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Diseases and Development

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DISEASES AND DEVELOPMENT*

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Abstract

This paper examines two related questions: what effects do infectious diseases exert on growth and development, and are they quantitatively important? We present evidence on the effect of health and infectious diseases on economic development using Hansen's (2000) endogenous threshold methodology. Taking into account various proxies for infectious diseases as potential threshold variables we show that countries are clustered in regimes that obey different growth paths and thus provide direct evidence of threshold effects. Motivated by this evidence we propose an epidemiological overlapping generations model where the transmission and incidence of an infectious disease depend upon economic incentives and rational behavior. The economic cost of the disease comes from its effect on mortality (infected individuals can die prematurely) and morbidity (lower productivity and/or lower flow of utility from a given consumption bundle). Our main theoretical finding is that if infectious diseases are particularly virulent or debilitating, growth- or development-traps are possible. Numerical results from a calibrated version of the model show that threshold effects of diseases are quantitatively important and in particular, significant health interventions are required to propel disease afflicted countries to a high-growth trajectory.

JEL Classification: O40, O47

Keywords: Infectious diseases, economic development, multiple growth paths, parameter heterogeneity, threshold variables

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1 Introduction

In studying Africa's persistently dismal economic performance, development economists have recently turned to health and infectious diseases for an answer. Citing evidence that 80% of the worldwide incidence of malaria is concentrated in Africa alone, Gallup and Sachs (2000) for instance, argue how such disease prevalence may be directly behind the continent's widespread poverty.

We begin with an exploration of the empirical relationship between infectious diseases and economic development. Despite the abundance of empirical work on diseases and growth, the aim of our empirical investigation is twofold.

First, we construct the most comprehensive list of proxies for infectious diseases to date. We consider well-documented aggregates such as life expectancy and adult mortality rates as well as more disaggregate measures such as indices for malaria, AIDS and tuberculosis. Our infectious disease data comes from various sources, notably the World Bank, the World Health Organization and publicly available ones such as Gallup and Sachs (2000) and McCarthy et al. (2000).

Second, we are specifically interested in seeing if there are non-linearities in the relationship between diseases and economic growth. To do so, we use Hansen's (2000) endogenous threshold methodology to search for multiple regimes. Hansen develops a statistical theory of threshold estimation in the regression context that allows for cross-section observations. Least squares estimation is considered and an asymptotic distribution theory for the regression estimates is developed. The main advantage of Hansen's methodology over, for instance, the Durlauf and Johnson (1995) regression-tree approach is that the former is based on an asymptotic distribution theory which can formally test the statistical significance of regimes selected by the data. Using our various proxies for infectious disease as potential threshold variables we show that countries are indeed clustered in regimes that obey different growth paths and thus give direct evidence of multiple equilibria.

We then propose a general equilibrium model of economic epidemiology to explain why health and diseases may induce non-linearities on the growth process. Epidemiological factors are introduced into a two-period overlapping generations model where the transmission and incidence of an infectious disease depend upon economic incentives and rational behavior. The economic cost of the disease comes from its effect on mortality (infected individuals can die prematurely) and morbidity (lower productivity and/or lower flow of utility from a given consumption bundle). Individuals born in the first period of their lives catch the disease from infected (old) individuals with whom

they are randomly matched. Their susceptibility to the disease from such encounters depends on preventive health investment undertaken early in life.

Individuals work in youth and invest in capital (broadly defined to include human and physical capital). Our aggregate technology is Ak to allow for endogenous growth. The interaction of rational disease behavior with savings-investment incentives generates an interesting pattern of development. If infectious diseases are particularly virulent or debilitating, thresholds effects are possible. Societies susceptible to such diseases, for example the tropics and underdeveloped regions, may simultaneously experience protracted, and high, incidence of infectious diseases and low economic growth. Regions where such diseases are not as debilitating, or that are relatively affluent, emerge from periods of slow growth and declining disease prevalence to take-off into sustained growth. In certain cases, these societies may temporarily experience contractionary behavior coupled with rising disease incidence before they start experiencing sustained growth and falling incidence of infectious diseases.

Finally, we calibrate our model to quantitatively assess the importance and likelihood of multiple growth regimes. Our results show that such regimes are plausible for reasonable parameter values, and that, substantial health interventions may be required to control infectious diseases and ensure that the economy takes off into sustained growth.

The paper is organized as follows. Section 2 describes our dataset and econometric methodology and presents empirical evidence on the effect of health and diseases on economic growth. In sections 2 and 3 we study a theoretical model of rational disease behavior that attempts to explain the evidence. Section 5 calibrates the model and presents numerical results on the quantitative effect of diseases on economic development.

2 Empirical Evidence

We begin by examining the empirical relationship between health, disease-prevalence and economic outcomes. We are interested specifically in non-linearities in the growth process. Accordingly we ask: do health/disease variables endogenously split cross-country data into multiple regimes obeying distinctly different growth paths? To answer this, we use Hansen's (2000) endogenous threshold methodology.

Table 1: Unconditional cross-sectional correlations between relevant variables

	$GROWTH$	$LIFEXP60$	$MALMORT$	$MALARIA$	$AIDS$
$GROWTH$	1				
$LIFEXP60$	0.0271	1			
$MALMORT$	-0.2542	-0.1135	1		
$MALARIA$	-0.2054	-0.1309	0.6693	1	
$AIDS$	-0.2412	—	—	—	1

2.1 Data

Our dataset is constructed using data series from the following sources: Penn World Table version 6.1 (PWT 6.1), the United Nations Statistics Division (UNSD), the World Health Organization (WHO), Barro and Lee (2001) and Gallup et al. (2001). For the typical Mankiw, Romer and Weil (1992) (MRW) growth regression variables, such as real per capita GDP (y), the share of investment to GDP (s_k), and population growth (n) we have used data from PWT 6.1 and schooling (s_h) from Barro and Lee (2001).[1] In addition, we have used different proxies for our health/disease variable including, *life expectancy* (UNSD-2001), *male mortality incidents*[2] (UN-2000), *malaria*[3] (Gallup et al.-2001) and *AIDS incidents*[4] (UNSD-2001).

The sample of countries considered is reduced from 96 countries (the original MRW sample using PWT 4.0) to 88 (using PWT 6.1). The countries excluded from the MRW sample are Algeria, Burma, Ecuador, Haiti, Liberia, Somalia, Sudan and W. Germany. PWT 6.1includes two additional countries, Botswana and Mauritius increasing the sample from 88 to 90 countries. Unfortunately, the schooling dataset from Barro and Lee (2001) is missing 17 observations. After subtracting the missing schooling observations our sample goes down to 73 countries. Finally, we drop one to three additional observations due to the health/disease data availability reducing our final sample further to 70-72 observations (depending on the health/disease proxy used). Summary statistics of key variables are provided in the data appendix.[5]

[1]For schooling we have used the average years of schooling for people over the age of 15. Our estimation results were robust to considering the alternative measure of average years of schooling for people over the age of 25.

[2]The male mortality proxy represents the number of mortality incidents in 100,000 people.

[3]The malaria proxy represents the percentage of a country's area with malaria.

[4]The AIDS proxy represents the incident rate. More specifically, it is the number of AIDS incidents per 100,000 people.

[5]The complete dataset used in this paper accompanied with detailed discussion regarding the data sources is available by the authors upon request.

Table 1 reports unconditional correlations of our relevant variables for our cross-section of 72 countries. Two points are worth noting here. First notice, that none of our four health/disease proxies are highly correlated with per capita GDP growth. Second, even though correlations have the expected sign, their magnitudes are not sizable. The correlation that stands out is the positive and high correlation between male mortality incidents and malaria.[6]

2.2 Methodology

In this paper we follow the endogenous threshold methodology of Hansen (2000). Hansen develops a statistical theory of threshold estimation in the regression context that allows for cross-section observations. Least squares estimation is considered and an asymptotic distribution theory for the regression estimates is developed. The main advantage of Hansen's methodology over, for instance, the Durlauf and Johnson (1995) regression-tree approach is that the former is based on an asymptotic distribution theory which can formally test the statistical significance of regimes selected by the data.[7]

In line with most empirical growth literature, we consider the following MRW growth regression equation:

$$\ln y_{i,2000} - \ln y_{i,1960} = a_0 + a_1 \ln y_{i,1960} + a_2 \ln s_{ik} + a_3 \ln s_{ih} + a_4 \ln(n_i + g + \delta) + \varepsilon_i, \qquad (1)$$

where y_i is per capita GDP for country i, s_k is physical capital investment (investment share to GDP), s_h is human capital investment (schooling), n is population growth, $g+\delta = 0.05$ as in MRW, and ε is a random error term.

We search for multiple regimes in the data by using four different proxies of health/disease variables, namely initial (1960) life expectancy ($LIFEXP60$), initial (1960) male mortality incidents ($MALMORT$), initial (1966) percentage of a country's area with malaria ($MALARIA$) and average (1979-2000) AIDS incidents ($AIDS$) as potential threshold variables. Consistent with Durlauf and Johnson (1995) and Hansen (2000), we have chosen *initial* values of these proxies to minimize the potential problem of endogeneity. However, we have made an exception with the AIDS data and have used average (rather than initial) values to minimize the enormous measurement error associated with initial periods of the new disease.

[6] Notice that we do not report the correlations between $AIDS$ (measured as the average from 1979 to 2000) and $LIFEXP60$, $MALMORT$, $MALARIA$ (measured in 1960) as they are meaningless.

[7] For a detailed discussion of the statistical theory for threshold estimation in linear regressions, see Hansen (2000).

2.3 Threshold Estimation using Health Variables

Since Hansen's (2000) statistical theory allows for one threshold for each threshold variable, we proceed using the heteroskedasticity-consistent Lagrange Multiplier test for a threshold developed by Hansen (1996). We start our threshold estimation exercise by considering the two aggregate measures of health, namely initial life expectancy and male mortality. Subsequently, we test for our more disaggregated proxies of health, namely malaria and AIDS.

First, we consider $LIFEXP60$ as a potential threshold variable. It is shown that the threshold model using $LIFEXP60$ is significant with p-value of 0.075, indicating that there exists a sample split based on initial life expectancy. The top panel in Figure 1 presents the normalized likelihood ratio sequence $LR_n^*(\gamma)$ statistic as a function of the output threshold. The least-squares estimate γ is the value that minimizes the function $LR_n^*(\gamma)$ which occurs at $\hat{\gamma} = 46.3$. The asymptotic 95% critical value (7.35) is shown by the dotted line and where it crosses $LR_n^*(\gamma)$ displays the confidence set $[35.5, 51.3]$. $LIFEXP60$ as a threshold variable divides our full sample of 70 countries into a low-life-expectancy regime (below or equal to 46.3) with 26 countries and a high-life-expectancy regime (above 46.3) with 44 countries.

Second, we consider $MALMORT$ as a threshold variable. We find that this threshold model is highly significant (in fact the most significant of all models considered) with p-value of 0.007, pointing to strong evidence of a split based on male mortality incidents. The second panel in Figure 1 presents the normalized likelihood ratio statistic as a function of $MALMORT$. The point estimate for the literacy threshold is $\hat{\gamma} = 438$ with the 95% confidence interval $[406, 571]$. $MALMORT$ splits our entire sample of 72 countries into a low-male-mortality regime (below or equal to 438) with 49 countries, and a high-male-mortality regime (above 438) with 23 countries.

Third, we consider $MALARIA$ as a threshold variable. We find that this threshold model is also significant with p-value of 0.059, pointing to evidence of an endogenous split based on the percentage of a country's area with malaria. The third panel in Figure 1 presents the normalized likelihood ratio statistic as a function of $MALARIA$. The point estimate for the literacy threshold is $\hat{\gamma} = 0.98$ with the 95% confidence interval $[0.98, 0.98]$. $MALMORT$ splits our sample of 72 countries into two regimes; a low-malaria regime (below or equal to 0.98) with 56 countries, and a high-malaria regime (above 0.98) with 16 countries.

Finally, $AIDS$ is considered as a threshold variable. The bootstrap test statistic for this variable

Figure 1: Likelihood ratio statistics as a function of threshold variables

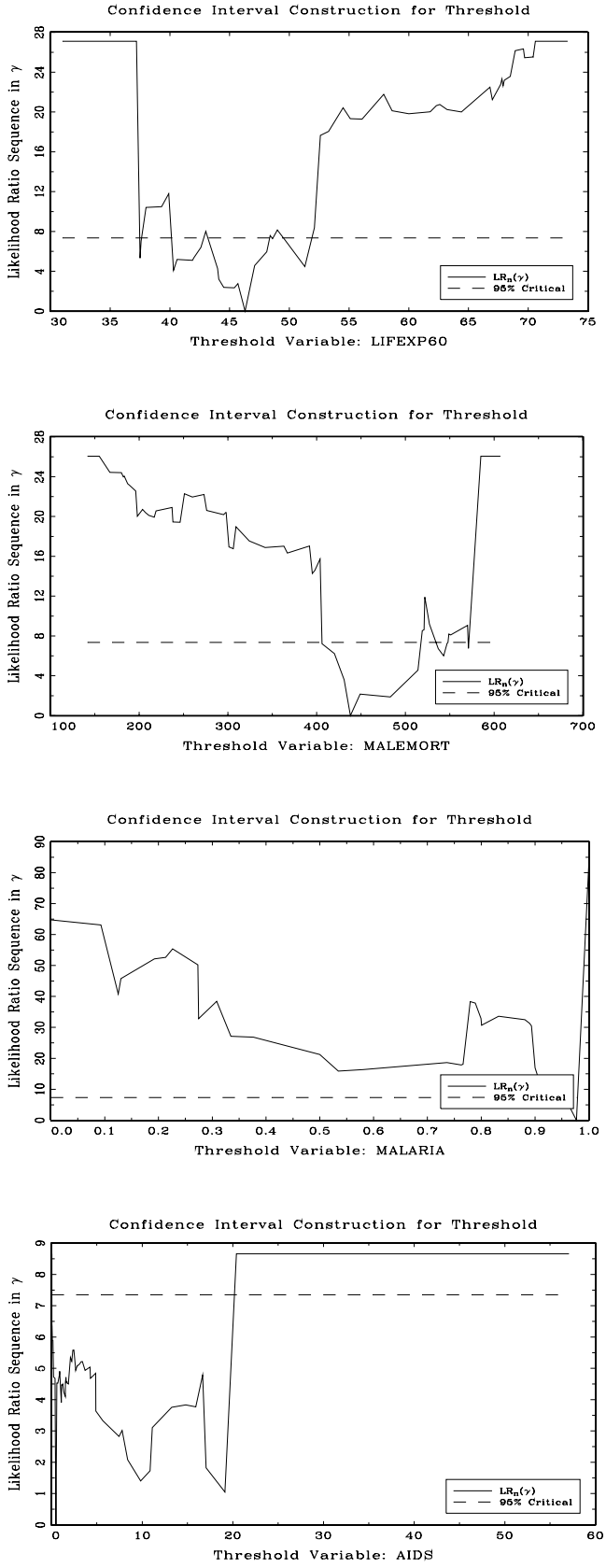
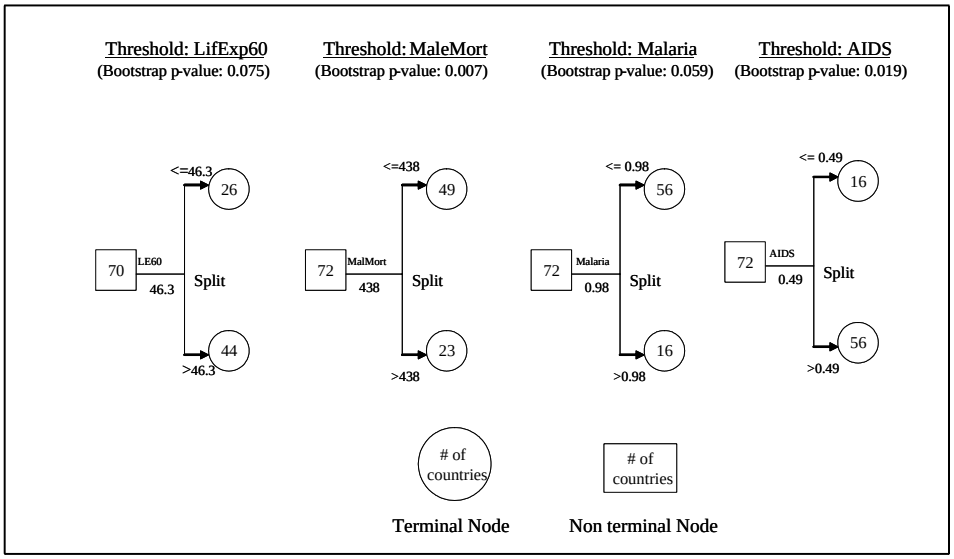


Figure 2: Regression trees obtained using threshold estimation



is quite highly significant as well with p-value of 0.019. In particular, $\hat{\gamma} = 0.49$ with the 95% confidence interval $[0.042, 19.12]$ and the entire sample of 72 countries can be split into two regimes with 16 countries (below or equal to 0.49) and 56 countries (above 0.49). The bottom panel in Figure 1 presents the normalized likelihood ratio statistic as a function of $AIDS$.[8]

Figure 2 presents regression tree diagrams that illustrate our threshold estimation results obtained under the four threshold variable models. Non-terminal nodes are illustrated by squares whereas terminal nodes are illustrated by circles. The numbers inside the squares and circles show the number of countries in each node. The point estimates for each threshold variable are presented on the rays connecting the nodes. Tables 2-3 present the countries in the four pairs of regimes, respective to the four threshold models.

These results suggest that there is strong evidence in favor of threshold effects. Our findings are quite remarkable because threshold effects emerge regardless of the health/disease proxy used in our estimation. In other words, our results are robust to health aggregated data (such as the

[8]In addition to these potential threshold variables, we have also considered two alternative datasets for malaria (Gallup et al.-2001 and WHO-1982) and female mortality incidents (UNSD). To save space we do not report these results as they are qualitatively similar to our baseline results. These results are available by the authors upon request. We have also collected data on Tuberculosis (UNSD). Unfortunately, data for initial periods (i.e. 1960-1970) existed only for a small subset (38 countries) of our sample which made threshold estimation unworkable.

Table 2: List of countries in subsamples using LifeExp60 and MalMort as threshold variables

Thresh.: LifeExp60			**Thresh.: MalMort**		
Regime 1 (*Low*)	*Regime* 2 (*High*)		*Regime* 1 (*Low*)		*Regime* 2 (*High*)
Bangladesh	Argentina	Mexico	Argentina	Mexico	Bangladesh
Bolivia	Australia	Netherlands	Australia	Netherlands	Bolivia
Cameroon	Austria	New Zealand	Austria	New Zealand	Botswana
Central Afr.	Belgium	Norway	Belgium	Nicaragua	Cameroon
Dominican Rep.	Botswana	Paraguay	Brazil	Norway	Central Afr.
Ghana	Brazil	Philippines	Canada	Pakistan	Ghana
Guatemala	Canada	Portugal	Chile	Panama	Guatemala
Honduras	Chile	Singapore	Colombia	Paraguay	Indonesia
India	Colombia	Spain	Costa Rica	Peru	Kenya
Indonesia	Costa Rica	Sri Lanka	Denmark	Portugal	Malawi
Jordan	Denmark	Sweden	Dominican Rep.	Singapore	Mali
Kenya	El Salvador	Switzerland	El Salvador	Spain	Mauritania
Malawi	Finland	Syria	Finland	Sri Lanka	Mozambique
Mali	France	Thailand	France	Sweden	Nepal
Mauritania	Greece	Trinidad	Greece	Switzerland	Niger
Mozambique	Ireland	Tunisia	Honduras	Syria	Papua
Nepal	Israel	Turkey	Hong Kong	Thailand	Philippines
Nicaragua	Italy	United Kingdom	India	Trinidad	Senegal
Pakistan	Jamaica	United States	Ireland	Tunisia	Sierra Leone
Papua	Japan	Uruguay	Israel	Turkey	Togo
Peru	Korea	Venezuela	Italy	United Kingdom	Uganda
Senegal	Malaysia	Zimbabwe	Jamaica	United States	Zambia
Sierra Leone			Japan	Uruguay	Zimbabwe
Togo			Korea	Venezuela	
Uganda			Malaysia		
Zambia					
(26)	(44)		(49)		(23)

commonly used in the empirical literature, life expectancy and mortality rates) as well as health disaggregated data (such as the malaria and AIDS). More importantly, these results provide strong support to the main implication of our theoretical model.

2.4 Subsample Regression Results

Next, we turn our attention to the estimation of equation (1) for the four threshold models and four pairs of regimes. Table 4 presents estimates for each regime. These estimates provide strong evidence in favor of parameter heterogeneity and the presence of threshold effects. The heterogeneity of the coefficient estimates across regimes is striking, as coefficient estimates vary considerably

Table 3: List of countries in subsamples using the four proxies of health variables

Thresh.: Malaria			**Thresh.: AIDS**		
Regime 1 (*Low*)		*Regime* 2 (*High*)	*Regime* 1 (*Low*)	*Regime* 2 (*High*)	
Argentina	Mauritania	Bangladesh	Bangladesh	Argentina	Mauritania
Australia	Mexico	Cameroon	Bolivia	Australia	Mexico
Austria	Nepal	Central Afr.	Finland	Austria	Mozambique
Belgium	Netherlands	Dominican Rep.	Hong Kong	Belgium	Netherlands
Bolivia	New Zealand	Ghana	India	Botswana	New Zealand
Brazil	Nicaragua	Kenya	Indonesia	Brazil	Niger
Canada	Niger	Korea	Japan	Cameroon	Norway
Chile	Norway	Malawi	Jordan	Canada	Panama
Colombia	Pakistan	Mozambique	Korea	Central Afr.	Papua
Costa Rica	Panama	Paraguay	Nicaragua	Chile	Paraguay
Denmark	Papua	Senegal	Pakistan	Colombia	Peru
El Salvador	Peru	Sierra Leone	Philippines	Costa Rica	Portugal
Finland	Philippines	Togo	Sri Lanka	Denmark	Senegal
France	Portugal	Uganda	Syria	Dominican Rep.	Sierra Leone
Greece	Singapore	Zambia	Tunisia	El Salvador	Singapore
Guatemala	Spain	Zimbabwe	Turkey	France	Spain
Honduras	Sri Lanka			Ghana	Sweden
Hong Kong	Sweden			Greece	Switzerland
India	Switzerland			Guatemala	Thailand
Indonesia	Syria			Honduras	Togo
Ireland	Thailand			Ireland	Trinidad
Israel	Trinidad			Israel	Uganda
Italy	Tunisia			Italy	United Kingdom
Jamaica	Turkey			Jamaica	United States
Japan	United Kingdom			Kenya	Uruguay
Jordan	United States			Malawi	Venezuela
Malaysia	Uruguay			Malaysia	Zambia
Mali	Venezuela			Mali	Zimbabwe
(56)		(16)	(16)	(56)	

in sign and magnitude. Below, we provide a brief summary (not a complete account) of the huge variation in estimates across the regime pairs in the four models.

Starting with the $LIFEXP60$ threshold model, notice how the point estimates for $\ln s_{ik}$ vary from -0.3098 and significant at the 5% level in Regime 1, to 0.4037 and significant at the 1% level in Regime 2. There is remarkable variation in the estimates associated with physical capital investment in the $MALMORT$ threshold model regimes as well. The coefficient estimates vary from -0.7960 and significant at the 1% level in Regime 1 to 0.2751 and significant at the 10% level in Regime 2.

Turning to the $MALARIA$ threshold model, it is once again pretty astonishing how different coefficient estimates are between the two regimes with regards to initial output ($\ln y_{i,60}$), investment ($\ln s_{ik}$) and schooling ($\ln s_{ih}$). Finally, a look at the coefficient estimates in the two regimes under the $AIDS$ threshold model, reveals the same trend as in the other three models. In particular, point estimates for $\ln s_{ik}$ vary from 1.6768 and significant at the 1% level in Regime 1, to 0.0435 and insignificant in Regime 2. Furthermore, point estimates for $\ln s_{ih}$ vary from 0.2960 and significant at the 10% level in Regime 1, to 0.5598 and significant at the 1% level in Regime 2.

These regression results reinforce our primary empirical and theoretical result of multiple growth paths due to health/disease variables.[9]

2.5 Robustness Analysis

Our threshold estimation results are clearly subject to the endogeneity problem that plagues the majority of growth regressions. Caner and Hansen (2004) have recently extended Hansen (2000) by proposing a method of estimating and conducting inference on the thresholds in a model with endogenous explanatory variables and an exogenous threshold variable. At the same time, Bloom, Canning, and Sevilla (2003) and others have argued that geographical and institutional variables are suitable instruments and have made important contributions to the literature using such variables. Using these two developments we examine the robustness of our results to an alterative methodology that, at least partly, corrects for the endogeneity problem. Our goal is to discover whether the thresholds previously found are in any way a product of the assumption of regressor exogeneity. See the technical appendix for a summary of the Caner-Hansen method.

[9]More generally, our results are consistent with Durlauf and Johnson (1995), Durlauf, Kourtellos and Minkin (2001), Liu and Stengos (1999), and Masanjala and Papageorgiou (2004), among others, who find strong nonlinearities in the growth process.

Table 4: Subsample regressions

Specification	**Extended Solow Model** (PWT 6.1)			
	LifeExp60		**MaleMort**	
	Regime 1 (*Low*)	**Regime 2** (*High*)	**Regime 1** (*Low*)	**Regime 2** (*High*)
Unrestricted				
Constant	2.9726 (1.8109)	5.9426*** (1.7662)	6.2131*** (1.7921)	2.9265* (1.7852)
$\ln y_{i,60}$	−0.3839** (0.1564)	−0.7159*** (0.1167)	−0.7960*** (0.0992)	0.2751* (0.1614)
$\ln s_{ik}$	−0.3098** (0.1264)	0.4037*** (0.1552)	0.4618*** (0.1609)	−0.0591 (0.2318)
$\ln s_{ih}$	0.3467*** (0.1366)	0.6122*** (0.2382)	0.7804*** (0.2377)	0.3739*** (0.1350)
$\ln(n_i + g + \delta)$	−0.4991* (0.2348)	−1.2806*** (0.4232)	−1.6331*** (0.4128)	−0.3090 (0.2300)
Adj. R^2	0.07	0.57	0.64	0.22
Obs.	26	44	49	23

Specification	**Extended Solow Model** (PWT 6.1)			
	Malaria		**AIDS**	
	Regime 1 (*Low*)	**Regime 2** (*High*)	**Regime 1** (*Low*)	**Regime 2** (*High*)
Unrestricted				
Constant	6.3490*** (1.0455)	0.7566 (2.4850)	10.252*** (1.8257)	3.4230*** (1.2896)
$\ln(Y/L)_{i,60}$	−0.6174*** (0.0763)	0.0783 (0.2171)	−0.8344*** (0.1265)	−0.3240*** (0.1089)
$\ln s_{ik}$	0.5025*** (0.1772)	−0.2408 (0.2096)	1.6768*** (0.2122)	0.0435 (0.1696)
$\ln s_{ih}$	0.5621*** (0.1071)	0.9239*** (0.3047)	0.2960* (0.1633)	0.5598*** (0.0878)
$\ln(n_i + g + \delta)$	−0.8177*** (0.3327)	−0.7935 (0.4525)	−0.4902 (0.4243)	−0.6670*** (0.2101)
Adj. R^2	0.58	0.24	0.83	0.38
Obs.	56	16	16	56

Notes: Standard errors are given in parentheses. White's heteroskedasticity correction was used. *** Significantly different from 0 at the 1% level. ** Significantly different from 0 at the 5% level. * Significantly different from 0 at the 10% level.

The instrument sets used are borrowed from Johnson and Papageorgiou (2005). In particular we use a set of five instruments. Three of our instruments are from Bloom, Canning, and Sevilla (2003): LAT, the absolute value of the latitude of the approximate center of the country; $COAST$, the percentage of land area within 100 km of the coast; $LAND$, an indicator variable that is one for landlocked countries and zero otherwise; and, HOM, the percentage of the population in the largest single group that shares the same ethnic, linguistic, and religious characteristics. We add to the these variables two variables constructed from the extensive set of landscape and climate variables in the Center for International Earth Science Information Network's (CIESIN) National Aggregates of Geospatial Data: Population, Landscape and Climate Estimates (PLACE) dataset. The first of these is $TEMPER$, the percentage of land area with a temperate climate and the second is $BIOME$, the percentage of land area in biome classes described as "temperate" or "Mediterranean."[10]

Figure 3 presents the normalized likelihood ratio sequence $LR_n^*(\gamma)$ statistic as a function of the threshold variable using the Caner-Hansen methodology. The top panel represents Life Expectancy, the middle panel Male Mortality and the bottom panel AIDS. It is obvious that these figures compare well with those in Figure 1. We could not confirm our results for Malaria as we failed to obtain convergence in the procedure used. The most likely reason for this is the large number of zeros and ones that appear in the Malaria data series.[11]

The main result from this exercise is that the thresholds found in our empirical analysis are not due to the regressor exogeneity assumption. More precisely, we find that exactly the same thresholds for Life Expectancy, Male Mortality and AIDS continue to exist when we use the Caner-Hansen methodology.

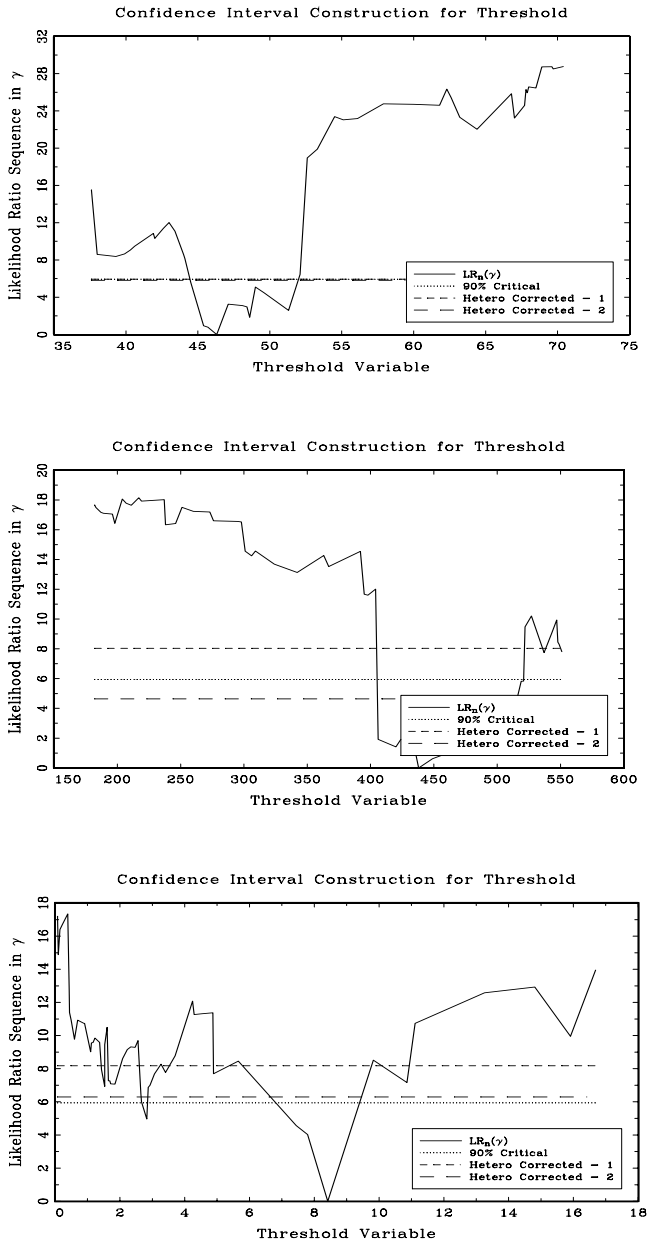
3 Model

Evidence supporting thresholds in cross-country data, as we uncovered in the previous section, are usually taken to imply non-ergodicity or path dependence. In this section we will propose a theoretical model motivated by the evidence and show how infectious diseases can adversely affect economic incentives and lead to poverty traps.

[10] For more details on the construction of these dataset we refer the interested reader to Johnson and Papageorgiou (2005).

[11] More precisely a matrix required for the estimation of coefficient estimates was not positive definite.

Figure 3: Likelihood ratio statistics as a function of threshold variables



Our framework is a discrete time, infinite horizon economy populated by overlapping generations of families. Each individual is born with an efficiency labor endowment of $(1, 0)$ and potentially lives for two periods. The modification we introduce to the standard model is the possibility of contacting an infectious disease early in life and premature death from it.

3.1 Infectious Diseases

Infectious diseases inflict three types of costs on an individual. First, he is less productive at work, supplying only $1 - \theta$ units of efficiency labor instead of unity. Secondly, there is an utility cost associated with being infected: he derives a utility flow of $\delta u(c)$ instead of $u(c)$ from a consumption bundle c, where $\delta \in (0, 1)$. Thirdly, an infected young individual faces the risk of premature death. In particular, he may not live through his entire old-age.[12]

Young individuals undertake preventive health investment, x_t, early in life. This may take the form of net food intake (that is, nutrients available for cellular growth), personal care and hygiene, accessing clinical facilities and related medical expenditure. It may even take the form of abstaining from risky behavior, particularly in the context of sexually transmitted infectious diseases such as AIDS. What is key is that such investment is privately costly and improves an individual's resistance to infectious diseases. We model these costs in terms of income, but just as likely, they can be foregone utility, for instance in sexually transmitted HIV (see Geoffard and Philipson, 1996, for example).

Diseases spread from infected older individuals to susceptible younger ones through a process of random matching. In particular, a susceptible young person randomly meets $\mu > 1$ older individuals during the course of his youth. Not all of these older individuals will be infected and not all encounters with infected persons result in transmission. In particular, given his preventive health investment x_t, the probability that a young individual gets infected from such a matching is $\pi(x_t)$, where $\pi' < 0$ and $-\pi'(0) > \infty$.[13]

Let p_t denote the probability of being infected for a typical member of generation t. If encounters are independent, the probability of not getting infected by the end of youth equals the product (across meetings) of not being infected. The probability of being infected after one match is

[12] We may think of an additional cost: infected individuals need to invest in curative health care. This is easily incorporated but does not add much to our analysis.

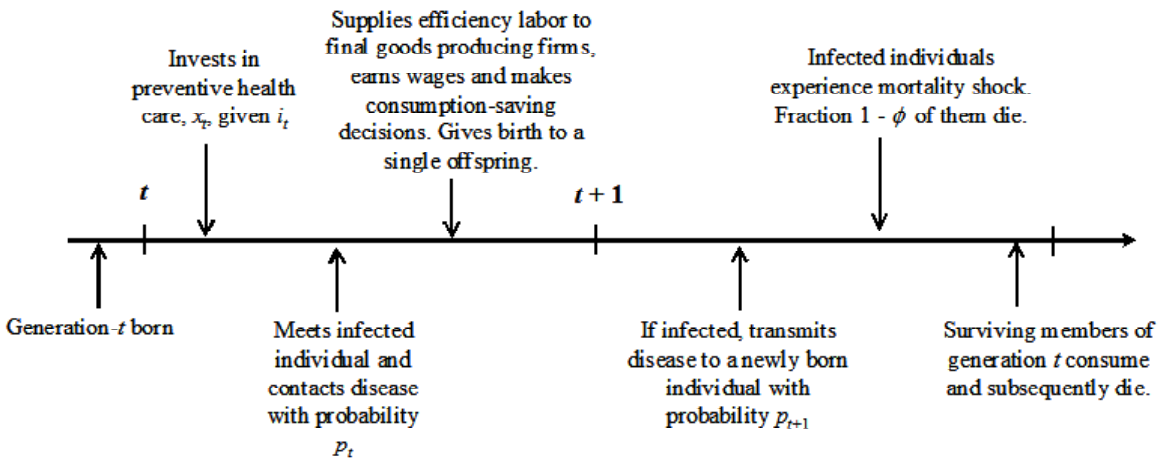
[13] This last assumption is not necessary to obtain multiple growth paths, it only makes the model's predictions richer.

the probability of meeting an infected individual (i_t) times the probability of getting infected in match (π_t), that is, $i_t\pi(x_t)$. Hence, the probability of not being infected after μ matches is simply $[1 - i_t\pi(x_t)]^\mu$. Thus,

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

We outline the timeline of events in Figure 4. Note that preventive health investments are chosen *ex ante*, before an individual meets an infected older person. Once a young individual's infection status is determined, his consumption and savings choices are determined in the usual manner.

Figure 4: Timeline of Events



3.2 Preferences

Preferences and individual behavior are disease contingent. We consider first decisions of an uninfected individual whose health investment has successfully protected him from the disease. The period utility function $u(c)$ is increasing, twice continuously differentiable with $u' > 0$, $u'' < 0$. In addition, it is homothetic, current and future consumptions are normal goods, and $-cu''(c)/u'(c) < 1$. The individual 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

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

$$c_{2t+1}^U = R_{t+1} z_t^U.$$

where w is the wage per efficiency unit of labor, z denotes savings and x is given, as per decisions made early in period t.[14] Hereafter we shall tag variables by U and I to denote decisions and outcomes for uninfected and infected individuals respectively.

An infected individual faces a constant probability $\phi \in (0,1)$ of dying from the disease in old-age. Assuming zero utility from death, he maximizes expected lifetime utility

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

subject to

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

where τ_{t+1} denotes lumpsum transfers received from the government. We assume an institutional setup whereby the government collects and distributes the assets of the prematurely deceased among surviving *infected* individuals.[15] Clearly transfers per surviving infected individual will be

$$\tau_{t+1} = \left(\frac{1-\phi}{\phi}\right) R_{t+1} z_t^I \tag{2}$$

in equilibrium.

For convenience, we assume the parametric utility function, $u(c) = c^{1-\sigma}$, $\sigma \in (0,1)$. Optimal savings for uninfected and infected individuals are easily verified to be

$$z_t^U = z^U(w_t; x_t, R_{t+1}) = \left[\frac{\beta^{1/\sigma} R_{t+1}^{1/\sigma-1}}{1+\beta^{1/\sigma} R_{t+1}^{1/\sigma-1}}\right](w_t - x_t),$$

$$z_t^I = z^I(w_t; x_t, R_{t+1}) = \left[\frac{(\beta\phi)^{1/\sigma} R_{t+1}^{1/\sigma-1}}{1+(\beta\phi)^{1/\sigma} R_{t+1}^{1/\sigma-1}}\right][(1-\theta)w_t - x_t] - \left[\frac{1}{1+(\beta\phi)^{1/\sigma} R_{t+1}^{1/\sigma-1}}\right]\frac{\tau_{t+1}}{R_{t+1}}.$$

[14] Implicitly we are assuming that x is financed by borrowings early in youth, at zero interest, and repaid after the labor market clears.

[15] Alternatively, we could have assumed perfect annuities market which would give the same qualitative results (see Chakraborty, 2004).

Substituting these savings decisions gives us the following two indirect lifetime utility functions

$$V^U(x_t; w_t) = \left[\left(w_t - x_t - z_t^U\right)^{1-\sigma} + \beta\left(R_{t+1} z_t^U\right)^{1-\sigma}\right]$$

$$V^I(x_t; w_t) = \delta\left[\left((1-\theta)w_t - x_t - z_t^I\right)^{1-\sigma} + \beta\phi\left(R_{t+1} z_t^I\right)^{1-\sigma}\right],$$

contingent on preventive health investment x_t.

Newborns choose the optimal level of x_t to maximize expected lifetime utility. Recall that i_t denotes the fraction of old agents who are infected. Given the random matching process mentioned above, a young individual's probability of catching the disease is $p_t = \mu\pi(x_t)i_t$ if less than one, and 1 otherwise. Hence, individuals choose x_t to maximize expected lifetime utility

$$\mu\pi(x_t)i_t V^I(x_t; w_t) + \left[1 - \mu\pi(x_t)i_t\right] V^U(x_t; w_t)$$

at the beginning of period t. The first order condition for this is

$$-\pi'(x_t)i_t\left(V_t^U - V_t^I\right) \geq \mu\pi_t i_t\left(-\frac{\partial V_t^I}{\partial x_t}\right) + \left[1 - \mu\pi_t i_t\right]\left(-\frac{\partial V_t^U}{\partial x_t}\right) \tag{3}$$

for $x_t \geq 0$. This conditions states that, for individuals to be willing to invest in disease prevention, the marginal benefit from living longer and experiencing a healthier life has to outweigh the marginal cost of foregoing current income.

All savings are invested in capital, which are rented out to final goods producing firms, earning a return equal to the rental rate. We assume that at $t = 0$, the initial old generation is endowed with a stock of capital K_0. An exogenously specified fraction i_0 of them also suffer from infectious diseases. The depreciation rate on capital is set equal to one without loss of generality.

3.3 Technology

A continuum of firms operate in perfectly competitive markets to produce the final good using capital and efficiency units of labor. To accommodate the possibility of endogenous growth we posit a firm-specific constant-returns technology exhibiting learning-by-doing externalities

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

where A, $b > 0$ and $\bar{k}$ denotes the average capital per effective unit of labor across firms. Standard factor pricing relationships under such externalities imply that

$$w_t = (1-\alpha)Ak_t + b \equiv w(k_t),$$

$$R_t = \alpha A \equiv R.$$

4 General Equilibrium Analysis

We begin by substituting equilibrium prices and transfers into the savings functions to obtain

$$z_t^U = s^U \left[w(k_t) - x(w_t, i_t) \right] \equiv z^U(k_t, i_t)$$

and

$$z_t^I = s^I \left[(1-\theta) w(k_t) - x(w_t, i_t) \right] \equiv z^I(k_t, i_t)$$

where,

$$s^U \equiv \left[\frac{\beta^{1/\sigma} R^{1/\sigma - 1}}{1 + \beta^{1/\sigma} R^{1/\sigma - 1}} \right], \; s^I \equiv \left[\frac{\phi (\beta\phi)^{1/\sigma} R^{1/\sigma - 1}}{1 + \phi (\beta\phi)^{1/\sigma} R^{1/\sigma - 1}} \right].$$

Note that $z_t^I < z_t^U$, as expected, since $\phi < 1$ and $\theta > 0$. Substituting these savings functions into the indirect utility functions, we obtain

$$\begin{aligned} V_t^{U*} &= \left[\left(1 - s^U\right)^{1-\sigma} + \beta R^{1-\sigma} \left(s^U\right)^{1-\sigma} \right] \left(w(k_t) - x_t \right)^{1-\sigma} \\ &\equiv \zeta^U \left(w(k_t) - x_t \right)^{1-\sigma} \end{aligned}$$

and

$$\begin{aligned} V_t^{I*} &= \phi^\sigma \left[\left(\frac{1 - s^I}{\phi + (1-\phi) s^I} \right)^{1-\sigma} + \beta R^{1-\sigma} \left(s^I\right)^{1-\sigma} \right] \left((1-\theta) w(k_t) - x_t \right)^{1-\sigma} \\ &\equiv \zeta^I \left((1-\theta) w(k_t) - x_t \right)^{1-\sigma} \end{aligned}$$

Next we substitute equilibrium prices and savings functions into the first order condition for the choice health investment. Note that individuals do not take into account equilibrium transfers (2) when making health investment decisions. Accordingly, (3) becomes

$$\begin{aligned} & \eta^I \mu \pi_t i_t \left((1-\theta) w(k_t) - x_t \right)^{-\sigma} + \eta^U \left[1 - \mu \pi_t i_t \right] \left(w(k_t) - x_t \right)^{-\sigma} \\ \leq \; & -\pi'(x_t) i_t \left[V_t^{U*} - V_t^{I*} \right] \end{aligned} \tag{4}$$

where

$$\begin{aligned} \eta^U &\equiv (1-\sigma) \left[\left(1 - s^U\right)^{1-\sigma} + \beta \left(R s^U \right)^{1-\sigma} \right], \\ \eta^I &\equiv \delta \phi (1-\sigma) \left[\frac{\left(1 - s^I\right)^{1-\sigma} + \beta \phi^\sigma \left(R s^I \right)^{1-\sigma}}{\phi + (1-\phi) s^I} \right]. \end{aligned}$$

Two possibilities arise depending on whether or not health investment yields positive returns. If, at $x_t = 0$, (4) holds as a strict inequality, optimal investment will be $x_t = 0$. The right-hand side of the expression above constitutes the marginal benefit, in the form of higher net utility, from lowering one's chance of catching the infectious disease. On the left, is the marginal utility cost of that investment, since health investment entails a lower current and, possibly, future consumption. Optimal health investment is zero as long as the utility cost dominates, that is, returns to health investment are negative at $x = 0$. Our assumption, $\pi'(0) > -\infty$, that is an individual cannot infinitely lower his disease risk through finite health investments, ensures that such a possibility can arise. Intuitively we expect this to occur at levels of low income and high prevalence rates of the infectious disease; private actions, in these situations, are likely to negligibly improve an individual's chance of leading a healthy life. Rewriting (4) above as,

$$\begin{aligned} & \left[\eta^U \mu\pi(0) + \eta^I (1-\theta)^{-\sigma} \pi(0)^U \left\{1 - \mu\pi(0) i_t\right\} / i_t\right] / w(k_t) \\ > \quad & -\pi'(0) \left\{\zeta^U - (1-\theta)^{-\sigma} \zeta^I\right\}, \end{aligned}$$

or,

$$\chi(k_t, i_t) > 0,$$

we note that $\partial\chi/\partial k > 0$ and $\partial\chi/\partial i > 0$, that is, private returns from preventive health investment are negative at low values of k and high values of i.

For (k_t, i_t) combinations such that $\chi(k_t, i_t) < 0$, optimal investment in health will be positive. In this case, at an interior optimum, we have

$$\begin{aligned} & \eta^I \mu\pi_t i_t \left((1-\theta) w(k_t) - x_t\right)^{-\sigma} + \eta^U \left[1 - \mu\pi_t i_t\right] \left(w(k_t) - x_t\right)^{-\sigma} \\ = \quad & -\pi'(x_t) i_t \left[\zeta^U \left(w(k_t) - x_t\right)^{1-\sigma} - \zeta^I \left((1 - \theta(w(k_t) - x_t)^{1-\sigma}\right]. \end{aligned} \tag{5}$$

Optimal health investment

$$x_t = \chi(k_t, i_t)$$

satisfies $\partial\chi/\partial k > 0$ and $\partial\chi/\partial i > 0$.

4.1 Dynamics

With a continuum of young agents of measure one, by the law of large numbers, aggregate savings at t is simply

$$S_t = p_t z_t^I + (1 - p_t) z_t^U,$$

and asset market clearing requires that

$$K_{t+1} = S_t.$$

To express this in terms of capital per efficiency unit of labor, note that efficiency labor supply comprises of the labor of infected and uninfected individuals, that is,

$$L_{t+1} = (1-\theta)p_{t+1} + (1-p_{t+1}) = 1 - \theta p_{t+1}.$$

Higher is θ, less productive are infected workers and hence, less is effective labor supply.

Given optimal health investment $x(k_t, i_t)$, $p_t = p(k_t, i_t)$. Under plausible parametric assumptions, we can establish that $\partial p_t/\partial i_t > 0$, that is, even though a higher prevalence rate leads to higher investment in preventive care ($\partial x_t/\partial i_t > 0$), it is not enough to lower an individual's overall susceptibility to the disease.

The asset market clearing condition now requires that

$$k_{t+1} = \frac{p(k_t, i_t) z^I(k_t, i_t) + [1 - p(k_t, i_t)] z^U(k_t, i_t)}{1 - \theta p(i_{t+1})}, \tag{6}$$

while equilibrium disease dynamics are governed by

$$i_{t+1} = p(k_t, i_t). \tag{7}$$

Assume $\pi(0) > 1/\mu$ so that, in the absence of any preventive investment, the infection rate rises over time.

We examine equilibrium dynamics using a phase-portrait of the economy. Consider first (k, i) combinations such that $\chi(k, i) > 0$, that is, $x_t = 0$. Since health investment is zero and $\mu\pi(0) > 1$, we now have $i_{t+1} = \mu\pi(0) i_t > i_t$, so that $\Delta i_t > 0$ for all i_t. The entire susceptible population at t becomes infected ($p_{t+1} = 1$), hence the phase line for capital per effective unit of labor is given by

$$\begin{aligned} \Delta k_t &\geq 0 \Leftrightarrow k_{t+1} \geq k_t \\ &\Leftrightarrow s^I \left[(1-\alpha) A k_t + b\right] \geq k_t \\ &\Leftrightarrow k_t \leq k_1^* \equiv \frac{b}{1 - (1-\alpha) s^I A}, \end{aligned}$$

where $(1-\alpha)A < 1/s^I$ by assumption. Given the technology parameters (α, A), this is equivalent to assuming that mortality from infectious diseases is particularly high (since $\partial s^I/\partial \phi > 0$).

When health investment is positive, that is, $\chi(k,i) \geq 0$, the phase line for capital per effective worker is

$$\begin{aligned} \Delta k_t &\geq 0 \\ &\Leftrightarrow \quad \mu\widehat{\pi}(k_t,i_t)z^I(k_t,i_t) + [1-\mu\widehat{\pi}(k_t,i_t)]\, z^U(k_t,i_t) - k_t\,[1-\theta\mu\widehat{\pi}(k_t,i_t)] \geq 0 \end{aligned}$$

where $\widehat{\pi}(k_t,i_t) \equiv \pi(k_t,i_t)i_t$, while that for the infection rate is

$$\Delta i_t \geq 0 \Leftrightarrow \mu\widehat{\pi}(k_t,i_t) \geq i_t.$$

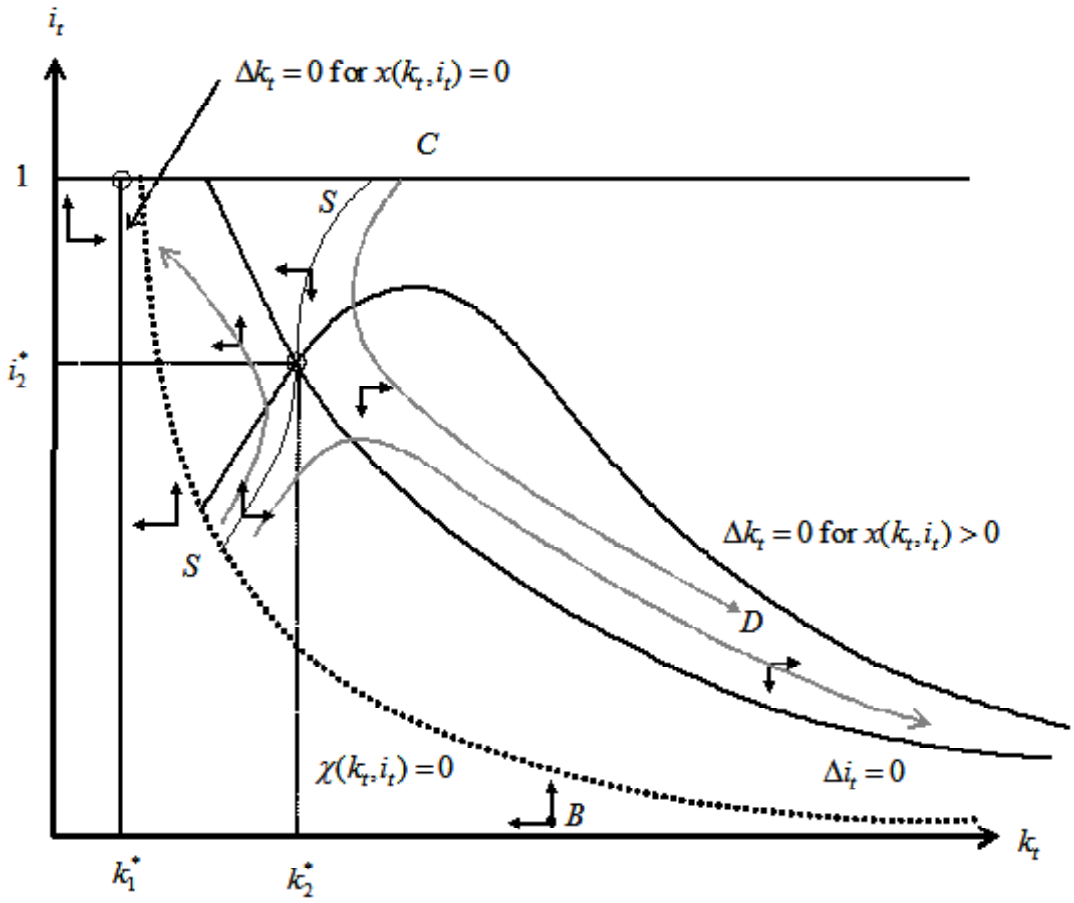
Figure 5 illustrates the phase-portrait with the dotted line corresponding to $\chi(k_t,i_t)=0$ below which $x_t = 0$. Vector fields indicate that the unique non-trivial steady-state $(k_1^*,1)$ is a sink. For capital per effective worker and infection rates lying above $\chi(k_t,i_t)$, the figure illustrates a single steady-state at (k_2^*,i_2^*) which is a saddle-point.

Recall that at $t=0$ the economy is endowed with K_0 units of capital owned by the initial old generation as well as with i_0, the fraction of that generation infected with diseases. Both k_0 and i_0 are predetermined variables, in other words. Hence, while $(k_1^*,1)$ is asymptotically stable, (k_2^*,i_2^*) is not. In particular, sequences of (k_t,i_t) which do not start exactly on the saddle-arm SS, converge either to $(k_1^*,1)$ or diverge to a sustained growth path along which infectious disease prevalence vanishes asymptotically.

Two specific features of equilibrium dynamics merit further discussion. First is the possibility of non-ergodicity in disease incidence and economic growth. At low levels of development (low k_0) and high disease prevalence (high i_0), equilibrium trajectories lead the economy to an inferior equilibrium of high disease prevalence and zero growth. For more favorable initial conditions, balanced growth results and infectious diseases disappear in the long-run. Note, however, that such favorable conditions do not preclude trajectories starting out with zero health investment, that is, below $\chi(k,i)=0$. Figure 5 depicts one scenario where the economy evolves along the trajectory comprising of point B and CD. Starting from B, the economy jumps to a point like C where disease prevalence is maximal (since individuals do not invest at all in preventive health care) and savings per worker lower. Thereafter diseases and the capital stock evolve along CD as diseases gradually decline and growth picks up. Overall, a period of epidemic (sharp increase in disease prevalence) is followed by gradual but steady economy progress.

Intuitively, the possibility of multiple growth paths depends on the economy's average propensity to invest. This propensity is a low s^I when everyone is infected and takes on the higher value s^U

Figure 5: Dynamics of Diseases and Capital per worker



for an uninfected population. Under our parametric restrictions, s^U allows for sustained economic growth but s^I does not (in our calibrations later we show why such restrictions are sensible). Hence, the initial infection rate i_0 determines an economy's willingness to invest in the future. Together with the initial capital stock per efficiency unit of labor, k_0, it also determines how affordable health investments are relative to income levels. For very high rates of infection and low levels of development, not only do people not invest much in the future, they do not undertake sufficient preventive investments either. This results in stagnation and endemic infectious diseases.

The second feature of the dynamics is the nature of transition to the high balanced growth path. Figure 5 shows that, for equilibrium trajectories starting with favorable initial conditions, the growth rate of output is initially low as individuals spend much of their labor incomes on disease prevention. Rewrite (6) in terms of the growth rate of capital per effective worker

$$\begin{aligned} 1+\gamma_t &\equiv \frac{k_{t+1}}{k_t} \\ &= \frac{1}{1-\theta p(i_{t+1})}\left[p(k_t,i_t)s^I\left\{\frac{(1-\theta)\left(b+(1-\alpha)Ak_t\right)-x(k_t,i_t)}{k_t}\right\}+\right. \\ &\quad \left.[1-p(k_t,i_t)]s^U\left\{\frac{b+(1-\alpha)Ak_t-x(k_t,i_t)}{k_t}\right\}\right]. \end{aligned}$$

It is clear that k_t has two effects on γ_t. First it lowers investment per unit of *capital* for both infected and uninfected individuals. At the same time, since i_t declines with sustained growth in income, health investments become increasingly less significant and this tends to boost the growth rate via higher savings. Declining infection incidence also shifts capital accumulation toward higher savings by uninfected workers.

The growth rate can exhibit a variety of interesting patterns. One possibility is threshold effects in the growth rate itself along equilibrium trajectories that converge to the sustained growth rate of $\gamma = (1-\alpha)s^U A - 1$. Here, the economy initially grows relatively slowly since infectious diseases require a significant fraction of labor income to be devoted to preventive care leaving little for investment opportunities. As the disease prevalence rate drops, savings per worker picks up and the economy grows rapidly. The initial phase of slow growth and disease declines is followed by a phase of rapid growth and still declining infection rates; eventually, as the infection rate drops arbitrarily close to zero, economic growth asymptotes the balanced growth path where capital and income per worker improve at the sustained rate γ.

Table 5: Benchmark parameter values

$\boldsymbol{\beta}$	0.99^{120}	$\boldsymbol{\alpha}$	0.67	$\boldsymbol{\theta}$	0.15
$\boldsymbol{\sigma}$	1	$\mathbf{A}$	23.82	$\boldsymbol{\phi}$	0.5
$\mathbf{b}$	1	$\boldsymbol{\mu}$	2	$\boldsymbol{\delta}$	0.9

5 Numerical Experiments

One of the multiple growth regime proposed by our theory is of particular concern since it is dynamically stable and characterized by zero growth, implying substantial human and economic costs. In this section, we explore whether these dynamic implications of the theory are robust to the choice of reasonable parameter values. The numerical exercises also help to assess the importance of the three types of costs inflicted by diseases — higher mortality, lower efficiency, and lower quality-of-life — in driving an economy towards a particular regime.

5.1 Calibration

Table 5 presents the benchmark values assigned to the different parameters. The model features overlapping generations of agents that potentially live for two periods. We follow de la Croix and Doepke (2003) and assume that one period, or generation, has a length of 30 years. We assign a value of 0.99^{120} to the discount factor (β); that is, 0.99 per quarter, which is standard in the real-business-cycle literature. The elasticity of consumption substitution (σ) is equalized to 1, another standard value. The production function displays three parameters: the technology parameter A, the capital elasticity α, and the labor productivity coefficient b. We normalize b to 1, and give α a value of 0.67. We are then looking at a broad concept of capital that includes physical, human and organizational capital. The value for A, in turn, is chosen so as to reproduce an annual long-run growth rate in the sustained growth equilibrium of 2%, approximately the average rate in the U.S.. This implies that A is chosen such that $s^U(1-\alpha)A$ equals 1.02^{30}.

We have no guidance about the technology that gives the probability of being infected in a match (π) nor the number of matches (μ). Hence, we choose the following simple form for the probability function, $\pi(x_t) = 1/(1+x_t)$, and assign $\mu = 2$, which satisfy the main properties, $\pi' < 0$, $\pi'(0) > -\infty$, $\pi(x_t) < 1$ for all $x_t > 0$, and $\mu\pi(0) > 1$.

We have more guidance about the parameters that govern the cost of diseases to individuals.

Dasgupta (1993), for example, finds that workers (in particular, farm workers in developing countries) are often incapacitated — too ill to work — for 15 to 20 days each year, and when they are at work, productivity may be severely constrained by a combination of malnutrition and parasitic and infectious diseases. His estimates suggest that potential income losses due to illness for poor nations are of the order of 15%. We then choose $\theta = 0.15$. Estimates by Birchenall (2004) for adult mortality rates from airbone diseases such as tuberculosis suggest a 50% chance of death. Then, we pick $\phi = 0.5$. Finally, there are some estimates on how ill health affects utility (or quality of life). In particular, Viscusi and Evans (1990) estimate that for injuries severe enough to generate a lost workday with an average duration of one month, the marginal utility of income falls to 0.92 in a logarithmic utility function model, although it can fall to 0.77 with a more flexible utility, where good health has a marginal utility of 1. This leads us to assign a value of 0.9 to the parameter δ.

5.2 Results

Figure 6 shows the different dynamics under the benchmark parameter values. All lines correspond to an initial infection rate of 0.3. We observe that the three different growth regimes implied by the theory are possible for reasonable parameters. For initial values of the stock of capital per capita below 0.3505, capital and output increase for the first 2 or 3 generations, but later decline and converge to a zero-growth steady-state. Over there, output remains constant, all the population gets infected from the disease, and no investment in prevention is carried out. When the initial capital per capita is, on the other hand, larger than the above value, the economy moves toward a balanced growth path characterized by sustained growth. In both scenarios, it takes output about 12 generations to reach its long-run path.

The solid line in Figure 6 corresponds to the unstable long-run equilibrium. This means, in our case, that for $i_0 = 0.3$ there is only one level of K_0 (in particular, $K_0 = 0.3505$) that generates an adjustment path that converges to that steady state. Over there, the values shown by the output variable and the infection rates are higher than in the other poverty trap, but the economy does not enjoys sustained long-run growth. Convergence to this third steady state is faster, it takes only 4 generations.

The second set of results are contained in Figure 7. It presents the effect of changes in the parameters related to the three types of costs induced by bad health. We consider a decrease in utility, with a new value of δ of 0.7. We analyze a decline in the probability of surviving if infected,

Figure 6: The three growth regimes, benchmark parameters

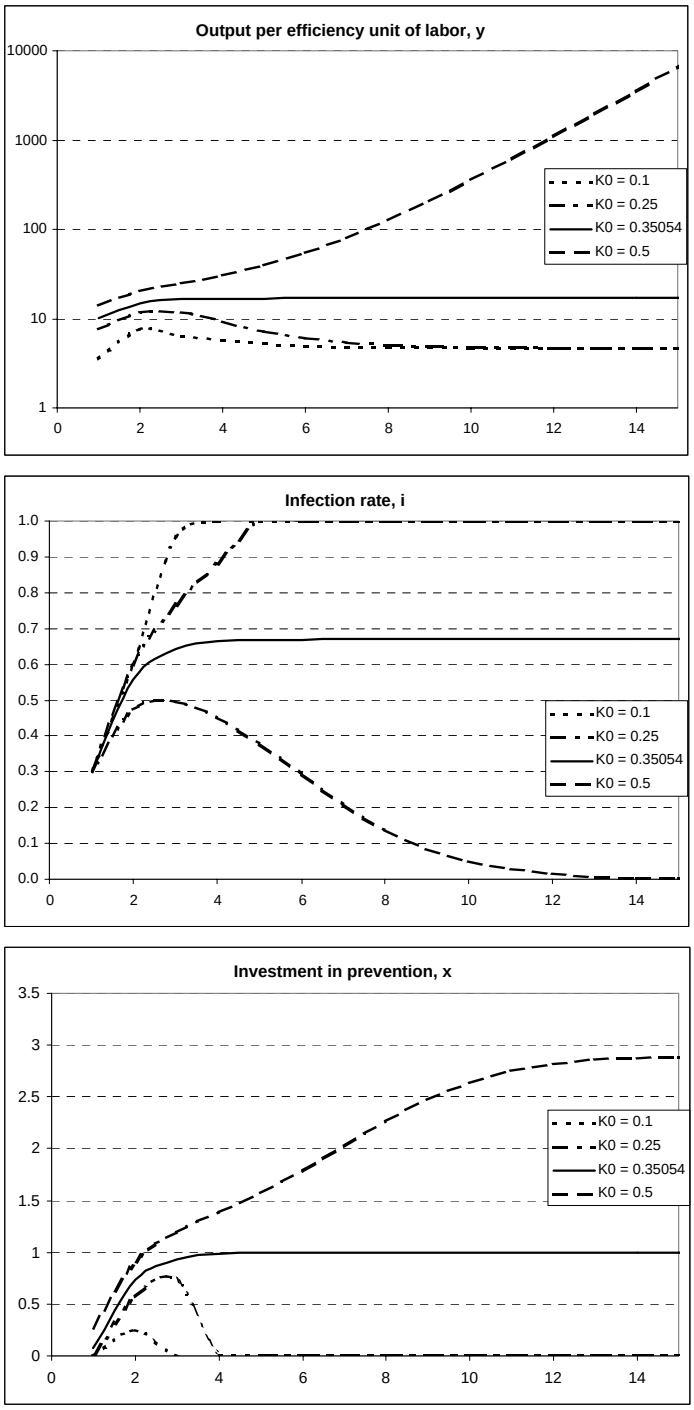
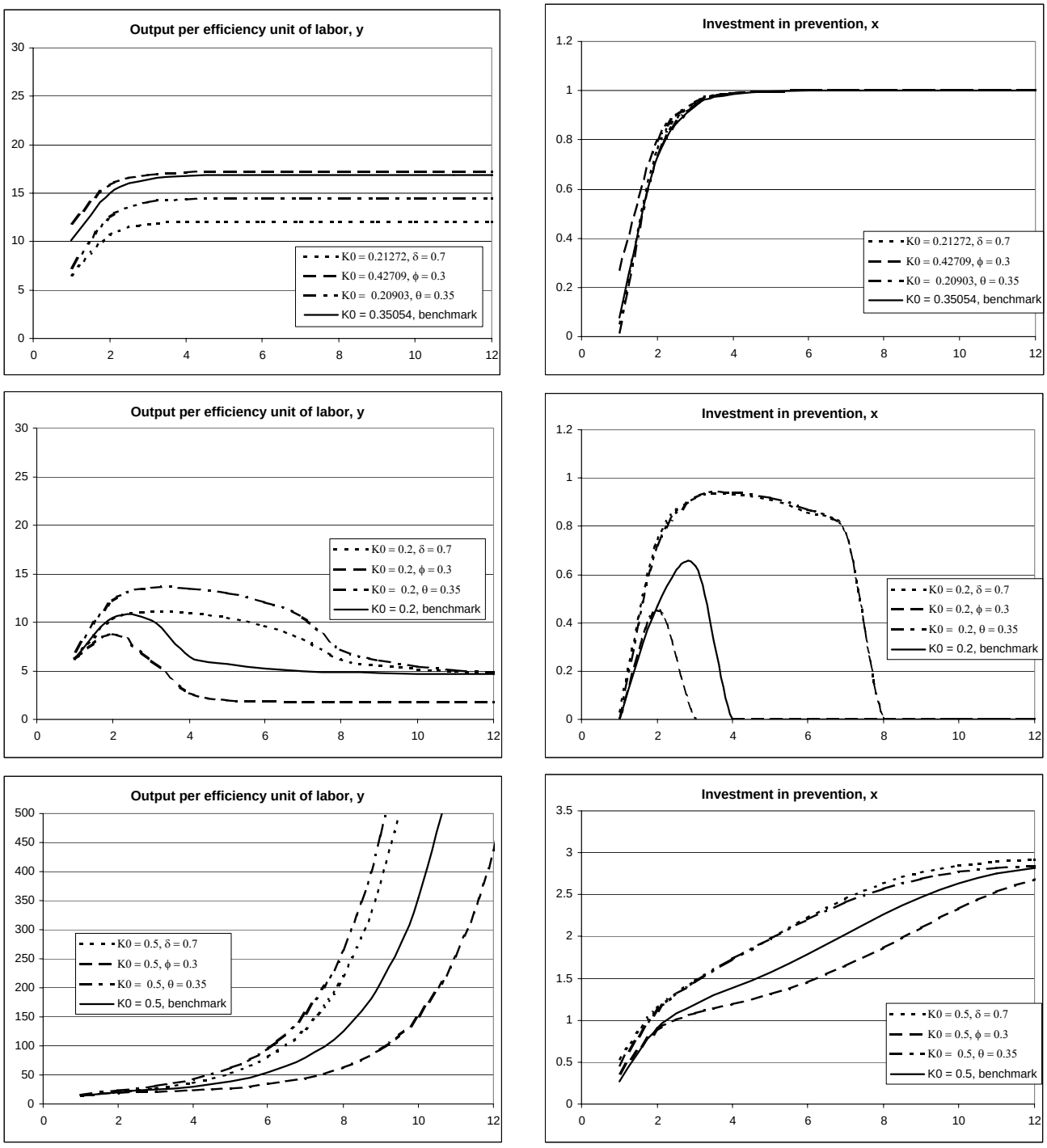


Figure 7: Effect of changes in three types of costs



with $\phi = 0.3$. Finally, we also check the effect of rising the efficiency loss for an infected adult to $\theta = 0.35$. The first, second, and third rows in Figure 7 present these effects on the unstable poverty trap, the stable poverty trap, and the sustained growth equilibrium, respectively. Only charts for output (first column) and for investment in prevention (second row) are presented. The evolution of the infection rate follows the same patterns as in Figure 6, and shows differences between different cases that are the same as the ones shown by prevention investment. For this reason the charts for i_t are omitted.

The first row of charts gives information about whether a higher cost of bad health implies a higher chance of ending in a poverty trap. We see that, compared to the benchmark case, only a higher probability of dying implies this negative effect. In particular, when ϕ declines from 0.5 to 0.3, the minimum stock of capital required to jump to perpetual growth rises from 0.3505 to 0.4271. A higher reduction in efficiency or in utility have the opposite impact. When $\theta = 0.35$ and $\delta = 0.7$, it becomes more difficult to fall into a poverty trap and, in particular, to achieve sustained growth the values of K_0 must be above 0.2090 and 0.2127, respectively.

The stable poverty trap is also affected by changes in the costs of bad health. The second row in Figure 7 suggests that output levels in the low equilibrium decline when the probability of not surviving to the infectious illness increases. This is also the case if the loss in efficient-labor units rises. Regarding this second scenario, notice that even thought the chart shows identical steady state values of y for the benchmark case and for $\theta = 0.35$, the former corresponds to $\theta = 0.15$ and, therefore, implies larger levels of steady-state output per capita. Only when the utility cost goes up, the level of output in the stable poverty trap does not decrease. However, agents in this last case are also worse off than in the benchmark, because they obtain lower utility for all levels of consumption. Finally, the last row of charts give the sustained-growth equilibrium path. In all cases, starting values are $i_0 = 0.3$ and $K_0 = 0.5$. The LHS chart simply shows that economies further away from its threshold level of K_0 (given in first row of charts) grow faster.

An interesting result is that in all scenarios, the steady-state investment in prevention is the same in the three equilibria. The second column of charts in Figure 7 also suggest that prevention may be key in explaining the effects. Notice that the scenario that provides the worse outcome, a higher probability of not surviving, is the only one that produces, on average, a lower investment in prevention than the benchmark case for the same initial K_0. Increases in the other two costs lead the economy to invest more in prevention, reducing the probability of falling into perpetual

poverty.

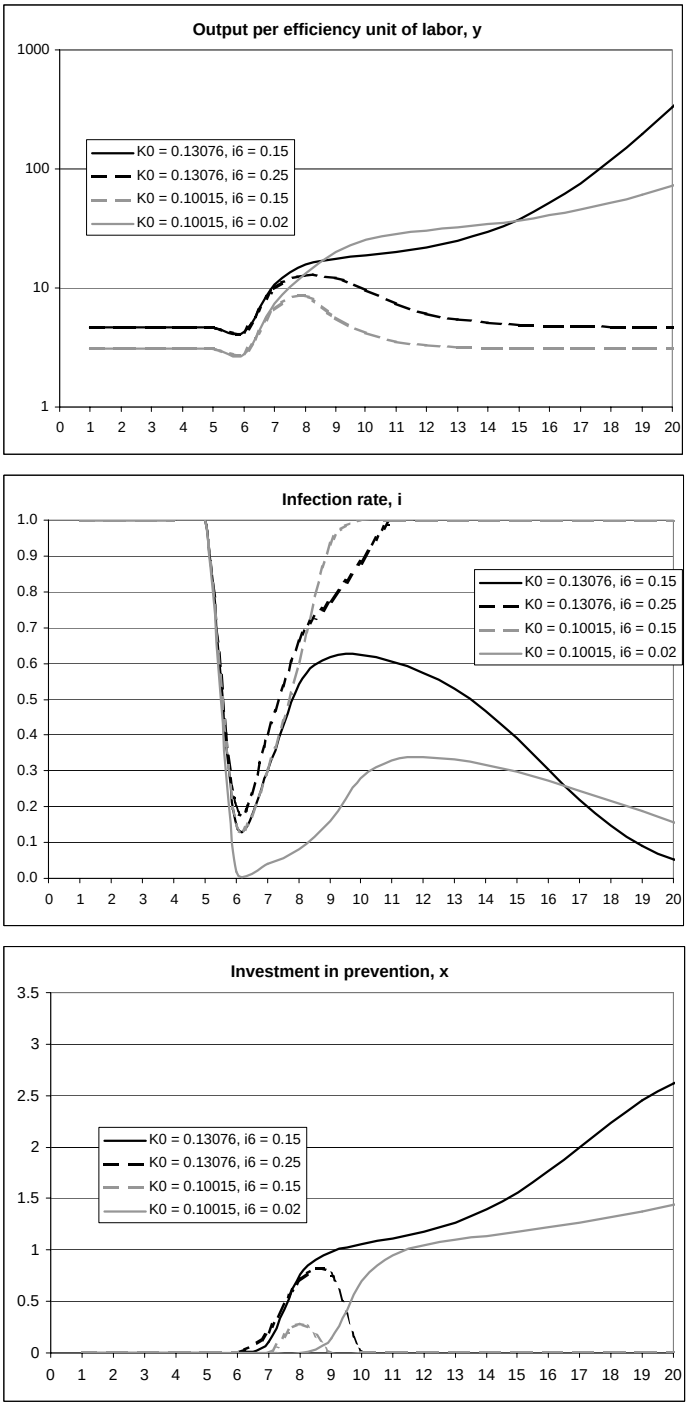
We can ask how costly can it be to get a nation out of a poverty trap induced by a high prevalence rate of infectious diseases. Suppose that the economy is at the worse steady-state of the benchmark scenario, that is, $i^* = 1$ and $K^* = 0.13076$. We ask: How much should the infection rate be reduced to generate perpetual growth? The answer is that it should be reduced at least to 0.15. Therefore, it requires a very big drop. The situation might be worse if the dynamic power of the economy is severely damaged. Let us assume that the initial situation of the economy corresponds to a value of $A = 17.8$. This represents a potential long-run annual growth rates of only 1%. The economy would be initially described by $i^* = 1$ and $K^* = 0.10015$. In this situation, a drop in the infection rate to 0.03 would be necessary to put the economy in the sustained growth path. The message from these experiments is that pulling economies out of the twin traps of poverty and disease may prove to be very costly, substantial reductions in the prevalence rate being necessary.

Figure 8 illustrates the capacity of prevalence rate reduction to take the economy into the growth path. We assume that the drop in i_t takes place in generation 6. When $A = 23.82$, the first five generations are characterized by $i^* = 1$ and $K^* = 0.13076$. For this case, if the reduction leads i_t to a value of 0.25, output starts growing fast at impact, and the economy invests heavily in prevention. However, the benefit of the initial drop only last around 5 generations. Generation 12's output is as low as generation 5's. This describes very well also the case $A = 17.8$, $i^* = 1$ and $K^* = 0.10015$, with $i_6 = 0.15$. Only if the drop is sufficiently large, for example, $i_6 = 0.15$ and $i_6 = 0.02$ for $A = 23.82$ and $A = 17.8$, respectively, the effect is permanent. In these last experiments, prevalence rates increase after the drop, but after generations 10 or 11 they fall toward zero and never grow again. The growth rate of output is negative at impact. This just reflects workers that recover their health and offer more efficient-labor units, but does not represents a fall in total output. After the initial drop, output shows its largest growth rates that later decline below their new balanced-growth-path value, and eventually recover.

6 Conclusion

There are several contributions this paper makes to the burgeoning literature on diseases and development. First, our empirical examination of how diseases affect aggregate outcomes is explicitly tailored to study possible non-linearities in the data. In this we adopt Hansen's (2000) threshold

Figure 8: Escaping the poverty trap



estimation technique. Results on infectious diseases, including malaria, suggest that geography and diseases may quite plausibly play a crucial role in shaping the destiny of many developing nations particularly those in the tropics that have consistently suffered from diseases and epidemics throughout history. Secondly, unlike exisiting works such as Acemoglu *et al.* (2002), Gallup and Sachs (2000) and McCarthy *et al.* (2000), we explicitly model the behavior of infectious diseases in an otherwise standard endogenous growth model. We show that such an epidemiological model can exhibit interesting dynamics and can, specifically, give rise to non-ergodicity in disease and growth paths. Finally, our numerical results from a calibrated version of the model suggest that multiple growth regimes are plausible for reasonable parameter values, and that, significant health interventions are required to control infectious diseases and ensure that the economy takes off into sustained growth.

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Technical Appendix

Caner and Hansen (2004) is the latest installment in a well developed theory of threshold estimation for linear models including, Caner (2002) and Hansen (1996, 2000). The particular contribution of Caner and Hansen (2004) is that their estimator and theory of inference permits endogenous explanatory variables. They develop a two-stage least squares estimator of the threshold parameter and a generalized method of moments estimator of the slope parameter. Here we give a brief, non-technical, outline of their methods.

The structural equation of interest is

$$\begin{aligned} y_i &= \theta_1' z_i + e_i, \quad q_i \leq \gamma \\ y_i &= \theta_2' z_i + e_i, \quad q_i > \gamma, \end{aligned}$$

where, for each i, y_i is the dependent variable, z_i is an m-vector of explanatory variables, q_i is the threshold variable assumed to be strictly exogenous, γ is the threshold parameter, θ_1 and θ_2 are m-vectors of slope parameters that may differ depending on the value of q_i, and e_i is a random disturbance term. The vector of explanatory variables is partitioned into a m_1 dimensional subset, z_{1i}, of exogenous variables uncorrelated with e_i, and a m_2 dimensional subset of endogenous variables, z_{2i}, correlated with e_i. The model can also be written as

$$y_i = \theta_1' z_i 1(q_i \leq \gamma) + \theta_2' z_i 1(q_i > \gamma) + e_i,$$

where $1(q_i \leq \gamma)$ is an indicator variable that is one if $q_i \leq \gamma$ and zero otherwise. In addition, this structural equation the model requires a suitable set of $k \geq m$ instrumental variables, x_i, that includes z_{1i}.

The threshold is estimated by $\hat{\gamma}$, the minimizer, over $\gamma \in \Gamma$, of the least squares residual sum of squares, $S_n(\gamma)$, from the regression of y_i on $\hat{z}_i 1(q_i \leq \gamma)$ and $\hat{z}_i 1(q_i > \gamma)$ where $\hat{z}_i$ is the predicted value of z_i from least squares estimation of the reduced form model $z_i = g(x_i, \pi) + u_i$, $E(u_i|x_i) = 0$ using a sample of n observations. The slope parameters θ_1 and θ_2 are estimated by application of GMM to each of the subsamples implied by the estimate $\hat{\gamma}$. The GMM estimators of θ_1 and θ_2 are shown to be consistent and asymtotically normal with covariance matrices that can be consistently estimated by the usual formulas.

To test for the presence of a threshold, Caner and Hansen propose testing $H_0 : \theta_1 = \theta_2$ using $SupW$, the maximizer over $\gamma \in \Gamma$ of $W_n(\gamma)$, the Wald statistic for H_0 given γ. They give the asymptotic distribution of $SupW$ and describe a procedure to simulate the distribution so that p-values can be obtained. Asymptotically valid confidence intervals for γ can be constructed as $\hat{\Gamma} = \{\gamma : LR_n(\gamma) \leq D\}$ where D is the 95% percentile of the asymptotic distribution of the "likelihood-ratio-like" statistic $LR_n(\gamma) = n\frac{S_n(\gamma) - S_n(\hat{\gamma})}{S_n(\hat{\gamma})}$. Caner and Hansen suggest using plots of $LR_n(\gamma)$ against γ to assess the estimator $\hat{\gamma}$ and its precision. We follow this suggestion but defer discussion of the interpretation of the plots until later in the paper. To test for the presence of a threshold, Caner and Hansen propose testing $H_0 : \theta_1 = \theta_2$ using $SupW$, the maximizer over $\gamma \in \Gamma$ of $W_n(\gamma)$, the Wald statistic for H_0 given γ. They give the asymptotic distribution of $SupW$ and describe a procedure to simulate the distribution so that p-values can be obtained.

Data Appendix

Country	GDP/L_{1960}	GDP/L_{2000}	Inv. Shr.	Sch.15	LE_{1960}	MalMort	Malaria	AIDS
Angola*	3127.01	2889.79	7.39	—	32	567	1.000	3.543
Argentina	8711.30	12790.55	17.57	6.97	64.4	217	0.065	2.665
Australia	12593.15	28479.77	24.68	10.25	70.41	207	0.000	2.872
Austria	8249.95	25820.15	25.98	7.60	68	211	0.000	1.429
B. Faso*	953.07	1256.60	8.51	—	33.9	586	1.000	11.231
Bangladesh	1329.38	2174.65	9.99	1.64	39.3	551	1.000	0.001
Belgium	8815.76	25233.67	23.95	8.53	69.7	189	0.000	1.690
Benin*	1339.08	1614.03	6.44	—	35.9	561	1.000	5.417
Bolivia	2995.62	3360.68	10.11	5.00	41.9	483	0.144	0.217
Botswana	1257.05	4391.11	16.06	3.52	49	537	—	57.084
Brazil	3032.10	8609.03	20.62	3.56	53.3	295	0.731	7.439
Burundi*	671.21	705.24	5.01	—	40.5	568	1.000	27.484
C. Afri. Rep.	2697.12	2230.58	4.64	1.46	37.5	519	1.000	20.396
Cameroon	2107.24	2592.84	6.84	2.52	38	602	1.000	10.862
Canada	12475.10	29408.37	21.86	10.20	70.6	183	0.000	3.064
Chad*	1512.28	1197.65	9.58	—	34	554	0.989	12.770
Chile	4798.46	11531.51	15.95	6.26	56.1	238	0.000	1.714
Colombia	3291.72	7028.28	11.51	4.16	55.1	298	0.491	1.526
Congo*	619.64	2267.26	22.97	—	40.6	583	1.000	168.6
Costa Rica	4556.99	7382.88	14.16	5.02	60	246	0.117	3.405
Denmark	12576.14	29214.81	23.52	9.13	72	154	0.000	2.467
Dom. Repub.	2213.66	6269.87	12.38	3.78	40.6	342	1.000	4.290
Egypt*	1875.87	5001.41	6.99	—	44.9	337	0.692	0.029
El Salvador	4272.25	5655.84	7.01	3.41	48.6	363	0.985	3.269
Ethiopia*	678.42	834.83	4.39	—	34.9	475	0.976	—
Finland	8833.28	26137.43	26.51	7.54	68	260	0.000	0.388
France	9012.38	24837.32	24.67	6.51	69.6	207	0.000	4.872
Ghana	1114.30	1743.42	10.05	2.99	44	514	1.000	16.679
Greece	4805.43	16211.37	25.85	6.73	67.9	180	0.000	1.226
Guatemala	3044.46	4686.98	8.08	2.44	44.1	514	0.548	2.223
Honduras	2202.64	2619.72	12.19	3.24	44.5	392	0.562	13.256
Hong Kong	3885.03	28985.27	25.83	7.59	—	301	0.500	0.494
I. Coast*	2045.07	2396.20	8.08	—	37.9	594	1.000	—
India	1057.29	3029.63	11.53	3.24	42.6	398	0.262	0.073
Indonesia	1170.83	4309.68	12.21	3.38	39.9	605	0.763	0.016
Ireland	6077.69	29673.53	17.91	7.69	68.9	187	0.000	1.095
Israel	6757.70	19731.21	28.12	8.72	67.8	146	0.001	0.883
Italy	7870.53	23409.35	24.85	5.94	68.5	155	0.000	4.531
Jamaica	3466.06	4398.90	19.10	3.99	62.6	251	0.000	11.113
Japan	5352.21	26607.24	31.09	8.39	66.8	238	0.000	0.095
Jordan	2938.15	4764.41	13.14	4.55	45.7	—	0.011	0.147
Kenya	1057.90	1660.26	11.19	2.91	43.4	547	0.961	24.953
Madagascar*	1560.13	1077.37	2.85	—	38.9	413	1.000	0.021
Malawi	543.02	1051.85	13.27	2.50	37.2	522	0.748	40.971

Note: * denotes the countries excluded from the original sample due to schooling data constraints. Our sample is therefore reduced from 90 possible countries to 73.

Data Appendix (cont.)

Country	GDP/L_{1960}	GDP/L_{1985}	Inv. Shr.	$Sch._{15}$	LE_{1960}	MalMort	Malaria	AIDS
Malaysia	2732.36	11881.36	20.13	4.94	52.1	438	0.983	1.642
Mali	1254.45	1266.79	7.32	0.53	34.1	589	0.997	3.707
Mauritania	1335.74	1980.26	5.95	4.78	37.5	587	0.992	2.082
Mauritius*	4113.44	15986.39	12.34	—	58.1	316	—	0.402
Mexico	5157.89	10517.05	18.30	4.91	55.1	306	0.208	2.927
Morocco*	1685.31	4360.63	12.89	—	45.4	370	0.954	0.207
Mozambique	1982.94	1220.98	2.48	0.77	33.7	592	1.000	9.823
N. Zealand	13810.97	21675.12	20.98	10.88	70.7	196	0.000	1.170
Nepan	962.16	1916.18	11.16	1.00	37.6	527	0.869	—
Netherlands	10876.95	26779.49	24.25	7.84	73	142	0.000	1.847
Nicaragua	3783.31	2262.50	10.84	3.29	45.4	431	0.225	0.431
Niger	2054.86	1147.25	6.99	0.59	—	543	0.994	4.239
Nigeria*	1336.88	1007.52	7.53	—	38.2	551	1.000	3.148
Norway	9463.86	30064.78	31.90	8.79	73.3	144	0.000	0.907
Pakistan	810.79	2373.30	13.10	2.34	43	420	0.710	0.011
Panama	2972.48	7183.22	20.20	6.33	—	273	0.940	7.794
Papua N. Gui.	2728.78	3911.93	11.80	1.82	37.2	521	0.962	1.527
Paraguay	3148.70	5870.30	10.68	4.94	63.2	219	1.000	0.695
Peru	4118.79	5509.87	20.01	5.45	46.3	404	0.186	2.335
Philippines	2633.35	4290.72	14.66	6.21	51.3	449	0.736	0.042
Portugal	4014.21	17372.31	20.87	3.73	62.3	198	0.000	4.889
Rwanda*	1206.77	1169.07	3.36	—	41.5	545	1.000	18.540
S. Africa*	2312.31	2177.97	12.35	—	48	525	0.005	2.247
S.Korea	1890.55	17871.16	27.34	7.68	52.6	406	1.000	0.031
Senegal	1338.46	1555.28	7.08	2.09	37.5	570	1.000	2.555
Sierra Leone	2756.36	12319.64	2.78	1.56	31	585	1.000	0.596
Singapore	6205.21	9009.19	41.20	5.64	63.2	309	1.000	1.366
Spain	5374.52	19526.76	24.41	5.48	67.7	167	0.000	8.412
Sri Lanka	1696.02	4135.50	10.26	5.37	58.6	204	0.100	0.047
Sweeden	11425.35	25994.72	22.24	9.37	72.7	146	0.000	1.120
Switzerland	16985.64	28795.71	27.73	9.25	70.7	189	0.000	5.656
Syria	1803.30	5126.30	12.44	3.62	48.4	367	0.384	0.036
Tanzania*	493.96	632.15	24.51	—	39.3	606	1.000	26.060
Thailand	1412.79	7888.54	29.44	4.89	54.5	395	0.846	17.047
Togo	1140.31	1121.38	7.07	1.90	38	548	1.000	21.910
Tri. & Tabago	5569.74	12713.71	9.95	6.32	61.8	237	0.000	15.906
Tunisia	2546.42	8021.32	18.25	2.79	47.1	324	0.968	0.442
Turkey	3385.51	8031.86	14.89	3.47	48.1	182	0.254	0.038
U.K.	10947.38	24535.04	18.31	8.33	70.4	198	0.000	1.604
U.S.A.	14527.60	37255.59	18.67	10.66	69.7	219	0.000	14.809
Uganda	729.18	1233.64	2.07	2.11	42	549	0.931	19.119
Uruguay	6823.21	10989.42	11.76	6.37	67	187	0.000	2.831
Venezuela	10188.71	7726.34	16.22	4.69	57.9	276	0.045	2.647
Zaire*	1232.06	969.50	4.83	—	—	568	1.000	—
Zambia	1557.93	1152.75	18.66	3.80	40.3	607	1.000	39.767
Zimbabwe	1595.46	3191.33	24.75	3.10	49.6	571	1.000	55.472

Note: * denotes the countries excluded from the original sample due to schooling data constraints.