Environment and Development Economics 10: 719–743 © 2005 Cambridge University Press
doi:10.1017/S1355770X05002500 Printed in the United Kingdom

Dynamics of tuberculosis and economic growth

DORIANA DELFINO
Environment Department, University of York, YO10 5DD, UK.
E-mail: dd109@york.ac.uk

PETER J. SIMMONS
Department of Economics and Related Studies, University of York, YO10 5DD, UK. E-mail: ps1@york.ac.uk

ABSTRACT. We find significant empirical links between the health structure of the population and the productive system of an economy that is subject to infectious disease, in particular tuberculosis. Consequently, development policy, aimed to improve the level of prosperity, has significant effects on the demographic-epidemiological dynamics of the population. Moreover, infectious diseases, such as tuberculosis, affect the size of the labour force and the productive capacity of the economy. We combine a Lotka-Volterra type system capturing the dynamics of TB epidemics with a Solow-Swan growth model where output is produced from capital and healthy labour. The demographic-epidemological parameters of the Lotka-Volterra type system are functions of GDP per healthy worker. We find significant differences between the most prosperous quartile and the rest of the world. In the former, the disease is eradicated whereas in the lowest three quartiles we predict damped capital and epidemic cycles converging to a population which is about 80 per cent of capacity and of whom about 2 per cent are TB infected. It follows that raising productivity in the lower quartiles is a critical policy aim.

1. Introduction

Infectious diseases have long been identified as a main cause of mortality in the human population. Despite improvements in medical knowledge, sanitation, personal hygiene, diet, and health education, disease-causing micro-organisms are reappearing throughout the world.[1] Some are newly identified (such as HIV/AIDS, Hanta Virus, Lassa Fever, and Ebola) and many are diseases (such as tuberculosis, malaria, plague, and measles) that, although thought to be well controlled, are emerging in more virulent forms, such as certain drug-resistant strains of bacteria (for instance, the emergence of a multidrug-resistant tuberculosis epidemic).

Tuberculosis (TB) has always been a serious public health threat both in developing and developed countries. During the nineteenth century and most of the first half of the twentieth century, TB, 'the white plague', was the

[1] Worldwide, infectious diseases killed more than 17 million people in 2001. By comparison, the death toll from cancer was 6.1 million, from cardiovascular diseases 9.7 million, and from cerebrovascular diseases, such as stroke, 4 million (WHO, 2002).

main cause of death throughout the World (Bates, 1992; Dubos and Dubos, 1987). As the living–working conditions of the population improved, TB morbidity and mortality declined (McKeown and Record, 1962). Local studies of TB mortality support the role of prosperity reforms affecting poverty and nutrition in controlling TB (Farmer, 1996; Freudenberg, 1995; Frieden, 1994; Lerner, 1993). However, historical studies also support the hypothesis that public health interventions, including housing policies, public education, improvements in infrastructures, and sanitation, leading to behavioural changes had an impact on the falling rates of mortality from TB (Cronje', 1984; Hardy, 1993; McFarlane, 1989). By the beginning of the 1970s, TB was no longer a significant health problem for developed countries. However, from 1985, TB became the leading cause of morbidity and mortality in developing countries (Raviglione and Luelmo, 1996) and of increasing morbidity in areas of poverty in developed and transition countries (Bhatti *et al.*, 1995; Elender *et al.*, 1998; Frieden *et al.*, 1995; Raviglione and Luelmo, 1996). Today, deaths from TB in Russia have risen to the level they were at two decades ago and TB is rising in many large cities in western Europe, such as London, where it strongly correlates with indices of deprivation (Bardsley *et al.*, 1998; Hayward, 1998; McKee and Jacobson, 2000).

Among the different factors that have led to the recent TB epidemic, poverty, malnutrition, overcrowded housing, together with poor hygienic conditions associated with rapidly increasing urbanization, stand out as primary causes (Dievler and Pappas, 1999; Ginsberg, 1998; Jaramillo, 1999; Young *et al.*, 1997).

The resurgence of TB epidemics and the insurgence of a new epidemic of multidrug-resistant tuberculosis initiated a controversy regarding the relative value of targeted general medical interventions (such as Directly Observed Therapy Short-course – DOTS) versus general economic prosperity reforms or public health infrastructure policies, involving investment in housing and infrastructures (Bayer and Dupuis, 1995; Brudney and Dobkin, 1991; Wallace, 1990). Some studies (Bayer, 1996; Sumartojo, 1993) claimed that current TB control measures have failed because they have not dealt with the underlying social conditions of poverty that predispose individuals to TB. Consequently, such limited interventions could by themselves achieve only partial and temporary success. Other studies (Brudney and Dobkin, 1991; Landesman, 1992), in contrast, interpreted the deterioration of the public health infrastructure since the 1960s – the loss of financing for TB screening, follow-up and treatment, housing and health care services both in urban and rural areas – as the main cause of the re-emergence of TB in the 1980s. Supporters of this view argue that control of TB epidemics requires a structured commitment to find an adequate public health infrastructure and urban planning policies independent of the need for general prosperity changes (Landesman, 1992; Rodrigues and Smith, 1990).

In this paper we divide the population into healthy susceptible and TB infected/infectious individuals and analyse their dynamic interactions where infected/infectious people behave as 'predators' of healthy susceptible 'preys'. We then identify four demographic–epidemiological parameters, which are critical in determining the evolution of TB. These are

the net growth rate of healthy/susceptible individuals, the force of infection, the recovery rate, and the overall mortality rate of TB infected/infectious cases.

Using cross-section data for 120 countries (World Bank, 2002; WHO, 2002) we find that there are significant links between these parameters and economic prosperity, working in the expected direction but in a non-linear way. We estimate the coefficients of a logistic type function for each of the four parameters to quantify these links and find that the predicted values of the parameters replicate the data by quartile of prosperity very closely. An additional important parameter is the carrying capacity of the environment, which bounds the total population above.

Using the predicted values of these five parameters in a two-equation Lotka–Volterra (1925–1926) type of model (including effects of recovery and of the limited carrying capacity (Murray, 2002)) for each quartile of prosperity, we find that the poorest three quartiles exhibit epidemic cycles, whereas the richest quartile has a time path of population in which TB dies out and the population grows to the carrying capacity of the environment. Thus, we have evidence for the importance of economic prosperity in controlling TB.

A critical quantity that determines the form of the dynamics that arises is the difference between the product of the force of infection with the carrying capacity of the environment and the sum of the recovery and TB mortality rates. This can be interpretated as the TB reproductive coefficient: TB infections from the existing stock decay at a rate equal to the sum of those recovering or dying, but new infections occur at a maximum rate equal to the product of the force of infection with the carrying capacity. If the reproductive coefficient is negative, then, in the long run, a positive stock of TB infections cannot be sustained.

However, this is only half the story, since a country is prosperous partly because it has a low incidence of TB and, hence, a healthy work force, i.e. there are feedback effects from TB to the level of prosperity. The latter is influenced by capital and healthy labour inputs, the savings ratio, and the productivity of technology. We combine the Lotka–Volterra type model with the one-sector Solow–Swan (1956) economic growth model. To analyse the economic–epidemiological interaction between the economic system and the disease we use estimated logistic type functions. Moreover, under the assumption that only healthy susceptible individuals are productive, variations in labour force participation are assumed to be a function of TB prevalence (defined here to be y/x). The savings rate and the productivity of technology are taken to be exogenously determined.

We find empirically that there are qualitative differences in behaviour between the most prosperous quartile and the remaining countries. Hence, we calibrate the production side of the economy, choosing common savings and capital depreciation rates and setting the productivity of technology (which is represented by a CES production function with capital and labour inputs) at levels that generate the GDP per healthy worker of respectively the richest quartile and of the remaining three quartiles.

Our results indicate that, in the lowest three quartiles, after a possible initial period of strong movements in capital stock and a relatively stable

population structure, the system exhibits damped cycles in capital, TB incidence, and the number of healthy susceptibles. Through time, the amplitude of these cycles falls and asymptotically the system converges to a stationary state in which there is a positive but relatively low level of TB prevalence (about 2 per cent of the population are infected/infectious). In this stationary state, total population is held down below the carrying capacity of the environment (at about 80 per cent of capacity). The two-dimensional projection of this 3-D phase space into the healthy susceptible and infected/infectious plane replicates the pattern that we observe when economic prosperity is taken to be exogenously fixed at the levels of the lower three quartiles. There are other stationary states. In particular, one where there is no disease and the healthy susceptible population is at the carrying capacity. However, these other stationary states are unstable.

In the richest quartile the pattern is quite different. The TB infection monotonely dies out in the early stages and there is balanced growth in capital and labour towards a stationary state in which the total population (consisting only of healthy susceptibles) is at the carrying capacity of the environment. There is no stationary state with positive TB incidence and positive capital stock.

The way in which the two types of pattern differ is in the productivity of the technology. In each case we use common values for savings and depreciation rates and the same estimated logistic type functions explaining the demographic–epidemiological parameters in each quartile. In fact variations in the saving or depreciation rates would not generate the quantitative differences in prosperity that we observe between the highest and lowest quartiles. The productivity differences yield prosperity differences, which then work through the logistic type functions to determine the demographic–epidemiological parameters of each quartile. This leads to a positive TB reproduction coefficient in the three lowest quartiles, but a negative value in the richest quartile. Variations in the parameter governing the productivity of technology lead to a bifurcation of the system. As productivity rises suddenly, the stationary state with positive TB and capital stock, which was the limit of almost all time paths, disappears. In the long run, so does TB. The key policy implication is to raise productivity in the lower quartiles through technology transfer or through education. Of course, any other policy that serves to reduce the reproductive coefficient also helps.

The plan of the paper is to examine the data on the demographic–epidemiological parameters and their links to prosperity in section 2. In section 3 we introduce the Lotka–Volterra type model and examine its dynamic properties for parameter values corresponding to those outlined in section 2. The economic model and its interaction with the Lotka–Volterra type model is analysed in section 4. In section 5 we highlight the policy implications of our results. Section 6 concludes.

2. Empirical links between TB epidemiology and economic prosperity

TB spreads by contact between a healthy susceptible and an infected/infectious individual. Having passed on one infection, there is still the same chance of an infected/infectious individual infecting the next healthy

Table 1. *World mean values of demographic–epidemiological parameters*

α	β	ω	ρ
0.0156	0.0012	0.220	0.742

Table 2. *Correlations of GDP/x with α, β, ω, and ρ (** = significant at the 1 per cent level; * = significant at the 5 per cent level)*

α	β	ω	ρ
-0.593^{**}	-0.178^{*}	-0.462	0.073

susceptible she meets. A TB infected/infectious individual can either recover from the disease or die from the disease (or, of course, from non-TB related causes). Those who recover from the disease do not have immunity to further infections. It follows that key factors in the development or control of TB are the force of infection (the chance of transmission of the disease in any meeting between a healthy susceptible and an infected/infectious person), the number of meetings between healthy susceptibles and the infected/infectious individuals, the mortality and recovery rates. The meetings between susceptibles and infected/infectious individuals partly depend on the relative numbers of people in each group, so that, in dynamic terms, the net growth rate of susceptibles is important.

We use cross-section data on 120 countries for the year 2000 from the World Bank and World Health Organization.

In table 1 the net annual percentage growth rate of susceptibles is represented by α. We define the force of infection, β, by using the basic idea that meetings between susceptibles and the infected/infectious individuals are proportional to the product of the two so that

$$\beta = \frac{\text{TB incidence}}{xy} \tag{1}$$

Moreover, ω is the percentage total mortality rate (from TB and other causes) and ρ is the percentage rate of recovery of TB infected/infectious individuals.

An indication of the variation of the demographic–epidemiological parameters with economic prosperity is given by their correlations with the gross domestic product per susceptible in \$00s (GDP/x), as shown in table 2.

Another way of exploring the links with economic prosperity is by looking at the descriptive statistics of the demographic–epidemiological parameters by groups of 30 countries. Each group corresponds to a quartile of the distribution of GDP/x. The mean values of GDP/x for the four quartiles (Ql, Q2, Q3, and Q4) are shown in table 3, whilst the mean values of the demographic–epidemiological parameters by quartile are shown in table 4.

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

Table 3. *Mean values by quartile of GDP/x*

Q1	*Q2*	*Q3*	*Q4*
$9.96	$23.07	$53.23	$163.24

Table 4. *Mean values by quartiles of α, β, ω, and ρ*

Quartile	α	β	ω	ρ
Ql	0.0237	0.0015	0.325	0.696
Q2	0.0177	0.0097	0.2355	0.7593
Q3	0.0132	0.0014	0.165	0.763
Q4	0.0076	0.00051	0.154	0.749

Table 5. *Correlations of GDP/x with α, β, ω, and ρ by quartile (** = significant at the 1 per cent level; * = significant at the 5 per cent level)*

	α	β	ω	ρ
Ql	−0.285	−0.055	−0.153	0.333
Q2	−0.132	−0.157	−0.481*	0.195
Q3	−0.520*	0.085	0.022	−0.169
Q4	−0.417*	−0.300	−0.388*	0.103

There are systematic differences in most of the demographic–epidemiological parameters between quartiles. However, there is also some intra-quartile variation (see table 5). Moreover, the parameters are intrinsically bounded, e.g. mortality and recovery rates must be in the unit interval, so we would expect the variation of the parameters with GDP/x to be non-linear.

Indeed plotting GDP/x against α, β, ω, and ρ, respectively, we find evidence of a logistic type of shape with clear upper and lower bounds on most variables (see figure 1).

Using the data on all the 120 countries, we apply non-linear least squares to estimate the relation between the parameters and economic prosperity.

For the net growth population rate, we try two models: firstly with the carrying capacity effect

$$\alpha = [\alpha_0 + \alpha_1 \exp(-\alpha_2 GDP/x/\alpha)](1 - \alpha_3 x) + \varepsilon\alpha \tag{2}$$

and then without it (i.e. imposing $\alpha_3 = 0$). In terms of the goodness of fit and the remaining coefficient estimates, there is very little difference.

As for β, there is evidence of heteroscedasticity if we use an additive error, so we run the regression

$$\ln(\beta) = \ln[\beta_0 + \beta_1 \exp(-\beta_2 GDP/x)] + \varepsilon_\beta \tag{3}$$

For ω and ρ, we estimate, respectively

$$\omega = [w_0 + w_1 \exp(-w_2 GDP/x)] + \varepsilon_\omega \tag{4}$$

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

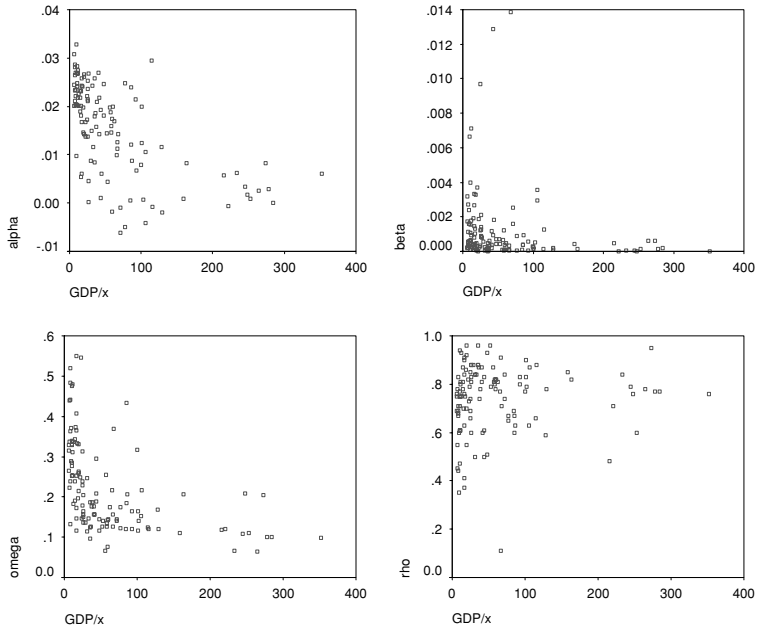

Figure 1. *Plots of GDP/x against* α, β, ω, *and* ρ, *respectively*

Table 6. *NLSQ estimates for the net growth population rate with* $\alpha_3 \neq 0$

	Estimate	*SE*
α_0	0.0033	0.0025
α_1	0.0228	0.0025
α_2	0.0147	0.0044
α_3	0.0004	0.0004
R^2	0.45656	
RSS	5.045*E* – 05	

and

$$\rho = [\rho_0 + \rho_1 \exp(-\rho_2 GDP/x)] + \varepsilon_\rho \tag{5}$$

The results are shown in tables 6 and 7.

When included in the equation for the net growth rate, the carrying capacity (λ) is insignificant and its estimated value is high (i.e. about 45 times the mean current population level).

From table 7 the results indicate that the poorest countries (if $GDP/x = 0$) have a net population growth rate of about 2.5 per cent, whilst the richest ($GDP/x \to \infty$) have a value of 0.03 per cent, which, although very small, is still positive. The significance of α_2 indicates the significance of variations in GDP/x.

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

Table 7. *NLSQ estimates for* α, β, ω, *and* ρ

	Estimate	*SE*		*Estimate*	*SE*
α_0	0.0034	0.0024	β_0	−0.0002	0.0002
α_1	0.0225	0.0023	β_1	0.0008	0.0002
α_2	0.0152	0.0045	β_2	0.0046	0.0031
R^2	0.45091		R^2	0.165	
RSS	5.053*E* – 05		*RSS*	2.133	
w_0	0.1486	0.0142	ρ_0	0.7625	0.0171
w_1	0.3082	0.0512	ρ_1	−0.3668	0.4571
w_2	0.0565	0.0158	ρ_2	0.1721	0.1532
R^2	0.41870		R^2	0.0548	
RSS	7.344*E* – 03		*RSS*	0.0202	

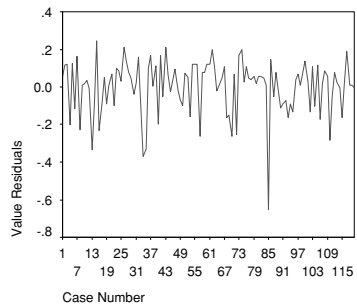

Figure 2. *Plot of residuals* ρ

For the force of infection, β, the richest countries effectively have no infection, the poorest countries do (β_1 is significant). Otherwise, GDP/x plays a relatively small role in fluctuations in β.

The total death rate for TB infected/infectious cases, ω, is three times higher in the poorest than the richest countries. However, even in the latter, it is about 15 per cent. For all countries GDP/x has a significant effect on ω.

The recovery rate, ρ, is twice as high in the richest than in the poorest countries, but even in the latter there is about a 40 per cent chance of recovery. Prosperity is marginally significant in determining recovery and the goodness of fit is rather poor, which appears to be due to three outlying countries. In figure 2 we plot the residuals for ρ, with the countries arranged by ascending GDP/x. There are three outliers: Brazil, Central African Republic, and Senegal, all of which have large negative residuals.

The estimated demographic–epidemiological parameters by quartile, evaluated at the mean of GDP/x in each quartile, are shown in table 8.

Comparing tables 4 and 8 indicates that the econometric estimates track the means of the demographic–epidemiological parameters in each quartile very closely.

The conclusion is that empirically there is a link between TB epidemiology and economic prosperity. Moreover, the non-linear sigmoid type of relationship captures the link quite well.

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

Table 8. *NLSQ estimates for the demographic–epidemiological parameters by quartile*

Quartile	α	β	ρ	ω
Ql	0.021	0.00061	0.696	0.325
Q2	0.016	0.00057	0.755	0.234
Q3	0.011	0.00048	0.762	0.165
Q4	0.0047	0.00023	0.762	0.149

The next step is to analyse the effect of prosperity on the dynamics of TB epidemiology and the feedback effects between the dynamic health structure of the population and economic prosperity.

3. The demographic–epidemiological model

The study of epidemics has a long history, with a vast variety of models and explanations for the spread and cause of epidemic outbreaks. As a benchmark for assessing the importance of economic effects on epidemics, we use a pure demographic–epidemiological model. Referring to the classical predator–prey Lotka–Volterra type model, the population dynamics of infectious diseases is represented as a 'predatory organism' searching for 'human prey' to consume.

We consider a homogeneously mixing population of $N(t)$ individuals in a given area at time t ($t \geq 0$), which is divided into two classes, according to their health status: healthy susceptible ($x(t)$) and TB infected/infectious *(y(t))* individuals.

$$\begin{cases} \dot{x}(t) = \alpha x(t)\left(1 - \dfrac{x(t)}{\lambda}\right) - \beta x(t)y(t) + \rho y(t) \\ \dot{y}(t) = \beta x(t)y(t) - (\rho + \omega)y(t) \end{cases} \tag{6}$$

According to (6) a susceptible/healthy individual can either stay susceptible/healthy or move out of this class to join the TB infected/infectious one. An infected/infectious individual can change status either through recovery from the disease (in this case she rejoins the susceptible/healthy class) or through death from any cause.

Here α, β, ω, and ρ are the demographic–epidemiological parameters as defined in the previous section.

The area in question has an upper bound to the population that can be sustained, so that net population growth tails off as $x(t)$ approaches λ, the carrying capacity of the area.

The number of new infections depends on the number of susceptibles, the number of infected/infectious, and the probability of transmission, β. As the latter is critically determined by the frequency and duration of an individual's contact with an infected/infectious person, the density dependent effect is particularly important in the dynamic analysis of the spread of TB. In more densely populated areas, the number of meetings between people is higher than in lower density areas. A standard approach in mathematical epidemiology (Hamer, 1906; Kermarck and McKendrick,

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

1927; De Palma and Lefevre, 1988) is to consider the probability that a susceptible individual meets a TB infected/infectious individual, given that a meeting occurs, to be $y(t)/N(t)$ (the proportion of TB infected/infectious individuals in a population with size $N(t)$). The meeting between susceptible and infected/infectious individuals leads to the disease transmission with probability that we take to be proportional to the population size, because of the density effect. It follows that the force of infection is given by

$$\beta N(t)\frac{y(t)}{N(t)}x(t) = \beta x(t)y(t) \tag{7}$$

Typically the parameters α, β, ρ, and ω are functions of the economic level of prosperity of the area. From table 2 we know the force of infection, the TB inclusive mortality rate, and the net growth population rate of susceptibles to fall with higher economic development, whilst the recovery rate rises.

We start by examining the behaviour of this dynamic population system at a given level of economic prosperity. The system potentially has three stationary states at

$$(x_1^* = y_l^* = 0) \tag{8}$$

$$(x_2^* = \lambda,\ y_2^* = 0) \tag{9}$$

$$\left(x_3^* = \frac{\rho+\omega}{\beta},\ y_3^* = \frac{\alpha(\rho+\omega)(\beta-(\rho+\omega)/\lambda)}{\omega\beta^2}\right) \tag{10}$$

The sign of $\beta\lambda - \rho - \omega$ is critical both in determining the number of stationary states and their dynamic properties. This can be thought of as the basic reproductive rate of the disease, which, if positive, allows epidemics to arise.

In the first stationary state (8) the population has become extinct.

The second stationary state (9) has no disease and the class of susceptibles is constant at the carrying capacity of the system.

The third stationary state (10) is infeasible if $y_3^* < 0$, i.e. if $\beta < (\rho+\omega)/\lambda$. This can occur either if the force of infection is low relative to the mortality and recovery rate of the infected/infectious individuals (i.e. there is not enough time for the disease to spread) or if the carrying capacity of the environment is relatively low, so that the net population growth rate of susceptibles is insufficient to maintain a pool of 'prey' who can still be infected.

The dynamics of the system can be analysed in two ways: local stability analysis around each stationary state to examine small movements and global simulation to investigate how these local dynamics join up.

From the linearization, we know that the first stationary state is characterized by a saddle point with eigenvalues

$$\left[\eta_1^1 = \alpha,\ \eta_2^1 = -(\rho+\omega)\right] \tag{11}$$

The system may be driven to extinction, but only along the unique stable seperatrix and only as time tends to infinity. Moreover, the second stationary state has an unstable nature only when the third stationary state exists and

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

is a point of attraction. Here, the eigenvalues are

$$\left[\eta_1^2 = -\alpha,\ \eta_2^2 = (\beta\lambda - \rho - \omega)\right] \tag{12}$$

If the third stationary state fails to exists or is unstable, then the second stationary state becomes unstable. When the third stationary exists, it is locally stable, possibly with complex conjugate roots.

3.1. Calibration

From the data we can compute the means of the demographic–epidemiological parameters either across all countries or by quartiles. We can use these values to see how TB either develops or becomes unimportant in countries with particular constant economic prosperity. First, we consider a country with parameter values equal to the overall means of the 120 countries. We think of this as exploring TB dynamics for a representative country with average values of each parameter. Second, we take the mean values of each quartile and repeat the analysis. For instance, this allows us to see the differences in the dynamic epidemiology of TB between a country in the middle of the poorest quartile and a country in the middle of the richest quartile.

3.1.1. World values

Given values of β, ω, and ρ, we know that there is a critical level of λ below which there is no stationary state with disease. Moreover, this critical level separates different dynamic patterns. If we use constant parameter values corresponding to the overall sample means, as in table 1, the critical value of the carrying capacity is $\lambda \cong 817$ (expressed in millions). This compares with a mean population of susceptibles of 20 and a mean population of infected/infectious individuals of 15. To illustrate the differences in behaviour, we have taken two possible values of λ above and below the critical value of λ.

For $\lambda = 100$, there are only two stationary states, both without infection ($y_1^* = 0$ and $y_2^* = 0$). The first has $x_1^* = 0$ and the second has $x_2^* = \lambda$. The eigenvalues around the first equilibrium are

$$\left[\eta_1^1 = 0.0156,\ \eta_2^1 = -0.962\right] \tag{13}$$

and around the second they are

$$\left[\eta_1^2 = -0.0156,\ \eta_2^2 = -0.842\right] \tag{14}$$

Here the origin is a saddle point with the stable seperatrix being vertical. With these parameters, extinction is only possible if initially there are no susceptibles (i.e. $x(0) = 0$). Some of the initial infected/infectious individuals may recover but not in numbers that allow the susceptible/healthy population to take off. However, starting with positive numbers of susceptible/healthy individuals, the infection dies out.

For $\lambda = 1000$, we have three stationary states. The additional third stationary state is at

$$(x_3^* = 802,\ y_3^* = 11) \tag{15}$$

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

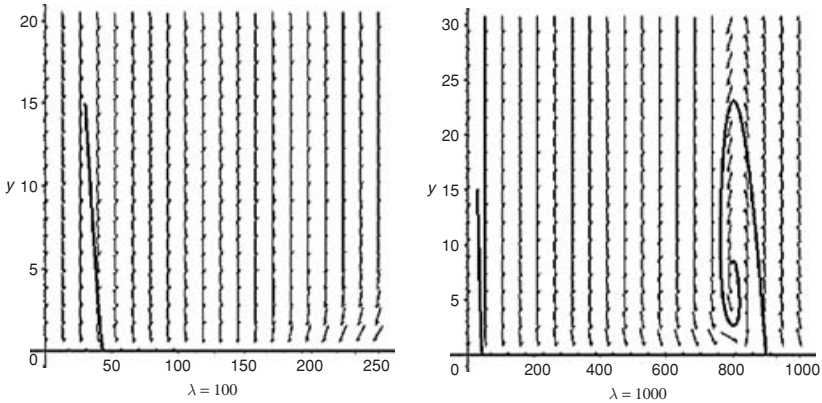

Figure 3. *Phase space diagrams*

The eigenvalues corresponding to this equilibrium are

$$\left[\eta^3_{1,2} = -0.0115 \pm 0.0533i\right] \tag{16}$$

For the equilibrium with no infections the eigenvalues are

$$\left[\eta^1_1 = 0.0156,\ \eta^1_2 = -0.962\right] \tag{17}$$

whilst at the equilibrium with $x^*_2 = \lambda$ the eigenvalues are

$$\left[\eta^2_1 = -0.0156,\ \eta^2_2 = 0.238\right] \tag{18}$$

Both the disease-free equilibria are saddle points, whilst the third equilibrium is locally stable. The stable direction at the carrying capacity equilibrium is horizontal so that this equilibrium is only reached (asymptotically) if initially there are no infected/infectious individuals (i.e. $y(0) = 0$).

The phase spaces with a path having the same initial conditions as the mean values of x and y is shown in figure 3.

3.1.2. Quartiles of economic prosperity values

Using the values of the demographic–epidemiological parameters from table 8 and the mean estimated value of the carrying capacity $\lambda = 2,257$, the calibration results for the poorest quartile indicate that the stationary states are at

$$(x^*_1 = y^*_1 = 0) \tag{19}$$

$$(x^*_2 = 2,257,\ y^*_2 = 0) \tag{20}$$

$$(x^*_3 = 1,663,\ y^*_3 = 31) \tag{21}$$

The dynamics around these stationary states suggest that, for any initial conditions with $y(0) > 0$, the system converges to a positive level of TB at $y^*_3 = 31$, with healthy susceptibles being contained below the capacity of the environment (i.e. $x^*_3 = 1,663 < \lambda = 2,257$).

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

Similar patterns occur in the next two quartiles.
For the second quartile we have stationary states at

$$(x_1^* = y_1^* = 0) \tag{22}$$

$$(x_2^* = 2,257,\ y_2^* = 0) \tag{23}$$

$$(x_3^* = 1,736,\ y_3^* = 33) \tag{24}$$

For the third quartile we have stationary states at

$$(x_1^* = y_1^* = 0) \tag{25}$$

$$(x_2^* = 2,257,\ y_2^* = 0) \tag{26}$$

$$(x_3^* = 1,946,\ y_3^* = 22) \tag{27}$$

However, the quartile with the highest values for GDP/x has quite different behaviour. Here the viable stationary states are

$$(x_1^* = y_1^* = 0) \tag{28}$$

$$(x_2^* = 2,257,\ y_2^* = 0) \tag{29}$$

So long as the system starts with $x(0) > 0$, it converges to a stationary state, where the disease is eradicated and with the number of healthy susceptible individuals set at the carrying capacity of the environment.

The dynamic diagrams are drawn for the same time horizon. Moreover, in each diagram the indicated paths have matching initial conditions. As indicated in figure 4, as economic prosperity rises, the ultimate prevalence of the disease stays roughly constant at 0.018 in the bottom two quartiles and falls in the third quartile to 0.011. The long-run susceptible population rises through the quartiles. In the third quartile, the number of TB infections attains a maximum at about 100, which corresponds to around half its peak in the first two quartiles. Also the speed of movement in the second quartile appears to be faster than in the first or third quartiles.

In the richest quartile, TB monotonically falls and is eliminated in finite time. Thereafter, the susceptibles just grow to the carrying capacity of the environment.

4. The economic model

In the economy there is an aggregate production function yielding a unique good that can be either consumed ($C(t)$) or invested ($\dot{k}(t)$)

$$F(x(t), k(t)) = C(t) + \dot{k}(t) \tag{30}$$

Inputs into production are provided by the healthy/susceptible individuals ($x(t)$) and by homogenous capital ($k(t)$).

The production function is assumed to satisfy neoclassical properties:

- $F(x(t), k(t))$ exhibits positive and decreasing returns to input ($F_x, F_k > 0$ and $F_{xx}, F_{kk} < 0$).

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

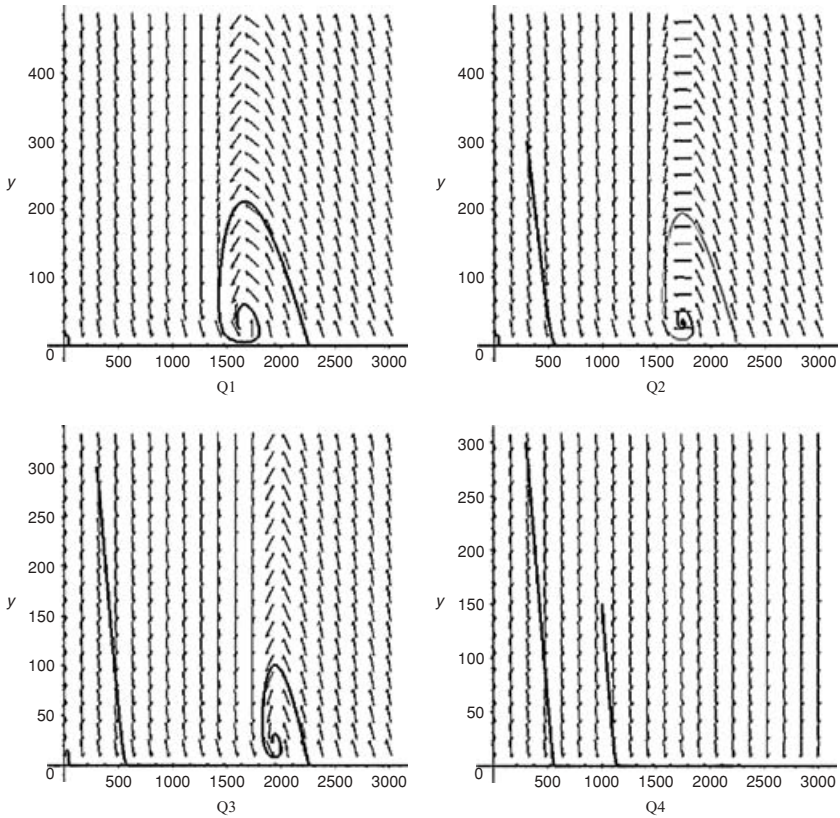

Figure 4. *Phase space diagrams*

- $F(x(t), k(t))$ is homogenous of degree one.
- Each input is essential in that $F(x(t), 0) = F(0, k(t)) = F(0, 0) = 0$.

Later, in a parametric example, we use a CES function with elasticity of substitution less than one.

The savings function is assumed to determine the composition of demand as between output to be consumed and output to be set aside for accumulation. Specifically, a fraction, s, of total output flow $(F(x(t), k(t)))$ is assumed to be saved and set aside to be added to the capital stock. This fraction of output devoted to investment is exogenous, positive, and constant. Moreover, existing capital is assumed to depreciate at rate ϕ with $\phi \in [0, 1]$.[2]

[2] In a decentralized system, where capital is equally owned by each individual, either susceptible or TB infected/infectious, we can think of

$$I_x = \psi x + r k_x \tag{f1}$$

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

The growth of the capital stock is given by the equation

$$\dot{k}(t) = sF(x(t),\ k(t)) - \phi k(t) \tag{31}$$

with $k(0) > 0$.

The demographic–epidemiological variables are endogenous to the economy, implying the net growth population rate (α), the TB transmission coefficient (β), the total death rate of the infected/infectious individuals (ω), and the recovery rate (ρ) are functions of economic variables. As only susceptible individuals are assumed to enter the labour force, output per healthy worker, $z(t)$, is chosen here as a measure for economic prosperity.

$$z(t) = \frac{F(x(t),\ k(t))}{x(t)} \tag{32}$$

To higher levels of z correspond lower levels of the TB transmission coefficient ($\beta'(z) < 0$) and the total death rate of the TB infected/infectious individuals ($\omega'(z) < 0$) and a higher level of the recovery rate ($\rho'(z) > 0$) (Adler *et al.*, 1993; Cigno, 1998; Ettner, 1996; Feinstein, 1993; Hadley and Osei, 1982; Haggerty and Johnson, 1996; Kitagawa and Hauser, 1973; Orcutt Duleep, 1985).

Higher output per healthy worker represents, here, improvements in general standards of living and working conditions, diet, education, availability and access to health services, which reduce both the TB transmission coefficient and the TB mortality rate of the TB infected/infectious individuals. Table 2 shows that the net growth population rate is always falling with GDP per healthy individual. So we also take $\alpha'(z) < 0$.

The two-way interactions between demographic–epidemiological factors and economic variables is described by an autonomous system in $\Re^3$ of three

and

$$I_y = rk_y \tag{f2}$$

being the income of the susceptible and TB infected/infectious individuals, respectively, with $k_x = \frac{x}{x+y}k$ and $k_y = \frac{y}{x+y}k$. There is a competitive rental market for capital with rental price r and competitive labour market wage ψ, all measured in units of output. The susceptible and TB infected/infectious individuals have the propensity to save out of their income of s_x and s_y, respectively. The total savings is

$$s = s_x I_x + s_y I_y \tag{f3}$$

By assuming

$$s_x = s_y = s \tag{f4}$$

then

$$s = s(I_x + I_y) = s(\psi x + rk_x + rk_y) = sF(x(t), k(t)) \tag{f5}$$

and we are back to the central planned model.

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

non-linear differential equations

$$\begin{cases} \dot{x}(t) = \alpha(z(t))x(t)\left[1 - \dfrac{x(t)}{\lambda}\right] - \beta(z(t))x(t)y(t) + \rho(z(t))y(t) \\ \dot{y}(t) = \beta(z(t))x(t)y(t) - \omega(z(t))y(t) - \rho(z(t))y(t) \\ \dot{k}(t) = sF(x(t), k(t)) - \phi k(t) \end{cases} \tag{33}$$

where, because of the assumption of constant returns to scale

$$z = \frac{F(x(t), k(t))}{k(t)} = F\left(\frac{x(t)}{k(t)}, 1\right) = f\left(\frac{x(t)}{k(t)}\right) \tag{34}$$

The system in (33), in the absence of capital ($k = 0$), reduces to the original predator–prey Lotka–Volterra type model. For $\beta = \omega = \rho = 0$ or $y = 0$, the system in (33) reduces to a version of the one-sector Solow–Swan model (1956). Also, if λ is arbitrarily large, the Solow–Swan model has a constant percentage net growth of susceptibles. Increases in economic prosperity improve the disease epidemiology through a reduced infection risk and an increased recovery rate. Moreover, since it reduces the net growth of susceptibles, improved economic prosperity also reduces the population at risk of infection. A countervailing force is that improved economic prosperity reduces the mortality of the infected/infectious, giving them a longer time in which to spread the infection. The dynamic forces at work include a rise in prosperity leading to reduced net population growth and initially a rise in the prevalence of the disease. This leads to an increase in the number of infected/infectious cases and a further worsening of TB prevalence. However, the fall in the workforce caused both by a fall in net population growth and a rise in infections in turn reduces prosperity. So there is scope for cycles arising out of the economic–epidemiological interaction.

To analyse the stability properties of the system (33), we find the stationary states by simultaneously solving the static equations $\dot{x}(t) = 0, \dot{y}(t) = 0$ and $\dot{k}(t) = 0$. As the demographic–epidemiological factors are constant when $x(t)$ and $k(t)$ are, there are four stationary states

$$(x_1^* = y_1^* = k_1^* = 0) \tag{35}$$

$$(x_2^* = \lambda,\ y_2^* = 0,\ k_2^* = 0) \tag{36}$$

$$\left(x_3^* = \lambda,\ y_3^* = 0,\ k_3^* = \left[f^{-1}\left(\frac{\phi}{s}\right)\right]^{-1}\right) \tag{37}$$

$$\left(x_4^* = \frac{\rho + \omega}{\beta},\ y_4^* = \frac{\alpha(\rho + \omega)(\beta - (\rho + \omega)/\lambda)}{\omega\beta^2},\ \left(\frac{k}{x}\right)_4^* = \left[f^{-1}\left(\frac{\phi}{s}\right)\right]^{-1}\right) \tag{38}$$

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

Here, $f^{-1}(\phi/s)$ is the inverse function[3] of f. It is easy to see that in the system (33) there is no balanced path along which $x(t)$, $y(t)$ and $k(t)$ each grow at a constant rate,[4] φ, even if λ is arbitrarily large.

However, there is a partially balanced growth path with $y \equiv 0$ and $z = [f^{-1}((\alpha + \phi)/s)]^{-1}$, where $z(t) = k(t)/x(t)$. Along this path, the growth rate of $x(t)$ and $k(t)$ is $\alpha(z(t))$ and there is no disease in the system. For any initial condition on this path, the disease cannot break out. For initial conditions starting away from this path, the system approaches this disease-free balanced growth time profile if $\dot{y}(t)/y(t)$ eventually becomes and stays negative. However, as $y(t) \to 0$, $\dot{x}(t)/x(t) \to \alpha(1 - x(t)/\lambda)$, which ultimately tends to zero with $x(t) \to \lambda$, as $\dot{y}(t)/y(t) = (\beta x(t) - \omega - \rho)$, where β, ω, and ρ are finite and positive. Thus, $\dot{y}(t)/y(t)$ can become and remain negative, if the reproductive rate, $\beta\lambda - \omega - \rho$, becomes and remains negative. If capital accumulation pushes the reproductive rate below zero, then rising economic prosperity leads to the elimination of the disease.

Next we focus on how the interaction of the economic growth system with the demographic–epidemiological process controls the dynamics of the population structure and economic prosperity.

First we look at the stationary states. Overall, the first stationary state corresponds to extinction. The second is characterized by a stationary healthy population, without any capital stock and consequently with zero output. The third corresponds to the state of an economic growth model, with healthy susceptibles at the capacity level of the environment. Only the fourth stationary state has presence of the disease and it is only viable if $y_1^* > 0$, requiring the familiar condition $\beta > (\rho + \omega)/\lambda$. If this is so, then the prevalence of the disease is given by $[\alpha(\beta - (\rho + \omega)/\lambda)]/[\omega\beta]$, which is increasing in α, β, and λ, but decreasing in ρ and ω.

4.1. Calibration of production

As a production function we take a CES with elasticity of substitution less than unity

$$F(x(t), k(t)) = g[vx(t)^m + rk(t)^m]^{\frac{1}{m}} \tag{39}$$

where $m < 0$ and $v, r > 0$. With these choices we have

$$F_x = gv\left[v + r\left(\frac{k(t)}{x(t)}\right)^m\right]^{\frac{(1-m)}{m}} \tag{40}$$

[3] From the third equation of (33) we know that

$$sF(x(t), k(t)) = \phi k(t) \tag{f6}$$

from which we have $sF(x(t)/k(t), 1) = \phi$ or $f(x(t)/k(t)) = \phi/s$ so that $x(t)/k(t) = f^{-1}(\phi/s)$ or $k(t)/x(t) = [f^{-1}(\phi/s)]^{-1}$.

[4] If there were a balanced growth path, φ, then (33) would require

$$\begin{cases} \varphi = \alpha - \beta y(t) \\ \varphi = \beta x(t) - \omega \end{cases} \tag{f7}$$

where along the balanced growth path α, β, ω, and ρ would be constants. However, setting $y(t) = y(0)e^{\varphi t}$, the only value of φ that satisfies (33) for all t is $\varphi = 0$.

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

$$\frac{F}{x} = g\left[v + r\left(\frac{k(t)}{x(t)}\right)^{m}\right]^{\frac{1}{m}} \tag{41}$$

$$F_k = gr\left[v\left(\frac{x(t)}{k(t)}\right)^{m} + r\right]^{\frac{(1-m)}{m}} \tag{42}$$

In this case the stationary states are at, respectively

$$(x_1^* = y_1^* = k_1^* = 0) \tag{43}$$

$$\left(x_2^* = \frac{\omega}{\beta},\ y_2^* = \frac{\alpha}{\beta},\ k_2^* = 0\right) \tag{44}$$

$$\left(x_3^* = \frac{\omega}{\beta},\ y_3^* = \frac{\alpha}{\beta},\left(\frac{k}{x}\right)_3^* = \left(\frac{\left(\frac{\phi}{sg}\right)^{m} - r}{v}\right)^{-\frac{1}{m}}\right) \tag{45}$$

Empirically, there is wide variation between countries in GDP/x: the highest quartile has GDP per healthy worker 16 times higher than that of the lowest quartile. This could be due to differences in initial factor endowments, differences in productivity or savings rates, or differences in depreciation rates. The labour endowments do not systematically differ by quartile, the composition of capital may differ by quartile, but variation in the depreciation or savings rates cannot generate such a substantial gap in GDP/x. Undoubtedly, initial capital stocks, as at present, differ substantially, but we have no data on this and anyway this is not really an explanation for a long-run gap, since the current capital must have been accumulated from the past. Thus, we identify the main determinant of the gap as productivity differences between quartiles and calibrate the scale factor g to values that generate approximately the quartile mean GDP/x in the TB-free stationary state, when susceptibles have attained the carrying capacity. Since the main difference is between the richest quartile and the others, we take two different values for g ($g = 25$ and $g = 75$). The other parameters are taken to have common values. Given that the depreciation rate of capital, ϕ, represents a weighted average of the depreciation rate of the different types of assets in the capital stock (i.e. buildings, machineries, vehicles), we set $\phi = 0.15$. The parameter values for v and r are determined by the stylized fact that the share of wages is about 70 per cent, which gives $r = 0.3$, $v = 0.7$, and $m = -1$, so that the elasticity of substitution is 0.5. The saving rate, s, is set at the average value of 10 per cent. Finally, we use the econometrically estimated functions for α, β, ρ, and ω and the mean estimate of the carrying capacity $\lambda = 2,257$.

For $g = 25$, the system has stationary states at

$$(x_1^* = 2,257,\ y_1^* = 0,\ k_1^* = 0) \tag{46}$$

$$(x_2^* = 1,311,\ y_2^* = 31,\ k_2^* = 0) \tag{47}$$

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

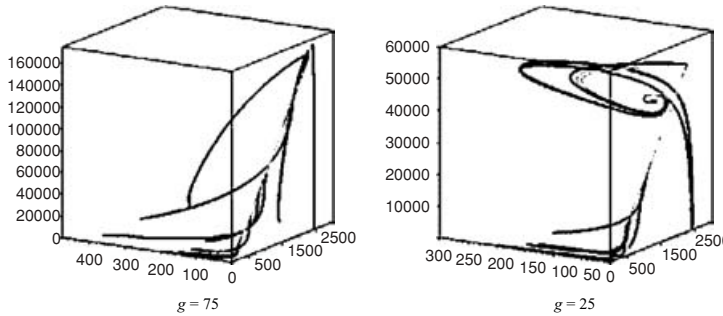

Figure 5. *Phase space diagrams*

$$(x_3^* = 2,257,\ y_3^* = 0,\ k_3^* = 52,777.824) \tag{48}$$

$$(x_4^* = 1,796,\ y_4^* = 32,\ k_4^* = 41,992.004) \tag{49}$$

At the third stationary state $GDP/x = 34.9$, which compares with the means of the two middle quartiles of \$23.07 and \$53.23 respectively. Around these the respective eigenvalues are

$$\left[\eta_1^1 = 0,\ \eta_2^1 = 0.6152,\ \eta_3^1 = 8.333\right] \tag{50}$$

$$\left[\eta_{1,2}^2 = -0.0143 \pm 0.0955i,\ \eta_3^2 = 8.333\right] \tag{51}$$

$$\left[\eta_1^3 = 0.0027,\ \eta_2^3 = 0,\ \eta_3^3 = 0.245\right] \tag{52}$$

$$\left[\eta_{1,2}^4 = -0.0111 \pm 0.056,\ \eta_3^4 = 0.003\right] \tag{53}$$

However, for $g = 75$, the system has stationary states at

$$(x_1^* = 2,257, y_1^* = 0,\ k_1^* = 0) \tag{54}$$

$$(x_2^* = 1,311, y_2^* = 31,\ k_2^* = 0) \tag{55}$$

$$(x_3^* = 2,257, y_3^* = 0,\ k_3^* = 160,268.30) \tag{56}$$

At the third stationary state $GDP/x = 106.5$, which compares with the mean of the top quartile of \$163.24.

Around these stationary states the eigenvalues are, respectively

$$\left[\eta_1^1 = 0,\ \eta_2^1 = 0.6152, \eta_3^1 = 25.0\right] \tag{57}$$

$$\left[\eta_{1,2}^2 = -0.0143 \pm 0.0155i,\ \eta_3^2 = 25.0\right] \tag{58}$$

$$\left[\eta_1^3 = 0.0009,\ \eta_2^3 = 0,\ \eta_3^3 = -0.144\right] \tag{59}$$

The dynamics of the system are shown in figure 5.

When $g = 75$, starting from an initial position with TB and where susceptibles are below the carrying capacity, the system moves on to a

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

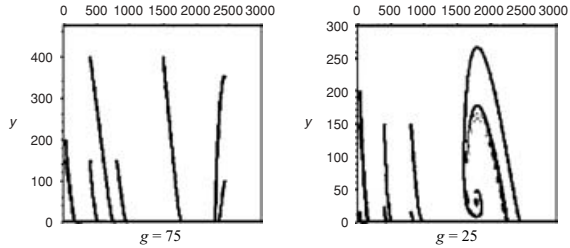

Figure 6. *Projections of phase space in $x - y$ plane*

path in which x and k grow monotonely until the healthy/susceptible population reaches the carrying capacity. Along this type of path the number of infected/infectious individuals falls monotonely to zero. However, if starting with susceptibles above the carrying capacity (mathematically possible but economically difficult to interpret!), the susceptibles fall to the carrying capacity, capital grows, and, although initially there may only be a slow reduction in the stock of infected/infectious, this then falls to zero again.

When $g = 25$, the global dynamics indicates that for any initial conditions with $y(0) > 0$ the system converges to the fourth stationary state. This is interesting since, locally, the second and fourth stationary states have a common pattern to the signs of the eigenvalues. However, the positive real root of the second solution is much higher than that of the fourth, which appears to dominate the dynamics.

The varying dynamic behaviour along different paths is of interest. Starting with a low but positive level of capital and a number of susceptibles below the fourth stationary state level, there is rapid decline of the disease. As this approaches zero, capital and the healthy population start growing towards the second stationary state, in which population is at the carrying capacity. However, its instability leads to cyclical convergence to the fourth stationary state. In the damped cycles, there is positive correlation between x and k but only weak correlation between y and x. A similar experience unfolds starting with abundant susceptibles but low levels of capital stock and TB infection. In this case, there is rapid growth of capital with near constant population and population structure. Here, the marginal product of capital is sufficiently high to allow growth in capital stock. Then, the number of infections rises and the system enters the same type of epidemic cycle.

We can look at the projection of the 3-D phase diagram into the $x - y$ plane (figure 6) to compare the systems with feedback links with those in which the demographic–epidemiological parameters vary with prosperity (figure 4). We notice remarkable similarities. Hence, we expect the first three quartiles to follow the dynamic pattern of cycles around a positive TB prevalence, where there is positive correlation between the number of susceptibles and capital stock, but both these having negative correlation with the stock of TB infections. However, the richest quartile should follow

https://doi.org/10.1017/S1355770X05002500 Published online by Cambridge University Press

a path leading to eradication of the disease and along which, after an initial period, the population of susceptibles grows to the carrying capacity.

5. Policy implications

The principal present universal policy initiative, aimed at the cure of infected cases, is the DOTS programme. Raising the recovery rate is one means of reducing the reproductive rate of the disease. However, the scope for improvement is limited, since, in all quartiles, the recovery rate is around 70 per cent. We find that GDP per healthy capita is important in determining the demographic–epidemiological position of a country. In the richest countries, prosperity gives favourable conditions, allowing TB to be eliminated without any additional direct policy intervention. In poorer countries, there has to be some policy initiative to control the disease. Overall, there are also elements of a vicious circle: high TB prevalence generates feedback effects on economic growth and prosperity, since it influences the healthy labour force. A critical expression is the reproductive rate, $\beta - (\rho + \omega)/\lambda$, so that as well as the force of infection, the recovery rate and the overall mortality rate of the TB infected/infectious, and the carrying capacity of the country are important. The higher the reproductive rate is, the more likely that there are endemic TB cycles.

As prosperity differences are critical in determining the dynamic impact of TB, it is important to determine the causes of these differences and to identify policies that can raise prosperity in poorer countries. We find that the quantitative differences between countries are such that raising the savings ratio to improve the capital base is not enough. The main factor causing the gap is the difference in labour productivity. One approach would be to make substantial capital donations to the poorer countries to shift their initial conditions. However, the necessary size of the donation to put them on an equal footing with the highest quartile is enormous. The alternative is to improve technology in the poorer countries through education, technical progress, and technology transfer, so that production possibilities approach those of the highest quartile. Another major benefit of narrowing the productivity gap would be to raise the living standards of the poorer countries. In our model, with a proportional savings function, if GDP per healthy capita increases, so does consumption per capita. Of course, this is a simple aggregate type of approach. There are also great environmental and social differences between different countries, so that, for example, the range of goods produced is quite different.

The alternative to a technology-based policy to raise productivity is to target particular critical demographic–epidemiological parameters. The recovery rate, ρ, is only marginally influenced by prosperity and it is already subject to a direct targeted policy, the DOTS programme, which has raised the recovery rate to around 70 per cent in all countries. The mortality rate, ω, is three times higher in the poorest than the richest countries and is significantly affected by economic prosperity. However, some of this difference is due to variation in non-TB causes of mortality, arising from prosperity differences. There is also significant variation in the force of infection, β, which is, again, three times higher in the poorest than the richest countries, and prosperity plays a significant role in determining its value

in the poorest countries. Overall, the indirect policy of raising productivity has primary effects via the death rate and the force of infection. A direct policy to control the force of infection is isolation of the infected/infectious cases, which harks back to the nineteenth century.

6. Conclusions

Infectious diseases have long been identified as a main cause of mortality in the human population. To date, although some of these diseases have been completely eradicated or kept at an endemic level in the population, in many parts of the world the exacerbation of old infections or the emergence of new ones are having dramatic effects on the population with strong repercussions on the economic structure.

As reported by the WHO, since the early 1980s TB has been the major health problem in developing countries, with about two million cases of death each year. Amongst the factors leading to the recent TB epidemics, major causes are poverty, malnutrition, overcrowded housing, together with poor hygienic conditions associated with rapidly increasing urbanization.

Most of the research has failed to analyse the complex nature of the recent TB epidemics.

In this paper we argue that diseases such as TB are indicators of wider social, environmental, and global conditions, and they must be seen within a wider concept of health. For TB, the empirical evidence supports this. It follows that a more complex and interdisciplinary model, integrating the contributing factors for the TB epidemic, is needed.

We classify the population into two groups: healthy susceptibles and TB infected/infectious. The dynamic interaction between them is governed by a variant of the Lotka–Volterra type model that includes the net growth rate of susceptibles, the carrying capacity of the environment, the force of infection, the overall mortality rate of the infected/infectious, and the recovery rate of the infected/infectious. Each of the main demographic–epidemiological parameters varies with economic prosperity in a non-linear way. We estimate how. Using values of the parameters corresponding to prosperity quartiles, we see that, in the lowest three quartiles, there are damped epidemic cycles around a stationary state with a positive TB prevalence and at which the total population is below the carrying capacity. There is another (unstable) stationary state with zero TB prevalence and population equal to the carrying capacity. There is also a stationary state corresponding to extinction of the population. In the lowest three quartiles, for almost all initial conditions, the system has damped cyclical convergence to a stationary state with TB prevalence. However, for parameter values of the richest quartile, this stationary state disappears and the system has monotone convergence to zero TB prevalence and population set at the carrying capacity of the environment.

Next we treat prosperity as being endogenously determined by economic production, using capital and healthy susceptibles as labour input. The production function is calibrated on two sets of parameters – one yielding GDP per healthy worker of the richest quartile, the other corresponding to the three lowest quartiles. Combining a Solow–Swan growth model,

using this production function and the Lotka–Volterra-type model gives a three-dimensional dynamical system.

For the first parameter set corresponding to the richest quartile, there are three stationary states and, so long as initially there is some capital, the system converges along a path of capital and healthy susceptible growth with falling TB to a state with no disease and a population equal to the carrying capacity.

With the second parameter set, corresponding to the lowest three quartiles with lower productivity of technology, there are four stationary states. Three of these match those of the richest quartile. The new possibility has positive TB prevalence and capital stock with population below the carrying capacity. This new stationary state is the limit of almost all paths and so is generically stable. Locally around it there are damped cycles in the healthy susceptibles, the TB infected/infectious and capital stock. Productivity variations cause a bifurcation of the system. As productivity rises, the undesirable fourth stationary state disappears. It follows that raising productivity in lower quartile countries is a key policy issue.

References

Adler, N.E., T. Boyce, M. Chesney, S. Folkman, and L. Syme (1993), 'Socio-economic inequalities in health – no easy solution', *AMA-Journal of American Medical Association* **269**: 3140–3145.

Bardsley, M. *et al.* (1998), *The Health of Londoners: A Public Health Report for London*, London: Health of Londoners Project.

Bates, B. (1992), *Bargaining for Life: A Social History of Tuberculosis, 1876–1938*, Philadelphia: University of Pennsylvania Press.

Bayer, R. (1996), 'Does anything work? Public health and the nihilist thesis', paper presented at the 124th Annual Meeting of the American Public Health Association, New York City.

Bayer, R. and L. Dupuis (1995), 'Tuberculosis, public health, and civil liberties', *American Review of Public Health* **16**: 307–326.

Bhatti, N., M.R. Law, J.K. Morris, R. Halliday, and J. Moore-Gillon (1995), 'Increasing incidence of tuberculosis in England and Wales: a study of the likely causes', *British Medical Association* **275**: 487–489.

Brudney, K. and J. Dobkin (1991), 'Resurgent tuberculosis in New York City: human immunodeficiency virus homelessness, and the decline of tuberculosis control programs', *American Review of Respiratory Diseases* **144**: 745–749.

Cigno, A. (1998), 'Fertility decisions when infant survival is endogenous', *Journal of Population Economics* **11**: 21–28.

Cronjé, G. (1984), 'Tuberculosis and mortality decline in England and Wales, 1851–1910', in R. Woods and J. Woodward (eds), *Urban Disease and Mortality in Nineteenth Century in England*, London and New York: Batsford Academic and Educational and St. Martin's Press.

De Palma, A. and C. Lefevre (1988), 'Population systems with (non)-extensive interaction rate', *Math. Comput. Modelling* **10**: 359–365.

Dievler, A. and G. Pappas (1999), 'Implications of social class and race for urban public health policy making: a case study of HIV/AIDS and TB policy in Washington, DC', *Social Science and Medicine* **48**: 1095–1102.

Dubos, R. and J. Dubos (1987), *The White Plague: Tuberculosis, Man and Society*, New Brunswick, NJ: Rutgers University Press.

Elender, F., G. Bentham, and I. Langford (1998), 'Tuberculosis mortality in England and Wales during 1982–1992; its association with poverty, ethnicity and AIDS', *Social Science and Medicine* **46**: 673–681.
Ettner, S.L. (1996), 'New evidence on the relationship between income and health', *Journal of Health Economics* **15**: 67–85.
Farmer, P. (1996), 'Social inequalities and emerging infectious diseases', *Emerging Infectious Diseases* **2**: 259–269.
Feinstein, J.S. (1993), 'The relationship between socioeconomic-status and health – a review of the literature', *The Milbank Quarterly* **71**: 279–322.
Feldberg, G.D. (1995), *Disease and Class: Tuberculosis and the Shaping of Modern North American Society*, New Brunswick, NJ: Rutgers University Press.
Freudenberg, N. (1995), 'A new role for community organizations in the prevention and control of tuberculosis', *Journal of Community Health* **20**: 15–28.
Frieden, T.R. (1994), 'Tuberculosis control and social change', *American Journal of Public Health* **84**: 1721–1723.
Frieden, T.R., P.I. Fujiwara, R. Washko, and M. Hamburg (1995), 'Tuberculosis in New York City: turning the tide', *New England Journal of Medicine* **333**: 229–233.
Ginsberg, A.M. (1998), 'The tuberculosis epidemic: scientific challenges and opportunities', *Public Health Report* **113**: 128–136.
Hadley, J. and A. Osei (1982), 'Does income affect mortality? – an analysis of the effects of different types of income on age: sex race specific mortality-rates in the United States', *Medical Care* **20**: 901–914.
Haggerty, M. and C. Johnson (1996), 'The social construction of the distribution of income and health', *Journal of Economic Issues* **30**: 525–532.
Hamer, W.H. (1906), Epidemic Disease in England, *The Lancet* **1**: 733–739.
Hardy, A. (1993), *The Epidemic Streets: Infectious Disease and the Rise of Preventive Medicine, 1856–1900*, Oxford: Clarendon Press.
Hayward, A. (1998), *Tuberculosis Control in London – The Need for Change: A Report for the Thames Regional Directors of Public Health*, London: NHS Executive.
Jaramillo, E. (1999), 'Encompassing treatment with prevention: the path for a lasting control of tuberculosis', *Social Science and Medicine* **49**: 393–404.
Kermarck, W.O. and A.G. McKendrick (1927), 'A contribution to the mathematical theory of epidemics', *Proceeding Royal Society of London*, Series A **115**: 700–721.
Kitagawa, E.M. and P.M. Hauser (1973), *Differential Mortality in the United States: A Study in Socioeconomic Epidemiology*, Cambridge, MA: Harvard University Press.
Landesman, S. (1992), 'Commentary: tuberculosis in New York City – the consequences and lessons of failure', *American Journal of Public Health* **83**: 76–768.
Lerner, B.H. (1993), 'New York City's tuberculosis control efforts: the historical limitations of the "War on Consumption"', *American Journal of Public Health* **83**: 758–766.
Lotka, A.J. (1925), 'Contribution of the analysis of malaria epidemiology', *American Journal of Hygiene* **3**: 1–21.
McFarlane, N. (1989), 'Hospitals, housing, and tuberculosis in Glasgow', *Social History of Medicine* **2**: 59–85.
McKee, M. and B. Jacobson (2000), 'Public health in Europe', *Lancet* **356**: 665–670.
McKeown, T. and R.G. Record (1962), 'Reasons for the decline of mortality in England and Wales during the nineteenth century', *Population Studies* **94**: 122.
Murray, J.D. (2002), *Mathematical Biology*, London, New York: Springer.
Orcutt Duleep, H. (1985), 'Measuring the effect of income on adult mortality using longitudinal administrative record data', *The Journal of Human Resources* **32**: 238–251.
Ravaglione, M.C. and F. Luelmo (1996), 'Update on the global epidemiology of tuberculosis', *Current Issues in Public Health* **2**: 192–197.

Rodrigues, L.C. and P.G. Smith (1990), 'Tuberculosis in developing countries and methods for its control', *Trans Royal Society for Tropical Medicine and Hygiene* **84**: 739–744.
Solow, R.M. (1956), 'A contribution to the theory of economic growth', *Quarterly Journal of Economics* **70**: 65–94.
Sumartojo, E. (1993), 'When tuberculosis treatment fails', *American Review of Respiratory Diseases* **147**: 1311–1320.
Swan, T.W. (1956), 'Economic growth and capital accumulation', *Economic Record* **32**: 343–361.
Volterra, V. (1926), 'Variazioni e Fluttuazioni del Numero d'Individui in Specie Animali Conviventi', *Mem. Acad. Lincei* **2**: 31–113.
Wallace, D. (1990), 'Roots of increased health inequality in New York', *Social Science and Medicine* **31**: 1219–1227.
World Bank (2002), 'World Development Indicators', http://www.worldbank.org/data
World Health Organization (2002), *World Health Report 2002*, Geneva, Switzerland.
Young Jr., R.C., R.E. Rachel, S.B. Bailey, H.L. Tate, and B. Nelson-Knuckles (1997), 'Strategies for suppression, containment and eradication of resurgent tuberculosis', *Journal of Health Care for the Poor and Underserved* **8**: 424–436.