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Infectious Disease and Economic Growth: the Case of Tuberculosis

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Abstract. We consider the links between the health structure of the population and the productive system of an economy that is subject to infectious disease, in particular tuberculosis. Reviewing the models of tuberculosis suggests that a Lotka-Volterra type system can capture the dynamics of epidemics. We combine this with a Solow-Swan growth model: output is produced from capital and healthy labour; the demographic parameters of the Lotka-Volterra type system are functions of the capital healthy labour ratio. We find three stationary states, two of which are extensions of population equilibria and the third of which has a positive capital healthy labour ratio. There is also a partial balanced growth path in which there is no disease and the healthy population and capital stock grow at a common rate but this path is unstable. We analyse the local dynamics and, in the context of global analysis of two examples, find that the epidemiological-economic stationary state is locally stable and an attractor for a wide range of initial conditions. The way in which the net birth rate of susceptibles responds to prosperity determines the level of the stationary state prevalence of the disease. The interaction between the disease and the economy can also decrease the amplitude of epidemic cycles.

1. Introduction

There is good evidence that the implementation of tuberculosis[1] (TB) control programmes based on the use of antitubercular drugs and BCG vaccinations has failed to prevent recent TB epidemics in many developing countries[2]. However, in most developed countries TB is no longer endemic yet was controlled before the development of comprehensive health care facilities.

[1] Two main forms of TB in humans exist: pulmonary TB and extra-pulmonary TB, the former is most common and is the only infectious form. TB develops in the human body in two stages. The first stage occurs when an individual who is exposed to micro-organisms from an infectious case of TB becomes infected (tuberculous infection). This stage is referred to as the *passive* TB infectious case. An individual's risk of infection depends on the extent of exposure to droplet nuclei and his/her susceptibility to infection. The risk of transmission of infection from a person with sputum smear-negative pulmonary TB is relatively low and with extra-pulmonary TB is even lower. The risk of infection of a susceptible individual is high with close, prolonged, indoor exposure to a person with sputum smear-positive pulmonary TB. The degree of crowding and of intimacy of exposure are therefore important factors. This suggests that the population dynamics of TB is extremely sensitive to urbanization. Subsequently, at a second stage, some of the individuals who have become infected develop the disease from this infection; the *active* TB infectious case. Individuals are most likely to develop disease in the period immediately following infection but they continue to be exposed to risk of TB throughout the remainder of their lives. The development of TB following infection with tuberculosis micro-organisms is usually prevented by the immunosystem. Only a relatively low proportion of those individuals who have been infected with TB develop the disease. Once infected, the likelihood of developing active TB is 10% in a lifetime. When the protection provided by the immunosystem is reduced, the TB micro-organisms which are dormant begin to multiply, causing TB disease. Comstock (1982); IUATLD (1996); Nardell et al. (1986); Sepkowitz (1996); Weatherall (1996); WHO (1998a).

[2] Alvi et al. (1998); De Cock, Chaisson (1999); Keynon et al. (1999); Kimerling et al. (1999); Madras Tuberculosis Institute Bangalore (1980); Netto et al. (1999); Sumartojo (1993); Zumla (1998).

In this paper, recognising that medical innovation and health targeted policies have limits in controlling TB, we explore whether economic growth can control the spread of the disease. In particular we examine the links between TB and rising economic prosperity which strengthens the basic immune system of individuals and improves the general infrastructure of the economy (in housing, diet, working conditions, transport and health infrastructures). Rising prosperity can slow the disease transmission process both through increasing TB resistance, reducing involuntary interpersonal contact arising from congestion and providing a better access to health care and services in urban as well as in rural areas.

Historically, the impact of changes in social and economic factors on the disease transmission process is observable in the TB epidemics in Europe and America from the late 18th century to the early 20th century. The industrial revolution, both in Europe and America and part of Africa, provided the ideal conditions for the establishment of TB that rapidly became the primary cause of death. The growth of industrial and urban centres, long working hours and poor working conditions, low wages, overcrowded living quarters, poor hygiene standards and inadequate diet caused a sharp increase in TB mortality[3]. The rise of TB in Europe and America was followed by a long period of decline in the prevalence of the disease which was largely independent of medical intervention (which remained appalling through most of the century); the control of TB was more related to improvements in housing, working conditions and nutrition[4]. Contemporary evidence is that poverty, malnutrition, overcrowded housing and poor hygienic conditions are the key factors behind the recent spread of TB[5]. This is particularly true in the context of developing countries (sub-Saharan Africa, Asia and Latin America) where there are similar bursts of economic growth and increases in inequality associated with rapidly increasing urbanisation[6]. Geographical extensions of urban areas are often associated with deteriorating and crowded living/working conditions, such as lack of transport, water, drainage and health care delivery, that have favoured the spread of communicable diseases (shanty towns). Health status has been therefore considered to be inversely correlated with the degree of urbanization[7].

Conversely, the health status of the population affects the productivity of the labour force and hence the prosperity of the economy. There are several historical examples: the import of smallpox to Mid-America in the 16th century amongst the local population who had no immune resistance to the disease led to their decimation which in turn eliminated the local labour force for working the silver mines; the plague in the Middle East had the effects of reducing the productive work force so heavily that localised famines emerged[8].

[3] Stephens (1995).

[4] Packard (1989).

[5] Together with the emergence of multidrug-resistant strains of TB (MDR-TB) and the growth of endemic HIV/AIDS infection which compromises the immune system's ability to control dormant TB mycobacteria. As stated by Weatherall et al. (1996) patients with congenital or acquired immunosuppression are particularly prone to TB. HIV infection considerably enhances the reactivation rate of TB in the infected person: about 10% of doubly infected (infected with both HIV and TB) susceptibles develop active TB each year. As reported by WHO (1999a, 1999b), a HIV-positive individual who is also passively TB infected is 30 times more likely to become sick with TB than a TB infected individual who is HIV negative. Barnhoorn, Adriaanse (1992), Chan-Yeung et al. (1971), Chaulet (1987); De Cock, Chaisson (1999), Farmer et al. (1992), Mata (1985), Menzies et al. (1993), Nightingale et al. (1990), Pilheu (1998), Reichman (1997), Rubel, Gallo (1992), Wallace, Wallace (1997), WHO (1998b, 1999a).

[6] As reported by UN, nearly two-thirds of the world's current urban population live in the developing world. UN(1996a, 1996b, 1997, 1998), UNU/WIDER (1998a, 1998b).

[7] Mutatkar (1995); Phillips (1993); WHO (1993, 1996).

[8] Watts (1997).

In more contemporary times there is evidence that health status affects labour force participation and labour income[9]. Dasgupta (1997) finds that early nutrition and morbidity have significant effects on long term work capacity.

From this we can analyse the two-way interaction between the economy and TB. On the one hand, poverty and low prosperity generate conditions in which the disease can flourish. On the other hand, with high prevalence of the disease the work force is reduced so that prosperity falls.

In this paper we look at the dynamics of this basic two way interaction by combining a simple Solow-Swan growth model with a simple representation of the epidemiology of TB. We call this the *epidemiological-economic growth system* and find that generally it has a partial balanced growth solution in which there is no disease and capital and healthy labour grow at a common rate. We find that an arbitrary path will not converge to the disease-free path so that economic forces cannot eliminate TB. However, they can control its prevalence, reducing to an endemic level. In addition there are three stationary states of the system. One is the origin leading to extinction of both healthy and sick; one is an epidemiological-economic stationary state in which there are constant nonzero levels of capital stock and of sick and healthy individuals; one is a pure epidemiological state in which the economy fails to function at all (capital stock and output are zero) and healthy and sick individuals coexist. Since we have a three dimensional dynamic system (the variables are the capital stock and the numbers of healthy and sick, respectively) with three stationary states, the dynamics of the system are quite rich. The origin is one stationary point; global analysis reveals that it is unstable. We find that locally the epidemiological-economic equilibrium, where all variables are positive, can display a wide variety of local dynamic patterns depending on parameter values. Linearising around the equilibrium, there may be: three real roots (two positive and one negative, an unstable saddle-node or three negative, a stable node) or a pair of complex conjugate together with a negative real root. The equilibrium in which the economy plays no role (zero capital) is locally a 3D centre: two pure imaginary eigenvalues and one real eigenvalue. The stationary state involving zero capital stock and positive population replicates the two-dimensional dynamic properties of the Lotka-Volterra model and adds a third real eigenvalue (and so gives a direction of either monotone convergence or divergence to the corresponding pure demographic equilibrium).

To explore the global dynamics of how solution paths behave between these three stationary states we select functional forms for the way in which demographic parameters respond to economic prosperity (essentially based on logistic functions) and for the production function (a CES with elasticity of substitution less than one between capital and labour) and numerically integrate solution paths. The results indicate that whether the net birth rate of susceptibles is increasing or decreasing in prosperity is important in determining the stationary state prevalence of the disease. However, the dynamic properties of the system are very similar whether α is increasing or decreasing with prosperity.

Numerical simulations for two examples indicate that the stationary state with a nonzero level of productive capital is locally stable and an attractor for a wide set of initial conditions. In the long run the two-way interaction between the population and the economic structure results in an epidemiological-economic equilibrium.

The paper is organized to outline alternative epidemiological models of TB in Section 2; to present the general analytics of the epidemiological-economic growth system structure in Section 3; to give numerical simulations in Section 4 and to conclude in Section 5.

[9] Bartel and Taubman (1979), Ettner (1996), Lee (1982), Luft (1995).

2. THE DISEASE PROCESS

We think of a population N_t of individuals in a given area at instant t. Generally individuals can be in one of four states: susceptible but healthy; latently infected and not infectious; actively infected and infectious; recovered from the disease[10]. Some of the epidemiological models distinguish more states than this e.g. in the case of various strains of TB which differ in the time gap between first infection and becoming actively infected and infectious (so called fast and slow TB)[11].

The population changes through time due to the births of susceptibles and to deaths either from causes other than TB or from the disease. Historically outbreaks of disease have generally followed an epidemic pattern. For example a common pattern in medieval England was for a geographical area to succumb to an outburst of plague over a period of five months or so, often concentrated at particular times of year, but then the disease would die away, subsequently breaking out again. This type of dynamic evolution is seen in predator-prey models; so a very common paradigm for modelling the disease dynamics is some version of the Lotka-Volterra system[12]:

$$N_t = X_t + Y_t + L_t + R_t \tag{1}$$

$$\begin{cases} \dot{X}_t = \alpha X_t + \dot{R}_t - \beta(X_t, L_t, Y_t) X_t Y_t \\ \dot{L}_t = \beta(X_t, L_t, Y_t) X_t Y_t - (\lambda_1 + \lambda_2 + \lambda_3) L_t \\ \dot{Y}_t = \lambda_2 L_t - \omega Y_t - \rho Y_t \\ \dot{R}_t = \rho Y_t + \lambda_3 L_t \end{cases} \tag{2}$$

where X_t is the stock of susceptibles; L_t is the stock of latently infected; Y_t is the stock of actively infected; R_t is the stock of recovered individuals; all at instant t. Furthermore, α is the net birth rate of susceptibles (birth rate minus death rate due to non-disease causes); $\beta(X_t, L_t, Y_t)$ is the probability that on meeting an actively infected person, a susceptible person becomes latently infected; λ_1 is the natural death rate of latently infected individuals from non-disease causes; λ_2 the proportion of the latently infected stock who become actively infected; λ_3 the proportion of latently infected who recover; ρ the proportion of actively infected who recover and ω the death rate of the actively infected either through the disease or natural causes. Assuming that the various rates of movement between groups are constant proportions ($\alpha, \lambda_i, \rho, \omega$ with $i = 1, 2, 3$) is a simplification, particularly so for λ_i where the assumption is that the length of time that one has been latently infected has no effect on the chances of recovery, developing active infection or death[13]. Obviously this system refers to the case in which the recovered do not gain immunity from the disease but rejoin the pool of susceptibles.

A major issue is the interpretation of the infection process $\beta(X_t, L_t, Y_t) X_t Y_t$. Usually this is in terms of $\beta(.)Y_t$ giving the probability of a given susceptible becoming infected and then multiplying this by X_t gives the number of newly infected susceptibles:

New infections = Pr (susceptible and infected meet) Pr (infection arising|meeting) X_t.

[10] The nature of recovery can also be heterogeneous: infected individuals who have recovered may have permanent immunity from the disease in which case they form a class on their own; or they may immediately become susceptible to a new attack of the disease in which case they join the existing group of susceptibles; for TB the latter is the case.

[11] Vynnycky and Fine(1997).

[12] Lotka (1925); Volterra (1926).

[13] Examples when the length of the latent period influences the probability of TB reactivation are in Chan-Yeung et al. (1971) and Grzybowski et al. (1965).

To model this we have to represent the meeting process and then the infection process arising from any meeting. For the latter the most common assumption is that the chance of infection out of any meeting between a susceptible and an infected is constant. The main exception to this is a form of density dependence where (e.g. with a high prevalence of the disease - high Y_t/X_t) individual behaviour may adjust to reduce the chance of infection in a meeting e.g. by wearing protective clothing or otherwise altering the nature of the meeting. For the former usually there is a model of social interaction and then, within that, some view of whether the interaction is between two susceptibles or a susceptible and an actively infected. It is plausible that the chance of a meeting being between a susceptible and an actively infected, *given that there is a meeting*, is Y_t/N_t. If the chance of a given susceptible meeting anyone else at all is proportional to N_t (i.e. a more densely populated region generates a higher number of meetings between people than a lower density area) then the probability that a given susceptible meets an infected is indeed Y_t. Alternatively if N_t were constant so that only the structure of the population but not its size were changing then Y_t *would* be the proportion of infected; with a randomly mixing population, where each person meets one other person every instant, this would be the probability of meeting an infected person.

If we adopt any of these assumptions, then β is a constant and the number of new latent infections is βXY. The equations (2) are then equivalent to

$$\begin{cases} \dot{X}_t = \alpha X_t + \rho Y_t + \lambda_3 L_t - \beta X_t Y_t \\ \dot{L}_t = \beta X_t Y_t - (\lambda_1 + \lambda_2 + \lambda_3) L_t \\ \dot{Y}_t = \lambda_2 L_t - \omega Y_t - \rho Y_t \end{cases} \tag{3}$$

From these equations it follows that total population changes according to

$$\dot{N}_t = \dot{X}_t + \dot{Y}_t + \dot{L}_t = \alpha X_t - \lambda_1 L_t - \omega Y_t \tag{4}$$

that is, the difference between the net birth rate of the susceptibles and the combined deaths of the latent and actively infected individuals. This system has two stationary points:

$$X^* = Y^* = L^* = 0 \tag{5}$$

$$\begin{aligned} X^* &= (\lambda_1 + \lambda_2 + \lambda_3)(\omega + \rho)/(\lambda_2 \beta) \\ Y^* &= \alpha(\omega + \rho)(\lambda_1 + \lambda_2 + \lambda_3)/[\lambda_2 \beta \omega + \beta \lambda_1 (\omega + \rho)] \\ L^* &= \alpha(\omega + \rho)^2(\lambda_1 + \lambda_2 + \lambda_3)/[\lambda_2^2 \beta \omega + \beta \lambda_1 \lambda_2 (\omega + \rho)] \end{aligned} \tag{6}$$

The first corresponds to extinction and the second to a constant population level and structure. There are no steady growth paths of the system. To investigate the local dynamics, around the origin we linearise and find that locally there are three real roots of the Jacobian[14]:

$$\alpha, \quad -(\lambda_1 + \lambda_2 + \lambda_3), \quad -(\omega + \rho) \tag{8}$$

[14] Linearisation around any stationary point gives a Jacobian of

$$\begin{bmatrix} \alpha - \beta Y & \lambda_3 & \rho - \beta X \\ \beta Y & -(\lambda_1 + \lambda_2 + \lambda_3) & -\beta X \\ \mathbf{0} & \lambda_2 & -(\omega + \rho) \end{bmatrix} \tag{7}$$

two of these roots are negative and one of them is positive, so we have a 3D saddlepoint with an additional stable direction[15].

Around the nonzero stationary point simple expressions for the roots are unavailable. However, as the determinant of the Jacobian is

$$-\alpha(\omega+\rho)(\lambda_1+\lambda_2+\lambda_3)<0 \tag{9}$$

and the trace is

$$-\left\{\alpha\left[\omega\lambda_3+\rho(\lambda_2+\lambda_3)\right]/\left[\lambda_2\omega+\lambda_1(\omega+\rho)\right]+\left[(\lambda_1+\lambda_2+\lambda_3)+(\omega+\rho)\right]\right\}<0 \tag{10}$$

the possible patterns of real parts of eigenvalues are either three negative real roots (3D node with positive attractor) or one negative and two positive real roots (focus sink with negative attractor or 3D saddlepoint with an additional unstable direction).

Many writers have applied this type of structure to analyse the dynamic transmission of TB. The microdynamics of TB is very complex. Interest centres on whether simple population interaction models can capture the main empirical trends in prevalence. A recent study is in Blower et al. (1995), who consider a population divided into three different classes: susceptible (defined as uninfected individuals), latent (infectious and non-infectious cases) and recovered. TB is described as having two pathogenic mechanisms: direct progression (when the disease develops soon after infection) and endogenous reactivation (when the disease can develop many years after infection). These two mechanisms are modelled by assuming that a constant proportion of the newly infected develop TB directly and a constant proportion of the newly infected enter the latent class.

The dynamic of the TB epidemic is described by a system of three differential equations. In Blower et al.'s notation,

$$\begin{cases} \dot{X}(t)=\pi-\beta T(t)\,X(t)-\mu\,X(t) \\ \dot{L}(t)=(1-p)\,\beta T(t)\,X(t)-v\,L(t)-\mu\,L(t) \\ \dot{T}(t)=v\,L(t)+p\,\beta T(t)\;X(t)-(\mu+\mu_T)\,T(t) \end{cases} \tag{11}$$

where $X(t)$, $L(t)$ and $T(t)$ represent the susceptible, latent and active infectious TB individuals, π the constant rate of recruitment to the susceptible population, β the probability that an infectious case successfully transmits the infection to a susceptible. Here, μ is the per capita average non-TB mortality rate, μ_T the per capita average TB mortality rate, v the rate at which the latent individuals develop active TB, p the proportion of newly infected who develop tuberculosis directly and, therefore, $(1-p)$ the proportion of the newly infected who enter the latent class. Individuals who recover from the disease are not modelled. Blower et al. simulate an epidemic arising from the introduction of one infectious individual into an uninfected population. They also analyse the epidemic doubling time as a function of the average number of secondary infections produced per infectious case per year and the threshold population-size (as the minimum number of susceptibles that have to be present before a TB epidemic can occur) for different transmission coefficients. The main outcome obtained in the analysis is that TB epidemics have slow intrinsic dynamics. Given this view the recent declining prevalence of TB infection over the past century in developed countries is the natural decline in a long epidemic which first increased during the 1600s. They do not examine the analytical properties of the

[15] We use the classification of Verhulst (1990).

dynamics; but actually the system has two stationary points at

$$\begin{aligned} X^* &= \frac{(\nu+\mu)(\mu+\mu_T)}{\beta(\nu+p\mu)}, \quad L^* = \frac{(\pi\beta(\nu+p\mu)-\mu(\nu+\mu)(\mu+\mu_T))(1-p)}{(\nu+\mu)(\nu+p\mu)\beta}, \\ T^* &= \frac{\pi\beta(\nu+p\mu)-\mu(\nu+\mu)(\mu+\mu_T)}{(\nu+\mu)(\mu+\mu_T)\beta} \end{aligned} \tag{12}$$

and

$$T^* = L^* = 0, \quad X^* = \pi/\mu \tag{13}$$

The stationary state with some disease typically has one real root and a pair of complex conjugate roots (focus sink/source with an additional positive/negative attractor). Locally the stationary state in which disease is eliminated has eigenvalues

$$-\mu, \quad -0.5\{\mu\upsilon+\mu\mu_T-p\beta\pi+2\mu^2\pm[\mu^2(\mu_T-\upsilon)^2-2\beta\pi\mu(p(\mu_T+\nu)-2\nu)+p^2\beta^2\pi^2]^{0.5}\}/\mu \tag{14}$$

so that there may be either three real roots (3D node positive/negative attractor) or a negative real root and a pair of complex conjugate roots (focus sink with an additional positive/negative attractor)[16].

Blower et al.'s approach is intermediate between a completely descriptively realistic treatment of the disease and a stylised paradigm. For example, the transition between passive and active infection is not governed by proportionality in reality; similarly, most population models would use geometric rather than arithmetic growth in the number of susceptibles; the recovered class are not analysed. Nevertheless, as the authors demonstrate, this level of complexity of modelling is appropriate to capture the salient long-run trends. Both (3) and (11) also demonstrate the tradeoff between descriptive realism and analytical generality; either one has a high dimensional descriptively accurate system whose dynamic behaviour can only be numerically simulated for a sample of initial conditions; or some of the realism is sacrificed to permit global analysis of the dynamics. For the latter the most that we can realistically manage is three dimensions.

For TB infectious diseases where there are no latents or where the latent class is negligible and infection switches susceptibles directly into active infection (e.g. $p = 1$) (3) or (11) collapses, in our notation, to

$$\begin{cases} \dot{X}_t = \alpha X_t - \beta X_t Y_t \\ \dot{Y}_t = \beta X_t Y_t - \omega Y_t \end{cases} \tag{16}$$

in which case there are again two stationary points

$$X^* = Y^* = 0 \tag{17}$$

$$X^* = \omega/\beta, Y^* = \alpha/\beta \tag{18}$$

As is well known the latter stationary point has two pure imaginary roots so long as $\alpha > 0$ so that there are closed cycles about this stationary point[17]. Notice that if

[16] Linearisation around any stationary point gives a Jacobian of

$$\begin{bmatrix} -\beta T-\mu & 0 & -\beta X \\ (1-p)\beta T & -(\mu+\nu) & (1-p)\beta X \\ p\beta T & \nu & -(\mu+\mu_T)+p\beta X \end{bmatrix} \tag{15}$$

[17] Linearisation around any stationary point gives a Jacobian of

$$\begin{bmatrix} \alpha-\beta Y & -\beta X \\ \beta Y & \beta X-\omega \end{bmatrix} \tag{19}$$

$\alpha \leq 0$ then we lose the centre as a viable stationary state; in this case the healthy just decay to zero through the combined effects of natural death and infection by the sick. Throughout this paper we assume that $\alpha > 0$. It is less well known that the origin is locally a saddlepoint. A typical phase diagram is shown in Fig. 2.1.

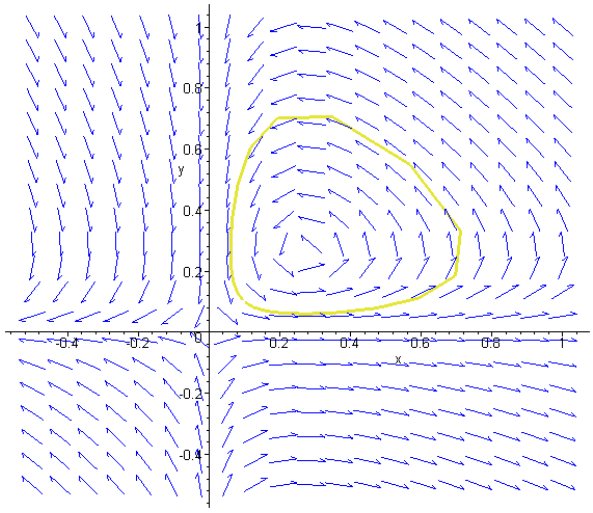

Figure 2.1

In similar vein to Blower et al., for the subsequent analysis we concentrate on the system without latents since our primary purpose is to analyse how the productive economy will impact on the dynamics of the system; having more extreme dynamics (closed cycles) will allow us to make the effects of economic structures more stark.

3. Growth and Prosperity

As we noted in the introduction, general prosperity brings with it improvements in diet, housing and somewhat less directly the socioeconomic infrastructure which have effects on the infection process and also possibly on the birth and death rates of the different population groups. In terms of the epidemiological model α, β and ω are in general functions of economic prosperity; there are a variety of ways of measuring this. We take the epidemiological parameters to be functions of the capital/productive labour ratio. This will be consistent with a variety of interpretations e.g. epidemiological parameters depending on per capita consumption of the healthy. However, we also want to model the causes of economic growth; there is some evidence that there are feedback effects of infectious disease on the ability of the economy to provide growth through debilitating the labour force[18]. A natural economic approach is to assume that infected individuals cannot work; the production possibilities of the economy depend on the productive labour force *inter alia*. Hence, if there is a high proportion of infected then output and economic prosperity is low *ceteris paribus*. Since economic prosperity affects demographics, with low prosperity, the net birth rate of susceptibles falls; the death rate of the infected rises and the infection rate rises. This leads to a fall in the productive labour force which in turn leads to a fall in consumption per capita. A vicious cycle emerges. Hence, it would appear that if output depends on susceptible labour alone the system with endogenous economic effects reinforces instability. However, this ignores the possibility of investment in capital.

[18] Bartel and Taubman (1979), Ettner (1996), Lee (1982), Luft (1995).

To model the economic system we assume there is a single good which can be consumed or invested: at any instant t, output of the single good is given by $F(K_t, X_t)$ where K_t is the existing capital stock. The production function satisfies the usual neoclassical properties ($F(K_t, X_t)$ is increasing, homogenous of degree one and has diminishing marginal productivity of each input). In addition we assume that each input is essential in that $F(0, X_t) = F(K_t, 0) = F(0,0) = 0$ but that the marginal product of each input remains finite as its quantity tends to zero i.e. that $\lim_{X\to 0} \frac{\partial F(K_t,X_t)}{\partial X}$ and $\lim_{K\to 0} \frac{\partial F(K_t,X_t)}{\partial K}$ each exist as finite positive numbers. For example we might think of a CES production function with elasticity of substitution less than unity[19].

Capital stock is owned equally by the healthy and infected individuals. A healthy individual receives labour income equal to the wage, W, for an inelastically supplied amount of work, and capital income equal to the product of the rental rate and his/her share of the capital stock. The wage and rental rate are set in competitive markets by the marginal productivity of inputs. The aggregate labour income is WX_t, all accruing to the healthy individuals. The aggregate capital income of the healthy is $\rho_t K_{Xt}$ where ρ_t is the rental rate on capital and $K_{Xt} = K_t X_t/(X_t + Y_t)$. Income of the infected individual is purely capital income $\rho_t K_{Yt}$ where $K_{Yt} = K_t Y_t/(X_t + Y_t)$. Each period, as population changes, capital is redistributed amongst the healthy and infected individual of that period. Capital depreciates linearly at a constant rate, ϕ. Each individual has a proportional saving function with saving rate s. Hence, in the aggregate, capital accumulates according to

$$\begin{aligned} \dot{K}_t &= s\left[WX_t + \rho(K_X + K_Y)\right] - \phi K_t \qquad (20) \\ &= sF(K_t, X_t) - \phi K_t \qquad (21) \end{aligned}$$

from the constant returns to scale. The demographic parameters α, β and ω are functions of K_t/X_t. In Section 4 we select functional forms for the production function and α, β and ω. Initially we just assume $\beta'(.) \leq 0$ and $\omega'(.) \leq 0$ in line with the predominant empirical findings[20] and that $\beta(0)$ and $\omega(0)$ are nonzero. The sign of $\alpha'(.)$ is less obvious; there is evidence that $\alpha'(.) \leq 0$ both because the birth rate may fall[21] and infant mortality may rise[22] with prosperity although the mortality rate generally falls[23]. However, the balance between movements in fertility and mortality may make $\alpha'(.) \geq 0$. It is also natural to assume that α, β and ω are bounded above by $\bar{\alpha}$, $\bar{\beta}$ and $\bar{\omega}$.

It is easy to see that there is no balanced growth path along which X_t, Y_t and K_t each grow at a nonzero rate g[24]. However, there is a partial balanced growth path with $Y_t \equiv 0$ and X_t/K_t constant solving $\alpha(K/X) = sf(X/K) - \phi$ where for $Z_t = X_t/K_t$

[19] If we write the production function as $F(K_t, X_t) = (rK_t^m + qX_t^m)^{\frac{1}{m}}$ where the elasticity of substitution is $1/(1-m)$ then for $m < 0$, $F(0, X_t) = F(0,0) = F(K_t, 0) = 0$ and since $\frac{\partial F}{\partial K} = (r + q(\frac{X_t}{K_t})^m)^{\frac{1-m}{m}}$, $\frac{\partial F(0,X_t)}{\partial K} = r^{\frac{1}{m}}$ and $\frac{\partial F(K_t,0)}{\partial K} = 0$.

[20] Ehrlich, Lui (1997); Lincoln (1993).

[21] Lee (1997); Robinson, Srinivasan (1997).

[22] Waldmann (1992).

[23] Anderson et al. (1997); Chapman and Hariharan (1993); Ehrlich, Lui (1997); Feinstein (1993); Mackenbach and Looman (1994).

[24] If there were a balanced growth path g then (16) would require

$$\begin{cases} g = \alpha - \beta Y_t \\ g = \beta X_t - \omega \end{cases} \qquad (22)$$

where along the balanced growth path α, β and ω would be constants. But then setting $Y_t = Y_0 \exp^{gt}$, the only value of g that satisfied (16) for all t is $g = 0$.

we define $f(Z_t) = F(1, Z_t)$. Along this path X_t and K_t both grow at the rate $\alpha(K/X)$ and there is no disease in the system. If initially the variables start on this path then disease cannot break out. A central question is if we start with arbitrary initial conditions involving some infection, then under what circumstances will the time path of the system approach this disease free balanced growth time profile? A necessary condition for this is that ultimately $\dot{Y}_t/Y_t$ eventually becomes and remains negative. Since Y_t is bounded below by zero and $Y_0 > 0, \lim_{t\to\infty} Y_t = 0$ implies $\dot{Y}_t < 0$ for sufficiently large t. But as $Y_t \to 0$, $\dot{X}_t/X_t \to \alpha > 0$. It follows that X_t will grow asymptotically at a positive rate $\lim_{t\to\infty} \alpha(K_t/X_t)$, but $\dot{Y}_t/Y_t = \beta X_t - \omega$ where ω and β are finite and positive. Hence it is impossible for $\dot{Y}_t/Y_t$ to ultimately remain negative as $t \to \infty$ and so it cannot be that $Y_t \to 0$. The result is that the system cannot eradicate the disease; interest then focuses on how the interaction of the economic growth system with the epidemiological process controls the dynamics of the population structure and economic prosperity.

To analyse the general dynamics we look at stationary states of the full system and behaviour around them: equating the LHS's of (16) and (21) to zero:

$$\begin{aligned} 0 &= \alpha X_t - \beta X_t Y_t \\ 0 &= \beta X_t Y_t - \omega Y_t \\ 0 &= sF(K_t, X_t) - \phi K_t \end{aligned} \tag{23}$$

and noting that α, β and ω are constant if X_t and K_t are, there are generally three stationary states[25]:

$$X^* = \frac{\omega}{\beta};\ Y^* = \frac{\alpha}{\beta};\ \left(\frac{K}{X}\right)^* = f^{-1}\left(\frac{\phi}{s}\right) \tag{24}$$

$$X^* = \frac{\omega}{\beta};\ Y^* = \frac{\alpha}{\beta};\ K^* = 0 \tag{25}$$

$$X^* = Y^* = K^* = 0 \tag{26}$$

The first of these has a nontrivial economic process with a stationary state capital healthy labour ratio that is independent of the behaviour of the epidemiological parameters; but in the other two the economy is irrelevant. In the stationary states that do not involve extinction, prevalence of the disease (Y_t/X_t or equivalently $Y_t/N_t = (1 + X_t/Y_t)^{-1}$) is given by α/ω. There is a lot of evidence that ω falls with rising prosperity[26]. Hence if the net birth rate of susceptibles increases with prosperity the effect of productive capital is to increase the stationary state disease prevalence. Conversely if the net birth rate of susceptibles falls with prosperity, whether economic effects increase or decrease the stationary state prevalence is ambiguous.

To see the local dynamics around these stationary states we use linearisations and then compute the relevant eigenvalues. Expanding around any stationary state to the first order gives the linearised system

[25] Whether the origin is an admissible stationary state depends on whether the demographic parameters have well defined values at $K_t = X_t = 0$.

[26] Anderson et al. (1997); Chapman and Hariharan (1993); Feinstein (1993); Mackenbach and Looman (1994).

$$\begin{bmatrix} \dot{X} \\ \dot{Y} \\ \dot{K} \end{bmatrix} = \begin{bmatrix} \alpha - \beta Y - \left(\alpha^{'} - \beta^{'} Y\right) \frac{K}{X} & -\beta X & \left(\alpha^{'} - \beta^{'} Y\right) \\ Y\beta - Y\left(\beta^{'} X - \omega^{'}\right) \frac{K}{X^2} & \beta X - \omega & \left(\beta^{'} X - \omega^{'}\right) \frac{Y}{X} \\ sF_X & 0 & sF_K - \phi \end{bmatrix} \begin{bmatrix} X \\ Y \\ K \end{bmatrix} \tag{27}$$

where X, Y and K are now measured as deviations from a particular stationary point and derivatives in the Jacobian are evaluated at that stationary point.

Summarising the results for the first stationary point (24) a wide variety of local dynamic patterns are possible. There may be a single real negative root with a pair of complex conjugate roots if the production function is not too concave relative to the marginal effects of prosperity on the epidemiological parameters (focus sink with either positive or negative attractor); or there may be three negative real roots (3D node positive attractor) or two positive and one negative real root (3D saddlepoint with an additional unstable direction) if the system is "very concave"; the appendix provides the technical conditions dividing these cases. It is easy to see that there are no roots with zero real part at this stationary state.

At the second stationary point (25) we have eigenvalues $sF_K - \phi$ and $\pm i\sqrt{\alpha\omega}$ where the sign of $sF_K - \phi$ is ambiguous at this stationary point. This replicates the roots of the pure Lotka-Volterra type system but adds the real root $sF_K - \phi$. There are then two possible dynamic patterns around (25); there is a two dimensional surface in (X, Y, K) space (in fact in the XY plane with $K = 0$) tangent to the complex eigenvector corresponding to the pure imaginary root. If the real root is negative then paths starting away from this surface spiral in a stable way towards the manifold except for a unique path through the "centre" of all such spirals which converges monotonely to the surface. Paths that start on the surface itself remain there and form pure cycles on the surface. If the real root is positive then there is the same pattern of paths but now they diverge away from the centre manifold.

At the third stationary point (26) we have $F(K, X) = 0$. To perform linearisations in a neighbourhood of the origin requires that F_K and F_X remain finite. In the case of a CES production function with elasticity of substitution less than unity whether this is so depends on the speed of convergence of K_t, X_t and Y_t to zero. The appendix gives details of the roots when such linearisations are possible; but in general we turn to global analysis to see how the origin relates to the other two stationary points.

There are various special cases of this structure (16) and (21) which are of interest:

(i) If $\beta = 0$ identically and α is constant then the system reduces to the Solow-Swan single sector growth model in (X_t, K_t) with the infected dying out gradually through time and effectively being segregated. This is analogous to the partial steady state with $Y_t \equiv 0$ in that X_t, K_t grow at a common rate but there is disease in the economy.

(ii) If α, β and ω are all nonzero constants then we still have three stationary states. The variation from the Solow Swan model is that X_t, the productive labour force, displays cycles. For this reason there is no steady state growth path. In fact in this case there are still three stationary states; the first of these has one negative real and two purely complex conjugate roots like the second whilst the third still has three real roots.

(iii) If $\beta = 0$ and α is a function of economic prosperity then we have an endogenous growth model in which the net birth rate of the work force varies with economic conditions.

(iii) If $\phi = 0$ then there are no stationary points and no steady state growth paths.

The results that we get here depend on the demographic structure assumed. In a related paper[27] we show that if we allow for capital and savings with the same sort of links between the demographic and economic structure as here but with homogenous demographics as in May and Anderson (1989) then the economy may settle down at a steady state.

4. A NUMERICAL EXAMPLE

As we have seen, the Lotka-Volterra demographic system combined with the Solow-Swan growth model gives us a new stationary state in which the prevalence of the disease is higher in the presence of growth than without growth if the net birth rate of susceptibles increases with the capital labour ratio; but prevalence is lower in this stationary state if the net birth rate of susceptibles falls with the capital labour ratio. In this section, we simulate the quantitative nature of the dynamics for each of these two cases by selecting particular functional forms. For the population dynamics there are quite strong arguments to select functional forms that give a bounded effect to economic prosperity on net population growth or the spread of disease. A simple form that will do this is related to logistic functions so we take

$$\begin{cases} \alpha(Z_t) = \alpha_0 - \alpha_1 \exp^{-\alpha_2/Z_t} \\ \beta(Z_t) = \beta_0 + \beta_1 \exp^{-\beta_2/Z_t} \\ \omega(Z_t) = \omega_0 + \omega_1 \exp^{-\omega_2/Z_t} \end{cases} \tag{28}$$

(remember $Z_t = X_t/K_t$). Here with zero capital, the baseline net growth of the healthy is $\alpha_0 - \alpha_1$; the baseline infection rate is $\beta_0 + \beta_1$ and the baseline death rate of the infected is $\omega_0 + \omega_1$. As consumption per capita tends to infinity the net growth rate of the healthy is α_0; the infection rate β_0 and the death rate of the infected ω_0. Consequently if α_1 is negative, the birth rate falls with the capital labour ratio; conversely, if α_1 is positive. The speed with which the upper bounds are reached is determined by the coefficients with subscript 2.

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

$$F(K, X) = (rK^m + qX^m)^{\frac{1}{m}} \tag{29}$$

where $m < 0$ and $r, q > 0$.

With these choices we have

$$F_X = q\left[r\left(\frac{K_t}{X_t}\right)^m + q\right]^{\frac{(1-m)}{m}} \tag{30}$$

$$\frac{F}{X_t} = \left[r\left(\frac{K_t}{X_t}\right)^m + q\right]^{\frac{1}{m}} \tag{31}$$

$$F_K = r\left[r + q\left(\frac{X_t}{K_t}\right)^m\right]^{\frac{(1-m)}{m}} \tag{32}$$

The three stationary states are, respectively, at

[27] Delfino, Simmons (1999).

$$X^* = \frac{\omega}{\beta};\ Y^* = \frac{\alpha}{\beta};\ \left(\frac{X}{K}\right)^* = \left[\left(\frac{\phi}{s}\right)^m /q - r/q\right]^{\frac{1}{m}} \tag{33}$$

$$X^* = \frac{\omega}{\beta};\ Y^* = \frac{\alpha}{\beta};\ K^* = 0 \tag{34}$$

$$X^* = Y^* = K^* = 0 \tag{35}$$

For the case in which the net birth rate increases with prosperity we select $m = -1$; $q = 0.7$; $r = 0.3$; $s = 0.2$; $\phi = 0.1$; $\alpha_0 = 0.06$; $\alpha_1 := 0.02$; $\alpha_2 = 1$; $\beta_0 = 0.1$; $\beta_1 := 0.3$; $\beta_2 = 1$; $\omega_0 = 0.2$; $\omega_1 = 0.2$; $\omega_2 = 1$. This implies that when capital stock is zero the net birth rate is 4%; the death rate of infected individuals is 40%; the infection rate is 40%. But when the capital labour ratio tends to infinity the net birth rate rises to 6%, the death rate of the infected falls to 20% and the infection rate falls to 10%. The elasticity of substitution is 0.5, the savings rate is 20% and the depreciation rate 10%.

With these parameter values the three stationary states are:

$$X^* = 1.721;\ Y^* = 0.461;\ K^* = 4.180 \tag{36}$$

$$X^* = 1.000;\ Y^* = .100;\ K^* = 0; \tag{37}$$

$$X^* = 0;\ Y^* = 0;\ K^* = 0; \tag{38}$$

We can calculate the eigenvalues corresponding to a linear approximation around the first two of these stationary states. Since the marginal products are not defined at $K_t = X_t = 0$ we do not attempt to explore the local stability of the origin. For the first stationary state (33) this gives us eigenvalues of:

$$\epsilon_1 = -.019 - .114i;\ \epsilon_2 = -.019 + .114i;\quad \epsilon_3 = -0.080 \tag{39}$$

which corresponds to the case of a pair of complex conjugates and one negative real root (focus sink positive attractor). Different parameter values would have generated the other combinations of eigenvalues near this stationary state. Around the second stationary state (37) the eigenvalues are:

$$\epsilon_1 = 0.126i;\quad \epsilon_2 = -0.126i;\quad \epsilon_3 = 0.567 \tag{40}$$

which reflects the analytical results above. In the pure epidemiological equilibrium when capital stock is zero, the stationary state prevalence of the disease is $Y/X = 10\%$ whereas in the epidemiological-economic equilibrium, the prevalence is more than twice as high at 27%; however, total population almost doubles.

We can then explore the local dynamics around each stationary state and the way these patch together to form a global phase space; to derive these we have numerically integrated the non-linear differential equations and not their linear approximations. So the phase spaces are globally accurate.

The results are given in Figures 4.1-4.2.

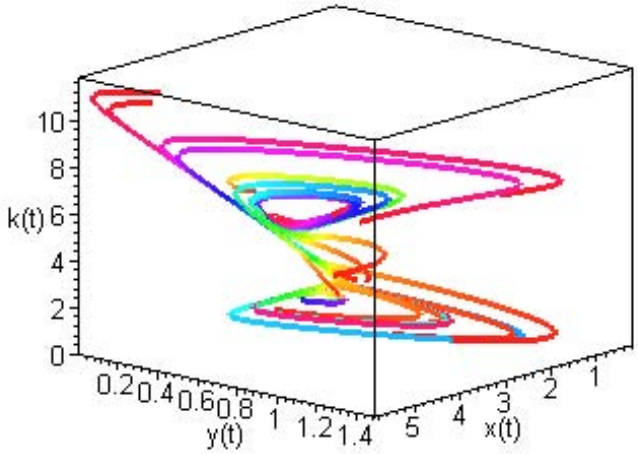

Fig. 4.1

Fig. 4.1 shows the global phase space with the three stationary states. Fig 4.2 shows small regions around the stationary states; the local dynamics of the stationary state with $K^* > 0$ shown in the top panel of Fig. 4.2 is a focus sink positive attractor and the dynamics around the stationary state with $K^* = 0$ in the middle panel is a 3D centre with an additional negative attractor. The stationary point at the origin has the dynamics described in bottom panel where paths start with a very low but positive value of K_t. The direction of movement is in terms of decreasing Y_t, increasing K_t and depending on initial conditions either increasing or approximately constant X_t. It is like the two dimensional saddlepoint of the pure Lotka-Volterra type system augmented by a direction of rapid growth in K_t.

The results indicate that the first stationary state is an attractor for paths which start near the other stationary points, and the paths rapidly converge to the first state. At the second and third stationary points the unstable direction in K_t seems to dominate the dynamics so that so long as initially $K_0 \neq 0$ even though it is small, economic growth takes over and the path converges in an oscillatory fashion to the first stationary state. However, for paths which start with relatively high values of K_0 , there is a three dimensional cycle in the variables with relatively rapid movement in K_t. If a path starts with K_0 actually equal to zero, then the system follows a closed Lotka-Volterra type cycle in the $X - Y$ plane; but for even small initial values of K_0, after some early cycles in X_t and Y_t, the path diverges to the first stationary state. From Figs 4.1-4.2 it is clear that there is virtually a linear direction of growth in K_t and X_t, and decay in Y_t as the effects of the economy take over until there is a cycle around the new epidemiological-economic stationary state.

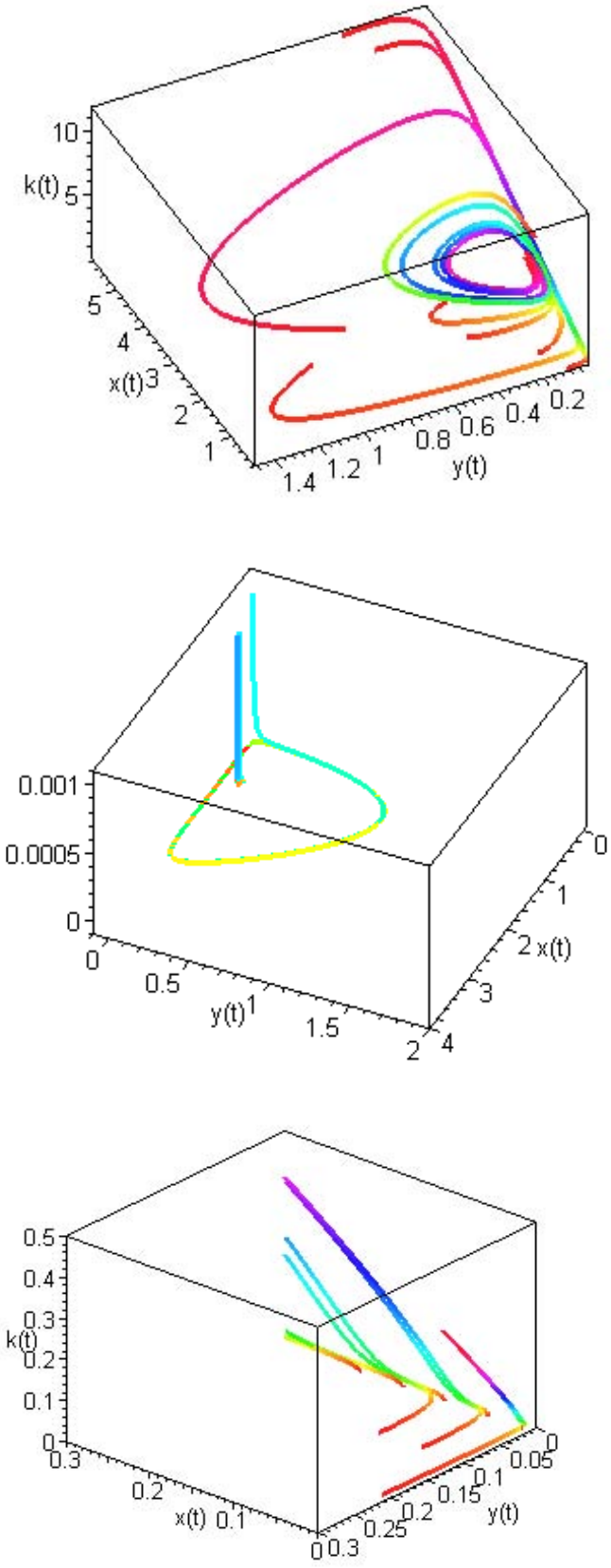
10
k(t) 5
5
4
x(t) 3
2
1
1.4 1.2 1 0.8 0.6 0.4 0.2
y(t)
0.001
0.0005
0
0 0.5 y(t) 1 1.5 2
0 1 2 x(t) 3 4
0.5
0.4
0.3
k(t) 0.2
0.1
0
0.3 0.2 x(t) 0.1 0
0 0.05 0.1 0.15 0.2 0.25 0.3
y(t)

Fig. 4.2

We can also compare the amplitude of cycles in X_t, Y_t with and without economic growth by integrating paths that start with identical values of X_0, Y_0 but in one case with $K_0 = 0$ and in the second case with $K_0 \neq 0$ over the same time horizon. Fig 4.3 demonstrates such a case where the paths start with $X_0 = 2.0, Y_0 = 0.4$ and with varying values of K_0. The result indicates that the cycles in X_t and Y_t have much lower amplitude when $K_0 \neq 0$.

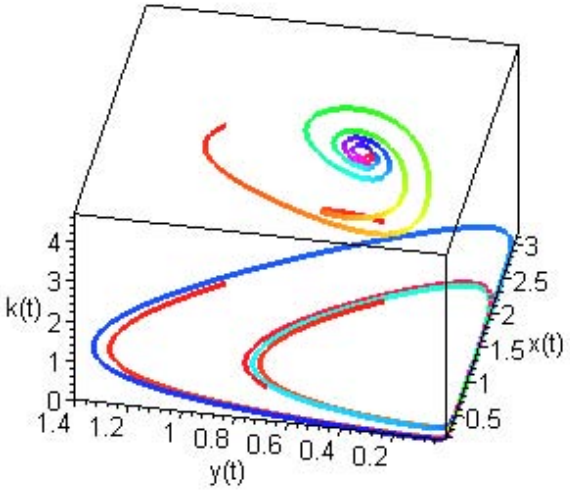

Fig. 4.3

For the case in which the net birth rate decreases with prosperity we select $m = -1$; $q = 0.7$; $r = 0.3$; $s = 0.2$; $\phi = 0.1$; $\alpha_0 = 0.02$; $\alpha_1 := -0.02$; $\alpha_2 = 1$; $\beta_0 = 0.1$; $\beta_1 := 0.3$; $\beta_2 = 1$; $\omega_0 = 0.2$; $\omega_1 := 0.2$; $\omega_2 = 1$. The net birth rate parameters are selected for comparison with the α increasing case to give a net birth rate when capital stock is zero of 4%. From (24)-(26) it is evident that only the stationary state level of the sick is affected by variation in the form of α; this is shown in the new stationary states which become

$$X^* = 1.721; Y^* = 0.1721; K^* = 4.180 \tag{41}$$

$$X^* = 1.000;\ Y^* = 0.100;\ K^* = 0; \tag{42}$$

$$X^* = 0;\ Y^* = 0;\ K^* = 0; \tag{43}$$

There is also little change in the eigenvalues of the linearisations: around the first stationary state (41) the roots are:

$$\epsilon_1 = -.007 + .007i;\ \epsilon_2 = -.007 - .007i;\quad \epsilon_3 = -0.078 \tag{44}$$

which corresponds to the case of a pair of complex conjugates and one negative real root (focus sink with positive attractor). Around the second stationary state (42) the eigenvalues are:

$$\epsilon_1 = 0.126i;\quad \epsilon_2 = -0.126i;\quad \epsilon_3 = 0.567 \tag{45}$$

Again since the derivatives of the production function are not uniquely defined as $K_t \to 0, X_t \to 0$, we do not attempt to linearise around the origin. Here the epidemiological-economic stationary state prevalence of the disease is identical to that where there are no economic effects on the epidemiological parameters; as stated above, in this case the prevalence may either rise or fall under the influence of economic effects. Total population has again increased by about 65% and in the epidemiological-economic equilibrium the net growth rate of susceptibles is 0.022. Partly these results are driven by the economic parameters; the savings rate, the depreciation rate and the elasticity of substitution are all important.

For this case where the net birth rate is a decreasing function of economic prosperity numerical integration of the nonlinear differential equations gives Fig. 4.4-4.5.

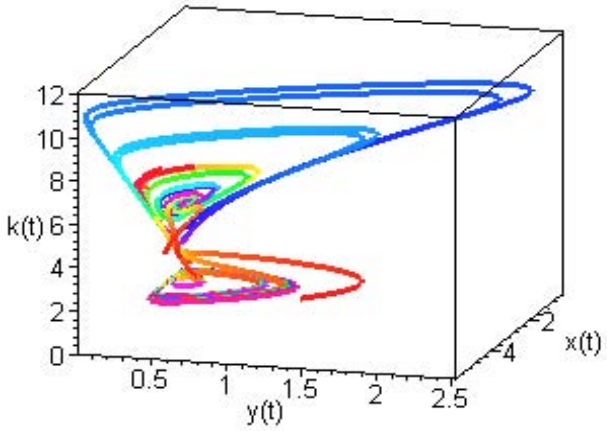

Fig. 4.4

Figure 4.4 shows the global phase space with the three stationary states; Figure 4.5 shows small regions around the stationary states other than the origin; the higher of these is a focus sink with positive attractor and the lower one is a 3D centre with an additional negative attractor. The interpretation of the dynamics is similar to the case in which the net birth rate rises with prosperity; the main difference between the two cases being that the prevalence of the disease in the first stationary state is lower here.

If we set $\beta = 0$ and $\alpha_2 = 0$ the system (23) reduces to a Solow-Swan growth model in which the healthy susceptibles grow at the exogenous rate $\alpha_0 - \alpha_1$ and the sick die out at the rate ω and have no infectious effect on the economy. If only $\beta = 0$ we have an endogenous growth model in which there is no interaction between the infected and the healthy people (i.e. segregation) and the sick again die out but the growth rate of the healthy susceptibles is endogenous with α varying with the capital healthy labour ratio. The effect of the two way interaction between the productive capacity of the economy and the epidemiological structure can be easily analysed by comparing these special cases with the epidemiological-economic stationary states. The Solow-Swan model with $\beta = 0$ and $\alpha_2 = 0$ where the healthy work force grows at a constant rate of 0.04 has a unique steady state capital labour ratio of 1.61 for the parameter values corresponding to Figure 4.1 (this solves $K/X = \left[\left[\left(\frac{\alpha_0 - \alpha_1 + \phi}{s}\right)^m - q\right]/r\right]^{1/m}$). By contrast, when only $\beta = 0$

the steady state K/X ratio is 1.413 and the common growth rate of capital and healthy labour is 0.055. The impact of rising prosperity on the net healthy growth rate leads to an increase in the steady state growth rate and a reduction in the steady state capital healthy labour ratio. In the epidemiological-economic stationary state the capital healthy labour ratio is 2.43 so that the infection process has a sufficiently strong effect in reducing the healthy work force to sharply increase the capital healthy labour ratio. Note that in the endogenous growth and Solow-Swan specialization, where $\beta = 0$ and there is segregation, in the long-run the TB prevalence is zero.

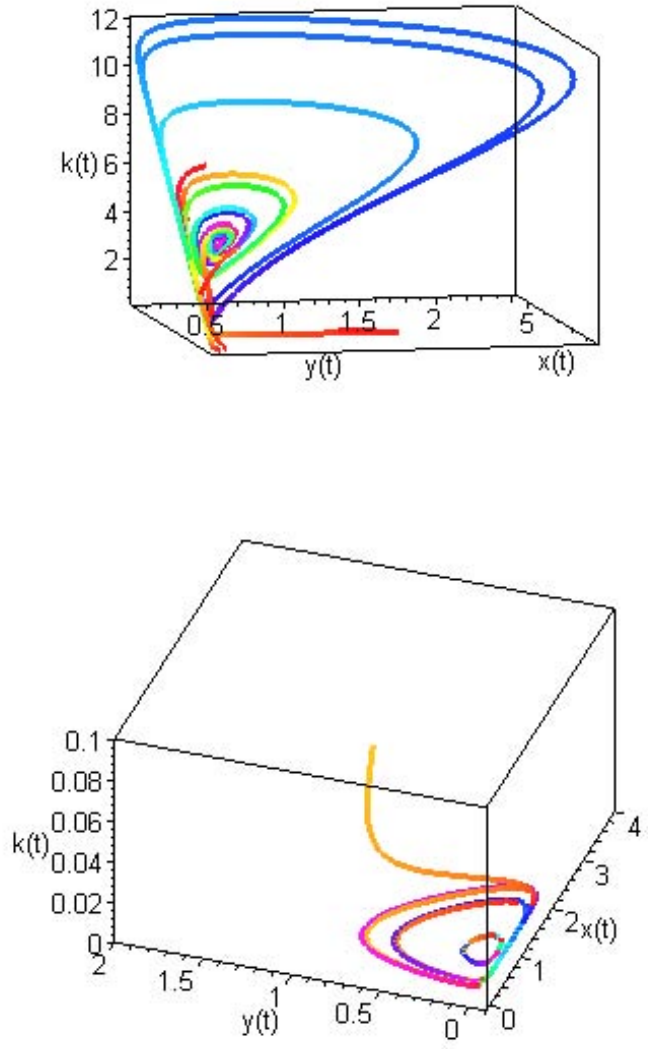

Fig. 4.5

Overall the impact of adding economic structure to the epidemiological process leads to:

(i) a new epidemiological-economic equilibrium with generally a higher healthy work force but with either a higher or lower prevalence of the disease depending on the behaviour of the epidemiological parameters;

(ii) the new equilibrium being an attractor for a wide range of initial conditions and whose dynamics pushes the system away from the epidemiological states with $K_t = 0$ towards the epidemiological-economic equilibrium;

(iii) shocks which displace the epidemiological-economic system away from its stationary state that may lead to cycles of much lower amplitude in the population structure than we would observe in the equivalent epidemiological model where $K_t = 0$.

The epidemiological equilibrium reappears in the epidemiological-economic model but as an unstable equilibrium; as well as this, the presence of disease eliminates the possibility of steady growth. However, compared with the pure epidemiological system, presence of the economy allows the system to settle down at a stationary state after some initial cyclical dynamics, thus avoiding perpetual epidemic cycles.

5. Conclusions

Much of the earlier literature on the dynamics of infectious diseases uses predator-prey type models; these are mechanistic but they allow us to understand the basic infection process. By surveying recent contributions to the analysis of TB, we show that, although there is a tension between descriptive realism and analytical tractability, the models are sufficiently flexible to accommodate some of the major interactions. Furthermore, by highlighting crucial steps in the chain of infection they allow us to focus on points at which economic forces can have an impact on the dynamics of the disease. Given this approach, our central purpose has been to lay out a framework of the two-way interactions between the disease, population structure and the economy. In an economic model with a productive capacity that depends on human inputs, the TB epidemic affects the productivity of the economic system. However, TB is partly controlled by the general level of economic prosperity; as such, even without any conscious policy initiative there are economic effects to control its spread.

We outline a model of the interaction between TB infectious disease and a dynamic economic growth model which uses capital stock and uninfected labour as inputs. We find that the dynamics are very rich: there are three stationary states, and whilst local dynamics around one of these is relatively straightforward, around the other two there is a possible diversity of behaviour. Two of the stationary states involve zero productive capital and replicate the basic predator-prey stationary states; the third is an epidemiological-economic equilibrium in which the economy and the disease coexist. When the local dynamics of the different stationary states are pasted together to give a global view of the phase space, there may be different types of transition between the different equilibria. Economic growth cannot drive the disease prevalence to zero but can make it settle down to an endemic level in the population. Using plausible parameter values in this model we find that the presence of productive capital generally pushes the system away from a pure epidemic cycle model towards the epidemiological-economic stationary state; that initially just a small injection of capital leads to a rapid rise in prosperity and shift of regime towards this new stationary state and that along the way the amplitude of cycles in the prevalence of the disease falls. At the new epidemiological-economic stationary state the prevalence of the disease may be higher or lower than in the absence of productive

capital depending on the behaviour of the epidemiological parameters.

Our initial concern was to explore the control of disease through economic growth in view of the evidence that targeted control programmes (e.g. vaccination) have limited success and the long term historical evidence that where TB has been largely eliminated, it was not achieved through targeted policy. We find that growth does have an impact but is unlikely itself to eliminate the disease. Of course the models here are stylised simplifications; in particular there is no spatial dimension so that the segregation/quarantine policies that have been historically important are not analysed.

Appendix

We can take the stationary states in turn: around (33) where $X^* = \frac{\omega}{\beta}$; $Y^* = \frac{\alpha}{\beta}$; $\left(\frac{K}{X}\right)^* = f^{-1}\left(\frac{\phi}{s}\right)$, we have

$$\begin{bmatrix} \dot{X} \\ \dot{Y} \\ \dot{K} \end{bmatrix} = \begin{bmatrix} -\left(\alpha' - \beta' Y\right)\frac{K}{X} & -\omega & \left(\alpha' - \beta' Y\right) \\ \alpha - Y\left(\beta' X - \omega'\right)\frac{K}{X^2} & 0 & \left(\beta' X - \omega'\right)\frac{Y}{X} \\ sF_X & 0 & sF_K - \phi \end{bmatrix} \begin{bmatrix} X \\ Y \\ K \end{bmatrix} \tag{46}$$

Using the fact that $F_X = \frac{F}{X} - F_K\left(\frac{K}{X}\right)$ in steady state sF_X can be written as $s\left(\frac{F}{X} - F_K\left(\frac{K}{X}\right)\right)$ in steady state. We can use a similarity transformation to deduce that the matrix in (46) has the same eigenvalues as $B = C^{-1}AC$ where

$$B = \begin{bmatrix} 0 & -\alpha\omega & -\omega\frac{Y}{X}\left(\beta' X - \omega'\right) \\ 1 & 0 & \left(\alpha' - \beta' Y\right) \\ -\frac{K}{X} & 0 & sF_K - \phi - \left(\frac{K}{X}\right)\left(\alpha' - \beta' Y\right) \end{bmatrix} = \begin{bmatrix} 0 & -\alpha\omega & b_{13} \\ 1 & 0 & b_{23} \\ -\frac{K}{X} & 0 & b_{33} \end{bmatrix} \tag{47}$$

and

$$C = \begin{bmatrix} 0 & 1 & 0 \\ -\frac{1}{\omega} & 0 & 0 \\ 0 & \frac{K}{X} & 1 \end{bmatrix}, C^{-1} = \begin{bmatrix} 0 & -\omega & 0 \\ 1 & 0 & 0 \\ -\frac{K}{X} & 0 & 1 \end{bmatrix} \tag{48}$$

Note that under our sign assumptions on the effects of growth on demographic functions, if $\alpha' > 0$, b_{13} is of ambiguous sign; $b_{23} > 0$ and $b_{33} < 0$. The characteristic equation of B has the form

$$f(\mu) = -\mu^3 + b_{33}\mu^2 - \left(\frac{K}{X}b_{13} + \alpha\omega\right)\mu + \alpha\omega\left(b_{33} + \frac{K}{X}b_{23}\right) \tag{49}$$

It is somewhat more convenient to change sign and consider the cubic equation $g(\mu) = -f(\mu) = 0$. Since $\alpha\omega(b_{33} + \frac{K}{X}b_{23}) = sF_k - \phi < 0$, there are no zero real roots. If $b_{33}^2/3 - (\frac{K}{X}b_{13} + \alpha\omega) \leqslant 0$, this cubic has no turning points and since it tends to $\pm\infty$ as μ does, it must then be always increasing hence giving a single real root. Moreover at $\mu = 0$, the cubic has a positive value; hence this single real root must be negative. Exploring this a bit further,

$$b_{33}^2/3 - \left(\frac{K}{X}b_{13} + \alpha\omega\right) = [sF_K - \phi - \frac{K}{X}\left(\frac{\alpha'}{\alpha} - \frac{\beta'}{\beta}\right)\alpha]^2/3 + \alpha\omega\left[\frac{K}{X}\left(\frac{\beta'}{\beta} - \frac{\omega'}{\omega}\right) - 1\right] \tag{50}$$

so if

$$\frac{K}{X}\left(\frac{\beta'}{\beta} - \frac{\omega'}{\omega}\right) - 1 \tag{51}$$

is sufficiently negative then we are sure there is a single negative real root.

However, if $b_{33}^2/3 - (\frac{K}{X}b_{13} + \alpha\omega) > 0$, then there are two turning points, μ_+ and μ_-, with $\mu_+ > \mu_-$. If $\alpha' > 0$ we know that $\mu_- < 0$ (since the derivative of the cubic is a

quadratic with a minimum at $\mu = b_{33}/3 < 0$). We also know that the cubic is decreasing between its turning points $g(\mu_+) \leqslant g(\mu_-)$. Then if $g(\mu_+)\ g(\mu_-) > 0$ so that the cubic does not change sign between its turning points we know that there is also a single real root which must be negative (Fig. A.1).

For $\alpha' > 0$ the condition for $g(\mu_+)\ g(\mu_-) > 0$ is that

$$\left[b_{33}\left(-2b_{33}^2 + 9b_{13}\frac{K}{X}\right) - 9\left(2b_{33} + 3\frac{K}{X}b_{23}\right)\alpha\omega\right]^2 > \left(b_{33}^2 - 3\left(\frac{K}{X}b_{13} + \alpha\omega\right)\right)^3 \quad (52)$$

But if the cubic does change sign between its turning points from positive at μ_- to negative at μ_+, then there are three negative real roots if $\mu_+ < 0$ and two positive and one negative real root if $\mu_+ > 0$ (Fig. A.2). The condition for $\mu_+ > 0$ is that $b_{13}K/X + \alpha\omega < 0$.

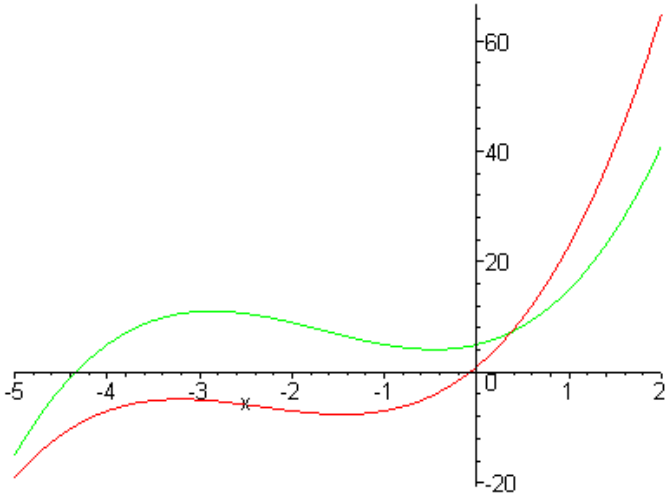

Figure A.1

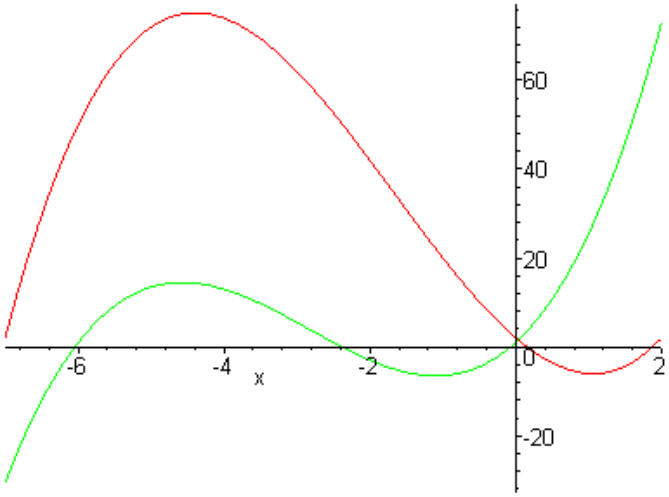

Figure A.2

If $\alpha' < 0$ then we may have $b_{33} > 0$; then when there are two turning points we know

that $\mu_+ > 0$ since μ_+ is above the value of μ which minimises the slope of the cubic (at $b_{33}/3$). It then again follows that there is either a single negative real root or two positive and one negative real roots. A summary is given in Table 1.

At the secondary stationary point (34) we have $F(K,X) = 0$ since $K^* = 0$ but X^* and Y^* are nonzero; hence $\frac{F(K,X)}{X} = 0$ and F_K has a finite value under our assumptions. From homogeneity of degree one we have $F = F_X X + F_K K$ and so at the stationary point we have $F_X = 0$ (since $K^* = F = 0$ but X^* is nonzero). Hence the linearised system becomes

$$\begin{bmatrix} \dot{X} \\ \dot{Y} \\ \dot{K} \end{bmatrix} = \begin{bmatrix} 0 & -\omega & \left(\alpha^{'} - \beta^{'} Y\right) \\ \alpha & 0 & \left(\beta^{'} X - \omega^{'}\right) \frac{Y}{X} \\ 0 & 0 & sF_K - \phi \end{bmatrix} \begin{bmatrix} X \\ Y \\ K \end{bmatrix} \tag{53}$$

which has eigenvalues $sF_K - \phi$ and $\pm i\sqrt{\alpha\omega}$

At the third stationary point (35) when $X^* = Y^* = K^* = 0$ we have $F(K,X) = 0$; and we assume that both F_X and F_K have large but finite values. Taking X_t Y_t and $K_t \to 0$ along the 45^o ray, then by L'Hôpitals rule $\frac{F}{X}$ approaches F_X; $\frac{Y}{X}$ approaches unity and so $F\frac{Y}{X^2}$ approaches F_x. Hence the linearised system has the form

$$\begin{bmatrix} \dot{X} \\ \dot{Y} \\ \dot{K} \end{bmatrix} = \begin{bmatrix} \alpha & 0 & \alpha^{'} \\ \omega' & -\omega & -\omega^{'} \\ sF_X & 0 & sF_K - \phi \end{bmatrix} \begin{bmatrix} X \\ Y \\ K \end{bmatrix} \tag{54}$$

and this matrix has eigenvalues

$$-\omega \tag{55}$$

$$0.5\left\{[sF_K - \phi + \alpha'] \pm \sqrt{[\alpha - sF_K - \phi]^2 + 4sF_X\alpha' - 4sF_K\phi + 4\alpha\phi}\right\} \tag{56}$$

These are real roots if

$$[\alpha - sF_K + \phi]^2 + 4sF_X\alpha' - 4sF_K\phi + 4\alpha\phi > 0 \tag{57}$$

When $\alpha^{'} > 0$ and $sF_K - \phi > 0$ at $K^* = 0$,

$$[\alpha - sF_K - \phi]^2 + 4sF_X\alpha' - 4sF_K\phi + 4\alpha\phi > [\alpha - sF_K + \phi]^2 \geq 0 \tag{58}$$

This gives us a lower bound on the upper root of (56) $(\alpha - \phi)$ and a nonnegative upper bound on the lower root of sF_K. Hence, if $\alpha < \phi$ both roots are negative but for $\alpha > \phi$ we only know that one root is positive. Overall, the third stationary point has a wide variety of local dynamic patterns shown in Table 2.

$b_{33}^2/3-(\frac{K}{X}b_{13}+\alpha\omega)>0$	$[b_{33}(-2b_{33}^2+9b_{13}\frac{K}{X})-9(2b_{33}+3\frac{K}{X}b_{23})\alpha\omega]^2>(b_{33}^2-3(\frac{K}{X}b_{13}+\alpha\omega))^3$		focus sink with positive/negative
$b_{33}^2/3-(\frac{K}{X}b_{13}+\alpha\omega)\leq 0$			3D centre with positive attractor
$b_{33}^2/3-(\frac{K}{X}b_{13}+\alpha\omega)>0$	$[b_{33}(-2b_{33}^2+9b_{13}\frac{K}{X})-9(2b_{33}+3\frac{K}{X}b_{23})\alpha\omega]^2<(b_{33}^2-3(\frac{K}{X}b_{13}+\alpha\omega))^3$	$\frac{K}{X}b_{13}+\alpha\omega>0$	3D node with positive attractor
$b_{33}^2/3-(\frac{K}{X}b_{13}+\alpha\omega)<0$	$[b_{33}(-2b_{33}^2+9b_{13}\frac{K}{X})-9(2b_{33}+3\frac{K}{X}b_{23})\alpha\omega]^2<(b_{33}^2-3(\frac{K}{X}b_{13}+\alpha\omega))^3$	$\frac{K}{X}b_{13}+\alpha\omega>0$	3D saddle with stable direction

Table 1

$\alpha + sF_K - \phi < 0$	$[\alpha - sF_K + \phi]^2 + 4sF_X\alpha' - 4sF_K\phi + 4\alpha\phi < 0$	focus sink positive attractor
$\alpha + sF_K - \phi > 0$	$[\alpha - sF_K + \phi]^2 + 4sF_X\alpha' - 4sF_K\phi + 4\alpha\phi < 0$	focus sink negative attractor
$\alpha < \phi$	$[\alpha - sF_K + \phi]^2 + 4sF_X\alpha' - 4sF_K\phi + 4\alpha\phi > 0$	3D node positive attractor
$\alpha > \phi$	$[\alpha - sF_K + \phi]^2 + 4sF_X\alpha' - 4sF_K\phi + 4\alpha\phi > 0$	3D saddle and stable/unstable direction

Table 2

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