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by

Doriana Delfino and Peter J Simmons

Department of Economics and Related Studies
University of York
Heslington
York, YO10 5DD

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Delfino D. and Simmons P. J.

University of York
Department of Economics and Related Studies
E-mail: dd109@york.ac.uk ps1@york.ac.uk

Abstract. We use a simple Lotka-Volterra model of the disease transmission process to analyse the dynamic population structure when a vaccine is available at a constant price through time which gives partial immunity to the disease. In contrast to earlier results for the full immunity case, we find that there may be multiple stationary states and instability. In contrast to earlier work which has only considered policies in steady states, we consider the dynamic effects of different dynamic vaccination policies on any solution path for the case of publicly subsidised vaccines. We find that in the partial immunity case a procyclical policy is desirable but for the full immunity case a countercyclical policy is desirable.

Infectious diseases have been and are undoubtedly economically and socially costly; e.g. in the UK in the 19th century 30% of deaths were caused by typhoid, tuberculosis (TB) and typhus; the World Health Organisation [17] estimates now that tuberculosis causes 3 million deaths and 8 million new infections per annum. Vaccination against such diseases can either work to give virtually certain immunity immediately following vaccination or can work to reduce the chance of infection. For example vaccines against polio, tetanus and diptheria appear to give certain immunity, although as with most vaccines, the degree of protection falls with time since vaccination. However, vaccination against cholera or malaria is problematic and vaccination against hepatitis B leaves 10-15% of middle aged males unprotected [5]. For analytical clarity, in this paper we classify vaccines as either giving certain and *permanent* immunity or as providing a reduction in the chance of infection.

If vaccines are provided in a market system then the individual incentive to purchase the vaccine is driven by the trade-off between its cost and the better life chances that vaccination offers. The higher the chance of infection and the greater the cost of being infected, the greater the willingness. However, to pay for the vaccine. It follows that the market demand for vaccination is sensitive to the risk of infection which itself is generally modelled as increasing with the prevalence of the disease in the population. With a heterogeneous population e.g. in incomes the aggregate demand for the vaccine will generally be a continuous function of the prevalence of the disease. However, a publicly provided vaccine will have effects on control of the disease which depend on the form of the vaccination programme. Is the vaccine offered in unlimited supply at every instant or is it offered in limited amounts either in time varying or time constant quantities? If the vaccine offers permanent immunity, is offered free to all and is taken up by all those at risk, then the disease can be most speedily eliminated. Typically the vaccine cost and the opportunity cost of public funds prevent this. The question is: with finite resources to fund vaccination, what time profile of its application is best?

In this paper we analyse the effects of market provided vaccines which offer partial immunity to the disease through decreasing the chance of infection. In this scenario we look at the stationary equilibria and also at the dynamics of the population structure. We also analyse the dynamic effects of dynamic public vaccination policies for both the

cases of vaccines giving sure, immediate immunity and vaccines giving partial immunity. Throughout, the dynamic population structure is governed by a variant of the Lotka-Volterra type predator-prey model.

Geoffard and Philipson [8] give a seminal analysis of the interaction between market provided vaccination programmes and a Lotka-Volterra type predator prey model of the population dynamics. Their paper focuses only on steady states in a model where vaccination gives permanent and certain immunity and in this context looks at the limits of private market solutions as compared with public subsidies for vaccination. They show that eradication of the disease is unlikely to be achieved either under a market system for delivering vaccination or under a public subsidy system.

By contrast we look at the behaviour of the population structure along nonstationary paths, mainly emphasising the case where vaccination changes the chance of subsequent infection. As we discuss in the sequel, the sort of disease we have in mind is TB where the effects of vaccination are quite uncertain depending on the nature of the disease and the social infrastructure[1]. This view of the uncertainty of the effects of preventive activity is closer to Geoffard and Philipson [7] although in that paper the emphasis is not on preventive policy.

The aim of this paper is to extend the steady state policy analysis to allow for dynamic policy in contexts that include both partial and full immunity; in addition, we explore how the dynamics of the disease varies with variations in the specification of the disease transmission process.

In Section 1 we outline the disease model; we use a slightly different demographic characterisation than Geoffard and Philipson [8] (in particular distinguishing only two health states) partly to avoid the curse of dimensionality in analysing the dynamics; in Section 2 we analyse the dynamics of the disease in a market setting; in Section 3 we look at regulatory solutions to disease control including targeted regulatory action.

The results indicate that when vaccination only offers partial immunity to infection, a market provided vaccine at a constant price will lead to choices of vaccination by individuals which may generate an additional stationary state for the population structure instead of the two stationary states which exist without vaccination. We give an example which has three stationary points, two of which are saddle points and the third a stable focus. The global phase space reveals that in this example the population structure will tend to settle down to either a stable low healthy/low disease level or will involve growth in both the numbers of healthy and sick.

However, if a dynamic subsidy policy is used to regulate vaccination then we find that in the case of partial immunity, a procyclical policy, vaccinating at instants when prevalence is high, is preferable to either a low prevalence policy or a constant vaccination policy. In the case of vaccination giving permanent immunity to infection and again considering dynamic subsidy policies, we find that a low prevalence subsidy policy is best. This result holds both in demographic dynamics used in the bulk of our analysis and in the demographic dynamics used by Geoffard and Philipson.

1. The Disease Process

We think of a population N_t of individuals in a given area at instant t. Individuals can be in one of two health states: susceptible but healthy, actively infected and infectious. Some epidemiological models distinguish many more states than this e.g. Geoffard and Philipson [8] allow for four states (susceptible, infected, recovered and out of the system);

[1] IUATLD [9], Weatherall [14], WHO [15],[16].

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), there may be five states (susceptible, latent slow infected, latent fast infected, active infected, recovered). The nature of recovery can also be heterogeneous: infected individuals who have recovered either may have permanent immunity from the disease forming a class of their own or may immediately become susceptible to a new attack of the disease joining the existing group of susceptibles[2]. The population changes through time due to the births of susceptibles (one cannot be born either a latent or active infected individual; nor as a recovered individual) and to deaths either from natural old age 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. To some extent this was due to the particular parasitic transmission mechanism; but partly it was due to the type of dynamic interaction seen in the very simplest predator-prey models which we use here. So a very common paradigm for modelling the disease dynamics is a simple version of the Lotka-Volterra system which ignores latents and the recovered:

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

$$\begin{cases} \dot{X}_t = \alpha X_t - \beta(X_t, Y_t) X_t Y_t \\ \dot{Y}_t = \beta(X_t, Y_t) X_t Y_t - \omega Y_t \end{cases} \tag{2}$$

where X_t is the stock of susceptibles at time t and Y_t is the stock of actively infected at t. Furthermore, α is the net birth rate of susceptibles (birth rate minus death rate due to non-disease causes); $\beta(X_t, Y_t)$ is the probability that on meeting an actively infected person, a susceptible person becomes latently infected; and ω is the death rate of the actively infected whether through the disease or natural causes. Assuming that (α, ω) are constant proportions is a simplification.

A major issue is the interpretation of the infection process $\beta(X_t, Y_t) X_t Y_t$. 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 at instant t:

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

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, under random matching, 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

[2]Chan-Yeung [3], Comstock [4], IUATLD [9], Weatherall, D. J. et al. [14].

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[3]. In the sequel we discuss the robustness of our conclusions to different epidemiological characterisations.

If we adopt any of these assumptions, then β is a constant. From these equations it follows that total population changes according to

$$\dot{N}_t = \dot{X}_t + \dot{Y}_t = \alpha X_t - \omega Y_t \tag{3}$$

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:

$$X^* = Y^* = 0 \tag{4}$$

$$X^* = \omega/\beta, Y^* = \alpha/\beta \tag{5}$$

The first corresponds to extinction and the second to a constant population level and structure. There are no steady growth paths of the system i.e. no paths along which total population is growing at a constant rate but the population structure is constant. Essentially this is because the differential equations are not homogeneous of degree one in the levels of the variables due to the product term $X_t Y_t$. Another way of putting it is that if the population initially doubles in each class (X_t, Y_t) the number of new infections quadruples; there is a built in tendency for more populous societies to face larger fluctuations in the health structure of the population.

As is well known, the latter stationary point (6) has two pure imaginary roots so long as $\alpha > 0$ so that there are closed cycles about this stationary point.

Notice that if $\alpha \leq 0$ 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. It is less well known that the origin is locally a saddle point. A typical phase diagram is shown in Fig. 1.

2. Decentralised Control: Market Solutions

Vaccination is a key preventive device. With market provision, a preventive device is available at a price p at time t. Such devices may either give permanent immunity to the disease in which case protected individuals drop out of the susceptible class; or may work by reducing β, the risk of infection. Geoffard and Philipson [8] analyse the situation in which vaccination of a susceptible gives certain and permanent immunity to the disease; in the absence of regulation there is a private demand for vaccination $D(p, Y_t)$ which depends on the constant price of the vaccine and the number of infected individuals in the economy. Their basic demographic system includes immune individuals who have been vaccinated and also a version of recovery in which some of the actively infected develop immunity. In contrast to our approach, the immune are no longer susceptible to the disease. They examine the stationary state of the system (unique in their model) with an exogenously given constant path of prices; they find that their system exhibits local stability of the nonzero stationary state rather than cycles.

[3]Geoffard and Philipson [8] treat X_t, Y_t as proportions of a varying population through having an extra nonmodelled class - those out of the system - which is another way of handling the issue.

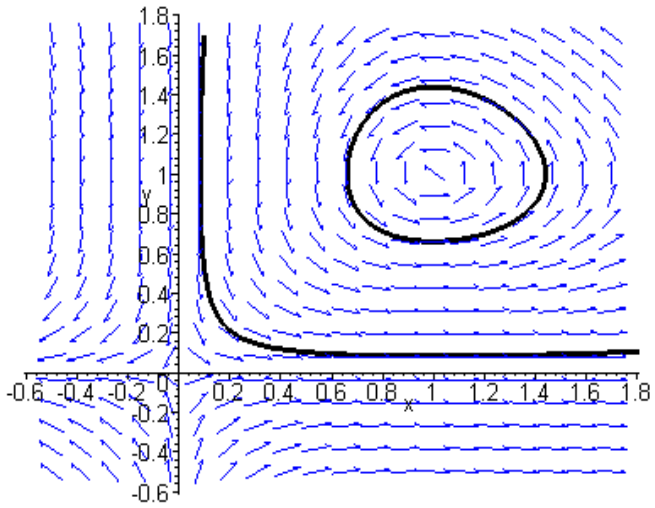

Figure 1:

The Geoffard-Philipson model [8] works through preventive action (vaccination) giving permanent immunity to susceptibles; in many cases this is fairly extreme. For example TB does not seem to fit this pattern. Two main forms of TB exist: pulmonary TB and extra-pulmonary TB, the former is most common and is the only infectious form. In the case of TB, BCG vaccination has only a limited effect on controlling the spread of infectious TB since, firstly, it prevents the non-infectious extra-pulmonary TB rather than infectious TB (IUALTLD [9]) and secondly, in developing countries environmental conditions may prohibit its efficacy (Madras Tuberculosis Institute Bangalore[11]).

So instead we take vaccination to reduce but not eliminate the chance of infection. In this case for each susceptible there would be a different level of β depending on whether that susceptible has been vaccinated. There are two levels of β, β_H and β_L ($\beta_H > \beta_L$). Individual choice of vaccination or not is based on utility maximisation: each susceptible individual i has income m_i that can be spent on consumption c_i or on vaccination at a relative price of p. For the ith susceptible if $u(h_i, c_i)$ represents utility with health state h_i (h_i is either infection I or susceptibility S) and is assumed strictly concave and increasing in c_i, vaccination expected utility of a susceptible i who has constant income m_i and has vaccinated is

$$\beta_L Y_t u(I, m_i - p) + (1 - \beta_L Y_t) u(S, m_i - p) \tag{6}$$

without vaccination at t or earlier it is

$$\beta_H Y_t u(I, m_i) + (1 - \beta_H Y_t) u(S, m_i) \tag{7}$$

Vaccination costs forgone consumption but gives more favourable odds between the good and bad state. Susceptible i will vaccinate if he gains expected utility from doing so; if all susceptibles are very poor ($m_i < p$ for all i) then none can afford vaccination anyway. If p were close to zero then all would vaccinate since it would have a negligible cost in terms of consumption but would improve the chance of the good state. Also the

difference in expected utilities between vaccination or not is monotone in p. Hence there exists a critical price of the vaccine $p = P(Y_t, m_i)$ which makes i just indifferent between vaccination or not. At $p < P(Y_t, m_i)$ i vaccinates and faces β_L; otherwise he does not vaccinate and faces β_H. Similarly if $Y_t = 0$ then no one will vaccinate since there is no risk of infection (so for $p > 0, \beta(p, 0) = \beta_H$). Let $\gamma(p, Y_t)$ be the proportion of susceptibles whose income is high enough to choose vaccination. This will be decreasing in p and increasing in Y_t. The average level of β is

$$\beta(p, Y_t) = \beta_H(1 - \gamma(p, Y_t)) + \beta_L \gamma(p, Y_t) \tag{8}$$

The number of new infections is then $\beta(p, Y_t)X_tY_t$ where $\beta(p, Y_t)$ is decreasing in Y_t and increasing in p. The population structure evolves according to

$$\begin{cases} \dot{X}_t = \alpha X_t - \beta(p_t, Y_t)X_tY_t \\ \dot{Y}_t = \beta(p_t, Y_t)X_tY_t - \omega Y_t \end{cases} \tag{9}$$

If prices and incomes are constant through time then effectively we can write $\beta = \beta(Y_t)$[4]. The origin is one stationary state of (9). There will generally be other stationary states; If the elasticity of β is globally less than -1 then $\beta(Y_t)Y_t$ is monotonically decreasing and so there is at most a single nonzero stationary point solving

$$\alpha = \beta(p, Y^*)Y^*; \quad \omega = \beta(p, Y^*)X^*$$

However since at $Y = 0$, $\beta(p, Y)Y = 0$ whilst at $Y = N - 1$, $\beta(p, Y)Y > 0$, it is unlikely that $\beta(Y)Y$ will be decreasing. Otherwise, there may be more than one non-zero stationary state each solving $\alpha = \beta(p, Y^*)Y^*$(yielding Y^*) and $\omega = \beta(p, Y^*)X^*$ (which then gives X^*)[5]. Generally the prevalence dependence of β will affect the stability of the system. If $\partial\beta/\partial Y < 0$ in the neighbourhood of non-zero stationary state, then locally the stationary state has at least one direction of stability (the trace of the Jacobian of the dynamical system evaluated at the nonzero-stationary point is $\omega\partial \ln \beta/\partial \ln Y < 0$). If locally the elasticity of β with respect to Y is less than -1, then locally it also has a direction of instability and is a saddle (the sign of the determinant is that of $[\partial \ln \beta/\partial \ln Y + 1]$). This is in contrast to the Geoffard and Phillipson [8] model[6] in which the unique non-zero stationary state is locally stable. Since each individual neglects the risk of future infection which he imposes on other susceptibles through not vaccinating,

[4]Alternatively we could derive the price and prevalence dependence of β from a dynamic programming approach as Geoffard and Philipson [8] do. If vaccination at any t gives a permanent change in risks of infection then we can interpret the utilities in lifetime terms; from t onwards let

$$V_t(Y_t, v) = \beta_L Y_t u(I, m_i - p) + (1 - \beta_L Y_t)u(S, m_i - p) + V_{t+1}(Y_{t+1}, v) \tag{10}$$

$$V_t(Y_t, nv) = \beta_H Y_t u(I, m_i) + (1 - \beta_H Y_t)u(S, m_i) + \max\{V_{t+1}(Y_{t+1}, v), V_{t+1}(Y_{t+1}, nv)\} \tag{11}$$

be the value functions of a susceptible who has not vaccinated prior to t and who respectively decides to either vaccinate $V_t(Y_t, v)$ or not vaccinate $V_t(Y_t, nv)$ in t. Here i vaccinates in t if $V_t(Y_t, v) > V_t(Y_t, nv)$. This comparison will again give us a critical income level defined in terms of the vaccine price and the current prevalence, together with expected future prevalences and future economic variables at which a susceptible is just indifferent between vaccination or not.

[5]There may be several solutions to the equation $\alpha = \beta(Y)Y$. If $\beta(0) > 0$ and the elasticity of $\beta(Y)$ with respect to $Y < -1$ then there is a unique solution since then $\beta(Y)Y$ is decreasing. It is plausible that $\beta(Y)Y$ has a minimum in which case there are likely to be at least two interior solutions for Y.

[6]It follows that the dynamic pattern is not robust to the epidemiological model. If for example we used the Geoffard and Philipson [8] model of demographics we would have

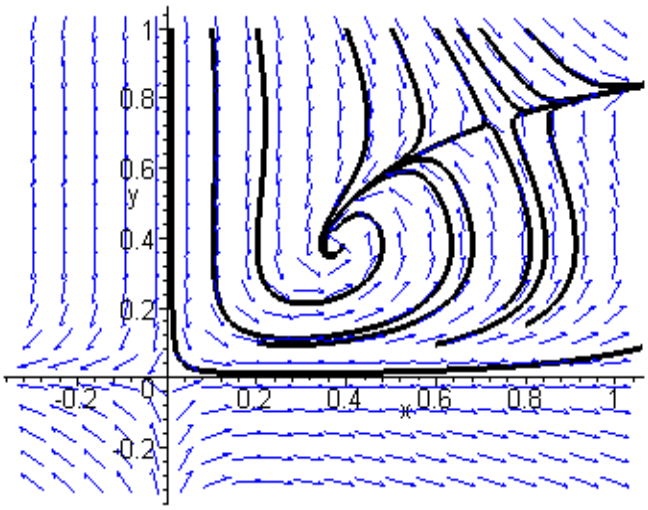

Figure 2:

the results are not Pareto optimal. Issues of market failure arising from this externality are discussed in Brito et al. [2].

To illustrate some of the dynamic possibilities with multiple stationary points we present an example which numerically integrates the nonlinear differential equations; this means the phase spaces are globally accurate; the linear approximations would just give us the local dynamics in the vicinity of the different stationary states.

To show this, in (9) we select $\alpha = 0.05$, $\beta_H = 0.2$, $\beta_L = 0.1\beta_H$, $\gamma(p, Y_t) = Y_t$ [7] and $\omega = 0.05$. This has three stationary points at $X^* = Y^* = 0$ which is a saddle point, $X^* = Y^* = 0.38$ (which has a stationary state level $\beta(p, Y) = 0.13$), which is a convergent focal point and $X^* = Y^* = 0.73$ (which has a stationary state level $\beta(p, Y^*) = 0.07$) which is also a saddle point. The eigenvalues corresponding to the stable focus are $-0.13 \pm 0.32i$; around the saddle point with $X^* = Y^*$ positive, the eigenvalues are 0.02, -0.12.

The global view of the phase space for these parameter values is in Fig. 2.

In this example the effect of marketed vaccination is to yield a system with three stationary states rather than the two stationary states in the basic Lotka-Volterra demographic system. In the vaccination model, there are asymptotically five types of behaviour for the population structure. It may tend to the stable focus or converge along the stable separatrix to the higher saddle point (if the initial conditions are on the stable separatrix). It may diverge away from the higher saddle point with both X_t and Y_t growing or travel down the vertical axis (the stable separatrix of the origin) or move outwards from the origin along the horizontal axis (the unstable separatrix of the origin). Which of

$$\begin{bmatrix} -\beta Y & -(\beta' Y + \beta)X \\ \beta Y & \beta' XY \end{bmatrix} \tag{12}$$

for the Jacobian of the dynamic system. The determinant of this is $\beta^2 XY$ which will generally be positive.

[7] Taking $\beta(p, Y)$ linear in Y is an approximation; the actual form used is consistent with various relations between β_H, β_L and the locally linear prevalence dependence.

these events occurs depends on the initial conditions. The effect is that either there will ultimately be a stable population with a constant structure or total population will be growing but with the numbers of healthy rising faster than the numbers of sick. In this last case the system follows an approximately linear path in the $X - Y$ plane.

3. Regulatory Policy

Policy can act either through targeted programmes on cure or prevention of the disease or indirectly through seeking to raise the level of prosperity of the economy. The historical evidence is that for some diseases where the risk of infection varies with the general level of health of susceptibles, raising general economic prosperity may be important (of course it gives other benefits as well). However, most contemporary interest is in targeted programmes of prevention either through education (e.g. for sexually transmitted diseases) or through vaccination. If there is an effective vaccine providing permanent and sure immunity then providing vaccine free to all, and ensuring that it is taken up by all, will eliminate the disease as susceptibles will always choose to take a vaccine offered at zero cost. Doing this may be prohibitively costly in which case the question of the most effective vaccination policy arises. Geoffard and Philipson [8] consider the effect on the steady state of their model of a continuous constant price subsidy to the vaccine. In contrast we examine the dynamic effects of dynamic rules for applying a subsidy on any solution path. Here, the issue we wish to focus on is the optimal timing of the vaccine. In the scenarios we envisage above, all susceptibles are medically identical so on medical grounds there is no reason to distinguish them. However, a given public budget for vaccination may have quite different effects if it is all spent at once either in a period with high prevalence giving a shift in the aggregate risk of infection in the period in which it is administered or in a period with low prevalence or if it is spent at a constant rate through time.

We continue to assume

$$\begin{cases} \dot{X}_t = \alpha X_t - \beta X_t Y_t \\ \dot{Y}_t = \beta X_t Y_t - \omega Y_t \end{cases} \tag{13}$$

and think of the partial immunity case as that in which vaccination works to give a step change in β when it is applied and (similarly to Geoffard and Phillipson [8]) the permanent immunity case where vaccination works by reducing the number of susceptibles at any instant where it is applied.

As examples we take three cases:

(i) the vaccine is administered along any path satisfying (13) only in periods of low prevalence when $Y_t < (\alpha/\omega)X_t$;

(ii) the vaccine is administered along any path satisfying (13) only in periods of high prevalence when $Y_t > (\alpha/\omega)X_t$[8];

(iii) the vaccine is administered at a constant rate independently of prevalence.

3.1. The Partial Immunity Case. With the vaccine giving partial immunity, the effect is to alter β. The idea is that susceptibles may either be vaccinated in which case they face β_L or not in which case the infection risk is β_H. If X_v and X_{nv} are respectively the numbers of vaccinated and nonvaccinated susceptibles, we can define the average infection rate β by

[8]As will be clear from the subsequent dynamic analysis, this particular definition of high and low prevalence is not crucial to the results; what matters is that the degree of prevalence is defined in terms of Y/X.

$$\beta = \beta_L \frac{X_v}{X_v + X_{nv}} + \beta_H \frac{X_{nv}}{X_v + X_{nv}} \tag{14}$$

To define an idea of equivalent shifts in β we assume there is a fixed lump sum budget of M and an interest rate of r. The budget can either be spent all in one period: if spent in period t, $e^{rt}M$ is available; if spent at a constant rate, then per period M/r can be spent; if spent at a constant rate, K over the interval $[T_1, T_2]$ e.g. corresponding to a sequence of periods of high prevalence an amount

$$K = (e^{rT_2} - e^{rT_1})/[r(T_2 - T_1)] \tag{15}$$

is available. Generally β_t is some decreasing function of m_t, vaccine spending in instant t.

For given funds continuous vaccination gives a lower effect on β at each instant than intermittent bouts of vaccination at the instants of vaccination. So if we can show that a given change in β at instants of vaccination is preferable if β is adjusted intermittently rather than continuously, then we are sure that intermittent is better than continuous vaccination. Any vaccination policy of this form shifts the nonzero stationary point along the ray $Y = \alpha/\omega X$ increasing both X^* and Y^* by shifting from β to a lower value $\bar{\beta}$.

To analyse intermittent vaccination consider a "high prevalence" vaccination policy where vaccination is undertaken whenever $Y_t > \alpha/\omega X_t$. The effect is that, in some parts of the region where $Y_t > \alpha/\omega X_t$, the gradient field changes when the policy switches on. In the region defined by $\alpha/\bar{\beta} > Y_t > \alpha/\beta$ and $\omega/\beta < X_t < \omega/\bar{\beta}$ the direction switches from one of rising Y_t and falling X_t In the region defined by $\alpha/\bar{\beta} > Y_t > \alpha/\beta$ and $X_t < \omega/\beta$ the direction of movement switches from one of falling X_t and Y_t to one of falling Y_t and rising X_t. When $\omega/\beta < X_t < \omega/\bar{\beta}$ and $Y_t > \alpha/\bar{\beta}$ the direction switches from increasing Y_t and falling X_t to one of falling Y_t and falling X_t. Combining these changes with the direction of movement in other areas of the phase space gives the final result of the high prevalence policy (Fig. 3). The effects are that the ray $Y = \alpha/\omega X$ develops some stability properties; on a path which approaches the ray at a point between ω/β and $\omega/\bar{\beta}$ the policy switches force the path to oscillate in a small neighbourhood of the ray with the policy continuously being switched on and off. Effectively the policy has eliminated the epidemic cycle in the original path. However, on a path which approaches the ray at $X_t < \omega/\beta$ there may initially be an oscillatory period before the path again settles down in a small neighbourhood of the ray. So depending on the initial conditions the high prevalence policy leads to a nearly stationary population structure in the long run with a ratio α/ω of sick.

A constant policy for the same cost will give a constant $\tilde{\beta}$ with $\beta > \tilde{\beta} > \bar{\beta}$. For the same initial condition the permanent fall in β switches the system from a low amplitude cycle around the original stationary point to a new high amplitude cycle around the new higher population level stationary point. The policy has actually increased the fluctuations in the system. Fig. 4 shows a closed cycle in the pre-policy phase together with a closed cycle in the post-policy phase; if the policy is introduced when the system is at a point like A, then for ever after the system follows the new closed cycle starting at A.

We could also consider a low prevalence policy; this might be thought sensible if a big push when the disease is unimportant can actually eliminate it. The idea is to vaccinate when $Y_t < \alpha/\omega X_t$. Similar consideration of the gradient field shows that this policy will be destabilising leading to an unstable spiral that is outside both stationary points. Fig. 5 portrays such an unstable path.

So for the same economic cost the high prevalence policy appears preferable: it eliminates fluctuations leading to a near constant population structure and the system settles down to a population level that depends on the initial conditions. That is with vaccination working through β the procyclical policy affects the whole dynamic path of the population favourably.

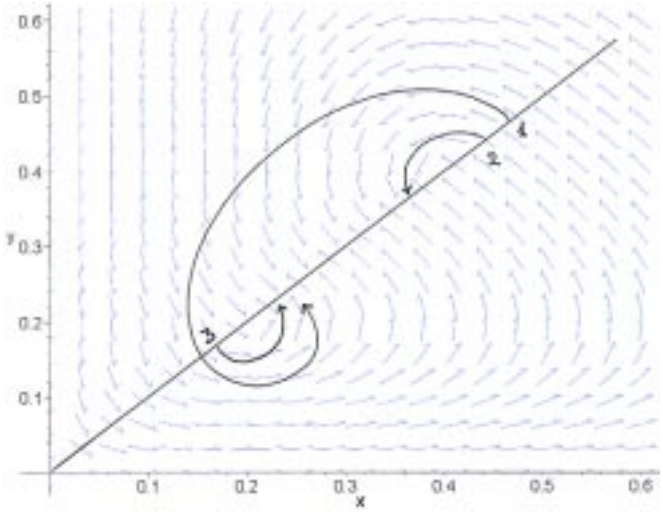

Figure 3:

3.2. The Full Immunity Case. Where the vaccine gives permanent immunity Geoffard and Phillipson examine the steady state effect of a public subsidy on the price of a market provided vaccine. They find that since the steady state prevalence of the disease is increasing with the price, an increase in the steady state subsidy (and so a decrease in the price) will have a direct effect in raising steady state demand for the vaccine but, since it reduces steady state prevalence, an indirect effect in reducing demand via prevalence. In our framework a relatively simple way of modelling the permanent immunity case is to assume that, when vaccination policy is in force, some of the net growth of susceptibles is diverted into immune individuals i.e. the policy works through reducing α. Without the policy the net growth of susceptibles is α; with the vaccination policy it is $\bar{\alpha} < \alpha$. The effect is that when the vaccination programme is active, the system has a stationary state that is vertically below that corresponding to inactive vaccination (i.e. $\bar{Y}^* = \bar{\alpha}/\beta < \alpha/\beta = Y^*$) as in Fig. 6. When the policy is active the system is following orbits around the lower stationary state; when inactive it follows orbits around the higher stationary state.

If we apply this policy in periods of high prevalence, again defined as $Y_t > \alpha/\omega X_t$, the effect is to create an unstable spiral. Starting from a path with the policy off, as soon as the ray $Y_t = \alpha/\omega X_t$ is reached, the path switches to an orbit around the new stationary point. The new orbit intersects the α/ω ray closer to the origin than the original orbit thus increasing the amplitude of movement[9]. On reaching the ray again from above, the

[9]By defining $w(\tau) = \beta/\omega x(\alpha t), z(\tau) = \beta/\alpha y(\alpha t)$ and using primes to denote differentiation wrt τ, (2)

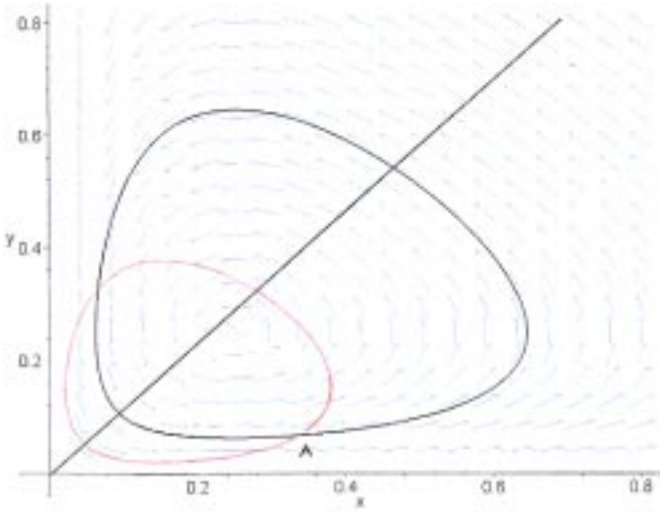

Figure 4:

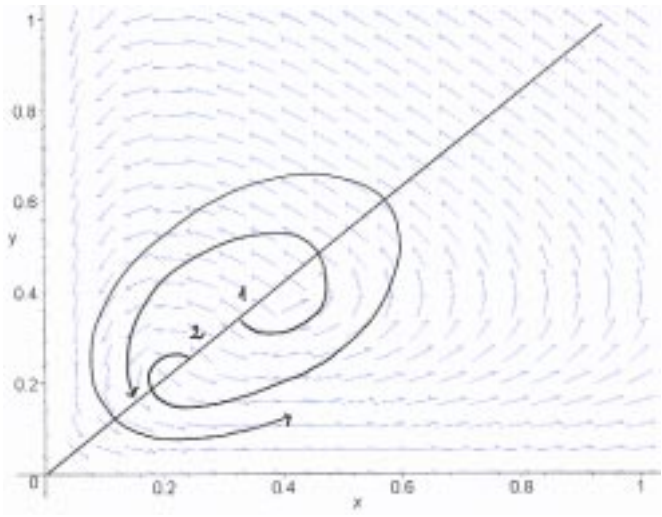

Figure 5:

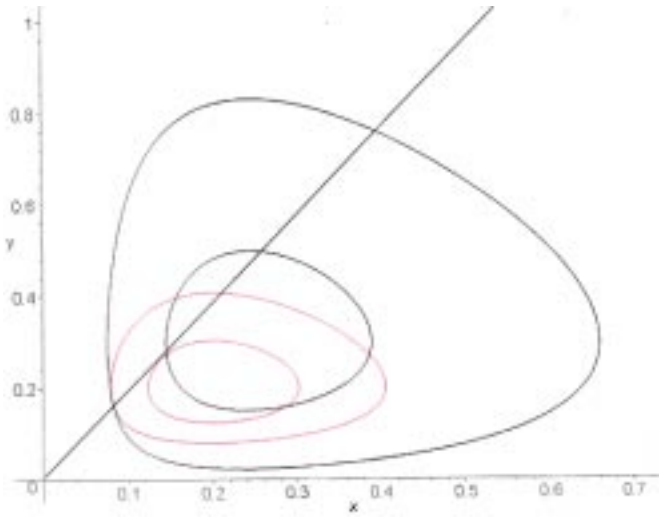

Figure 6:

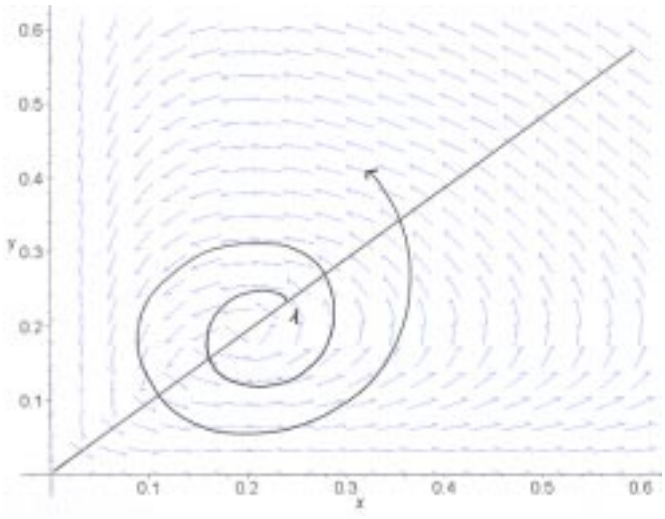

Figure 7:

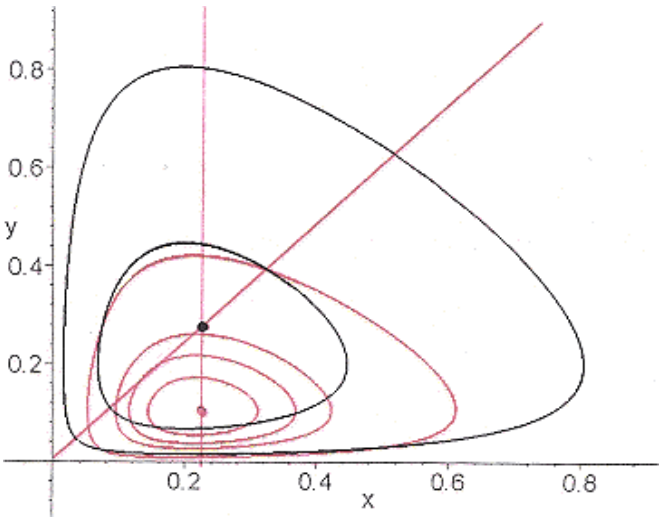

Figure 8:

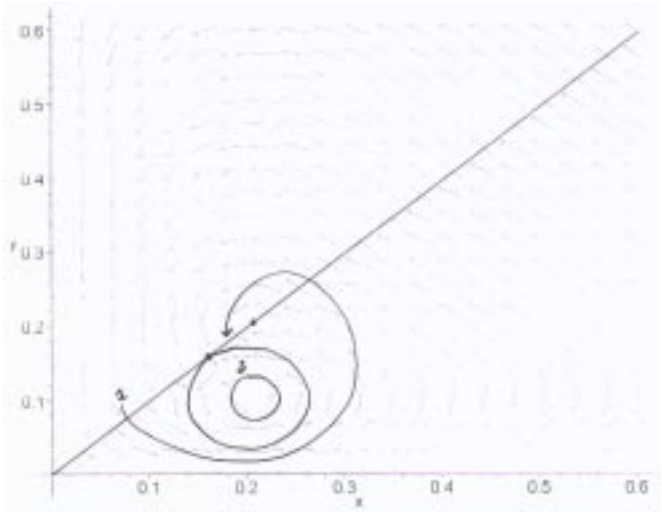

Figure 9:

policy is turned off and the path switches on to a new orbit about the original stationary point which lies outside the starting orbit. Continuing in this way produces an asymmetric unstable spiral. If we look at the phase diagram combining the two switches we get Fig. 7; here we can only see the no vaccination stationary state. The lower stationary state and orbits close to it and below the ray never occur because the policy is switched off there.

However, a low prevalence policy will generate quite complex dynamics with two nonzero stationary states and also the part of the ray $Y = \alpha/\omega X$ will become a region of attraction so that once in the vicinity of this part of the ray the system oscillates between the vaccination policy being on and off. Fig. 8 shows simultaneous operations of the two systems. Note that there is an orbit around the lower vaccination stationary point that is just tangent to the α/ω ray, say where $Y = Y^*$. If the system ever reaches a point on the ray between $Y = Y^*$ and $Y = \alpha/\beta$ then it remains at that point. Again because orbits around the vaccination stationary state cross the ray closer to the origin than orbits around the no vaccination stationary state for the same initial conditions, there is a generic pattern of a stable cycle which will converge to some point in the region of attraction of the ray. Typically, the low prevalence policy will leave roughly the same amplitude fluctuations in X_t but will pass through a region of values of Y_t lower than without the policy (Fig. 9).

The low prevalence vaccination policy can also be considered preferable within the Geoffard and Phillipson demographic structure. For given demographic parameters there is a unique stable stationary state to the system

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

at $X^* = \omega/\beta, Y^* = \alpha/\omega$ (see Fig. 10). Vaccination works again to reduce α to $\bar{\alpha}$ so that in the system with vaccination there is again a unique stable stationary state at the same level of susceptibles but a lower level of infected. An example of the two systems together is shown in Fig. 11. If a high prevalence policy is used (vaccinate whenever $Y_t > \alpha\beta X_t/\omega^2$) then the system cannot converge to the lower vaccination stationary state since in an open region about this stationary point the system is following the dynamics of the no vaccination system. The high prevalence policy system will thus either converge to the no vaccination stationary state or will follow a closed cycle that includes this stationary state in its interior (Fig. 12). However, a low prevalence policy gives the opportunity of converging to the vaccination policy stationary state. Indeed paths must converge to one of the two stationary states since both dynamic systems are stable and trajectories always diminish in amplitude (they "point inwards"). If eventually a trajectory enters a with vaccination phase that keeps the path below the ray $Y_t = \alpha\beta X_t/\omega^2$, then the dynamics

becomes $w'(\tau) = w(\tau)(1 - z(\tau))$ and $z'(\tau) = (\omega/\alpha)z(\tau)(w(\tau) - 1)$. This system has an interior stationary point at $w^* = z^* = 1$. For any initial condition, the equation for the closed orbit in phase space is $w - \ln(w) + \alpha/\omega(z - \ln(z)) = C$ where C is a constant determined by initial conditions. High prevalence is defined by $z \geq w$. On any given orbit, the two points of the orbit that are on the 45^o line are the roots of $w - \ln(w) = \omega C/(\alpha + \omega)$. Now take two systems: the no vaccination system with α and the vaccination system with $\bar{\alpha} < \alpha$. Select an arbitrary orbit from the no vaccination system and find the higher root where this orbit crosses the 45^o line; say at w_0. At w_0 start travelling along the orbit of the vaccination system; this new orbit will cross the 45^o line at points w_1 which satisfy $[w_1 - \ln(w_1)][1 + \bar{\alpha}/\omega] = C$
$= [w_0 - \ln(w_0)][1 + \alpha/\omega]$.

As $\bar{\alpha} < \alpha$ and $w - \ln(w)$ is a convex function with a minimum, the two roots in the vaccination system are each below the corresponding root in the no vaccination system.

of the vaccination system are in force at every instant and so the system converges to the stationary state of the vaccination system. Otherwise, the path will converge to the no vaccination stationary state (Fig. 13). Thus with the demographic dynamics of (16) the low prevalence policy gives preferable in that there is no risk of a closed cycle and a positive chance of attaining the with vaccination stationary state with a lower prevalence of the disease.

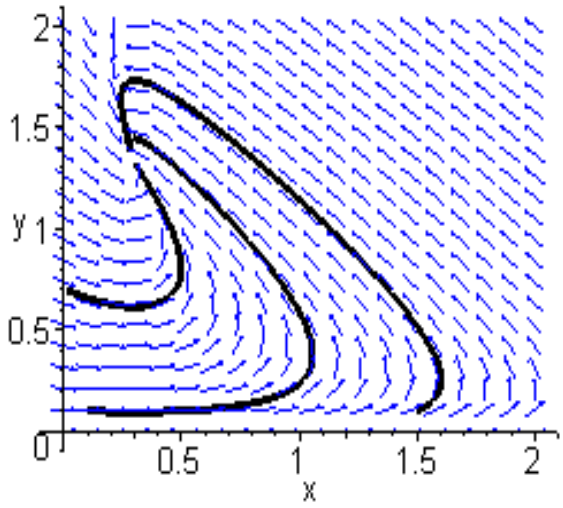

Figure 10:

We conclude that generally the emphasis on procyclical vaccination policy has desirable effects when the vaccination does not give permanent immunity but that countercyclical policy is better if the vaccine does give permanent immunity, in both of the demographic systems considered. This is also in contrast to Geoffard and Phillipson's steady state analysis. Obviously the desirability of any of these policies also depends on the opportunity cost of the public funds.

4. Conclusions

We use a similar demographic structure to that of Geoffard-Phillipson [8] and start by analysing the stationary states and dynamic paths of market provided vaccines that offer a reduction in the chance of infection from the disease. The economic incentive for the individual to take vaccination is similar to that of the permanent immunity case analysed by Geoffard-Phillipson. But in the partial immunity case we find that there may be more stationary states and that the "extra" stationary state is locally a saddlepoint. This is in addition to the stationary states of extinction and of a low level of the population which, like Geoffard-Phillipson, gives a stable focus. The effect is that in more populous societies with a fair proportion of infection the population may grow, with both the healthy and sick groups growing. This can also happen if initially there is a low population with a high proportion of infected and infectious individuals. We conclude that in our framework vaccines offering partial immunity and provided through a market system can control the

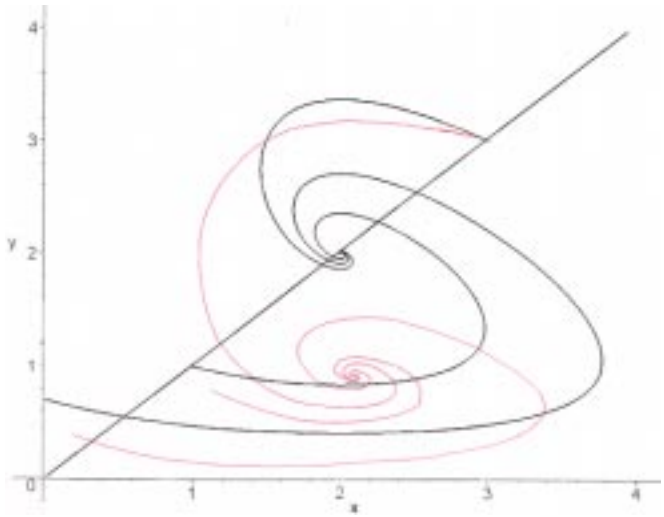

Figure 11:

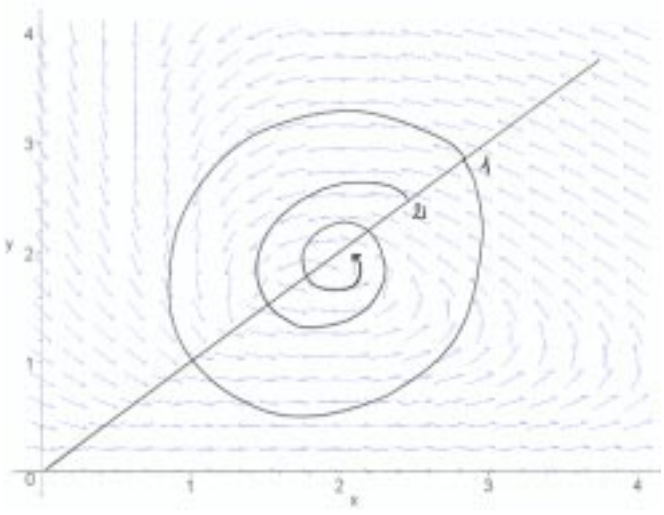

Figure 12:

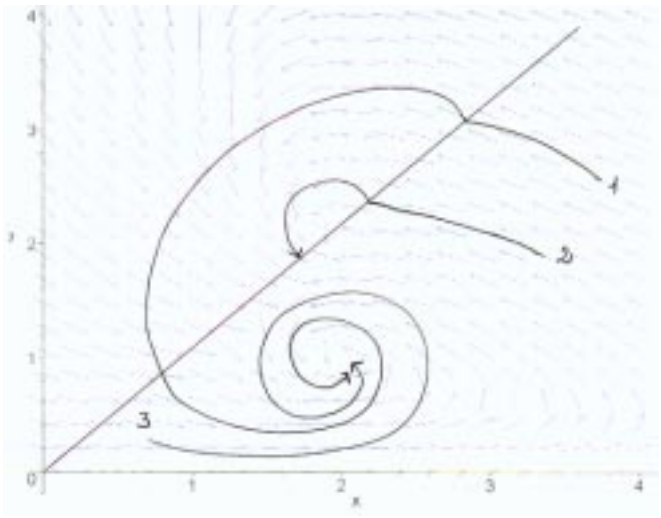

Figure 13:

disease sufficiently to prevent extinction but have elements of instability. The dynamic pattern is more complex than in the case of vaccines offering permanent immunity.

When vaccines are publicly provided through possibly time varying policies we find that the effects of different policies varies a lot with the form of the vaccine. Firstly, we compare alternative policies in the context of vaccination that gives partial immunity. We find that if the criterion function depends mainly on control of the absolute number of infected or on the system being stable and not exhibiting epidemics, then a high prevalence policy (i.e. vaccinate when prevalence is high) is generally more efficient than vaccination at a steady rate which is more efficient in turn than vaccination when prevalence of the disease is low. Secondly, in the full immunity case where vaccination works to control the net growth rate of the susceptible population, we find that a high prevalence policy generates instability whereas it is now the low prevalence policy that leads to reduced fluctuations in the population structure. This conclusion extends to the demographic dynamics used by Geoffard and Phillipson [8].

Much of the earlier epidemiological literature uses predator-prey type models; these are mechanistic but by highlighting crucial steps in the chain of infection they allow us to focus on points at which the disease can be controlled. Vaccination is obviously relevant but the way in which it affects the disease process depends on whether the vaccine gives partial or full immunity. There is also the question of whether intervention policy is necessary to ensure efficient vaccination policy, and if so what form this policy should take. Some reasons for intervention are to correct the dynamic externalities between individuals (especially with infectious disease the individual is motivated by his own chances of infection and the cost of changing this; he ignores the costs on others in the future if he becomes infected); and also to correct for distributional considerations (with many market provided vaccines there is a body of empirical evidence that in the early stages of vaccination the cost of the vaccine is so high that only high percentiles of the income distribution use it). There are also potential monopoly supply problems. Of course vaccination is not the only means of disease control; historically, segregation/quarantine and

also the effects of economic growth on the social infrastructure have been important. In a related paper [6], we look at the interaction between economic growth and the health structure of the population.

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