Infectious Disease Control by Vaccines Giving Full or Partial Immunity

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Abstract

We use a simple Lotka-Volterra model of the disease transmission process to analyse the dynamic population structure in two scenarios. Firstly a vaccine is available on the market at a constant price through time. Secondly, the vaccine is publicly provided. The vaccine works either by giving partial or full immunity to the disease. We analyse market provision for vaccines providing partial immunity and public provision of both types of vaccine.

Infectious diseases have been and are economically and socially costly. In the UK in the 19th century 30% of deaths were caused by typhoid, tuberculosis (TB) and typhus [13]. The World Health Organisation [18] estimates that TB causes about three million deaths and eight million new infections per annum. Vaccination against such diseases can either work to give full 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. 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 full and permanent immunity or as providing a reduction in the chance of infection (partial immunity).

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 to pay for vaccination. 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 generally is a continuous function of the prevalence of the disease and the effectiveness of the vaccine. Here the question is: what are the effects of a market provided vaccine on the dynamic health structure of the population?

Alternatively, if the vaccine is publicly provided, its effects on control of the disease depend on the form of the vaccination programme. Since the vaccine is costly, scarcity of public resources prevents offering the vaccine free to everybody in unlimited quantities at all times. So the question is: with finite resources to fund vaccination, what time profile of publicly provided vaccination is best?

In earlier work Geoffard and Philipson [8] analyse market and publicly provided vaccination programmes in a Lotka-Volterra type predator prey model of the population dynamics for the case of vaccine giving permanent immunity. They show that in the unique stationary state the eradication of the disease is unlikely to be achieved either under a market system for delivering vaccination or under a public subsidy system. With an exogenously given constant path of prices, they find that their system exhibits local stability of the nonzero stationary state rather than cycles. However, they exclusive focus on stationary states so that the global dynamics escape investigation. Furthermore, they work with preventive action which gives permanent immunity. Often this is inappropriate. For example TB does not 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 [9], [11].

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 along non-stationary paths. We also analyse the dynamic effects of public vaccination policies for both the cases of vaccines giving full immunity and vaccines giving partial immunity. Throughout the paper, the dynamic population structure is governed by a variant of the Lotka-Volterra type predator-prey model.

As we discuss in the sequel, the sort of diseases we have in mind are pulmonary TB and polio when vaccination provides partial immunity and full immunity, respectively[1].

In Section 1 we outline the disease model. To avoid the curse of dimensionality in analysing the dynamics we use a slightly different demographic characterisation than Geoffard and Philipson [8] (in particular distinguishing only two health states). 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 leads to choices of vaccination by individuals which may generate additional stationary states for

[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. IUATLD [9], Weatherall [14], WHO [15],[18].

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 tends to settle down to either a stable low healthy/low disease level or involves growth in both the numbers of healthy and infected individuals.

Alternatively, 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 full immunity to infection, 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 [8].

1 The Disease Process

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). 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). [14] 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 the susceptible class (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 occurrence 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. However, partly it was due to the type of dynamic interaction seen in the very simplest predator-prey models which we use here. It follows that a very common paradigm for modelling the disease dynamics is a simple version of the Lotka-Volterra system which combines the latent and the susceptible individuals[3] and ignores the recovered individuals.

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 and latent or actively infected and infectious.

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

[3] This is justified since TB has a short incubation period of TB and only 10% of the latents develop the disease in active form.

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

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

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) and ω is the death rate of the actively infected whether through the disease or natural causes. The probability that a susceptible person becomes infected is represented by βY_t so that the total number of new infections is $\beta X_t Y_t$. The probability of infection reflects prevalence of the disease, the frequency of interaction between individuals (density dependent effect) and the virulence of the disease.

From (2) 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:

$$X_1^* = Y_1^* = 0 \tag{4}$$

$$X_2^* = \omega/\beta, \quad Y_2^* = \alpha/\beta \tag{5}$$

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 and 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 $X_t Y_t$. That is, 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 second stationary point (5) has two pure imaginary roots so long as $\alpha > 0$ so that there are closed cycles about this stationary point.

Notice that if α □ 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 infected individuals. It is less well known that the origin is locally a saddle point. A typical phase diagram is shown in Fig. 1.

Figure 1: Phase plane for system (2)

2 Marketed Vaccination Providing Partial Immunity

With market provision, a preventive device is available at a price p at time t. The vaccine works by reducing β, the risk of infection. In this case for each susceptible there would be a different level of β depending on whether that susceptible has been vaccinated or not. 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 status h_i (h_i is either infected I or susceptible S) and is assumed strictly concave and increasing in c_i, 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 vaccinates if he/she gains expected utility from doing so.

Given that expected utility depends on current prevalence Y_t and the cost of the vaccine p, individual i would be more likely to vaccinate the higher is current

prevalence and the lower the vaccine price. Since the effect of vaccination is to alter the risk of infection faced by the individual, then the average risk of infection varies with the proportion of vaccinated individuals. It follows that we can write $\beta = \beta(p, Y_t)$.

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

$$\begin{cases} \dot{X}_t = \alpha X_t - \beta(p_t, Y_t)X_tY_t \\ \dot{Y}_t = \beta(p_t, Y_t)X_tY_t - \omega Y_t \end{cases} \tag{8}$$

If prices and incomes are constant through time then effectively we can write $\beta = \beta(Y_t)$.[4] The origin is always one stationary state of (8). There are generally other stationary states. Since at $Y = 0$, $\beta(Y)Y = 0$ we know that the origin is the unique stationary state if $\beta(Y)Y$ is bounded above by α. However, if it is not, then the number of nonzero stationary states depends on the number of turning points in $\beta(Y)Y$. Since the sign of the derivative of $\beta(Y)Y$ is given by $(1 + \delta \ln(\beta Y)/\delta \ln Y)$, if the elasticity is either bounded above or below by $|1|$, then the function is monotonic and there is at most one nonzero stationary point solving

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

Otherwise, there may be more than one nonzero stationary state each solving $\alpha = \beta(Y^*)Y^*$(yielding Y^*) and $\omega = \beta(Y^*)X^*$ (which then gives X^*)[5]. Generally the prevalence dependence of β affects the stability of the system. As $\partial\beta/\partial Y < 0$ 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

[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

$$V_t(Y_t, v) = \beta_L Y_t u(I, m_i - p) + (1 - \beta_L Y_t)u(S, m_i - p) + V_{t+1}(Y_{t+1}, v) \tag{9}$$

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

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 again gives 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.

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, 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 in which we numerically integrate the nonlinear differential equations. 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 (8) we select $\alpha = 0.05$, $\beta(Y_t) = 0.2 - 0.1Y_t$ and $\omega = 0.05$. This has three stationary points at $X_1^* = Y_1^* = 0$ which is a saddle point, $X_2^* = Y_2^* = 0.38$ (which has a stationary state level $\beta(Y) = 0.13$), which is a convergent focal point and $X_3^* = Y_3^* = 0.73$ (which has a stationary state level $\beta(Y^*) = 0.07$) which is also a saddle point. The eigenvalues corresponding to the stable focus are $[-0.13 \pm 0.32i]$; around the saddle point with $X_3^* = Y_3^*$ 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 these events occurs depends on the initial conditions. The effect is that either there ultimately is a stable population with a constant structure or total population is 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 through targeted programmes of prevention. If there is an effective vaccine providing permanent immunity then providing vaccine free to all,

[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

$$\begin{bmatrix} -\beta Y & -(\beta' Y + \beta)X \\ \beta Y & \beta' XY \end{bmatrix} \tag{11}$$

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

Figure 2: Phase plane for market provided vaccine

and ensuring that it is taken up by all, can eliminate the disease as susceptibles will always choose to take a vaccine offered at zero cost. As this may be prohibitively costly, 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

$$\begin{cases} \dot{X}_t = \alpha X_t - \beta X_t Y_t \\ \dot{Y}_t = \beta X_t Y_t - \omega Y_t \end{cases} \tag{12}$$

In the absence of any policy the transmission coefficient β takes the value β_H. However, if the vaccine gives partial immunity, a public policy of complete coverage of the susceptibles by vaccination leads to a step change in β from β_H to β_L.

If the vaccine provides permanent immunity then, similarly to [8], 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 (12) only in periods of low prevalence when $Y_t < (\alpha/\omega)X_t$;

(ii) the vaccine is administered along any path satisfying (12) only in periods of high prevalence when $Y_t > (\alpha/\omega)X_t$[7];

(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 this case they face β_L) or not (in this 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

$$\beta = \beta_L \frac{X_v}{X_v + X_{nv}} + \beta_H \frac{X_{nv}}{X_v + X_{nv}} \tag{13}$$

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

$$K = (e^{rT_2} - e^{rT_1})/[r(T_2 - T_1)] \tag{14}$$

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 when 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

[7] 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.

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. It follows that 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 infected individuals.

A constant policy for the same cost gives 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.

For the same economic cost the high prevalence policy appears preferable as it eliminates fluctuations leading to a near constant population structure. Furthermore, 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.

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) has 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

Figure 3: Phase plane for high prevalence policy - partial immunity

Figure 4: Phase plane with constant vaccination policy - partial immunity

Figure 5: Phase plane for low prevalence policy - partial immunity

Figure 6: Phase plane for intermittent policy - full immunity

Figure 7: Phase plane for high prevalence policy - full immunity

Figure 8: Phase plane for low prevalence policy - full immunity

Figure 9: Amplitude of low prevalence policy - full immunity

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[8]. On reaching the ray again from above, the policy is turned off and

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

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 $Y = \alpha/\omega X$ becomes 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 converges to some point in the region of attraction of the ray. Typically, the low prevalence policy leaves roughly the same amplitude fluctuations in X_t but passes 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

$$\begin{cases} \dot{X}_t = \alpha - \beta(X_t, Y_t)X_tY_t \\ \dot{Y}_t = \beta(X_t, Y_t)X_tY_t - \omega Y_t \end{cases} \tag{15}$$

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 thus either converges to the no vaccination stationary state or follows 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 phase where the vaccination policy is in effect that keeps the path below the ray $Y_t = \alpha\beta X_t/\omega^2$, then the dynamics 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 converges to the no vaccination stationary state (Fig. 13). Thus with the demographic dynamics of (15) the low prevalence policy is preferable in that there is no risk of a closed cycle and a positive chance of attaining the stationary state of a vaccinated population with a lower prevalence of the disease.

Figure 10: Low prevalence policy in Geoffard and Phillipson's framework

We conclude that generally in both of the demographic systems considered 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. This is 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. However, 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 infected 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 disease sufficiently to pre-

Figure 11: Geoffard and Phillipson's framework with and without the low prevalence policy

Figure 12: Geoffard and Phillipson's framework with high prevalence policy

Figure 13: Geoffard and Phillipson's framework low prevalence policy

vent 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].

We have found that vaccination has important effects on the dynamic structure of the health status of the population. Moreover, some of the effects are sensitive to the forms that the disease and the vaccination takes. Of course vaccination is not the only means of disease control. Historically, segregation/quarantine and also the effects of economic growth/public health on the social infrastructure have been important.

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