The Optimal Control of Infectious Diseases via Prevention and Treatment*

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Abstract. This paper fully characterizes the optimal control of a recurrent infectious disease through the use of (non-vaccine) prevention and treatment. The dynamic system may admit multiple steady states and the optimal policy may be path dependent. We find that an optimal path cannot end at a point with maximal prevention; it is necessarily zero or at an intermediate level. In contrast, an optimal path must end at a point at which treatment is either maximal or minimal. We show that treatment and prevention are imperfect substitutes and may or may not be used in conjunction, depending on the state of the system. This means that optimal paths do not generally approach steady states as rapidly as possible. We show that for some parameterizations, it is always optimal to go to a specific steady state (either a high or a low prevalence one) while for others, the optimal path and steady state depend on initial conditions and thus there is hysteresis. We find that the comparative statics with respect to the rates of infectivity and recovery may radically differ across steady states, which has important policy implications. Last, we illustrate the main conclusions of the formal analysis by simulations.

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1. Introduction

Despite significant achievements in the battle against infectious diseases, effective infection control remains a formidable challenge.[1] Infectious diseases remain one of the major causes of morbidity and mortality in both developing and developed countries and are a major strain on public budgets. In parallel with rapid advancements in the biomedical field, there is an ongoing effort to develop strategies to better deploy existing tools and resources. In particular, it is a priority to determine how different interventions work at different stages of an epidemic (separately and in conjunction) and to determine optimal policy.

An old adage holds that *an ounce of prevention is worth a pound of cure.* In the case of infectious diseases, the relationship between prevention and treatment is complicated by the presence of externalities. It turns out that determining the right mix of prevention

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[1]See Piot et al. (2008).

and treatment is a delicate matter and significantly more complicated than folk wisdom might suggest.

To aid policy makers in formulating sensible public policies, it is important to conduct a systematic analysis of how different interventions work within a unified framework and to carefully determine how such interventions interact. To this end, we study a simple susceptible-infected-susceptible (or SIS) model, in which individuals can be either infected or susceptible (but never immune).[2] We assume that both the infection and recovery rates can be partially controlled by a benevolent social planner. Specifically, we assume that the planner can lower disease incidence (i.e. the rate of new infections) through costly preventive effort and/or lower disease prevalence (i.e. the number of infected people) through costly therapeutic effort.[3] We fully characterize the planner's problem and in so doing, derive both the optimal policy, steady states and transition paths.

One of the distinct advantages of considering treatment and prevention within a unified framework, is that it helps organize and clarify results that are known from single-instrument models. Thus we can both analyze the interaction of multiple policies and obtain existing models as special cases. This makes it easier to trace different effects to specific policy instruments. Despite superficial similarities, prevention and treatment are profoundly different in their effects and desirability for different levels of disease prevalence. In particular, while optimal prevention will tend to push prevalence towards intermediate levels, i.e. towards an interior steady state, optimal treatment will tend to push prevalence towards the extremes, i.e. towards corner steady states with either very high or very low infection levels.

In understanding the way prevention and treatment work in reducing infection, it is instructive to first consider each in isolation. There are several important differences between these two interventions. First, they target different groups in the population. While prevention directly targets susceptible individuals and thus disease *incidence*, treatment directly targets infected individuals and thus disease *prevalence*.[4] The second (and more important) difference, lies in the way that the marginal costs and benefits from employing an instrument vary with disease prevalence. For both interventions, we assume that marginal costs are independent of prevalence.[5] But it turns out that the main difference lies in the way that marginal benefits accrue. Whereas the marginal benefit of prevention is an increasing function of disease prevalence, the marginal benefit of treatment is *decreasing*. In the terminology of Brock and Starrett's (2003) analysis of shallow lake management and other non-convex systems, with treatment there is *destabilizing positive feedback*. By the same token, with prevention, there is *stabilizing negative feedback*. It is the destabilizing effect of treatment that creates the scope for multiple steady states.

In general, for extreme levels of disease prevalence, treatment and prevention will tend

[2] Diseases that fall in this category include sexually transmitted diseases like chlamydia and gonorrhea. Applications also include a wide range of other bacterial, viral, fungal and parasitic infections, but infections have very varied characteristics so care should be taken in determining applicability for a given disease.

[3] We focus on temporary measures that must be sustained through time in order to remain effective. In particular, we exclude vaccinations which confer prolonged (or permanent) immunity.

[4] Since incidence and prevalence are intimately related, prevention indirectly affects prevalence while treatment indirectly affects incidence.

[5] Note that this is the *marginal* cost, i.e. the cost of treating or protecting "one" more individual. The total cost of the intervention is trivially increasing in the number of targeted individuals.

to be strong substitutes and used in very asymmetric proportions, whereas intermediate prevalence levels lead them to be weaker substitutes, such that it may be optimal to use them in conjunction.[6] Along optimal paths, treatment and prevention are always at their maximum or minimum possible levels, whereas this is not true once a steady state is reached.

More generally, we find that the system admits a large number of potential steady states, some of which may coexist. We delineate several possible regimes (which depend on parameters), which characterize the optimality and multiplicity/uniqueness of steady states. In Regime I, there is a unique saddle point which is always the endpoint of the optimal path. In Regime II, there are two saddle points, but only one of these can be the end point of an optimal path. In Regime III, there are also two saddle points, each of which is an optimal endpoint for appropriate initial conditions. In this regime, the optimal policy is path dependent. There is hysteresis in the sense that history, captured by the initial level of disease prevalence, will determine where it is optimal to take the system. This means that there may be a discontinuity: as the initial level of infection is increased, there may be a shift in which steady state is optimal and the solution therefore changes qualitatively.[7]

While we prove that the system cannot display limit cycles or spiral sinks, the dynamics may still exhibit complicated behavior such as spiral paths. We show that an optimal policy never involves such paths, but is a relatively simple function of disease prevalence.

Last, we find that the comparative statics results for steady state prevalence with respect to infectiousness and the rate of recovery, may be radically different across steady states. E.g., we find that while decreasing the infectiousness of the disease is always welfare enhancing, the manner in which these gains are realized differ from one steady state to the other. If there is no prevention in the benchmark steady state, then the optimal policy response to decreased infectiousness may be to increase costly treatment in the short run in order to drive down infection prevalence to the new steady state. In turn, steady state welfare is higher in the new steady state, outweighing the additional costs incurred during the transition. On the other hand, if there is positive prevention in the benchmark steady state, then the optimal policy response to decreased infectiousness is to decrease prevention in the short run to increase disease prevalence to the new steady state. In this new steady state, welfare is lower than before the transition, but this welfare loss is outweighed by the cost savings due to lack of prevention during the transition to the new steady state. Because of this lack of robust prescriptions across steady states, caution is advisable when using comparative statics results to inform public policy.

1.1. Related Literatures. The literature on economic epidemiology is varied and growing and there are several good surveys, such as Philipson (2000), Gersovitz and Hammer (2003) and Klein et al. (2007). Of direct relevance to the present work is research that deals with prevention and treatment, separately or in conjunction.

The earliest contributions, by Sanders (1971), Sethi (1974) and Sethi and Staats (1978), consider treatment in different versions of the SIS model from a planner's perspective. Goldman and Lightwood (1995) consider treatment in the SIS model under

[6] To be precise, treatment and prevention may be used in conjunction for *some* intermediate prevalence levels.

[7] This is a property shared by many economic/ecological models, as surveyed in Dasgupta and Mäler (2003).

learning, while Goldman and Lightwood (2002) also study treatment in the controlled SIS model, but considers different cost structures than the earlier literature.[8] Rowthorn (2006) and Anderson et al. (2010) extend the analysis of the controlled SIS model to settings with budget and wealth constraints. Toxvaerd (2009a) considers decentralization to strategic decision makers, while Toxvaerd (2009b) considers the effects of treatment when recovery confers immunity to further infection.

The literature on prevention is more varied than that on treatment. Sethi (1978) considers quarantines, while Geoffard and Philipson (1996) and Aadland et al. (2010) consider non-vaccine prevention in the SI and SIS models respectively. Reluga (2009) analyzes prevention by strategic individuals in linked subpopulations, while Reluga (2010) considers prevention through social distancing**.** Toxvaerd (2010) analyzes continual prevention in the SIS model and decentralization of optimal policy to strategic decision makers. There are also important literatures on vaccination and on abstinence, exemplified by Brito et al. (1991) and Kremer (1996), respectively. The issues dealt with in those papers are somewhat orthorgonal to the present work and are reviewed in more detail in Toxvaerd (2010). Greenwood et al. (2009) consider a search-theoretic matching model of the SI variety and analyze the incentives to form long and short term partnerships.

There are a few papers that explicitly consider multiple instruments. Most related to our work is that of Gersovitz and Hammer (2004) who, like us, consider prevention and treatment in an SIS framework. In contrast to us, they bypass the issue of multiplicity by assuming that there is a unique steady state and that it is an interior one. As we shall show, this assumption has radical consequences for both the analysis and the conclusions derived from it. In a short note, Zaman et al. (2007) consider vaccination and treatment in an SIR setting and simulate optimal paths. A similar exercise is done in Almeder et al. (2007) for an HIV type disease. Goyal and Vigier (2010) consider a static two stage model with vaccination and abstinence. Dodd et al. (2010) consider multiple concurrent interventions and discuss when there are likely to be synergies between these in the sense that raising the level of one instrument increases the benefit to increasing the level of other instruments. Last, Blayneh et al. (2009) consider prevention and treatment in a setting with a vector-borne disease. Apart from Gersovitz and Hammer (2004), these papers are similar to ours only in spirit and their analyses are not directly comparable to the one we carry out.

For completeness, we should also mention some related contributions that do not deal directly with infection control, but which share structural features with our work. Feichtinger (1984) studies conditions for synergies between multiple controls in non-linear dynamic systems. Although related to our work, his results do not apply to our setting. Behrens et al. (2000) analyze a model of the spread of drug use, in which both treatment and prevention can reduce the prevalence of addiction and in which the habit of drug use spreads in the population like an infection. Interestingly, they find that at early stages of the epidemic, prevention should take precedent whereas at later stages, the optimal policy is to treat the addiction. Such a policy is the mirror image of the optimal policy in the present setting, in which high treatment (and low prevention) is the optimal response at low levels of disease prevalence and high prevention (and no treatment) is optimal for high levels of disease prevalence.

[8] Goldman and Lightwood (2002)'s analysis focuses mainly on necessary conditions for optimality and provide an informal analysis using phase diagrams.

Last, our paper contributes to an important literature on equilibrium multiplicity and history dependence in systems with non-convexities, as surveyed in Dasgupta and Mäler (2003) and Deissenberg et al. (2004). Of particular relevance to our work is the literature on the optimal management of shallow lake systems, such as Brock and Starrett (2003), Mäler et al. (2003) and Wagener (2003). It turns out that important results from that literature can be brought to bear on the management of infectious diseases.

The remainder of the paper is structured as follows. In Section 2, we outline the classical susceptible-infected-susceptible model. In Section 3, we introduce the economic version of the model and partially characterize the optimal policies. In Section 4, we characterize the steady states of the system and the optimal paths formally. In Section 5, we describe the equilibria and dynamics of the model and interpret the central features driving the results. In Section 6, we perform some simple comparative analysis, consider welfare and draw some policy conclusions. In Section 7 we illustrate some of the main points of the analysis via simulated examples. In Section 8, we outline a number of extensions of our model and discuss robustness of our results to these changes. Section 9 concludes. Most proofs are found in the Appendix.

2. The Classical Model

To make the exposition self-contained, we will start by expounding the classical epidemiological version of the susceptible-infected-susceptible model in some detail. This will not only aid in understanding the economic model that follows, but also highlight the contrast in predictions based on the separate modeling approaches.

The classical susceptible-infected-susceptible model is simple to describe.[9] Time is continuous and runs indefinitely. A population $\mathcal{P} =[0, 1]$ consists of a continuum of infinitely lived individuals who can at each instant $t \geq 0$ each be in one of two states, namely *susceptible* or *infected*. The set of infected individuals is denoted by $\mathcal{I}(t)$ and has measure $I(t)$, while the set of susceptible individuals is denoted by $\mathcal{S}(t)$ and has measure $S(t)$. Because the population size is normalized to unity, these measures can be interpreted as fractions. Henceforth, $I(t)$ shall be referred to as *disease prevalence*.

At each instant, the population mixes homogeneously. This corresponds to pairwise random matching where each individual has an equal chance of meeting any other individual, irrespective of the health status of the two matched individuals. Whereas a match between two infected individuals or two susceptible individuals does not create any new infection, a match between an infected and a susceptible individual may. The rate at which infection is transferred in such a match is denoted by $\beta > 0$. This parameter captures the infectivity of the disease. Coupled with the assumption of homogeneous mixing, this means that the rate at which susceptible individuals become infected is given by the simple expression $\beta I(t)S(t)$. Thus the rate of new infection, or *disease incidence*, is proportional to disease prevalence.[10] Note that while disease incidence is a flow, disease prevalence is a stock.

Infected individuals recover spontaneously at rate $\gamma \geq 0$. This means that the rate

[9] See Anderson and May (1991), Daley and Gani (2001) or Keeling and Rohani (2008) for good introductions and applications.

[10] The term $\beta I(t)S(t)$ should be thought of as the rate at which susceptible individuals have contact with other individuals, multiplied by the probability of the contact being with an infectious individual, multiplied by the probability that the infection is transmitted in such a contact. See e.g. Keeling and Rohani (2008) for a detailed derivation.

at which infected individuals become susceptible is given by $\gamma I(t)$. Figure 1 shows the stocks and flows of susceptible and infected individuals diagrammatically. The dynamics of the model are described by the following system of differential equations:

$$\dot{S}(t) = I(t)\left[\gamma - \beta S(t)\right] \tag{1}$$
$$\dot{I}(t) = I(t)\left[\beta S(t) - \gamma\right] \tag{2}$$
$$I(t) = 1 - S(t), \quad I(0) = I_0 \tag{3}$$

Using the normalization, this system reduces to the following simple logistic growth equation:

$$\dot{I}(t) = I(t)\left[\beta(1 - I(t)) - \gamma\right], \quad I(0) = I_0 \tag{4}$$

The steady states of this system are

$$I^* = 0, \quad I^* = \frac{\beta - \gamma}{\beta} \tag{5}$$

For $\beta > \gamma$, the stable steady state is endemic while for $\beta < \gamma$, the relevant and stable steady state involves eradication. In other words, if the rate at which individuals become infected surpasses the rate at which they recover, then some positive fraction of the population will always be infected. If recovery is not possible, the entire population ends up being infected. On the other hand, if individuals recover at a higher rate than the rate at which they become infected, then the disease eventually dies out. Last, note that the endemic steady state disease prevalence is increasing in infectivity and decreasing in the rate of recovery.

At the aggregate level, there is no uncertainty and thus the probability that a randomly chosen individual is infected must coincide with the fraction of infected individuals. From the perspective of an infected individual, the transition to susceptibility is governed by a Poisson process with rate γ, which is memoryless. Similarly, for a fixed level of aggregate infection $I(t)$, the transition to infectivity for a susceptible individual is governed by a Poisson process with rate $\beta I(t)$. Thus transition probabilities are memoryless, a fact that greatly simplifies the analysis that follows. This completes the description of the classical SIS model.

3. The Economic Model and Optimal Policies

In the economic version of the model, each individual earns flow payoffs that depend on the state of their health. For simplicity, assume that an individual earns flow payoff $\omega_{\mathcal{S}}$ while susceptible and $\omega_{\mathcal{I}} < \omega_{\mathcal{S}}$ while infected. It shall prove useful to introduce the health premium $\omega \equiv \omega_{\mathcal{S}} - \omega_{\mathcal{I}} > 0$. The future is discounted at rate $\rho > 0$. The basic epidemiological parameters $\beta > 0$ (infectiousness) and $\gamma > 0$ (background rate of spontaneous recovery) are retained from the classical model.

The two policy instruments at the planner's disposal are *prevention* and *treatment*. These instruments influence the flows from $\mathcal{S}(t)$ to $\mathcal{I}(t)$ and from $\mathcal{I}(t)$ to $\mathcal{S}(t)$ respectively. Specifically, the planner can set some level of prevention $\pi(t) \in [0, 1]$ at time $t \geq 0$, which translates into effective disease incidence $(1 - \pi(t))\beta I(t)S(t)$. The factor $(1 - \pi(t))$ can be thought of as the proportion of susceptible individuals who is exposed at time $t \geq 0$. Turning to treatment, the planner can set the level of treatment $\tau(t) \in [0, 1]$ at time $t \geq 0$, which translates to an effective recovery rate $(\tau(t)\alpha + \gamma)$. Here, $\alpha > 0$ is the efficiency

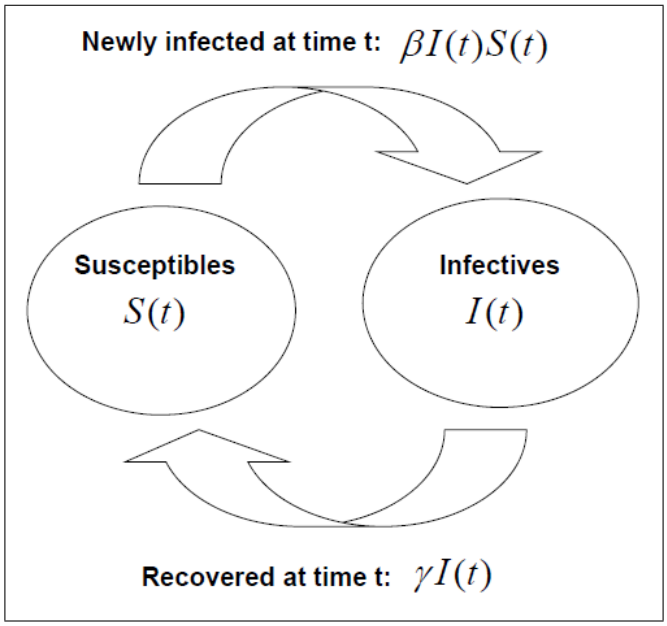

Figure 1: Stocks and flows in the classical model.

of treatment in inducing recovery. Last, the individual costs of protection and treatment are $c_P \geq 0$ and $c_T \geq 0$ respectively. Note the mnemonic notation: τ denotes *treatment* and π denotes *prevention*. We should add that an equivalent interpretation is that each susceptible individual is exposed at intensity $(1-\pi(t))$ and that each infected individual is treated at intensity $\tau(t)$. Figure 2 shows the stocks and flows in the controlled version of the model.

We now consider the optimal control of the SIS system from the perspective of a benevolent social planner. The planner's objective is assumed to be a straightforward sum of the individuals' infinite horizon, discounted expected utilities. The planner's problem is therefore to solve the following programme:

$$\max_{\tau(t),\pi(t)\in[0,1]} \int_0^\infty e^{-\rho t} \left[I(t) \left[\omega_{\mathcal{I}} - c_T \tau(t) \right] + (1 - I(t)) \left[\omega_{\mathcal{S}} - c_P \pi(t) \right] \right] dt \tag{6}$$

$$s.t. \quad \dot{I}(t) = I(t) \left[(1-\pi(t))\beta(1-I(t)) - \gamma - \tau(t)\alpha \right], \quad I(0) = I_0 \tag{7}$$

The optimal value function for this programme is denoted by $V^*(I_0)$, where dependence on the parameters has been suppressed for ease of notation.

Throughout, we maintain the following:

Assumption We assume that (i) $\omega - c_P > 0$ and (ii) $\beta - \gamma - \alpha > 0$.

The former inequality implies that a policy without any treatment, but with a strictly interior level of prevention, cannot eradicate infection even asymptotically. The latter inequality implies that a policy without prevention, but with maximal treatment, cannot eradicate infection even asymptotically.

Note that this environment is stationary and that the problem to be solved is autonomous, i.e. time enters in the integrand only through the discount term $e^{-\rho t}$.

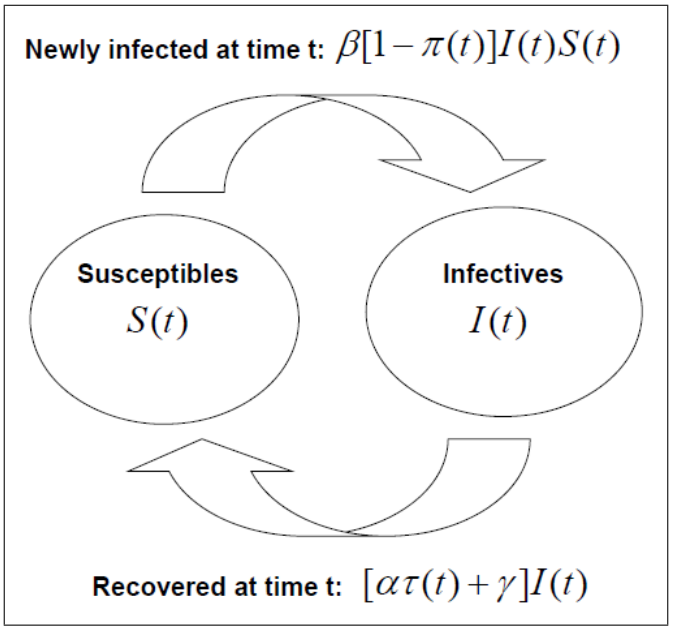

Figure 2: Stocks and flows in the controlled model.

An admissible solution is a triple of functions $(I(t), \tau(t), \pi(t))$ in which for all $t \geq 0$, $I(t)$ satisfies the logistic growth equation (7)and where $\tau(t), \pi(t) \in [0, 1]$. Furthermore, the policies $\tau(t), \pi(t)$ must be piecewise continuous. Let $\lambda(t)$ denote the current-value costate variable (or multiplier). It is required to be piecewise continuously differentiable.

Before embarking on the detailed analysis of the model, we ensure that the planner's problem admits an optimal solution:

Theorem 1. *An optimal solution $(I^*(t), \tau^*(t), \pi^*(t))$ exists if at least one of the fixed points A, B, A_0, B_0 (to be specified below) is feasible.*

Proof: See Appendix A ■

The existence proof relies on existence of optimal solutions in truncated versions of the problem and is by contradiction. The qualification in the statement of the result is that at least one fixed point be *feasible* and does not require that the steady state be optimal. This is an implicit restriction on the allowable parameter constellations and is a sensible requirement.

Turning to the characterization of the optimal policy, the current-value Hamiltonian is given by

$$\begin{aligned} H &= -\omega I(t) - c_P\pi(t)(1-I(t)) - c_T\tau(t)I(t) \\ &\quad +\lambda(t)I(t)\left[(1-\pi(t))\beta(1-I(t)) - \gamma - \tau(t)\alpha\right] \end{aligned} \tag{8}$$

Note that the current-value Hamiltonian is linear in both control variables, which has important implications for the characterization of optimal policies.

The evolution of the costate variable is given by

$$\begin{aligned}\dot{\lambda}(t) &= \rho\lambda(t) - \frac{\partial H}{\partial I(t)} &(9)\\ &= \lambda(t)\left[\rho+\gamma+\alpha\tau(t)+\beta(2I(t)(1-\pi(t))+\pi(t)-1)\right] \\ &\quad +\left[\omega+\tau(t)c_T-\pi(t)c_P\right] &(10)\end{aligned}$$

In general, the steady state is given by the solution to the system $\dot{I}(t) = \dot{\lambda}(t) = 0$, i.e.

$$\begin{aligned}I(t) &= \frac{\beta(1-\pi(t))-\gamma-\alpha\tau(t)}{\beta(1-\pi(t))} &(11)\\ \lambda(t) &= \frac{\omega+\tau(t)c_T-\pi(t)c_P}{\gamma-\beta(1-\pi(t))-\rho+\alpha\tau(t)} &(12)\end{aligned}$$

For a path to be optimal, the policy instruments $(\tau(t), \pi(t))$ must maximise the Hamiltonian (8). This yields the following necessary conditions for optimality. Optimal treatment is given by the bang-bang solution

$$\begin{aligned}\tau(t) &= 0 \quad if \quad \alpha\lambda(t) > -c_T &(13)\\ \tau(t) &\in [0,1] \quad if \quad \alpha\lambda(t) = -c_T &(14)\\ \tau(t) &= 1 \quad if \quad \alpha\lambda(t) < -c_T &(15)\end{aligned}$$

In turn, optimal prevention is given by the bang-bang solution

$$\begin{aligned}\pi(t) &= 0 \quad if \quad \beta\lambda(t)I(t) > -c_P &(16)\\ \pi(t) &\in [0,1] \quad if \quad \beta\lambda(t)I(t) = -c_P &(17)\\ \pi(t) &= 1 \quad if \quad \beta\lambda(t)I(t) < -c_P &(18)\end{aligned}$$

These policies simply state that if the marginal benefit of increasing an instrument (i.e. treatment or prevention) exceeds the marginal cost of doing so, then it is optimal to increase the level of the instrument. Similarly, if the marginal cost exceeds the marginal benefit, then it is optimal to decrease the level of the instrument. Last, when the marginal cost equals the marginal benefit, the optimal policy is not determined.

Recall that $\lambda(t) < 0$ is the (negative) social utility associated with a marginal increase in disease prevalence. With this in mind, it is straightforward to interpret the optimal policies in terms of the marginal costs and benefits of intervention. In the case of treatment, the marginal benefit of intervention is given by $-\alpha\lambda(t)$, which follows from the fact that α is the rate at which increased treatment induces recovery (i.e. it is the efficiency of treatment) and each recovery benefits society at level $-\lambda(t)$. In the case of preventive effort, the marginal benefit of intervention is given by $-\beta I(t)\lambda(t)$. This follows since $\beta I(t)$ is the rate at which unprotected susceptible individuals become infected and each infected individual costs society $\lambda(t)$.

Figure 3 illustrates the areas in $(I(t), \lambda(t))$-space in which the different policy combinations are optimal and indicates the different feasible steady states.

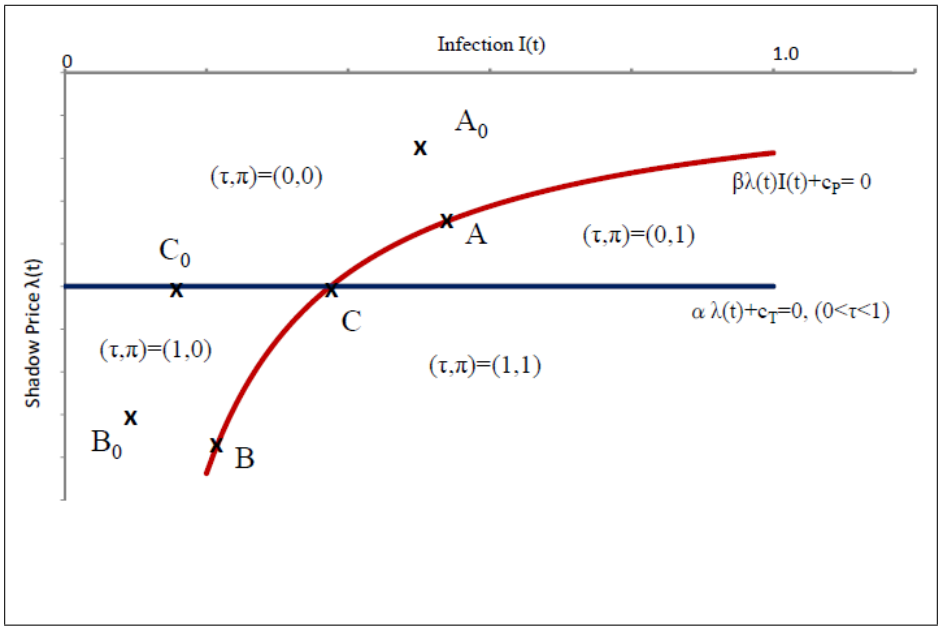

Figure 3: Optimal policies and typology of steady states.

4. Optimal Paths and Steady States

We will now proceed with a detailed analysis of the optimal paths and the steady states of the system, through a number of propositions. In the next section, we will offer a more informal description of these results.

The system of differential equations for the state variable (disease prevalence) and the costate variable has six potential fixed points, which we will denote by (A, B, C, A_0, B_0, C_0) respectively. As will become clear, the fixed points can be sensibly grouped as (A, B, C) and (A_0, B_0, C_0).

Proposition 2. *The dynamic system admits six potential steady states. These are characterized as follows: Solution* A *:* $\tau^* = 0$ *and* $\pi^* \in (0,1)$*. Solution* B*:* $\tau^* = 1$ *and* $\pi^* \in (0,1)$*. Solution* C*:* $\tau^* \in (0,1)$ *and* $\pi^* \in (0,1)$*. Solution* A_0*:* $\tau^* = 0$ *and* $\pi^* = 0$*. Solution* B_0*:* $\tau^* = 1$ *and* $\pi^* = 0$*. Solution* C_0*:* $\tau^* \in (0,1)$ *and* $\pi^* = 0$*.*

From this proposition, it follows that in steady state, treatment can be either at the highest possible level, the lowest possible level or at an intermediate level. Prevention, in contrast, is either at the lowest possible level or at an intermediate level. Steady states with subscript "0" are those that involve no prevention, whereas those without a subscript denote steady states with a positive amount of prevention. The different steady state values are listed in Section 4.1. For each set of parameters, only a subset of these steady states are feasible. The relevant feasibility conditions are set out in Appendix B.

Whereas the steady state may involve keeping prevention at an interior level, the approach to a steady state always involves maximal or minimal levels of the two policy instruments, as the following result shows:

Proposition 3. *The optimal policy is always of the bang-bang variety. Along the approach path to a steady state, both* $\tau(t), \pi(t) \in \{0,1\}$ *for all* $t \geq 0$*, except at a finite*

number of points where there is an instantaneous switch from one control regime to another.

Proof: Follows directly from the characterization of the optimal policies and the phase diagram ■

Since optimal policies generically take extreme values on transition paths while possibly intermediate values once steady state is reached, optimal policies may be expected to have discontinuities in steady state (for some parameter constellations).

Because the planner's problem is autonomous and involves a single state variable, optimal prevalence paths are necessarily monotone time. This means that an optimal path cannot bend back on itself in $(I(t), \lambda(t))$-space. Owing to the bang-bang nature of the optimal policies, the monotonicity implies that only a limited number of policy regime switches can occur in approaching a steady state. In particular, we have the following result:

Proposition 4. *Along an optimal path, there will be at most four switches of regime. At most one switch from $\tau(t) = 0$ to $\tau(t) = 1$, at most one switch from $\tau(t) = 1$ to $\tau(t) = 0$, at most one switch from $\pi(t) = 0$ to $\pi(t) = 1$ and at most one switch from $\pi(t) = 1$ to $\pi(t) = 0$.*

The monotonicity of disease prevalence along optimal paths, coupled with the bang-bang characteristic, means that optimal policies can be characterized as simple functions of disease prevalence as follows:

Corollary 5. *Along an optimal path, if there are regime switches in treatment or prevention respectively, optimal policies are as follows: (i) For fixed $\overline{\pi} \in [0,1]$, there is a unique $\hat{I}(\overline{\pi}) \in [0,1]$ such that*

$$\begin{aligned} \pi(t) &= 0 \quad if \quad I(t) > \hat{I}(\overline{\pi}) && (19)\\ \pi(t) &\in [0,1] \quad if \quad I(t) = \hat{I}(\overline{\pi}) && (20)\\ \pi(t) &= 1 \quad if \quad I(t) < \hat{I}(\overline{\pi}) && (21) \end{aligned}$$

(ii) For fixed $\overline{\tau} \in [0,1]$, there is a unique $\hat{I}(\overline{\tau}) \in [0,1]$ such that

$$\begin{aligned} \pi(t) &= 0 \quad if \quad I(t) < \hat{I}(\overline{\tau}) && (22)\\ \pi(t) &\in [0,1] \quad if \quad I(t) = \hat{I}(\overline{\tau}) && (23)\\ \pi(t) &= 1 \quad if \quad I(t) > \hat{I}(\overline{\tau}) && (24) \end{aligned}$$

This means that any policy along optimal paths can be fully characterized in terms of a few critical levels of disease prevalence which indicate when the policy instruments should be switched between their extreme levels (until steady state is reached).

That full prevention is not possible in an optimal steady state is shown next.

Proposition 6. *At any optimal steady state (I^*, λ^*), the level of prevention $\pi^* < 1$.*

Proof: See Appendix C ■

The proof shows that a policy that keeps $\pi^* = 1$ in steady state does not satisfy the transversality condition for an optimal path.

While there may be several potential steady states, it turns out that the fully interior ones, in which the treatment instrument is kept at an interior level, are never the end points of optimal paths. This result significantly simplifies the description of the optimal policy. Formally, we have the following:

Proposition 7. *No optimal path converges to either C or C_0.*

Proof: See Appendix D ■

Since the interior points that we have just ruled out are characterized by interior levels of the treatment instrument, the following corollary is immediate:

Corollary 8. *An optimal path always converges to a steady state at which $\tau^* \in \{0, 1\}$.*

In order to further reduce the set of steady states that should be considered for a given set of parameters, the following result usefully shows that one can focus attention on one or the other of two sets of steady states as follows:

Proposition 9. *Depending on the parameter values, at least one and at most two of the steady states A, A_0, B, B_0 is the end point of an optimal path. For any given set of parameter values, it is not possible for both A and A_0, for both B and B_0 or for both C and C_0 to be fixed points.*

Proof: Follows directly from the parameter restrictions in Appendix B ■

Potential steady states are indicated in Figure 3. The triple (A, B, C) straddle the boundary between the no prevention and full prevention areas, whereas the triple (A_0, B_0, C_0) is situated in the no prevention area. Similarly, whereas the steady states (A, A_0) are in the no treatment area and the steady states (B, B_0) are in the full treatment area, the steady states (C, C_0) both straddle the boundary between the no treatment and the full treatment areas.

4.1. Steady State Values. The different steady states are given as follows:

Solution A: This case corresponds to $\tau(t) = 0$ and $\pi(t) \in (0, 1)$. The steady state solution is then

$$I_A \equiv \frac{\rho c_P}{\beta(\omega - c_P)} \tag{25}$$

$$\lambda_A \equiv \frac{c_P - \omega}{\rho} \tag{26}$$

$$\pi_A \equiv \frac{c_P(\beta - \gamma + \rho) + \omega(\gamma - \beta)}{c_P(\beta + \rho) - \beta\omega} \tag{27}$$

$$\tau_A \equiv 0 \tag{28}$$

Solution B: This case corresponds to $\tau(t) = 1$ and $\pi(t) \in (0,1)$. The steady state solution is then

$$I_B \equiv \frac{\rho c_P}{\beta(c_T + \omega - c_P)} \tag{29}$$

$$\lambda_B \equiv \frac{c_P - \omega - c_T}{\rho} \tag{30}$$

$$\pi_B \equiv \frac{c_P(\beta - \gamma + \rho - \alpha) + (\omega + c_T)(\alpha + \gamma - \beta)}{c_P(\beta + \rho) - \beta(\omega + c_T)} \tag{31}$$

$$\tau_B \equiv 1 \tag{32}$$

Solution C: This case corresponds to $\tau(t) \in (0,1)$ and $\pi(t) \in (0,1)$. The steady state solution is then

$$I_C \equiv \frac{\alpha c_P}{\beta c_T} \tag{33}$$

$$\lambda_C \equiv \frac{-c_T}{\alpha} \tag{34}$$

$$\pi_C \equiv \frac{2\alpha c_P - \alpha\omega + c_T(\gamma + \rho - \beta)}{\alpha c_P - \beta c_T} \tag{35}$$

$$\tau_C \equiv \frac{\alpha c_P - \alpha\omega + \rho c_T}{\alpha c_T} \tag{36}$$

Solution A_0: This case corresponds to $\tau(t) = 0$ and $\pi(t) = 0$. The steady state solution is then

$$I_{A_0} \equiv \frac{\beta - \gamma}{\beta} \tag{37}$$

$$\lambda_{A_0} \equiv \frac{-\omega}{\beta - \gamma + \rho} \tag{38}$$

$$\pi_{A_0} \equiv 0 \tag{39}$$

$$\tau_{A_0} \equiv 0 \tag{40}$$

Solution B_0: This case corresponds to $\tau(t) = 1$ and $\pi(t) = 0$. The steady state solution is then

$$I_{B_0} \equiv \frac{\beta - \gamma - \alpha}{\beta} \tag{41}$$

$$\lambda_{B_0} \equiv \frac{\omega + c_T}{\alpha - \beta + \gamma - \rho} \tag{42}$$

$$\pi_{B_0} \equiv 0 \tag{43}$$

$$\tau_{B_0} \equiv 1 \tag{44}$$

Solution C_0: This case corresponds to $\tau(t) \in (0,1)$ and $\pi(t) = 0$. The steady state

solution is then

$$I_{C_0} \equiv \frac{\alpha\omega + c_T(\beta - \gamma - \rho)}{2\beta c_T} \tag{45}$$

$$\lambda_{C_0} \equiv \frac{-c_T}{\alpha} \tag{46}$$

$$\pi_{C_0} \equiv 0 \tag{47}$$

$$\tau_{C_0} \equiv \frac{-\alpha\omega + c_T(\beta - \gamma + \rho)}{2\alpha c_T} \tag{48}$$

Based on these values, some important observations follow:

Proposition 10. *(i) Steady states with positive treatment have lower disease prevalence than steady states with no treatment, i.e.* $I_A > I_B$ *and* $I_{A_0} > I_{B_0}$. *(ii) Steady states with positive prevention have lower disease prevalence than steady states with no prevention, i.e.* $I_A < I_{A_0}$ *and* $I_B < I_{B_0}$.

Proof: Part (i) follows from direct inspection. Part (ii) follows from the fact that the conditions that ensure that the no prevention steady state prevalence levels are higher than the positive prevention steady state prevalence levels, are exactly the opposite of the conditions that must hold for prevention to be zero in the no-prevention steady states ■

These results are not trivial, since prevention and treatment both work to reduce infection. It is therefore conceivable that the lack of one instrument is compensated for by an increase in the other instrument to the extent that prevalence ends up at a lower level than it otherwise would have been.

The next result follows from direct inspection of the relevant steady state prevention levels:

Proposition 11. *In the steady states with positive prevention, the no treatment steady state involves more prevention than the full treatment steady state, i.e.* $\pi_A > \pi_B$.

We can summarize the ranking of the steady state prevalence levels as follows:

$$I_B \leq \min\{I_A, I_{B_0}\} \leq \max\{I_A, I_{B_0}\} \leq I_{A_0}$$

The prevalence levels I_A and I_{B_0} are not unambiguously ranked.[11] But the condition that ensures that $\pi_{B_0} = 0$ implies the condition that ensures that $I_A \geq I_{B_0}$.

When multiple steady states coexist, we can talk of a high prevalence steady state and a low prevalence steady state. In the former, prevention is at a high level while treatment is at a low level. In the latter, prevention is at a low level while treatment is at a high level.

An important observation is in order. In the steady states involving no prevention, i.e. (A_0, B_0), steady state disease prevalence levels depend *only* on biological parameters that characterize the disease (such as infectiousness and recovery rate) and not on the economic parameters (such as costs and payoffs). In contrast, in the steady states involving positive

[11] It is easy to check that $I_A \geq I_{B_0}$ if and only if $c_P \geq \omega\left(\frac{\beta-\gamma-\alpha}{\beta-\gamma-\alpha+\rho}\right)$.

prevention, i.e. (A, B), steady state disease prevalence levels depend on *both* the biological and the economic parameters of the problem. In fact, in the no prevention steady states, the prevalence levels closely mirror those of the endemic steady state of the classical model. In the steady state with no treatment, the correspondence is exact, whereas in the steady state with full treatment, the effective recovery rate is modified to $(\gamma + \alpha)$.

5. Description of the Dynamics

In order to clearly draw out the effects of treatment and prevention on the overall behavior of the system, it is instructive to consider the polar cases in which there is either treatment or prevention alone. When the only feasible intervention is treatment, the system will display a similar overall behavior as the present model. Specifically, there will (subject to the right parameter constellations) be two corner steady states, one with low disease prevalence (or eradication) and another with high disease prevalence. In the former, the optimal policy is to fully treat whereas in the latter, the optimal policy is to not treat at all. As in the present model, there is an interior unstable steady state which is a spiral source (see Rowthorn, 2006 and Toxvaerd, 2009a). The upshot of this is that the presence of prevention does not alter the steady state levels of treatment, although it does alter the steady state levels of disease prevalence and the equilibrium paths.

Turning to a model with prevention only, in such a setting it turns out that there is a unique steady state, in which the optimal policy (i.e. level of preventive effort) is interior and in which disease prevalence is at an intermediate level (see Toxvaerd, 2010). It follows that the presence of treatment has a very stark effect on the system, influencing both the steady state levels and the equilibrium paths, but also the number of steady states.

The key to understanding the differences between treatment and prevention is to consider how the marginal benefits of each instrument depend on disease prevalence. In the case of prevention, the marginal benefits are *increasing* in prevalence: other things being equal, higher disease prevalence increases the risk of infection for susceptible individuals and hence increases the return from prevention.

Turning to treatment, the time profile of the benefits is more complex than that for prevention in that the benefits accrue in the future. Treatment increases the proportion of time that a typical individual will spend in the susceptible state. For a given susceptible individual, the probability of infection (or reinfection) is proportional to disease prevalence. The value of treating an individual in the present is therefore a decreasing function of future prevalence. As current treatment is increased, future prevalence decreases, making current and future treatment even more attractive. This virtuous circle (which is formally a complementarity property of the planner's problem) means that with treatment, the marginal benefits are decreasing in prevalence. This is exactly what creates the scope for multiple steady states. In the low infection steady state, the marginal benefits from treatment are high and treatment is thus exerted at the highest possible level, thereby maintaining low infection. In the high infection steady state, the marginal benefits of treatment are low and therefore there is no treatment at all. This keeps the infection at a high level.

Once both instruments are available, the forces described above are essentially superimposed. The presence of treatment creates the potential for multiple steady states, even in the presence of prevention (although the levels are altered accordingly). In the full treatment steady state, disease prevalence is relatively modest. But this means that the

marginal benefit of prevention is relatively low, resulting in a low steady state level of prevention. In contrast, in the no treatment steady state, disease prevalence is relatively high, leading to high marginal benefits of prevention. As a consequence, the prevention level is relatively high.

The coexistence of these effects has curious implications, as the following feature of the system shows. For sufficiently low prevention costs, the unique steady state involves a low level of infection (point B). In this steady state, there is full treatment of infected individuals and relatively little preventive effort, targeted at susceptible individuals. In contrast, for sufficiently high prevention costs, the unique steady state involves a high level of infection (point A). In this steady state, there is no treatment at all but a relatively high level of prevention. On the face of it, this seems to be highly counter-intuitive and even Giffenesque.[12]

How does one make sense of the observation that higher prevention costs lead to a steady state with a higher level of prevention? Before answering this question, we note that a similar result holds with respect to the treatment costs. Namely, for low enough treatment costs, the unique steady state is the high prevalence steady state with no treatment, while for sufficiently high treatment costs, the unique steady state is the low prevalence steady state with full treatment of infected individuals.

An explanation of this seemingly paradoxical phenomenon is as follows. For sufficiently high costs of disease reduction, i.e. high treatment or prevention costs, the steady state will involve a high level of infection. But for high levels of infection, the marginal benefits of treatment are low, while the marginal benefits of prevention are high. Consequently, the optimal policy in such a steady state is to have no treatment but to have a relatively high level of preventive effort. Similarly, for low costs of disease reduction, the steady state will involve a low level of infection. For low prevalence levels, the marginal benefits of treatment are high, while the corresponding marginal benefits of preventive effort are low. Thus in such a steady state, the optimal policy prescribes full treatment coupled with a more modest level of prevention. In each steady state, prevention and treatment are imperfect substitutes and the intervention with the highest marginal benefits dominates in the optimal policy mix.

5.1. Informal Bifurcation Analysis. Following Wagener (2003), we can usefully divide the parameter space into three different regimes as follows. In Regime I, there is a unique optimal steady state from the set $\{A, B, A_0, B_0\}$. Which of these is feasible depends on the particular parameter constellation in question. In Regime II, there are four potential pairs of stable equilibria, namely $\{(A, B), (A_0, B_0), (A, B_0), (A_0, B)\}$, each possibly with an accompanying unstable equilibrium from the set $\{C, C_0\}$. From each such pair of stable steady states, one or the other equilibrium is always optimal, i.e. is the end point of an optimal path for all initial conditions (i.e. the steady state is *globally* optimal). In Regime III, there are again four possible pairs of stable equilibria (possibly with corresponding unstable equilibria) like in Regime II, but different initial conditions render different equilibria optimal. In this scenario, there is an indifference (or Skiba) point $I_S \in (0, 1)$ such that for prevalence levels above this threshold, the high infection

[12]These comments do not apply to the fixed points (A_0, B_0), since these involve no prevention. In these cases, high treatment costs lead to a low treatment steady state being feasible, while low treatment costs lead to a high treatment steady state being feasible. In either case, sufficiently large prevention costs ensure that there is no prevention in steady state.

steady state is optimal, while for prevalence levels below it, the low infection steady state is optimal.

Even though the interior solutions cannot be end points of optimal paths, it is tempting to think that they demarcate intervals of the state variable from which it is optimal to go to one steady state or the other. For example, it might seem natural that for prevalence levels $I(t) < I_C$, the optimal policy is to go to the low infection steady state I_B while for prevalence levels $I(t) > I_C$, the optimal policy is to go to the high infection steady state I_A. In fact, this turns out to be wrong. While the optimal policy may indeed have the threshold character just described, the critical prevalence level I_S is generically different from the interior steady state.[13]

For a given set of parameters, it is a routine matter to check the conditions in Appendix B and determine whether Regime I obtains or not. In order to determine whether the system is in Regime II or III, there is no option but to compute values along all (typically two) paths satisfying the necessary conditions for optimality. This is because the existence of the indifference (or Skiba) point that distinguishes Regimes II and III cannot be formally characterized by a local condition in the same way that local extrema can (see Deisssenberg et al. 2004). This is so since the indifference point is obtained as the point of intersection of two functions for which there are no closed form solutions, namely the value functions evaluated along the different candidate paths.[14]

To emphasize the richness of possibilities, note how the number and character of equilibria of the model changes when one moves through the different possible parameter constellations of the model. The following possibilities can occur: (i) There is a unique steady state in which there is low prevalence, little prevention and high treatment; (ii) there are two steady states, one with low prevalence, low prevention and high treatment and another with high prevalence, high prevention and low treatment; it is always optimal to converge to the former steady state; (iii) there are two steady states, one with low prevalence, low prevention and high treatment and another with high prevalence, high prevention and low treatment; which steady state is optimal depends on the initial conditions; (iv) there are two steady states, one with low prevalence, low prevention and high treatment and another with high prevalence, high prevention and low treatment; it is always optimal to converge to the latter steady state; (v) there is a unique steady state in which there is high prevalence, high prevention and low treatment. In all cases except (iii), it is easy for the planner to determine where to steer the system, but the optimal policy mix must still be determined. In case (iii), there is the additional complication that there are two competing steady states and the planner must therefore compute the values of steering the system efficiently to either steady state and then compare these.

5.2. Optimal Paths, Spiral Sources and Limit Cycles. Although the interior points C and C_0 cannot be end points of optimal paths, it is necessary to consider the behavior of paths starting at these points. Our simulations show that such paths may be spirals, but formally showing that this is the case is complicated by the fact that standard results for the local behavior around such points do not apply to our problem. This is due to the discontinuities in the optimal policies in steady state. In characterizing the candidate solutions for optimal paths, there is a further potential complication, namely

[13] This property *does* hold when the Hamiltonian is concave, as described in Deissenberg (2004).

[14] Wagener (2003) develops sufficient conditions for such a point to exist and be unique.

the possibility that the paths close to the interior steady states constitute limit cycles (i.e. closed orbits around the interior point). We will now show two results. First, we show that the interior solutions are indeed spiral sources, i.e. exploding spirals. We prove this result by appealing to a theorem due to Wagener (2003), which excludes limit cycles. We then extend his reasoning to exclude that the interior points are spiral sinks. By implication, the points must be spiral sources. Second, having established the spiraling nature of paths originating at the interior solutions, we characterize the candidate optimal paths.

Proposition 12. *The points C and C_0 are clock-wise spiral sources.*

Proof: The proof is in two parts. In Appendix E, we prove that the movement around the interior solutions is characterized by clock-wise rotation. In Appendix F, we show that the movement is necessarily an exploding spiral ■

As discussed earlier and emphasized by the fact that the interior points are spiral sources, the Hamiltonian conditions do not pin down candidate optimal paths uniquely. It turns out that there is a simple way to determine these from a given spiraling path, as the next result shows:

Proposition 13. *A candidate optimal path starting at the prevalence levels associated with points C or C_0 is the highest or lowest monotone segment of the spiral.*

Proof: See Appendix G ■

Since we know that optimal paths may form part of an explosive spiral, this result is of direct practical importance.

In our simulations, we have identified the following interesting pattern. In Regime II, where one steady state dominates the other steady state for all initial conditions, the candidate optimal path to one steady state forms part of a spiral, whereas the candidate optimal path to the other does not. In both scenarios, the non-spiraling path turns out to be the optimal one. In Regime III, i.e. the case in which there is a Skiba point, paths to both steady states form parts of nested spirals emanating from a common source. Wagener (2003) and Mäler et al. (2003) show that if there are two nested spirals that lead to distinct equilibrium points, then there exists a unique Skiba point, which is also what we find in simulations. Of course, this does not a priori mean that if there is only one spiraling path, then there is necessarily not a Skiba point. While we have not attempted a formal analysis of these observations in our setting, these seem worthwhile pursuing in future work.[15]

To conclude, we have found that the fixed points (A, B, A_0, B_0) are saddle points (if feasible), while the fixed points (C, C_0) are spiral sources.

[15] Note however that *when* there are two spiraling paths to the high and low infection steady states respectively, then the results of Wagener (2003) and Mäler et al. (2003) apply and there exists a unique Skiba point. This observation formally confirms a similar point made by Goldman and Lightwood (2002).

5.3. Substitutes, Complements and Speeds of Convergence. In a static model, a common definition of complementarities is that an increase in the level of one instrument increases the marginal rate of return on the other instrument. An important question in the present context is whether prevention and treatment display a similar property. For non-linear multiple-instrument optimal control problems, there are instances in which one may cleanly characterize "synergies" between the control variables, i.e. instances in which raising one control variable makes it more desirable to also raise the other (see Feichtinger, 1984). In the present model, the desirability of increasing one instrument depends on the level of the other instrument through its effects on disease prevalence. In fact, changing the level of *either* instrument influences disease prevalence, which in turn changes the desirability of further changing *both* instruments.

To see this, consider an increase in the level of prevention. Such an increase will decrease disease prevalence, thereby increasing the marginal benefits of treatment, but also decreasing the marginal benefit of prevention. Similarly, an increase in treatment will cause a decrease in disease prevalence, thereby increasing the marginal benefits of treatment, but decreasing the marginal benefits of prevention.

These interactions are simply a reflection of the insight that treatment induces a positive feedback effect, whereas prevention induces a negative feedback effect.

Almost no existing work discusses the optimal phasing of prevention and treatment. An exception is Gersovitz and Hammer (2004), who arrive at the conclusion that

> "...[optimal] subsidization [to treatment and prevention] is at equal rates because it is equally beneficial in preventing further infection to get a person out of the infected pool as to have prevented the person from getting into it in the first place [...]"

This statement seems to suggest that treatment and prevention are perfect substitutes in the steady state of their model that they consider. Our analysis shows that prevention and treatment are imperfect substitutes. Having said that, there are clearly instances in which the two instruments are used in conjunction. This stems from the fact that at some levels of disease prevalence, the strength of substitutability is low enough to render the use of both instruments optimal. This observation is intimately connected to the property of optimal paths being of the most rapid approach variety (MRAPs for short), to which we turn next.

When each policy is considered in isolation, optimal paths are known to be of this type in the prevention model but not in the treatment model (see Toxvaerd 2009a, 2010).[16] But in the present setting, this is not necessarily the case. The reason lies in the fact that the marginal benefits of treatment are decreasing in prevalence whereas the marginal benefits of prevention are increasing in prevalence. This feature of the planner's problem implies that when approaching a steady state from below and starting from very low prevalence levels, the optimal policy may involve no prevention coupled with full treatment of the (relatively few) infected individuals. As discussed earlier, this is because for low prevalence levels, the probability of reinfection is relatively modest, making treatment worthwhile, but prevention suboptimal. This implies that infection is not increasing as

[16] More precisely, paths are always MRAPs in a setting in which recovery can only happen via treatment. If there is also spontaneous recovery, then the optimal path to the steady state from above involves no treatment, which is not an MRAP.

fast as it could. Once prevalence has increased to a level that makes further treatment undesirable, the path does become a MRAP. Similarly, when approaching a steady state from above, the optimal path may involve no treatment even though there is full prevention. Again, this is because for very high prevalence levels, reinfection probabilities are so high that treatment becomes suboptimal but the marginal benefits of prevention are high enough to justify using this instrument to its fullest extent. But this means that disease prevalence does not decrease as fast as possible towards its steady state level. When (and if) prevalence has decreased to a level that makes treatment optimal, the remaining path also becomes a MRAP. In Regime III, i.e. in the case where there is a Skiba point, there is also an interior region in which optimal paths are not most rapid approach paths.

Formally, any path that spends time in areas in which $(\tau(t), \pi(t)) = (0, 1)$ or $(\tau(t), \pi(t)) = (1, 0)$, are not of the most rapid approach type. The same is true for any decreasing path in the area $(\tau(t), \pi(t)) = (1, 0)$. This implies the following observations:

Proposition 14. *(i) The optimal path to point A from the right is not a MRAP, while the optimal path from the left is potentially a MRAP. (ii) The optimal path to point B from the left is not a MRAP, while the optimal path from the right is potentially a MRAP. (iii) The optimal path to point A_0 from the right is not a MRAP, while the optimal path from the left is potentially a MRAP. (iv) Optimal paths to point B_0 are not MRAPs from either direction.*

We can further state the following:

Proposition 15. *For all paths that are potentially MRAPs, the closing segments of the paths are MRAPs.*

The previous two propositions deserve some further comments. As can be seen from 3, all paths described as "potential MRAPs" *may* involve initial segments in which the system does not approach the steady state as fast as possible. It is in this sense that they are *potentially* most rapid approach paths. Having said that, all these paths share the feature that as the system moves close enough to the steady state, the paths enter regions where they *do* approach steady state as rapidly as possible. Thus, although some paths are not MRAPs along their entire length, their closing segments have this property.

We now turn to the behavior of the system close to the steady states. The speed of convergence towards a steady state (I^*, τ^*, π^*) is found via the first-order Taylor approximation[17] of the logistic growth equation around the steady state, i.e.

$$\sigma(I^*, \tau^*, \pi^*) \equiv -\left[(1-\pi^*)\beta(1-2I^*) - \alpha\tau^* - \gamma\right] \tag{49}$$

Because the optimal amount of preventive effort may have a discontinuity at some steady states, we need to distinguish speeds of convergence when approaching the steady state from the left and from the right respectively. We will denote by $\sigma_-(I^*, \tau^*, \pi^*)$ and $\sigma_+(I^*, \tau^*, \pi^*)$ the speeds when approaching from the left and right respectively, and

[17] It is given by the equation

$$\dot{I}(t) \approx I^* \left[(1-\pi^*)\beta(1-I^*) - \gamma - \alpha\tau^*\right] + (I(t) - I^*)\left[(1-\pi^*)\beta(1-2I^*) - \alpha\tau^* - \gamma\right]$$

$\sigma(I^*, \tau^*, \pi^*)$ when there is no need to distinguish the direction (because the two speeds coincide). With this notation, the speeds are given as follows:

$$\sigma_+(I_A, 0, 1) = \gamma \tag{50}$$

$$\sigma_-(I_A, 0, 0) = \gamma + \frac{c_P(\beta + 2\rho) - \beta\omega}{\omega - c_P} \tag{51}$$

$$\sigma_+(I_B, 1, 1) = \alpha + \gamma \tag{52}$$

$$\sigma_-(I_B, 1, 0) = \alpha + \gamma + \frac{c_P(\beta + 2\rho) - \beta(\omega + c_T)}{c_T + \omega - c_P} \tag{53}$$

$$\sigma(I_{A_0}, 0, 0) = \beta - \gamma \tag{54}$$

$$\sigma(I_{B_0}, 1, 0) = \beta - \gamma - \alpha \tag{55}$$

It should be emphasized that these speeds of convergence are approximations that are valid only close to the steady states in question. In particular, this means that the speed of approach of paths that contain an initial non-MRAP segment may be overstated.

Second, it is interesting to note that there is no unambiguous ranking of the speeds of convergence from the left and right to points A and B. In other words, it is not generally true that descending to points A or B with the aid of full prevention is faster than ascending to points A or B with no prevention. It depends on the cost of prevention and the relevant conditions are not implied by any of the other constraints we have maintained.[18]

6. Comparative Analysis and Welfare

The main focus of the present paper is the optimal control of infectious diseases through prevention and treatment, taking the efficiency of these interventions as given. In other words, the parameters β, α and γ are not directly controlled. Some interventions, such as the administration of antiretroviral drugs to non-infected individuals, can be interpreted as a direct change in the infectiousness of the disease (see Toxvaerd, 2010 for a discussion and a survey of that literature). It is thus also of interest to conduct comparative statics analysis with respect to these parameters and to analyze their welfare and policy implications. We shall do so in this section.

From the steady state levels listed above, the following results immediately follow:

Proposition 16. *(i) In steady states with no prevention, steady state prevalence is increasing in infectivity and decreasing in the rate of recovery. (ii) In steady states with positive prevention, steady state prevalence is decreasing in infectivity and independent of the rate of recovery.*

While infectivity is always measured by β, the rate of recovery may be γ or $(\gamma + \alpha)$, depending on steady state treatment intensity.

These results have important and surprising policy implications. They show that in the absence of prevention, the steady state comparative statics of disease prevalence with respect to infectiousness and the recovery rate, are qualitatively the same as those in the classical model. But surprisingly, when the steady state involves positive preventive

[18] Specifically, we have that $\sigma_+(I_A, 0, 1) > \sigma_-(I_A, 0, 0)$ if and only if $c_P < \frac{\beta\omega}{\beta+2\rho}$. Also, $\sigma_+(I_B, 1, 1) > \sigma_-(I_B, 1, 0)$ if and only if $c_P < \frac{\beta(\omega+c_T)}{\beta+2\rho}$.

effort, the comparative statics results are *reversed*. This is an important observation, because the decrease in infectiousness and the improvement in therapeutic technologies are an important vehicle through which medical scientists and epidemiologists seek to control epidemics. What the present results show, is that changing the basic biological parameters through direct intervention may have unexpected consequences.

To fully draw out the welfare and policy implications, we first derive two further results. First, we consider the overall welfare effects of such parameter changes and then consider the effects on steady state welfare. With these results in hand, we will be able to give a sharp characterization of the welfare tradeoff involved in changing the biological and medical parameters.

Consider the overall effects of parameter changes on welfare. These are captured by changes in the optimal value function. We have the following results:

Proposition 17. *(i) An increase in infectiousness β decreases overall welfare. (ii) An increase in the rate of recovery $(\gamma + \alpha)$ increases overall welfare.*

Proof: From the dynamic envelope theorem, it follows that in some steady state $(I^*, \tau^*, \pi^*, \lambda^*)$, the effect of a change in a parameter x is given by[19]

$$\frac{\partial V^*(I_0)}{\partial x} = \int_0^\infty \frac{\partial H(I^*, \tau^*, \pi^*)}{\partial x} dt$$

Therefore we have that

$$\begin{aligned}
\frac{\partial V^*(I_0)}{\partial \beta} &= \int_0^\infty \lambda^* I^* (1 - I^*)(1 - \pi^*) dt < 0 \\
\frac{\partial V^*(I_0)}{\partial \alpha} &= -\int_0^\infty \lambda^* I^* \tau^* dt \geq 0 \\
\frac{\partial V^*(I_0)}{\partial \gamma} &= -\int_0^\infty \lambda^* I^* dt > 0
\end{aligned}$$

and the result follows ■

It should be noted that the results with respect to α are strict only when the treatment level is positive (and weak if the treatment level is zero).

The comparative dynamics results with respect to α, γ, β are hardly surprising. They also follow from a simple revealed preferences argument, as noted in Toxvaerd (2010). Consider a decrease in β or an increase in either α or γ. Ceteris paribus, infection is now easier to control and the planner can always choose the same paths for disease prevalence and the policy instruments as before the change in parameters. Thus overall welfare cannot be lower after the decrease in infectiousness or the increase in the rate of recovery.

It turns out that the gains in overall welfare may have an unexpected source, depending on the steady state in question. To see this, we first determine the effects of parameter changes on steady state welfare. We find the following results:

Proposition 18. *(i) In steady states with no prevention, steady state welfare is decreasing in infectivity and increasing in the rate of recovery. (ii) In steady states with positive*

[19] In this result, the Hamiltonian is first differentiated with respect to the parameter and only then is the resulting expression evaluated at the relevant steady state values. See Caputo (2005) for details.

prevention, steady state welfare is increasing in infectivity if $\rho + \alpha > \gamma$ *and increasing in the rate of recovery.*[20]

Proof: The steady state levels of welfare associated with the non-interior steady states are given as follows:

$$H(I_A, \tau_A, \pi_A, \lambda_A) = \frac{-c_P(\beta - \gamma + \rho)}{\beta} \tag{56}$$

$$H(I_B, \tau_B, \pi_B, \lambda_B) = \frac{-c_P(\beta - \gamma + \rho - \alpha)}{\beta} \tag{57}$$

$$H(I_{A_0}, \tau_{A_0}, \pi_{A_0}, \lambda_{A_0}) = \frac{-\gamma\omega}{\beta} \tag{58}$$

$$H(I_{B_0}, \tau_{B_0}, \pi_{B_0}, \lambda_{B_0}) = \frac{(\alpha - \beta + \gamma)(c_T + \omega)}{\beta} \tag{59}$$

The results then follow from inspection ■

Again, note that the results with respect to α are strict only when the treatment level is positive (and weak if the treatment level is zero).

Taken together, these above results have interesting implications. Start from a situation in which the system is in steady state and consider an decrease in infectiousness β. Assume furthermore that this change does not cause a shift in regime, so that the set of equilibria and their optimality remains unchanged.

In steady states without prevention, i.e. (A_0, B_0), a decrease in β causes both overall welfare and steady state welfare to increase. On the other hand, the new steady state level of disease prevalence is lower, so the planner may have to expend resources on forcing down prevalence through additional treatment, until steady state is reached.[21] Since overall welfare is higher, the extra costs borne during the transition are outweighed by the increase in the resulting steady state welfare (both suitably discounted).

In steady states with positive prevention, i.e. (A, B), a decrease in β must also increase overall welfare, as we have seen. But we also know that such a decrease in infectiousness actually decreases steady state welfare. The upshot of this is that all gains in overall welfare stem from the transition to the new steady state. Indeed, since decreasing β increases steady state prevalence when prevention is positive, the planner forces prevalence up by reducing the level of preventive effort. The cost savings associated with not having any prevention during the transition to the new steady state are so large, that they outweigh the losses in steady state welfare (both suitably discounted).

To sum up, decreasing infectiousness must always improve overall welfare. But in order to reap the benefits of lower infectiousness, the planner must pay special attention to the steady state the system is in. In some steady states, the optimal policy response is to *reduce* prevalence through increased treatment, trading a short term increase in infection

[20] This condition ensures the stated result (on the effects of changes in infectiousness) for steady state B. The weaker condition $\rho > \gamma$ ensures that the result holds for steady state A. We also note that the conditions that ensure that steady state welfare in steady states A and B is increasing in infectivity β are sufficient conditions for the shadow values of infection being negative in steady states A_0 and B_0 respectively.

[21] This is the case if starting at point B_0. If starting at point A_0, the decrease will happen without further costly infection control.

control costs for a long term increase in steady state welfare. In other steady states, the optimal policy response is conversely to *increase* prevalence through a reduction in prevention, trading short term cost savings from reduced infection control for a long term decrease in steady state welfare.

Turning to changes in the efficiency of treatment α, some interesting patterns emerge. While changing the infectiousness parameter β could have opposing effects on overall welfare and steady state welfare, changes in α never move these two welfare measures in opposite directions. In steady states (A, A_0), there is no treatment and thus both overall welfare and steady state welfare are in fact independent of α. There are therefore no tradeoffs to consider. In steady states (B, B_0), there is full treatment and therefore overall welfare and steady state welfare are (increasing) functions of α. In this case, there is no tradeoff between the short term costs and steady state welfare since the new steady states (if different) are reached without any changes in the steady state levels of the policy instruments.

To sum up, whether steady state prevalence changes as the efficiency of treatment α is varied, depends on whether there is any prevention in steady state. In contrast, whether such a change in efficiency has any impact on welfare (overall or in steady state), depends on whether there is any treatment in steady state.

The results show that the key ingredient in creating rational disinhibition (as discussed in Toxvaerd, 2010 and Gersovitz, 2010) is prevention rather than treatment, as it is the former that gives rise to the non-classical comparative statics results.

For completeness, we would also like to comment on a seemingly counter intuitive feature of steady states with positive preventive effort. Whereas the steady state welfare levels in points (A_0, B_0), in which there is no prevention, are functions of all the relevant deep parameters, the corresponding values for points (A, B) are not. In particular, steady state welfare in point A is independent of the health premium ω whereas in point B, it is independent of both the health premium ω and the treatment cost c_T.[22] The reason for this feature is that the optimal prevention level in these steady states are such that they exactly counterweight these parameters. In other words, the parameters are present in the optimal prevention levels, which in turn cancels out these parameters in the expressions for steady state disease prevalence.

7. Simulated Paths and Steady States

In order to further illustrate the results of the preceding sections, we now consider some sample simulations of optimal paths and steady states. The simulations were done using a fourth-order Runge-Kutta procedure with the following parameter values:

Parameters	α	β	γ	ω	ρ	c_P	c_T
Values	$\{0.2, 0.4, 0.5\}$	3	0.1	1	0.11	0.5	10

With this choice of the parameters $(\beta, \gamma, \omega, \rho, c_P, c_T)$, the feasible steady states are (A, B, C) and the system is either in Regime II or III, depending on the magnitude of the efficiency of treatment α. This means that both the low and the high infection steady

[22] That point A is independent of c_T is not surprising since this steady state involves no treatment.

states exist. The following table shows the ranges for α where each regime obtains:

Interval	$\alpha \in [0, 0.3]$	$\alpha \in [0.3, 0.41]$	$\alpha \in [0.41, 1]$
Opt. steady state	Point A (Reg. II)	Point A or B (Reg. III)	Point B (Reg. II)

In Example 1, $\alpha = 0.2$ and it is optimal to pursue the path to steady state A for any initial level of disease prevalence (this case is in Regime II). The paths to the two steady states A and B are illustrated in the upper part of Figure 4. In the lower part, we show the total discounted value of following the paths to steady states A and B respectively, for different initial prevalence levels. It is clear from this figure that the value of going to (and staying at) point A is everywhere higher than the value of going to (and staying at) point B.

In Example 2, $\alpha = 0.5$ and it is optimal to follow the path to steady state B for any initial prevalence level (this case is also in Regime II). The paths to A and B are shown in Figure 5, which also shows the corresponding values of following the different paths. It is clear from the figure that going to (and staying at) point B always dominates going to (and staying at) point A.

In Example 3, $\alpha = 4$ and the system is in Regime III in which the optimal steady state depends on the initial level of infection. This case is illustrated in Figure 6. For prevalence levels below $I_S = 0.1629$, the optimal path leads to the low infection steady state B while for prevalence levels above $I_S = 0.1629$, the optimal path leads to the high infection steady state A. Thus for this parameter constellation, the optimal path is history dependent in the sense that the initial conditions matter for where it is optimal for the system to settle. Note that in the lower part of Figure 6, $I_S = 0.1629$ is the prevalence level at which the value functions for the paths to A and B intersect.

For completeness, note that the kinks in the optimal paths in the three graphs correspond to switches in the control regimes. The optimal policies corresponding to the paths in the three examples are summarized in the following table.

Example 1 ($\alpha = 0.2$)			**Example 2** ($\alpha = 0.5$)		
Optimal path goes to A	$\tau(t)$	$\pi(t)$	Optimal path goes to B	$\tau(t)$	$\pi(t)$
$I \in [0, 0.0031]$	1	0	$I \in [0, 0.0018]$	1	0
$I \in [0.0031, 0.0370]$	0	0	$I = 0.0018$	1	0.7996
$I = 0.0370$	0	0.9654	$I \in [0.0018, 0.0176]$	1	1
$I \in [0.0370, 1]$	0	1	$I \in [0.0176, 1]$	0	1
Example 3 ($\alpha = 0.4$)			**Example 3** ($\alpha = 0.4$)		
Optimal path goes to B	$\tau(t)$	$\pi(t)$	Optimal path goes to A	$\tau(t)$	$\pi(t)$
$I \in [0, 0.0017]$	1	0	$I \in [0.0163, 0.0370]$	0	0
$I = 0.0017$	1	0.7996	$I = 0.0370$	0	0.9654
$I \in [0.0017, 0.0115]$	1	1	$I \in [0.0370, 1]$	0	1
$I \in [0.0115, 0.0163]$	0	1			

It is interesting to note that when there is a globally optimal steady state, i.e. when the system is in Regime II, the path to the optimal steady state does not form part of a spiral, whereas the path to the sub-optimal steady state does.

In the Skiba case, i.e. in Regime III, the paths to both steady states form part of spirals emanating from the interior steady state.

For all three simulated cases, it is interesting to consider the corresponding optimal paths in terms of prevention and treatment levels. With reference to the discussions in earlier sections, a number of interesting patterns emerge. First, optimal treatment is decreasing in prevalence along optimal paths, while optimal prevention is increasing. Second, for extreme prevalence levels, the optimal treatment levels slow down the system's approach to the optimal steady state. Thus in general, it is not the case that optimal paths are of the most rapid approach variety. Third, paths approaching high prevalence steady states from below do so as rapidly as possible (if close enough to the steady state), while never when approaching steady state from above. Similarly, paths approaching low prevalence steady states from above do so as rapidly as possible (if close enough to the steady state) while never when approaching from below. This holds true in both Regimes II and III.

The previous exercise is a simple example of the kind of bifurcation analysis known from the shallow lake literature (see references in Section 1.1). In the basic shallow lake system, there are only two central parameters to vary (apart from the discount rate). In contrast, in the present model there is a much larger number of parameters to be chosen, making a systematic bifurcation analysis considerably harder to accomplish. Last, while the present model leads to bang-bang policies, the shallow lake system has policies that are continuous. This makes the present model more difficult to characterize.

8. Robustness and Extensions

The present model incorporates a number of implicit simplifying assumptions. In this section, we briefly touch on some of these and indicate when our results can be expected to carry over when these assumptions are relaxed. Indeed, some of these extensions seem to us to be good starting points for further exploration.

1. We have assumed that both the incubation period and the latency period have zero length. Furthermore, there is no uncertainty about individuals' health status. This means that individuals in each category, i.e. infected and susceptible, can be perfectly targeted for treatment and prevention respectively. Relaxing these assumptions seems worthwhile but not straightforward.

2. One simplifying assumption is that the model has no demographics, i.e. births and deaths. Adding these features do not seem to alter the analysis qualitatively. In the simple case in which infected individuals may die at some exogenous rate, but are immediately replaced by susceptible individuals in order to maintain stationarity, the formal model is identical to the one studied here, but with increased recovery rate. This is because death in this case corresponds to immediate recovery. From a welfare perspective, introducing deaths from infection decreases the losses due to infection, making intervention less valuable. Adding births to the model should not change our analysis qualitatively. This is because the presence of future generations (whose welfare features in the planner's objective) would simply increase the benefits of both prevention and treatment, as these interventions not only protect current generations from infection, but also benefit future generations.

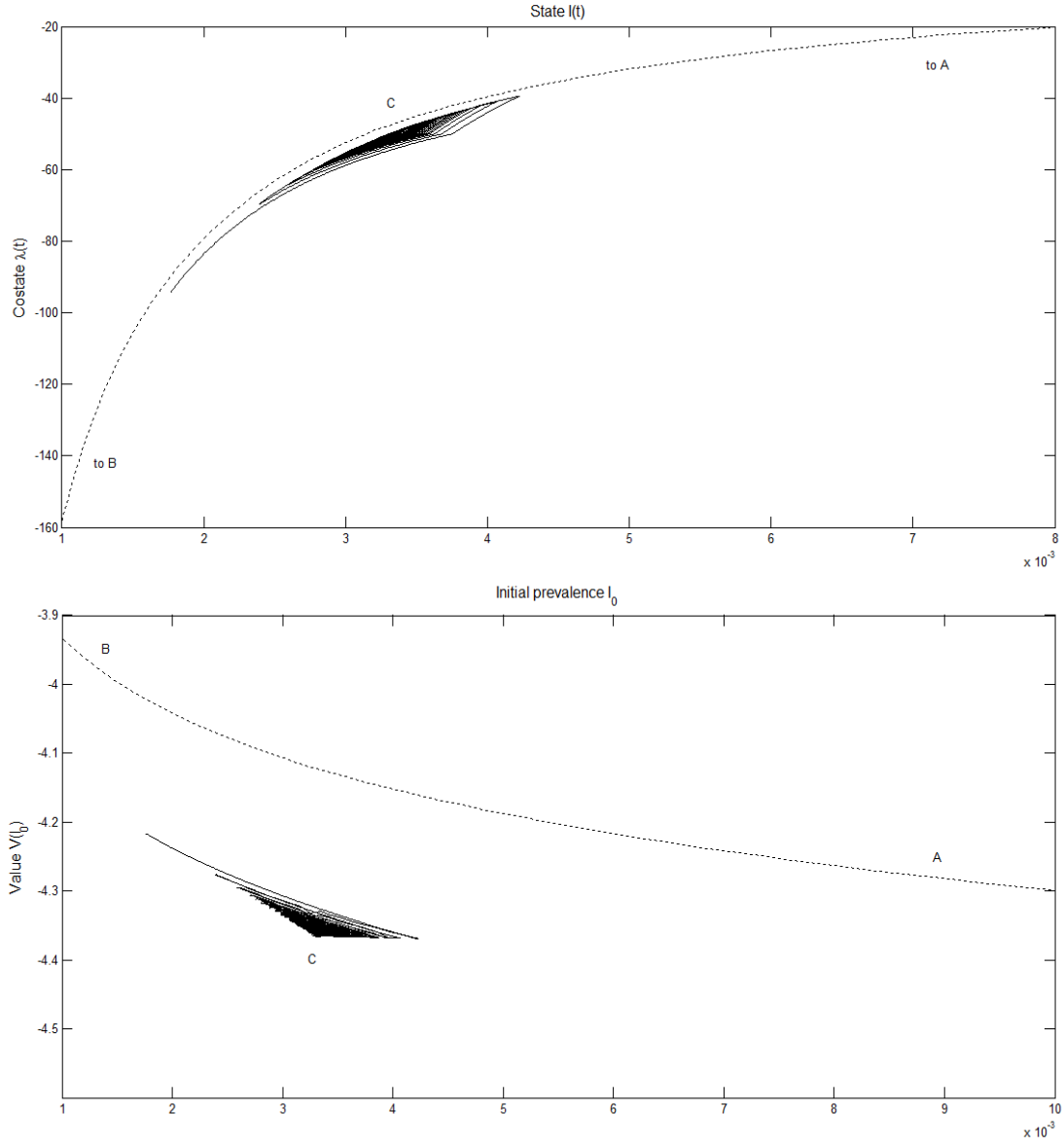

Figure 4: Optimal path and steady state with $\alpha = 0.2$. Path goes to steady state A.

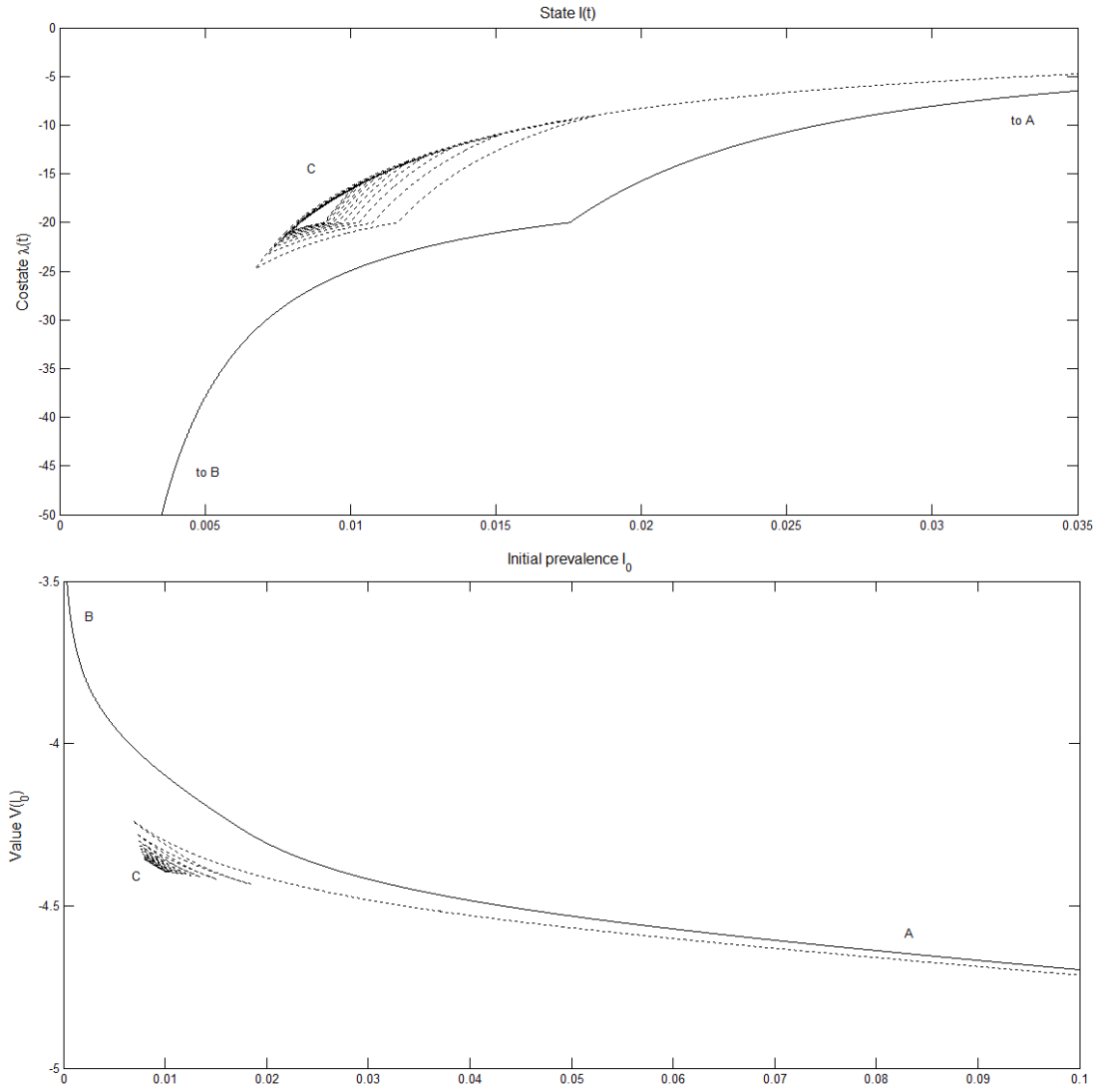

Figure 5: Optimal path and steady state with $\alpha = 0.5$. Path goes to steady state B.

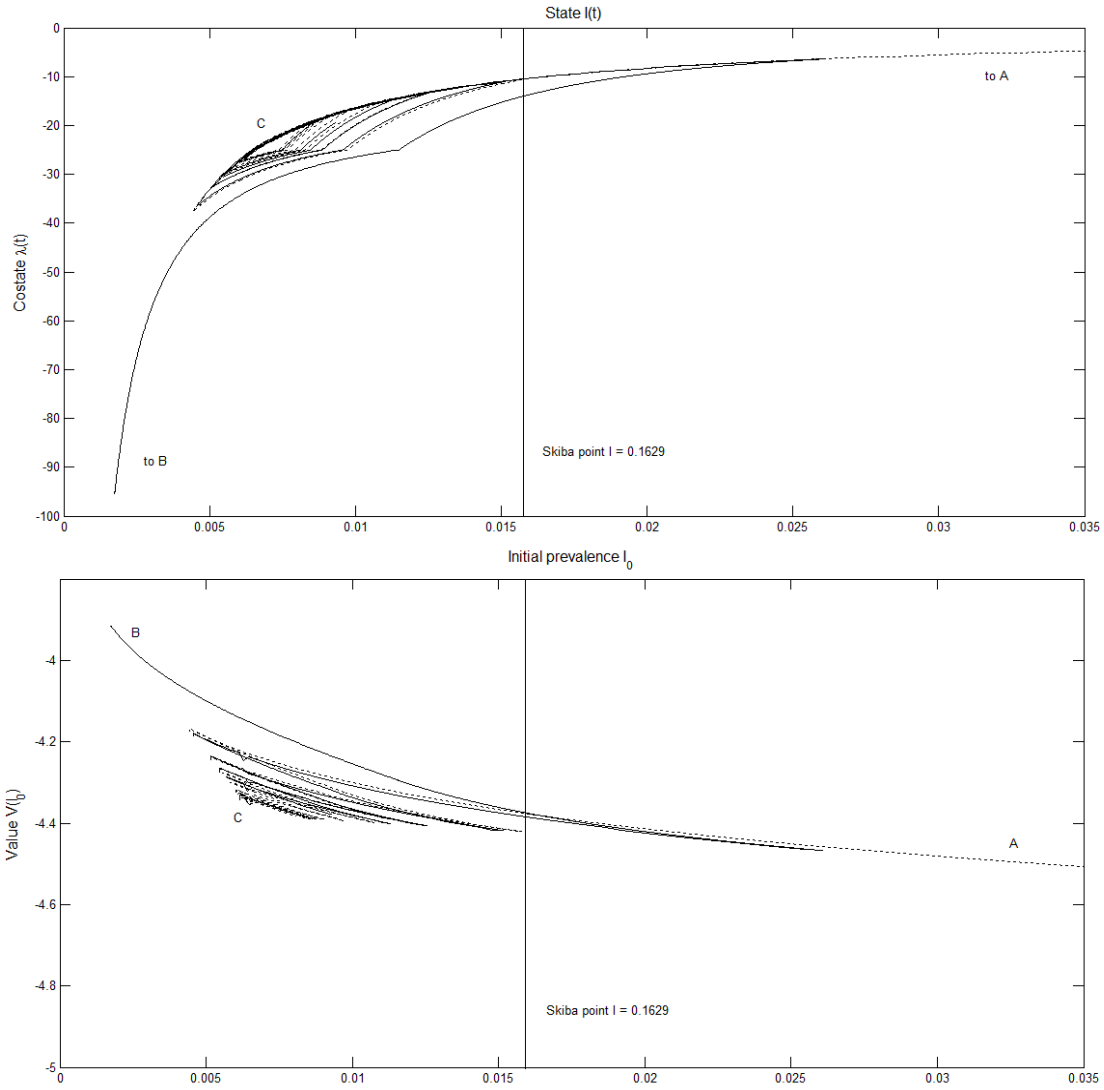

Figure 6: Optimal path and steady states with $\alpha = 0.4$. Path goes to steady state A for $I(t) > 0.1629$ and to steady state B for $I(t) < 0.1629$.

3. The vast majority of diseases fall under the broad category of susceptible-infected-recovered-susceptible models (or SIRS for short). The present analysis is conducted within the simpler SIS framework, but extends in a straightforward manner to the SIRS setting. The SIRS model is only quantitatively different from the SIS model that we consider, in that the benefits from recovery (both direct and indirect) are higher since temporary immunity is socially beneficial.

4. For simplicity, the population under consideration is assumed to be homogeneous in all respects. Heterogeneity can be modeled in a number of ways, with individuals differing in either the economic or the biological parameters. Heterogeneity will induce priority classes into the planner's problem, such that some individuals are treated or protected before others. But the basic forces at work are essentially those identified in our simplified setup. Heterogeneity can arise endogenously in the model because of features of the disease and the interventions. For example, if prevention provides some measure of protection over time (but not forever, as in vaccination), then there will at any given point of time be different kinds of non-infected individuals in the population. This corresponds to some version of the SIRS model discussed above. Similarly, if recovery confers partial immunity over a period of time, then this would also correspond to a version of the SIRS model.[23]

5. While our model covers a wide variety of infectious diseases, we have ruled out the possibility of immunity, acquired either through treatment, vaccination or spontaneous recovery. While such extensions are outside the scope of the present analysis, they are certainly worth pursuing. One complication with these extensions is that the number of state variables increases (which is well known from the analysis of SIR type models) and that such models do not have closed form solutions.

6. Due to the bang-bang nature of optimal policies, it may be conjectured that linearity of costs (in the number of individuals targeted for intervention) plays a central role in the analysis. This turns out not to be the case. As shown by Goldman and Lightwood (2002), when the SIS model is controlled through treatment, the positive destabilizing feedback present in our model remains in settings with convex costs. Since preventive effort is interior even with linear costs, clearly this result will be present with convex costs too. So linearity does not play an essential role, but does allow us to derive a number of properties of the model explicitly.

7. We would like to note an apparent contradiction in the literature, which relates to the monotonicity of optimal treatment policies. Using a dynamic programming approach, Sanders (1971) finds that optimal treatment intensity in an SIS environment is decreasing in disease prevalence. Specifically, he finds that for low levels of disease prevalence, it is optimal to treat all infected individuals while for high levels of disease prevalence, it is optimal to not treat anyone. Sethi (1974) revisits the Sanders analysis by using optimal control theory and focuses on the possibility of a singular solution, something not considered by Sanders (1971). Surprisingly, Sethi (1974) finds that for disease prevalence above the singular steady state, it is

[23] In the special case where recovery confers a permanently lower level of susceptibility to reinfection, the model reduces to a heterogeneous version of the SIS model.

optimal to treat everyone while for levels below it, it is optimal to not treat anyone. Goldman and Lightwood (2002) set out to generalize Sanders' result to non-linear cost structures but overlook the fact that their results are seemingly at odds with Sethi's findings.[24] Anderson et al. (2010) explicitly note the contradicting results but do not seeks to reconcile them. It turns out that the analysis in Goldman and Lightwood (2002) is not directly comparable to that of Sanders (1971) or Sethi (1974) since it relies on a subtly different model. All three contributions make similar assumptions about the benefits of treatment. Namely, they all assume that the social benefit of treatment is a decreasing function of prevalence, because treatment has fewer positive externalities when a large fraction of the population is already infected. However, they make different assumptions about costs. In Sanders (1971) and Sethi (1974), it is tacitly assumed that individual treatment cost is a sharply decreasing function of prevalence.[25] At high levels of prevalence, this cost is so low that full treatment is optimal, despite its limited social benefits. Goldman and Lightwood (2002) consider a variety of cost functions. In every case, at high levels of prevalence, the cost of treatment exceeds its benefits, so the optimal policy is to set treatment at zero. To sum up, our analysis formally confirms the monotonicity result in Goldman and Lightwood (2002), which is in turn an important independent result, rather than a generalization of earlier findings.

8. We have worked under the assumption that the population mixes homogeneously and we have done so for tractability. While we are believe that our results would reappear in some form or other in a wide variety of settings, we do not wish to make blanket statements about extensions in this direction. A possible extension that we find very appealing, is to consider optimal control via prevention and treatment on an explicit network.

9. In conducting our analysis, we have taken the perspective of a benevolent social planner that can dictate policies and does not have to consider the incentives of the individuals in the population. This raises the important question of the possible decentralization of optimal policy. This is an important, but not straightforward, extension of our analysis. One direction is to consider a representative agent, as in Goldman and Lightwood (2002) or Gersovitz and Hammer (2004). Alternatively, one may consider fully decentralized decision making by strategically sophisticated individuals and characterize the equilibrium outcomes in such a setting, as done in Toxvaerd (2009a) and Toxvaerd (2010) for the models with only prevention and only treatment, respectively. A full analysis of decentralized decision making seems to be a very difficult task to achieve. This is because the presence of treatment introduces an element of strategic complementarities, which may in turn create multiple (expectations-driven) equilibria (see Toxvaerd 2009a).

[24] In reviewing the work of Sanders (1971) and Sethi (1974), they state that "In those works, there is some *critical rate of infection* below which it is optimal to treat fully and above which treatment is set to zero."

[25] Sanders (1971) and Sethi (2004) assume that the total cost of treatment is independent of disease prevalence. Using Sethi's notation, let x be the total size of the infected population. In his model, it costs $K\gamma$ to cure γx individuals where $K > 0$ is some constant and γ belongs to some bounded interval of the positive real line. This works out at K/x for each individual who is cured.

10. The way we have modeled treatment and prevention, masks an implicit asymmetry in the effectiveness with which the two instruments reduce infection. While full prevention reduces incidence to zero instantaneously, full treatment only gradually reduces prevalence (as long as α is finite). In other words, while the rate of transition from infected to susceptible can be brought down only to a finite speed, the transition rate from susceptible to infected can be brought instantaneously to zero. It turns out that this asymmetry has only quantitative effects. We show this formally in Appendix H, where we describe the dynamics and derive the steady states of the model with imperfect prevention.

11. We have modeled preventive measures as efforts directed at the susceptible population. This type of intervention can be thought of as the use of condoms, in the case of sexually transmitted diseases. For other diseases, an alternative way to prevent the transmission of infection is to directly target the infected individuals. One instance of such a measure is a quarantine that cuts off infected individuals from the general population. In a sense, the way we have modeled prevention is tantamount to a quarantine of the susceptible individuals. The alternative scenario in which it is the infected that are quarantined is important and deserves further study. We outline such a model in Appendix I and note that the dynamics of such a model may differ from the present one in substantive ways.

12. We have focused attention on the infinite horizon case, in which the problem to be solved is stationary and autonomous. In Appendix A, we touch upon the finite horizon version of the model in which time runs until some terminal time $T < \infty$. As we discuss in the appendix, as long as the horizon is sufficiently distant, the qualitative analysis is unchanged. However, a critical time is eventually reached, after which the optimal paths in the finite and infinite horizon versions differ. The main qualitative difference is that in the finite horizon version, optimal paths or policies may fail to be monotone in time.

9. Conclusion

For the past four decades, the field of infectious disease control and public health has benefited from formal mathematical analyses of epidemics and their management through different policies that influence disease propagation. By employing techniques from dynamic optimization, economists have added valuable insights on how best to control infectious diseases and thereby informed public policy in this important field.

Although significant progress has been made in the analysis of single instrument models, such as those with vaccination, quarantines, condom use, mosquito nets or treatment, little is known about optimal disease control through multiple interacting instruments. One important question, from both a theoretical and a practical policy perspective, is to determine how different instruments and policies interact and how such interventions should be combined at different stages of the epidemic. Answering this type of question is the central aim of this paper.

Our analysis is not simply an abstract exercise, but one that has concrete, practical relevance to the formulation of policy. A case in point is the recent outbreak of swine flu. In early July 2009, the UK Department of Health announced that in its battle against the swine flu pandemic, it had now entered a "treatment phase" under which treatment was

to be the main policy instrument in controlling the outbreak of the disease.[26] It stated that

> "As swine flu spreads and more people start to catch it, it makes sense to move from intensive efforts to contain the virus to focusing efforts on treating the increasing number of people who have the disease."

While the exact reasoning behind this change of tack was not made explicit, it's clear from this statement that the relative desirability of treatment and prevention was thought by policy makers to be a function of disease incidence and prevalence.[27] To the best of our knowledge, there is no existing research that formally shows that such a policy switch may be optimal or that formally links the optimal policy mix to the level of disease prevalence or incidence.

In this paper, we have analyzed the optimal economic control of a susceptible-infected-susceptible model, in which a benevolent social planner can influence the rates of infection and recovery through costly intervention. Although this is a difficult problem, we have made significant progress in characterizing both the steady states of the system and the equilibrium paths. While out characterization is wholly analytical, we have added sample simulations of the system in order illustrate the dynamics and the optimal policies.

A number of results emerge from this analysis. First, treatment and prevention work in fundamentally different ways. Although both reduce infection, the former directly targets prevalence whereas the latter directly targets incidence. More importantly, we find that treatment induces a destabilizing positive feedback effect, since the marginal benefits of treatment are decreasing in disease prevalence. Since treatment reduces prevalence, the desirability of further treatment is increased as treatment efforts are intensified. This complementarity between current and future treatment efforts creates the potential for multiple steady states. In contrast, prevention induces a stabilizing negative feedback effect, since the marginal benefit of prevention is increasing in disease prevalence. This means that as preventive measures are intensified, prevalence levels decrease, thereby making further prevention less desirable. When these effects are superimposed, interesting interactions occur. This is evident in a number of different instances. For example, we find that the optimal policy will typically involve treatment when prevalence is low but no treatment when prevalence is high. Since prevention and treatment are imperfect substitutes, if there is any prevention at all, a low infection, high treatment steady state will be associated with relatively little prevention. Similarly, a high infection, low treatment steady state will involve a relatively high level of prevention.

We find that conducting comparative statics analysis is at best a very delicate matter. In steady states with no prevention, the comparative statics of steady state prevalence with respect to infection and recovery rates mirror those of the classical epidemiological SIS model. Namely, steady state prevalence is increasing in infectivity and decreasing in

[26] See *Swine Flu: From Containment to Treatment*, UK Department of Health (2009).

[27] In a news release by the Scottish Government, Health Secretary Nicola Sturgeon is reported to have stated that "In recent weeks we have, as expected, seen a significant increase in the number of cases of pandemic flu throughout the UK [...]. Given the number of cases, and the evidence of community transmission, we believe now is the right time to move to the treatment phase of dealing with the pandemic [...]. This does not mean that the virus is getting more severe or that there is any cause for alarm. It simply means that we are seeing a rise in the number of cases and are adapting our approach to dealing with these." See http://www.scotland.gov.uk/News/Releases/2009/07/02125359.

the effective rate of recovery. In steady states with positive preventive effort, these results are partially reversed. In particular, higher infectivity yields lower steady state disease prevalence (because of lower prevention levels) while prevalence is wholly independent of the rate of recovery. These results highlight the importance of careful formal analysis in conducting policy aimed at reducing infectivity or at increasing the effectiveness of treatment.

An interesting and worthwhile extension of the present work that we have not yet pursued, is to conduct a careful bifurcation analysis. While such analysis has been carried out for the related shallow lake systems, as described in connection with the equilibrium dynamics of the model, a number of different regimes are possible for different parameter constellations. It would be interesting to carry out a systematic analysis of these regimes, in order to get a clearer picture of when the system is in Regime I, II or III respectively (in which there is either a unique steady state, multiple steady states with a unique optimal one, or multiple steady states in which the optimal one depends on initial conditions). Such an analysis would likely entail a significant amount of simulations, but should be worth pursuing in order to conduct policy experiments.

We should emphasize that when treatment or prevention efforts are found to be zero in steady state, the model does not reduce to the special case models in which no treatment or prevention is possible. This is because it may be optimal to treat and/or prevent infection along the equilibrium paths even if it ceases to be optimal once steady state is reached. Thus the dynamics of the present model in those cases differ from the corresponding dynamics of the single-instrument models. Furthermore, for some parameter constellations, steady states from the treatment only model and the prevention only model coexist.

The modeling assumptions that we have adopted, in particular linearity of costs in the measure of targeted individuals, pose some difficulties but afford us some advantages as well. The simplicity of the optimal solutions makes a characterization of the optimal paths and steady states very clear. In particular, we get closed form solutions for multiple steady states, which allows us to give a sharp characterization and comparative analysis with respect to parameters. On the other hand, the concavity of the disease propagation function, and the resulting convexity of the Hamiltonian, together with the bang-bang nature of the optimal controls, makes it impossible to use many of the standard results in optimal control theory. Specifically, sufficiency conditions for local extrema, such as that of Arrow, as well as conditions for local stability, are inapplicable.[28] Despite this, we make very substantial progress in completely characterizing the behavior and optimal control of the system analytically. We achieve this by using both novel approaches and by using techniques recently developed to study the optimal management of other ecological systems.

[28] There are very few general results on linear control problems. See Caputo (2005) for a discussion.

A. Existence of an Optimal Solution

In this appendix, we prove that the planner's problem admits an optimal solution. The existence proof proceeds in two steps. In Step 1, we consider finite horizon versions of the model and show that in these, an optimal solution exists. In Step 2, we show by contradiction that because optimal solutions exists for all finite horizons, an optimal solution must also exist for the infinite horizon version.

Step 1: Consider a finite horizon version of the model in which $t \in [0, T]$, with $T < \infty$. Define the set

$$N(I,U,t) \equiv \left\{ e^{-\rho t}\left(-\omega I - c_P(1-I)\pi - c_T I\tau\right) + \xi, I\left(\beta(1-I)(1-\pi) - \gamma - \alpha\tau\right) : (\tau,\pi) \in U \right\} \tag{60}$$

where $\xi \leq 0$ is some constant and $U = [0,1] \times [0,1]$ is the space of feasible control pairs.

Consider two points $y_1, y_2 \in N(I,U,t)$ given by

$$y_1 \equiv \left\{ e^{-\rho t}\left(-\omega I - c_P(1-I)\pi_1 - c_T I\tau_1\right) + \xi_1, I\left(\beta(1-I)(1-\pi_1) - \gamma - \alpha\tau_1\right) \right\} \tag{61}$$

$$y_2 \equiv \left\{ e^{-\rho t}\left(-\omega I - c_P(1-I)\pi_2 - c_T I\tau_2\right) + \xi_2, I\left(\beta(1-I)(1-\pi_2) - \gamma - \alpha\tau_2\right) \right\} \tag{62}$$

Let $\varphi \in [0,1]$ and let $y_3 \equiv \varphi y_1 + (1-\varphi)y_2$. We will prove that $y_3 \in N(I,U,t)$ and thus that the set $N(I,U,t)$ is convex. Let $\varphi y_1 + (1-\varphi)y_2 = (z_1, z_2)$. Taking the first element, we have that

$$\begin{aligned} z_1 &= \varphi\left[e^{-\rho t}\left(-\omega I - c_P(1-I)\pi_1 - c_T I\tau_1\right) + \xi_1\right] \\ &\quad + (1-\varphi)\left[e^{-\rho t}\left(-\omega I - c_P(1-I)\pi_2 - c_T I\tau_2\right) + \xi_2\right] \qquad (63) \\ &= e^{-\rho t}\left(-\omega I - c_P(1-I)\pi_3 - c_T I\tau_3\right) + \xi_3 \qquad (64) \end{aligned}$$

where $\tau_3 \equiv \varphi\tau_1 + (1-\varphi)\tau_2$, $\pi_3 \equiv \varphi\pi\tau_1 + (1-\varphi)\pi_2$ and $\xi_3 \equiv \varphi\xi_1 + (1-\varphi)\xi_2 \leq 0$.

Similarly, taking the second element we have that

$$\begin{aligned} z_2 &= \varphi\left[I\left(\beta(1-I)(1-\pi_1) - \gamma - \alpha\tau_1\right)\right] \\ &\quad + (1-\varphi)\left[I\left(\beta(1-I)(1-\pi_2) - \gamma - \alpha\tau_2\right)\right] \qquad (65) \\ &= I\left[\beta(1-I)(1-\pi_3) - \gamma - \alpha\tau_3\right] \qquad (66) \end{aligned}$$

We can now conclude that: (i) there exist an admissible triple $(I(t), \tau(t), \pi(t))$; (ii) the set $N(I,U,t)$ is convex for each $(I(t),t)$; (iii) the set U is closed and bounded; (iv) there exists a bound $b = 1$ such that $\|I(t)\| < b$ for all $t \geq 0$ and admissible triples $(I(t), \tau(t), \pi(t))$. By the Filippov-Cesari Theorem, we can then conclude that an optimal solution $(I^*(t), \tau^*(t), \pi^*(t))$ exists and the optimal policy $(\tau^*(t), \pi^*(t))$ is measurable. See Seierstad and Sydsaeter (1987) for details.

Step 2: We will consider the case in which the relevant steady states are (A, B). The case (A_0, B_0) follows similar steps. In the finite horizon version of our problem, we impose no condition on the terminal value $I(T)$. This implies that the relevant transversality condition is $\lambda(T) = 0$. As we have shown, this problem has an optimal solution. Moreover, if T is large enough, there are at at most two candidates for an optimum. Each path satisfies the necessary conditions for optimality, including the aforementioned transversality condition. Of these two candidate optimal paths, one goes to solution A as in the infinite horizon case but then at time $t = T - T_A$, peels off along the unstable branch to

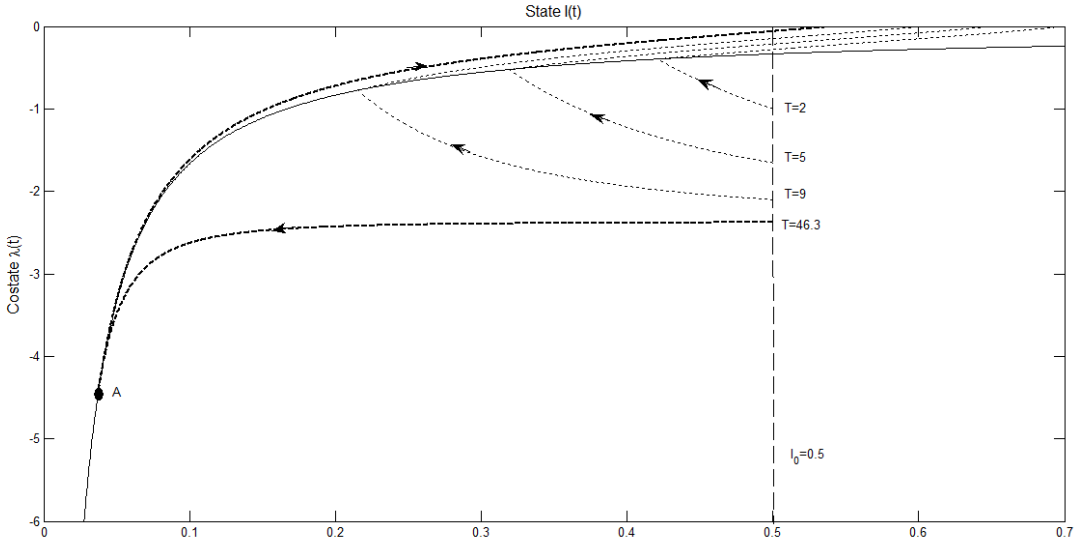

Figure 7: Paths in finite horizon model around point A.

increase monotonically, reaching $\lambda(t) = 0$ at time $t = T$. The other path goes to solution B as in the infinite horizon case, but then at time $t = T - T_B$, peels off along the unstable branch to increase monotonically, reaching $\lambda(t) = 0$ at time $t = T$. Note that the times T_A and T_B are fixed.

In Figure 7, we illustrate the idea by plotting optimal paths around the point A. The parameters are the same as those in Example 1. The optimal paths from initial condition $I_0 = 0.5$ and different horizons T are represented by dashed curves. Note that the light paths reach the $\lambda(T) = 0$ line faster than the heavy dashed path that goes through point A, since the latter stays at point A until time $t = T - T_A$ regardless of how long it took that path to reach point A. In contrast, the light dashed paths do not rest at any point until they reach their destination. For all horizons $T \geq 46.3$, the optimal path reaches point A before making the transition to the $\lambda(T) = 0$ line, while for horizons $T < 46.3$, the point A is not reached along an optimal path. While we have shown only a case where $I_0 > I_A$, similar analysis applies for the case $I_0 < I_A$. Similar analysis also applies for optimal finite horizon paths in the vicinity of the other steady states.

From the point A, there is a unique path satisfying the Hamiltonian conditions and starting from $(I(0), \lambda(0)) = (I_A, \lambda_A)$ and is such that $\lambda(T_A) = 0$ for some $T_A > 0$. The time T_A is unique. Denote the value of the integral along this path as follows:

$$W_A \equiv \int_0^{T_A} e^{-\rho t} \left[I(t) \left[\omega_{\mathcal{I}} - c_T \tau(t) \right] + (1 - I(t)) \left[\omega_{\mathcal{S}} - c_P \pi(t) \right] \right] dt \tag{67}$$

From the point B, there is similarly a unique path satisfying the Hamiltonian conditions and starting from $(I(0), \lambda(0)) = (I_B, \lambda_B)$ and is such that $\lambda(T_B) = 0$ for some $T_B > 0$. The time T_B is unique. Denote the value of the integral along this path as follows:

$$W_B \equiv \int_0^{T_B} e^{-\rho t} \left[I(t) \left[\omega_{\mathcal{I}} - c_T \tau(t) \right] + (1 - I(t)) \left[\omega_{\mathcal{S}} - c_P \pi(t) \right] \right] dt \tag{68}$$

From now on, we shall consider only paths that begin at $I(0) = I_0$. Let

$$V_A^{\overline{T}} \equiv \int_0^{\overline{T}} e^{-\rho t} \left[I(t) \left[\omega_{\mathcal{I}} - c_T \tau(t) \right] + (1 - I(t)) \left[\omega_{\mathcal{S}} - c_P \pi(t) \right] \right] dt \tag{69}$$

where the integral is evaluated along the Hamiltonian path and terminates at point A at time $\overline{T}$. Also, let

$$V_B^{\overline{T}} \equiv \int_0^{\overline{T}} e^{-\rho t} \left[I(t) \left[\omega_{\mathcal{I}} - c_T \tau(t) \right] + (1 - I(t)) \left[\omega_{\mathcal{S}} - c_P \pi(t) \right] \right] dt \tag{70}$$

where the integral is evaluated along the Hamiltonian path and terminates at point B at time $\overline{T}$.

Finally, let

$$X_A^T \equiv \int_0^T e^{-\rho t} \left[I(t) \left[\omega_{\mathcal{I}} - c_T \tau(t) \right] + (1 - I(t)) \left[\omega_{\mathcal{S}} - c_P \pi(t) \right] \right] dt \tag{71}$$

where the integral is evaluated along the Hamiltonian path that goes to point A and sits

there until time $t = T - T_A$ and then peels off to reach $\lambda(t) = 0$ at time $t = T$. Also, let

$$X_B^T \equiv \int_0^T e^{-\rho t} \left[I(t) \left[\omega_{\mathcal{I}} - c_T \tau(t) \right] + (1 - I(t)) \left[\omega_{\mathcal{S}} - c_P \pi(t) \right] \right] dt \tag{72}$$

where the integral is evaluated along the Hamiltonian path that goes to point B and sits

there until time $t = T - T_B$ and then peels off to reach $\lambda(t) = 0$ at time $t = T$.

It is clear that

$$X_A^T = V_A^{T-T_A} + e^{-\rho(T-T_A)} W_A \tag{73}$$
$$X_B^T = V_B^{T-T_B} + e^{-\rho(T-T_B)} W_B \tag{74}$$

Suppose without loss of generality that in the infinite horizon case, it is better to go to point B and stay there than to go to point A and stay there. Then

$$\lim_{T \to \infty} V_B^{T-T_B} = V_B^{\infty} > V_A^{\infty} = \lim_{T \to \infty} V_A^{T-T_A} \tag{75}$$

From the above equations, it then follows that

$$\lim_{T \to \infty} X_B^T > \lim_{T \to \infty} X_A^T \tag{76}$$

Thus, in the finite horizon case, it is optimal for large T to go to point B and then peel off at time $t = T - T_B$.

Suppose there is no optimal path in the infinite horizon case. Then there is some path starting from $I(0) = I_0$ for which the value of the integral is greater than V_B^{∞}. Let

$$Z^T \equiv \int_0^T e^{-\rho t} \left[I(t) \left[\omega_{\mathcal{I}} - c_T \tau(t) \right] + (1 - I(t)) \left[\omega_{\mathcal{S}} - c_P \pi(t) \right] \right] dt \tag{77}$$

where the integral is evaluated along this alternative path.

By assumption,

$$\lim_{T \to \infty} Z^T = Z^{\infty} > V_B^{\infty} = \lim_{\overline{T} \to \infty} V_B^{\overline{T}} \tag{78}$$

This implies that there exist $\overline{T}^*, T^*, \varepsilon > 0$ such that for all $\overline{T} > \overline{T}^*$ and $T > T^*$, the inequality $Z^T > V_B^{\overline{T}} + \varepsilon$ holds. Hence, for $T > \max\left\{ \overline{T}^* + T_B, T^* \right\}$, it follows that

$$Z^T > V_B^{T-T_B} + \varepsilon \tag{79}$$

Now, for sufficiently large T, $\varepsilon > e^{-\rho(T-T_B)} W_B$ and hence

$$Z^T > V_B^{T-T_B} + e^{-\rho(T-T_B)} W_B = X_B^T \tag{80}$$

But this is not possible, since X_B^T is optimal. This contradiction establishes that there must be an optimal solution to the infinite horizon problem. This concludes the proof ■

B. Parameter Restrictions for Steady States

Throughout this paper, we have maintained the assumption that $\omega - c_P > 0$ and $\beta - \gamma - \alpha > 0$. In this appendix, we list additional assumptions that ensure that the different fixed points are feasible.

B.1. Fixed Point A. For this steady state to be feasible, we need the following additional restrictions:

- For $I(t) \in (0,1)$ need $c_P < \frac{\beta\omega}{\rho+\beta}$.
- For $\lambda(t) < 0$ need $c_P < \omega$.
- For $\pi(t) \in (0,1)$ need $c_P < \omega$.
- For $\tau(t) = 0$ need $c_P > \omega - c_T\left(\frac{\rho}{\alpha}\right)$.

B.2. Fixed Point B. For this steady state to be feasible, we need the following additional restrictions:

- For $I(t) \in (0,1)$ need $c_P < \frac{\beta(\omega+c_T)}{\rho+\beta}$.
- For $\lambda(t) < 0$ need $c_P < \omega + c_T$.
- For $\pi(t) \in (0,1)$ need $c_P < \left(\frac{\beta-\alpha-\gamma}{\beta-\alpha-\gamma+\rho}\right)(\omega + c_T)$.
- For $\tau(t) = 1$ need $c_P < \omega + c_T\left(\frac{\alpha-\rho}{\alpha}\right)$.

B.3. Fixed Point C. For this steady state to be feasible, we need the following additional restrictions:

- For $I(t) \in (0,1)$ need $c_P < \frac{\beta c_T}{\alpha}$.
- For $\lambda(t) < 0$, no extra restriction.
- For $\pi(t) \in (0,1)$ need $c_P < \min\left\{\frac{\omega}{2} + c_T\left(\frac{\beta-\gamma-\rho}{2\alpha}\right), \frac{\beta c_T}{\alpha}\right\}$.
- For $\tau(t) \in (0,1)$ need $c_P \in (\omega - c_T\left(\frac{\rho}{\alpha}\right), \omega + c_T\left(\frac{\alpha-\rho}{\alpha}\right))$.

B.4. Fixed Point A_0. For this steady state to be feasible, we need the following additional restrictions:

- For $I(t) \in (0,1)$ need $\beta > \gamma$.
- For $\lambda(t) < 0$ need $\beta > \gamma - \rho$.
- For $\pi(t) = 0$ need $c_P > \frac{\omega(\beta-\gamma)}{\beta-\gamma+\rho}$.
- For $\tau(t) = 0$ need $c_T > \frac{\alpha\omega}{\beta-\gamma+\rho}$.

B.5. Fixed Point B_0. For this steady state to be feasible, we need the following additional restrictions:

- For $I(t) \in (0,1)$, no extra restriction.
- For $\lambda(t) < 0$, no extra restriction.
- For $\pi(t) = 0$ need $c_P > \frac{(\omega+c_T)(\beta-\gamma-\alpha)}{\beta-\gamma+\rho-\alpha}$.
- For $\tau(t) = 1$ need $c_T < \frac{\alpha\omega}{\beta-\gamma+\rho-2\alpha}$.

B.6. Fixed Point C_0. For this steady state to be feasible, we need the following additional restrictions:

- For $I(t) \in (0,1)$, need $c_T \in (\frac{\alpha\omega}{\beta+\gamma+\rho}, \frac{\alpha\omega}{\gamma+\rho-\beta})$.
- For $\lambda(t) < 0$, no extra restriction.
- For $\pi(t) = 0$ need $c_P > \frac{\alpha\omega+c_T(\beta-\gamma-\rho)}{2\alpha}$.
- For $\tau(t) \in (0,1)$ need $c_T \in (\frac{\alpha\omega}{\beta-\gamma+\rho}, \frac{\alpha\omega}{\beta-\gamma+\rho-2\alpha})$.

C. Non-Optimality of Maximal Prevention

In this appendix, we prove that an optimal path cannot end at a point at which prevention is at its maximum possible level. We prove this result by contradiction. Suppose that $\pi^* = 1$. Consider a trajectory for which there exists $\hat{t}$ such that $(\tau(t), \pi(t)) = (\overline{\tau}, 1)$ for all $t \geq \hat{t}$, where $\overline{\tau} \in [0,1]$ is a fixed level of treatment. Such a policy will eradicate the disease asymptotically, i.e. will be such that $\lim_{t\to\infty} I(t) = 0$. Assume this trajectory is optimal. There are two cases to consider. First, suppose that $\overline{\tau} < 1$. Since the policy is optimal, it must be the case that $\lambda(t) \geq -c_T/\alpha$ for $t \geq \hat{t}$. Hence, $\beta\lambda(t)I(t) \geq -\beta I(t)c_T/\alpha$ for $t \geq \hat{t}$. Since $\lim_{t\to\infty} I(t) = 0$ it follows that $\lim_{t\to\infty} \beta\lambda(t)I(t) \geq 0$. This implies the existence of $\tilde{t}$ such that $\beta\lambda(t)I(t) > -c_P$ for $t \geq \tilde{t}$. By the Hamiltonian conditions this in turn implies that $\pi(t) = 0$ for $t \geq \tilde{t}$, contradicting the assumption that $\pi(t) = 1$ for sufficiently large t.

Next, suppose that $\overline{\tau} = 1$. For clarity assume that $\hat{t} = 0$. From the logistic growth equation, it follows that along such a path, prevalence evolves according to

$$\dot{I}(t) = -(\gamma + \alpha)I(t) \tag{81}$$

Integrating this equation yields

$$I(t) = e^{-(\gamma+\alpha)t} I_0 \tag{82}$$

where $I_0 = I(0)$ is the initial condition. Hence

$$\dot{I}(t) = -(\gamma + \alpha)e^{-(\gamma+\alpha)t} I_0 \tag{83}$$

From the law of motion of the costate variable, we have that

$$\dot{\lambda}(t) = \lambda(t)\left[\rho + \gamma + \alpha\right] + \left[\omega - c_P + c_T\right] \tag{84}$$

This differential equation can be rewritten as

$$\dot{z}(t) = z(t)\left[\rho + \gamma + \alpha\right] \tag{85}$$

where

$$z(t) = \lambda(t) + \frac{\omega - c_P + c_T}{\rho + \gamma + \alpha} \tag{86}$$

Integrating yields

$$z(t) = e^{(\rho+\gamma+\alpha)t} z_0 \tag{87}$$

where

$$z_0 = \lambda_0 + \frac{\omega - c_P + c_T}{\rho + \gamma + \alpha} \tag{88}$$

and $\lambda_0 = \lambda(0)$.

It follows that

$$\lambda(t) = e^{(\rho+\gamma+\alpha)t}\left(\lambda_0 + \frac{\omega - c_P + c_T}{\rho + \gamma + \alpha}\right) - \frac{\omega - c_P + c_T}{\rho + \gamma + \alpha} \tag{89}$$

and thus

$$\begin{aligned} e^{-\rho t}\lambda(t)\dot{I}(t) &= -\left(\lambda_0 + \frac{\omega - c_P + c_T}{\rho + \gamma + \alpha}\right)(\gamma + \alpha)I_0 \\ &\quad + e^{-(\gamma+\alpha)t}\left(\frac{\omega - c_P + c_T}{\rho + \gamma + \alpha}\right)(\gamma + \alpha)\, I_0 \end{aligned} \tag{90}$$

Taking the infinite horizon limit, gives

$$\lim_{t\to\infty} e^{-\rho t}\lambda(t)\dot{I}(t) = -\left(\lambda_0 + \frac{\omega - c_P + c_T}{\rho + \gamma + \alpha}\right)(\gamma + \alpha)I_0 \tag{91}$$

Next, note that

$$\lambda_0 < \lambda_{C_T} = -\left(\frac{\omega - c_P + c_T}{\rho}\right) < -\left(\frac{\omega - c_P + c_T}{\rho + \gamma + \alpha}\right) \tag{92}$$

Hence

$$\lambda_0 + \frac{\omega - c_P + c_T}{\rho + \gamma + \alpha} < 0 \tag{93}$$

and it follows that

$$\lim_{t\to\infty} e^{-\rho t}\lambda(t)\dot{I}(t) > 0 \tag{94}$$

Note also that

$$e^{-\rho t}H = e^{-\rho t}\left[-\omega - c_P\pi(t)(1 - I(t)) - c_T I(t)\right] + e^{-\rho t}\lambda\dot{I}(t) \tag{95}$$

Taking limits yields

$$\lim_{t\to\infty} e^{-\rho t}H = \lim_{t\to\infty} e^{-\rho t}\lambda(t)\dot{I}(t) > 0 \tag{96}$$

According to Michel (1982), a necessary condition for a trajectory to be optimal is that the transversality condition $\lim_{t\to\infty} e^{-\rho t}H = 0$ holds. This condition is clearly not satisfied along any trajectory with $(\tau(t), \pi(t)) = (1, 1)$ for all $t \geq 0$. This concludes the proof ■

D. Non-Optimality of Points C and C_0

In this appendix, we formally establish the non-optimality of the interior points C and C_0. To this end, we first prove a useful relationship between the value function and the Hamiltonian. This part of the proof is related to a result by Mäler et al. (2003), but theirs applies only to fully interior controls and we must therefore make suitable changes and exploit that controls are constant almost everywhere along optimal paths.[29]

Lemma 19. $\rho V(I_0) = H(I_0, \tau(0), \pi(0), \lambda(0))$.

Proof: Consider a path which starts from the point $I(0) = I_0$, for which the control variables $\tau(t)$ and $\pi(t)$ are piecewise continuous and which satisfies the first order Hamiltonian conditions. For any path that satisfies these conditions together with the transversality condition and the laws of motion for state and costate variables, the following are true:
(1) Suppose that

$$-\left[c_T + \alpha\lambda(t)\right] I(t) < 0 \tag{97}$$

Then $\tau(t) = 0$ is optimal. Since $\lambda(t)$ and $I(t)$ are continuous along the path in question, it follows that

$$-\left[c_T + \alpha\lambda(t+\varepsilon)\right] I(t+\varepsilon) < 0 \tag{98}$$

for sufficiently small $\varepsilon > 0$ and hence $\tau(t+\varepsilon) = 0$. Thus, $d\tau(t)/dt = 0$ at time t. Likewise, $d\tau(t)/dt = 0$ if

$$-\left[c_T + \alpha\lambda(t)\right] I(t) > 0 \tag{99}$$

which makes $\tau(t) = 1$ optimal. Finally, if

$$\left[c_T + \alpha\lambda(t)\right] I(t) = 0 \tag{100}$$

then the Hamiltonian is independent of the treatment rate and therefore $\partial H/\partial\tau(t) = 0$. Thus, it is always the case that

$$\frac{\partial H}{\partial \tau(t)} \frac{d\tau(t)}{dt} = 0 \tag{101}$$

(2) Suppose

$$-\left[c_P + \beta\lambda(t)I(t)\right](1 - I(t)) < 0 \tag{102}$$

Then $\pi(t) = 0$ is optimal. Since $\lambda(t)$ and $I(t)$ are continuous along the path in question, it follows that

$$-\left[c_P + \beta\lambda(t+\varepsilon)I(t+\varepsilon)\right](1 - I(t+\varepsilon)) < 0 \tag{103}$$

for sufficiently small ε and hence $\pi(t+\varepsilon) = 0$. Thus, $d\tau(t)/dt = 0$ at time t. Likewise, $d\tau(t)/dt = 0$ if

$$-\left[c_P + \beta\lambda(t+\varepsilon)I(t+\varepsilon)\right](1 - I(t+\varepsilon)) > 0 \tag{104}$$

[29] We have an alternative proof of the non-optimality of the interior points but the present derivation is more elegant.

which makes $\pi(t) = 1$ optimal. Finally, if

$$[c_P + \beta\lambda(t)I(t)]\,(1 - I(t)) = 0 \tag{105}$$

then the Hamiltonian is independent of the prevention rate and therefore $\partial H/\partial\pi(t) = 0$. Thus, it is always the case that

$$\frac{\partial H}{\partial \pi(t)}\frac{d\pi(t)}{dt} = 0 \tag{106}$$

The current-value Hamiltonian H is a function of $I(t), \lambda(t), \tau(t)$ and $\pi(t)$. Hence totally differentiating the Hamiltonian yields

$$\begin{aligned}
\frac{dH}{dt} &= \frac{\partial H}{\partial I(t)}\frac{dI(t)}{dt} + \frac{\partial H}{\partial \lambda(t)}\frac{d\lambda(t)}{dt} + \frac{\partial H}{\partial \tau(t)}\frac{d\tau(t)}{dt} + \frac{\partial H}{\partial \pi(t)}\frac{d\pi(t)}{dt} && (107)\\
&= \frac{\partial H}{\partial I(t)}\frac{dI(t)}{dt} + \frac{dI(t)}{dt}\left(\rho\lambda(t) - \frac{\partial H}{\partial I(t)}\right) + \frac{\partial H}{\partial \tau(t)}\frac{d\tau(t)}{dt} + \frac{\partial H}{\partial \pi(t)}\frac{d\pi(t)}{dt} && (108)\\
&= \rho\lambda(t)\frac{dI(t)}{dt} && (109)
\end{aligned}$$

where we have used that

$$\begin{aligned}
\dot{I}(t) &= \frac{\partial H}{\partial \lambda(t)} && (110)\\
\dot{\lambda}(t) &= \rho\lambda(t) - \frac{\partial H}{\partial I(t)} && (111)
\end{aligned}$$

Next, we have that

$$\begin{aligned}
\frac{d(e^{-\rho t}H)}{dt} &= \rho e^{-\rho t}\left[-H + \lambda(t)\frac{dI(t)}{dt}\right] && (112)\\
&= \rho e^{-\rho t}\left[\omega I(t) + c_P\pi(t)(1 - I(t)) + c_T\tau(t)I(t)\right] && (113)
\end{aligned}$$

Since the transversality condition $\lim_{t\to\infty} e^{-\rho t}H(t) = 0$ must hold, it follows that

$$\int_0^\infty \left[\frac{d(e^{-\rho t}H)}{dt}\right] dt = \lim_{t\to\infty} e^{-\rho t}H(t) - H(0) = -H(0) \tag{114}$$

Thus

$$\begin{aligned}
H(x_0, u(0), \lambda(0)) &= -\int_0^\infty \left[\frac{d(e^{-\rho t}H)}{dt}\right] dt && (115)\\
&= -\rho\int_0^\infty e^{-\rho t}\left(\omega I(t) + c_P\pi(t)(1 - I(t)) + c_T\tau(t)I(t)\right) dt && (116)\\
&= \rho V(I_0) && (117)
\end{aligned}$$

Hence

$$\rho V(I_0) = H(I_0, \tau(0), \pi(0), \lambda(0)) \tag{118}$$

This completes the proof ■

We now turn to the proof of the non-optimality of the interior solutions. Suppose there is a path that starts at $I(0) = I_C$ and has

$$\lambda(0) = \lambda_C^* > \lambda_C = \frac{-c_T}{\alpha} \tag{119}$$

and hence $\beta\lambda_C^* I_C > \beta\lambda_C I_C = -c_P$. The first inequality implies that $\tau(t) = 0$ is optimal and the second implies that $\pi(t) = 0$ is optimal. Equation (118) implies that value of the integral for the stationary path that remains at I_C is given by

$$\begin{aligned} \rho V_C &= -\omega - c_P\pi_C(1 - I_C) - c_T\tau_C I_C + \lambda_C I_C\left[(1 - \pi_C)\beta(1 - I_C) - \gamma - \alpha\tau_C\right] && (120)\\ &= -\omega + \lambda_C I_C\left[\beta(1 - I_C) - \gamma\right] && (121)\end{aligned}$$

The value of the integral along the alternative path is found by setting $I(0) = I_C$, $\lambda(0) = \lambda_C^*$, $\tau(0) = 0$ and $\pi(0) = 0$. Using (118), this yields the following expression for the integral along this path:

$$\rho V^* = -\omega + \lambda_C^* I_C\left[\beta(1 - I_C) - \gamma\right] \tag{122}$$

By subtraction,

$$\rho(V^* - V_C) = (\lambda_C^* - \lambda_C) I_C\left[\beta(1 - I_C) - \gamma\right] \tag{123}$$

Note that $\dot{I}(t) = 0$ if $I(t) = I_C, \tau(t) = \tau_C, \pi(t) = \pi_C$. Hence

$$\dot{I}(t) = I_C\left[(1 - \pi_C)\beta(1 - I_C) - \gamma - \tau_C\alpha\right] = 0 \tag{124}$$

Since $I_C, \tau_C, \pi_C > 0$, if follows that

$$I_C\left[\beta(1 - I_C) - \gamma\right] > I_C\left[(1 - \pi_C)\beta(1 - I_C) - \gamma - \tau_C\alpha\right] = 0 \tag{125}$$

Since $\lambda_C^* > \lambda_C$, it follows that $V^* > V_C$. Thus it is better to choose the alternative path than to remain at C. These arguments also apply to the point C_0. This concludes the proof ■

E. Rotation Around Interior Solutions

In this appendix, we prove that the movement around the interior points is a clock-wise rotation. Suppose that the interior stationary solution is C. The diagram in Figure 8 shows a linearized segment of a path in the vicinity of C and the angles θ_i, $i = 1, ..., 5$. We shall now show that

$$\begin{aligned} 90^0 &> \theta_1, \theta_3, \theta_4, \theta_5 > 0 && (126)\\ 180^0 &> \theta_2 > 0 && (127)\\ 90^0 &> \theta_1 - \theta_5, \theta_5 - \theta_3, \theta_4 - \theta_5 > 0 && (128)\\ 180^0 &> \theta_2 - \theta_5 > 0 && (129)\end{aligned}$$

Let $t_i = \tan\theta_i$. Then it follows that

$$t_i = \frac{\dot{\lambda}(t)}{\dot{I}(t)} \tag{130}$$

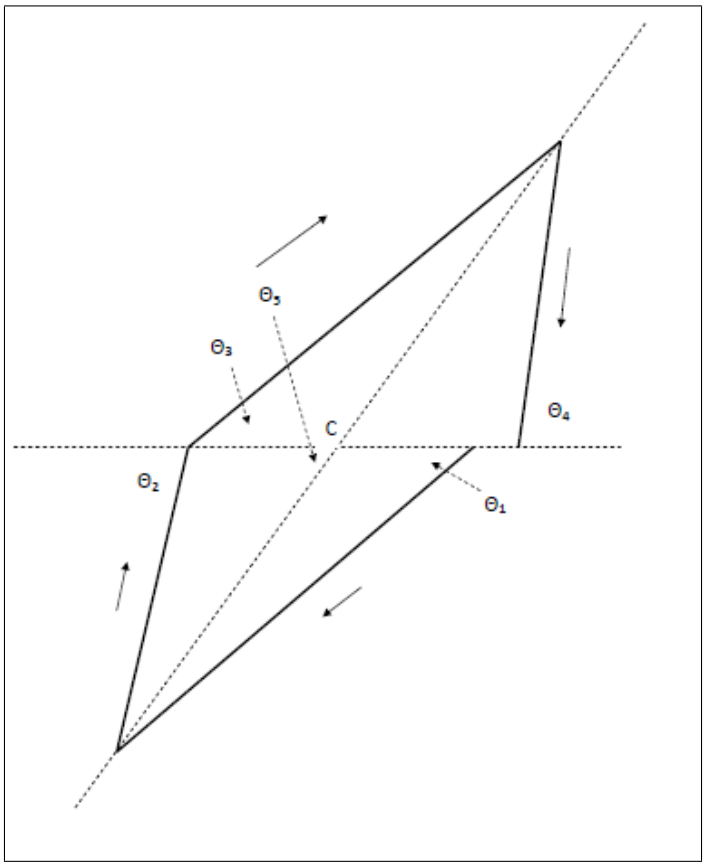

Figure 8: Rotation around interior solution C with linearized system.

For $i = 1, ..., 4$, the rates of change $\dot{I}(t)$ and $\dot{\lambda}(t)$ are calculated by choosing the appropriate values of $\tau(t)$ and $\pi(t)$ and inserting the equilibrium values I_C and λ_C into the laws of motion for the state and costate variables, i.e.

$$\dot{I}(t) = I(t)\left[(1-\pi(t))\beta(1-I(t)) - \gamma - \alpha\tau(t)\right] \tag{131}$$

$$\dot{\lambda}(t) = \lambda(t)\left[\rho + \gamma + \alpha\tau(t) + (1-\pi(t))\beta(2I(t)-1)\right] + \left[\omega - \pi(t)c_P + \tau(t)c_T\right] \tag{132}$$

We now proceed to consider each angle in turn:

Angle θ_1: $\tau(t) = 1, \pi(t) = 1$. This yields the laws of motions

$$\begin{aligned}
\dot{I}(t) &= I_C\left[-\gamma - \alpha\right] && (133)\\
&= -\frac{\alpha c_P}{\beta c_T}(\gamma + \alpha) < 0 && (134)\\
\dot{\lambda}(t) &= \lambda_C\left[\rho + \gamma + \alpha\right] + \left[\omega - c_P + c_T\right] && (135)\\
&= -\frac{c_T}{\alpha}\left(\rho + \gamma\right) + \left(\omega - c_P\right) < 0 \text{ if } C \text{ is allowable} && (136)
\end{aligned}$$

and hence

$$\begin{aligned}
t_1 &= \frac{\dot{\lambda}(t)}{\dot{I}(t)} && (137)\\
&= \frac{\frac{c_T}{\alpha}\left(\rho + \gamma\right) - \left(\omega - c_P\right)}{\frac{\alpha c_P}{\beta c_T}(\gamma + \alpha)} > 0 && (138)
\end{aligned}$$

Thus, $90^0 > \theta_1 > 0$.

Angle θ_2: $\tau(t) = 1, \pi(t) = 0$. This yields the laws of motion

$$\begin{aligned}
\dot{I}(t) &= I_C\left[\beta(1-I_C) - \gamma - \alpha\right] && (139)\\
&= \frac{\alpha c_P}{\beta c_T}\left[\beta\left(1 - \frac{\alpha c_P}{\beta c_T}\right) - \gamma - \alpha\right] && (140)
\end{aligned}$$

$$\begin{aligned}
\dot{\lambda}(t) &= \lambda_C\left[\rho + \gamma + \alpha + \beta(2I_C - 1)\right] + \left[\omega + c_T\right] && (141)\\
&= -\frac{c_T}{\alpha}\left[\rho + \gamma - \beta\right] + \left[\omega - 2c_P\right] > 0 \text{ if } C \text{ is allowable} && (142)
\end{aligned}$$

and hence

$$\begin{aligned}
t_2 &= \frac{\dot{\lambda}(t)}{\dot{I}(t)} && (143)\\
&= \frac{-\frac{c_T}{\alpha}\left[\rho + \gamma - \beta\right] + \left[\omega - 2c_P\right]}{\frac{\alpha c_P}{\beta c_T}\left[\beta\left(1 - \frac{\alpha c_P}{\beta c_T}\right) - \gamma - \alpha\right]} && (144)
\end{aligned}$$

Since $\dot{\lambda}(t) > 0$, it follows that $180^0 > \theta_2 > 0$.

Angle θ_3: $\tau(t) = 0, \pi(t) = 0$. This yields the law of motion for prevalence as

$$\begin{aligned} \dot{I}(t) &= I_C\left[\beta(1 - I_C) - \gamma\right] && (145) \\ &= \frac{\alpha c_P}{\beta c_T}\left[\beta\left(1 - \frac{\alpha c_P}{\beta c_T}\right) - \gamma\right] > 0 \text{ since } I_C < I_{A_0} = \frac{\beta - \gamma}{\beta} && (146) \end{aligned}$$

Note that $I(t)$ converges to $\frac{\beta-\gamma}{\beta}$ if there is no treatment or protection. Since there is some treatment and some protection at C, it must be the case that $I_C < \frac{\beta-\gamma}{\beta}$. The law of motion for the multiplier is given by

$$\dot{\lambda}(t) = -\frac{c_T}{\alpha}\left[\rho + \gamma - \beta\right] + \left[\omega - 2c_P\right] > 0 \text{ if } C \text{ is allowable} \quad (147)$$

and thus it follows that

$$\begin{aligned} t_3 &= \frac{\dot{\lambda}(t)}{\dot{I}(t)} && (148) \\ &= \frac{-\frac{c_T}{\alpha}\left[\rho + \gamma - \beta\right] + \left[\omega - 2c_P\right]}{\frac{\alpha c_P}{\beta c_T}\left[\beta\left(1 - \frac{\alpha c_P}{\beta c_T}\right) - \gamma\right]} > 0 && (149) \end{aligned}$$

Thus, $90^0 > \theta_3 > 0$.

Angle θ_4: $\tau(t) = 0, \pi(t) = 1$. This yields the laws of motion

$$\begin{aligned} \dot{I}(t) &= -\gamma I_C && (150) \\ &= -\gamma\frac{\alpha c_P}{\beta c_T} < 0 && (151) \end{aligned}$$

$$\begin{aligned} \dot{\lambda}(t) &= \lambda_C\left[\rho + \gamma\right] + \left[\omega - c_P\right] && (152) \\ &= -\frac{c_T}{\alpha}\left[\rho + \gamma\right] + \left[\omega - c_P\right] < 0 \text{ if } C \text{ is allowable} && (153) \end{aligned}$$

$$\begin{aligned} t_4 &= \frac{\dot{\lambda}(t)}{\dot{I}(t)} && (154) \\ &= \frac{\frac{c_T}{\alpha}\left[\rho + \gamma\right] - \left[\omega - c_P\right]}{\gamma\frac{\alpha c_P}{\beta c_T}} > 0 && (155) \end{aligned}$$

Thus, $90^0 > \theta_4 > 0$.

To find t_5, note that the curve with this slope satisfies the equation $\beta\lambda(t)I(t) = -c_P$ and

hence at C,

$$t_5 = \frac{d\lambda(t)}{dI(t)} \tag{156}$$
$$= \frac{c_P}{\beta\left(I_C\right)^2} \tag{157}$$
$$= \frac{c_P}{\beta\left(\frac{\alpha c_P}{\beta c_T}\right)^2} \tag{158}$$
$$= \frac{\beta}{c_P}\left(\frac{c_T}{\alpha}\right)^2 > 0 \tag{159}$$

Thus, $90^0 > \theta_5 > 0$.

Angle $\theta_1 - \theta_5$:

$$J\left(t_1 - t_5\right) = \frac{c_T}{\alpha}\left(\rho+\gamma\right) - \left(\omega - c_P\right) - \frac{\beta}{c_P}\left(\frac{c_T}{\alpha}\right)^2 \frac{c_P\alpha}{\beta c_T}(\gamma+\alpha) \tag{160}$$
$$= \frac{c_T}{\alpha}\left(\rho+\gamma\right) - \left(\omega - c_P\right) - \frac{c_T}{\alpha}(\gamma+\alpha) \tag{161}$$
$$= \frac{c_T}{\alpha}\rho - \left(\omega + c_T - c_P\right) < 0 \text{ if } C \text{ exists} \tag{162}$$

where

$$J \equiv \frac{\alpha c_P}{\beta c_T}(\gamma+\alpha) > 0 \tag{163}$$

Thus $90^0 > \theta_5 - \theta_1 > 0$.

Angle $\theta_2 - \theta_5$:

$$K(t_2 - t_5) = -\frac{c_T}{\alpha}\left[\rho+\gamma-\beta\right] + \left[\omega - 2c_P\right] - \frac{c_T}{\alpha}\left[\beta\left(1 - \frac{c_P\alpha}{\beta c_T}\right) - \gamma - \alpha\right] \tag{164}$$
$$= -\frac{c_T}{\alpha}\left[\rho-\alpha\right] + \left[\omega - c_P\right] > -\frac{c_T}{\alpha}\left[\rho-\alpha\right] + \rho\frac{c_T}{\alpha} - c_T = 0 \text{ if } C \text{ exists} \tag{165}$$

where

$$K \equiv \frac{\alpha c_P}{\beta c_T}\left[\beta\left(1 - \frac{\alpha c_P}{\beta c_T}\right) - \gamma - \alpha\right] \tag{166}$$

Thus, $180^0 > \theta_2 - \theta_5 > 0$.

Angle $\theta_3 - \theta_5$:

$$L(t_3 - t_5) = -\frac{c_T}{\alpha}\left[\rho+\gamma-\beta\right] + \left[\omega - 2c_P\right] - \frac{c_T}{\alpha}\left[\beta\left(1 - \frac{c_P\alpha}{\beta c_T}\right) - \gamma\right] \tag{167}$$
$$= -\frac{c_T}{\alpha}\rho + \left[\omega - c_P\right] < 0 \text{ if } C \text{ exists} \tag{168}$$

where

$$L \equiv \frac{\alpha c_P}{\beta c_T}\left[\beta\left(1 - \frac{\alpha c_P}{\beta c_T}\right) - \gamma\right] > 0 \tag{169}$$

Thus, $90^0 > \theta_5 - \theta_3 > 0$.

Angle $\theta_4 - \theta_5$:

$$t_4 - t_5 = \frac{\frac{c_T}{\alpha}[\rho+\gamma] - [\omega - c_P]}{\gamma\frac{\alpha c_P}{\beta c_T}} - \frac{\beta}{c_P}\left(\frac{c_T}{\alpha}\right)^2 \tag{170}$$

$$\begin{aligned} M(t_4 - t_5) &= \frac{c_T}{\alpha}[\rho+\gamma] - [\omega - c_P] - \frac{c_T}{\alpha}\gamma & (171)\\ &= \frac{c_T}{\alpha}\rho - [\omega - c_P] > 0 \text{ if } C \text{ exists} & (172)\end{aligned}$$

where

$$M \equiv \gamma\frac{\alpha c_P}{\beta c_T} > 0 \tag{173}$$

Thus, $90^0 > \theta_4 - \theta_5 > 0$.

This establishes the inequalities we wished to show. There is therefore a clockwise rotation around C. The diagram refers to the case in which $90^0 > \theta_2$. The diagram is slightly different if $180^0 > \theta_2 > 0$, but there is still a clockwise rotation around C.

Next, suppose the interior stationary solution is C_0. Then in the region of this point, there is no prevention and the local dynamics are the same as in the treatment-only model examined by Rowthorn (2006), who showed that there is a clockwise rotation around the interior stationary solution. This concludes the proof ■

F. Points C and C_0 are Spiral Sources

In this appendix, we prove that the rotations around the interior points C and C_0 are necessarily exploding spirals. First, consider paths $(\tau(t), \pi(t))$ that maximize the planner's problem. Then the resulting system

$$\begin{aligned} \dot{I}(t) &= I(t)\left[(1-\pi(t))\beta(1-I(t)) - \gamma - \tau(t)\alpha\right] & (174)\\ \dot{\lambda}(t) &= \lambda(t)\left[\rho + \gamma + \alpha\tau(t) + \beta(2I(t)(1-\pi(t)) + \pi(t) - 1)\right] \\ &\quad + \left[\omega + \tau(t)c_T - \pi(t)c_P\right] & (175)\end{aligned}$$

evaluated along these paths, cannot display limit cycles. This was shown by Wagener (2003) and his argument is as follows. In $(I(t), \lambda(t))$-space, consider the vector field

$$F = \left(\frac{\partial H}{\partial \lambda(t)}, \rho\lambda(t) - \frac{\partial H}{\partial I(t)}\right) \tag{176}$$

Let Φ_t denote the flow mapping of the system (174)-(175). Then for some initial conditions $(I(0), \lambda(0))$, we have $\Phi_t(I(0), \lambda(0)) = (I(t), \lambda(t))$, where $(I(t), \lambda(t))$ is a solution to the system for the given initial conditions. Next, consider a set of initial conditions $\Lambda(0)$. Then Φ_t maps this set into a new set $\Lambda(t)$ as follows:

$$\Lambda(t) = \{(I(t), \lambda(t)) : (I(t), \lambda(t)) = \Phi_t(I(0), \lambda(0)) \text{ for some } (I(0), \lambda(0)) \in \Lambda(0)\} \tag{177}$$

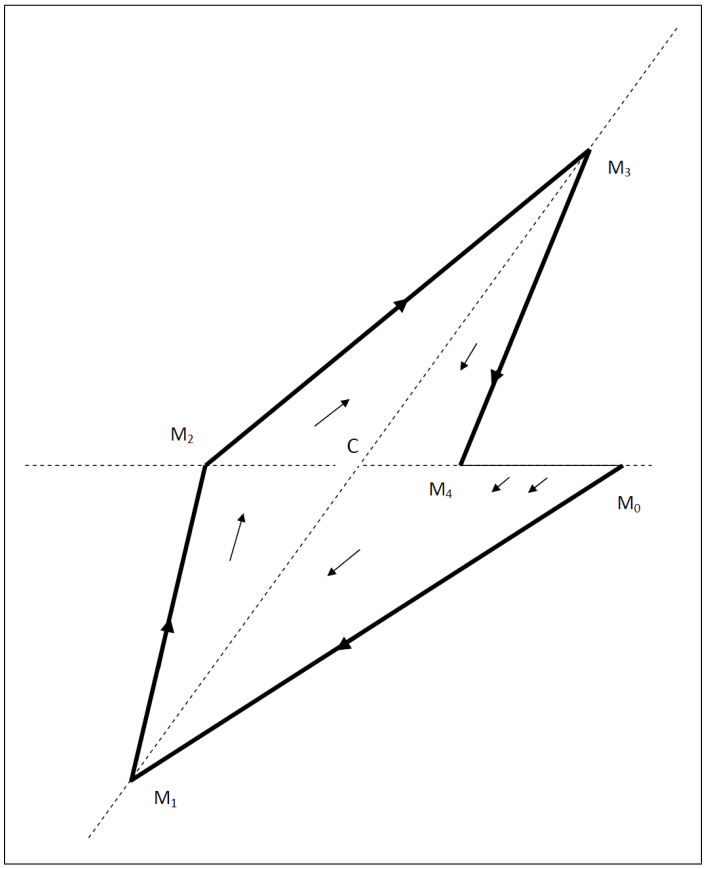

Figure 9: Rotation around point C when it is a spiral sink.

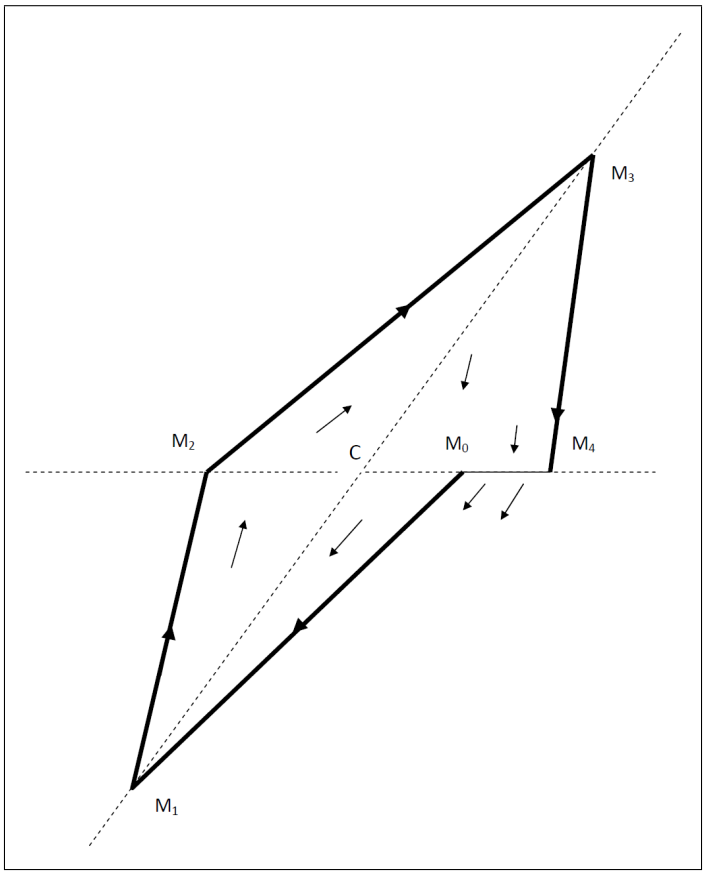

Figure 10: Rotation around point C when it is a spiral source.

The last step is to note[30] that

$$\frac{d\text{Area}\Lambda(t)}{dt}|_{t=0} = \rho\text{Area}\Lambda(0) > 0 \tag{178}$$

In other words, if we start by considering a set of initial conditions $\Lambda(0)$ with strictly positive area, then the invariant region delineated by the system must be strictly increasing over time. But this rules out limit cycles, as they would imply the existence of a bounded invariant region.

Next, we consider the possibility that the interior points are sinks. Figure 9 illustrates a segment of the trajectory around C when the point is a spiral sink. Let $\Lambda(0)$ be the closed set enclosed by the line $M_0M_1M_2M_3M_4$ together with the line segment M_4M_0. As can be seen from the figure, the initial direction of movement of every point in the set $\Lambda(0)$ is into this set, either along the boundary or into the interior. Thus

$$\frac{d\text{Area}\Lambda(t)}{dt}|_{t=0} \leq 0 \tag{179}$$

However, we have already seen that

$$\frac{d\text{Area}\Lambda(t)}{dt}|_{t=0} = \rho\text{Area}\Lambda(0) > 0 \tag{180}$$

This contradiction establishes that the point C cannot be a spiral sink. Thus, the point C must be a spiral source (with clock-wise rotation), as illustrated in figure 10. A similar argument holds for point C_0. This concludes the proof ■

G. Non-Optimality of Spiraling

In this appendix, we prove that it is never optimal to follow a spiral path. Suppose that the interior fixed point C is feasible and consider two paths which satisfy the Hamiltonian conditions and start directly above C at the points (I_C, λ_C^*) and (I_C, λ_C^{**}). Suppose $\lambda_C^{**} > \lambda_C^*$. Initially both paths satisfy the inequalities $\beta\lambda(0)I(0) > -c_P$ and $\lambda(0) > -c_T/\alpha$, and thus in each case $\pi(0) = \tau(0) = 0$.The integral along these paths satisfy the following equations:

$$\rho V^* = H^* = -\omega + \lambda_C^* I_C \left[\beta(1 - I_C) - \gamma\right] \tag{181}$$

$$\rho V^{**} = H^* = -\omega + \lambda_C^{**} I_C \left[\beta(1 - I_C) - \gamma\right] \tag{182}$$

Thus,

$$\rho(V^{**} - V^*) = (\lambda_C^{**} - \lambda_C^*) I_C \left[\beta(1 - I_C) - \gamma\right] > 0 \tag{183}$$

Hence, the path with the higher initial value $\lambda(t)$ is better. In the case of a spiral around the point C in $(I(t), \lambda(t))$-space, this means that it is best to choose the outermost path. This has been shown for paths that begin above the point C. A similar argument applies to paths that start below C. The rule is always choose an outermost path. These arguments also apply to the point C_0 ■

[30] See Wagener (2003) for details.

H. Imperfect Prevention

In this appendix, we consider the effects of imperfect prevention on the steady states and dynamics of the system. Assume that for some $\mu > 0$, the infection rate is given by

$$I(t)\left[\mu + (1 - \pi(t))\beta\right] \tag{184}$$

In this formulation, given the infection level $I(t)$, the infection rate for any level of prevention is uniformly higher than in the standard formulation, since it can be brought down no further than to the level $\mu I(t)$. The Hamiltonian conditions are unchanged by this imperfection, but the dynamics change to

$$\begin{aligned}
\dot{I}(t) &= I(t)\left[(\mu + (1 - \pi(t))\beta)(1 - I(t)) - \gamma - \tau(t)\alpha\right] && (185)\\
\dot{\lambda}(t) &= \lambda(t)\left[\rho + \gamma + \alpha\tau(t) + \beta(2I(t)(1 - \pi(t)) + \pi(t) - 1) + \mu(2I(t) - 1)\right] \\
&\quad + \left[\omega + \tau(t)c_T - \pi(t)c_P\right] && (186)
\end{aligned}$$

The steady state prevalence values for points A^μ, B^μ, C^μ, A_0^μ, B_0^μ, C_0^μ are as follows:

$$\begin{aligned}
I_{A^\mu} &\equiv \frac{\rho c_P}{\beta(\omega - c_P) - \mu c_P} > I_A && (187)\\
I_{B^\mu} &\equiv \frac{\rho c_P}{\beta(\omega - c_P) - \mu c_P + \beta c_T} > I_B && (188)\\
I_{C^\mu} &\equiv \frac{\alpha c_P}{\beta c_T} = I_C && (189)\\
I_{A_0^\mu} &\equiv \frac{\beta - \gamma + \mu}{\beta + \mu} > I_{A_0} && (190)\\
I_{B_0^\mu} &\equiv \frac{\beta - \gamma - \alpha + \mu}{\beta + \mu} > I_{B_0} && (191)\\
I_{C_0^\mu} &\equiv \frac{\alpha\omega + c_T(\beta - \gamma - \rho + \mu)}{2c_T(\beta + \mu)} > I_{C_0} && (192)
\end{aligned}$$

Note that all the relevant steady state prevalence levels are higher than under perfect prevention.[31] It is clear from these results that nothing qualitative changes if prevention becomes imperfect.

I. Quarantine versus Prevention

Consider the setting in which the planner can choose the fraction $q(t) \in [0, 1]$ of infected individuals that are quarantined. Quarantine costs $c_Q \geq 0$ per instant per infected individual. Quarantine reduces the contact rates between infected and susceptible individuals and hence disease incidence becomes

$$(1 - q(t))\beta I(t)(1 - I(t)) \tag{193}$$

This is virtually the same as under prevention as we have modeled it so far. The main difference appears in the cost of the intervention, which depends on which class of individuals is being targeted.

[31] The ranking $I_{C_0^\mu} > I_{C_0}$ holds if and only if $c_T > \frac{\alpha\omega}{\gamma+\rho}$.

The planner's problem is given by

$$\max_{\tau(t),q(t)\in[0,1]} \int_0^\infty e^{-\rho t} \left[I(t)(\omega_I - c_T\tau(t)) + (1 - I(t))\omega_S - I(t)q(t)c_Q \right] dt \tag{194}$$

Disease prevalence evolves according to the differential equation

$$\dot{I}(t) = I(t) \left[(1 - q(t))\beta(1 - I(t)) - \gamma - \tau(t)\alpha \right] \tag{195}$$

The necessary conditions for optimality (for an interior level of prevalence) are then given by

$$c_T + \lambda(t)\alpha = 0 \tag{196}$$

$$c_Q + \beta\lambda(t)(1 - I(t)) = 0 \tag{197}$$

Note that the optimality condition for treatment is unchanged, but that the condition for optimal quarantine differs from that characterizing optimal prevention.

Last, the multiplier evolves according to the differential equation

$$\dot{\lambda}(t) = \lambda(t) \left[\rho + \gamma + \tau(t)\alpha - \beta(1 - q(t))(1 - 2I(t)) \right] + \left[\omega + q(t)c_Q + \tau(t)c_T \right] \tag{198}$$

This version of our model is in fact a generalization of a model analyzed by Sethi (1978). He characterizes the optimal quarantine policy in the SIS model, but without treatment as a control instrument.

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