The Optimal Control of Infectious Diseases: Decentralization, Externalities and Implementation

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Background

- In 14th century Europe, bubonic plague claimed lives of 25 million out of population of 100 million
- In 1918-1920, world death toll from influenza epidemic estimated at 50 million or higher
- More recently, AIDS has claimed more than 25 million lives since 1981 while an estimated 33 million currently live with HIV
- Also, SARS, bird flu, swine flu and designed vira
- Most recently, COVID-19...
- In short, impossible to ignore impact of infectious disease
- Public policy in this area is of first order importance and economists can contribute

Economics and Epidemiology

- Economics and epidemiology have developed separately but in parallel
  - early contributors were Daniel Bernoulli (1700-1782) and Nicolaus I Bernoulli (1687-1759)
- But policy has always been a raison d'être of epidemiology
- Bernoulli (1766) stated that "I simply wish that, in a matter which so closely concerns the well-being of mankind, no decision shall be made without all the knowledge which a little analysis and calculation can provide."
- Daley and Gani (2001): "One of the purposes of modelling epidemics is to provide a rational basis for policies designed to control the spread of a disease."
- Classical mathematical epidemiology devoid of behavioral aspects or optimization

Related Literature

- **Models with protection** (quarantine, prophylaxis, abstinence, mosquito nets, vaccines) include Sethi (1978), Geoffard and Philipson (1996, 1997), Kremer (1996), Auld (2003), Aadland and Finnoff (2007), Francis (2003), Gersovitz and Hammer (2004), Boulier et al. (2007), Brito et al. (1991), Barrett (2003), Chen and Cottrell (2009), Toxvaerd (2010)
- **Models with treatment** include Sanders (1971), Sethi (1974), Goldman and Lightwood (1995, 2002), Rowthorn (2006), Toxvaerd (2010)
- **Models with combinations of instruments** include Gersovitz and Hammer (2004), Gersovitz (2010), Zaman et al. (2007), Goyal and Vigier (2010) and Dodd et al. (2010)

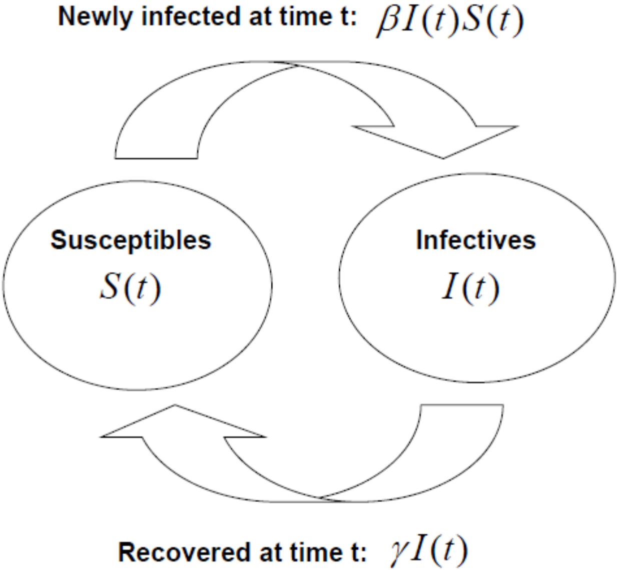
Overview

- Two things important to understand structural properties and control
- Nature of underlying disease dynamics (SI, SIS, SIRS, SIR)
- Available policies (vaccination, social distacing, treatment, test and trace...)
- Recent work mostly SIR w. lockdowns and non-controlled vaccines/treatments
- Consider two models (time allowing):
- SIS with treatment and social distancing
- SEIRS with social distancing

Overview

- The biological and economic models
  - Rates of infection and recovery, instruments and costs
- Centralized decision making
  - Social planner, benchmark
- Decentralized decision making (uncontrolled)
  - Market equilibrium, external effects
- Decentralized decision making (controlled)
  - Implementation, taxes and subsidies

The Classical SIS Model



The Classical SIS Model

- The classical susceptible-infected-susceptible model has two compartments, namely
  - the set of infected individuals $\mathcal{I}(t)$, of measure $I(t)$
  - the set of susceptible individuals $\mathcal{S}(t)$, of measure $S(t)$
- Time is continuous and discounted at rate $\rho > 0$
- There is a continuum $[0, 1]$ of individuals in the population
- At each instant, the population mixes homogeneously
  - pair-wise random matching with equal probabilities

The Classical SIS Model

- The dynamics of the SIS model are given by

$$\begin{aligned} \dot{S}(t) &= I(t)\left[\gamma - \beta S(t)\right] \\ \dot{I}(t) &= I(t)\left[\beta S(t) - \gamma\right] \\ I(t) &= 1 - S(t), \quad I(0) = I_0 \end{aligned}$$

- System reduces to simple logistic growth equation

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

- The steady states of this system are

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

- For $\beta > \gamma$, stable steady state is endemic; for $\beta < \gamma$, (relevant and) stable steady state involves eradication

Infection Dynamics in Classical SIS Model

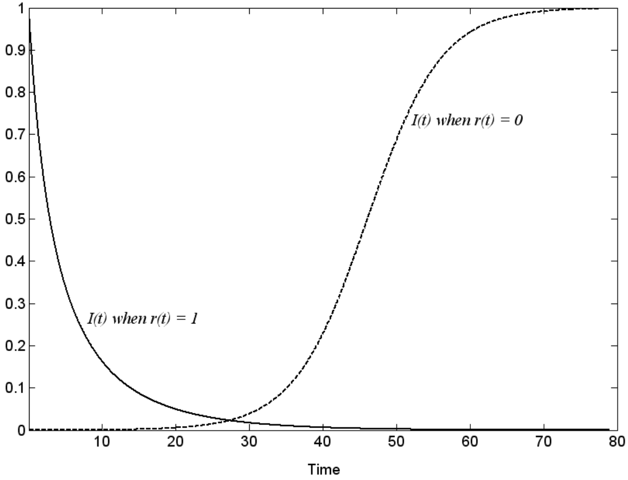


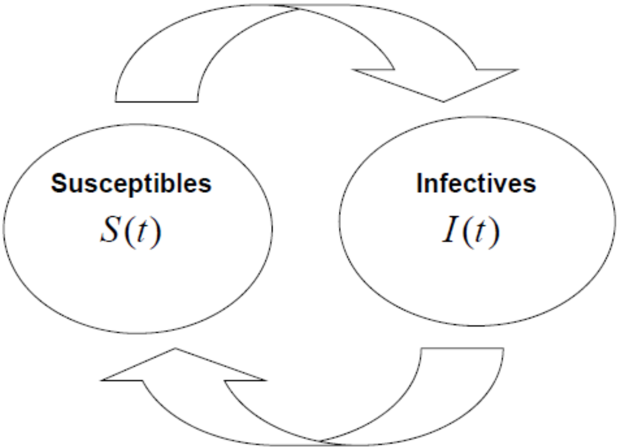
Figure: Evolution of Disease

The Economic Model

- Suppose one wants to conduct welfare analysis and to design sensible policy
- Classical model is unsuitable vehicle because no clear tradeoffs
- Turn model into an economic setting by assigning costs and benefits and defining measure of welfare
- Assume that there is a welfare loss ω associated with being infected ($\omega = \omega_{\mathcal{S}} - \omega_{\mathcal{I}}$)
- Assume that prevention and treatment involves a (linear) cost

The Economic SIS Model

Newly infected at time t: $\beta[1-\pi(t)]I(t)S(t)$



Recovered at time t: $[\alpha\tau(t)+\gamma]I(t)$

The Planner's Problem

- Choose functions $\tau(t), \pi(t) \in [0,1]$ to maximize

$$\int_0^\infty e^{-\rho t}\left[-\omega I(t) - I(t)\tau(t)c_T - (1-I(t))\pi(t)c_P\right]dt$$

- The modified growth equation is given by

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

- Letting multiplier on constraint be $\lambda(t)$, Hamiltonian is

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

- The costate variable evolves according to

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

Optimal Intervention

- The optimal treatment decision given by

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

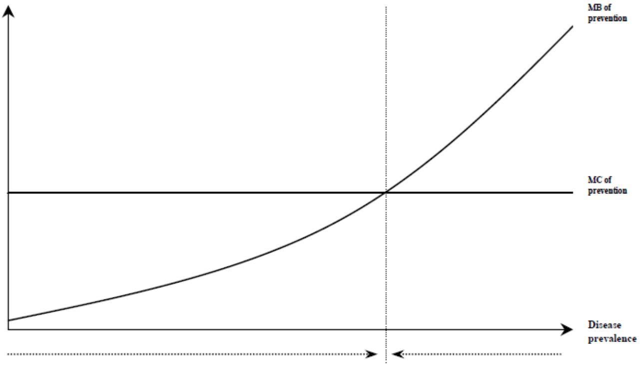
- The optimal prevention decision given by

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

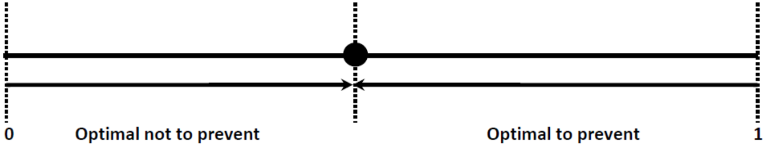
Optimal Intervention

- While the MC is constant, the MB of reducing the infection level turns out to be a function of disease prevalence
- The MB of treatment is *decreasing* in prevalence
- The MB of prevention is *increasing* in prevalence
- This fundamental difference accounts for the differences between the two policies
- Why and how do marginal benefits of treatment and prevention depend on infection levels?
- Marginal benefit of prevention stems from reduction in probability of infection, which is increasing in prevalence
- Marginal benefit of treatment stems from subsequent spell as recovered, whose expected duration is decreasing in prevalence

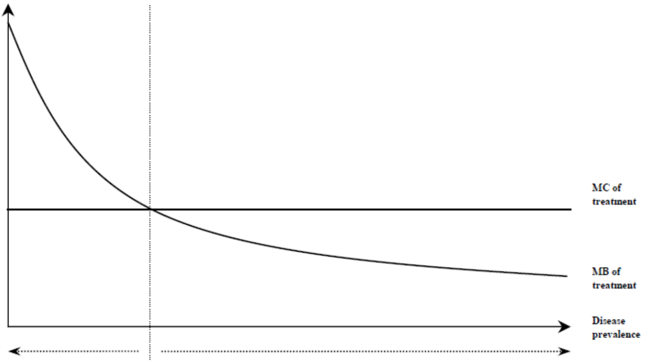
Costs and Benefits with Prevention



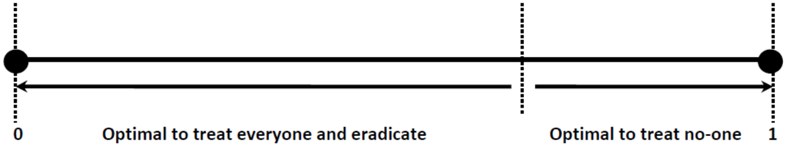
The Model with Prevention Only



Costs and Benefits with Treatment



The Model with Treatment Only



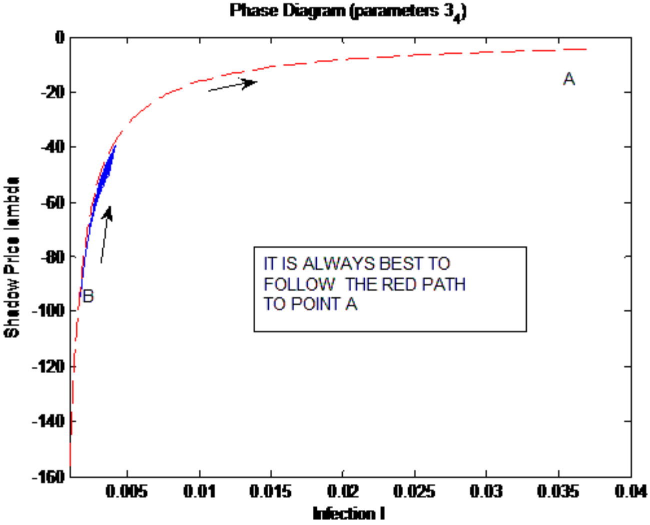
Steady States of the Model

- If prevention is used, there are two types of steady states
  - one steady state has low infection, full treatment and low prevention
  - one steady state has high infection, no treatment and high prevention
- If prevention is not used, there are two types of steady states
  - one steady state has low infection and full treatment
  - one steady state has high infection and no treatment
- Note that in all cases, both treatment and prevention may be used on equilibrium path

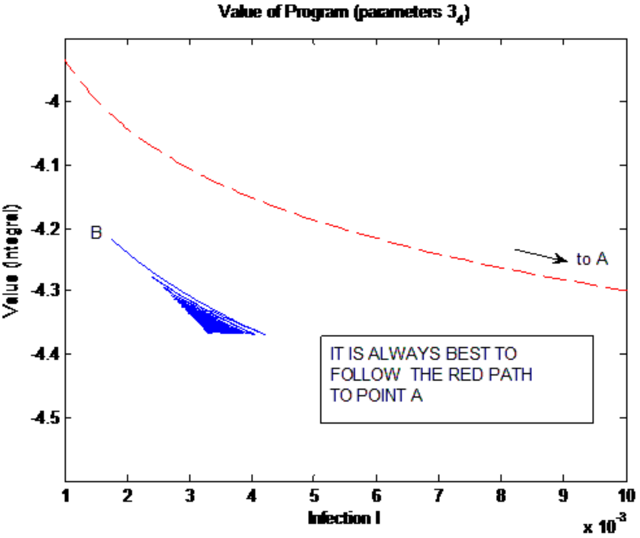
Steady States and Regimes

- There are three possible regimes (depend on parameters)
- Regime I: there is unique steady state
- Regime II: there are two stable steady states but one always dominates the other
- Regime III: there are two stable steady states and optimal one depends on initial conditions

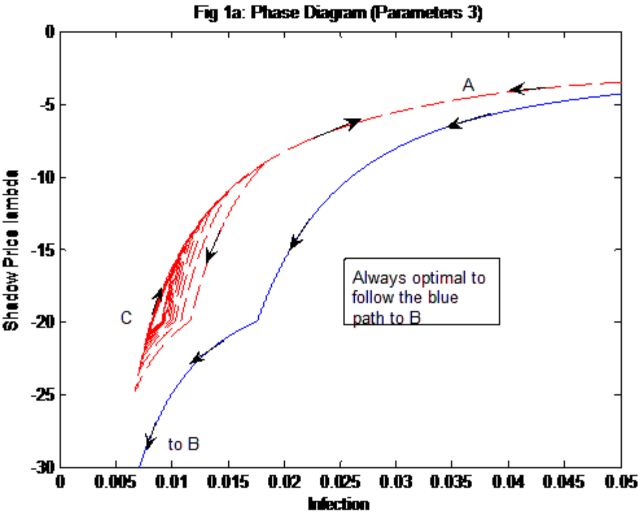
Steady State A Optimal



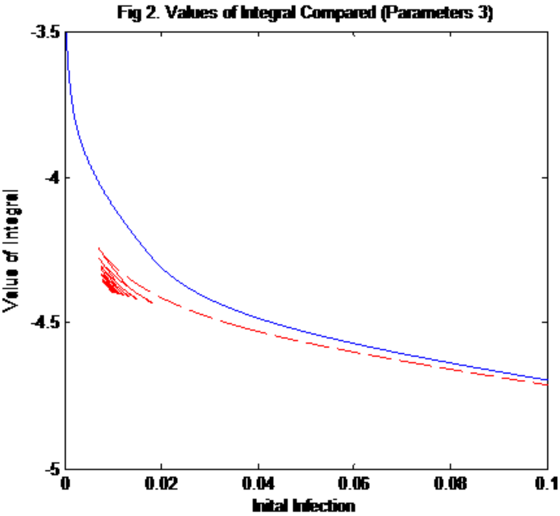
Steady State A Optimal



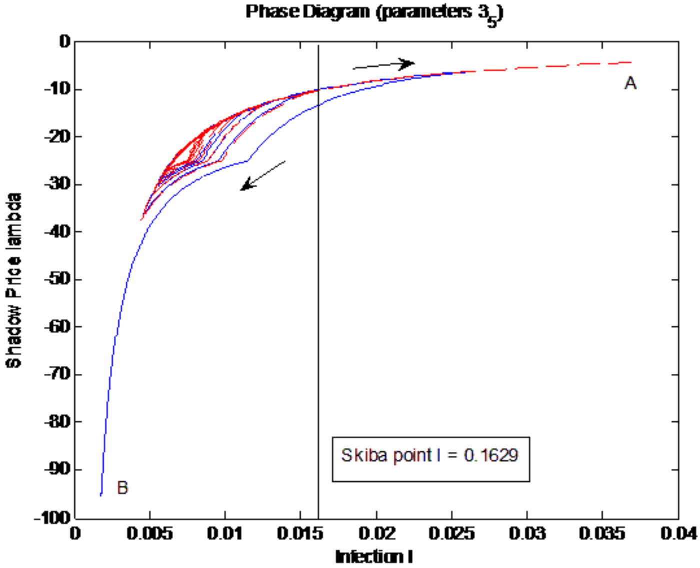
Steady State B Optimal



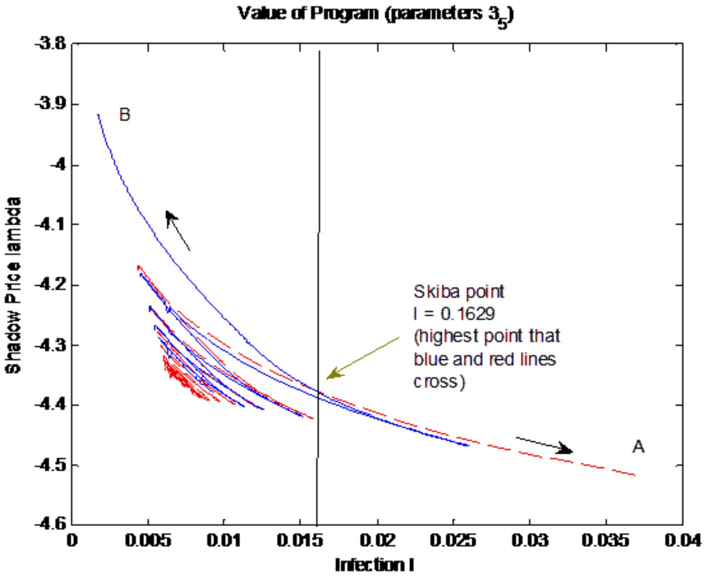
Steady State B Optimal



Steady State A or B Optimal



Steady State A or B Optimal



Equilibrium under Decentralization

- Choose functions $\tau_i(t), \pi_i(t) \in [0, 1]$ to maximize

$$\int_0^\infty e^{-\rho t} \left[-q_i(t)\omega - q_i(t)\tau_i(t)c_T - (1 - q_i(t))\pi_i(t)c_P \right] dt$$

- Constraint for individual is now

$$\dot{q}_i(t) = (1 - q_i(t))(1 - \pi_i(t))\beta I(t) - (\gamma + \tau_i(t)\alpha)q_i(t)$$

- The modified growth equation is given by

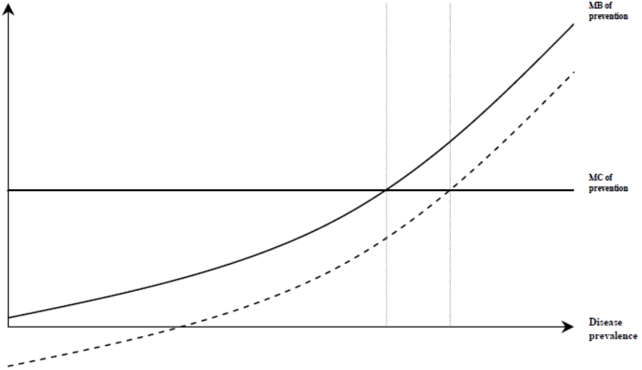
$$\dot{I}(t) = I(t) \left[(1 - \pi)\beta(1 - I(t)) - \gamma - \tau\alpha \right]$$

- Here π and τ are aggregate prevention and treatment levels and $I(t)$ exogenous to agent

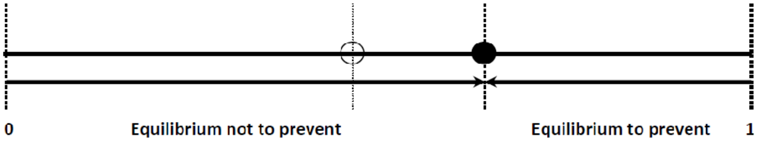
Equilibrium under Decentralization

- Overall typography of steady states similar to planner's problem but dynamics and levels different
- Under decentralized decision making, steady state infection is weakly larger than socially optimal levels
- There are two distinct reasons
  - Agents do not internalize external effects → pure externality effect
  - Agents are infinitesimally small → risk effect

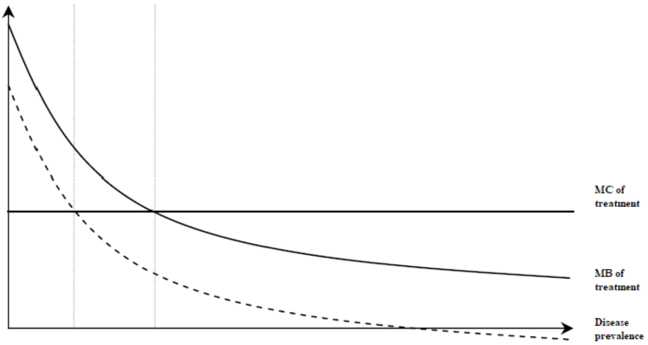
Equilibrium under Decentralization with Prevention



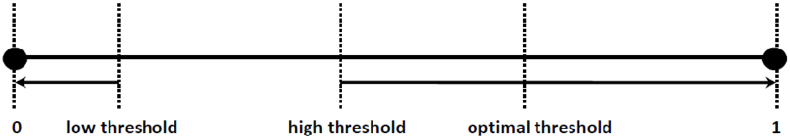
Decentralization and Equilibrium: Prevention



Equilibrium under Decentralization with Treatment



Decentralization and Equilibrium: Treatment



Decentralizing Social Optimum: Decomposition

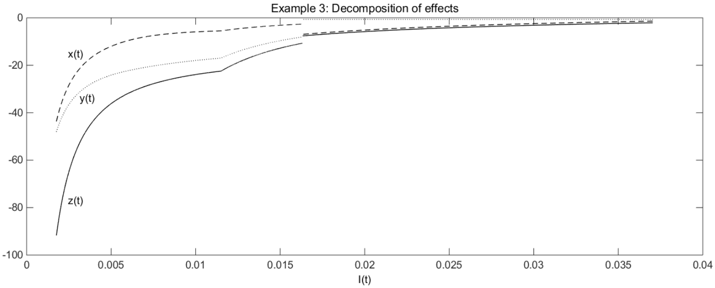
- Let individual's costate be denoted by $\mu(t)$
- We then have that $z(t) \equiv \lambda(t) - \mu(t) \leq 0$
- We can decompose shadow price gap into two effects so

$$\underbrace{\lambda(I) - \mu(I)}_{z(t)} = \underbrace{[\lambda(I) - \eta(I)]}_{x(t)} + \underbrace{[\eta(I) - \mu(I)]}_{y(t)}$$

- There is a pure externality effect $x(t) \equiv \lambda(I) - \eta(I) \leq 0$
  - Because agents don't internalize external effects
- There is a risk effect $y(t) \equiv \eta(I) - \mu(I) \leq 0$
  - Because agents non-atomic and take path as given
- In these calculations, $\eta(I)$ is the costate of a maverick that optimizes against socially optimal path

Decentralizing Social Optimum

- Decomposition into externality and risk effects



Decentralizing Social Optimum: Implementation

- There are two ways of implementing socially optimal path
  - State dependent (dynamic) subsidies $s_P(t)$ and $s_T(t)$ to prevention and treatment $\longrightarrow$ flow subsidies
  - State dependent (dynamic) subsidy $T(t)$ to the healthy (or tax on the infected) $\longrightarrow$ stock subsidies
- These subsidies are prevalence-dependent (i.e. not constant) and very complicated
  - Not easily approximated by simple non-optimal schemes
- Also, may be difficult to implement (depending on context)
  - Subsidizing prevention may be infeasible (e.g. *use* of condoms rather than acquisition)
  - Taxing the infected may be morally/politically untenable

Decentralizing Social Optimum: Implementation

- Let subsidized individual's costate on equilibrium path be denoted by $\phi(t)$
- The optimal flow subsidies are given by

$$
\begin{aligned}
s_P(t) &\equiv \beta I(t)[\phi(t) - \lambda(t)] \geq 0 \\
s_T(t) &\equiv \alpha[\phi(t) - \lambda(t)] \geq 0
\end{aligned}
$$

- These equal rates at which instruments abate social harm from infection
- The relative subsidies given by

$$
\frac{s_P(t)}{s_T(t)} = \left(\frac{\beta}{\alpha}\right) I(t)
$$

- This is a suprisingly simple property

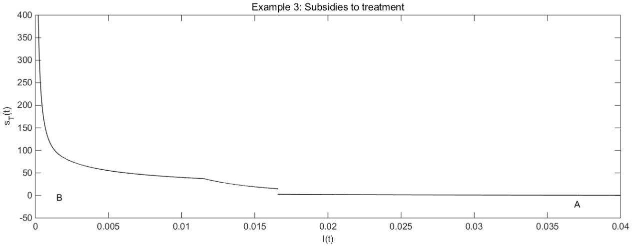
- The optimal stock subsidy is given by

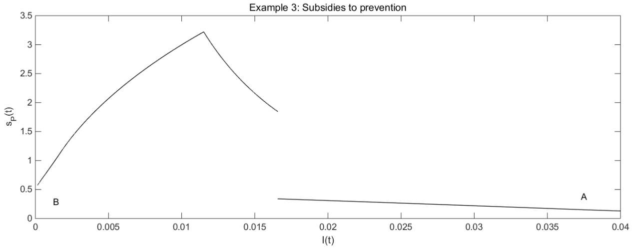
$$T(t) = -\lambda(t)(1 - \pi(t))\beta(1 - I(t)) \geq 0$$

- This subsidy is expected damage per unit of time that individual will cause society
- Note that these schemes not Pigouvian
  - Must correct for both externality and risk effect

Decentralizing Social Optimum: Implementation

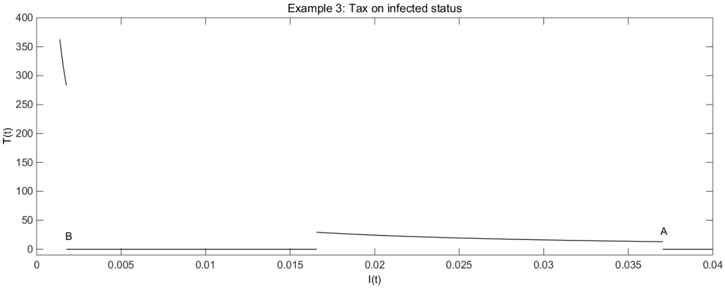
- Subsidies to prevention and treatment





Decentralizing Social Optimum: Implementation

- Subsidies to healthy (or tax on infected)



Conclusion and extensions

- Other policy interventions that change parameters of model
  - Anti-retrovirals and rational disinhibition
- Extension to more general models
  - Susceptible-Infected-Removed-Susceptible model
  - Imperfect protection
  - Eradication
- Online appendix:
  - Existence proofs
  - Characterization of spiral sources
  - Comparative statics and dynamics
  - Speeds of convergence and most rapid approach paths
  - Bifurcation analysis
  - Welfare analysis
  - And more...

Thank you!