Review of Classical Epidemic Models

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Outline

- Introduction
- Types of epidemic models
- Simple deterministic epidemic models
- Qualitative analysis on SIR and SIS models
  - Basic Reproduction Number
- Comments and questions

Introduction

Mathematical modeling of infectious diseases is a tool to investigate the mechanisms for outbreak and spread of diseases and to predict the future course in order to control an epidemic.

- The earliest mathematical epidemic model: (by Daniel Bernoulli) The results showed that the universal inoculation against smallpox could increase the life expectancy.

- Modern mathematical epidemic modeling: A. G. McKendrick and W. O. Kermack (1927) formulated a simple deterministic model, which was successful in predicting the behavior of outbreaks in many recorded epidemics.

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Questions of interests

* Will an infection spread?
* How does it evolve over time?
* When will the spread disappear and what is the final outcome?

Types of mathematical epidemic models

1. Stochastic models:
   The epidemic process has random nature. Stochastic models are used to estimate the probabilistic quantities for the outcome events, such as the probability distribution of extinction time, the probability distribution of final epidemic size, the associate mean and so on.

2. Deterministic compartmental models:
   The transition rate from one class (compartment) to the other one is characterized by derivative mathematically. If we assume that the population size is differentiable with respect to time, in the limiting of large population, the time evolution of behavior of each subgroup can be approximated by the deterministic dynamics.

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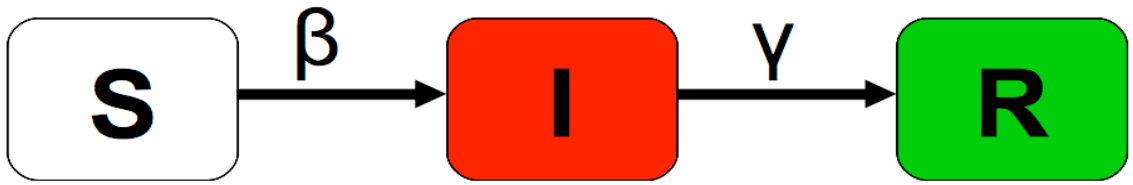
Deterministic compartmental models

1. without demography:
   - SIR
   - SIS
   - SIRS
2. with demography:
   - SIR

Deterministic compartmental models: SIR (Kermack and McKendrick)



where

S – the number of susceptibles

I – the number of infectives

R – the number of recoveries

β – contact rate

γ – recovery rate

SIR, ctd

Let N be the total size of the population and we assume homogeneity, that is, each individual in the population has an equal probability of contacting the disease with a rate of β.

- The number of contacts made by one infective to transmit the disease in the population is βN per unit time.
- The fraction of contacts by one infected individual with a susceptible is $\frac{S}{N}$
- The number of infectives is I

Therefore the consumption rate of susceptibles, which is due to infection, is $\{\beta N\}\left\{\frac{S}{N}\right\}\{I\}(=\beta SI)$. i.e.

$$\frac{dS}{dt} = -\beta SI$$

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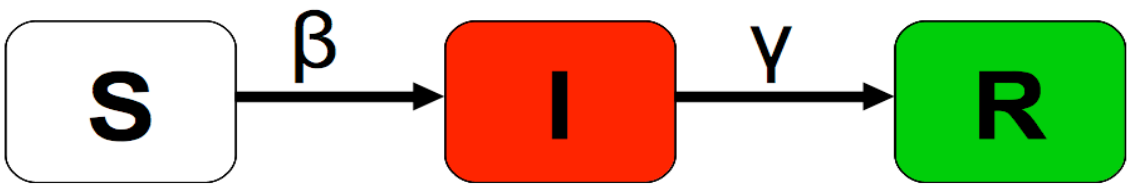
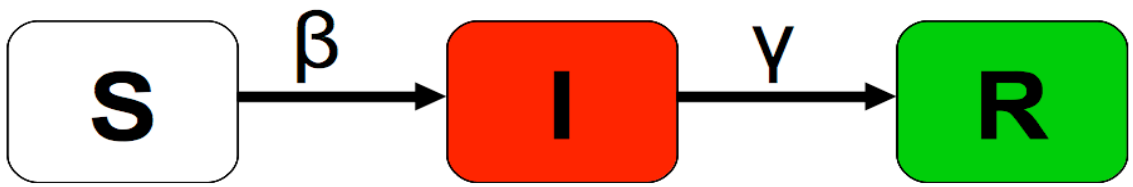


$$\frac{dS}{dt} = -\beta SI$$

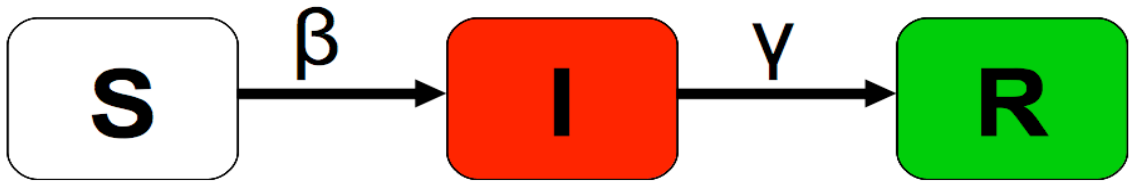
$$\frac{dI}{dt} = ?$$

SIR, ctd



$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \gamma I$$

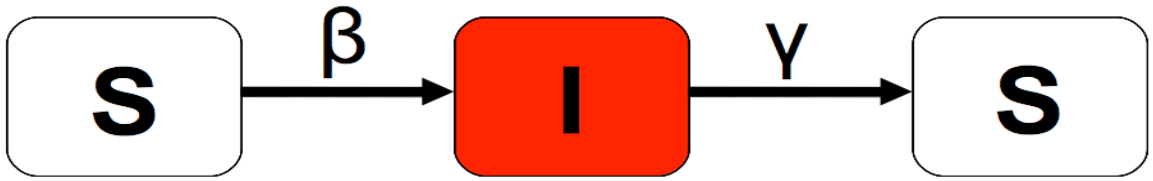
SIR, ctd



$$\begin{aligned} \frac{dS}{dt} &= -\beta SI \\ \frac{dI}{dt} &= \beta SI - \gamma I \\ \frac{dR}{dt} &= \gamma I \end{aligned} \tag{1}$$

Deterministic compartmental models: SIS

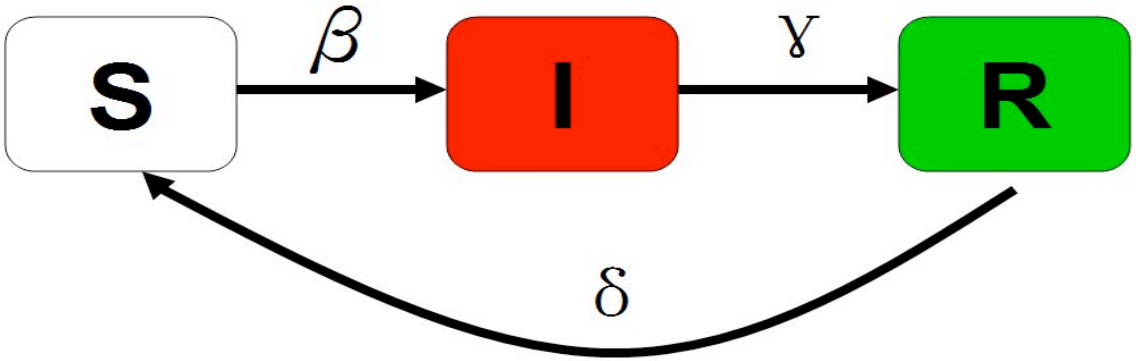
SIS epidemic (disease w/o immunity)



$$\frac{dS}{dt} = -\beta SI + \gamma I$$
$$\frac{dI}{dt} = \beta SI - \gamma I \tag{2}$$

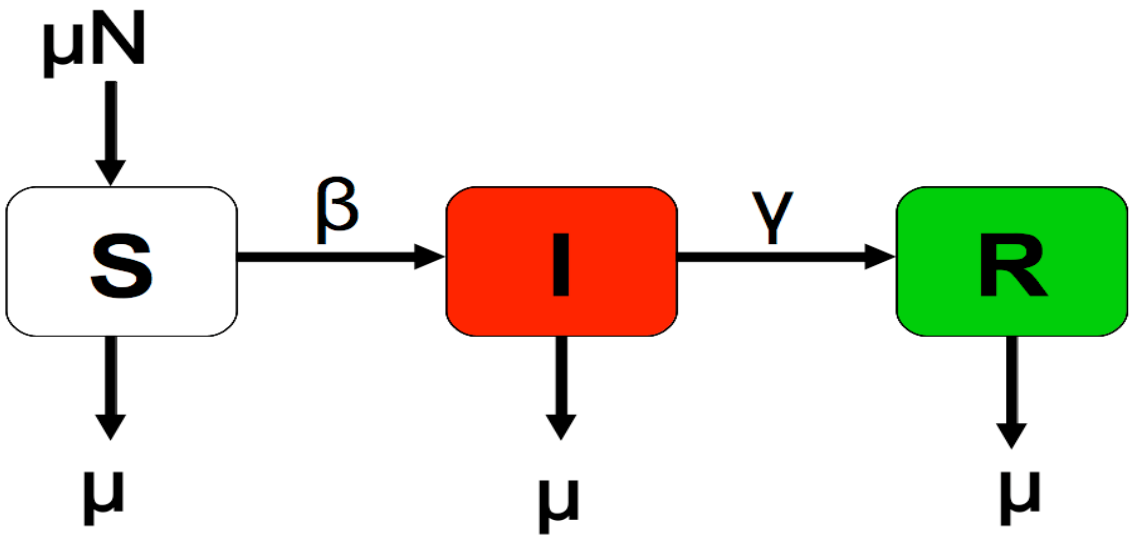
Deterministic compartmental models: SIRS

SIRS epidemic (disease with finite-time immunity)



$$\begin{aligned}
\frac{dS}{dt} &= -\beta SI + \delta R \\
\frac{dI}{dt} &= \beta SI - \gamma I \\
\frac{dR}{dt} &= \gamma I - \delta R
\end{aligned} \tag{3}$$

Deterministic SIR model with demography



$$\begin{aligned}
\frac{dS}{dt} &= -\beta SI + \mu(N - S) \\
\frac{dI}{dt} &= \beta SI - \gamma I - \mu I \\
\frac{dR}{dt} &= \gamma I - \mu R
\end{aligned} \tag{4}$$

Qualitative analysis on SIR:

Recall

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI \\ \frac{dI}{dt} &= \beta SI - \gamma I \\ \frac{dR}{dt} &= \gamma I \end{aligned} \tag{5}$$

Nodimensionlization: $P_S = \frac{S}{N}, P_I = \frac{I}{N}, P_R = \frac{R}{N}, \text{ and } \tau = \gamma t.$

$$\begin{aligned} P_S' := \frac{dP_S}{d\tau} &= -R_0 P_S P_I \\ P_I' := \frac{dP_I}{d\tau} &= (R_0 P_S - 1) P_I \\ P_R' := \frac{dP_S}{d\tau} &= P_I \end{aligned} \tag{6}$$

Qualitative analysis on SIR: ctd

Basic reproduction number: $R_0 = \dfrac{\beta N}{\gamma}$

- βN is the number of contacts made by one infective in an otherwise susceptible population per unit time that leads to an infection.
- $\dfrac{1}{\gamma}$ represents infectious period.

Therefore R_0 describes the total number of secondary infections produced when one infected individual is introduced into a host virgin population.

Qualitative analysis on SIR: ctd

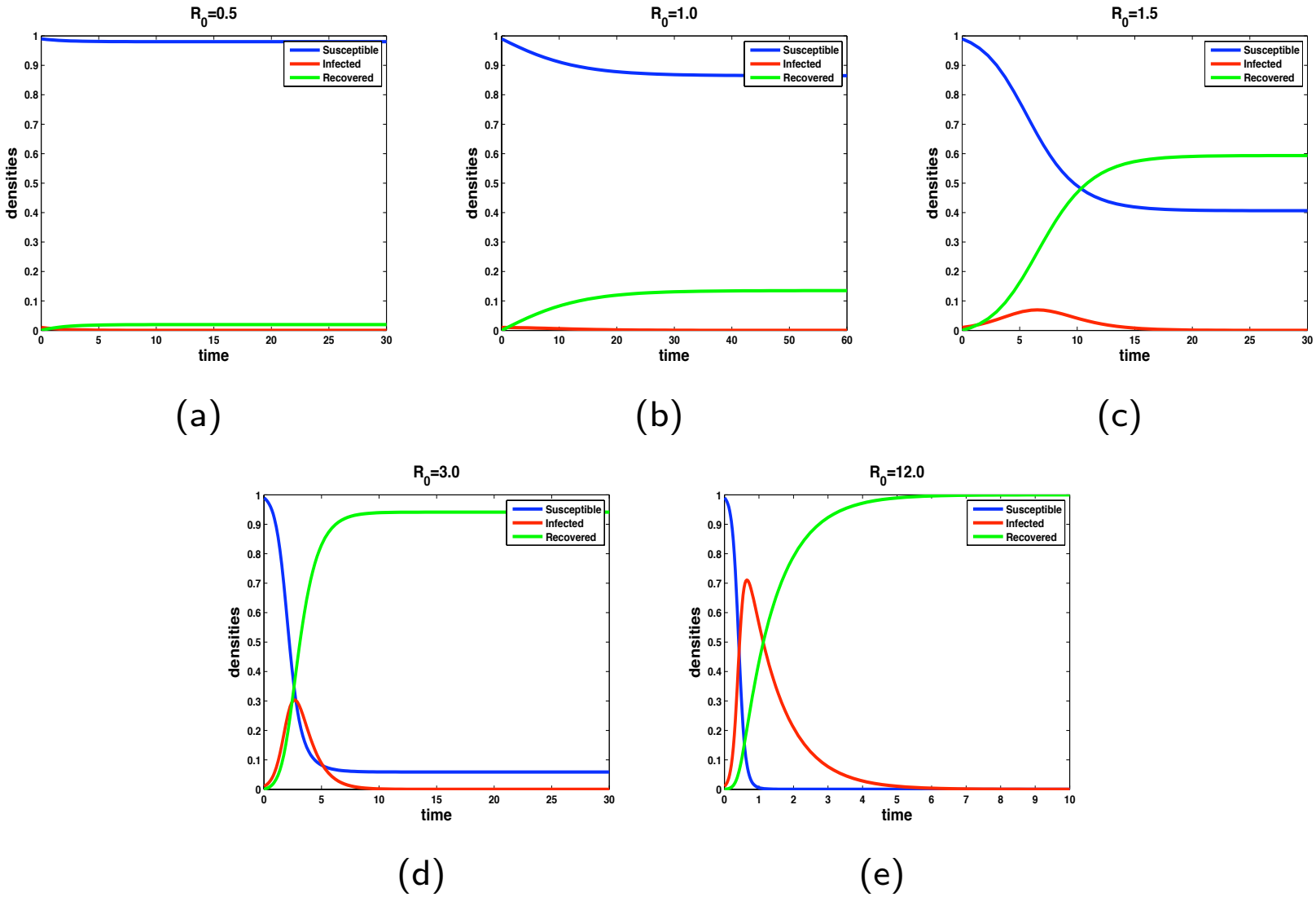
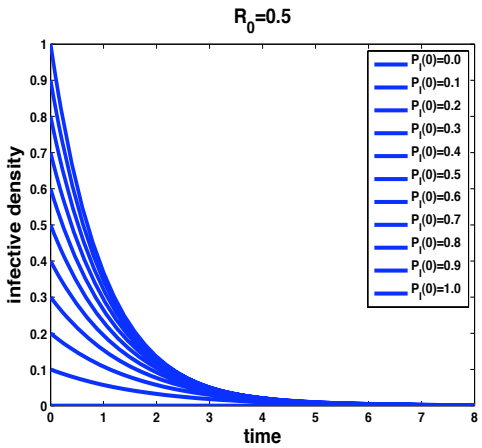


Figure: densities vs. time τ: $P_S = 0.99, P_I = 0.01$ and $P_R = 0$.

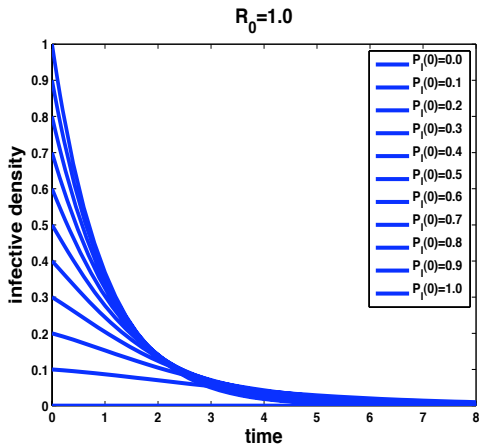
Qualitative analysis on SIR: ctd

$$
\begin{aligned}
P_S' &= -R_0 P_S P_I \\
P_I' &= (R_0 P_S - 1) P_I \\
P_R' &= P_I
\end{aligned}
\tag{7}
$$

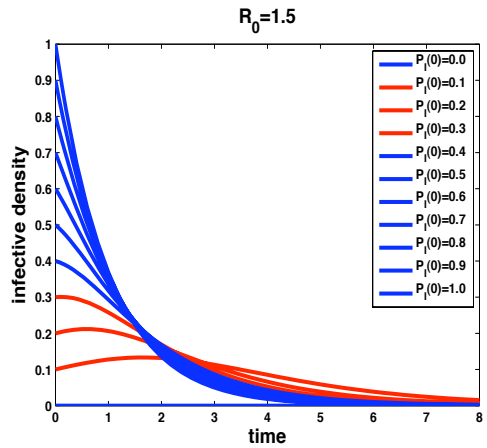
1. $R_0 \leq 1$: P_I decreases to zero. The infection dies out. No epidemics.
2. $R_0 > 1$: P_I increases initially when $P_S(0) > \dfrac{1}{R_0}$ and decays to zero as $t \to \infty$ (since $R_0 P_S(\tau) = R_0 P_S(0) \exp(-R_0 \int_0^\tau P_I(\xi) d\xi)$). Infection spreads out and finally disappears. Epidemics occur.



(a)

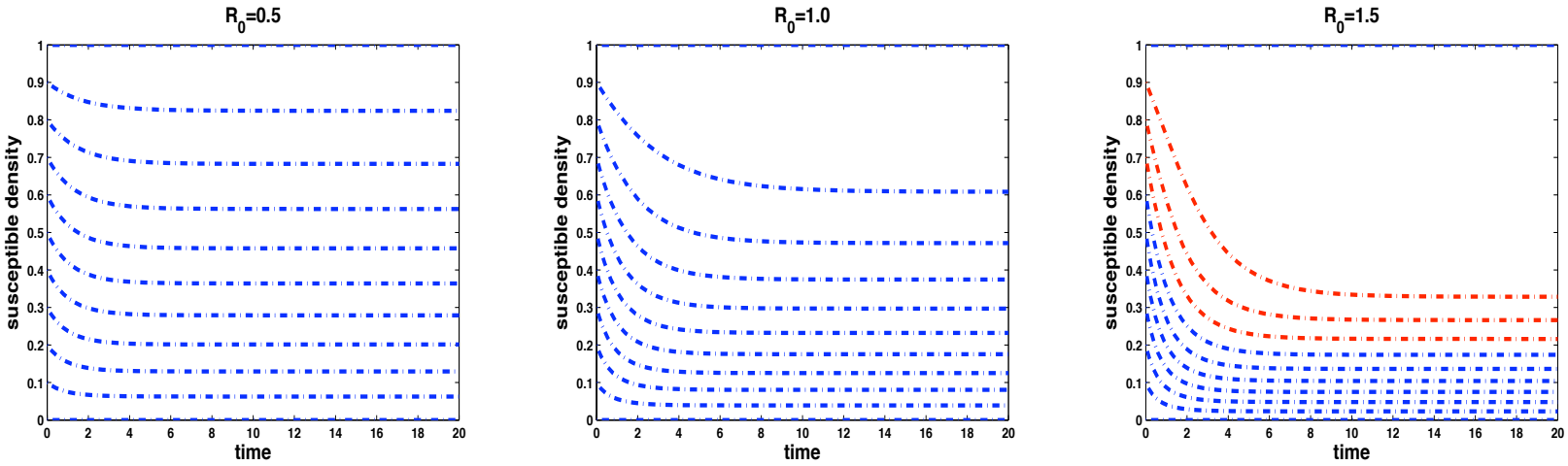


(b)



(c)

Qualitative analysis on SIR: ctd



(a) (b) (c)

Figure: P_S vs. time τ

Notice that

$$P_S + P_I + P_R = 1 \tag{8}$$

We have

$$(P_S + P_I + P_R)' = 0 \tag{9}$$

Qualitative analysis on SIR: ctd

By (7), $P_R' = P_I = -\frac{P_S'}{R_0 P_S}$. Hence

$$\left(P_S + P_I - \frac{1}{R_0} \ln P_S \right)' = 0. \tag{10}$$

(10) implies

$$P_S + P_I - \frac{1}{R_0} \ln P_S = P_S(0) + P_I(0) - \frac{1}{R_0} \ln P_S(0). \tag{11}$$

As $t \to \infty$, $P_I(\infty) \to 0$. The final size of susceptible populations, $P_S(\infty)$, satisfies

$$P_S(\infty) - \frac{1}{R_0} \ln P_S(\infty) = P_S(0) + P_I(0) - \frac{1}{R_0} \ln P_S(0) \tag{12}$$

Qualitative analysis on SIS:

SIS epidemic (disease w/o immunity)

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI + \gamma I \\ \frac{dI}{dt} &= \beta SI - \gamma I \end{aligned} \tag{13}$$

Since $S + I = N$, (13) can be reduced to a single equation

$$\frac{dI}{dt} = \beta I \left(\frac{\beta N - \gamma}{\beta} - I \right). \tag{14}$$

Let $K = \frac{\beta N - \gamma}{\beta}$. The solution of (14) is given by

$$I(t) = \begin{cases} K \left[1 + \left(\frac{K}{I(0)} - 1 \right) \exp(-sign\{I(0)\}\beta K t) \right]^{-1}, & K \neq 0 \\ \left(\frac{1}{I(0)} + \beta t \right)^{-1}, & \text{K=0.} \end{cases}$$

Qualitative analysis on SIS: ctd

$$\frac{dI}{dt} = \beta I (K - I). \tag{15}$$

Equilibrium $\left(\frac{dI}{dt} = 0\right)$: $I_* = 0$ (disease free) and $I_* = K$ (endemic).

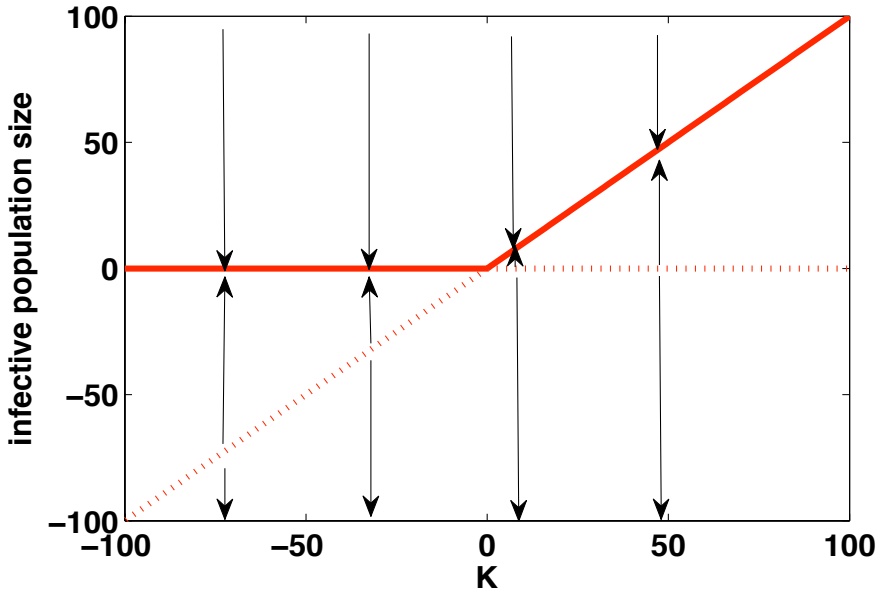
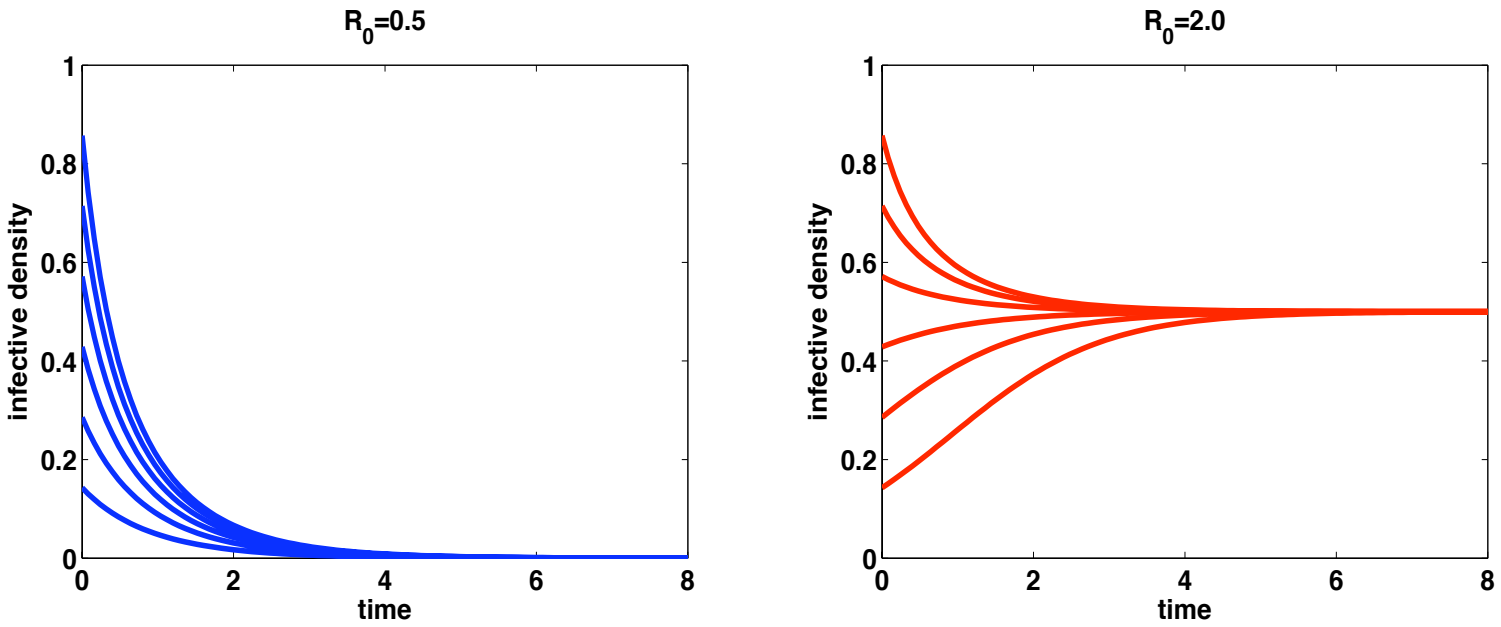


Figure: bifurcation diagram: I vs. K

Qualitative analysis on SIS: ctd

Note that $K = \frac{\gamma}{\beta}(R_0 - 1)$.



(a) (b)

Figure: P_I vs. time t

Qualitative analysis on SIS: ctd

Notice that

$$K > 0 \Longleftrightarrow R_0 \equiv \frac{\beta N}{\gamma} > 1.$$

1. $R_0 \leq 1$: I monotonically decreases to zero. Infection dies out. No epidemics.

2. $R_0 > 1$:
   - If $I(0) < K$, I monotonically increases to its 'carry capacity', K;
   - If $I(0) > K$, I monotonically decreases to K.

   Infection spreads out and an endemics happens.

Comments:

1. The deterministic compartmental models presented here are only valid in case of sufficiently large populations. How large will be sufficient for population sizes?
2. How to estimate the parameters in the models?
3. The population structure and randomness are neglected. When population structure or randomness have to be considered in the models?

Thanks!