

Foundations of dynamic modeling: The SIR Model Family

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Goals

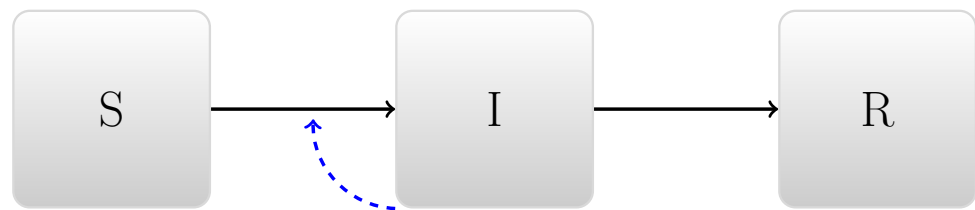
- This lecture will:
 - introduce the idea of dynamical modeling
 - explain why dynamical modeling is a key tool for understanding infectious disease
 - discuss and demonstrate simple dynamical models from the SIR model family
 - investigate some insights that can be gained from these models

Dynamical modeling connects scales

- Start with rules about how things change in short time steps
 - Usually based on *individuals*
- Calculate results over longer time periods
 - Usually about *populations*

Compartmental models

Divide people into categories:



- Susceptible \rightarrow Infectious \rightarrow Recovered

What determines transition rates?

- People get better independently
- People get infected by infectious people

Conceptual modeling

- What is the final result?
- When does disease increase, decrease?

Dynamic implementation

- Requires assumptions about recovery and transmission
- The *conceptually simplest* implementation uses Ordinary Differential Equations (ODEs)
 - Other options may be more realistic
 - Or simpler in practice

Recovery

- Infectious people recover at *per capita* rate γ
 - Total recovery rate is γI
 - Mean time infectious is $D = 1/\gamma$

Transmission

- Susceptible people get infected by:
 - Going around and contacting people (rate c)
 - Some of these people are infectious (proportion I/N)
 - Some of these contacts are effective (proportion p)
- Per capita rate of becoming infected is $cpI/N \equiv \beta I/N$
- Population-level transmission rate is $\mathcal{T} = \beta SI/N$

Another perspective on transmission

- Infectious people infect others by:
 - Going around and contacting people (rate c)
 - Some of these people are susceptible (proportion S/N)
 - Some of these contacts are effective (proportion p)
- Per capita rate of infecting others is $cpS/N \equiv \beta S/N$
- Population-level transmission rate is $\mathcal{T} = \beta SI/N$

The basic reproductive number

- \mathcal{R}_0 is the number of people who would be infected by an infectious individual *in a fully susceptible population*.
- $\mathcal{R}_0 = \beta/\gamma = \beta D = (cp)D$
 - c : Contact Rate
 - p : Probability of transmission (infectivity)
 - D : Average duration of infection
- A disease can invade a population if and only if $\mathcal{R}_0 > 1$.

ODE implementation

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

Spreadsheet implementation

<http://tinyurl.com/SIR-MMED-2023>

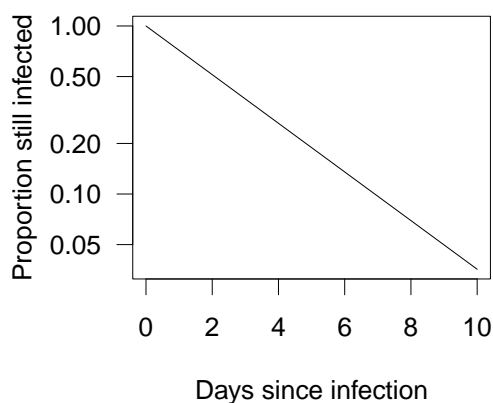
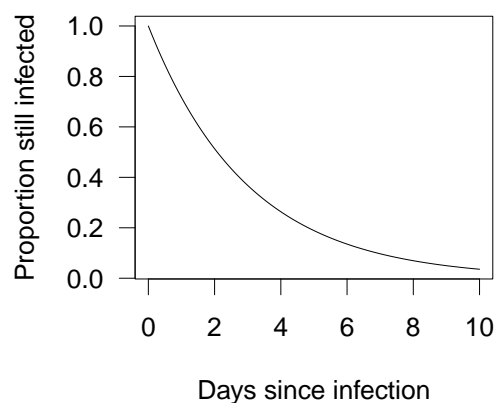
ODEs and mechanistic models

- What is the relationship between the spreadsheet and the ODE model we started with?
 -
 -

ODE assumptions

- Lots and lots of people
- Perfectly mixed

ODE assumptions



- Waiting times are exponentially distributed
- Rarely realistic
 - but sometimes OK for a particular application

Scripts vs. spreadsheets

- Scripts are more transparent, less redundant
- Spreadsheets are more intuitive for simple problems

More about transmission

- $\beta = pc$
 - What is a contact?
 - What is the probability of transmission?
- Sometimes this decomposition is clear
- But usually it's not
- So we often start by estimating β directly

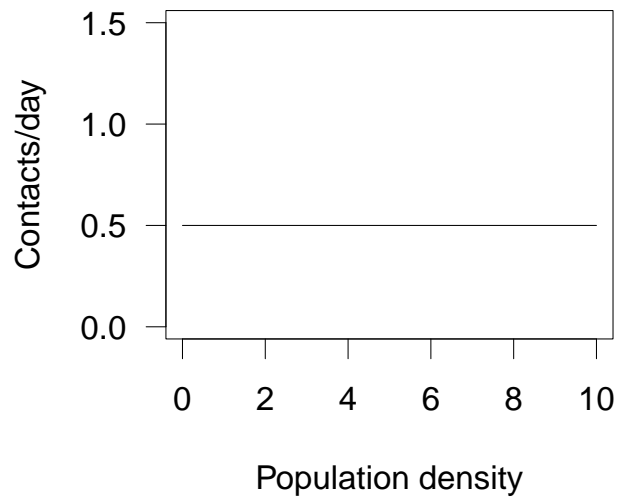
Population sizes

- How does β change with population size?
- We can make different assumptions about this
 - It may increase with population size, or not
- If population size changes we have to *consider* the question

Population sizes

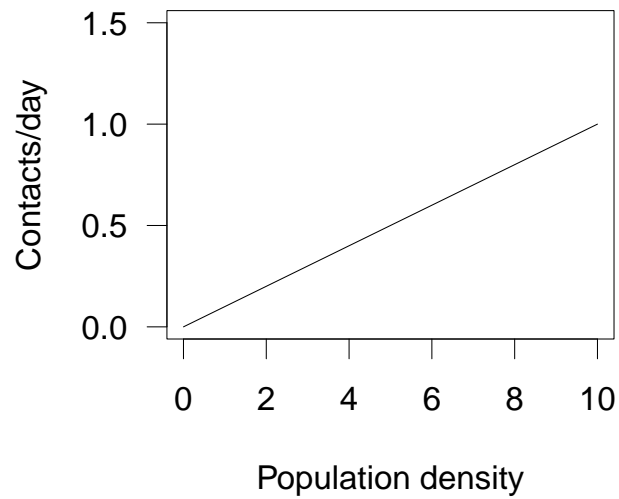
$$\begin{aligned}\frac{dS}{dt} &= -\beta(N)\frac{SI}{N} \\ \frac{dI}{dt} &= \beta(N)\frac{SI}{N} - \gamma I \\ \frac{dR}{dt} &= \gamma I\end{aligned}$$

Standard incidence



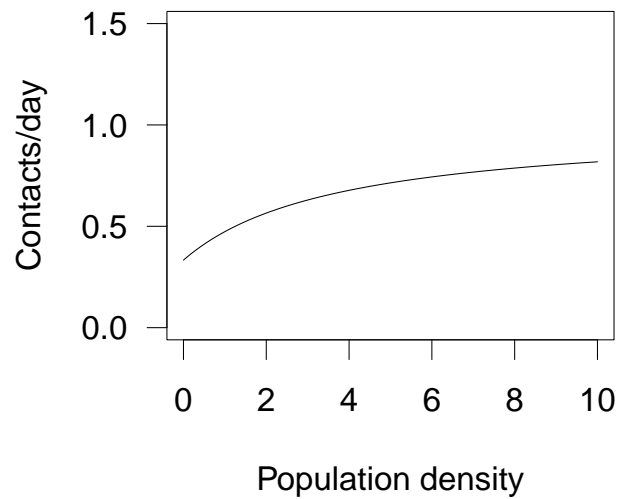
- $\beta(N) = \beta_0$
- $\mathcal{T} = \frac{\beta_0 SI}{N}$
- Also known as *frequency-dependent* transmission

Mass action



- $\beta(N) = \alpha_0 N$
- $\mathcal{T} = \alpha_0 SI$
- Also known as *density-dependent* transmission

General



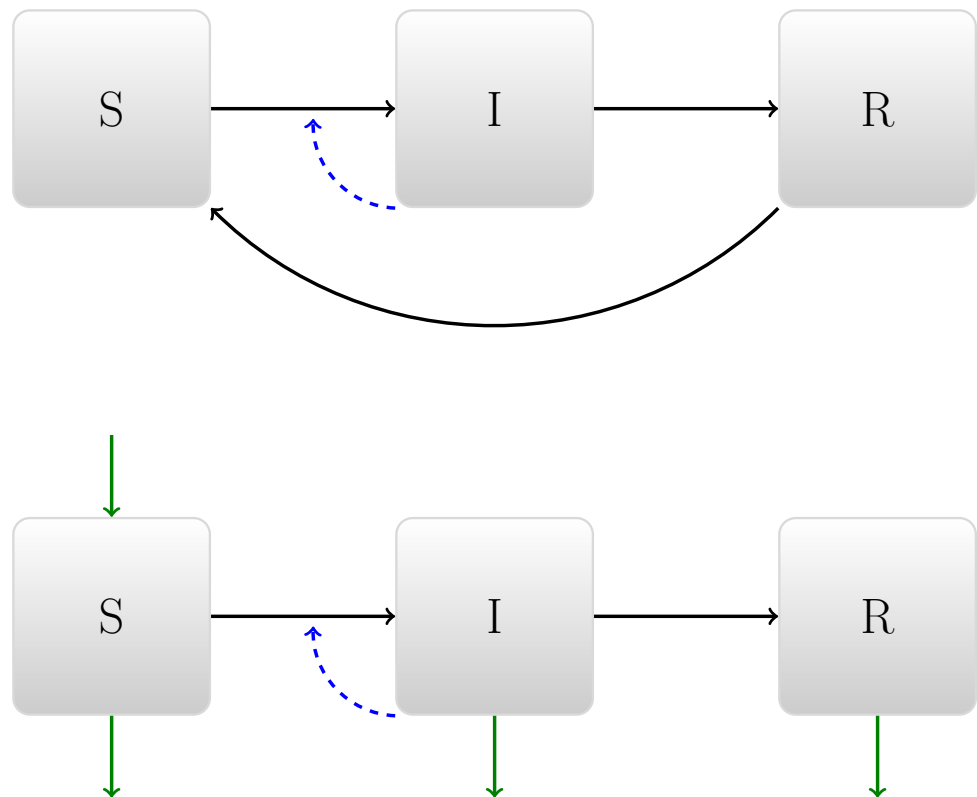
- Per-capita rate:
 - May not go to zero when N does

- May not go to ∞ when N does

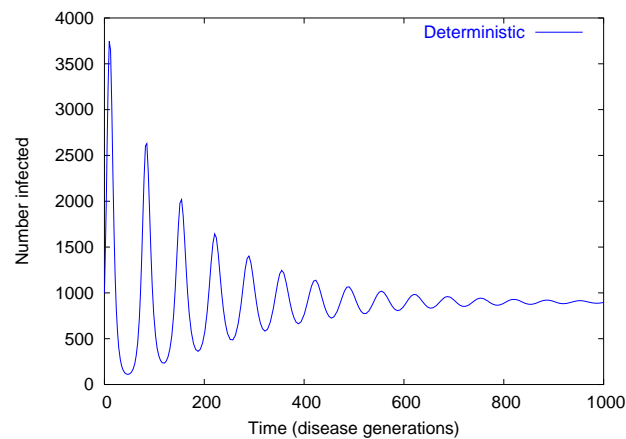
Digression – units

- $\mathcal{T} = \beta SI/N : [\text{ppl}/\text{time}]$
- $\beta : [1/\text{time}]$
 - The true β always has people in the numerator and the denominator
 - $\beta/\gamma = \beta D : [1]$
- $\mathcal{T} = \alpha SI : [\text{ppl}/\text{time}]$
 - Mass-action incidence, $\alpha : [1/(\text{people} \cdot \text{time})]$

Closing the circle

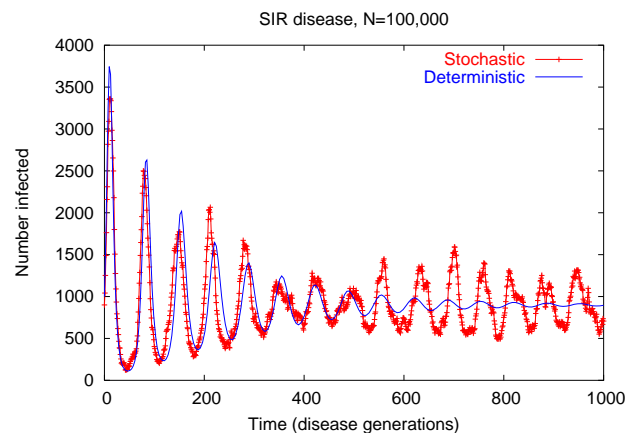


Tendency to oscillate



- Many susceptibles \rightarrow many infections \rightarrow few susceptibles \rightarrow few infections \rightarrow ...
- Oscillations in simple models tend to be “damped”

With individuality



- Treating individuals as individuals can produce substantial oscillations even in large populations
- Interaction between random effects and the different time scales (of infection and recovery)

Summary

- Dynamic models are an essential tool because they allow us to link between scales
- There are many ways to construct and implement dynamic models

- Very simple models can provide useful insights
 - Reproductive numbers and thresholds
 - Tendency for oscillation (and tendency for damping)
- More complex models can provide more detail, but also require more assumptions, and more choices
- Understanding simple models can help guide our understanding of more complicated models