

Foundations of dynamic modeling: The SIR Model Family

Jonathan Dushoff, McMaster University

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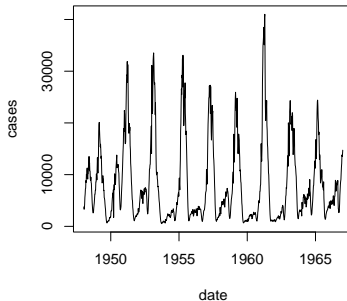
Goals

- ▶ 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



Measles reports from England and Wales

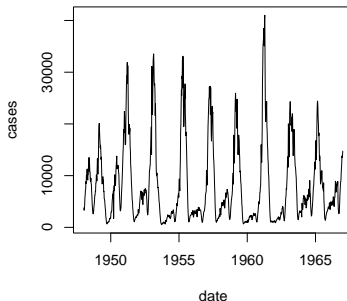


- ▶ Start with rules about how things change in short time steps
 - ▶ Usually based on *individuals*
- ▶ Calculate results over longer time periods
 - ▶ Usually about *populations*

Dynamical modeling is a bridge



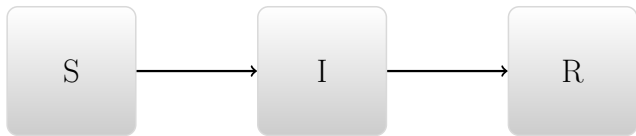
Measles reports from England and Wales



- ▶ If we see (or change) actions, how can we predict population-level outcomes?
- ▶ If we observe population-level outcomes (or their changes), what can we infer about individual-level actions?

Compartmental models (repeat)

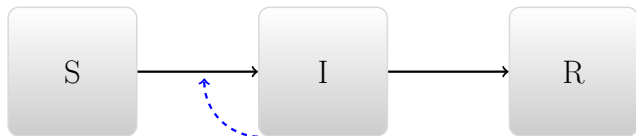
Divide people into categories:



- ▶ Susceptible \rightarrow Infectious \rightarrow Recovered
- ▶ How well does this reflect the simple transmission model?
- ▶ What's missing?

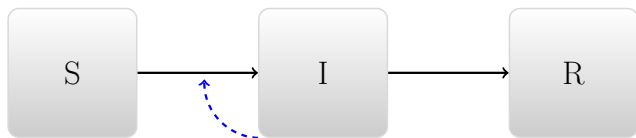
Compartmental models (repeat)

Divide people into categories:



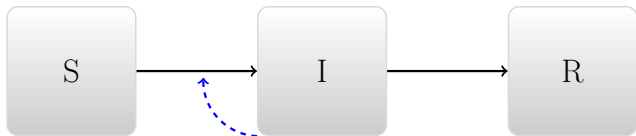
► Susceptible \rightarrow Infectious \rightarrow Recovered

What determines transition rates?

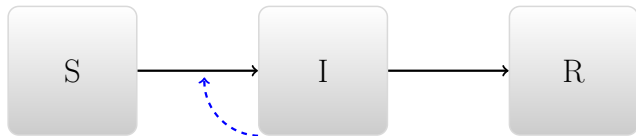


- ▶ People get better independently
- ▶ People get infected by infectious people

Conceptual modeling (preview)

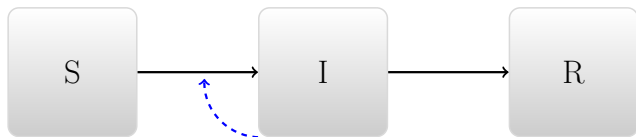


Conceptual modeling



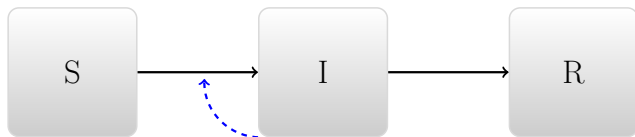
- ▶ What is the final result?
- ▶ When does disease increase, decrease?

Dynamic implementation



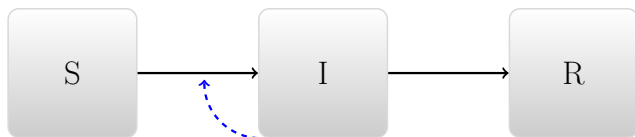
- ▶ Requires assumptions about recovery and transmission
- ▶ The *conceptually simplest* implementation uses Ordinary Differential Equations (ODEs)
 - ▶ Other options may be more realistic
 - ▶ Or simpler in practice

Recovery



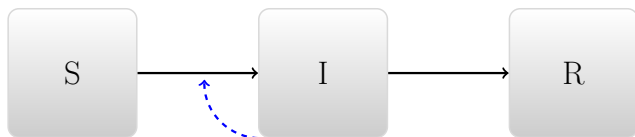
- ▶ Infectious people recover at *per capita* rate γ
 - ▶ Total recovery rate is γI
 - ▶ Mean time infectious is $D = 1/\gamma$

Transmission



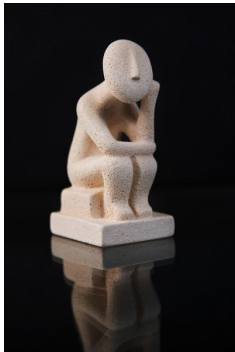
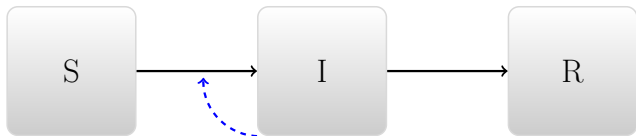
- ▶ Susceptible people get infected by:
 - ▶ Going around and contacting people (rate c)
 - ▶ Some of these people are infectious (proportion I/N)
 - ▶ Some of these contacts are effective (proportion p)
- ▶ Per capita rate of becoming infected is $cpI/N \equiv \beta I/N$
- ▶ Population-level transmission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

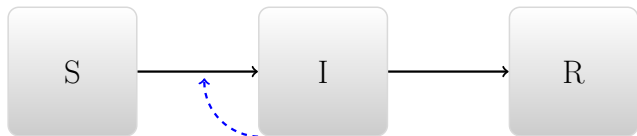
Conceptual modeling (repeat)



The basic reproductive number

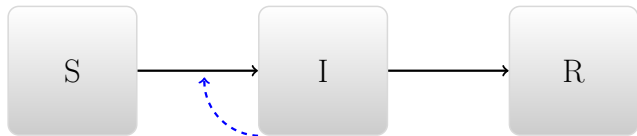
- ▶ \mathcal{R}_0 is the expected number of people infected by an infectious individual *in a fully susceptible population*.
- ▶ $\mathcal{R}_0 = \beta/\gamma = \beta D = (cp)D$
 - ▶ c : Contact Rate
 - ▶ p : Probability of transmission (infectivity)
 - ▶ D : Average duration of infection
- ▶ A disease 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-2024>

What have we learned?

- ▶ Why does the number of infected decline?
 - ▶ * In our model world, the only reason for change is that we have fewer susceptibles
 - ▶ * In the real world, there may be other reasons
- ▶ \mathcal{R}_0 is the expected number of people infected by an infectious individual *in a fully susceptible population*.
- ▶ \mathcal{R}_{eff} is the expected number of people infected by an infectious individual
 - ▶ * In this model $\mathcal{R}_{\text{eff}} = \mathcal{R}_0 S/N$

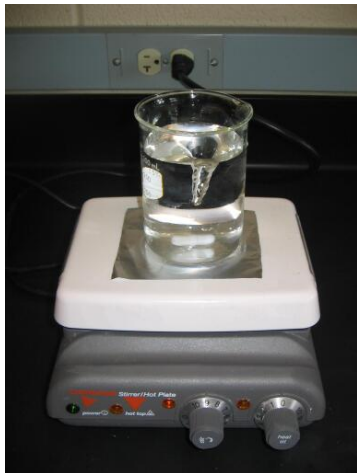
What is our graph showing?

- ▶ Prevalence?
 - ▶ * The number (or proportion) of the population currently infected
- ▶ Incidence?
 - ▶ * The number (or proportion) of the population currently *getting* infected *per unit time*
- ▶ * The graph shows prevalence (number in box 1))
- ▶ * Incidence is found in our transmission column

ODEs and mechanistic models

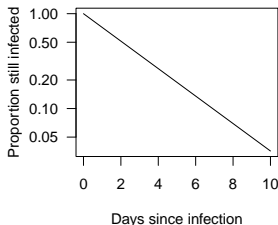
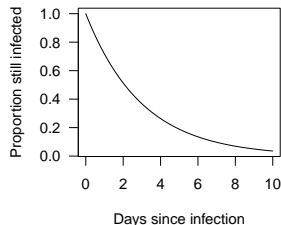
- ▶ What is the relationship between the spreadsheet and the ODE model we started with?
 - ▶ * The ODE is the limit when $\Delta T \rightarrow 0$
 - ▶ * We need technical software to do this well

Simple model assumptions (population)



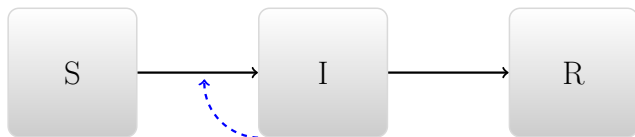
- ▶ Lots and lots of people
- ▶ Perfectly mixed

Simple model assumptions (time)



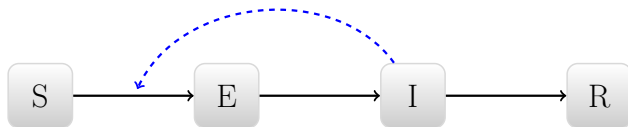
- ▶ Probability of leaving does not depend on time-in-box
 - ▶ → Waiting times are exponentially distributed
- ▶ Rarely realistic
 - ▶ but sometimes OK for a particular application
 - ▶ We will talk more about “model worlds”

Simple model assumptions (structure)

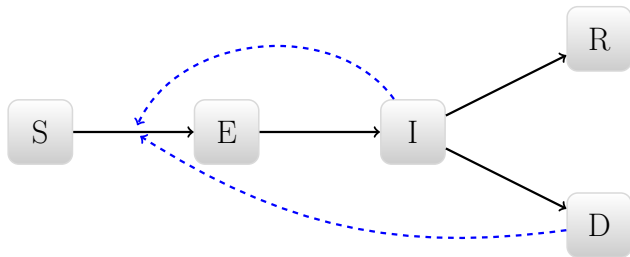


- ▶ How realistic is this picture?
 - ▶ * Infected people are not always sick
 - ▶ * Infected people are not always *infectious*

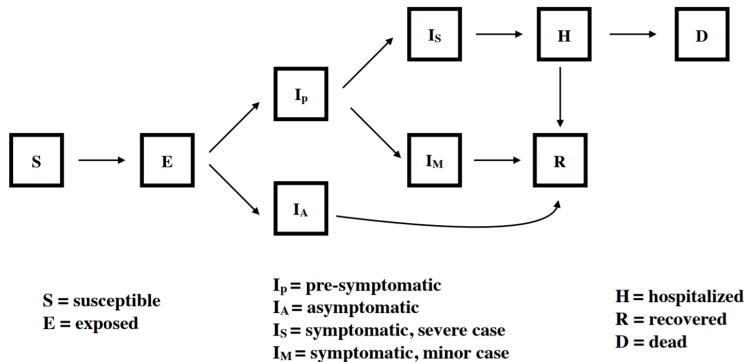
Delayed infectiousness



Ebola

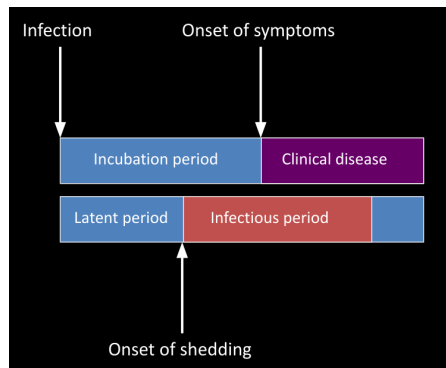


COVID-19



Childs et al., <http://covid-measures.stanford.edu/>

Time distributions



- ▶ Latent period is time until disease can spread
- ▶ Incubation period is time until symptoms start
- ▶ The relationship is very important!

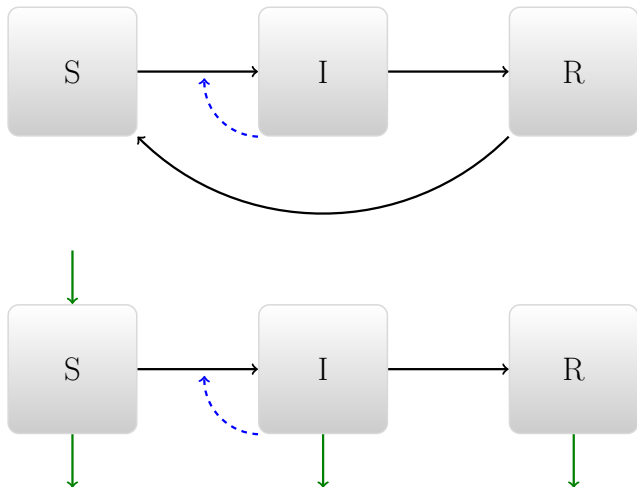
Time distributions

Table 3.1 Incubation, latent and infectious periods (in days) for a variety of viral and bacterial infections. Data from Fenner and White (1970), Christie (1974), and Benenson (1975)

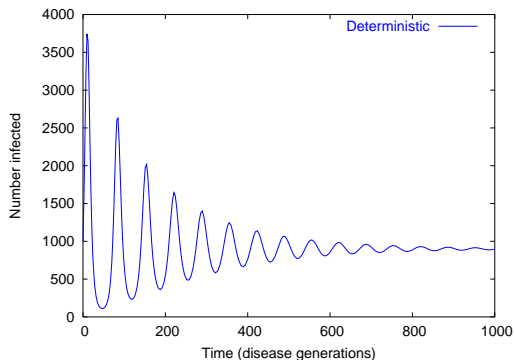
Infectious disease	Incubation period	Latent period	Infectious period
Measles	8–13	6–9	6–7
Mumps	12–26	12–18	4–8
Whooping cough (pertussis)	6–10	21–23	7–10
Rubella	14–21	7–14	11–12
Diphtheria	2–5	14–21	2–5
Chicken pox	13–17	8–12	10–11
Hepatitis B	30–80	13–17	19–22
Poliomyelitis	7–12	1–3	14–20
Influenza	1–3	1–3	2–3
Smallpox	10–15	8–11	2–3
Scarlet fever	2–3	1–2	14–21

Anderson and May (1982) Science

Closing the circle

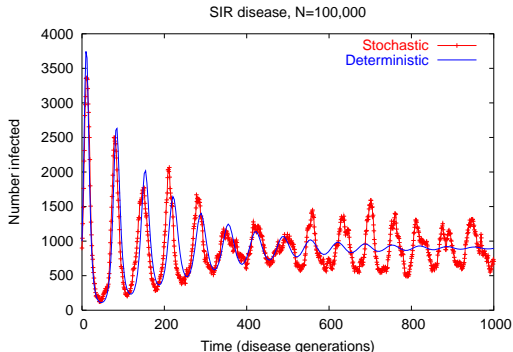


Tendency to oscillate



- ▶ 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



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