Foundations of dynamic modeling:
The SIR Model Family

Jonathan Dushoff, McMaster University

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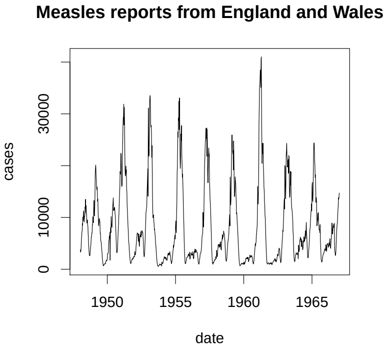
Goals

- 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

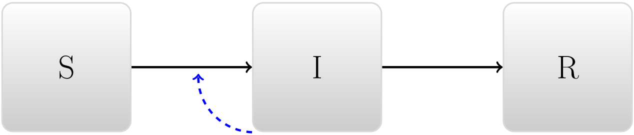
Dynamical modeling connects scales



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

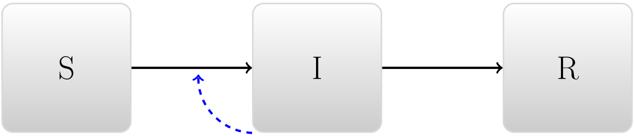
Compartmental models

Divide people into categories:



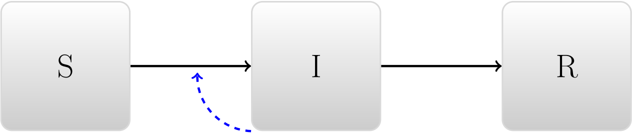
- Susceptible $\rightarrow$ Infectious $\rightarrow$ Recovered

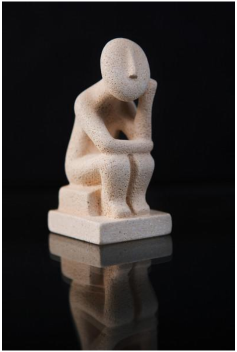
What determines transition rates?



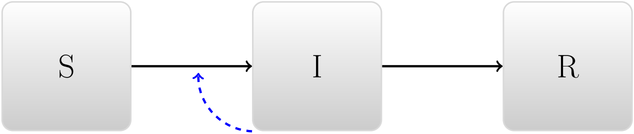
- People get better independently
- People get infected by infectious people

Conceptual modeling (present)



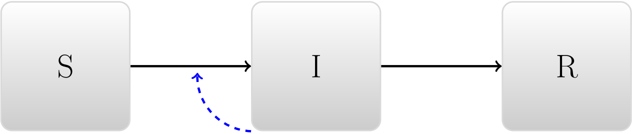


Conceptual modeling



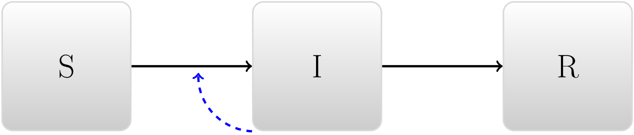
- What is the final result?
- When does disease increase, decrease?

Dynamic implementation



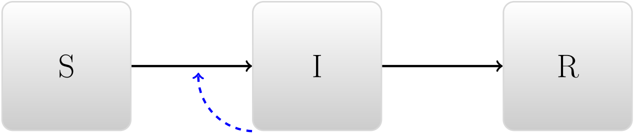
- Requires assumptions about recovery and transmission
- The *conceptually simplest* implementation uses Ordinary Differential Equations (ODEs)
  - Other options may be more realistic
  - Or simpler in practice

Recovery



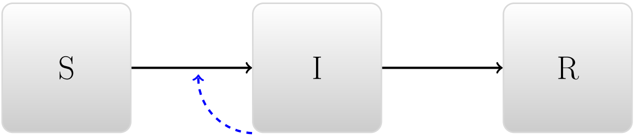
- Infectious people recover at *per capita* rate γ
  - Total recovery rate is γI
  - Mean time infectious is $D = 1/\gamma$

Transmission



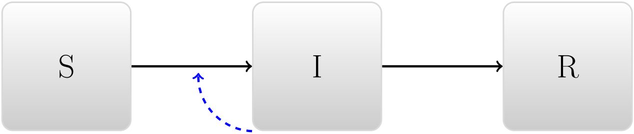
- Susceptible people get infected by:
  - Going around and contacting people (rate c)
  - Some of these people are infectious (proportion I/N)
  - Some of these contacts are effective (proportion p)
- Per capita rate of becoming infected is $cpI/N \equiv \beta I/N$
- Population-level transmission rate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

Another perspective on transmission



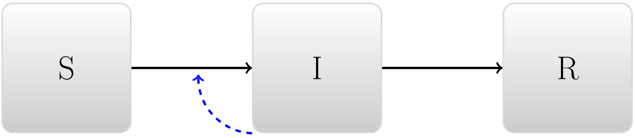
- Infectious people infect others by:
  - Going around and contacting people (rate c)
  - Some of these people are susceptible (proportion S/N)
  - Some of these contacts are effective (proportion p)
- Per capita rate of infecting others is $cpS/N \equiv \beta S/N$
- Population-level transmission rate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

ODE implementation



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

Spreadsheet implementation

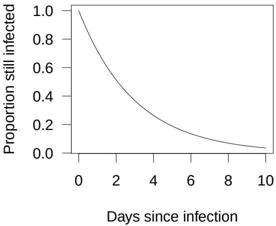


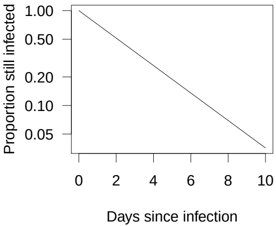
`http://tinyurl.com/SIR-MMED-2019`

ODE assumptions

- Lots and lots of people
- Perfectly mixed

ODE assumptions





- Waiting times are exponentially distributed
- Rarely realistic

Scripts vs. spreadsheets

	Susceptibles	Infectious	Removed	Total
	people	people	people	peopl
0	999	1	0	
1	998.6004	1.1996	0.2	
2	998.121231584064	1.438848415936	0.43992	
3	997.546773522873	1.72553679393953	0.7276896831872	
4	996.858252058318	2.06895089970737	1.07279704197511	
5	996.033271747327	2.48014103075661	1.48658722191658	
6	995.045150553223	2.97223401870901	1.9826154280679	
7	993.862147734573	3.56079003361749	2.5770622318097	
8	992.446573962396	4.26420579907117	3.2892202385332	
9	990.753775388012	5.10416321364044	4.14206139834744	
10	988.730987798368	6.1061181605567	5.16289404107552	
11	986.316064502168	7.29981782464567	6.38411767318686	
12	983.436093466813	8.71982529507146	7.844081238116	
13	980.005937097253	10.4060166056164	9.58804629713029	

Sheet1 Sheet2

```
infec <- bet*S*I/N
recov <- I/D

Sdot <- -infec
Idot <- infec - recov
Rdot <- recov
```

- Scripts are more transparent, less redundant
- Spreadsheets are more intuitive for simple problems
- I don’t personally use spreadsheets for science, just teaching

ICI3D

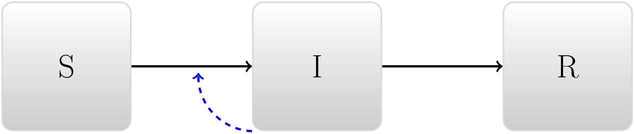
More about transmission

- $\beta = pc$
  - What is a contact?
  - What is the probability of transmission?
- Sometimes this decomposition is clear
- But usually it's not

Population sizes

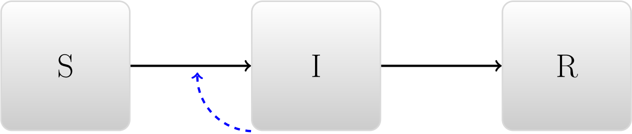
- How does β change with population size?
- Recall that β is the *per capita* rate of contacts

Population sizes (present)



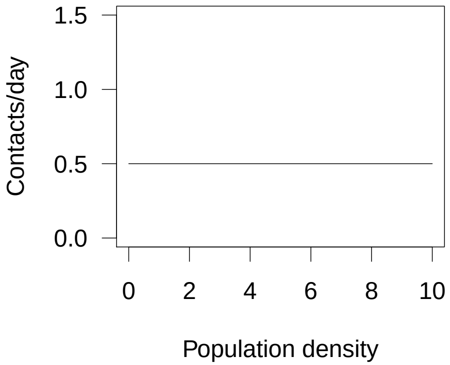
$$
\begin{aligned}
\frac{dS}{dt} &= -\beta\frac{SI}{N} \\
\frac{dI}{dt} &= \beta\frac{SI}{N} - \gamma I \\
\frac{dR}{dt} &= \gamma I
\end{aligned}
$$

Population sizes



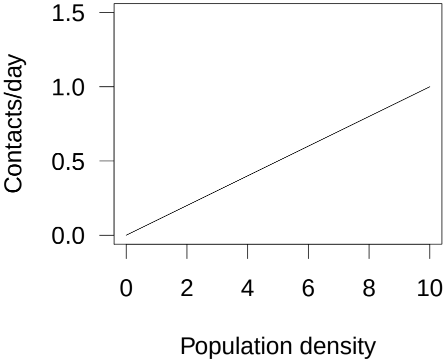
$$\frac{dS}{dt} = -\beta(N)\frac{SI}{N}$$
$$\frac{dI}{dt} = \beta(N)\frac{SI}{N} - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

Standard incidence



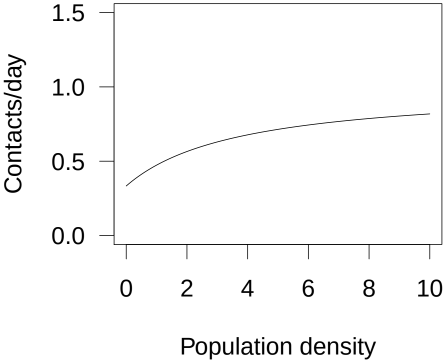
- $\beta(N) = \beta_0$
- $\mathcal{T} = \frac{\beta_0 SI}{N}$
- Also known as *frequency-dependent* transmission

Mass action



- $\beta(N) = \beta_1 N$
- $\mathcal{T} = \beta_1 SI$
- Also known as *density-dependent* transmission

General

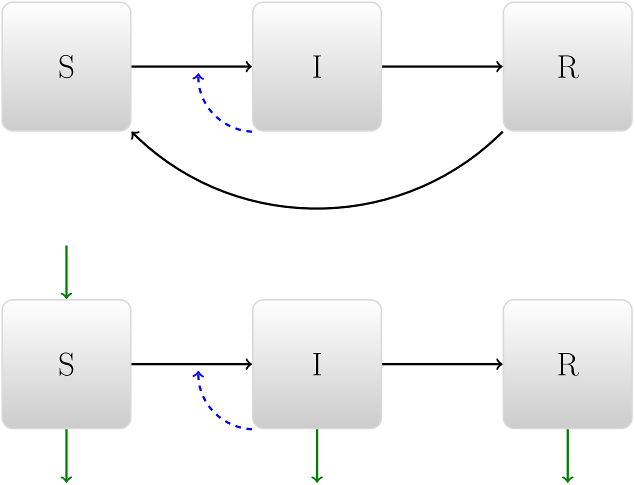


- Per-capita rate:
  - May not go to zero when N does
  - May not go to ∞ when N does

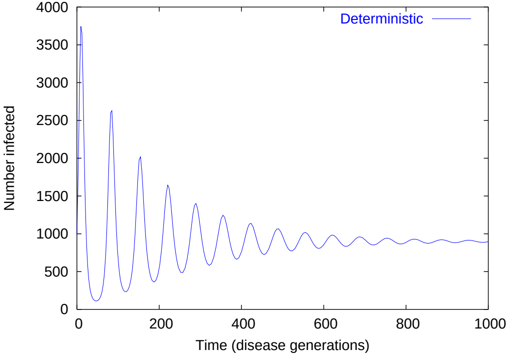
Digression – units

- $\mathcal{T} = \beta SI/N$: [ppl/time]
- β : [1/time]
  - $\beta/\gamma = \beta D$: [1]
  - Standard incidence, β_0 : [1/time]
  - Mass-action incidence, β_1 : [1/(people · time)]

Closing the circle

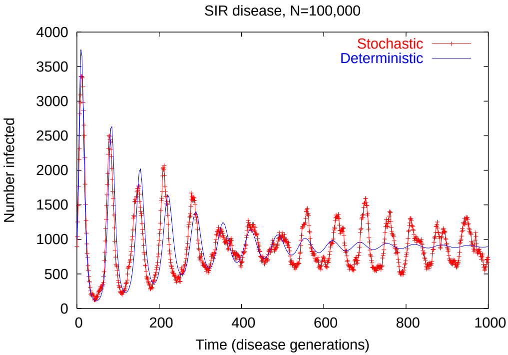


Tendency to oscillate



- Many susceptibles $\rightarrow$ many infections $\rightarrow$ few susceptibles $\rightarrow$ few infections $\rightarrow \ldots$
- 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