UNIT 8: Infectious disease

1 Introduction

Infectious disease

- Extremely common
- Huge impacts on ecological interactions
- A form of exploitation, but doesn't fit well into our previous modeling framework
  - How many people are there?
  - How many influenza viruses are there?
  - How do they find each other?

Disease agents

- Poll: Name an infectious agent that causes disease in humans.
- Disease agents vary tremendously:
  - Most **viruses** have just a handful of genes that allow them to hijack a cell and get it to make virus copies
    *
  - **Bacteria** are independent, free-living cells with hundreds or thousands of chemical pathways
    *
  - **Eukaryotic** pathogens are nucleated cells who are more closely related to you than they are to bacteria
    *

Microparasites

- For infections with small pathogens (viruses and bacteria), we don't attempt to count pathogens, but instead divide disease into stages
  - Latently infected (infected but not yet infectious)
  - Infectious
  - Recovered

Microparasite models

- We model microparasites by counting the number of hosts in various **states**:
  - **Susceptible** individuals can become infected
  - **Infectious** individuals are infected and can infect others
  - **Resistant** individuals are not infected and cannot become infected
- More complicated models include other states:
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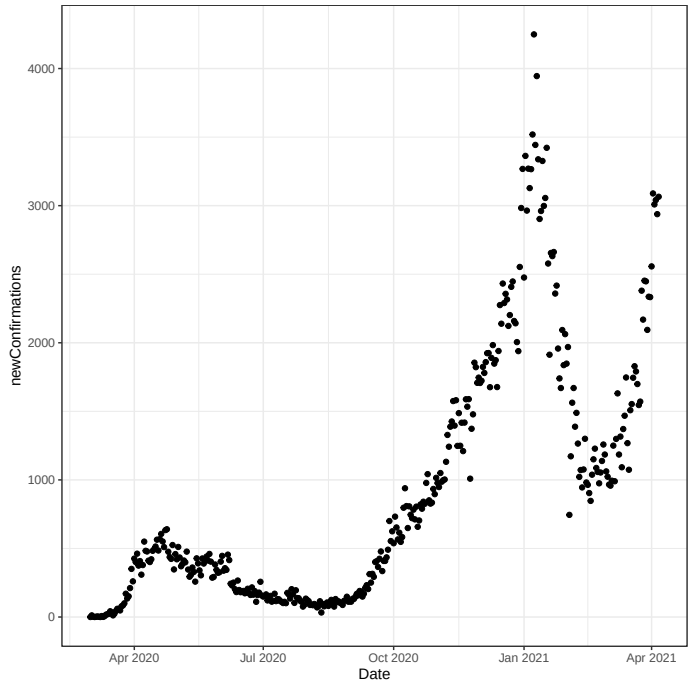
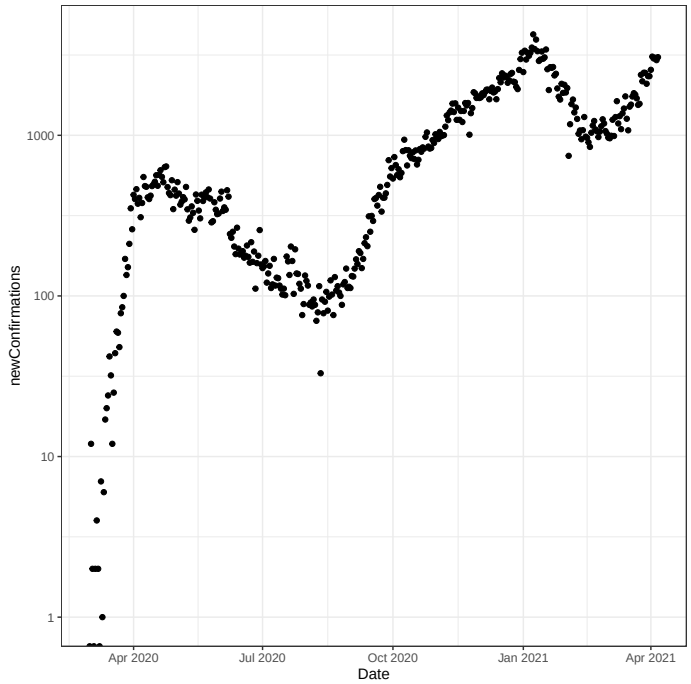
Models as tools

- Models are the tools that we use to connect scales:
  - individuals to populations
  - single actions to trends through time

2 Rate of spread

COVID in Ontario





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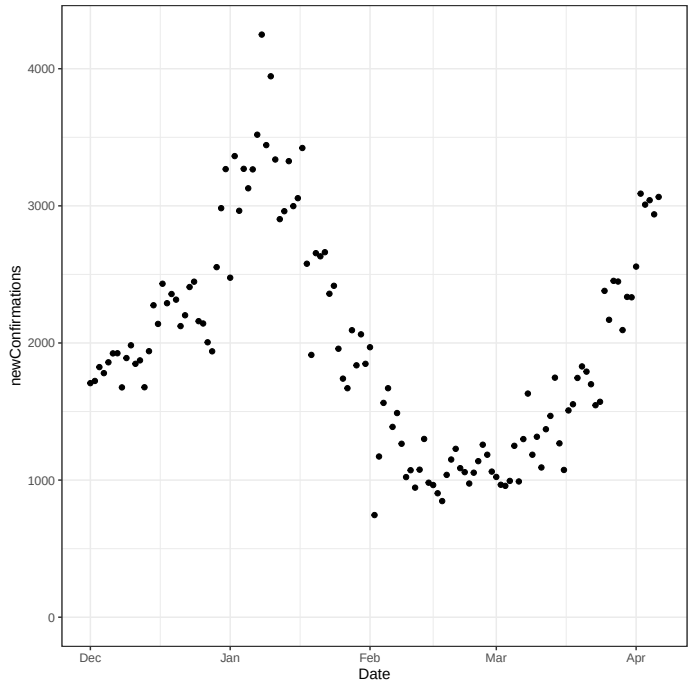

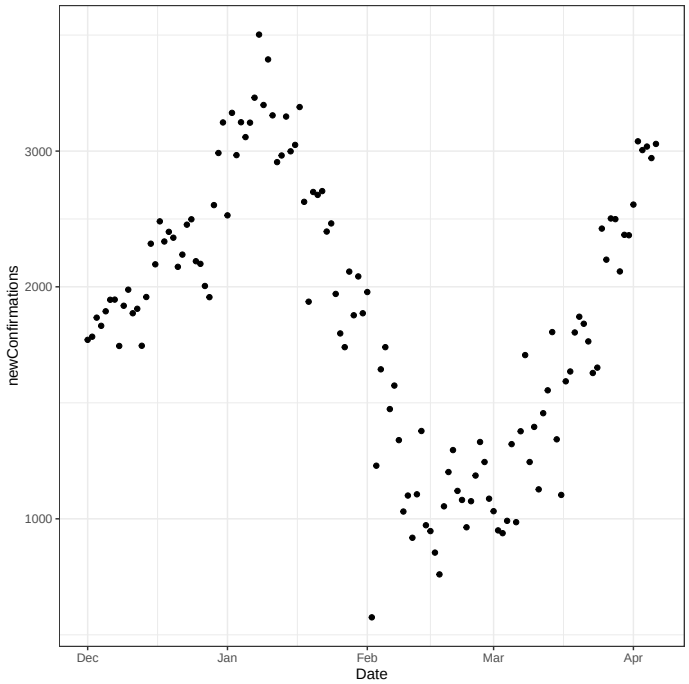

Scales

- Which scale should we look at?
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Population biology

- What quantities do we want to look at?
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Instantaneous rate of growth r

- What are the components?
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    - *
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- How do you think we estimate?
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    - *
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    - * Individuals $\leftrightarrow$ Populations

Reproductive number $\mathcal{R}$

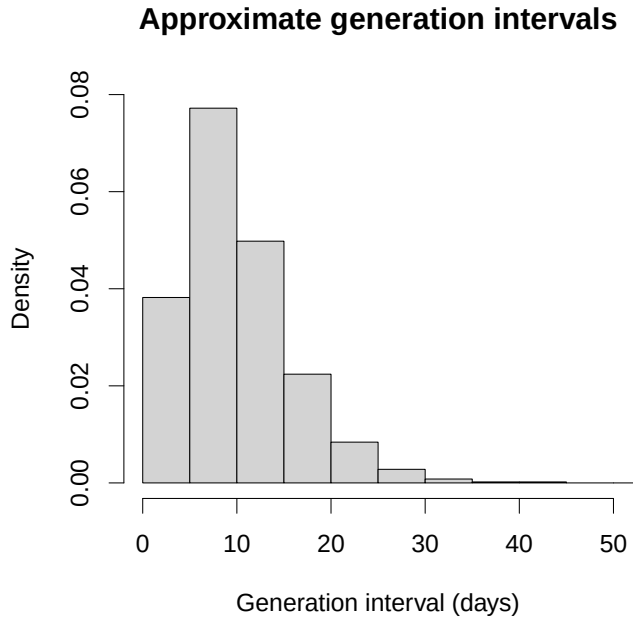
- What is it?
  -
- Why do we want this?
  -
- How do we calculate it?
  -

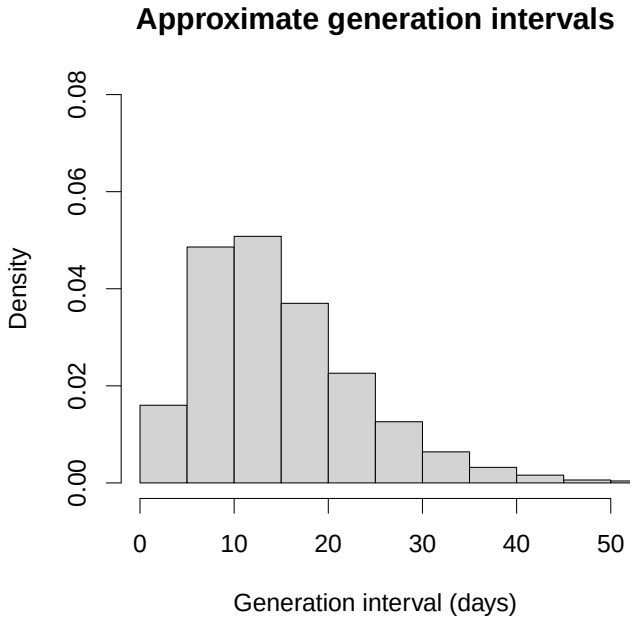
Example

- $r \approx 0.14/\,\text{day}$
- What is our estimate of $\mathcal{R}$?
  - When average length of infection $L = 5\,\text{day}$?
  - When average length of infection $L = 10\,\text{day}$?

Generation intervals

- Researchers try to estimate the *proportion* of transmission that happens for different **ages of infection**
- How long from the time you are *infected* to the time you *infect someone else*?
- Analogous to a life table
- The effective generation time $\hat{G}$ has units of time
  - And is hard to calculate, like λ in a structured model





Speed and risk

- Which is more dangerous, a fast disease, or a slow disease?
  - How are we measuring speed?
  - How are we measuring danger?
  - *What do we already know?*

Generation time and risk

- If we know $\mathcal{R}$, what does the generation time tell us about r?
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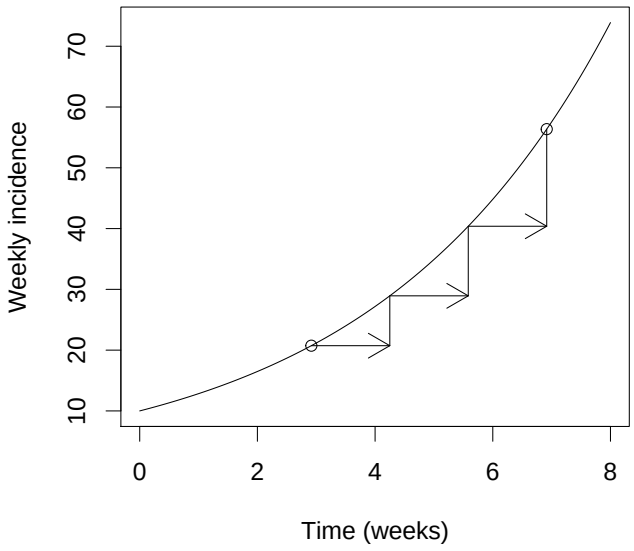
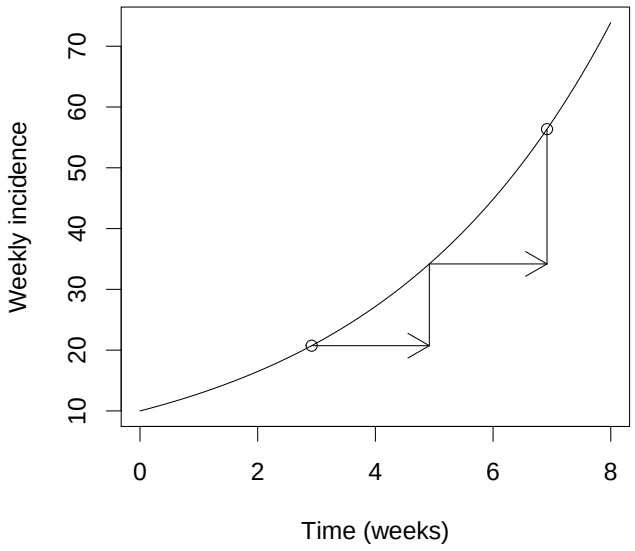
- If we know r, what does the generation time tell us about $\mathcal{R}$?
  -
- $\mathcal{R} = \exp(r\hat{G})$





- $\mathcal{R} = \exp(r\hat{G})$
- An intuitive view:
  - Epidemic speed = Generation strength $\times$ Generation speed
- If we know generation speed, then a faster epidemic speed means:
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- If we know epidemic speed, a faster generation speed means
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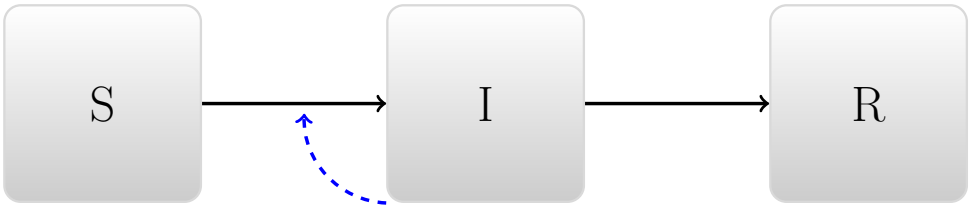
3 Single-epidemic model

- Susceptible $\rightarrow$ Infectious $\rightarrow$ Recovered
- We also use N to mean the total population

Transition rates



- What factors govern movement through the boxes?
  - People get better independently
  - People get infected by infectious people

Conceptual modeling

- Poll: What happens in the long term if we introduce an infectious individual?
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Interpreting

- Why might there not be an epidemic?
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- Why doesn't everyone get infected?
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Quantities

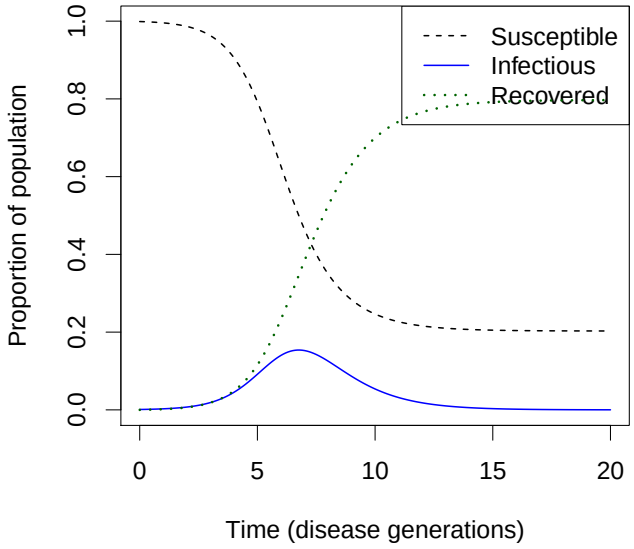
State variables

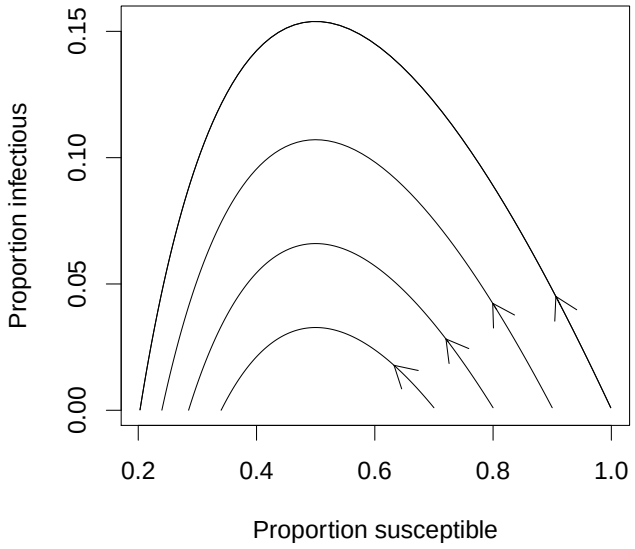
- S, I, R, N: [people] or [people/ha]

Parameters

- Susceptible people have **potentially effective** contacts at rate β (units [1/time])
  - These are contacts that would lead to infection if the person contacted is infectious
  - Total infection rate is $\beta I/N$, because I/N is the proportion of the population infectious
- Infectious people recover at *per capita* rate γ (units [1/time])
  - Total recovery rate is γI
  - Mean time infectious is $D = 1/\gamma$ (units [time])

Simulating the model





Basic reproductive number

- Poll: What *unitless* parameter can you make from the model above?
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- Poll: What happens early in the epidemic if $\mathcal{R}_0 > 1$?
  -
- What happens early in the epidemic if $\mathcal{R}_0 < 1$?
  -

Effective reproductive number

- The effective reproductive number gives the number of new infections per infectious individual in a partially susceptible population:
  - –
- Is the disease increasing or decreasing?
  - –
  - –
- Why doesn't everyone get infected?
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4 Recurrent epidemic models

- Poll: If epidemics tend to burn out, why do we often see repeated epidemics?
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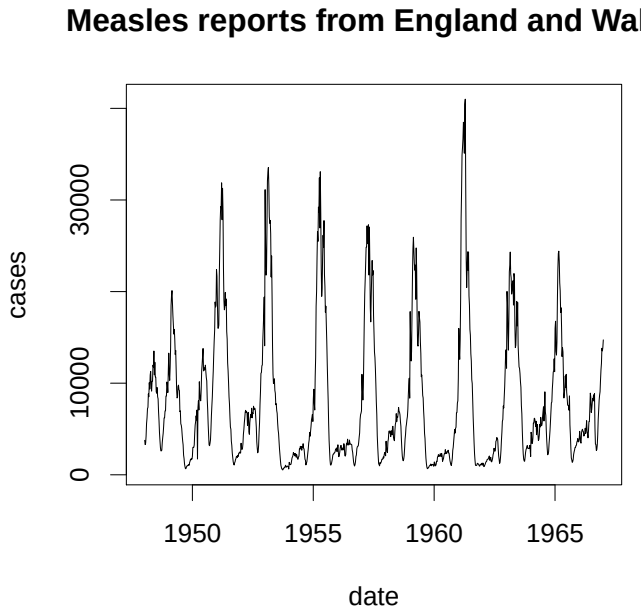
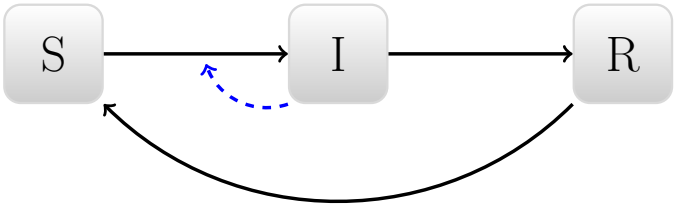


Closing the circle



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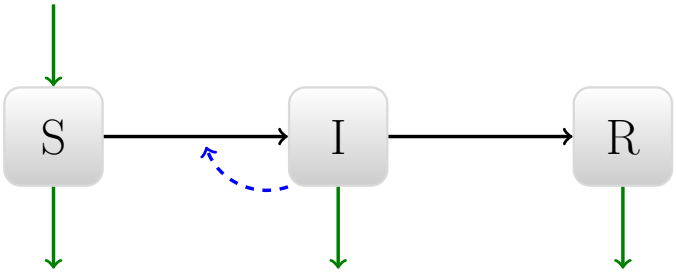
Closing the circle



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Equilibrium

- At equilibrium, we know that $\mathcal{R}_e = 1$
  - One case per case
  - Number of susceptibles at equilibrium determined by the number required to keep infection in balance
    - $S/N = 1/\mathcal{R}_0$

- What does this remind you of?
  -

- Number of infectious individuals determined by number required to keep susceptibles in balance.
- As susceptibles go up, what happens?
  - Rate of susceptible increase goes down
    - per capita, at least
  - Infections required goes down

Reciprocal control

- What happens to *equilibrium* if we protect susceptibles (move them to R class)?
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- What else could happen?
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Reciprocal control

- Poll: What happens if we remove infectious individuals at a constant rate (find them and cure them or isolate them)?
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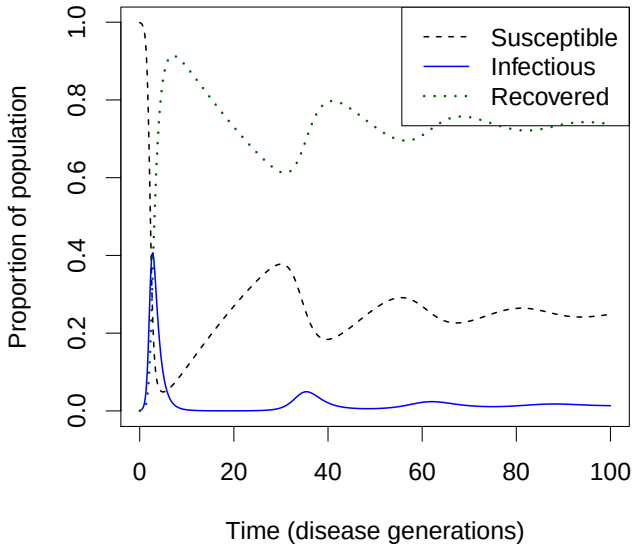

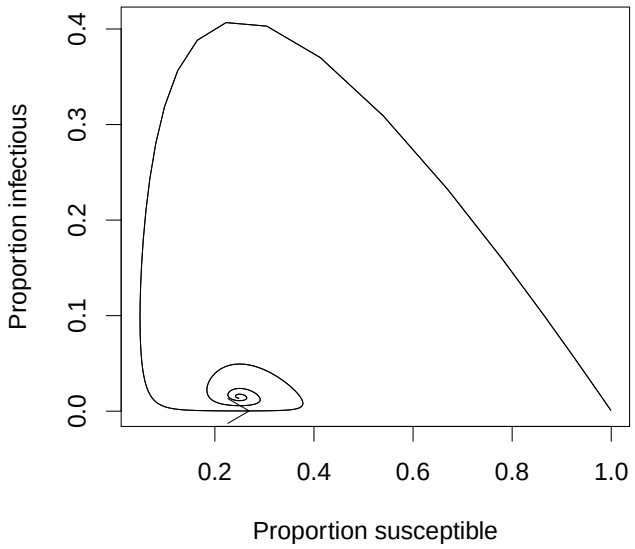

- "Closed-loop" SIR models (ie., with births or loss of immunity):
  - Tend to show damped oscillations
    - System reaches an **endemic** equilibrium – disease persists

Source of oscillations

- Similar to predator-prey systems
- What happens if we start with too many susceptibles?
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Persistent oscillations

- Poll: If oscillations tend to be damped in simple models, why do they persist in real life?
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