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
    * **Answer:** influenza virus, Ebola virus, HIV, SARS-CoV-2
  - **Bacteria** are independent, free-living cells with hundreds or thousands of chemical pathways
    * **Answer:** Tuberculosis, anthrax, pertussis
  - **Eukaryotic** pathogens are nucleated cells who are more closely related to you than they are to bacteria
    * **Answer:** Malaria, various worms

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

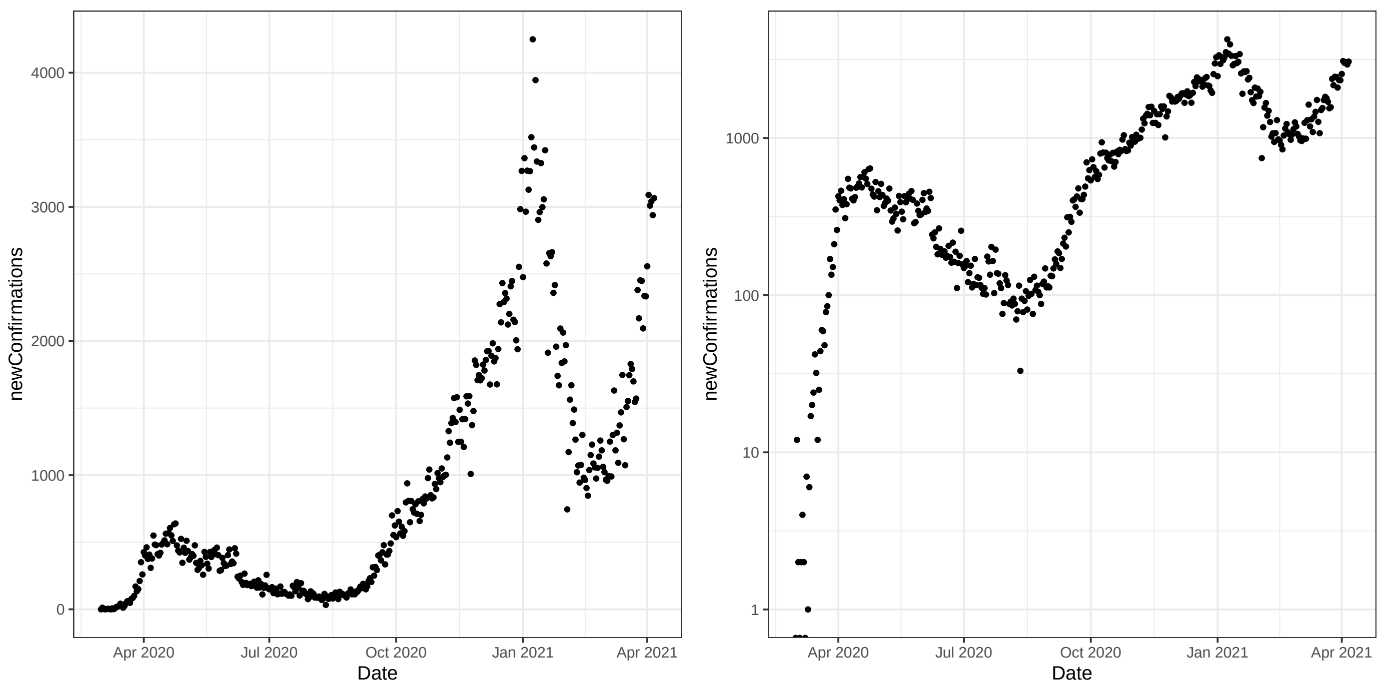
- We model microparasites by counting the number of hosts in various **states**:
  - **Susceptible** individuals can become infected
  - **Infectious** individuals are infected and can infect others
  - **Resistant** individuals are not infected and cannot become infected
- More complicated models include other states:
  - **Answer:** Not yet infectious
  - **Answer:** Severe infections
  - **Answer:** Asymptomatic but infectious

Models as tools

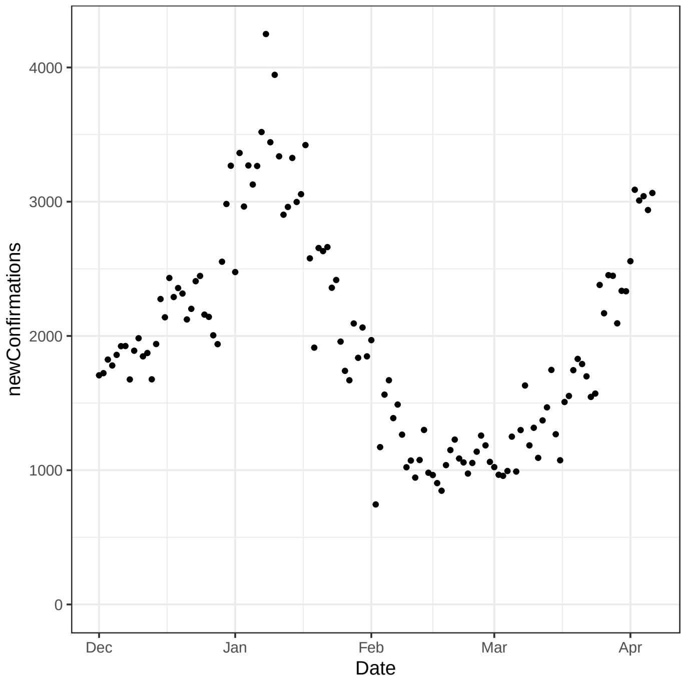
- Models are the tools that we use to connect scales:
  - individuals to populations
  - single actions to trends through time

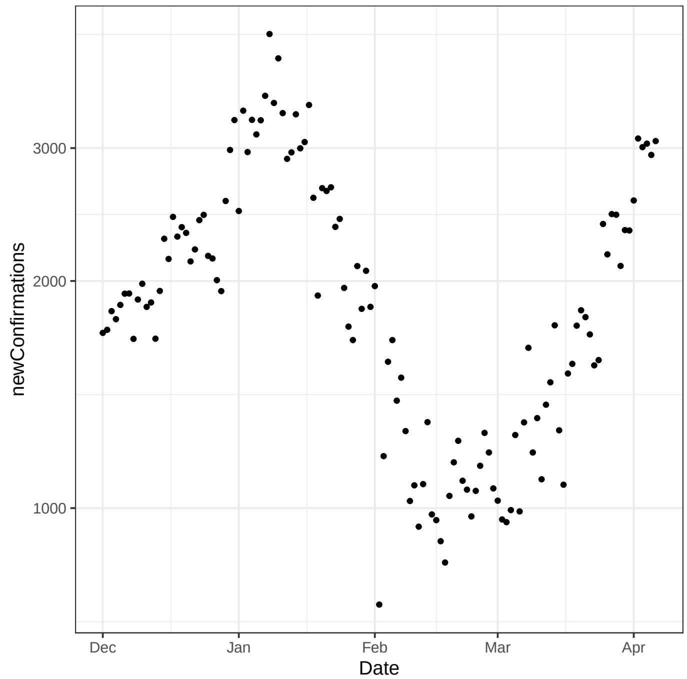
2 Rate of spread

COVID in Ontario



COVID in Ontario





Scales

- Which scale should we look at?
  - **Answer:** Log scale is better for looking at trends
  - **Answer:** Linear scale is better for looking at impacts

Population biology

- What quantities do we want to look at?
  - **Answer:** Speed of exponential growth r
  - **Answer:** Finite rate of increase λ
    - **Answer:** Skipped this year
  - **Answer:** Lifetime reproduction

Instantaneous rate of growth r

- What are the components?
  - **Answer:** Birth rate
    - **Answer:** Instantaneous rate of a case producing new cases
    - **Answer:** [case/(case $\cdot$ time]
  - **Answer:** Death rate
    - **Answer:** Virus-centered!
    - **Answer:** Rate of death, recovery, or effective quarantine

- How do you think we estimate?
  - **<u>Answer</u>:** We estimate r from the population-level increase in disease
    - **<u>Answer</u>:** Then using that to estimate b
  - **<u>Answer</u>:** Models go both directions!
    - Individuals $\leftrightarrow$ Populations

Reproductive number $\mathcal{R}$

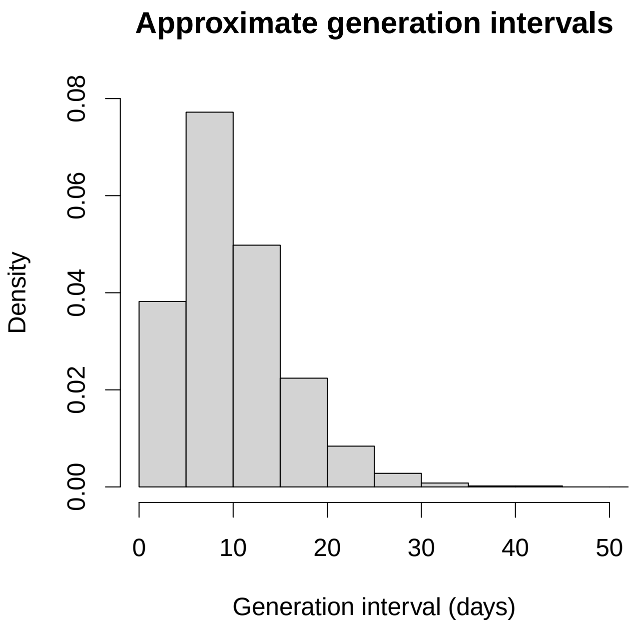
- What is it?
  - **<u>Answer</u>:** Expected number of new cases per case over the lifetime of a case
- Why do we want this?
  - **<u>Answer</u>:** An important measure of how hard the epidemic will be to stop
- How do we calculate it?
  - **<u>Answer</u>:** $\mathcal{R} = b/d$; if we can estimate those

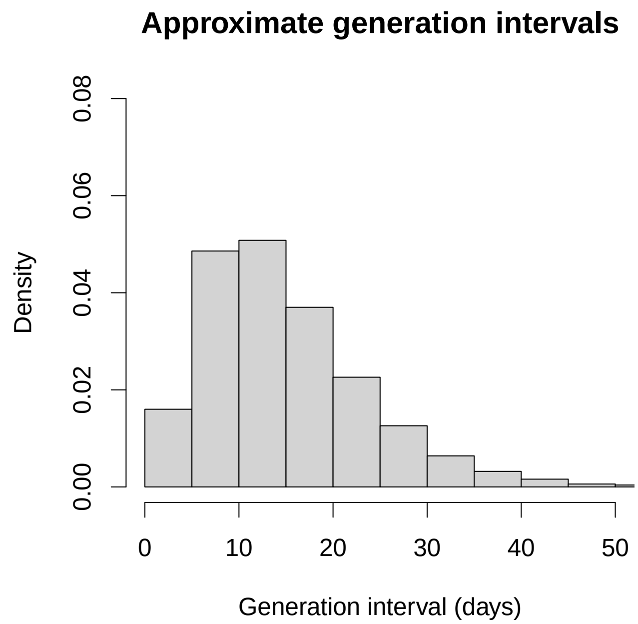
Example

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

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



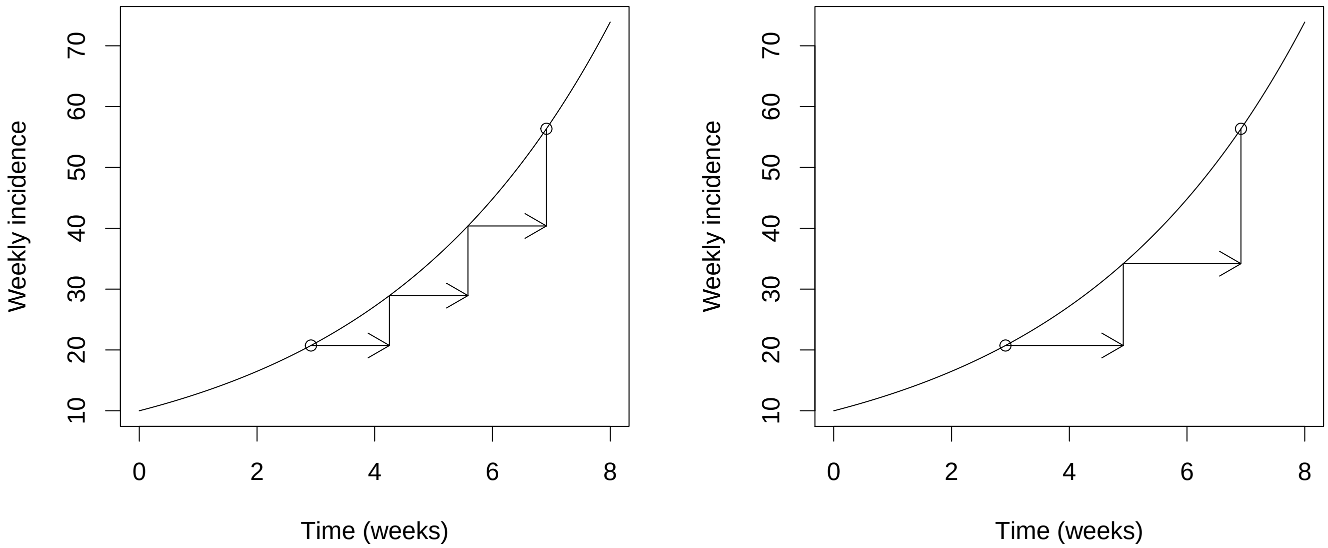


Speed and risk

- 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?
  - **Answer:** The faster the generations (small $\hat{G}$), the faster the exponential growth (large r)
- If we know r, what does the generation time tell us about $\mathcal{R}$?
  - **Answer:** The faster the generations (small $\hat{G}$), the the *smaller* the strength of the epidemic (small reproductive number $\mathcal{R}$)
- $\mathcal{R} = \exp(r\hat{G})$

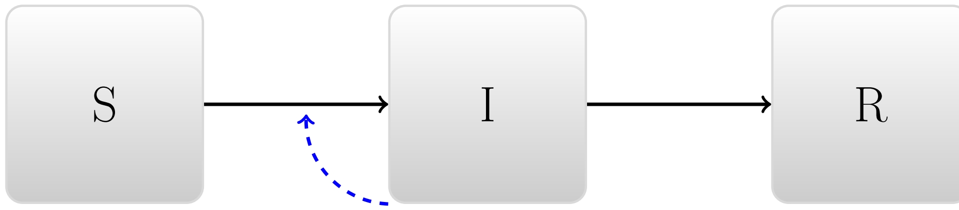


- $\mathcal{R} = \exp(r\hat{G})$
- An intuitive view:
  - Epidemic speed = Generation strength × Generation speed
  - *<u>Comment</u>:* Mathematically: $r = \log(\mathcal{R}) * (1/\hat{G})$
- If we know generation speed, then a faster epidemic speed means:
  - **<u>Answer</u>:** More strength required (greater $\mathcal{R}$)
- If we know epidemic speed, a faster generation speed means
  - **<u>Answer</u>:** Less strength required (smaller $\mathcal{R}$)

3 Single-epidemic model

- Susceptible → Infectious → 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?
  - **Answer:** The *may be* an **epidemic** – an outbreak of disease
  - **Answer:** Disease burns out
  - **Answer:** Everyone winds up either recovered …
    - **Answer:** or susceptible!

Interpreting

- Why might there not be an epidemic?
  - **Answer:** If the disease can't spread well enough in the population
    - **Answer:** Could depend on season, or immunity …
  - **Answer:** Demographic stochasticity: if we only start with one individual, we expect an element of chance
- Why doesn't everyone get infected?
  - **Answer postponed:**

Quantities

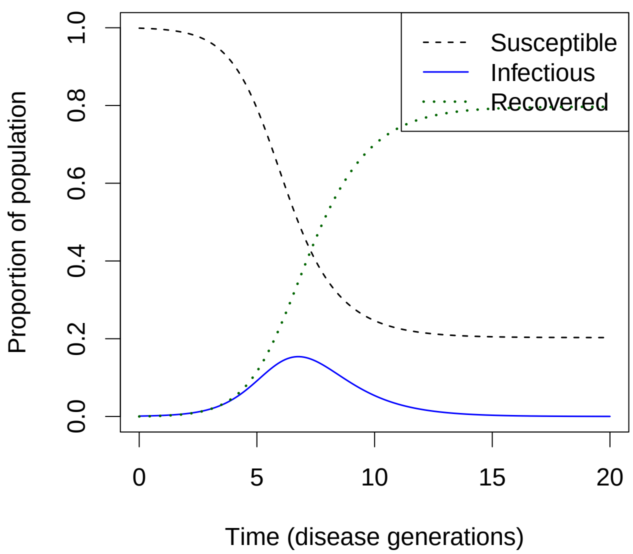
State variables

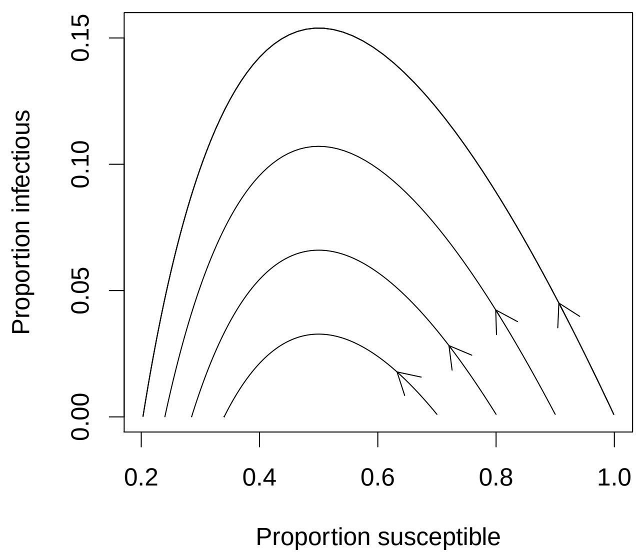
- S, I, R, N: [people] or [people/ha]

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?
  - **Answer:** $\mathcal{R}_0 = \beta D = \beta/\gamma$ is the **basic reproductive number**
  - **Answer:** The *potential* number of infections caused by an average infectious individual
    - **Answer:** That is: the number they would cause on average if everyone else were susceptible
  - **Answer:** The product of the rate β (units [1/t]) and the duration D ([t])

- Poll: What happens early in the epidemic if $\mathcal{R}_0 > 1$?
  - **Answer:** Number of infected individuals grows exponentially
- What happens early in the epidemic if $\mathcal{R}_0 < 1$?
  - **Answer:** Number of infected individuals does not grow (disease cannot invade)

Effective reproductive number

- The effective reproductive number gives the number of new infections per infectious individual in a partially susceptible population:
  - **Answer:** $\mathcal{R}_e = \mathcal{R}_0 S/N$
- Is the disease increasing or decreasing?
  - **Answer:** It will increase when $\mathcal{R}_e > 1$ (more than one case per case)
  - **Answer:** This happens when $S/N > 1/\mathcal{R}_0$
- Why doesn't everyone get infected?
  - **Answer:** When susceptibles are low enough $\mathcal{R}_e < 1$
  - **Answer:** When $\mathcal{R}_e < 1$, the disease dies out on its own (less than one case per case)