Day 2 Lecture 2: Understanding the phases of an epidemic





Short course on modelling infectious disease dynamics in R

Ankara, Türkiye, September 2025

Dr Juan F Vesga

Aims of the session

- Understand why epidemics rise
- Understand why epidemics peak
- Understand why epidemics fade out
- Examine the concept of heard immunity
- Understand R₀ and R_{eff}

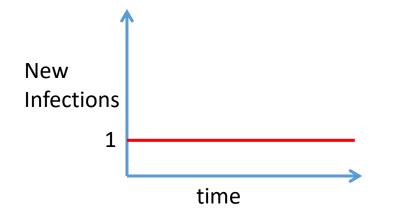
What it takes for an epidemic to rise?

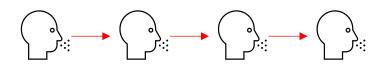
- A rate of infection -> transmission!
- A window of opportunity -> infectious period!

In general an epidemic starts if a pathogen is able to transmit quickly enough in that window of opportunity

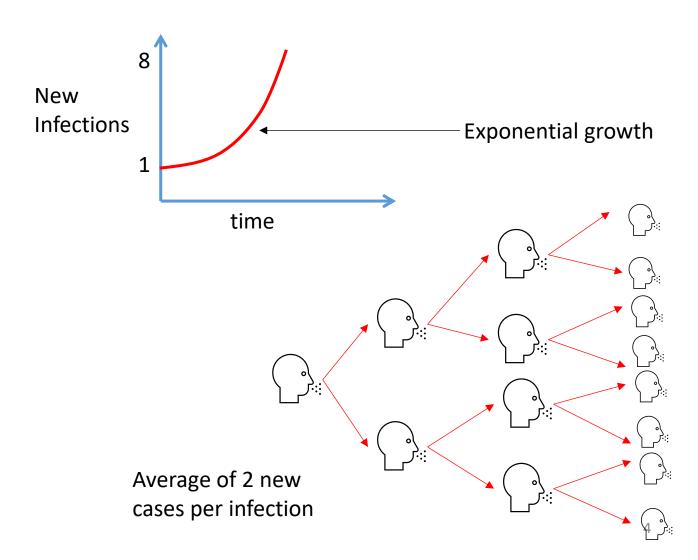
Other factors play a role too, like virulence behaviours etc.

What it takes for an epidemic to rise?





Average of 1 new case per infection

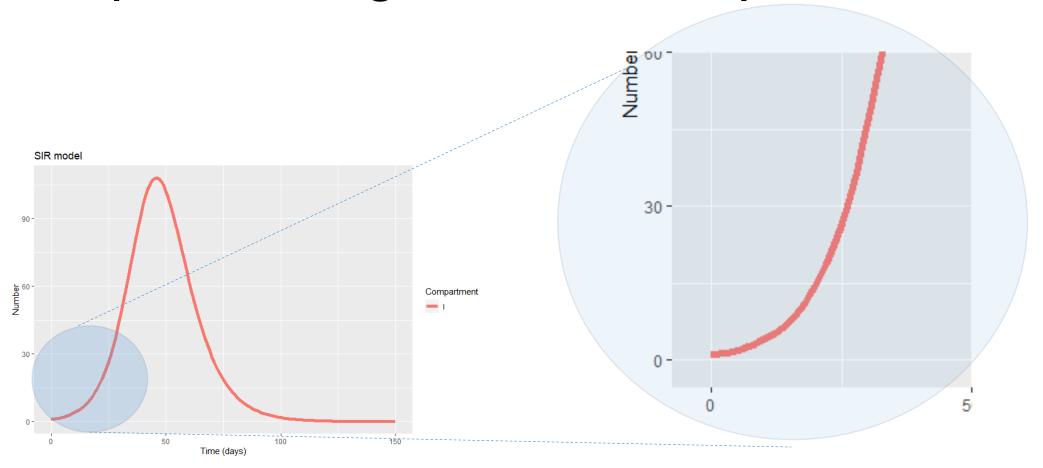


R₀ (basic reproduction number)

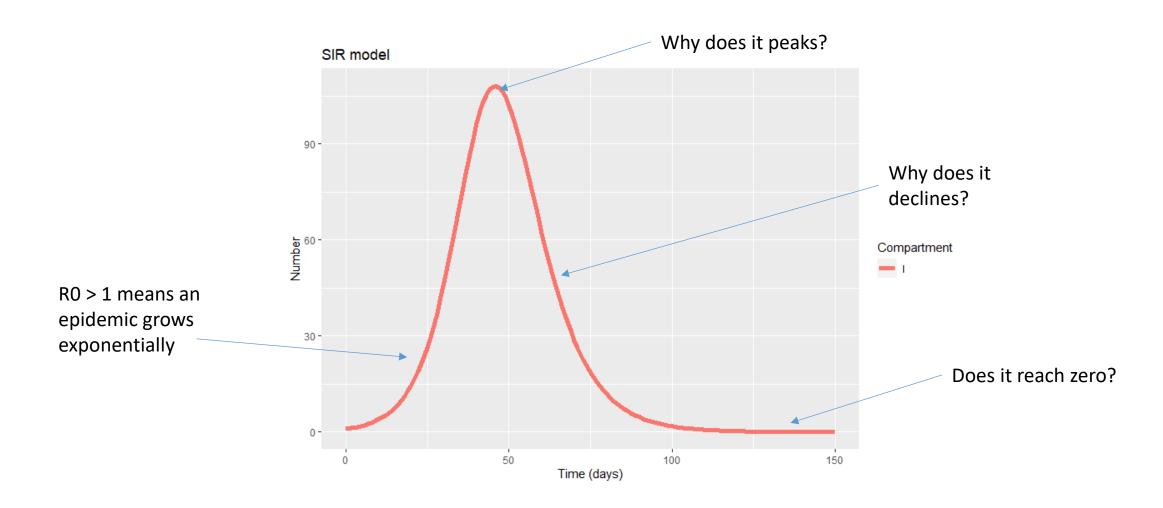
Average number of secondary infections caused by a single infectious case in a fully susceptible population

- If $R_0 > 1$, an epidemic start
- If $R_0 < 1$, an epidemic fails

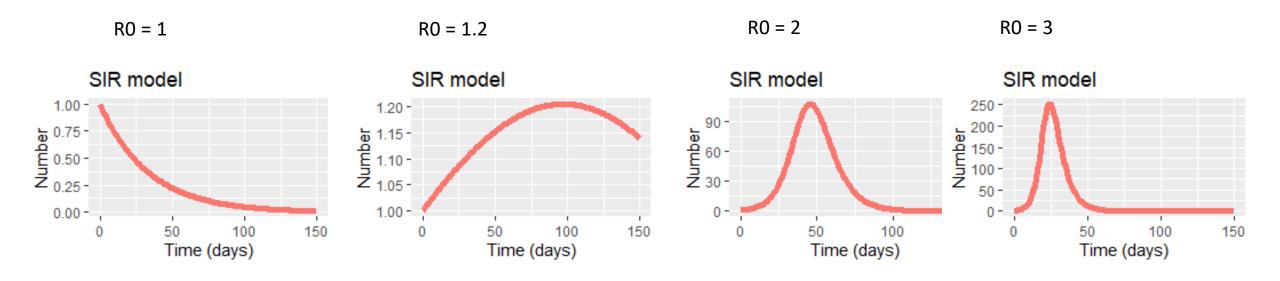
Exponential growth of an epidemic



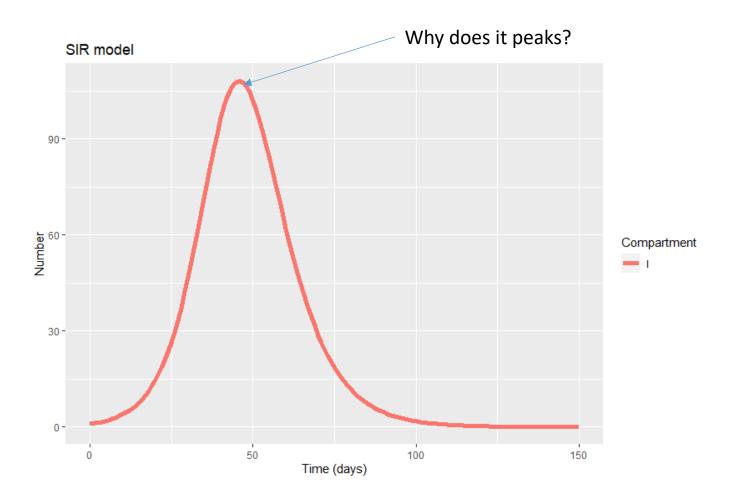
What are the phases?



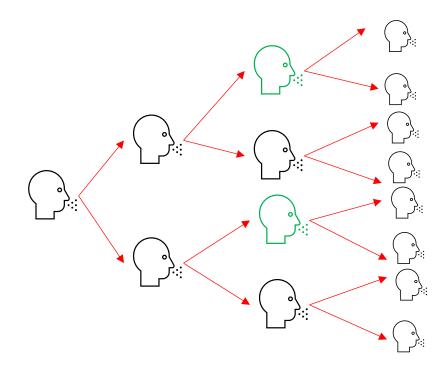
Epidemic growth and R₀



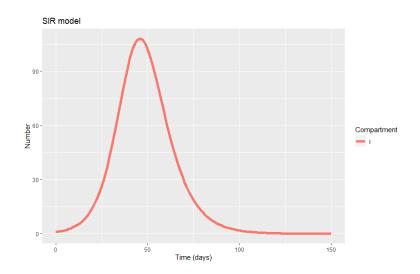
Behaviour of the number infected under different assumptions of R0 for an SIR model with a close population

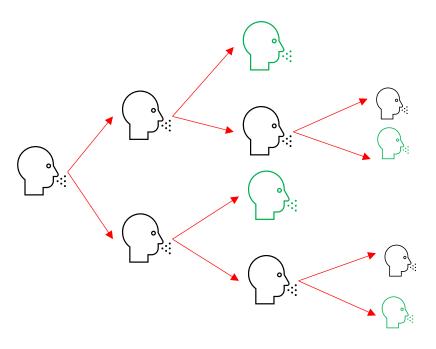


- We know that R0 plays a role in the epidemic rise.
- A second actor is immunity

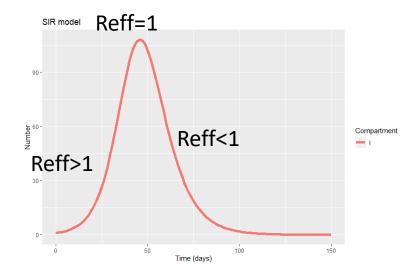


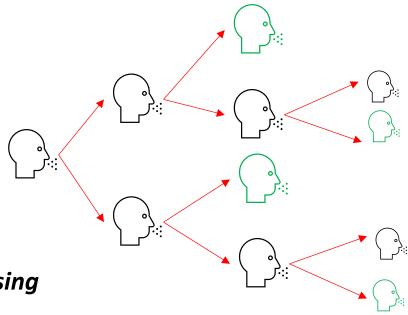
- We know that R0 plays a role in the epidemic rise.
- A second actor is immunity
- At the peak, the fraction susceptible in the population is insufficient to satisfy R0 for each infected case





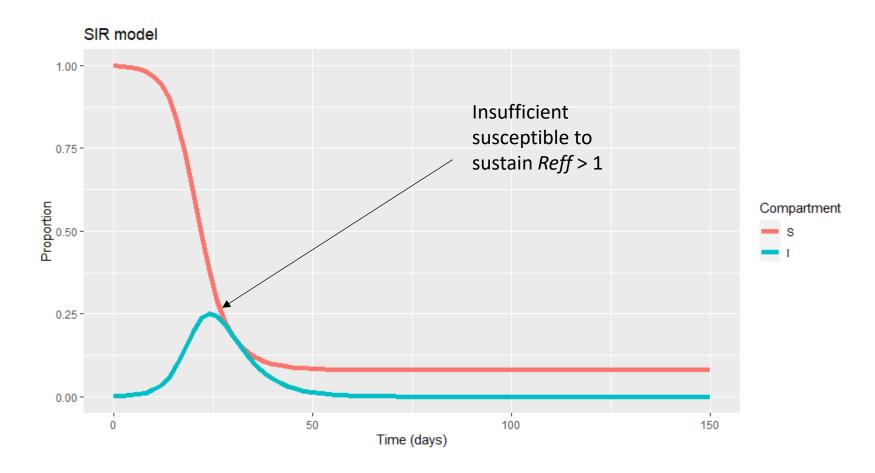
- R0 is defined for a fully susceptible population
- When immunity plays a role, we can define R_{eff}
- A depletion of Susceptible drives the epidemic down





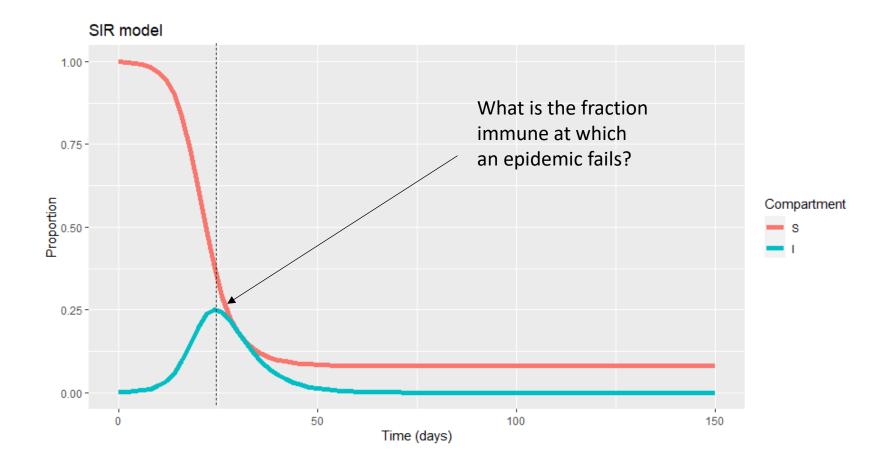
 $R_{\it eff}$ is the average number of secondary cases arising from an infected case at a given point in time

- Does everybody gets infected?
- No!

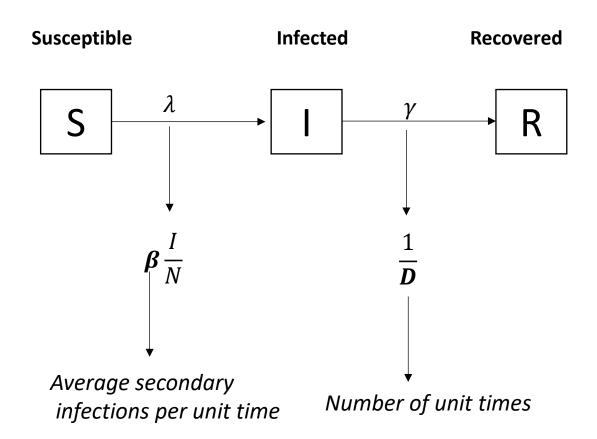


Herd immunity

- Reff =1
- Can be raised artificially with vaccination



How can this be explained in terms of SIR



$$R_0 = \beta D = \beta \frac{1}{\gamma} = \frac{\beta}{\gamma}$$

Average number of secondary infections in the total duration of the infectious period

And R_{eff} ?

 If we said that the depletion of the susceptible compartment is what drives down an epidemic. Then R_{eff} is proportional to the fraction that remains susceptible at each time t

$$R_{eff} = R_0 \frac{S(t)}{N}$$

- Herd immunity
- Fraction immune necessary to bring epidemic down

$$R_{eff} = 1$$

$$R_0 \frac{S(t)}{N} = 1$$

$$\underbrace{\frac{S(t)}{N}} = \frac{1}{R_0}$$

Fraction susceptible below which epidemic declines

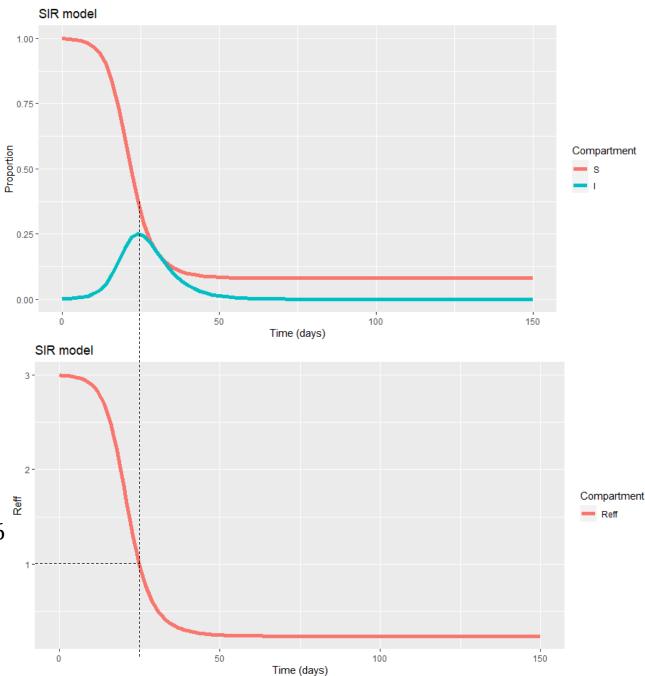
Example for R0=3

$$\frac{S(t)}{N} = \frac{1}{R_0}$$

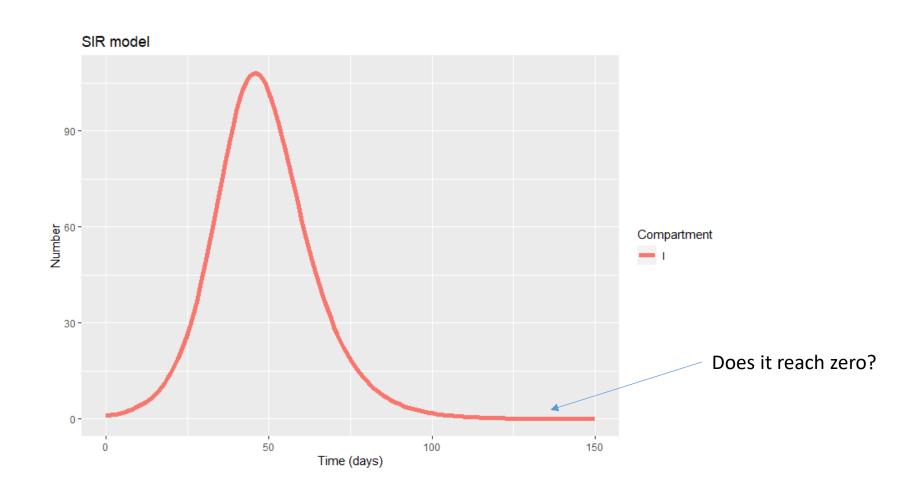
$$\frac{S(t)}{N} = \frac{1}{3} = 0.33$$

$$HIT = 1 - 0.33 = 0.66$$

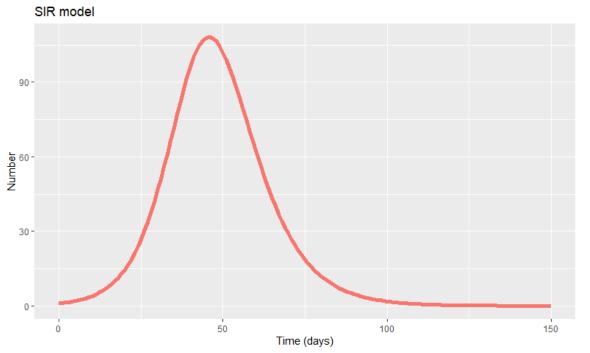
Herd immunity threshold



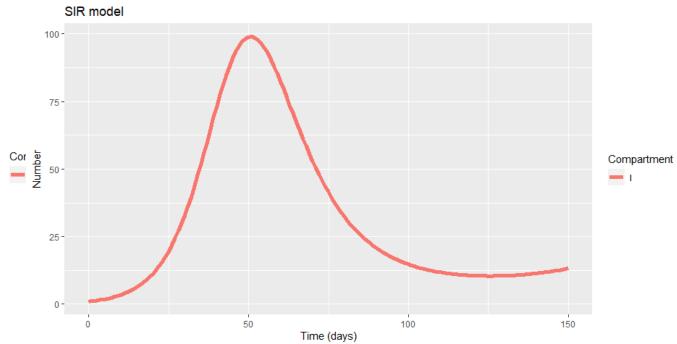
What if we have susceptible renewal?



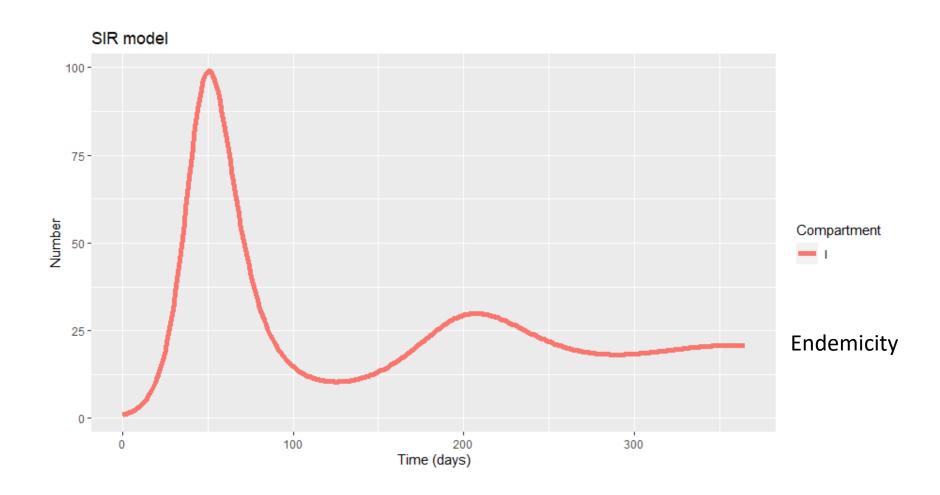
Close population



Susceptible renewal (birth rate)

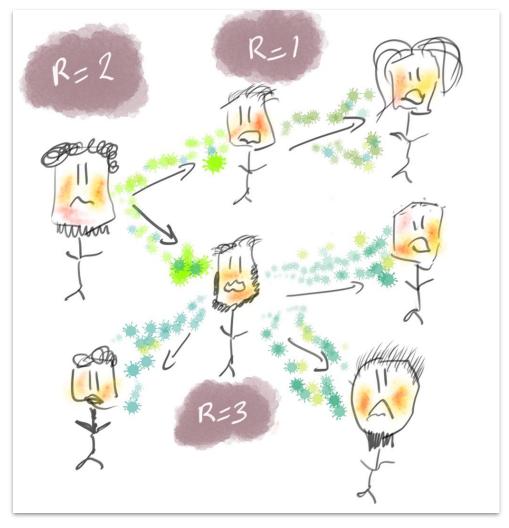


Long term dynamics



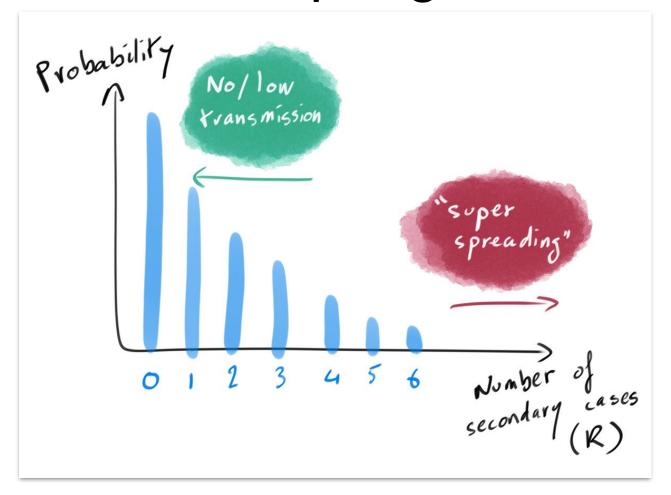
Some added complexity when understanding reproduction numbers

The reproduction number(s)



- Generalities
 - number of secondary cases by infected individual
 - R > 1 = exponential growth
 - R < 1 = exponential decline
- Basic reproduction number (R_0) : R in a fully susceptible population
- Effective / case reproduction number (R_c) : actually realised R
- Instantaneous reproduction number $(R_i(t))$: average R_c of infectors with symptoms at time t if transmissibility remains the same

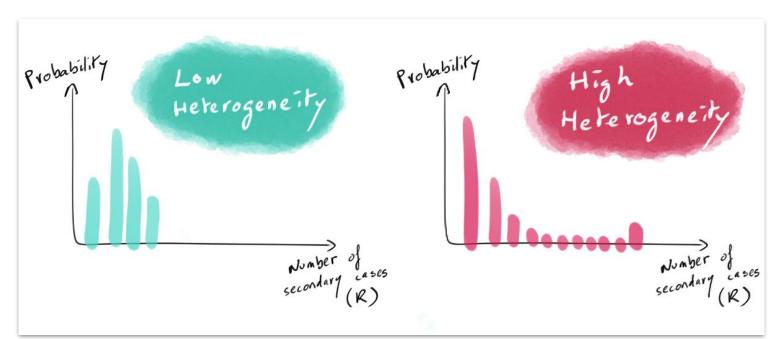
The offspring distribution



- Distribution of the number of secondary cases per infected case
- Its average is R
- Further indications of how transmission occurs:
 - Do all individuals transmit?
 - Are there super-spreading events?

With thanks to Thibaut Jombart

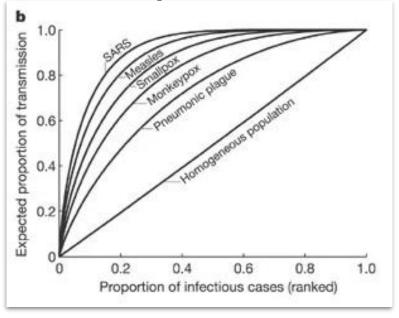
Heterogeneity in R

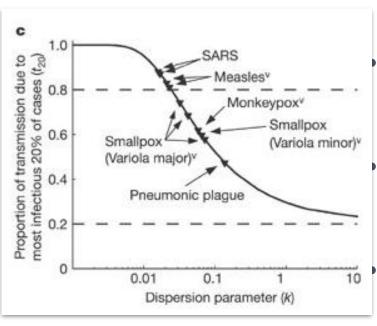


- Heterogeneity in R reflects differences in infectiousness between cases
- Higher heterogeneity
 - A smaller fraction of the population drives transmission
 - Presence of super-spreading events
 - Skewed offspring distribution

With thanks to Thibaut Jombart

Impact of heterogeneity in R for epidemic control





Source: Lloyd-Smith et al. (2005)

Higher heterogeneity in R

Stochastic extinction more likely

 Rarer, more 'explosive' outbreaks

Individual-based interventions better suited than population-wide

Targeted interventions require investigation of predictors of infectiousness

May need contact tracing, genetic data, etc. to reconstruct transmission chains

What we should know by now

- Why epidemics rise
- What is R0 and Reff and how it relates to the SIR model
- What is herd immunity
- How we calculate HIT if we know R0
- How introducing demographics can change the picture of the long term dynamics
- In simple models we assess the population infectiousness, but in a real outbreak, estimation of R becomes more complex