Basics of SIR Models

Some Terms

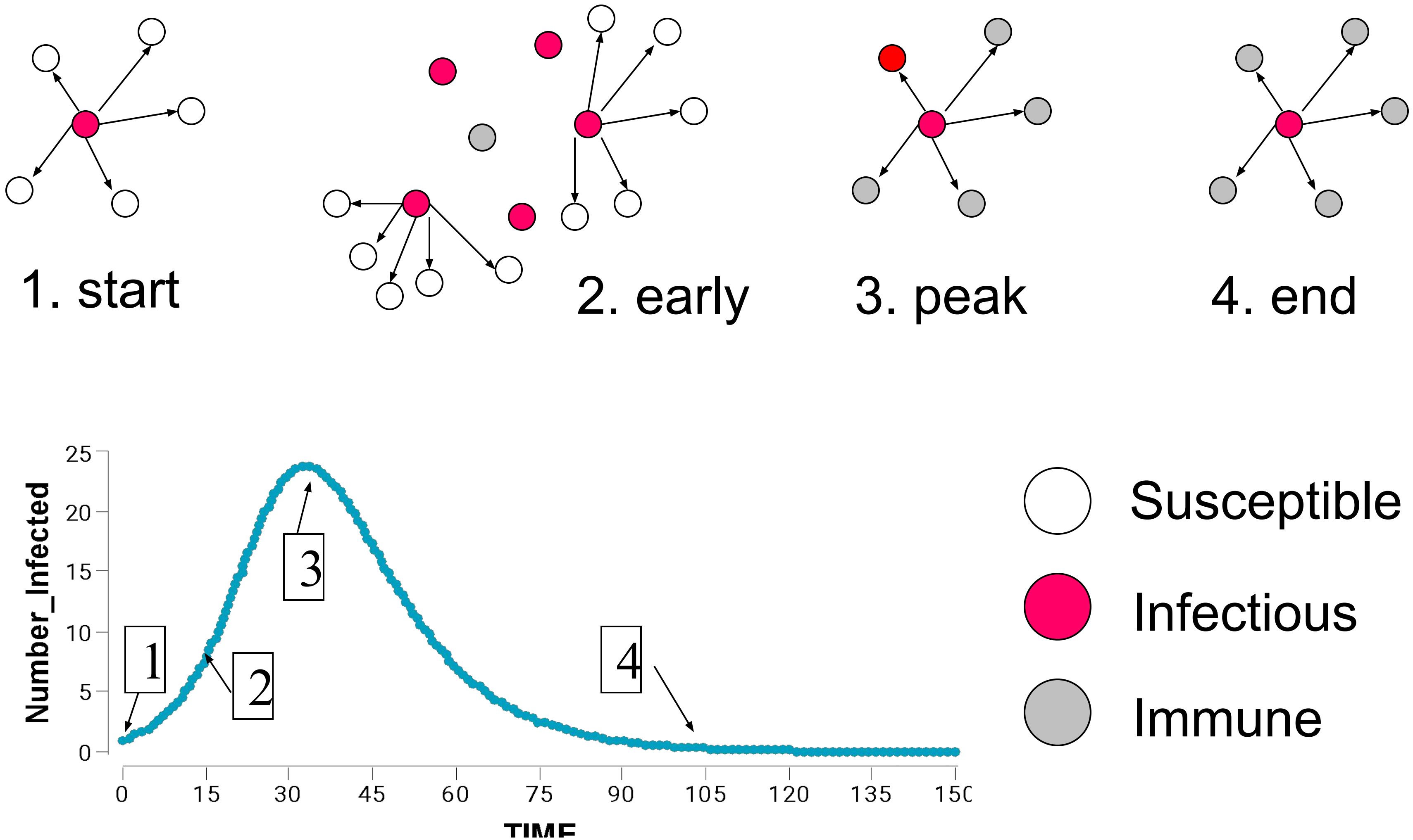
Closed v Open populations

- Closed populations have no additions due to births or immigration and no losses due to death or emigration – all dynamics due ***only*** to infection
  - Unrealistic, but simple and a good place to start
- Open populations can have births, immigration, deaths, emigration
  - Either explicitly or implicitly

Equilibrium and Transient

- At equilibrium, the values of all states are constant – individuals may still get sick, recover, etc, but all changes balance each other out
- Dynamics are transient if the states are continuing to change … more nuance later

Phases of an Epidemic

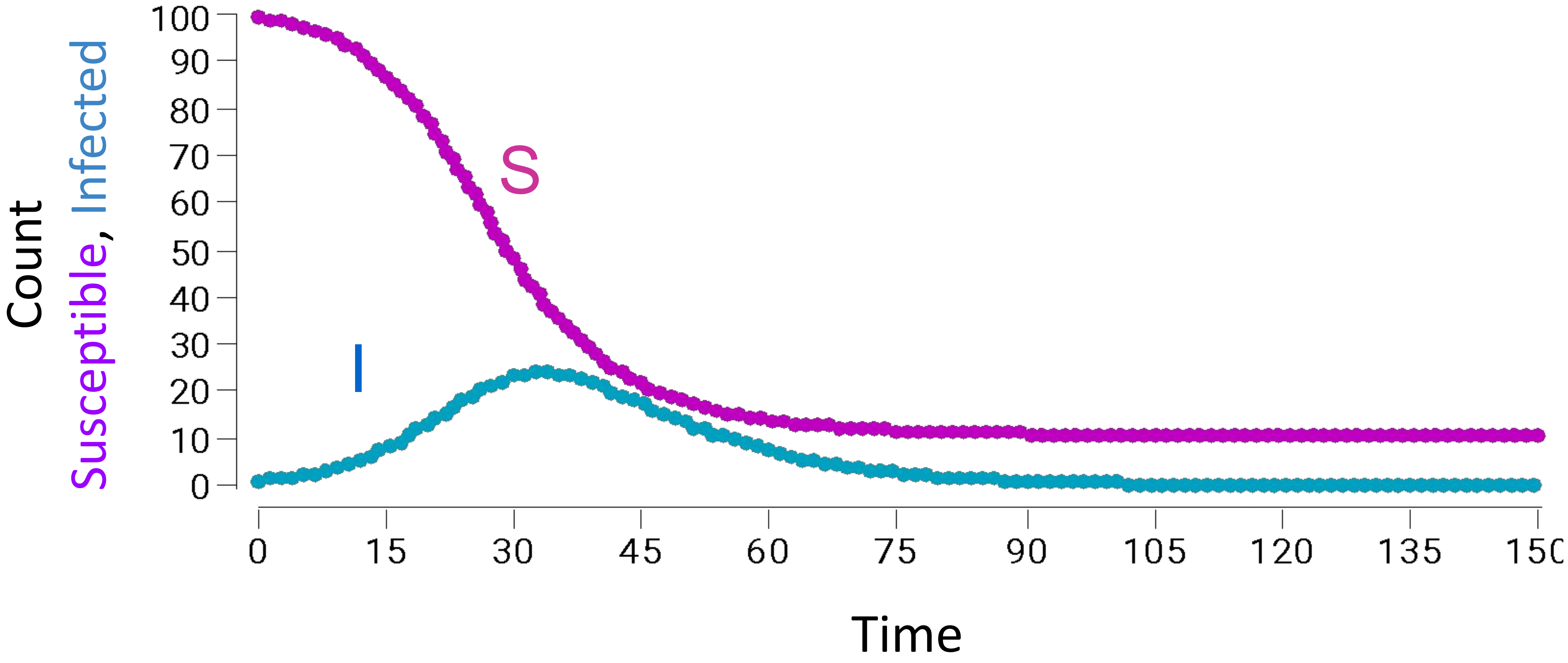


The SIR Model

The States

S, **I**, and **R** reflect the number of individuals that are currently **susceptible**, **infected** (and infectious), and **recovered,** respectively

Idealized Epidemic in a Closed Community



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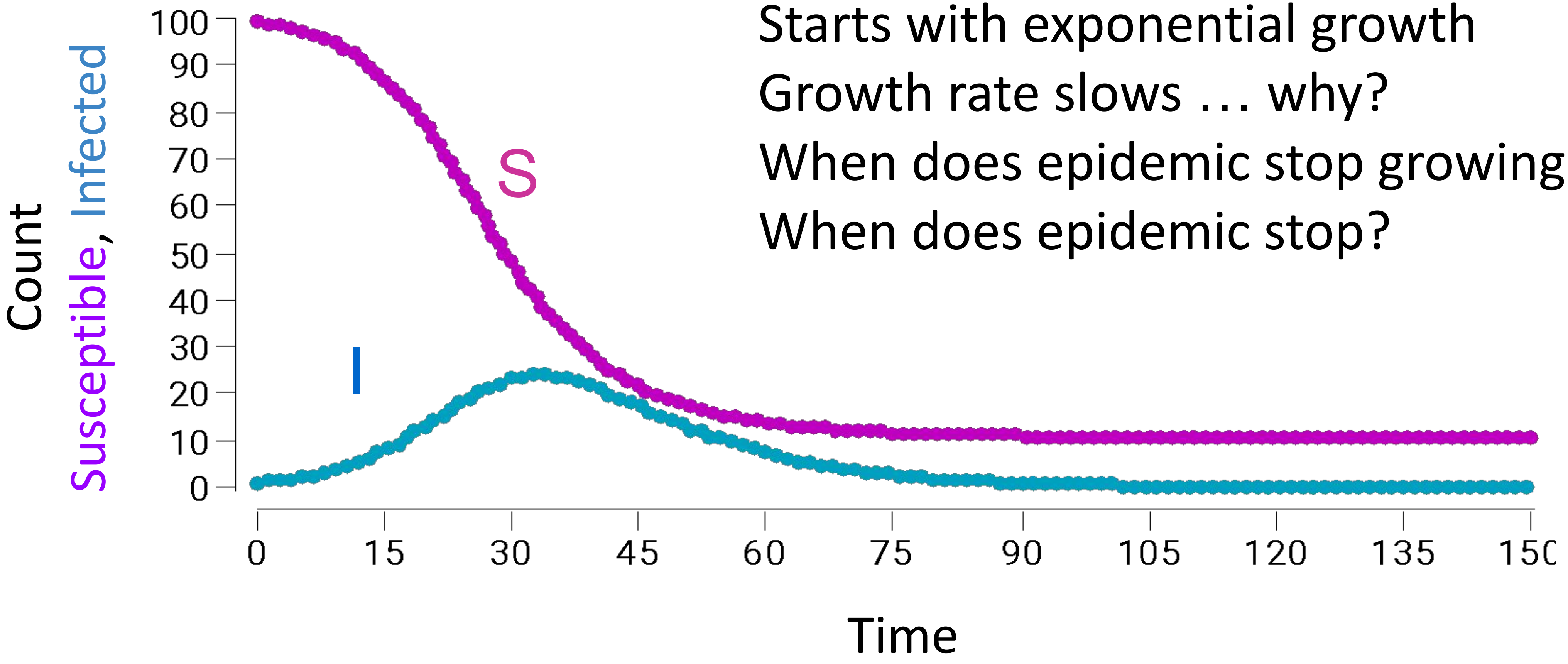


Starts with exponential growth
Growth rate slows … why?
When does epidemic stop growing?
When does epidemic stop?

The SIR Model

$$\frac{dS}{dt} =$$
$$\frac{dI}{dt} =$$
$$\frac{dR}{dt} =$$

Since we're talking about how the epidemic grows, or how the **states change over time**, we write the SIR model in terms of the derivative, with respect to time, of each of the states.

The SIR Model

$$\frac{dS}{dt} = -\beta SI$$

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

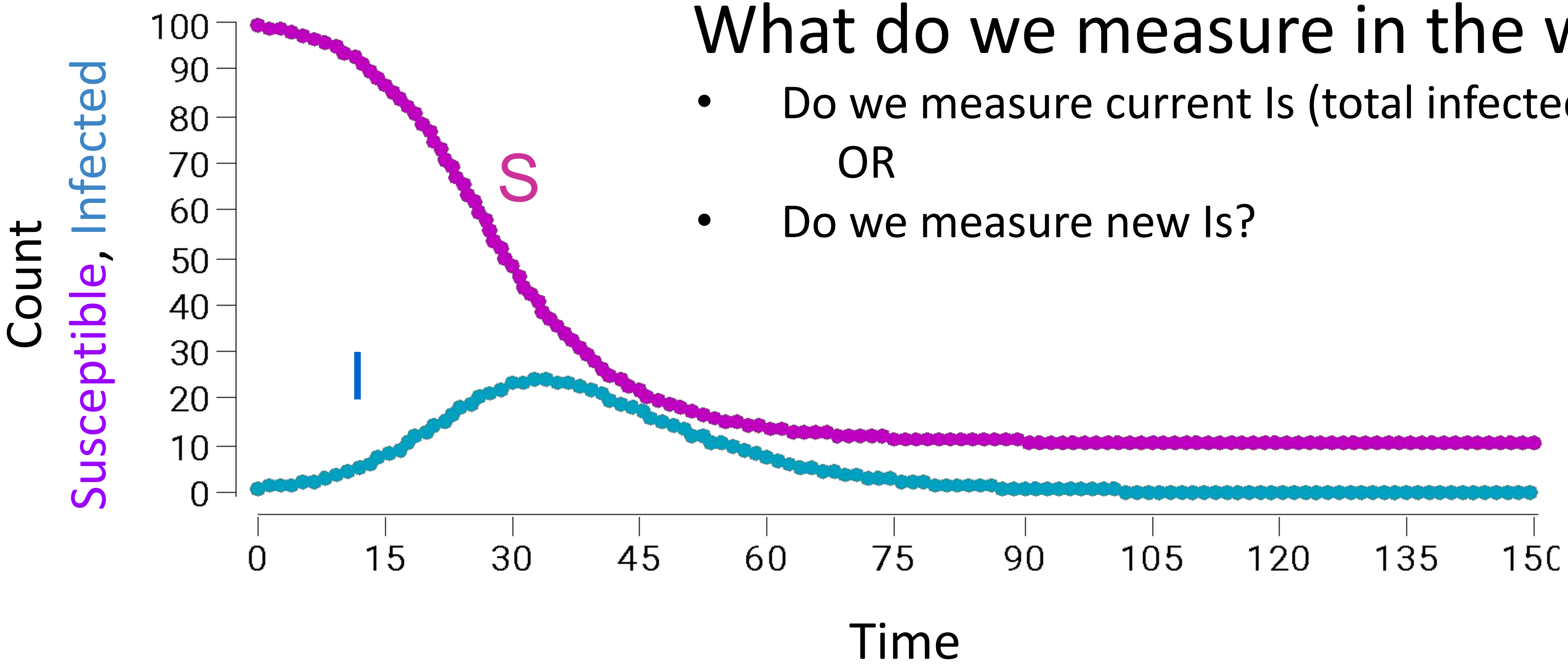
$$\frac{dR}{dt} = \gamma I$$

The States

S, **I**, and **R** reflect the number of individuals that are currently **susceptible**, **infected** (and infectious), and **recovered,** respectively

Because the states are linked (an S becomes and I, and an I becomes an R) the states show up in each other's equations

Idealized Epidemic in a Closed Community

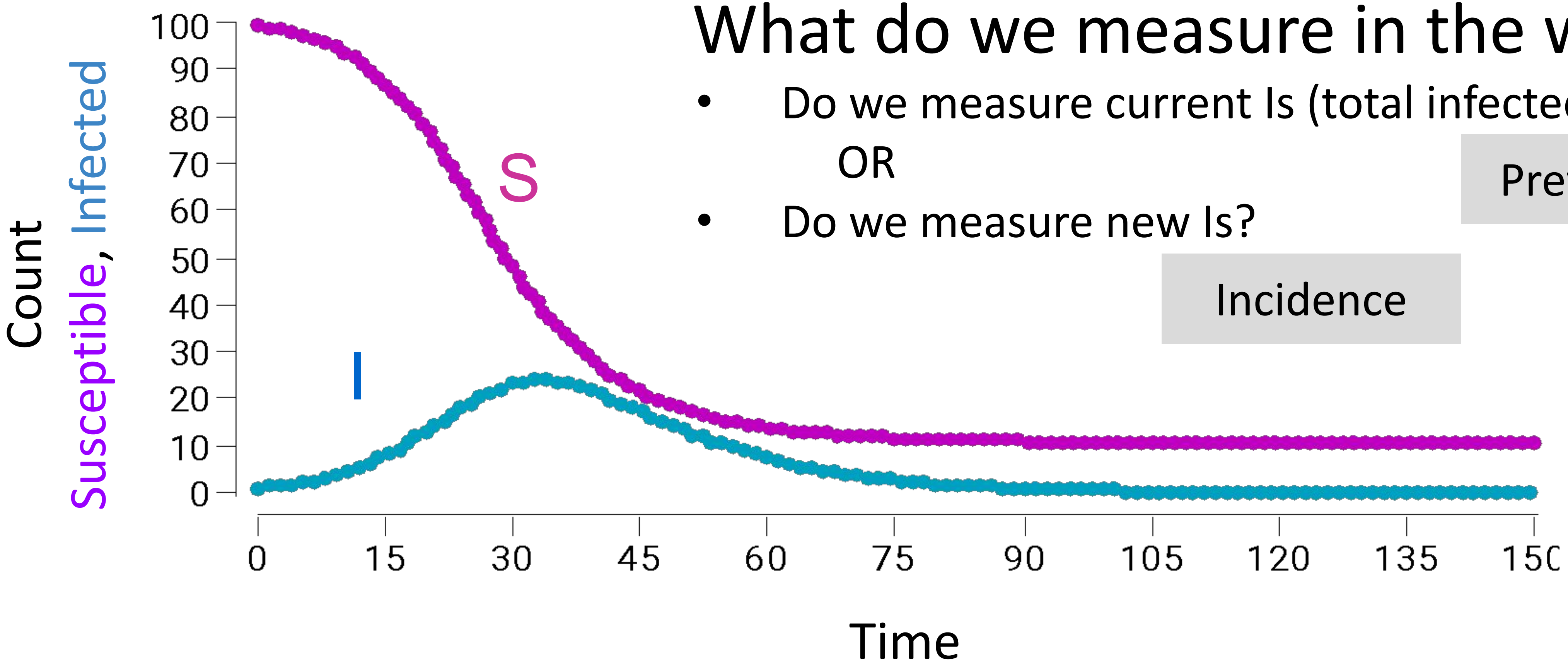


What do we measure in the world?

- Do we measure current Is (total infected),

  OR
- Do we measure new Is?

Idealized Epidemic in a Closed Community



What do we measure in the world?

- Do we measure current Is (total infected),

  OR
- Do we measure new Is?

Prevalence

Incidence

The SIR Model

$$\frac{dS}{dt} = -\beta S I$$

$$\frac{dI}{dt} = \beta S I - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

What do we measure in the world?

- Do we measure current Is (total infected),

  OR
- Do we measure new Is?

How could we measure current Is?

How could we measure new Is?

The SIR Model

$$\frac{dS}{dt} = -\beta SI$$

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

$$\frac{dR}{dt} = \gamma I$$

Contact Process

- This quantifies the rate at which susceptibles and infecteds interact
  - Increases with number (or proportion) of each
  - Creates non-linearity

The SIR Model

$$\frac{dS}{dt} = -\beta \boxed{SI}$$

$$\frac{dI}{dt} = \beta \boxed{SI} - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

Contact Process

- There are lots of ways to adjust this … the most (in)famous of which is: $S\frac{I}{N}$

The SIR Model

$$\frac{dS}{dt} = -\beta SI$$

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

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Contact Process

- There are lots of ways to adjust this … the most (in)famous of which is: $S\frac{I}{N}$

Begon: A clarification of transmission terms in host-microparasite models
https://pmc.ncbi.nlm.nih.gov/articles/PMC2869860/

The SIR Model

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

Contact Process

- What other ways might contacts change with the amount of infection?

Hint: think about behavior over the last 5 years

The SIR Model

$$\frac{dS}{dt} = -\boxed{\beta} SI$$

$$\frac{dI}{dt} = \boxed{\beta} SI - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

Transmission Parameter

- *SI* defines the shape of contacts. β turns that into infectious contacts:
  - Rate of infectious contacts (not all contacts are infectious)
  - Probability of infection *given* contact

The SIR Model

$$\frac{dS}{dt} = -\beta SI$$

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

$$\frac{dR}{dt} = \gamma I$$

Recovery Rate

- This is the rate, number per time, of recovery (or removal)
- If rate is high, many events happen per unit time and time between events is small

The SIR Model

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

Recovery Rate

- This is the rate, number per time, of recovery (or removal)
- So large ***γ*** means that the average duration of infection is short

The SIR Model

$$\frac{dS}{dt} = -\beta SI$$

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

$$\frac{dR}{dt} = \gamma I$$

Recovery Rate

- This is the rate, number per time, of recovery (or removal)
- Mean duration of infection, L, is $\frac{1}{\gamma}$

if the distribution of infectious periods is exponential

How realistic is this?
Why do we do this?

Basic Epidemic Theory

$$S + I \xrightarrow{\beta} I + I \qquad \text{Infection}$$

$$I \xrightarrow{\gamma} R \qquad \text{Recovery}$$

$$\frac{dS}{dt} = -\beta SI$$

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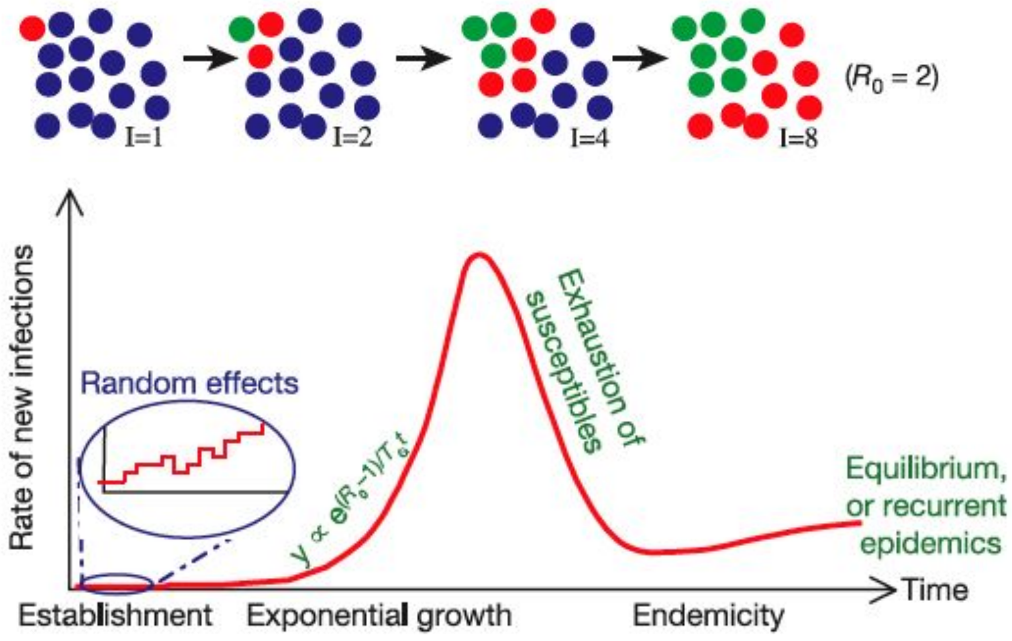

figures from Ferguson et al. *Nature* 2003

At the start, when there are few infections, an epidemic grows (almost) exponentially

Basic Epidemic Theory

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$$I \xrightarrow{\gamma} R \quad \text{Recovery}$$

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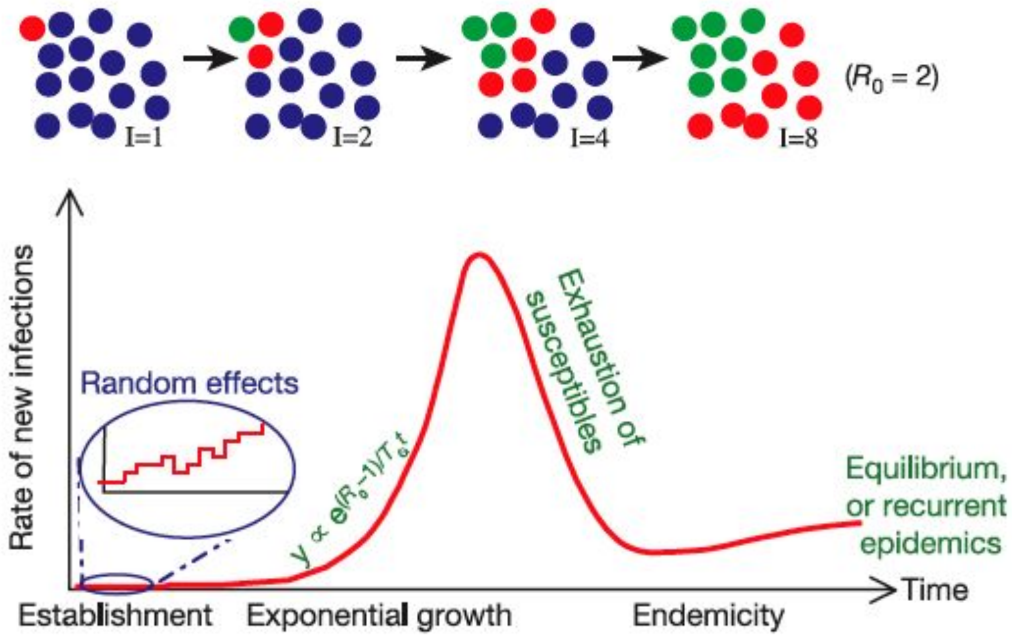

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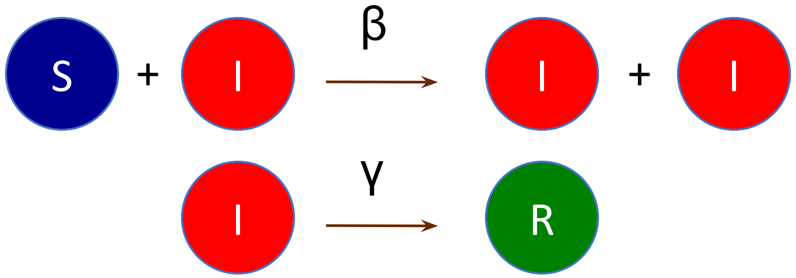
At the start, when there are few infections, an epidemic grows (almost) exponentially

We'll use this property later to estimate the transmission rate

Basic Epidemic Theory



Infection

Recovery

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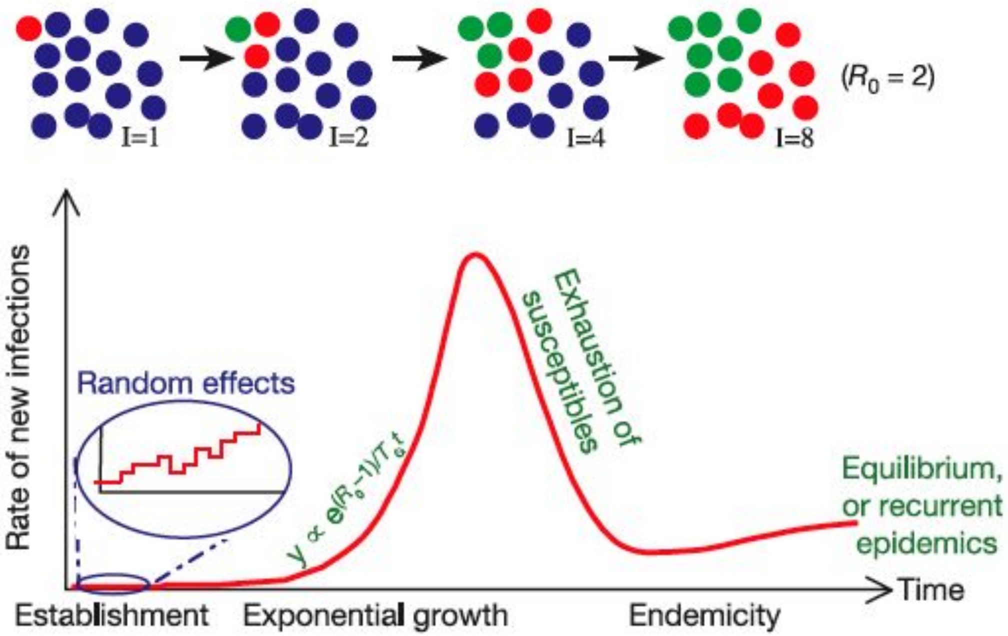

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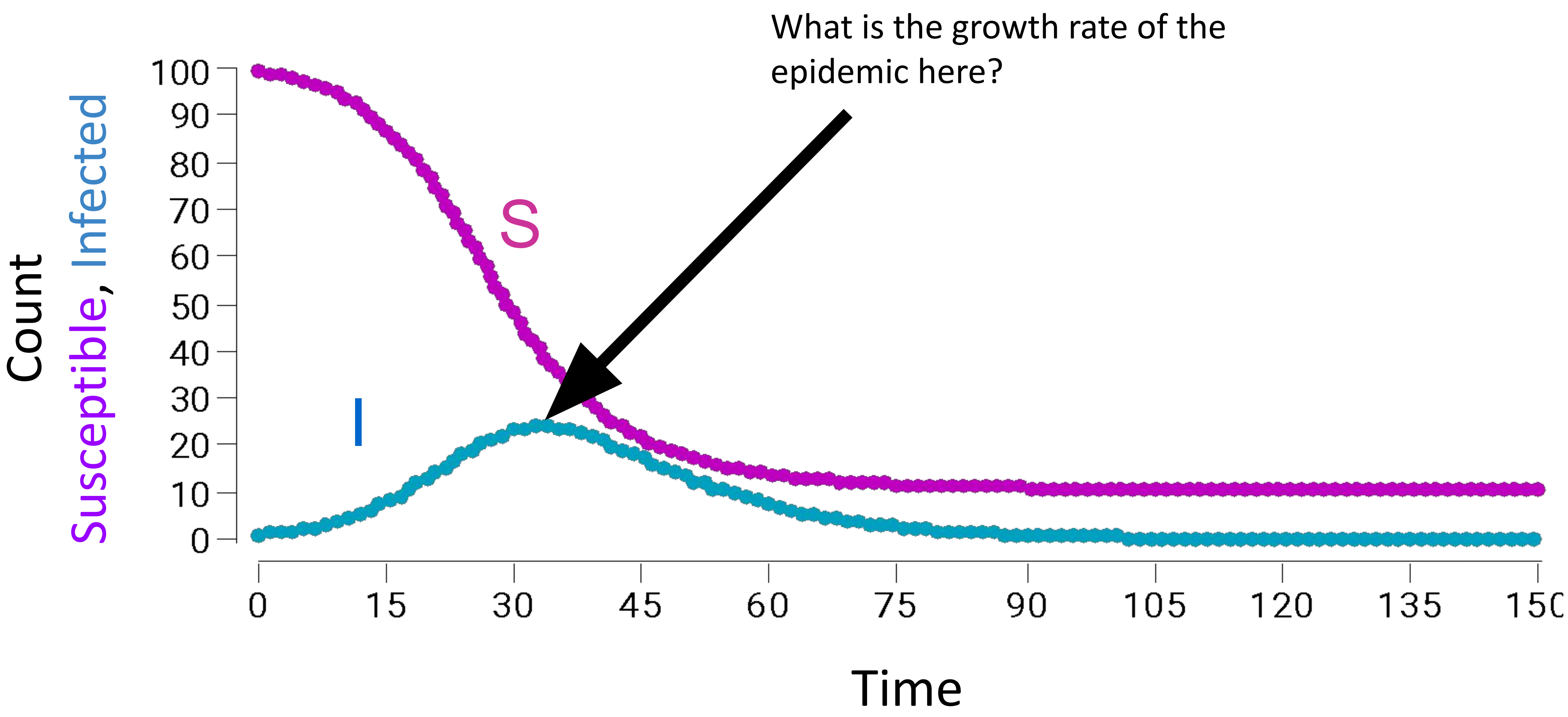
As individuals recover and the number susceptible declines, that growth slows because Susceptibles are being depleted

When does epidemic stop growing?

Idealized Epidemic in a Closed Community



Basic Reproduction Number

$$\frac{dS}{dt} = -\beta SI$$

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Under what conditions can the infectious compartment grow?

Basic Reproduction Number

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Under what conditions can the infectious compartment grow?

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

$$0 = \ \beta SI \ - \gamma I$$

$$0 = \ \beta S \ - \gamma$$

$$\beta S = \gamma$$

$$1 = \frac{\beta S}{\gamma}$$

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$$1 = \frac{\beta S}{\gamma}$$

Condition under which I doesn't change

Basic Reproduction Number

$$\frac{dS}{dt} = -\beta SI$$

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

$$\frac{dR}{dt} = \gamma I$$

Under what conditions can the infectious compartment grow?

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

$$0 > \ \beta SI \ - \gamma I$$

$$0 > \ \beta S \ - \gamma$$

$$\beta S < \gamma$$

$$1 > \frac{\beta S}{\gamma}$$

Condition under which I declines … epidemic fades

Basic Reproduction Number

$$\frac{dS}{dt} = -\beta SI$$

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

$$\frac{dR}{dt} = \gamma I$$

Under what conditions can the infectious compartment grow?

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

$$0 < \ \beta SI \ - \gamma I$$

$$0 < \ \beta S \ - \gamma$$

$$\beta S > \gamma$$

$$1 < \frac{\beta S}{\gamma}$$

Condition under which I grows … epidemic grows

Basic Reproduction Number

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

Under what conditions can the infectious compartment grow?

$$\frac{dI}{dt} = \beta SI \ - \gamma I$$
$$0 < \ \beta SI \ - \gamma I$$
$$0 < \ \beta S \ - \gamma$$
$$\beta S > \gamma$$
$$1 < \beta SL$$

Recall that $\frac{1}{\gamma} = L$ is the mean duration of infection

R_0: The Basic Reproduction Number

$$R_0 = \frac{\beta S}{\gamma} = \beta S L$$

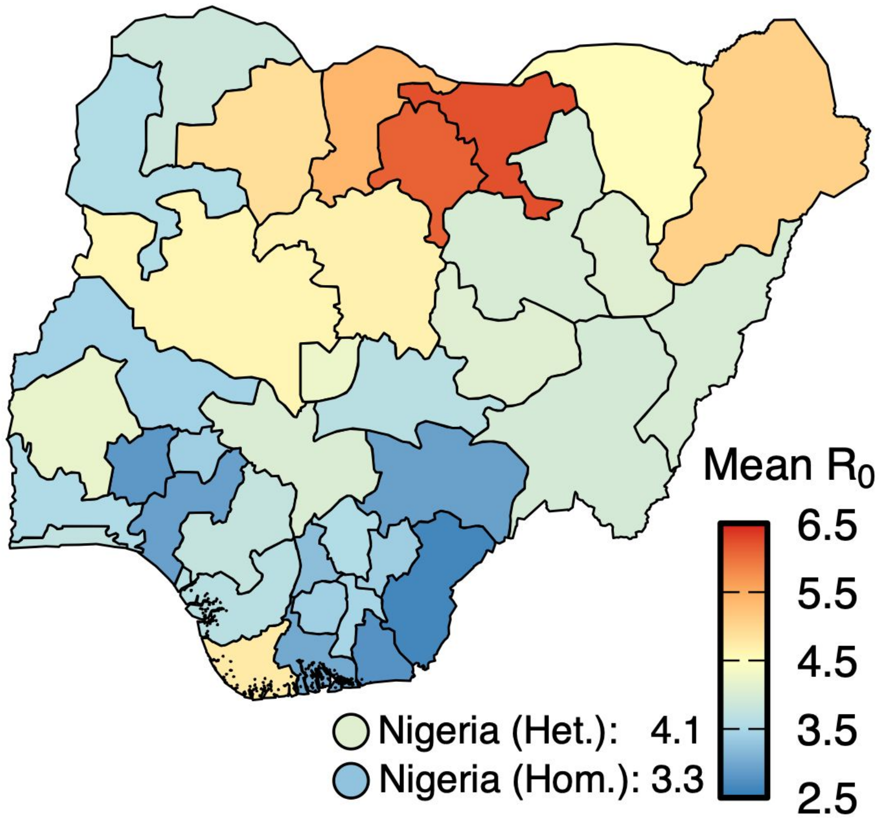
The expected number of new infections due to the first infection in a susceptible population

- A common currency
  - A function of the pathogen and the population (recall what β is)
  - Rarely observable directly
  - But closely related to many observable phenomena, as we'll see

Estimated values of R0 for various infections

Measles	England	1947	13-14
	Nigeria	1968	16-17
	Kansas	1920	5-6
Pertussis	England	1944-78	16-18
	Canada	1912	7-8
Chickenpox	USA	1912	7-8
	USA	1944	10-11

Basic Reproduction Number for Rubella



Estimated R0 varies over 2x even within a single country

Nakase et al 2024 Vaccine

What Does This Mean for Interventions?

$$R_0 = \frac{\beta S}{\gamma} = \beta S L$$

What does this suggest for interventions?

- Reduce β
- Reduce L (increase gamma)
- Reduce S

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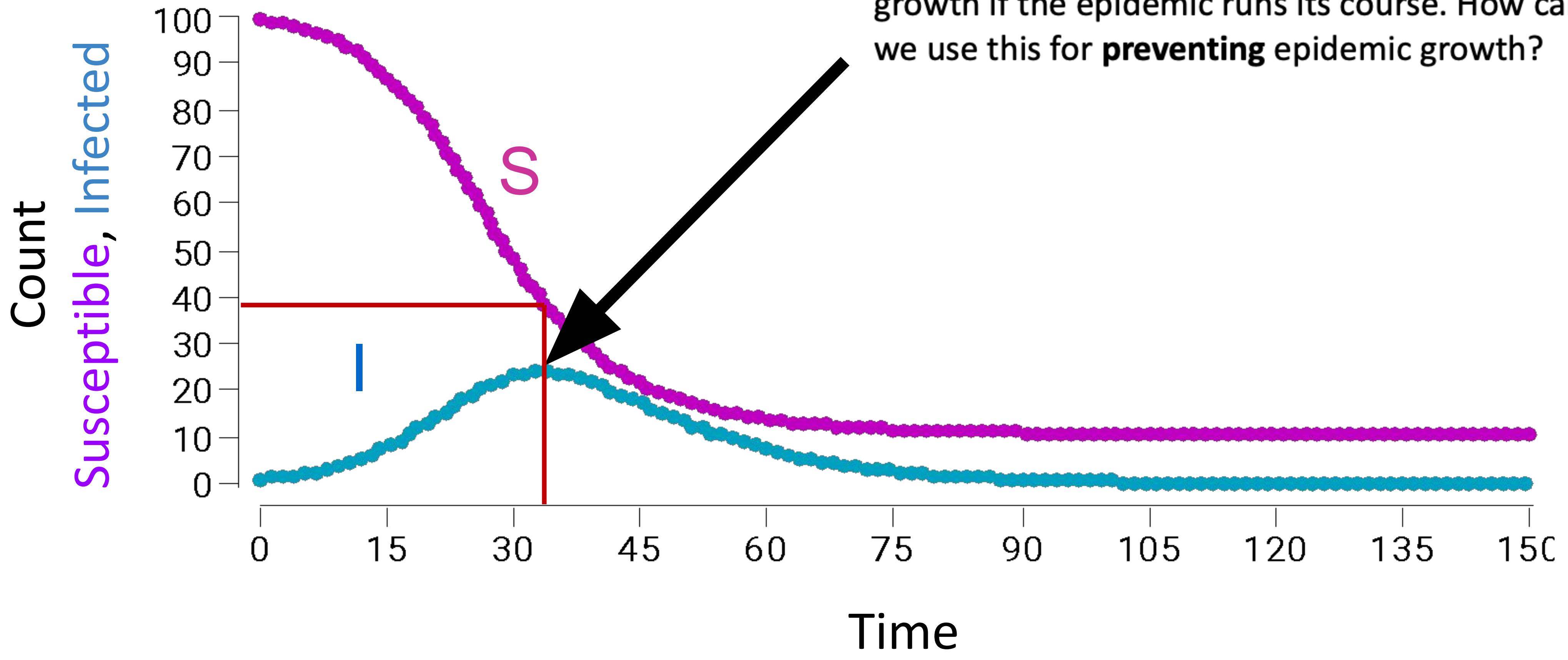
Idealized Epidemic in a Closed Community



Here is where S is small enough to prevent growth if the epidemic runs its course. How can we use this for **preventing** epidemic growth?

R_E : The Effective Reproduction Number

$$R_E = \frac{\beta p S}{\gamma} = \beta p S L$$

p is the fraction susceptible
1-*p* is the fraction immune

What does this suggest for interventions?

- Reduce β
- Reduce L (increase gamma)
- Reduce S

The expected number of new infections due to each infection in a population with some immunity

Herd Immunity Threshold

$$R_0 = \frac{\beta S}{\gamma} = \beta S L$$

$$R_0 = \beta S L$$

$$1 = \frac{\beta S L}{R_0}$$

$$1 = \frac{1}{R_0} S \beta L$$

What fraction of Susceptibles need to be immune in order for

$$\frac{1}{R_0} S$$

to remain?

Herd Immunity Threshold

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What fraction of Susceptibles need to be immune in order for

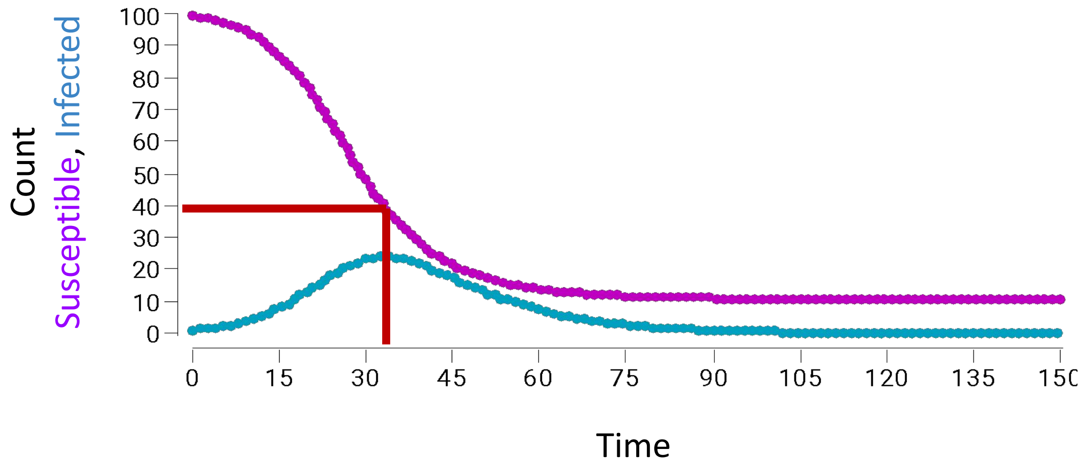
$$\frac{1}{R_0} S$$

to remain?

$$T_c = 1 - \frac{1}{R_0}$$

Herd Immunity Threshold

If $T_c = 1 - \frac{1}{R_0}$ are immune ***before*** an outbreak then it won't be able to grow (on average)



If an outbreak takes off it ***WILL NOT*** stop when $T_c = 1 - \frac{1}{R_0}$ are immune

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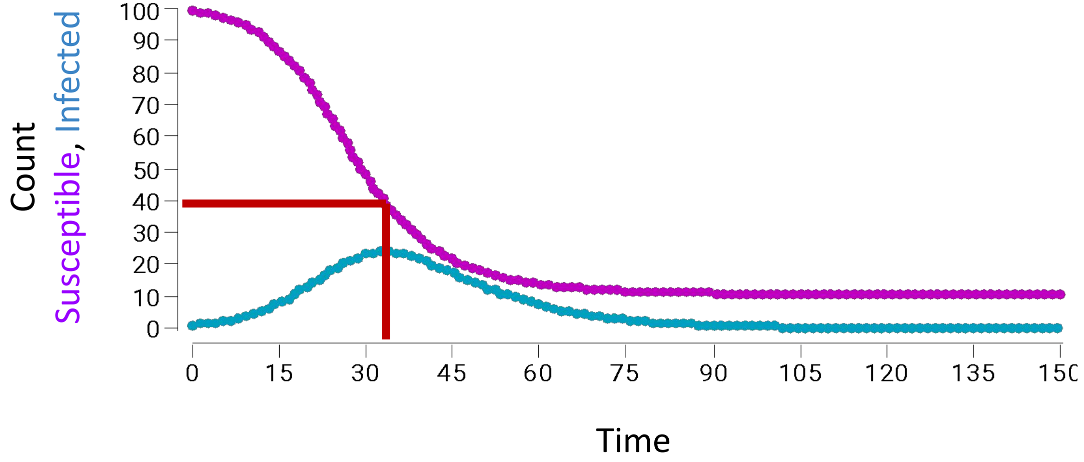


If an outbreak takes off it ***WILL NOT*** stop when $T_c = 1 - \frac{1}{R_0}$ are immune

Why not?

Final Size Calculation

$$R_\infty = 1 - e^{-R_0 R_\infty}$$

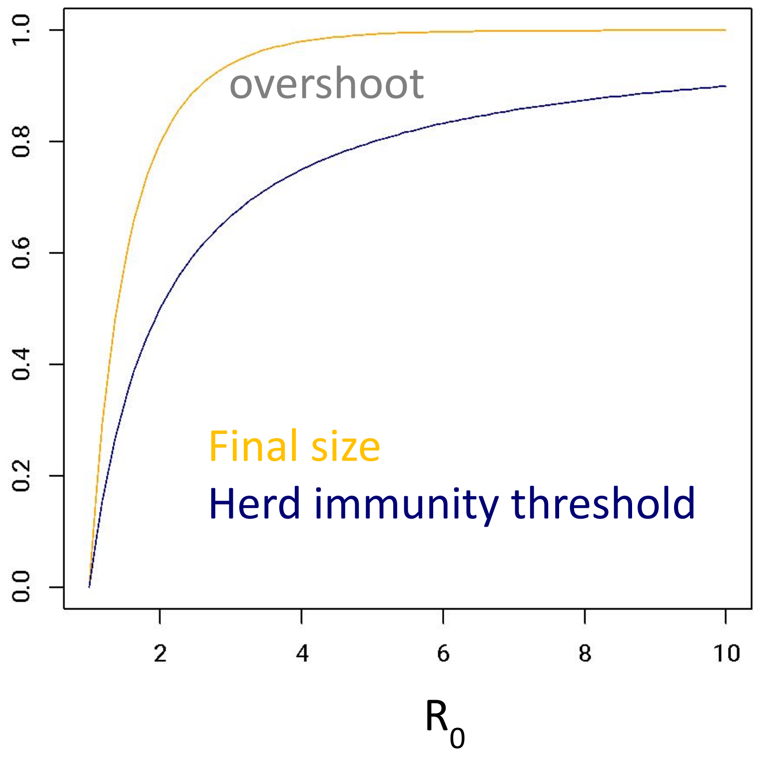
Where R_∞ is the proportion of the population infected at the end of the epidemic (the proportion in the R class at the end)

Citation:https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3506030/

Comparing T_c and Final Size

Many more individuals will become infected in an epidemic (on average) than need to be immunized ***BEFORE*** an epidemic

Herd Immunity is a relevant concept throughout an epidemic (and helps stop them), the Herd Immunity Threshold is only relevant for preventing, not stopping outbreaks.



What Happens When Births Are Added?

$$\frac{dS}{dt} = \delta N - \beta SI - \delta S$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \alpha I - \delta I$$

$$\frac{dR}{dt} = \gamma I - \delta R$$

δ is birth and death rate

α is disease induced death rate

What Happens When Births Are Added?

$$\frac{dS}{dt} = \delta N - \beta SI - \gamma I - \alpha I - \delta S$$

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$$\frac{dR}{dt} = \delta N - \beta SI - \gamma I - \alpha R - \delta R$$

When there’s NO infection?

δ is birth and death rate

α is disease induced death rate

What Happens When Births Are Added?

$$\frac{dS}{dt} = \delta N - \beta SI - \gamma I - \alpha I - \delta S$$

$$\frac{dI}{dt} = \delta N - \beta SI - \gamma I - \alpha I - \delta I$$

$$\frac{dR}{dt} = \delta N - \beta SI - \gamma I - \alpha R - \delta R$$

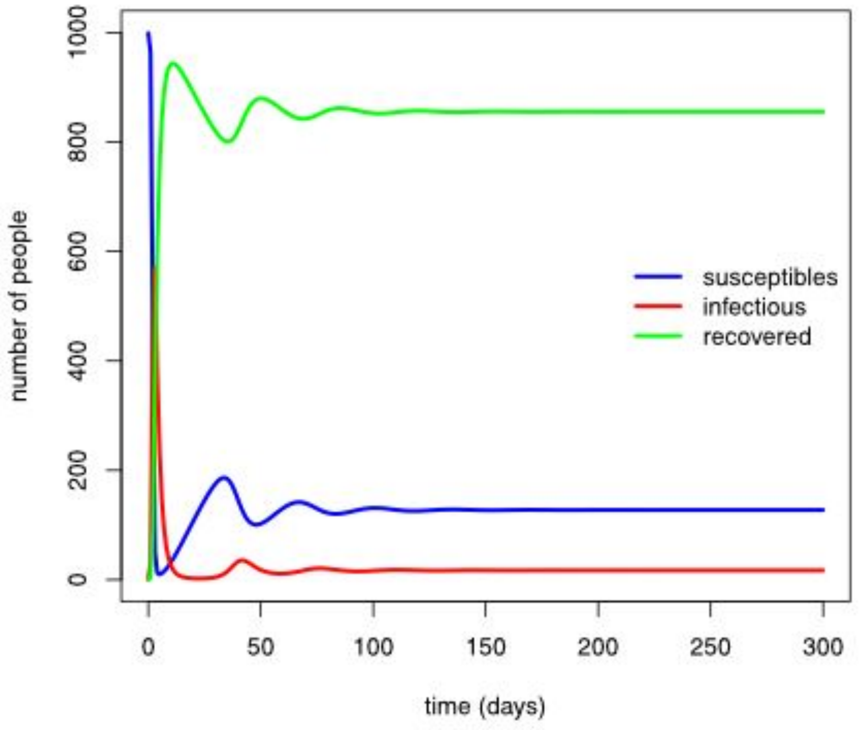
When there’s very little infection?

δ is birth and death rate

α is disease induced death rate

Dynamics Over Time

Note that after initial overshoot, susceptibles build back up until a new outbreak occurs, the second is smaller, and the third smaller than that



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- Open populations can have births, immigration, deaths, emigration
  - Either explicitly or implicitly

Equilibrium and Transient

- At equilibrium, the values of all states are constant – individuals may still get sick, recover, etc, but all changes balance each other out
- Dynamics are transient if the states are continuing to change … more nuance later (really)

When Does *I* Stop Growing?

$$\frac{dI}{dt} = \beta SI \ - \gamma I \ - \alpha I - \delta I$$

$$0 < \beta SI \ - \gamma I \ - \alpha I - \delta I$$

$$\beta SI > \ \gamma I + \alpha I + \delta I$$

$$\beta S > \ \gamma + \alpha + \delta$$

$$1 < \frac{\beta S}{\gamma + \alpha + \delta} \equiv R_0$$

δ is birth and death rate

α is disease induced death rate

When Does *I* Stop Growing?

$$\frac{dI}{dt} = \beta SI \ - \gamma I \ - \alpha I - \delta I$$
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$$\beta SI > \ \gamma I + \alpha I + \delta I$$
$$\beta S > \ \gamma + \alpha + \delta$$
$$1 < \frac{\beta S}{\gamma + \alpha + \delta} \equiv R_0$$

Why do these terms show up in equation for R_0?

δ is birth and death rate

α is disease induced death rate

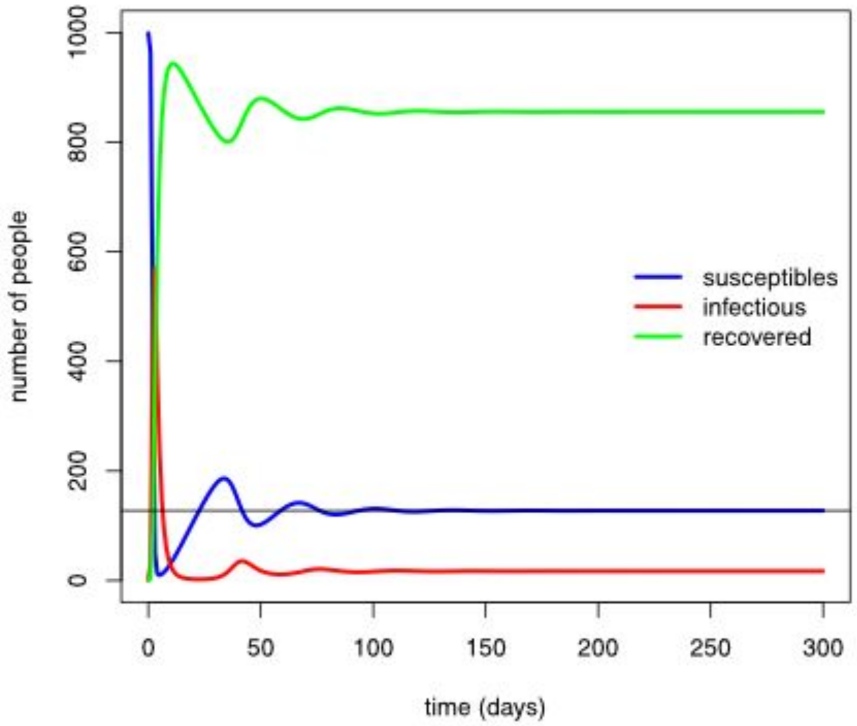
Equilibrium Dynamics

The stable equilibrium proportion susceptible is

$$\sim \frac{1}{R_0}$$

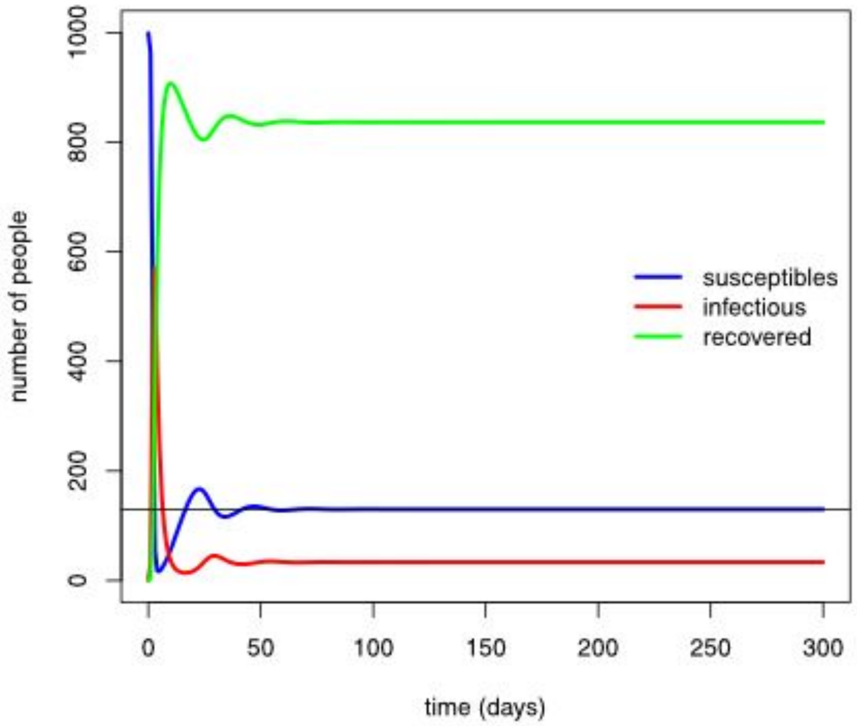
and the stable proportion recovered (immune) is

$$\sim 1 - \frac{1}{R_0}$$



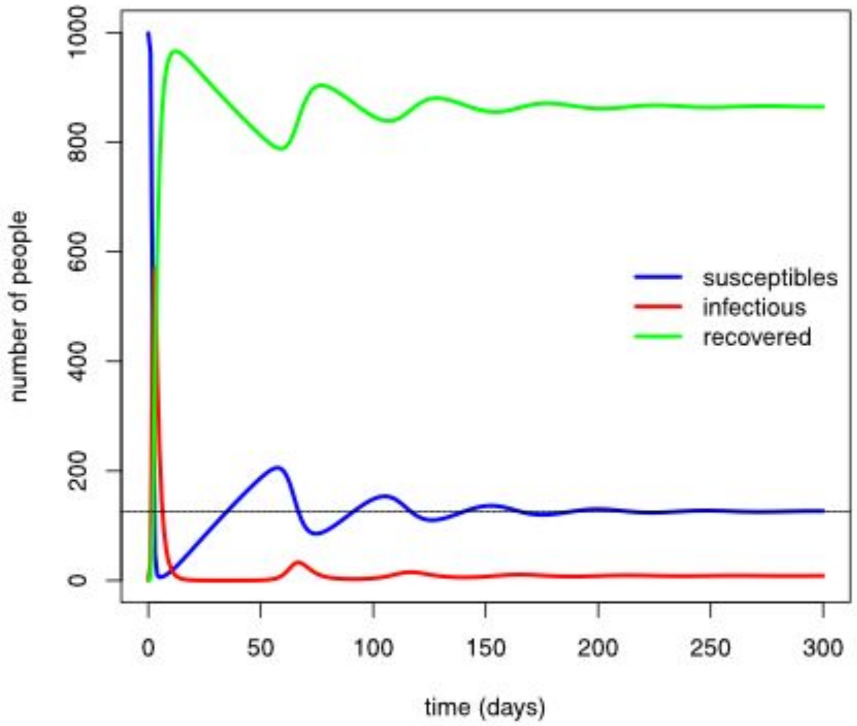
Birth Rate Changes the Speed to Equilibrium

If we increase the birth rate, it takes less time to reach equilibrium under the assumption that the population isn't growing



Birth Rate Changes the Speed to Equilibrium

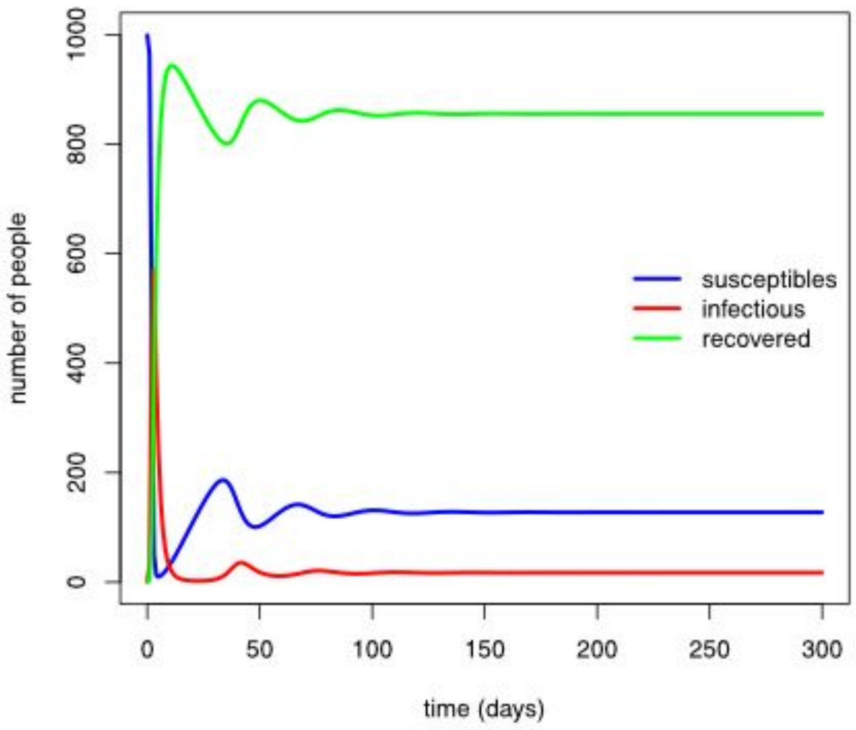
If we decrease the birth rate, it takes longer to reach equilibrium under the assumption that the population isn't growing



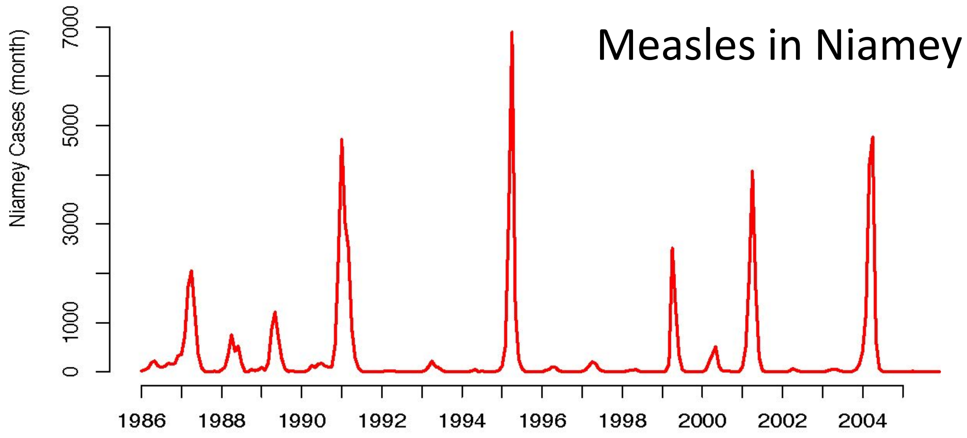
What about growing populations?

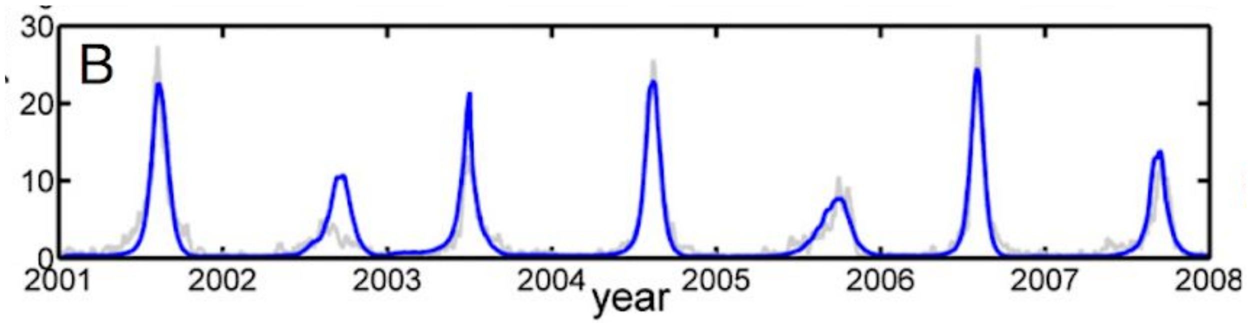
- Growing populations have more susceptibles added than recovereds being taken away (by death)
  - So a greater fraction susceptible, less indirect protection, and more transmission
- More of those susceptibles are young, so if young and old have different contact rates, then transmission and dynamics will differ in young vs. old populations …

Seasonality and Cycles



Measles in Niamey





Influenza in Jerusalem

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Equilibrium and Transient

- At equilibrium, the values of all states are constant – individuals may still get sick, recover, etc, but all changes balance each other out
- An *attractor* is collection of states towards which a system tends – it's regular and predictable, but not static.
- Dynamics are transient if they are neither of these … which is most of time

Seasonality and Cycles

- Long term dynamics often exhibit regular fluctuations around the equilibrium levels because of seasonal changes in
  - Environmental conditions
  - Behavior
  - Population movement/aggregation
  - Vector seasonality

Examples
Influenza
Lassa fever
Legionellosis
Leptospirosis
Meningococcal meningitis
Polio
Typhoid

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Pertussis
Rubella

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Modeled as a temporal change in β

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Examples
Measles
Meningococcal meningitis

Modeled as a temporal change in β ***or*** S

Seasonality and Cycles

- Long term dynamics often exhibit regular fluctuations around the equilibrium levels because of seasonal changes in
  - Environmental conditions
  - Behavior
  - Population movement/aggregation
  - Vector seasonality

Examples
Chikungunya
Dengue
Malaria
Trypanosomiasis
West Nile Virus
Yellow Fever

Requires a new compartment for the vector populations