

# What's your favorite animal so far?



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0%



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0%



0%



0%

# What's your favorite animal so far?



0%



0%



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0%

# Fundamentals of Ecology

Week 8, Ecology Lecture 5

Cara Brook

February 20, 2024

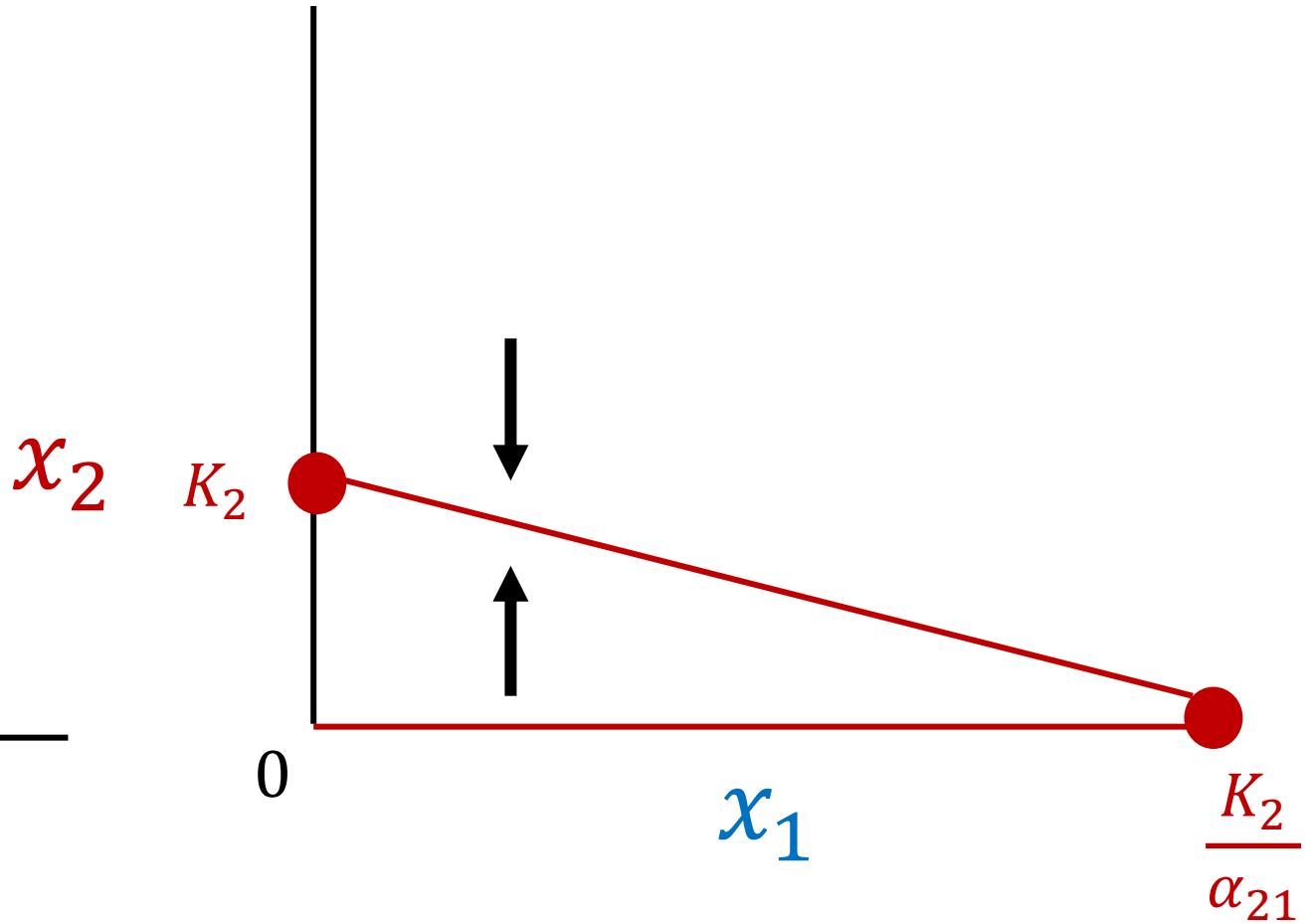
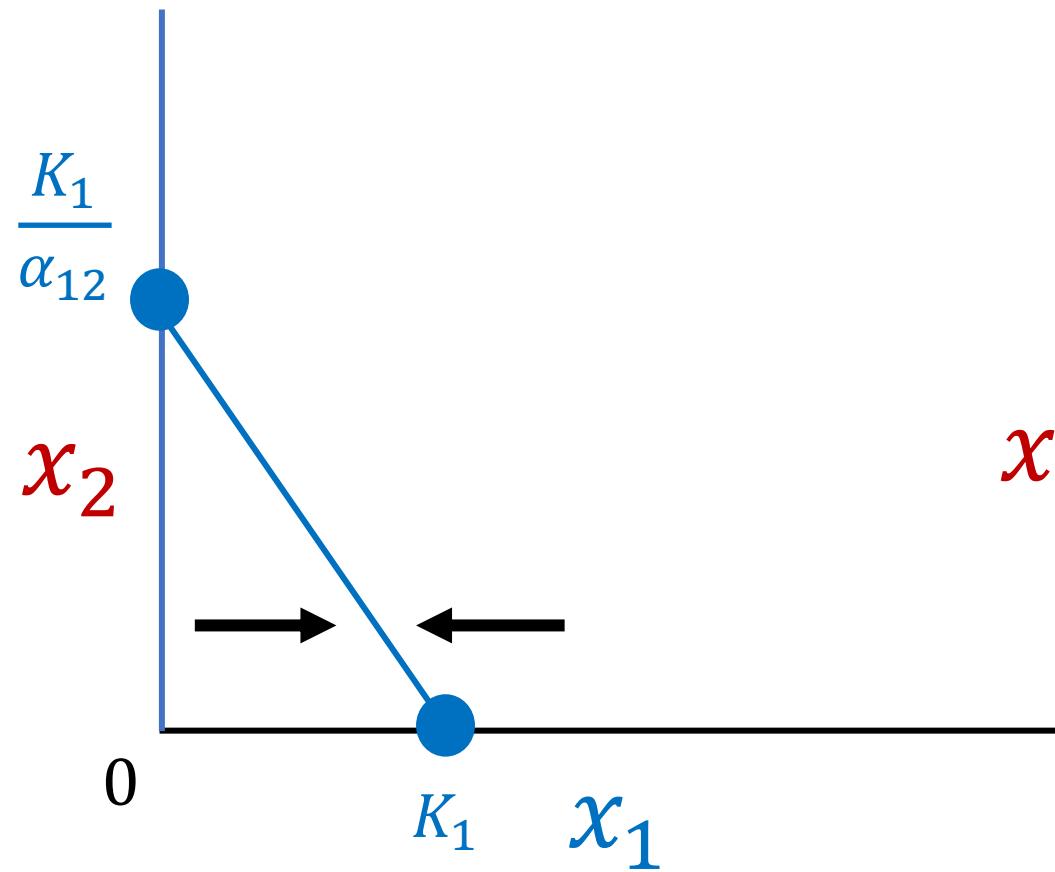
# Let's recap a bit!

## ***Interspecies Dynamics***

- The Lotka-Volterra competition model describes a minimum of two interacting species whose populations are regulated both by intra- and interspecific competition for limited resources.
- We can draw the nullclines (or zero-growth isoclines) of this model. These lines describe the conditions under which the RATE of change for one species population is not changing.
- The four equilibria of the system give the conditions under which the rate of change is 0 for both species populations. Under certain parameter conditions, we can find an equilibrium in which the two species demonstrate stable coexistence.
- We can determine the stability of the system using vector addition on the nullcline planes.

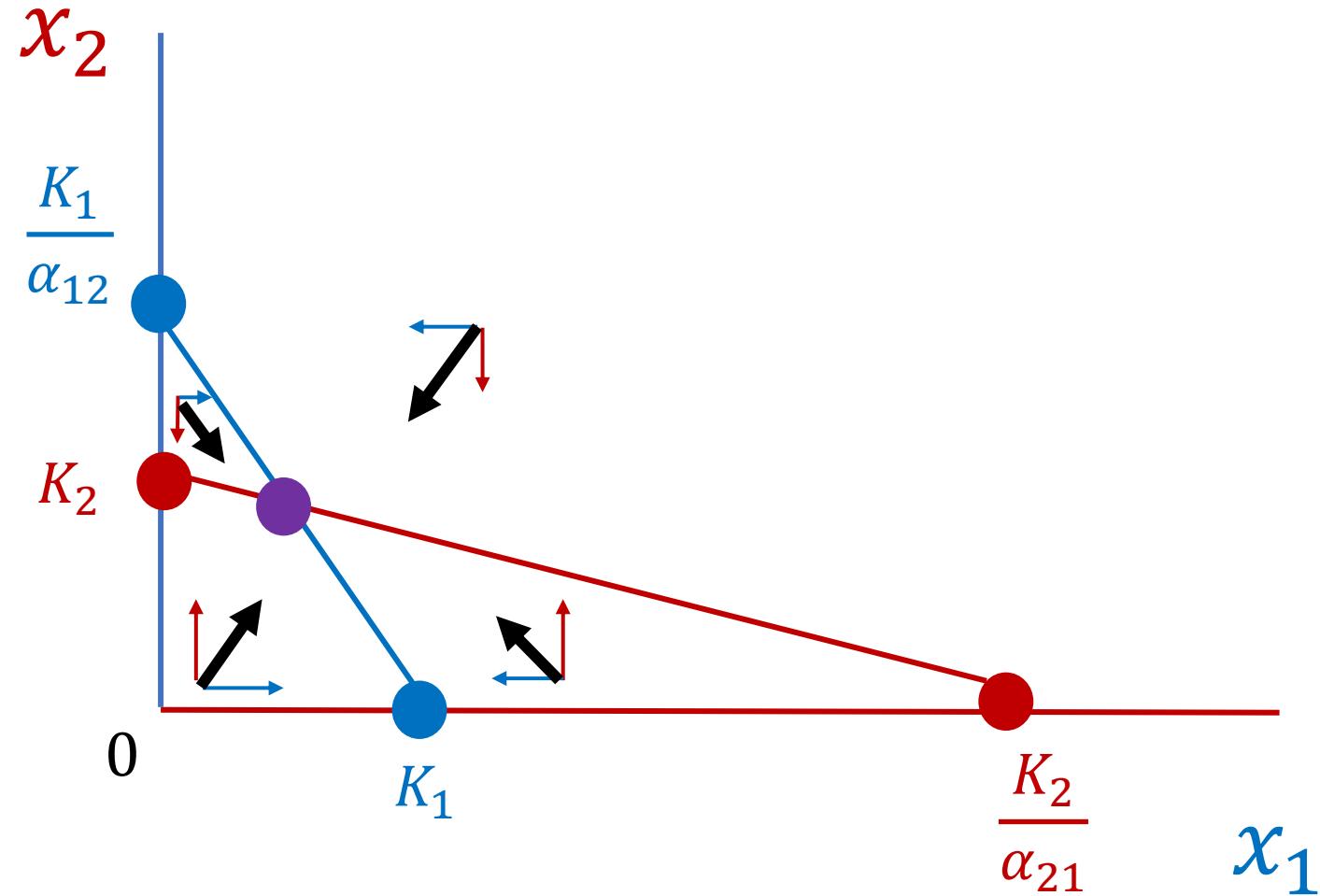
# Let's recap a bit!

## Interspecies Dynamics



Let's recap a bit!

### Interspecies Dynamics

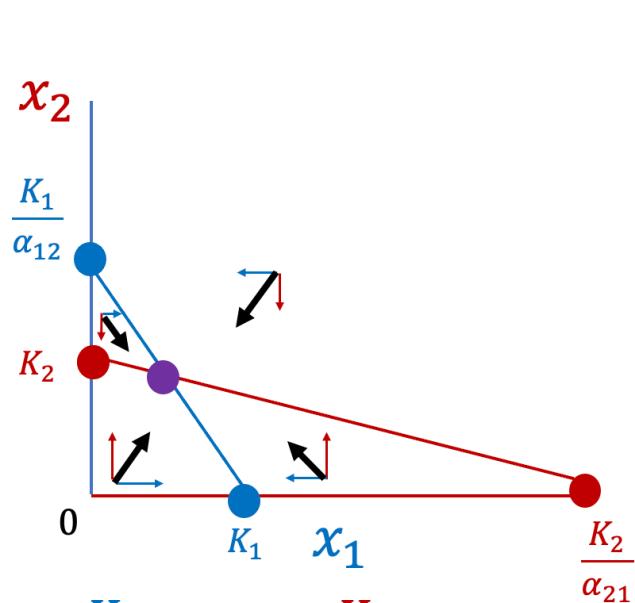


# Let's recap a bit!

## Interspecies Dynamics

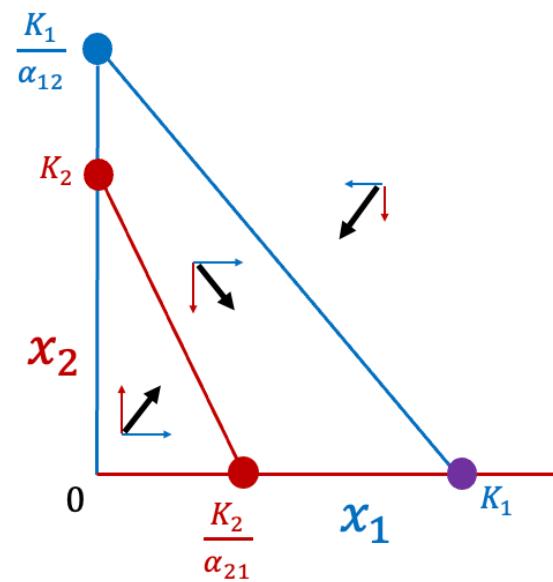
- There are four possible outcomes of competition that can be observed from drawing the nullclines of the system.

**Case 1: Stable coexistence**



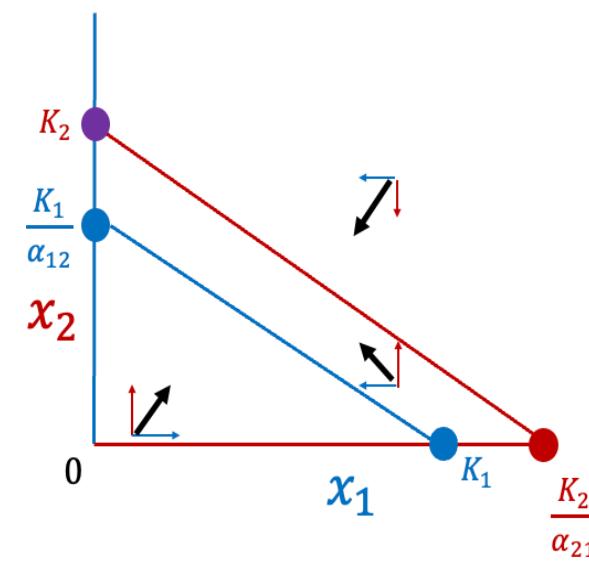
$$\frac{K_1}{\alpha_{12}} > K_2 \text{ and } \frac{K_2}{\alpha_{21}} > K_1 \\ (\alpha_{12} * \alpha_{21} < 1)$$

**Case 2: Spp. 1 wins**



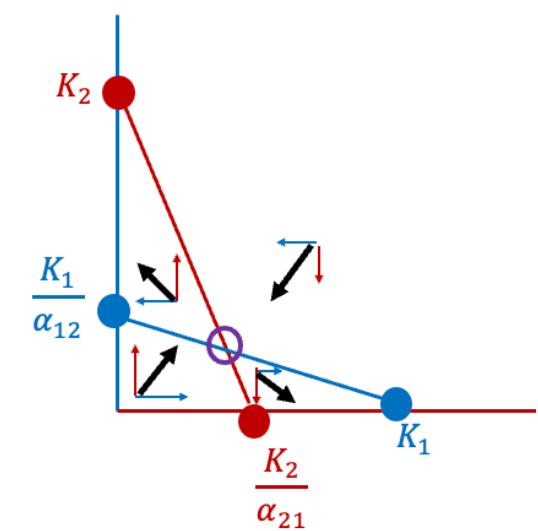
$$\frac{K_1}{\alpha_{12}} < K_2 \text{ and } \frac{K_2}{\alpha_{21}} > K_1$$

**Case 3: Spp. 2 wins**



$$\frac{K_1}{\alpha_{12}} < K_2 \text{ and } \frac{K_2}{\alpha_{21}} > K_1$$

**Case 4: Precedence**



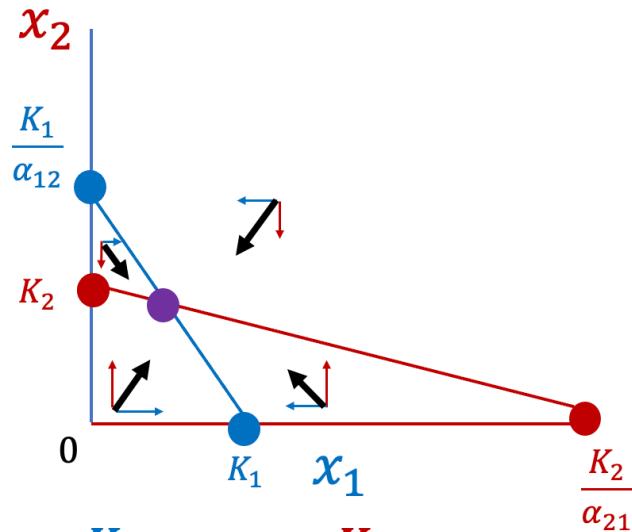
$$\frac{K_1}{\alpha_{12}} < K_2 \text{ and } \frac{K_2}{\alpha_{21}} < K_1 \\ (\alpha_{12} * \alpha_{21} > 1)$$

# Let's recap a bit!

## Interspecies Dynamics

- There are four possible outcomes of competition that can be observed from drawing the nullclines of the system.

### Case 1: Stable coexistence



$$\frac{K_1}{\alpha_{12}} > K_2 \text{ and } \frac{K_2}{\alpha_{21}} > K_1$$

$(\alpha_{12} * \alpha_{21} < 1)$

Stable coexistence is possible when intraspecies competitive interactions are greater than interspecies interactions.

# Gause's experiments:

Competitive exclusion was observed:



*Paramecium aurelia*



*Paramecium caudatum*

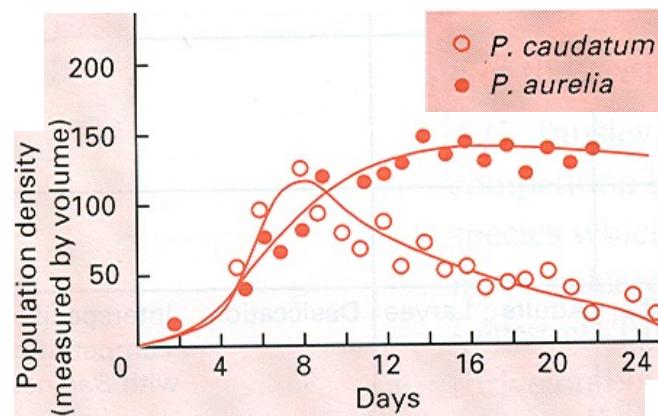
Coexistence was observed:



*Paramecium caudatum*

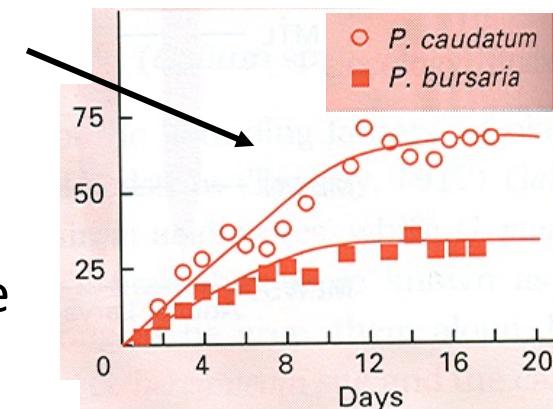


*Paramecium bursaria*



Species coexisted below each species' respective individual carrying capacity.

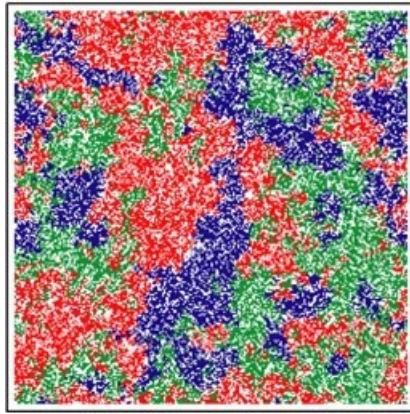
The two coexisting species were largely partitioned in space. *P. bursaria* ate yeast at the bottom and *P. caudatum* consumed bacteria suspended in the medium.



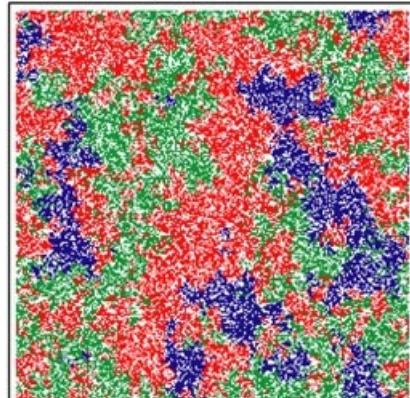
Gause 1934. *J Experimental Biology*.  
Gause 1935. *Science*.

# Sometimes **stochasticity** and **space** are all you need to ensure **coexistence** in a competitive environment

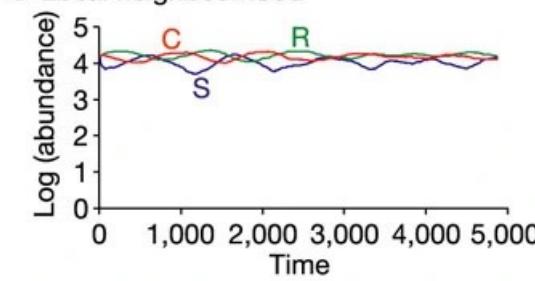
a Time step 3,000



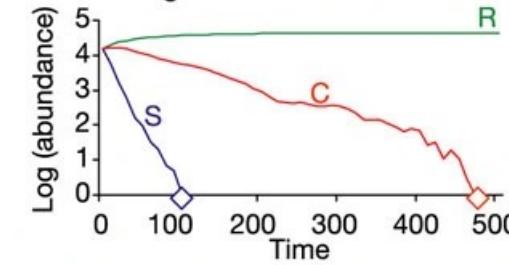
b Time step 3,200



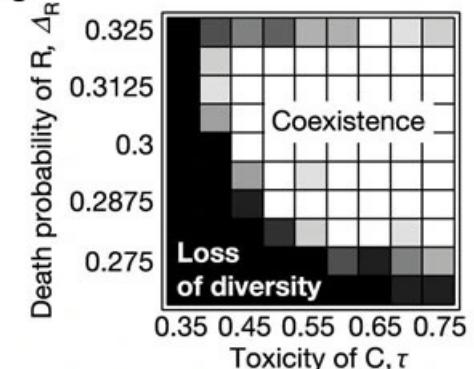
c Local neighbourhood



d Global neighbourhood

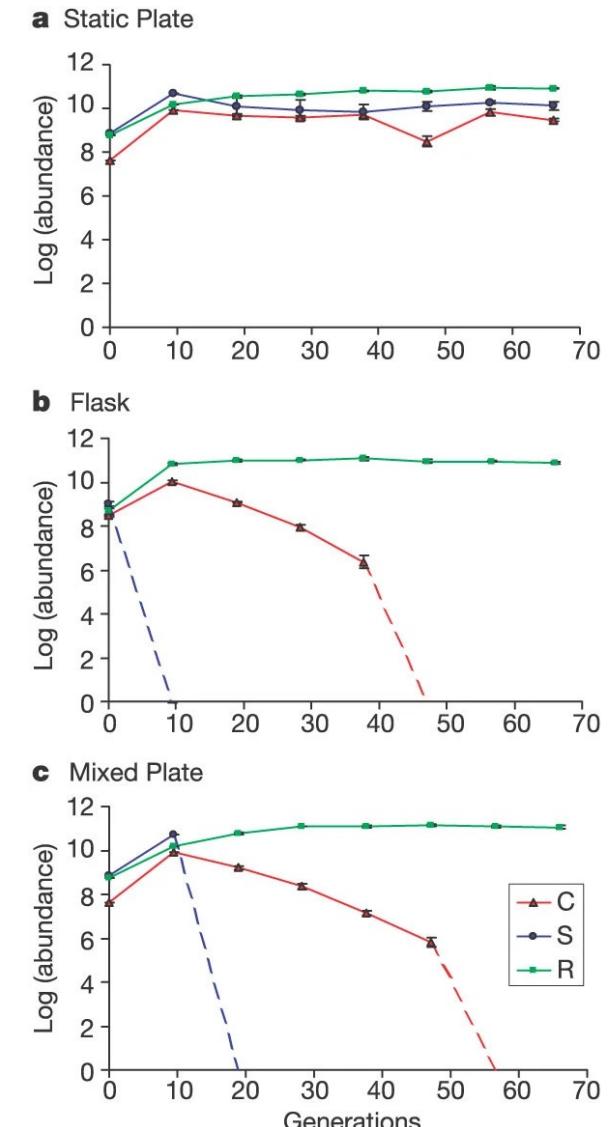
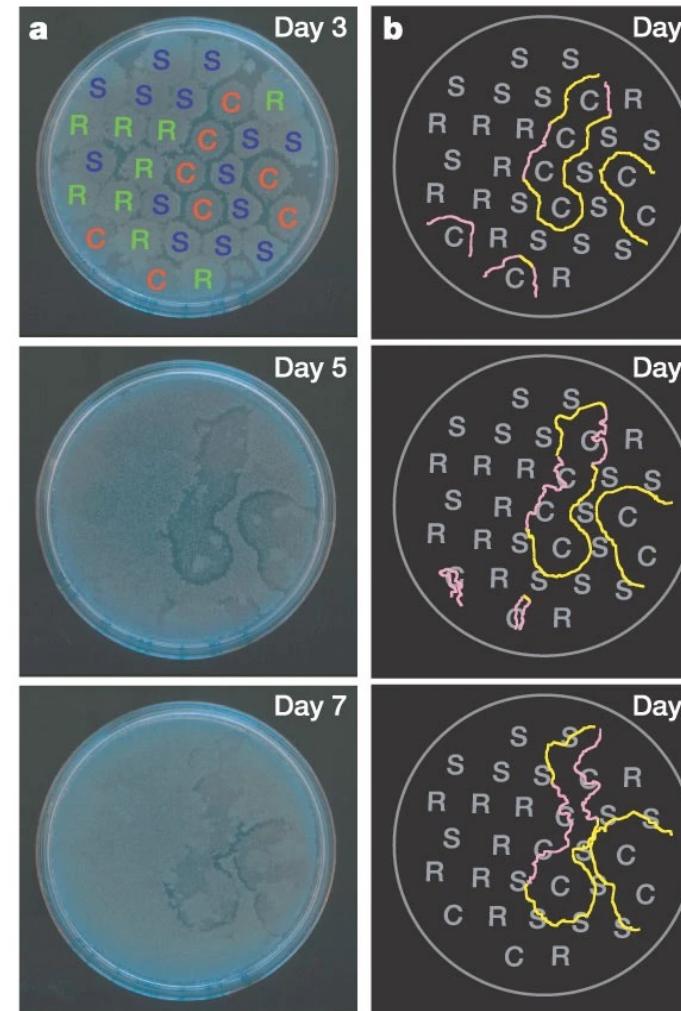
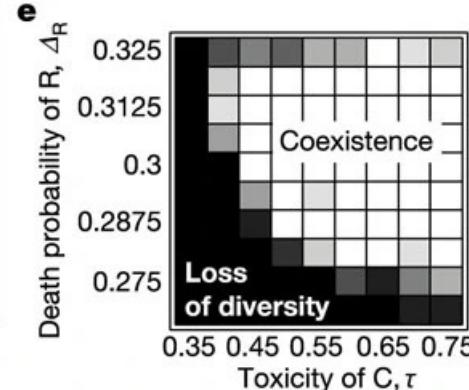
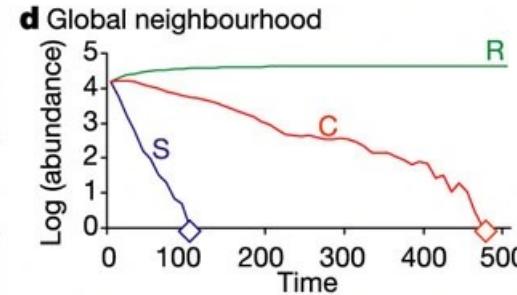
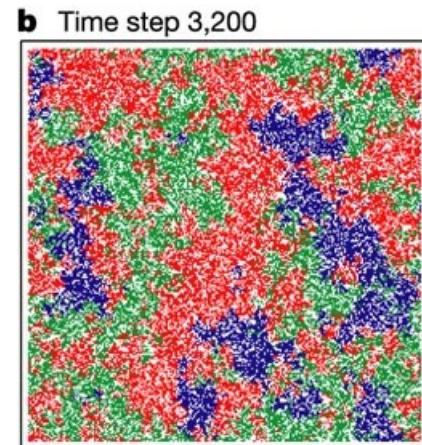
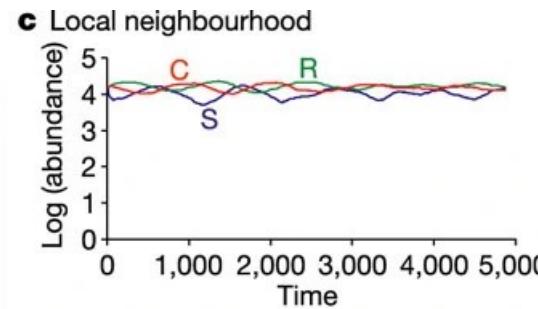
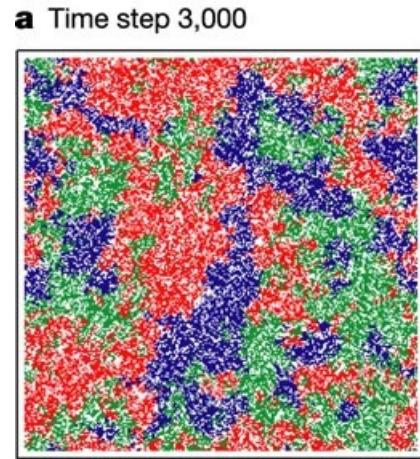


e



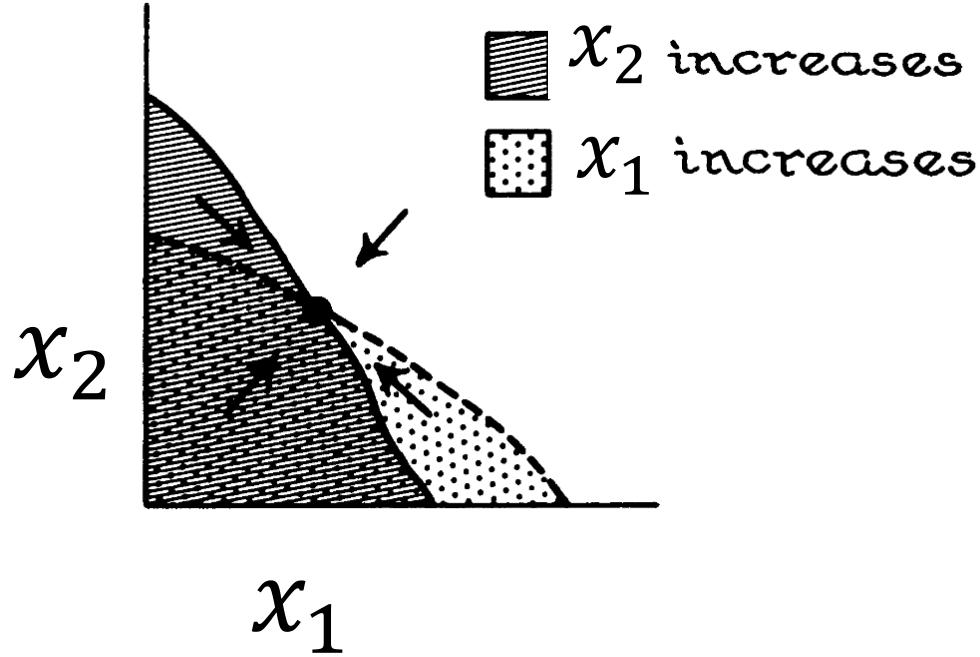
- The authors modeled a community of 3 strains of *E. coli* with **overlapping resource requirements** (C,R, and S), occupying distinct spatial patches in a metapopulation.
- They produced simulations allowing for a perfectly mixed population (global neighborhood), or a population in which dispersal (mixing) happened only locally.
- **Local interactions allowed for the coexistence of all three strains** (in theory). What about experimentally?

# Sometimes adding **stochasticity** and **space** (remember metapopulations!) is all you need to ensure **coexistence**!

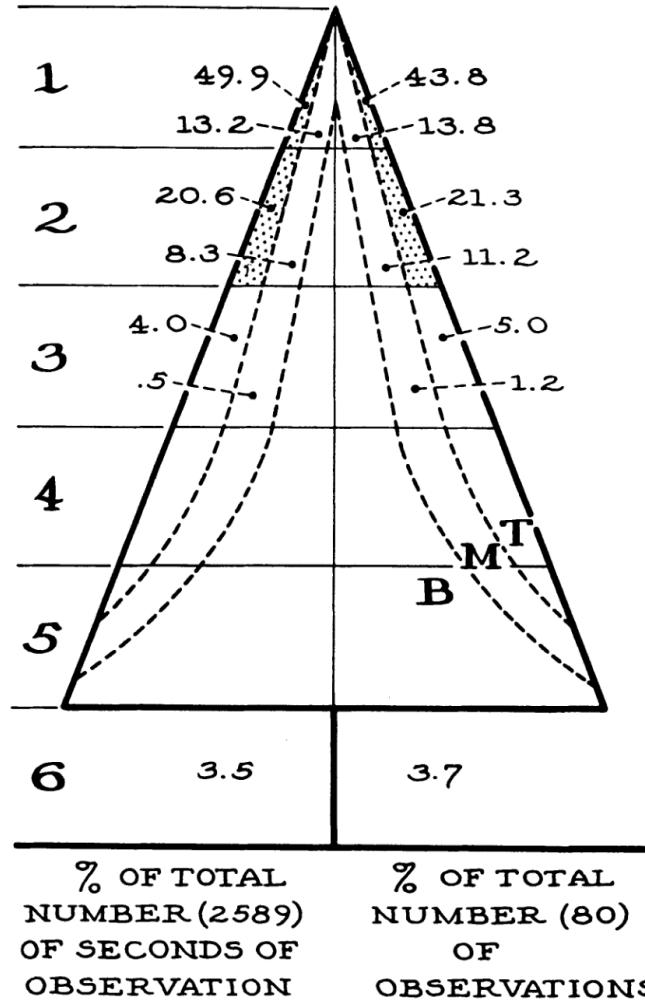


# Niche partitioning enables organisms to avoid competitive exclusion

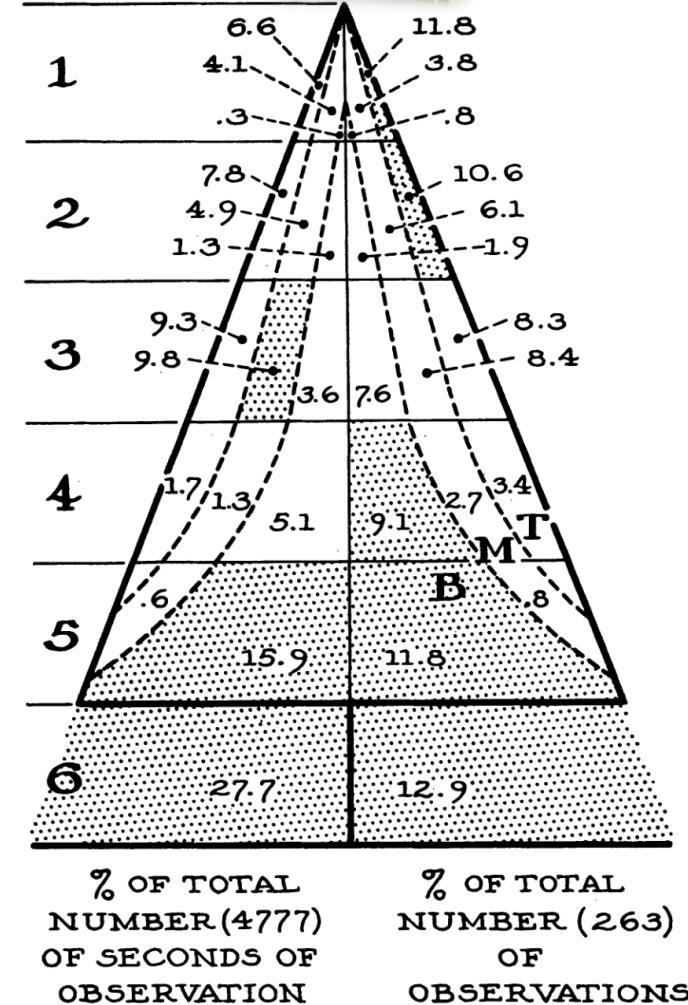
**Niche**: a match of a species to a specific environmental condition



Cape May  
Warbler

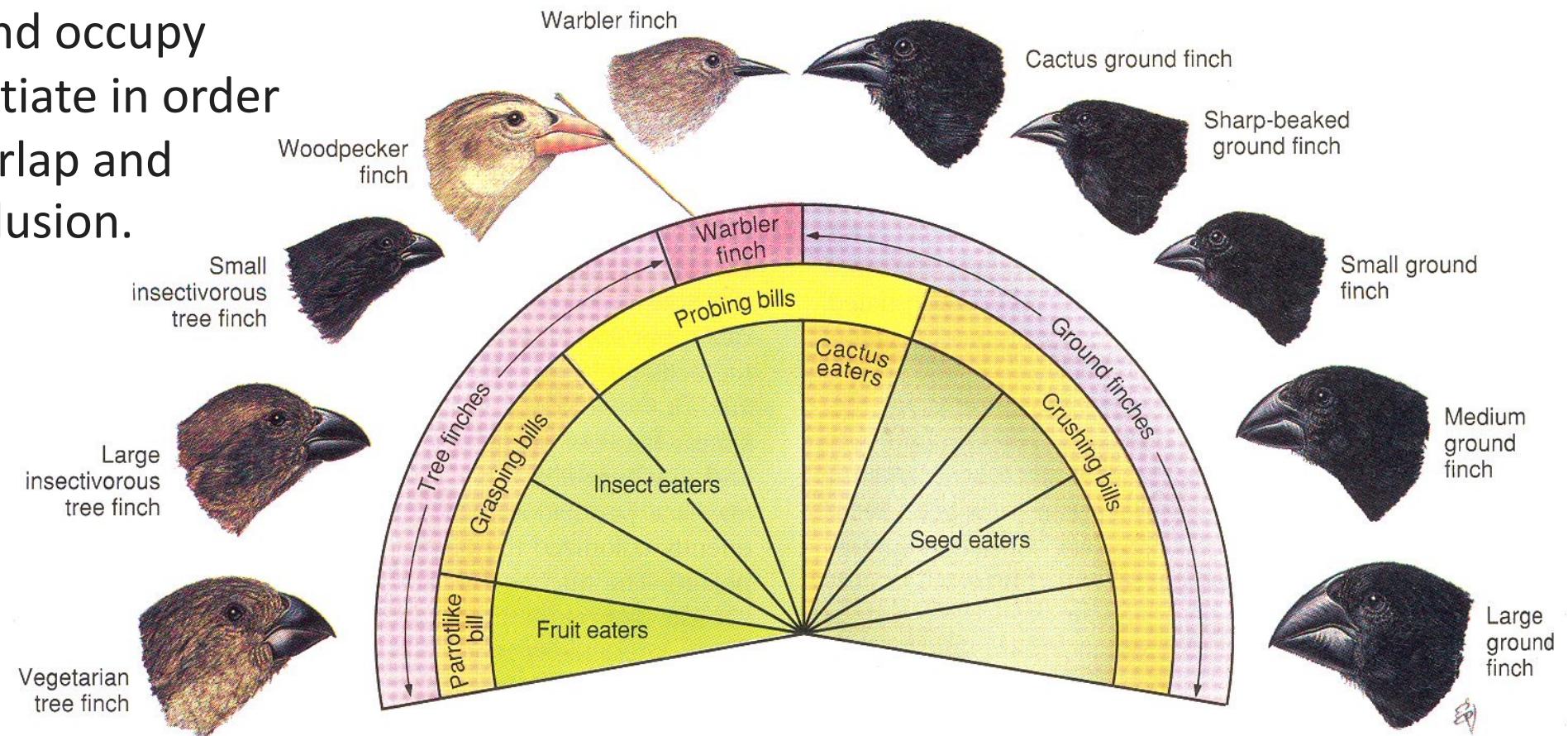


Myrtle  
Warbler



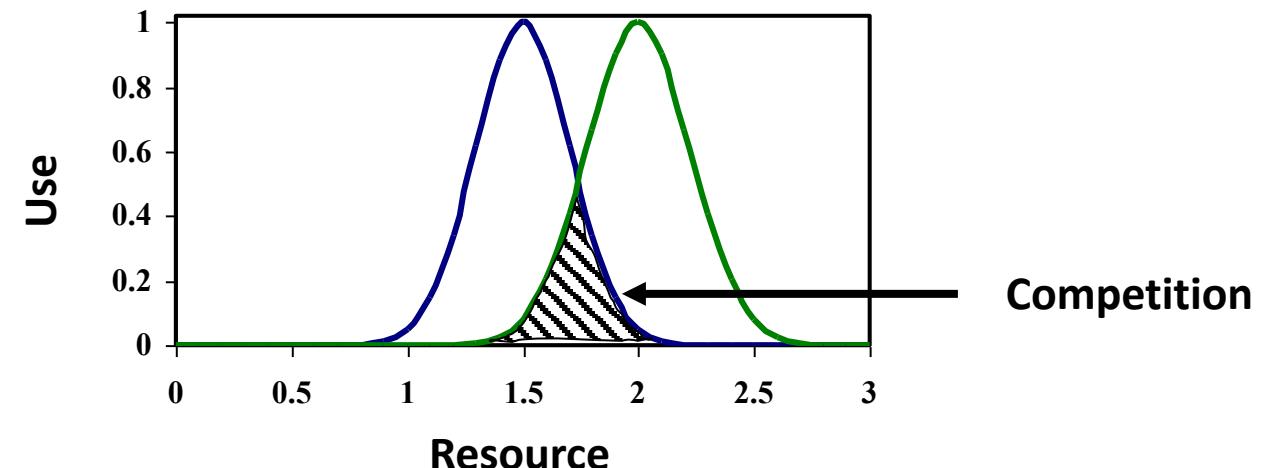
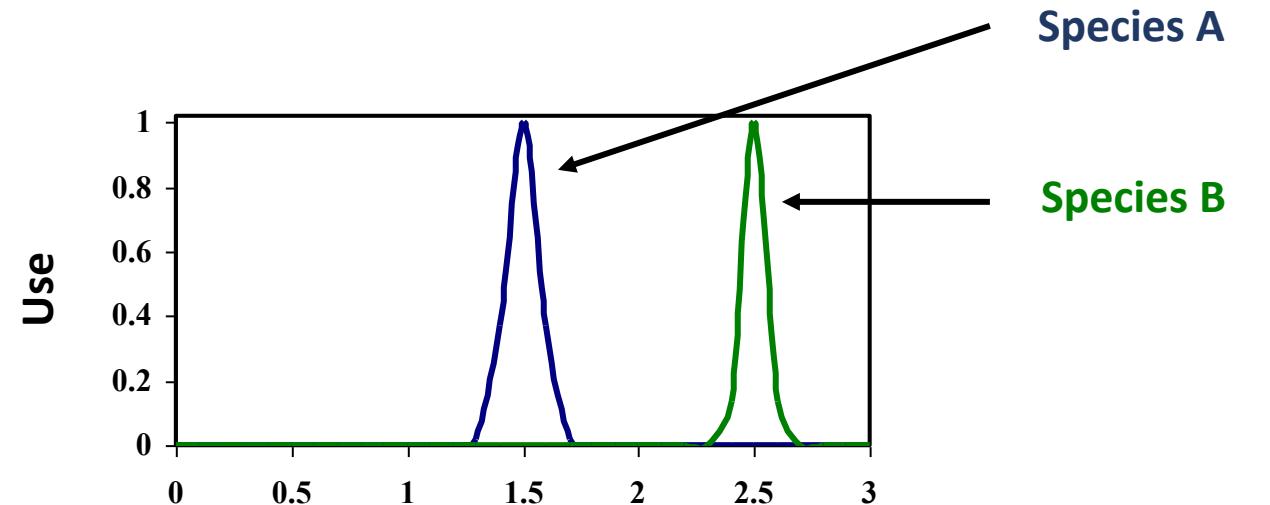
# Niche partitioning enables organisms to avoid competitive exclusion

**Character displacement:** similar species that live in the same geographical region and occupy similar niches differentiate in order to minimize niche overlap and avoid competitive exclusion.



# Niche partitioning enables organisms to avoid competitive exclusion

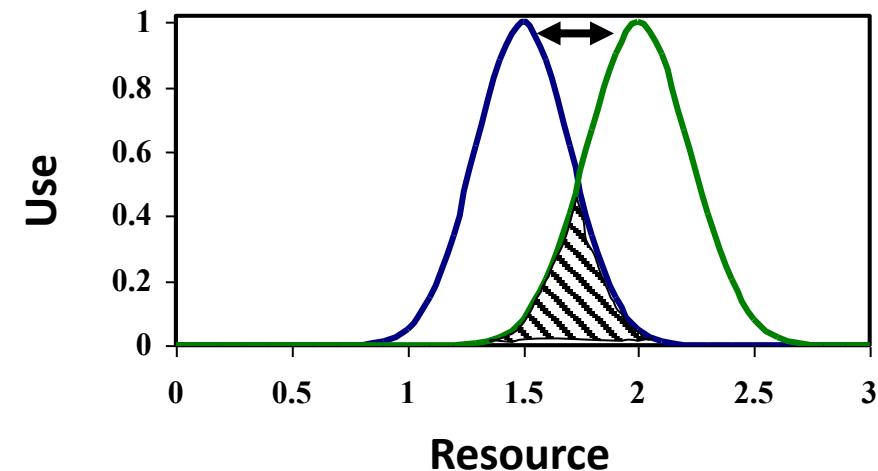
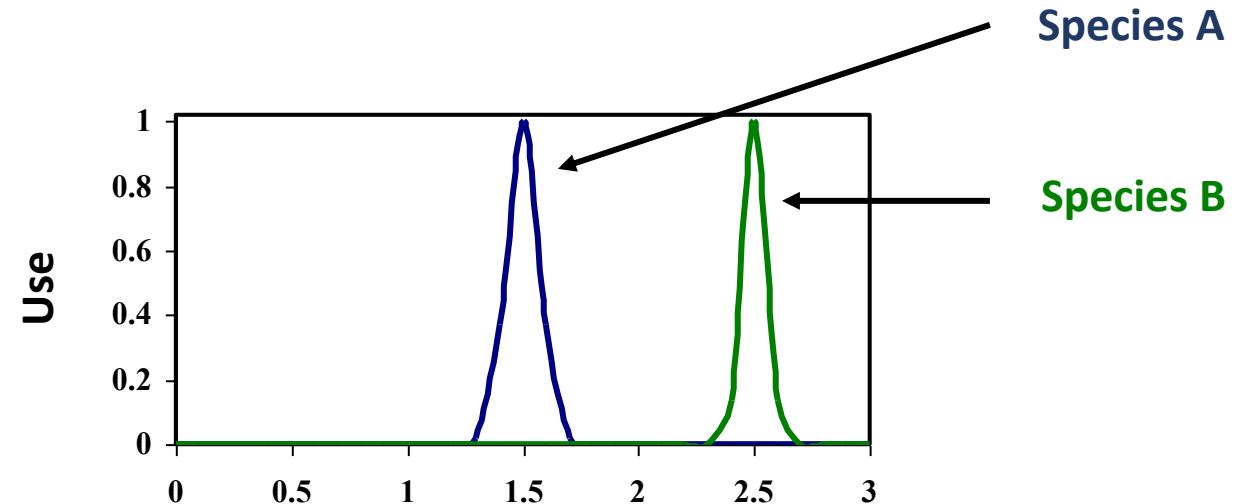
**Character displacement:** similar species that live in the same geographical region and occupy similar niches differentiate in order to minimize niche overlap and avoid competitive exclusion.



When niches overlap, competition results

# Niche partitioning enables organisms to avoid competitive exclusion

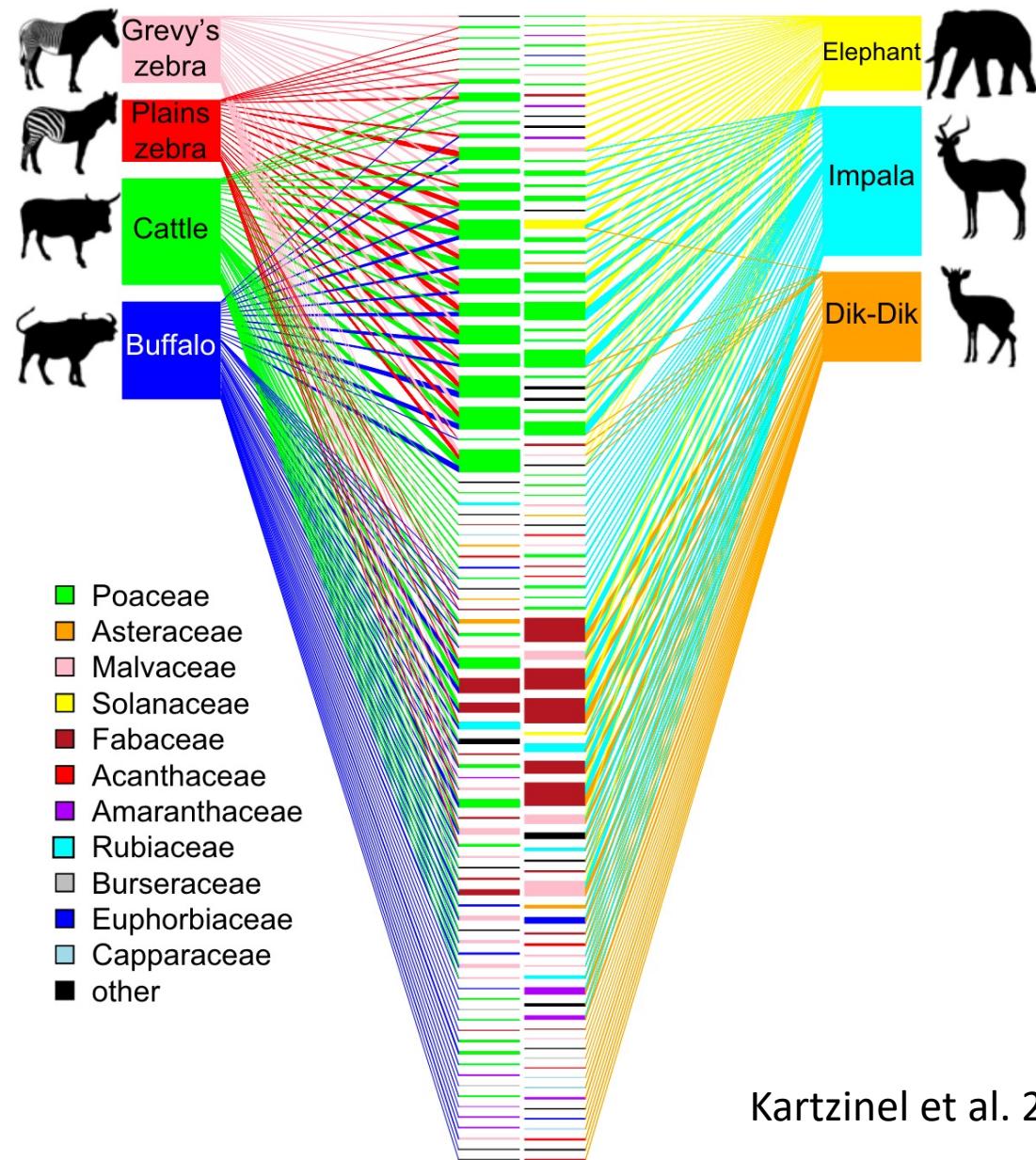
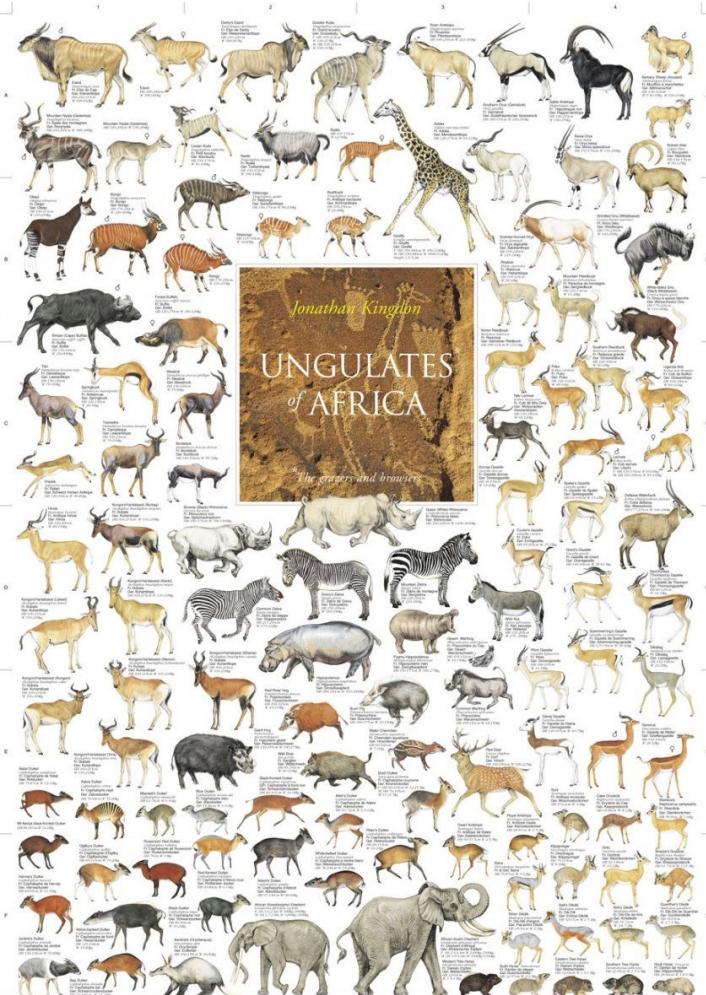
**Character displacement:** similar species that live in the same geographical region and occupy similar niches differentiate in order to minimize niche overlap and avoid competitive exclusion.



Character displacement forces niches apart.

# Niche partitioning enables organisms to avoid competitive exclusion

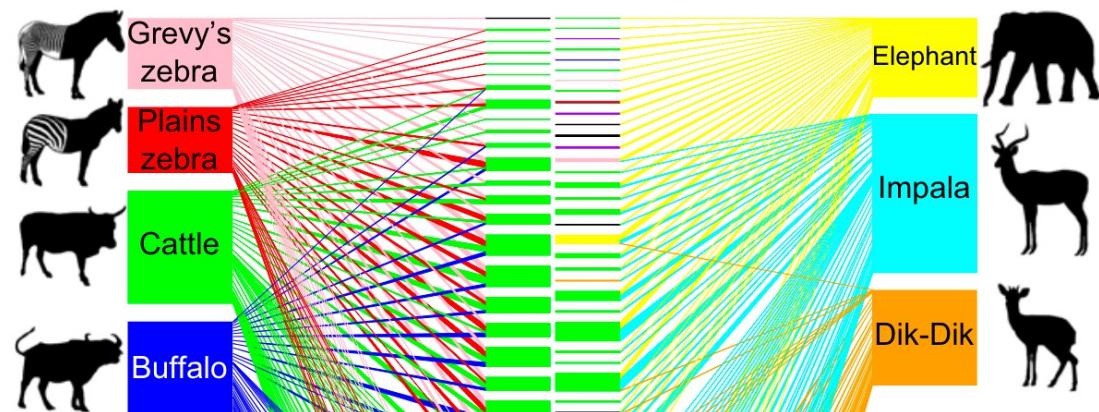
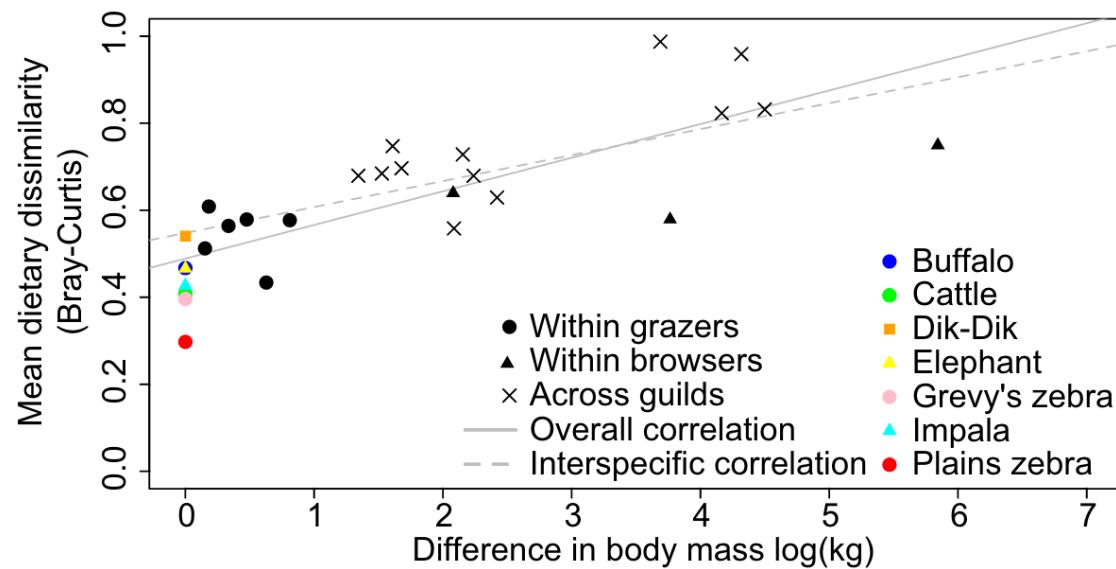
Often 10-25 large mammalian herbivores coexisting in the same African savanna!



Kartzin et al. 2015. PNAS.

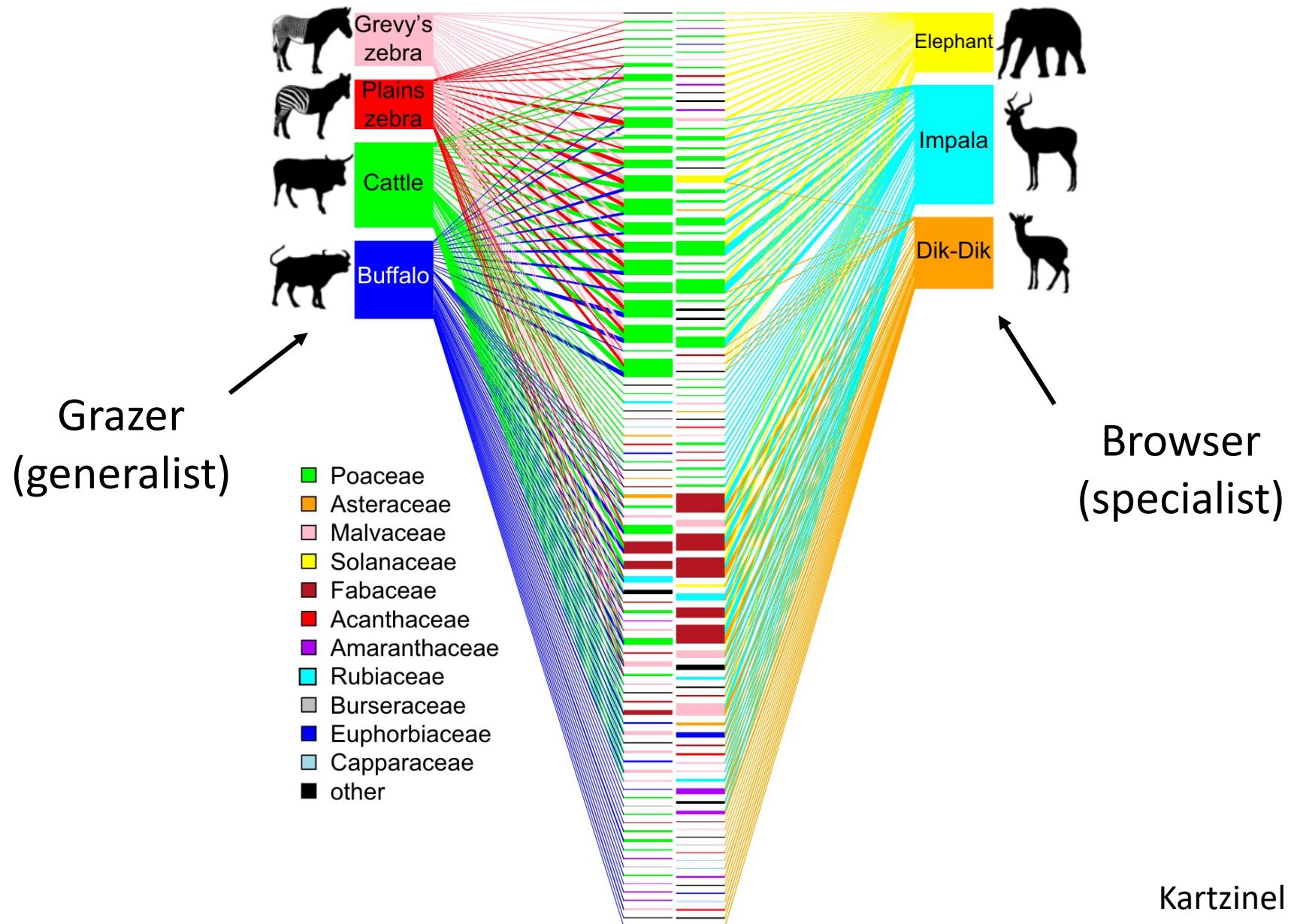
# Niche partitioning enables organisms to avoid competitive exclusion

Dietary overlap is higher in similar-sized mammals



- Poaceae
- Asteraceae
- Malvaceae
- Solanaceae
- Fabaceae
- Acanthaceae
- Amaranthaceae
- Rubiaceae
- Burseraceae
- Euphorbiaceae
- Capparaceae
- other

# Consumer resource partitioning in the African savanna



## Que. 1 "Complete competitors cannot coexist" unless:

- a. there is a precedence effect
- b. space, low dispersal, and stochasticity are important
- c. there is character displacement

## Que. 1 "Complete competitors cannot coexist" unless:

a. there is a precedence effect

0%

b. space, low dispersal, and stochasticity are important

0%

c. there is character displacement

0%

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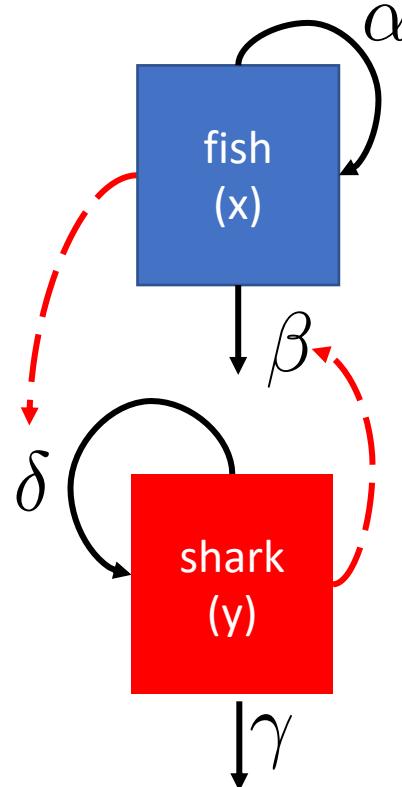
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0%

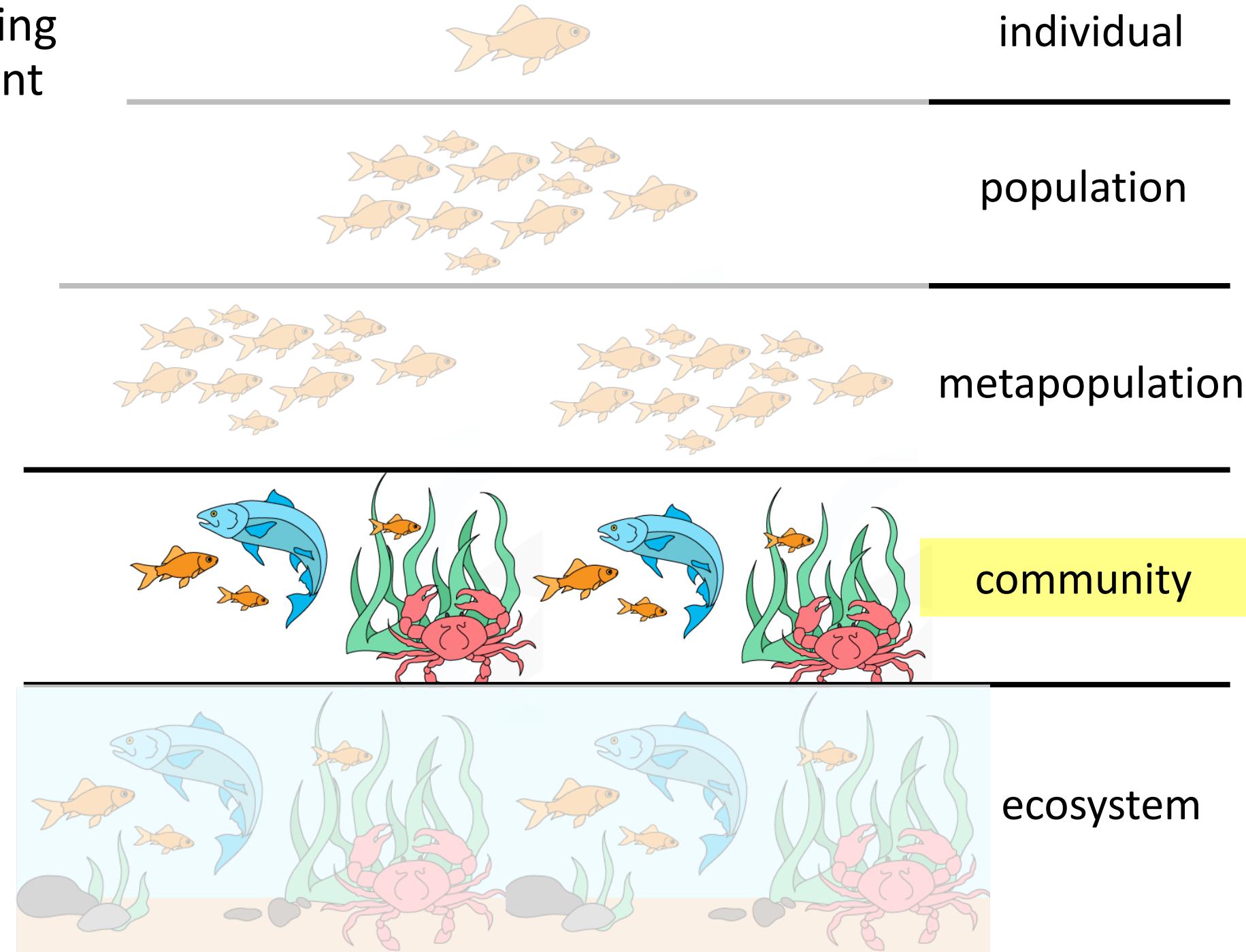
c. there is character displacement

0%

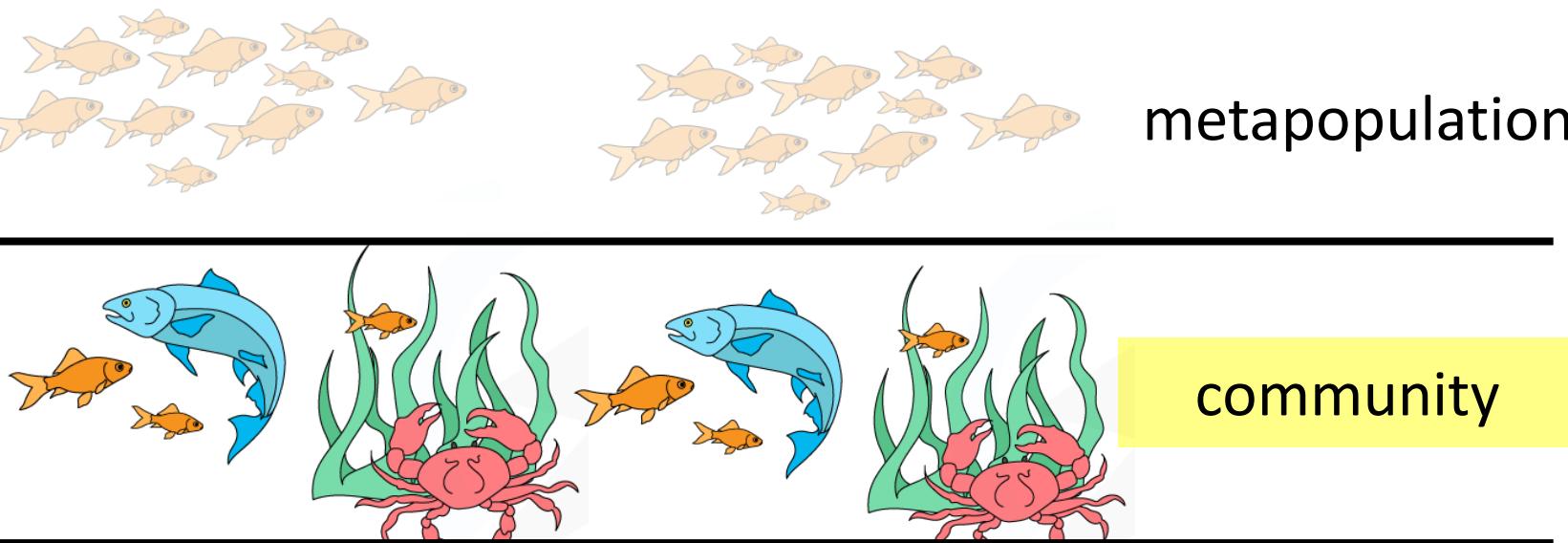
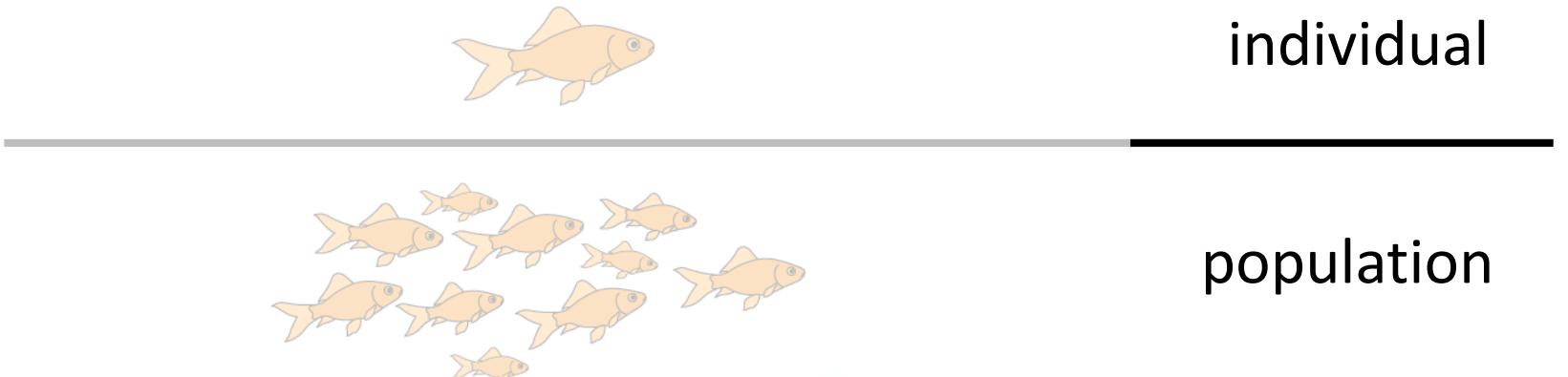
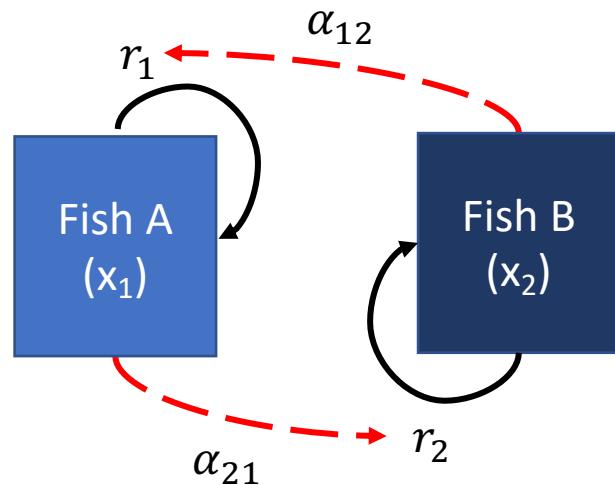
**Community** = interacting populations of different species



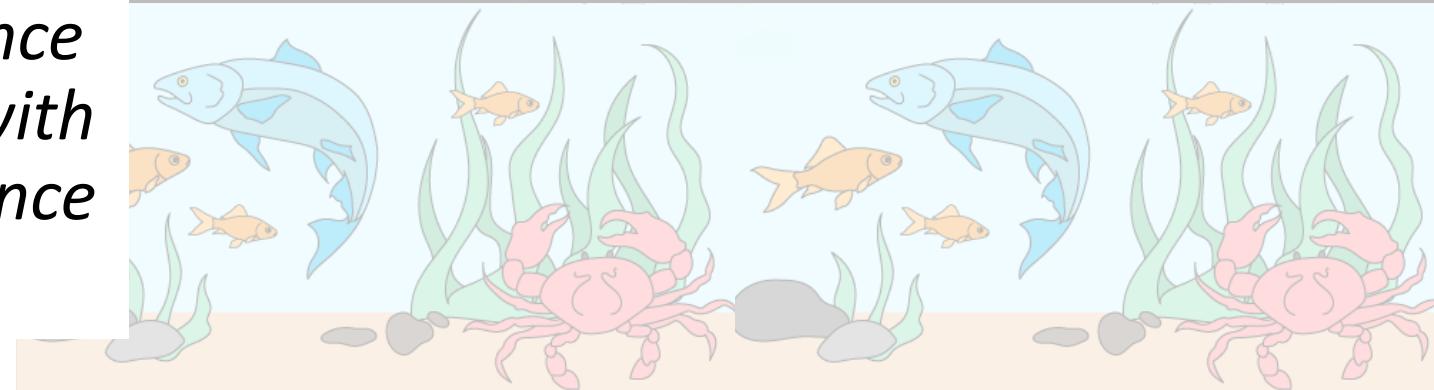
How does fish abundance **vary** with changes in shark abundance?



**Community** = interacting populations of different species

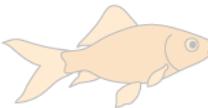


How does the abundance of **fish species A** vary with changes in the abundance of **fish species B**?

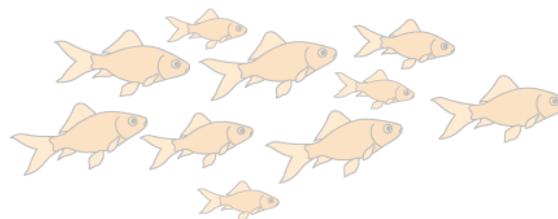


ecosystem

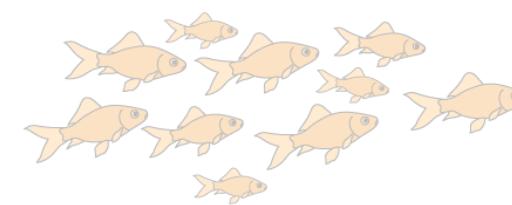
**Community** = interacting populations of different species



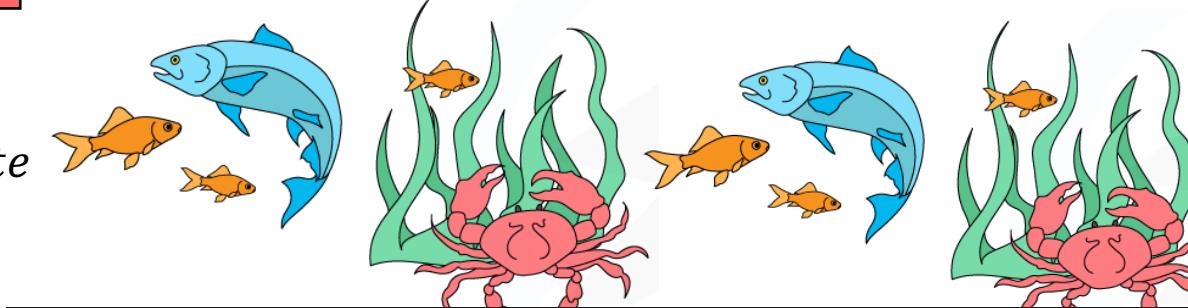
individual



population

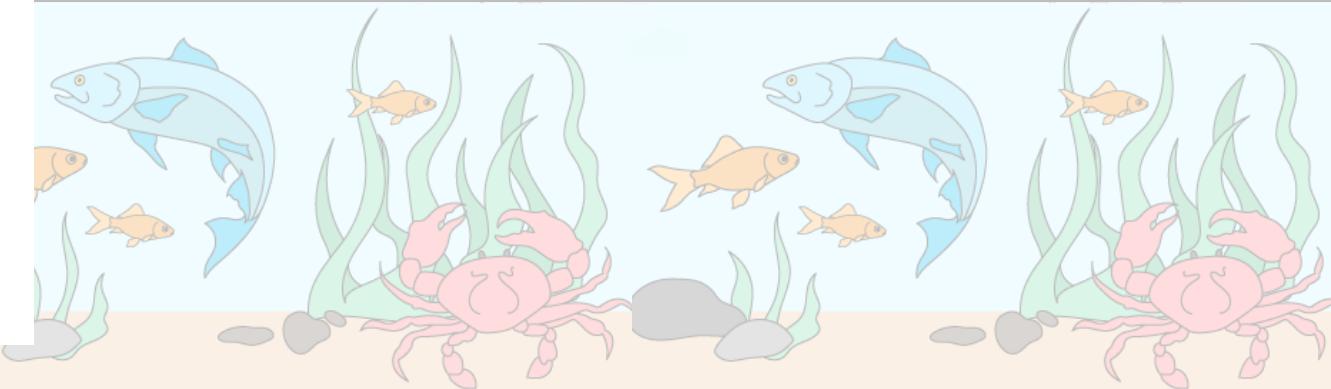


metapopulation



community

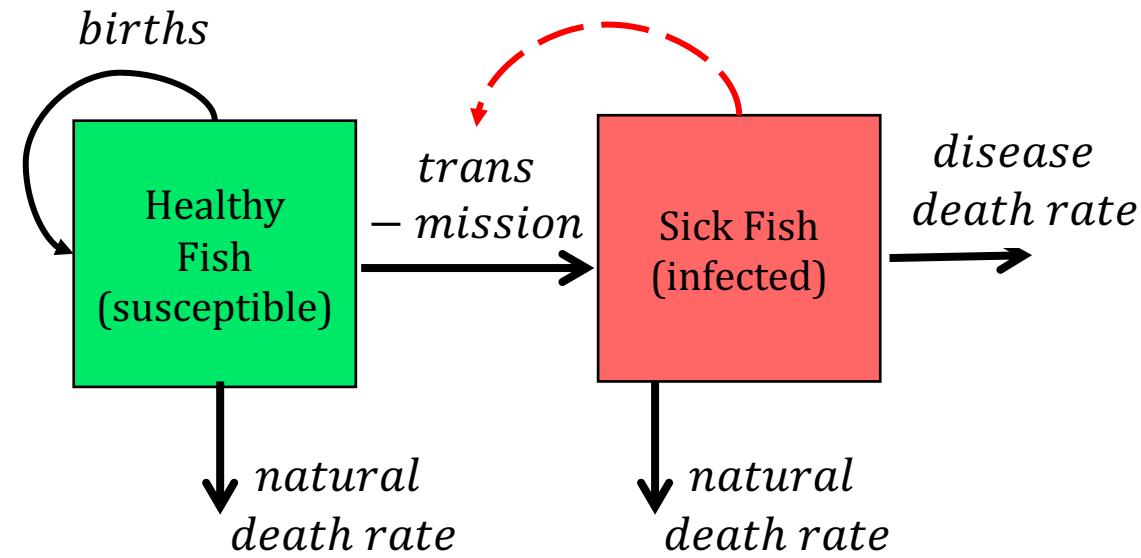
How does the abundance of **fish** change based on **infection with Mycobacterium marinum** (wasting disease)?



ecosystem

**Community** = interacting populations of different species

Species can interact in several distinct ways.



How does the abundance of **fish** change based on **infection with Mycobacterium marinum** (wasting disease)?

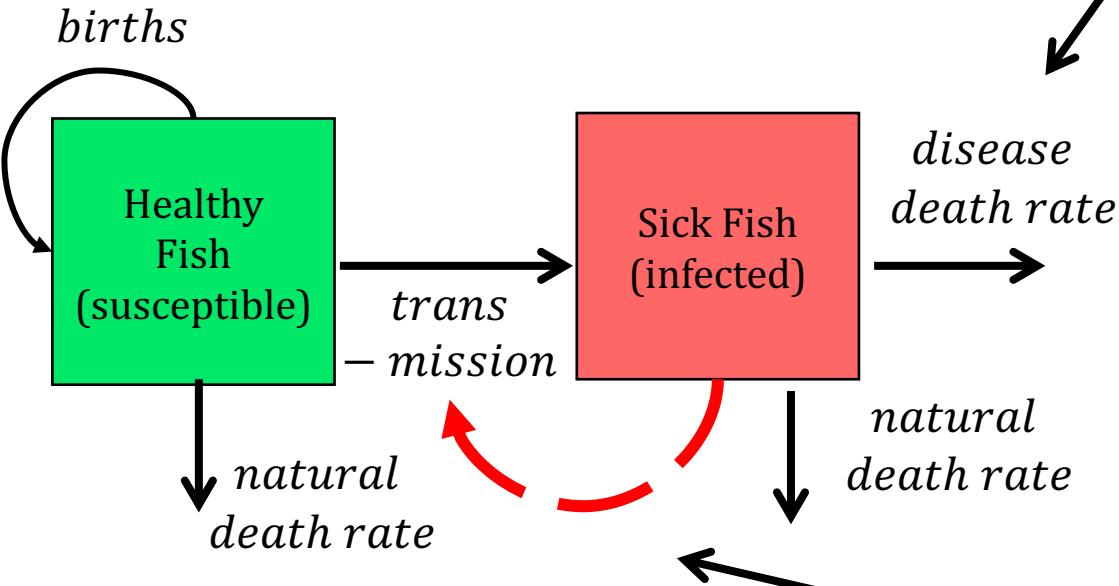
- **Mutualism** – both species benefit
- **Commensalism** – one species benefits, the other is unaffected
- **Predation** – one species benefits, the other is harmed (eaten!)
- **Competition** – two species compete for the same limiting resource, both harmed by the interaction
  - Direct = wolves and coyote at a moose carcass
  - Indirect = diurnal cheetah, nocturnal leopard at a giraffe carcass
- **Parasitism** – one species (the parasite) lives *in* or *on* the other species (the host)

# Population Biology

Each box is a distinct population!

When we model these populations, everyone in the box is considered the same.

In a continuous time model (ODE), each box would get its own differential equation!

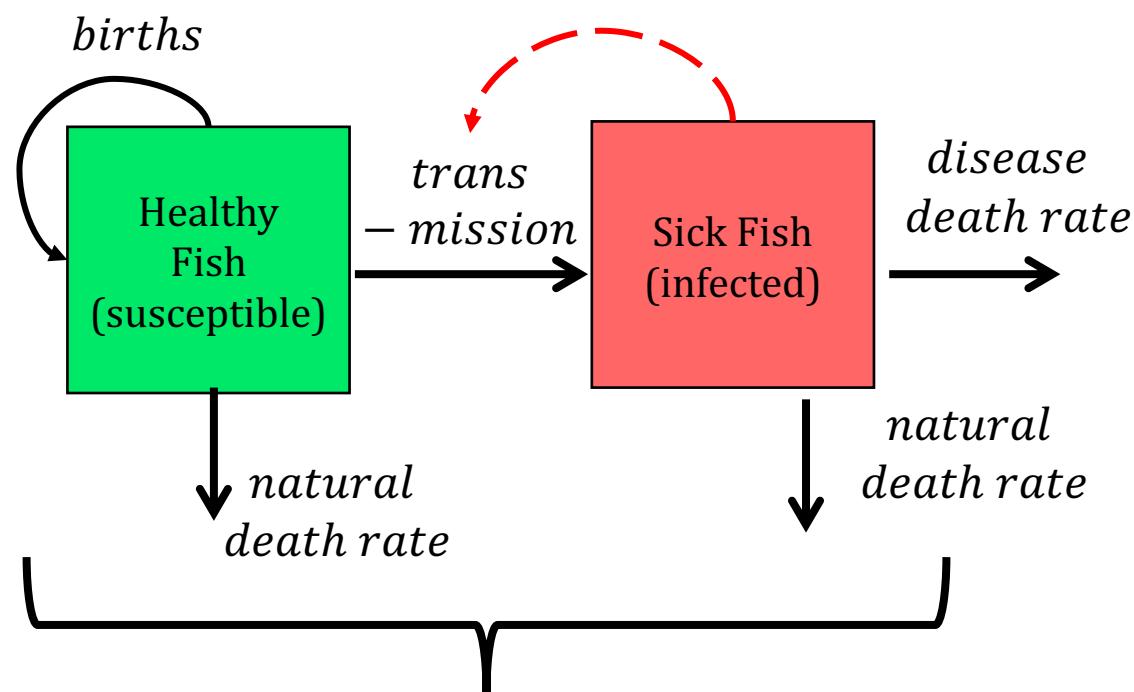


*How does the abundance of **fish** change based on **infection with Mycobacterium marinum** (wasting disease)?*

Solid lines are processes by which individuals in each population move between boxes.

Dashed lines are influences of populations on rates (transmission is higher when there are more sick fish)

# *Population Biology*



In disease ecology, we model populations based on their **infection status**.

*How does the abundance of fish change based on infection with **Mycobacterium marinum** (wasting disease)?*

Previously, we modeled populations of **different species**, or of **distinct life history classes within a species**.

# *Population Biology*

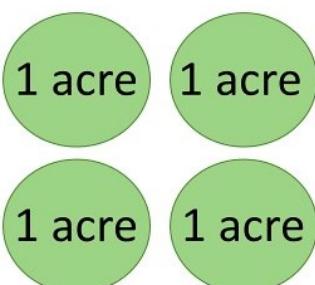
## Conservation Biology

- Goal:
  - protect **populations** from extinction
- Concept:
  - **Minimum Viable Population** size (MVP)
- Approach:
  - protected area **reserves**

Single Large

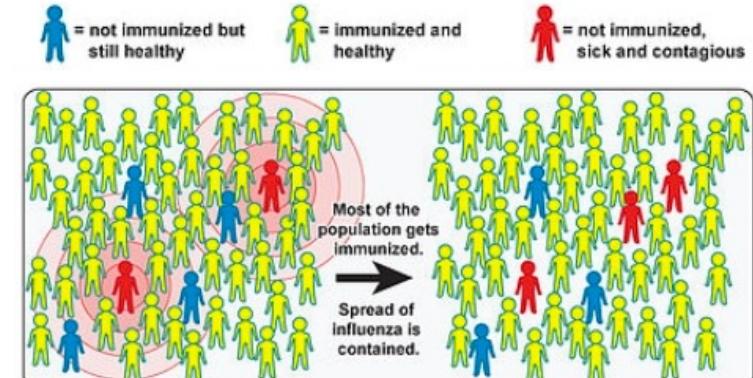


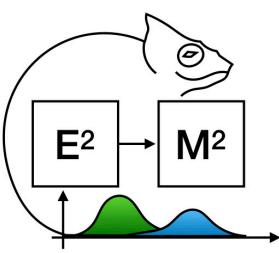
Several Small



## Disease Ecology

- Goal:
  - protect **populations** from disease via pathogen **extinction**
- Concept:
  - **Critical Community Size** (CCS)
- Approach:
  - sanitation
  - **vaccination**





# E<sup>2</sup>M<sup>2</sup>: Ecological and Epidemiological Modeling in Madagascar



December 16, 2022  
[E2M2.org](http://E2M2.org)

# Parasites and Pathogens

- Parasite: an organism that lives in or on another organism and benefits at the expense of others.
  - Ex: helminths (parasitic worms: tapeworms, roundworms, hookworms), ectoparasites (ticks, fleas)



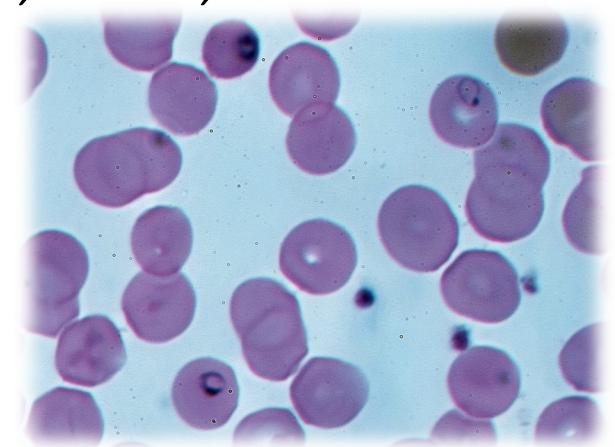
# Parasites and Pathogens

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- Pathogen: a microorganism that can cause disease
  - Ex: bacterium, virus, protozoan

Pathogen: *Yersinia pestis*  
Disease: Plague

Pathogen: SARS-CoV-2  
Disease: COVID-19

Pathogen: *Plasmodium falciparum, P. vivax, P. malariae, P. ovale, P. knowlesi*  
Disease: malaria



# Parasites and Pathogens

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  - Ex: bacterium, virus, protozoan
- Host: the organism on/in which the parasite/pathogen lives
  - Ex: Soay sheep, grapes

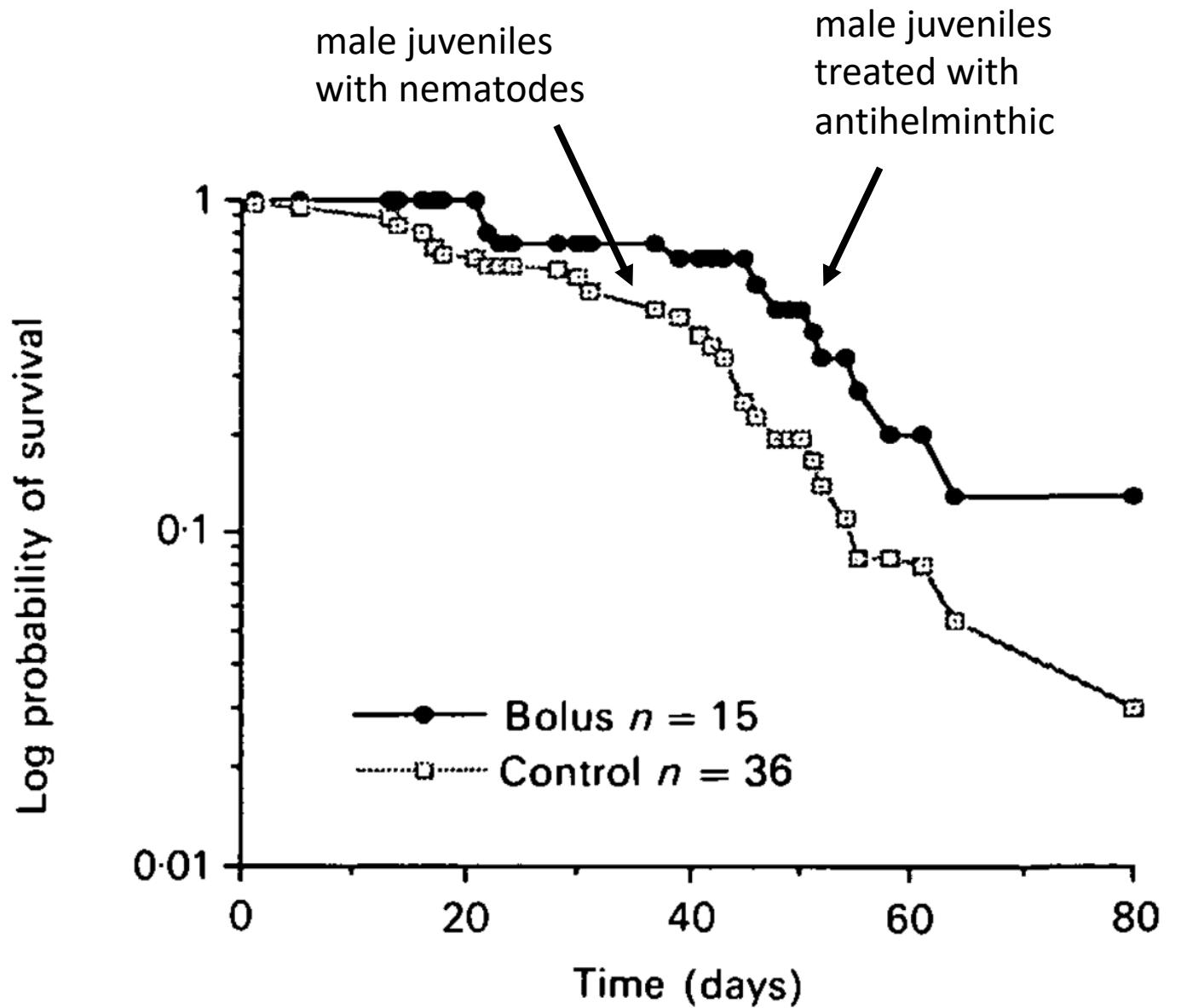
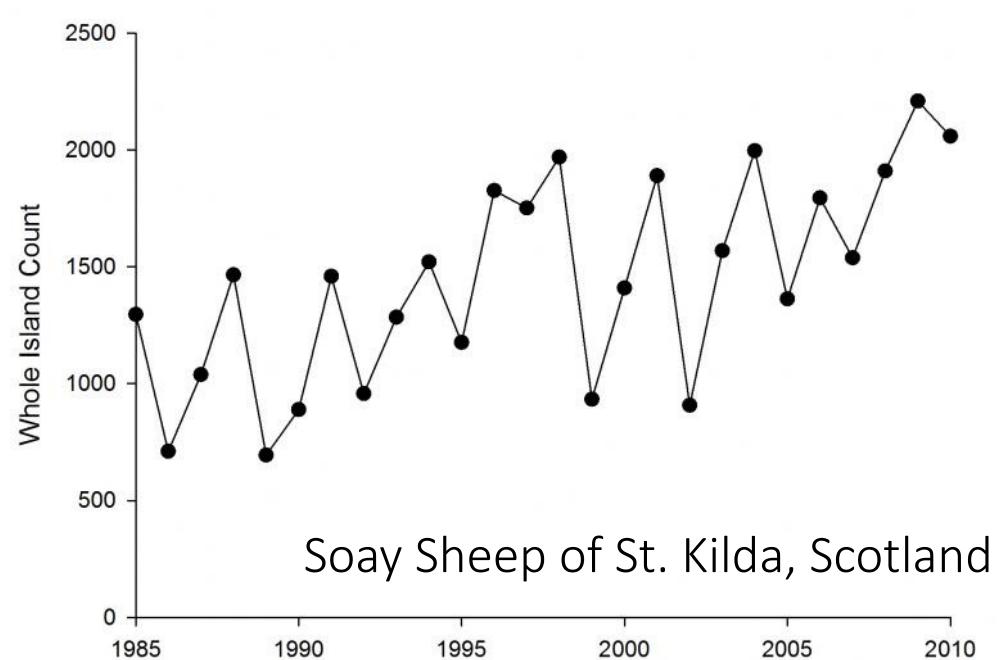


# Parasites and Pathogens

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- Pathogen: a microorganism that can cause disease
  - Ex: bacterium, virus, protozoan
- Host: the organism on/in which the parasite/pathogen lives
  - Ex: Soay sheep, grapes
- Vector: an arthropod agent that carries and transmits a pathogen or parasite from host to host
  - Ex: mosquitoes, ticks, fleas



We already know that  
parasites can play a role in  
regulating populations!



Gulland 1992. *Parasitology*.

## Que 2: What factors do we know contribute to annual fluctuations in the population density of the Soay sheep of St Kilda?

A Density-dependent effects on lamb survival

B Higher mortality in bad weather years

C Higher ewe mortality at high population sizes

D Lowered survival in juveniles with high worm count

E Geometric growth

F A, B

G A, B, C, D, E

## Que 2: What factors do we know contribute to annual fluctuations in the population density of the Soay sheep of St Kilda?

A Density-dependent effects on lamb survival 0%

B Higher mortality in bad weather years 0%

C Higher ewe mortality at high population sizes 0%

D Lowered survival in juveniles with high worm count 0%

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F A, B 0%

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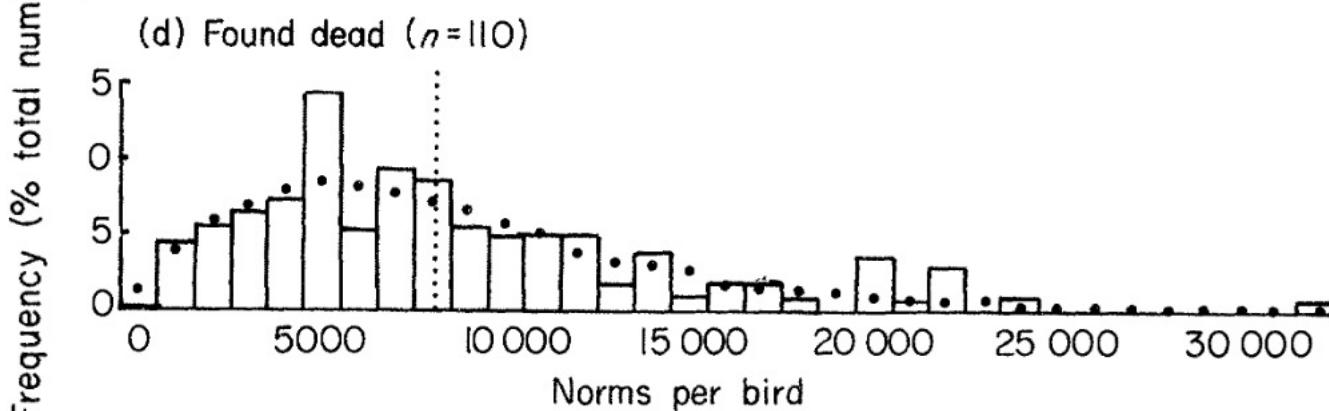
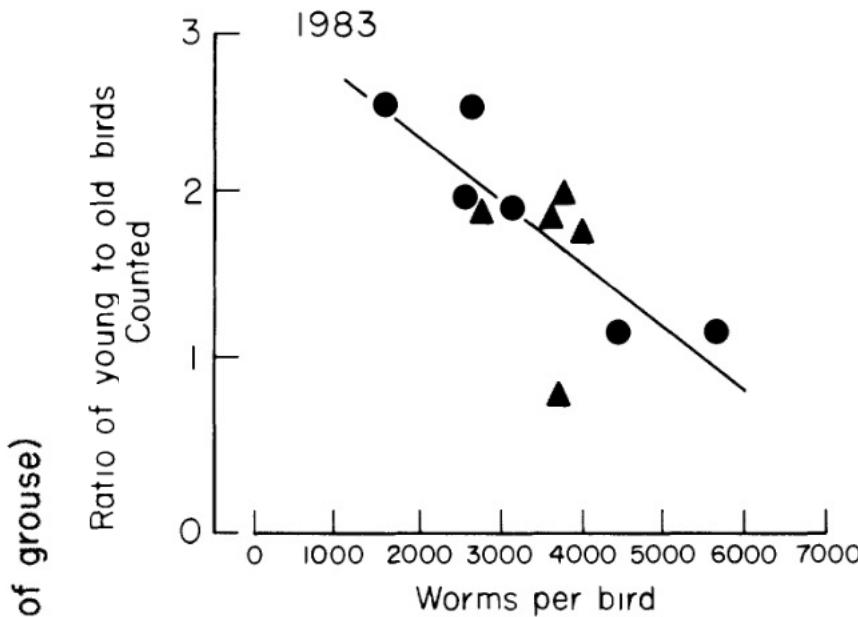
G A, B, C, D, E 0%

Early work in red grouse experimentally demonstrated the power of parasites to regulate populations.



Hudson 1986. *J Animal Ecology*.  
Hudson et al 1992. *J Animal Ecology*.  
Hudson et al 1998. *Science*.

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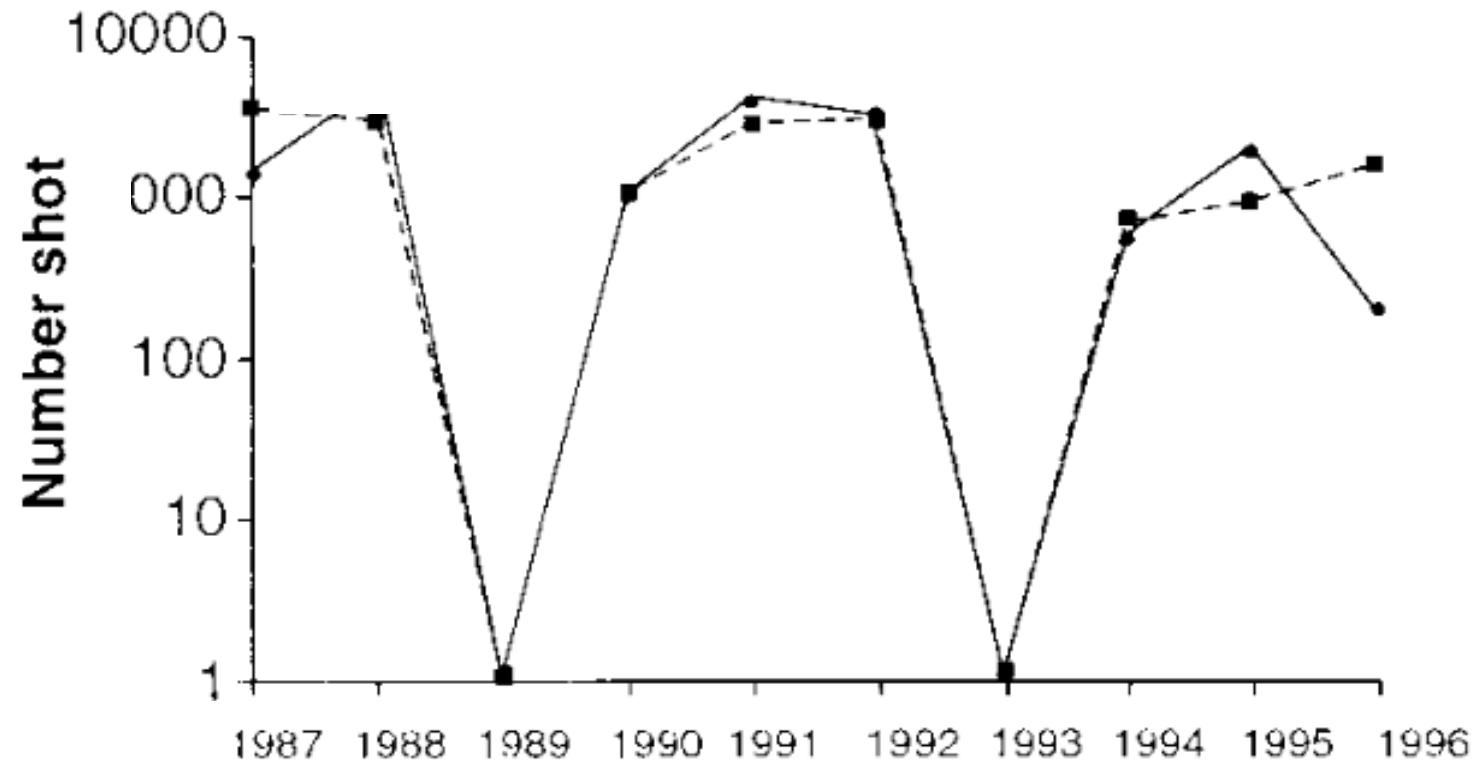


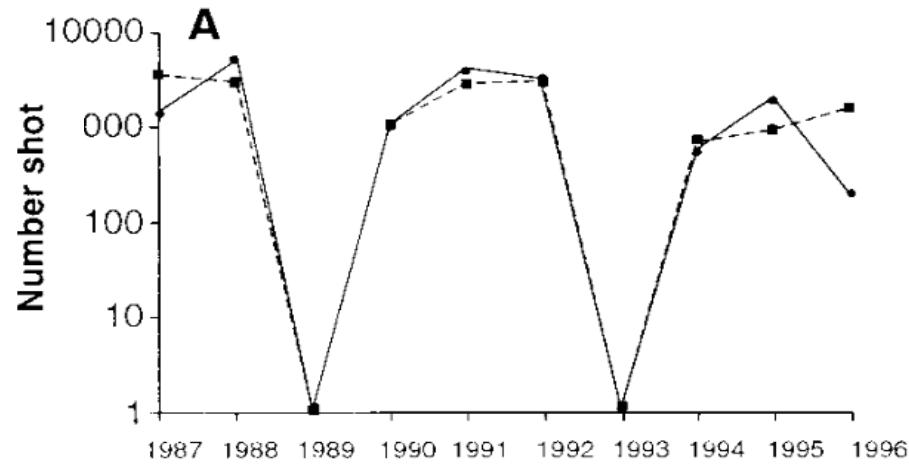
Higher burden of the intestinal strongyle worm, *Trichostrongylus tenuis*, both reduces breeding success and increases mortality in red grouse.

Hudson 1986. *J Animal Ecology*.

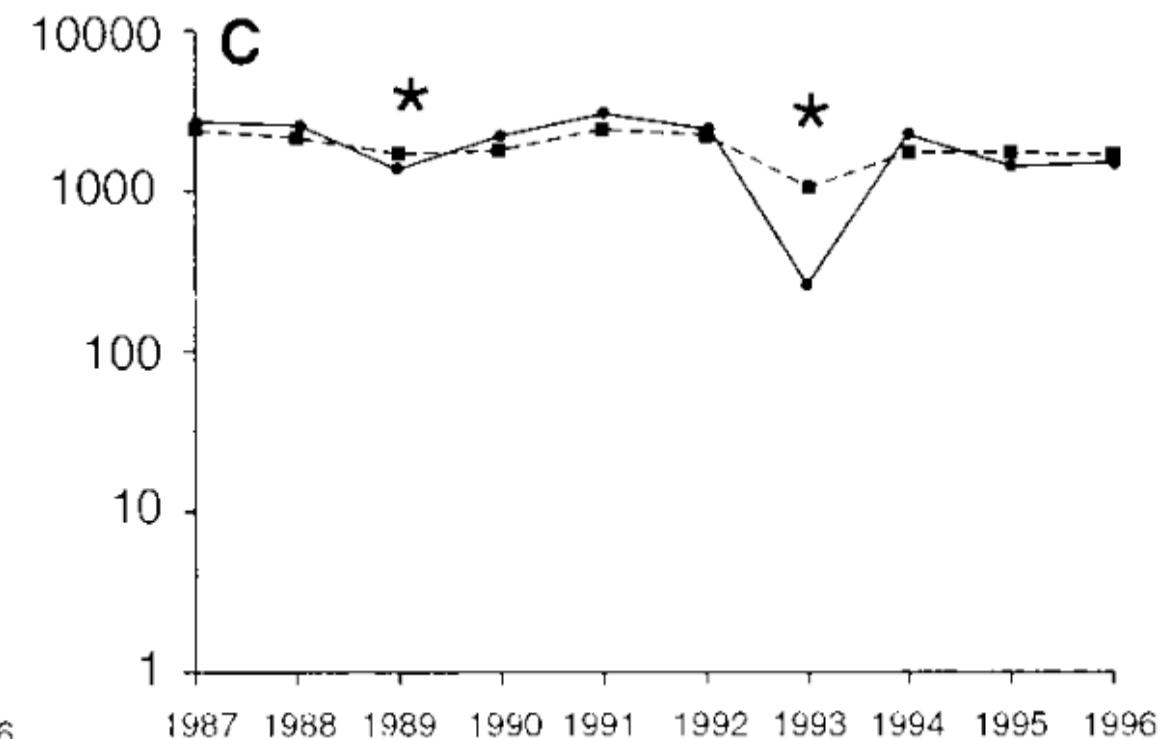
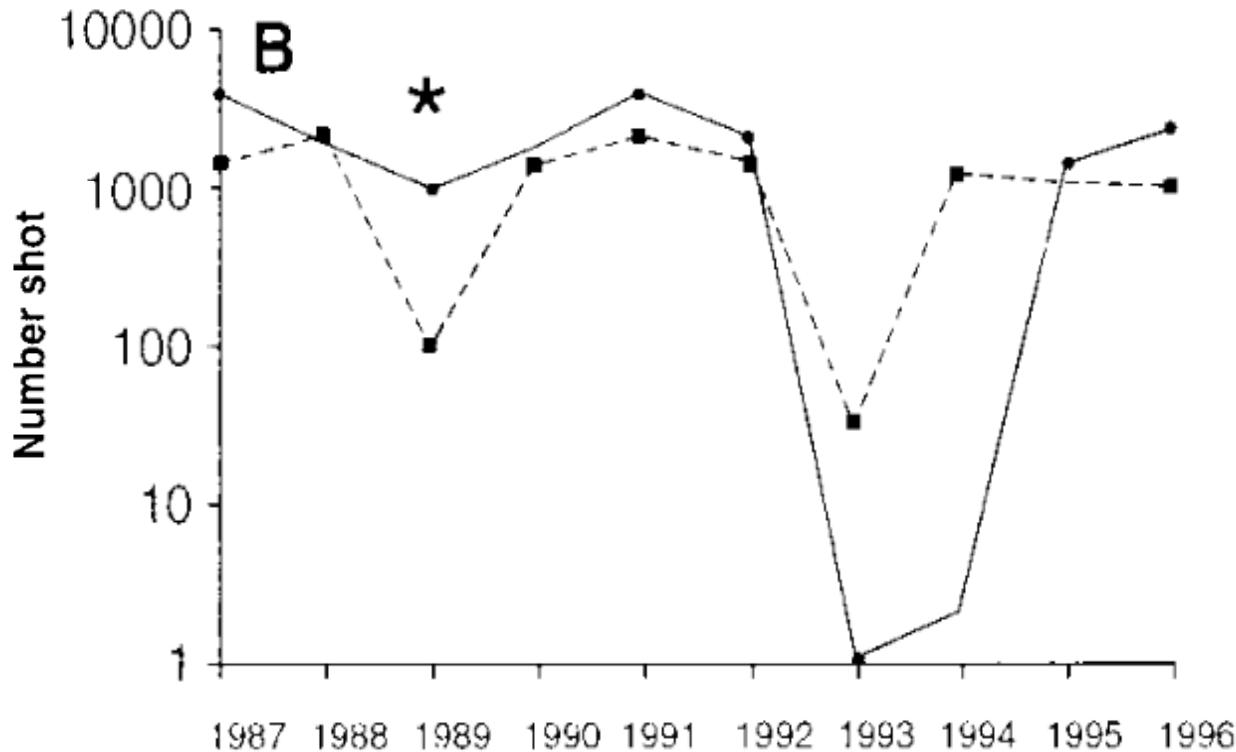
Hudson et al 1992. *J Animal Ecology*.

Worms were hypothesized to be responsible for the observed population cycles in ‘bag data’ from northern England.





Deworming eliminated population cycles to prove this effect!

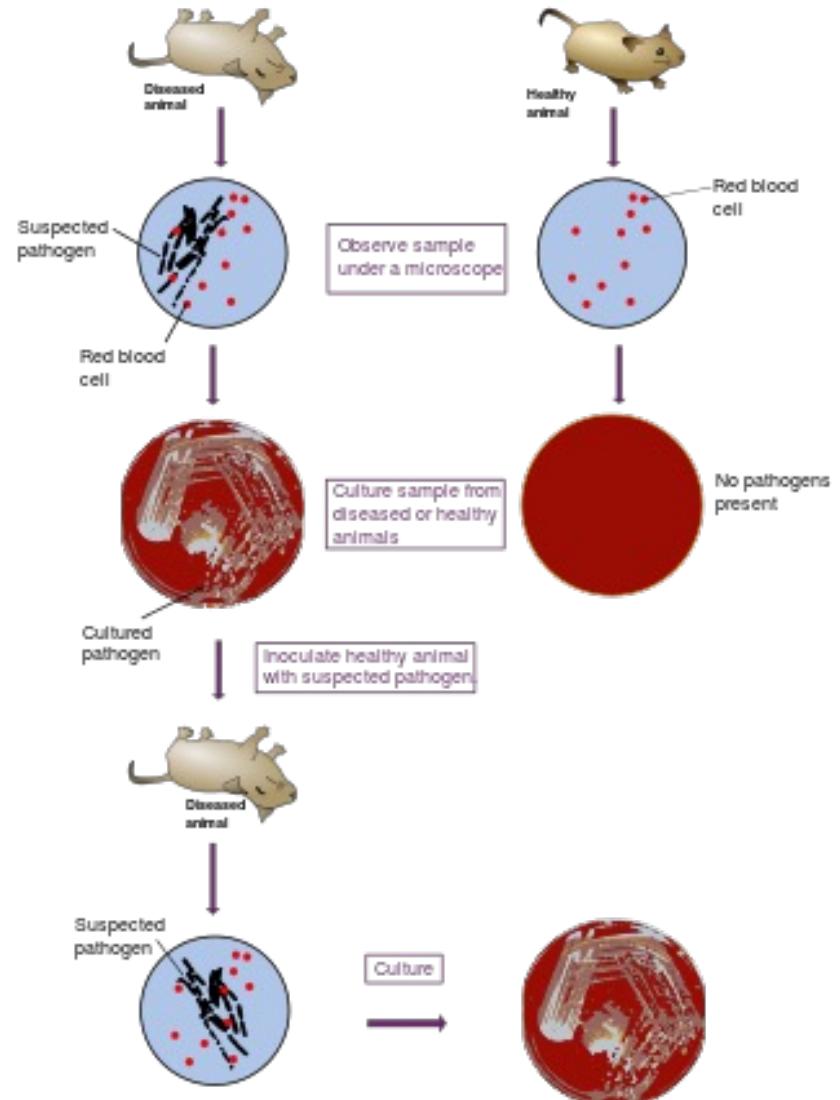


Parasites and pathogens have also shaped human history.

# Parasites and pathogens have also shaped human history.

## But we have not always known they were responsible for disease!

1. **Four Humors:** Hippocrates (c. 400 BC) wrote that disease results from an imbalance of the four humors
2. **Miasmatic Theory:** Extension of Hippocrates that lasted through the 1800s
  - idea that disease was caused by bad air.
  - Popularized by Florence Nightengale
3. **Germ Theory of Disease:** Idea that disease results from germs
  - Leuwenhoek's microscope (1675)
  - Koch's postulates (1890)



Parasites and pathogens have also shaped human history.

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4. **Classic epidemiology**

- Risk factors for disease = John Snow (1854)



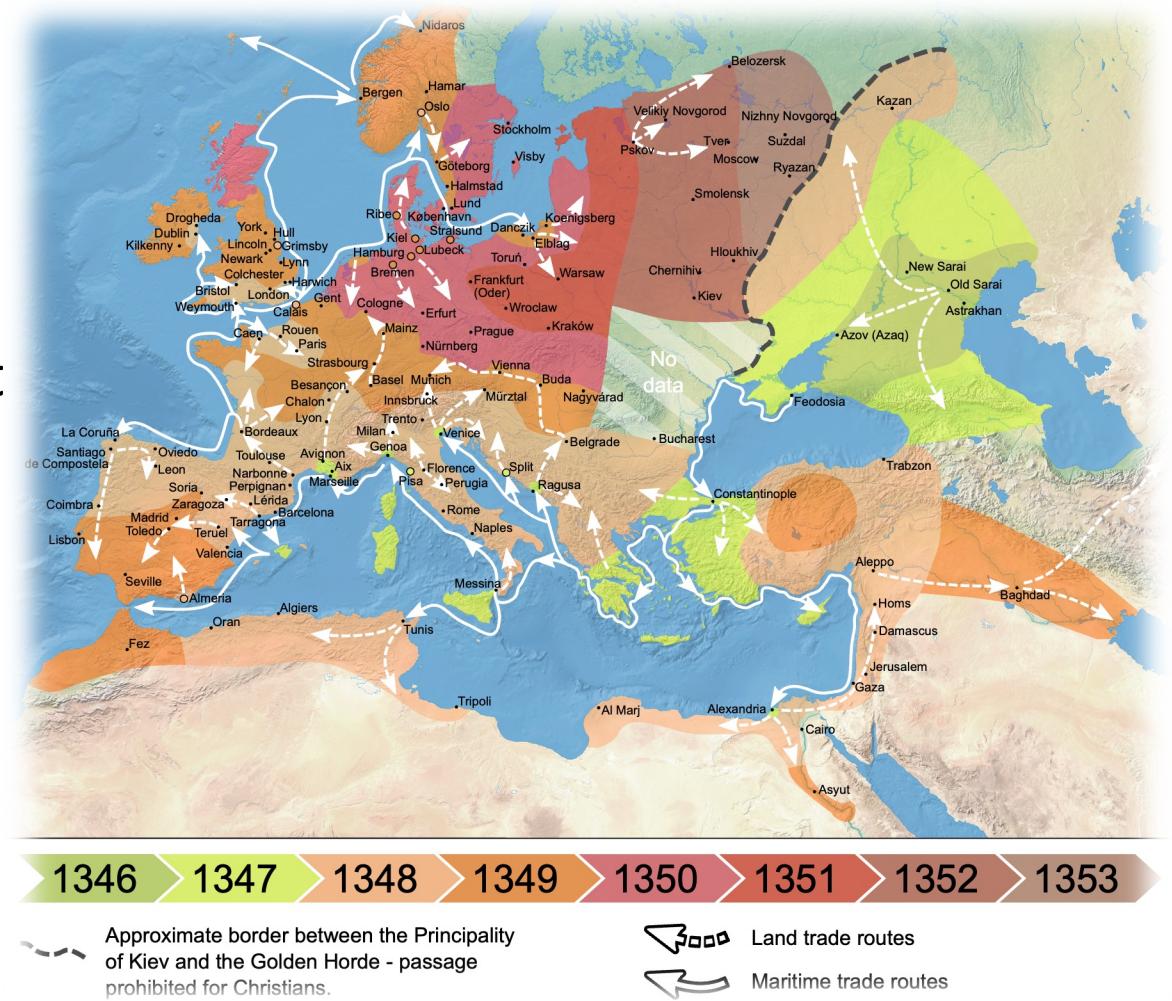
# Parasites and pathogens have also shaped human history.

- Plague of Justinian (541-549 AD)
  - First historically recorded pandemic of *Yersinia pestis*
  - Launched the ‘first plague pandemic’ resulting in the deaths of 15-100 million people, 25-60% of Europe’s population at the time



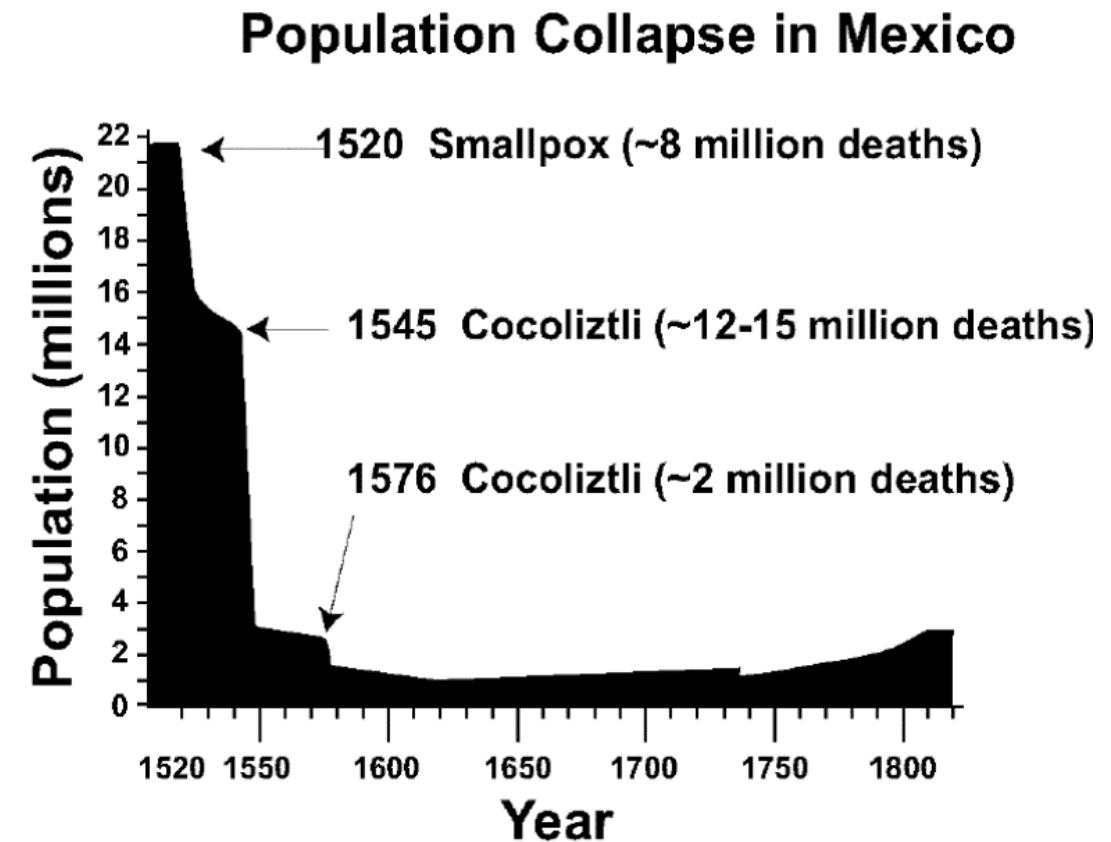
# Parasites and pathogens have also shaped human history.

- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
  - Most fatal pandemic in human history, resulting in deaths of 75-200 million people
  - Killed 30-60% of Europe's population at the time; 17-54% of global population



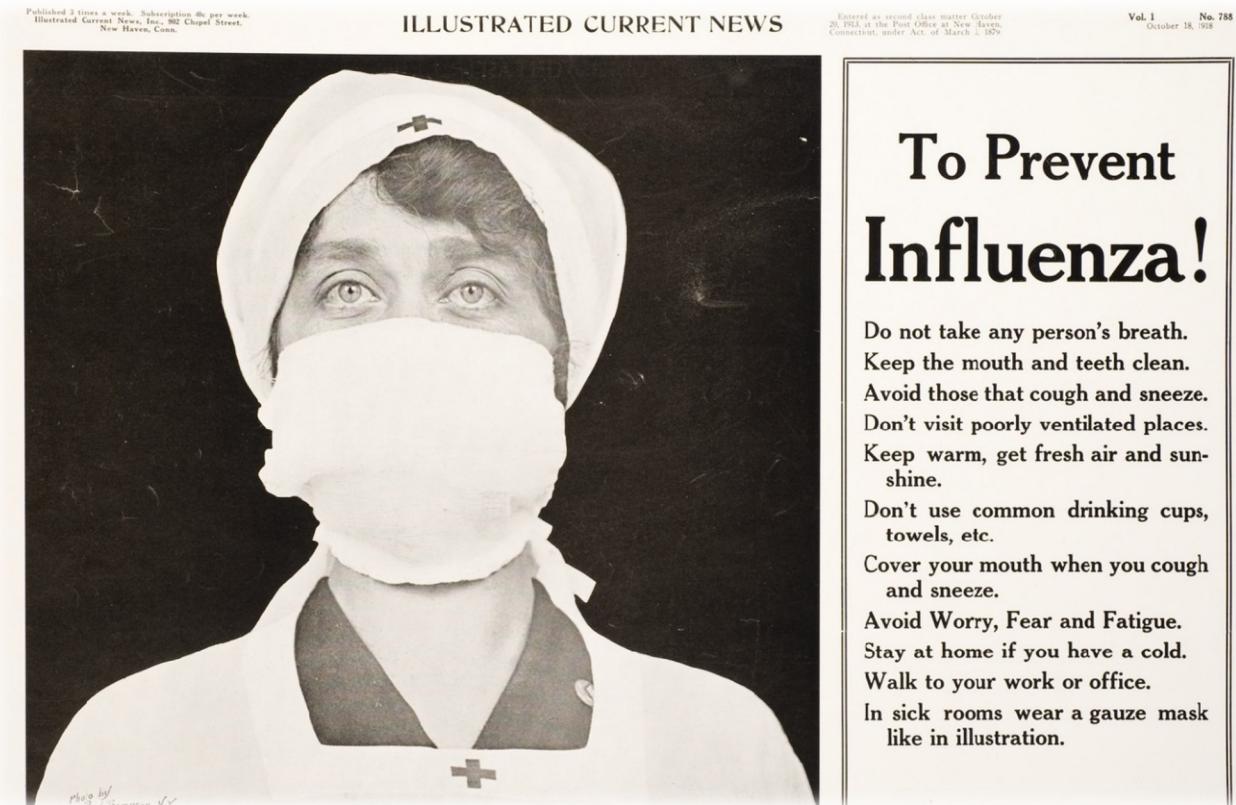
# Parasites and pathogens have also shaped human history.

- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
  - Pathogen still unknown! Maybe viral hemorrhagic fever, maybe bacterium
  - Killed 80% of the population of Mexico



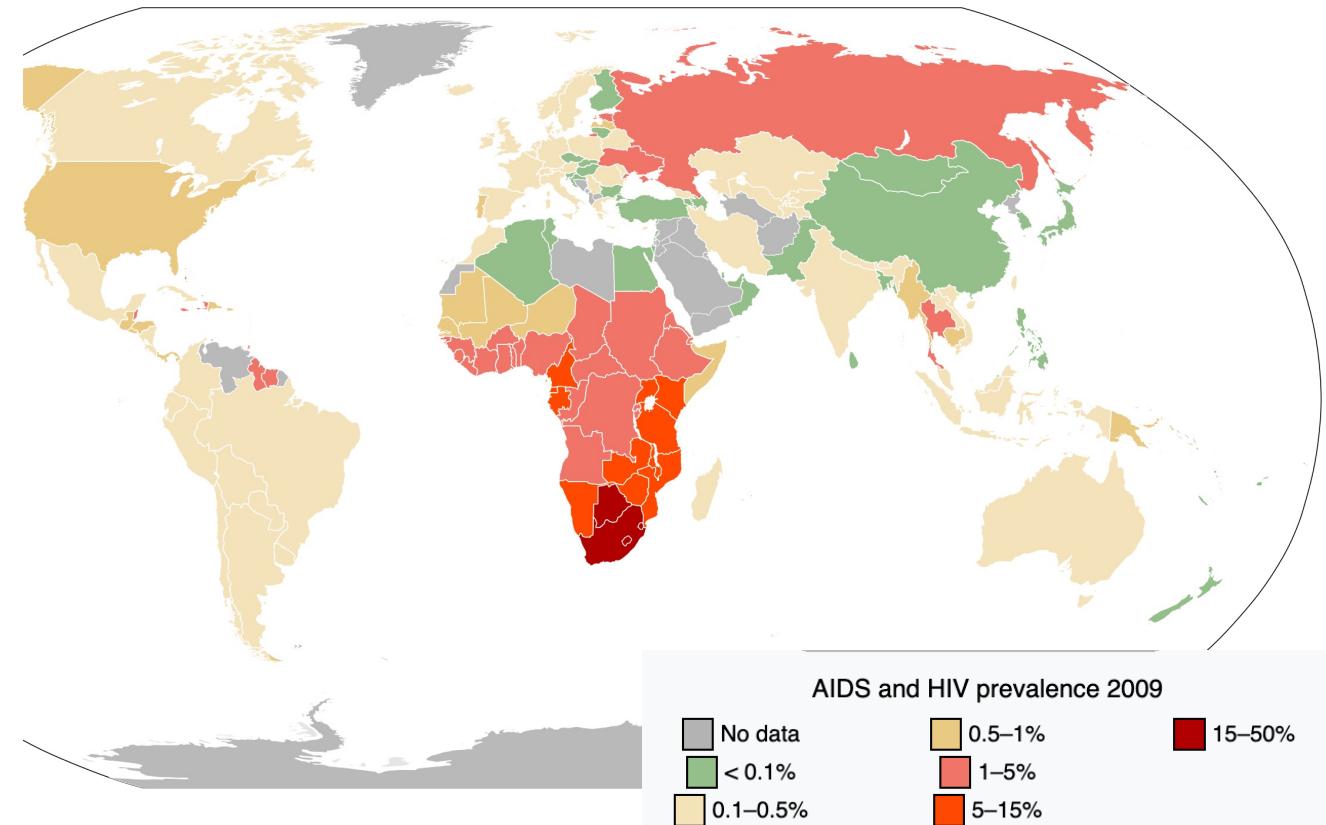
# Parasites and pathogens have also shaped human history.

- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
- Spanish Influenza (1918-1920)
  - 17-100 million deaths worldwide.
  - 1-5% of global population
  - 2<sup>nd</sup>-most devastating pandemic in history (after Black Death)



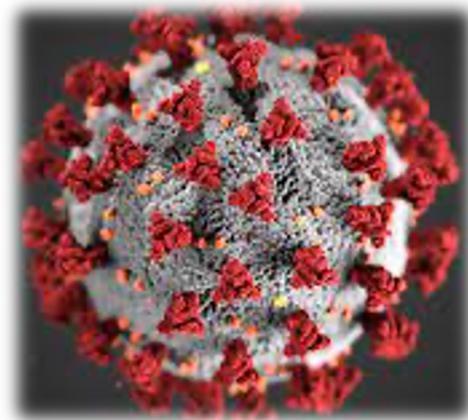
# Parasites and pathogens have also shaped human history.

- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
- Spanish Influenza (1918-1920)
- HIV (~1960-now)
  - >40 million deaths and counting
  - Prevalence still >20% in some countries in southern Africa



# Parasites and pathogens have also shaped human history.

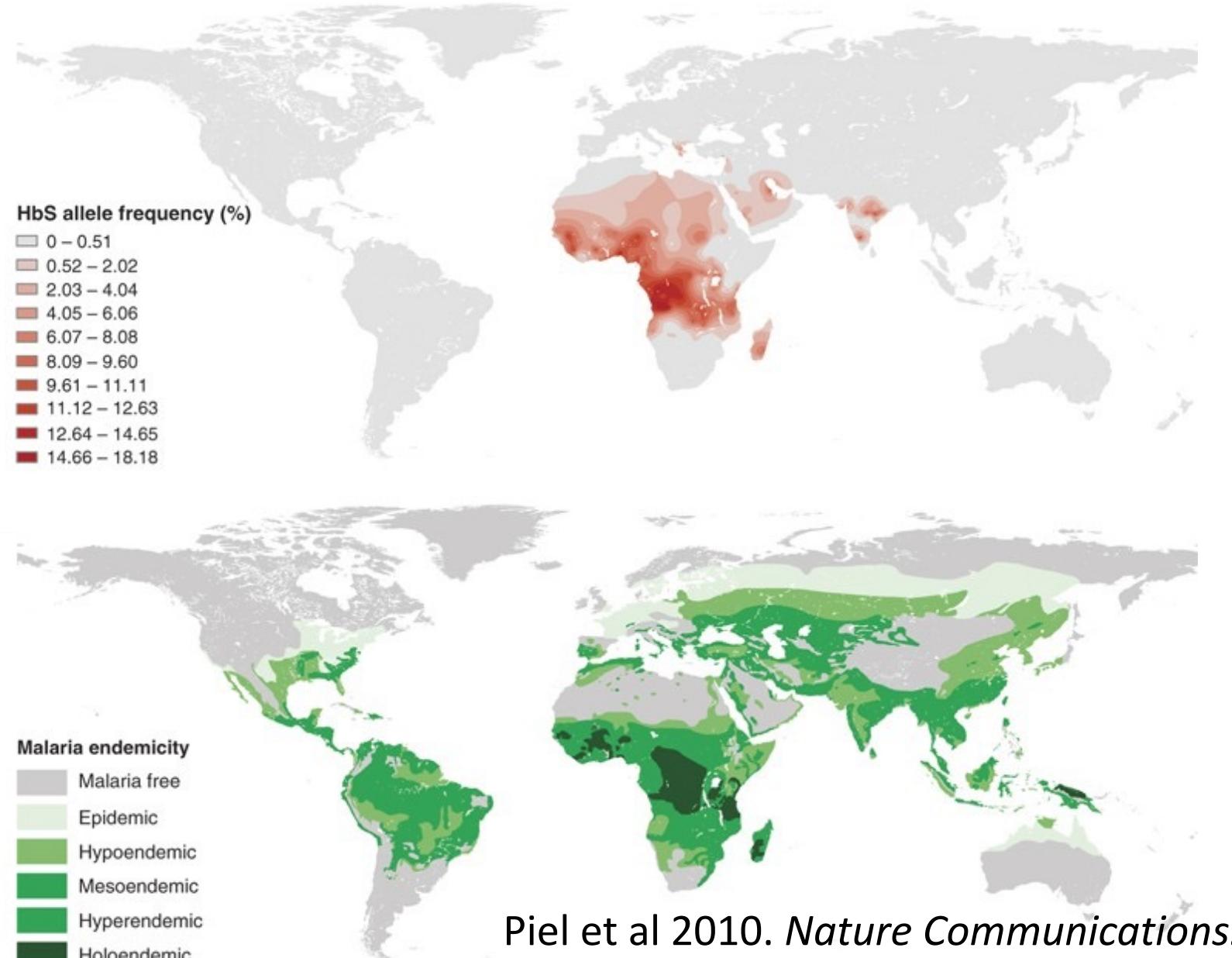
- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
- Spanish Influenza (1918-1920)
- HIV (~1960-now)
- COVID-19 (2019-now)
  - ~7-29 million deaths worldwide
  - ~0.1-0.4% of population



# Parasites and pathogens have also shaped human DNA.

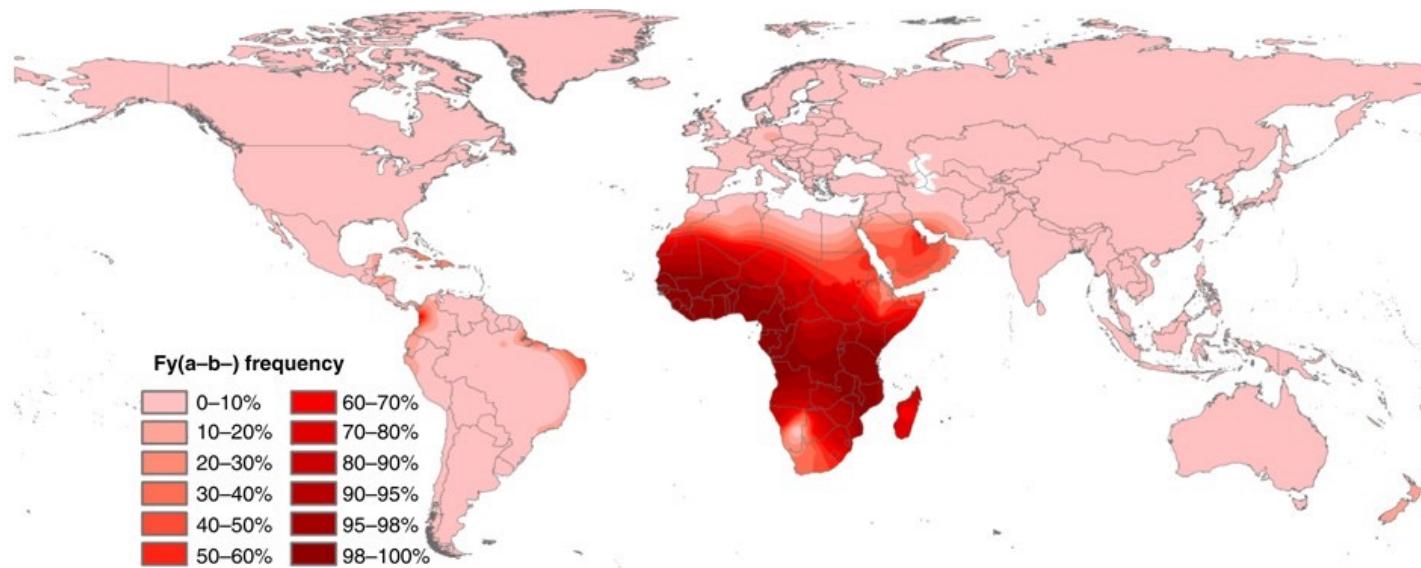
## Sickle cell anemia

- The HbS allele confers resistance to malaria but also results in sickle cell anemia when homozygous.
- Natural selection has favored this trait in malaria-endemic regions of the planet.
- As of 2021, WHO estimates 247 million malaria cases worldwide and >600,000 deaths, 95% in Africa.
- Children <5 account for 80% of malaria deaths.



Parasites and pathogens have also shaped human DNA.

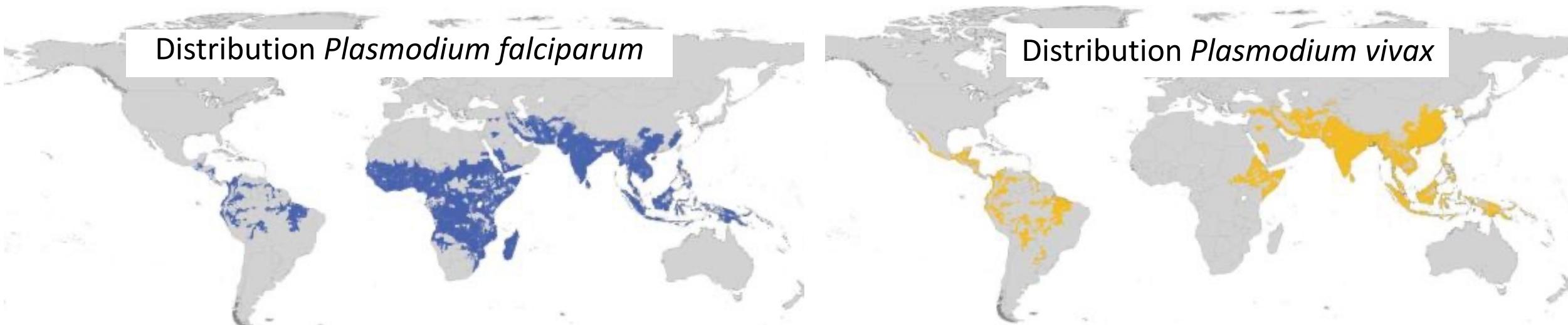
## Duffy antigen



- Modeled distribution of Duffy-negative human population

Distribution *Plasmodium falciparum*

Distribution *Plasmodium vivax*



Guerra et al. 2006. *Trends in Parasitology*  
Howes et al 2011. *Nature Communications*.

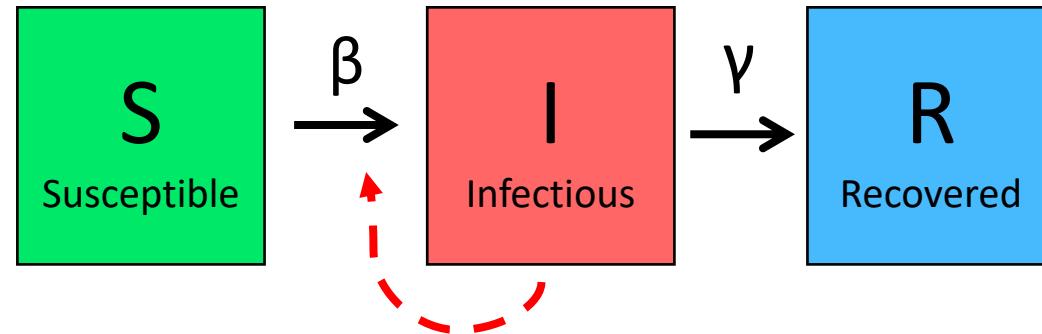
# Epidemiology vs. Disease Ecology

---

- Epidemiology = “the study of **what** is on the people”
  - Coined by Spanish physician Villalba in 1802
- Emphasis on the study and analysis of the **distribution and determinants** of health and disease (“risk factors”)
  - Including chronic diseases!
- Often uses **cross-sectional** data to demonstrate associations of variables with outcome (disease)
- More statistical ( $y=mx+b$ )
  - **Pattern**

- Disease Ecology = the study of **how** a disease spreads
  - Emphasis on the **interactions** of organisms with each other and the environment...when interactions result in disease
- Emphasis on understanding the **transmission dynamics** of infectious disease
- Typically involves fitting dynamical (population) models to **time series** data
- More mathematical ( $dN/dt$ )
  - **Process**

# The SIR Model

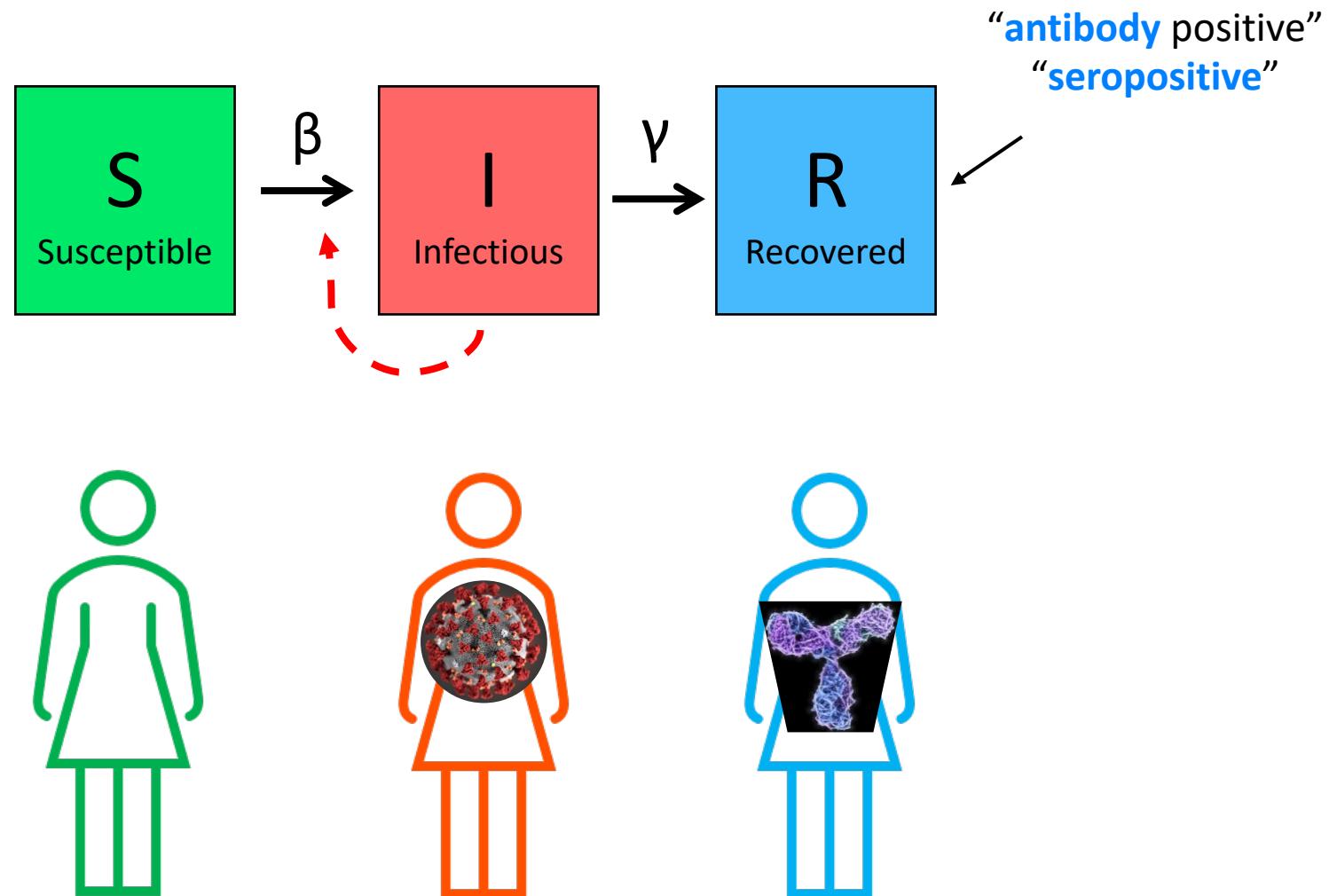


$\beta$  = transmission rate

$\gamma$  = recovery rate

Kermack and McKendrick 1927 *Proc Roy Soc A*

We class hosts into categories of **susceptible**, **infectious**, and **recovered** to model **pathogen dynamics**.



$\beta$  = transmission rate  
 $\gamma$  = recovery rate

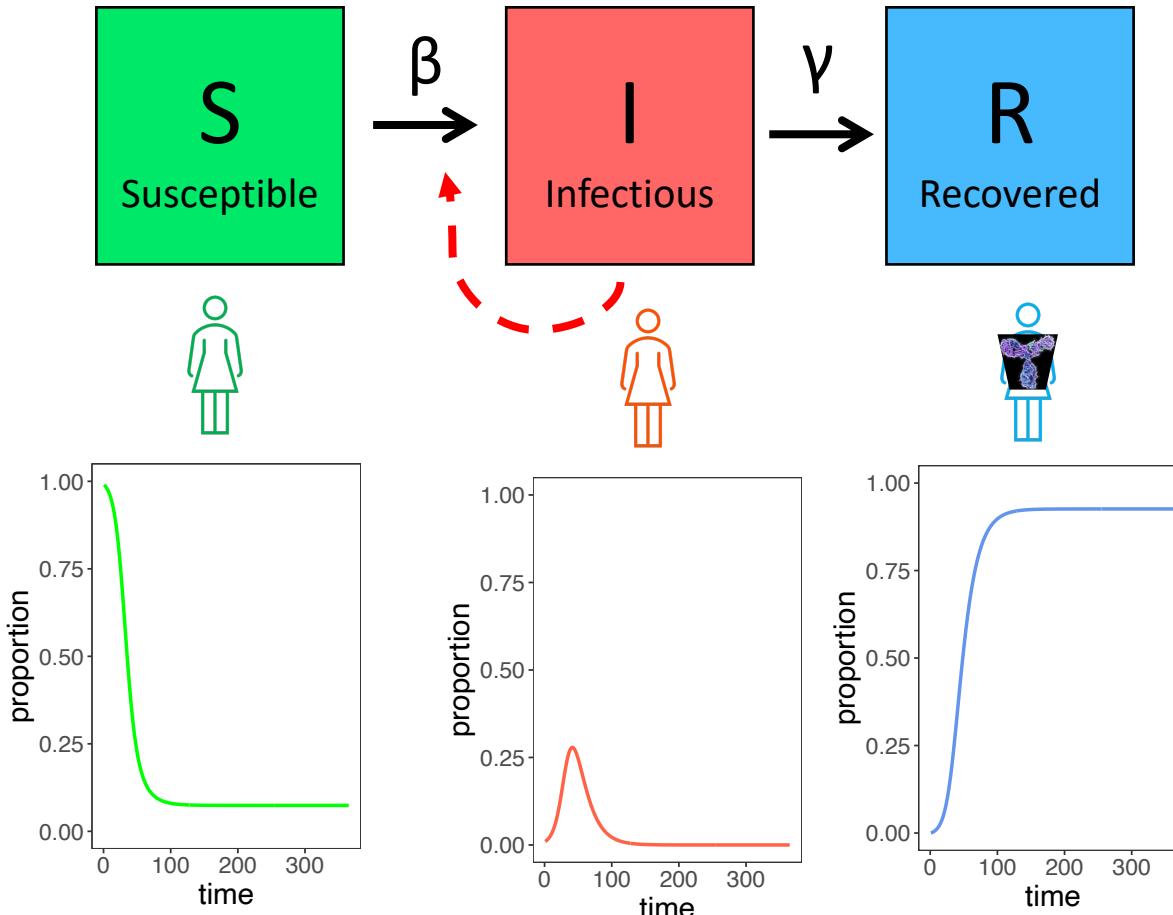
Kermack and McKendrick 1927 *Proc Roy Soc A*

We use computers to simulate systems of equations in the SIR framework.

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

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

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



$\beta$  = transmission rate

$\gamma$  = recovery rate

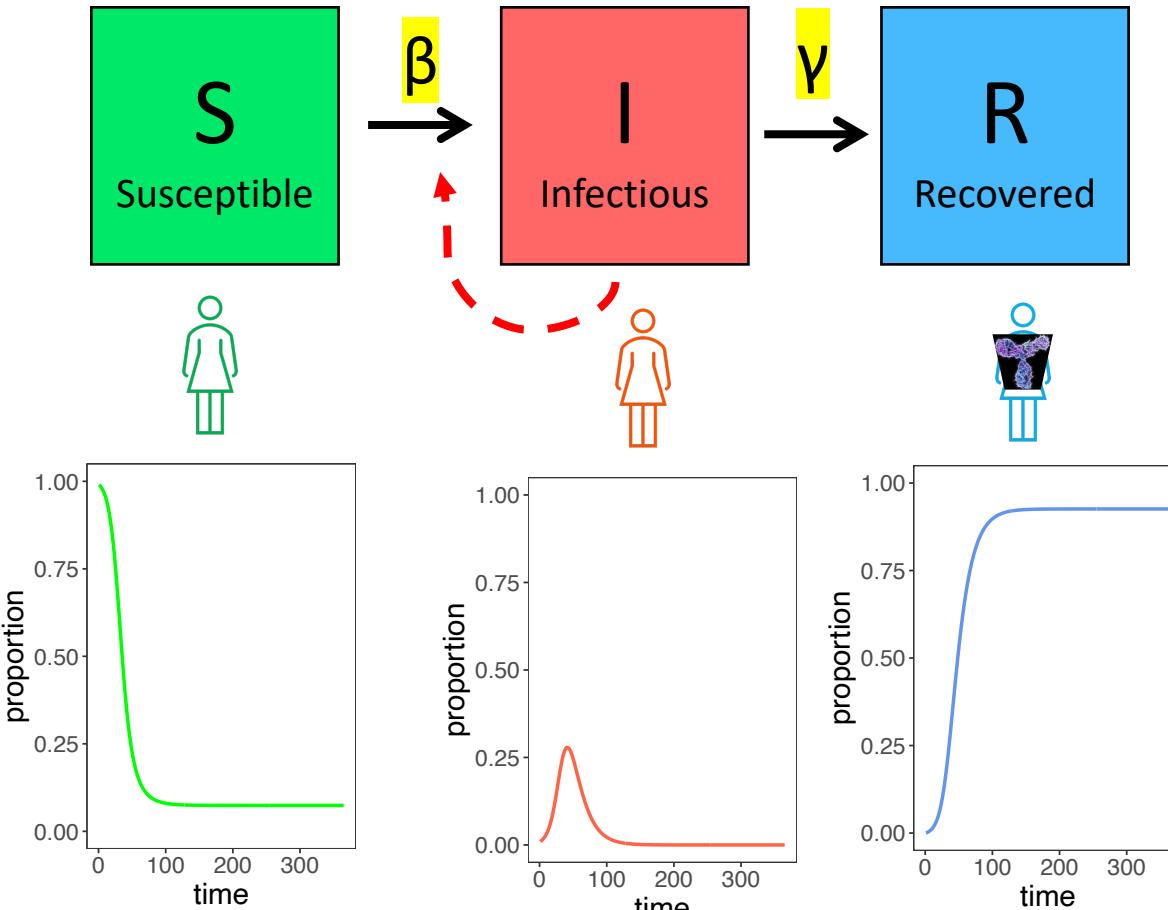
We use computers to simulate systems of equations in the SIR framework.

$R_0$  is the pathogen **basic reproduction number**.

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

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

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



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

infections  
created  
infections  
lost

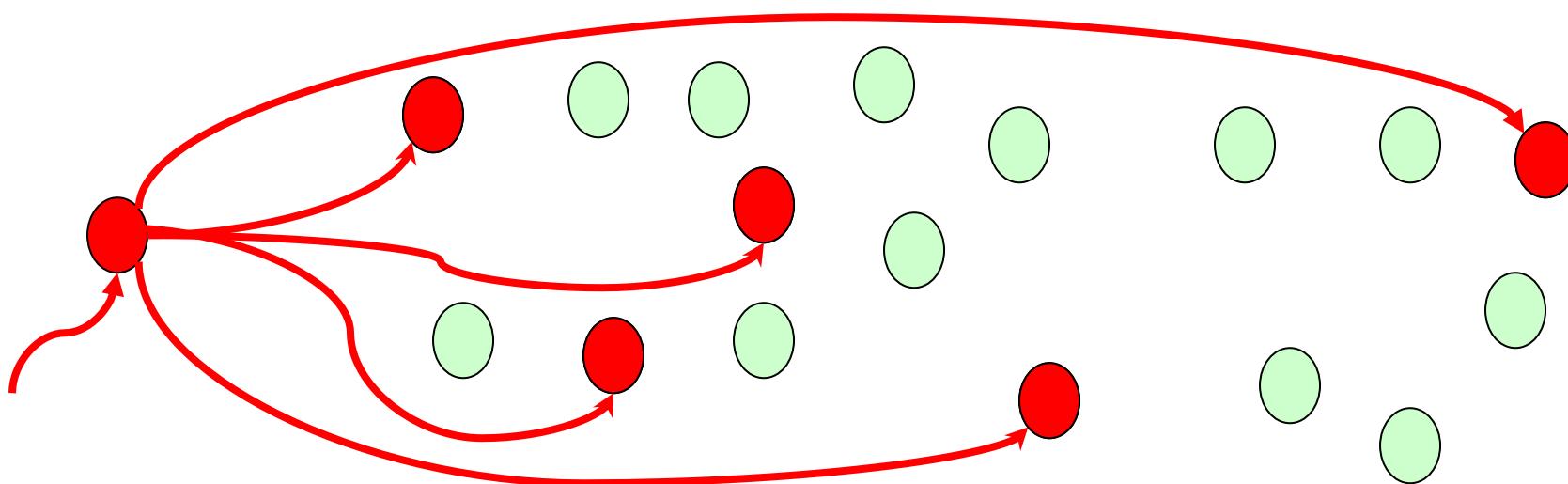
$R_0$  must be  $>1$  for  
a disease to start  
spreading!

$\beta$  = transmission rate

$\gamma$  = recovery rate

# $R_o$

- The **basic reproduction number** for a pathogen
- Defined as: the number of new cases caused by one infectious case in a **completely susceptible** population



What is  $R_o$ ?

$R_o = 5$

We can add realism to our models with births and deaths to **maintain** endemic pathogens.

$$\frac{dS}{dt} = b(S + I + R) - \beta SI - \mu S$$

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

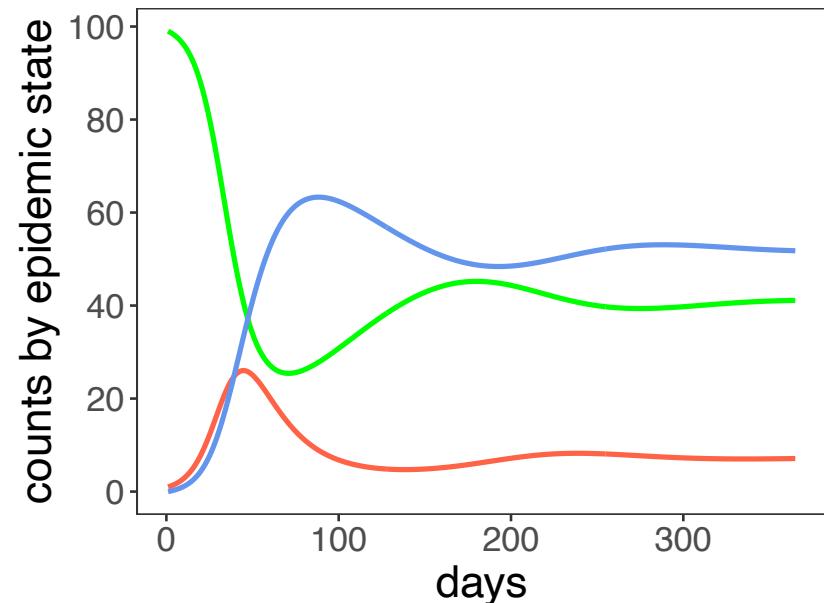
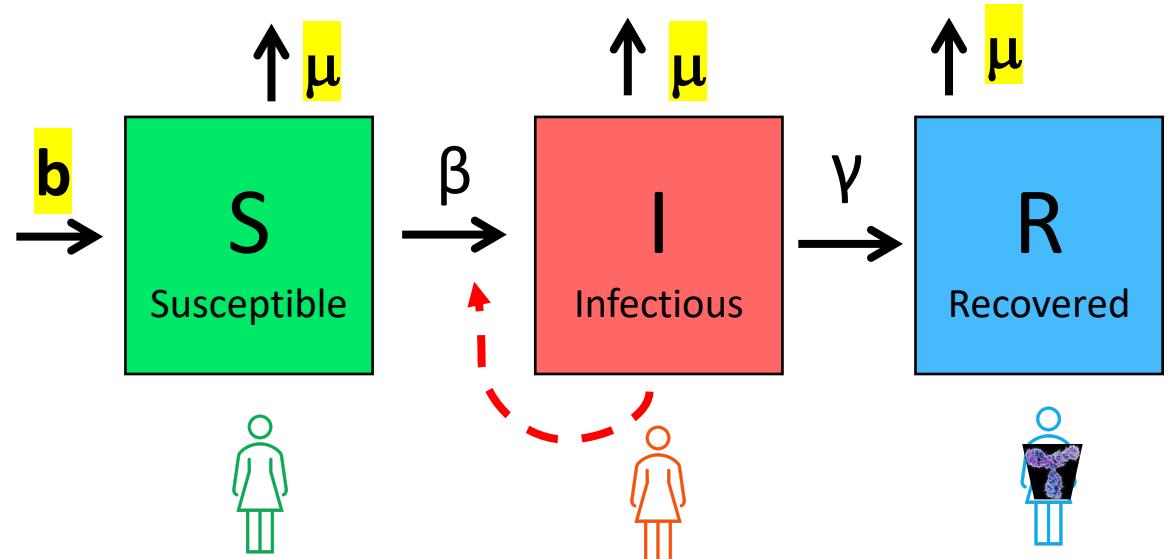
$$\frac{dR}{dt} = \gamma I - \mu R$$

**b** = birth rate

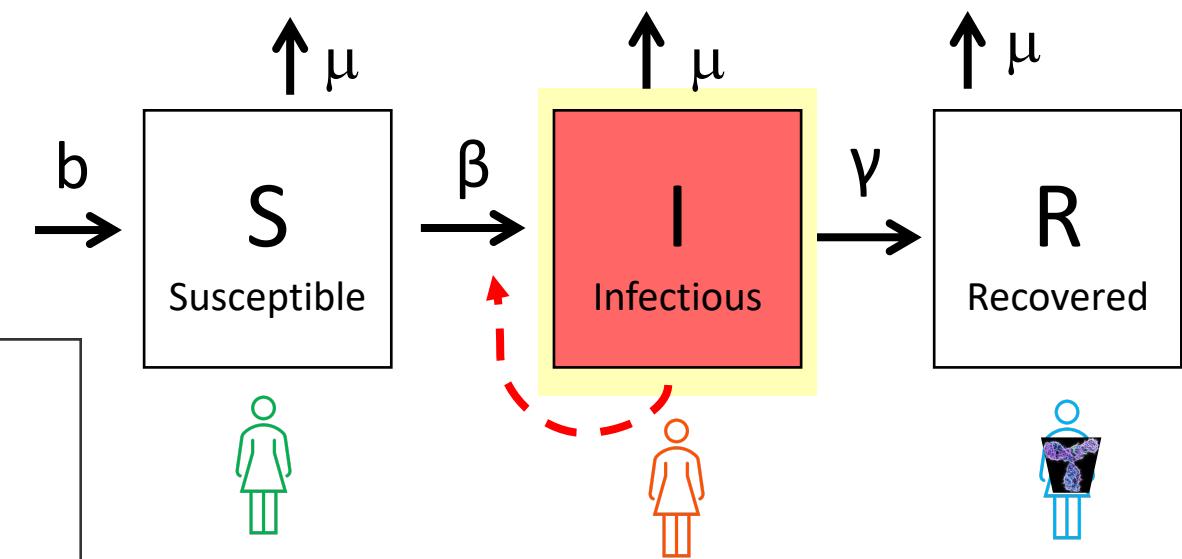
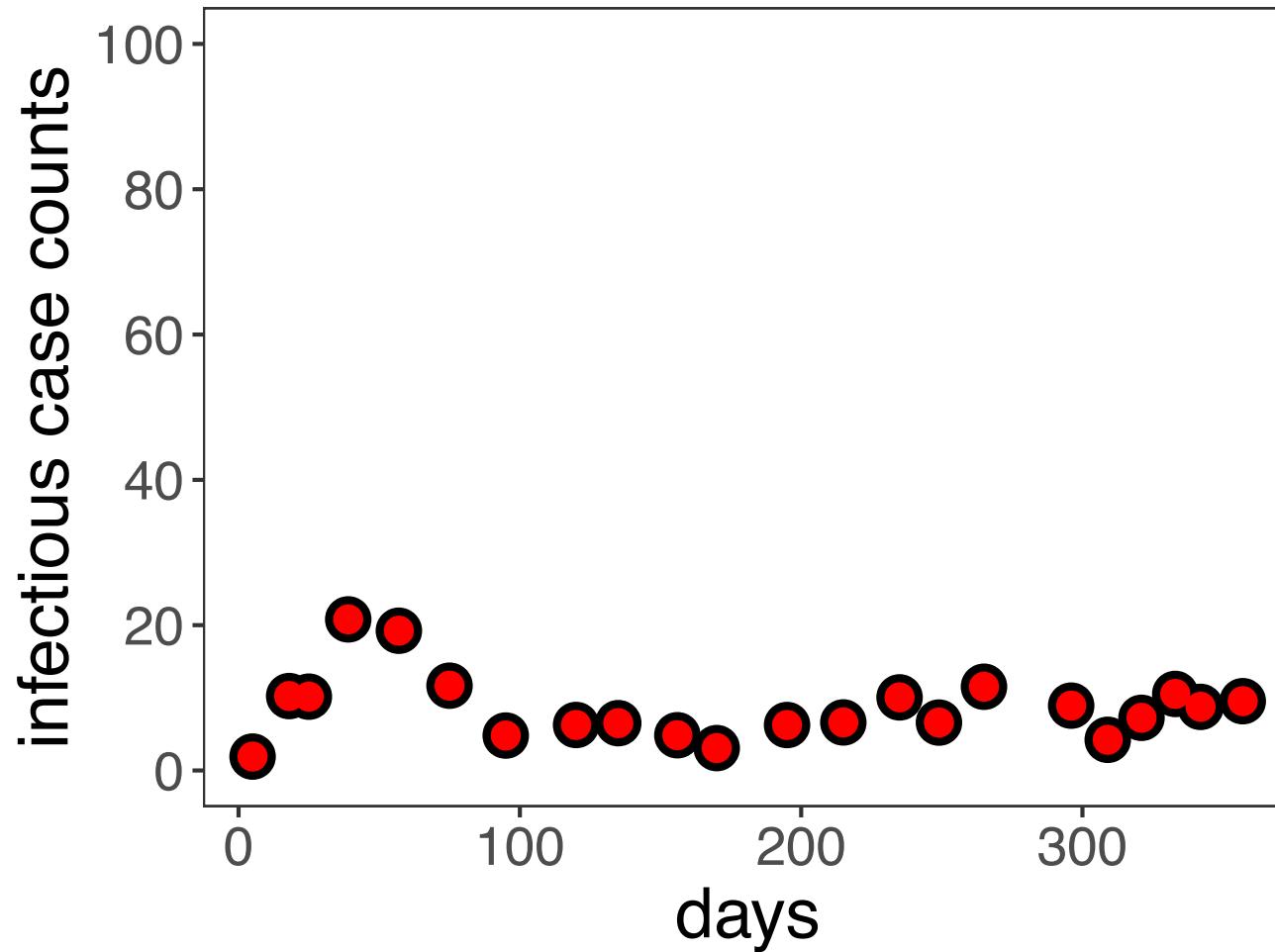
**$\mu$**  = death rate

$\beta$  = transmission rate

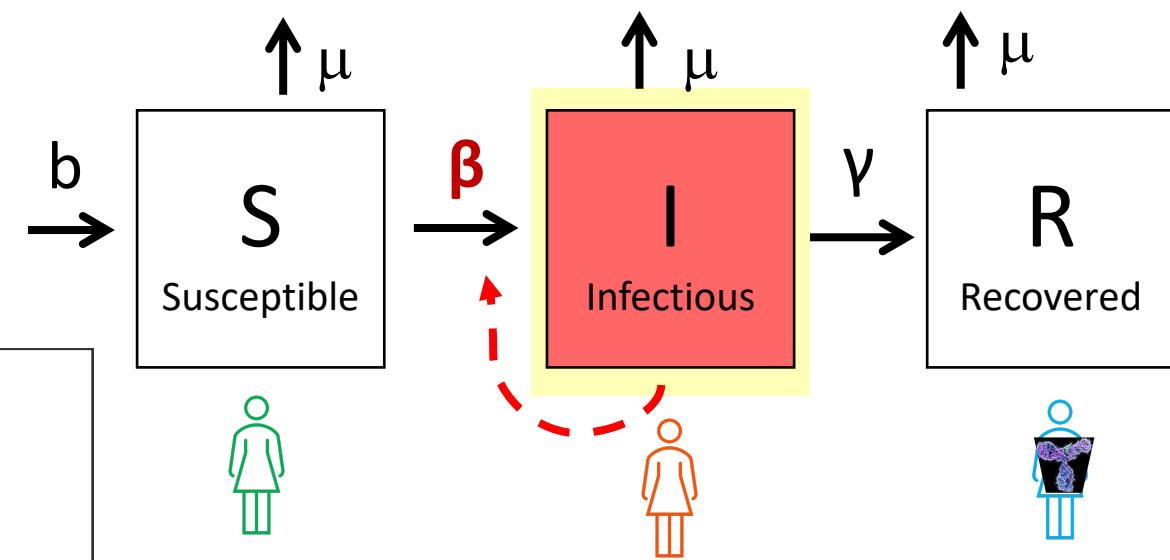
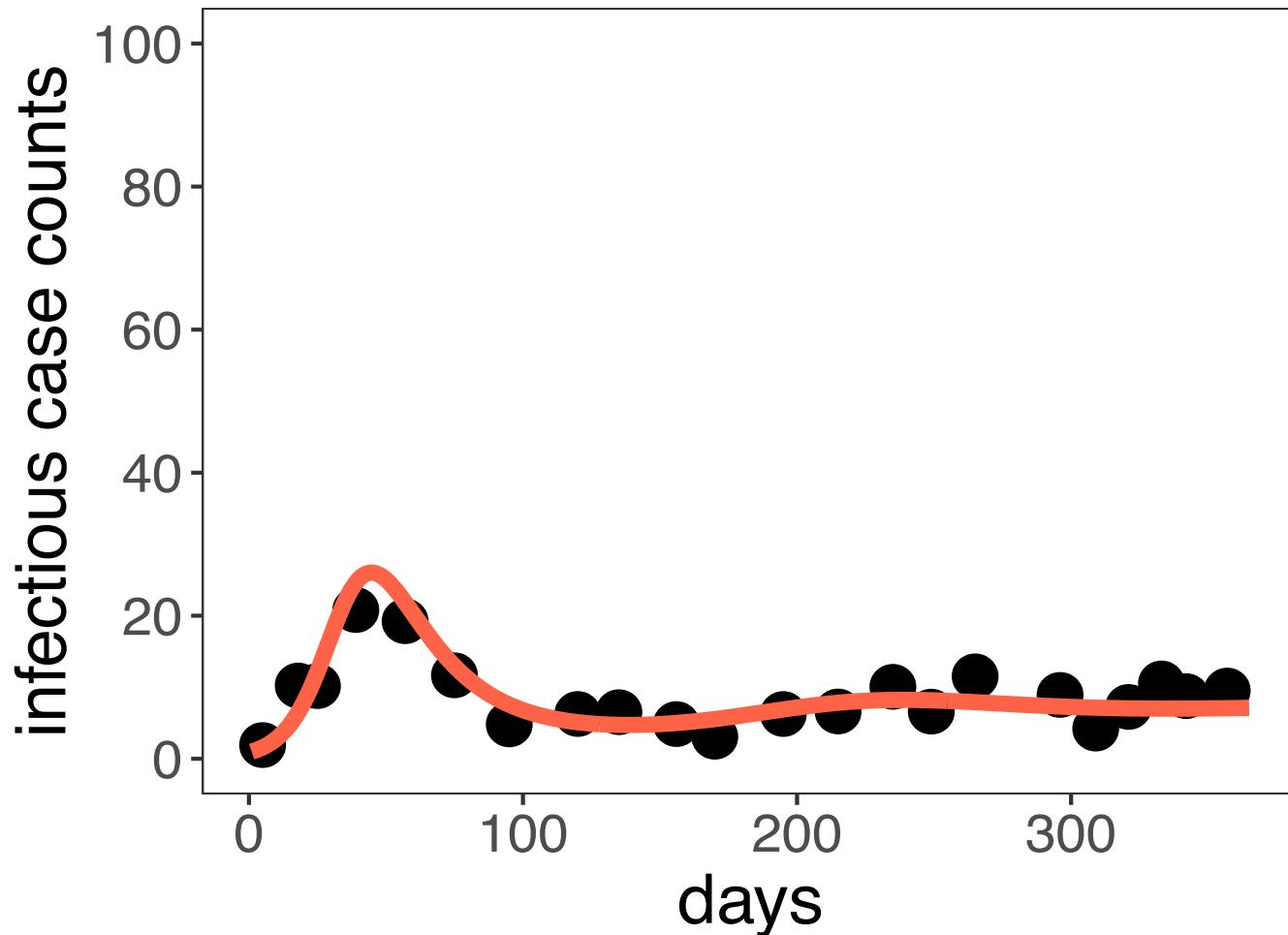
$\gamma$  = recovery rate



We can **estimate epidemic trajectories**  
by fitting SIR models to infectious case  
count data.



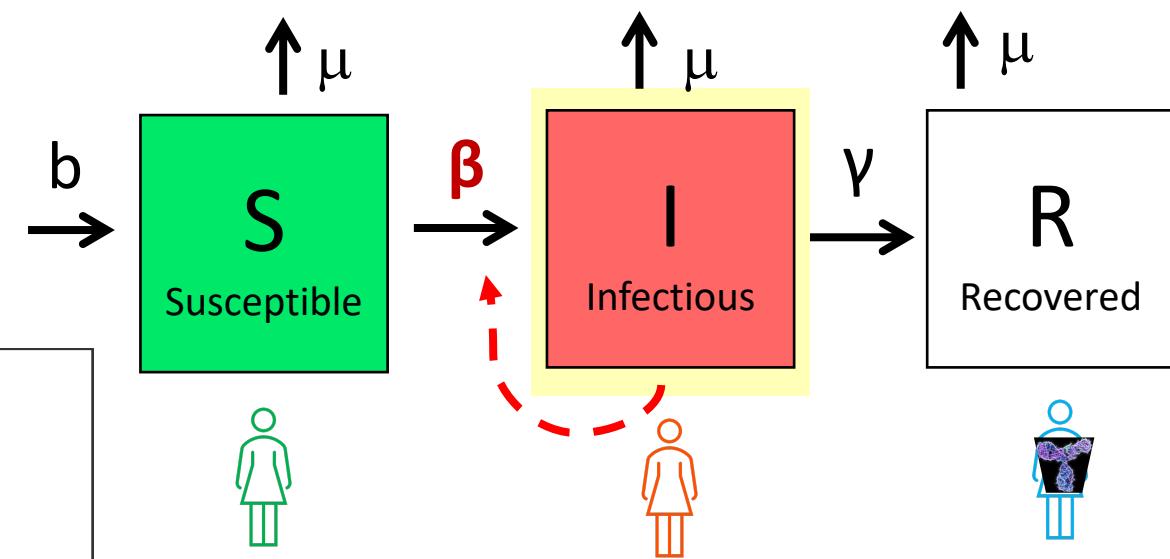
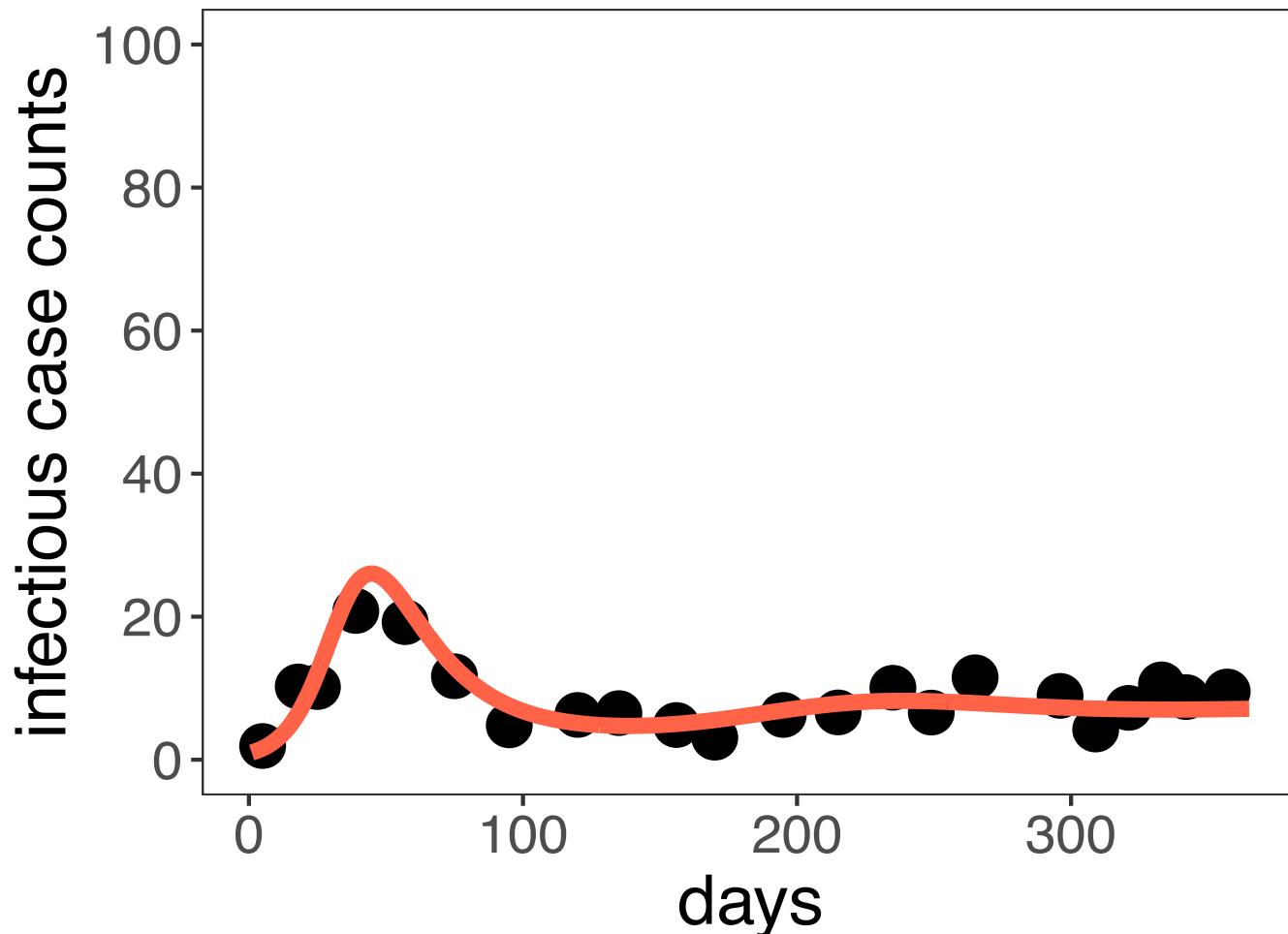
We can **estimate epidemic trajectories**  
by fitting SIR models to infectious case  
count data.



$$R_0 = \frac{\beta}{\gamma + \mu}$$

$b$  = birth rate  
 $\mu$  = death rate  
 $\beta$  = **transmission rate**  
 $\gamma$  = recovery rate

We can **estimate epidemic trajectories**  
by fitting SIR models to infectious case  
count data.



$$R_0 = \frac{\beta}{\gamma + \mu}$$

$$R_E = R_0 \frac{S}{N}$$

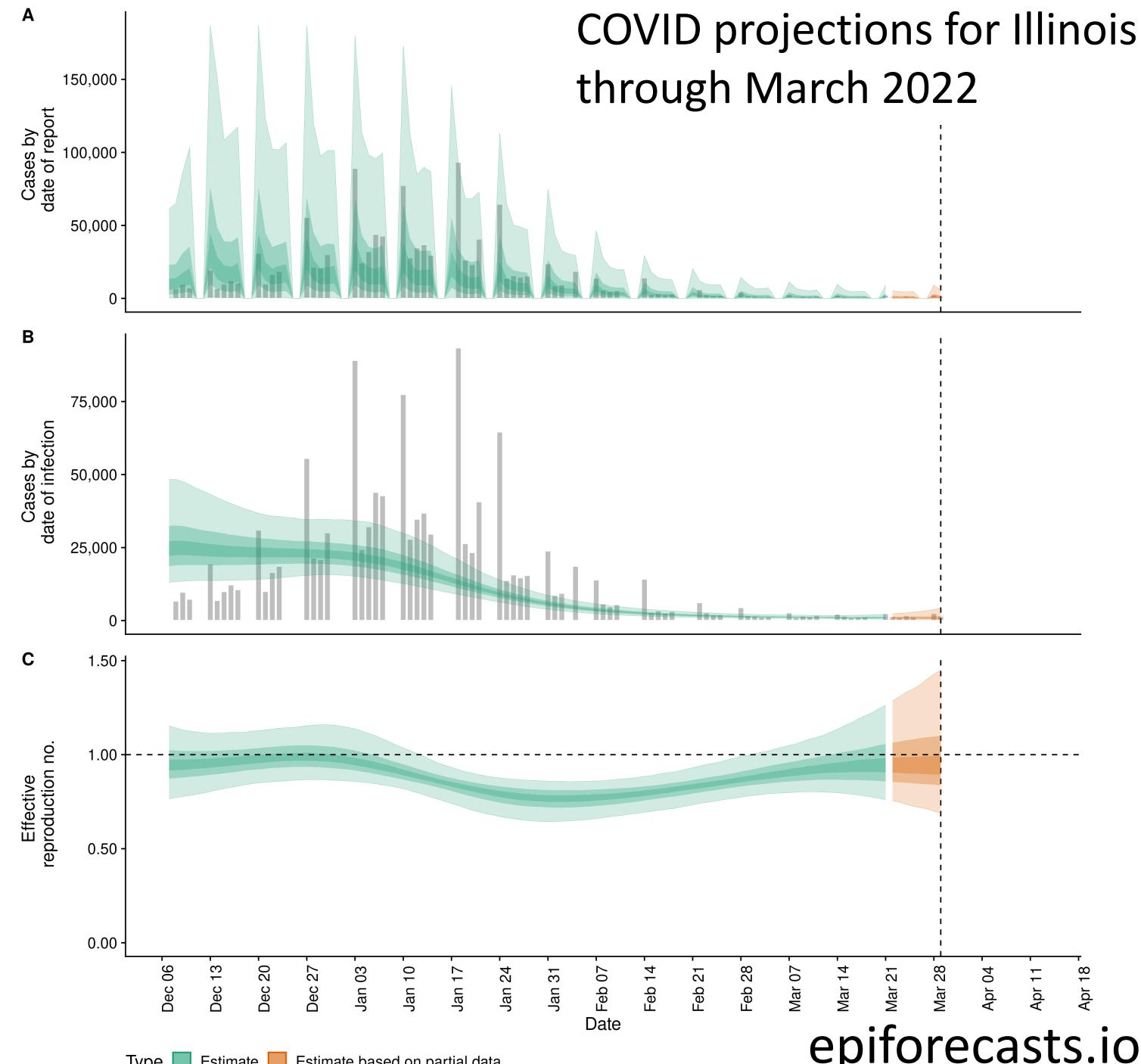
$b$  = birth rate  
 $\mu$  = death rate  
 $\beta$  = **transmission rate**  
 $\gamma$  = recovery rate

$\approx \lambda$  for a  
population model

(epidemics spread @ $R_E > 1$  and decline @ $R_E < 1$ )

# $R_E$ OR $R_t$

- The **effective reproduction number** for a pathogen
  - Defined as: the number of new cases caused by one infectious case in a **partially susceptible** population
  - Calculated as  $R_0^*$ \*proportion susceptible
  - Gives a realistic pulse of the current pace of the epidemic!
- $$R_E = R_0 \frac{S}{N}$$

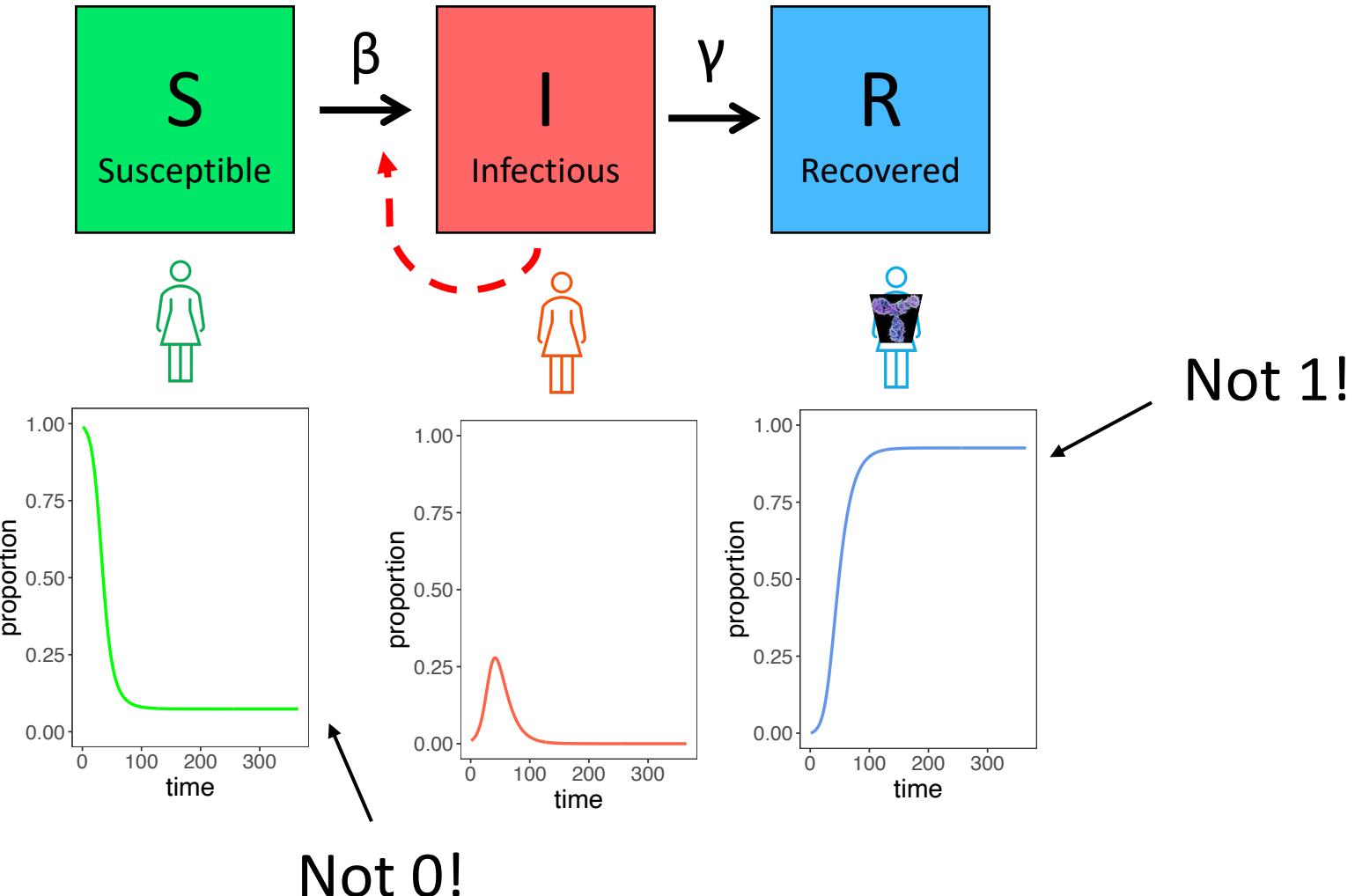


No matter the dynamics, not everyone gets infected before the epidemic ends!

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

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

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



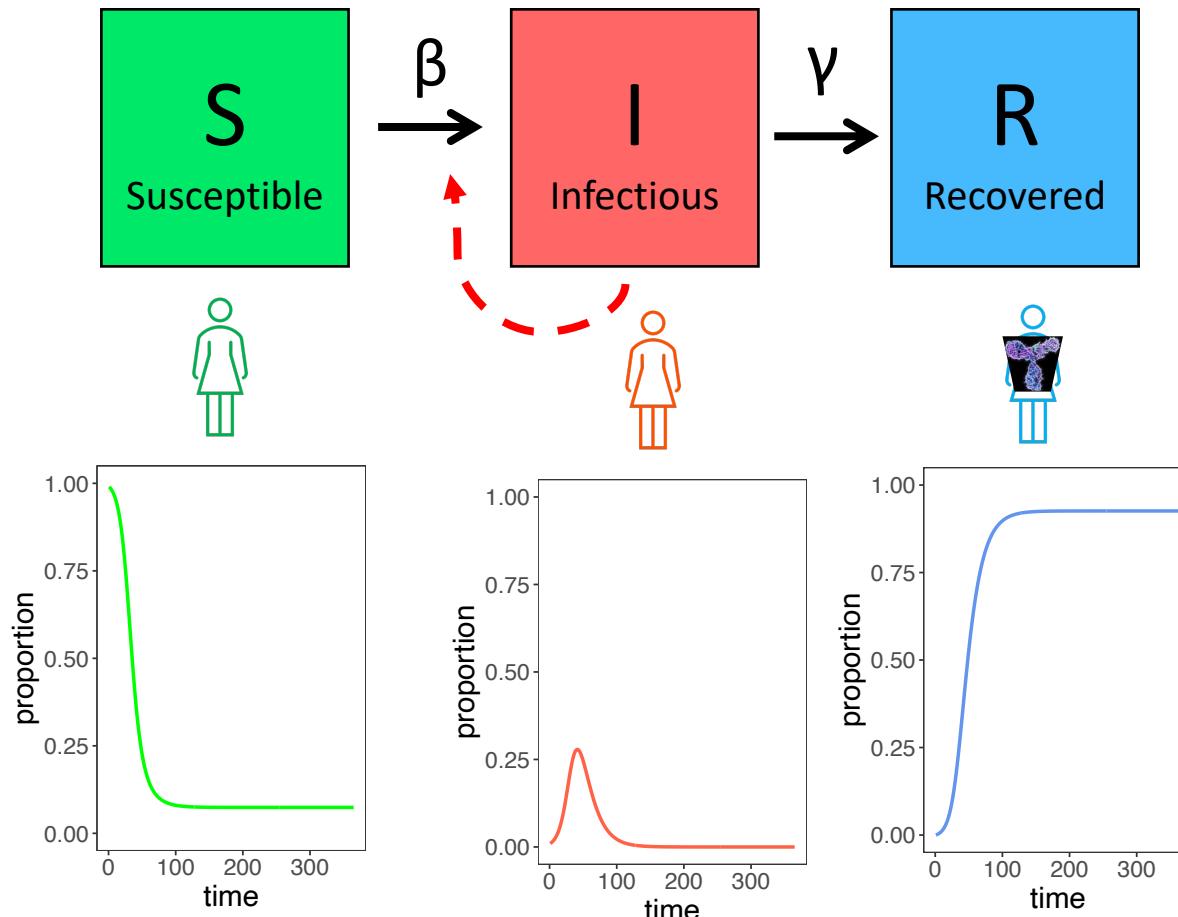
$\beta$  = transmission rate  
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# No matter the dynamics, not everyone gets infected before the epidemic ends!

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

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

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



- The epidemic does not end because all individuals have been infected and have either died or recovered.
- Rather, finding new susceptibles becomes more difficult and  $R_E < 1$

$\beta$  = transmission rate

$\gamma$  = recovery rate

**Public health interventions** can be employed to reduce both  $R_0$  and  $R_E$

## **$R_0$** interventions

- Social distancing
- Masking
- Limits to gathering sizes
- Drugs that shorten the infectious period



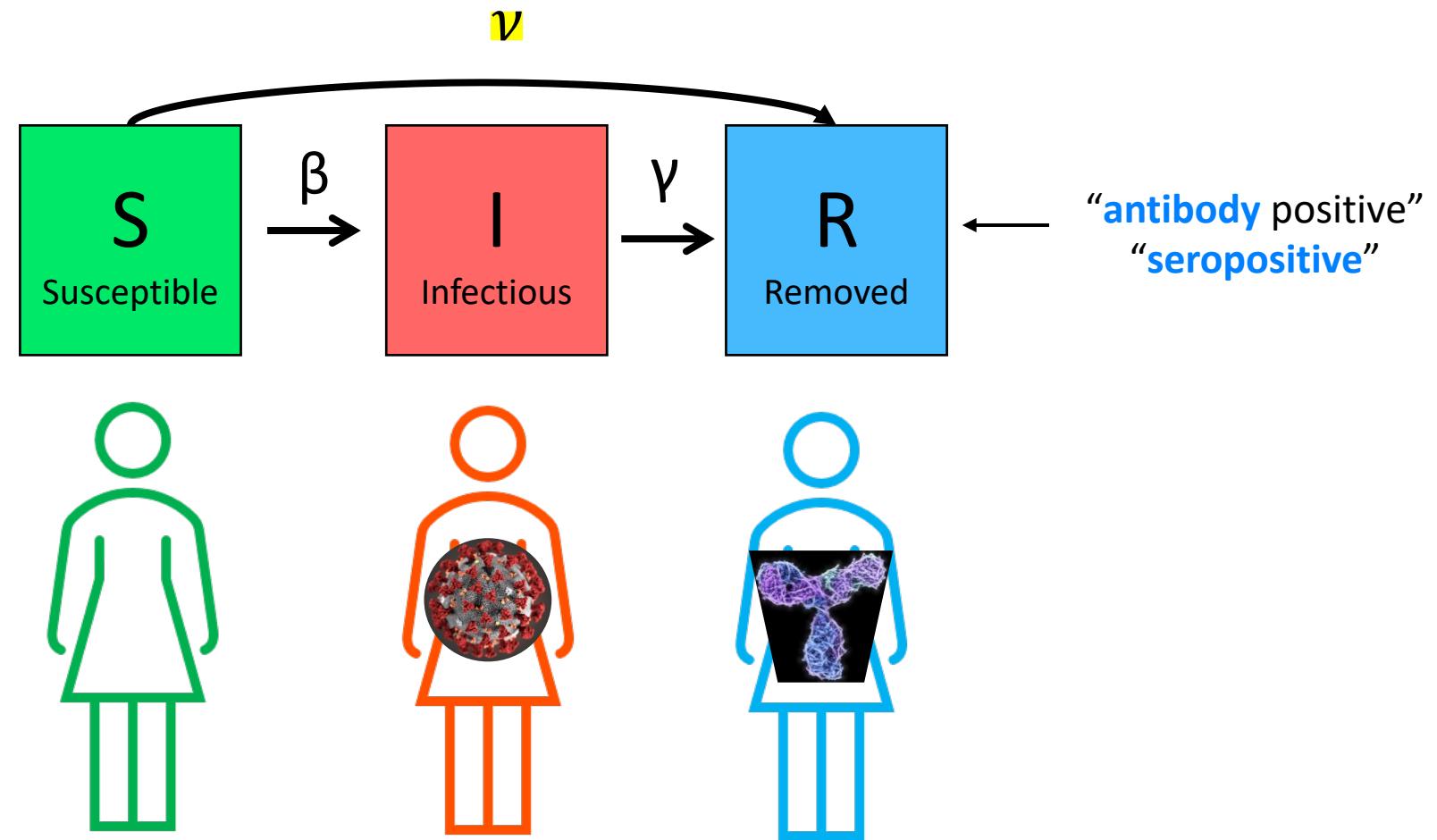
## **$R_E$** interventions

- Vaccination



# Mathematics of Vaccination

- Goal: **Reduce  $R_E < 1$**  by removing individuals from the susceptible population.



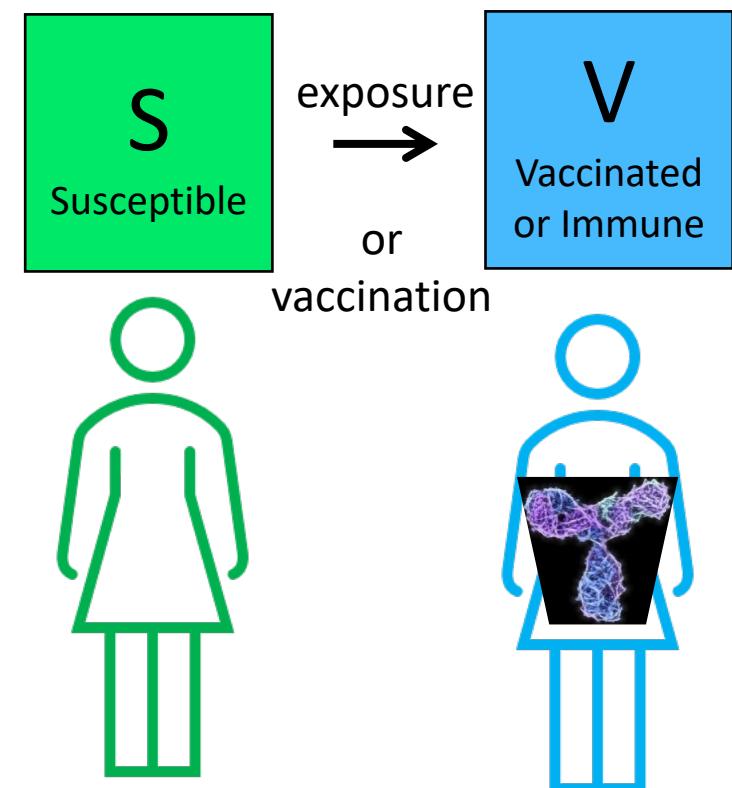
$\beta$  = transmission rate

$\gamma$  = recovery rate

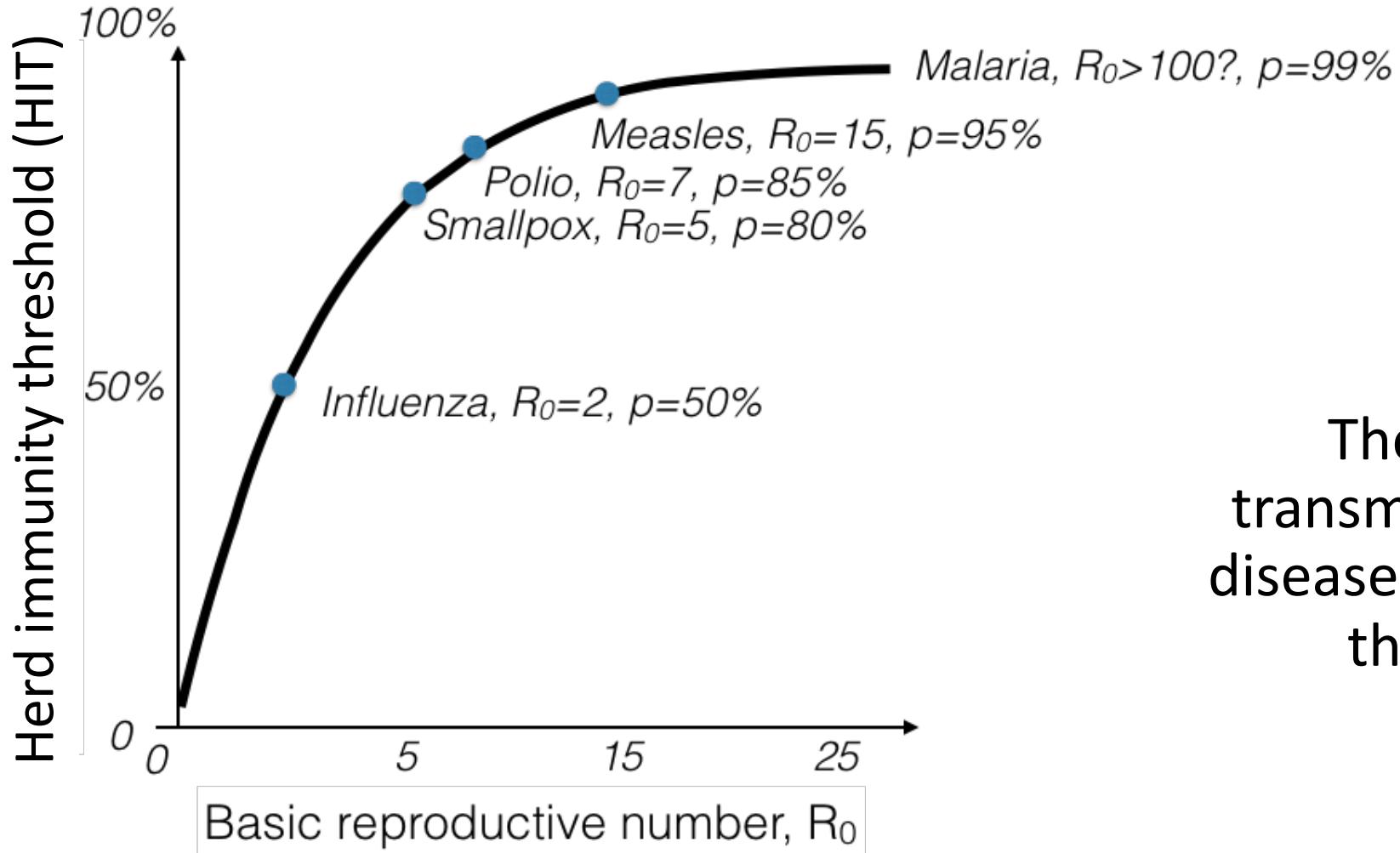
$\nu$  = vaccination rate

# Mathematics of Vaccination

- Goal: **Reduce  $R_E < 1$**  by removing individuals from the susceptible population.
- Because infectious periods tend to be short-lived (*depending on the pathogen!*), we can theoretically divide the population into two classes: S (susceptible) and V (vaccinated, or immune)
- If  $S + V = N$ , then  
Prop. Susceptible + Prop. Vaccinated = 1.
- Remember,  $R_E = R_0 P_S$  or  $R_E = R_0(1 - P_V)$
- $R_E < 1 \approx (1 - P_V)R_0 < 1$
- Rearranging,  $P_V > 1 - \frac{1}{R_0}$
- **This is the herd immunity threshold.**
- Even susceptibles will not become infected because the disease will not spread ( $R_E < 1$ ).



# $R_0$ and the Herd Immunity Threshold



The more  
transmissible the  
disease, the higher  
the HIT!

## Que. 3 What is $R_E$ ? It is

100

- A. the herd immunity threshold during an epidemic
- B. the rate of change of a disease in a completely susceptible population
- C.  $R_0$  adjusted for the current fraction of the population that is susceptible

## Que. 3 What is $R_E$ ? It is

100

A. the herd immunity threshold during an epidemic

8%

B. the rate of change of a disease in a completely susceptible population

3%

c.  $R_0$  adjusted for the current fraction of the population that is susceptible

89%

## Que. 3 What is $R_E$ ? It is

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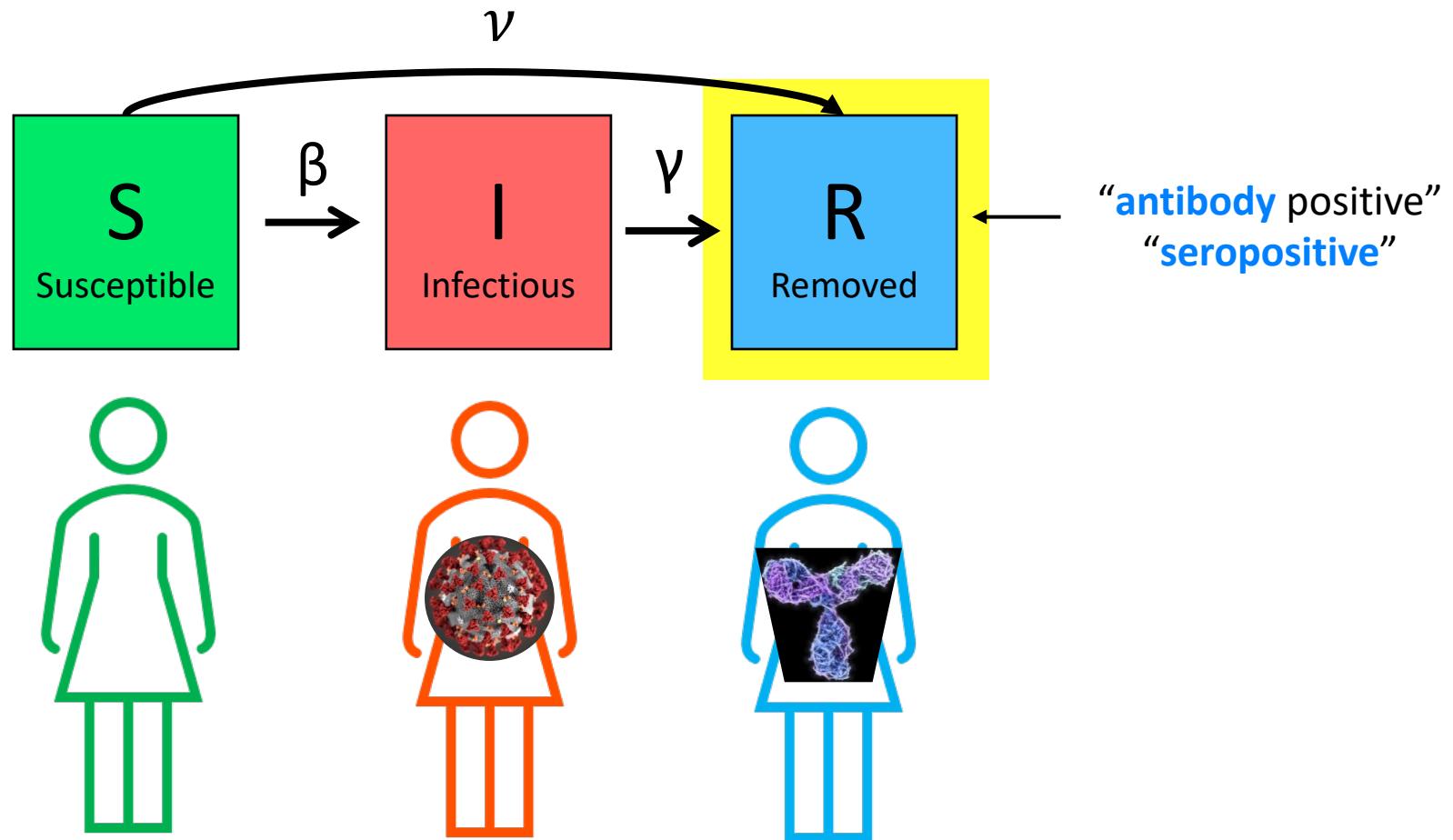
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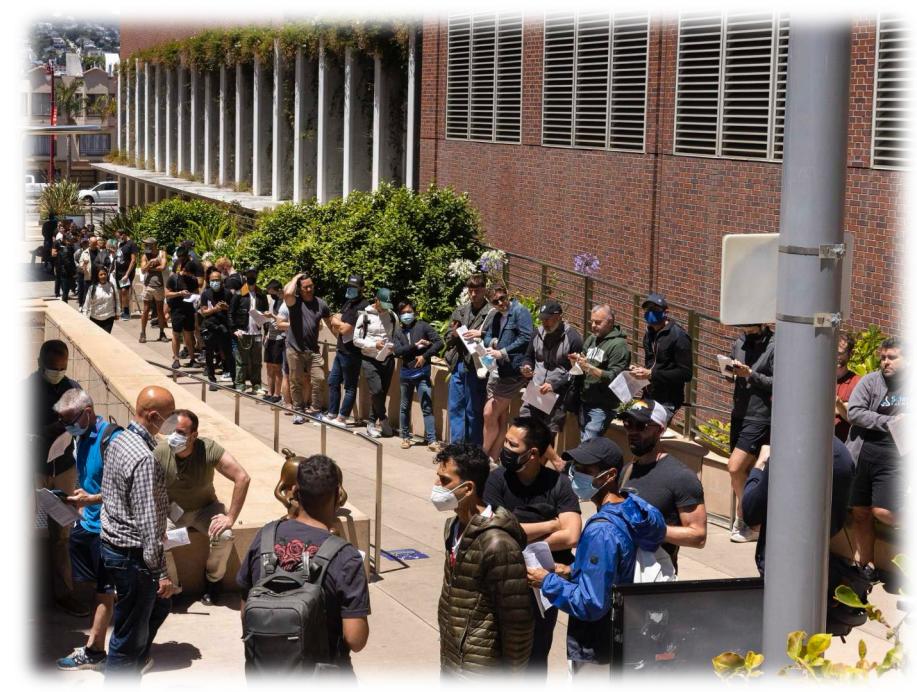
89%

# Vaccination stems from a long history

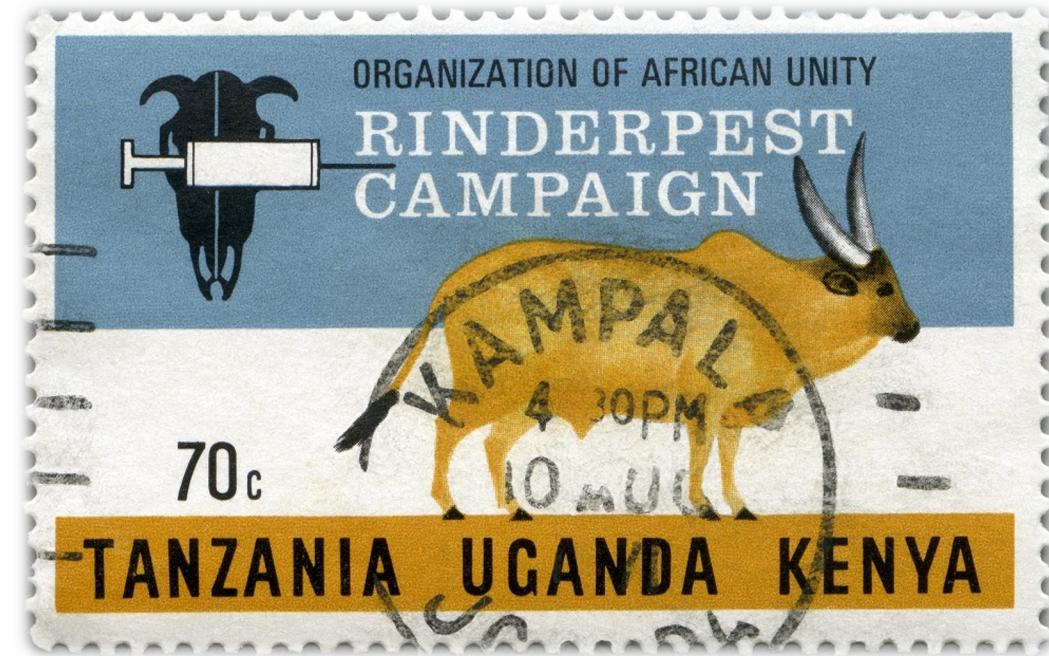


# Vaccination stems from a long history

- Variolation: Early attempts to provide protection against smallpox (*Variola virus*) via inoculation with scab material from a recent patient infected with *Variola minor*
  - First described in China in the 10<sup>th</sup> century
  - Caused 1% mortality!
- 1789 Edward Jenner used cowpox vesicles to inoculate an 8-year-old boy
  - Later inoculated with smallpox and boy was unaffected
  - The first vaccine, taken from *vacca*, cow in Latin
- Smallpox was globally eradicated in 1977, following a massive international campaign
- Today, we are seeing enhanced transmission of monkeypox partly resulting from a lack of circulating immunity to closely related smallpox

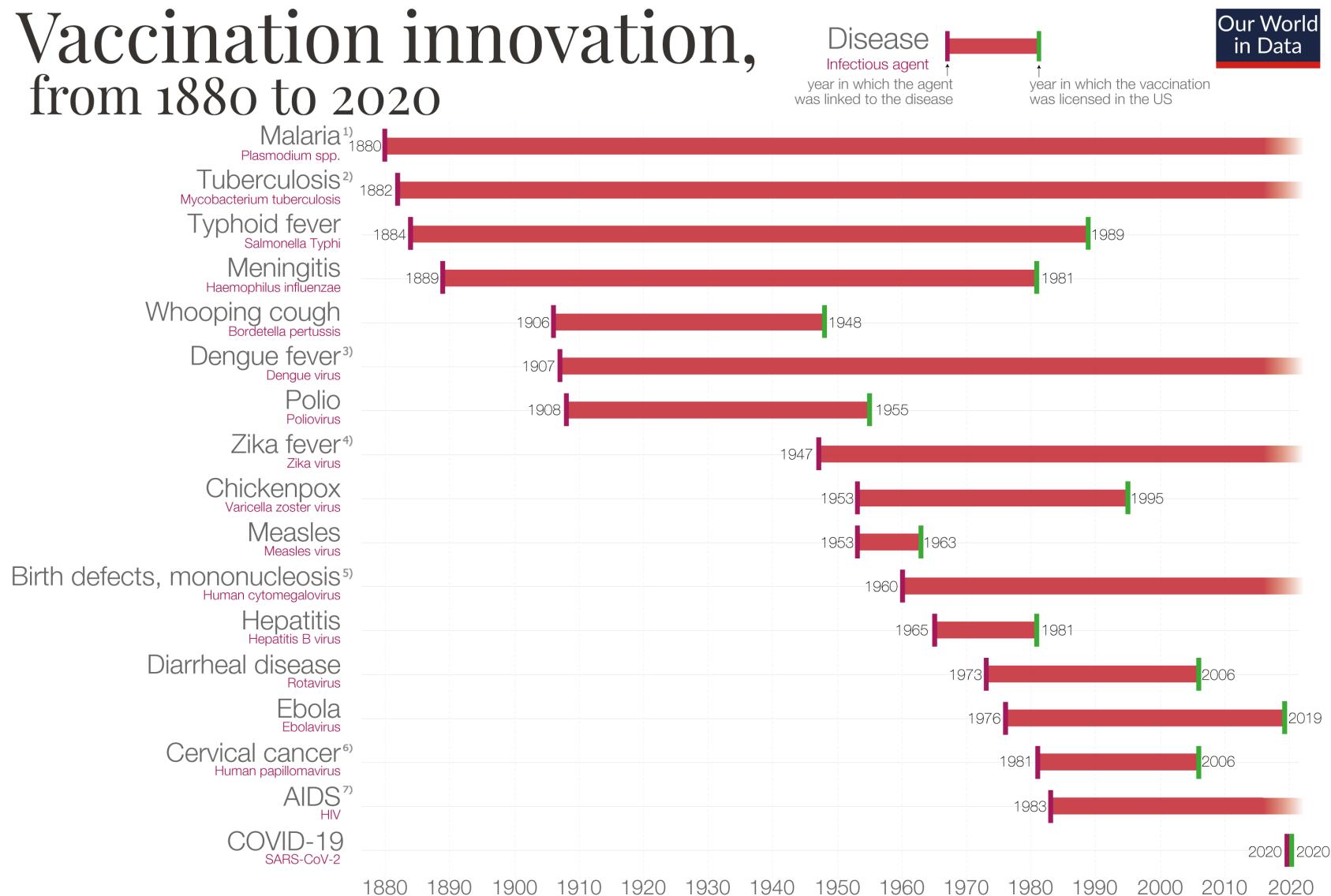


# Only two global vaccination success stories



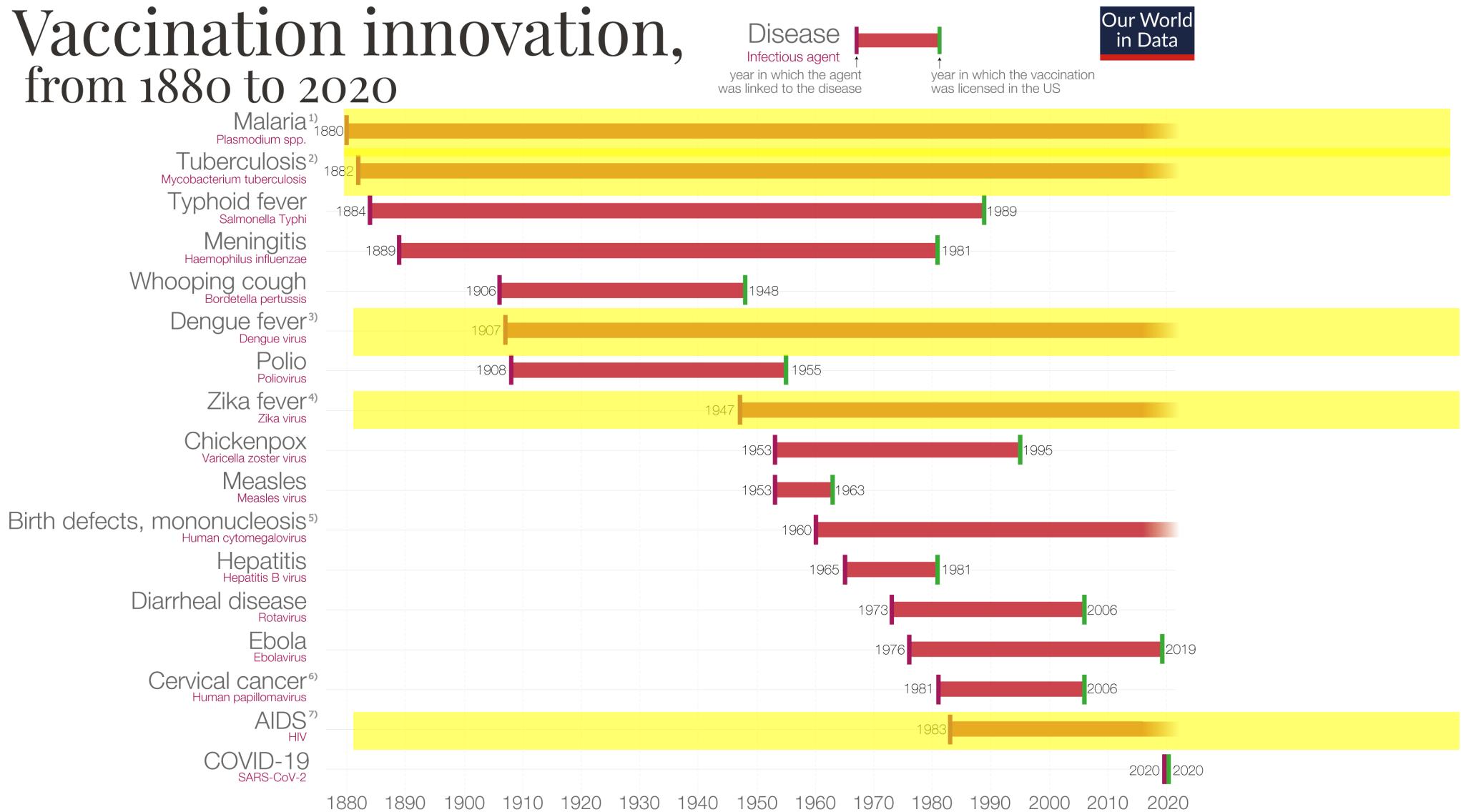
# The pace of vaccine development has accelerated drastically

## Vaccination innovation, from 1880 to 2020



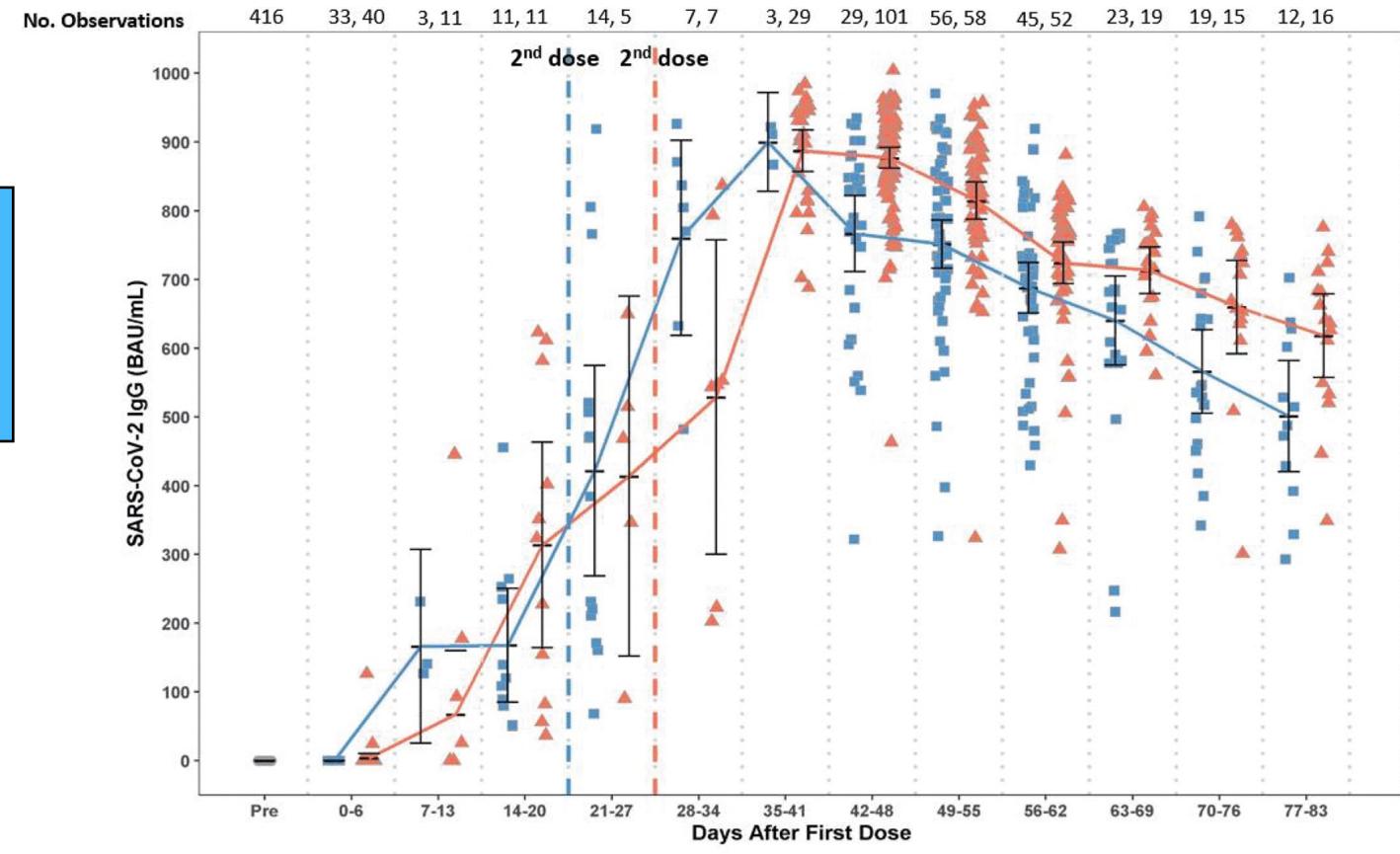
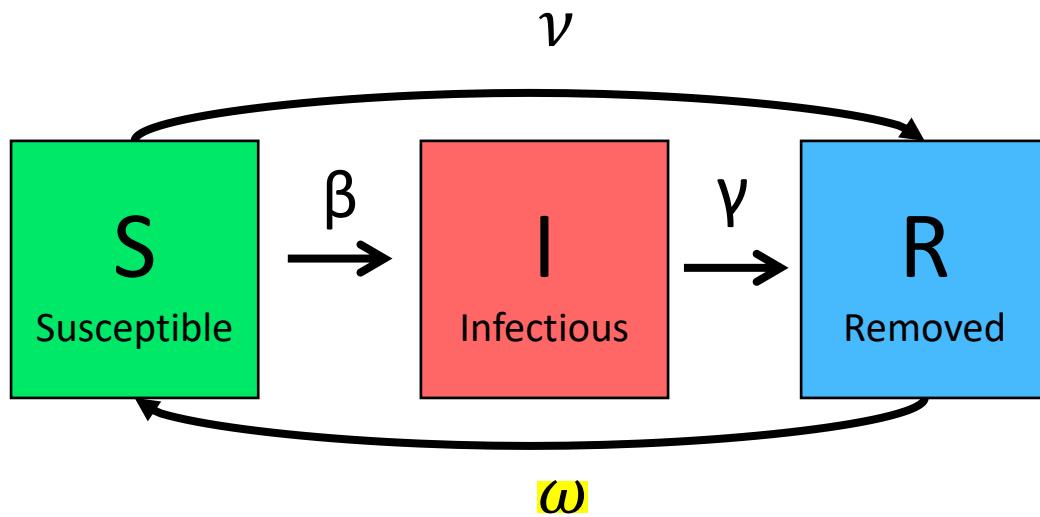
# But we still lack vaccines for several important diseases.

## Vaccination innovation, from 1880 to 2020



# Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens



$\beta$  = transmission rate

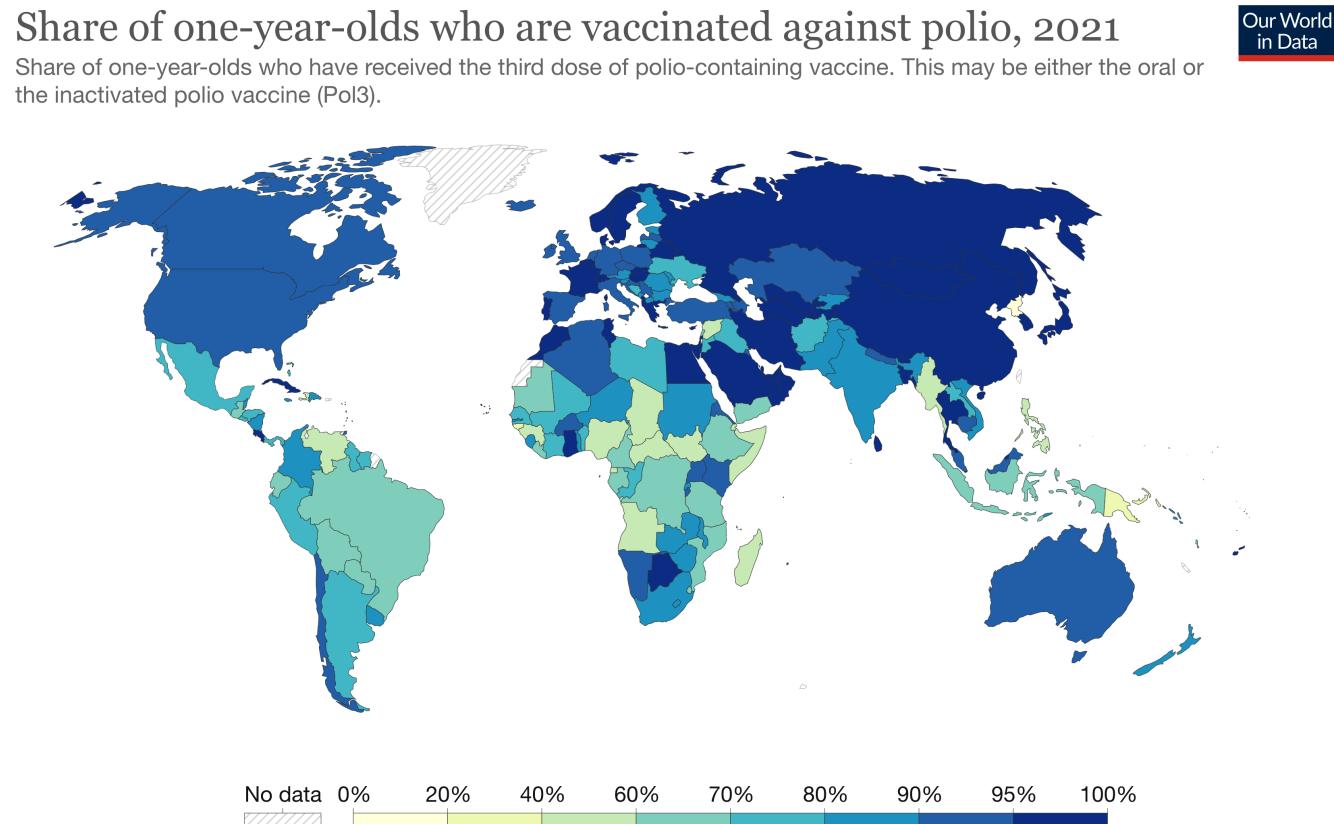
$\gamma$  = recovery rate

$\nu$  = vaccination rate

$\omega$  = rate waning immunity

# Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access



Source: WHO; UNICEF (2022)

Note: Polio is a highly infectious viral disease. The polio virus invades the nervous system and can cause irreversible paralysis.

[OurWorldInData.org/polio/](https://OurWorldInData.org/polio/) • CC BY

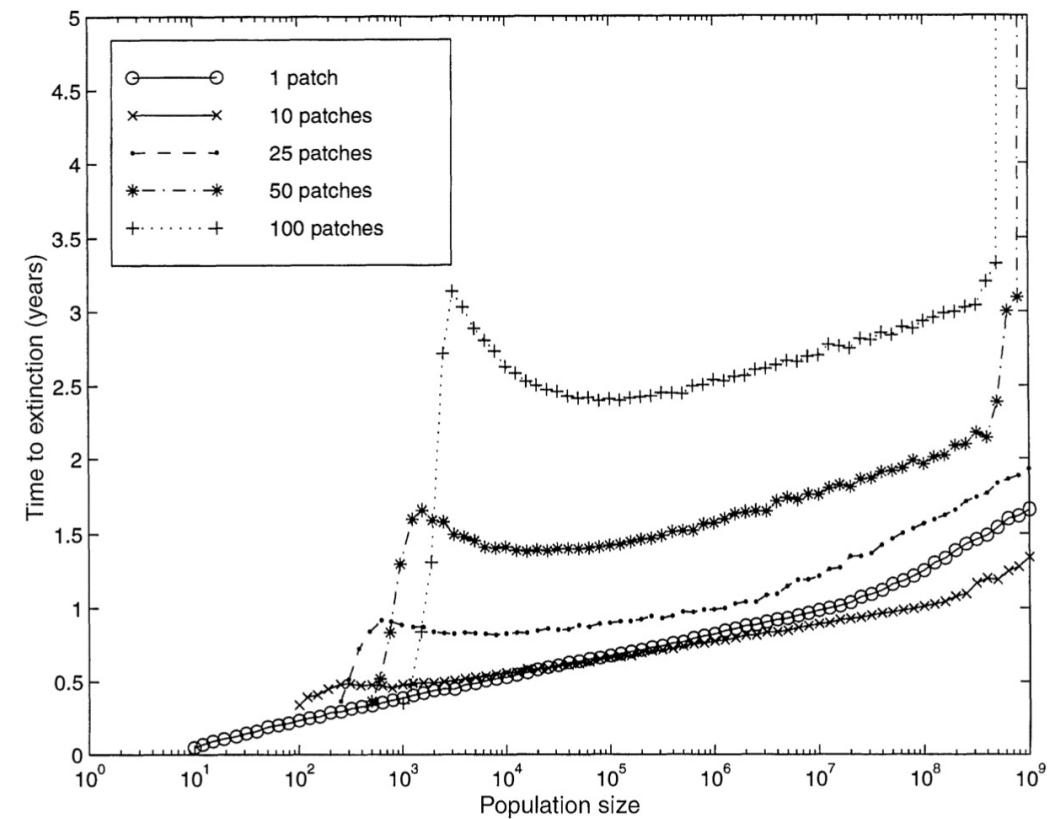
# Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access
- Continuous births
- Animal reservoirs



# Challenges to Vaccination

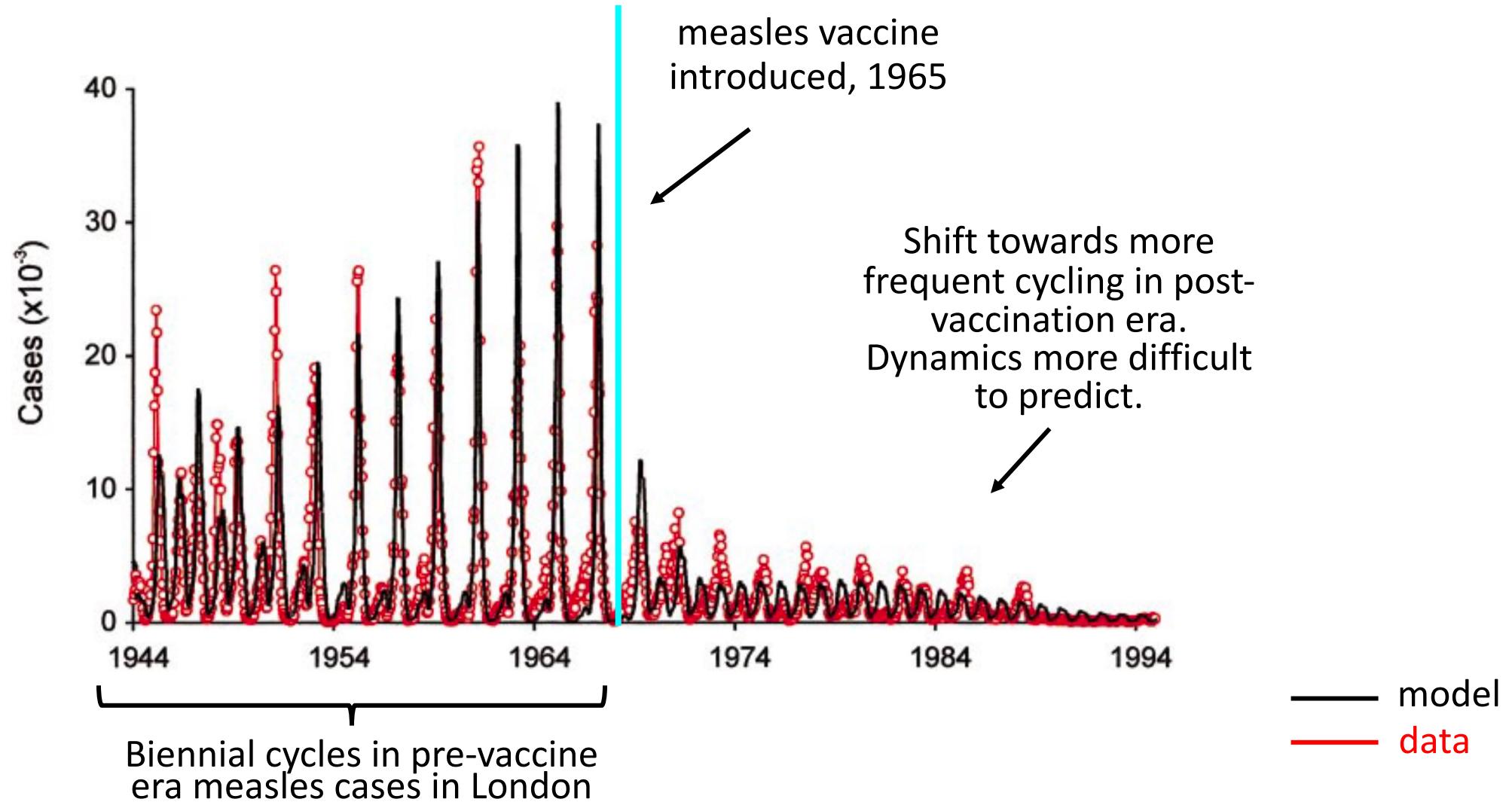
- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access
- Continuous births
- Animal reservoirs
- Spatial structure (metapopulation rescue)



# Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access
- Continuous births
- Animal reservoirs
- Spatial structure (metapopulation rescue)
- More complex pathogens!

# Much of the mathematical theory underlying vaccination was first developed for measles



Even for measles, stochastic dynamics mean that predictions become more challenging at smaller population sizes.

