Fundamentals of Ecology

Week 8, Ecology Lecture 5

Cara Brook

February 20, 2024

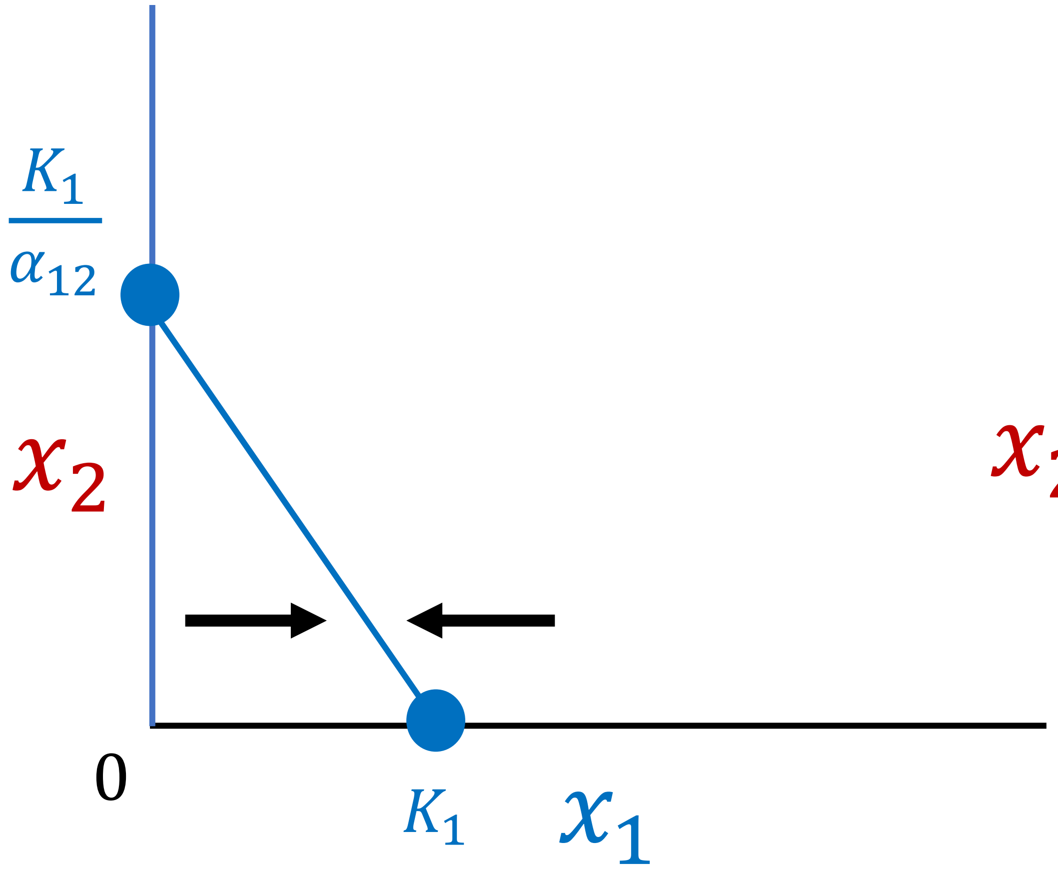
Let’s recap a bit!

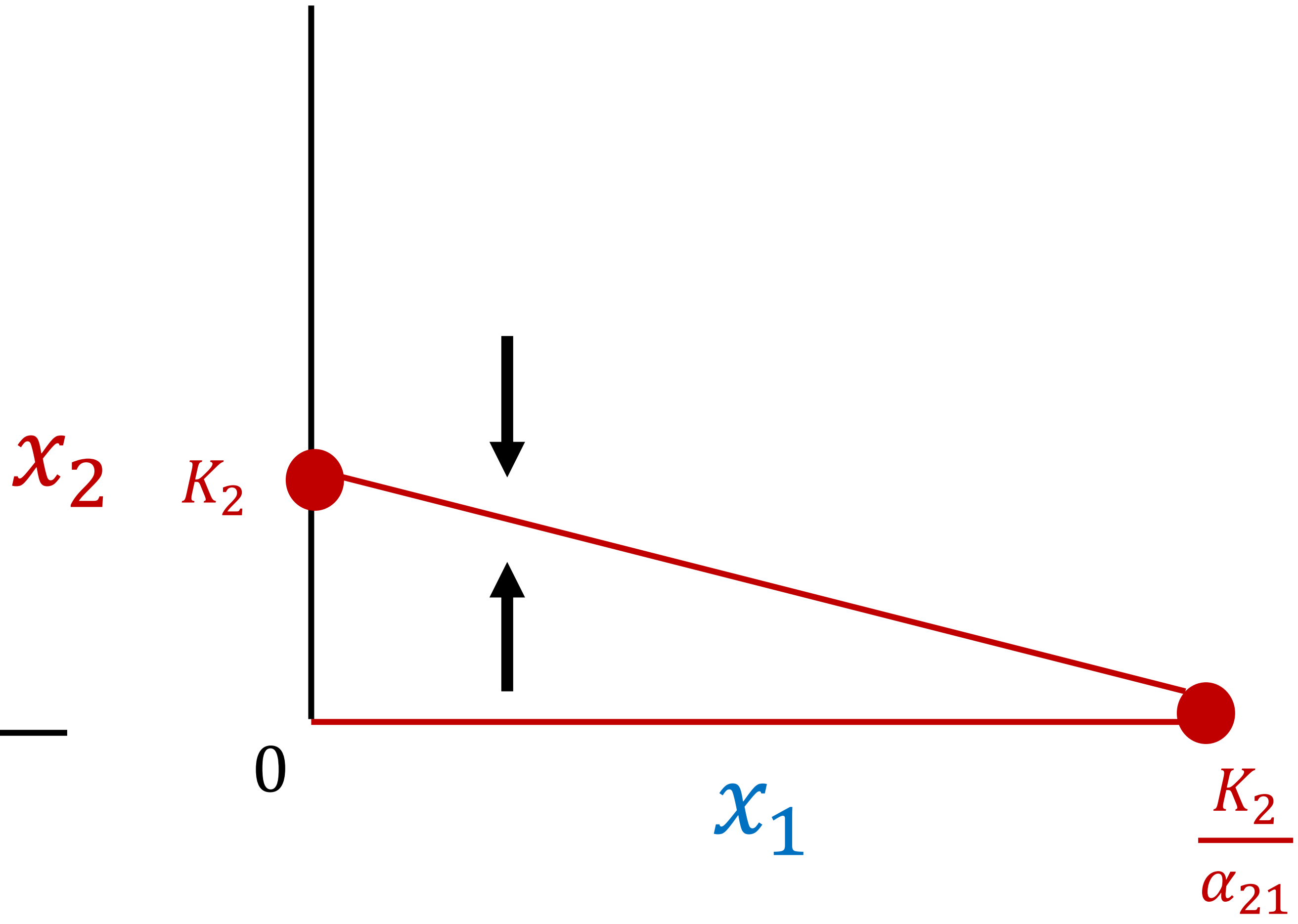
Interspecies Dynamics

- 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





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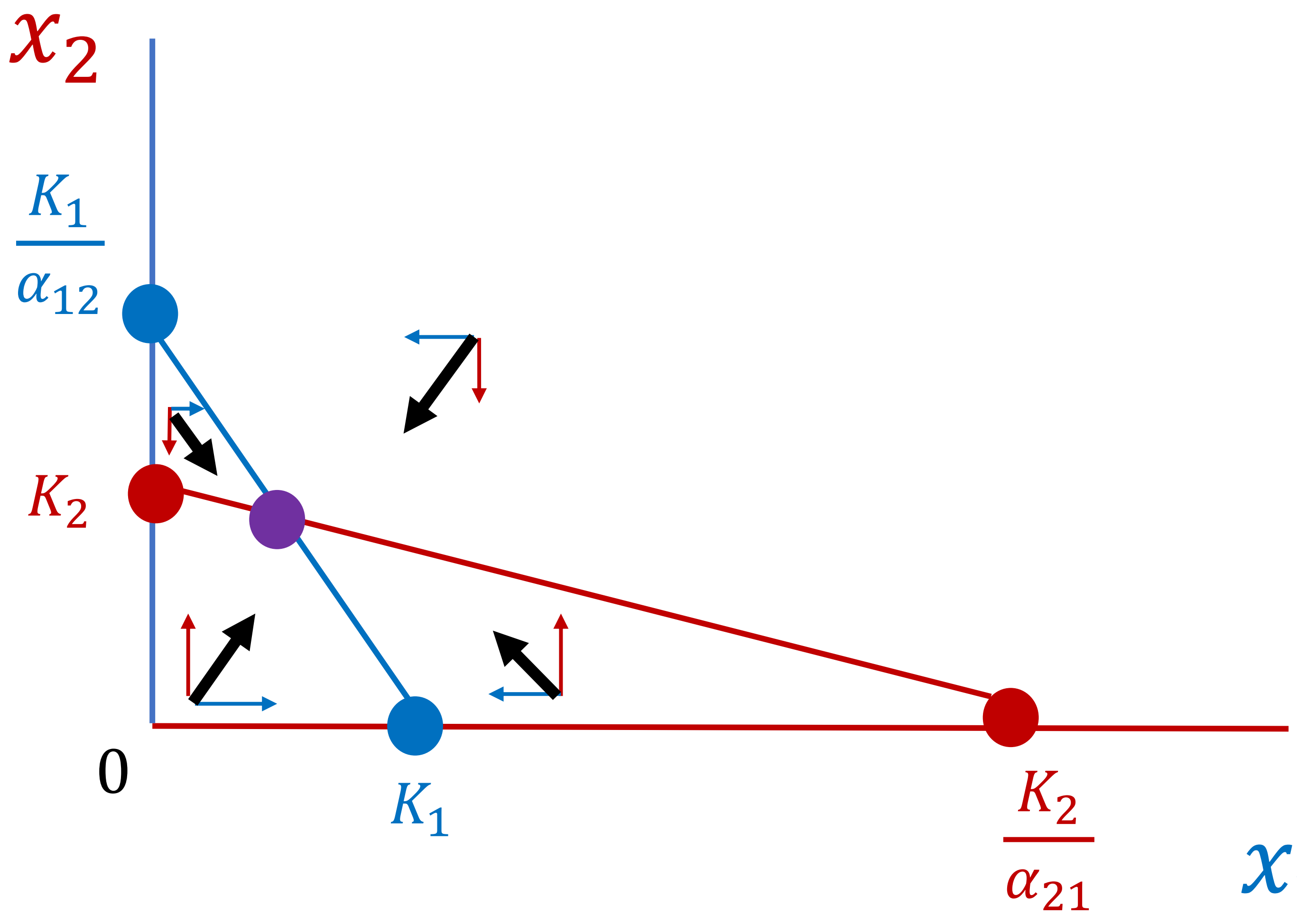
Interspecies Dynamics

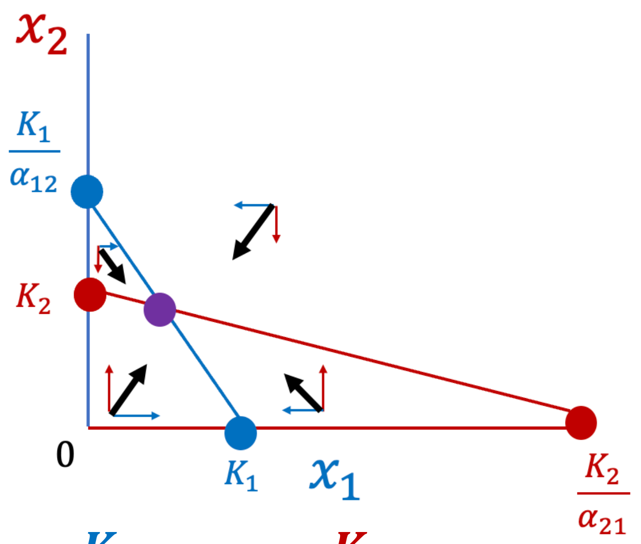


Let's recap a bit!

Interspecies Dynamics

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

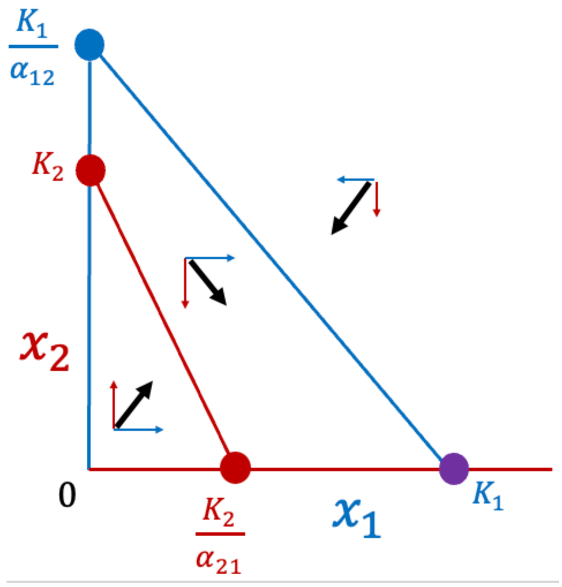
Case 1: Stable coexistence



$$\frac{K_1}{\alpha_{12}} > K_2 \ \& \ \frac{K_2}{\alpha_{21}} > K_1$$

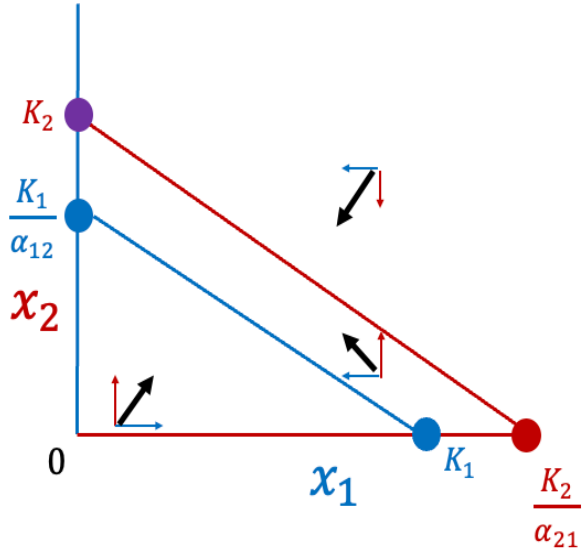
$$(\alpha_{12} * \alpha_{21} < 1)$$

Case 2: Spp. 1 wins



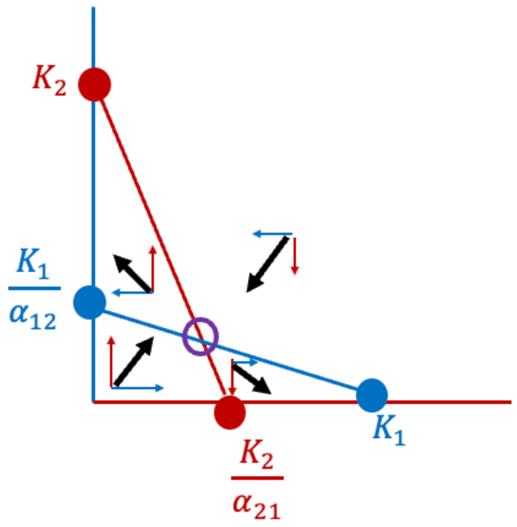
$$\frac{K_1}{\alpha_{12}} < K_2 \ \& \ \frac{K_2}{\alpha_{21}} > K_1$$

Case 3: Spp. 2 wins



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

Case 4: Precedence



$$\frac{K_1}{\alpha_{12}} < K_2 \ \& \ \frac{K_2}{\alpha_{21}} < K_1$$

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

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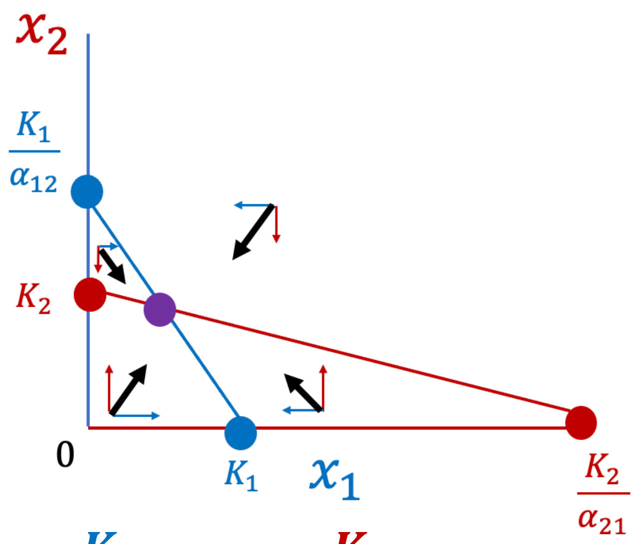
Case 1: Stable coexistence



$$\frac{K_1}{\alpha_{12}} > K_2 \ \& \ \frac{K_2}{\alpha_{21}} > K_1$$

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

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

Gause's experiments:

Competitive exclusion was observed:

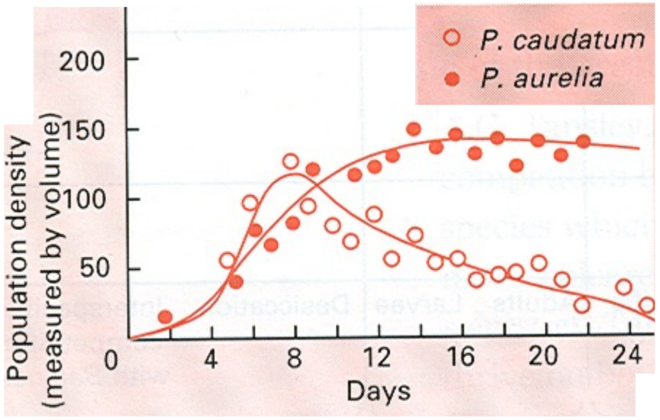
Paramecium aurelia

Paramecium caudatum

Coexistence was observed:

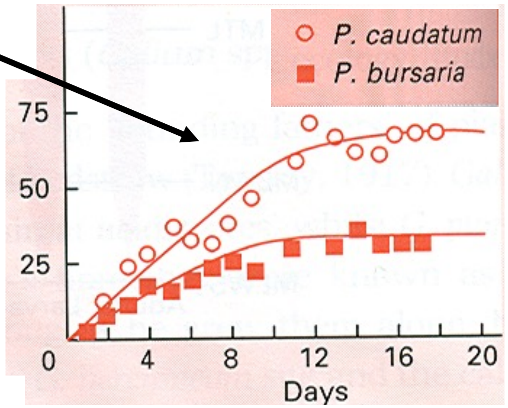
Paramecium caudatum

Paramecium bursaria



Species coexisted below each species' respective individual carrying capacity.

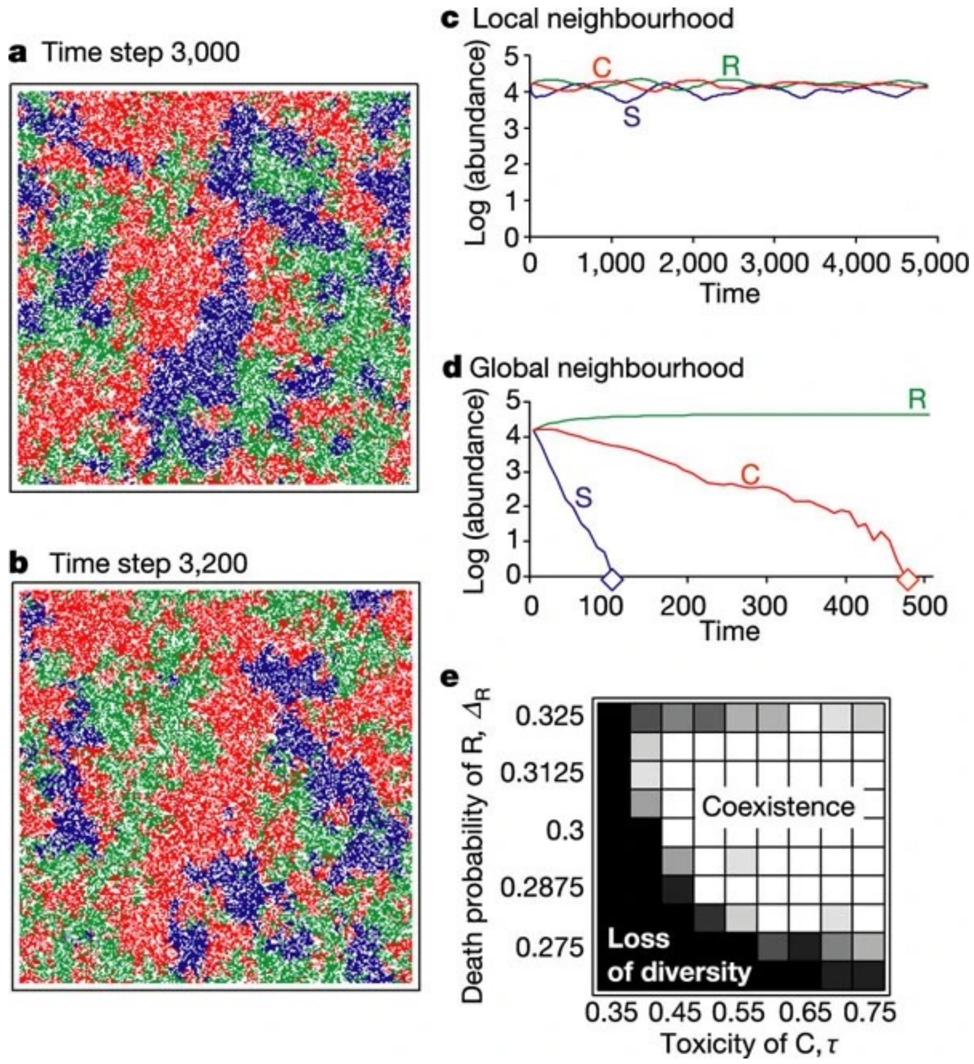
The two coexisting species were largely partitioned in space. *P. bursaria* ate yeast at the bottom and *P. caudatum* consumed bacteria suspended in the medium.



Gause 1934. *J Experimental Biology.*

Gause 1935. *Science.*

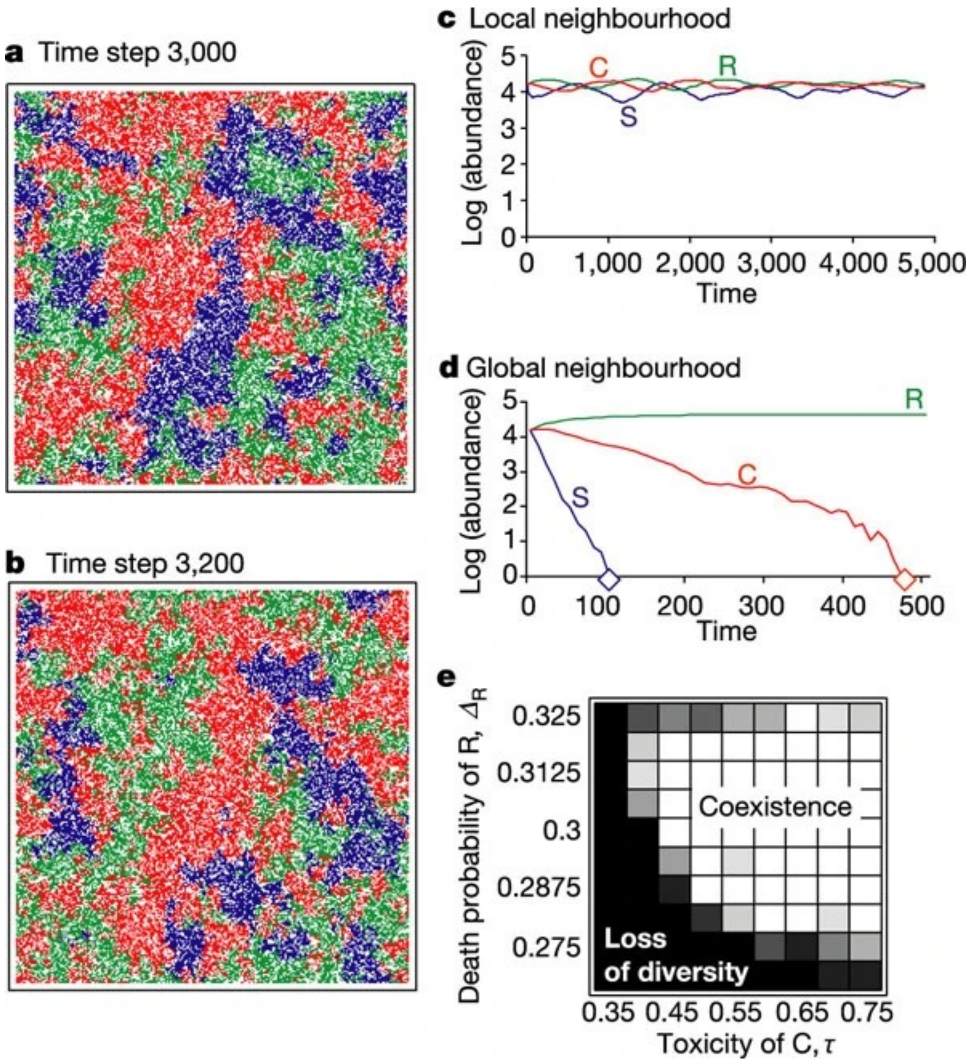
Sometimes **stochasticity** and **space** are all you need to ensure **coexistence** in a competitive environment

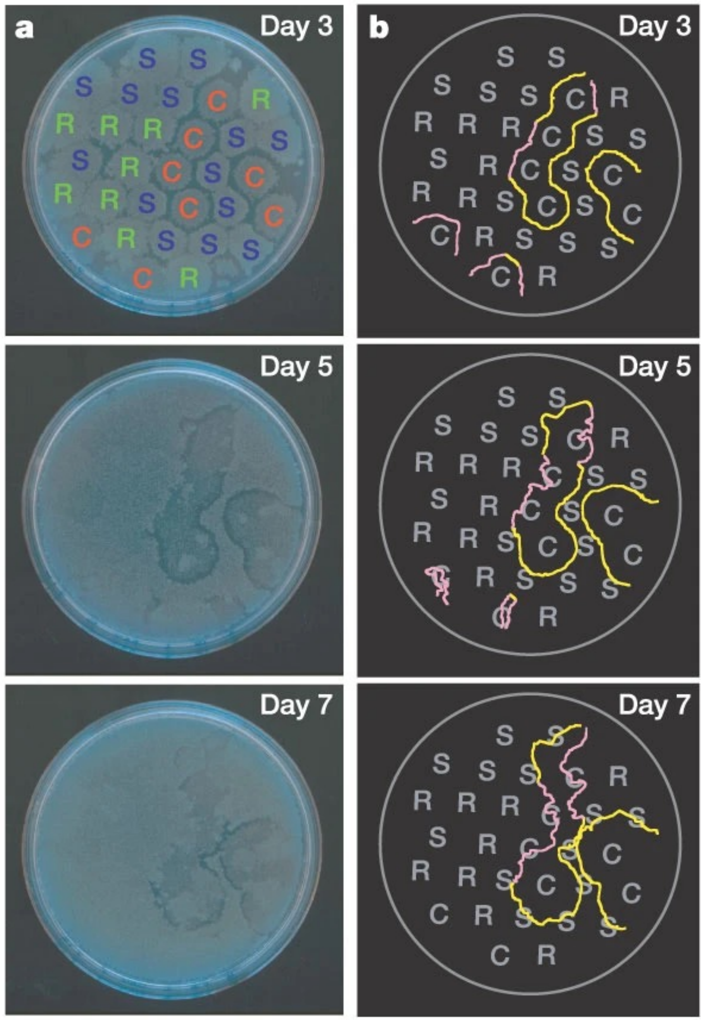


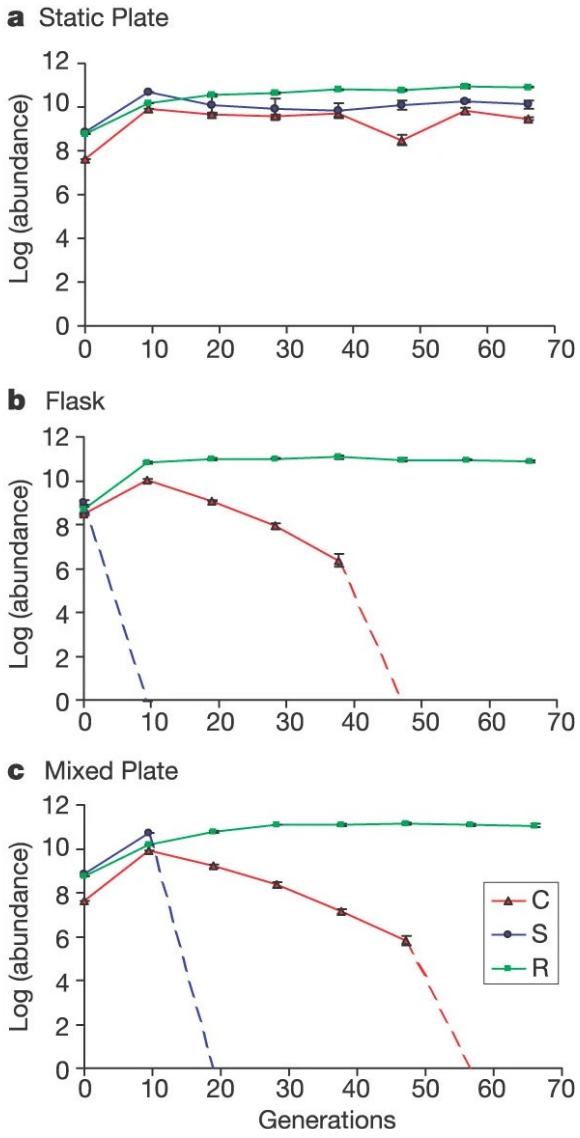
Kerr et al. 2002. *Nature.*

- 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**!



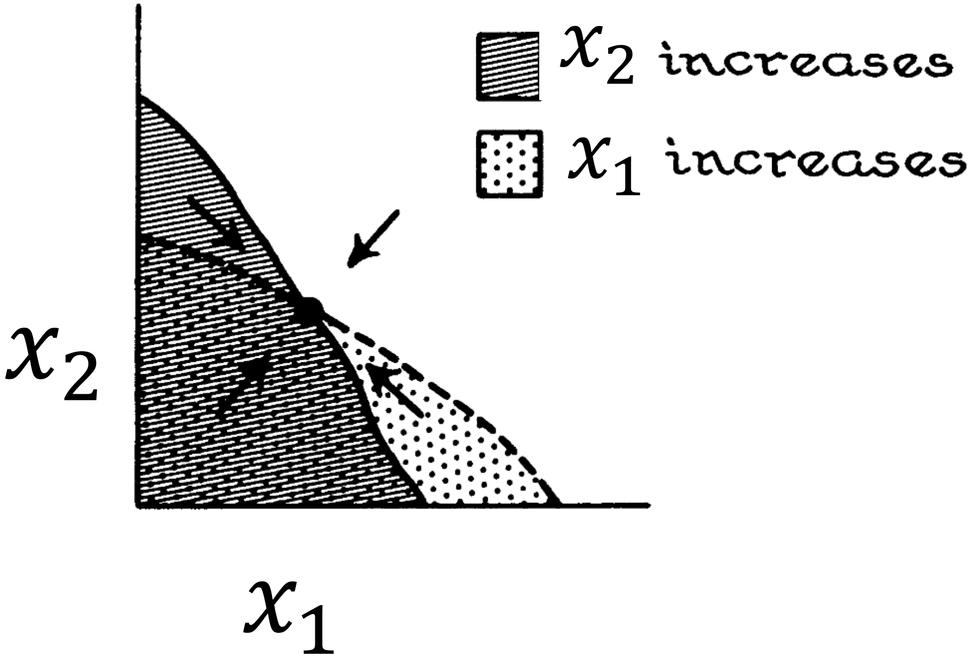




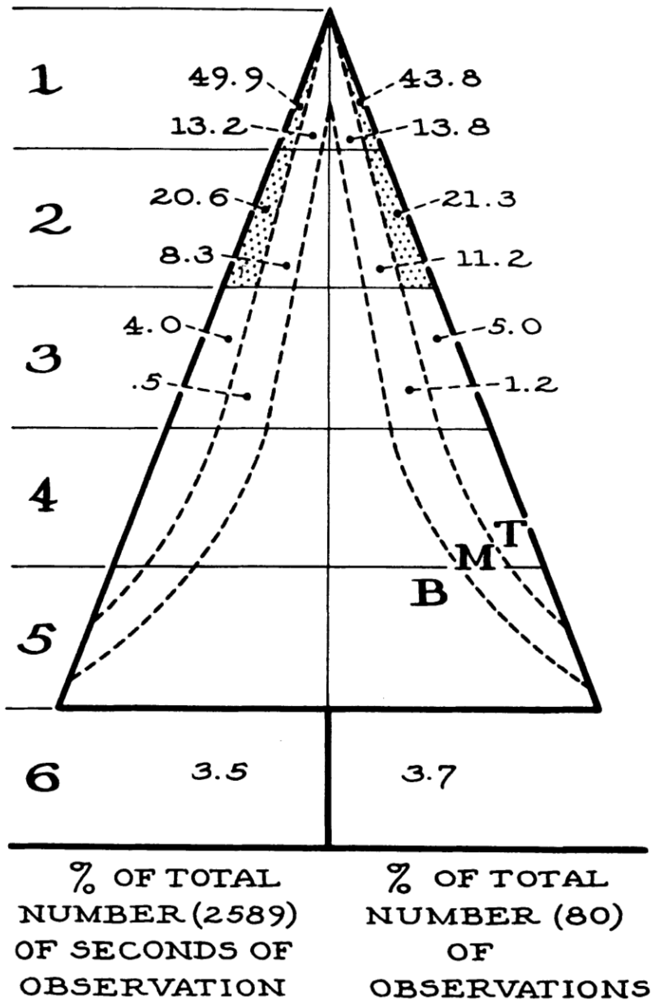
Kerr et al. 2002. *Nature.*

Niche partitioning enables organisms to avoid competitive exclusion

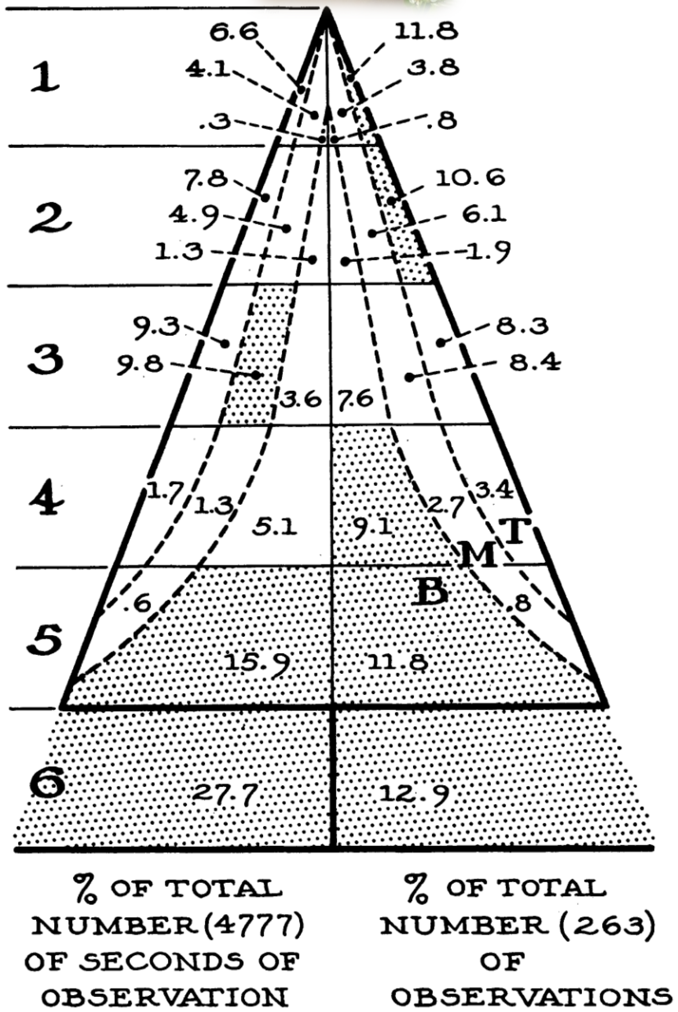
Niche: a match of a species to a specific environmental condition



Cape May Warbler



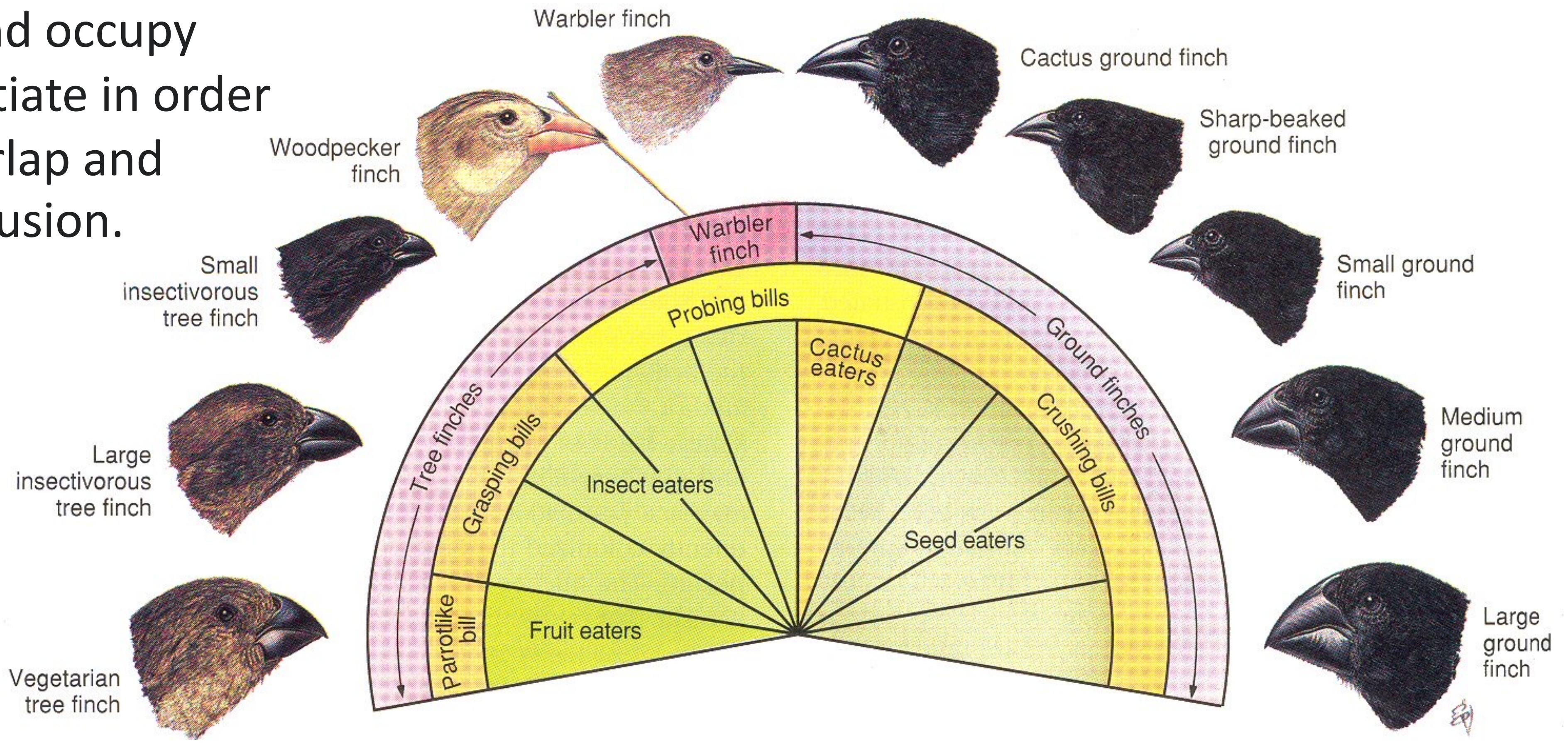
Myrtle Warbler



MacArthur 1968. *Ecology.*

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.



Grant & Grant 2006. *Science.*

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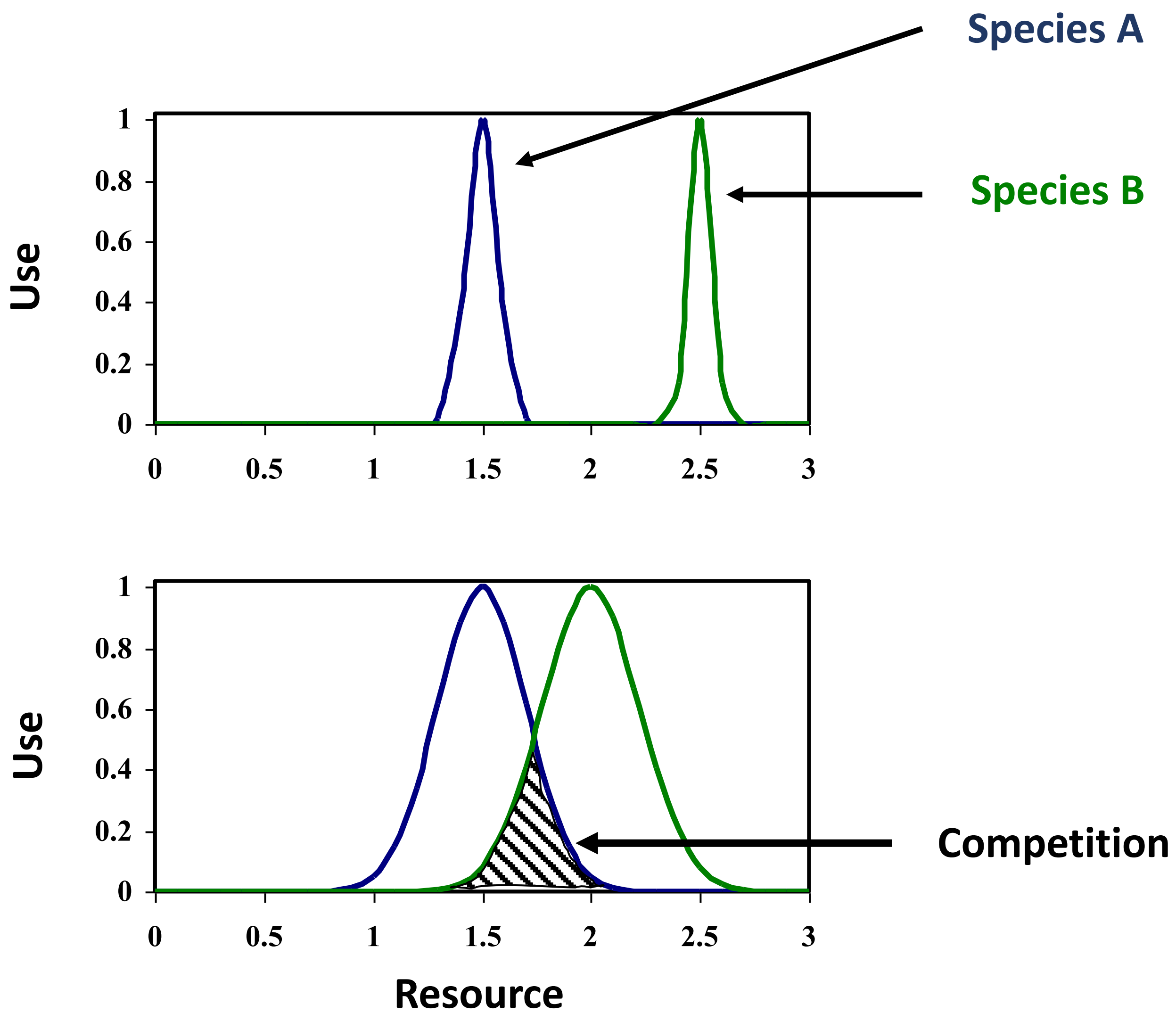

When niches overlap, competition results

Grant & Grant 2006. *Science.*

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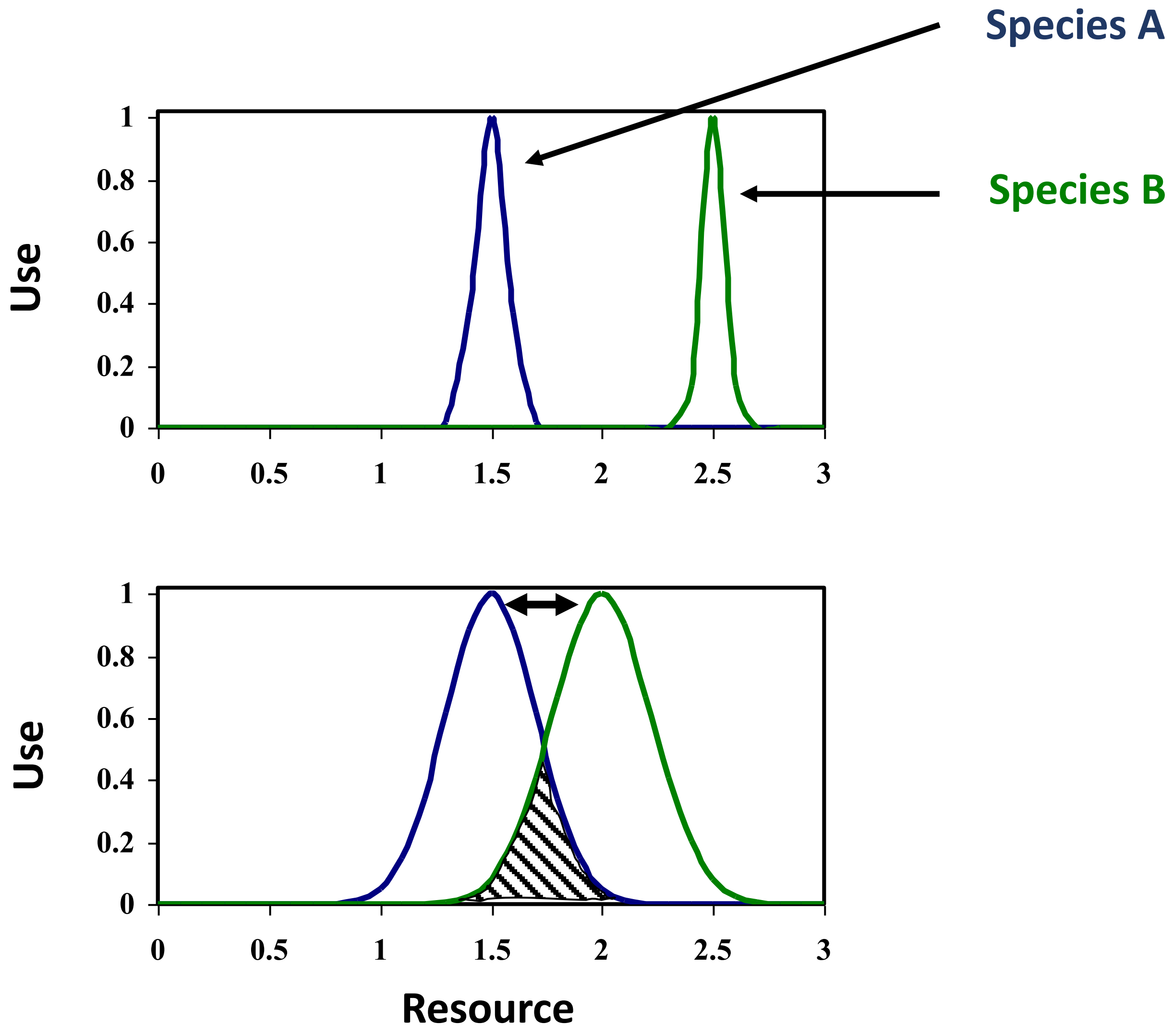
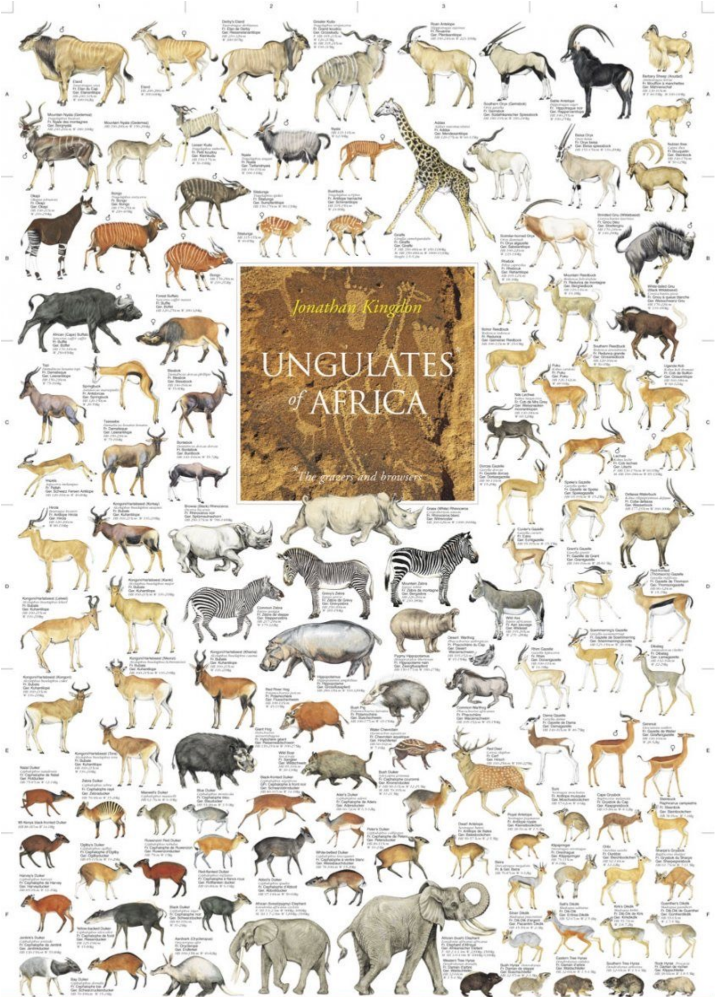
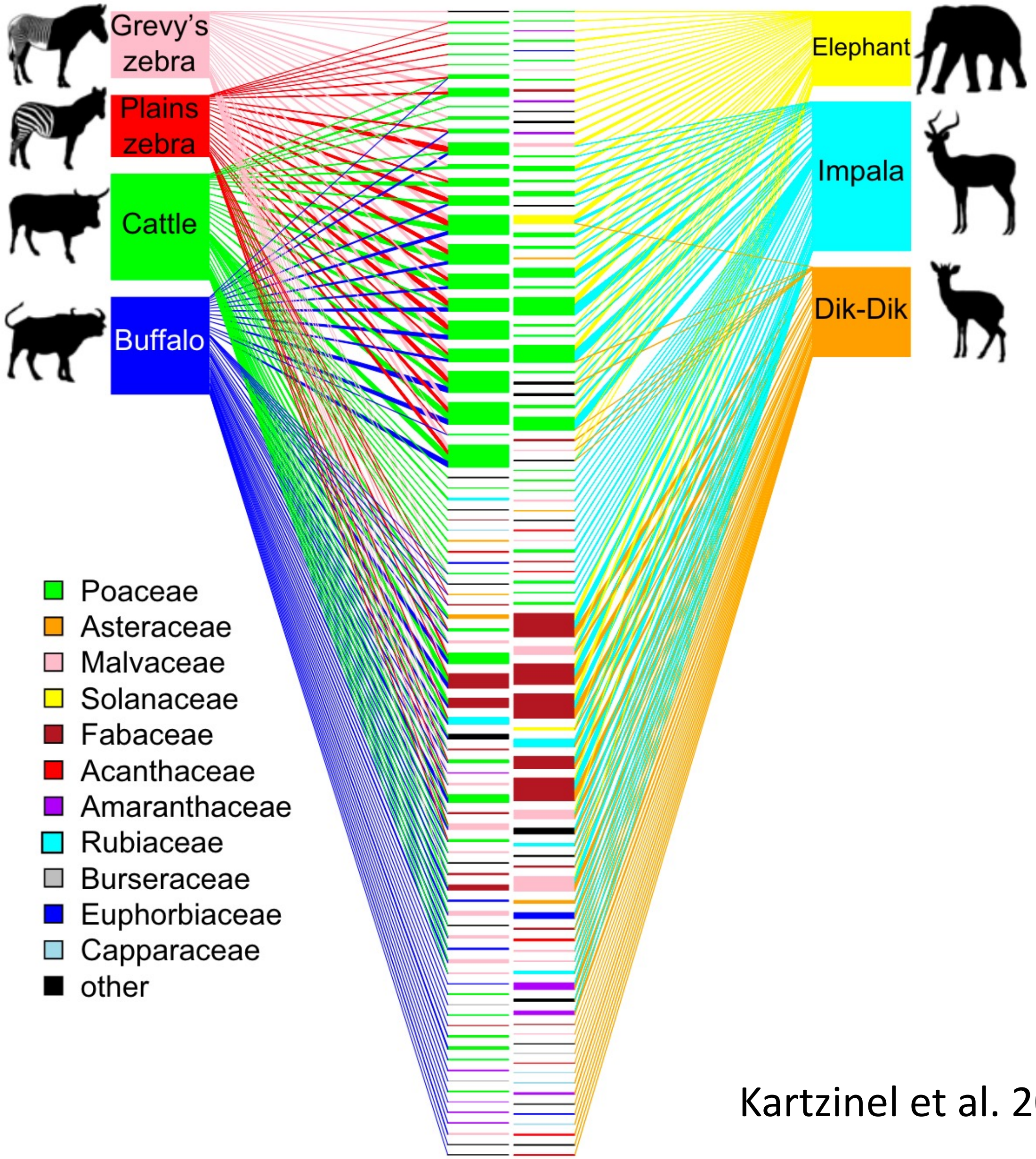


Character displacement forces niches apart.

Niche partitioning enables organisms to avoid competitive exclusion

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

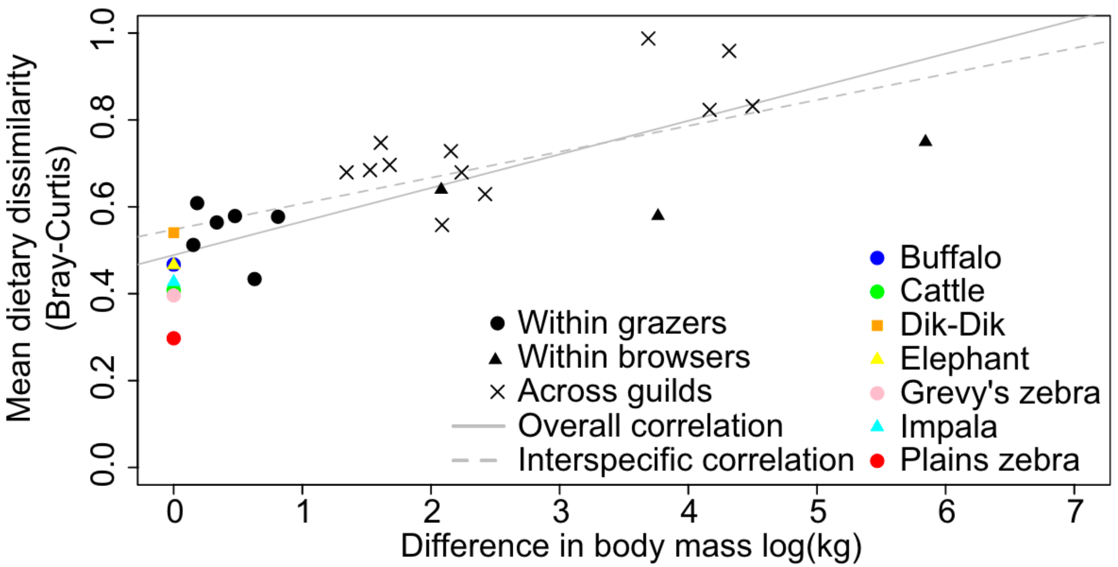


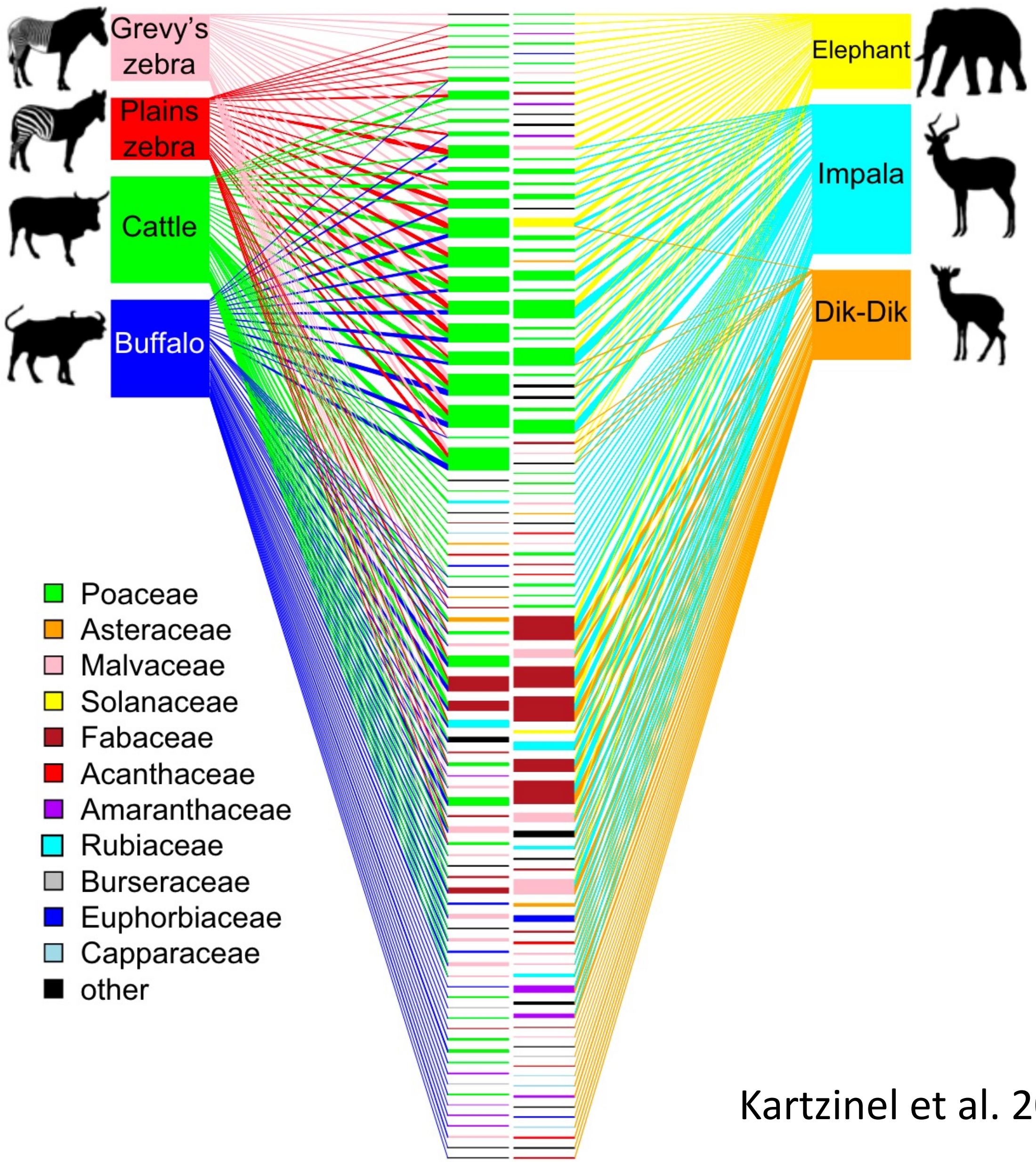


Kartzinel et al. 2015. *PNAS.*

Niche partitioning enables organisms to avoid competitive exclusion

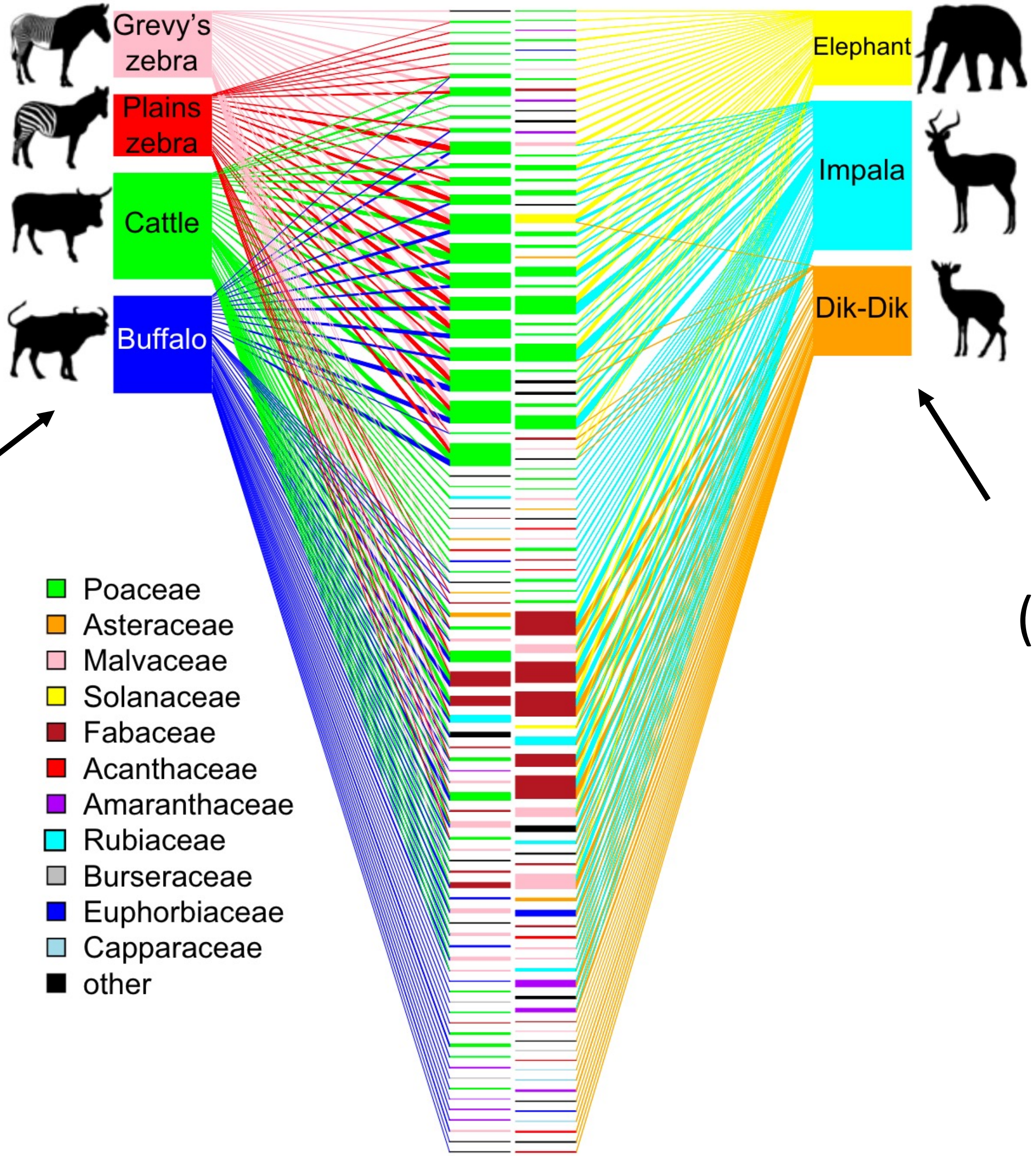
Dietary overlap is higher in similar-sized mammals





Kartzinel et al. 2015. *PNAS.*

Consumer resource partitioning in the African savanna

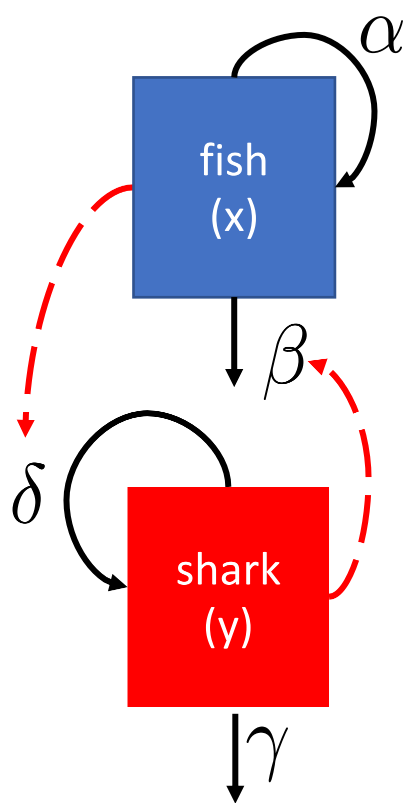


Grazer (generalist)

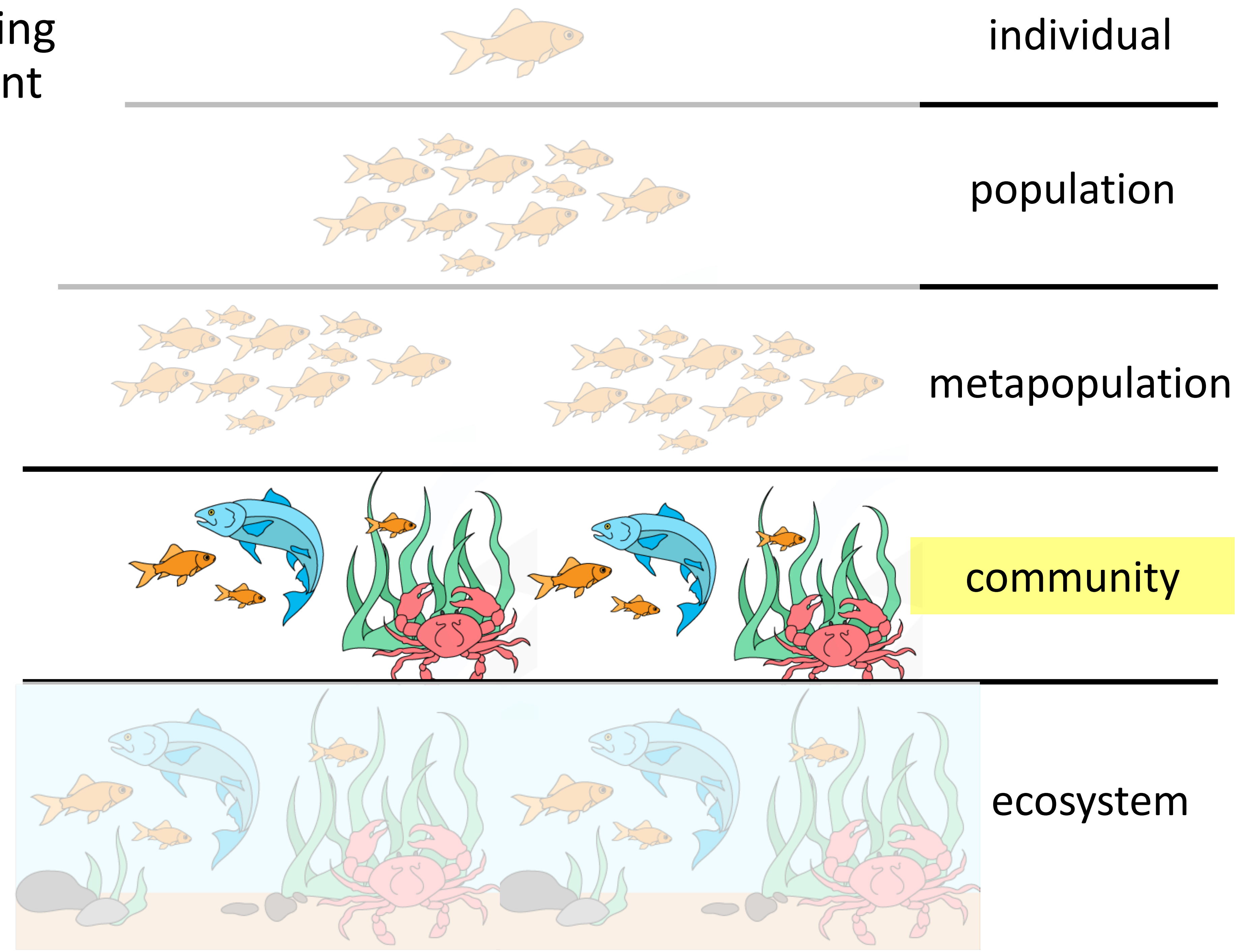
Browser (specialist)

Kartzinel et al. 2015. *PNAS.*

Community = interacting populations of different species



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



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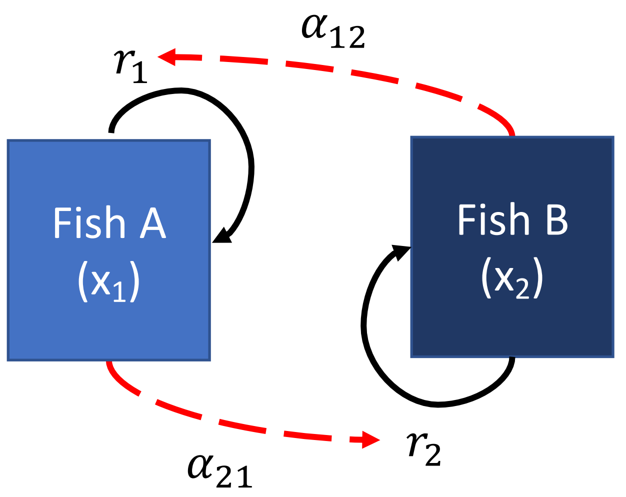
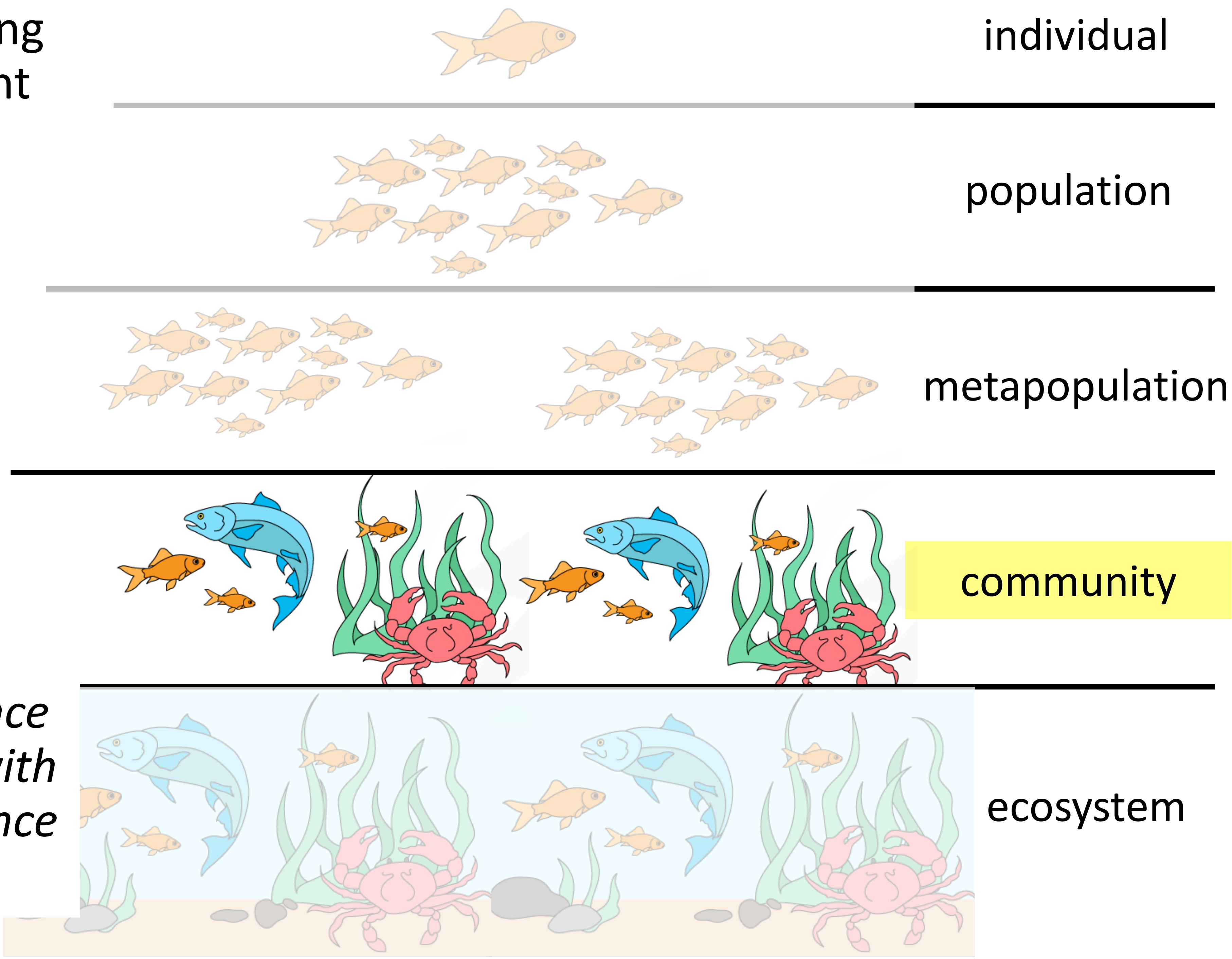


*How does the abundance of **fish species A** vary with changes in the abundance of **fish species B**?*



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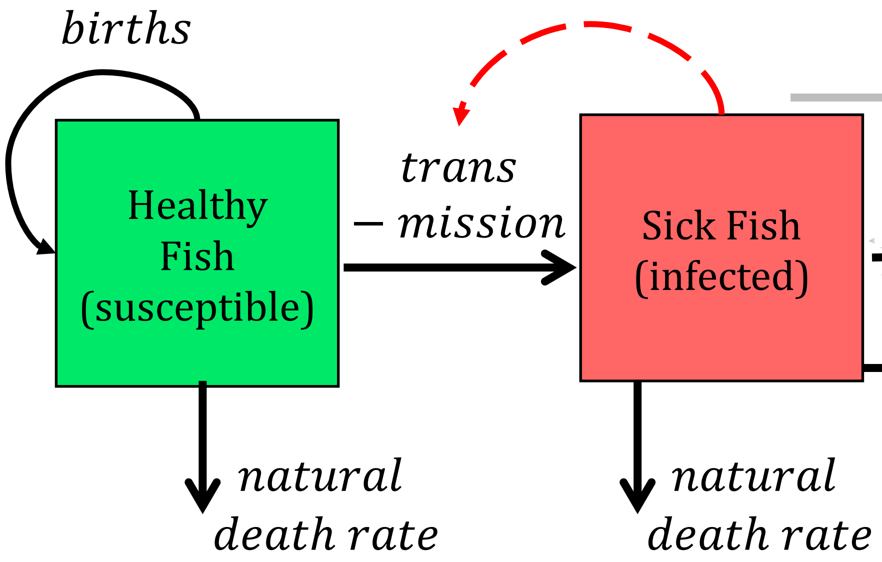
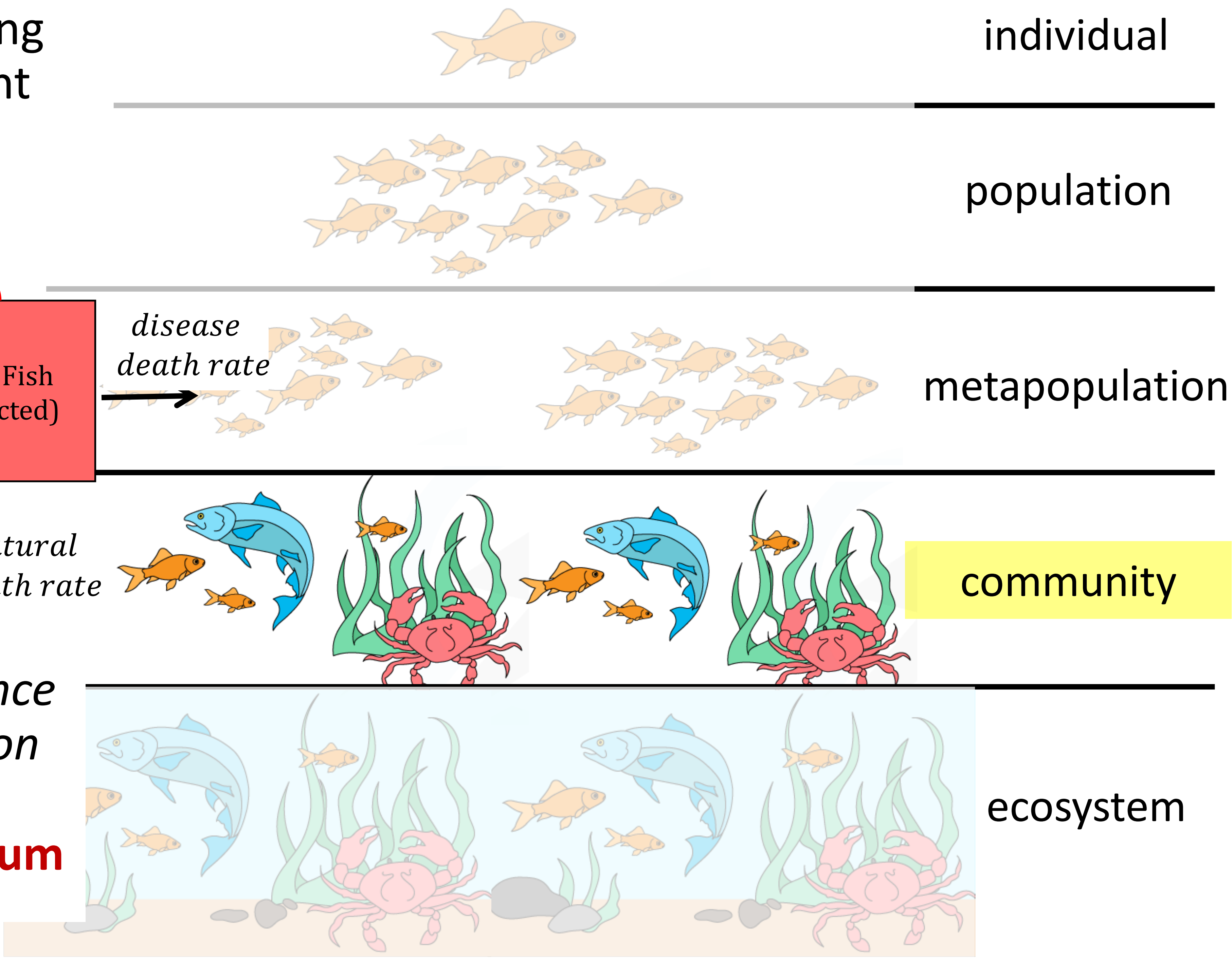
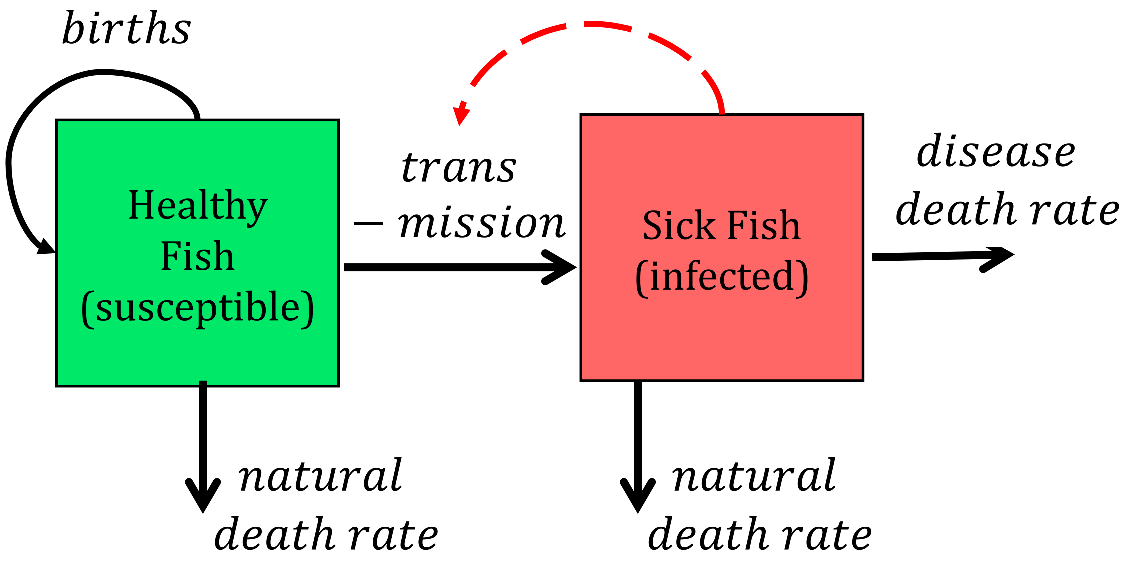




*How does the abundance of **fish** change based on **infection with Mycobacterium marinum** (wasting disease)?*

Community = interacting populations of different species



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

Species can interact in several distinct ways.

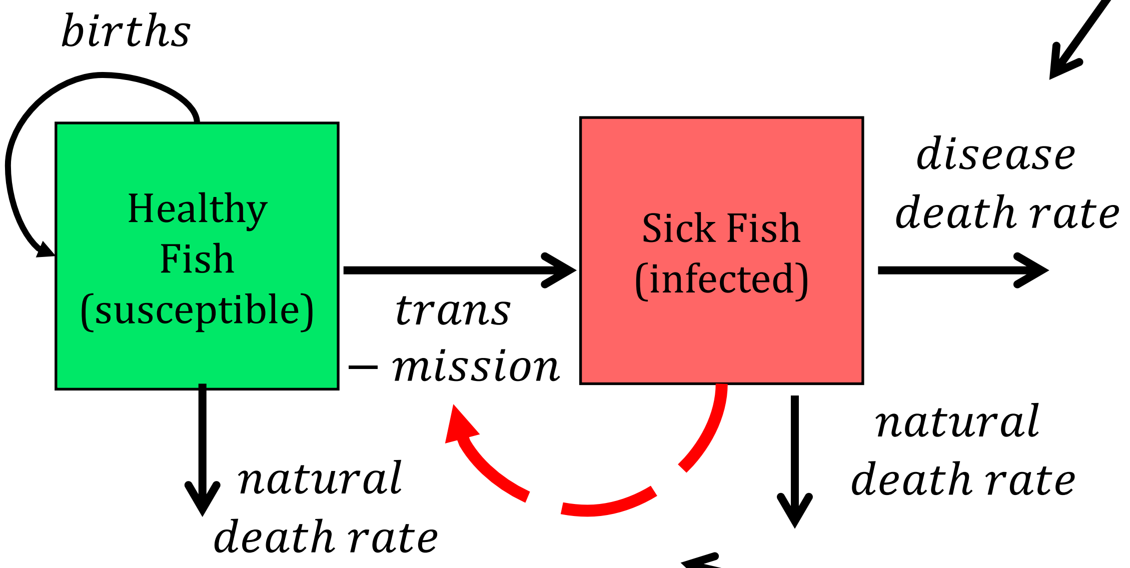
- **Mutualism** – both species benefit
- **Commensalism** – one species benefits, the other is unaffected
- **Predation** – one species benefits, the other is harmed (eaten!)
- **Competition** – two species compete for the same limiting resource, both harmed by the interaction
  - Direct = wolves and 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!

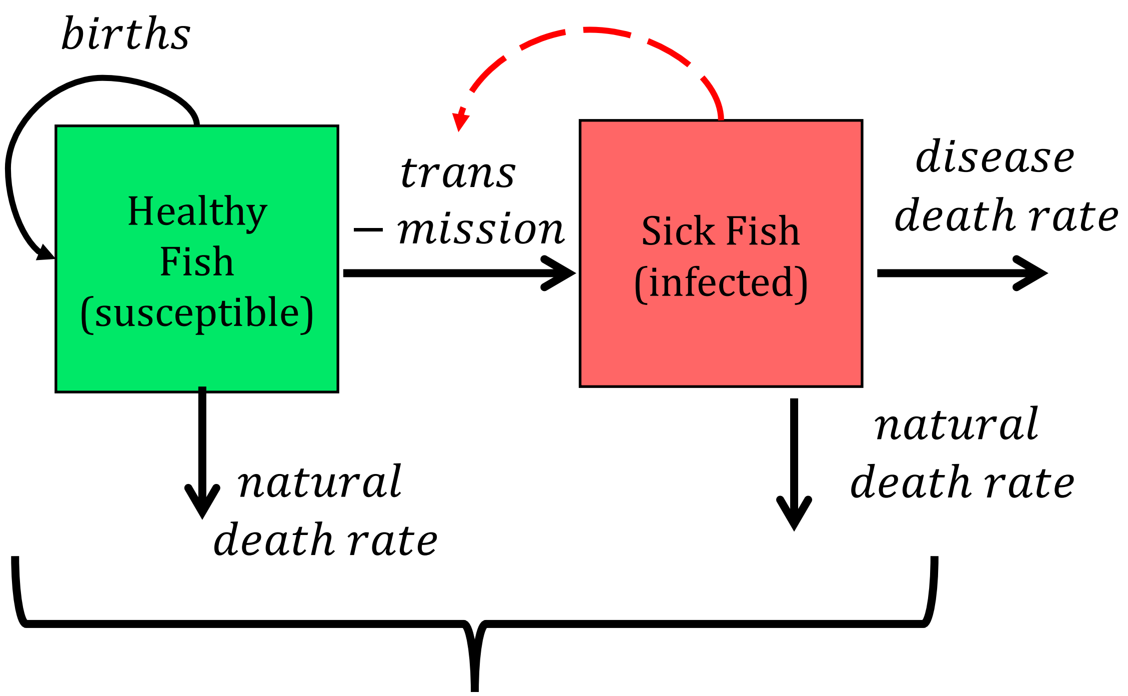


Solid lines are processes by which individuals in each population move between boxes.

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

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

Population Biology



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

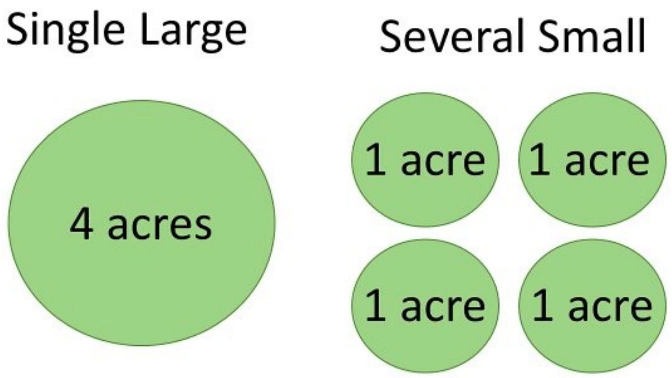
In disease ecology, we model populations based on their **infection status**.

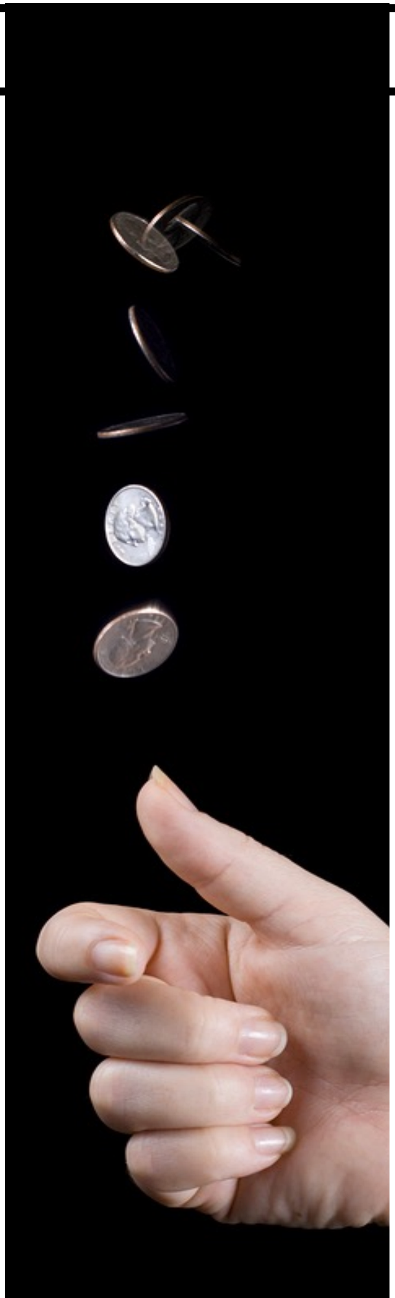
Previously, we modeled populations of **different species**, or of **distinct life history classes within a species**.

Population Biology

Conservation Biology

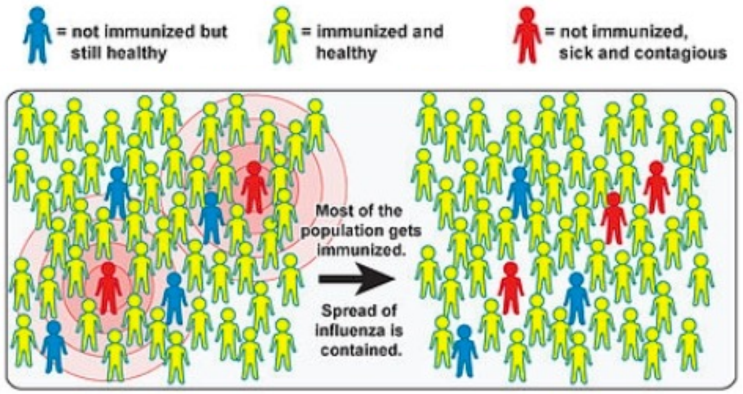
- **Goal:**
  - protect **populations** from **extinction**
- **Concept:**
  - **Minimum Viable Population** size (MVP)
- **Approach:**
  - protected area **reserves**





Disease Ecology

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



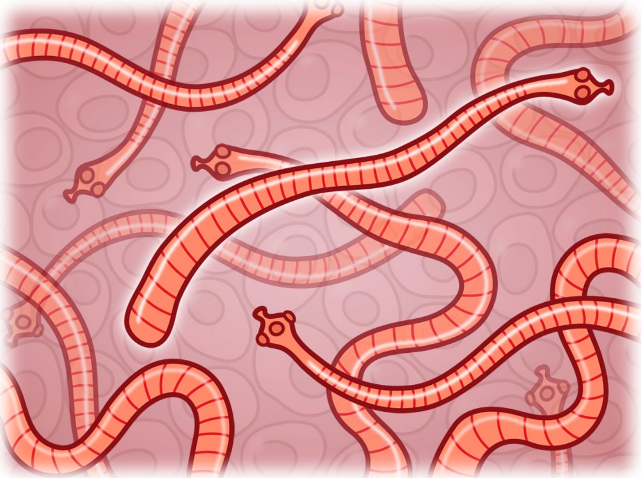
E^2M^2: Ecological and Epidemiological Modeling in Madagascar

December 16, 2022

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)



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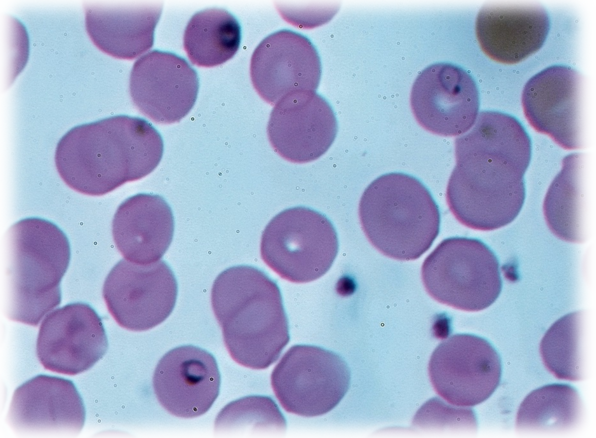
- Pathogen: a microorganism that can cause disease
  - Ex: bacterium, virus, protozoan

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



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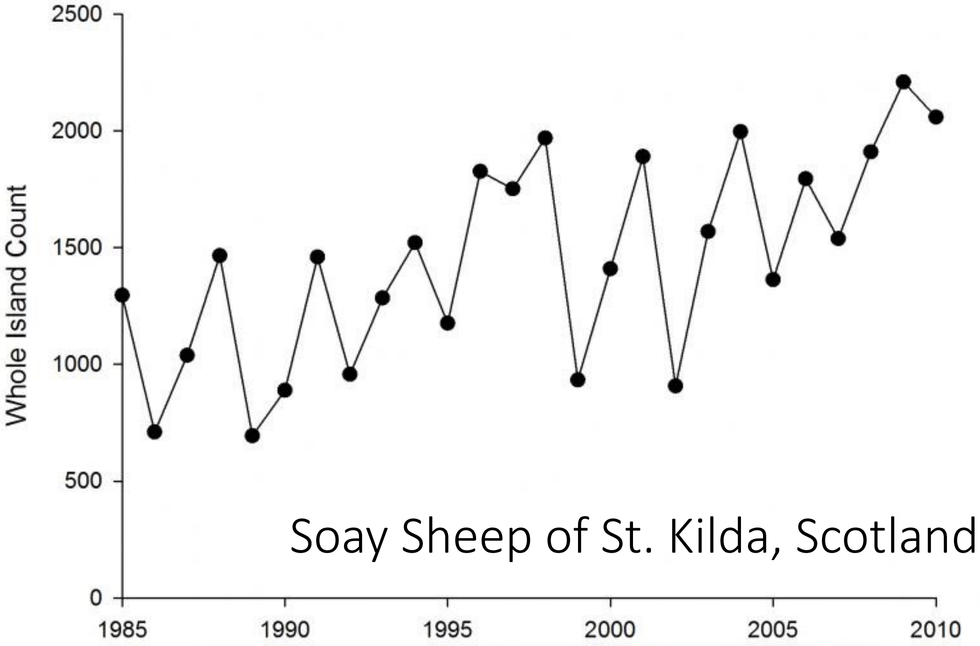
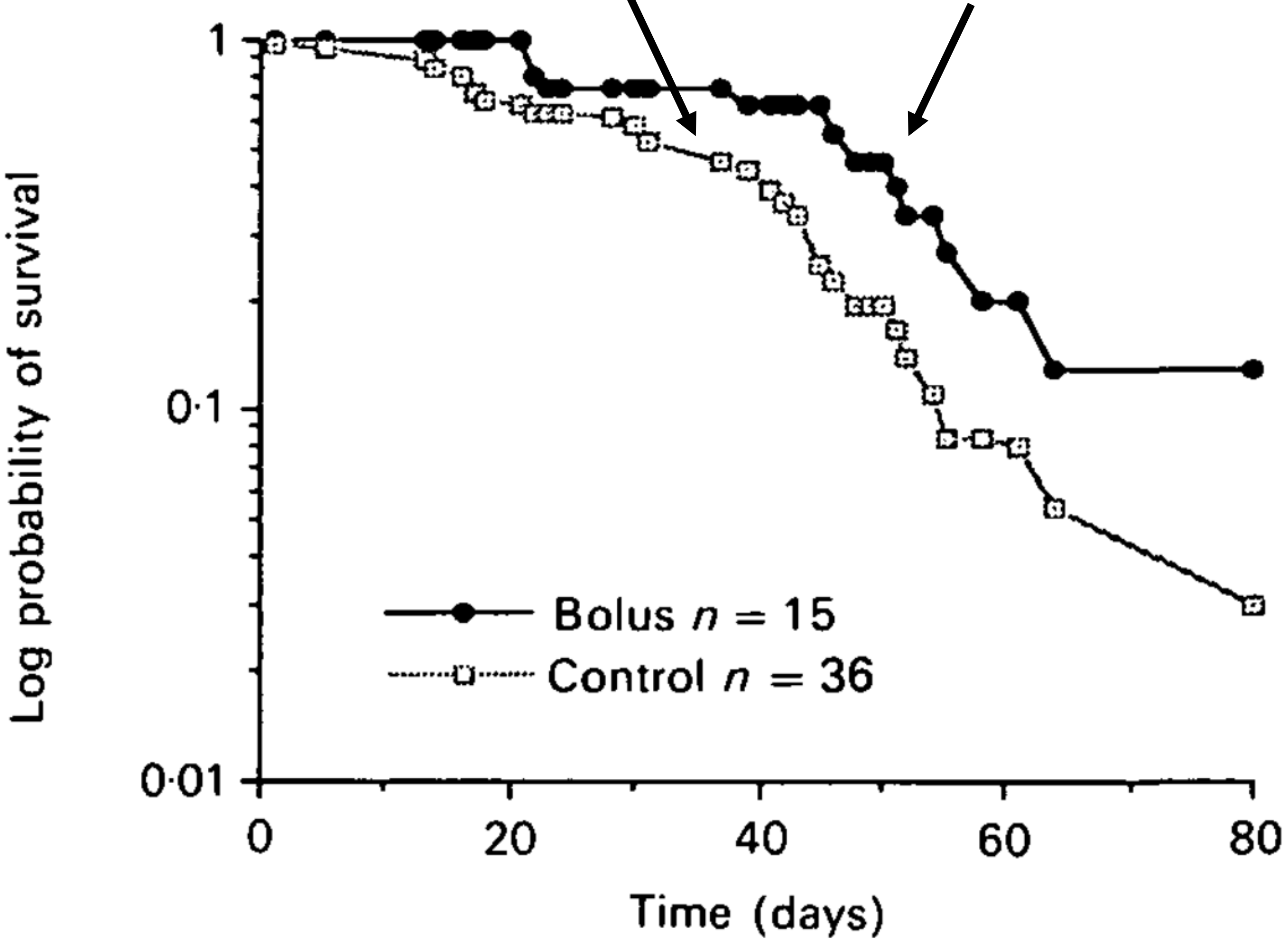
- Host: the organism <u>*on/in which*</u> the parasite/pathogen lives
  - Ex: Soay sheep, grapes
- Vector: an arthropod agent that carries and transmits a pathogen or parasite from host to host
  - Ex: mosquitoes, ticks, fleas

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



male juveniles with nematodes

male juveniles treated with antihelminthic



Gulland 1992. *Parasitology.*

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

Hudson 1986. *J Animal Ecology.*
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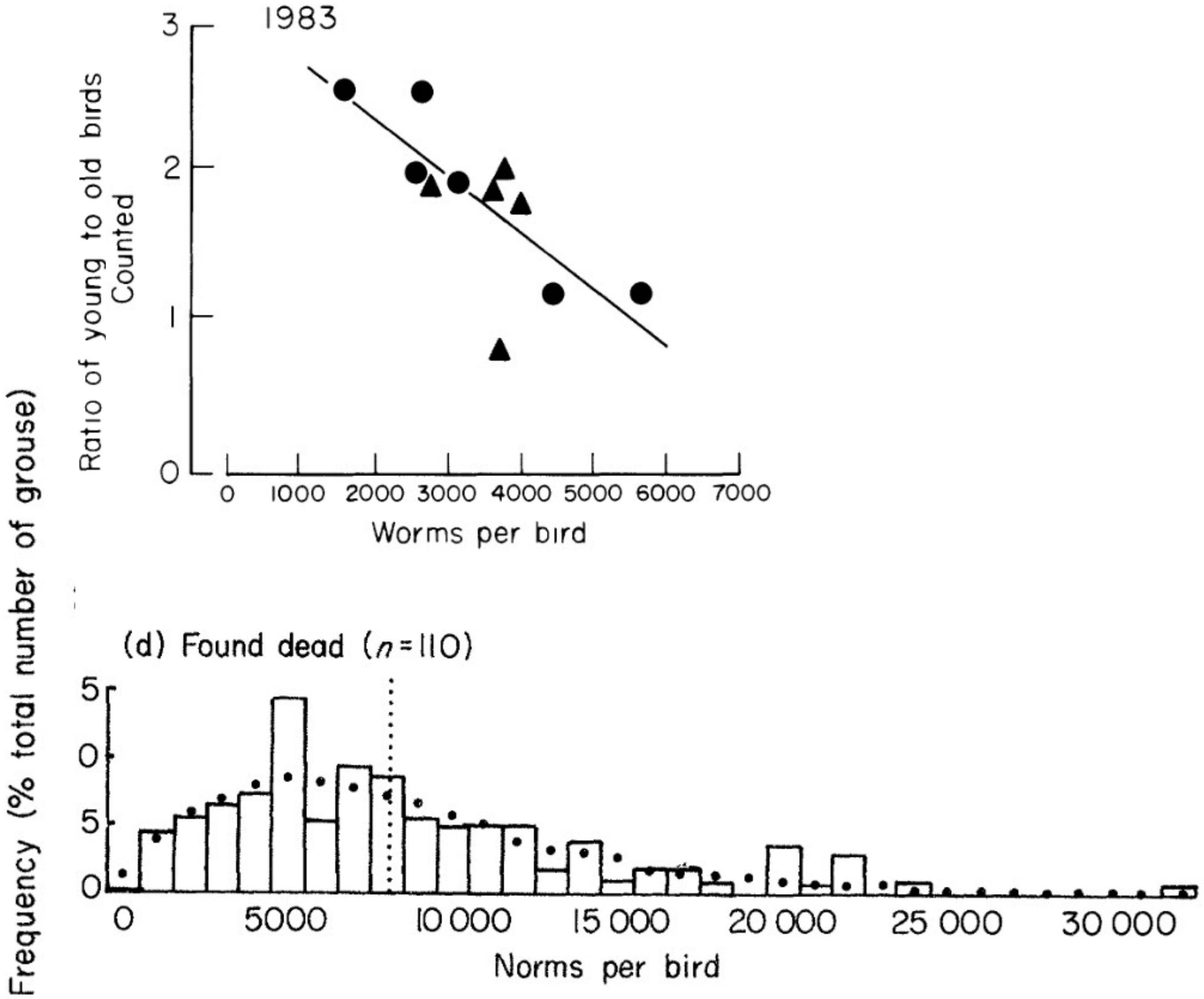

Higher burden of the intestinal strongyle worm, *Trichostrongylus tenuis,* both reduces breeding success and increases mortality in red grouse.

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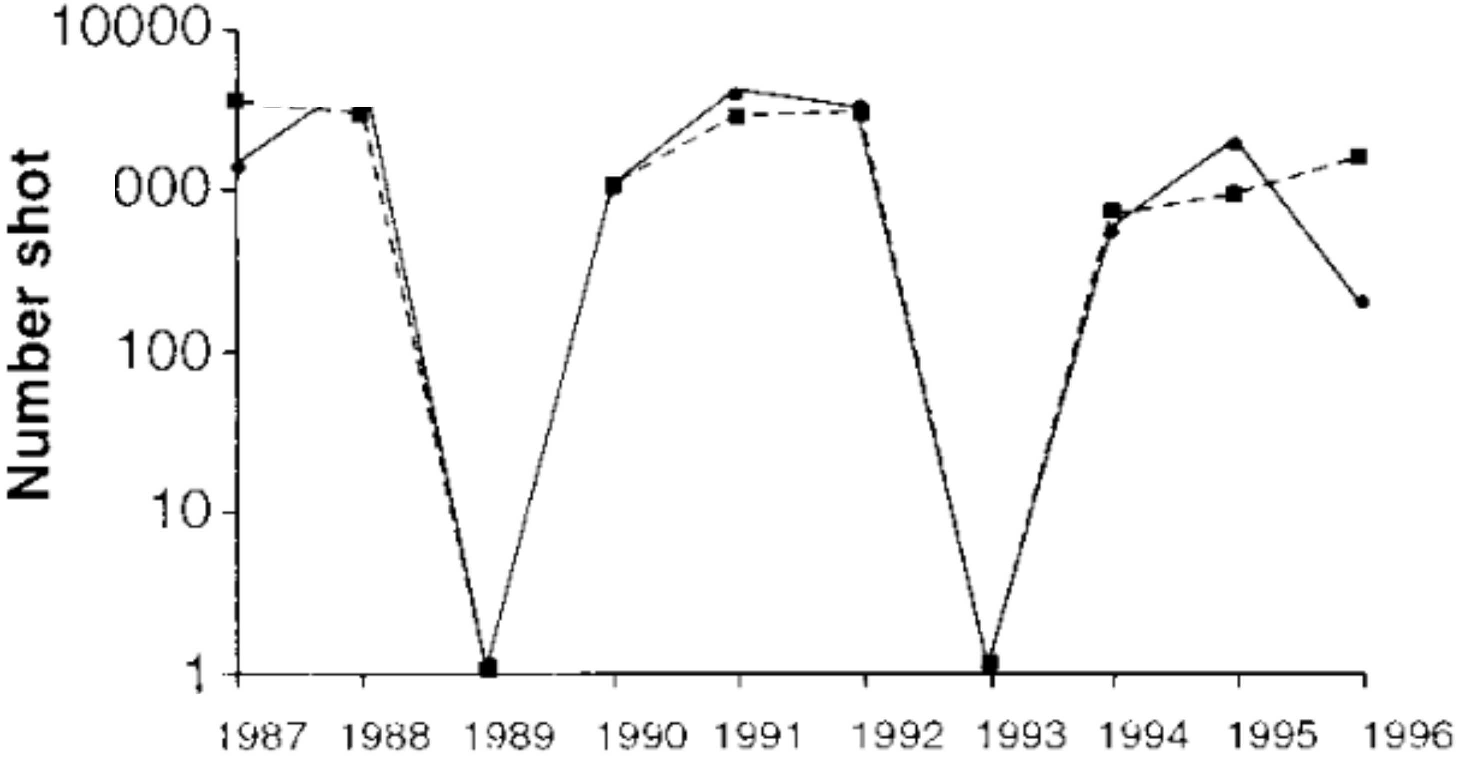
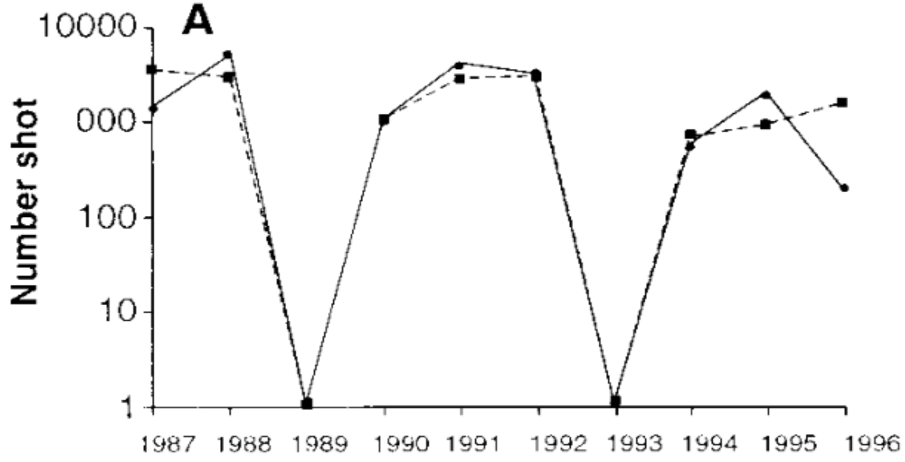
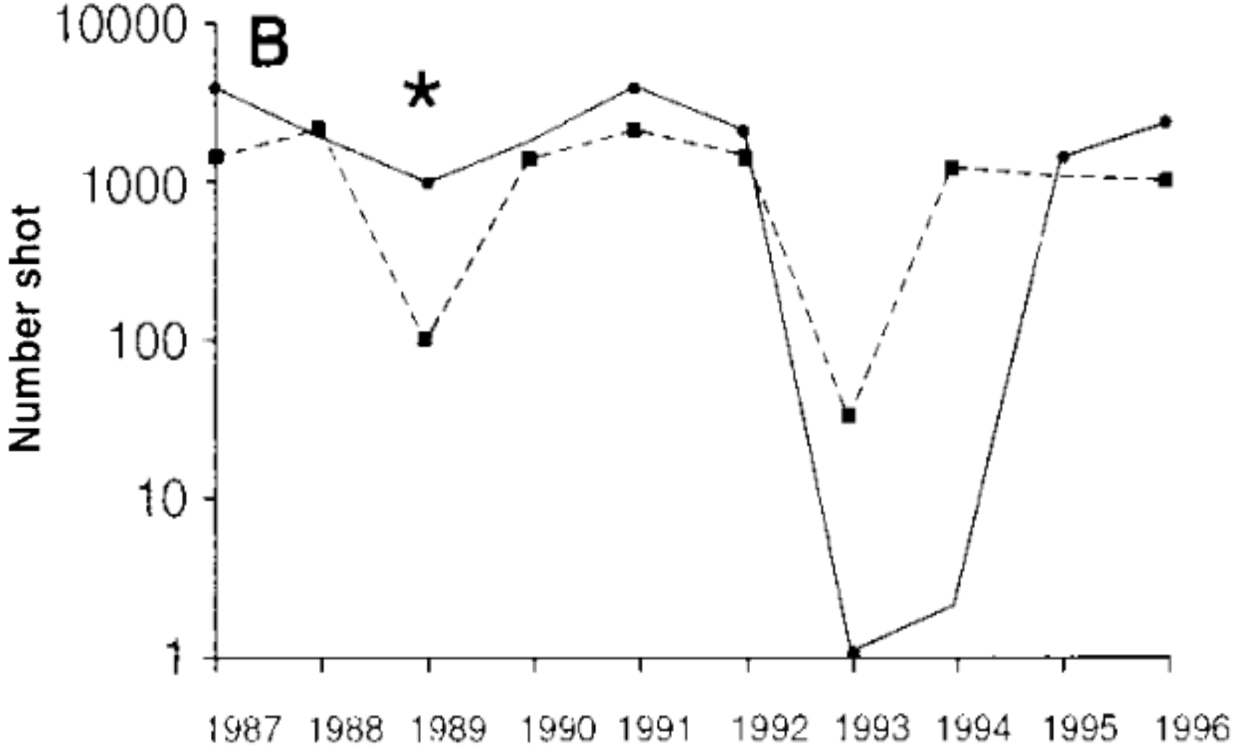
Worms were hypothesized to be responsible for the observed population cycles in ‘bag data’ from northern England.

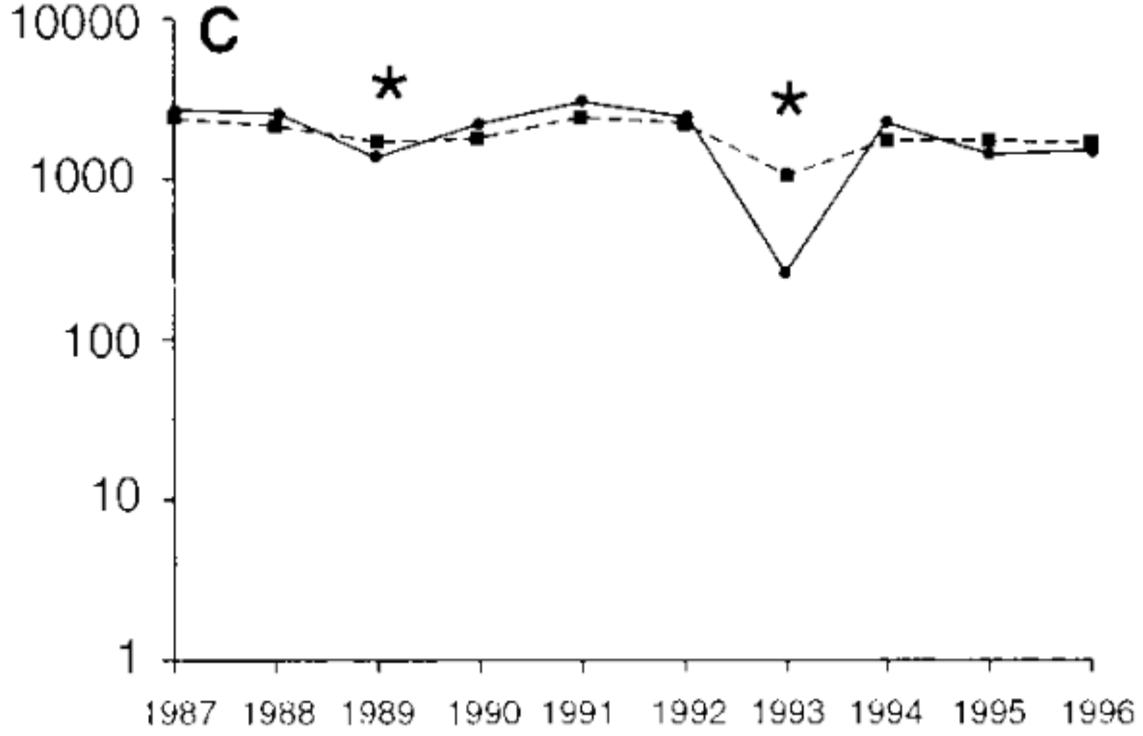


Hudson et al 1998. *Science.*



Deworming eliminated population cycles to prove this effect!





Hudson et al 1998. *Science.*

Parasites and pathogens have also shaped human history.

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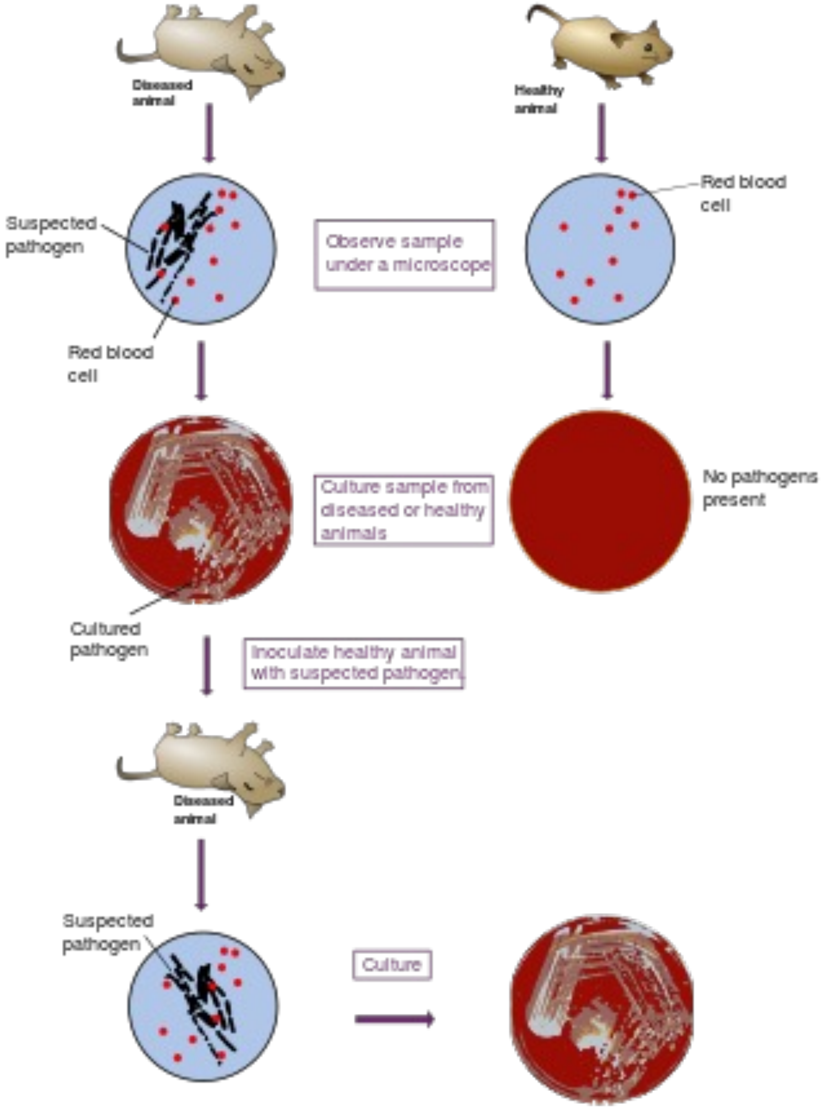
But we have not always known they were responsible for disease!

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



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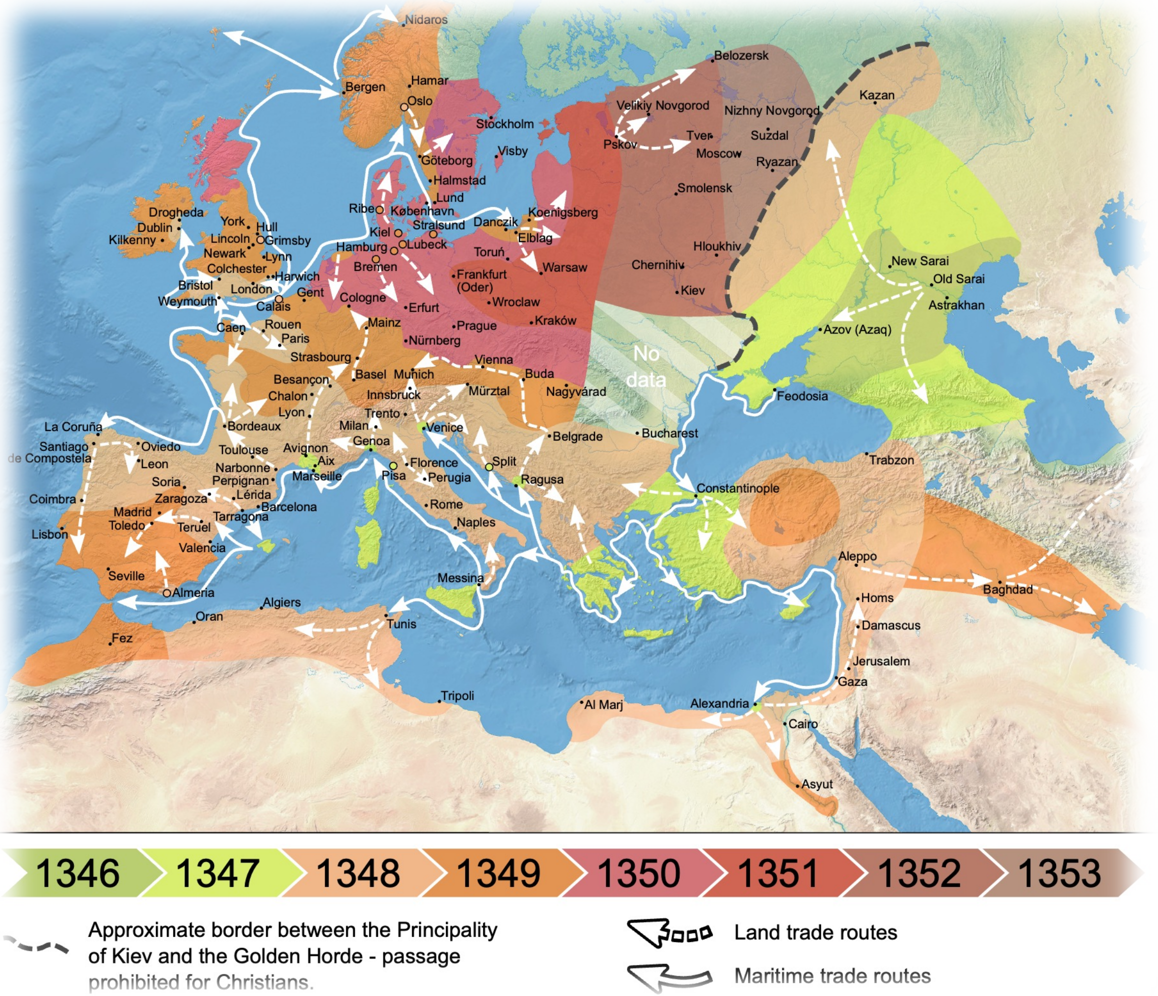
   - Leuwenhoek’s microscope (1675)
   - Koch’s postulates (1890)
4. **<u>Classic epidemiology</u>**
   - Risk factors for disease = John Snow (1854)

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

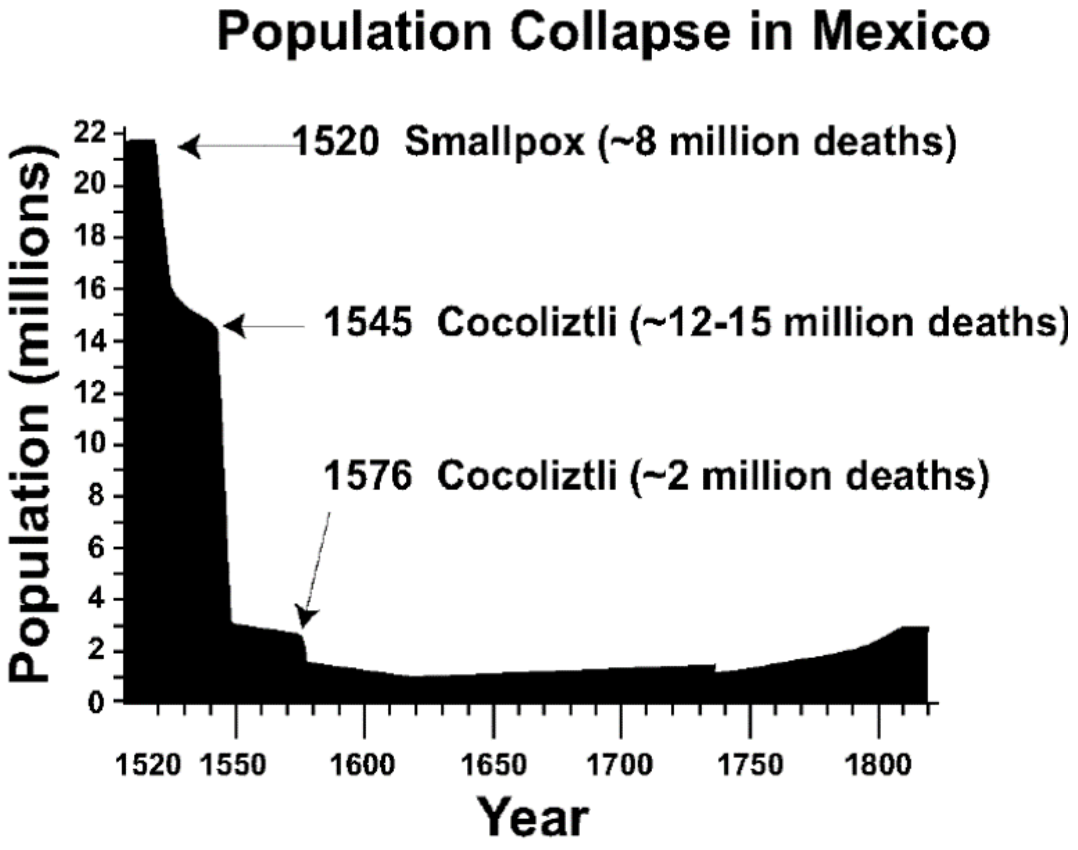
Parasites and pathogens have also shaped human history.

- 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

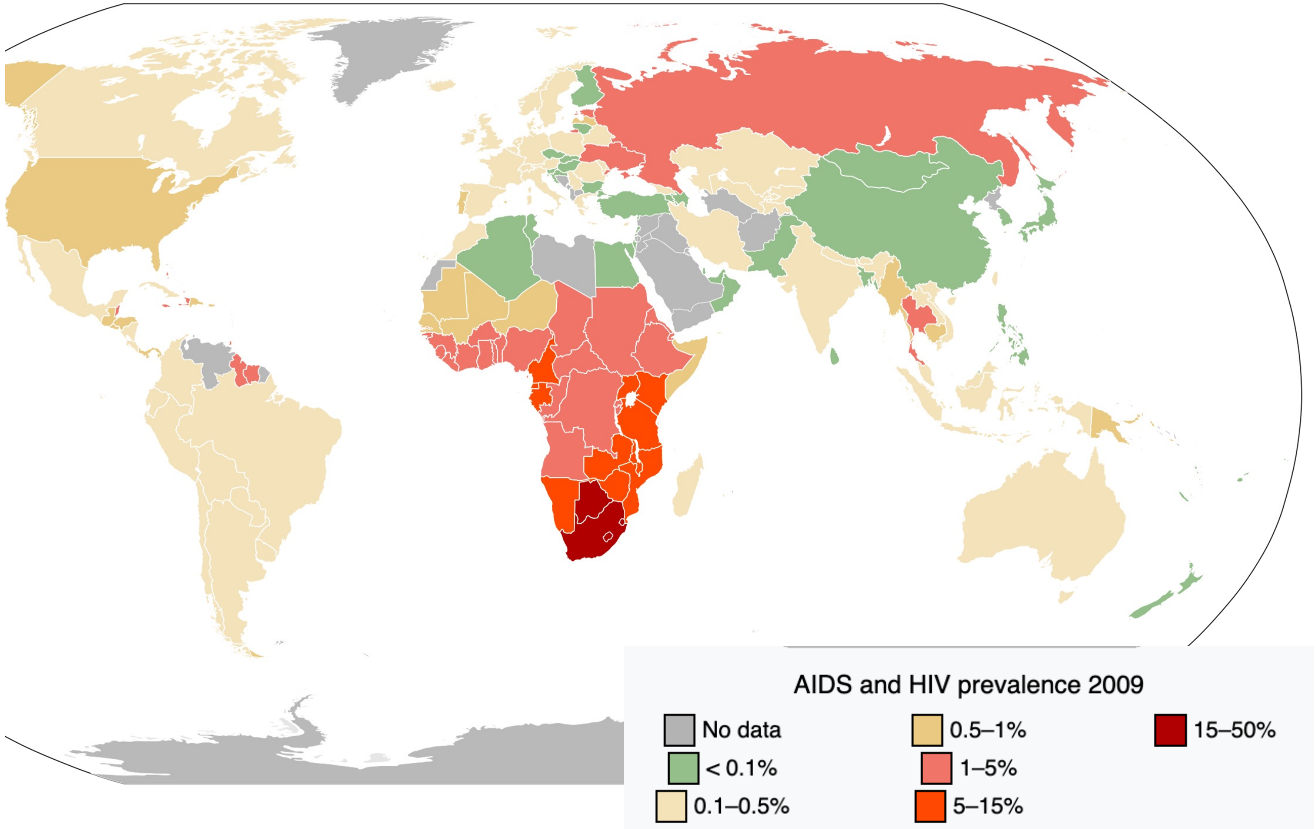


Parasites and pathogens have also shaped human history.

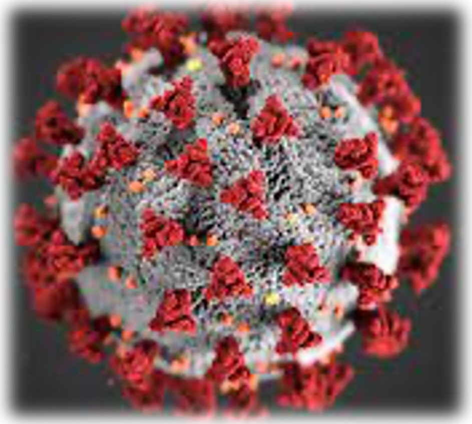
- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
- Spanish Influenza (1918-1920)
  - 17-100 million deaths worldwide.
  - 1-5% of global population
  - 2nd-most devastating pandemic in history (after Black Death)

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.

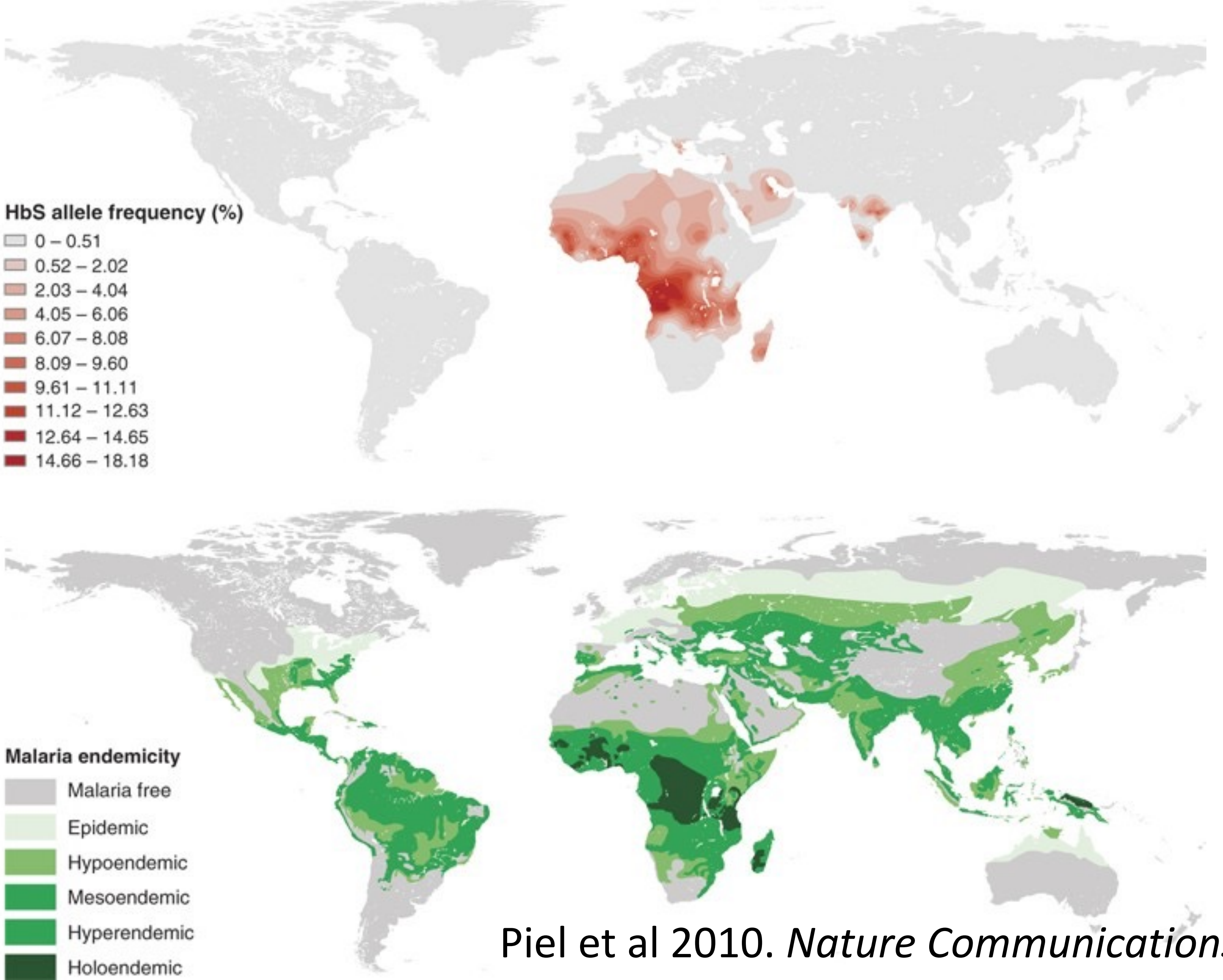


- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
- Spanish Influenza (1918-1920)
- HIV (~1960-now)
- COVID-19 (2019-now)
  - ~7-29 million deaths worldwide
  - ~0.1-0.4% of population

Parasites and pathogens have also shaped human DNA.

Sickle cell anemia

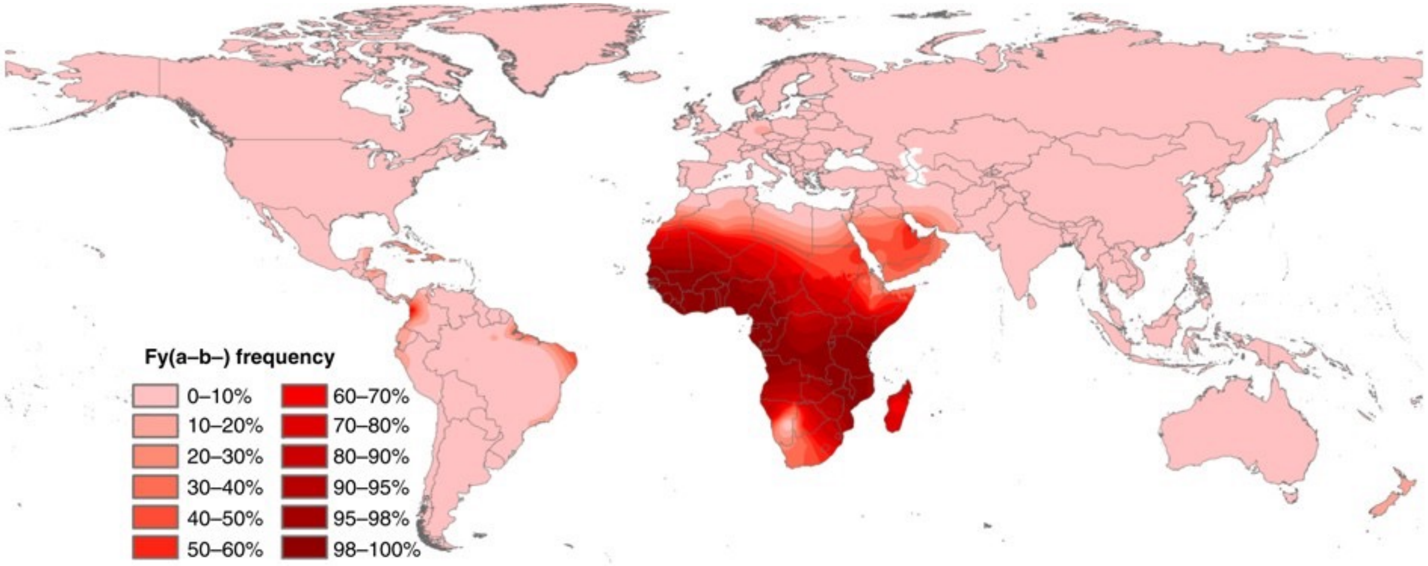
- The HbS allele confers resistance to malaria but also results in sickle cell anemia when homozygous.
- Natural selection has favored this trait in malaria-endemic regions of the planet.
- As of 2021, WHO estimates 247 million malaria cases worldwide and >600,000 deaths, 95% in Africa.
- Children <5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



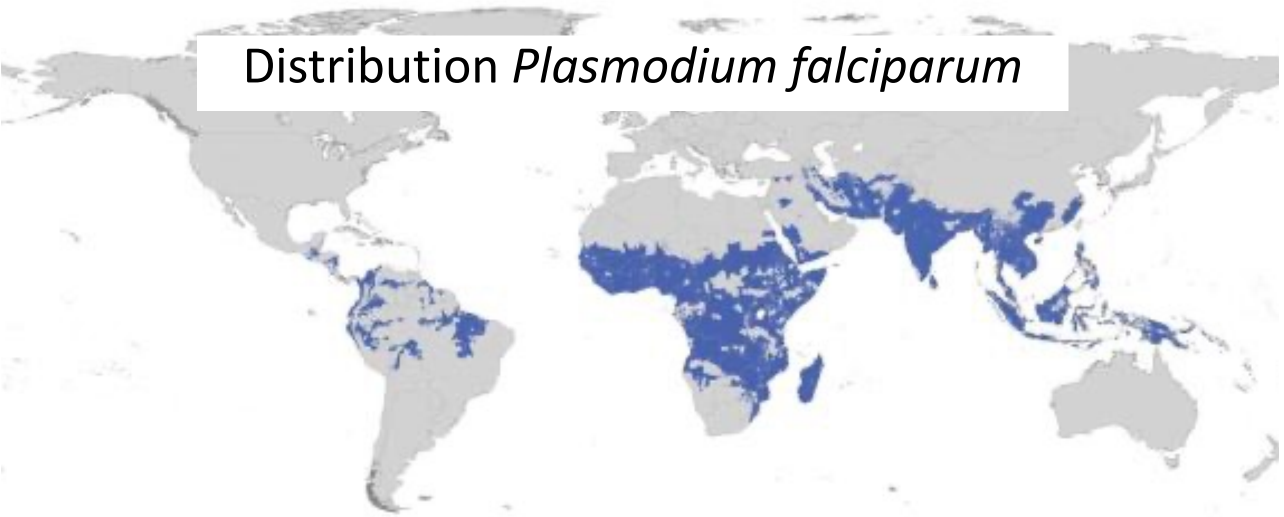
Piel et al 2010. *Nature Communications*.

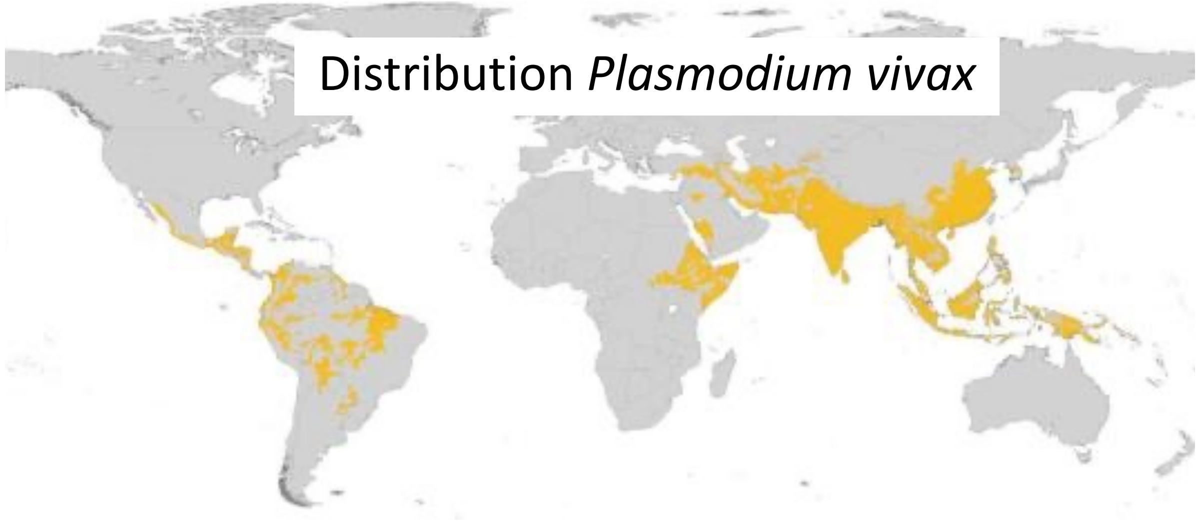
Parasites and pathogens have also shaped human DNA.

Duffy antigen



- Modeled distribution of Duffy-negative human population





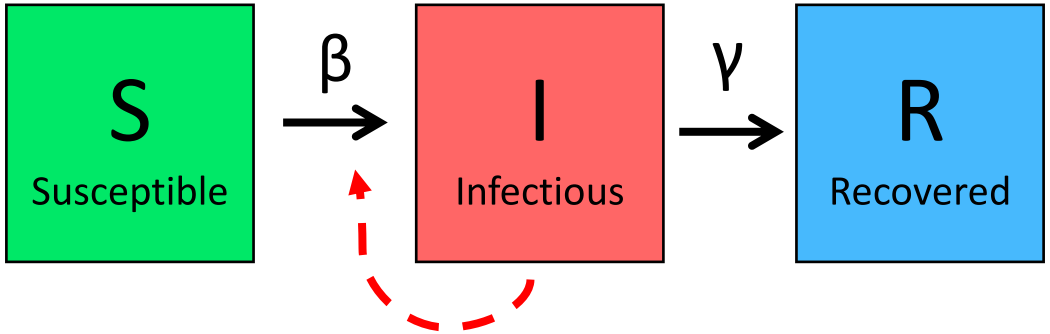
Guerra et al. 2006. *Trends in Parasitology*
Howes et al 2011. *Nature Communications.*

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

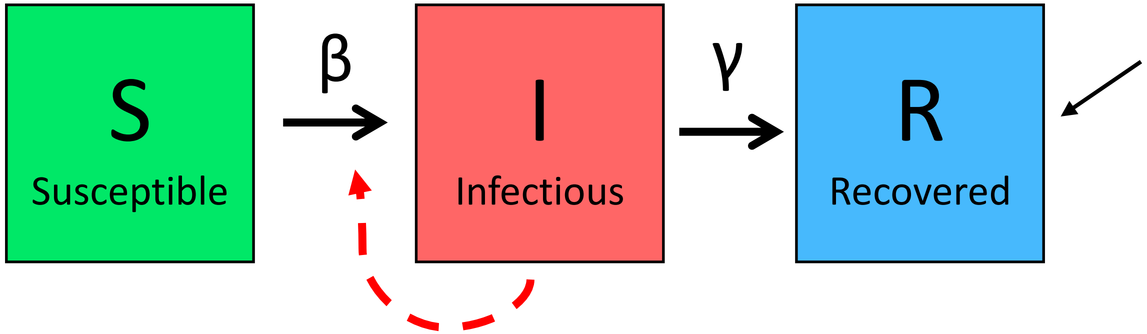


β = transmission rate
γ = recovery rate

Kermack and McKendrick 1927 *Proc Roy Soc A*

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

“**antibody** positive”
“**seropositive**”



β = transmission rate
γ = recovery rate

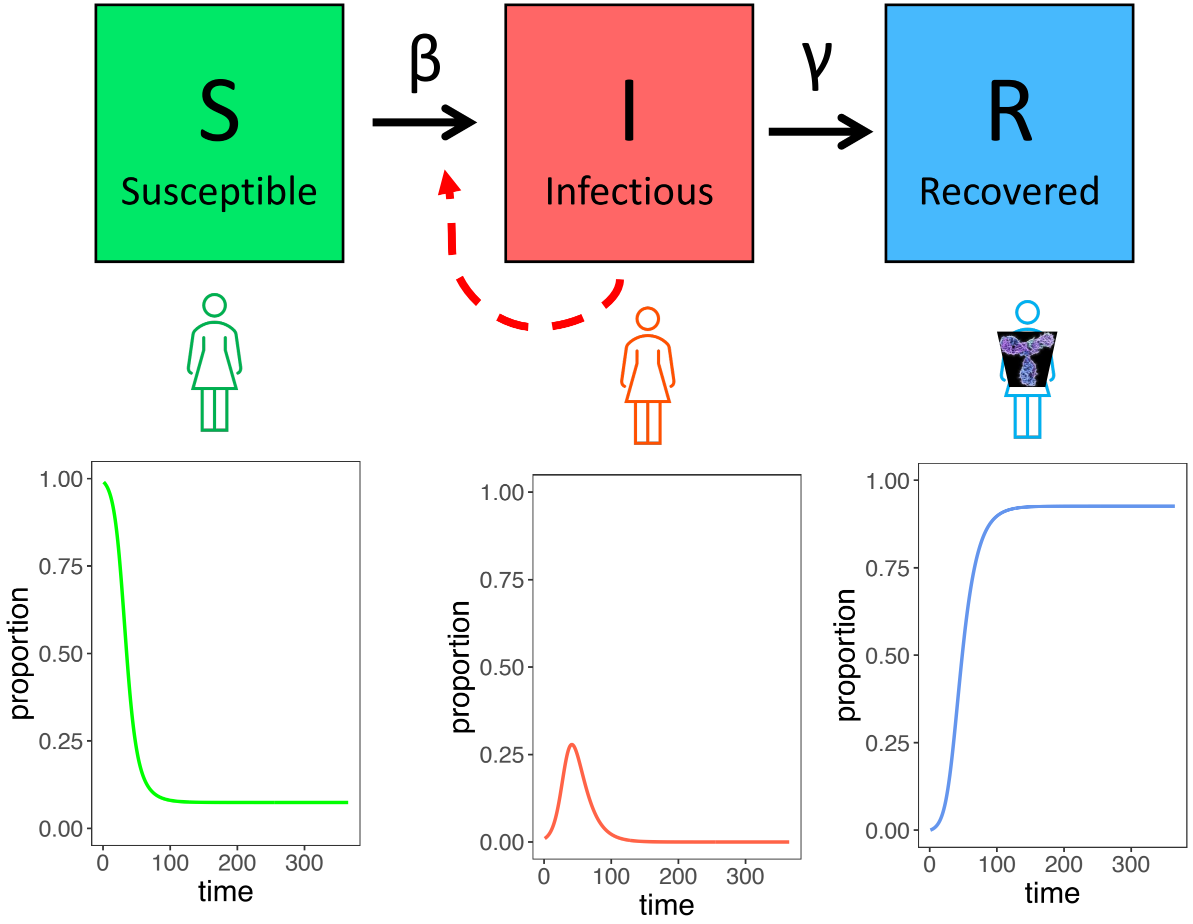
Kermack and McKendrick 1927 *Proc Roy Soc A*

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

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

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

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



β = transmission rate
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Kermack and McKendrick 1927 *Proc Roy Soc A*

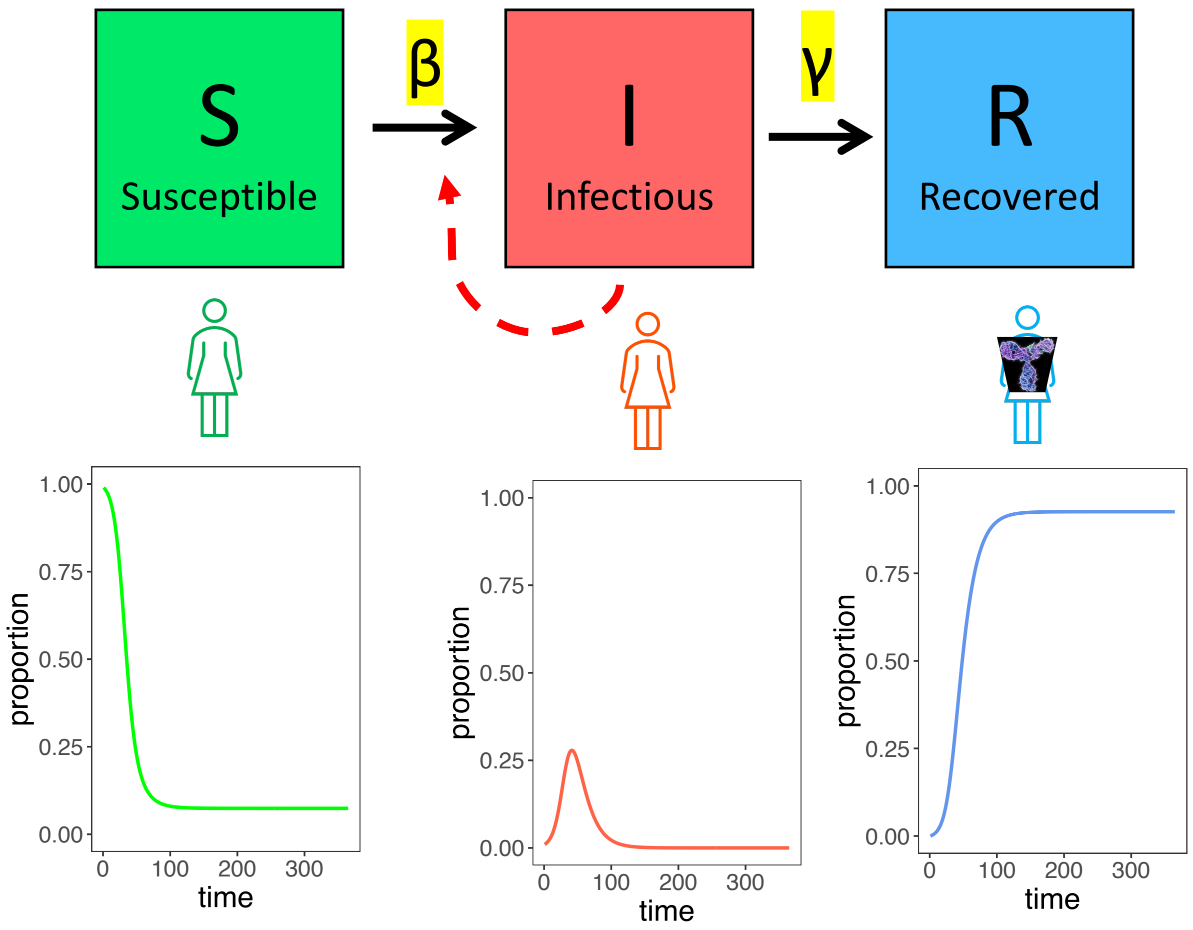
We use computers to simulate systems of equations in the SIR framework.

R₀ *is the pathogen* ***basic reproduction number.***

$$\frac{d\boldsymbol{S}}{dt} = -\beta \boldsymbol{S}\boldsymbol{I}$$

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

$$\frac{d\boldsymbol{R}}{dt} = \gamma \boldsymbol{I}$$



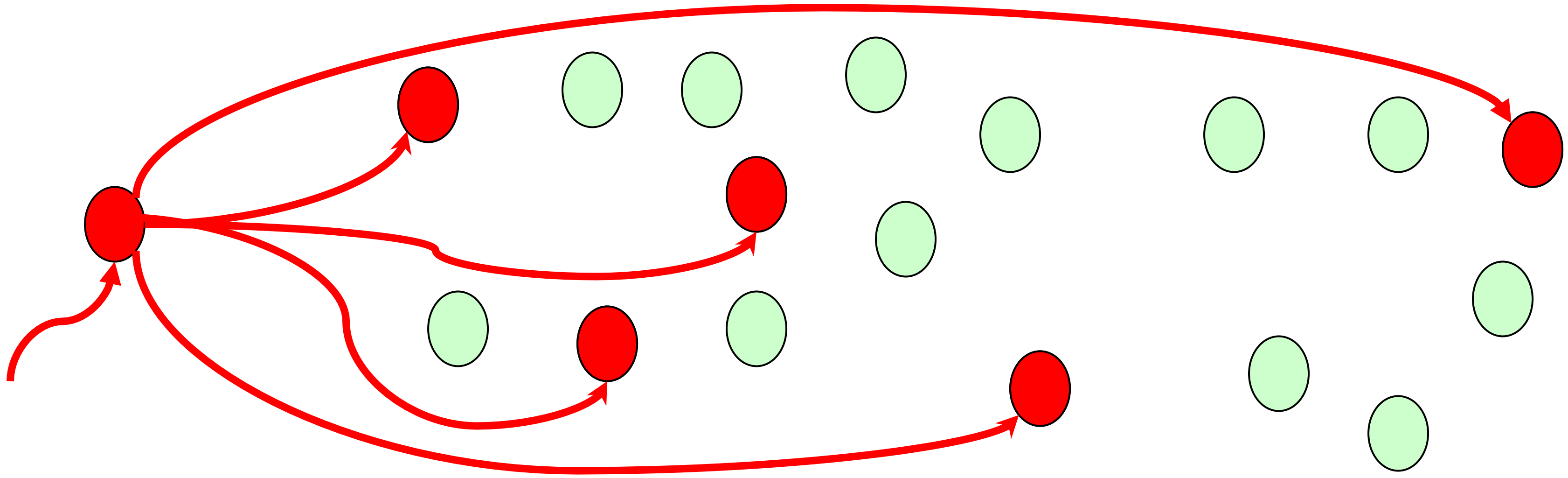
$$\mathbf{R_0} = \frac{\overbrace{\boldsymbol{\beta}}^{\text{infections created}}}{\underbrace{\boldsymbol{\gamma}}_{\text{infections lost}}}$$

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

β = transmission rate
γ = 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?

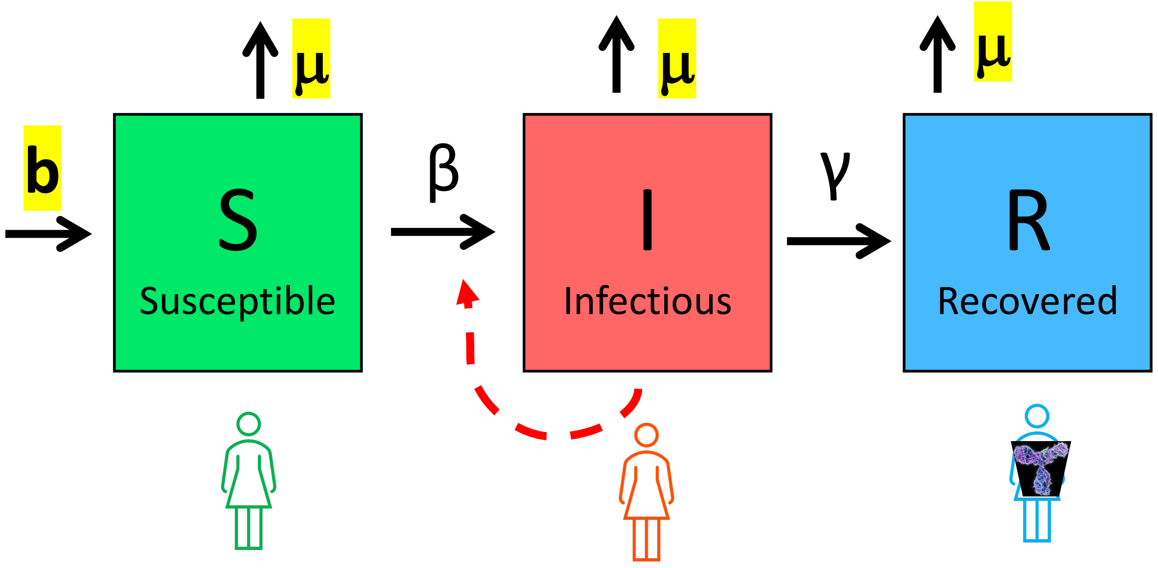
R_o = 5

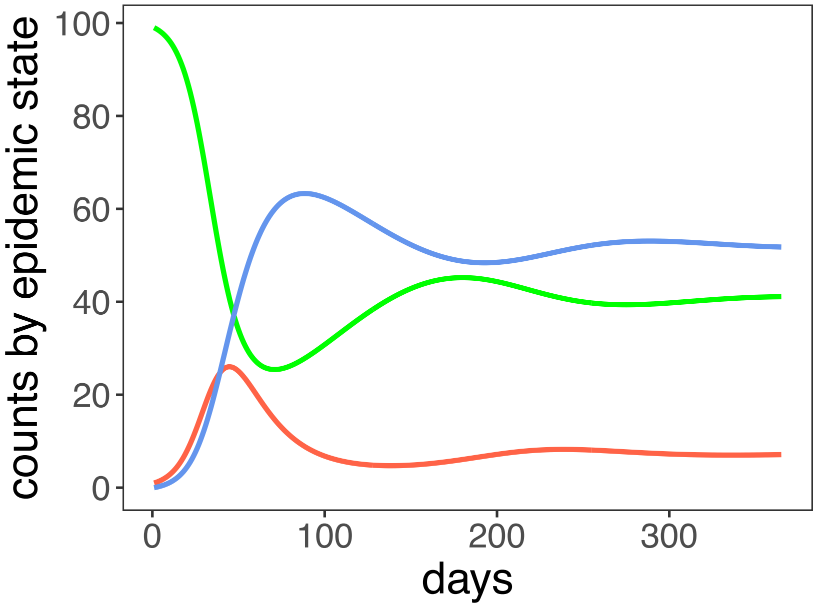
We can add realism to our models with births and deaths to **maintain** endemic pathogens.

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

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

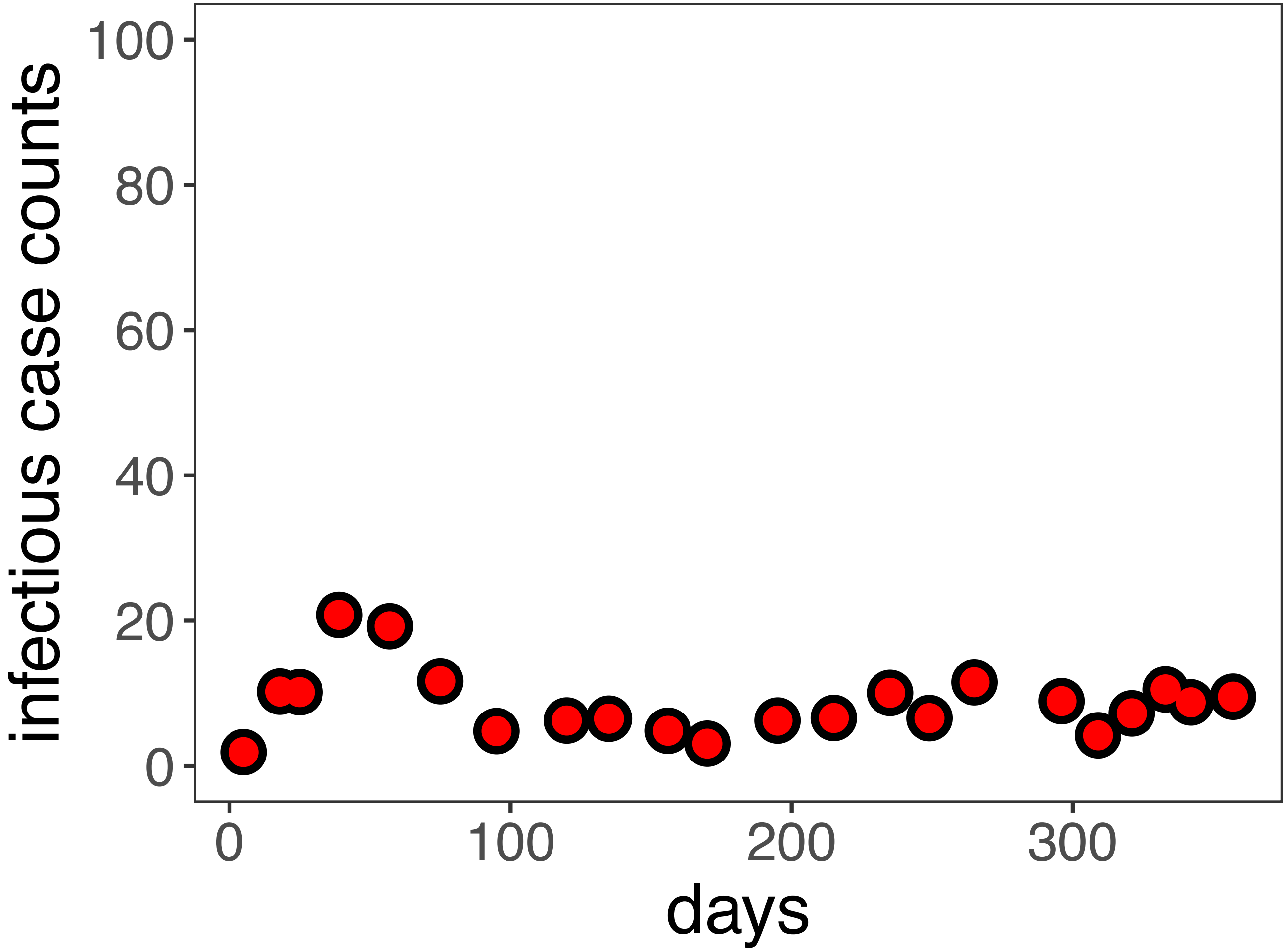
$$\frac{dR}{dt} = \gamma I - \mu R$$

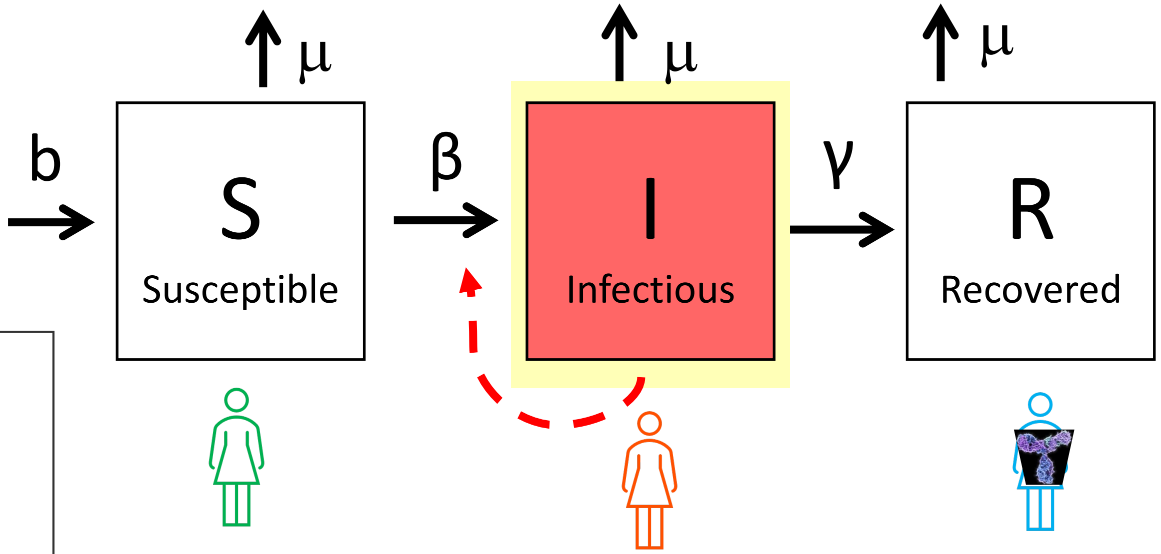




b = birth rate
μ = death rate
β = transmission rate
γ = recovery rate

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





Anderson & May 1991. *Infectious Diseases of Humans*

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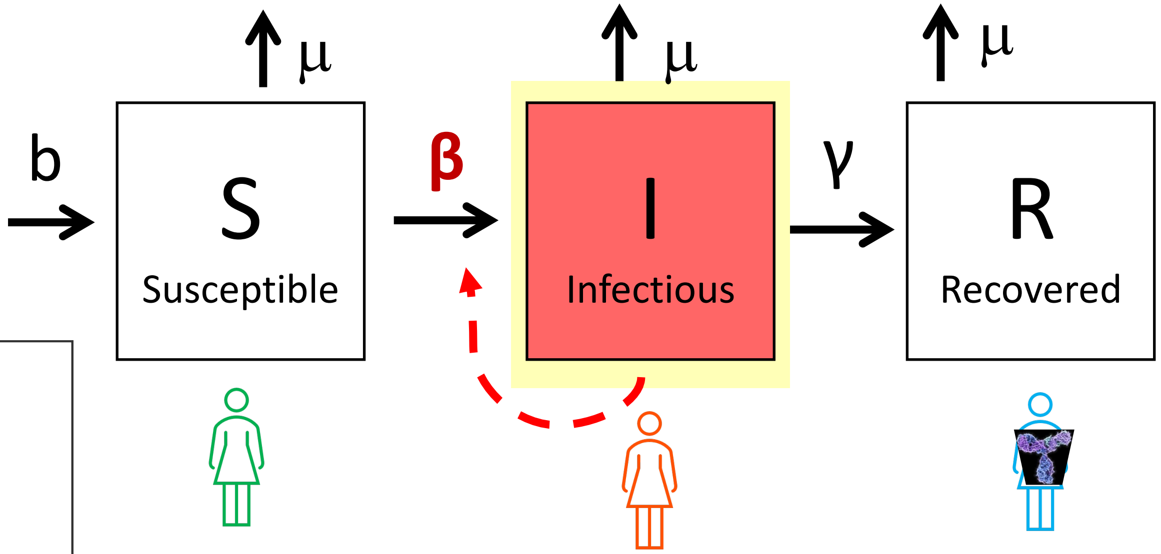
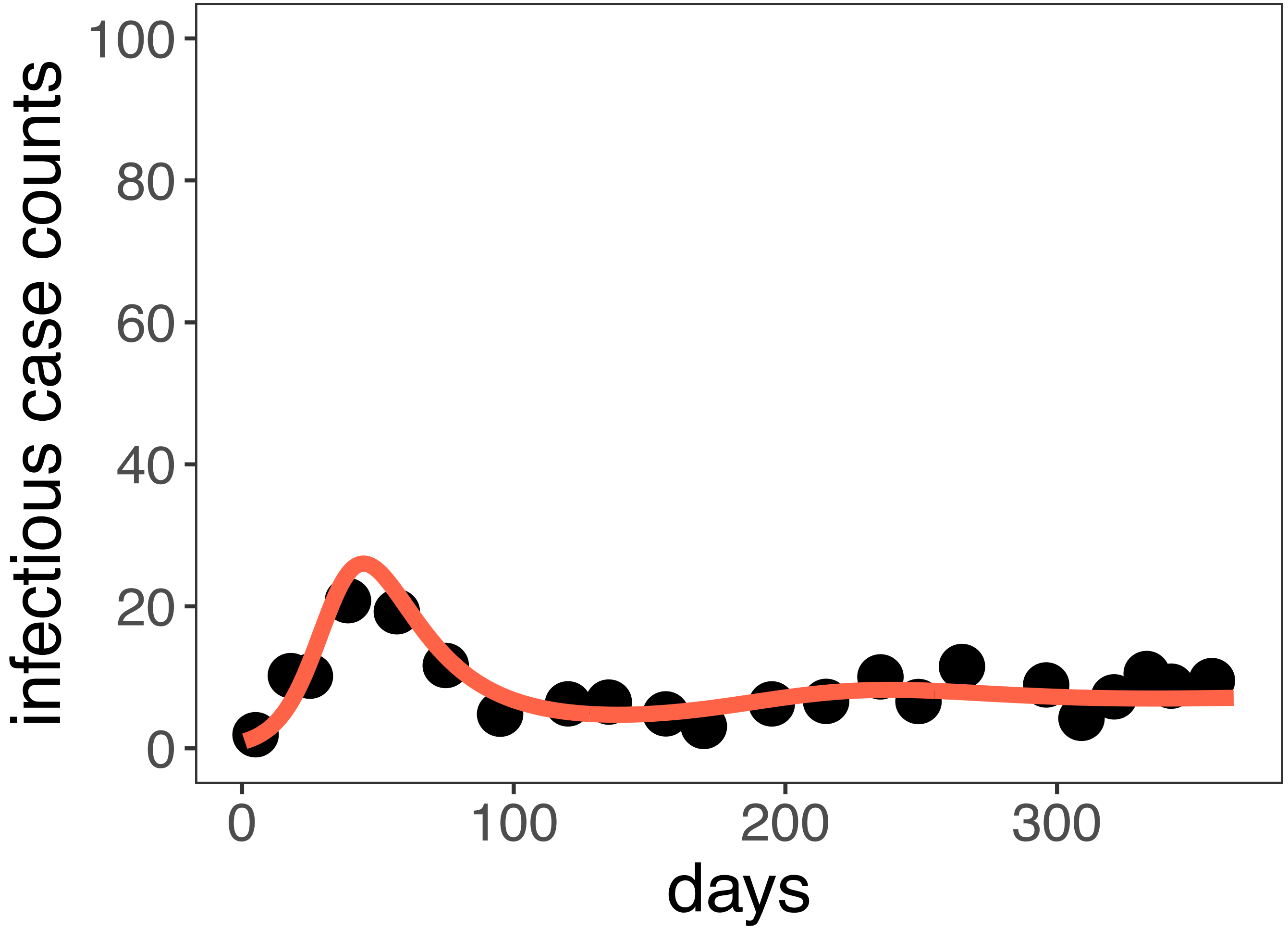


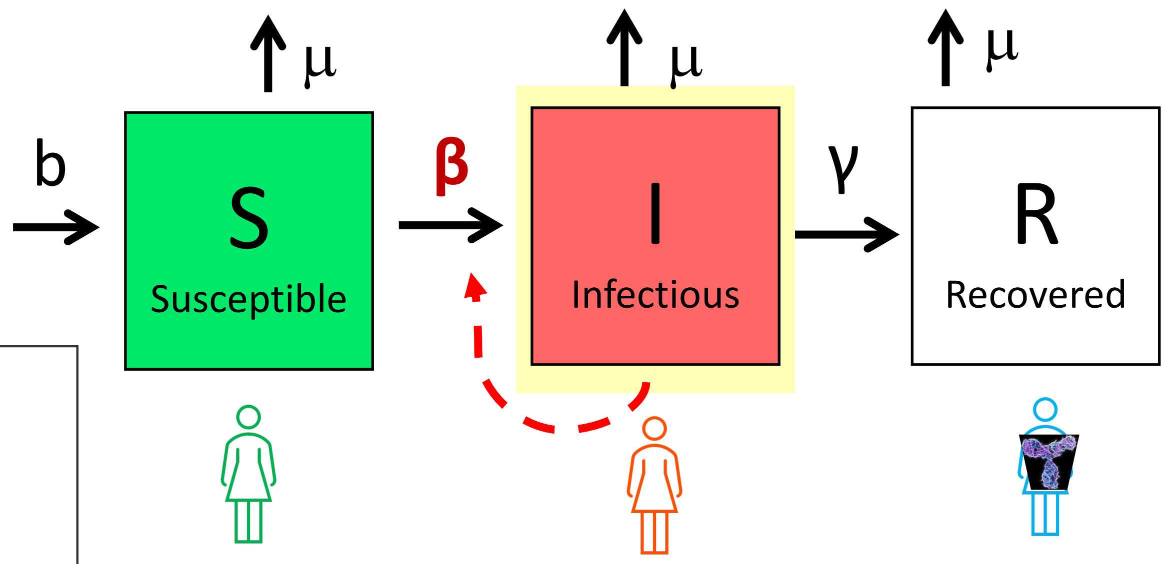


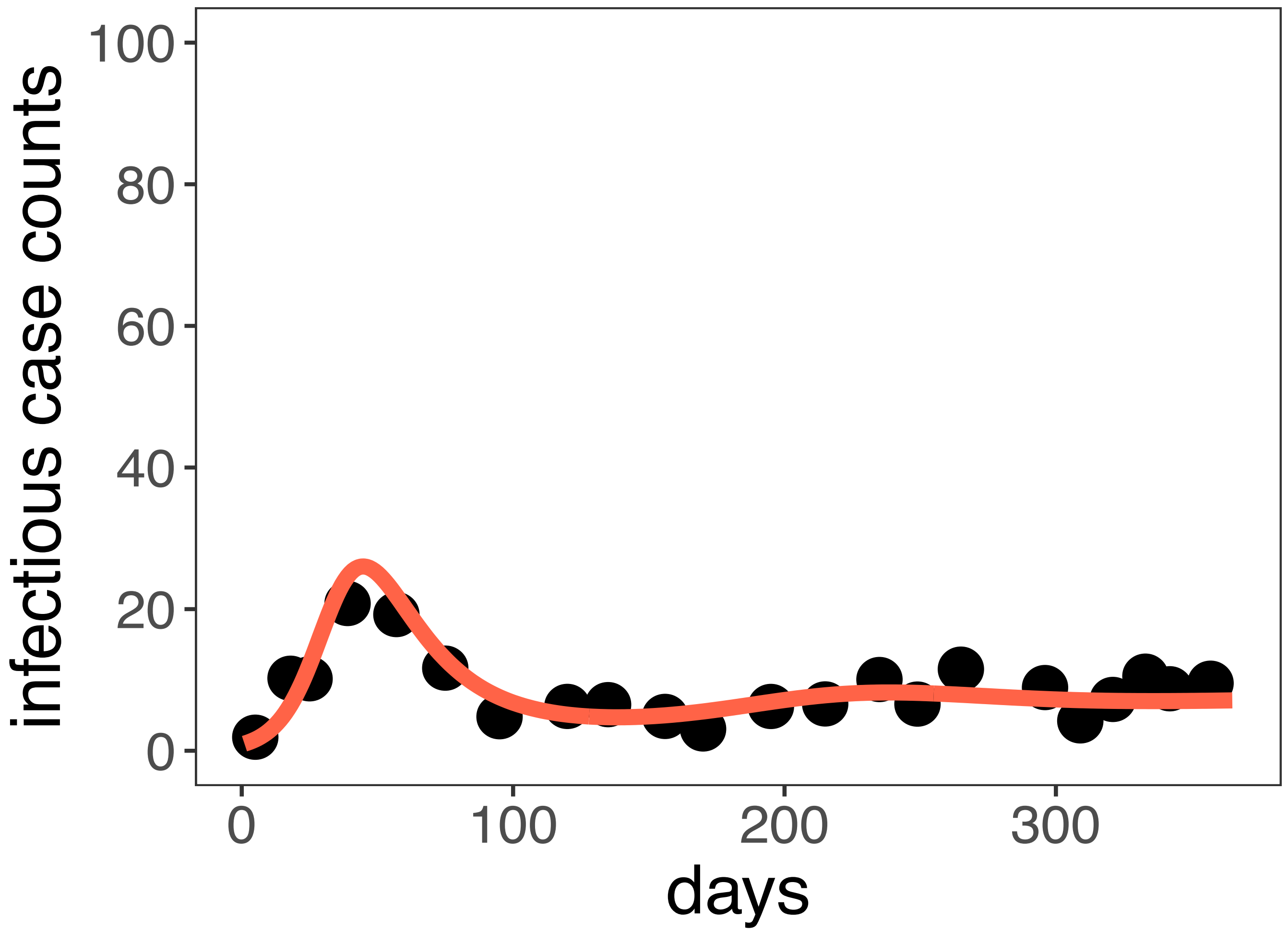
$$R_0 = \frac{\beta}{\gamma + \mu}$$

b = birth rate
μ = death rate
β = transmission rate
γ = recovery rate

Anderson & May 1991. *Infectious Diseases of Humans*

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





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

b = birth rate
μ = death rate
β = transmission rate
γ = recovery rate

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

$\approx \lambda$ *for a population model*

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

Anderson & May 1991. *Infectious Diseases of Humans*

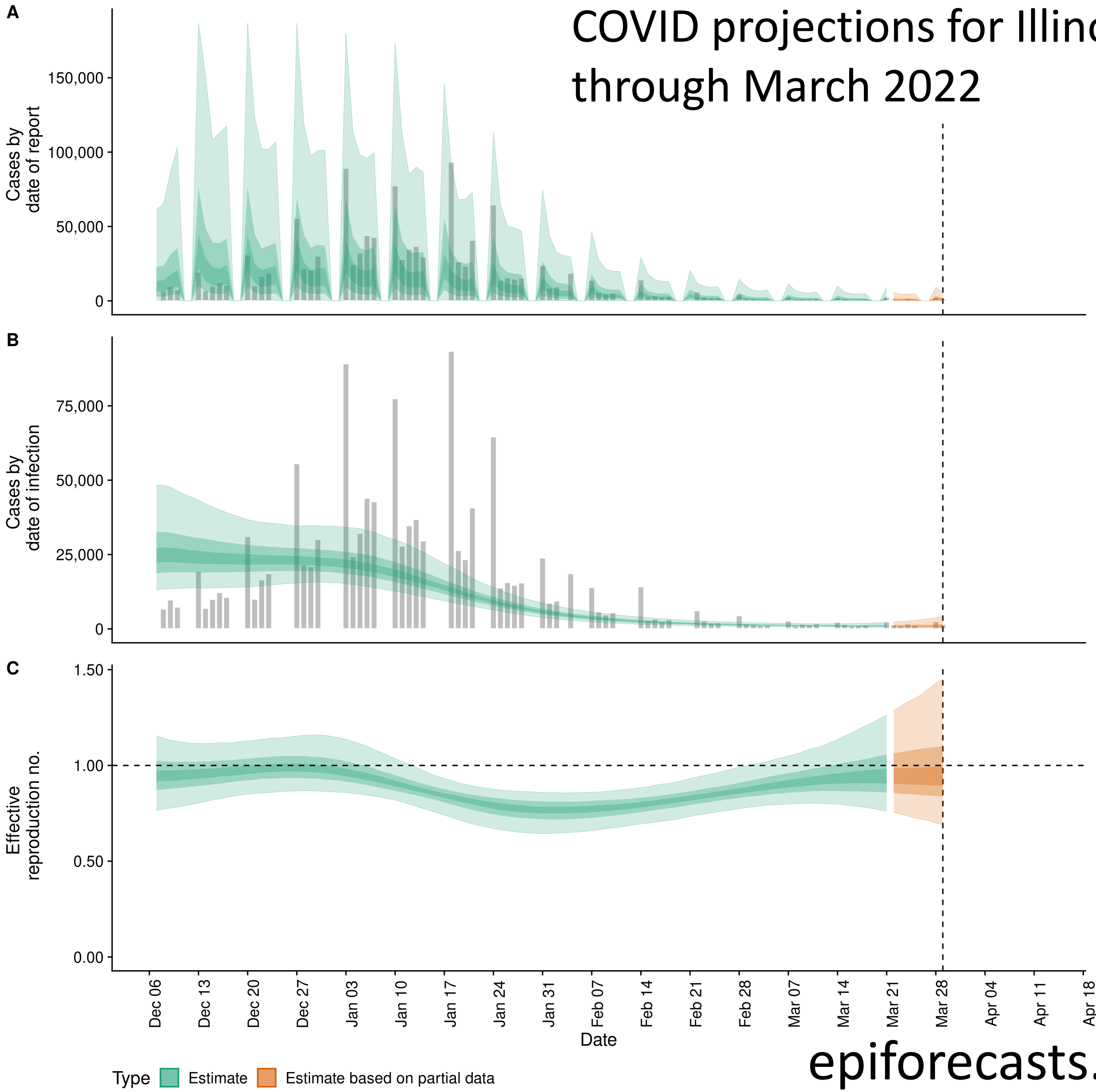
R_E OR R_t

- 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 R0*proportion susceptible

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

- Gives a realistic pulse of the current pace of the epidemic!

COVID projections for Illinois through March 2022



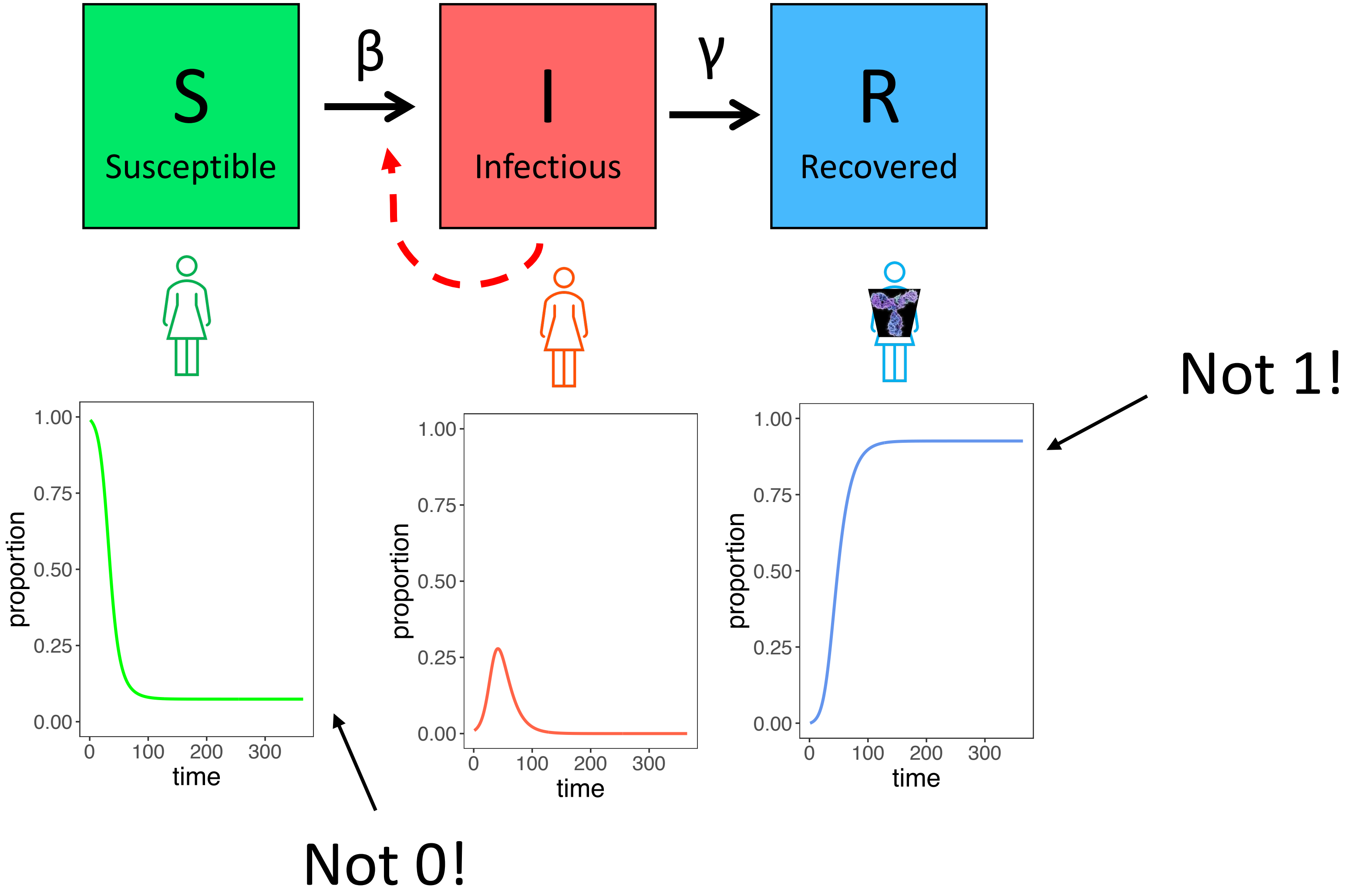
epiforecasts.io

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

$$\frac{d\mathbf{S}}{dt} = -\beta \mathbf{S}\mathbf{I}$$

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

$$\frac{d\mathbf{R}}{dt} = \gamma \mathbf{R}$$



β = transmission rate
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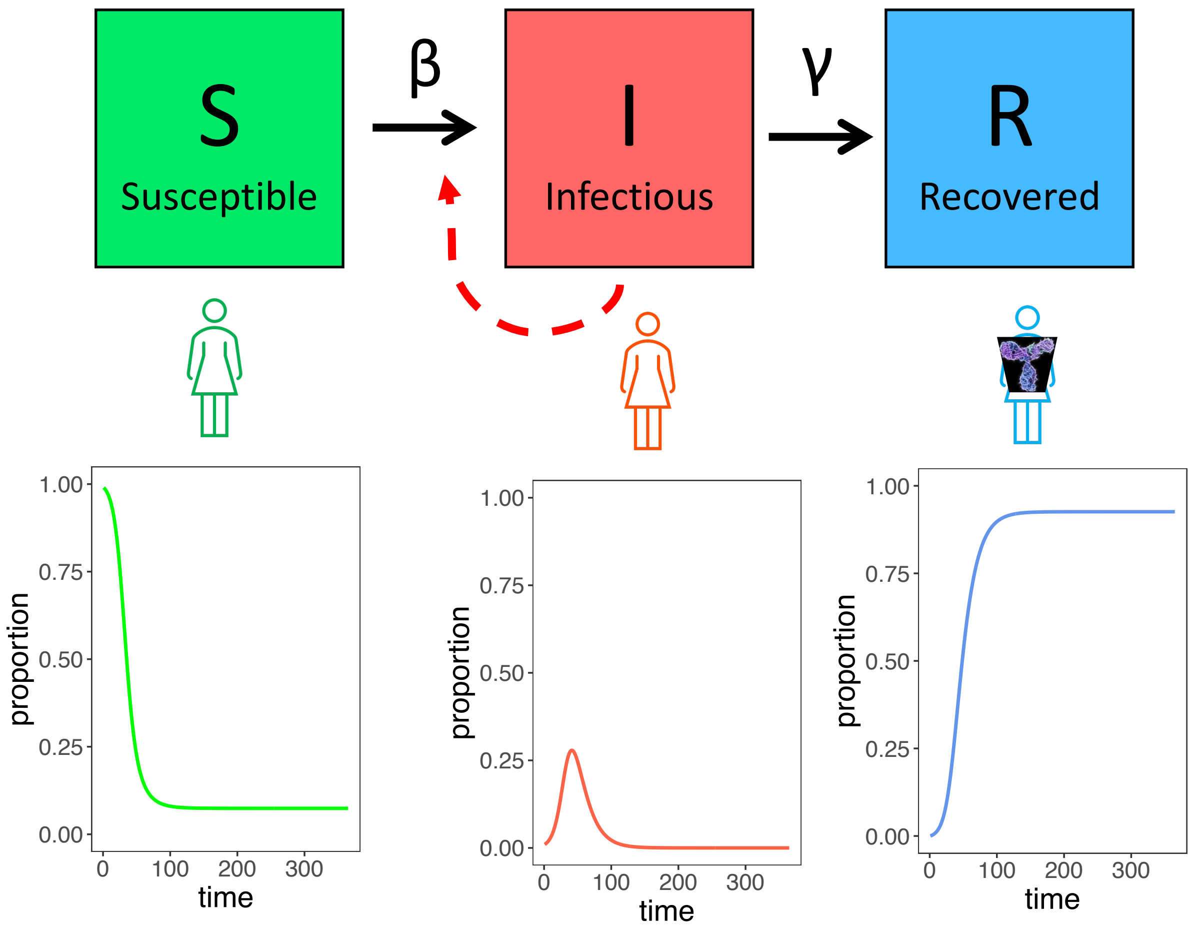


- The epidemic does not end because all individuals have been infected and have either died or recovered.
- Rather, finding new susceptibles becomes more difficult and $R_E < 1$

β = transmission rate
γ = recovery rate

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

$\boldsymbol{R_0}$ interventions

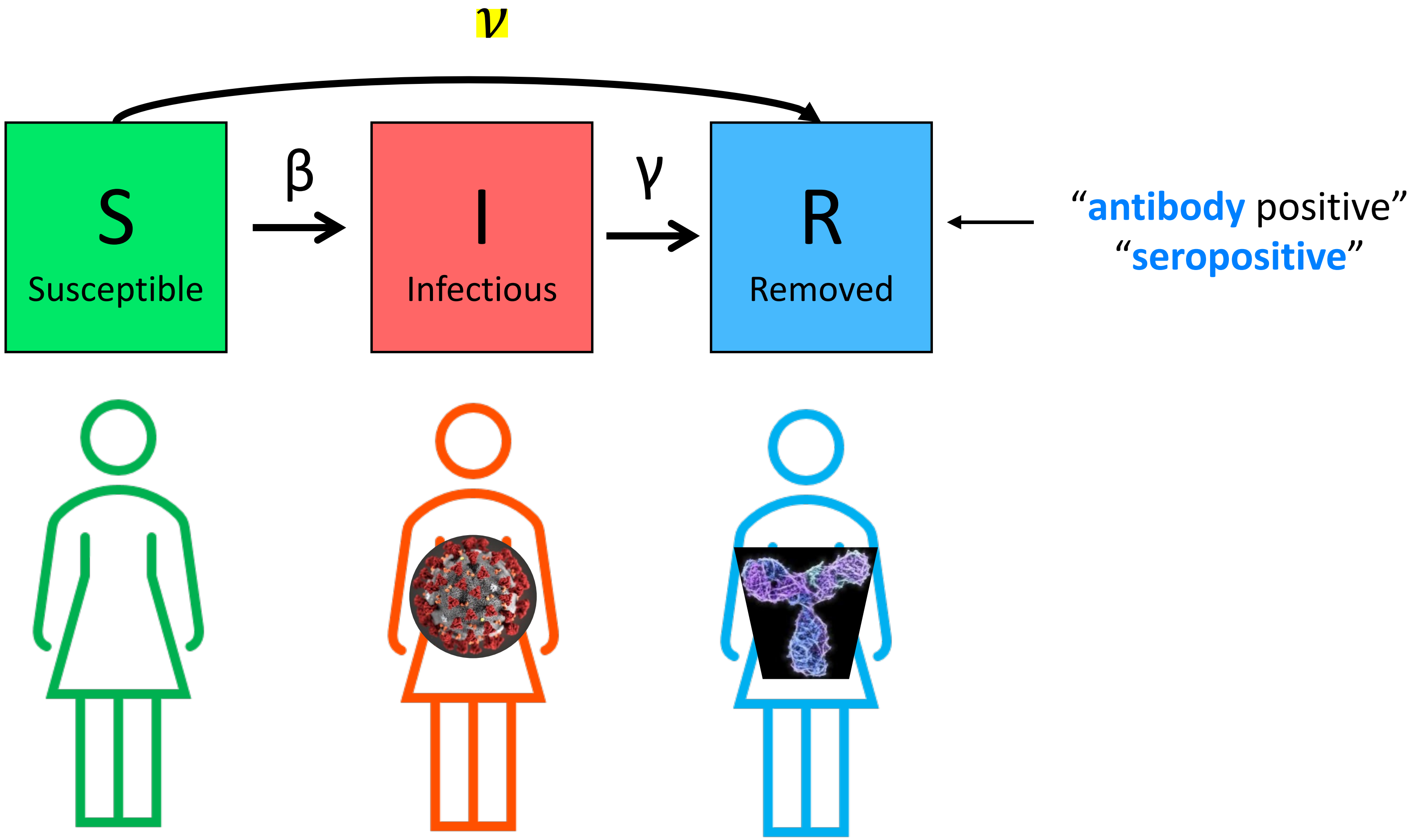
- Social distancing
- Masking
- Limits to gathering sizes
- Drugs that shorten the infectious period

$\boldsymbol{R_E}$ interventions

- Vaccination

Mathematics of Vaccination

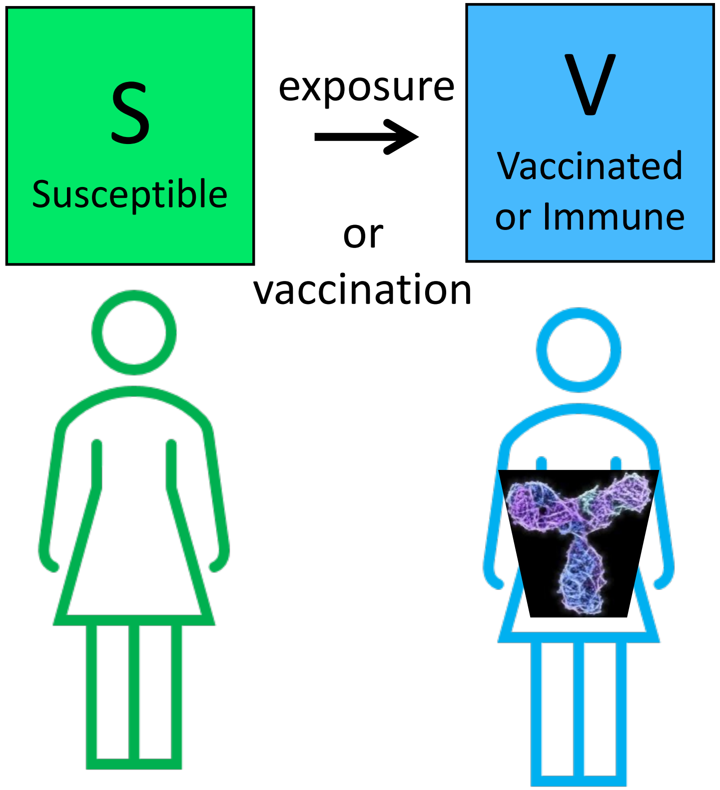
- Goal: **Reduce $R_E < 1$** by removing individuals from the susceptible population.



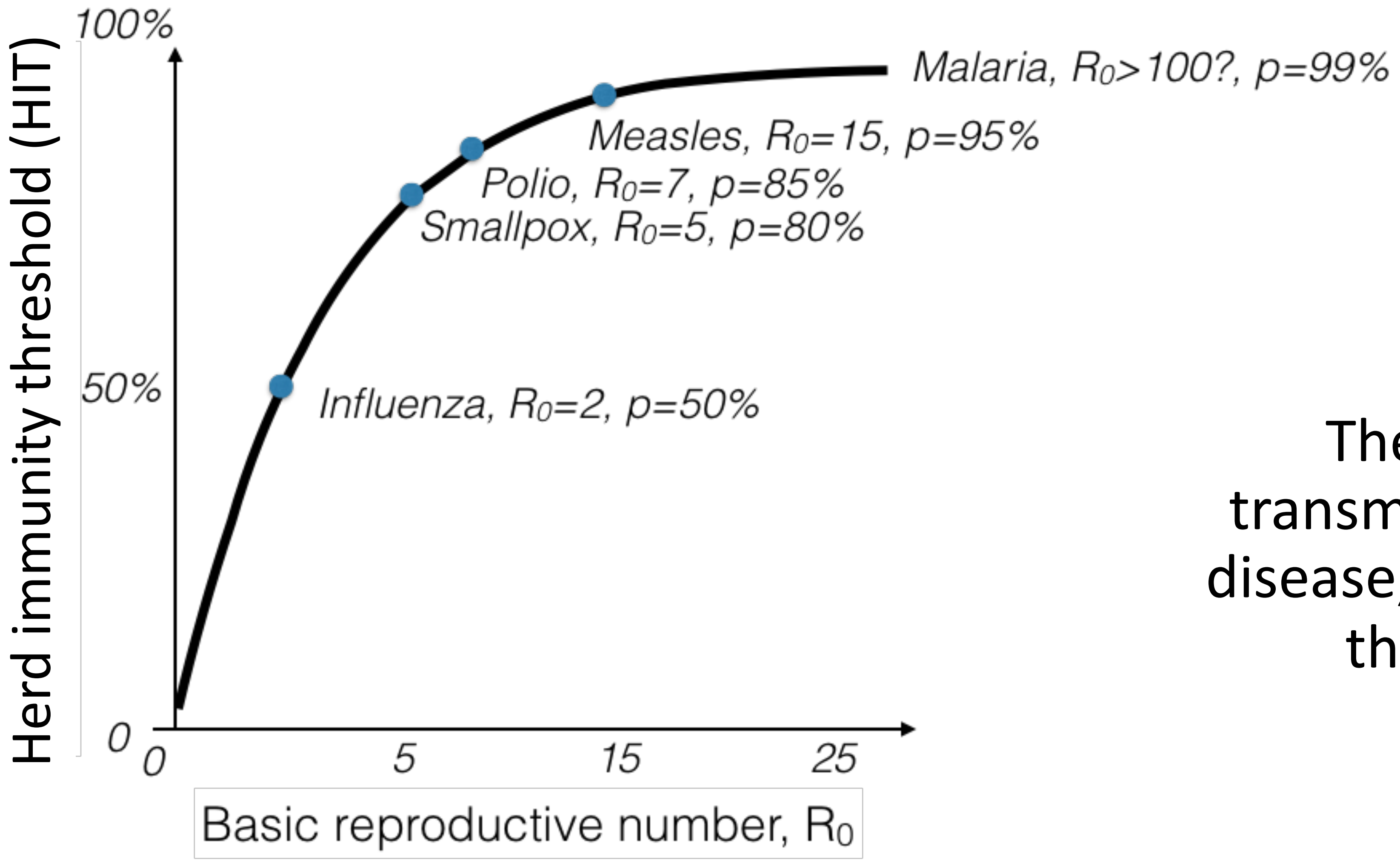
β = transmission rate
γ = recovery rate
ν = vaccination rate

Mathematics of Vaccination

- Goal: **Reduce $\boldsymbol{R_E < 1}$** 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, $\boldsymbol{P_V > 1 - \frac{1}{R_0}}$
- **This is the herd immunity threshold.**
- **Even susceptibles will not become infected because the disease will not spread ($\boldsymbol{R_E < 1}$).**

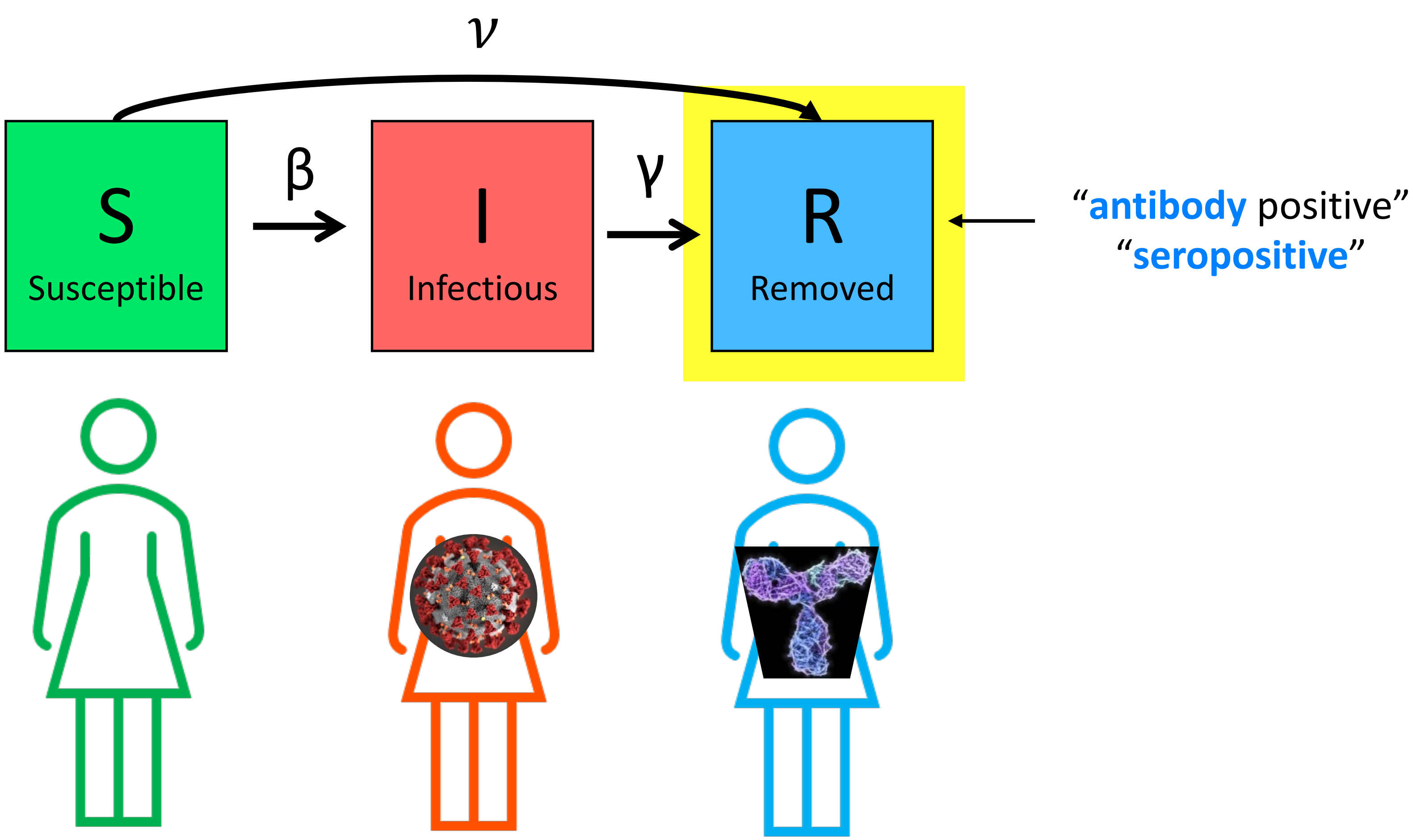


R_0 and the Herd Immunity Threshold



The more transmissible the disease, the higher the HIT!

Vaccination stems from a long history

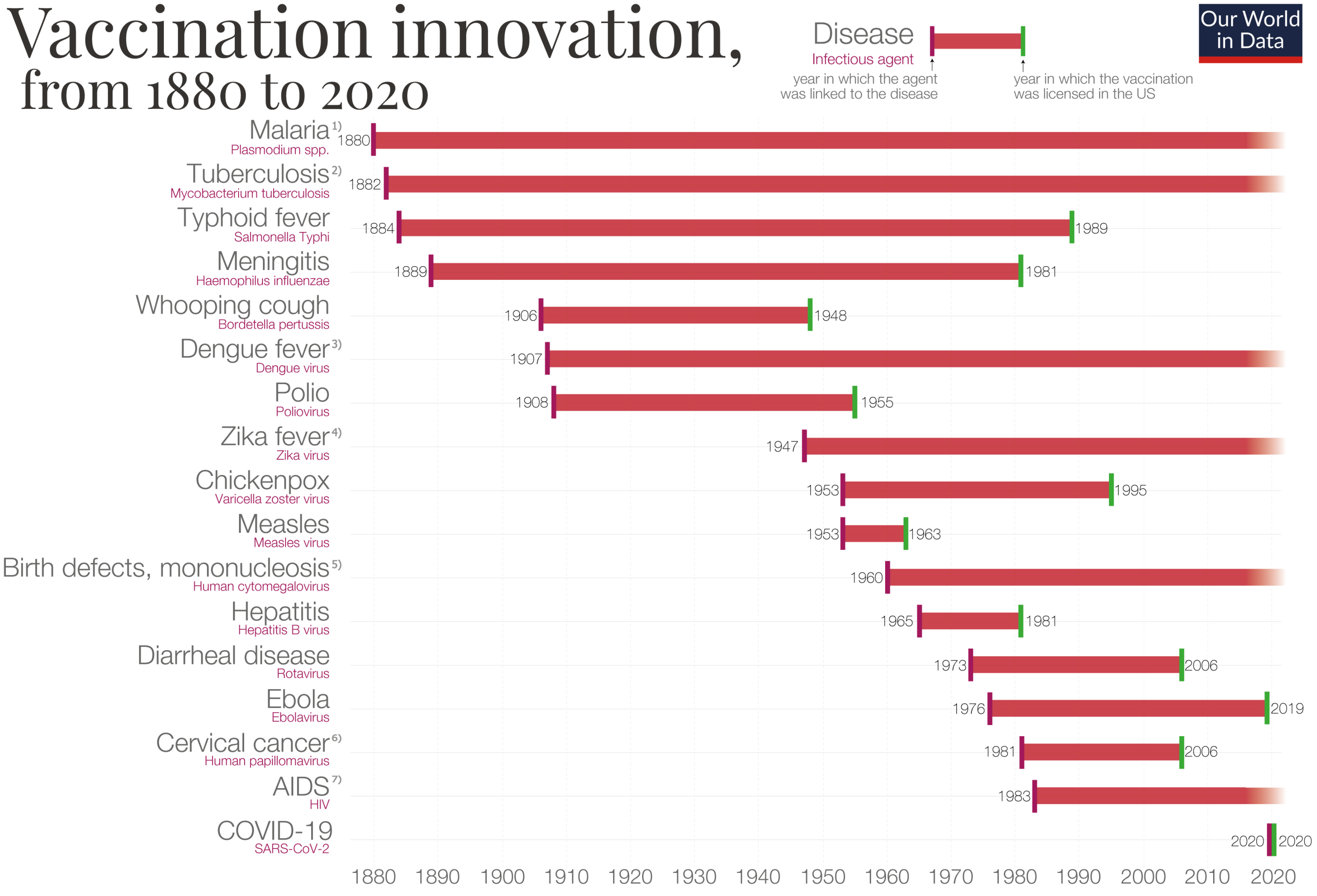


Vaccination stems from a long history

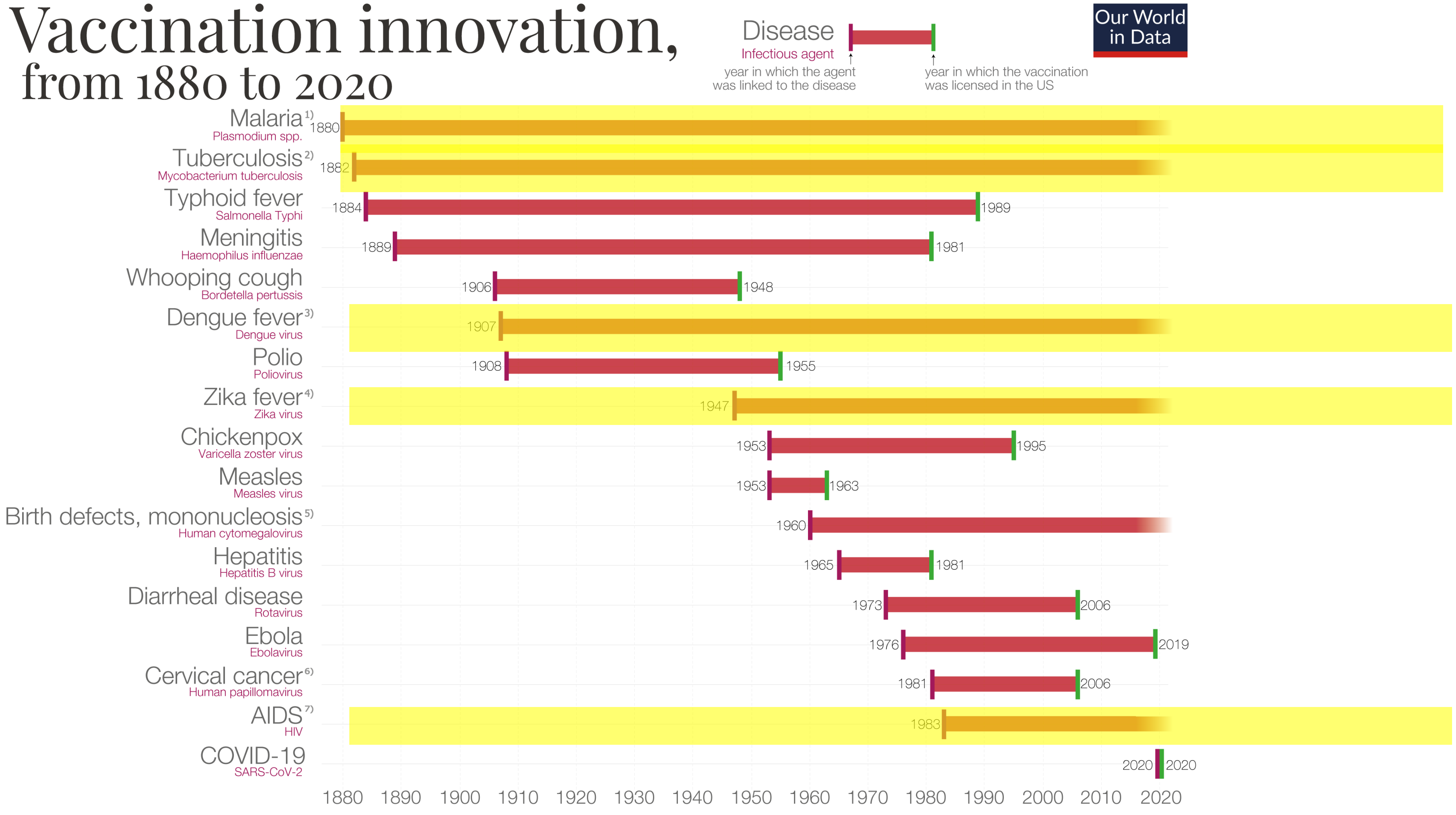
- Variolation: Early attempts to provide protection against smallpox (*Variola virus*) via inoculation with scab material from a recent patient infected with Variola minor
  - First described in China in the 10^{th} century
  - Caused 1% mortality!
- 1789 Edward Jenner used cowpox vesicles to inoculate 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



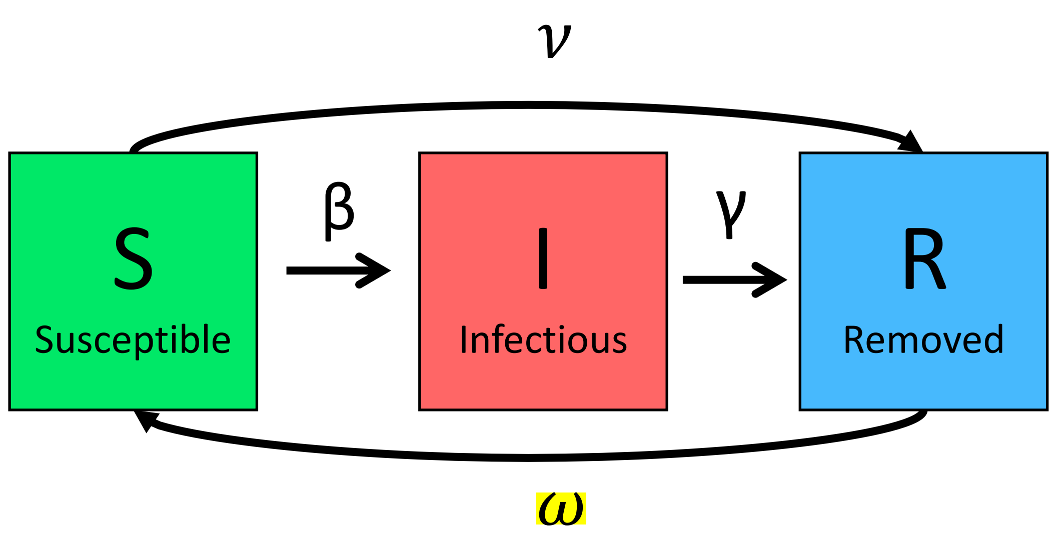
But we still lack vaccines for several important diseases.

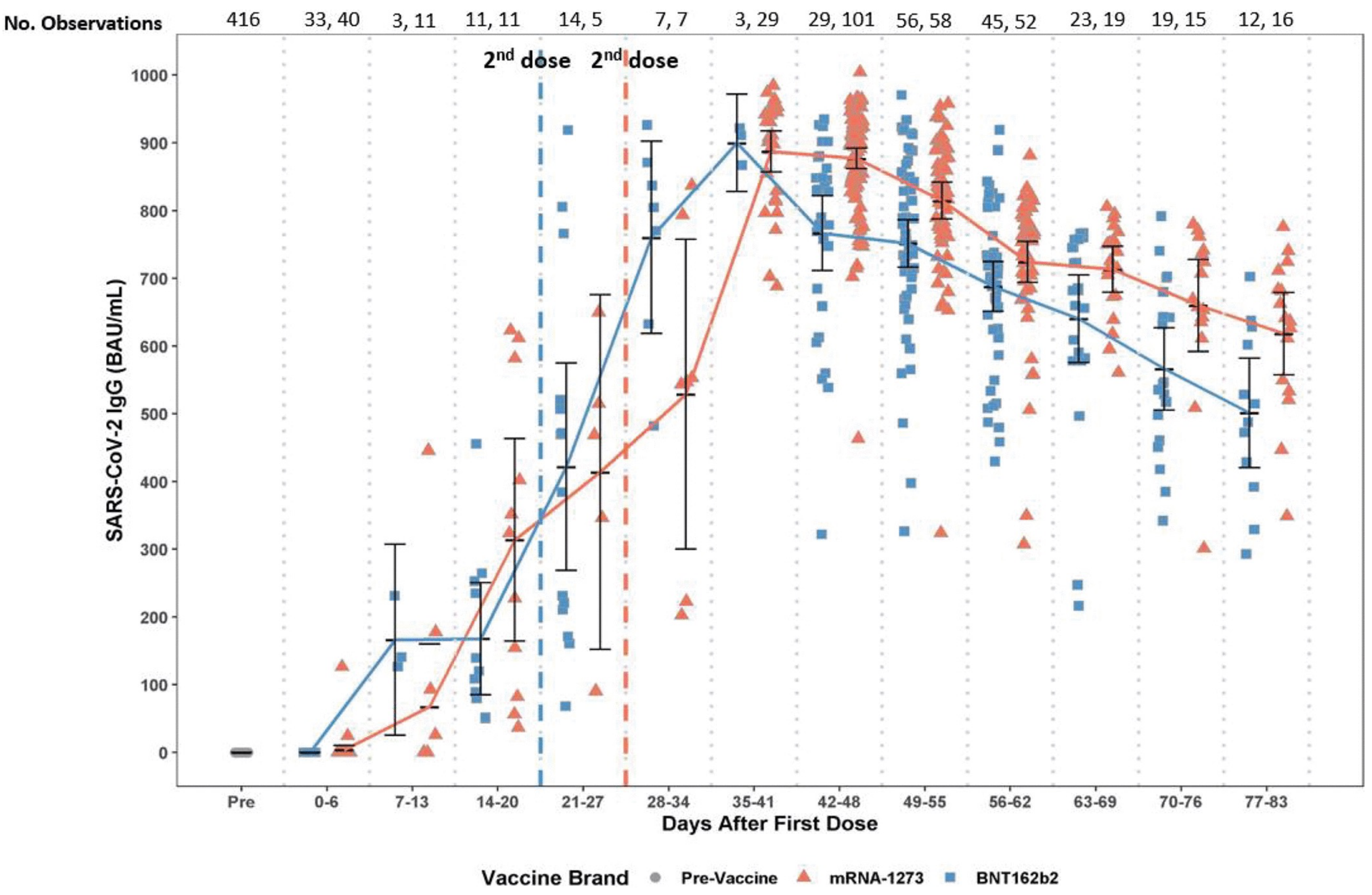


OurWorldinData.org – Research and data to make progress against the world's largest problems.

Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens



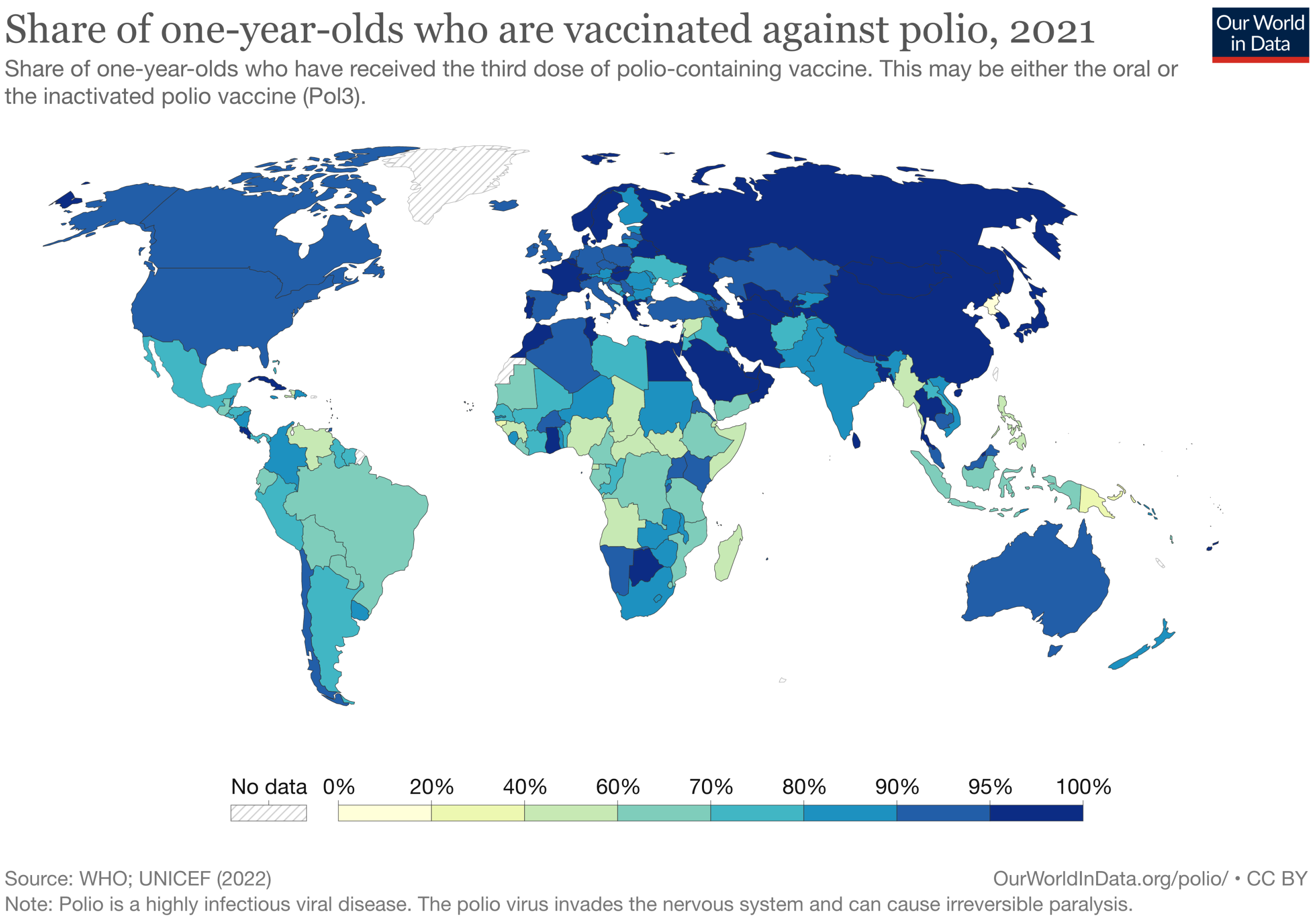


β = transmission rate
γ = recovery rate
ν = vaccination rate
ω = rate waning immunity

Montoya et al. 2021 *Microbiology Spectrum*

Challenges to Vaccination

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

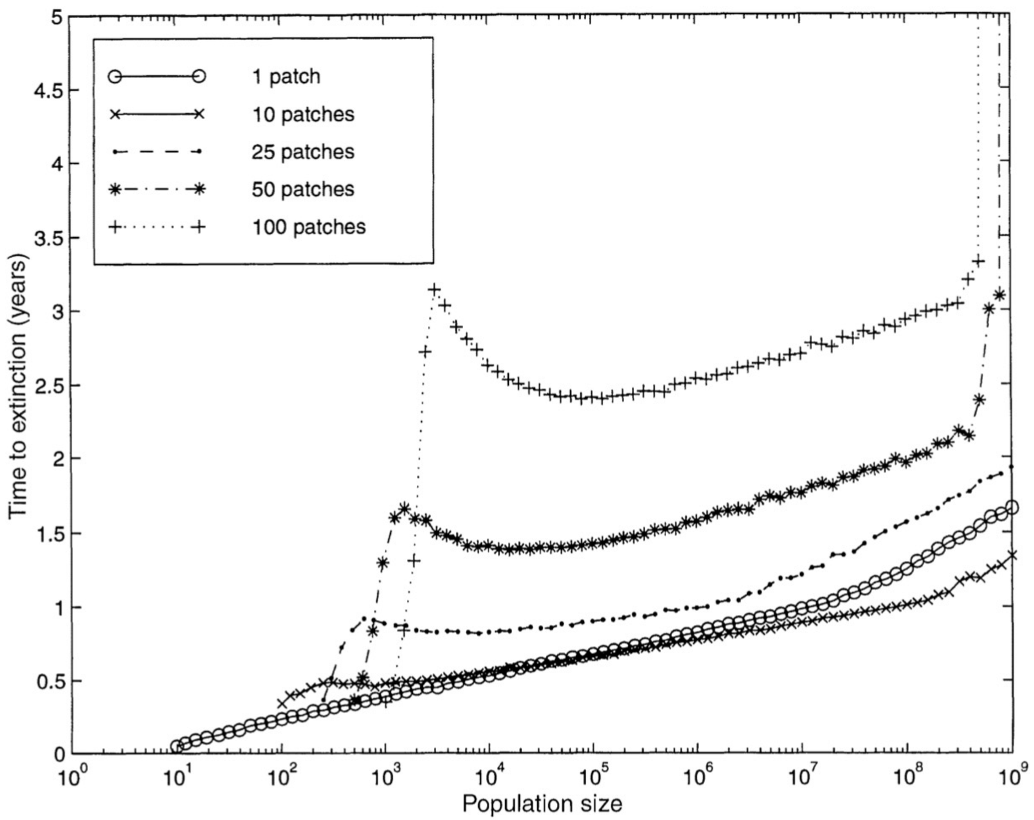


Challenges to Vaccination

- 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)

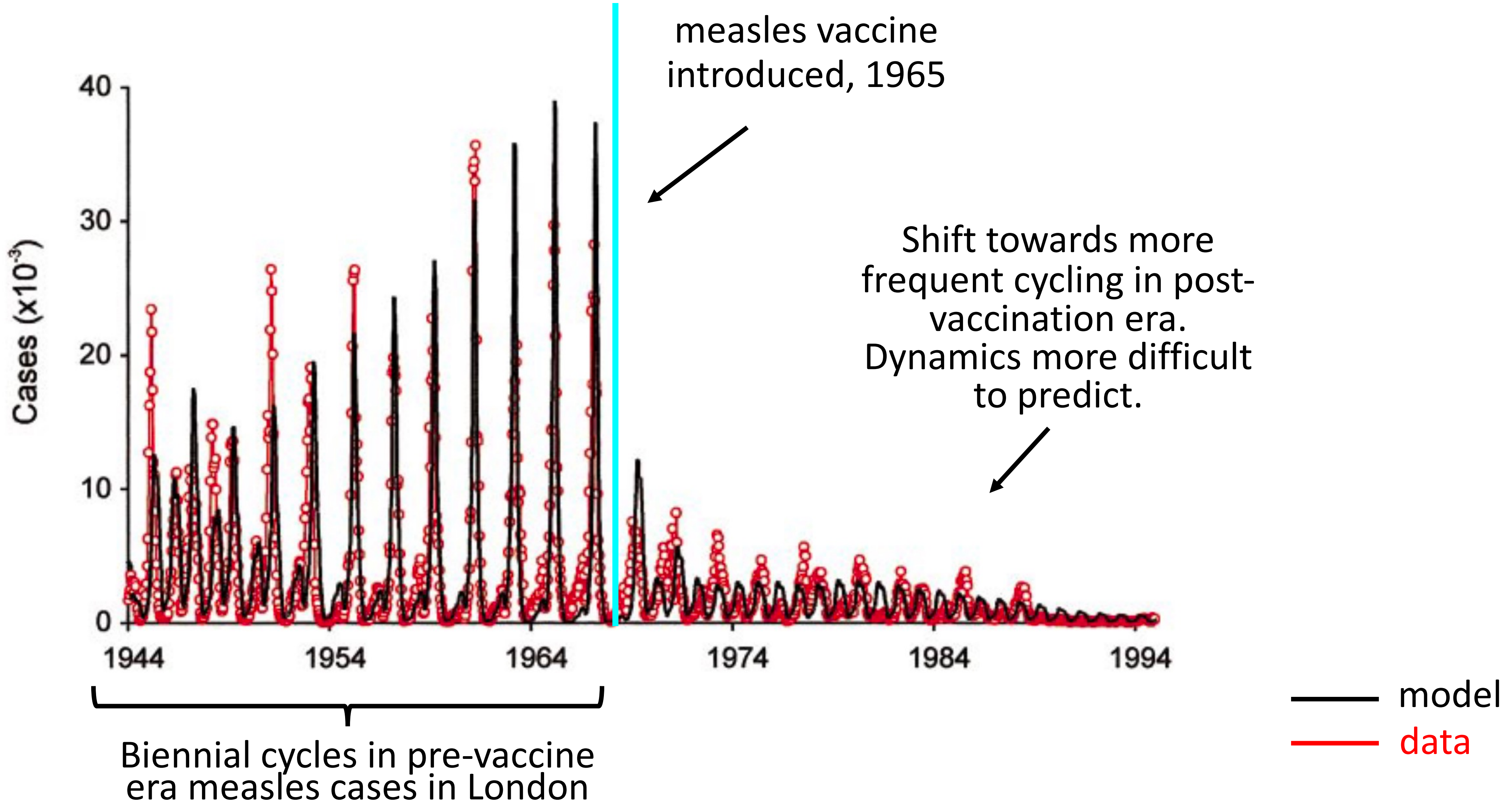


Swinton et al. 1998
Bartlett 1957

Challenges to Vaccination

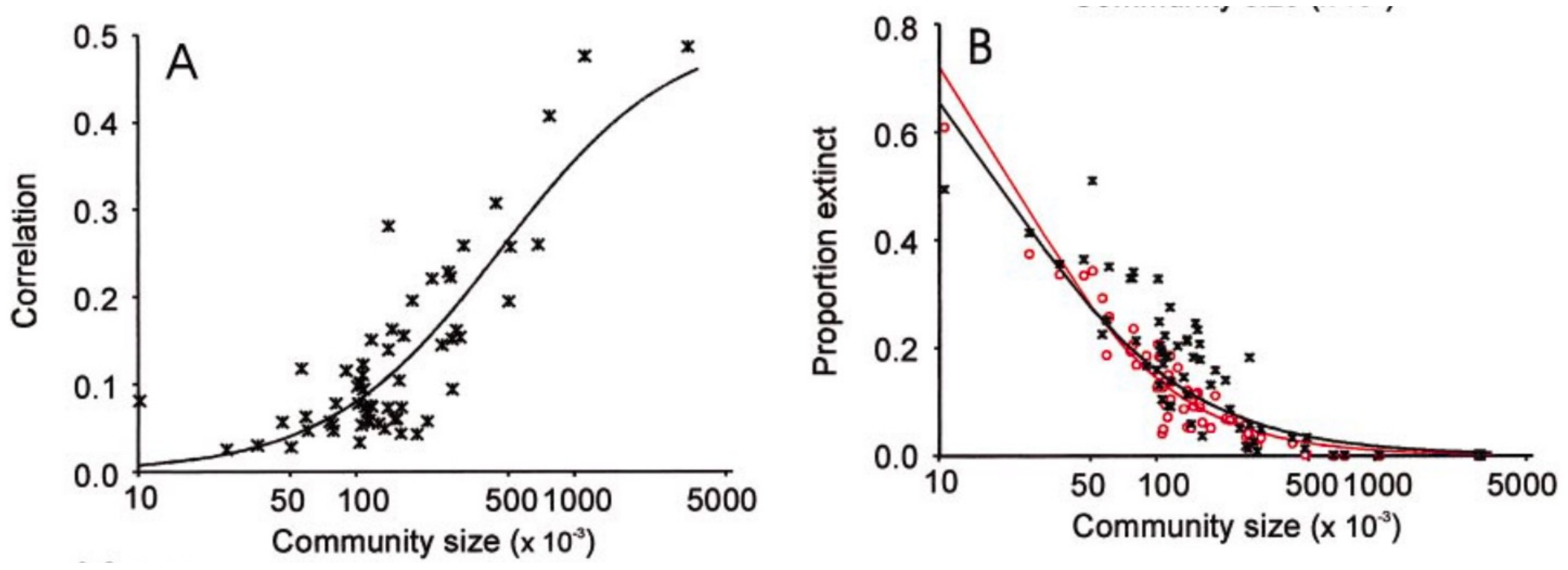
- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access
- Continuous births
- Animal reservoirs
- Spatial structure (metapopulation rescue)
- More complex pathogens!

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



Grenfell et al. 2002. *Ecological Monographs*

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



Grenfell et al. 2002. *Ecological Monographs*