

Fundamentals of Ecology

Week 8, Ecology Lecture 6

Cara Brook

February 25, 2025

Office hours: On ZOOM

Friday, Feb 28, 2025

4-5pm

I will email out a link!

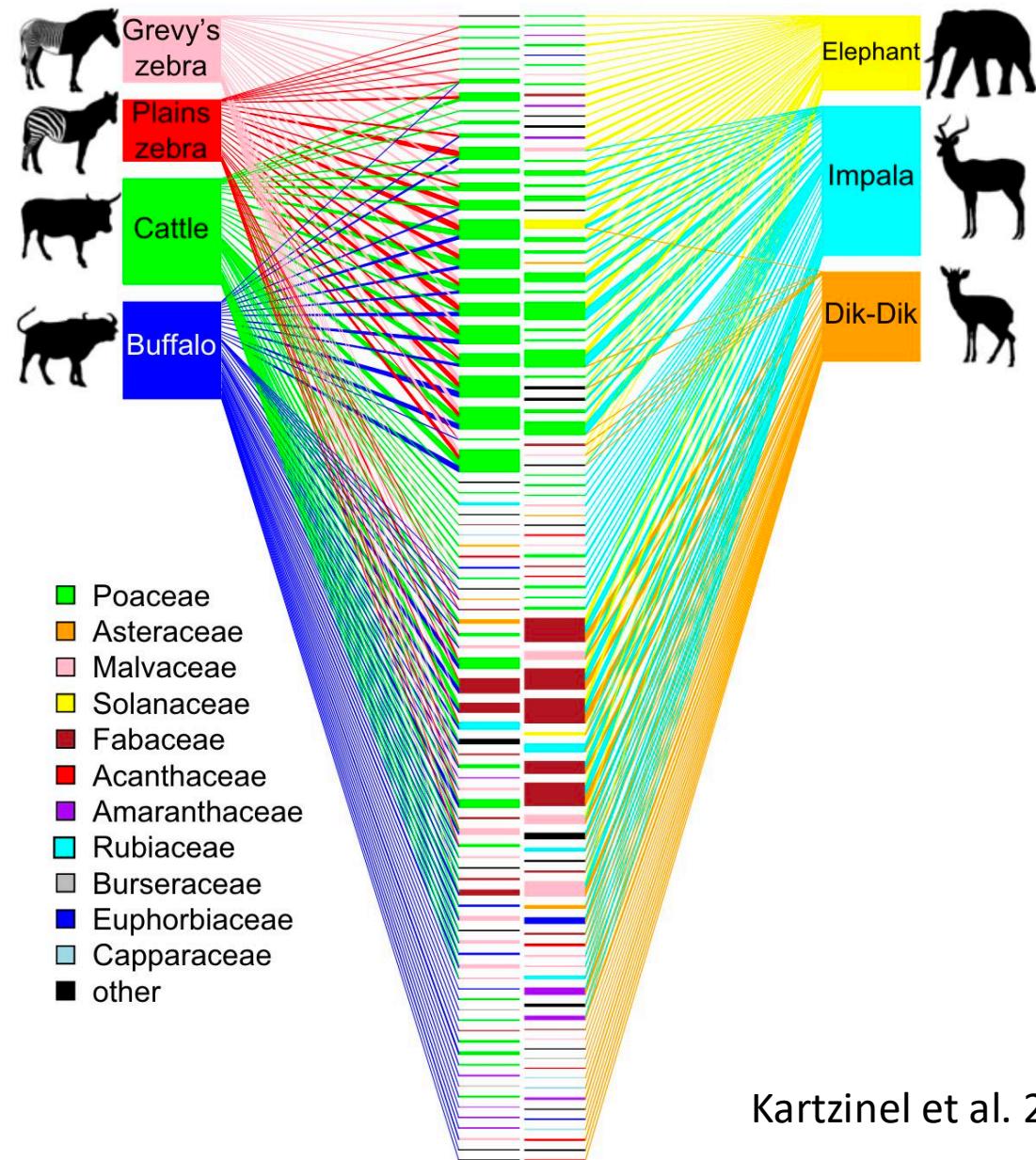
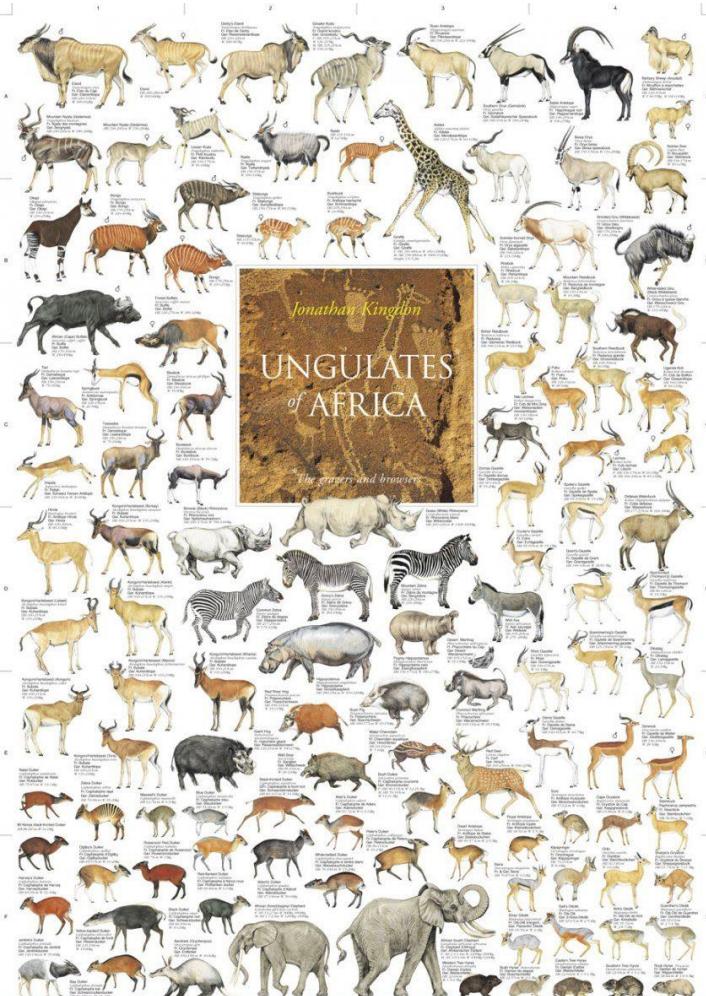
Learning objectives from Lecture 5

You should be able to:

- Recognize which components of the Lotka-Volterra competition equation correspond to intraspecies interactions vs. interspecies interactions.
- Analyze nullclines from the competition equations and predict, from the shape of the graph, which equilibrium the two populations will eventually move to, given a starting point.
- Given a set of competition equations and a graph of nullclines with variables labeled, understand which nullcline corresponds to which species.
- Understand niche partitioning, character displacement, and why these processes occur

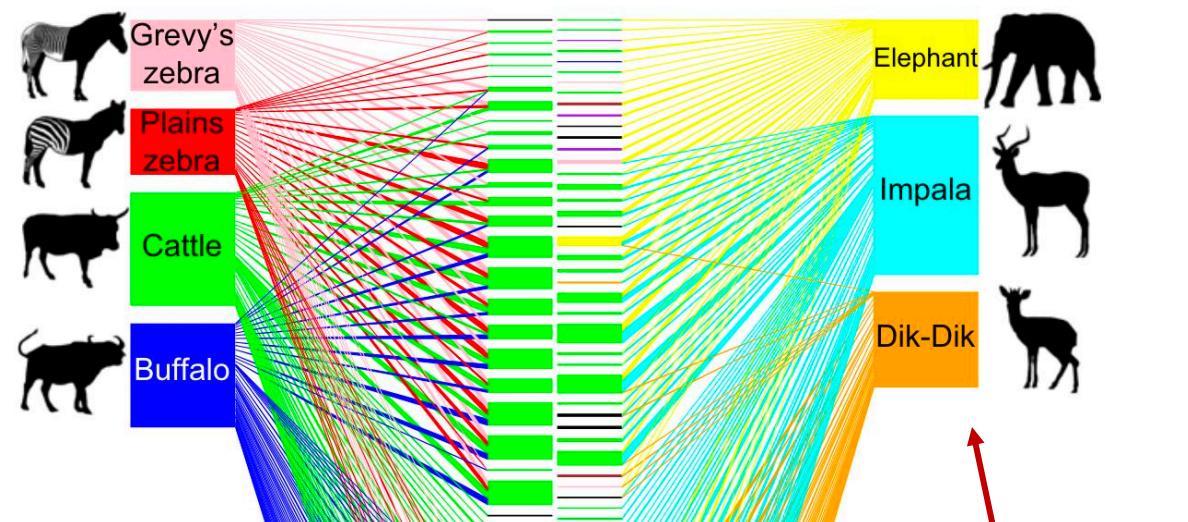
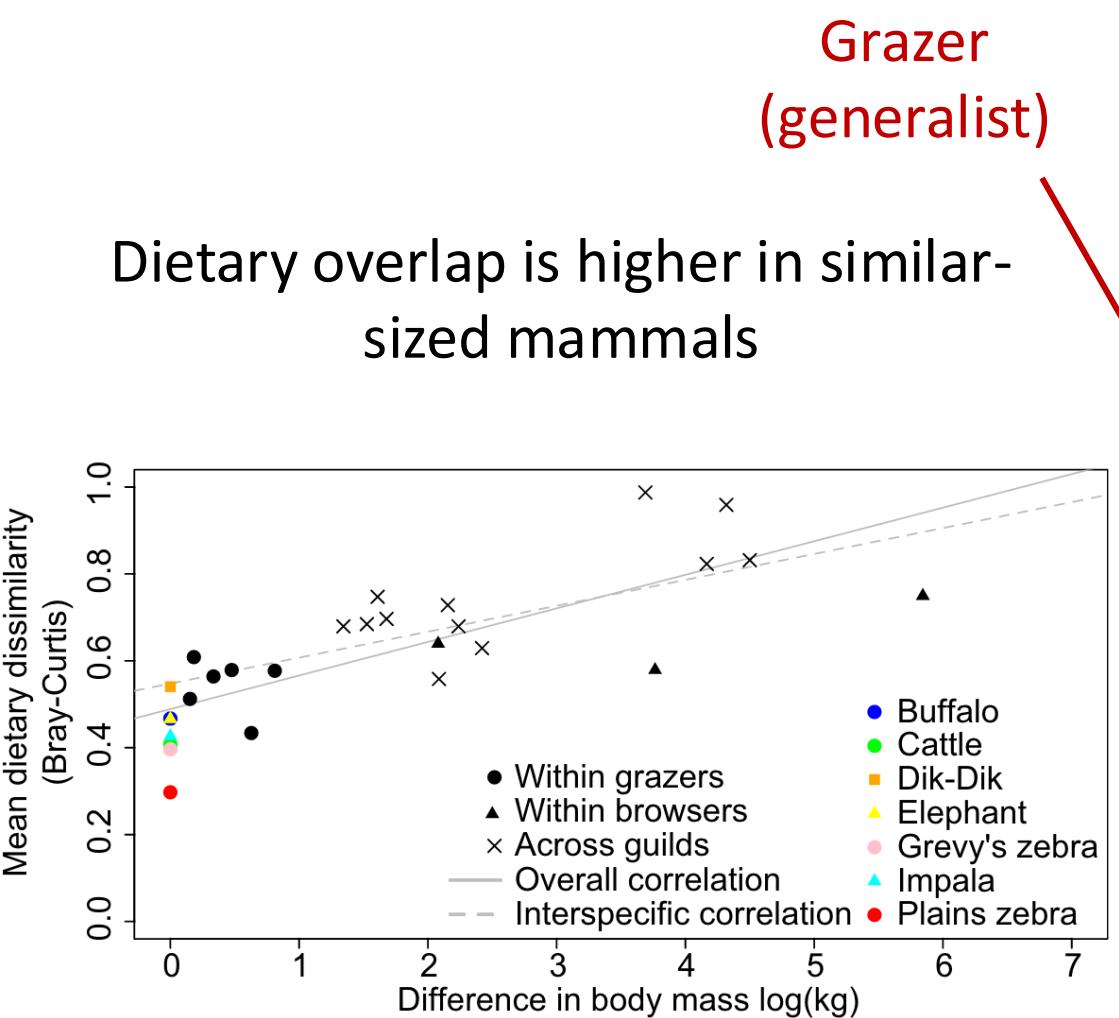
Niche partitioning enables organisms to avoid competitive exclusion

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



Kartzin et al. 2015. PNAS.

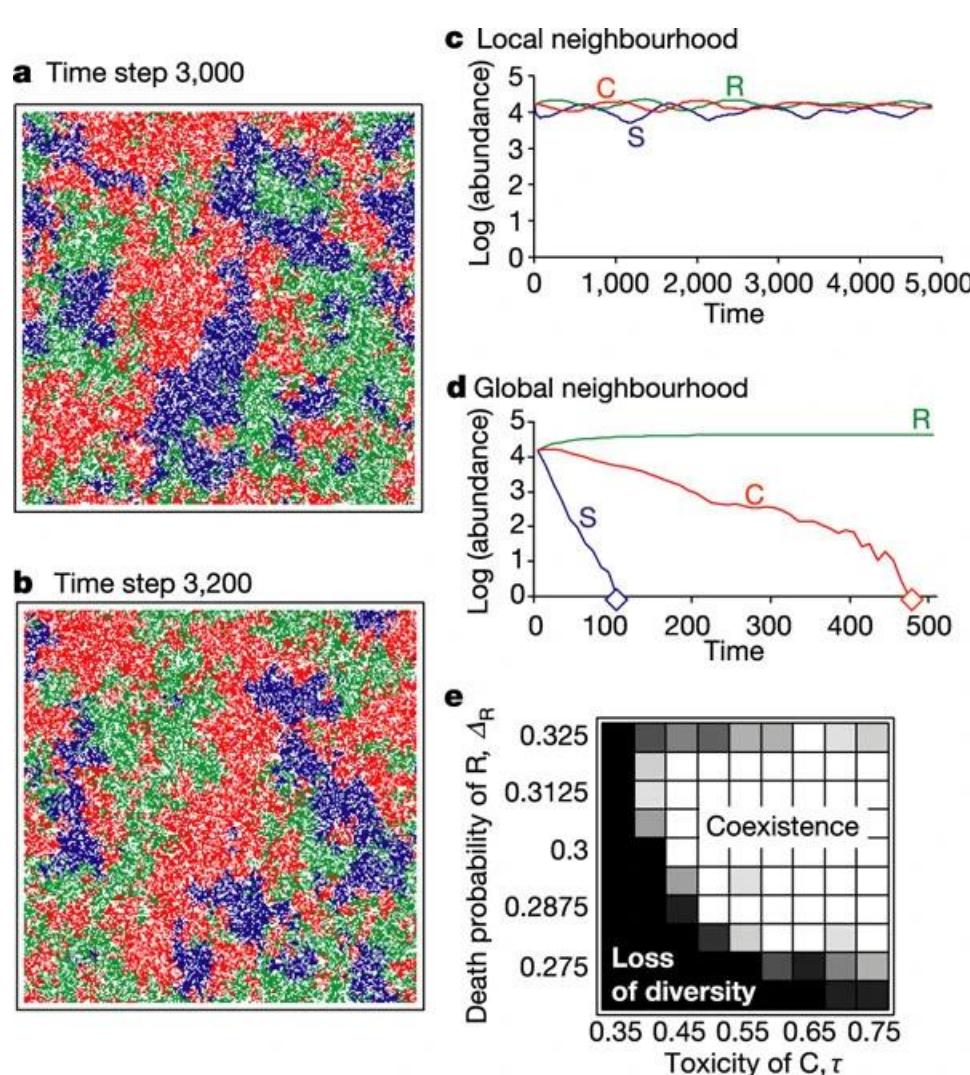
Niche partitioning enables organisms to avoid competitive exclusion



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

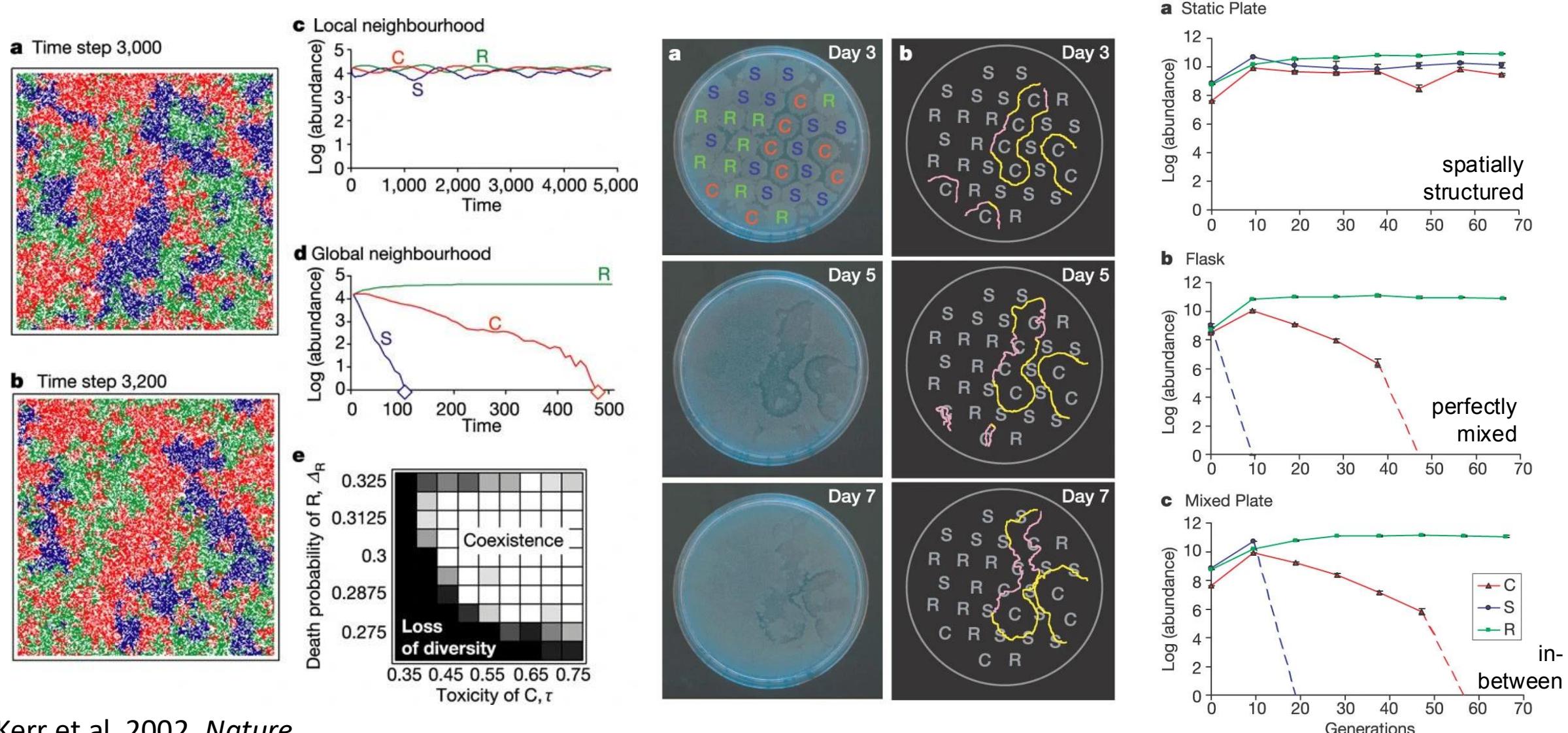
**Browser
(specialist)**

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

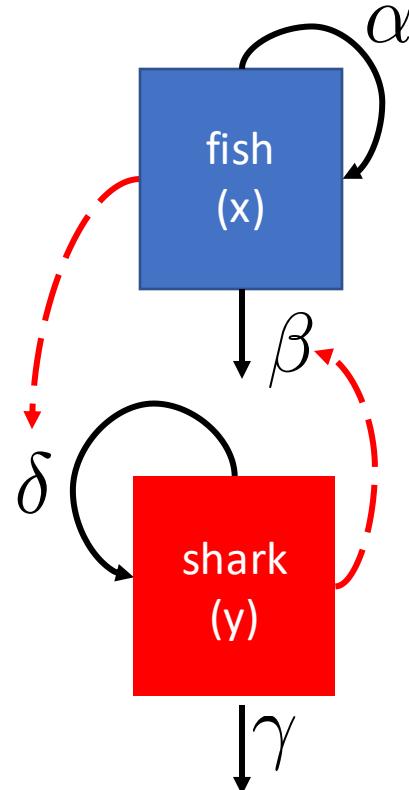


- The authors modeled a community of 3 strains of *E. coli* with **overlapping resource requirements** (C,R, and S), occupying distinct spatial patches in a metapopulation.
- They produced simulations allowing for a perfectly mixed population (global neighborhood), or a population in which dispersal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

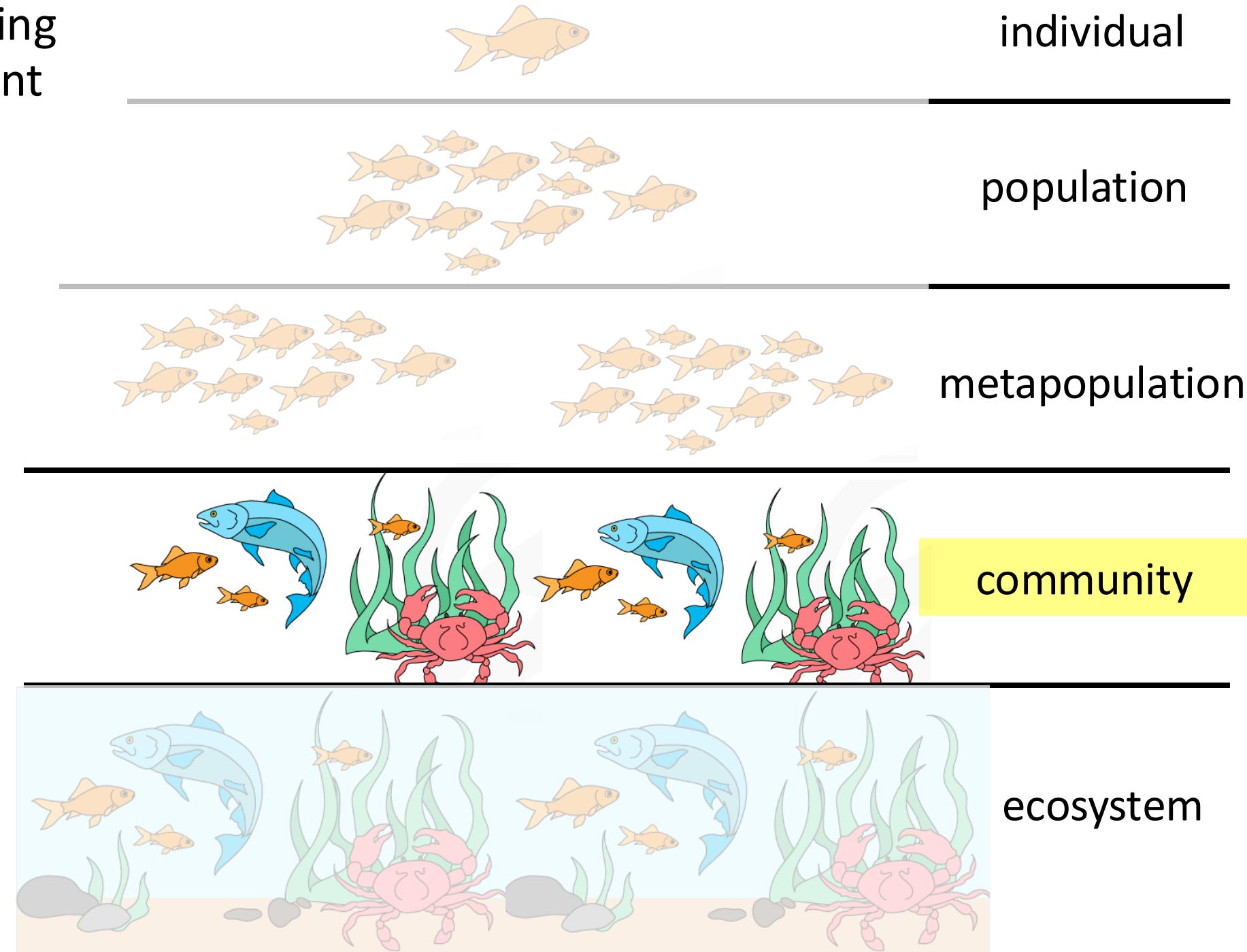
Sometimes **stochasticity** and **space** are all you need to ensure **coexistence** in a competitive environment



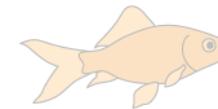
Community = interacting populations of different species



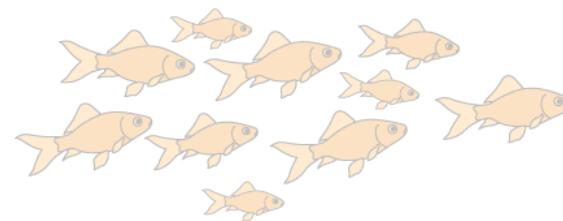
How does fish abundance **vary** with changes in shark abundance?



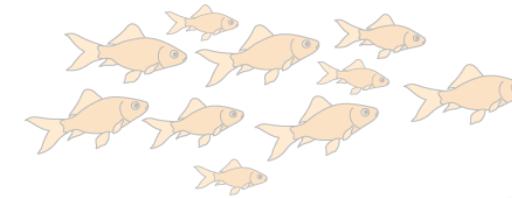
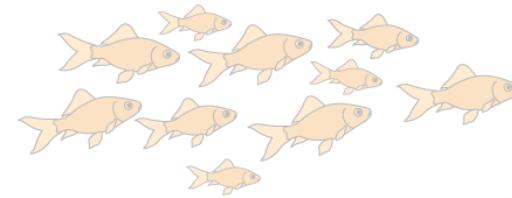
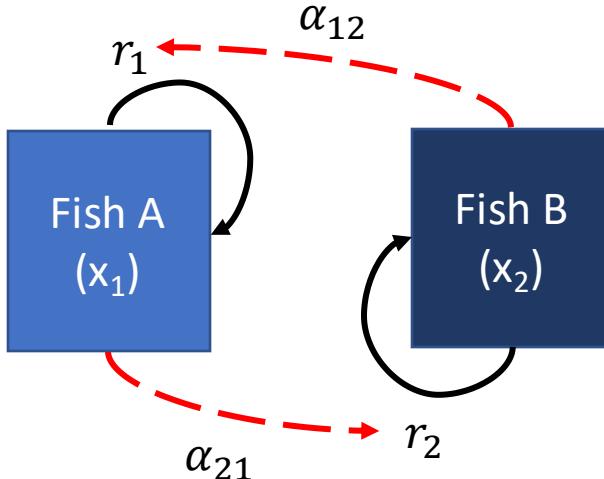
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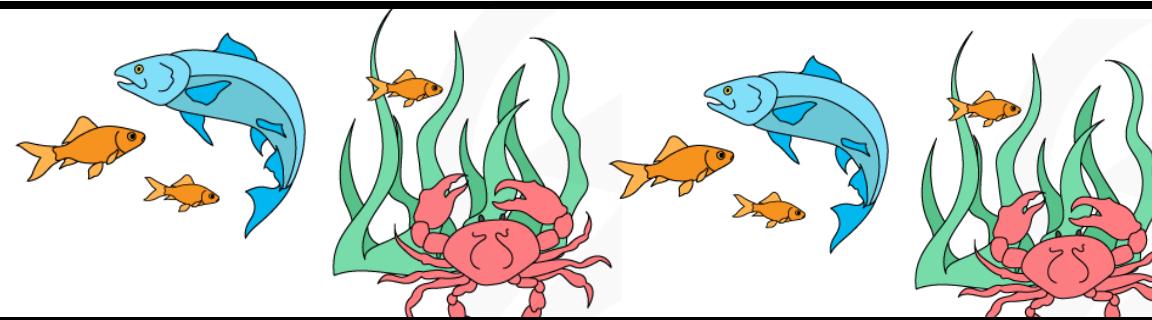
individual



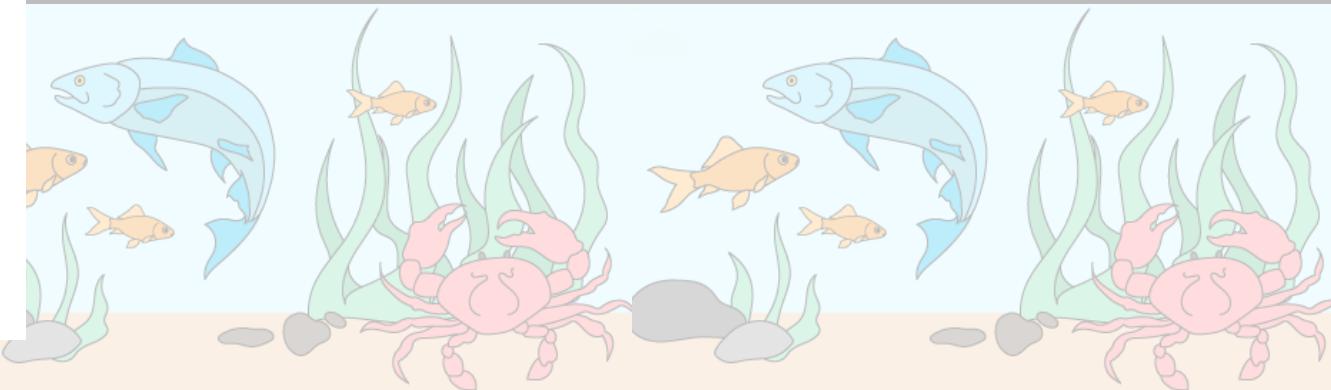
population



metapopulation

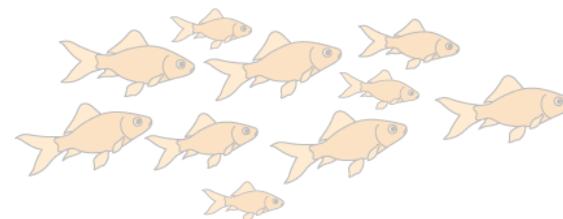
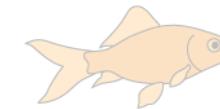


How does the abundance of **fish species A** vary with changes in the abundance of **fish species B**?

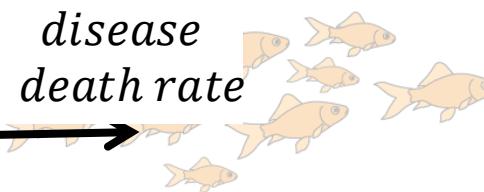


Community = interacting populations of different species

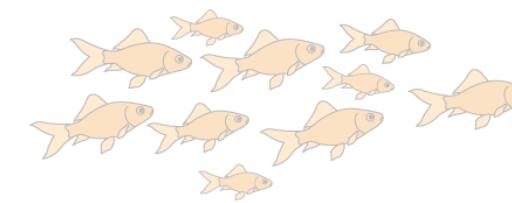
individual



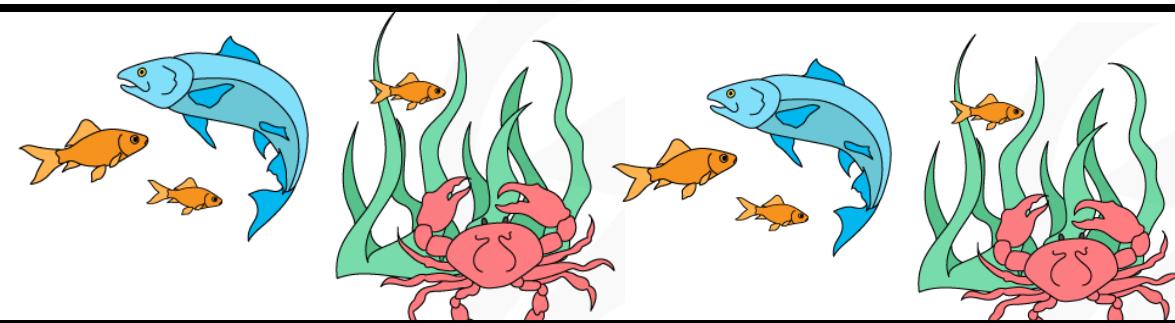
population



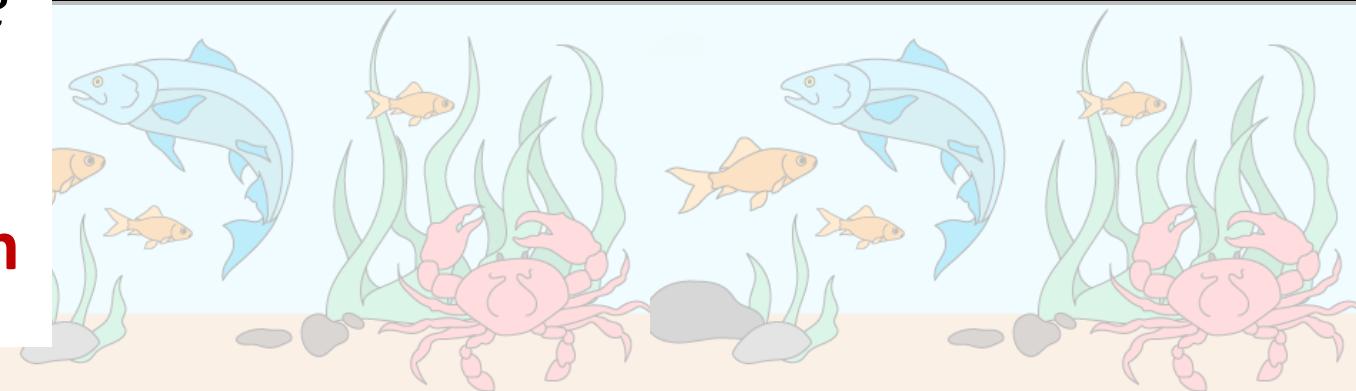
metapopulation



community



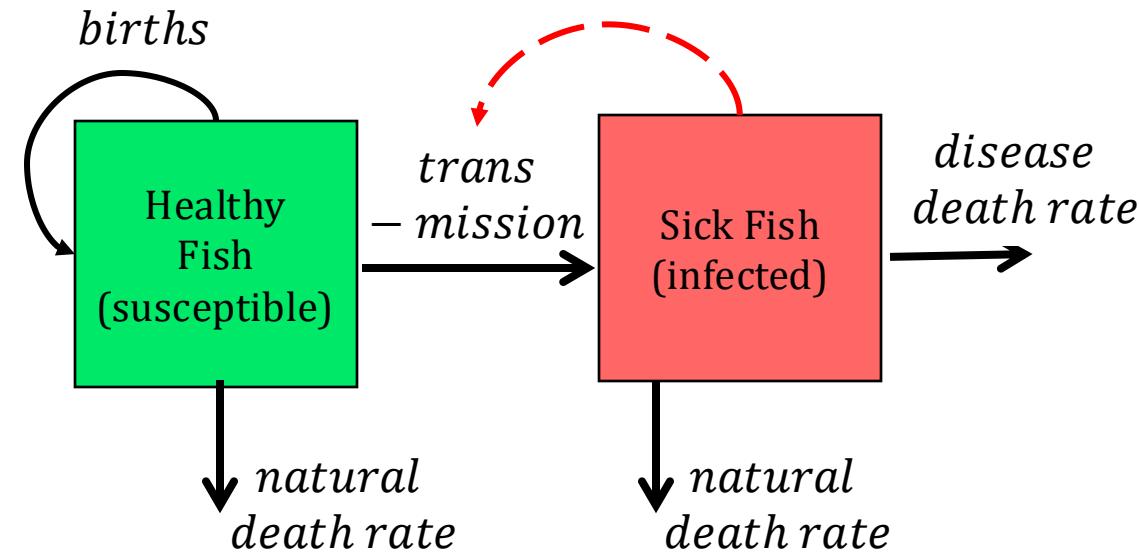
ecosystem



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

Community = interacting populations of different species

Species can interact in several distinct ways.



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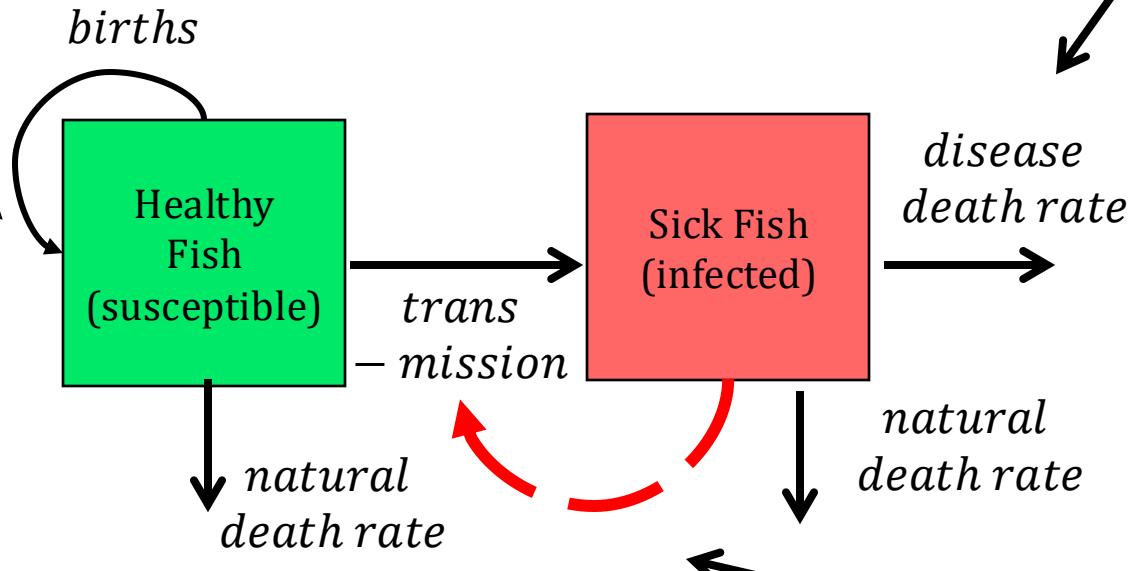
- **Mutualism** – both species benefit
- **Commensalism** – one species benefits, the other is unaffected
- **Predation** – one species benefits, the other is harmed (eaten!)
- **Competition** – two species compete for the same limiting resource, both harmed by the interaction
 - Direct = wolves and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

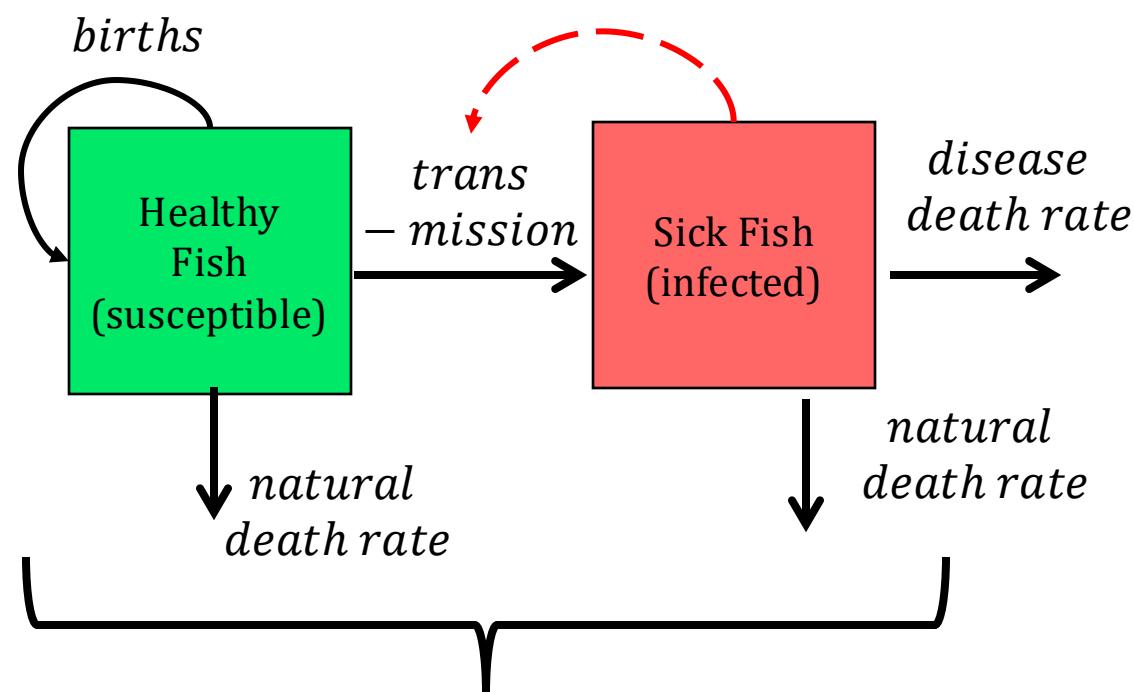


Solid lines are processes by which individuals in each population move between boxes.

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



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**.

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

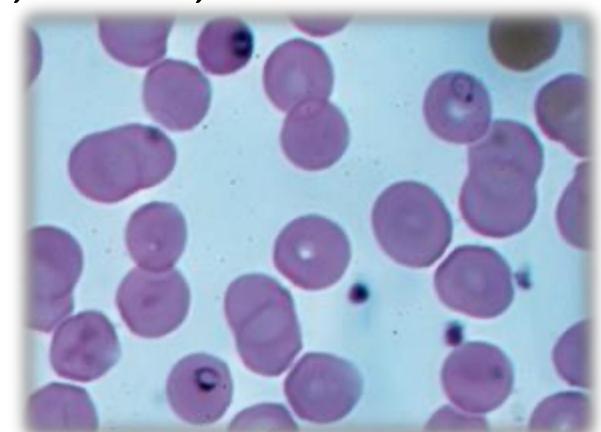
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Pathogen: *Yersinia pestis*
Disease: Plague

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

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

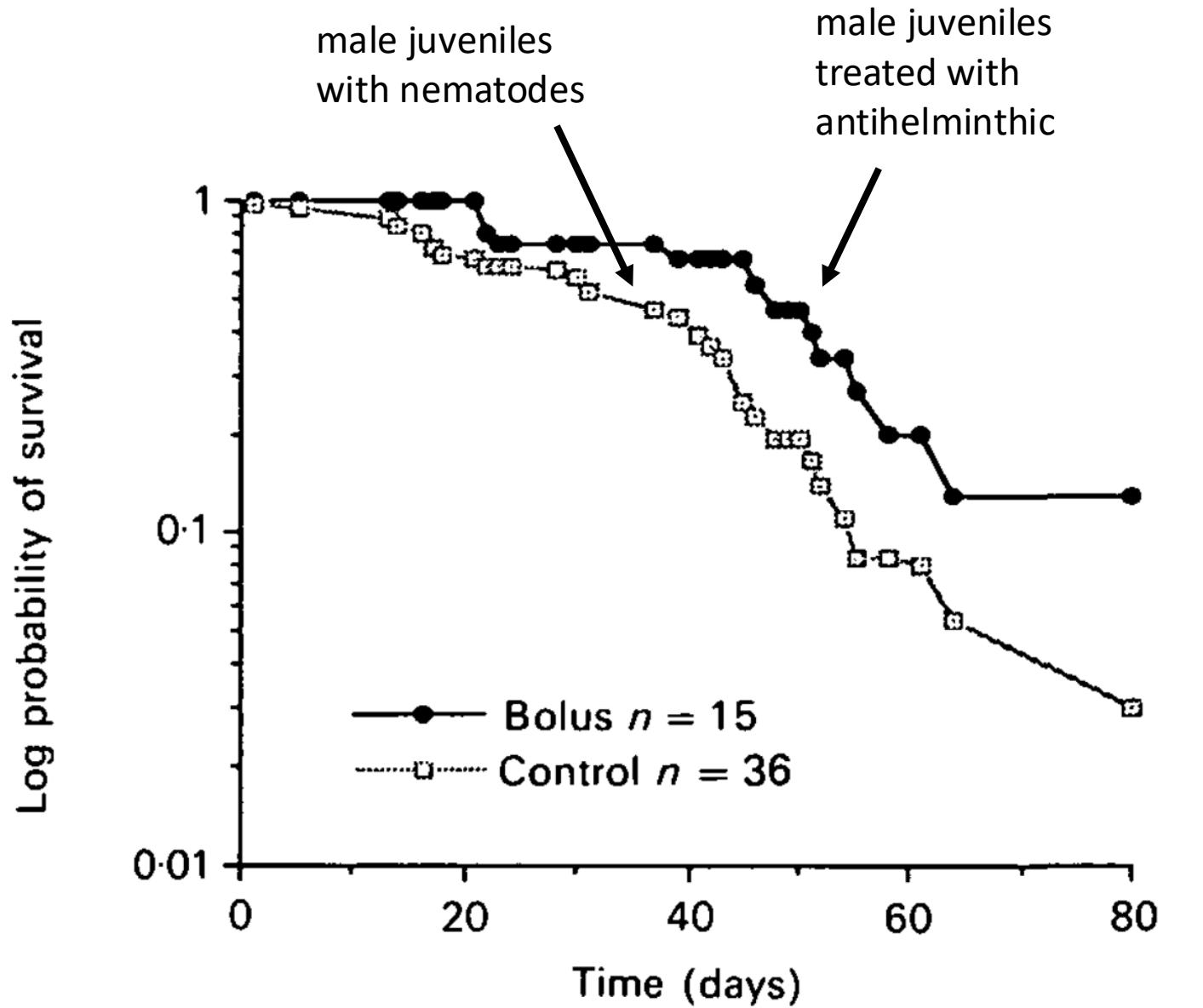
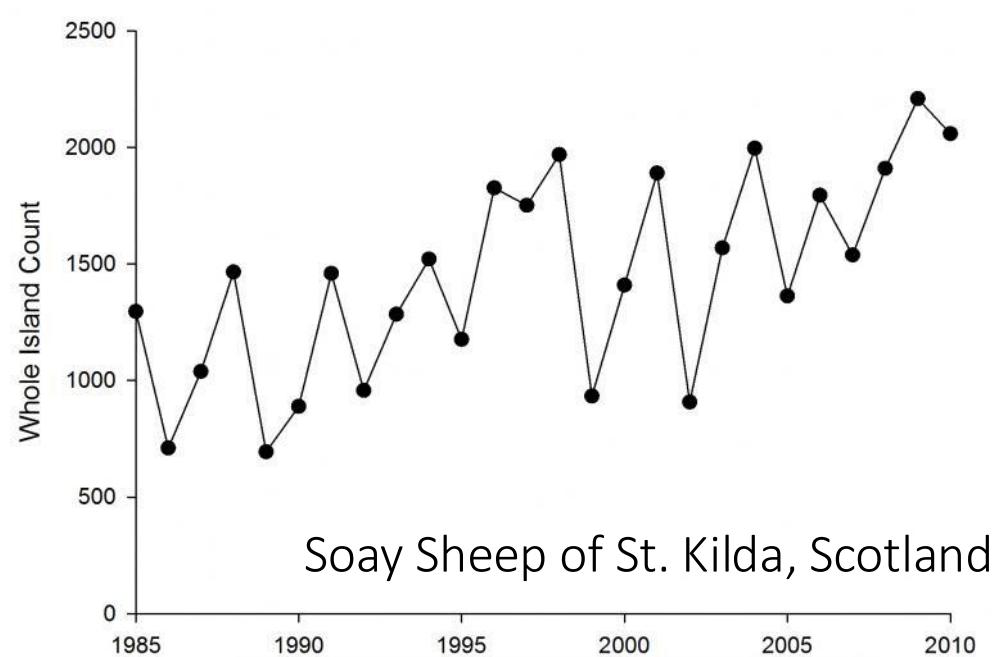


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- Pathogen: a microorganism that can cause disease
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- Host: the organism on/in which the parasite/pathogen lives
 - Ex: Soay sheep, grapes
- Vector: an arthropod agent that carries and transmits a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



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

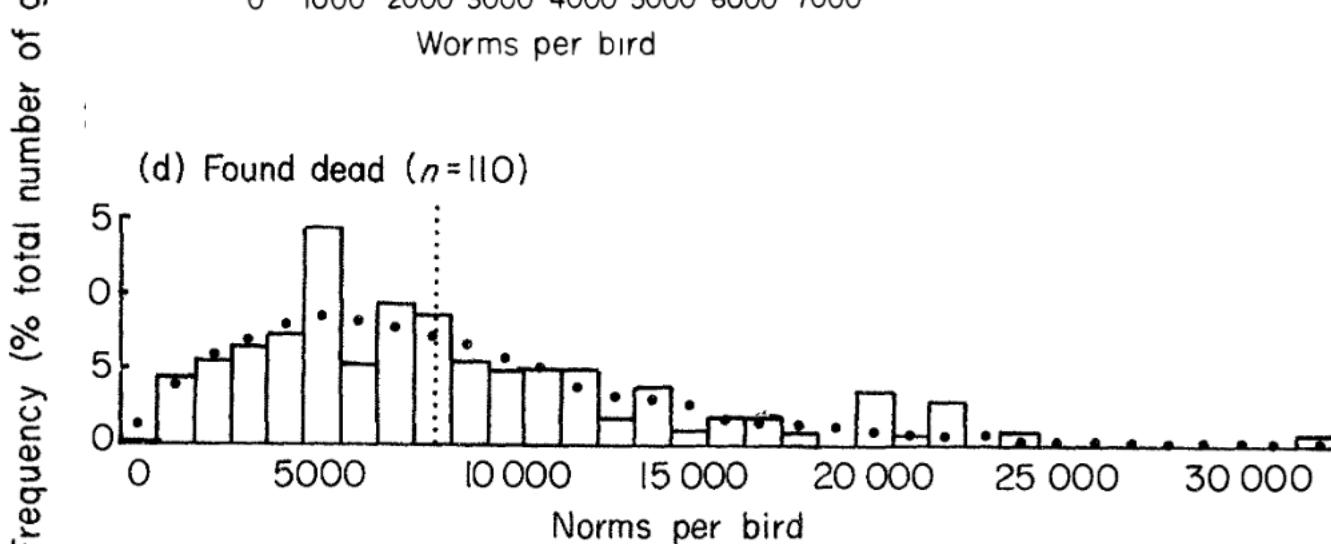
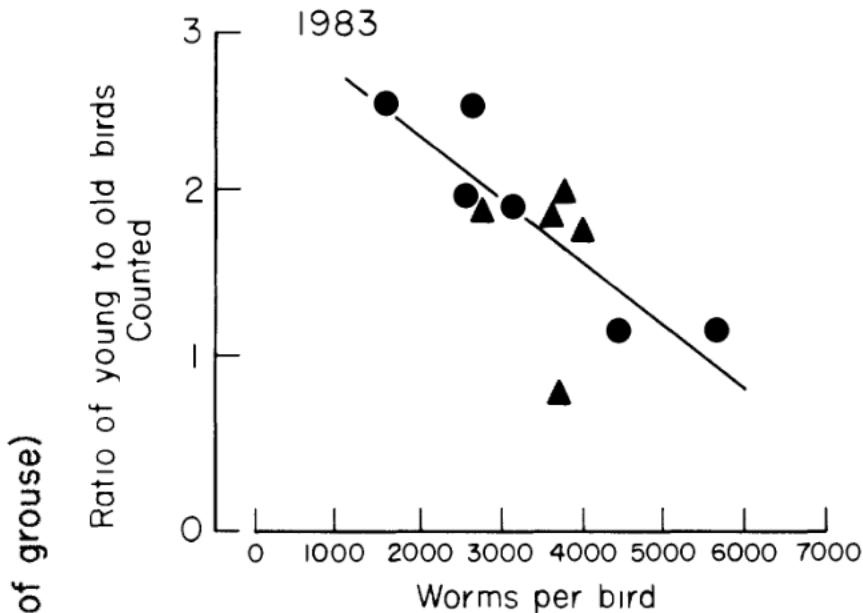


Early work in red grouse experimentally demonstrated the power of parasites to regulate populations.



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

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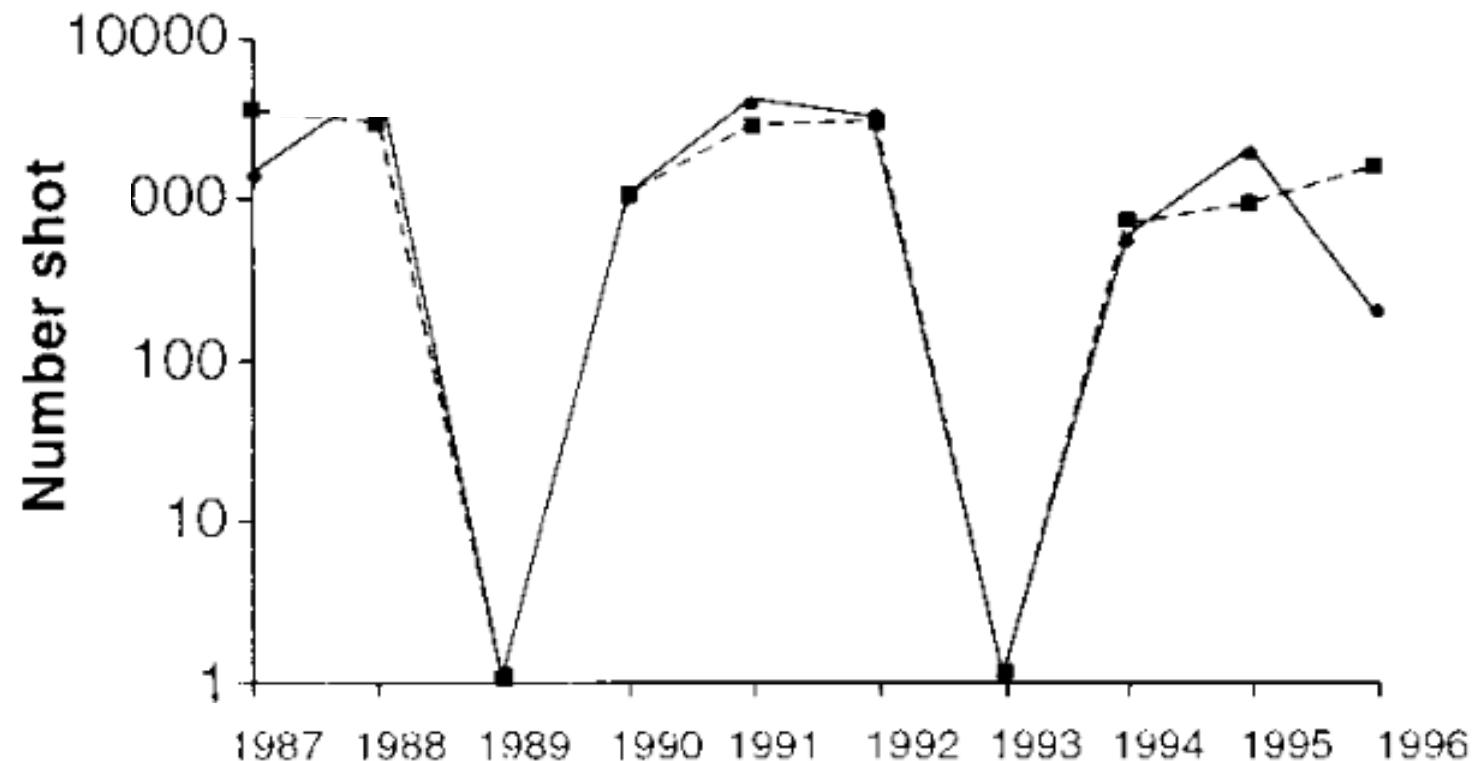


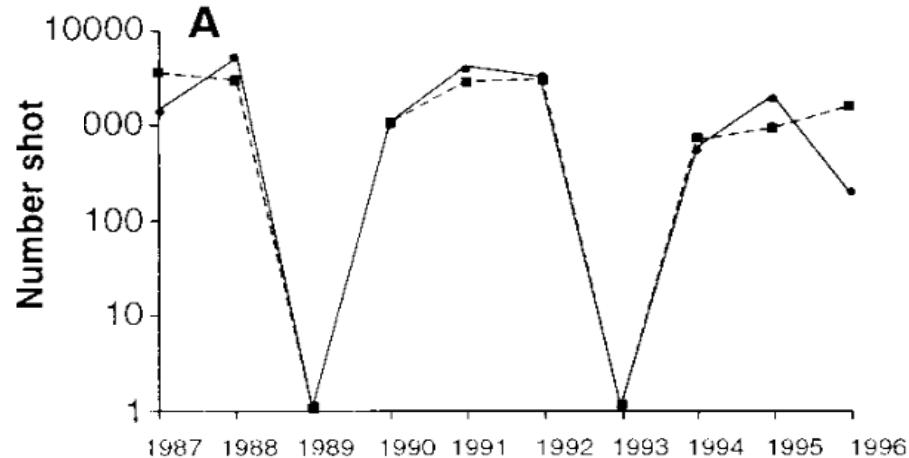
Higher burden of the intestinal strongyle worm, *Trichostrongylus tenuis*, both reduces breeding success and increases mortality in red grouse.

Hudson 1986. *J Animal Ecology*.

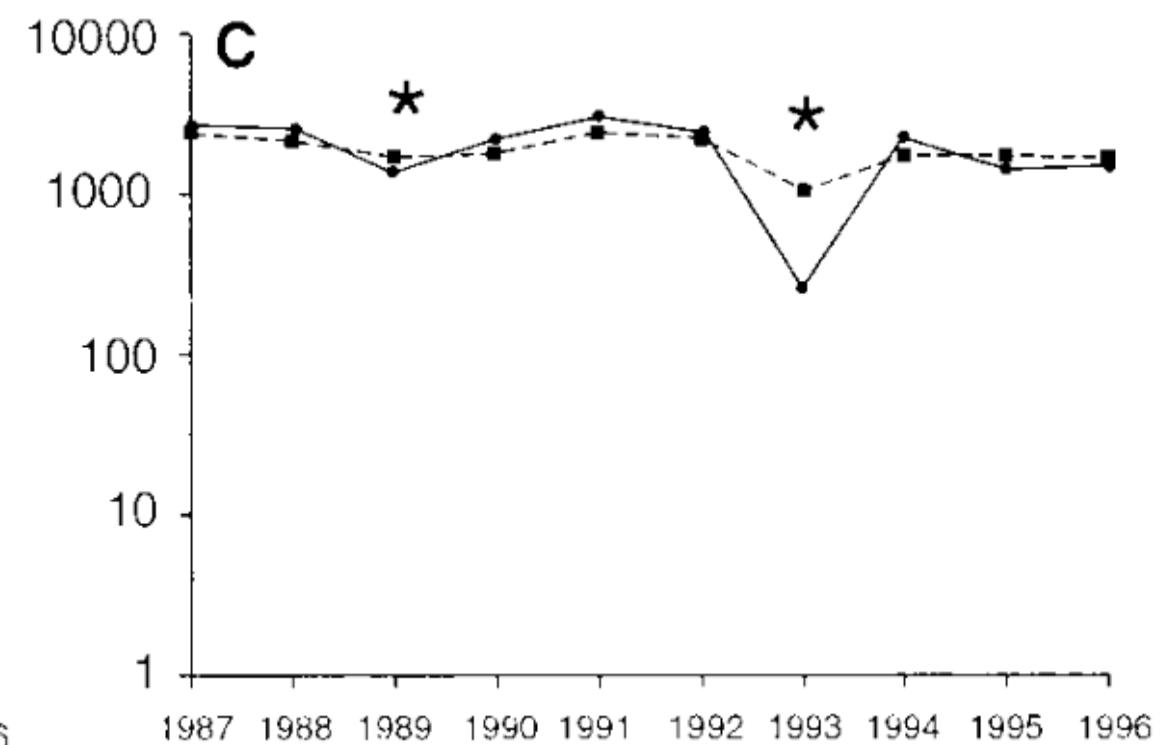
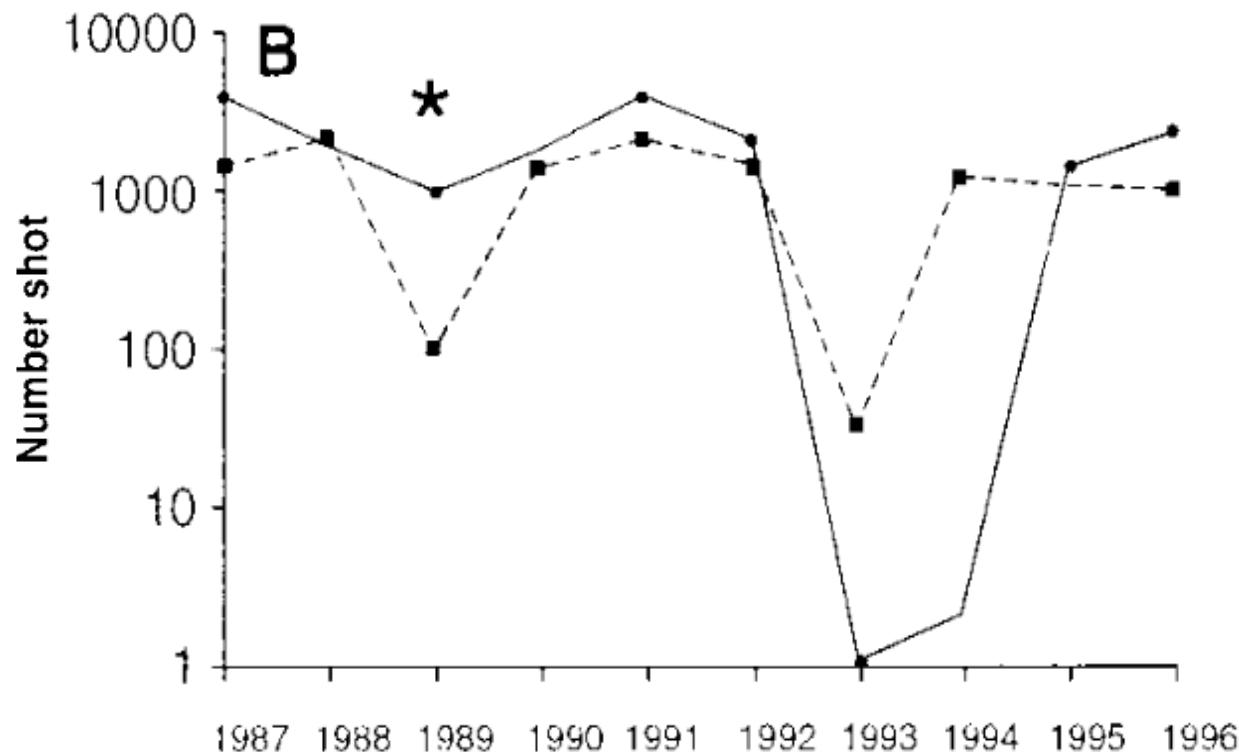
Hudson et al 1992. *J Animal Ecology*.

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!

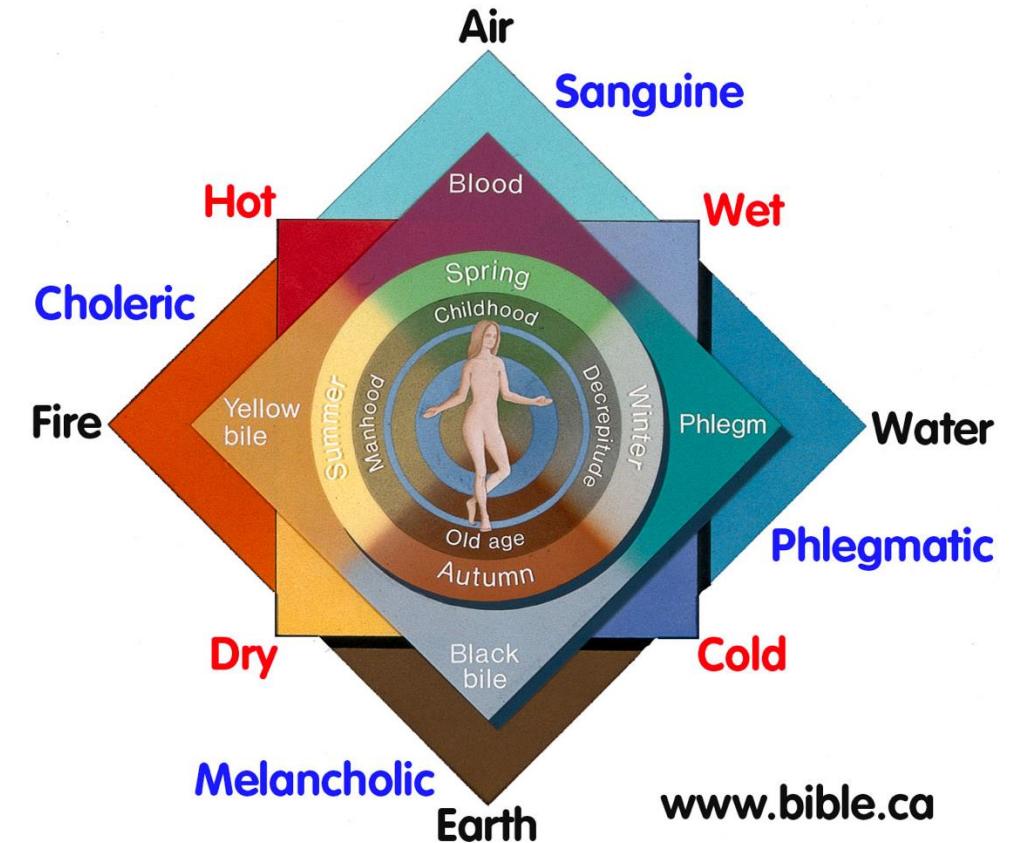


Parasites and pathogens have also shaped human history.

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

The Four Humors of Hippocratic Medicine
450 BC - 1858 AD
Melancholy Blood (depression)



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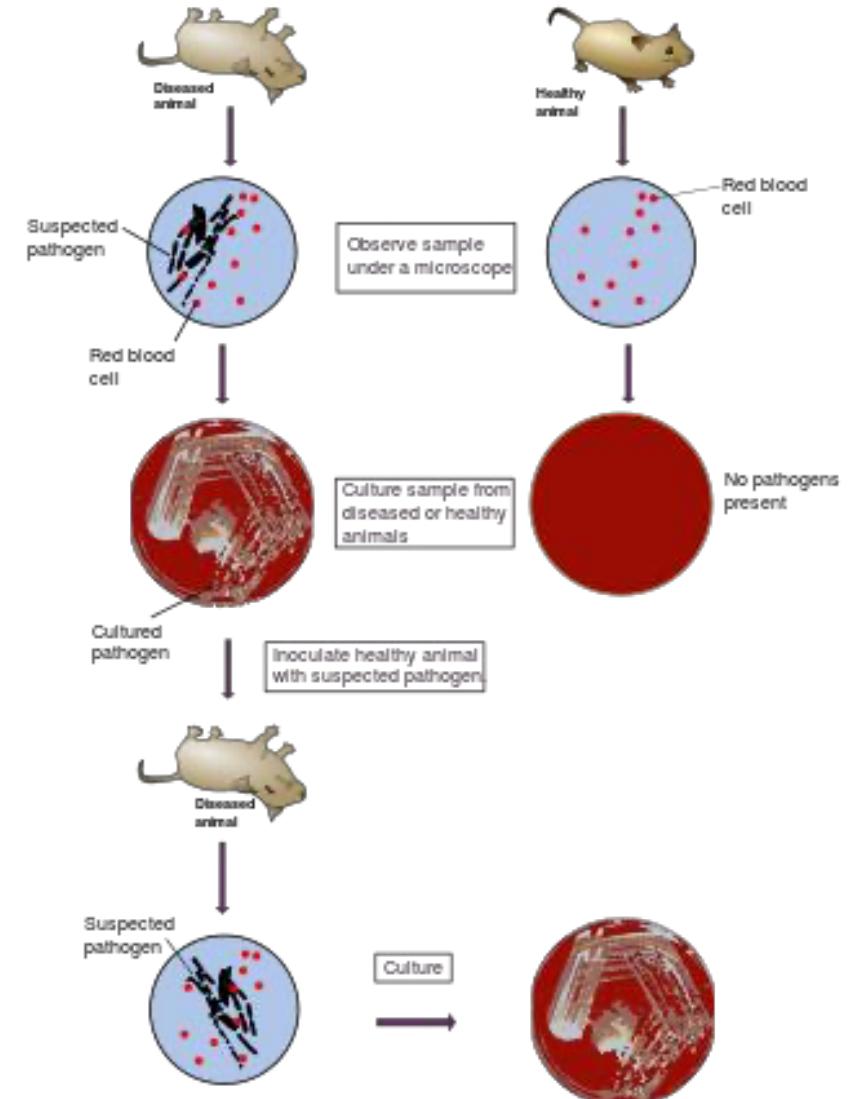
1. **Four Humors:** Hippocrates (c. 400 BC) wrote that disease results from an imbalance of the four humors
2. **Miasmatic Theory:** Extension of Hippocrates that lasted through the 1800s – idea that disease was caused by bad air. Popularized by Florence Nightengale



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 - Popularized by Florence Nightengale
3. **Germ Theory of Disease:** Idea that disease results from germs
 - Leuwenhoek's microscope (1675)
 - Koch's postulates (1890)



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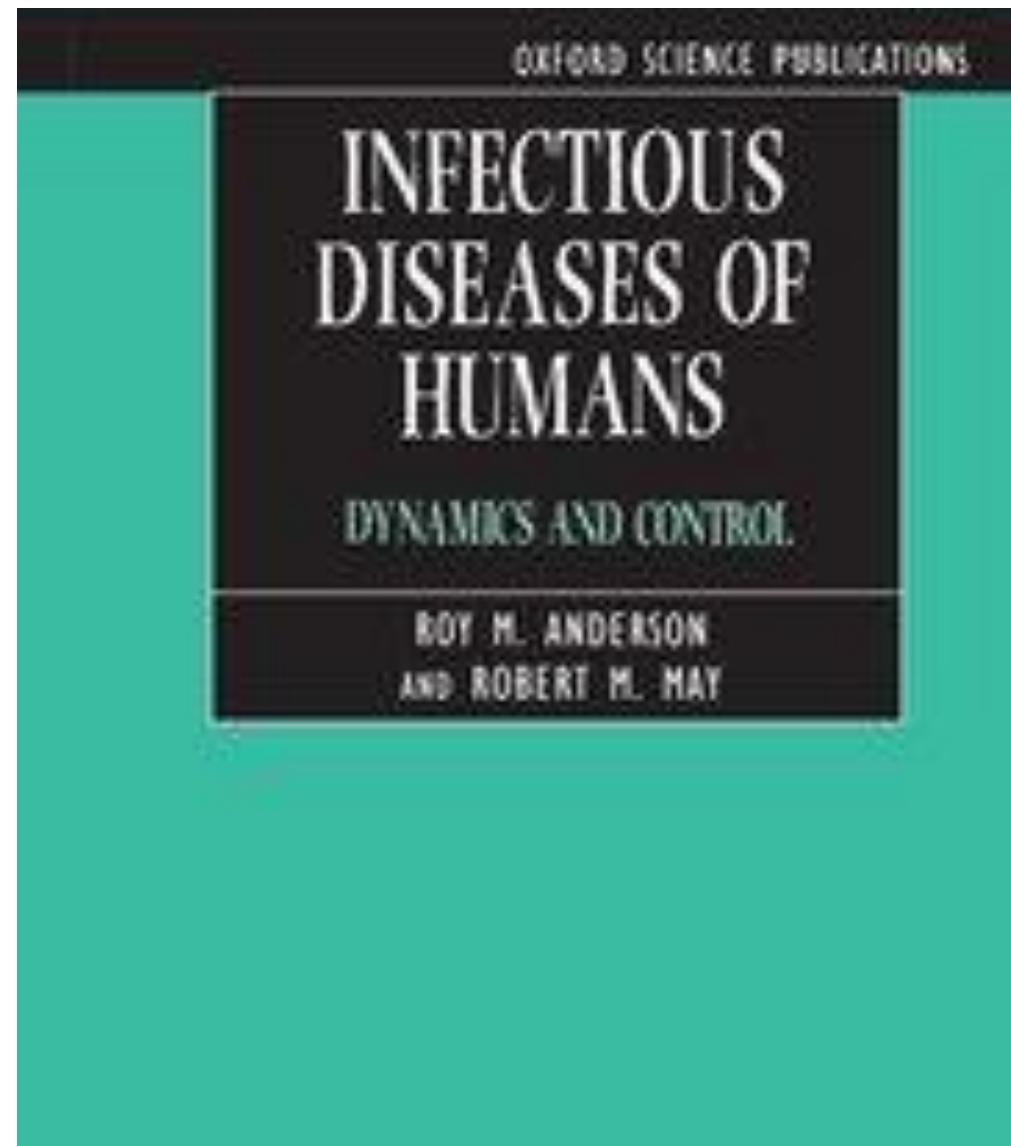
4. **Classic epidemiology**

- Risk factors for disease = John Snow (1854)



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4. **Classic epidemiology**
 - Risk factors for disease = understand ***patterns***
5. **Population biology of infectious disease**
 - Understanding the ***process*** of infectious disease ***transmission***. = Kermack and McKendrick (1927); Anderson and May (1991).



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



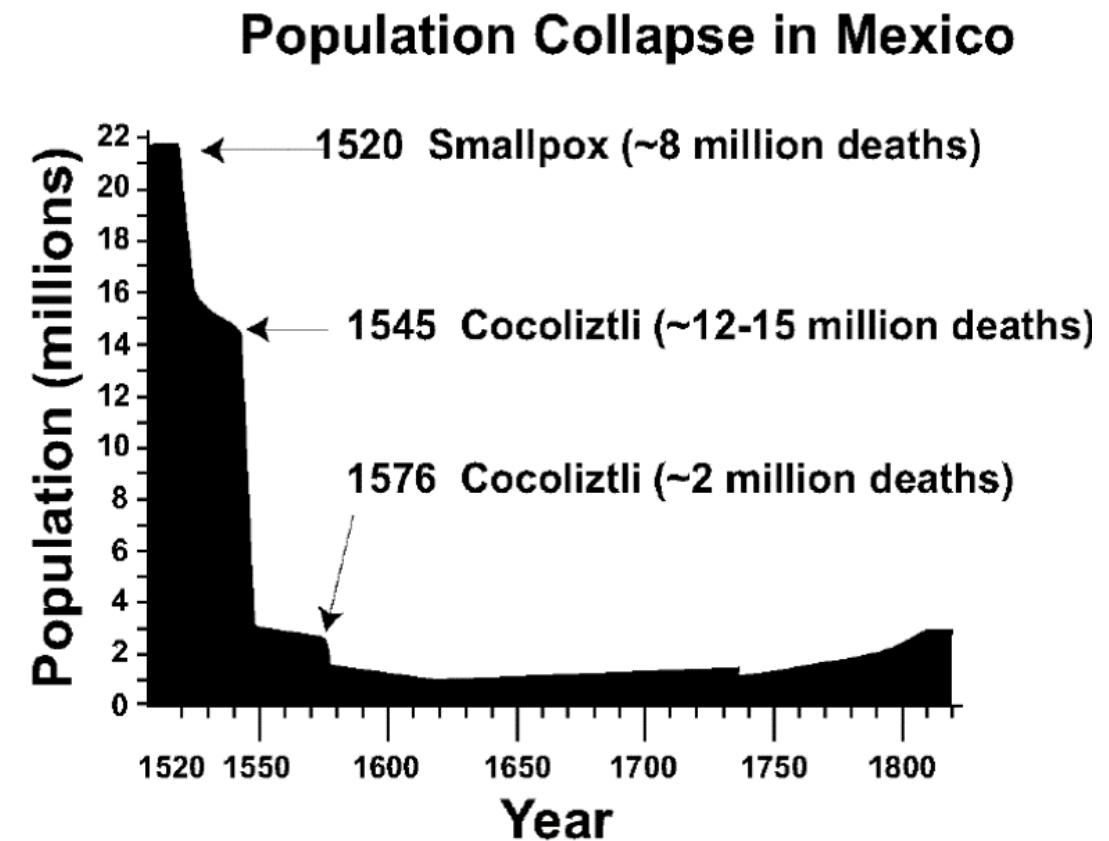
Parasites and pathogens have also shaped human history.

- 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



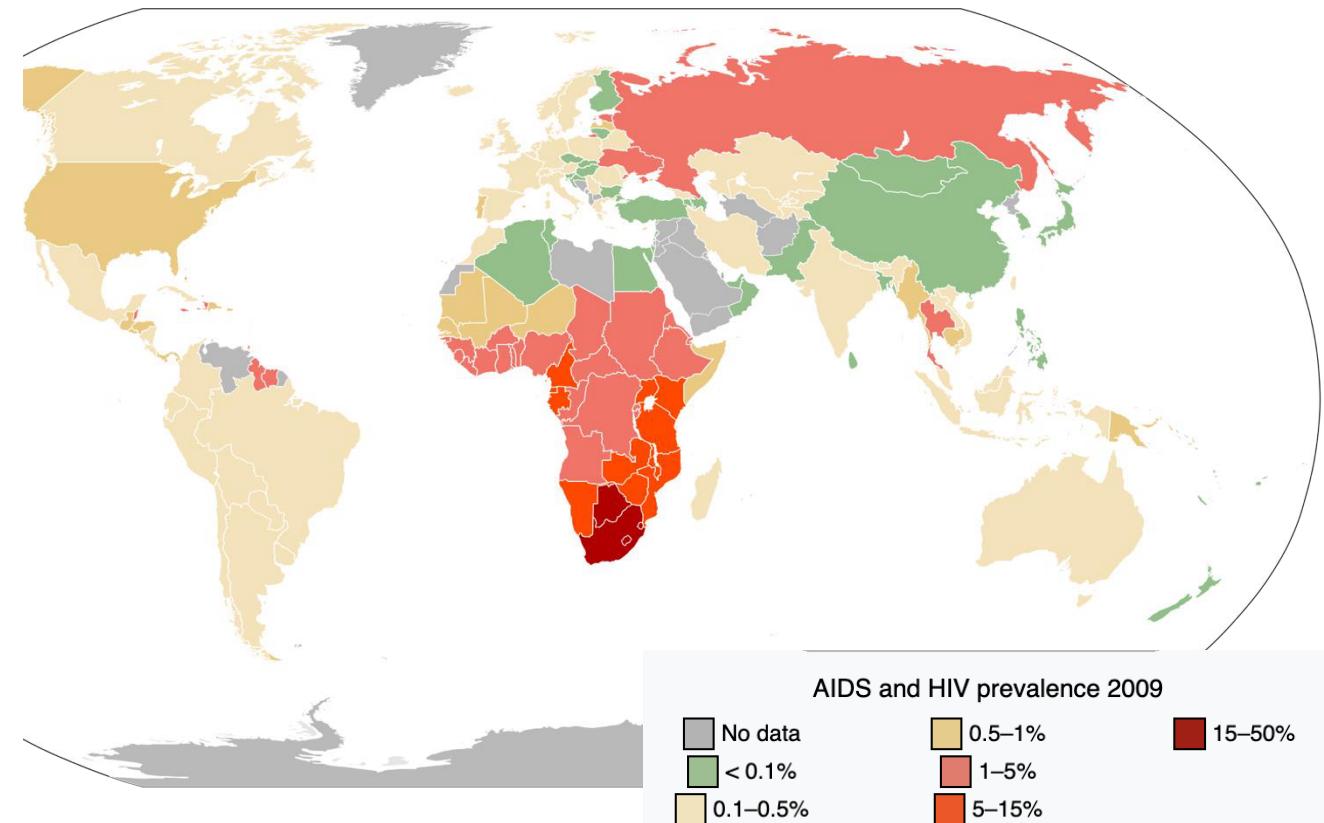
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- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
- Spanish Influenza (1918-1920)
 - 17-100 million deaths worldwide.
 - 1-5% of global population
 - 2nd-most devastating pandemic in history (after Black Death)



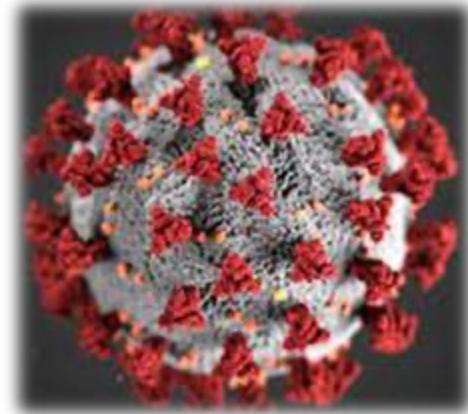
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- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
- Spanish Influenza (1918-1920)
- HIV (~1960-now)
 - >40 million deaths and counting
 - Prevalence still >20% in some countries in southern Africa



Parasites and pathogens have also shaped human history.

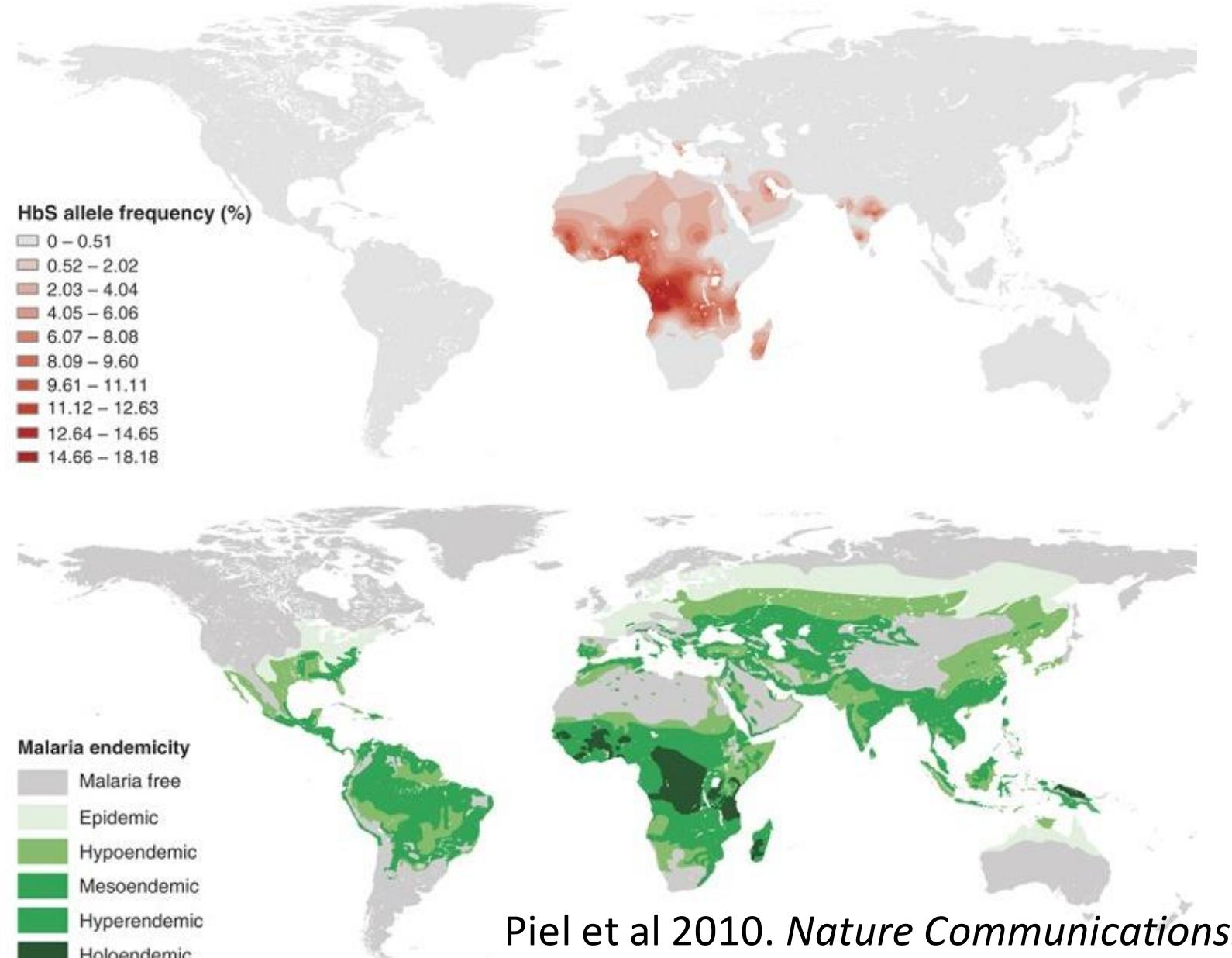
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- Cocoliztli (1545-1548)
- Spanish Influenza (1918-1920)
- HIV (~1960-now)
- COVID-19 (2019-2022)
 - ~7-29 million deaths worldwide
 - ~0.1-0.4% of population



Parasites and pathogens have also shaped human DNA.

Sickle cell anemia

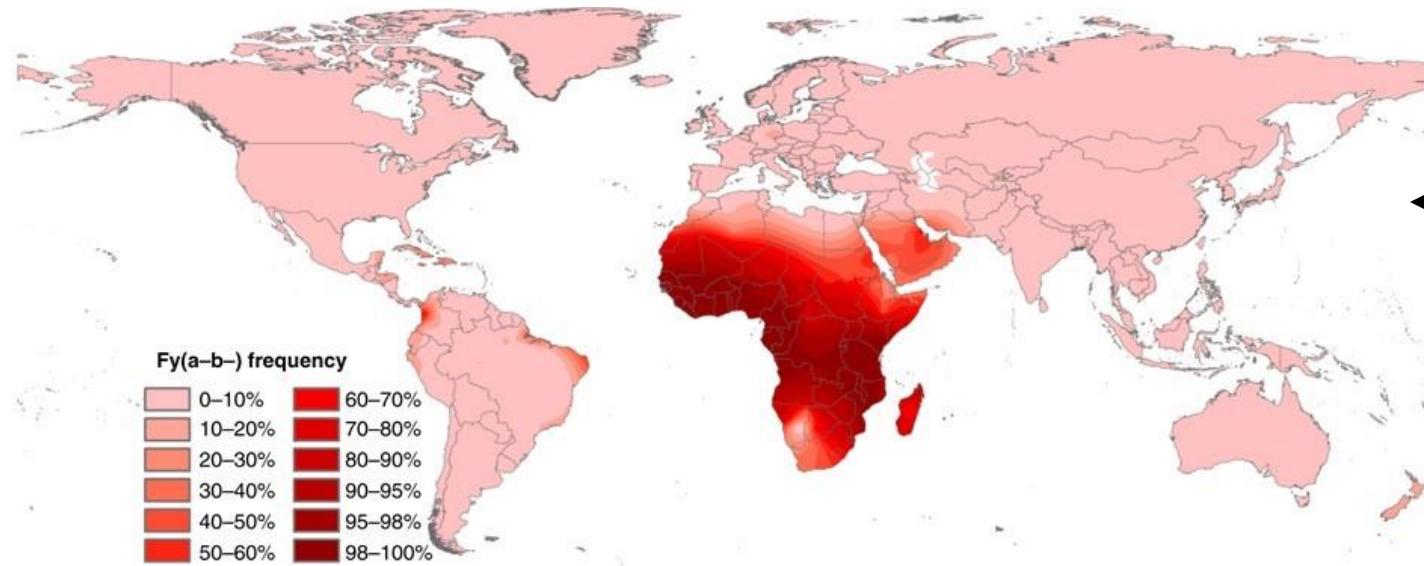
- The HbS allele confers resistance to malaria but also results in sickle cell anemia when homozygous.
- Natural selection has favored this trait in malaria-endemic regions of the planet.
- As of 2021, WHO estimates 247 million malaria cases worldwide and >600,000 deaths, 95% in Africa.
- Children <5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



Parasites and pathogens have also shaped human DNA.

Duffy antigen

Required for *P. vivax* to enter RBCs

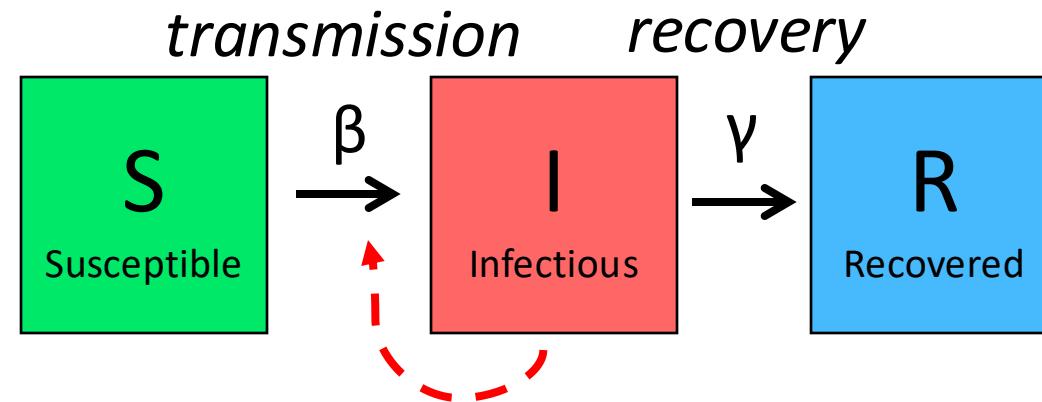


← Modeled distribution of Duffy-negative human population

Distribution *Plasmodium falciparum*

Distribution *Plasmodium vivax*

The SIR Model

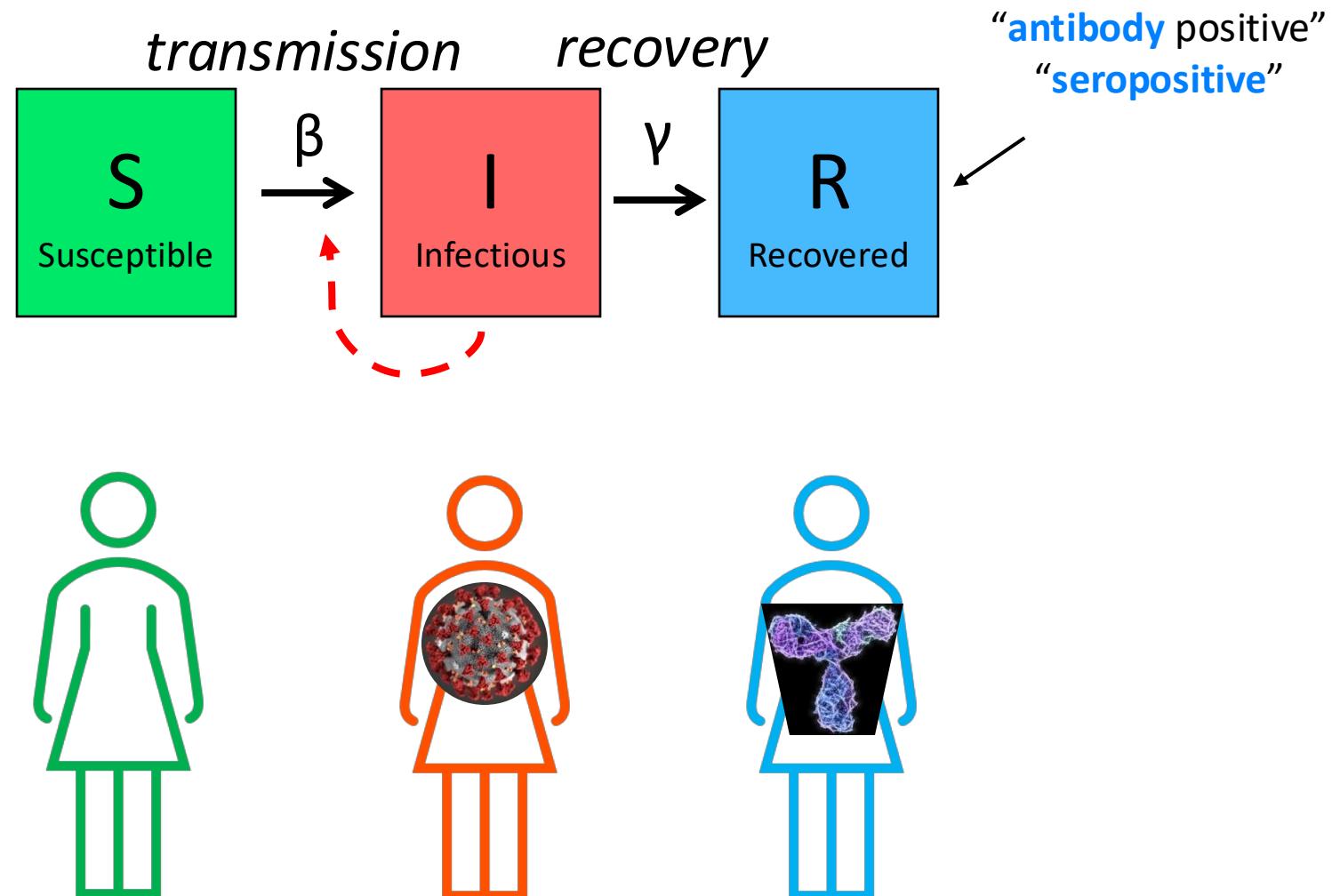


β = transmission rate

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**.



β = transmission rate

γ = recovery rate

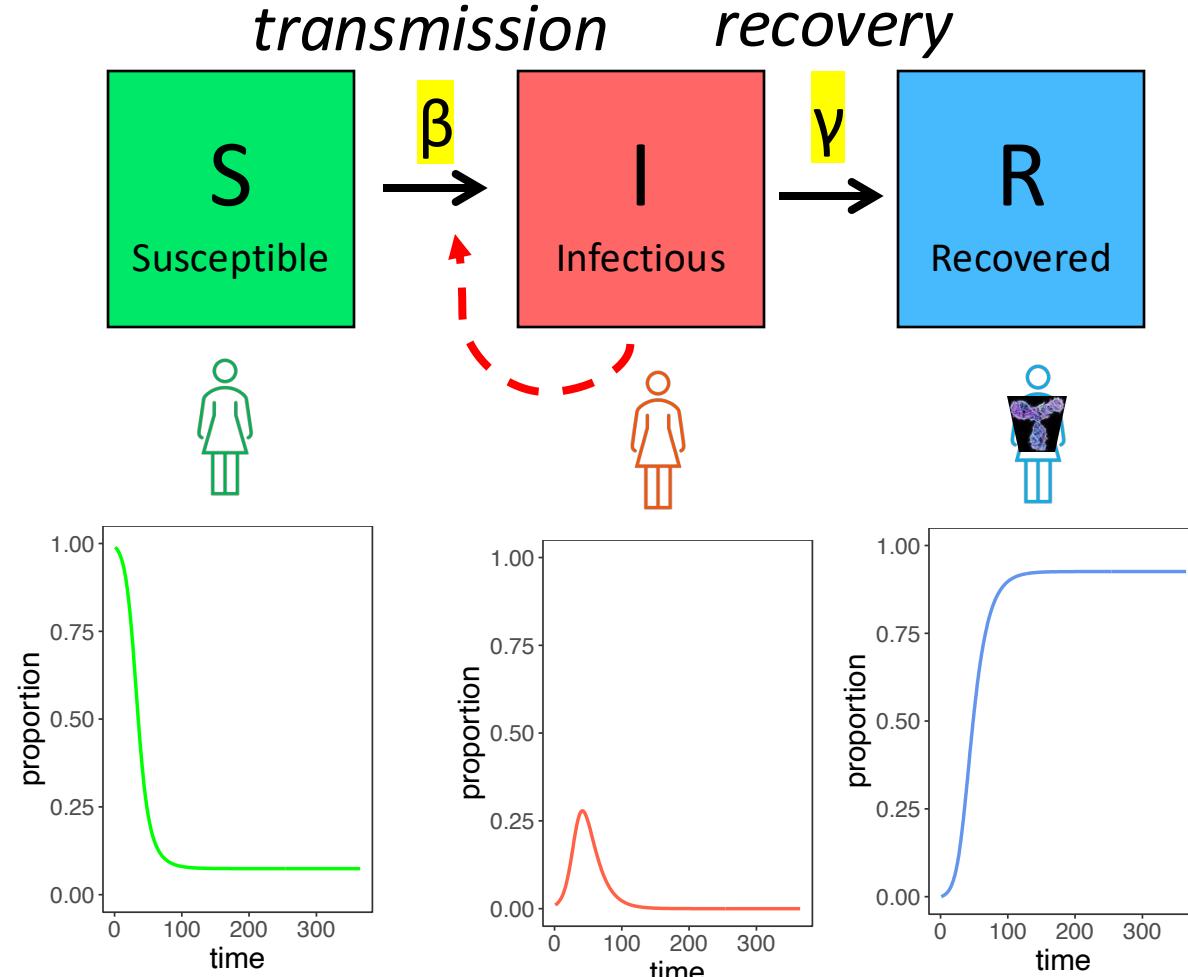
We use computers to simulate systems of equations in the SIR framework.

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

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

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

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



$$R_0 = \frac{\beta}{\gamma} \frac{\text{transmission}}{\text{recovery}}$$

β = transmission rate

γ = recovery rate

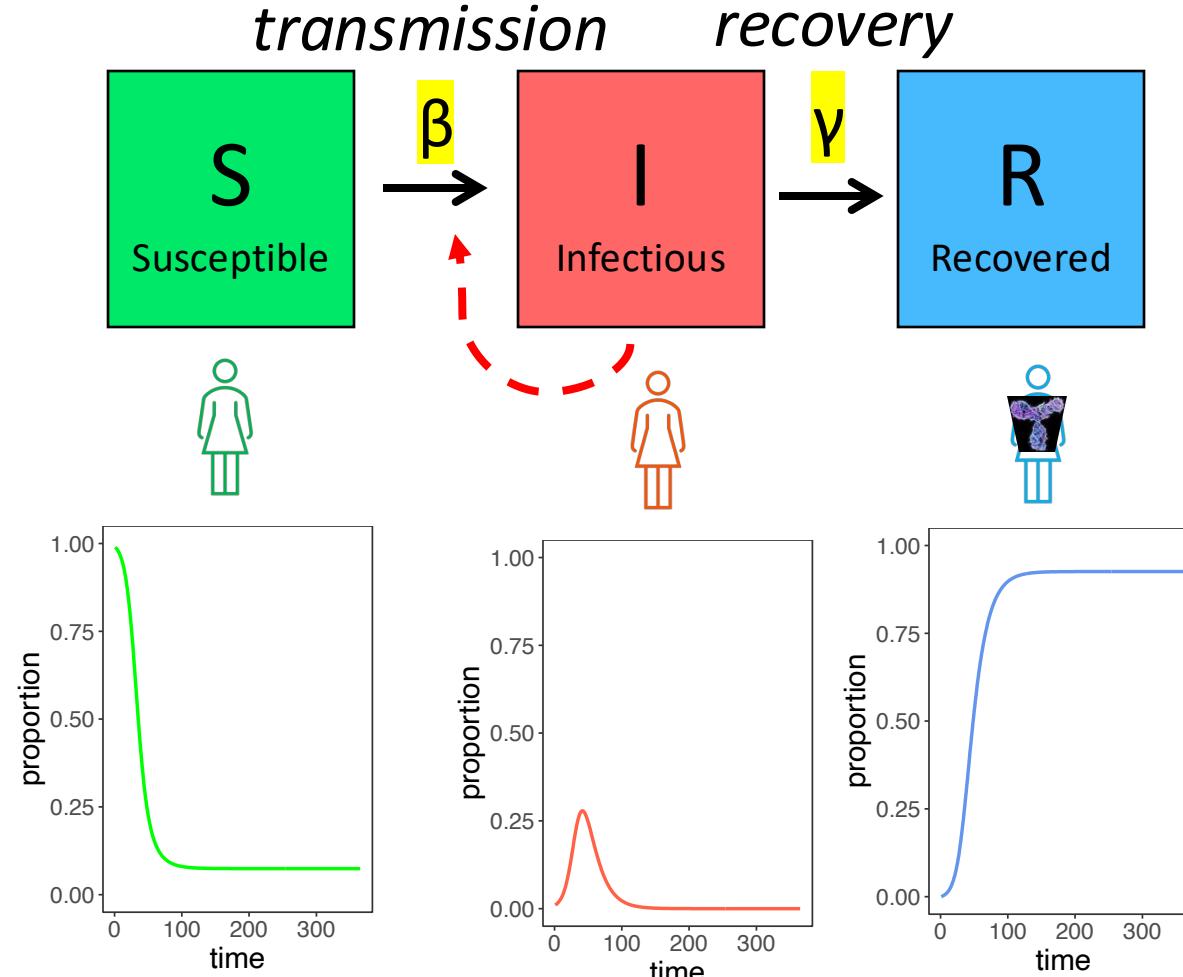
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$$\frac{dS}{dt} = -\beta SI$$

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

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

infections
created
infections
lost

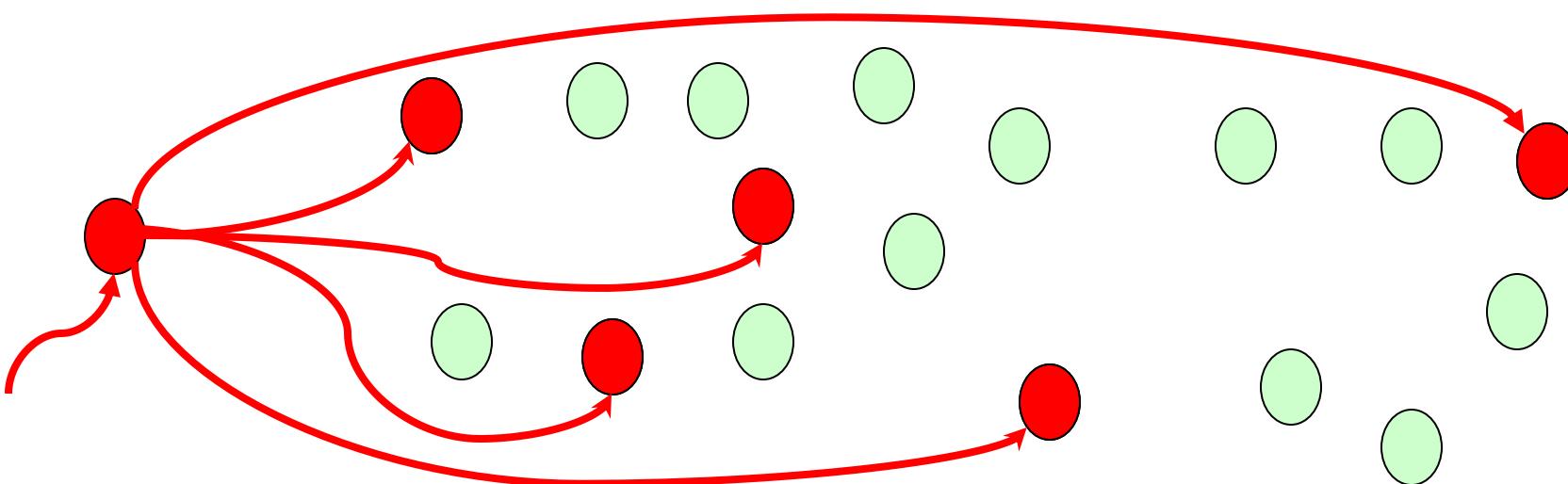
R_0 must be >1 for
a disease to start
spreading!

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

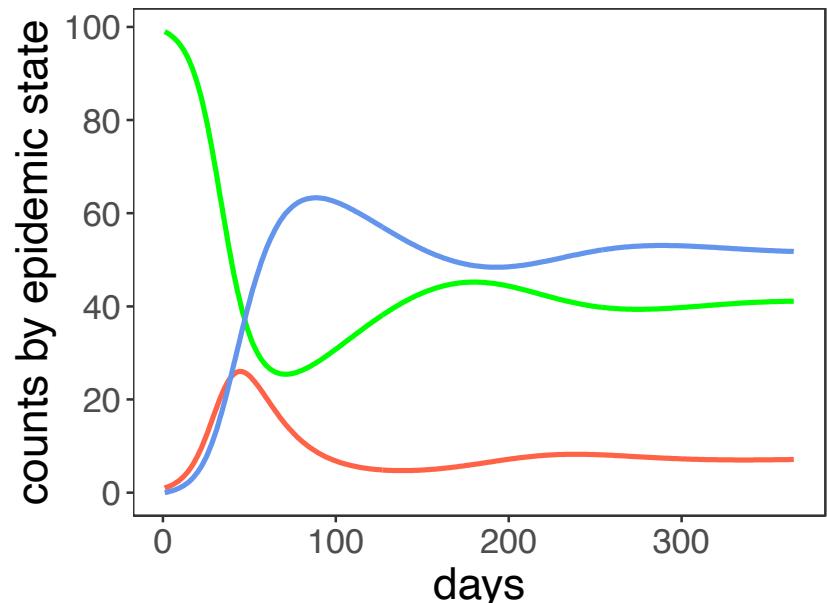
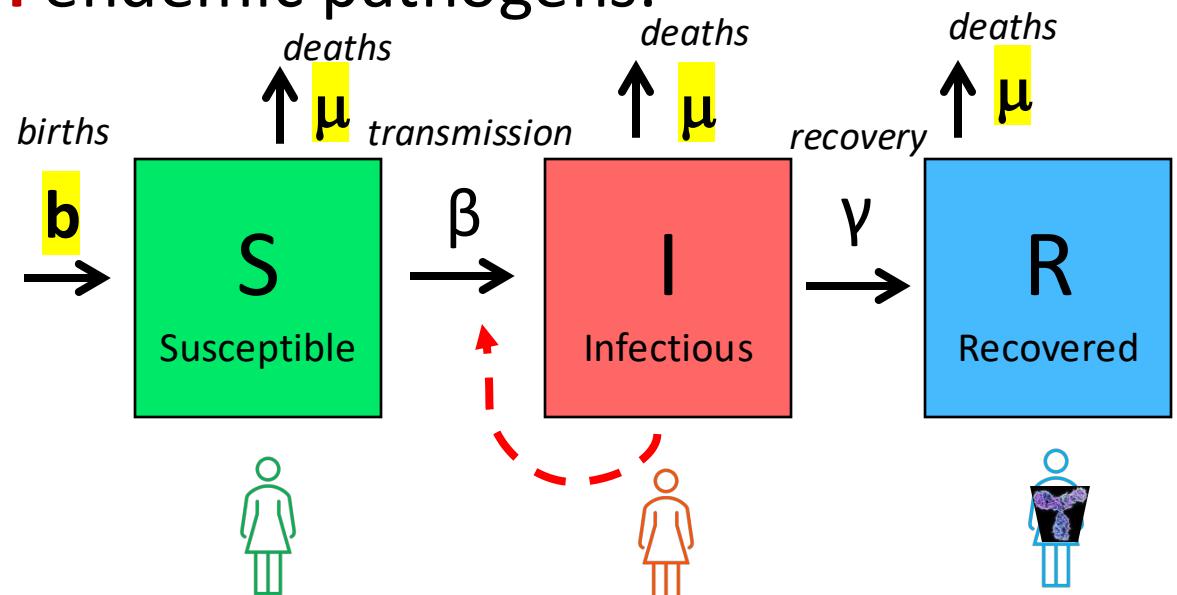


What is R_o ?

$R_o = 5$

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

$$\begin{aligned}\frac{dS}{dt} &= b(S + I + R) - \beta SI - \mu S \\ \frac{dI}{dt} &= \beta SI - \gamma I - \mu I \\ \frac{dR}{dt} &= \gamma I - \mu R\end{aligned}$$



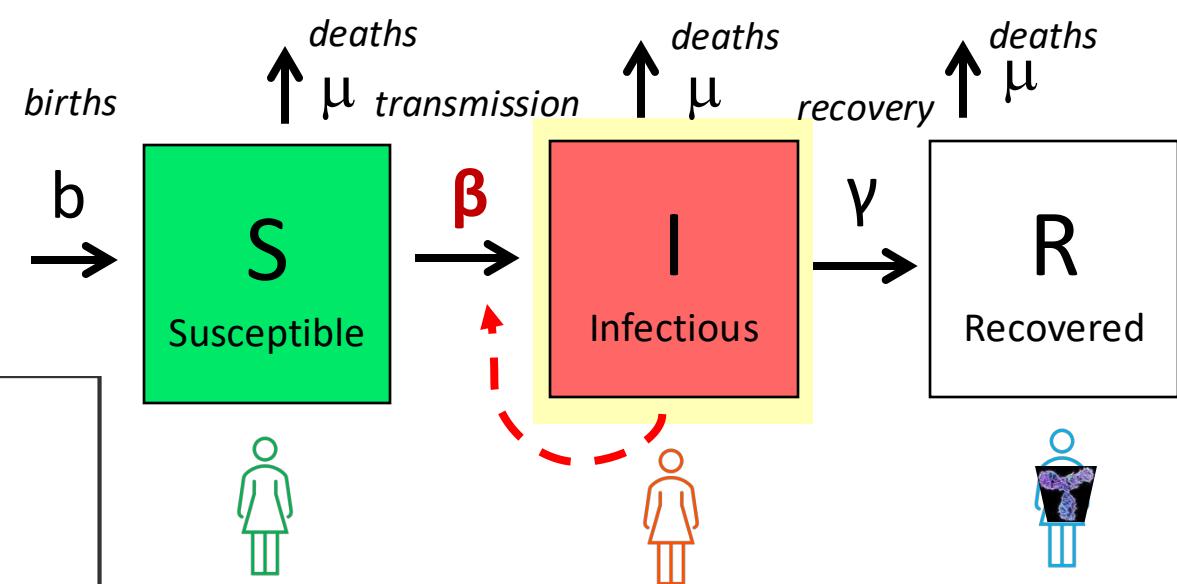
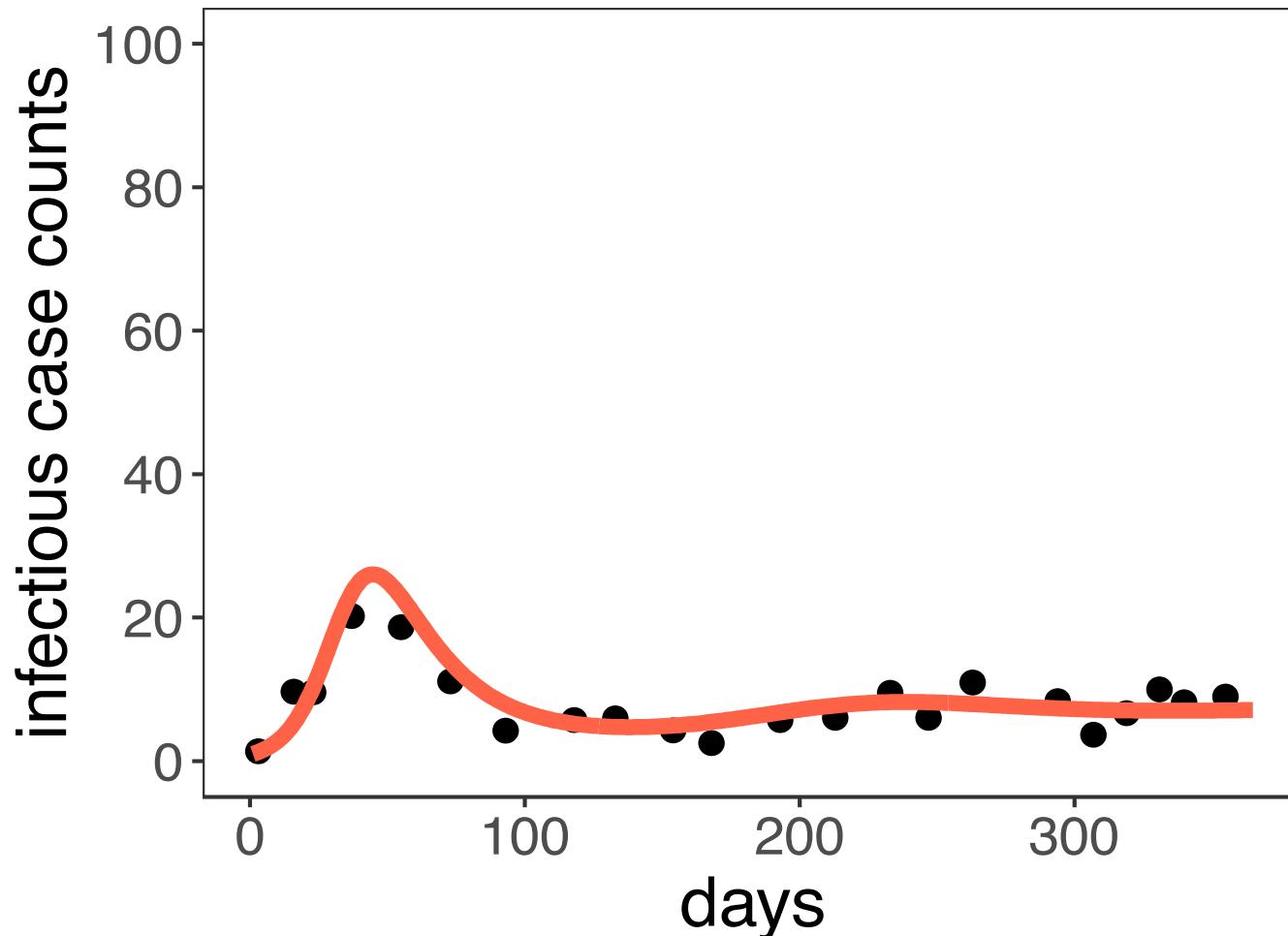
b = birth rate

μ = death rate

β = transmission rate

γ = recovery rate

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



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

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

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

$\approx \lambda$ for a
population model

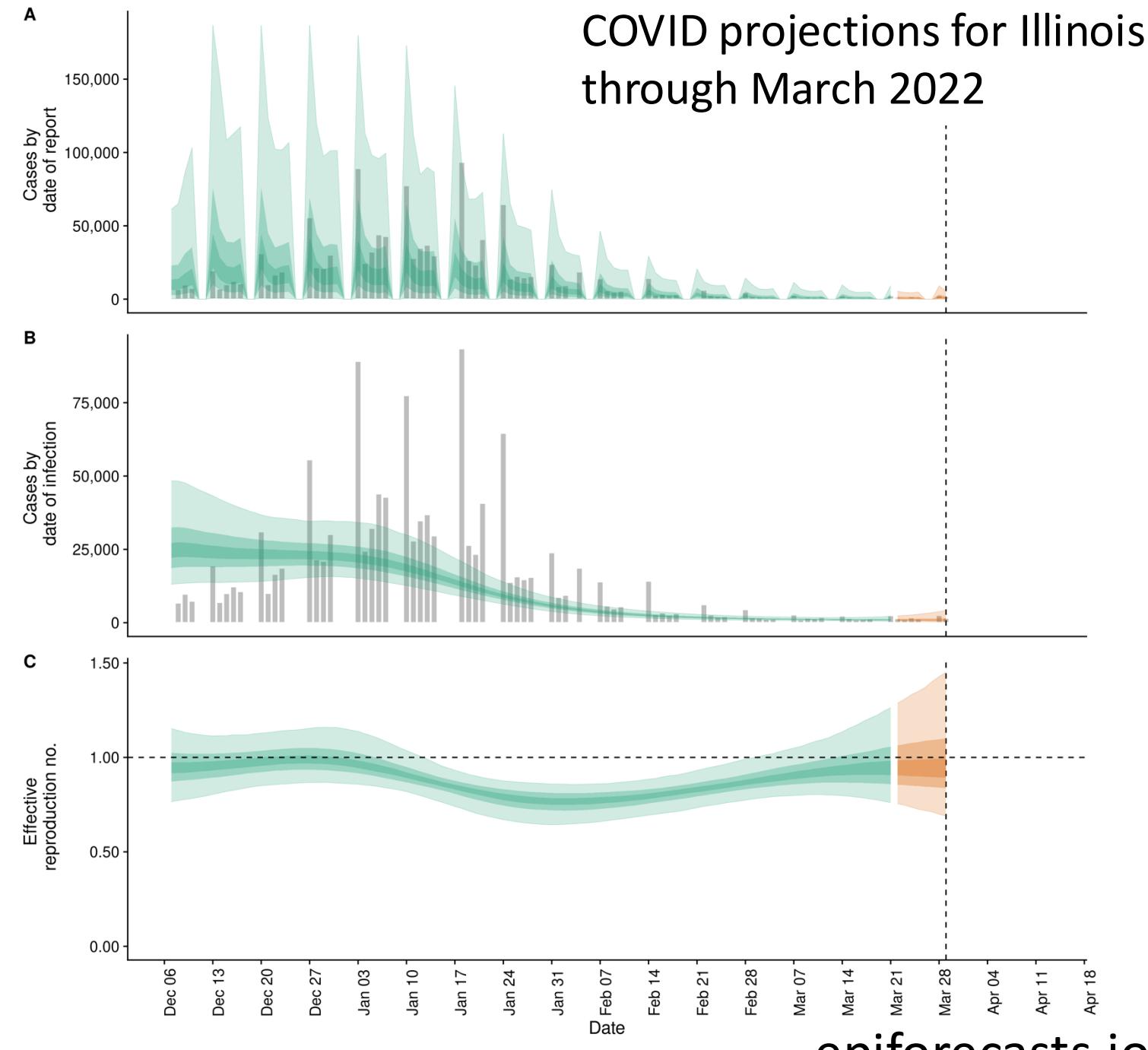
(epidemics spread @ $R_E > 1$ and decline @ $R_E < 1$)

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 $R_0 * \text{proportion susceptible}$

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

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

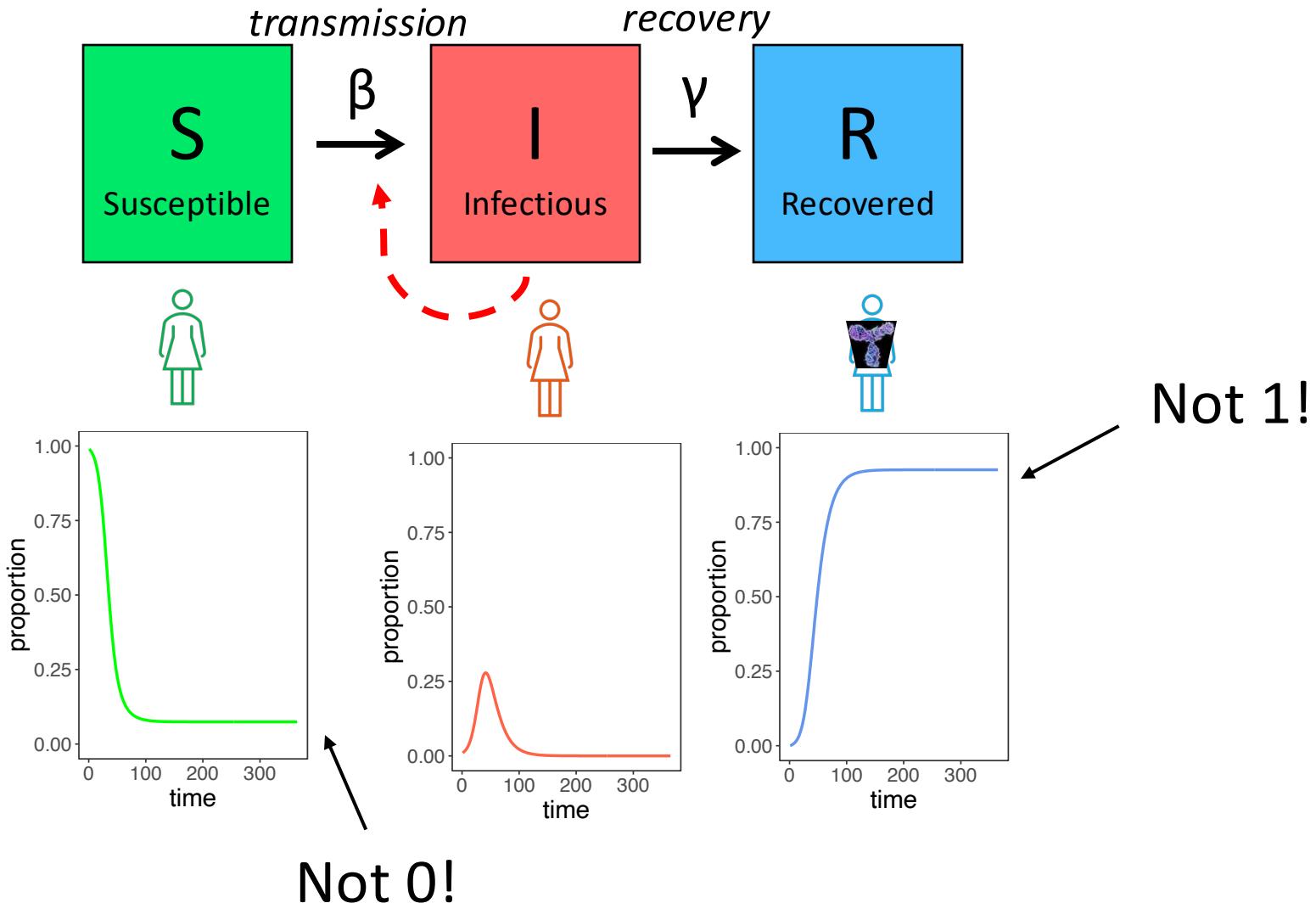


No matter the dynamics, not everyone gets infected before the epidemic ends!

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

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

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



β = transmission rate

γ = recovery rate

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

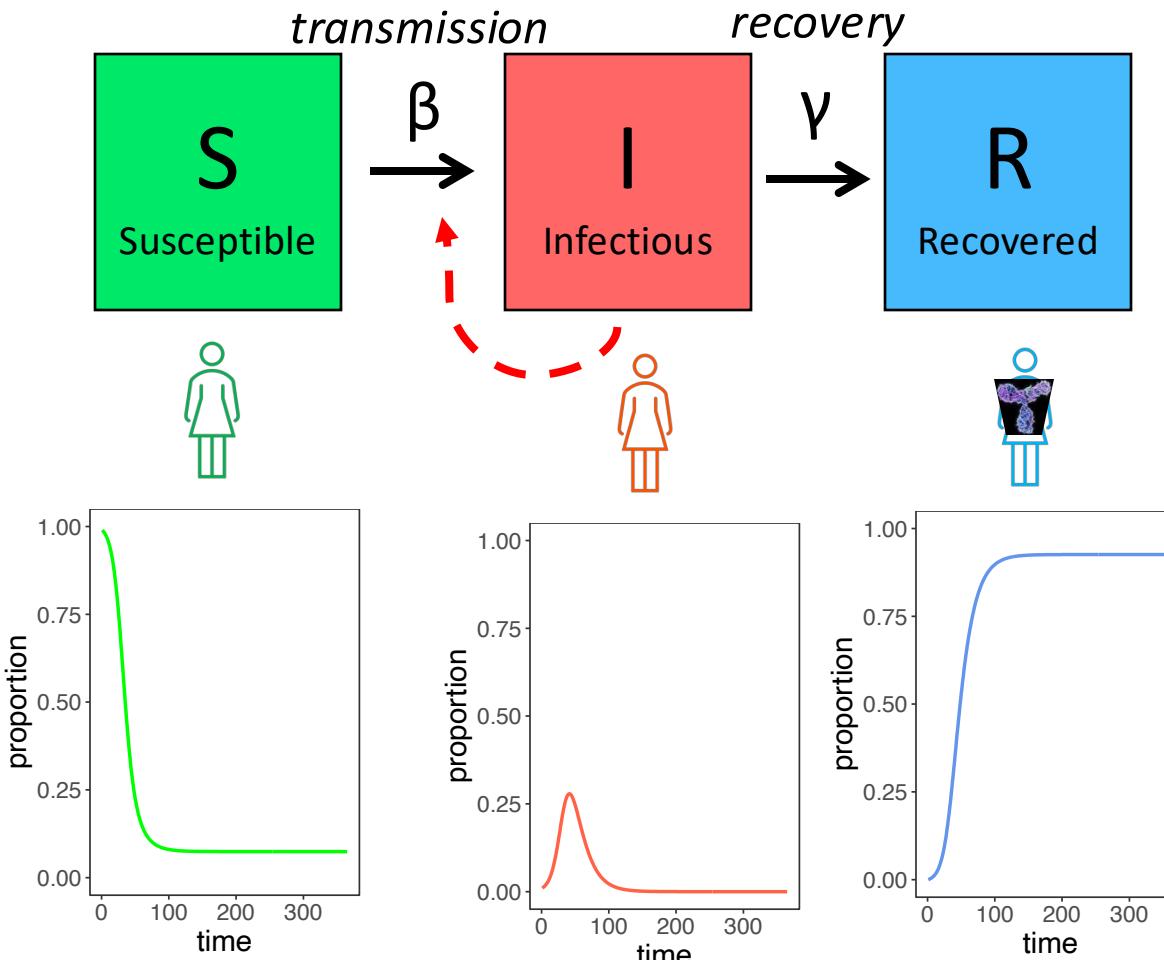
$$\frac{dS}{dt} = -\beta SI$$

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

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

β = transmission rate

γ = recovery rate



- 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 $R_E < 1$

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



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R_0 interventions

- Social distancing
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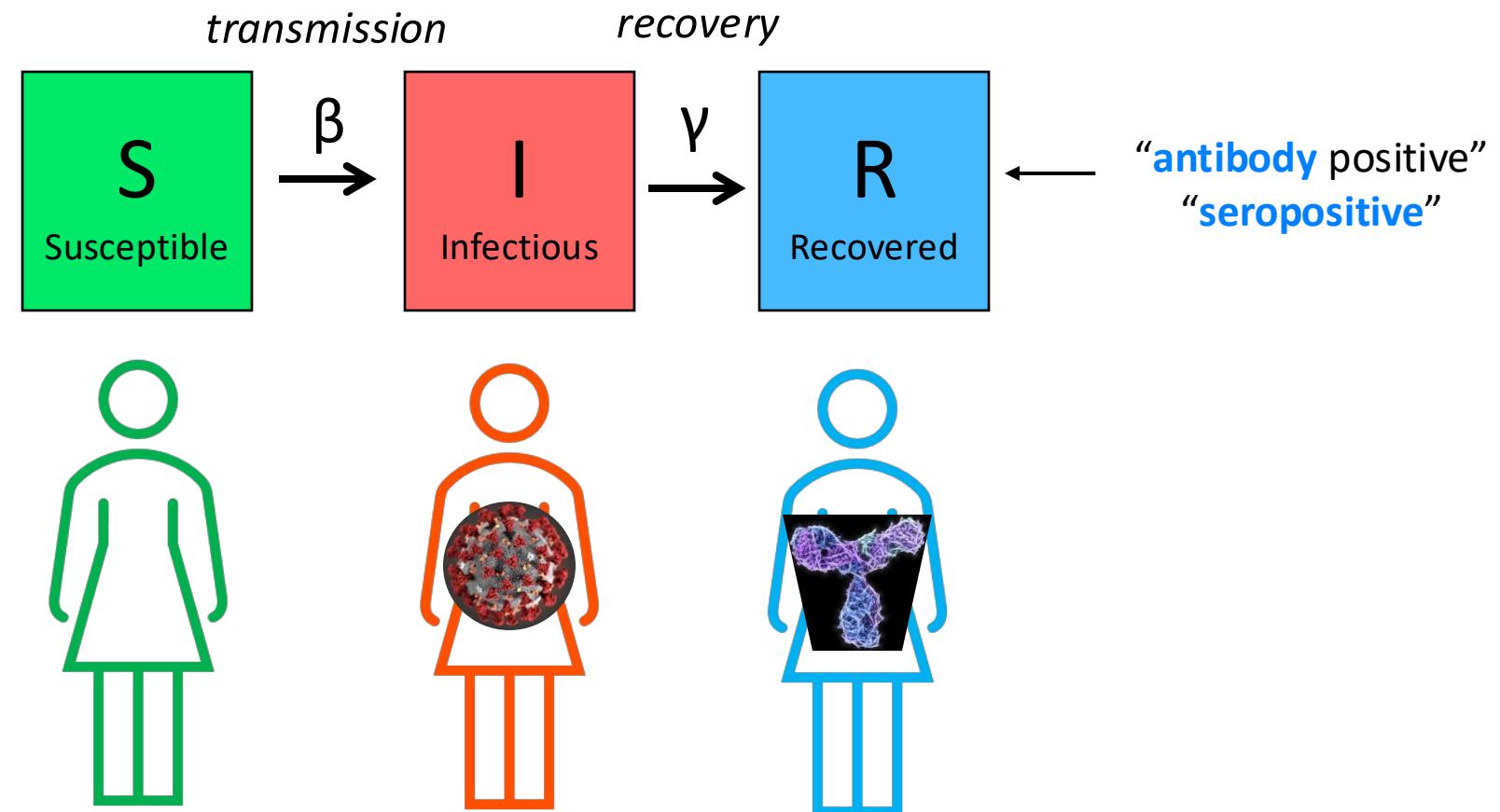
R_E interventions

- Vaccination



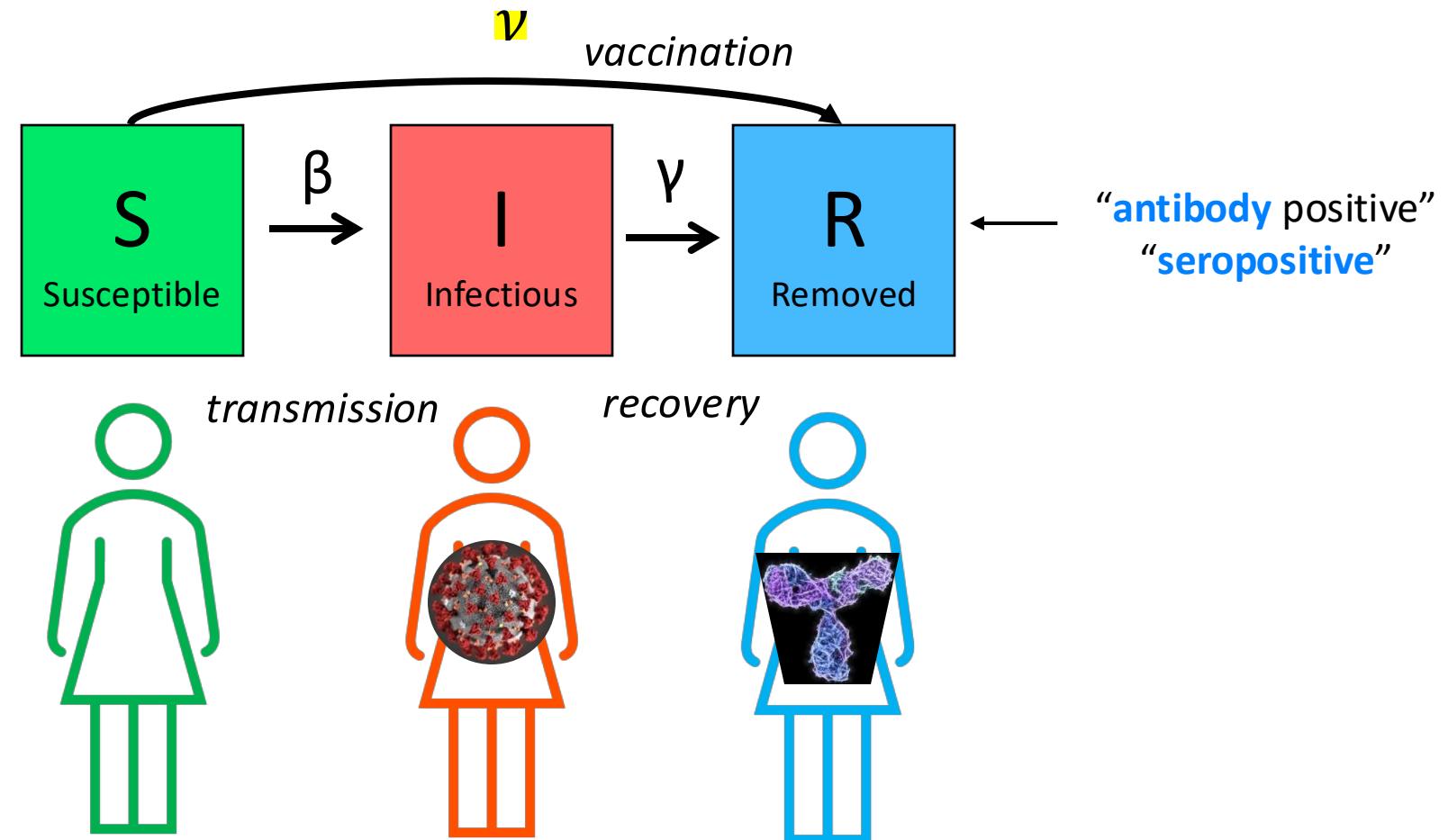
Mathematics of Vaccination

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



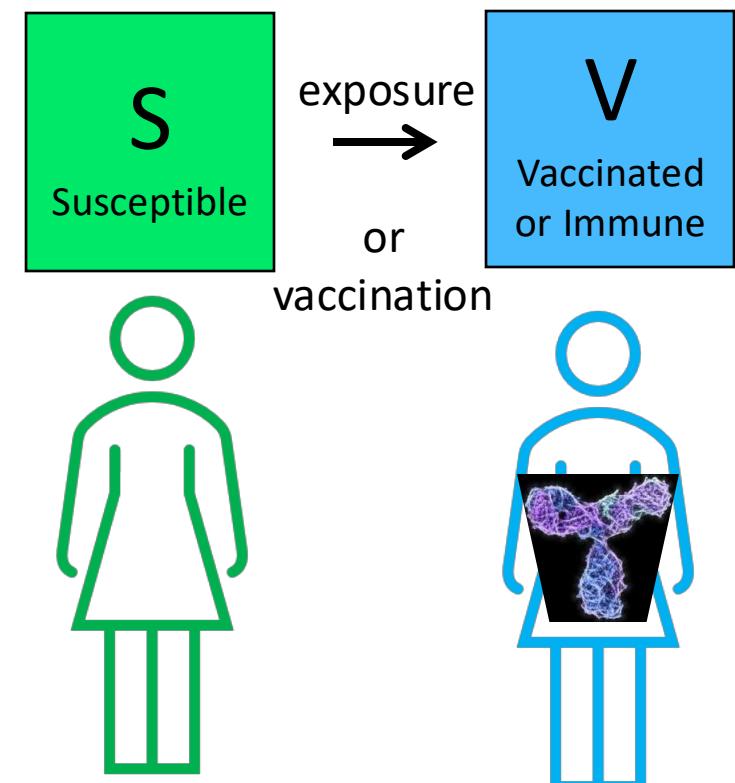
Mathematics of Vaccination

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

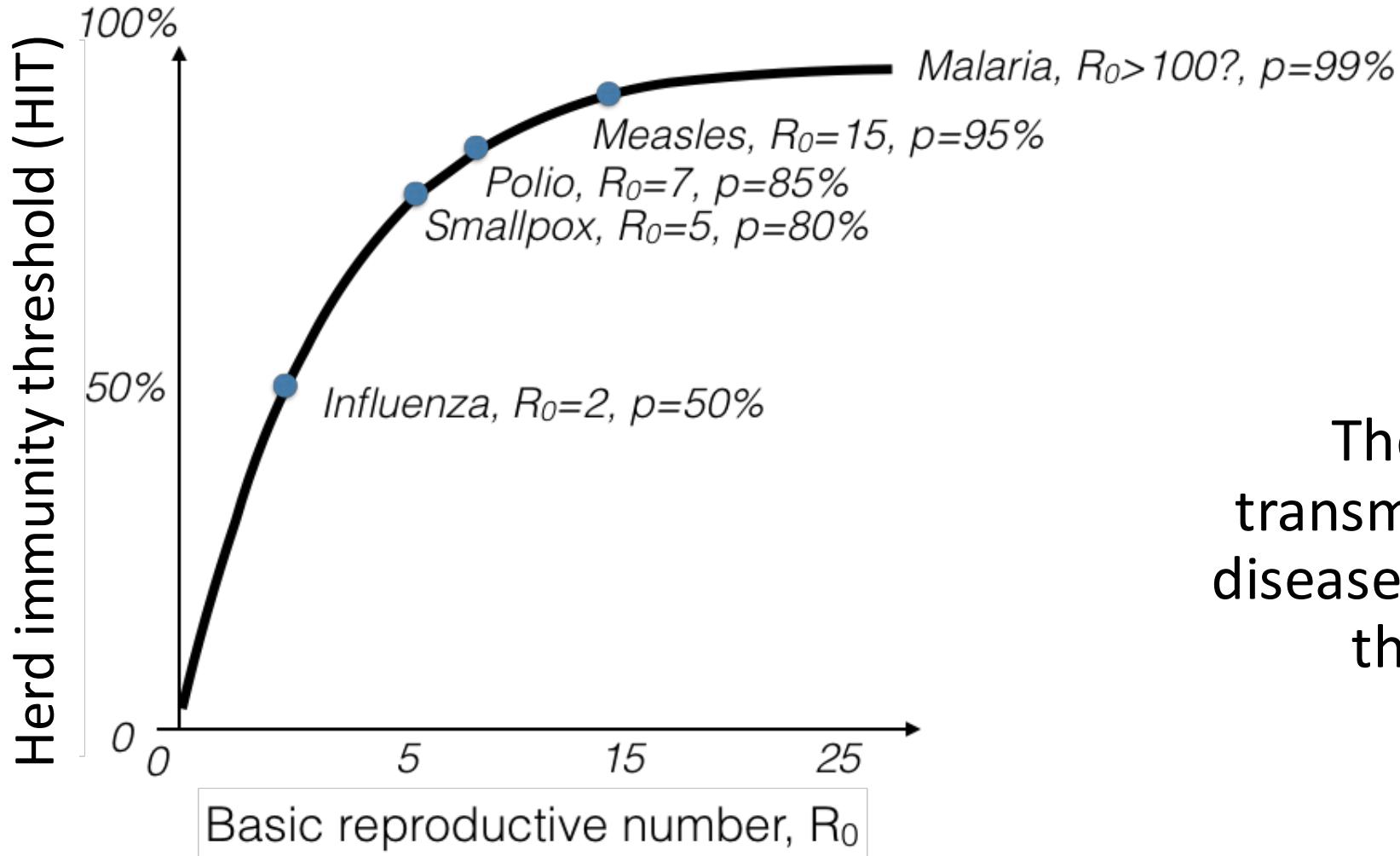


Mathematics of Vaccination

- 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 ($R_E < 1$).

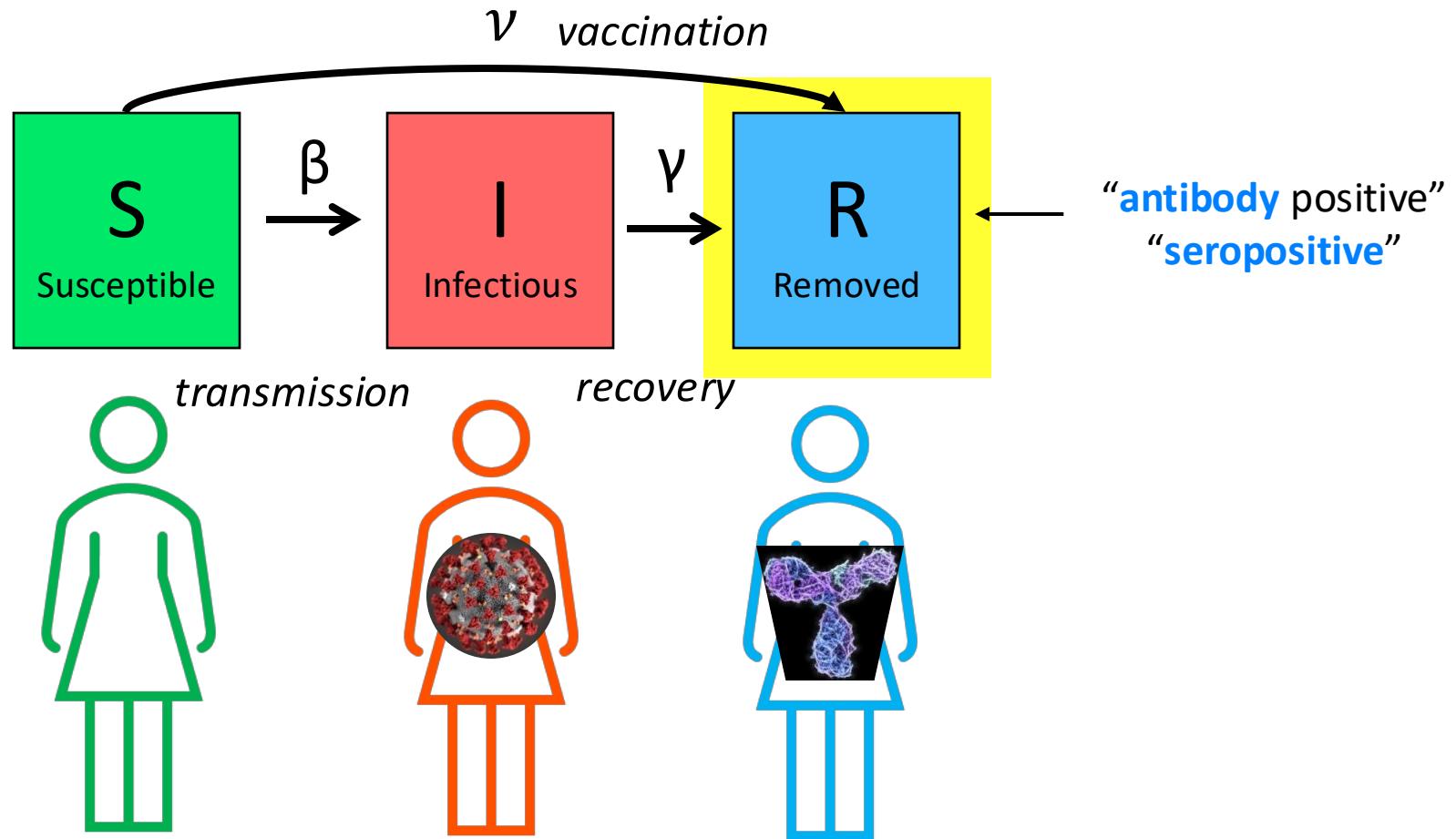


R_0 and the Herd Immunity Threshold



The more
transmissible the
disease, the higher
the HIT!

Vaccination stems from a long history

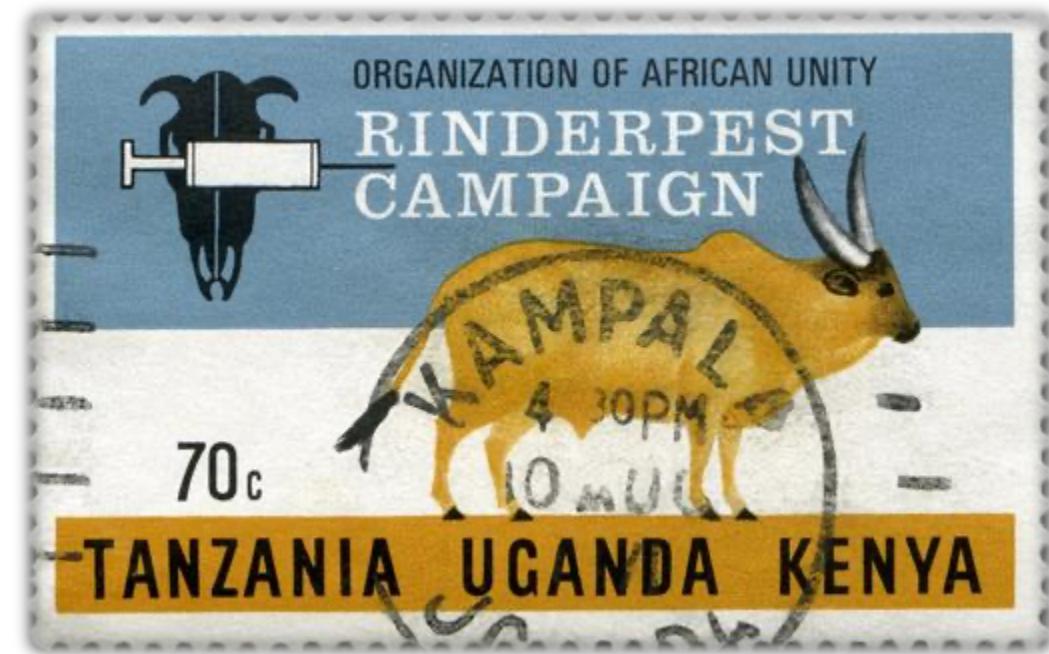


Vaccination stems from a long history

- 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 10th 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



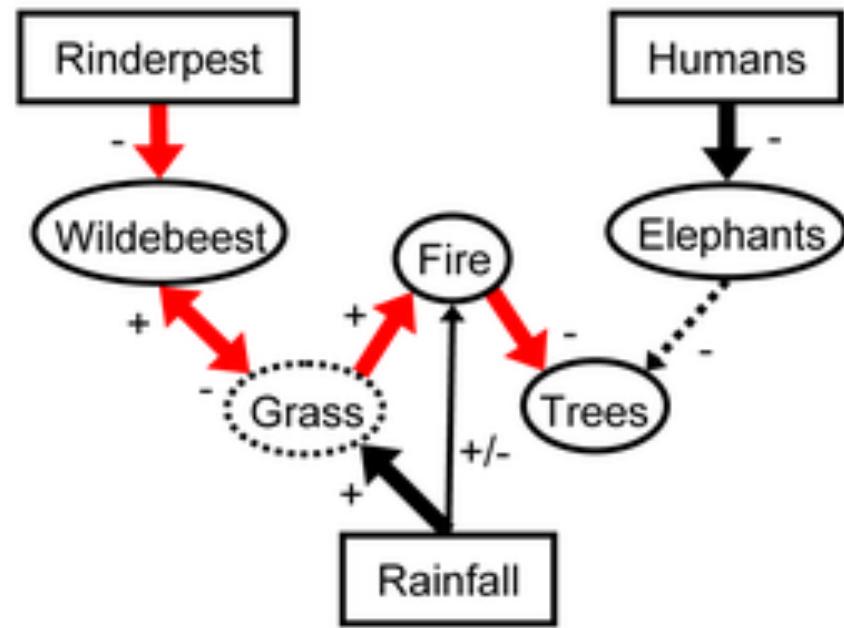
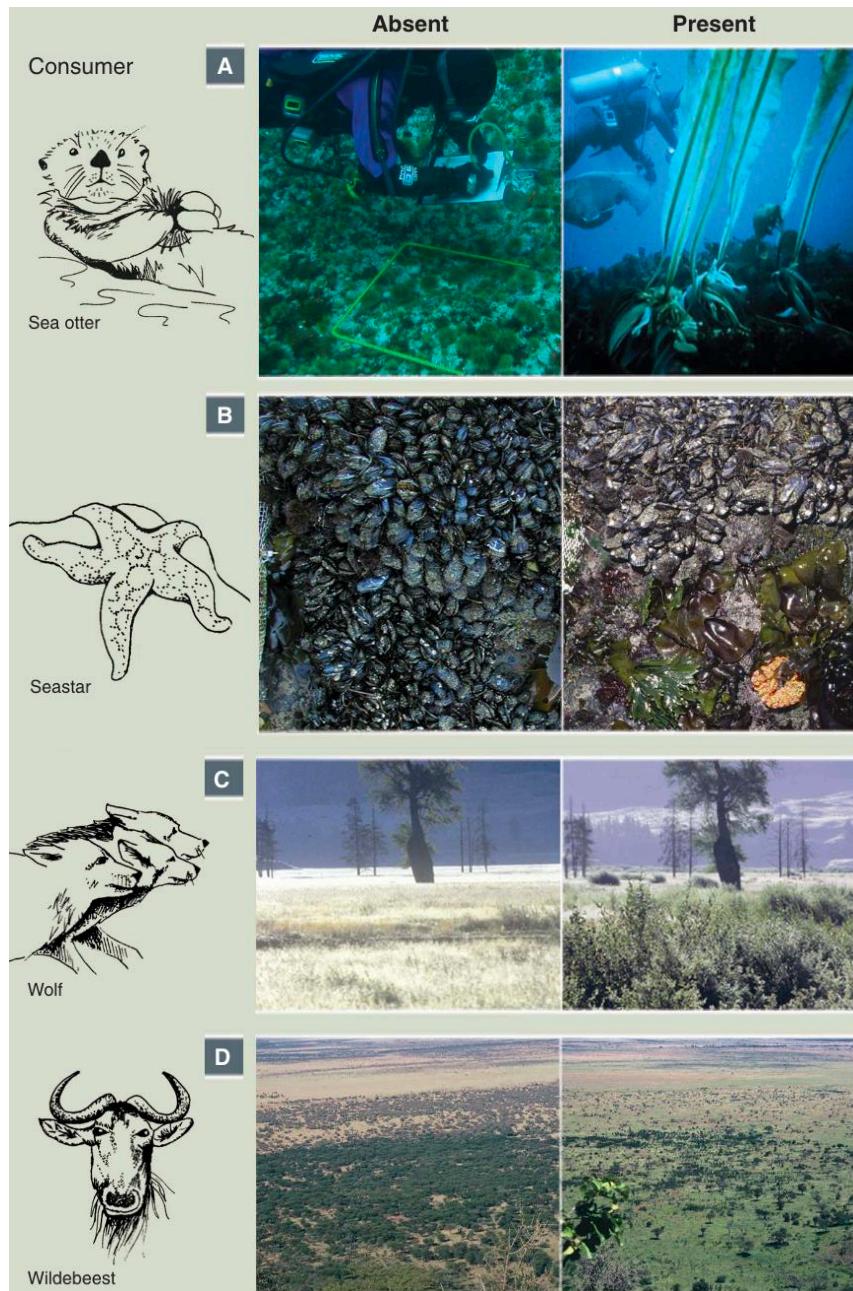
Only two global vaccination success stories



Rinderpest was globally eradicated in 2011, though inoculation efforts date back to the 1700s!

Remember those **trophic cascades**...

Estes et al. 2011. *Science*.



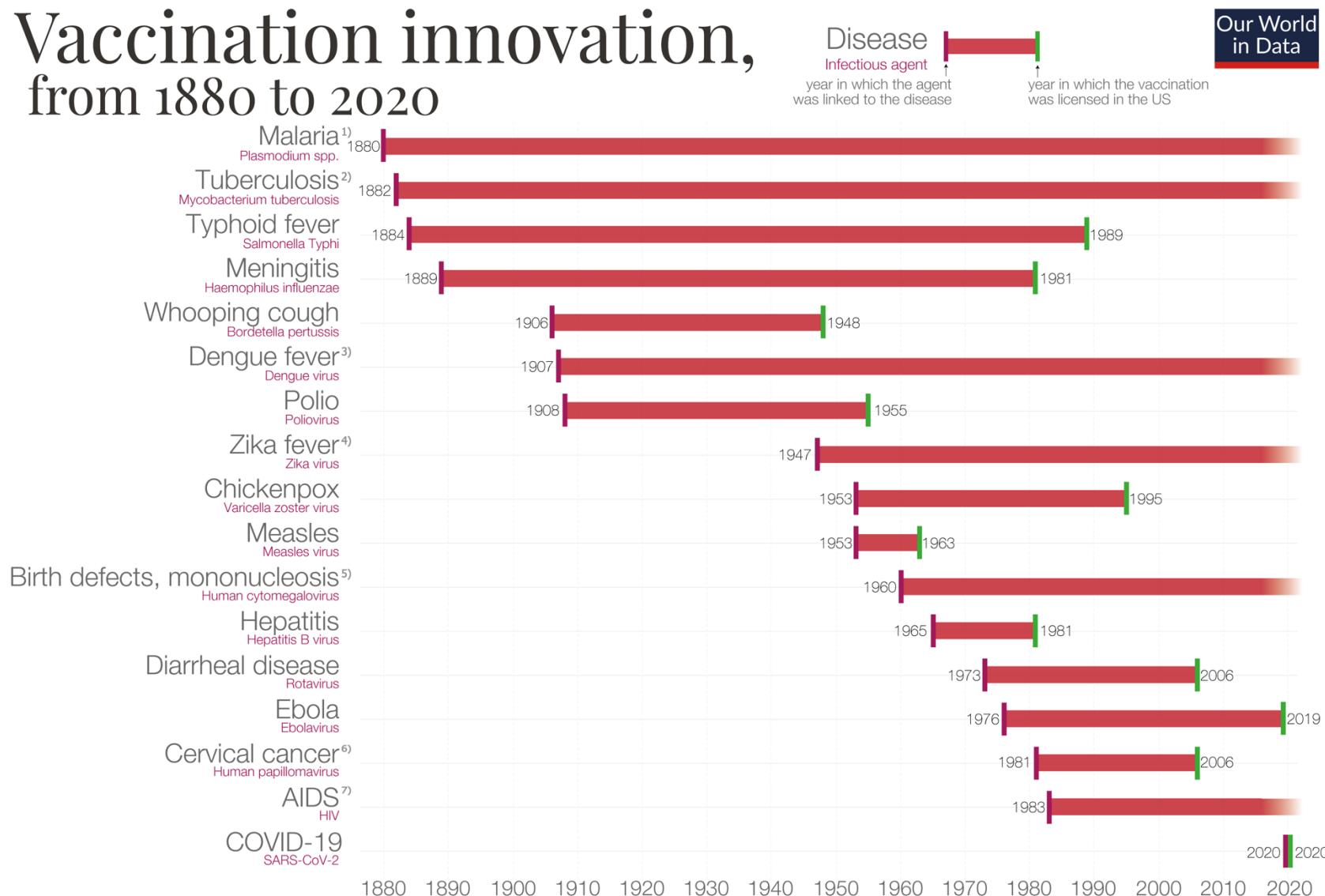
1. ↓ rinderpest
2. ↑ wildebeest
3. ↓ grass
4. ↓ fire
5. ↑ trees

Rinderpest eradication releases wildebeest populations that control savanna, limit fire, and promote tree regrowth

Holdo et al. 2009. *PLoS Biology*

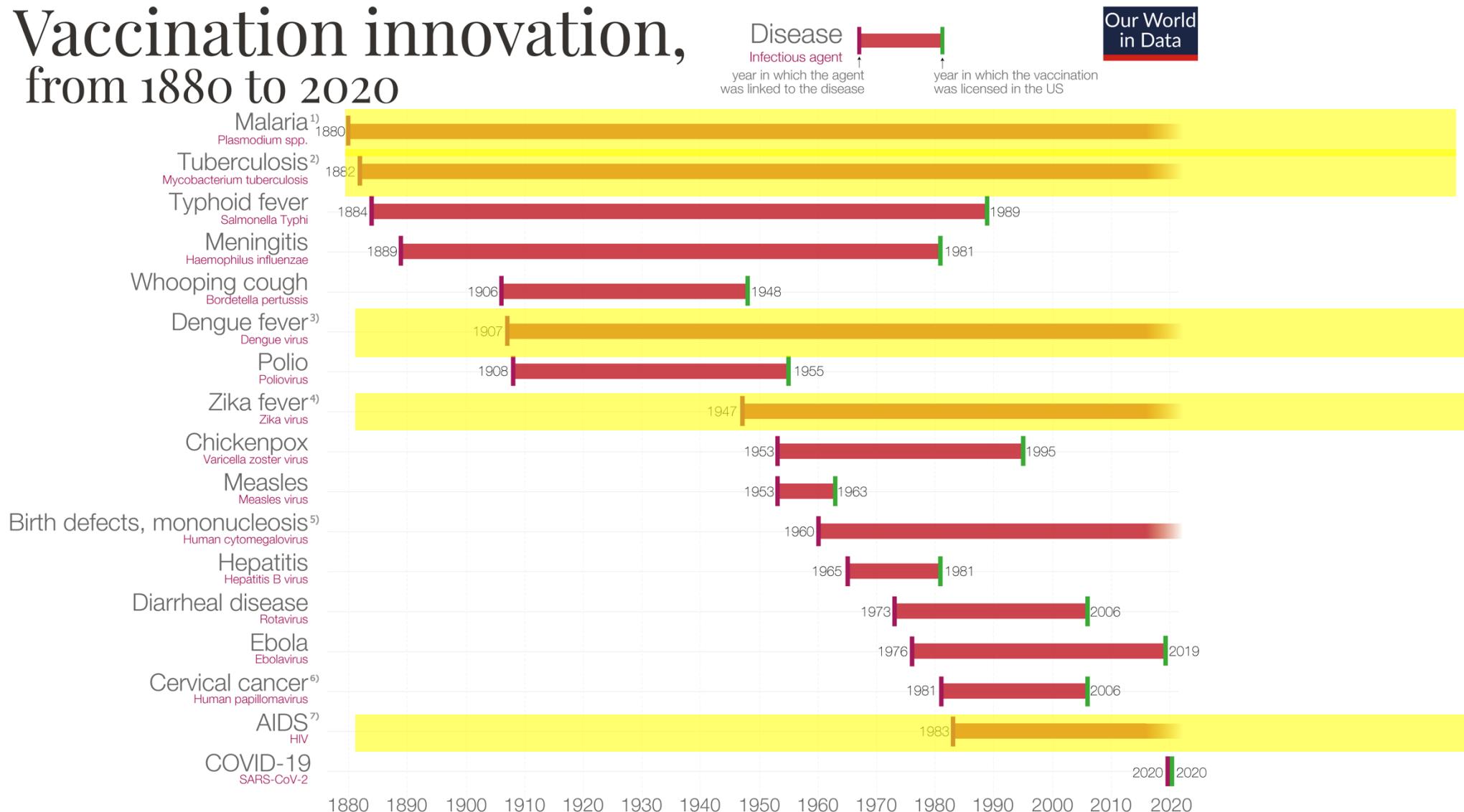
The pace of vaccine development has accelerated drastically

Vaccination innovation, from 1880 to 2020



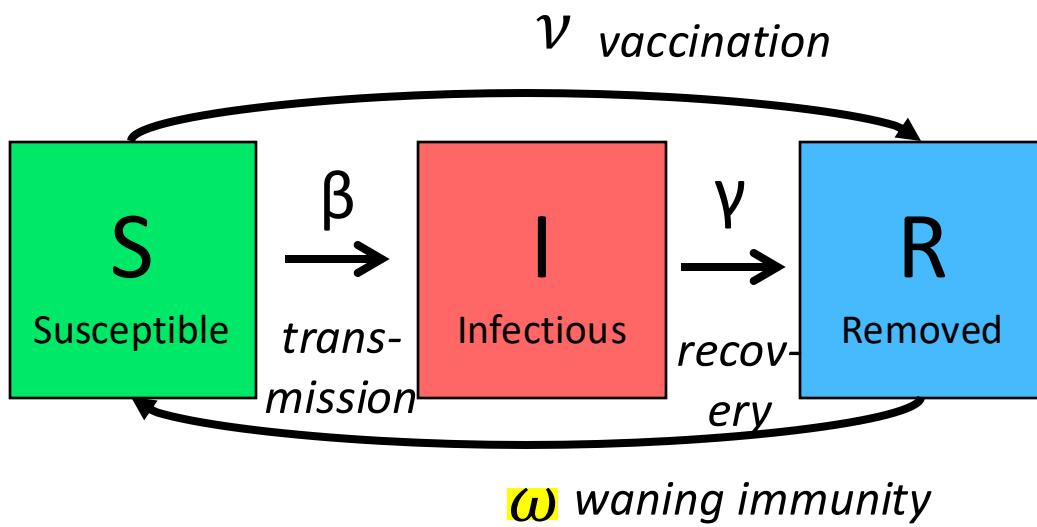
But we still lack vaccines for several important diseases.

Vaccination innovation, from 1880 to 2020



Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens

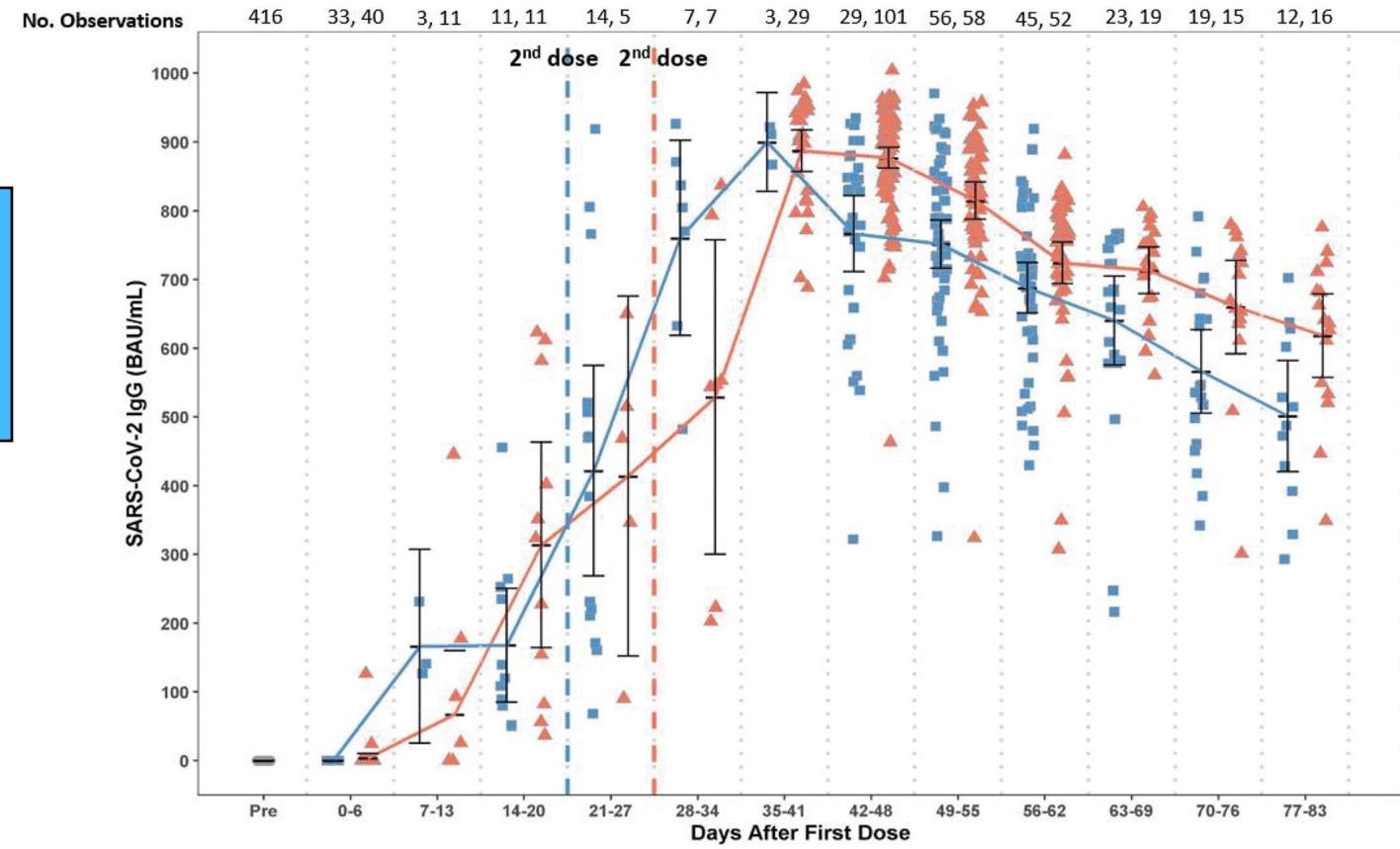


β = transmission rate

γ = recovery rate

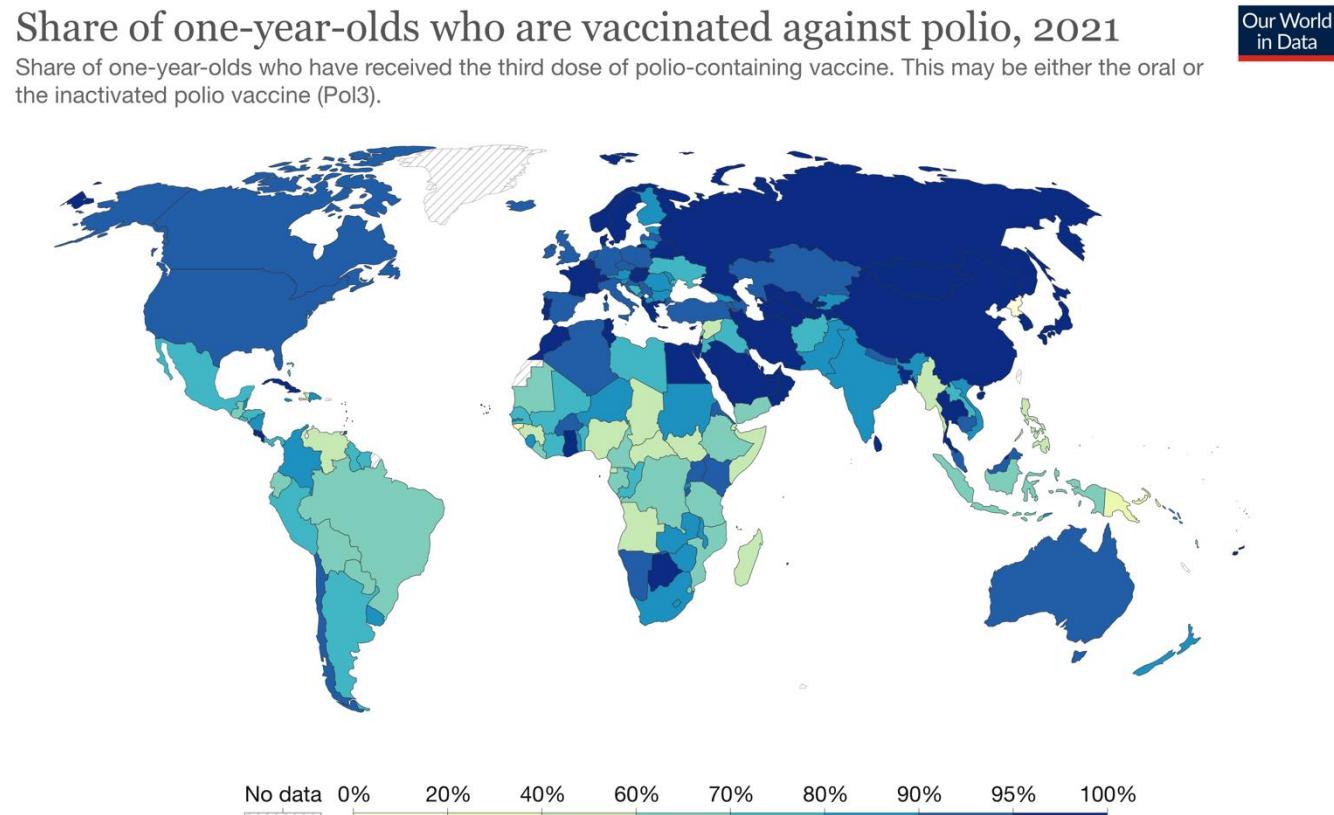
ν = vaccination rate

ω = 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/ • CC BY

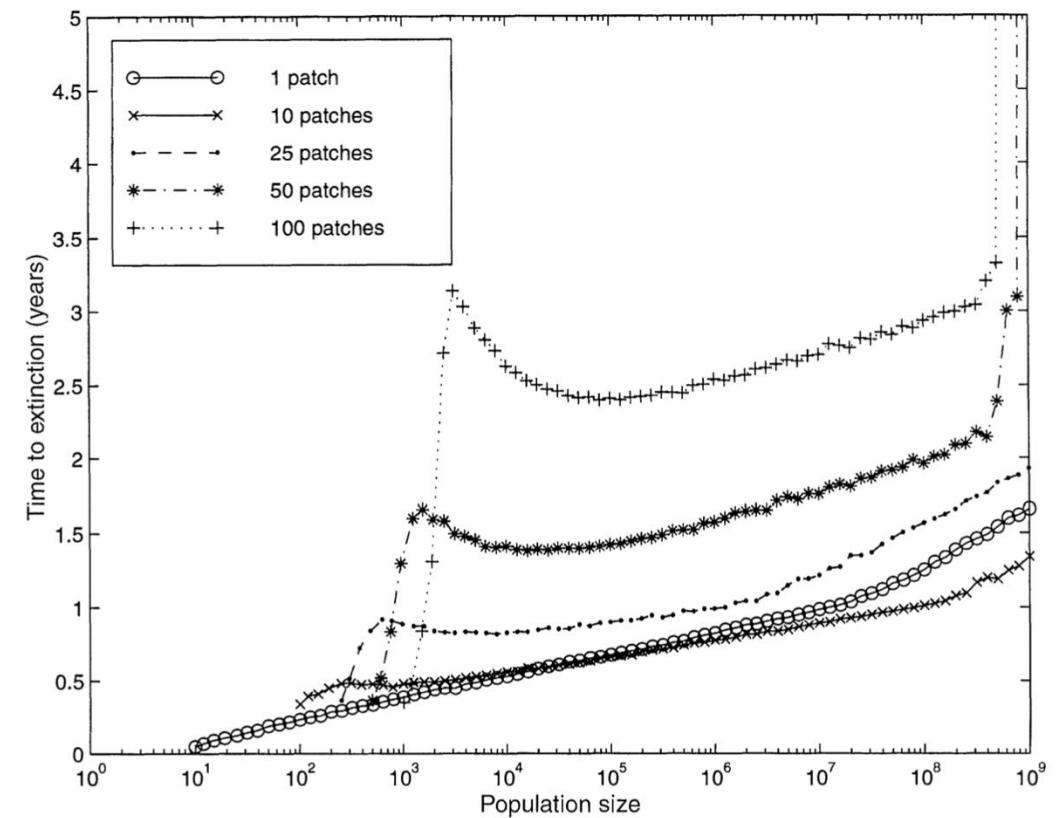
Challenges to Vaccination

- 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

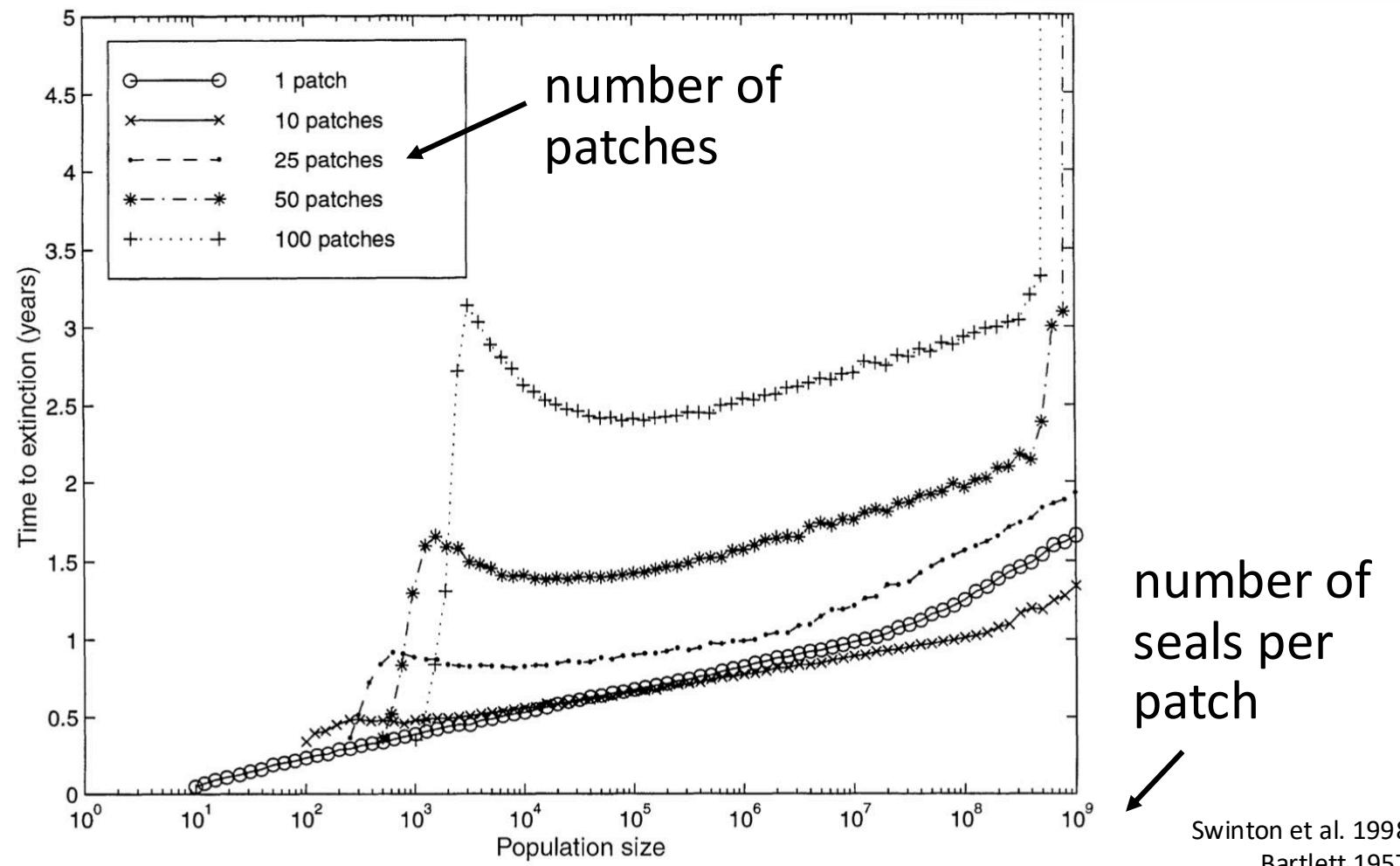
- Spatial structure (**metapopulation rescue**)



time until all patches go extinct (remember metapopulations!)

All else equal, **increasing the number of patches** will slow the time to extinction.

...though remember **source-sink** dynamics!



Population Biology

Conservation Biology

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

MVP = the minimum number of individuals in a population needed to sustain the population 1000 years into the future



Disease Ecology

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

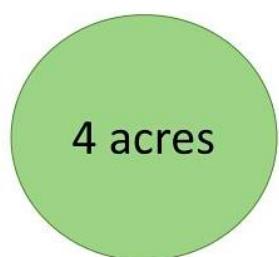
CCS = the minimum number of hosts needed to sustain endemic transmission of a pathogen indefinitely into the future

Population Biology

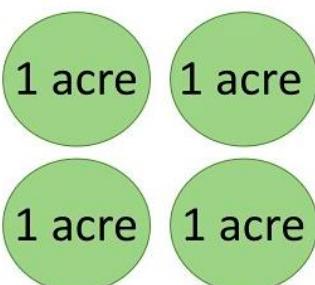
Conservation Biology

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

Single Large

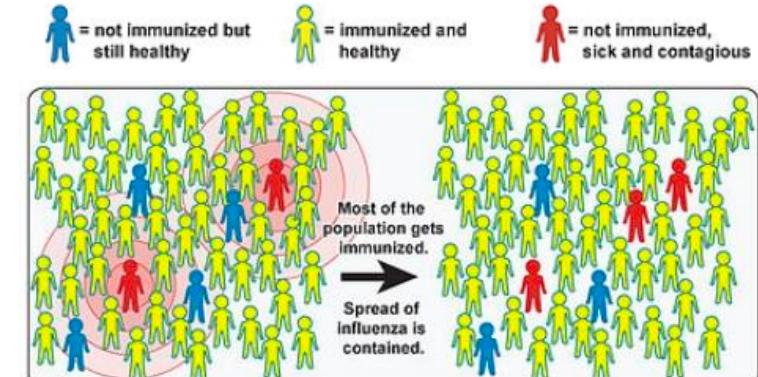


Several Small



Disease Ecology

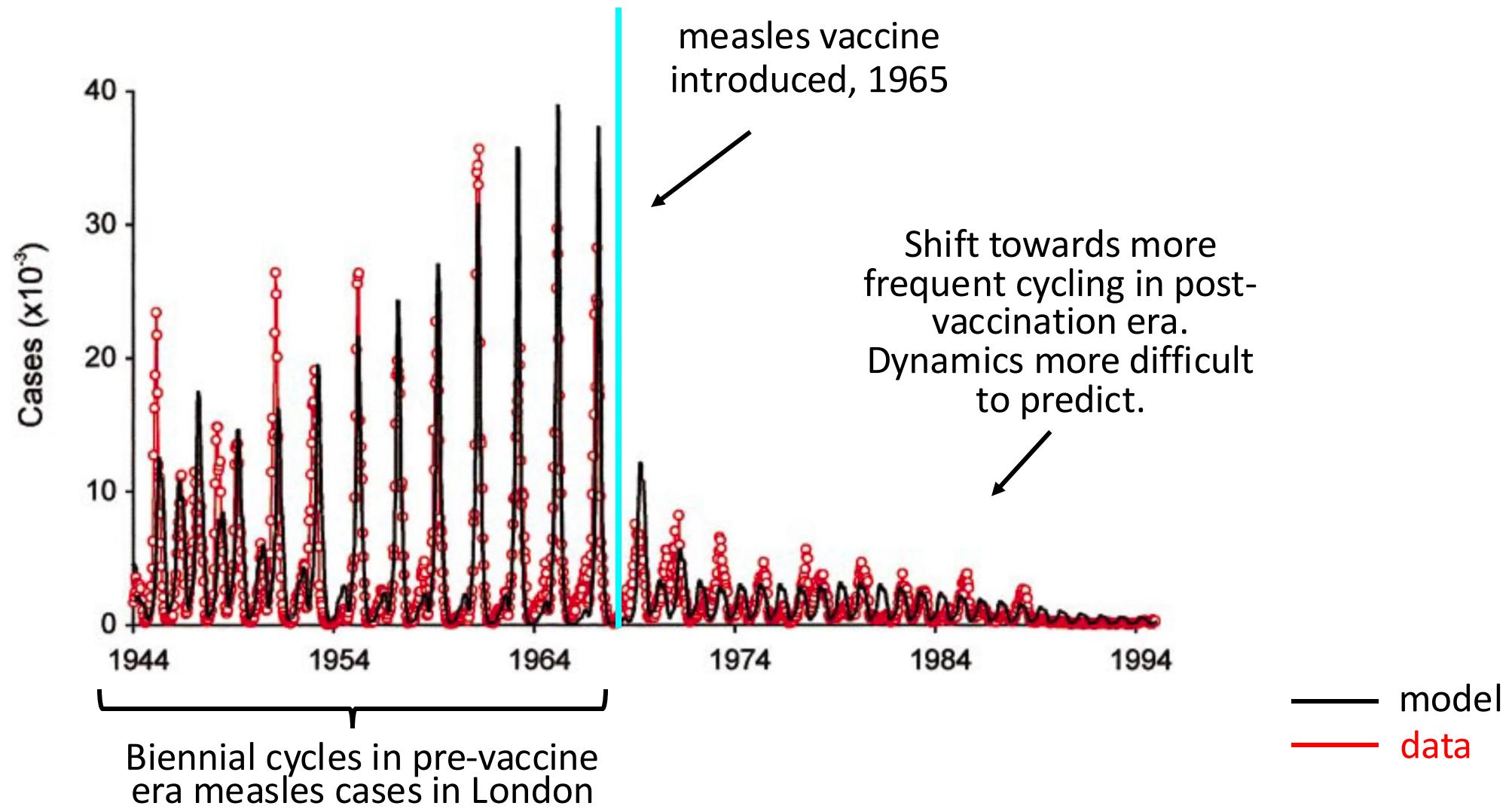
- Goal:
 - protect **populations** from disease via pathogen **extinction**
- Concept:
 - **Critical Community Size** (CCS)
- Approach:
 - sanitation
 - **vaccination**



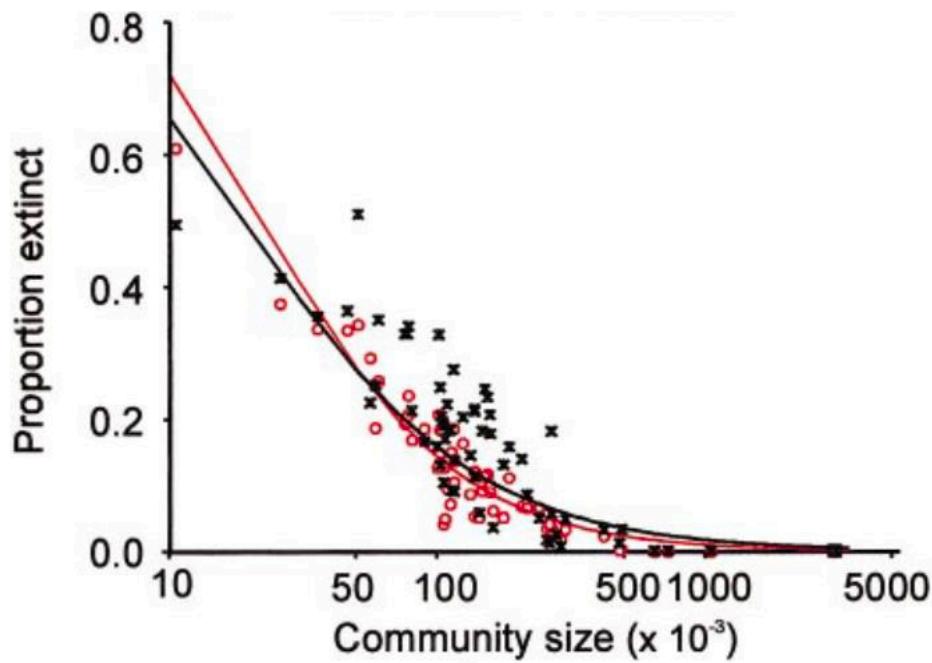
Challenges to Vaccination

- 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.



CCS is the **minimum number of hosts** needed to sustain **endemic transmission** of a pathogen indefinitely into the future.

