Fundamentals of Ecology

Week 8, Ecology Lecture 5

Cara Brook

February 21, 2023

Let’s recap a bit!

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Interspecies Dynamics

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- We can determine the stability of the system using vector addition on the nullcline planes.

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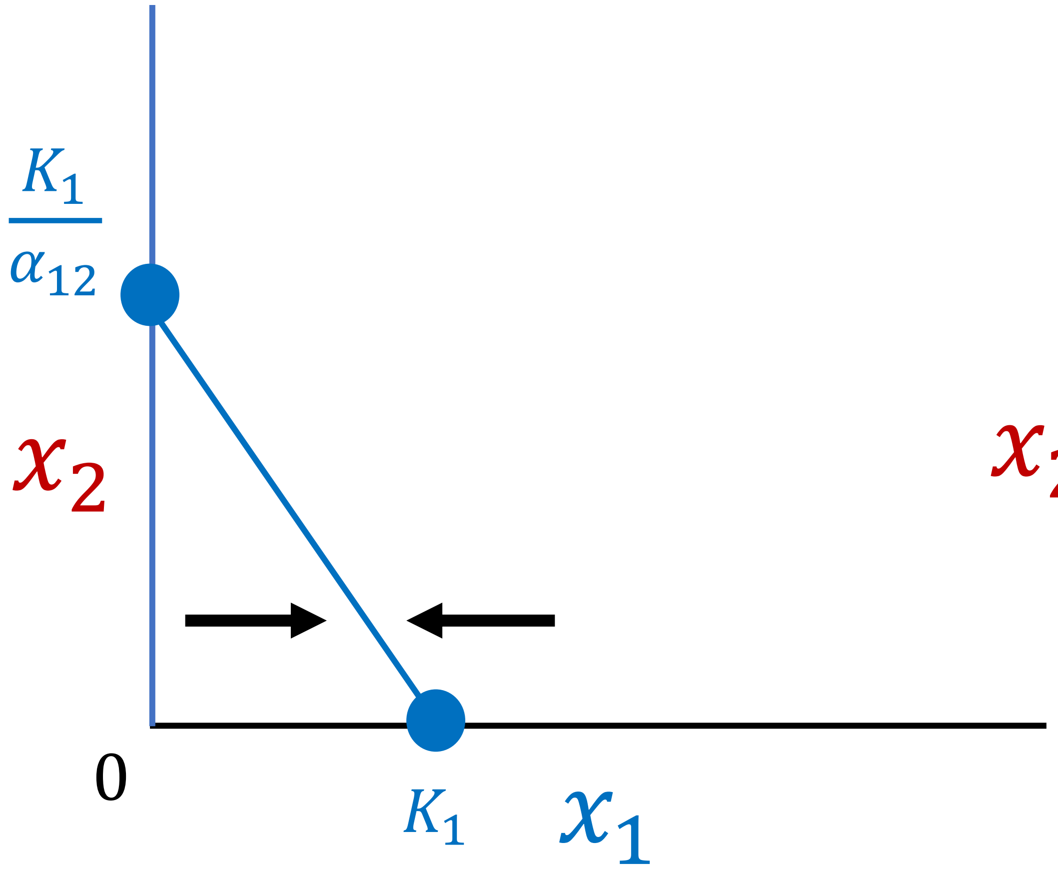
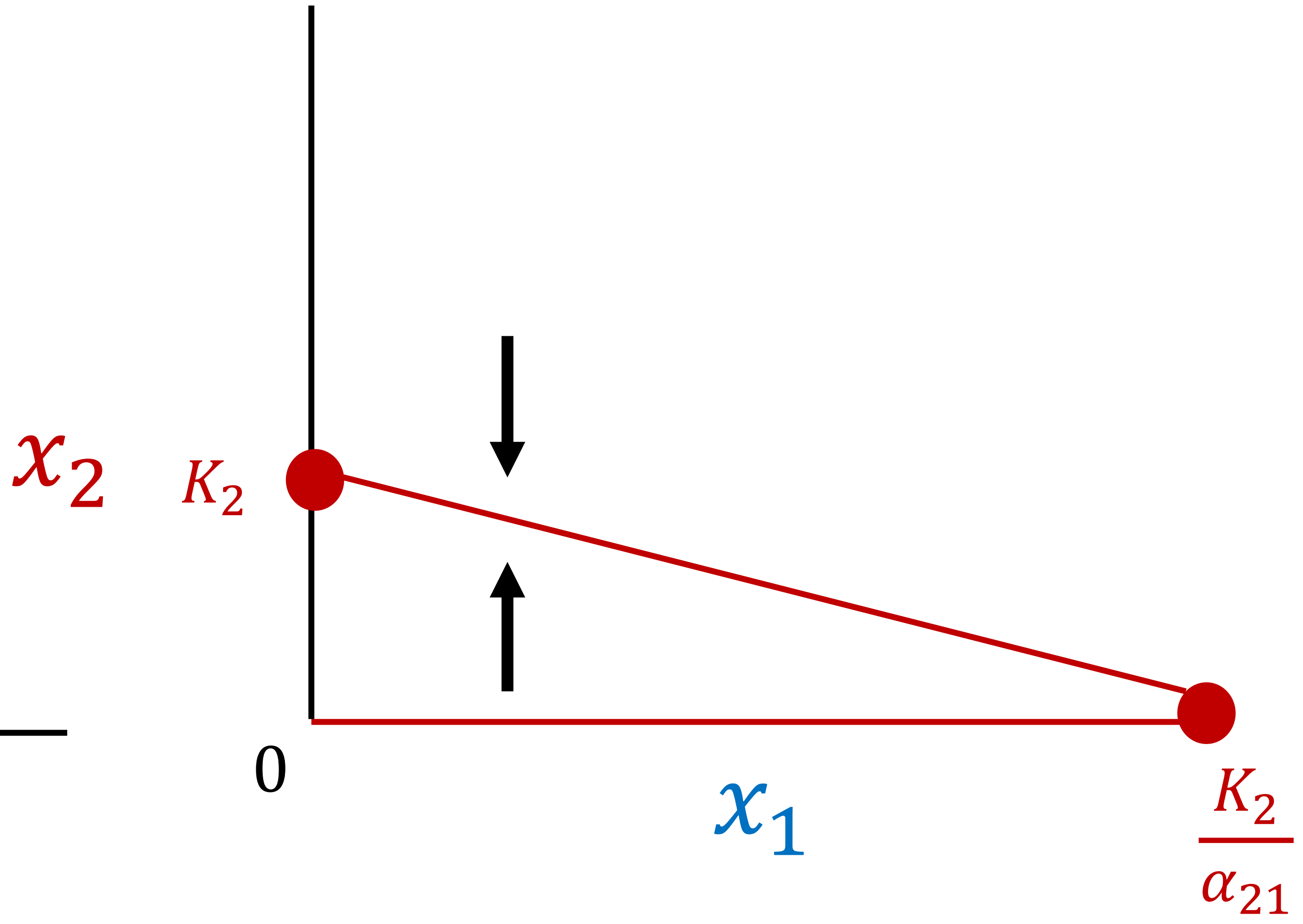
Interspecies Dynamics





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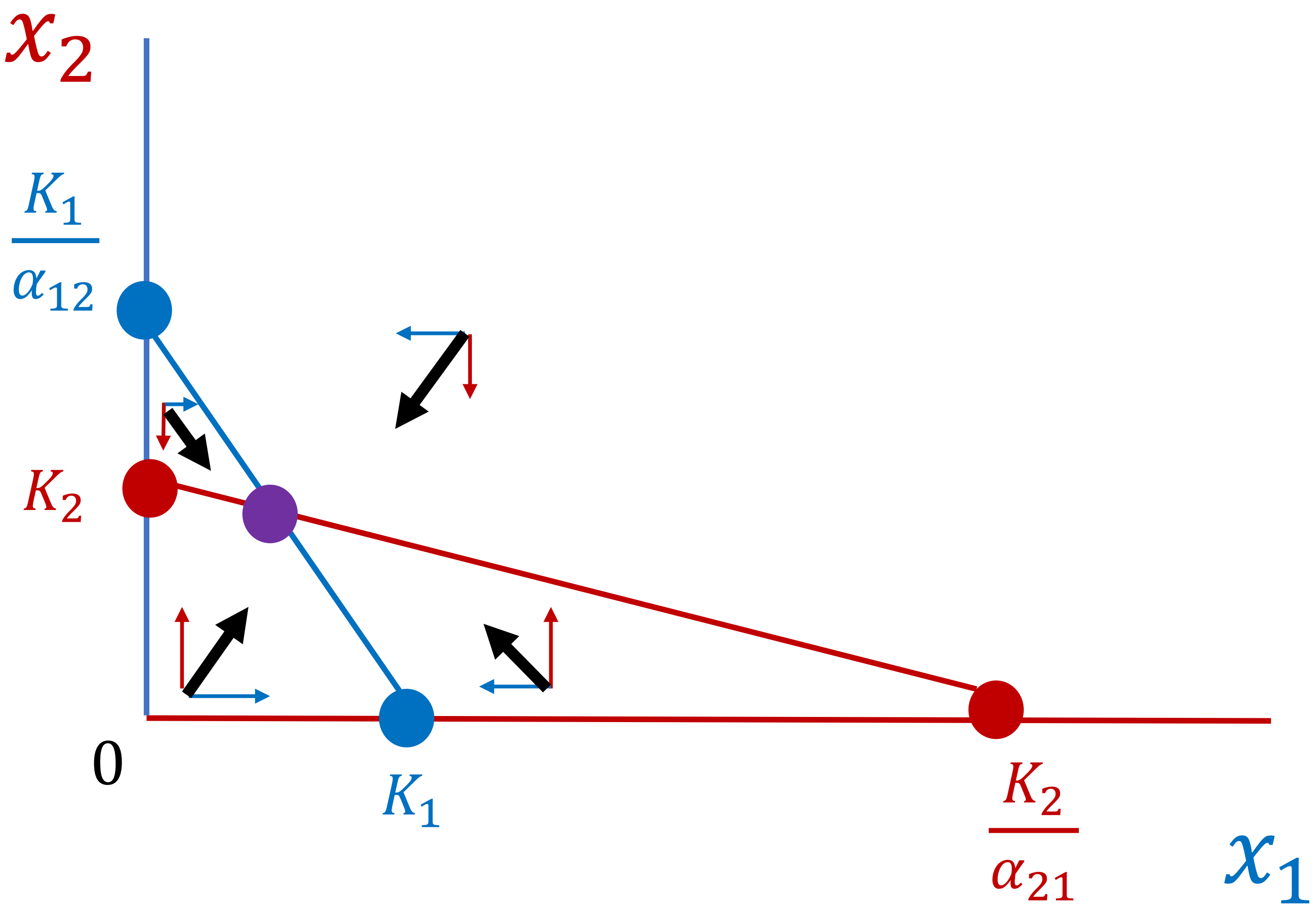

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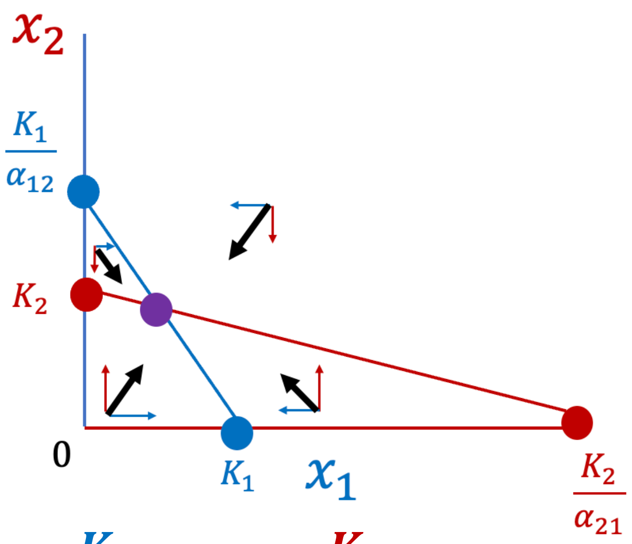
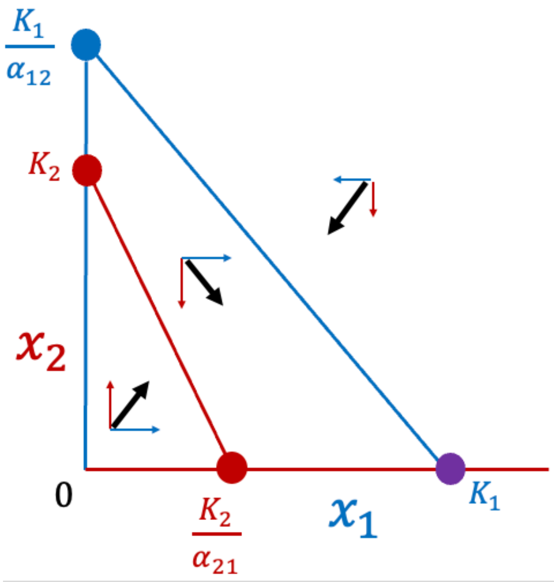
Case 1: Stable coexistence



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

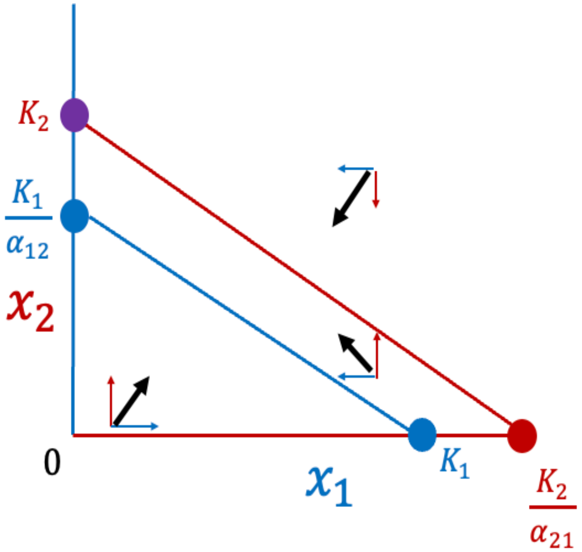
$$(\alpha_{12} * \alpha_{21} < 1)$$

Case 2: Spp. 1 wins



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

Case 3: Spp. 2 wins



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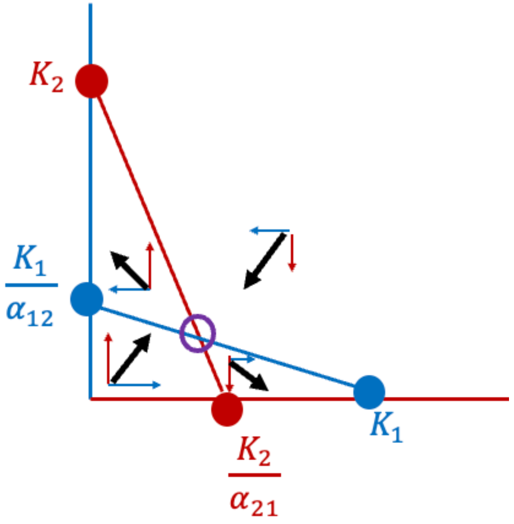
Case 4: Precedence



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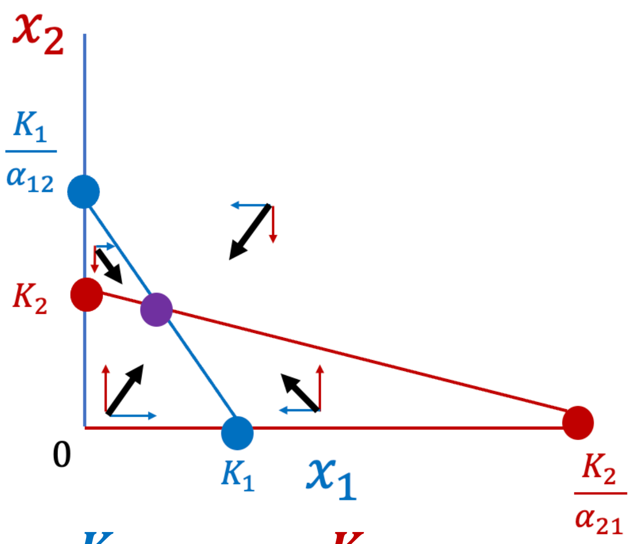

$$\frac{K_1}{\alpha_{12}} > K_2 \ \& \ \frac{K_2}{\alpha_{21}} > K_1$$

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

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

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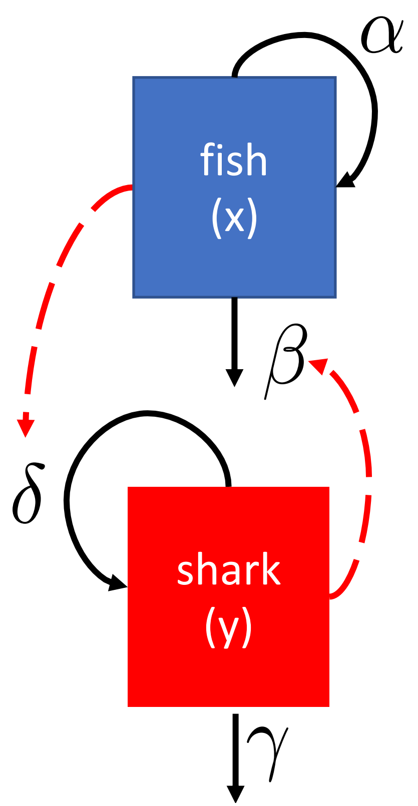
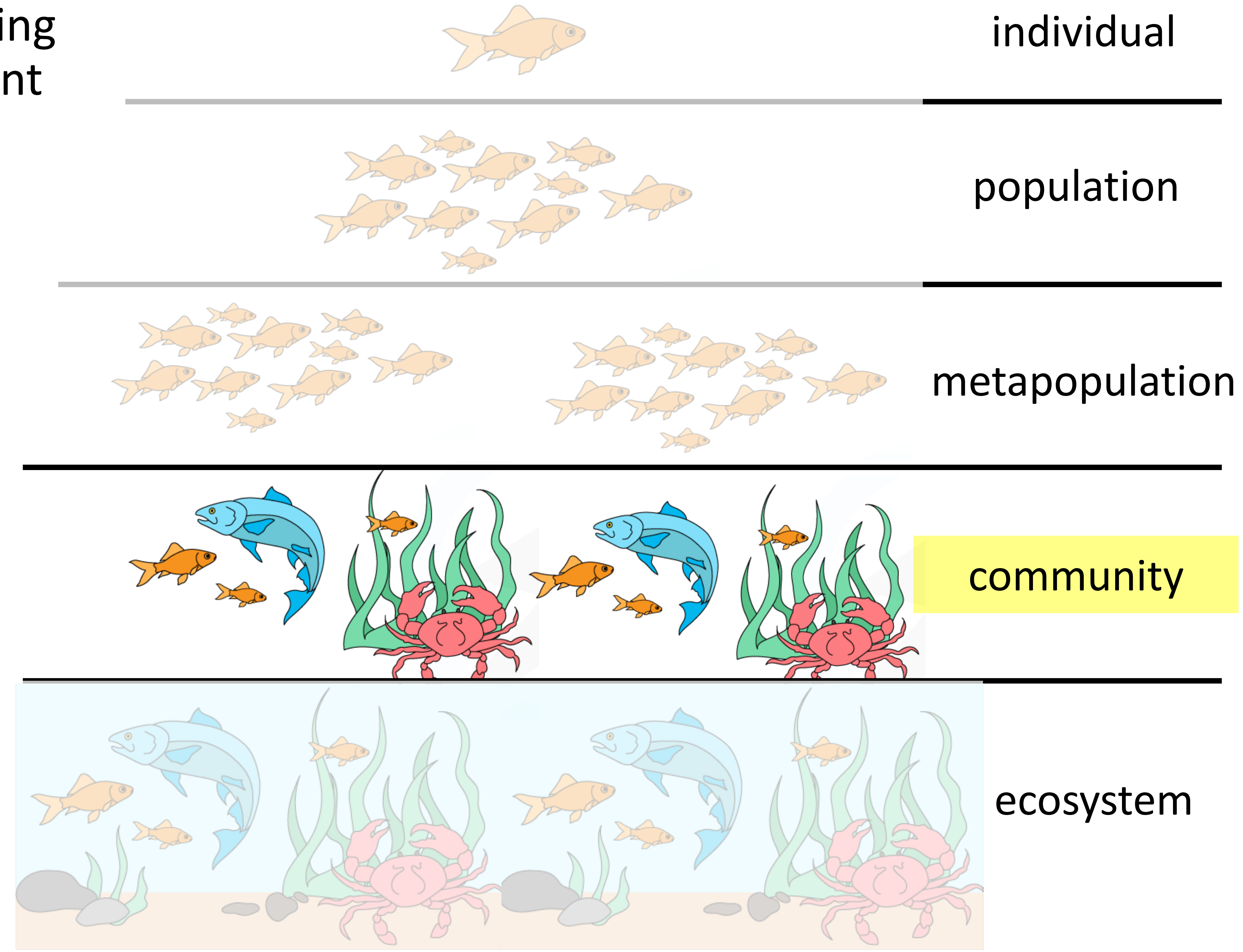
- Character displacement is the evolutionary process by which species occupying similar habitats accentuate differences to avoid competition.
- Consumer resource models developed by MacArthur and Tilman offered a mechanistic explanation of how species compete for specific resources.
- Tilman showed how if multiple species compete for the same limiting resource, the species with the lowest equilibrium resource level (e.g. the species able to live on the lowest quantity of resource) would win.

Community = interacting populations of different species



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



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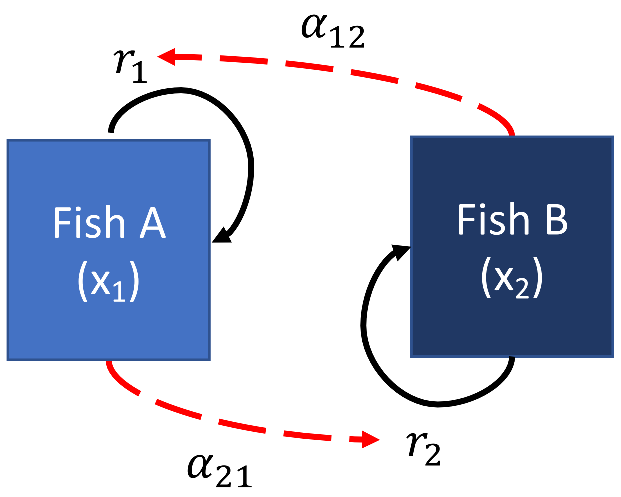

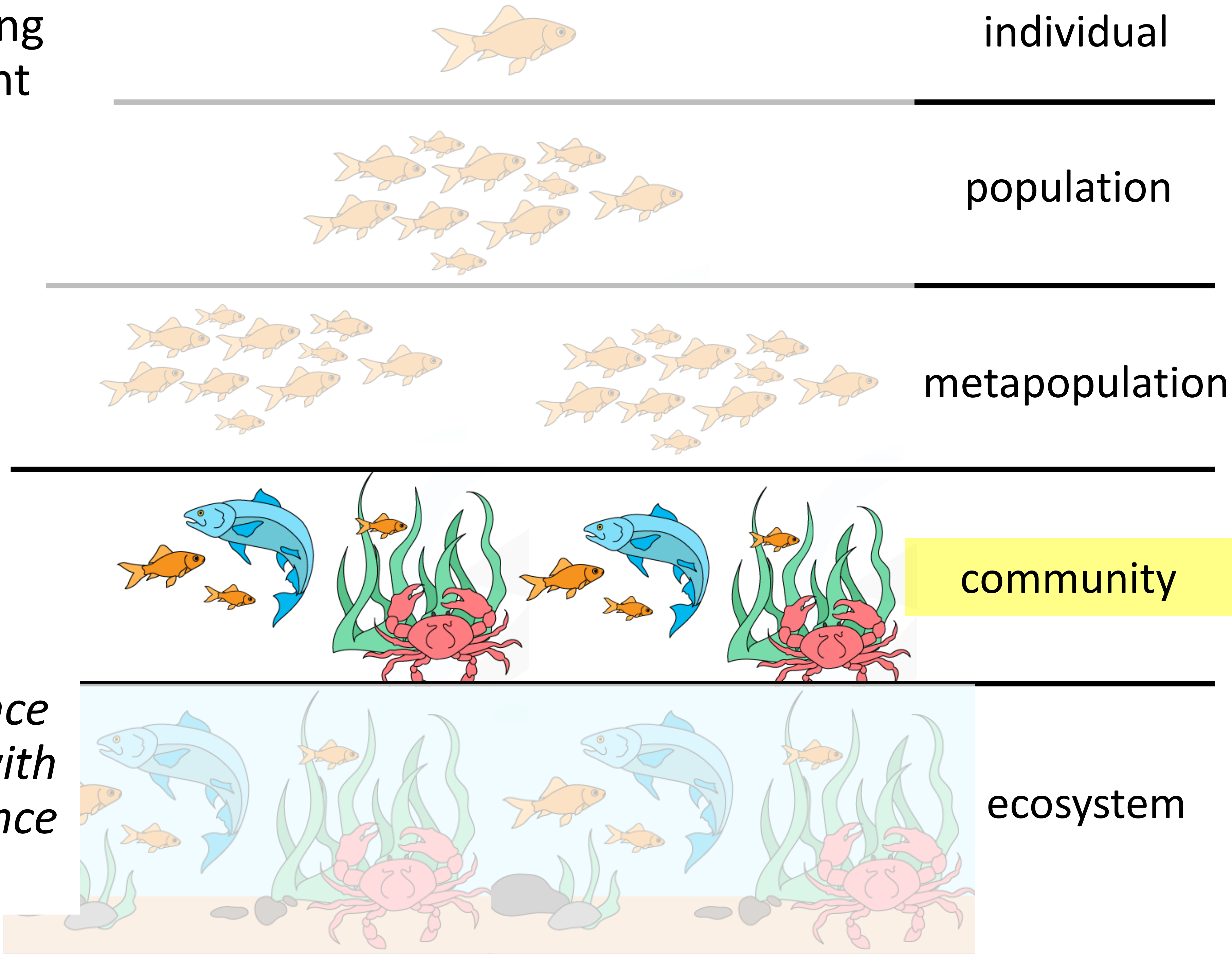

*How does the abundance of **fish species A** vary with changes in the abundance of **fish species B**?*

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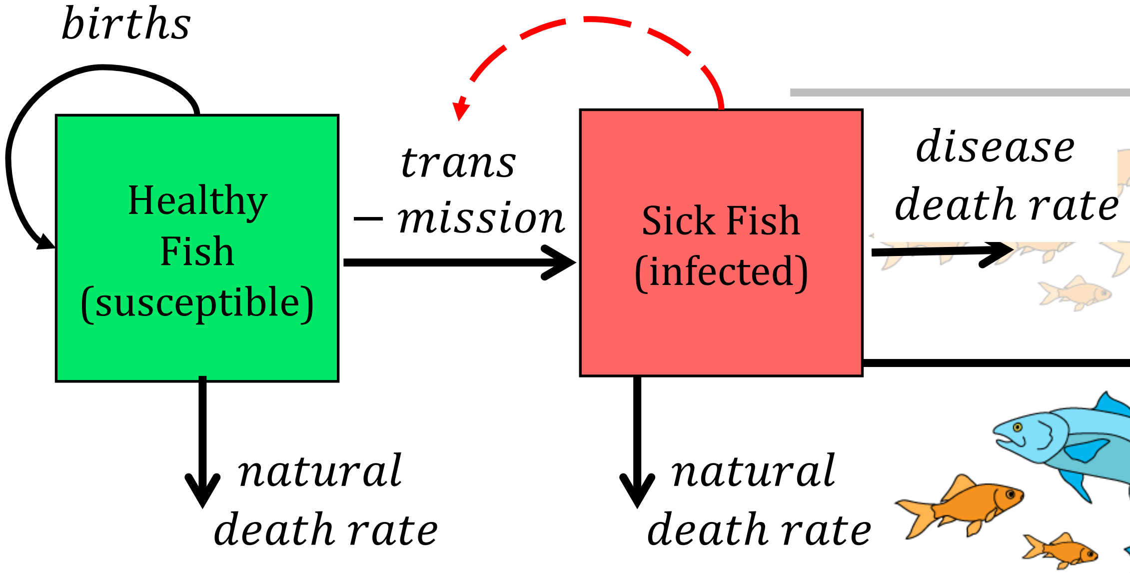

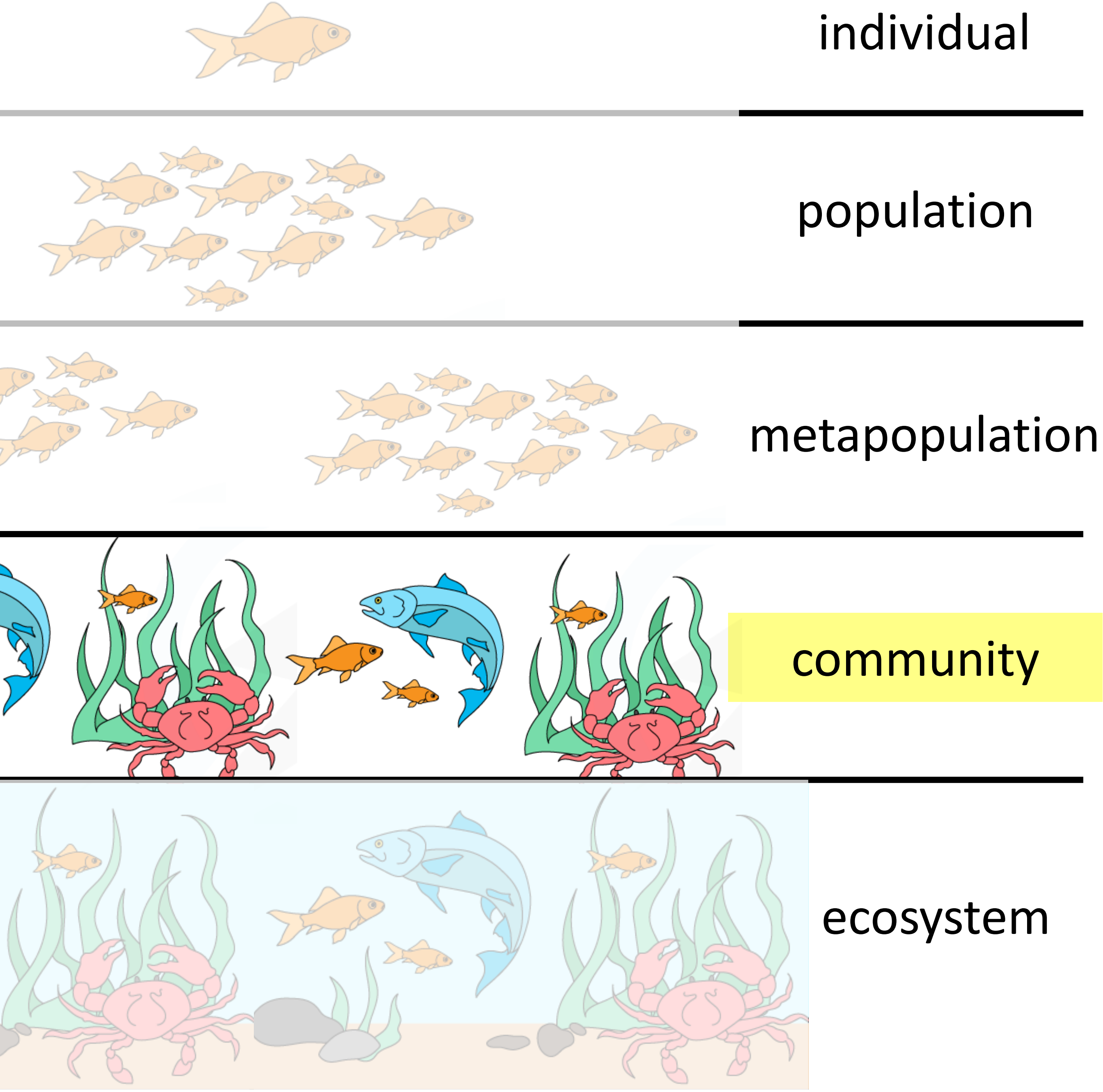

How does the abundance of ***fish*** *change based on* ***infection with Mycobacterium marinum*** *(wasting disease)?*

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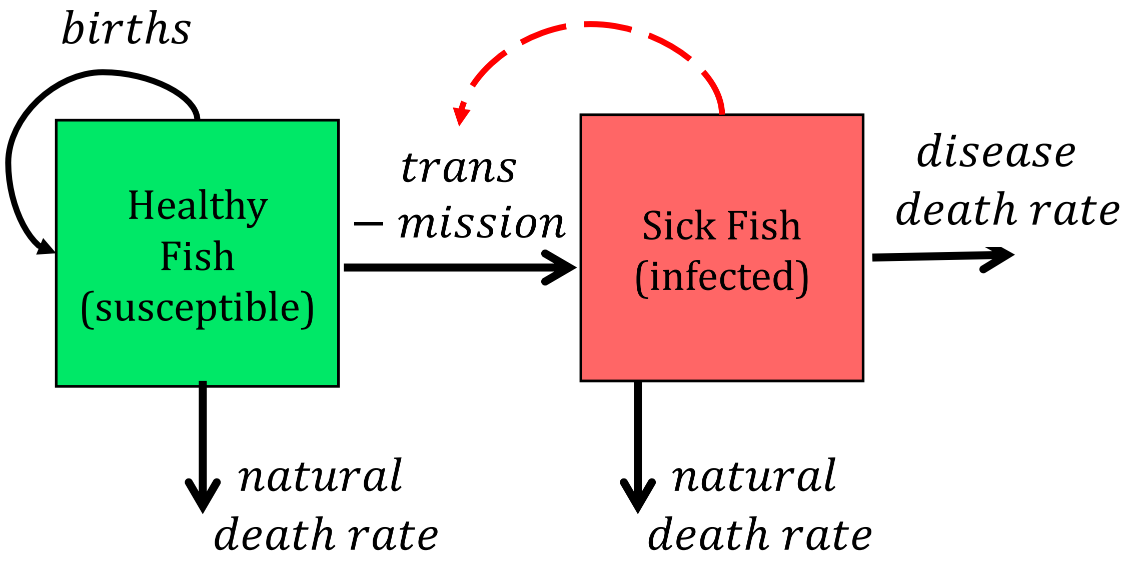

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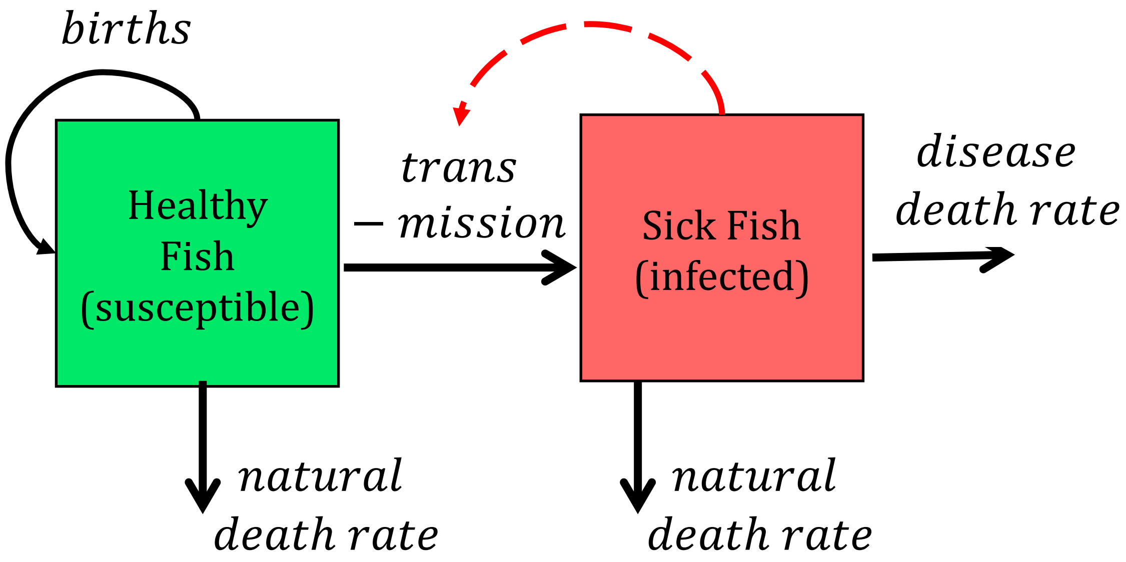

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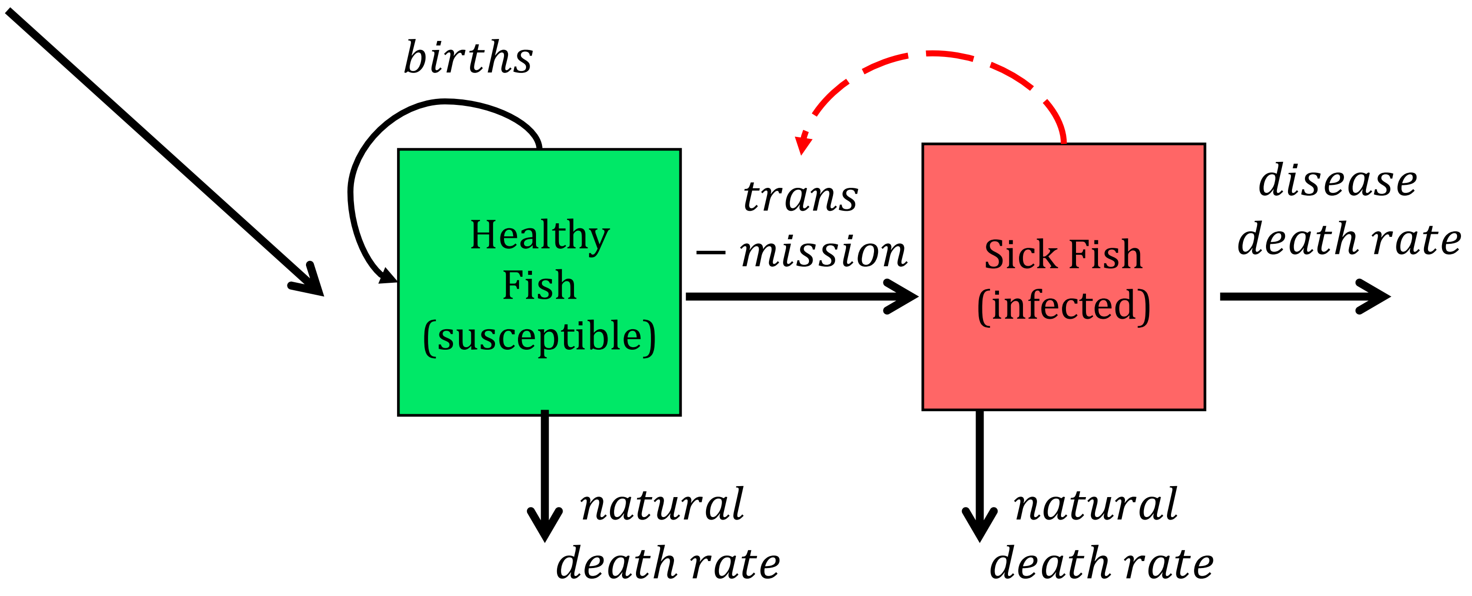
Species can interact in several distinct ways.

- **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)

Each box is a distinct population!

Population Biology



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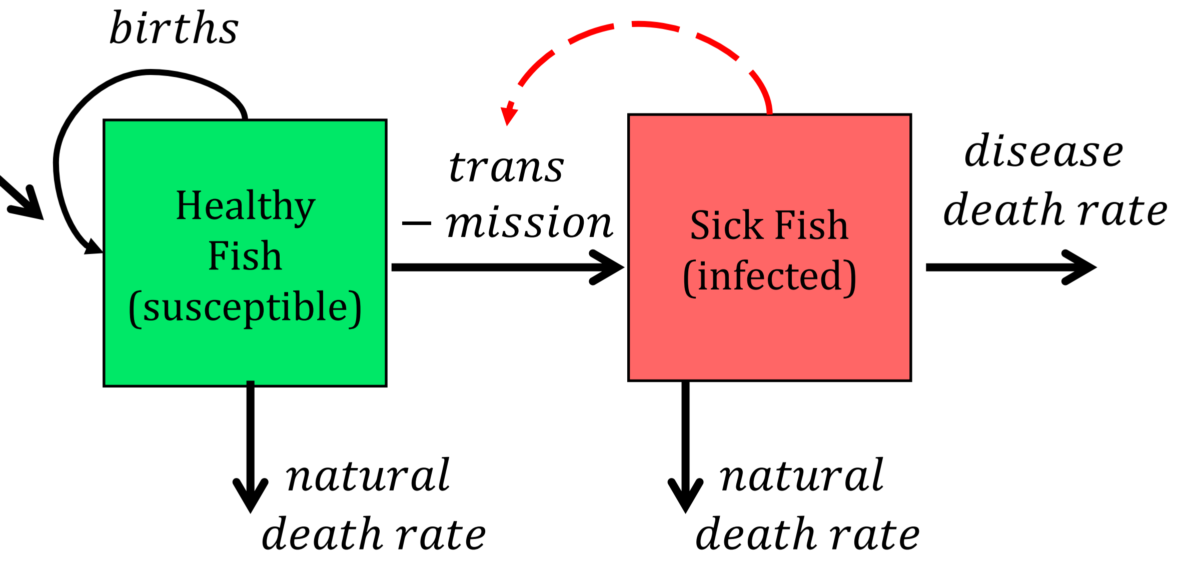
When we model these populations, everyone in the box is considered the same.



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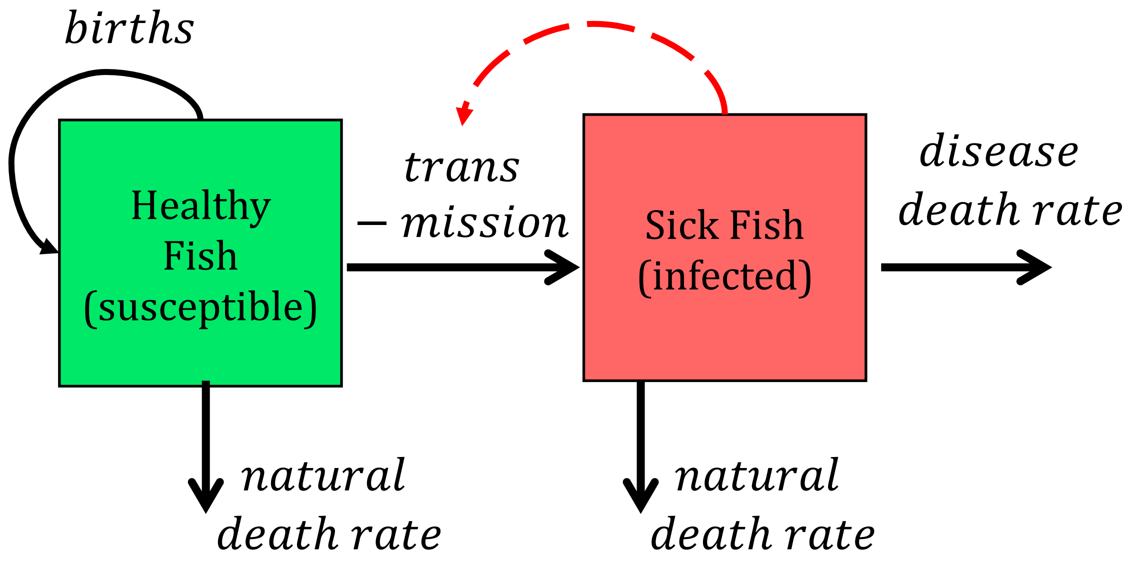
In a continuous time model (ODE), each box would get its own differential equation!



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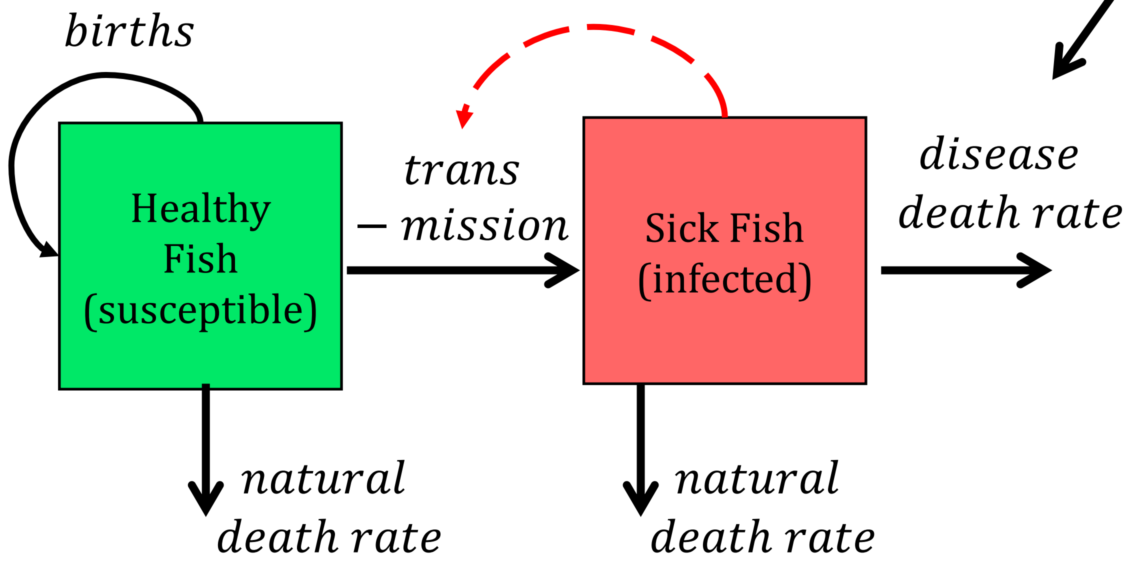

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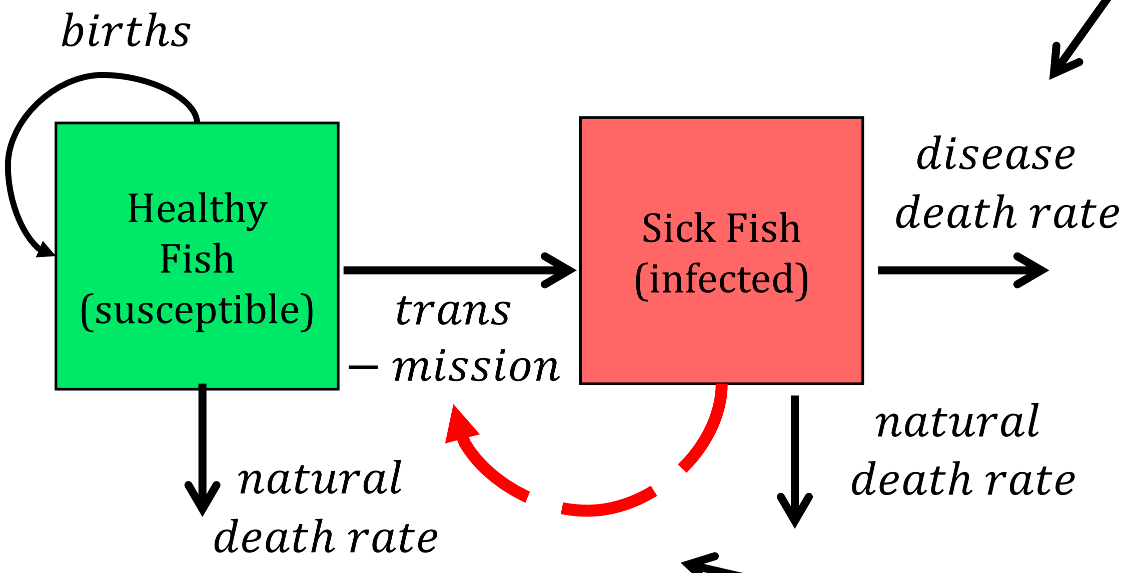

Solid lines are processes by which individuals in each population move between boxes.

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

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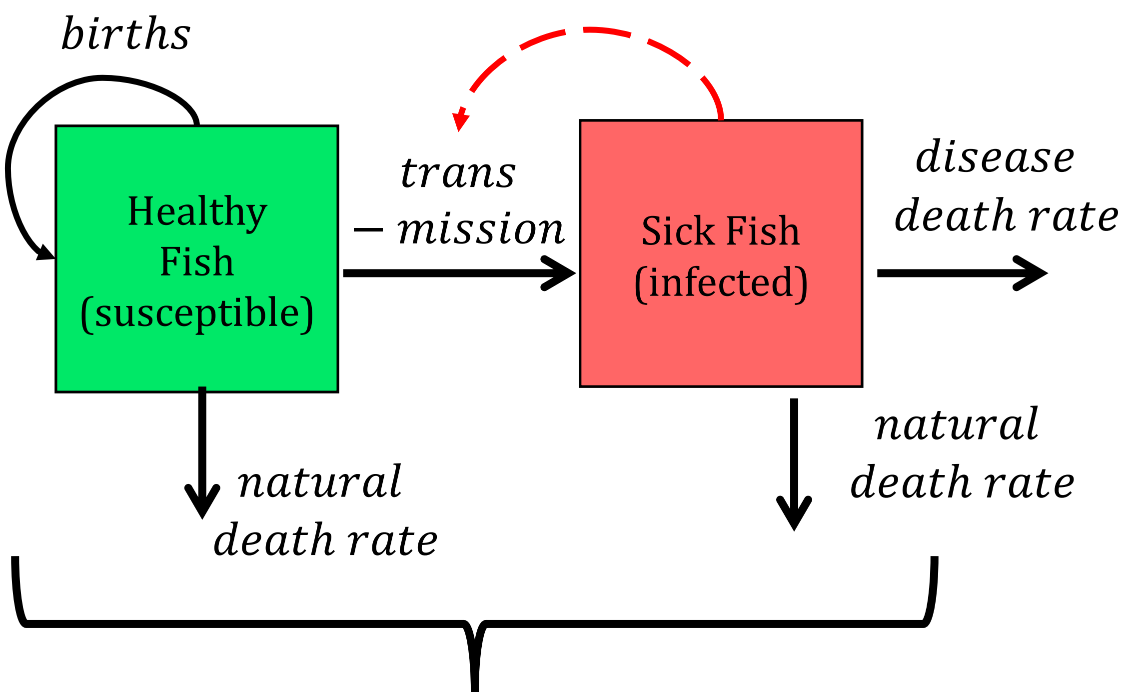

In disease ecology, we model populations based on their **infection status**.

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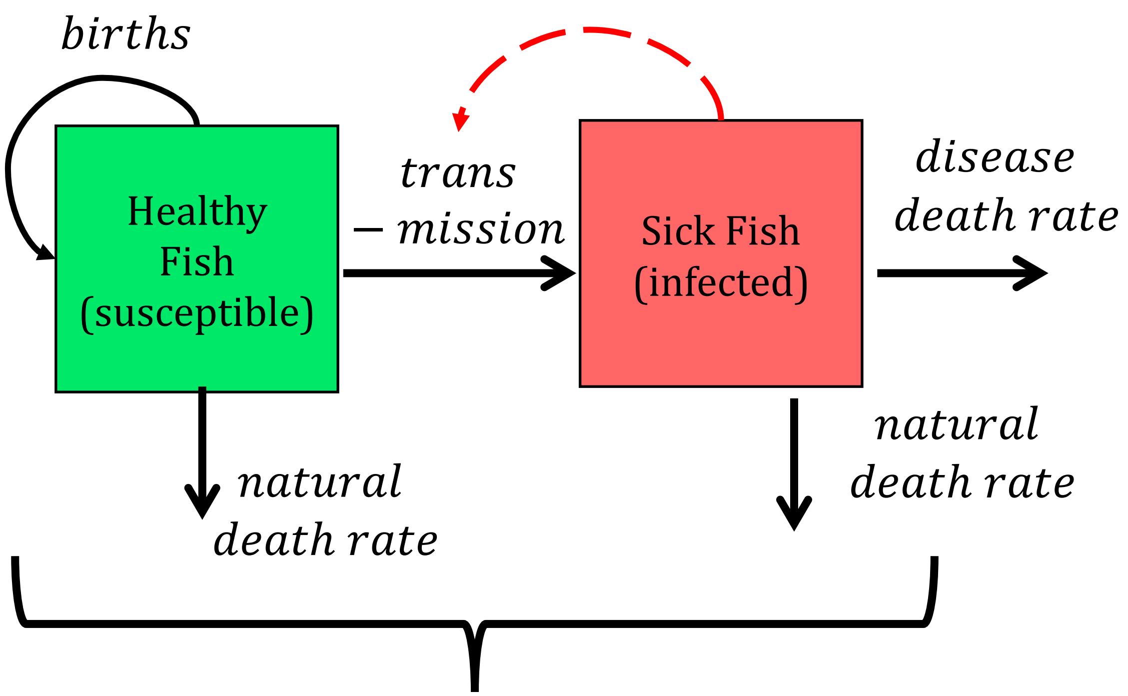

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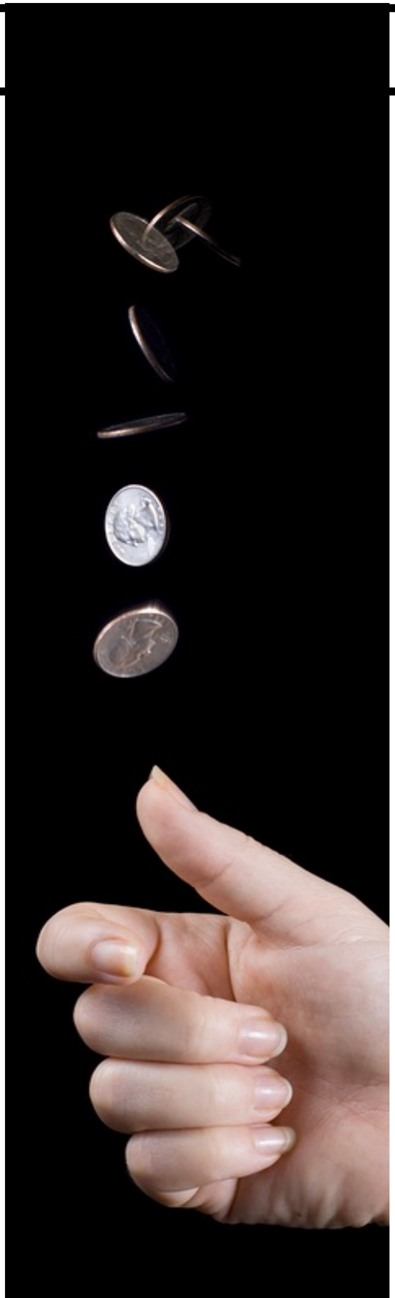
In disease ecology, we model populations based on their **infection status**.

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

Population Biology

Conservation Biology	Disease Ecology



Population Biology

Conservation Biology	Disease Ecology

- **Goal:**
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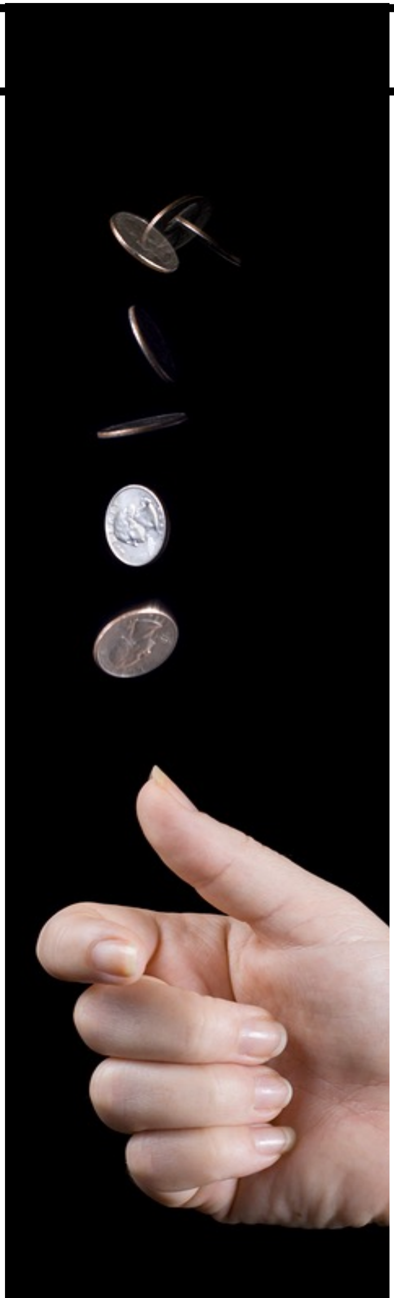

Population Biology

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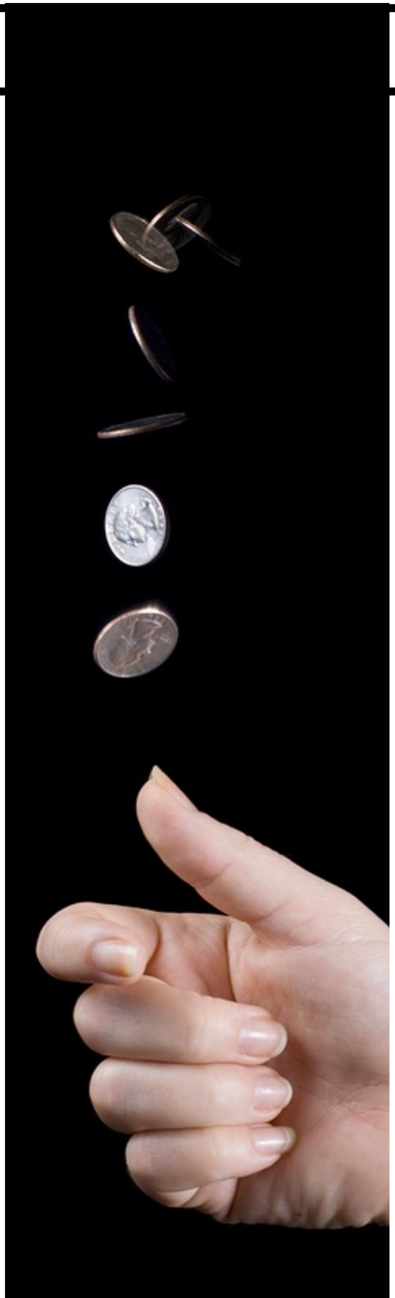

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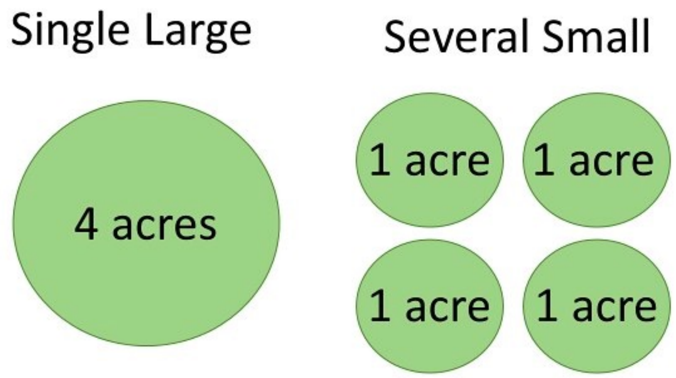

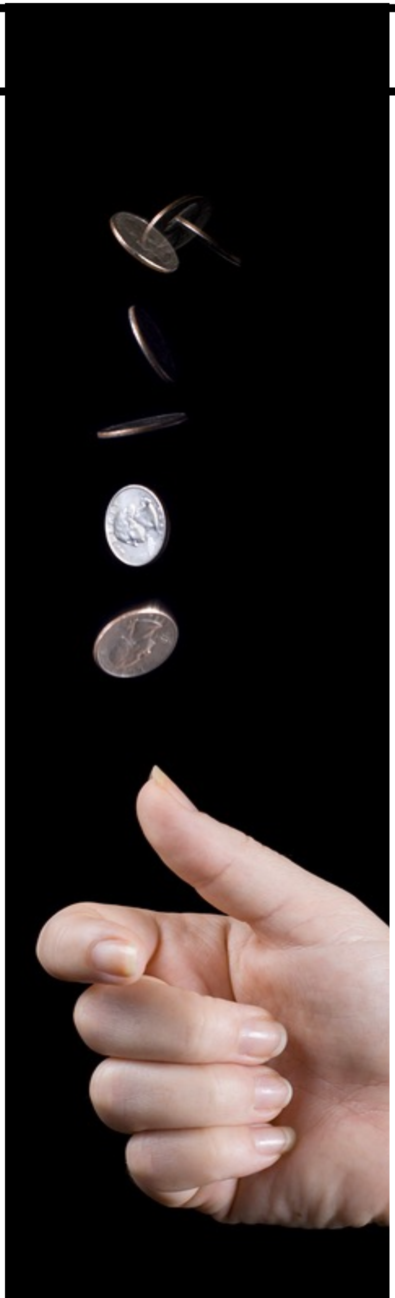

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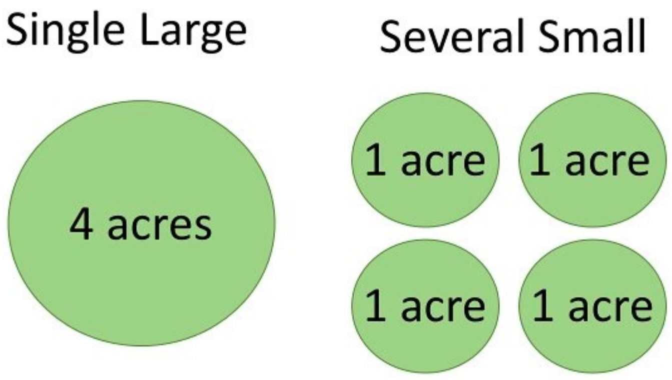

Disease Ecology

- **Goal:**
  - protect **populations** from disease via pathogen **extinction**

Population Biology

Conservation Biology

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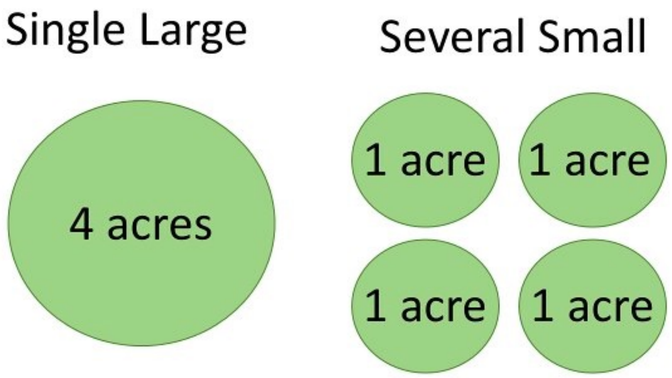

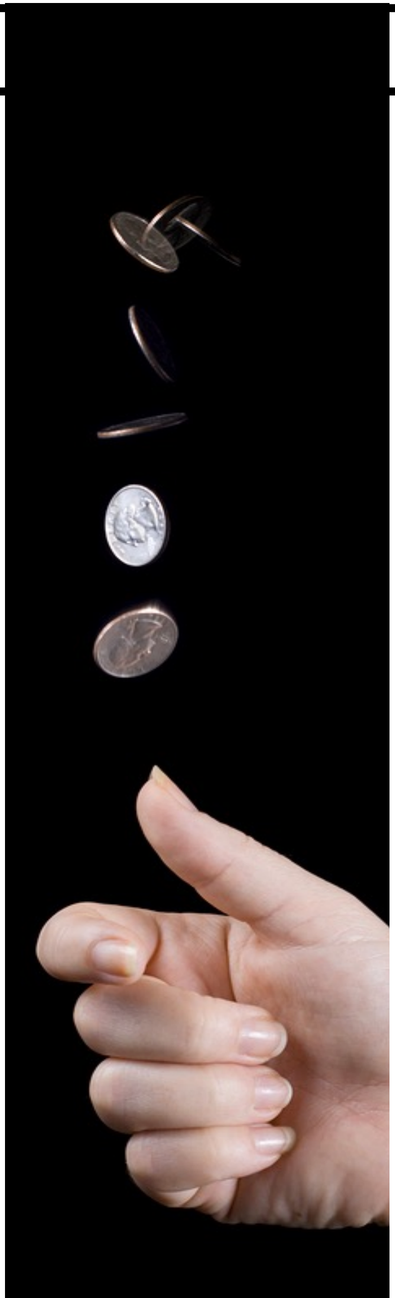

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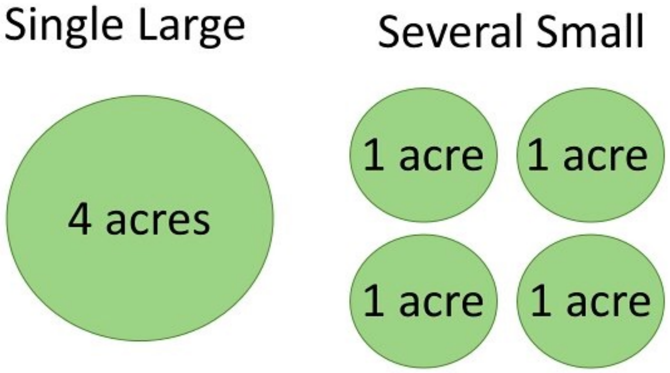

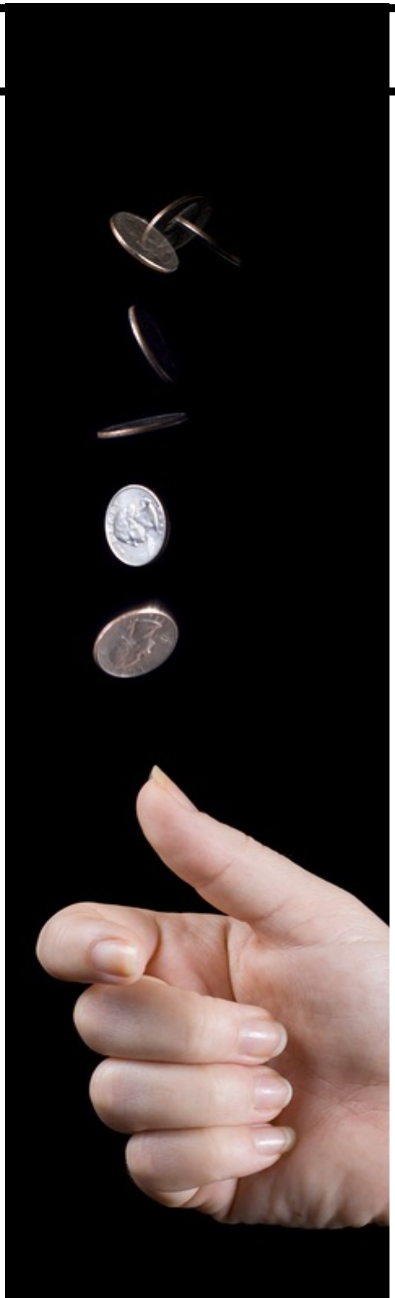

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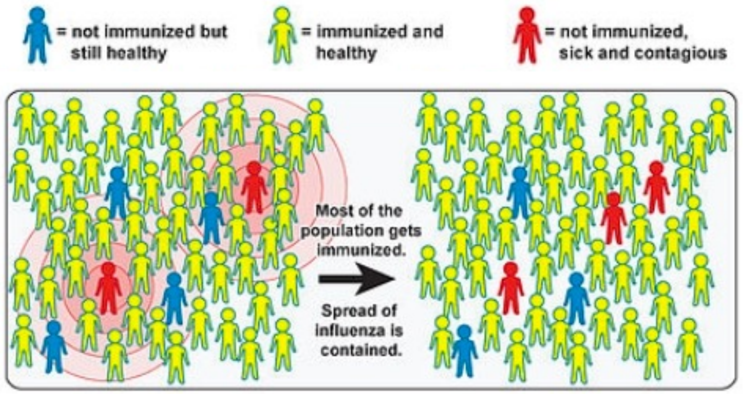
  - **vaccination**

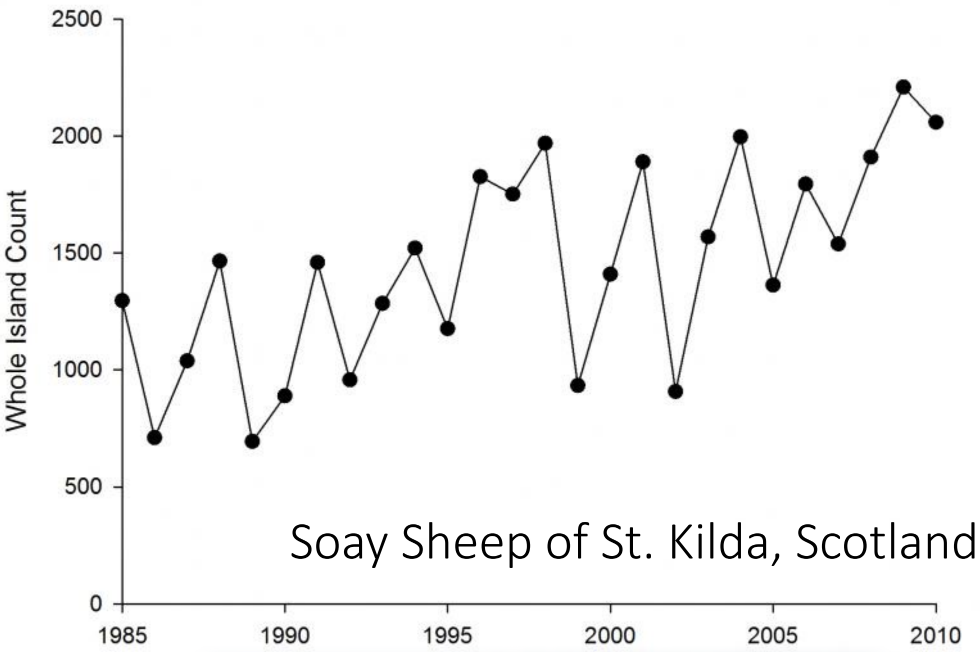


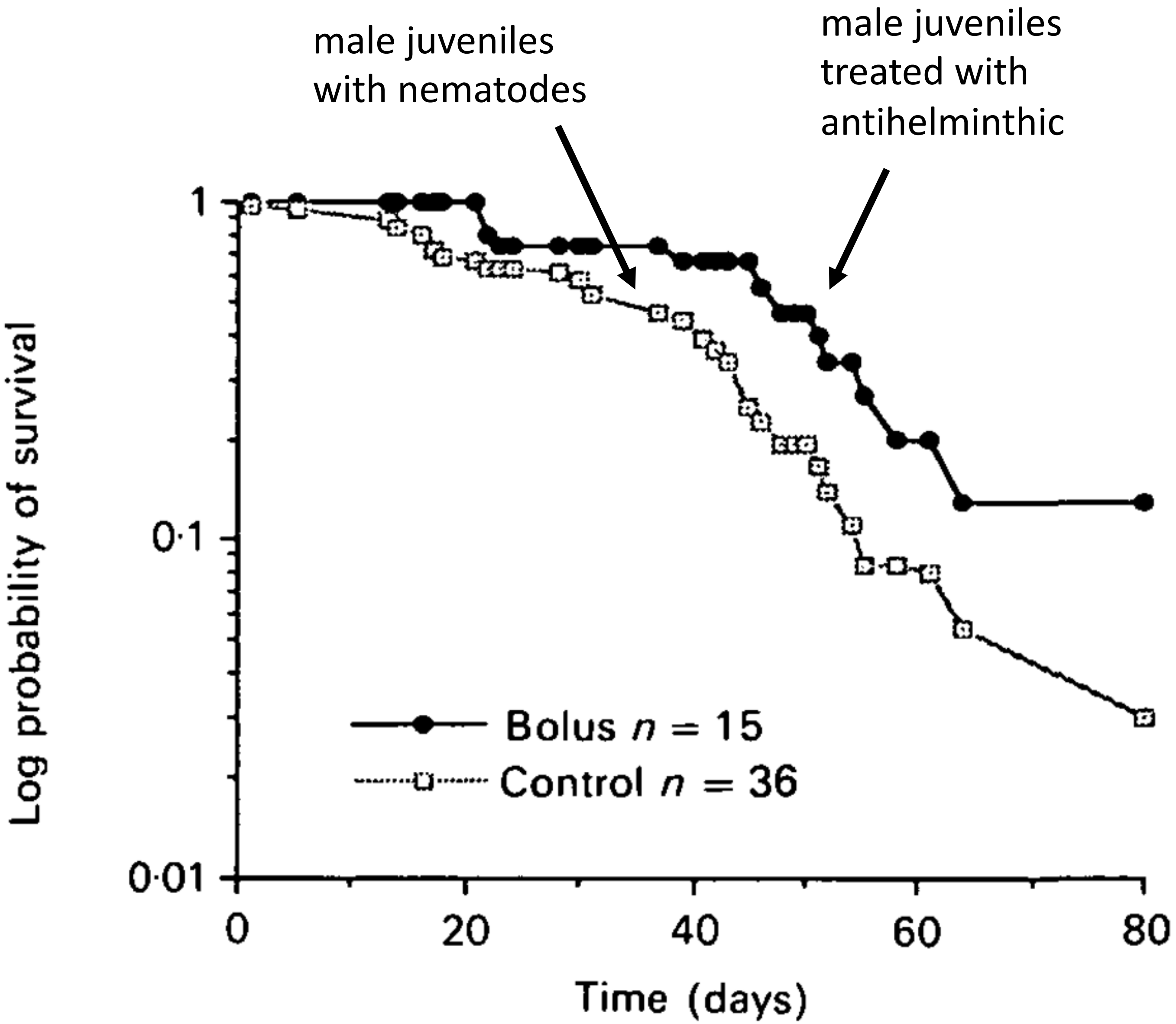
E^{2}M^2: **Ecological** and **Epidemiological** Modeling in Madagascar

December 16, 2022

E2M2.org

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





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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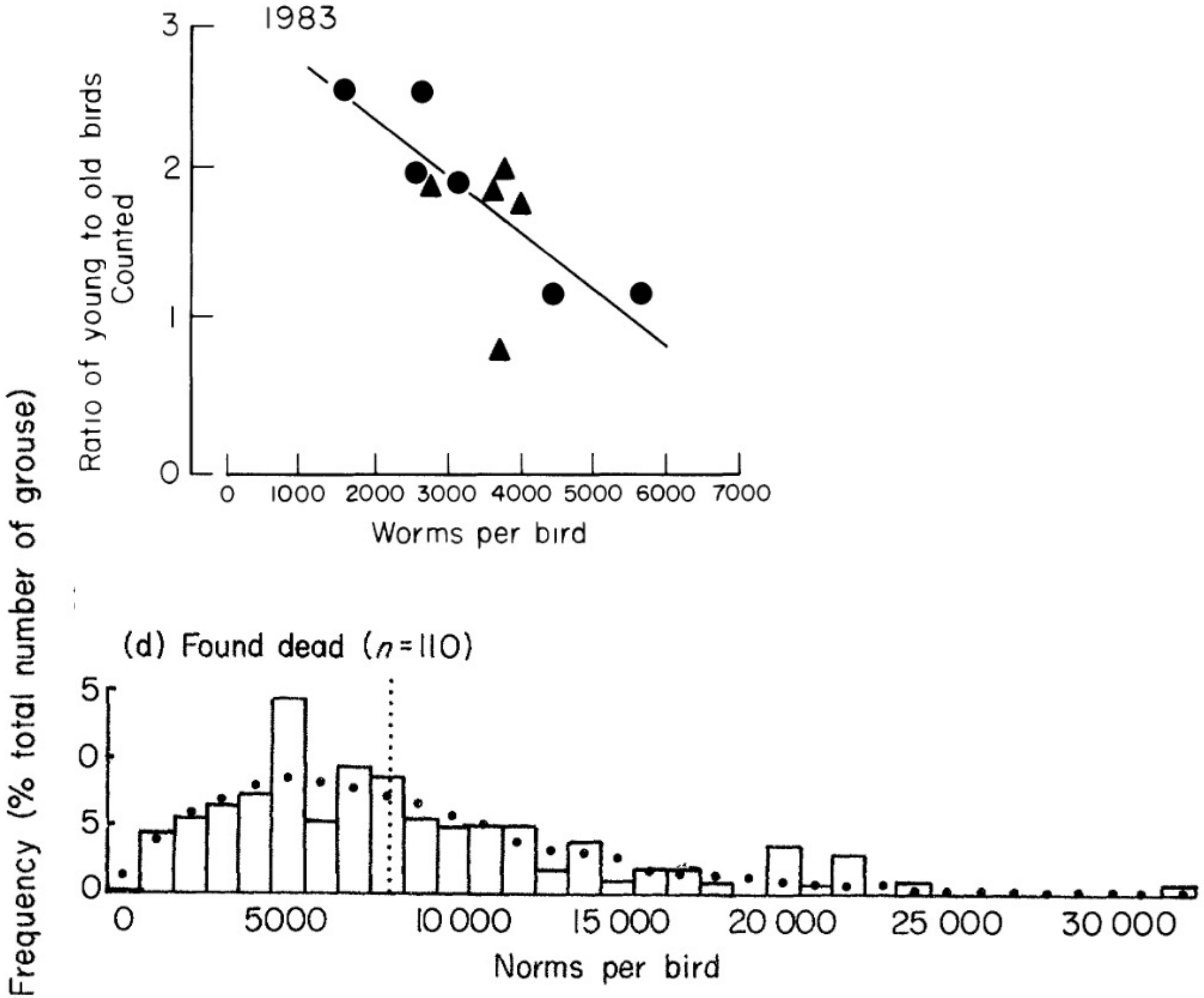

Higher burden of the intestinal strongyle worm, *Trichostrongylus tenuis,* both reduces breeding success and increases mortality in red grouse.

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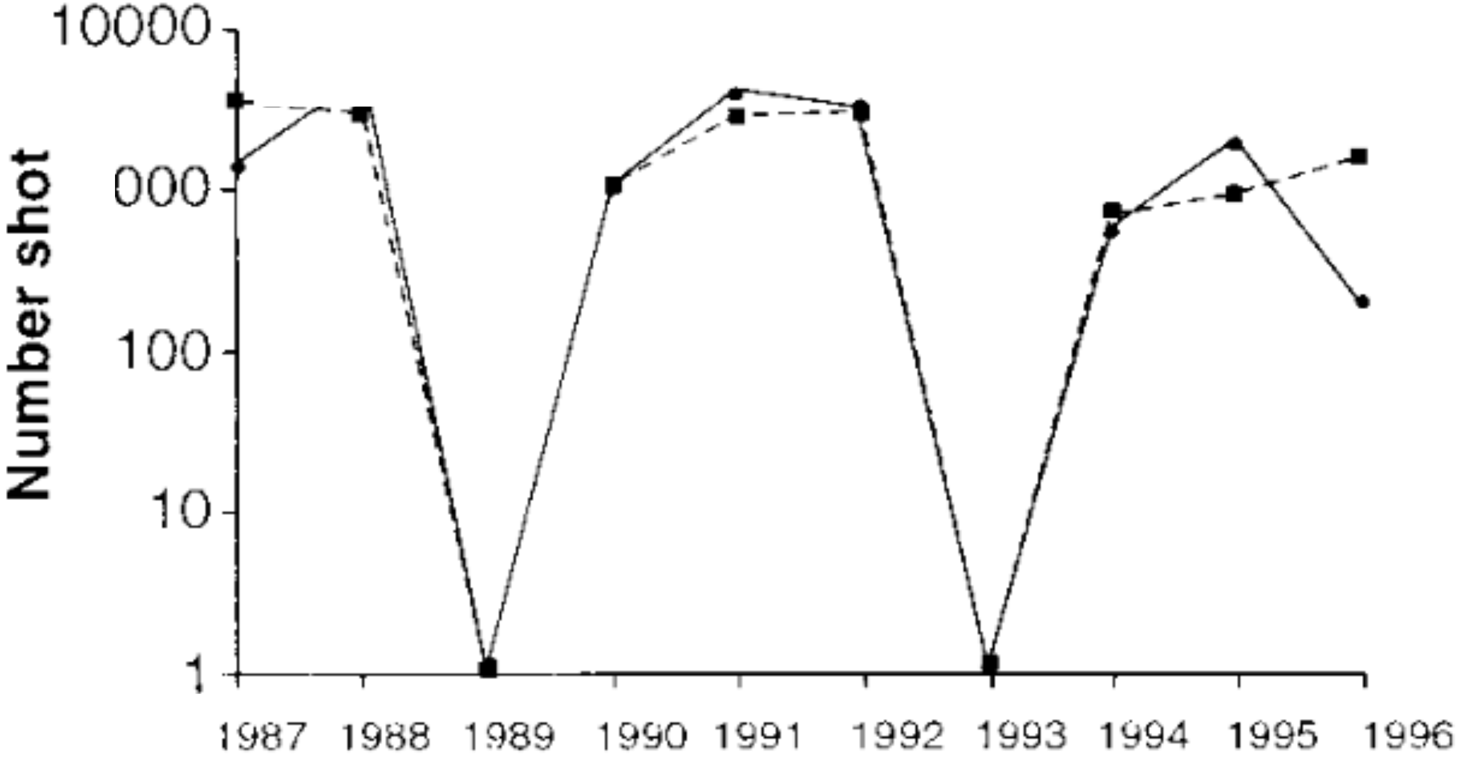
Worms were hypothesized to be responsible for the observed population cycles in ‘bag data’ from northern England.



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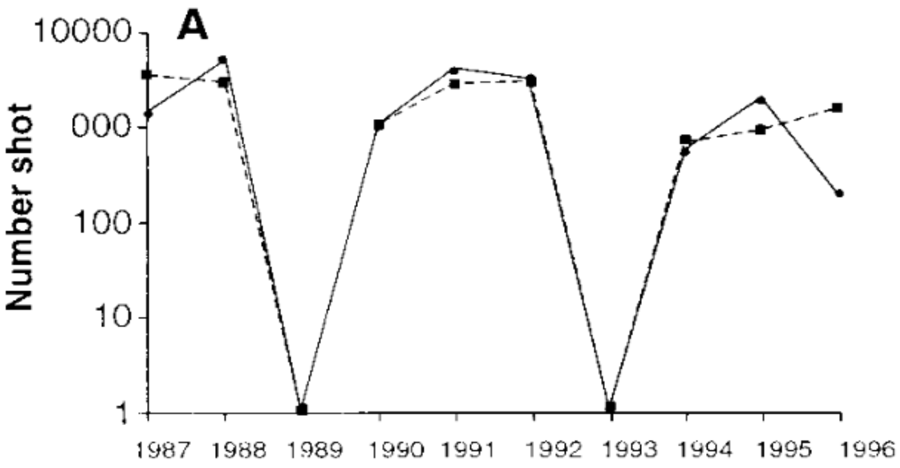
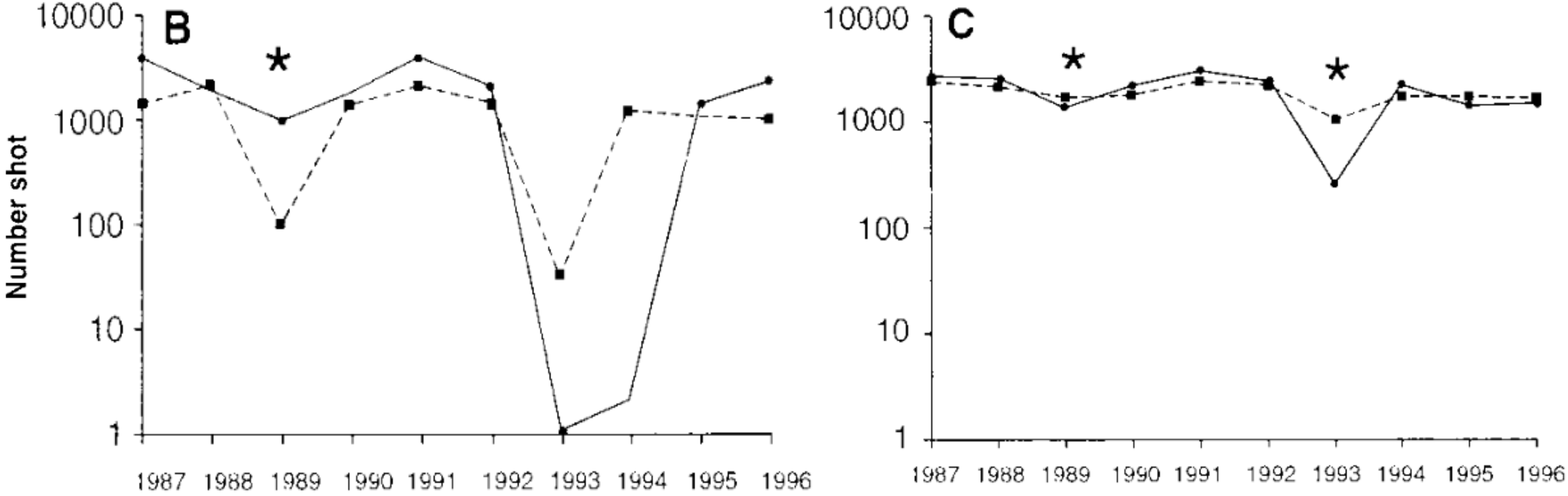


Deworming eliminated population cycles to prove this effect!



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

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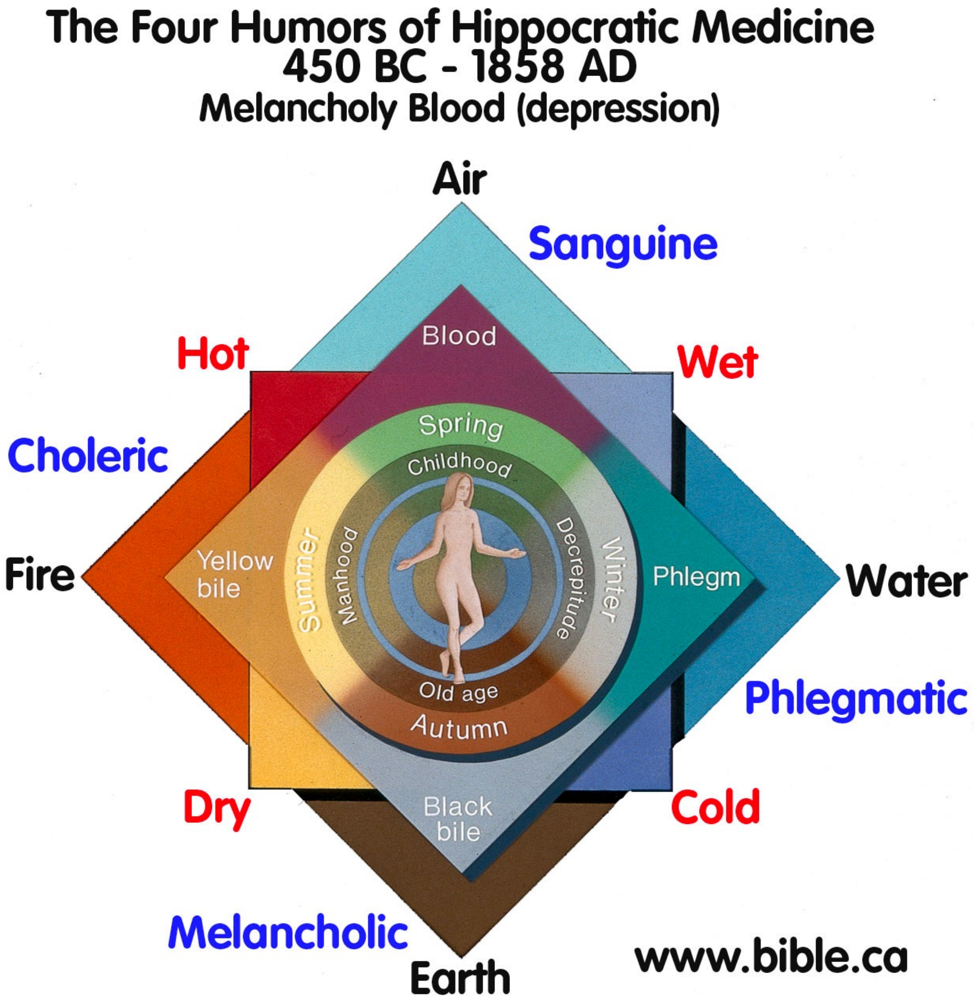

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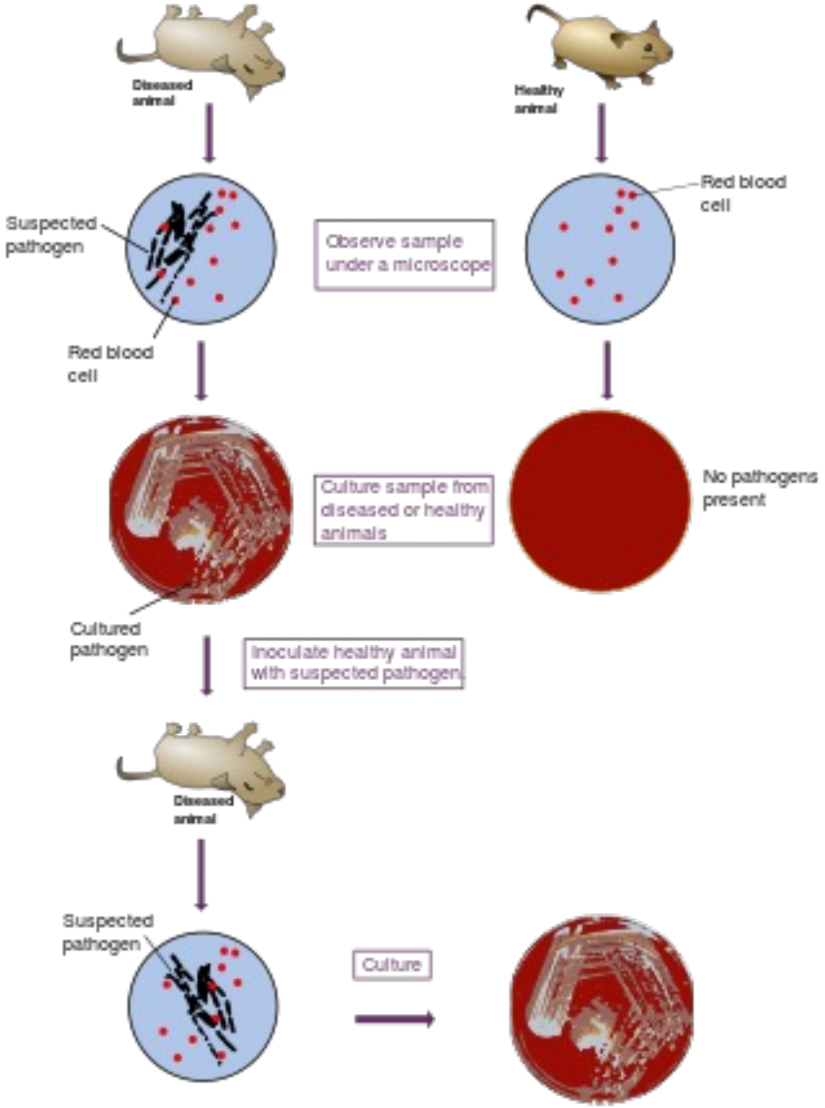
3. **Germ Theory of Disease:** Idea that disease results from germs
   - Leuwenhoek's microscope (1675)
   - Koch's postulates (1890)



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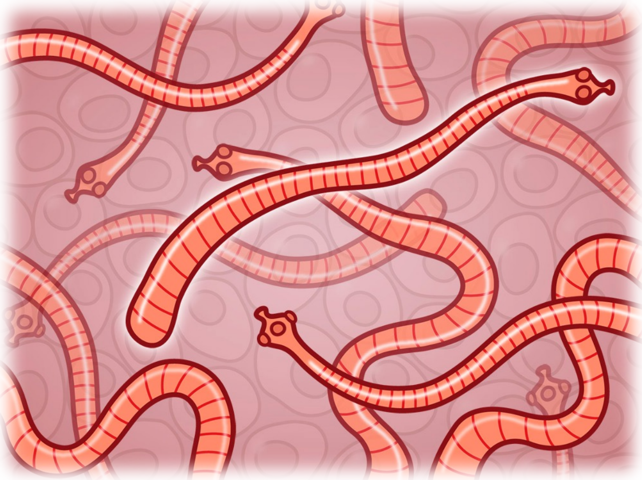
4. **<u>Classic epidemiology</u>**
   - Risk factors for disease = John Snow (1854)

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: *Yersinia pestis*
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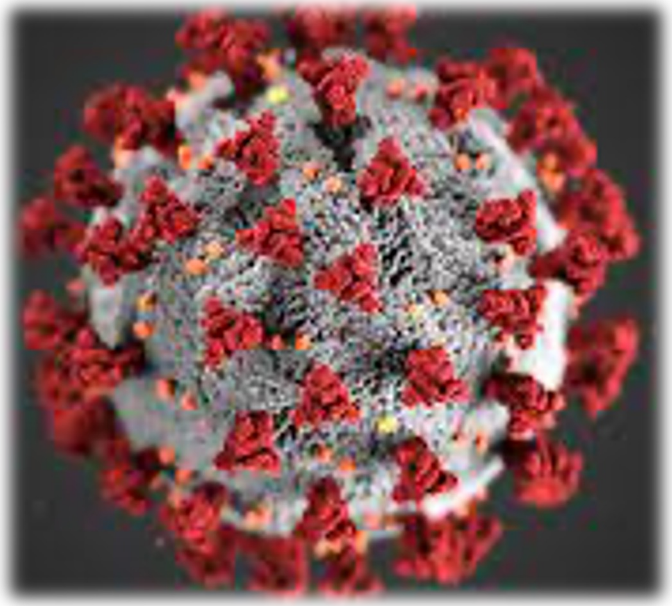
Pathogen: SARS-CoV-2
Disease: COVID-19



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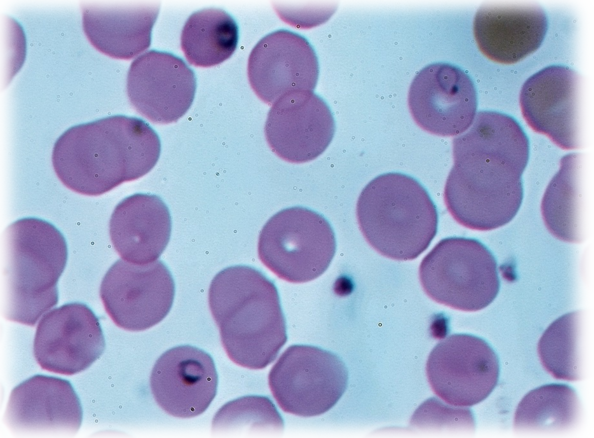
Pathogen: SARS-CoV-2
Disease: COVID-19

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

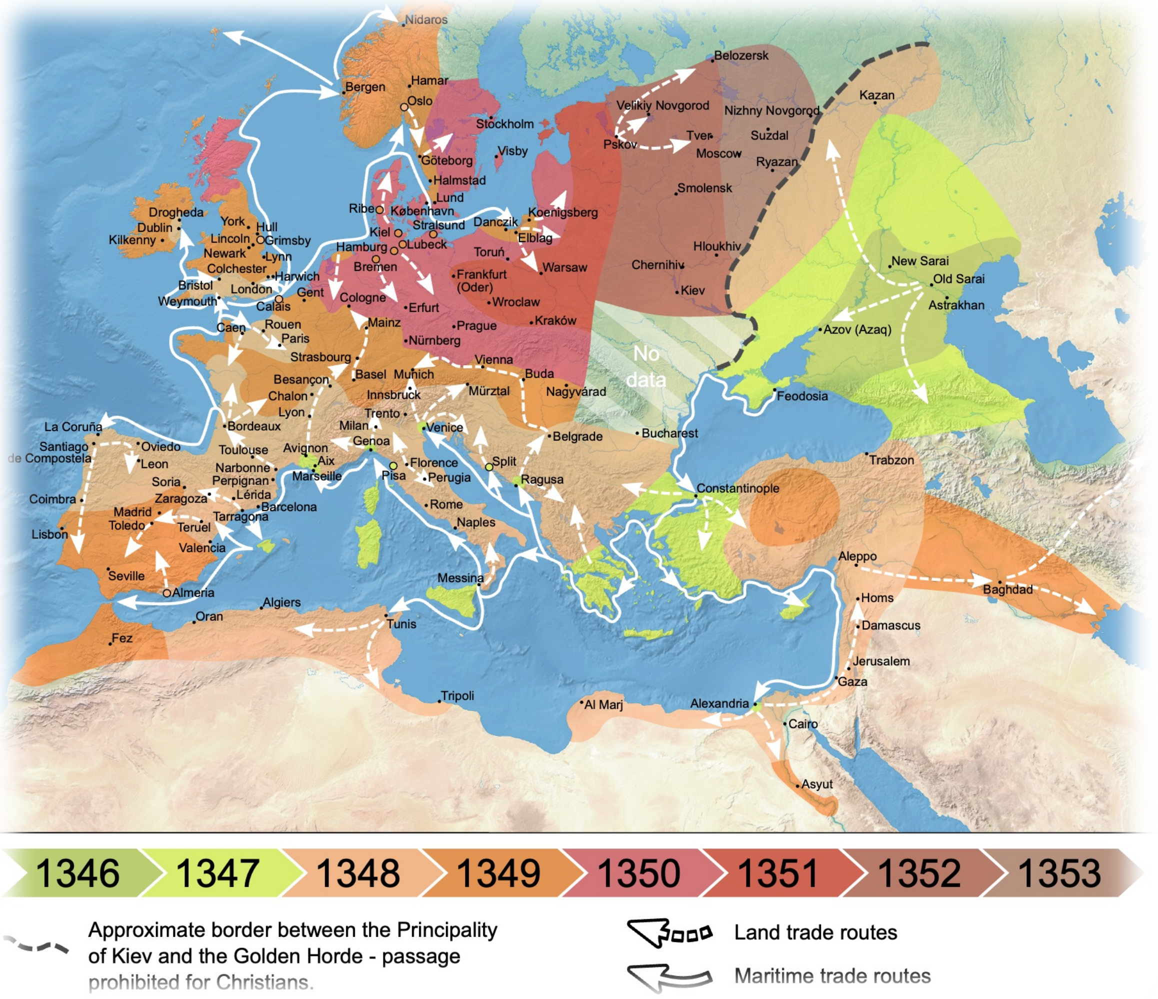


Parasites and pathogens have also shaped human history.

- 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



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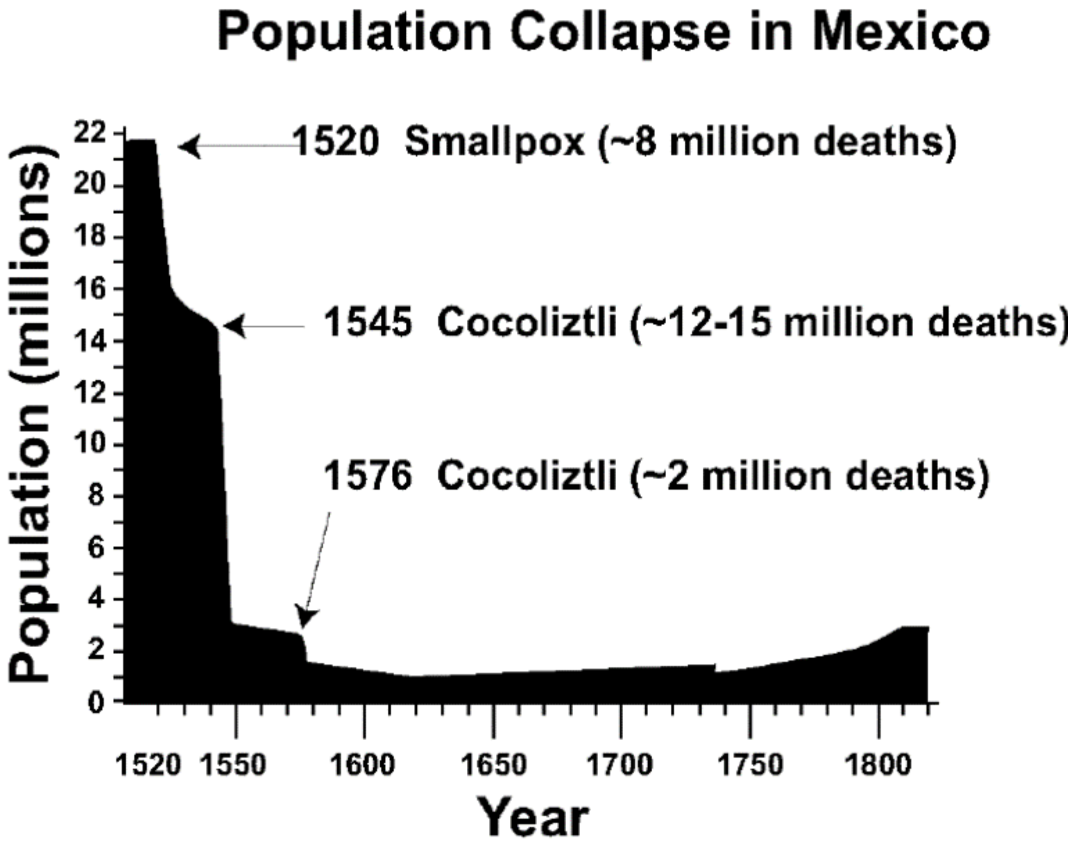
- Plague of Justinian (541-549 AD)
- Black Death (1346-1353 AD)
- Cocoliztli (1545-1548)
  - Pathogen still unknown! Maybe viral hemorrhagic fever, maybe bacterium
  - Killed 80% of the population of Mexico



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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)

Parasites and pathogens have also shaped human history.

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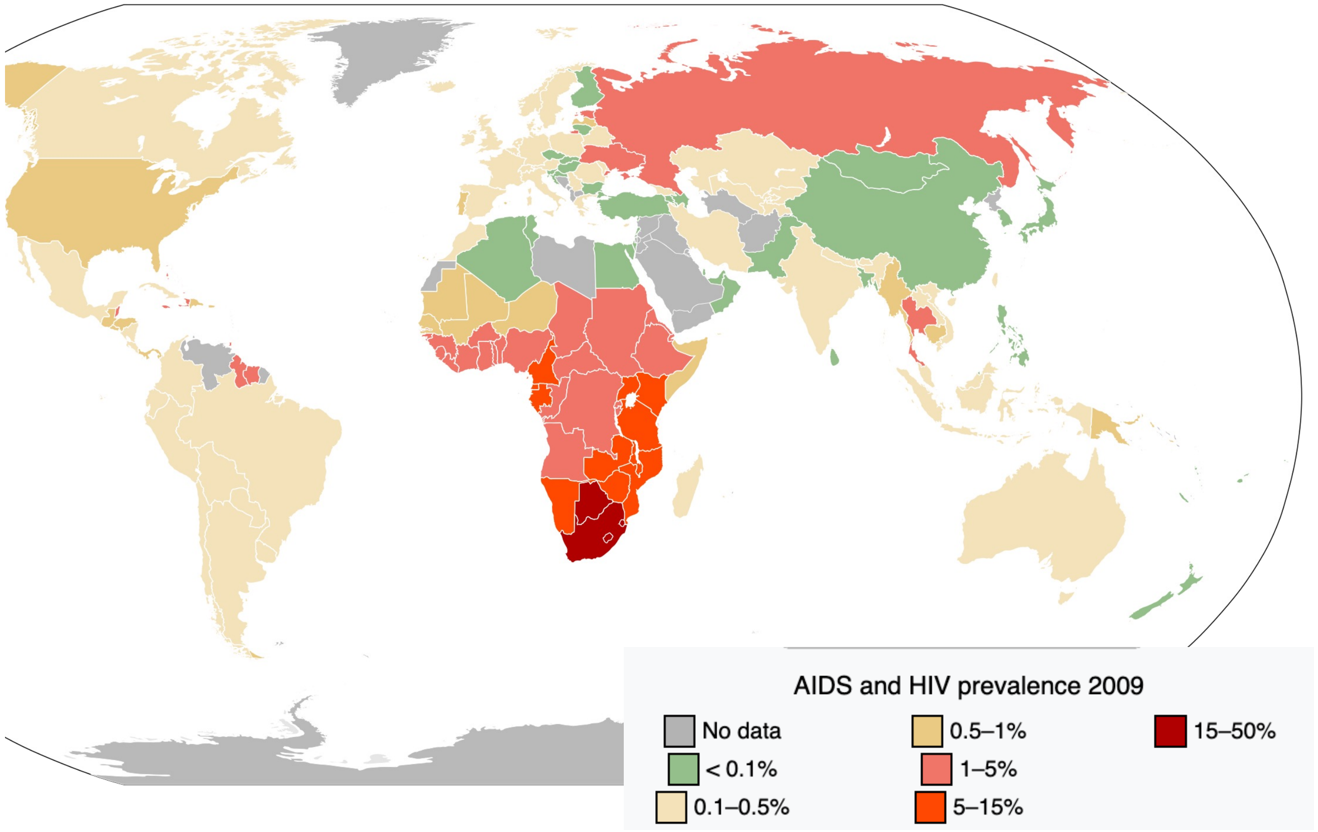
- Spanish Influenza (1918-1920)
- HIV (~1960-now)
  - >40 million deaths and counting
  - Prevalence still >20% in some countries in southern Africa



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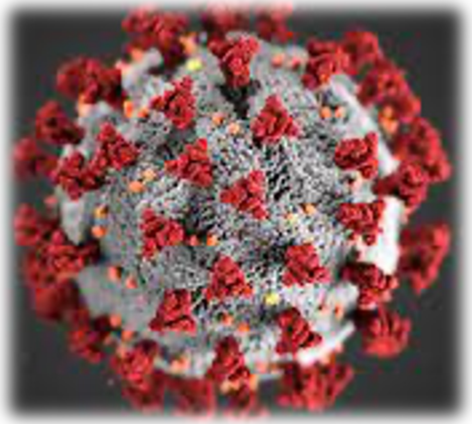
- Spanish Influenza (1918-1920)
- HIV (~1960-now)
- COVID-19 (2019-now)
  - ~7-29 million deaths worldwide
  - ~0.1-0.4% of population

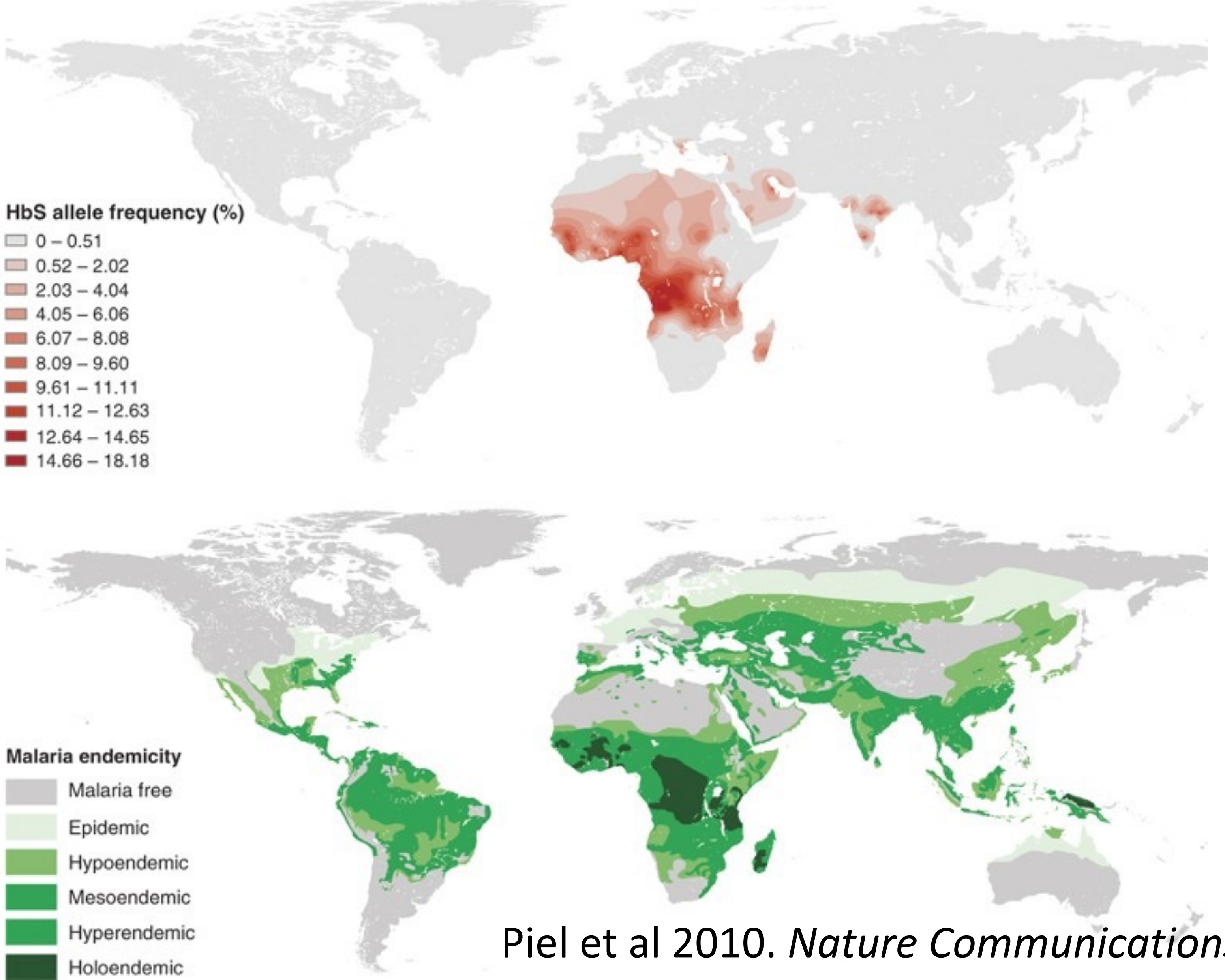


Parasites and pathogens have also shaped human DNA.

Parasites and pathogens have also shaped human DNA.

Sickle cell anemia

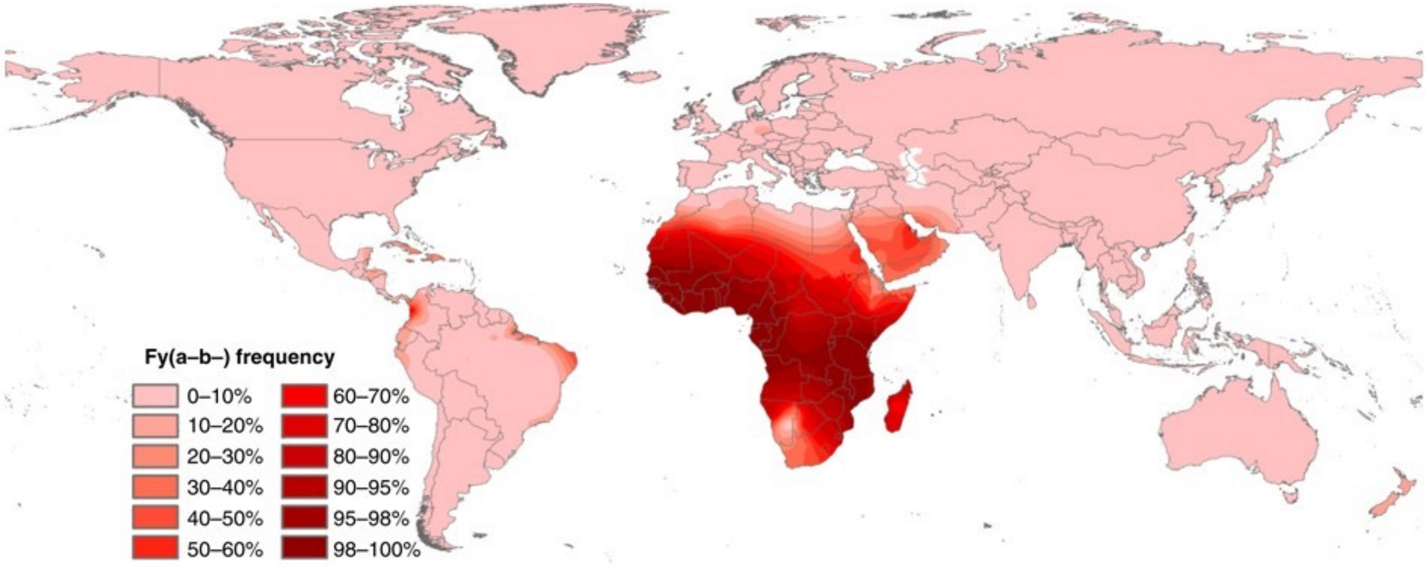
- The HbS allele confers resistance to malaria but also results in sickle cell anemia when homozygous.
- Natural selection has favored this trait in malaria-endemic regions of the planet.
- As of 2021, WHO estimates 247 million malaria cases worldwide and >600,000 deaths, 95% in Africa.
- Children <5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



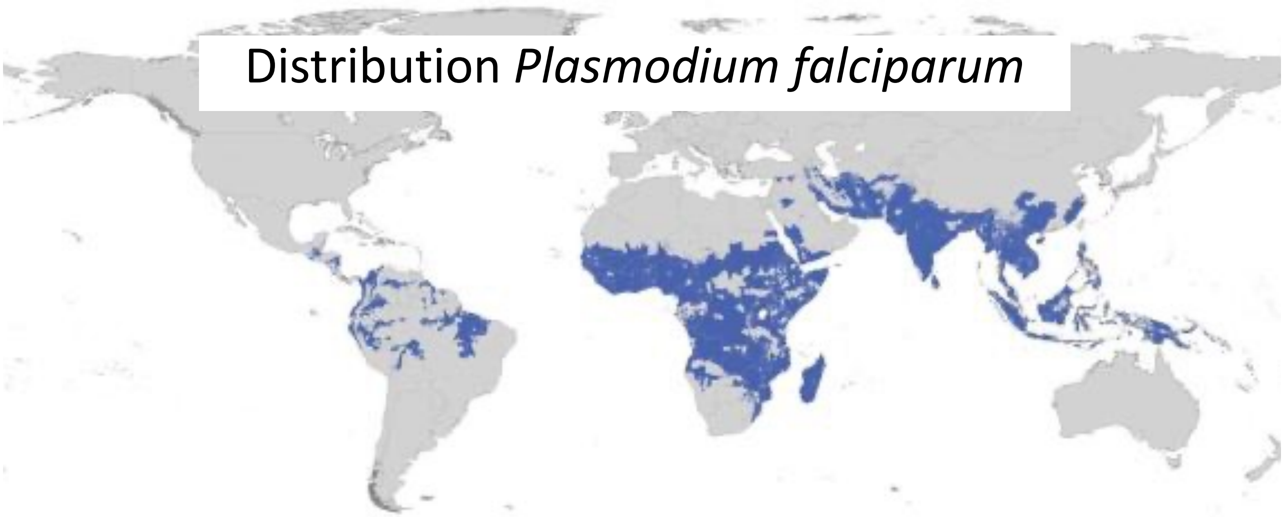
Piel et al 2010. *Nature Communications.*

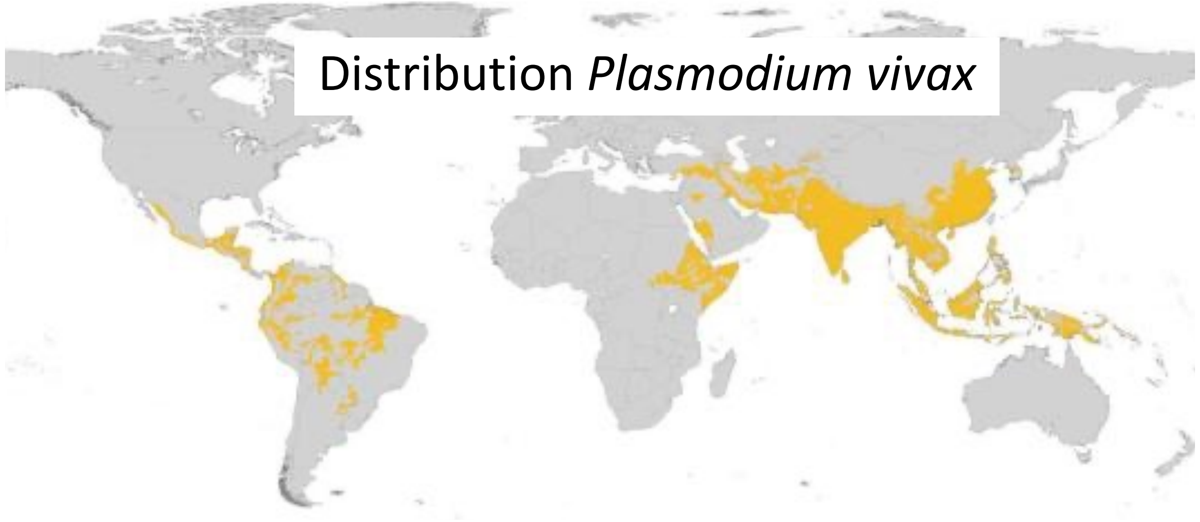
Parasites and pathogens have also shaped human DNA.

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

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- Disease Ecology = the study of **how** a disease spreads
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- 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**

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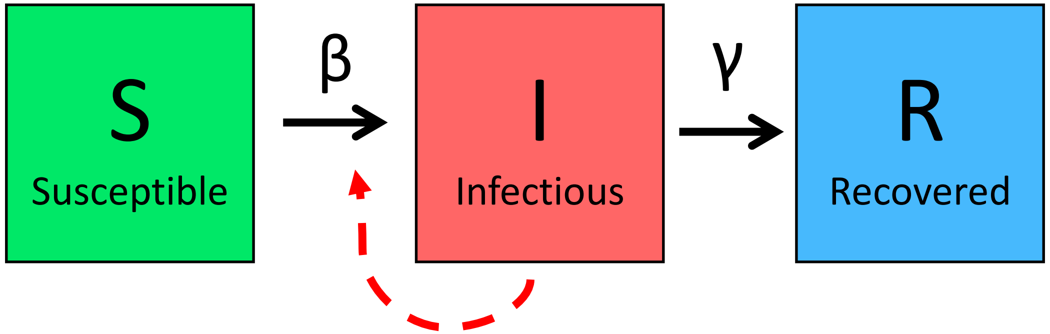
  - Emphasis on the **interactions** of organisms with each other and the environment…when interactions result in disease
- Emphasis on understanding the **transmission dynamics** of infectious disease
- Typically involves fitting dynamical (population) models to **time series** data
- More mathematical (dN/dt)
  - **Process**

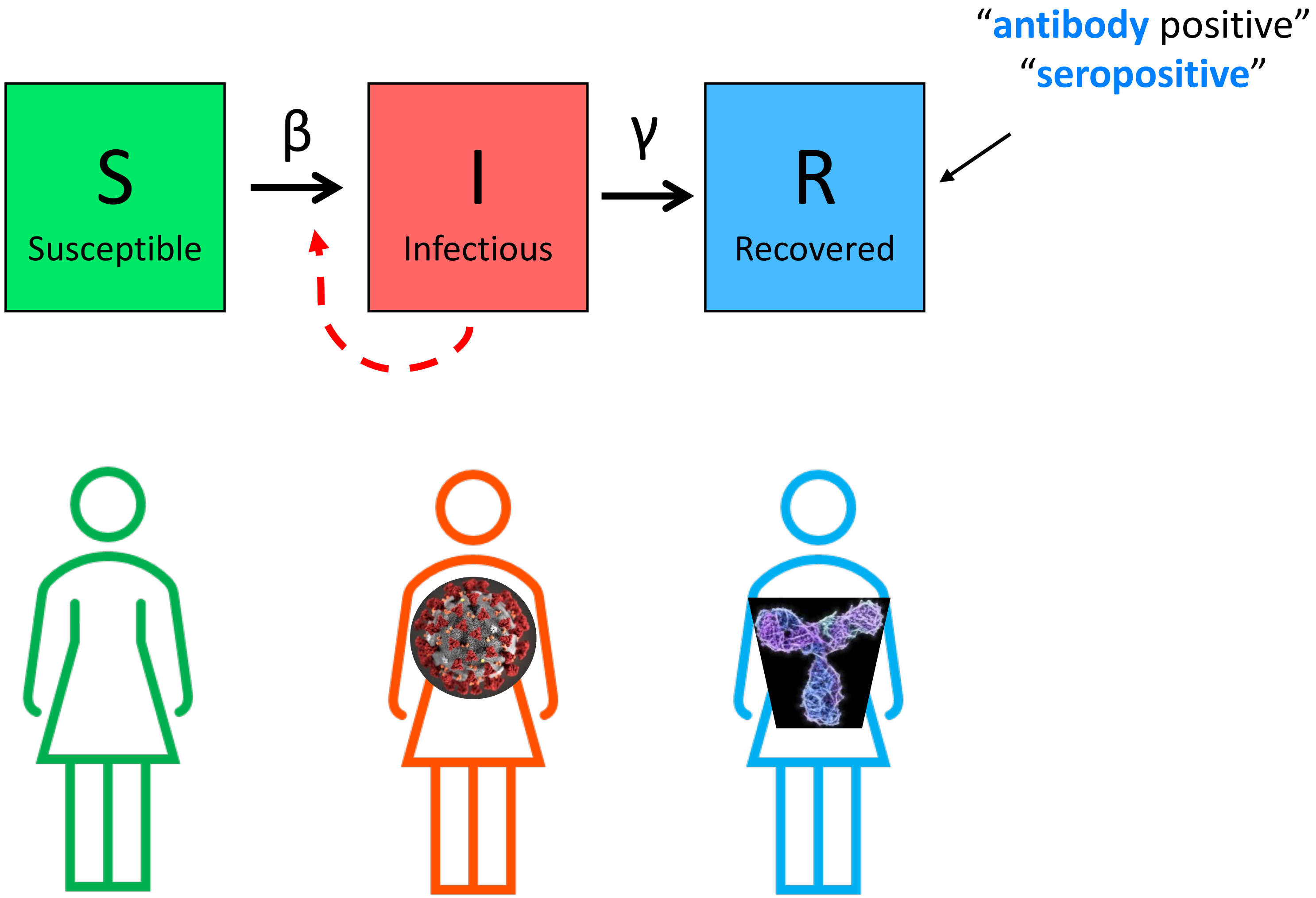
The SIR Model



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**.



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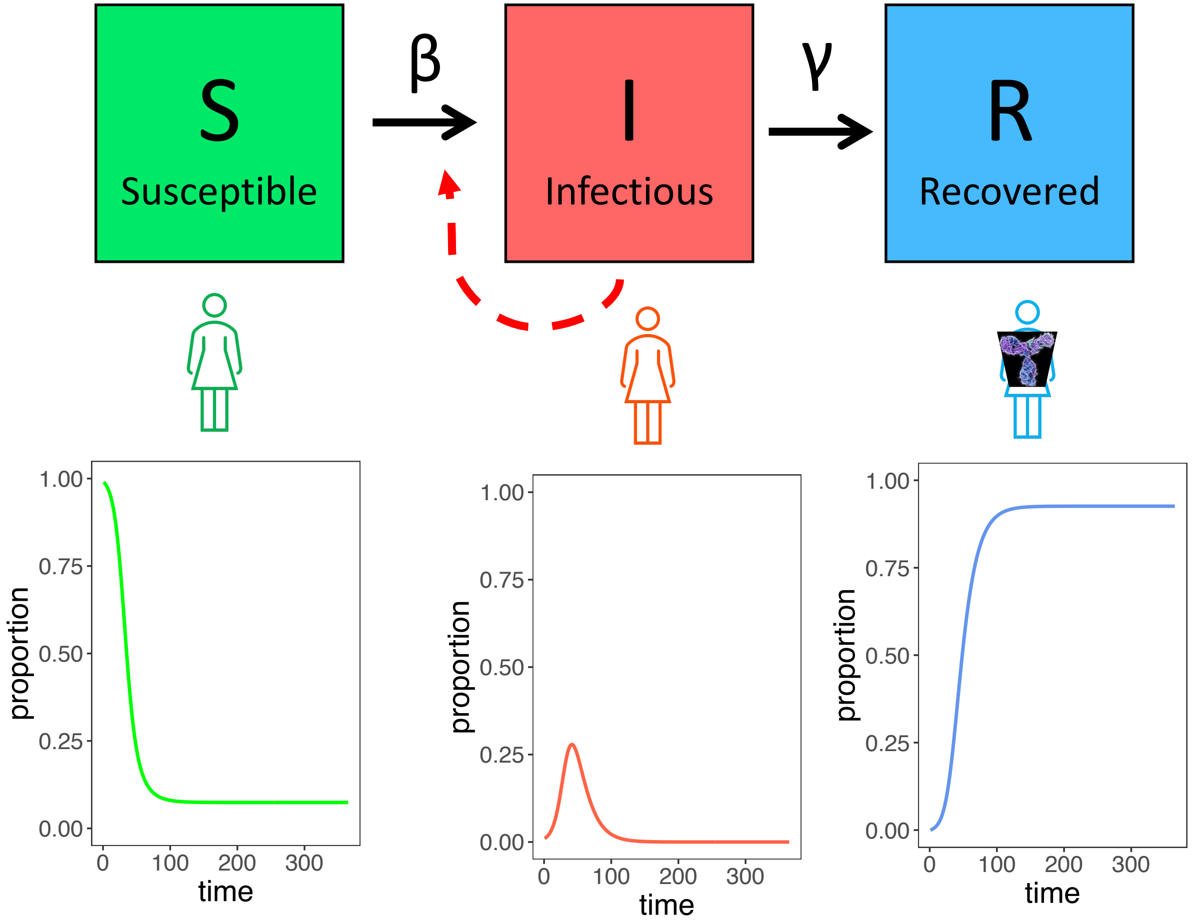
We use computers to simulate systems of equations in the SIR framework.

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

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

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



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

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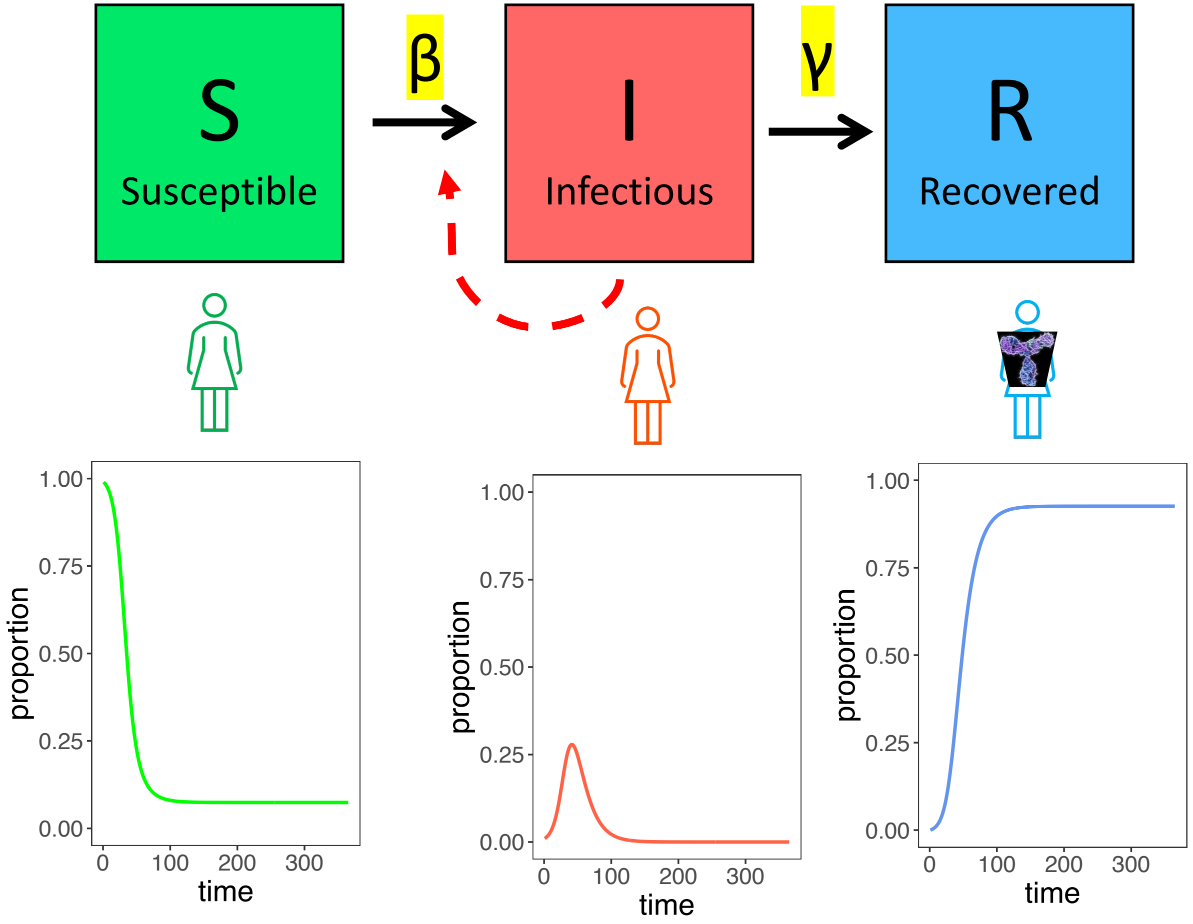

$$R_0 = \frac{\beta}{\gamma}$$

β = transmission rate

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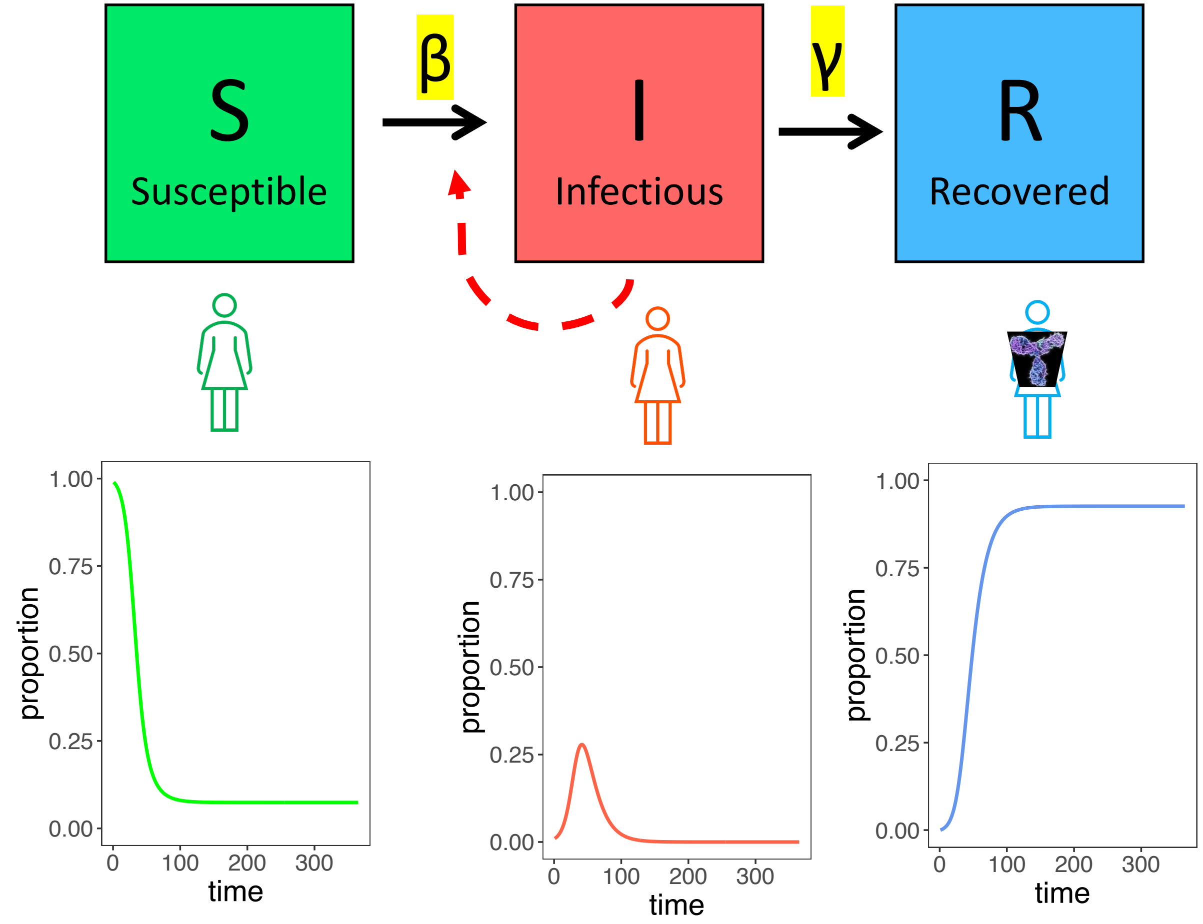

$$R_0 = \frac{\beta}{\gamma}$$

β } infections created

γ } infections lost

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

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

β = transmission rate

γ = recovery rate

R_o

- The **basic reproduction number** for a pathogen

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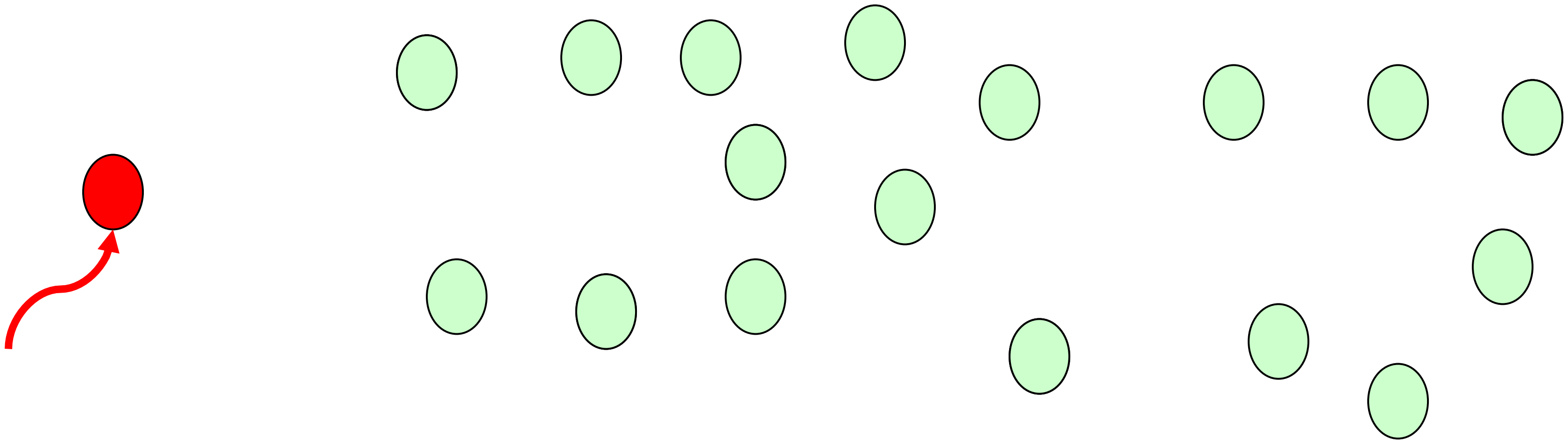

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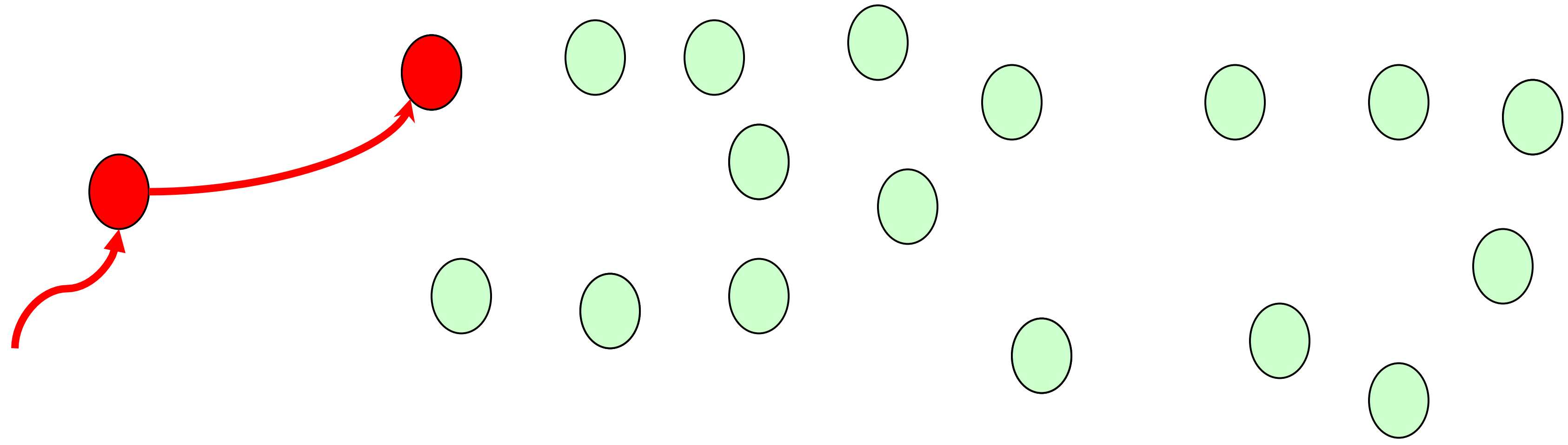

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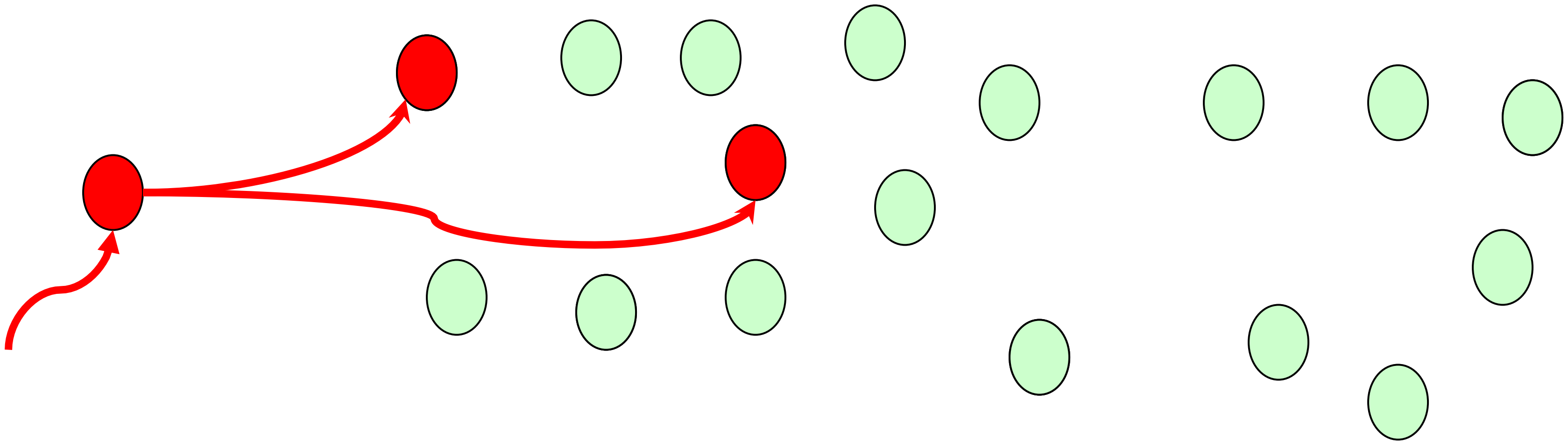

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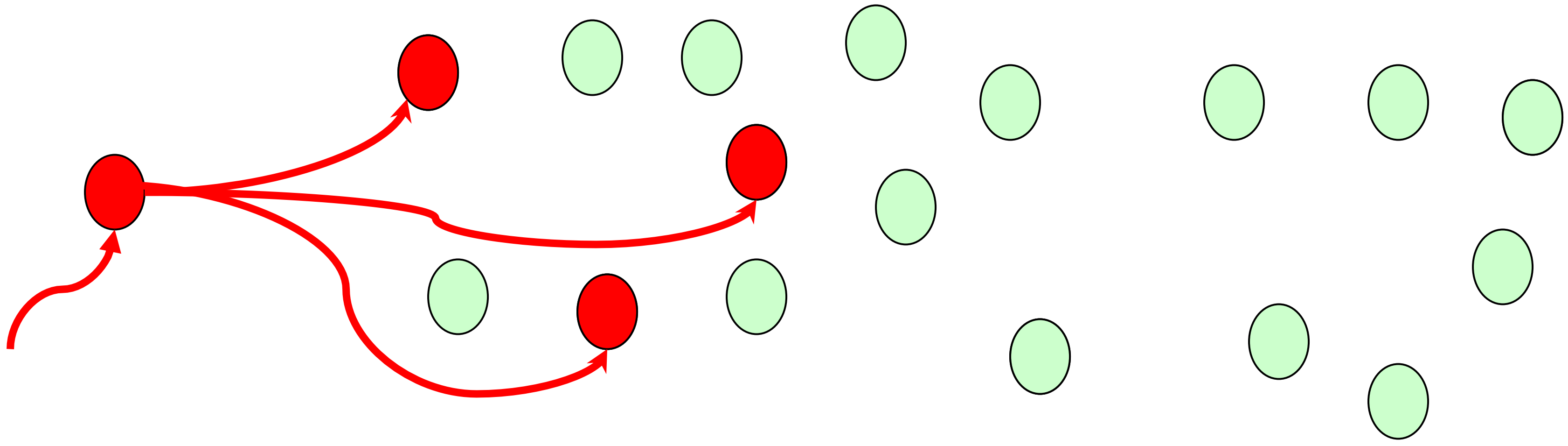

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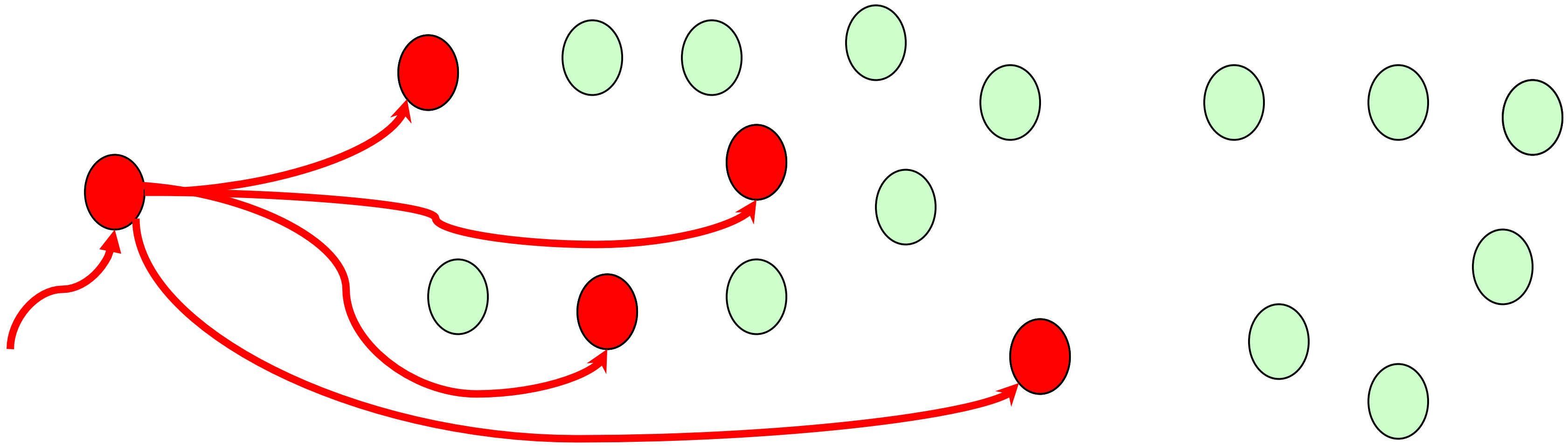

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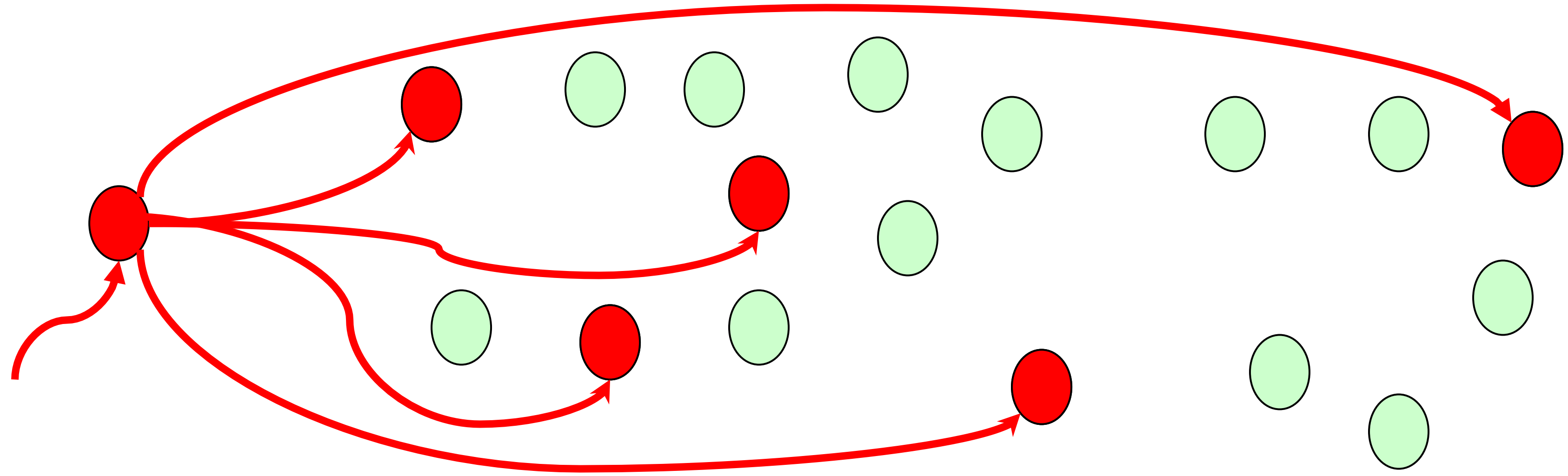

What is R_o?

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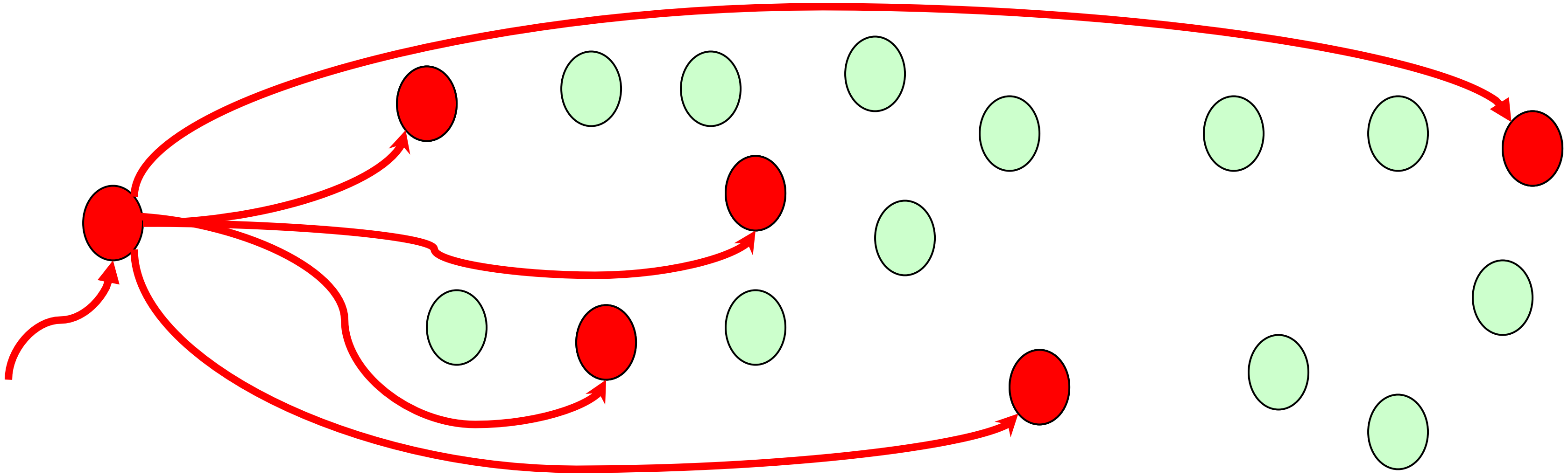


What is R_o?

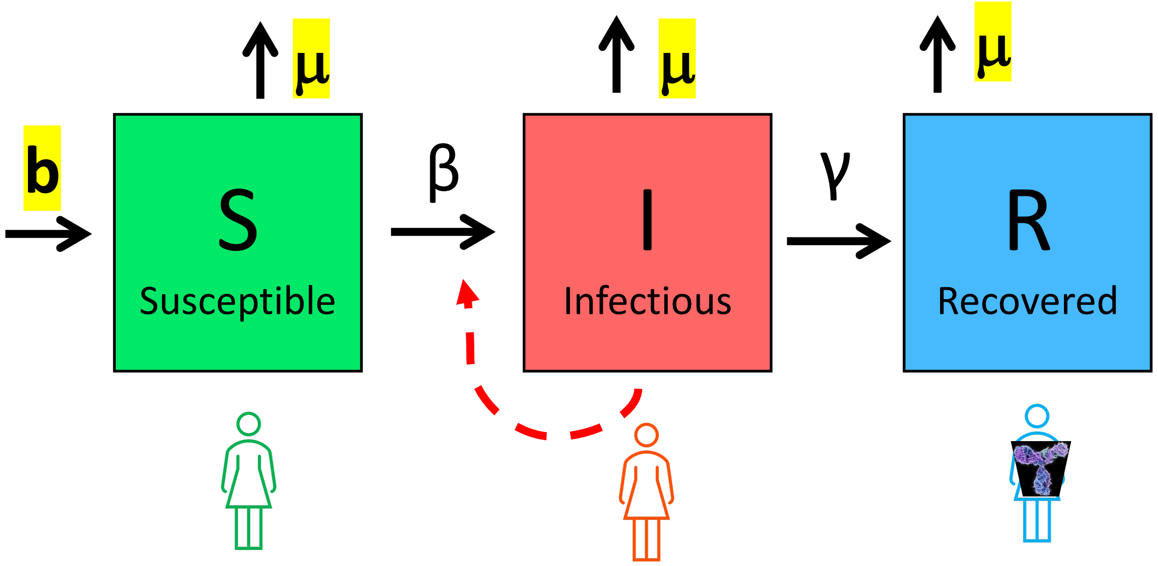
R_o = 5

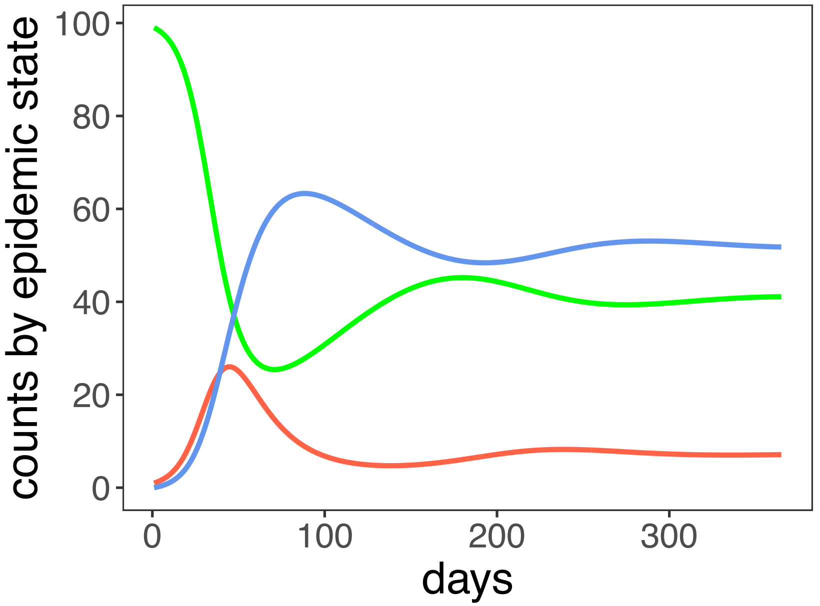
We can add realism to our models with births and deaths to **maintain** endemic pathogens.

$$\frac{dS}{dt} = bS - \beta SI - \mu S$$

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

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





b = birth rate
μ = death rate
β = transmission rate
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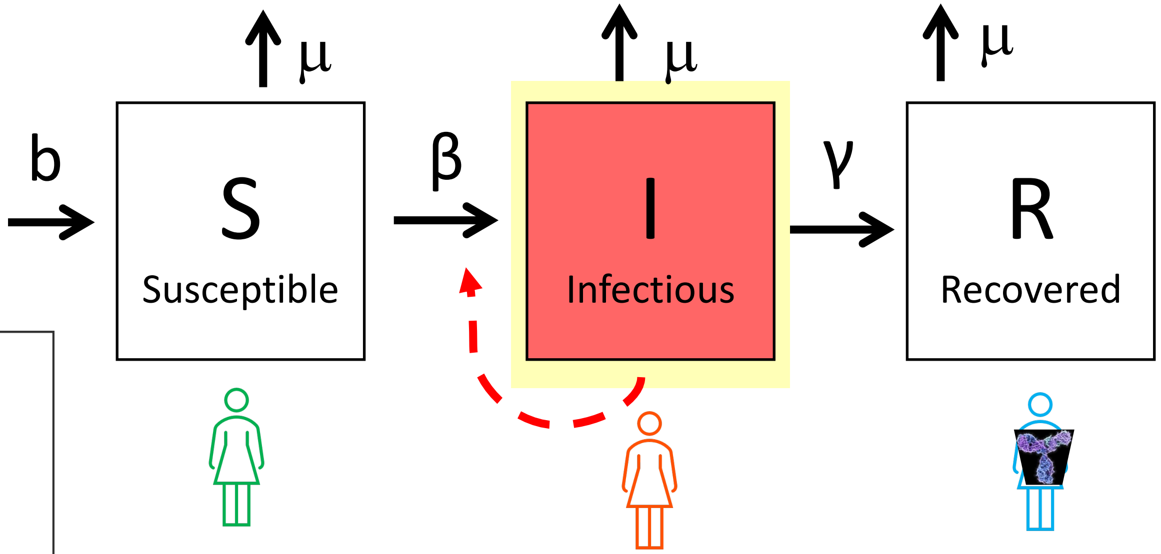
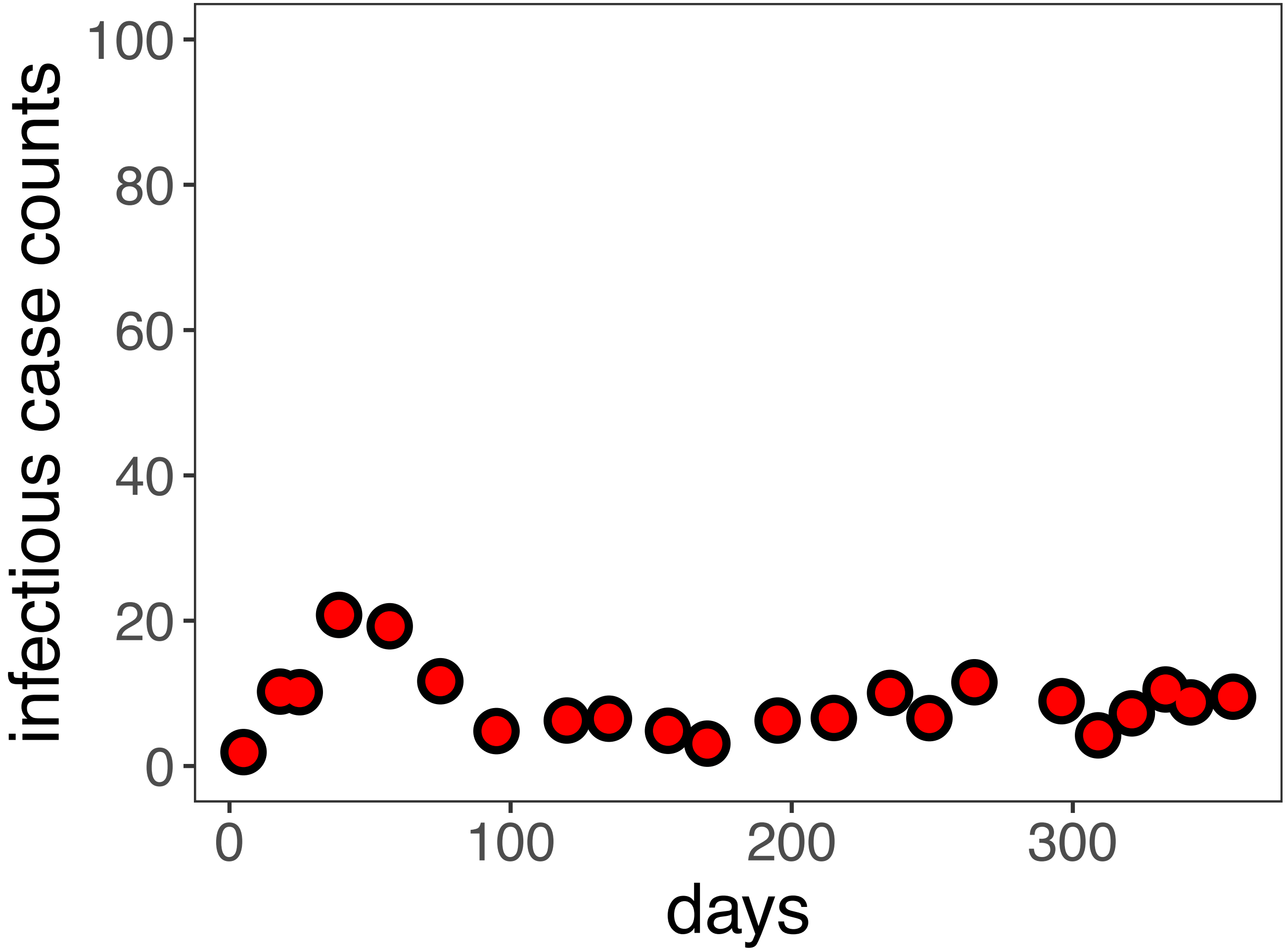
We can **estimate epidemic trajectories** by fitting SIR models to infectious case count data.





Anderson & May 1991. *Infectious Diseases of Humans*

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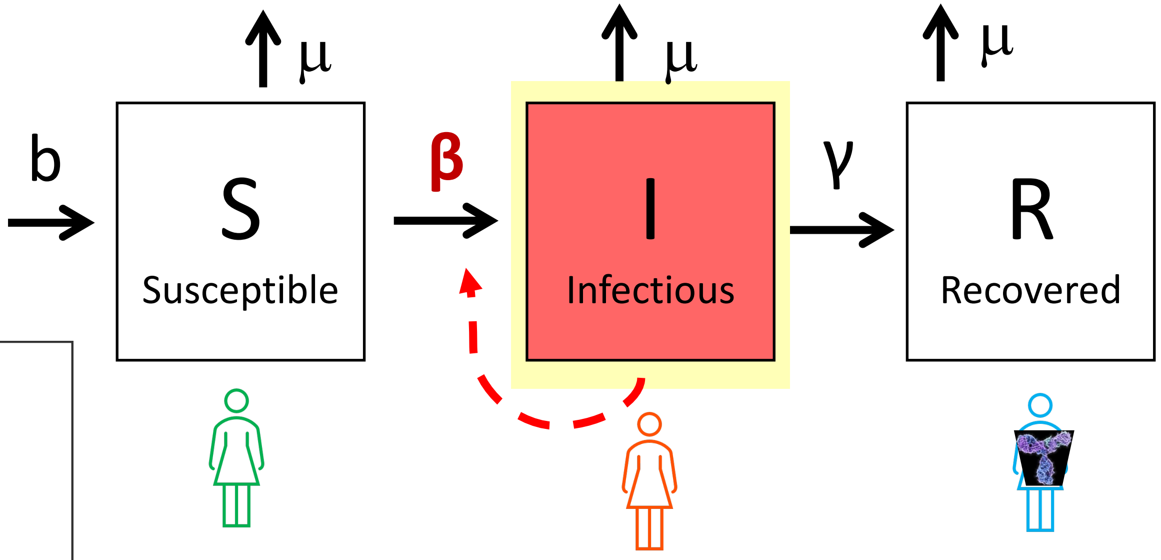

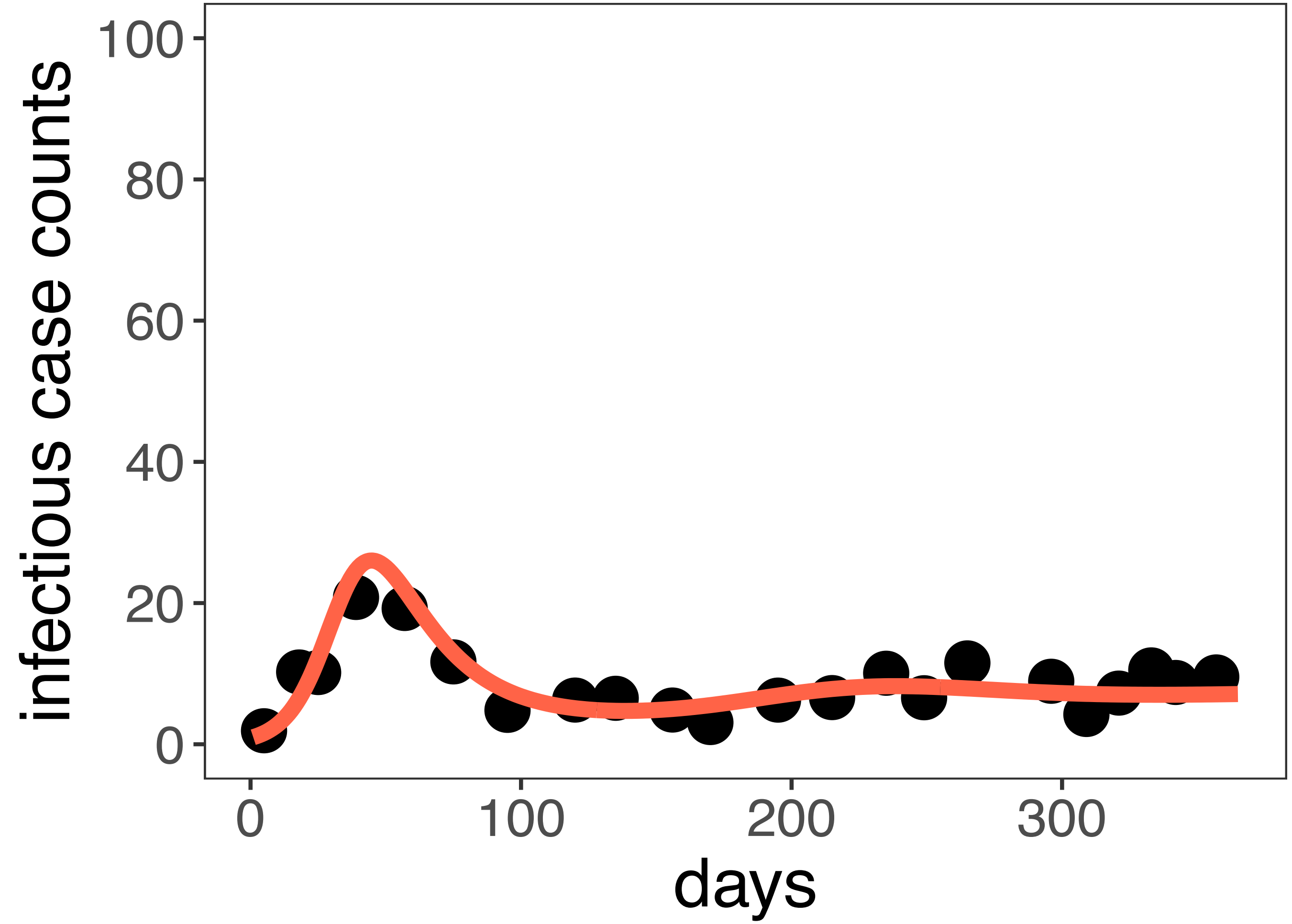

$$R_0 = \frac{\beta}{\gamma + \mu}$$

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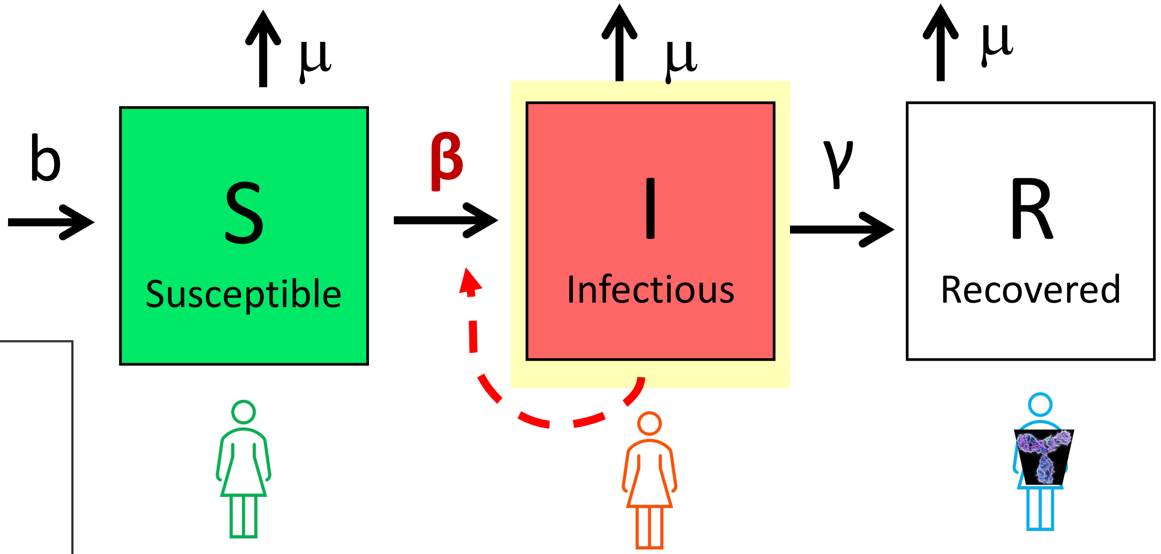

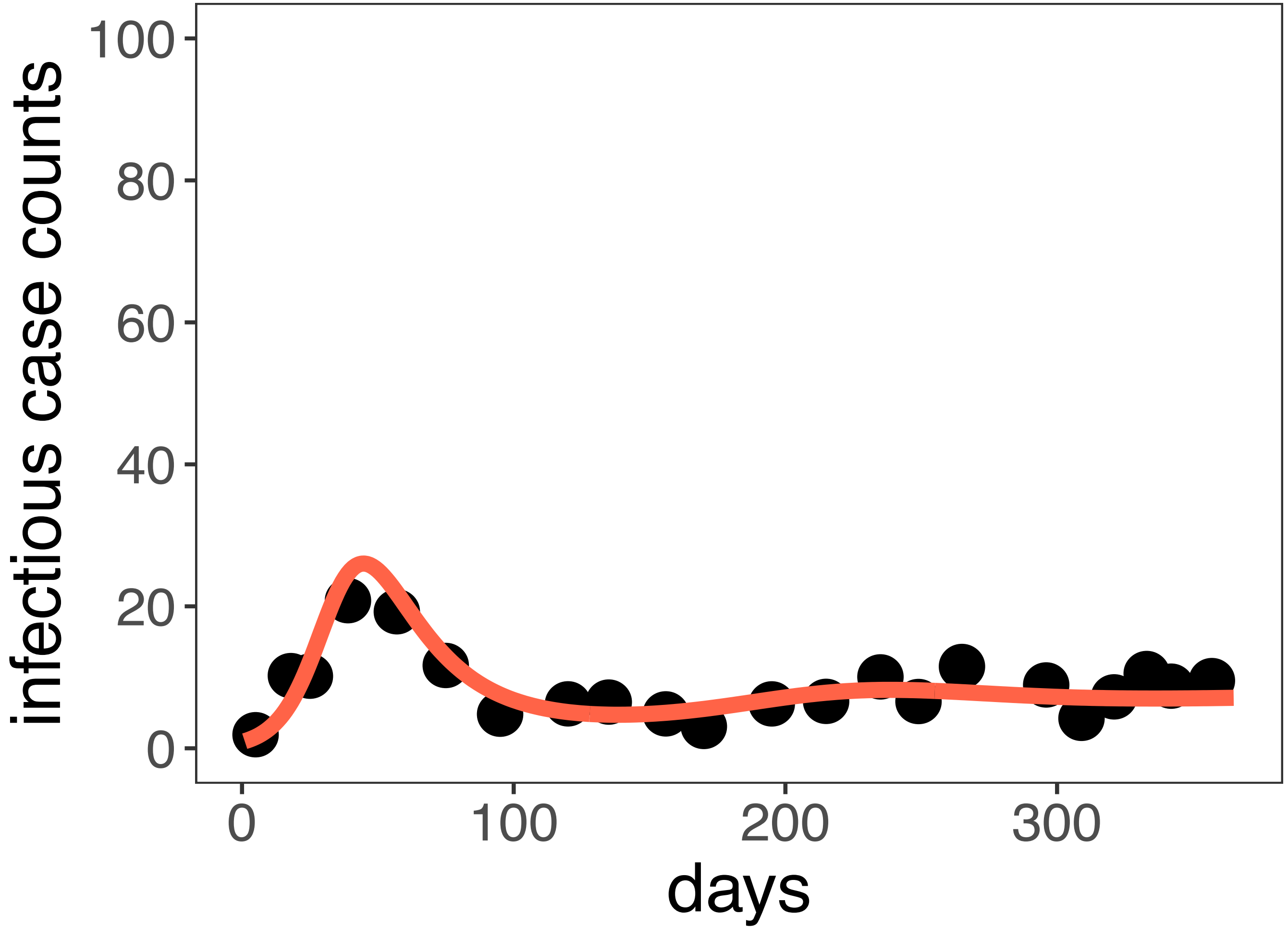

$$\mathbf{R_0} = \frac{\boldsymbol{\beta}}{\boldsymbol{\gamma} + \boldsymbol{\mu}}$$

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

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

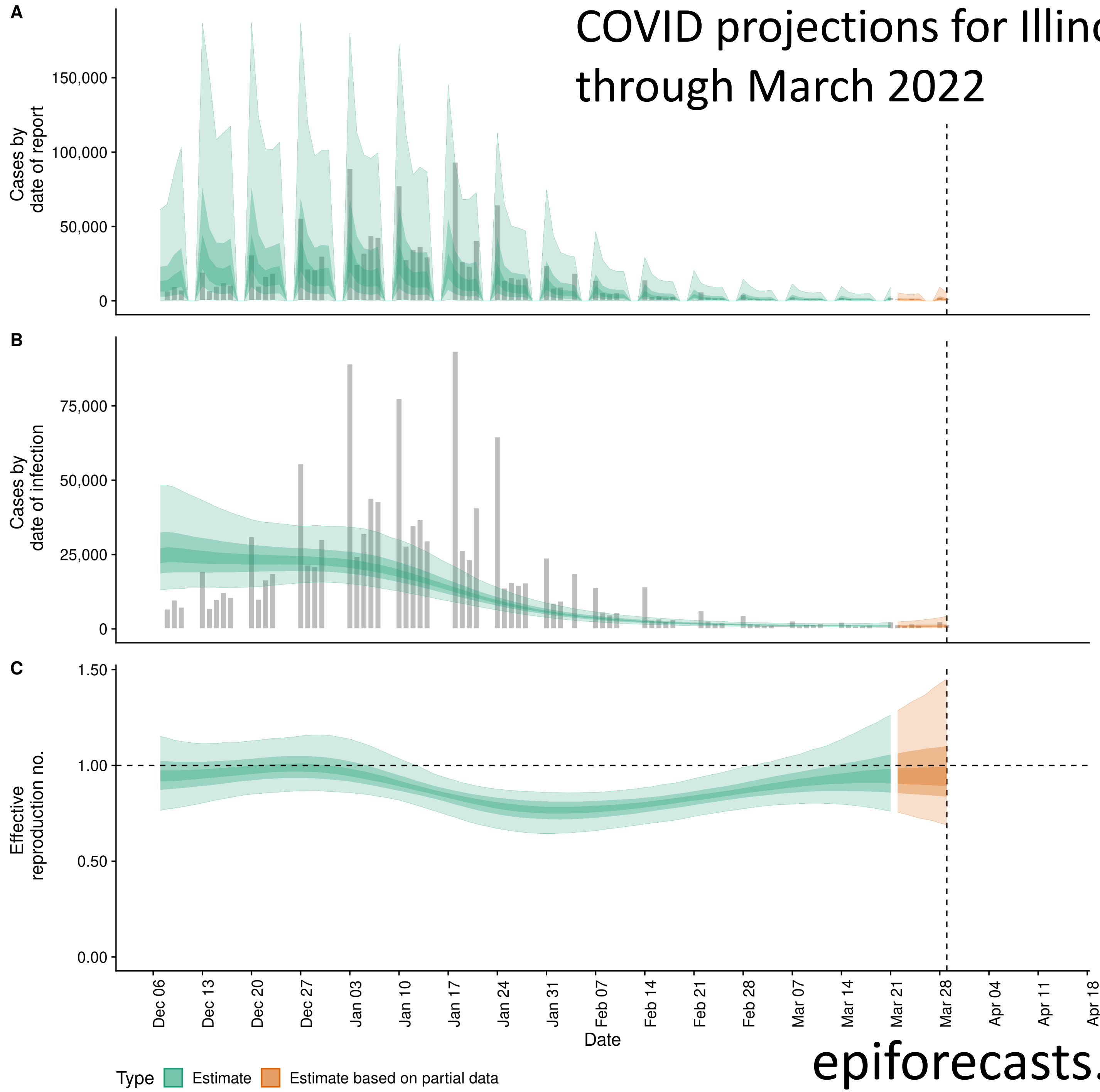
(epidemics spread @R_E>1 and decline @R_E<1)

Anderson & May 1991. *Infectious Diseases of Humans*

R_E OR R_t

- The **effective reproduction number** for a pathogen

COVID projections for Illinois through March 2022



epiforecasts.io

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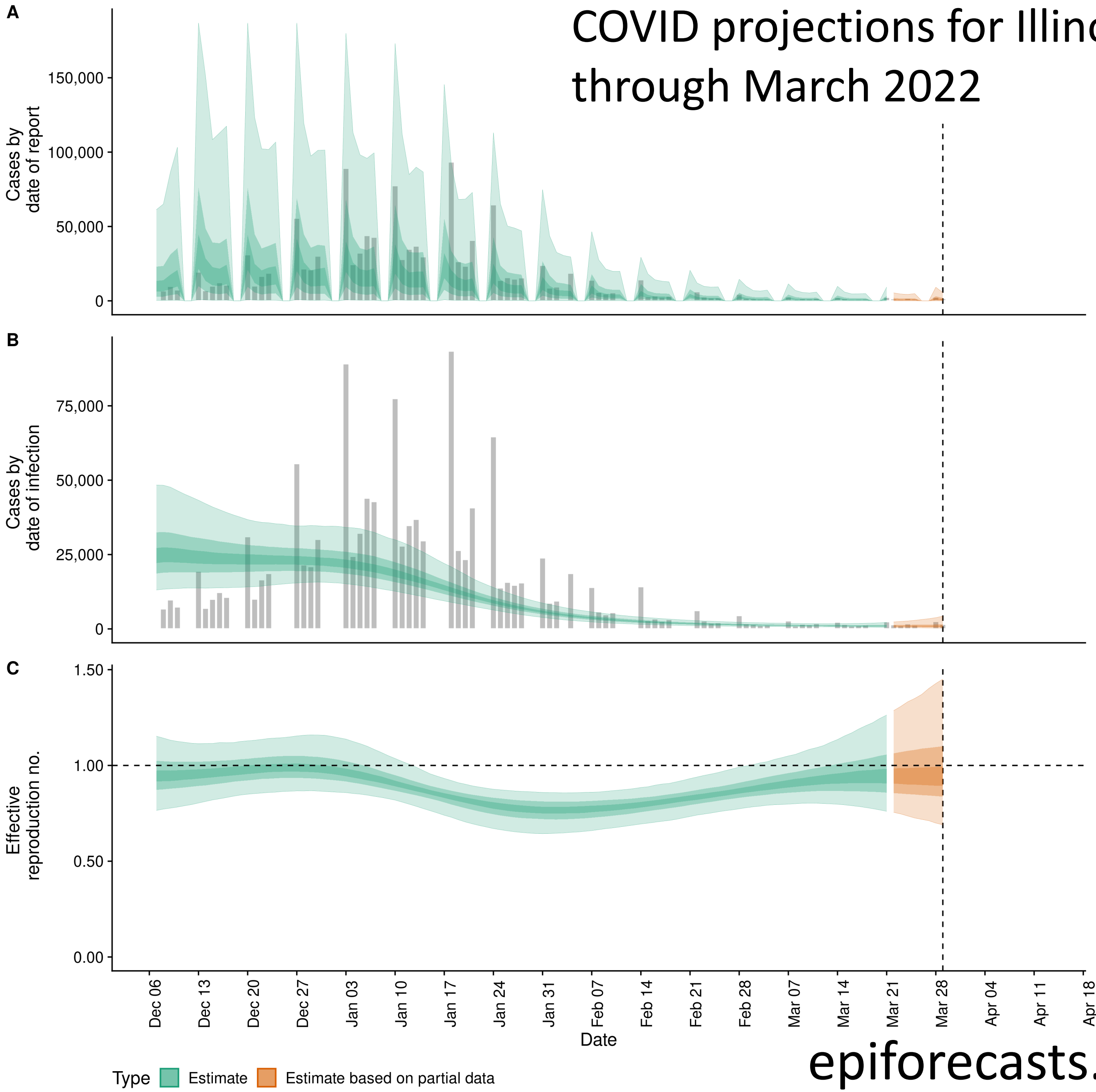

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$$R_E = R_0 \frac{S}{N}$$

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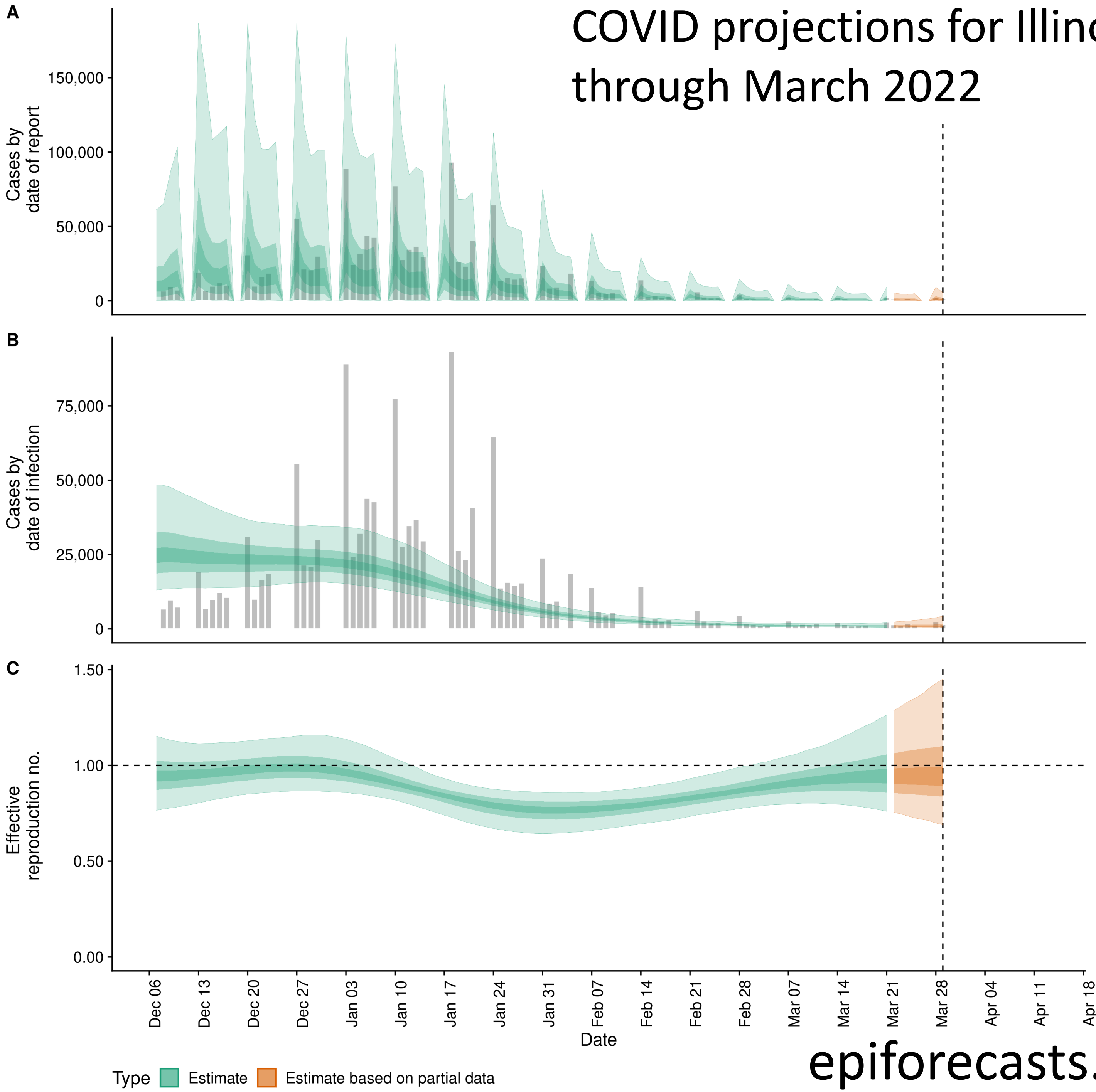

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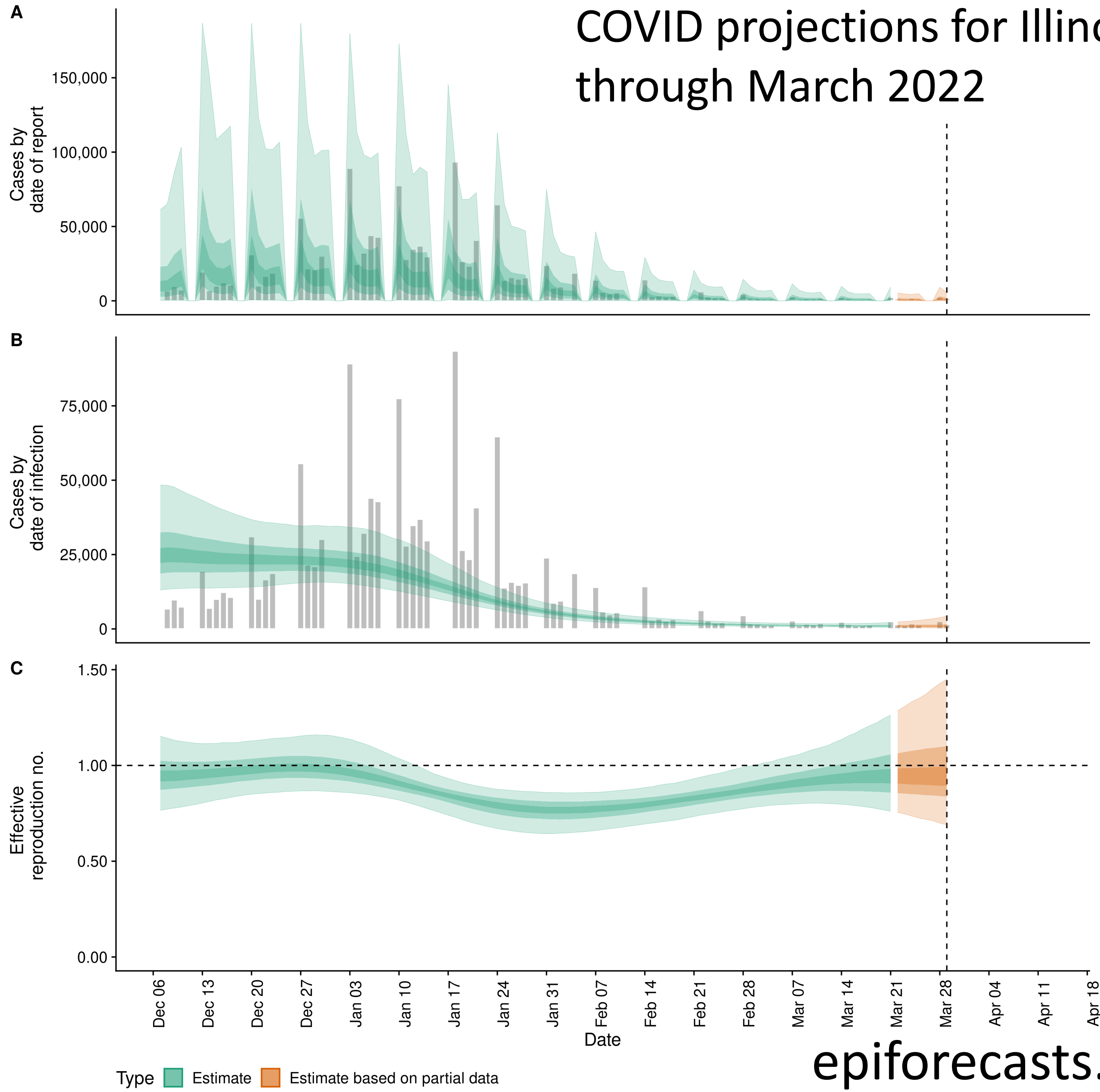
- Gives a realistic pulse of the current pace of the epidemic!

COVID projections for Illinois through March 2022



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

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

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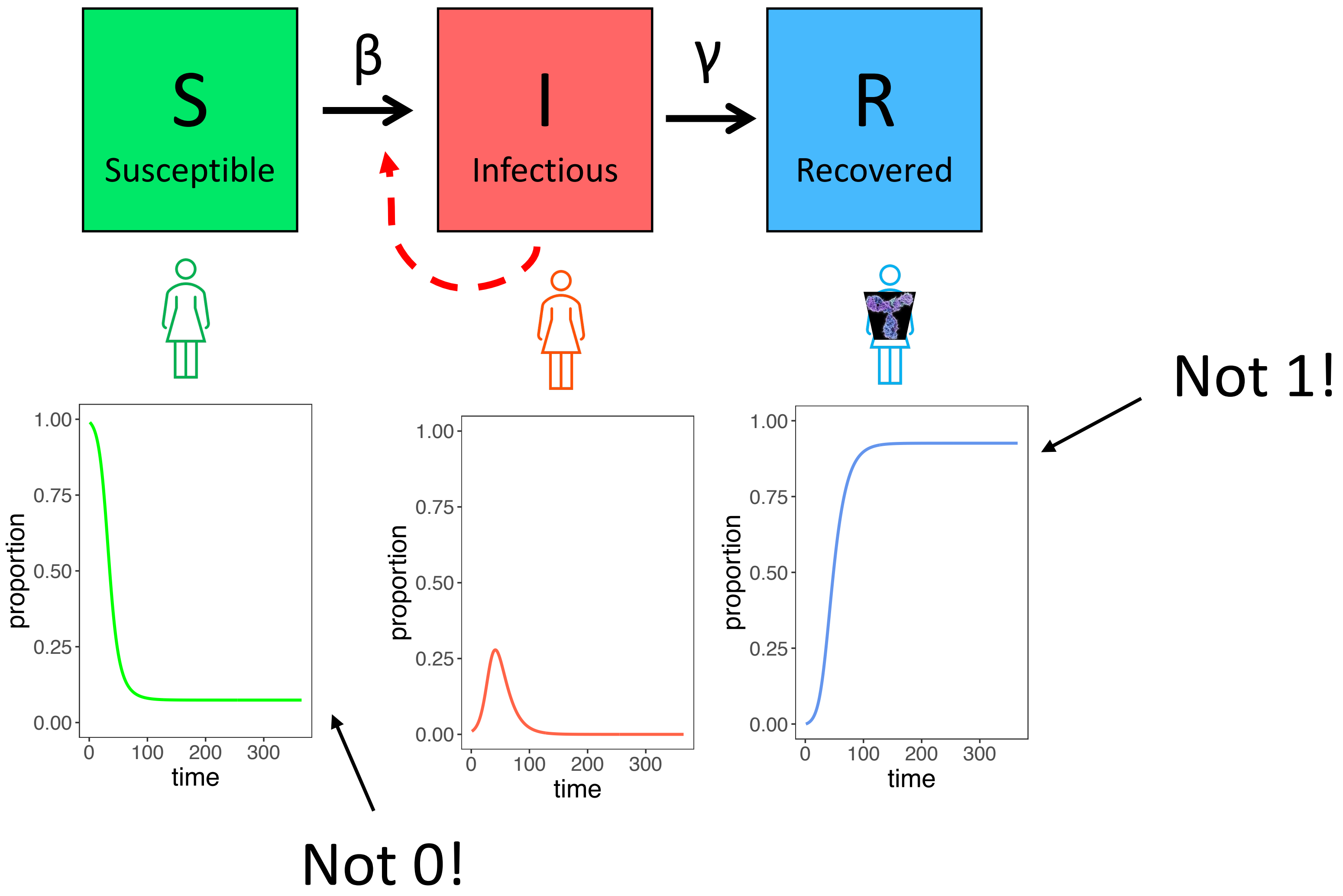
$$\frac{dR}{dt} = \gamma R$$



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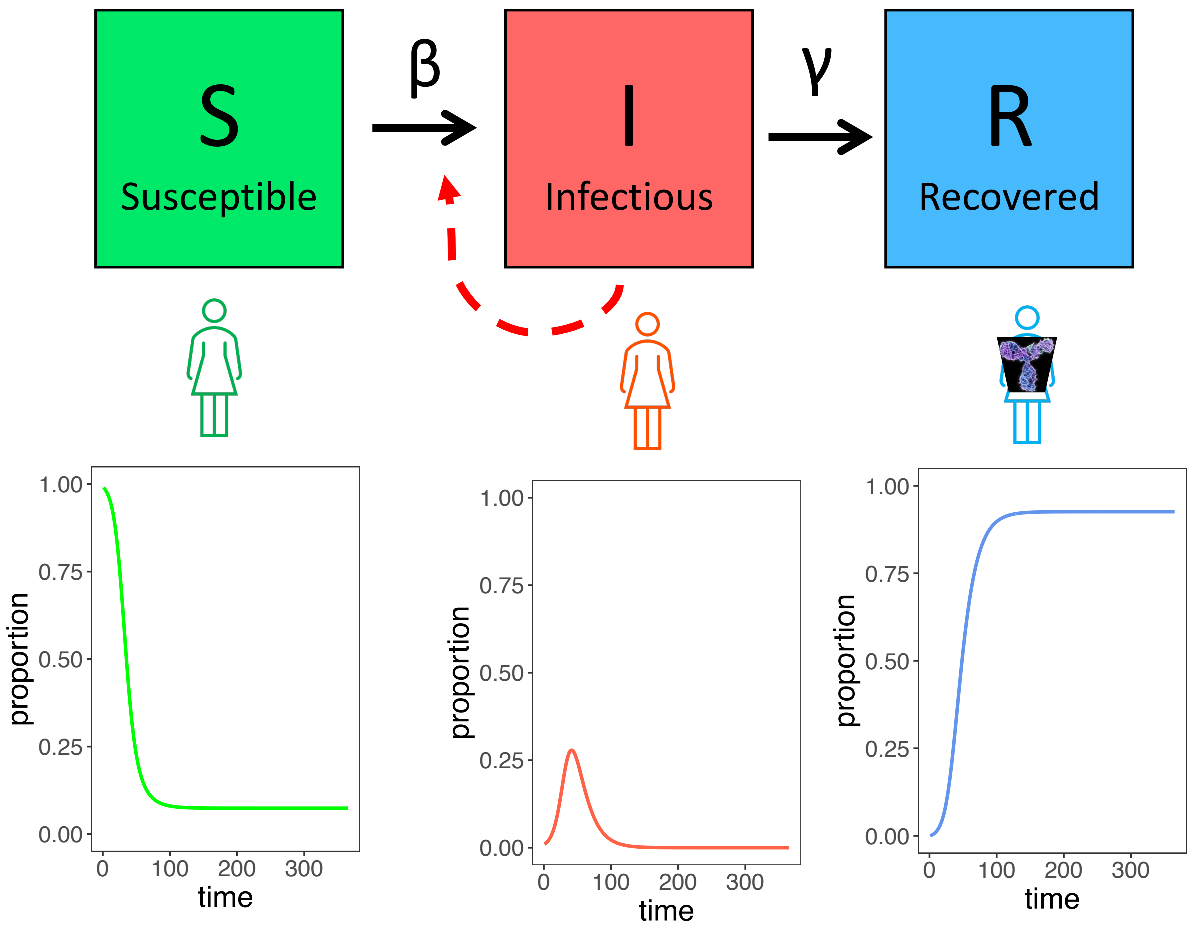

- The epidemic does not end because all individuals have been infected and have either died or recovered.
- Rather, finding new susceptibles becomes more difficult and $R_E < 1$

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

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R_E interventions

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Mathematics of Vaccination

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

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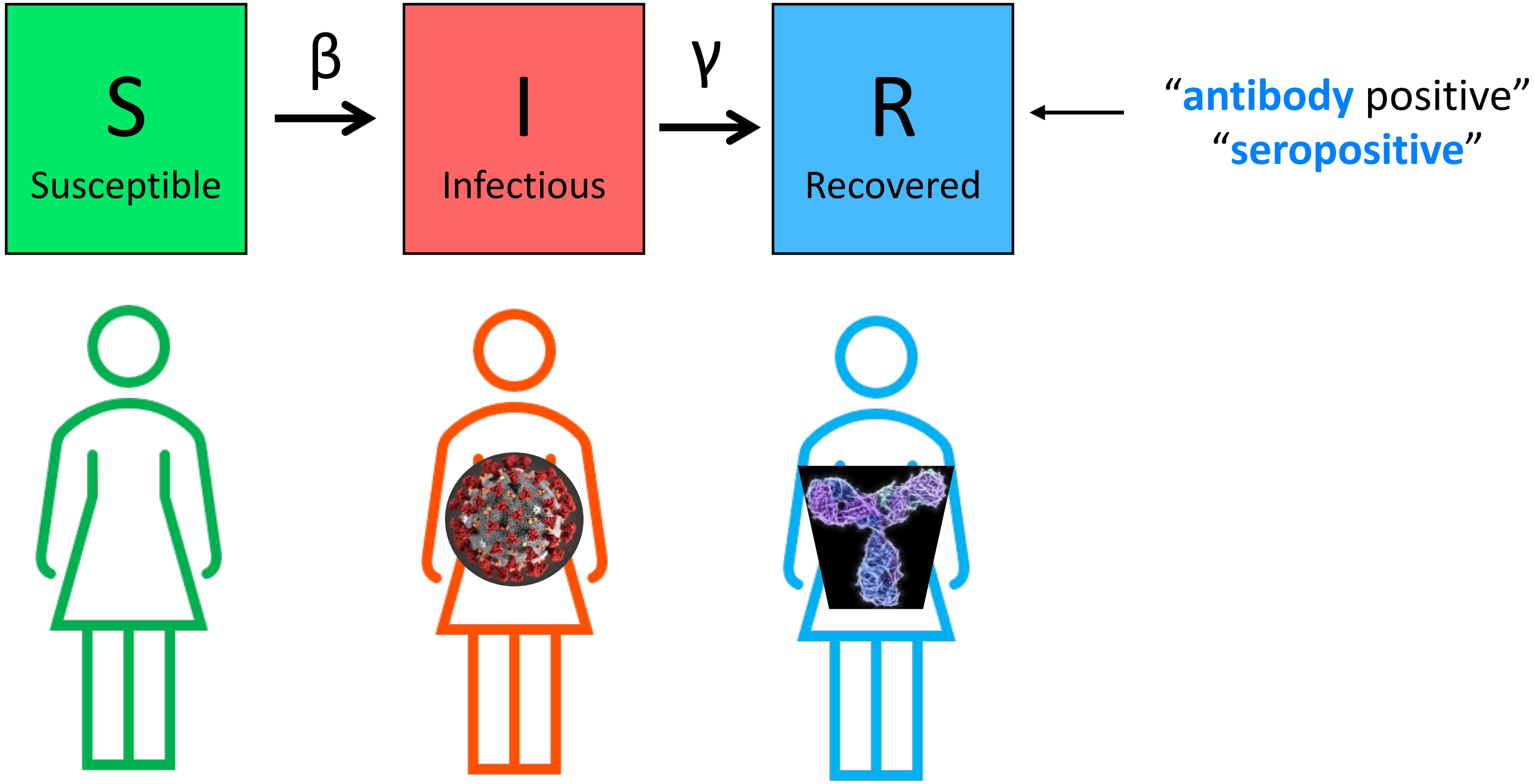

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Mathematics of Vaccination

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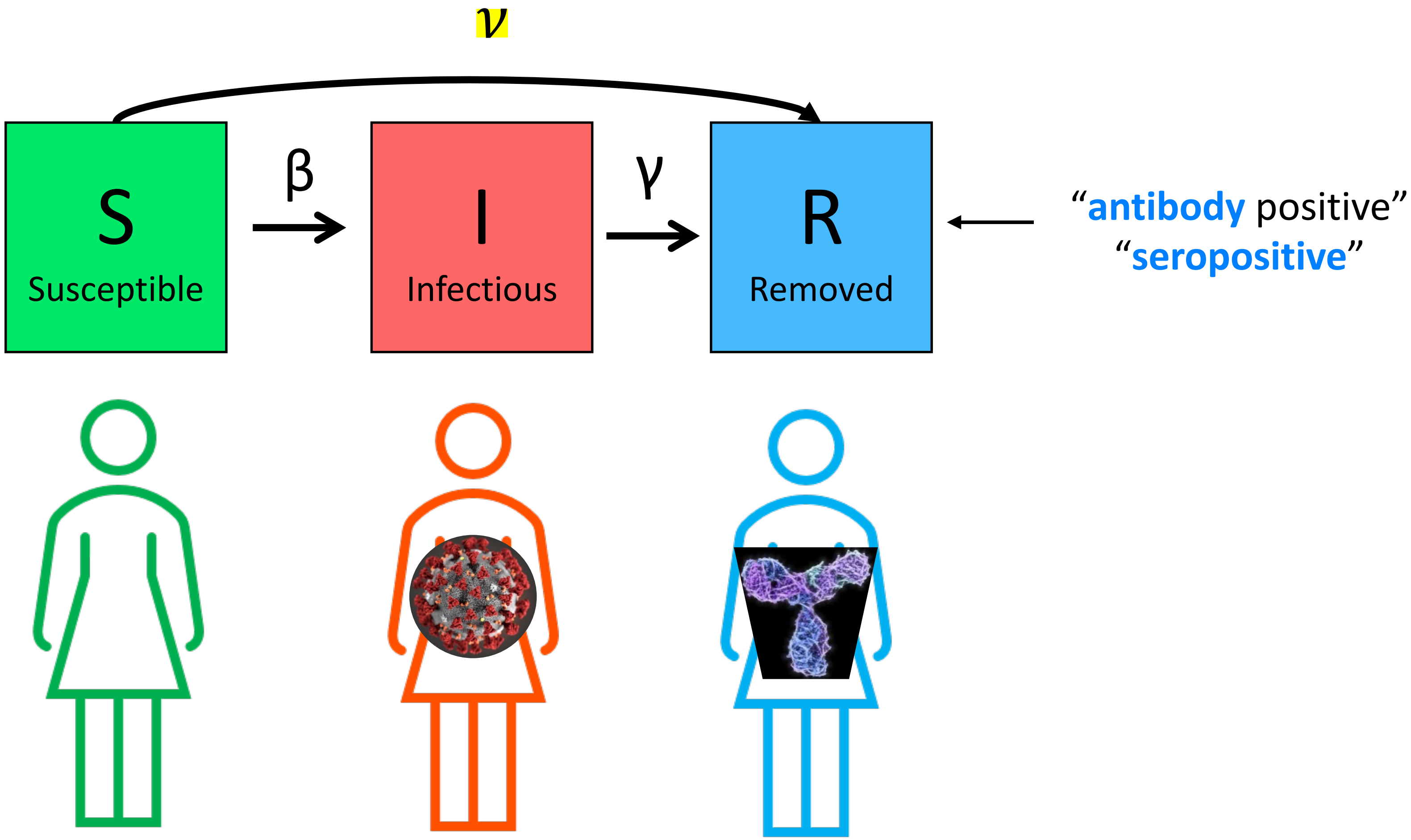
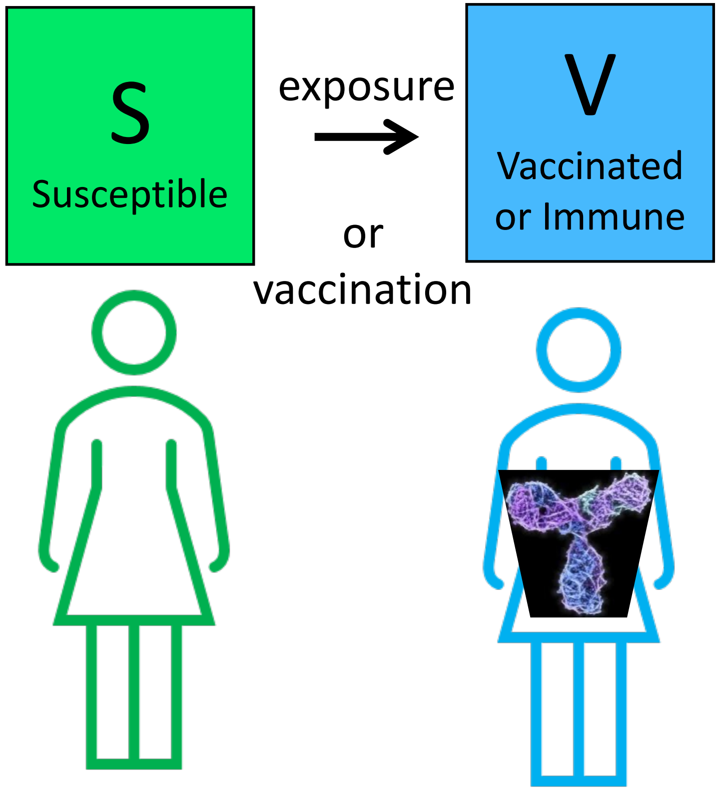


β = transmission rate
γ = recovery rate
ν = vaccination rate

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)



Mathematics of Vaccination

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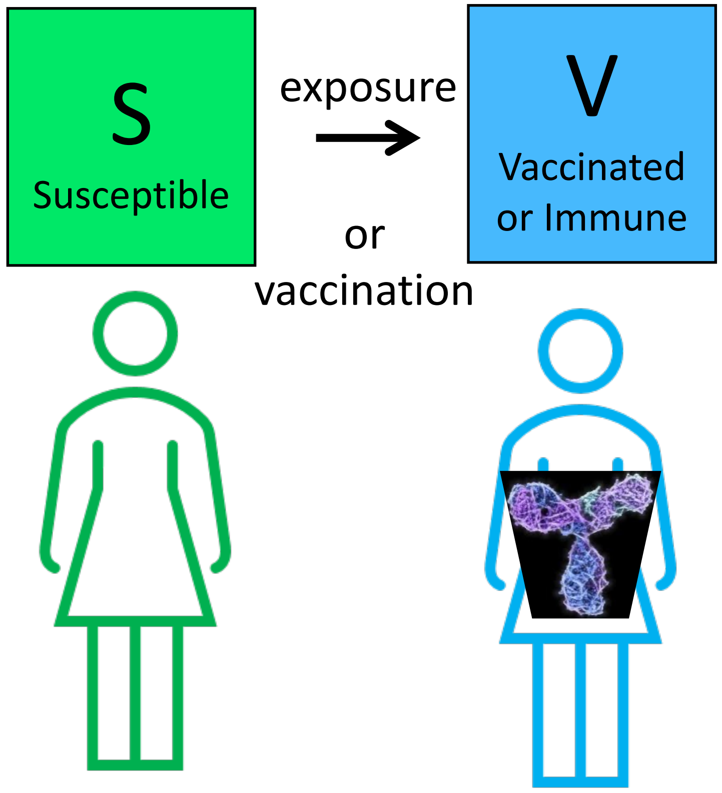

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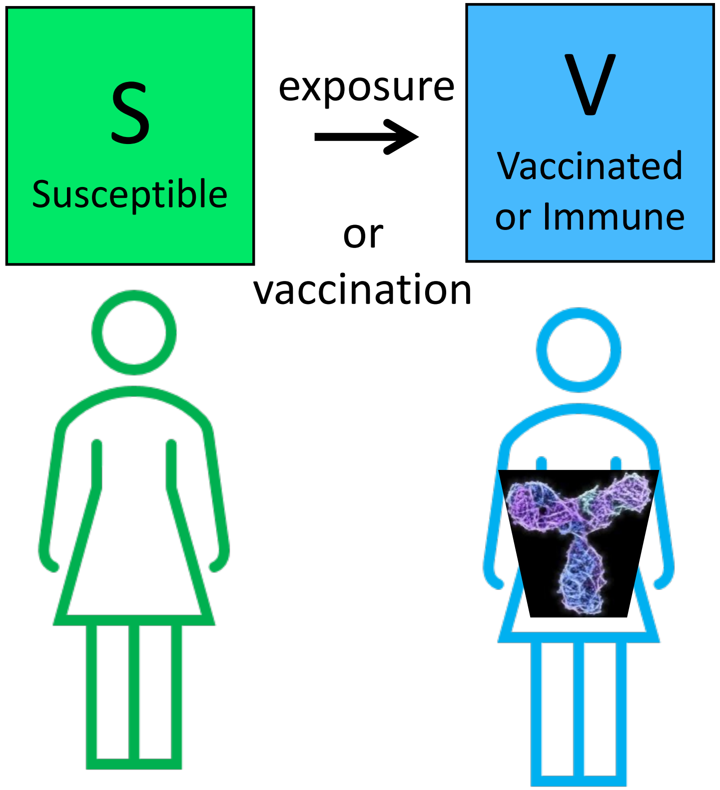

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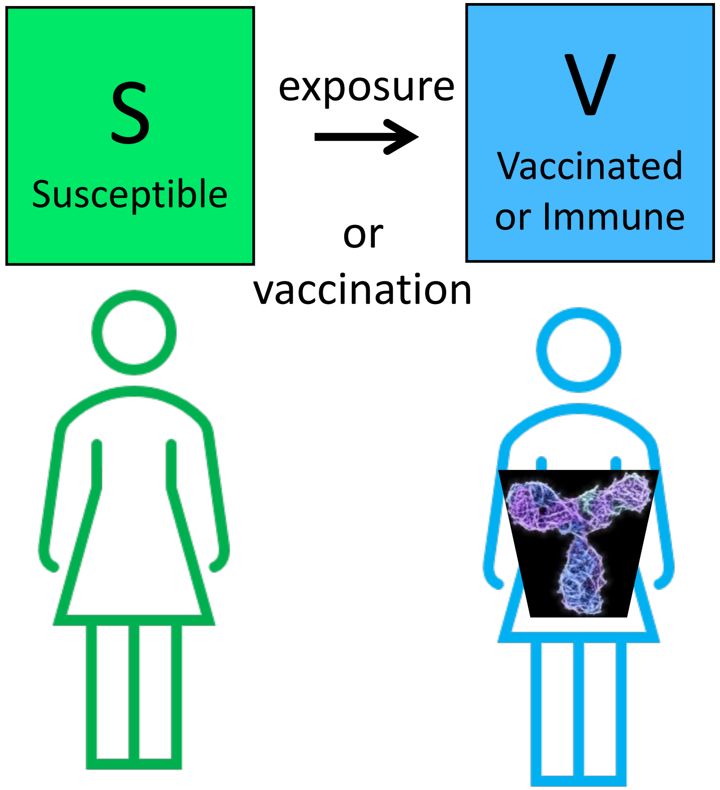

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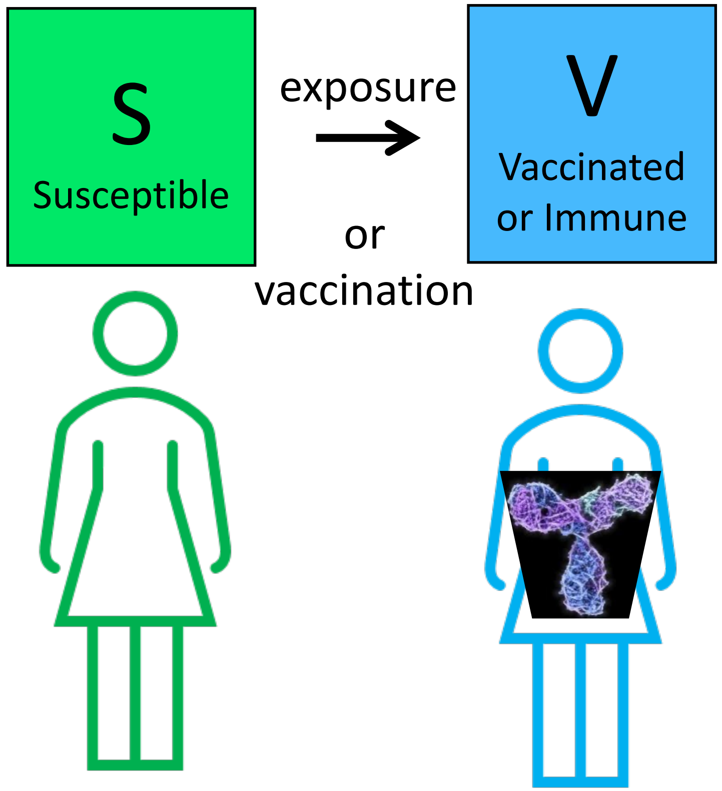

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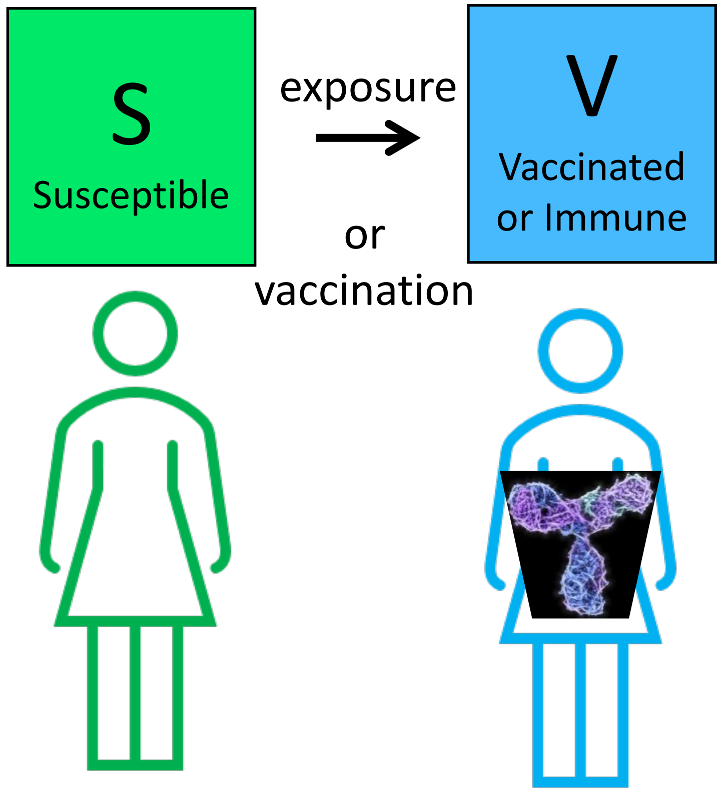

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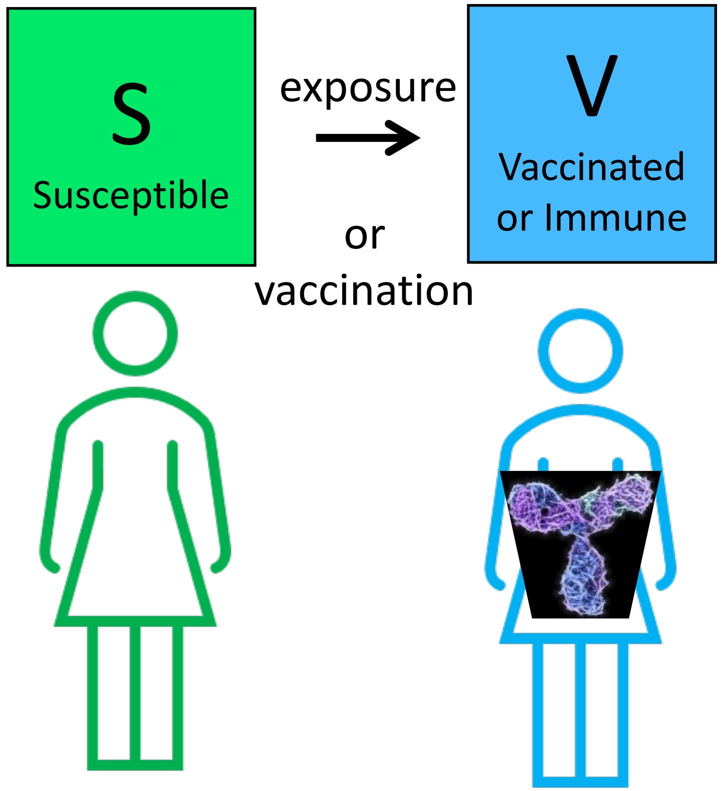
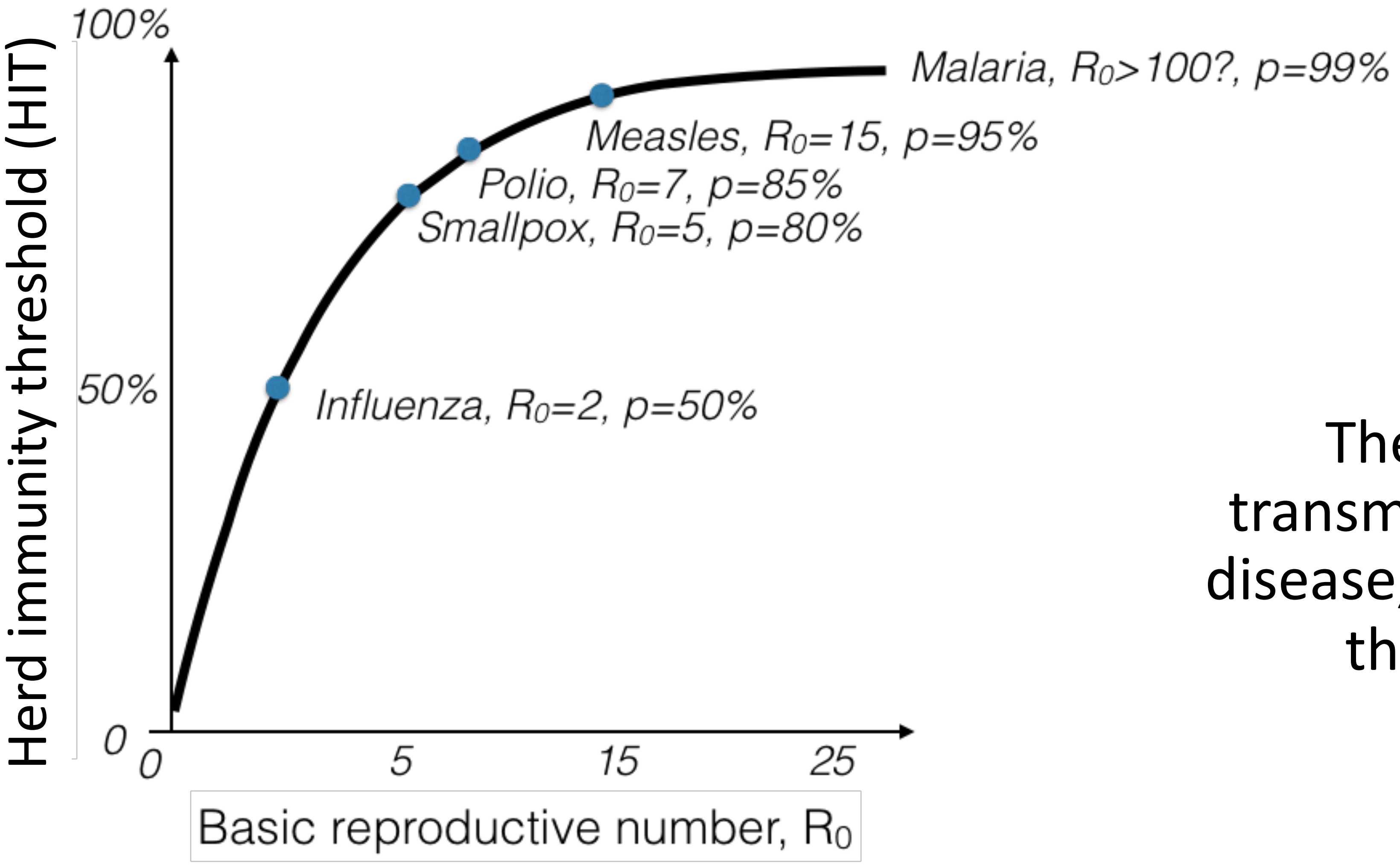
- **Even susceptibles will not become infected because the disease will not spread ($R_E < 1$).**

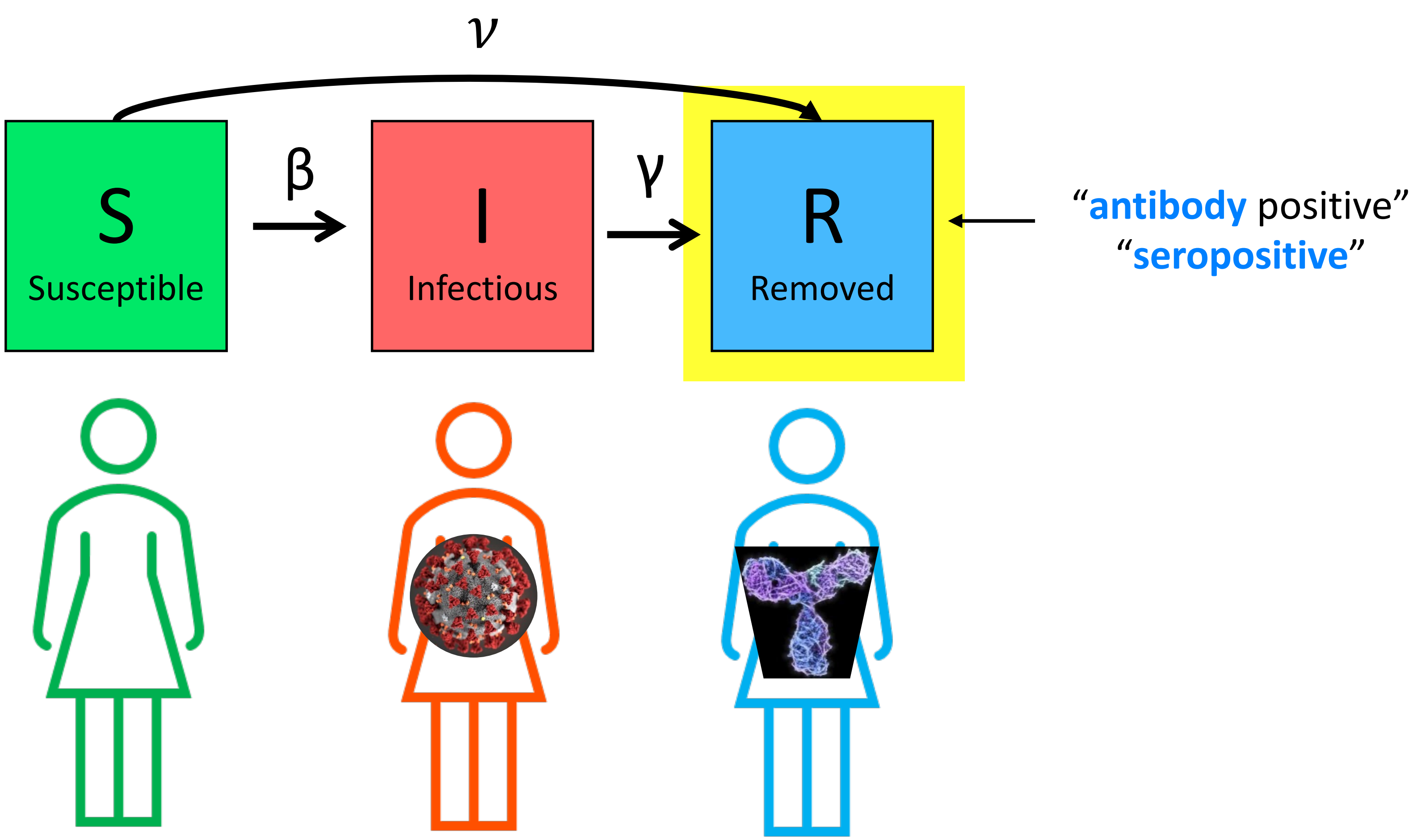


R_0 and the Herd Immunity Threshold



The more transmissible the disease, the higher the HIT!

Vaccination stems from a long history



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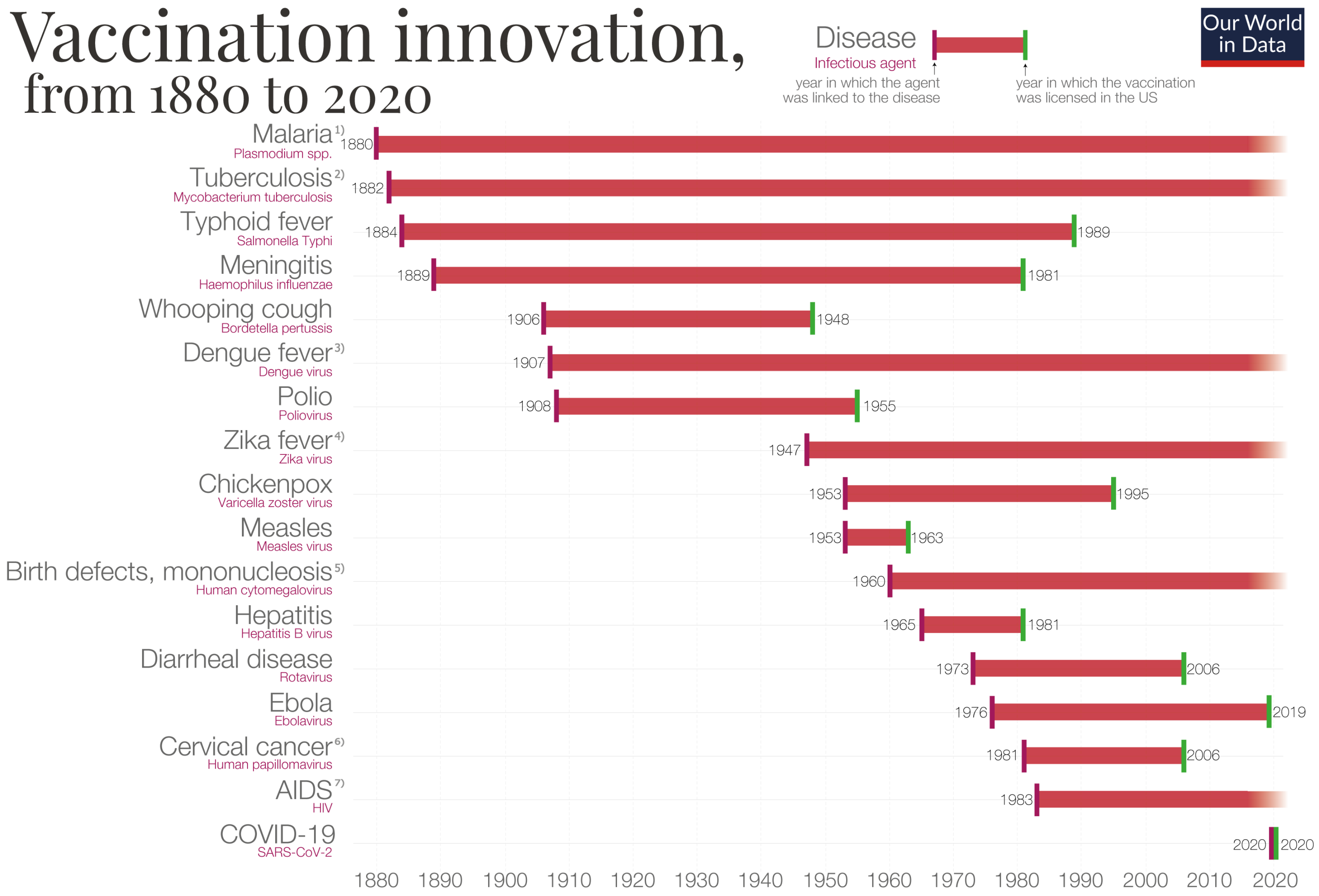
- Smallpox was globally eradicated in 1977, following a massive international campaign
- Today, we are seeing enhanced transmission of monkeypox partly resulting from a lack of circulating immunity to closely related smallpox

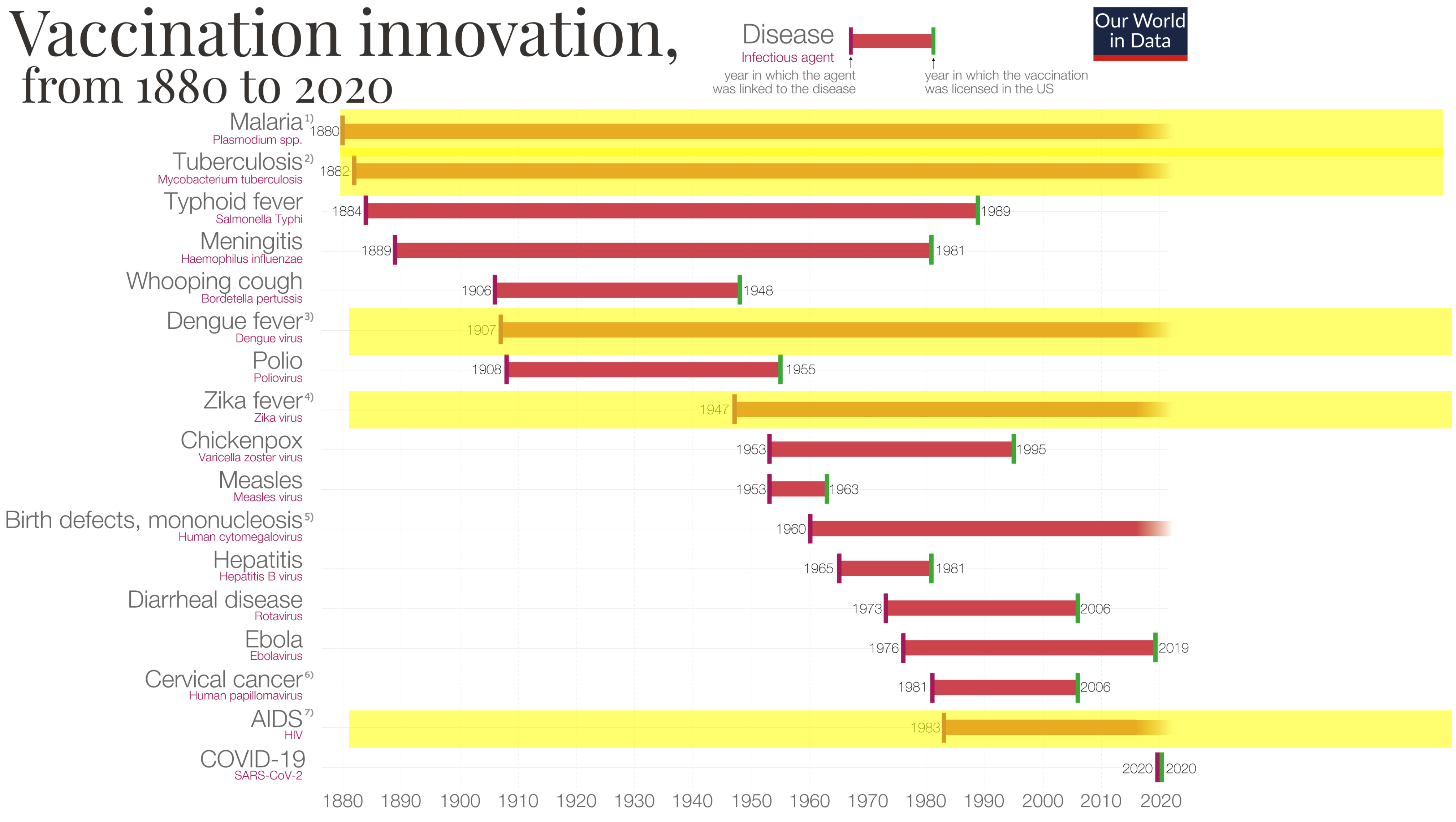
Only two global vaccination success stories

The pace of vaccine development has accelerated drastically



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

But we still lack vaccines for several important diseases.



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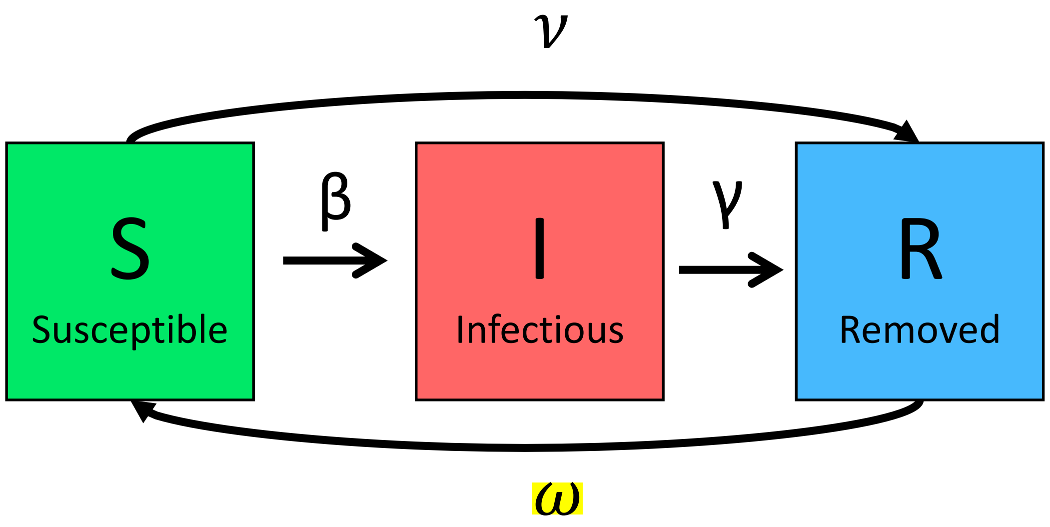
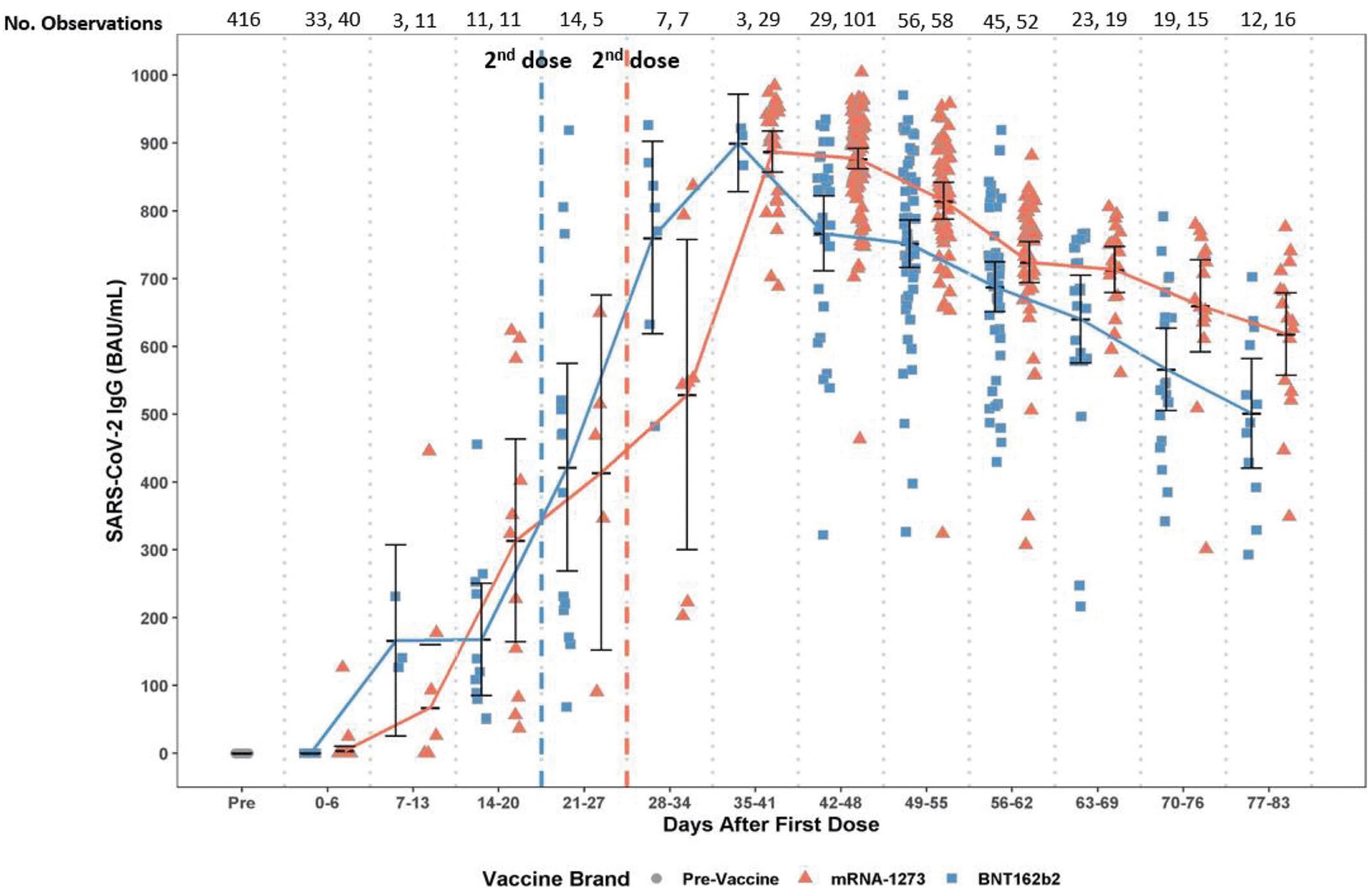
Challenges to Vaccination

- 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*

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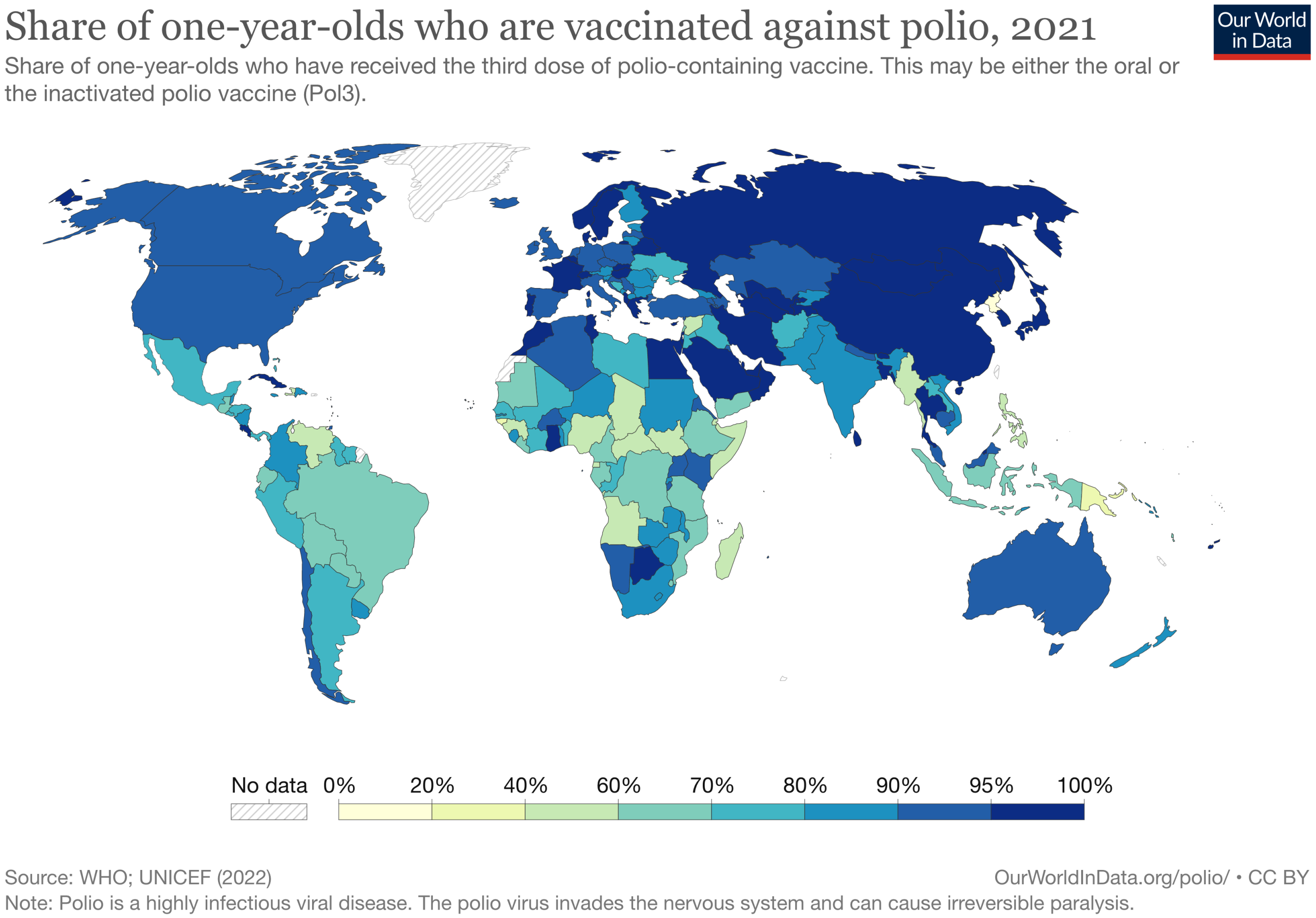

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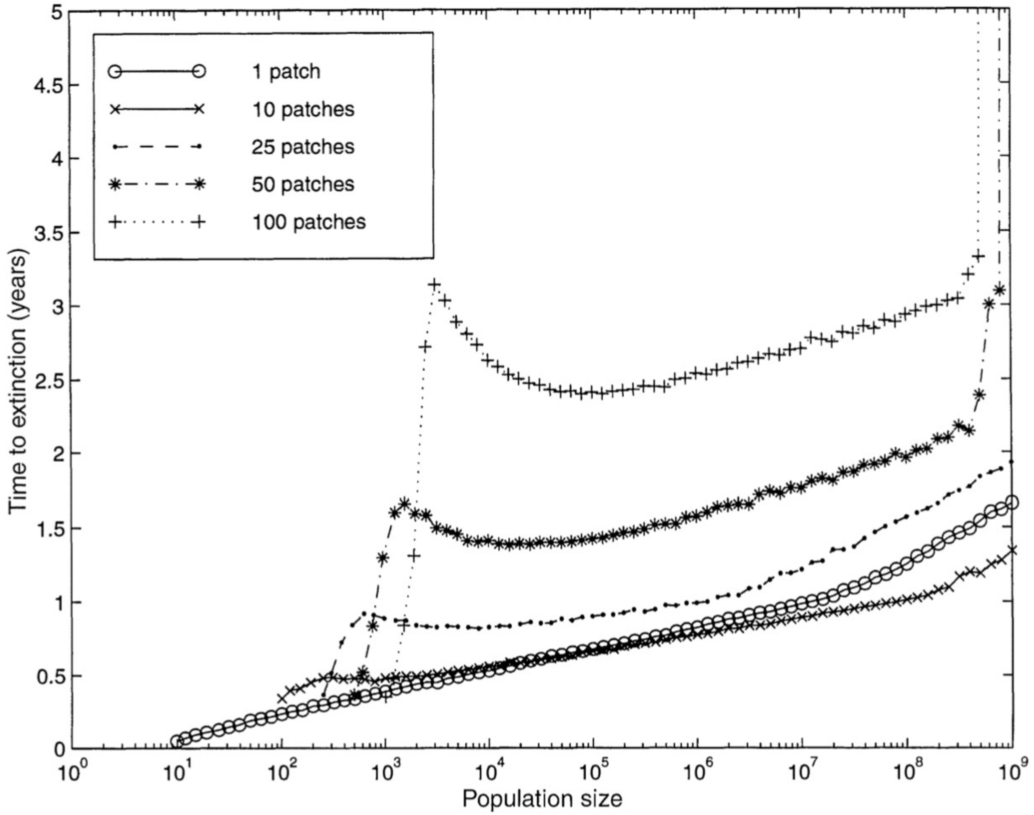
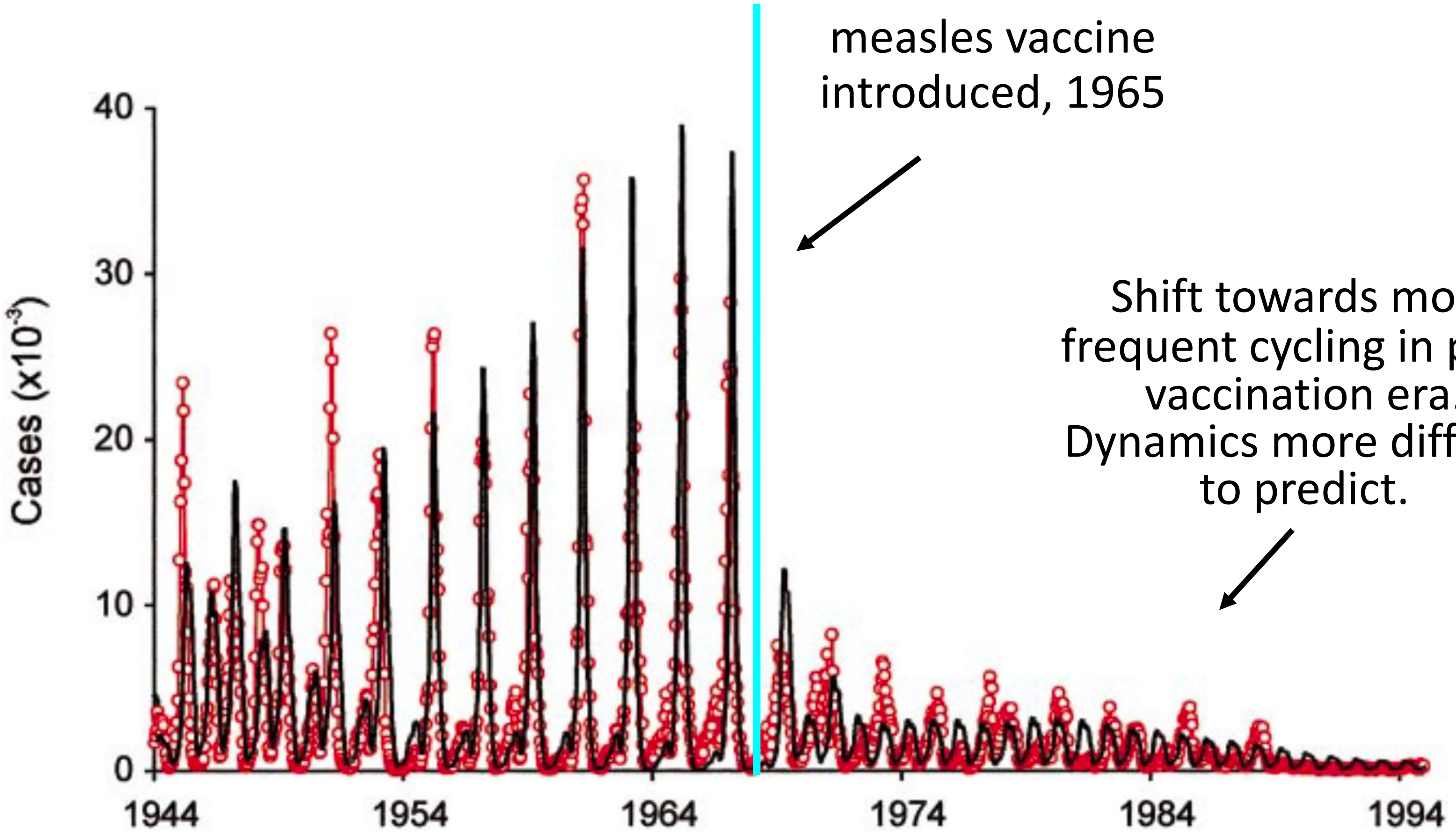


Swinton et al. 1998
Bartlett 1957

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



measles vaccine introduced, 1965

Shift towards more frequent cycling in post-vaccination era. Dynamics more difficult to predict.

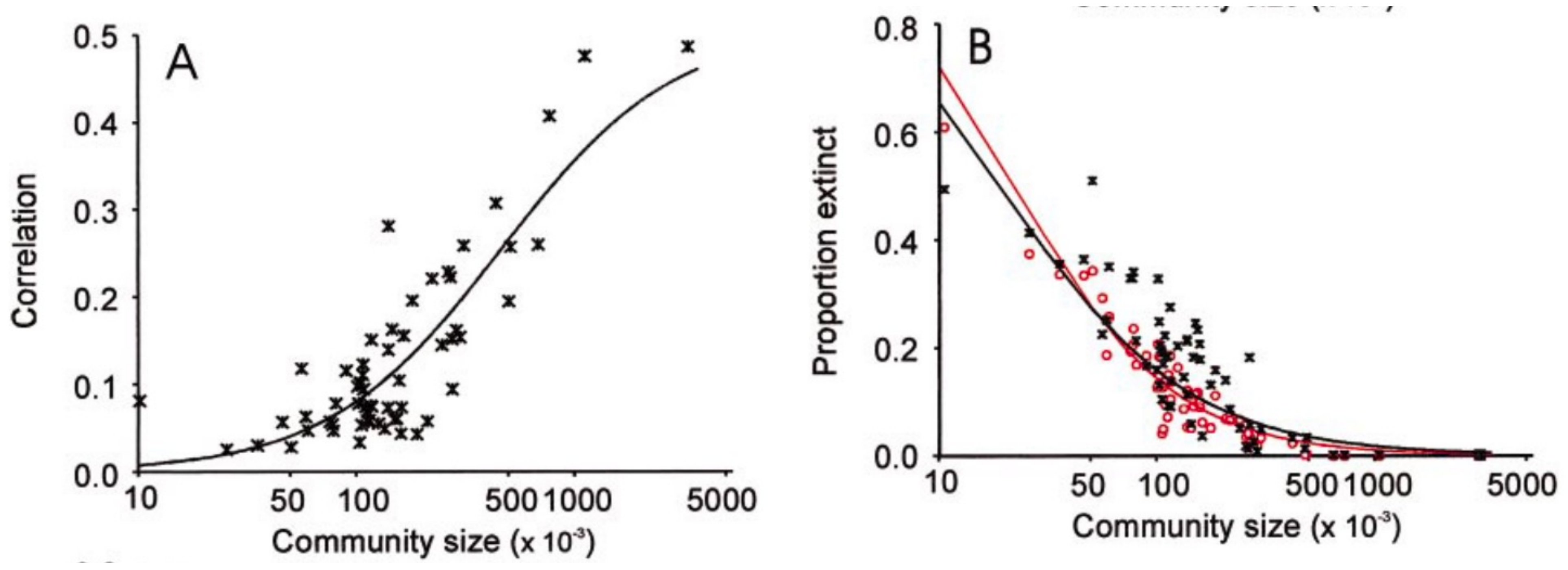
Biennial cycles in pre-vaccine era measles cases in London

model

data

Grenfell et al. 2002. *Ecological Monographs*

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



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