Wk9-Lect8-Mar4

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What's your favorite parasite or pathogen so far?

A. Measles

0%

B. Malaria

0%

SEE MORE

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Fundamentals of Ecology

Week 9, Ecology Lecture 8

Cara Brook

March 3, 2025

Office hours: On ZOOM
Thursday, March 6, 2025
4-5pm
I will email out a link!

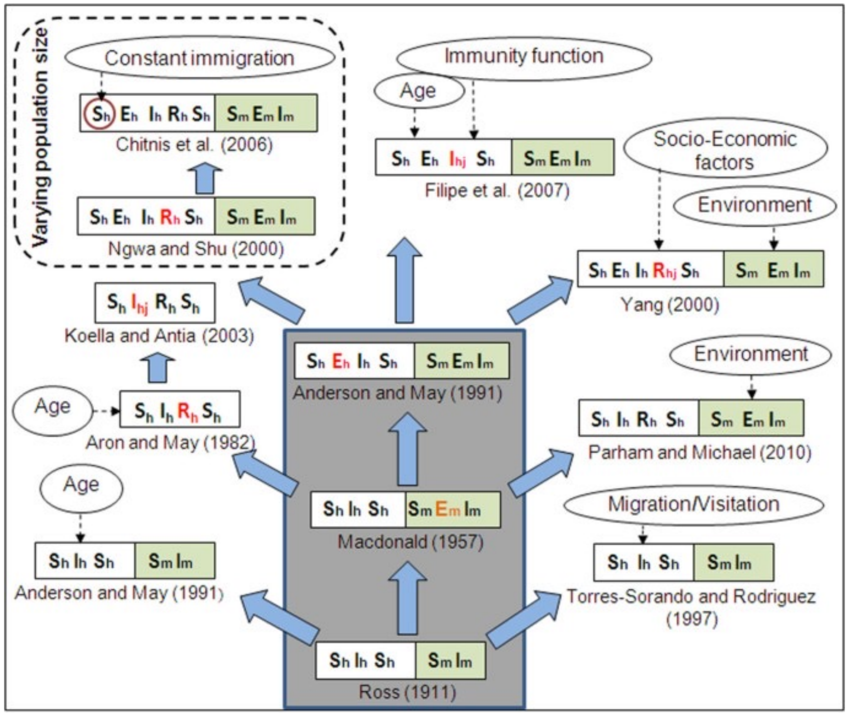
Learning objectives from Lecture 7

You should be able to:

- Understand the relationship between herd immunity and R0
- Understand the direction of time in a disease epidemic from a phase plane graph (from lab)
- Know the two global vaccination success stories and be able to list some of the challenges to vaccination in other cases
- Know the term ‘critical community size’
- Recognize different types of pathogen transmission and acknowledge that unique approaches are needed capture this transmission in a model
- Given a model diagram, recognize if the disease is transmitted directly or has a vector or animal reservoir

Malaria models have played a critical role in public health policy for over a century.

- 1911: British medical Dr. Sir Ronald Ross developed the first model of malaria while working in the Indian Medical Service.
- 1957: MacDonald modified this model to include the latent period of the parasite in the mosquito.
- This led to a widespread WHO campaign for malaria elimination using DDT in the 1950s!
- 1991: Anderson and May extended model to show latency in the human population.



Mandal et al. 2011. *Malaria Journal.*

Trash

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Which statement about malaria models is accurate?

A Ross-McDonald malaria models demonstrate mosquito recovery from infection.

2%

B The Ronald Ross malaria models influenced public health policy through t… an exposed…

SEE MORE

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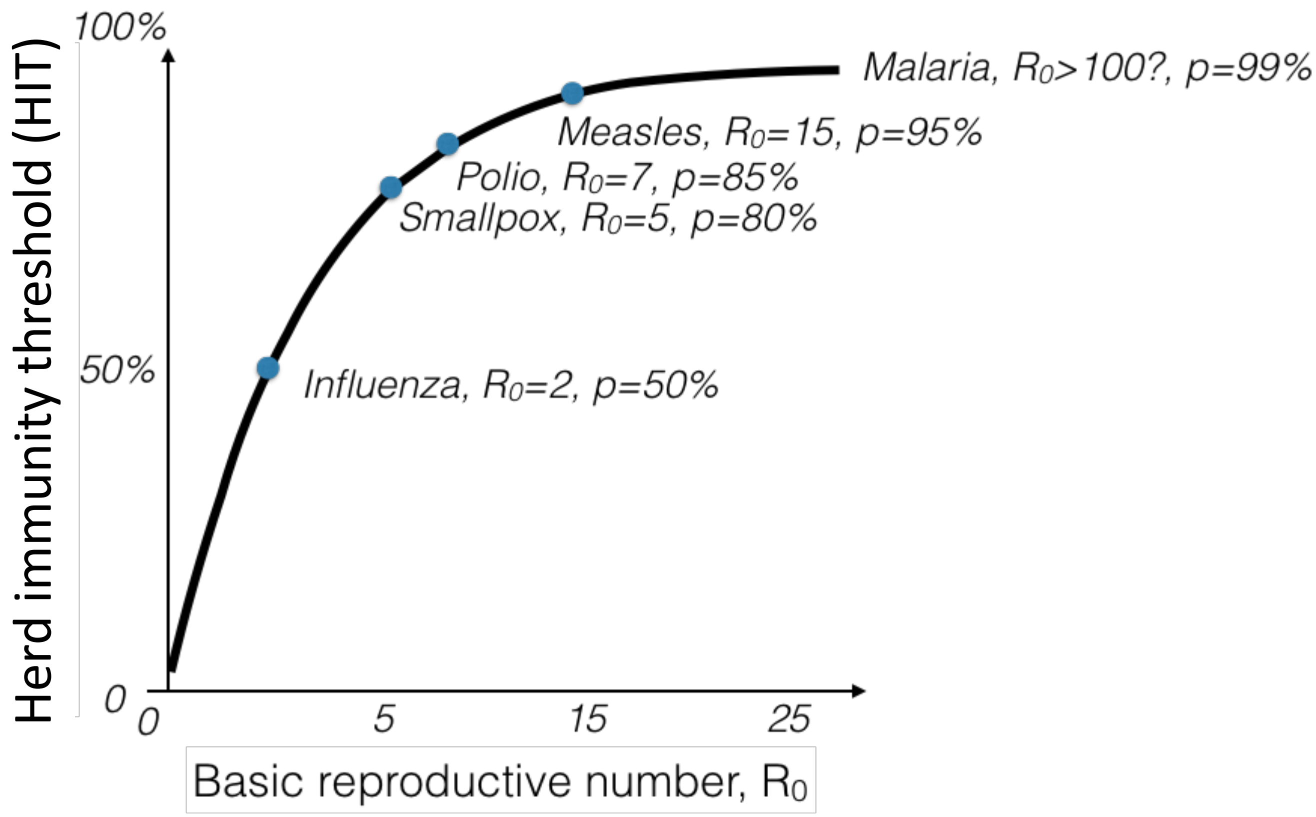
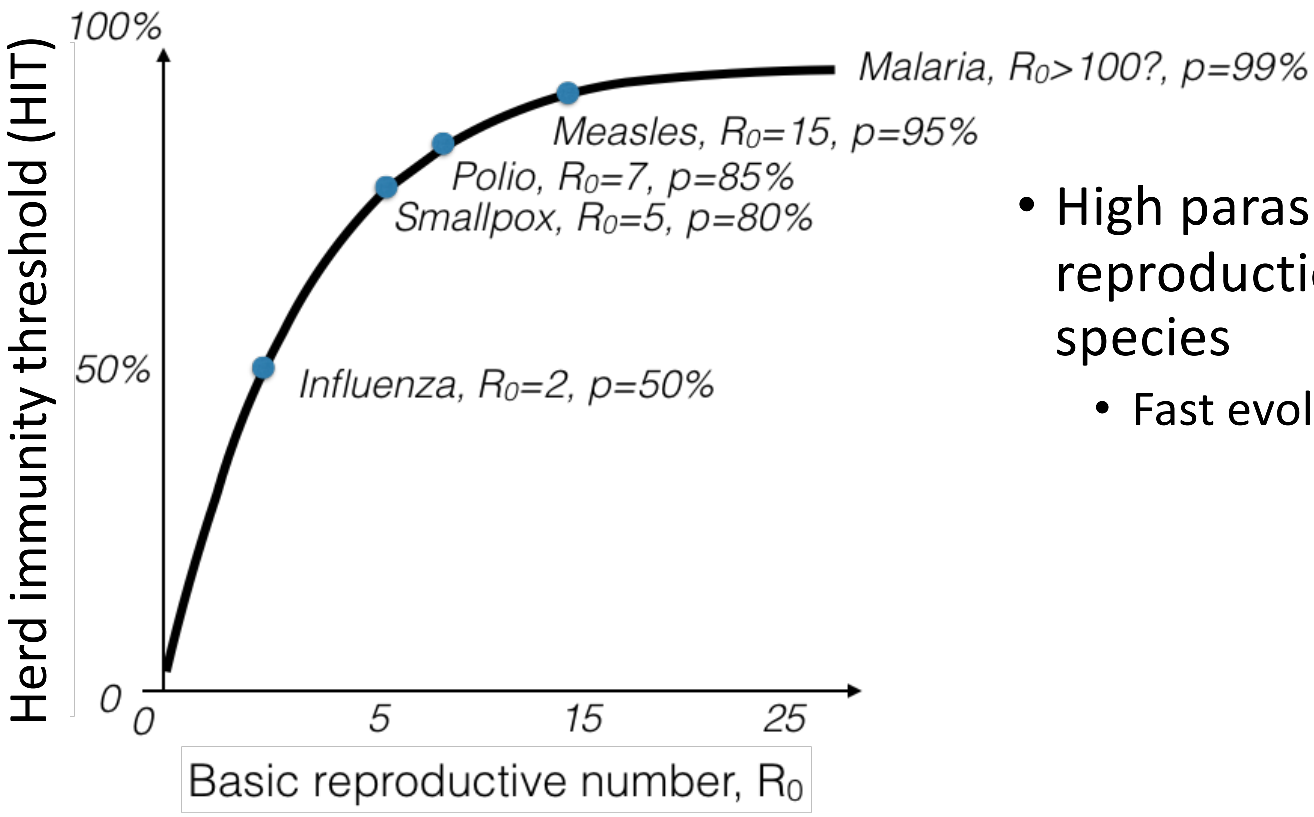
Challenges to malaria elimination

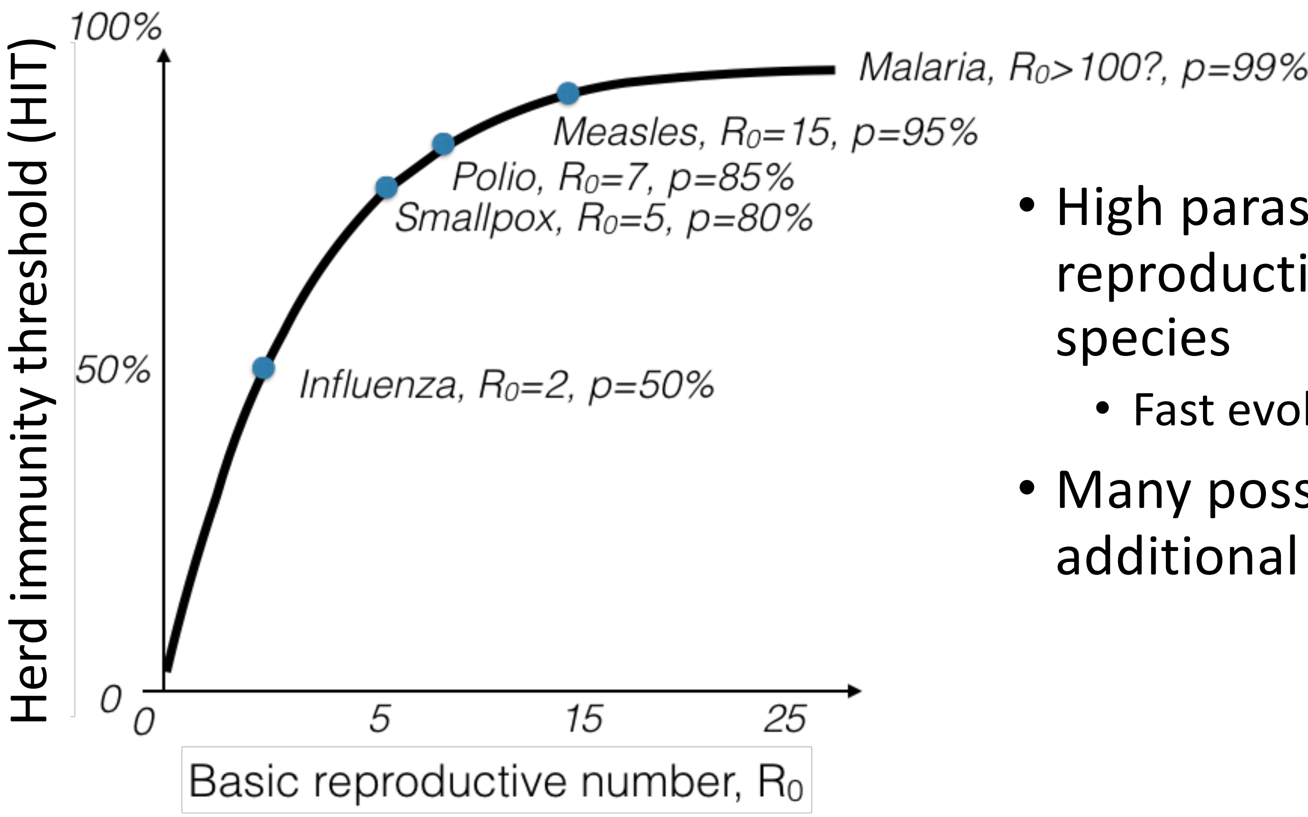


Challenges to malaria elimination



- High parasite diversity: sexual reproduction in 4+ *Plasmodium* species
  - Fast evolution of resistance *(e.g. to drugs)*

Challenges to malaria elimination



- High parasite diversity: sexual reproduction in 4+ *Plasmodium* species
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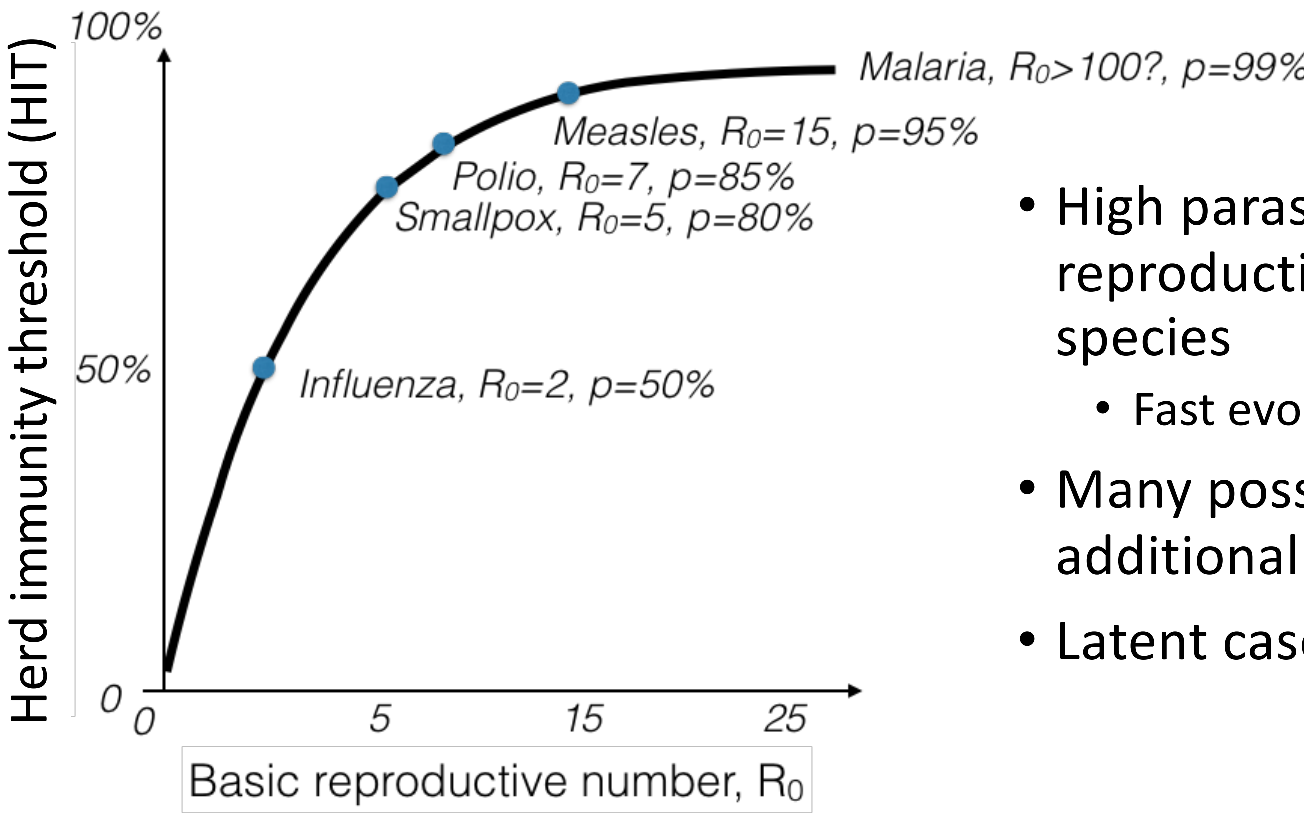
- Many possible vectors! Potentially additional possible reservoirs!

Challenges to malaria elimination



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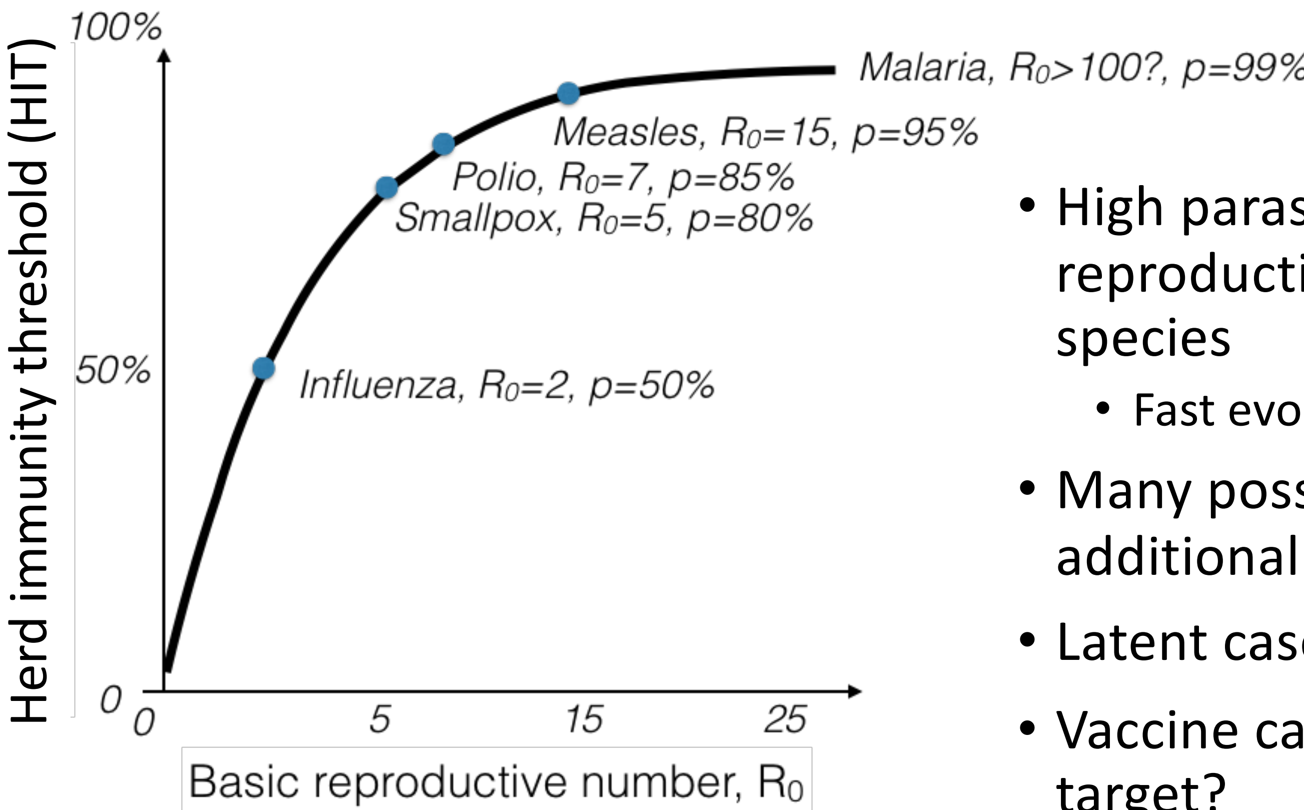
Challenges to malaria elimination



- High parasite diversity: sexual reproduction in 4+ *Plasmodium* species
  - Fast evolution of resistance *(e.g. to drugs)*
- Many possible vectors! Potentially additional possible reservoirs!
- Latent cases as burden is reduced
- Vaccine candidates: what life stage to target?

Pathogens exhibit **diverse transmission mechanisms** that require tailored modeling structures

- **Vector-borne** diseases (a type of indirect transmission) are transmitted via blood-feeding arthropod (mosquitoes, ticks, fleas)
  - Malaria: Mosquito-borne protozoan *Plasmodium spp.*
  - "Arboviruses": Mosquito-borne viruses, including Dengue, Zika, Yellow fever virus, West Nile virus, Chikungunya virus
  - Sleeping sickness, also known as African trypanosomiasis: tsetse fly vector and protozoan pathogen (trypanosome)
  - Chagas disease: kissing bug vector and trypanosome pathogen
  - Plague: flea vector and bacterial pathogen (*Yersinia pestis)*

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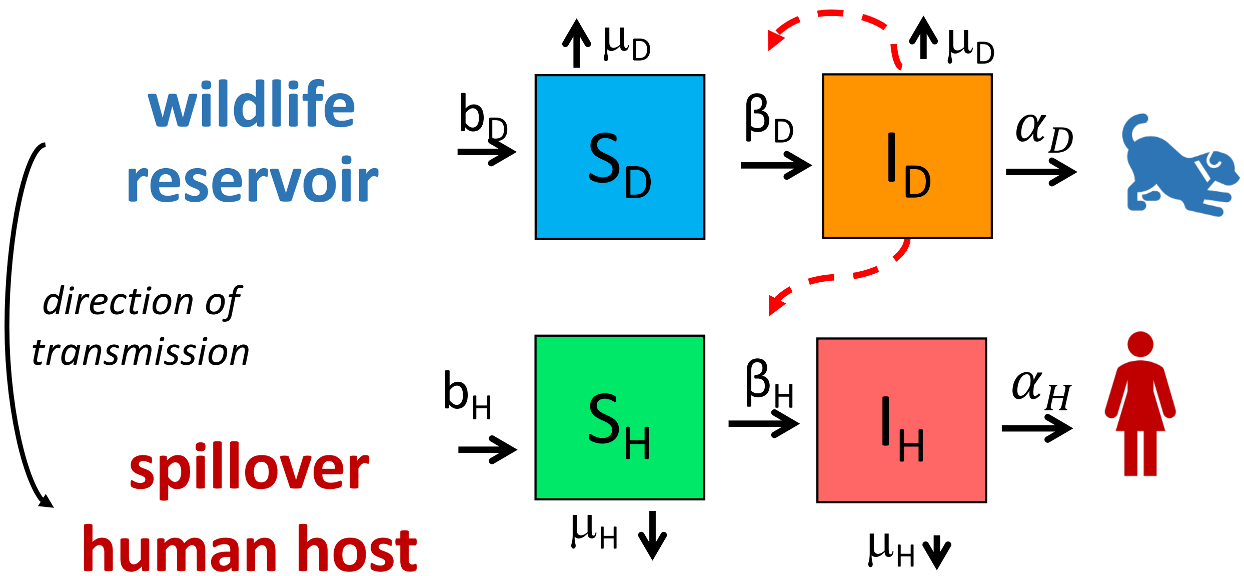
Plague is BOTH vector-borne and zoonotic!

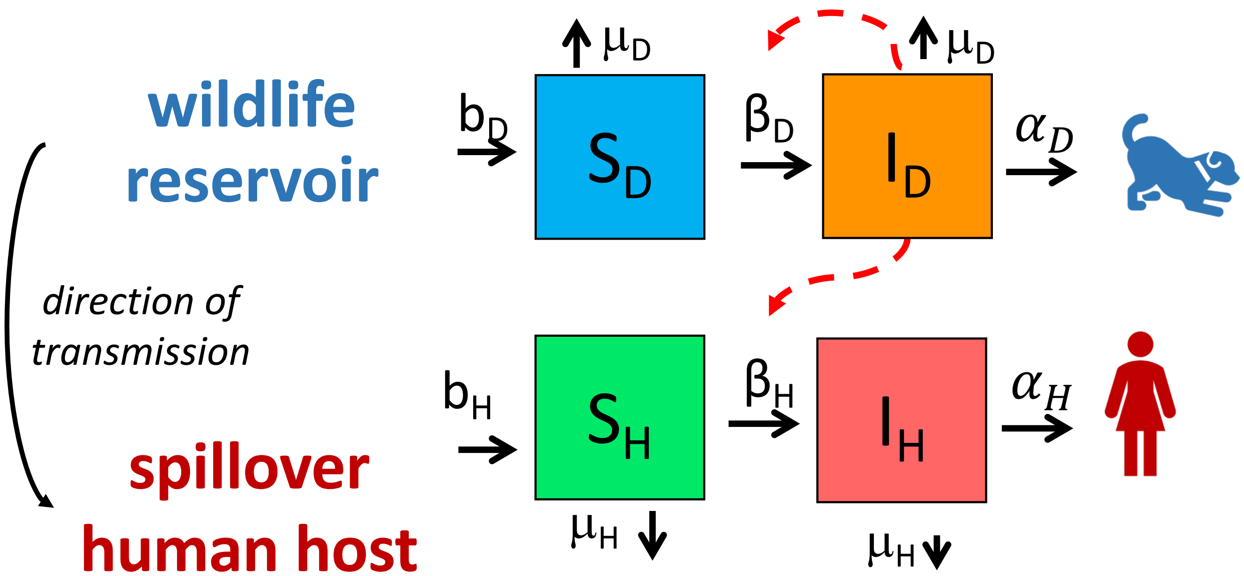
Zoonoses are pathogens transmitted from a **wildlife reservoir** to a **spillover human** host.



ex: rabies

Haydon et al. 2002. *Emerging Infectious Diseases.*

Zoonoses are pathogens transmitted from a wildlife reservoir to a spillover human host.

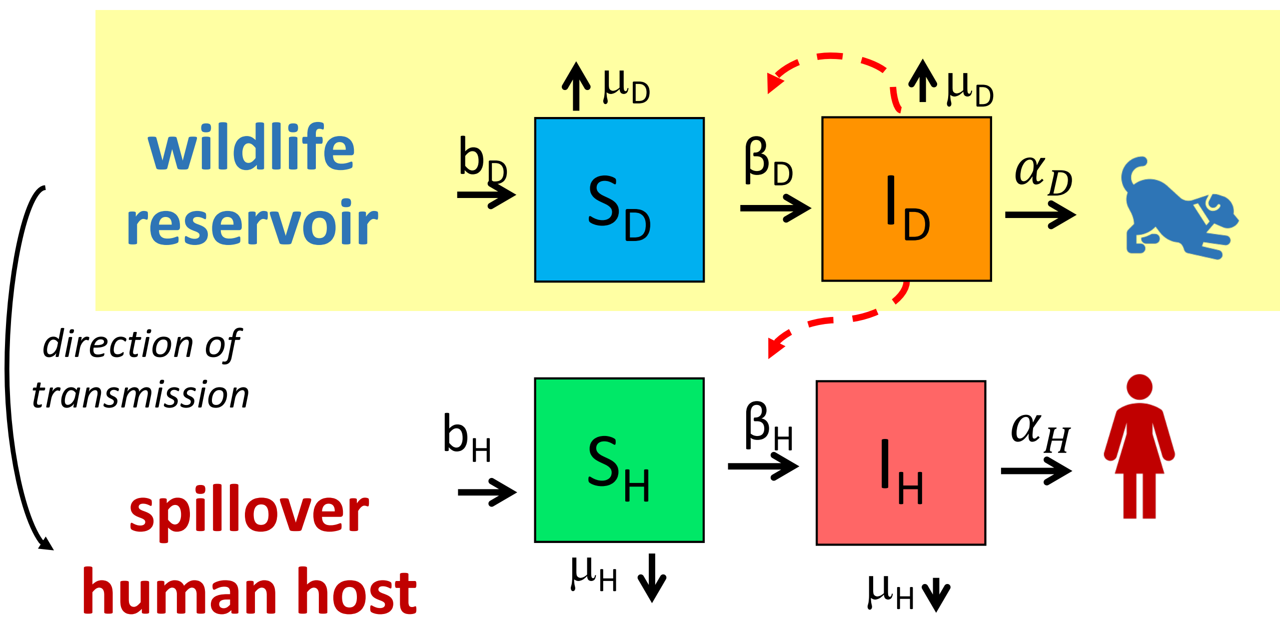


Typically modeled with the reservoir as a distinct state variable!

ex: rabies

Haydon et al. 2002. *Emerging Infectious Diseases.*

Zoonoses are pathogens transmitted from a **wildlife reservoir** to a **spillover human** host.



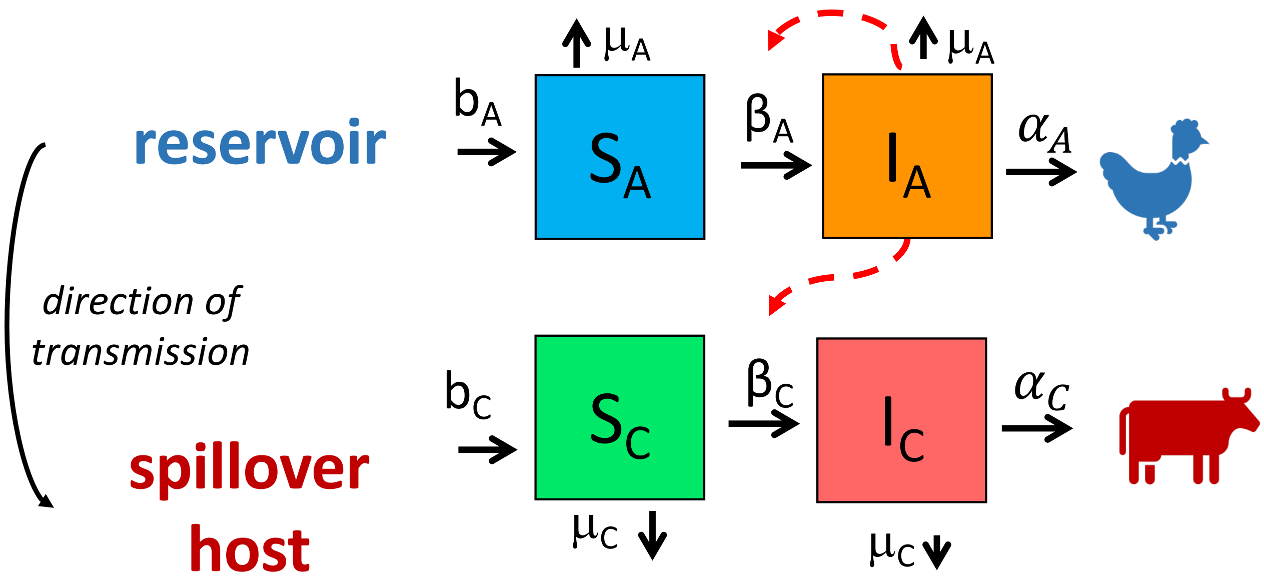
The **reservoir** host must be able to independently maintain the pathogen, with **population size > CCS**!

Animal hosts are not vectors!

ex: rabies

Haydon et al. 2002. *Emerging Infectious Diseases.*

Spillover is the term used to describe pathogen transmission between any two different species.



ex: avian flu

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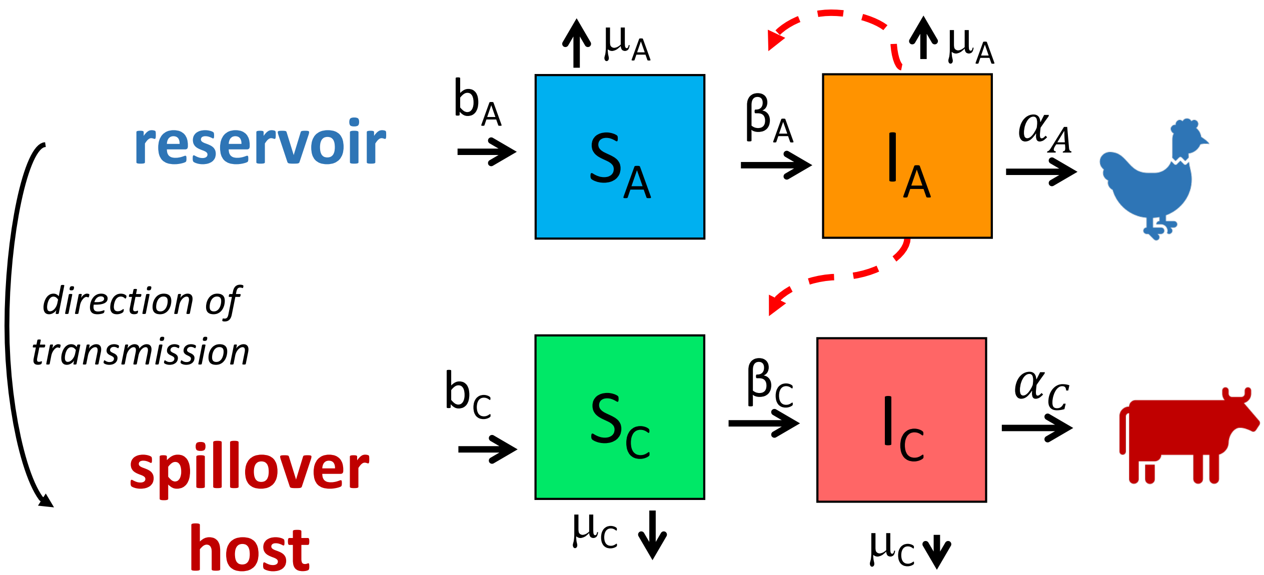
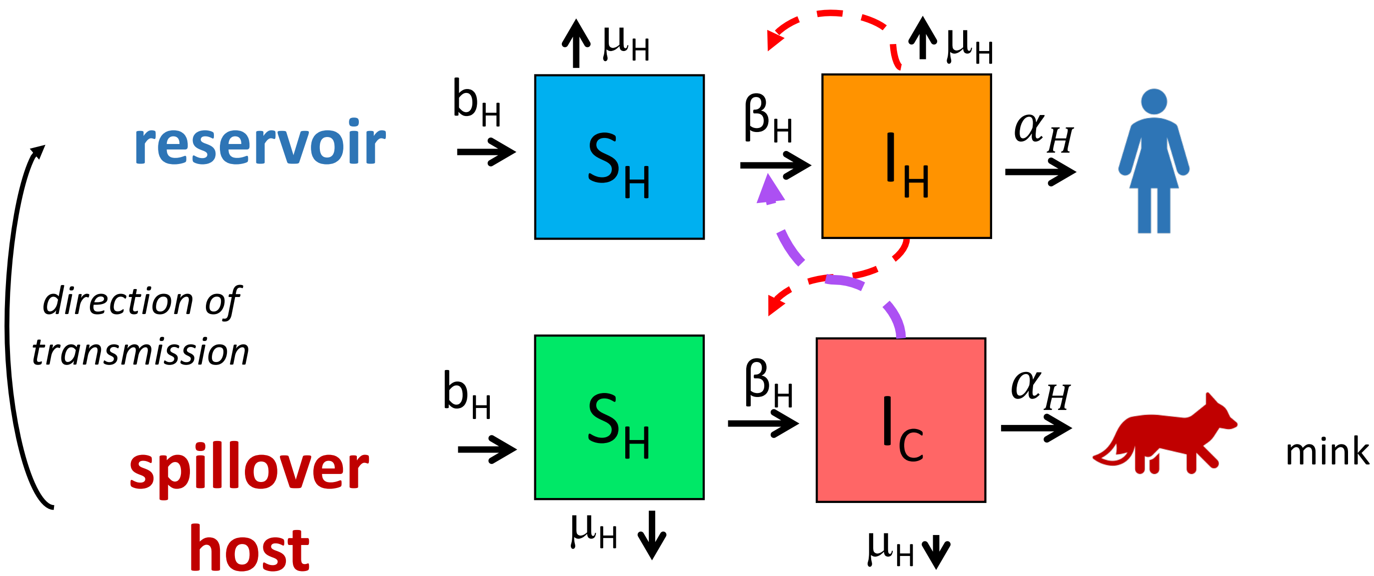


A zoonosis always requires spillover, but a spillover does not necessarily mean a zoonosis!

ex: avian flu

Spillback is the term used to describe pathogen transmission back to a **reservoir host** from a **spillover host**.



ex: SARS-CoV-2

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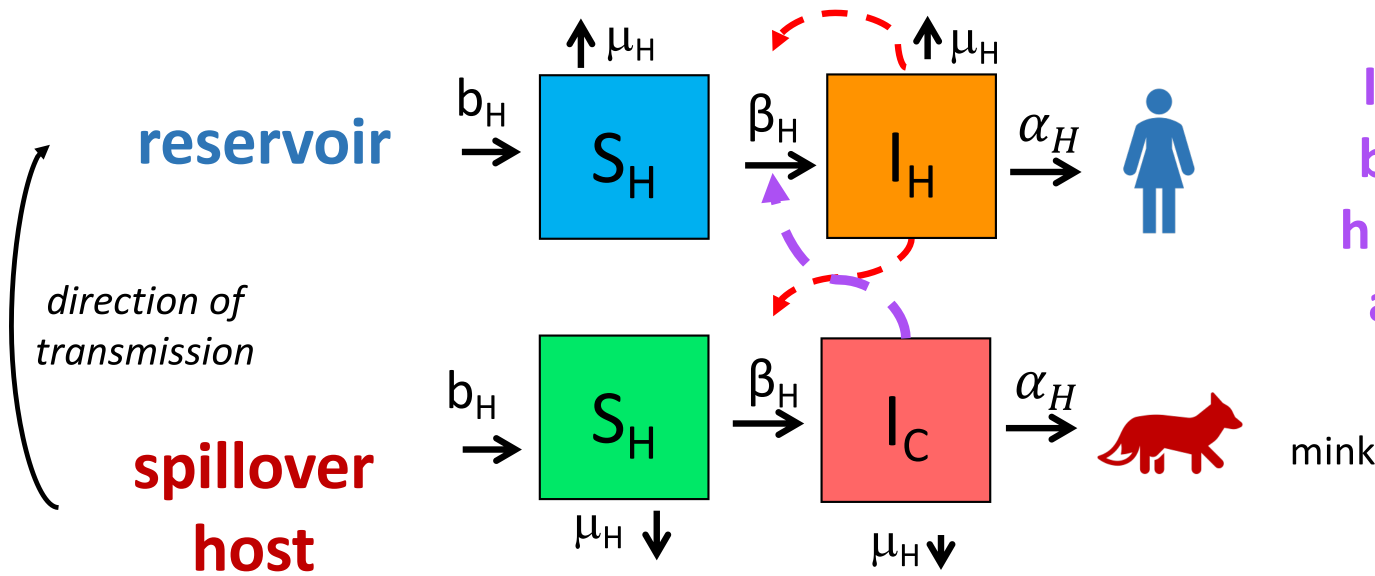
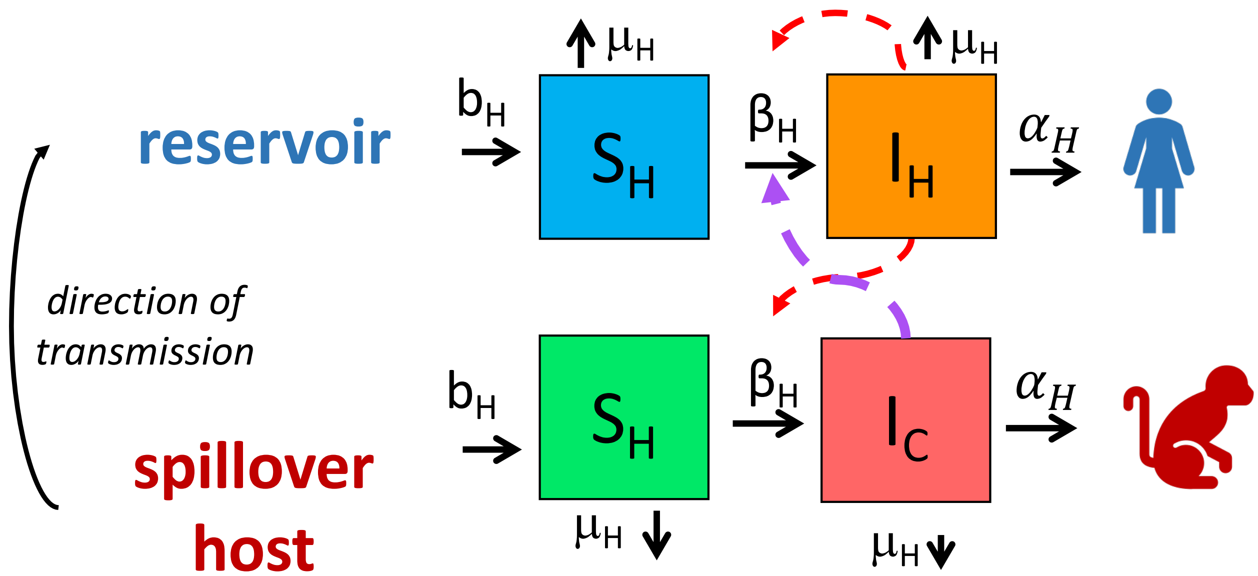


Interesting case because neither humans nor mink are the original reservoir host!

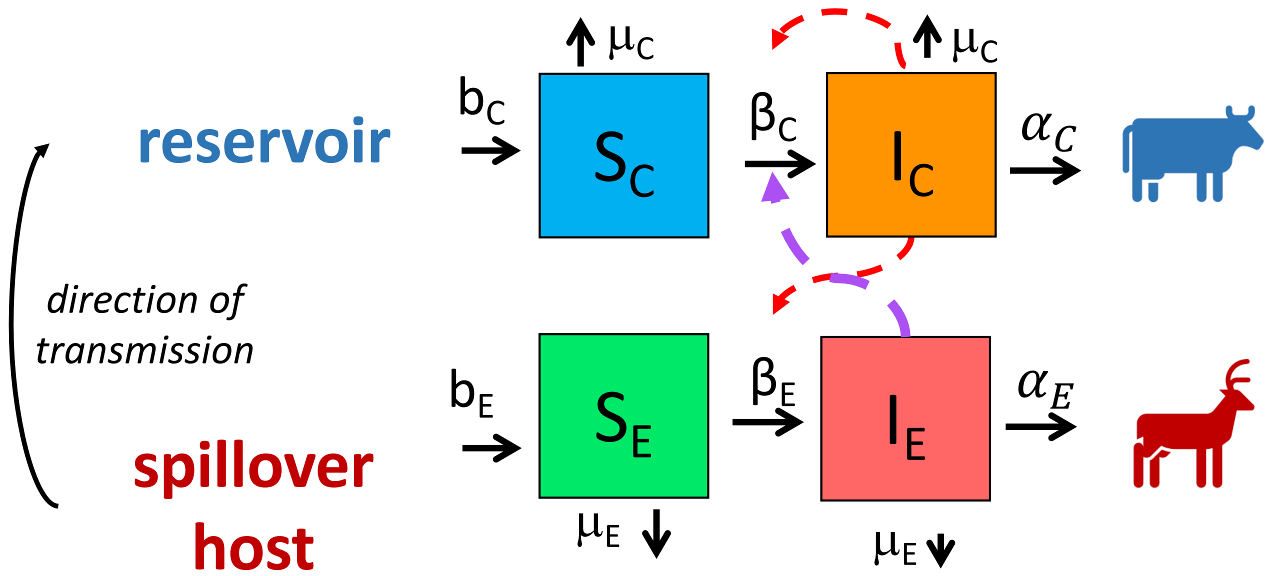
ex: SARS-CoV-2

Spillback is the term used to describe pathogen transmission back to a **reservoir host** from a **spillover host**.



ex: Yellow Fever in the Americas

Spillback occurs among wildlife as well.

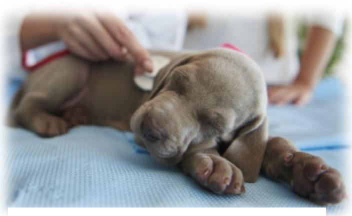


ex: *Brucella* spp. in Yellowstone National Park

Pathogens can be classed according to their host relationships.

Stage I

Transmits exclusively in animals



canine parvovirus

Stage II

Human cases from spillovers only

rabies virus

Stage III

Stuttering chains of transmission in humans

monkeypox (pre-2022)

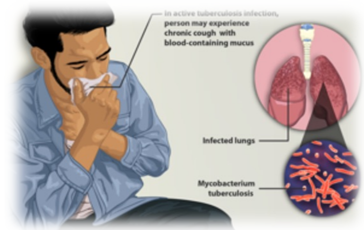
Stage IV

Sustained transmission and human outbreaks

Ebola virus (especially post-2014)

Stage V

Transmits exclusively in humans



Tuberculosis

Lloyd-Smith et al. 2009. *Science.*

Pathogens can be classed according to their host relationships.

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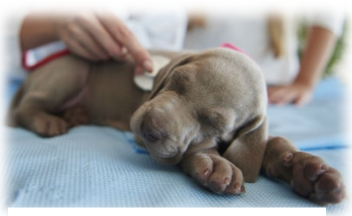


canine parvovirus

Stage II

Human cases from spillovers only

rabies virus

$R_0 < 1$

Stage III

Stuttering chains of transmission in humans

monkeypox (pre-2022)

$R_0 \approx 1$

Stage IV

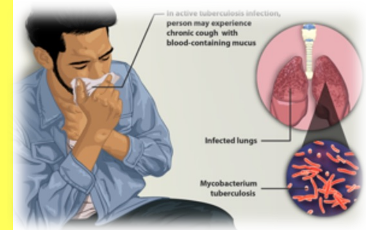
Sustained transmission and human outbreaks

Ebola virus (especially post-2014)

$R_0 > 1$

Stage V

Transmits exclusively in humans

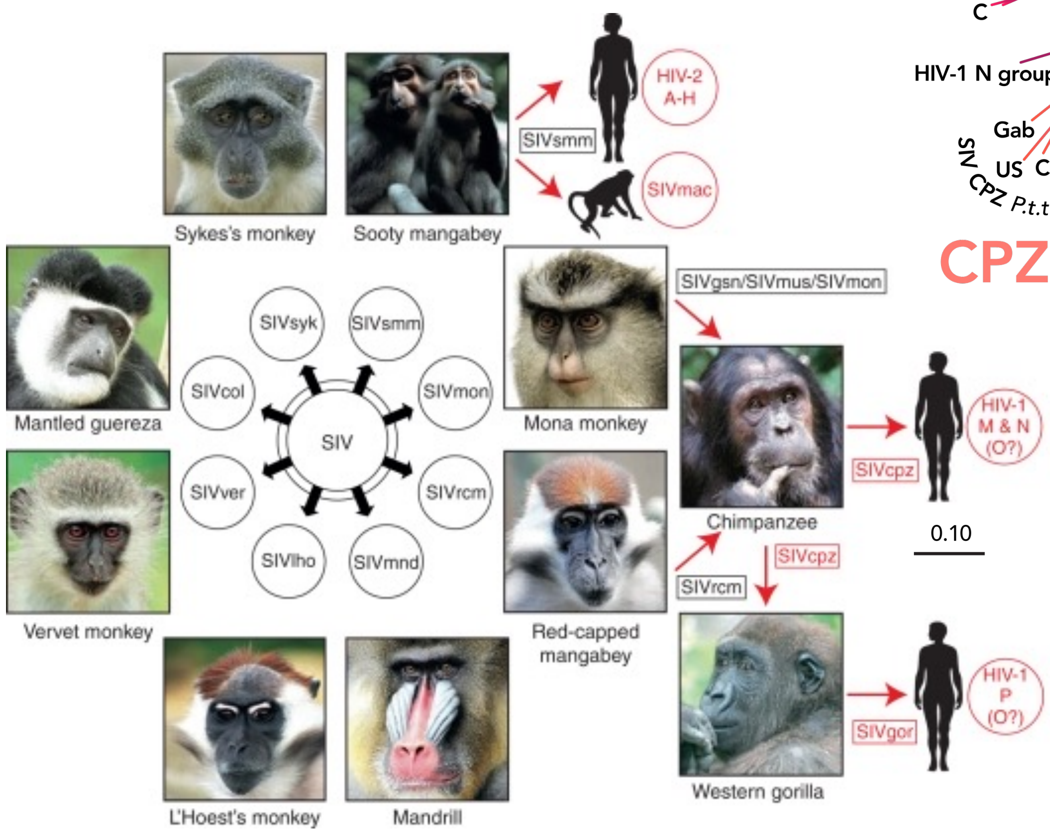


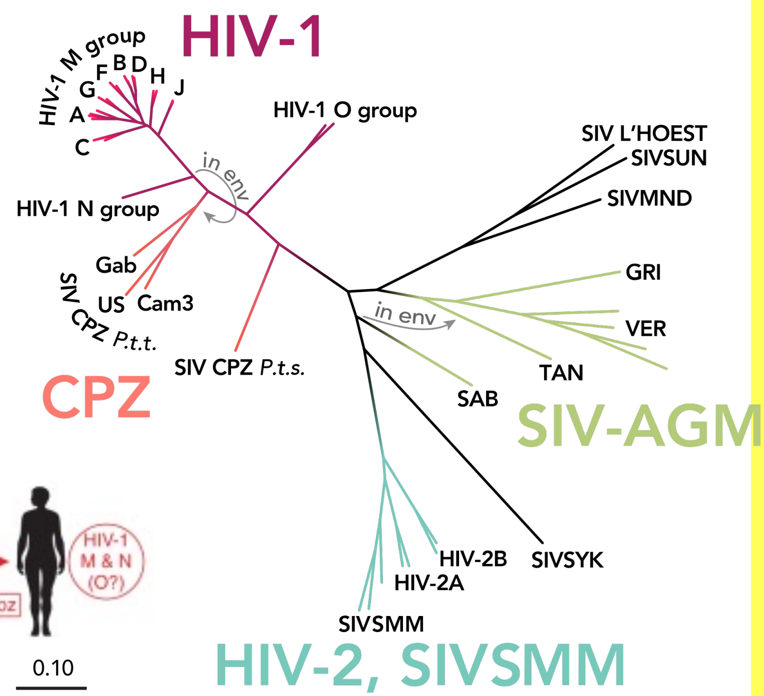
Tuberculosis

Zoonotic pathogens can be classed according to their R_0 in humans.

Lloyd-Smith et al. 2009. *Science.*

Most stage V pathogens once had an animal origin, as well!





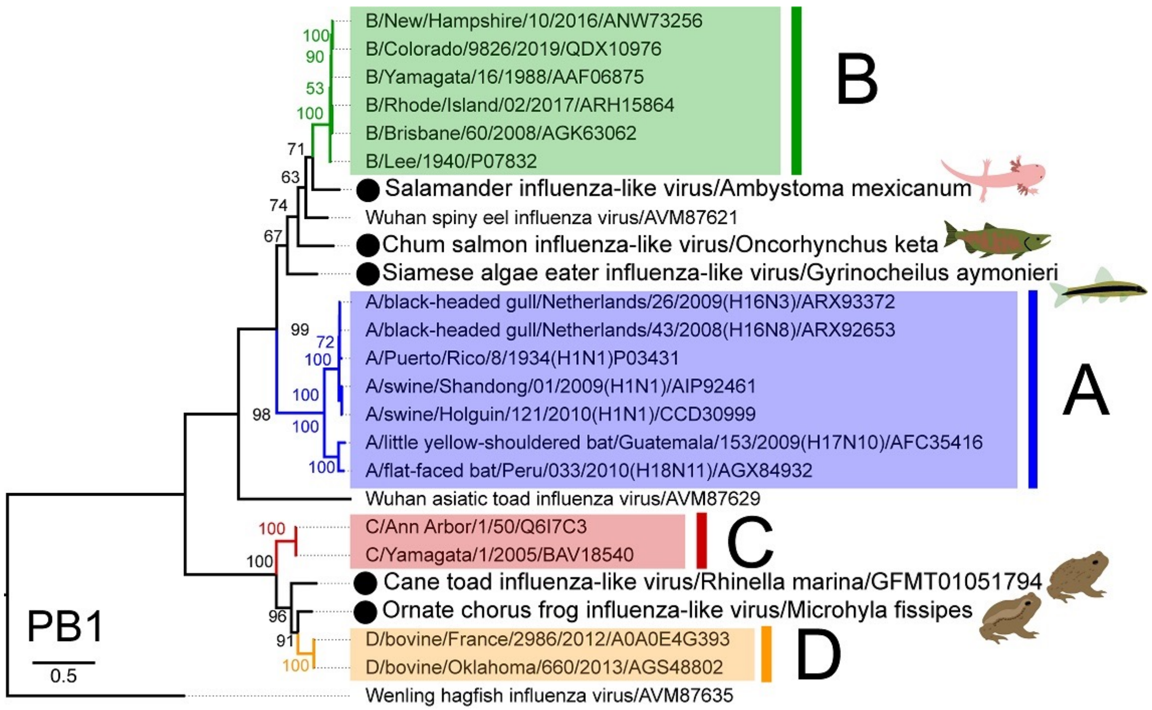
Stage V

Transmits exclusively in humans

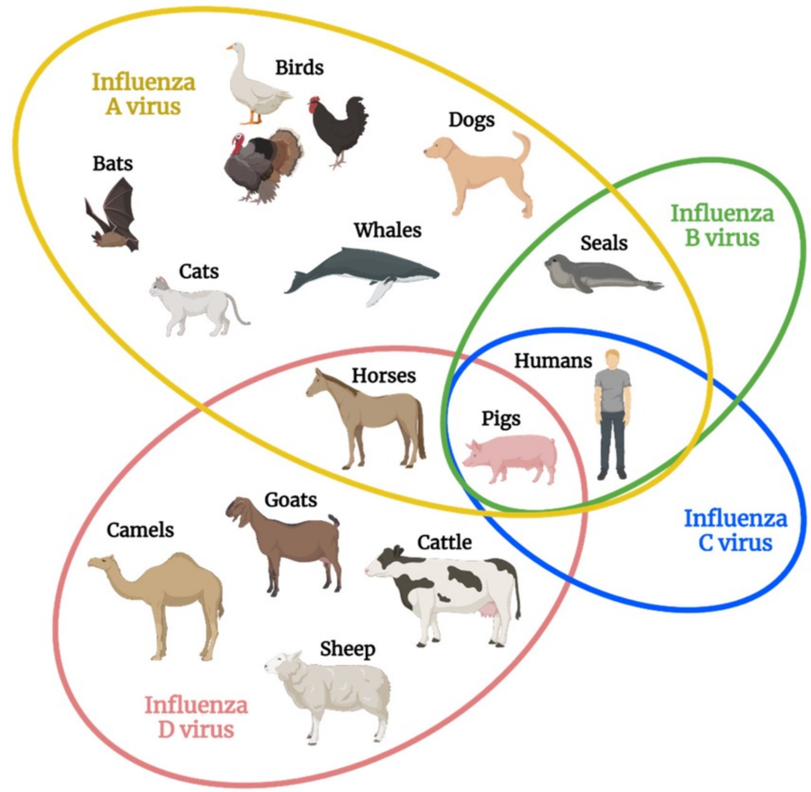
HIV

Sharp & Hahn. 2011. *Cold Spring Harb Perspect Med.*

When is influenza zoonotic?

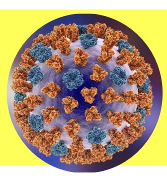


Parry et al. 2020. *Viruses.*



Skelton & Huber. 2022. *Viruses.*

Stage V Transmits exclusively in humans



Influenza

Wk8-Lect7-Feb27

Visual settings

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Which of the following is never a zoonotic disease?

A Plasmodium knowelsi - a macaque malaria t…	0%
B Influenza B	0%
C Toxoplasma gondii - a protozoan circulating…	0%
D Q fever - a bacterial pathogen of livestock t…	0%
E Influenza A	0%

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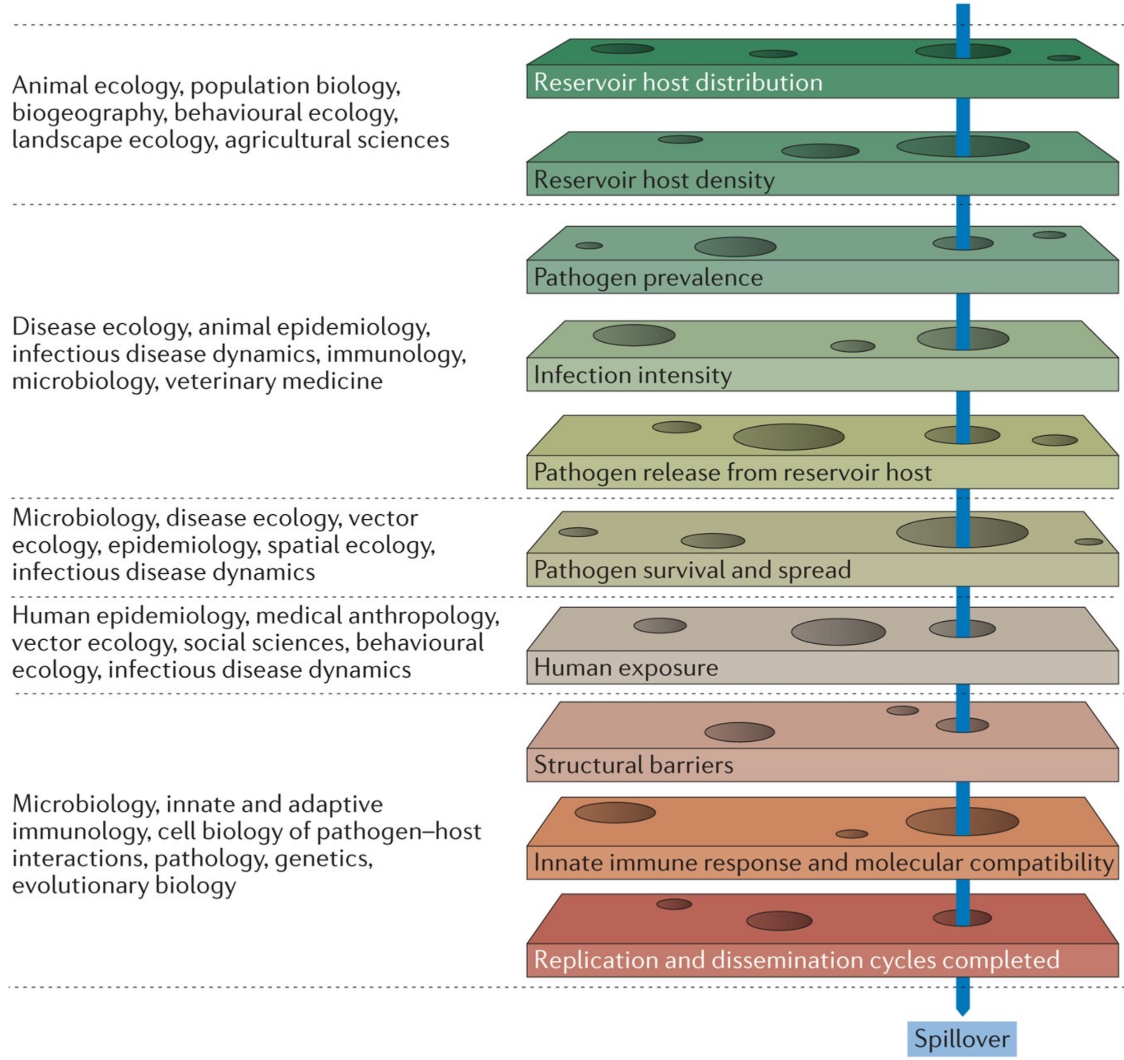
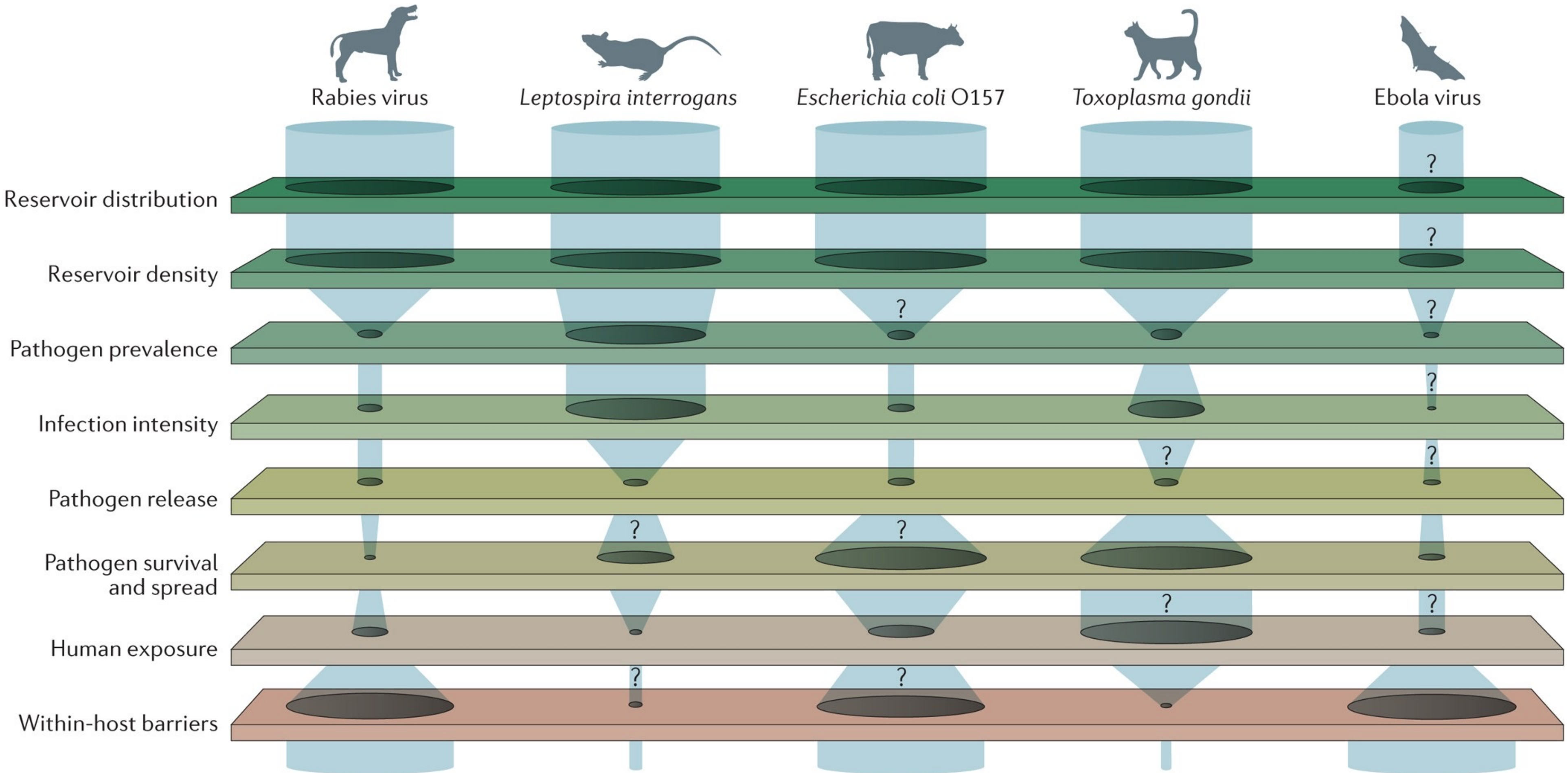
There are **many barriers** to cross-species transmission.

We can think of zoonosis as a series of **improbable events** multiplied together.



Plowright et al. 2017. *Nature Reviews Microbiology.*



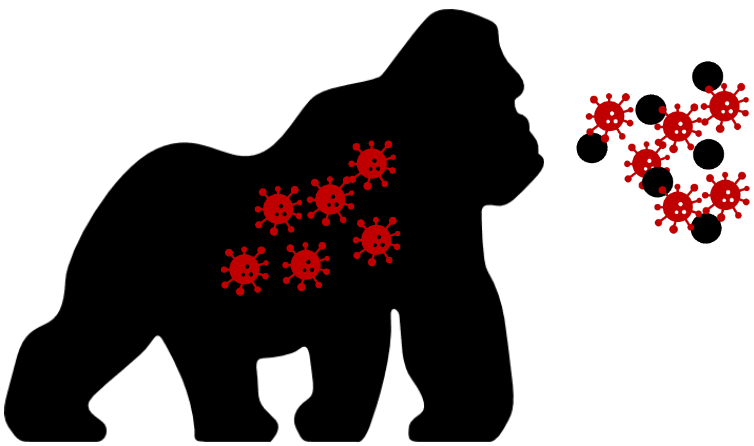
Bottlenecks to zoonotic transmission vary for different pathogens.

Plowright et al. 2017. *Nature Reviews Microbiology.*

Why do pathogens make us sick?

A virus will evolve to **maximize** its capacity for **between-host infections** (R_0).

Why do pathogens make us sick?



Alizon et al. 2008. *J Evolutionary Biology*
Anderson and May 1982. *Parasitology.*

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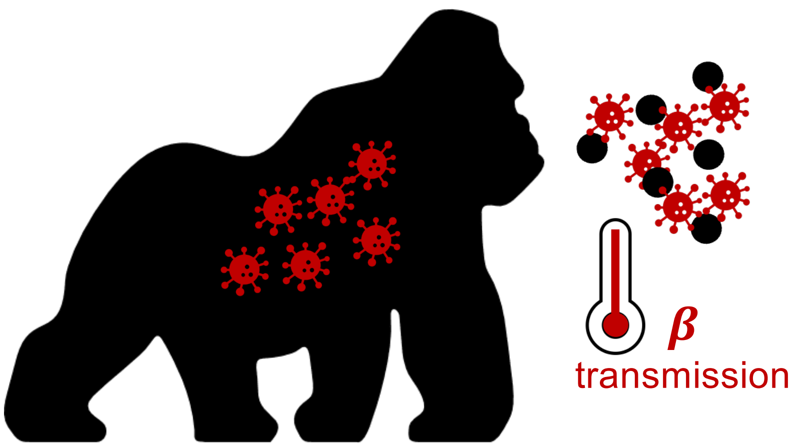

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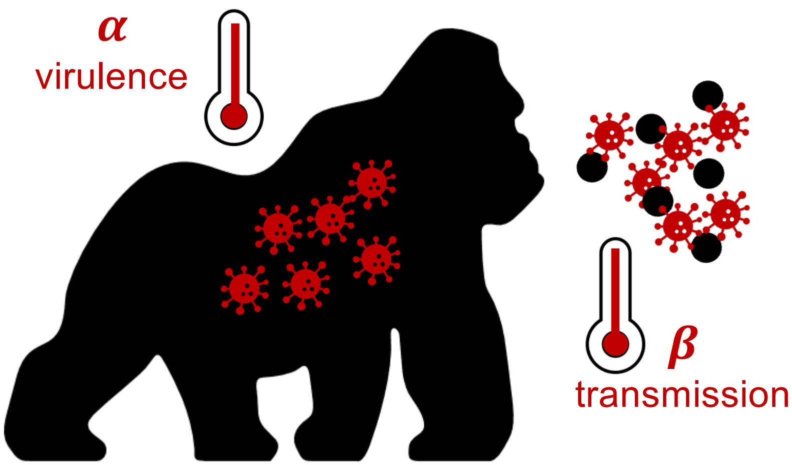

Mechanisms that promote **transmission** may also enhance **virulence** to the host.

Alizon et al. 2008. *J Evolutionary Biology*
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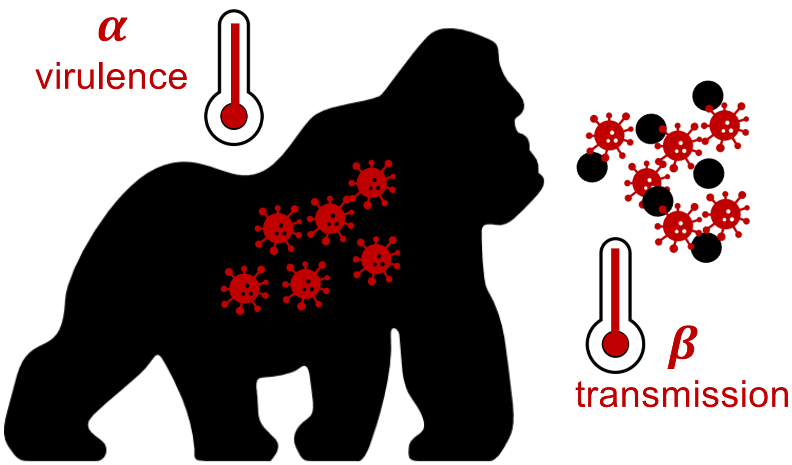

Mechanisms that promote **transmission** may also enhance **virulence** to the host.

Why do pathogens make us sick?

Virulence, then, is a by-product of a pathogen's need to transmit for reproduction!

Alizon et al. 2008. *J Evolutionary Biology*
Anderson and May 1982. *Parasitology.*

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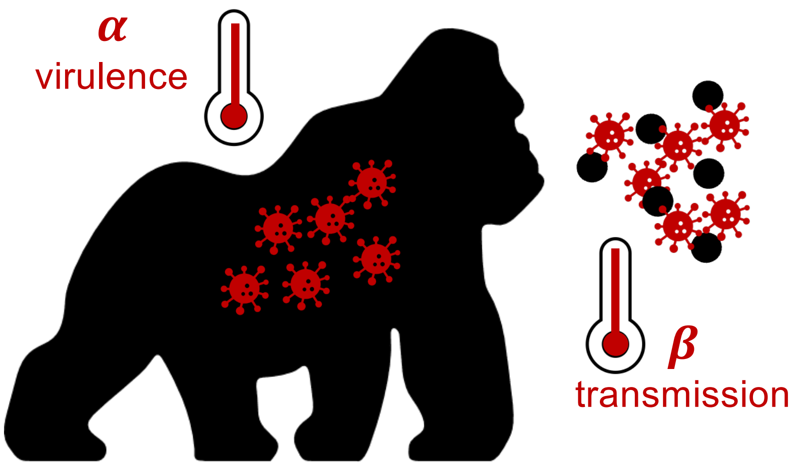
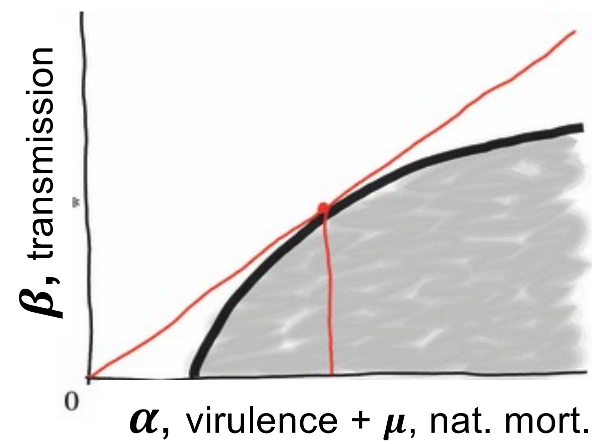
Why do pathogens make us sick?



As a result, we predict the evolution of "**optimal virulence**."



Mechanisms that promote **transmission** may also enhance **virulence** to the host.

$$\mathbf{R_0} = \frac{\boldsymbol{\beta}(virus\ density)}{\boldsymbol{\gamma} + \boldsymbol{\mu} + \boldsymbol{\alpha}(virus\ density)} \quad \begin{matrix} \} \text{ infections created} \\ \} \text{ infections lost} \end{matrix}$$

Alizon et al. 2008. *J Evolutionary Biology*
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Why do pathogens make us sick?

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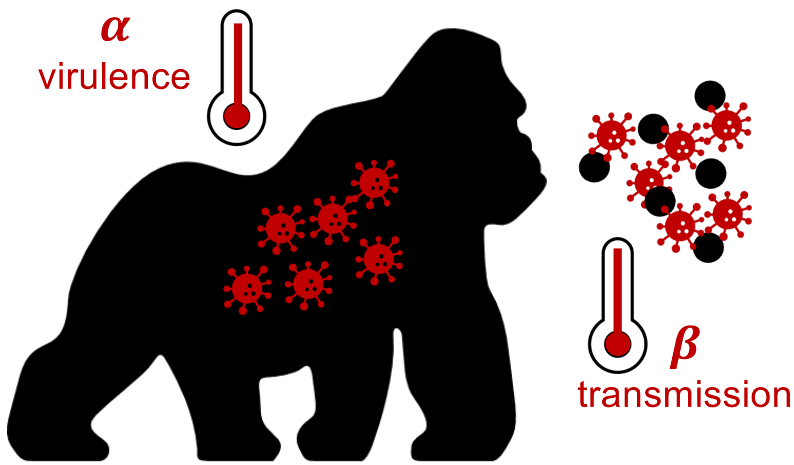

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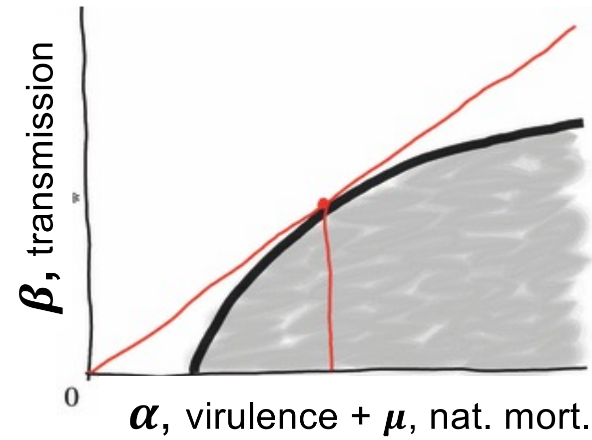

Note that originally Anderson and May (1982) represented this link to virus density as acting on the disease recovery rate, though it is now more commonly expressed as a function of virulence!

$$\mathbf{R_0} = \frac{\boldsymbol{\beta}(\textit{virus density})}{\boldsymbol{\gamma} + \boldsymbol{\mu} + \boldsymbol{\alpha}(\textit{virus density})} \quad \begin{matrix} \} \text{ infections created} \\ \} \text{ infections lost} \end{matrix}$$

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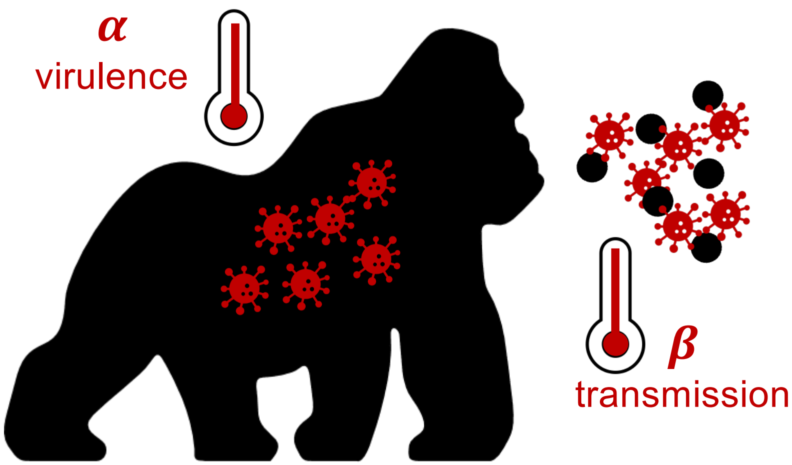

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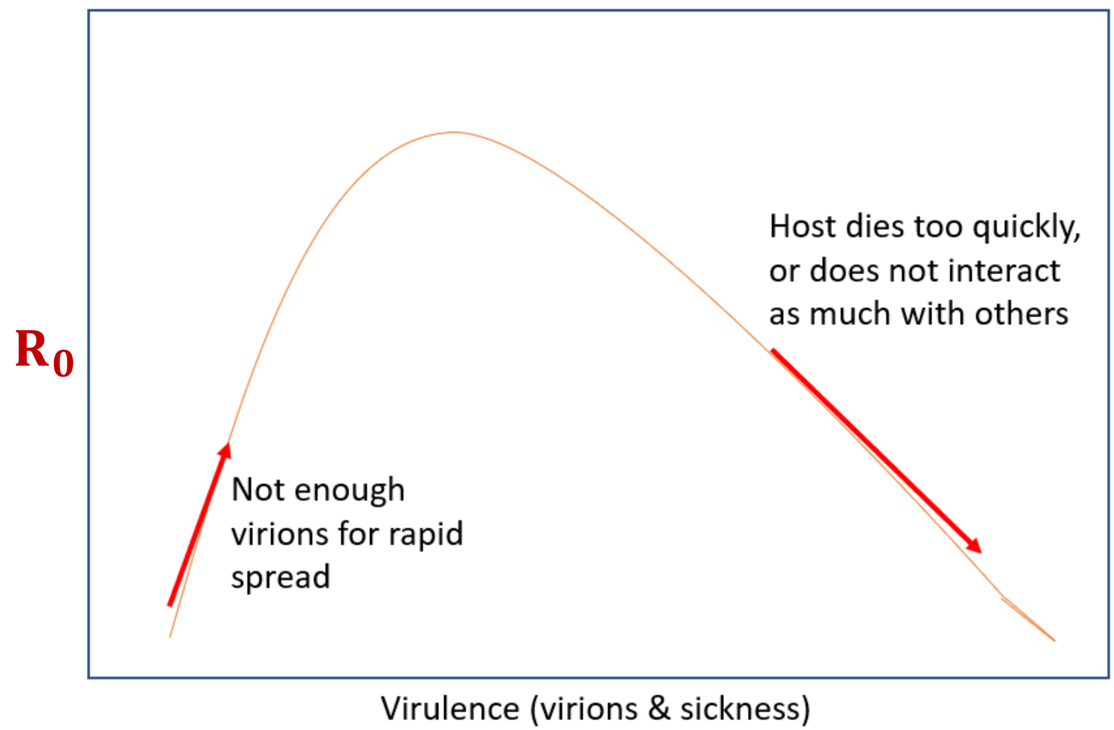


The **virulence case study** of rabbit Myxoma virus

The **virulence case study** of rabbit Myxoma virus

- 1788: European rabbits brought to Australia as a food source
- Rabbits quickly became feral and numbers soared.
- 1901: Australia constructed the famous “rabbit-proof fence” to attempt to keep rabbits out of agriculture in the West.

The **virulence case study** of rabbit Myxoma virus

- 1788: European rabbits brought to Australia as a food source
- Rabbits quickly became feral and numbers soared.
- 1901: Australia constructed the famous "rabbit-proof fence" to attempt to keep rabbits out of agriculture in the West.
- Government looked to control measures, including biological controls in the 1930s.
- Tried Myxoma virus, a highly virulent European poxvirus infecting rabbits. with a CFR >99%.

Myxoma virus evolved to **intermediate virulence** in just a single year.

TABLE 4. THE VIRULENCE OF STRAINS OF MYXOMA VIRUS RECOVERED FROM THE FIELD IN AUSTRALIA BETWEEN 1951 AND 1981, EXPRESSED AS PERCENTAGES

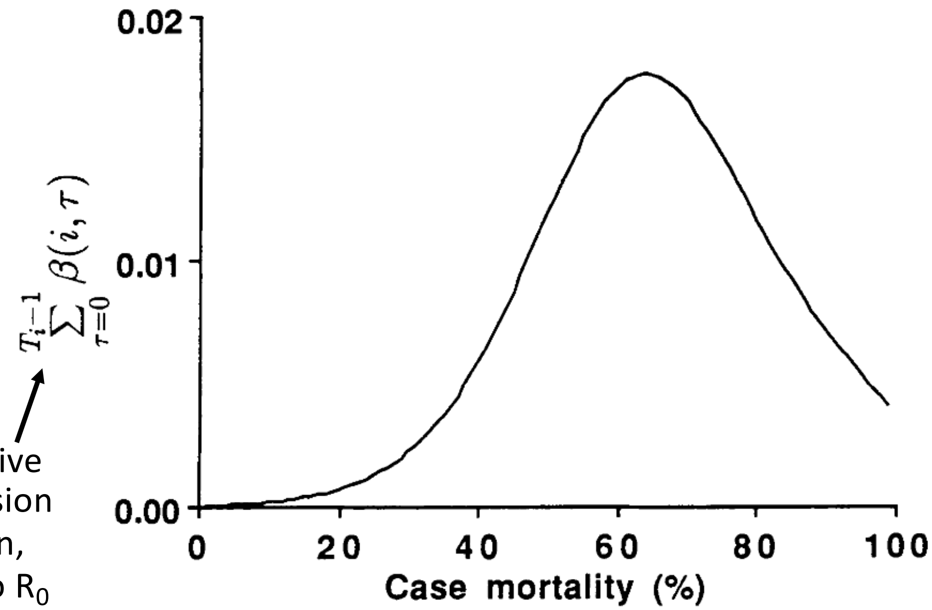
virulence grade	I	II	III	IV	V	
case fatality rate (%)	> 99	95–99	70–95	50–70	< 50	number of
mean survival time/day	< 13	14–16	17–28	29–50	—	samples
1950–51†	100‖					1
1952–55†	13.3	20.0	53.3	13.3	0	60
1955–58†	0.7	5.3	54.6	24.1	15.5	432
1959–63‡	1.7	11.1	60.6	21.8	4.7	449
1964–66‡	0.7	0.3	63.7	34.0	1.3	306
1967–69‡	0	0	62.4	35.8	1.7	229
1970–74‡	0.6	4.6	74.1	20.7	0	174
1975–81§	1.9	3.3	67.0	27.8	0	212

† Data from Marshall & Fenner (1960).
‡ Data from Edmonds *et al.* (1975).
§ Data from J. W. Edmonds and R. C. H. Shepherd (personal communication, 1982).
‖ Although only one strain was tested, the very high mortality rates in the initial outbreaks justify this extrapolation.

Fenner 1983. *Proc Roy Soc B*

For Myxoma virus, **intermediate virulence evolution** resulted from **optimization of the tradeoffs between virulence and transmission**.

Rabbits around a waterhole in the myxomatosis trial site on Wardang Island, Australia, 1938



Cumulative transmission function, similar to R_0

A SIMULATION MODEL OF THE POPULATION DYNAMICS AND EVOLUTION OF MYXOMATOSIS[1]

GREG DWYER
Department of Zoology, University of Washington, Seattle, Washington 98195 USA

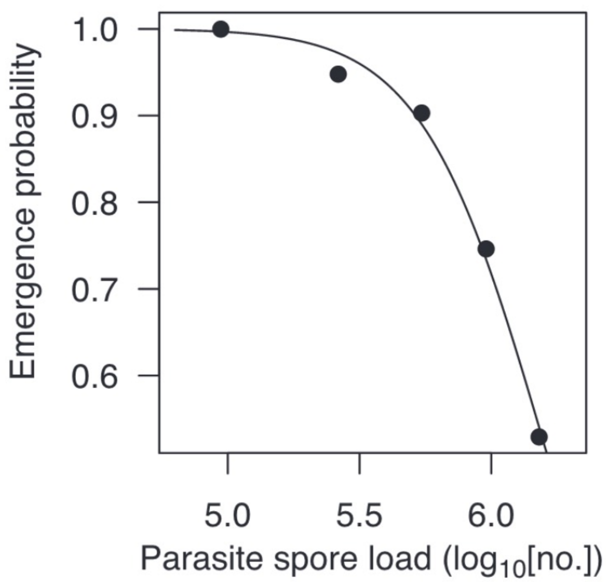
SIMON A. LEVIN
Section of Ecology and Systematics, Corson Hall, Cornell University, Ithaca, New York 14853 USA

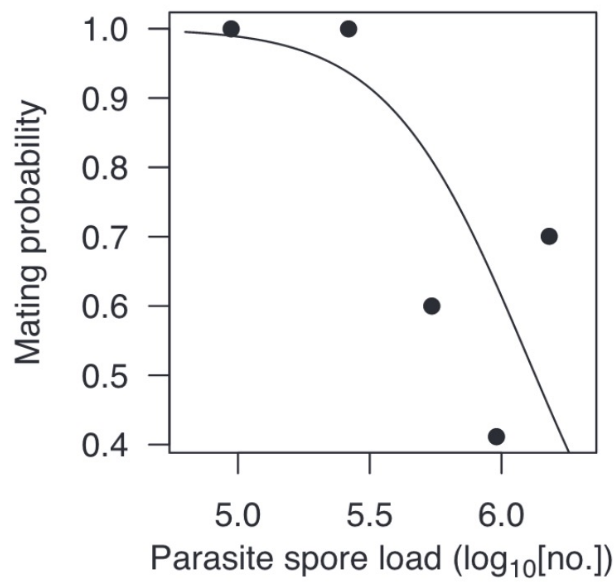
LINDA BUTTEL
Ecosystems Research Center, Corson Hall, Cornell University, Ithaca, New York 14853 USA

Dwyer, Levin, and Buttel. 1990. *Ecological Monographs.*

Another classic **transmission-virulence tradeoff:** parasites of monarch butterflies

- Monarch butterflies infected with the protozoan parasite, *Ophryocystis elektroscirrha*, demonstrate reduced emergence and mating probabilities at higher parasite spore load (**virulence**).

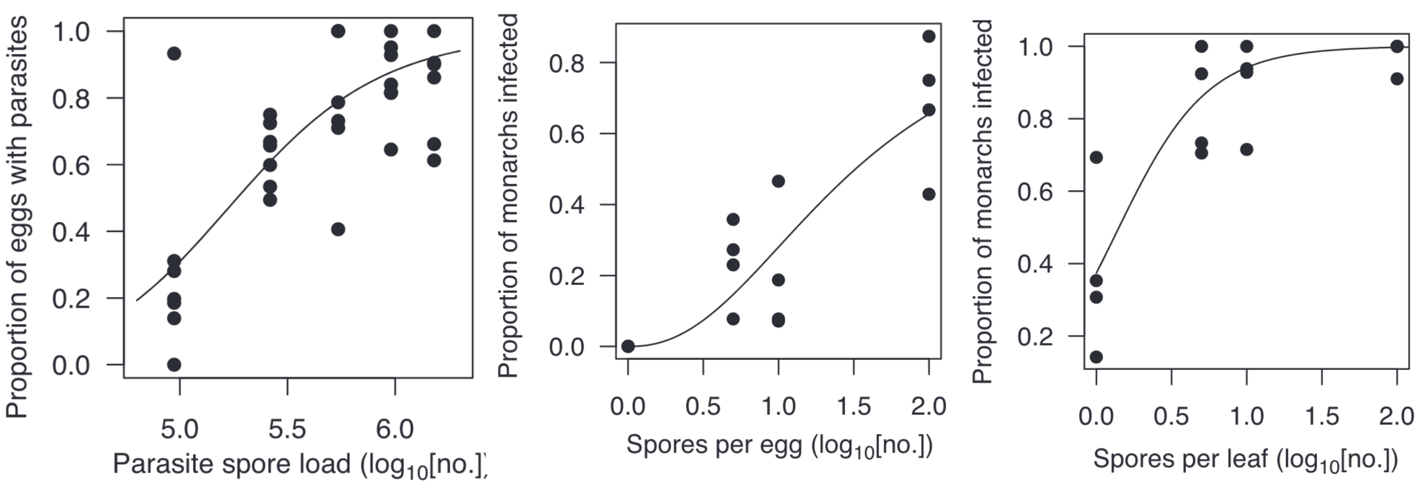




de Roode et al. 2008. *PNAS.*

Another classic **transmission-virulence tradeoff:** parasites of monarch butterflies

- Monarch butterflies infected with the protozoan parasite, *Ophryocystis elektroscirrha*, demonstrate reduced emergence and mating probabilities at higher parasite spore load (**virulence**).
- On the flip side, higher parasite loads also led to increased **transmission** through higher proportions of monarch eggs that acquired spores and higher numbers of parasites per egg and milkweed leaf, which increased the probability of infection.



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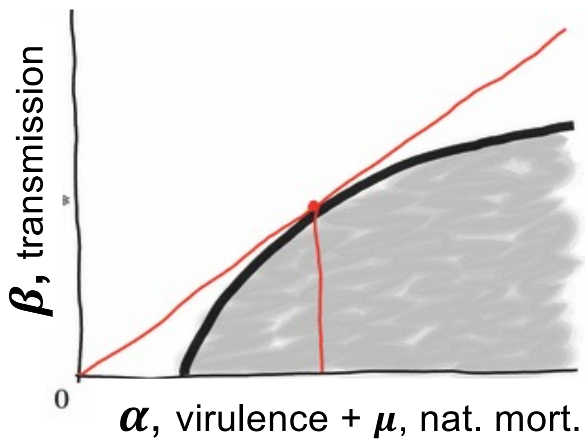
Another classic **transmission-virulence tradeoff:** parasites of monarch butterflies

- Monarch butterflies infected with the protozoan parasite, *Ophryocystis elektroscirrha*, demonstrate reduced emergence and mating probabilities at higher parasite spore load (**virulence**).
- On the flip side, higher parasite loads also led to increased **transmission** through higher proportions of monarch eggs that acquired spores and higher numbers of parasites per egg and milkweed leaf, which increased the probability of infection.
- **Parasite fitness is calculated to be maximized at intermediate spore load.**



de Roode et al. 2008. *PNAS.*

Limitations of the tradeoff model

- The 'trade-off hypothesis' offers an explanation for the disease inflicted by parasites and pathogens on their original hosts. While well-designed theoretically, it has not been historically well-supported empirically!

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    - Sometimes, virulence is the result of the host's immune response, rather than the direct impact of the parasite itself, further complicating dynamics

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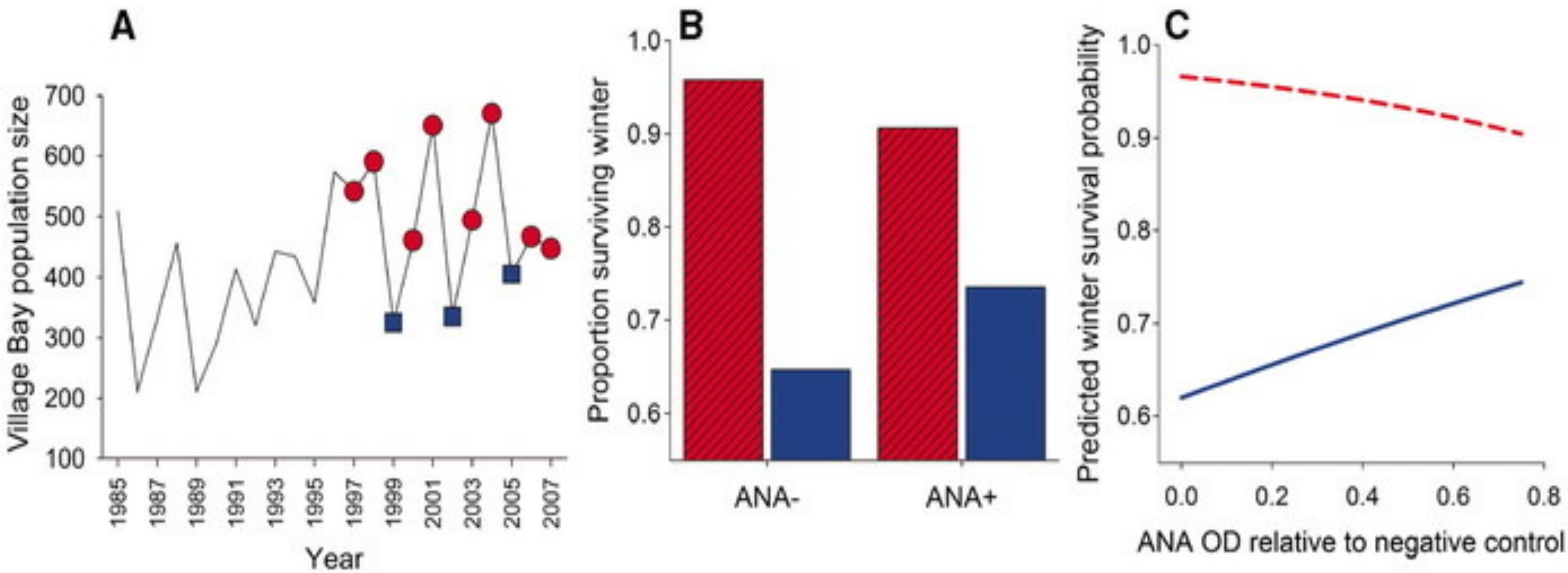

Self-reactive antibodies (ANA) **promote survival by downregulating worms in crash years** but **impede survival via immunopathology in peak years!**

Graham et al. 2010. *Science.*

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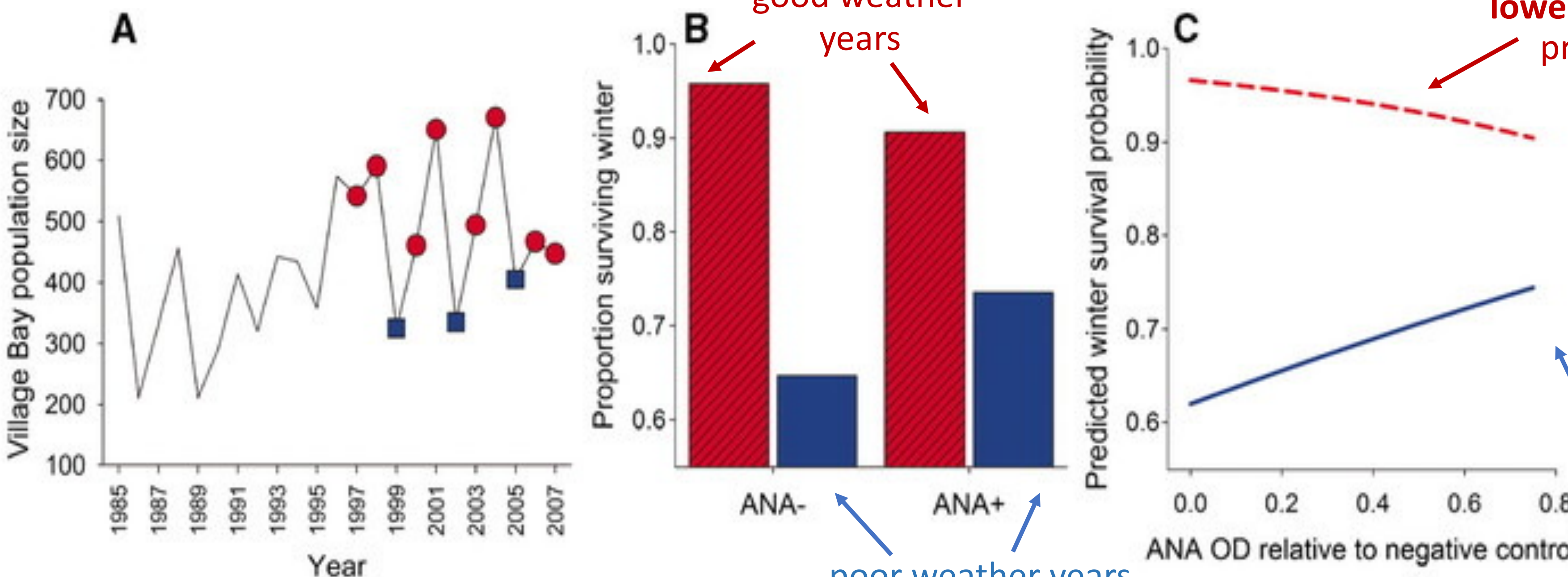

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  - In the case of zoonoses, the bulk of our measurements may be derived from a different host than the one in which the virus evolved
- Many examples of cases in which transmission is decoupled from virulence, due to more complex transmission dynamics.
  - Ex: COVID (transmission high in the respiratory tract; morbidity low in the RT)

Wk9-Lect8-Mar4

Visual settings

Edit

In the 2014 West African Ebola epidemic, several people contracted infections during burial services by encountering the virus via contact with a recently-deceased patient. What would we expect for selective pressures on virulence evolution?

A We know Ebola beca…	0%
B Because virulenc…	0%
C The virus will be selected…	0%
D The virus is in a stutteri…	0%

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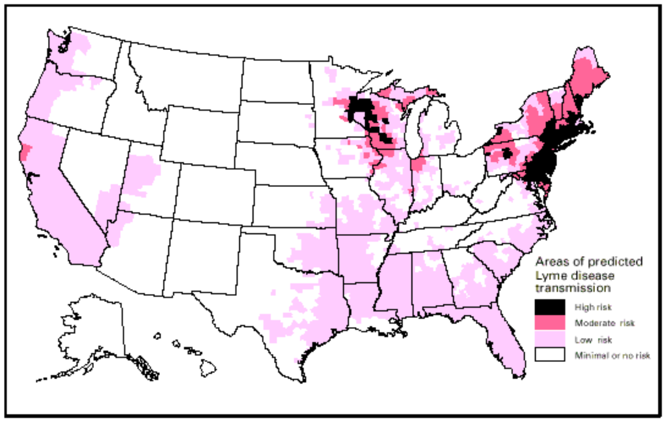
Disease dynamics in the **broader community**

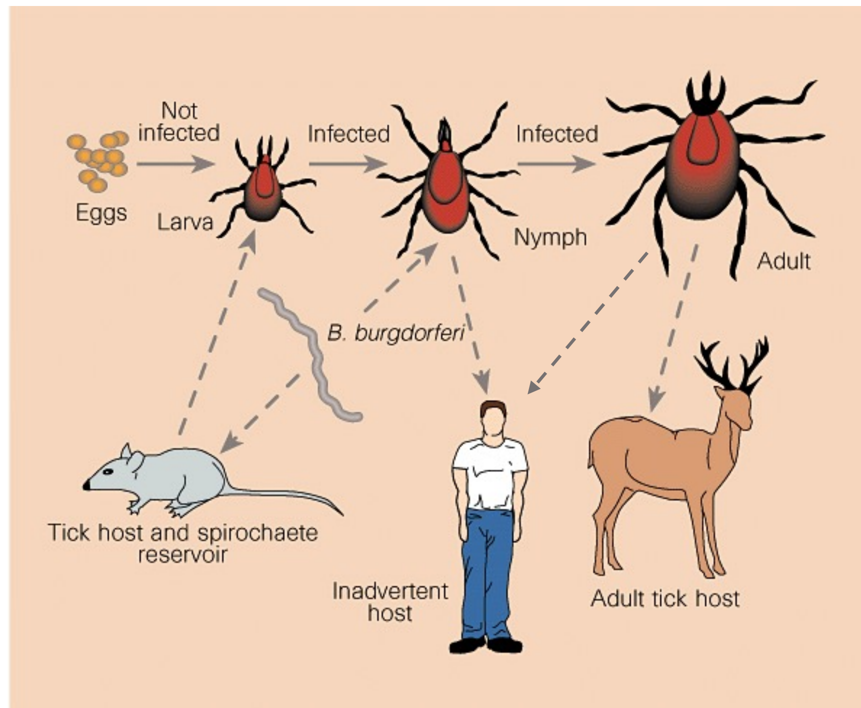
Disease dynamics in the **broader community**

Example: Lyme Disease

National Lyme disease risk map with four categories of risk



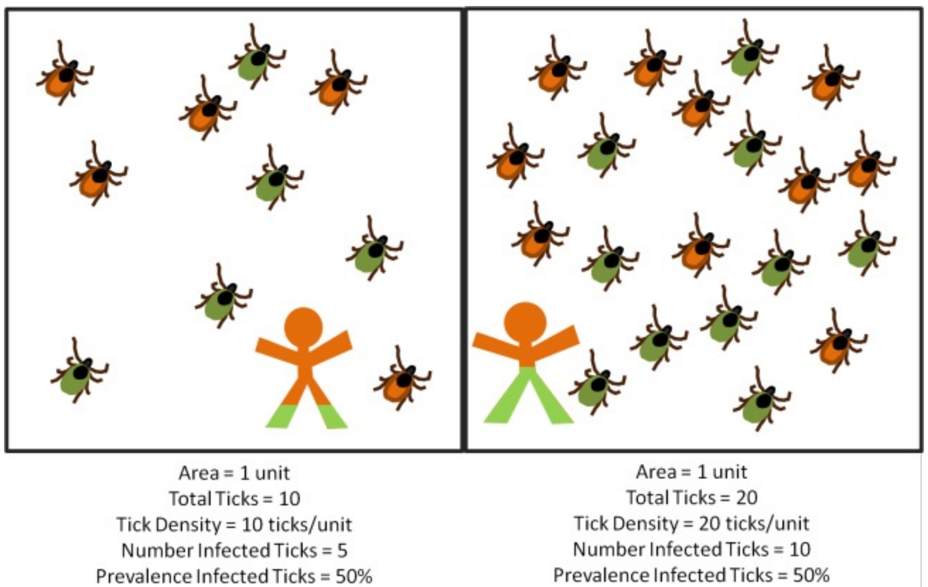
- Lyme disease is a vector-borne disease caused by the bacterium, *Borrellia burgdoferi,* vectored by *Ixodes* especially *Ixodes scapularis*) ticks.
- Nymph ticks are borne in the spring, feed on small mammal hosts through the summer, then reproduce (particularly on deer) in the fall before going dormant in the winter.
- Human cases are largely concentrated in the spring and summer and result from infected tick bites.



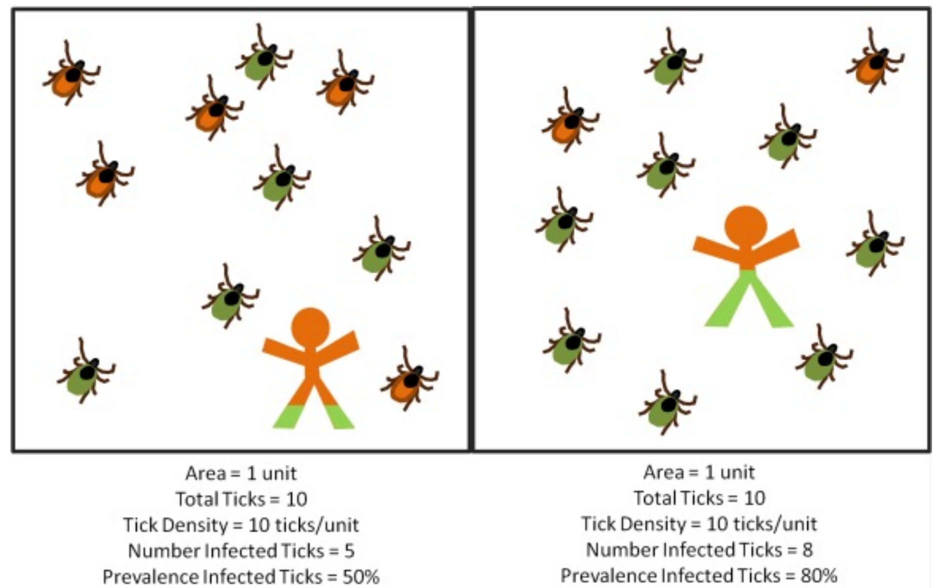
Barbour and Zuckert 1997. *Nature.*

Human infection probability varies with both the **density of infected ticks** and the **prevalence of Lyme** in the tick population.

Scenario 1: Density of infected ticks



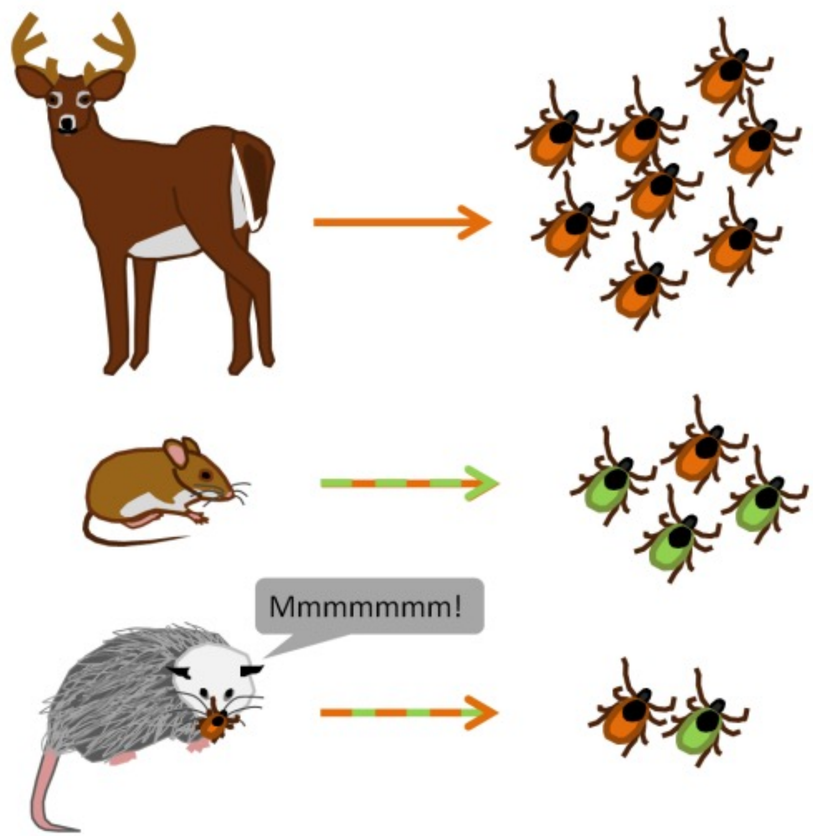
Scenario 2: Prevalence of Lyme in tick population



lime = infected with Lyme

parasiteecology.wordpress.com

The broader **wildlife community impacts Lyme disease risk** for humans



(1) Wildlife hosts vary in the extent to which they offer blood meals to ticks, thereby modulating tick abundance.

(2) Wildlife hosts also vary in their permissibility to *B. burgdoferi* infection.

lime = **infected with Lyme**

parasiteecology.wordpress.com

The **dilution effect** highlights buffering effects of **biodiversity on disease transmission**.

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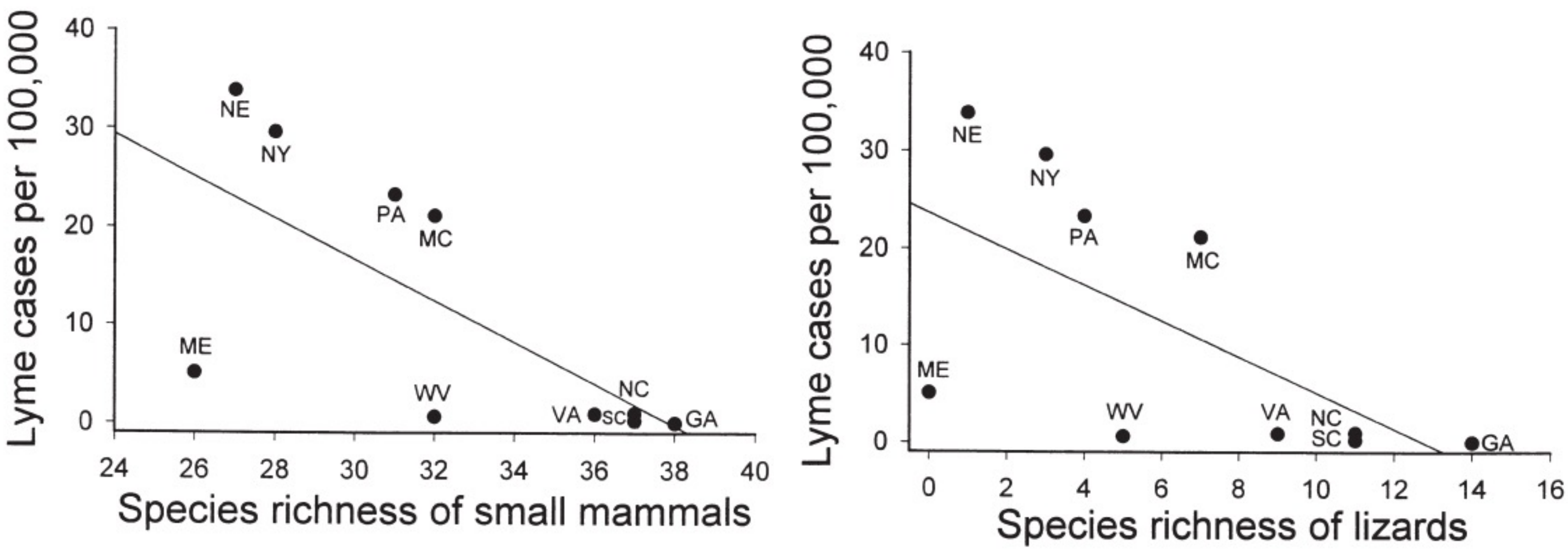
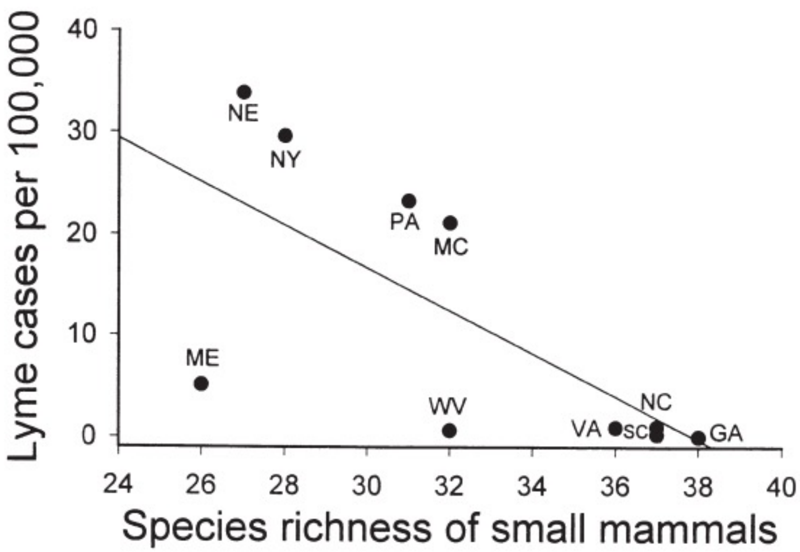


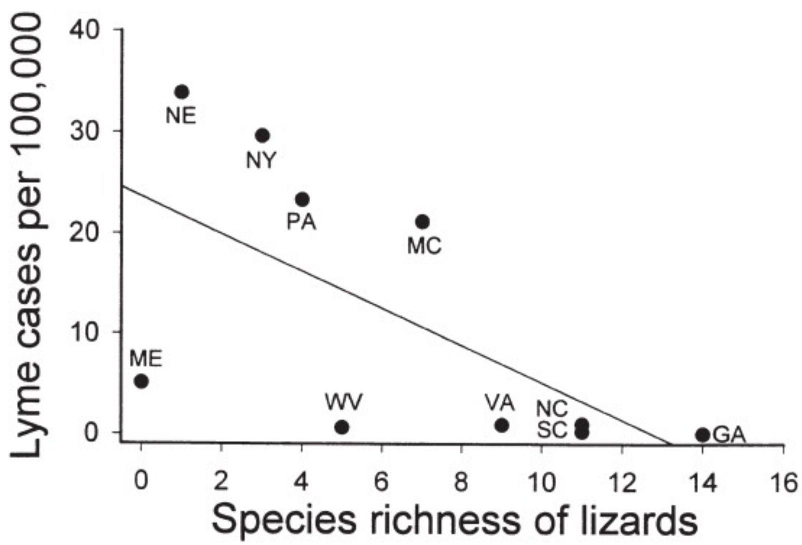
In the case of Lyme, many examples demonstrate a **negative correlation between host biodiversity and Lyme prevalence**.

Ostfeld and Keesing. 2000. *Conservation Biology.*

The **dilution effect** highlights buffering effects of **biodiversity on disease transmission**.

While a popular concept, it only holds in select cases!

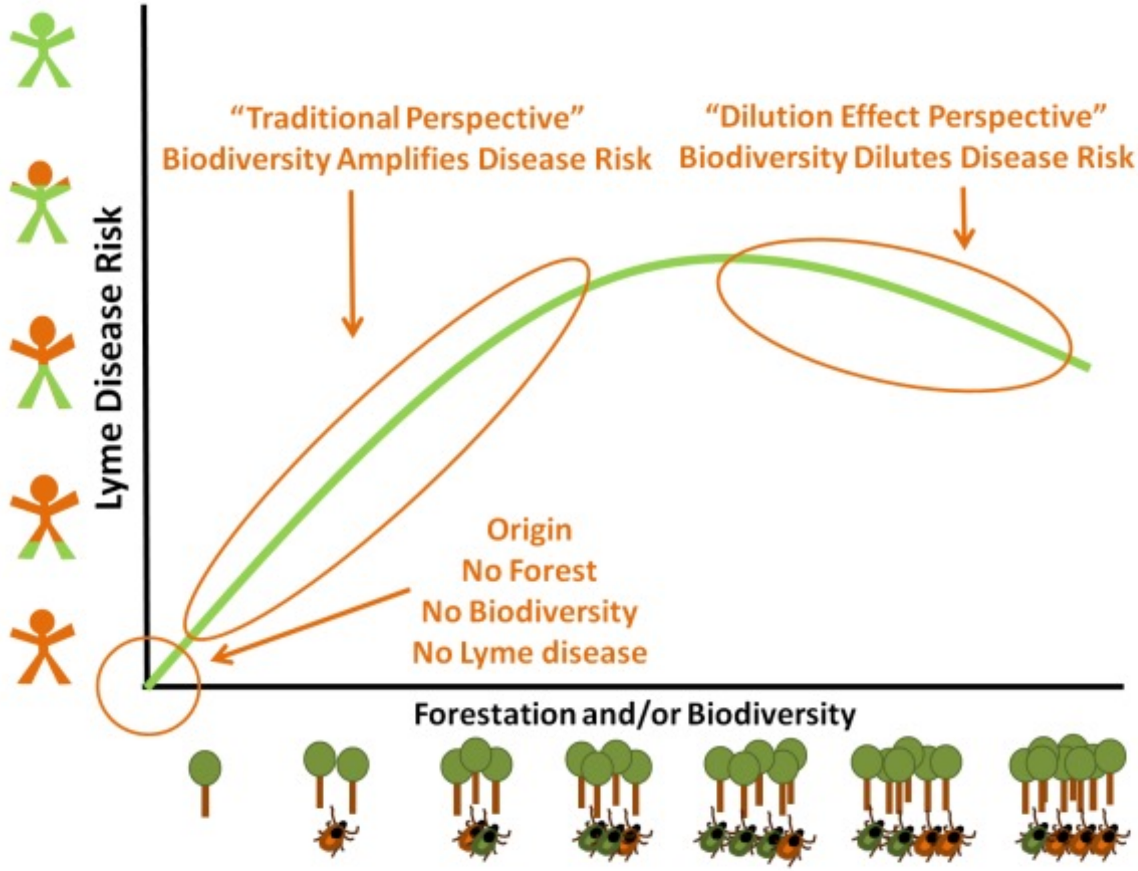




In the case of Lyme, many examples demonstrate a **negative correlation between host biodiversity and Lyme prevalence**.

Patterns depend on the context of the wildlife community!

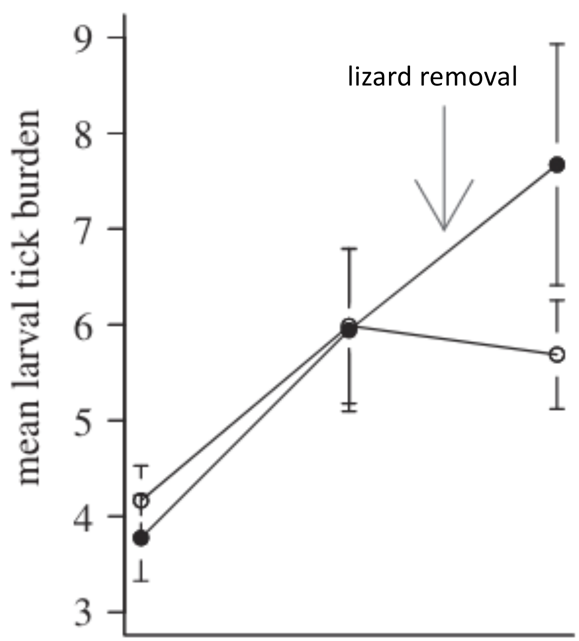
Human infection probability varies with both the **density of infected ticks** and the **prevalence of Lyme** in the tick population.



parasiteecology.wordpress.com

"**Zooprophylaxis**" is the diversion of pathogen-transmitting arthropods from humans to animals

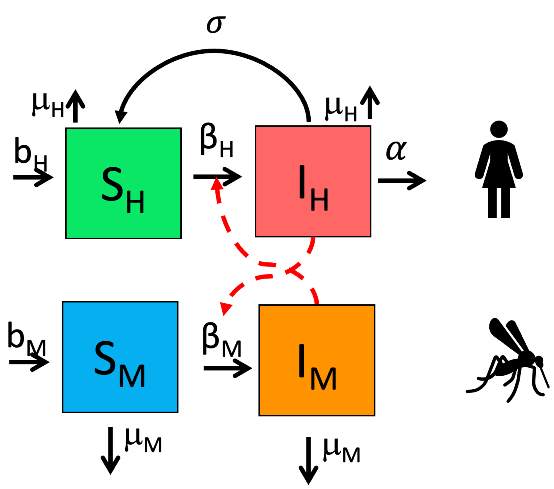
Tick burden on woodrats in Marin County, CA following removal of western fence lizard (*Sceloporus occidentalis*) at the end of the 2007 year in experimental (solid) and control (open circle) plots

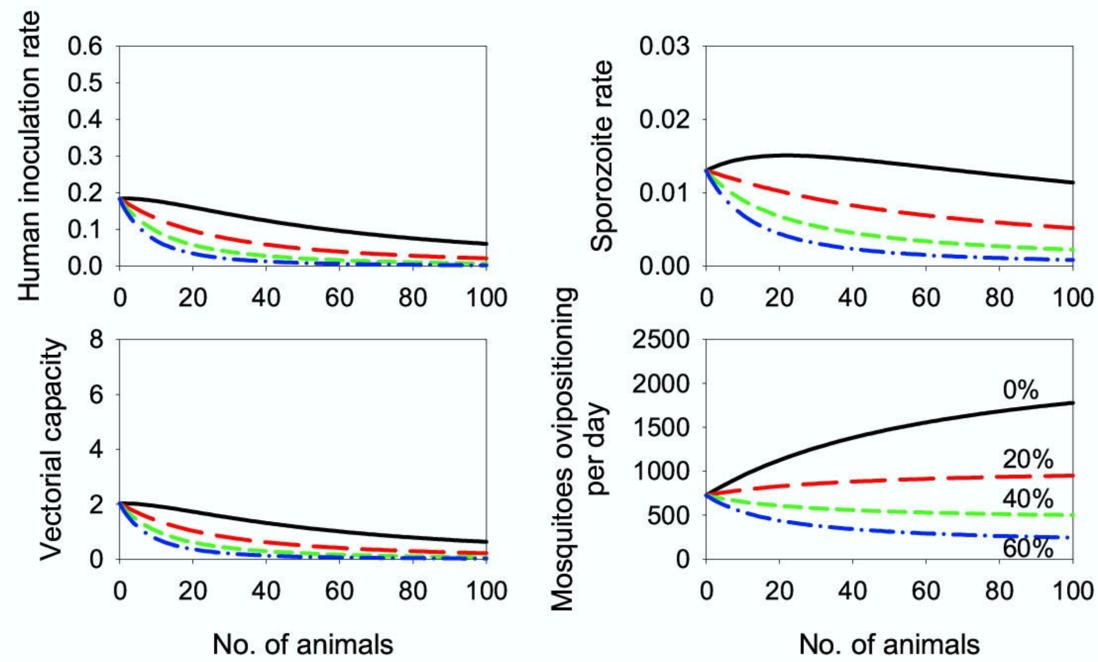


Sceloporus occidentalis

Swei et al. 2011. *Proc Roy Soc B.*

"**Zooprophylaxis**" has been suggested for malaria control – but only works in cases by which livestock are used as bait to draw mosquitoes closer to insecticides.



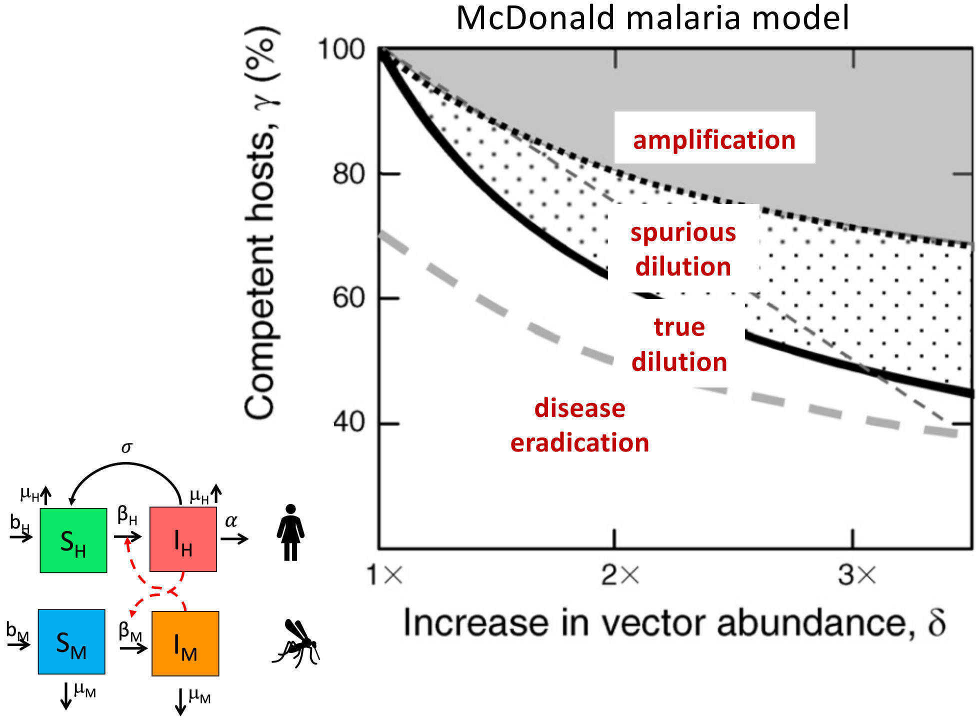


Black, red, green, blue = 0, 20, 40 or 60% chance of mosquito being killed as a result of feeding on animals

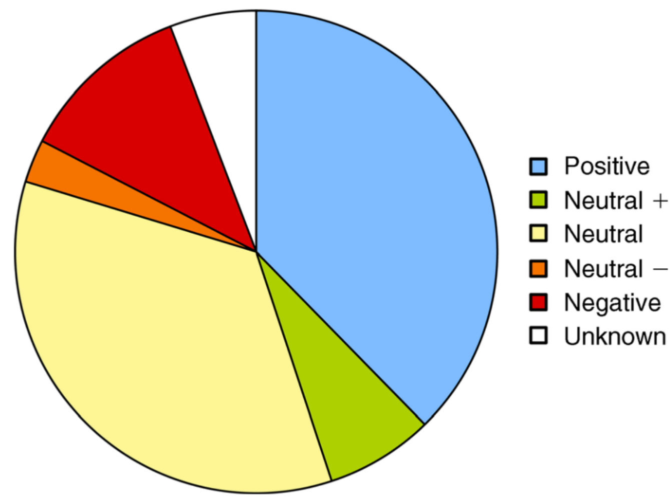
Asale et al. 2017. *Malaria Journal*

Understanding the **underlying transmission dynamics** of the system can help predict how **wildlife biodiversity** might **amplify or dilute** human disease risk in different contexts.

Results from an extended Ross-McDonald malaria model



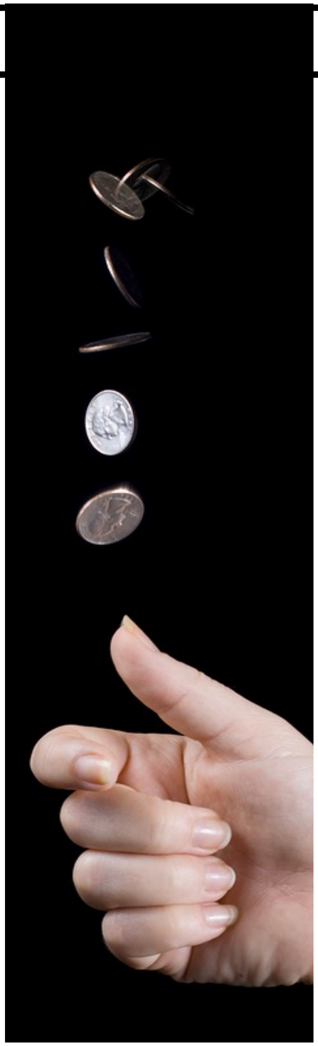
Meta-analysis of biodiversity impacts on 69 common human pathogens



Wood et al. 2014. *Ecology*

Population Biology

Conservation Biology	Disease Ecology
• **Goal:**	• **Goal:**
• protect **populations** from **extinction**	• protect **populations** from disease via pathogen **extinction**



Population Biology

Conservation Biology	Disease Ecology

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)

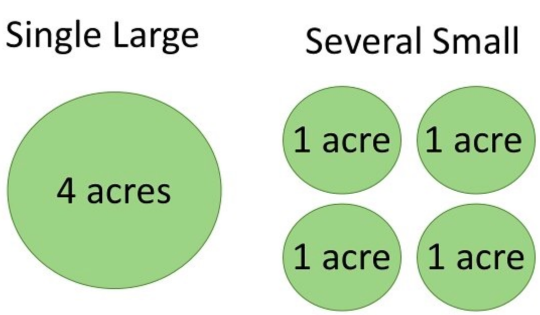
CCS = the minimum number of hosts needed to sustain endemic transmission of a pathogen indefinitely into the future

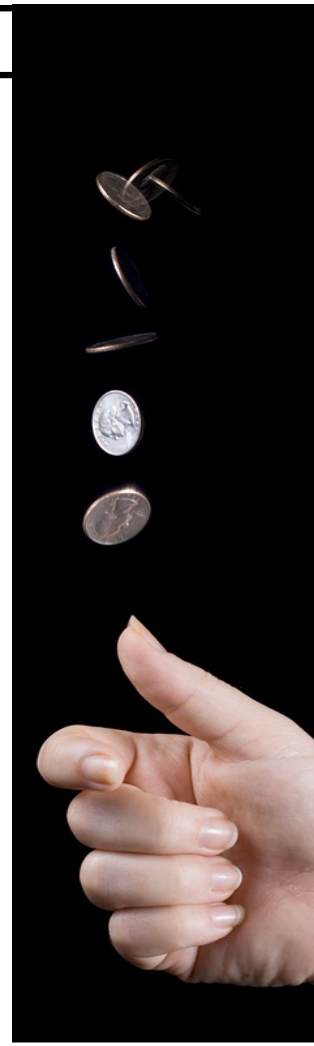
Population Biology

Conservation Biology	Disease Ecology

Conservation Biology

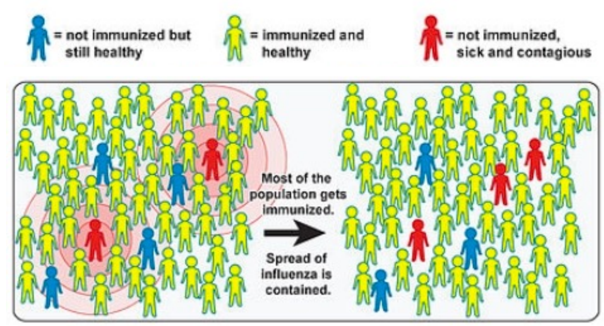
- **Goal:**
  - protect **populations** from **extinction**
- **Concept:**
  - **Minimum Viable Population** size (MVP)
- **Approach:**
  - protected area **reserves**





Disease Ecology

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



Wk9-Lect8-Mar4

Visual settings

Edit

Imagine a forest with endemic Lyme disease and a high population of deer. What outcome would you expect if the deer were experimentally removed, but the increase in grass availability leads to an explosion in the population of white-footed mice?

A. Lyme risk to humans …	0%
B. Lyme risk to humans …	0%
C. Lyme risk will not change	0%
D. We'll need to build a mo…	0%

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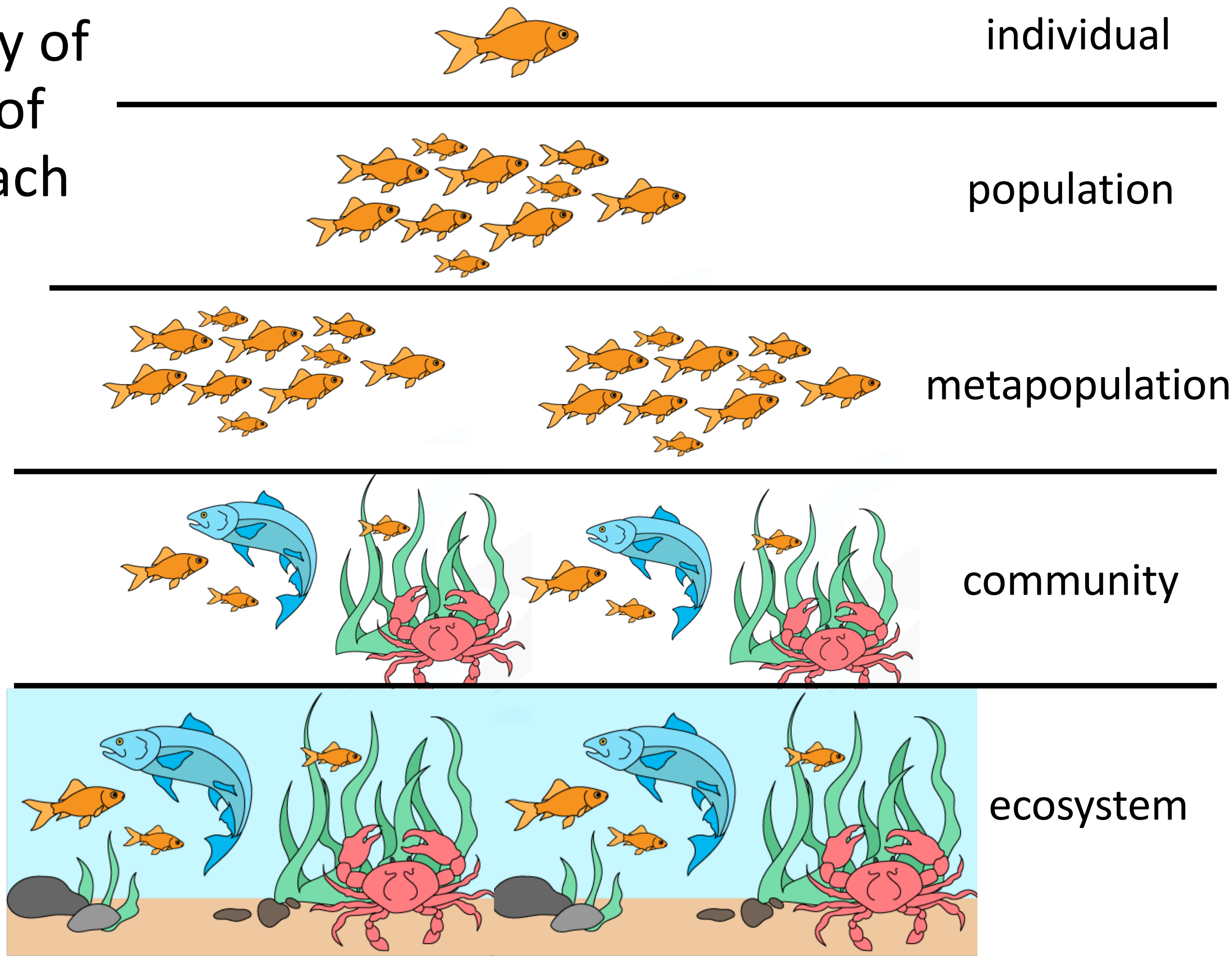
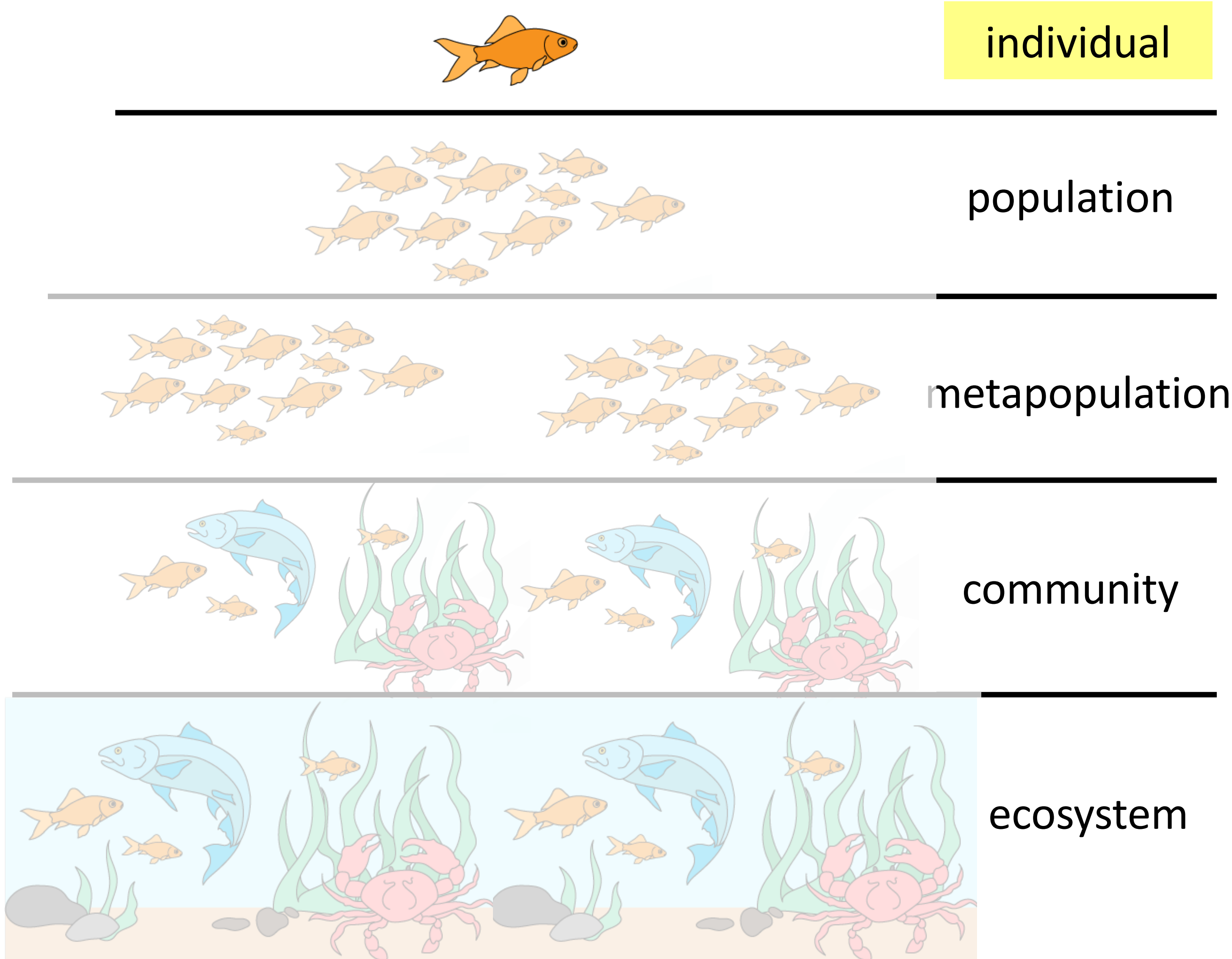
Ecology is the study of the **interactions** of **organisms** with each other and their **environment.**



Individual:
metabolism, behavior, life history.

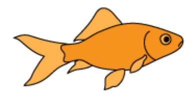
interactions of an individual with the environment



Individual:
metabolism,
behavior, life history.

Dynamic Energy Budget (DEB) Model

How does a fish's metabolism ***change*** *with temperature?*



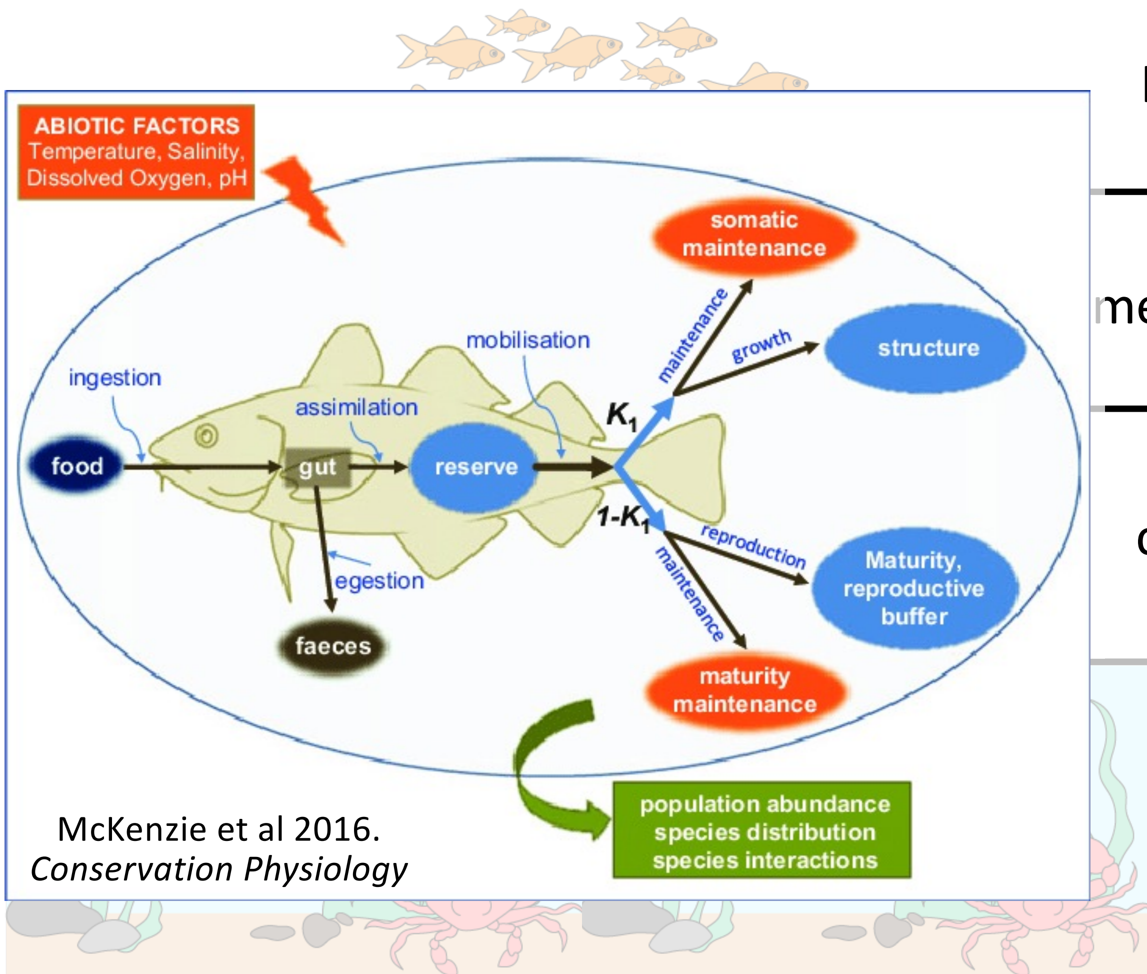
individual

population

metapopulation

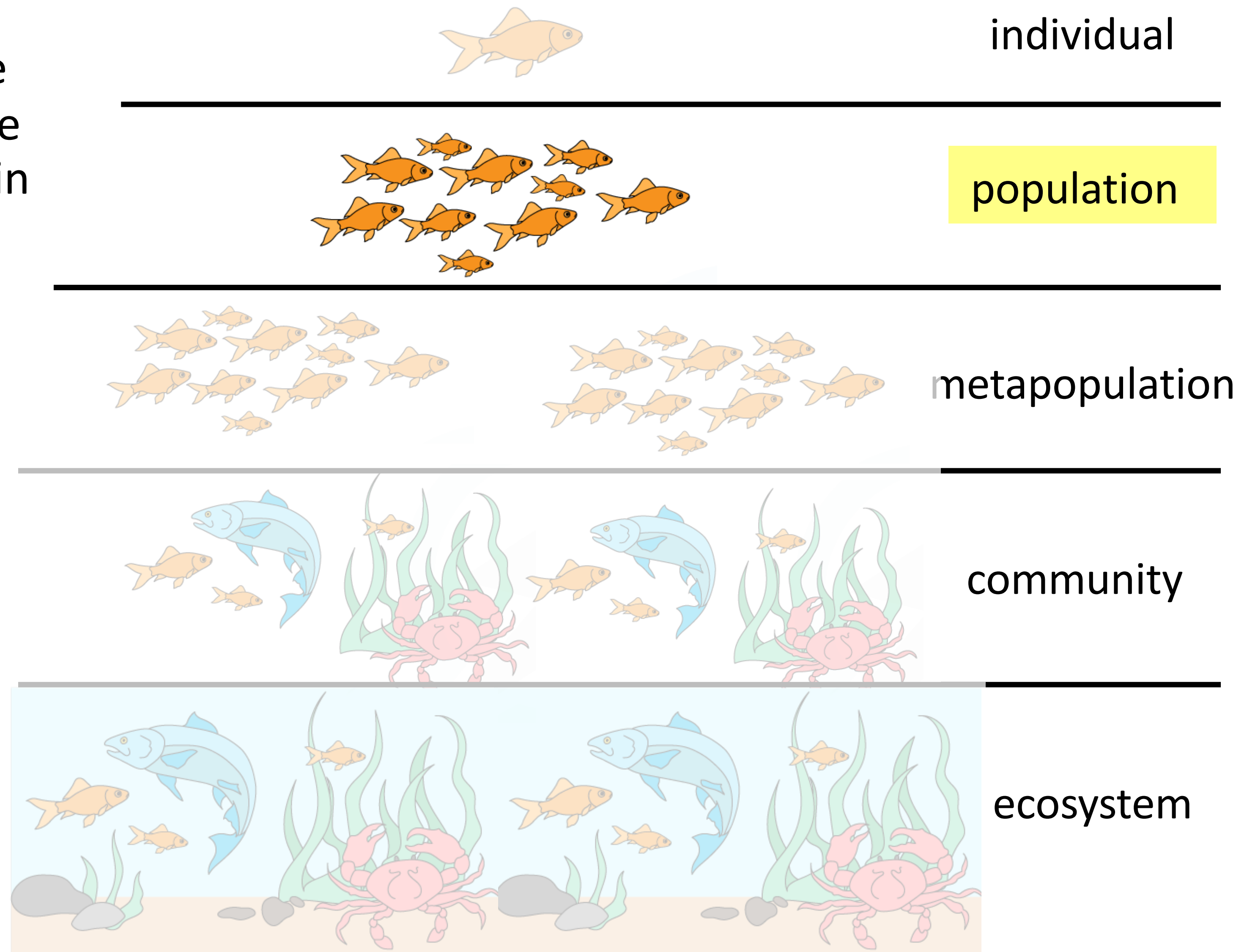
community

ecosystem



McKenzie et al 2016. *Conservation Physiology*

Population = multiple individuals of the same species (**conspecifics**) in the same habitat



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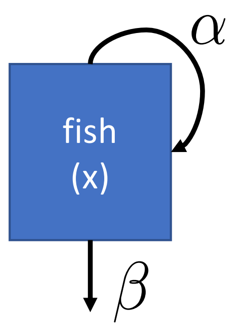
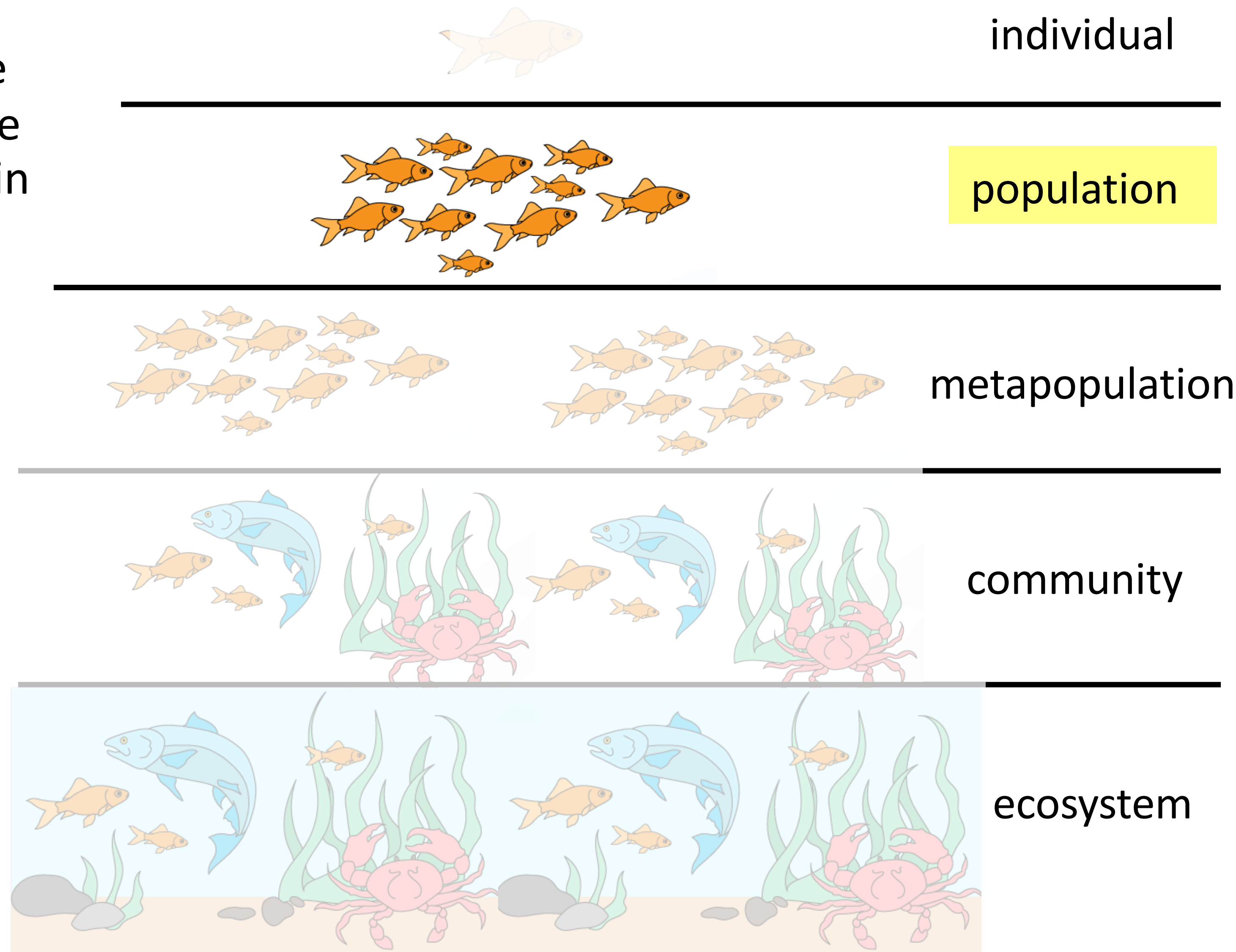
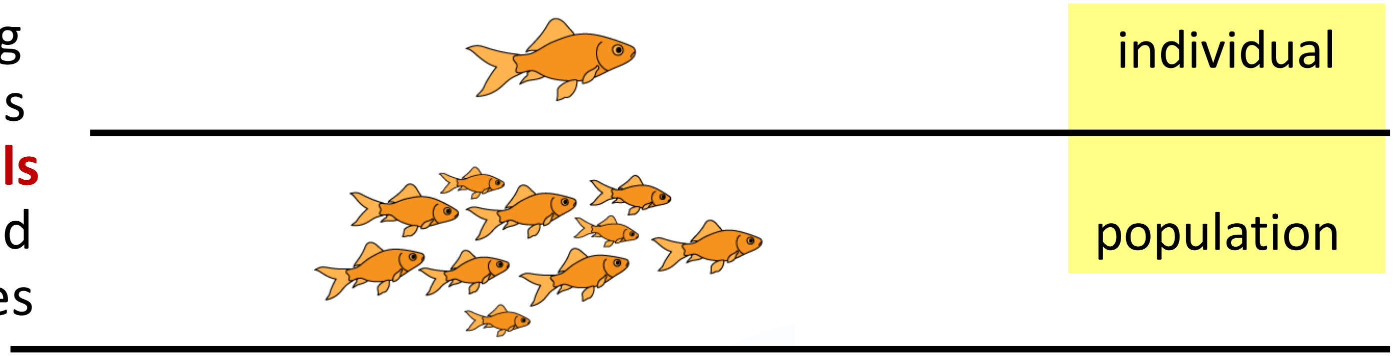
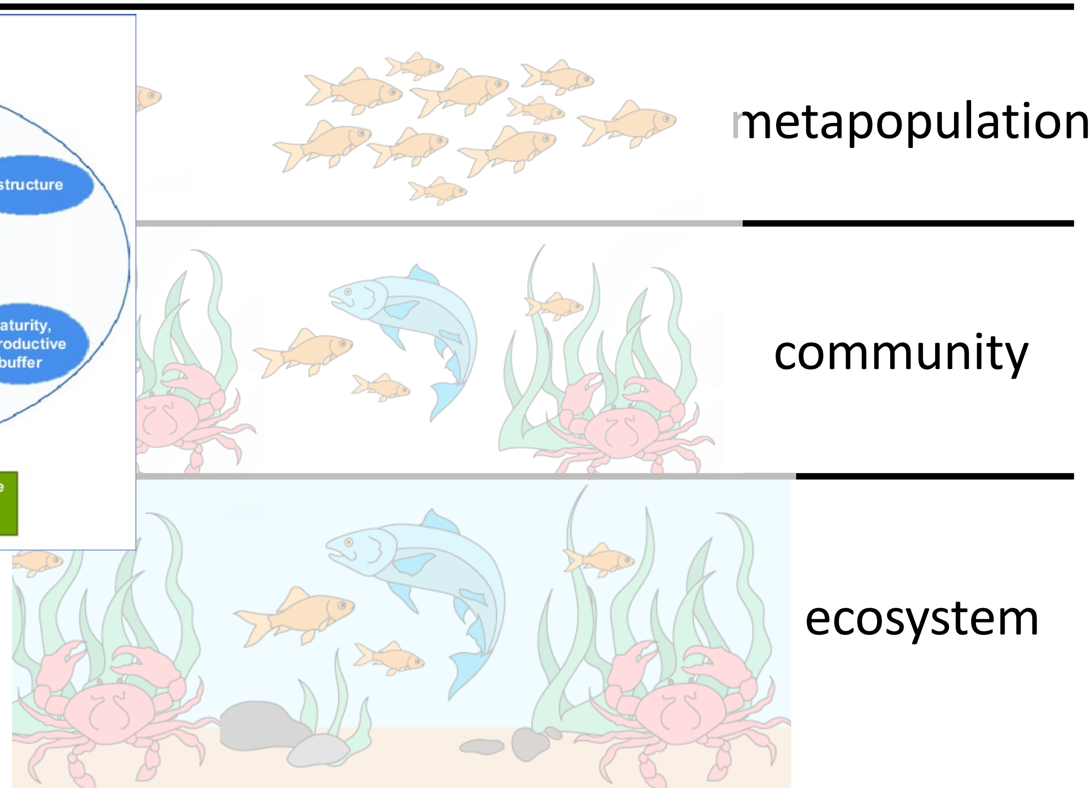


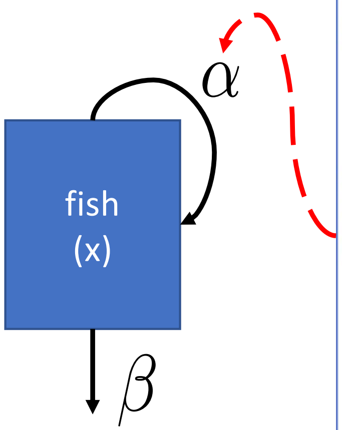
*How does the abundance of fish **change** through time?*

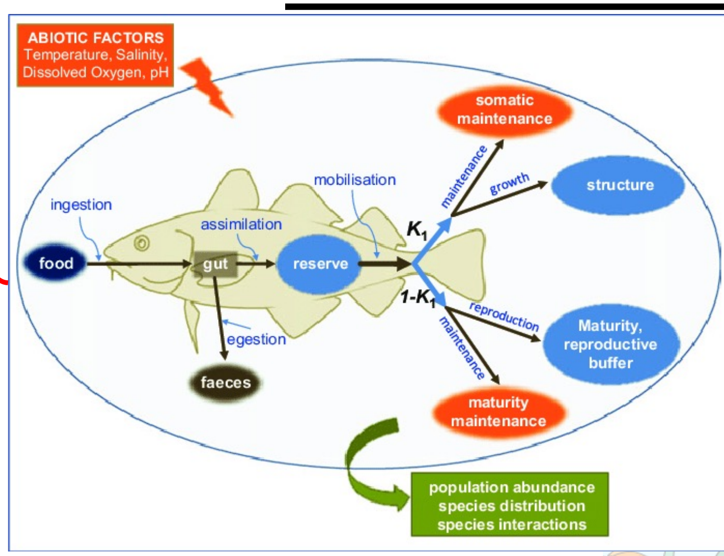


Nested Models, including a class of model known as **Integral Projection Models** (IPMs), link individual- and population-level processes



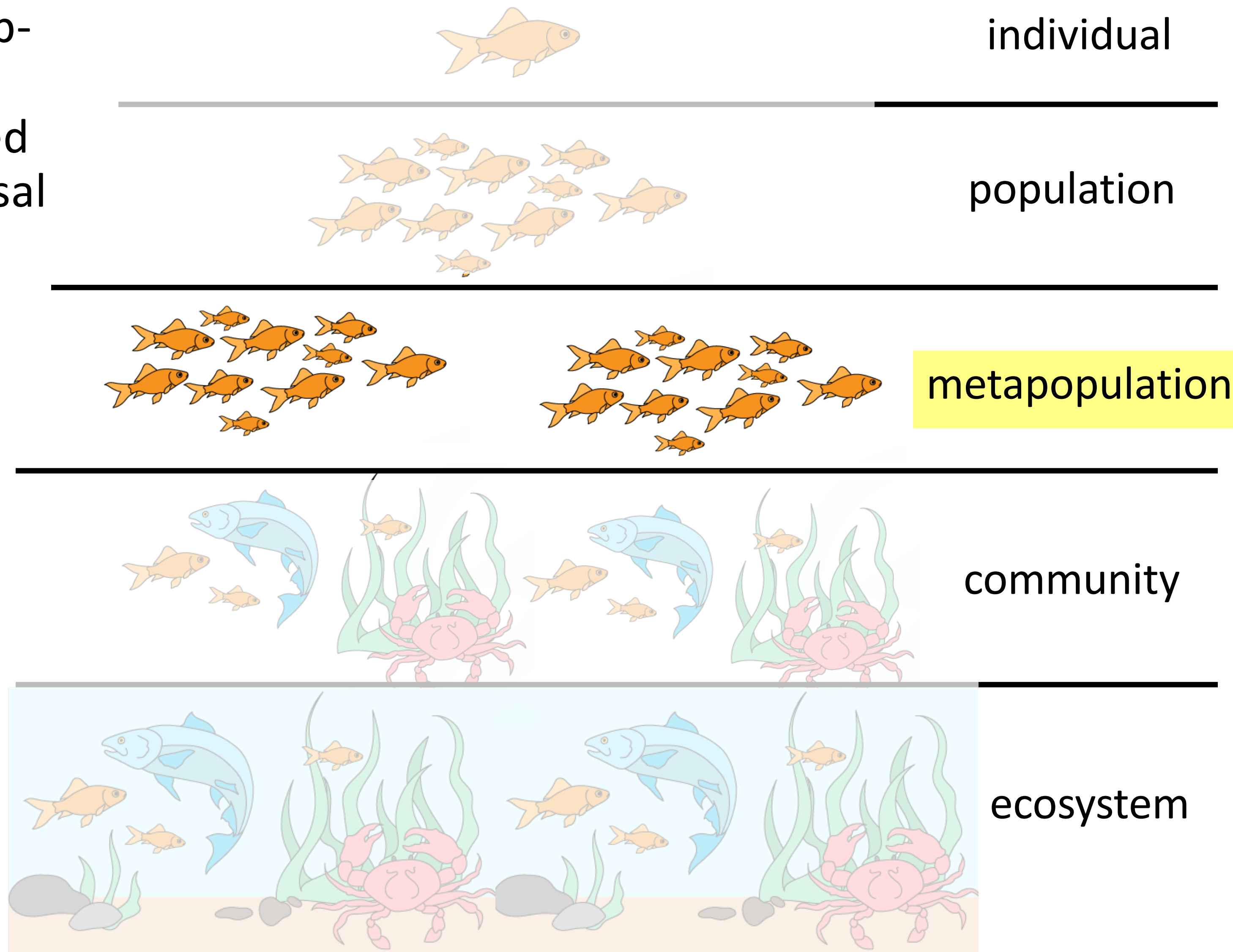






How does the abundance of fish ***change*** *through time as temperature* ***changes*** *metabolism?*

Metapopulation = sub-populations of conspecifics connected by migration or dispersal



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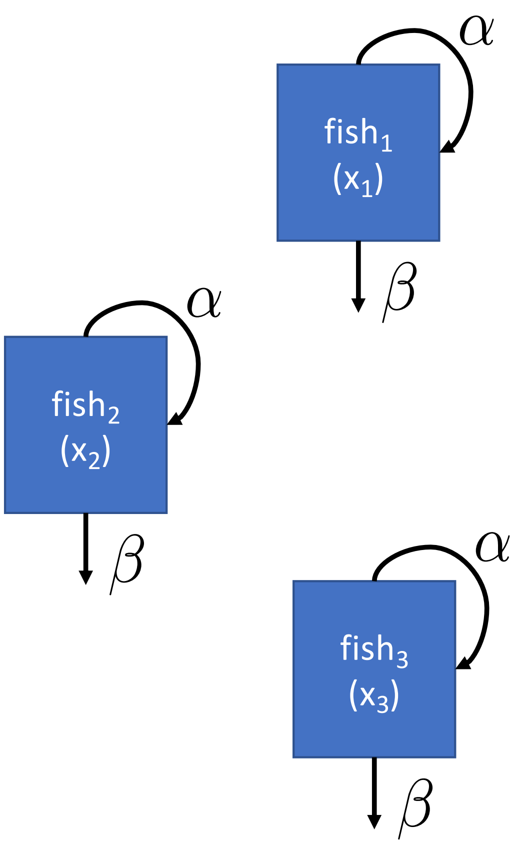
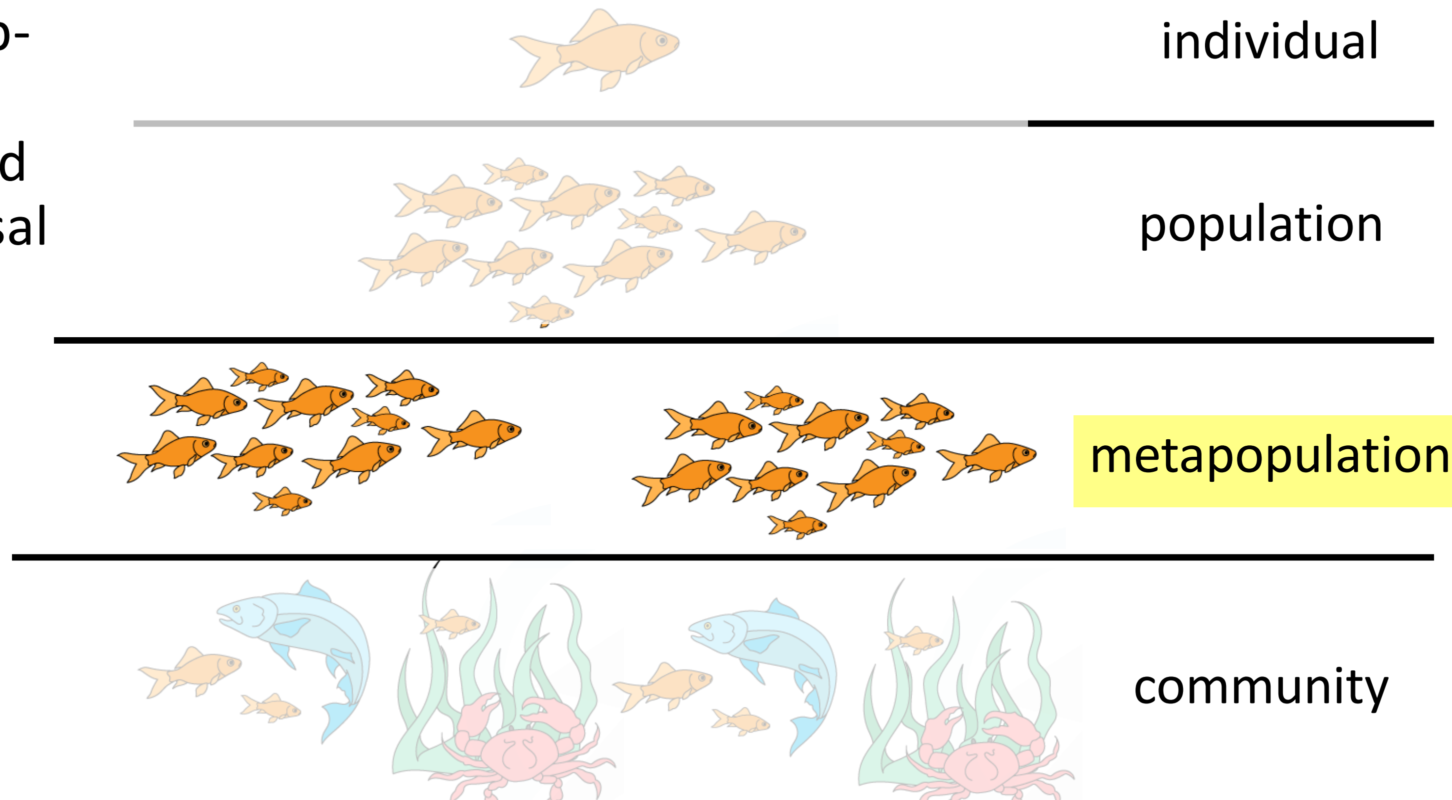
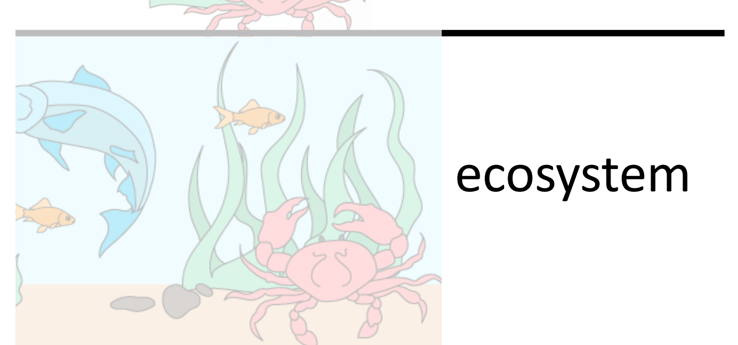




*How do the abundances of different subpopulations of the same fish **vary** in space [and time]?*



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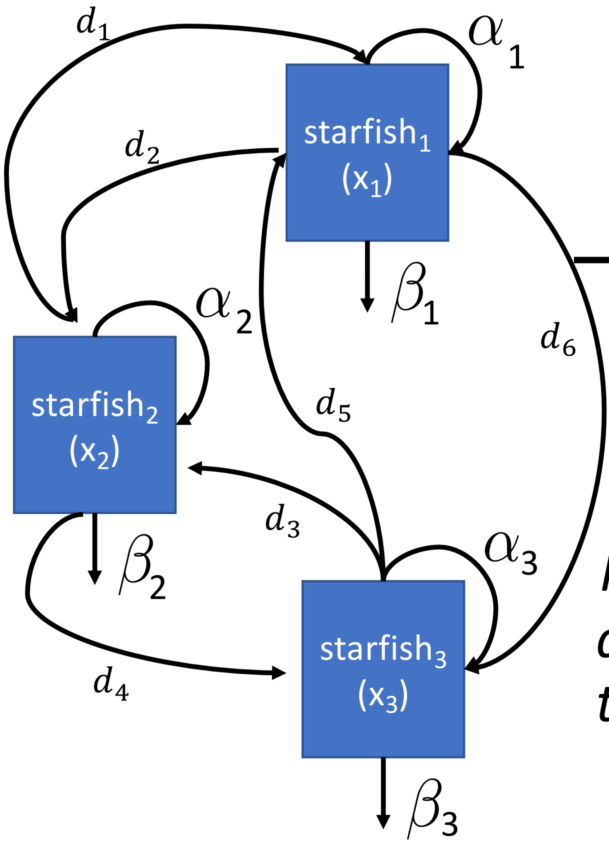

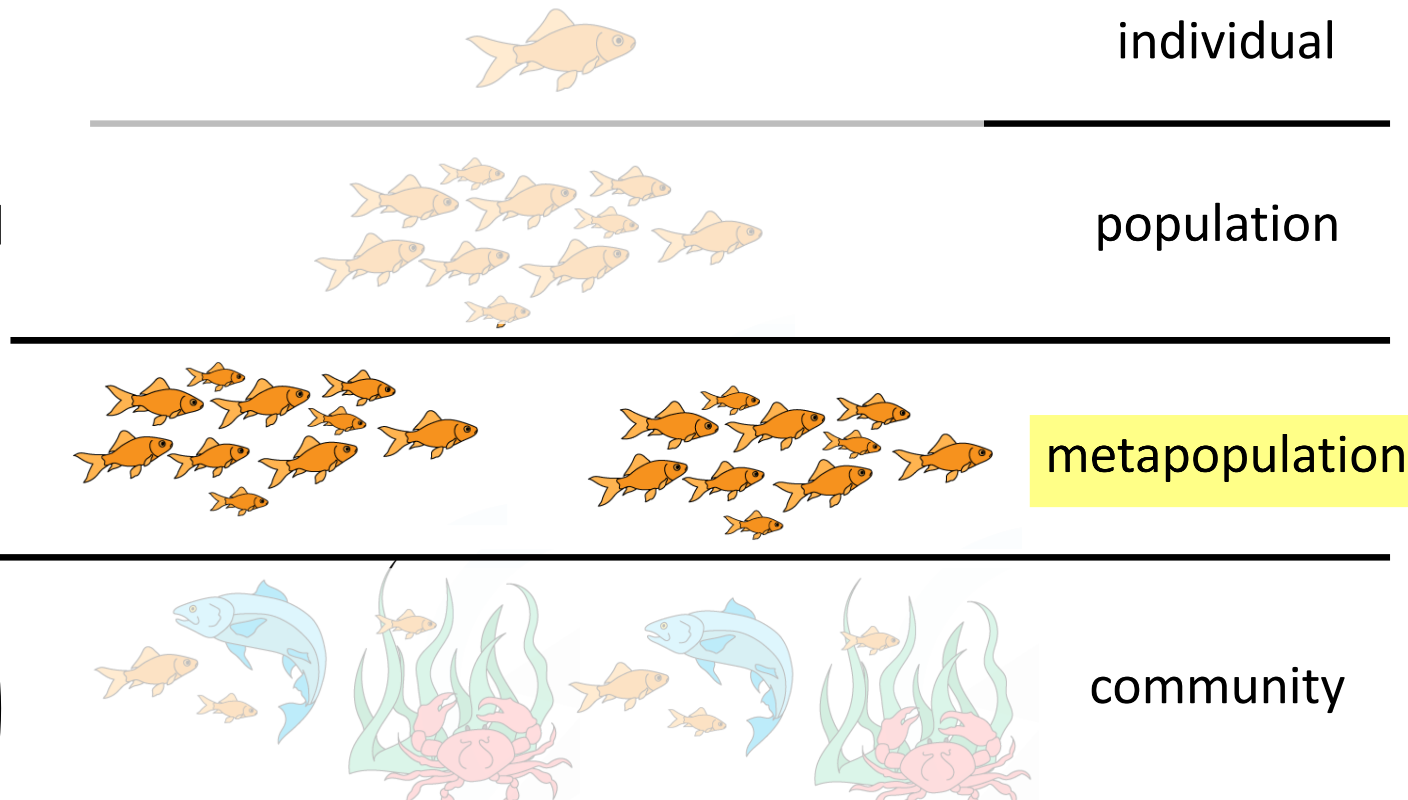

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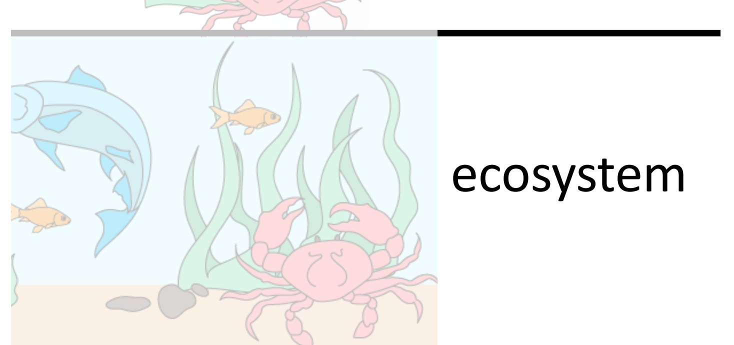
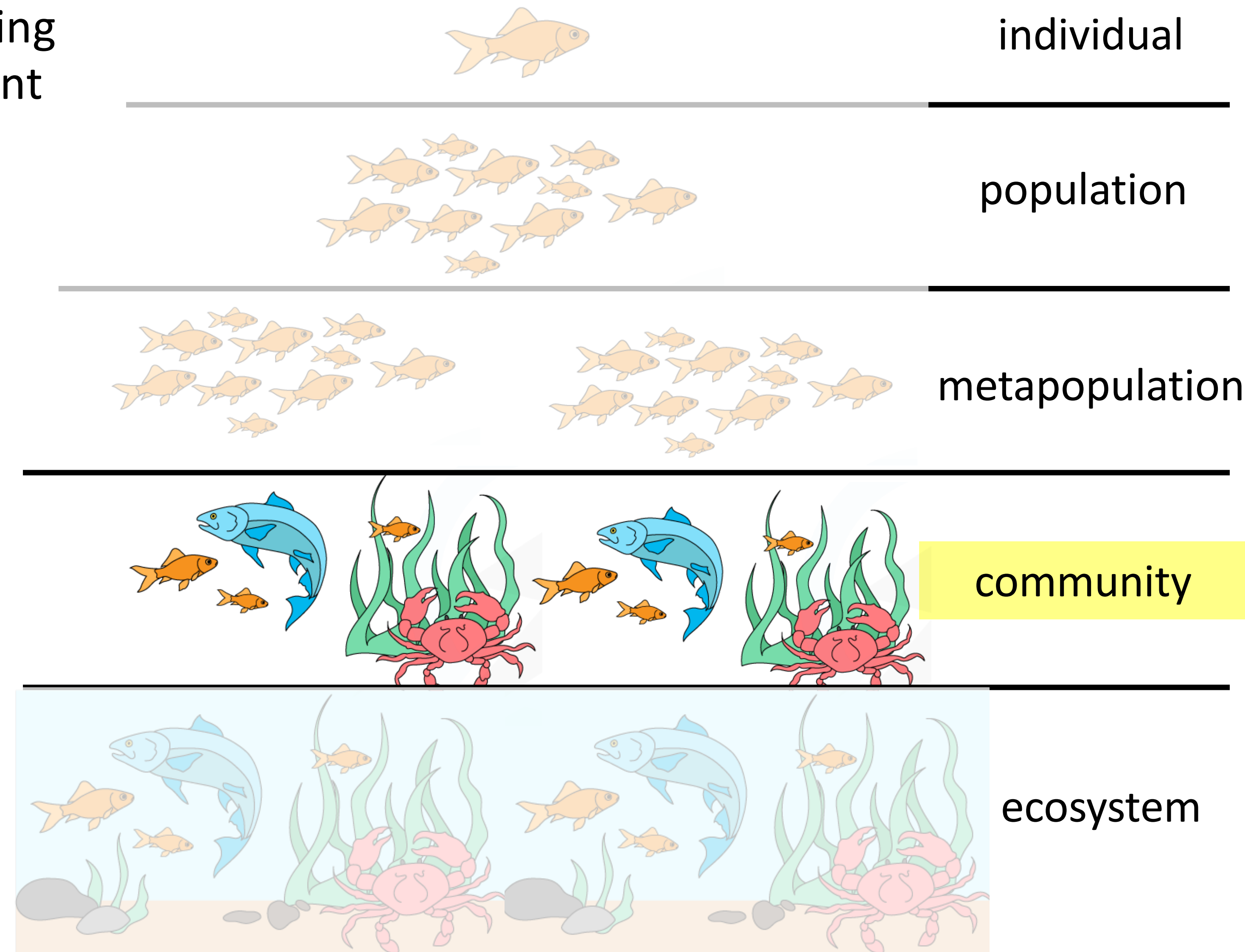


Community = interacting populations of different species



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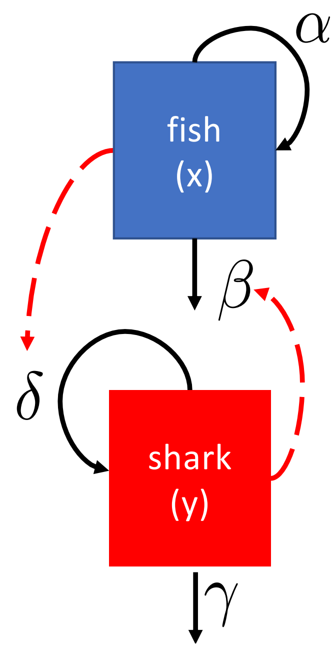
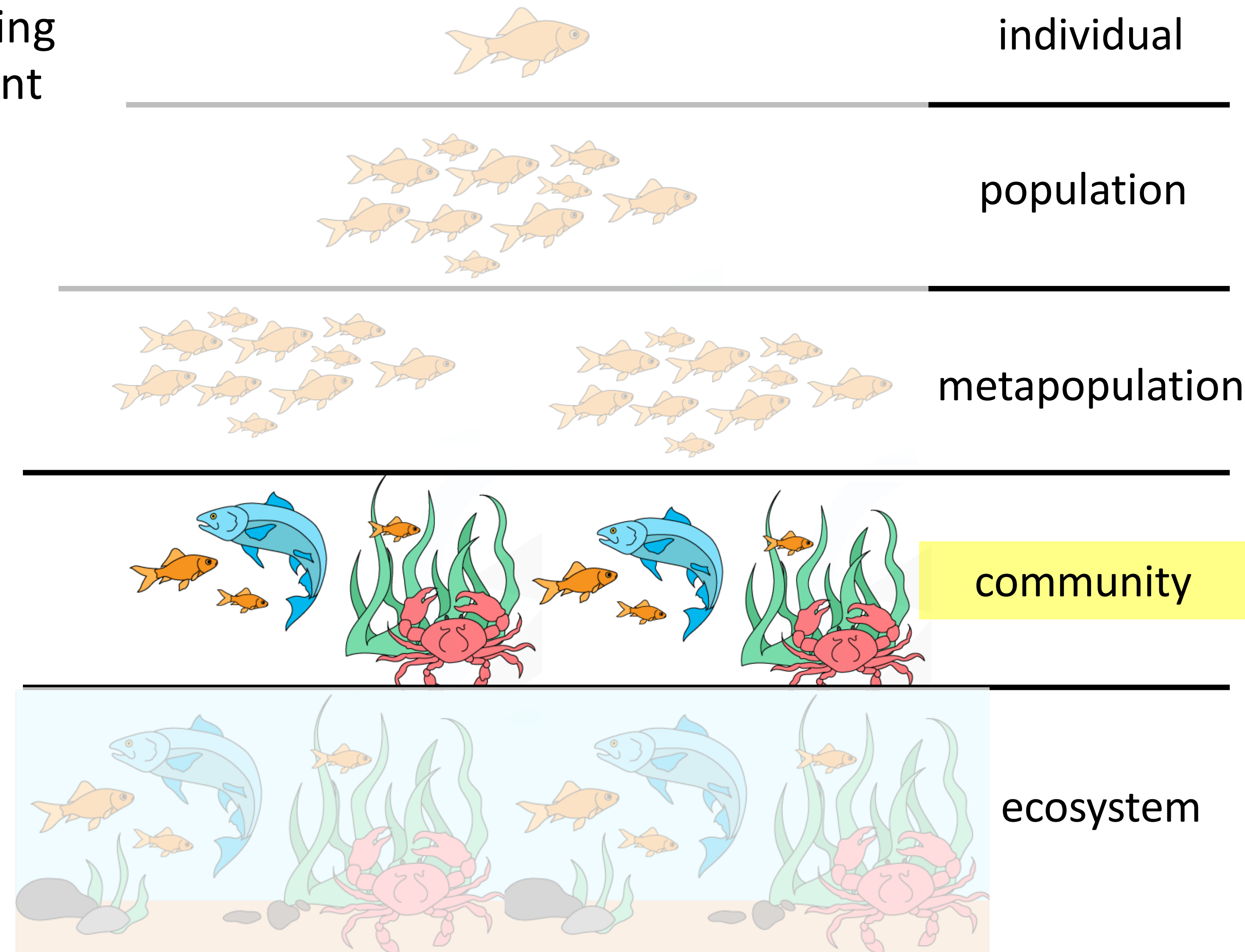
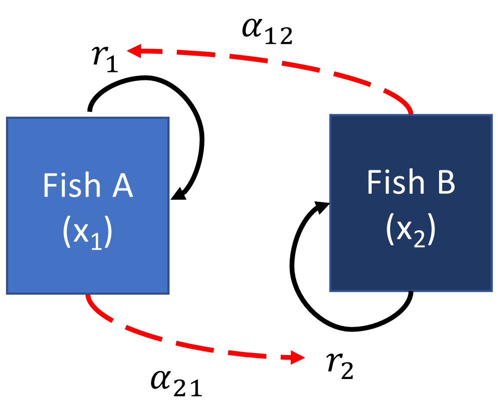


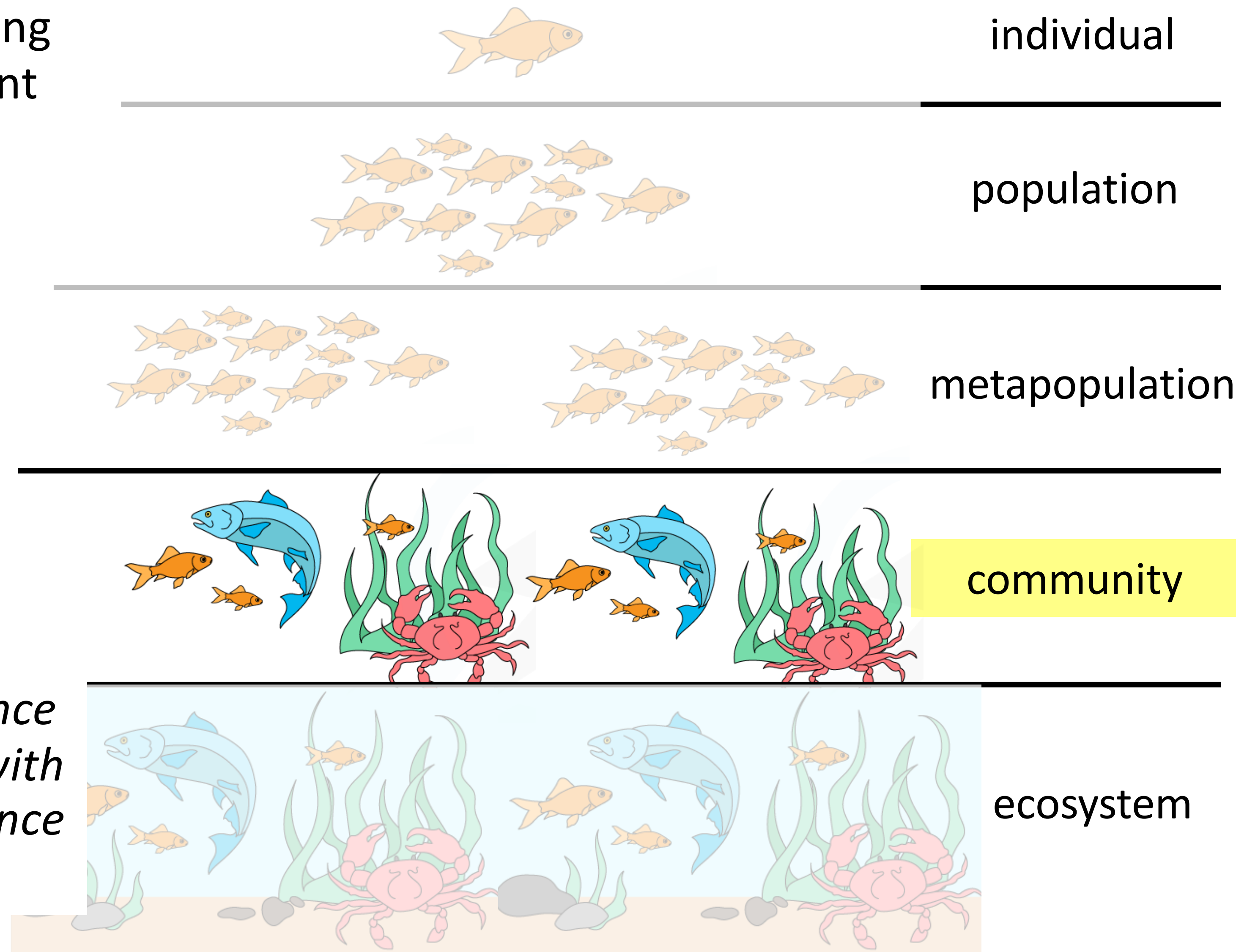
*How does fish abundance **vary** with **changes** in shark abundance?*



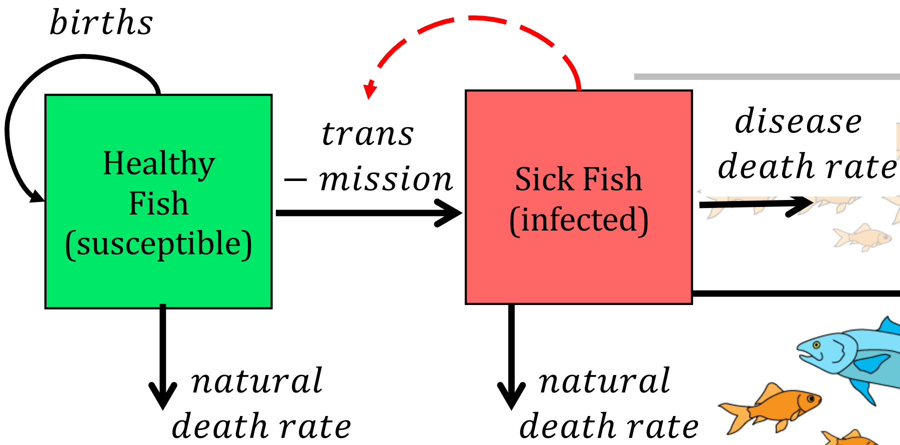
Community = interacting populations of different species

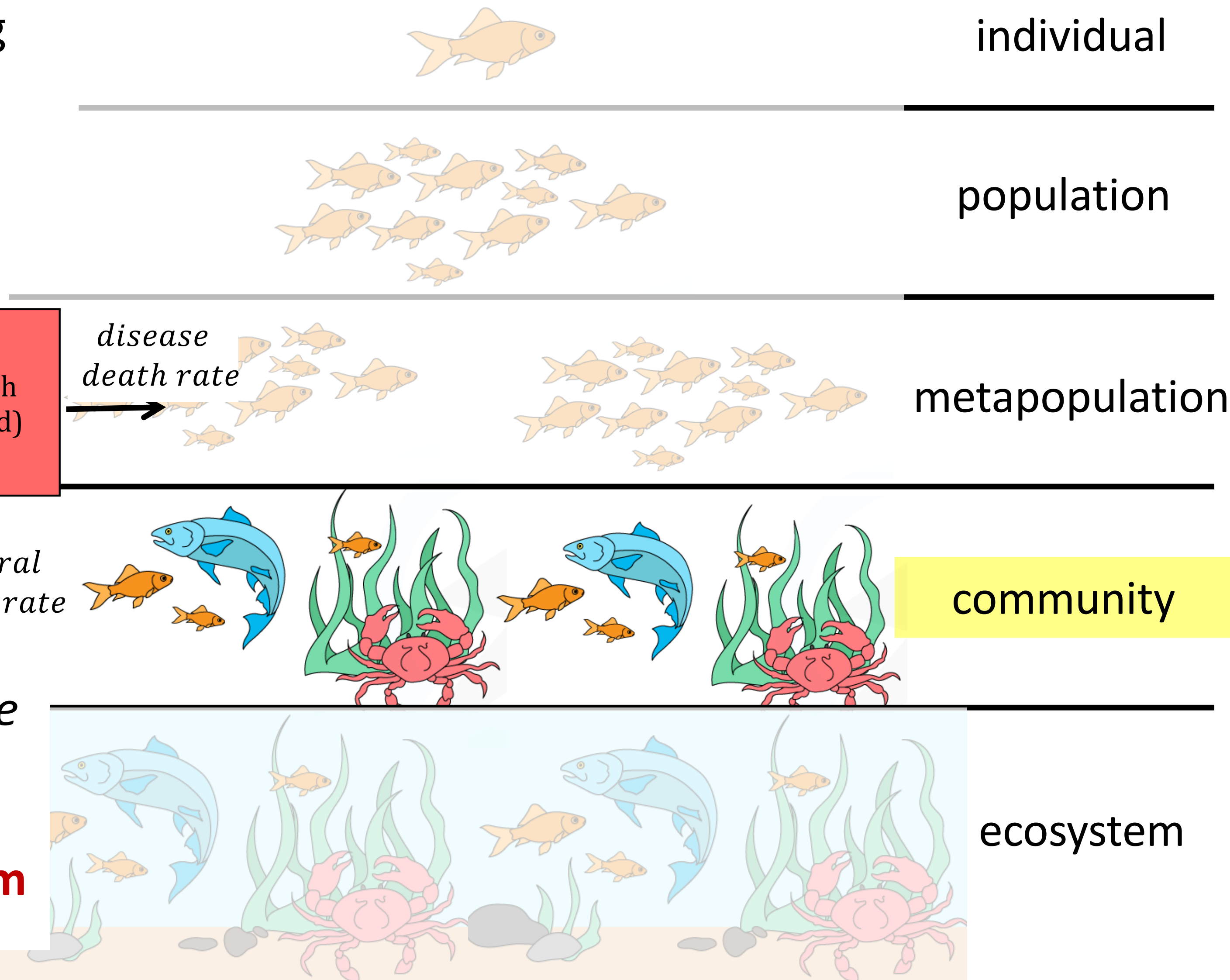


How does the abundance of ***fish species A*** *vary with changes in the abundance of* ***fish species B****?*



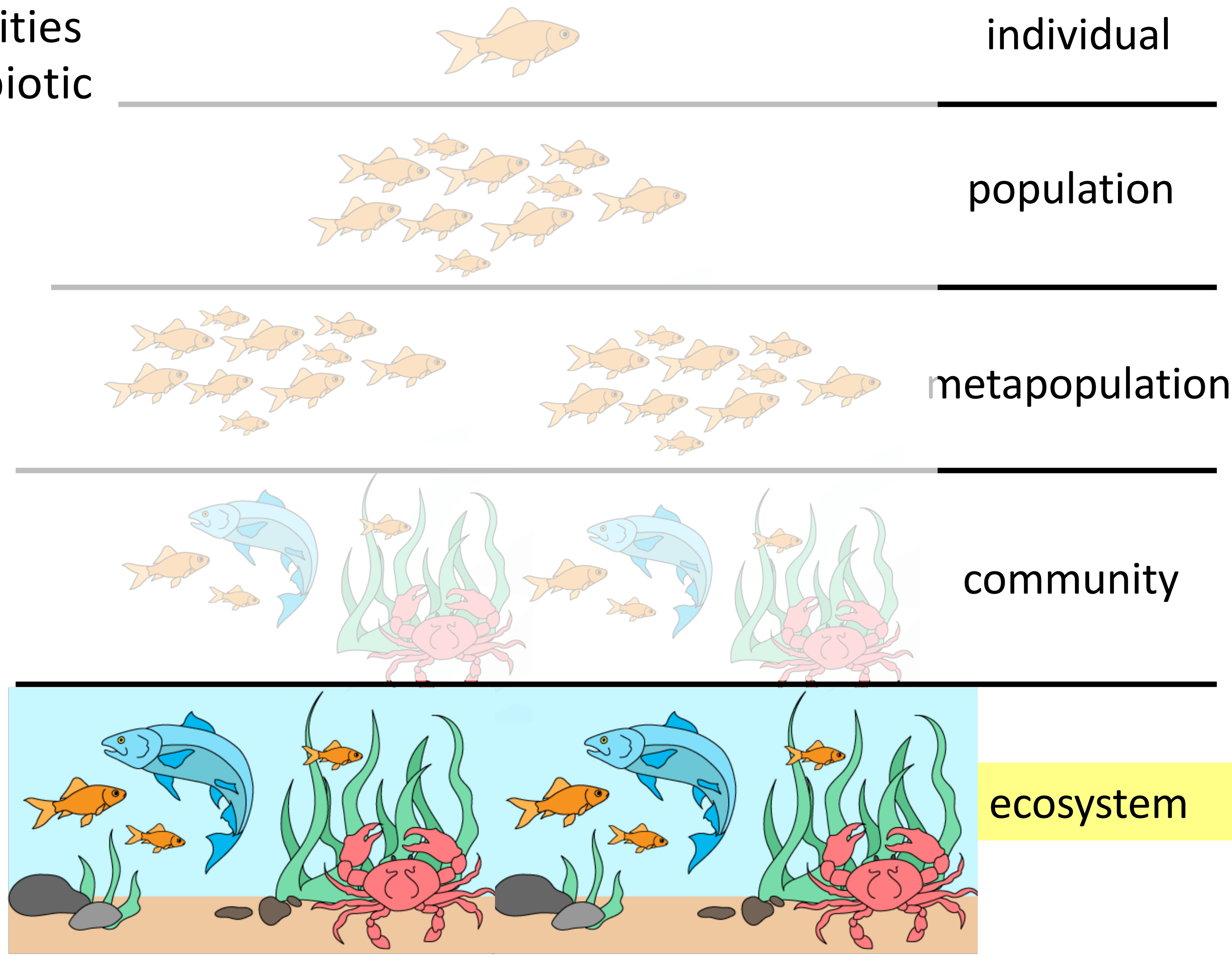
Community = interacting populations of different species



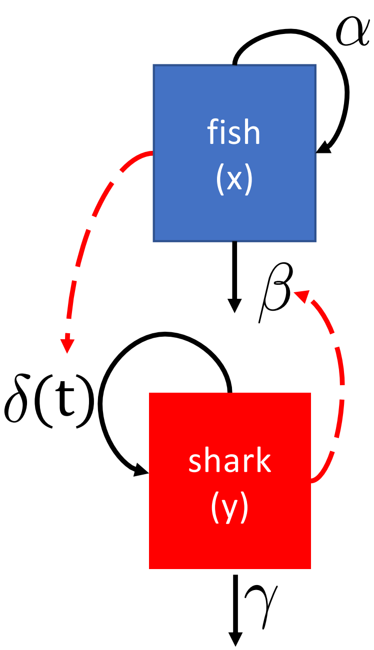


*How does the abundance of **fish** change based on **infection with** ***Mycobacterium marinum*** (wasting disease)?*

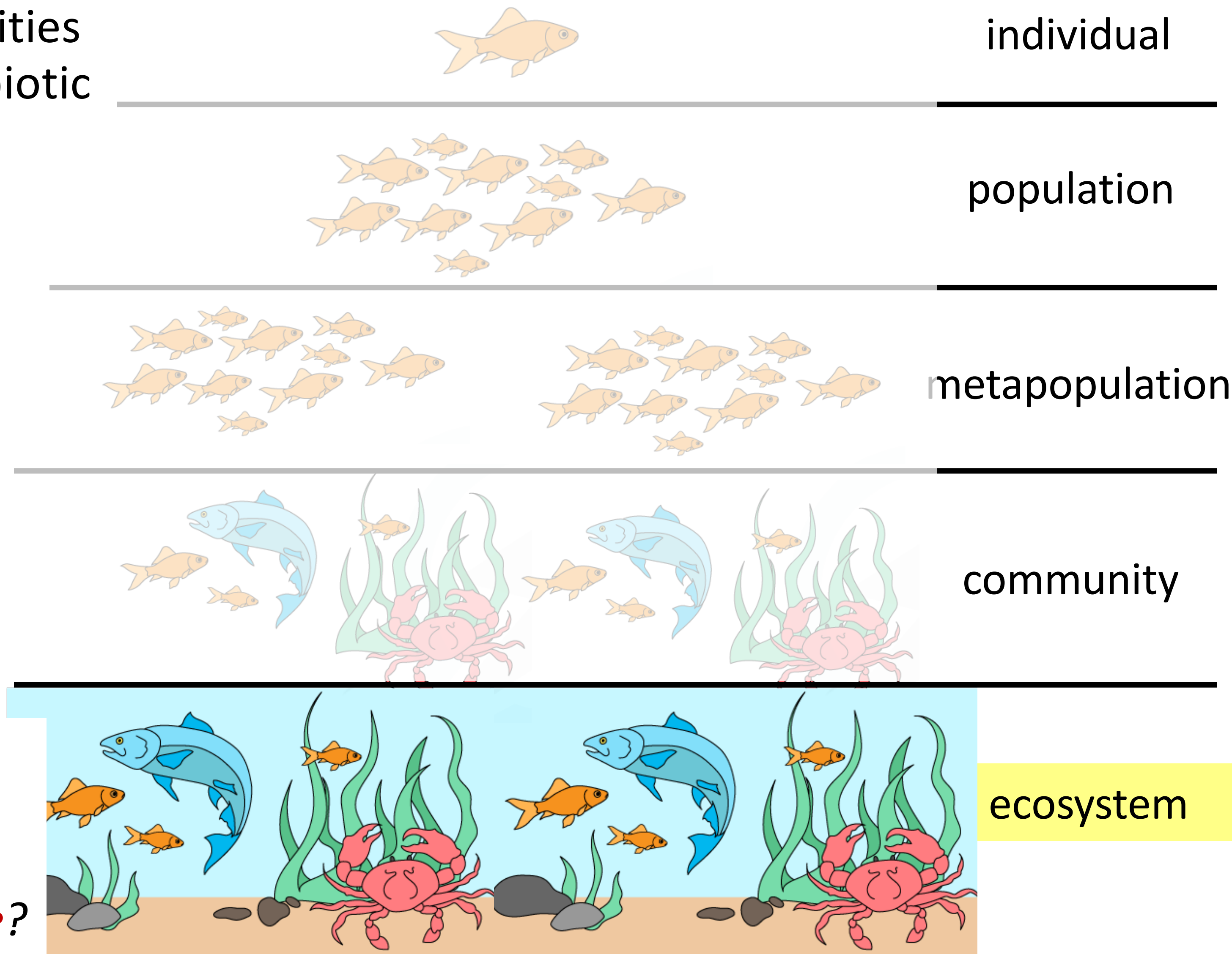
Ecosystem = communities interacting with the abiotic environment



Ecosystem = communities interacting with the abiotic environment

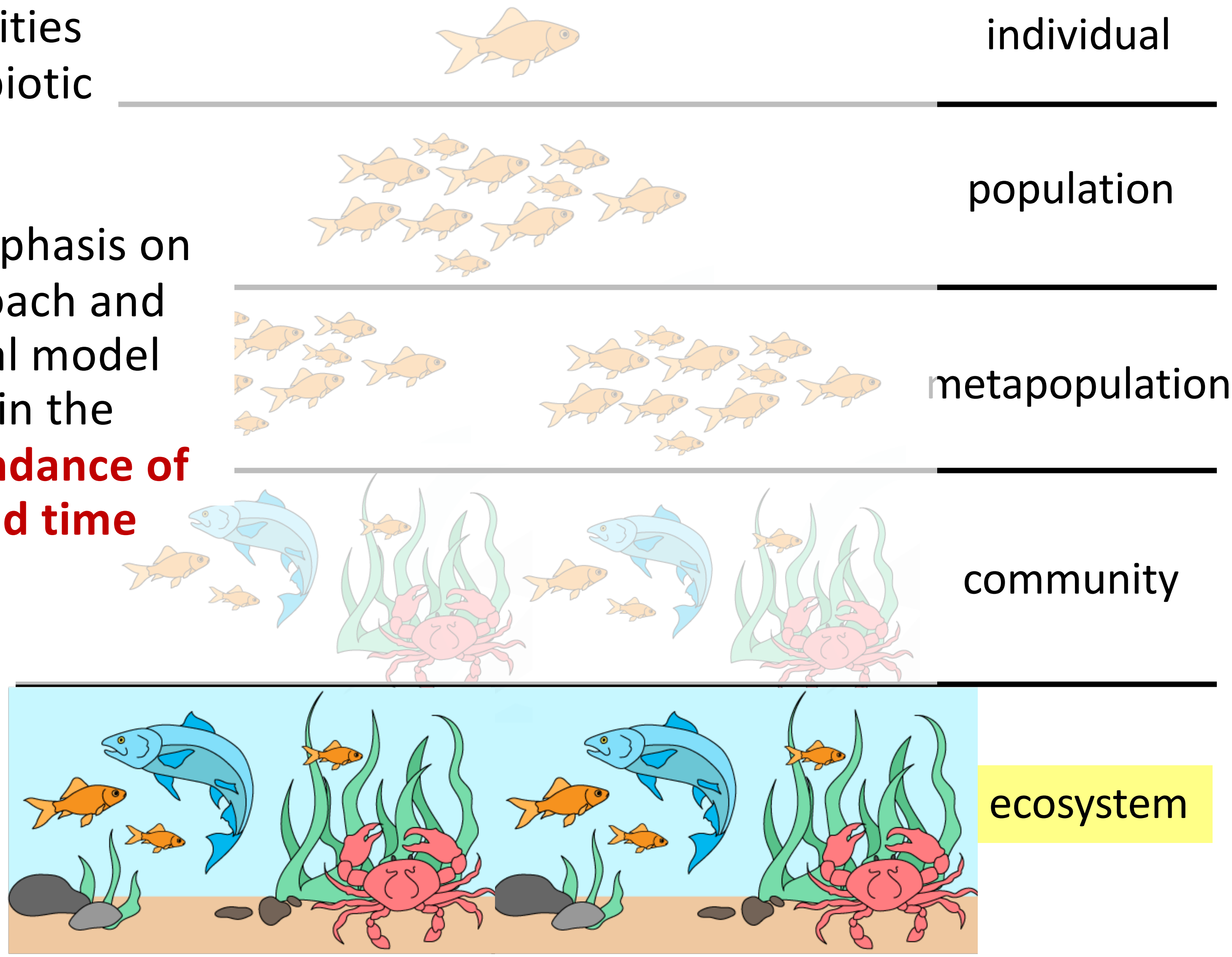


*How does fish abundance **vary** with **changes** in shark birth rates with **temperature**?*



Ecosystem = communities interacting with the abiotic environment

There is often less emphasis on the box model approach and more of a conceptual model approach to explain the **distribution and abundance of species in space and time**



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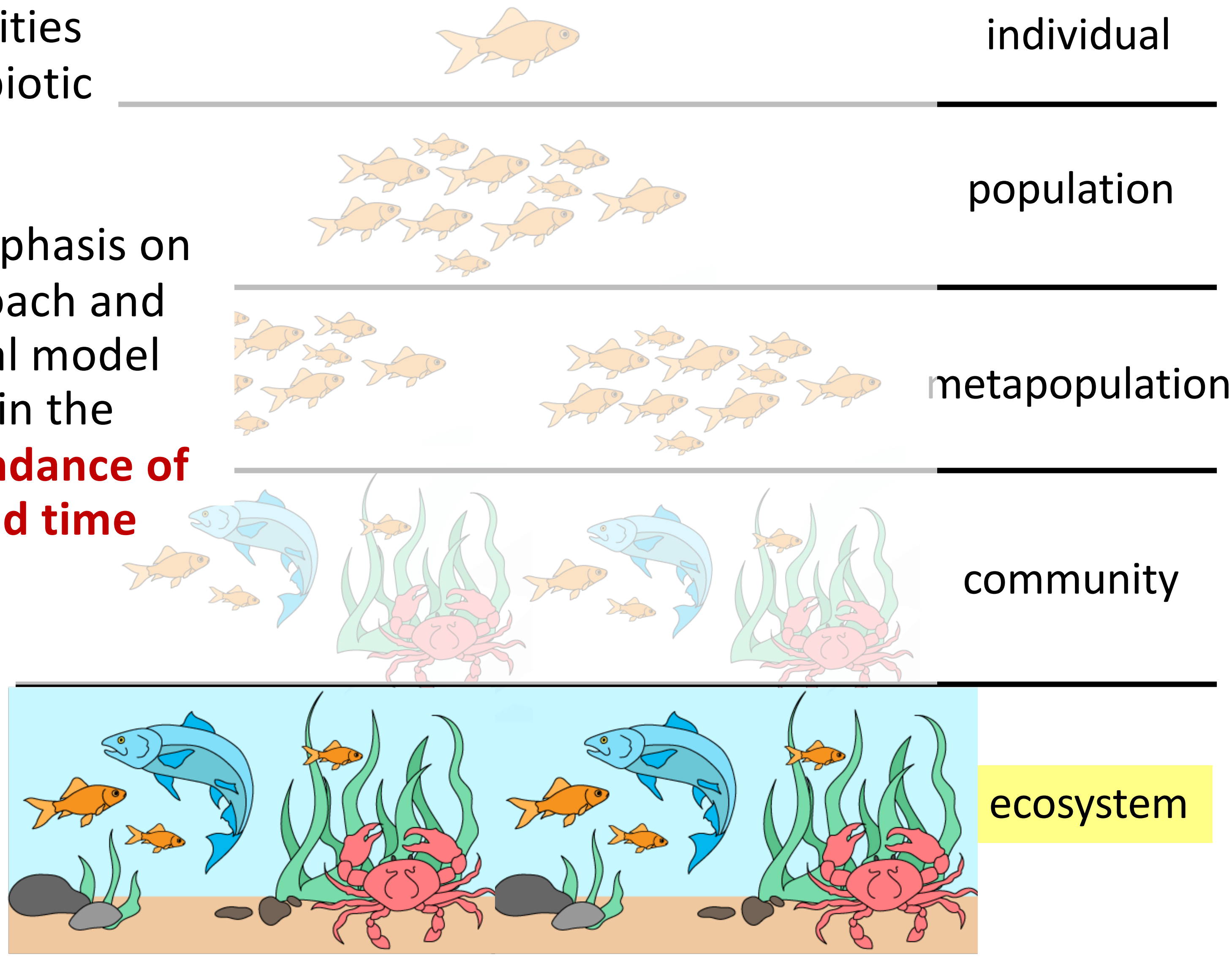
How do communities assemble?

Do they end up the same in different environments?

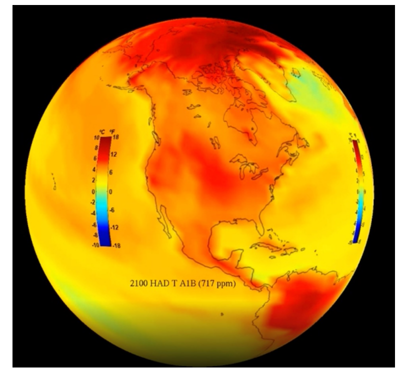


What is a model? an abstract representation of a phenomenon

Human

Solar System

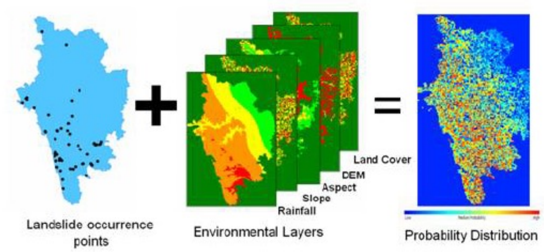
Climate



Human Genetics

Human Disease

Species Distribution



What is a model? an abstract representation of a phenomenon

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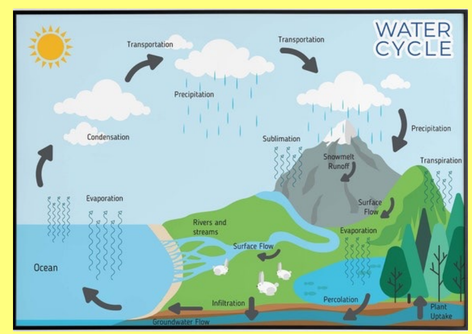
Mathematical

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

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

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

Conceptual



Community assembly is the study of the **processes** that shape the **identity and abundance** of species within ecological communities.

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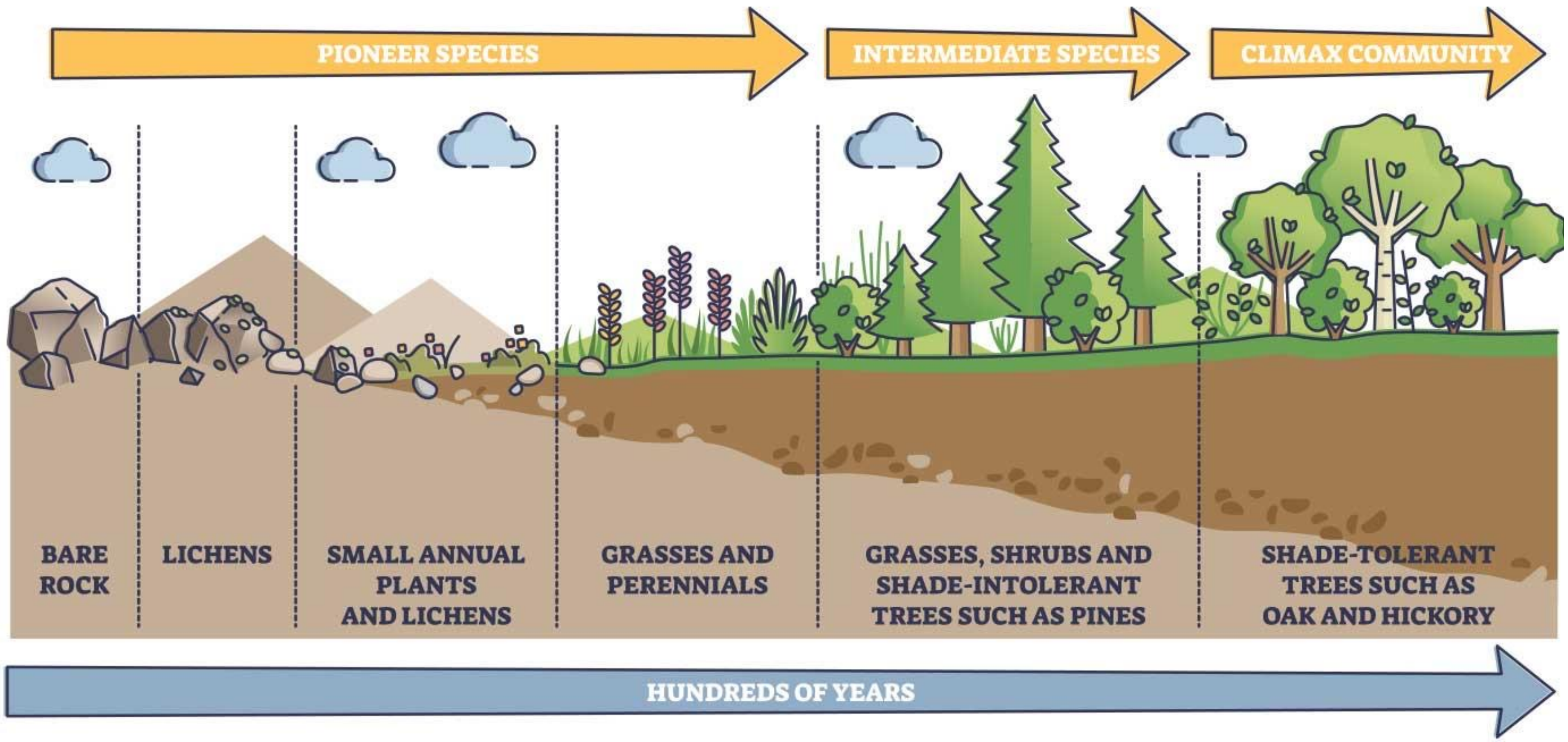
- Succession is the **process of change** in the **species structure** of ecological communities with time.
- Community begins with **pioneer species**, then develops with increasing complexity that self-reinforces to establish a **climax community**.
- Henry Chandler Cowles, a professor at the University of Chicago, developed the first formal concept of succession while observing **vegetation on dunes of different ages at the Indiana Dunes**. Differently aged dunes offered a proxy for time.

Primary succession occurs when species colonize a bare substrate.



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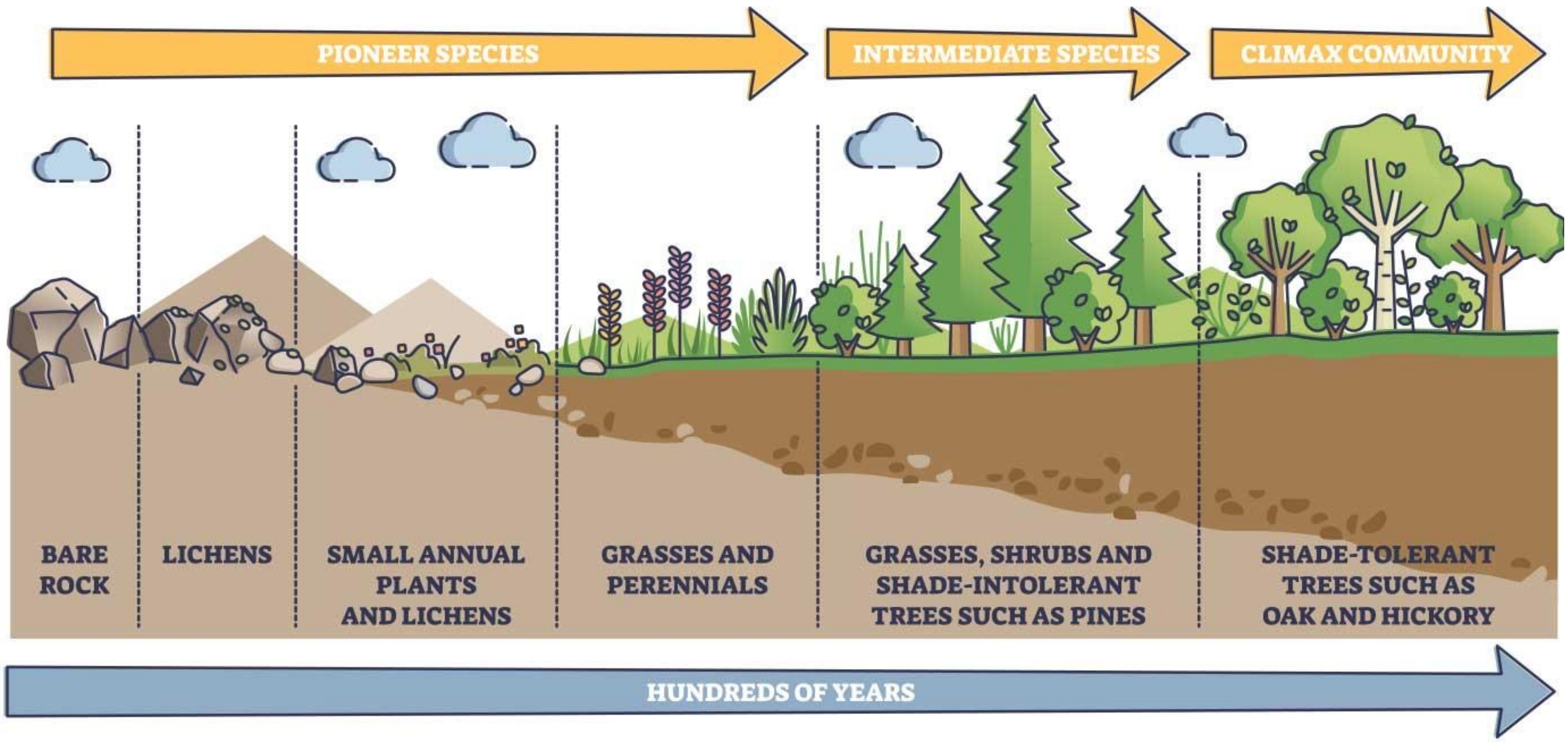
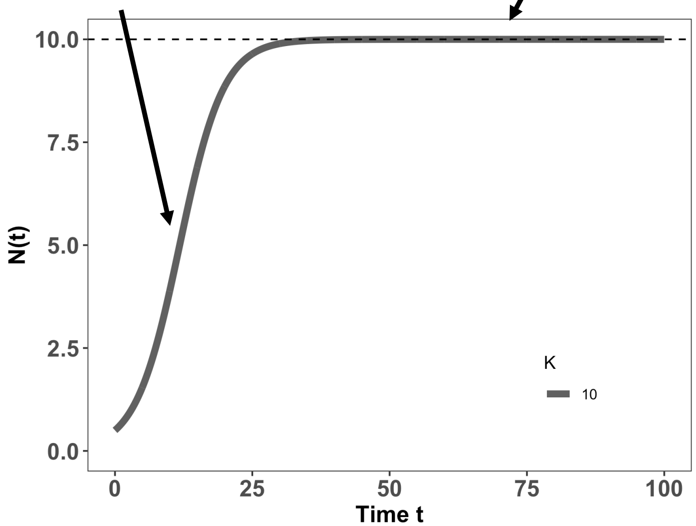


*Continuum from "**r-selected**" → "**K-selected**" species.*

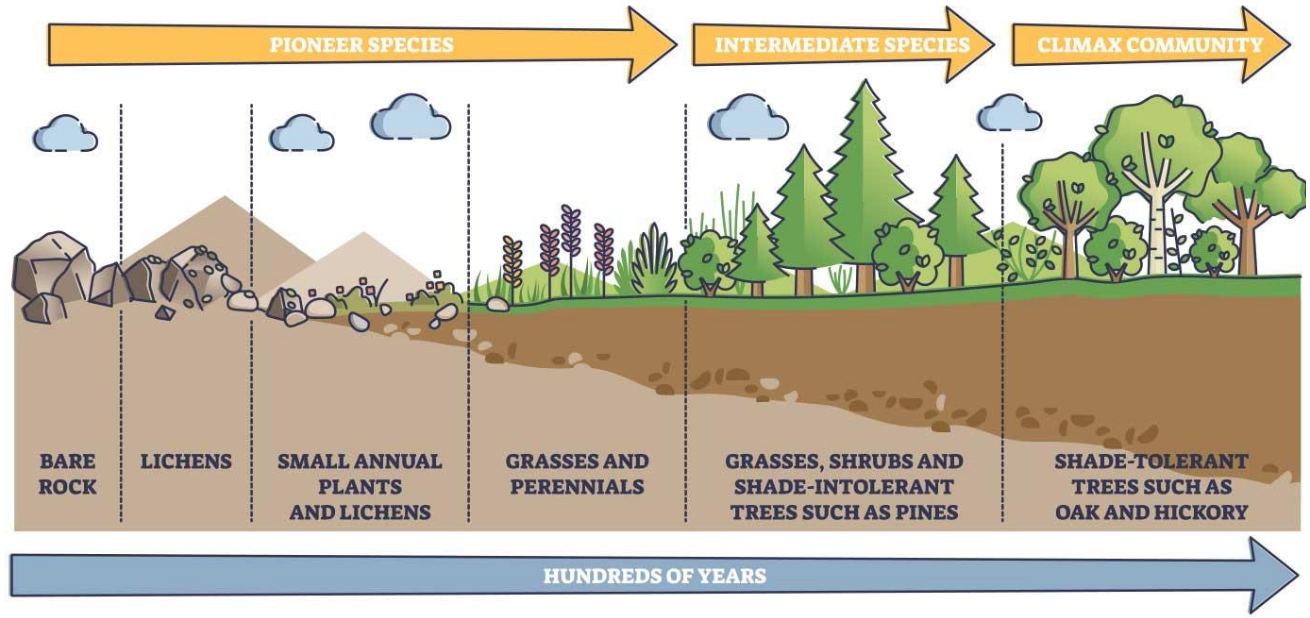
Primary succession occurs when species colonize a bare substrate.

K-selected species

r-selected species

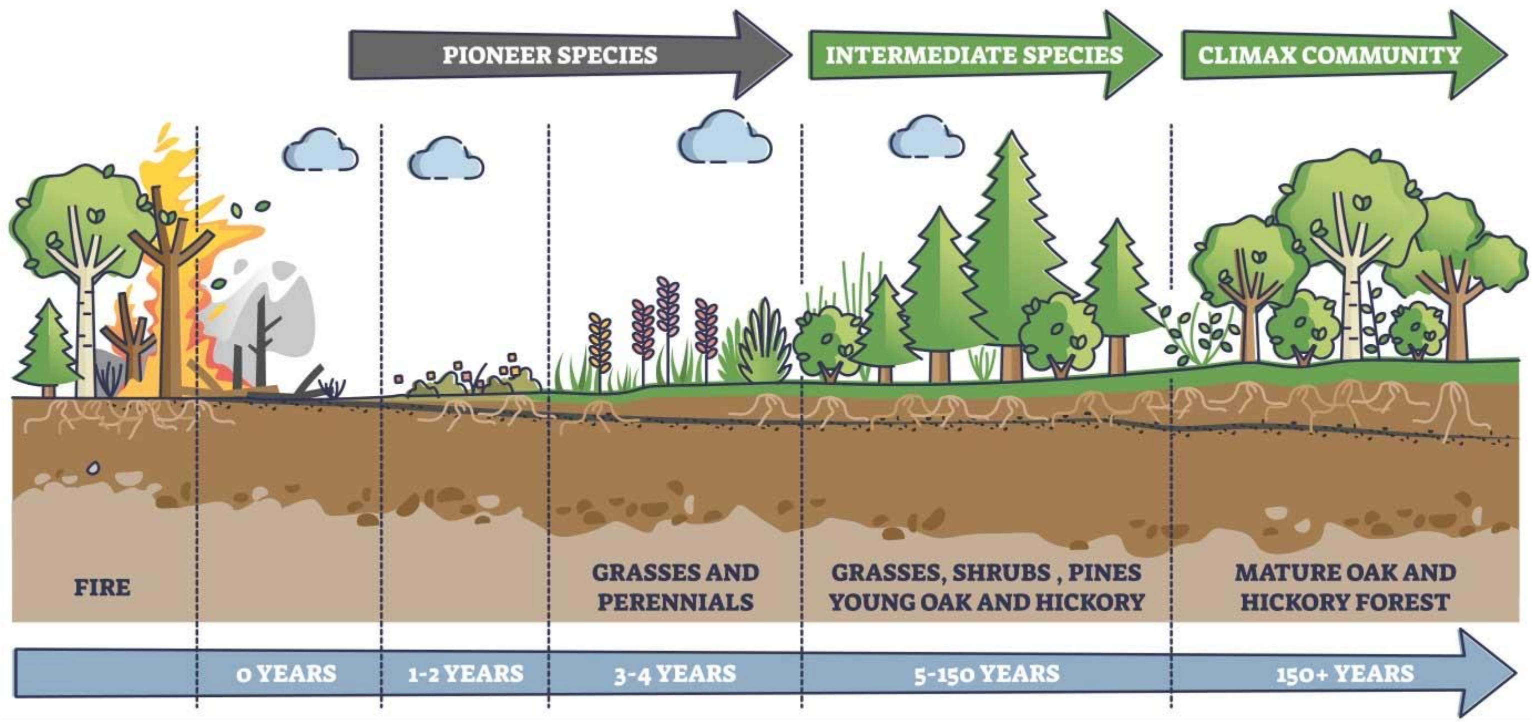


$$\frac{dN}{dt} = \boldsymbol{r}N\left(1 - \frac{N}{\boldsymbol{K}}\right)$$



Continuum from "r-selected" → "K-selected" species.

Secondary succession occurs when an environmental disturbance displaces a climax community, but soil and nutrients are still retained.



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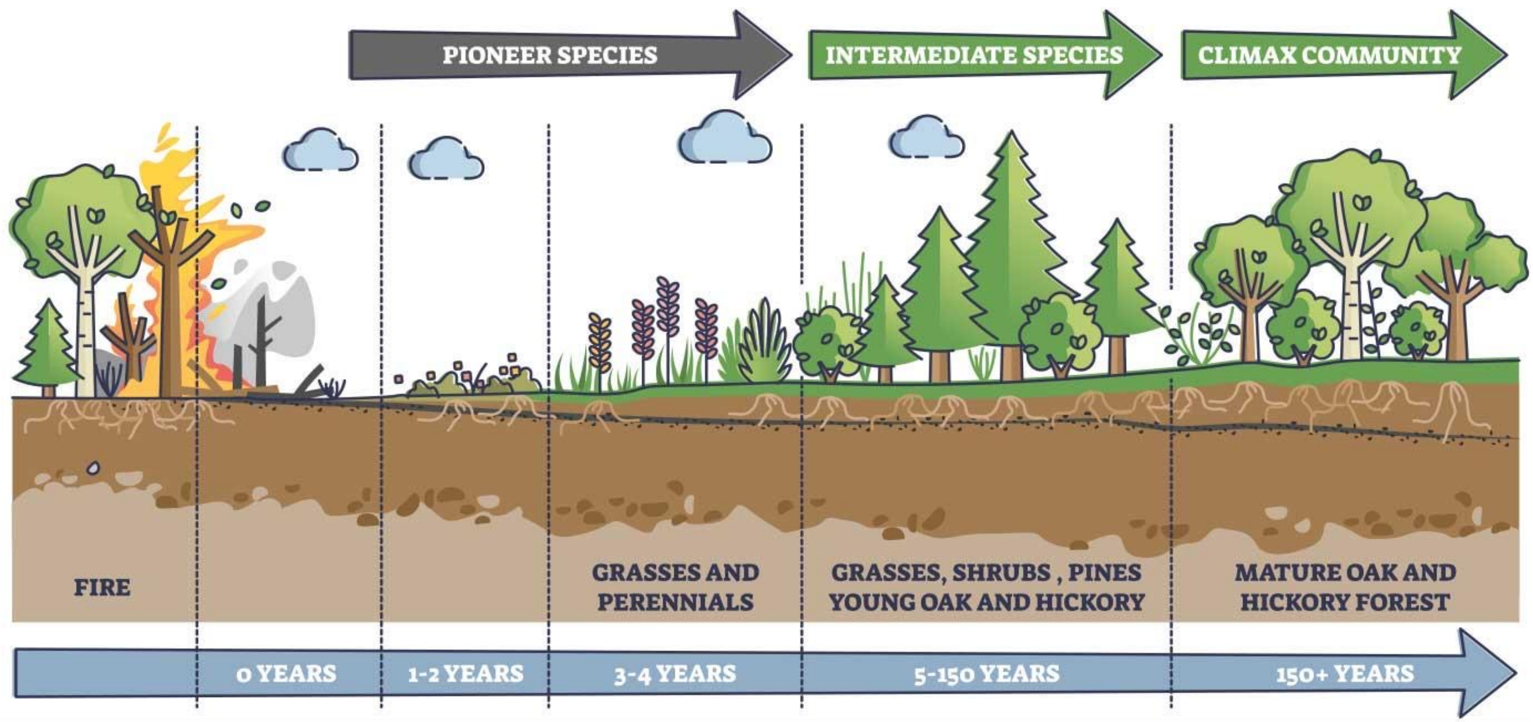
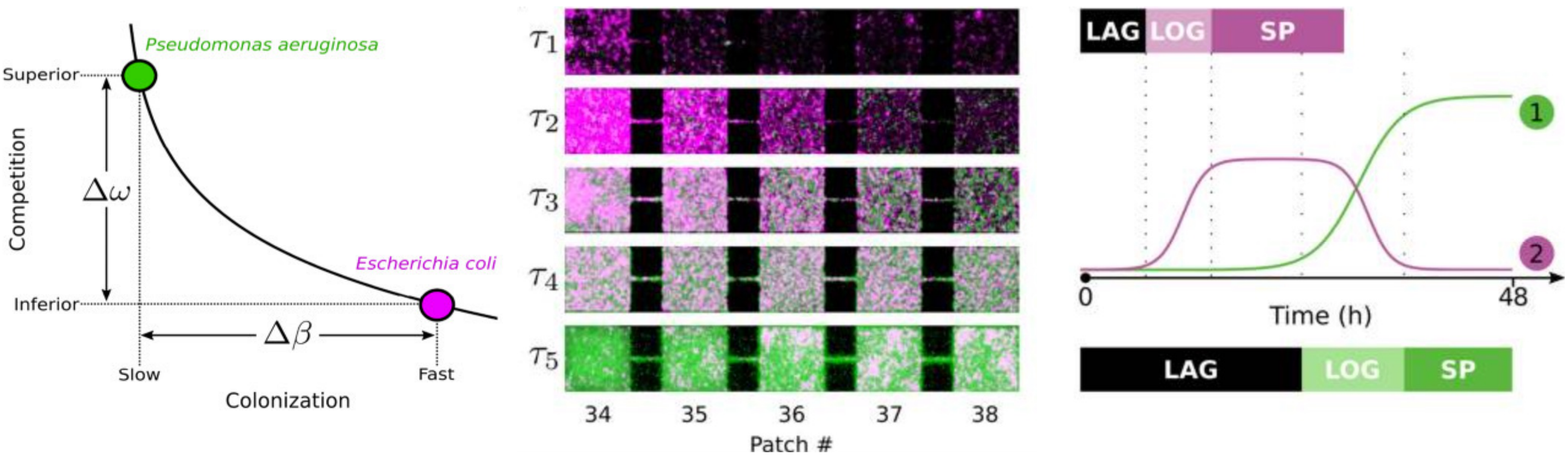


Continuum from "r-selected" → "K-selected" species.

Succession also occurs in microbial systems.

Here, the "***K-selected***" superior competitor eventually replaces the "***r-selected***" fast colonizer.



Wetherington et al. 2022. *BMC Biology*

Superorganisms vs. **Loose Collections** of Species

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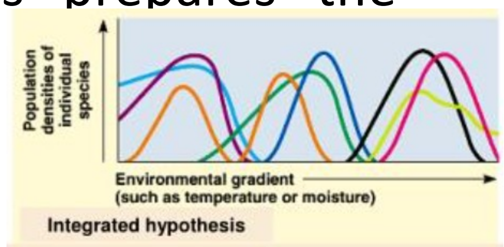

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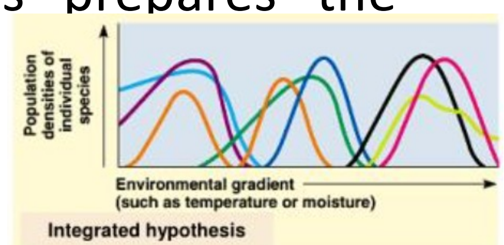
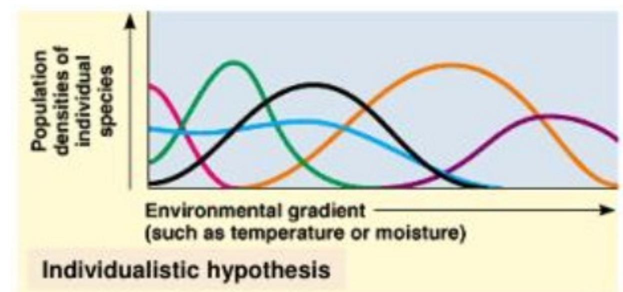
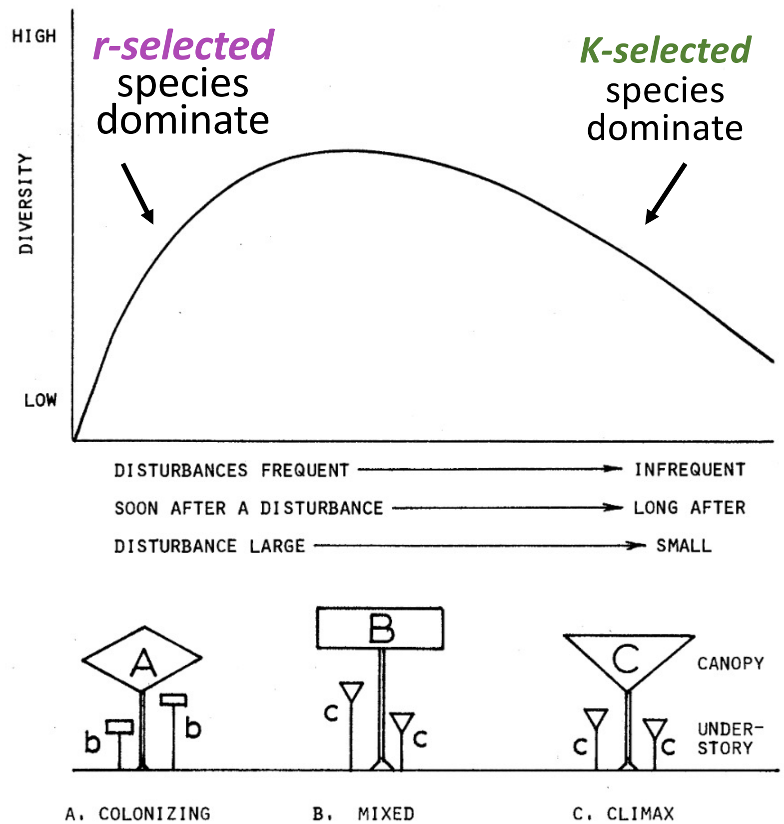


- Henry Gleason (1926) argued instead that chance favored the dispersal of nearby species into available habitat for succession, leading to **stochastic** assembly of communities
- Closer to Cowles' original thinking



The **Intermediate Disturbance Hypothesis** states that species diversity should be maximized at levels of intermediate disturbance in which both r-selected and K-selected species can coexist.



Connell 1978. *Science.*

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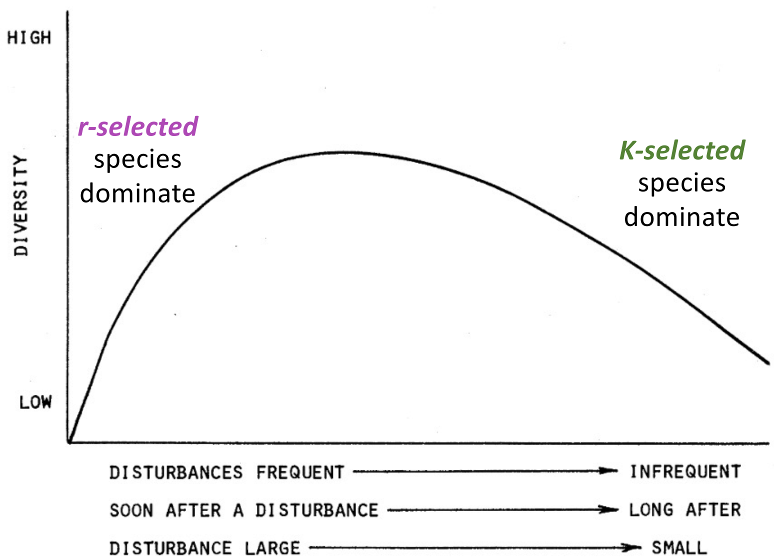

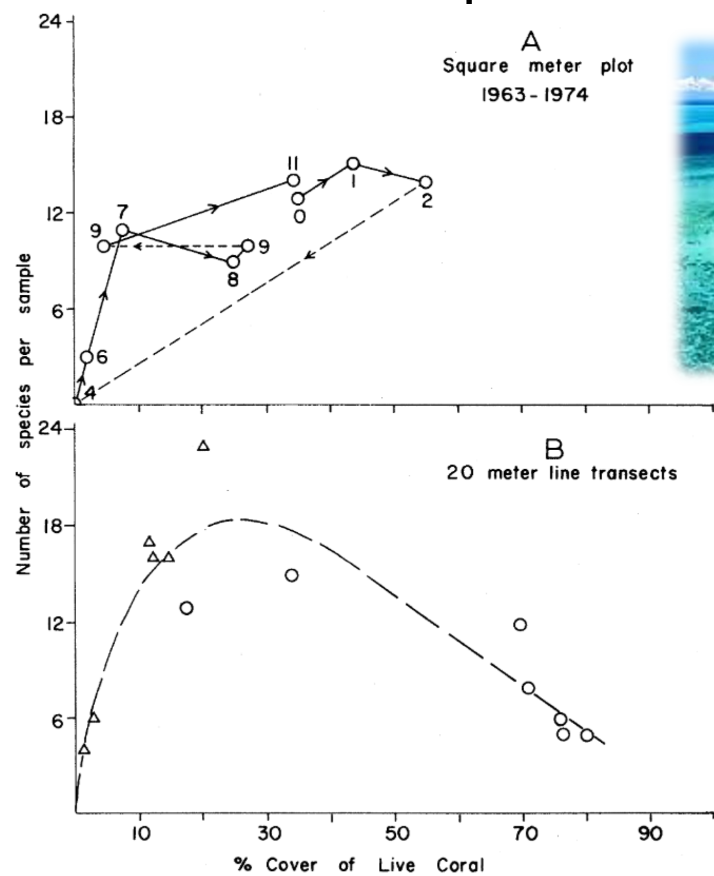

Heron Island, Australia

Sometimes it seems to be correct!

Connell 1978. *Science.*

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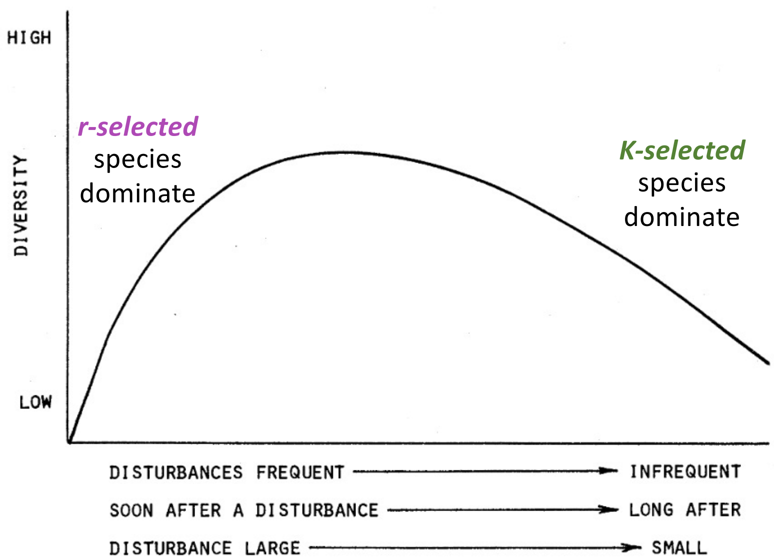
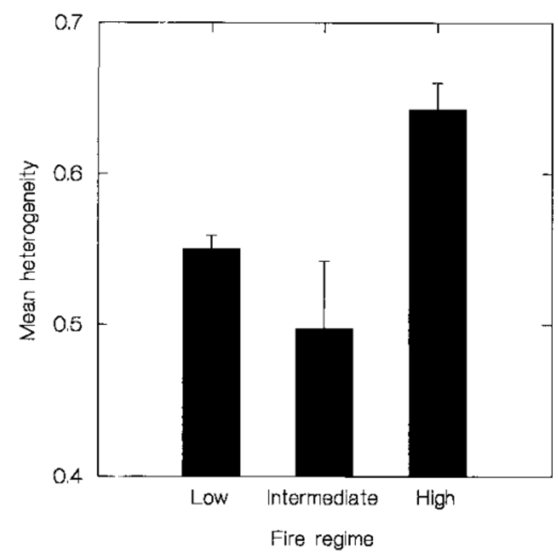


South African Fynbos

Sometimes it is not so well-supported empirically!



Schwilk et al. 1997. *Plant Ecology*

The **IDH** gives an example of a model that is often wrong but still useful in generating testable hypotheses.

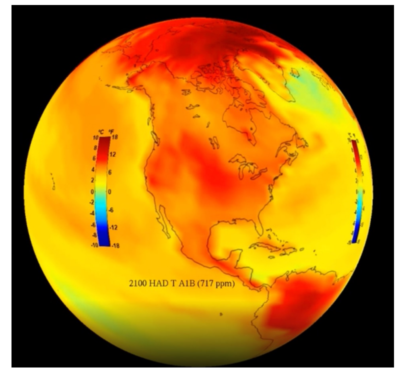
What is a model?

an abstract representation of a phenomenon

Human

Solar System

Climate



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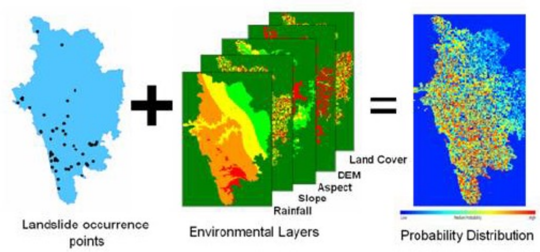
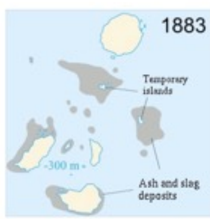


Wk9-Lect8-Mar4

Visual settings

Edit

Volvanic eruptions on Krakatoa, Indonesia in 1883 led to the complete destruction of a large part of the land mass and buried the remnant regions in ash and slag. What type of succession best describes the return of vegetation to these islands?

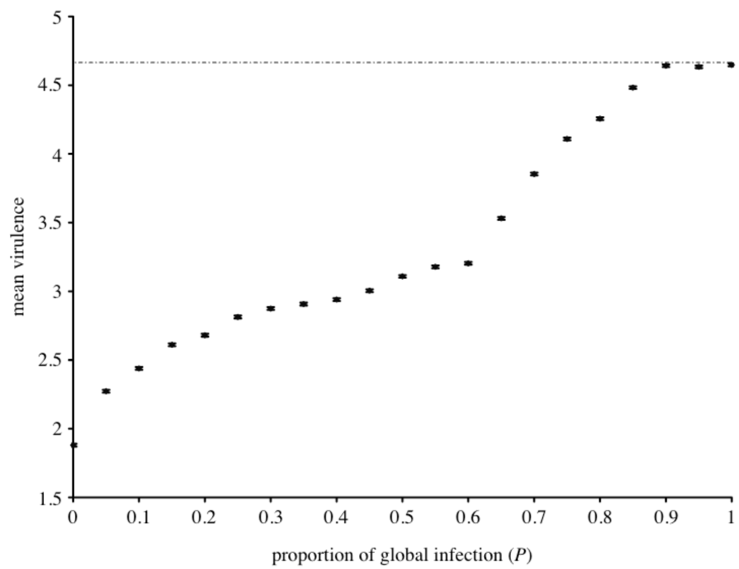


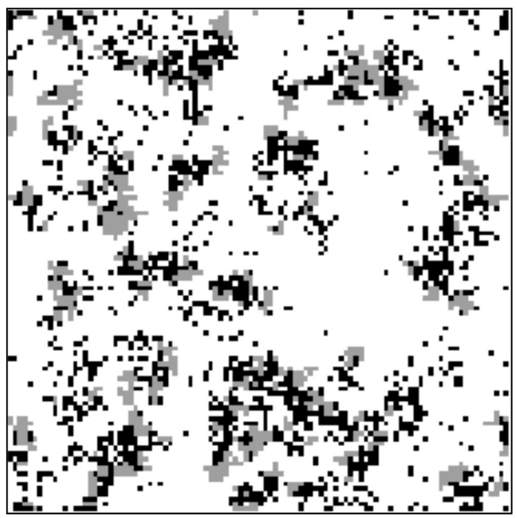
A primary		0%
B secondary		0%
C tertiary		0%
D quarternary		0%

Powered by Poll Everywhere

Extra

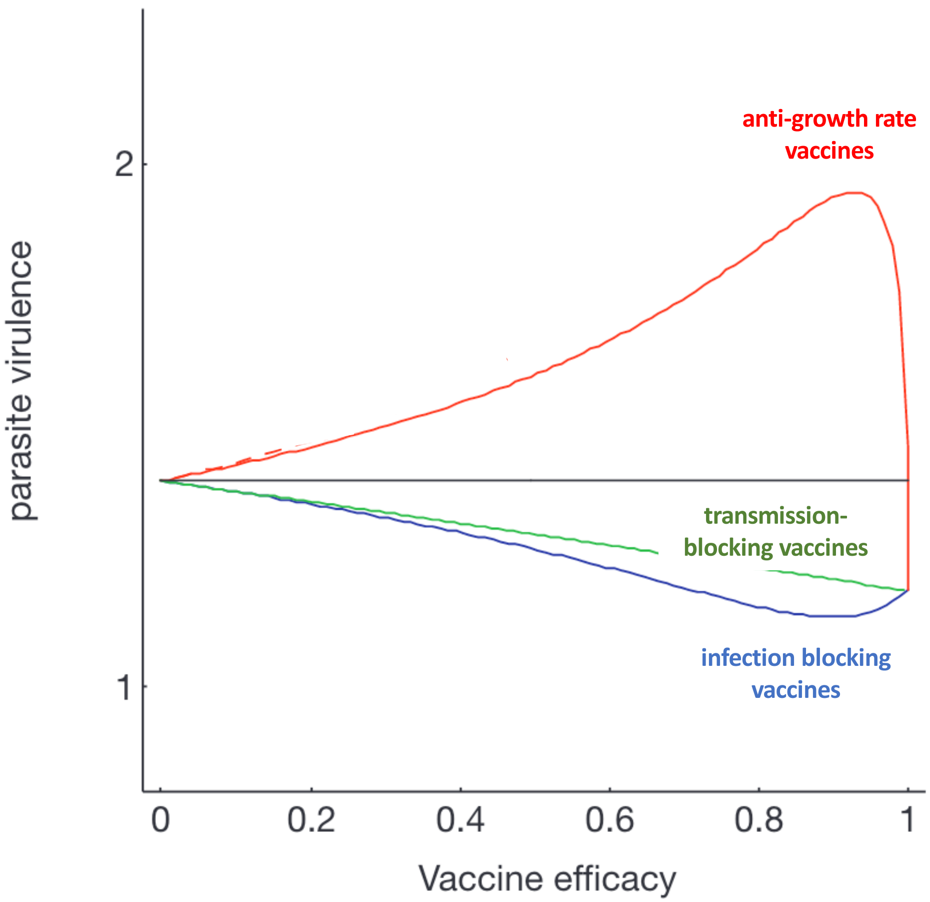
Spatial structuring generally favors **reduced pathogen virulence**.





Boots and Sasaki. 1999. *Proc Roy Soc B.*

Imperfect vaccination can support the evolution of higher virulence, depending on the lifestage of the pathogen that is targeted.



Gandon et al. 2001. *Nature.*

Malaria vaccines