

Fundamentals of Ecology

Week 9, Ecology Lecture 7

Cara Brook

February 27, 2024

Let's recap a bit!

Disease Ecology

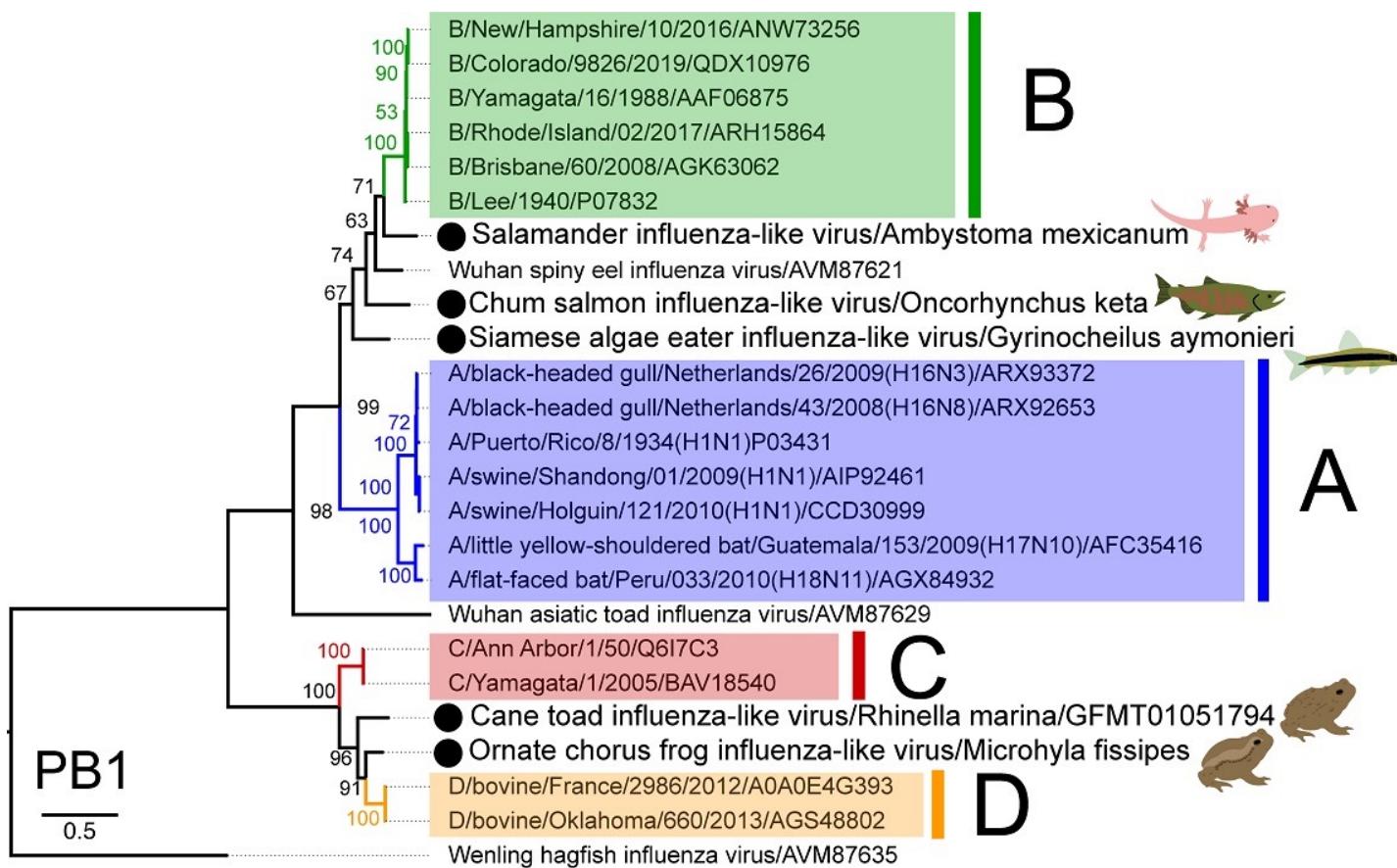
- We learned about extensions to the SIR model that can be made to better represent diverse types of pathogens (e.g. dynamics different from classic measles).
- These extensions included incorporating inherited maternal immunity into a disease model (important in the case of natural vs. vaccinated immunity in mothers), as well as modeling different types of pathogen transmission.
- Environmental disease transmission occurs when the pathogen is maintained outside the host in an environmental reservoir. We learned a few approaches to modeling this type of pathogen. Example: cholera, with water-borne transmission.
- Vertical transmission occurs when a pathogen is passed mother-to-infant *in utero*.
- Vector-borne transmission occurs when a pathogen is transmitted via blood-feeding arthropod (tick, flea, mosquito). We learned the specifics of how vector-borne disease are modeled in the case of malaria.

Let's recap a bit!

Disease Ecology

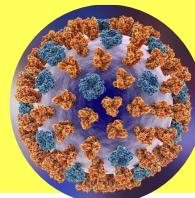
- We explored advances in malaria modeling, starting with the simplest Ross model, then adding a mosquito incubation period in the MacDonald model, then adding a human incubation period in the Anderson and May model.
- Incorporation of the mosquito incubation period in the MacDonald model led to the insight that the survival of the female mosquito is a weak point in the malaria transmission cycle, which inspired decades of WHO malaria control via spraying of the insecticide DDT.
- We learned terminology for zoonotic diseases, those transmitted from wildlife reservoirs to human spillover hosts.
- Spillback occurs when pathogens transmit from the spillover host back to the reservoir that sourced the infection, as in the case of mink transmission of SARS-CoV-2 to humans.
- We can classify pathogens into stage I-V based on their R_0 in the human population. Zoonotic pathogens encompass stages II-IV.

When is influenza zoonotic?

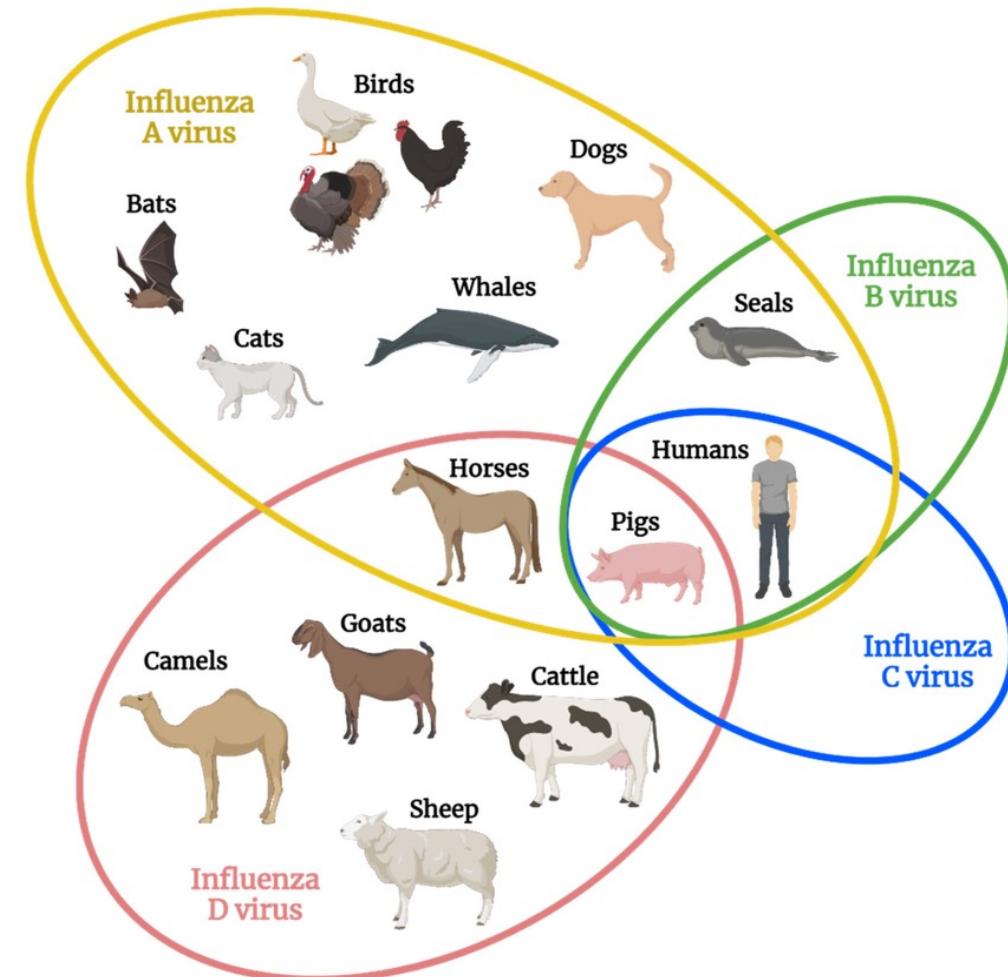


Parry et al. 2020. *Viruses*.

Stage V Transmits exclusively in humans

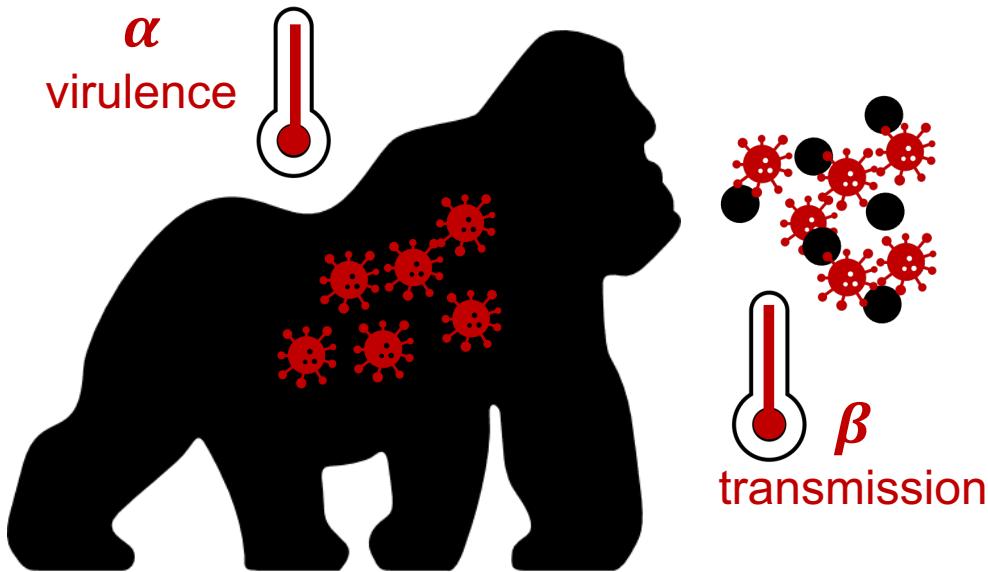


Influenza



Skelton & Huber. 2022. *Viruses*.

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

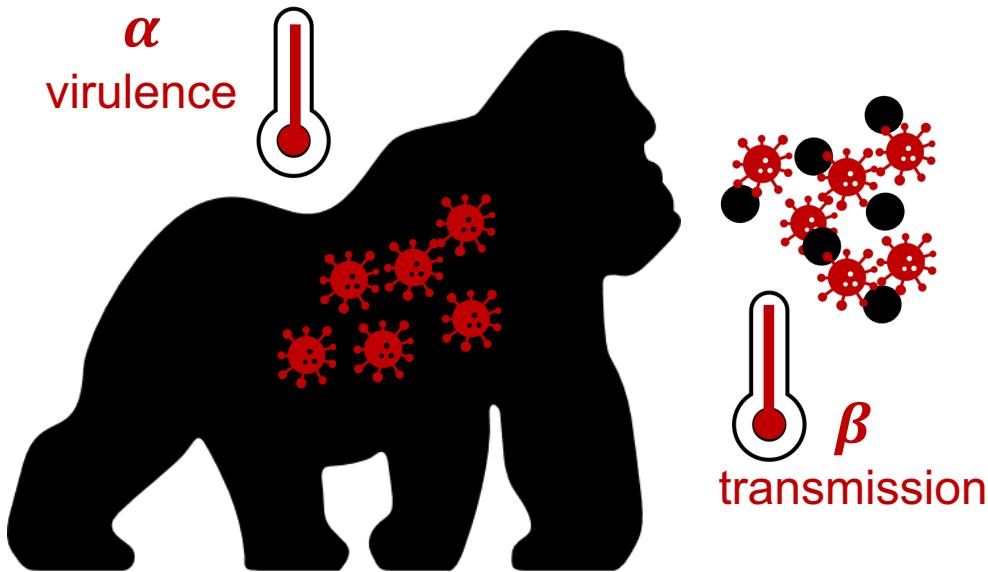


Mechanisms that promote **transmission** may also enhance **virulence** to the host.

Why do pathogens make us sick?

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

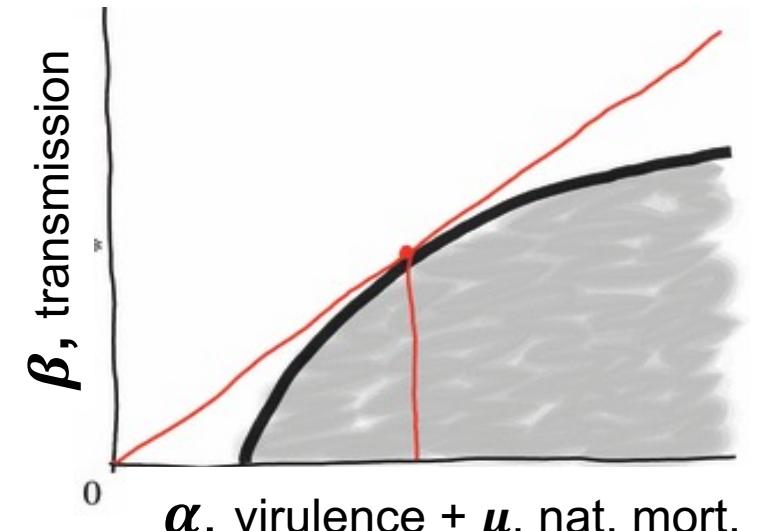
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Why do pathogens make us sick?

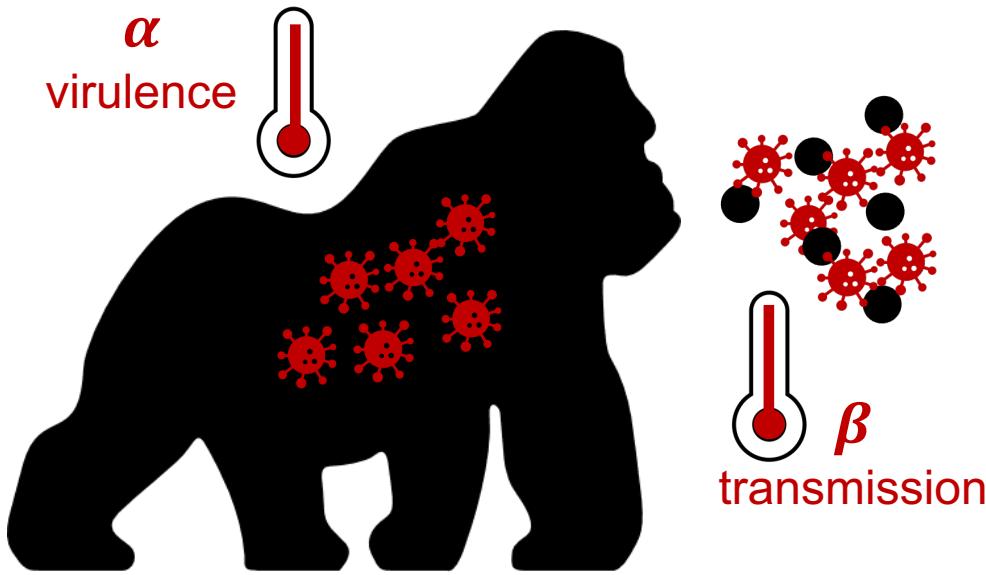
As a result, we predict the evolution of "**optimal virulence**."



$$R_0 = \frac{\beta(\text{virus density})}{\gamma + \mu + \alpha(\text{virus density})}$$

} infections created
} infections lost

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



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

Why do pathogens make us sick?

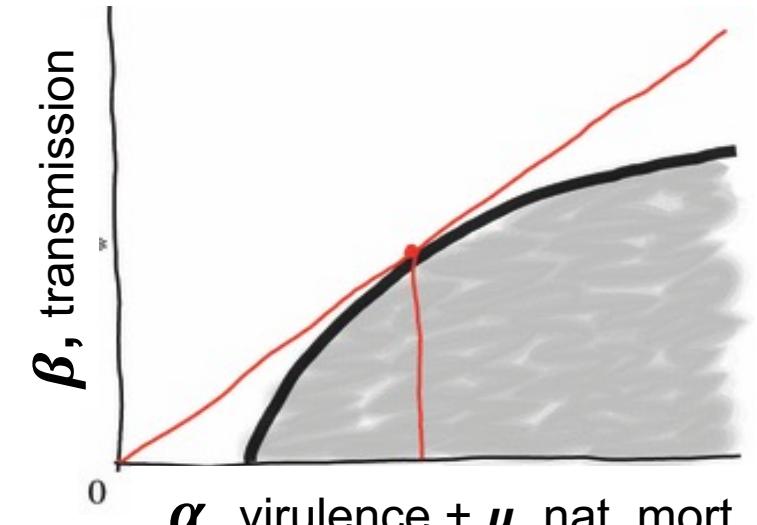
As a result, we predict the evolution of "**optimal virulence**."

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!

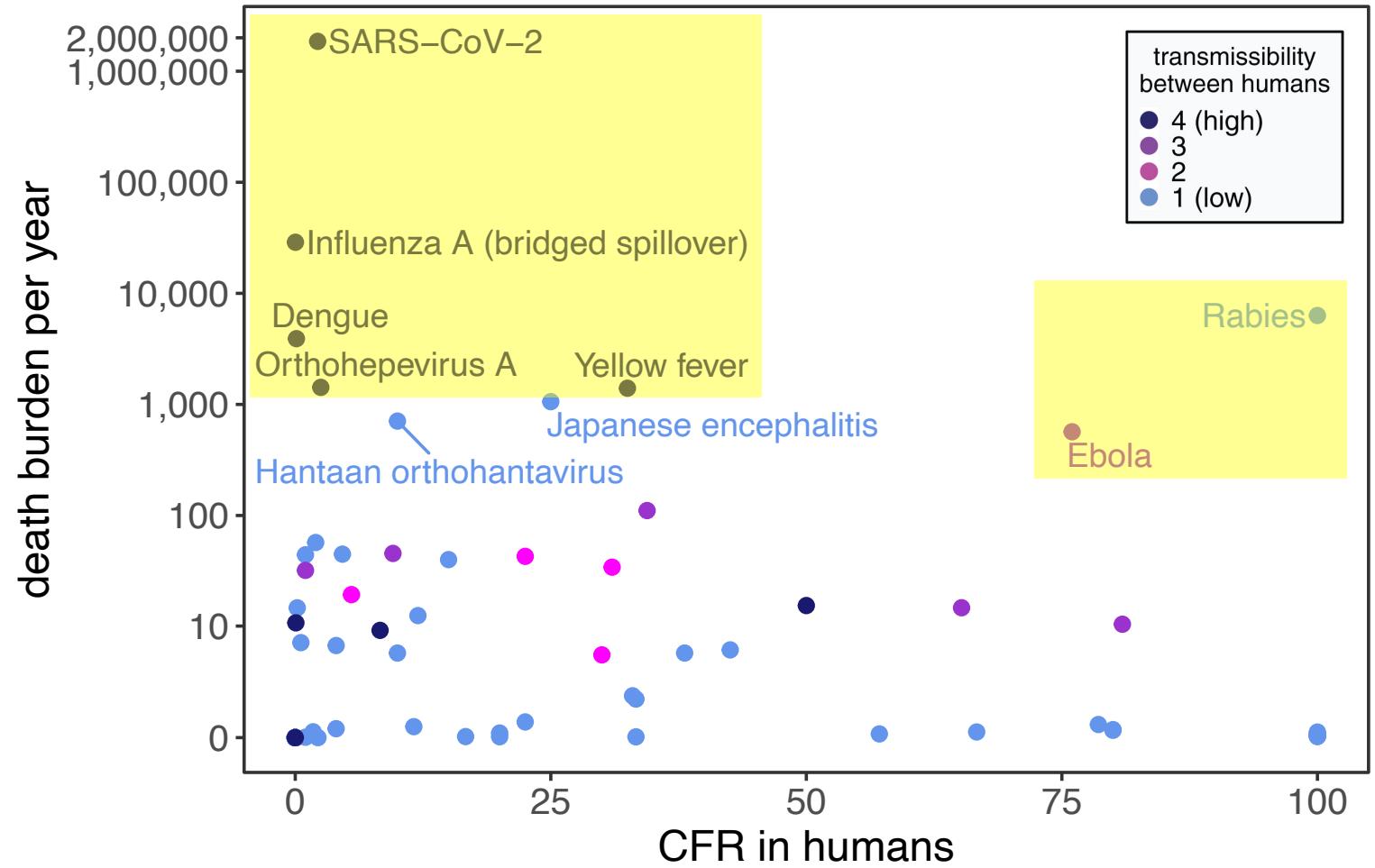
$$R_0 = \frac{\beta(\text{virus density})}{\gamma + \mu + \alpha(\text{virus density})}$$

Legend:

- β (yellow box): infections created
- α (yellow box): infections lost



For zoonoses,
**virulence and
transmission
tradeoff** to result in
total death burden.



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 >99	II 95–99	III 70–95	IV 50–70	V <50	number of samples
case fatality rate (%)						
mean survival time/day	< 13	14–16	17–28	29–50	—	
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).

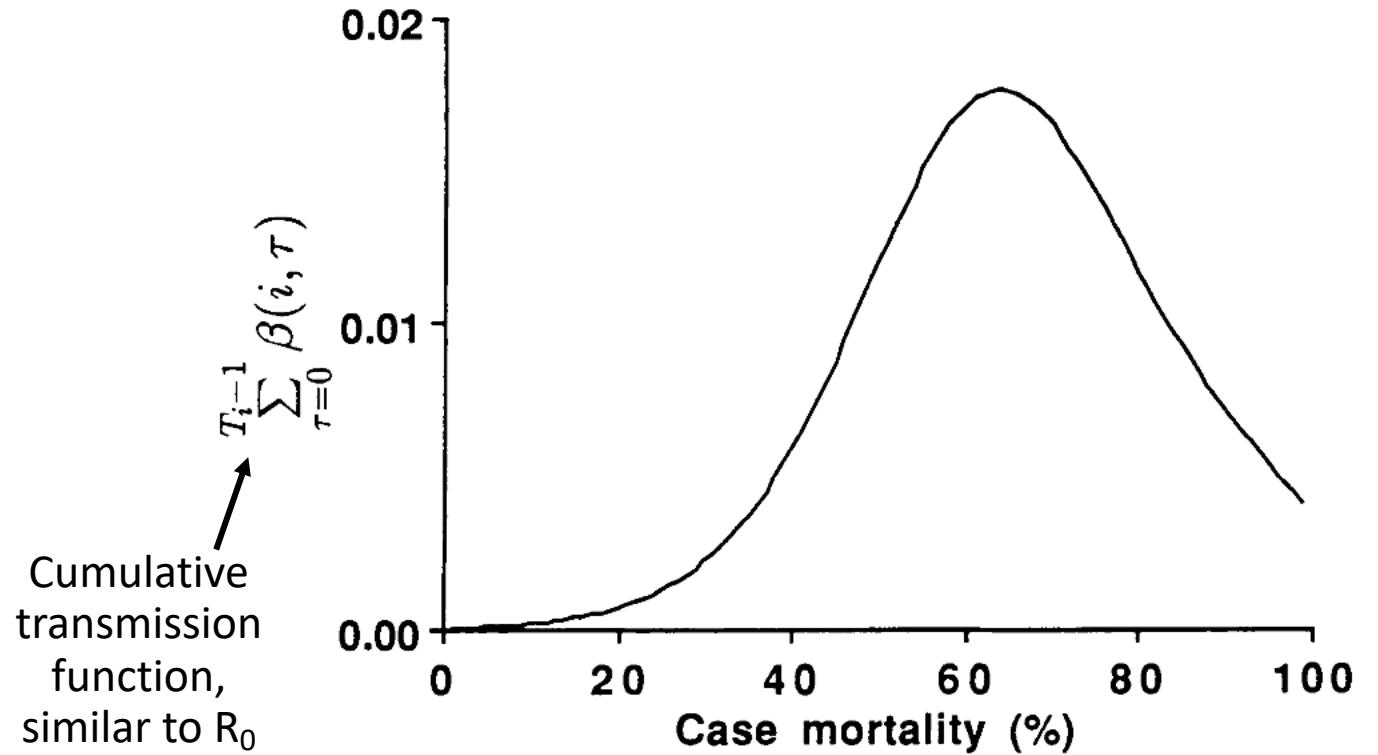
§ Data from J. W. Edmonds and R. C. H. Shepherd (personal communication, 1982).

|| Although only one strain was tested, the very high mortality rates in the initial outbreaks justify this extrapolation.

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



A SIMULATION MODEL OF THE POPULATION DYNAMICS
AND EVOLUTION OF MYXOMATOSIS¹

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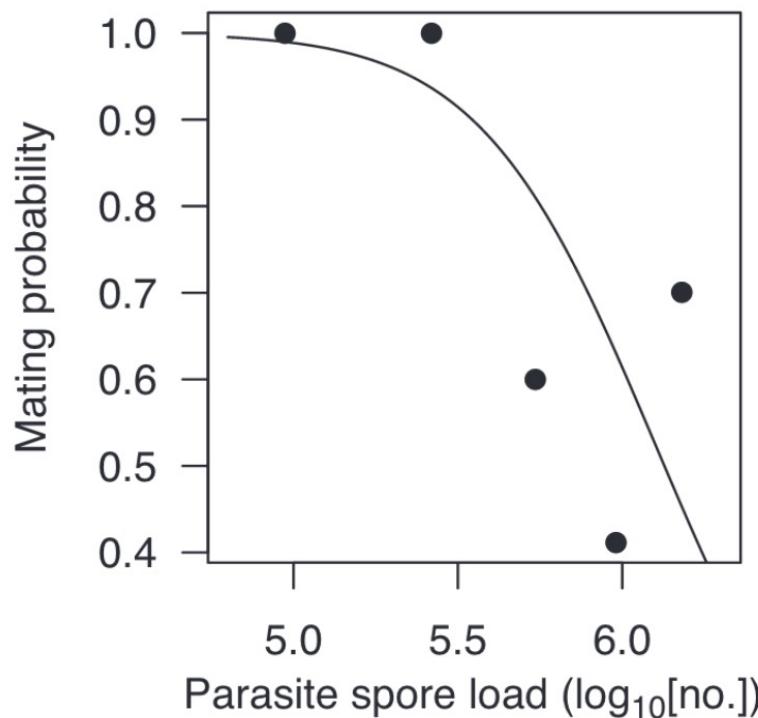
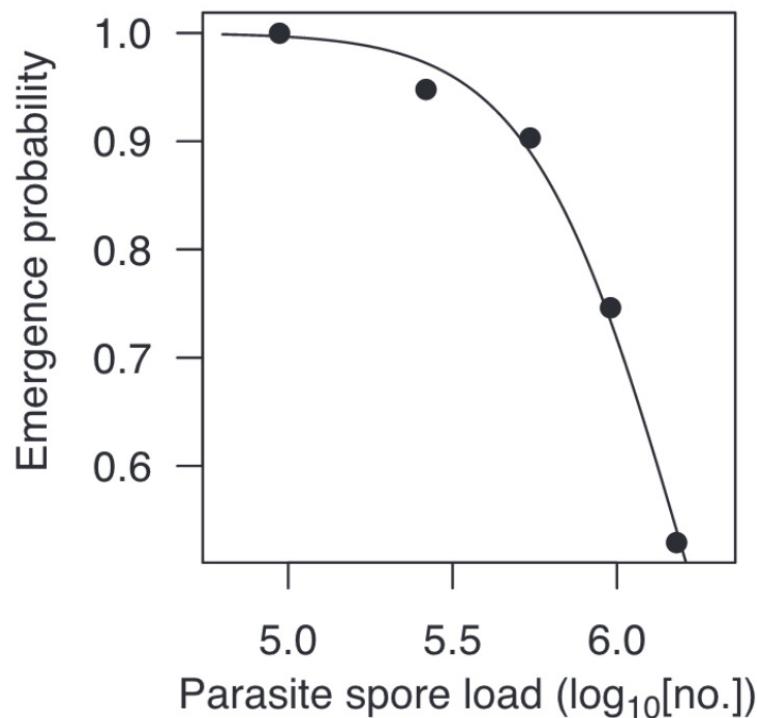
LINDA BUTTEL

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Ithaca, New York 14853 USA

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

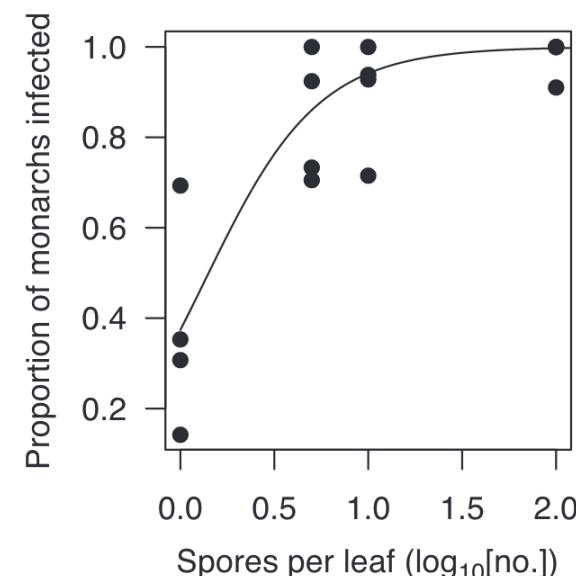
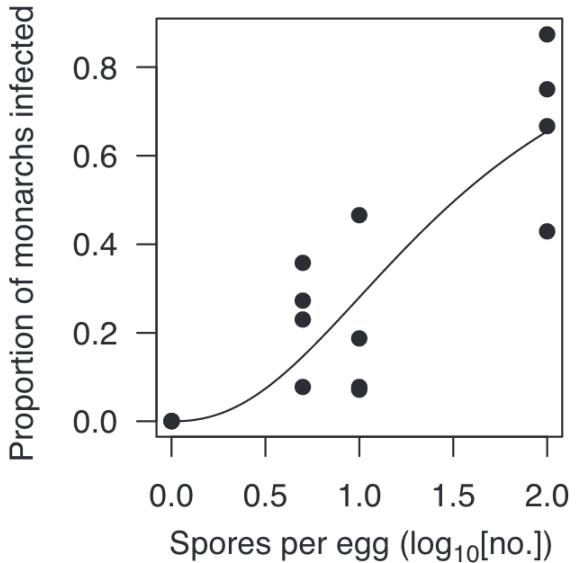
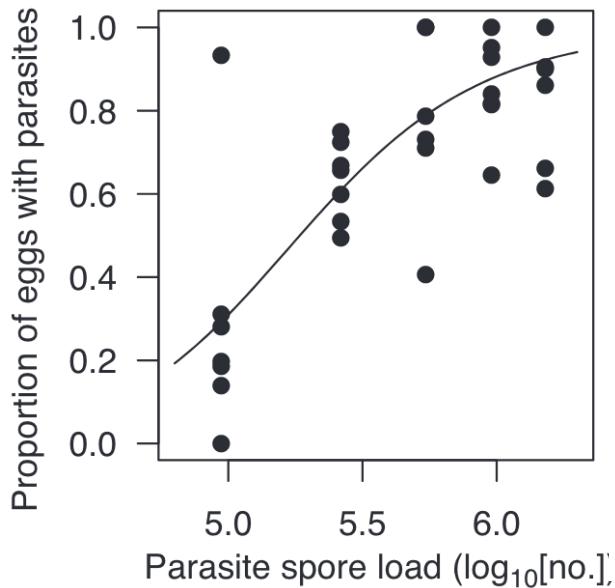
Another classic **transmission-virulence tradeoff**: parasites of monarch butterflies

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



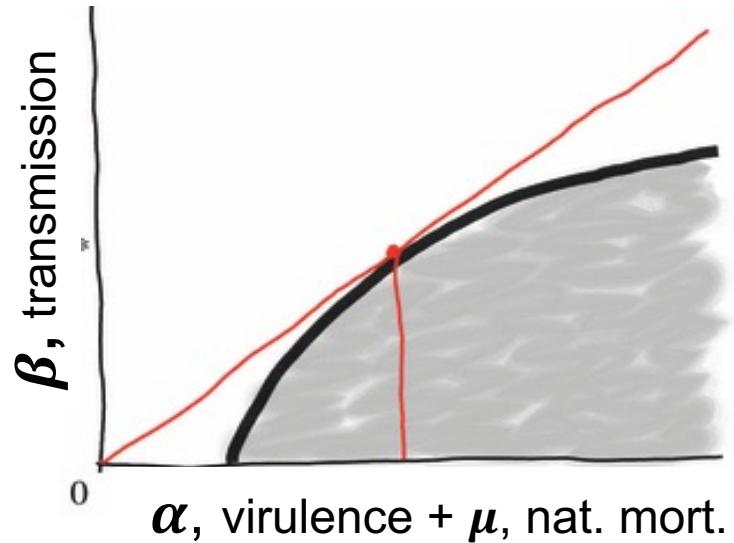
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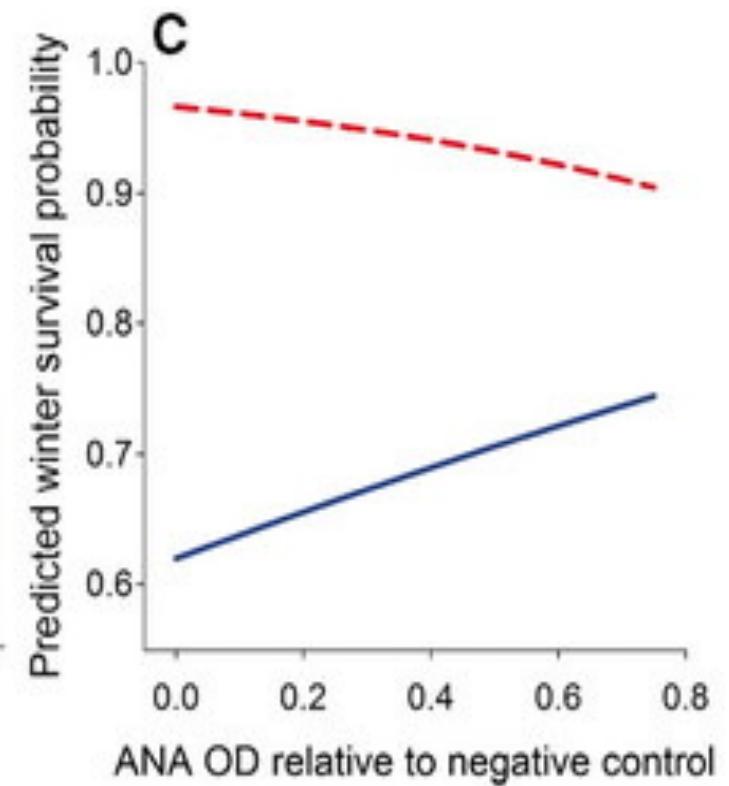
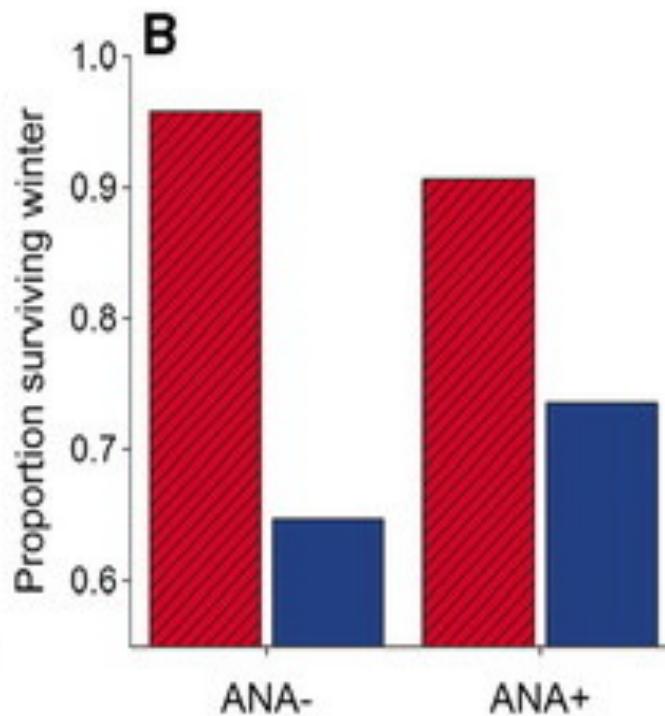
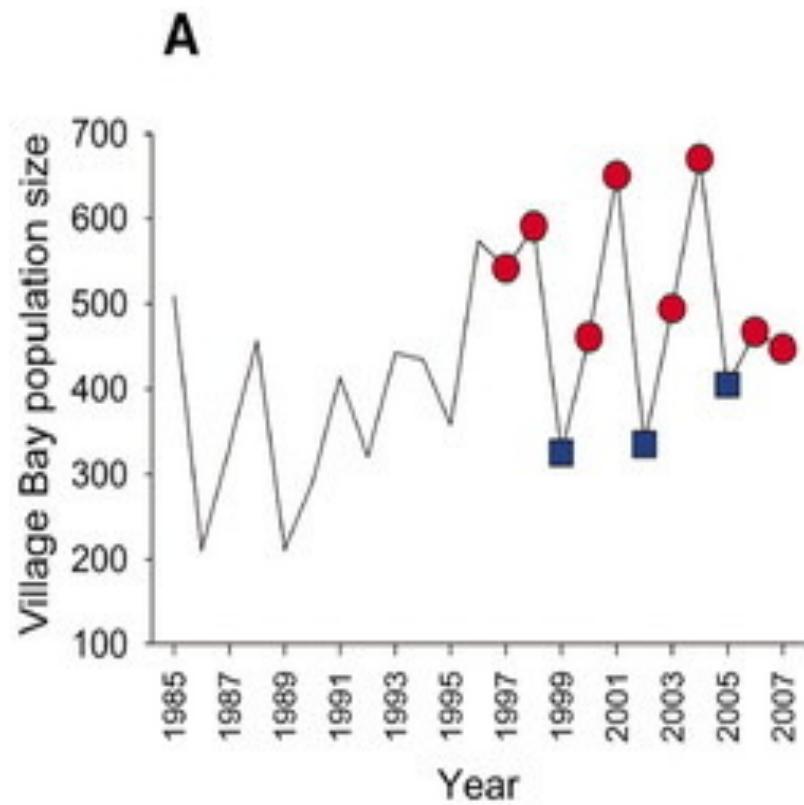
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- **Parasite fitness is calculated to be maximized at intermediate spore load.**



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!
- This is partly due to challenges arising from the difficulty of measuring (and defining) transmission and virulence.
 - Virulence is a fitness cost that the parasite inflicts on the host, but these can take diverse forms, with differing consequences for the evolution of virulence.
 - For example: Fitness effects on reproduction vs. adult mortality
 - 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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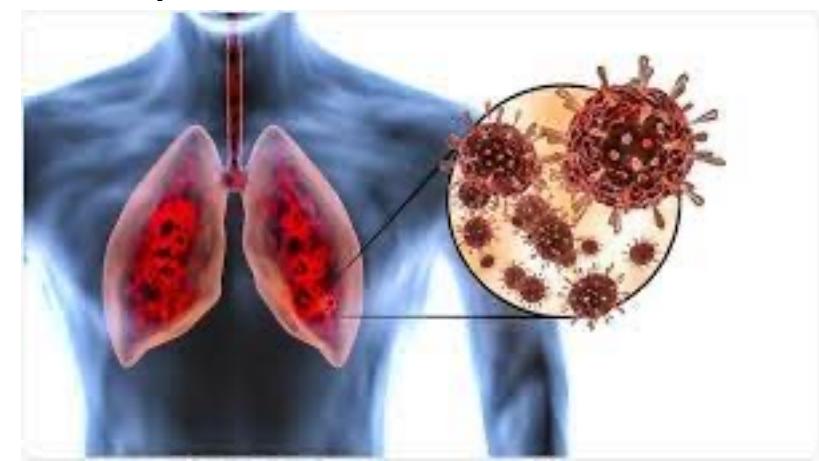


Self-reactive antibodies (ANA) **promote survival by downregulating worms in crash years** but **impede survival via immunopathology in peak years!**

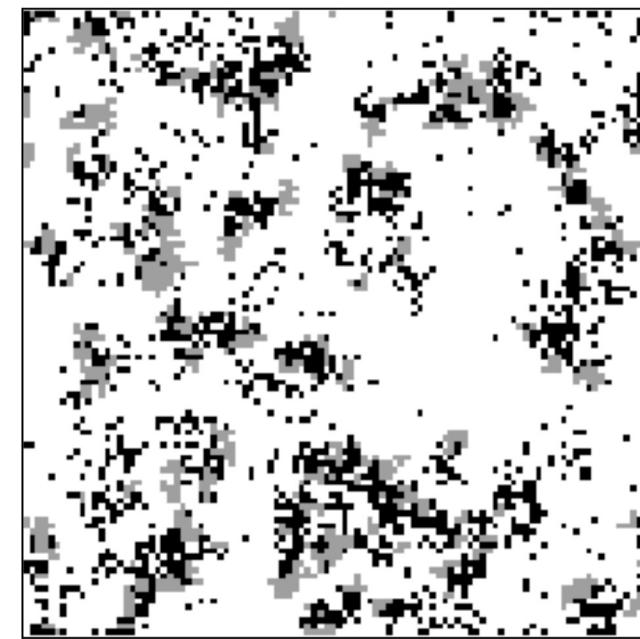
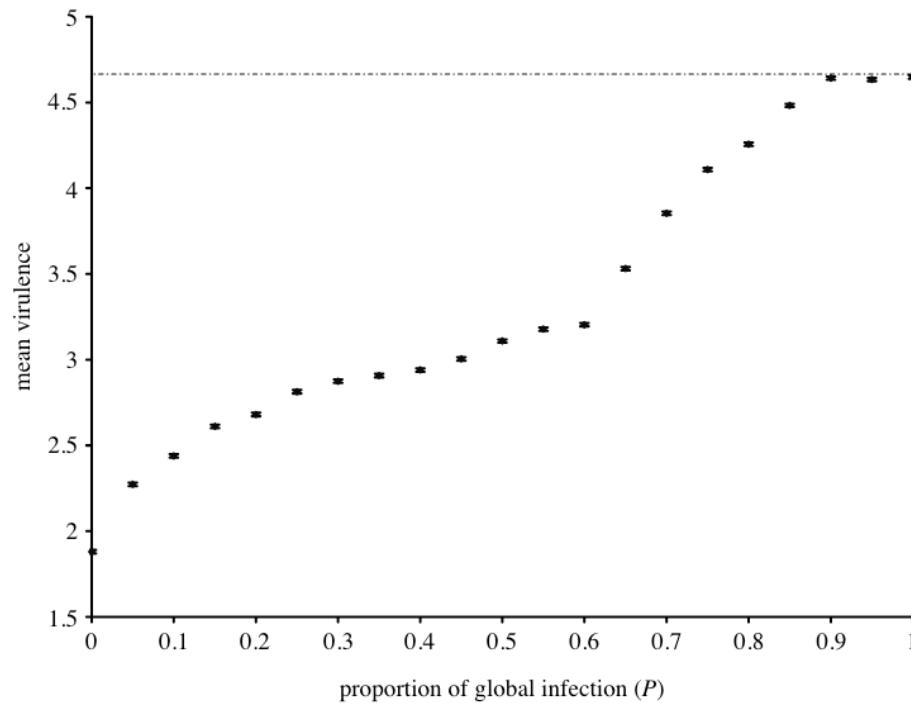


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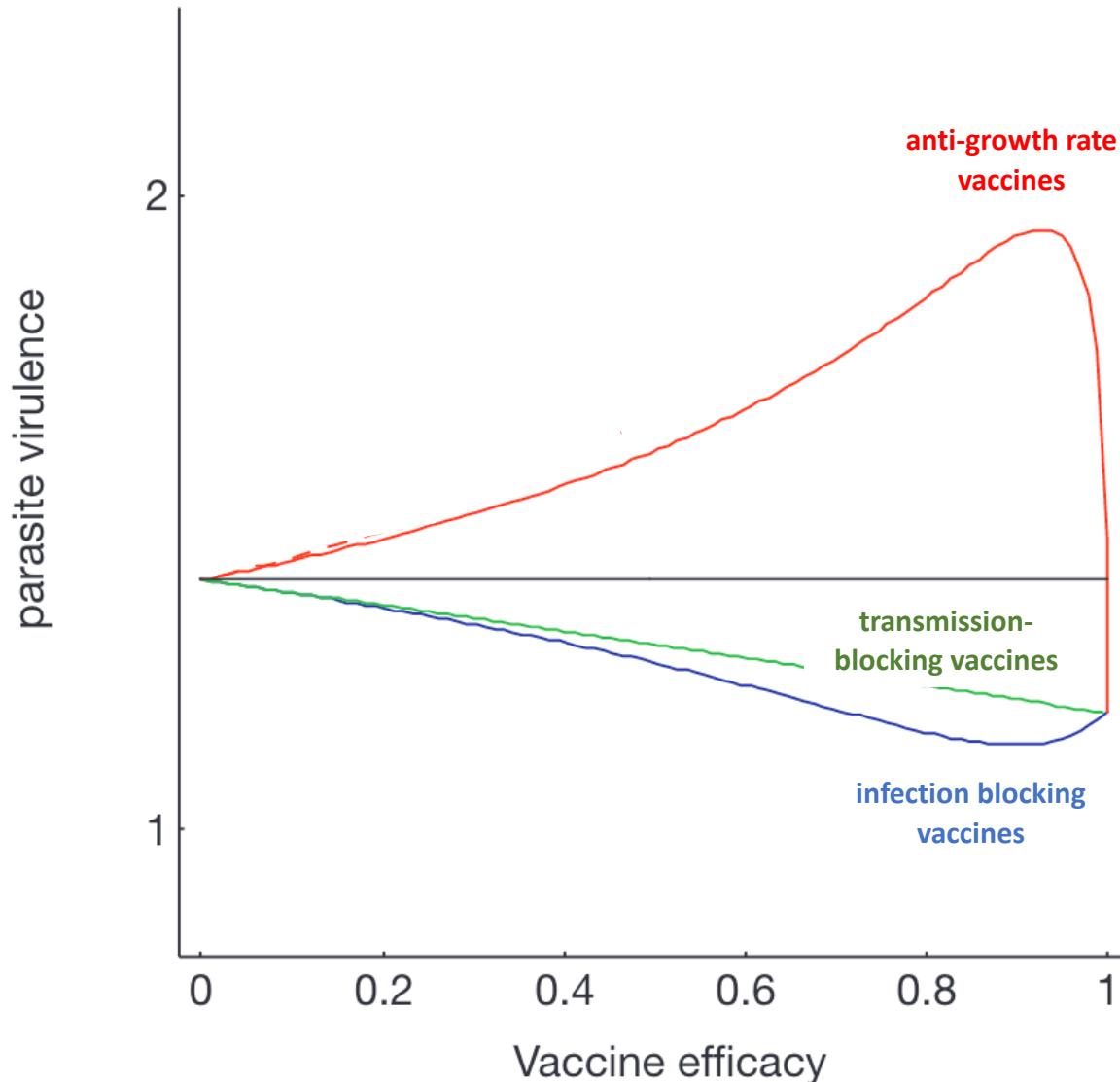
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 - For example: Fitness effects on reproduction vs. adult mortality
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 - In the case of zoonoses, the bulk of our measurements may be derived from a different host than the one in which the virus evolved
- 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)



Spatial structuring generally favors reduced pathogen virulence.

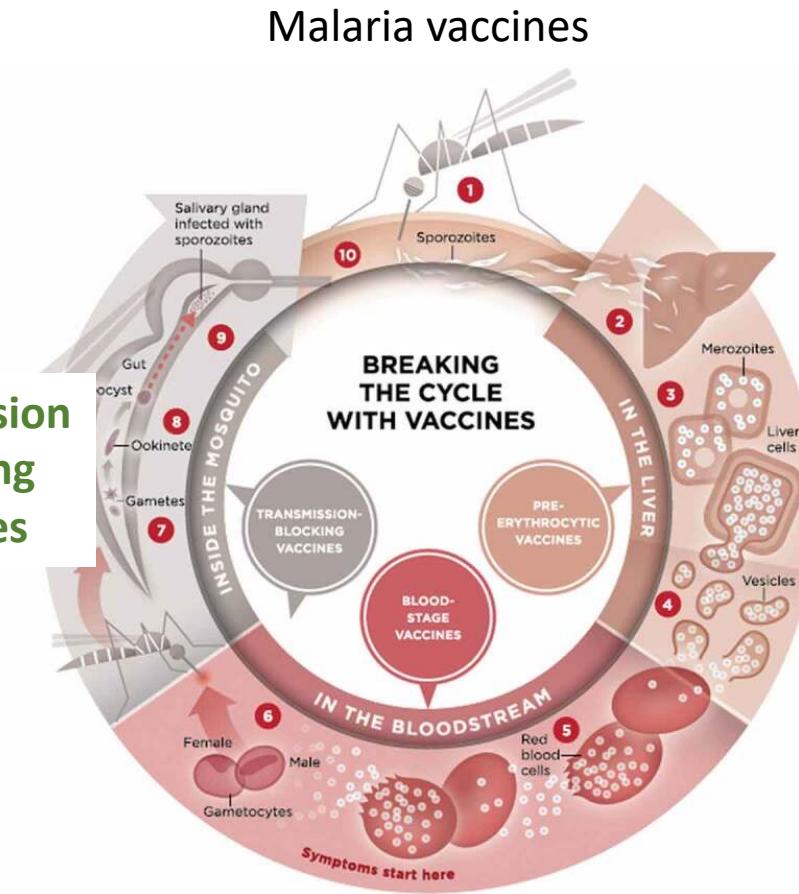


Imperfect vaccination can support the evolution of higher virulence, depending on the lifestage of the pathogen that is targeted.



transmission
-blocking
vaccines

anti-growth
rate vaccines



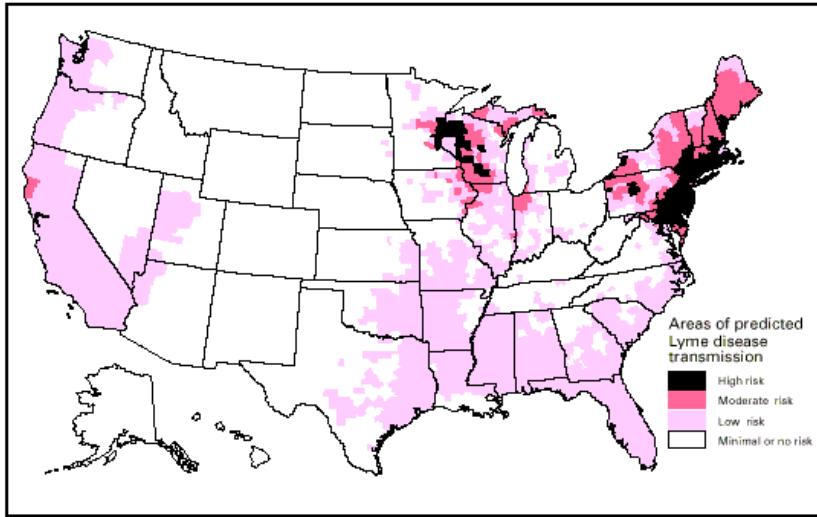
infection
blocking
vaccines

Malaria vaccines

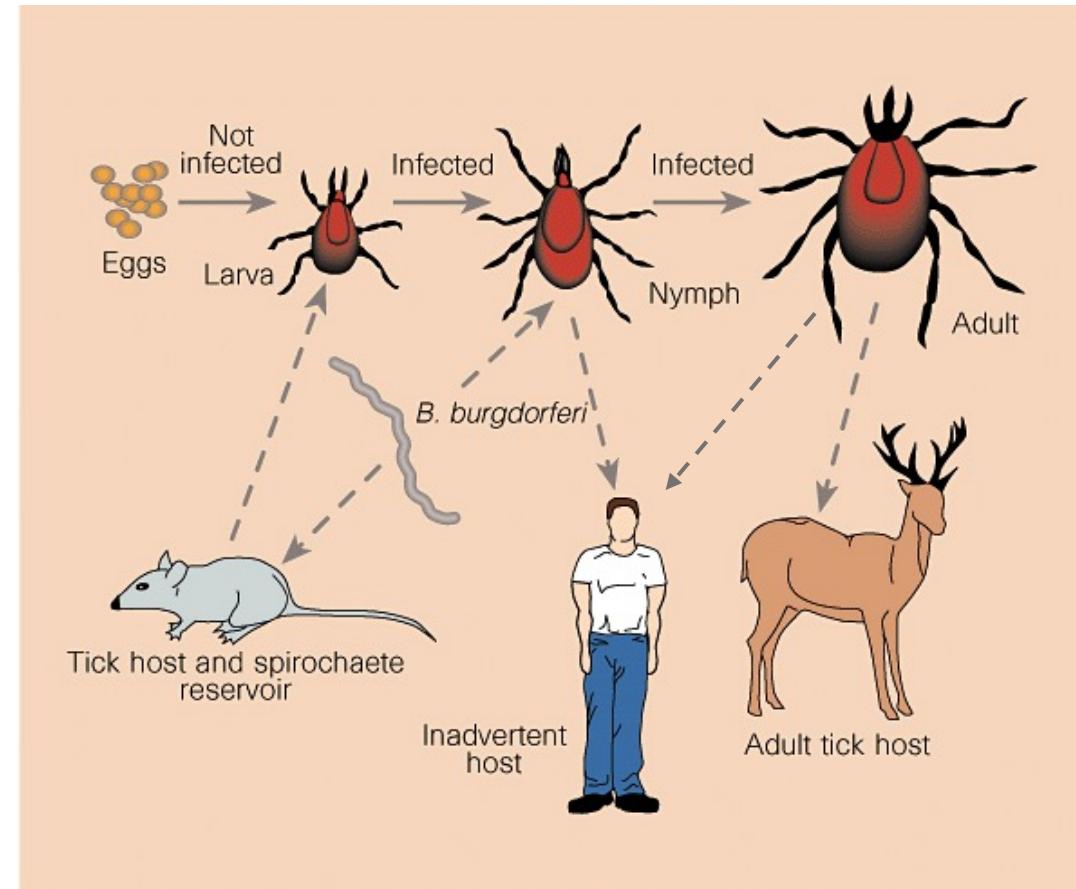
Disease dynamics in the broader community

Example: Lyme Disease

National Lyme disease risk map with four categories of risk

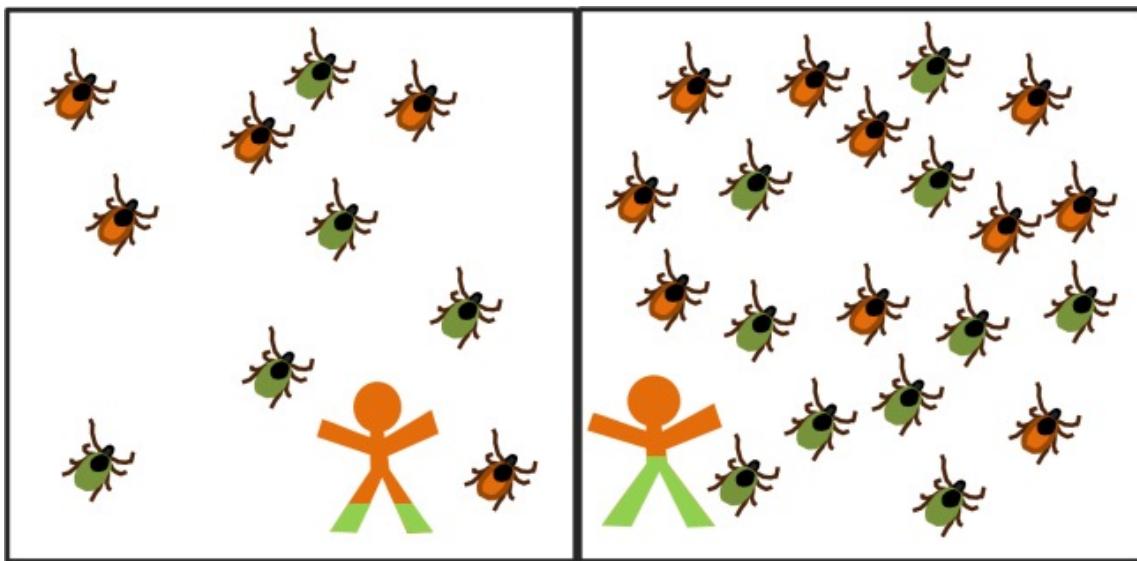


- Lyme disease is a vector-borne disease caused by the bacterium, *Borrelia burgdorferi*, vectored by *Ixodes* especially *Ixodes scapularis* ticks.
- Nymph ticks are borne in the spring, feed on small mammal hosts through the summer, then reproduce (particularly on deer) in the fall before going dormant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



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

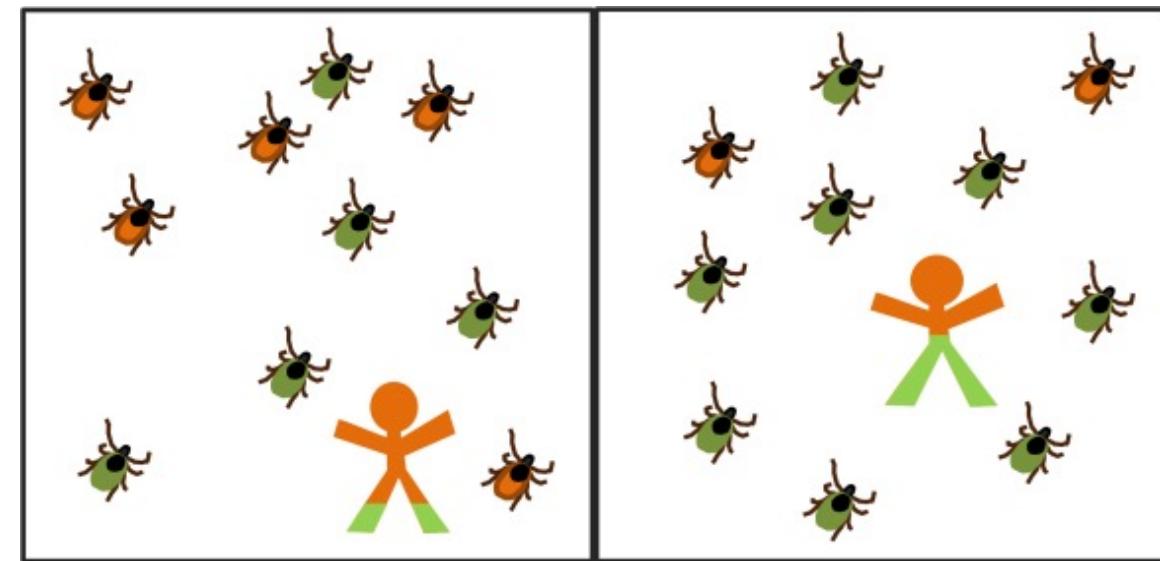
Scenario 1: Density of infected ticks



Area = 1 unit
Total Ticks = 10
Tick Density = 10 ticks/unit
Number Infected Ticks = 5
Prevalence Infected Ticks = 50%

Area = 1 unit
Total Ticks = 20
Tick Density = 20 ticks/unit
Number Infected Ticks = 10
Prevalence Infected Ticks = 50%

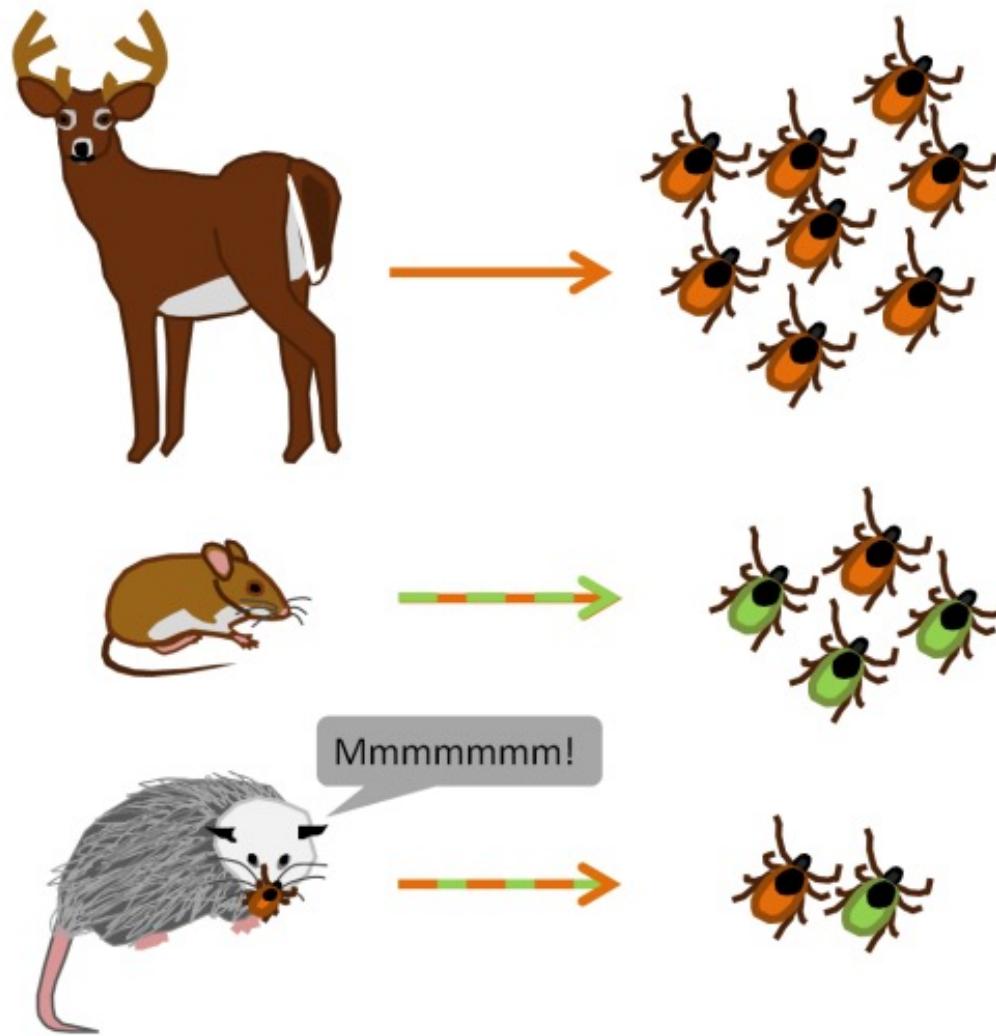
Scenario 2: Prevalence of Lyme in tick population



Area = 1 unit
Total Ticks = 10
Tick Density = 10 ticks/unit
Number Infected Ticks = 5
Prevalence Infected Ticks = 50%

Area = 1 unit
Total Ticks = 10
Tick Density = 10 ticks/unit
Number Infected Ticks = 8
Prevalence Infected Ticks = 80%

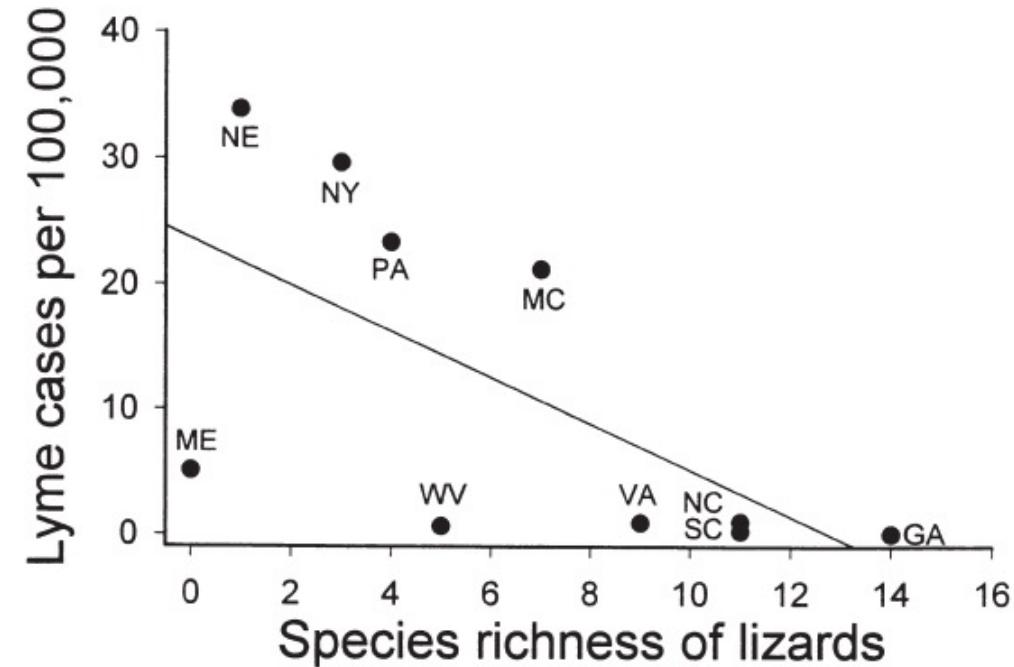
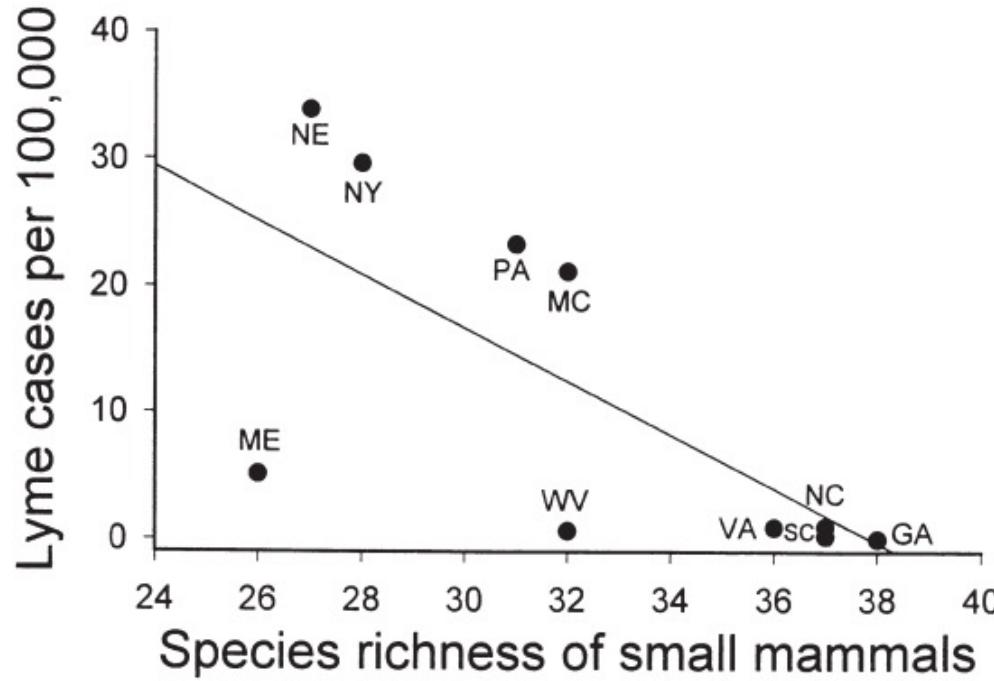
The broader **wildlife community impacts Lyme disease risk** for humans



- (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. burgdorferi* infection.

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

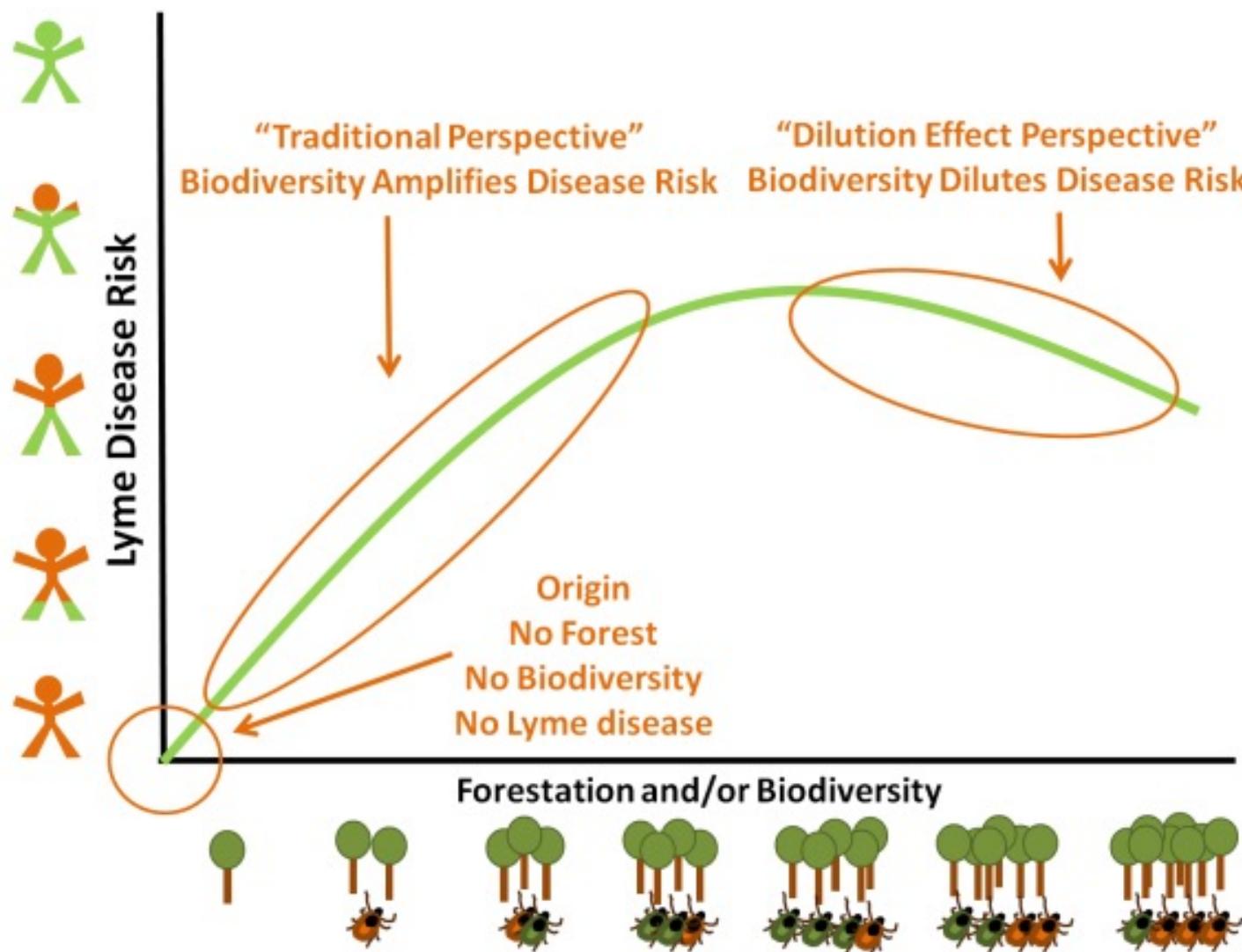
While a popular concept, it only holds in select cases!



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

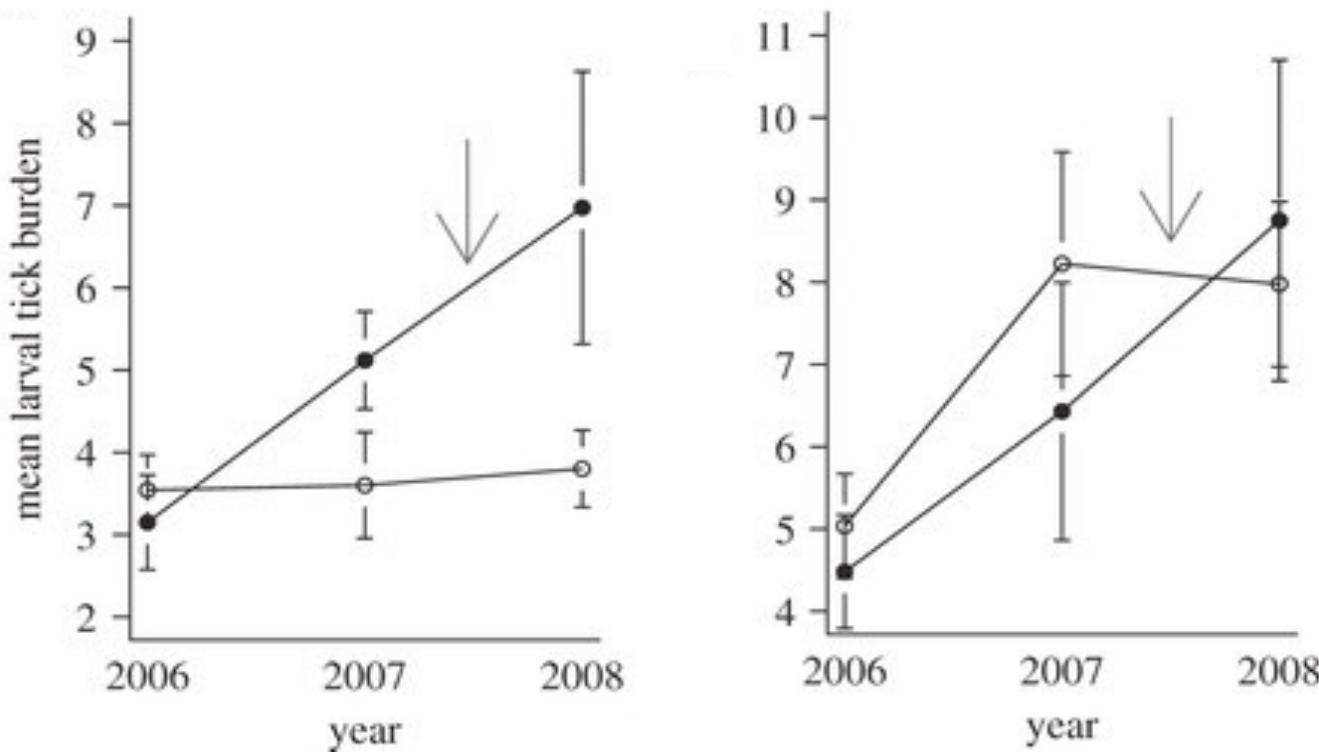
Patterns depend on the context of the wildlife community!

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



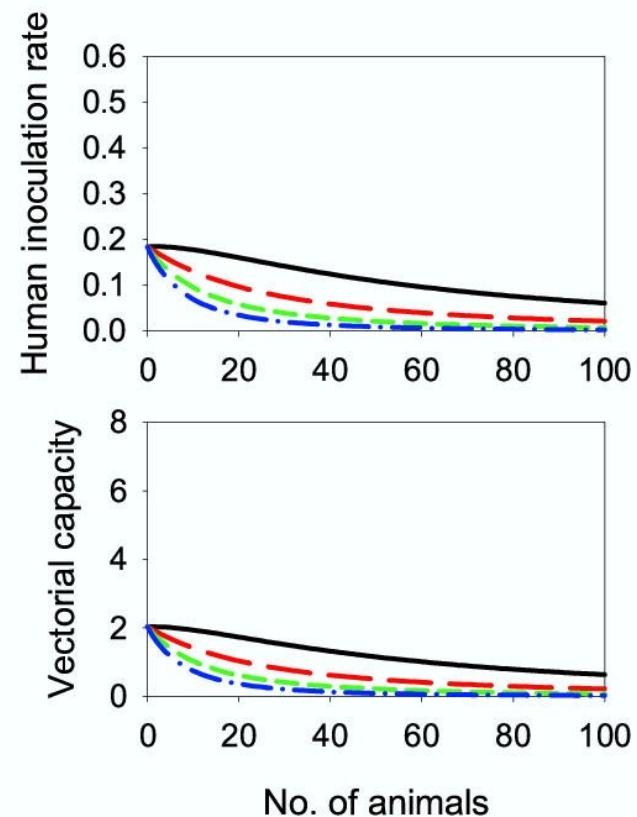
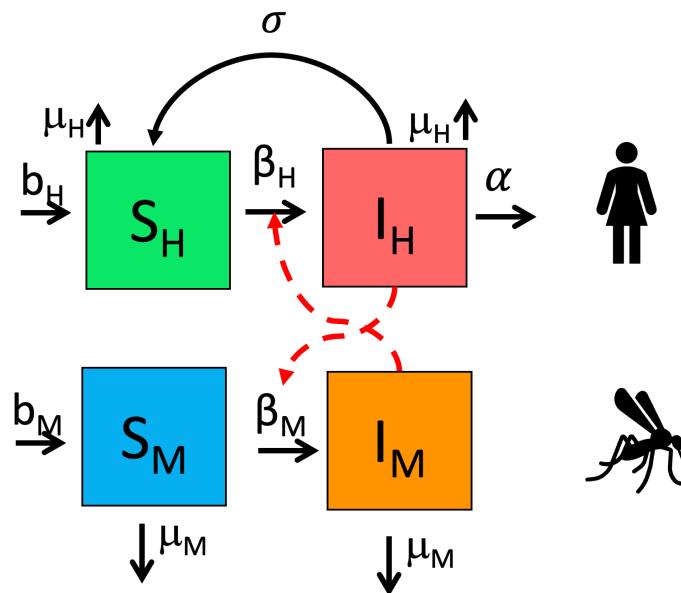
“Zooprophylaxis” is the diversion of pathogen-transmitting arthropods from humans to animals

Tick burden on female (left) and male (right) 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



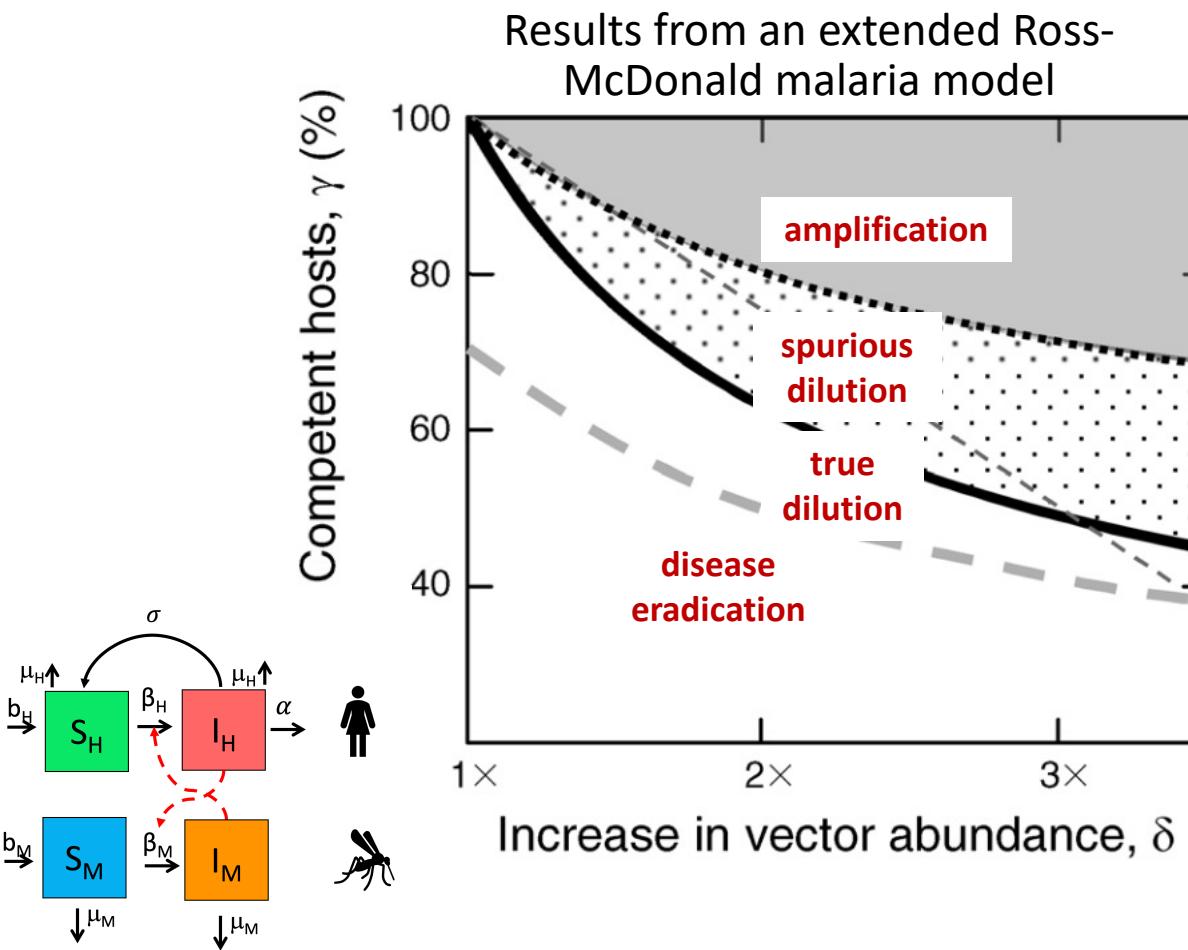
Sceloporus occidentalis

“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.

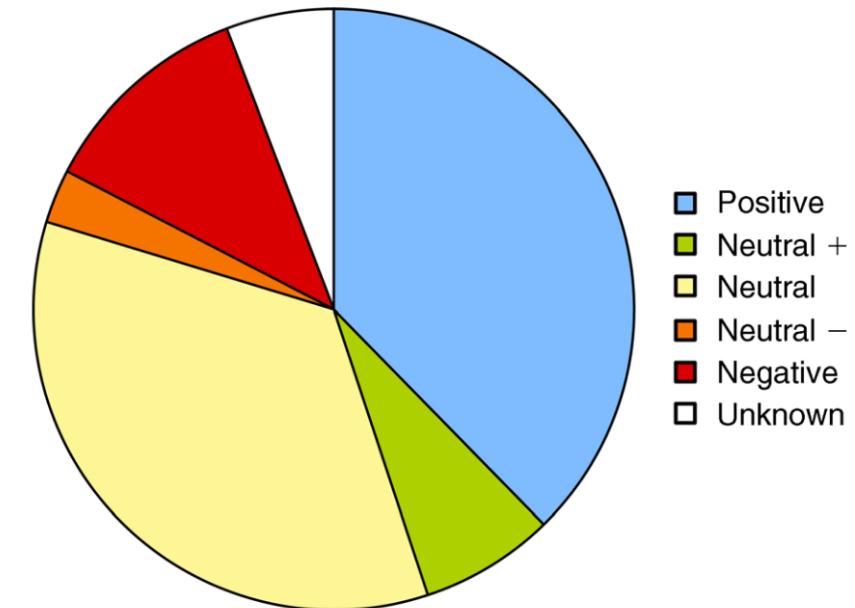


Black, red, green, blue = 0, 20, 40 or 60% chance
of being killed as a result of feeding on animals

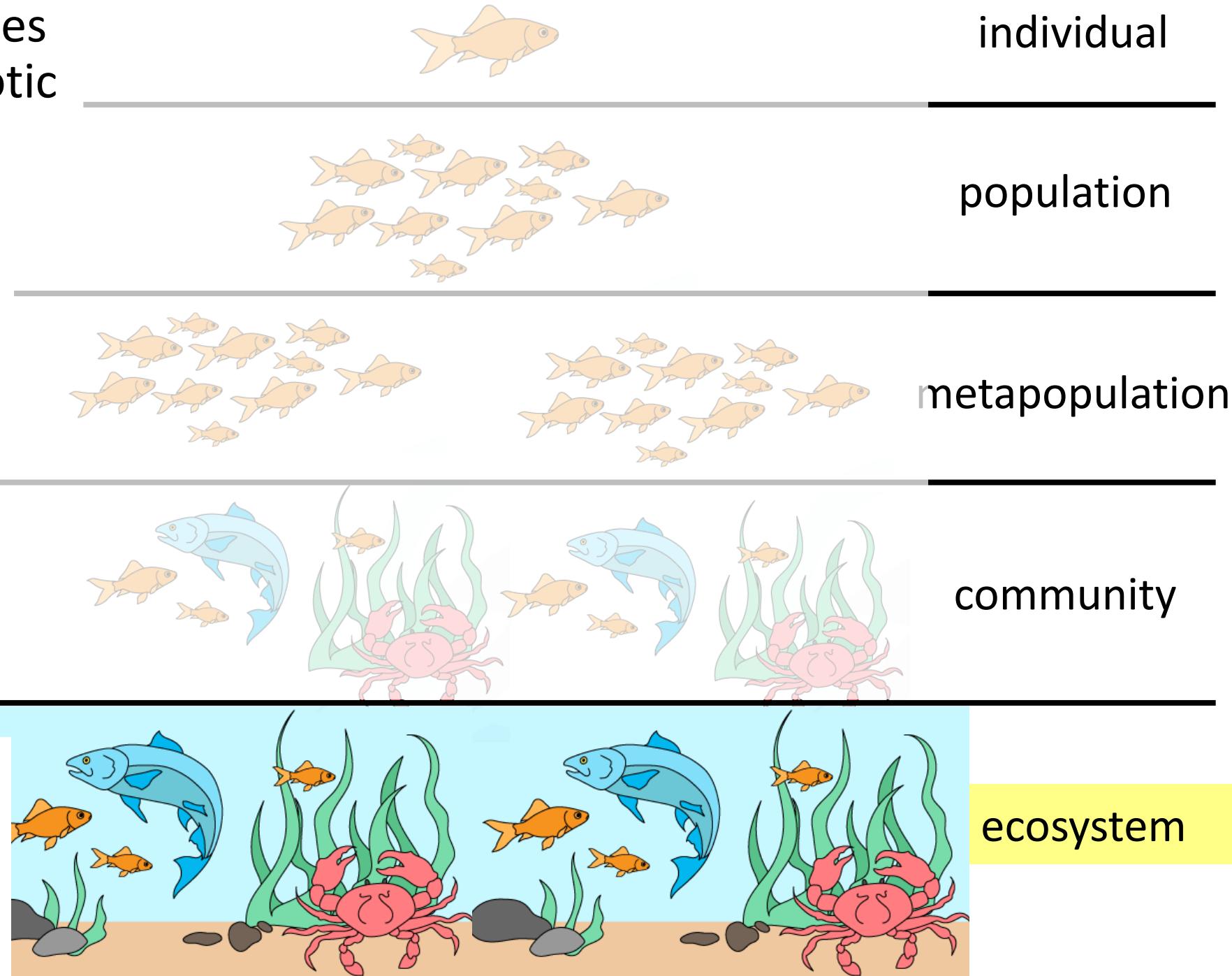
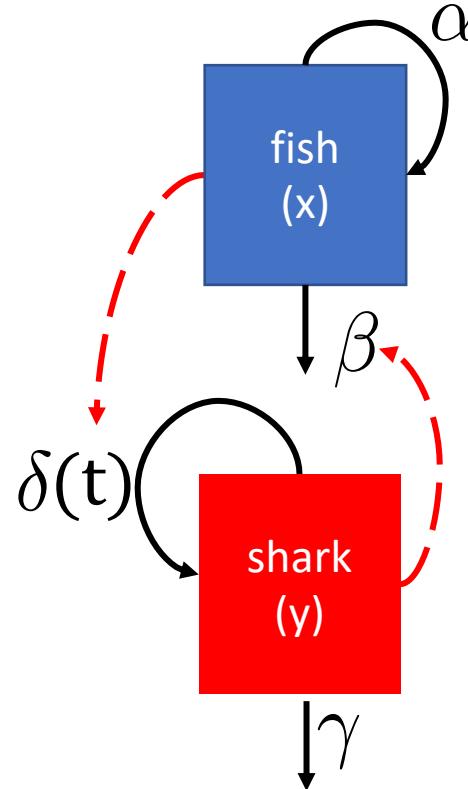
Understanding the **underlying transmission dynamics** of the system can help predict how **wildlife biodiversity** might **amplify or dilute** human disease risk in different contexts.



Meta-analysis of biodiversity impacts on 69 common human pathogens



Ecosystem = communities interacting with the abiotic environment



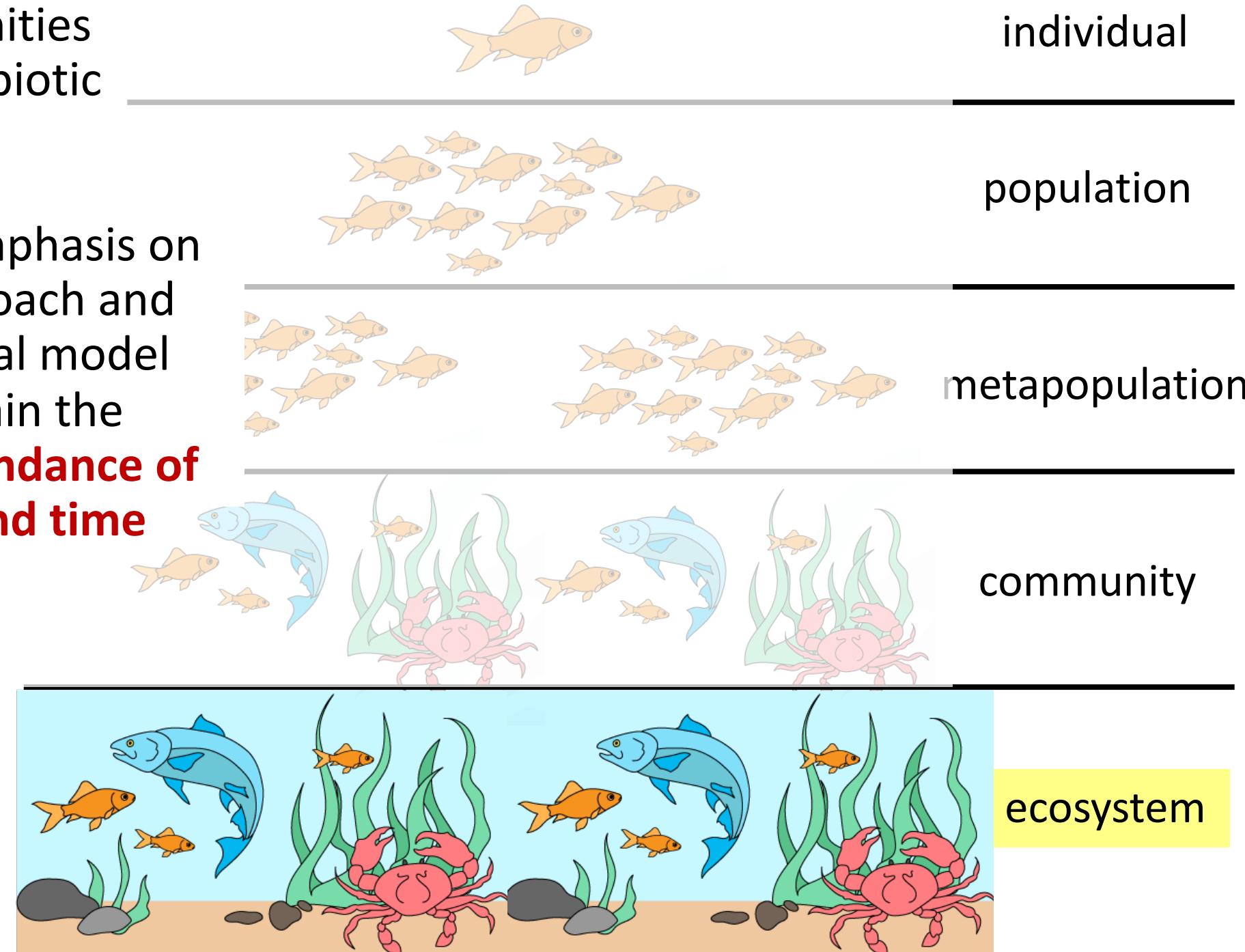
*How does fish abundance **vary** with changes in shark birth rates with **temperature**?*

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**

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



Mathematical

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

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

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

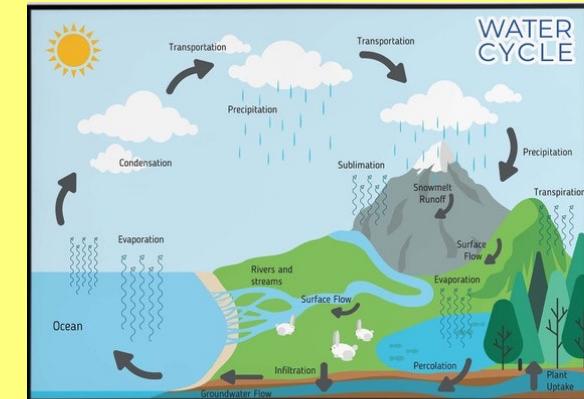
Human Genetics



Human Disease



Conceptual



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

One process of community assembly is ecological **succession**.

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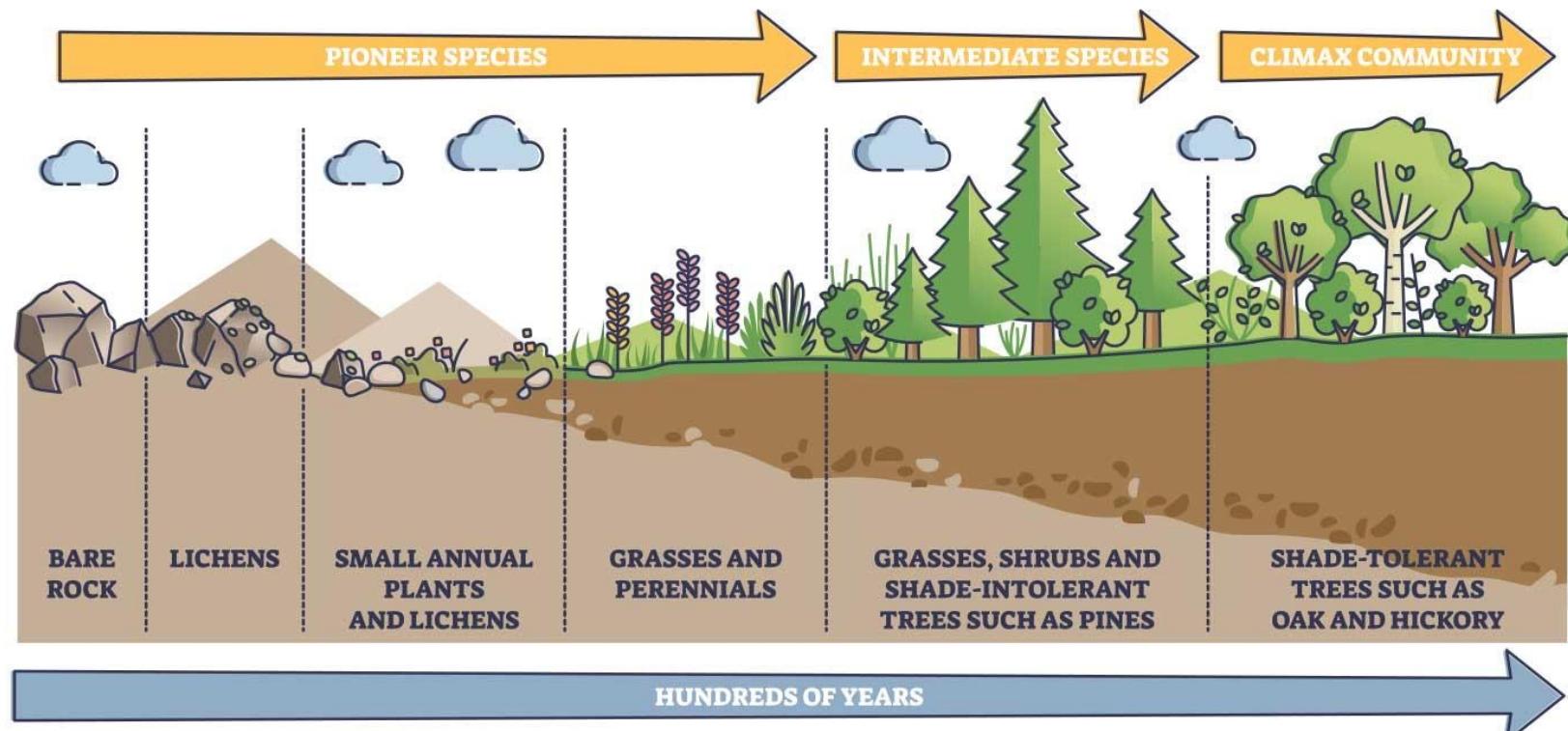
- Succession is the **process of change** in the **species structure** of ecological communities with time.
- Community begins with **pioneer species**, then develops with increasing complexity that self-reinforces to establish a **climax community**.
- Henry Chandler Cowles, a professor at the University 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.

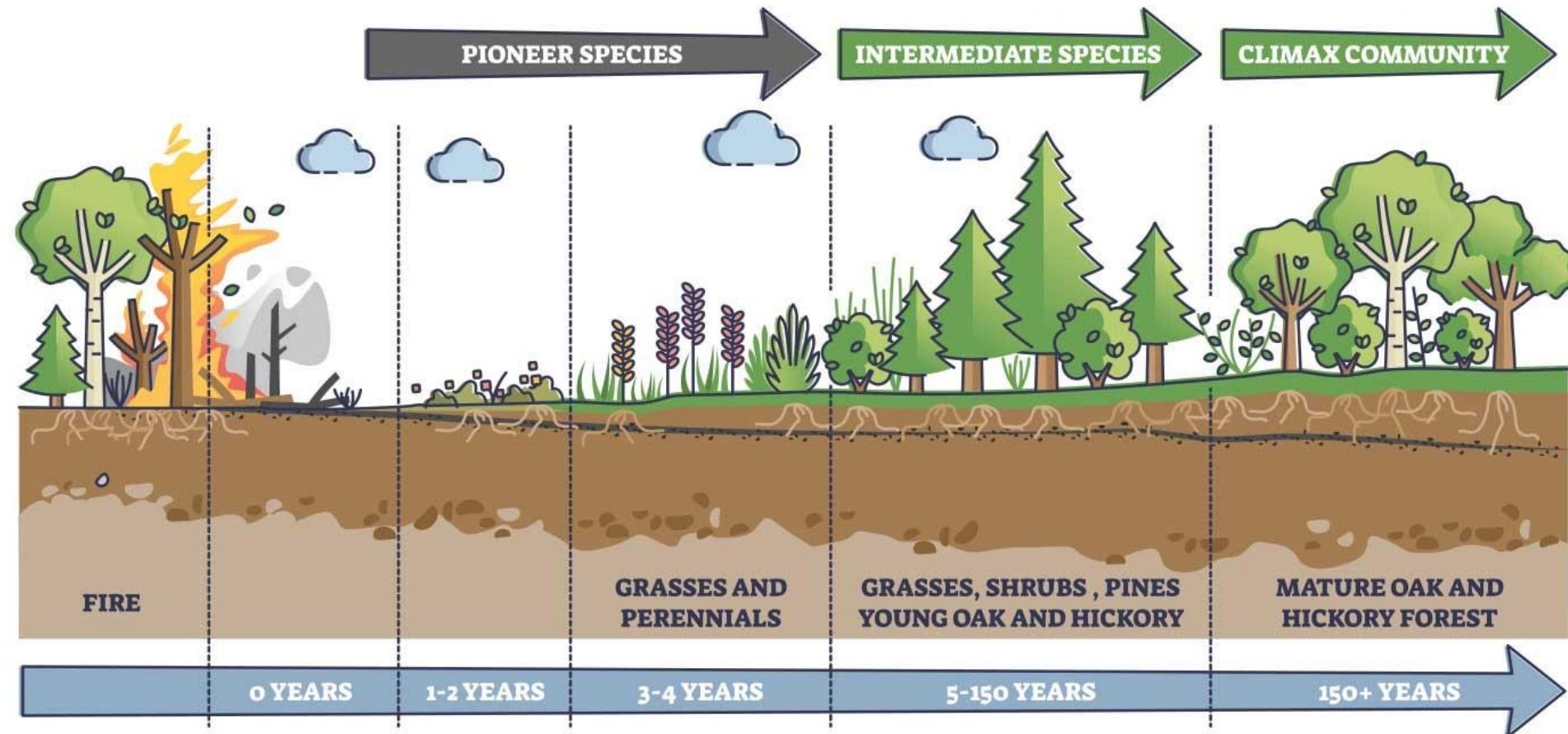


$$\frac{dN}{dt} = rN \left(1 - \frac{N}{K}\right)$$



*Continuum from “*r-selected*” → “*K-selected*” species.*

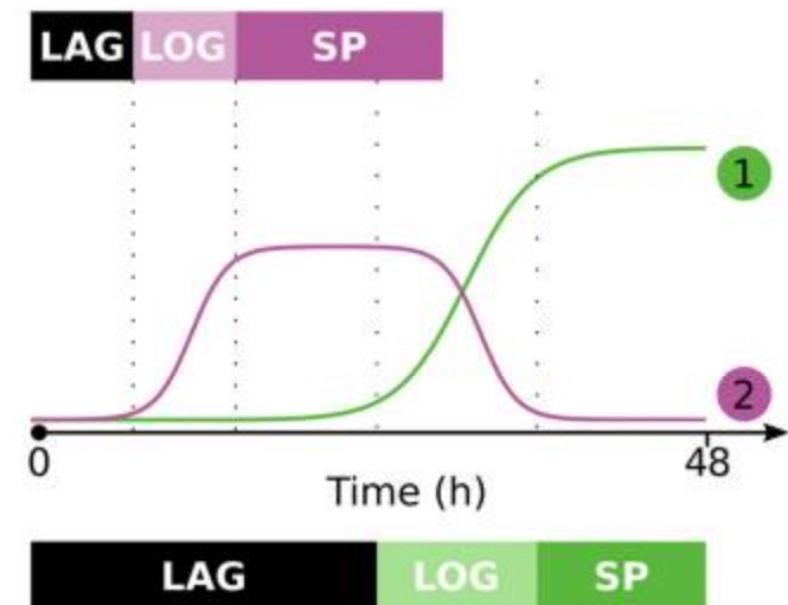
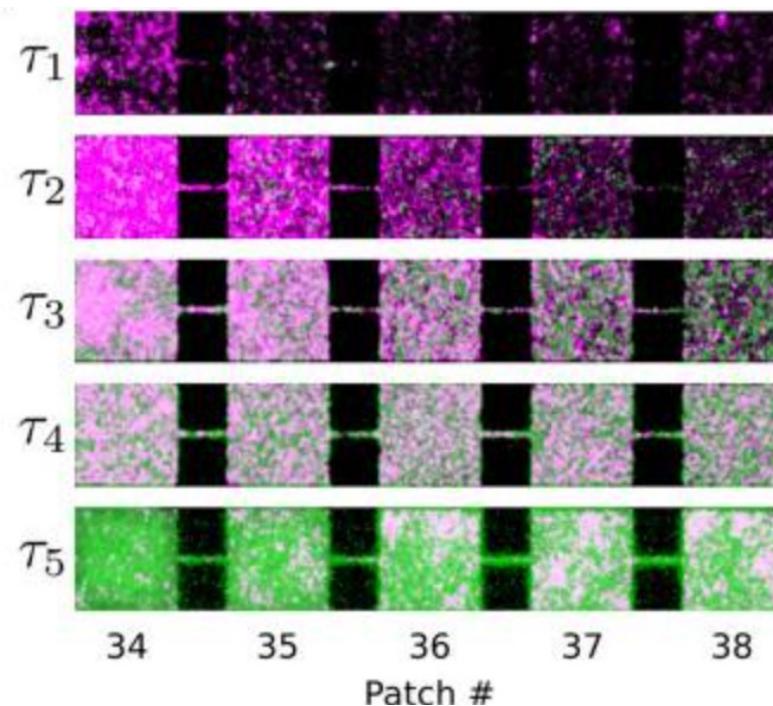
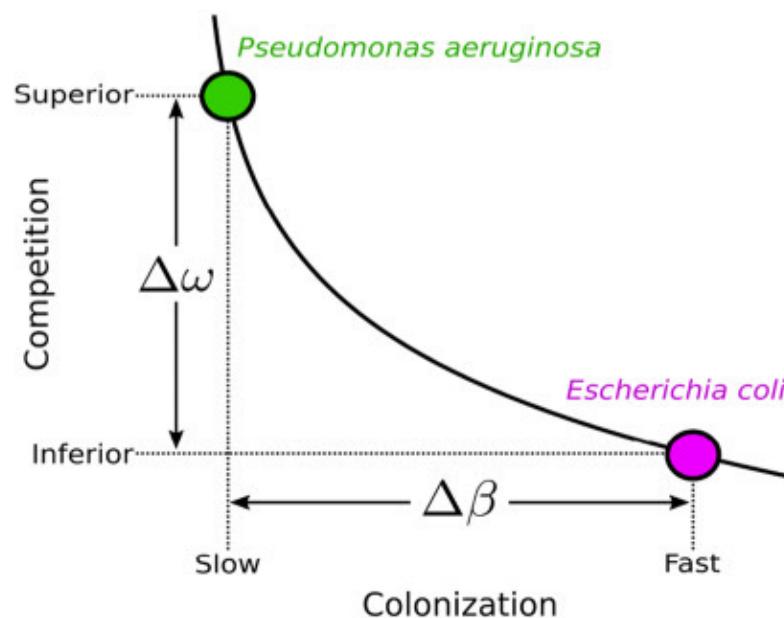
Secondary succession occurs when an environmental disturbance displaces a climax community, but soil and nutrients are still retained.



*Continuum from “**r-selected**” → “**K-selected**” species.*

Succession also occurs in microbial systems.

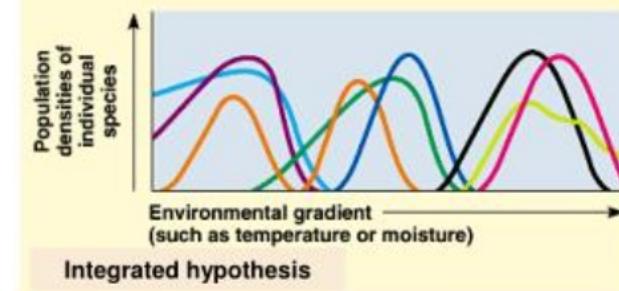
Here, the “**K-selected**” superior competitor eventually replaces the “**r-selected**” fast colonizer.



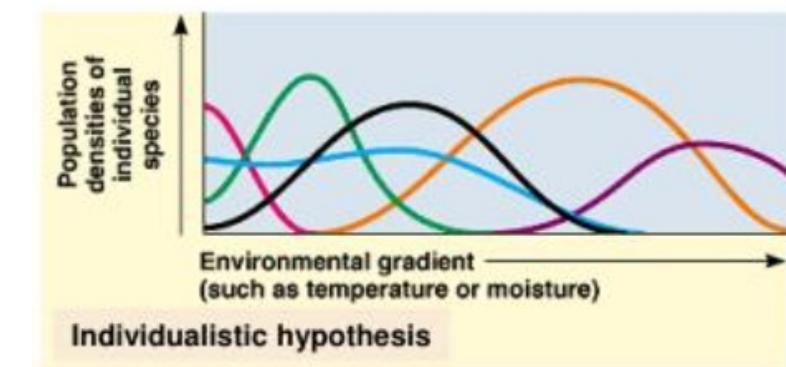
Superorganisms vs. Loose Collections of Species

- Frederic Clements (1916) argued that community succession was predictable and **deterministic**, much like ontogenetic development in individual organisms, moving always towards some superorganism.

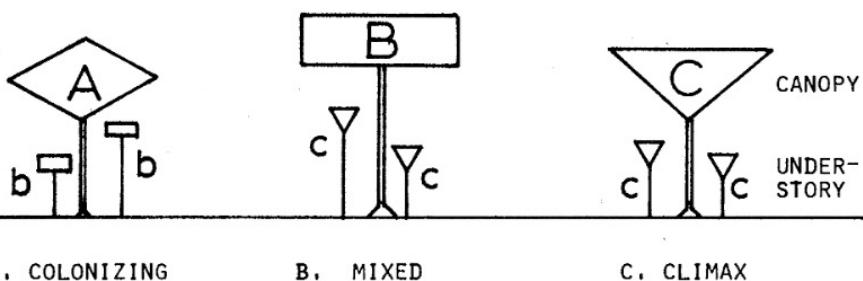
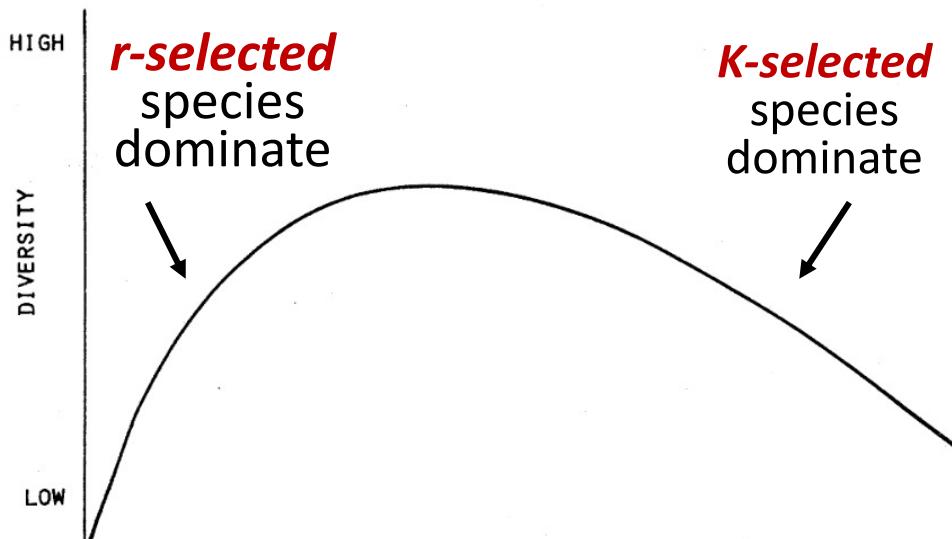
- **Priority effects:** inhibitory or facilitative priority effects occur when one species “prepares” the environment for the next species in succession



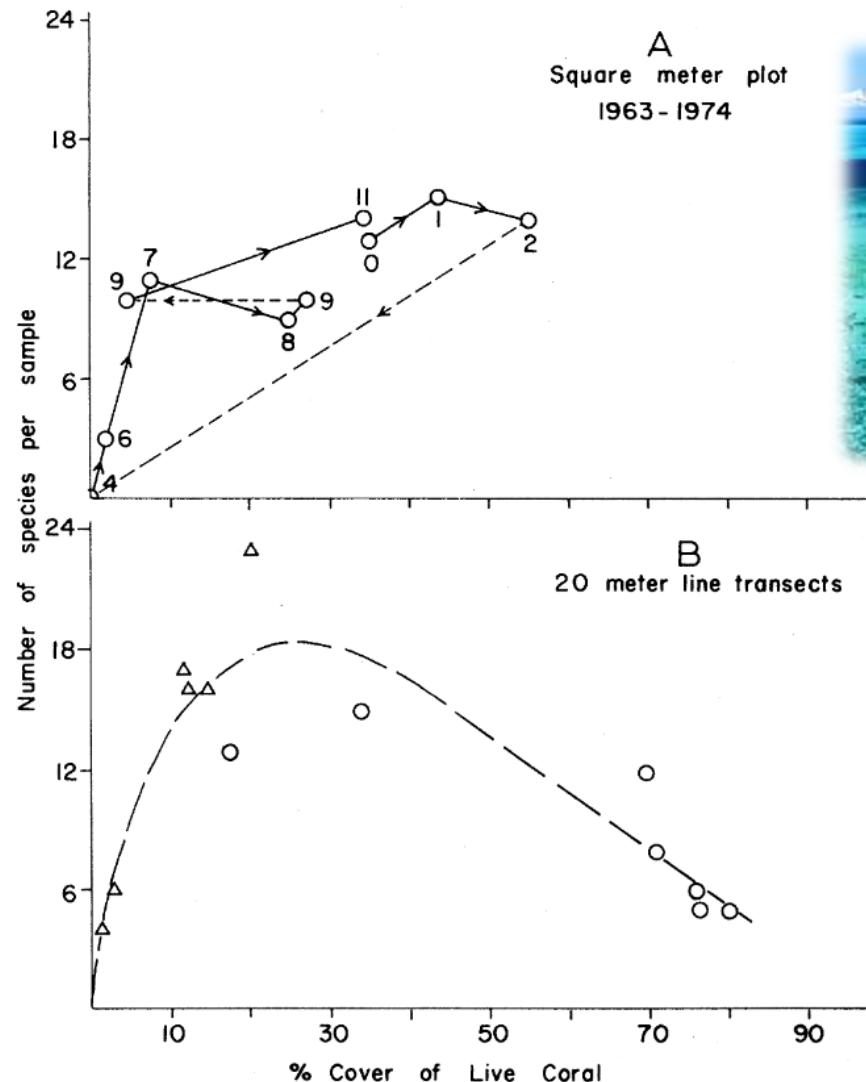
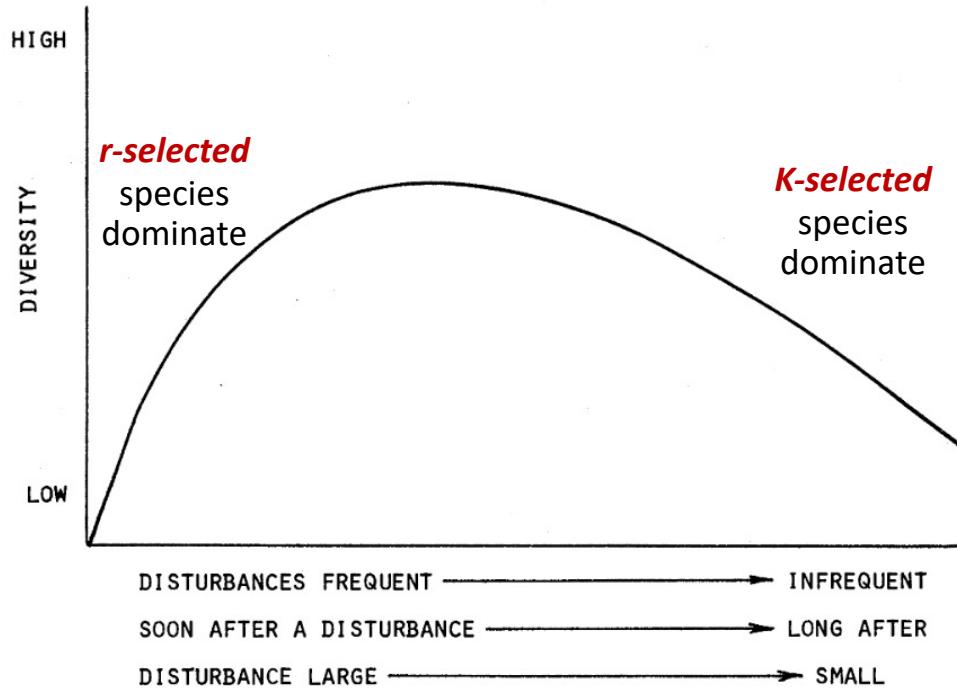
- Henry Gleason (1926) argued instead that chance favored the dispersal of nearby species into available habitat for succession, leading to **stochastic** assembly of communities
- Closer to Cowles' original thinking



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.



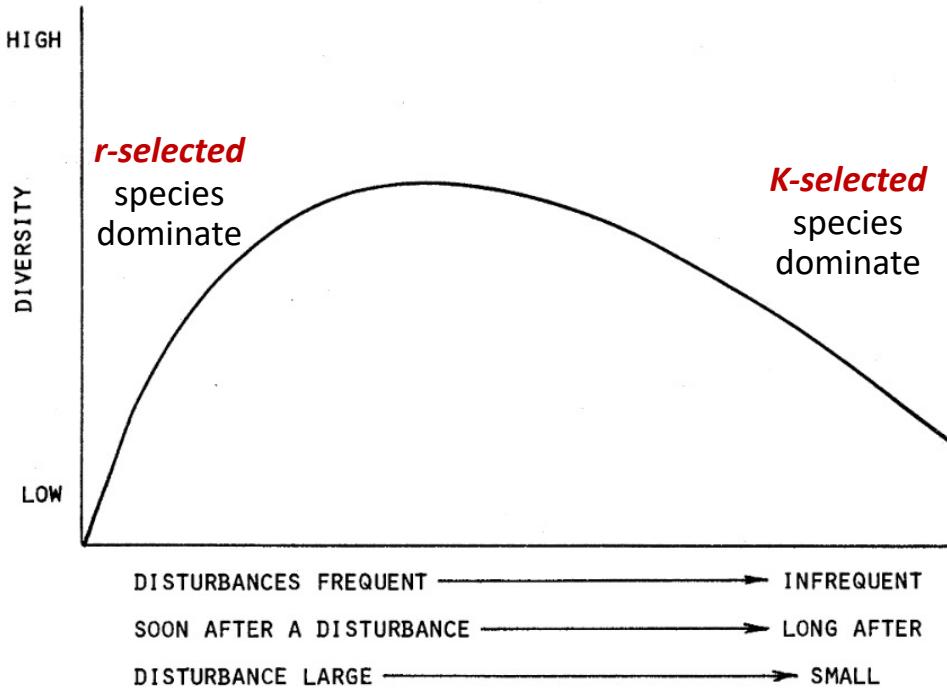
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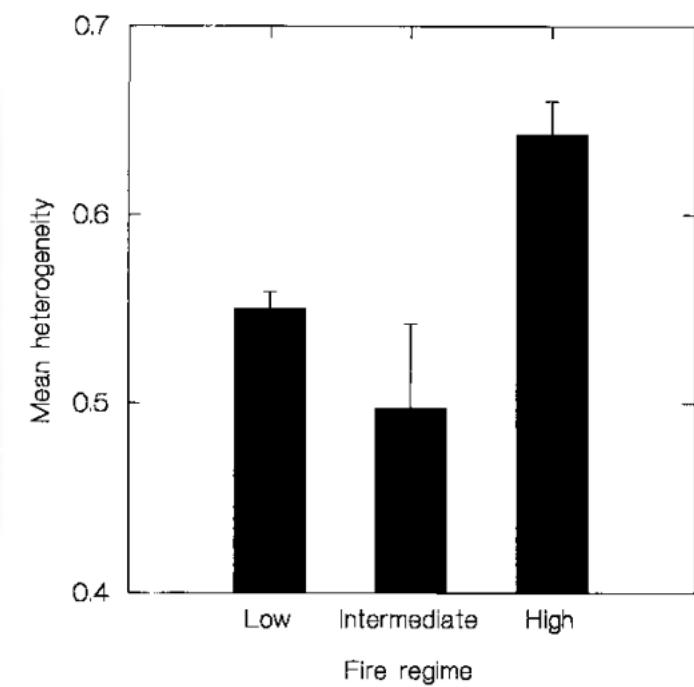
Heron Island, Australia

Sometimes it seems to be correct!

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.



Sometimes it is not so well-supported empirically!



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

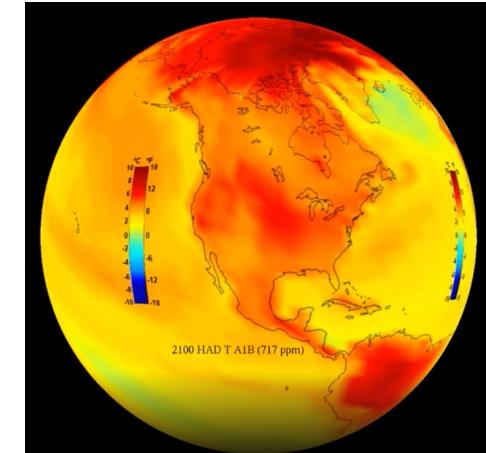
Human



Solar System



Climate



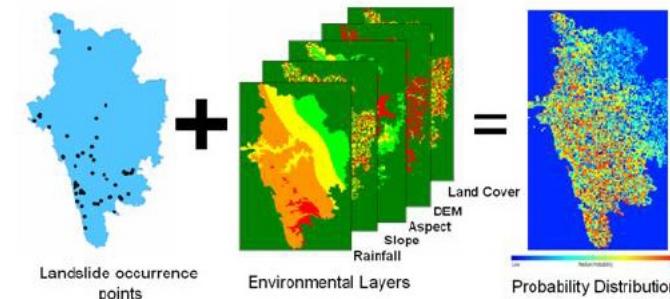
Human Genetics



Human Disease



Species Distribution



The field of **biogeography** studies the geographical distribution of plants and animals

- Larger areas have more species!

$$S = cA^z$$

↑ ↑ ← slope of
number of habitat relationship
species area in log-log
 space

constant based on
unit of area
(standardizes to
expected number of
species per single
unit area)

The field of **biogeography** studies the geographical distribution of plants and animals

- Larger areas have more species!

$$S = cA^z$$

- The slope of the log-log relationship (z) will differ across diverse communities and ecosystems.

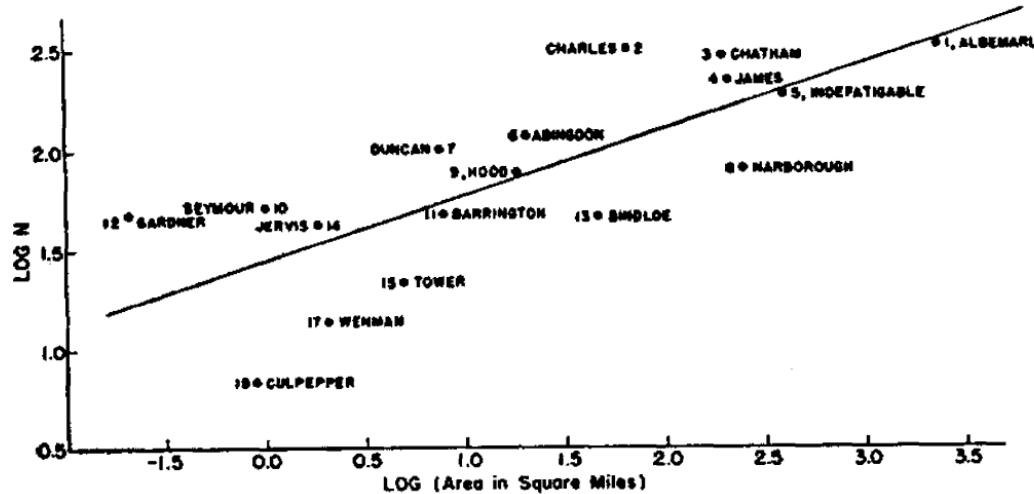
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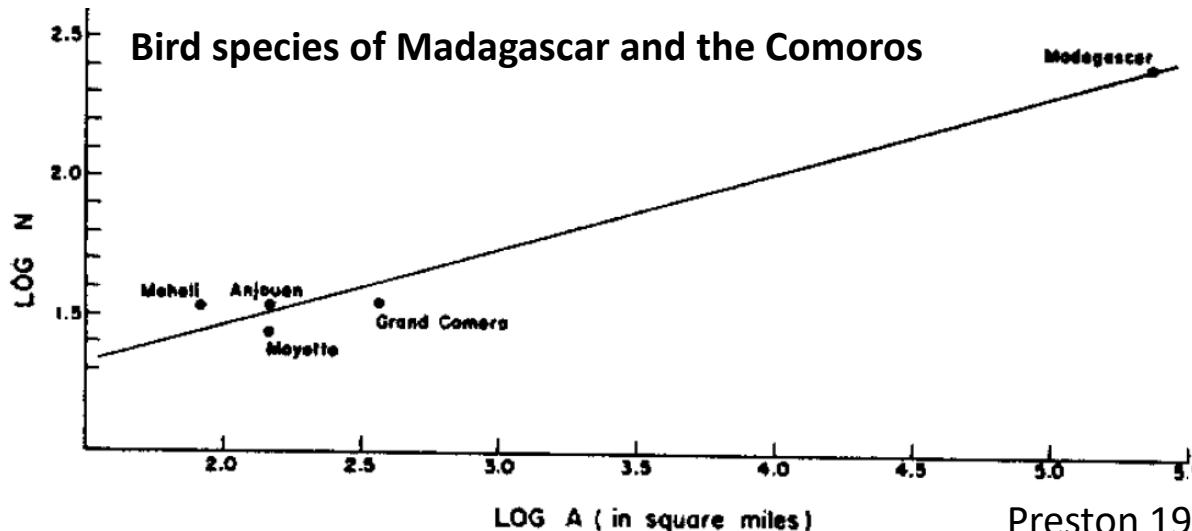
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Plant species of the Galapagos islands



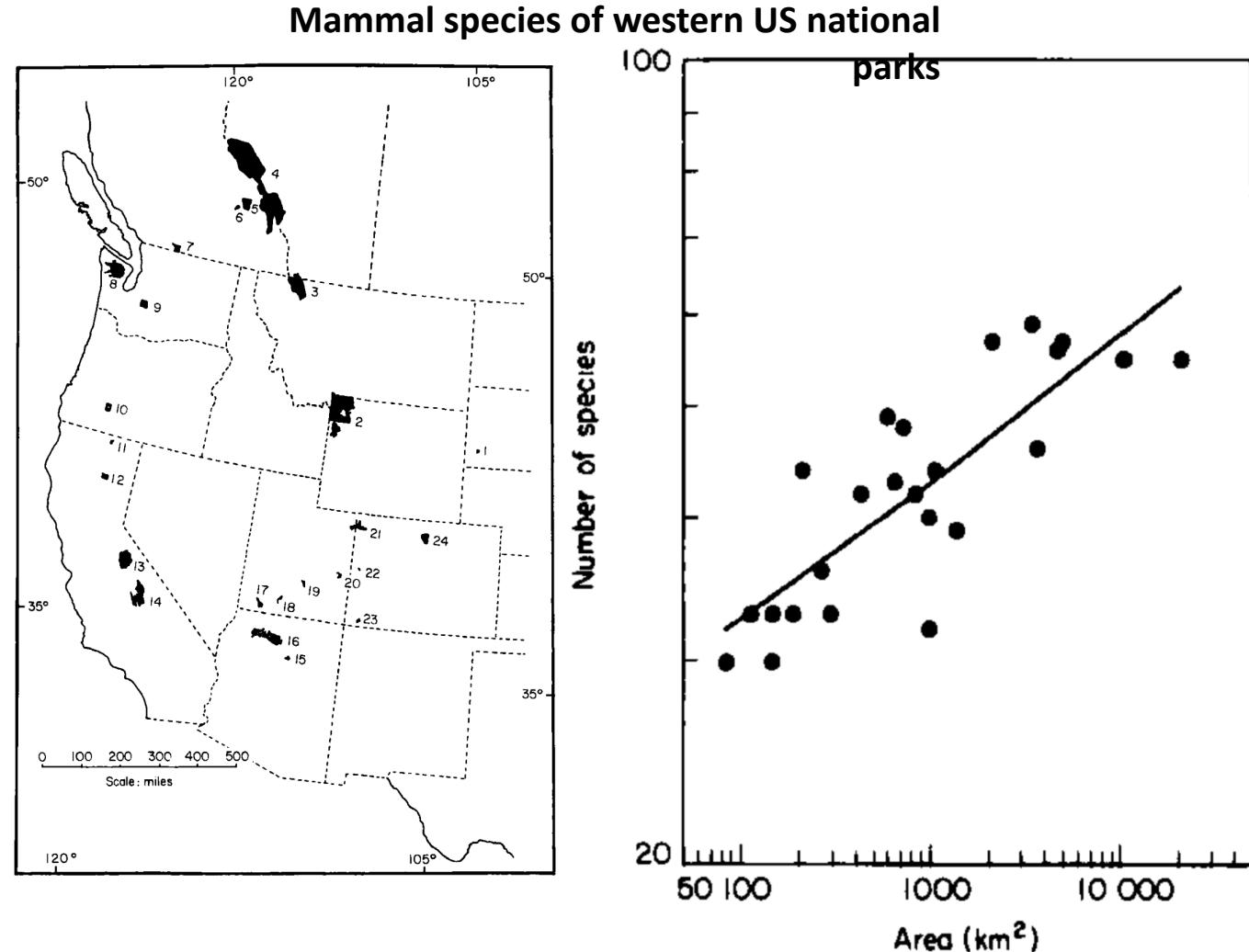
Bird species of Madagascar and the Comoros



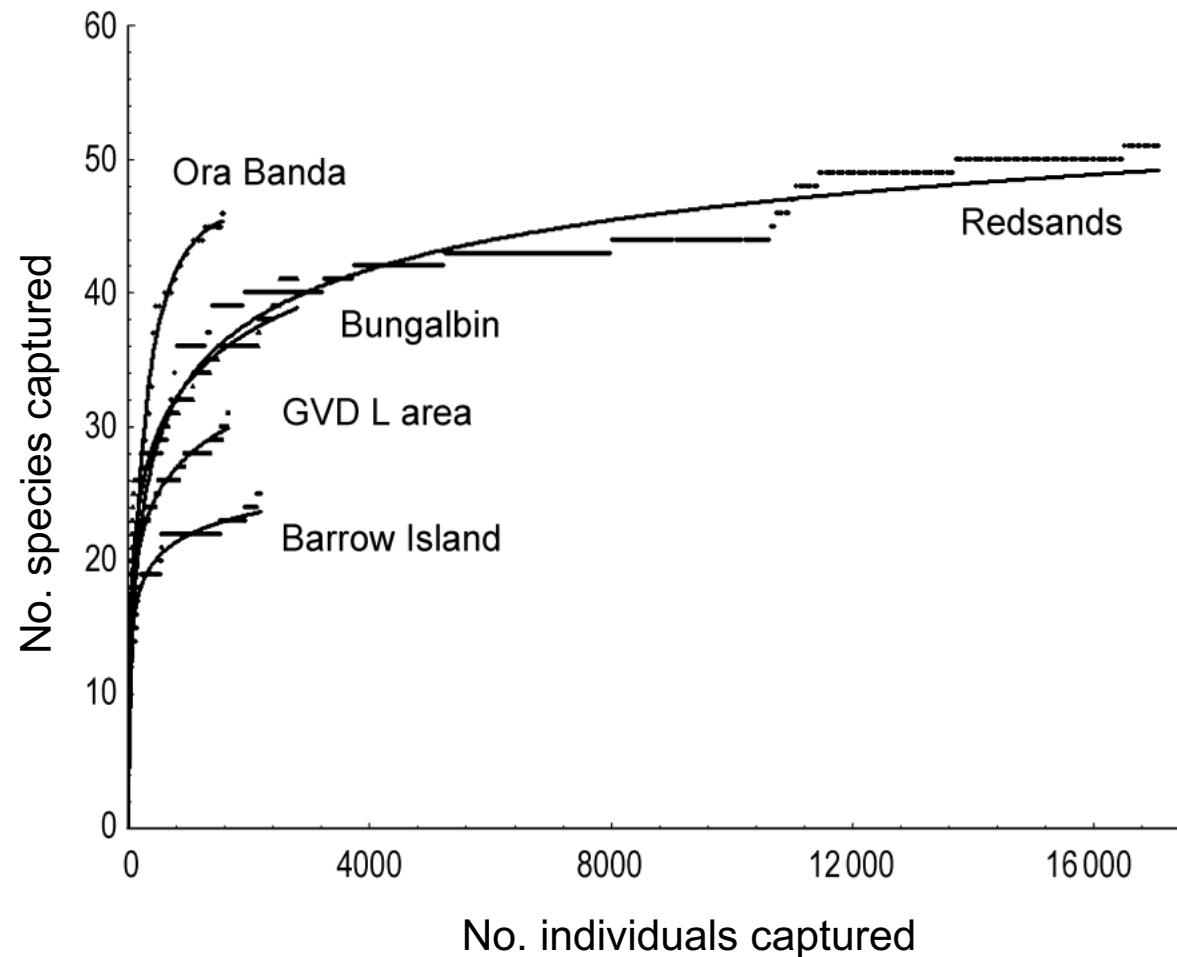
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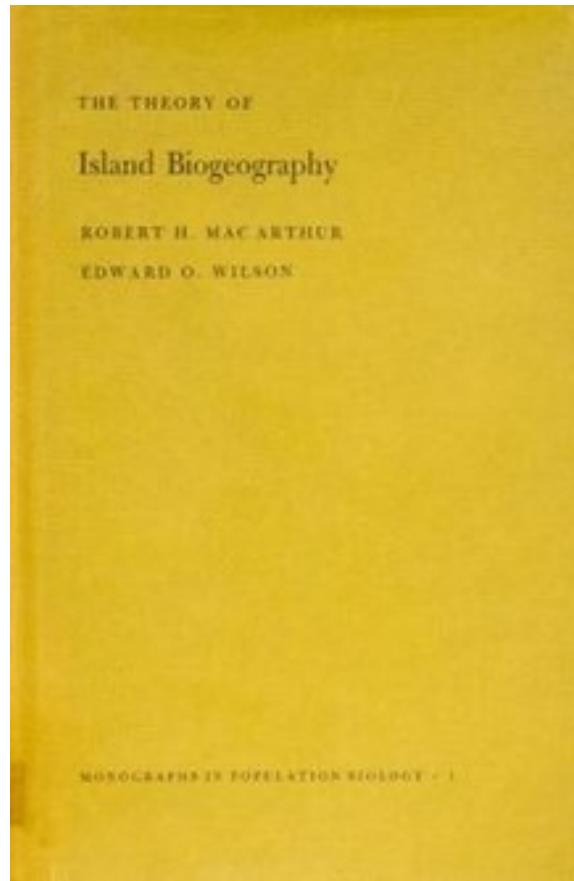


We can use the **species-area relationship (SAR)** to build **species accumulation curves** to understand if we have representatively sampled a population in field studies.



Building on the species-area relationship (SAR), MacArthur and Wilson proposed the **theory of island biogeography**.

This theory offers a null model for the number of species found in a given habitat, predictable from both the **size** of the habitat and its **distance** from a source population.



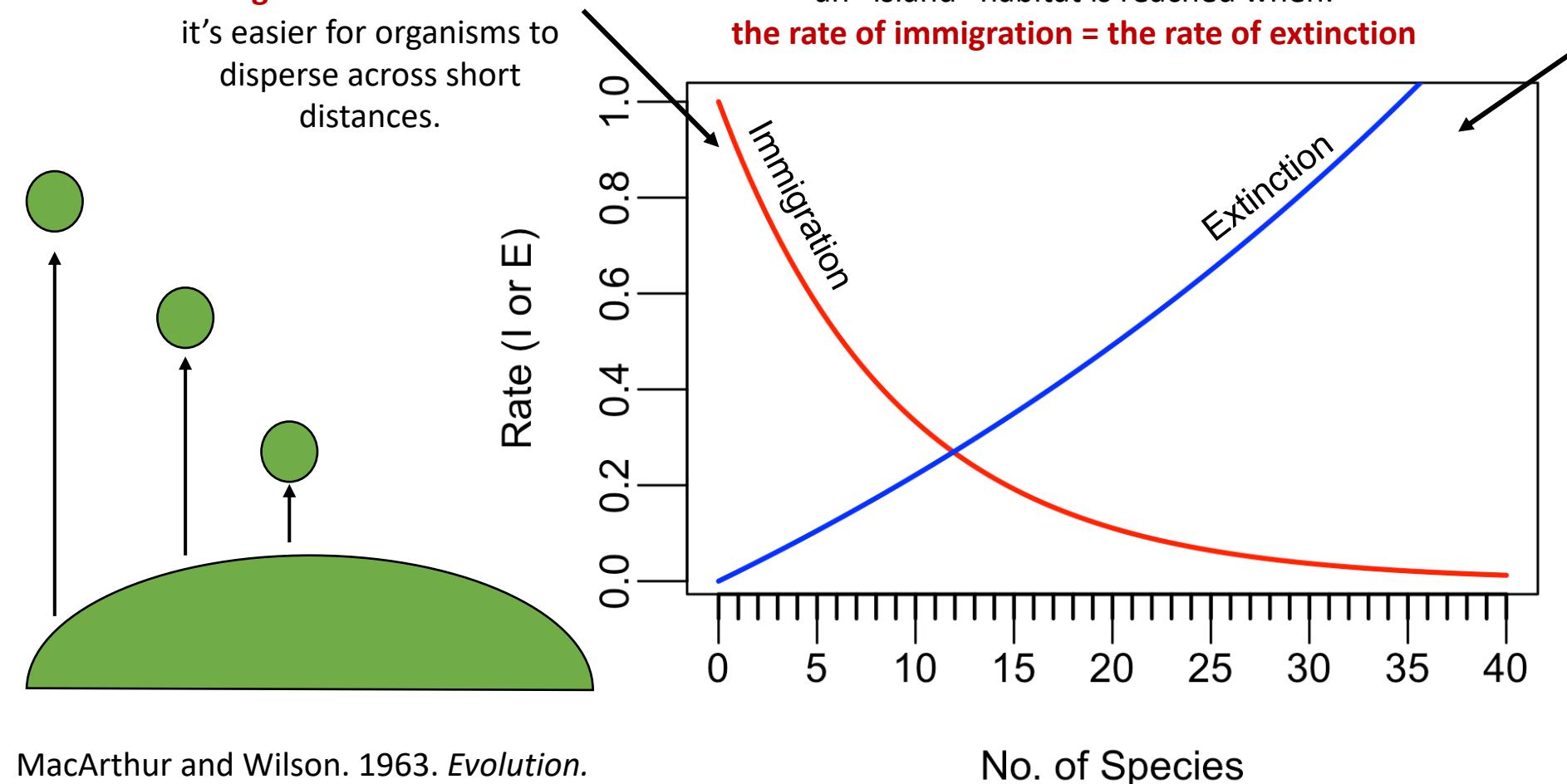
MacArthur and Wilson. 1963. *Evolution*.
MacArthur and Wilson. 1967. *The Theory of Island Biogeography*.

The **theory of island biogeography** offers a null model for the number of species found in a given habitat, predictable from both the **size** of the habitat and its **distance** from a source population.

Distance effect: **Closer islands have higher immigration rates** because it's easier for organisms to disperse across short distances.

The equilibrium number of species in an “island” habitat is reached when:
the rate of immigration = the rate of extinction

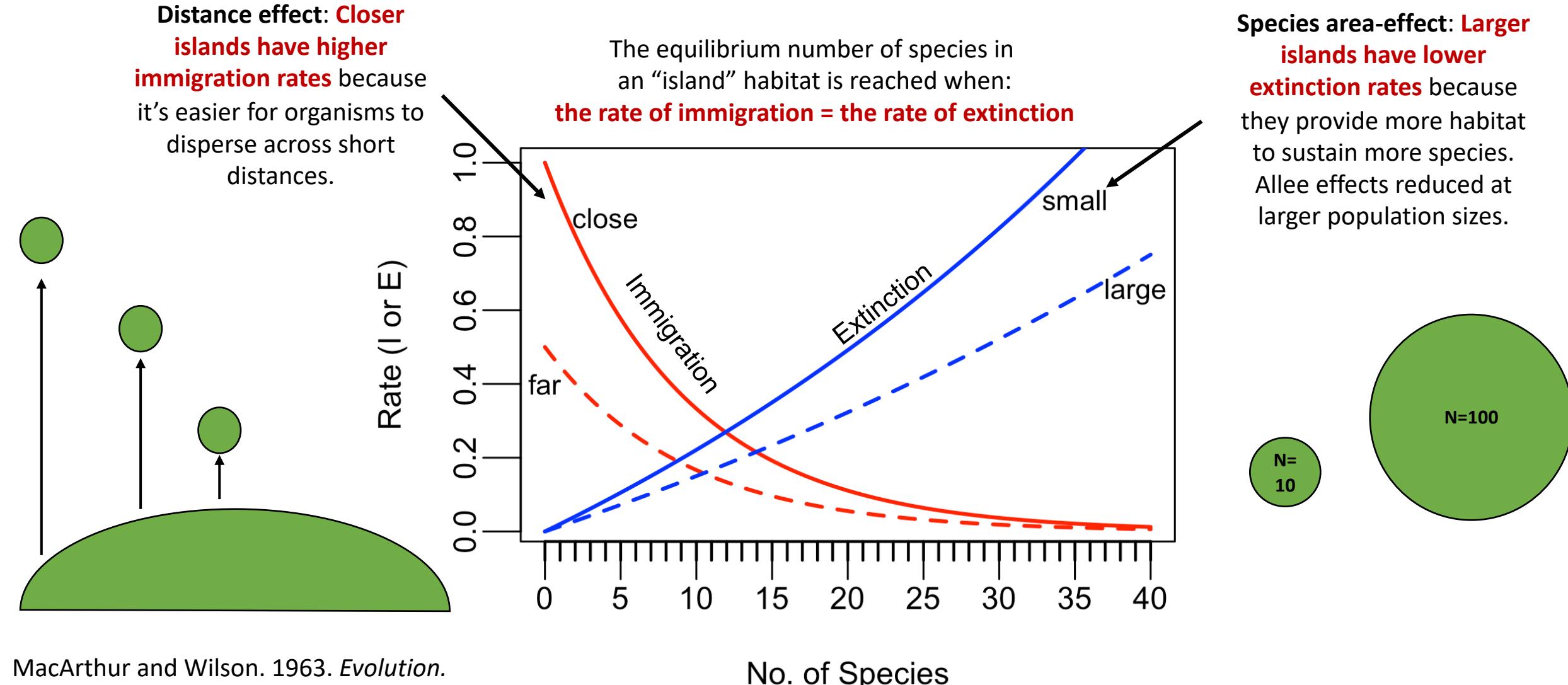
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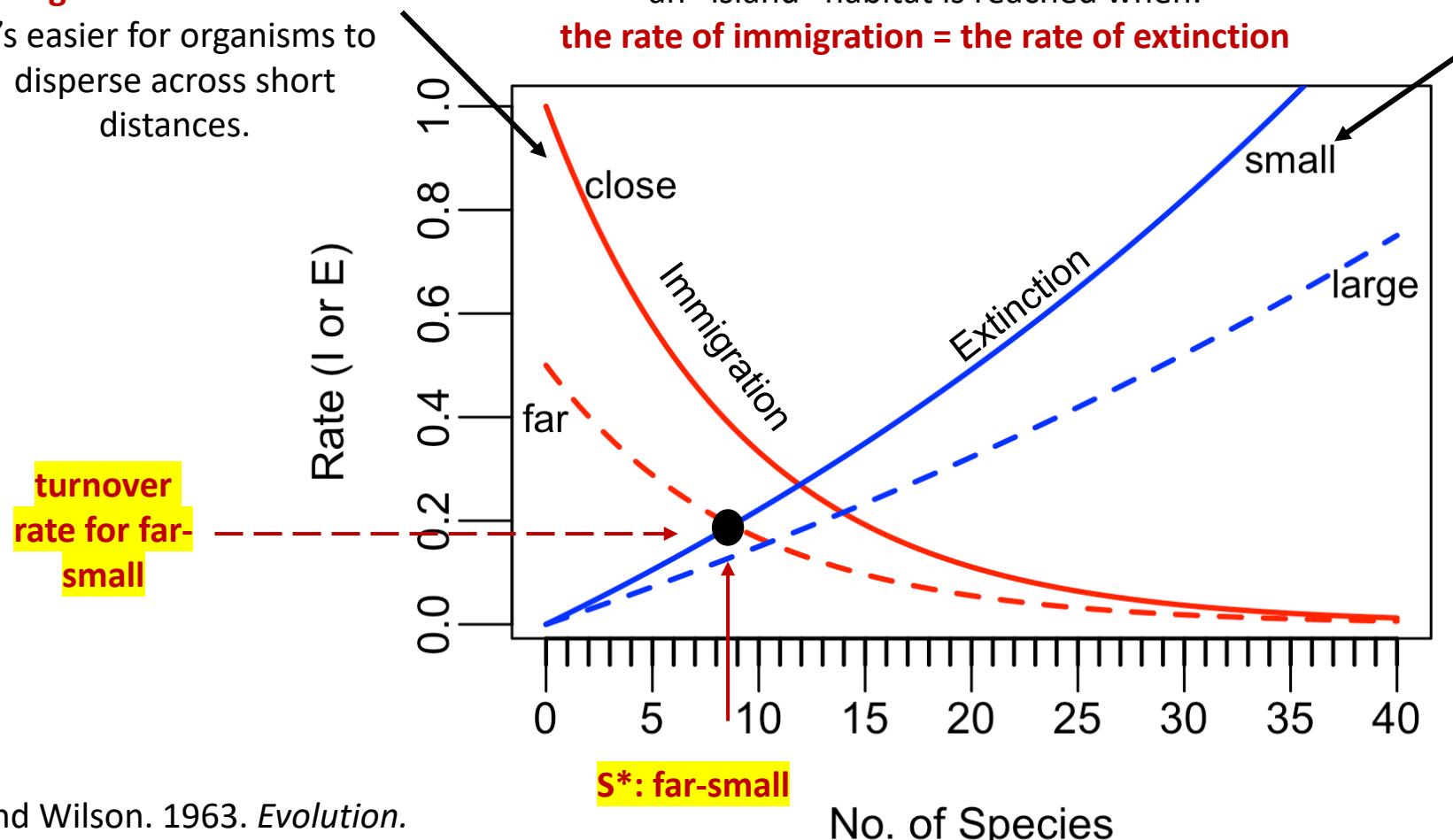


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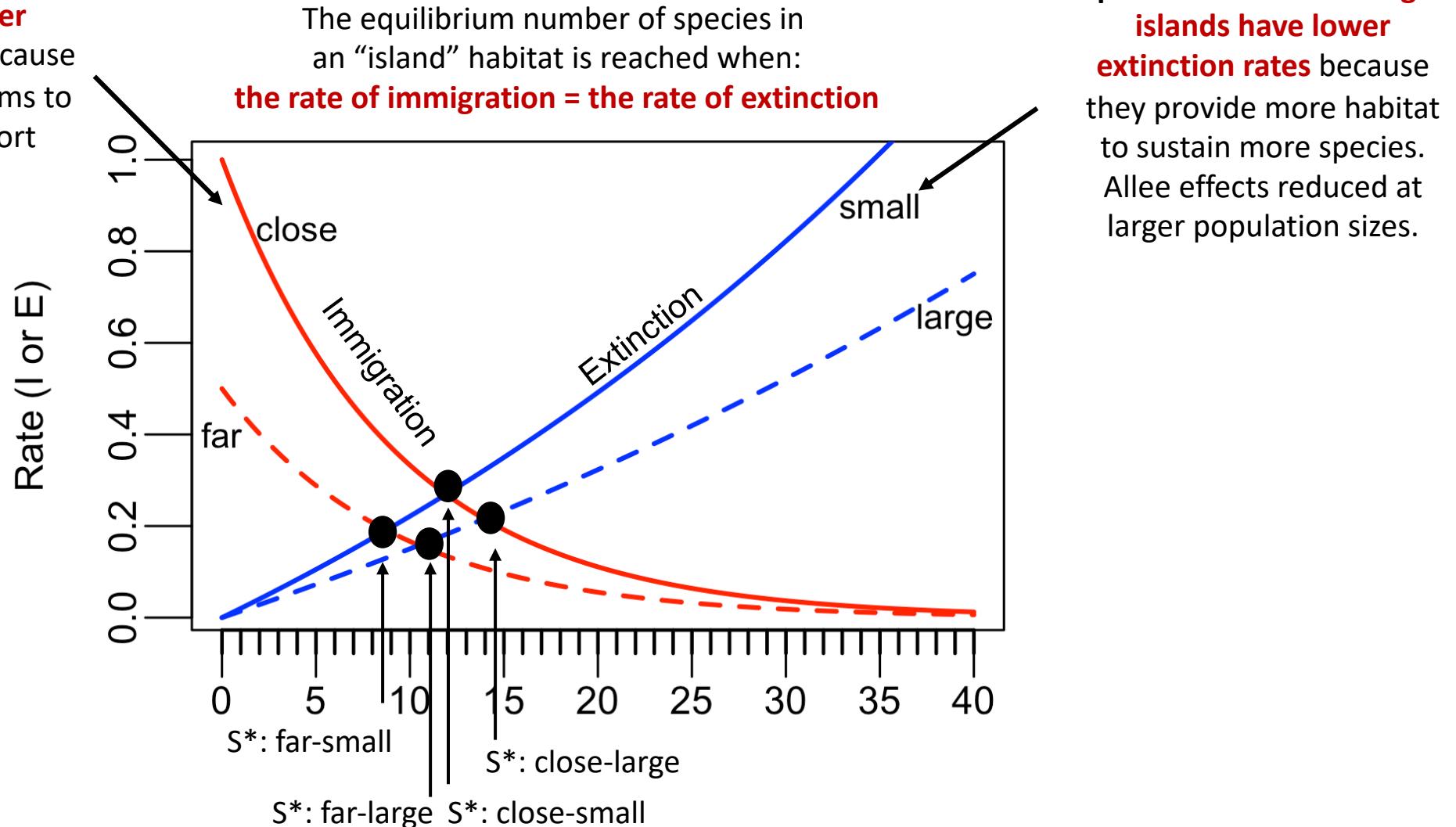
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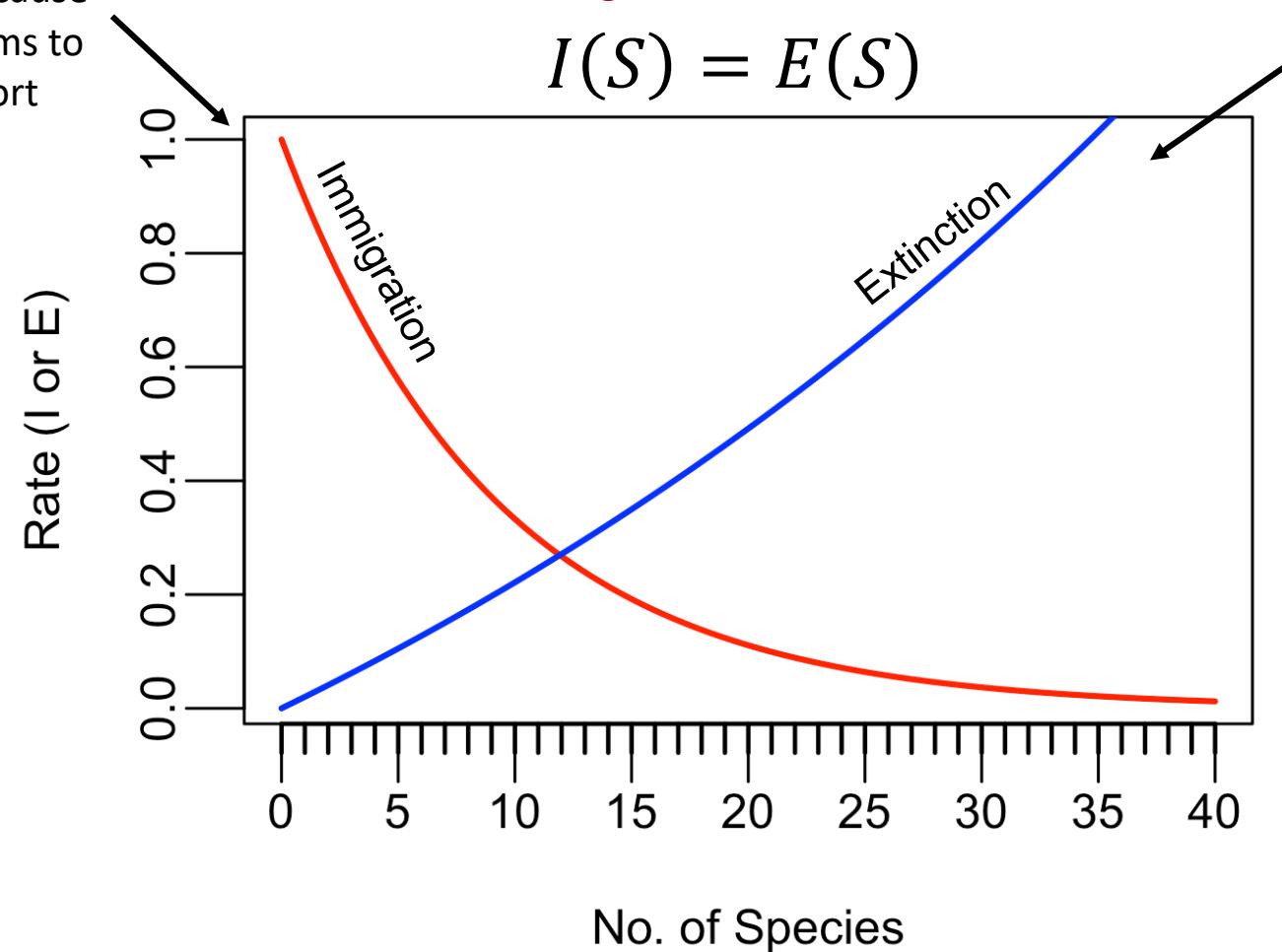


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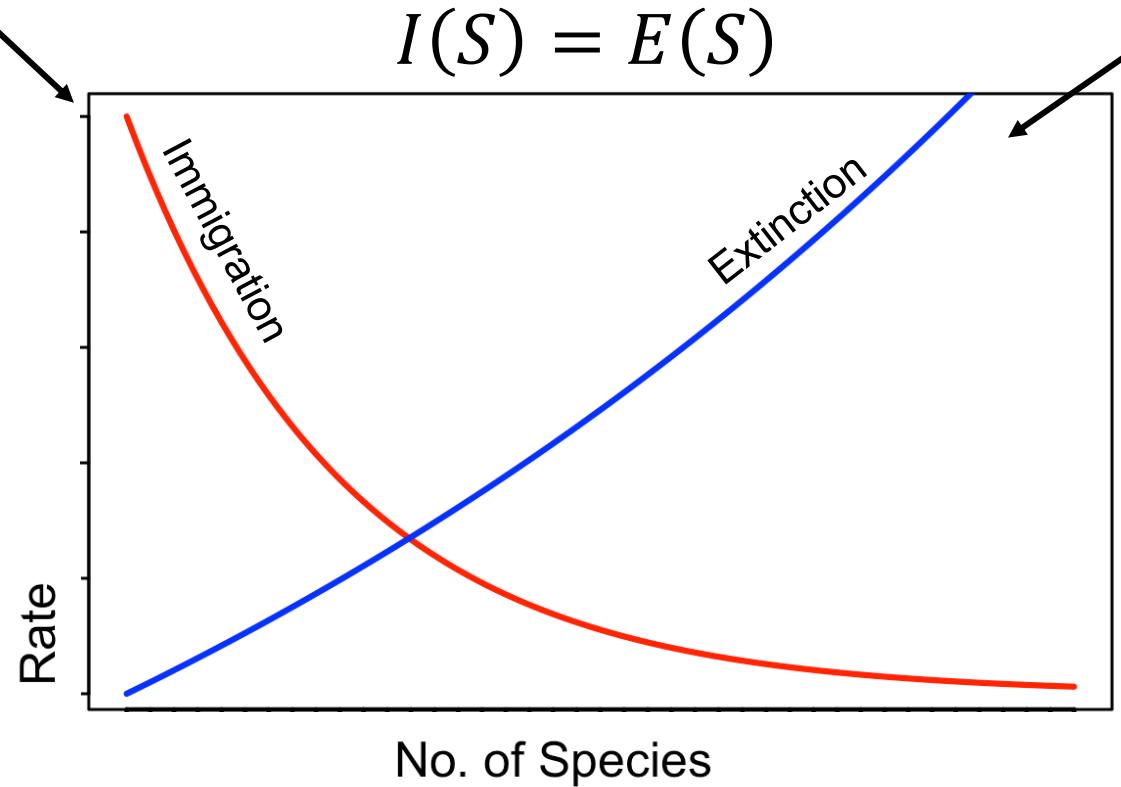
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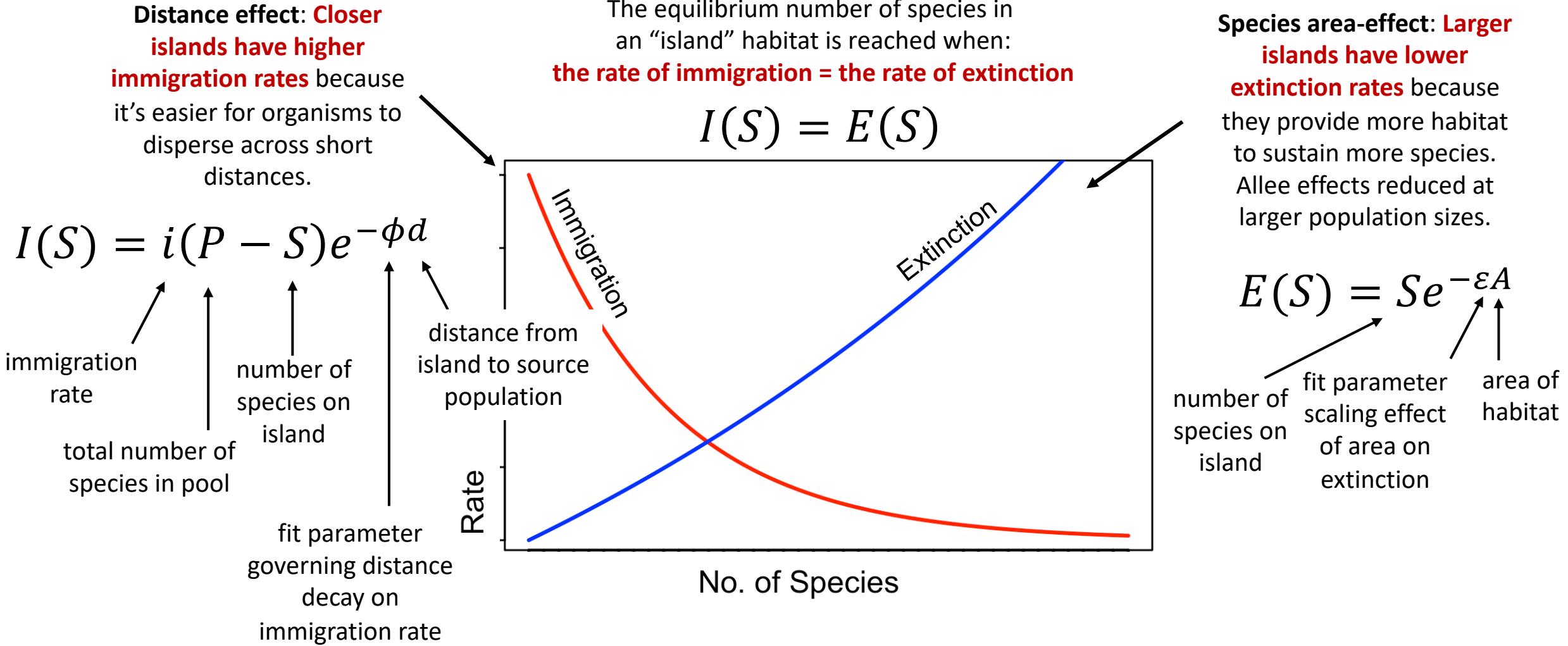


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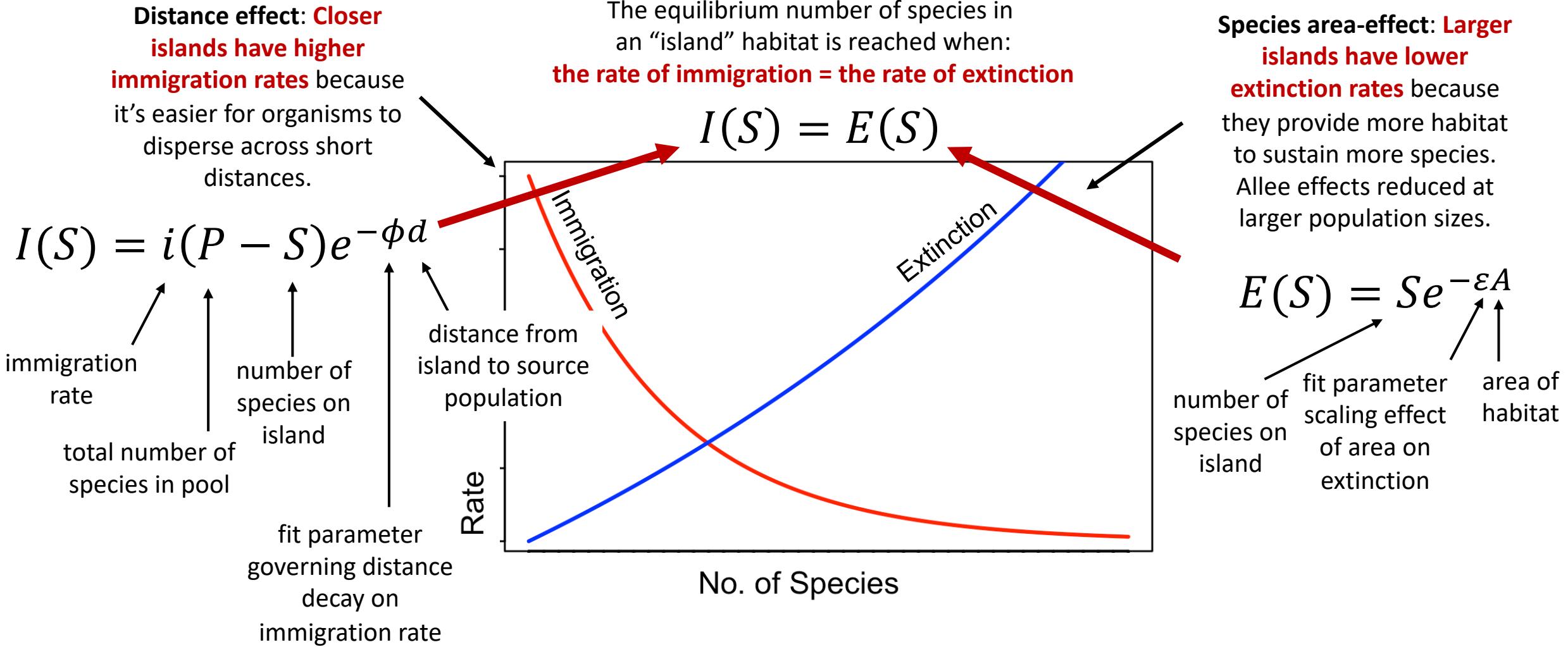
$$E(S) = S e^{-\varepsilon A}$$

number of species on island fit parameter scaling effect of area on extinction area of habitat

The **theory of island biogeography** offers a null model for the number of species found in a given habitat, predictable from both the **size** of the habitat and its **distance** from a source population.



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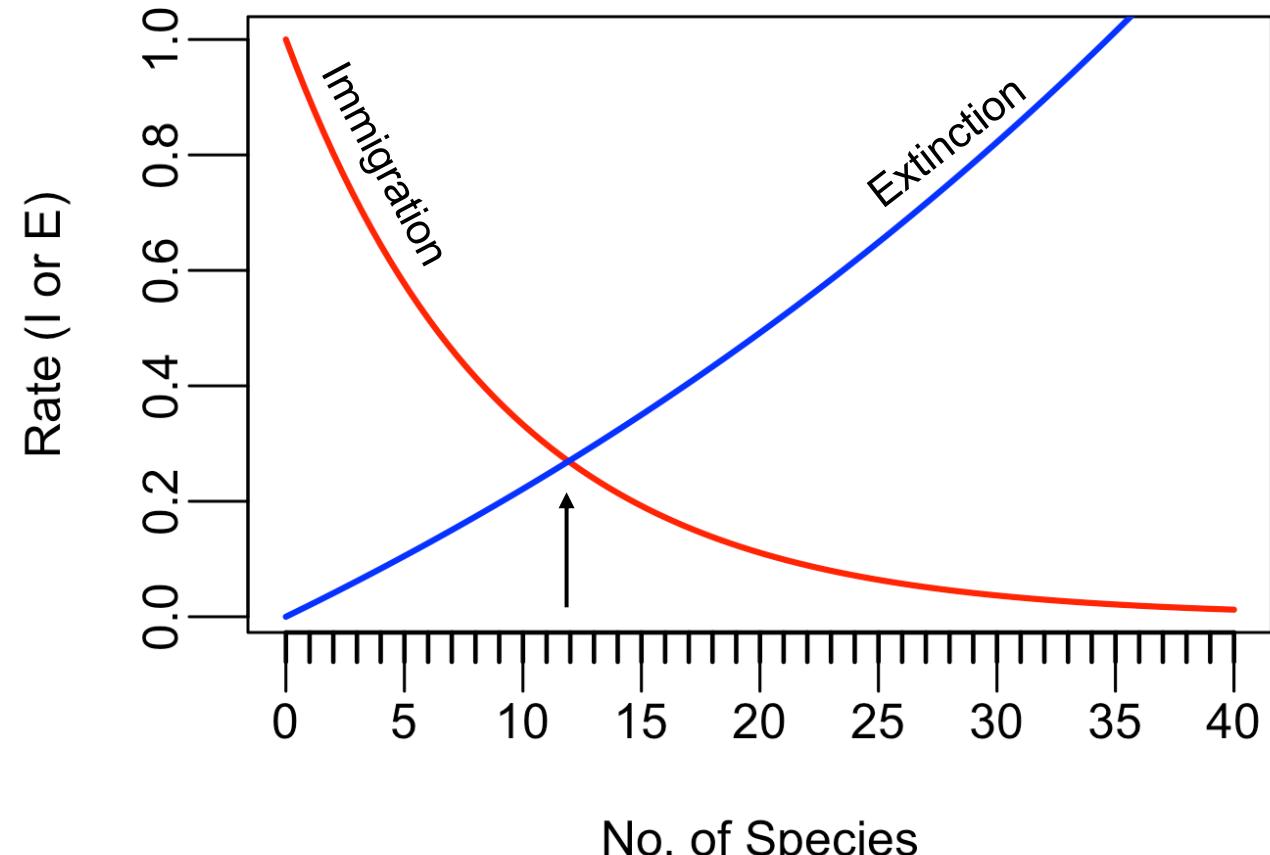
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$$I(S) = E(S)$$

$$S^* = \frac{iPe^{\varepsilon A}}{ie^{\varepsilon A} + e^{\phi d}}$$



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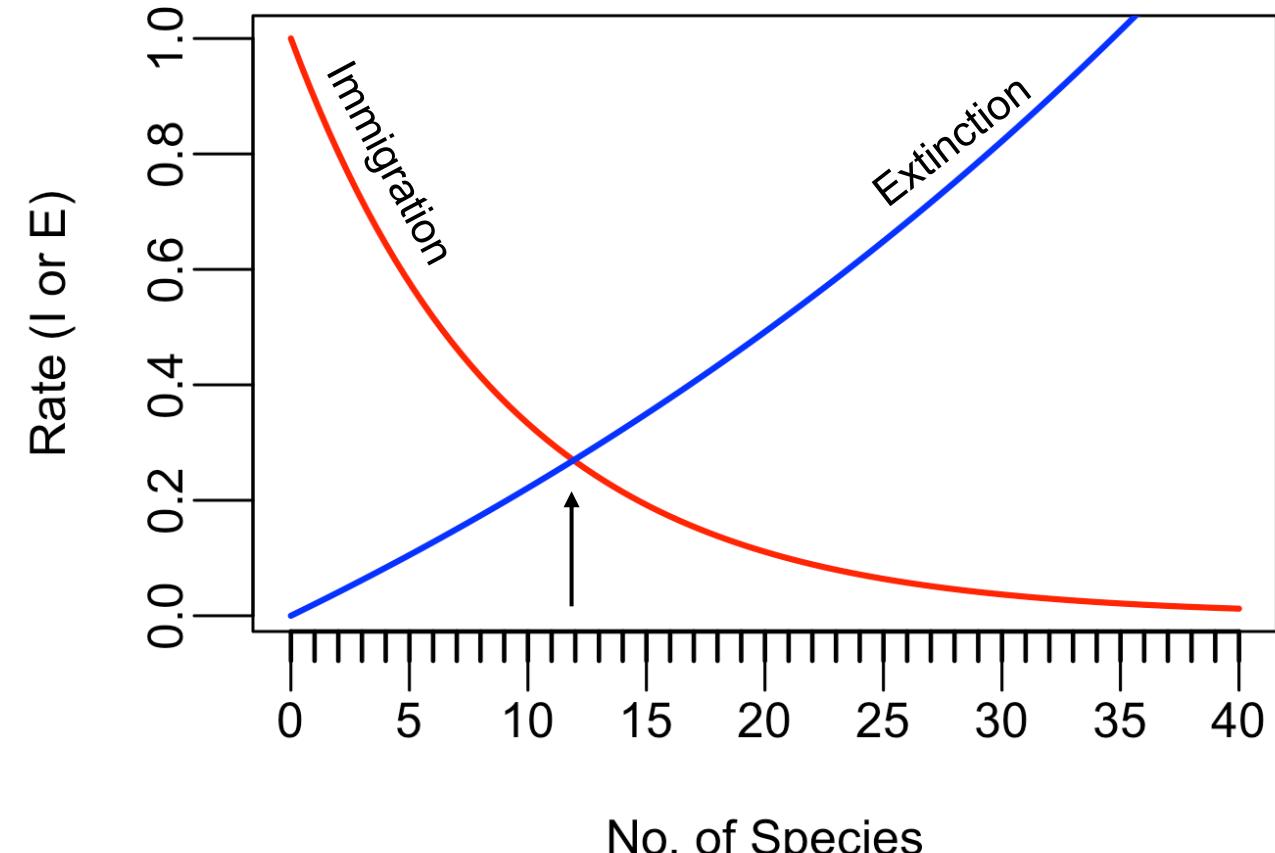
$$S^* = \frac{iPe^{\varepsilon A}}{ie^{\varepsilon A} + e^{\phi d}}$$

↑ equilibrium number of species

increases with number of species in source pool

increases with area of island

decreases with distance from mainland

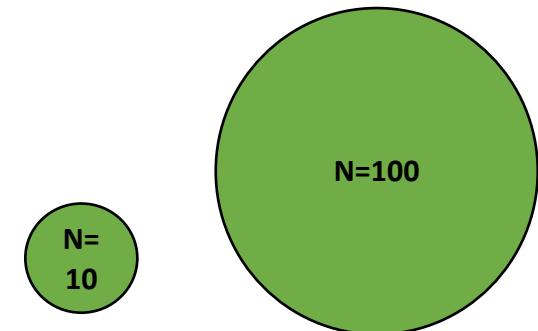
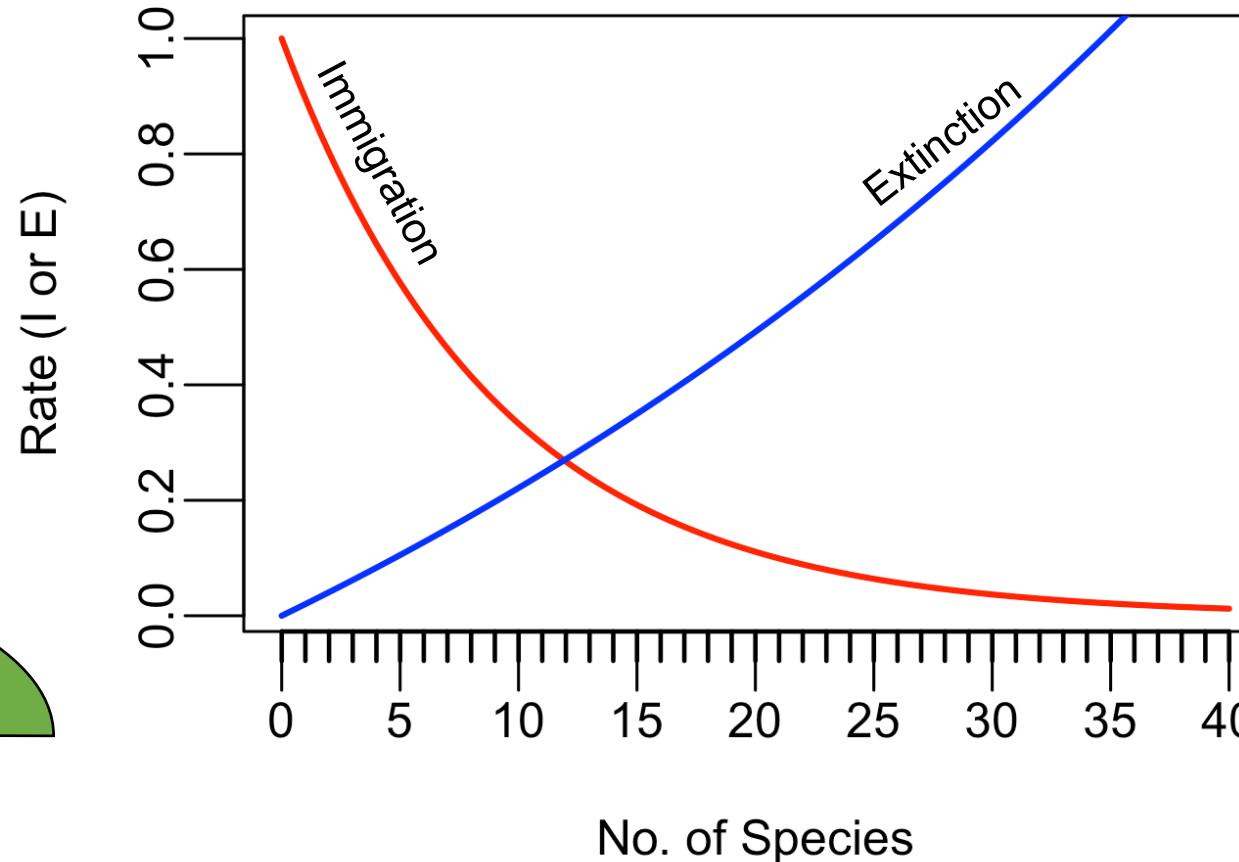
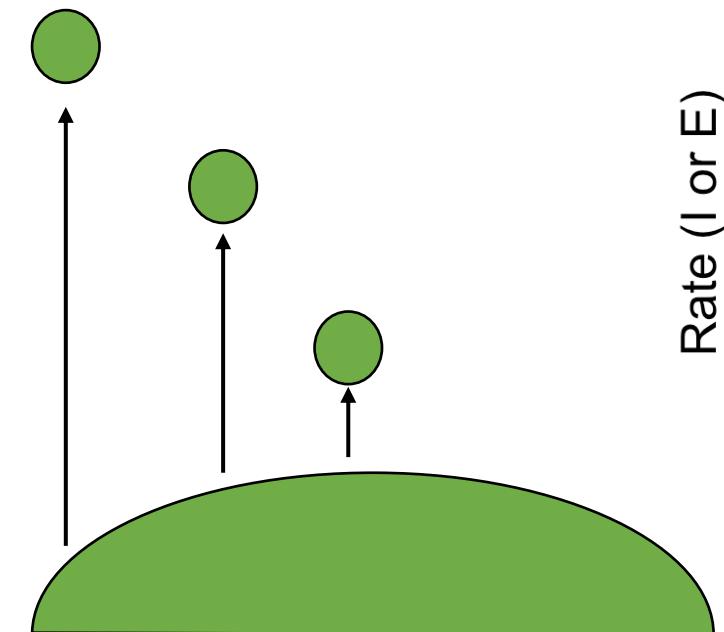


Extensions to the theory of island biogeography...

Distance effect: Closer islands have higher immigration rates because it's easier for organisms to disperse across short distances.

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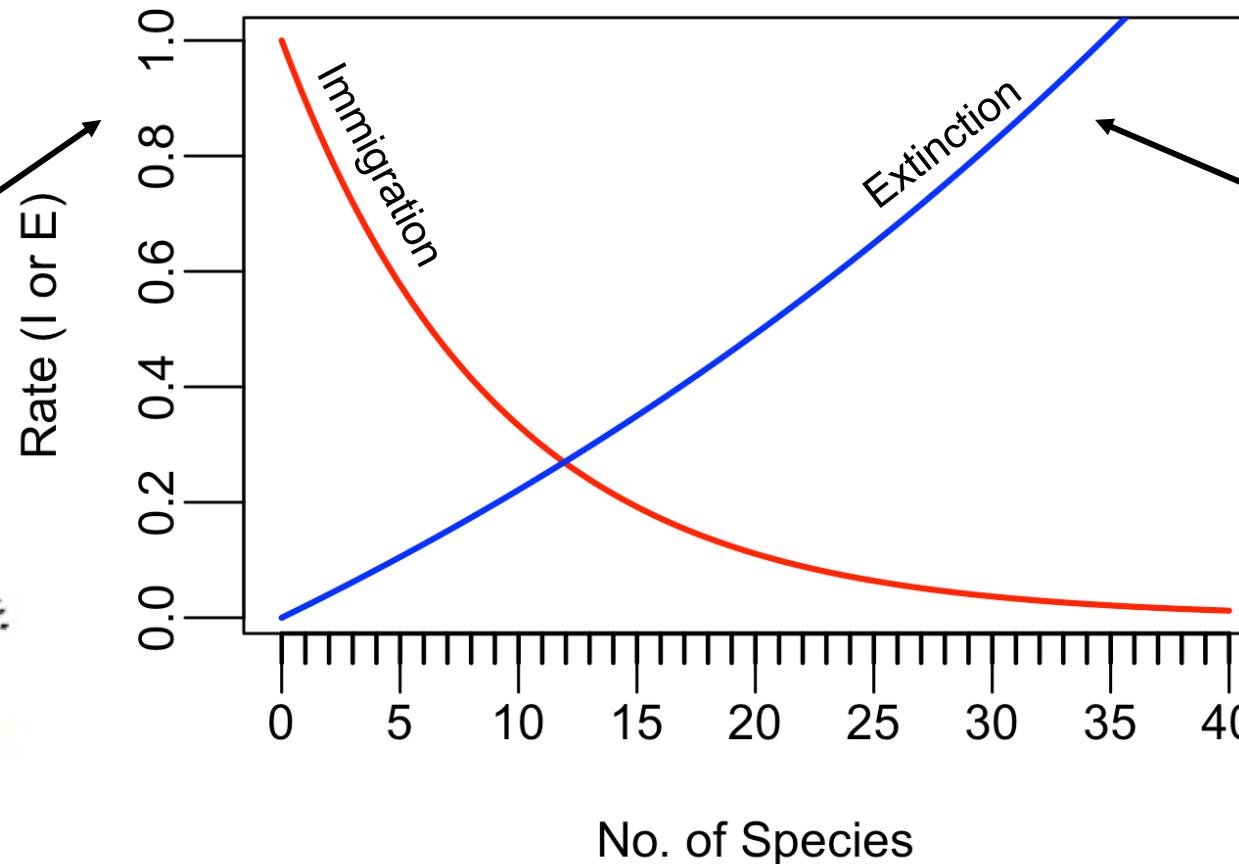
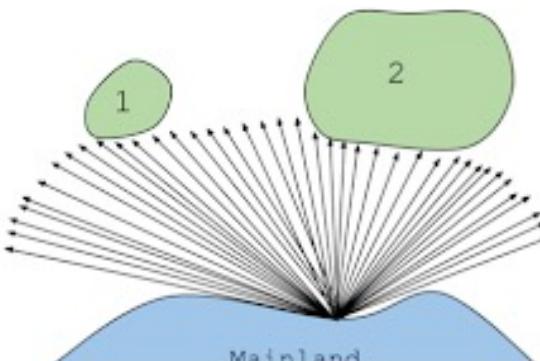
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Extensions to the theory of island biogeography...

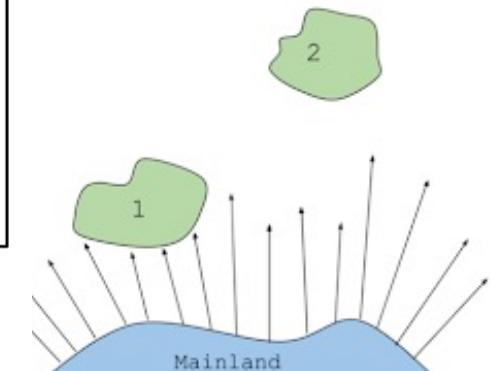
Distance effect: Closer islands have higher immigration rates because it's easier for organisms to disperse across short distances.

Target effect: larger islands have higher immigration rates because they offer a bigger target to land on!

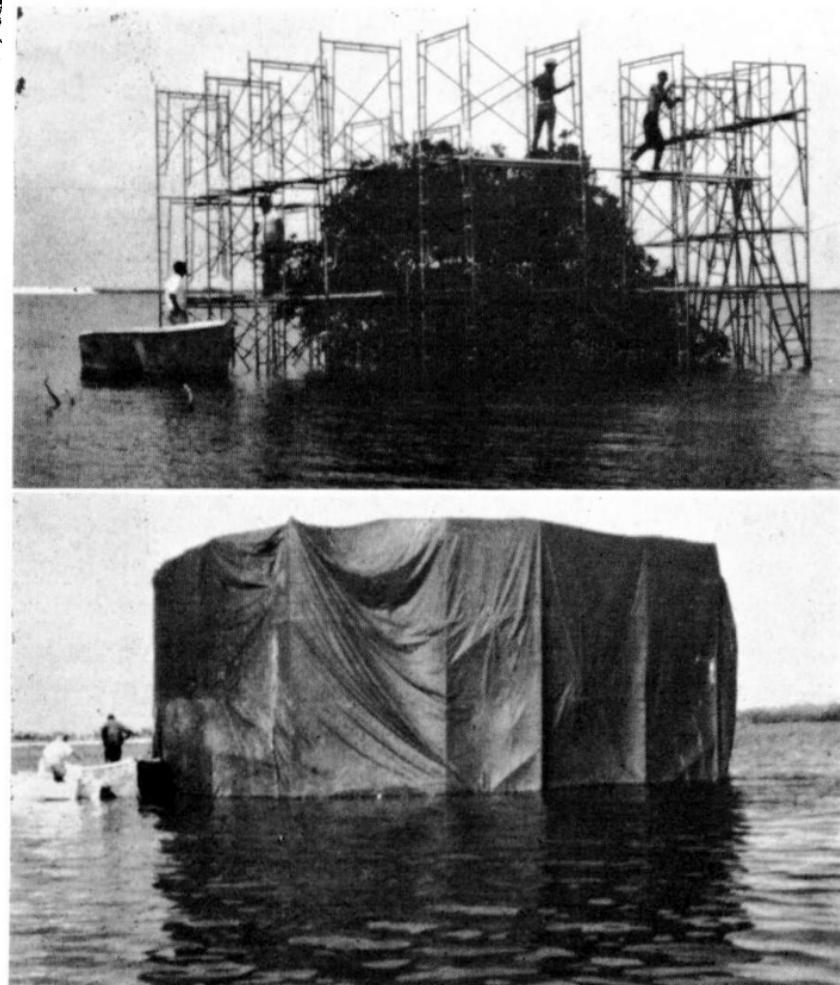
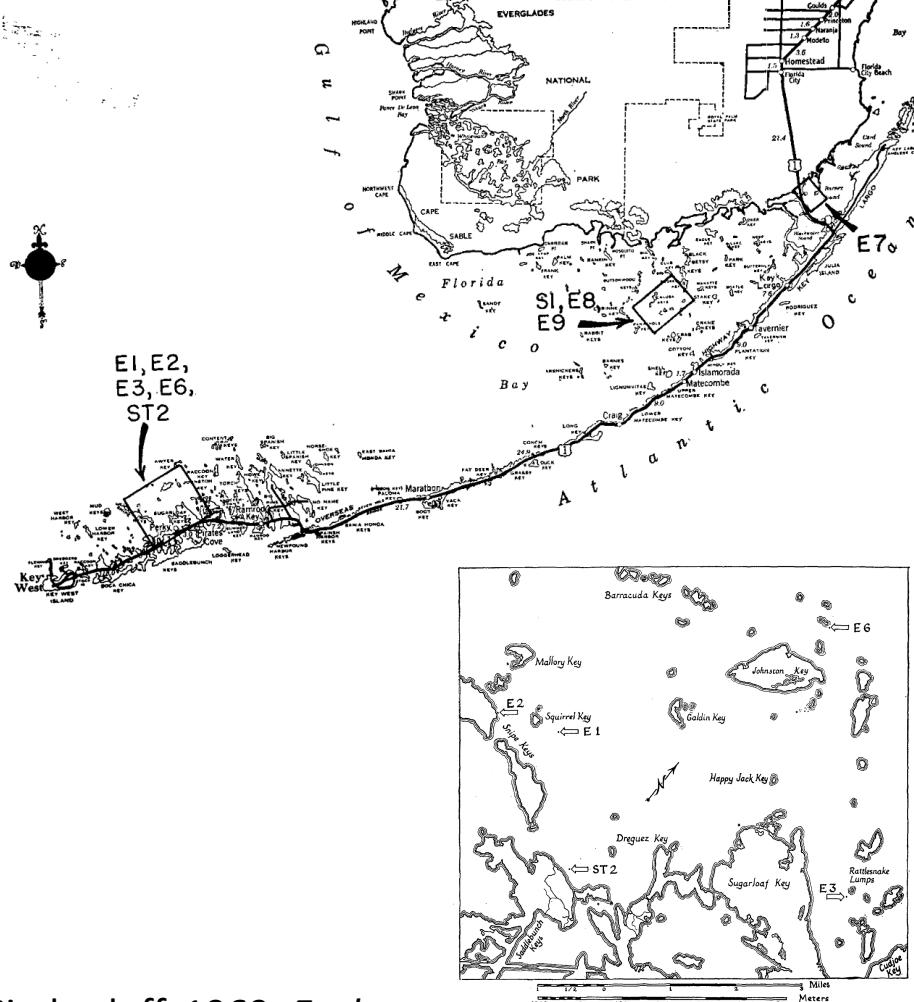


Species area-effect: Larger islands have lower extinction rates because they provide more habitat to sustain more species. Allee effects reduced at larger population sizes.

Rescue effect: Closer islands have lower extinction rates because they can be repopulated from the mainland!



Wilson and Simberloff field-tested this theory in the Florida Keys!



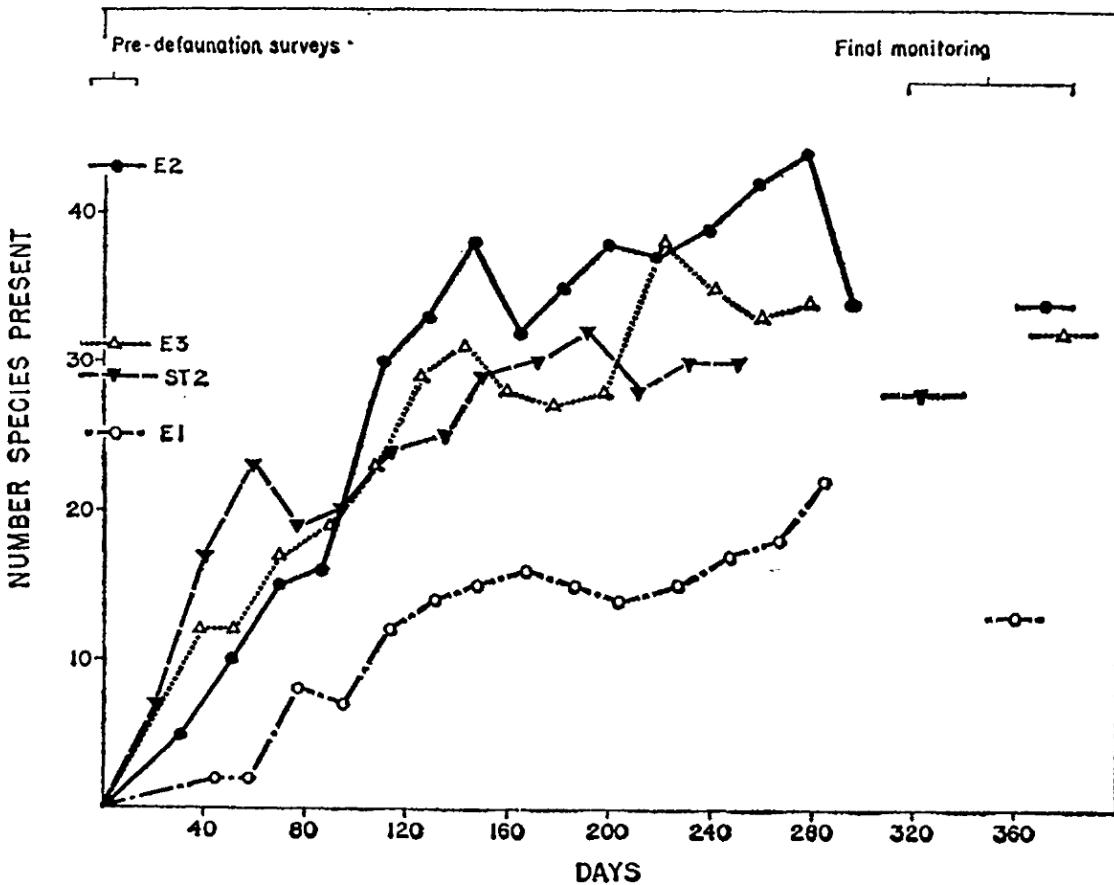
- Wilson and Simberloff identified seven mangrove islands in the Florida Keys varying in size from 11-18m in diameter and catalogued their fauna (all arthropods).
- They also conducted surveys across the keys to quantify the entire possible “source” pool to the islands.
- The experimental islands were then fumigated with methyl bromide at levels lethal to arthropods but not to plants.
- They catalogued their progressive recolonization after fumigation, tracking its predictability based on size and distance of these islands to the source pool.

Simberloff. 1969. *Ecology*.

Wilson and Simberloff. 1969. *Ecology*.

Simberloff and Wilson. 1969. *Ecology*.

Wilson and Simberloff field-tested this theory in the Florida Keys!



- Islands were recolonized to pre-defaunation levels rapidly, within a year!
- Though the equilibrium number of species was the same, the identity was quite different – only about 40% similar to the original censuses.
- Strong flyers recolonized first but were eventually replaced (outcompeted) by better competitors (typically ants) – in keeping with theories of faunal succession.
- The islands farthest from the mainland were the slowest to recolonize.

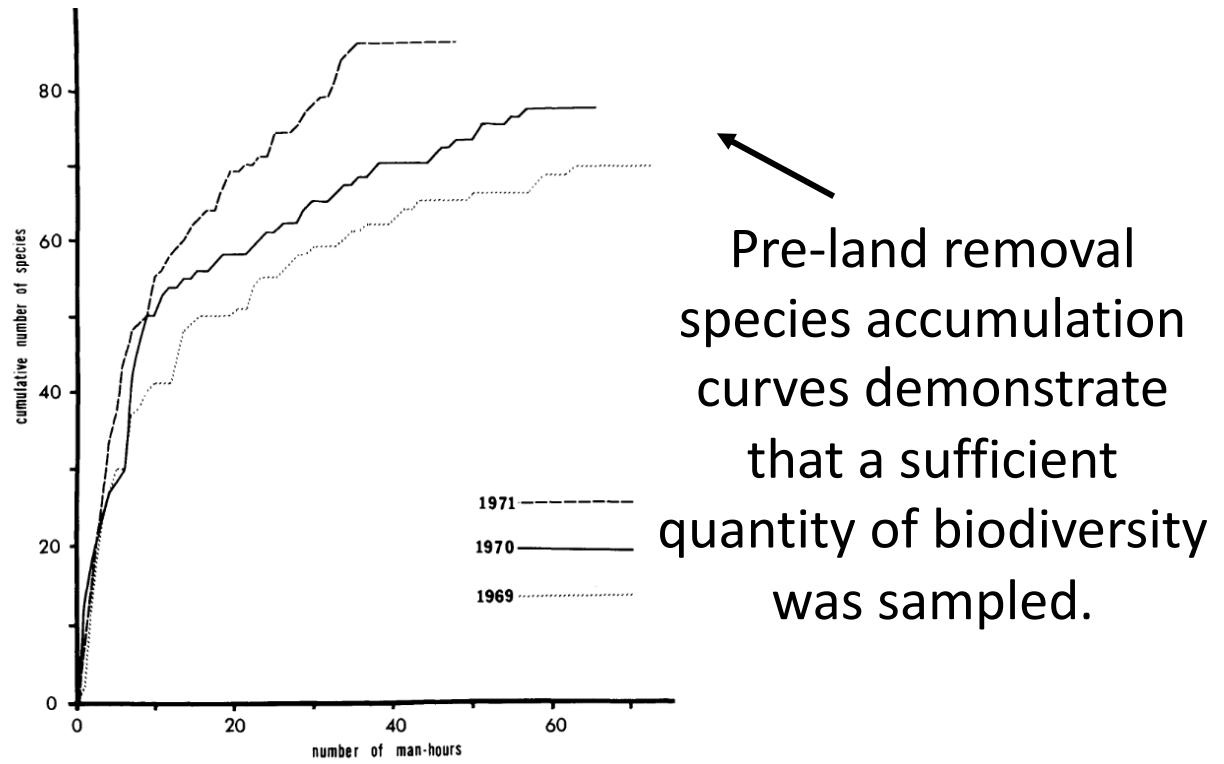


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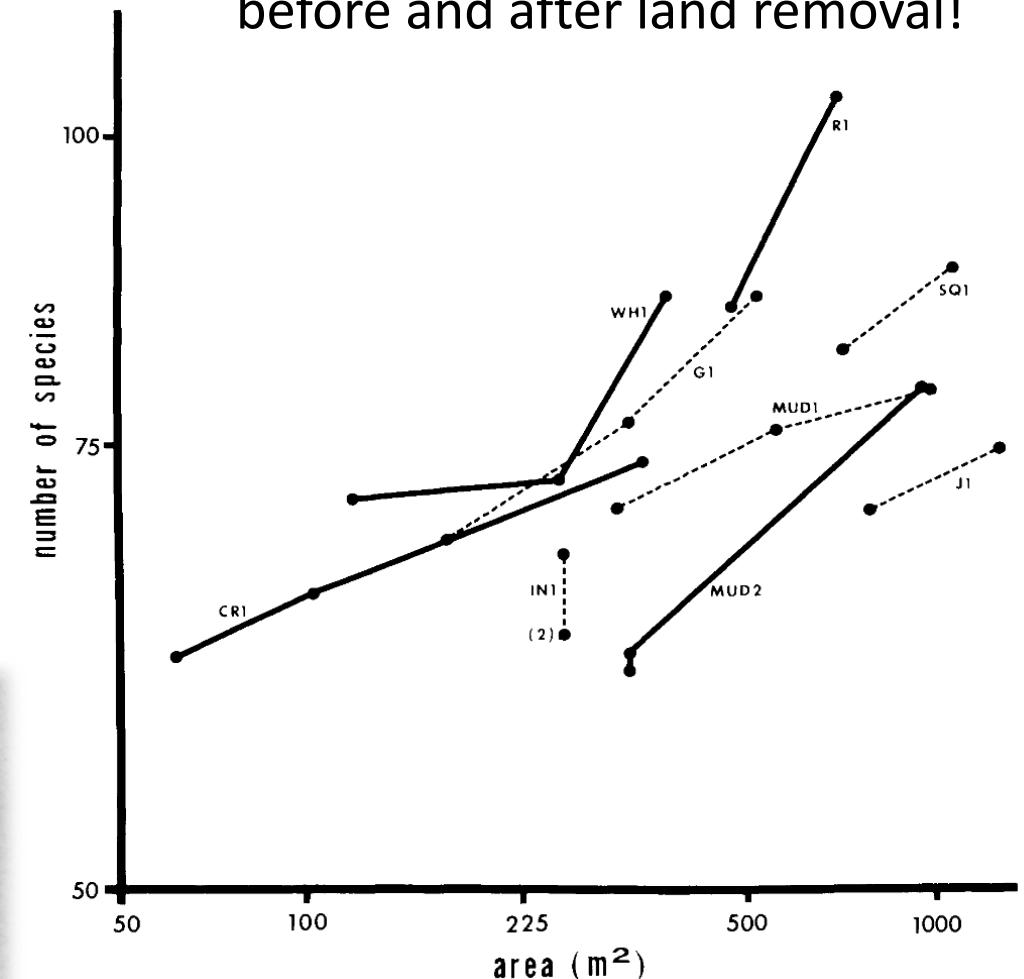
Simberloff and Wilson. 1969. *Ecology*.

In subsequent work, Simberloff demonstrated the area effect by actually removing entire chunks out of islands and censusing species!



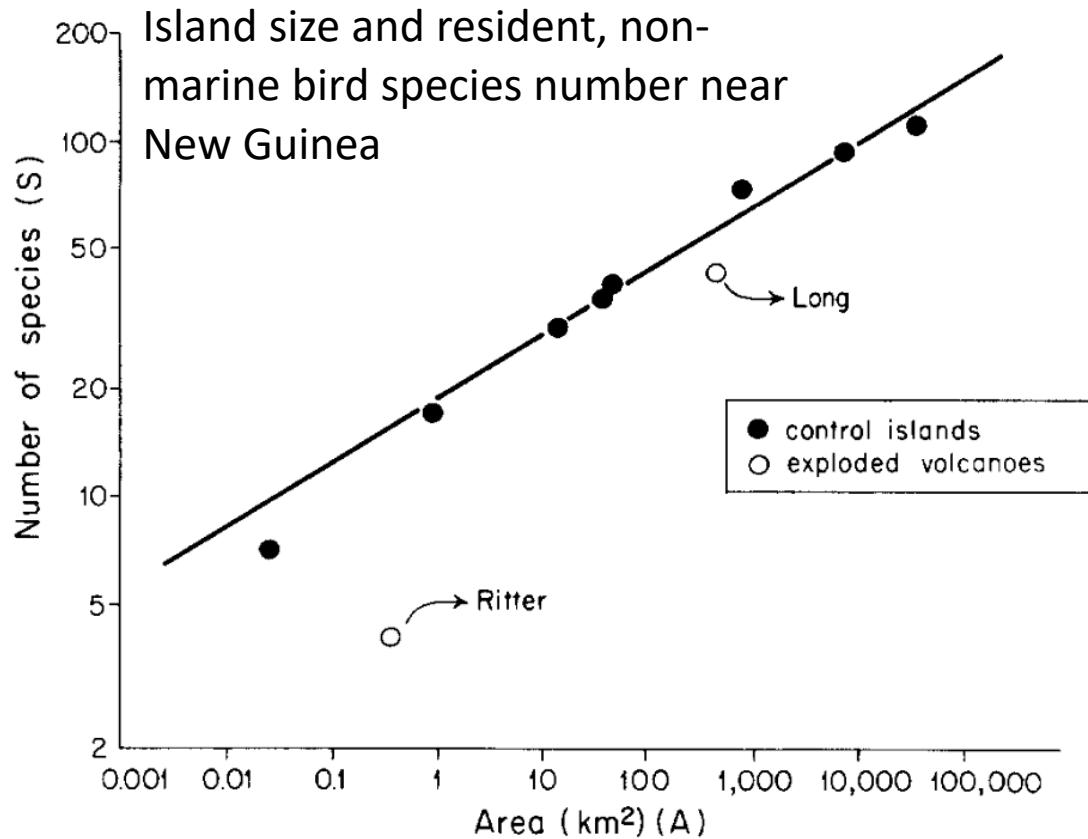
Pre-land removal
species accumulation
curves demonstrate
that a sufficient
quantity of biodiversity
was sampled.

log-log plot of island area and species count
before and after land removal!

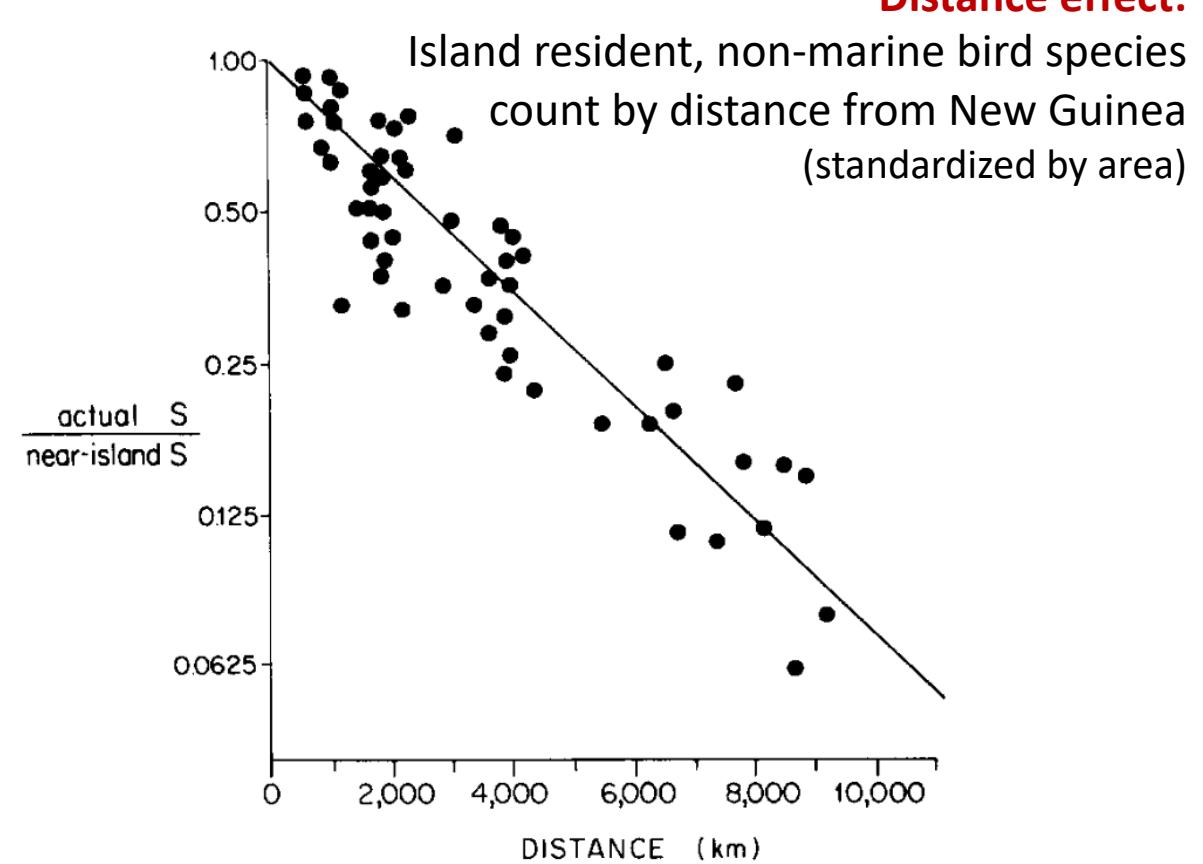


Island biogeography has greatly influenced the design of protected area reserves

Area effect:

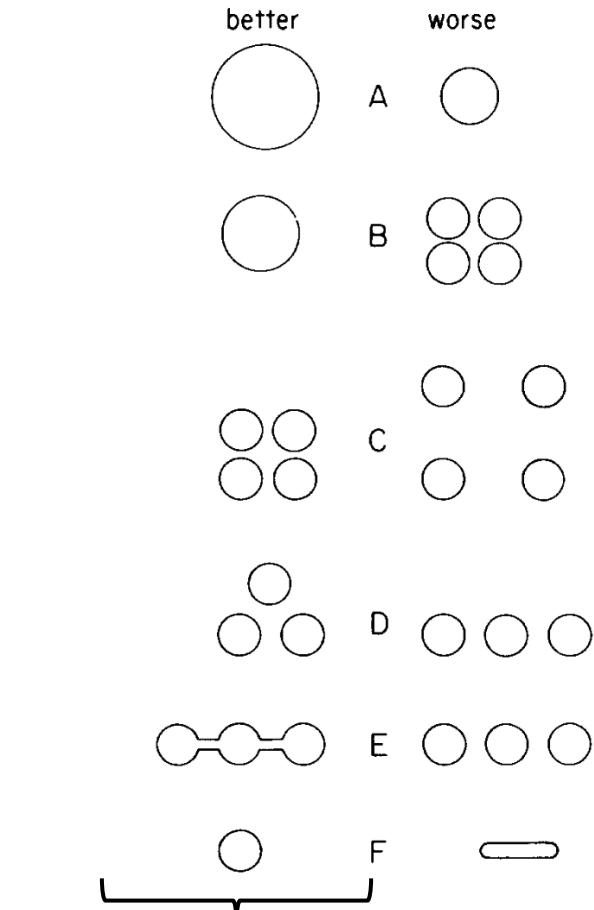
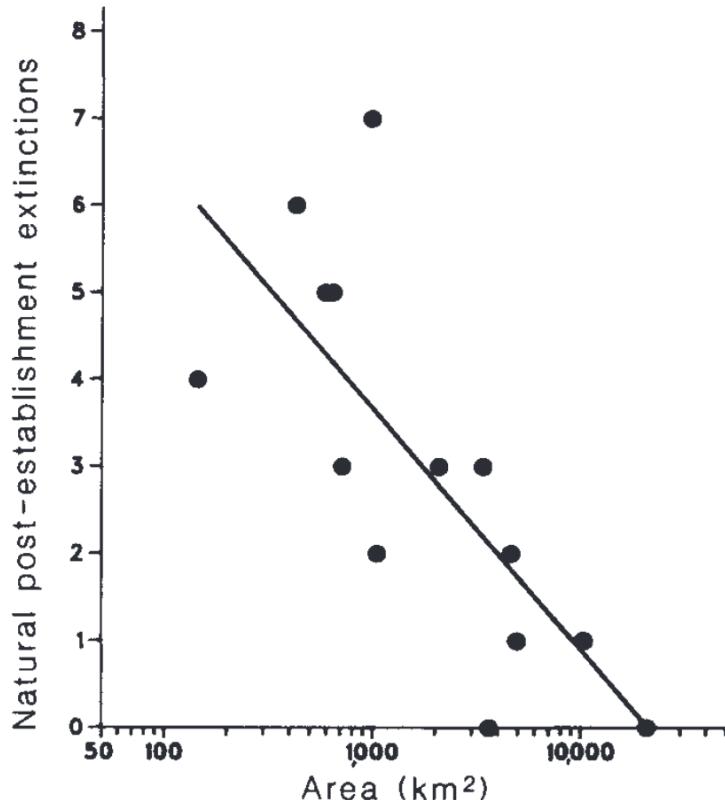


Distance effect:

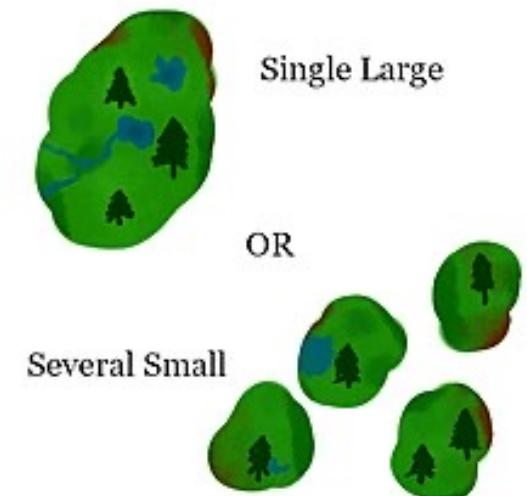


Island biogeography has greatly influenced the design of protected area reserves

Mammalian extirpations post-establishment by area size in western North American national parks



Reserve design estimated to protect the largest number of species based on island biogeography theory

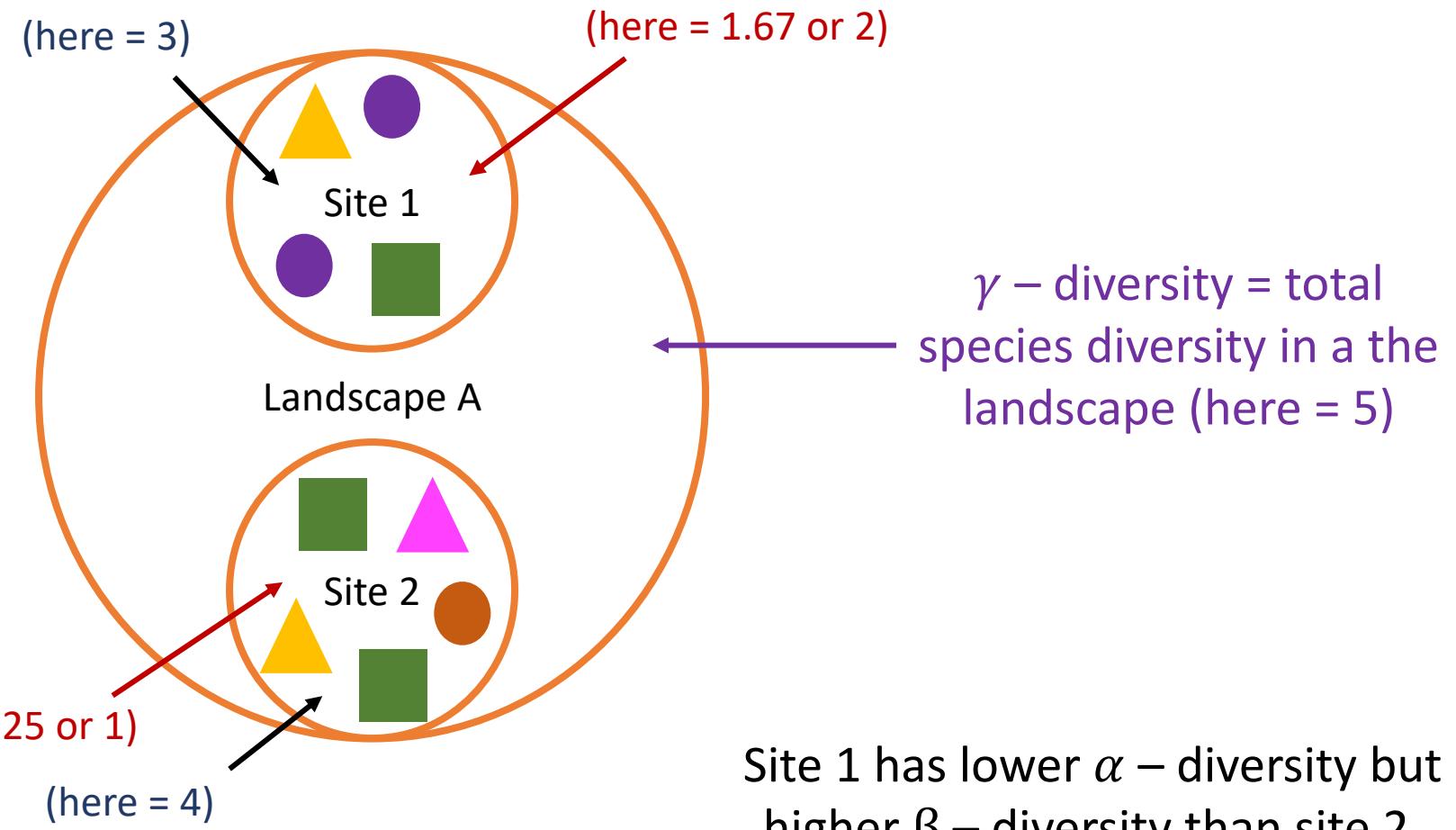


“SLOSS” debate
(1970s-1980s)

Biodiversity can be measured in several ways!

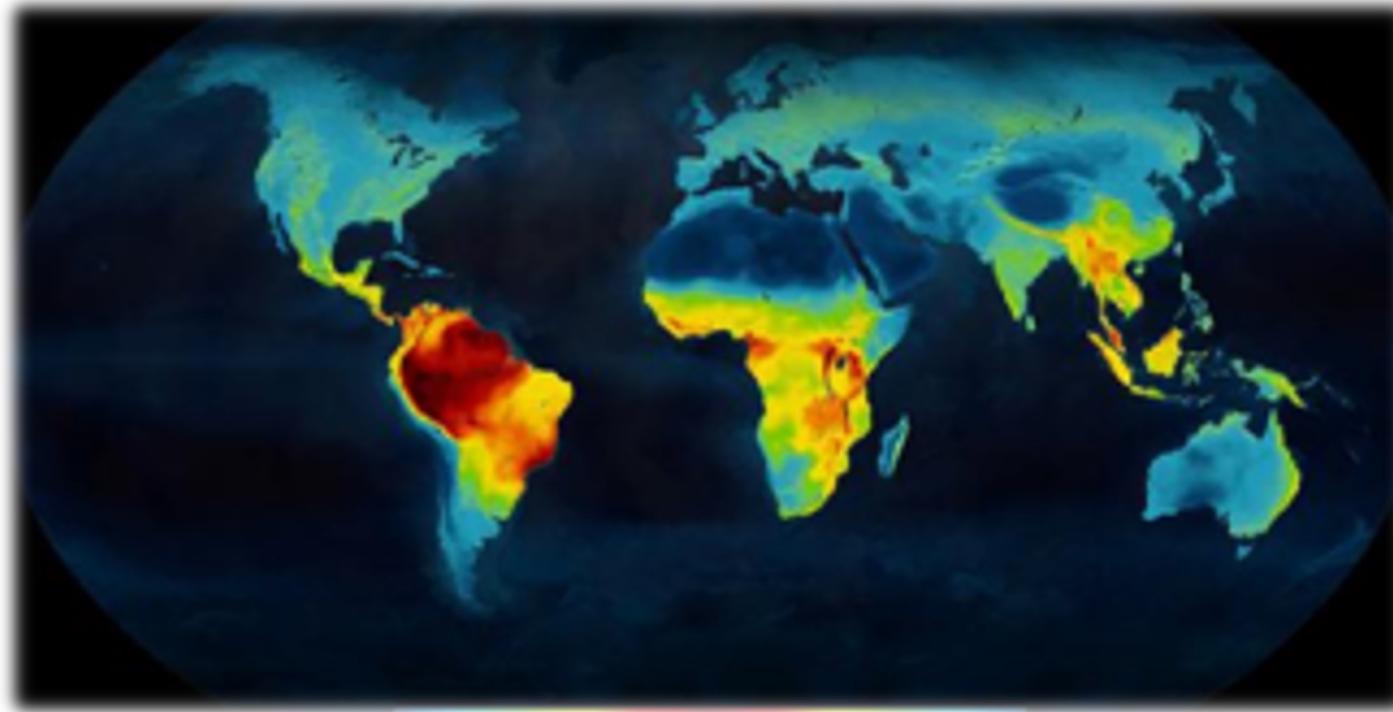
α – diversity = species richness, typically within a small specified region

β – diversity = ratio between landscape and local species diversity, either $(\frac{\gamma}{\alpha})$ or $(\gamma - \alpha)$



Site 1 has lower α – diversity but higher β – diversity than site 2. Both are important!

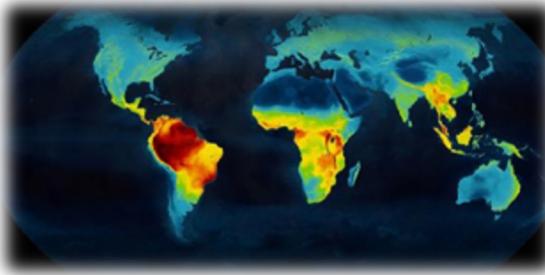
Biodiversity is concentrated in the tropics.



We still lack a satisfying model to explain why.

(terrestrial vertebrate diversity)

Mannion. 2014. *Trends in Ecology & Evolution*.



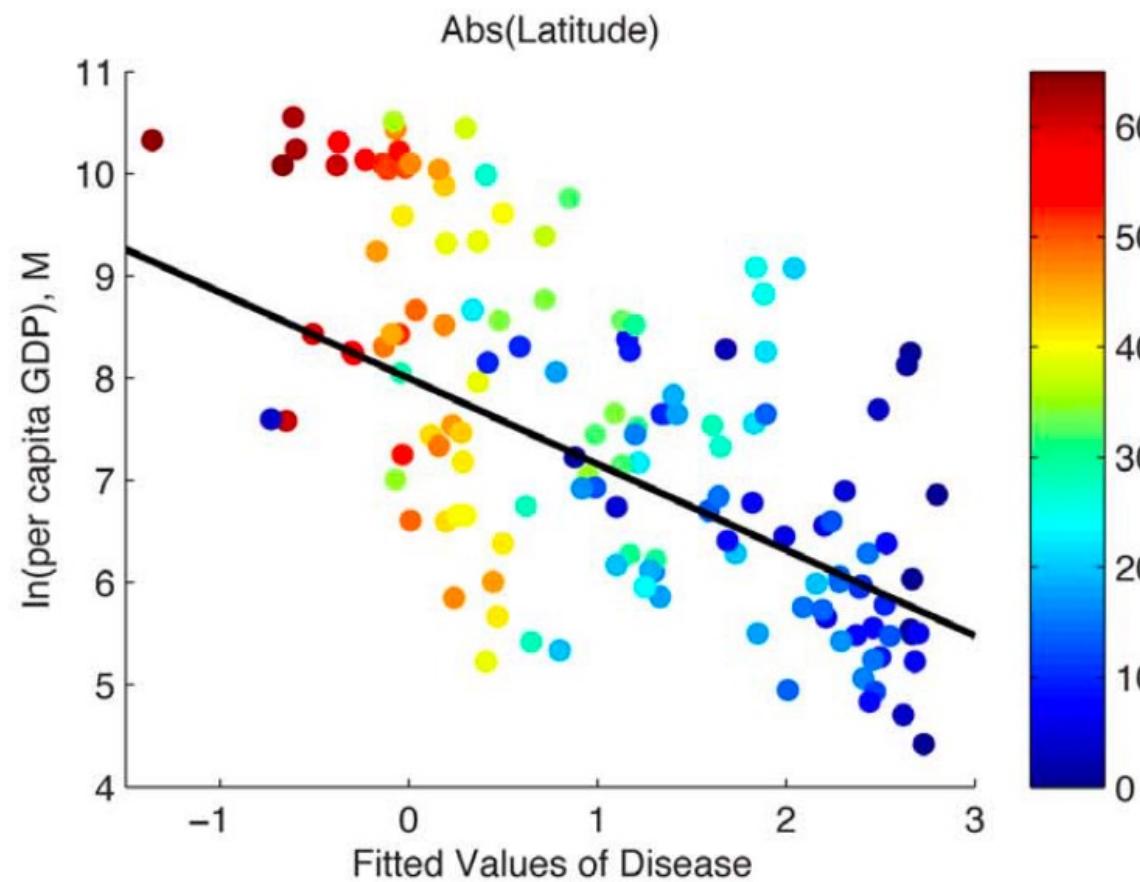
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Some candidate hypotheses:

1. **Geographical area hypothesis.** More area in the tropics = more species... *but there's just as much area north of the tropics and fewer species!*
2. **Species-energy hypothesis.** Increased solar energy at low latitudes causes increased net primary productivity (or photosynthesis) and drives accumulation of species up the food web...*but offers a better prediction for abundance and biomass than for numbers of species.*
3. **Historical perturbation hypothesis.** Polar regions have not yet recovered equilibrium species numbers after glaciation... *but does not hold for marine systems, where the latitudinal gradient still exists...*
4. **Biotic interactions hypothesis.** More species yield more species as processes of competition, predation, etc. are intensified in the tropics... *but cannot provide the basal cause for the accumulation of more species to begin with!*

Vector-borne and parasitic diseases are also **concentrated in the tropics** - where income is correspondingly low.



Why do we care about biodiversity?

