

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!

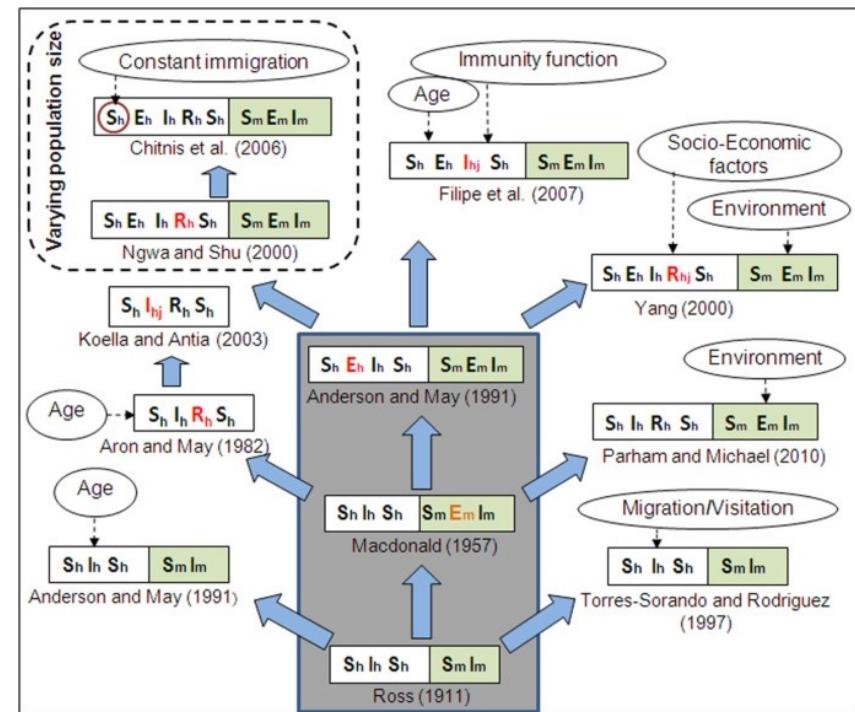
Learning objectives from Lecture 7

You should be able to:

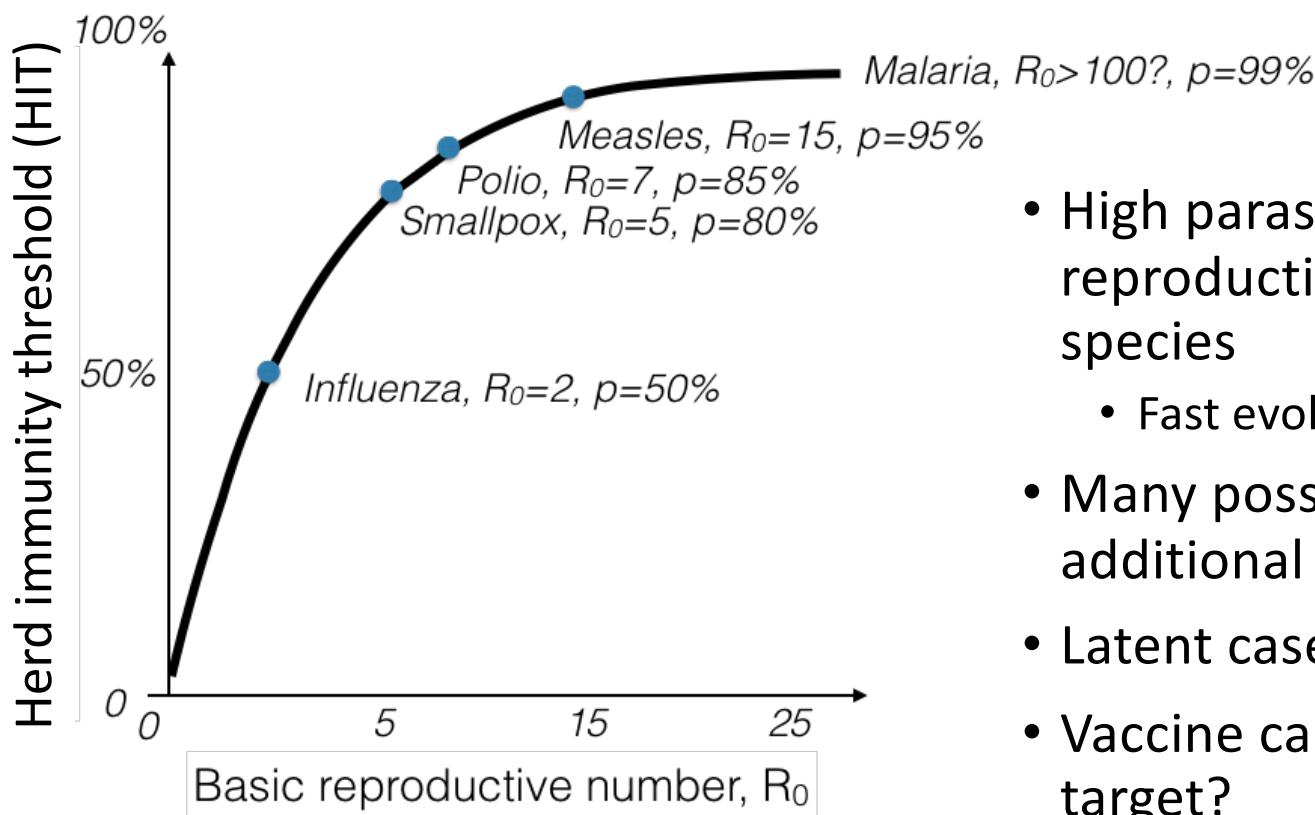
- Understand the relationship between herd immunity and R_0
- Understand the direction of time in a disease epidemic from a phase plane graph (from lab)
- Know the two global vaccination success stories and be able to list some of the challenges to vaccination in other cases
- Know the term ‘critical 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.



Challenges to malaria elimination



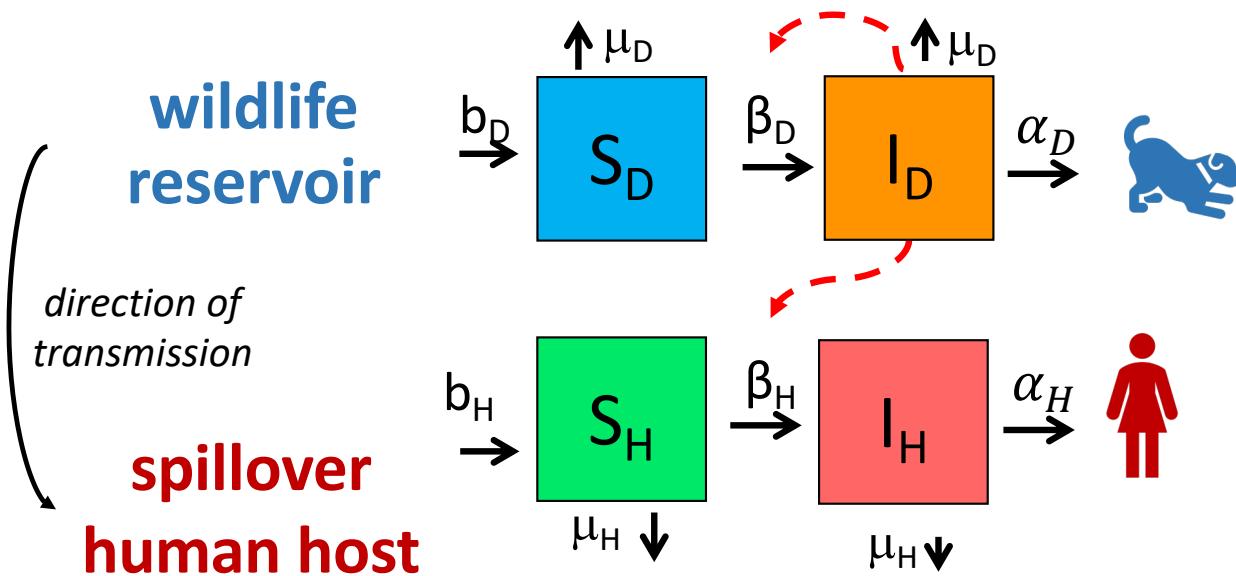
- High parasite diversity: sexual reproduction in 4+ *Plasmodium* species
 - Fast evolution of resistance (e.g. to drugs)
- Many possible vectors! Potentially additional possible reservoirs!
- Latent cases as burden is reduced
- Vaccine candidates: what life stage to target?

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*)

Plague is BOTH vector-borne and zoonotic!

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

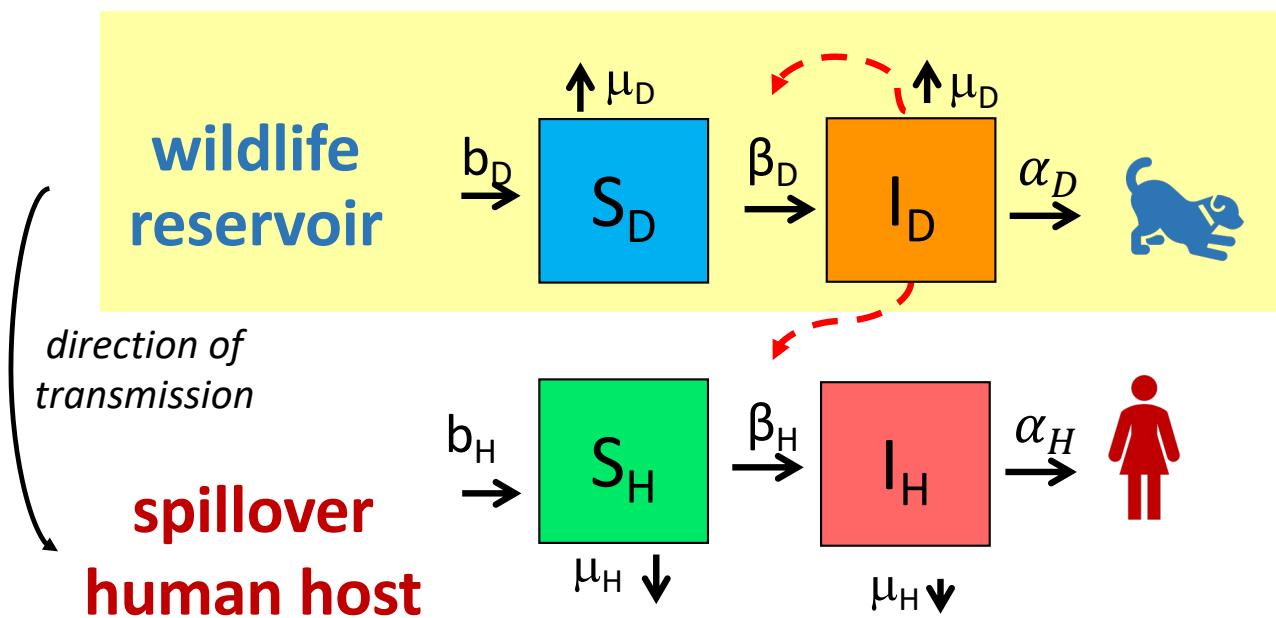


Typically modeled with the reservoir as a distinct state variable!

ex: rabies

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

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



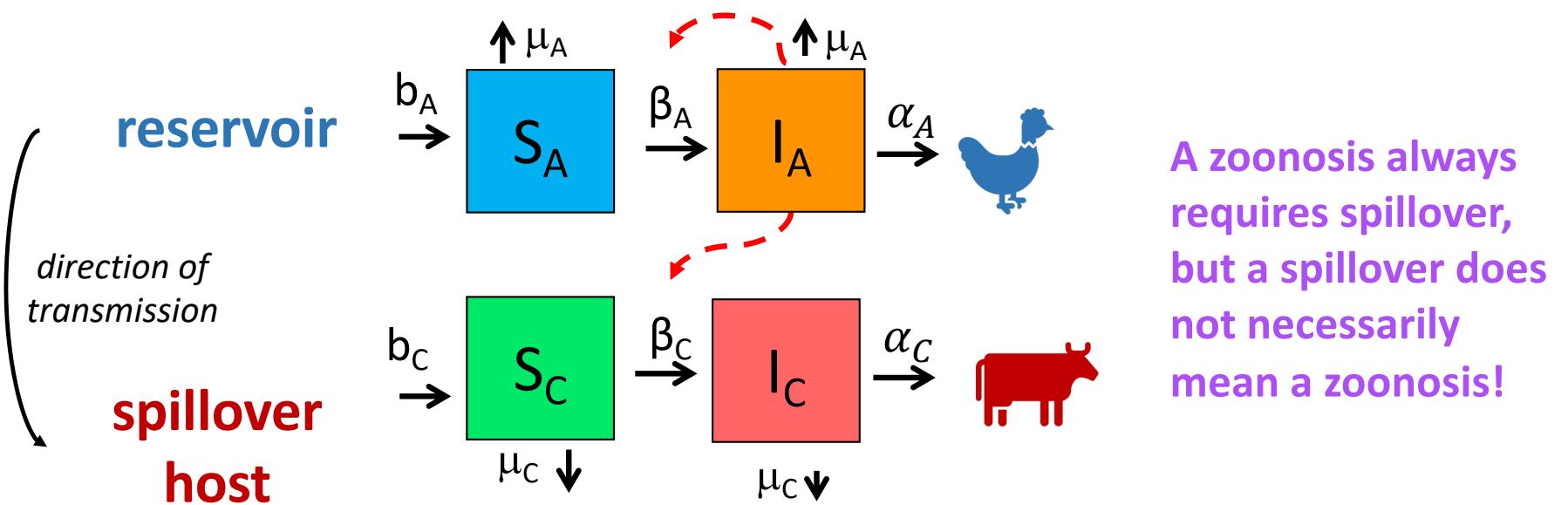
The **reservoir** host must be able to independently maintain the pathogen, with **population size > CCS!**

Animal hosts are not vectors!

ex: rabies

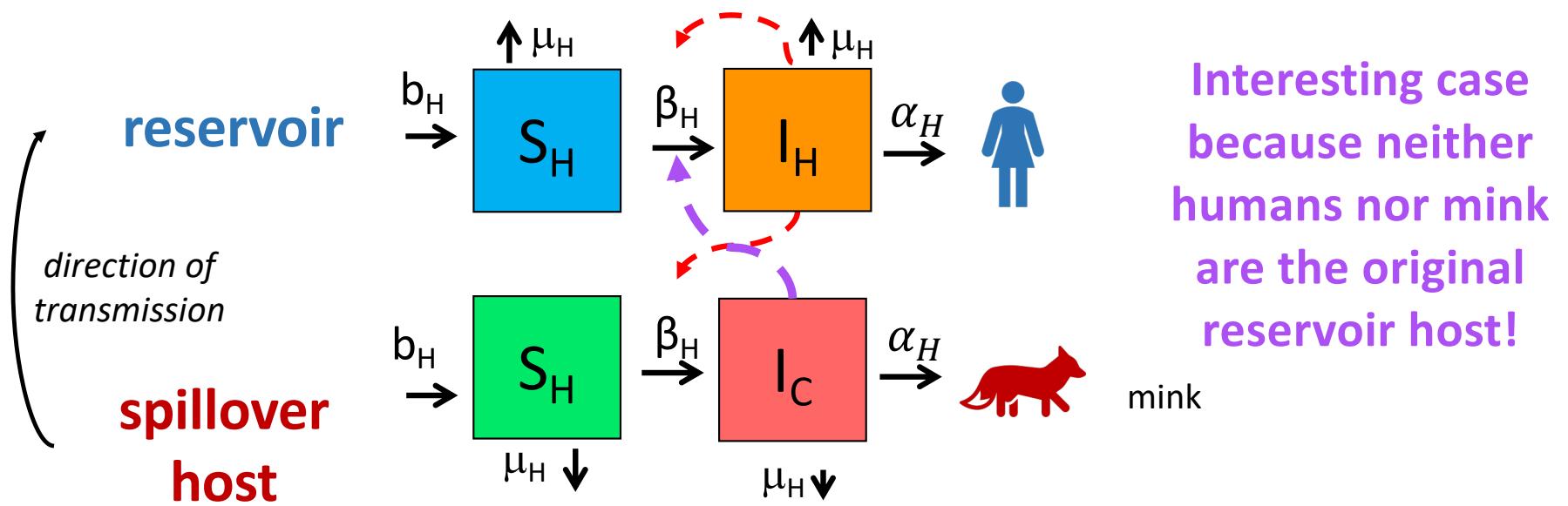
Haydon et al. 2002. *Emerging Infectious Diseases*.

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



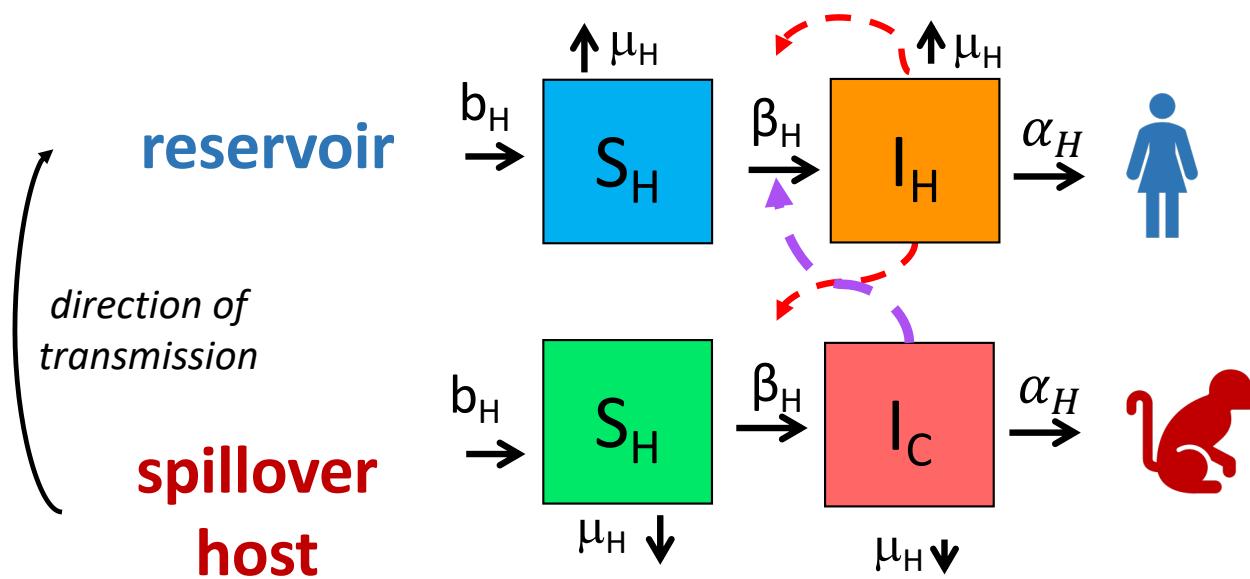
ex: avian flu

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



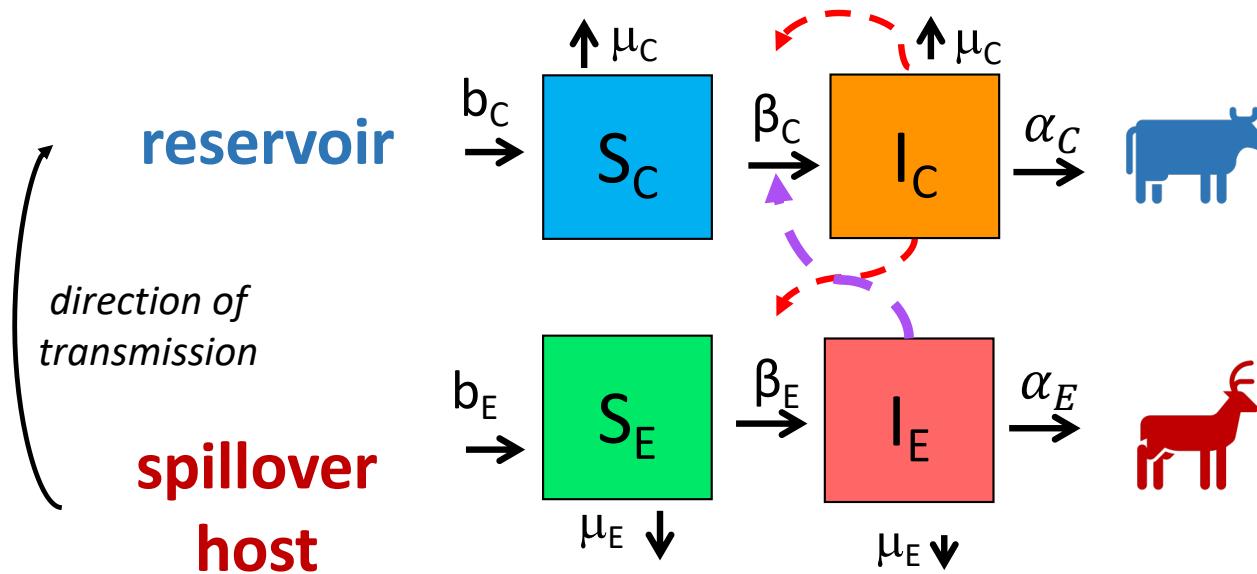
ex: SARS-CoV-2

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

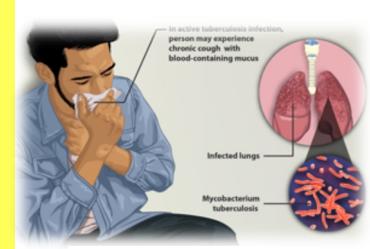
Sustained transmission and human outbreaks



Ebola virus (especially post-2014)

Stage V

Transmits exclusively in humans



Tuberculosis

$$R_0 < 1$$

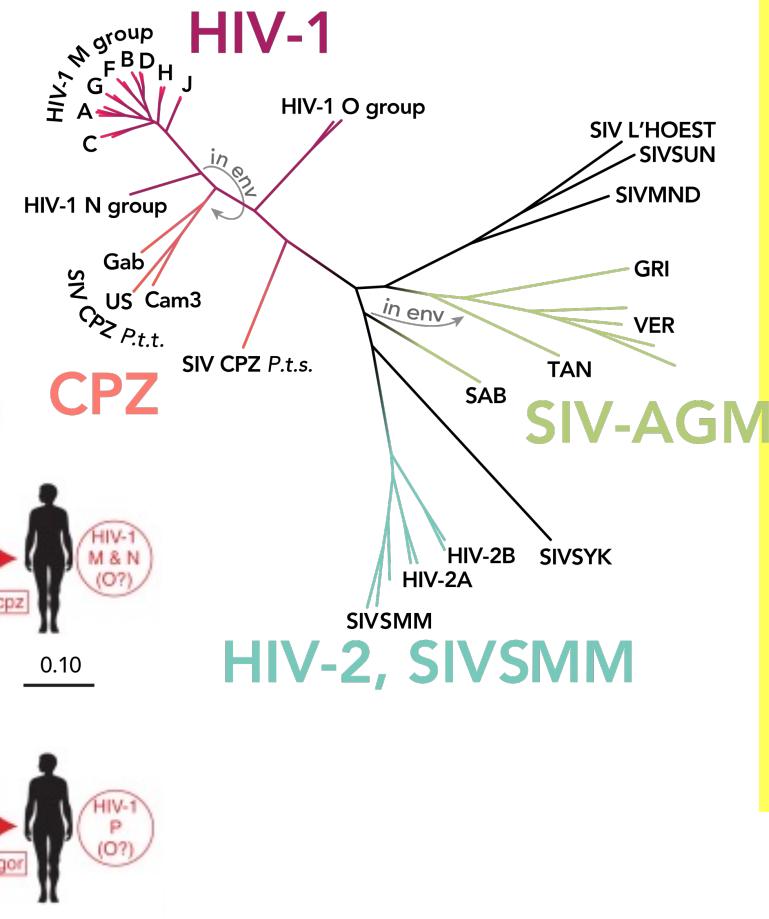
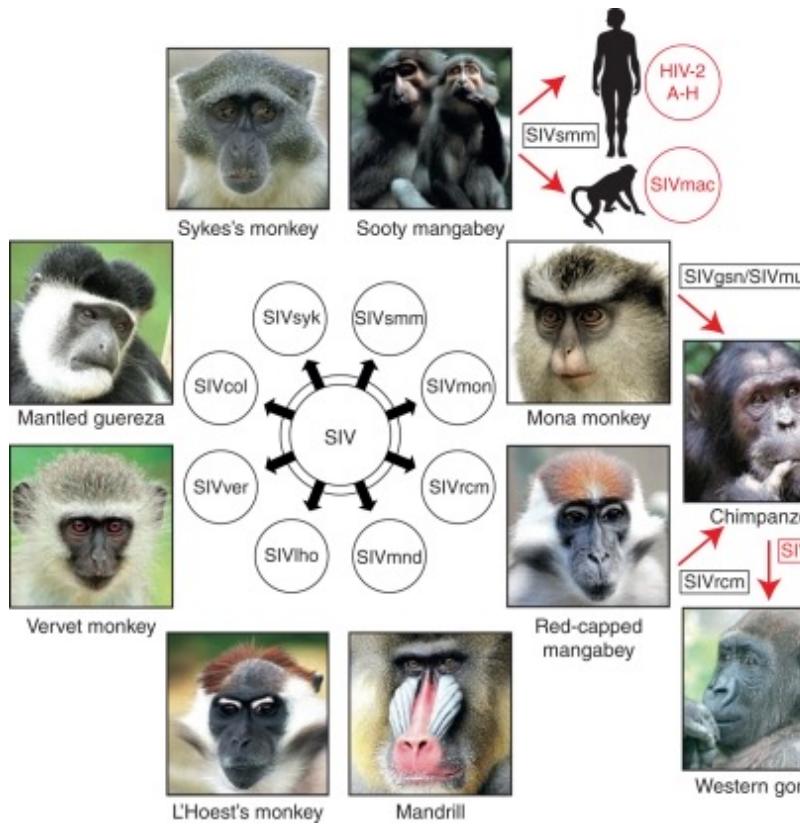
$$R_0 \approx 1$$

$$R_0 > 1$$

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!



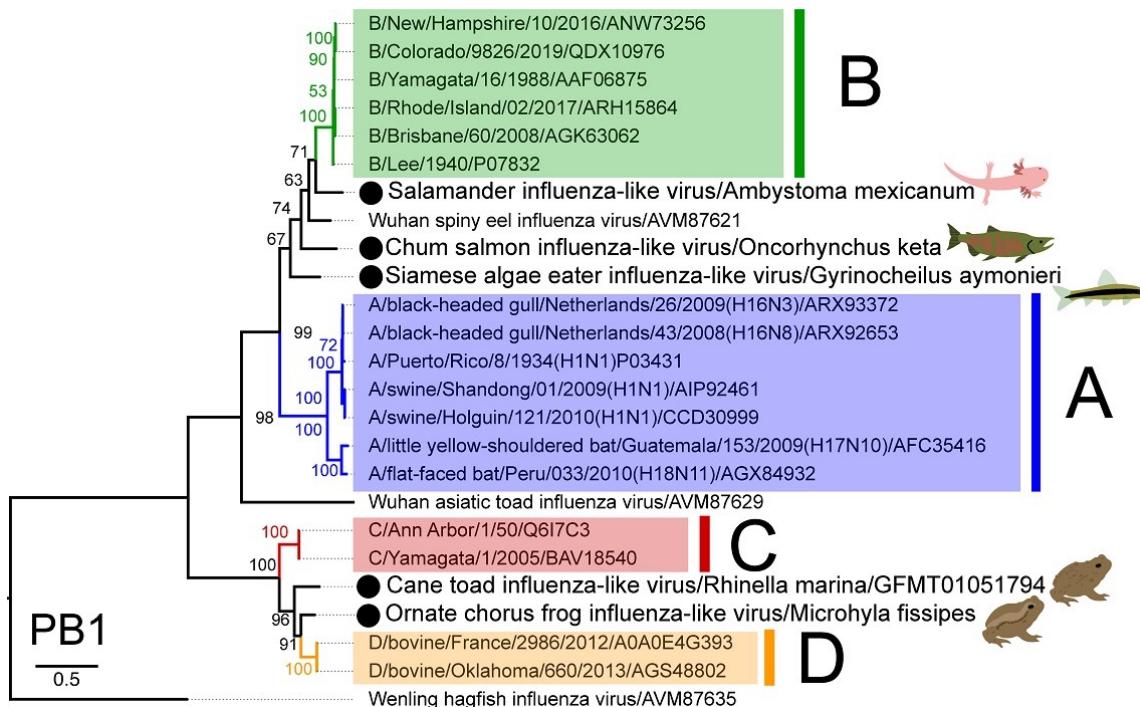
Stage V
Transmits exclusively in humans



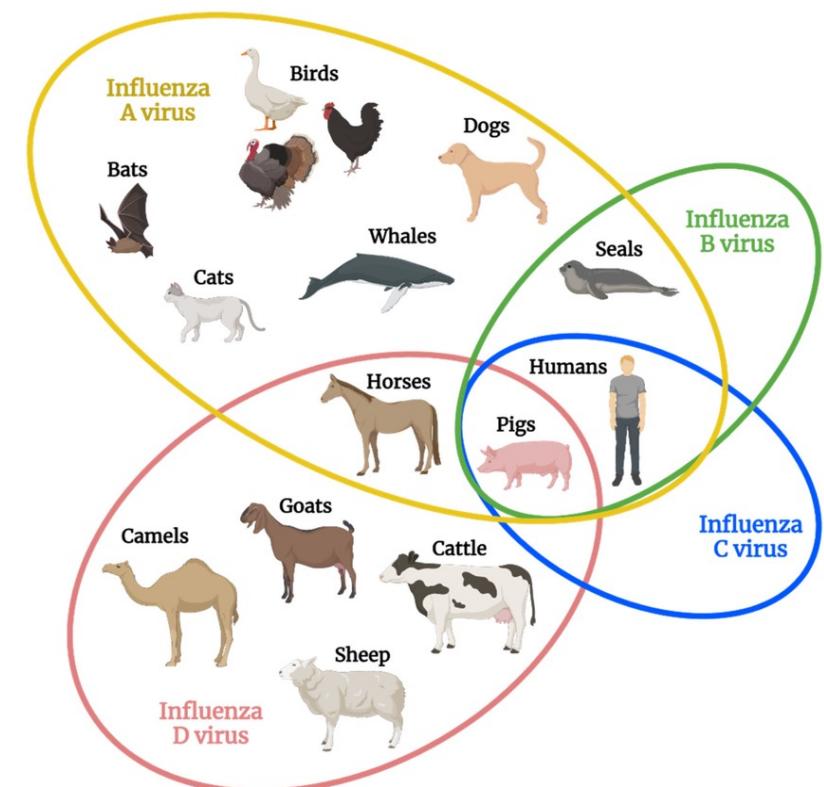
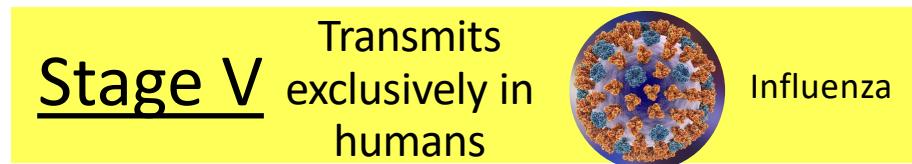
HIV

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

When is influenza zoonotic?



Parry et al. 2020. *Viruses*.



Skelton & Huber. 2022. *Viruses*.

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

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

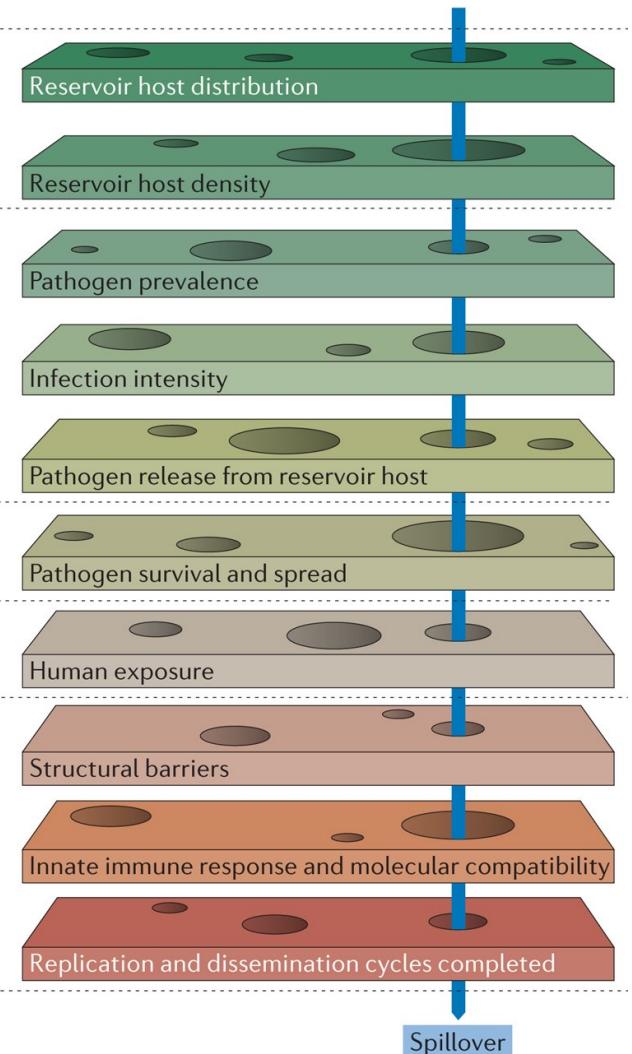
Animal ecology, population biology,
biogeography, behavioural ecology,
landscape ecology, agricultural sciences

Disease ecology, animal epidemiology,
infectious disease dynamics, immunology,
microbiology, veterinary medicine

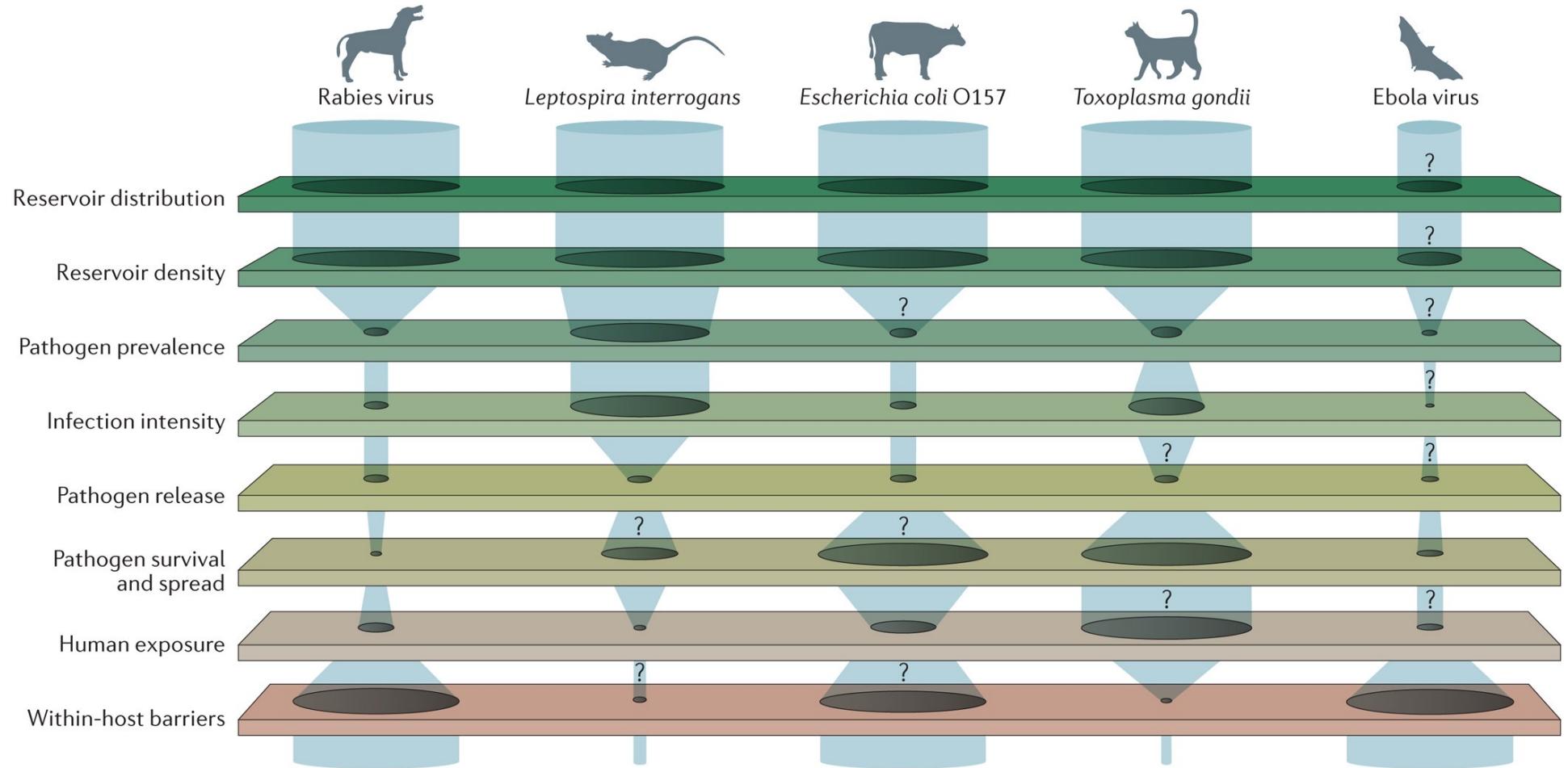
Microbiology, disease ecology, vector
ecology, epidemiology, spatial ecology,
infectious disease dynamics

Human epidemiology, medical anthropology,
vector ecology, social sciences, behavioural
ecology, infectious disease dynamics

Microbiology, innate and adaptive
immunology, cell biology of pathogen-host
interactions, pathology, genetics,
evolutionary biology



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

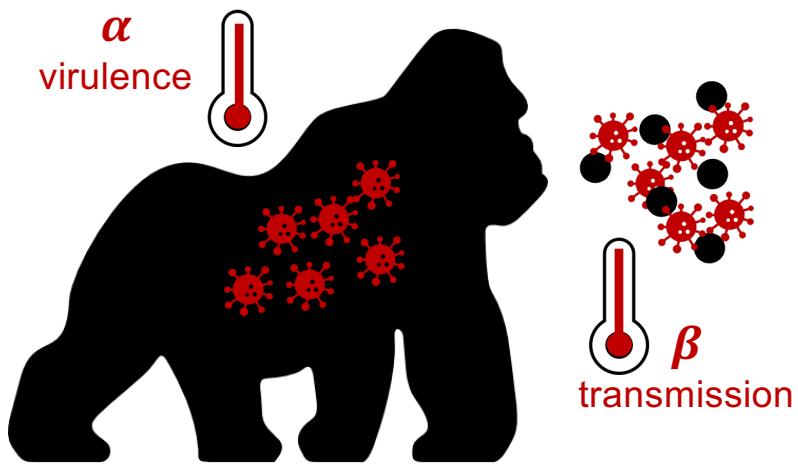


Bottlenecks to zoonotic transmission vary for different pathogens.

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).



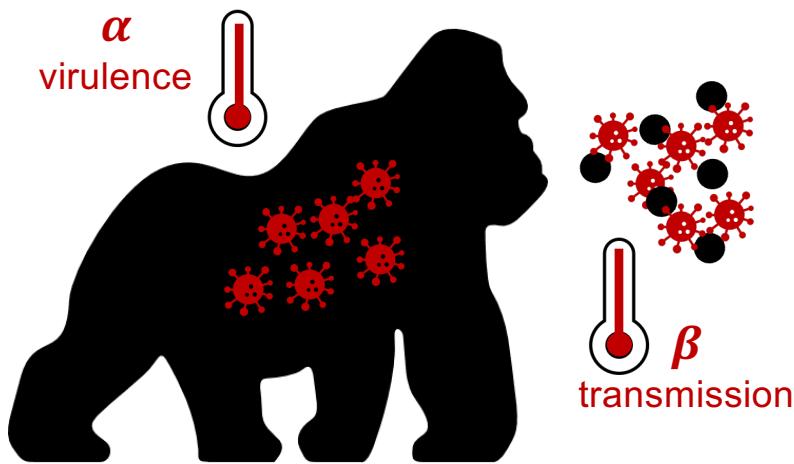
Mechanisms that promote **transmission** may also enhance **virulence** to the host.

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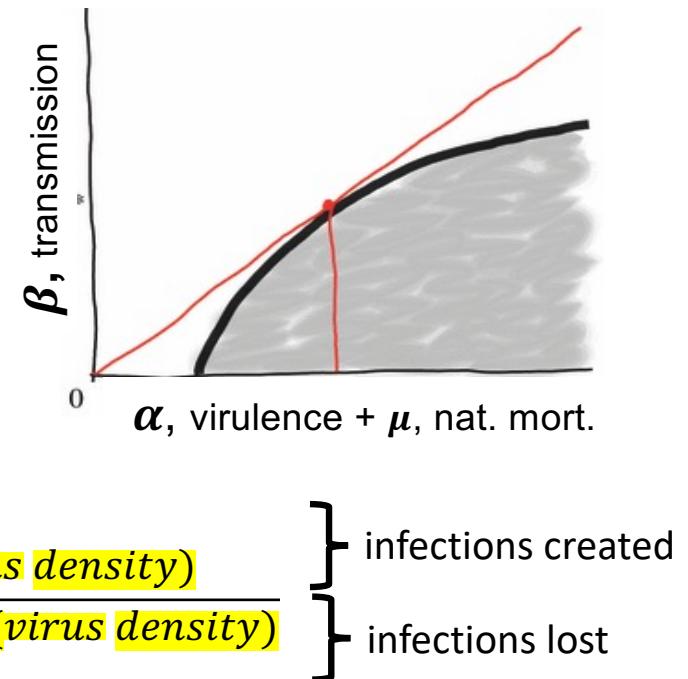
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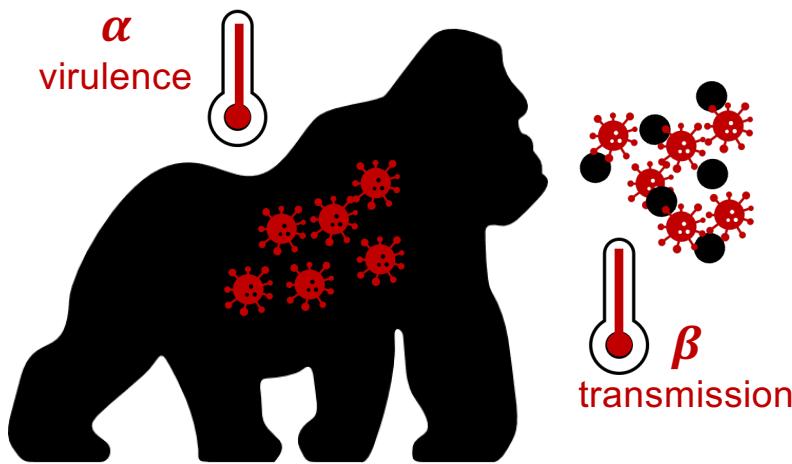
As a result, we predict the evolution of "**optimal virulence**".

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!



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

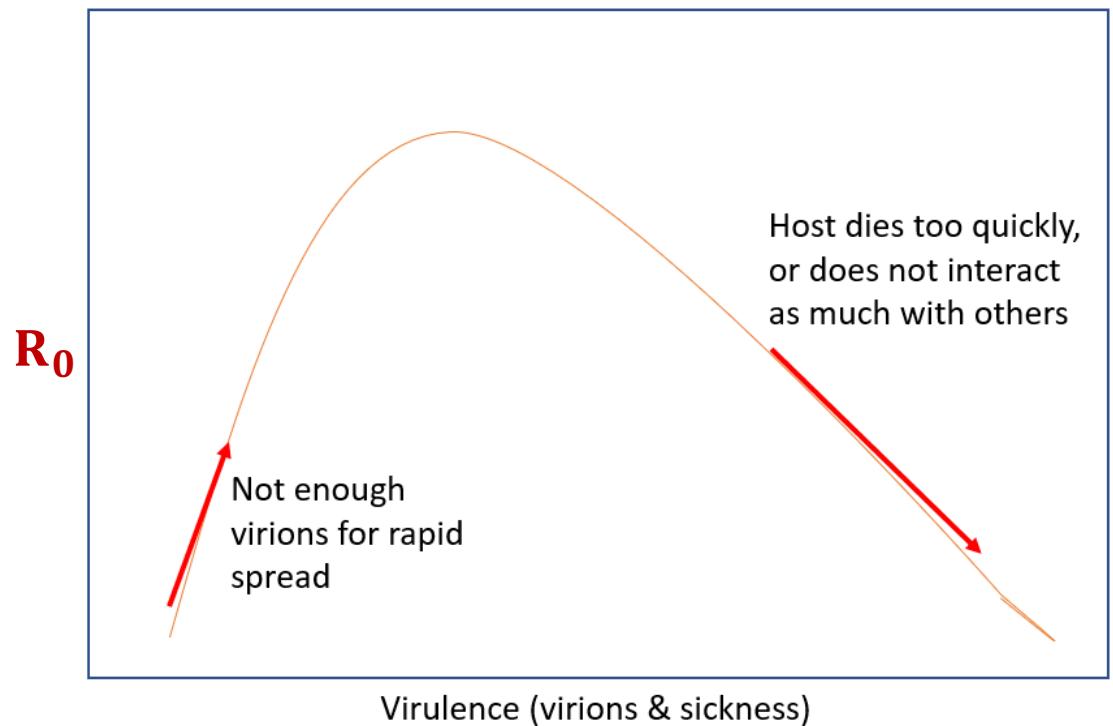
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Why do pathogens make us sick?

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



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

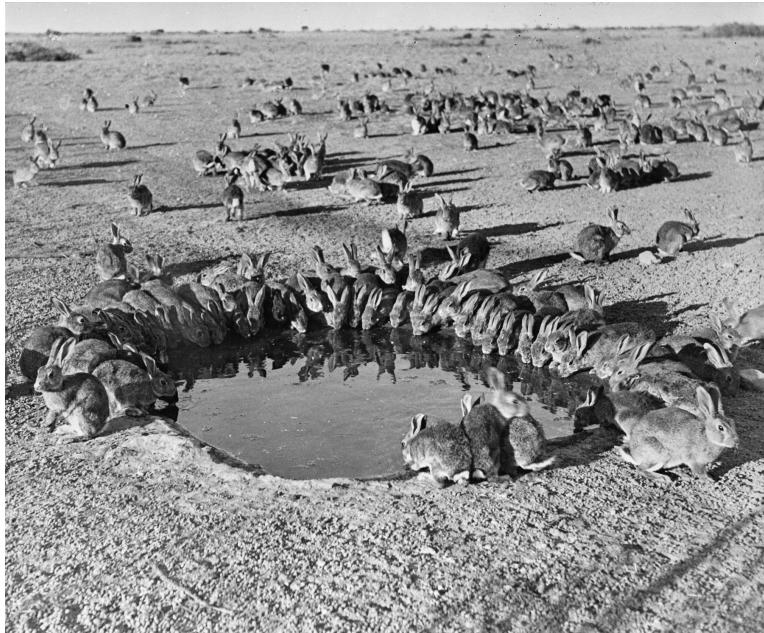
† Data from Marshall & Fenner (1960).

‡ Data from Edmonds *et al.* (1975).

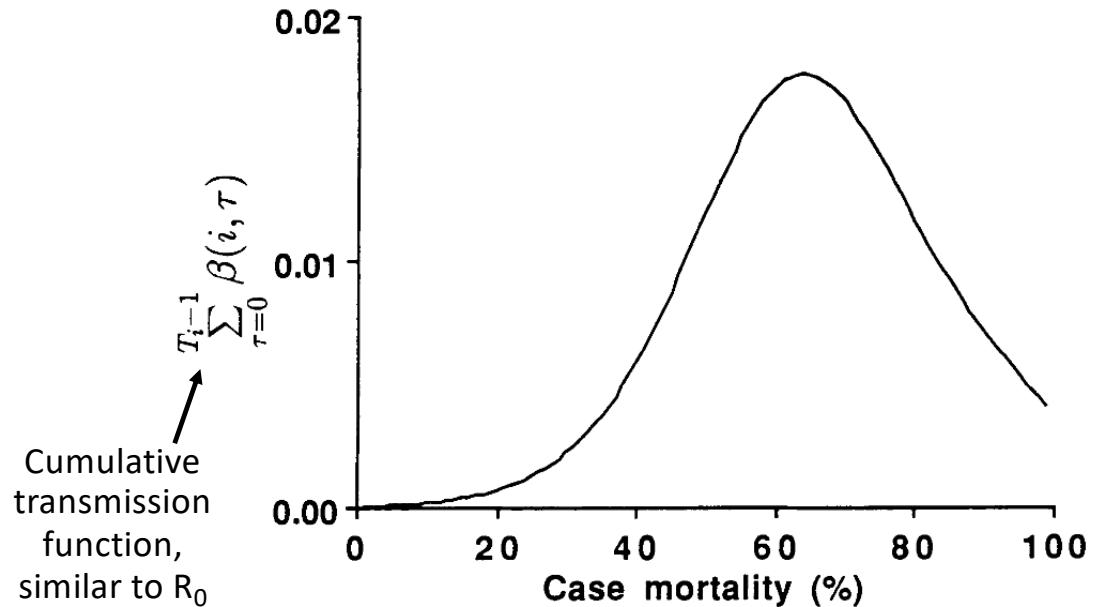
§ Data from J. W. Edmonds and R. C. H. Shepherd (personal communication, 1982).

|| 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

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

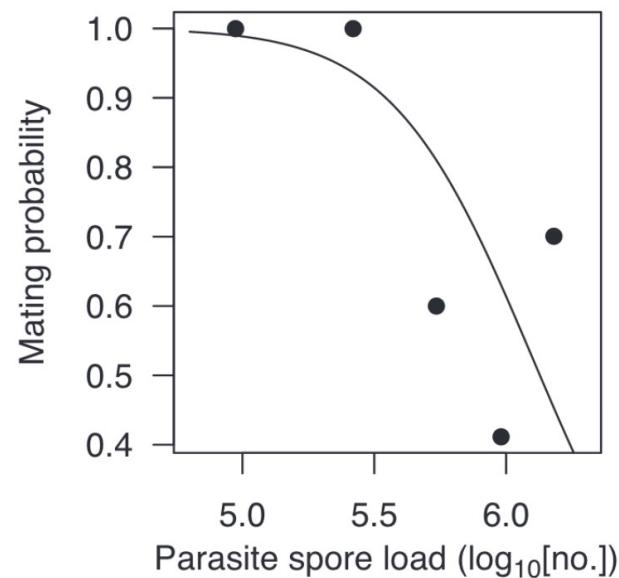
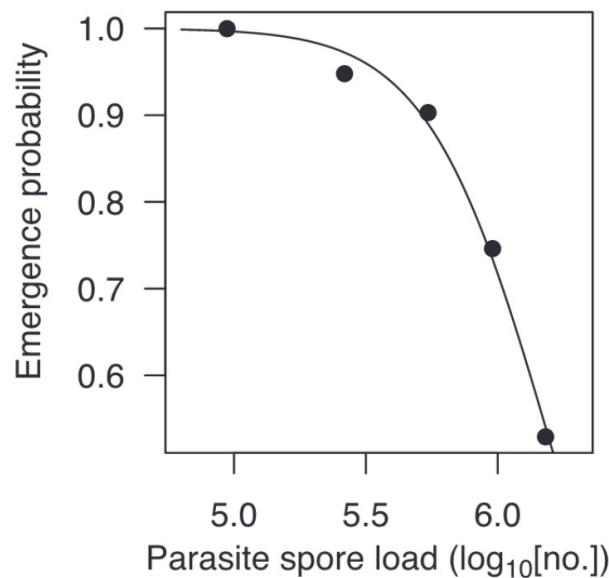
SIMON A. LEVIN
*Section of Ecology and Systematics, Corson Hall, Cornell University,
Ithaca, New York 14853 USA*

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

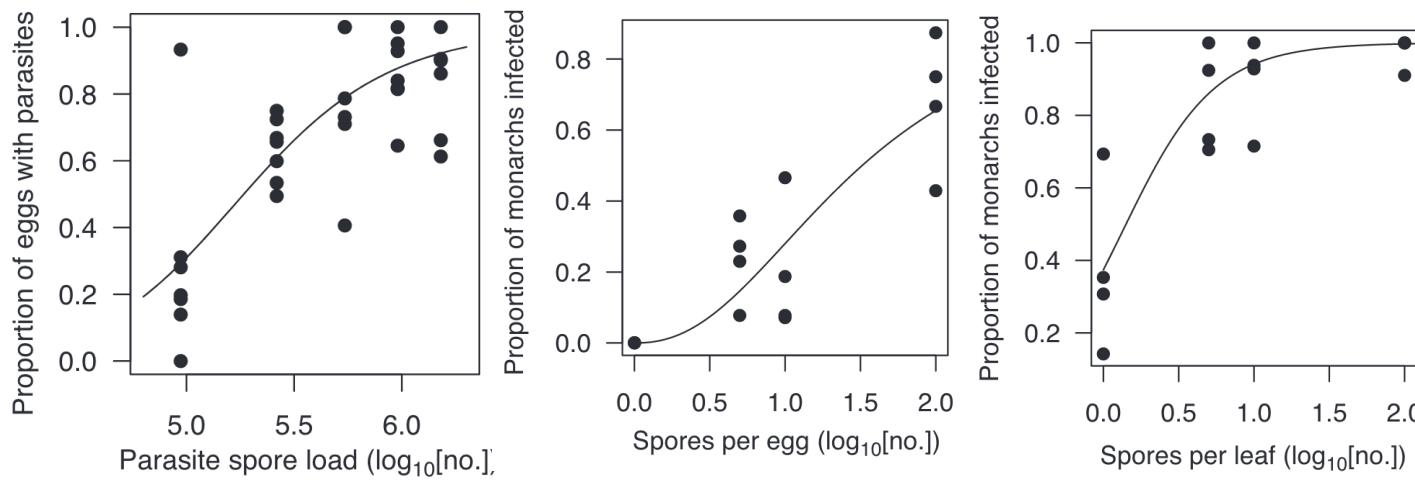
- Monarch butterflies infected with the protozoan parasite, *Ophryocystis elektroscirrha*, demonstrate reduced emergence and mating probabilities at higher parasite spore load (**virulence**).



de Roode et al. 2008. PNAS.

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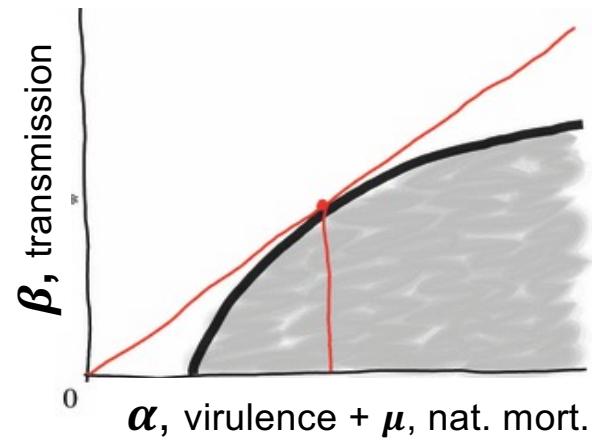
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- On the flip side, higher parasite loads also led to increased **transmission** through higher proportions of monarch eggs that acquired spores and higher numbers of parasites per egg and milkweed leaf, which increased the probability of infection.



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- 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.**

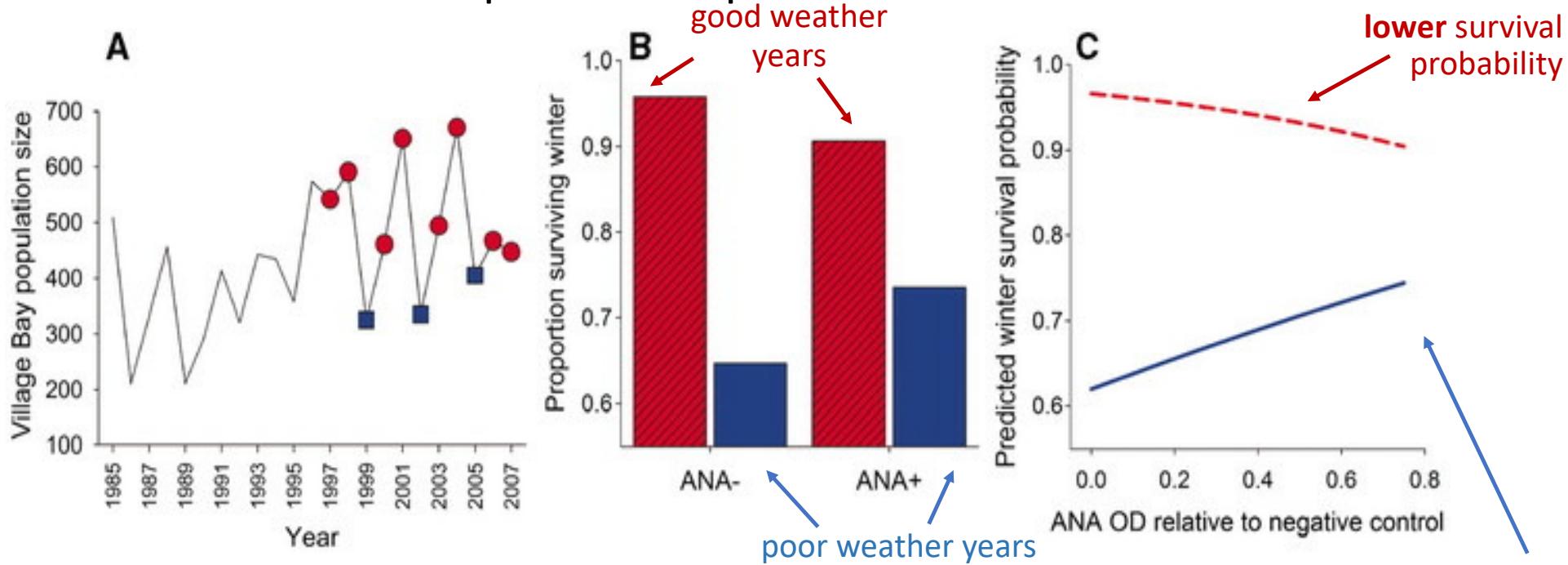


de Roode et al. 2008. PNAS.

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!
- 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

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



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

In good weather years, higher ANA leads to **lower survival probability**
In poor weather years, higher ANA leads to **higher survival probability**

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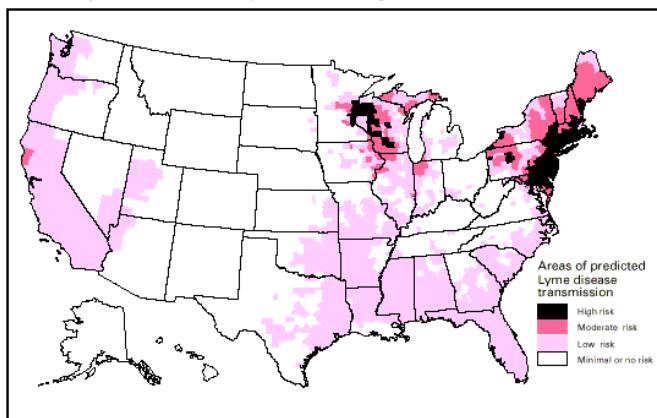
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 - In the case of zoonoses, the bulk of our measurements may be derived from a different host than the one in which the virus evolved
- Many examples of cases in which transmission is decoupled from virulence, due to more complex transmission dynamics.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



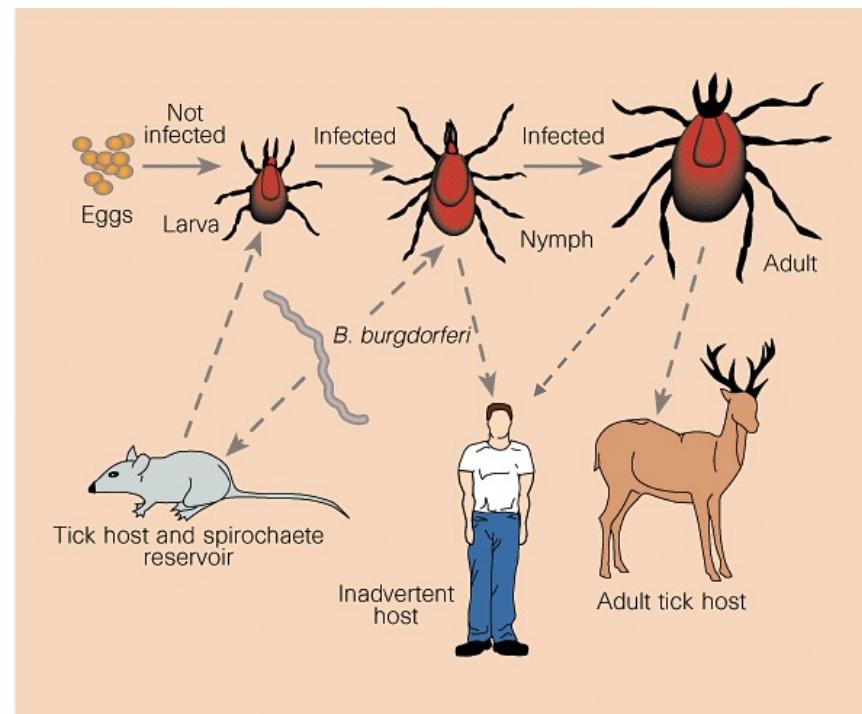
Disease dynamics in the **broader community**

Example: Lyme Disease

National Lyme disease risk map with four categories of risk



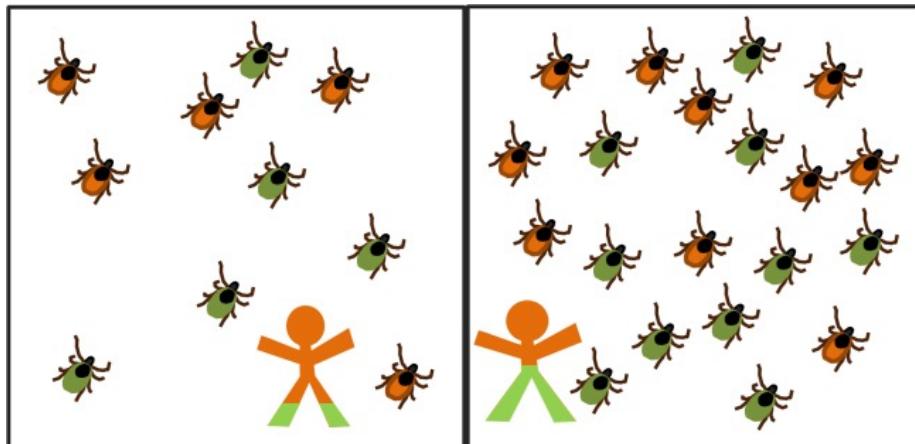
- Lyme disease is a vector-borne disease caused by the bacterium, *Borrelia burgdorferi*, vectored by *Ixodes* especially *Ixodes scapularis* ticks.
- Nymph ticks are borne in the spring, feed on small mammal hosts through the summer, then reproduce (particularly on deer) in the fall before going dormant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



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.

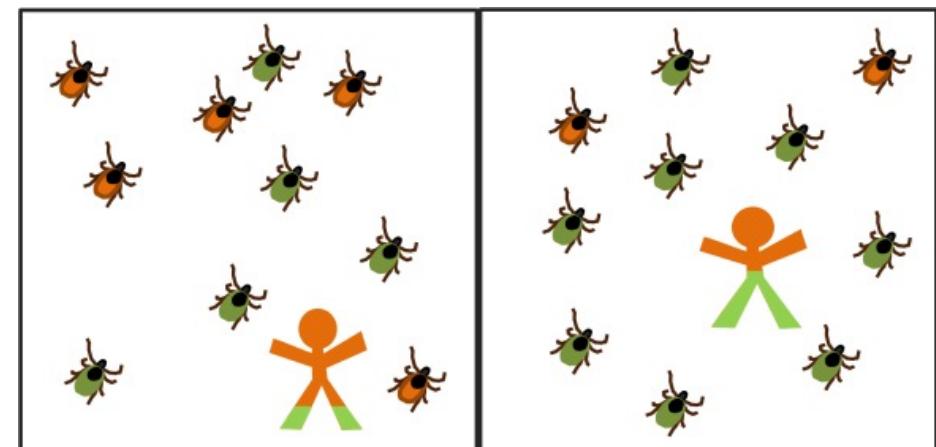
Scenario 1: Density of infected ticks



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



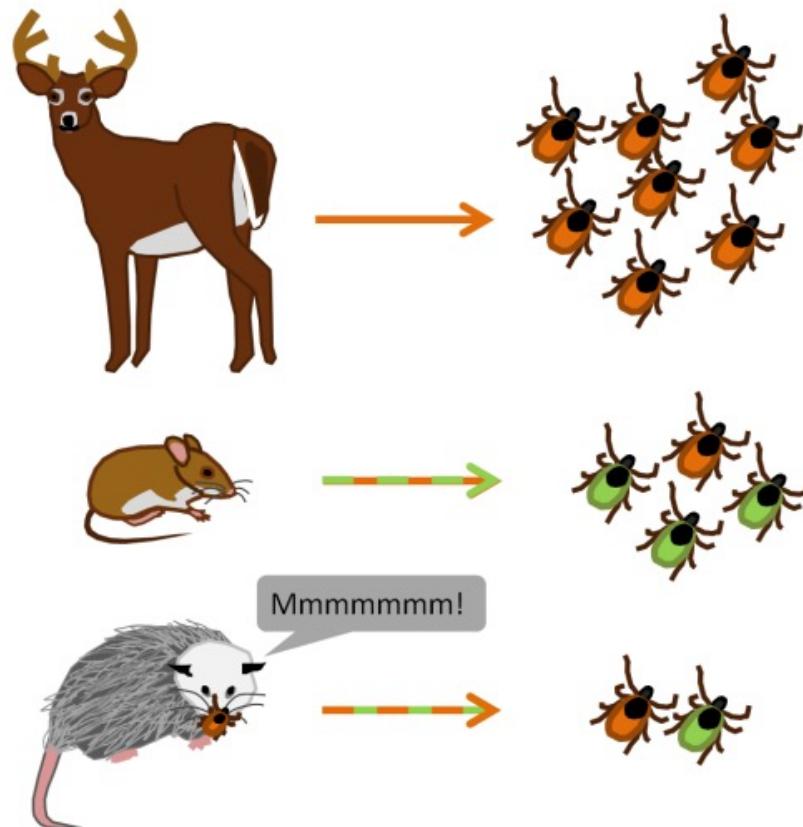
Area = 1 unit
Total Ticks = 10
Tick Density = 10 ticks/unit
Number Infected Ticks = 5
Prevalence Infected Ticks = 50%

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

lime = infected with Lyme

parasiteecology.wordpress.com

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

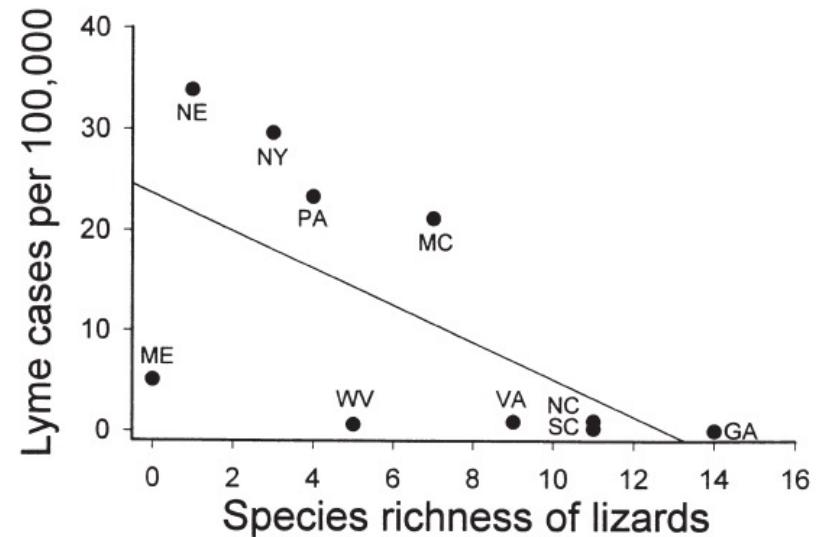
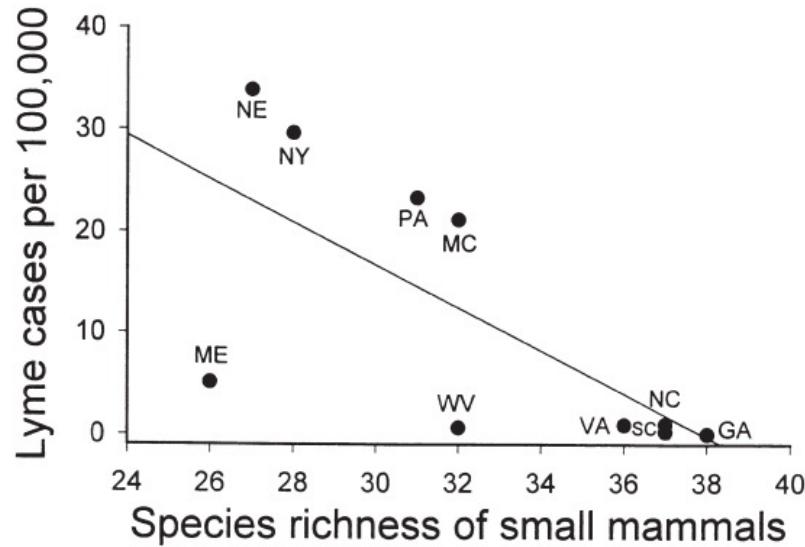


lime = infected with Lyme

- (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**.

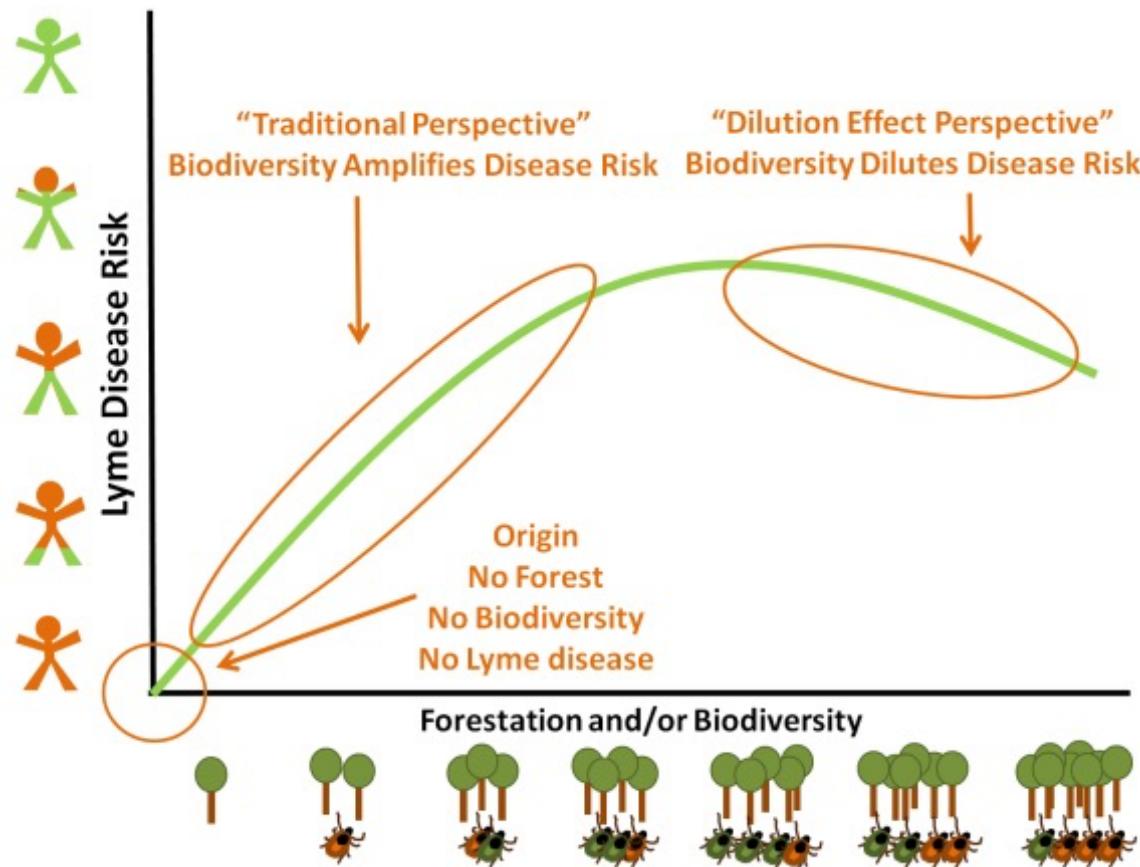
While a popular concept, it only holds in select cases!



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

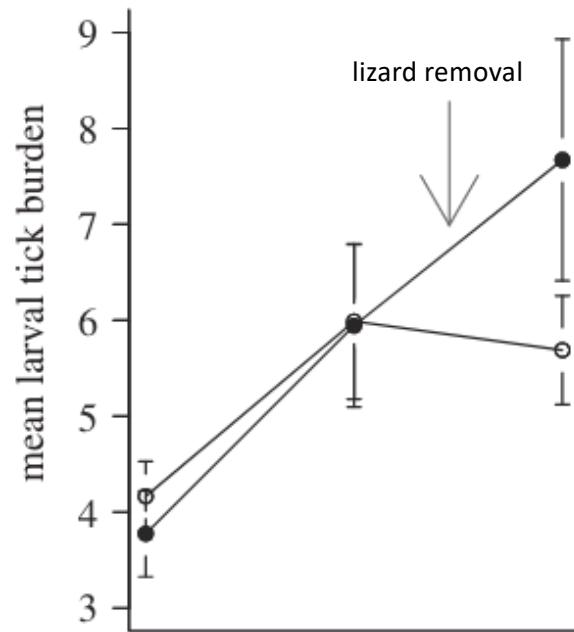
Patterns depend on the context of the wildlife community!

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



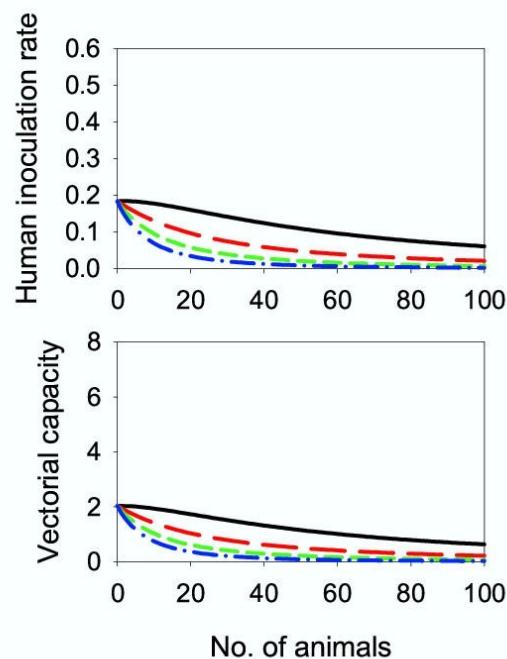
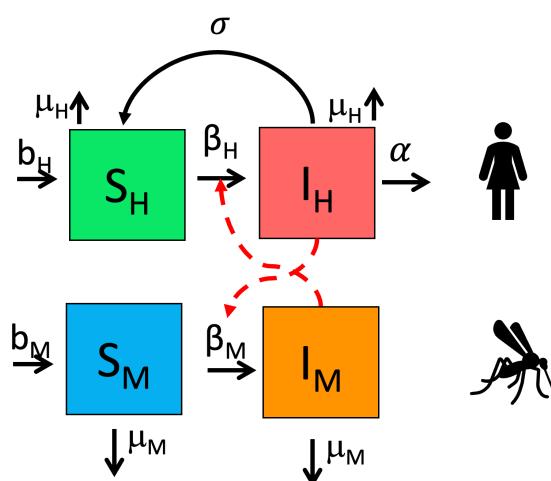
“Zooprophylaxis” is the diversion of pathogen-transmitting arthropods from humans to animals

Tick burden on 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

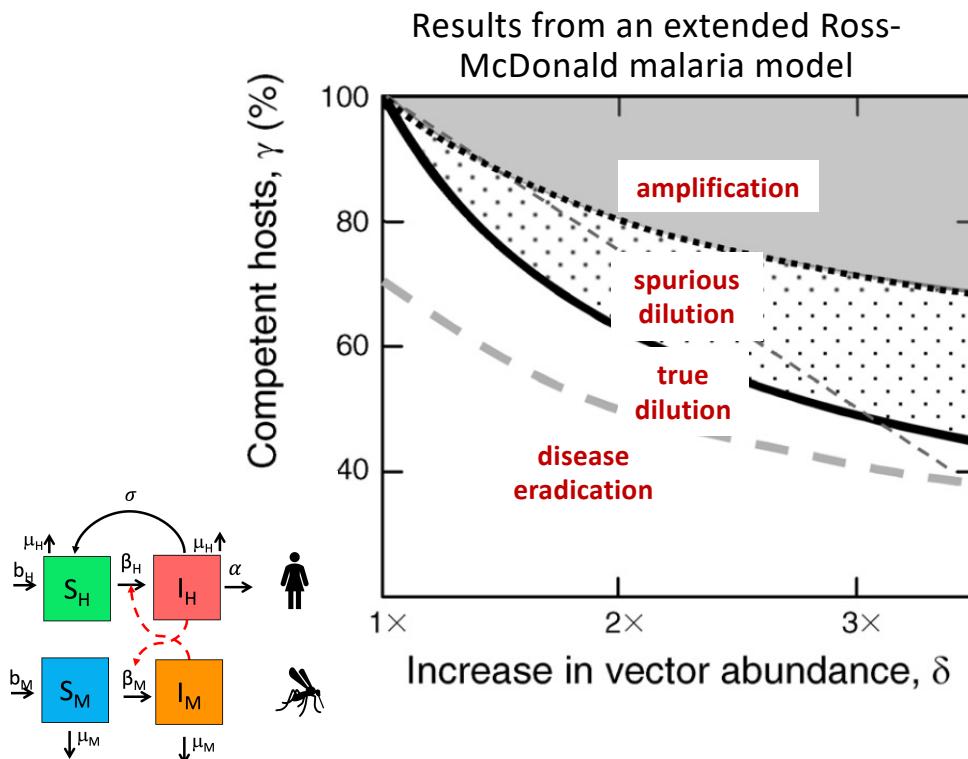
“Zooprophylaxis” has been suggested for malaria control –
but only works in cases by which livestock are used as bait to
draw mosquitoes closer to insecticides.



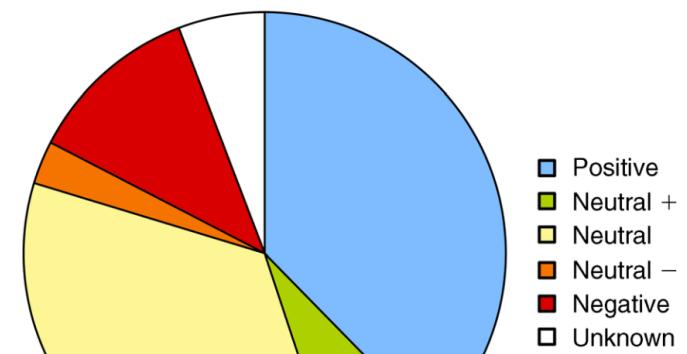
Black, red, green, blue = 0, 20, 40 or 60% chance of
mosquito being killed as a result of feeding on animals

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.



Meta-analysis of biodiversity impacts on 69 common human pathogens



Wood et al. 2014. *Ecology*