Fundamentals of Ecology

Week 9, Ecology Lecture 7

Cara Brook

February 28, 2023

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- These extensions included incorporating <u>inherited maternal immunity</u> into a disease model (important in the case of natural vs. vaccinated immunity in mothers), as well as modeling <u>different types of pathogen transmission</u>.
- <u>Environmental disease transmission</u> occurs when the pathogen is maintained outside the host in an <u>environmental reservoir</u>. We learned a few approaches to modeling this type of pathogen. Example: cholera, with water-borne transmission.

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- Vertical transmission occurs when a pathogen is passed mother-to-infant *in utero.* We learned how to model this in the case of HIV- and also learned a few details about this pathogen.

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- 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.

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- 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.

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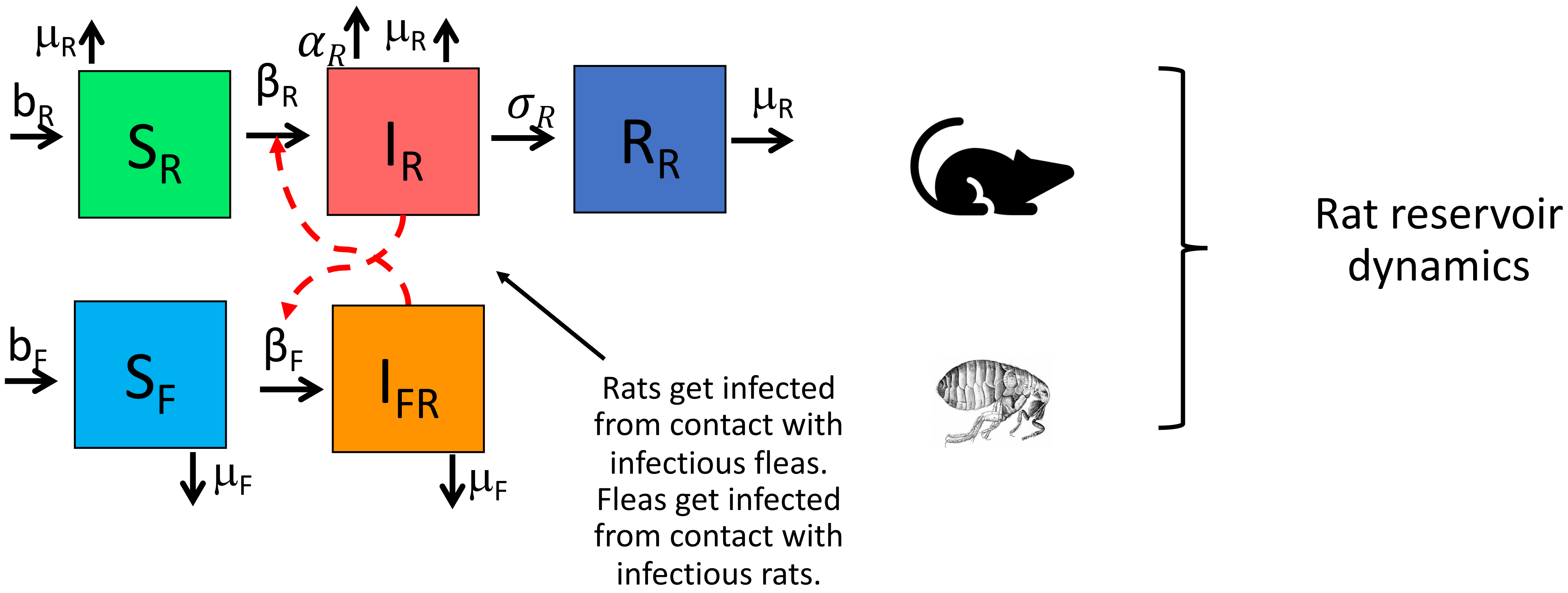

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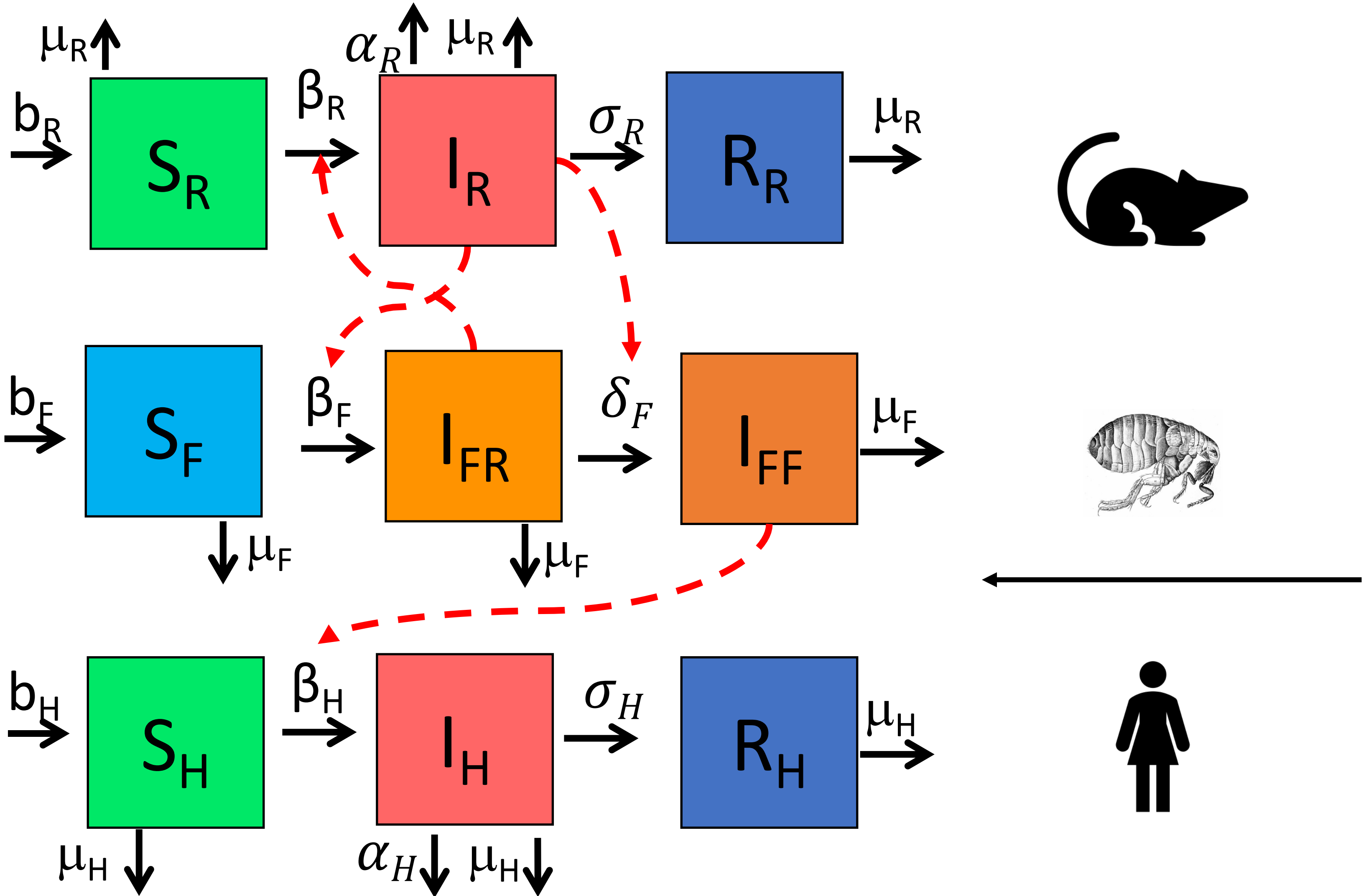

Humans get infected from contact with free-living infectious fleas!

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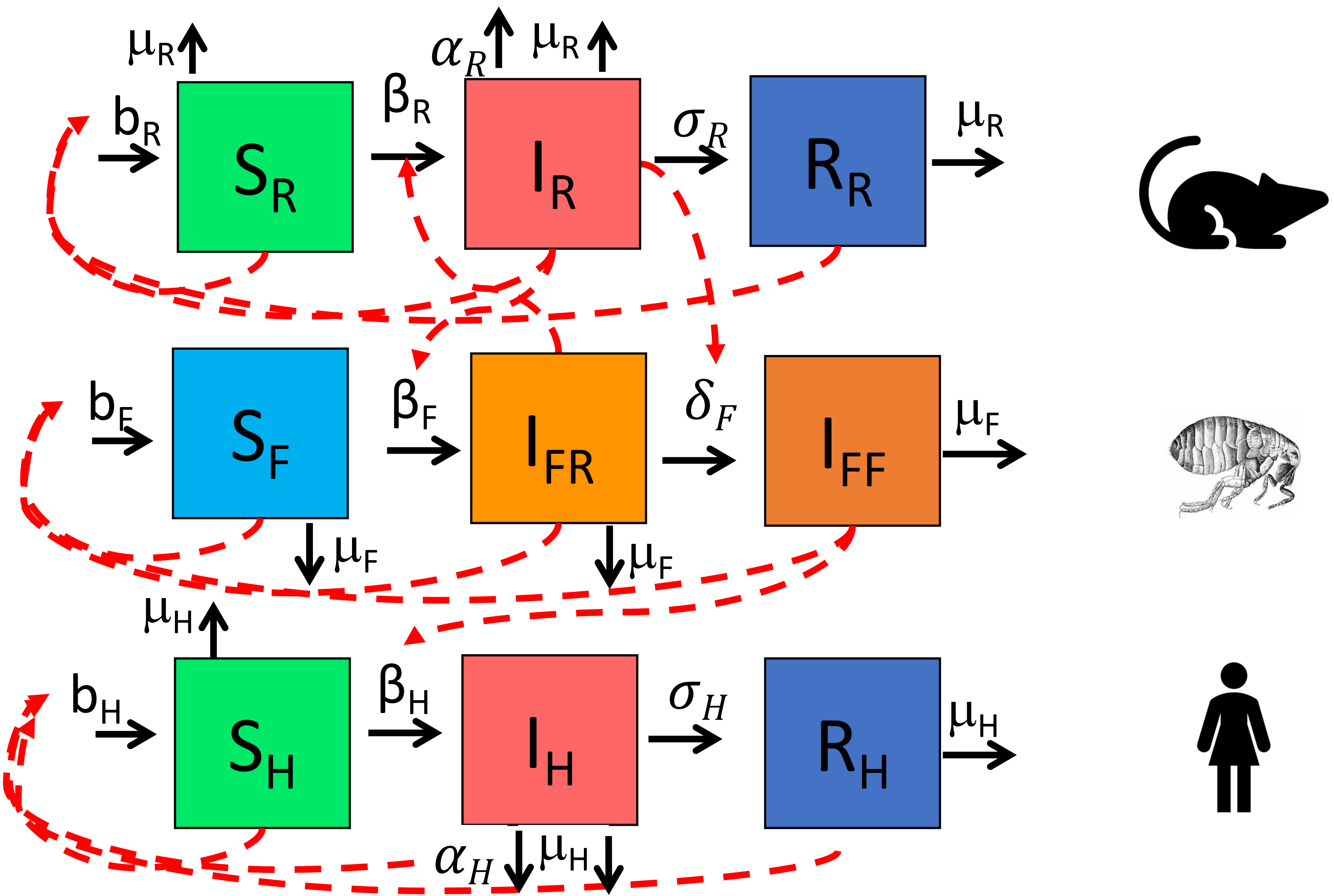
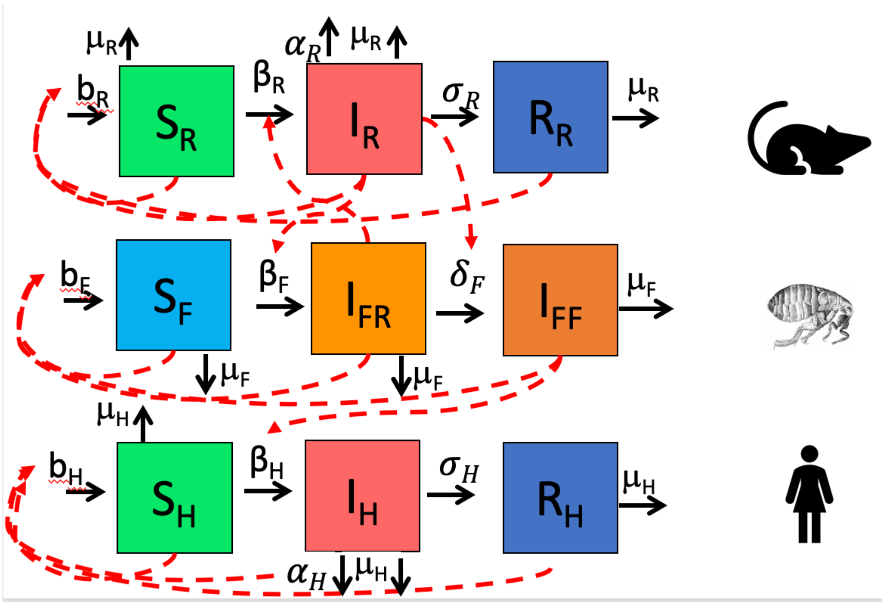


How many equations would we need to model this system?

A simple plague model



$$\left.\begin{aligned}
\frac{dS_R}{dt} &= b_R(S_R + I_R + R_R) - \beta_R S_R I_{FR} - \mu_R S_R \\
\frac{dI_R}{dt} &= \beta_R S_R I_{FR} - \mu_R I_R - \alpha_R I_R - \sigma_R I_R \\
\frac{dR_R}{dt} &= \sigma_R I_R - \mu_R R_R
\end{aligned}\right\} \text{Rats}$$

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\frac{dS_F}{dt} &= b_F(S_F + I_{FR} + I_{FF}) - \beta_F S_F I_R - \mu_F S_F \\
\frac{dI_{FR}}{dt} &= \beta_F S_F I_R - \delta I_{FR} - \mu_F I_{FR} \\
\frac{dI_{FF}}{dt} &= \delta I_{FR} - \mu_F I_{FF}
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\frac{dS_H}{dt} &= b_H(S_H + I_H + R_H) - \beta_H S_H I_{FF} - \mu_H S_H \\
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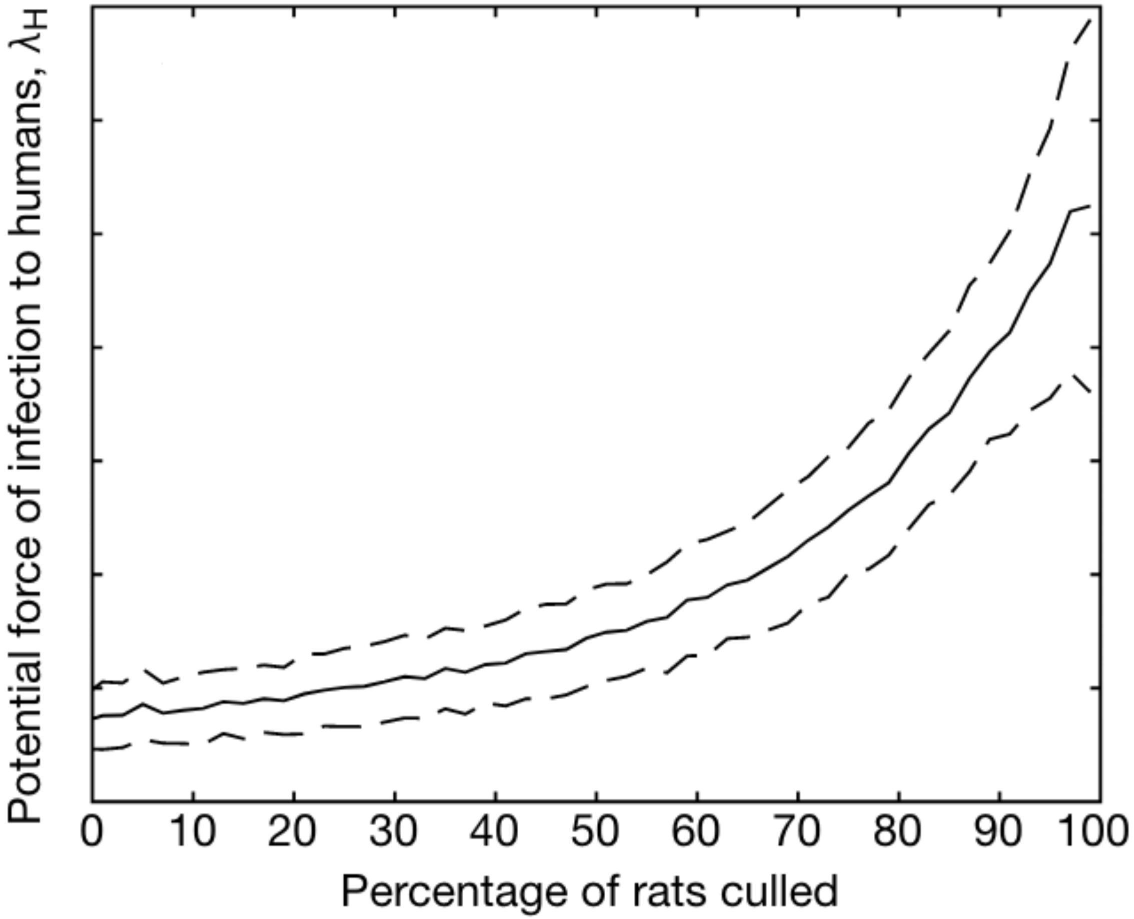
Fleas get infected from rats.
Humans get infected from free-living infected fleas!



"…from April 18 onwards, quantities of dead or dying rats were found in factories and warehouses…From the outer suburbs to the center of the town, in all the byways where the doctor's duties took him, in every thoroughfare, rats were piled up in garbage cans or lying in long lines in the gutters…On the fourth day the rats began to come out and die in batches…"

--*La Peste,* Albert Camus (1948)

Keeling and Gilligan. 2000. *Nature.*

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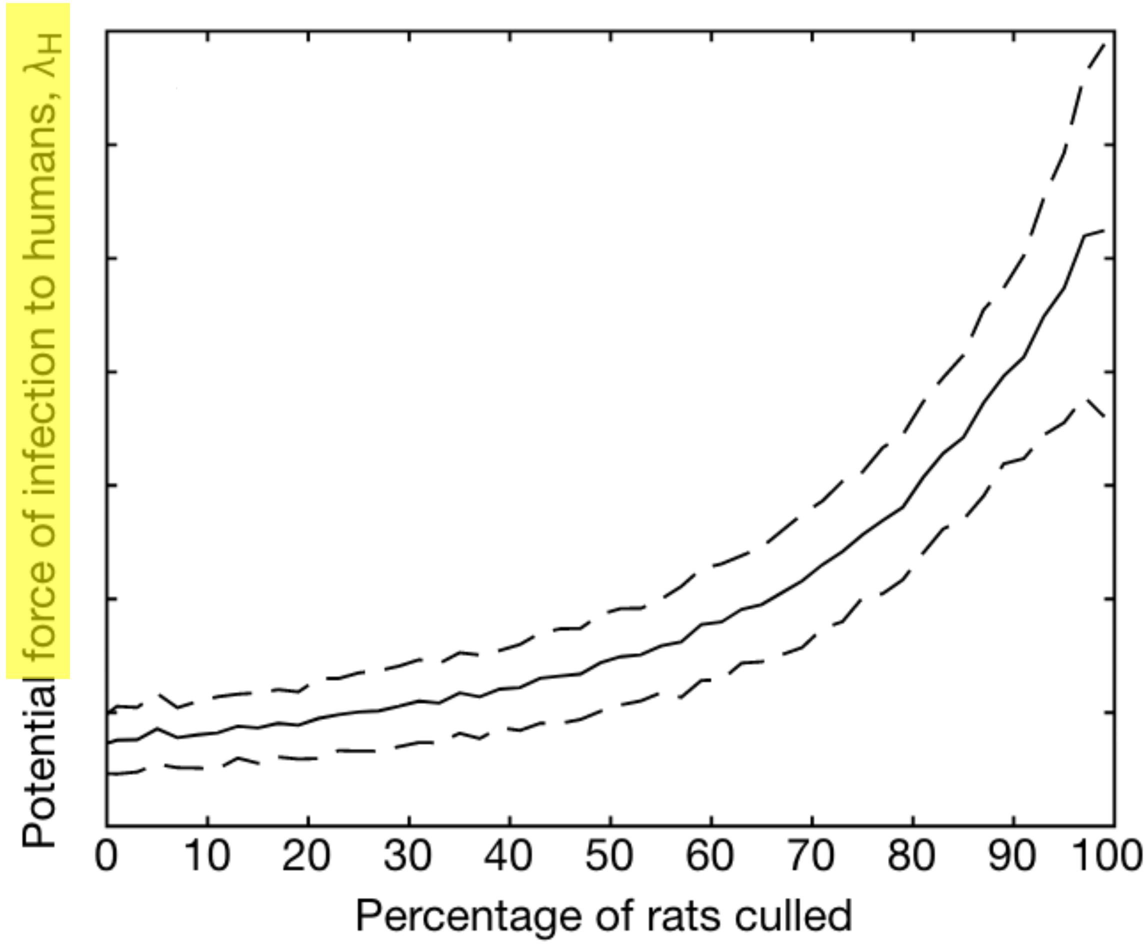

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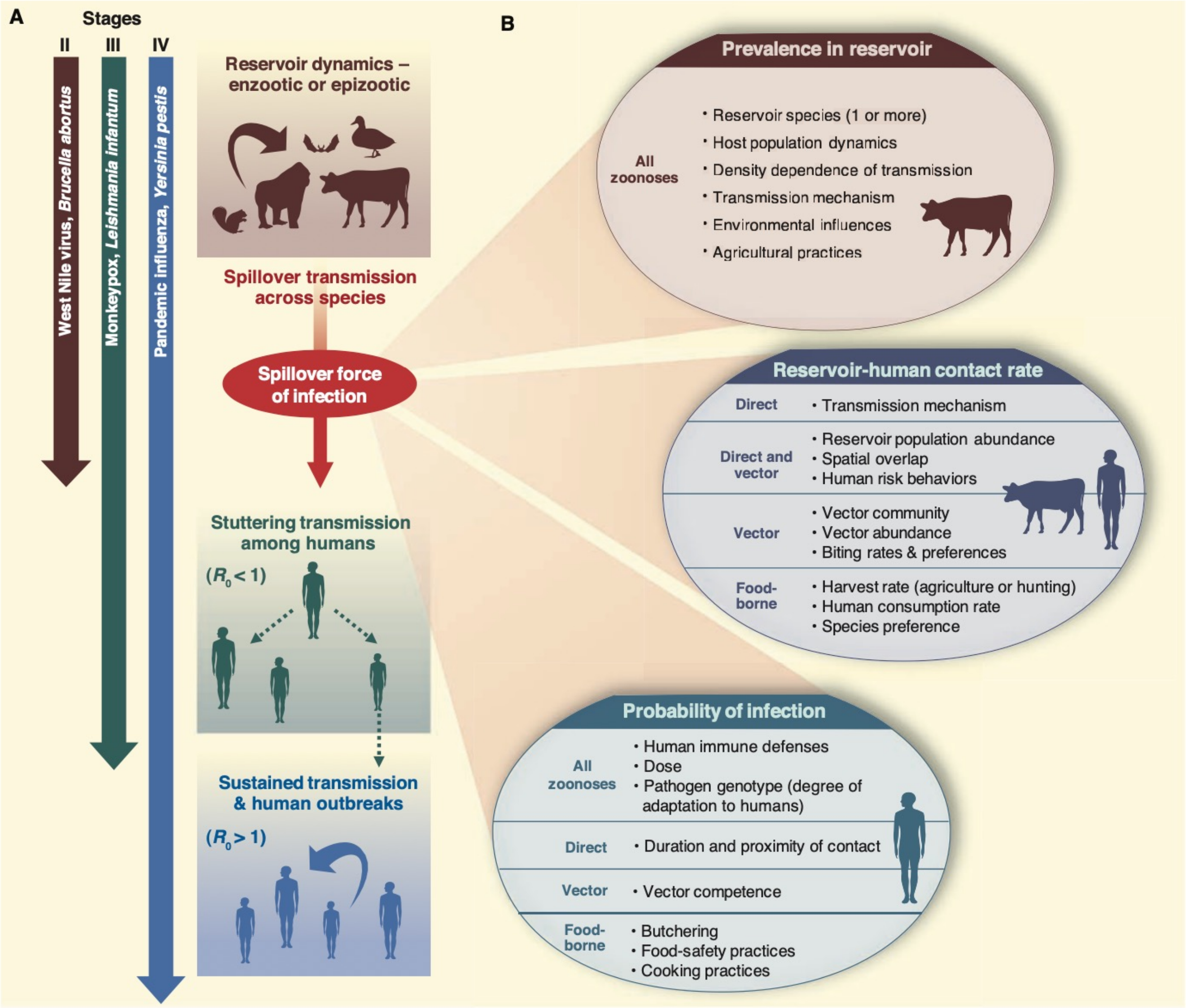
- For a single host pathogen: $\lambda = R_0 \frac{I}{N}$
- (**FOI = R_0 * proportion infected**)



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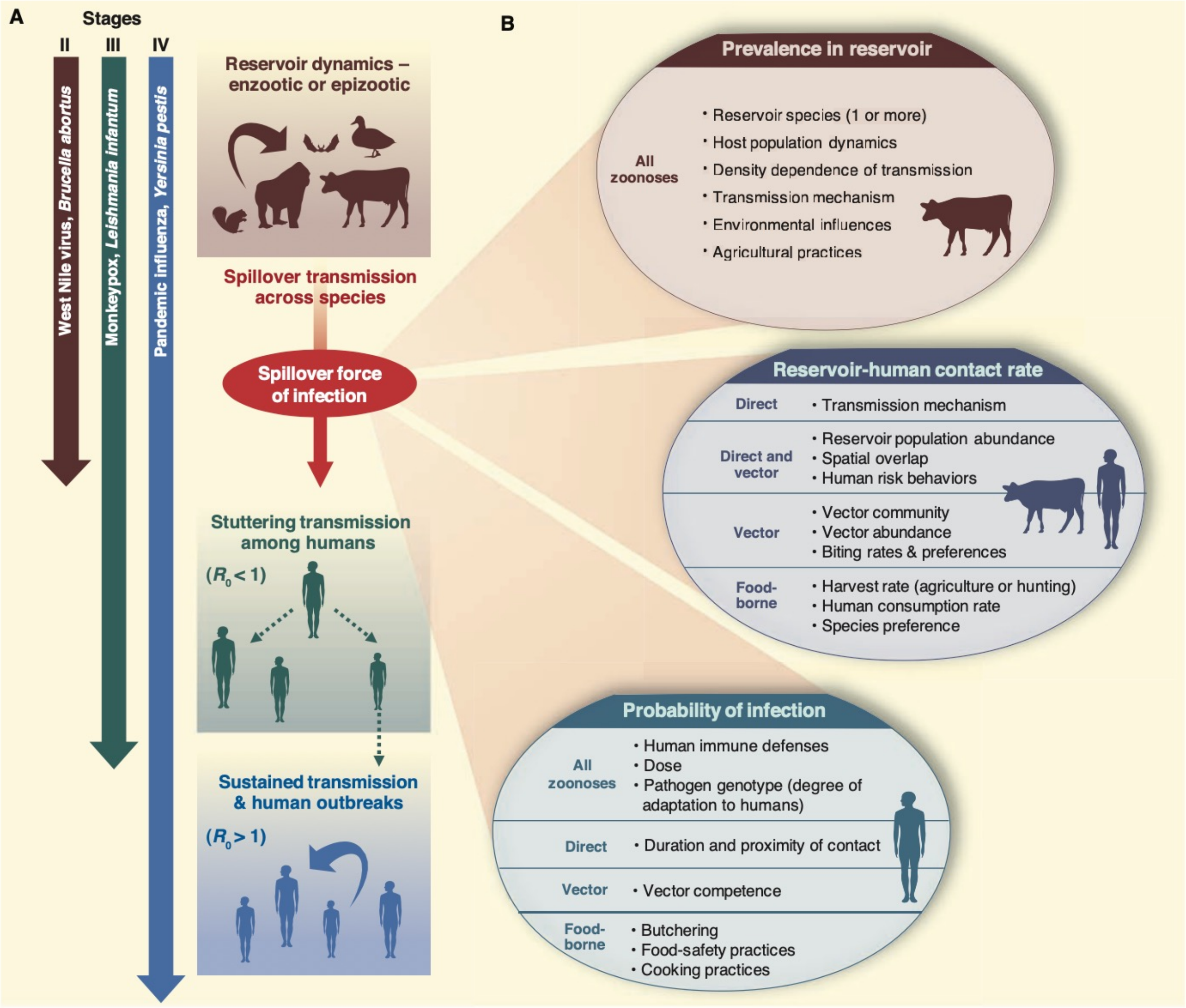

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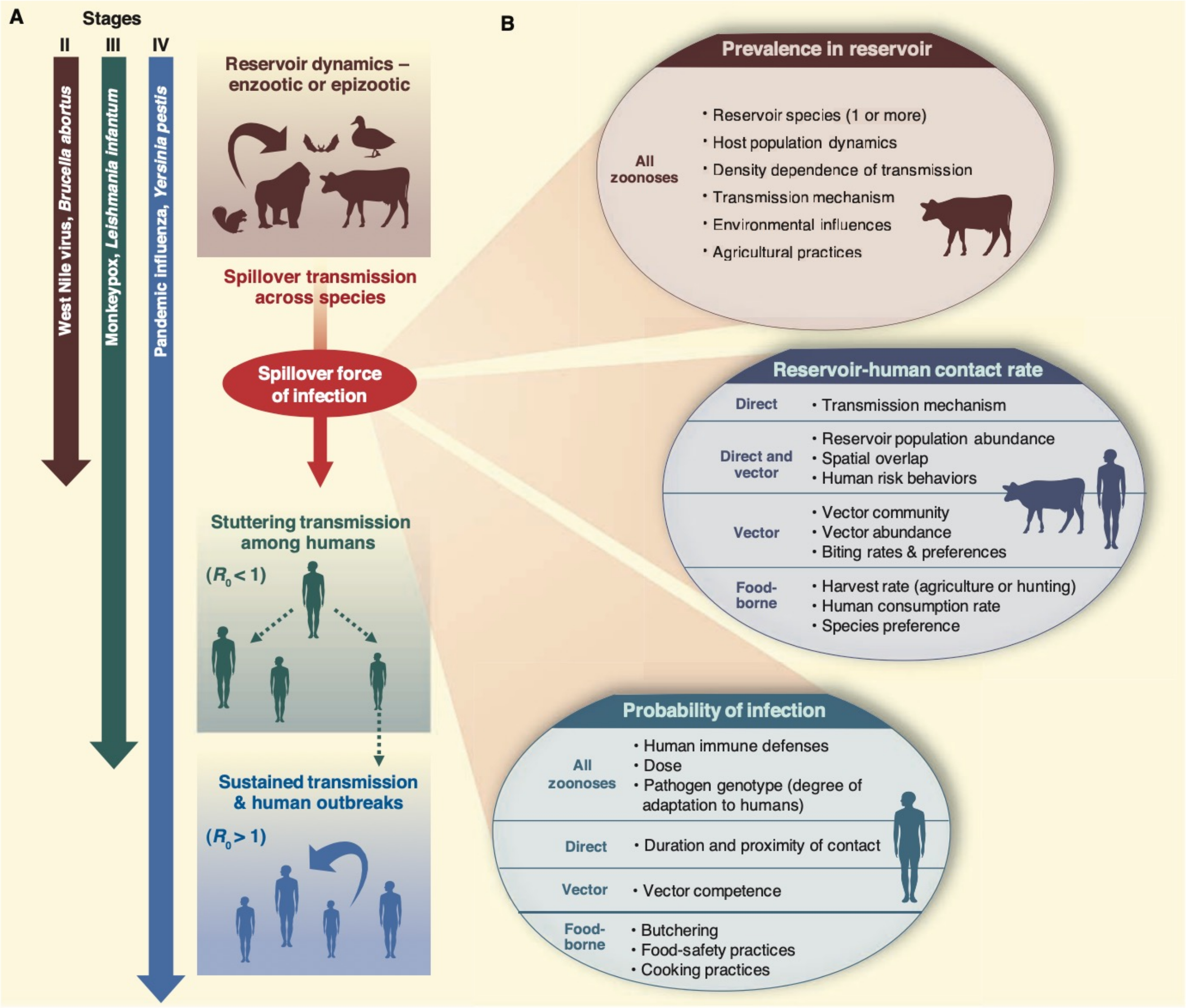

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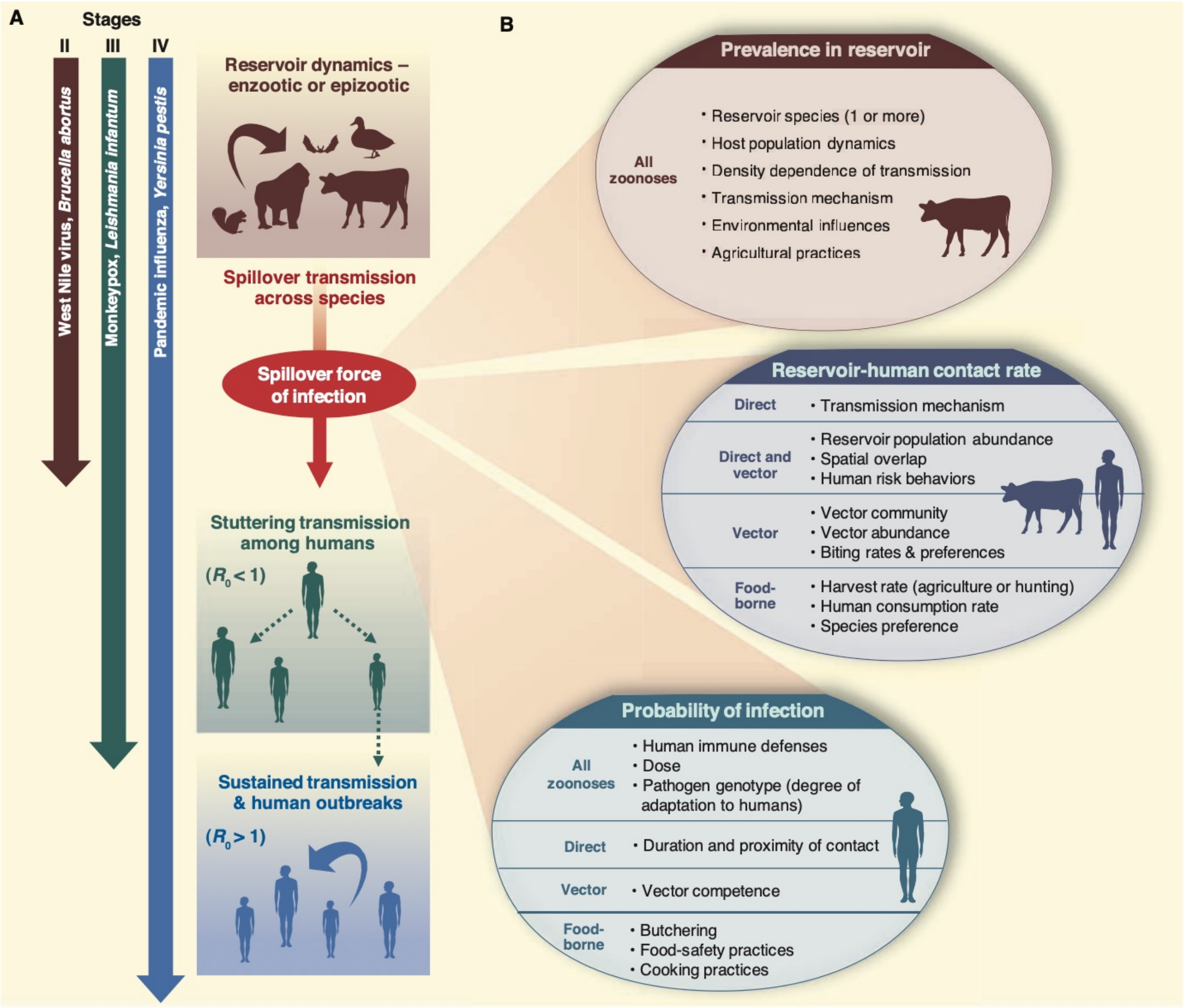

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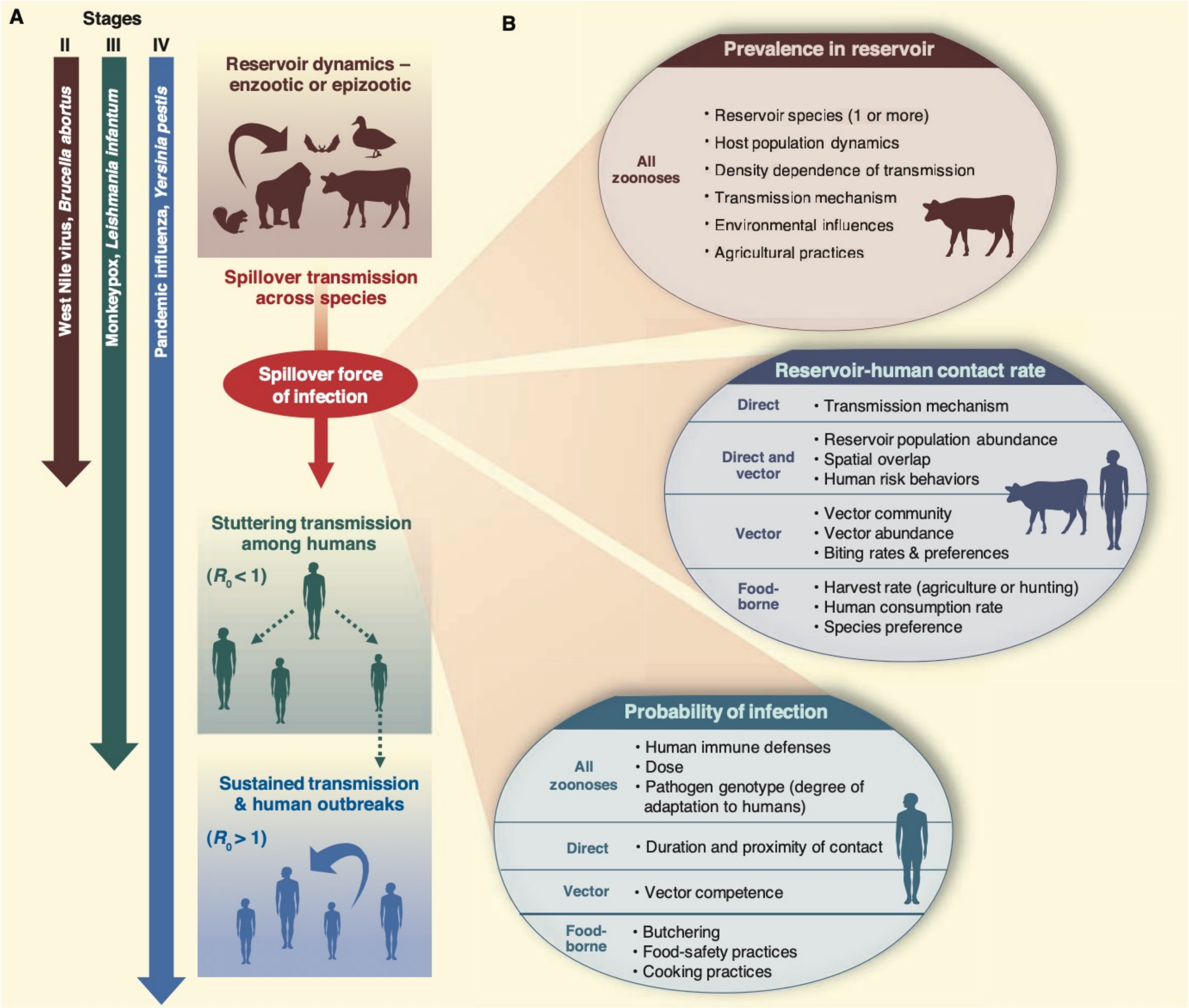
Keeling & Gilligan model λ_H for plague as proportional to the abundance of free-living infected fleas.



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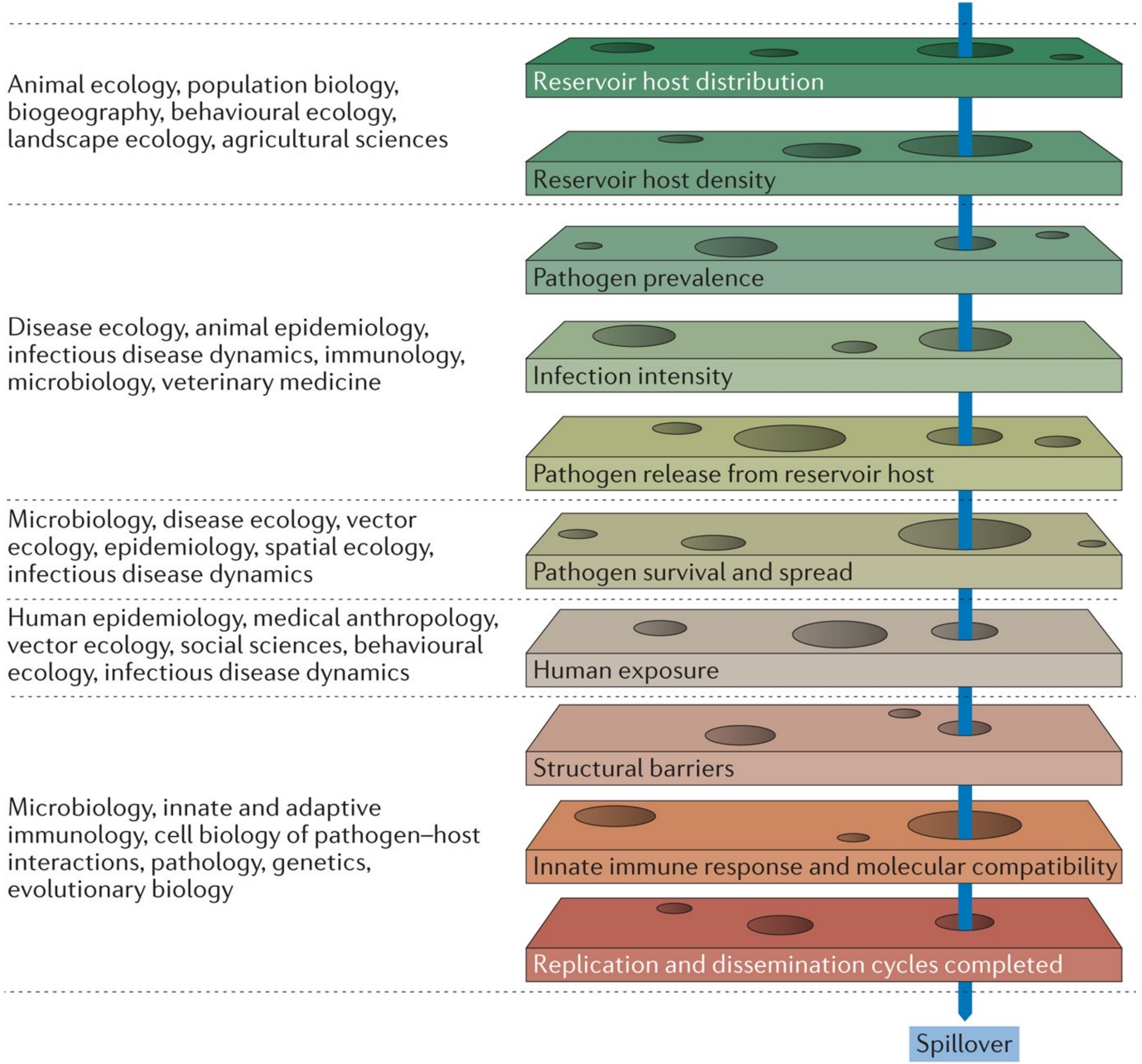
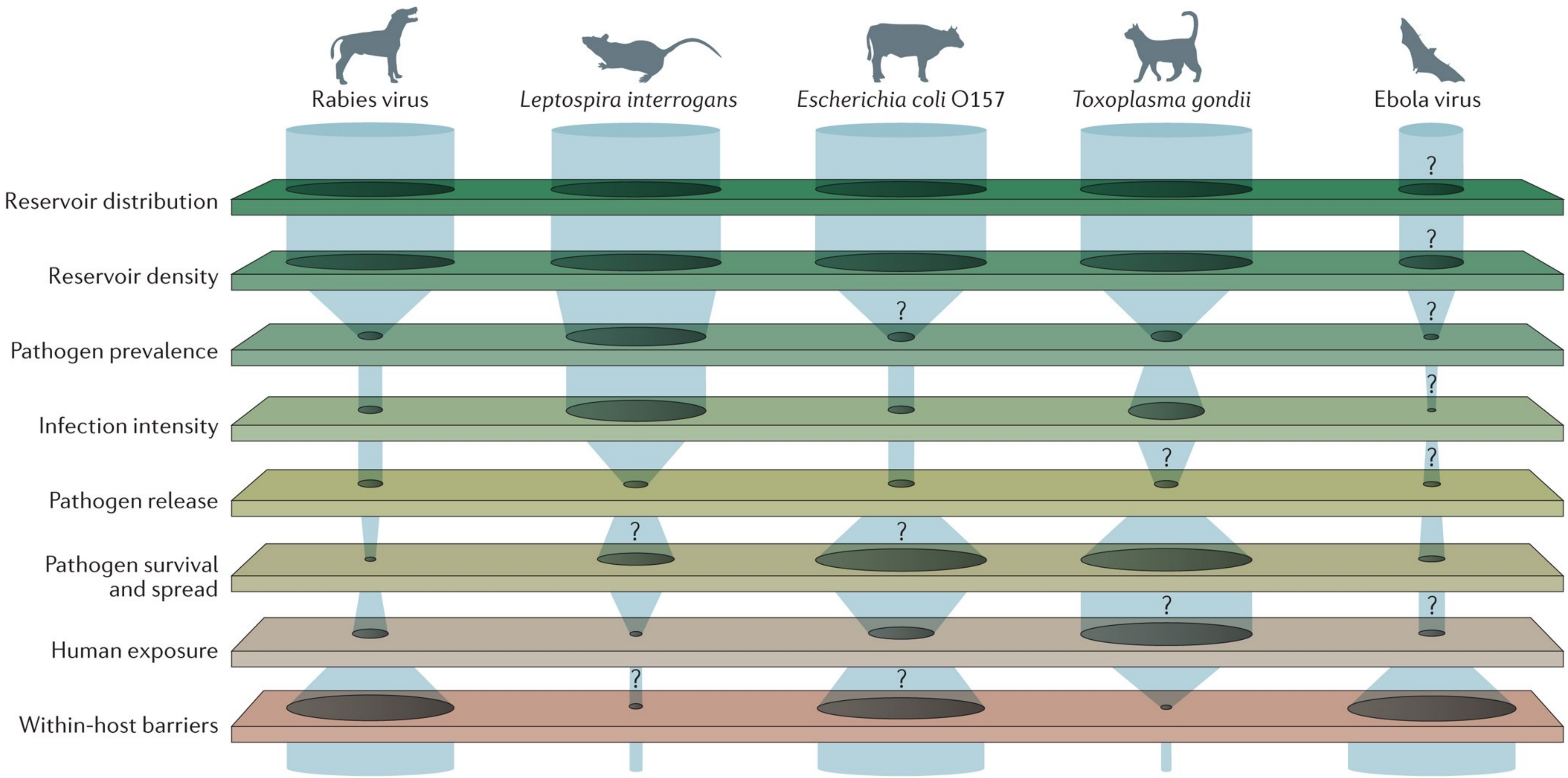
Zoonosis is a series of **improbable events**.

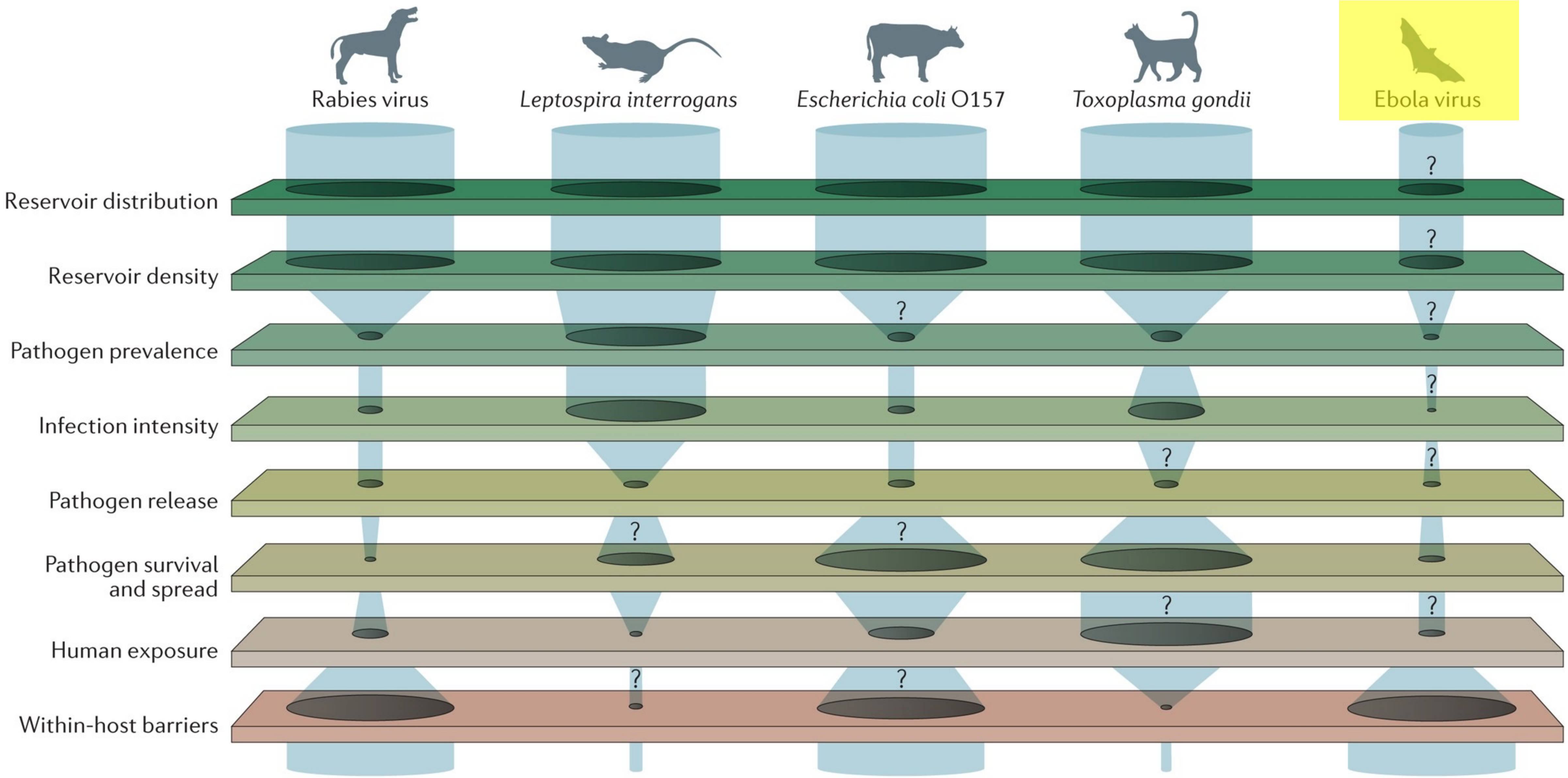


Plowright et al. 2017. *Nature Reviews Microbiology.*



Bottlenecks to spillover.

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

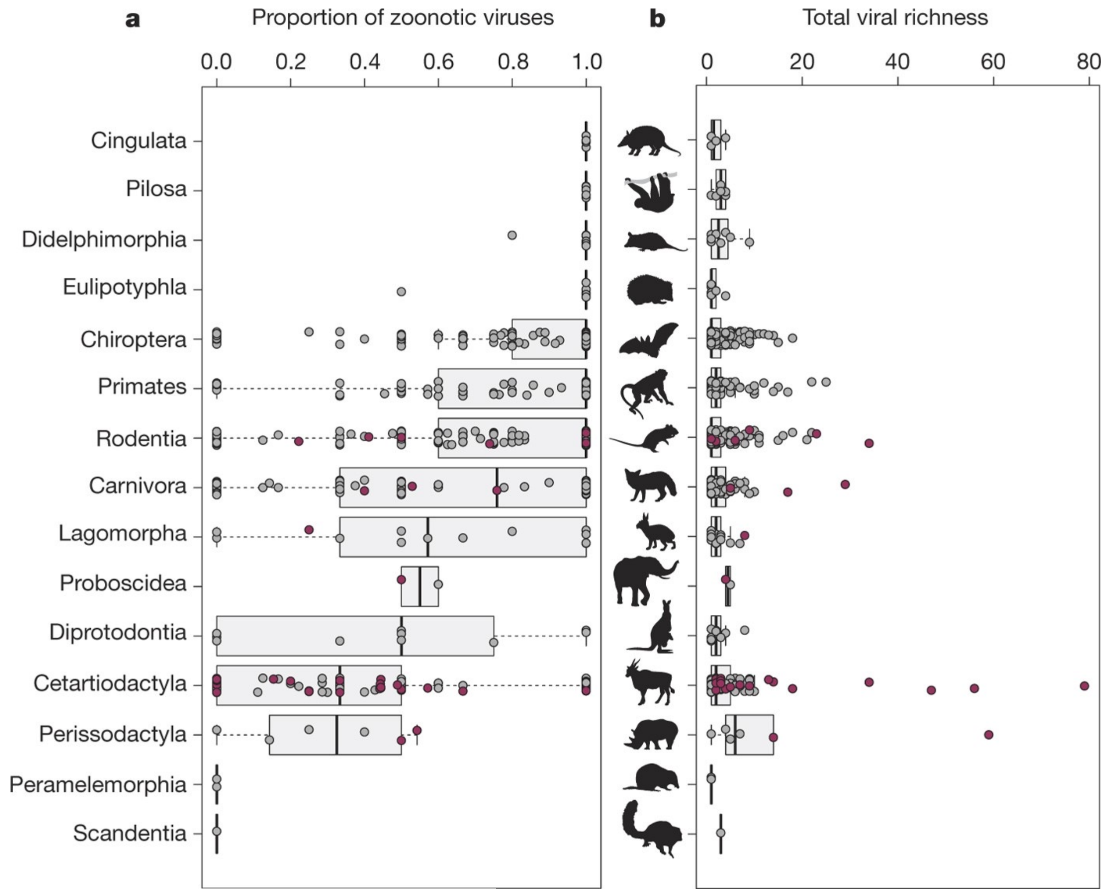


What's so special about bats?

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

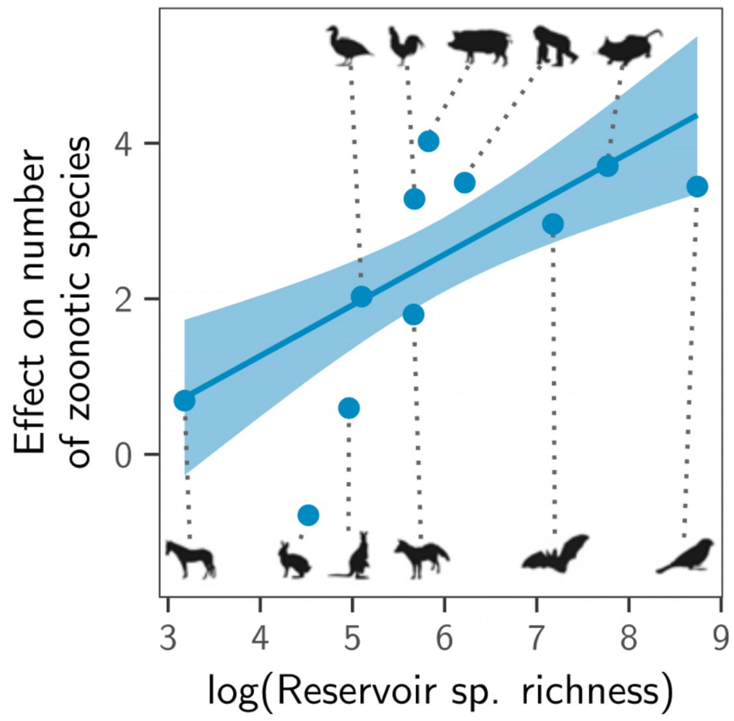
It is **debated** whether bats are reservoir hosts for **more** zoonotic viruses than other mammals.

Bats host a higher proportion of zoonotic viruses than other mammalian orders.

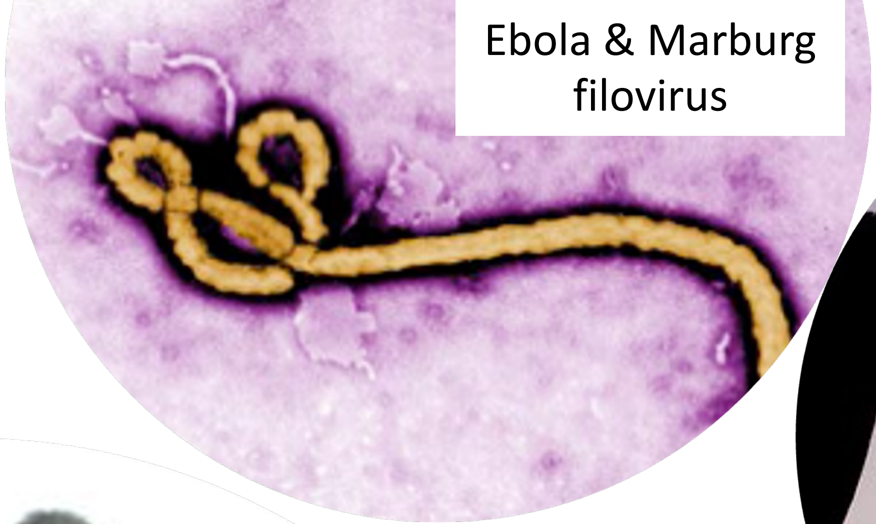


Olival et al. 2017. *Nature.*

The number of zoonotic viruses hosted by bats scales with the total number of bat species in existence (>1400 spp.; 20% of mammalian diversity).



Mollentze & Streicker. 2020. *PNAS.*



Ebola & Marburg filovirus

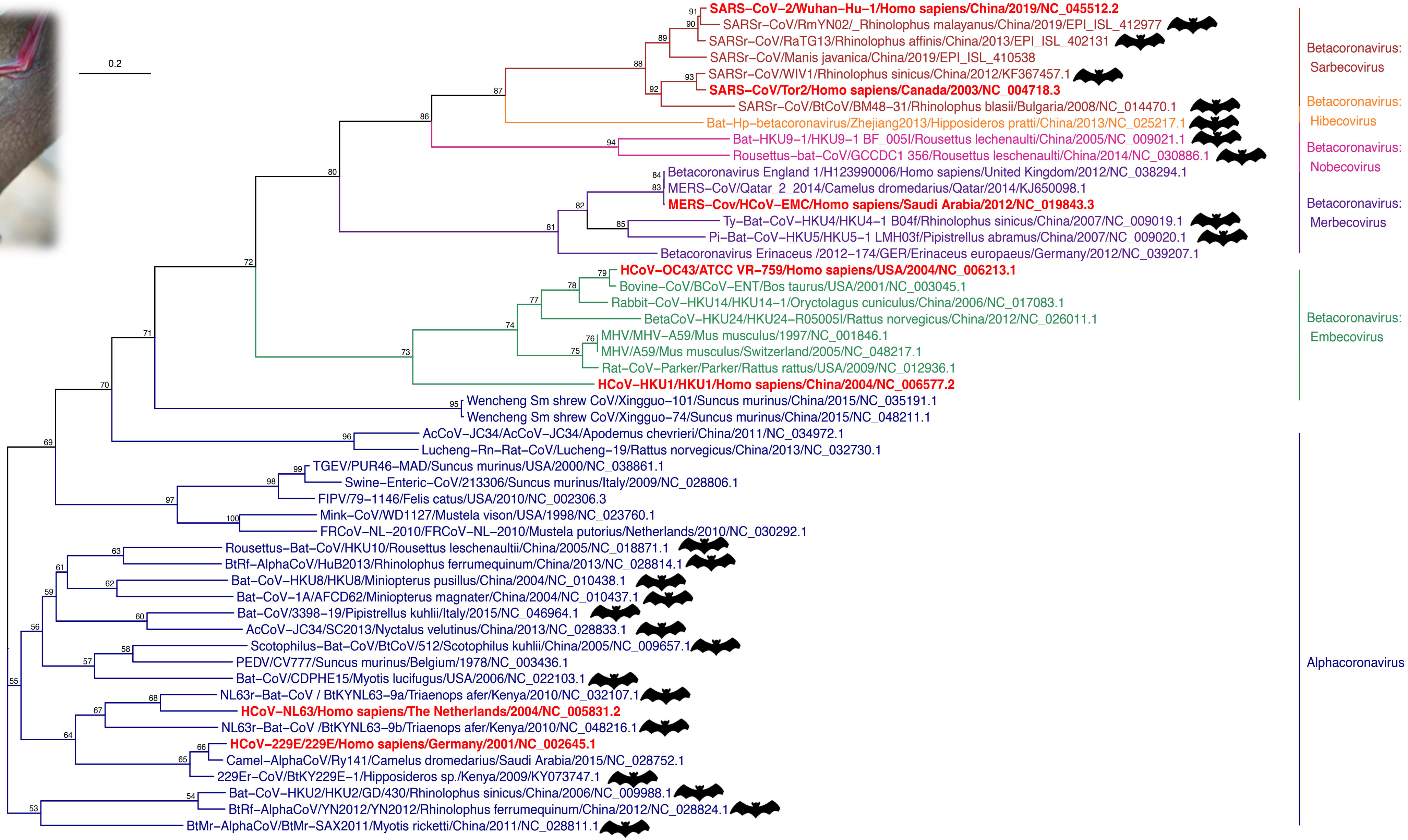
Hendra & Nipah henipavirus

Rabies lyssavirus

SARS, MERS, SARS-CoV-2 coronaviruses

But it is known that bats do act as natural **reservoirs and maintenance hosts** for the majority of the world’s **most virulent zoonotic viruses**.

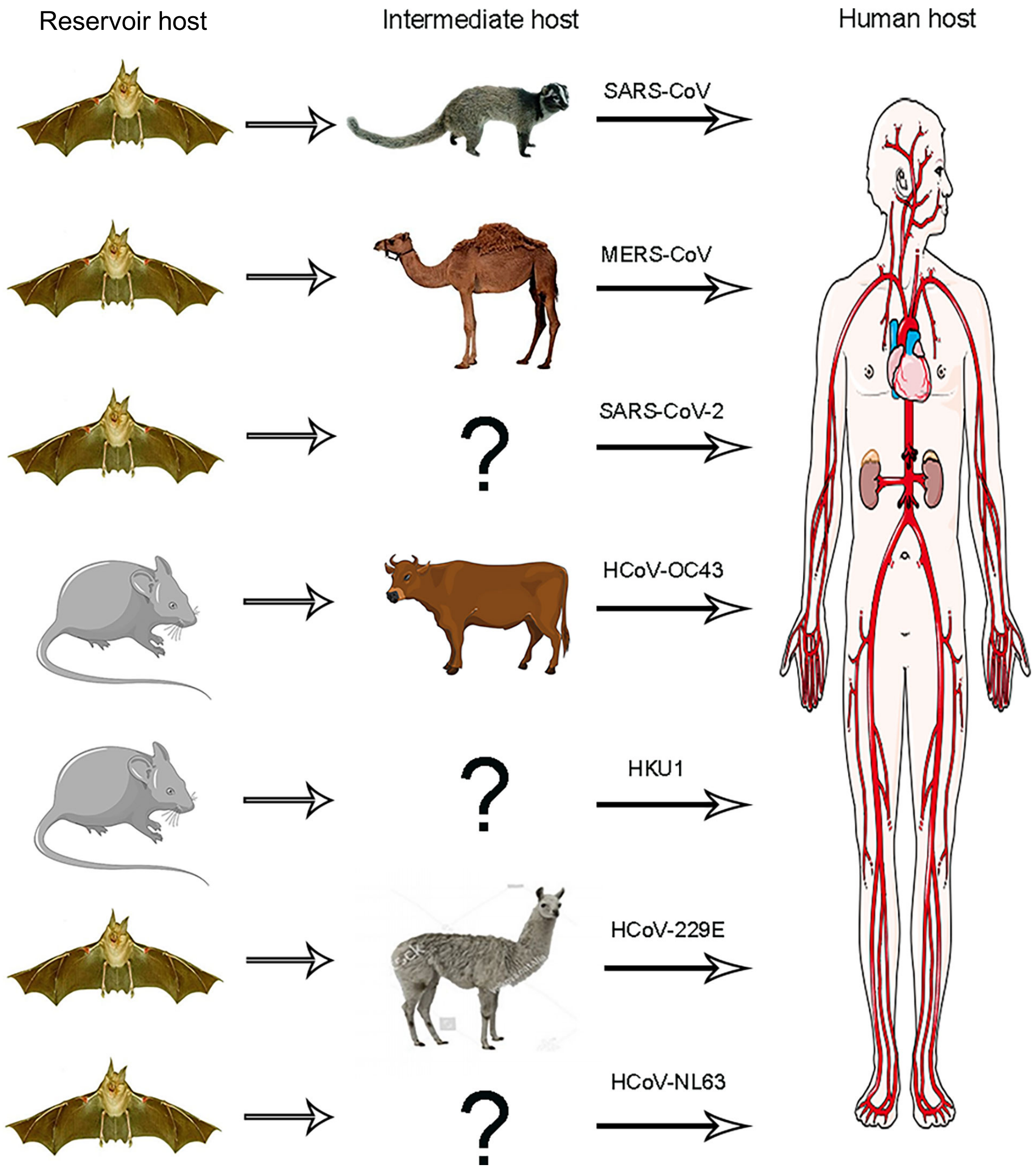
Bats are natural reservoir hosts for several subgenera of ***Alpha– and Betacoronaviruses***.
Rhinolophus* spp. bats** are reservoirs for the SARS-like ***Sarbecoviruses.



7 Zoonotic CoVs

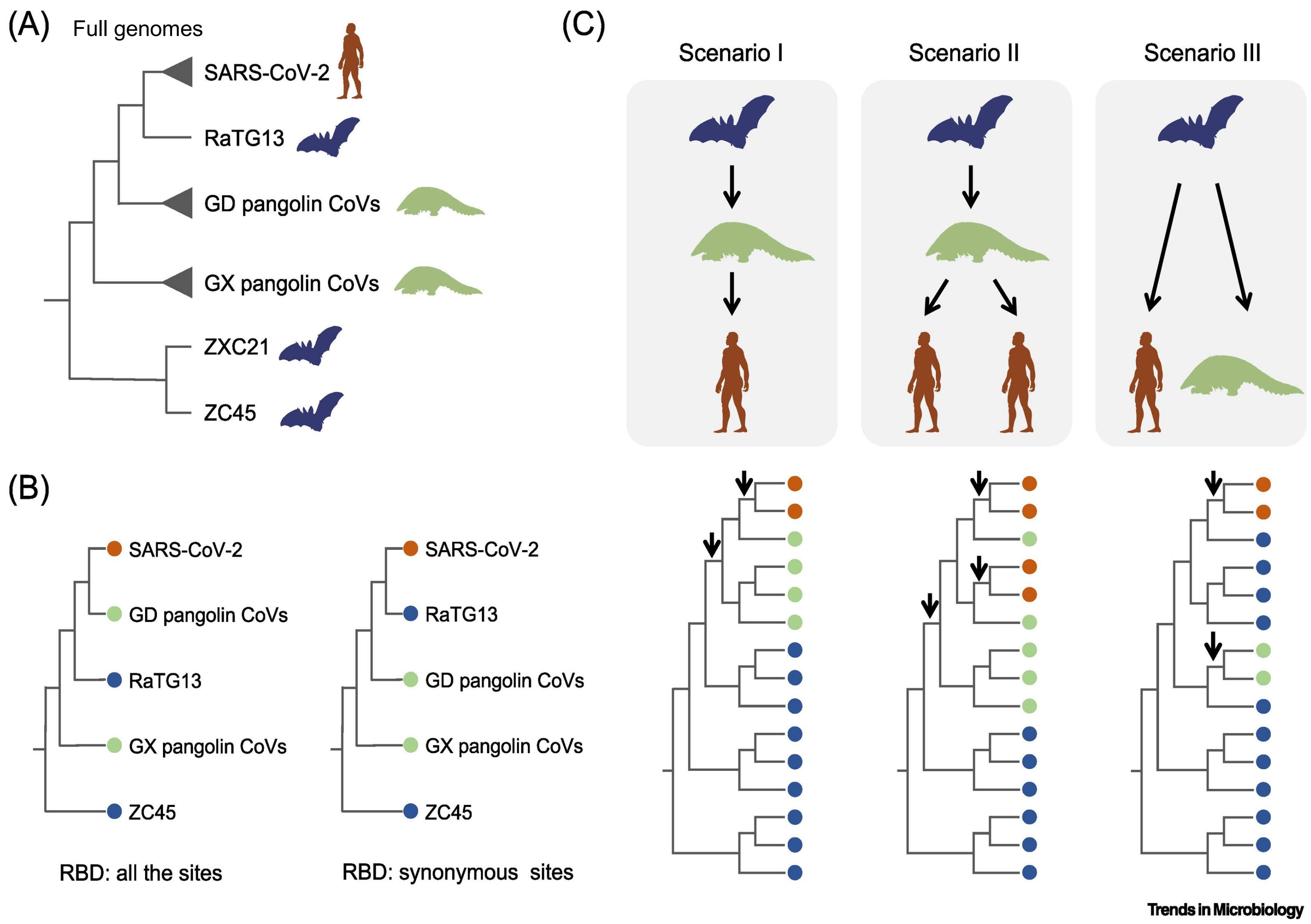
Ravelomanantsoa et al. 2020. *Emerging Topics in Life Sciences*.

Many CoVs transmit (and adapt) via **intermediate hosts** prior to infecting humans. An intermediate host for SARS-CoV-2 has **not yet been identified**.



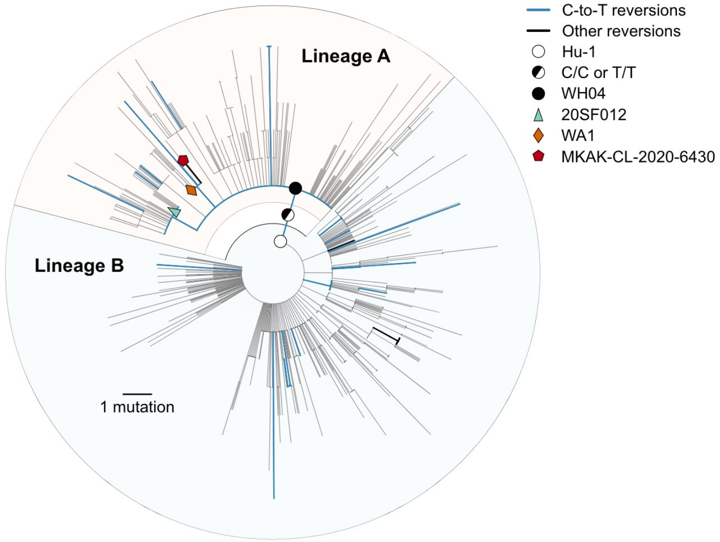
Ning et al. 2021. *Front. Cell. Infect. Microbiol.*

Bats likely sourced infections to both humans and pangolins in **disparate spillover events** (Scenario III)

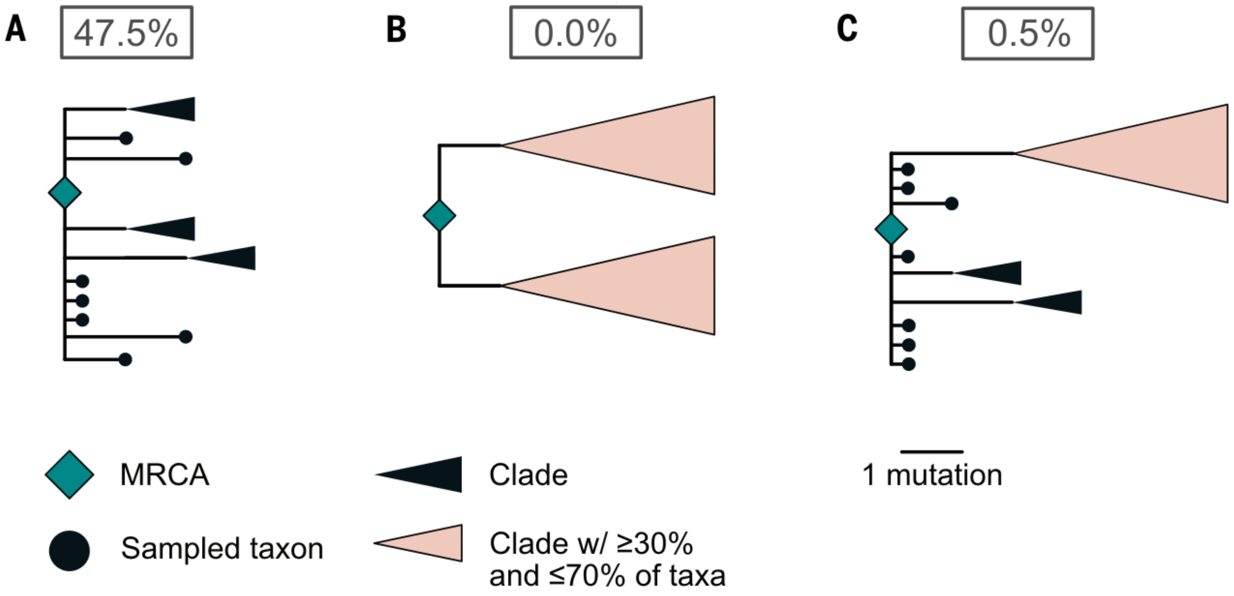


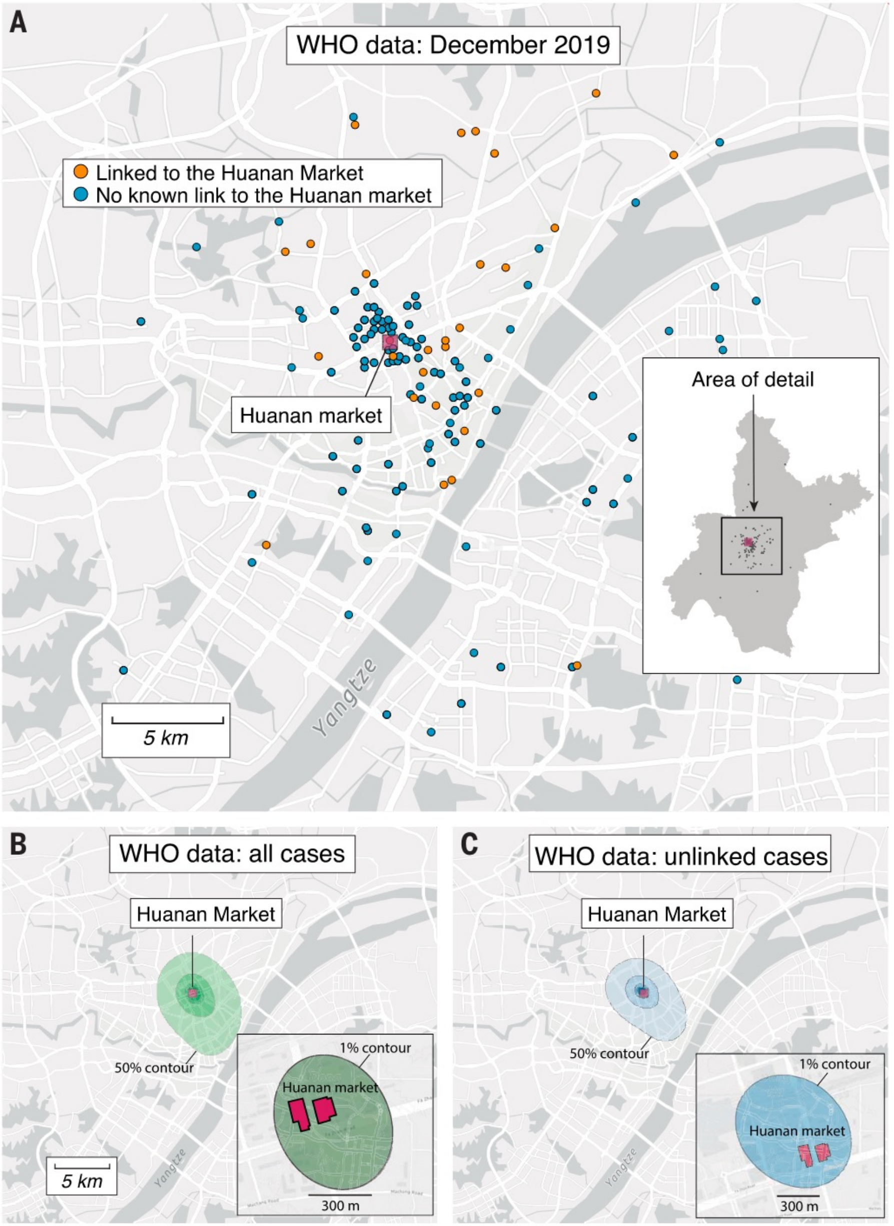
Han 2020. *Trends in Microbiology.*

Phylogenetic analysis suggests multiple spillover origins of SARS-CoV-2 to humans. Both lineages appear to have emerged from the Huanan Seafood Market.



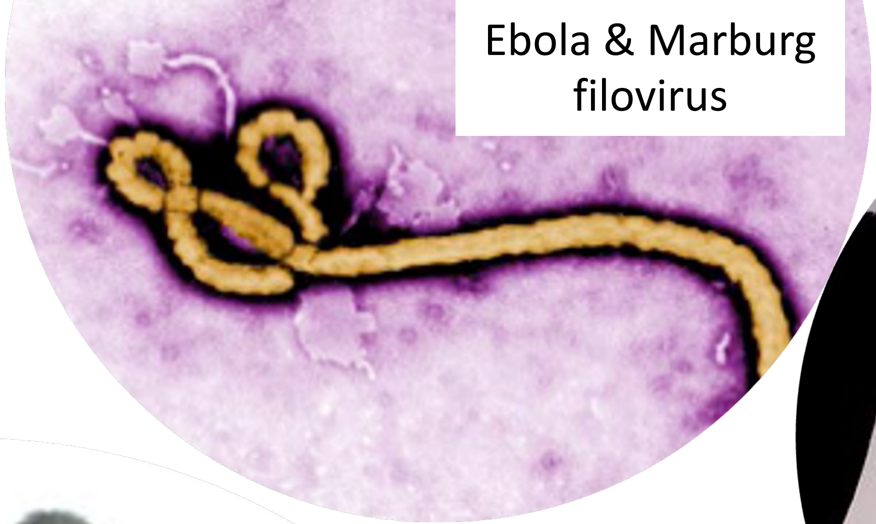
Polytomy: a node on a phylogeny where more than 2 lineages descend from an ancestral lineage





Pekar et al. 2022. *Science.*

Worobey et al. 2022. *Science.*



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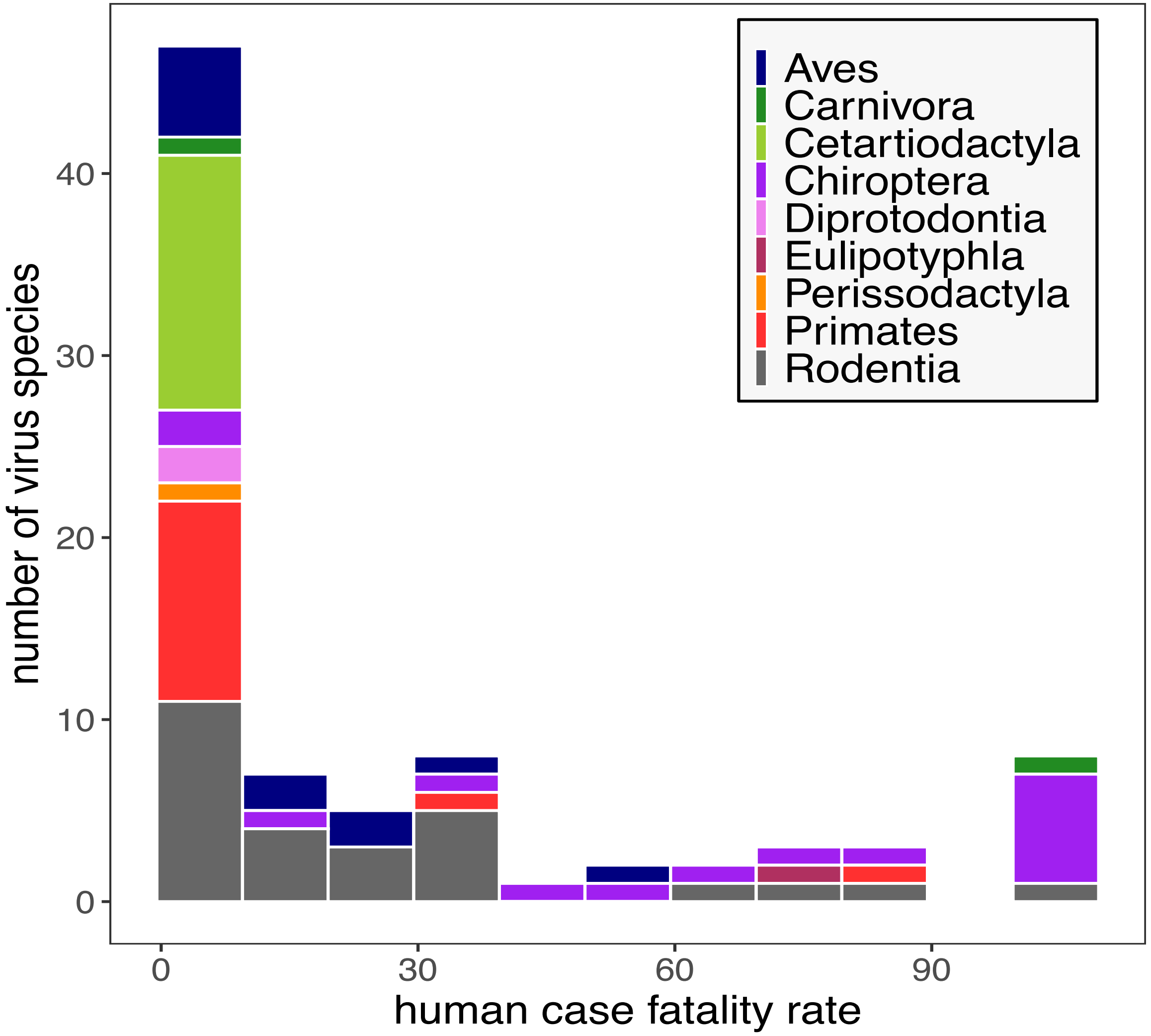
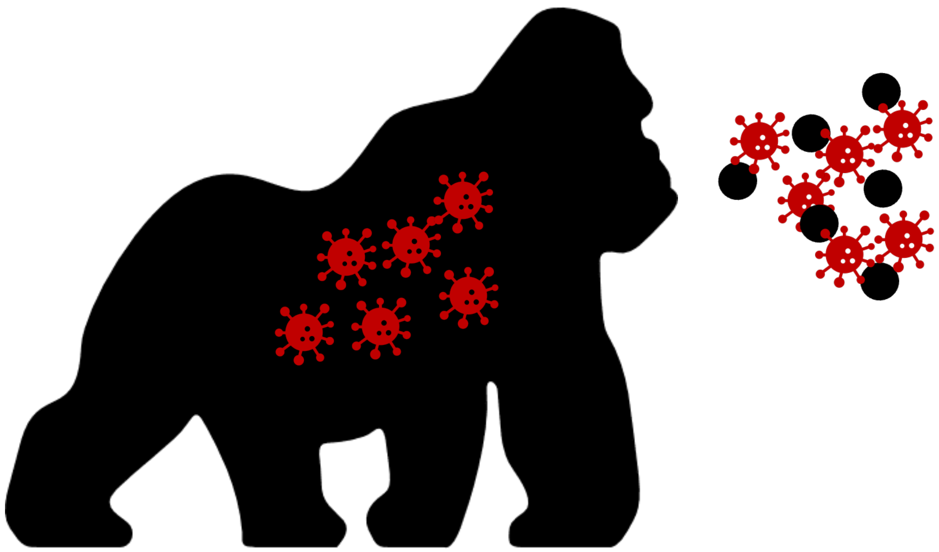


Guth et al. 2019. *Phil Trans Roy Soc.*
Guth et al. 2022. *PNAS.*

Why do pathogens make us sick?

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



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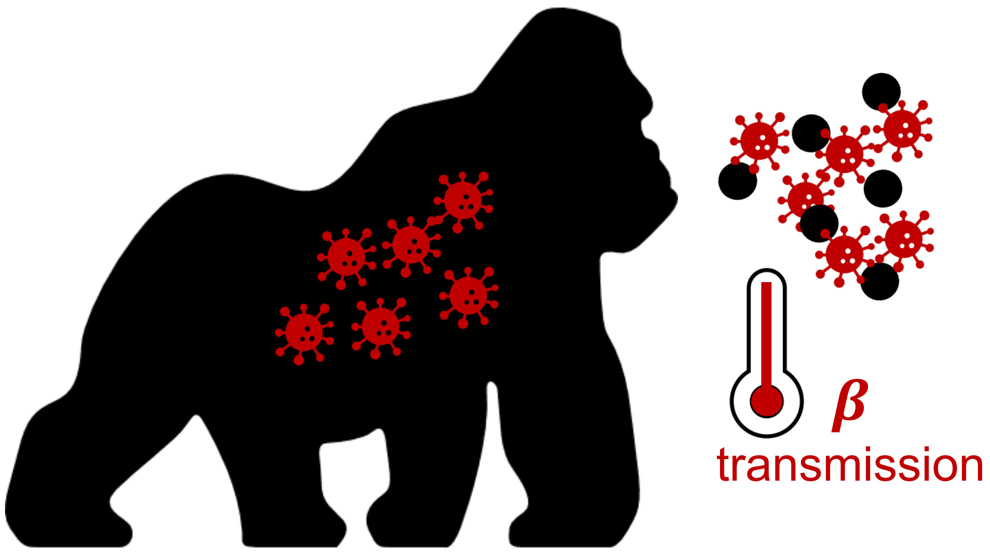

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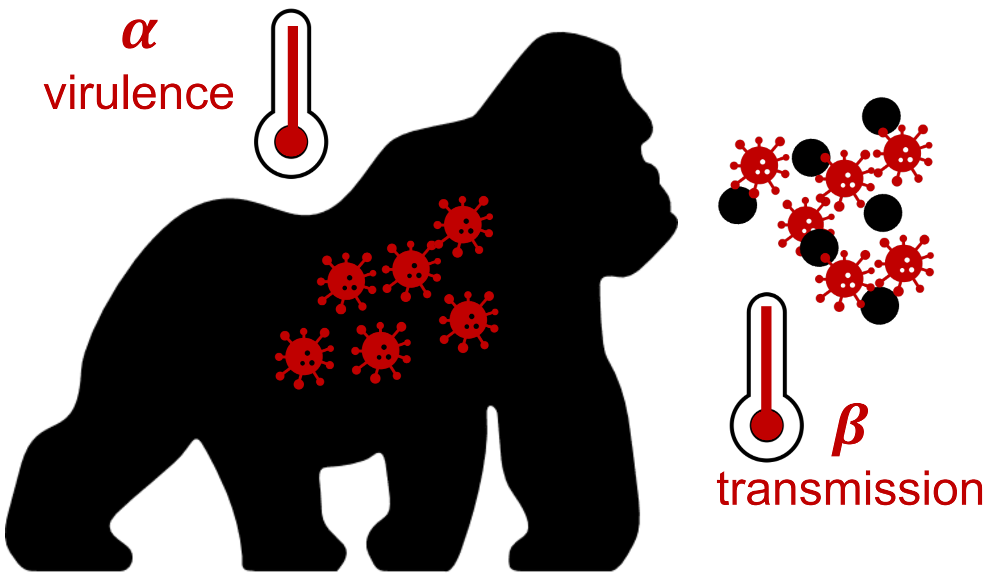

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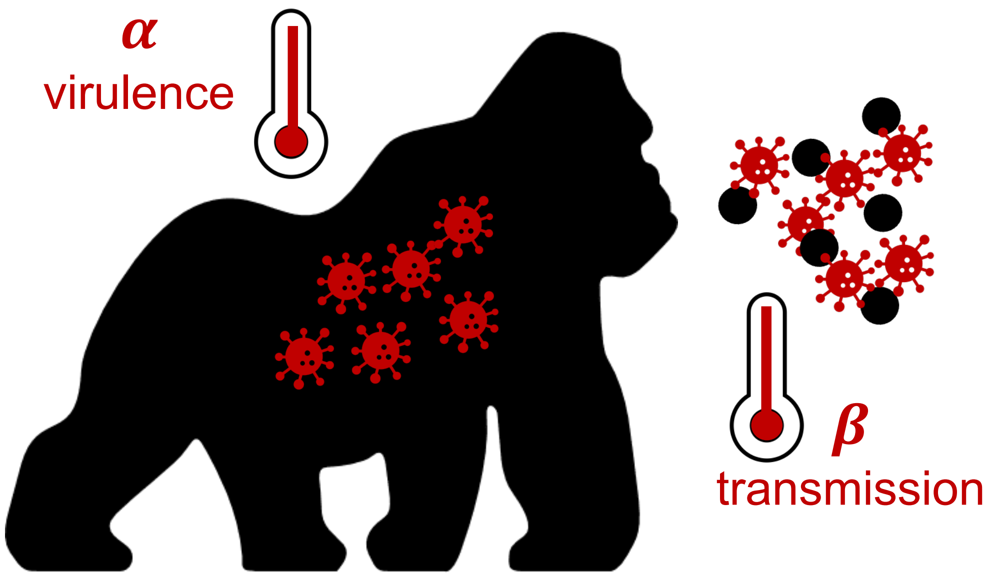

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Virulence, then, is a by-product of a pathogen's need to transmit for reproduction!

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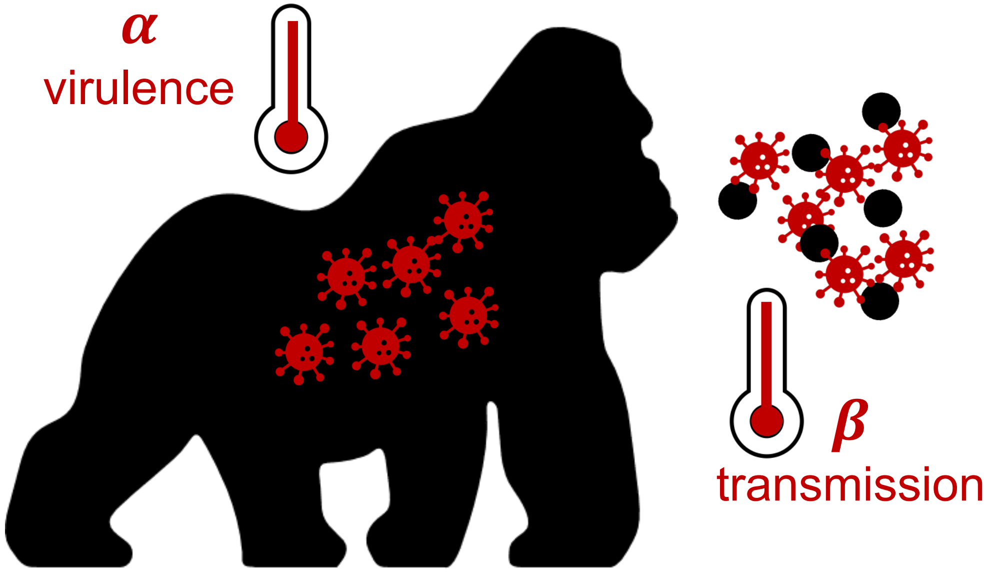

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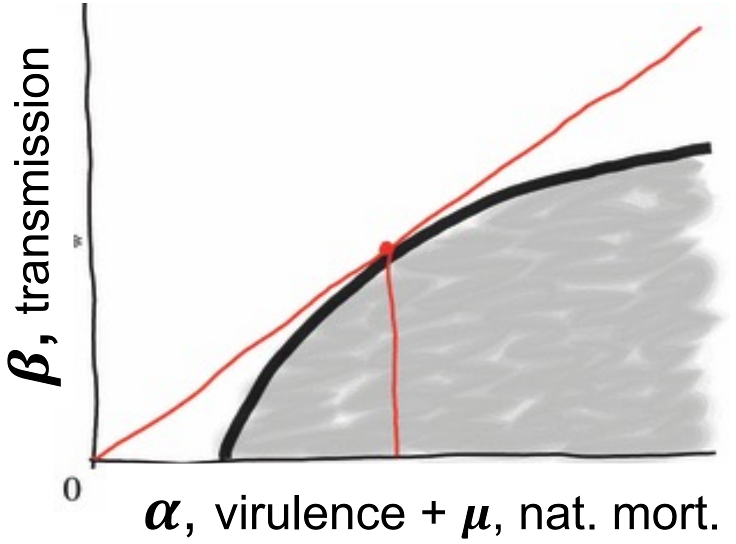
As a result, we predict the evolution of "**optimal virulence**."



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

infections created

infections lost

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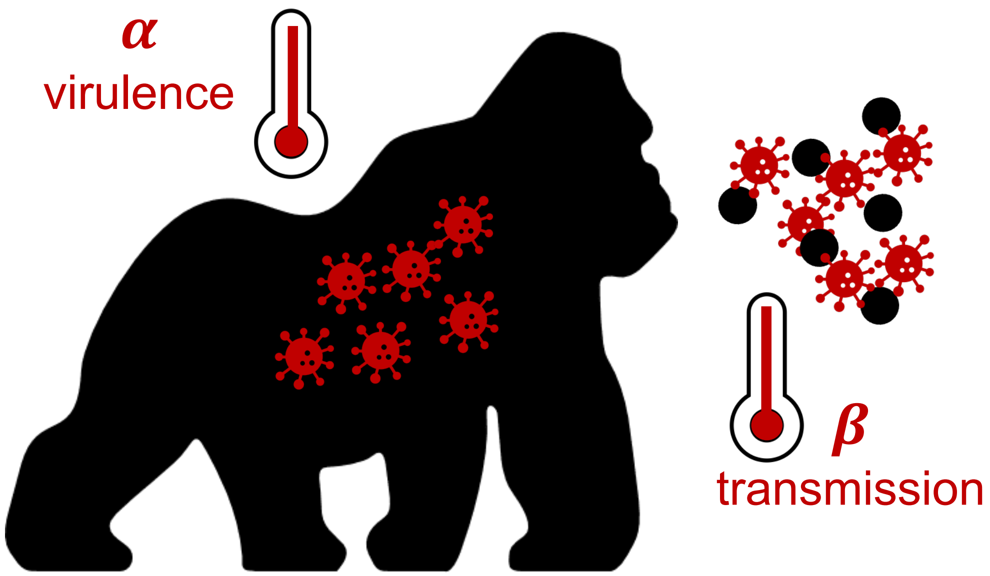

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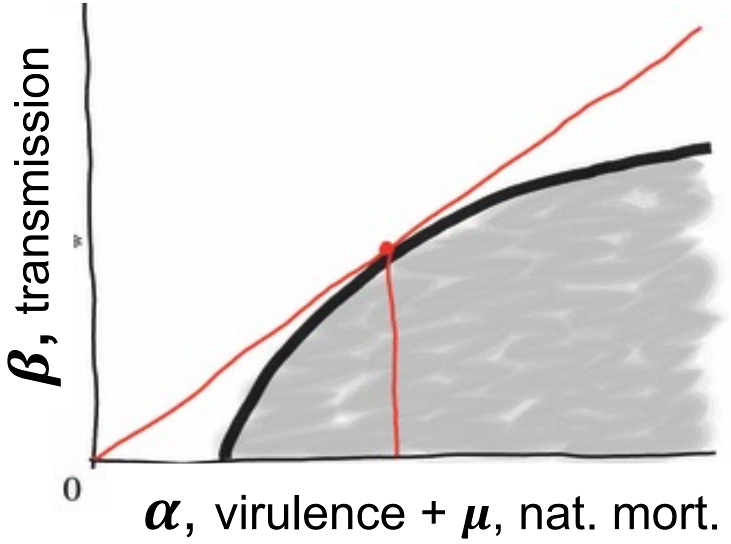

Note that originally Anderson and May (1982) represented this link to virus density as acting on the disease recovery rate, though it is now more commonly expressed as a function of virulence!

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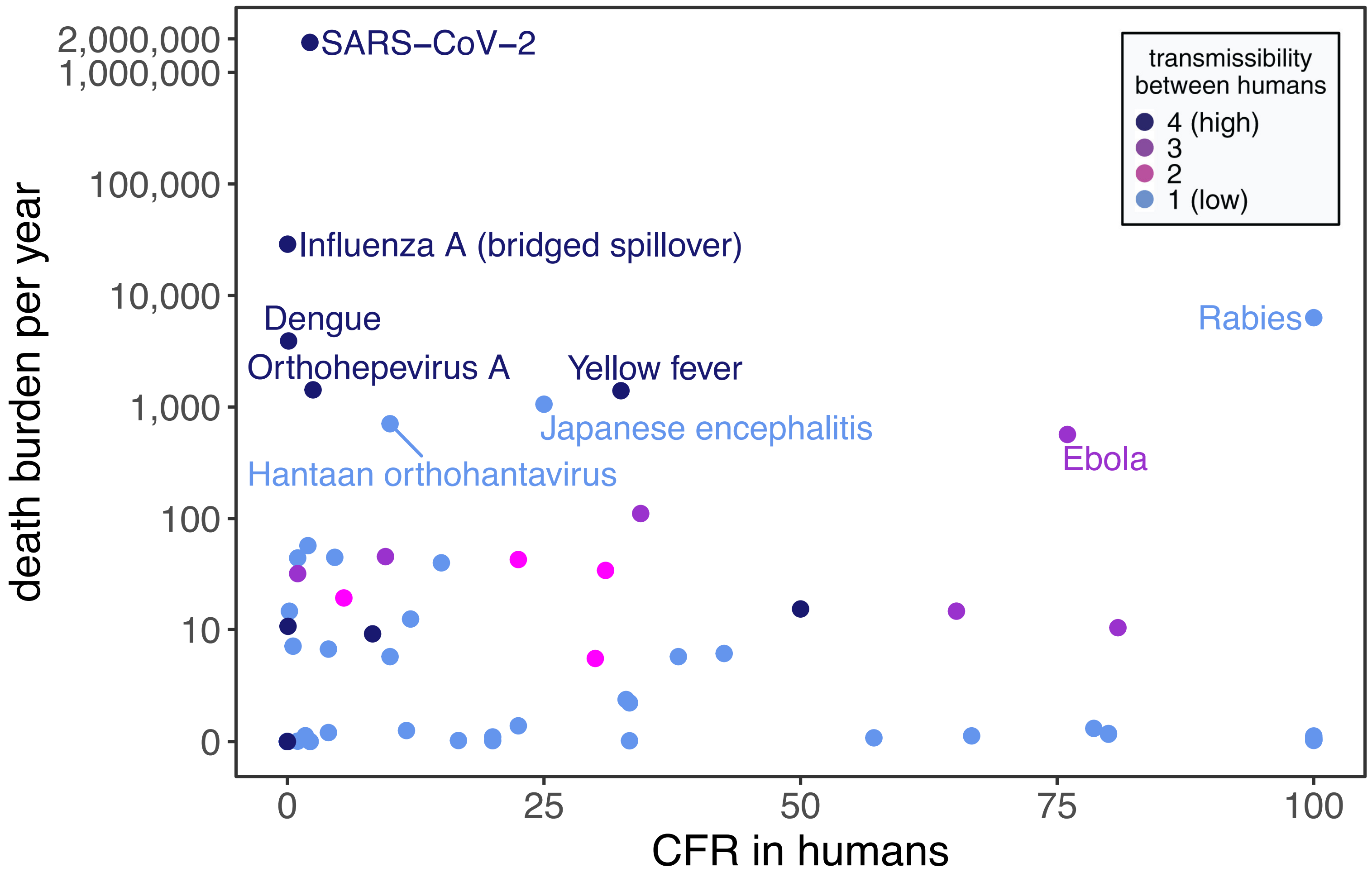

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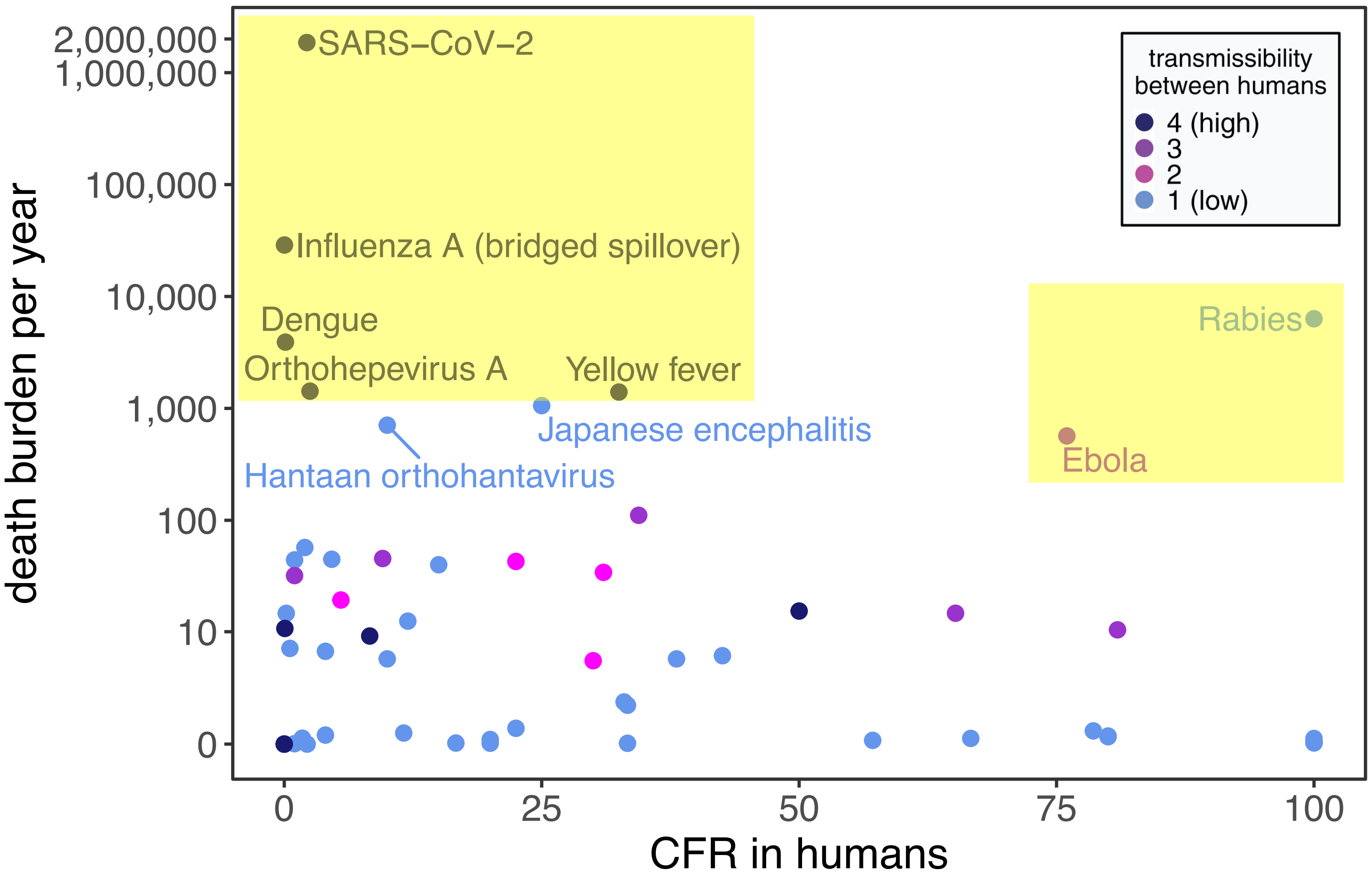

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The **virulence case study** of rabbit Myxoma virus

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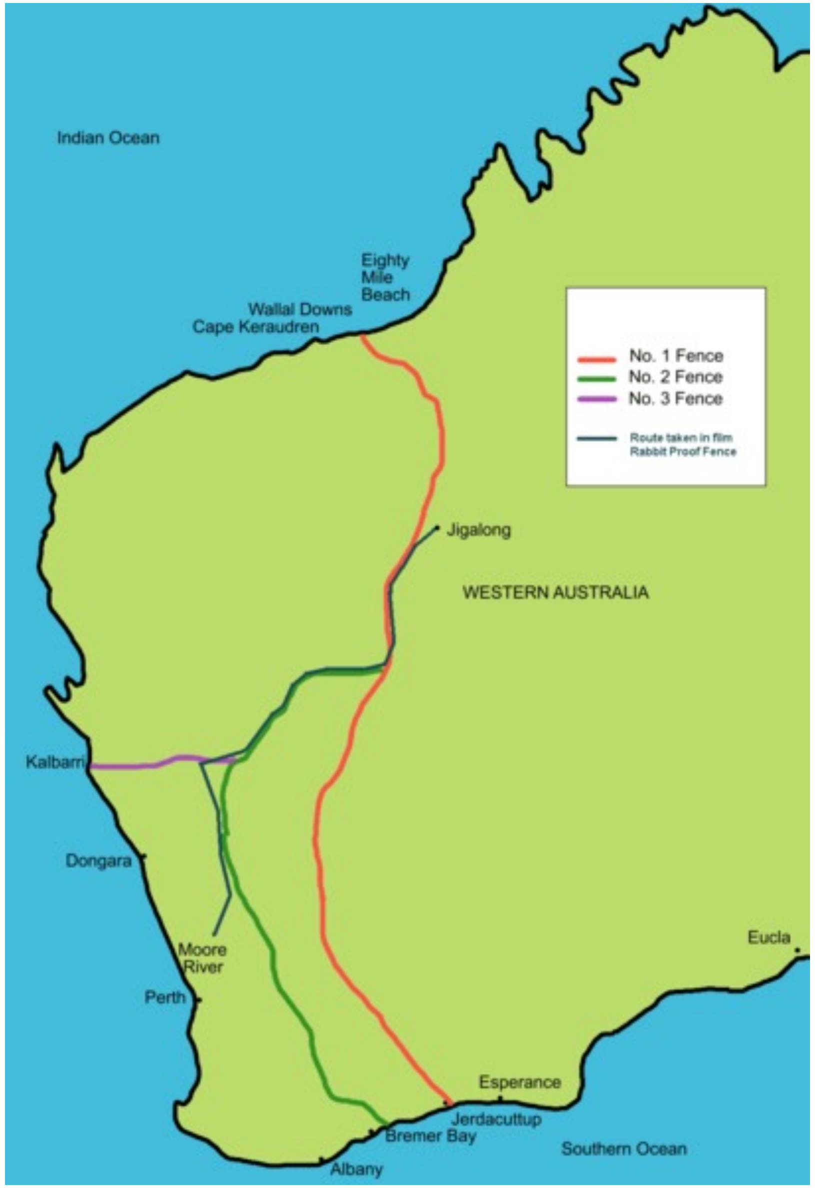
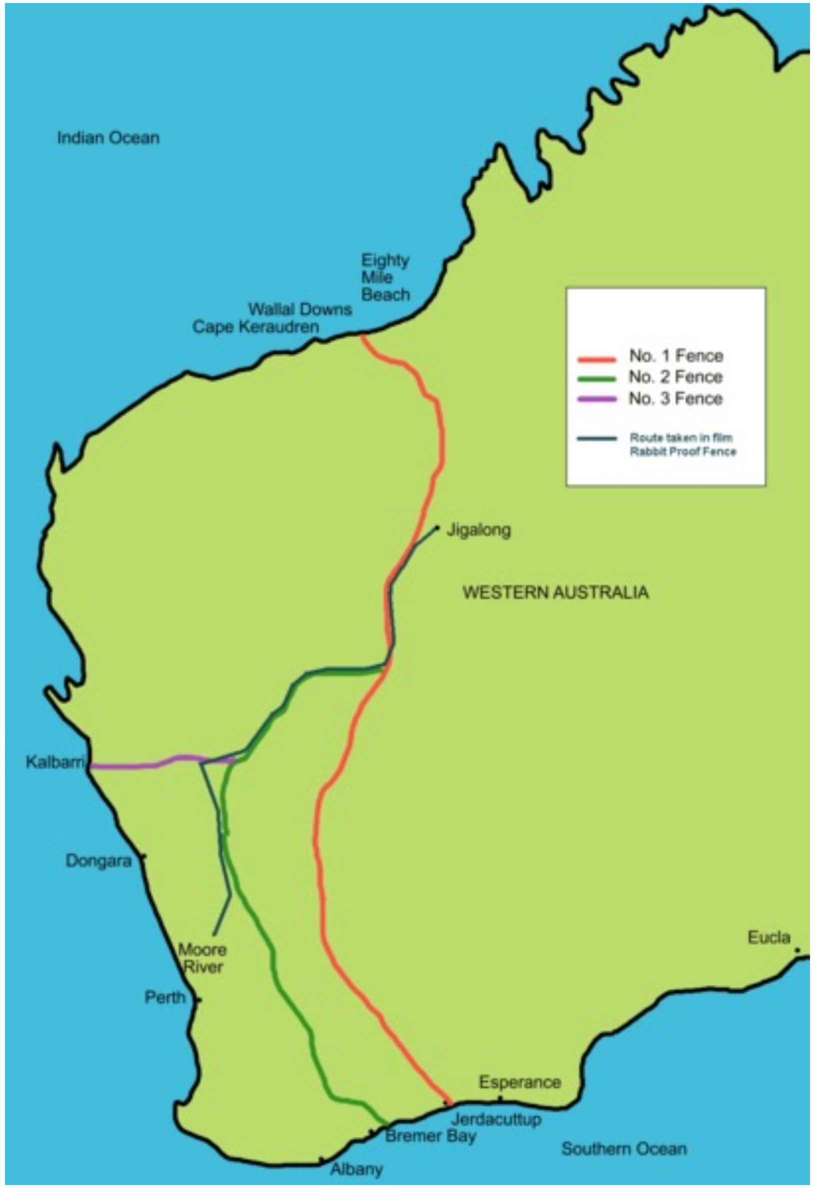
- 1788: European rabbits brought to Australia as a food source
- Rabbits quickly became feral and numbers soared.
- 1901: Australia constructed the famous "rabbit-proof fence" to attempt to keep rabbits out of agriculture in the West.



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.

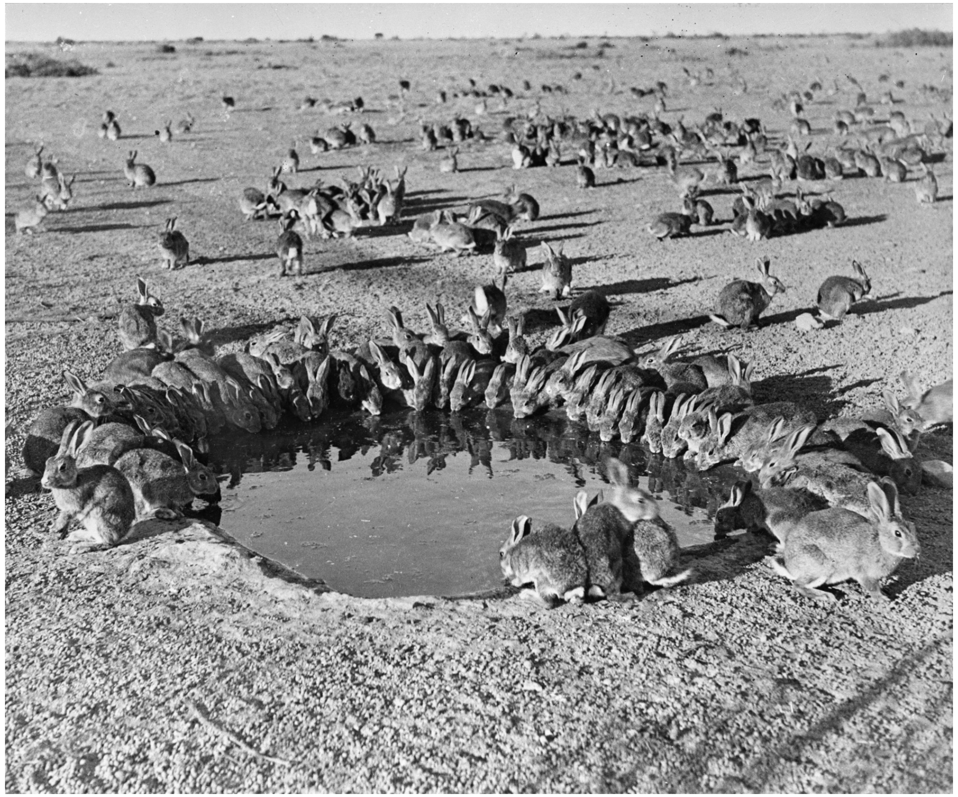
TABLE 4. THE VIRULENCE OF STRAINS OF MYXOMA VIRUS RECOVERED FROM THE FIELD IN AUSTRALIA BETWEEN 1951 AND 1981, EXPRESSED AS PERCENTAGES

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.

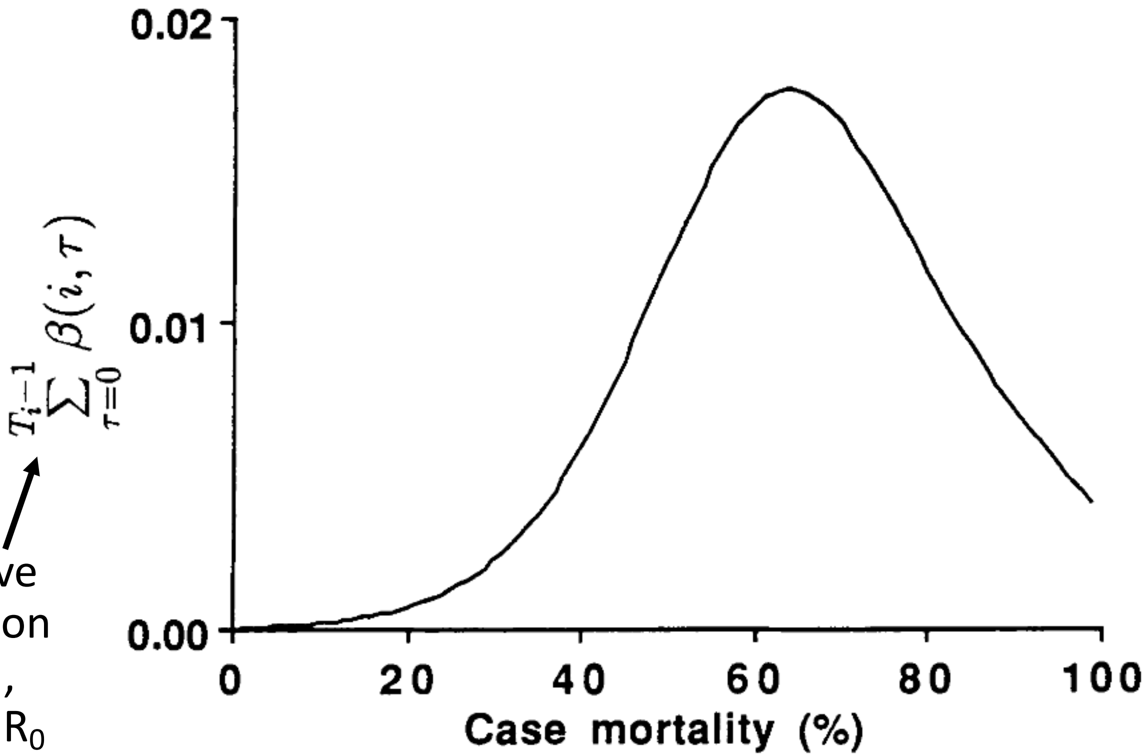
Fenner 1983. *Proc Roy Soc B*

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

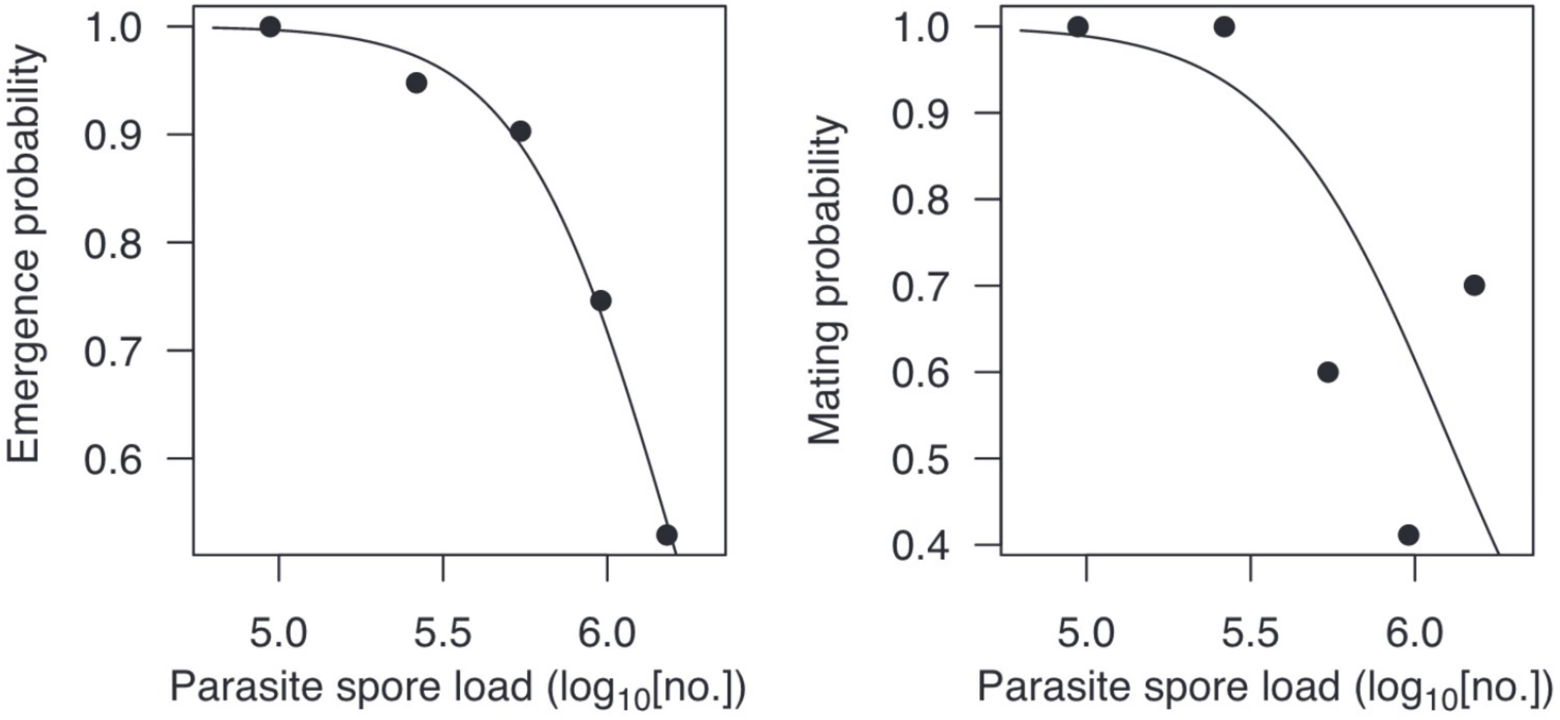
Simon A. Levin
Section of Ecology and Systematics, Corson Hall, Cornell University, Ithaca, New York 14853 USA

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**).



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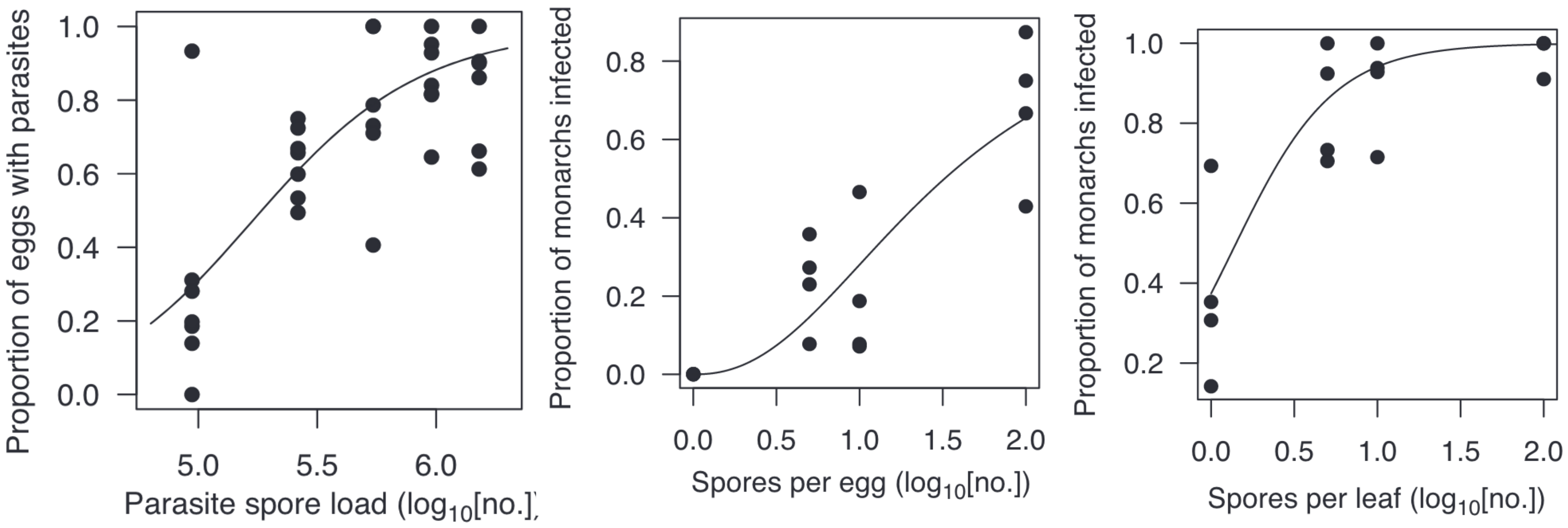

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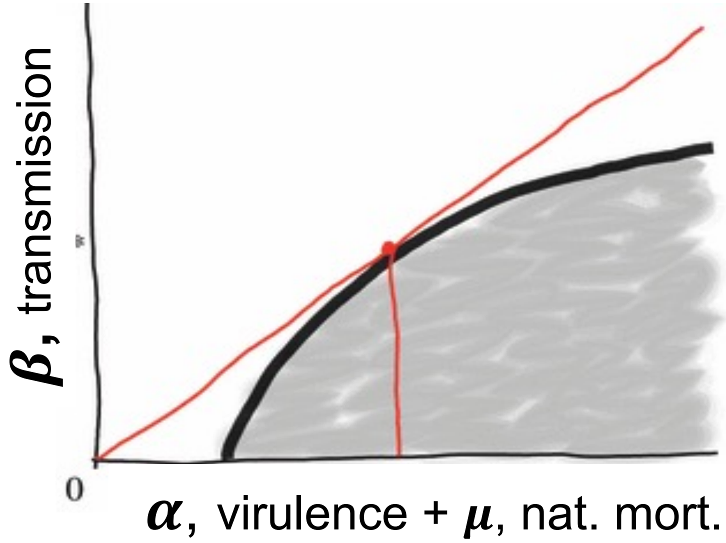
- **Parasite fitness is calculated to be maximized at intermediate spore load.**



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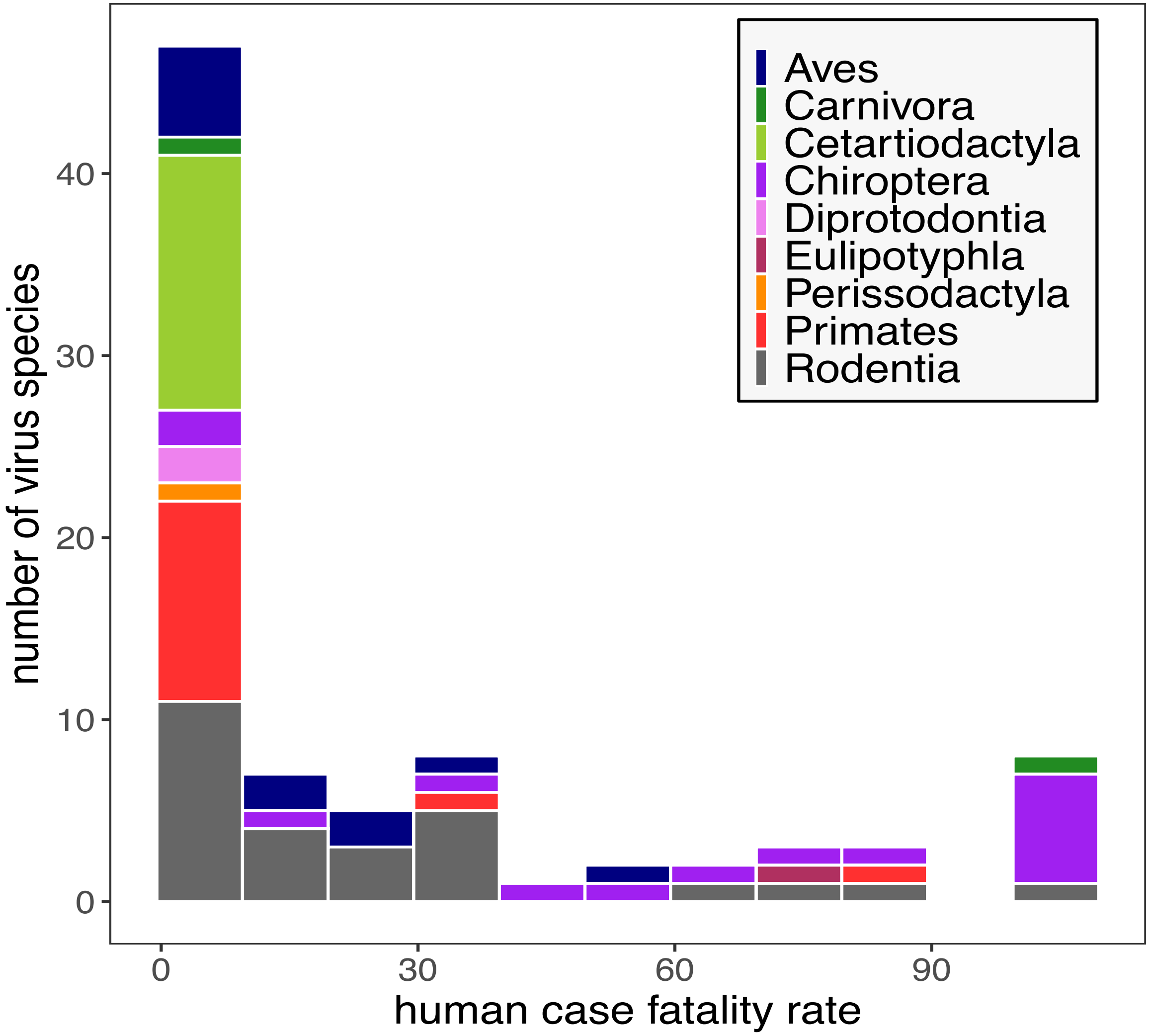
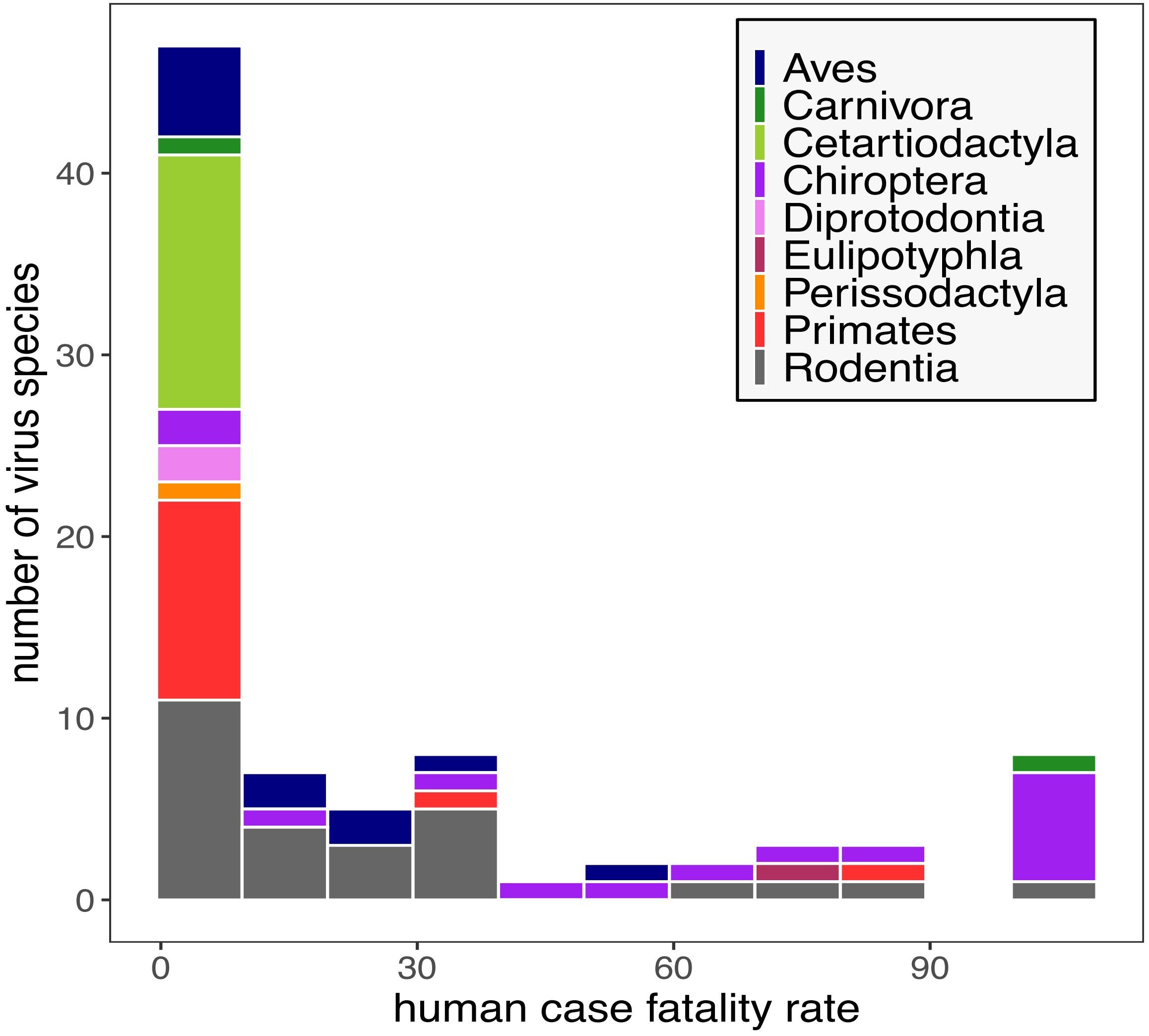
Why then are bat viruses so virulent [**to humans**]?



Guth et al. 2019. *Phil Trans Roy Soc.*
Guth et al. 2022. *PNAS.*

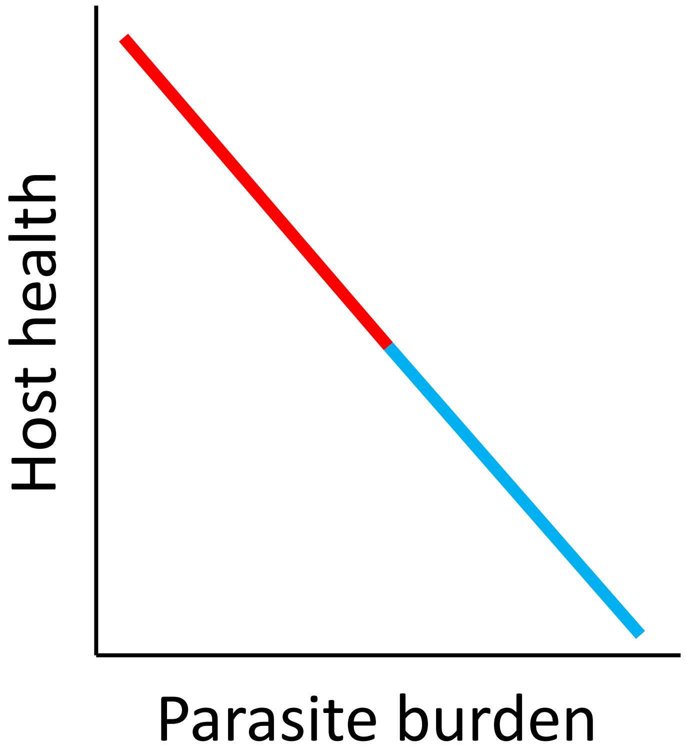
Why then are bat viruses so virulent [**to humans**]?

Mismatch of optimal virulence evolved in the **reservoir host** and a lack of **viral tolerance** in the spillover (human) host.

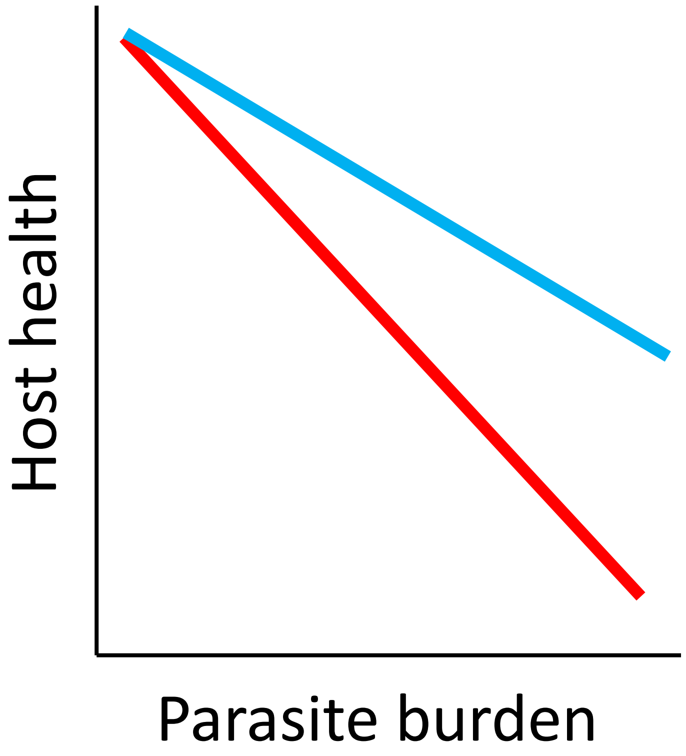


Guth et al. 2019. *Phil Trans Roy Soc.*
Guth et al. 2022. *PNAS.*

Bats appear **highly tolerant** of virus infection!
(They host viruses without experiencing disease)



resistance



tolerance

Boots 2008. *TREE.*
Raberg et al. 2007 *Science.*

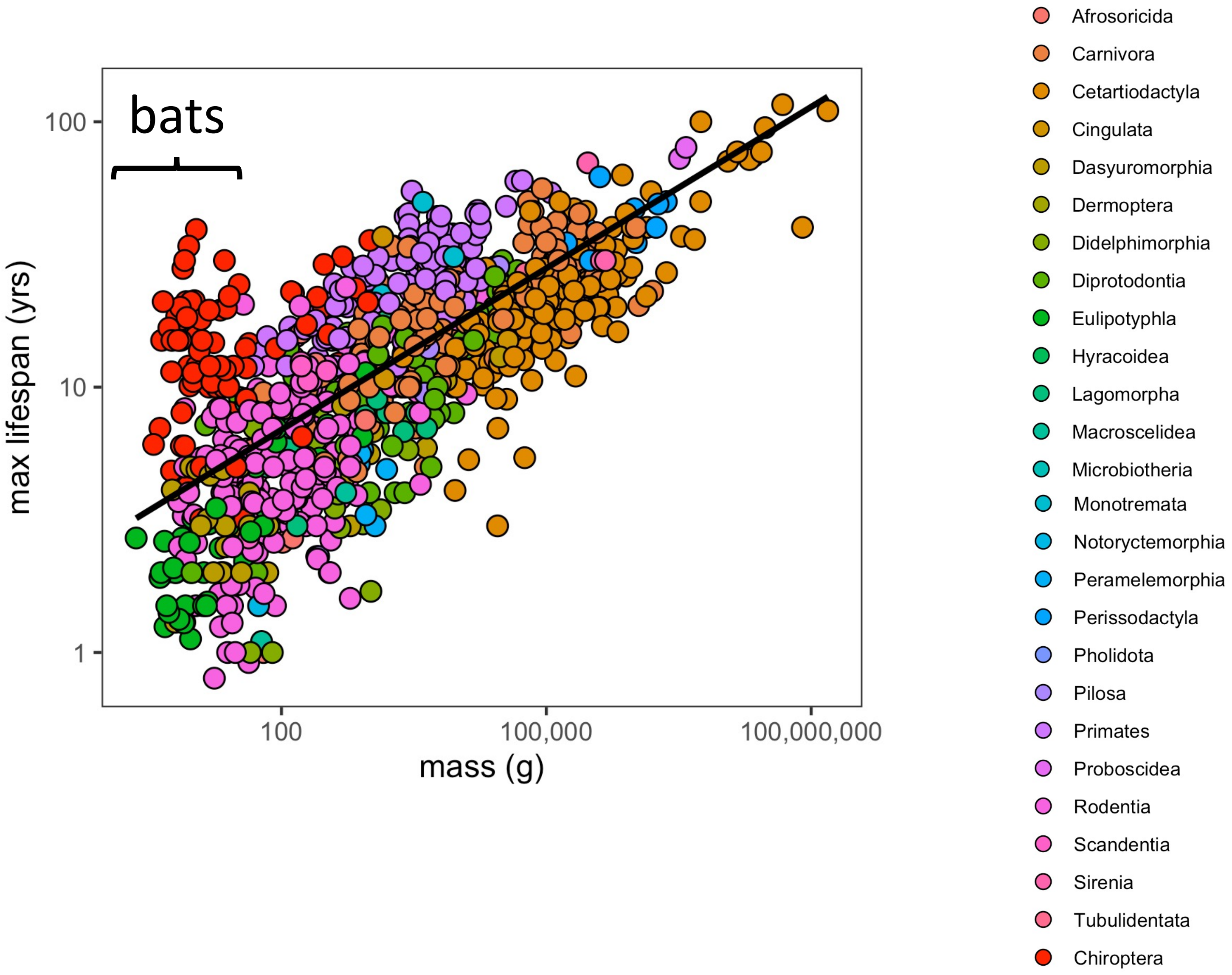
Virus **tolerance** is linked to the **evolution of flight**.

Bats are the only **flying mammals**.

Flight is **more metabolically expensive** than any form of terrestrial locomotion.

O'Shea et al. 2014 *EID*
Brook and Dobson 2015 *Trends Micro*

Bats have **elongated lifespans** despite **high metabolic rates**.



Jones et al. 2009 *Ecology*

Brook et al. In Revision. *PLoS Biology.*

We hypothesize that:

unique **anti-inflammatory** pathways

evolved to **enable flight**

Brook and Dobson 2015 *Trends Micro*

We hypothesize that:

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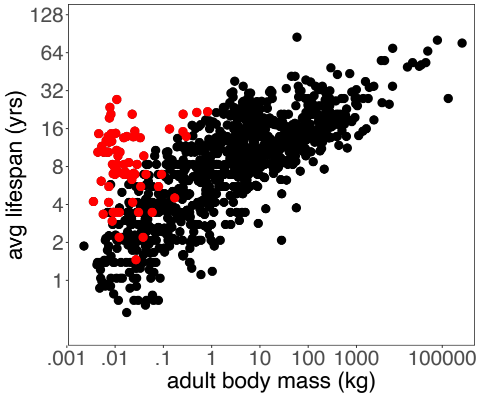
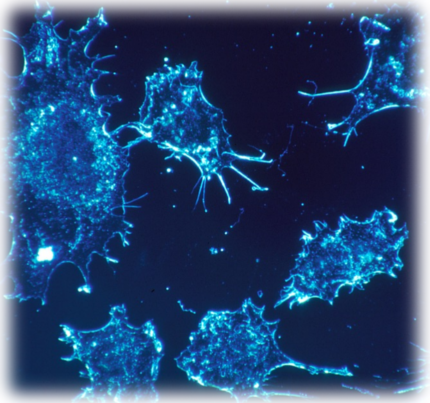
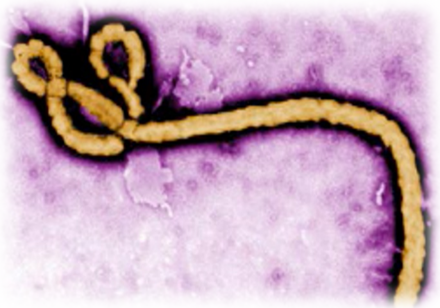
bat **longevity**



anti-**cancer** defenses

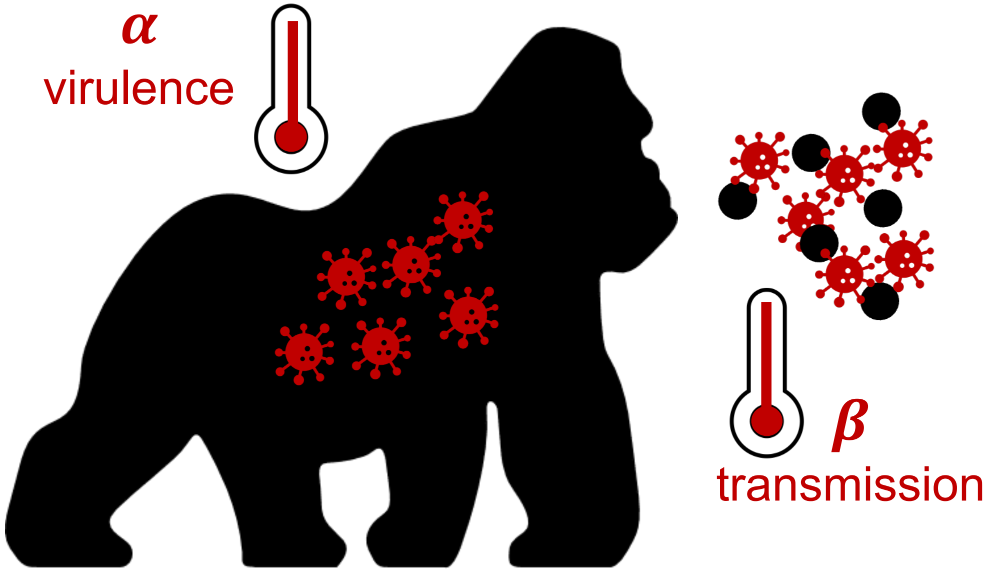


virus tolerance



Brook and Dobson 2015 *Trends Micro*

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.

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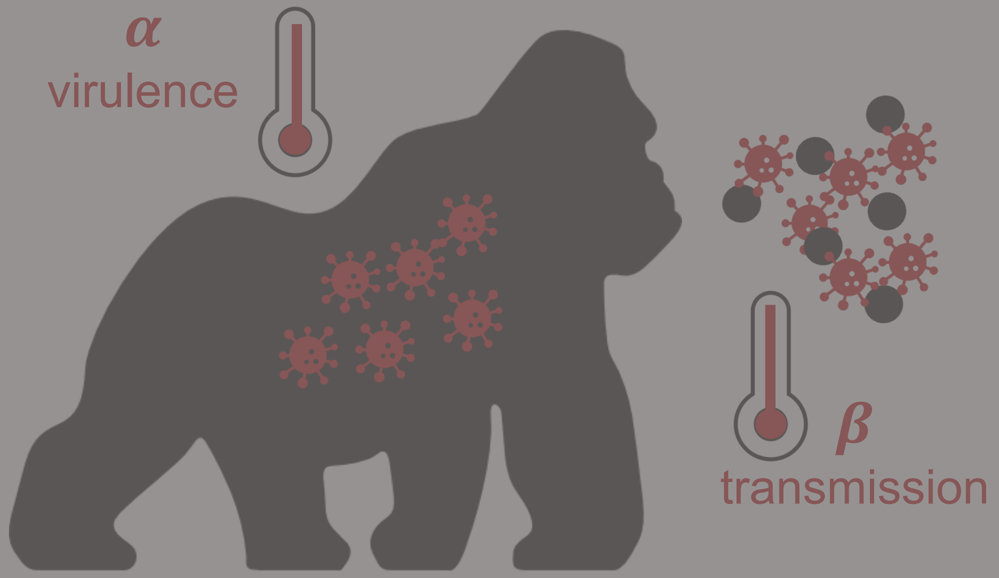

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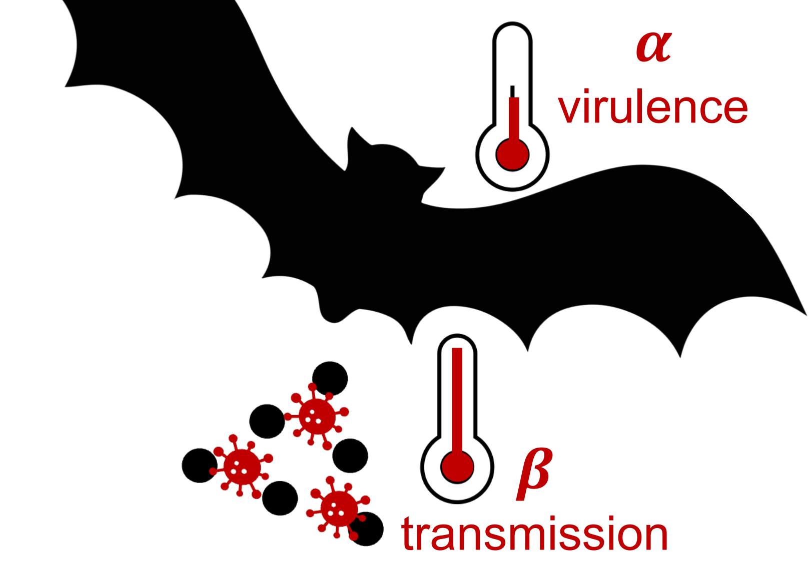
Bats accrue **less virulence** for a given level of transmission.



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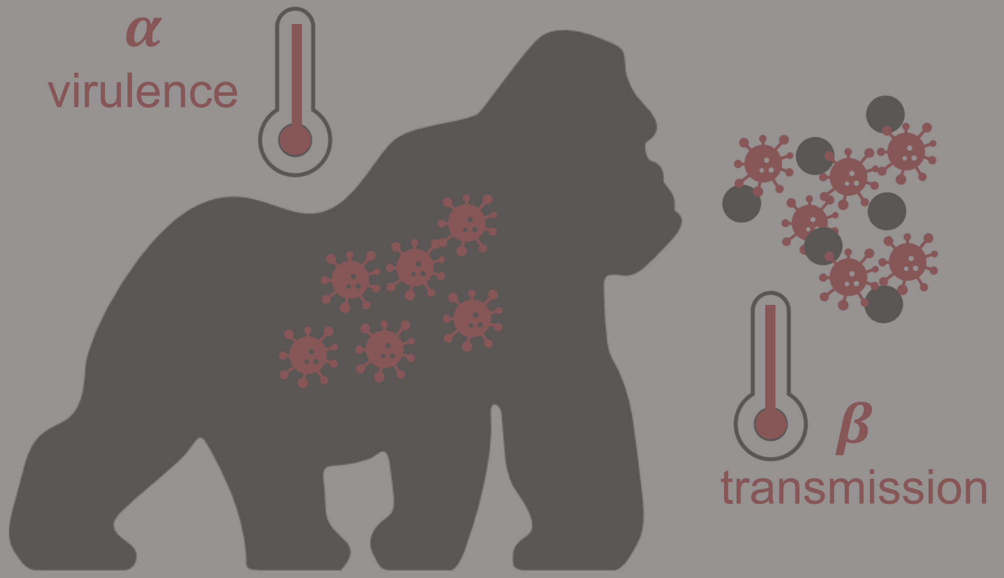

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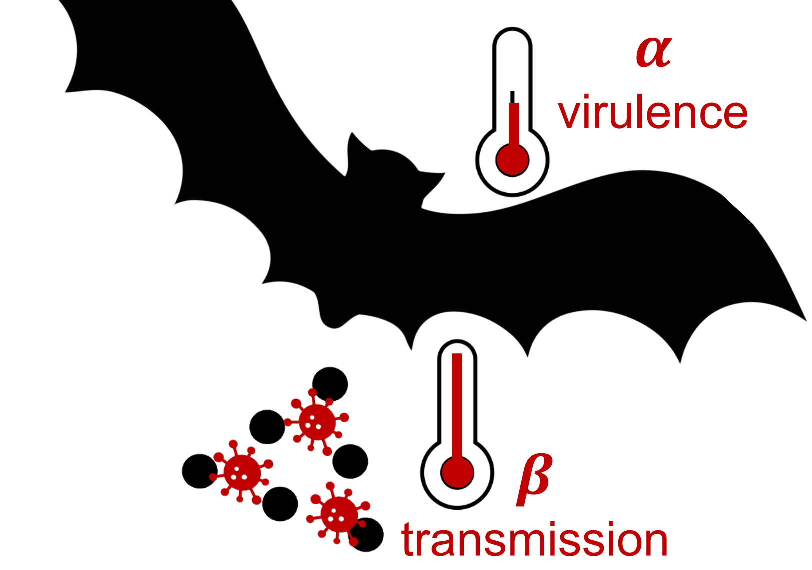

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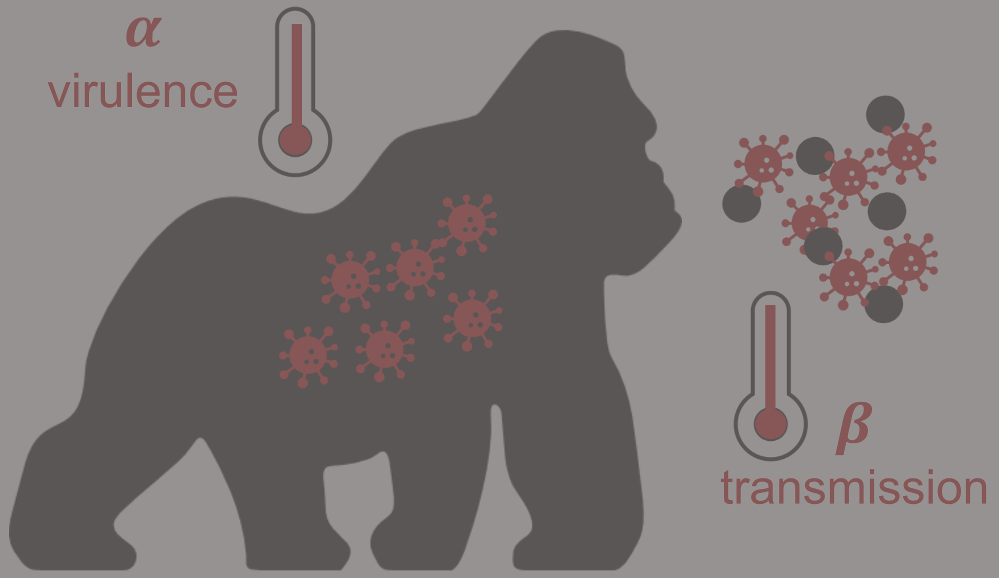

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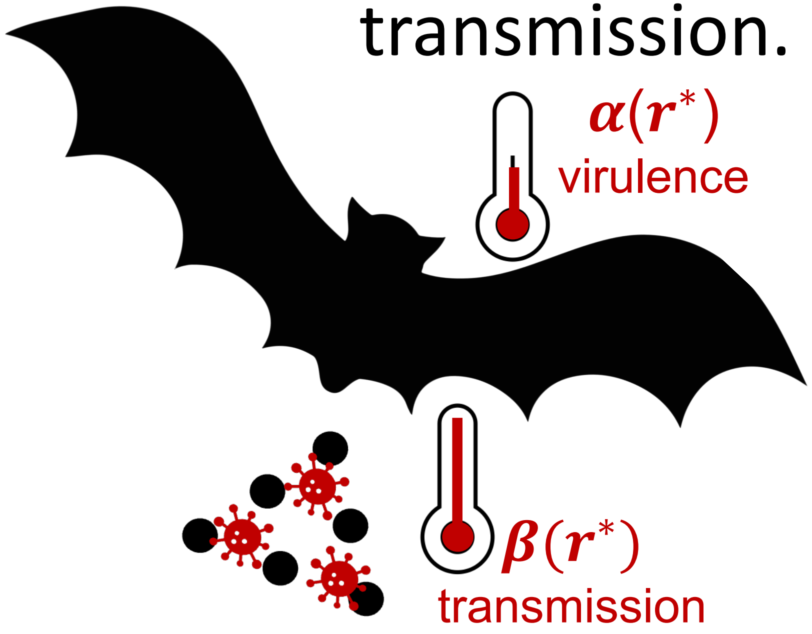

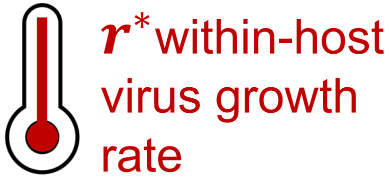

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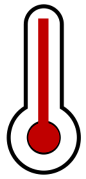
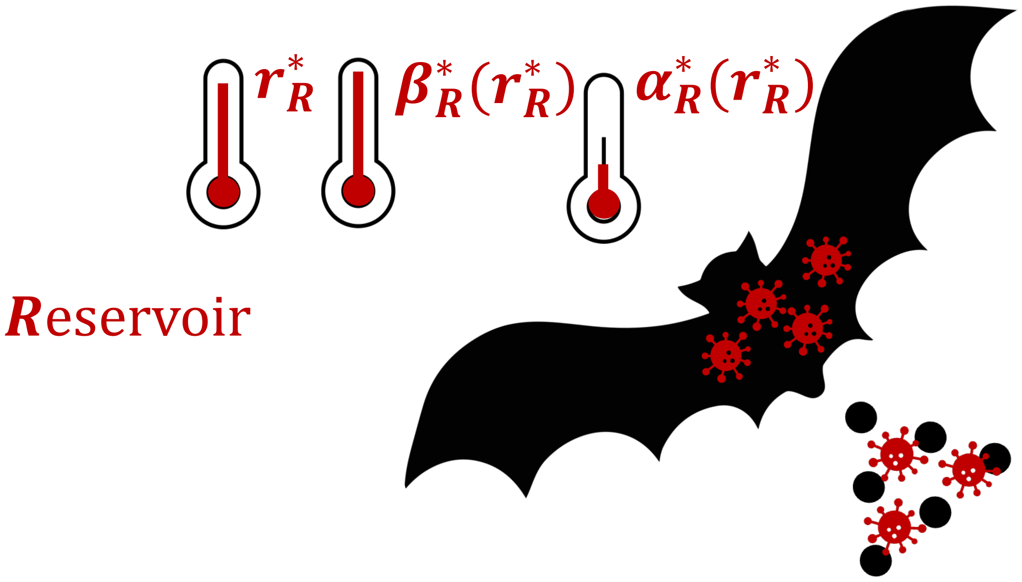


r^* within-host virus growth rate



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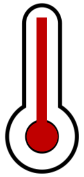

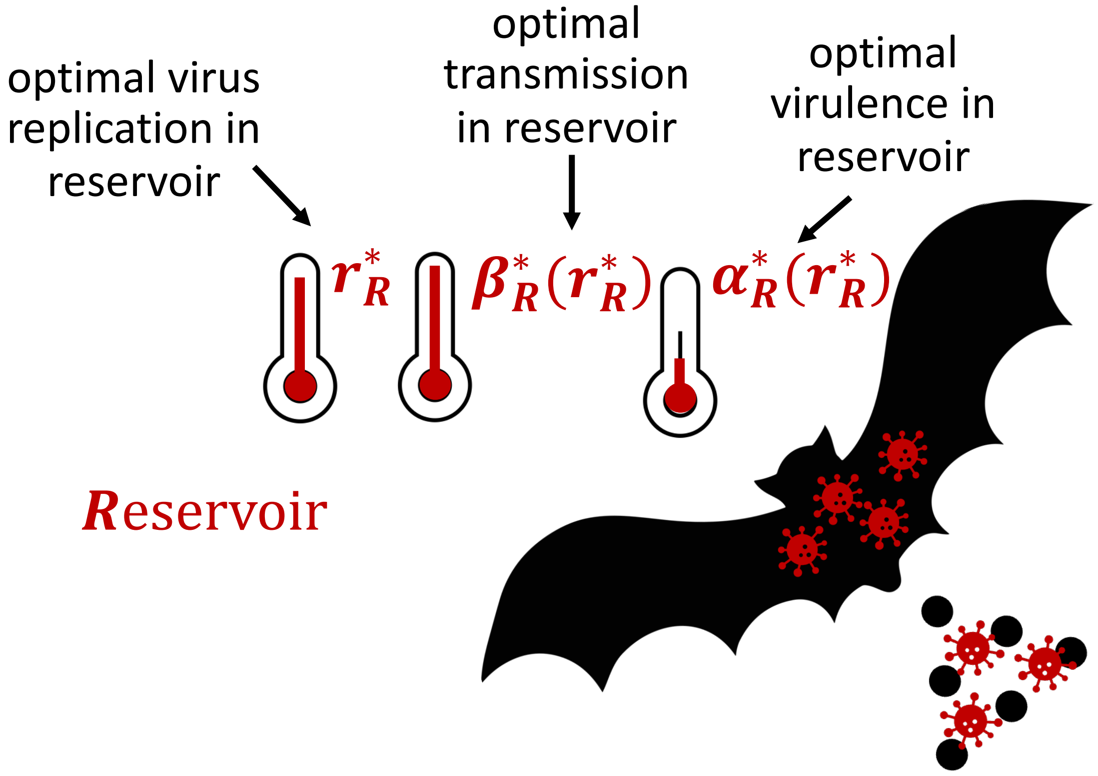

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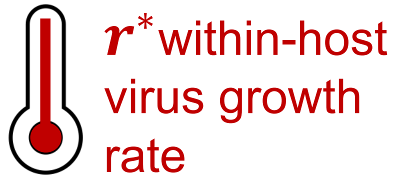

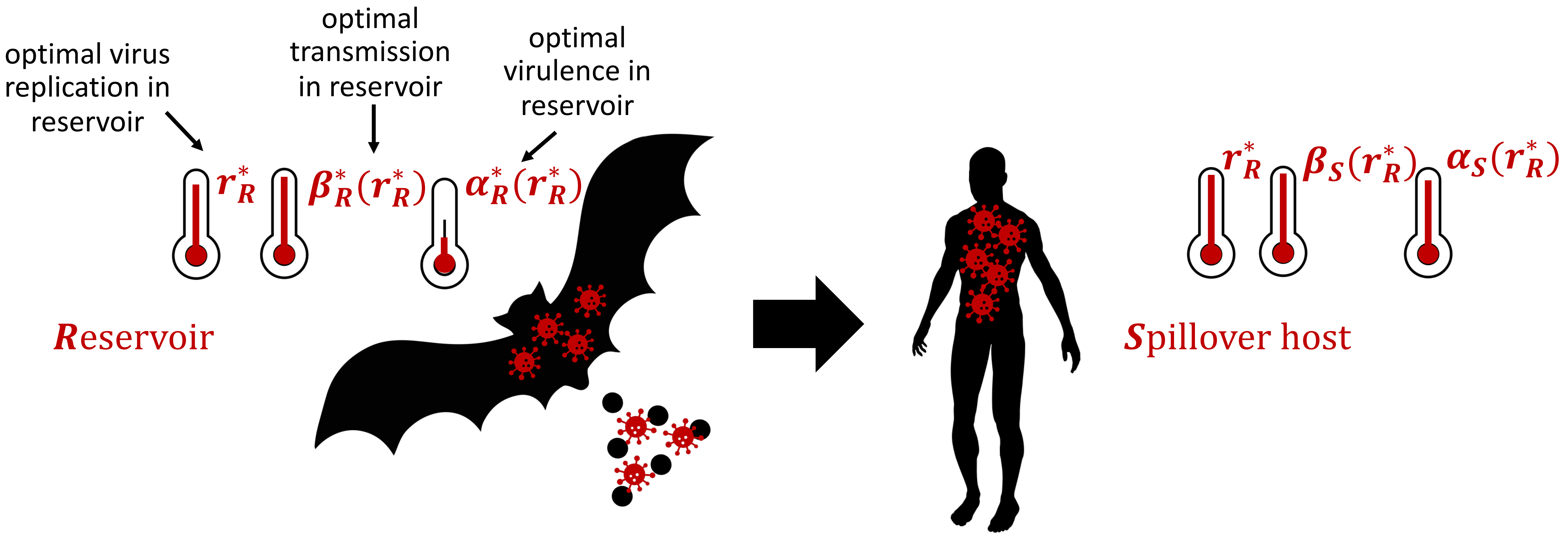

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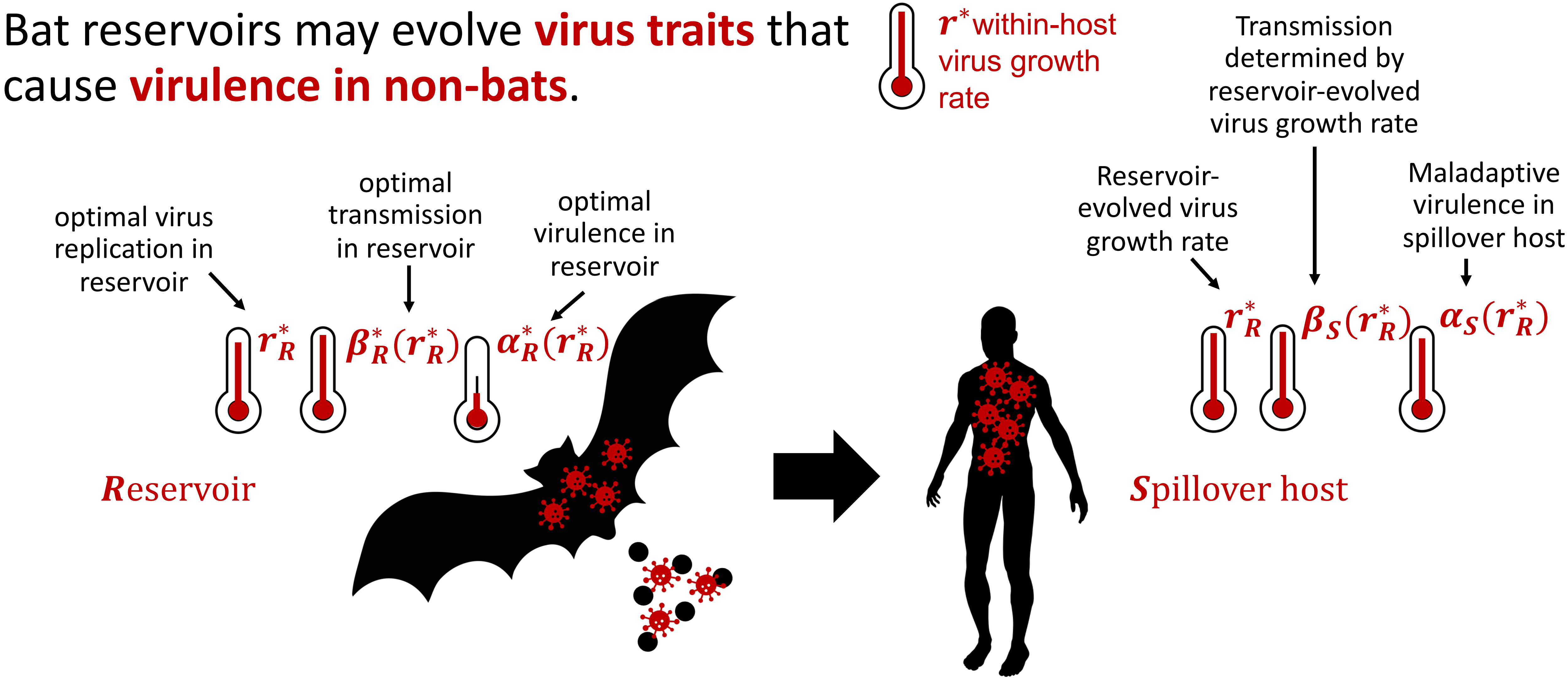

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Limitations of the tradeoff model

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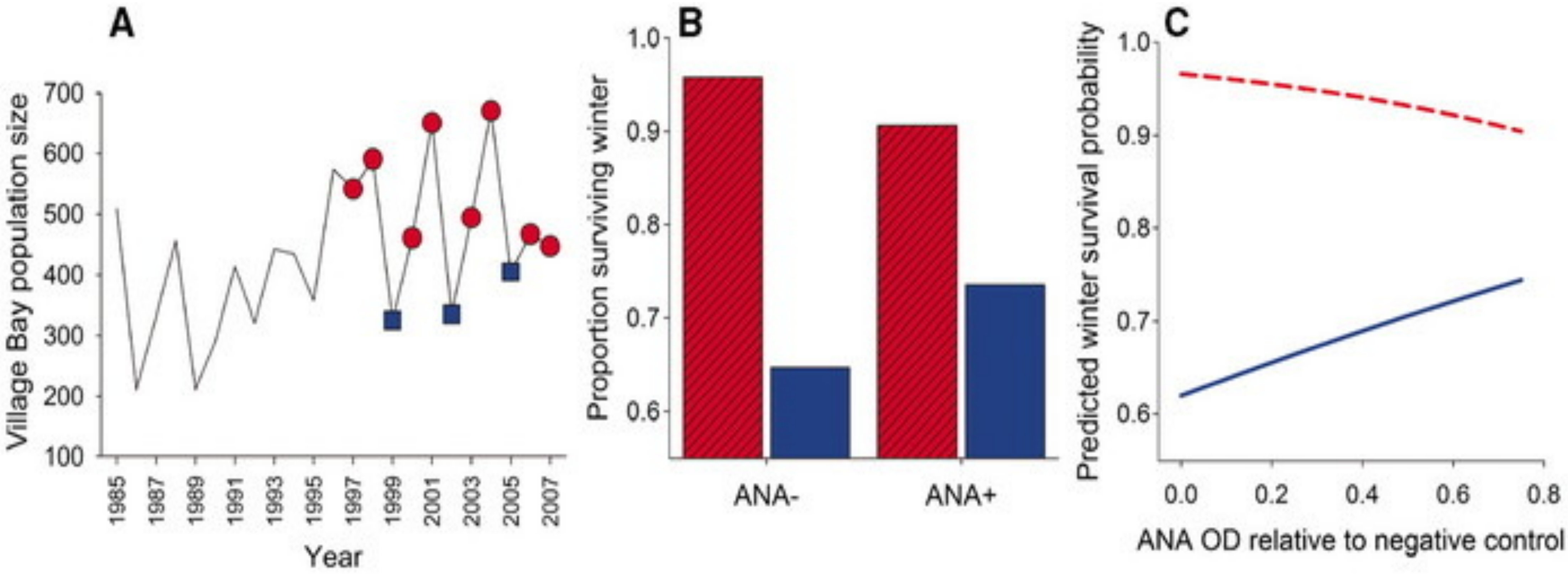

Self-reactive antibodies (ANA) **promote survival by downregulating worms in crash years** but **impede survival via immunopathology in peak years!**

Graham et al. 2010. *Science.*

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  - Ex: Ebola (transmission after death)
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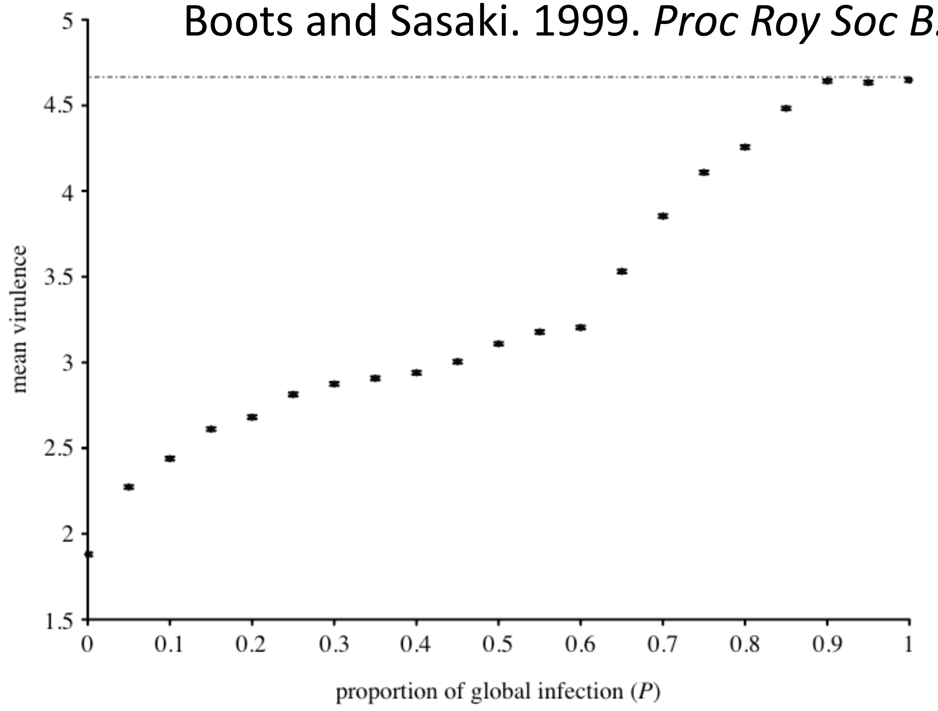
- A few specific applications of tradeoff theory…

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

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



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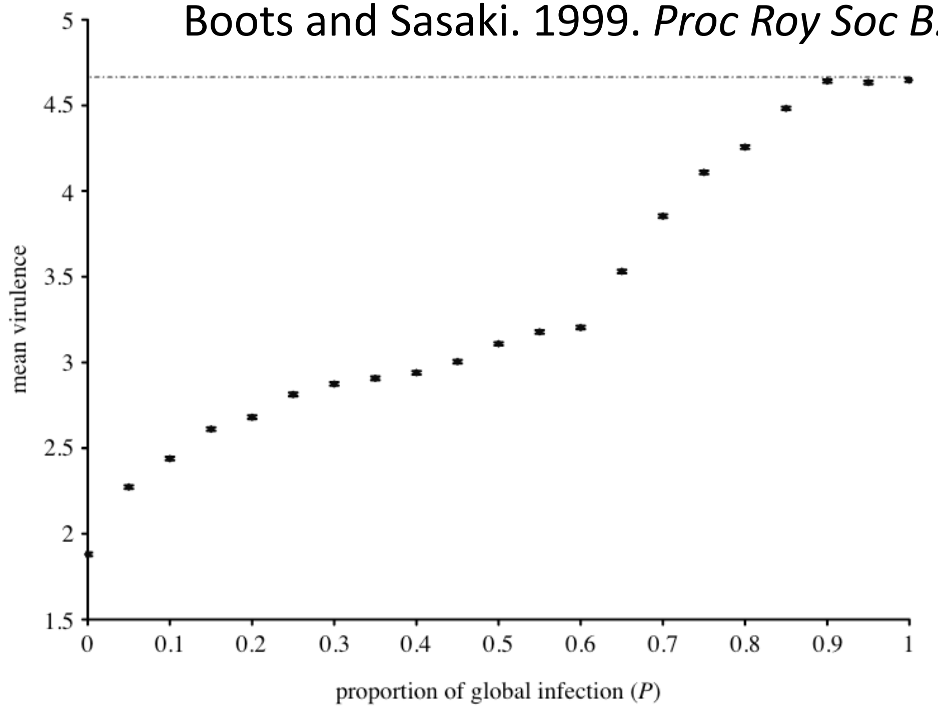
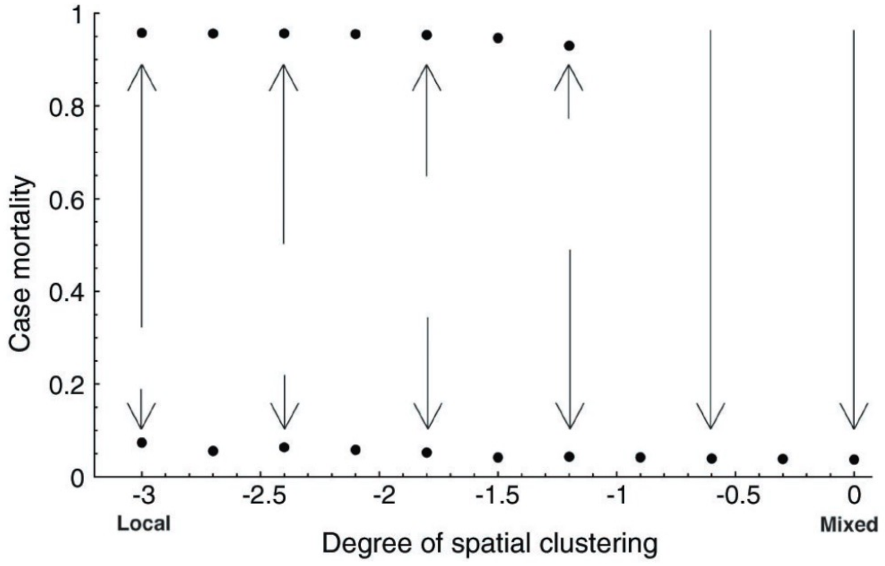


High and low virulence are **both stable evolutionary outcomes** when pathogens generate acquired immunity in spatially structured populations.



Boots et al. 2004. *Science.*

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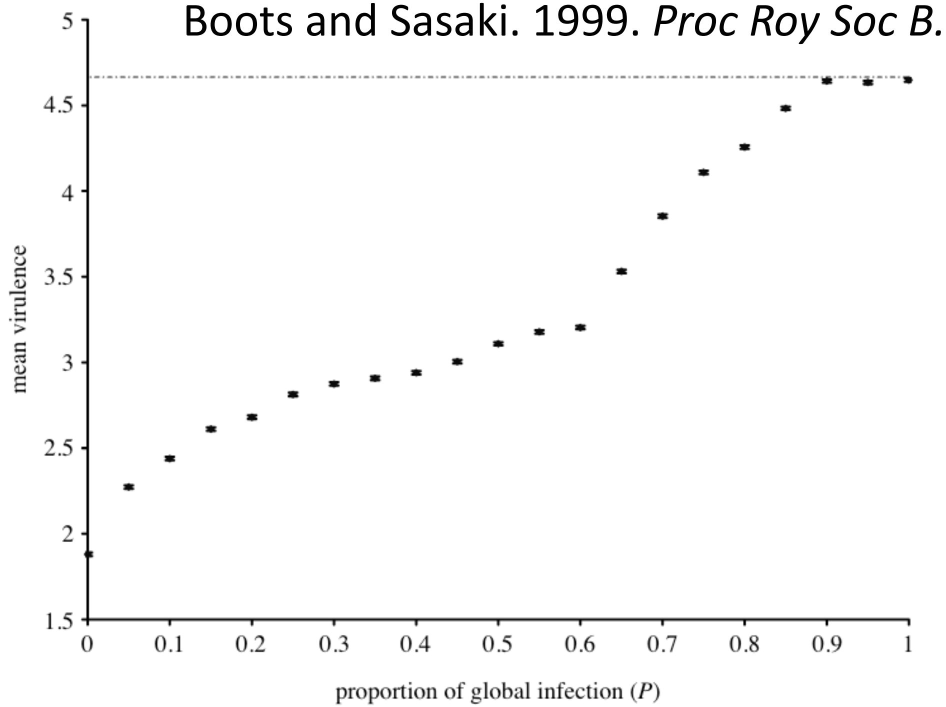

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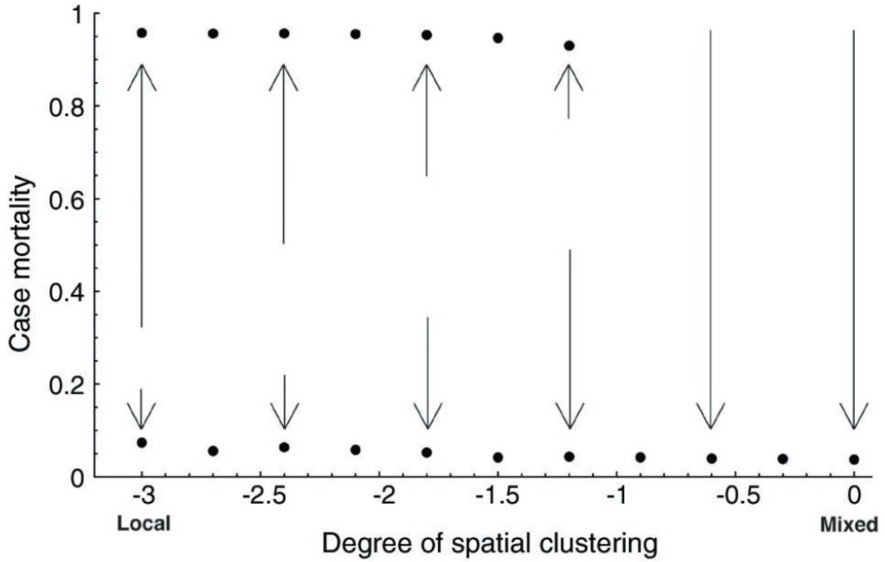
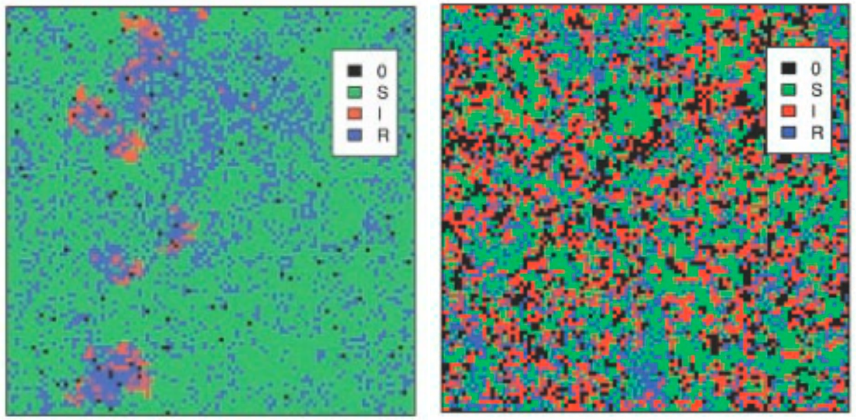
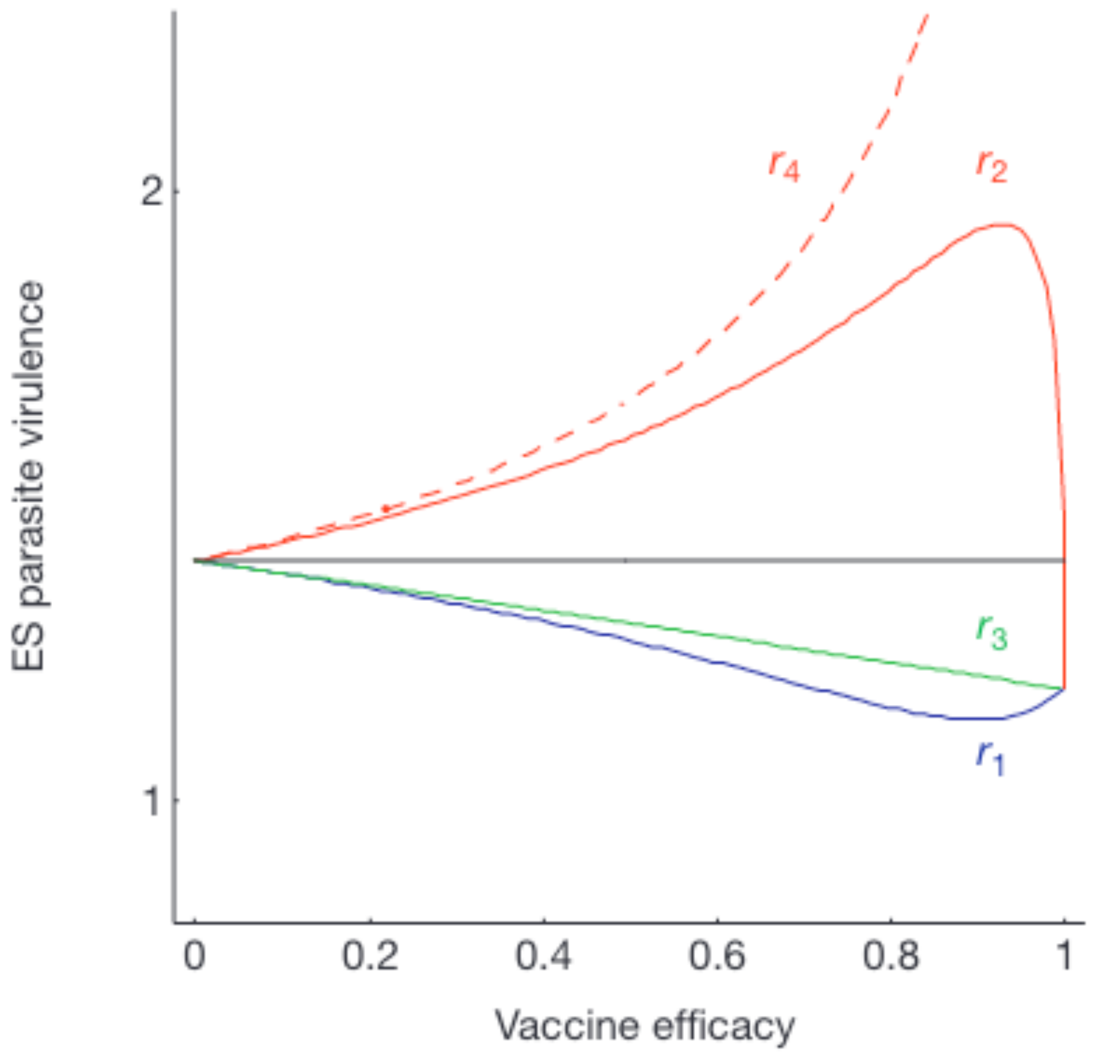


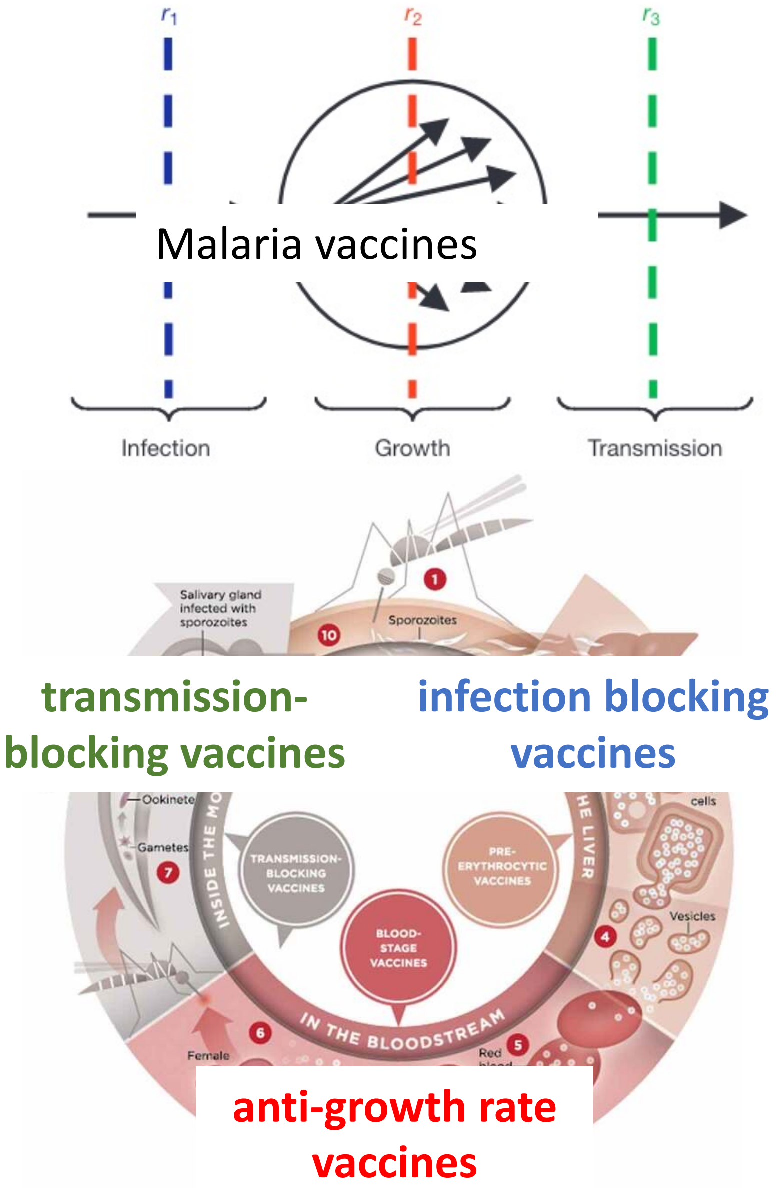
Boots et al. 2004. *Science.*

Here, **low virulence is favored in dense environments** and **high virulence in sparse environments**.



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





Gandon et al. 2001. *Nature.*

Is this ecology or evolution?

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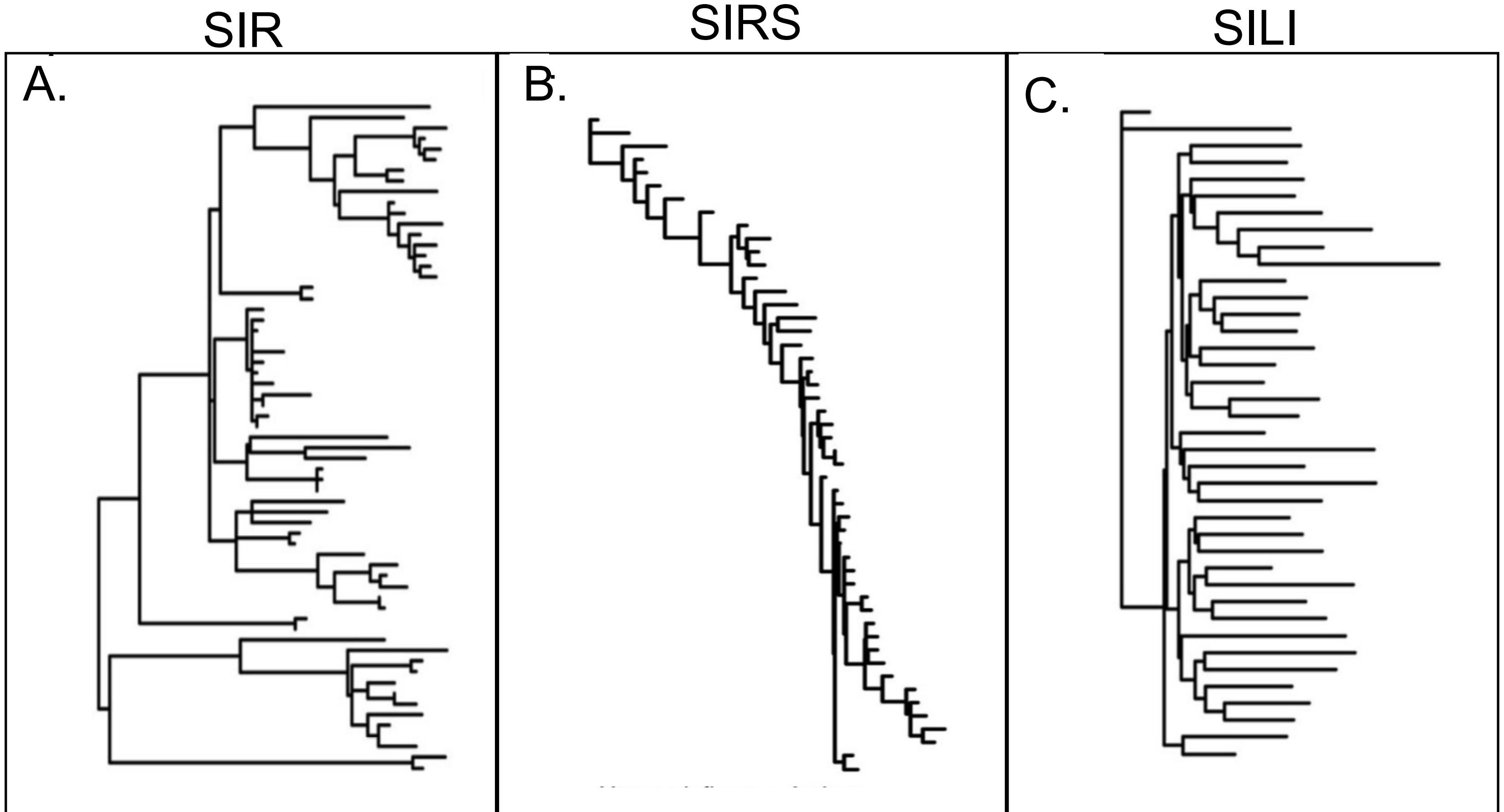
Phylodynamics unites both

Grenfell et al. 2004. *Science.*

Is this ecology or evolution? *Phylodynamics* unites both

Unifying the Epidemiological and Evolutionary Dynamics of Pathogens

Bryan T. Grenfell,[1]* Oliver G. Pybus,[2] Julia R. Gog,[1] James L. N. Wood,[3] Janet M. Daly,[3] Jenny A. Mumford,[3] Edward C. Holmes[2]



measles population phylogeny

influenza A population phylogeny

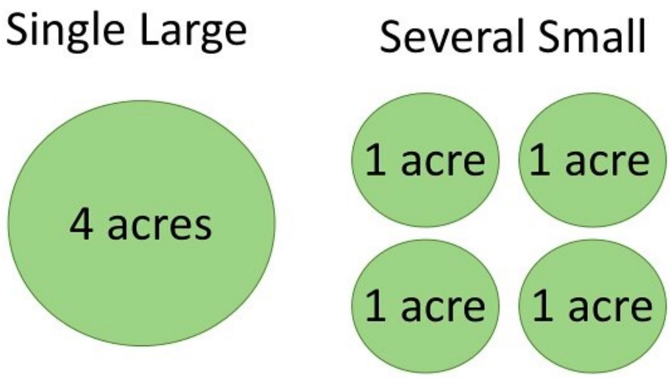
HIV population phylogeny

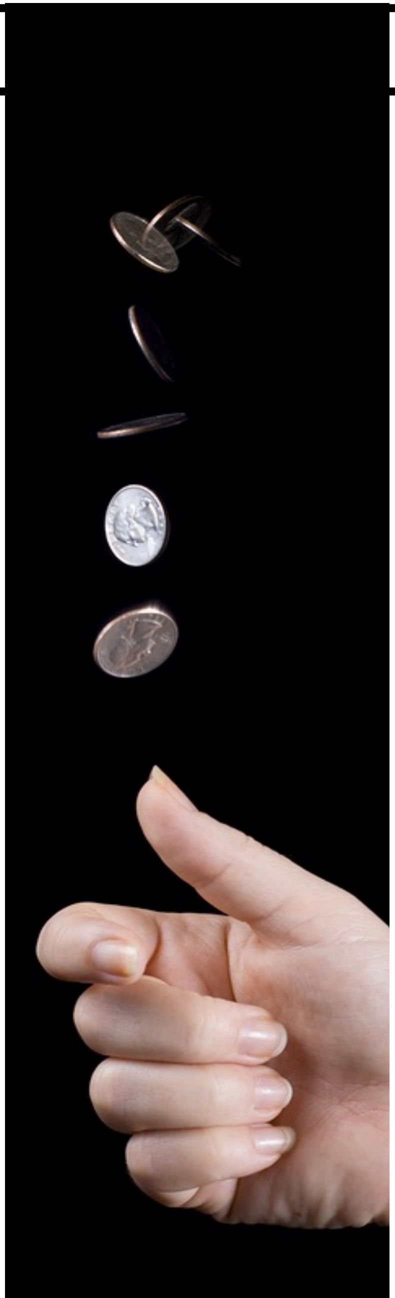
Grenfell et al. 2004. *Science.*

Population Biology

Conservation Biology

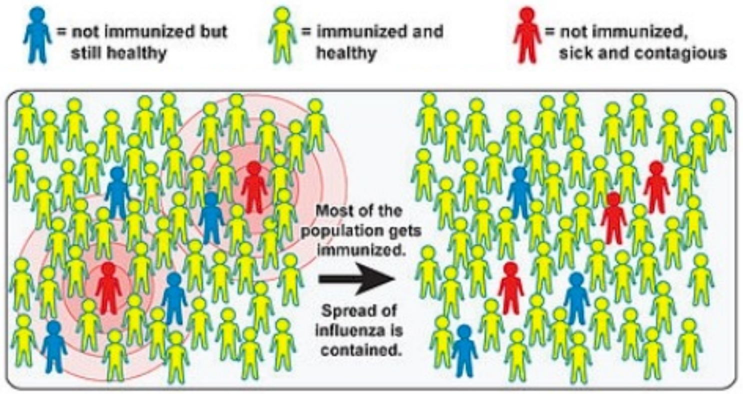
- **Goal:**
  - protect **populations** from **extinction**
- **Concept:**
  - **Minimum Viable Population** size (MVP)
- **Approach:**
  - protected area **reserves**



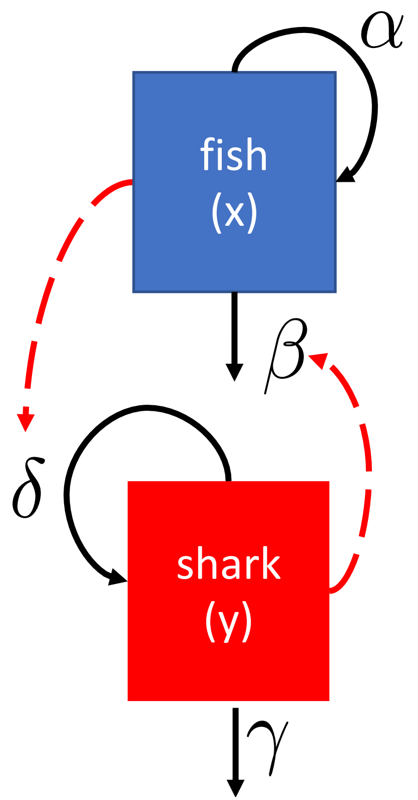


Disease Ecology

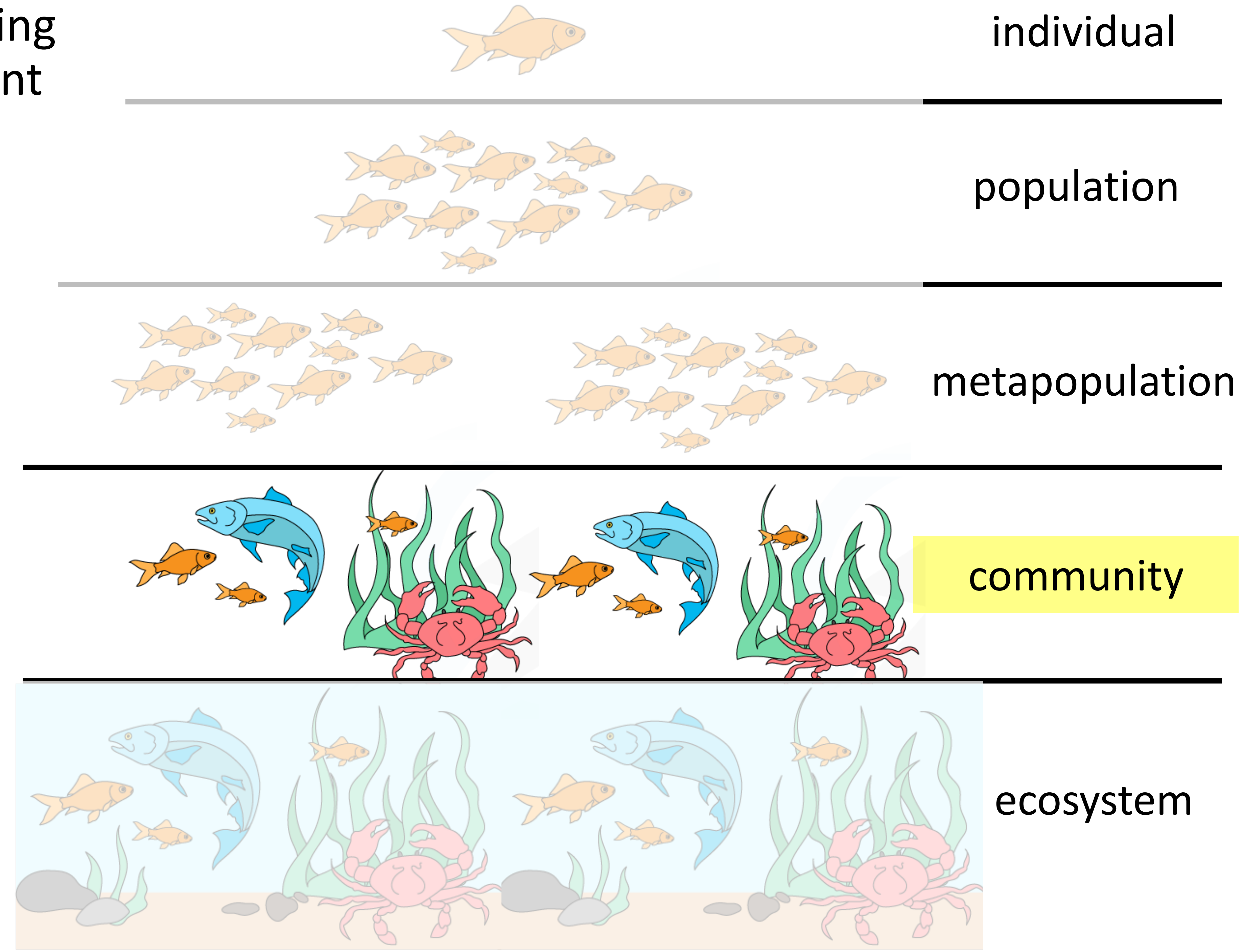
- **Goal:**
  - protect **populations** from disease via pathogen **extinction**
- **Concept:**
  - **Critical Community Size** (CCS)
- **Approach:**
  - sanitation
  - **vaccination**



Community = interacting populations of different species



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

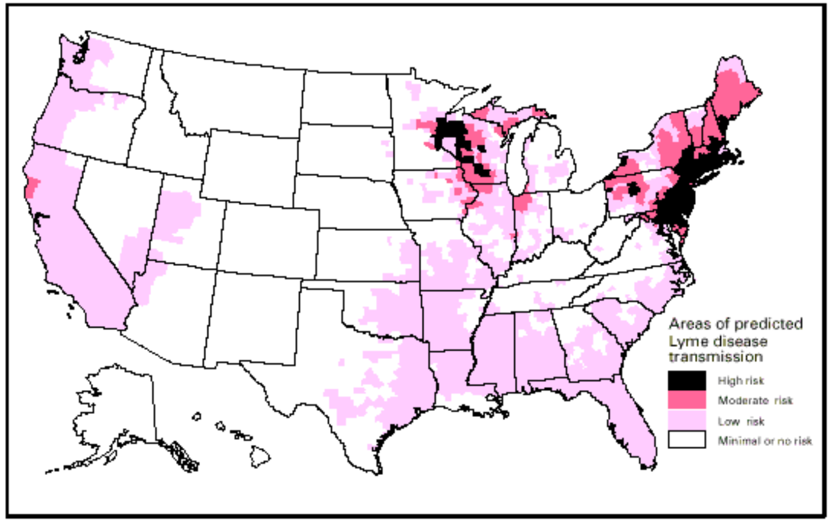


Disease dynamics in the **broader community**

Disease dynamics in the **broader community**

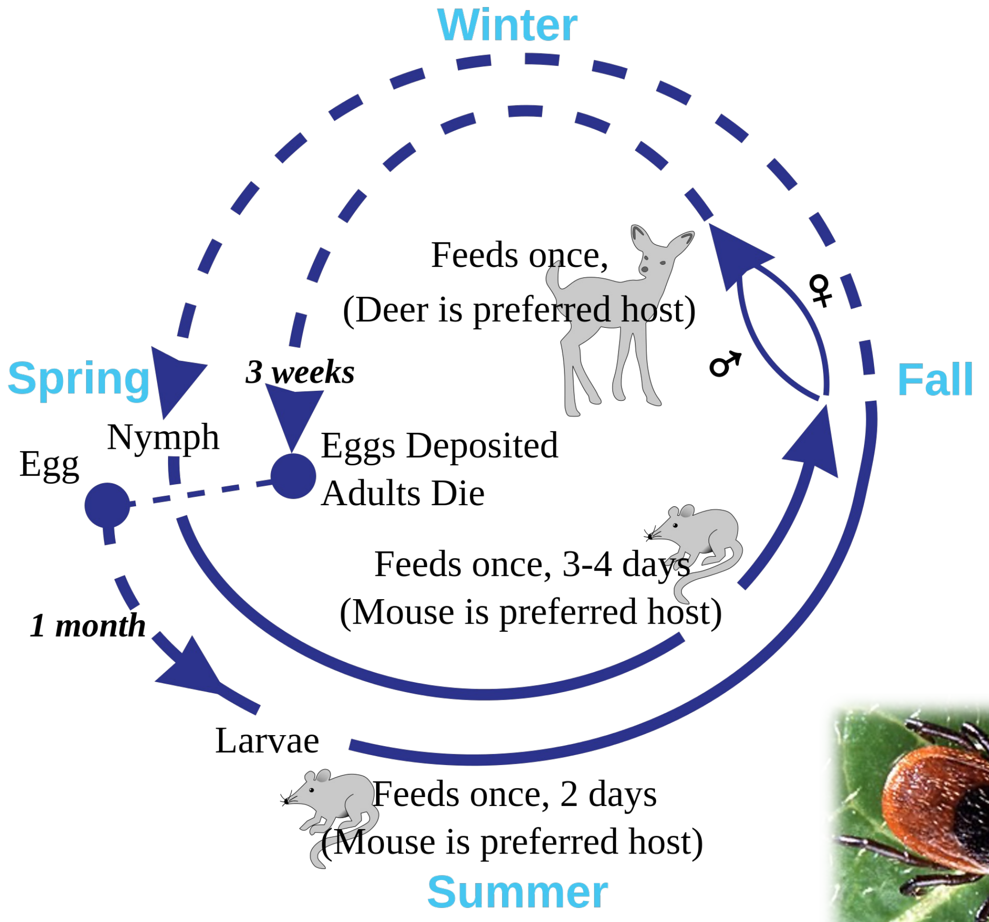
Example: Lyme Disease

National Lyme disease risk map with four categories of risk



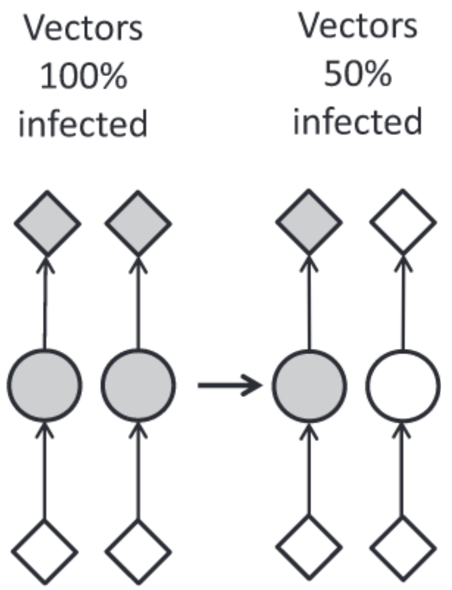
- Lyme disease is a vector-borne disease caused by the bacterium, *Borrellia burgdoferi,* vectored by *Ixodes* especially *Ixodes scapularis*) ticks.
- Nymph ticks are borne in the spring, feed on small mammal hosts through the summer, then reproduce (particularly on deer) in the fall before going dormant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

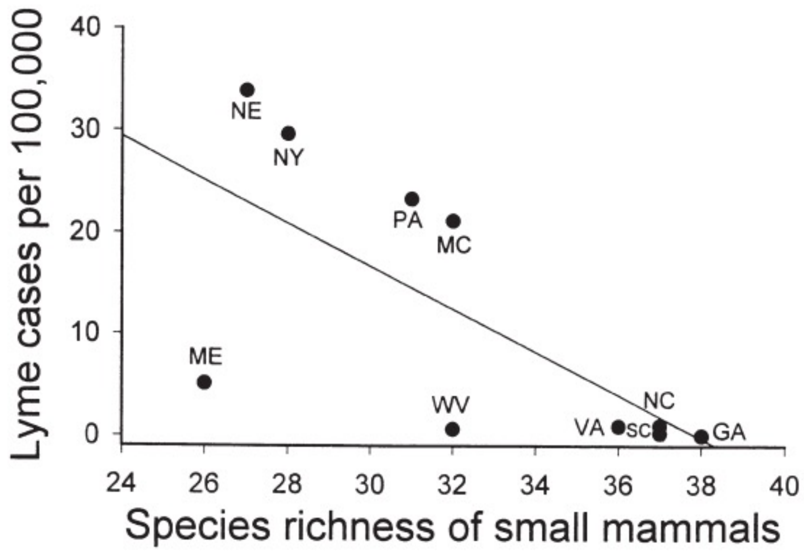
Life Cycle of the *Ixodes scapularis* Tick

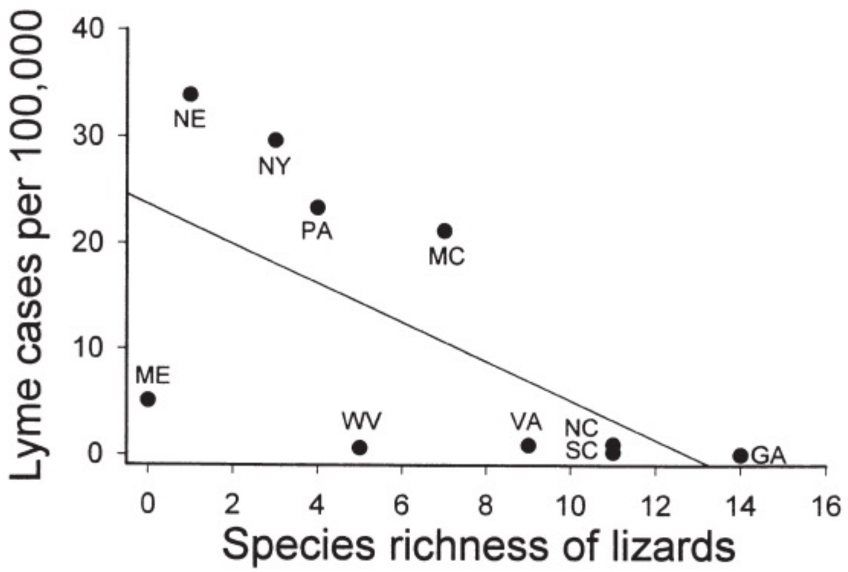


The **dilution effect** highlights buffering effects of **biodiversity on disease transmission**.

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







In the case of Lyme, **increasing host biodiversity tends to be associated with decreasing Lyme prevalence**.

Dobson and Auld. 2016. *The American Naturalist.*

Ostfeld and Keesing. 2000. *Conservation Biology.*

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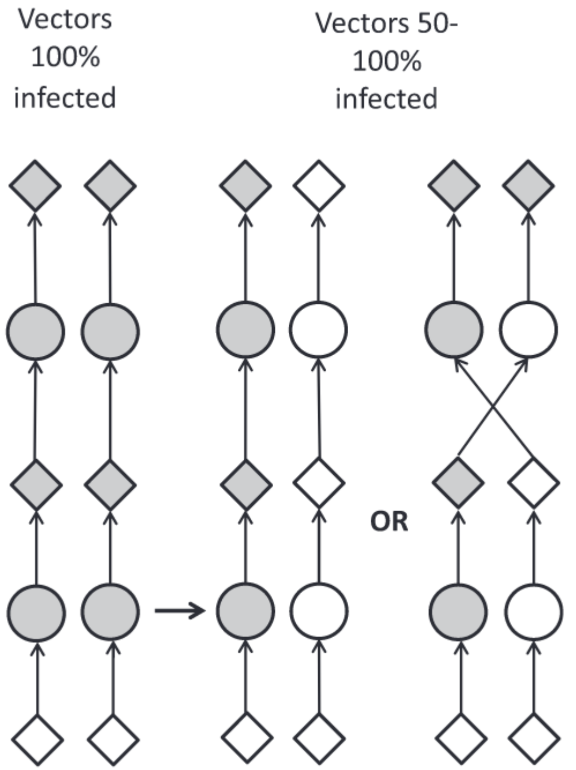

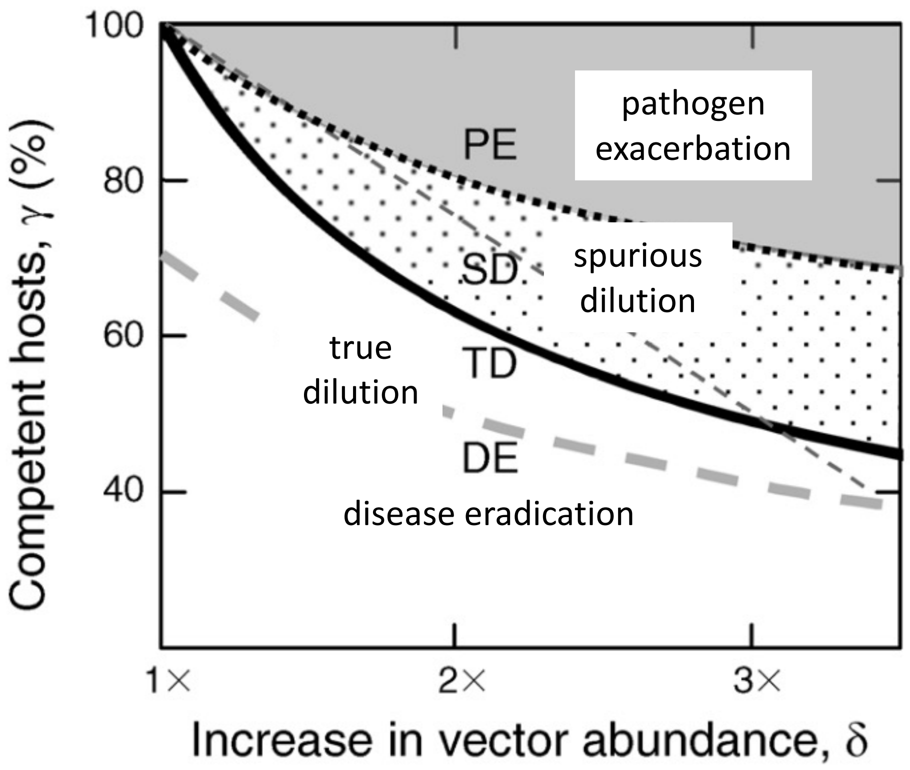

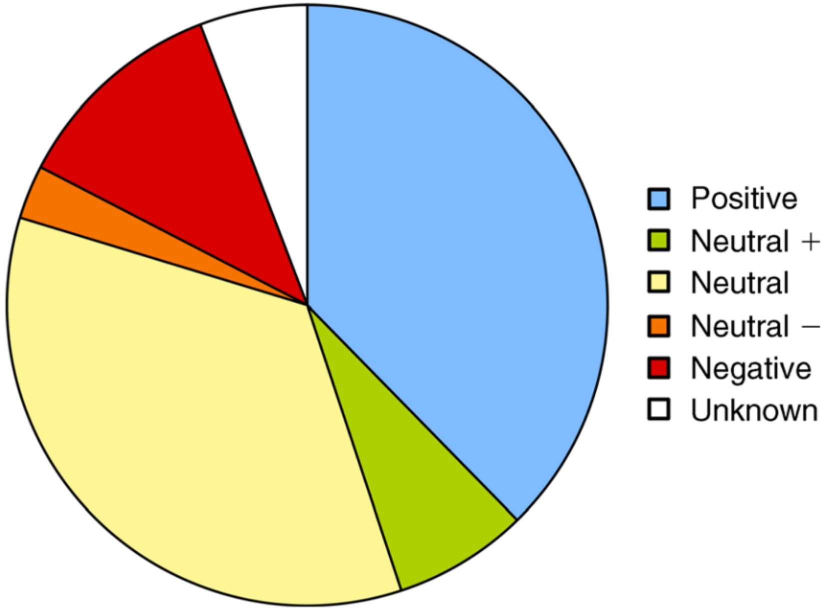

Dobson and Auld. 2016. *The American Naturalist.*

Wood et al. 2014. *Ecology*