

Fundamentals of Ecology

Week 8, Ecology Lecture 6

Cara Brook

February 22, 2024

Let's recap a bit!

Disease Ecology

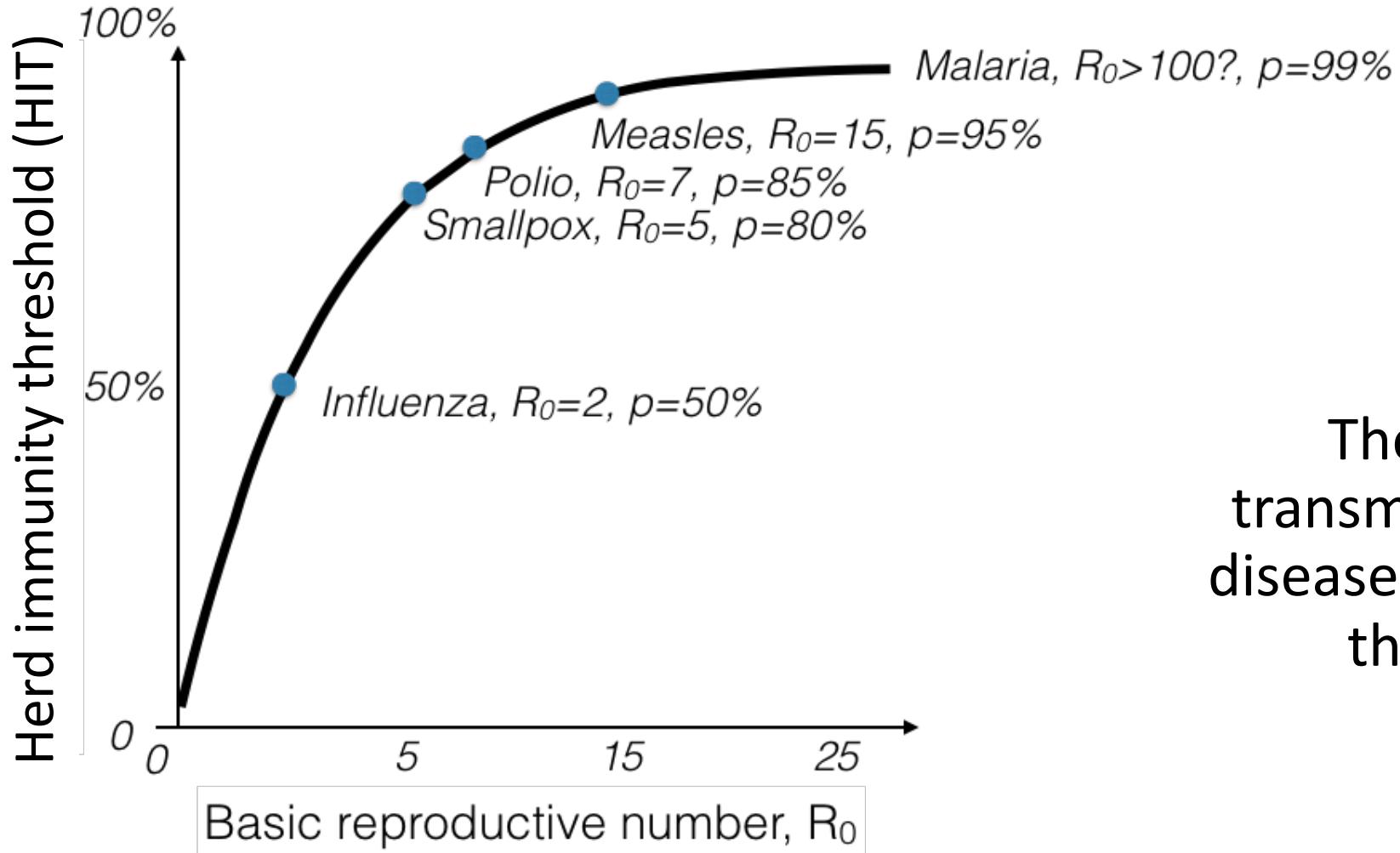
- Parasitism is a species interaction in which a parasite or pathogen lives in or on a host and benefits at the expense of the hosts.
- Pathogens play a key role in regulating population cycles in wildlife (e.g. Soay sheep, red grouse) and also impact human demography (e.g. Plague of Justinian, Black Death, Cocoliztli, HIV, COVID)
- The recognition that microorganisms (bacteria, viruses, protozoa) cause disease dates back to the origins of the microscope (van Leeuwenhoek) and was later formalized in Koch's postulates in the late 1800s
- In addition to demography, pathogens have also shaped human evolution (malaria → sickle cell anemia, Duffy negative antigen)
- Epidemiology is focused on understanding the risk factors associated with disease, while disease ecology is focused on understanding their transmission

Let's recap a bit!

Disease Ecology

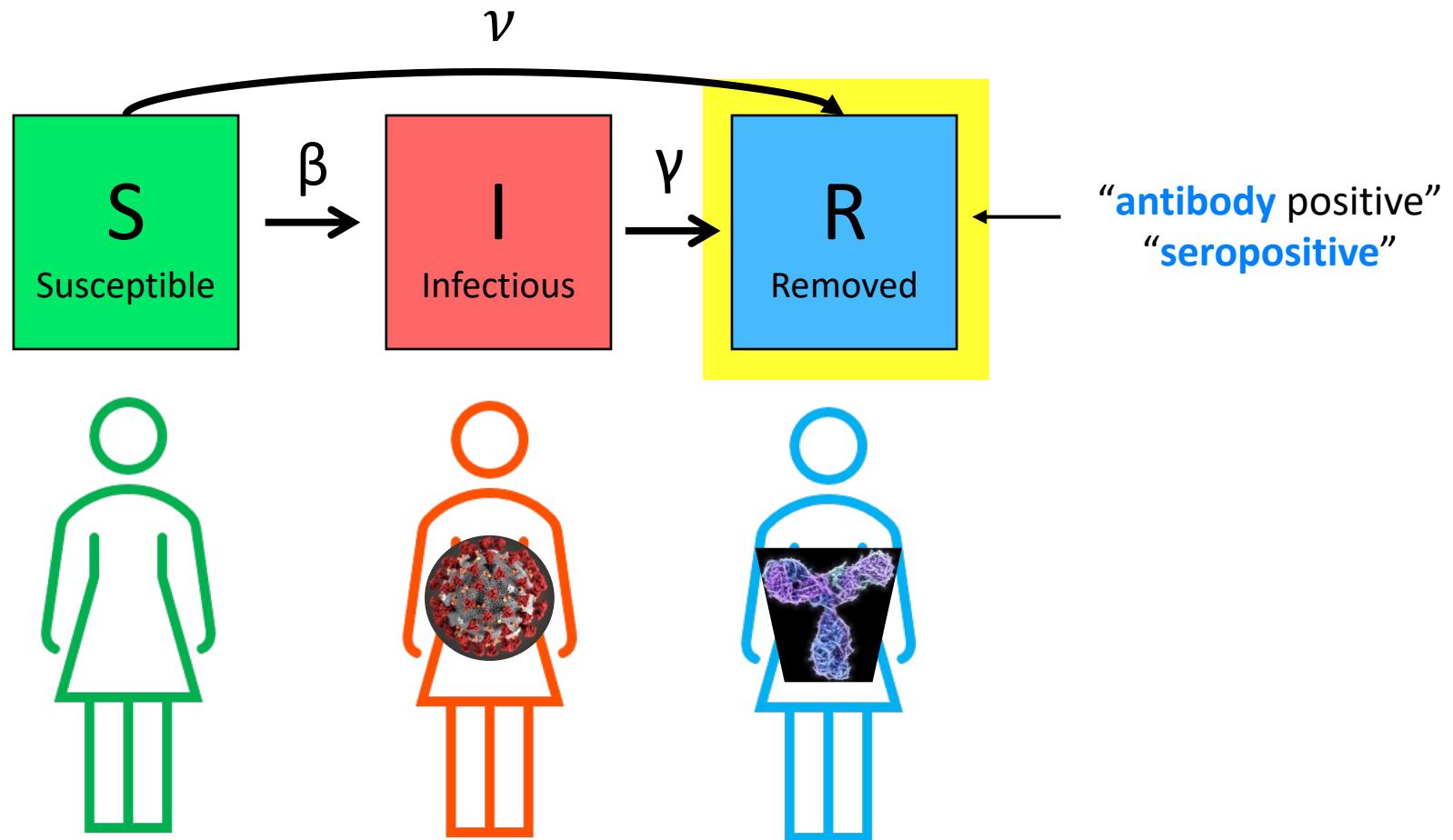
- We model transmission dynamics using the Susceptible-Infected-Recovered (SIR) model, where hosts are classed according to their infectious status.
- R_0 , the basic reproduction number for a pathogen, describes the number of new infections generated by a single infection in a completely susceptible population. It is expressed with the terms of infections gained / infections lost.
- R_E , or R_t , is the effective reproduction number. It is $R_0 * \text{the proportion susceptible}$. Epidemics grow at $R_E > 1$ and decline at $R_E < 1$.
- We can leverage this RE equation to describe the proportion of the population needed to vaccinate to eradicate a pathogen: $P_V = 1 - 1/R_0$
- Vaccination dates back to antiquity but does not work equally well for all pathogen types and faces many challenges because most pathogens do not follow the simplistic dynamics of measles.

R_0 and the Herd Immunity Threshold



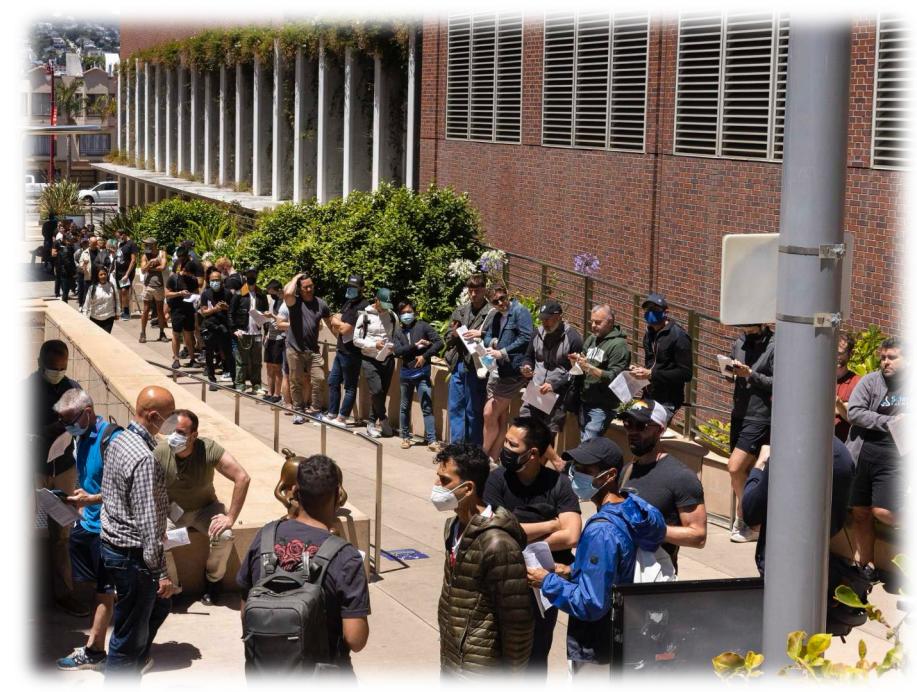
The more
transmissible the
disease, the higher
the HIT!

Vaccination stems from a long history

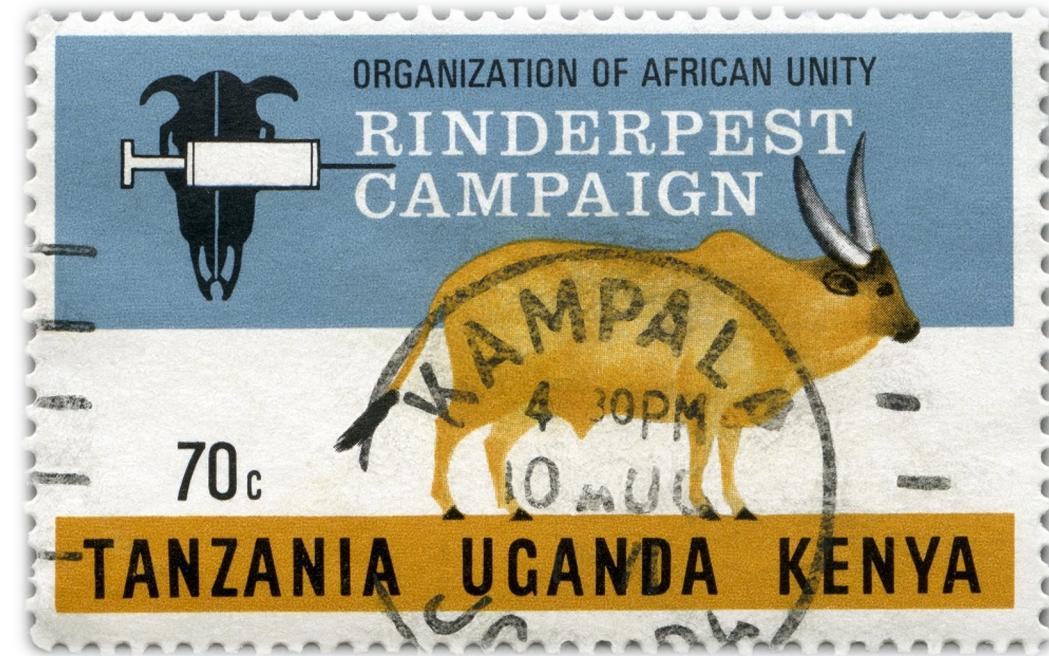


Vaccination stems from a long history

- Variolation: Early attempts to provide protection against smallpox (*Variola virus*) via inoculation with scab material from a recent patient infected with *Variola minor*
 - First described in China in the 10th century
 - Caused 1% mortality!
- 1789 Edward Jenner used cowpox vesicles to inoculate an 8-year-old boy
 - Later inoculated with smallpox and boy was unaffected
 - The first vaccine, taken from *vacca*, cow in Latin
- Smallpox was globally eradicated in 1977, following a massive international campaign
- Today, we are seeing enhanced transmission of monkeypox partly resulting from a lack of circulating immunity to closely related smallpox

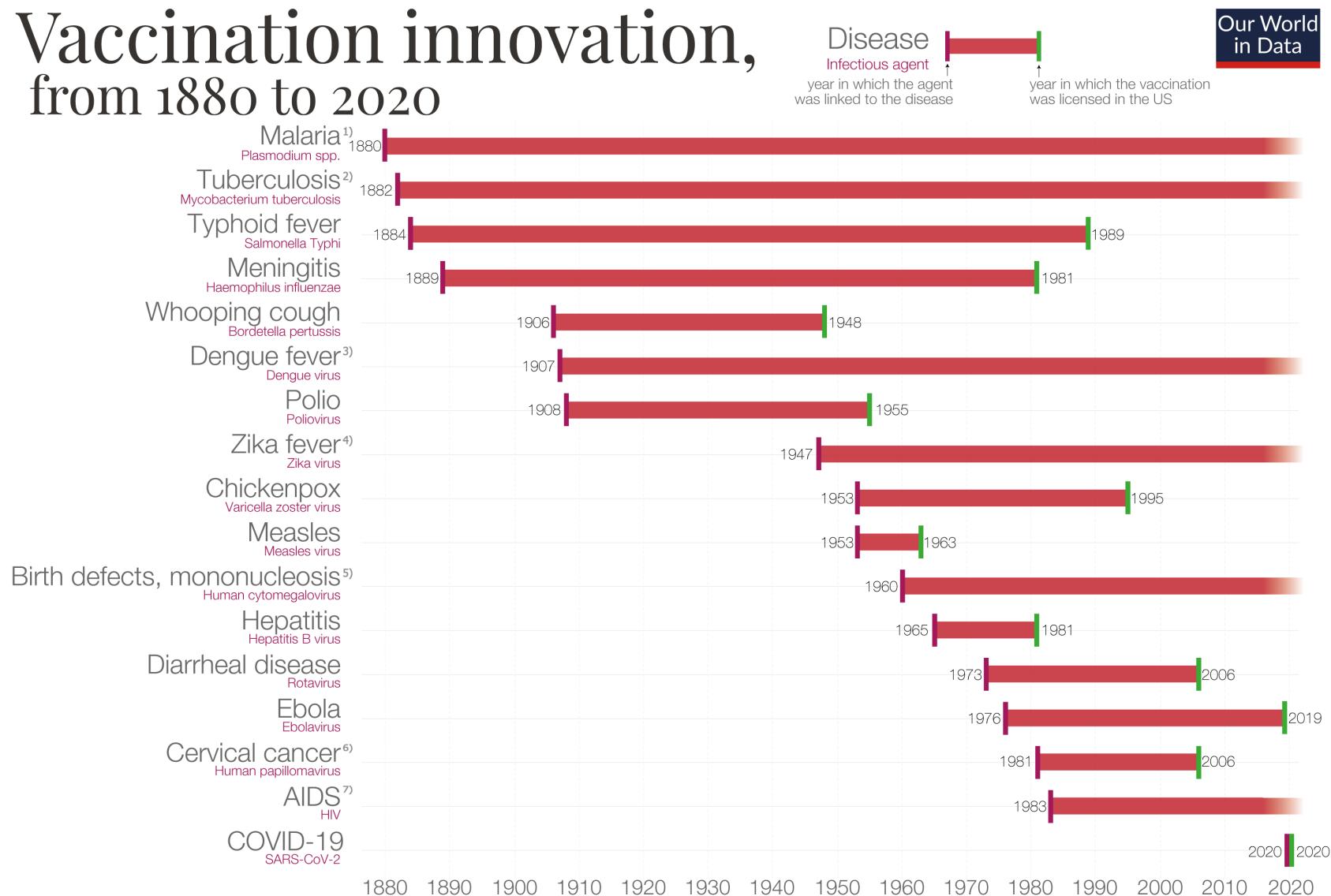


Only two global vaccination success stories



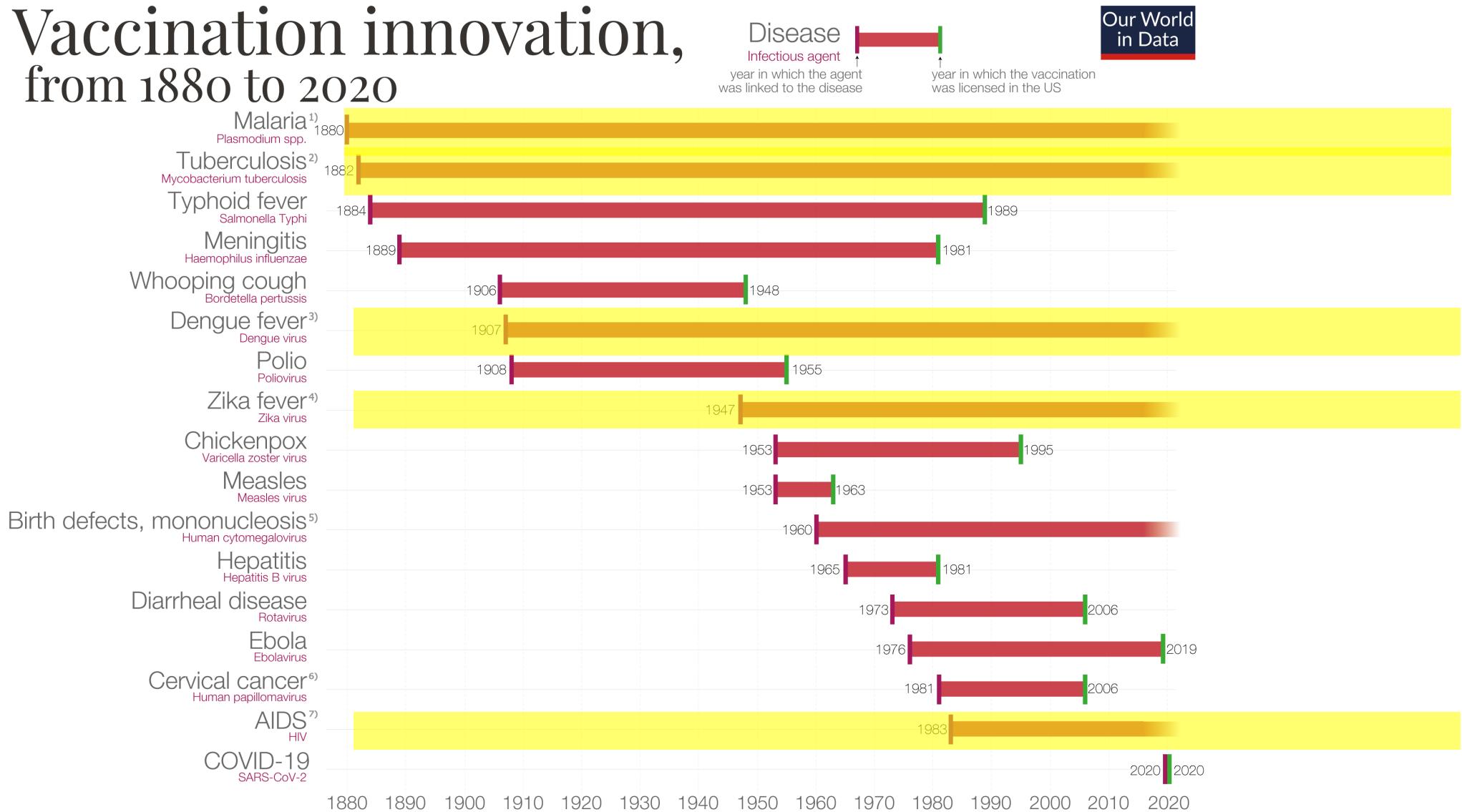
The pace of vaccine development has accelerated drastically

Vaccination innovation, from 1880 to 2020



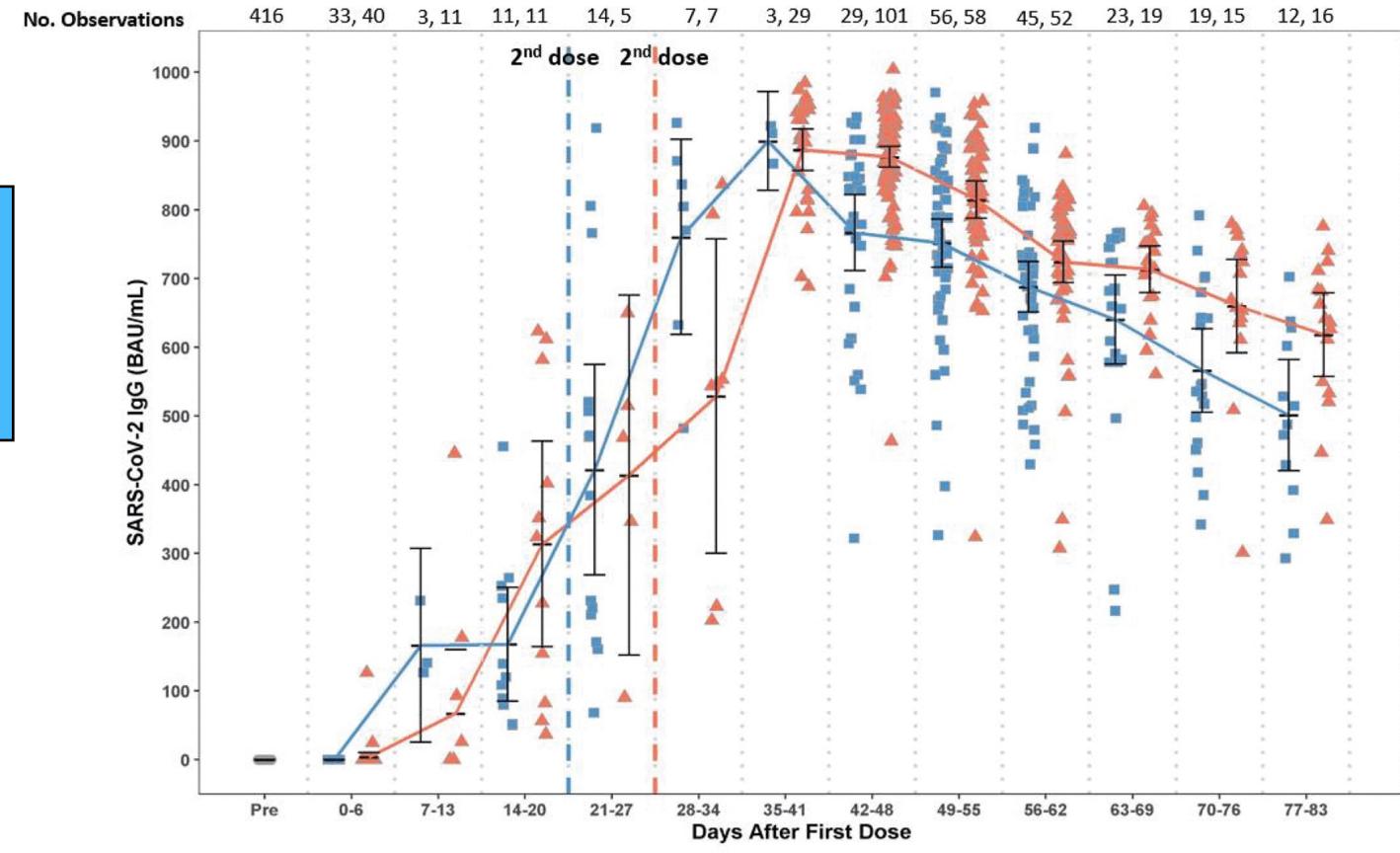
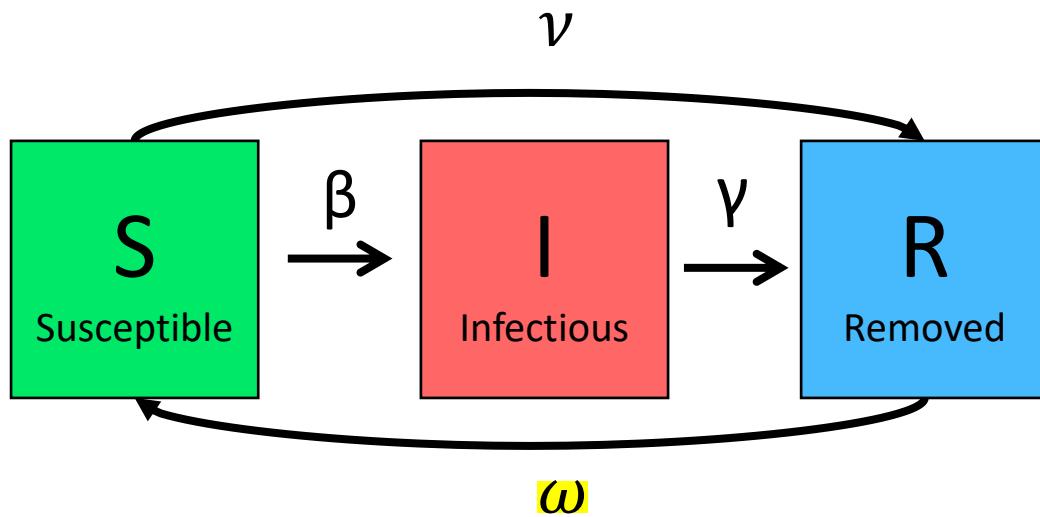
But we still lack vaccines for several important diseases.

Vaccination innovation, from 1880 to 2020



Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens



β = transmission rate

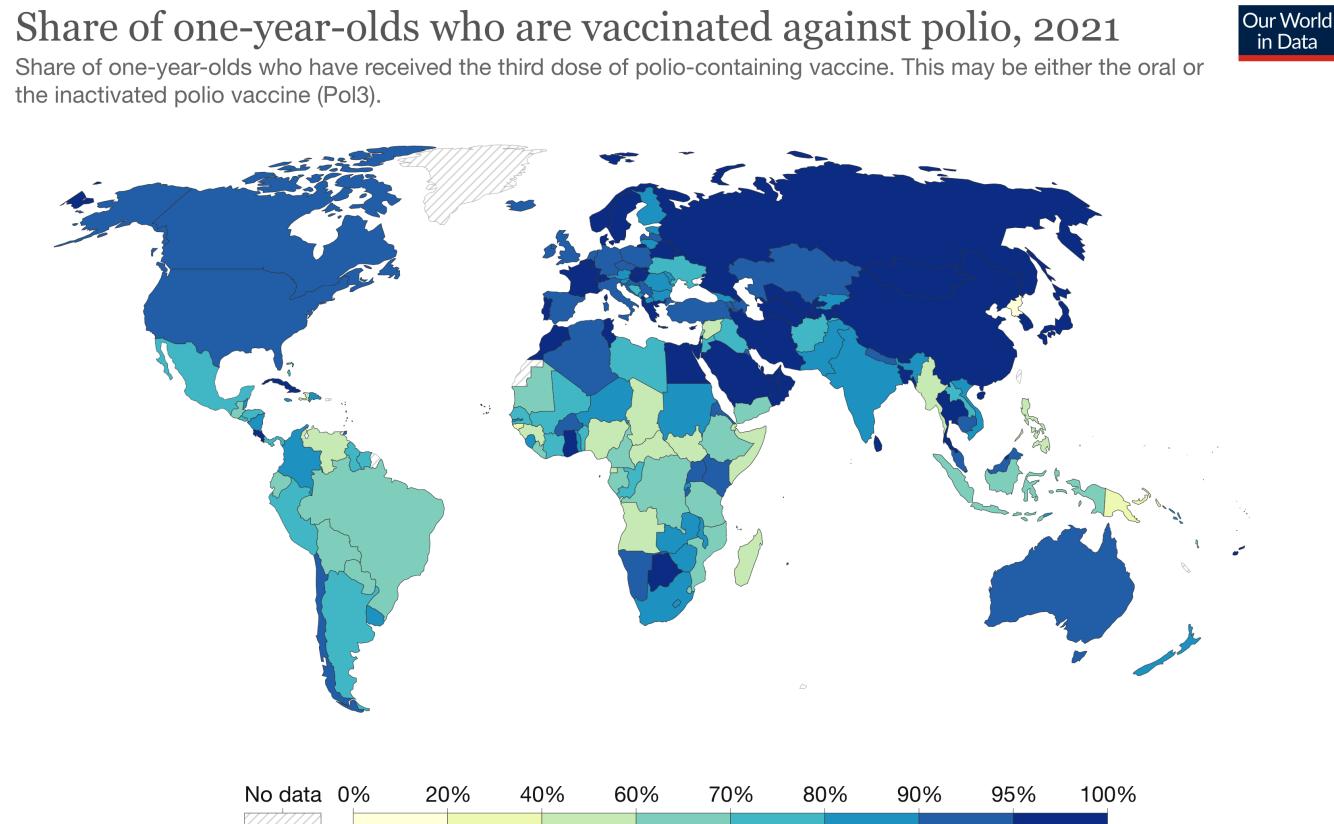
γ = recovery rate

ν = vaccination rate

ω = rate waning immunity

Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access



Source: WHO; UNICEF (2022)

Note: Polio is a highly infectious viral disease. The polio virus invades the nervous system and can cause irreversible paralysis.

OurWorldInData.org/polio/ • CC BY

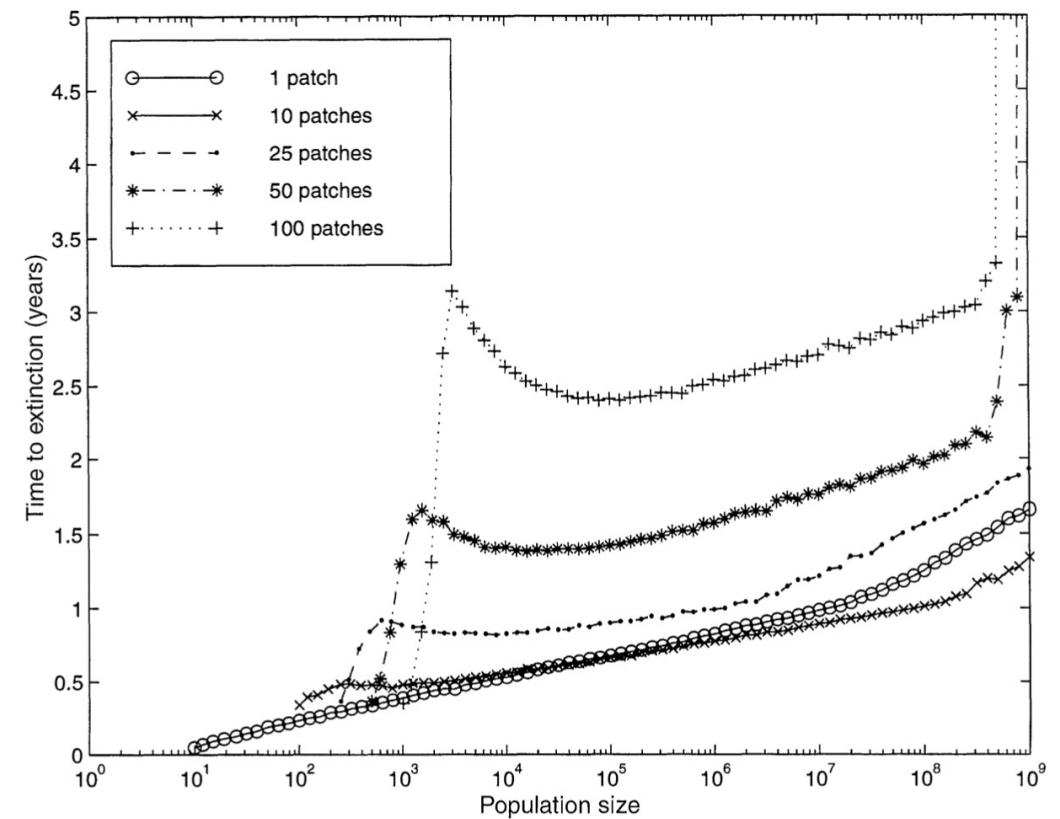
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- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access
- Continuous births
- Animal reservoirs



Challenges to Vaccination

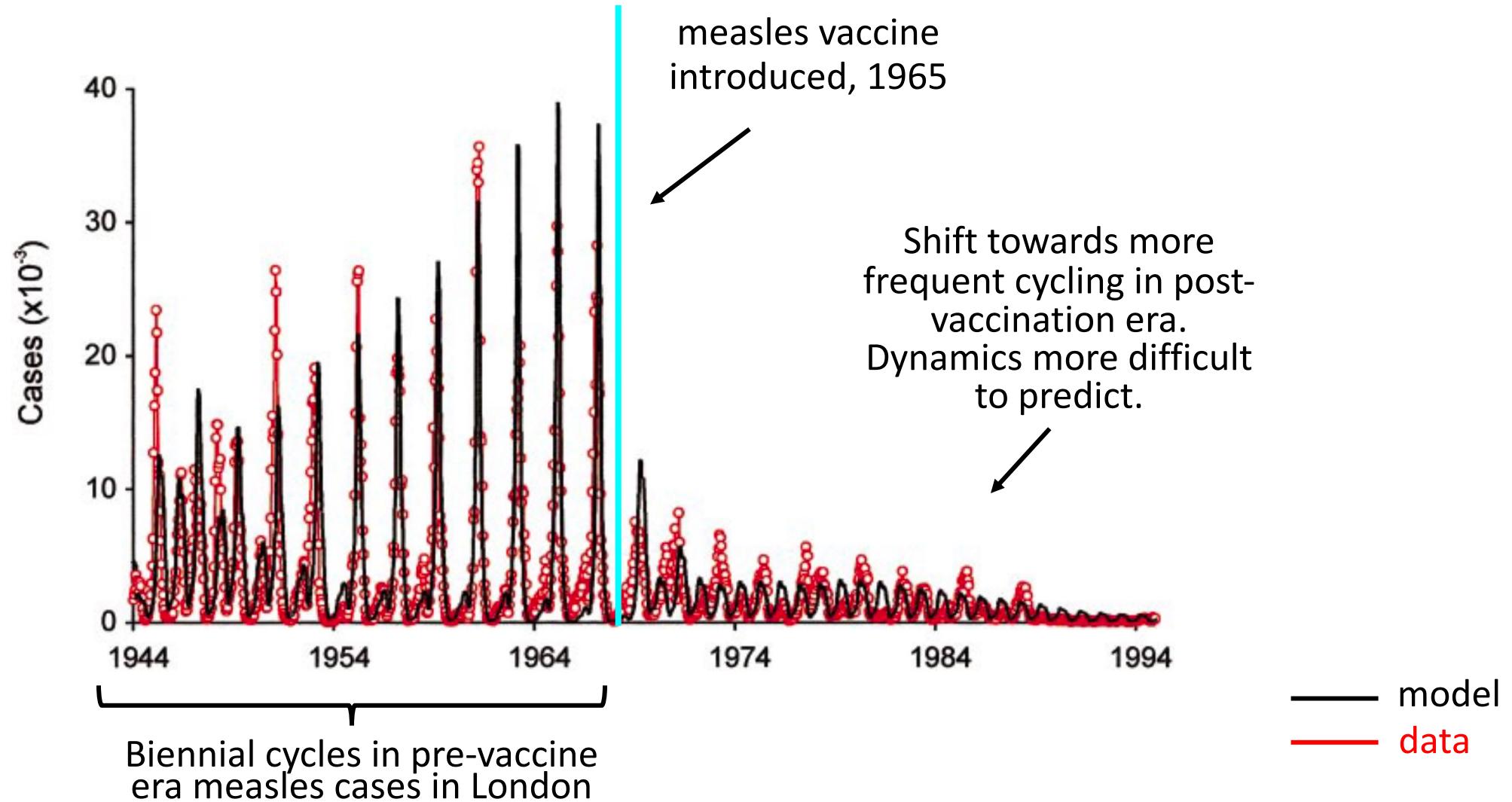
- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access
- Continuous births
- Animal reservoirs
- Spatial structure (metapopulation rescue)



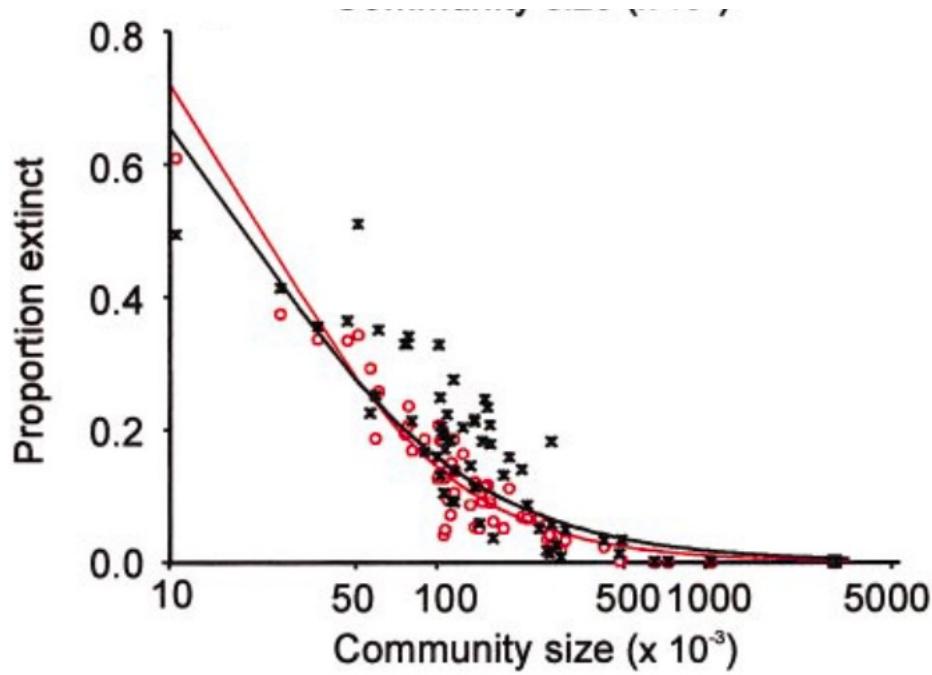
Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens
- Geographic differences in public health policy and access
- Continuous births
- Animal reservoirs
- Spatial structure (metapopulation rescue)
- More complex pathogens!

Much of the mathematical theory underlying vaccination was first developed for measles



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

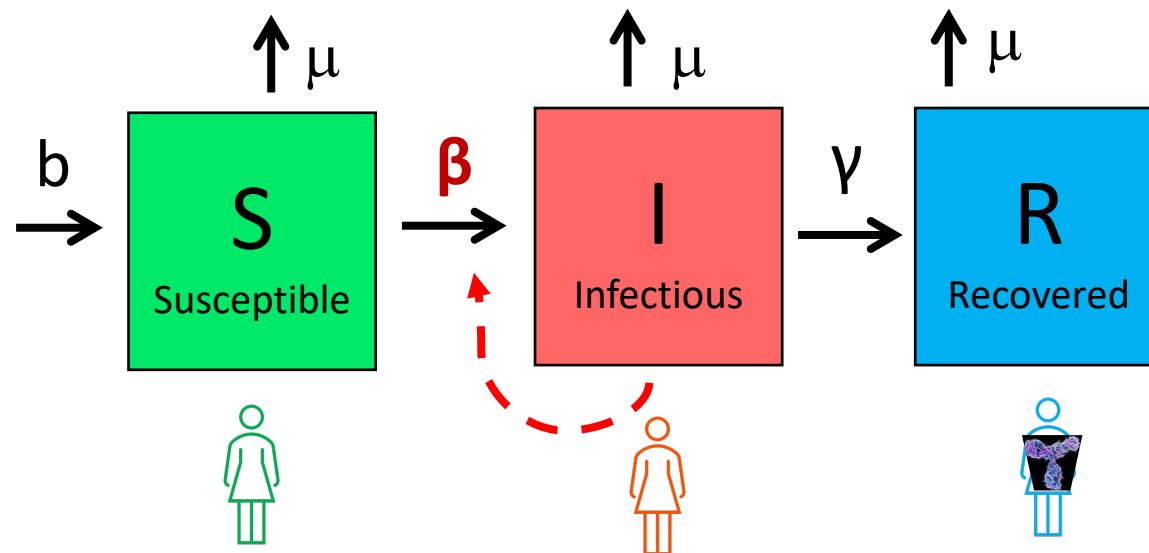


We can adapt the simple SIR model to better match our pathogen of interest and our corresponding data.

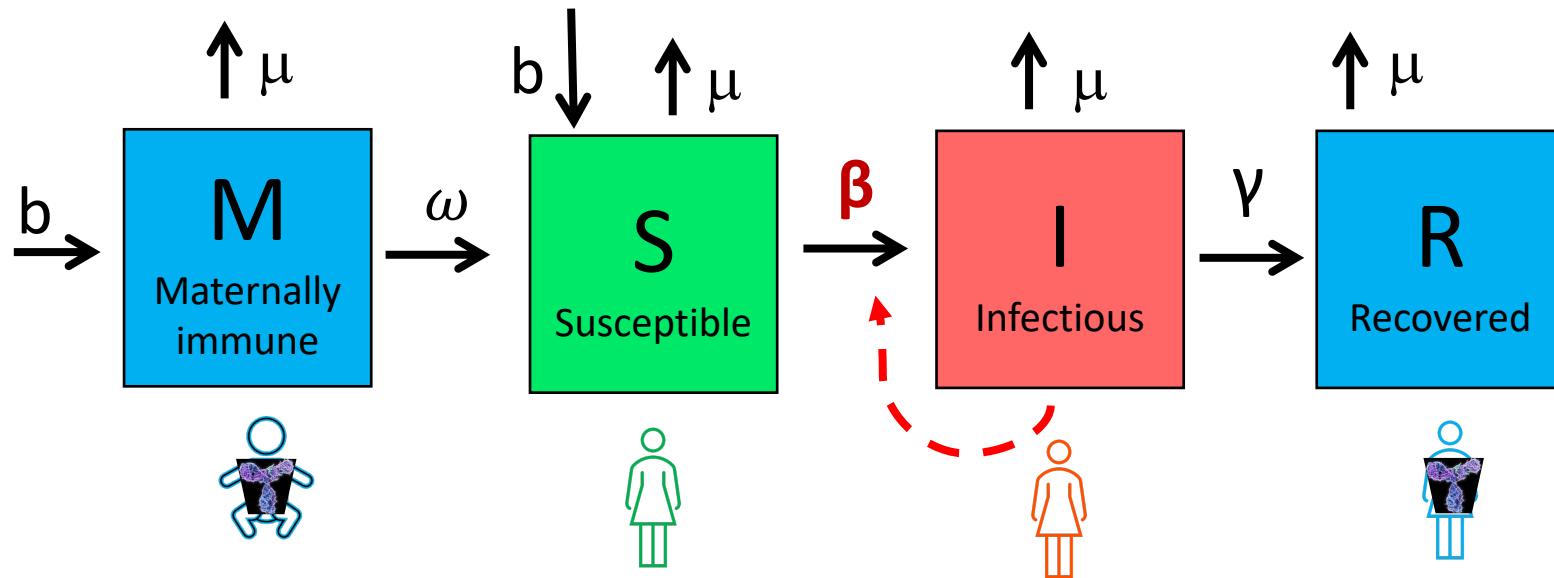
$$\frac{dS}{dt} = b(S + I + R) - \beta SI - \mu S$$

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

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



Incorporating Maternal Immunity



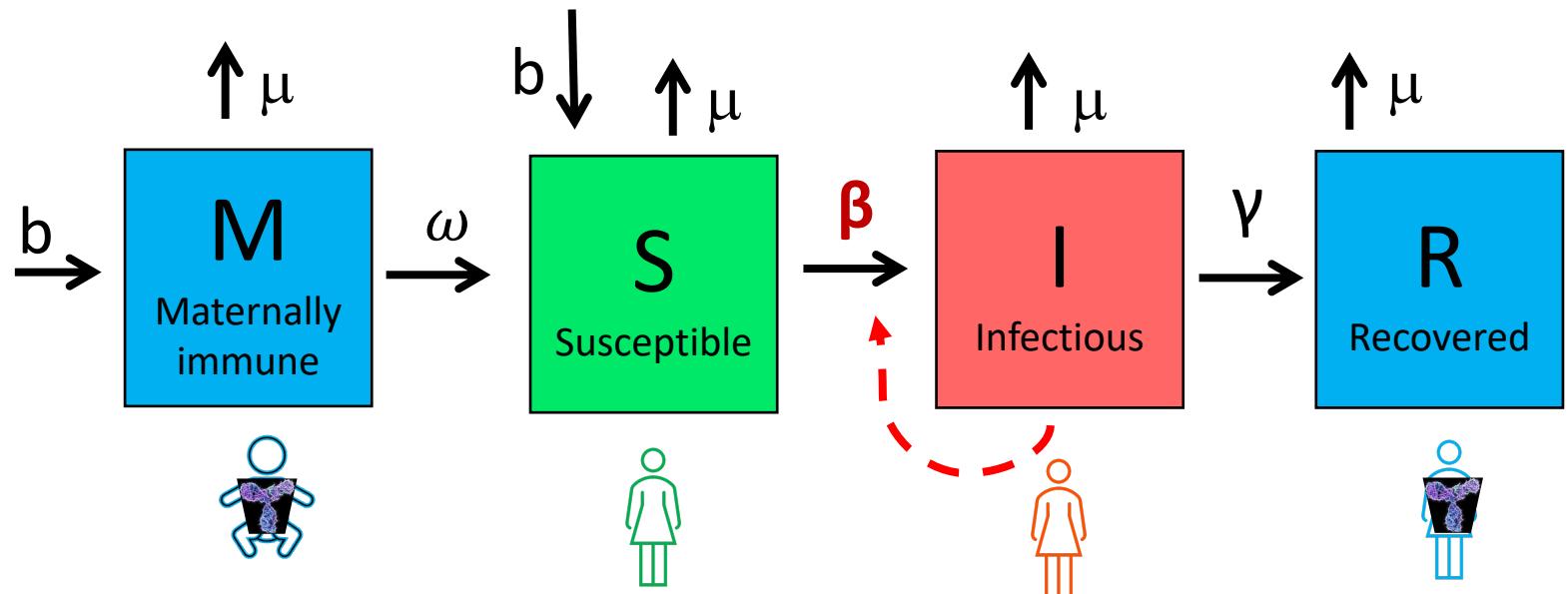
Incorporating Maternal Immunity

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

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

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

What does our new equation look like?



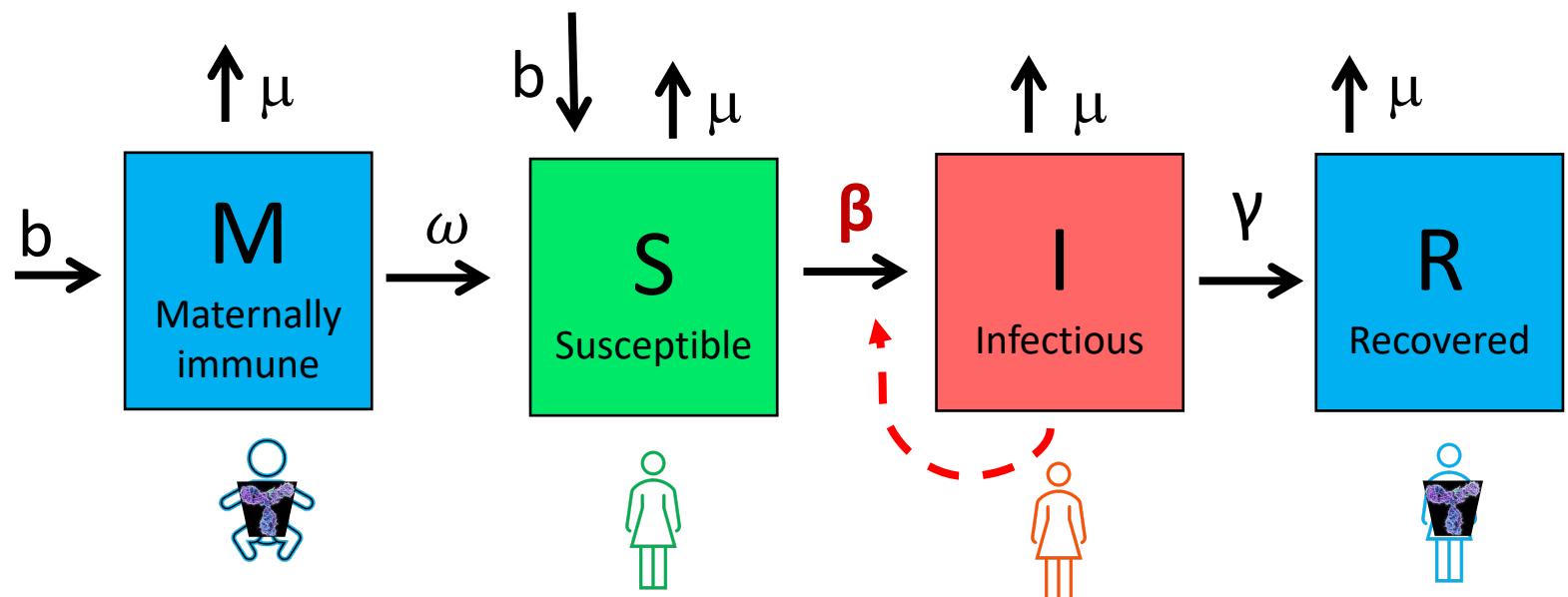
Incorporating Maternal Immunity

$$\frac{dM}{dt} = b(I + R) - \omega M - \mu M$$

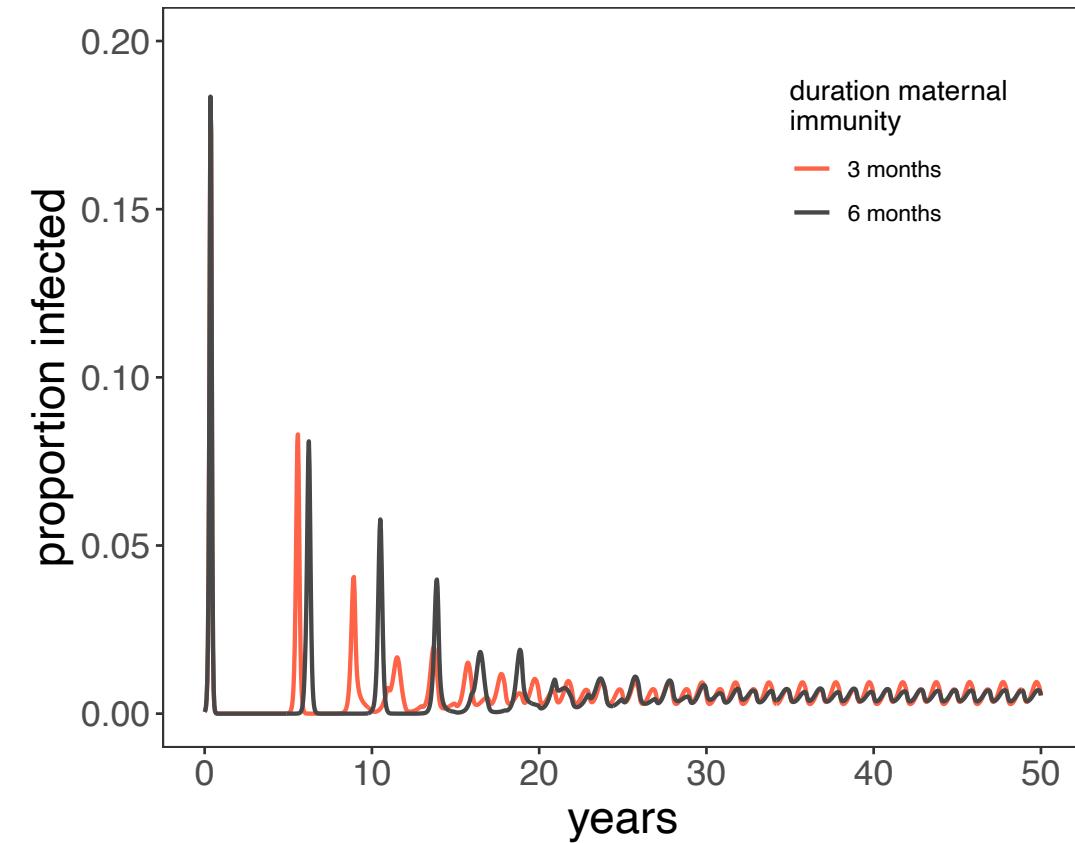
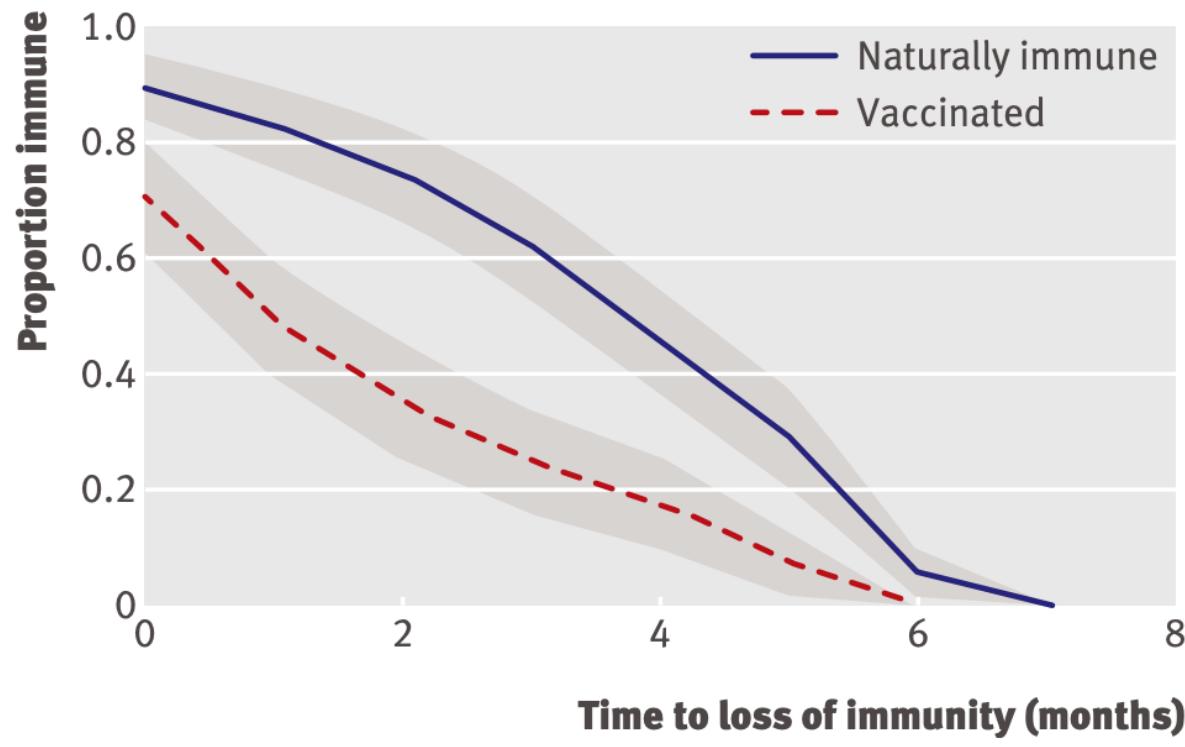
$$\frac{dS}{dt} = \omega M + bS - \beta SI - \mu S$$

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

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



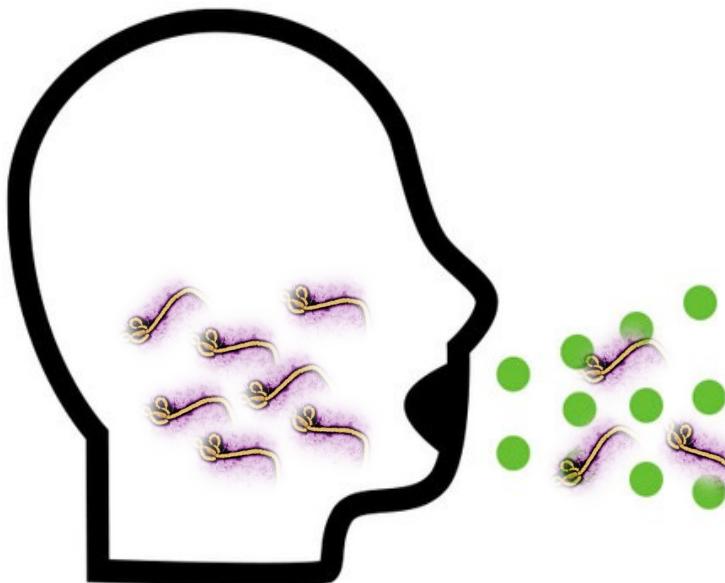
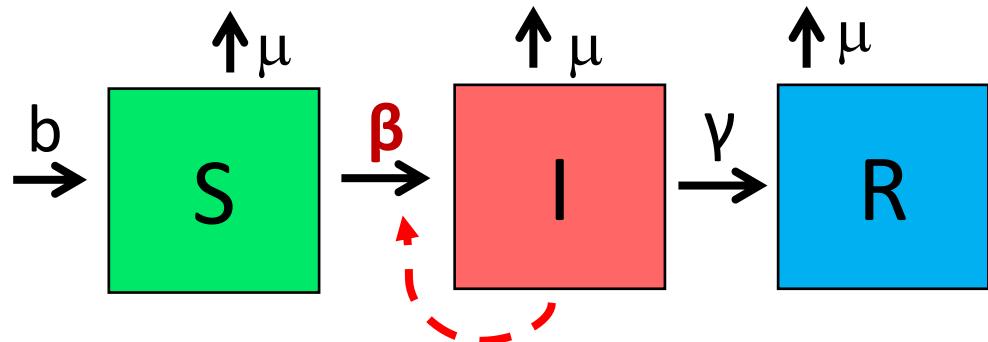
Duration of maternal immunity for measles for naturally infected vs. vaccinated mothers → will impact dynamical predictions!



incorporating maternal immunity

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

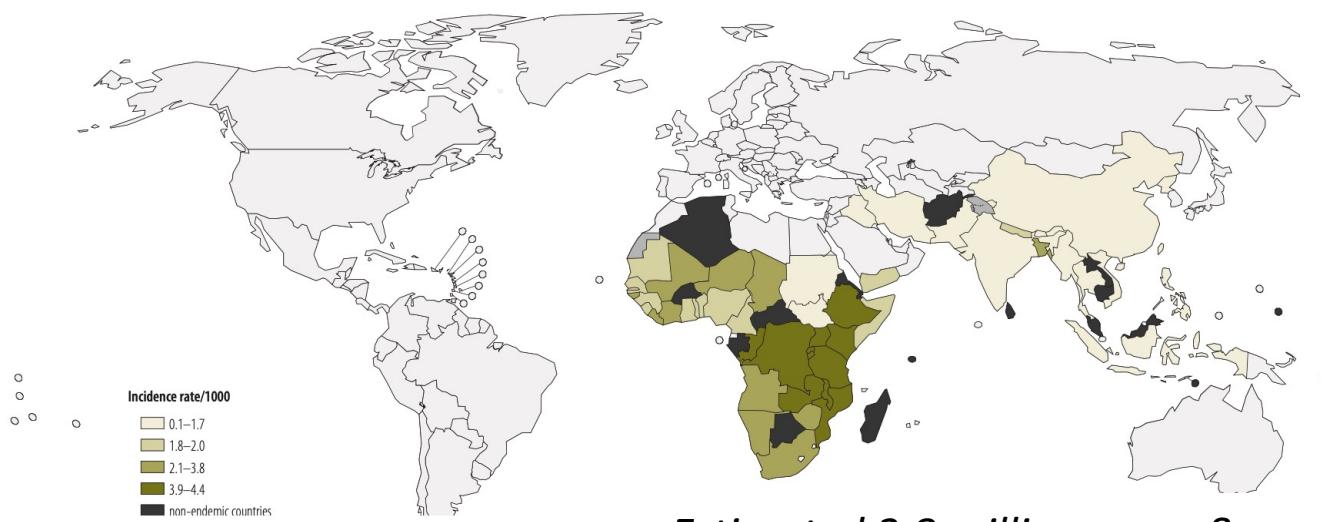
- **Directly-transmitted** diseases are transmitted via exchange of bodily fluids
 - Droplet (> 5 microns) spread or direct contact
 - Includes sexually-transmitted pathogens, though often modeled with a more complex contact network
 - Smallpox (*Variola* spp.), HIV, Mononucleosis (*Epstein Barr virus*)
- **Indirectly-transmitted** diseases are transmitted via droplets retained in air
 - Droplets < 5 microns in diameter
 - Measles, COVID (SARS-CoV-2)



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

- **Environmentally-transmitted** pathogens are transmitted outside of the host (e.g. water-borne, food-borne)
 - Examples: **Cholera (*Vibrio cholerae*)**, Salmonellosis (*Salmonella* spp. bacteria), White-Nosed Syndrome (*Pseudogymnoascus destructans*)

Global Burden of Cholera, 2012



*Estimated 2.8 million cases &
95,000 deaths annually*



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

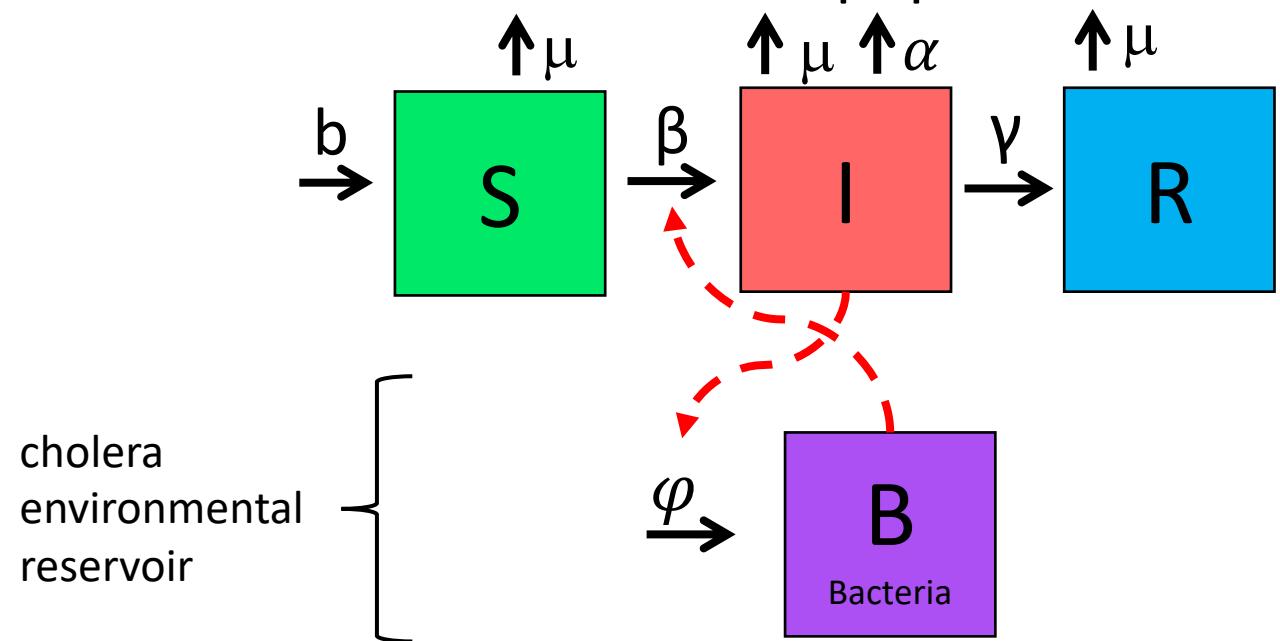
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 - Examples: Cholera (*Vibrio cholerae*), Salmonellosis (*Salmonella* spp. bacteria), White-Nosed Syndrome (*Pseudogymnoascus destructans*)
 - Here, the **environmental reservoir** is often modeled as its own population

$$\frac{dB}{dt} = \varphi IB$$

$$\frac{dS}{dt} = b(S + I + R) - \beta SB - \mu S$$

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

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



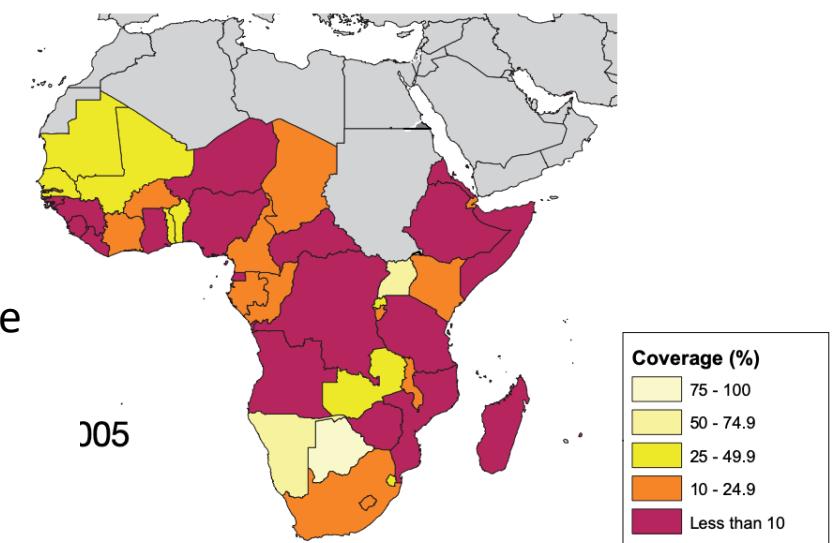
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- **Vertically-transmitted** pathogens are transmitted mother-to-child *in utero*
 - Examples: **HIV**, *Herpes simplex virus*, *Cytomegalovirus*, Rubella, Zika



- In untreated HIV+ mothers, rate of vertical transmission for HIV = 15-45%
- Reduced to <1% for those on ART, though global access to ART is geographically heterogeneous

ART coverage
for those in
need, from
WHO



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)
 - Euclidean **vector**: a quantity with a magnitude and direction
→
 - Epidemiological **vector**: an agent that carries and transmits an infectious patient into another living organism



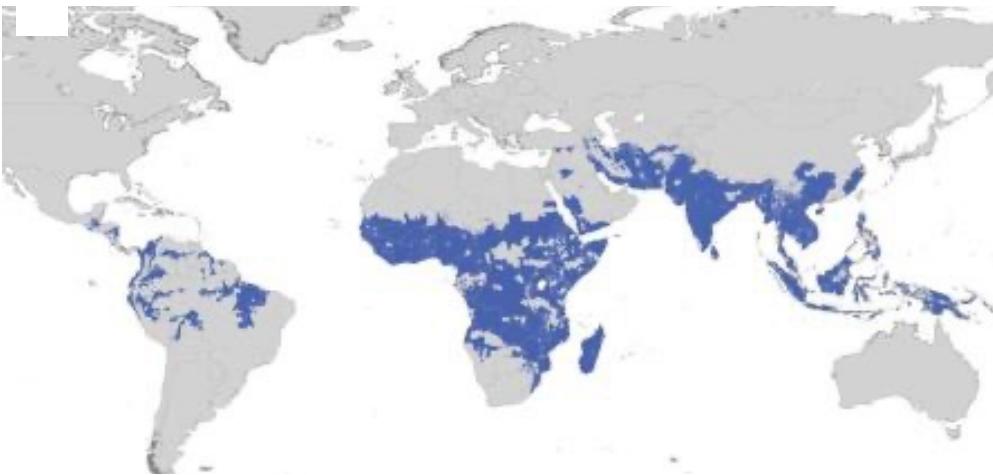
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 - Malaria: Mosquito-borne protozoan *Plasmodium spp.*
 - “Arboviruses”: Mosquito-borne viruses, including Dengue, Zika, Yellow fever virus, West Nile virus, Chikungunya virus
 - Sleeping sickness, also known as African trypanosomiasis: tsetse fly vector and protozoan pathogen (trypanosome)
 - Chagas disease: kissing bug vector and trypanosome pathogen
 - Plague: flea vector and bacterial pathogen (*Yersinia pestis*)

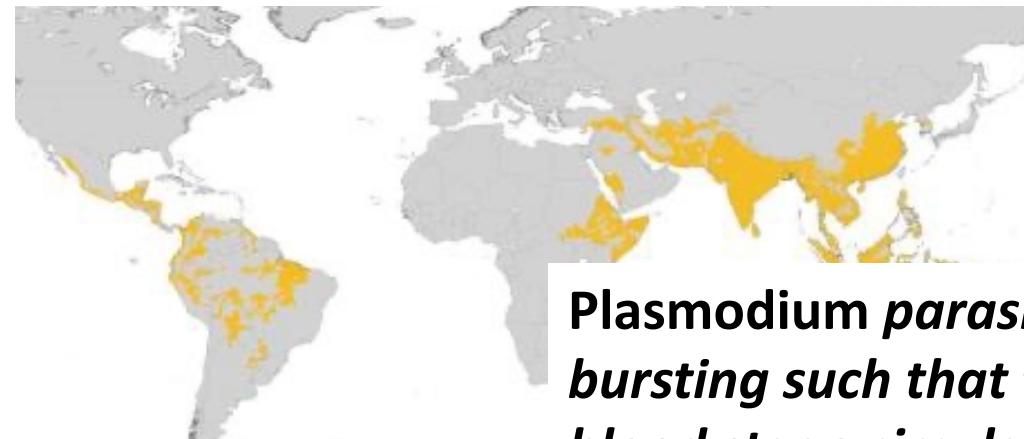
Malaria

- 4 main human *Plasmodium* parasites (*falciparum*, *vivax*, *malariae*, *ovalae*).
- Over 200 *Plasmodium* spp. globally, infecting birds, reptiles, and other mammals (rodents, bats, primates)

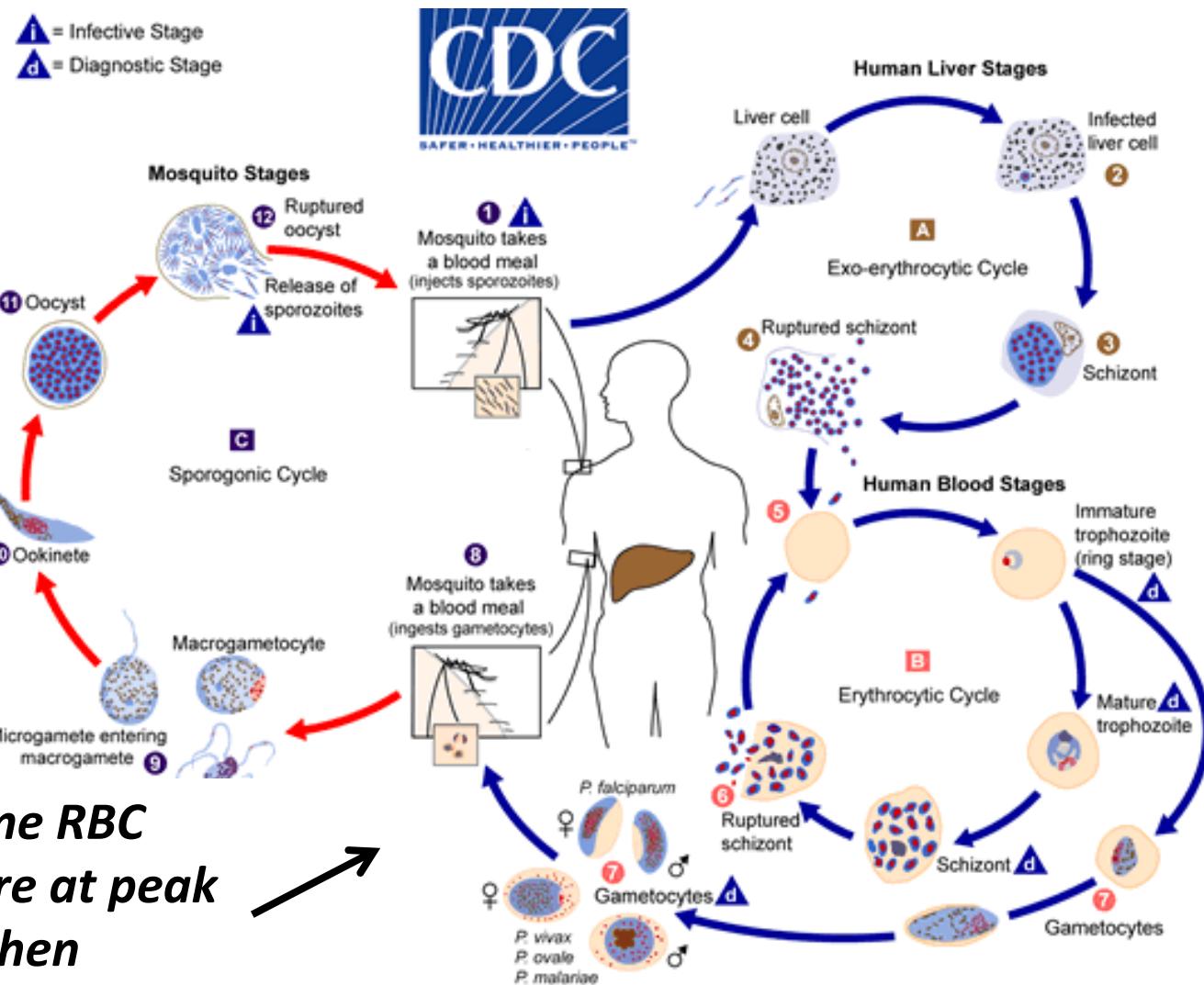
Distribution *Plasmodium falciparum*



Distribution *Plasmodium vivax*



Plasmodium parasites time RBC bursting such that they are at peak blood stage circulation when mosquito vectors are feeding at dusk!



Malaria

- 4 main human *Plasmodium* parasites (*falciparum*, *vivax*, *malariae*, *ovalae*).
- Over 200 *Plasmodium* spp. globally, infecting birds, reptiles, and other mammals (rodents, bats, primates)
- >400 global species of *Anopheles* mosquito, >100 that can transmit human malaria
- ~30-40 *Anopheles* spp. most commonly implicated in human malaria transmission!

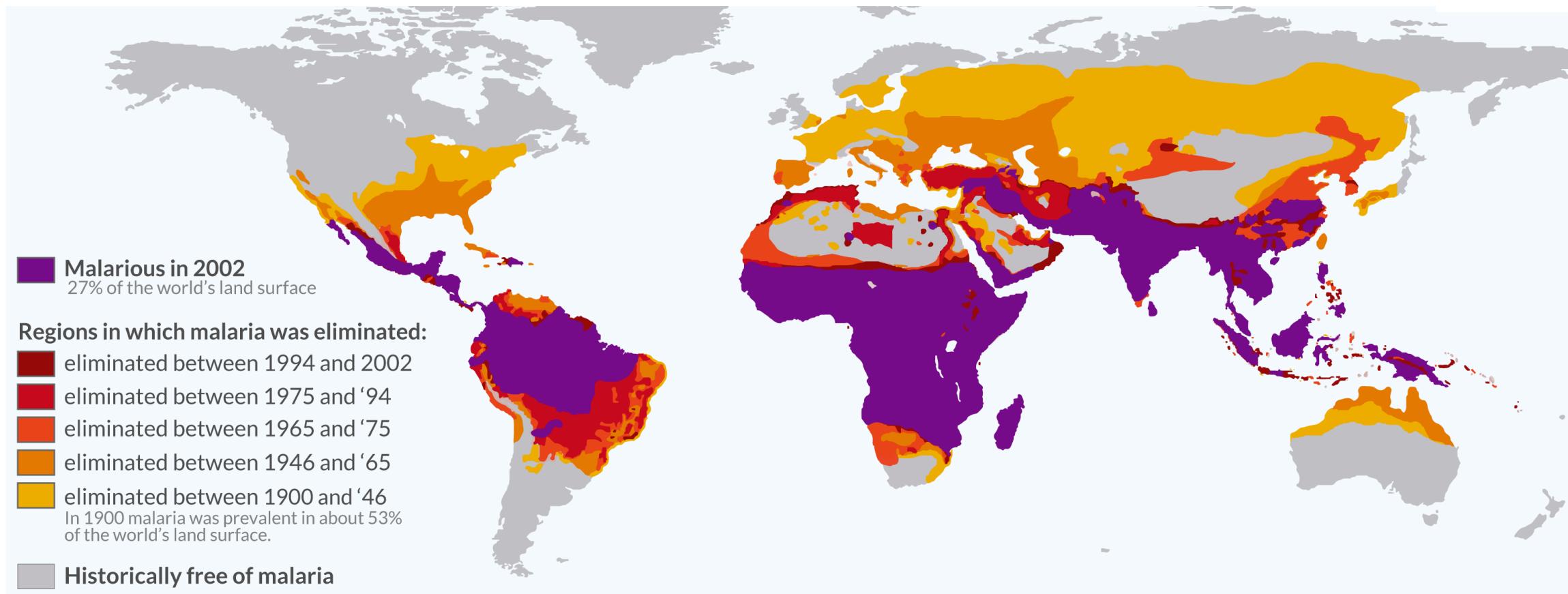


Only female mosquitoes feed on blood!
Male mosquitoes feed on plant nectar.



Anopheles	
No vector	
albimanus	funestus and arabiensis
annularis	melas
anthropophagus	barbirostris
arabiensis	funestus, arabiensis and gambiae s.s.
arabiensis and funestus	culicifacies
aquasalis	funestus and gambiae s.s.
atroparvus	dirus
	gambiae s.s.
	farauti
	gambiae s.s. and funestus
	flavirostris
	labranchiae
	nunez-tovari
	darlingi and marajoara
	pseudopunctipennis
	freeborni
	quadrivittatus
	minimus
	sacharovi
	multicolor
	superpictus
	sinensis
	pharaohensis
	maculatus
	fluvialis
	darlingi and marajoara

Malaria has been eliminated from many regions where it was previously endemic, including the US.



Still one of the leading causes of child mortality globally – responsible for about half a million childhood deaths a year, 80% in Africa.

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

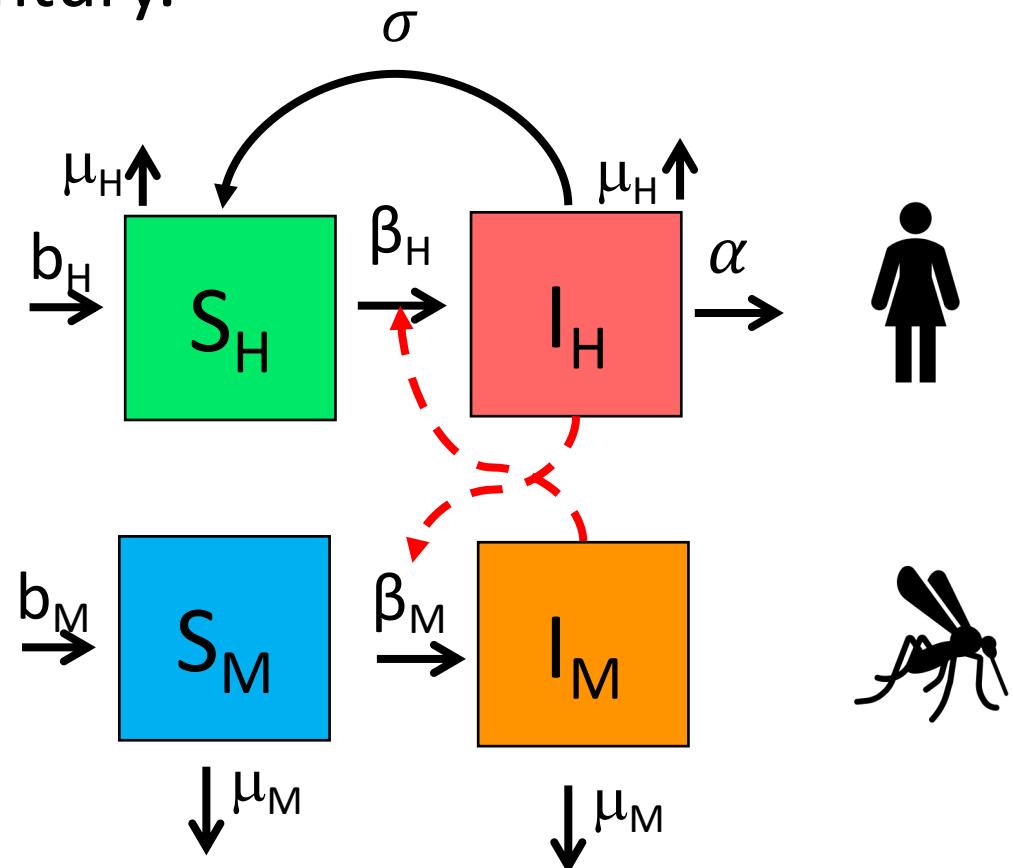
- 1911: British medical Dr. Sir Ronald Ross developed the first model of malaria while working in the Indian Medical Service.
 - He had already won the 1902 Nobel prize in physiology and medicine for discovering the life cycle of avian malaria

$$\frac{dS_H}{dt} = b_H(S_H + I_H) + \sigma I_H - \beta_H S_H I_M - \mu_H S_H$$

$$\frac{dI_H}{dt} = \beta_H S_H I_M - \sigma I_H - \mu_H I_H - \alpha I_H$$

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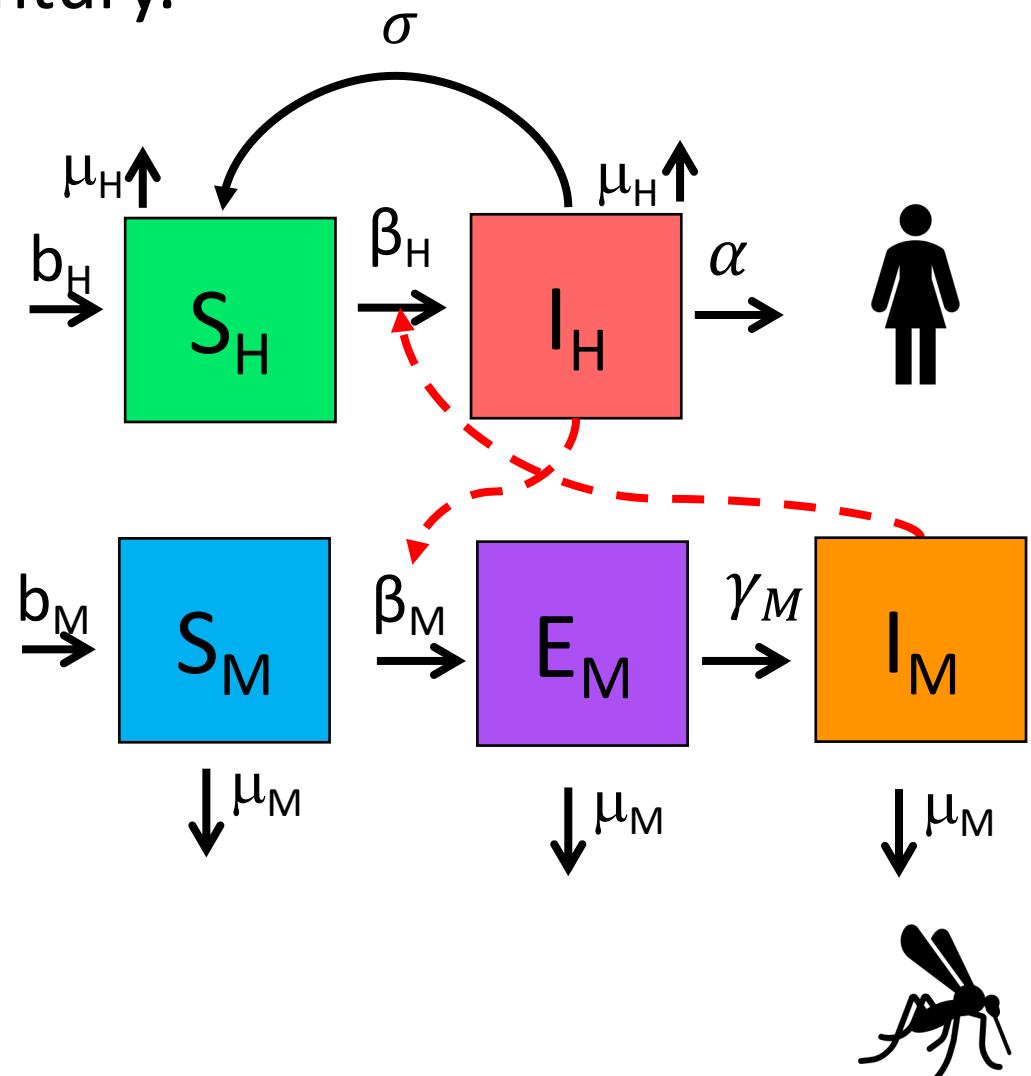
Malaria models have played a critical role in public health policy for over a century.

- 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 developing in the mosquito.
 - He implicated the survivorship of the female mosquito as the weakest link in the life cycle!

$$\frac{dS_M}{dt} = b_M(S_M + E_M + I_M) - \beta_M S_M I_H - \mu_M S_M$$

$$\frac{dE_M}{dt} = \beta_M S_M I_H - \mu_M E_M - \gamma_M E_M$$

$$\frac{dI_M}{dt} = \gamma_M E_M - \mu_M I_M$$



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 developing in the mosquito.
- This led to a widespread WHO campaign for malaria elimination using DDT in the 1950s!
 - Swiss chemist Paul Müller awarded 1948 Nobel Prize in medicine and physiology for discovering DDT and its impacts on arthropods
 - DDT played a major role in eliminating malaria from Europe and North America
 - DDT bioaccumulates and is carcinogenic – led to development of an environmental backlash in the US, culminating in Rachel Carson's 1962 book *Silent Spring*
 - DDT banned globally in 2004, excepting in cases of WHO-recommended indoor residual spraying (IRS) for vector control in malaria-endemic regions



“Ecology has a synonym which is ALL.”

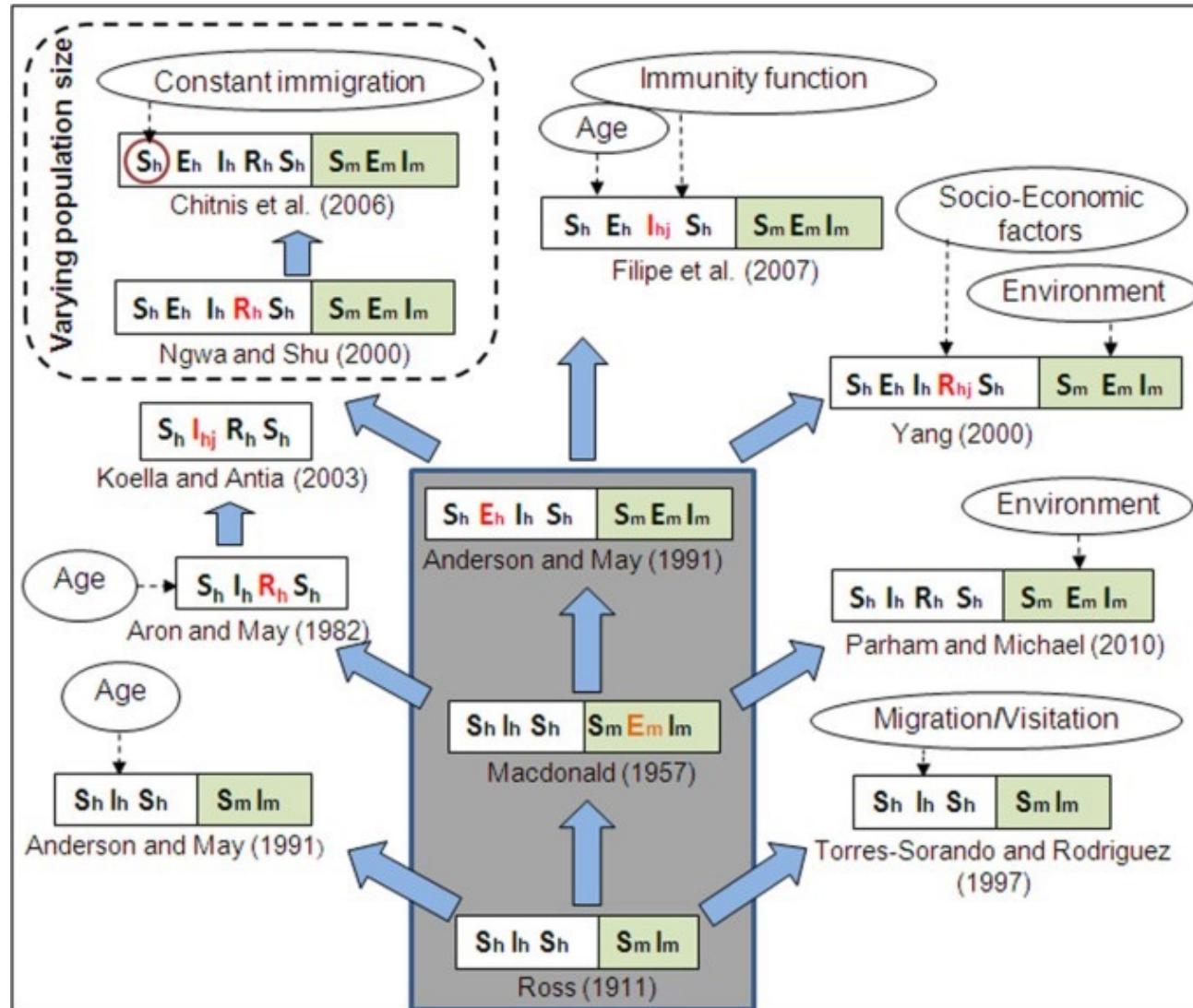
-John Steinbeck

The Log from the Sea of Cortez (1941)

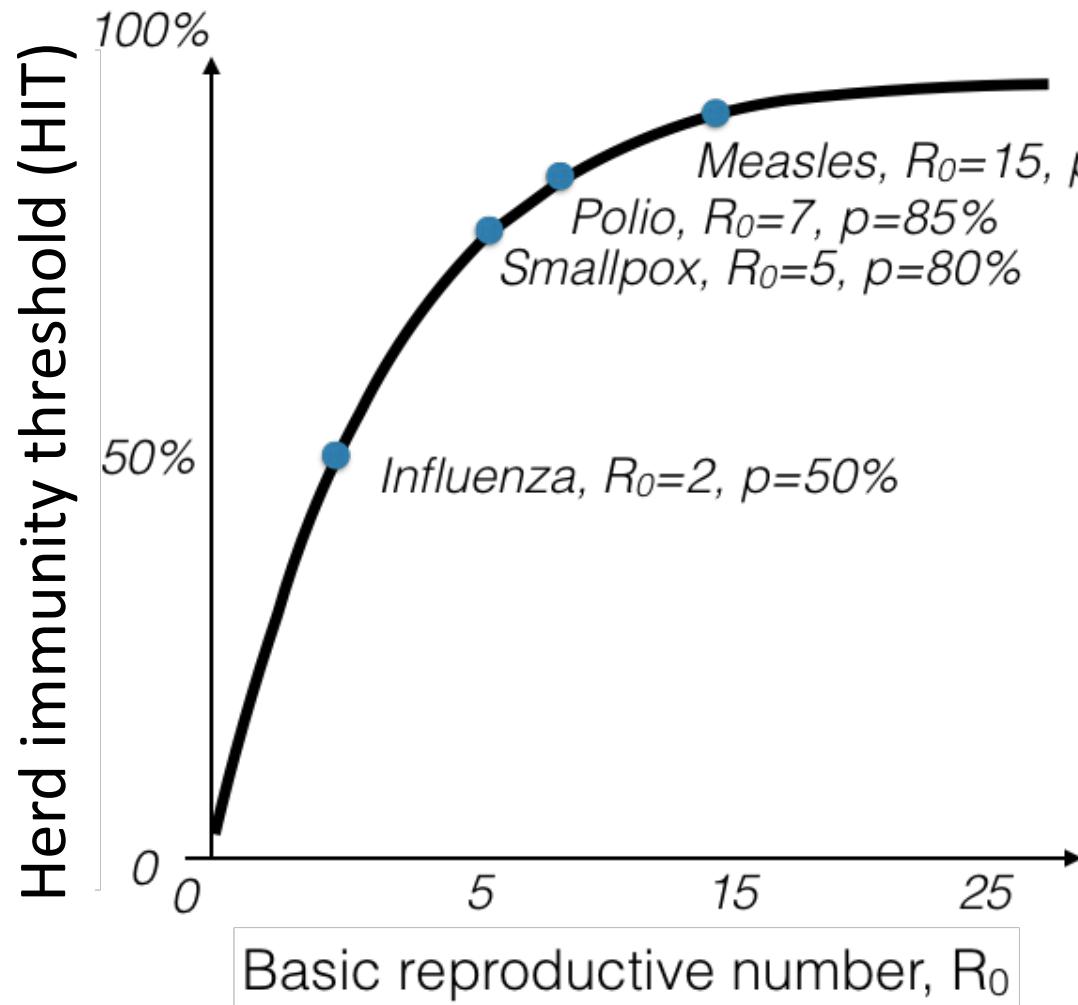
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- 1991: Anderson and May extended model to show latency in the human population.

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



Challenges to malaria elimination

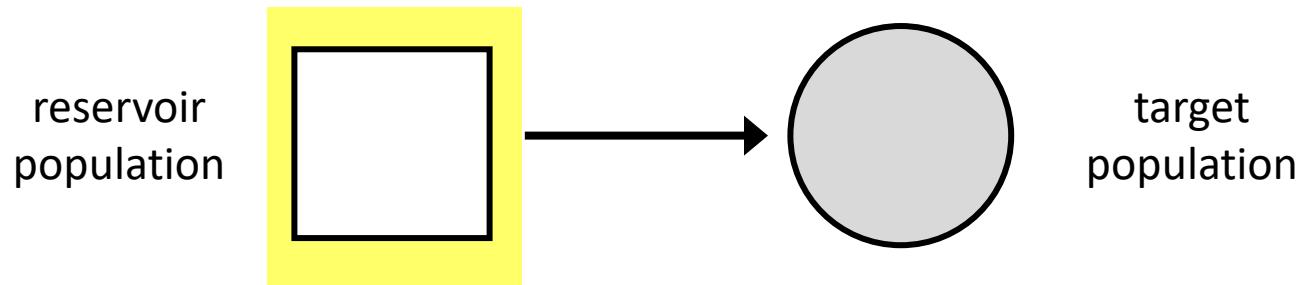


- High parasite diversity: sexual reproduction in 4+ *Plasmodium* species
 - Fast evolution of resistance (e.g. to drugs)
- Many possible vectors! Potentially additional possible reservoirs!
- Latent cases as burden is reduced
- Vaccine candidates: what life stage to target?

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

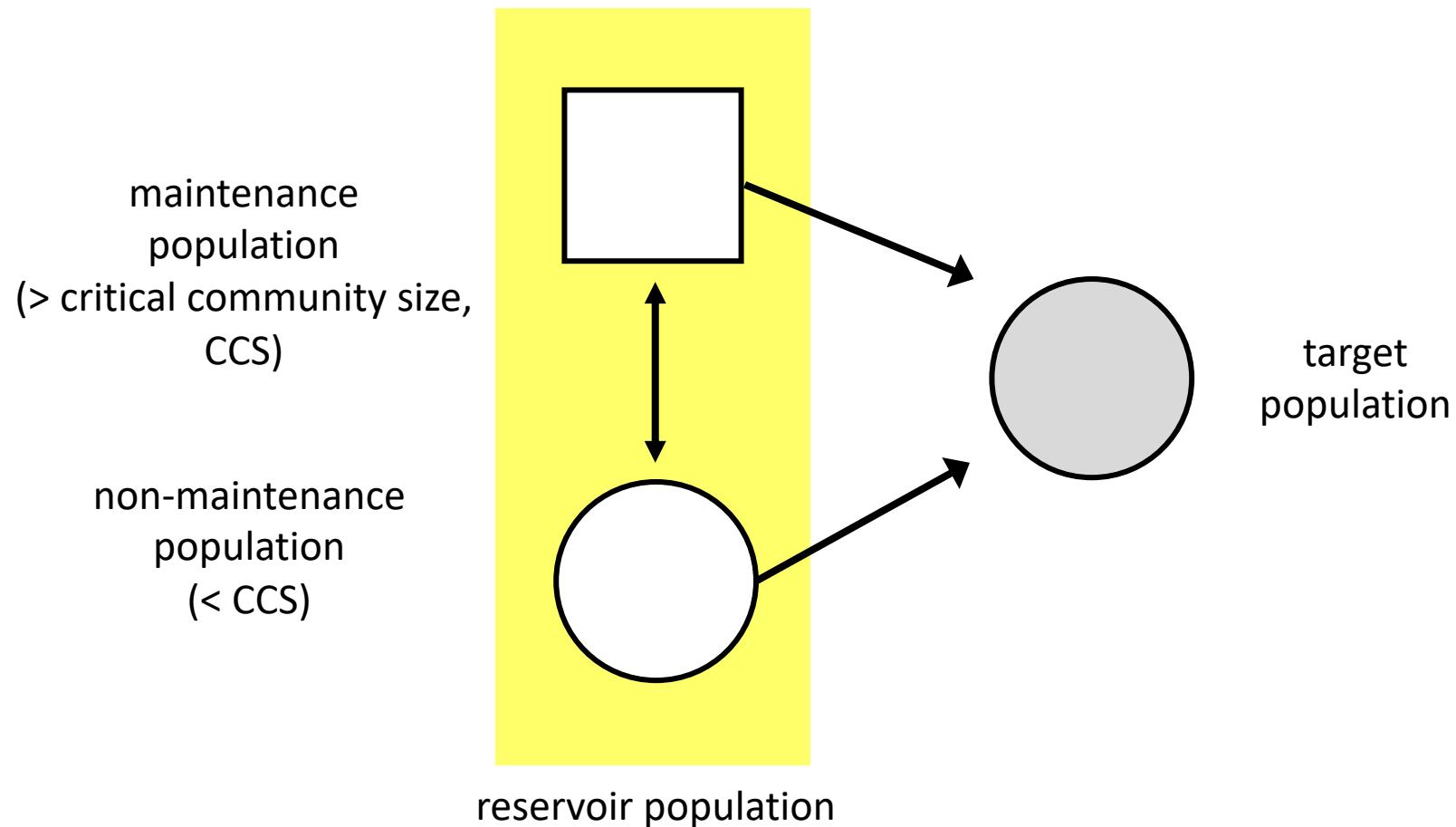
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 - Sleeping sickness, also known as African trypanosomiasis: tsetse fly vector and protozoan pathogen (trypanosome)
 - Chagas disease: kissing bug vector and trypanosome pathogen
 - **Plague**: flea vector and bacterial pathogen (*Yersinia pestis*) **Plague is BOTH vector-borne and zoonotic!**

A pathogen **reservoir** sources infections to
a **target** population.



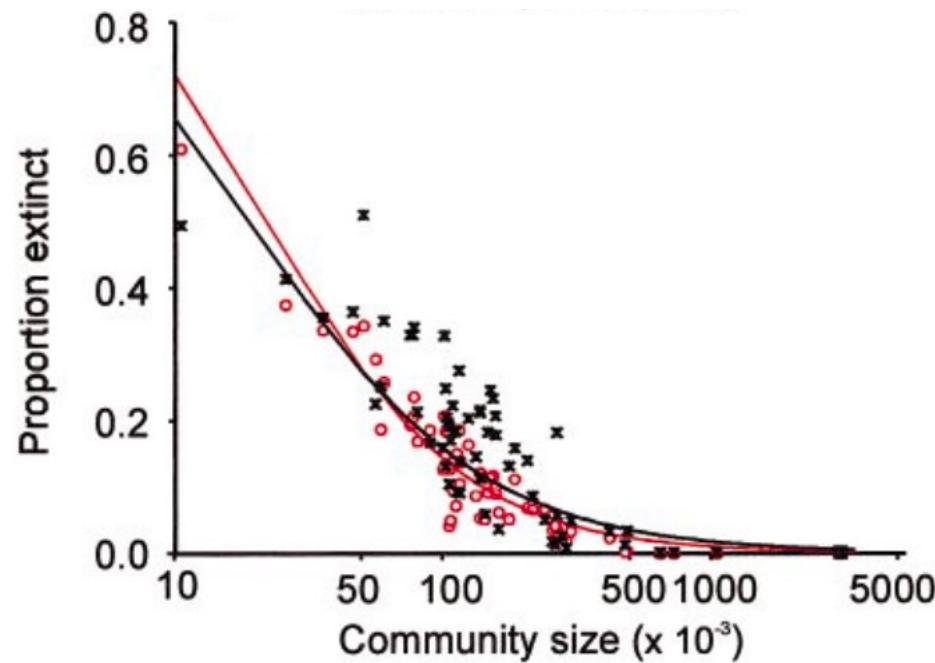
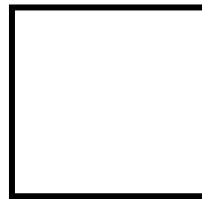
All of these populations are still called **hosts**!
Some are **reservoir hosts** and some are **spillover hosts**.

Reservoir hosts can comprise a single species population or multiple sub-populations of different species.

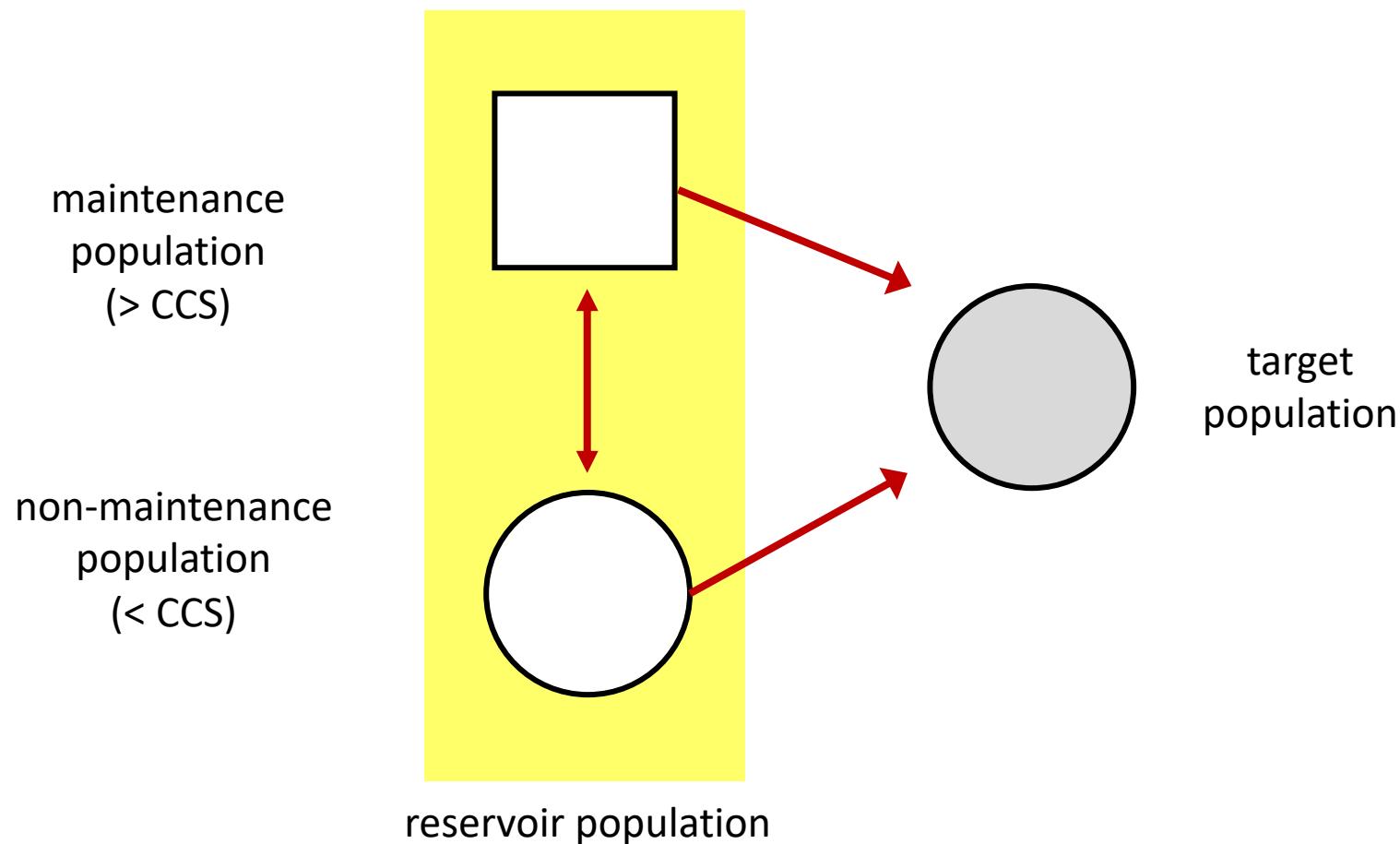


CCS is the **minimum number of hosts** needed to sustain **endemic transmission** of a pathogen indefinitely into the future.

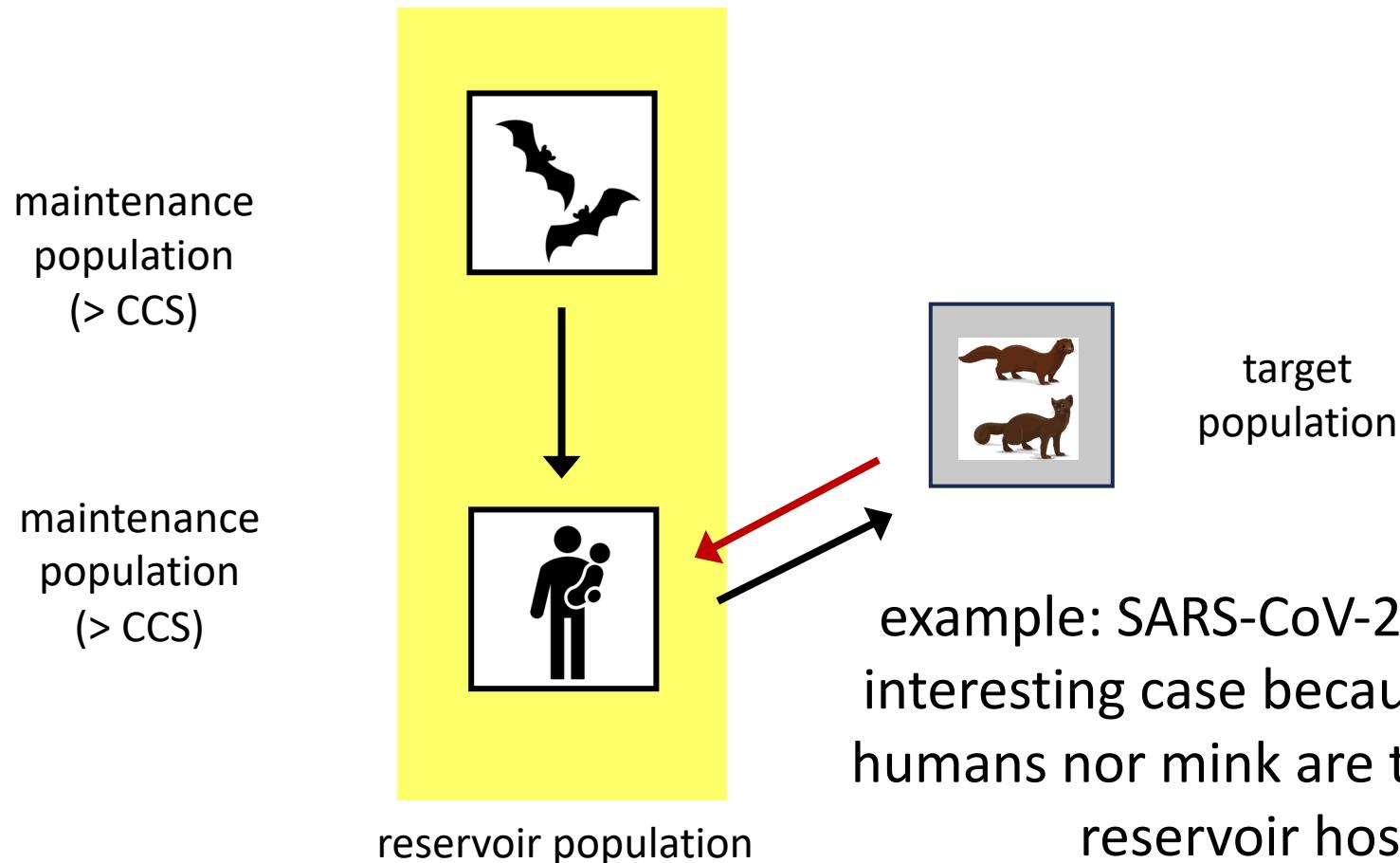
maintenance population
(> critical community size,
CCS)



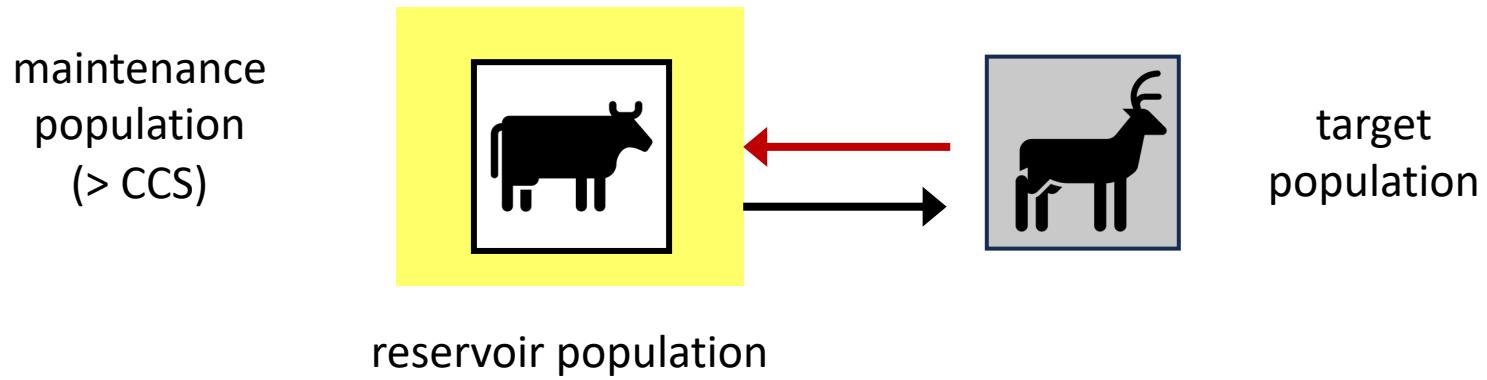
Spillover is the transmission of that pathogen **between populations** of different host species.



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

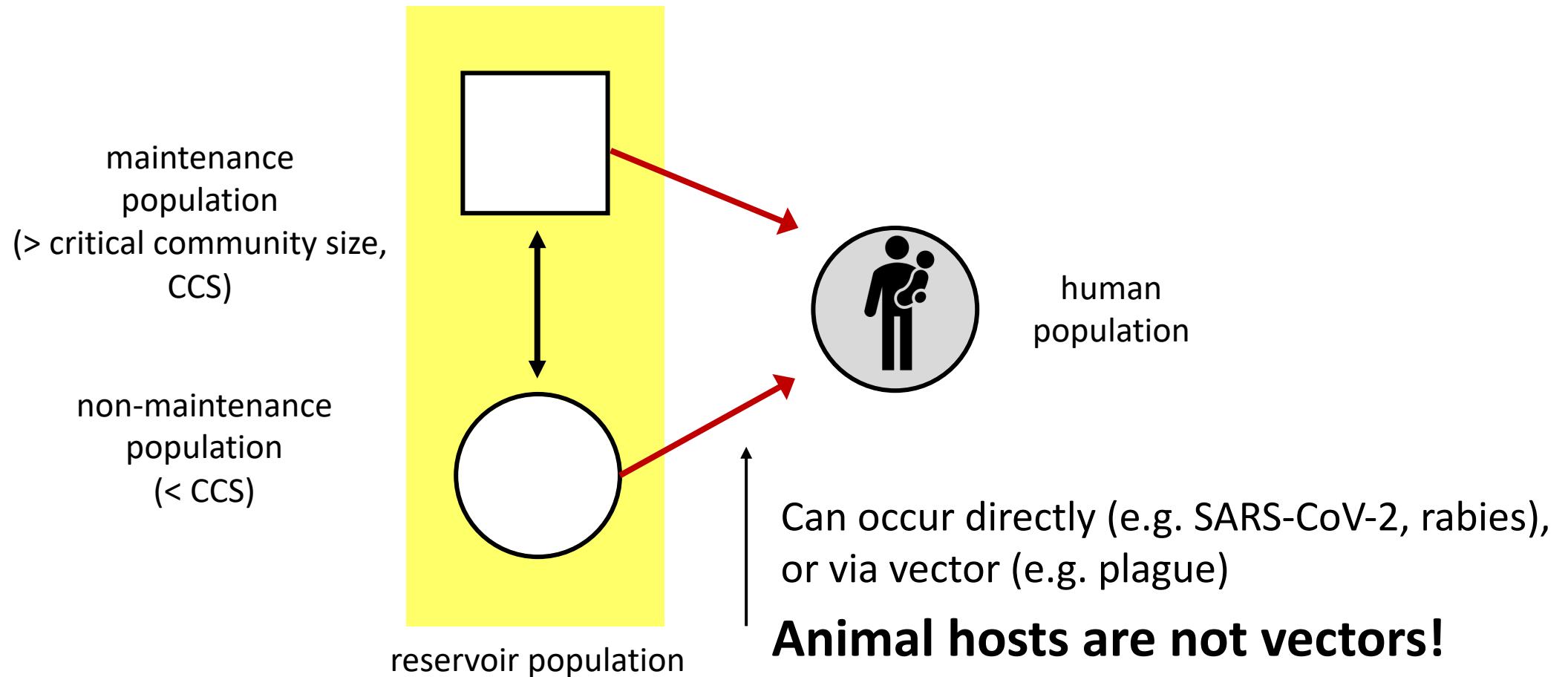


Spillback occurs among wildlife as well.

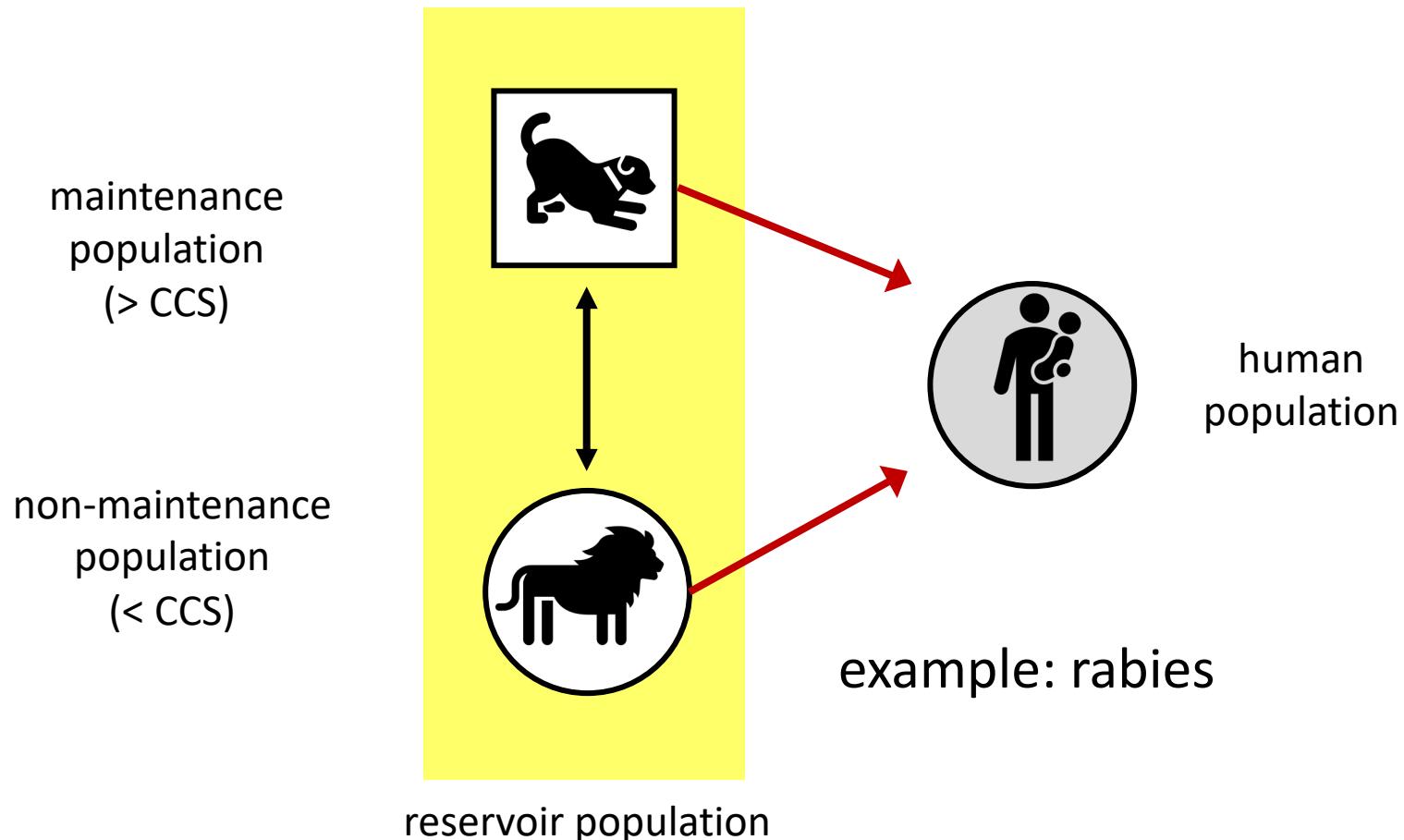


example: Cattle sourced *Brucella* to wild ungulates in Yellowstone National Park, which now serve as a source for reinfection to cattle.

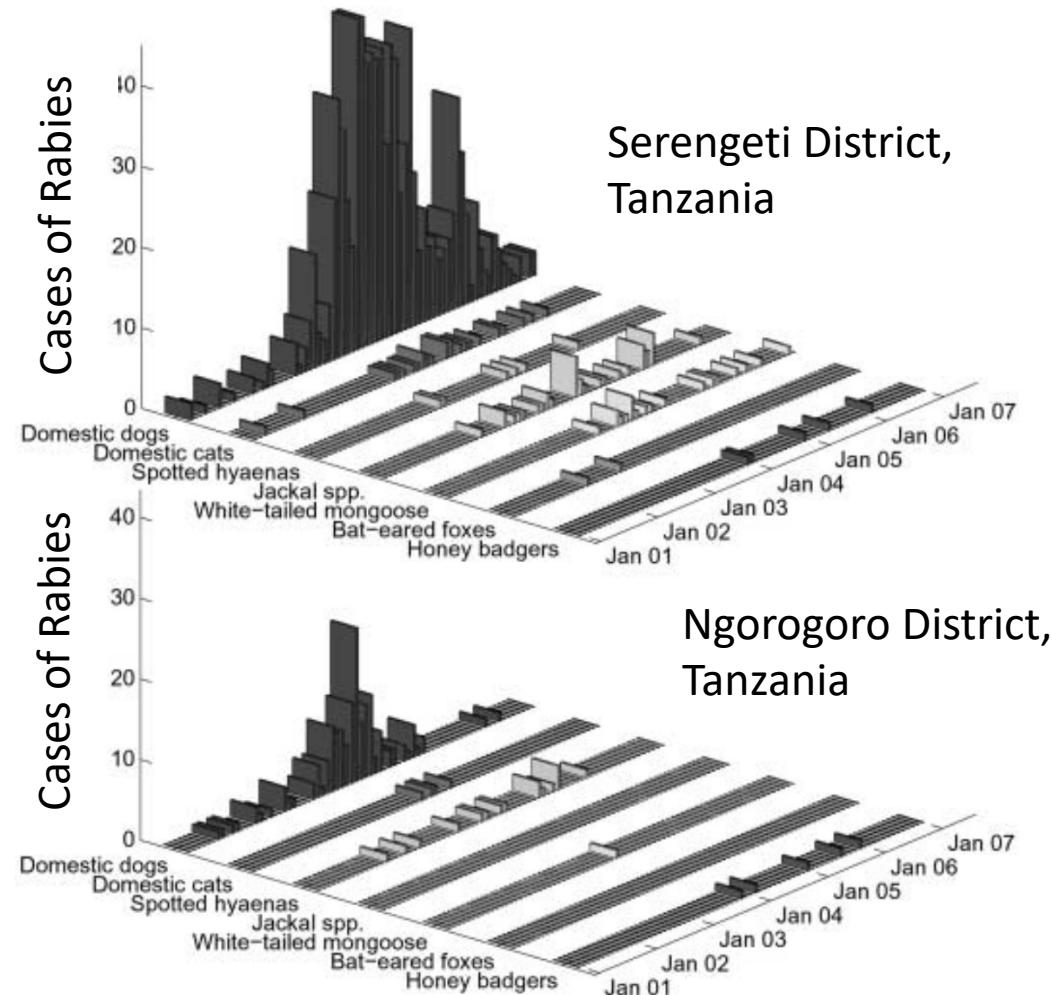
Zoonosis is the transmission of a pathogen from a **wildlife reservoir host** to a **human target host** population.



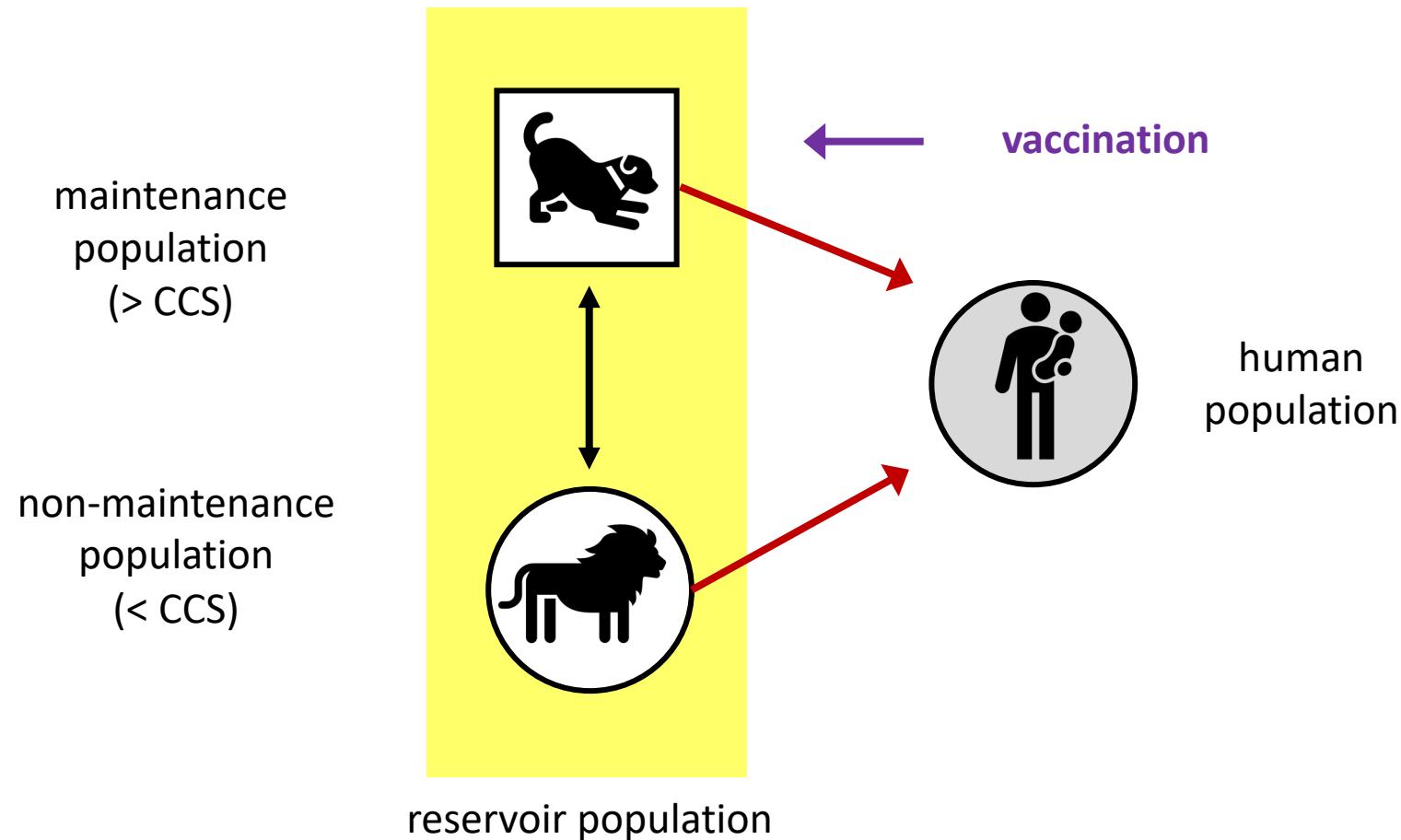
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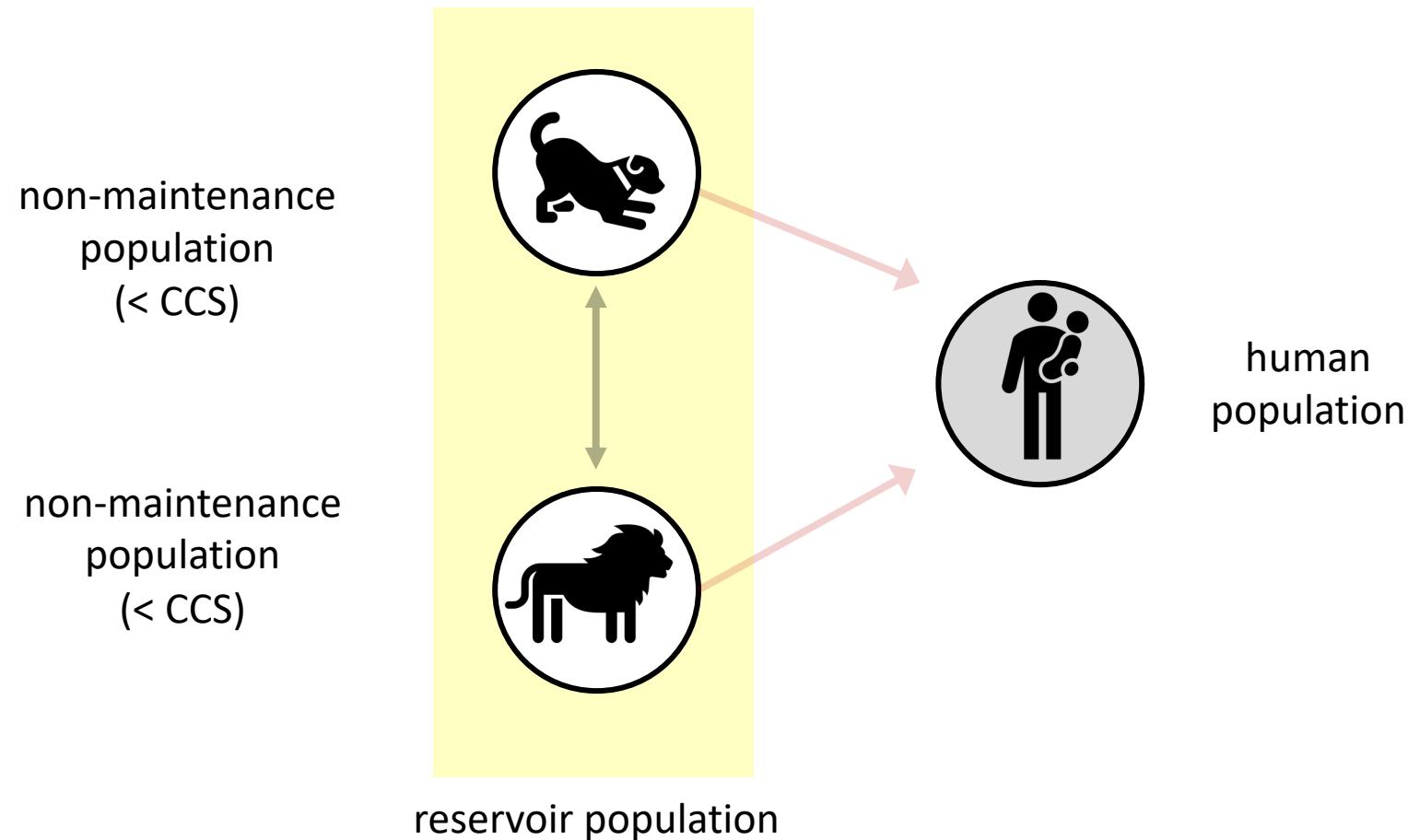
Rabies in the Serengeti is **maintained in domestic dogs** but can spillover to wild African carnivores.



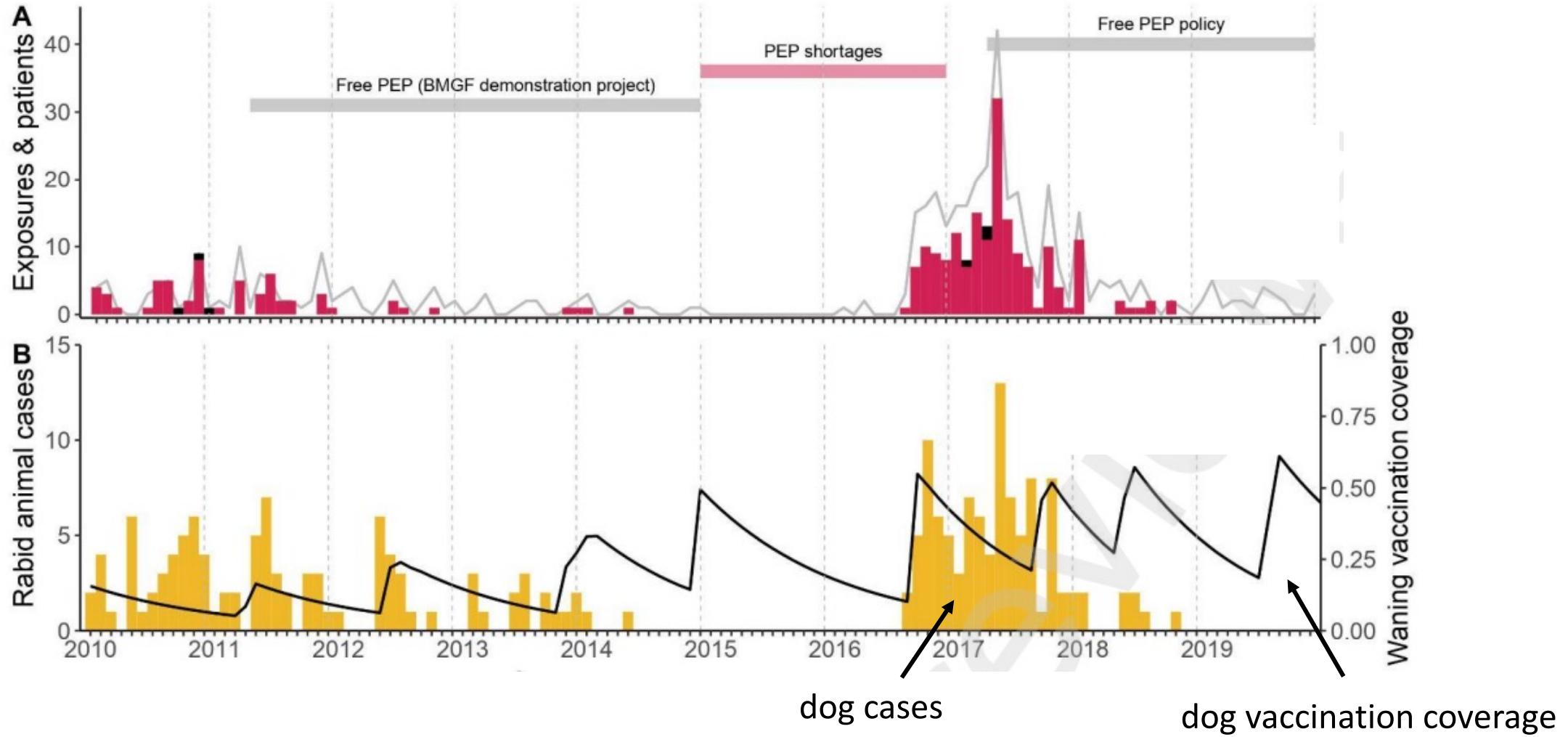
Understanding processes which **maintain zoonoses in reservoirs** **can guide interventions** to reduce spillover to humans.



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Vaccination of domestic dogs can successfully eradicate rabies from some systems.



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

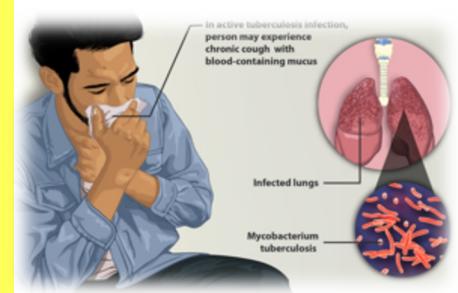
Sustained transmission and human outbreaks



Ebola virus (especially post-2014)

Stage V

Transmits exclusively in humans



Tuberculosis

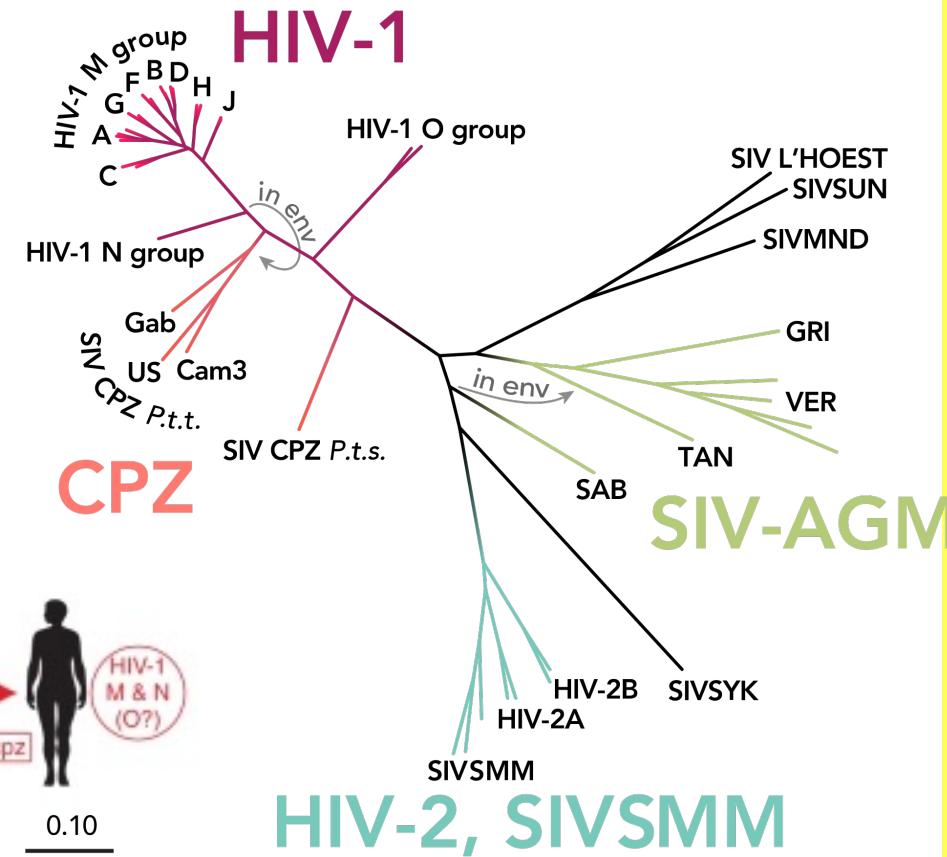
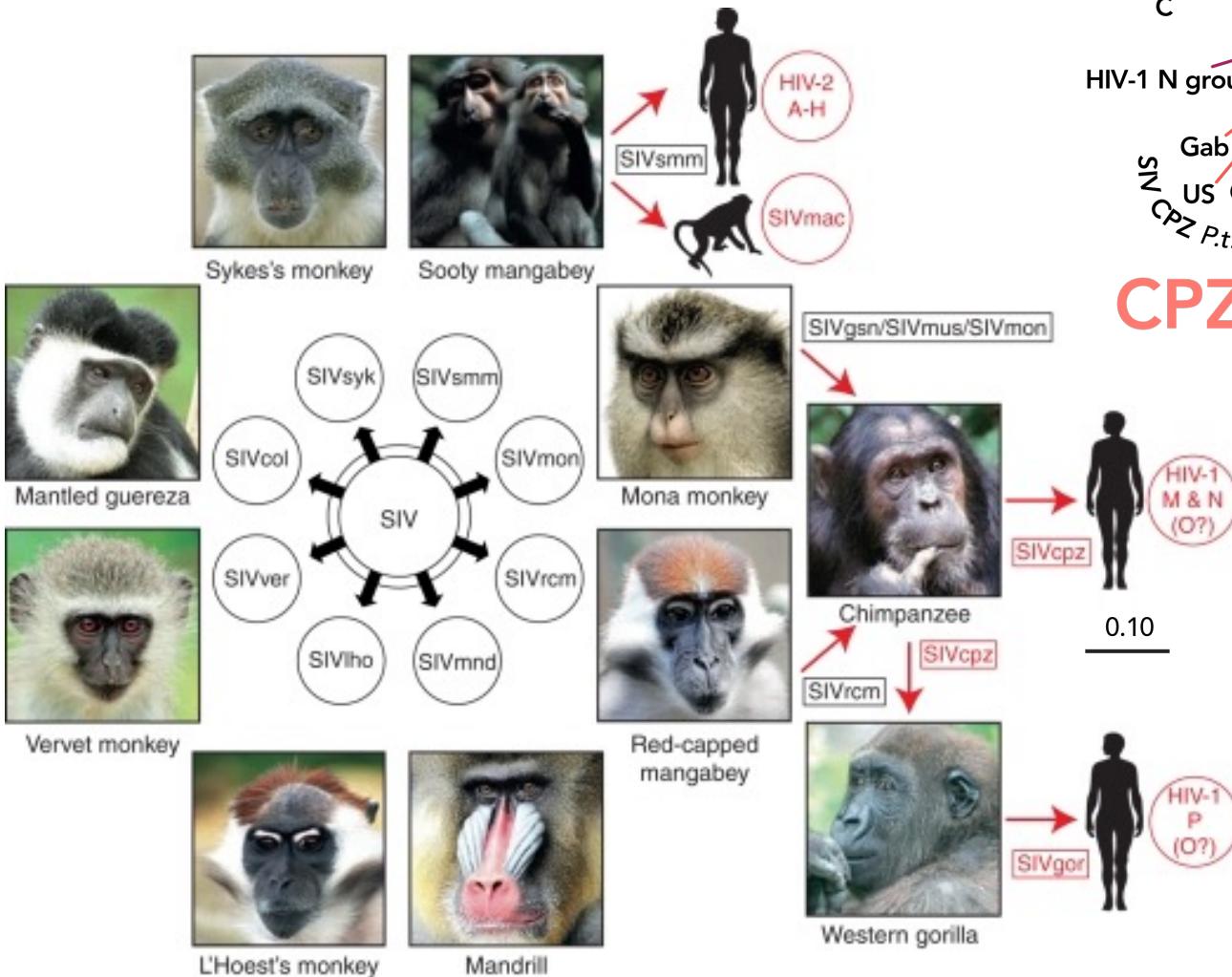
$$R_0 < 1$$

$$R_0 \approx 1$$

$$R_0 > 1$$

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

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



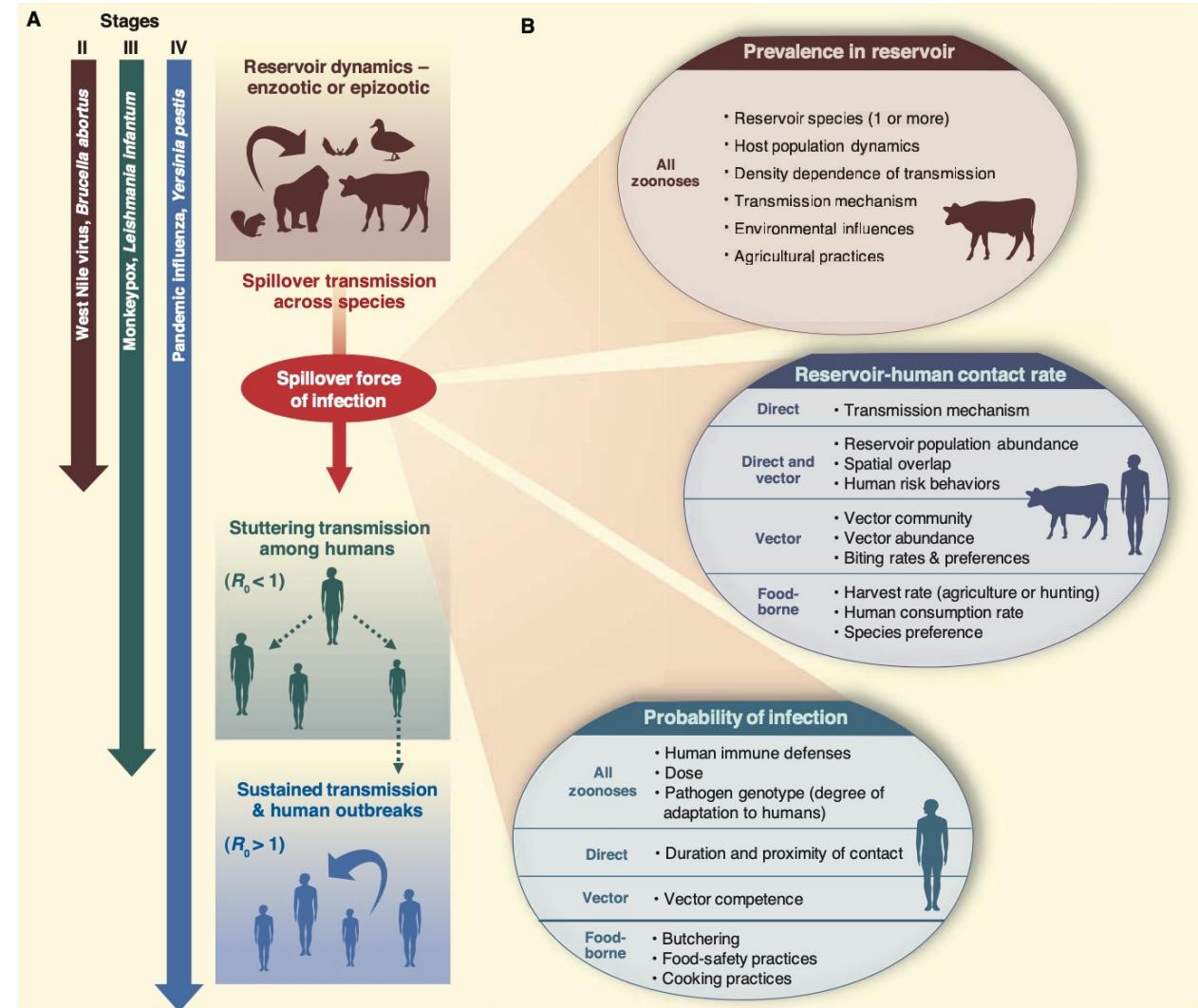
Stage V
Transmits exclusively in humans



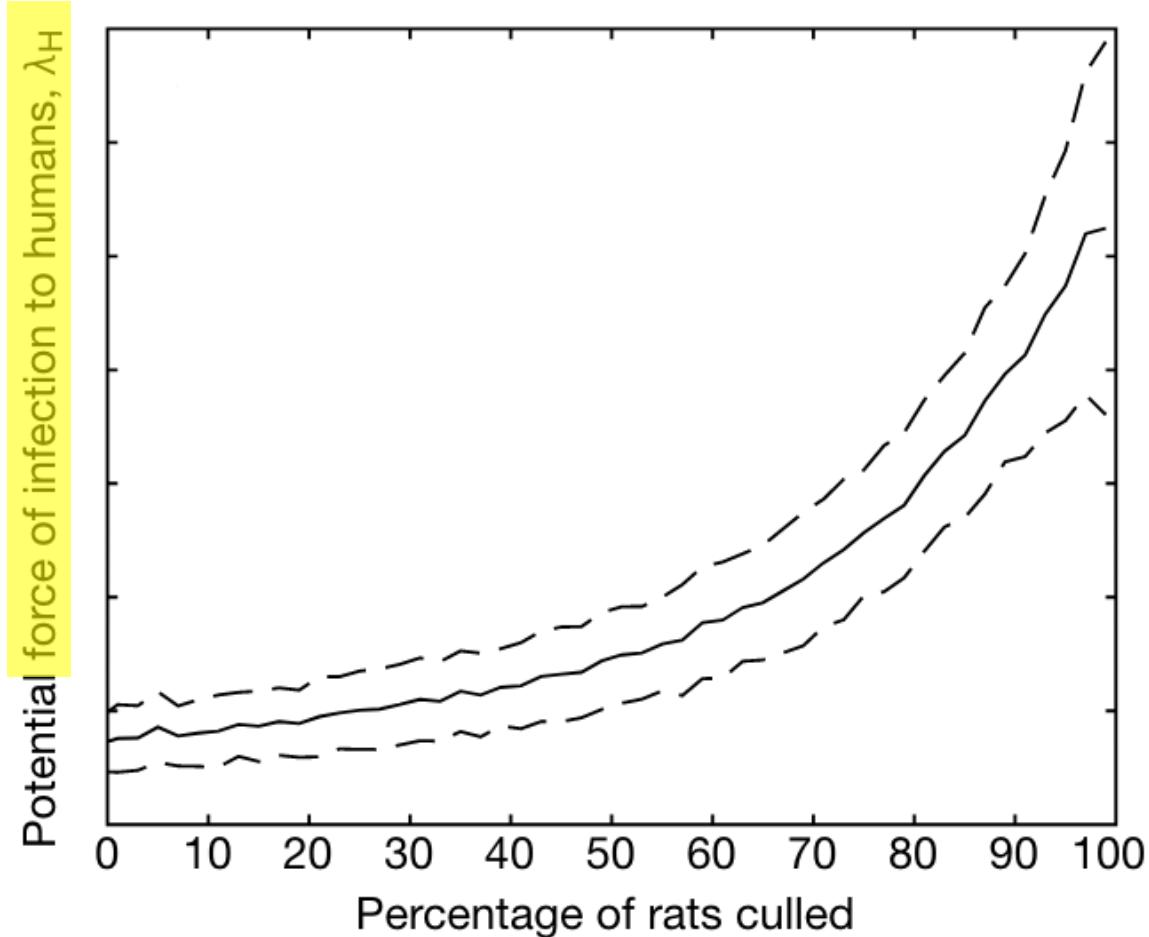
The force of infection (FOI), λ , is the rate at which susceptibles become infected

- For a single host pathogen: $\lambda = R_0 \frac{I}{N}$
- (**FOI = $R_0 * \text{proportion infected}$**)
- \approx mirror image of $R_E = R_0 \frac{S}{N}$
- Also a finger on the epidemic pulse
- For multi-host pathogens, we can define the **spillover force of infection**
 - Comprised of:
 - Infectiousness** of the reservoir
 - Reservoir-human **contact rates**
 - Susceptibility** in the human host

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



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)

Plague is BOTH vector-borne and zoonotic!

Zoonosis is a series of improbable events.

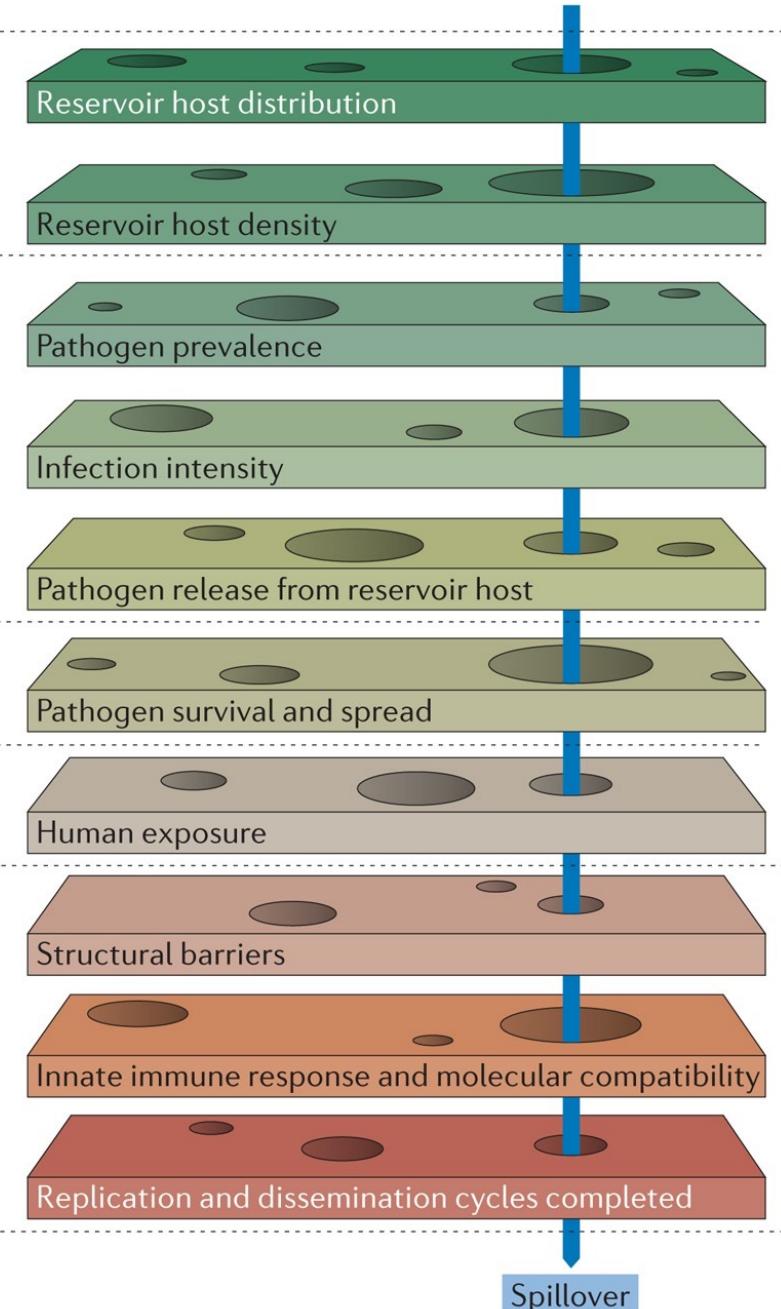
Animal ecology, population biology, biogeography, behavioural ecology, landscape ecology, agricultural sciences

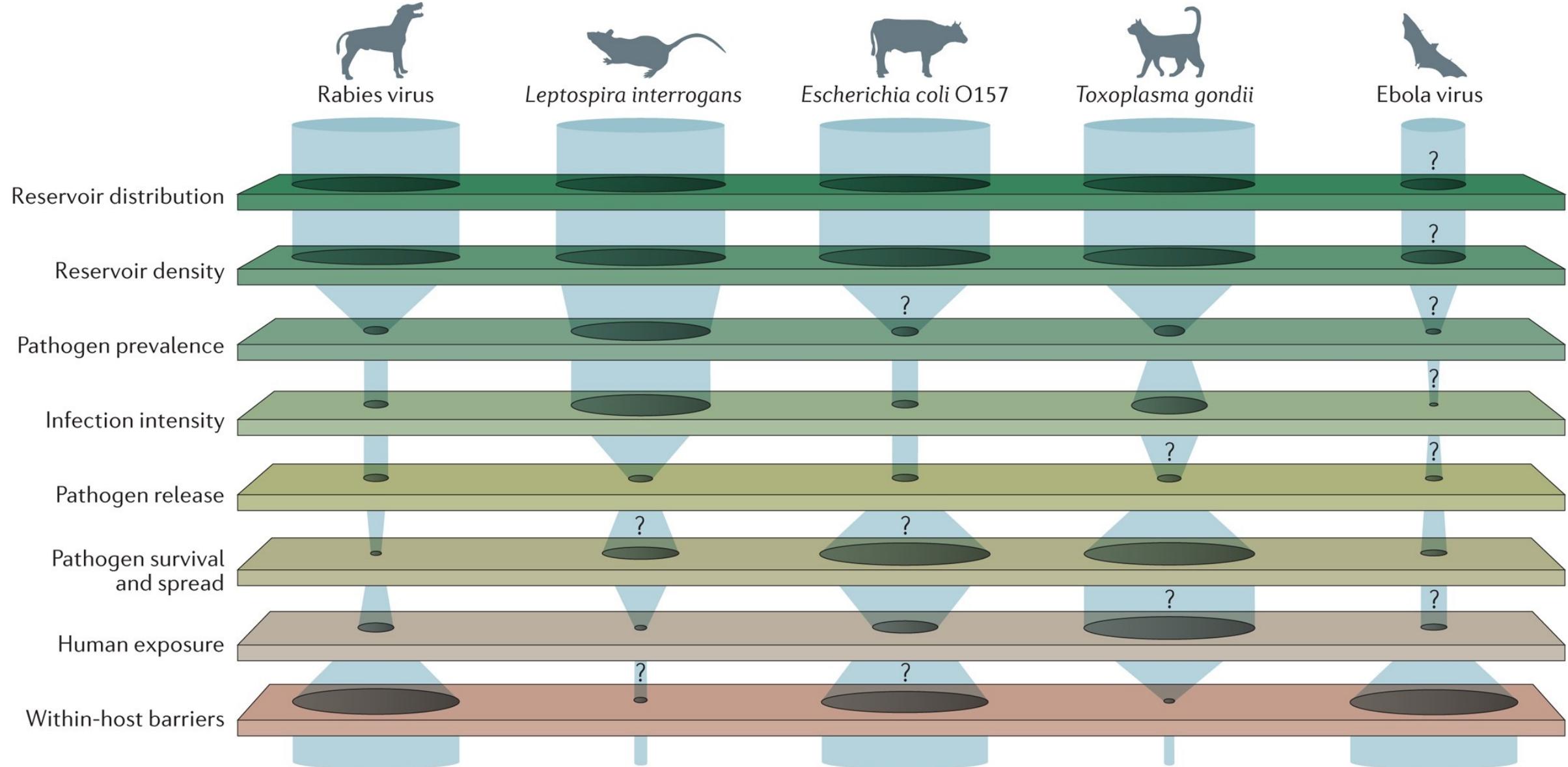
Disease ecology, animal epidemiology, infectious disease dynamics, immunology, microbiology, veterinary medicine

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

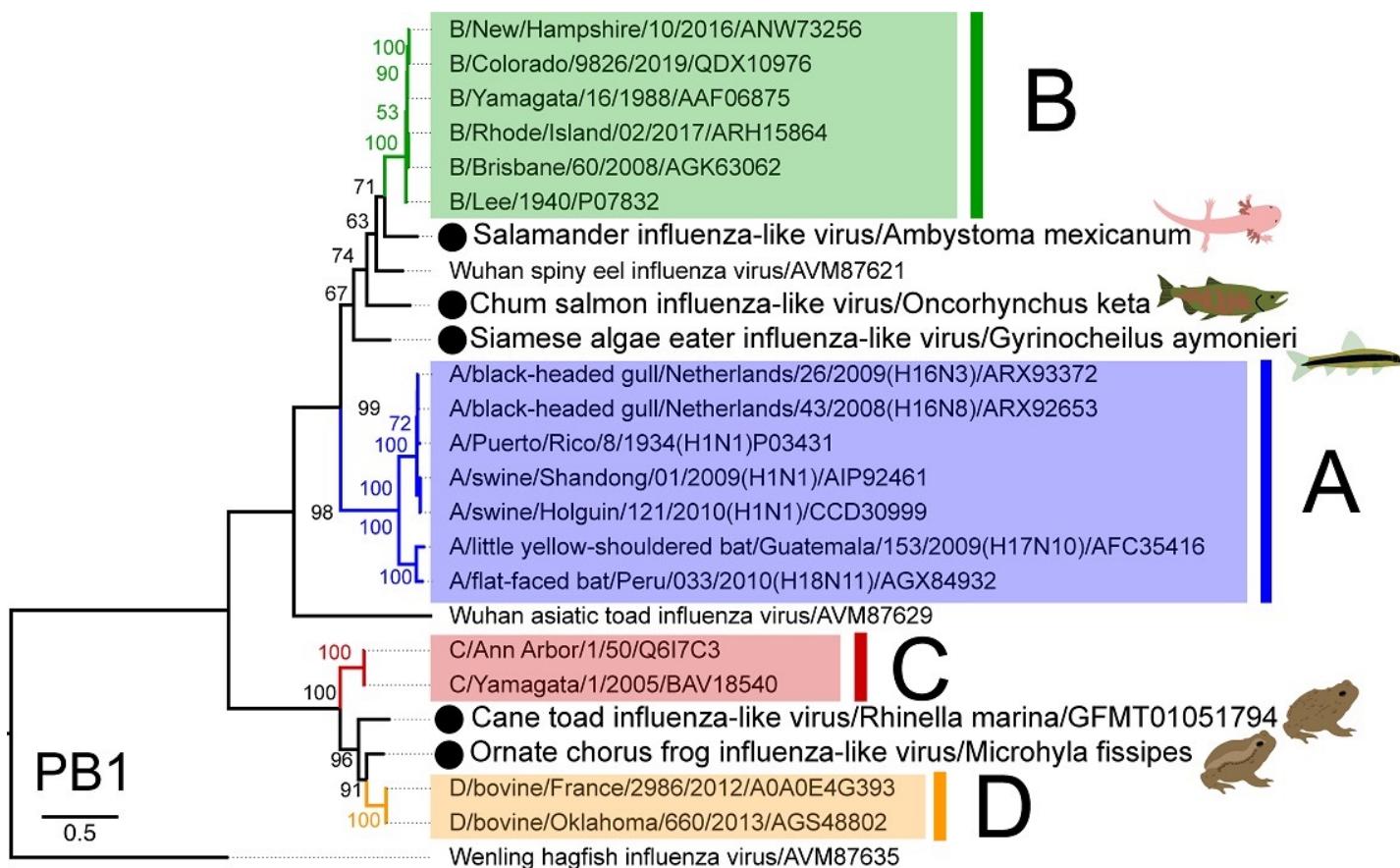




Bottlenecks to spillover.

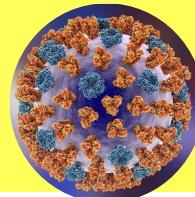
Plowright et al. 2017. *Nature Reviews Microbiology*.

When is influenza zoonotic?

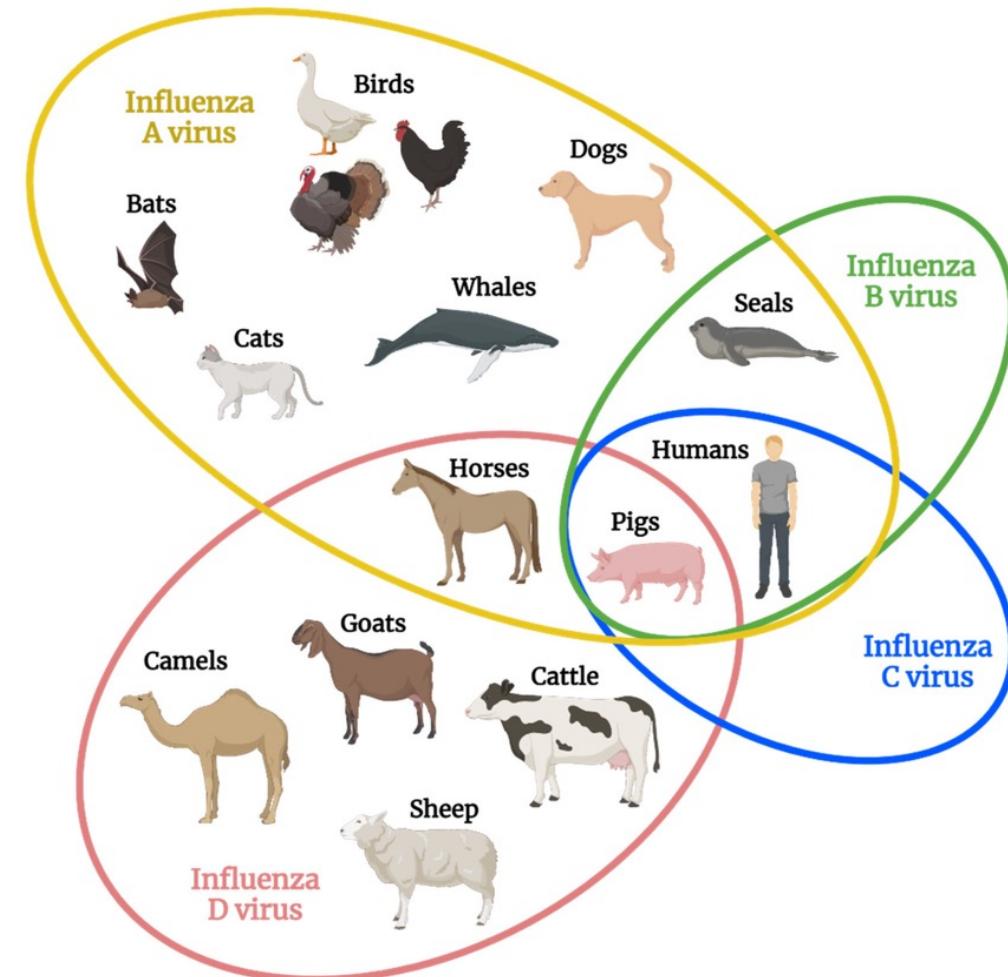


Parry et al. 2020. *Viruses*.

Stage V Transmits exclusively in humans



Influenza

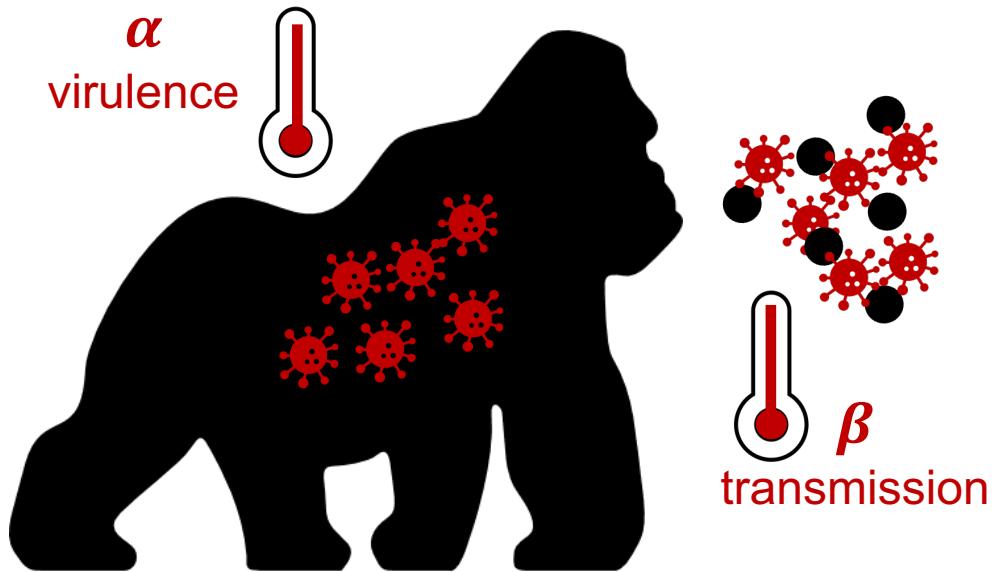


Skelton & Huber. 2022. *Viruses*.

Why do pathogens make us sick?

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

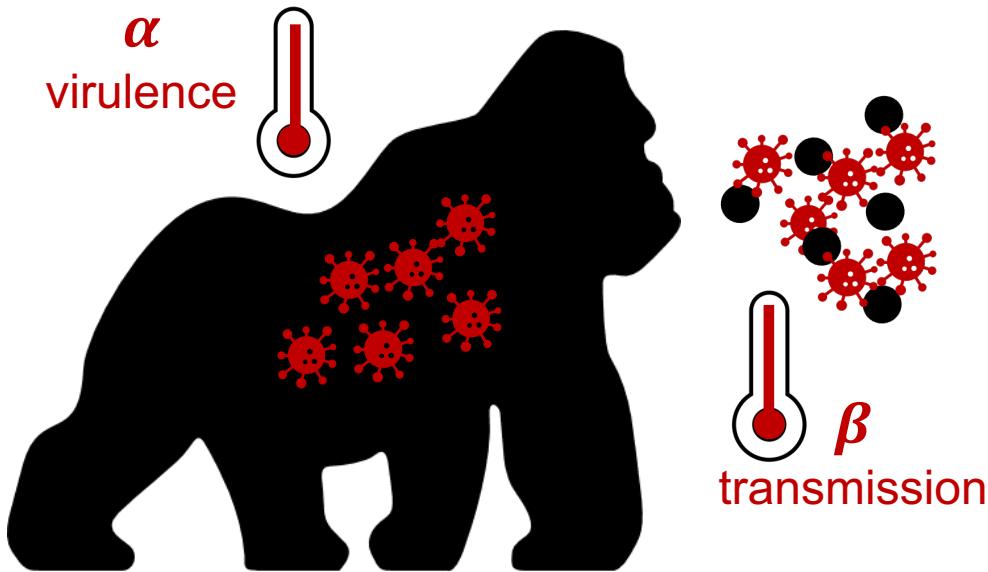
*Why do pathogens
make us sick?*



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

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

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

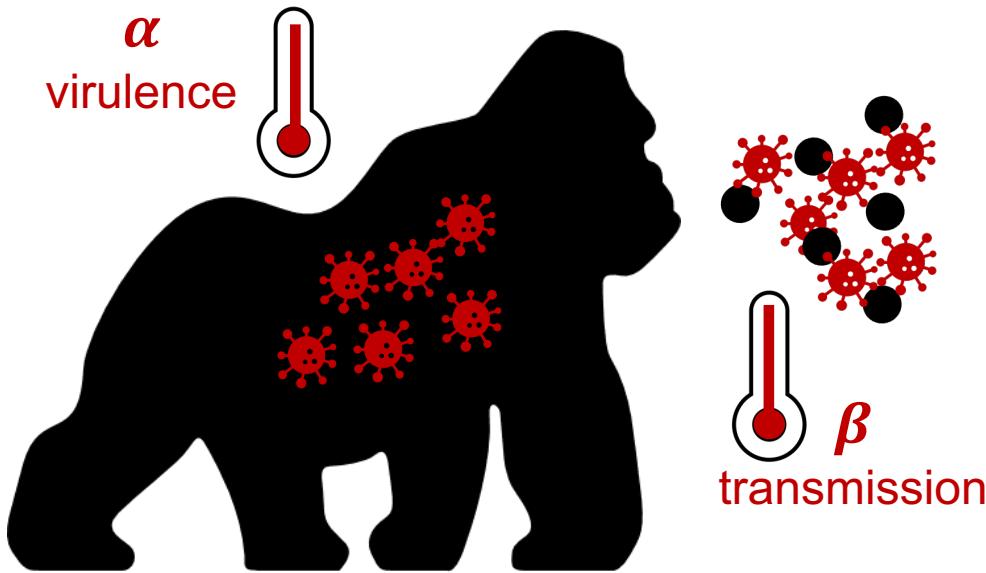


Mechanisms that promote **transmission** may also enhance **virulence** to the host.

Why do pathogens make us sick?

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

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?

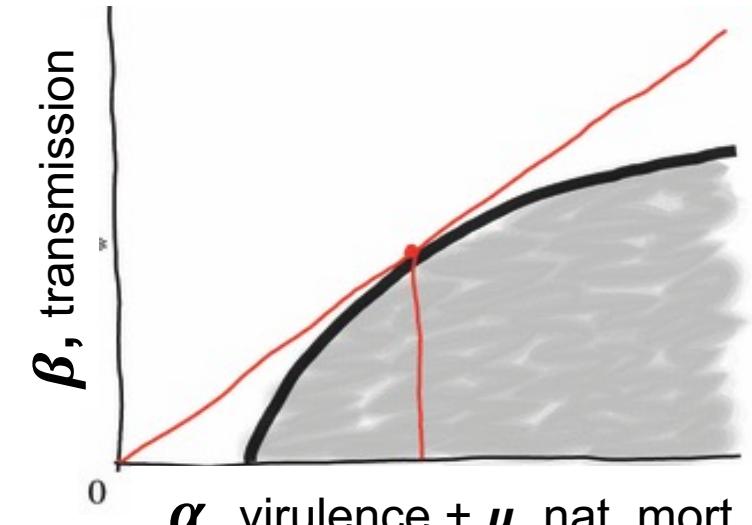
As a result, we predict the evolution of "**optimal virulence**."

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!

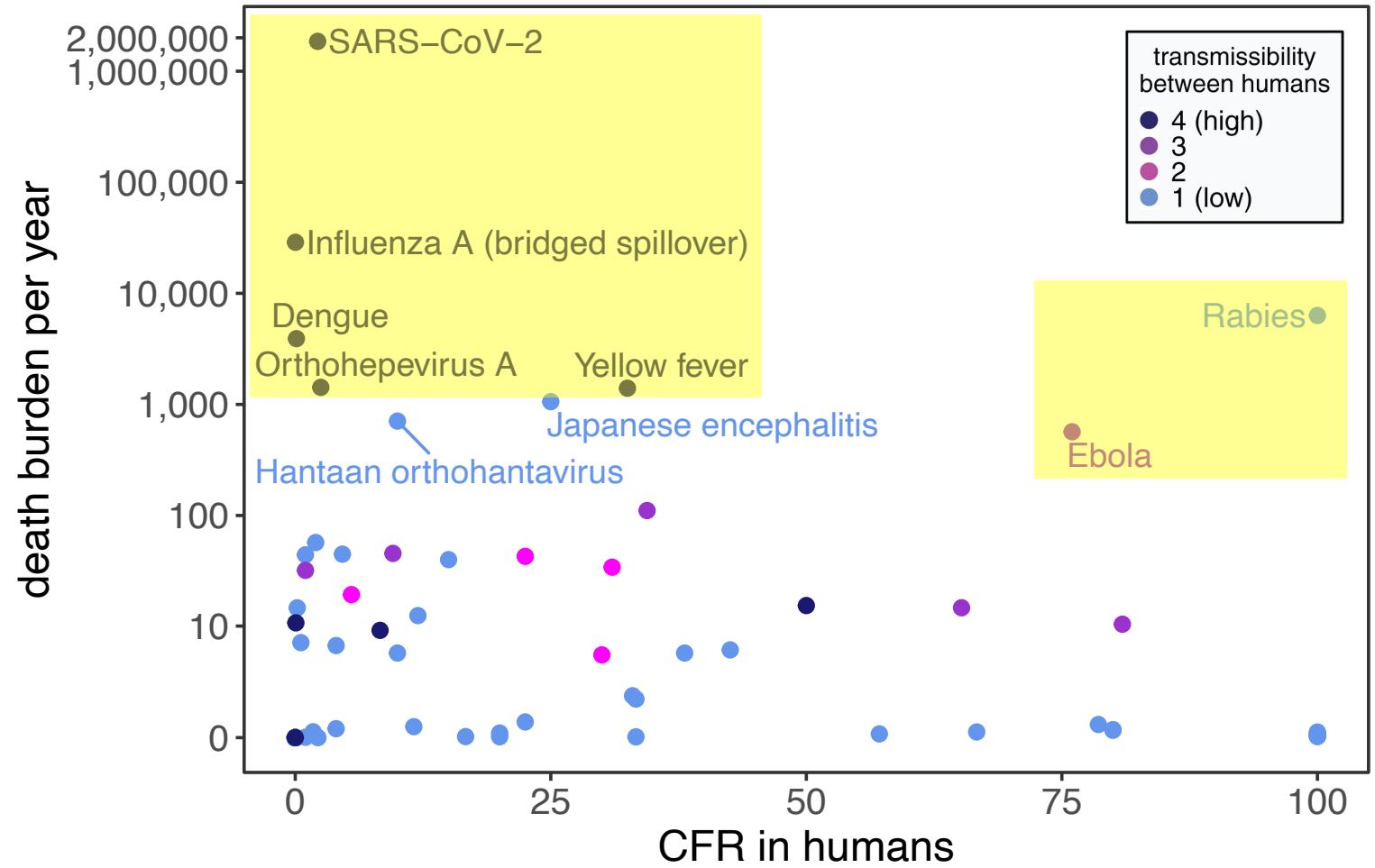
$$R_0 = \frac{\beta(\text{virus density})}{\gamma + \mu + \alpha(\text{virus density})}$$

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).

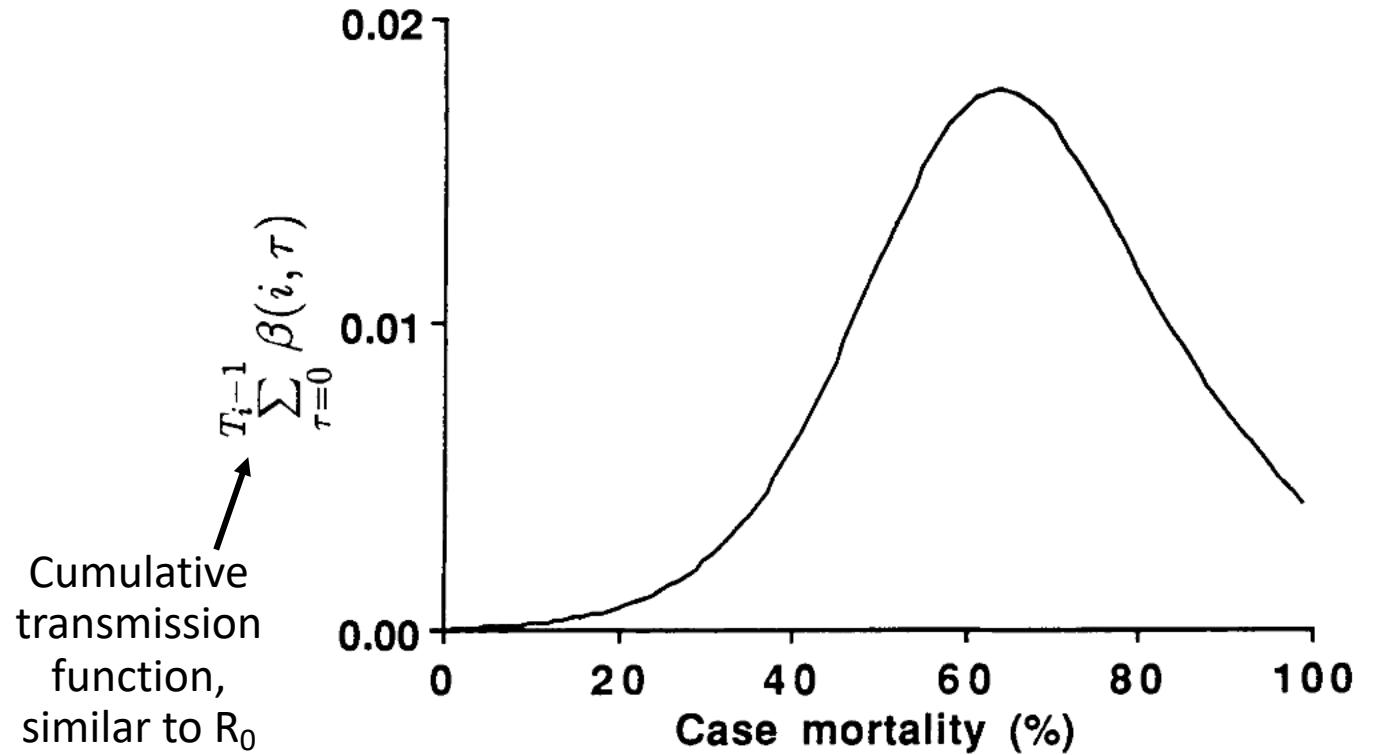
§ Data from J. W. Edmonds and R. C. H. Shepherd (personal communication, 1982).

|| 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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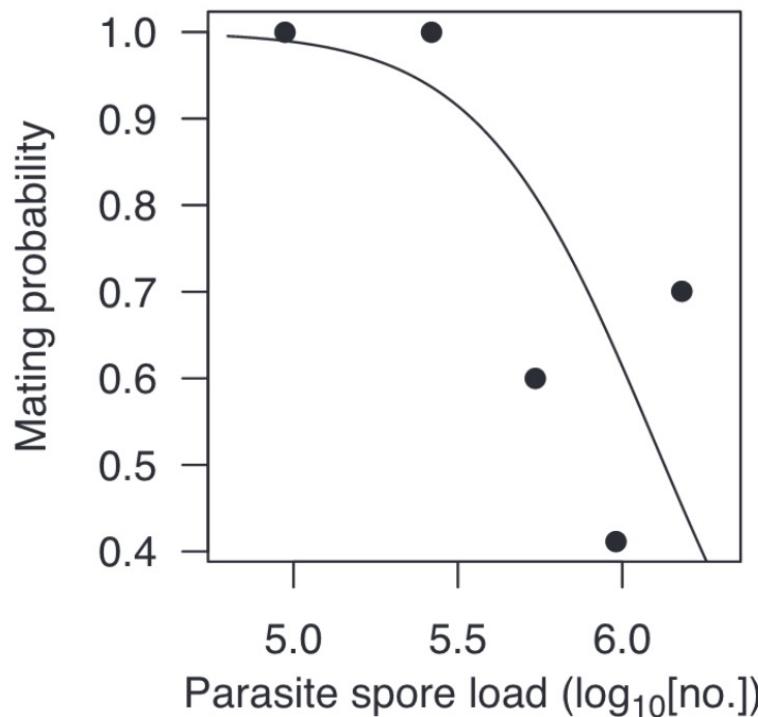
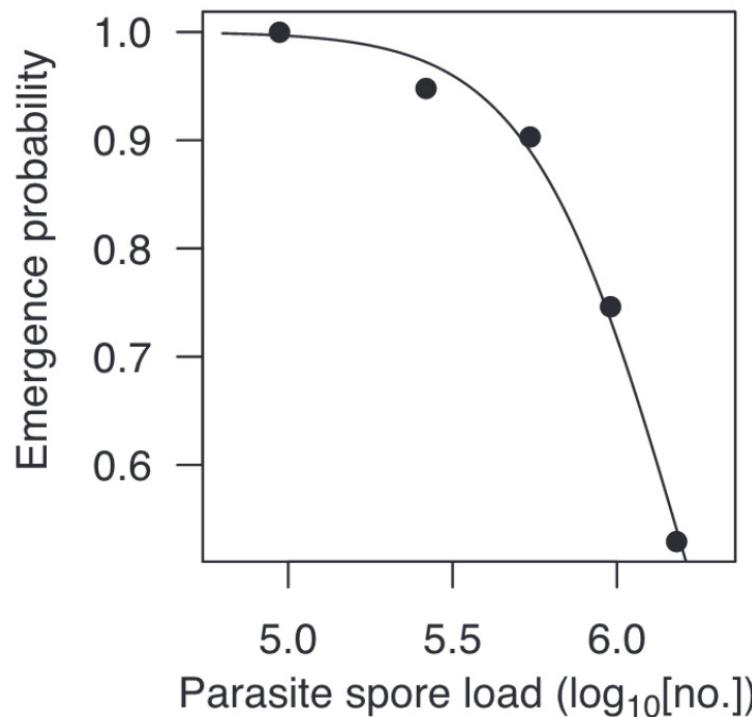
LINDA BUTTEL

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

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

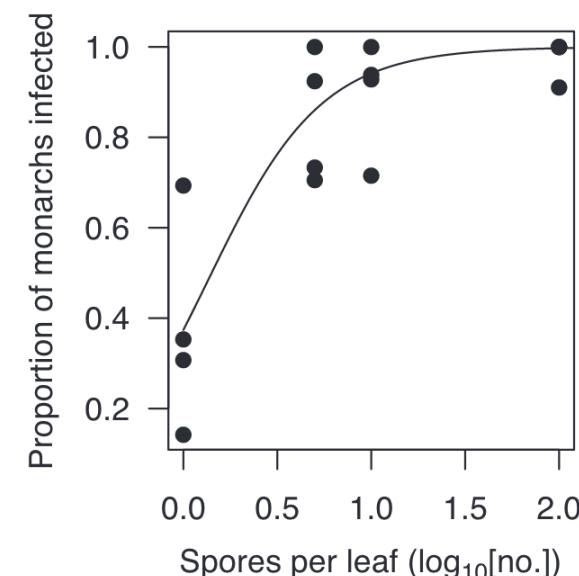
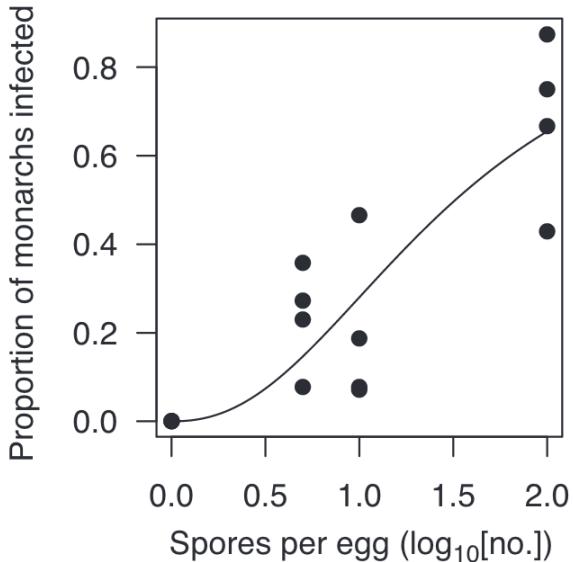
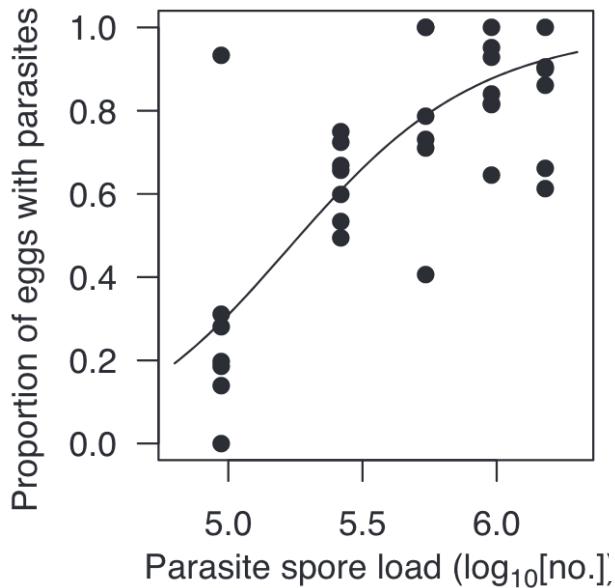
Another classic **transmission-virulence tradeoff**: parasites of monarch butterflies

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



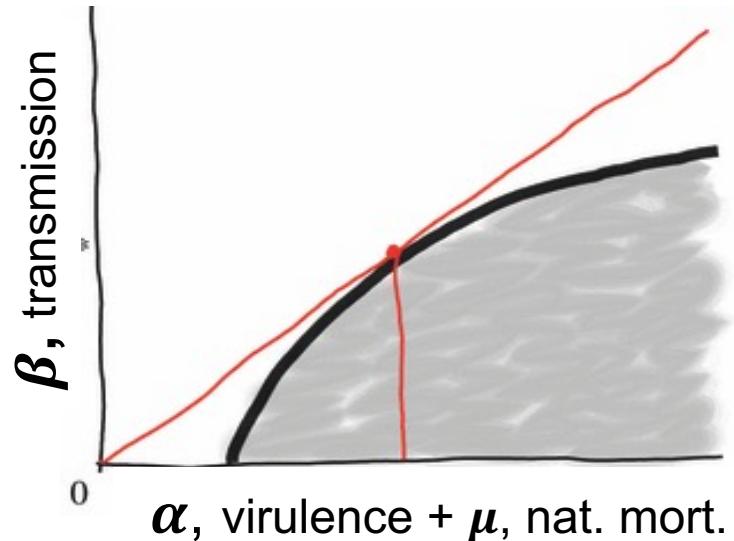
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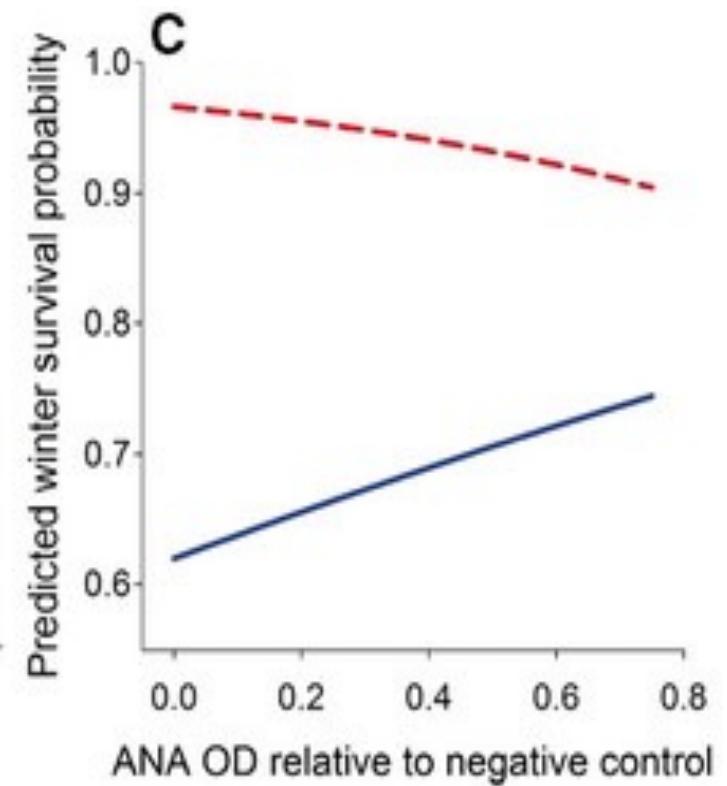
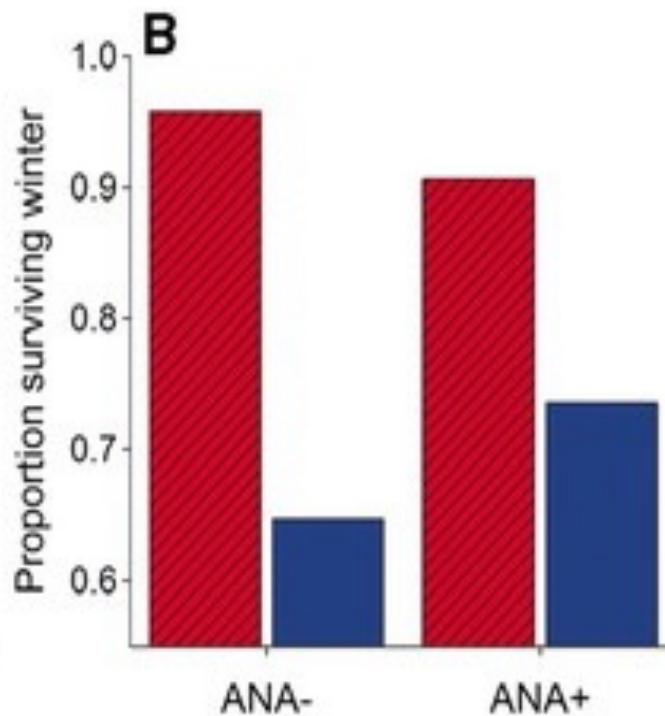
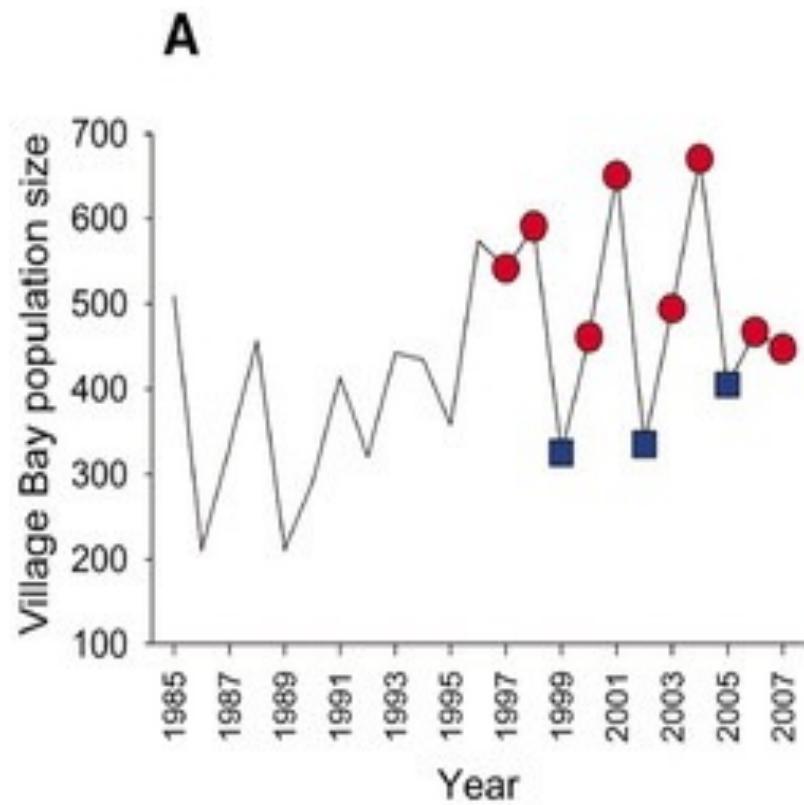
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- On the flip side, higher parasite loads also led to increased **transmission** through higher proportions of monarch eggs that acquired spores and higher numbers of parasites per egg and milkweed leaf, which increased the probability of infection.
- **Parasite fitness is calculated to be maximized at intermediate spore load.**



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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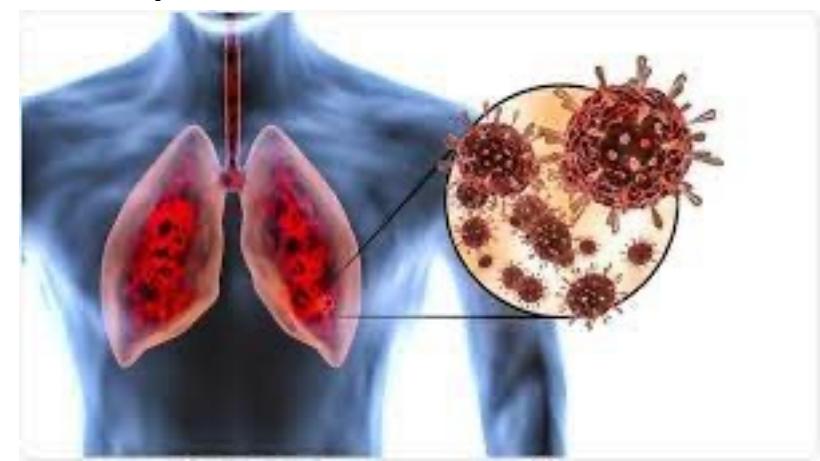


Self-reactive antibodies (ANA) **promote survival by downregulating worms in crash years** but **impede survival via immunopathology in peak years!**



Limitations of the tradeoff model

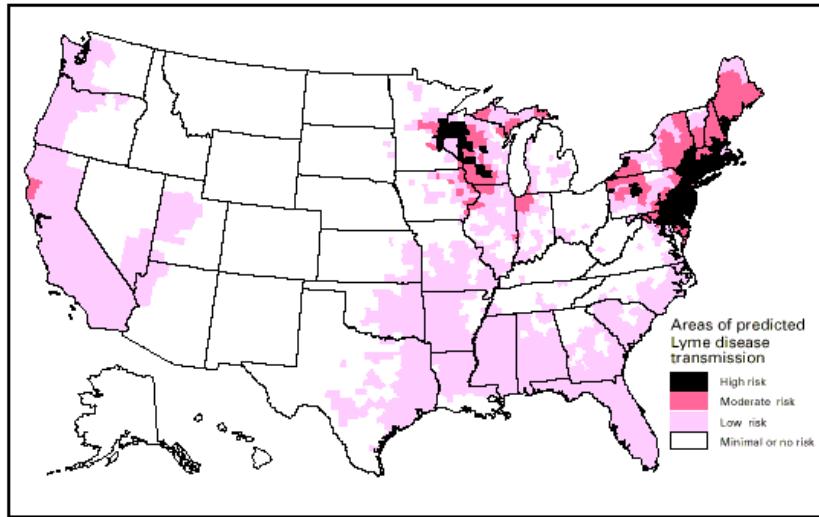
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 - In the case of zoonoses, the bulk of our measurements may be derived from a different host than the one in which the virus evolved
- Many examples of cases in which transmission is decoupled from virulence, due to more complex transmission dynamics.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



Disease dynamics in the **broader community**

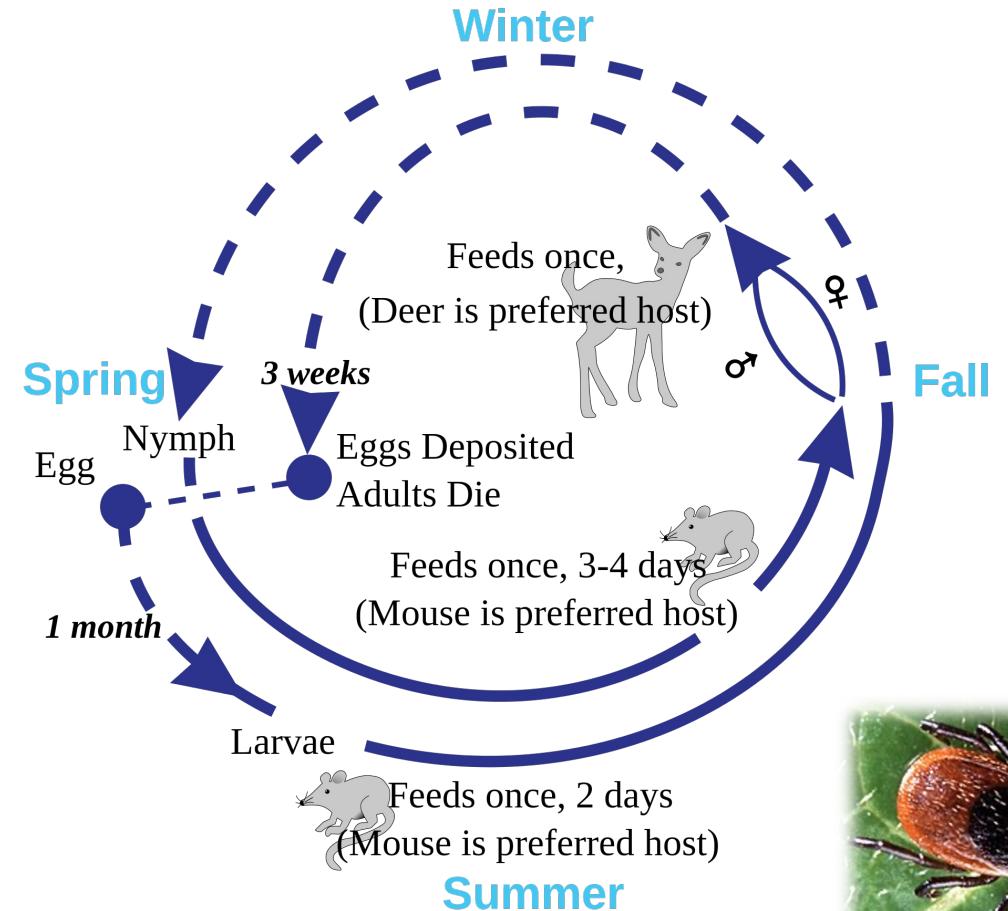
Example: Lyme Disease

National Lyme disease risk map with four categories of risk



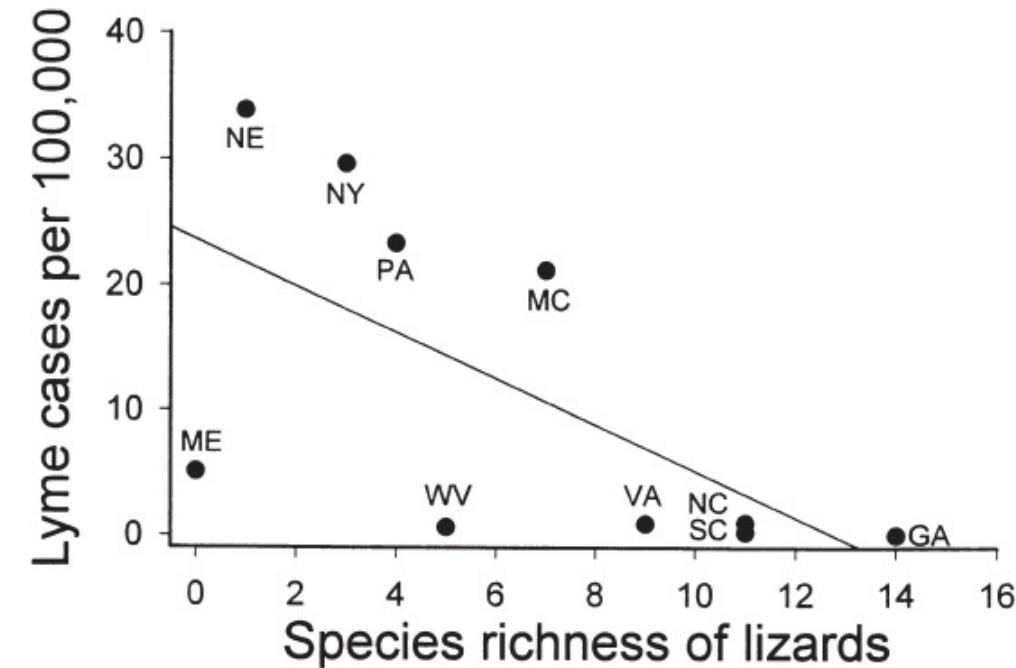
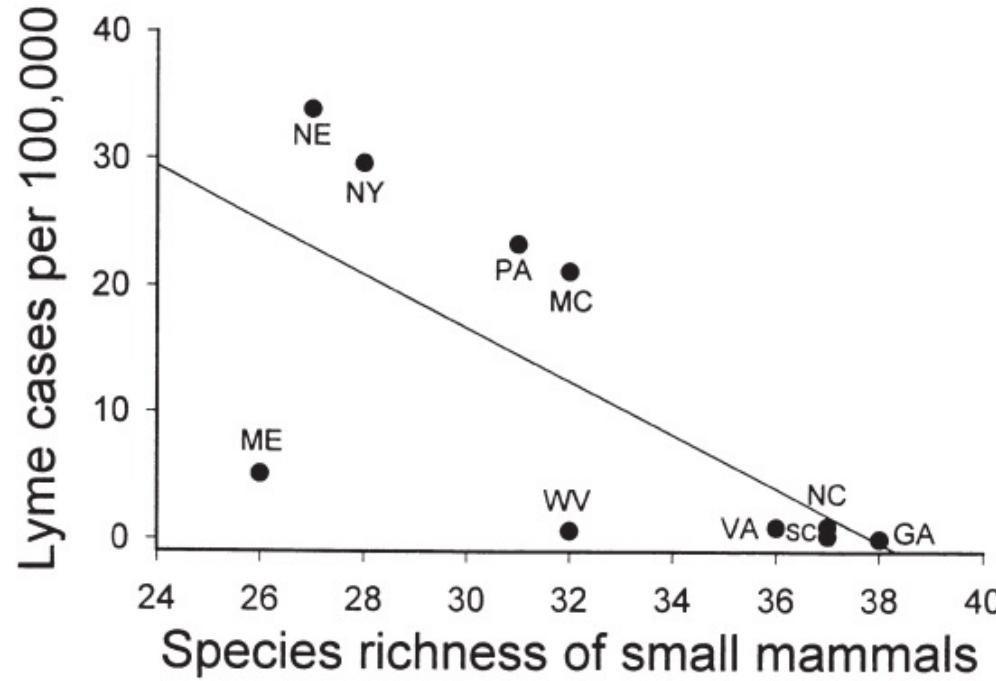
- Lyme disease is a vector-borne disease caused by the bacterium, *Borrelia burgdorferi*, vectored by *Ixodes* especially *Ixodes scapularis* ticks.
- Nymph ticks are borne in the spring, feed on small mammal hosts through the summer, then reproduce (particularly on deer) in the fall before going dormant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

Life Cycle of the *Ixodes scapularis* Tick



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

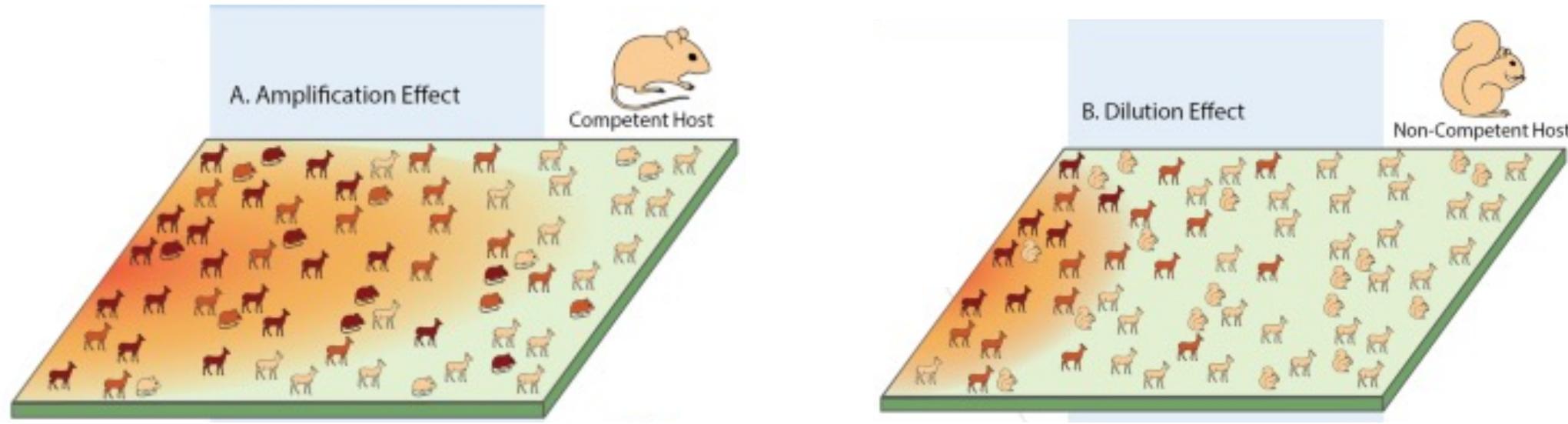
While a popular concept, it only holds in select cases!



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

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

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



In other cases, increasing host diversity will result in **amplification** of pathogen transmission.

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

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

