

Fundamentals of Ecology

Week 8, Ecology Lecture 7

Cara Brook

February 27, 2025

**Office hours: On ZOOM
Friday, Feb 28, 2025
4-5pm
*I will email out a link!***

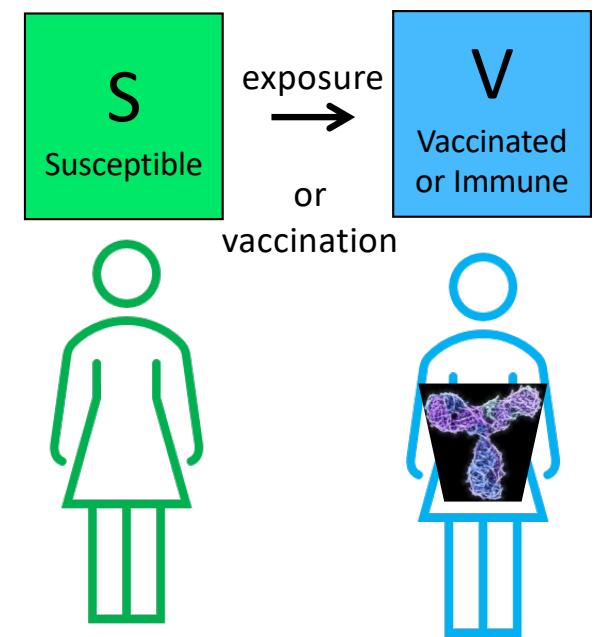
Learning objectives from Lecture 6

You should be able to:

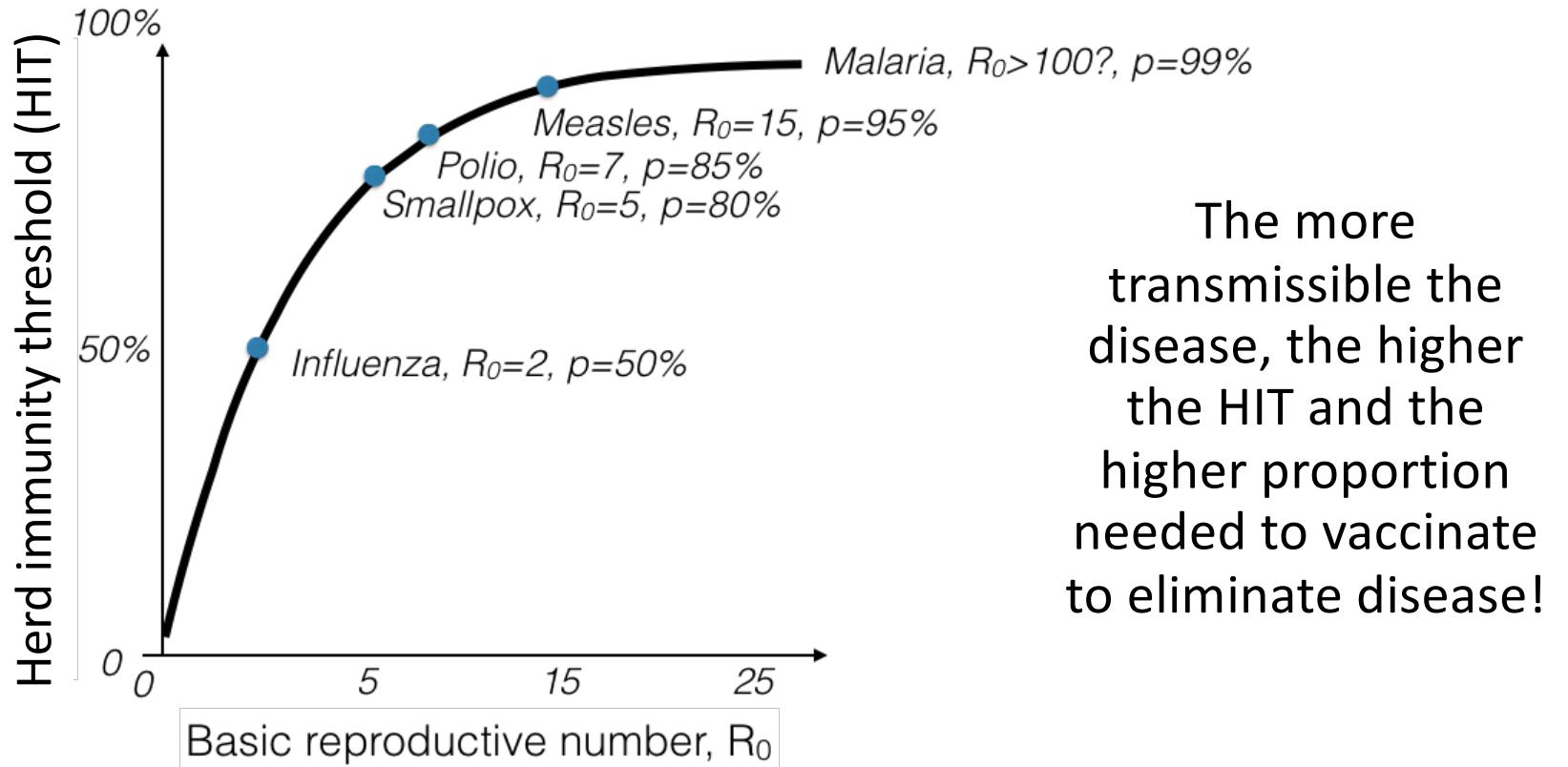
- Recognize that coexistence is rare without spatial structuring or niche partitioning (from competition/coexistence lessons)
- Know the history of disease impacts on human population growth and evolution
- Understand different classes of parasite or pathogen and how they manifest as disease
- Understand the SIR equation and be able to convert between diagrams and equations. Know the processes that connect the different boxes
- Understand the herd immunity concept (also the proportion needed to vaccinate to eradicate disease) and identify on a phase plane graph (from lab)
- Understand the direction of time in a disease epidemic from a phase plane graph (from lab)
- Understand the relationship between R_0 and R_t/RE

Mathematics of Vaccination

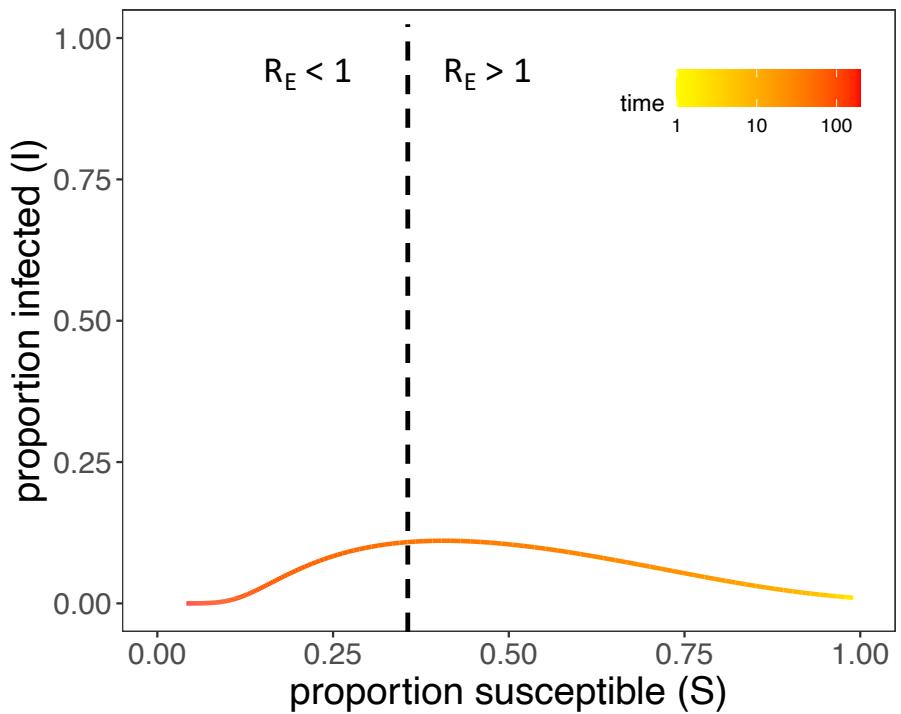
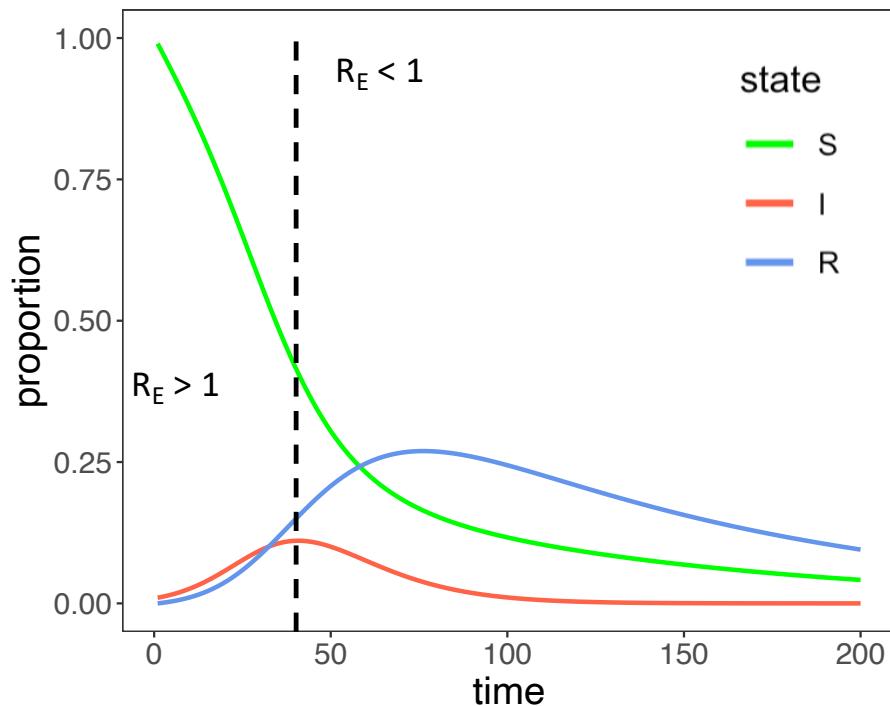
- Goal: **Reduce $R_E < 1$** by removing individuals from the susceptible population.
- Because infectious periods tend to be short-lived (*depending on the pathogen!*), we can theoretically divide the population into two classes: S (susceptible) and V (vaccinated, or immune)
- If $S + V = N$, then
Prop. Susceptible + Prop. Vaccinated = 1.
- Remember, $R_E = R_0 P_S$ or $R_E = R_0(1 - P_V)$
- $R_E < 1 \approx (1 - P_V)R_0 < 1$
- Rearranging, $P_V > 1 - \frac{1}{R_0}$
- **This is the herd immunity threshold.**
- **Even susceptibles will not become infected because the disease will not spread ($R_E < 1$).**



R_0 and the Herd Immunity Threshold



R_0 and the Herd Immunity Threshold

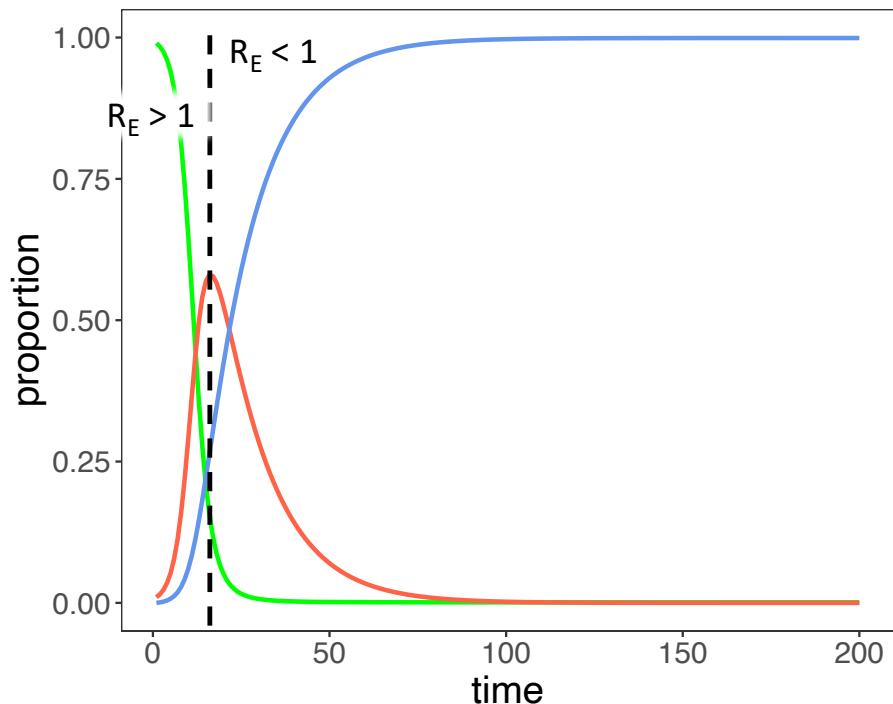


$$R_0 = 2.8$$

$$P_v = 1 - 1/R_0 = 1 - (1/2.8) = 0.64$$

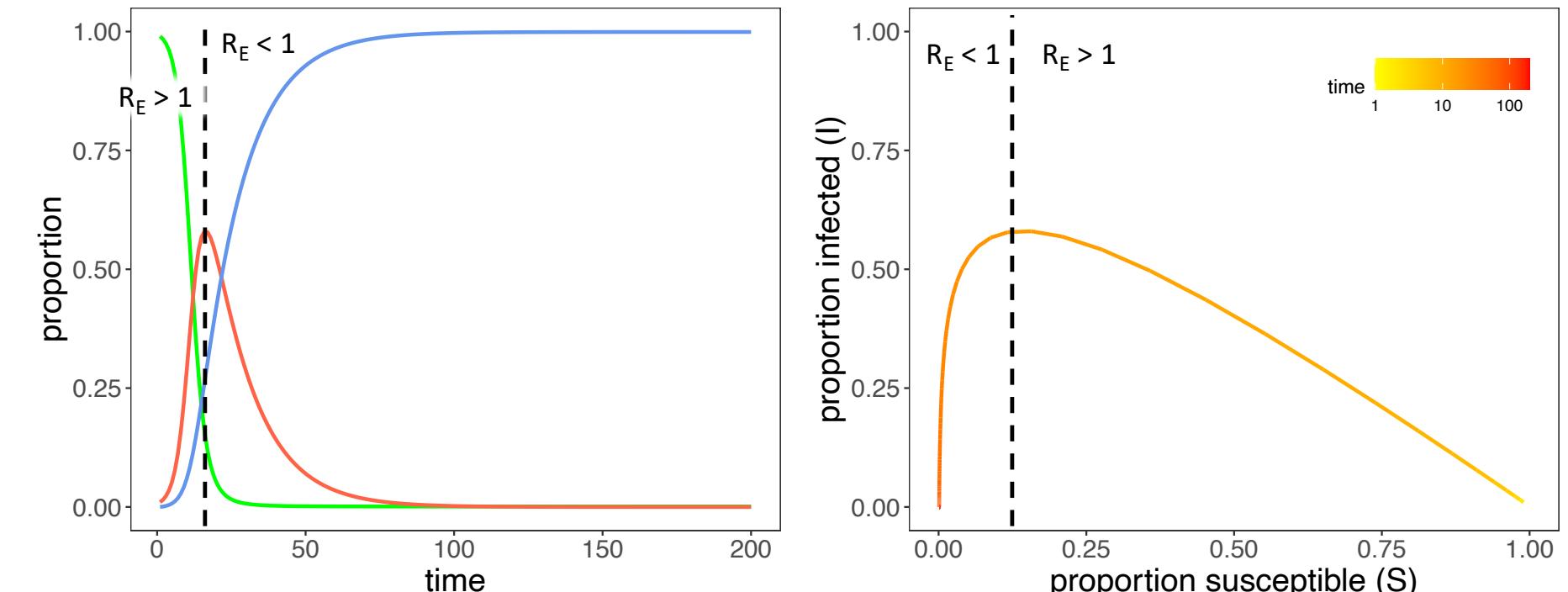
64% need to be immune to stop the disease from spreading.
That means only 36% remaining susceptibles are permitted!

R_0 and the Herd Immunity Threshold



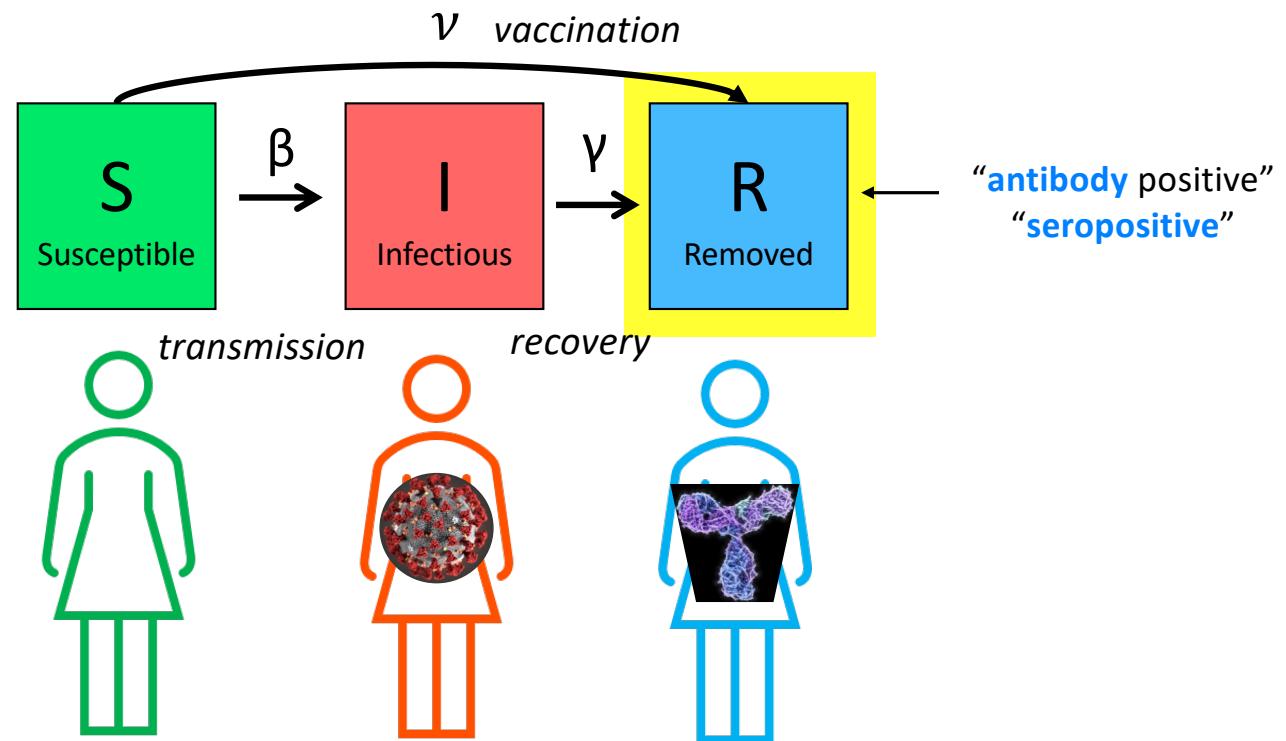
$$R_0 = 7$$

$$P_v = 1 - 1/R_0 = 1 - (1/7) = 0.86$$



86% need to be immune to stop the disease from spreading.
That means only 14% remaining susceptibles are permitted!

Vaccination stems from a long history

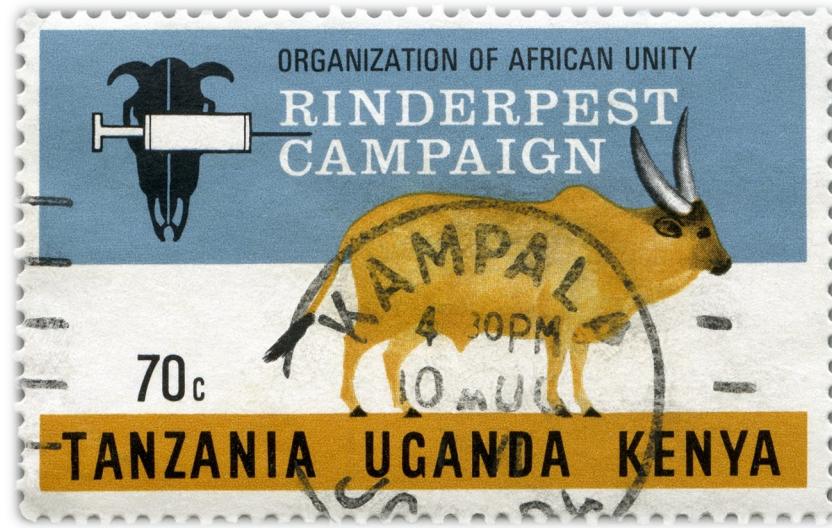


Vaccination stems from a long history

- 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



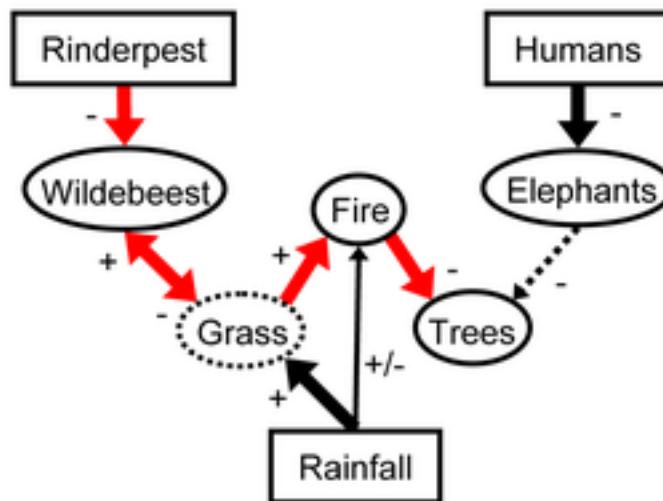
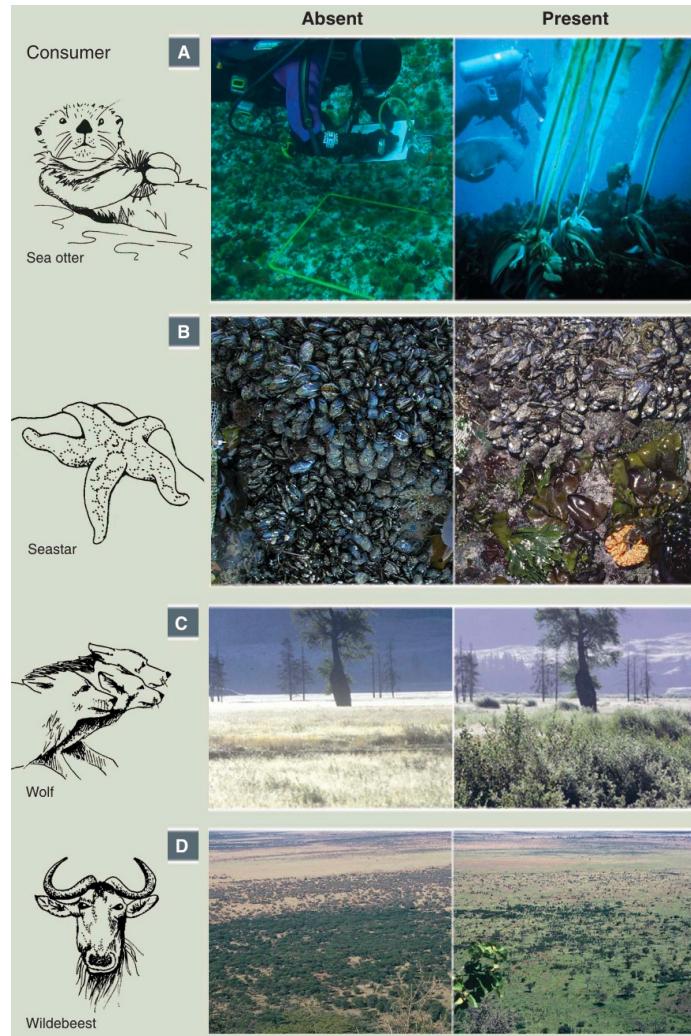
Only two global vaccination success stories



Rinderpest was globally eradicated in 2011, though inoculation efforts date back to the 1700s!

Remember those **trophic cascades**...

Estes et al. 2011. *Science*.



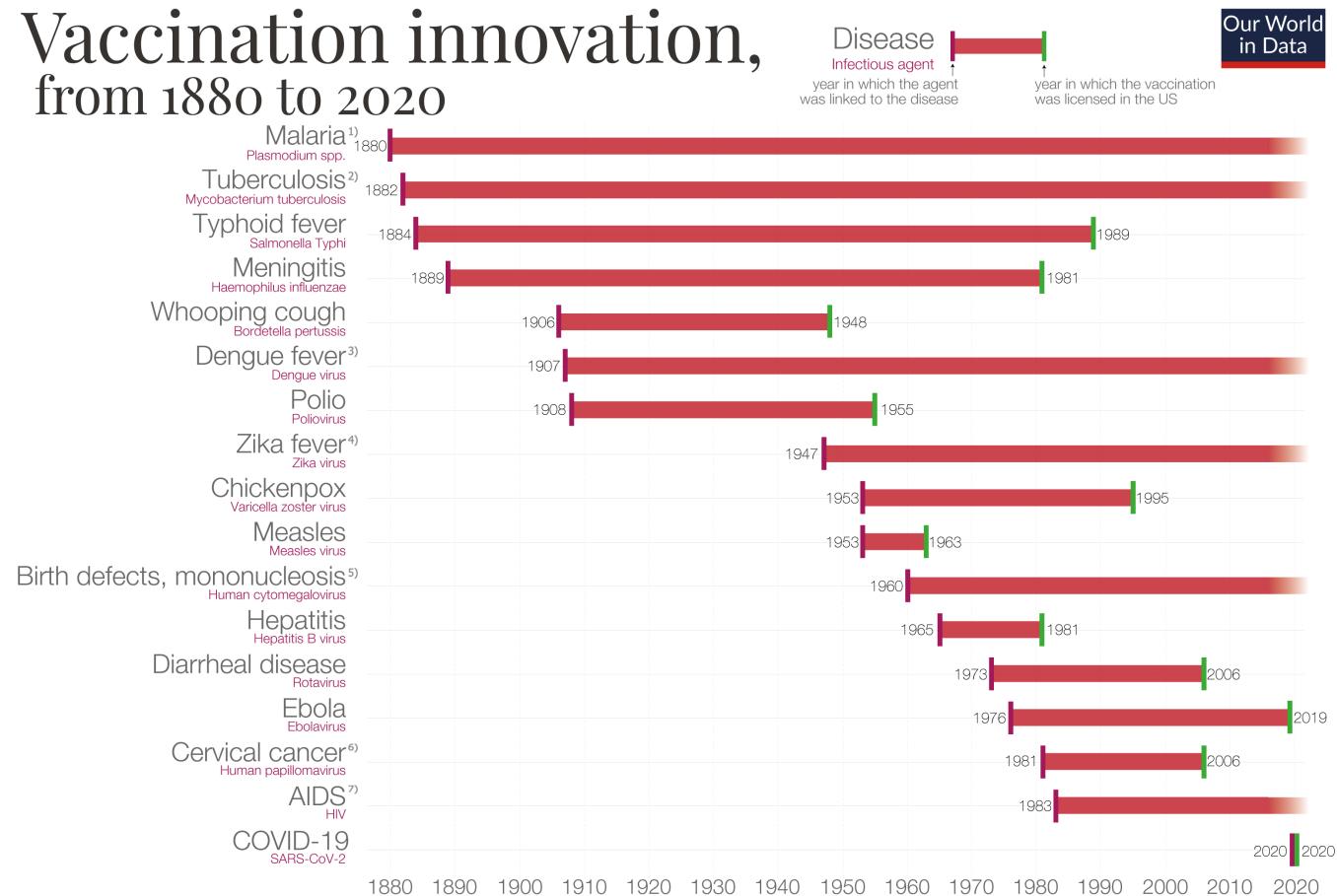
1. ↓ rinderpest
2. ↑ wildebeest
3. ↓ grass
4. ↓ fire
5. ↑ trees

Rinderpest eradication releases wildebeest populations that control savanna, limit fire, and promote tree regrowth

Holdo et al. 2009. *PLoS Biology*

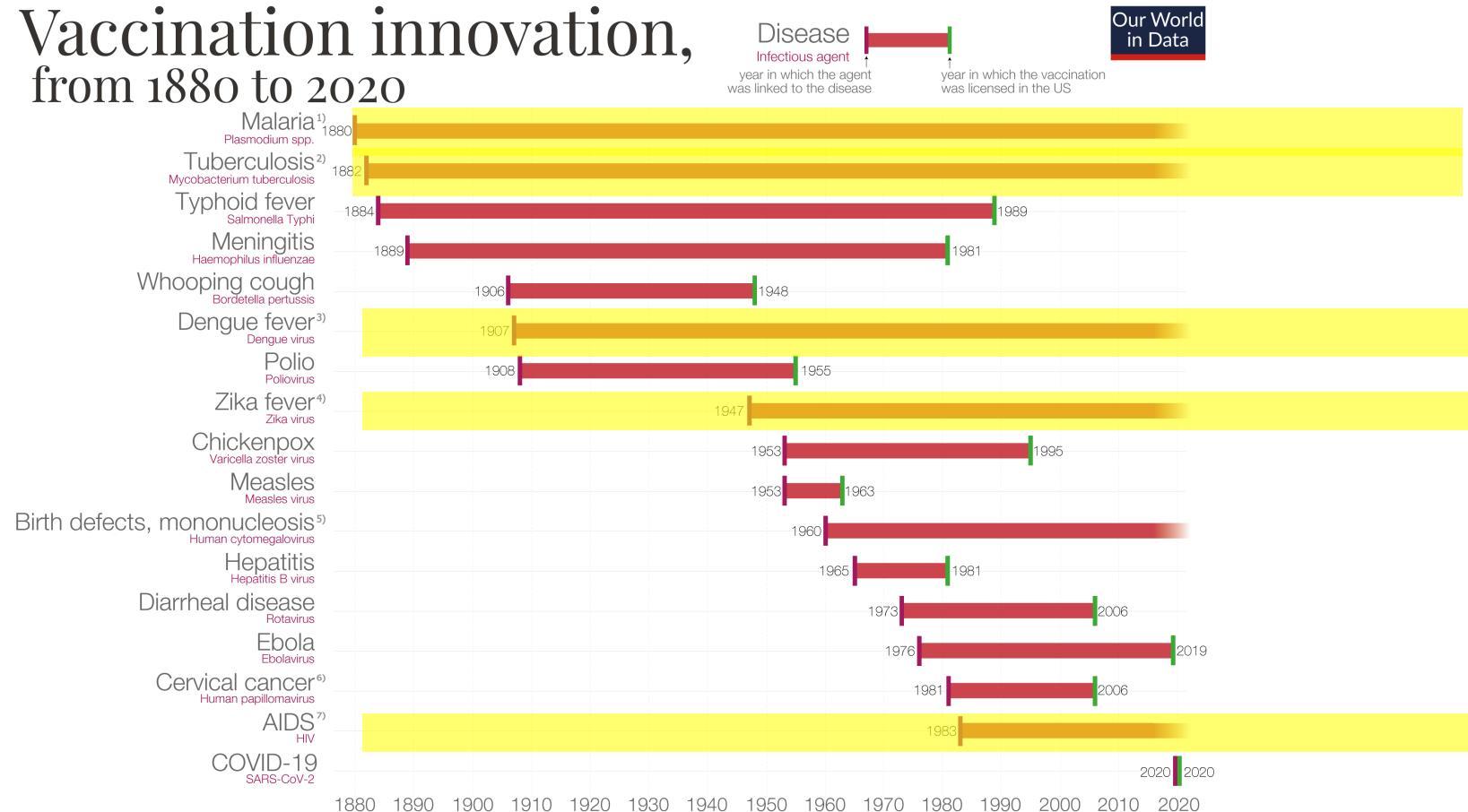
The pace of vaccine development has accelerated drastically

Vaccination innovation, from 1880 to 2020



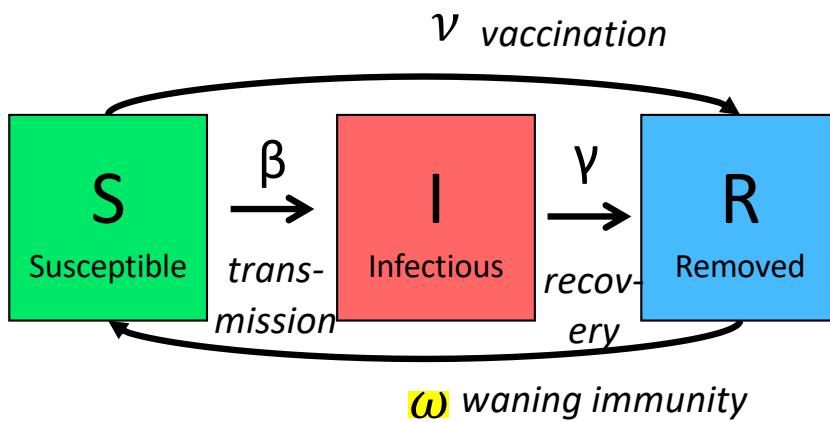
But we still lack vaccines for several important diseases.

Vaccination innovation, from 1880 to 2020



Challenges to Vaccination

- Imperfect immunity, especially with non-viral pathogens

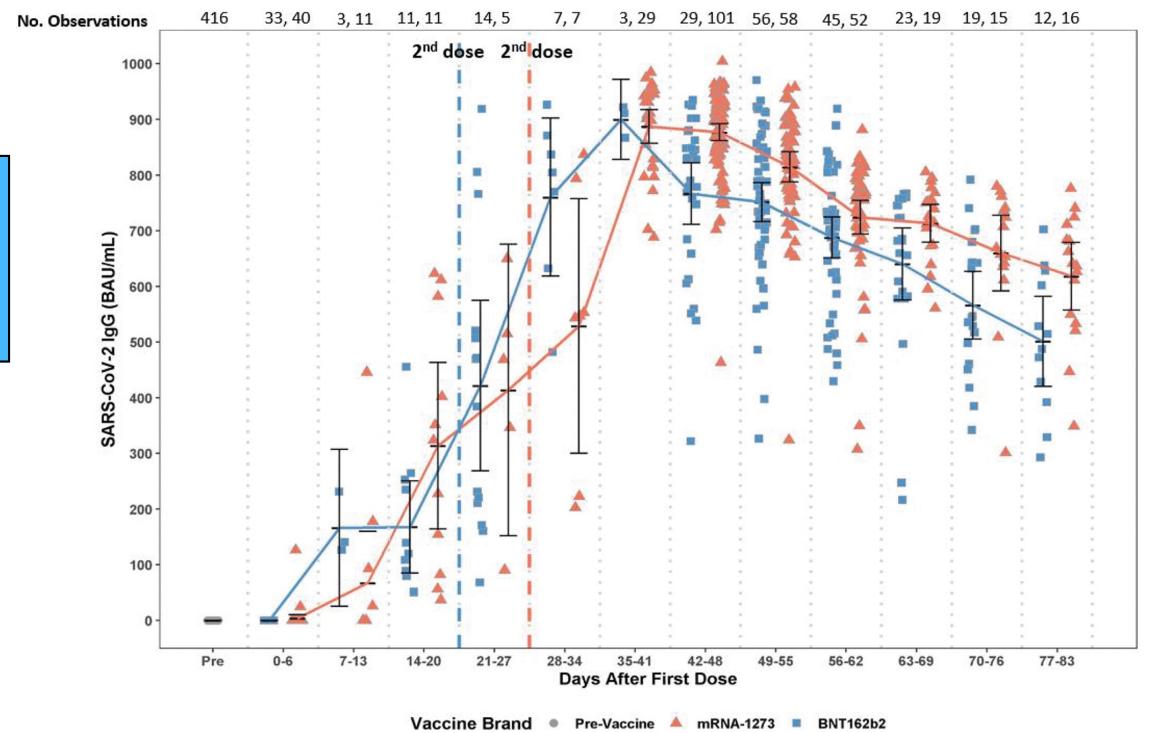


β = transmission rate

γ = recovery rate

ν = vaccination rate

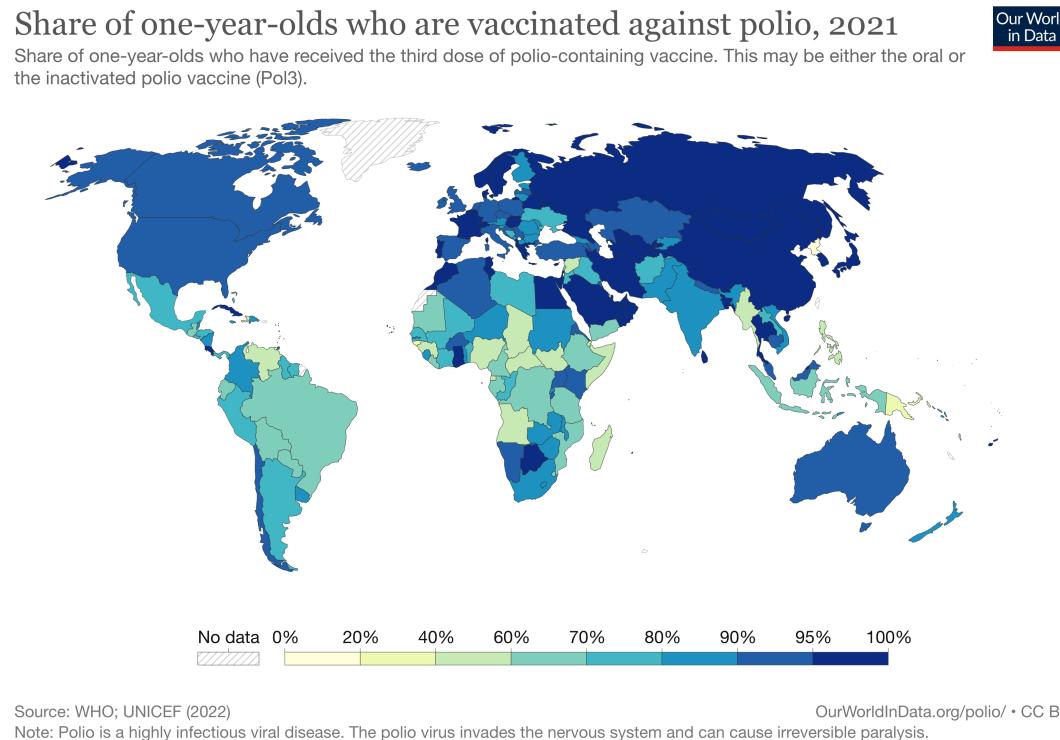
ω = rate waning immunity



Montoya et al. 2021 *Microbiology Spectrum*

Challenges to Vaccination

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



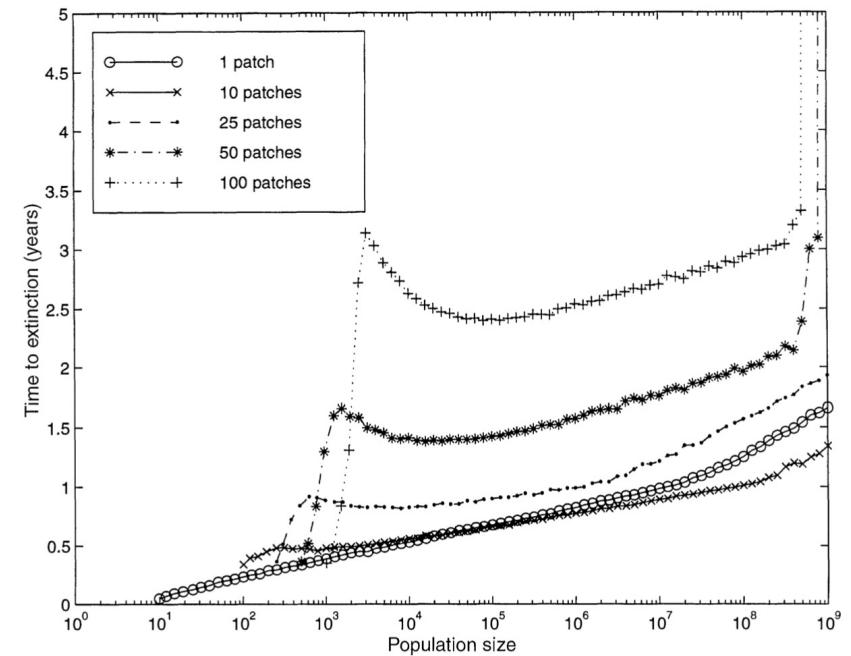
Challenges to Vaccination

- 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)



Swinton et al. 1998
Bartlett 1957

Challenges to Vaccination

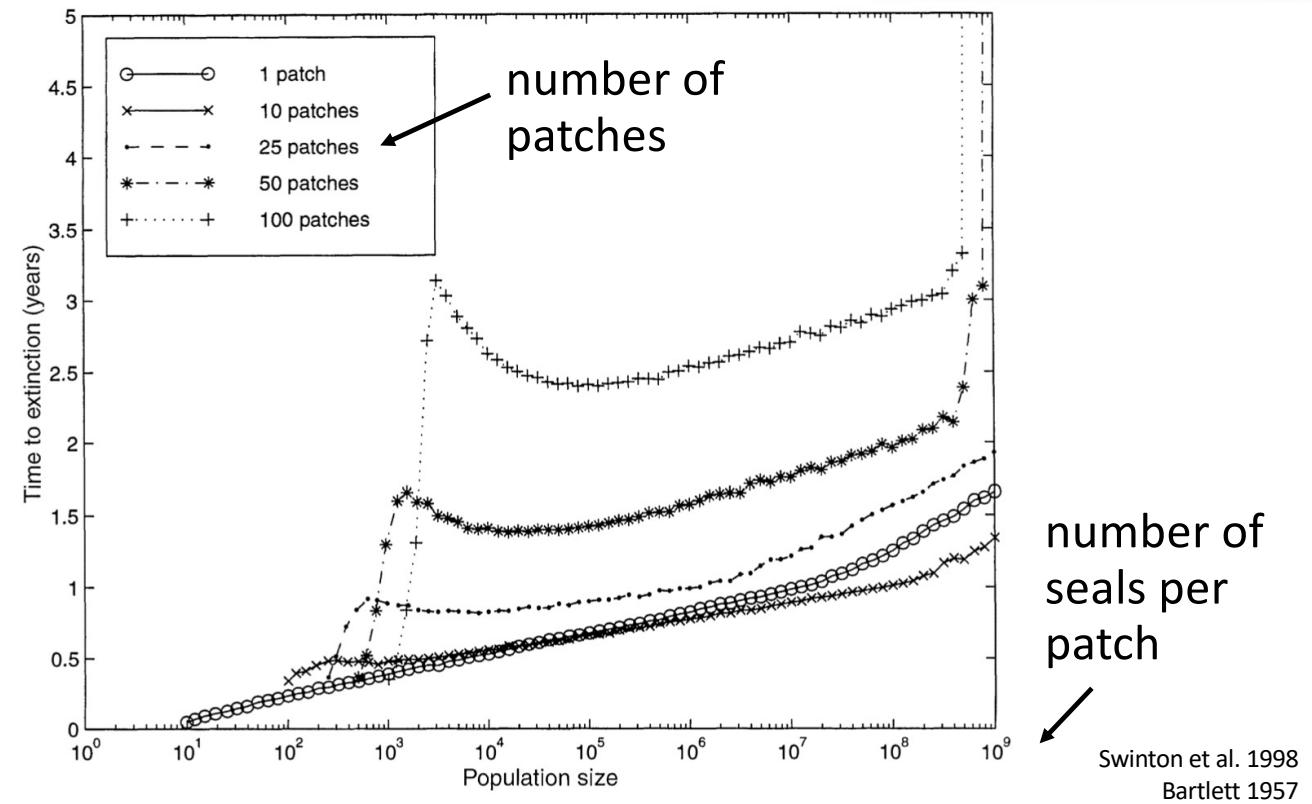
- Spatial structure (**metapopulation rescue**)



time until all patches go extinct (remember metapopulations!)

All else equal, **increasing the number of patches will slow the time to extinction.**

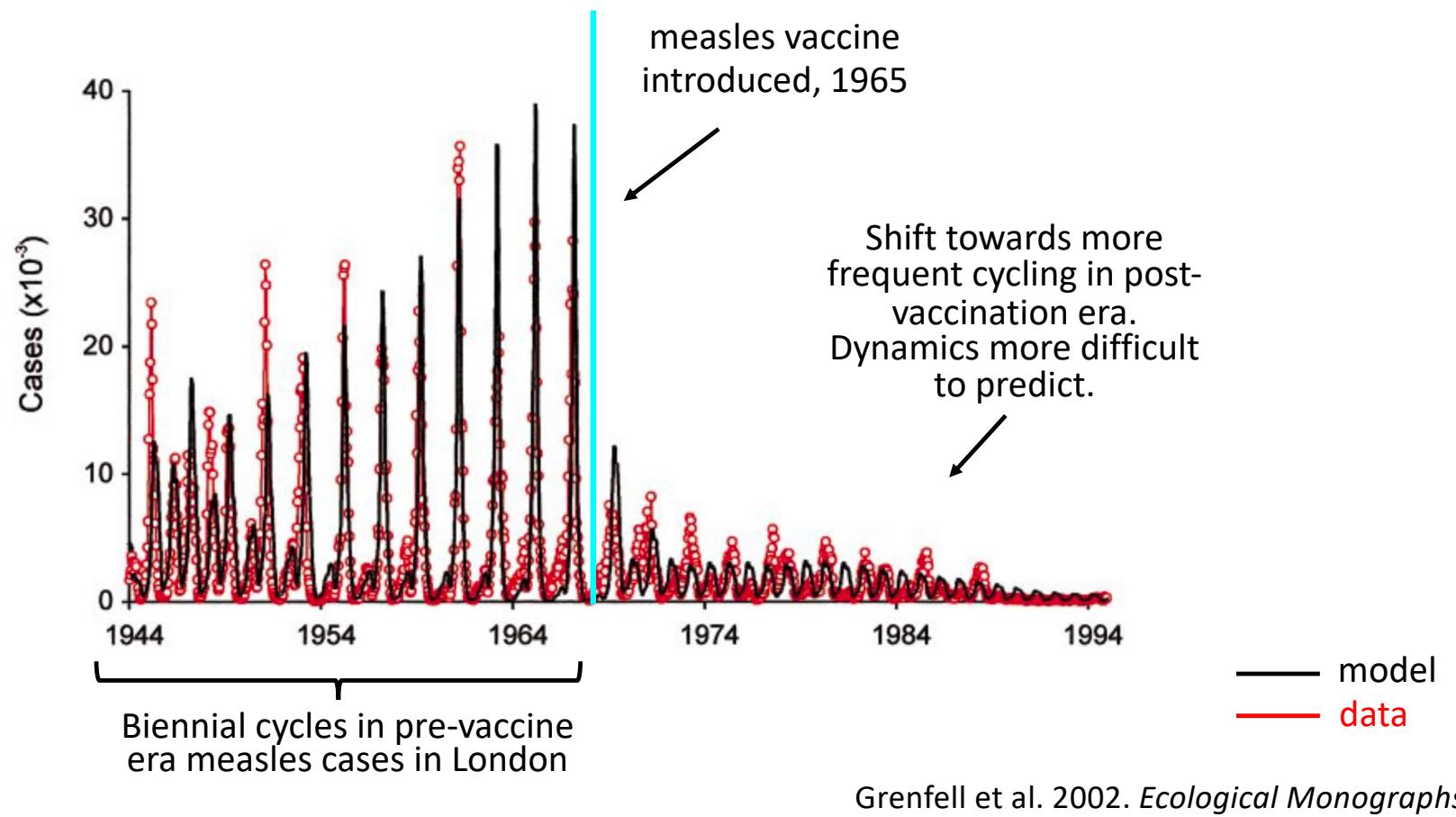
...though remember **source-sink dynamics!**



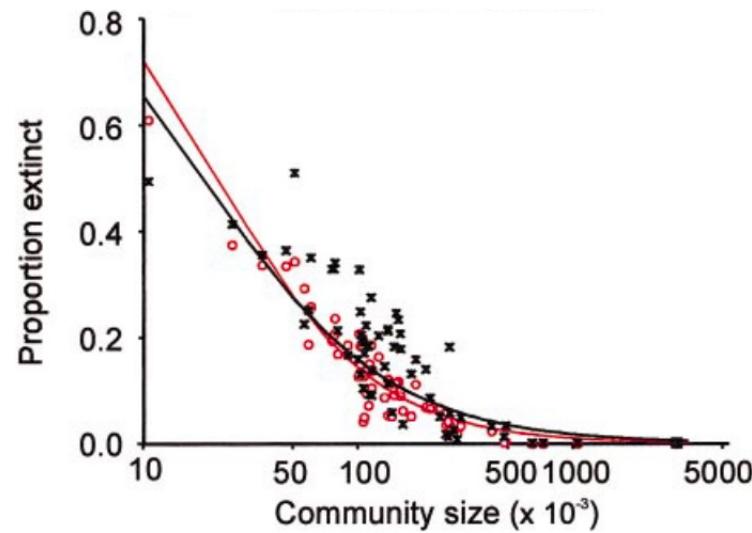
Challenges to Vaccination

- 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

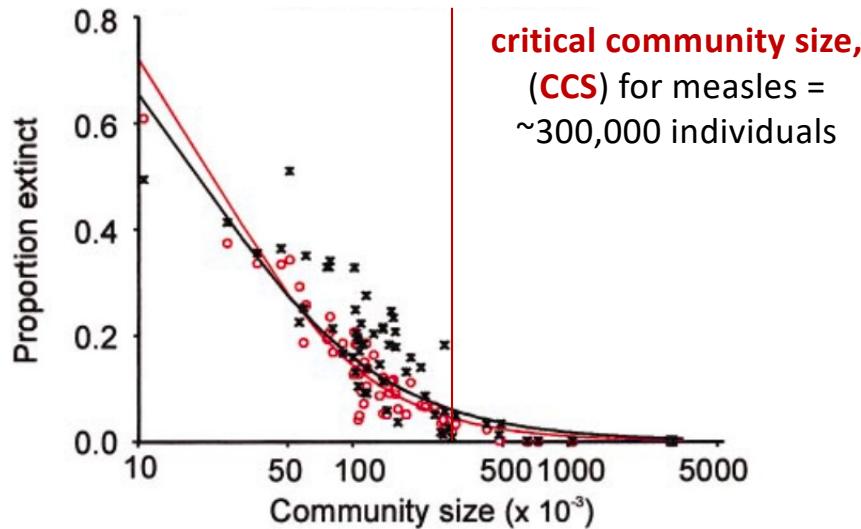


Even for measles, stochastic dynamics mean that predictions become more challenging at smaller population sizes.



Grenfell et al. 2002. *Ecological Monographs*

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



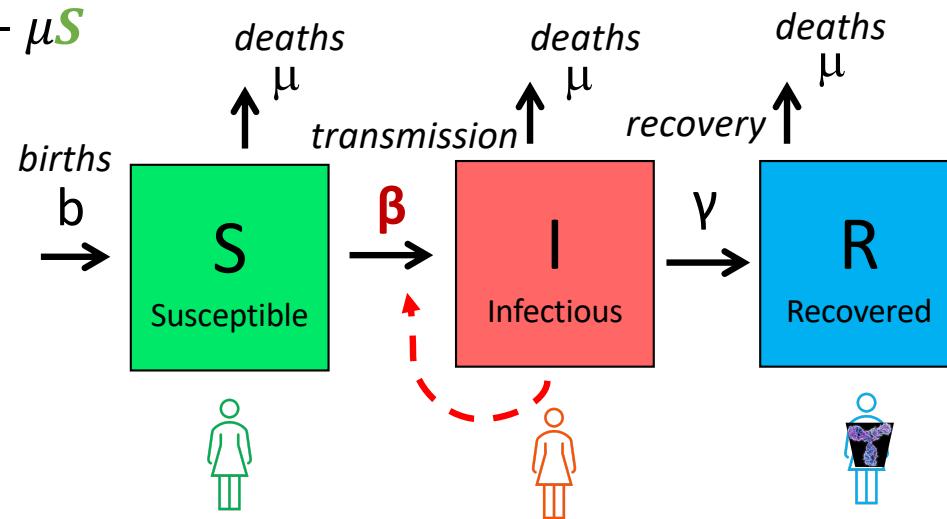
Bartlett 1957. *J of Roy Stat Soc.*
Grenfell et al. 2002. *Ecological Monographs*.
Haydon et al. 2002. *Emerging Infectious Diseases*.

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

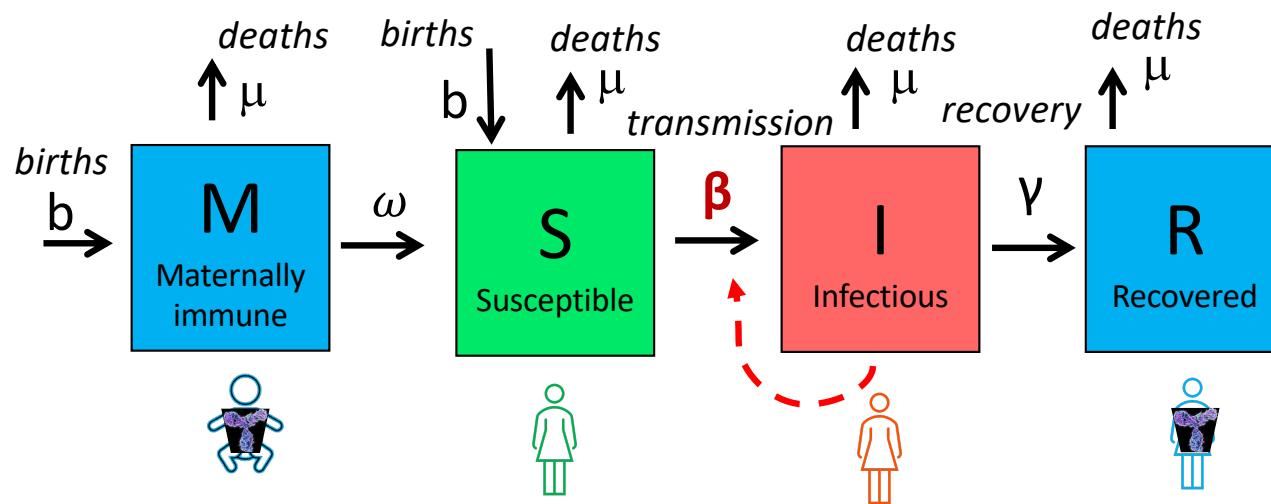
$$\frac{dS}{dt} = b(S + I + R) - \beta SI - \mu S$$

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

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



Incorporating Maternal Immunity



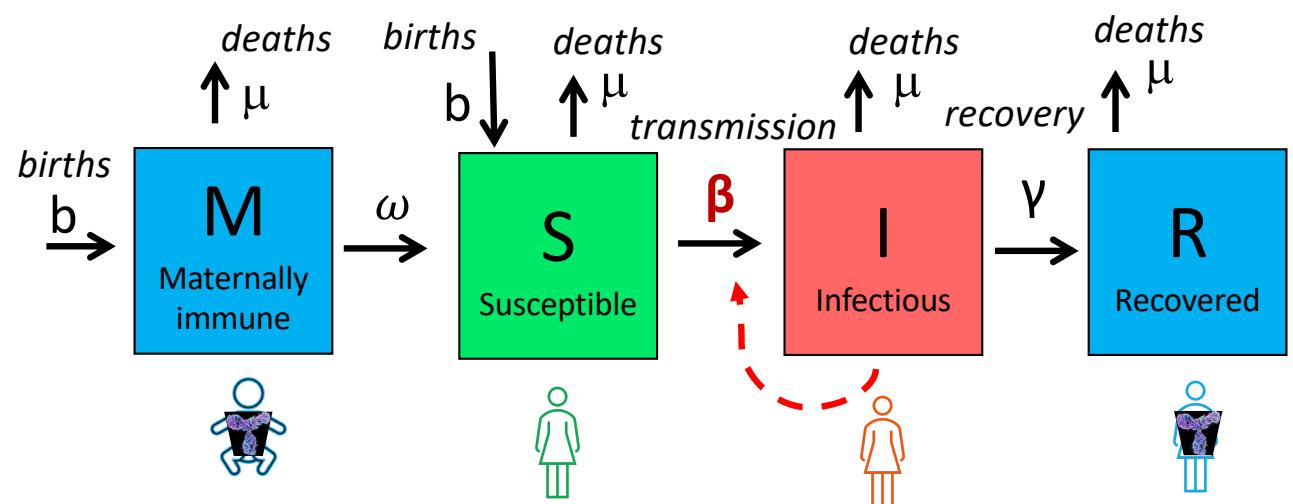
Incorporating Maternal Immunity

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

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

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

What does our new equation look like?



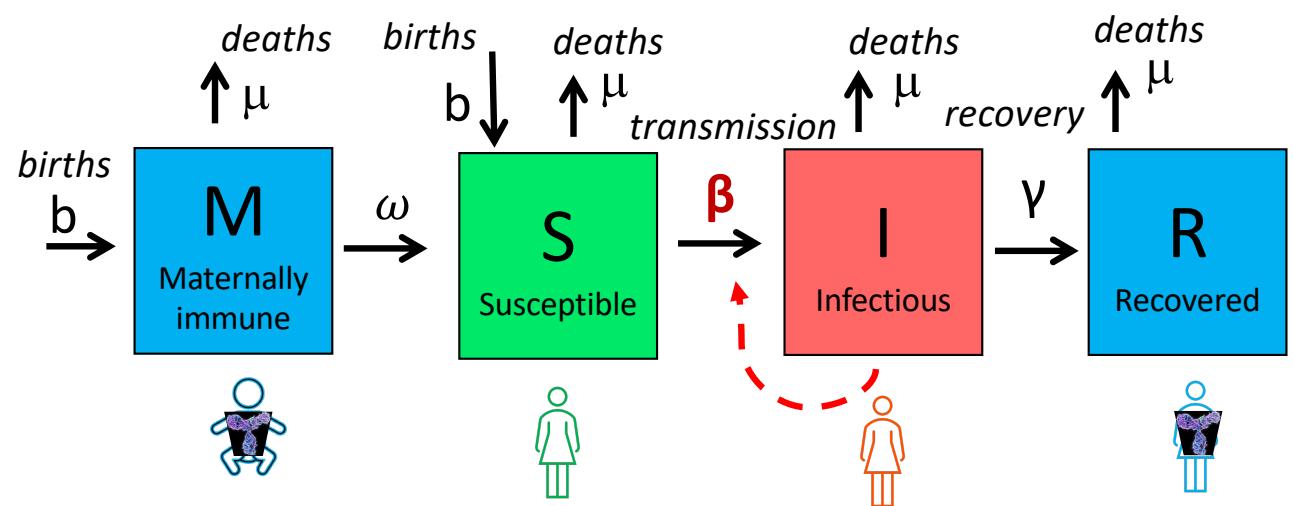
Incorporating Maternal Immunity

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

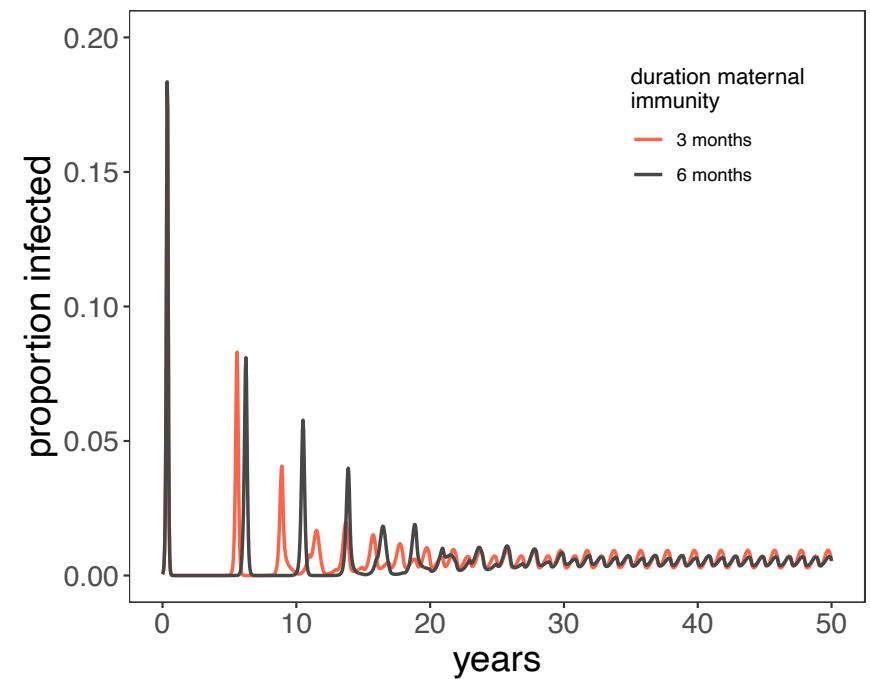
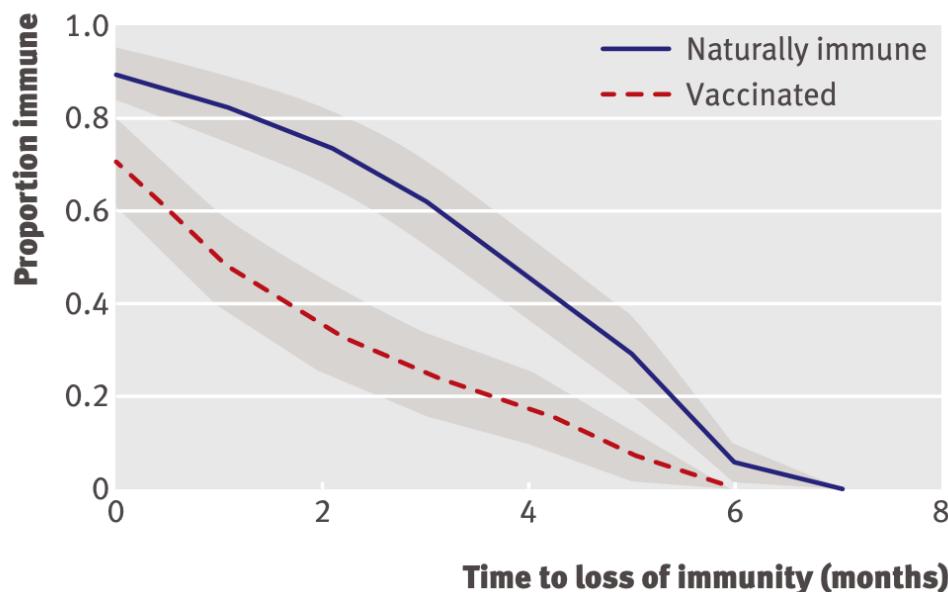
$$\frac{dS}{dt} = \omega M + bS - \beta SI - \mu S$$

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

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



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

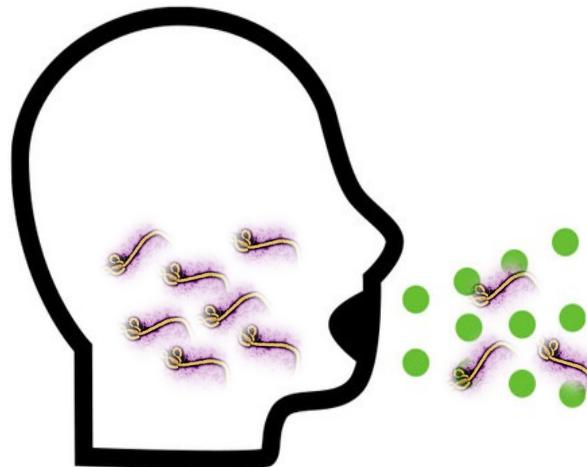
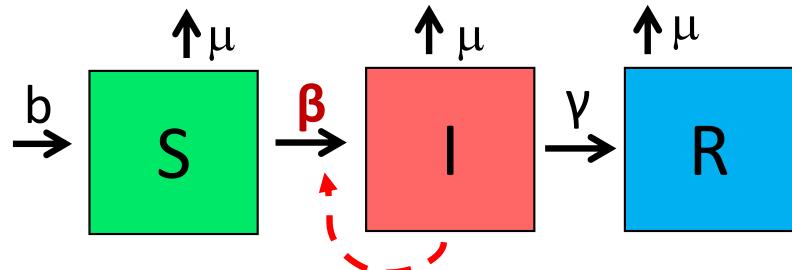


Leuridan et al. 2010. *BMJ*.

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)



**Modeled
using the
classic SIR
structure!**

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

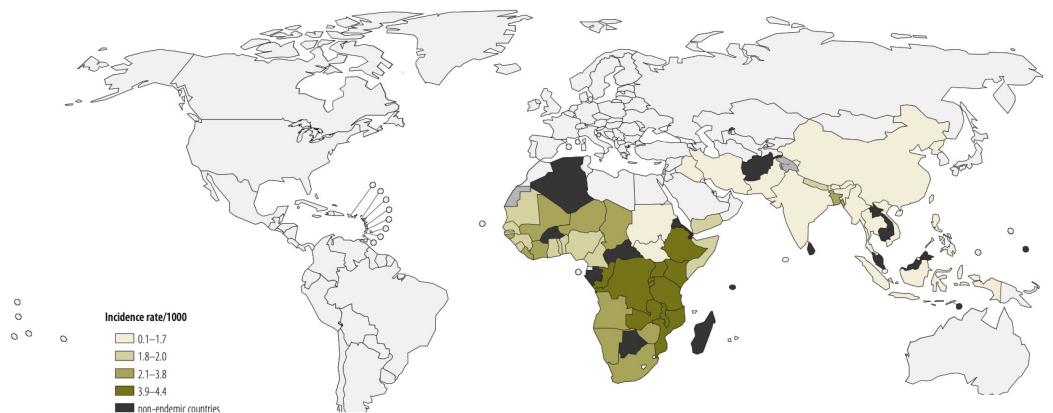
- **Environmentally-transmitted** pathogens are transmitted outside of the host (e.g. water-borne, food-borne)
 - Examples: Cholera (*Vibrio cholerae*), Salmonellosis (*Salmonella* spp. bacteria), White-Nosed Syndrome (*Pseudogymnoascus destructans*)

**Modeled with the
environmental
reservoir as a
distinct state
variable!**

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



Ali et al. 2012, WHO Bulletin.

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

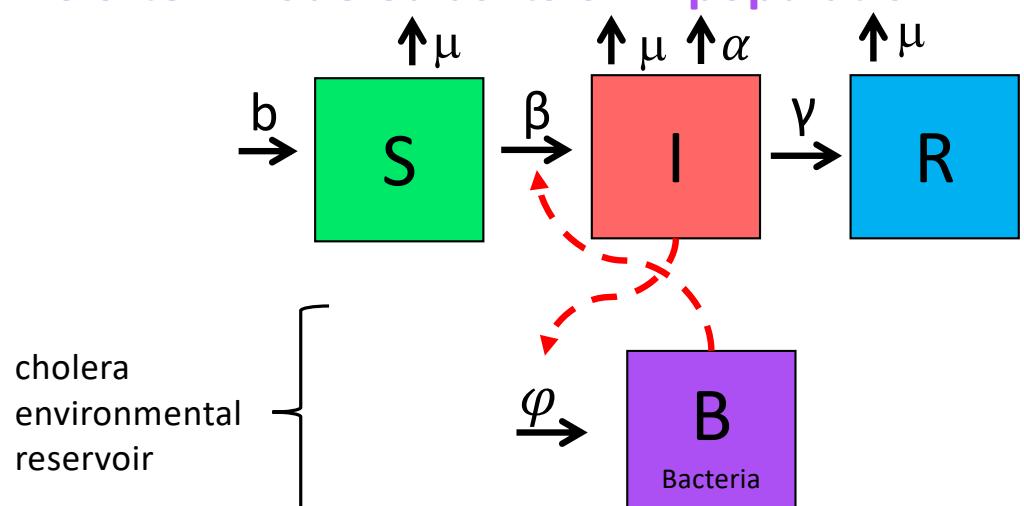
- **Environmentally-transmitted** pathogens are transmitted outside of the host (e.g. water-borne, food-borne)
 - Examples: Cholera (*Vibrio cholerae*), Salmonellosis (*Salmonella* spp. bacteria), White-Nosed Syndrome (*Pseudogymnoascus destructans*)
 - **Here, the environmental reservoir is often modeled as its own population**

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

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

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

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



Codeço 2001. *BMC Infectious Diseases*.

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

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



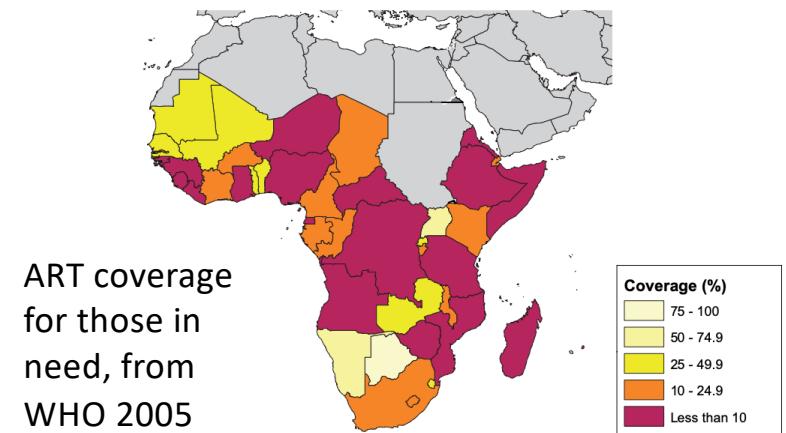
**Modeled with
inherited infection
rather than
through contact!**

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

- **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



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



Typically
modeled with
the vector as a
distinct state
variable!

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*)

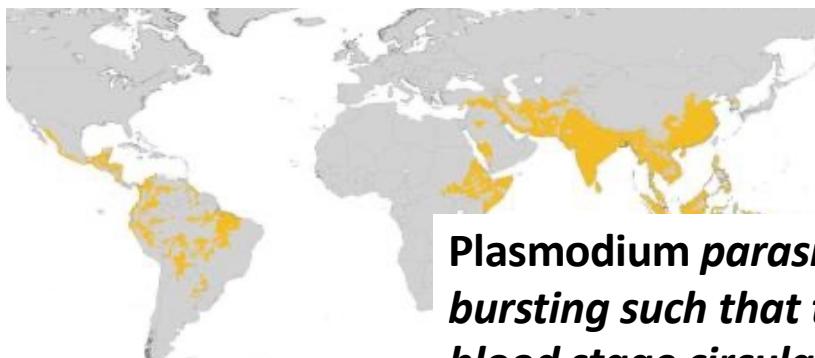
Malaria

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

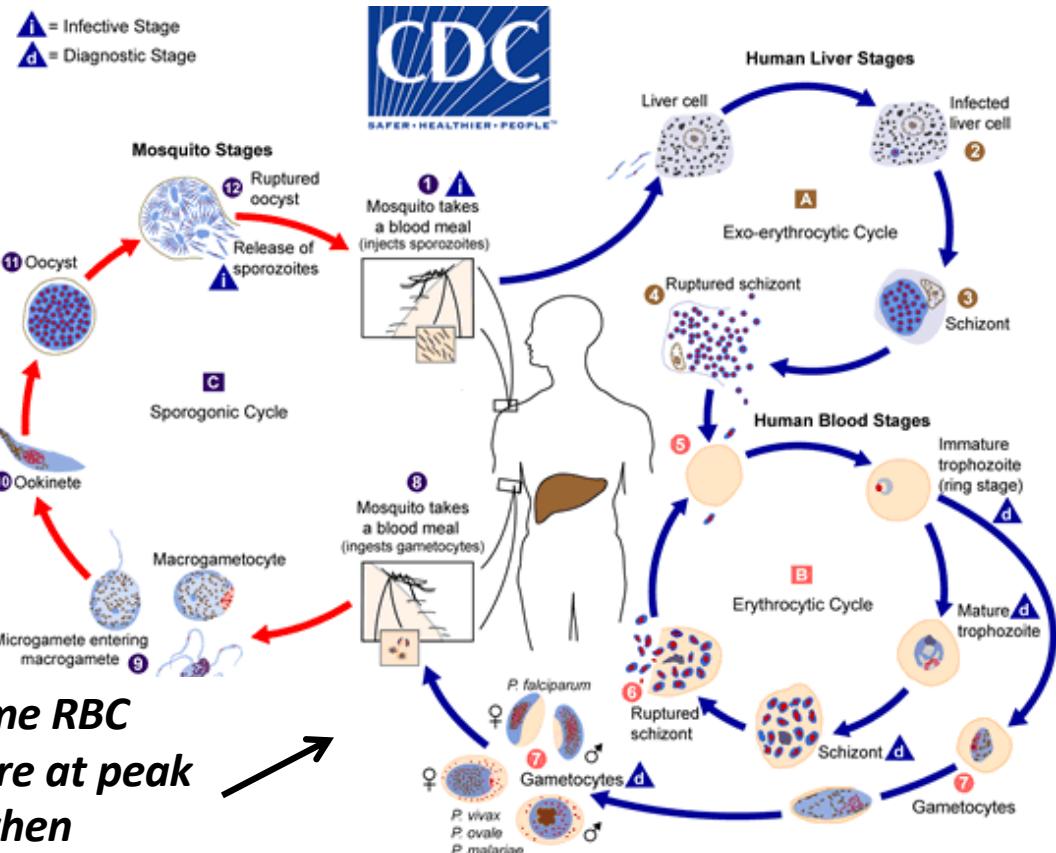
Distribution *Plasmodium falciparum*



Distribution *Plasmodium vivax*



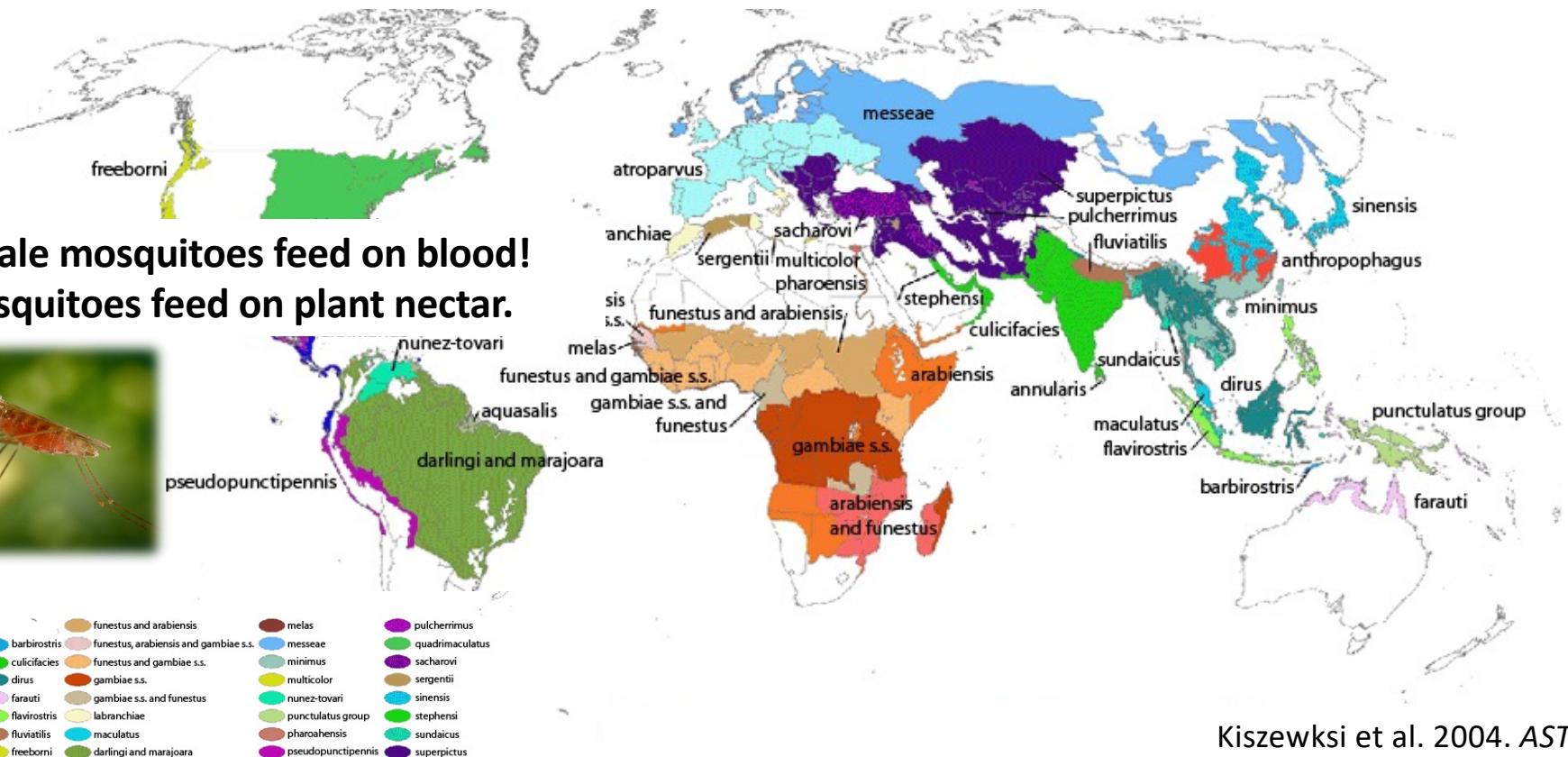
Plasmodium parasites time RBC bursting such that they are at peak blood stage circulation when mosquito vectors are feeding at dusk!



Guerra et al. 2006. Trends in **mosquito vectors are feeding at dusk!**

Malaria

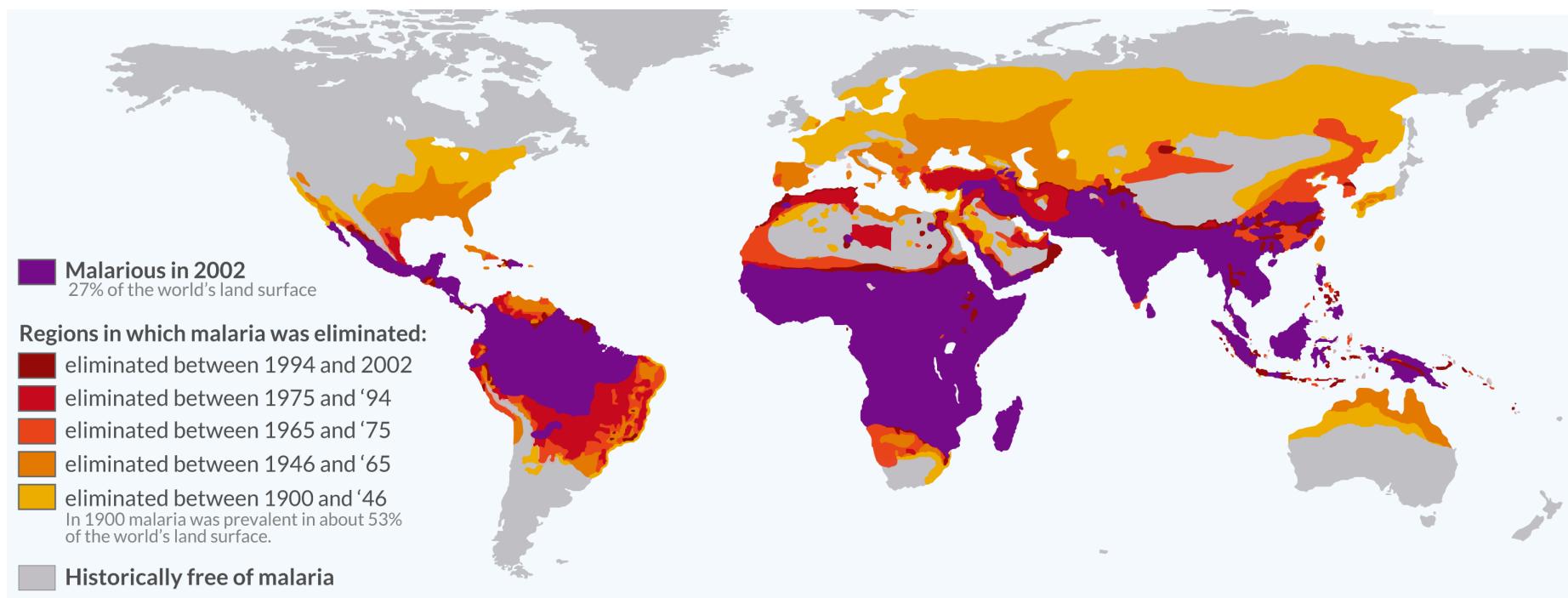
- 4 main human ***Plasmodium* parasites** (*falciparum*, *vivax*, *malariae*, *ovalae*).
- Over 200 *Plasmodium* spp. globally, infecting birds, reptiles, and other mammals (rodents, bats, primates)
- >400 global species of ***Anopheles* mosquito**, >100 that can transmit human malaria
- ~30-40 *Anopheles*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



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

Kiszewksi et al. 2004. *ASTMH*.

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.

OurWorldinData.org

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

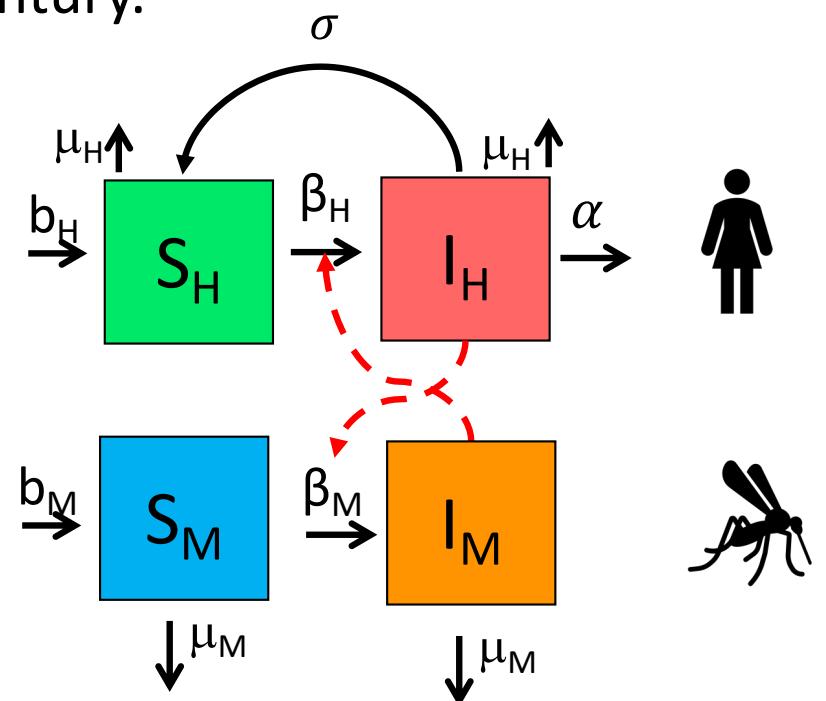
- 1911: British medical Dr. Sir Ronald Ross developed the first model of malaria while working in the Indian Medical Service.
 - He had already won the 1902 Nobel prize in physiology and medicine for discovering the life cycle of avian malaria

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

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

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

$$\frac{dI_M}{dt} = \beta_M S_M I_H - \mu_M I_M$$



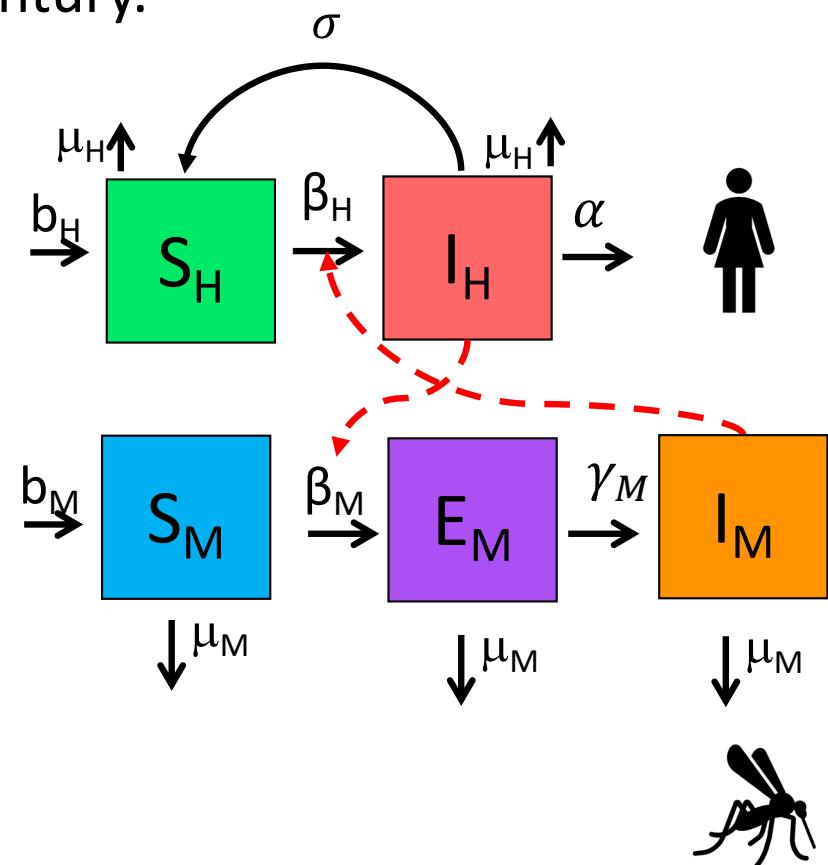
Malaria models have played a critical role in public health policy for over a century.

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

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

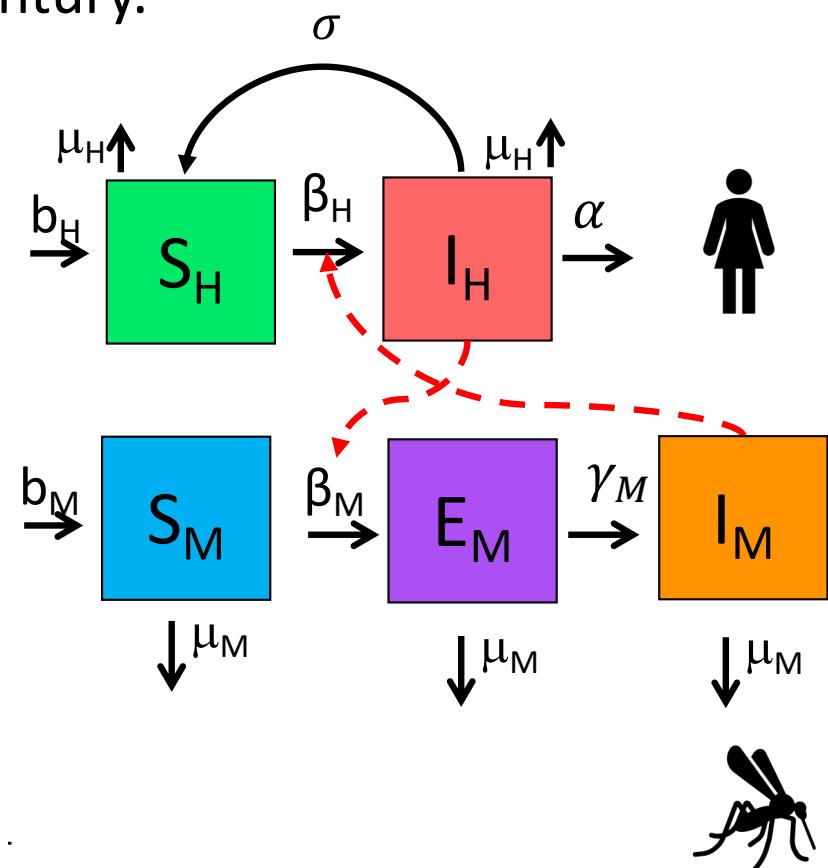
$$\frac{dE_M}{dt} = \beta_M S_M I_H - \mu_M E_M - \gamma_M E_M$$

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



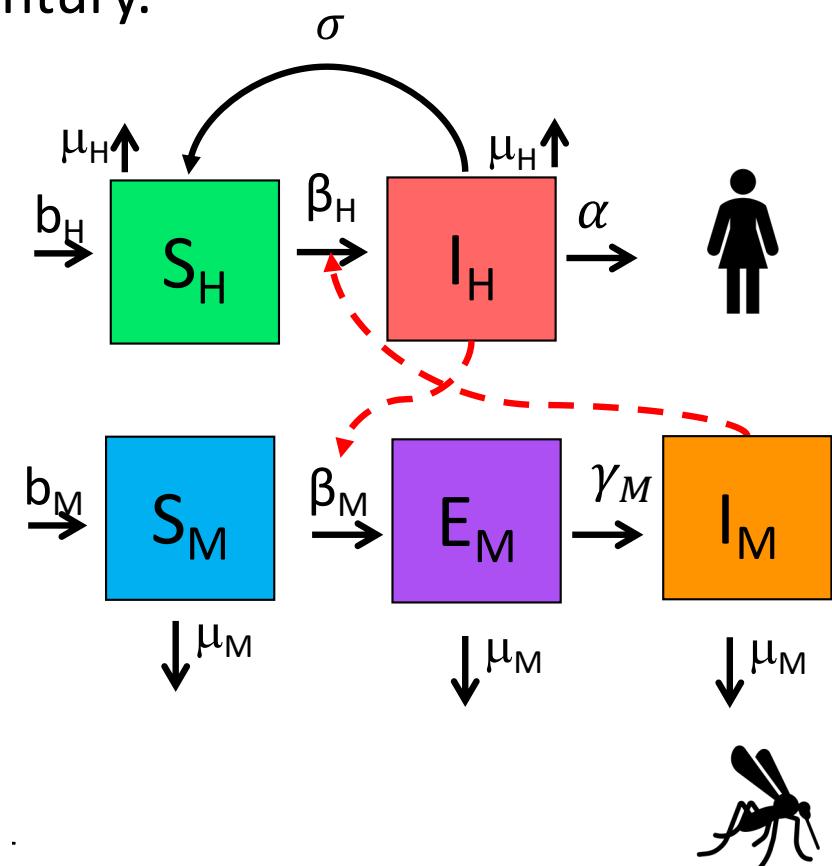
Malaria models have played a critical role in public health policy for over a century.

- 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!



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



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*

Bioaccumulation:

the gradual accumulation of substances in an organism through time; particularly dangerous for **high trophic level consumers**



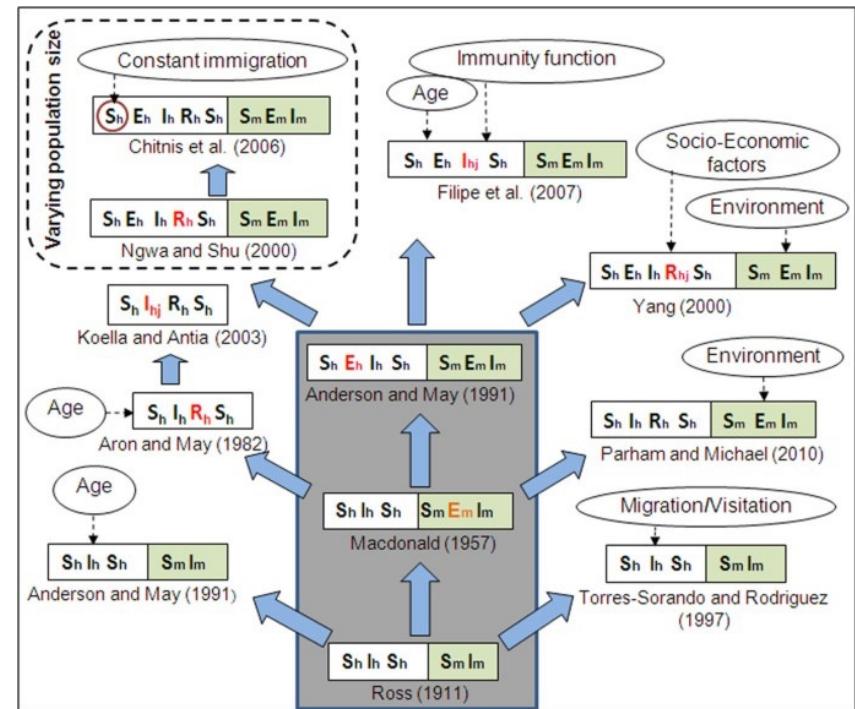
Malaria models have played a critical role in public health policy for over a century.

- 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



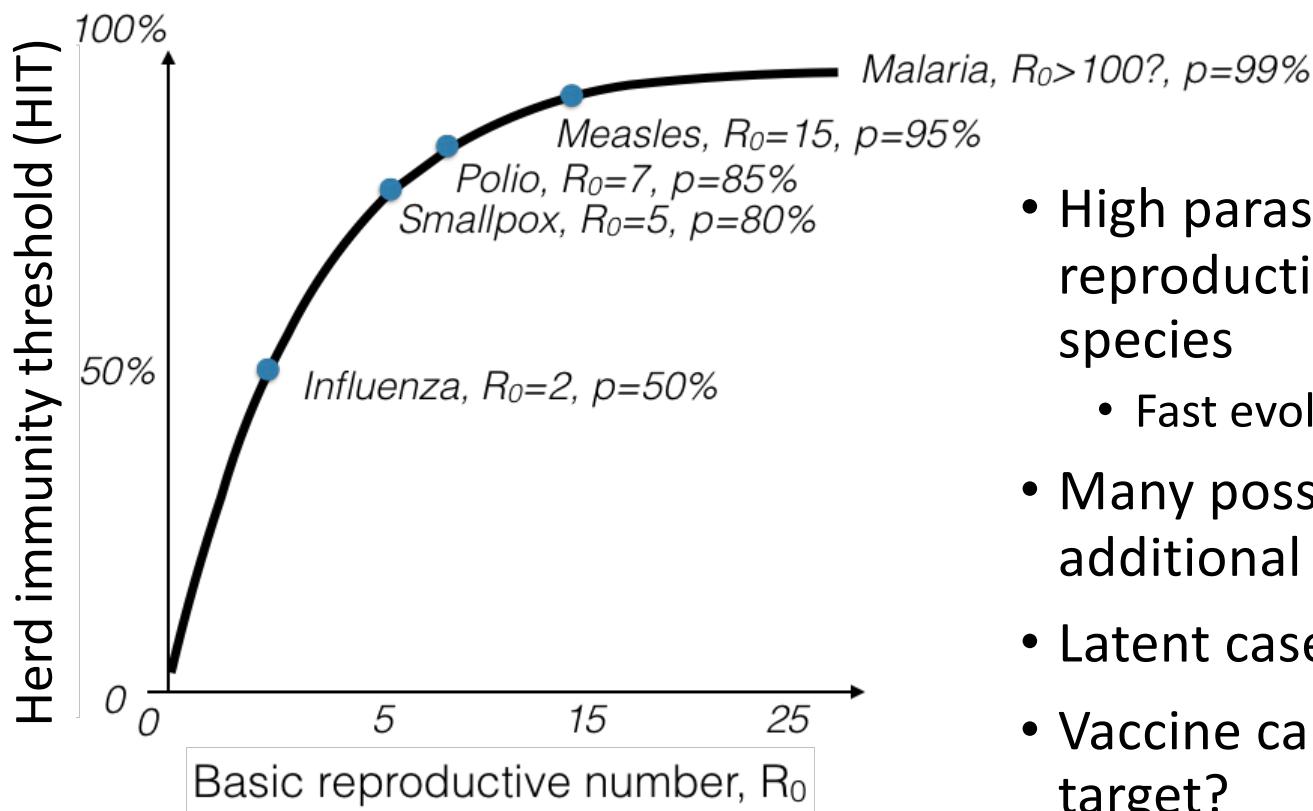
Malaria models have played a critical role in public health policy for over a century.

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



Mandal et al. 2011. *Malaria Journal*.

Challenges to malaria elimination



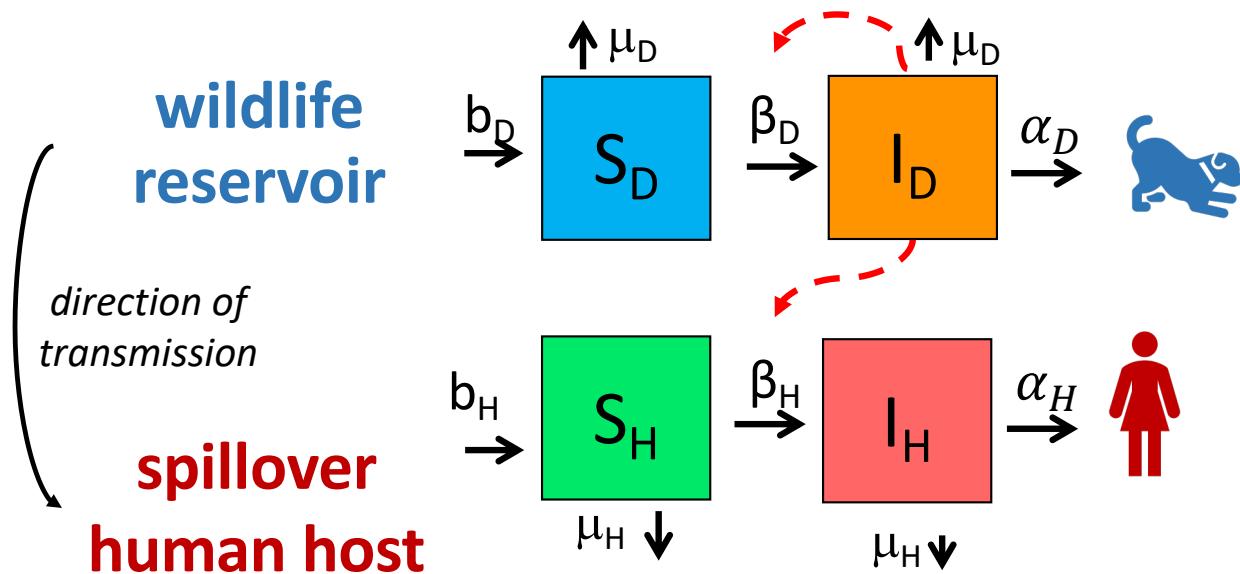
- High parasite diversity: sexual reproduction in 4+ *Plasmodium* species
 - Fast evolution of resistance (e.g. to drugs)
- Many possible vectors! Potentially additional possible reservoirs!
- Latent cases as burden is reduced
- Vaccine candidates: what life stage to target?

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.

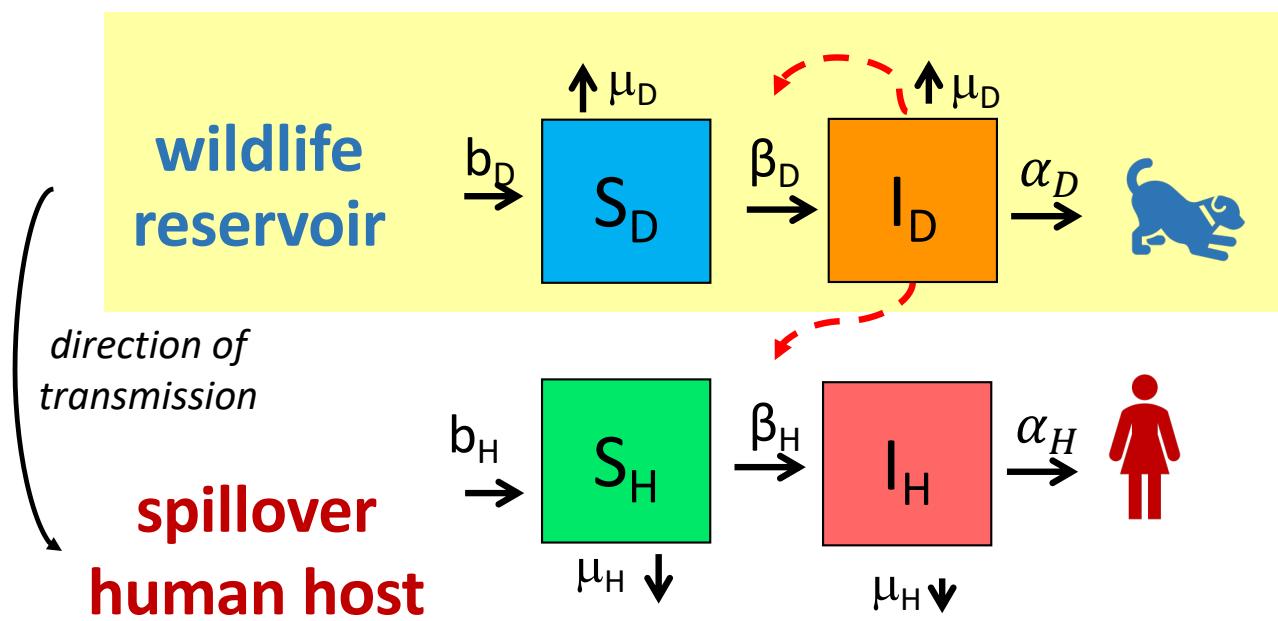


Typically modeled with the reservoir as a distinct state variable!

ex: rabies

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

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



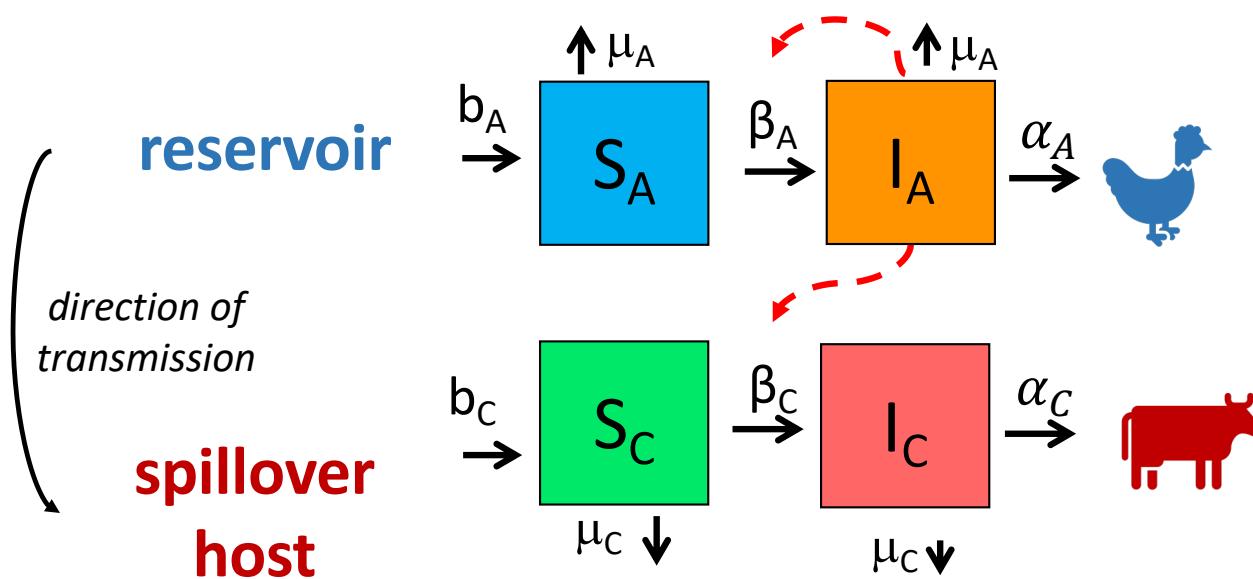
The **reservoir** host must be able to independently maintain the pathogen, with **population size > CCS!**

Animal hosts are not vectors!

ex: rabies

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

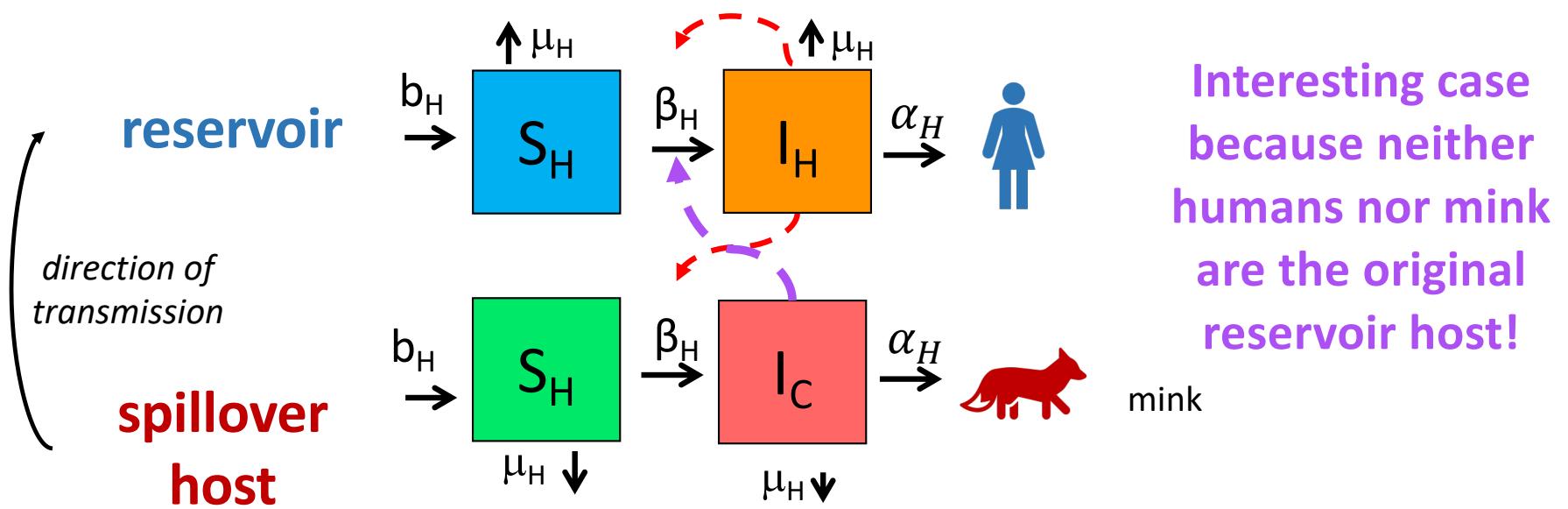
Spillover is the term used to describe pathogen transmission between any two different species.



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

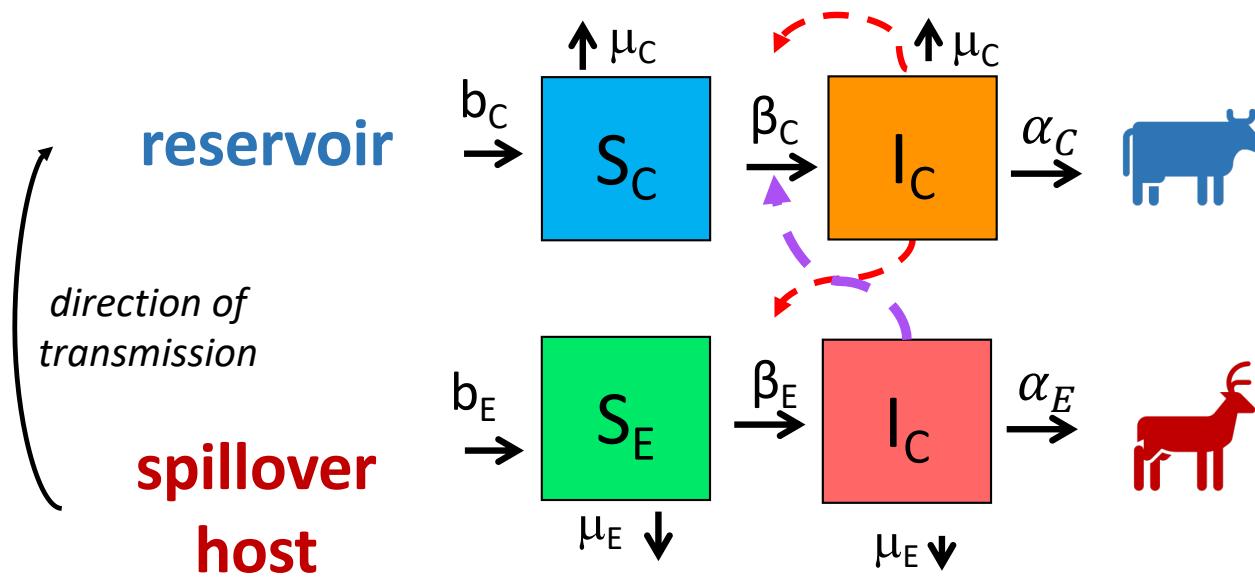
ex: avian flu

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



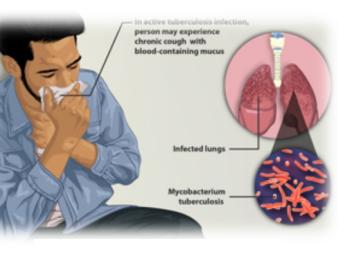
ex: SARS-CoV-2

Spillback occurs among wildlife as well.



ex: *Brucella* spp. in Yellowstone National Park

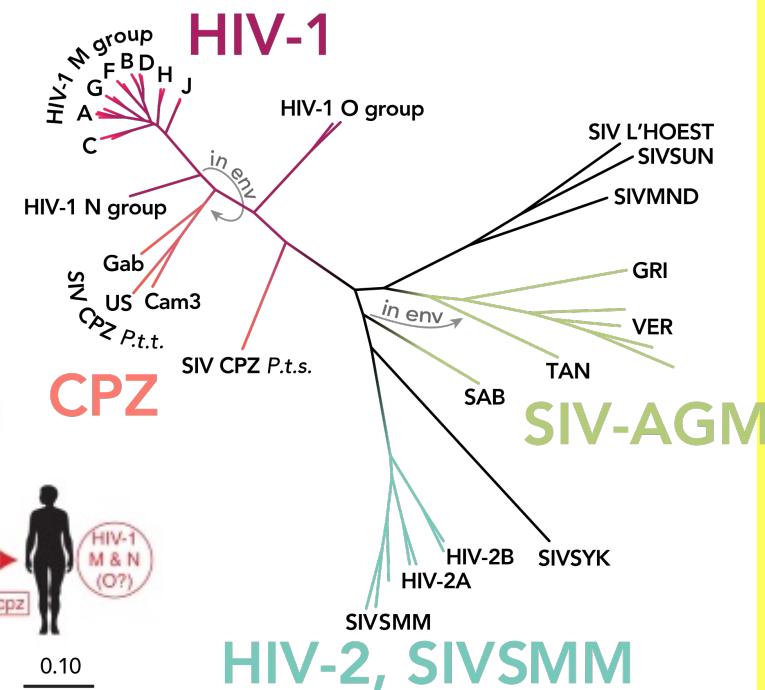
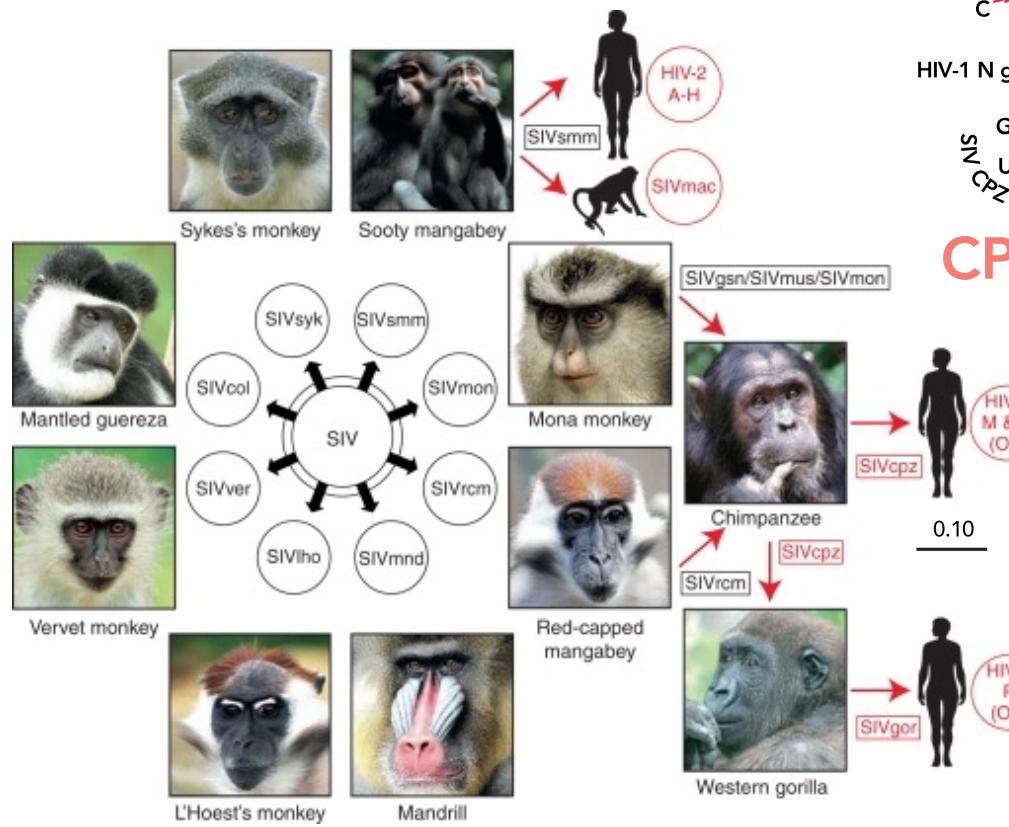
Pathogens can be classed according to their host relationships.

| <u>Stage I</u> | <u>Stage II</u> | <u>Stage III</u> | <u>Stage IV</u> | <u>Stage V</u> |
|--|--|---|--|--|
| Transmits exclusively in animals | Human cases from spillovers only | Stuttering chains of transmission in humans | Sustained transmission and human outbreaks | Transmits exclusively in humans |
|  canine parvovirus |  rabies virus |  monkeypox (pre-2022) |  Ebola virus (especially post-2014) |  Tuberculosis |

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

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

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



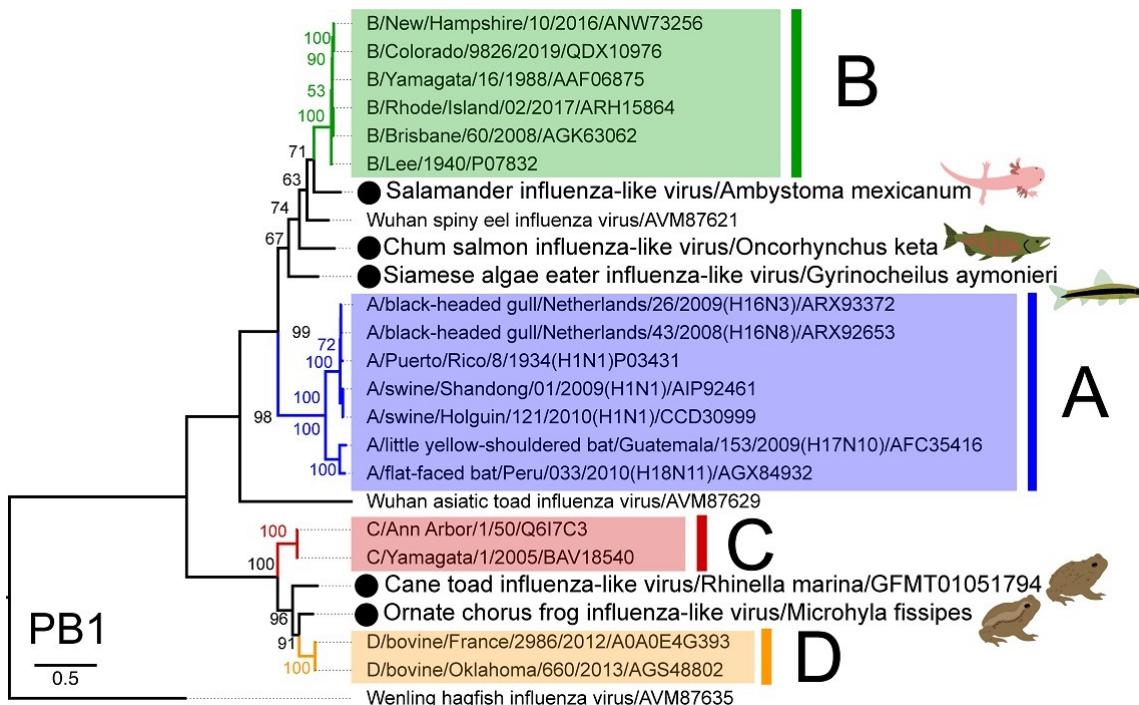
Stage V
Transmits exclusively in humans



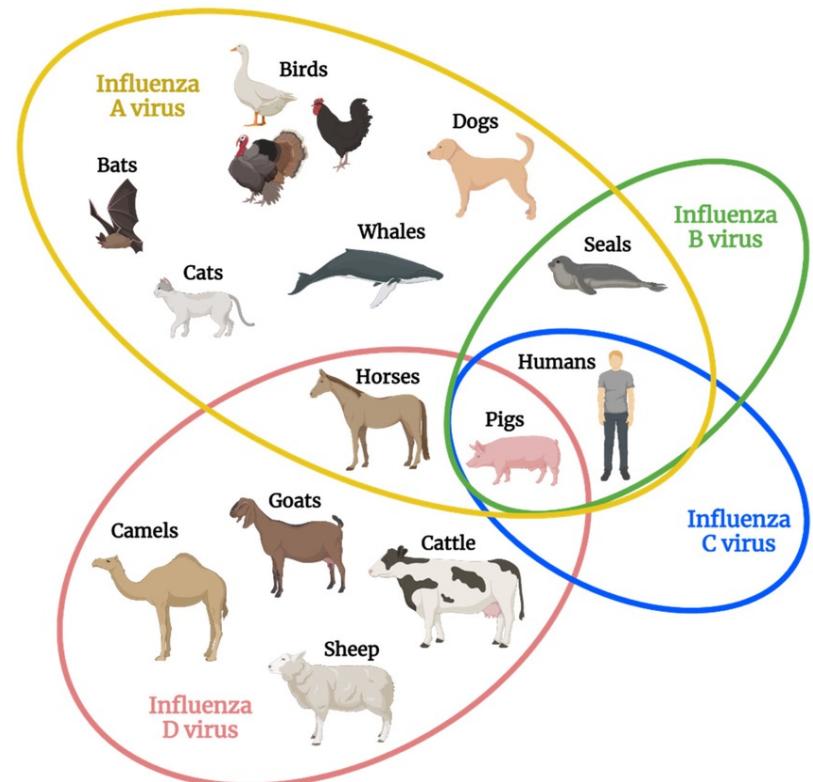
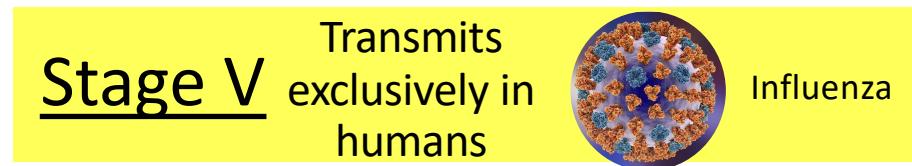
HIV

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

When is influenza zoonotic?



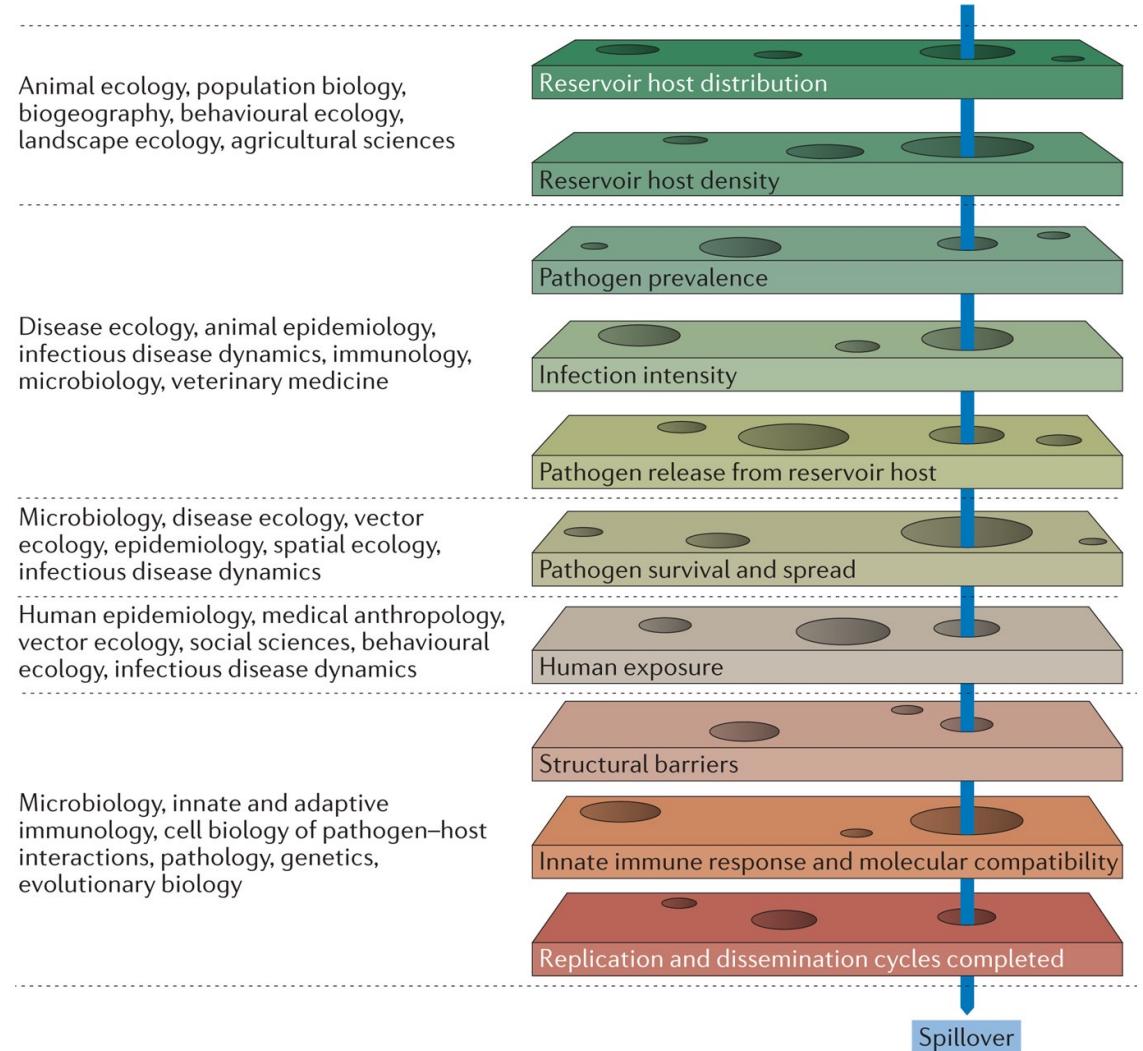
Parry et al. 2020. *Viruses*.



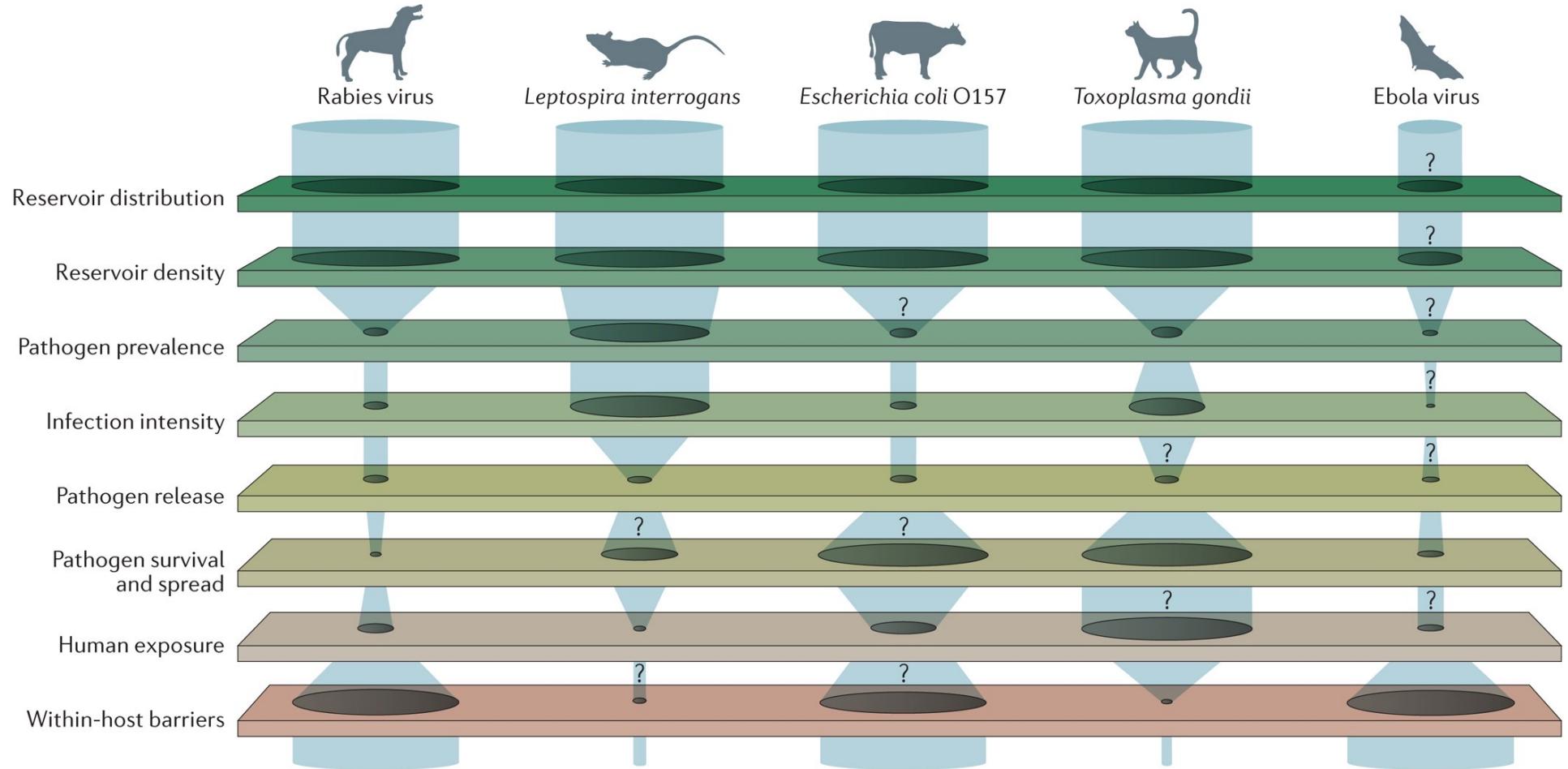
Skelton & Huber. 2022. *Viruses*.

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

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



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



Bottlenecks to zoonotic transmission
vary for different pathogens.

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