Ecology 8310
Population (and Community) Ecology

Disease ecology

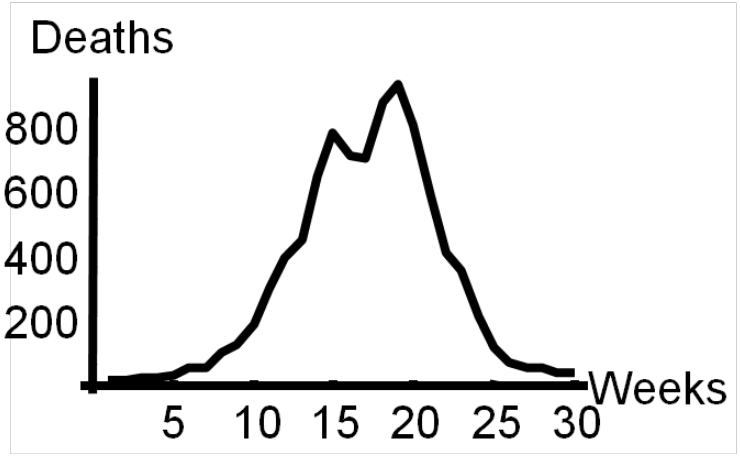
- Examples (e.g., measles)
- SIR models
- Epidemics
- Vaccinations
- Persistence (endemics)

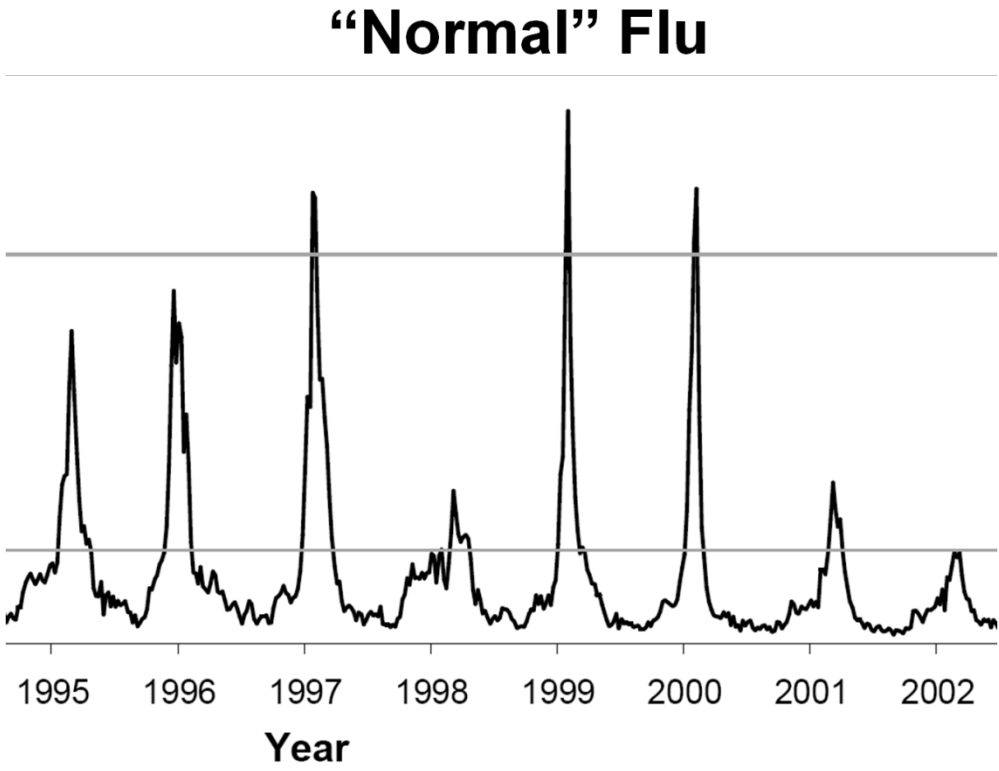
Thanks to Drew Kramer

Why study host-parasite interactions?

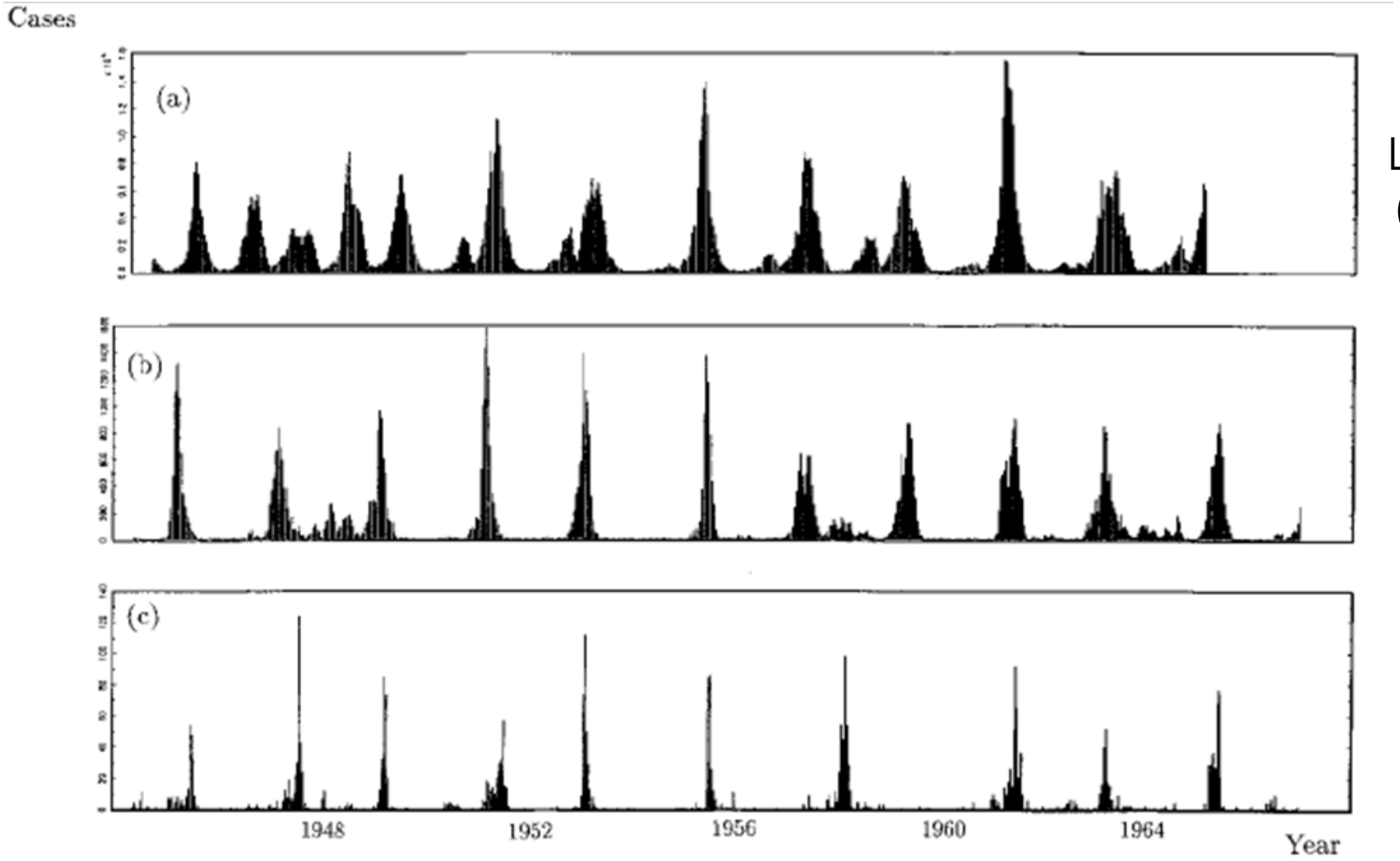
- Interesting dynamics (e.g., cycles)

Plague: Weekly deaths in Bombay (1906):





Measles dynamics (pre-vaccination): 1944-1966



Large city (London)

Medium

Small

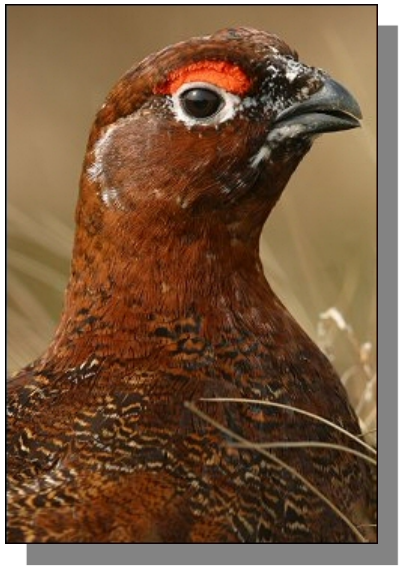
Finkenstadt, Bjornstat & Grenfell (2002)

Why study host-parasite interactions?

- Major regulator of wildlife population dynamics

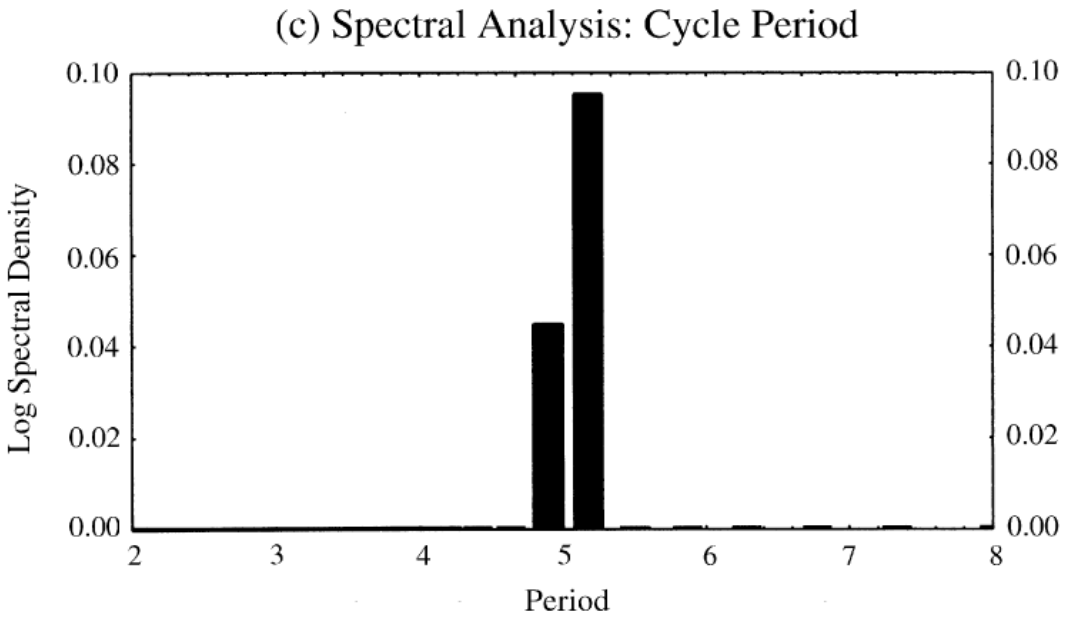
Red Grouse

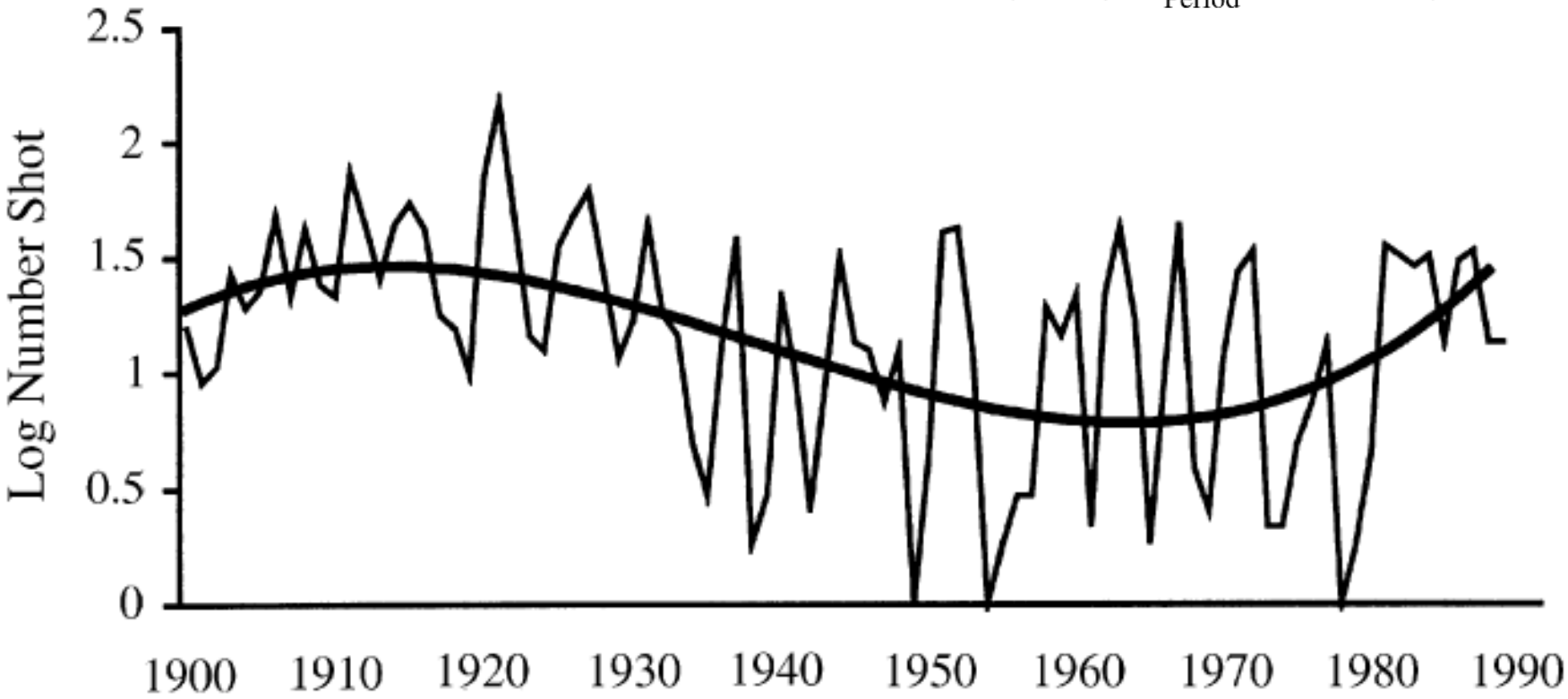
(Lagopus lagopus scoticus)

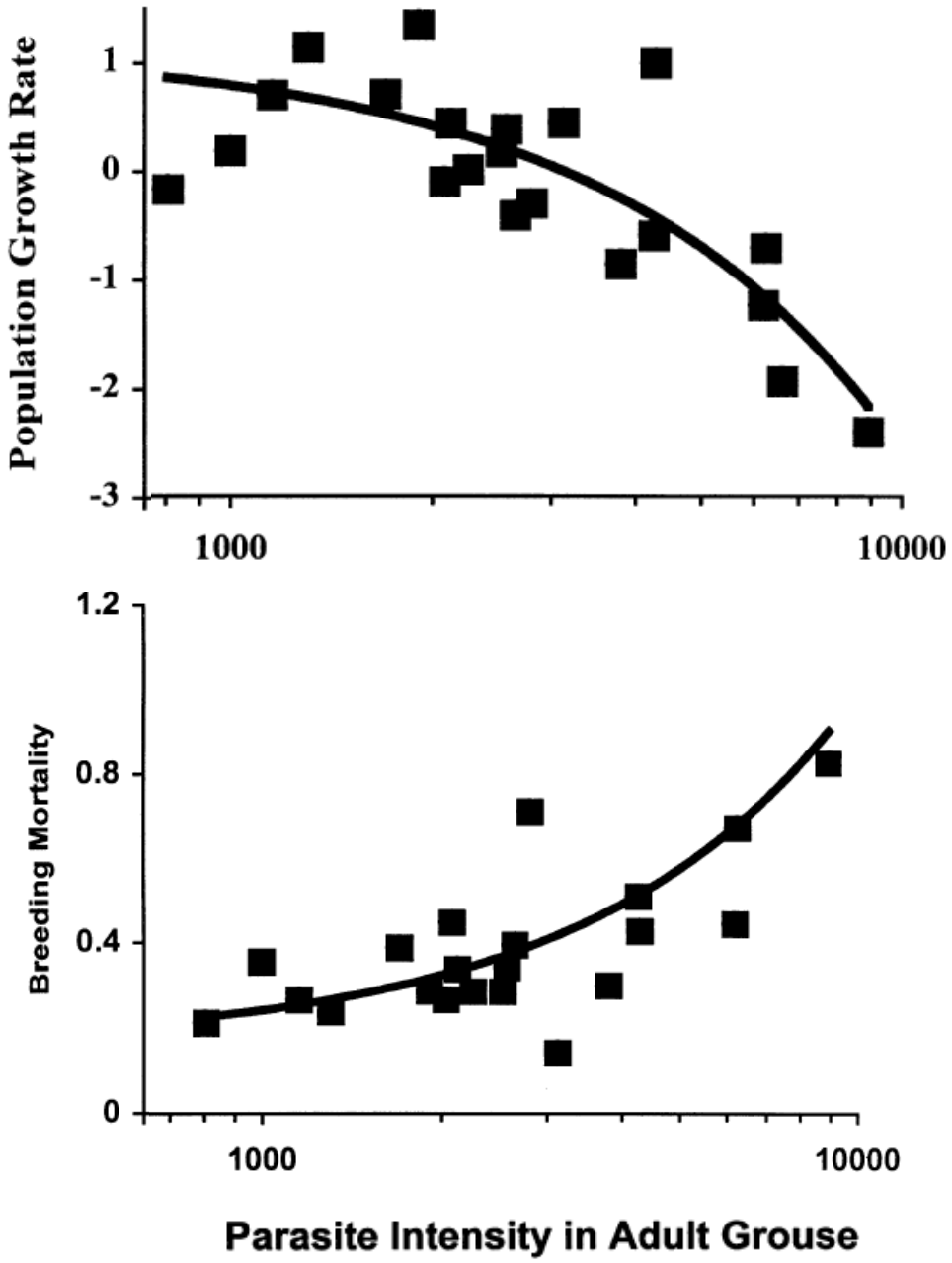


Trichostrongylus tenuis

Red grouse dynamics:

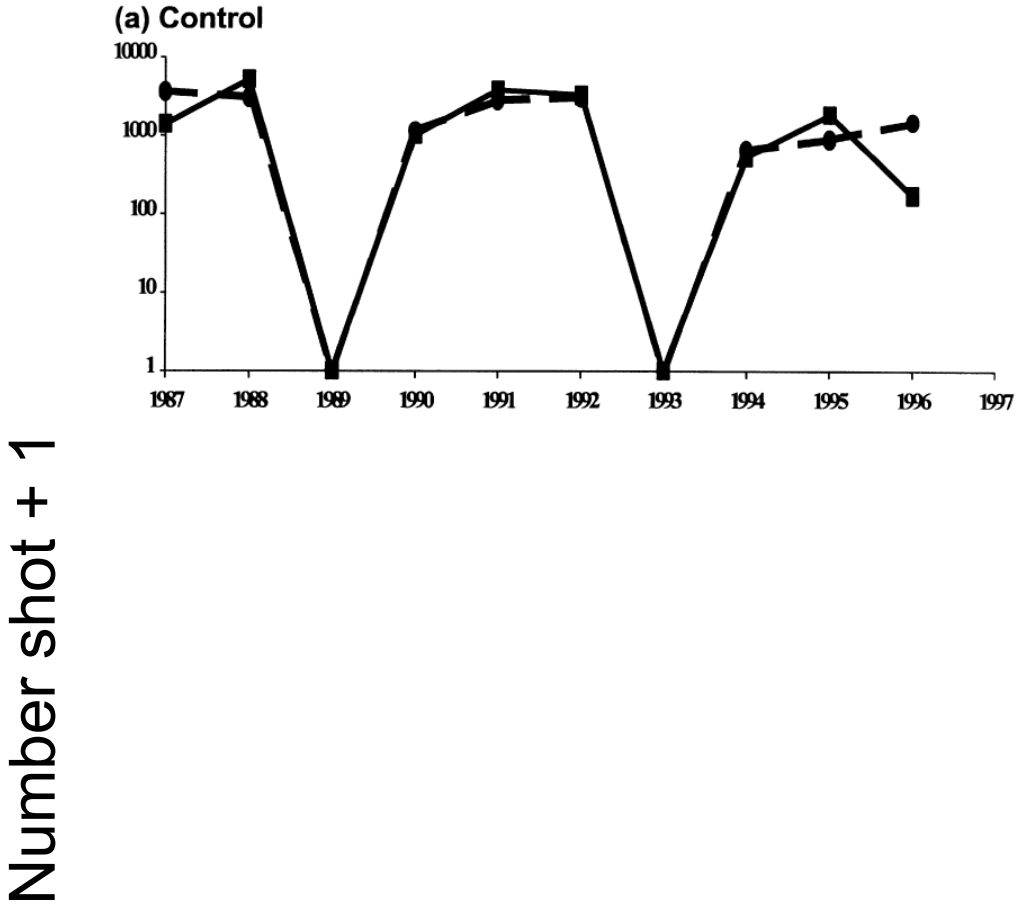





Population Growth Rate
1
0
-1
-2
-3
1000
10000
Breeding Mortality
1.2
0.8
0.4
0
1000
10000
Parasite Intensity in Adult Grouse

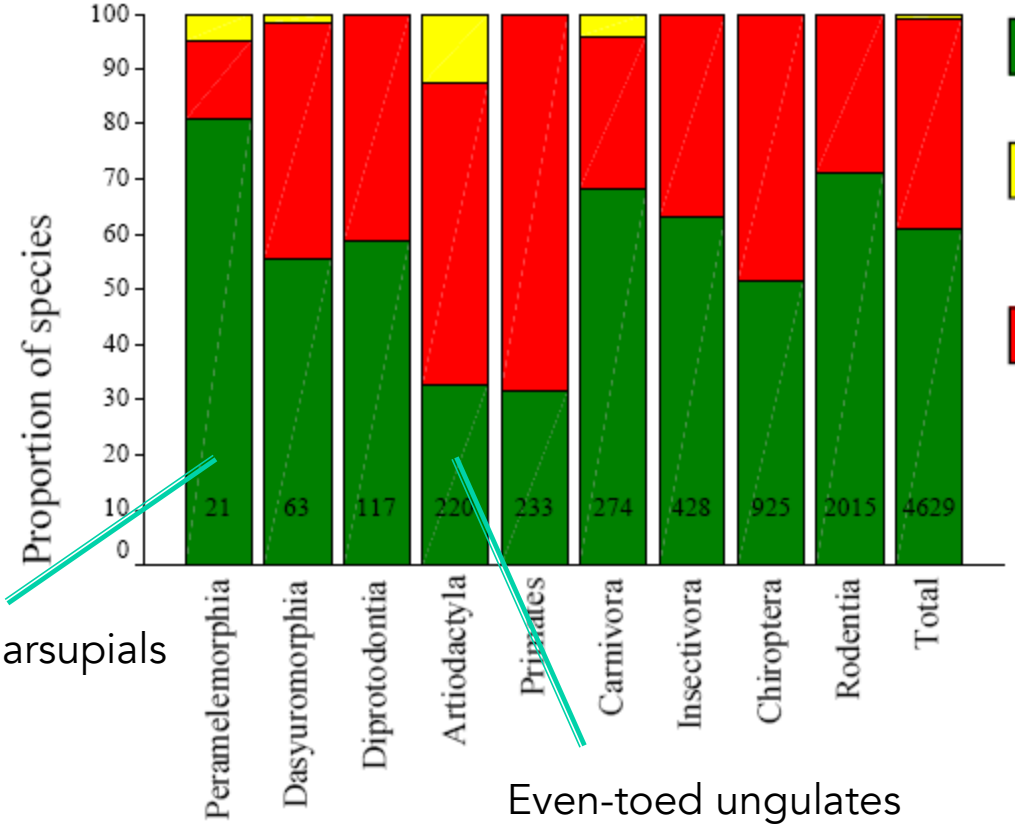
Field experiment (n=2):

Remove parasites in ~20% of grouse



Why do ecologists study host-parasite interactions?

- Cause of decline for threatened and endangered species

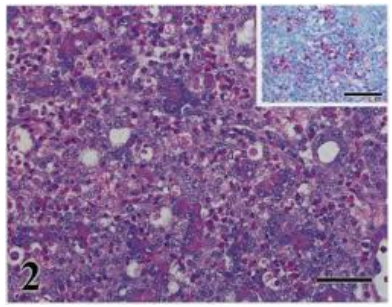


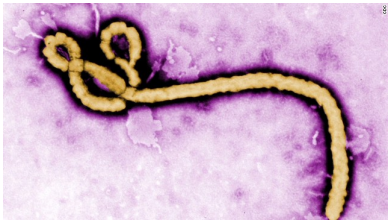
Omnivorous marsupials

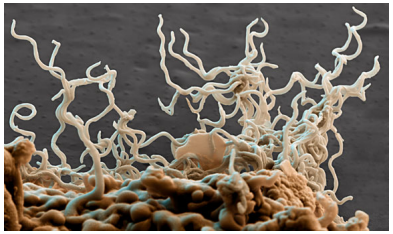
Even-toed ungulates

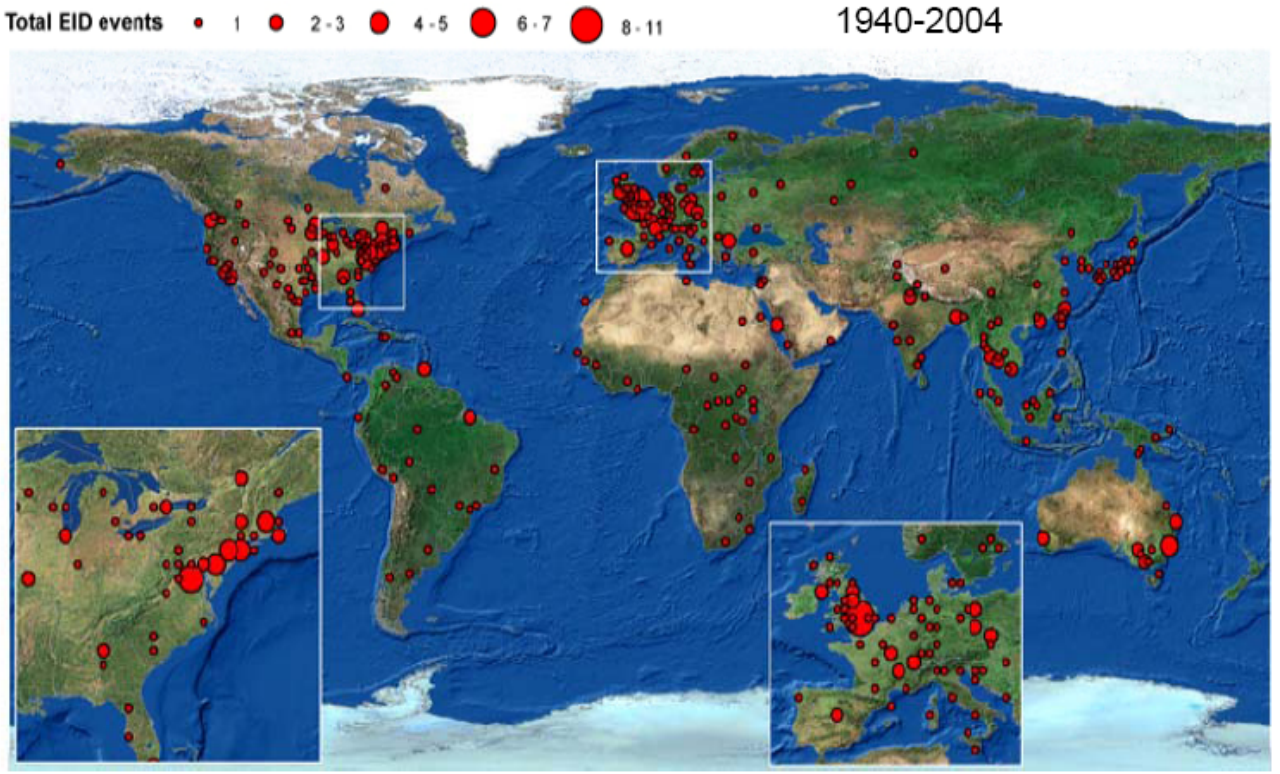
Why do ecologists study host-parasite interactions?

- Zoonoses are the main source of emerging infectious diseases in humans









Jones et al. 2008

Issues:

What determines if there will be an epidemic?

Why does it die out?

Why does it recur?

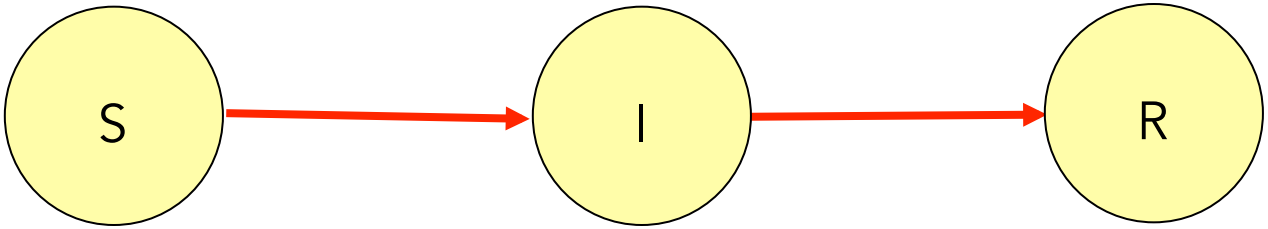
Let’s start by building a model…

SIR model:

Three states of host:

1) Susceptible (S)
2) Infected (I)
3) Removed (R): dead or immune

[N = S+I+R]



No host demography (no births or deaths)

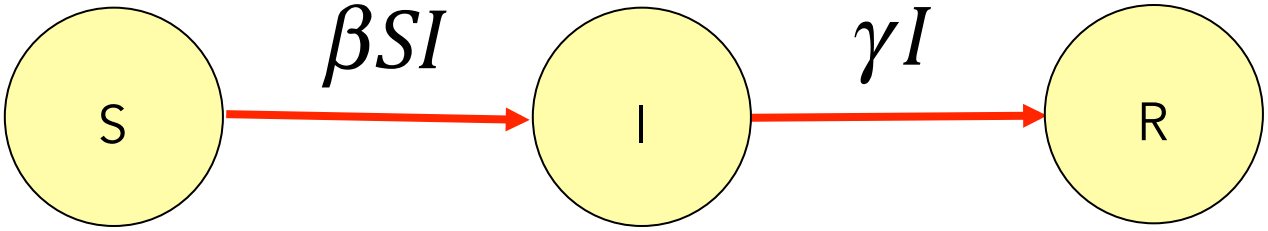
All hosts start as “S”

SIR model:

Need to specify:

1) Conversion of S to I

2) Removal (I to R):



β is "transmission rate": set by contact rate and infection probability ("random encounter")

γ is removal rate

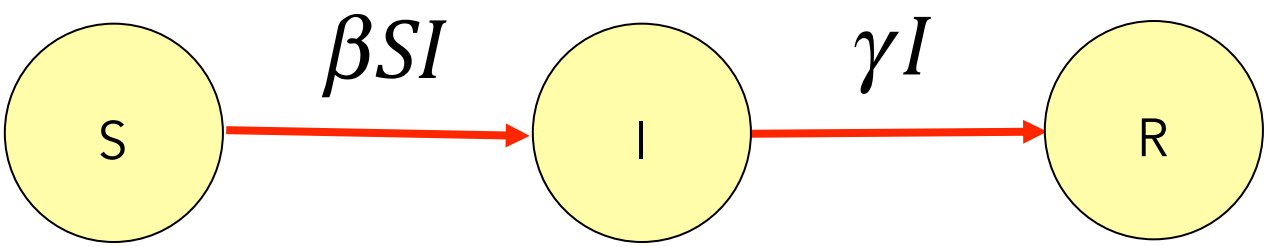
$1/\gamma$ is mean time an individual remains infective

SIR model:

$$dS/dt = -\beta SI$$

$$dI/dt = \beta SI - \gamma I$$

$$dR/dt = \gamma I$$



Is there an epidemic?

does an Infected infect a Susceptible before s/he Recovers (or dies)?

SIR model:

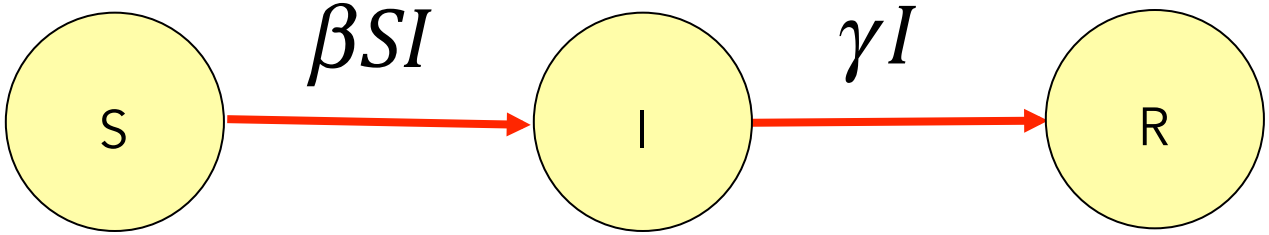
An epidemic requires:

$dI/dt > 0$

i.e., $\beta S > \gamma$

i.e., $\beta S / \gamma > 1$

recall $1/\gamma$ is duration of infection
so $\beta S / \gamma$ is the number of new infections per infection



Basic Reproductive Number:

$$R_0 = \beta S / \gamma$$

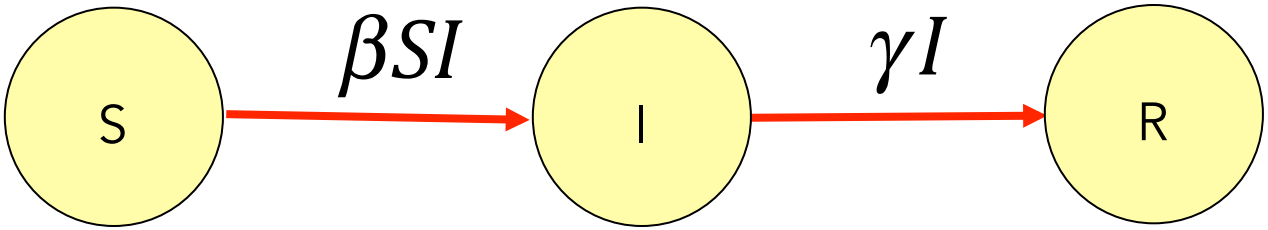
SIR model:

Is there an epidemic?

At start, all individuals are susceptible:

N=S, so is

$$R_0 = \beta N / \gamma > 1?$$



SIR model:

Epidemic more likely if:

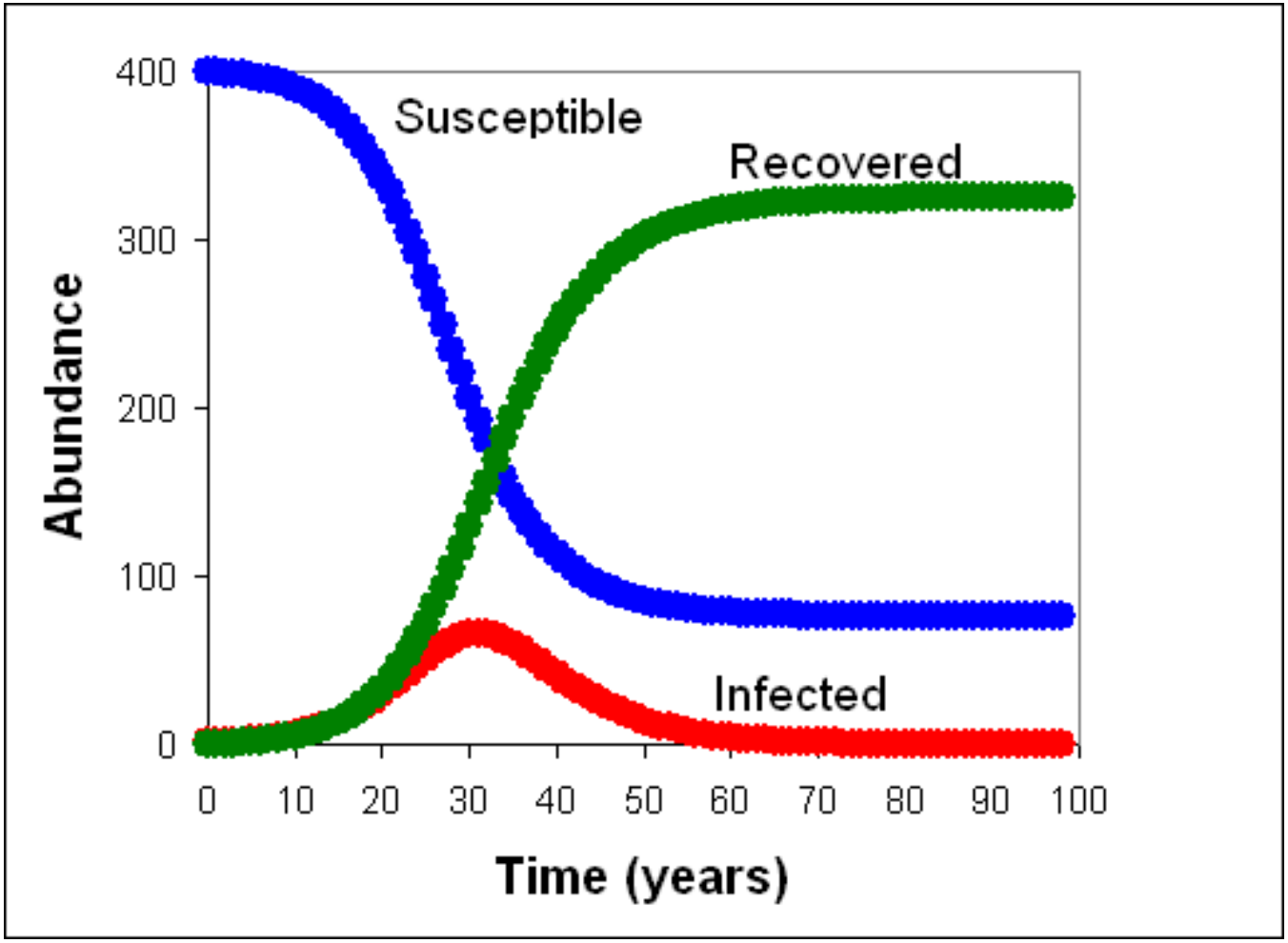
1) **N is large** (more contact with susc.)
2) β **is large** (more contacts; more likely to transmit given contact)
3) γ **is small** (stay infectious longer)

Critical community size:

$$N = \gamma / \beta$$

SIR model:

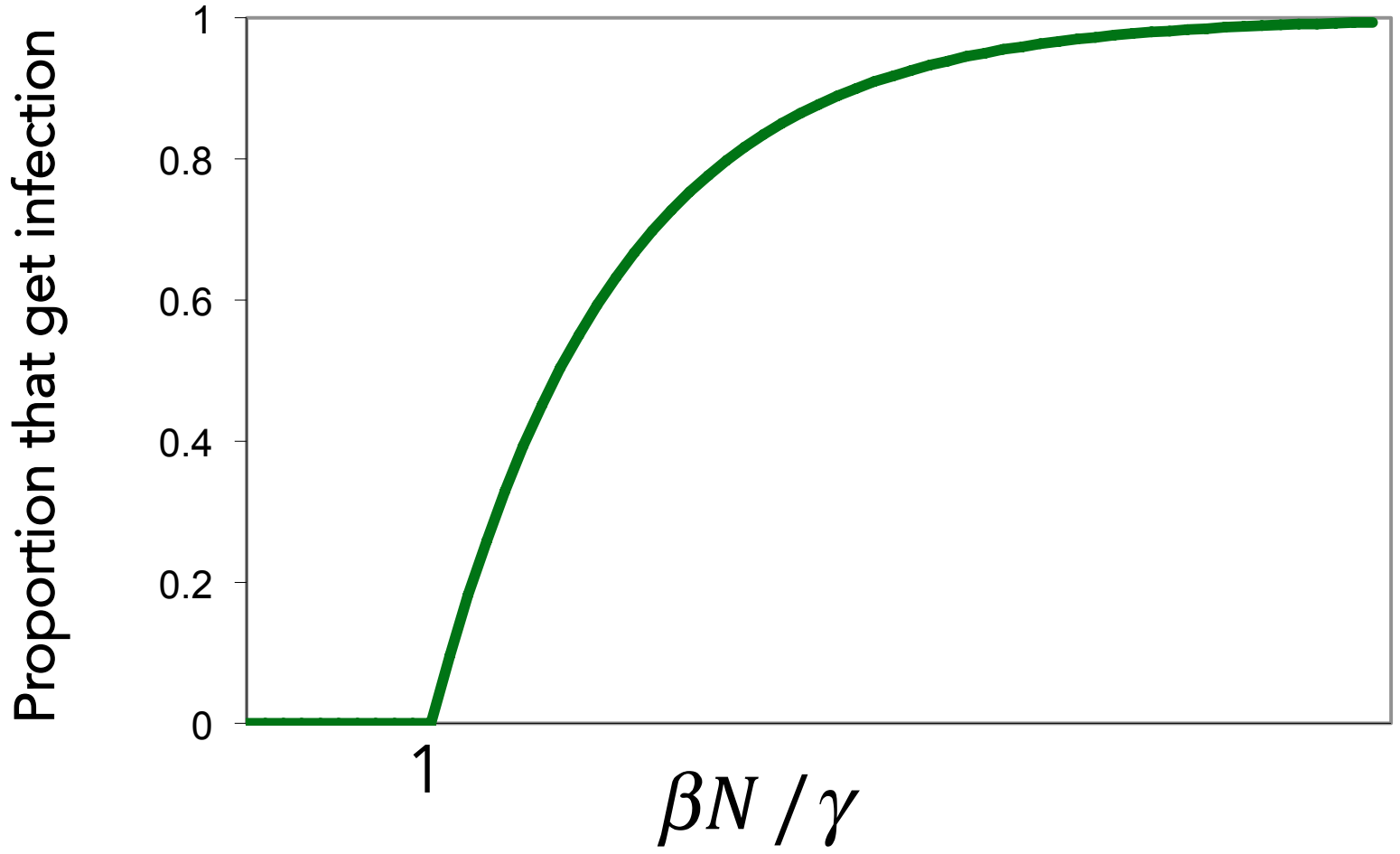
What do the dynamics look like?



Why doesn't everyone get the disease?

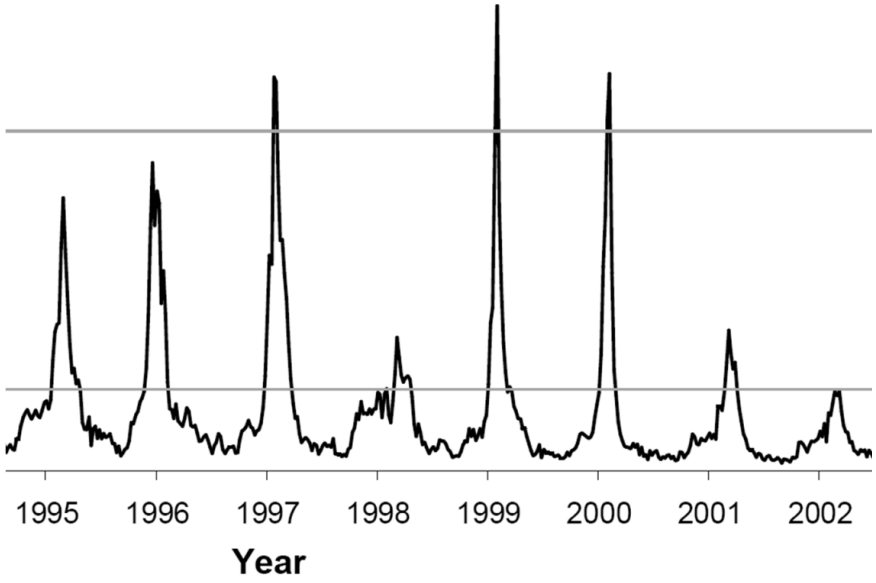
SIR model:

What fraction of population gets infected?



Sequential epidemics? (cycles)

Endemics:



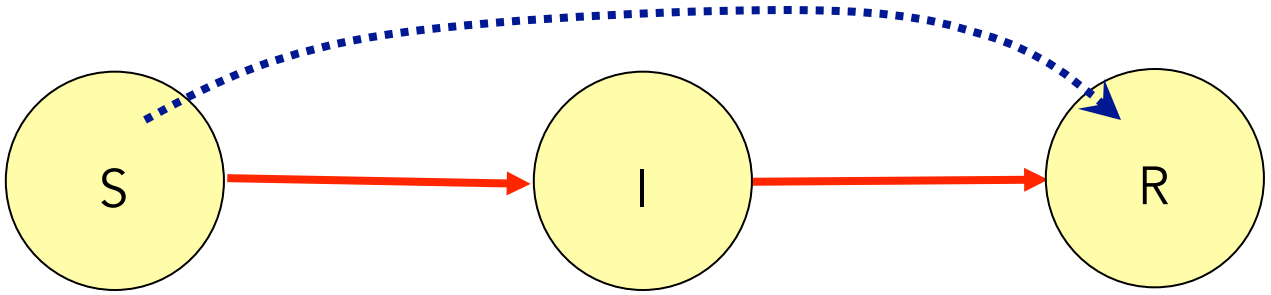
Why does pathogen persist?

- Birth of new hosts
- Immigration of new hosts
- Loss of immunity
- Evolution of pathogen
- Reintroduction of pathogen

Vaccinations

Vaccines:

Vaccines convert S to R



- protects immunized person
- reduces Pr(epidemic): herd immunity
- What proportion, p, of population do we need to immunize to prevent epidemic if infected individual enters our community?

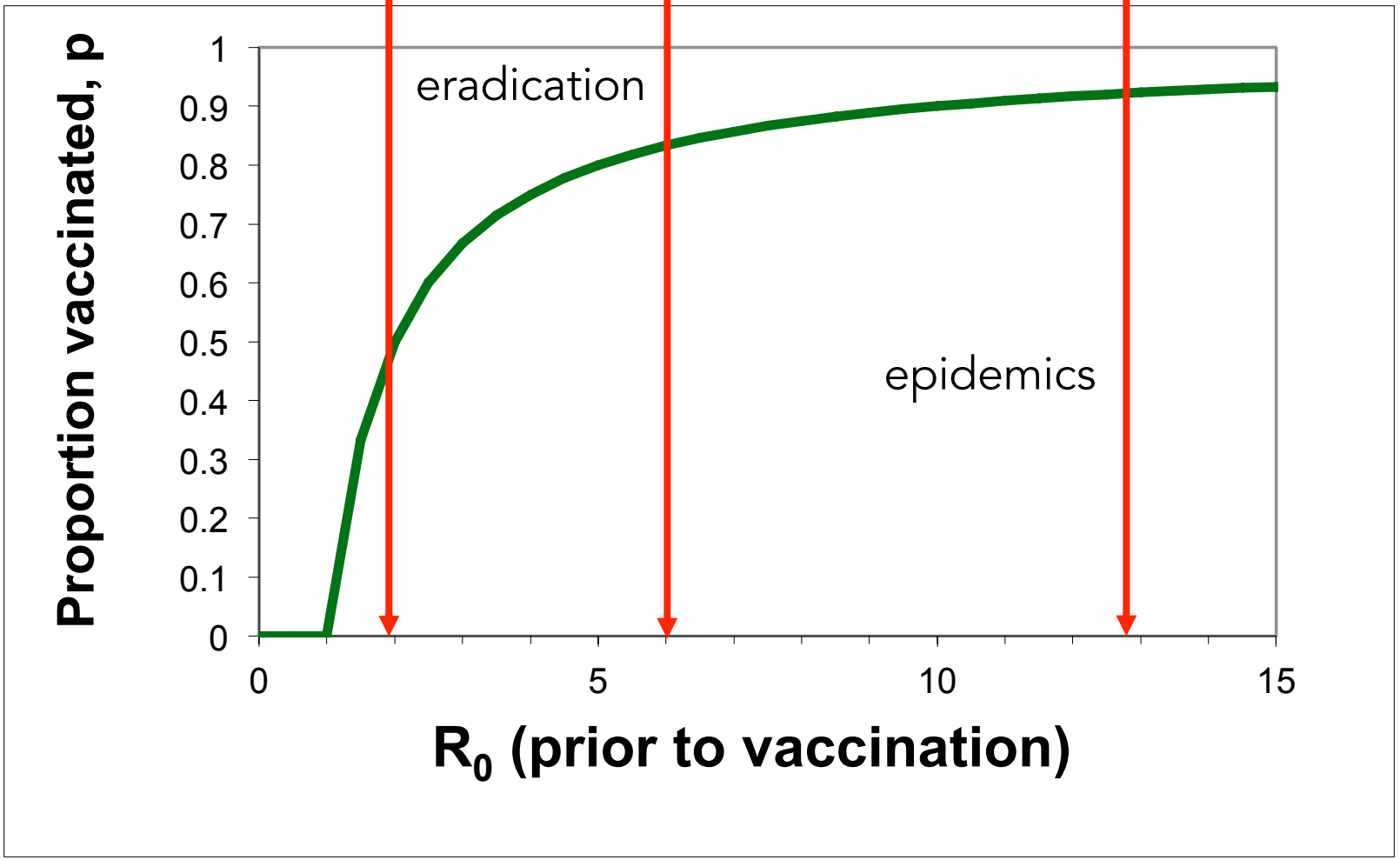
Vaccines:

Prior to vaccine: $R_0 = \beta N / \gamma > 1$

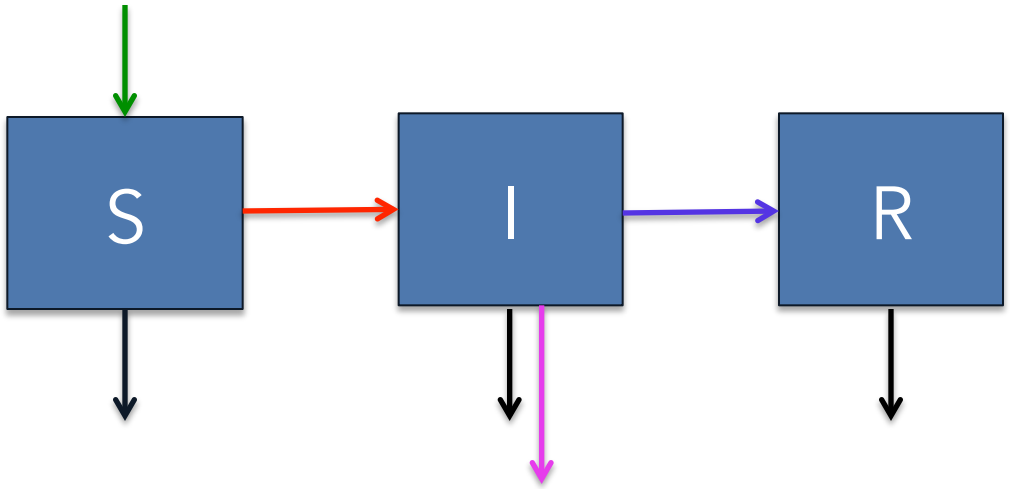
After vaccine: $R_0 = \beta N(1-p) / \gamma$

So to prevent epidemic: $p > 1-(1/R_0)$

Vaccines:


Measles
Rubella
Smallpox
eradication
epidemics
Proportion vaccinated, p
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
5
10
15
R0 (prior to vaccination)

Adding in host demography



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

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I - \alpha I \longrightarrow \beta SI - \gamma I - \mu I - \alpha I > 0$$

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

Epidemic criterion:

$$R_0 = \beta N / (\gamma + \mu + \alpha) > 1$$

STDs?

Transmission?

Transmission (per infected): $\beta SI/I = \beta S$

Contact: cN

Pr(contact is with susceptible): S/N

Pr(infection|contact with S): a

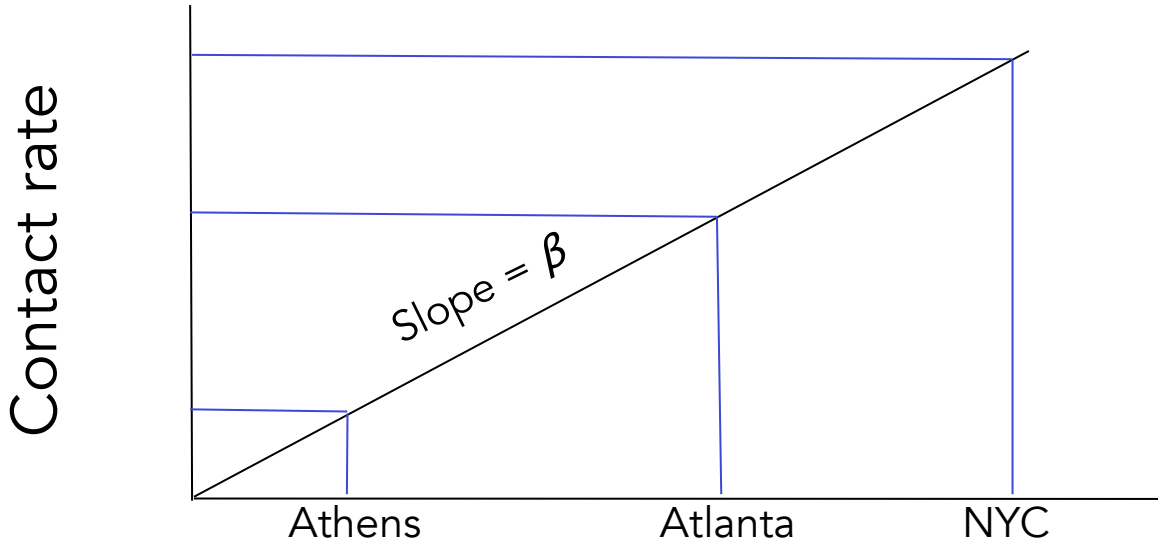
Thus, transmission = acS,

and

β = ca

Contact rate (cN)

(e.g., how many people do you bump into on a sidewalk?)

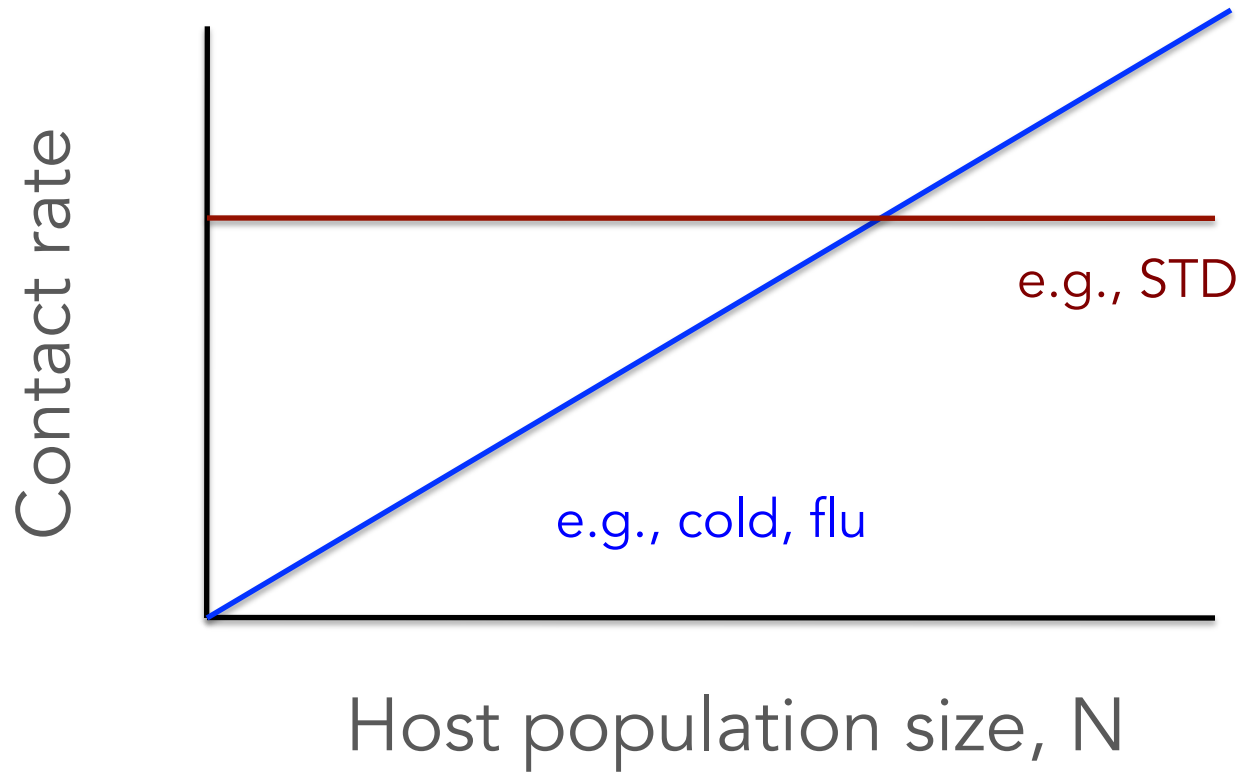


Is this a good model for all pathogens (e.g., STDs)?

An alternative?

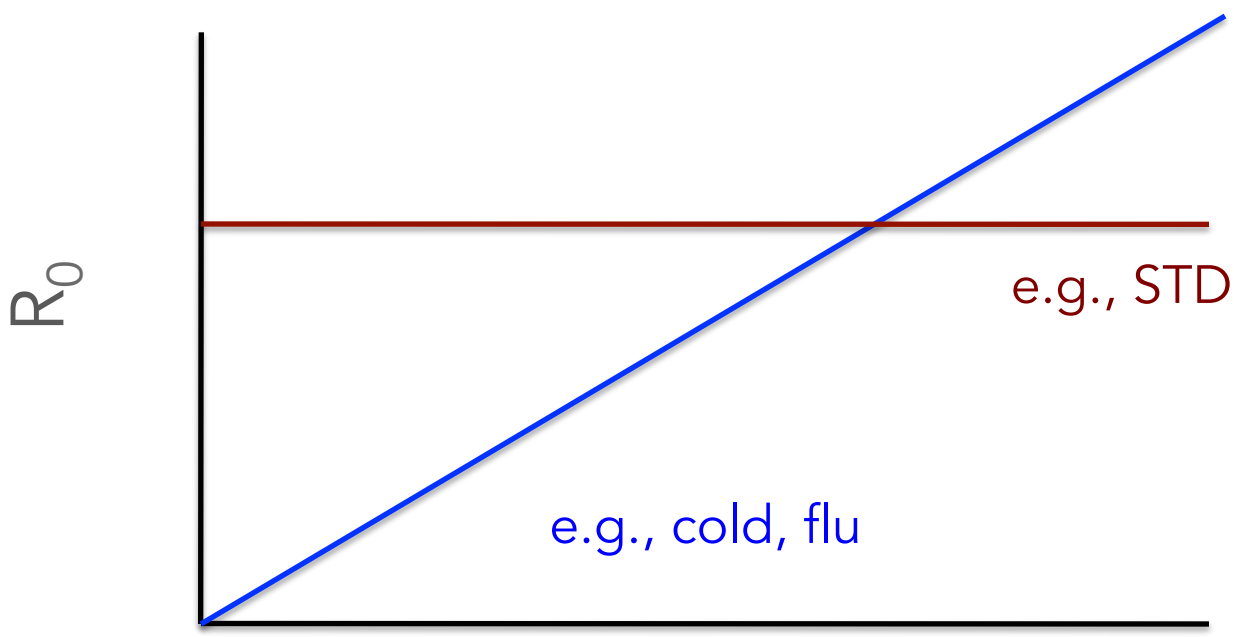
Density-dependent transmission

Frequency-dependent transmission



Density-dependent transmission

Frequency-dependent transmission

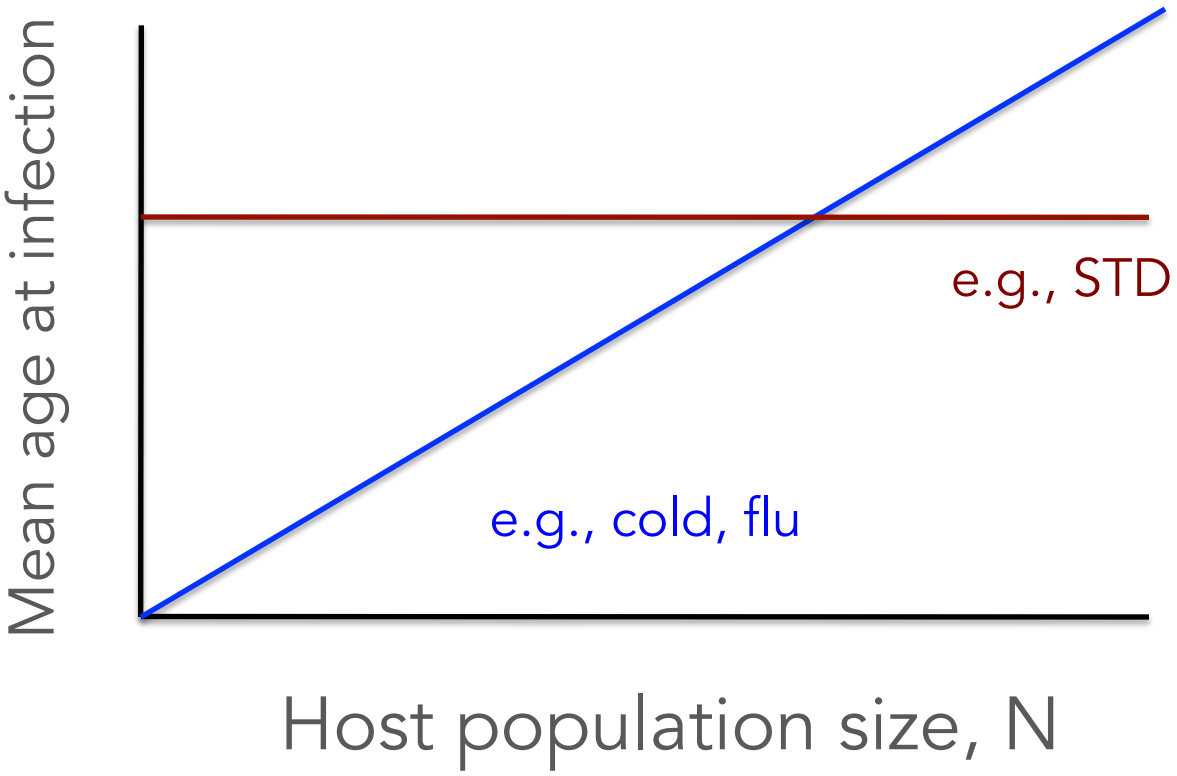


Thus, the existence of threshold populations depends on the type of transmission

With frequency-dependent transmission there is no threshold population size!

Density-dependent transmission

Frequency-dependent transmission



R_0 & age at infection

It is possible to derive that if $R_0>1$ and there is a supply of susceptibles, then the equilibrium proportion of susceptibles is $S/N=1/R_0$

Suppose the average age at infection is A

Individuals < A are susceptible

Individuals > A are immune

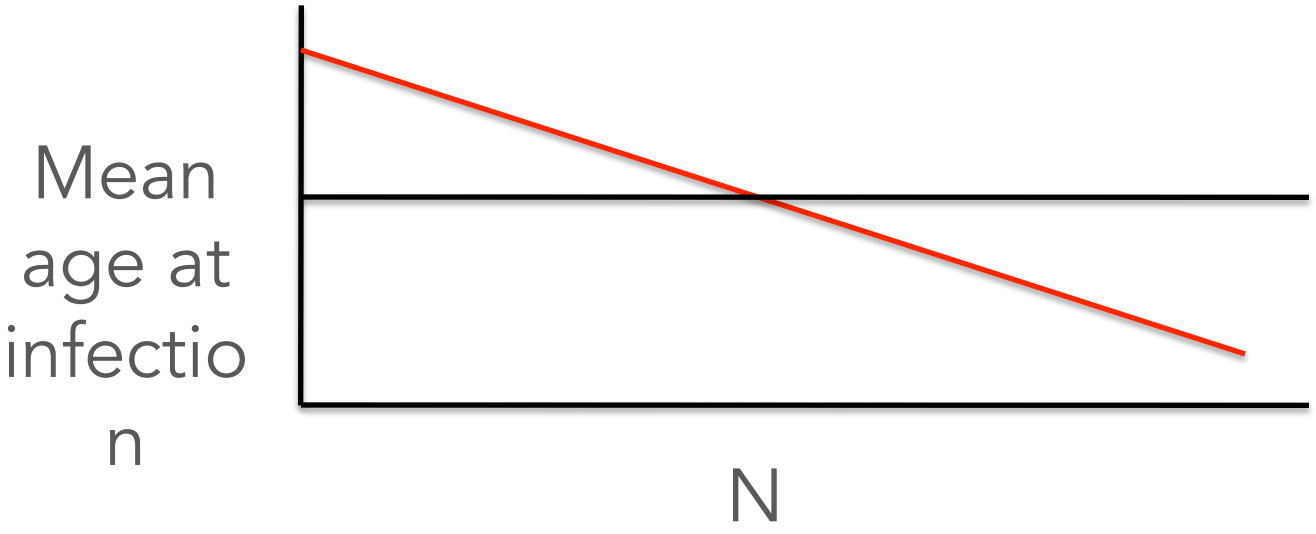
For simplicity assume a rectangular age distribution up to life expectancy L

Then the proportion susceptible = A/L

This means $R_0=L/A$

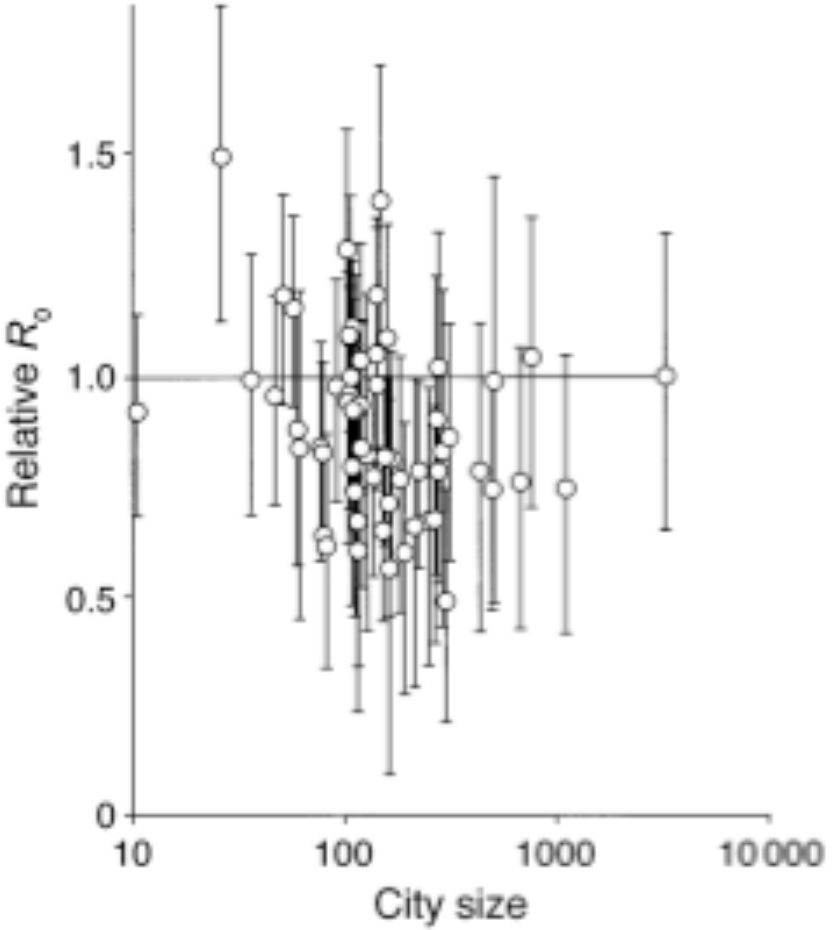
Predictions based on transmission

Density- and frequency-dependent transmission



More surprises

Measles in UK



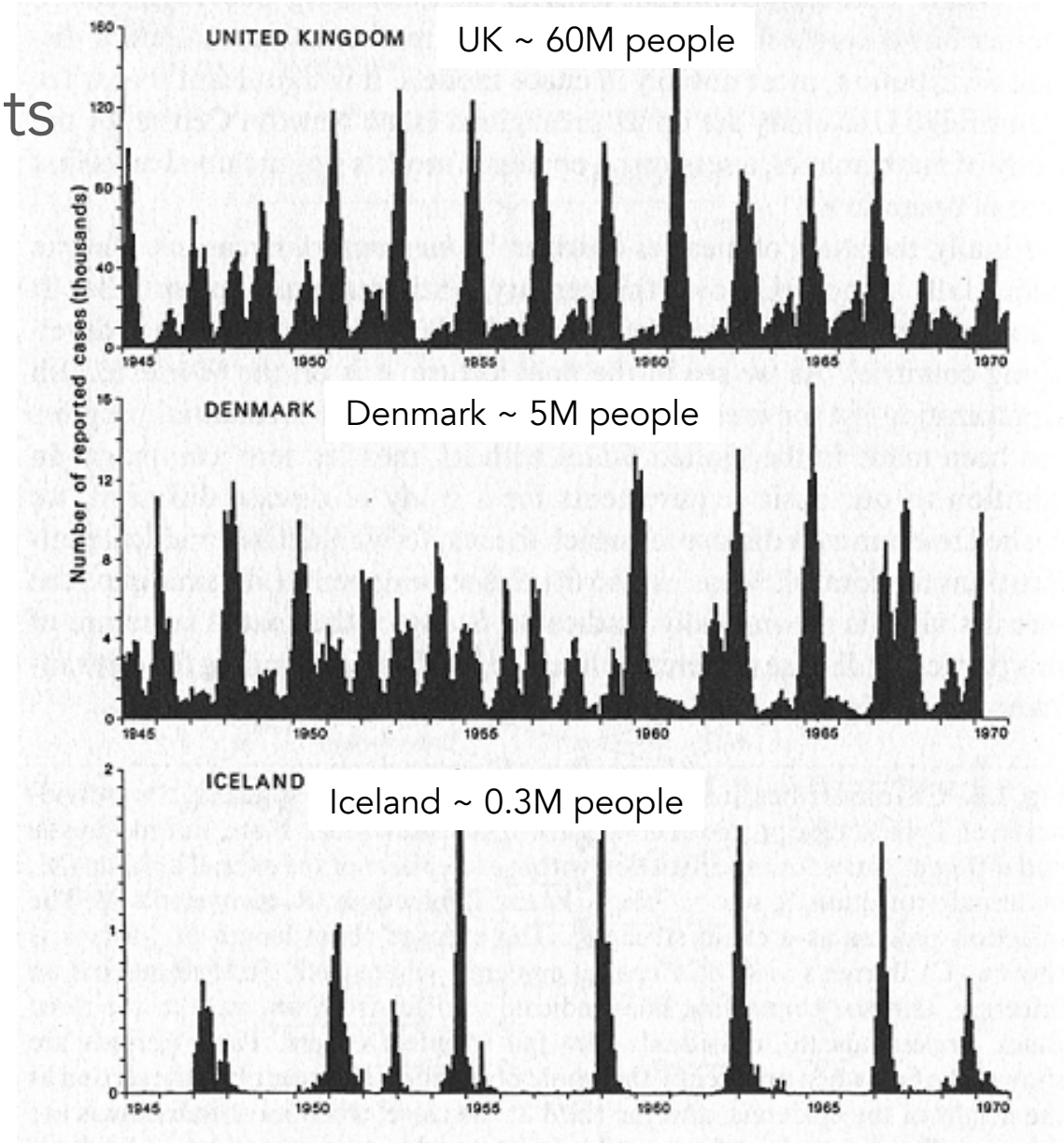
- suggests frequency-dependent transmission
- why?

Bjørnstad et al. 2002, Ecol. Monographs

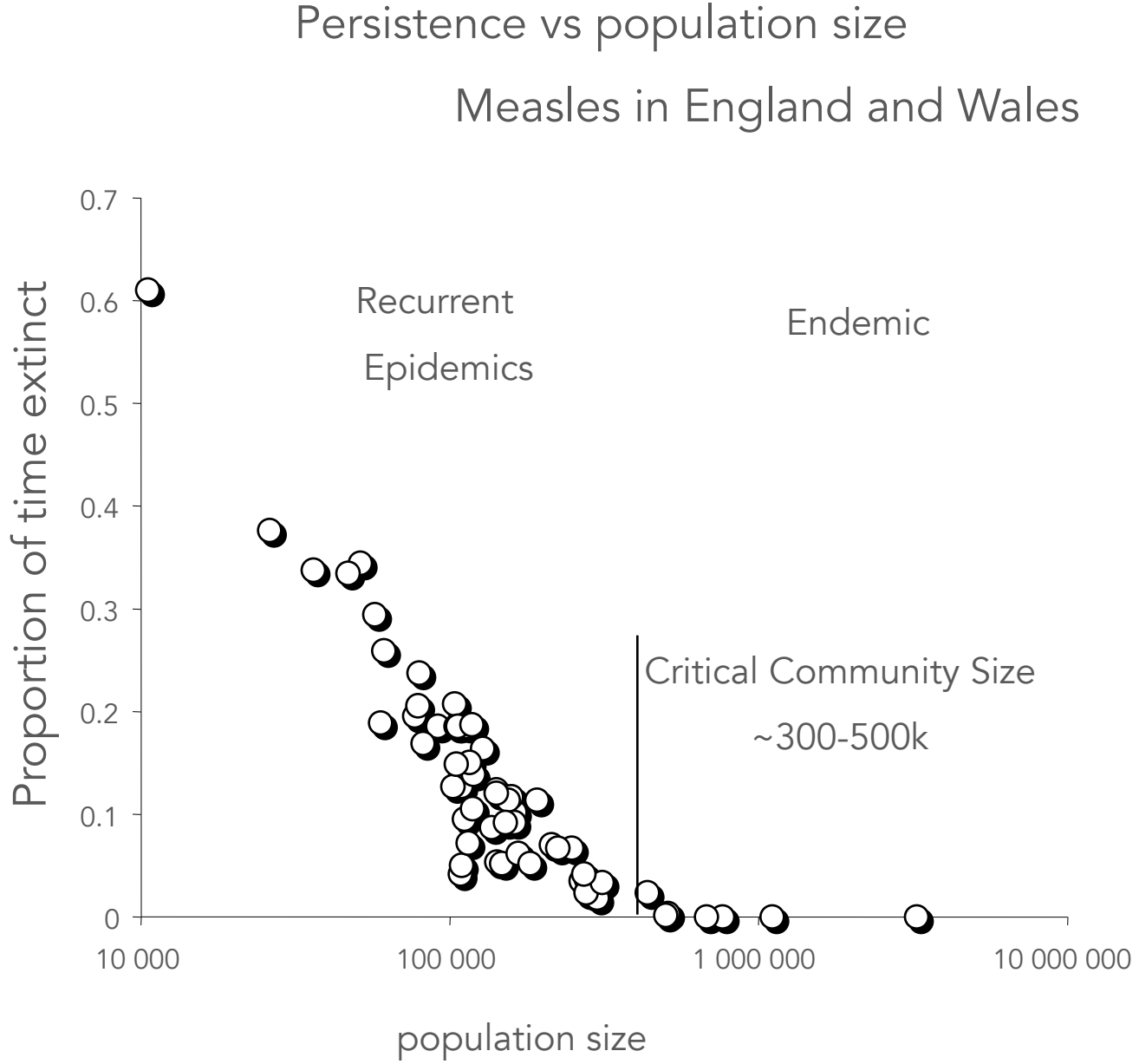
But isn't there a critical community size?

Epidemic fade-outs

e.g. Measles



Cliff et al. 1981


Persistence vs population size
Measles in England and Wales
Proportion of time extinct
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
Recurrent
Epidemics
Endemic
Critical Community Size
~300-500k
10 000
100 000
1 000 000
10 000 000
population size

Issues we haven't addressed:

- SEIR models (exposed/latent period)
- Vector-borne diseases
- Age structure
- Spatial structure