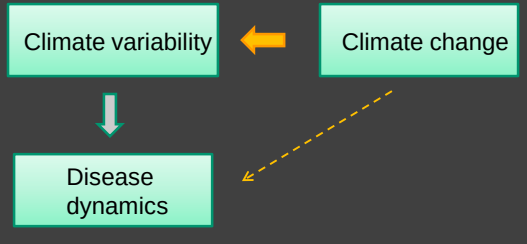
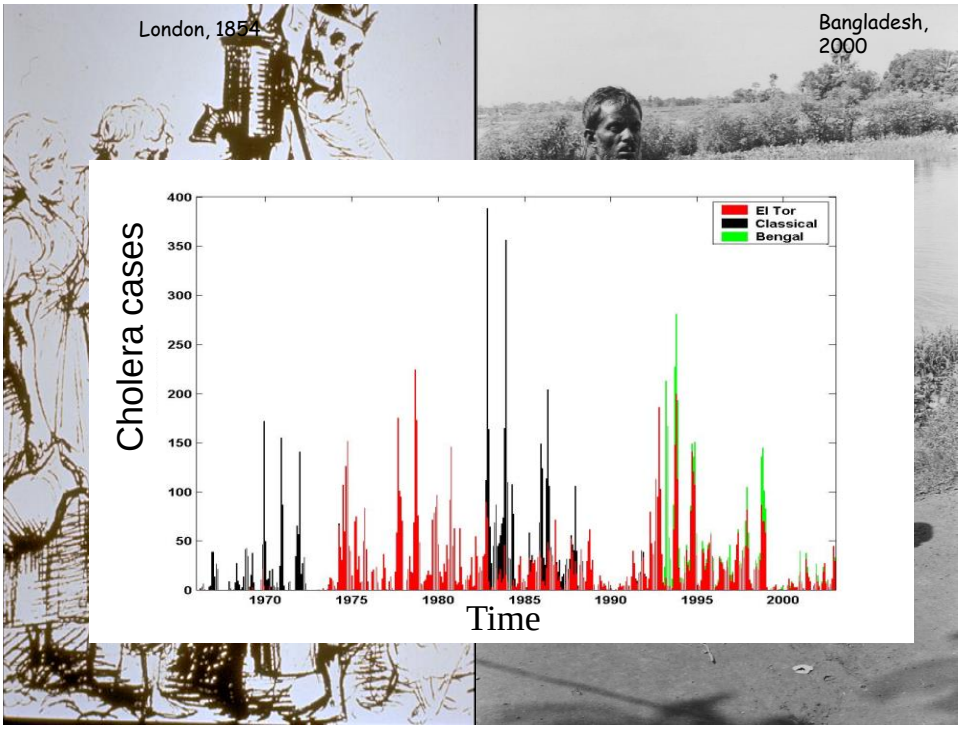
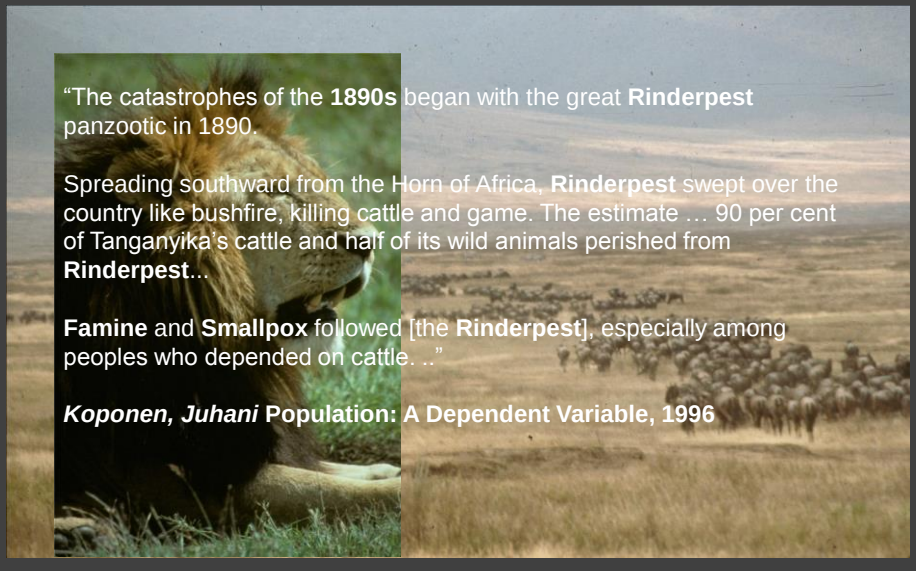
Introduction and Microparasitic models (the basics)

Mercedes Pascual





Population regulation: examples in the wild



"The catastrophes of the **1890s** began with the great **Rinderpest** panzootic in 1890.

Spreading southward from the Horn of Africa, **Rinderpest** swept over the country like bushfire, killing cattle and game. The estimate … 90 per cent of Tanganyika's cattle and half of its wild animals perished from **Rinderpest**...

Famine and **Smallpox** followed [the **Rinderpest**], especially among peoples who depended on cattle. .."

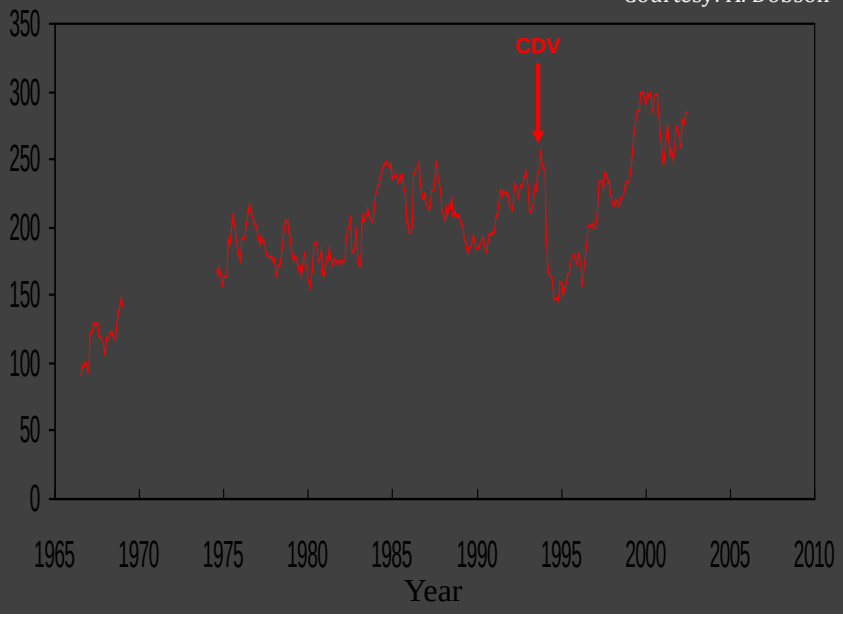
***Koponen, Juhani* Population: A Dependent Variable, 1996**

Widespread rinderpest vaccination of cattle

Although no wildebeest were vaccinated, their numbers increased by x8.

Lion population: Serengeti study area

Courtesy: A. Dobson



Predators versus Pathogens

PREDATORS	PATHOGENS
• Usually same size as victim.	• Much smaller than victims
• Similar, or slower, rate of population increase	• Much faster rate of population increase
• Tend to satiate	• Insatiable, unless vectored, or STD's

From population regulation to ecosystem regulation

OPEN ACCESS Freely available online

PLOS BIOLOGY

A Disease-Mediated Trophic Cascade in the Serengeti and its Implications for Ecosystem C

Ricardo M. Holdo[1]*, Anthony R. E. Sinclair[2], Andrew P. Dobson[3], Kristine L. Metzger[2], Benjamin M. Bolker[1], Mark E. Ritchie[4], Robert D. Holt[1]

1 Department of Biology, University of Florida, Gainesville, Florida, United States of America, 2 Department of Zoology, University of British Columbia, Vancouver, British Columbia, Canada, 3 Department of Ecology and Evolutionary Biology, Princeton University, Princeton, New Jersey, United States of America, 4 Department of Biology, Syracuse University, Syracuse, New York, United States of America

Abstract

Tree cover is a fundamental structural characteristic and driver of ecosystem processes in terrestrial ecosystems, and trees are a major global carbon (C) sink. Fire and herbivores have been hypothesized to play dominant roles in regulating trees in African savannas, but the evidence for this is conflicting. Moving up a trophic scale, the factors that regulate fire occurrence and herbivores, such as disease and predation, are poorly understood for any given ecosystem. We used a Bayesian state-space model to show that the wildebeest population irruption that followed disease (rinderpest) eradication in the Serengeti ecosystem of East Africa led to a widespread reduction in the extent of fire and an ongoing recovery of the tree population. This supports the hypothesis that disease has played a key role in the regulation of this ecosystem. We then link our state-space model with theoretical and empirical results quantifying the effects of grazing and fire on soil carbon to predict that this cascade may have led to important shifts in the size of pools of C stored in soil and biomass. Our results

Biodiversity and disease

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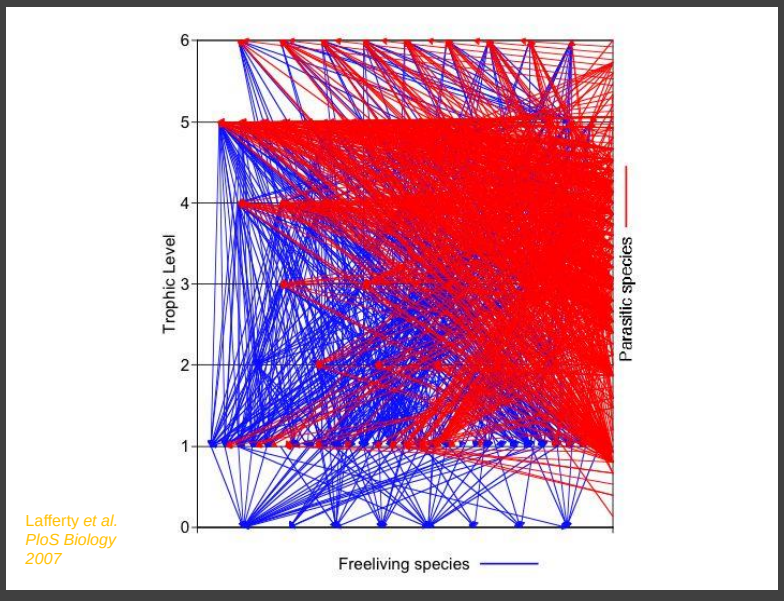
PLOS MEDICINE

Essay

Sacred Cows and Sympathetic Squirrels: The Importance of Biological Diversity to Human Health

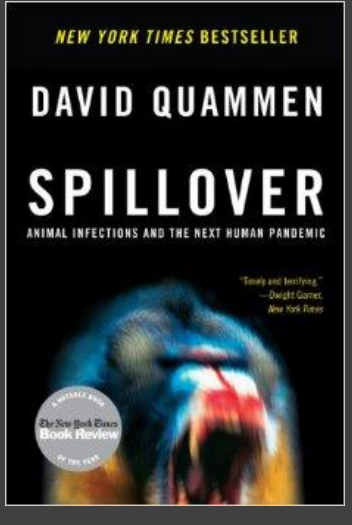
Andy Dobson*, Isabella Cattadori, Robert D. Holt, Richard S. Ostfeld, Felicia Keesing, Kristle Krichbaum, Jason R. Rohr, Sarah E. Perkins, Peter J. Hudson

Why are cows sacred? Travel anywhere in India and they have the right of way. Travel anywhere in the eastern United States and you'll see squirrels, more than likely, as roadkill. Yet both species serve a similar epidemiological function: they receive bites from infected vectors that might otherwise have bitten humans, and they break the chain of pathogen transmission. In the case of Indian cattle, the bites are from mosquitoes infected with malaria parasites; the squirrels, on the other hand, receive



Lafferty *et al. PloS Biology 2007*

Pathogen emergence



This course/workshop is a bit like a marathon …

- The basic models
- Variations of these models for different kinds of infectious diseases, and to incorporate structure in the host population (e.g. age, space) or in the community of hosts (e.g. species diversity)
- Different kinds of epidemiological models (deterministic, stochastic, individual-based)
- Sources of climate data and climate-based predictions
- Different kinds of model applications, especially at the interface with environmental forcing

- Current research in the field

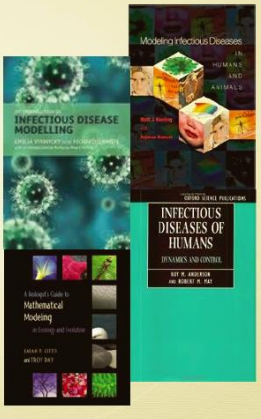
- Parameter inference for process-based models

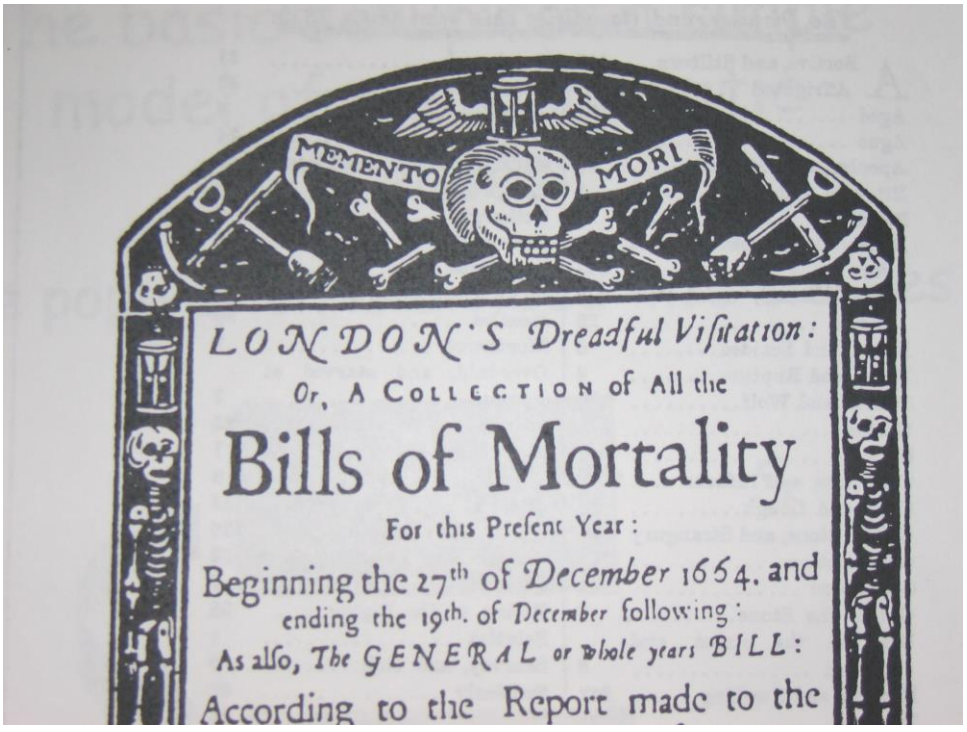
- Group projects

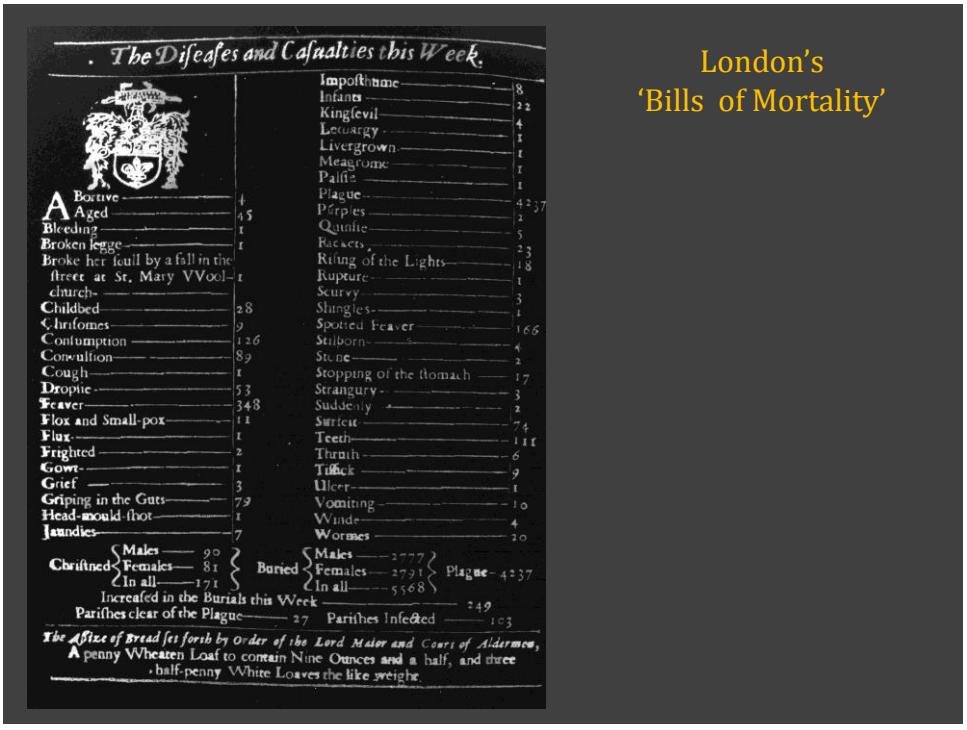
all in two weeks!!!!

Resources:

- Keeling & Rohani (2008)
- Vynnycky & White (2010)
- Anderson & May (1991)
- Otto & Day (2007)



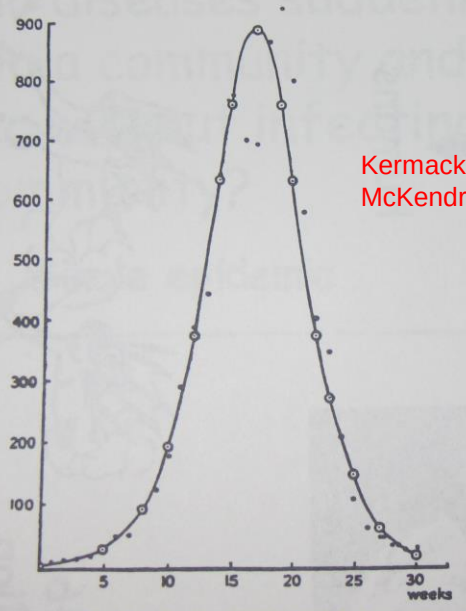




London's
'Bills of Mortality'

form in which many statistics are given

$$\frac{dz}{dt} = \frac{i^3}{2z_0 x^2}\sqrt{-q}\,\operatorname{sech}^2\left(\frac{\sqrt{-q}}{2}lt - \phi\right). \qquad \text{(thirty-one)}$$

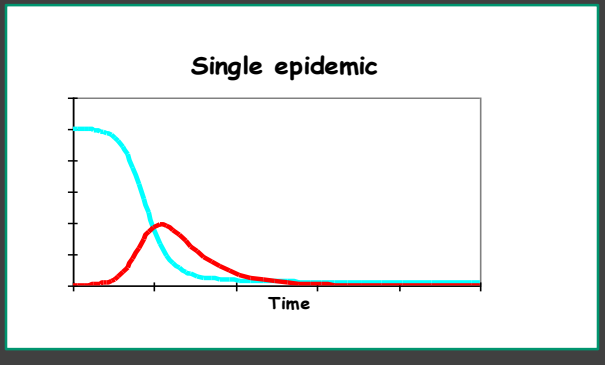


Kermack and McKendrick, 1927

The accompanying chart is based upon figures of deaths from plague in the island of Bombay over the period December 17, 1905, to July 21, 1906. The ordinate represents

Some key questions:

❖ Why do diseases suddenly develop in a community and then disappear without infecting the entire community?

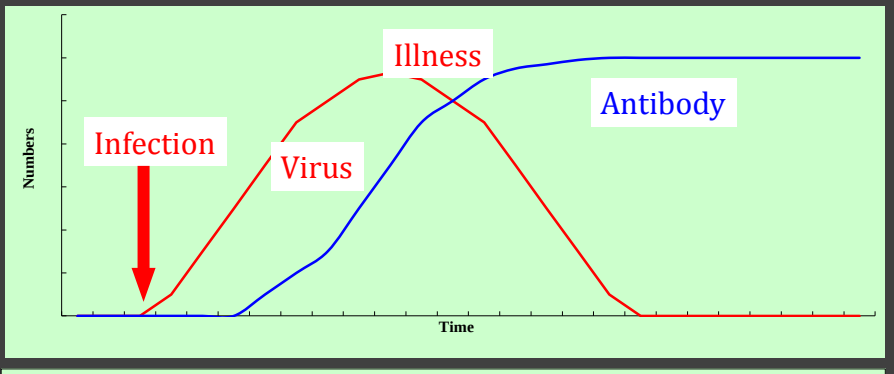


❖ Once a pathogen invades a population, what determines whether an epidemic will take place?

Main conceptual points:

- The idea of a population threshold for epidemic behavior
- The concept of R_0: the basic reproductive number of a disease
- The concept of herd immunity
- Why do we observe recurrent epidemics (i.e. cycles)?

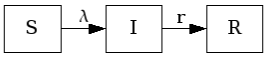
Basic variables: microparasitic infections



Susceptible	Infectious	Recovered
S	I	R
Years	Days	Many years

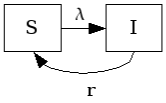
The population is divided into classes

SIR



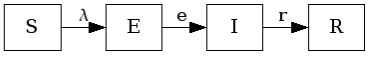
Infection confers lifelong immunity

SIS



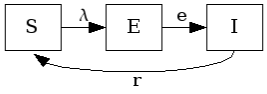
No immunity

SEIR

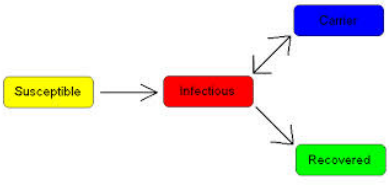


Latent class

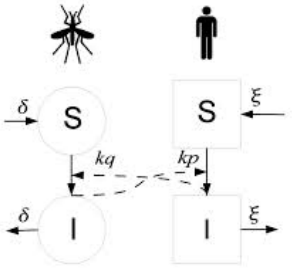
SEIS



Other variations of this theme…



Asymptomatic carriers that can transmit infection



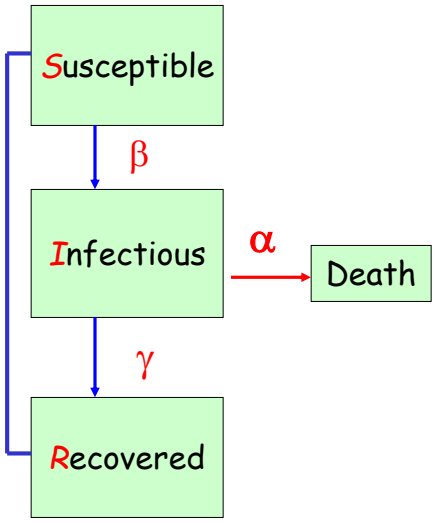
Vector-borne infection

SIR equations

$$\frac{dS}{dt} = \cdots$$

$$\frac{dI}{dt} = \cdots$$

$$\frac{dR}{dt} = \cdots$$



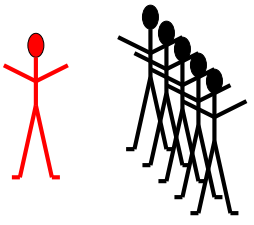
The transmission rate

Number of contacts / day

= contact rate κ

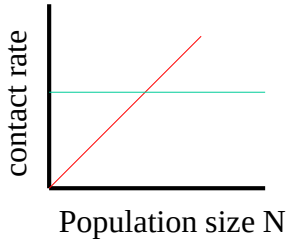
Probability of transmission given contact

= infectivity ν



Fraction of individuals that are susceptible

= S/N

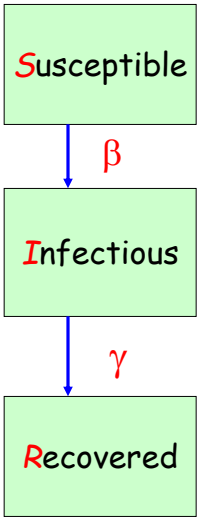


Density-dependent transmission

Frequency-dependent transmission

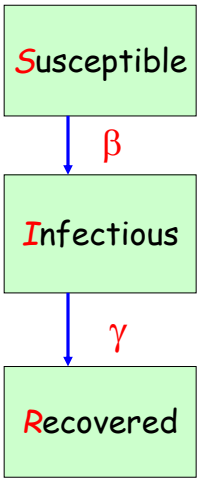
Transmission

$$\frac{dS}{dt} = -\beta S \frac{I}{N}$$
$$\frac{dI}{dt} = +\beta S \frac{I}{N} - \cdots$$
$$\frac{dR}{dt} = \cdots$$



add recovery

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = +\beta SI - \gamma I$$
$$\frac{dR}{dt} = +\gamma I$$



Mean recovery time – Mean infectious period

Consider recovery of a single infectious individual

$$I(t) = e^{-\gamma t}$$

$$1 = \int_o^\infty ce^{-\gamma t}dt \quad = \frac{c}{\gamma}$$

Hence, probability density function is $\gamma e^{-\gamma t}$

$$\tau = \int_0^\infty t\gamma e^{-\gamma t}dt \; = \frac{1}{\gamma}$$

For a random variable x, with probability density function f(x), the mean is given by $\int_0^\infty xf(x)dx$

Deriving the transmission rate

- Contact rate assumed to be κ per unit time
- For a susceptible, a fraction Υ/N of contacts is with infecteds
- In a small time interval, δt, the number of contacts with infecteds is $\kappa\,(\Upsilon/N)\,\delta t$
- If c is probability of transmission, given contact, then probability of escaping infection in δt is
  - $1-\delta q = (1-c)^{\kappa\,(\Upsilon/N)\,\delta t}$
- Define $\beta = -\kappa \log(1-c)$, then
  - $\delta q = 1 - e^{-\beta\Upsilon\delta t/N}$

    $\delta q = 1 - (1 - \beta\Upsilon\delta t/N + (\beta\Upsilon\delta t/N)^2 - (\beta\Upsilon\delta t/N)^3 + \ldots)$
  - $\delta q/\delta t = \beta\Upsilon/N \Rightarrow dq/dt = \beta\Upsilon/N$

$$e^x = 1 + x + \frac{x^2}{2!} + \ldots$$

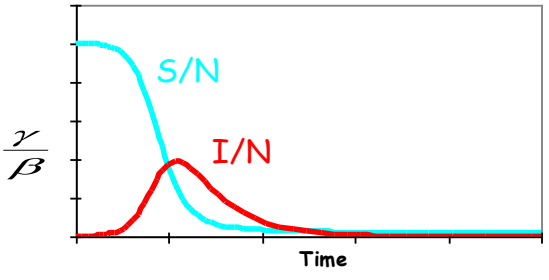
When does the number of infecteds increase?

$$\frac{dI}{dt} = (\beta \frac{S}{N} - \gamma)I$$

- When $\frac{S}{N} < \frac{\gamma}{\beta}$, the number of infected individuals decreases
- When $\frac{S}{N} > \frac{\gamma}{\beta}$, the number of infected individuals increases

➡ EPIDEMIC

But what is happening to S/N?



Concept of a population threshold

The famous R_0 or basic reproductive number

THOUGHT EXPERIMENT:
introduce a small number of infected individuals in a population and ask whether there will be an epidemic

We need

$$R_0 = \frac{\beta}{\gamma} > 1$$

A biological interpretation of R_0

$$\underbrace{\beta}_{\text{Number of susceptibles that come into contact with an infected}} . \underbrace{\frac{1}{\gamma}}_{\text{Average duration of infection}}$$

R_0 is the average number of secondary infections arising from an average primary infection (in an entirely susceptible population)

Density-dependent transmission

$$S_0 \cdot \beta \cdot \frac{1}{\gamma}$$

Number of susceptibles that come into contact with an infected

Average duration of infection

Estimates of Ro

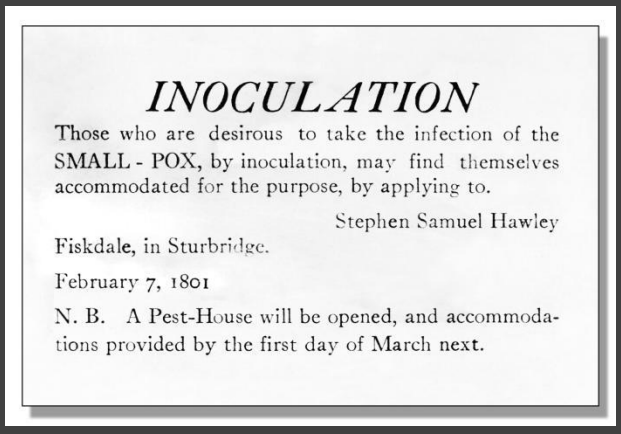
Disease	Host	Estimated R_0	Reference
FIV	domestic cats	1.1-1.5	Smith (2001)
Rabies	dogs (Kenya)	2.44	Kitala *et al.* (2002)
Phocine Distemper	seals	2 - 3	Swinton *et al.* (1998)
Tuberculosis	cattle	2.6	Goodchild & Clifton-Hadley (2001)
Influenza	human	3 - 4	Murray (1989)
Foot and Mouth Disease	livestock farms (UK)	3.5 - 4.5	Ferguson *et al.* (2001b)
Smallpox	human	3.5 - 6	Gani & Leach (2001)
Rubella	human (UK)	6 - 7	Anderson & May (1991)
Chickenpox	human (UK)	10 - 12	Anderson & May (1991)
Measles	human (UK)	16 - 18	Anderson & May (1982)
Whooping Cough	human (UK)	16 - 18	Anderson & May (1982)

Keeling & Rohani (2008)

Other estimates of interest:

- HIV: among homosexual men in UK ~ 4
- HIV: among female sex workers in Kenya ~ 11
- 2003 SARS epidemic: ~ 3
- 1918 Influenza pandemic: ~ 1.5-3
- 2009 Swine 'flu epidemic: ~ 1.2-1.5

How many people should we vaccinate?



INOCULATION

Those who are desirous to take the infection of the SMALL - POX, by inoculation, may find themselves accommodated for the purpose, by applying to.

Stephen Samuel Hawley

Fiskdale, in Sturbridge.

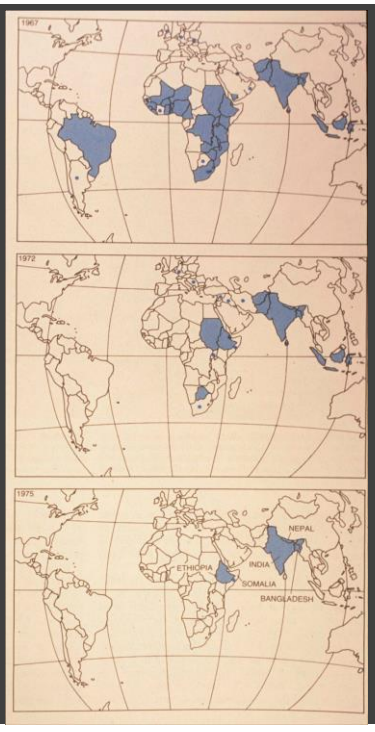
February 7, 1801

N. B. A Pest-House will be opened, and accommodations provided by the first day of March next.

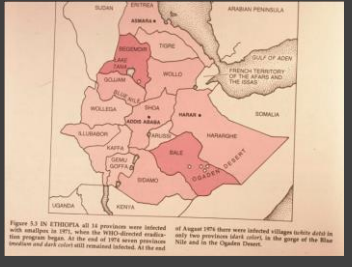
1967

1972

1975



Poster campaign



The final few cases

The basic idea behind vaccination:

Reduce R_0 below 1 by reducing the effective number of susceptibles.

Proportion p of the population that needs to be vaccinated

$$p > 1 - \frac{1}{R_0}$$

Concept of herd immunity

Results with SIR model

- Epidemic occurs if and only if $R_0 > 1$
- Single epidemic, then disease disappears

- Cannot explain diseases that persist
- Cannot explain recurrent cycles of epidemics