Basic compartmental models of mathematical epidemiology

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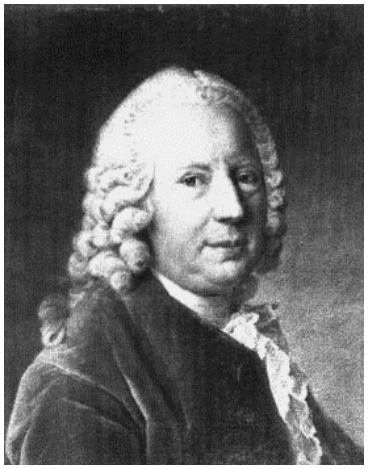
Szeged Summer School

Why do we need to do modelling?

- to have an understanding of the behavior of disease spread
- to make sense of data
- to forecast future behavior, estimate the severity of an epidemic
- to inform future collection of data: what type of data needed, when and where to collect to obtain information required for predictions
- to assess and compare potential intervention strategies
- to optimize control measures
- to estimate key parameters
- to run „experiments” (simulations)
- by qualitative analysis to provide information about all possible scenarios

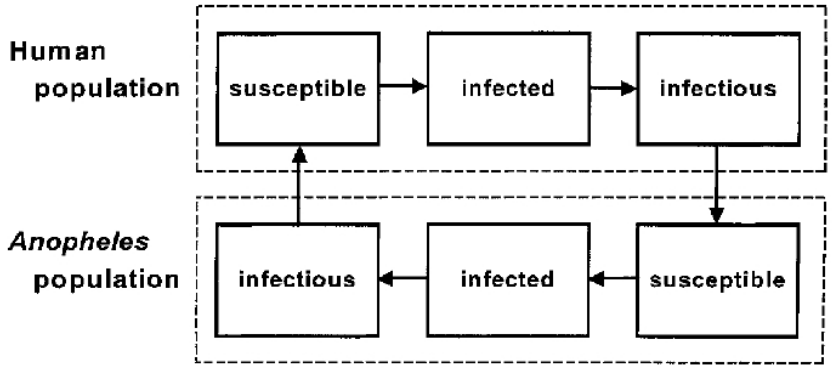


I simply wish that, in a matter which so closely concerns the well-being of the human race, no decision shall be made without all knowledge which a little analysis and calculation can provide.

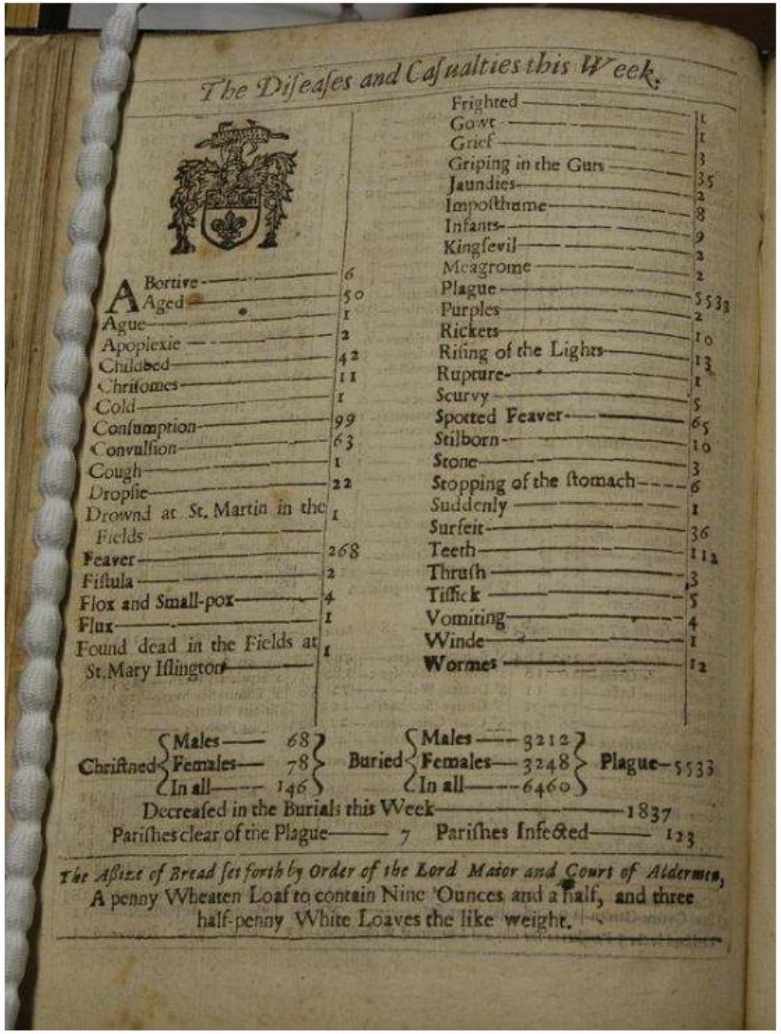
Daniel Bernoulli, 1760, on smallpox inoculation

As a matter of fact all epidemiology, concerned as it is with variation of disease from time to time or from place to place, *must* be considered mathematically (...) and the mathematical method of treatment is really nothing but the application of careful reasoning to the problems at hand.

Sir Ronald Ross, 1911, *The Prevention of Malaria*



London 1665-1666

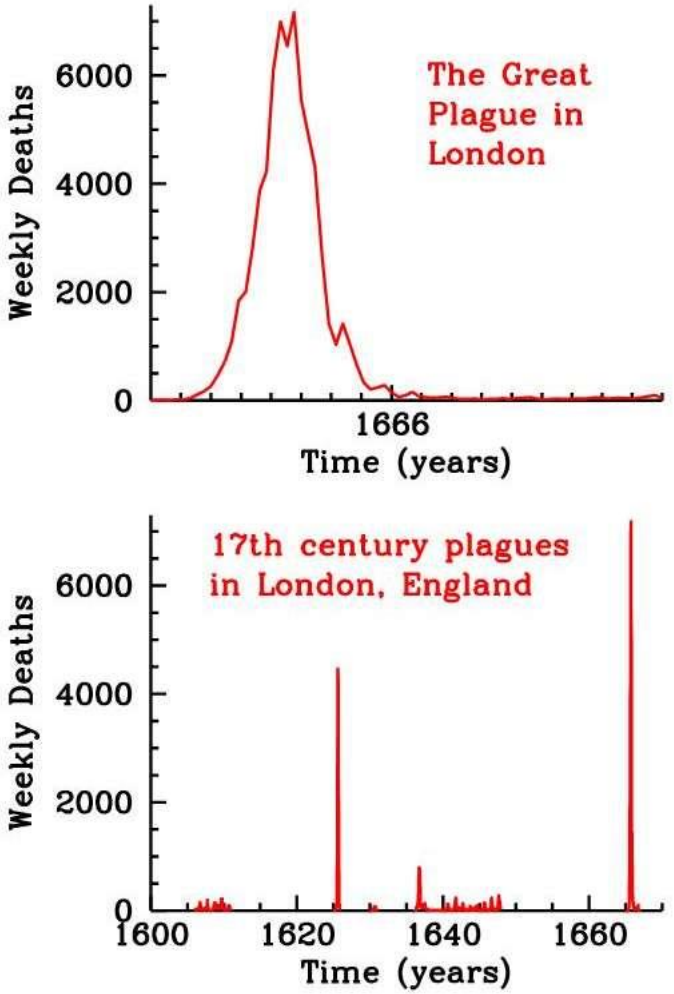


The Diſeaſes and Caſualties this Week.

Abortive	6	Frighted	1
Aged	50	Gowt	1
Ague	1	Grief	3
Apoplexie	2	Griping in the Guts	35
Childbed	42	Jaundies	2
Chriſomes	11	Impoſthume	8
Cold	1	Infants	9
Conſumption	99	Kingſevil	2
Convulſion	63	Meagrome	2
Cough	1	Plague	5533
Dropſie	22	Purples	2
Drowned at St. Martin in the Fields	1	Rickets	10
Feaver	268	Riſing of the Lights	13
Fiſtula	2	Rupture	1
Flox and Small-pox	4	Scurvy	5
Flux	1	Spotted Feaver	65
Found dead in the Fields at St. Mary Iſlington	1	Stilborn	10
		Stone	3
		Stopping of the ſtomach	6
		Suddenly	1
		Surfeit	36
		Teeth	112
		Thruſh	3
		Tiſſick	5
		Vomiting	4
		Winde	1
		Wormes	12

Chriſtned: Males 68, Females 78, In all 146
Buried: Males 3212, Females 3248, In all 6460
Plague 5533
Decreaſed in the Burials this Week 1837
Pariſhes clear of the Plague 7 Pariſhes Infected 123

The Aſſize of Bread ſet forth by Order of the Lord Maior and Court of Aldermen,
A penny Wheaten Loaf to contain Nine Ounces and a half, and three half-penny White Loaves the like weight.

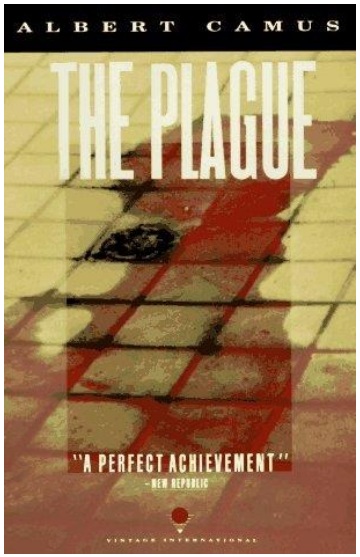


in: D Earn, A Light Introduction to Modelling Recurrent Epidemics, Lecture Notes in Mathematical Epidemiology, 2009

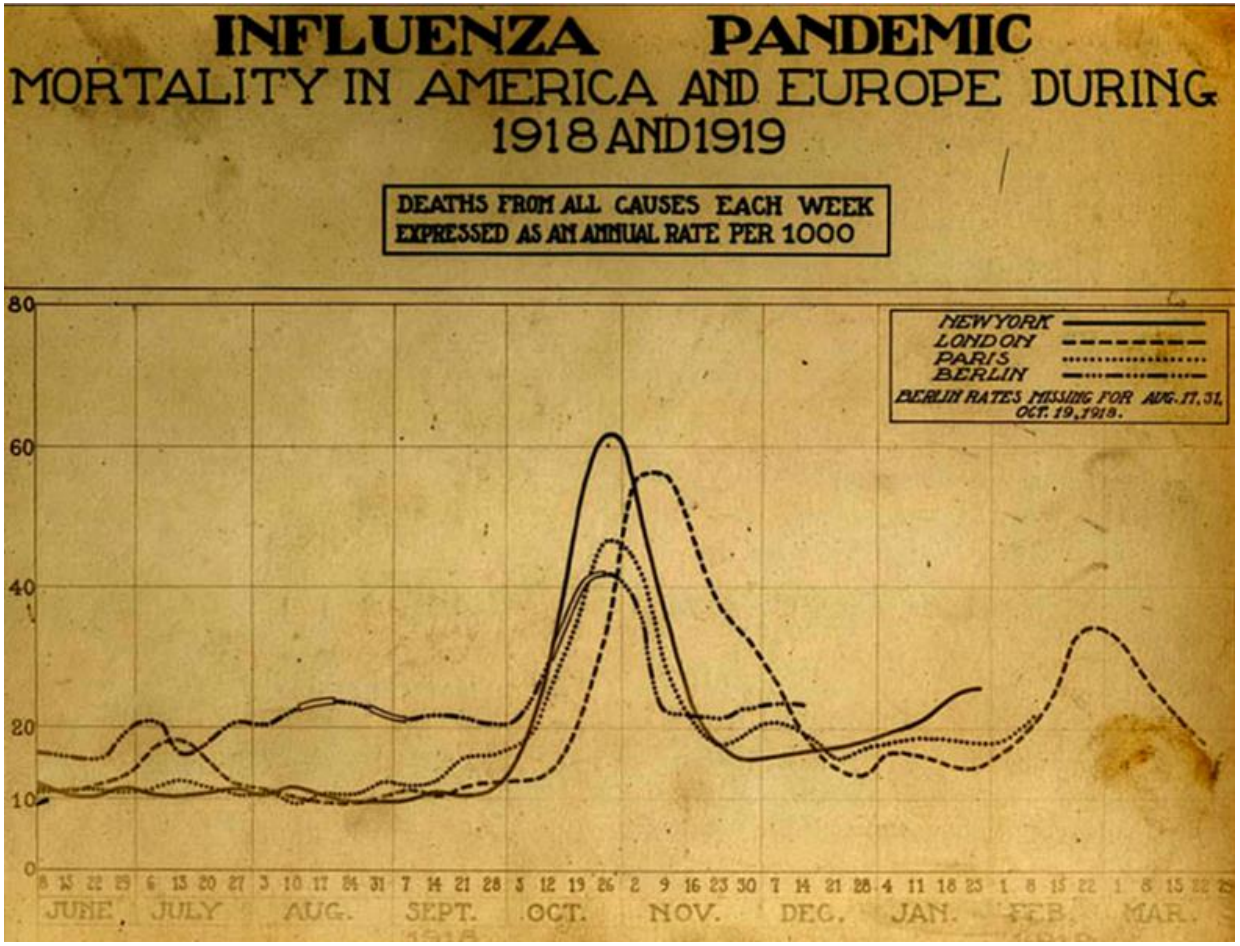
More than 15% of the population of London died in the Great Plague. It appeared quite suddenly, grew in intensity, and then disappeared, leaving part of the population untouched. Why? We will understand by the end of this lecture (I hope ☺)

Our strategy had not changed, but whereas yesterday it had obviously failed, today it seemed triumphant. Indeed, one's chief impression was that the epidemic had called a retreat after reaching all its objectives; it had, so to speak, achieved its purpose.

Albert Camus, The Plague (La Peste, 1948)



Why are these mortality curves are so similar in distant cities?



First serious mathematical treatment of a problem in epidemiology
1766 Daniel Bernoulli (1700-1782)

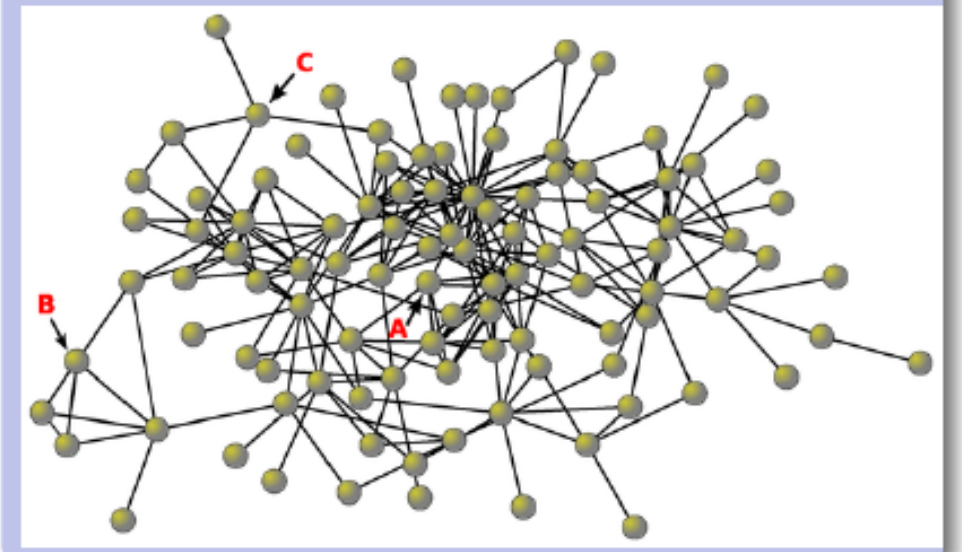
Smallpox: very high mortality rate
Variolation (inoculation): dangerous practice of immunization, heated debates, hundreds of articles on pros and cons

Bernoulli's question: how life expectancies would change if we could completely eliminate smallpox by inoculation? The gain in life expectancy after elimination of this cause of death can be explicitly expressed in terms of the case fatality and the endemic prevalence of susceptibles.

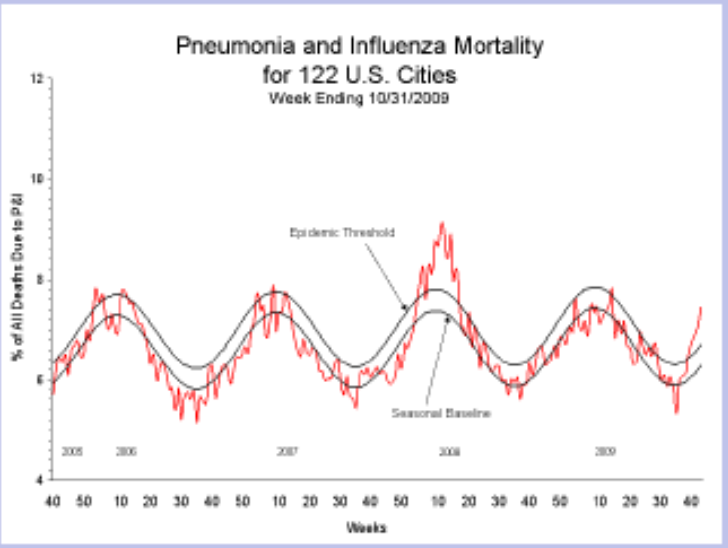
Related to financial mathematics, as in that time life annuities were sold.

Modelling approaches for infectious diseases

Agent-based (network) models



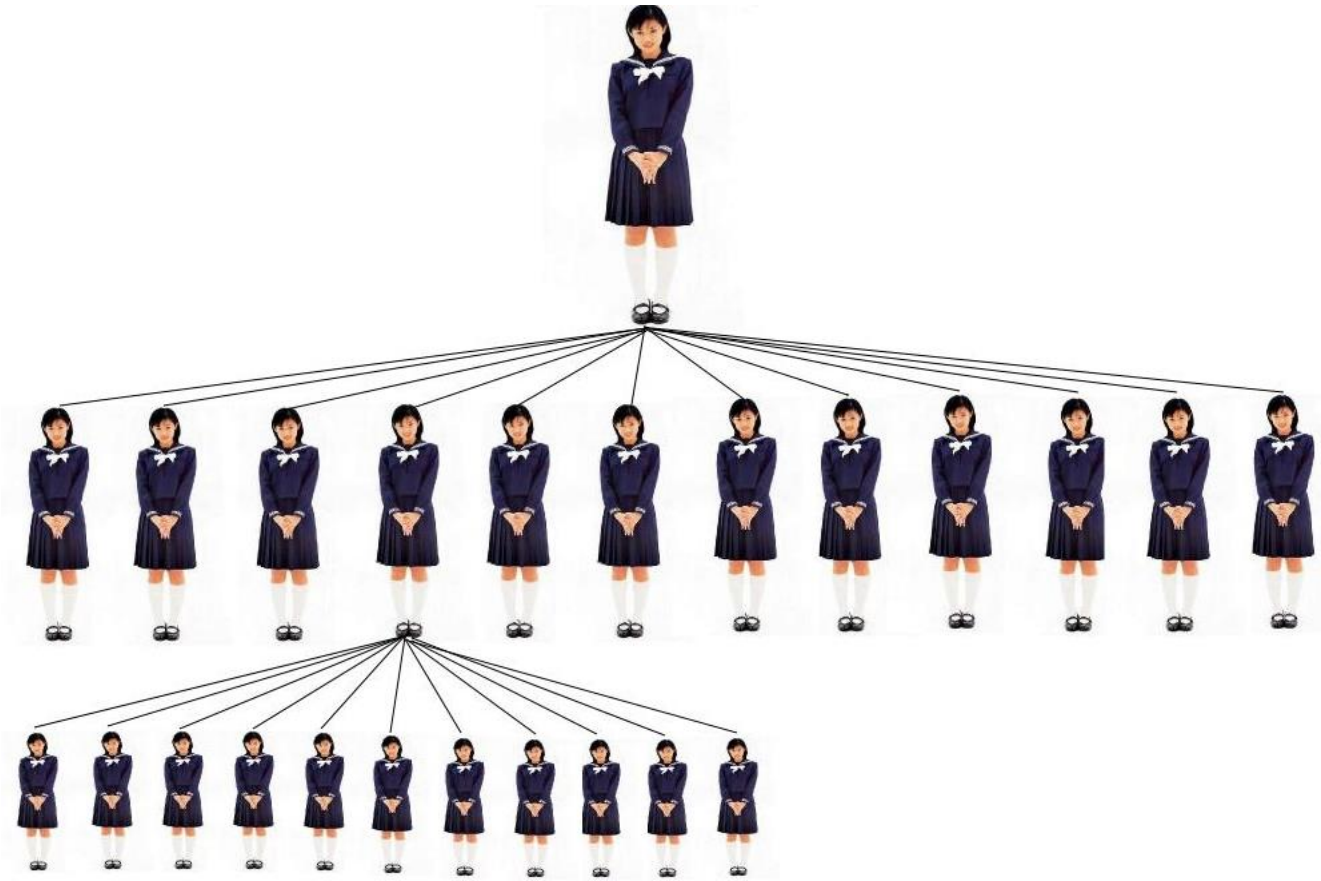
Stochastic models



Deterministic compartmental models

Basic idea: Population is divided into disjoint classes according to key characteristics. Then we describe the movement of individuals from one class to another by differential equations.

Let’s start with an incredibly naive approach



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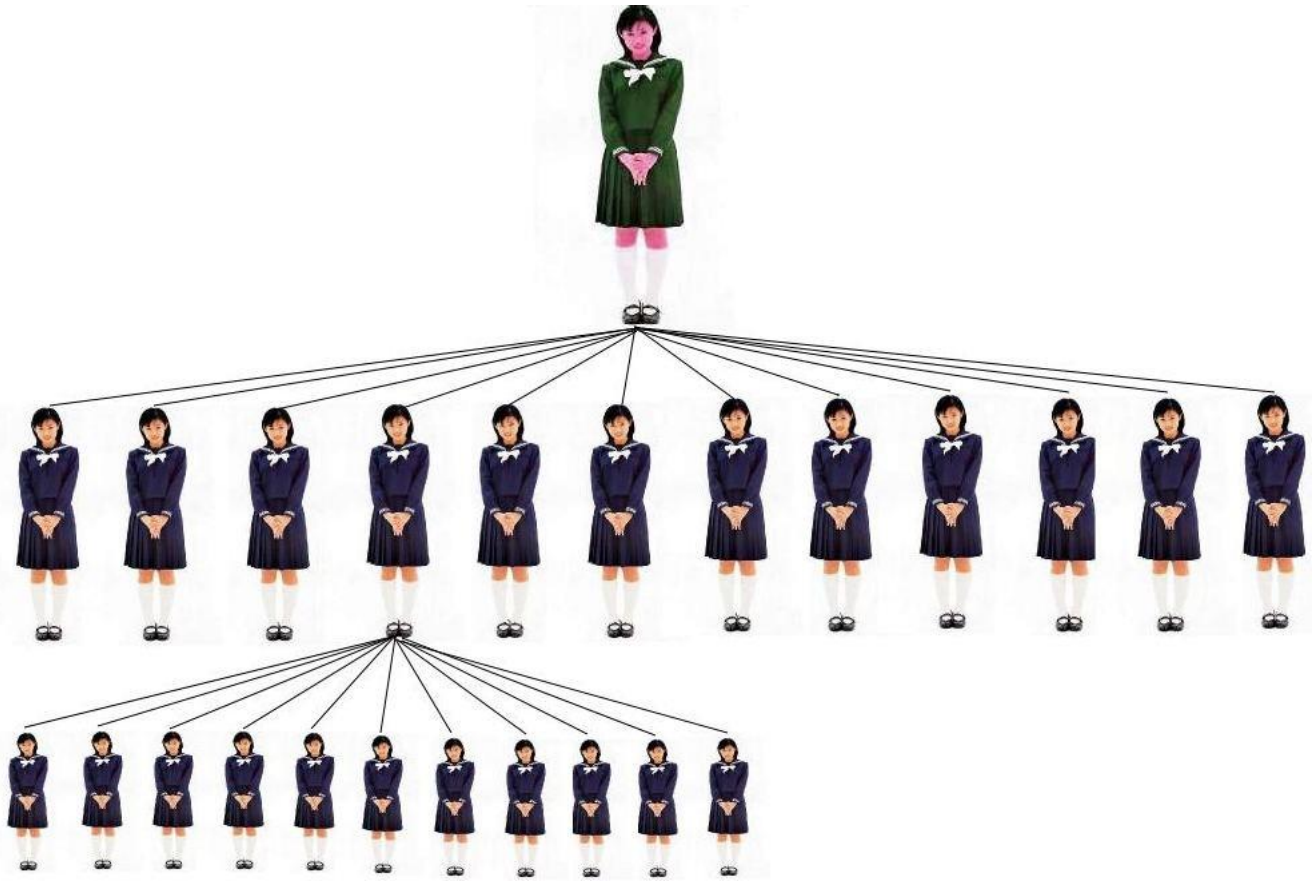

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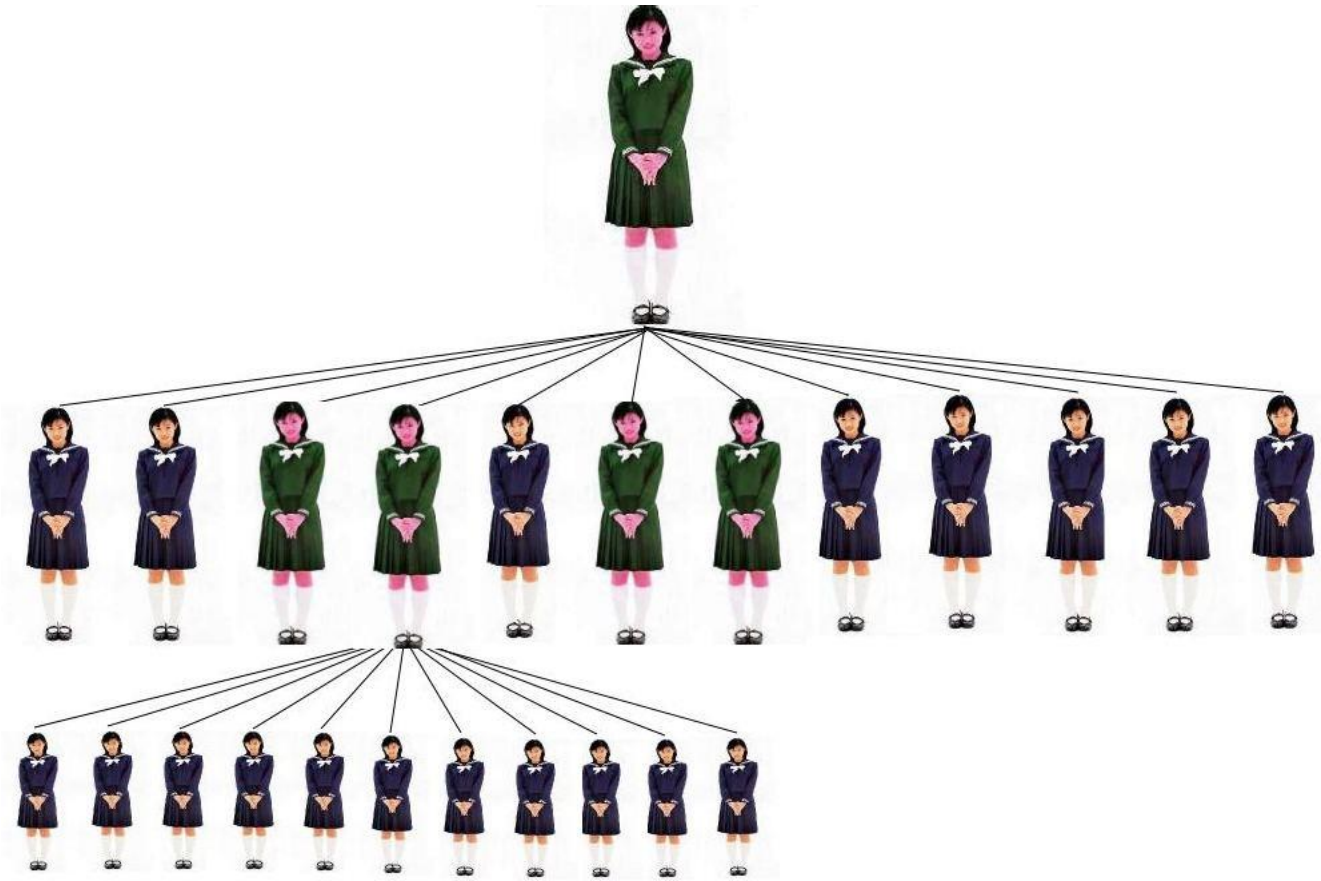

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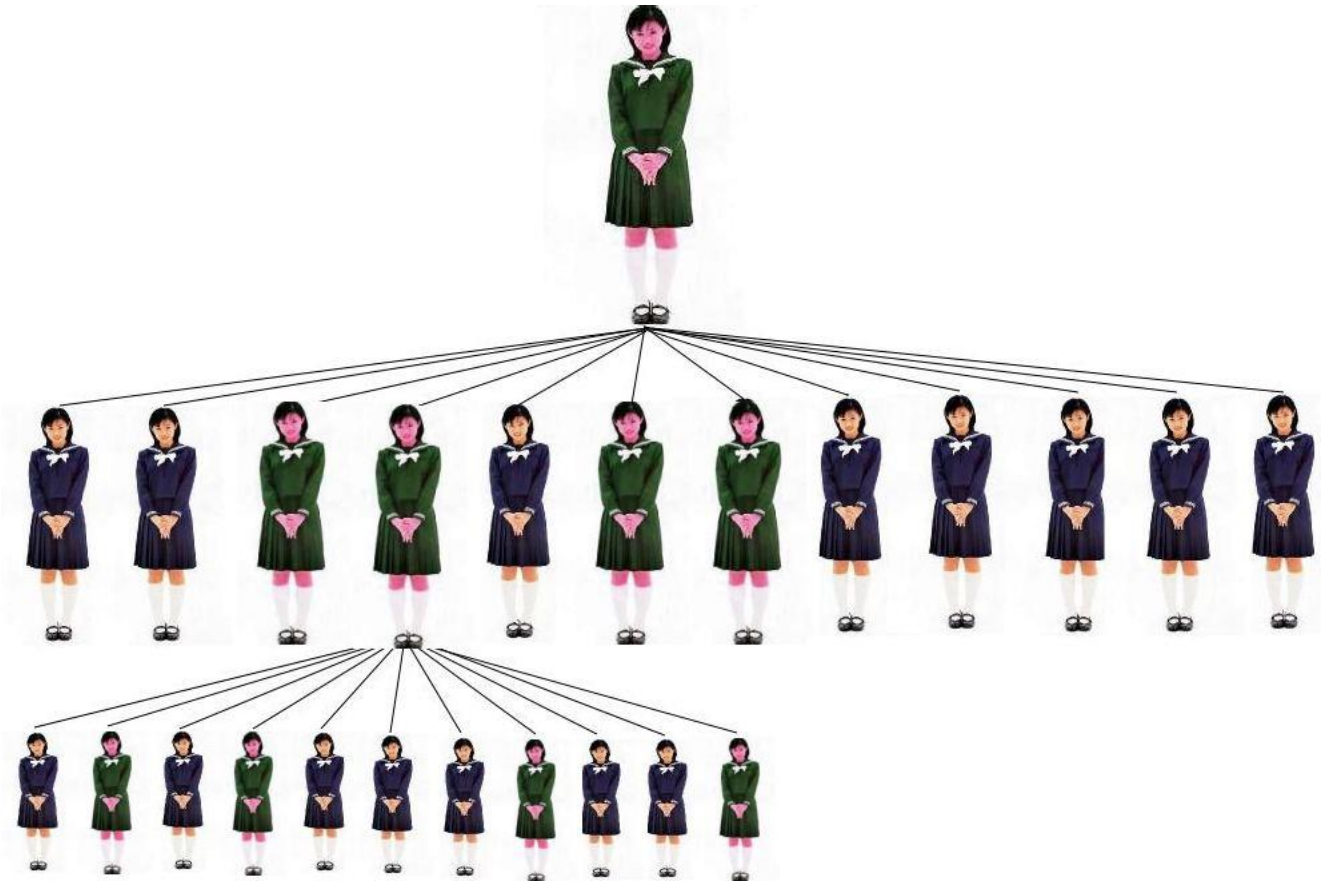


Assume that every infected student infects q other students.

What will be the total number of infections? High school math:

$$1 + q + q^2 + q^3 + \cdots + q^n = \frac{1 - q^{n+1}}{1 - q}$$

$$\rightarrow \frac{1}{1 - q} \quad \text{or} \quad +\infty$$

Depending on $q < 1$ or $q > 1$

Clearly a very poor model (but not as bad as it seems at first), because at a later stage of the epidemic, this q can not be sustained.

Simulation file

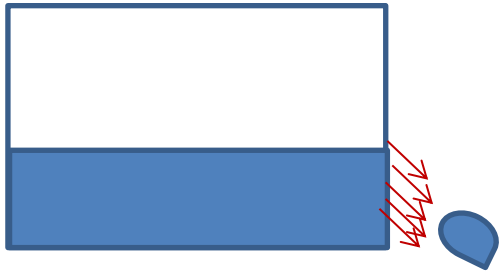
What is a compartmental model?

Consider a compartment, in which material can flow through (in an out)

Put in some water

$W(t)$

Make some holes

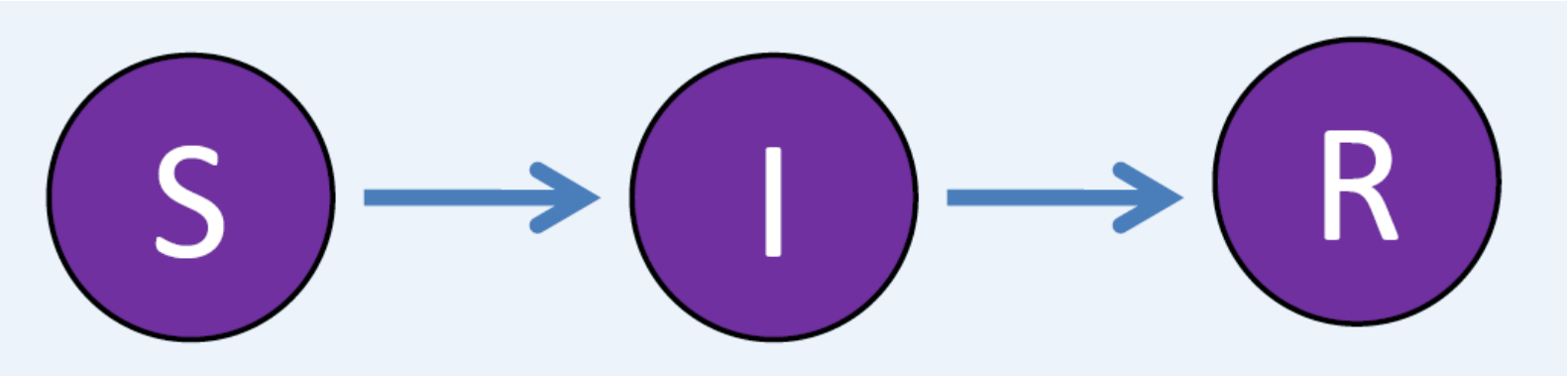


$$\frac{dW(t)}{dt} = -aW(t)$$

Add water at constant rate *K*

$$\frac{dW(t)}{dt} = K - aW(t)$$

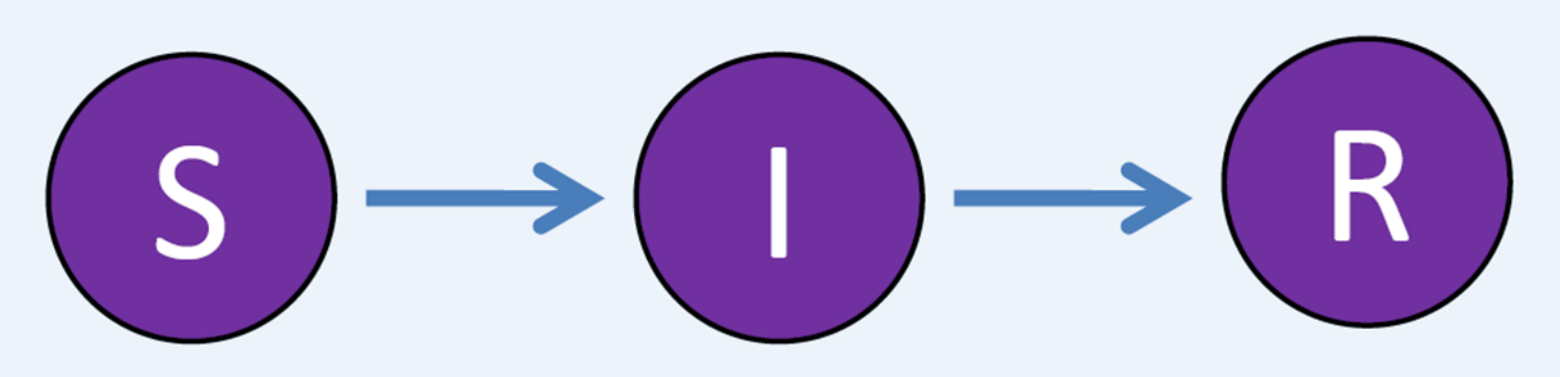
Now consider multiple compartments, each including a group of people, who are allowed to move from an to another. Then we use differential equations to describe the flow of people.



Population is divided into three disjoint groups:

- ***S***usceptible: individuals in the population who have not been infected
- ***I***nfectious: infected individuals who are contagious
- ***R***emoved: individuals who have recovered from the disease and immune (or who have died)

SIR model (without demography)



S(t), I(t), R(t) denotes the number of individuals in the given compartments at time ***t***.

Assumptions:

- Homogeneous population
- Closed population (***S(t)+I(t)+R(t)=N*** constant), no birth and death
- Disease spread by contacts between persons
- Well-mixed population (any two persons are equally likely to encounter each other)
- Time homogeneity (parameters don't change)
- Infectious individuals recover at a given per capita rate

SIR model (without demography)

C(N) per capita contact rate

P transmission probability per contact per unit time

Number of new infections per unit time (transfer rate from ***S*** to ***I*** at time ***t***):

$$\boldsymbol{I(t)\ X\ C(N)\ X\ (S(t)\ /\ N)\ X\ P = \beta\, S(t)\, I(t),}$$

with the notation $\boldsymbol{\beta = C(N)P/N}$.

Average length of infectiousness: ***1/ α***

$$S' = -\beta SI,$$

$$I' = \beta SI - \alpha I,$$

$$R' = \alpha I\ .$$

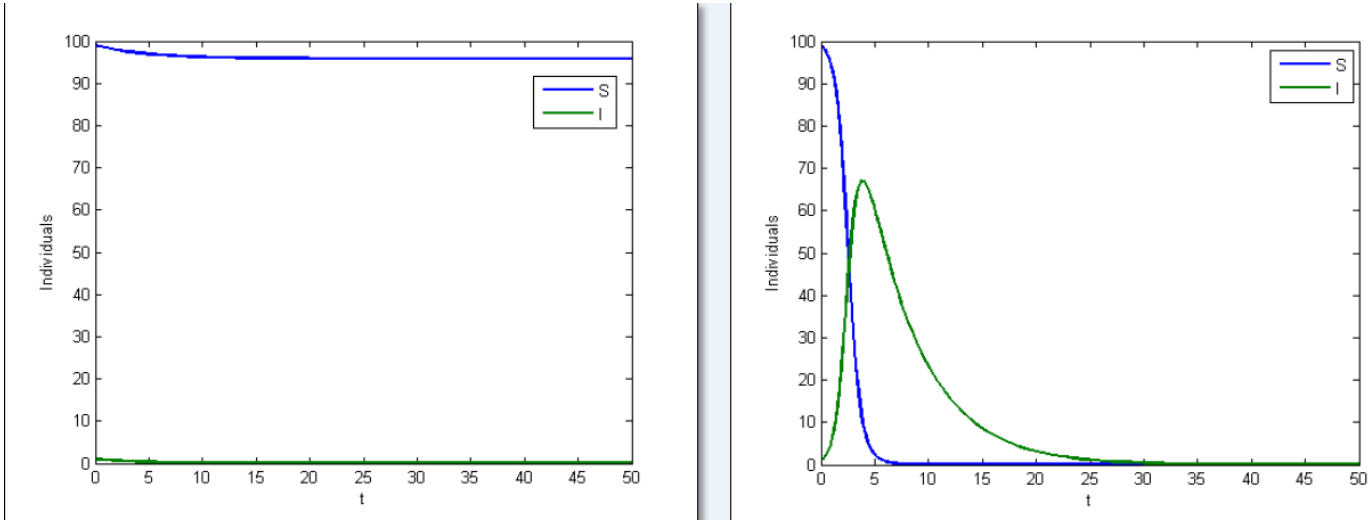
SIR model analysis

$$S' = -\beta SI,$$
$$I' = \beta SI - \alpha I, \qquad S(0) = S_0 > 0, \quad I(0) = I_0 > 0, \quad R(0) = 0.$$
$$R' = \alpha I\ .$$

Constant population size built in: $\mathbf{S' + I' + R' = N' = 0}$

Computer can solve special cases for us. Two different scenarios:

β=0.7 $\alpha = 0.9$ N=100 ***β=2 $\alpha = 0.2$ N=100***



Disease dies out quickly vs large outbreak. What's the difference?
How can we distinguish?

$\mathcal{R}_0$

$$I' = (\beta S - \alpha)I$$

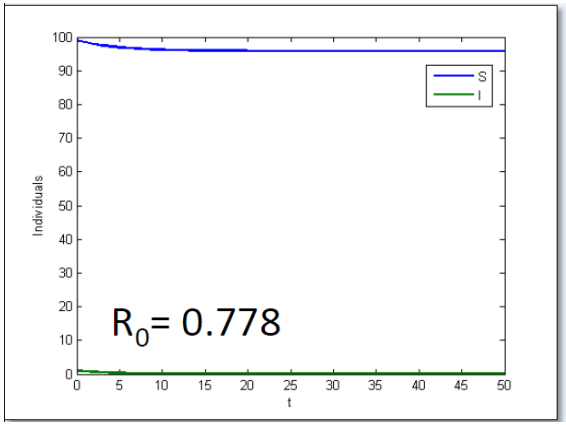
Number of infectious individuals increasing when $\boldsymbol{\beta S(t) > \alpha}$

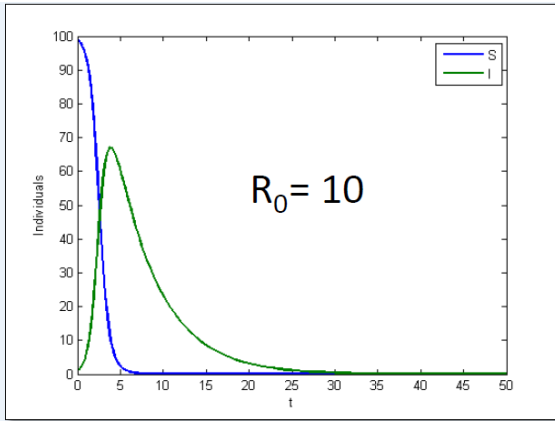
At the beginning (***t=0***): $\boldsymbol{\beta S(0) > \alpha}$

Equivalently, $\mathbf{R_0 = \frac{\beta S(0)}{\alpha} > 1}$

Basic reproduction number = length of infectious period X
number of new infections per unit time per one infectious individual
= total number of secondary infection by a single infected individual
Introduced into a wholly susceptible population ($N \approx S(0)$)

$\mathbf{R_0}$ is a threshold parameter



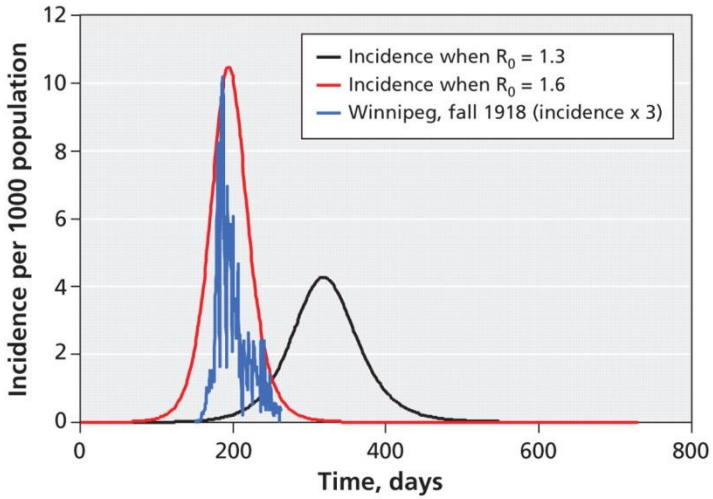


How to decrease R_0 ?

$$\mathbf{R_0 = \frac{\beta S(0)}{\alpha}}$$

- Decrease $\boldsymbol{\beta}$ – reduce transmission probability, or reduce contacts
- Decrease $\mathbf{S(0)}$ – vaccination
- Increase $\boldsymbol{\alpha}$ – treatment (shortening infectious period)

Now we understand Camus: after many people got infected, once ***S(t)*** drops below $\boldsymbol{\alpha/\beta}$, the disease dies out quickly.



Mathematics of vaccination / herd immunity

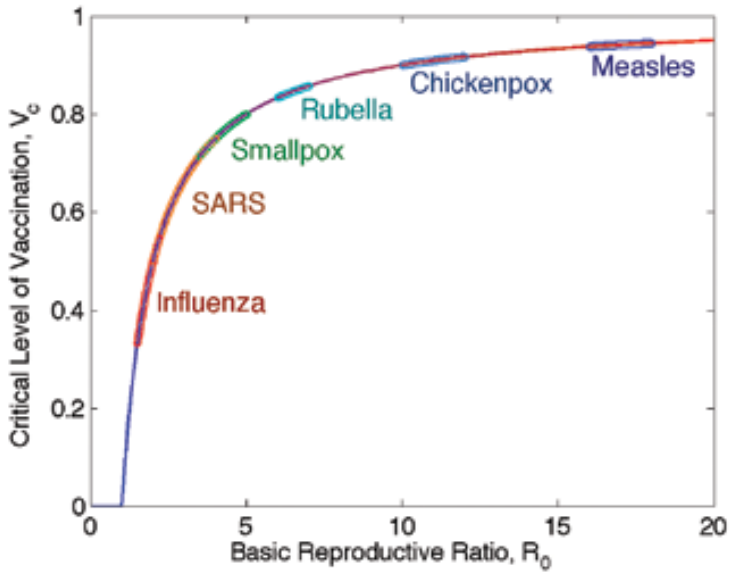
Values of R_0 of well-known infectious diseases[1]

Disease	Transmission	R_0
Measles	Airborne	12–18
Pertussis	Airborne droplet	12–17
Diphtheria	Saliva	6–7
Smallpox	Airborne droplet	5–7
Polio	Fecal-oral route	5–7
Rubella	Airborne droplet	5–7
Mumps	Airborne droplet	4–7
HIV/AIDS	Sexual contact	2–5
SARS	Airborne droplet	2–5[2]
Influenza (1918 pandemic strain)	Airborne droplet	2–3[3]
Ebola	Bodily Fluids	1–4

$$\mathbf{R_0} = \frac{\boldsymbol{\beta S(0)}}{\boldsymbol{\alpha}}$$

$$\mathbf{R_V} = \frac{\boldsymbol{\beta S(0)(1-V)}}{\boldsymbol{\alpha}} = \mathbf{(1-V)R0}$$

$$\mathbf{R_V < 1} \Leftrightarrow \boldsymbol{V > 1 - 1/R_0}$$



More detailed mathematical analysis of SIR model

$$S' = -\beta SI,$$
$$I' = \beta SI - \alpha I,$$
$$R' = \alpha I\ .$$

- Solutions remain nonnegative
- ***S(t)*** is monotone decreasing
- ***I(t)*** is converging to **0** (can be proven from ***S'+I'= −α I*** **).**
- ***S(t)*** is also converging, but where (final size, that gives also the total number of infections) ?
- What is the peak size?

Consider ***dI/dS***, separate variables and integrate to obtain:

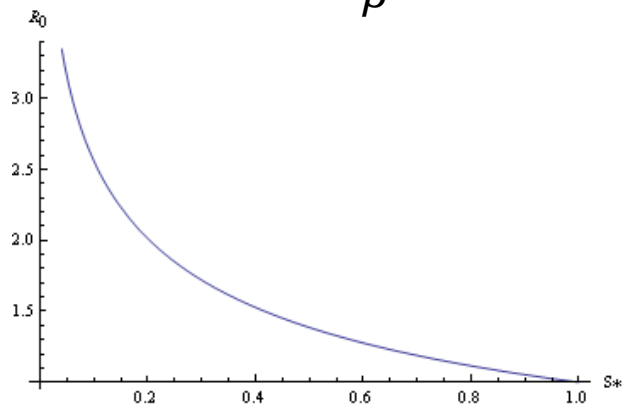
$$I(t) + S(t) - \frac{\alpha}{\beta}\log S(t) = \quad \text{constant}$$

This is called a first integral (invariant) of the system. While the SIR differential equations can not be solved explicitly, the first integral essentially solves it.

$$I(t) + S(t) - \frac{\alpha}{\beta}\log S(t) = I(0) + S(0) - \frac{\alpha}{\beta}\log S(0) = S(\infty) - \frac{\alpha}{\beta}\log S(\infty)$$

This leads to the Final Size Relation:

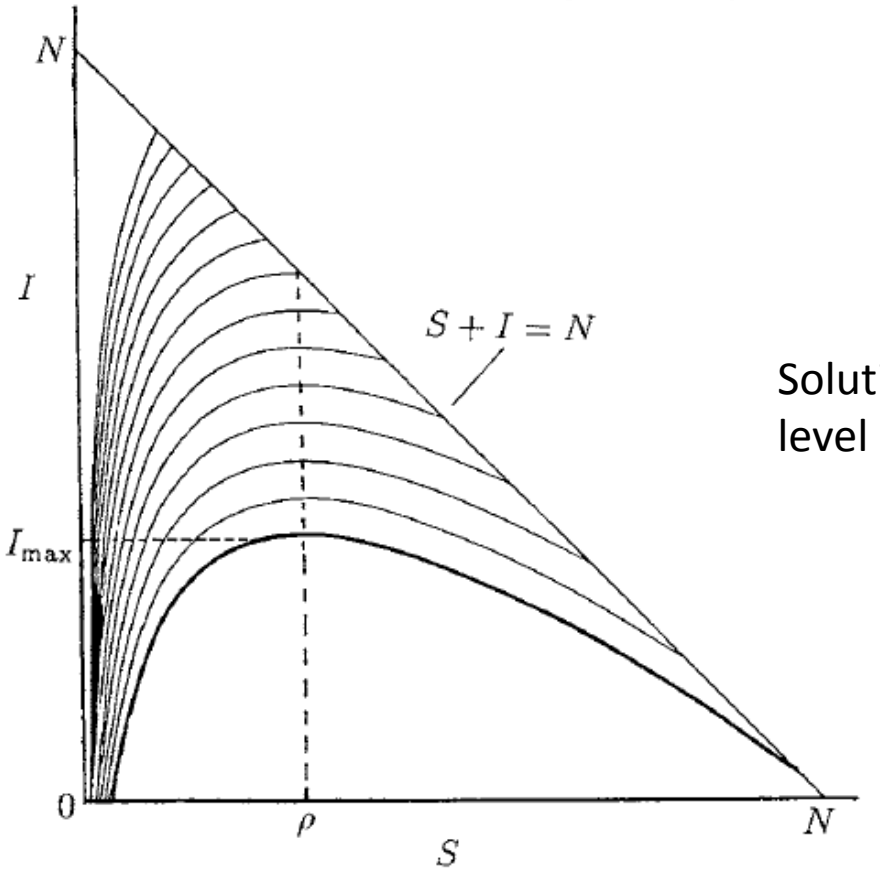
$$\log\frac{S_0}{S_\infty} = \mathcal{R}_0\left[1 - \frac{S_\infty}{N}\right]$$



The maximum number of infectives at any time is the number of infectives when the derivative of I is zero, that is, when $S = \alpha/\beta$. This maximum is given by

$$I_{max} = S_0 + I_0 - \frac{\alpha}{\beta}\log S_0 - \frac{\alpha}{\beta} + \frac{\alpha}{\beta}\log\frac{\alpha}{\beta},$$

obtained by substituting $S = \alpha/\beta, I = I_{max}$



Solutions move along the level curves of the first integral

Does this simple SIR model work?

$\mathcal{R}_0 = 3.8$

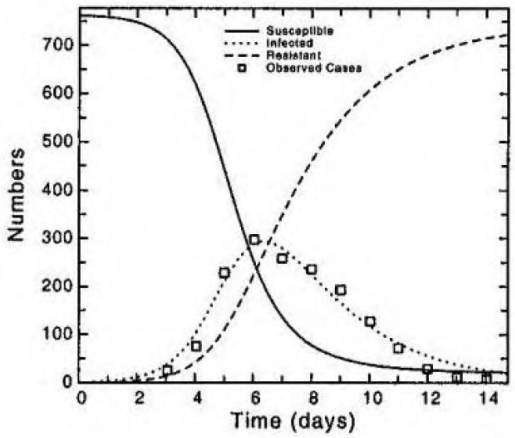


Figure 15.2: Model and data for a 1978 flu epidemic in an English boarding school for boys. Parameters are: $S(0) = 762$, $I(0) = 1$, $\alpha = 0.00218$, $\beta = 0.4404$, $N = 763$.

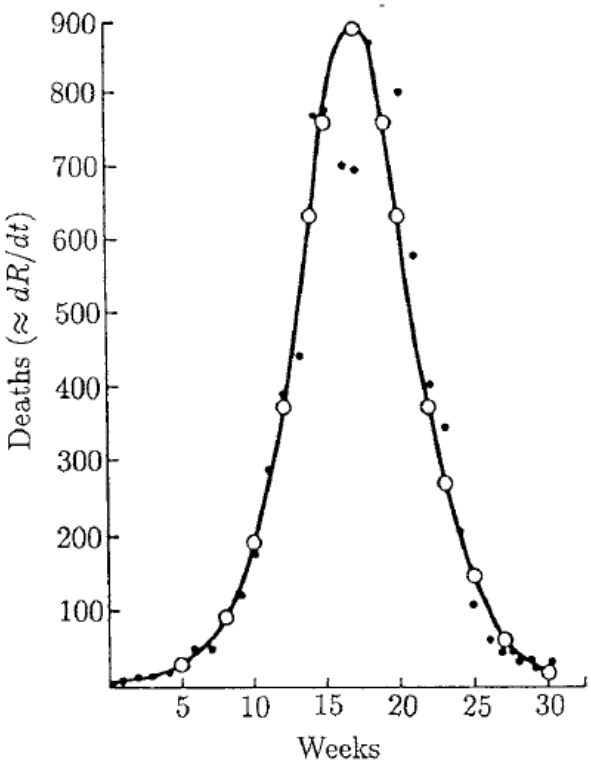


Figure 10.2. Bombay plague epidemic of 1905–1906. Comparison between the data (•) and theory (○) from the (small) epidemic model and where the number of deaths is approximately dR/dt given by (10.16). (After Kermack and McKendrick 1927)

Bombay Plague Epidemic 1905–1906

Taken from Brauer & Castillo-Chavez book (2011)

Example 2. (The Great Plague in Eyam) The village of Eyam near Sheffield, England, suffered an outbreak of bubonic plague in 1665–1666 the source of which is generally believed to be the Great Plague of London. The Eyam plague was survived by only 83 of an initial population of 350 persons. Since detailed records were preserved and the community was persuaded to quarantine itself to try to prevent the spread of disease to other communities, the disease in Eyam has been used as a case study for modeling [Raggett (1982)]. Detailed examination of the data indicates that there were actually two outbreaks, of which the first was relatively mild. Thus we shall try to fit the model (9.2) over the period from mid-May to mid-October 1666, measuring time in months with an initial population of 7 infectives and 254 susceptibles, and a final population of 83. Raggett (1982) gives values of susceptibles and infectives in Eyam on various dates, beginning with $S(0) = 254$, $I(0) = 7$, shown in Table 9.1.

The final size relation with $S_0 = 254$, $I_0 = 7$, $S_\infty = 83$ gives $\beta/\alpha = 6.54 \times 10^{-3}$, $\alpha/\beta = 153$. The infective period was 11 days, or 0.3667 month, so that $\alpha = 2.73$. Then $\beta = 0.0178$. The relation (9.5) gives an estimate of 30.4 for the maximum number of infectives. We use the values obtained here for the parameters β and τ in the model (9.2) for simulations of both the phase plane, here the (S, I)-plane, and for graphs of S and I as functions of t (Figures 9.3, 9.4, 9.5). Figure 9.6 plots these data points together with the phase portrait given in Figure 9.3 for the model (9.2).

The actual data for the Eyam epidemic are remarkably close to the predictions of this very simple model. However, the model is really too good to be true. Our model assumes that infection is transmitted directly between people. While this is

Date (1666)	Susceptibles	Infectives
July 3/4	235	14.5
July 19	201	22
August 3/4	153.5	29
August 19	121	21
September 3/4	108	8
September 19	97	8
October 4/5	Unknown	Unknown
October 20	83	0

Table 9.1 Eyam Plague data.

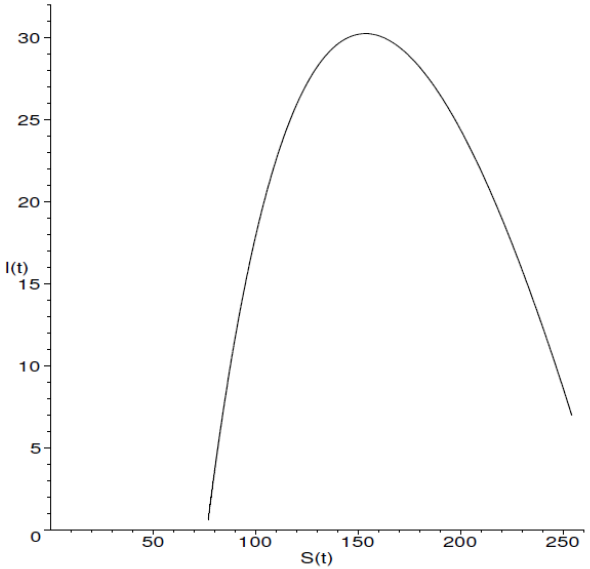


Fig. 9.3 The *S*-*I* plane.

possible, bubonic plague is transmitted mainly by rat fleas. When an infected rat is bitten by a flea, the flea becomes extremely hungry and bites the host rat repeatedly, spreading the infection in the rat. When the host rat dies, its fleas move on to other rats, spreading the disease further. As the number of available rats decreases, the fleas move to human hosts, and this is how plague starts in a human population (although the second phase of the epidemic may have been the pneumonic form of bubonic plague, which can be spread from person to person). One of the main reasons for the spread of plague from Asia into Europe was the passage of many trading ships; in medieval times ships were invariably infested with rats. An accurate model of plague transmission would have to include flea and rat populations, as well as movement in space. Such a model would be extremely complicated, and its predictions might well not be any closer to observations than our simple unrealistic model. Very recent study of the data from Eyam suggests that the rat population may not have been large enough to support the epidemic and human to human transmission

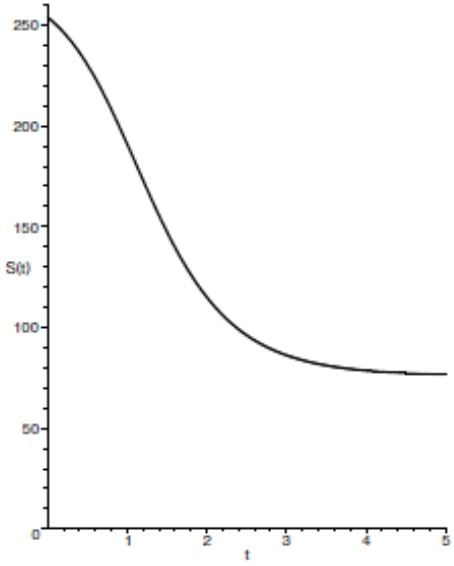


Fig. 9.4 S as a function of t.

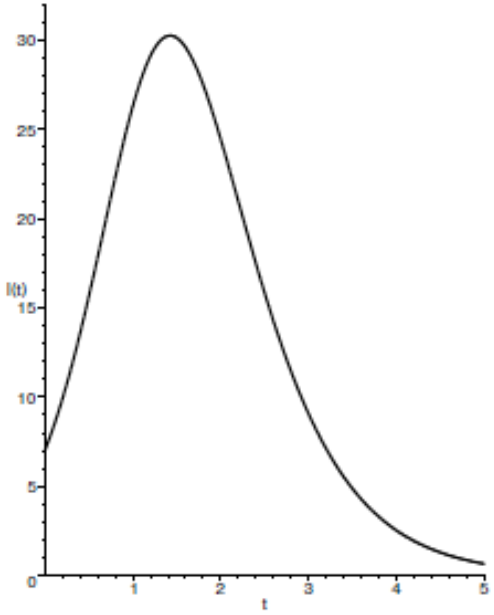


Fig. 9.5 I as a function of t.

was also a factor. Raggett (1982) also used a stochastic model to fit the data, but the fit was rather poorer than the fit for the simple deterministic model(9.2).

In the village of Eyam the rector persuaded the entire community to quarantine itself to prevent the spread of disease to other communities. One effect of this policy was to increase the infection rate in the village by keeping fleas, rats, and people in close contact with one another, and the mortality rate from bubonic plague was much higher in Eyam than in London. Further, the quarantine could do nothing to prevent the travel of rats and thus did little to prevent the spread of disease to other communities. One message this suggests to mathematical modelers is that control

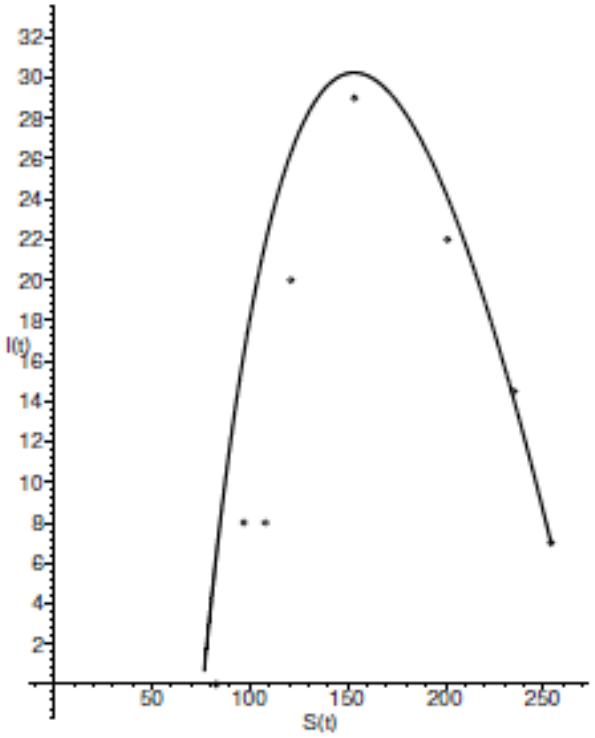
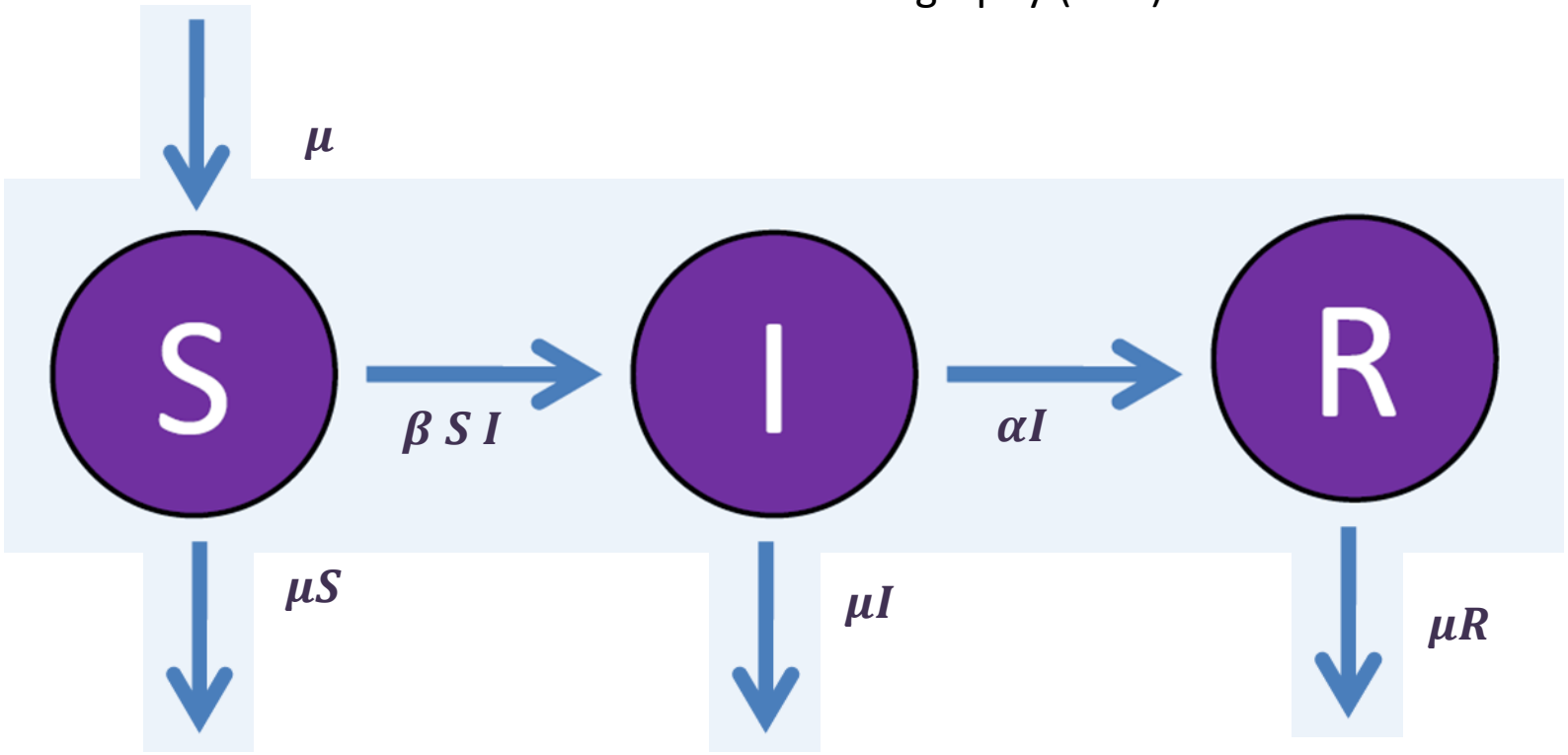


Fig. 9.6 The *S-I* plane, model and data.

strategies based on false models may be harmful, and it is essential to distinguish between assumptions that simplify but do not alter the predicted effects substantially, and wrong assumptions that make an important difference.

SIR with demography (N=1)



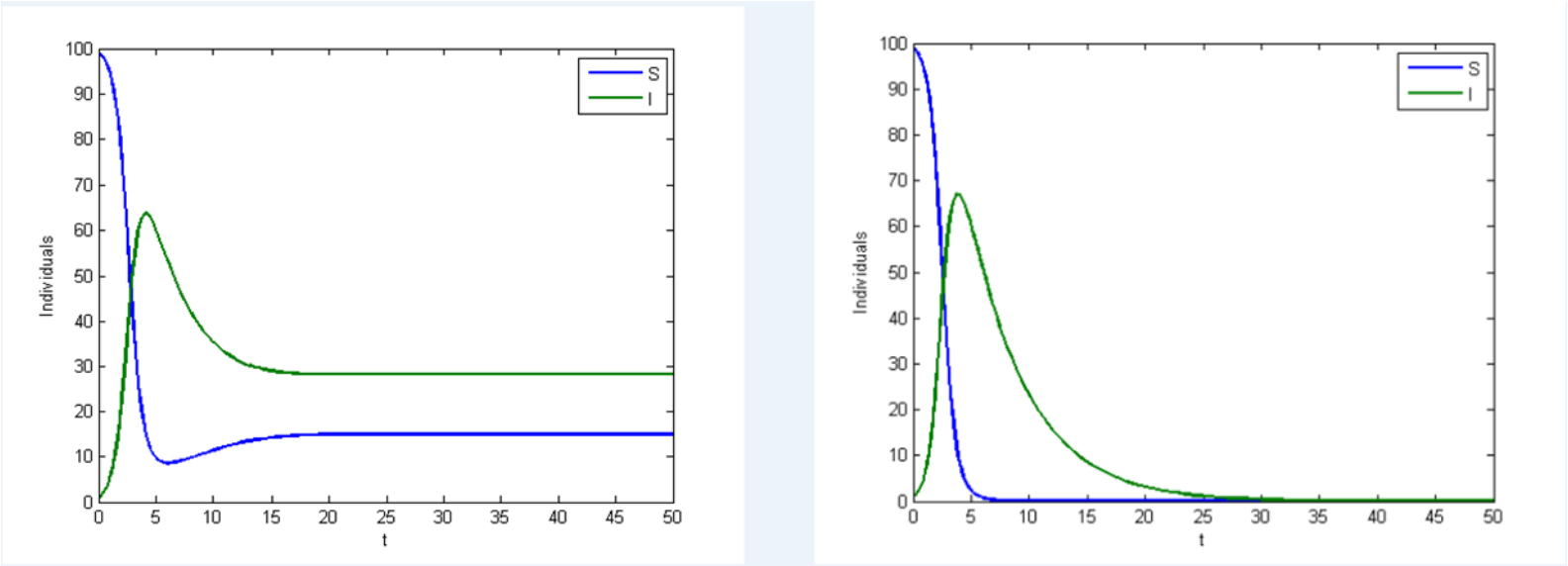
Disease Free Equilibrium (1,0,0)

$$R_0 = \frac{\beta}{\alpha + \mu}$$

Endemic Equilibrium (1/ R_0 , I^*,R^*)

R_0 is a threshold that determines which equilibrium is globally asymptotically stable

SIR with vs without demography



$\beta = 2, \alpha = 0.2$, **μ=0.1 vs. 0**

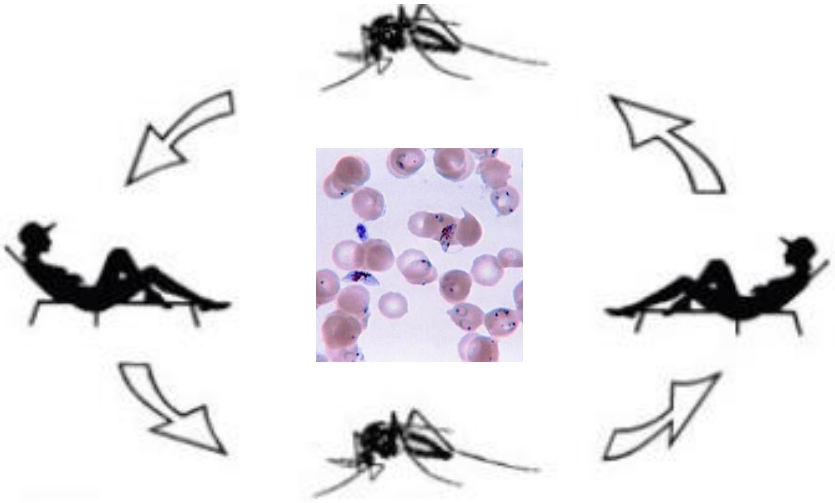
Threshold property: disease will be eradicated, or disease remains endemic.

A vector borne disease: brief history of malaria modeling

- BC 2700 in Chinese medical texts

  Mal'aria = 'bad air' in Latin

- 1898: malaria is caused by parasites transmitted by mosquitoes



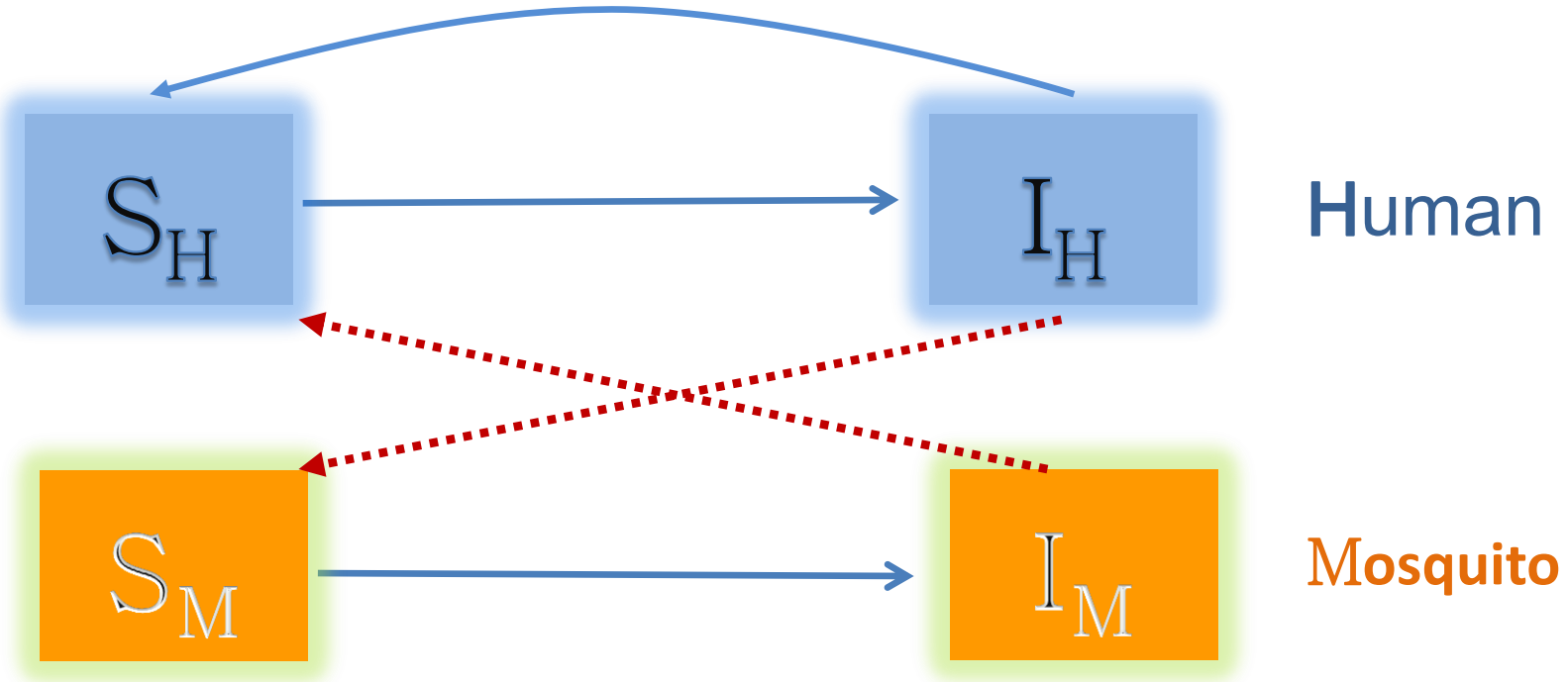
Ronald Ross

Mosquitoes transmit malaria.

COOL! LET'S GO AND FINISH THEM ALL.

That's pointless.
We can't kill all of them anyway.

We don't have to kill all mosquitoes.
Look at my model.



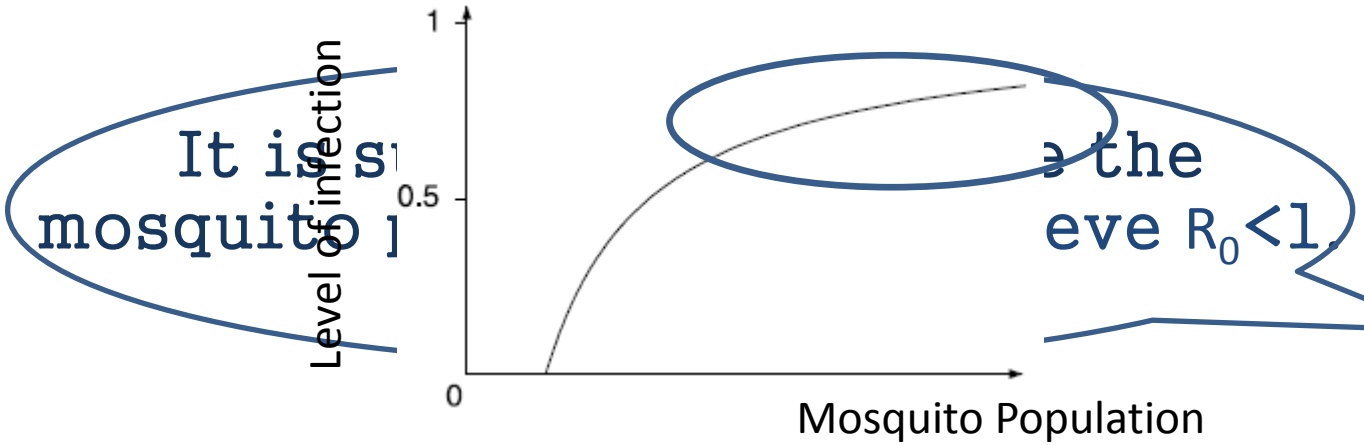
$$\frac{dI_H}{dt} = p'bI_M \frac{H - I_H}{H} - rI_H$$

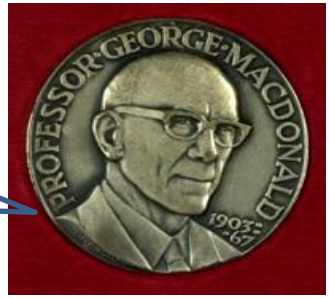
$$\frac{dI_M}{dt} = pb(M - I_M)\frac{I_H}{H} - mI_M$$

Threshold dynamics for Ross-Macdonald models

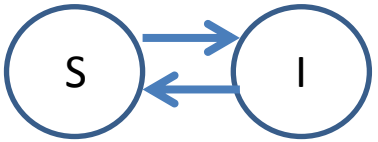
- If $R_0<1$, malaria disappears.
- If $R_0>1$, malaria persists (endemic)

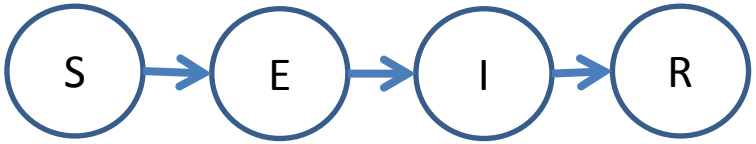
$$R_0^2 = C\,\frac{(\text{contact rate}) \times (\text{mosquito population})}{(\text{recovery rate})}$$

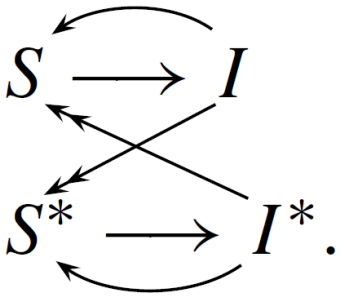


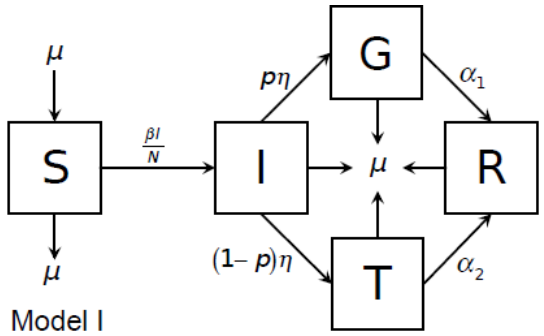


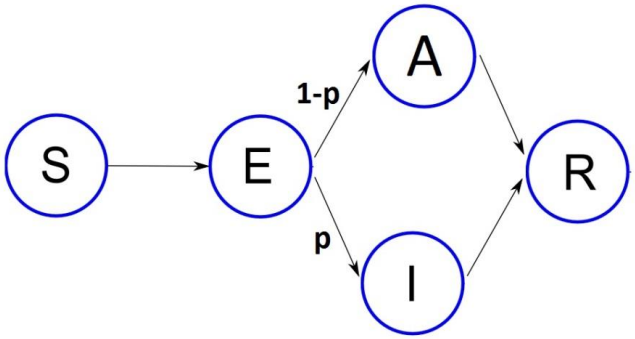
Extensions of the basic model: SIS, SEIR, SEIRS, SLAIR, treatment, vaccination, quarantine, two strains…



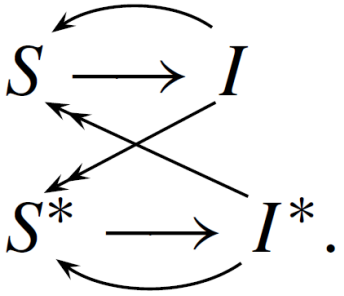








$$\dot{S}(t) = -\beta S(t)(I(t) + \delta A(t)),$$
$$\dot{E}(t) = \beta S(t)(I(t) + \delta A(t)) - \mu_E E(t),$$
$$\dot{A}(t) = (1-p)\mu_E E(t) - \mu_A A(t),$$
$$\dot{I}(t) = p\mu_E E(t) - \mu_I I(t),$$
$$\dot{R}(t) = \mu_A A(t) + \mu_I I(t)$$



Heterogeneity: age, sex, location…

Basic reproduction number: secondary infections by a single infected individual. But in a heterogeneous model there are many types of individuals! How can we define R_0 ?

Next Generation Matrix – as an example, let there be three types of persons. Construct a matrix

$$M = \begin{pmatrix} R(1,1) & R(1,2) & R(1,3) \\ R(2,1) & R(2,2) & R(2,3) \\ R(3,1) & R(3,2) & R(3,3) \end{pmatrix}$$

where ***R(i,j)*** expresses that how many secondary infections generated among type ***j*** persons by a type ***i*** person.

R_0 is defined as the spectral radius of this matrix (dominant real eigenvalue).

Example: (hetero)Sexually transmitted diseases: $M = \begin{pmatrix} 0 & A \\ B & 0 \end{pmatrix}$ and $R_0 = \sqrt{AB}$

Example: an age structured influenza model with vaccination

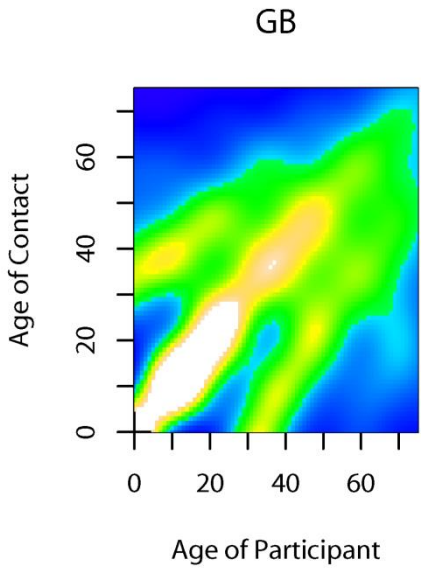
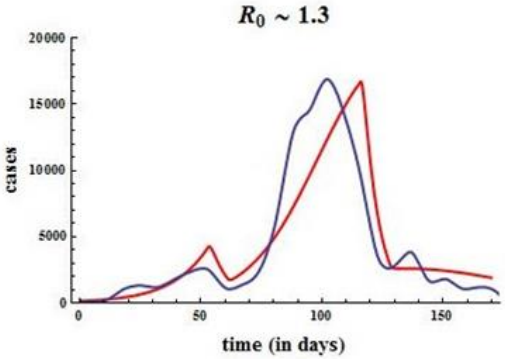
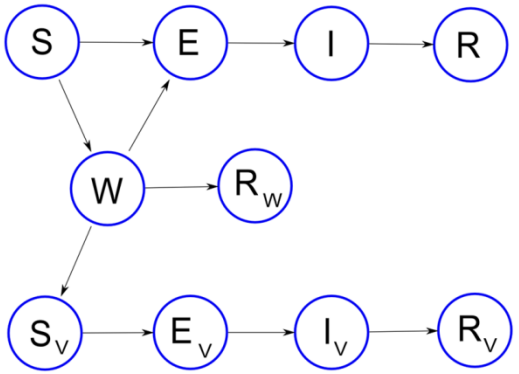


Figure from Mossong et al

	00-04	05-09	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70+
00-04	2,512358	0,715402	0,230419	0,131918	0,312627	0,39074	0,644092	0,569294	0,486006	0,344638	0,237785	0,226478	0,248656	0,171056	0,154336
05-09	1,129217	6,020013	0,919965	0,354768	0,223829	0,351701	0,694929	0,913365	0,653525	0,339602	0,434154	0,413509	0,276253	0,311982	0,258132
10-14	0,444652	1,229884	8,616317	1,177201	0,259255	0,231693	0,452891	0,655259	0,817767	0,521273	0,882193	0,491112	0,42846	0,261536	0,561207
15-19	0,327782	0,338254	1,552874	9,182383	1,310065	0,566779	0,447831	0,624206	1,056077	1,356961	0,970471	0,374456	0,36124	0,271604	0,585689
20-24	0,447924	0,289668	0,39492	1,342387	3,681434	1,94421	0,745387	0,649074	0,993901	0,768742	0,883455	0,637425	0,453804	0,289164	0,315059
25-29	0,755581	0,607605	0,416299	0,640255	1,852325	2,421122	1,392598	1,038678	0,883325	0,949513	1,100216	0,859887	0,714828	0,588644	0,460695
30-34	1,104155	0,912903	0,56788	0,466437	1,086752	1,686657	2,04434	1,450039	1,217251	1,082328	0,918856	0,905328	0,880329	0,716529	0,495146
35-39	1,006314	1,039015	0,963011	0,803092	0,846464	1,097758	1,746209	2,211847	1,42368	1,203872	0,982144	0,866633	0,918954	0,828398	0,48703
40-44	0,670499	1,048962	1,222623	0,952284	0,791679	0,942851	1,236204	1,824605	1,90436	1,425513	1,118822	0,991312	0,892044	0,870134	0,723835
45-49	0,44411	0,515931	0,772672	1,105645	1,002383	0,918615	0,783045	1,078251	1,386477	1,683196	1,167289	0,954483	0,764105	0,630791	0,687712
50-54	0,431302	0,39123	0,499789	0,61663	0,792194	0,89471	0,822741	0,796846	1,017834	1,179911	1,265395	1,131831	0,721695	0,624814	0,573115
55-59	0,405564	0,3176	0,260644	0,339128	0,505306	0,664289	0,574051	0,552103	0,464662	0,651198	1,031867	1,500609	1,053937	0,61288	0,518528
60-64	0,320396	0,296946	0,189185	0,19065	0,269948	0,429697	0,527745	0,542105	0,516995	0,425934	0,516361	0,850866	0,938125	0,857512	0,568151
65-69	0,256757	0,230012	0,201335	0,140435	0,134876	0,220179	0,261126	0,439805	0,293448	0,189294	0,216956	0,477602	0,656456	0,894606	0,685851
70+	0,392157	0,479821	0,376735	0,224435	0,318317	0,378067	0,320636	0,494041	0,582925	0,616799	0,58854	0,786284	0,800018	1,100269	1,214828

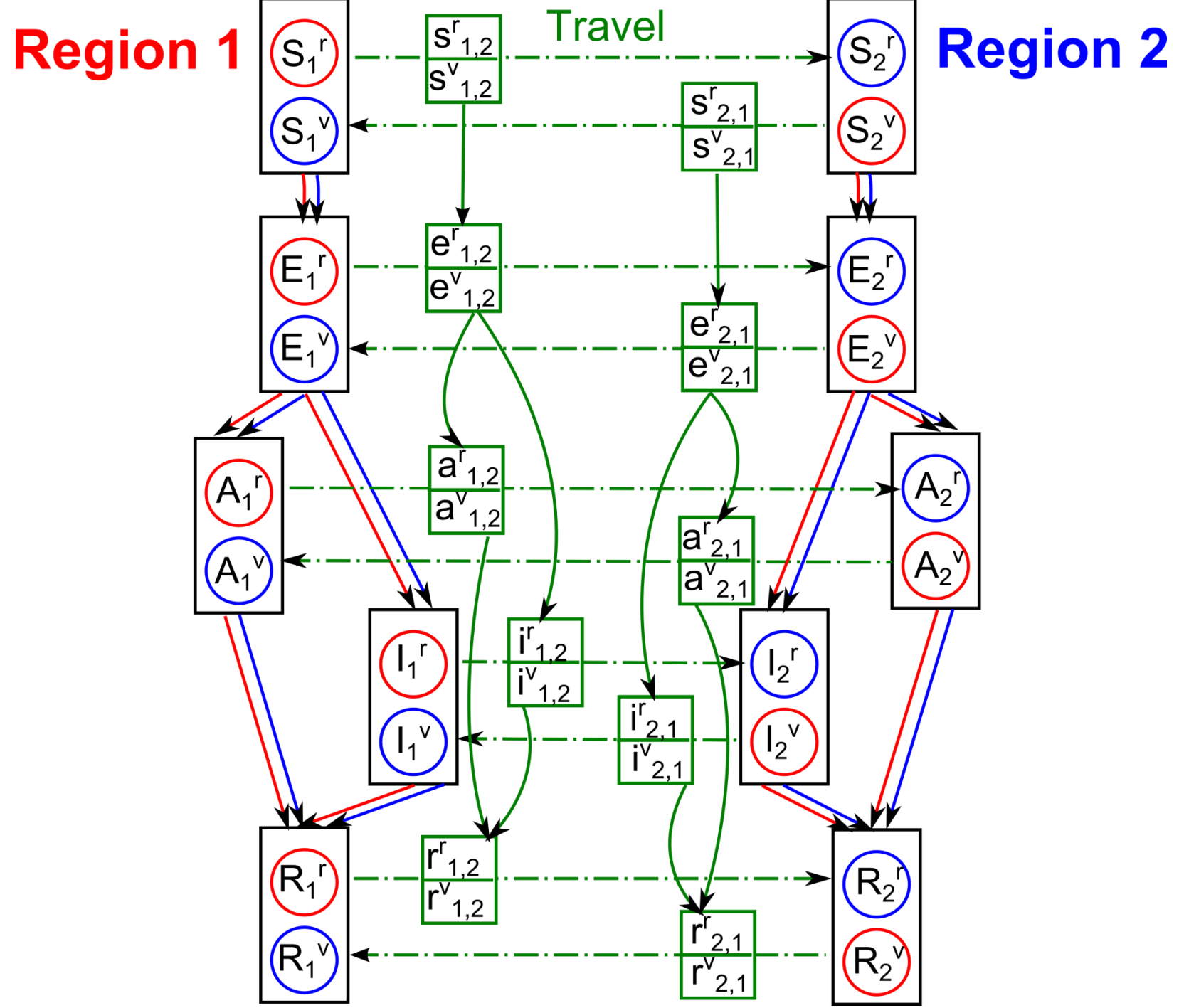
$$\mathcal{C} = \begin{pmatrix} 5,3580 & 1,0865 & 3,0404 & 2,4847 & 0,8150 \\ 0,9507 & 10,2827 & 2,8148 & \mathbf{3,6215} & 0,7752 \\ 1,1201 & 1,1852 & 6,5220 & 4,1938 & 0,9016 \\ 0,8027 & 1,3372 & 3,6776 & 5,2632 & 1,3977 \\ \mathbf{0,5187} & 0,5638 & 1,5573 & 2,7531 & 2,0742 \end{pmatrix}$$





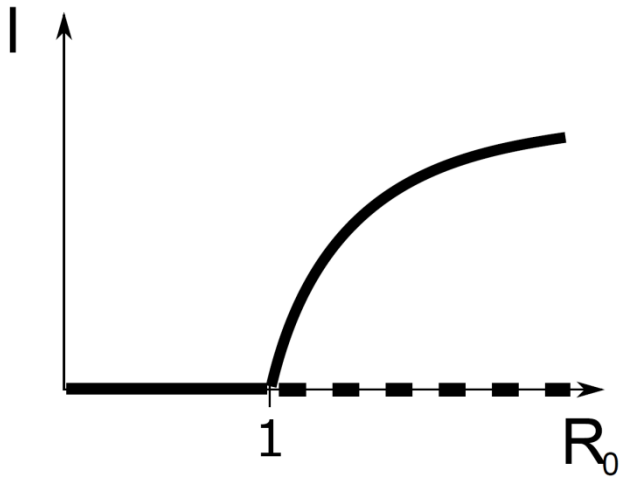
$$\dot{S}^i(t) = -S^i(t)\lambda^i(t) - V^i(t) \qquad \dot{S}^i_V(t) = (1-q_i)\mu_W W^i(t) - S^i_V(t)\lambda^i(t)$$
$$\dot{E}^i(t) = (S^i(t) + W^i(t))\lambda^i(t) - \mu^i_E E^i(t) \qquad \dot{E}^i_V(t) = S^i_V(t)\lambda^i(t) - \mu^i_{E_V} E^i_V(t)$$
$$\dot{I}^i(t) = \mu^i_E E^i(t) - \mu^i_I I^i(t) \qquad \dot{I}^i_V(t) = \mu^i_{E_V} E^i_V(t) - \mu^i_{I_V} I^i_V(t)$$
$$\dot{R}^i(t) = \mu^i_I I^i(t) \qquad \dot{R}^i_V(t) = \mu^i_{I_V} I^i_V(t)$$
$$\dot{W}^i(t) = V^i(t) - W^i(t)\lambda^i(t) - \mu_W W^i(t) \qquad \dot{R}^i_W(t) = q_i \mu_W W^i(t)$$

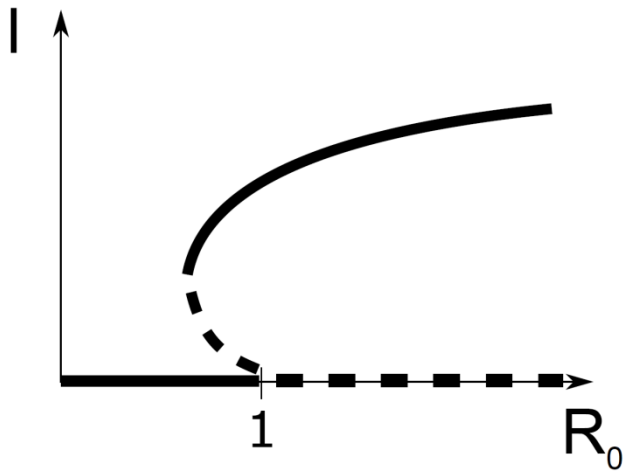
force of infection: $\lambda^i(t) = \sum_{j=1}^{5}(\beta_{j,i}(I^j(t) + \delta I^j_V(t)))$, age groups: $i = 1, 2, \ldots 5$

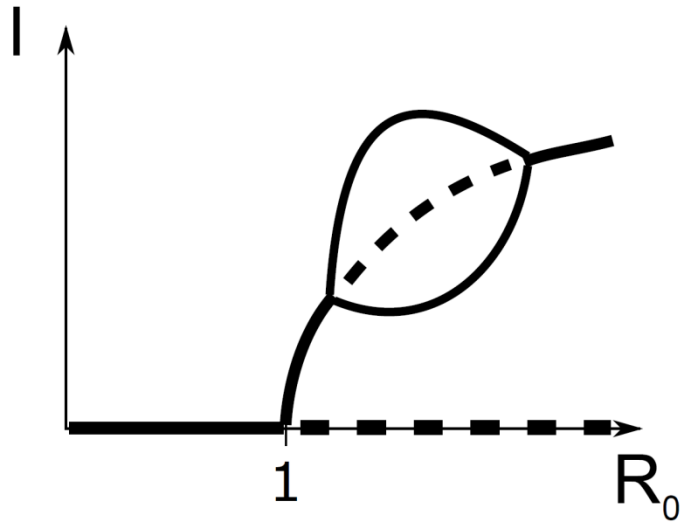

Region 1
Travel
Region 2
S_1^r
S_1^v
$s^r_{1,2}$
$s^v_{1,2}$
$s^r_{2,1}$
$s^v_{2,1}$
S_2^r
S_2^v
E_1^r
E_1^v
$e^r_{1,2}$
$e^v_{1,2}$
$e^r_{2,1}$
$e^v_{2,1}$
E_2^r
E_2^v
A_1^r
A_1^v
$a^r_{1,2}$
$a^v_{1,2}$
$a^r_{2,1}$
$a^v_{2,1}$
A_2^r
A_2^v
I_1^r
I_1^v
$i^r_{1,2}$
$i^v_{1,2}$
$i^r_{2,1}$
$i^v_{2,1}$
I_2^r
I_2^v
R_1^r
R_1^v
$r^r_{1,2}$
$r^v_{1,2}$
$r^r_{2,1}$
$r^v_{2,1}$
R_2^r
R_2^v

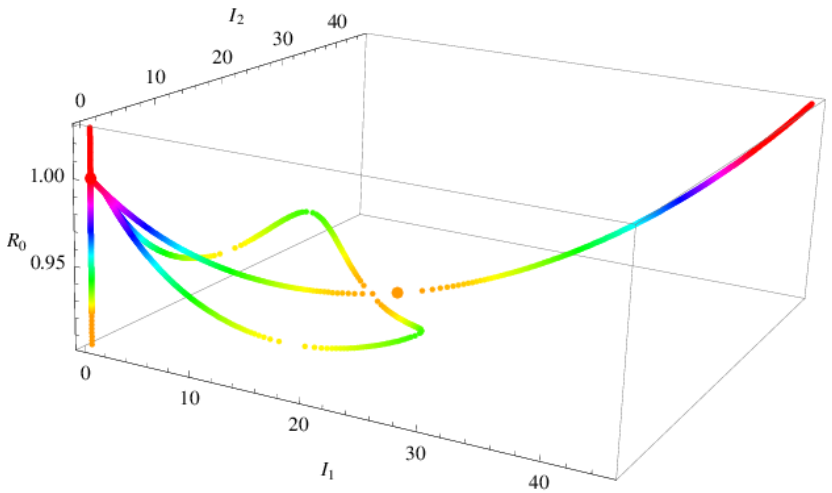
videok

Does R_0 tell us the whole story? Sometimes, but not always.



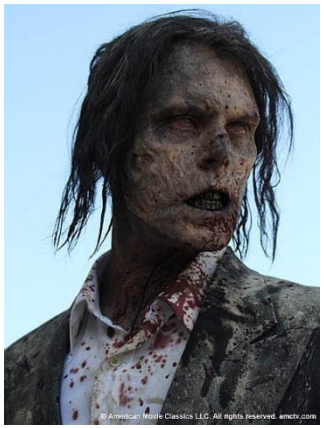






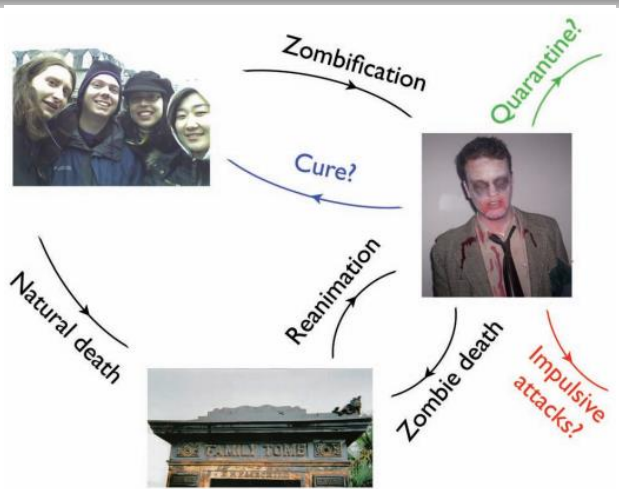
After all this serious stuff, at the end let us consider a cheerful disease

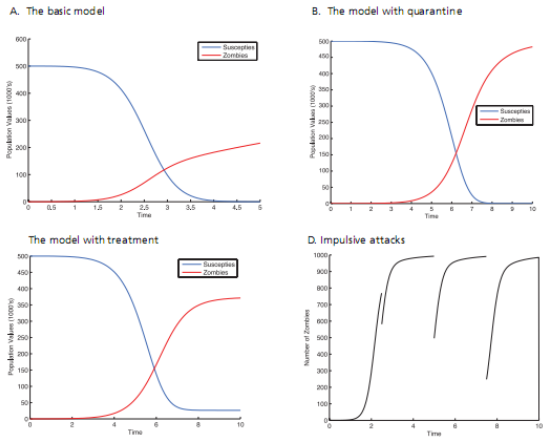
Containing a zombie outbreak



AMC

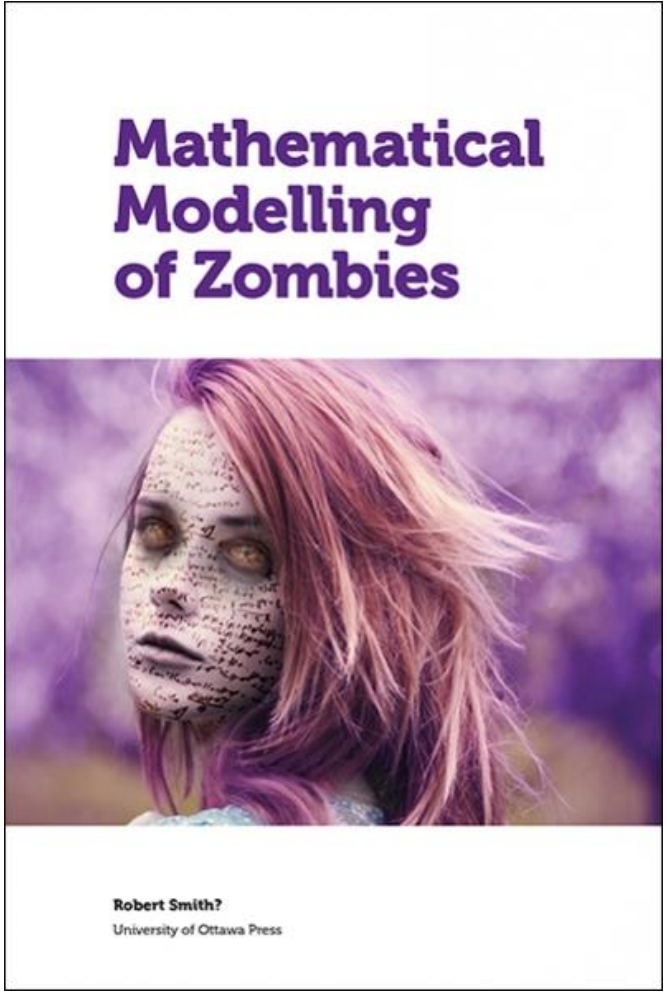
Robert Smith?, A report on the zombie outbreak of 2009: How mathematics can save us (no really), Canadian Medical Association Journal, 2009, 181: E297-E300
P Munz, I Hudea, J Imad, RJ Smith? When zombies attack!: Mathematical modelling of an outbreak of zombie infection, Infectious Disease Modelling Research Progress 2009, 133-150





Selected media appearances

- Zombies lurk at the end of a gravel road in Virginia. And they must die. (The Washington Post)
- How MATHS could save you from the zombie apocalypse: Equation shows why the living dead are viruses of the monster world (The Daily Mail)
- Surviving a Zombie Apocalypse: Just Do the Math (Live Science)
- The disease, math, zombie connection (CBC Kelowna - 6 minutes)
- A light-hearted look at zombies, math, and serious disease (UBC Okanagan)
- Zombies + math = serious disease (Castanet News, Kelowna)
- Using mathematical models to eradicate disease (blog for Mathematics of Planet Earth 2013)
- Skeptic Check on Big Picture Science (8 minutes)
- Science Scoop: Zombies (part of Virtual Researchers on Call, which also promotes careers in science - 5 min)
- Mathematical models, not guns, key to zombie survival kit (Hallowe'en blog post for the Ottawa Gazette)
- Ottawa's Zombieland (discussing the impact of a zombie invasion on Canada's capital city)
- "Ottawa's hidden math scene: Revealing the numbers behind the city's art and culture" Apartment 613 (discussing chaos theory)
- NPR's "Bluff the Listener", a segment on "Wait, Wait Don't Tell Me" tries to convince you Bieber Fever isn't real
- A Mathematical Explanation Of The Justin Bieber Phenomenon (Business Insider)
- CBC News item on my zombie class (2 min)
- Zombies as Brain Food (The Ottawa Citizen review of Braaaiiinnnsss!)
- University of Ottawa professor Robert Smith? publishes zombie essay collection (The Fulcrum)
- This book wants to eat your braaaiiinnnsss! (The uOttawa Gazette)
- CBC's All in a Day, promoting the launch of Braaaaiiinnnsss
- A profile of me for the "Math at Work" segment of the Canadian Mathematics Society webpage
- Zombie Zeitgeist (The Agenda, with Steve Paikin)
- Remember long division? (The Fulcrum)
- A documentary profile of me by Jes Ellacott (6 min)
- Predicting the zombie apocalypse (The Fulcrum)
- Immediate, aggressive spending could halt the AIDS epidemic (where the media was interested in my serious research for once)
- The Science of the Living Dead (with zombie luminaries George Romero and Max Brooks)
- An article about my question mark in The Toronto Star
- Hungry Beast (ABC TV, Australia)
- NPR zombie interview (with transcript)
- The Wall Street Journal interview (I still can't believe I get to type those words)
- BBC News (where it was the number 1 story for 24 hours; yes, it was a slow news week)



Forthcoming (September 2014)
Paper $49.95 CAD

Chapter 17: Baneling dynamics in Legend of the Seeker
by Gergely Röst