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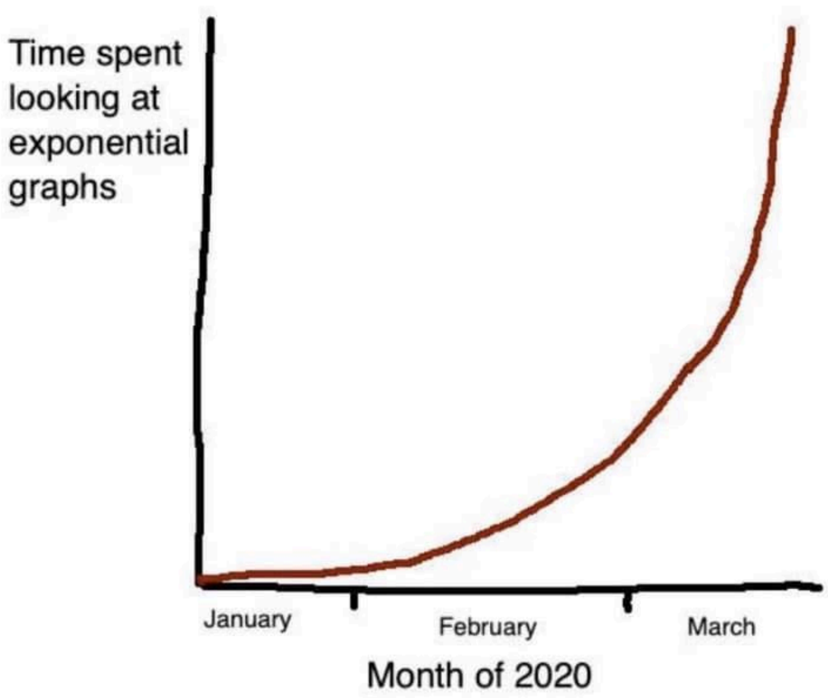
TÉCNICO LISBOA

Introduction to epidemic models



Network Science, 2021/2022

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Last class:
Resilience of complex networks
and cascading effects

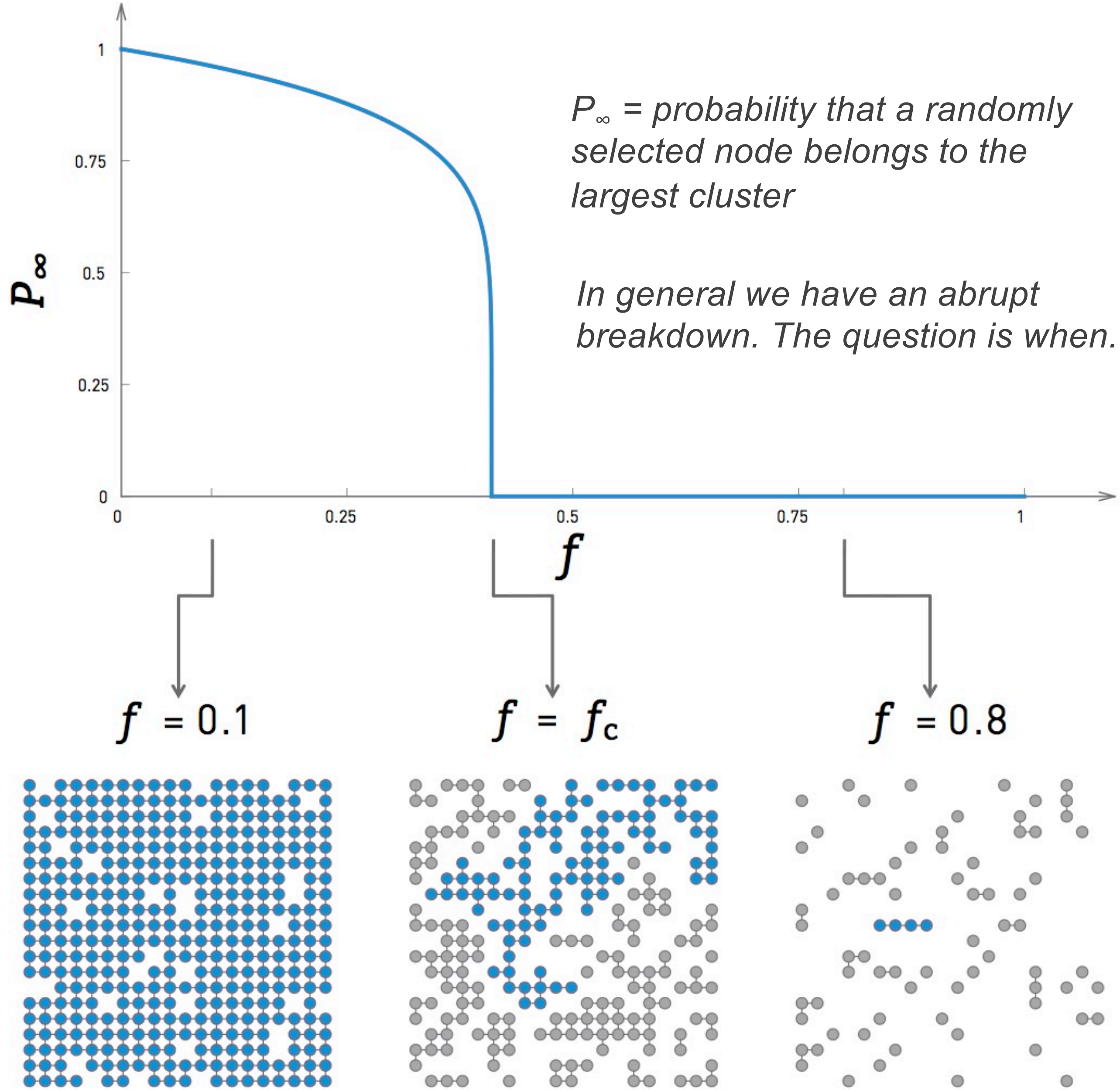
Network Science, 2021/2022

Robustness as an inverse percolation problem

Fraction of removed nodes:

$$f = 1 - p$$

e.g., the fraction of nodes that fail



$0 < f < f_c$:

There is a giant component.

$P_\infty \sim |f - f_c|^\beta$

$f = f_c$:

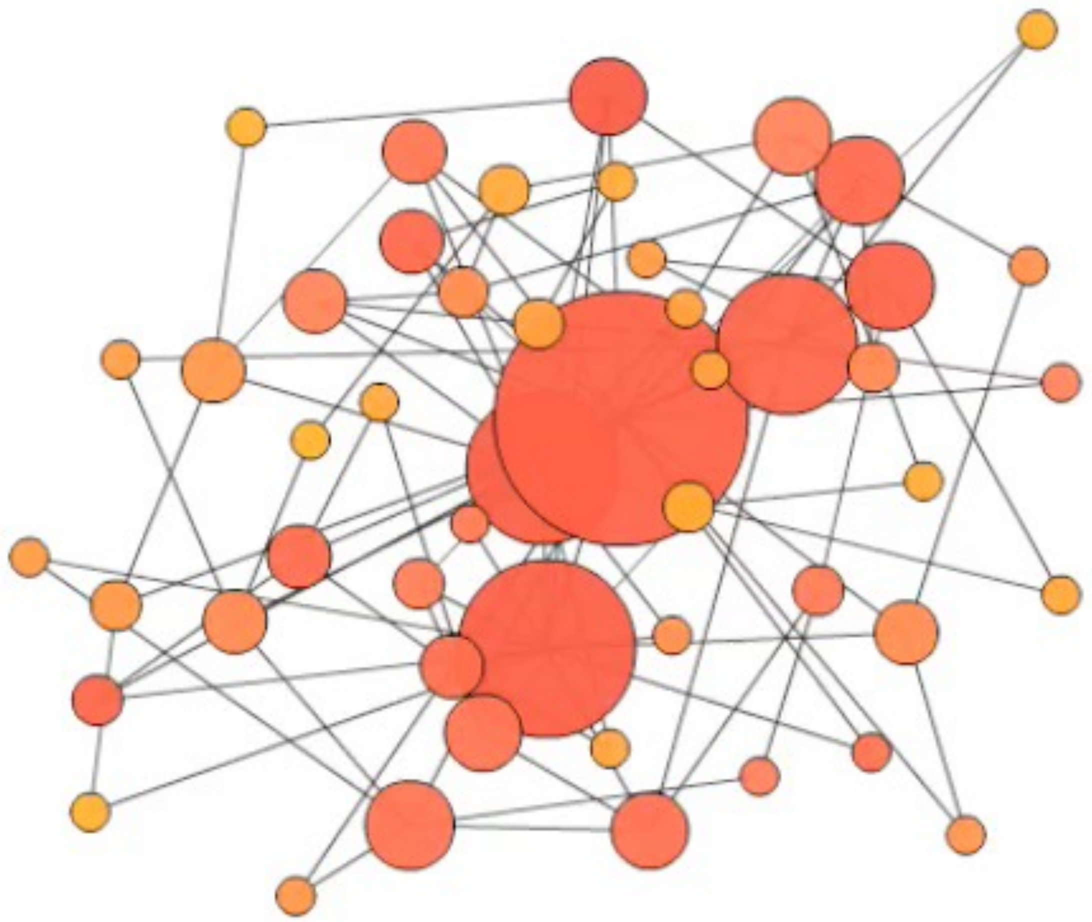
The giant component vanishes.

$f > f_c$:

The lattice breaks into many tiny components.

Scale-free networks under node failures

Randomly select and remove nodes, one by one.



How many nodes do we have to delete to fragment the network into isolated components?

Scale-free nets show an usual behavior: we must remove almost all of its nodes to destroy a given network.

Example of a scale-free network (BA model) under random node failure
Video by Dashun Wang

In general…

For details see for instance, Barrat et al. Dynamical processes in complex networks, 2008, section 6.4.

R Cohen et al.
Resilience of the Internet to random breakdowns,
Phys. Rev. Lett. (2000)

R Cohen

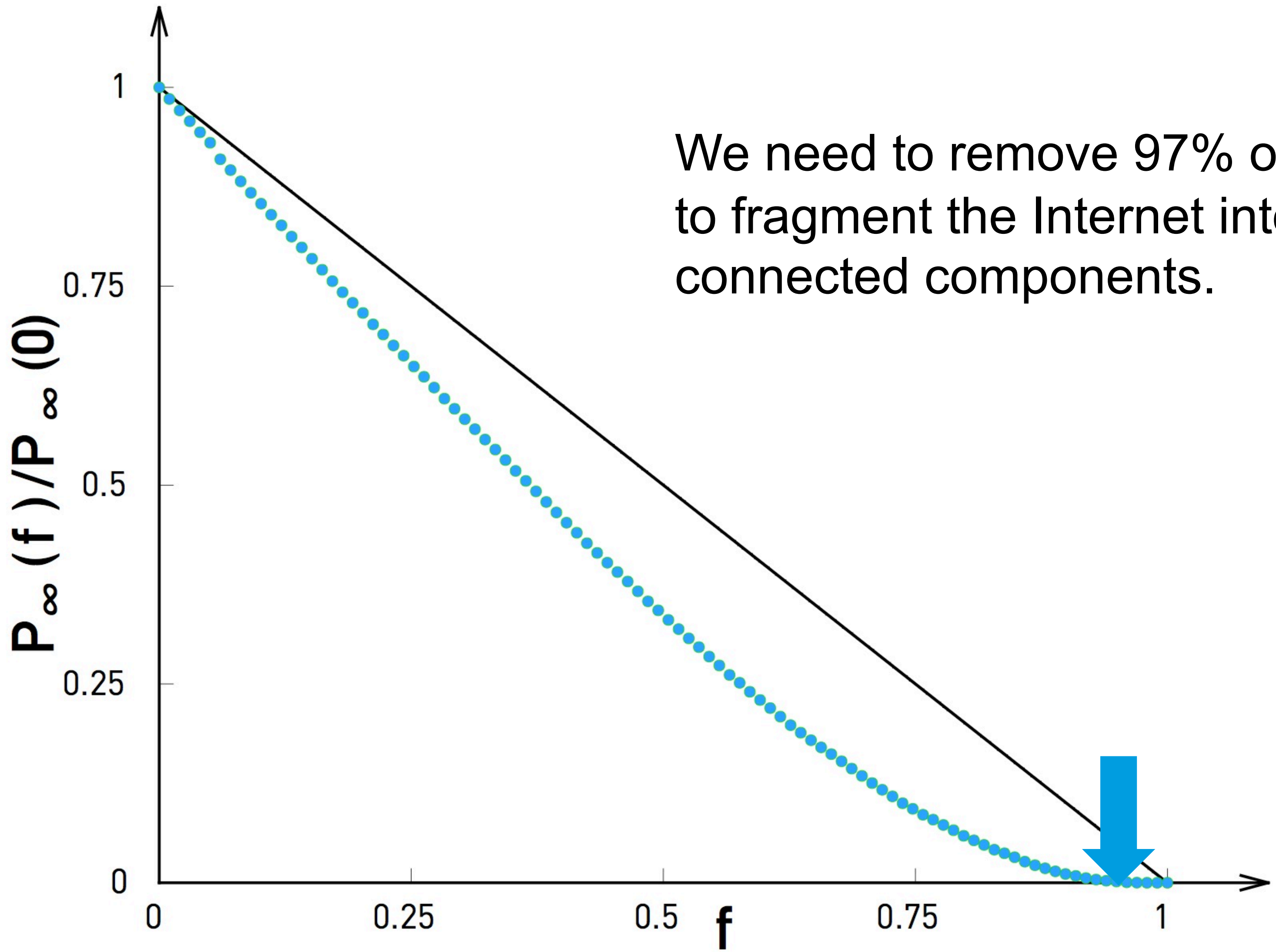
S Havlin

- From this, it can be shown that the giant component is present if the fraction f of removed nodes is

$$f < f_C = 1 - \frac{1}{\frac{\langle k^2 \rangle}{\langle k \rangle} - 1}$$

- As you may remember, in ***scale-free nets*** with $2 \leq \gamma < 3$ the second moment $\langle k^2 \rangle \to \infty$
- Thus, when $2 \leq \gamma < 3$, we get a giant component whenever ***p=1-f>0*** (or ***f<1***), i.e., **always**!!

Scale-free networks (Internet / Simulations)

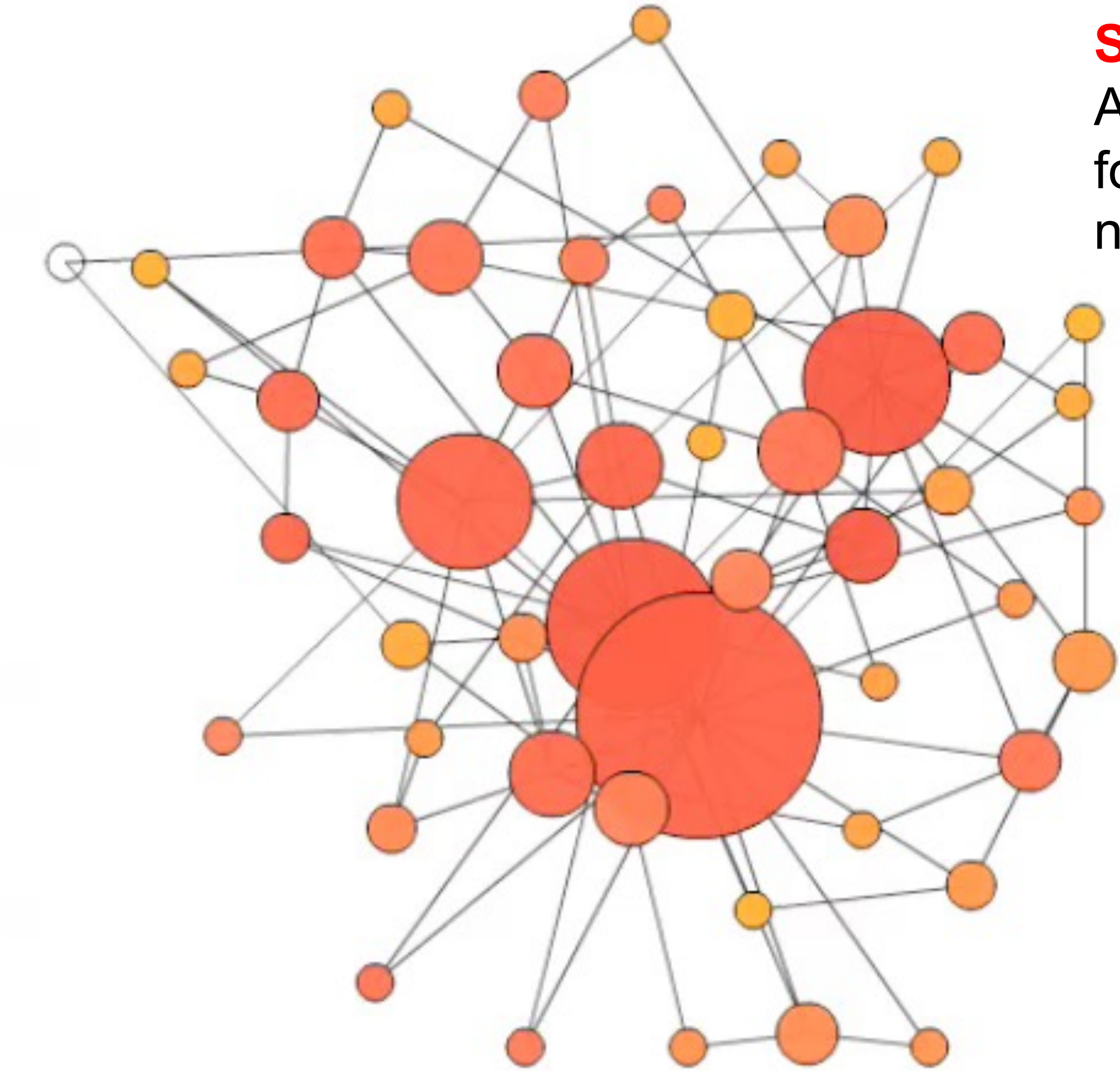


We need to remove 97% of the routers to fragment the Internet into dis-connected components.

Global contact networks

Air transportation network

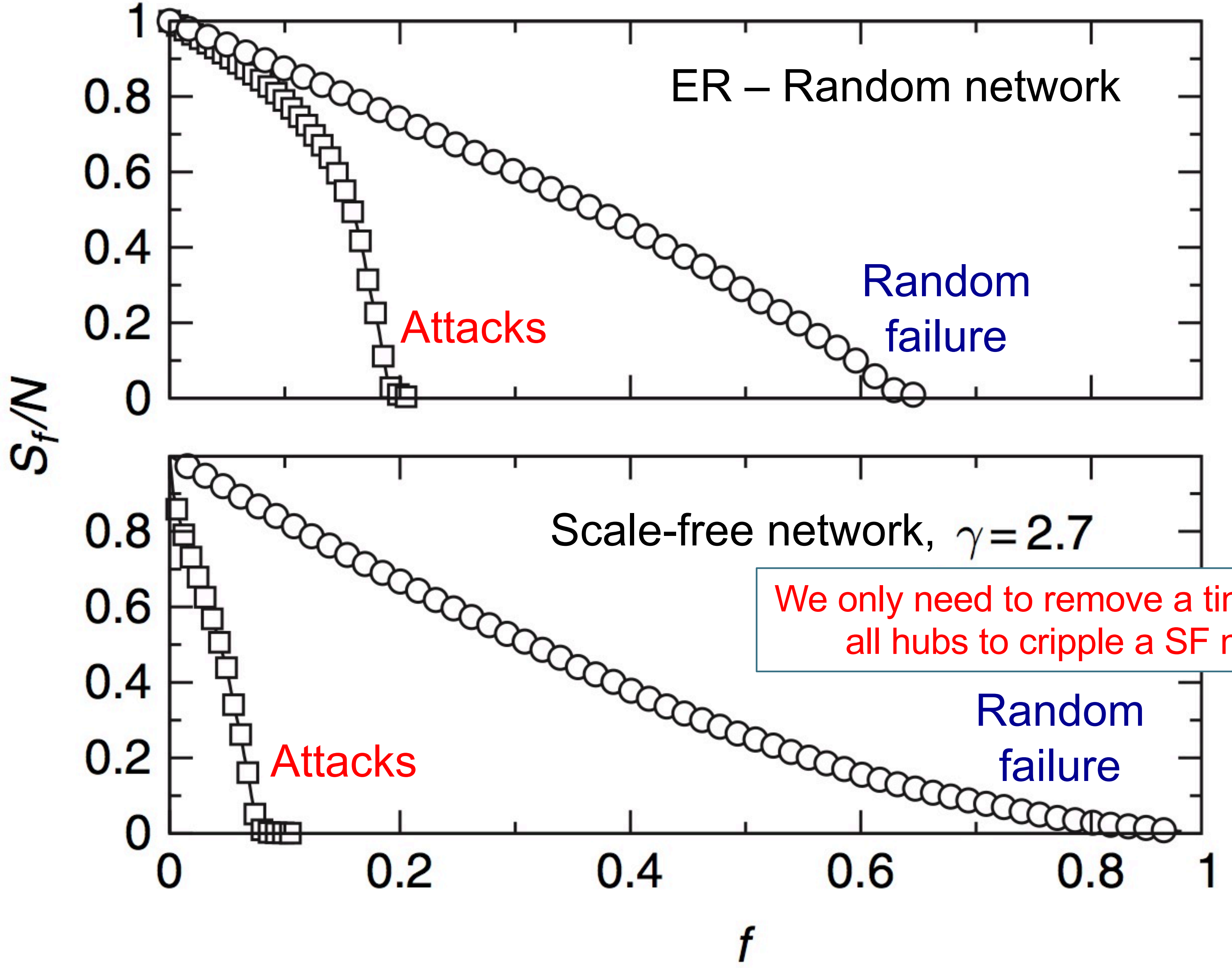
Achilles' heel of scale-free networks



Scale-free networks under attack
Attack first the highest-degree node, followed by the next highest degree node, and so on.

Video by Dashun Wang

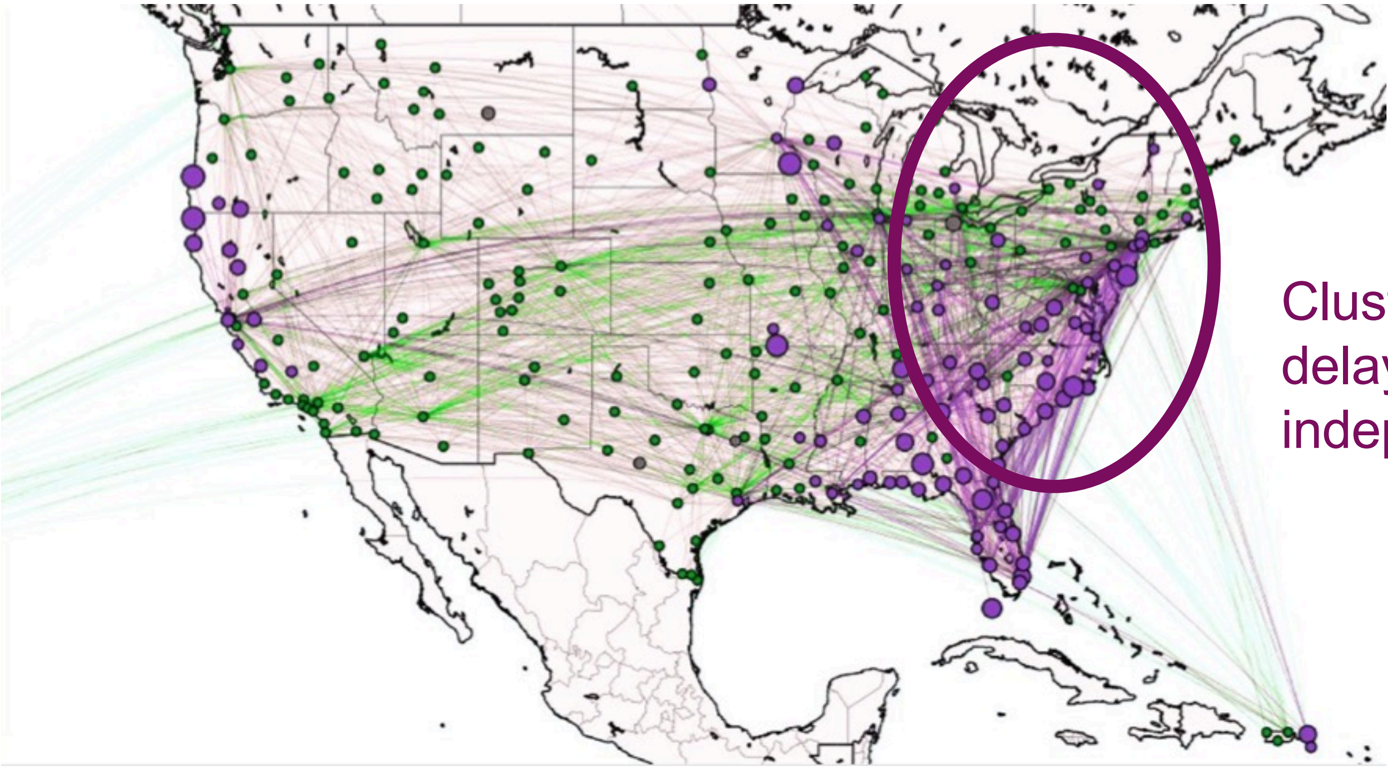
Robustness against targeted attacks



We only need to remove a tiny fraction of all hubs to cripple a SF network.

Do nodes of a network fail independently of each other!?

Flight delays in the U.S. have an economic impact of over $40 billion per year!!

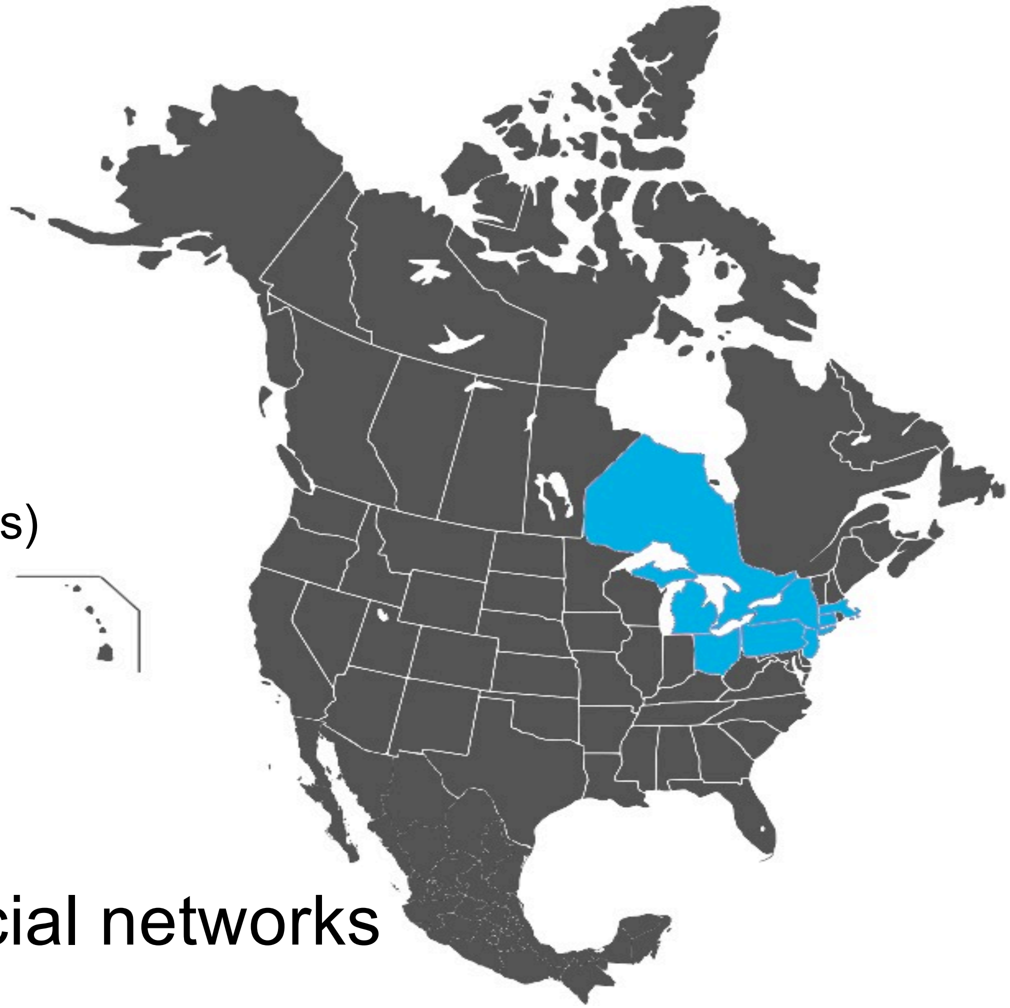


Clusters indicate that delays are not independent of each other

Cascading events & avalanches

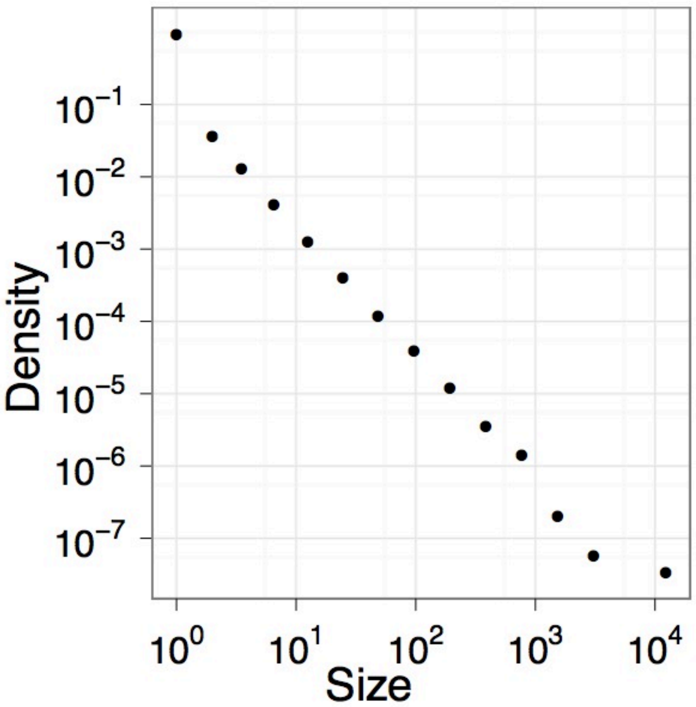
Nodes of a network fail independently of each other?

- Airports
- Power grids
- Internet (e.g. Denial of Service Attacks)
  C. Labovitz, A. Ahuja and F. Jahasian. Proc. of IEEE FTCS, Madison, WI, 1999
- Financial networks
  Haldane & May. Nature, 469: 351-355, 2011.
  Roukny, Bersini, et al. Sci. Rep., 3: 2759, 2013.
  Tedechi et al., PLoS One 7: e52749, 2012
- Information cascades in social networks



Information cascades in Twitter

The distribution of cascade sizes on Twitter. While most tweets go unnoticed, a tiny fraction of tweets are shared thousands of times.



Avalanche exponent: $\alpha \approx 1.75$

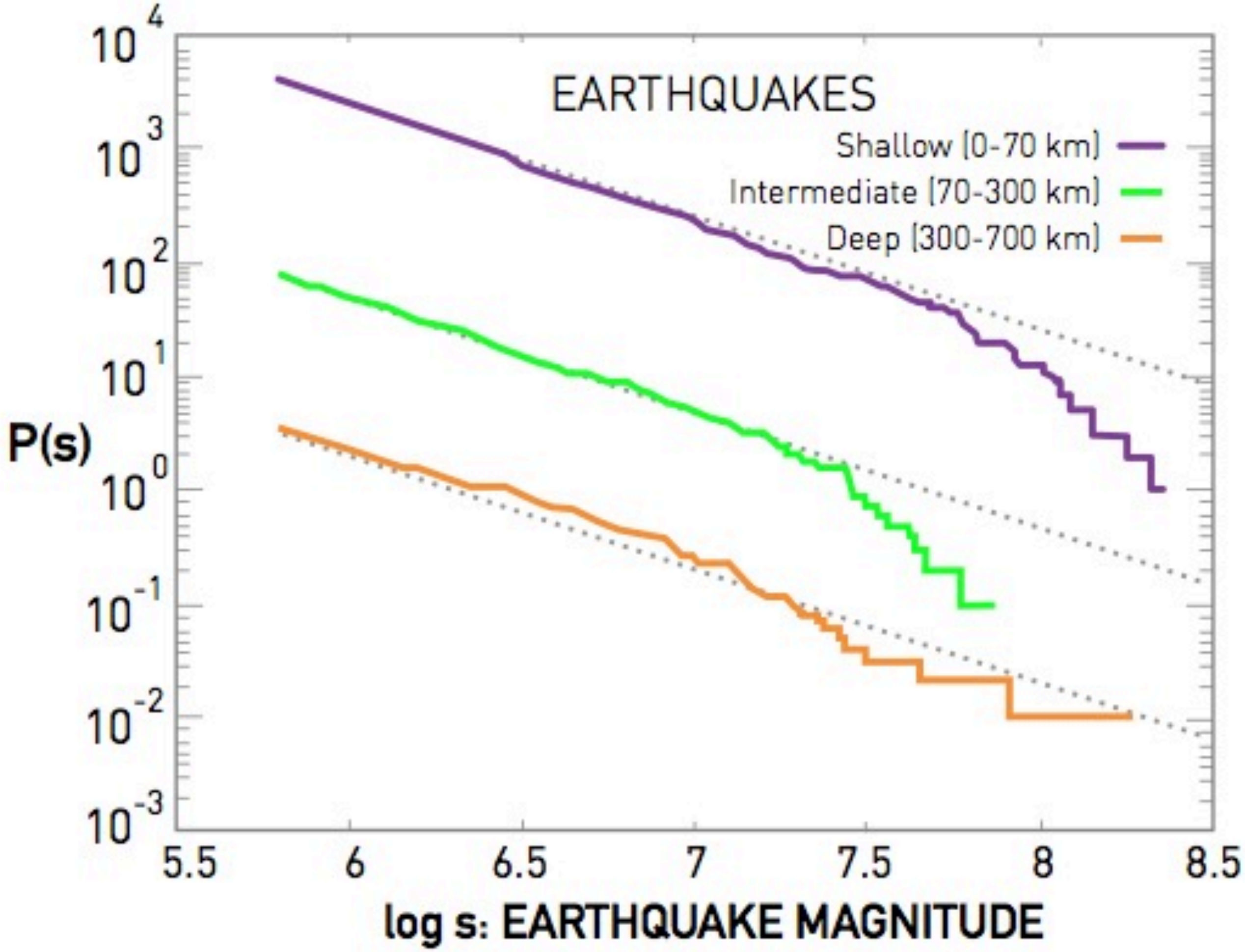
The power law indicates that the vast majority of posted URLs do not spread at all!!

Indeed, the average cascade size is only 1.14.

Yet, a small fraction of URLs are reposted thousands of times.

E. Bakshy, et al. ***Everyone's an influencer: quantifying influence on twitter.*** WSDM '11, 65-74, 2011.

Earthquakes magnitudes



Cascading events and power-laws

Avalanche exponents, from earthquakes and power failures, to twitter and flights delay, are surprisingly close (1.6-2.0)

SOURCE	EXPONENT	CASCADE
Power grid (North America)	2.0	Power
Power grid (Sweden)	1.6	Energy
Power grid (Norway)	1.7	Power
Power grid (New Zealand)	1.6	Energy
Power grid (China)	1.8	Energy
Twitter Cascades	1.75	Retweets
Earthquakes	1.67	Seismic Wave

Cascading events and power-laws

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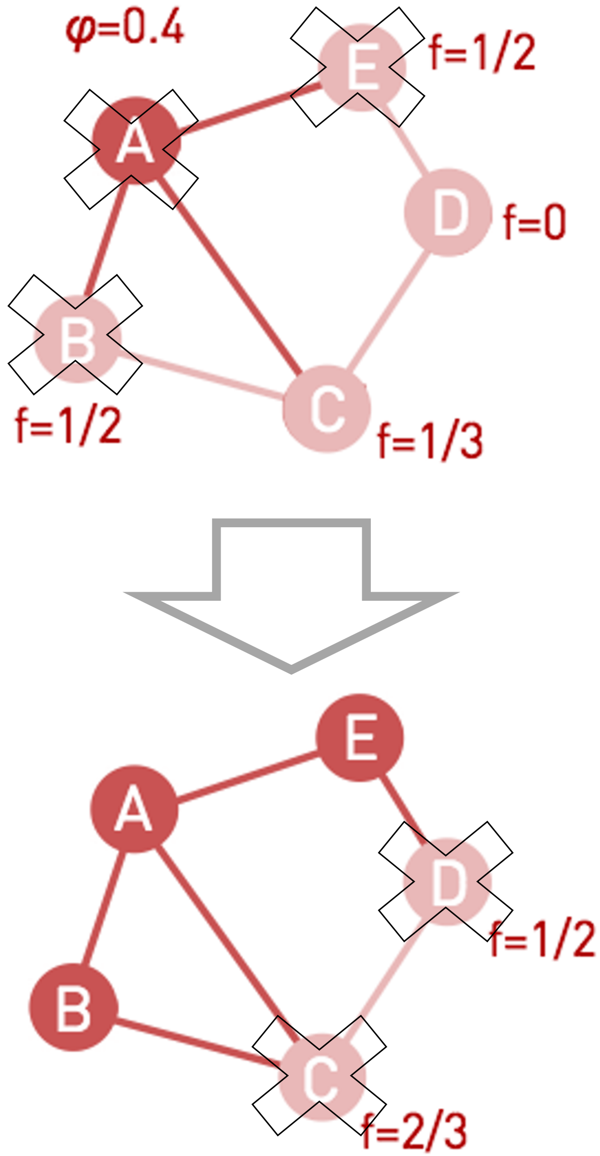
Can we create a model that helps us to explain the emergence of these properties?

A simple model for cascading events

- Consider a network of size N
- All nodes have a state initialized to “0” (healthy)
- At each time step, each node will turn “1” if at least a fraction φ of its neighbors is also “1” (i.e., have also failed).



Kong & Yeh, Resilience of degree-dependent and cascading node failures in random geometric networks, IEEE Transactions in Information Theory, 2010

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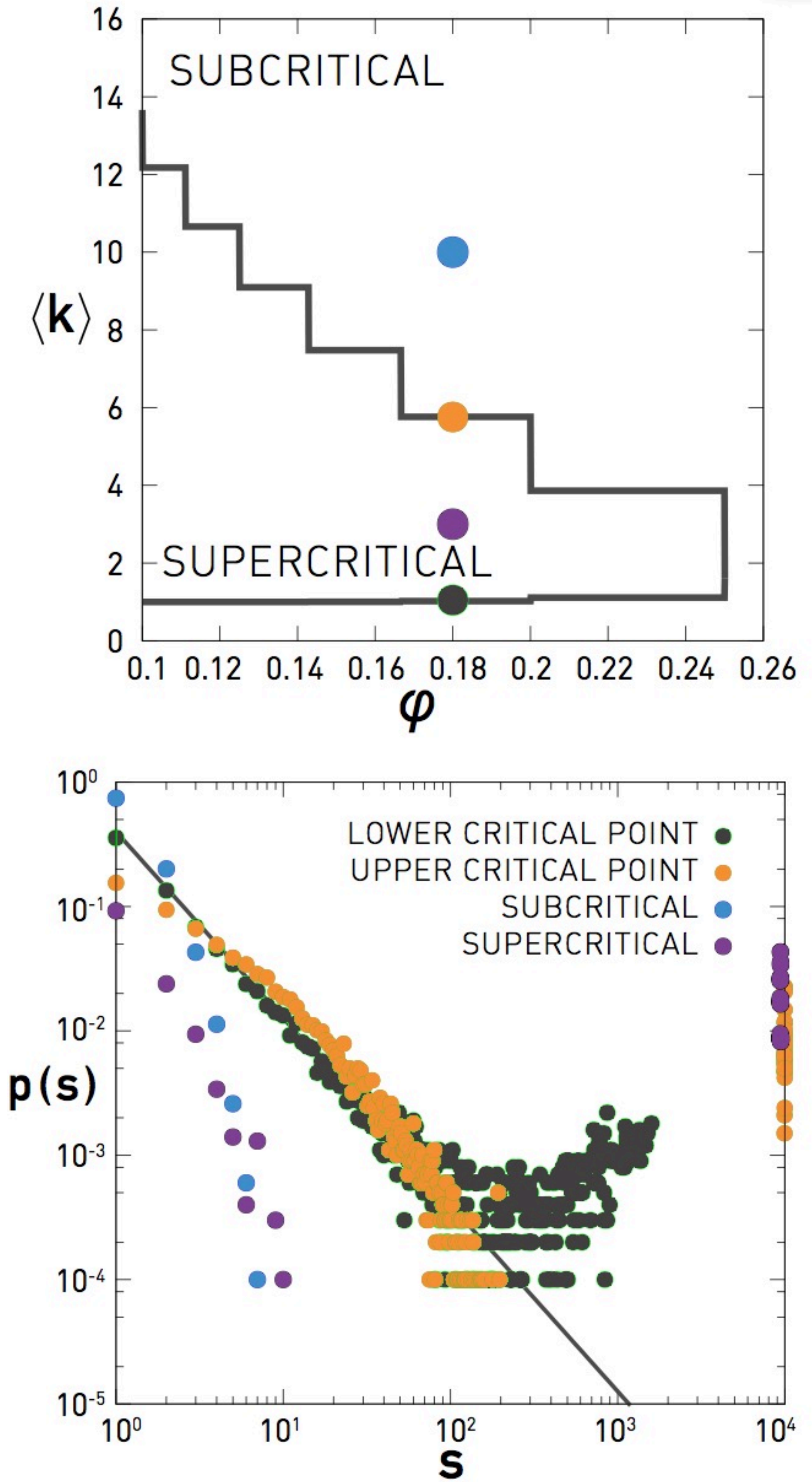


Kong & Yeh, Resilience of degree-dependent and cascading node failures in random geometric networks, IEEE Transactions in Information Theory, 2010

Motter, Lai, PRE 66 (6), 065102 (2002)
Motter, PRL 93(9), 098701 (2004)

Other example
Motter-Lai congestion model (2002)

- All nodes, at equal rate, send data packages to each other along shortest paths.
- Therefore, the permanent load of a node *i* is proportional to its betweeness centrality, $B_{0,i}$
- This model assumes that each node i has a limiting capacity given by

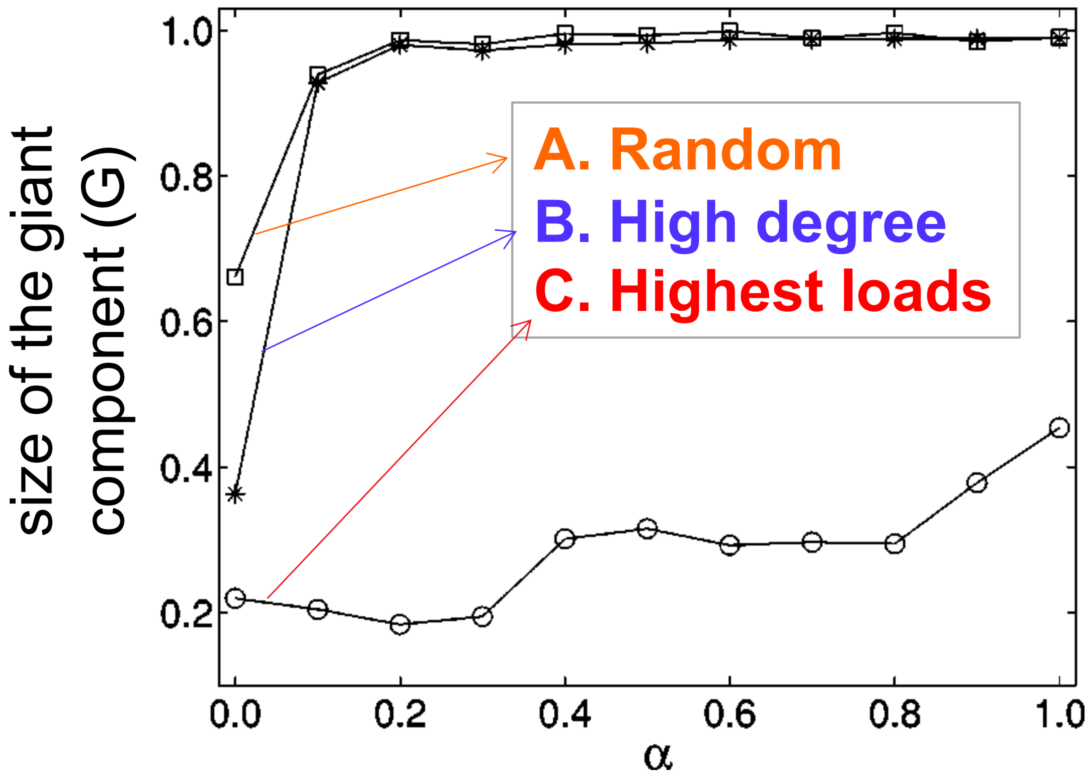
$$c_i = (1+\alpha) B_{0,i}$$

above this, the node colapses, leading to further collapses elsewhere.

Motter, Lai, PRE 66 (6), 065102 (2002)
Motter, PRL 93(9), 098701 (2004)

Other example
Motter-Lai congestion model (2002)

1. Compute all Betweeness centralities, $B_{0,i}$
2. Remove a given node using a given criterion: **A**, **B** or **C**.
3. Compute all B's and delete nodes with $B_i > (1+\alpha)B_{0,i}$
4. Repeat (3) until no overloaded nodes remain.



Degree-based attacks are surprisingly ineffective

Halting avalanches

Can we avoid cascading failures?

Reinforce the network after the first failure by adding new links? Does not work... The time needed to establish a new link is much larger than the timescale of a cascading failure (e.g.. financial and legal barriers, new transmission line on the power grid, etc.)

Can we reduce cascading failures through selective node and link removal (fighting fire with fire)?

Challenge: What is the most efficient strategy?

- Remove nodes with low or high loads?
- Remove links with low or large loads?

AE Motter, Cascade control and defense in complex networks, Phys Rev Lett 93(9), 098701 (2004)

What's next? How does the shape of our contact networks influence the spreading of information, diseases, human decisions, etc.?

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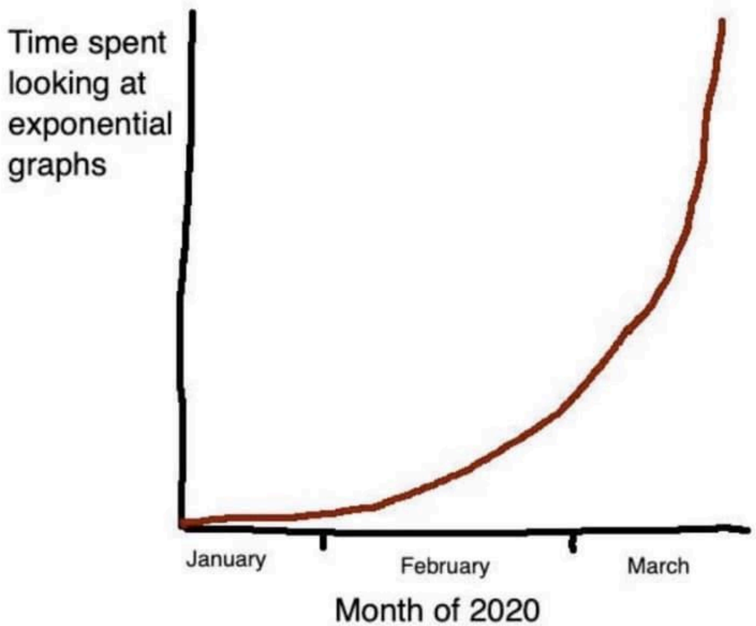
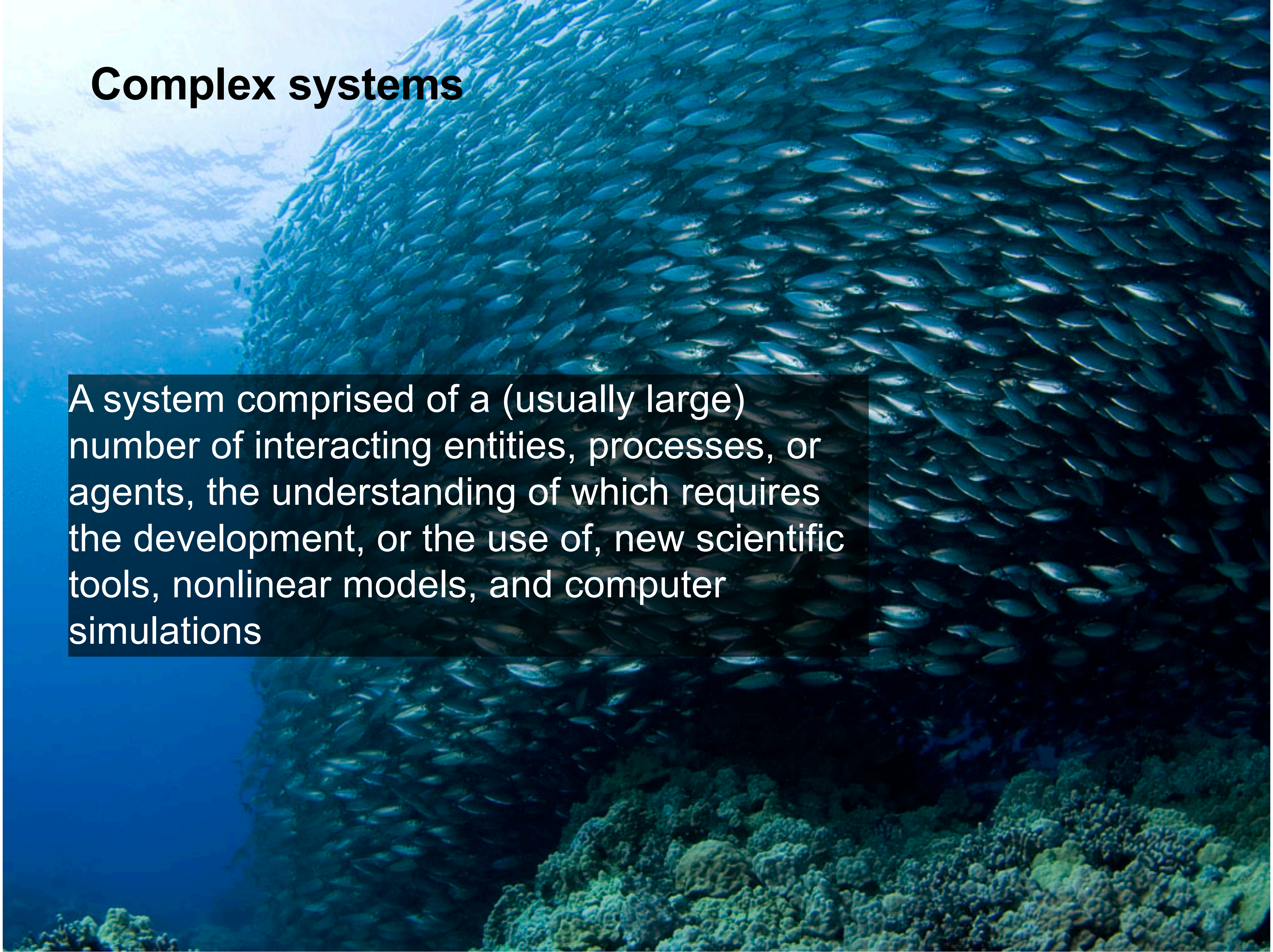
Introduction to epidemic models



Network Science, 2021/2022

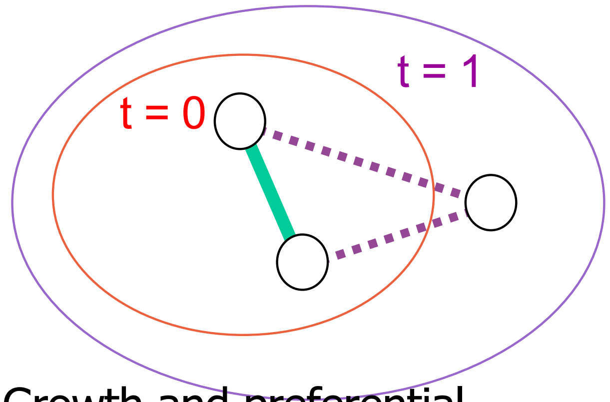


Complex systems

A system comprised of a (usually large) number of interacting entities, processes, or agents, the understanding of which requires the development, or the use of, new scientific tools, nonlinear models, and computer simulations

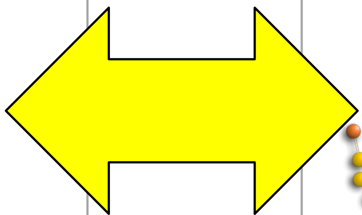
The ultimate goal of modelling complex systems

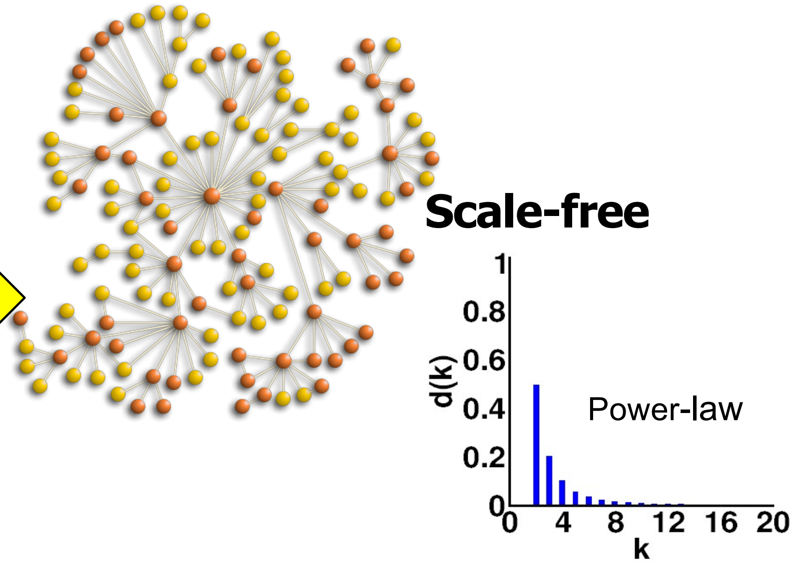
Ex: Scale-free networks



Growth and preferential attachment

Microscopic interactions



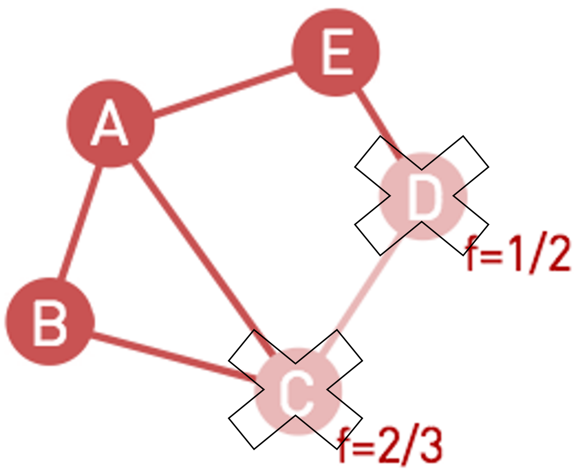


Emergent collective phenomena / properties

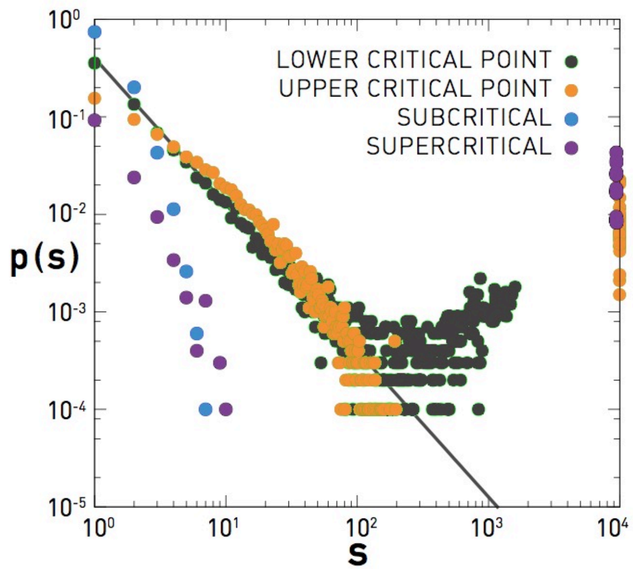
local ***global***

The ultimate goal of modelling complex systems

Ex: Cascading effects



Microscopic interactions



Emergent collective phenomena / properties

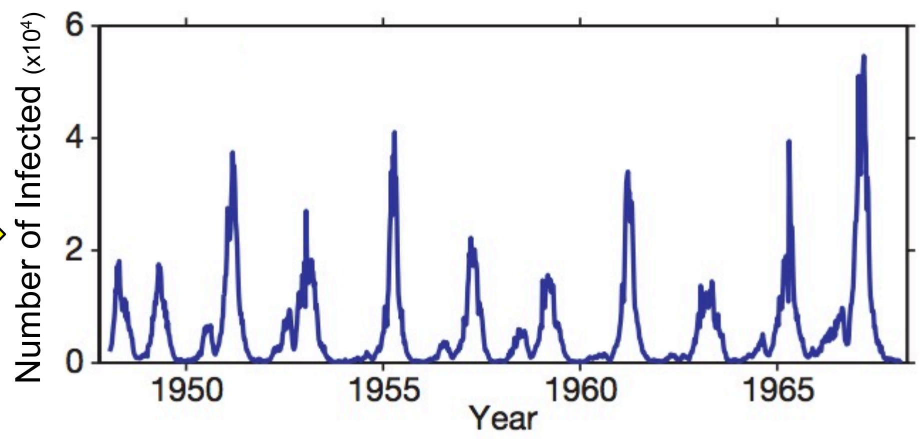
local

global

The ultimate goal of modelling complex systems

Ex: Disease spreading

Microscopic interactions

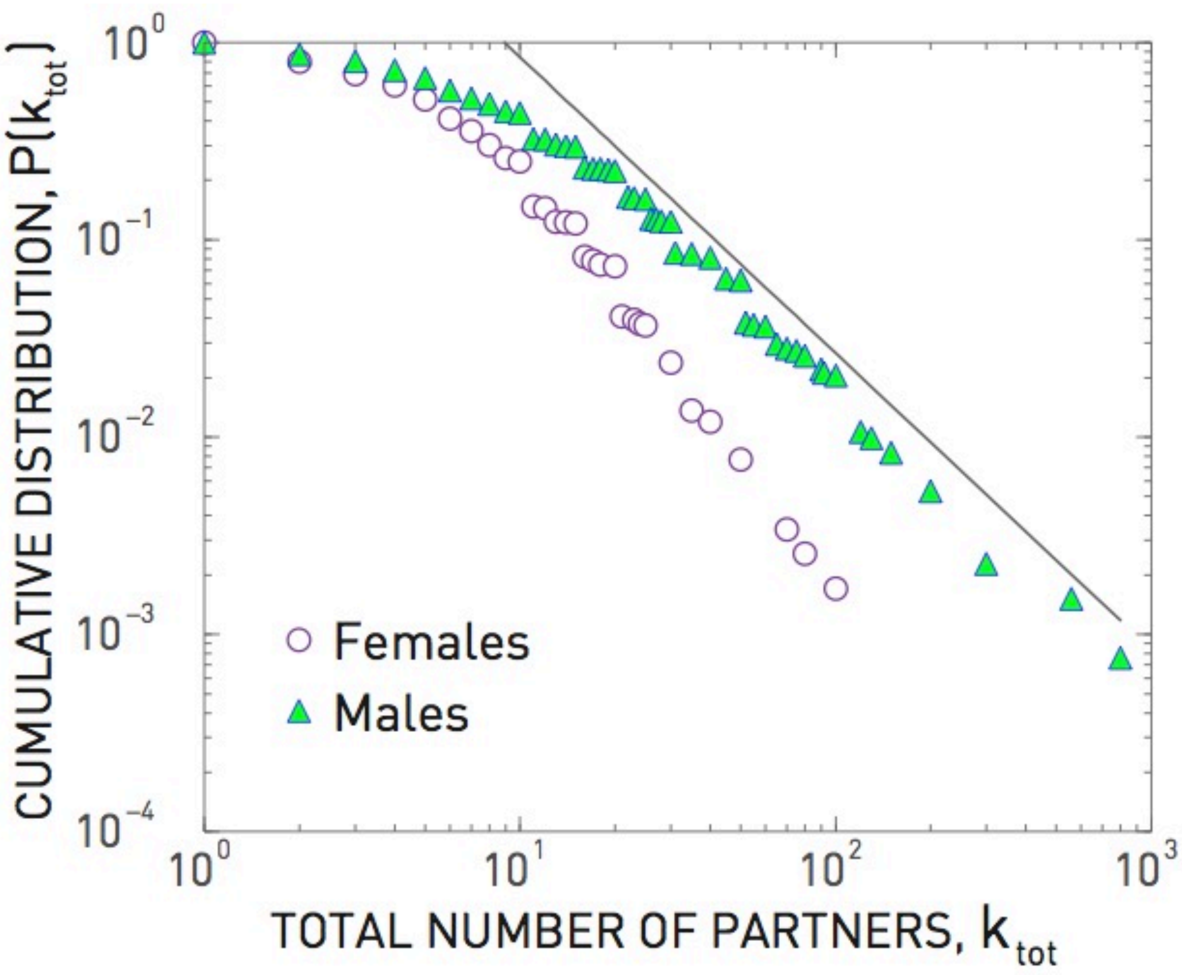


Emergent collective phenomena / properties

local

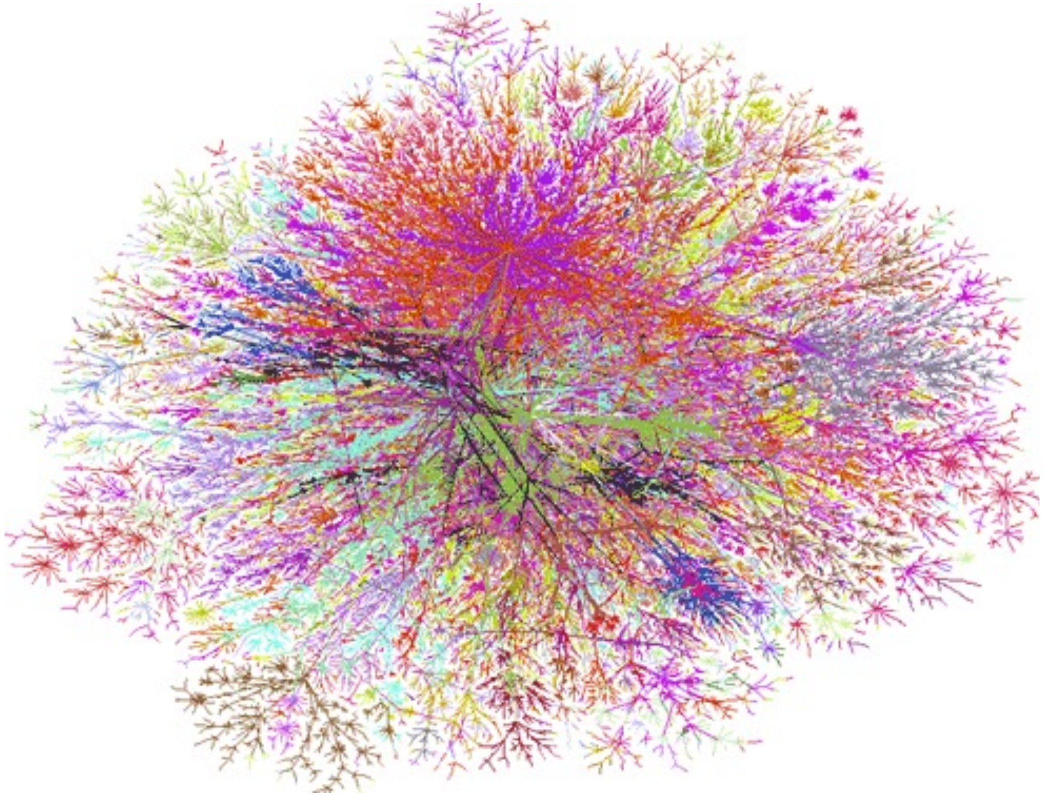
global

Sexually transmitted infections (STIs)

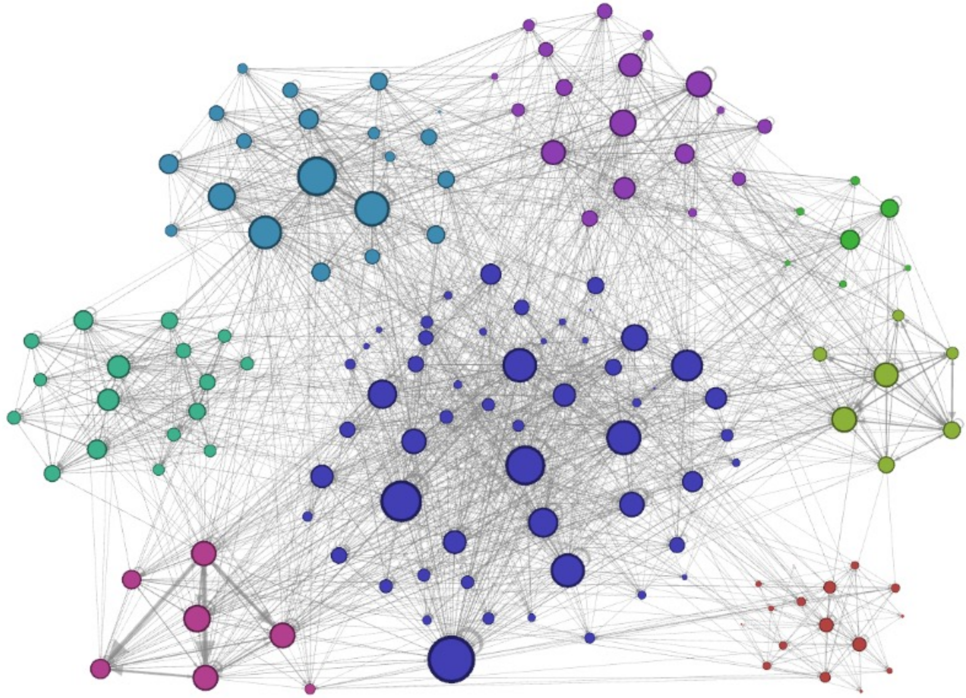


Computer viruses, mobile phone viruses, etc.

Internet

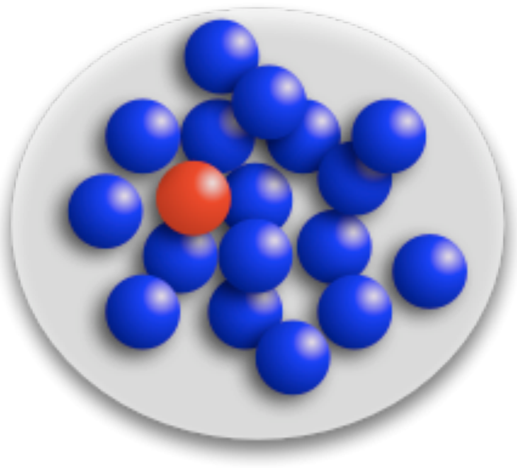


Email networks



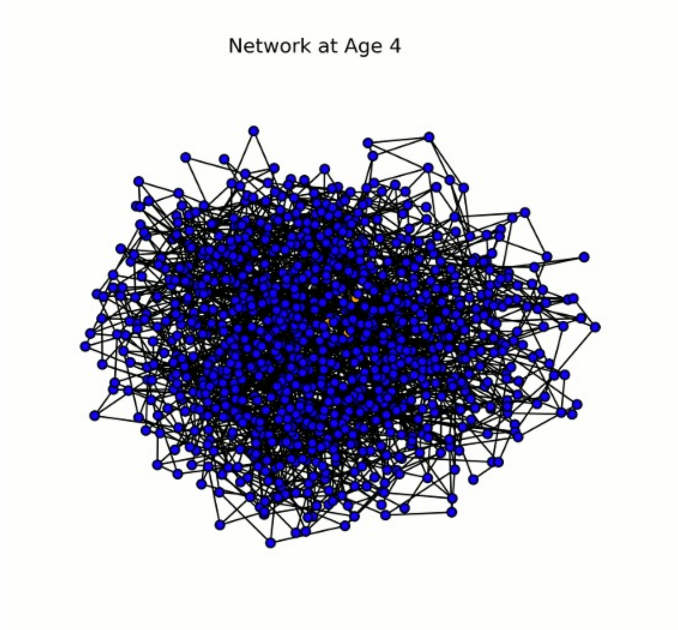
Synopsis

Part I



Introduction to theoretical epidemiology

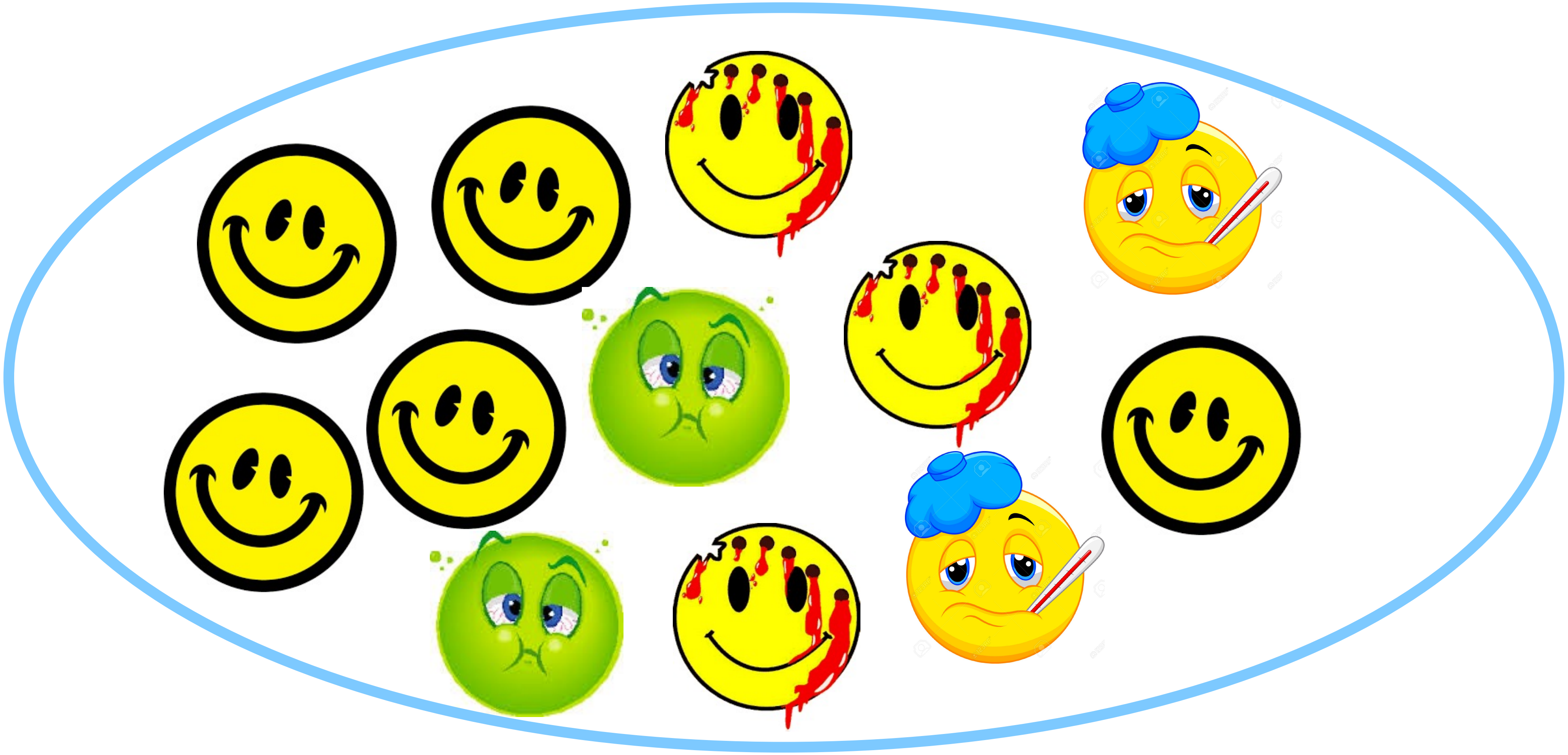
Part II



Network epidemics

Compartment models of disease spreading

Let's start from the basics. Consider a population where each individual has a disease state



Compartment models of disease spreading

Let’s start from the basics. Consider a population where each individual has a disease state

S

Susceptible
(healthy)

x

I

Infected

y

E

Exposed
(latency period)

w

R

Vaccinated
or Recovered

z

(…)

Compartment models of disease spreading

Populations are large and “well-mixed”

- anyone can potentially interact with anyone else (a.k.a., “mean-field” approach)
- all individuals in the same state are equivalent;
- the parameters defining the population dynamics are provided by the fraction of individuals that are in a given state.

x y w z (…)

Compartment models of disease spreading

G. McKendrick (Scottish military physician)
W. O. Kermack (Scottish biochemist)
(key papers: 1927, 1932, and 1933)

x y w z

(...)

Compartment models of disease spreading

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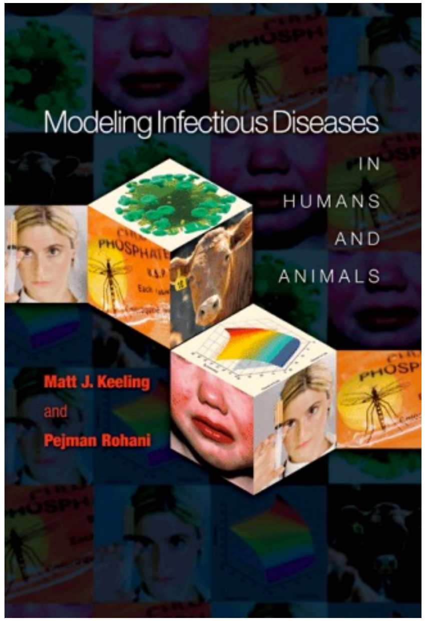
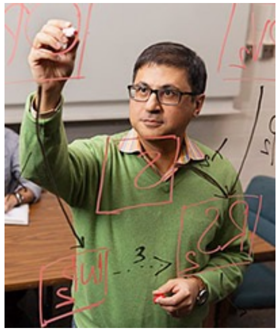


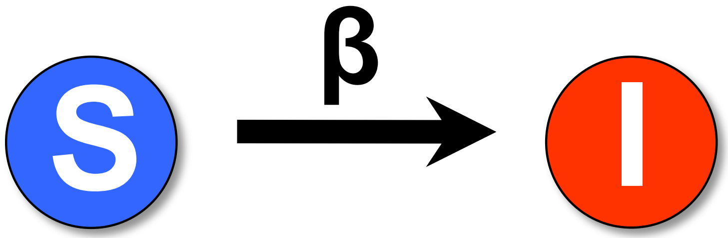
Modeling Infectious Diseases in Humans and Animals
By Matt J. Keeling & Pejman Rohani
Princeton University Press, 2007



Traditional models

β: contact infection rate

SI model

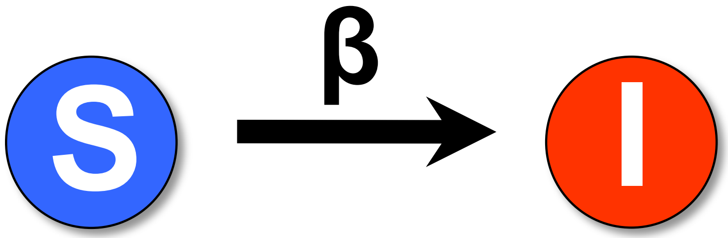


Ex: AIDS

Traditional models

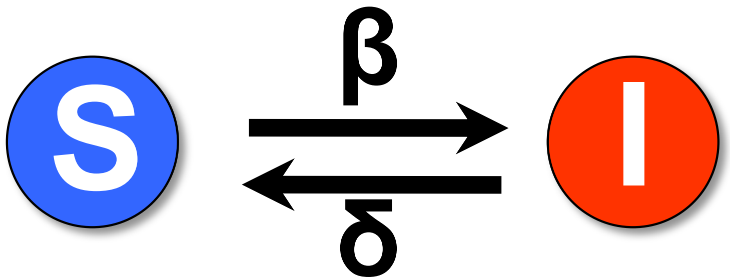
β: contact infection rate
δ: recovery rate

SI model



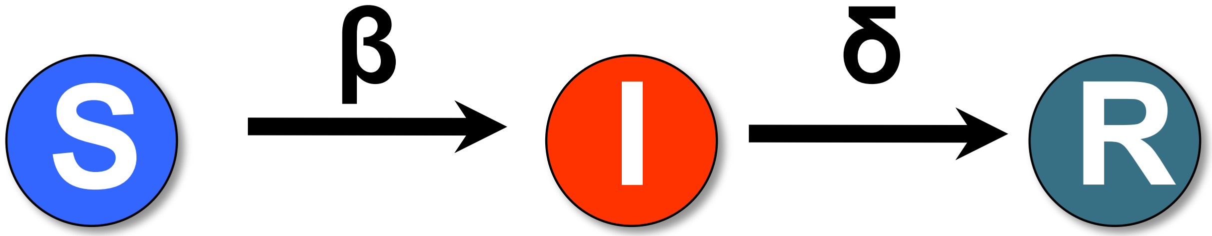
Ex: AIDS

SIS model



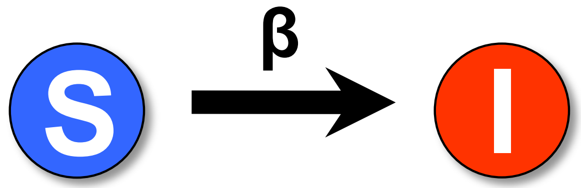
Ex: gonorrhea, athlete's foot, …

SIR model



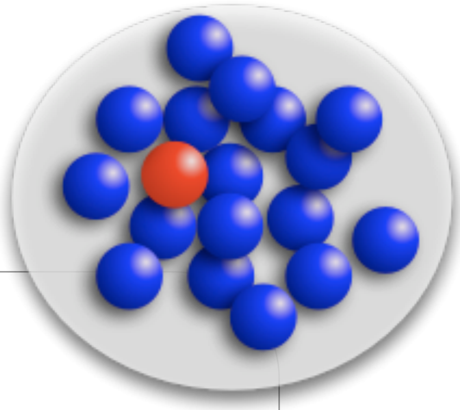
Ex: single season flu

Building a model (ex: SI model)



SI model

Assumptions:
N = population size (large number)
Everyone is equally likely to interact with everyone else.
S(t) susceptible and I(t) infected individuals at time t.

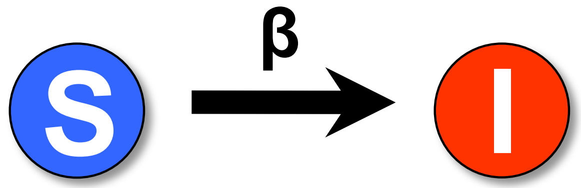


Goal:

Let us say that at time t=0 everyone is susceptible (S(0)=N) and no one is infected (I(0)=0).

If a single individual becomes infected at time *t=0* (i.e. *I*(0)=1), how many individuals will be infected at some later time *t*?

Building a model (ex: SI model)



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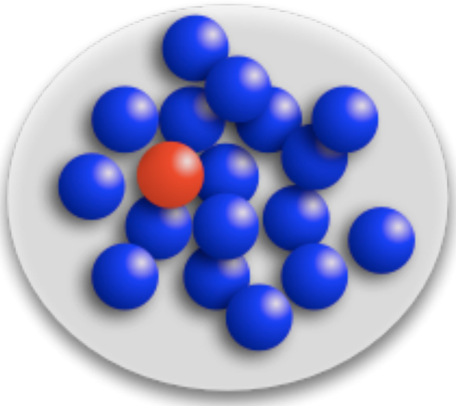
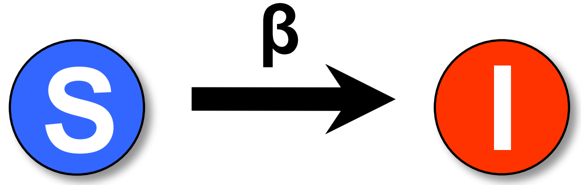


The probability that an infected person encounters a susceptible in a random interaction is

$$\frac{S(t)}{N}$$

Building a model (ex: SI model)



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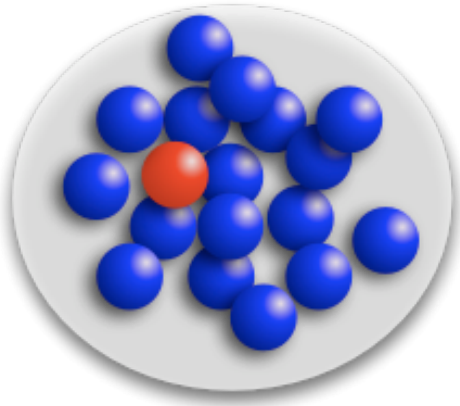
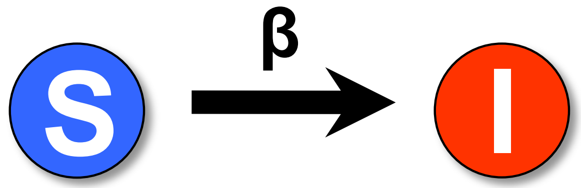


If everyone interacts <k> times at each time-step, we get that each infected encounters (on average)

$$\langle k \rangle \frac{S(t)}{N}$$

susceptible individuals

Building a model (ex: SI model)



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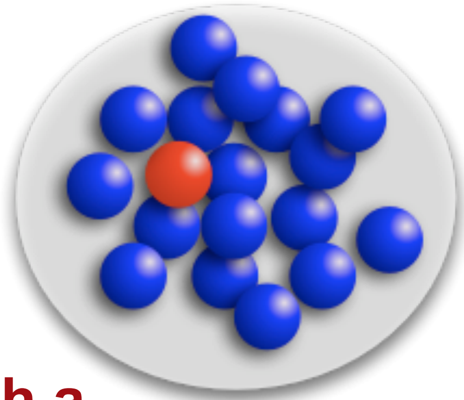
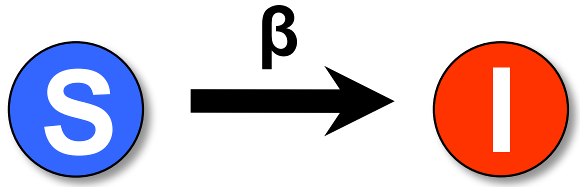


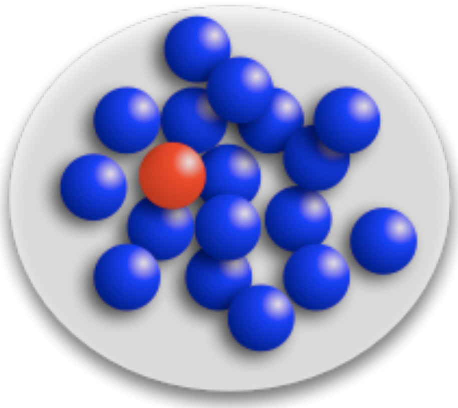
If each of the I(t) infected transmits the disease with a rate β, the number of new infected individuals at each time-step is

$$I(t)\beta\langle k\rangle \frac{S(t)}{N}$$

Building a model (ex: SI model)



SI model

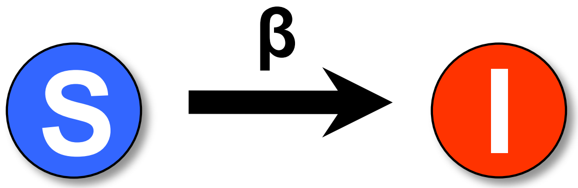


Assumptions:
N = population size (large number)
Everyone is equally likely to interact with everyone else.
S(t) susceptible and I(t) infected individuals at time t.

- **Therefore, the change in I(t) is given by**

$$I(t+1) - I(t) = I(t)\beta\langle k\rangle\frac{S(t)}{N}$$

Building a model (ex: SI model)



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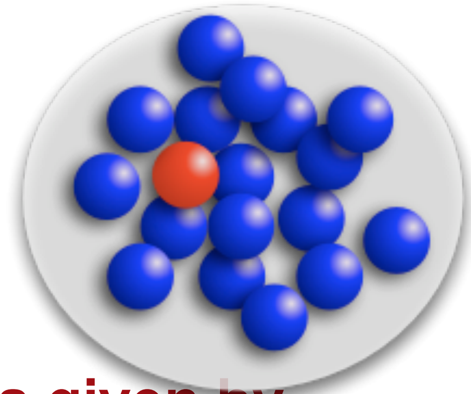
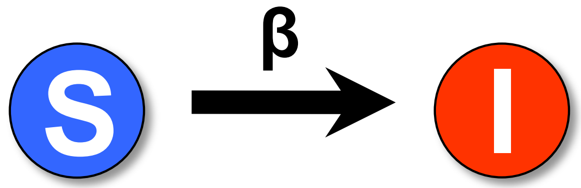


..or, in the other words, the rate of change dI(t)/dt is given by

$$\frac{dI(t)}{dt} = I(t)\beta\langle k\rangle\frac{S(t)}{N}$$

Building a model (ex: SI model)



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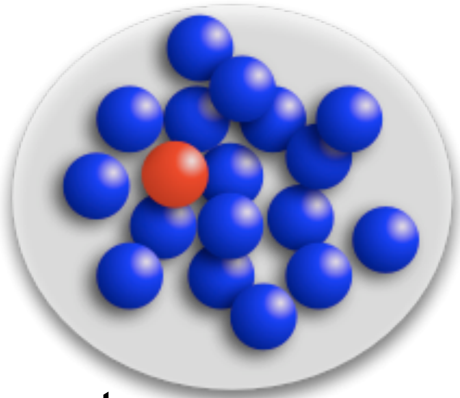
Everyone is equally likely to interact with everyone else.
S(t) susceptible and I(t) infected individuals at time t.



Using the frequencies $x=I(t)/N$ and $y=S(t)/N=1-x$ we get

$$\frac{dx}{dt} = x(1-x)\beta\langle k\rangle$$

SI model

β = infection rate
<k> = average number of contacts of a given individual
x = fraction of infected in the population
y = 1-x = fraction of susceptible

$$S \xrightarrow{\beta} I$$

$$\frac{dx}{dt} = x(1-x)\boxed{\beta\langle k\rangle}$$

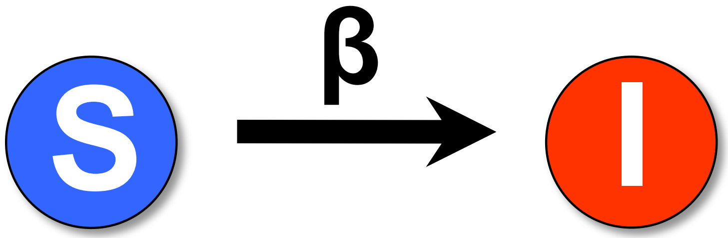
Effective spreading rate = β<k>

i.e., an infected individual is able to transmit the disease with β<k> others per unit time. Or, if you prefer, the characteristic timescale of the disease is

$$\tau = \left(\beta\langle k\rangle\right)^{-1}$$

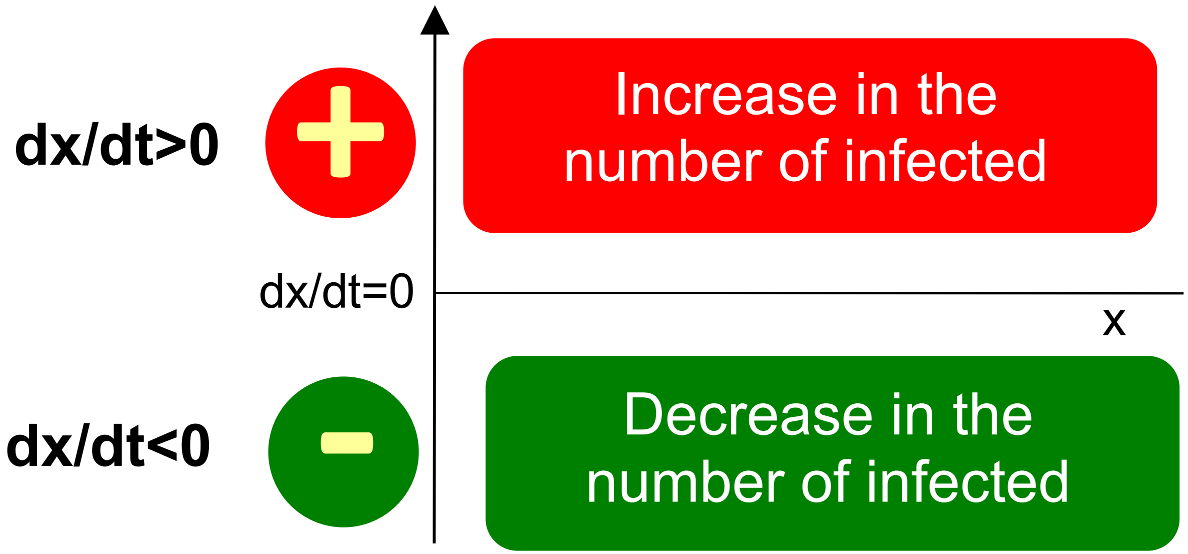
SI model

β = infection rate
<k> = average number of contacts of a given individual
x = fraction of infected in the population
y = 1-x = fraction of susceptible



$$\frac{dx}{dt} = x(1-x)\beta\langle k\rangle$$

Gradient of infection:



SI model

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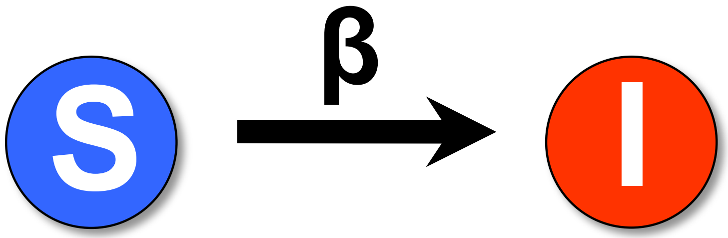

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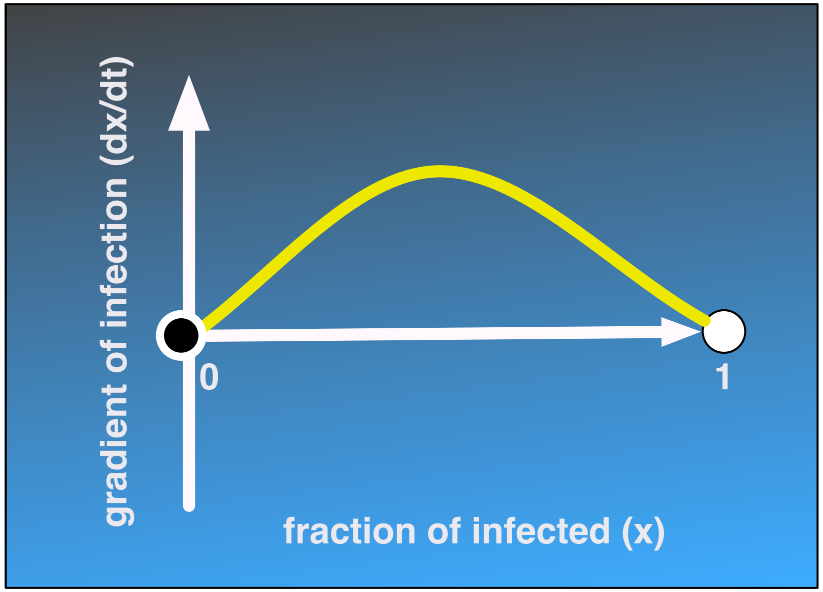

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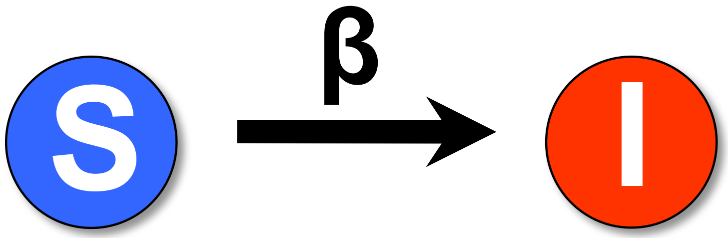

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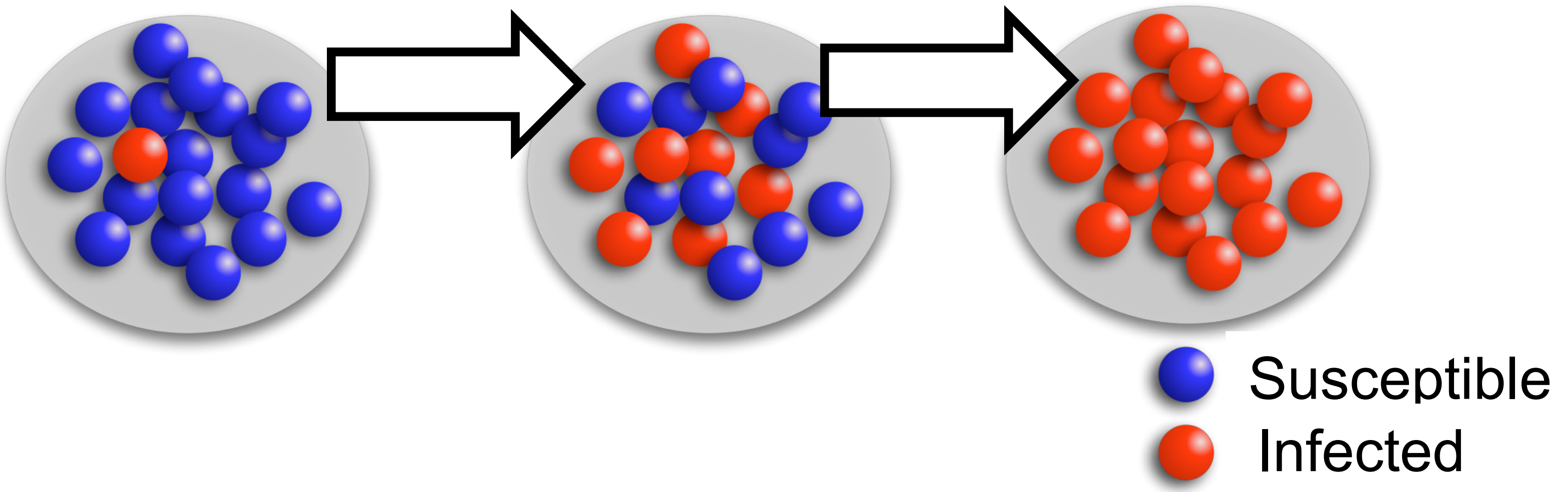

SI model

β = infection rate
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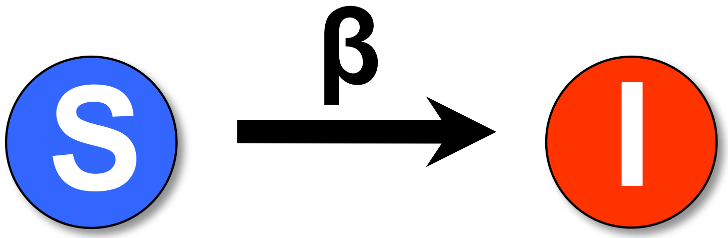


$$\frac{dx}{dt} = x(1-x)\beta\langle k\rangle$$

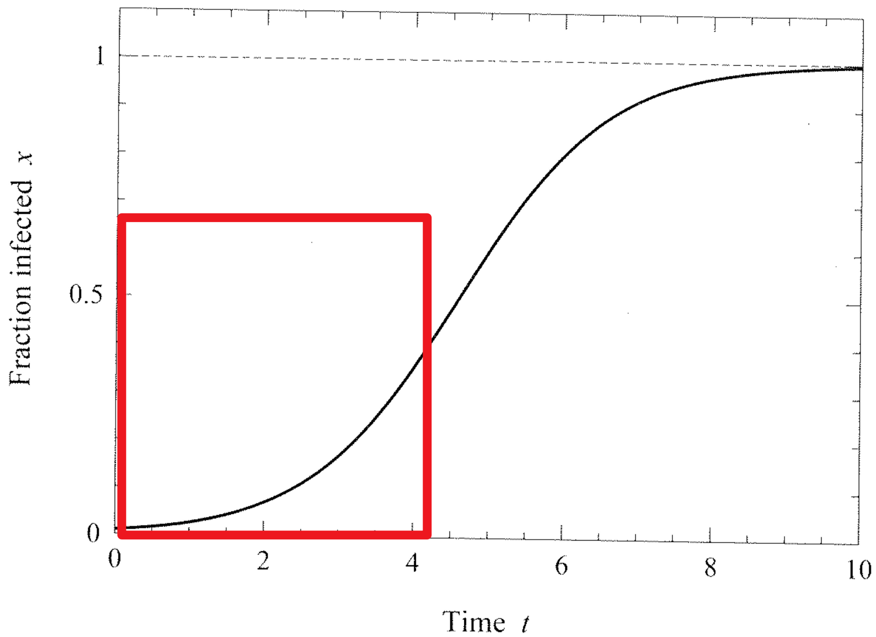
$$\tau = \left(\beta\langle k\rangle\right)^{-1}$$

$$x(t) = \frac{x_0 e^{t/\tau}}{1 - x_0 - x_0 e^{t/\tau}}$$

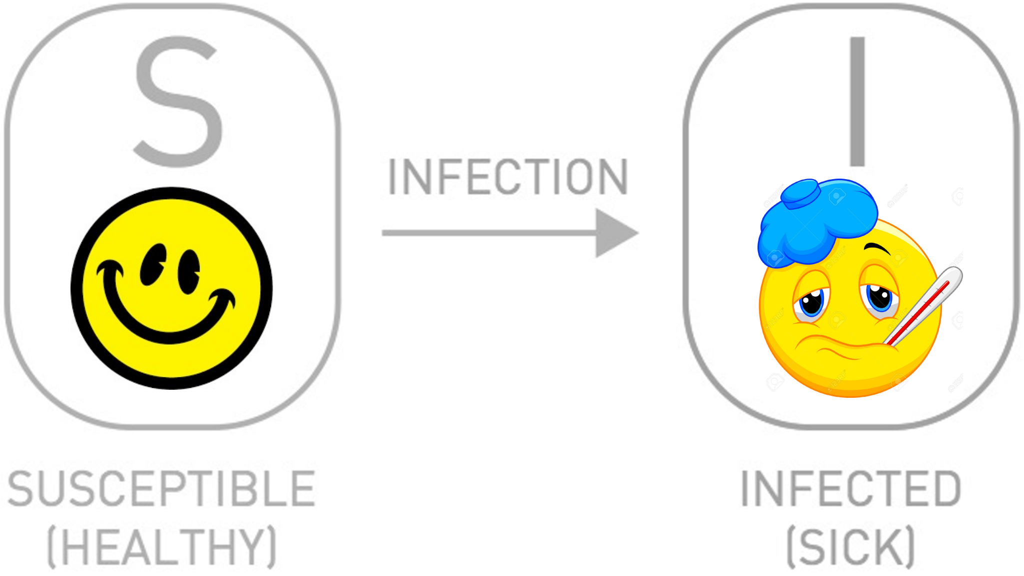
$$x(t) << 1$$

$$x(t) \approx x_0 e^{t/\tau}$$

Logistic curve:



SI model

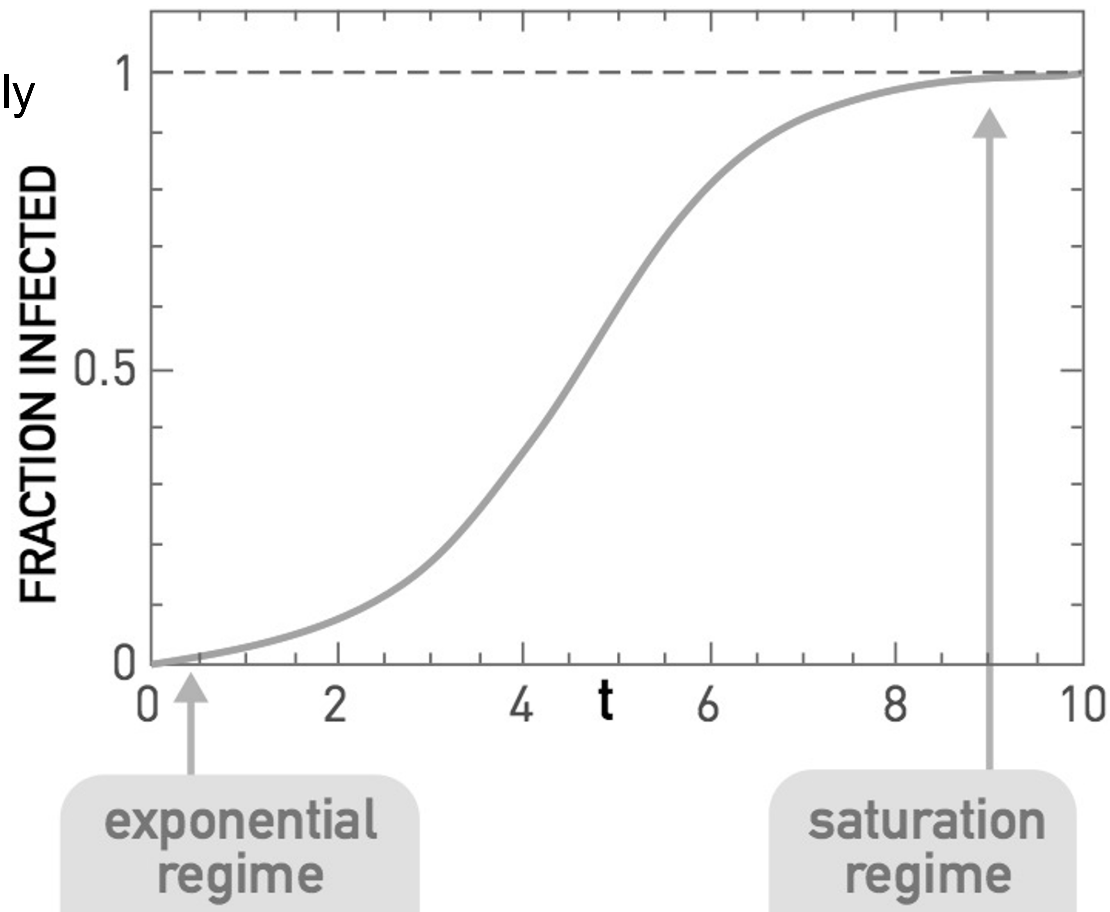


What are we missing here?

In some diseases pathogens are eventually defeated by the immune system.

Thus we need to consider the possibility that individuals recover, stopping to spread the disease.

Thus, we need an extra parameter:
The rate of recovery: **δ**



Traditional models

β: contact infection rate
δ: recovery rate

SIS model

$$S \underset{\delta}{\overset{\beta}{\rightleftarrows}} I$$

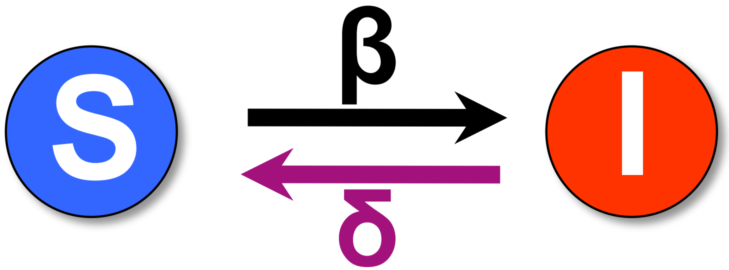
$$\dot{x} \equiv {}^{dx}\!/_{dt} = \underbrace{x(1-x)\beta\langle k\rangle}_{\text{infection}} \underbrace{-\,\delta x}_{\text{recovery}}$$

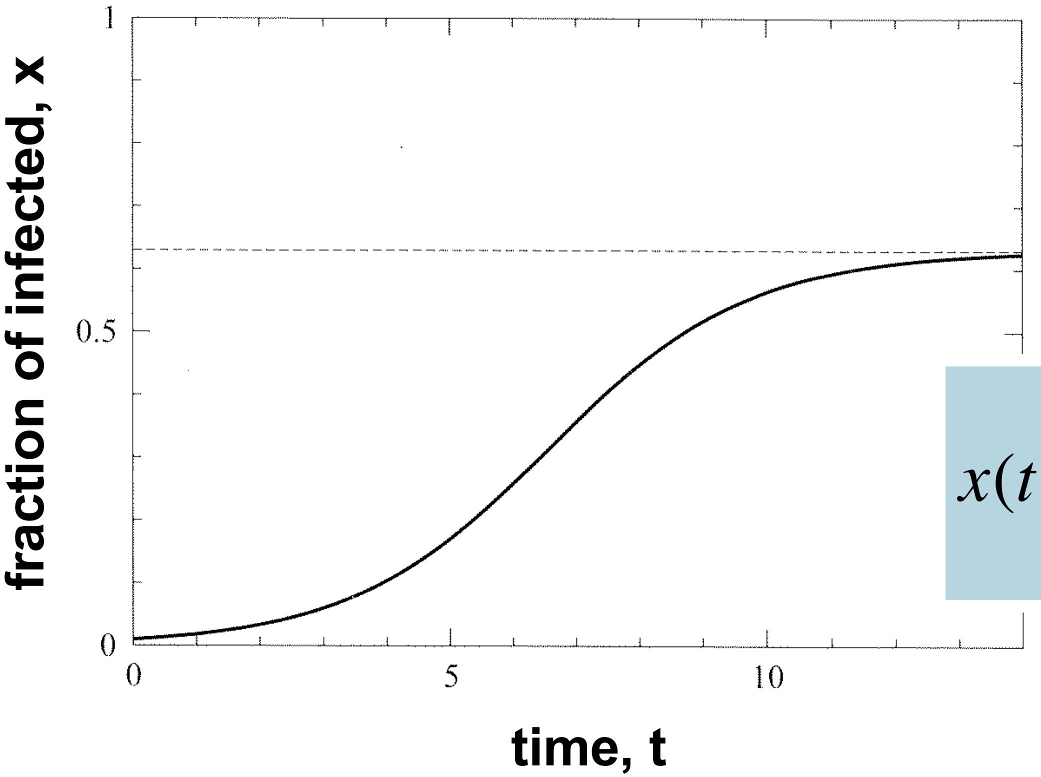
<k> – average number of contacts of a given individual
x – fraction of infected in the population
y = 1-x – fraction of susceptible

Traditional models

β: contact infection rate
δ: recovery rate

SIS model





Endemic disease

$$x(t) = \left(1 - \frac{\delta}{\beta\langle k\rangle}\right)\frac{Ce^{t/\tau}}{1 + Ce^{t/\tau}}$$

$$\tau = \left(\beta\langle k\rangle - \delta\right)^{-1}$$

Traditional models

β: contact infection rate
δ: recovery rate

SIS model

$$S \underset{\delta}{\overset{\beta}{\rightleftharpoons}} I$$

- Endemic state:

  For low recovery rate, the disease will never disappear.

$$\delta < \beta \langle k \rangle$$

- Disease free-state:

  For large recovery rates the number of new infections will be lower than the number of new recovered individuals, and the disease decreases exponentially in time.

$$\delta > \beta \langle k \rangle$$

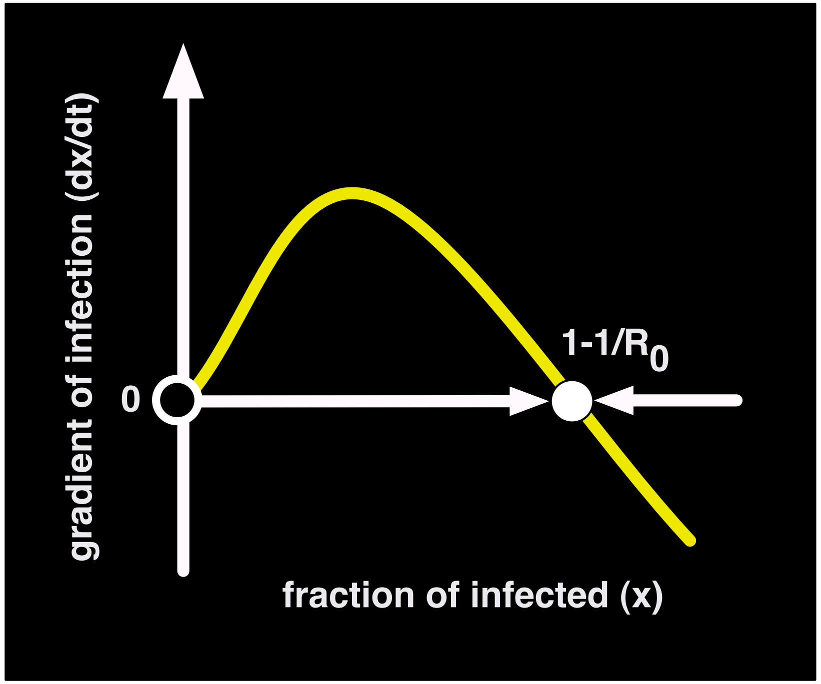
Endemic states

β: contact infection rate
δ: recovery rate

SIS model

$$S \underset{\delta}{\overset{\beta}{\rightleftarrows}} I$$

$$\dot{x} = {dx}/{dt} = x(1-x)\beta\langle k\rangle - \delta x$$

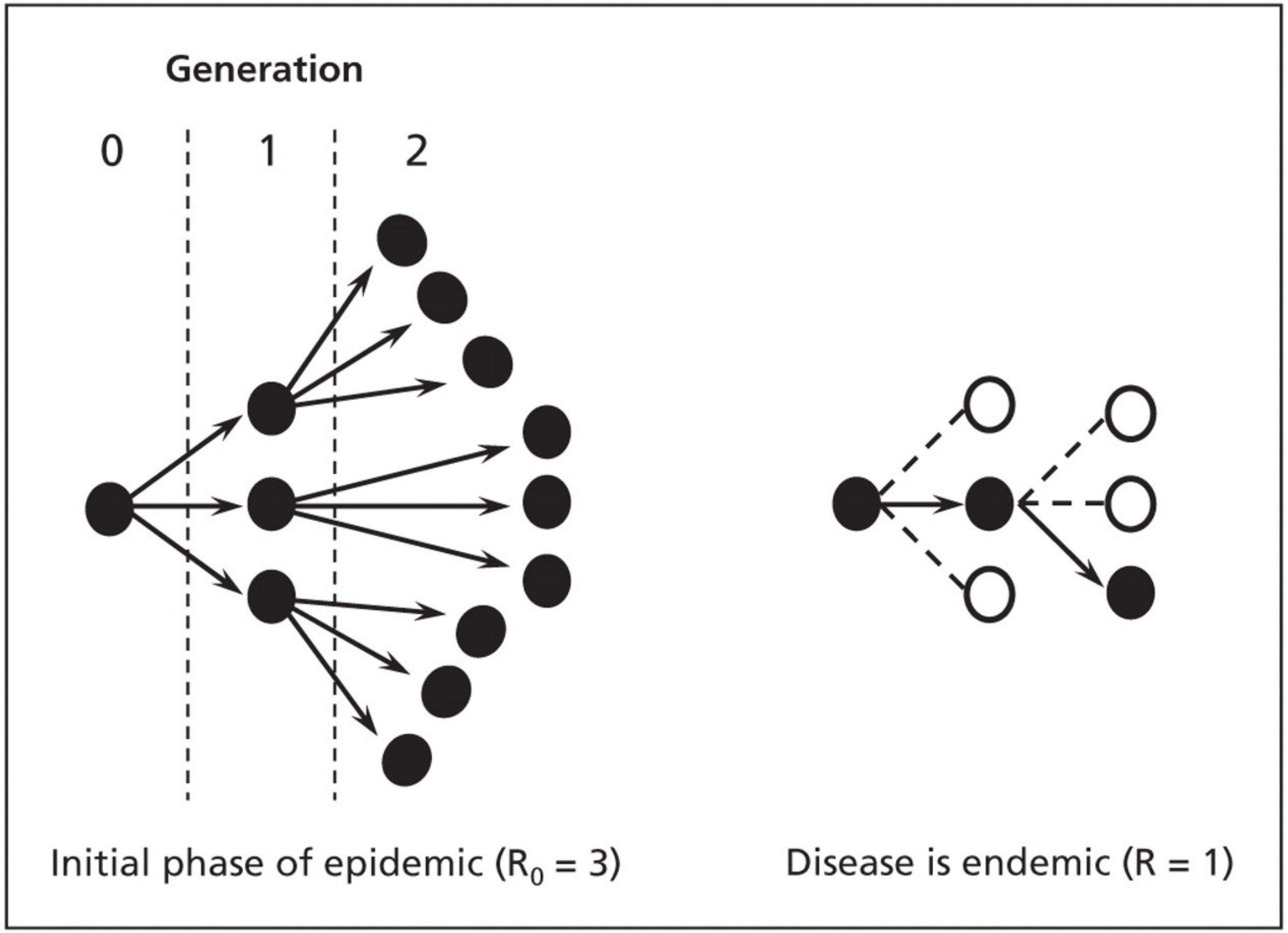


Basic Reproductive Ratio & Epidemic Threshold

Endemic for $R_0 \equiv \dfrac{\beta\langle k\rangle}{\delta} > 1$

The basic reproductive number (R-naught)

The reproductive number R_0 provides the number of individuals an infected infects if all its contacts are susceptible.



The basic reproductive number (R-naught)

The reproductive number $\boldsymbol{R_0}$ provides the number of individuals an infected infects if all its contacts are susceptible.

For $\boldsymbol{R_0}<1$ the disease dies out.

For $\boldsymbol{R_0}>1$ the pathogen will spread and persist in the population.

Higher the value of $\boldsymbol{R_0}$, faster the the spreading process.

The basic reproductive number (R-naught)

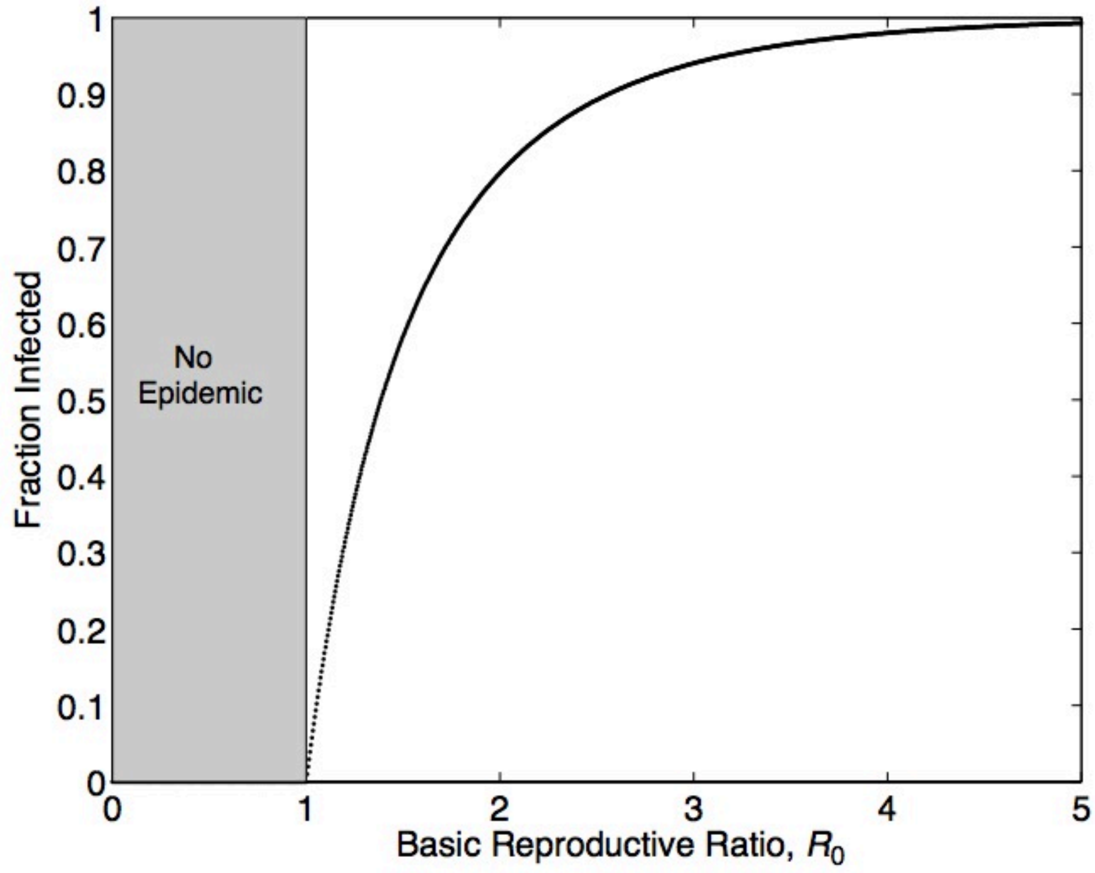
The reproductive number R_0 provides the number of individuals an infected infects if all its contacts are susceptible.

Disease	Transmission	R_0
Measles	Airborne	12-18
Pertussis	Airborne droplet	12-17
Diphtheria	Saliva	6-7
Smallpox	Social contact	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
Influenza (1918 pandemic strain)	Airborne droplet	2-3

The basic reproductive number

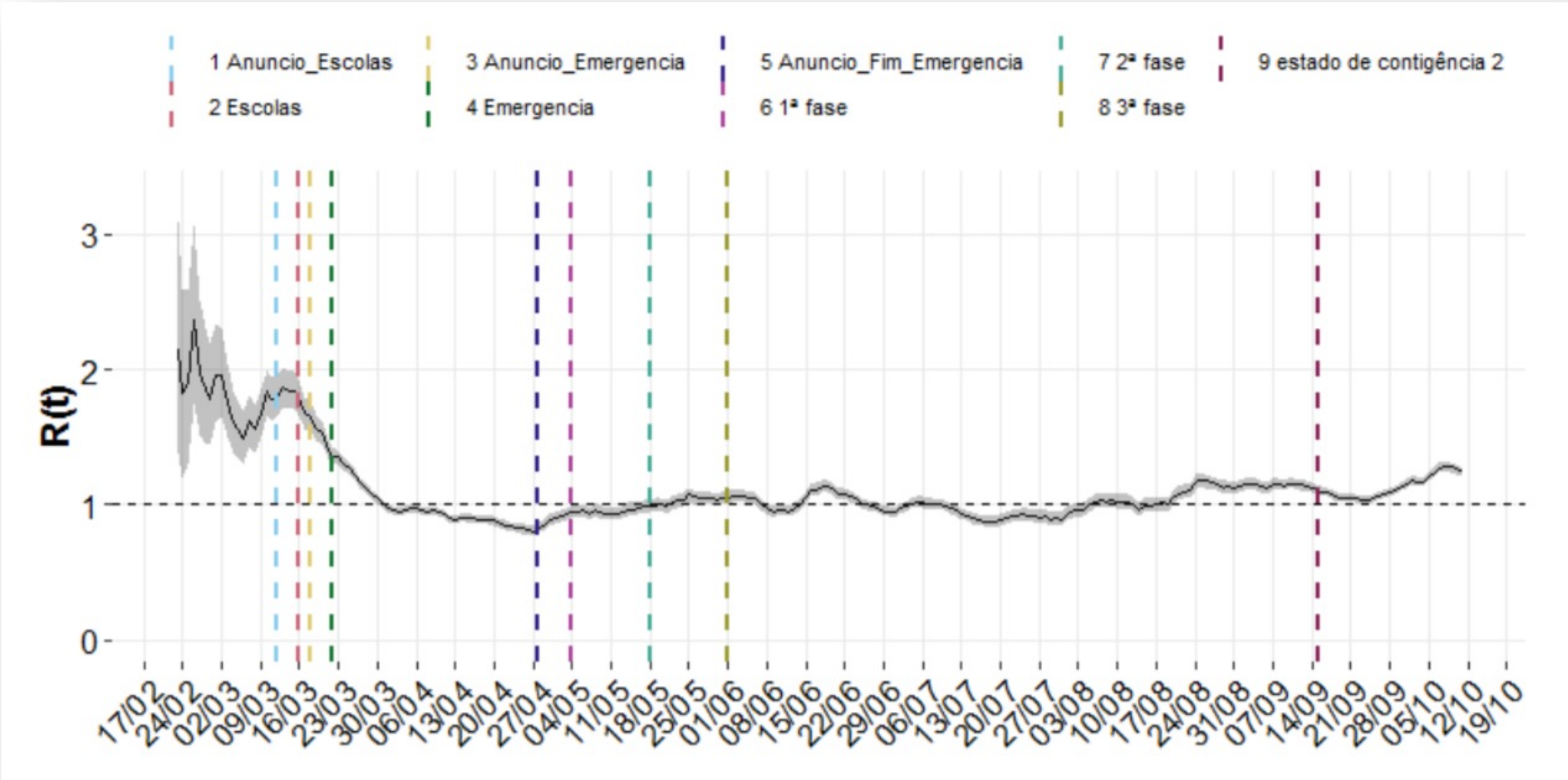
The reproductive number $\boldsymbol{R_0}$ provides the number of individuals an infected infects if all its contacts are susceptible.

$$R_0^{SIS,SIR} \equiv \frac{\beta \langle k \rangle}{\delta}$$



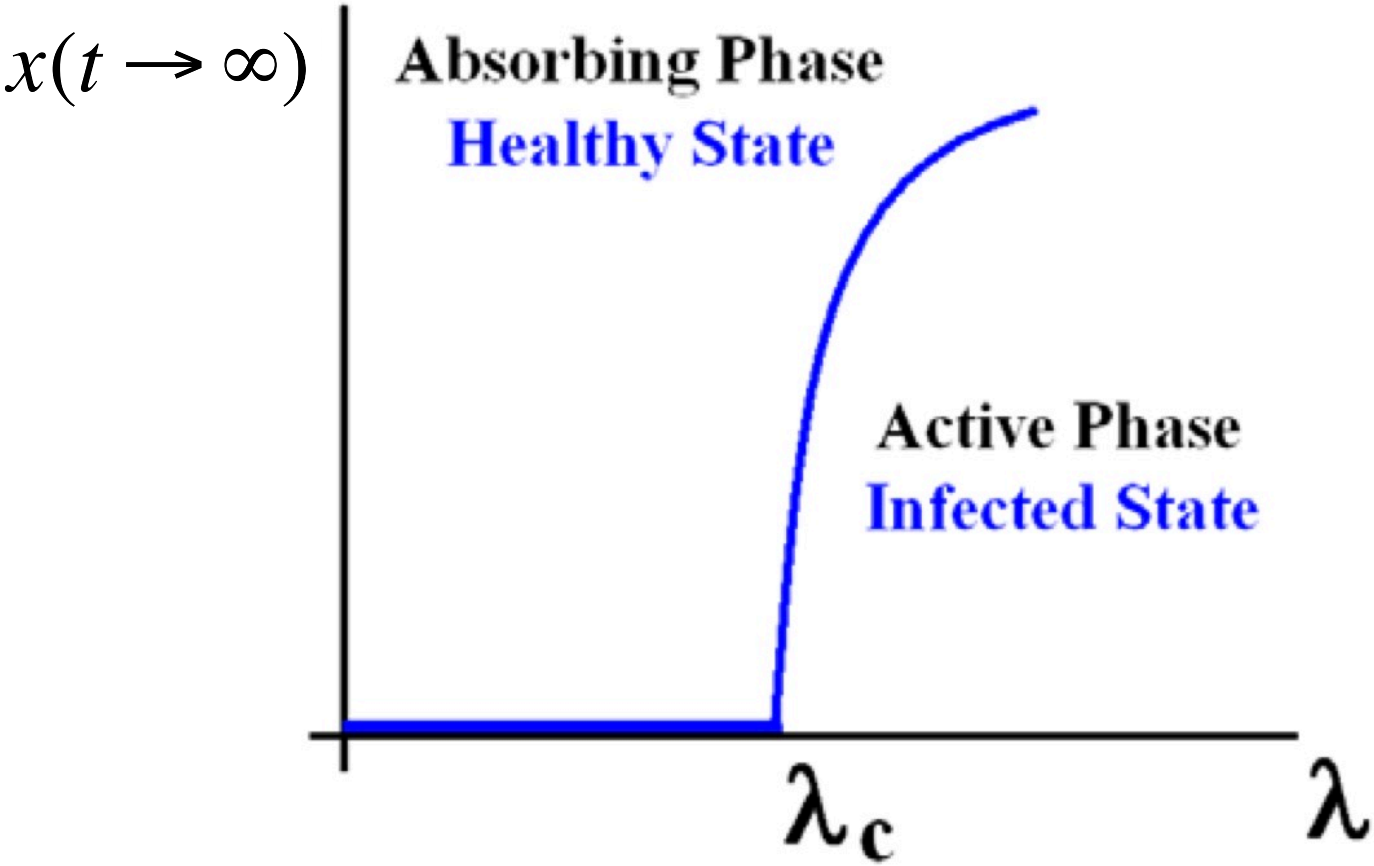
The effective reproductive number (R_t)

The effective reproductive number (***R*** or $\boldsymbol{R_t}$ or $\boldsymbol{R_e}$) provides the number of individuals an infected creates in the current state of a population, which does not have to be the uninfected state.



Epidemic threshold

Equivalently, one may define an epidemic threshold $\lambda_c = \beta_c / \delta$ which also splits the phase space into the endemic state and the healthy state.



$$R_0^{SIS,SIR} \equiv \frac{\beta \langle k \rangle}{\delta}$$

SIR model

β: contact infection rate
δ: recovery rate

Often individuals develop immunity after recovery
(e.g., Influenza) or can be removed from the population.

S

Susceptible (healthy)

$1-x-r$

$\xrightarrow{\beta}$

I

Infected

x

$\xrightarrow{\delta}$

R

Recovered / Removed / Dead

r

Traditional models

β: contact infection rate
δ: recovery rate

SIR model

$$S \xrightarrow{\beta} I \xrightarrow{\delta} R$$

$$\dot{r} = \delta x$$

$$\dot{x} = \beta \langle k \rangle x(1 - x - r) - \delta x$$

<k> – average number of contacts of a given individual
x – fraction of infected in the population
r – fraction of recovered in the population
y = 1-x-r – fraction of susceptible in the population

Traditional models

β: contact infection rate
δ: recovery rate

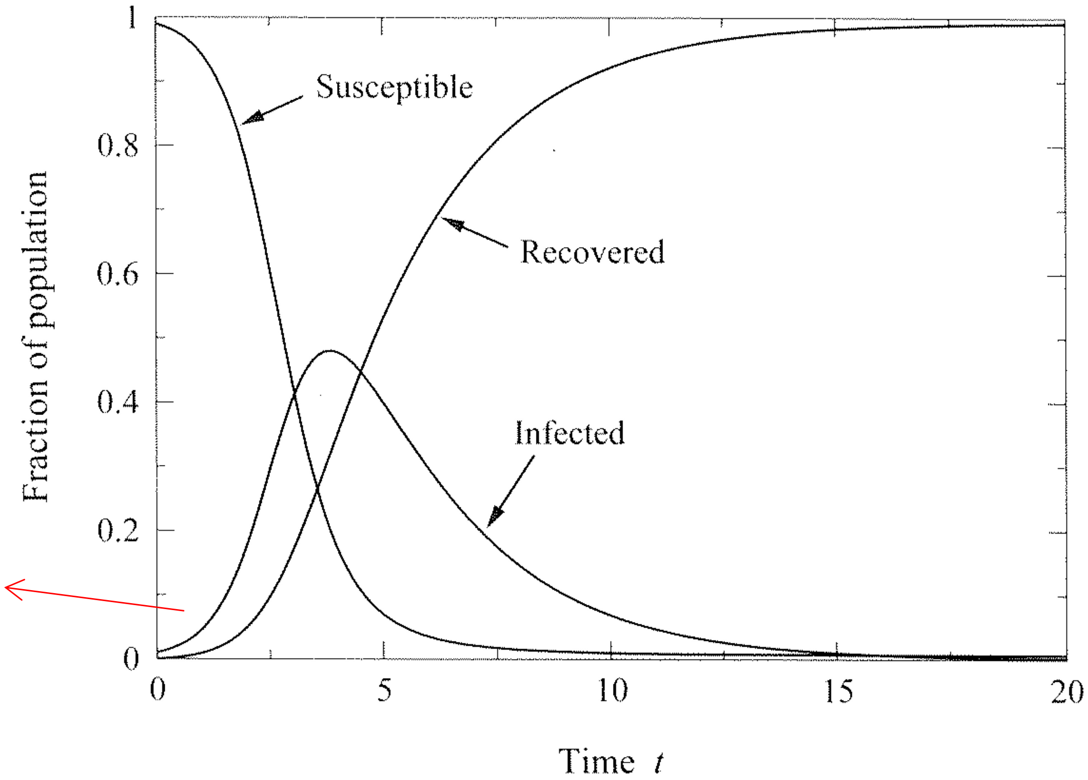
SIR model

$$S \xrightarrow{\beta} I \xrightarrow{\delta} R$$

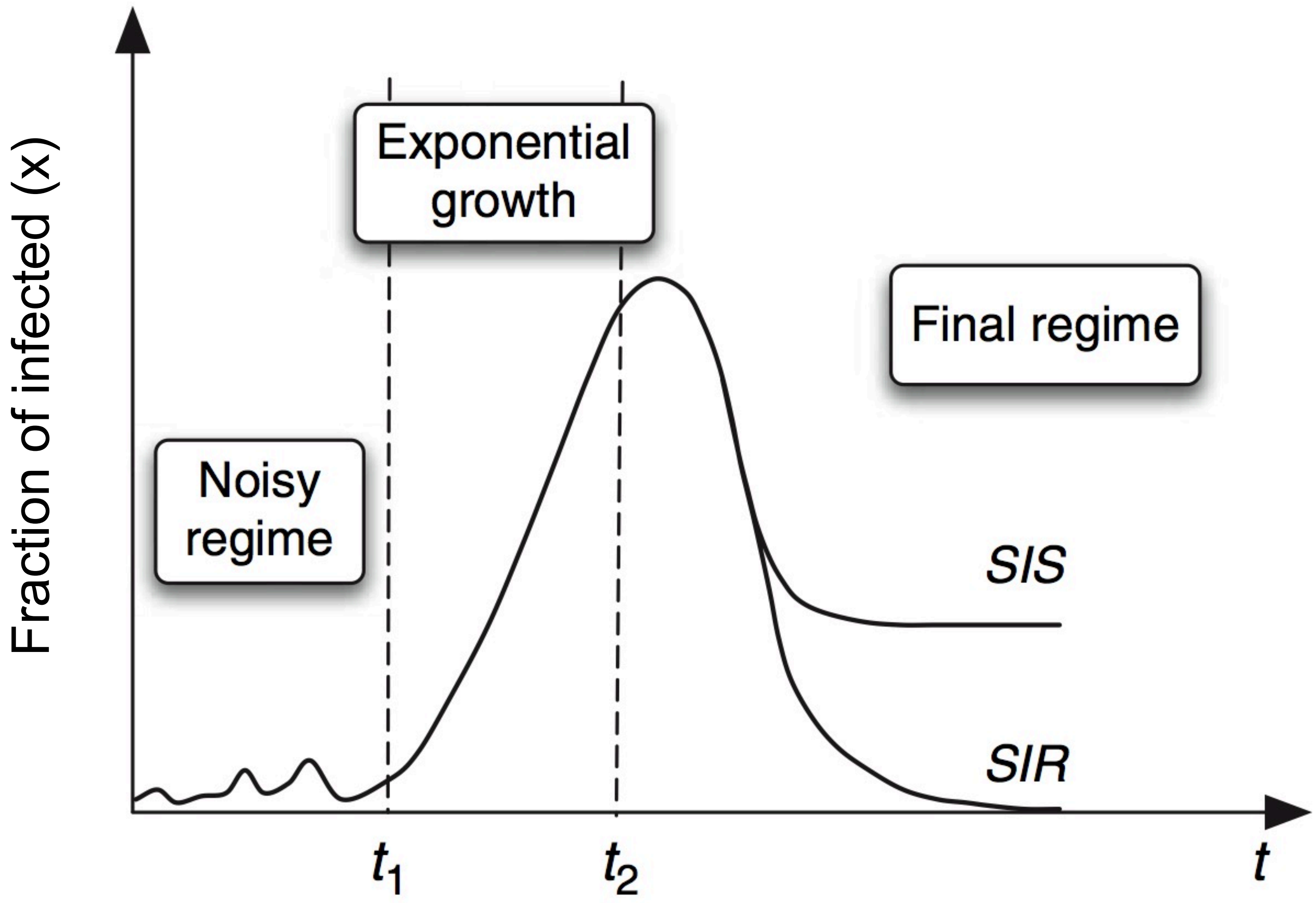
$$\dot{x} = \beta \langle k \rangle x(1 - x - r) - \delta x$$

$$\dot{r} = \delta x$$

$$R_0 = \frac{\beta \langle k \rangle}{\delta} > 1$$



Stochastic effects



Working SIR example

- Outbreak of influenza in a British boarding school in 1978.
- Soon after the start of the Easter term, three boys were reported to the school infirmary with the typical symptoms of influenza.
- Over the next few days, a very large fraction of the 763 boys in the school had contracted the infection

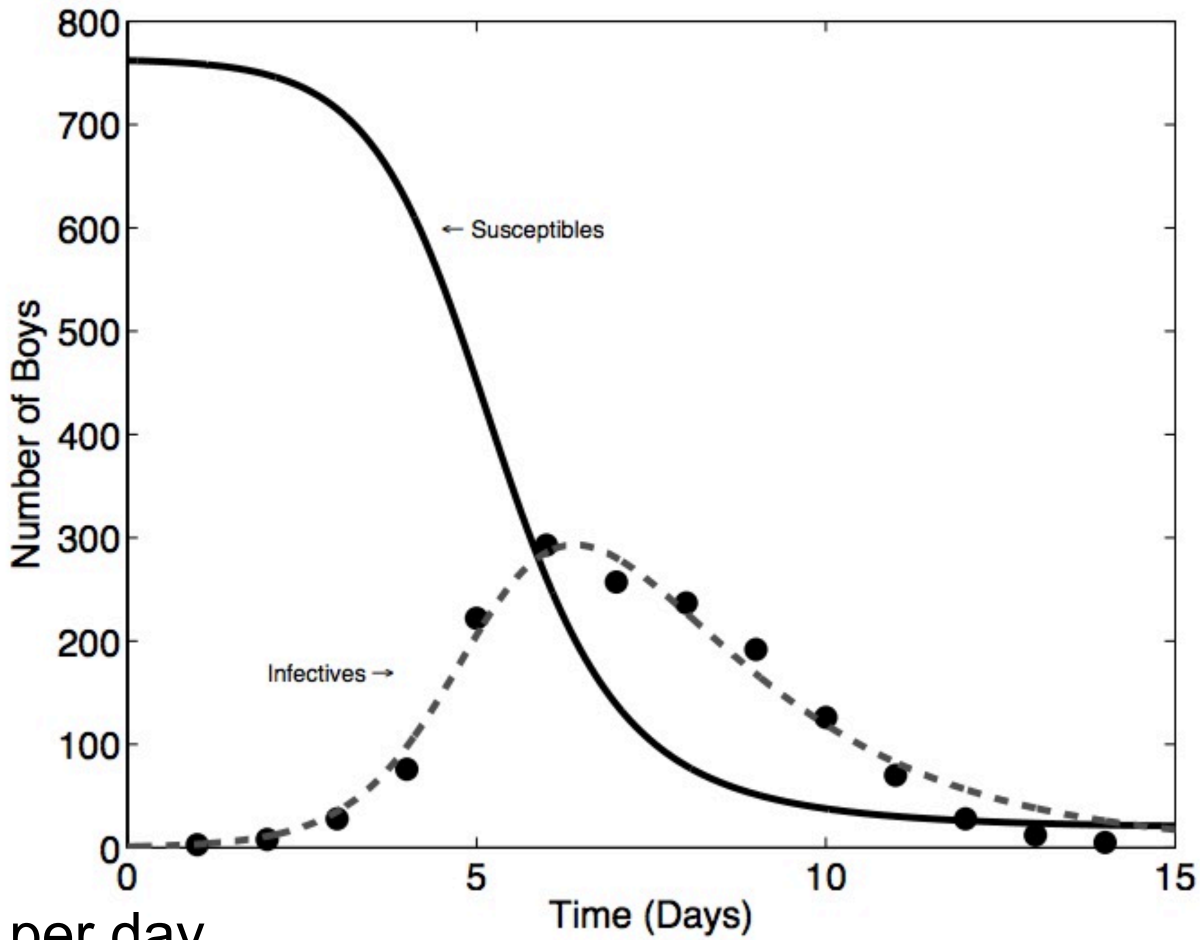
$$\dot{x} = \beta \langle k \rangle x(1 - x - r) - \delta x$$

$$\dot{r} = \delta x$$

(Murray 1989)
(Keeling & Rohani, Section 2.1.1.3)
(check their Python code!)

Working SIR example

Within two weeks, the infection had become extinguished, as predicted by the simple SIR model

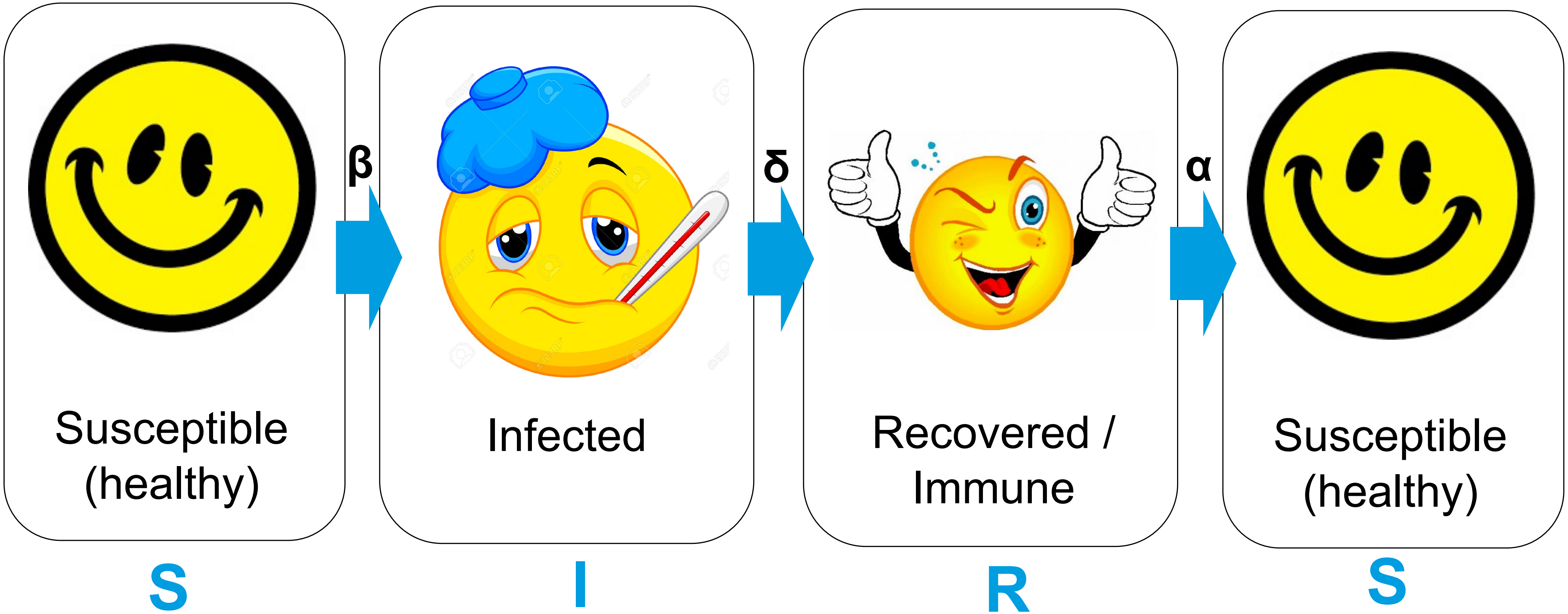


Estimated parameters:
transmission rate ($\beta\langle k\rangle$) =1.66 per day
$1/\delta$ = 2.2 days,
giving an R_0 of 3.65.

(Murray 1989)
(Keeling & Rohani, Section 2.1.1.3)
(check their Python code!)

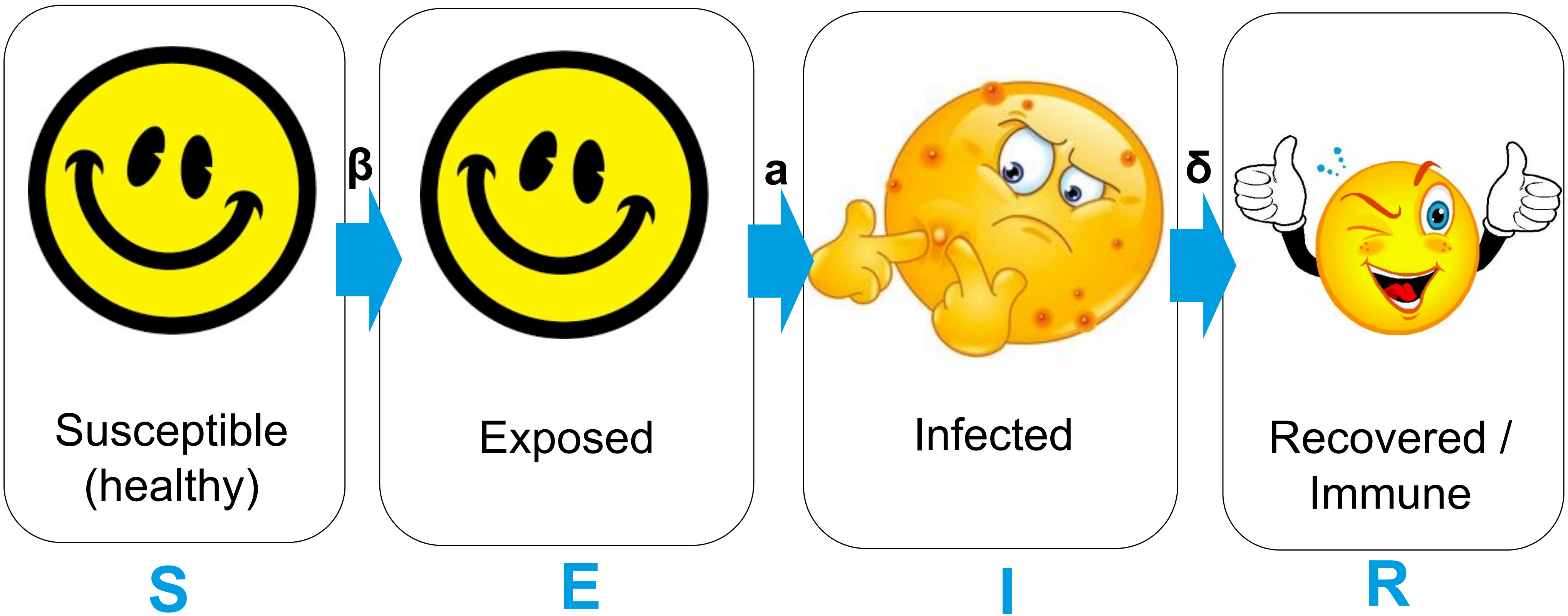
Other variants: SIRS model

This model is simply an extension of the SIR model. The only difference is that it allows members of the recovered class to be free of infection and rejoin the susceptible class.



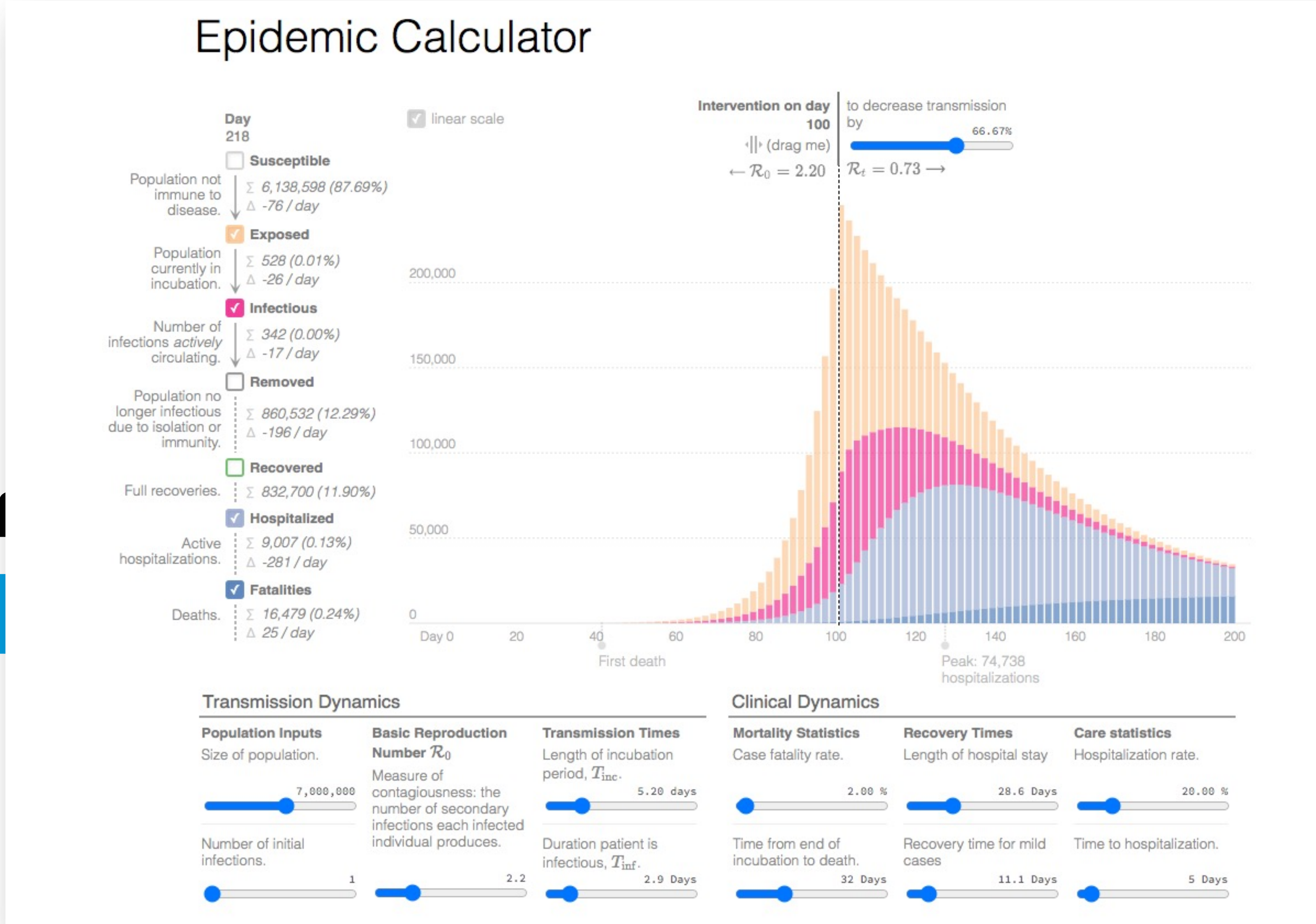
Other variants: SEIR and SEIR-S models

For many important infections there is a significant incubation period during which the individual has been infected but is not yet infectious themselves. During this period the individual is in compartment E (for exposed).



COVID-19

Ex: SARS-CoV-2
Severe acute respiratory syndrome (2019-?)
http://gabgoh.github.io/COVID/index.html



Susceptible
(healthy)

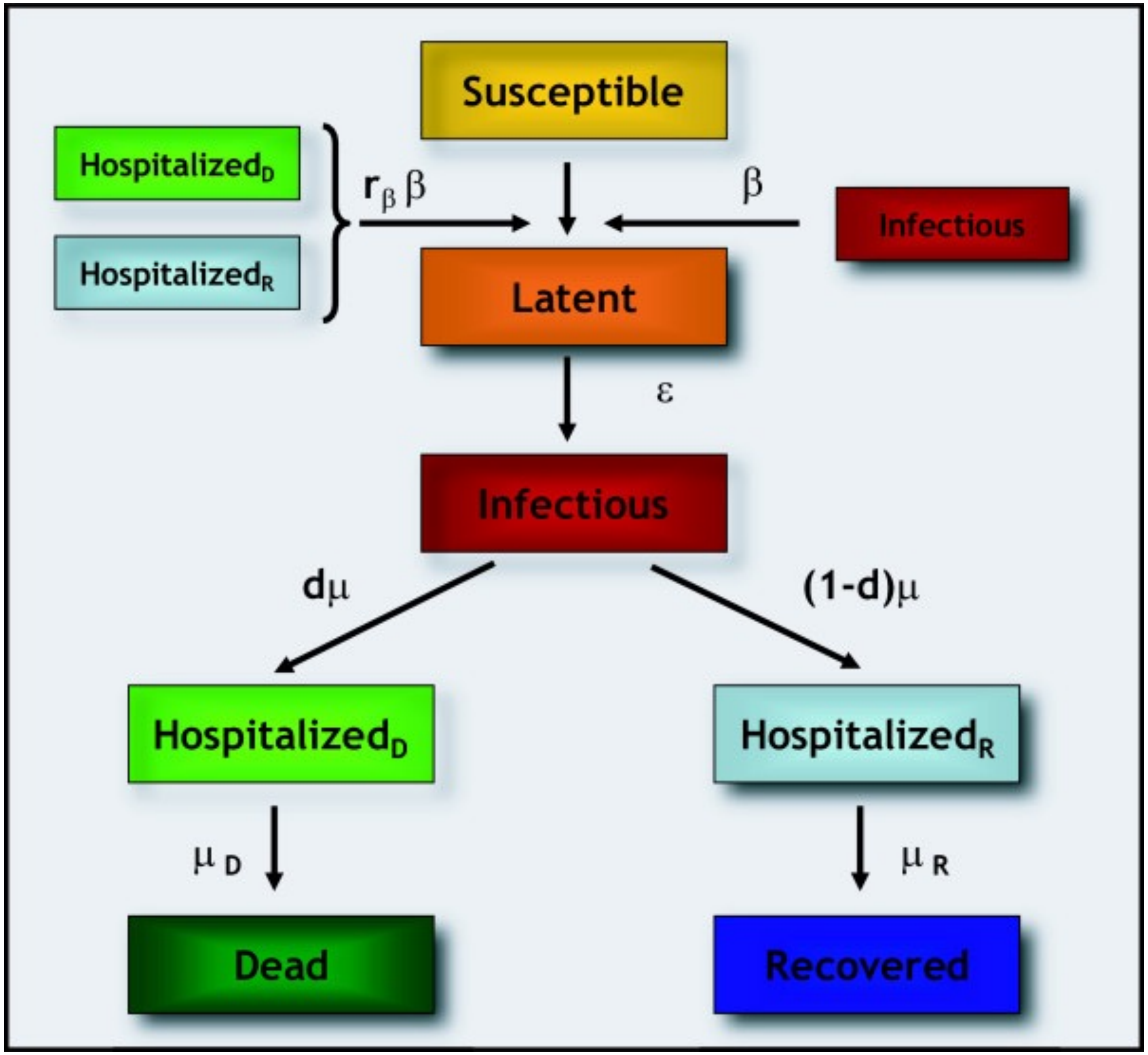
S

E

I

R

Other variants: SARS (SARS-CoV-1, 2002-2003 outbreak)



Colizza et al. BMC Medicine 2007 5:34

Epidemic control

Aiming at β

Transmission-Reducing Interventions

Face masks, gloves, hand washing, condoms, etc.

$$\dot{x} = \beta \langle k \rangle x (1 - x - r) - \delta x$$

Aiming at <k>

Contact-Reducing Interventions

For diseases with severe health consequences officials can quarantine patients, close schools and limit access to frequently visited public spaces, like movie theaters and malls, or impose global or partial lockdowns.

Aiming at the number of susceptible

Vaccination

Removal of vaccinated nodes from the network and their links. Reduces the spreading rate, enhancing the likelihood that the pathogen dies out.

Epidemic control

vaccination in the SIR model

- Disease control is one the most important application of epidemic models.
- Can you estimate the critical proportion ***w_c*** to be immunized in order to avoid a disease outbreak?

$$R_0 = \frac{\beta\langle k \rangle}{\delta} < 1 \quad \Rightarrow \quad R_0 = \frac{\beta\langle k \rangle (1-w)}{\delta} < 1$$

$$R_0(w) = R_0(1-w) < 1$$

$$\boxed{w_c = 1 - \frac{1}{R_0}}$$

Epidemic control

vaccination in the SIR model

- Disease control is one the most important application of epidemic models.
- Can you estimate the critical proportion w_c to be immunized in order to avoid a disease outbreak?

w_c	R_0
0.50	2
0.66	3
0.80	5
0.90	10

$$w_c = 1 - \frac{1}{R_0}$$

Epidemic control
vaccination in the SIR model

- Disease control is one of the most important application of epidemic models.
- Can you estimate the critical proportion w_c to be immunized in order to avoid a disease outbreak?

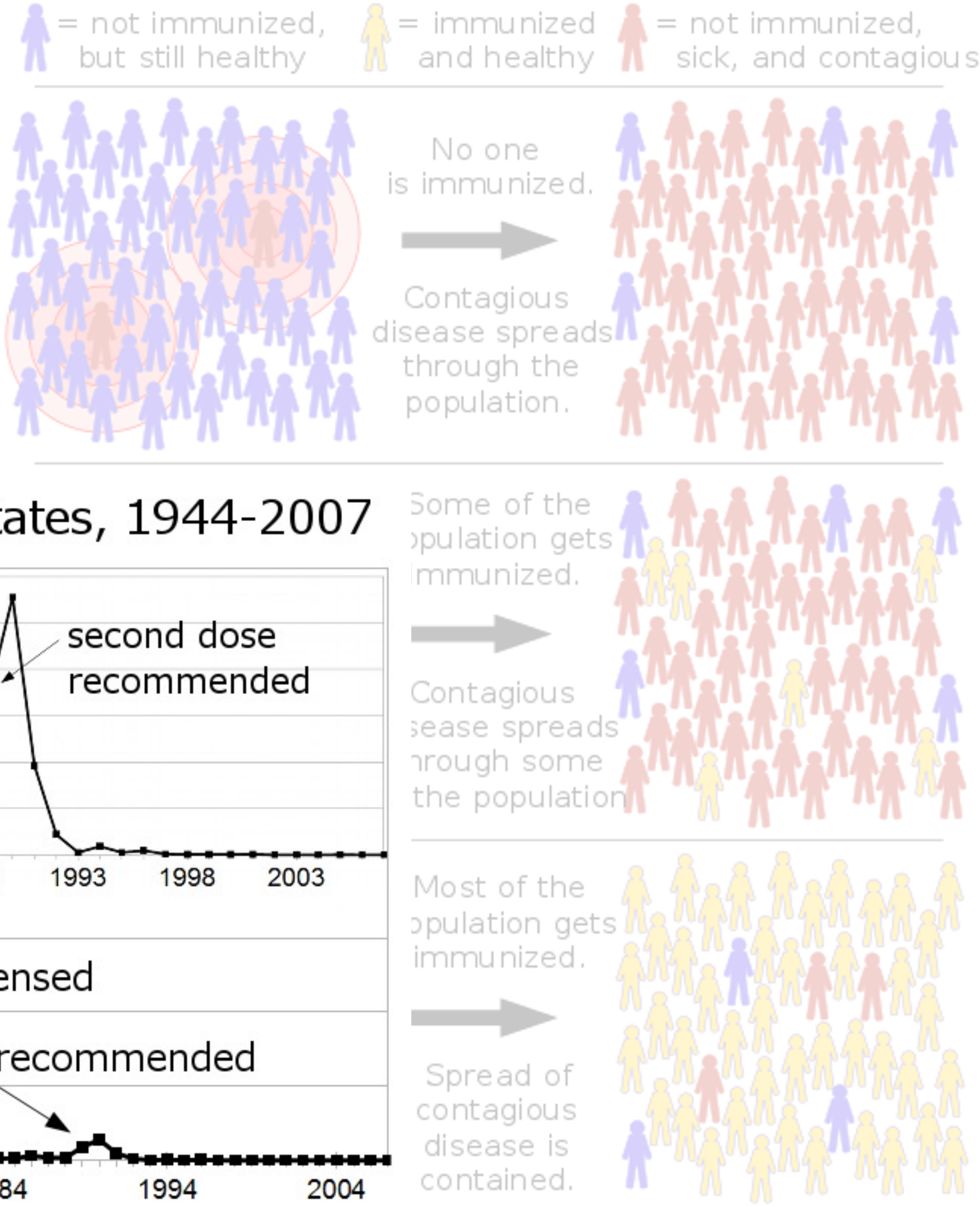
Challenge: Vaccines are not perfect!
If we consider efficacy (φ) for a vaccine, can you compute the efficacy below which it is impossible to stop a disease outbreak, even if the entire population is vaccinated?

w_c	R_0
0.50	2
0.66	3
0.80	5
0.90	10

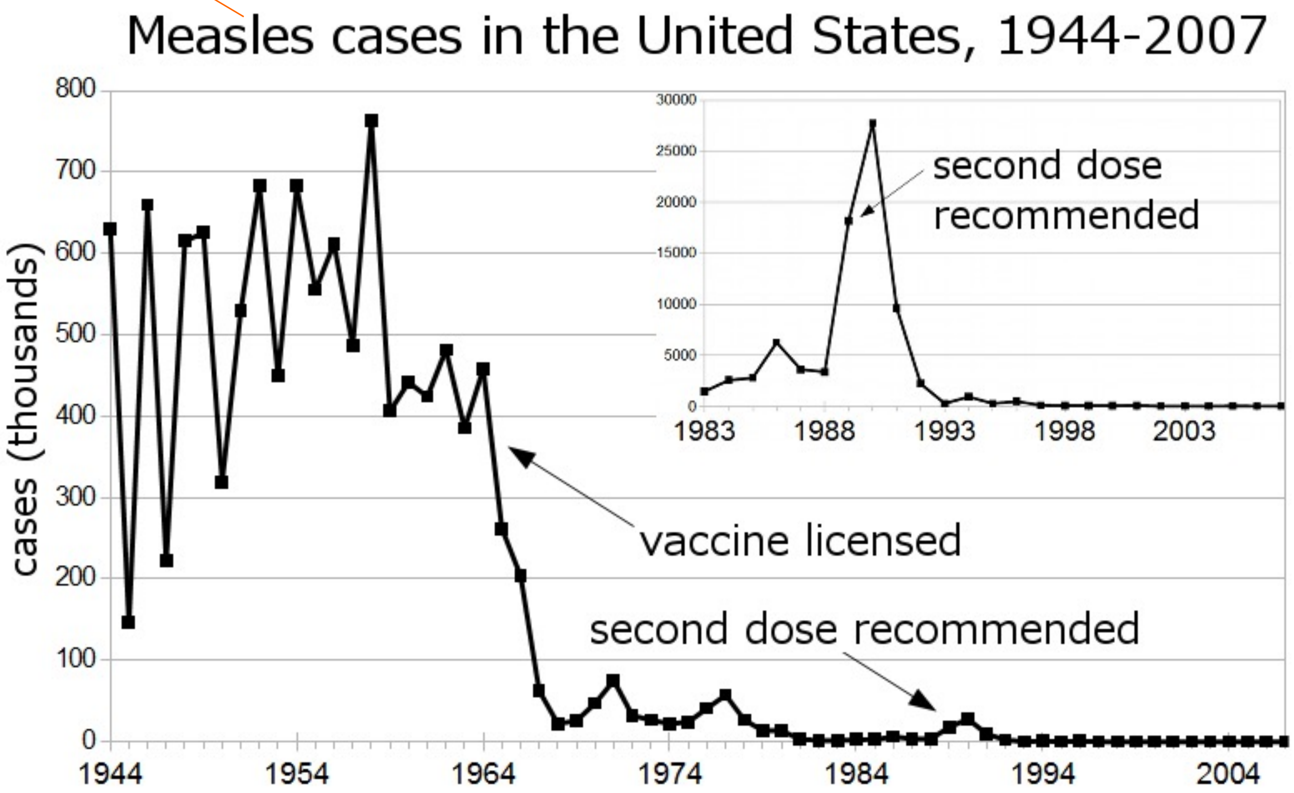
$$w_c = 1 - \frac{1}{R_0}$$

Herd immunity

when the fraction of immune *w* (or *r*) is larger than w_c



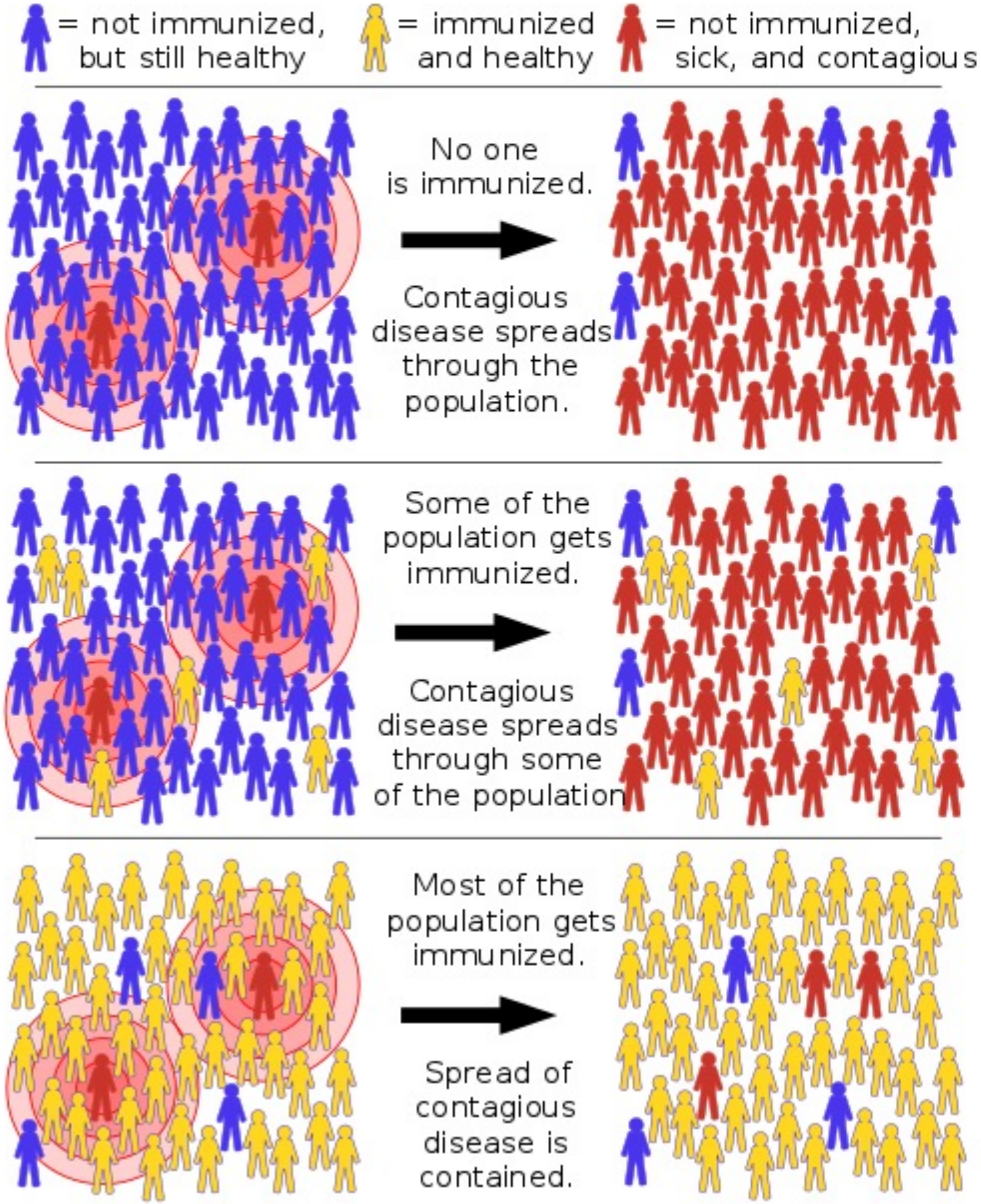
("*Sarampo*", "*Mässling*", "*Mazelen*", …)



Herd immunity

when the fraction of immune *w* (or *r*) is larger than w_c

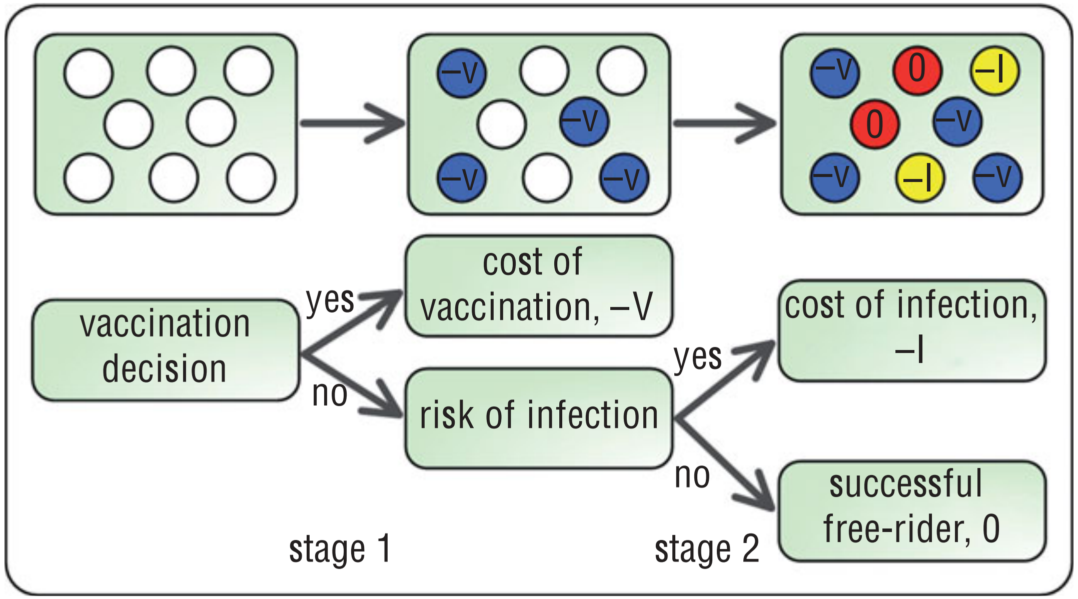
w_c	R_0
0.50	2
0.66	3
0.80	5
0.90	10



Herd immunity

when the fraction
of immune *w* (or *r*)
is larger than w_c

Herd immunity & vaccination
is vulnerable to the
free-rider problem!!



Fu, F., Rosenbloom, D. I., Wang, L., & Nowak, M. A. (2011). Imitation dynamics of vaccination behaviour on social networks. Proceedings of the Royal Society of London B: Biological Sciences, 278(1702), 42-49.

Epidemic control: age does matter

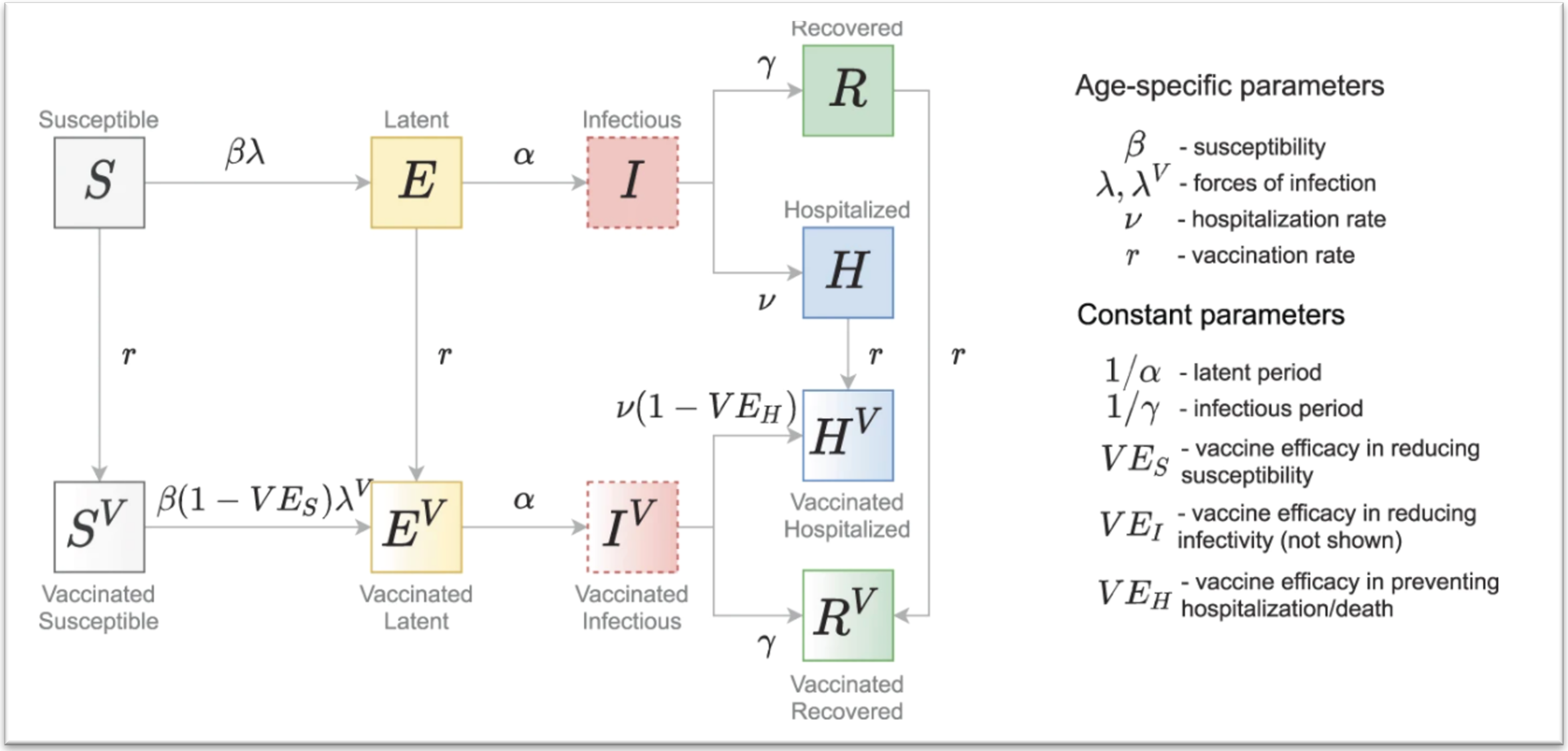
Solution: age-structured epidemic models

- split the population into k age groups
- define k^2 contact rates among these groups

- Age structure is able to disentangle social and biological factors.

Epidemic control: age does matter

Ganna Rozhnova
Utrecht & U Lisbon



Ana Nunes
FCUL, U Lisbon

& many colleagues

Manuel Carmo Gomes
FCUL, U Lisbon

Viana, et al. "Controlling the pandemic during the SARS-CoV-2 vaccination rollout." Nature Communications 12.1 (2021): 1-15.

Code available here:
https://github.com/lynxgav/COVID19-vaccination

Epidemic control

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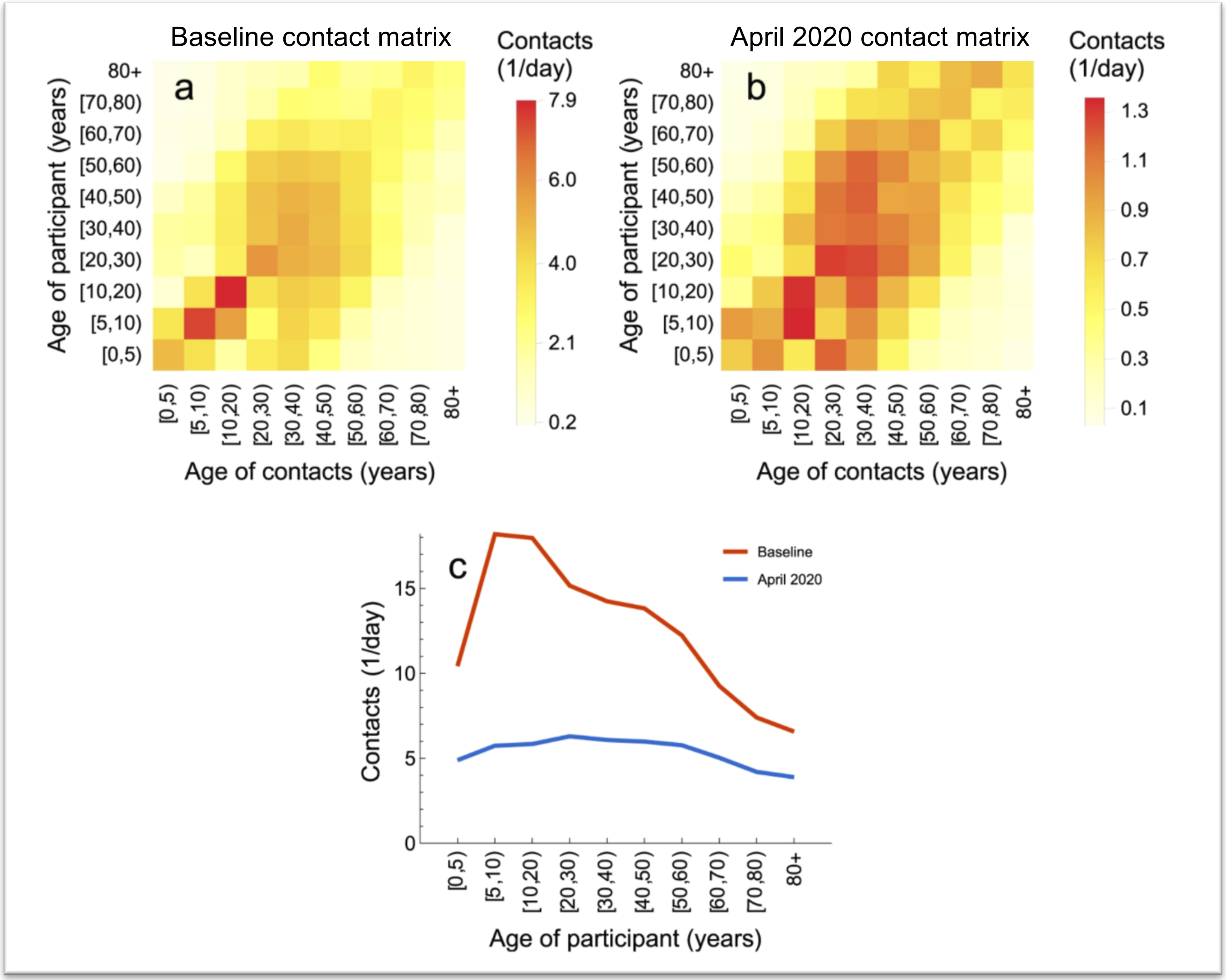

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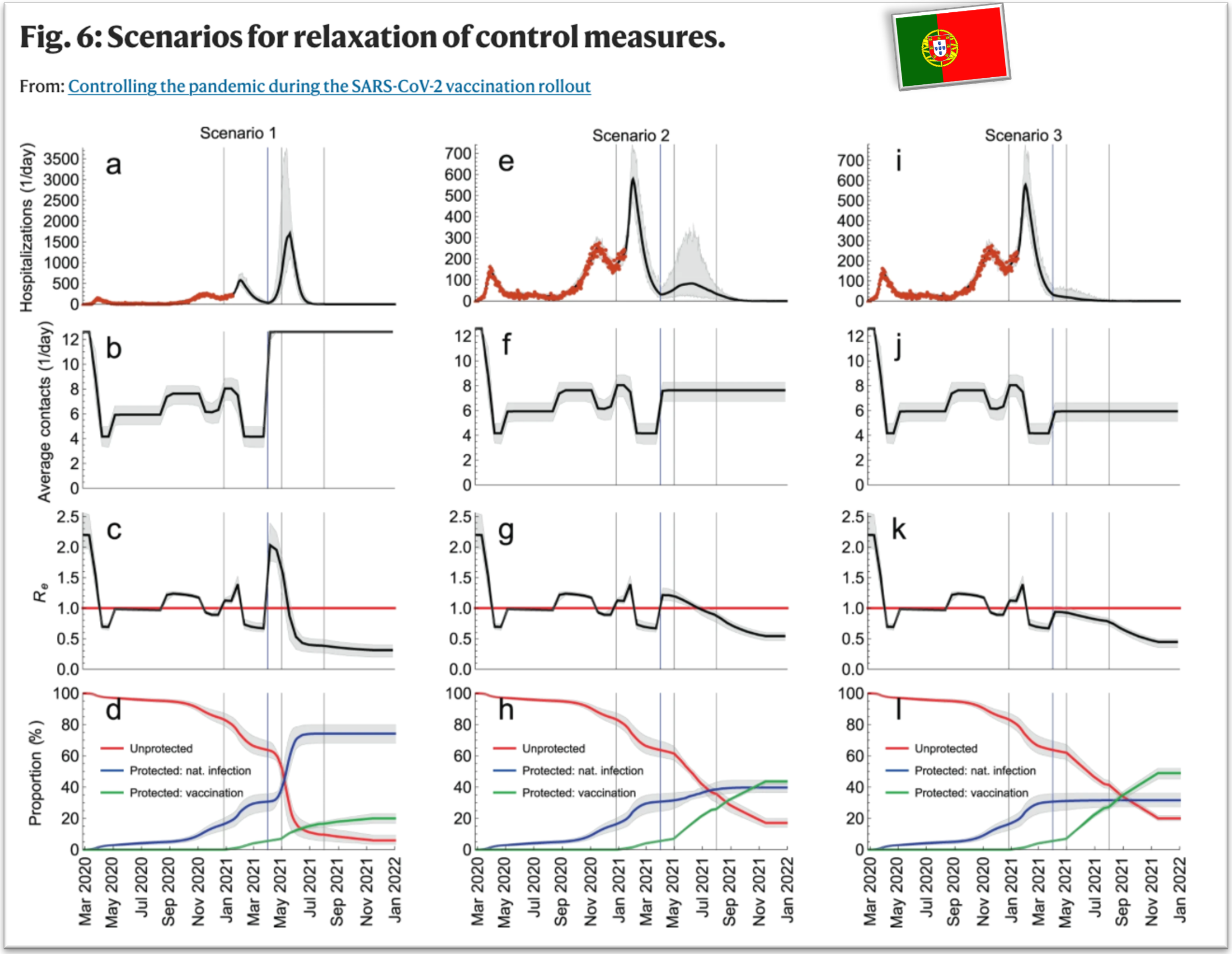
Epidemic control



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Contact tracing and time-delays

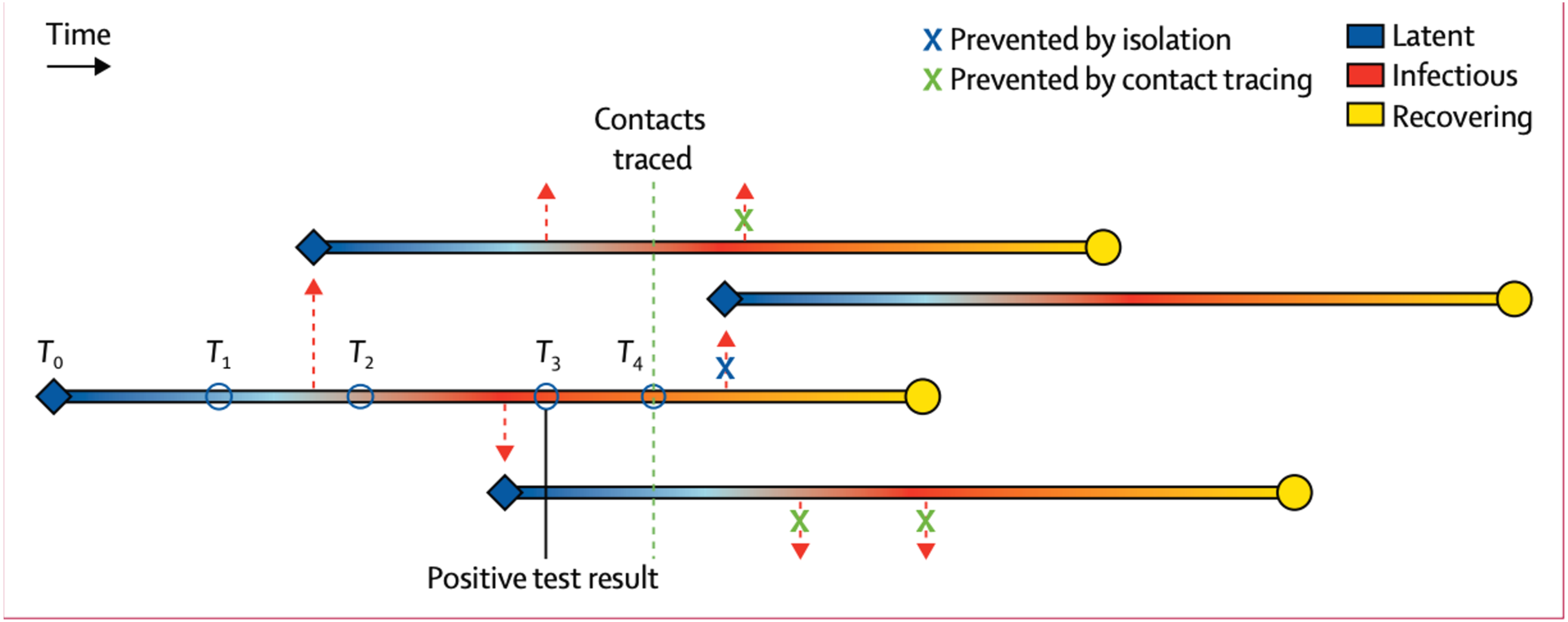


***Figure 1:* Schematic of the contact tracing process and its time delays**

T_0=time of infection of index case. T_1=onset of infectiousness. T_2=symptom onset. T_3=time of positive diagnosis. T_4=time of tracing and quarantining of contacts.

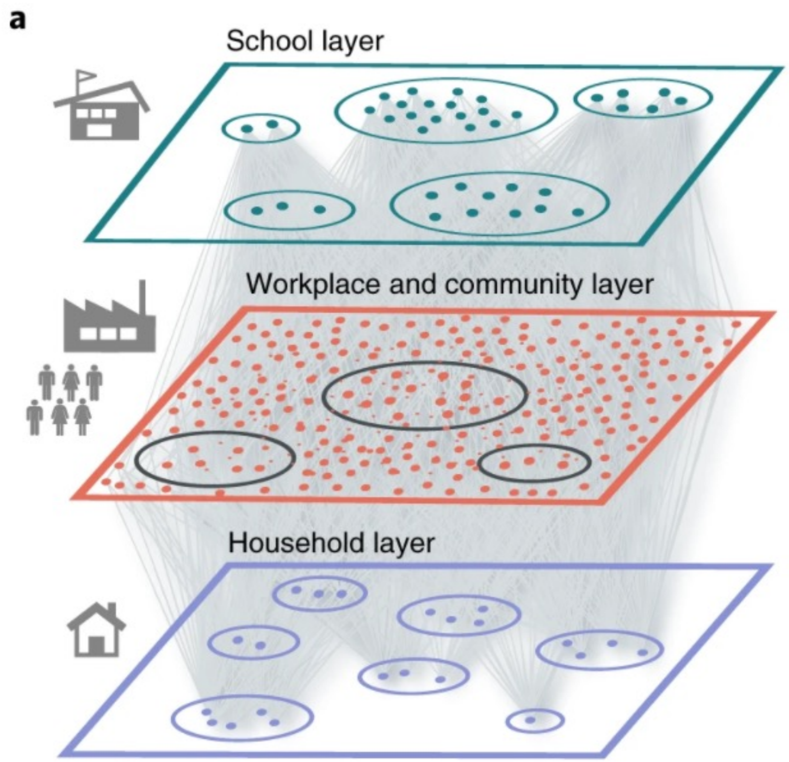
Contact tracing will only contribute to containment of COVID-19 if it can be organised such that delays in the process from symptom onset to isolation is very short.

Kretzschmar, M. E., Rozhnova, G., et al. (2020). Impact of delays on effectiveness of contact tracing strategies for COVID-19: a modelling study. The Lancet Public Health, 5(8), e452-e459.

Epidemic control:
not all interactions are equivalent

Solution: context dependent interactions

- split interactions by context
- measure a large set contact rates per typology
- Disentangle several sociological factors.
- Allow for a good assessment of the impact of lockdown measures



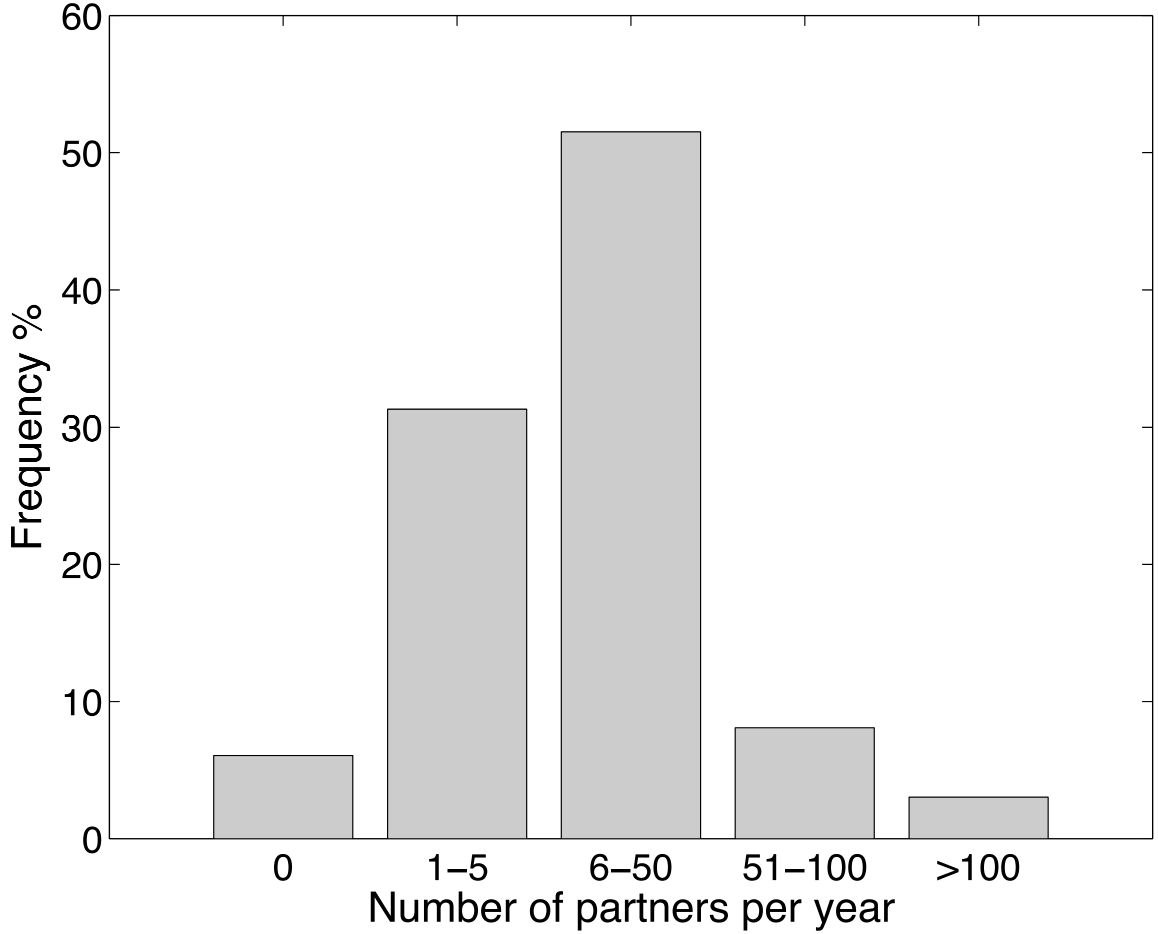
Aleta et al. *Nature Human Behaviour* 4, 964–971(2020)

Epidemic control in STIs

Models with risk structure in Sexually transmitted infections (STIs)

Traditional division into 2 classes:

- High risk
- Low risk



Frequency distribution of the number of sexual partners of male homosexuals in London in the mid-1980’s (MacManus and McEnvoy, 1987) (Anderson & May, 1991)

Epidemic control in STIs

Models with risk structure in Sexually transmitted infections (STIs)

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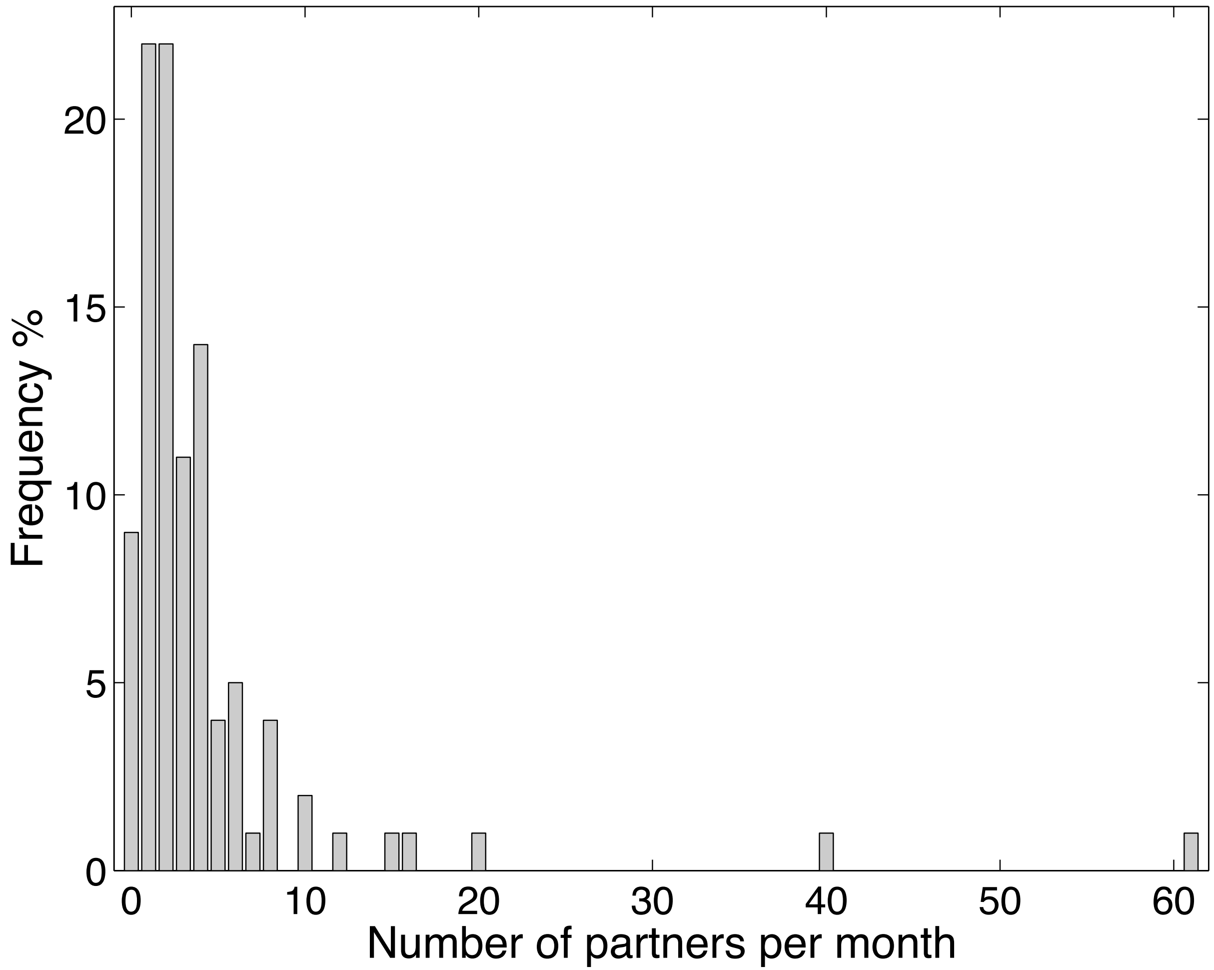
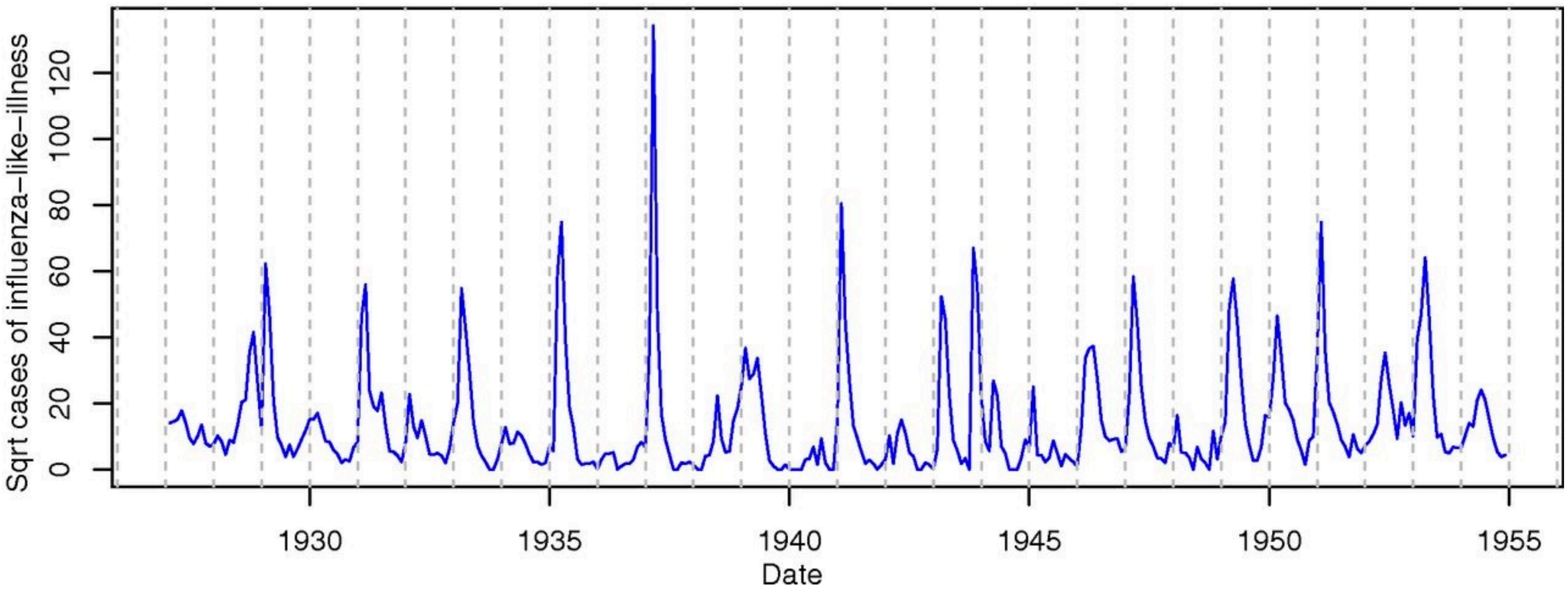


Frequency distribution of the number of sexual partners of male homosexuals in London in the mid-1980's (MacManus and McEnvoy, 1987) (Anderson & May, 1991)

Epidemics of many infectious diseases occur periodically. Why?

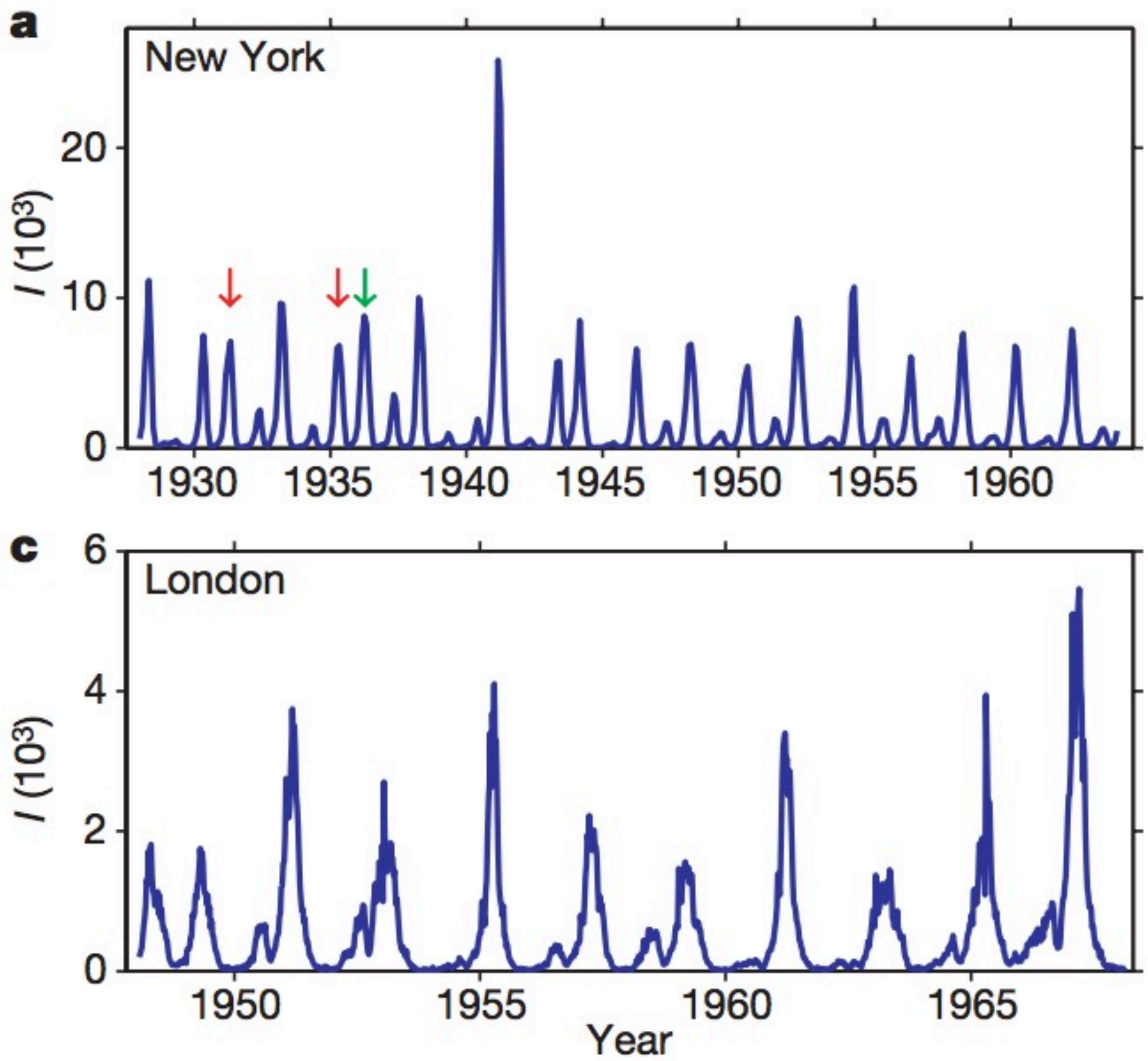
Ex: Influenza



Bjørnstad and Viboud, Timing and periodicity of influenza epidemics, PNAS 113 (46) 12899-12901 (2016)

Epidemics of many infectious diseases occur periodically. Why?

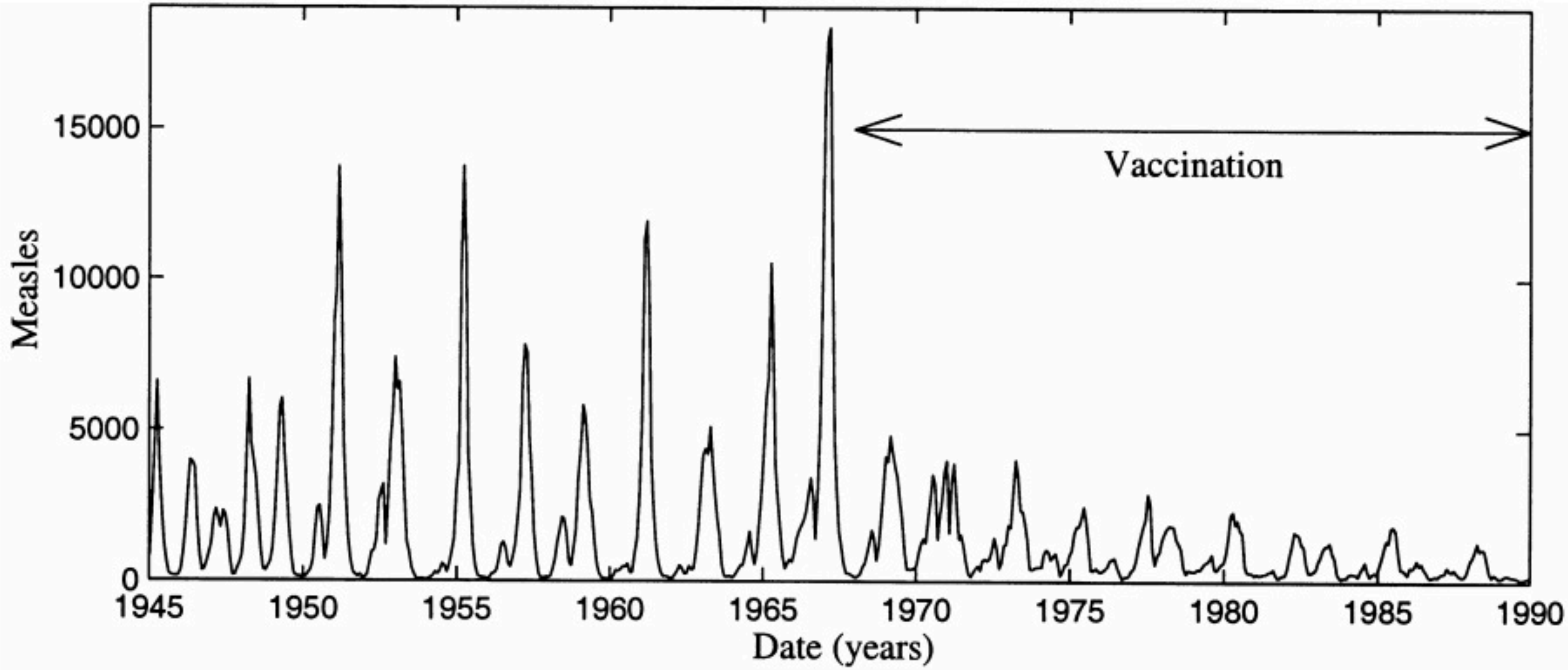
Ex: Measles (“*Sarampo*”, “*Mässling*”, “*Mazelen*”, …)



Stone, Ronen, Olinky, Huppert, Nature 2007

Epidemics of many infectious diseases occur periodically. Why?

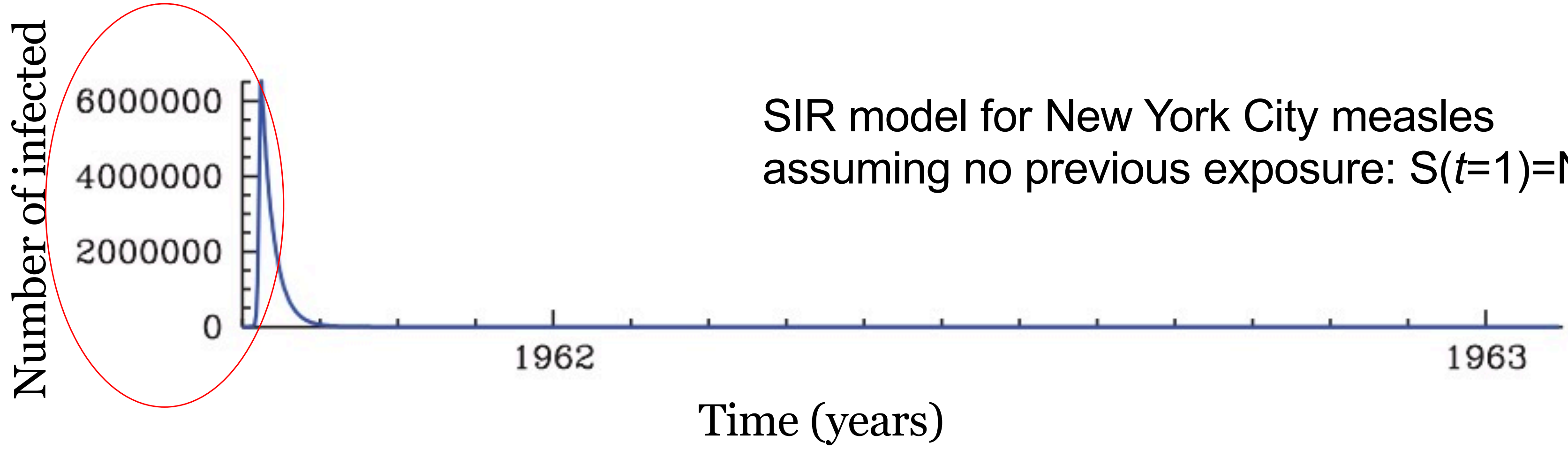
Ex: Measles (“*Sarampo*”, “*Mässling*”, “*Mazelen*”, …)



M.J. Keeling et al. / Physica D 148 (2001) 317–335

Recurrent epidemics

Ex: Measles ("*Sarampo*", "*Mässling*", "*Mazelen*", …)



SIR model for New York City measles assuming no previous exposure: $S(t=1)=N-1$; $I(t=1)=1$

The profile does not look much like the real data for the 1962 epidemic in New York. So is there something wrong with our model?

No, but there is something very wrong with our initial conditions!

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Recurrent epidemics

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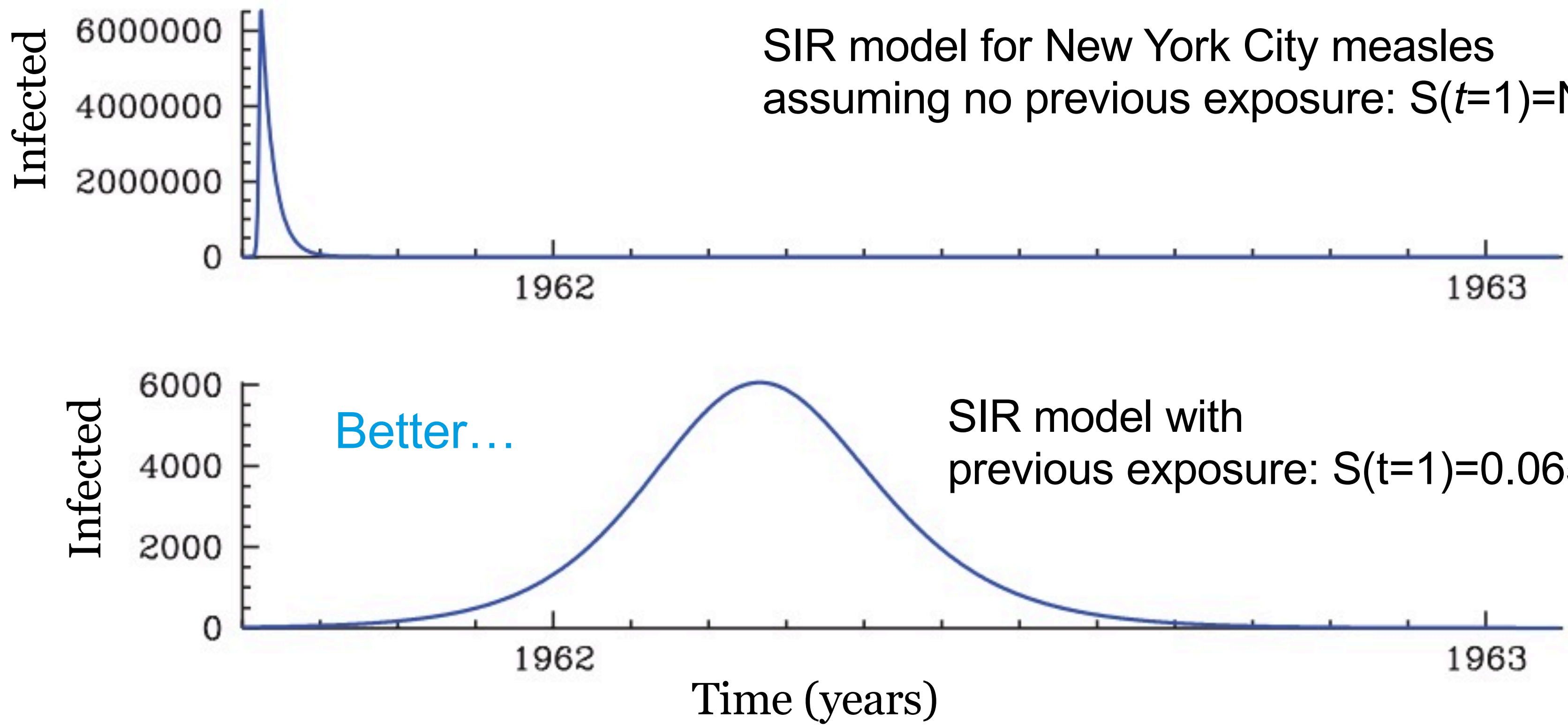


SIR model for New York City measles assuming no previous exposure: S(*t*=1)=N-1 ;I(*t*=1)=1

Better…

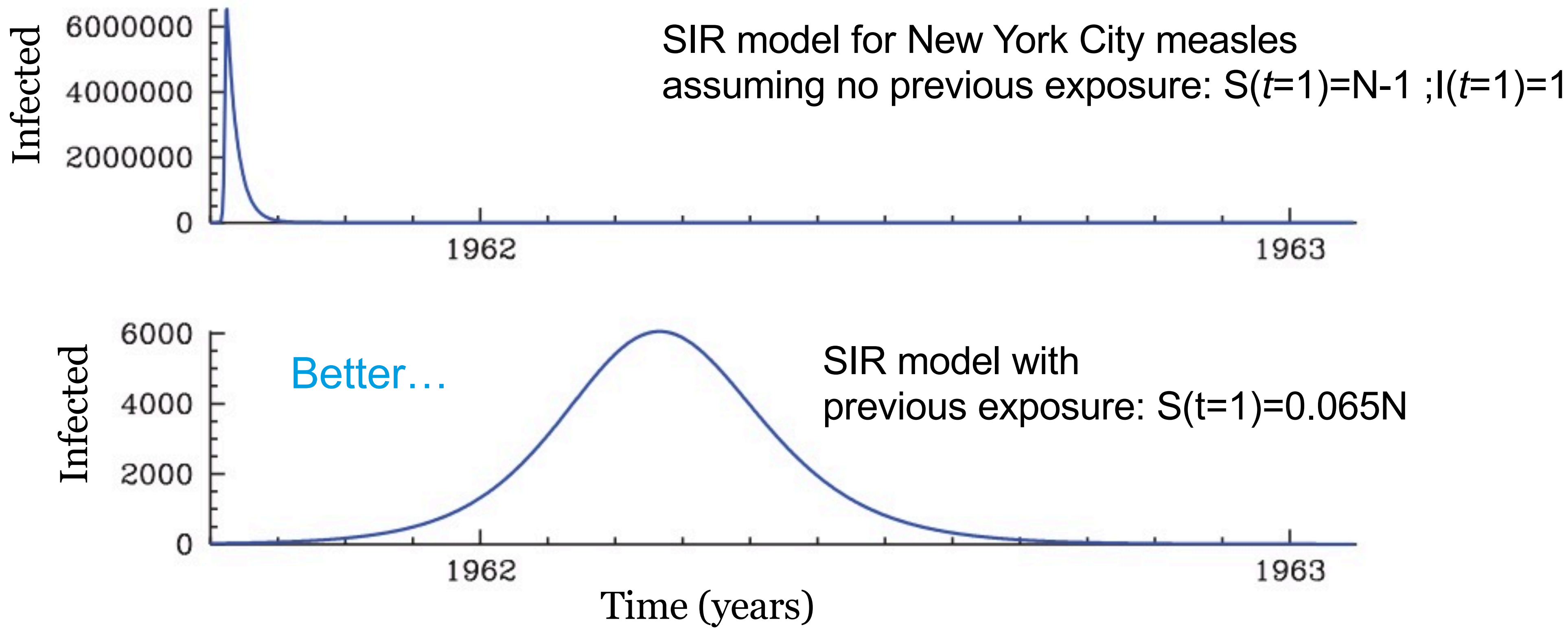
SIR model with previous exposure: S(t=1)=0.065N

Yet, we’re still missing recurrent epidemics… What’s missing in your model?

M.J. Keeling et al. / Physica D 148 (2001) 317–335

Recurrent epidemics

Ex: Measles (“*Sarampo*”, “*Mässling*”, “*Mazelen*”, …)



- The characteristic turnover of disease occurs because the pathogen is running out of susceptible individuals.
- To stimulate a second epidemic, there must be a source of susceptible individuals.
- For measles, that source cannot be previously infected people, because of its lifelong immunity to the virus. So who is it?

M.J. Keeling et al. / Physica D 148 (2001) 317–335

Recurrent epidemics & Vital Dynamics

Ex: Measles (“*Sarampo*”, “*Mässling*”, “*Mazelen*”, …)

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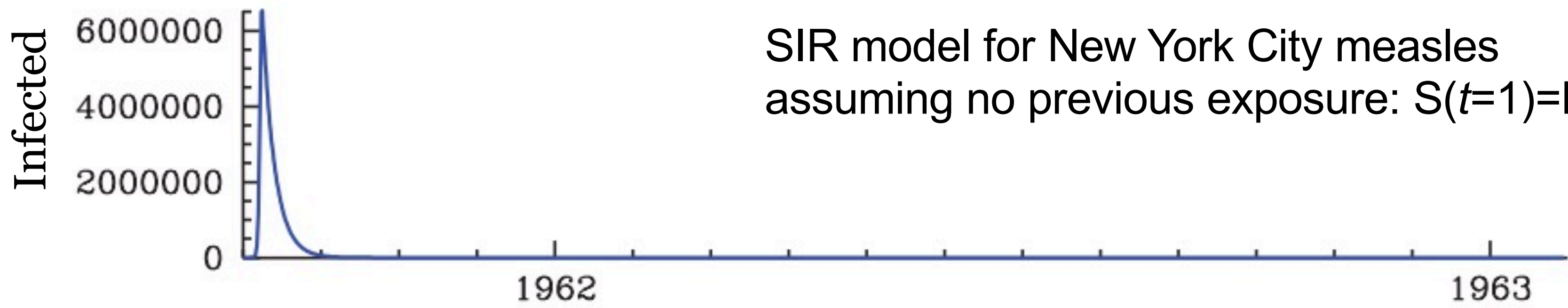

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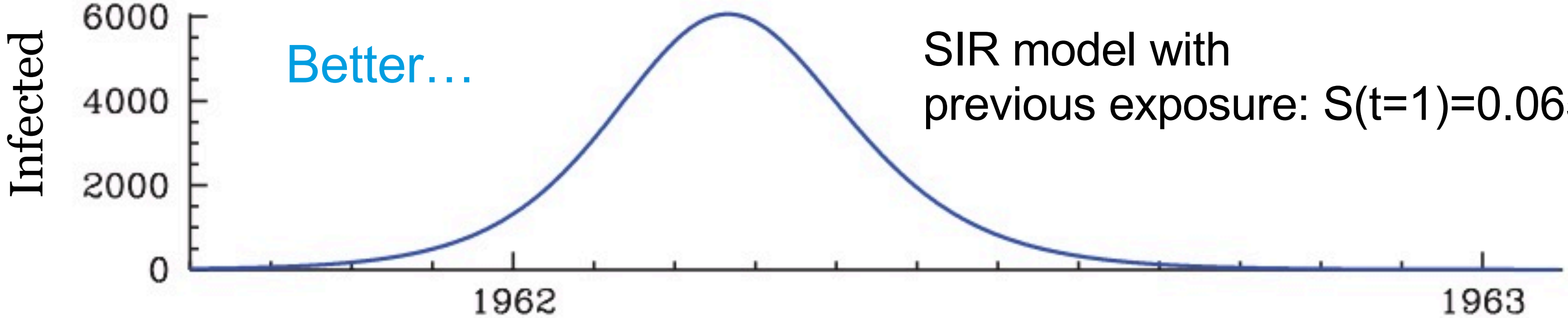
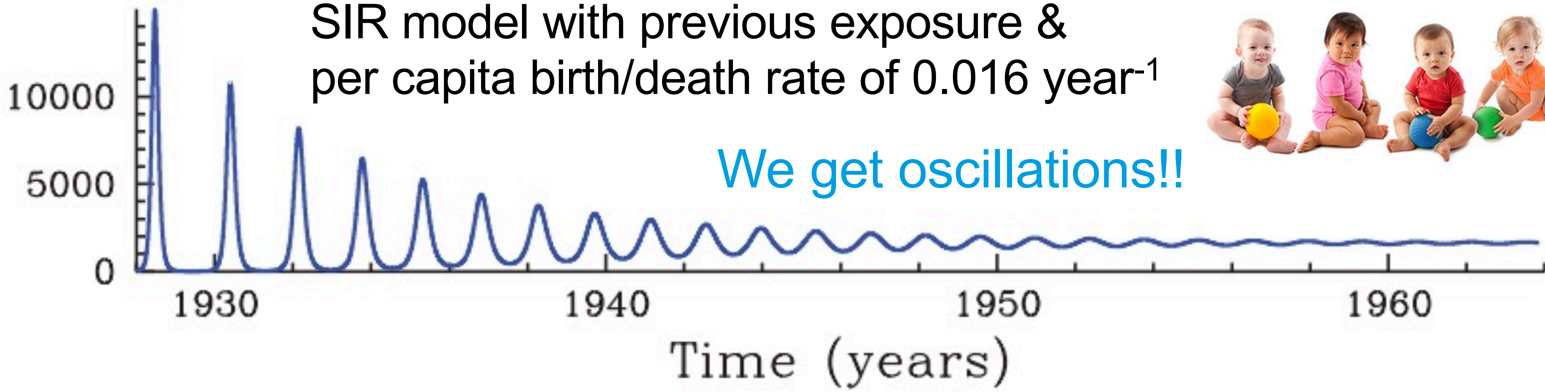


SIR model with previous exposure &
per capita birth/death rate of 0.016 $year^{-1}$

We get oscillations!!



SIR with Vital Dynamics

Ex: Measles (“*Sarampo*”, “*Mässling*”, “*Mazelen*”, …)

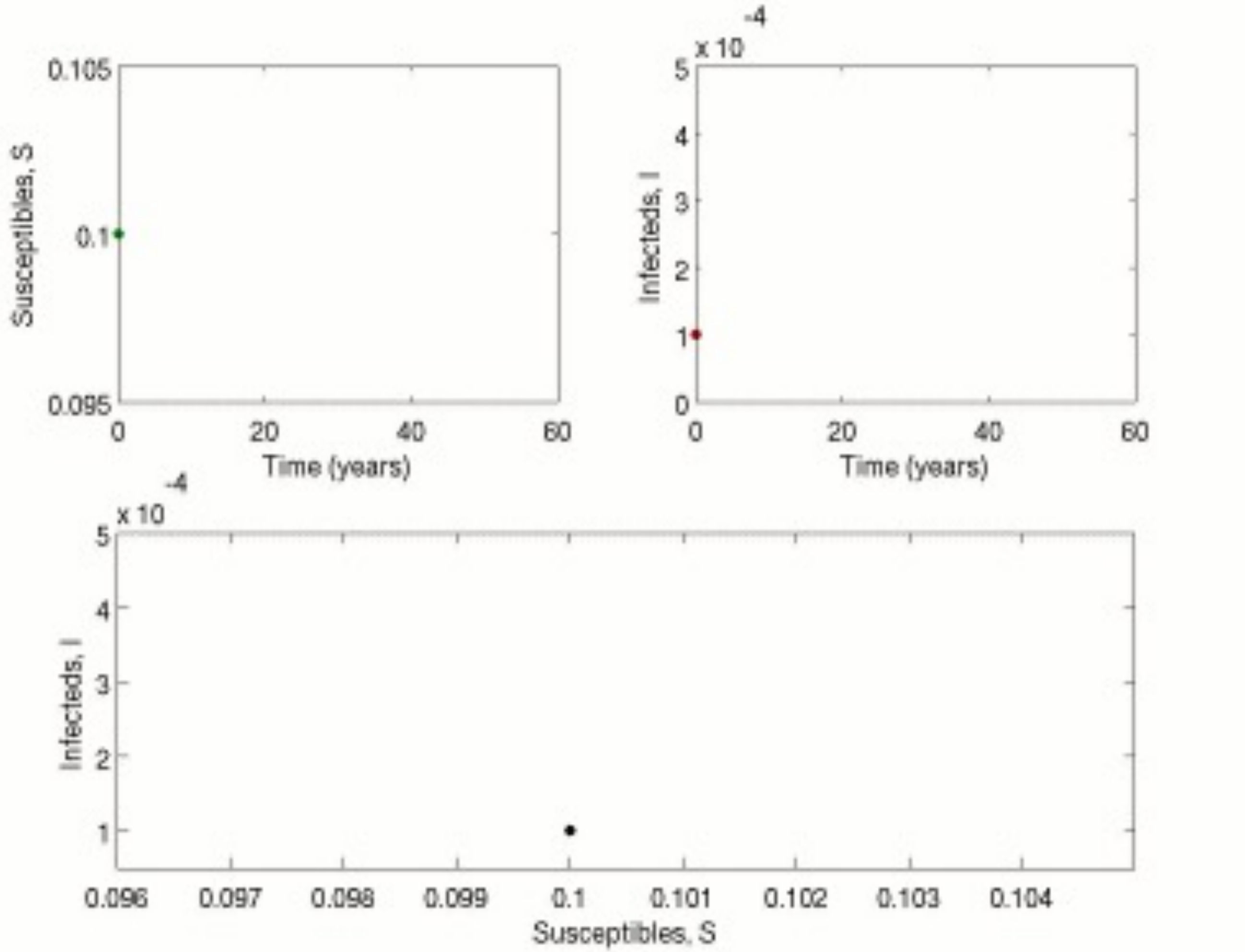
Newborns provide a constant stream of susceptibles.

Birth rate

Death rate

$$\frac{dS}{dt} = B - \beta\langle k\rangle SI - \mu S$$

$$\frac{dI}{dt} = \beta\langle k\rangle SI - \mu S$$



Nice! We have oscillations but they are damped… What are we still missing?

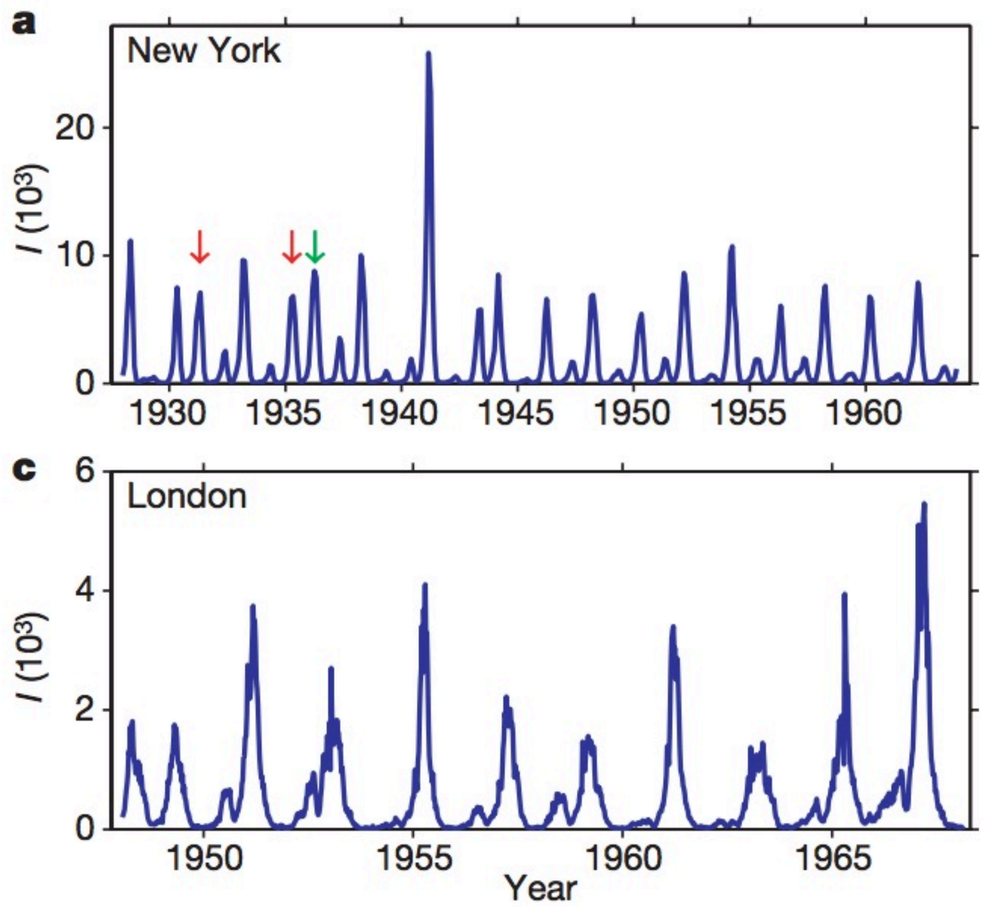
Seasonal Forcing

Ex: Measles (“*Sarampo*”, “*Mässling*”, “*Mazelen*”, …)

In reality, the transmission rate β<k> is not constant…

To see that, consider the fact that most susceptibles are children.

Children are in closer contact when school is in session, so the transmission rate must vary seasonally.



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Seasonal Forcing

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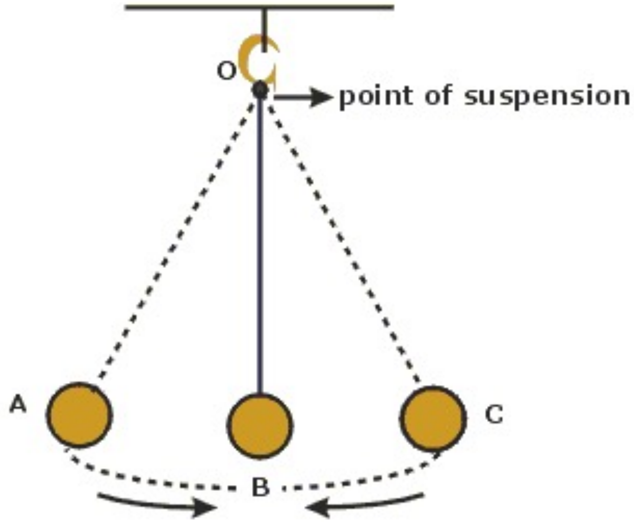
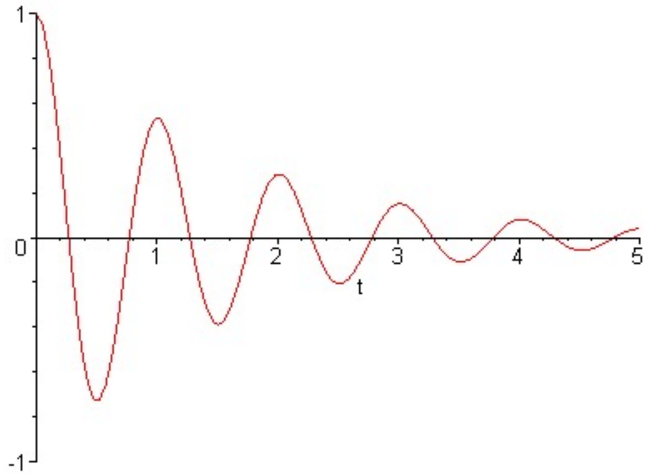
It’s just like a pendulum w/friction periodically tapped by a hammer!





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Seasonal Forcing

Ex: Measles (“*Sarampo*”, “*Mässling*”, “*Mazelen*”, …)

A crude approximation of this seasonality:

Mean transmission force

$$\langle k \rangle \beta(t) = \langle k \rangle \beta_0 (1 + \beta_1 \cos \omega t)$$

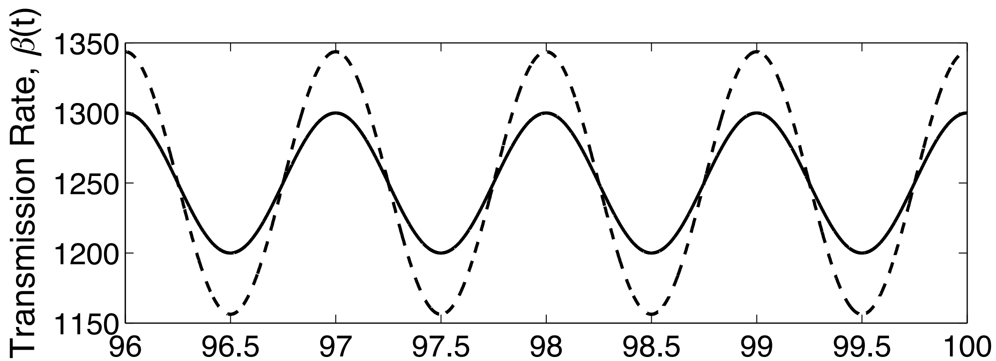
Amplitude of seasonal variation

Birth rate

Death rate

$$\frac{dS}{dt} = B - \beta \langle k \rangle SI - \mu S$$

$$\frac{dI}{dt} = \beta \langle k \rangle SI - \mu S$$



Seasonal Forcing

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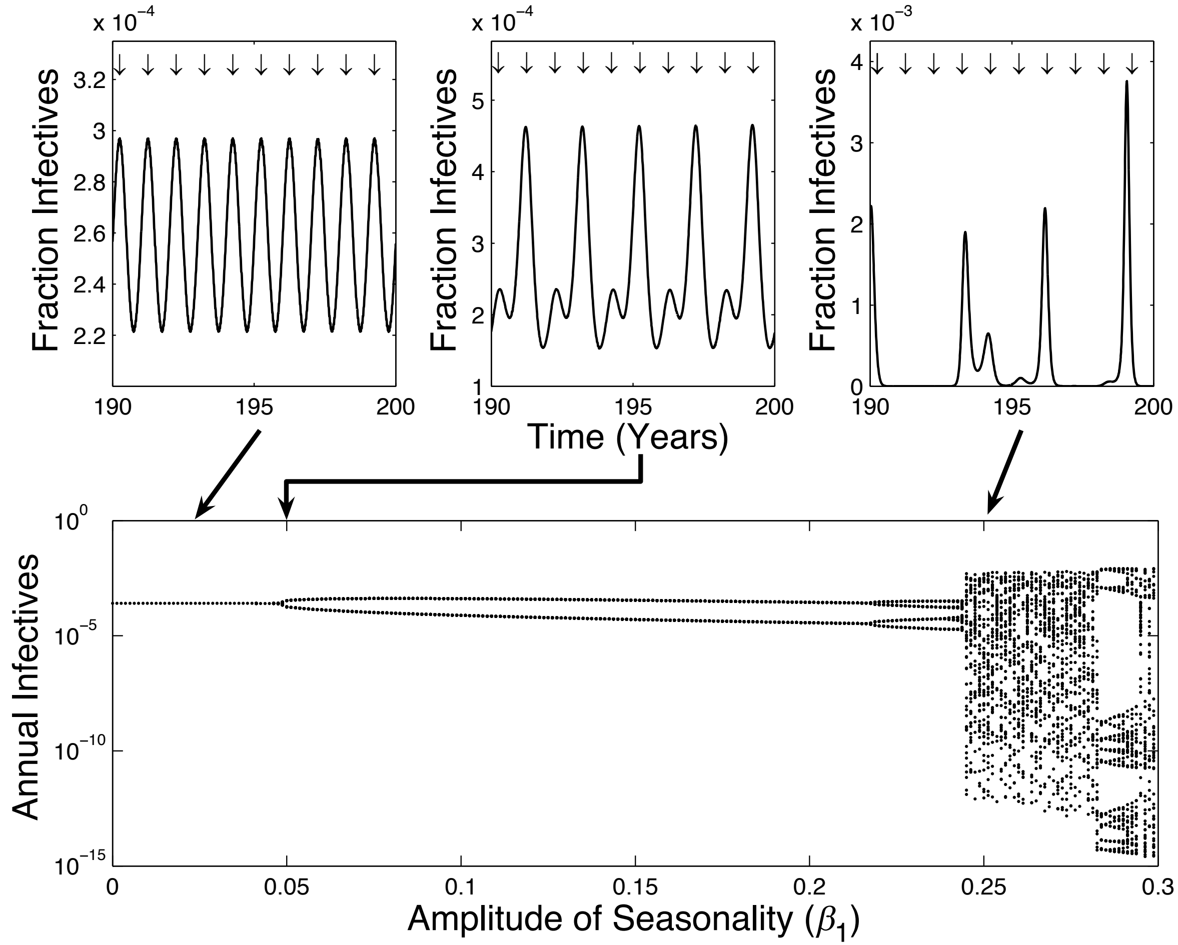
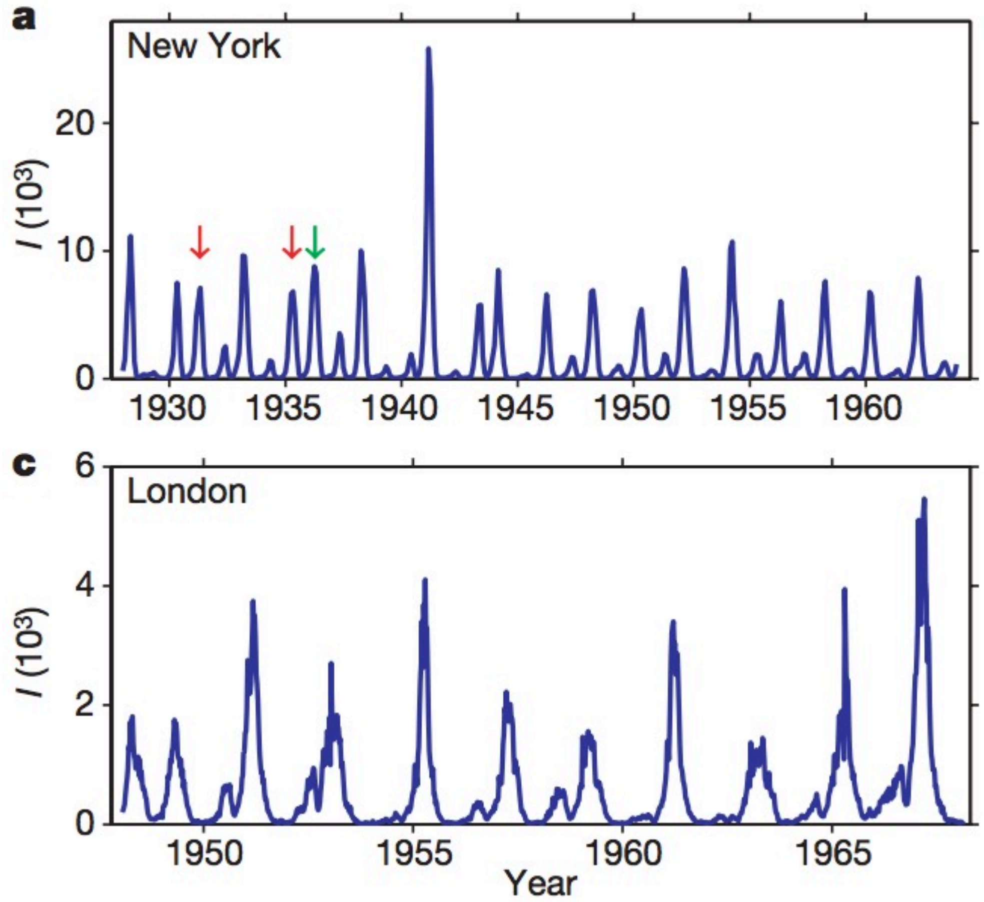


Noise & demographic stochasticity

Ex: Measles (“*Sarampo*”, “*Mässling*”, “*Mazelen*”, …)

- a relatively small amount of noise is sufficient to prevent the oscillations of the basic SIR model from damping out.

- Whether we recast the SIR model as a stochastic process or simply add a small noise term to the deterministic equations, we can sustain the oscillations.



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What do I mean?

The nano-fox problem & determinist dynamics

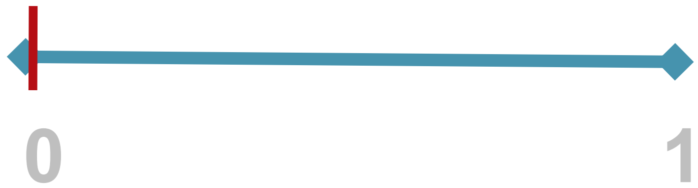
...populations are finite

Finite populations

$\Delta x = 1/N \neq dx$

Stochastic description

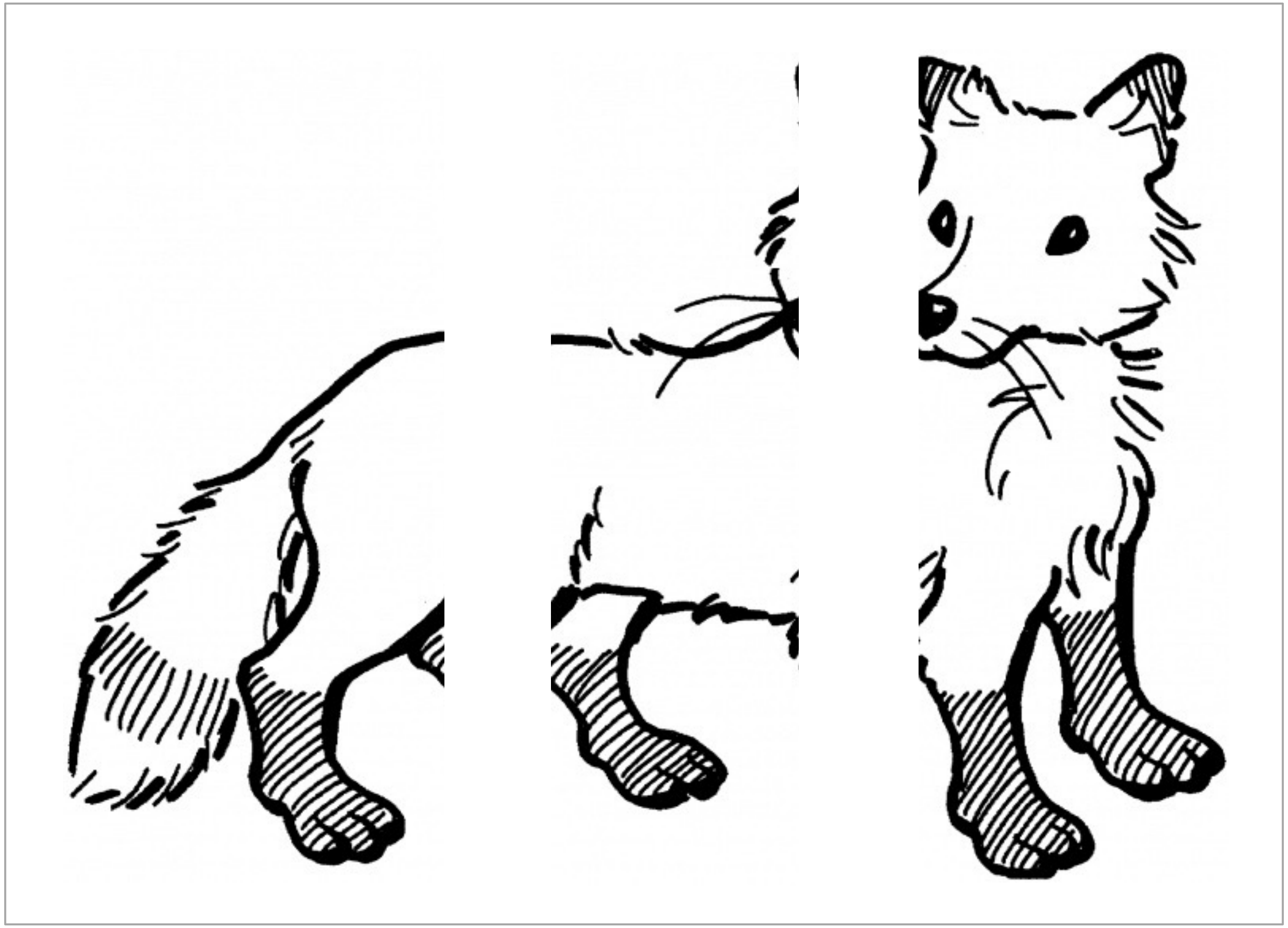
Infinite populations

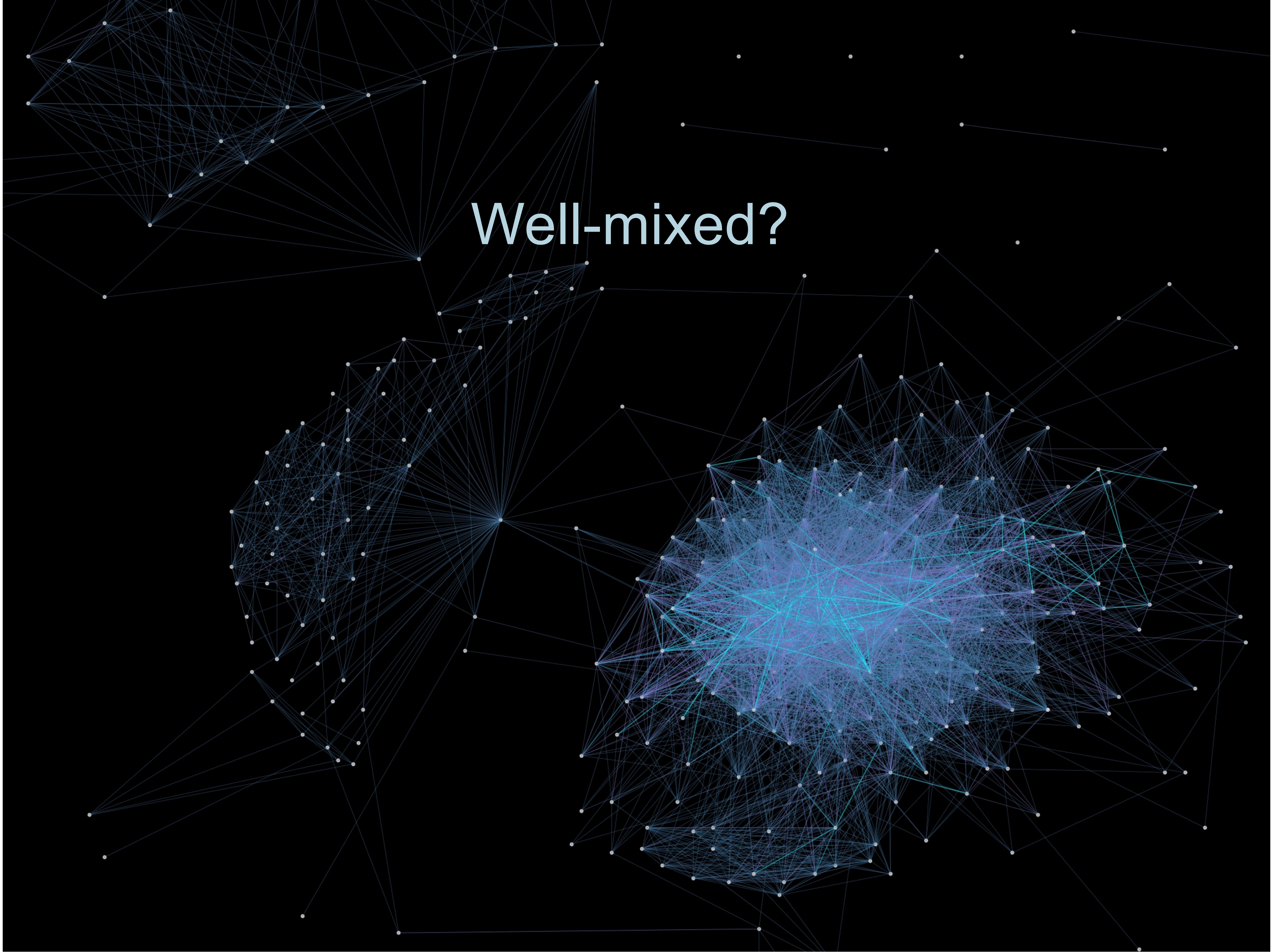


$\Delta x = 1/N = dx$

Deterministic description

The nano-fox problem & determinist dynamics




Well-mixed?