DEI
DEPARTAMENTO
DE ENGENHARIA INFORMÁTICA
TÉCNICO LISBOA

Disease spreading

part II

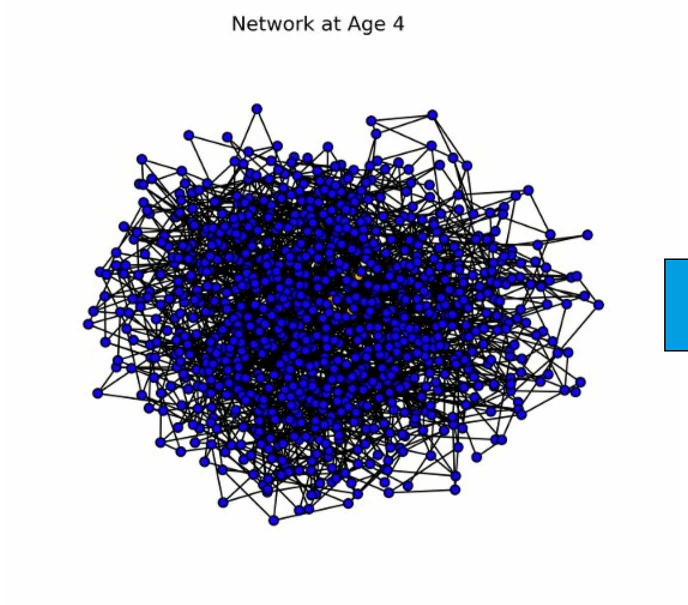
Network Science, 2021/2022

Today

Part I (last class)

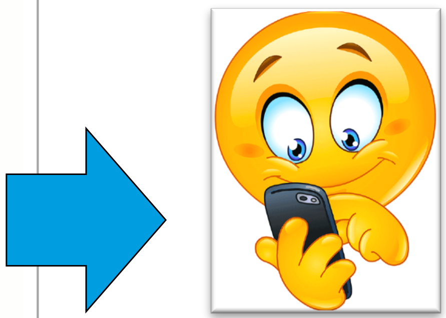
Introduction to theoretical epidemiology

Part II



Network epidemics

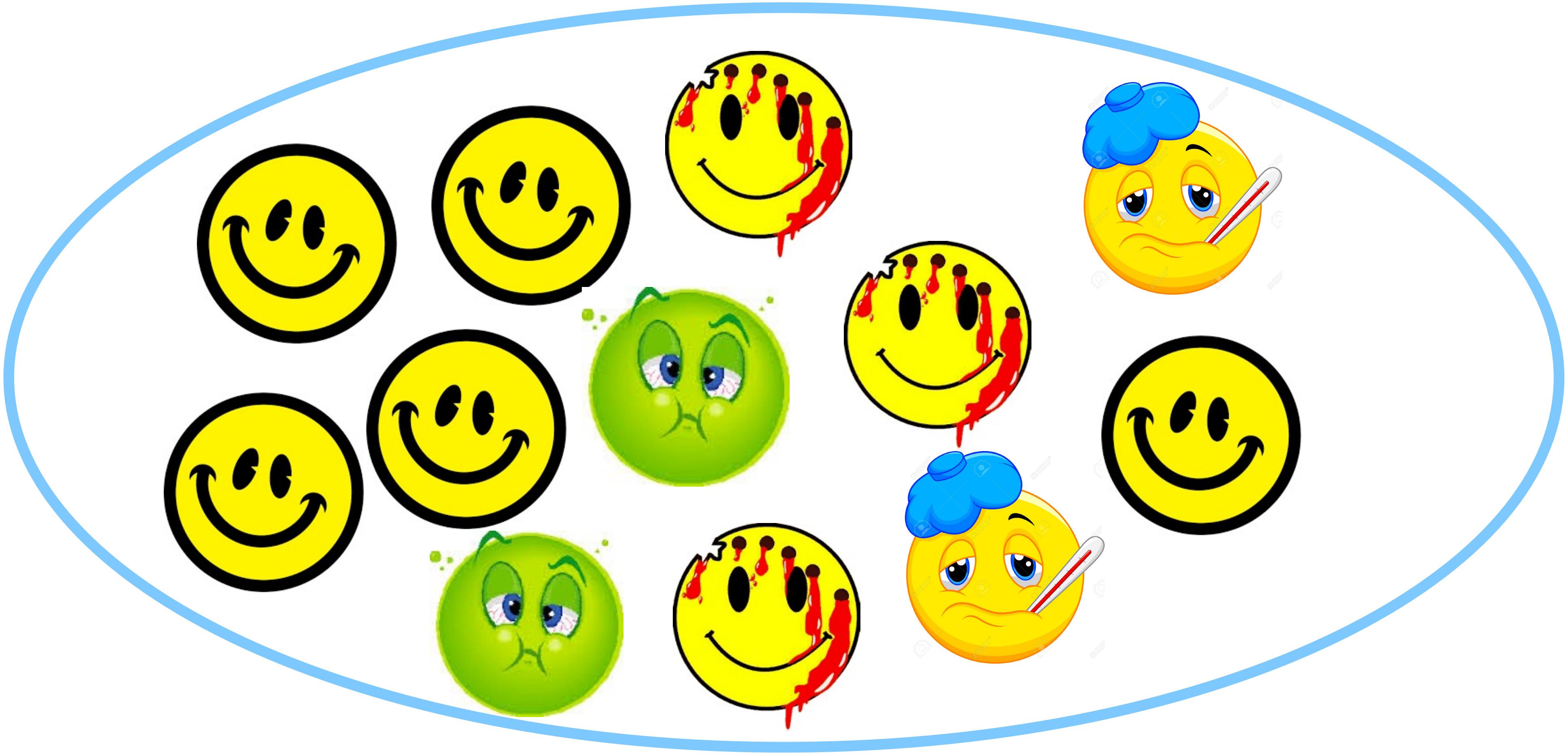
Part III



Social Dynamics

Compartment models of disease spreading

Consider a population where each individual has a disease state

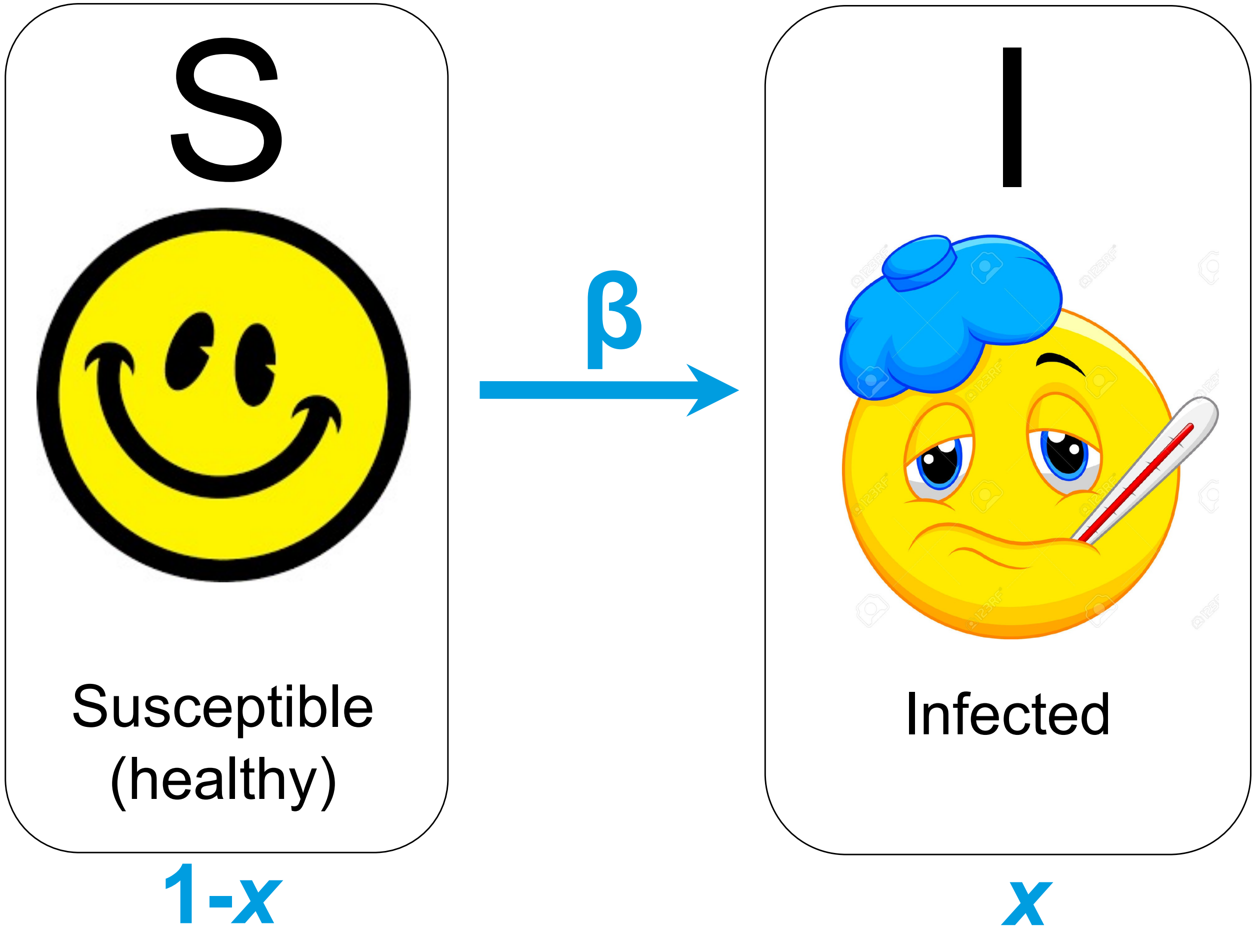


SI model

Ex: AIDS

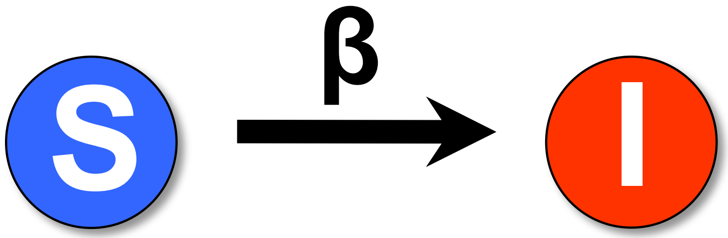
β: infection rate per contact

The most elementary model…



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



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

Transmission rate / force of infection = 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

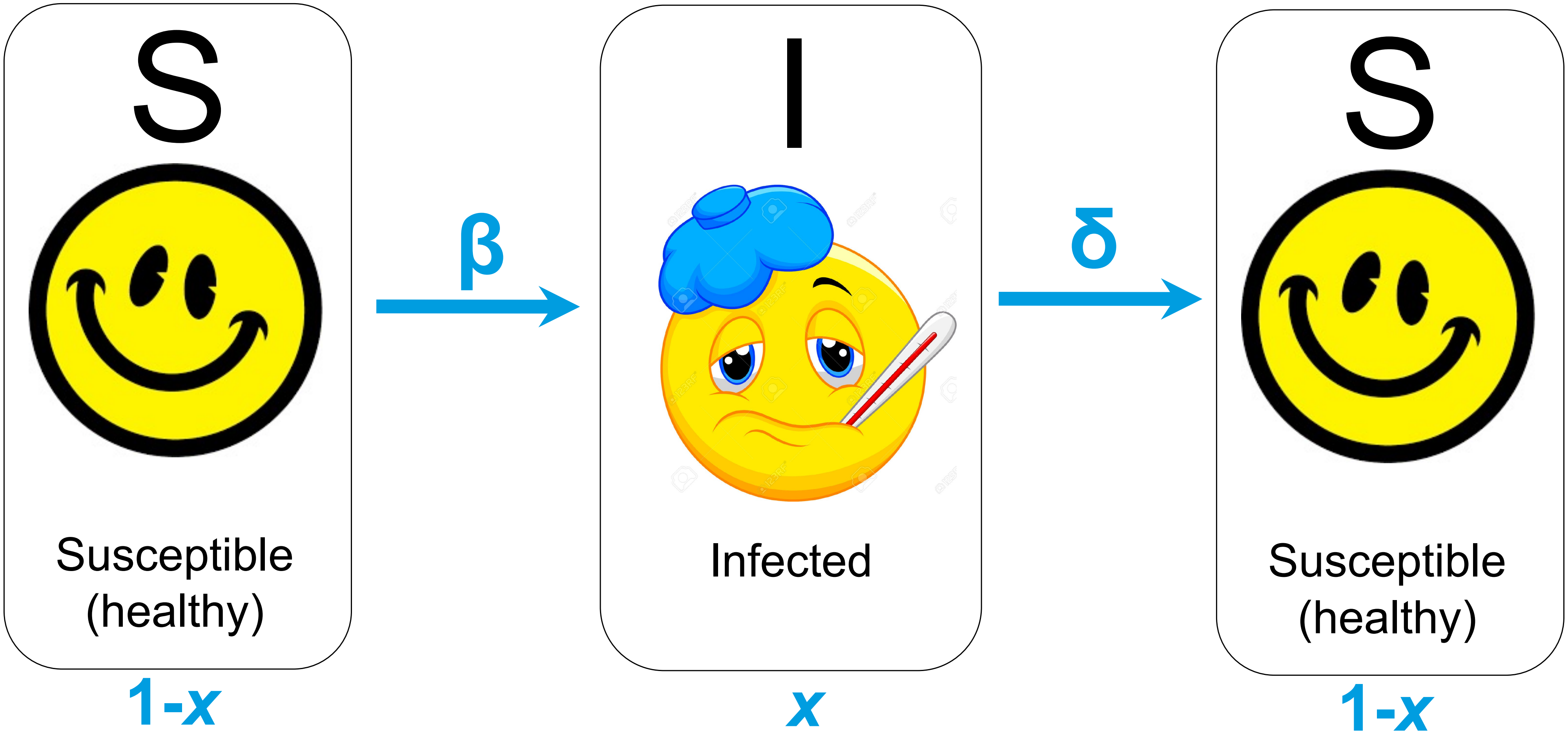
$$\tau = \left(\beta\langle k\rangle\right)^{-1}$$

SIS model

Ex: gonorrhea, athlete's foot, …

β: contact infection rate
δ: recovery rate

Let’s add recovery!



SIS model

β: contact infection rate
δ: recovery rate

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

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

<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

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

- Endemic state:

  For low recovery rate, the disease will never disappear.

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

- Disease free-state:

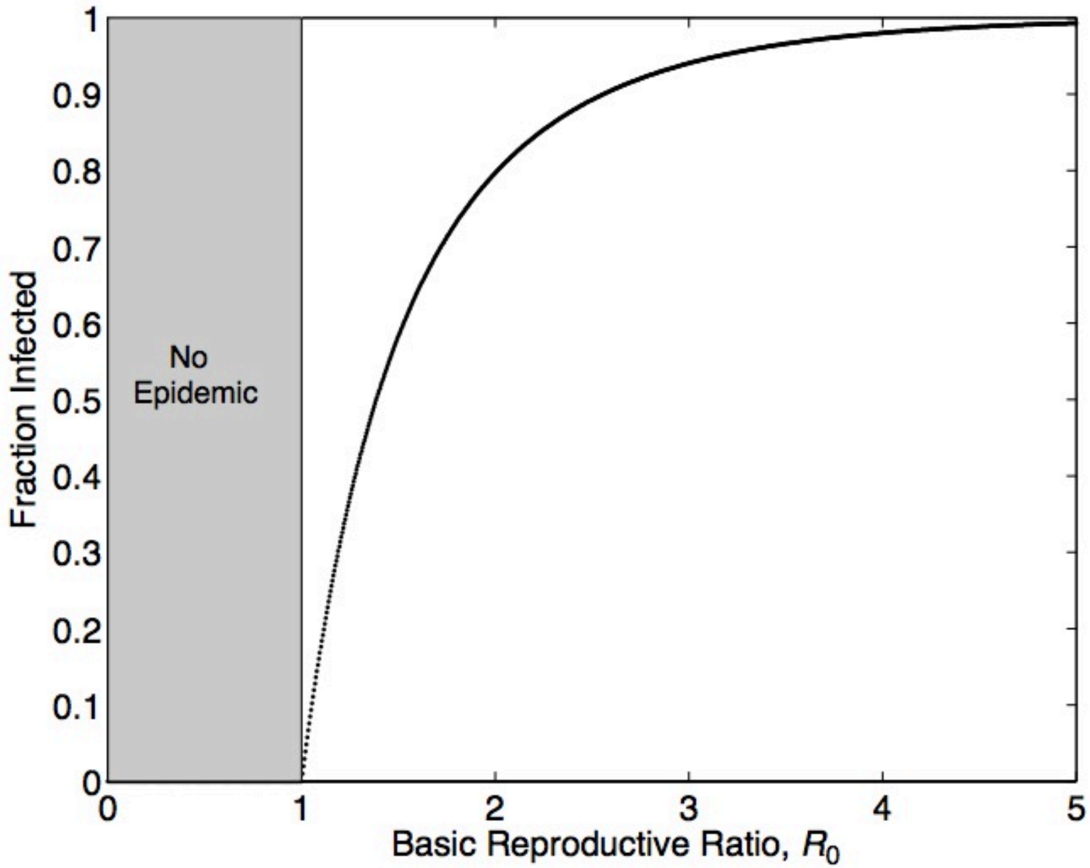
  For large recovery rates the number of new infections will be lower than the number of new recovered individuals, and the disease decreases exponentially in time.

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

The basic reproductive number

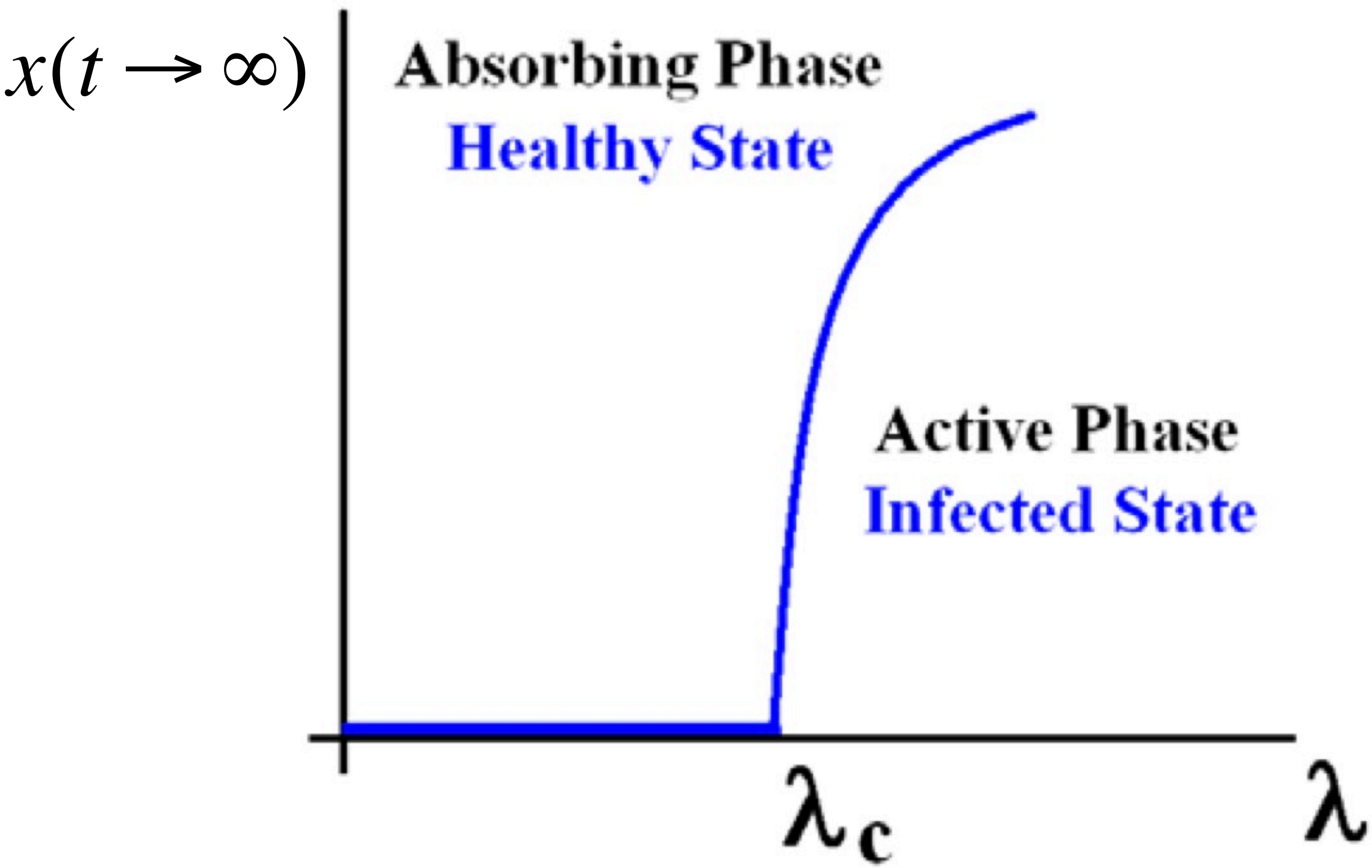
The reproductive number $\boldsymbol{R_0}$ provides the number of individuals an infected infects if all its contacts are susceptible.

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



Epidemic threshold

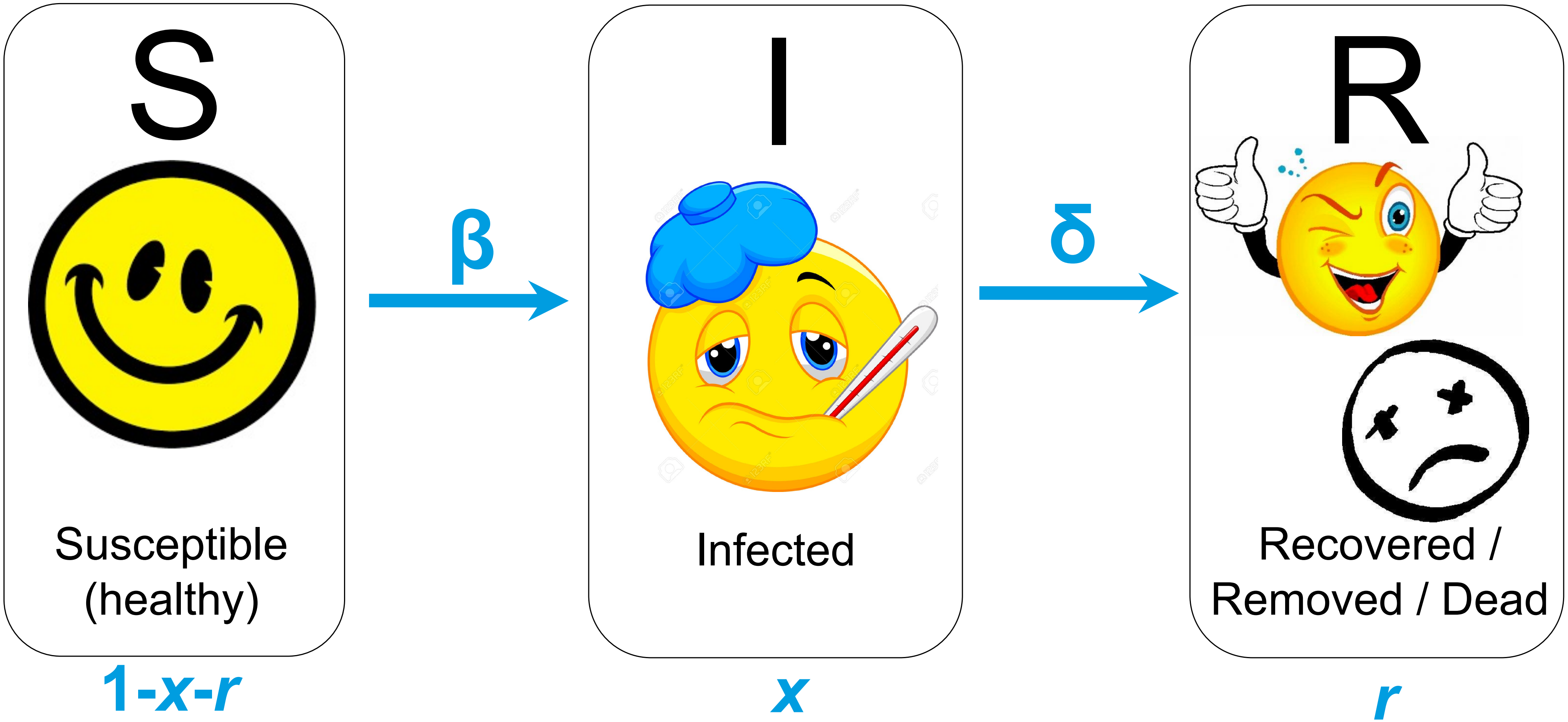
Equivalently, one may define an epidemic threshold $\lambda_c = \beta_c / \delta$ which also splits the phase space into the endemic state and the healthy state.



SIR model

β: contact infection rate
δ: recovery rate

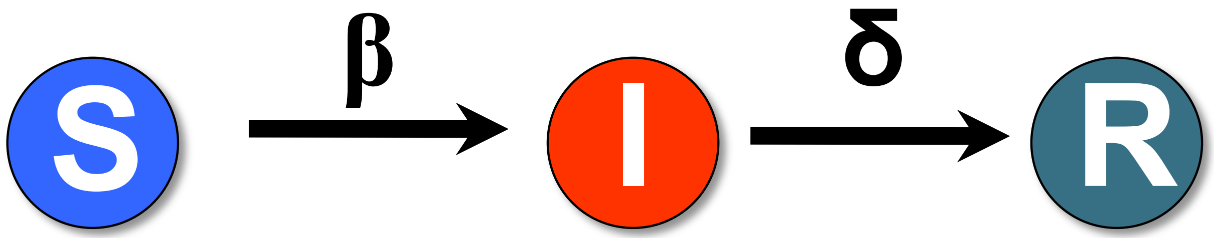
Often individuals develop immunity after recovery
(e.g., Influenza) or can be removed from the population.



Traditional models

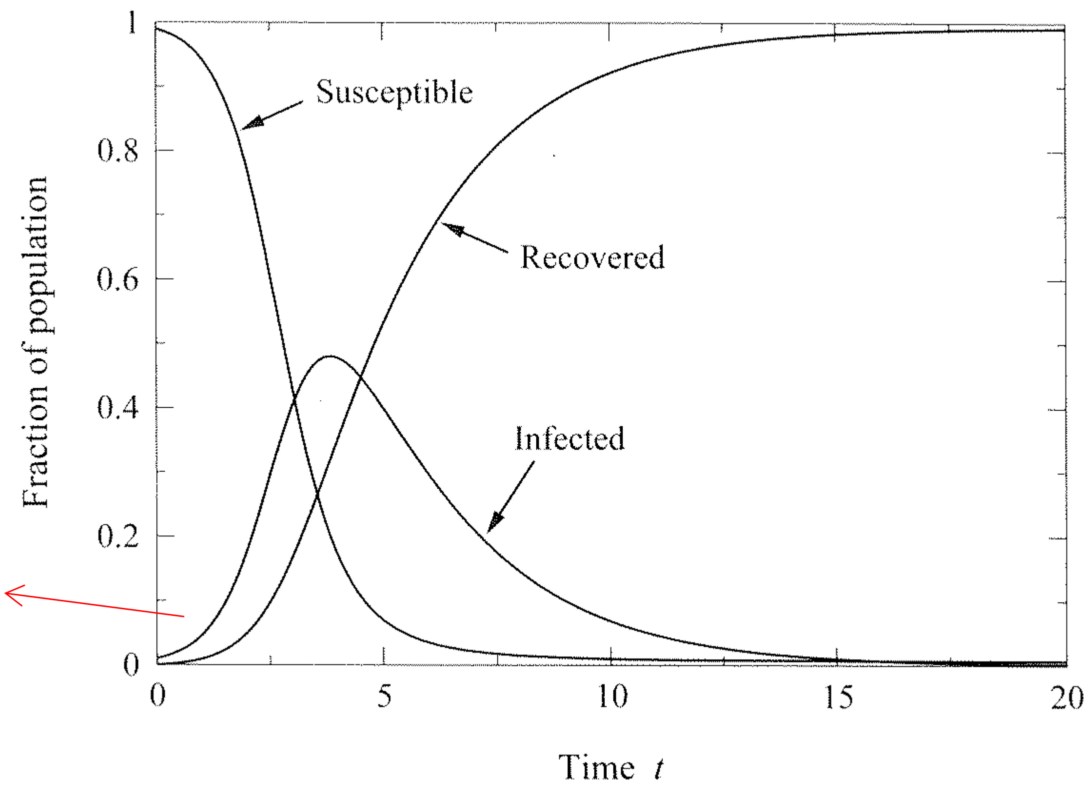
β: contact infection rate
δ: recovery rate

SIR model



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

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



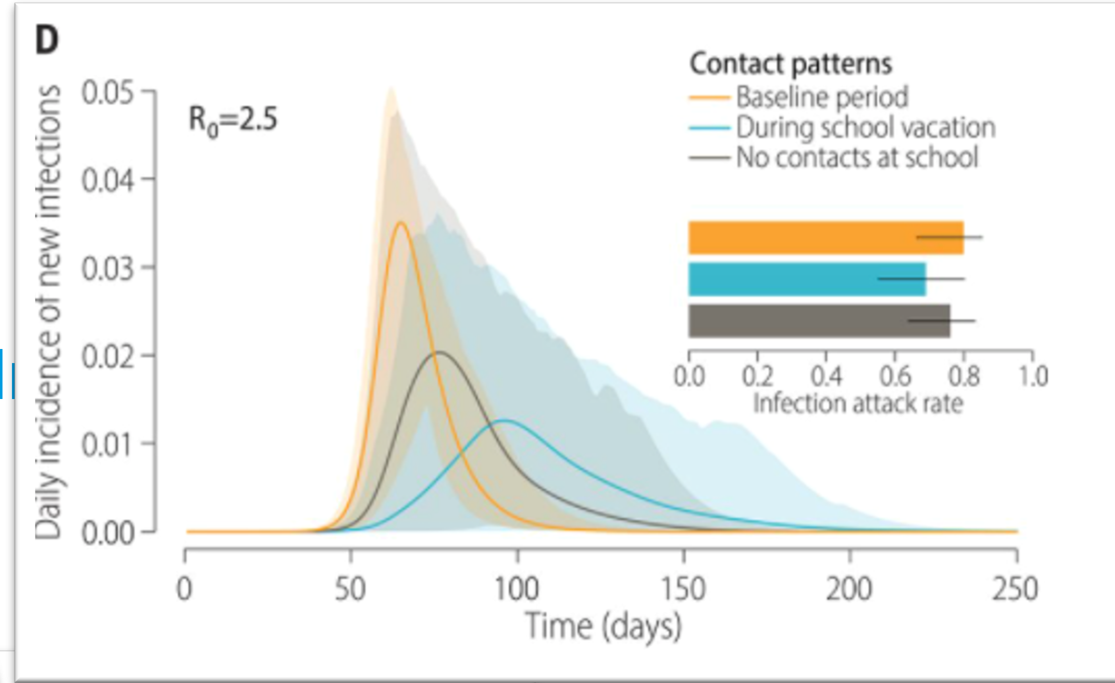
$$R_0 = \frac{\beta\langle k\rangle}{\delta} > 1$$

Epidemic control

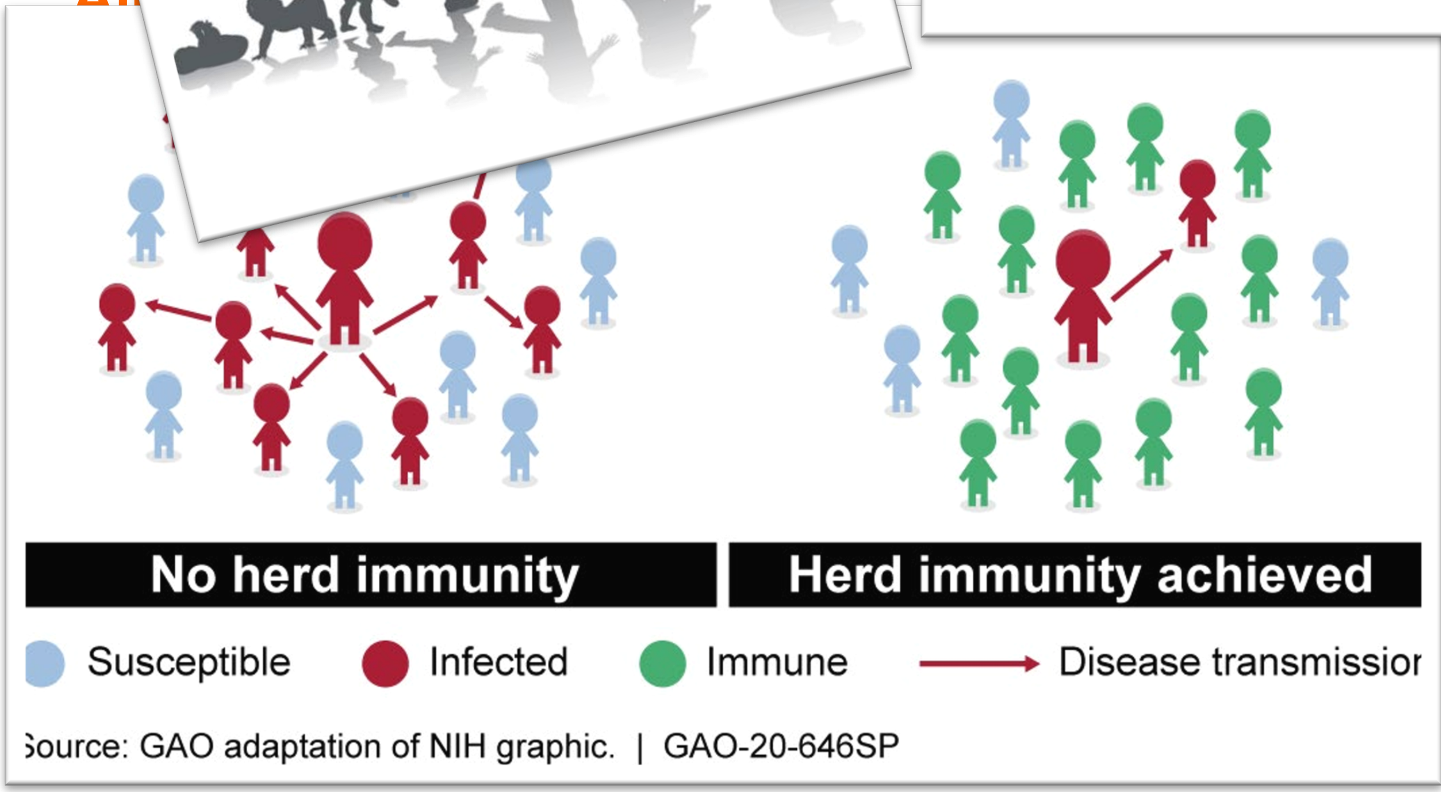
Aiming at β

Tran… g I…

F… ng,



Ai…



ource: GAO adaptation of NIH graphic. | GAO-20-646SP

…n quarantine
…d public spaces, like
…owns.

…nks. Reduces the
…dies out.

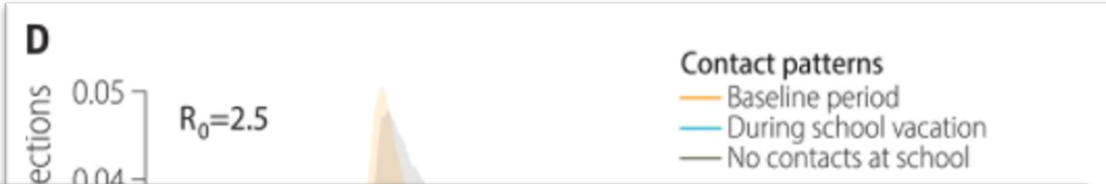
Epidemic control

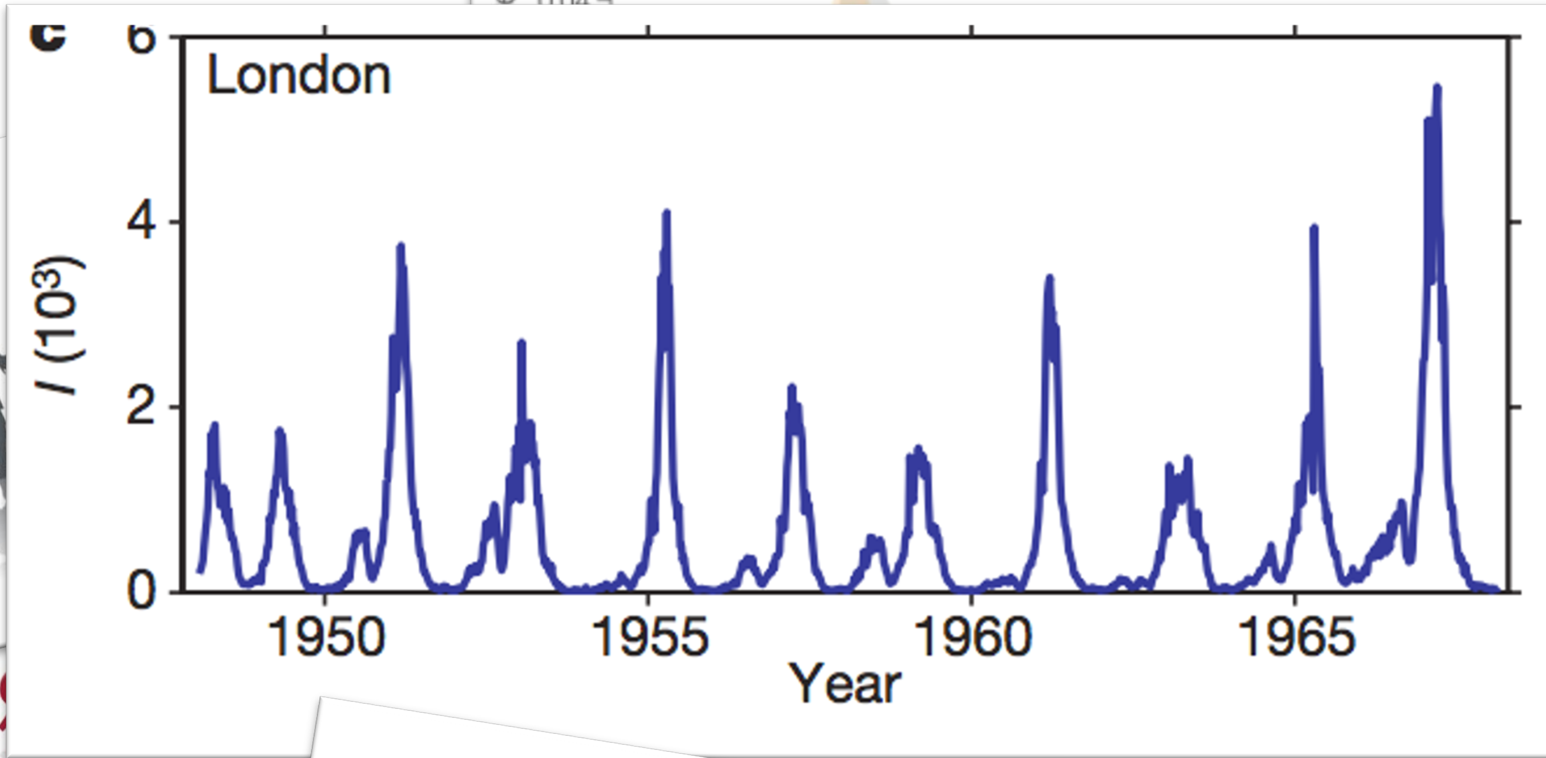
Aiming at β

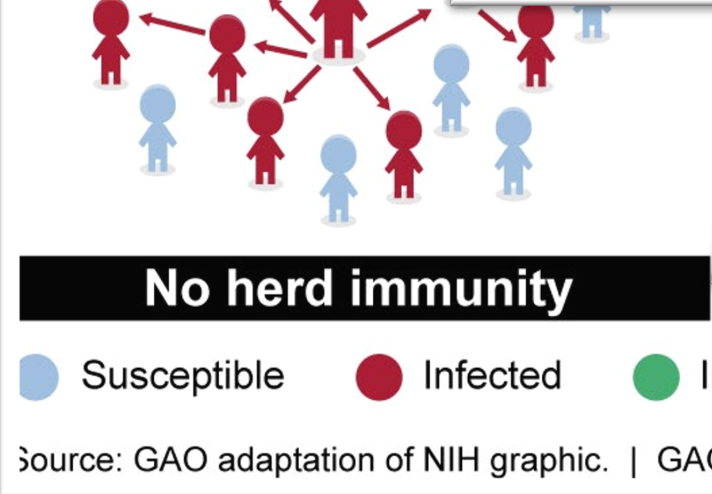
Tran

F

Ai



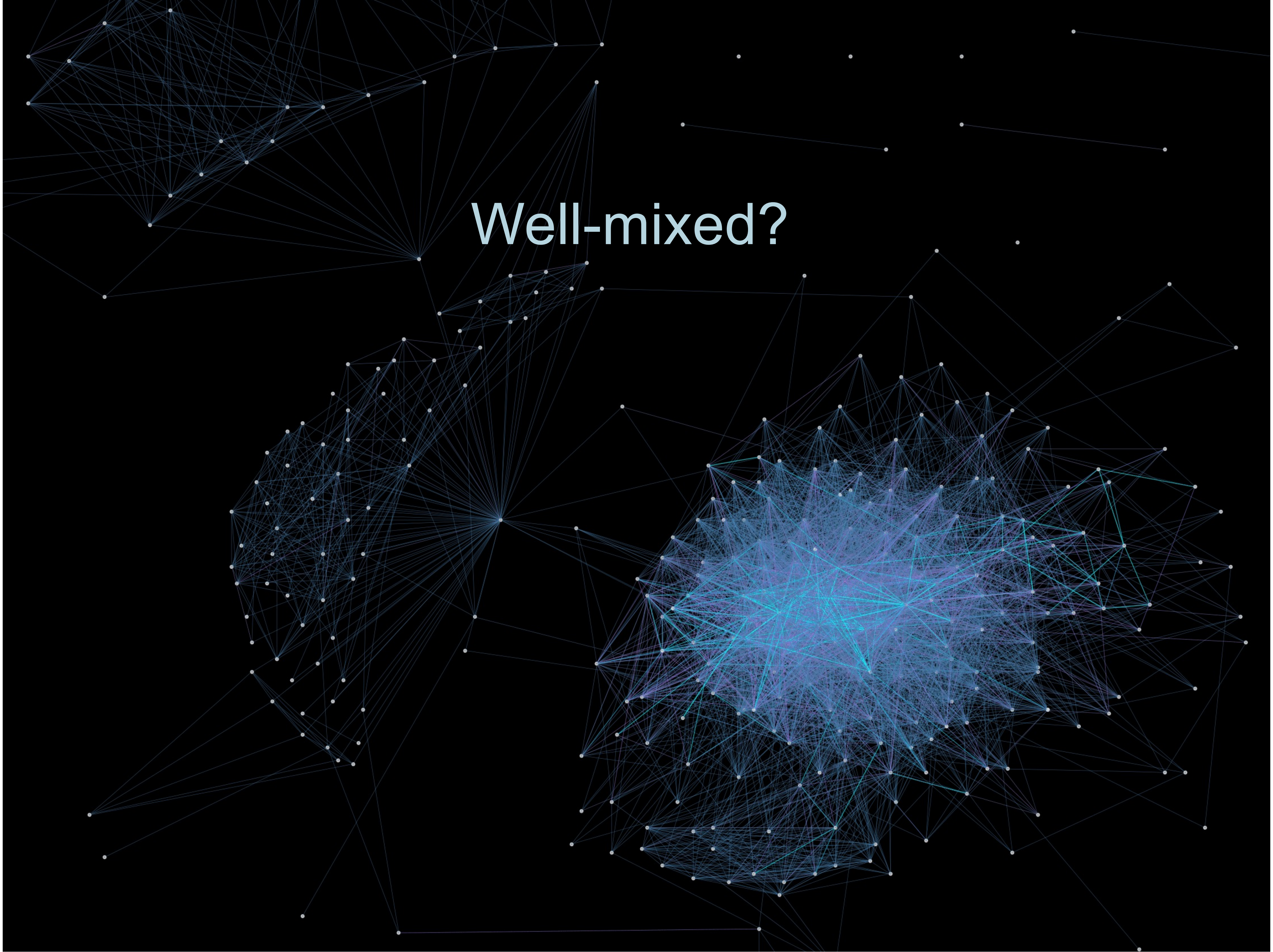




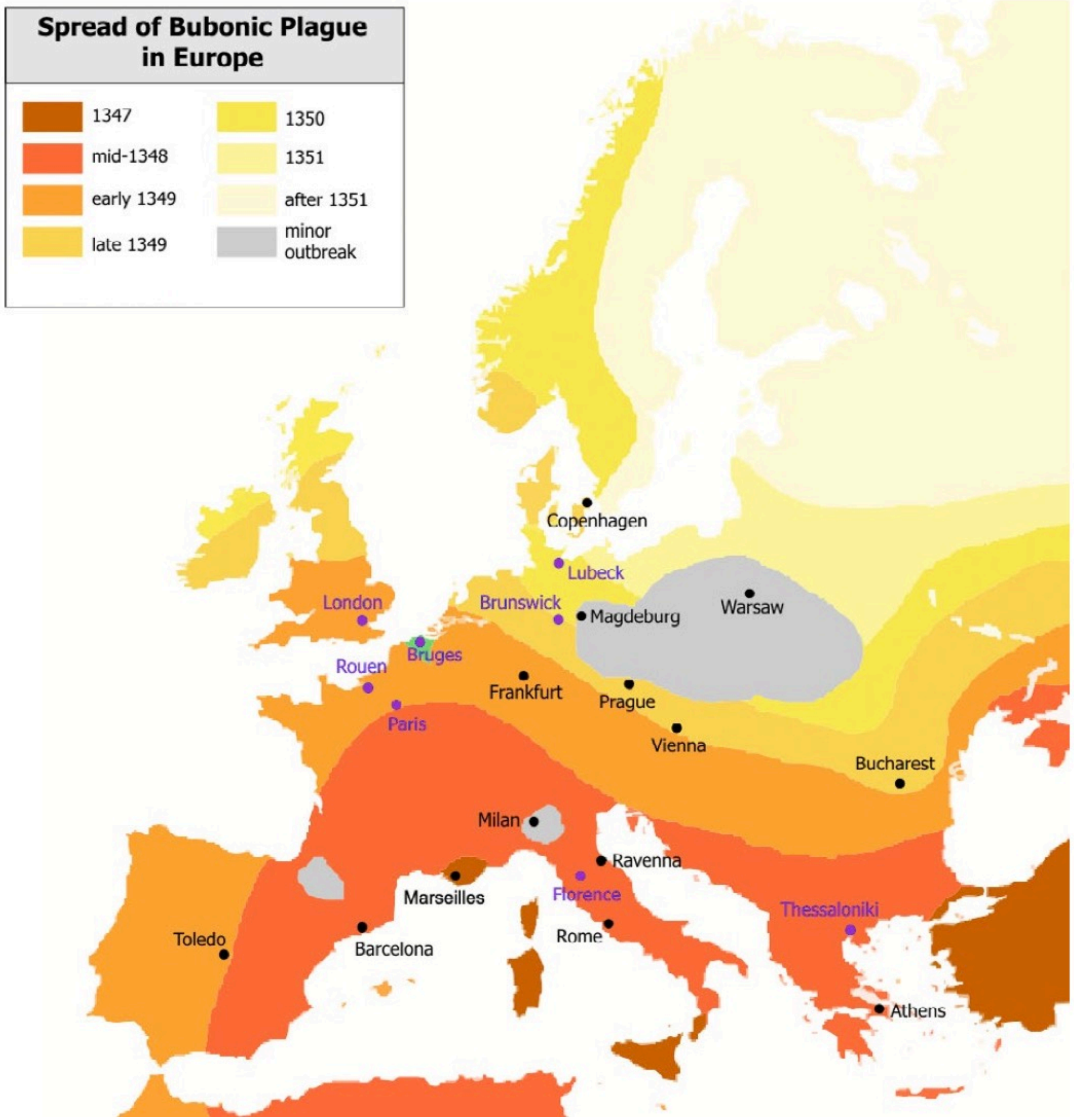
owns.

dies

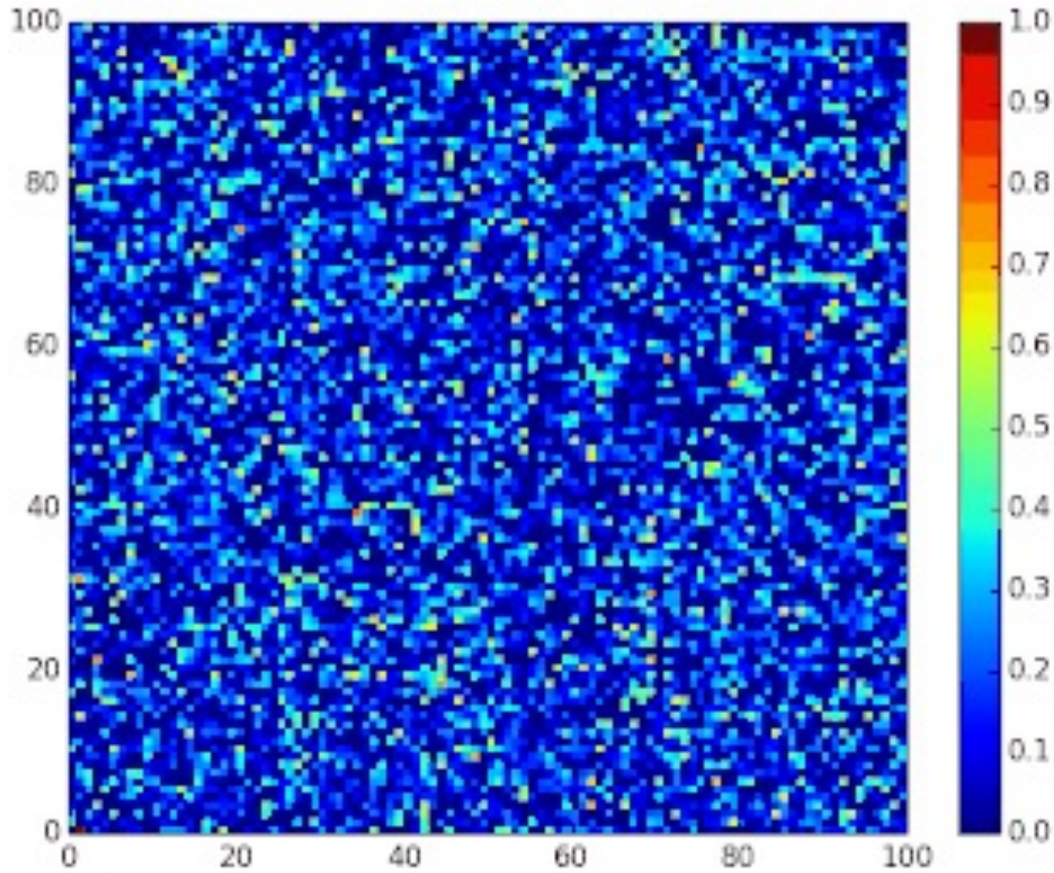
Well-mixed?



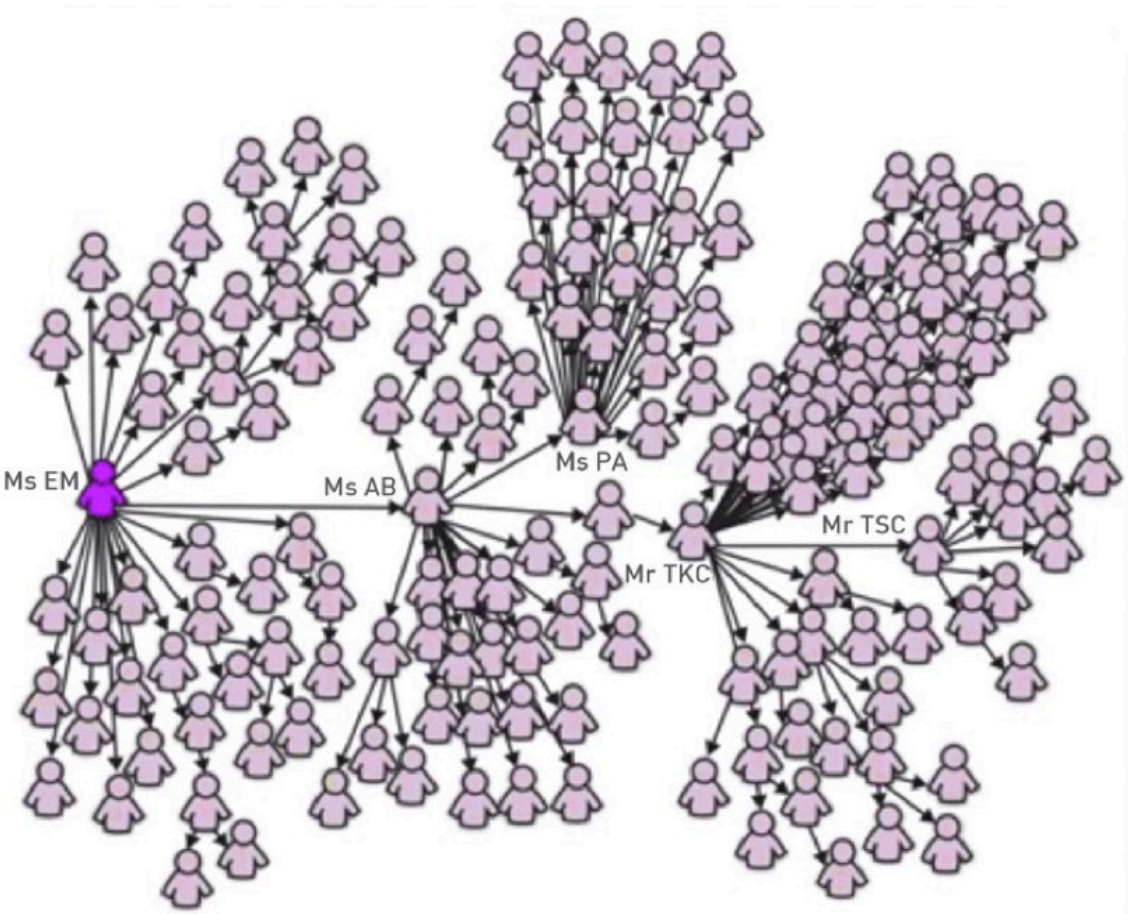
Spatial spreading



Spatial SIR model simulation

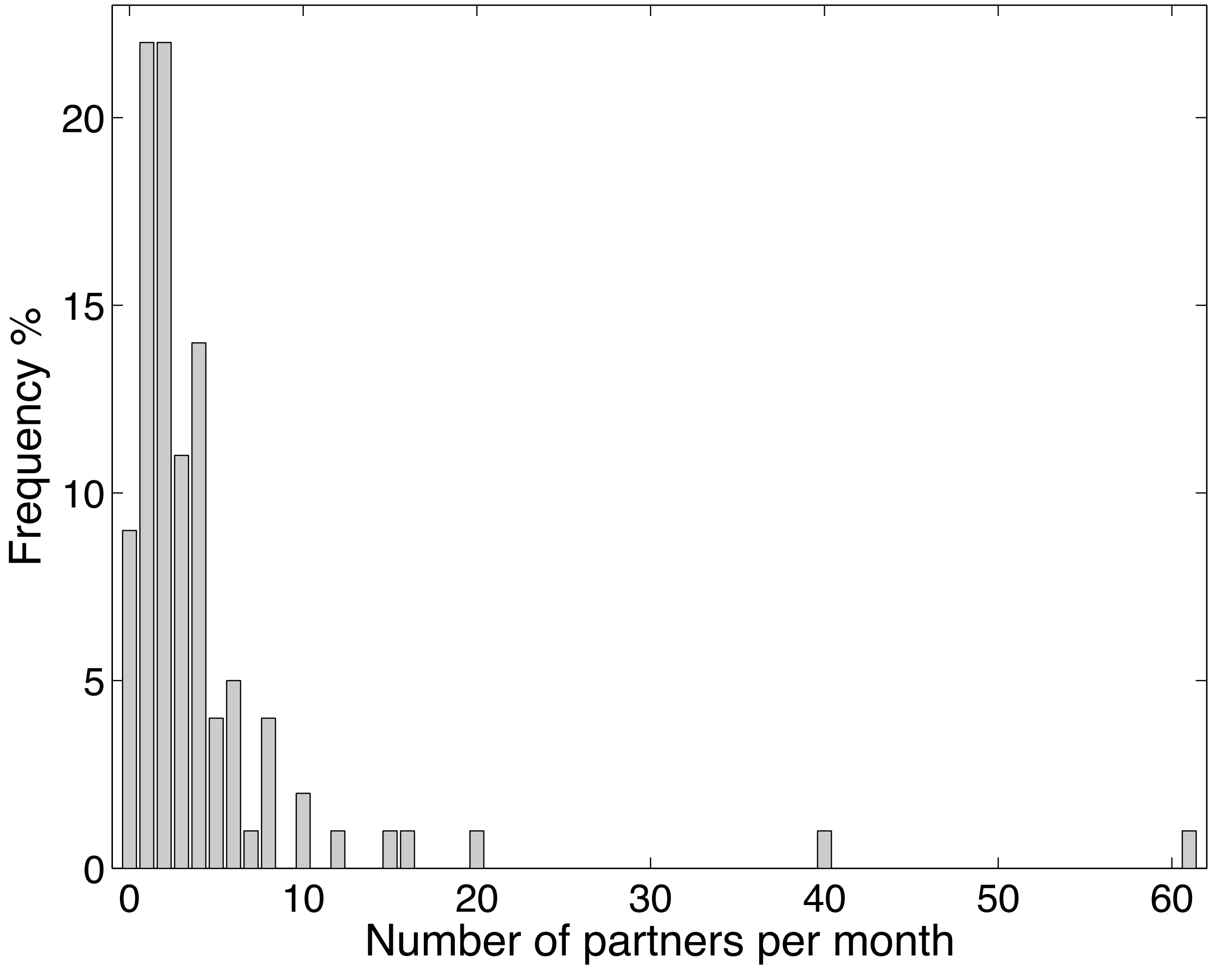


Heterogeneous networks and super-spreaders



Sexually transmitted infections (STIs)

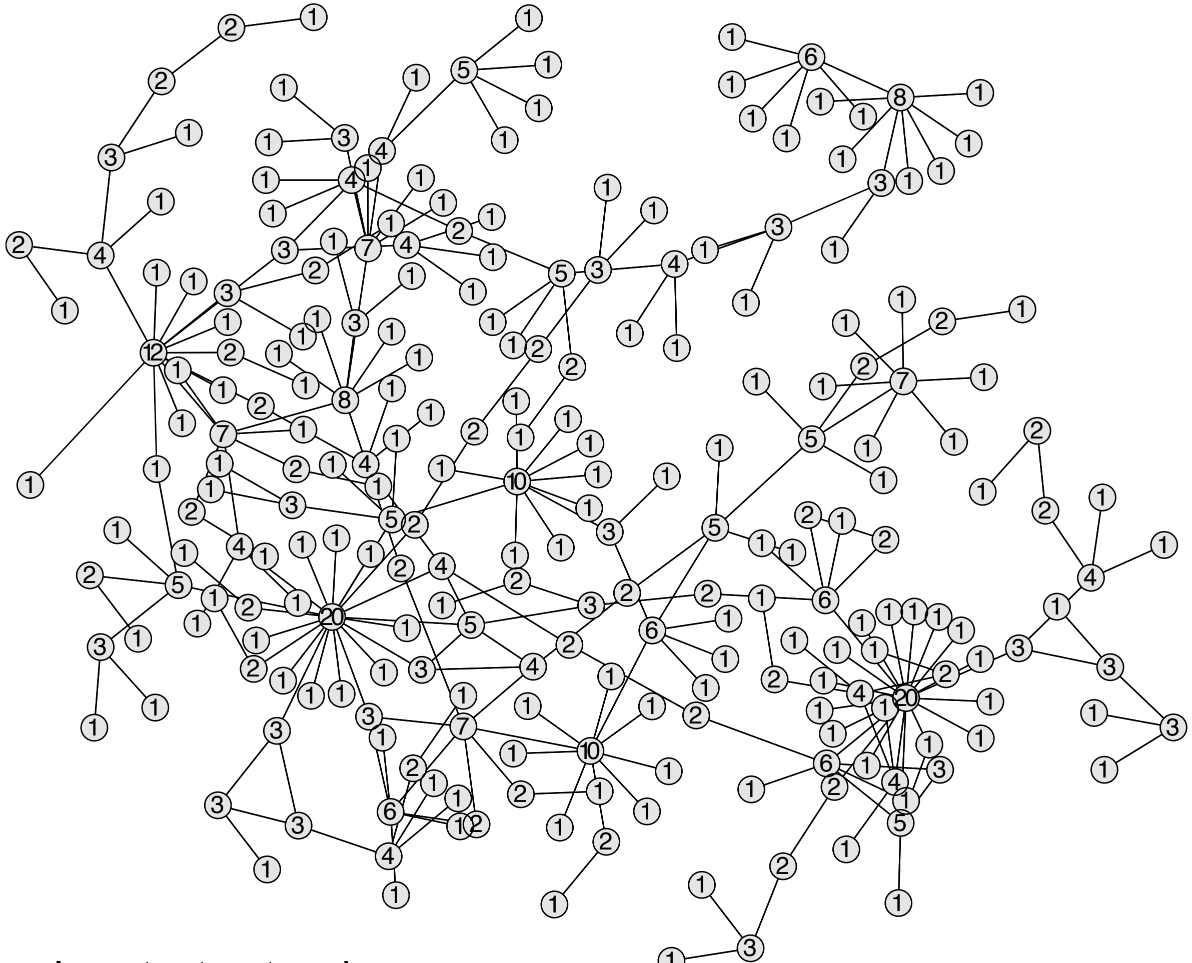
Networks are everywhere



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)

Sexually transmitted infections (STIs)

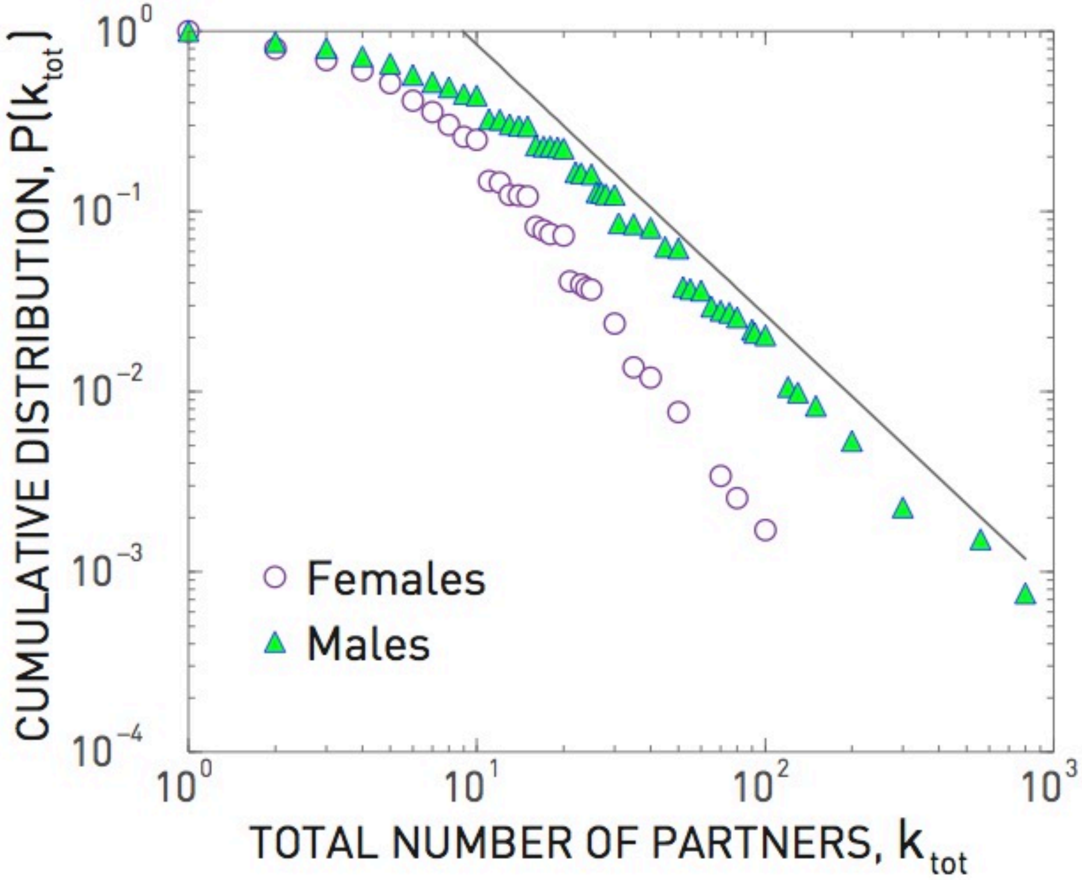
Networks are everywhere



An example of a sexual contact network,
taken from the study of HIV transmission in Colorado Springs
(Potterat et al. 2002)

Sexually transmitted infections (STIs)

Networks are everywhere



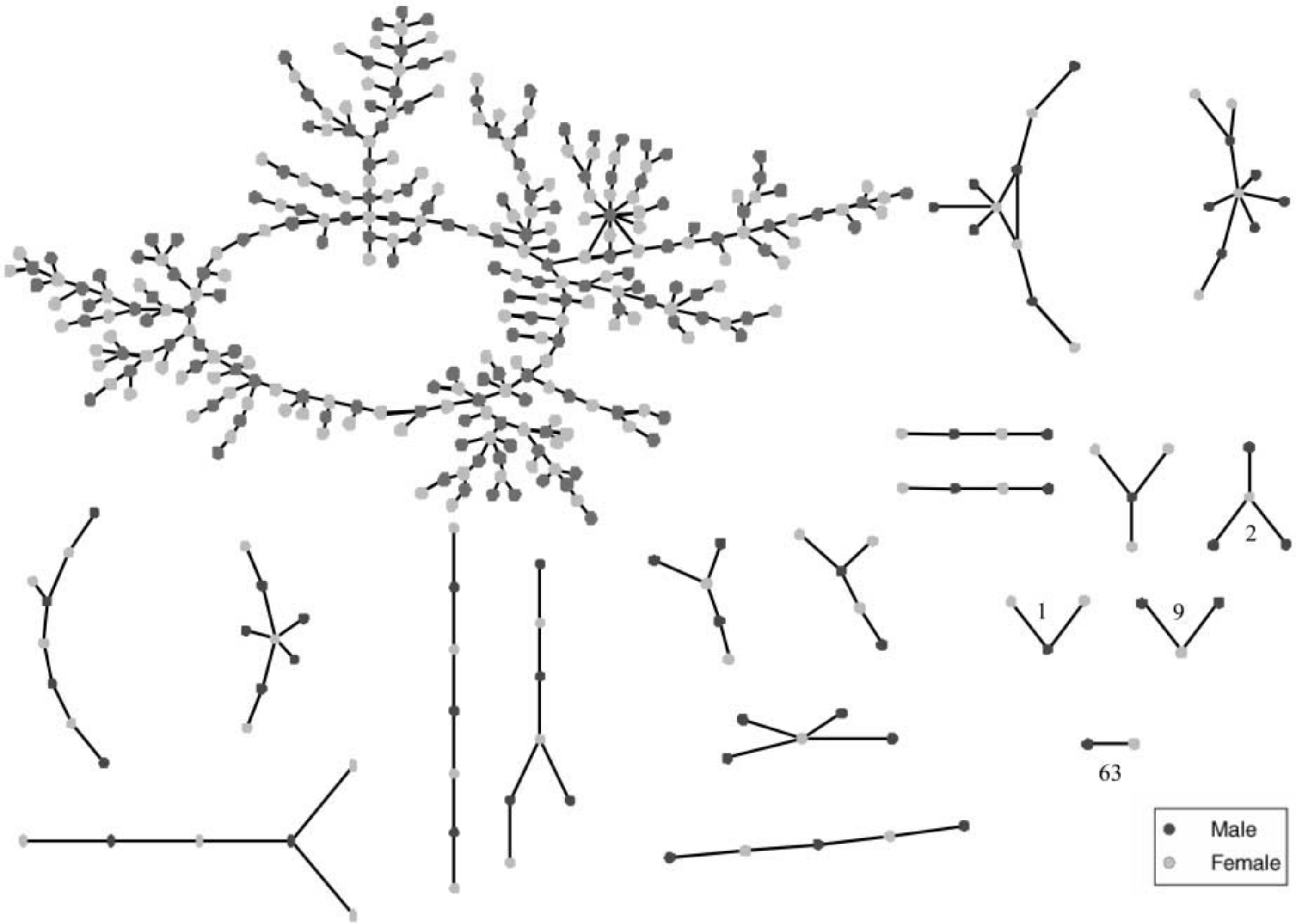
Fredrik Liljeros
Dep. Sociology,
Stockholm Univ.

Liljeros, F., Edling, C. R., Amaral, L. A. N., Stanley, H. E., & Åberg, Y. (2001). The web of human sexual contacts. Nature, 411(6840), 907-908.

Sexually transmitted infections (STIs)

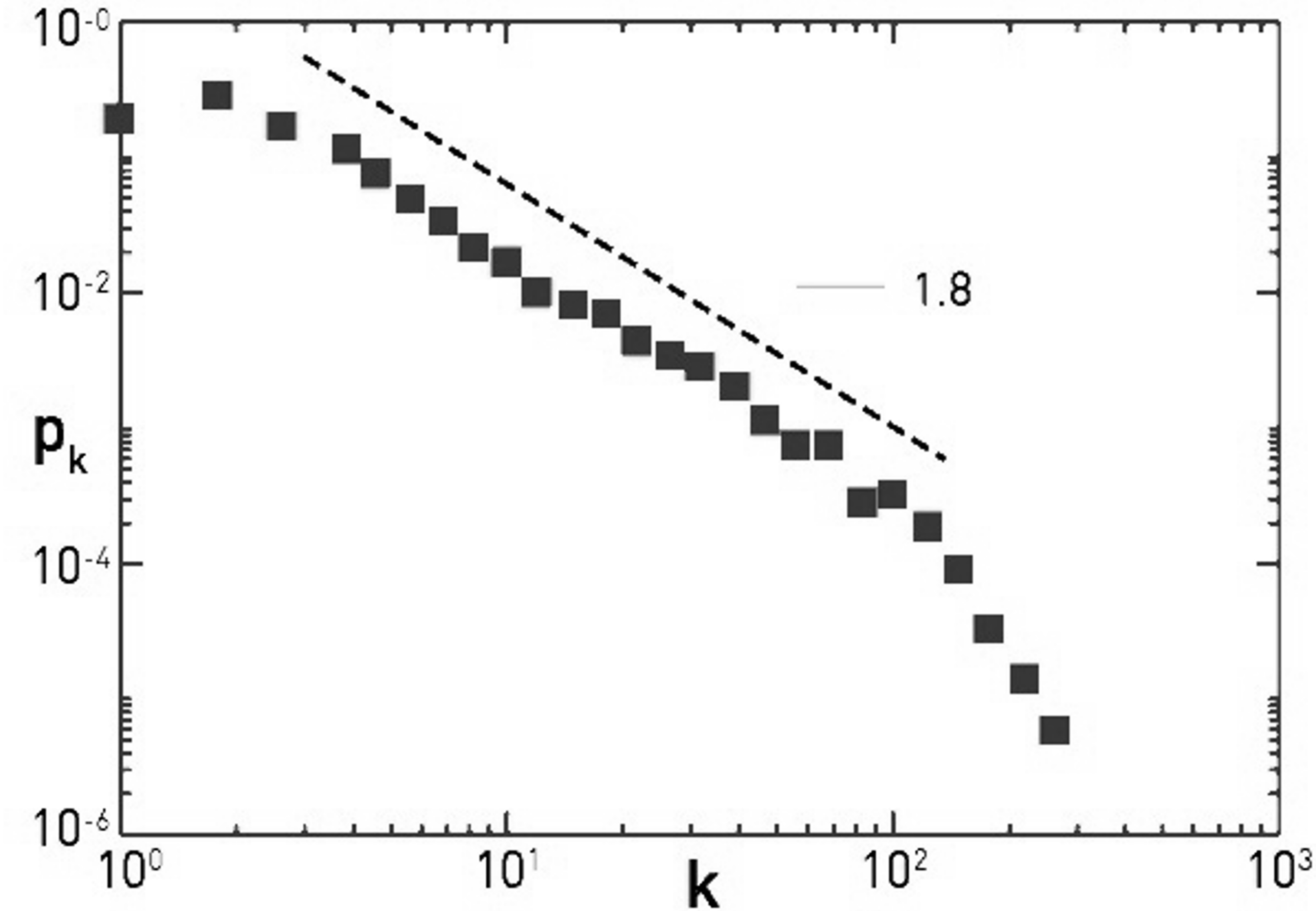
Connected components

P. S. Bearman, J. Moody, and K. Stovel. Chains of affection: the structure of adolescent romantic and sexual networks. Am J Sociol., 110:44-91, 2004.



Global contact networks

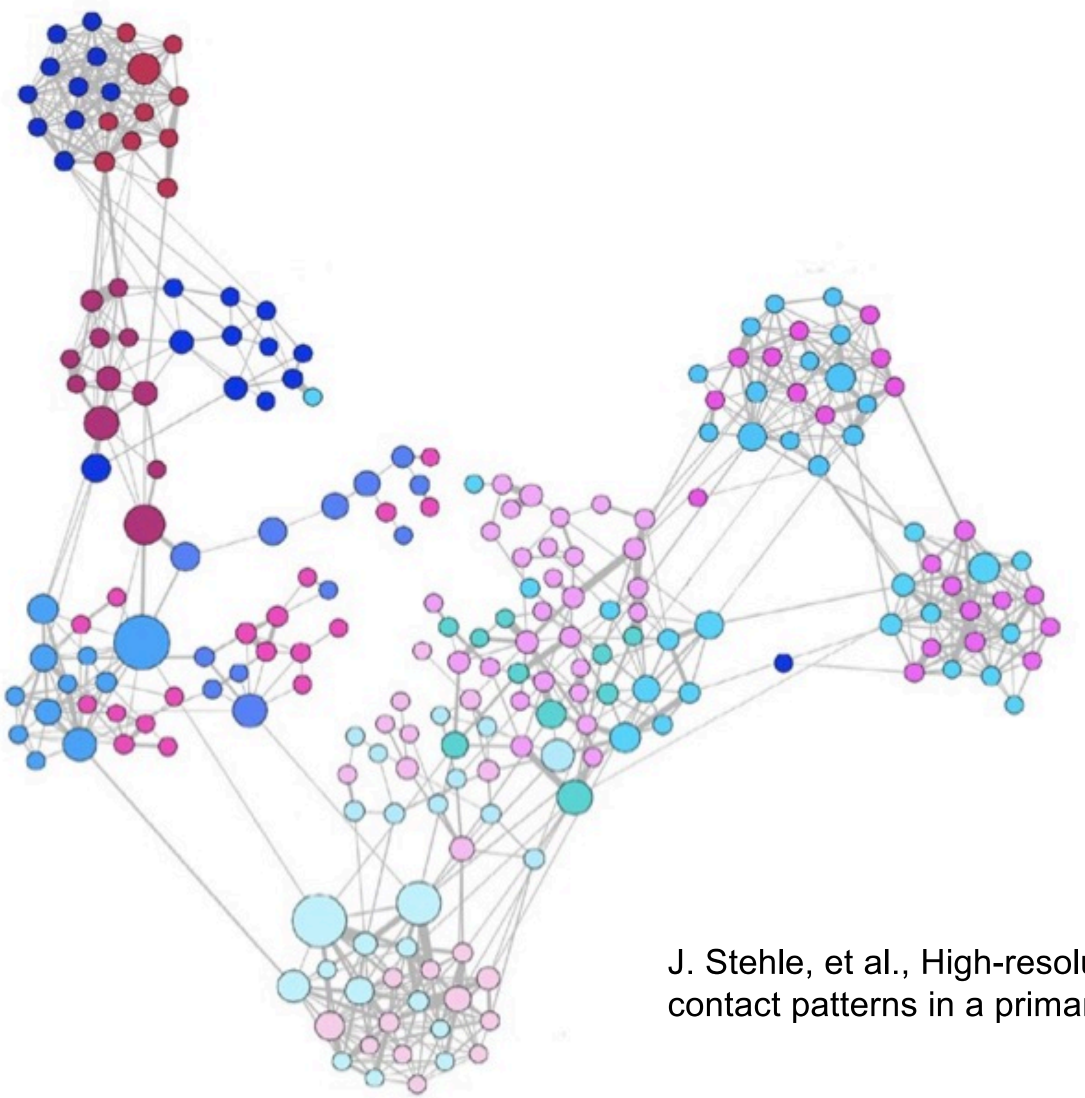
Air transportation network



Local contact networks

Face-to-face interactions (RFID experiments)

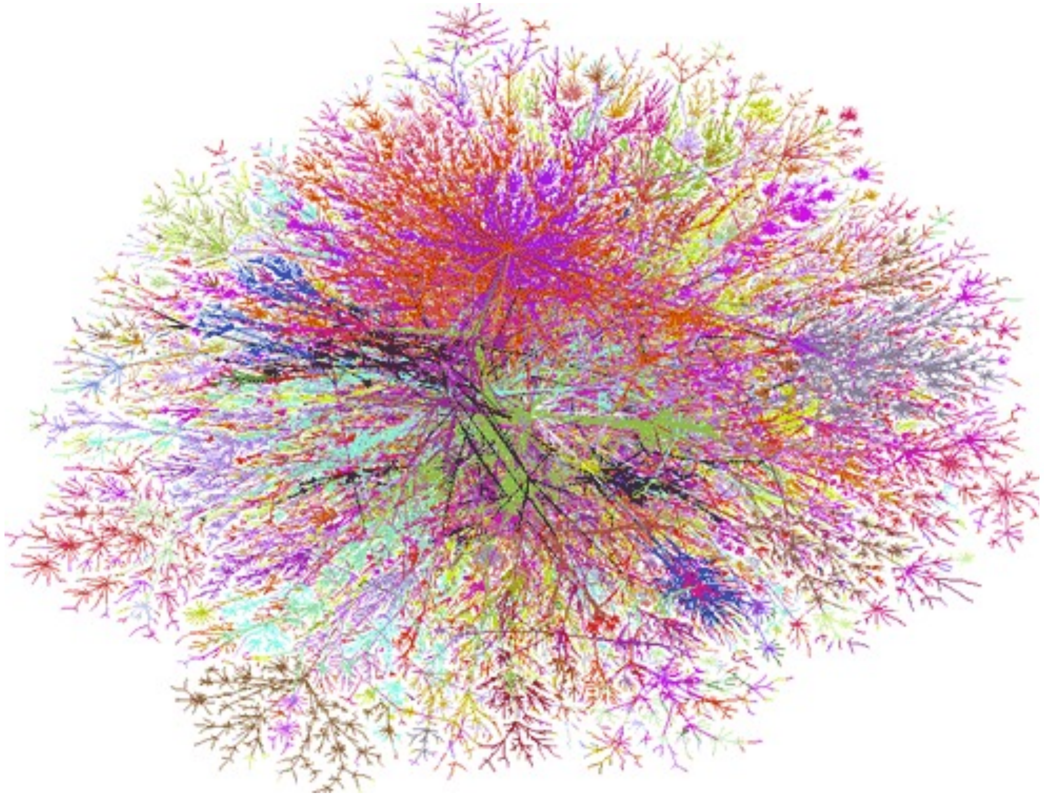
Radio-frequency identification



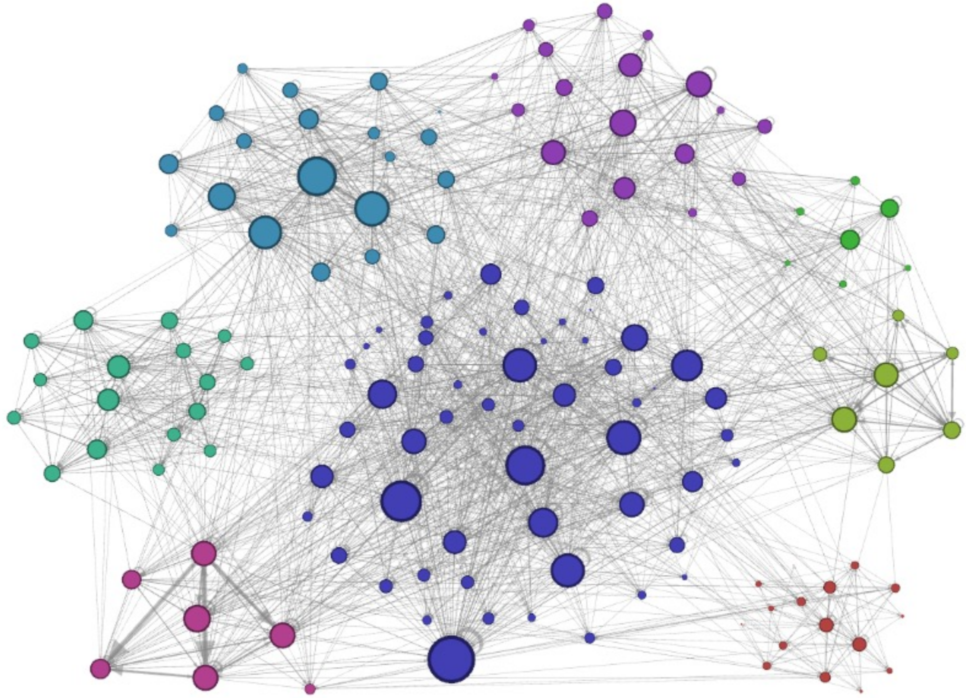
J. Stehle, et al., High-resolution measurements of face-to-face contact patterns in a primary school. PLoS ONE, 6:e23176, 2011.

Computer viruses, mobile phone viruses, etc.

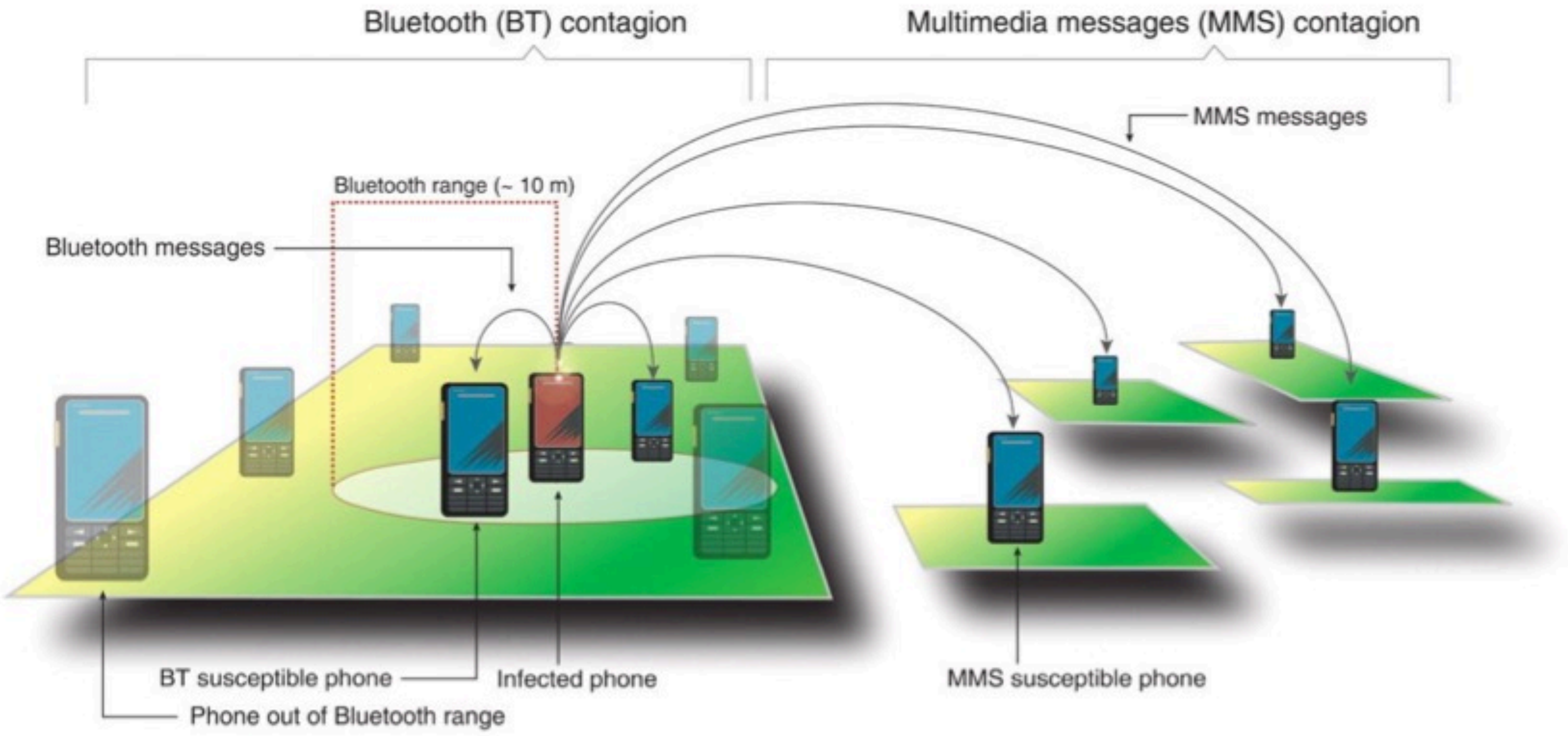
Internet



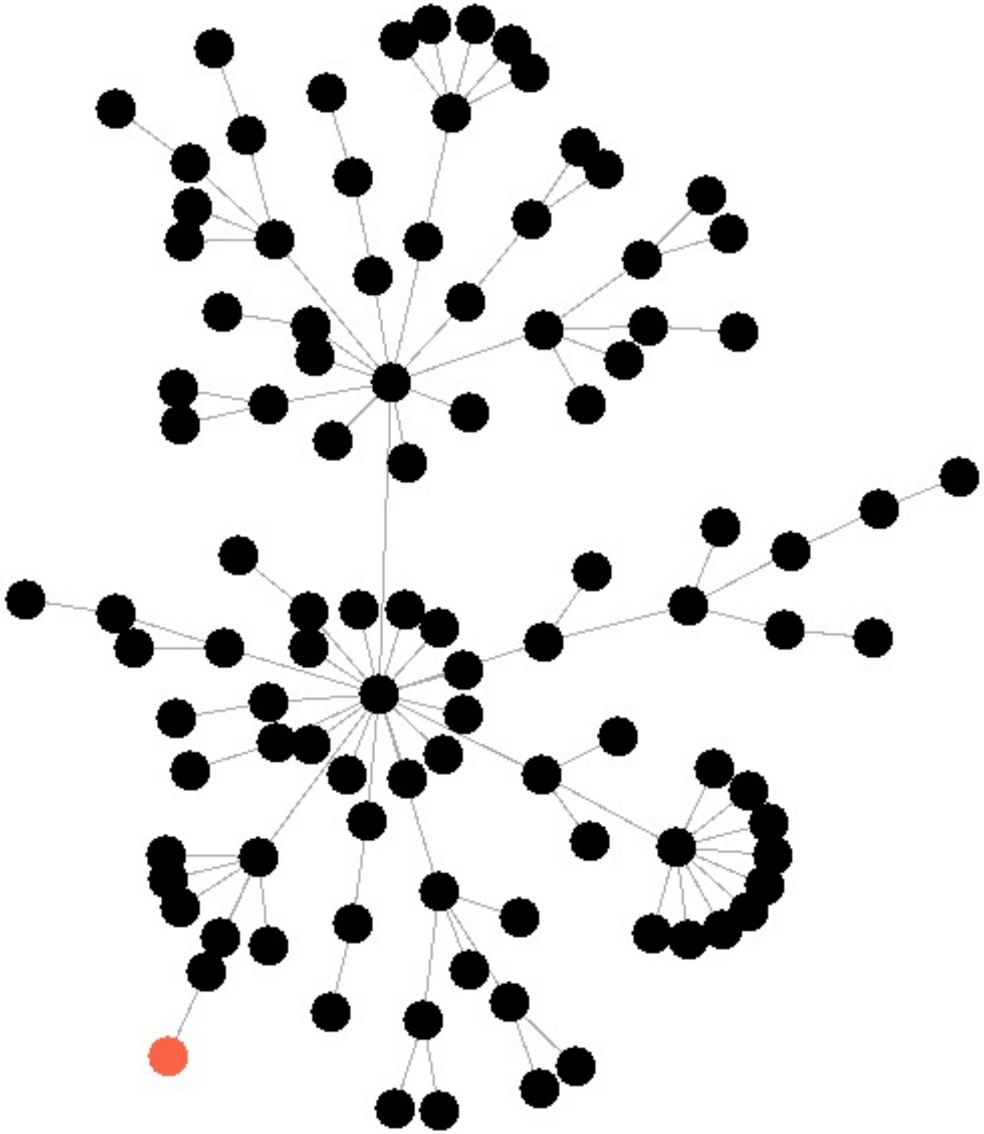
Email networks



Computer viruses, mobile phone viruses, etc.

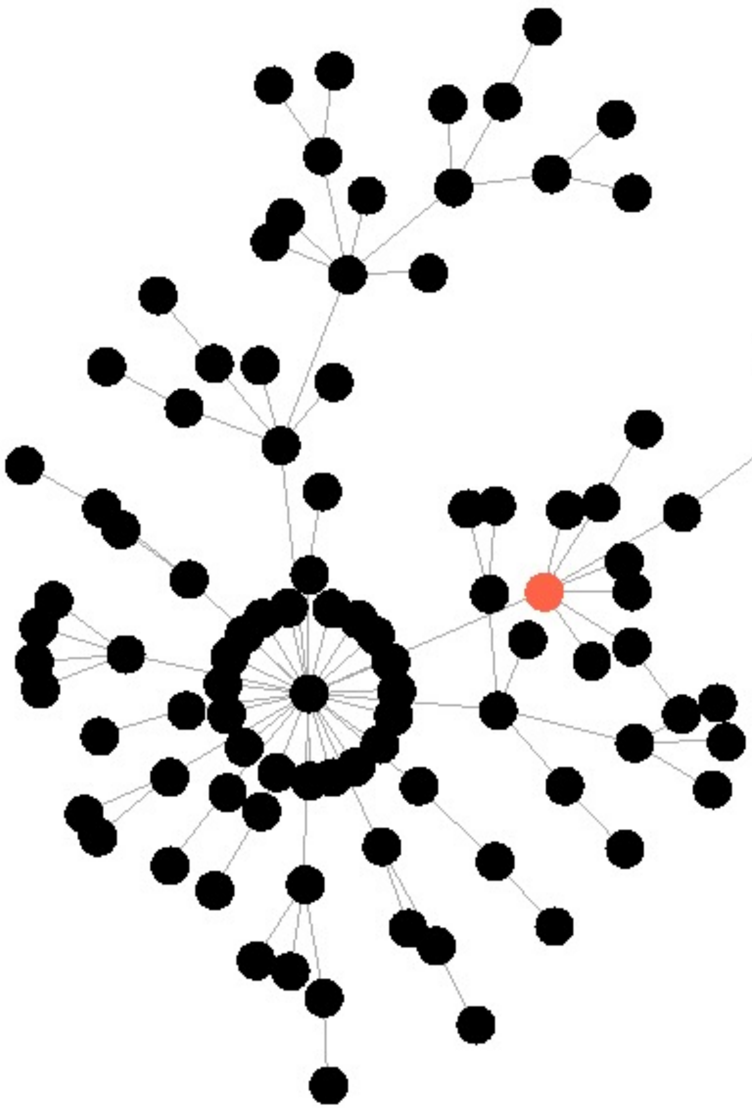


Example: SI model on networks



https://kurtjlewis.com/visualizing-epidemic-models-using-graphstream/

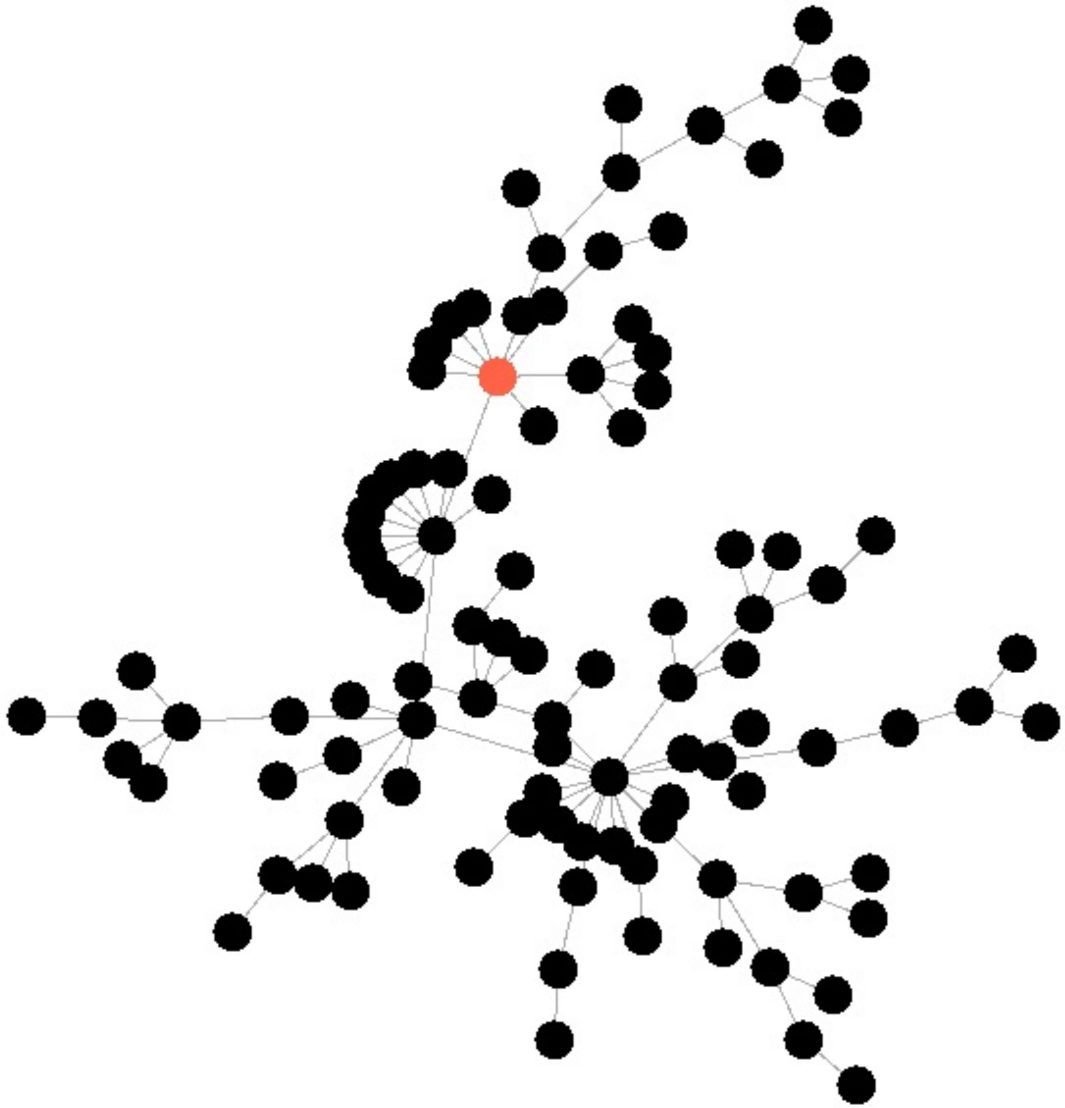
Example: SIS model on networks



Example of an endemic state

https://kurtjlewis.com/visualizing-epidemic-models-using-graphstream/

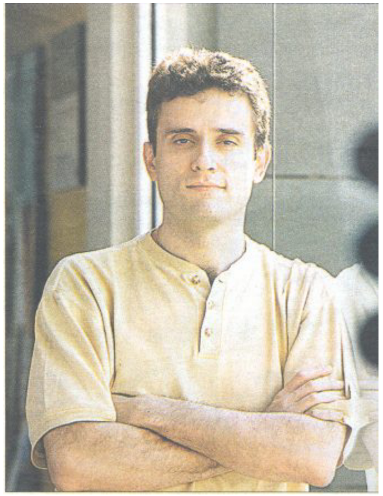
Example: SIR model on networks



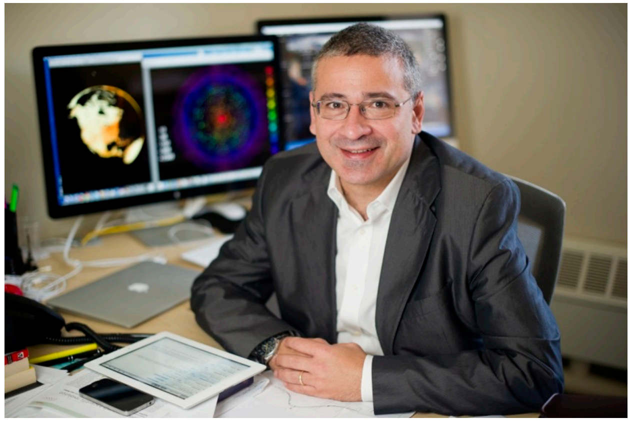
https://kurtjlewis.com/visualizing-epidemic-models-using-graphstream/

Minimal approach to network epidemics

(Pastor-Satorras & Vespignani, PRL 2001)



Romualdo Pastor-Satorras
Universitat Politècnica de Catalunya



Alessandro Vespignani
Northeastern University, Boston

Minimal approach to network epidemics

(Pastor-Satorras & Vespignani, PRL 2001)

- populations are infinite (or very large)
- network of contacts

1. Individuals are vertices of a network

1. Edges define who interacts with whom

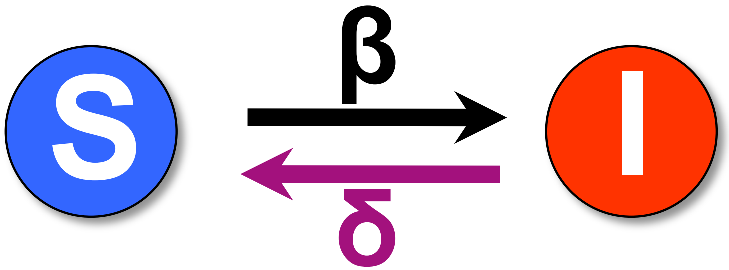
2. The graph is infinite and uncorrelated

3. The degree distribution P(k) such that $\int dk P(k) = 1$

and $< k^n >= \int dk P(k) k^n$

The general idea (e.g., SIS model)

β: contact infection rate
δ: recovery rate

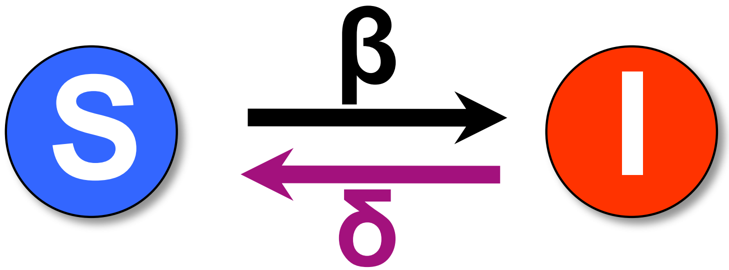


$$\dot{x} \equiv {dx}/{dt} = \underbrace{(1-x)\beta\langle k\rangle x}_{\text{infection}} \underbrace{- \delta x}_{\text{recovery}}$$

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

The general idea (e.g., SIS model)

β: contact infection rate
δ: recovery rate



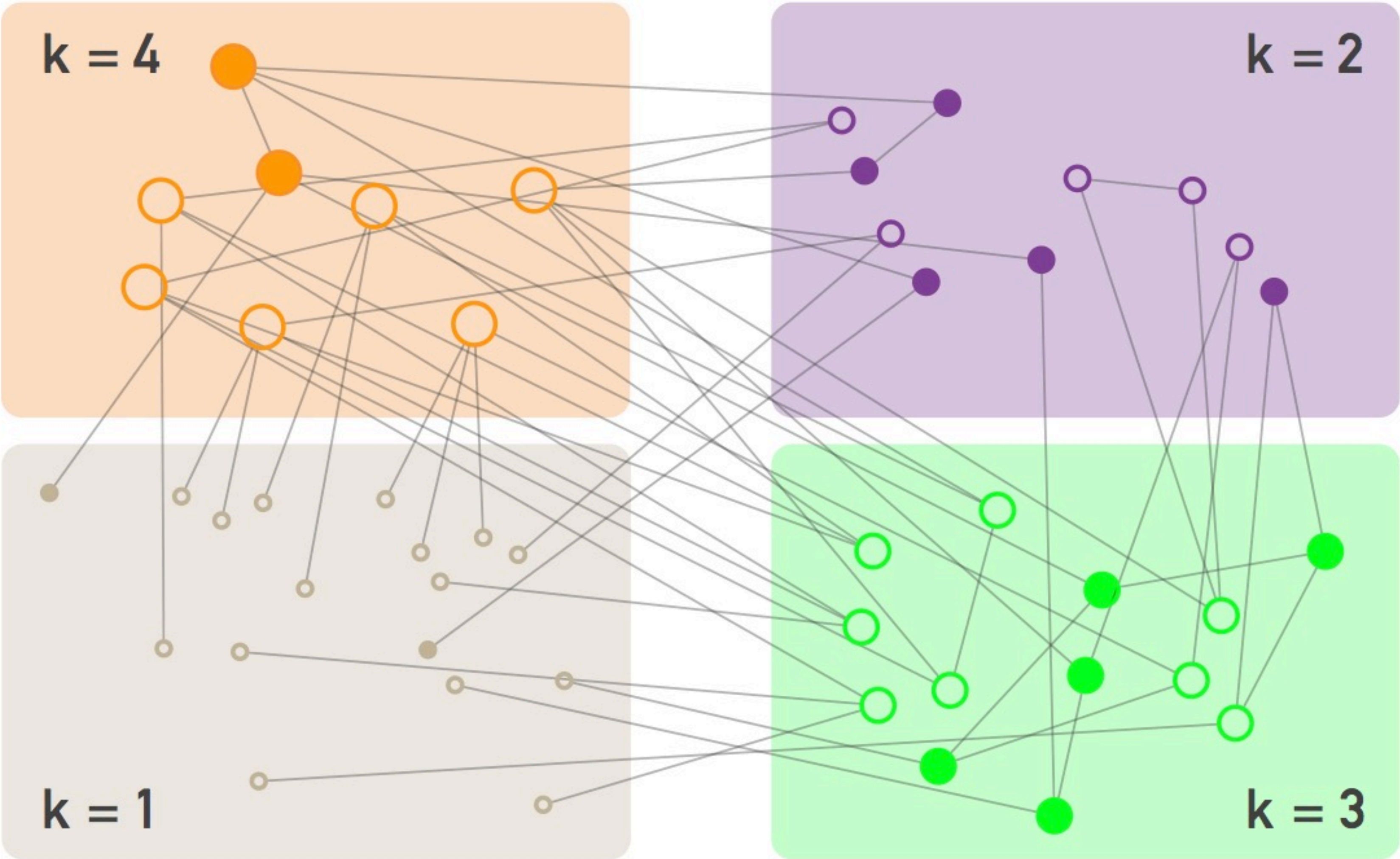
$$\dot{x} \equiv {dx}/{dt} = (1-x)\beta\langle k\rangle \boxed{x} - \delta x$$

Now, this will depend on the density of infected in a neighborhood!

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

The general idea

(Pastor-Satorras & Vespignani, PRL 2001)



The general idea (e.g., SIS model)

(Pastor-Satorras & Vespignani, PRL 2001)

- In networks, we shall have, for each degree

$$\frac{dx_k}{dt} = (1 - x_k)\beta k \boxed{\Theta_k(t)} - \delta x_k$$

Fraction of infected nodes

$$x = \sum_k P(k) x_k$$

Degree dist.

fraction of nodes with degree k that are infected

Density function, representing the fraction of infected neighbors of a susceptible node with degree k **(this was equal to x in the previous well-mixed & homogeneous scenarios)**

The general idea (e.g., SIS model)

(Pastor-Satorras & Vespignani, PRL 2001)

- In networks, we shall have, for each degree

$$\frac{dx_k}{dt} = (1 - x_k)\boxed{\beta k \Theta_k(t)} - \delta x_k$$

Fraction of infected nodes

$$x = \sum_k P(k) x_k$$

Degree dist.

fraction of nodes with degree k that are infected

This quantity redefines our previous "transmission rate", into something which is time, model and network dependent…

Yet Θ(t) is degree independent.

The general idea (e.g., SIS model)

(Pastor-Satorras & Vespignani, PRL 2001)

- For each degree k we have (for small x)

$$\frac{dx_k}{dt} \approx x_0 \beta k \frac{\langle k \rangle - 1}{\langle k \rangle} e^{t/\boxed{\tau}}$$

Fraction of infected nodes

$$x = \sum_k P(k) x_k$$

$$\cancel{\tau_{\text{well-mixed}} = \left(\beta \langle k \rangle - \delta\right)^{-1}}$$

$$\tau_{\text{networks}} = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - \delta \langle k \rangle}$$

Degree dist.

fraction of nodes with degree k that are infected

The general idea (e.g., SIS model)

(Pastor-Satorras & Vespignani, PRL 2001)

- For each degree k we have (for small x)

$$x_k \approx x_0 \left[1 + \frac{k(\langle k \rangle - 1)}{\langle k^2 \rangle - \langle k \rangle} \left(e^{t/\boxed{\tau}} - 1 \right) \right]$$

$$\tau = \frac{\langle k \rangle}{\boxed{\beta \langle k^2 \rangle - \delta \langle k \rangle}}$$

When $\beta \langle k^2 \rangle - \delta \langle k \rangle > 0$

the disease persists.

When $\beta \langle k^2 \rangle - \delta \langle k \rangle < 0$

the disease dies out.

The general idea (e.g., SIS model)

(Pastor-Satorras & Vespignani, PRL 2001)

- For each degree k we have (for small x)

$$x_k \approx x_0 \left[1 + \frac{k(\langle k \rangle - 1)}{\langle k^2 \rangle - \langle k \rangle} \left(e^{t/\tau} - 1 \right) \right]$$

Using a convenient definition of spreading rate $\lambda = \frac{\beta}{\delta}$

for ***random networks*** we get an endemic state if

$$\langle k^2 \rangle = \langle k \rangle (\langle k \rangle + 1)$$

$$\lambda = \frac{\beta}{\delta} > \frac{\langle k \rangle}{\langle k^2 \rangle} = \frac{1}{\langle k \rangle + 1}$$

The general idea (e.g., SIS model)

(Pastor-Satorras & Vespignani, PRL 2001)

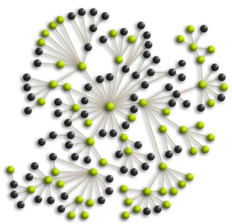
- For each degree k we have (for small x)

$$x_k \approx x_0 \left[1 + \frac{k\left(\langle k \rangle - 1\right)}{\langle k^2 \rangle - \langle k \rangle} \left(e^{t/\tau} - 1 \right) \right]$$

Using a convenient definition of spreading rate $\lambda = \dfrac{\beta}{\delta}$

for *scale-free networks* we get an endemic state if

$$\langle k^2 \rangle \xrightarrow{N \to \infty} = \infty$$



$$\lambda = \frac{\beta}{\delta} > \frac{\langle k \rangle}{\langle k^2 \rangle} \to 0$$

Vanishing epidemic threshold in SF nets (SIS)

In large scale-free networks,

$$\tau = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - \delta \langle k \rangle} \rightarrow 0$$

which means that

a virus can instantaneously reach most nodes

In large scale-free networks,

$$\lambda_C = \frac{\beta}{\delta} = \frac{\langle k \rangle}{\langle k^2 \rangle} \rightarrow 0$$

which means that,

even a virus with a small spreading rate can persist and spread.

Vanishing epidemic threshold in SF nets

(SIR)

Same as a percolation threshold (see robustness class)

In large scale-fre

$$\tau = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - (\delta + \beta)\langle k \rangle} \to 0$$

which means that

a virus can instantaneously reach most nodes

n large scale-free networks,

$$\lambda_C = \frac{\beta}{\delta} = \frac{1}{\dfrac{\langle k^2 \rangle}{\langle k \rangle} - 1} \to 0$$

which means that,

even a virus with a small spreading rate can persist and spread.

Vanishing epidemic threshold in SF nets (SIS)

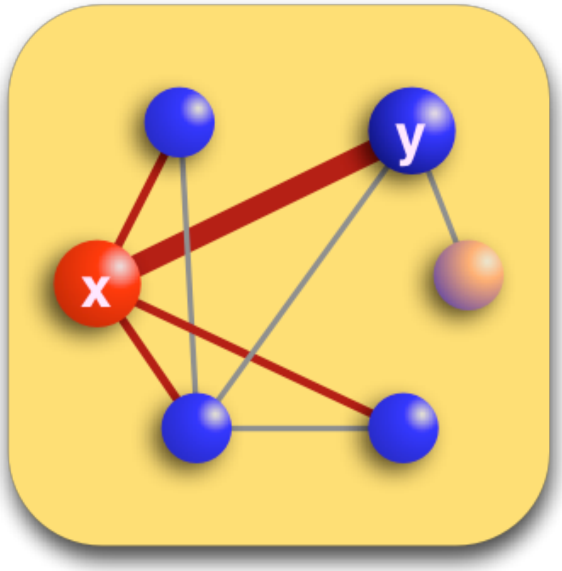
Simulations:

1. For each node, if S check if there's any I in the neighborhood. In that case, S will become I with a probability β. If the node is infected, it will recover with with a given probability δ.
2. Repeat 1. until you reach an equilibrium.
3. Repeat this simulation for many networks and different values of β.
4. For each class of network, compute the value of

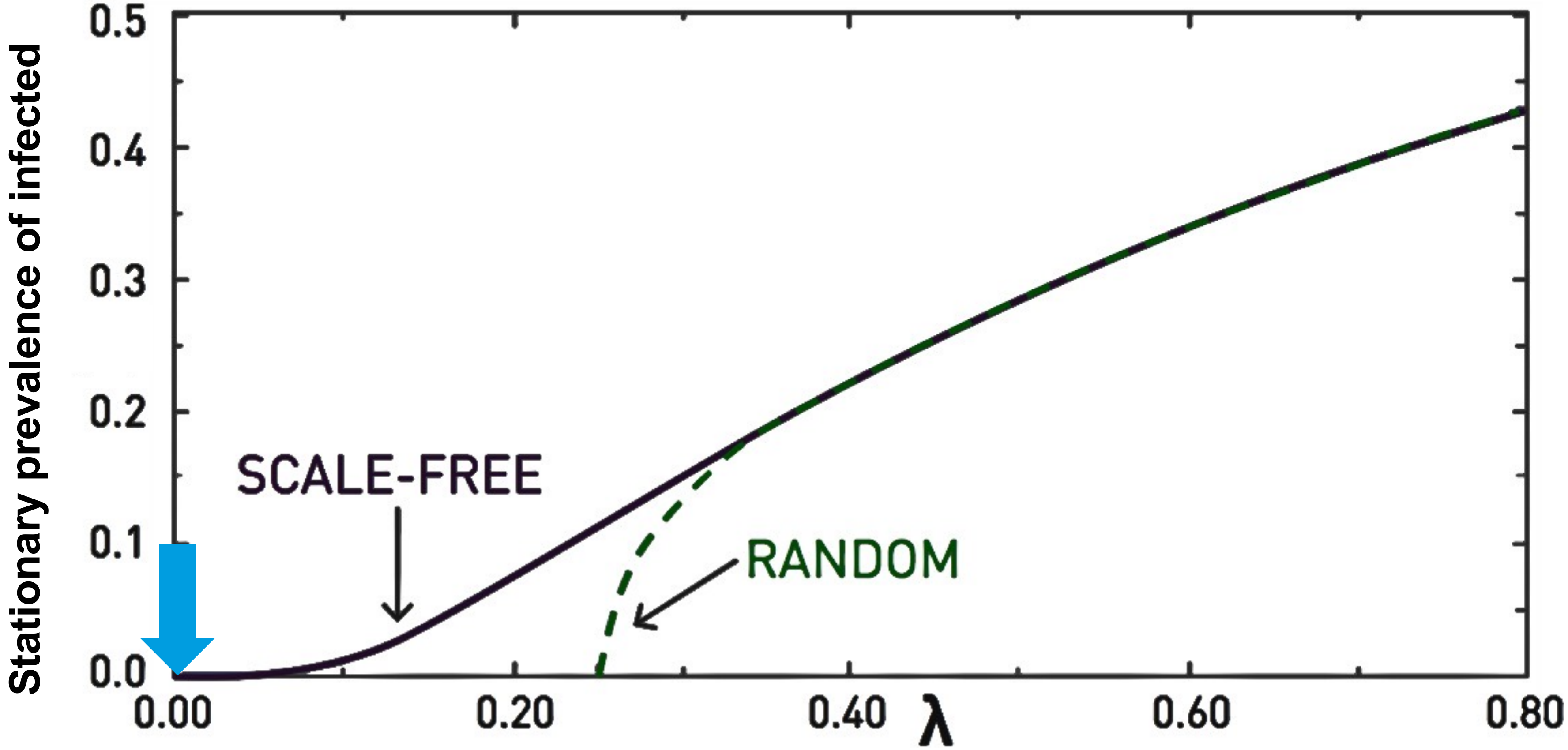
$$\lambda = \frac{\beta}{\delta}$$

below which the disease disappears.

Note: Please note that previously δ and β were rates. Now they are referred as probabilities.



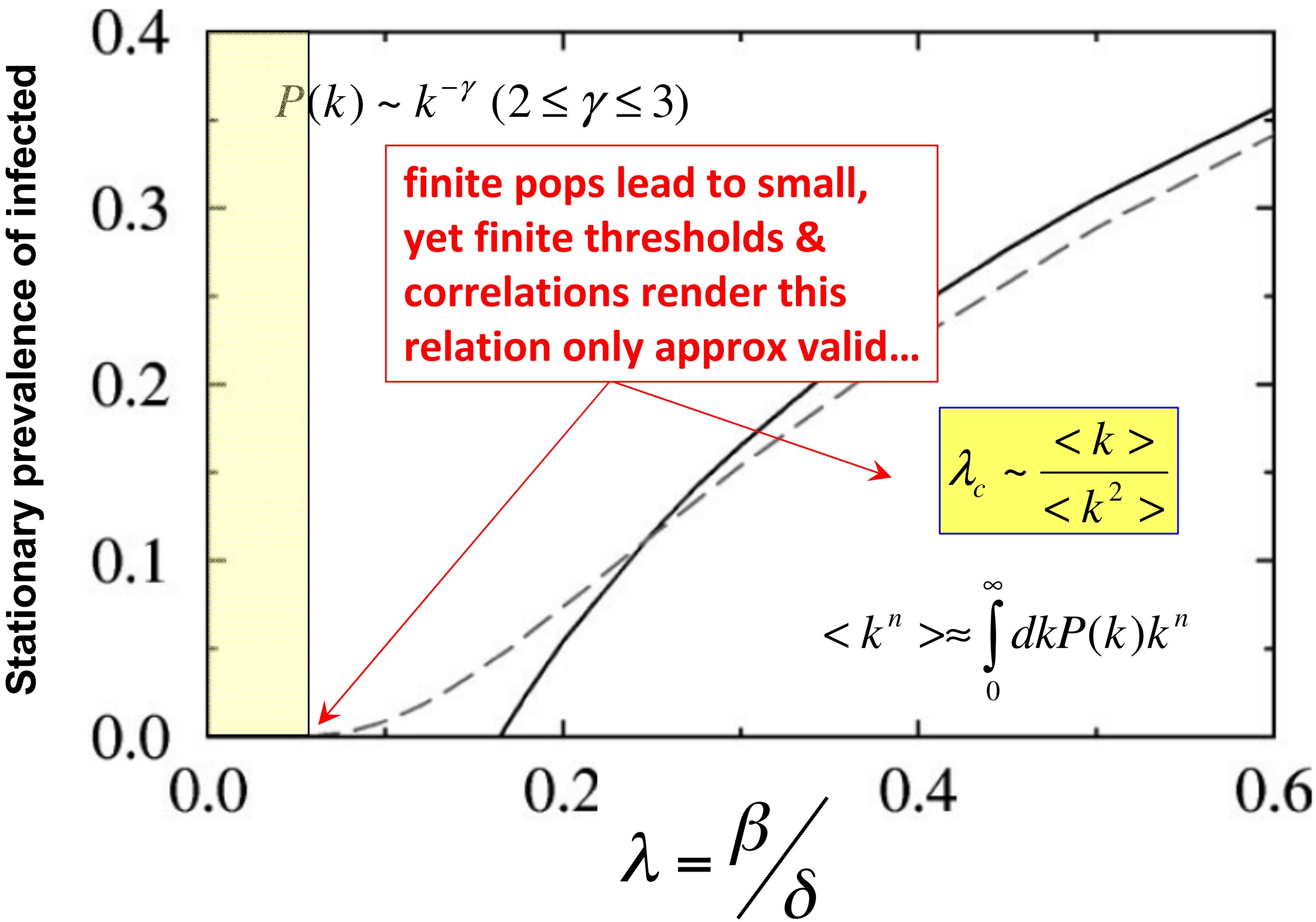
Vanishing epidemic threshold in SF nets (SIS)



$$\lambda = \frac{\beta}{\delta}$$

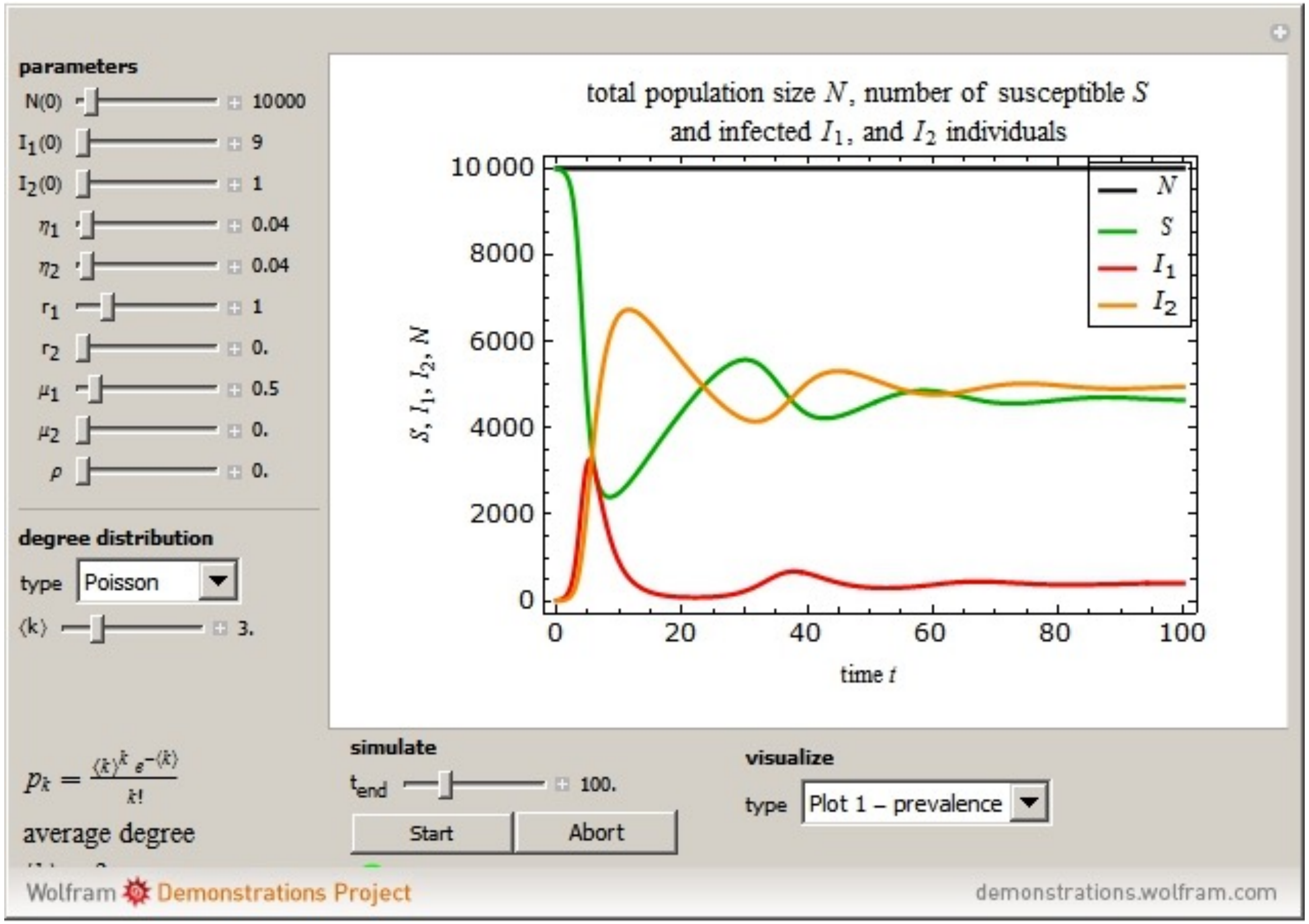
Disease spreading in finite networks

Challenge: Reproduce this result via computer simulations



Online simulation tools

Wolfram Demonstrations Project™



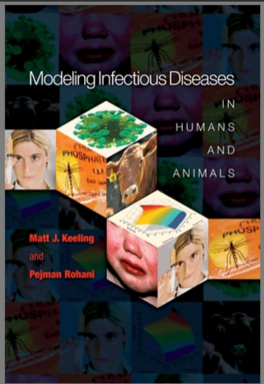
Example: "Epidemic Spread and Transmission Network Dynamics" from the Wolfram Demonstrations Project
http://demonstrations.wolfram.com/EpidemicSpreadAndTransmissionNetworkDynamics/

Online simulation tools

http://www.modelinginfectiousdiseases.org/

Modeling Infectious Diseases in Humans and Animals
Matt J. Keeling & Pejman Rohani

Simple SIR model (page 19)



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  - Program 2.5
  - Program 2.6
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- Contributions
- Erratum
- Reviews

Consider a "closed population" without demographics (no births, deaths, or migration). The scenario we have in mind is a large naive population into which a low level of infectious agent is introduced and where the resulting epidemic occurs sufficiently quickly that demographic processes are not influential. We also assume homogeneous mixing, whereby intricacies affecting the pattern of contacts are discarded, yielding βSI as the transmission term. Given the premise that underlying epidemiological rates are constant, we get the following *SIR* equations:

$$\frac{dS}{dt} = -\beta SI,$$

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

$$\frac{dR}{dt} = \gamma I.$$

Parameters

β	is the transmission rate and incorporates the encounter rate between susceptible and infectious individuals together with the probability of transmission.
γ	is called the removal or recovery rate, though often we are more interested in its reciprocal ($1/\gamma$) which determines the average infectious period.
$S(0)$	is the initial proportion of the population that are susceptible.
$I(0)$	is the initial proportion of the population that are infectious.

All rates are specified in **days**.

Requirements.
All parameters must be positive, and $S(0)+I(0) \leq 1$

Files
C++ Program, Python Program, Fortran Program, Parameters, MATLAB Code.

Back to our previous question:

How should we halt an epidemic?

Aiming at β

Transmission-Reducing Interventions

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

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 nodes

Vaccination

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

Vaccination strategies

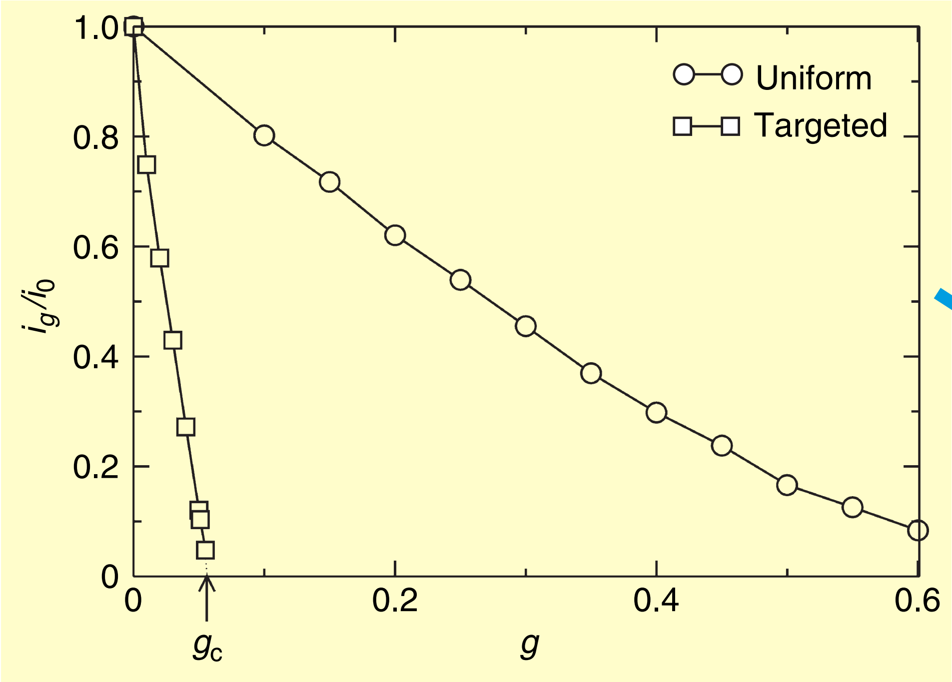
(Pastor-Satorras & Vespignani, 2002)

The immunization (ex: vaccination) of a fraction g ***randomly chosen*** from the population is equivalent to a simple rescaling of the effective spreading rate:

$$\lambda_C \to \lambda_C(1-g_C) \sim \frac{\langle k \rangle}{\langle k^2 \rangle}$$

Differently, we may try a ***targeted immunization***, aiming at high degree nodes. In this case, the immunization threshold:

$$g_C \sim e^{-2\delta / \lambda k_{\min}}$$



Agent-based simulations

What's the biggest problem with with this strategy?

Vaccination strategies

Cohen, Havlin and ben Avraham, 2003



Vaccination without global knowledge?

Group 0. Choose randomly a p fraction of nodes, like we do during random immunization. Call these nodes Group 0.

Group 1. Select randomly a partner for each node in Group 0. We call these partners, Group 1. For example, we ask each individual from Group 0 to nominate one of its acquaintance with whom he/she engaged in an activity that could have resulted in the transmission of the pathogen. In the case of HIV, ask them to name a sexual partner. Vaccinate group 1.

Repeat the same procedure for partners of group 1.

IS THIS STRATEGY EFFICIENT?

CAN YOU SIMULATE IT?

Vaccination as an optimization process

Holme, Petter, and Nelly Litvak. "Cost-efficient vaccination protocols for network epidemiology." PLoS Computational Biology 13, no. 9 (2017): e1005696.

To carry out a targeted vaccination campaign, one would first need to gather information about the network, then use this information to vaccinate.

Thus, there are 3 major costs involved :

- the cost of the disease itself,
- the cost of gathering the information about the network (c_{info})
- the cost of vaccinating (c_{vaac}).

Vaccination may then be framed as an optimization process with a goal function that combines these variables.

Fraction vaccinated

Number of inquiries needed

$$N\chi(f) = \Omega - \Omega'(f) - Nfc_{\text{vacc}} - n(f)c_{\text{info}}$$

Outbreak size without and with vaccination

Vaccination as an optimization process

Holme, Petter, and Nelly Litvak. "Cost-efficient vaccination protocols for network epidemiology." PLoS Computational Biology 13, no. 9 (2017): e1005696.

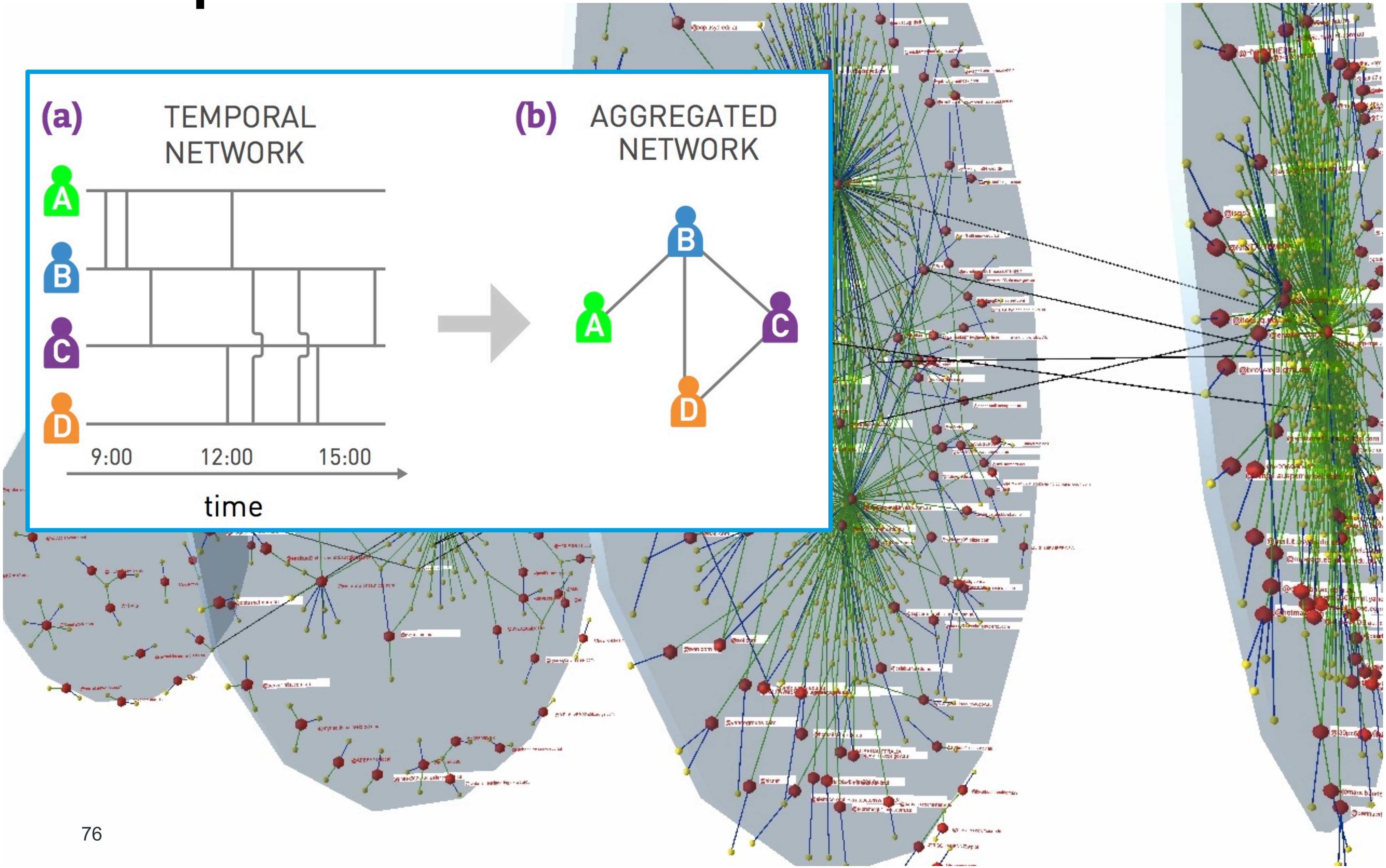
Strategies:

1. *Random.* Null model, low information cost.
2. *Acquaintance vaccination.* slighter larger information cost.
3. *Random-walk vaccination.* random walks can be an efficient method to find high-degree nodes.
4. *Two-step heuristic.* random walk on a sample.
5. *Degree centrality.* high information cost
6. *K-coreness.* high information cost.
7. *Collective influence.* similar to closeness centrality, high information cost.

Project/Challenge:
Optimize vaccination!

Beyond degree distributions:

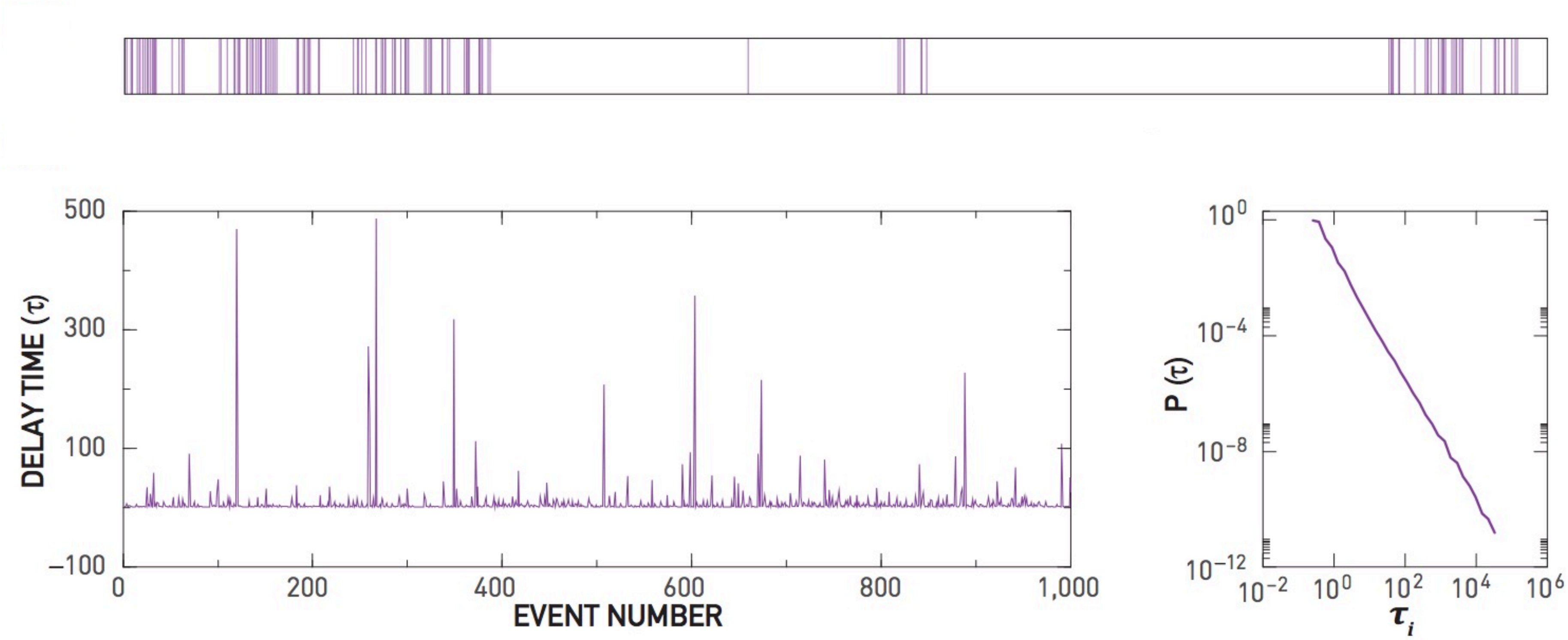
Temporal networks



Beyond degree distributions:
Bursty interactions

Project/Challenge: What's the impact of Bursty contact patterns in disease spreading?

Measurements indicate each connected pair of individuals will have periods of frequent interactions, when multiple contacts follow each other within a relatively short time frame, and long periods without any further contact.



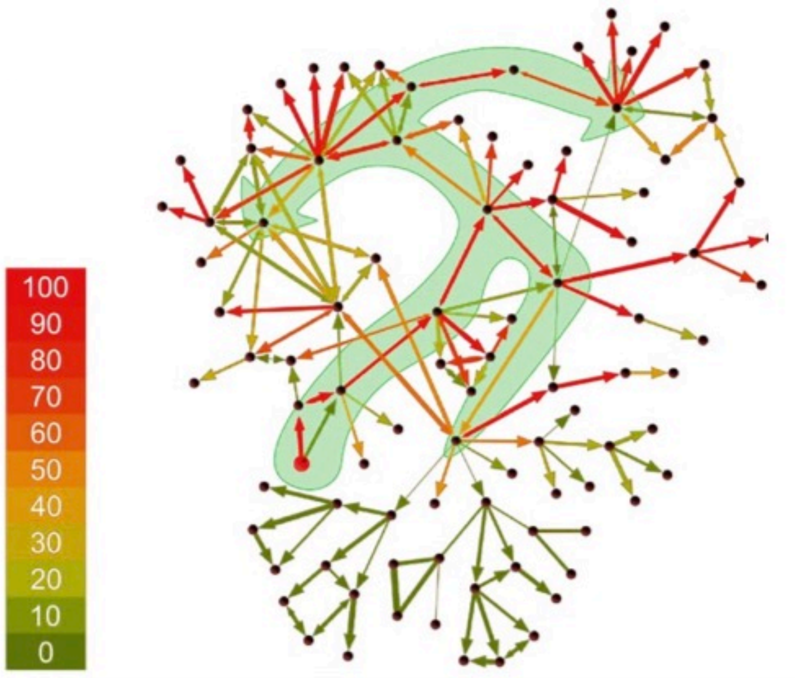
M. Salathé, et al., PNAS, 107:22020–22025, 2010.
Barabási et al., Nature, 435:207-11, 2005.

Beyond degree distributions:
Communities, assortativity, clustering, and weights

- Degree correlations. What should be the impact of degree correlations in disease spreading?

- Weights and community structure?

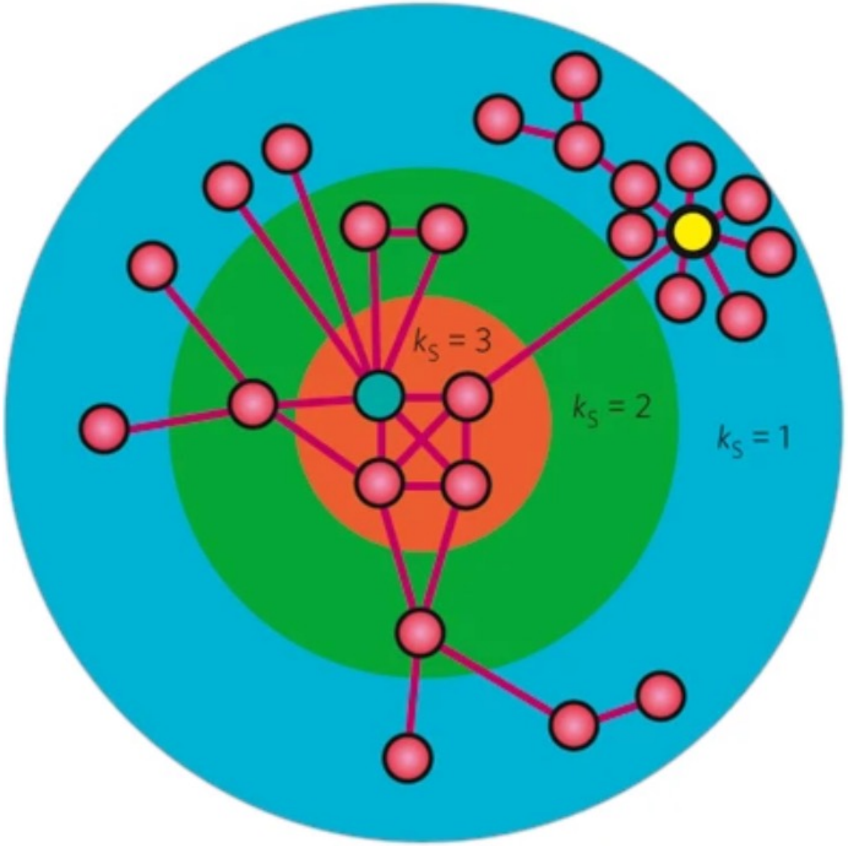
- What about clustering?



NOTE: Plenty of nice projects here…

Beyond degree distributions

Example: when the hubs are not the best spreaders



Identifying the most efficient ‘spreaders’ in a network is crucial.

Often the best spreaders do not correspond to the most highly connected or the most central people.

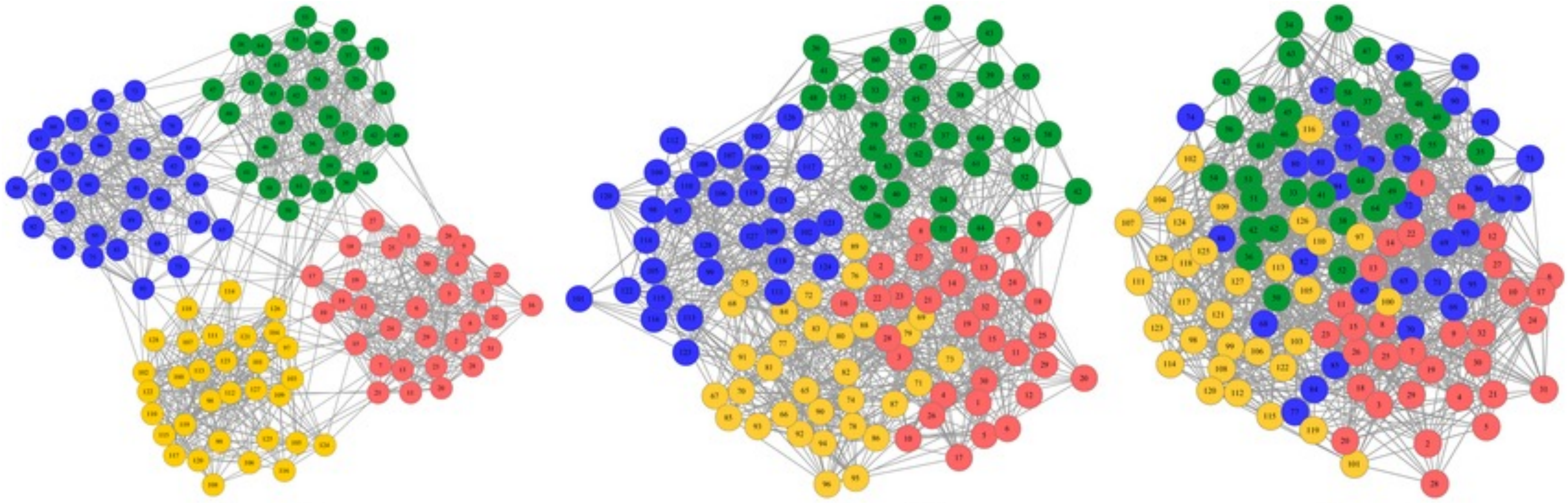
Instead, the most efficient spreaders are those located within the core of the network (identified by the k-shell decomposition).

Maksim, et al. "Identification of influential spreaders in complex networks." *Nature physics* 6.11 (2010): 888-893.

NOTE: Plenty of nice projects here…

Beyond degree distributions

Example: What about communities?



(a) $z_{in}=15$ (b) $z_{in}=11$ (c) $z_{in}=8$

Suggestion: Simulate a given epidemic dymamics with GN or LFT benchmarks of synthetic networks with different levels of community structure

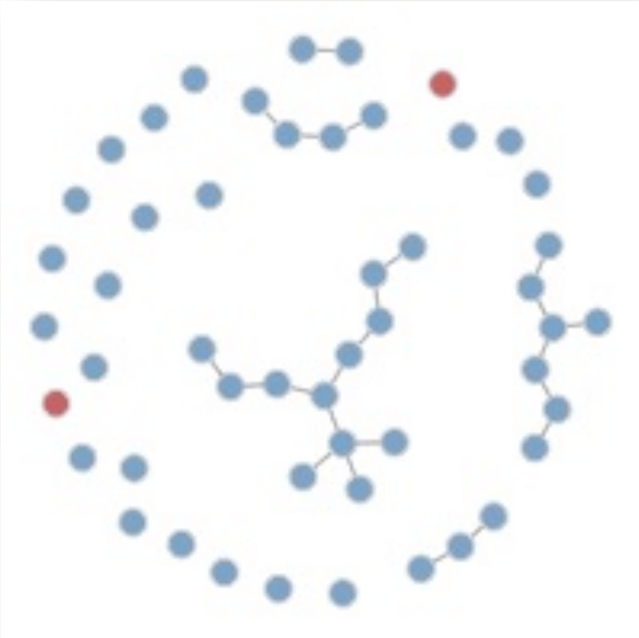
See also: Salathé, M., & Jones, J. H. (2010). Dynamics and control of diseases in networks with community structure. *PLoS computational biology*, *6*(4), e1000736.

NOTE: Plenty of nice projects here…

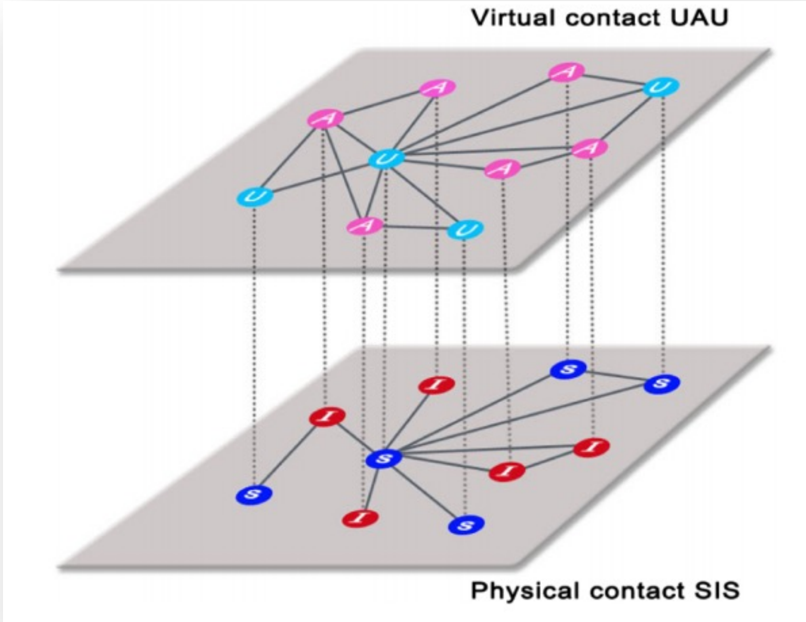
Beyond degree distributions

Behaviour matters! Awareness and epidemics

Temporal networks



Multi-layer networks



Ex: Van Segbroeck, S., Santos, F. C., & Pacheco, J. M. (2010). Adaptive contact networks change effective disease infectiousness and dynamics. PLoS Comput Biol, 6(8), e1000895.

Ex: Granell, C., Gómez, S., & Arenas, A. (2013). Dynamical interplay between awareness and epidemic spreading in multiplex networks. Physical review letters, 111(12), 128701.

NOTE: Plenty of nice projects here…

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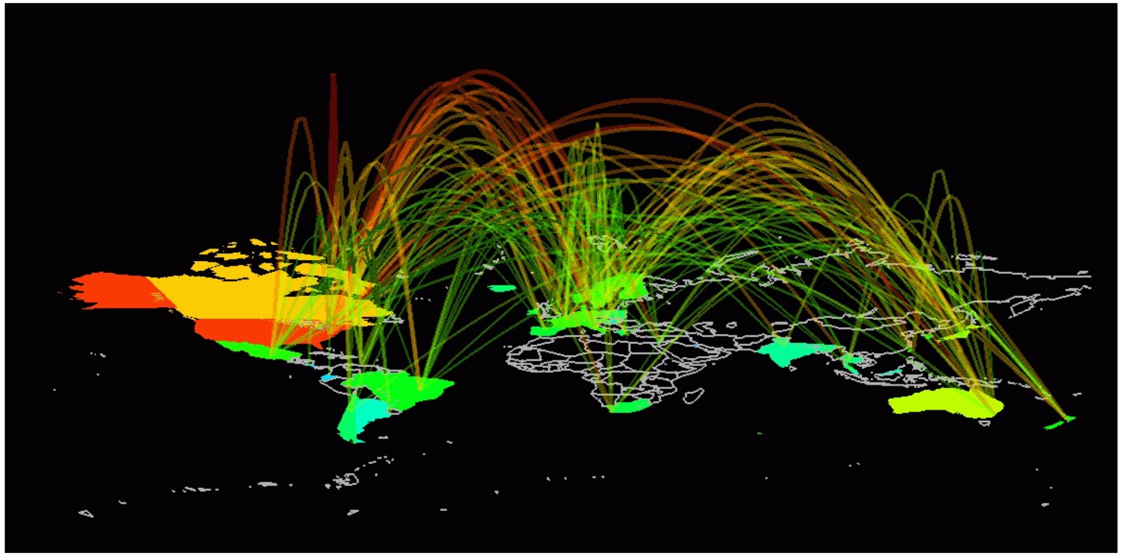
Computational epidemiology at a global scale

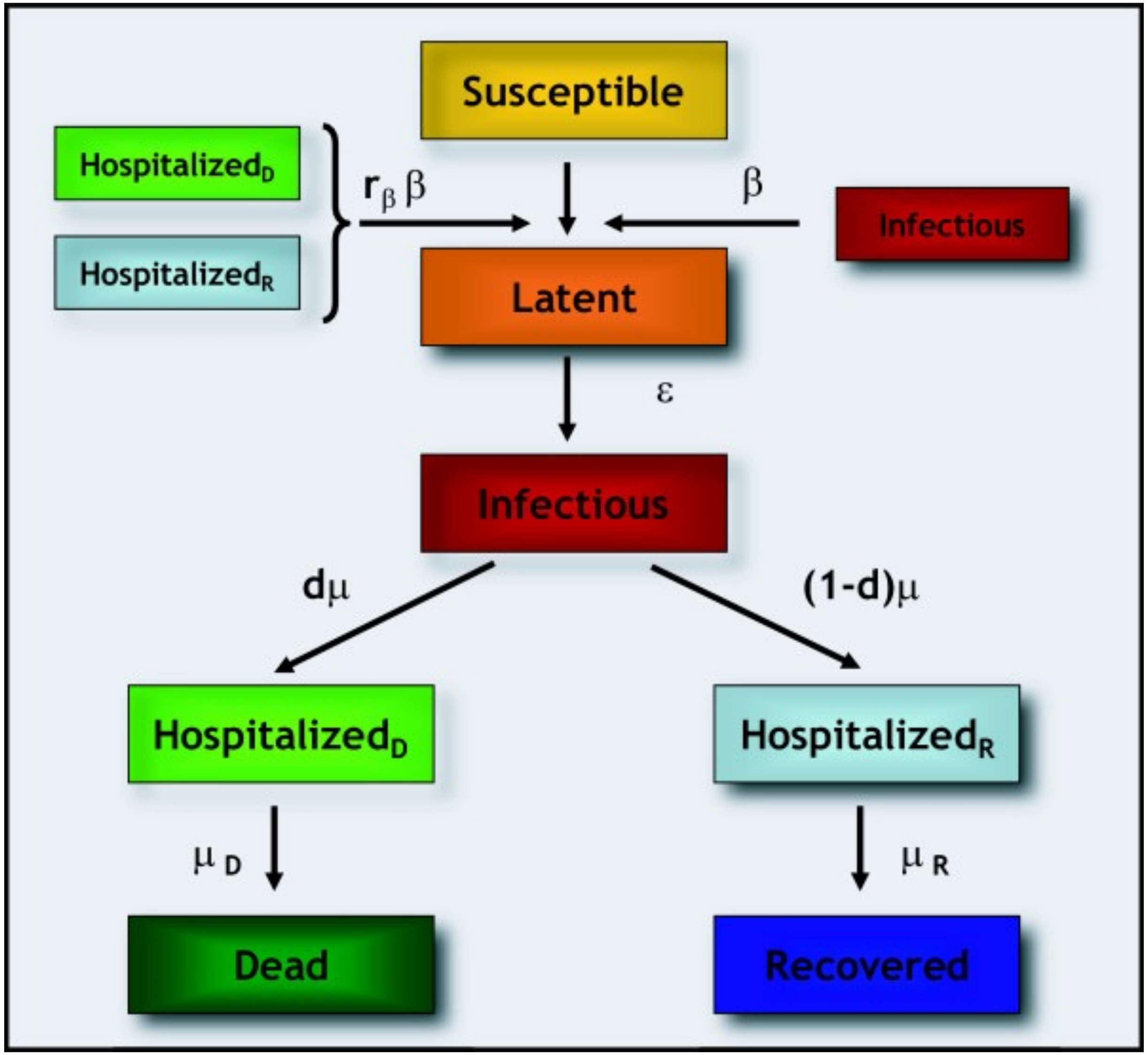


Network Science, 2021/2022

Overview

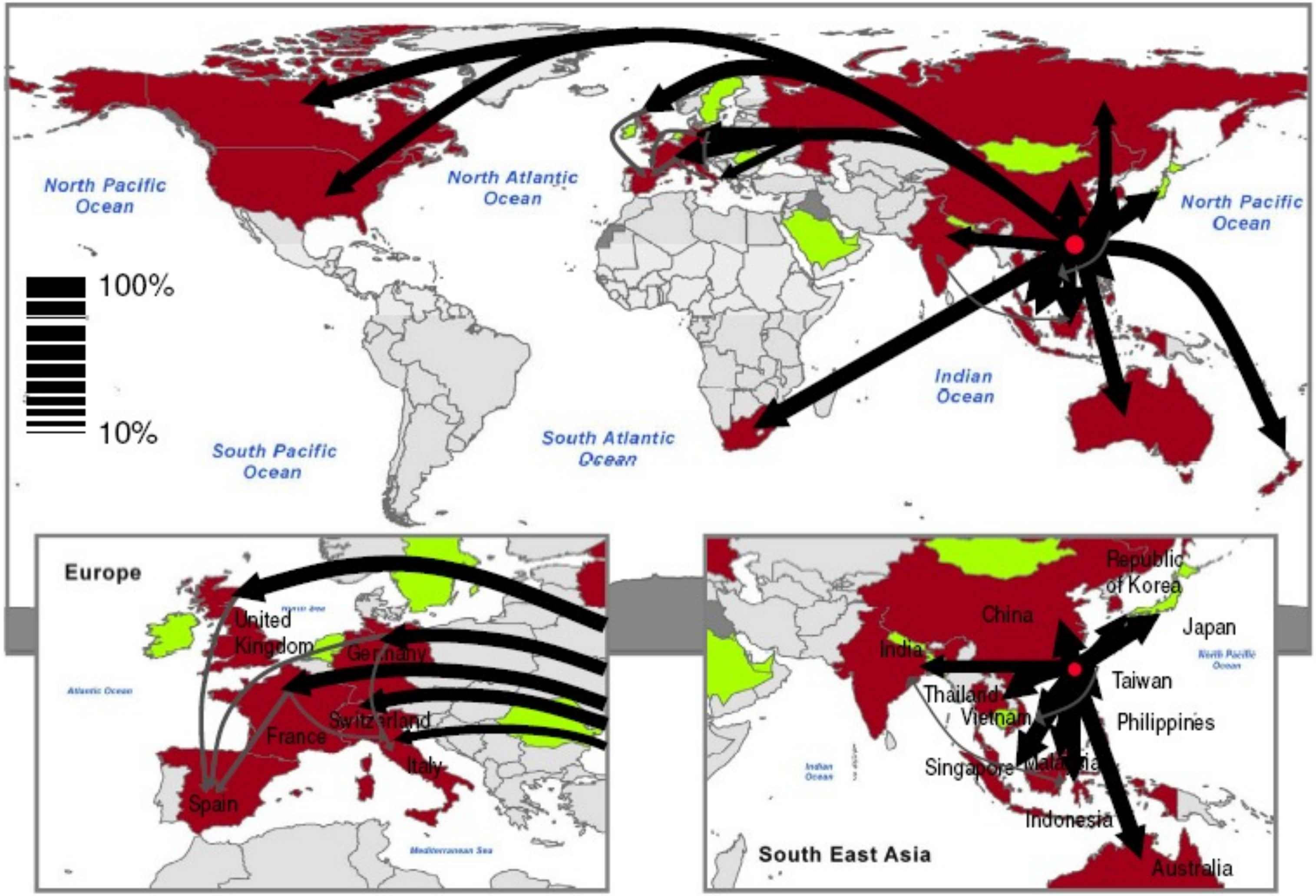
- Example: SARS (2002-03), H1N1 (2009), COVID-19, some future pandemia . . .
- can one make large scale predictions of pandemic scenarios in the same way as we can predict the weather ?
- so-far yes, on a global scale, combining well-mixed (in city) disease models (with lots of local parameters) with weighted networks describing global population mobility (air-transportation).
- Health governance: test the impact of travel restrictions, antiviral treatment, modifications on effective distances, etc.

Models used in SARS pandemic



Colizza et al. BMC Medicine 2007 5:34

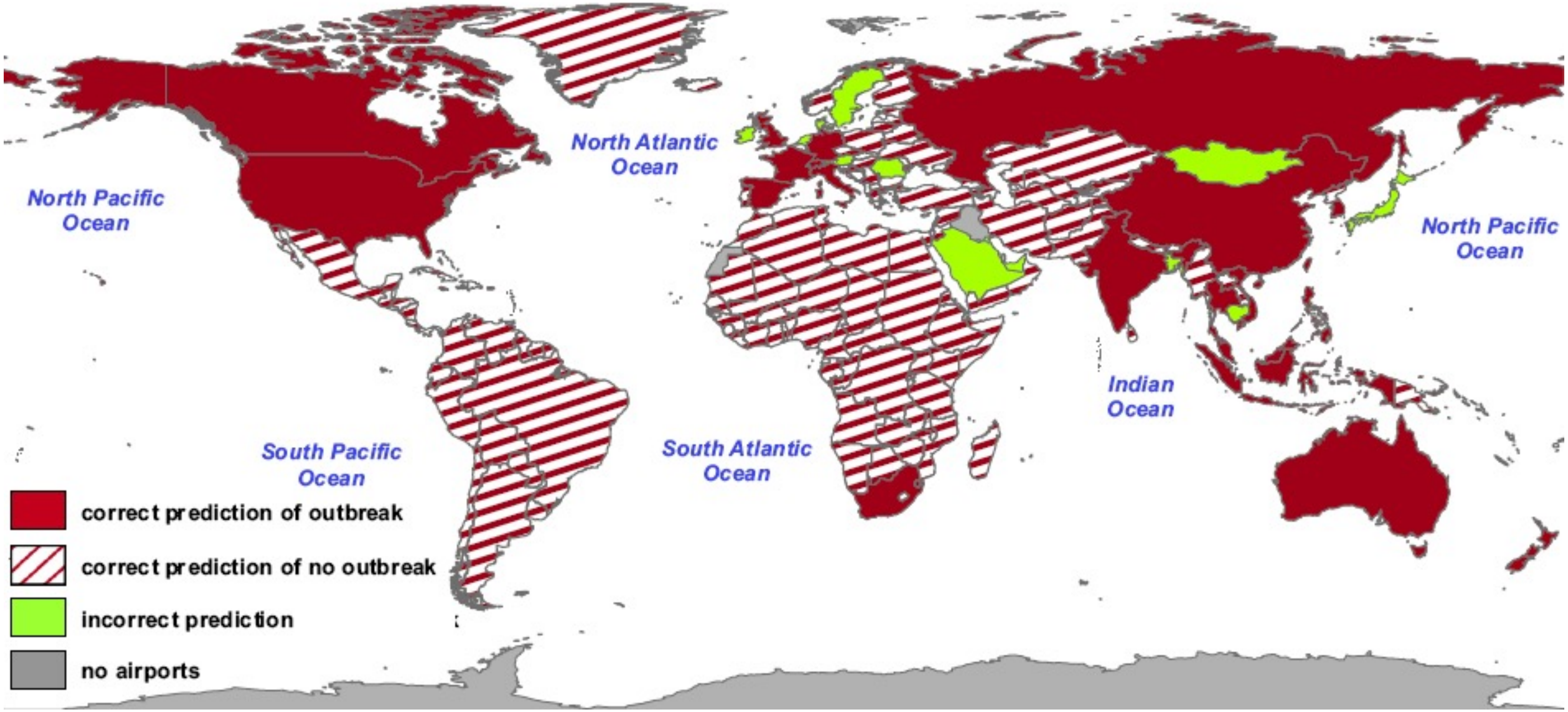
Computacional SARS



network: air-traffic between airports
weight : #-pass-year-on-route / #-tot-pass

Colizza et al. BMC Medicine 2007 5:34

Computacional SARS



network: air-traffic between airports
weight : #-pass-year-on-route / #-tot-pass

Colizza et al. BMC Medicine 2007 5:34

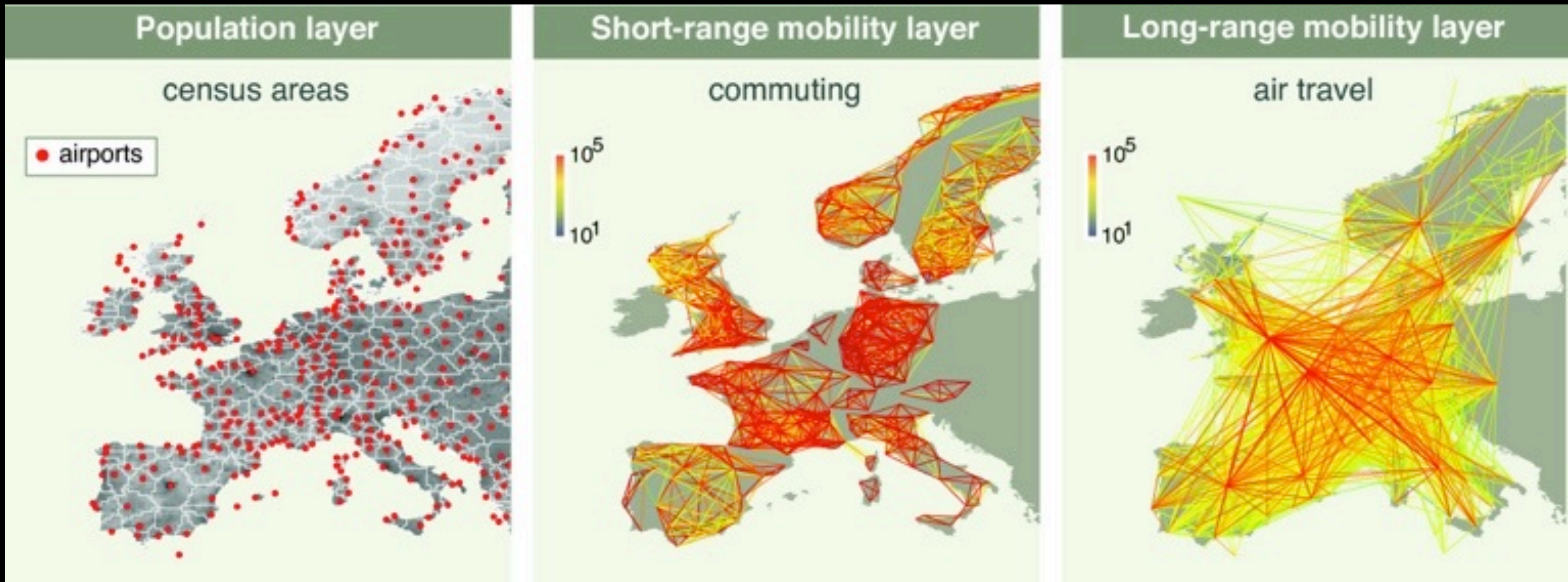
Example: GLEAM

GLEAM

GLOBAL EPIDEMIC AND MOBILITY MODEL

Van den Broeck et al, The GLEaMviz computational tool, a publicly available software to explore realistic epidemic spreading scenarios at the global scale, BMC Infectious Diseases 11:37 (2011)

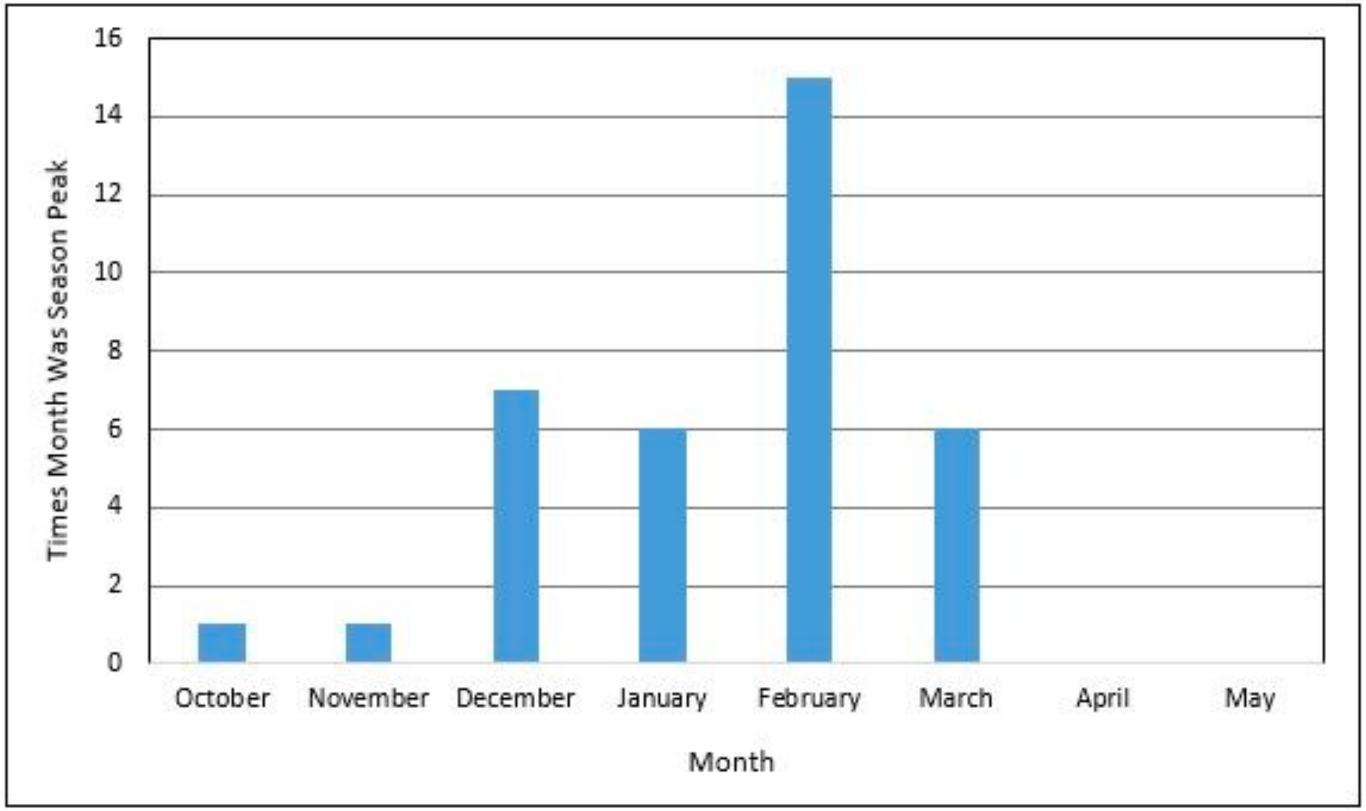
Example: GLEAM



Van den Broeck et al, The GLEaMviz computational tool, a publicly available software to explore realistic epidemic spreading scenarios at the global scale, BMC Infectious Diseases 11:37 (2011)

Peak Month of Flu Activity 1982-1983 through 2017-2018

Centers for Disease Control and Prevention
CDC 24/7: Saving Lives, Protecting People™

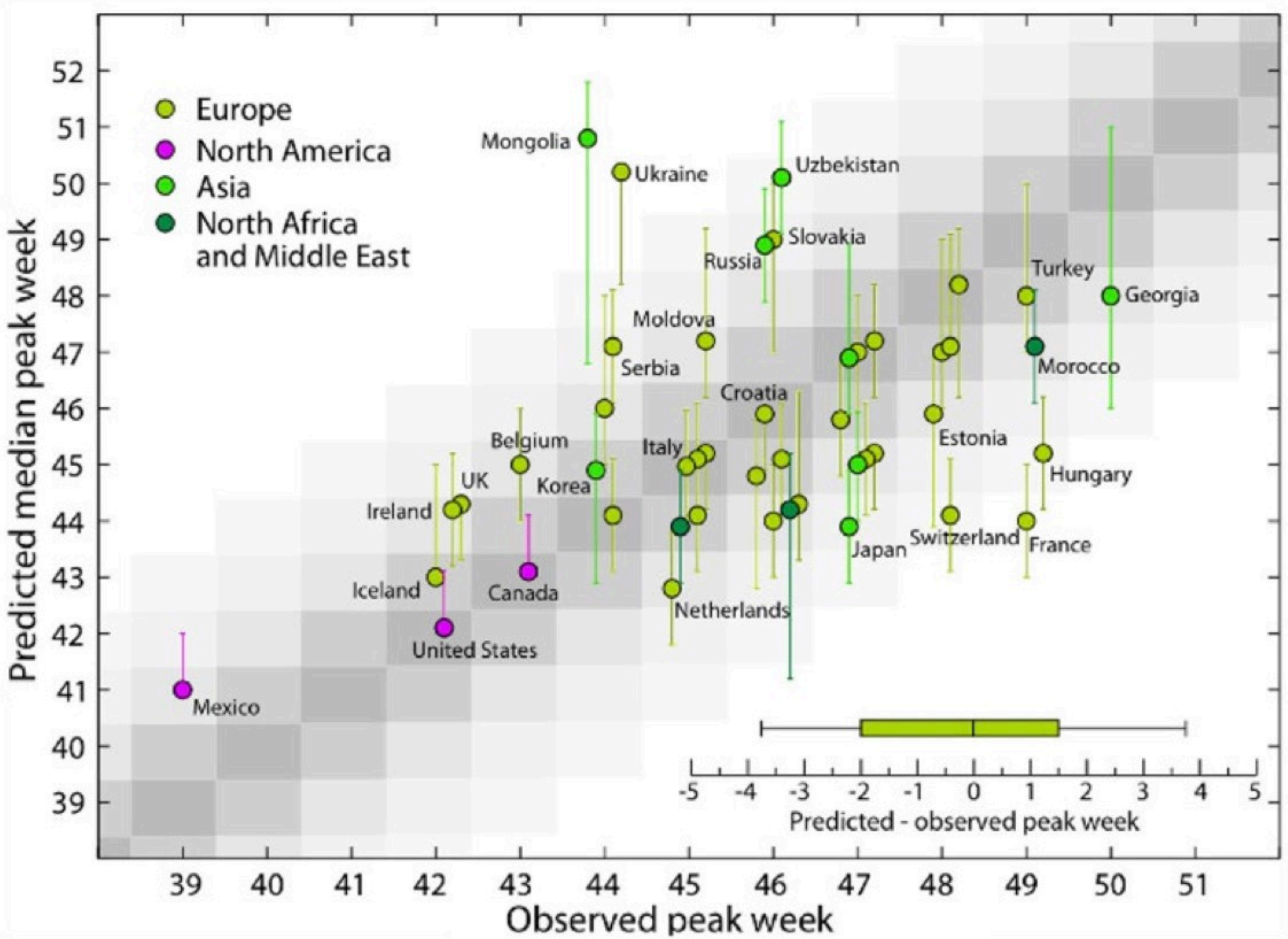


The exact timing and duration of flu seasons can vary, but influenza activity often begins to increase in October. Most of the time flu activity peaks between December and February, although activity can last as late as May.

Spread of H1N1 virus (2009)

GLEAM predicted that the H1N1-2009 epidemic will peak out in November, rather than in January or February, the typical peak time of influenza-like viruses.

Spread of H1N1 virus (2009)



P. Bajardi, et al. Human Mobility Networks, Travel Restrictions, and the Global Spread of 2009 H1N1 Pandemic. PLoS ONE, 6:e16591, 2011.

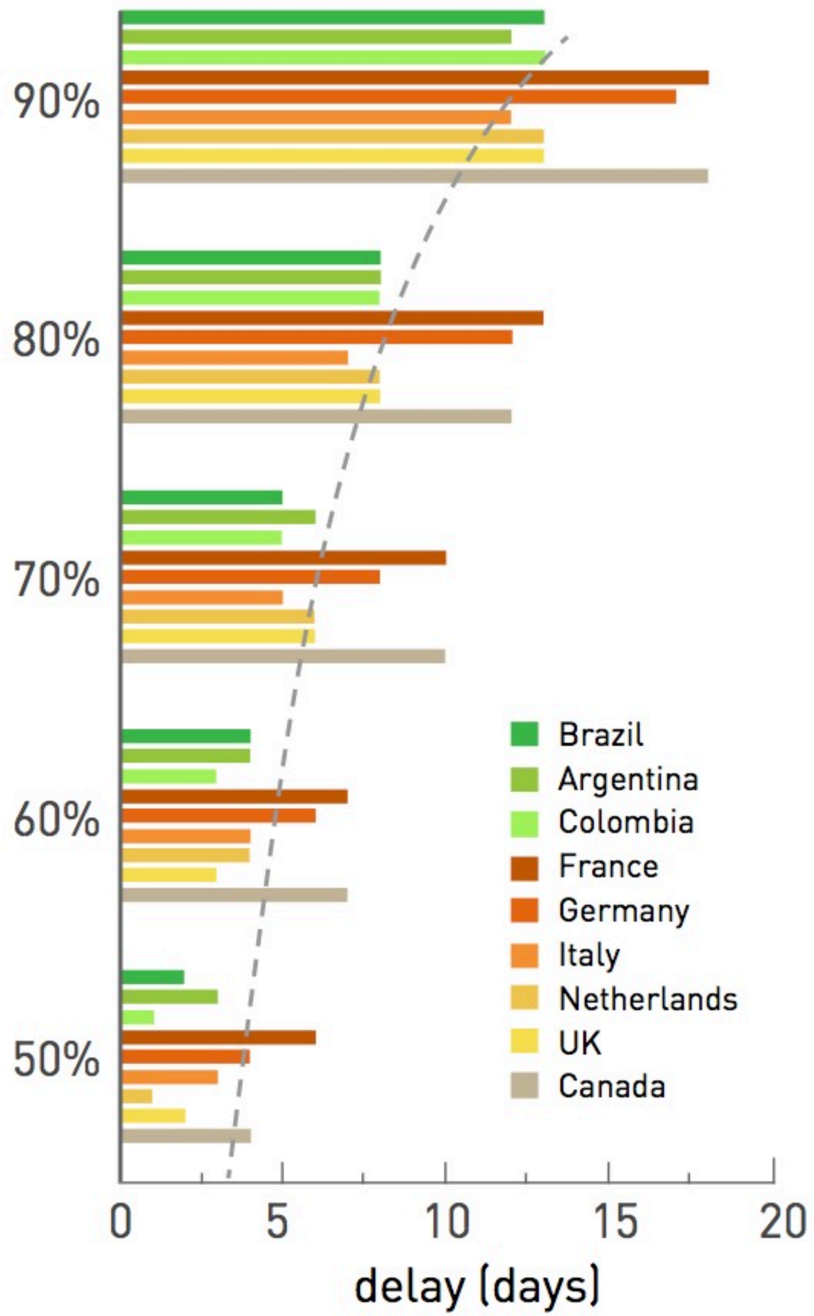
Travel restrictions and the spread of H1N1 (2009)

Similar studies predicted marginal impact in case of significant travel restrictions.

T. D. Hollingsworth et al. Nature Medicine, 12:497-499, 2006.

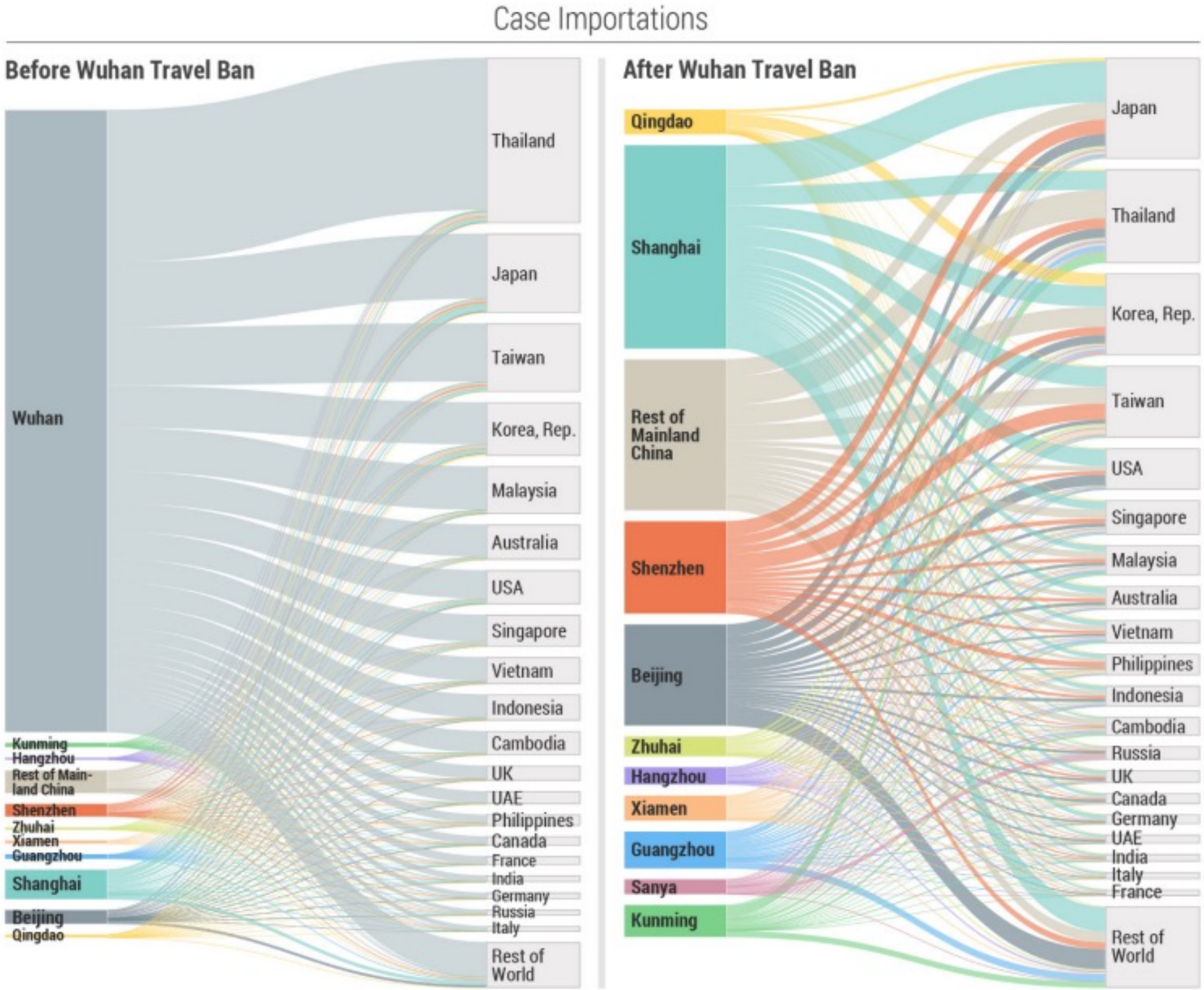
Yet, widespread antiviral treatment may have delayed in 3-4 weeks.

A.C. Singer, et al. Environ Health Perspect., 116:1563-1567, 2008.



Travel restrictions and the spread of Covid-19 (2020-21)

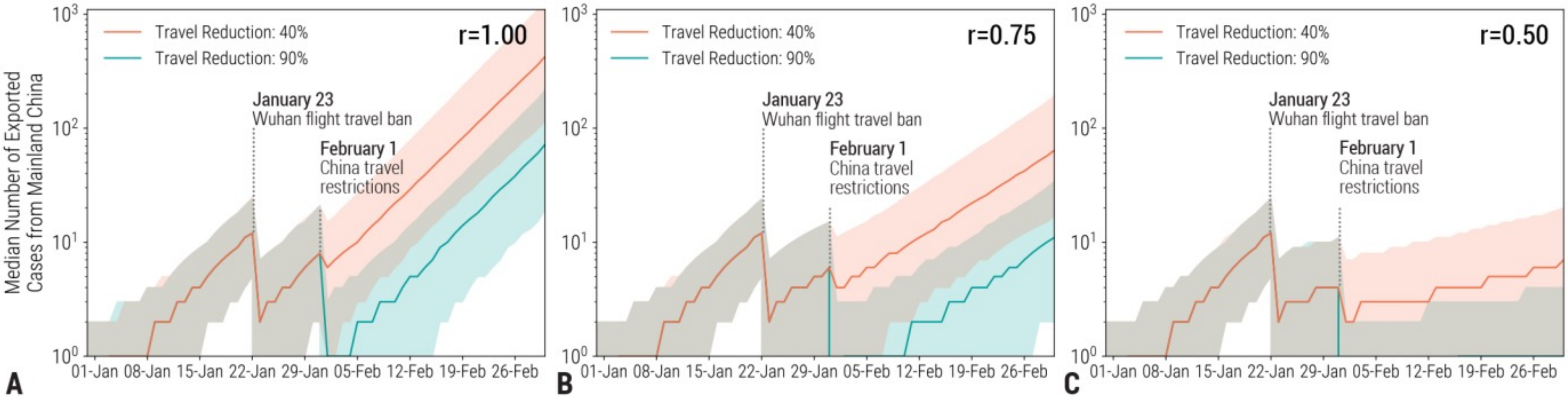
The travel quarantine of Wuhan (Jan, 2020) delayed the overall epidemic progression by only 3 to 5 days in Mainland China; yet at the international scale, importation were reduced by nearly 80% until mid February.



Chinazzi, M., Davis, J. T., Ajelli, M., Gioannini, C., Litvinova, M., Merler, S., ... & Vespignani, A. (2020). The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak. *Science*, *368*(6489), 395-400.

Travel restrictions and the spread of Covid-19 (2020-21)

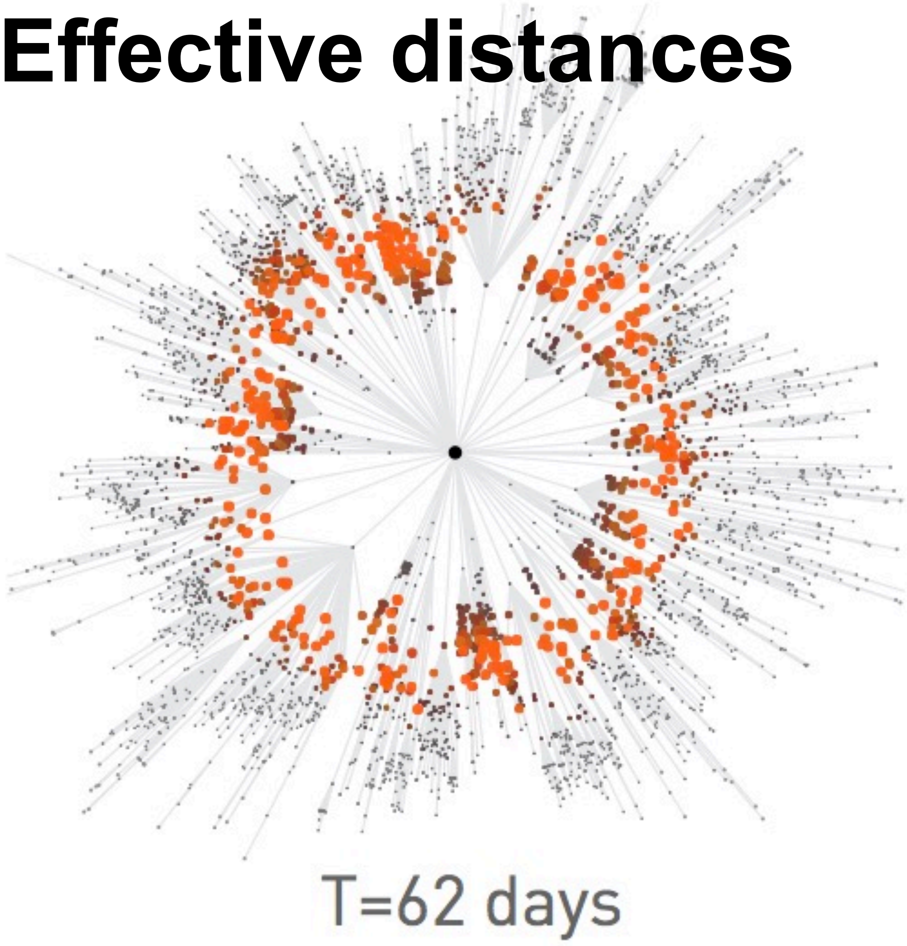
r = transmissibility reduction factor

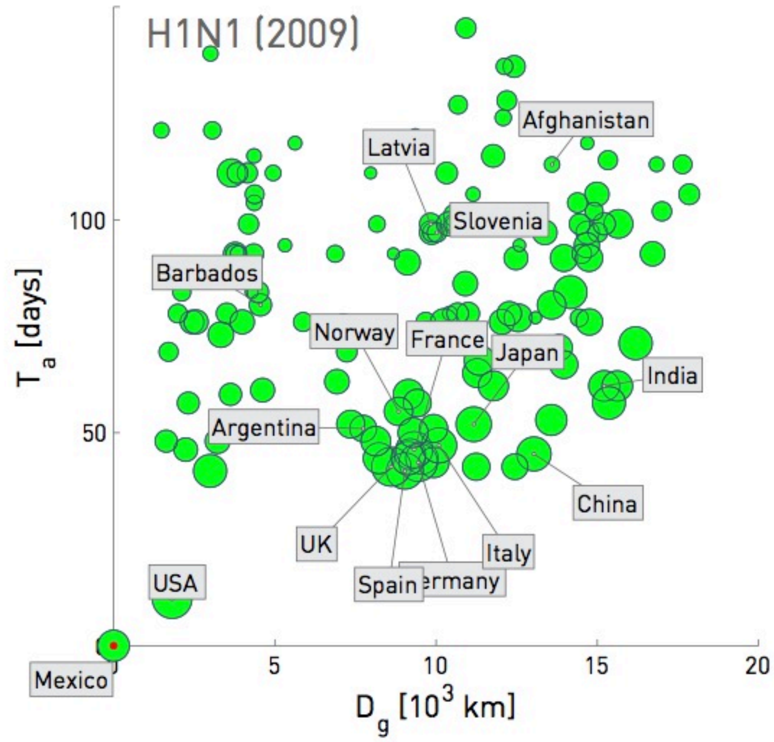


Results also indicate that sustained 90% travel restrictions to and from Mainland China only modestly affect the epidemic trajectory unless combined with a 50% or higher reduction of transmission in the community.

Chinazzi, M., Davis, J. T., Ajelli, M., Gioannini, C., Litvinova, M., Merler, S., ... & Vespignani, A. (2020). The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak. *Science*, *368*(6489), 395-400.

Effective distances



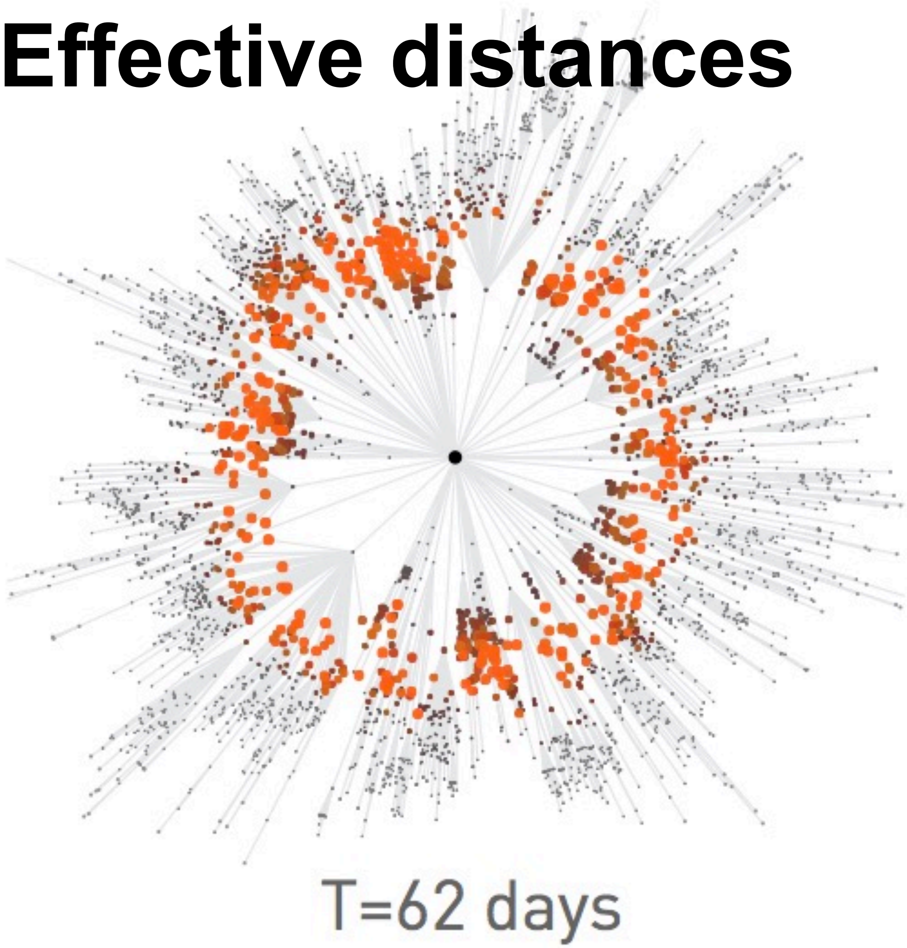


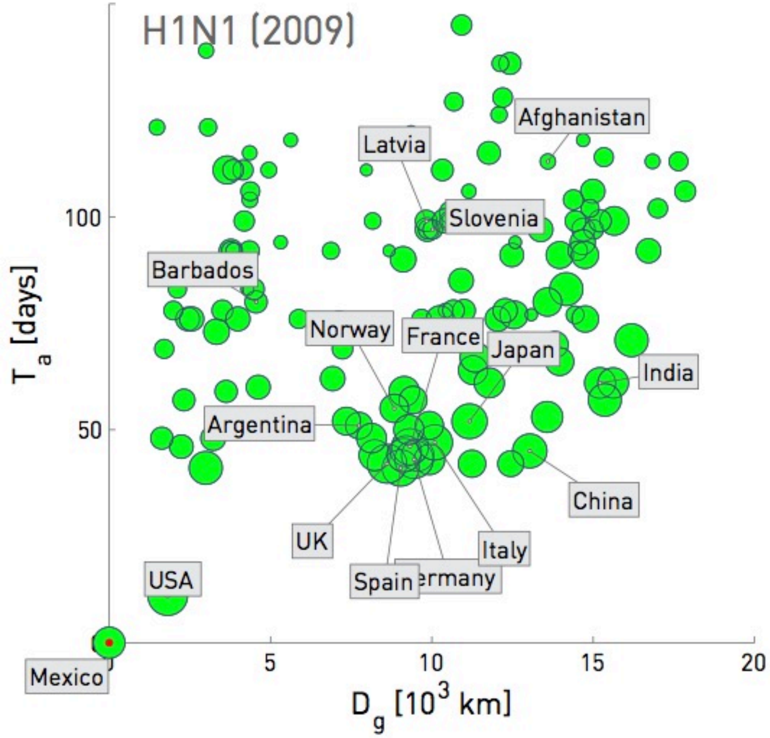
Is there a better space to view the spread of an epidemic than the physical space?

What if we replace the geographic distance with an ***effective distance*** derived from the mobility network?

D. Brockmann and D. Helbing. The Hidden Geometry of Complex, Network-Driven Contagion Phenomena. Science, 342:1337-1342, 2014.

Effective distances





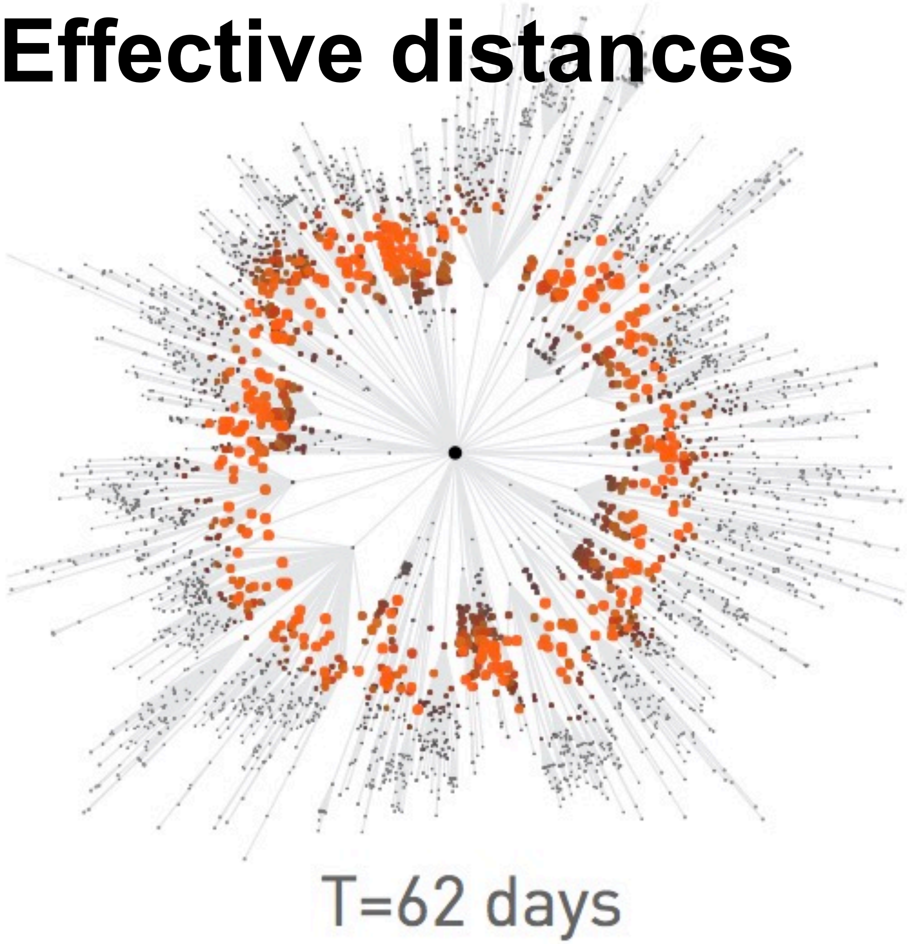
$$d_{ij} = (1 - \ln p_{ij})$$

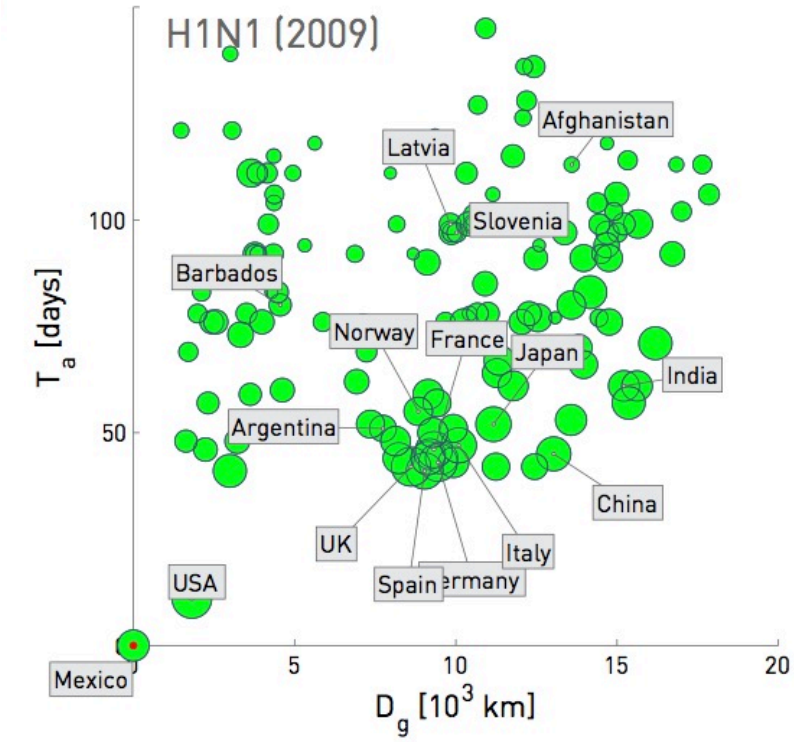
Effective distance between cities i and j.

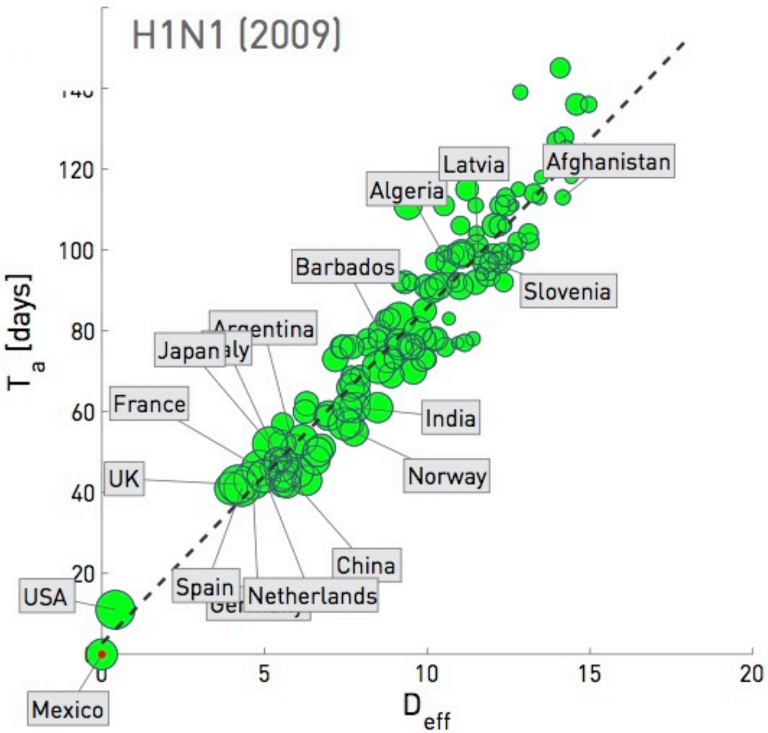
Normalized flux of people traveling from i to j

D. Brockmann and D. Helbing. The Hidden Geometry of Complex, Network-Driven Contagion Phenomena. Science, 342:1337-1342, 2014.

Effective distances







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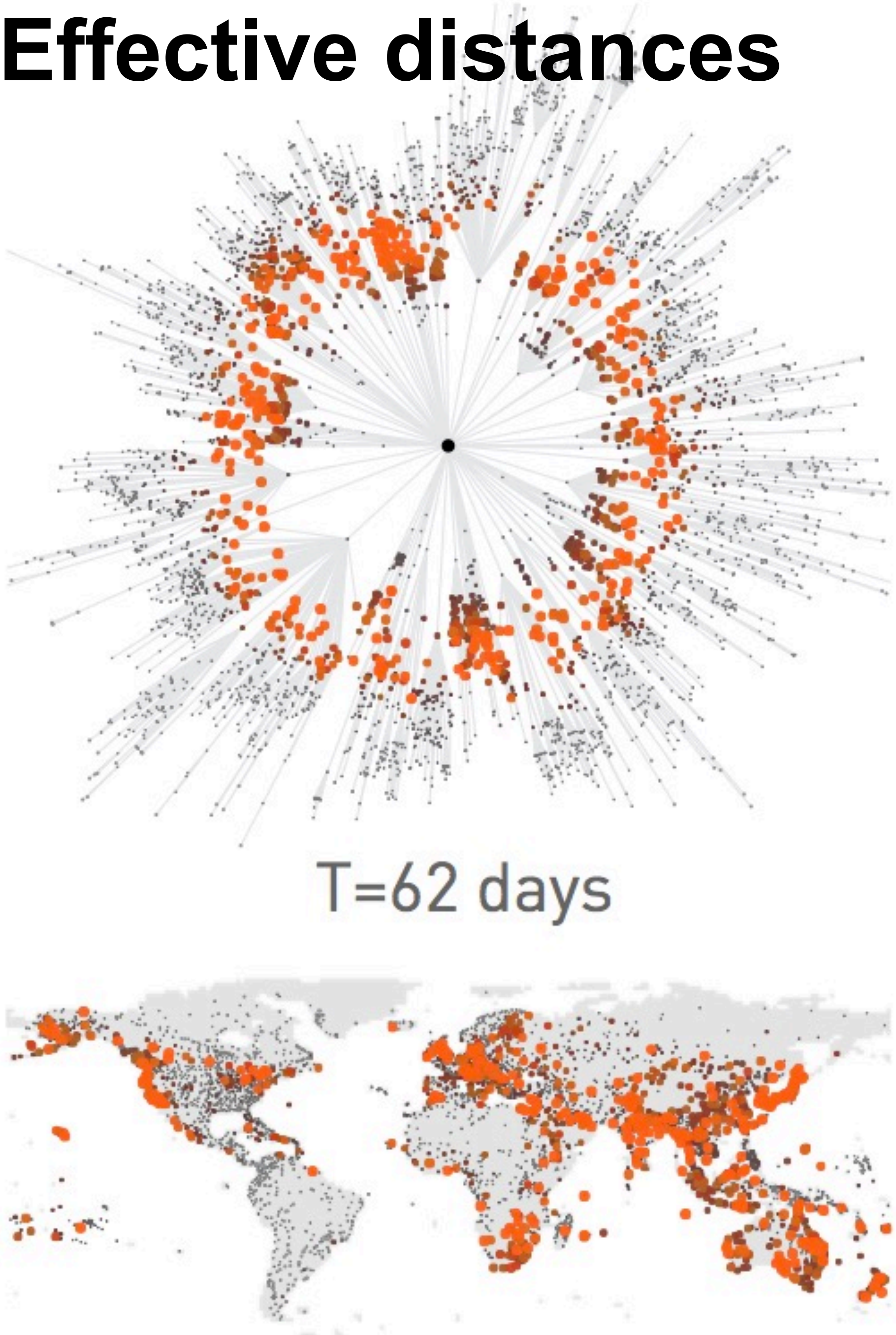
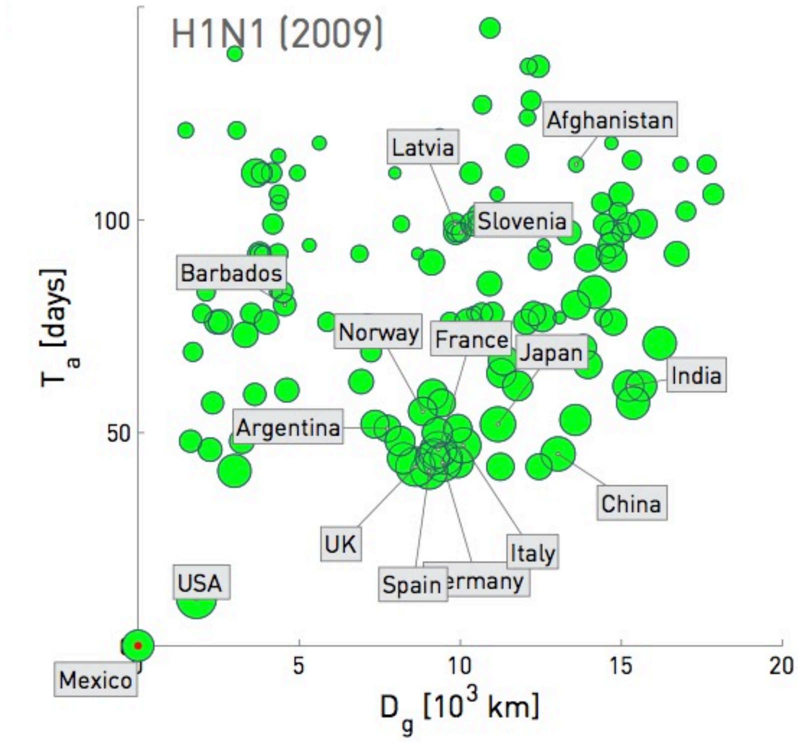
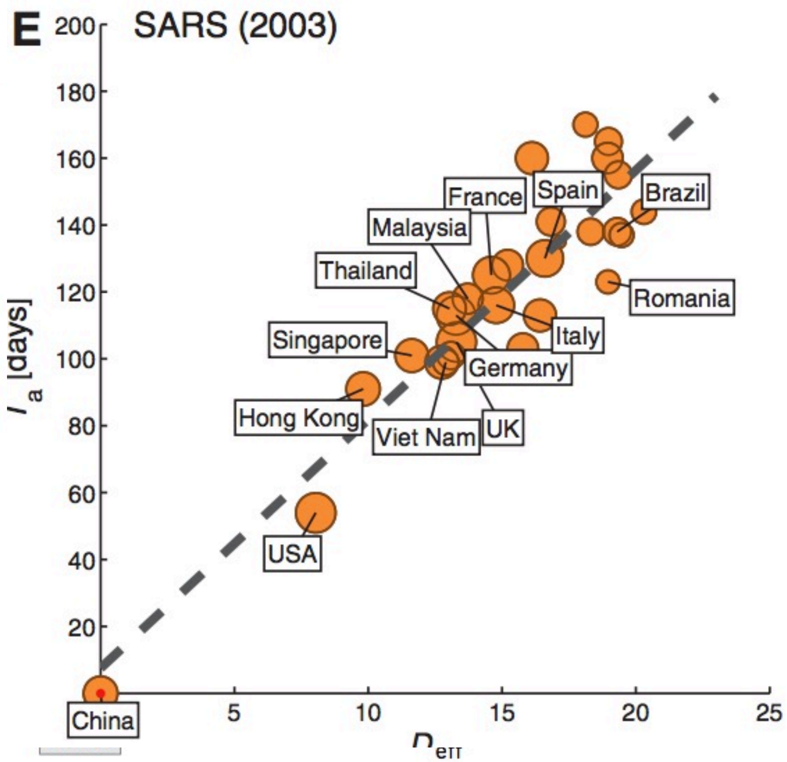
Effective distances



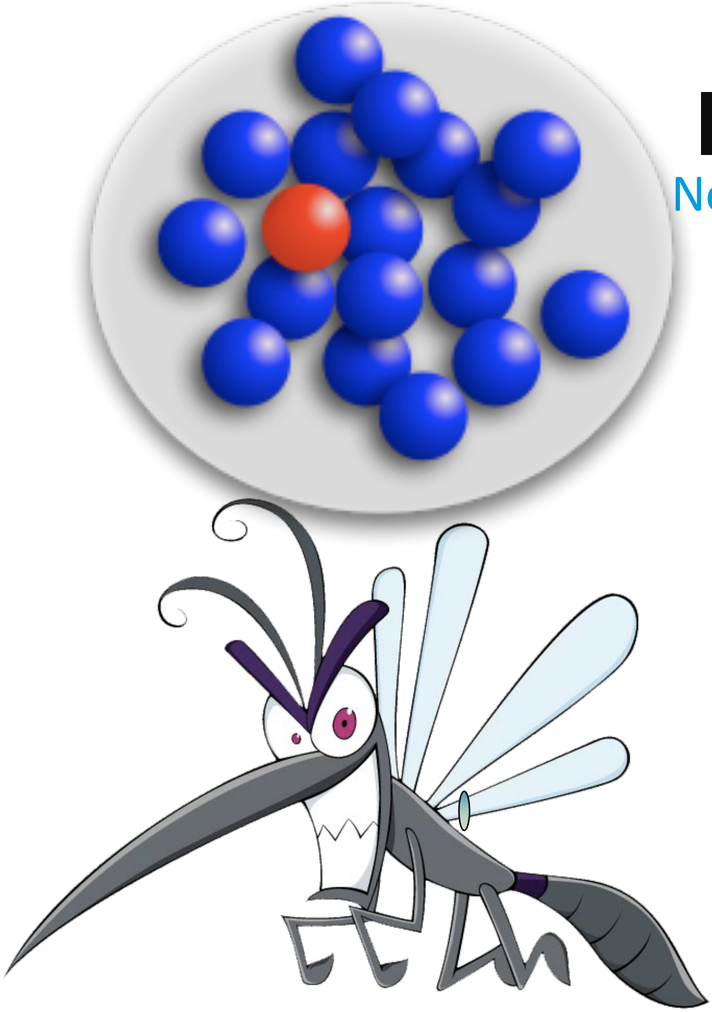


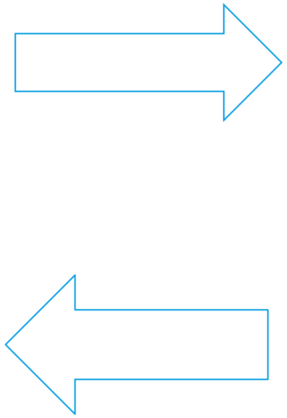


D. Brockmann and D. Helbing. The Hidden Geometry of Complex, Network-Driven Contagion Phenomena. Science, 342:1337-1342, 2014.

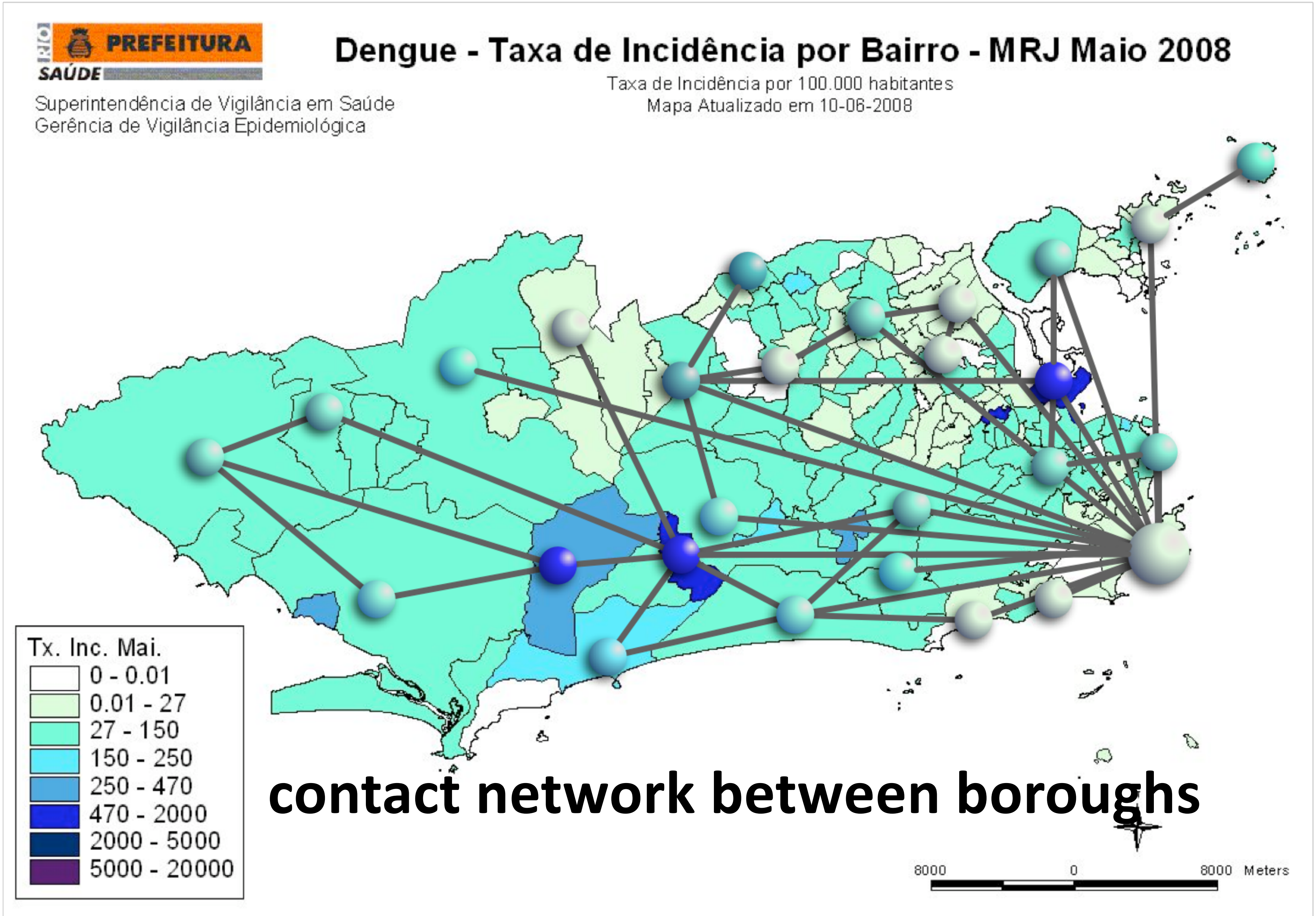
DENGUE

Not only a Brazilian problem





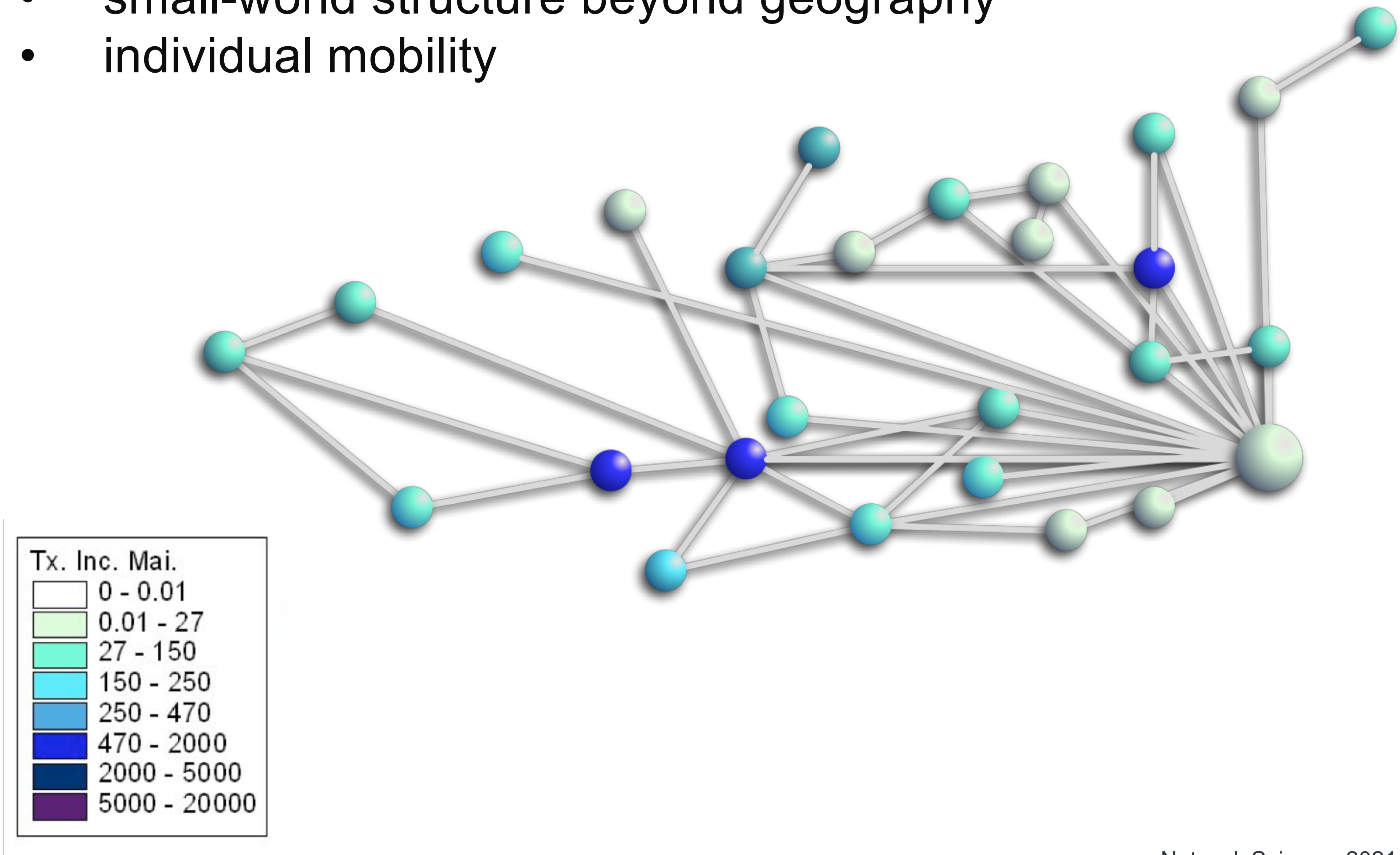
Two interacting populations



contact network between boroughs

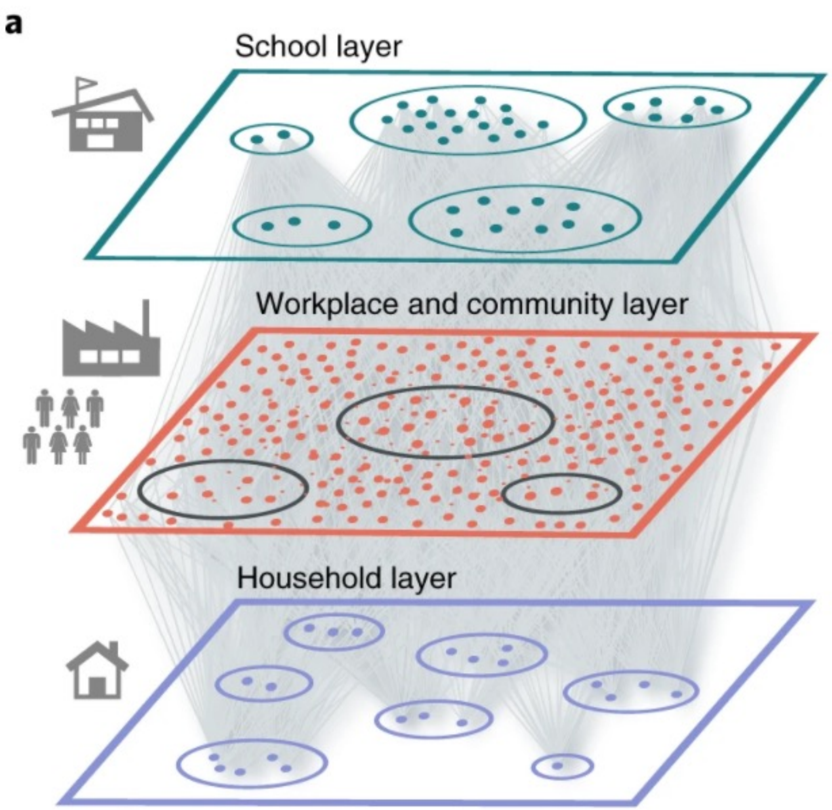
network theory

- heterogeneous graphs
- small-world structure beyond geography
- individual mobility

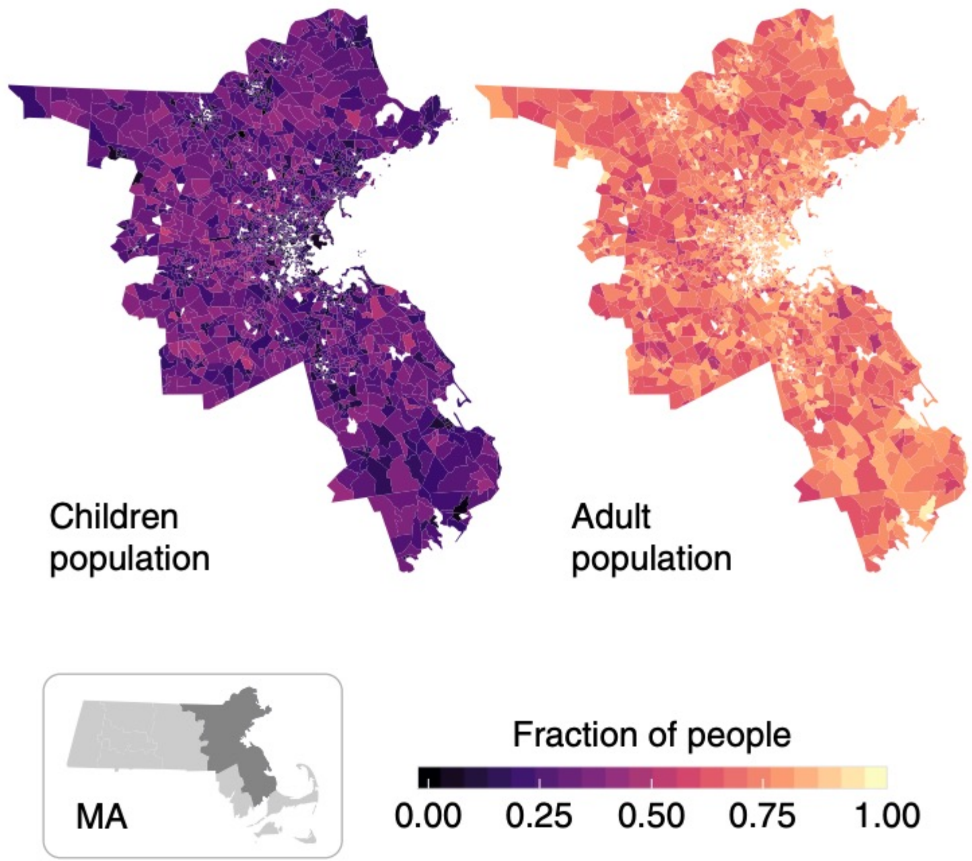


Agent-based models

(COVID-19, 2020)



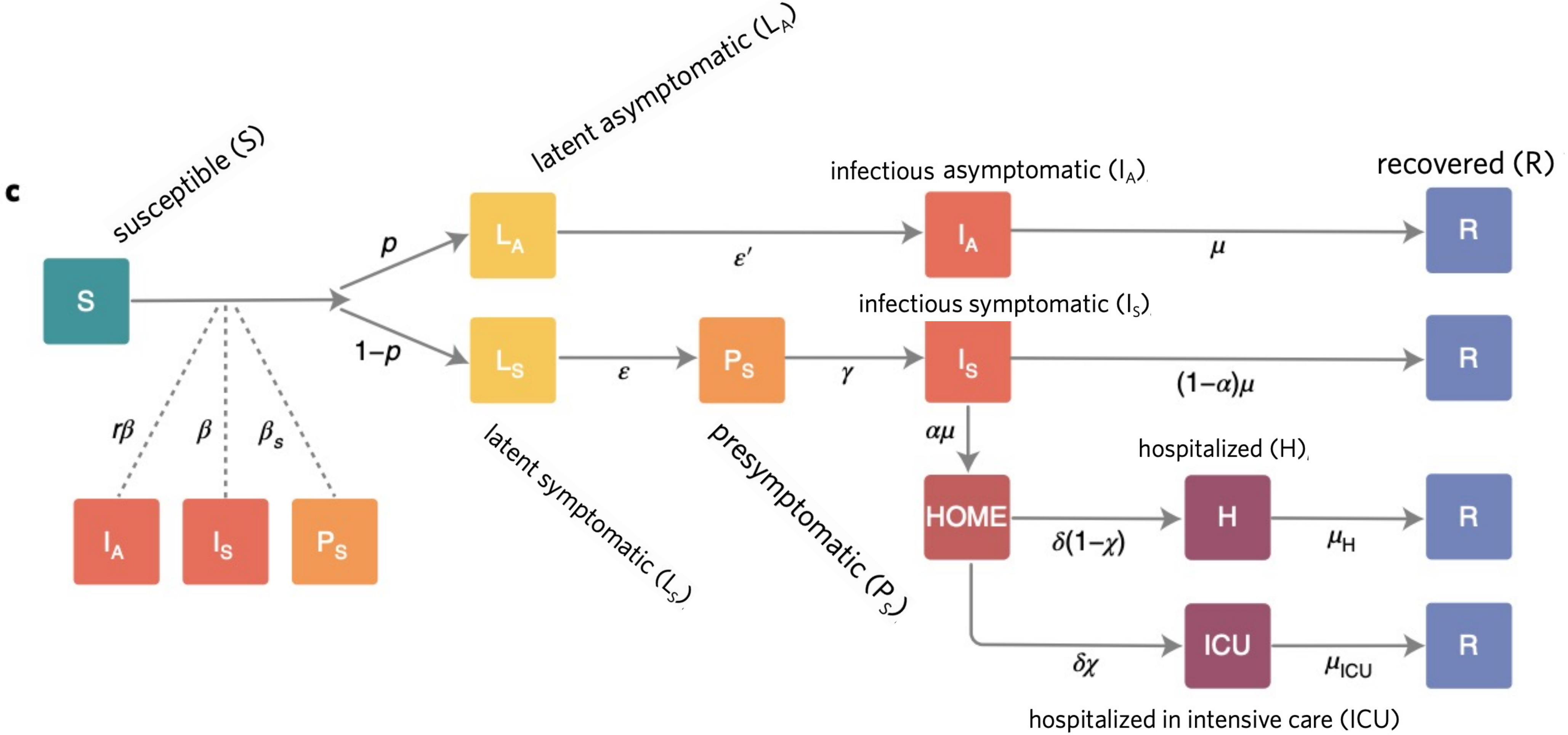
Schematic illustration of the weighted multilayer synthetic population built from mobility data in the metropolitan area of Boston. The agent-based system comprises around 64,000 adults and 21,000 children



Geographical distribution of the agents.

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

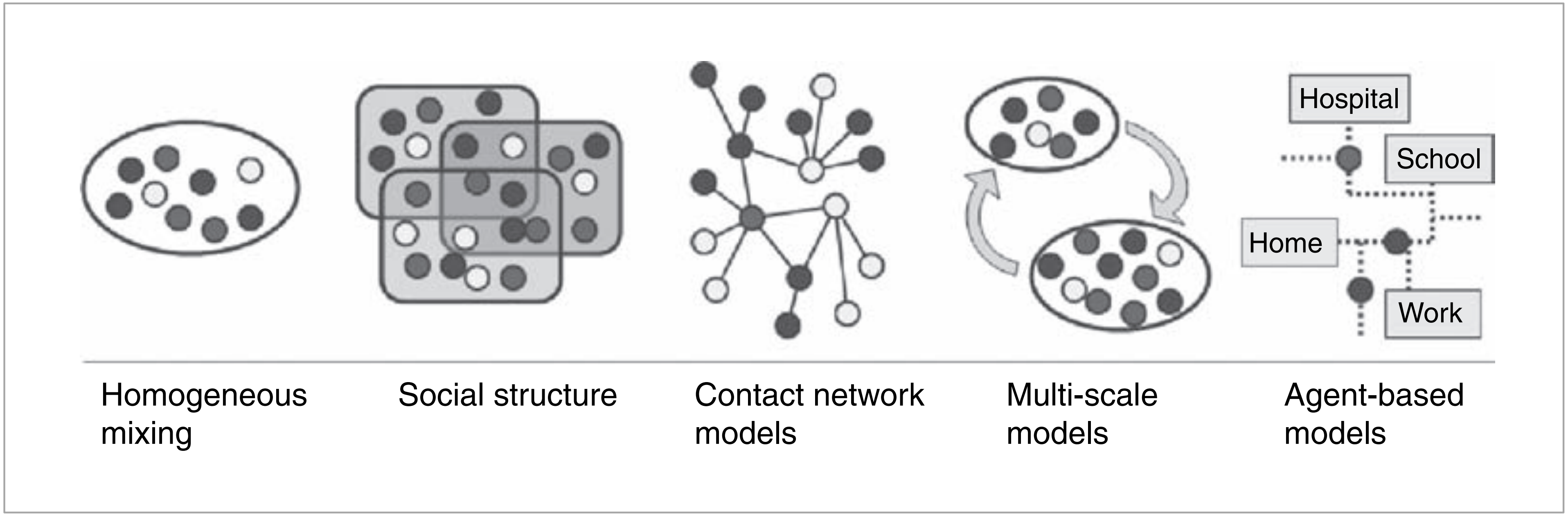
Agent-based models



Stochastic simulations of COVID-19 dynamics

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

OVERVIEW:
Classes of models in computational epidemiology



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Can we resort to similar techniques to address social systems?