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Lecture 15.ns10

Course: Complex Networks Analysis and Visualization
Sub-Module: NetSci



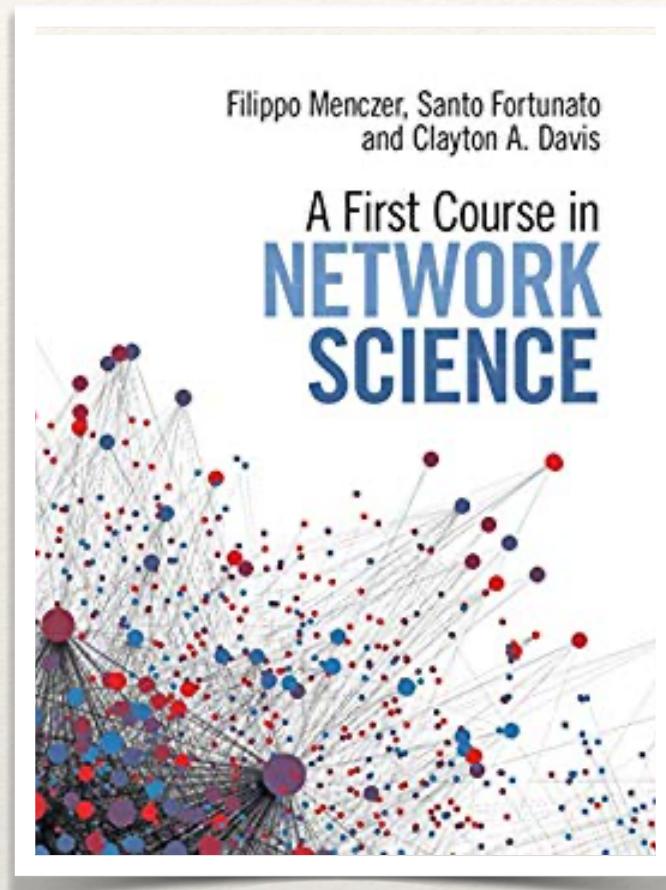
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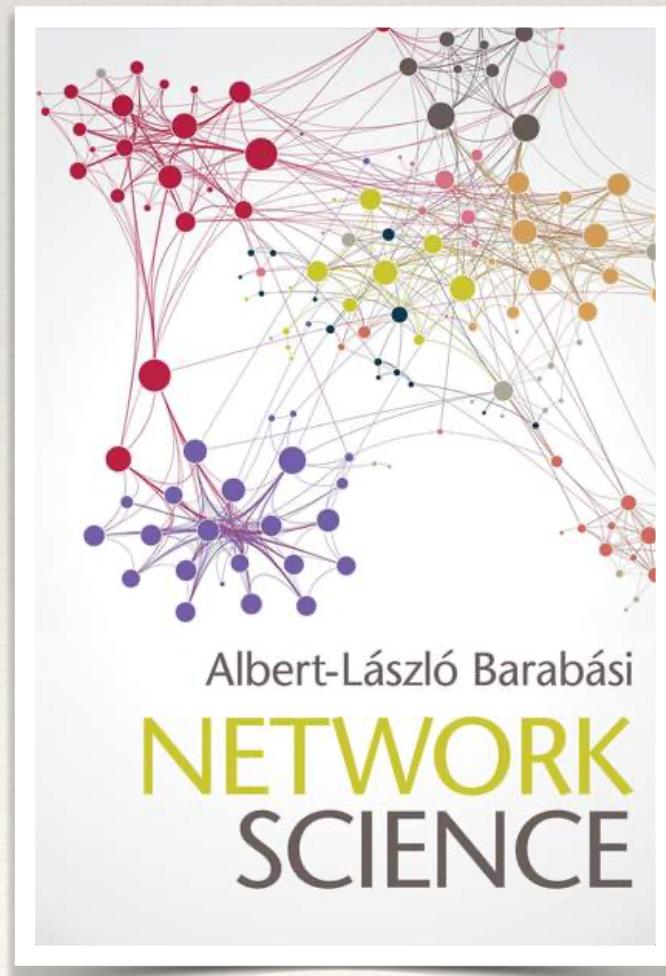


Epidemic spreading in heterogeneous networks

References



[ns1] Chapter 7: Dynamics
Study 7.2: Epidemic Spreading
(sections: 7.1, 7.3, 7.4 contain a very quick review of dynamics we have studied more deeply in our previous lectures - not necessary)



[ns3] Chapter 10: Spreading Phenomena
Sections: 10.1-10.7
<http://networksciencebook.com/chapter/10>

Agenda

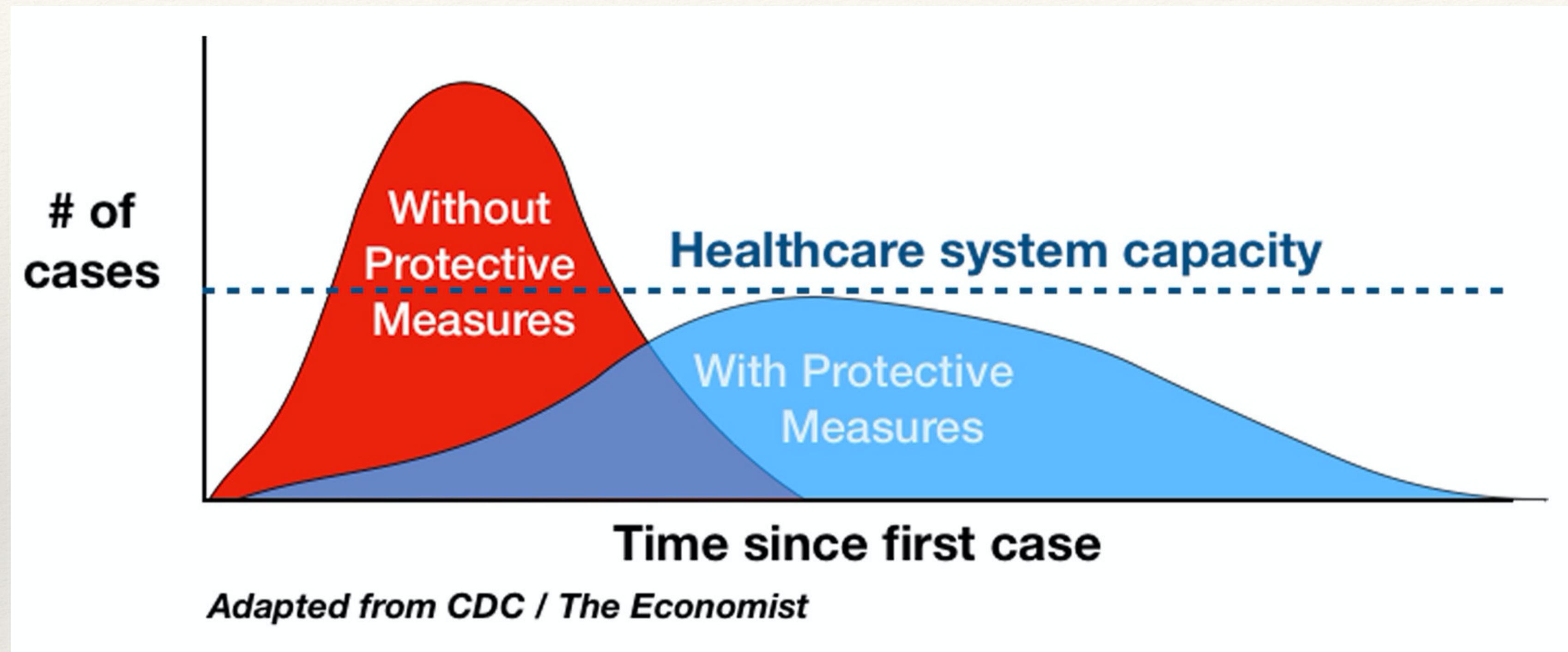
- ❖ Epidemic spreading in heterogeneous networks
- ❖ A continuum approach to model epidemics
- ❖ Heterogeneous networks epidemics
- ❖ Contact networks
- ❖ Immunization
- ❖ Predictions
- ❖ Surveillance, and more

Epidemic spreading in heterogeneous networks

Recap

- ❖ We learnt how to model epidemic spreading
 - ❖ branching processes
 - ❖ compartmental models (i.e., SIR, SIS, SIRS)
- ❖ We found that the *basic reproductive number* $R_0 = \frac{\beta k}{\mu}$, a measure on how many people an infect individual can spread the diseases, returns a useful **threshold**
 - ❖ if $R_0 < 1$, the epidemic will die out spontaneously
 - ❖ if $R_0 > 1$, the epidemic has a chance to survive forever
- ❖ Small changes on variables defining R_0 can have large effects on the spread of the disease

Flatten the curve



"Keeping the curve down — diminishing the rate at which new cases occur — prevents overtaxing the finite resources (represented by the dotted line) available to treat it."

“Real” networks

- ❖ If we want to add more reality to our models, we need make some observations
- ❖ We made some assumptions that aren't realistic
 - ❖ real networks are not trees (as in branching processes)
 - ❖ *fully mixed hypothesis* - assuming that each individual has the same chance to coming into contact with an infected one (basically, we are assuming that people meet completely at random - *homogeneously*)

Recap: Heterogeneity

- ❖ Let's define the **heterogeneity parameter** $\kappa = \frac{\langle k^2 \rangle}{\langle k \rangle^2}$, where
 - $\langle k \rangle = \frac{1}{N} \sum_i k_i$ that is the **mean**, and
 - $\langle k^2 \rangle = \frac{1}{N} \sum_i k_i^2$, that is the **variance**
- ❖ This is a measure on how broad is the degree distribution
- ❖ In random networks: $\langle k^2 \rangle \approx \langle k \rangle^2 \Rightarrow \kappa \approx 1$
- ❖ Conversely, if the distribution is very heterogeneous: $\kappa \gg 1$

Network	Nodes (N)	Links (L)	Average degree ($\langle k \rangle$)	Maximum degree (k_{max})	Heterogeneity parameter (κ)
Facebook Northwestern Univ.	10,567	488,337	92.4	2,105	1.8
IMDB movies and stars	563,443	921,160	3.3	800	5.4
IMDB co-stars	252,999	1,015,187	8.0	456	4.6
Twitter US politics	18,470	48,365	2.6	204	8.3
Enron Email	36,692	367,662	10.0	1,383	14.0
Wikipedia math	15,220	194,103	12.8	5,171	38.2
Internet routers	190,914	607,610	6.4	1,071	6.0
US air transportation	546	2,781	10.2	153	5.3
World air transportation	3,179	18,617	11.7	246	5.5
Yeast protein interactions	1,870	2,277	2.4	56	2.7
C. elegans brain	297	2,345	7.9	134	2.7
Everglades ecological food web	69	916	13.3	63	2.2

Recap: Power laws

In many cases, the degree distribution is characterized by (an approx. of) a **power law function**
 $p_k \propto k^{-\gamma}$

It is possible to prove that when $2 < \gamma \leq 3$, then $\langle k^2 \rangle \rightarrow \infty$

That would imply that, with real data:

- (i) the variance will be different order of magnitude greater than the mean \Rightarrow the average has not statistical significance (**scale-free regime**)
- (ii) $\kappa \rightarrow \infty$, hence the distribution is highly heterogeneous

However, *power-law distributions* and *scale-free networks* are just particular cases of extreme heterogeneity; we want to focus on **heterogeneous networks** (that are very common in the real world)

Preferential Attachment

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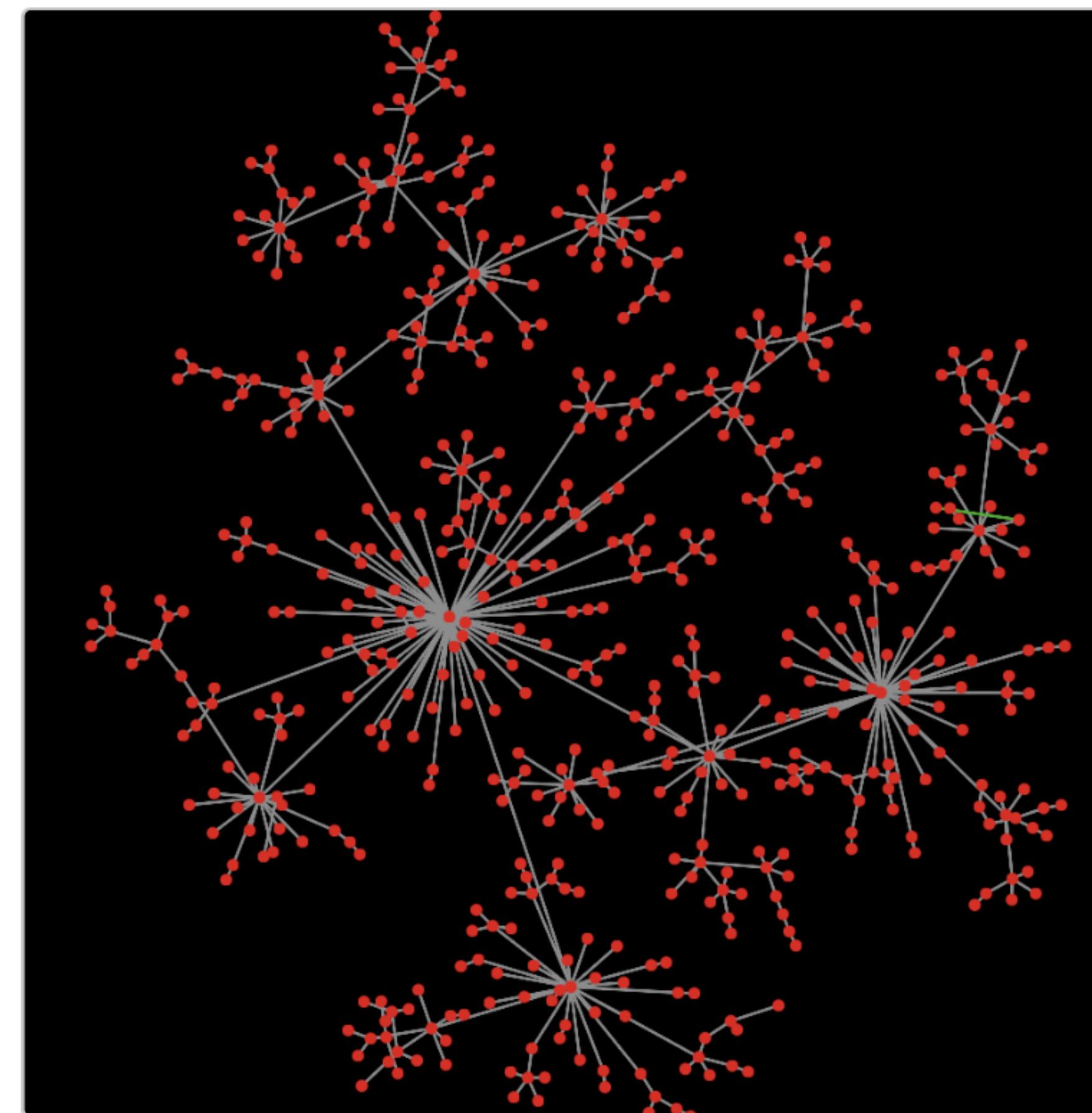
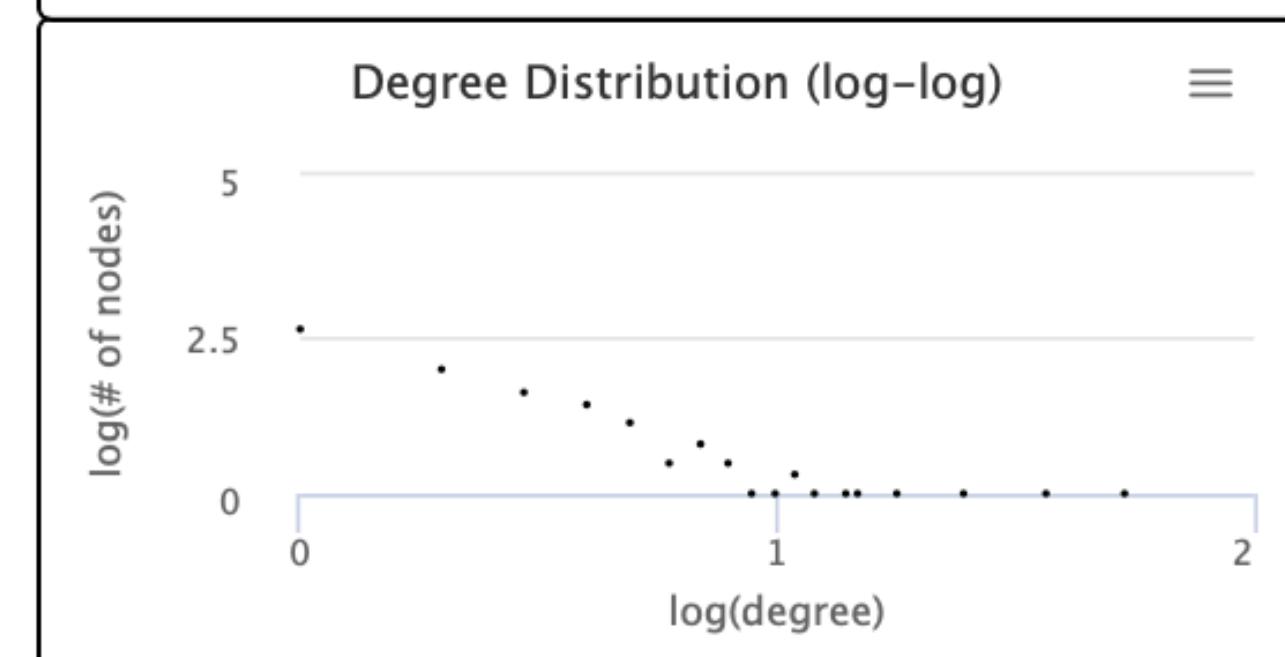
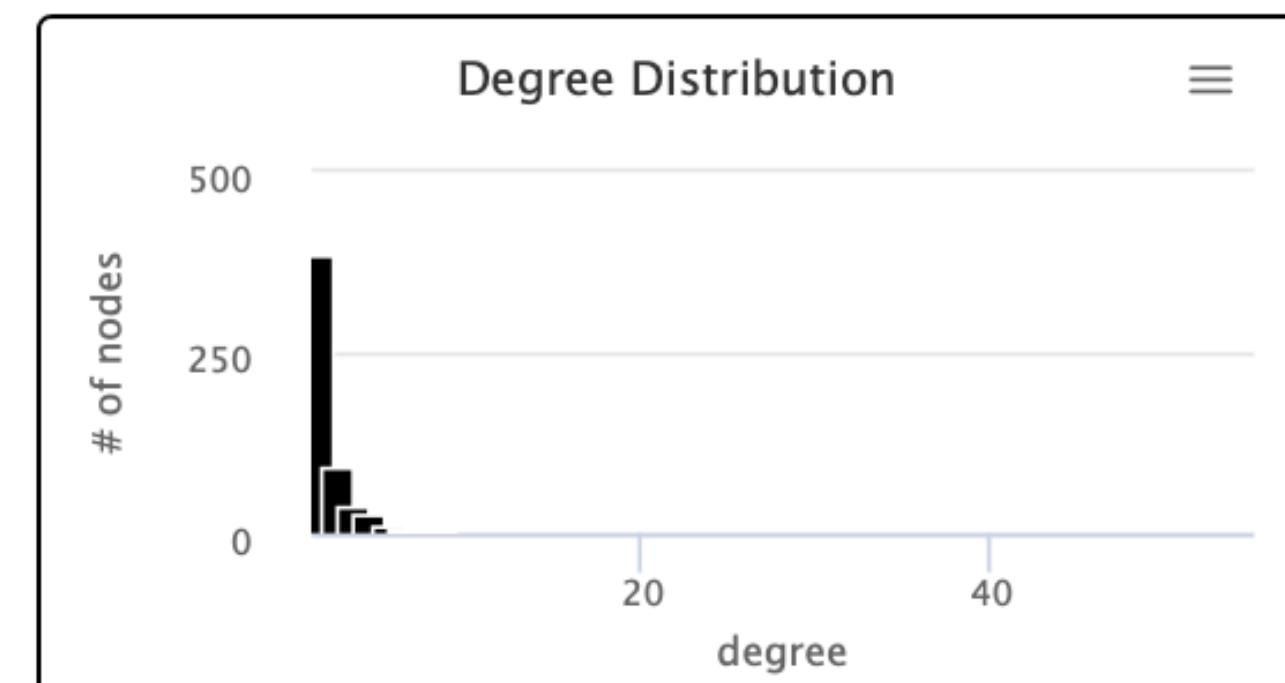
model speed

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setup
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plot?
 layout?

of nodes
574



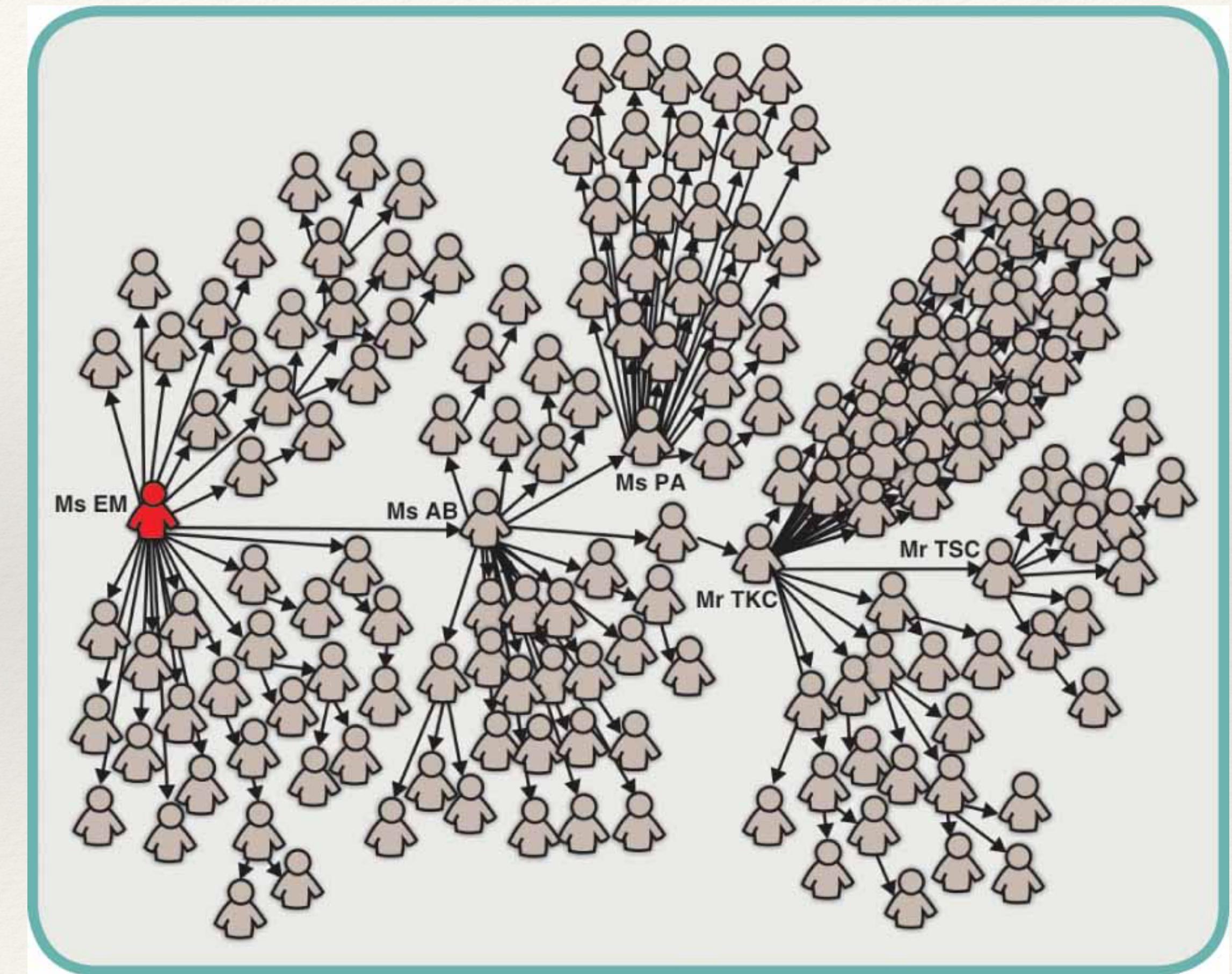
Hubs are super-spreaders

One-hundred-forty-four of Singapore's 206 probable SARS cases were traced to a chain of five individuals that included four "superspreaders."

CREDIT: WHO/WPRO

The fully-mixed hypothesis cannot hold:
contacts are not homogeneous

What does it change?



A continuum approach to model epidemics

Revisiting compartmental modeling

- ❖ To capture heterogeneity, we need to get rid of the fixed k contacts
- ❖ As a first approximation, we use an average $\langle k \rangle$
- ❖ An individual can be, as before, in one of these *states* : **S**, **I**, and **R**
- ❖ It also turns out that the best way to capture network dynamics is to move to a **continuous description of time t**
- ❖ $s(t)$, $i(t)$, and $r(t)$: the fraction of individuals who are susceptible, infectious, and removed at time t
- ❖ Initial conditions ($t=0$) - with only one infectious individual:
$$i(0) = \frac{1}{N}, \quad s(0) = 1 - i(0) \quad \text{and, of course, } r(0) = 0$$

SI model

- ❖ an infectious node transmit the diseases with prob. β : $i + s \xrightarrow{\beta} i + i$
- ❖ Let's assume there is no recovery and no one will be ever removed: when individuals enter the I compartment, they stay there forever
- ❖ Not hard to guess that everyone, when $t \rightarrow \infty$, will enter the infectious state.
- ❖ The question is: **how soon?**
- ❖ The plan: Let's find a formula for $i(t)$.
- ❖ *Full mixed hypothesis*: an infected individual comes into contact with other $\langle k \rangle s(t)$ susceptible individuals in a unit time

- ❖ This means that the new infections $di(t)$ during a time dt is:

$$di(t) = \beta\langle k \rangle s(t)i(t)dt$$

- ❖ observe that $s(t) = 1 - i(t)$. Moreover, for the sake of simplicity, let's get rid the (t) variable

$$\frac{di}{dt} = \beta\langle k \rangle i(1 - i)$$

- ❖ We can solve the above equation to obtain:

$$\frac{di}{i} + \frac{di}{1 - i} = \beta\langle k \rangle dt$$

- ❖ Integrating both sides:

$$\ln i - \ln(1 - i) + C = \beta\langle k \rangle t$$

- ❖ If we set a generic initial condition $i_0 = i(0)$, from the above equation:

$$\ln i - \ln(1 - i) + C = \beta\langle k \rangle t$$

- ❖ we get C :

$$C = \ln(1 - i_0) - \ln i_0$$

- ❖ Finally, after some algebraic substitutions:

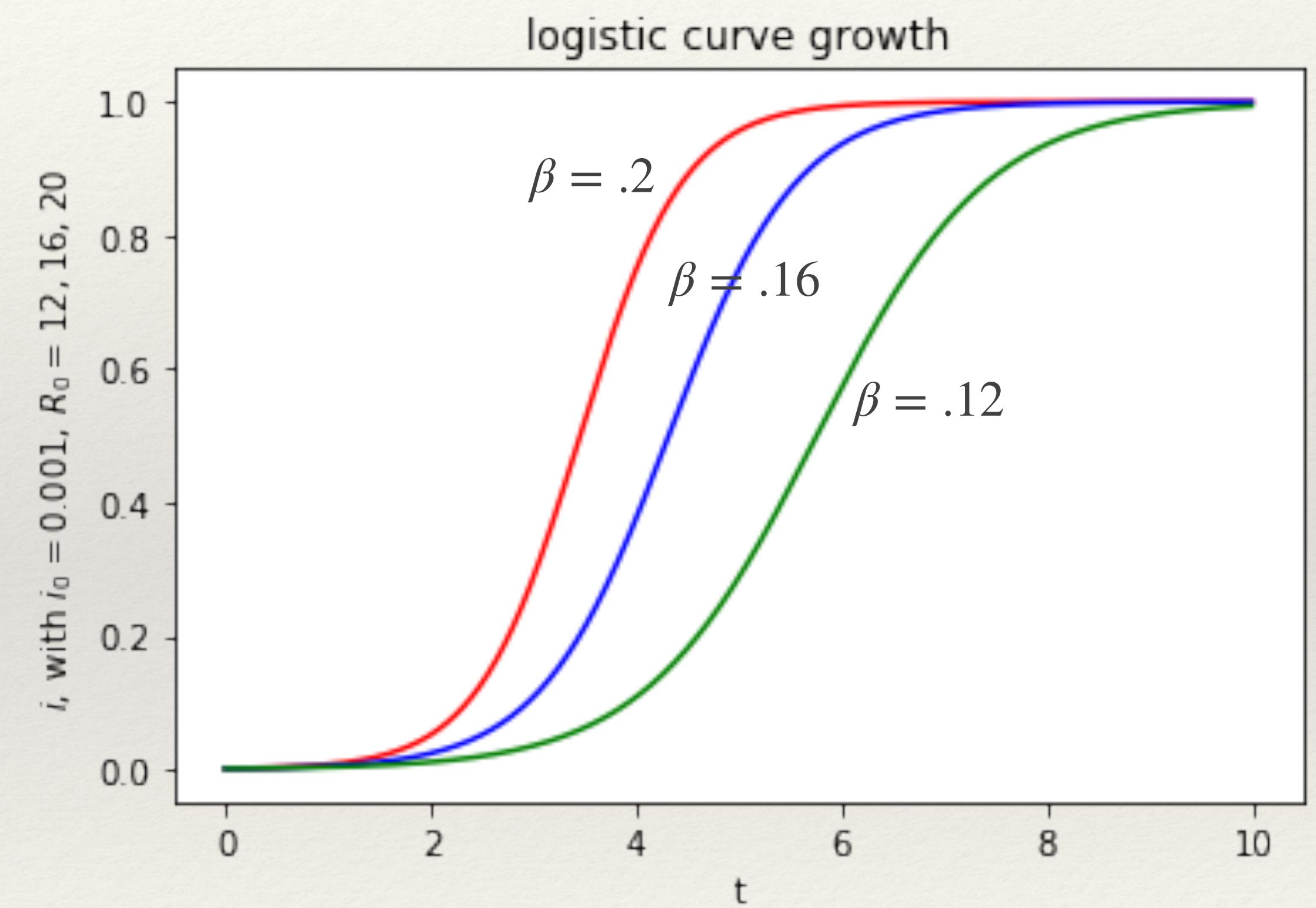
$$\ln i - \ln(1 - i) + \ln(1 - i_0) - \ln i_0 = \beta\langle k \rangle t$$

$$\Rightarrow \frac{i}{1 - i} \frac{1 - i_0}{i_0} = e^{\beta\langle k \rangle t} \Rightarrow \frac{1 - i_0}{i_0} i = e^{\beta\langle k \rangle t} - e^{\beta\langle k \rangle t} i$$

$$\Rightarrow \frac{1 - i_0 + i_0 e^{\beta\langle k \rangle t}}{i_0} i = e^{\beta\langle k \rangle t} \Rightarrow i = \frac{i_0 e^{\beta\langle k \rangle t}}{1 - i_0 + i_0 e^{\beta\langle k \rangle t}}$$

Logistic growth curve

- ❖ The equation $i = \frac{i_0 e^{\beta \langle k \rangle t}}{1 - i_0 + i_0 e^{\beta \langle k \rangle t}}$ is called *logistic growth curve*.
 - ❖ it grows exponentially at the beginning
 - ❖ After a while, there are very few susceptible around, and it is harder to infect some new individual
 - ❖ $\beta \langle k \rangle$ is the speed of the infection (no threshold!)



Observation on SI epidemics

- ❖ Very few diseases can be modeled with SI processes: the diseases that do not kill their victims, are eventually defeated by the immune system
- ❖ However, SI models are still useful to understand that even the worst case scenario can be treated by delaying as much as possible the so called *characteristic time* τ , i.e., you can move to the right the inflection point of the logistic
- ❖ This time depends on β and $\langle k \rangle$: $\tau = \frac{1}{\beta \langle k \rangle}$

SIS model

- ❖ An infectious node transmit the diseases with prob. β : $i + s \xrightarrow{\beta} i + i$
- ❖ Infectious individuals recover with rate μ : $i \xrightarrow{\mu} s$
- ❖ New question: is there a threshold for $R_0 = \frac{\beta\langle k \rangle}{\mu}$?

- ❖ The equation describing the dynamics of the model is:

$$\frac{di}{dt} = \beta\langle k \rangle i(1 - i) - \mu i$$

- ❖ The solution for this equation is:

$$i = \left(1 - \frac{\mu}{\beta\langle k \rangle}\right) \frac{Ce^{(\beta\langle k \rangle - \mu)t}}{1 + Ce^{(\beta\langle k \rangle - \mu)t}}$$

- ❖ where the initial condition $i(0) = i_0$, gives

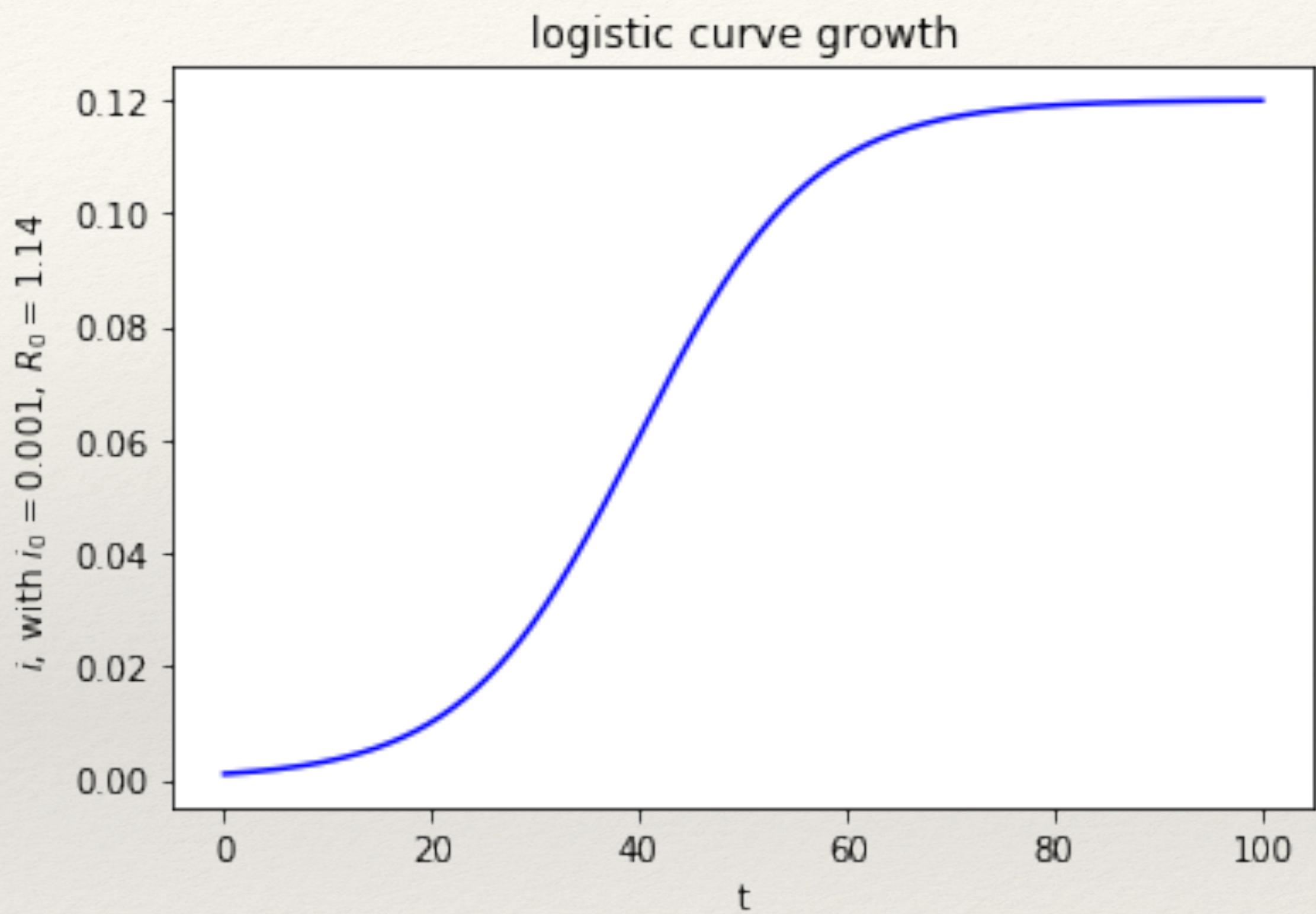
$$C = \frac{i_0}{1 - i_0 - \frac{\mu}{\beta\langle k \rangle}}$$

- ❖ What happens to

$$i = \left(1 - \frac{\mu}{\beta\langle k \rangle}\right) \frac{Ce^{(\beta\langle k \rangle - \mu)t}}{1 + Ce^{(\beta\langle k \rangle - \mu)t}}$$

when $R_0 = \frac{\beta\langle k \rangle}{\mu}$ changes and $t \rightarrow \infty$?

- ❖ if $R_0 > 1 \Rightarrow \beta\langle k \rangle > \mu$,
 then $\lim_{t \rightarrow \infty} i(t) = 1 - \frac{\mu}{\beta\langle k \rangle}$

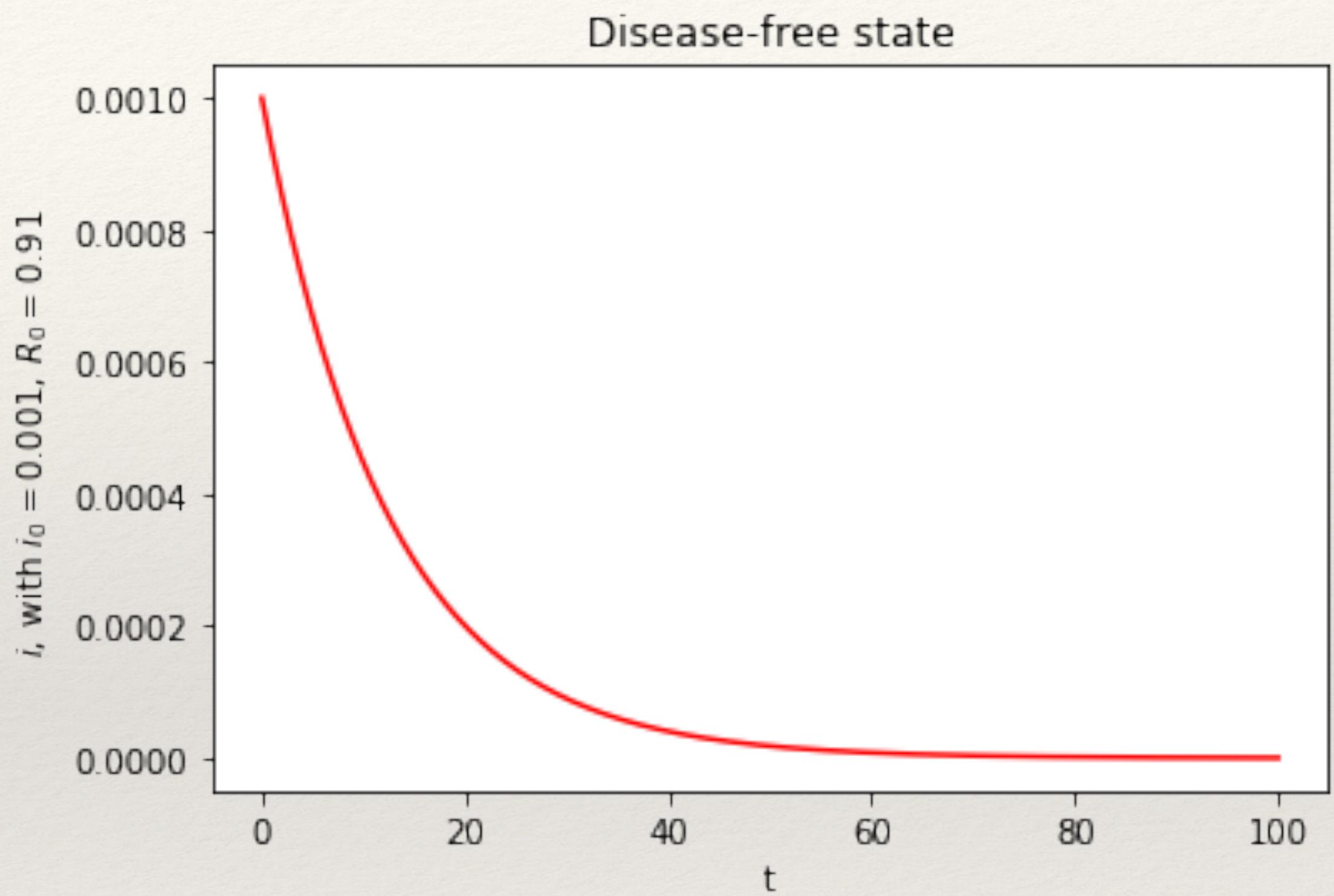


- ❖ What happens to

$$i = \left(1 - \frac{\mu}{\beta\langle k \rangle}\right) \frac{Ce^{(\beta\langle k \rangle - \mu)t}}{1 + Ce^{(\beta\langle k \rangle - \mu)t}}$$

when $R_0 = \frac{\beta\langle k \rangle}{\mu}$ changes and $t \rightarrow \infty$?

- ❖ if $R_0 < 1 \Rightarrow \beta\langle k \rangle < \mu$,
then $\lim_{t \rightarrow \infty} i(t) = 0$



SIR model

- ❖ An infectious node transmit the diseases with prob. β : $i + s \xrightarrow{\beta} i + i$
- ❖ Infectious individuals recover with rate μ : $i \xrightarrow{\mu} r$
- ❖ The question is: is there a threshold for $R_0 = \frac{\beta\langle k \rangle}{\mu}$ even here?

- ❖ The **equations** describing the dynamics of the **SIR model** are:

$$\frac{ds}{dt} = -\beta \langle k \rangle i s = -\beta \langle k \rangle i (1 - r - i)$$

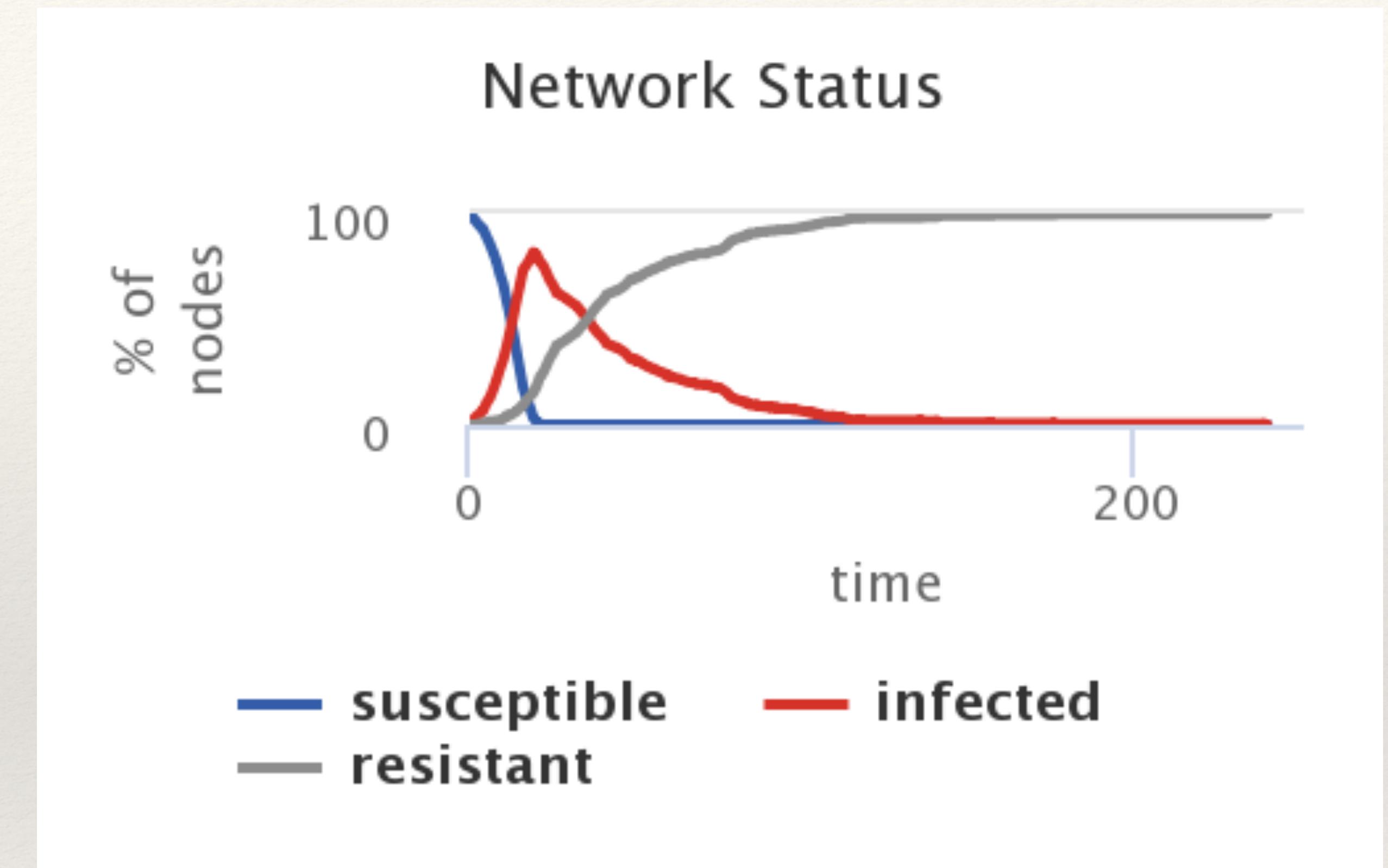
$$\frac{di}{dt} = \beta \langle k \rangle i (1 - r - i) - \mu i$$

$$\frac{dr}{dt} = \mu i$$

- ❖ and also: $s + r + i = 1$, $s(0) = s_0$, $r(0) = 0$, $i(0) = i_0 = 1 - s_0$

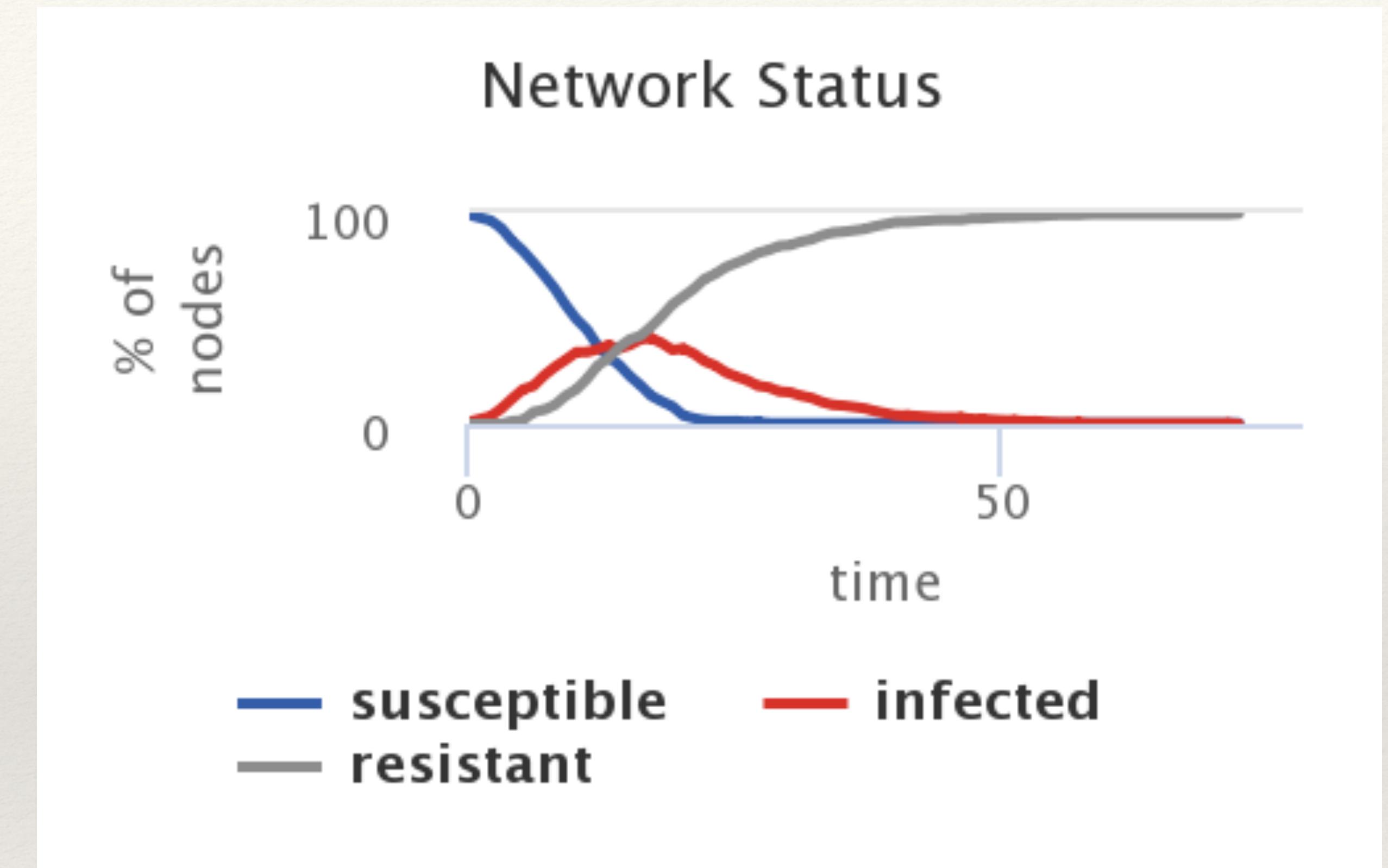
one netlogo simulation

- ❖ $s_0 = 0.99, i_0 = 0.01, r_0 = 0$
- ❖ $\beta = 10\%, \langle k \rangle = 10, \mu = 2.5\%$
 $R_0 = 40$
- ❖ peak: $i(19) \approx 74\%$



one netlogo simulation

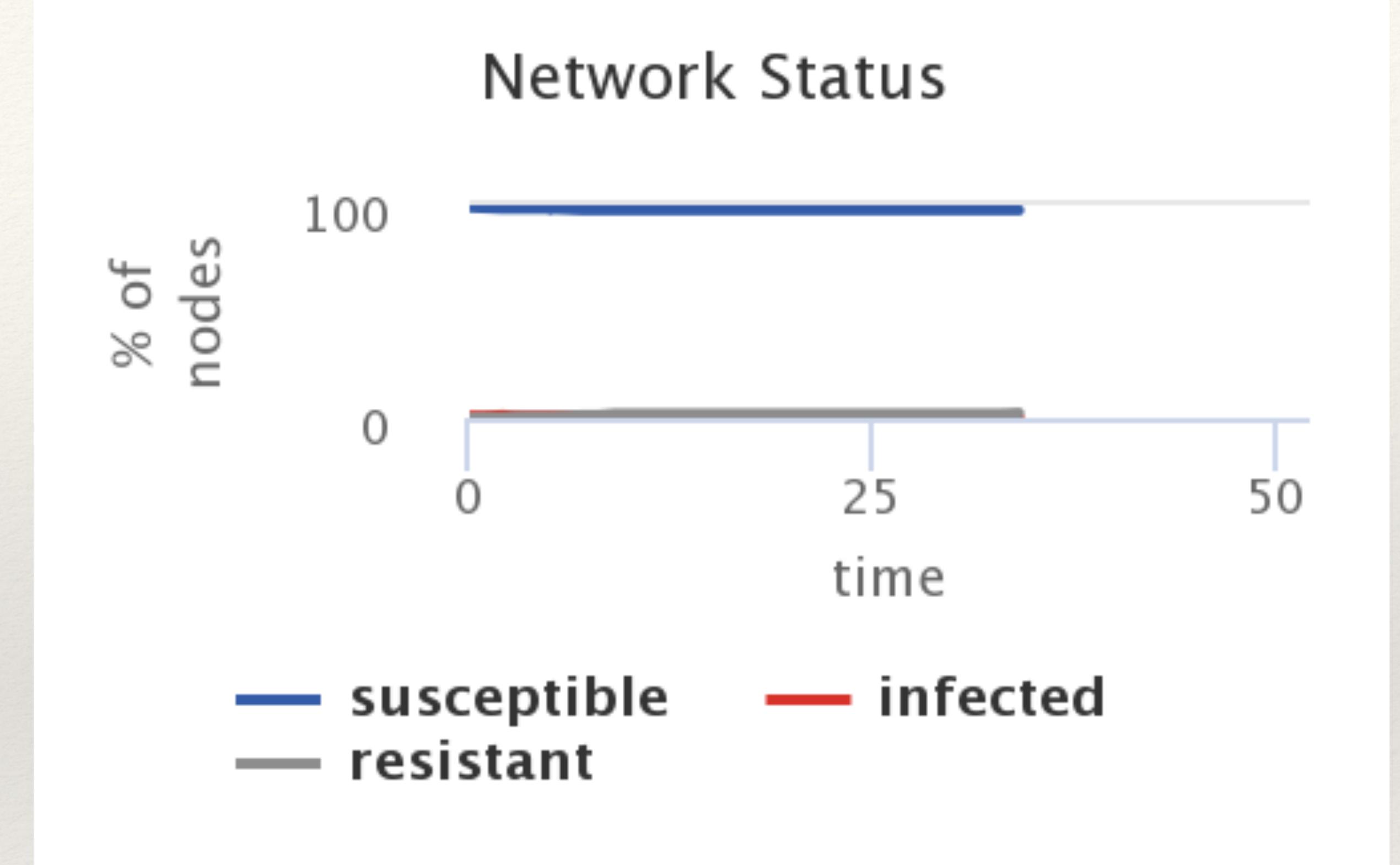
- ❖ $s_0 = 0.99, i_0 = 0.01, r_0 = 0$
- ❖ $\beta = 10\%, \langle k \rangle = 10, \mu = 10\%$
 $R_0 = 10$
- ❖ peak: $i(17) \approx 31\%$



We are flattening the curve!

one netlogo simulation

- ❖ $s_0 = 0.99, i_0 = 0.01, r_0 = 0$
- ❖ $\beta = 0.9\%, \langle k \rangle = 10, \mu = 10\%$
 $R_0 < 1$
- ❖ peak: $i(10) \approx 0.33\%$



Finally, the epidemic dies out

Calculating the asymptotic value of r

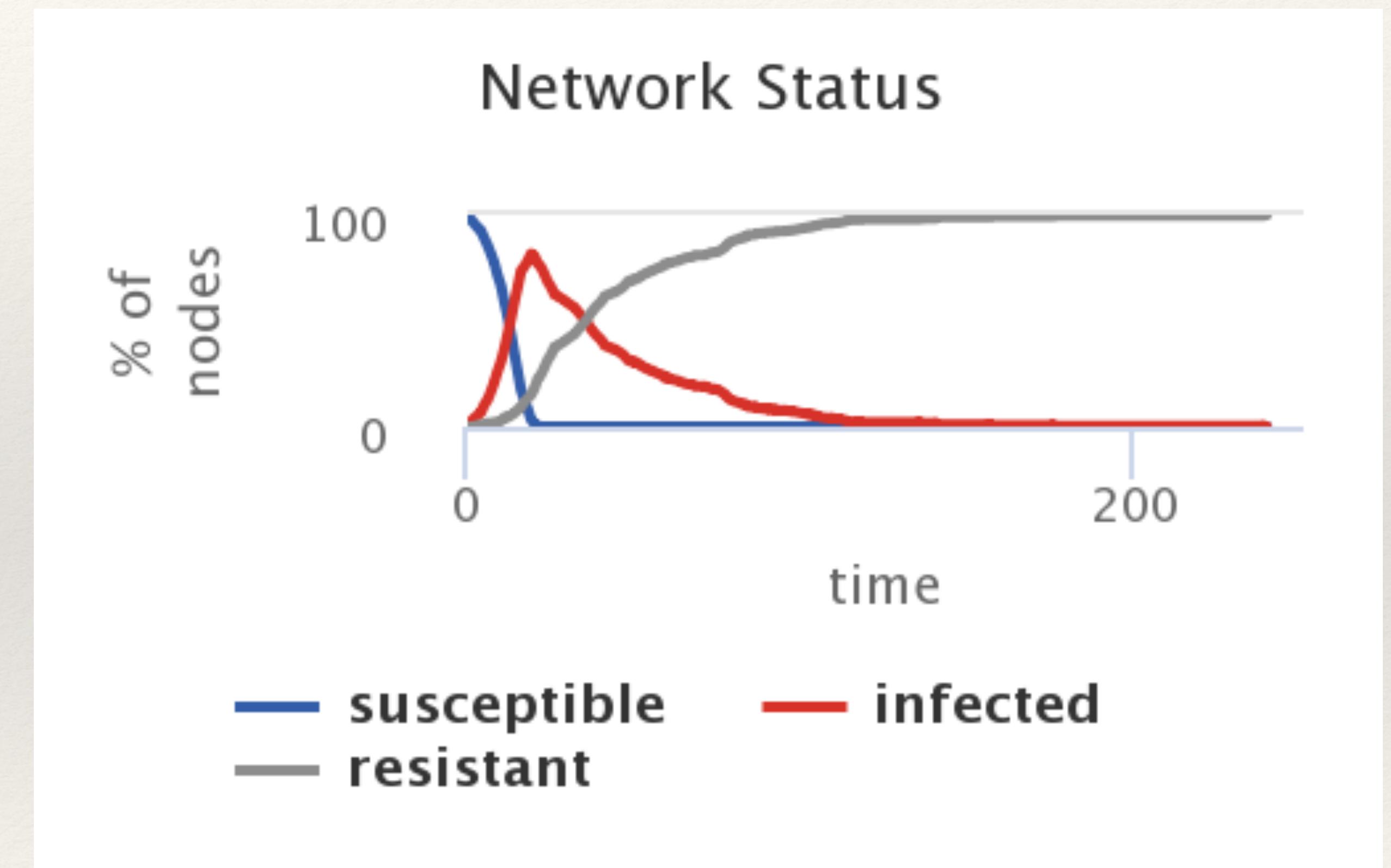
$$\frac{dr}{dt} = \mu(1 - r - s_0 e^{-R_0 r}) = 0$$

$$\Rightarrow r = 1 - s_0 e^{-R_0 r}$$

for large networks ($N \rightarrow \infty$): $s_0 \approx 1$

$$\Rightarrow r = 1 - e^{-R_0 r}$$

- ❖ if $R_0 > 1$,
then $\lim_{t \rightarrow \infty} r = 1$
- ❖ if $R_0 < 1$,
then $\lim_{t \rightarrow \infty} r = 0$



Proof: finding a formula for $r(t)$

$$\begin{aligned} \frac{ds}{dt} = -\beta\langle k \rangle s i \Rightarrow i &= -\frac{1}{\beta\langle k \rangle s} \frac{ds}{dt} \\ &\Rightarrow \frac{1}{\mu} \frac{dr}{dt} = -\frac{1}{\beta\langle k \rangle s} \frac{ds}{dt} \\ &\Rightarrow \frac{\beta\langle k \rangle}{\mu} \frac{dr}{dt} = -\frac{1}{s} \frac{ds}{dt} \\ &\Rightarrow \frac{1}{s} \frac{ds}{dt} = -R_0 \frac{dr}{dt} \end{aligned}$$

$$\frac{1}{s} \frac{ds}{dt} = -R_0 \frac{dr}{dt}$$

integrating both sides

$$\Rightarrow \ln s = -R_0 r + C$$

calculating C at $t = 0$: $C = \ln s_0$ (because $r_0 = 0$)

$$\Rightarrow \ln s = -R_0 r + \ln s_0$$

hence, we get

$$\Rightarrow s = s_0 e^{-R_0 r}$$

remember that $s + r + i = 1$ and $\frac{dr}{dt} = \mu i$

$$\Rightarrow \frac{dr}{dt} = \mu(1 - r - s_0 e^{-R_0 r})$$

Heterogeneous networks epidemics

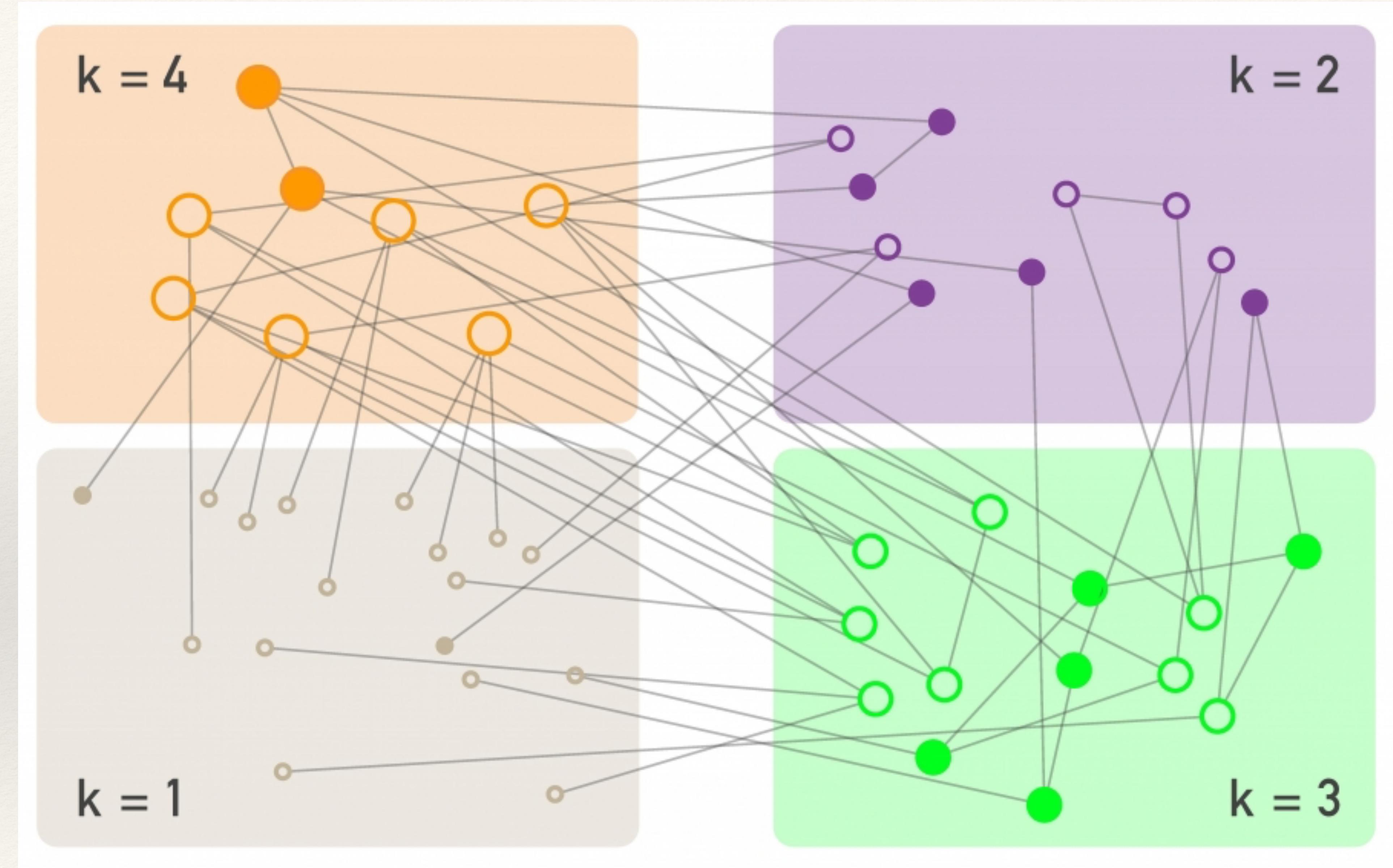
Getting rid of the full-mixed hypothesis

- ❖ Now we have a framework that allows us to run our processes on every networks
- ❖ We need to get rid of the *full-mixed hypothesis* because our networks have hubs, super-spreaders and are, basically, **heterogeneous**
- ❖ The change of the paradigm is due to Romualdo Pastor-Satorras and Alessandro Vespignani with their 2001 paper.

Degree-block approximation

- ❖ In heterogeneous networks, individuals with more links are more likely to be in contact with an infected individual, hence they are more likely to be infected
- ❖ The degree-block approximation distinguishes nodes based on their degree and **assumes** that nodes with the same degree are statistically equivalent
- ❖ $i_k = I_k/N_k$ is the fraction of nodes with degree k that are infected among all N_k degree- k nodes in the network. The total fraction of infected nodes can be calculated as it follows:

$$i = \sum_k p_k i_k$$



From: Albert-László Barabási, Network Science [ns3]

SI model on a network

- ❖ We write the SI model for each degree k separately

$$\frac{di_k}{dt} = \beta(1 - i_k)k\Theta_k$$

- ❖ Same structure as before, but
 - ❖ the average $\langle k \rangle$ is replaced by each node's actual degree k
 - ❖ we have the **density function** Θ_k : the fraction of infected neighbors of a susceptible node with degree k
 - ❖ now we have a system of k_{\max} coupled equations

Exploring early-time behavior of i_k

- ❖ If we lack a cure, the only way to alter the course of an epidemic is to try to slow the spread of the epidemics as much as possible
- ❖ When i_k is small, the higher order term of $\beta i_k \Theta_k$ can be neglected:

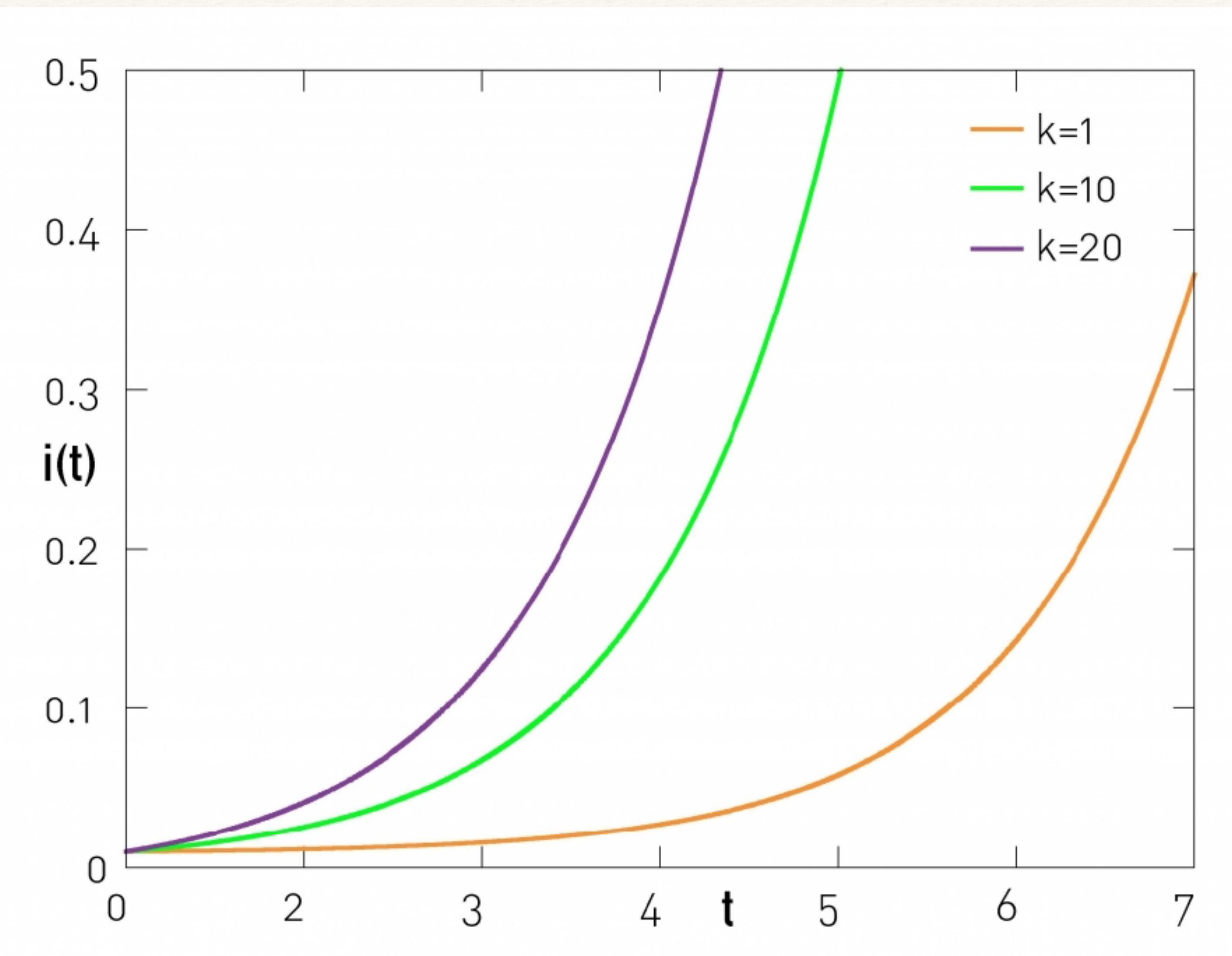
$$\frac{di_k}{dt} \approx \beta k \Theta_k$$

- ❖ Assuming no degree correlation, we can prove that Θ_k is independent of k :
$$\Theta_k \approx i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} e^{1/\tau}, \text{ where } \tau = \frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)}$$
- ❖ τ is the *characteristic time* for the spread of the pathogen

Fraction of Infected Nodes in the SI model

Observe that the group of nodes with higher degree has a higher fraction of infected nodes

Consequently, at any time virtually all hubs are infected, but small-degree nodes tend to be disease free. Hence the disease is maintained in the hubs, which in turn broadcast the disease to the rest of the network.



Observations on τ

- ❖ The characteristic time $\tau = \frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)}$ depends on the heterogeneity of the network
- ❖ In **random networks**, i.e., $\kappa = \frac{\langle k^2 \rangle}{\langle k \rangle^2} \approx 1 \Rightarrow \langle k^2 \rangle \approx \langle k \rangle^2 \Rightarrow \tau \approx \frac{1}{\beta \langle k \rangle}$
(recovering the results under homogeneous assumption)
- ❖ In **heterogeneous networks**, i.e., $\kappa = \frac{\langle k^2 \rangle}{\langle k \rangle^2} \gg 1 \Rightarrow \langle k^2 \rangle \gg \langle k \rangle^2 \Rightarrow \tau \ll \frac{1}{\beta \langle k \rangle}$
- ❖ In **scale-free networks** (special case) we have that $\langle k^2 \rangle \xrightarrow{N \rightarrow \infty} \infty \Rightarrow \tau \rightarrow 0$

SI model and heterogeneity

- ❖ We already knew that with SI dynamics the diseases would reach all the individual
- ❖ We use characteristic time τ to understand the speed of the diffusion
 - ❖ In homogeneous networks $\tau = \frac{1}{\beta\langle k \rangle}$: some countermeasures can delay the propagation
 - ❖ **In heterogeneous networks the spread is almost instantaneous!**

SIS model in a network

- ❖ To understand the full impact of the network topology, let's explore the SIS model further
- ❖ The continuum equation describing the dynamics of the extension of the SIS model on a network is the following:

$$\frac{di_k}{dt} = \beta(1 - i_k)k\Theta_k(t) - \mu i_k$$

- ❖ The presence of the recovery term $-\mu i$ changes the characteristic time to:

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

Epidemic threshold

- ❖ Let's define $\lambda = \frac{\beta}{\mu}$ as the **spreading rate**: it depends only on biological characteristics of the pathogen
- ❖ Note that $R_0 = \lambda \langle k \rangle$
- ❖ The higher is λ , the more likely that the diseases will spread
- ❖ Let's look for the **epidemic threshold** λ_c : the pathogen can spread only if $\lambda > \lambda_c$

- ❖ Recall that $\tau = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - \mu \langle k \rangle}$, we want to find when the pathogen has a chance to persist:

$$\tau > 0 \Rightarrow \frac{\langle k \rangle}{\beta \langle k^2 \rangle - \mu \langle k \rangle} > 0 \Rightarrow \beta \langle k^2 \rangle > \mu \langle k \rangle \Rightarrow \lambda = \frac{\beta}{\mu} > \frac{\langle k \rangle}{\langle k^2 \rangle};$$

$$\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}$$

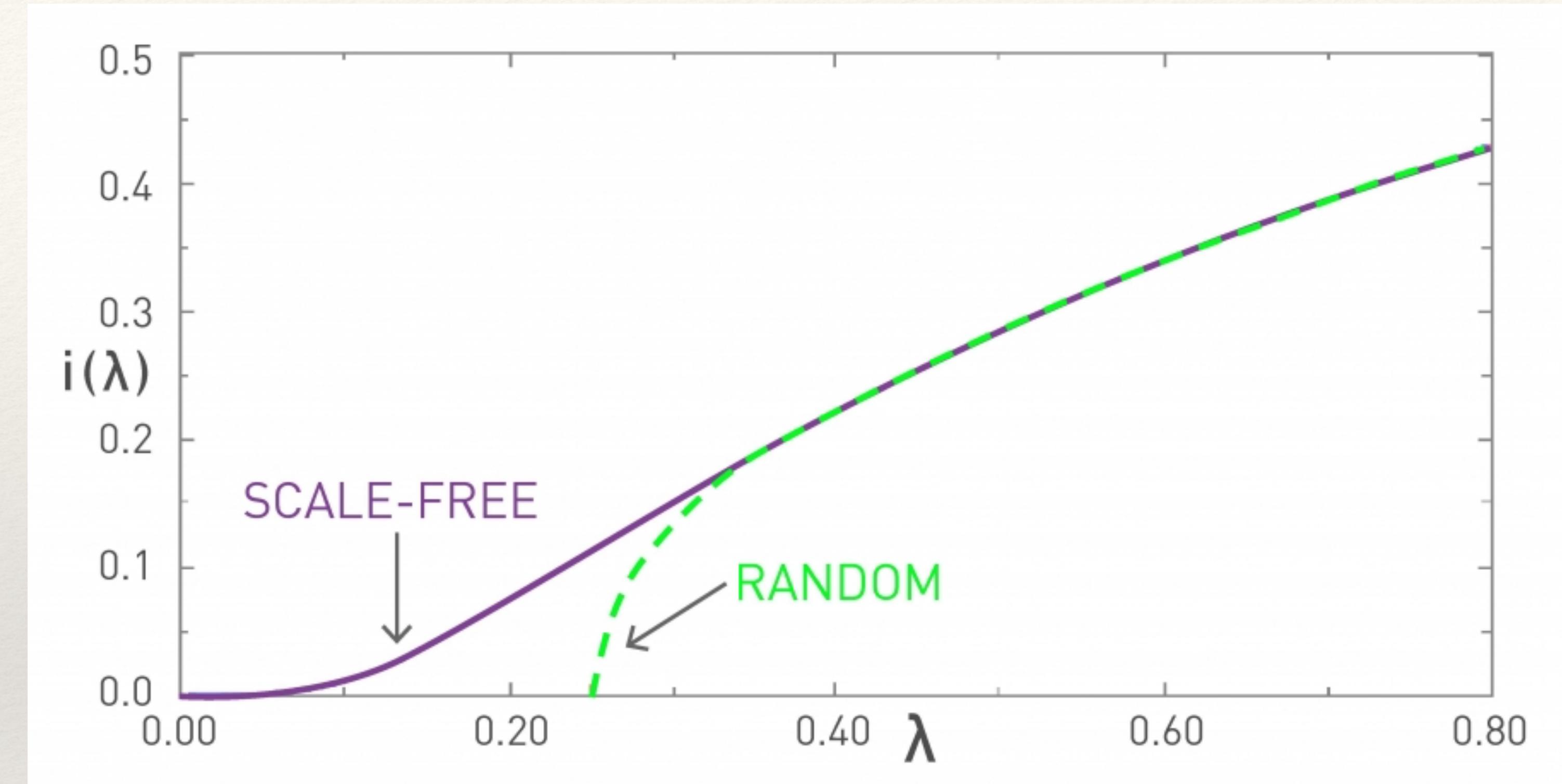
- ❖ In **random networks**, i.e., $\langle k^2 \rangle \approx \langle k \rangle^2 \Rightarrow \lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle} \approx \frac{1}{\langle k \rangle}$
 that confirms that, under homogeneous hypothesis, the pathogen has a
 chance to persist when $\lambda > \frac{1}{\langle k \rangle}$, or analogously, when $R_0 > 1$
- ❖ In **heterogeneous networks**, i.e., $\langle k^2 \rangle \gg \langle k \rangle^2$
 $\Rightarrow \lambda_c$ is expected to vanish for increasingly heterogeneous networks
- ❖ In **scale-free networks** (special case), we have that $\langle k^2 \rangle \xrightarrow{N \rightarrow \infty} \infty \Rightarrow \lambda_c \rightarrow 0$

Epidemic Threshold in the SIS model

The random network has a finite epidemic threshold λ_c , implying that a pathogen with a small spreading rate ($\lambda < \lambda_c$) must die out, i.e. $i(\lambda) = 0$.

If, however, the spreading rate of the pathogen exceeds λ_c , the pathogen becomes **endemic** and a finite fraction of the population is infected at any time.

For a **scale-free network** we have $\lambda_c = 0$, hence even viruses with a very small spreading rate λ can persist in the population.



No epidemic threshold and hubs

- ❖ Similar results also with SIR models: **vanishing threshold in heterogeneous networks**
- ❖ A direct consequence of hubs: even a weakly infectious pathogen **can** spread widely in a population through **super-spreaders**
- ❖ If the pathogen fails to infect a hub, it can die out very soon

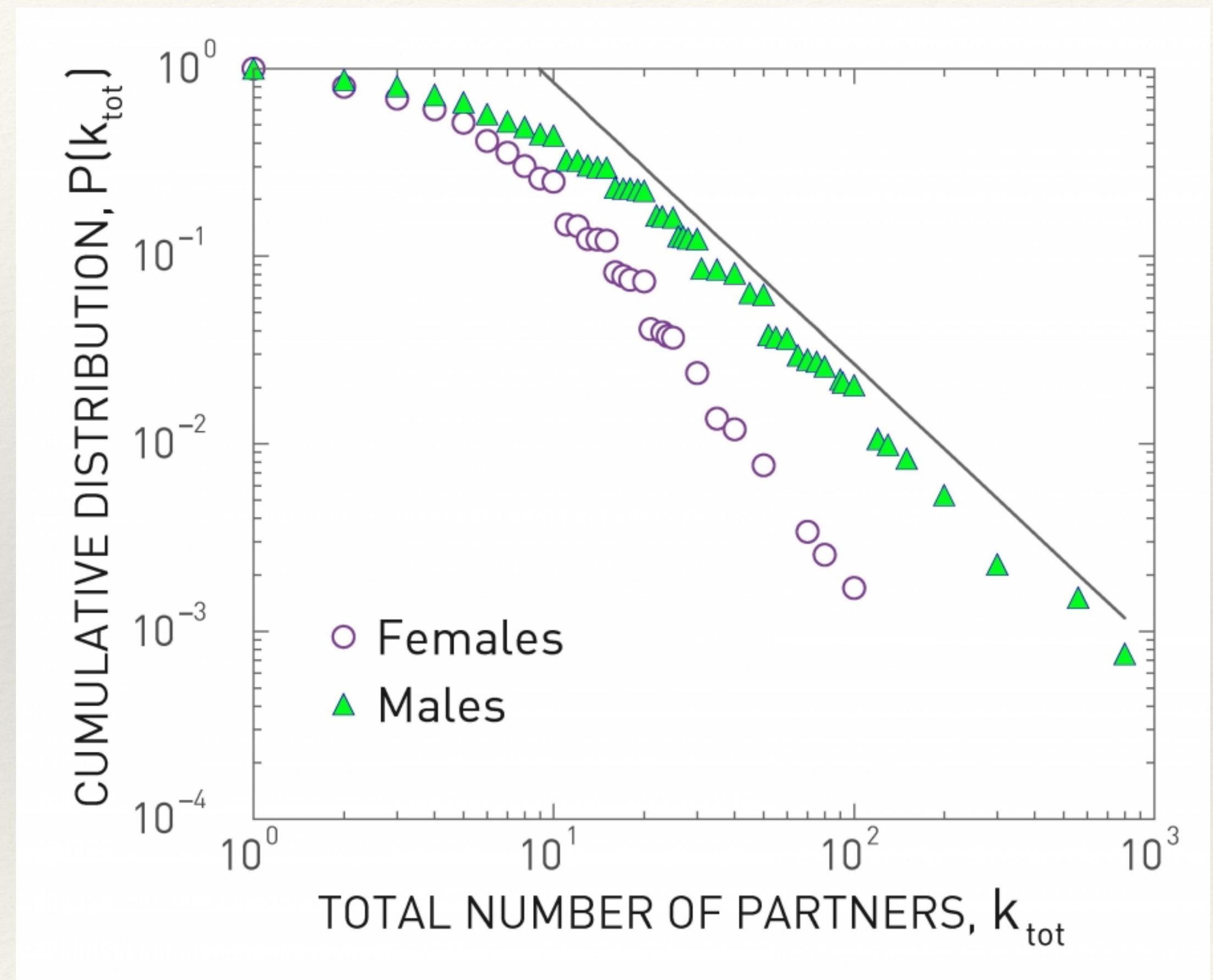
Notes on degree correlation

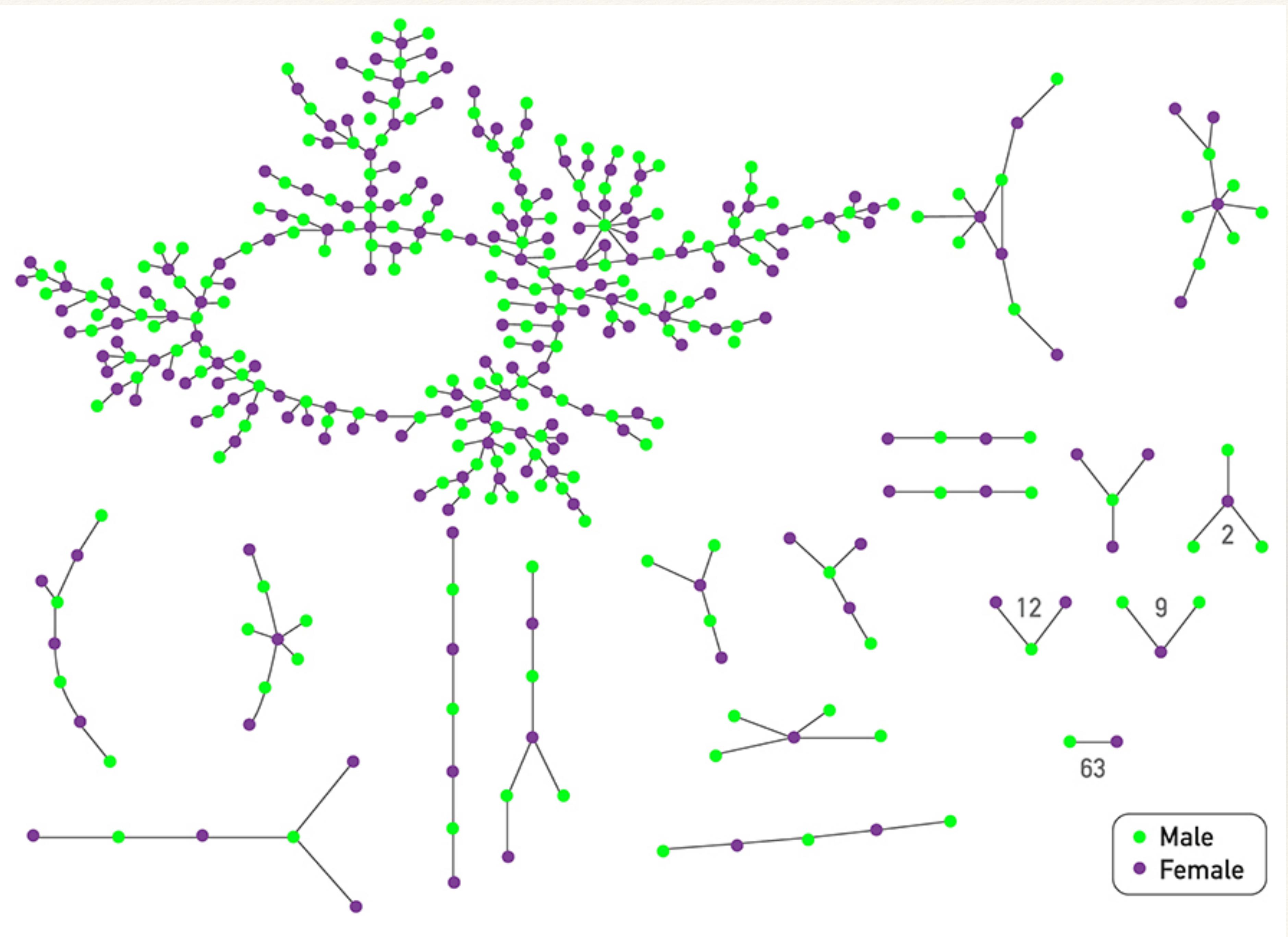
- ❖ These results have been obtained with the assumption of **no degree correlation**
- ❖ But degree correlations alter the epidemic threshold λ_c : **assortative correlations decrease λ_c and dissassortative correlations increase it**
- ❖ Despite the changes in λ_c , for the SIS model the epidemic threshold vanishes for a scale-free network with diverging second moment, whether the network is assortative, neutral or disassortative
- ❖ Given that hubs are the first to be infected in a network, **assortativity accelerates the spread of a pathogen**. In contrast **disassortativity slows the spreading process**.
- ❖ Finally, in the **SIR model assortative correlations were found to lower the prevalence but increase the average lifetime of an epidemic outbreak**

Contact networks

Sexually transmitted diseases

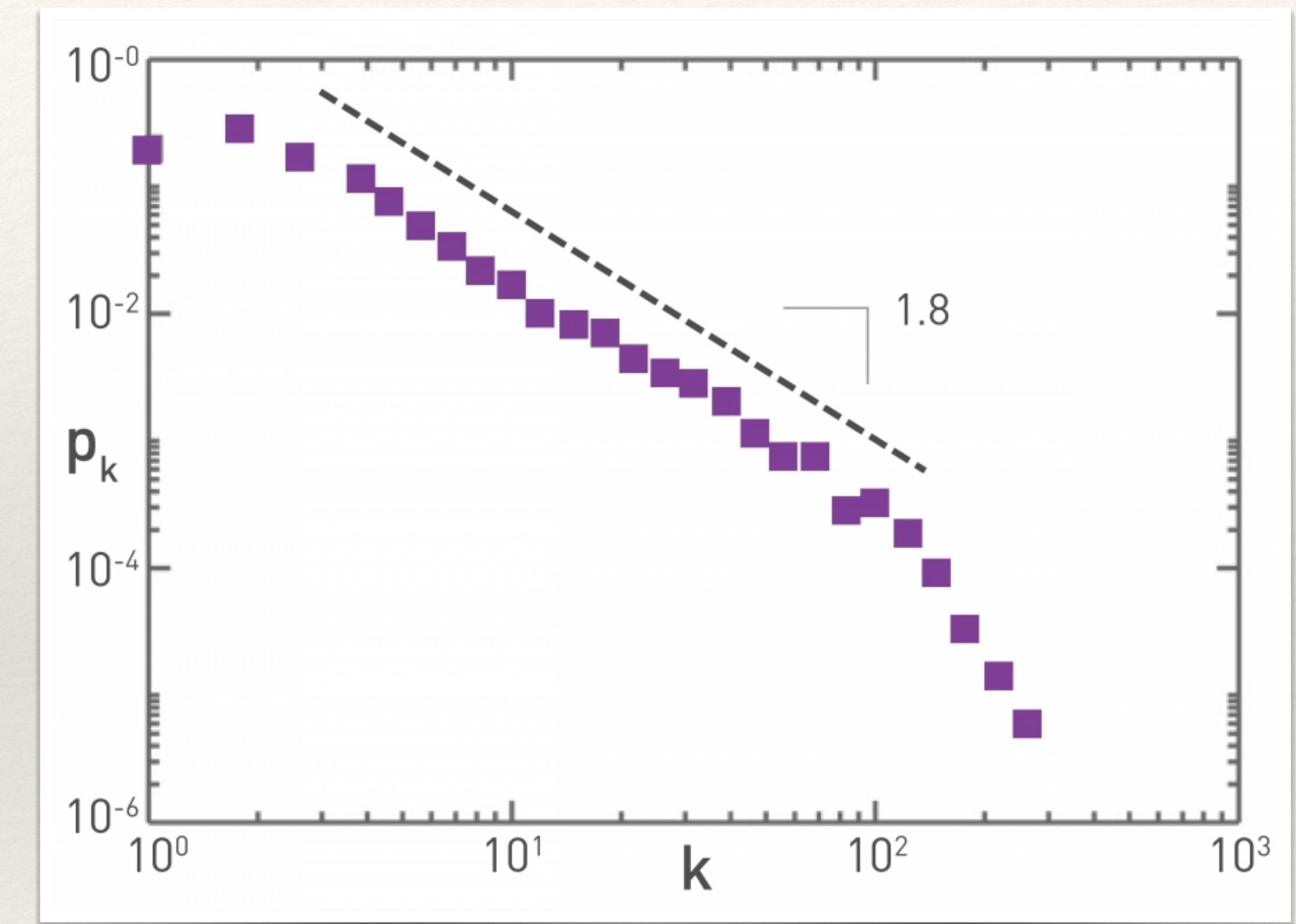
- ❖ In 1998, researchers collected information from 4,781 randomly chosen Swedes of ages 18 to 74. The participants were not asked to reveal the identity of their sexual partners, but only to estimate the number of sexual partners they had during their lifetime.
- ❖ Degree distributions are well approximated by power laws





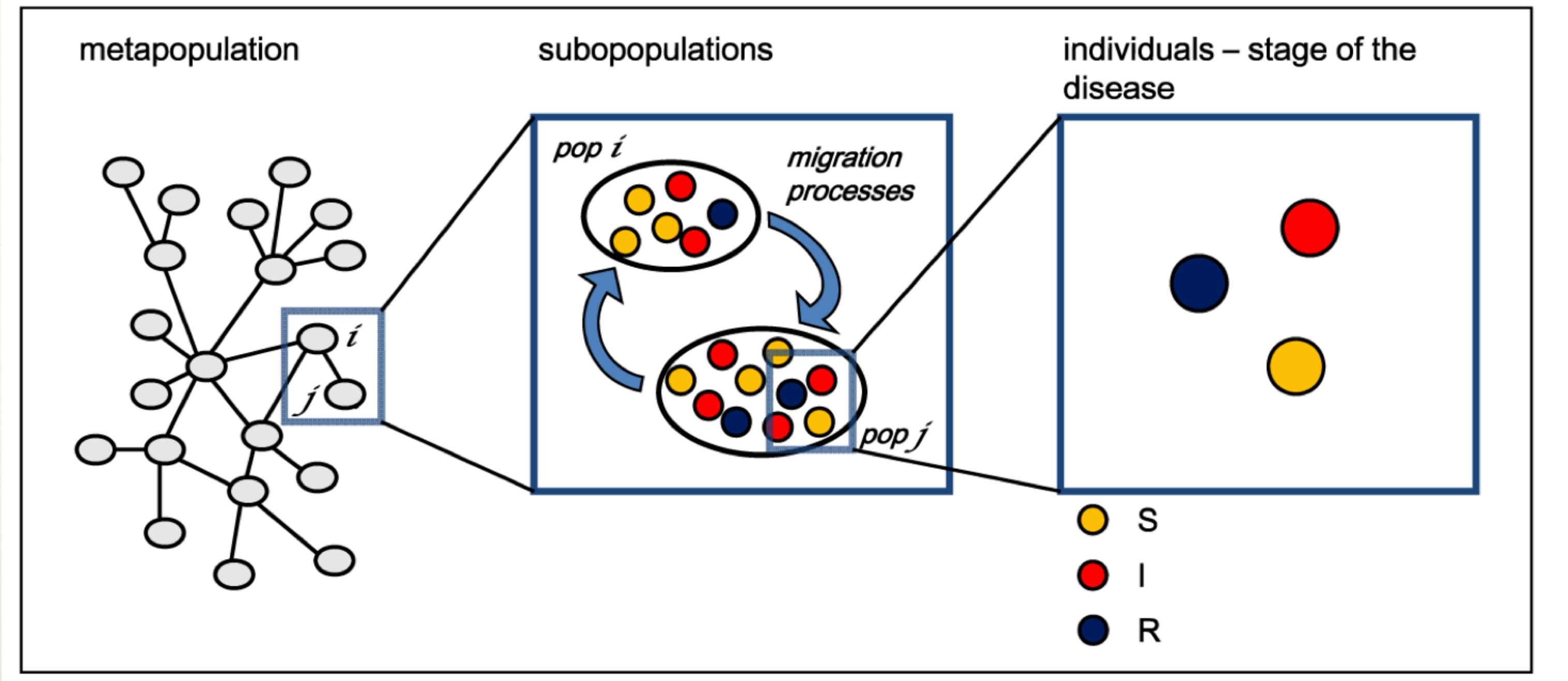
Global travel network

- ❖ Air transportation network, that connects airports with direct flights, plays a key role in modeling and predicting the spread of pathogens
- ❖ The resulting network is a **weighted graph** containing the $N=3,100$ largest airports as nodes that are connected by $L=17,182$ direct flights as links, together accounting for 99% of the worldwide traffic



T. D. Hollingsworth, N.M. Ferguson, and R.M. Anderson. Will travel restrictions control the International spread of pandemic influenza? Nature Med., 12:497-499, 2006.

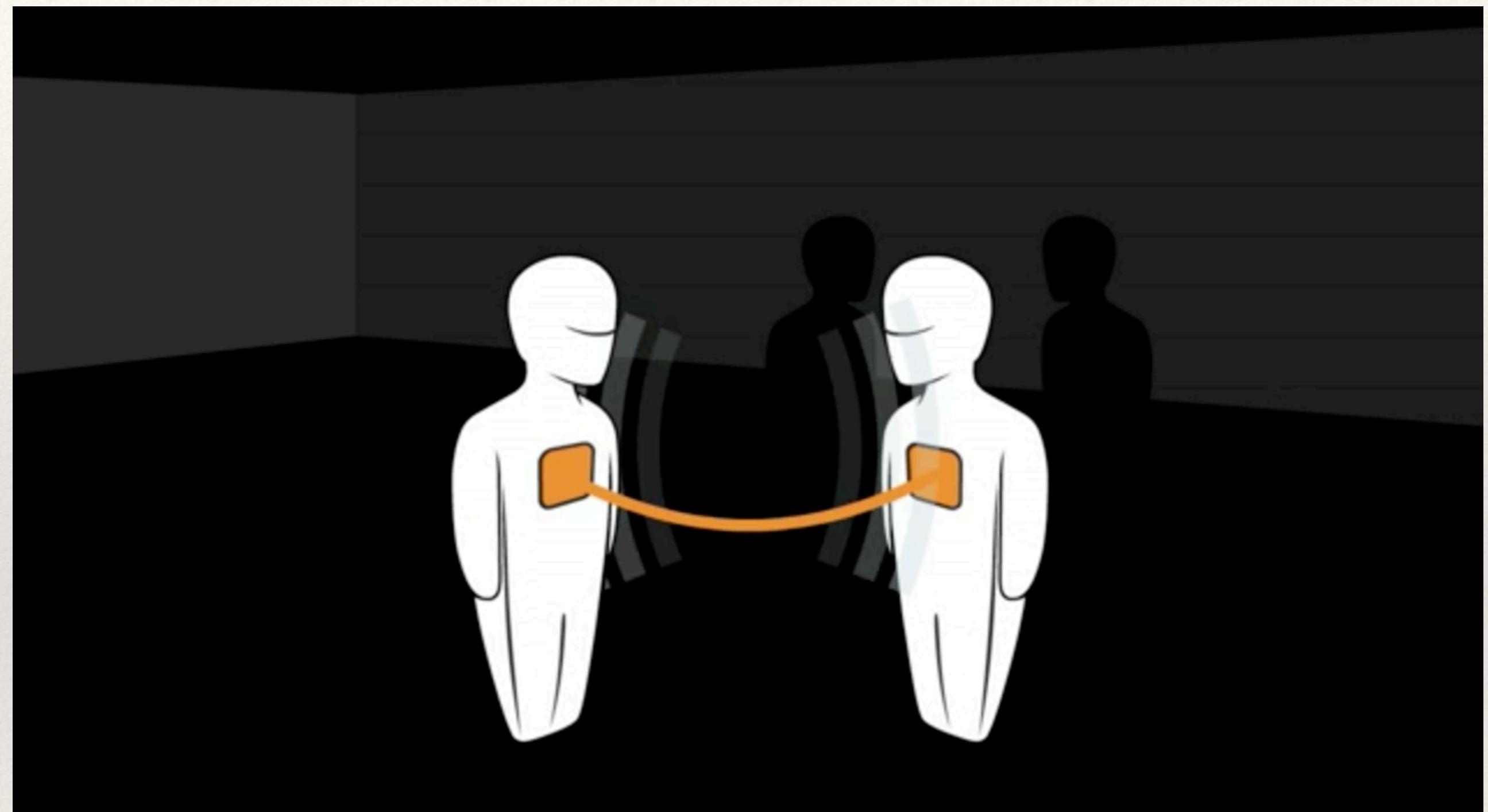
V. Colizza, A. Barrat, M. Barthélemy, A.-J. Valleron, and A. Vespignani. Modeling the world-wide spread of pandemic influenza: baseline case and containment interventions. PLoS Med, 4:e13, 2007.



- ❖ Schematic representation of a **metapopulation** model.
- ❖ The system is composed of a heterogeneous network of subpopulations, connected by migration processes.
- ❖ Individuals can move from a subpopulation to another on the network of connections among subpopulations.

Local contact patterns

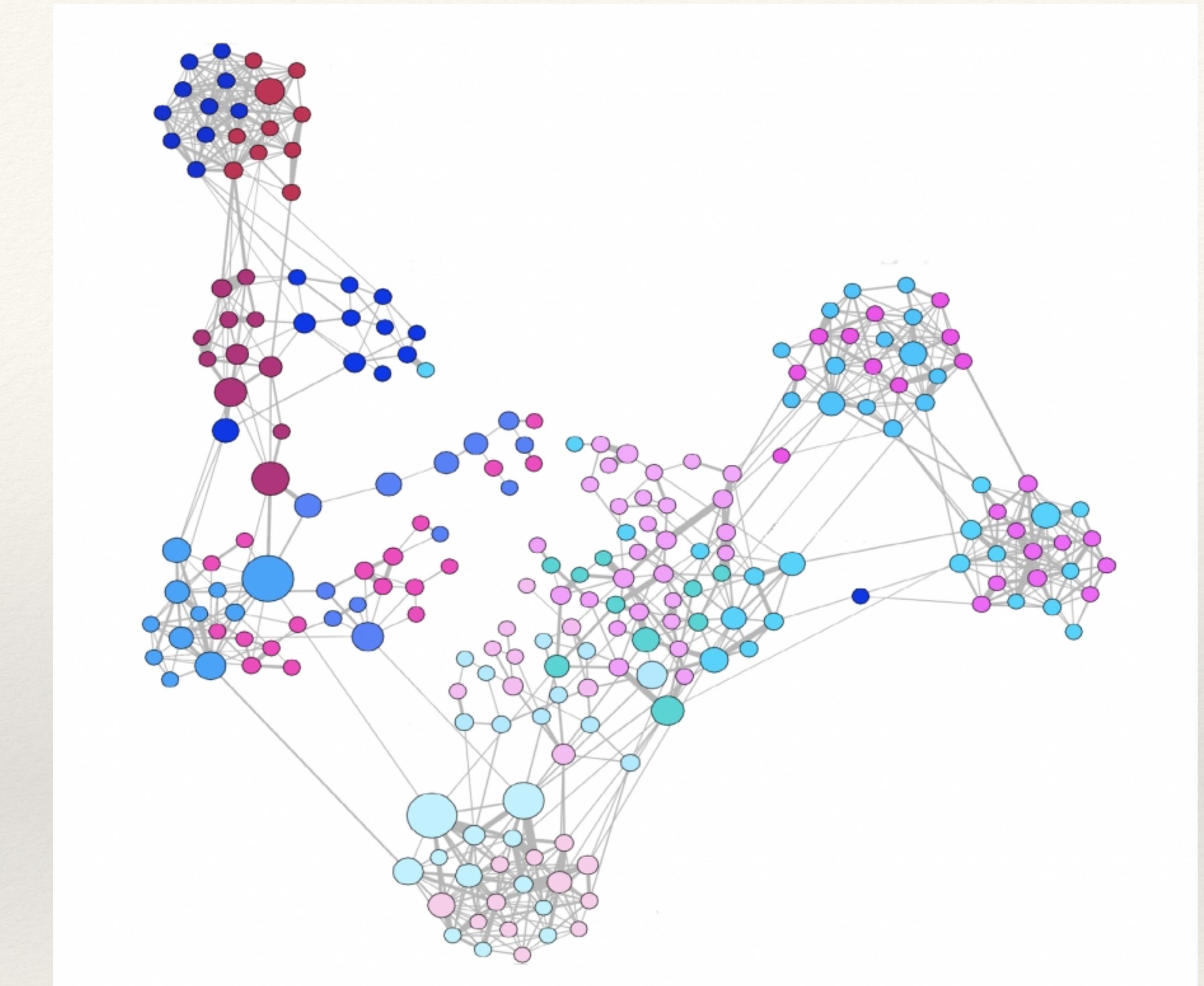
- ❖ Many airborne diseases spread thanks to face-to-face interactions. These interaction patterns can be monitored using Radio-Frequency Identification Devices (RFID)
- ❖ See **SocioPattern** project (led by Ciro Cattuto and Alain Barrat)



<http://www.sociopatterns.org>

<https://vimeo.com/6590604>

A face-to-face contact network mapped out using RFA tags, capturing interactions between 232 students and 10 teachers across 10 classes in a school



Stehlé, J., Voirin, N., Barrat, A., Cattuto, C., Isella, L., Pinton, J. F., Quaggiotto, M., Van den Broeck, W., Régis, C., Lina, B., & Vanhems, P. (2011). High-resolution measurements of face-to-face contact patterns in a primary school. *PloS one*, 6(8), e23176. <https://doi.org/10.1371/journal.pone.0023176>

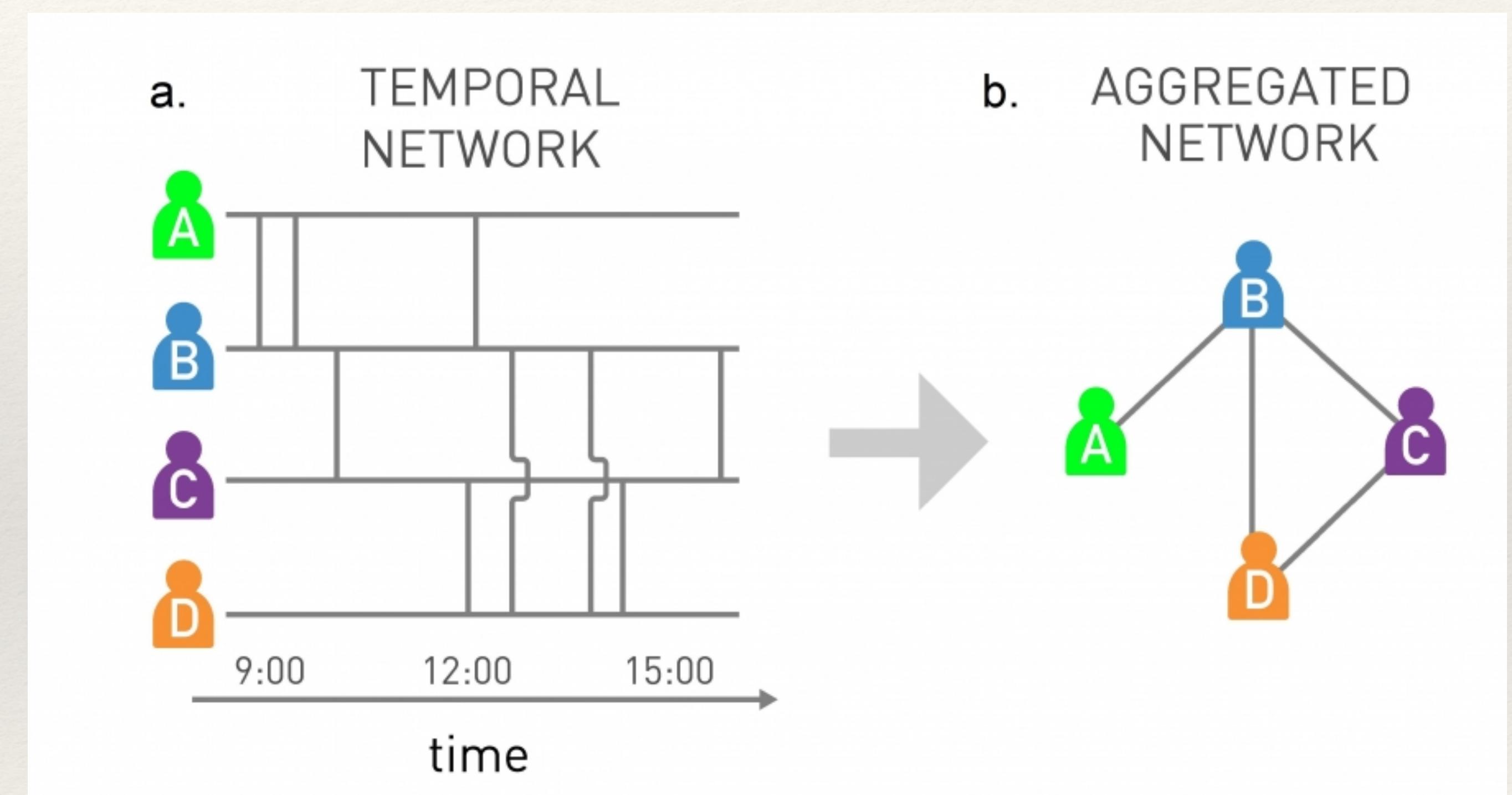
Open problems in identifying contact networks

- ❖ To correctly validate epidemics models, and also to run efficient simulations and reliable predictions, an accurate snapshot of the contact network would be of a paramount importance
- ❖ How to get an accurate model of the many ever-changing contact networks?
- ❖ When we work with meta-populations, we can use public transportation data, but what to do to get a picture of finer-grained pictures?
 - ❖ ethical concerns

Temporal Networks

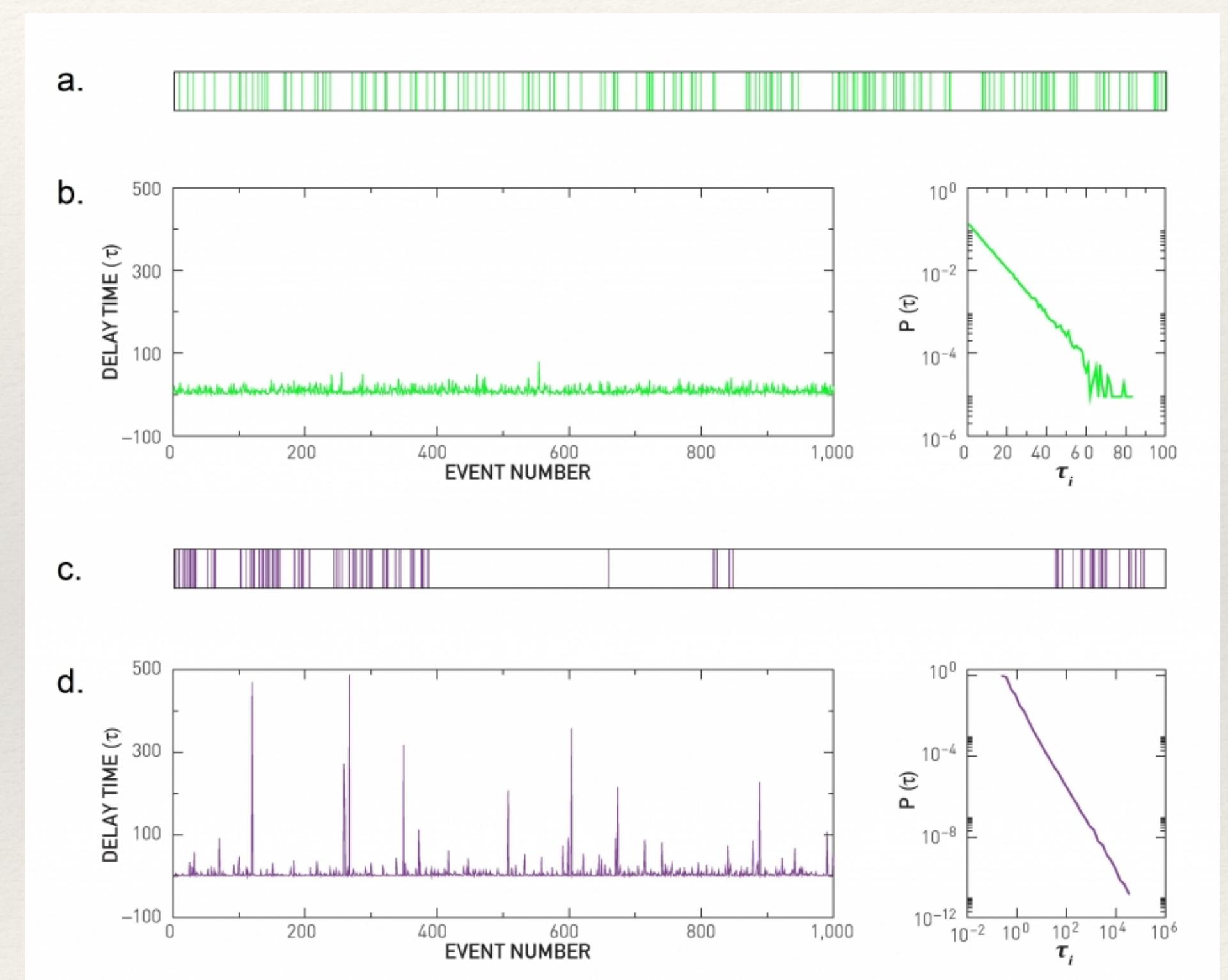
Temporal vs Aggregated Networks

- ❖ **Temporal Network:** Most interactions in a network are not continuous, but have a finite duration. We must therefore view the underlying networks as temporal networks.
- ❖ The timeline of the interactions between four individuals. Each vertical line marks the moment when two individuals come into contact with each other. If A is the first to be infected, the pathogen can spread from A to B and then to C, eventually reaching D. If, however, D is the first to be infected, the disease can reach C and B, but not A. This is because there is a temporal path from A to D.
- ❖ **Aggregated Network:** The network obtained by merging the temporal interactions shown in (a). If we only have access to this aggregated representation, the pathogen can reach all individuals, independent of its starting point.



Bursty Contact Patterns

- ❖ If the pattern of activity of an individual is random, the times follow a Poisson process, which assumes that in any moment an event takes place with the same probability q . The horizontal axis denotes time and each vertical line corresponds to an event whose timing is chosen at random.
- ❖ The absence of long delays is visible if we show the inter-event times τ_i for 1,000 consecutive random events. The height of each vertical line corresponds to the gaps seen in (a).
- ❖ The probability of finding exactly n events within a fixed time interval follows the Poisson distribution predicting that the inter-event time distribution follows shown on a log-linear plot.
- ❖ The succession of events for a temporal pattern whose interevent times follow a power-law distribution. While most events follow each other closely, forming bursts of activity, there are a few exceptionally long interevent times, corresponding to long gaps in the contact pattern. The time sequence is not as uniform as in (a), but has a bursty character.
- ❖ The waiting time τ_i of 1,000 consecutive events, where the mean event time is chosen to coincide with the mean event time of the Poisson process shown in (b). The large spikes correspond to exceptionally long delays.
- ❖ The delay time distribution $P(\tau_i) \sim \tau_i^{-2}$ for the bursty process shown in (d) and (e). After [35].
- ❖ Bursty interactions are observed in a number of contact processes of relevance for epidemic phenomena, from email communications to call patterns and sexual contacts. To be specific, power law interevent times increase the characteristic time τ . The number of infected individuals decays slower than predicted by a random contact pattern.



Immunization

Immunization strategies

- ❖ Immunization strategies specify how vaccines, treatments or drugs are distributed in the population.
- ❖ Immunization strategies are guided by monitoring the pathogen's spreading rate λ
- ❖ Random immunization vs selective immunization

Random immunization

- ❖ Let us assume that the pathogen follows the SIS model
- ❖ Let us also consider the situation when a **randomly selected g fraction of individuals** are immunized in a population
- ❖ Only the remaining $(1-g)$ fraction of the nodes can contact and spread the disease
- ❖ Consequently, the **effective degree of each susceptible** node changes from $\langle k \rangle$ to $\langle k \rangle(1 - g)$
- ❖ The **spreading rate** $\lambda = \beta/\mu$ decreases to $\lambda' = \lambda(1 - g)$

Random immunization in random networks

- ❖ If the pathogen spreads on a random network, for a sufficiently high g_c the spreading rate λ' could fall **below** the epidemic threshold λ_c

$$\lambda_c = \lambda(1 - g_c) = (1 - g_c) \frac{\beta}{\mu} \approx \frac{1}{\langle k \rangle}$$

obtaining

$$g_c \approx 1 - \frac{\mu}{\beta \langle k \rangle}$$

- ❖ If we have a fraction of immunized individuals above g_c , our spreading rate will be under the epidemic threshold: the pathogen will die out
- ❖ This fraction is **very high!**
- ❖ Hence, it is important to vaccinate massively against the "normal" flu, or to use appropriate protections for casual sexual intercourses!

Random immunization in heterogeneous networks

- ❖ If the pathogen spreads on a network with $\kappa \gg 1$, random immunization will allow us to determine the critical g_c :

$$\lambda_c = \lambda(1 - g_c) = (1 - g_c) \frac{\beta}{\mu} \approx \frac{\langle k \rangle}{\langle k^2 \rangle}$$

obtaining

$$g_c \approx 1 - \frac{\mu}{\beta} \frac{\langle k \rangle}{\langle k^2 \rangle}; \text{ and in heterogeneous networks } \langle k^2 \rangle \gg \langle k \rangle^2$$

- ❖ In scale-free networks, $\langle k^2 \rangle \rightarrow \infty$, hence $g_c \rightarrow 1$: **we need to immunize virtually all nodes to stop the epidemics**
- ❖ For example, measles require a *herding immunity* of 80%-100% of the population to eradicate the pathogen

Selective immunization

- ❖ Let's put it simply: random immunization in heterogeneous networks is inefficient because of the vanishing epidemic threshold
- ❖ We must find a way to increase the epidemic threshold: reduce the variance $\langle k^2 \rangle$ of the underlying contact network
- ❖ Hubs are to blame: we need to immunize them (all nodes whose degree is exceeding some preselected k'_{\max})

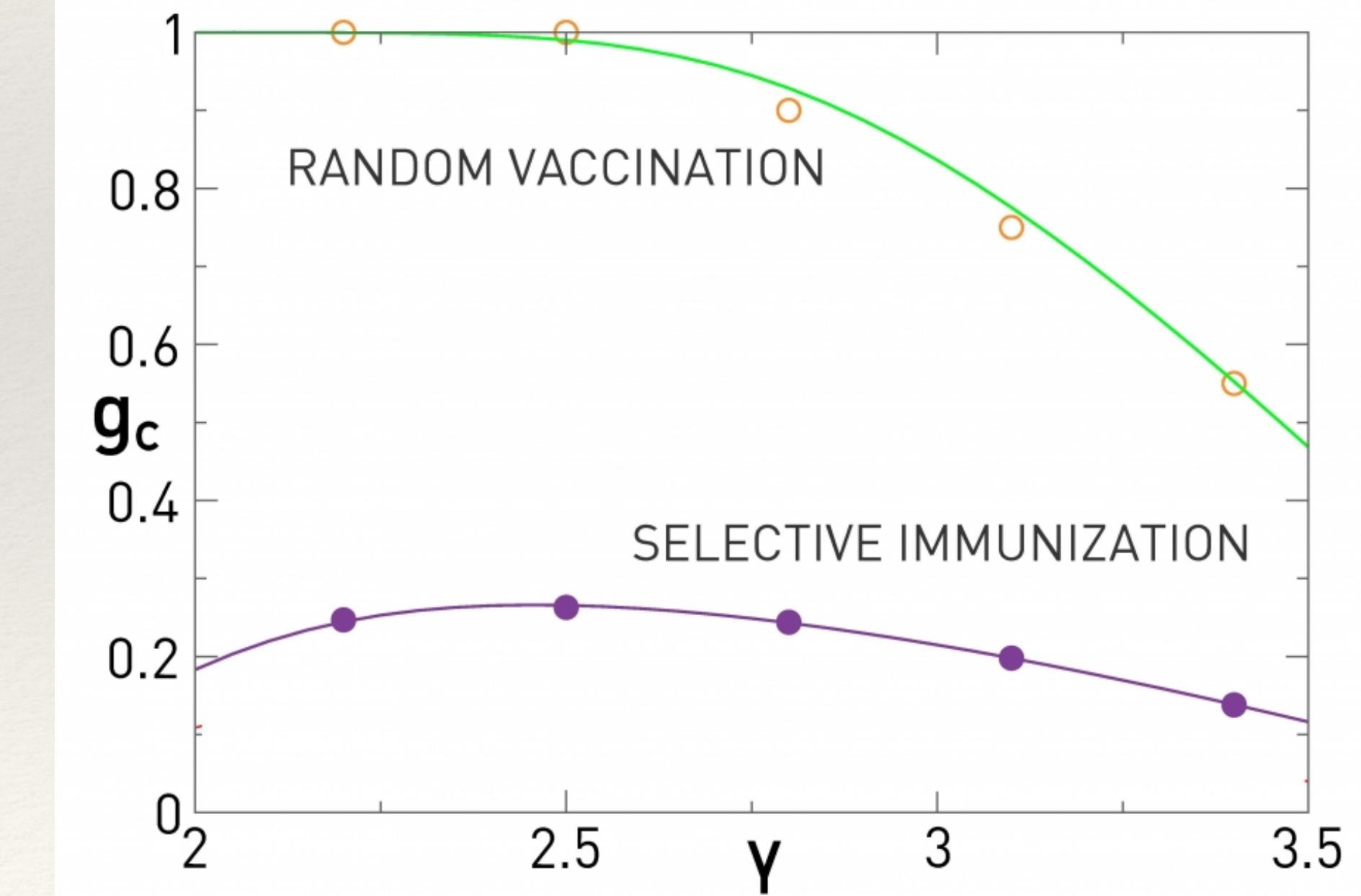
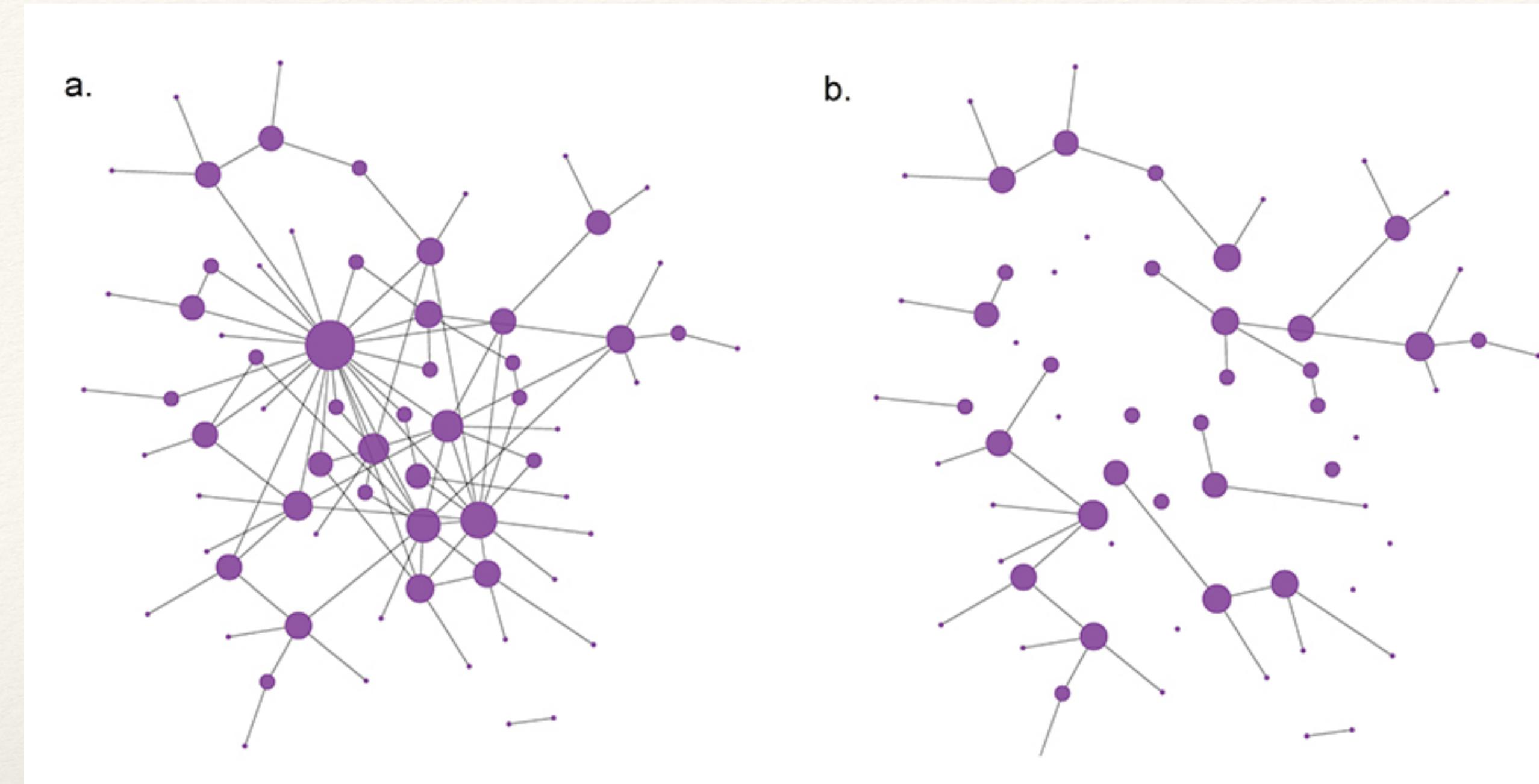
Robustness: Heterogeneous networks are resistant against random node and link failures, but are vulnerable to attacks.

Analogously, random immunization is unable to eradicate a disease, but targeting the hubs, can restore a finite critical threshold, helping us eradicate the disease.

The critical immunization threshold g_c in function of the degree exponent γ of the underlying contact network whose degree distribution follows a power law $p_k \propto k^{-\gamma}$.

Z. Dezső and A-L. Barabási. Halting viruses in scale-free networks. Physical Review E, 65:055103, 2002.

R. Pastor-Satorras and A. Vespignani. Immunization of complex networks. Physical Review E, 65:036104, 2002.



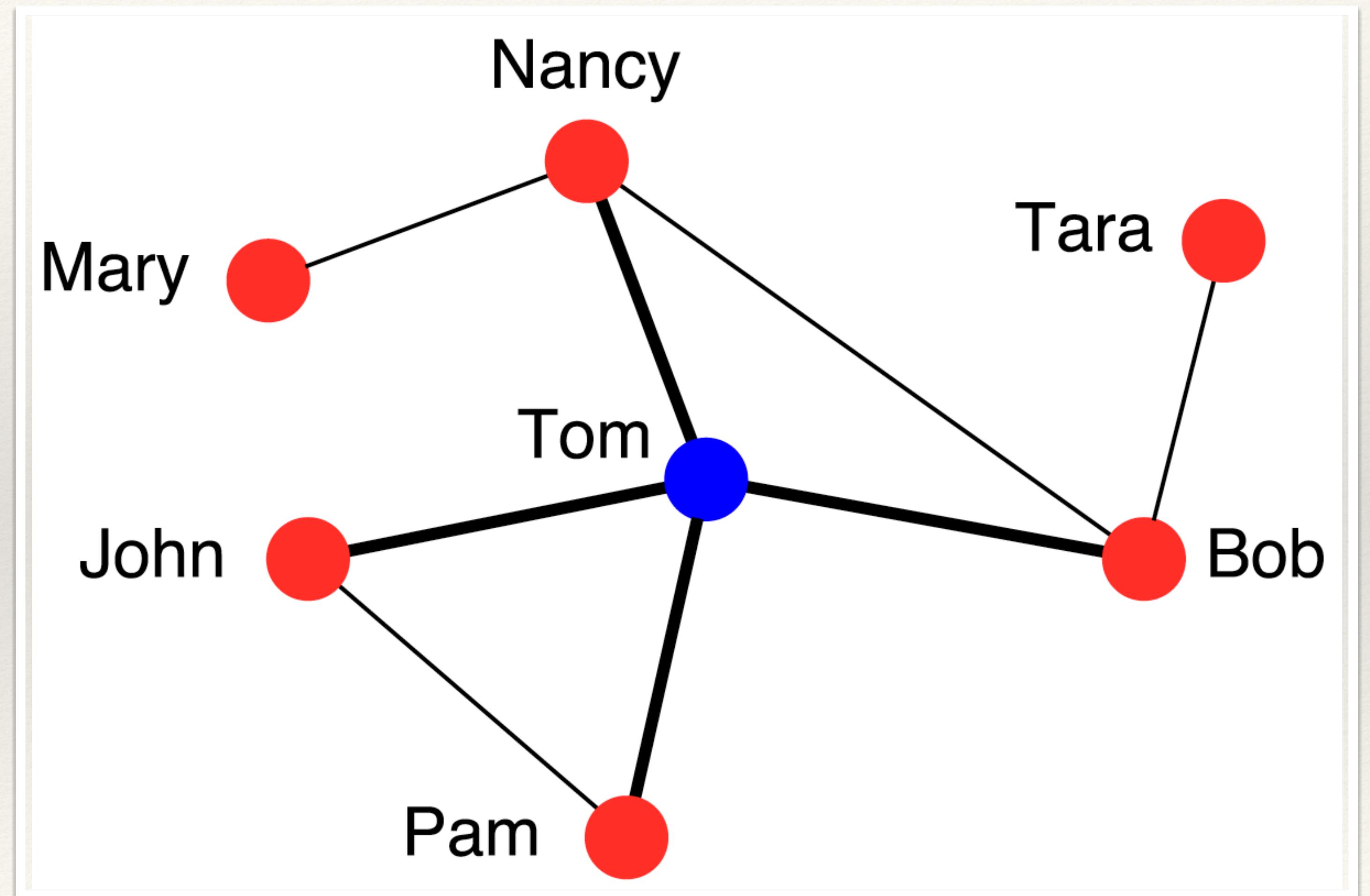
From: Albert-László Barabási, Network Science [ns3]

How can we identify hubs?

- ❖ We can exploit the **friendship paradox**:
 - ❖ Group 0: a p fraction of nodes randomly chosen
 - ❖ Group 1: Select randomly a link for each node in Group 0. We stop at the set of nodes to which these links connect to.
 - ❖ Immunize the Group 1 individuals.

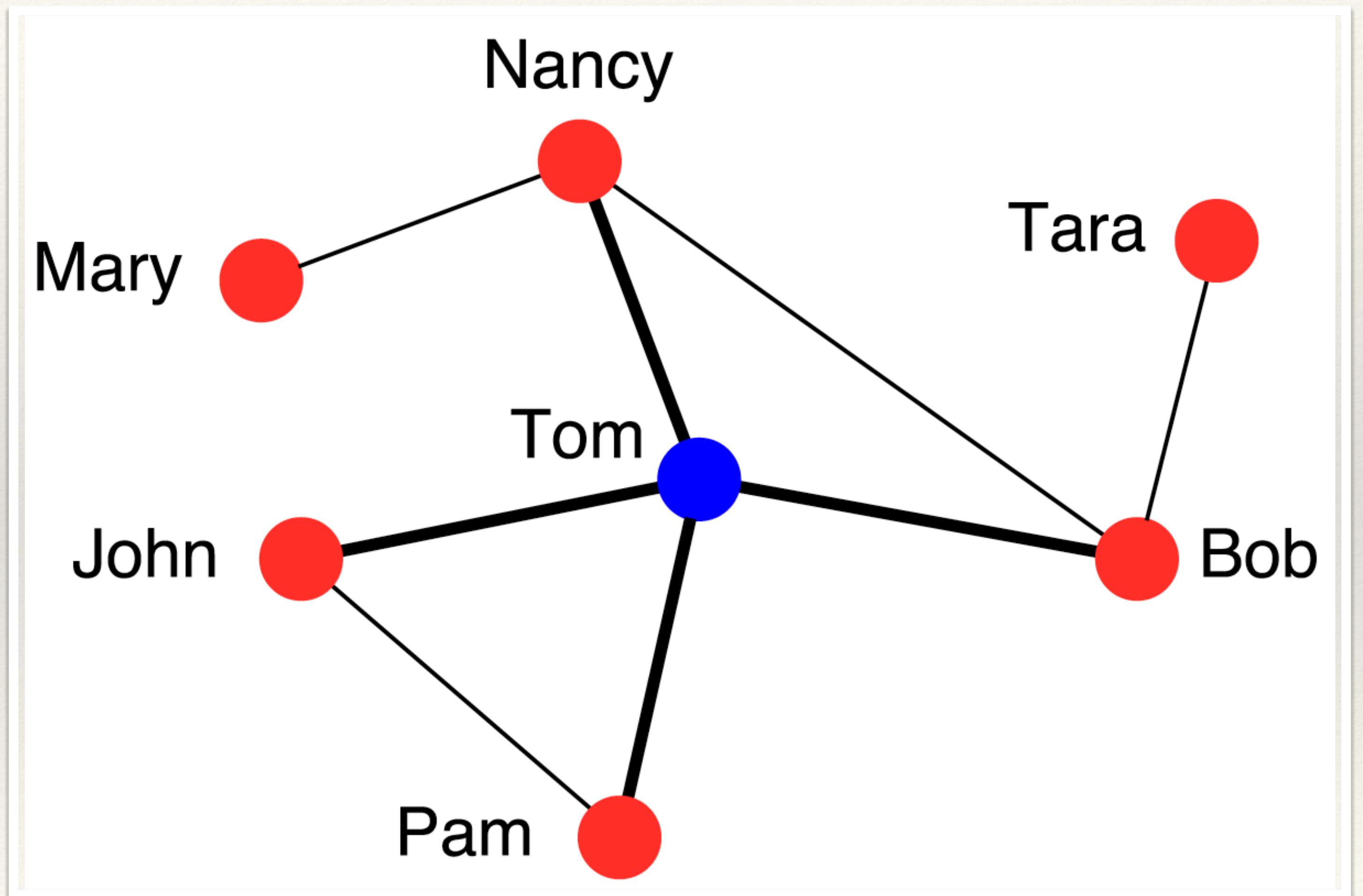
Friendship paradox

- ❖ By choosing nodes at random, Tom has the same chance to be picked as everybody else
- ❖ By choosing links at random, Tom has a higher chance to be picked than everybody else
- ❖ By following links, the chance to hit a hub increases



Friendship paradox

- ❖ Average degree of a node = 2.29
- ❖ Average degree of the neighbors of a node = $2.83 > 2.29$
- ❖ Our friends have more friends than we do, on average (**friendship paradox**)



Predictions

Key questions

- ❖ The emergence of any new pathogen raises several key questions:
 - ❖ Where did the pathogen originate?
 - ❖ Where do we expect new cases?
 - ❖ When will the epidemic arrive at various densely populated areas?
 - ❖ How many infections are to be expected?
 - ❖ What can we do to slow its spread?
 - ❖ How can we eradicate it?

- ❖ These questions are addressed using powerful epidemic simulators that consider as input demographic, mobility-related, and epidemiological data.
- ❖ The algorithms behind these tools range from **stochastic meta-population models** to **agent-based computer simulations** that capture the behavior and the **interactions of millions of individuals**.

Real time forecasts

- ❖ **Epidemic forecast** aims to foresee the real time spread of a pathogen, predicting the number of infected individuals expected each week in each major city.
- ❖ The first successful real time pandemic forecast based on network science relied on the **Global Epidemic and Mobility (GLEAM)** computational model, a stochastic framework that uses as input high-resolution data on worldwide human demography and mobility.

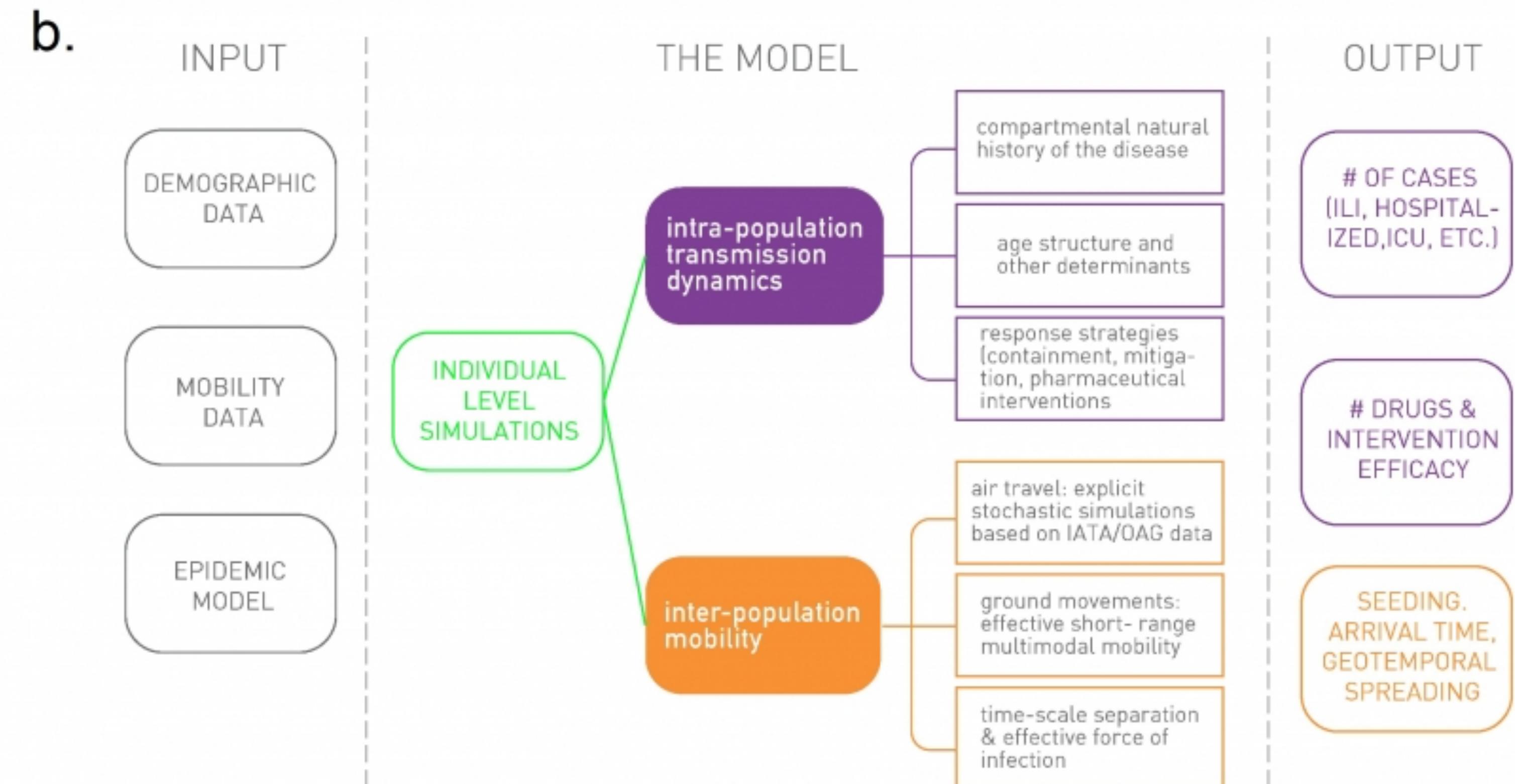
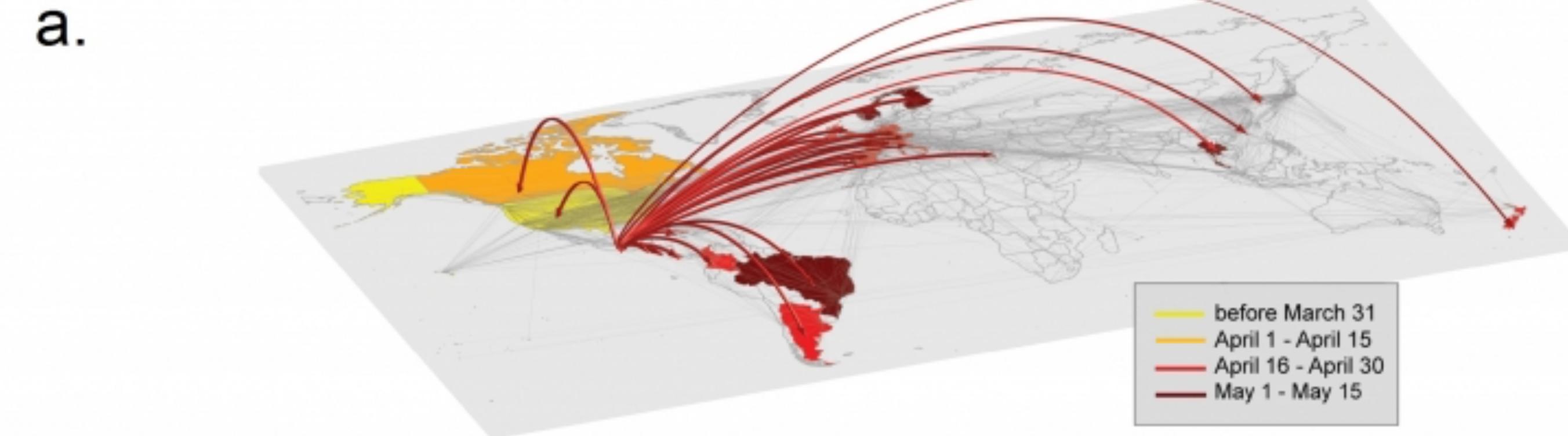
I. M. Hall, R. Gani, H.E. Hughes, and S. Leach. Real-time epidemic forecasting for pandemic influenza. *Epidemiol Infect.*, 135:372-385, 2007

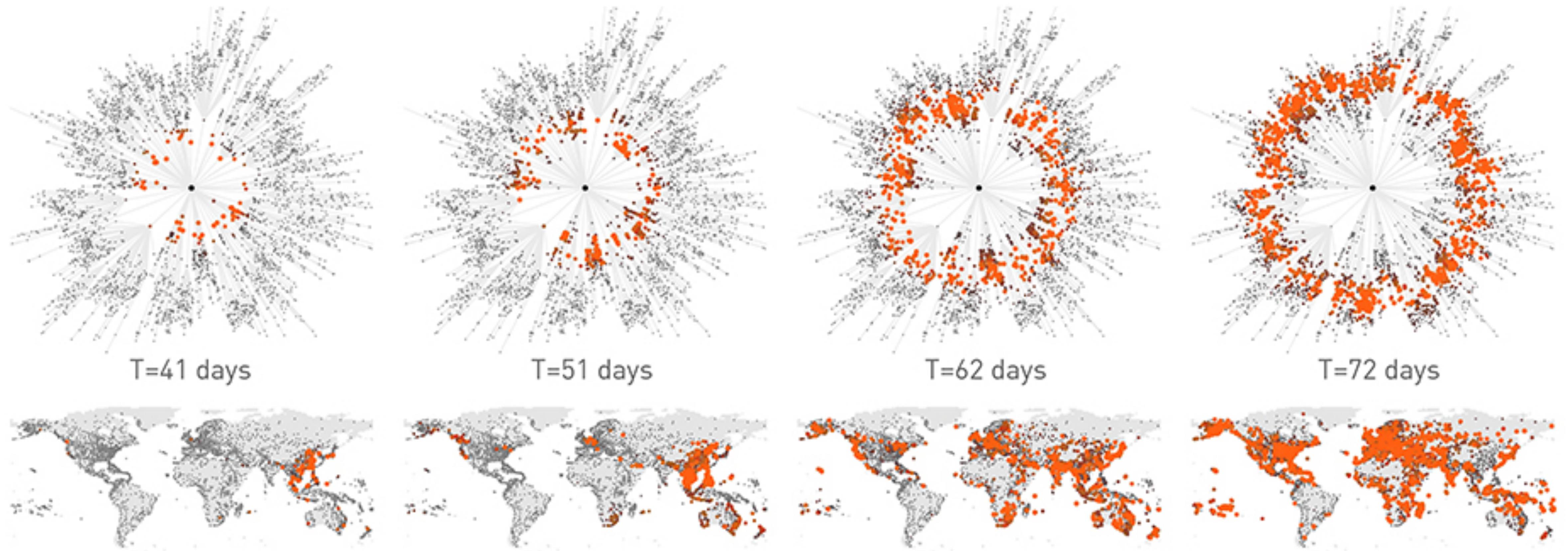
M. Tizzoni, P. Bajardi, C. Poletto, J. J. Ramasco, D. Balcan, B. Gonçalves, N. Perra, V. Colizza, and A. Vespignani. Real-time numerical forecast of global epidemic spreading: case study of 2009 A/H1N1pdm. *BMC Medicine*, 10:165, 2012.

- ❖ GLEAM employs a network-based computational model:
 - ❖ It maps each geographic location into the nodes of a network.
 - ❖ Transport between these nodes, representing the links, are provided by global transportation data, like airline schedules
 - ❖ It estimates the epidemic parameters, like the transmission rate or reproduction number, using a network-based approach: It relies on **chronological data** that captures the worldwide spread of the pandemic, rather than medical reports.

(a) The spread of the H1N1 virus during the early stage of the 2009 outbreak. The arrows represent the **arrival of the first infections** in previously unaffected countries. The **color code** indicates the **time of the virus' arrival**.

(b) The flowchart of GLEAM computational model, used to predict the real-time spread of pathogens like H1N1 or Ebola. The left column (Input) represents the **input databases, capturing demographic, mobility and epidemiological information**. The center column (model) describes the **network-based dynamic processes** that are modeled at each time step. The right column (Output) offers **examples of quantities the model can predict**.





The spread of a pandemic with an initial outbreak in Hong Kong. Regions with a large number of infections are shown as red nodes. Each panel compares the state of the system in the conventional geographic representation (bottom) with the **effective distance** representation (top). The complex spatial pattern observed in the geographic representation becomes a circular wave that moves outwards at constant speed in the effective distance representation

Surveillance, and more



Dove va l'influenza, diccelo tu.

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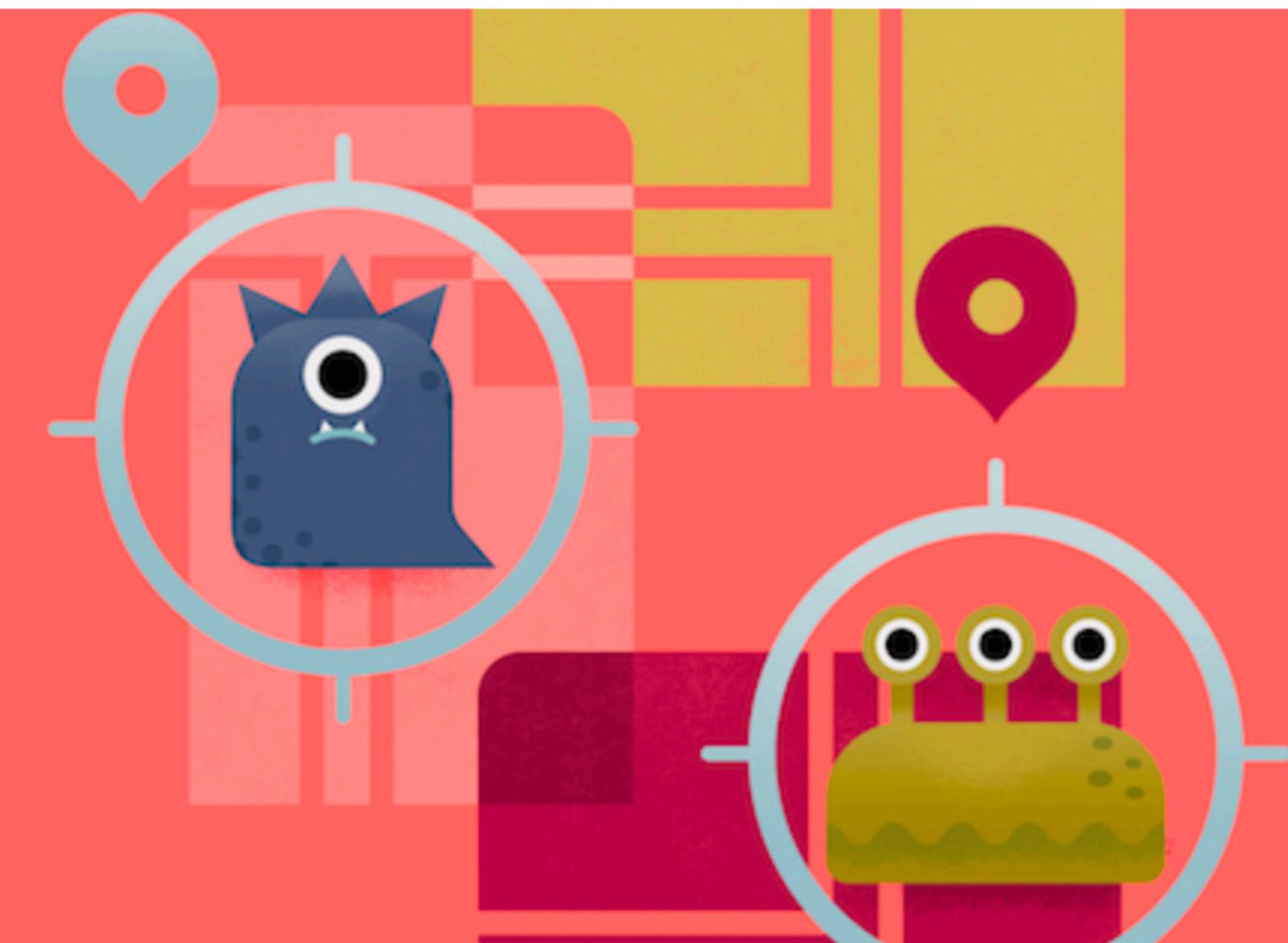
Raccogliamo insieme le informazioni sull'epidemia di corona virus.
Riportaci il tuo stato di salute in tempo reale.

[Partecipa!](#)

Ultime notizie

Ultime notizie sul coronavirus, 11 marzo 2020

Dall'Organizzazione Mondiale della Sanità, è appena giunta la notizia che l'epidemia di corona virus è stata dichiarata pandemia
[Continua a leggere](#)

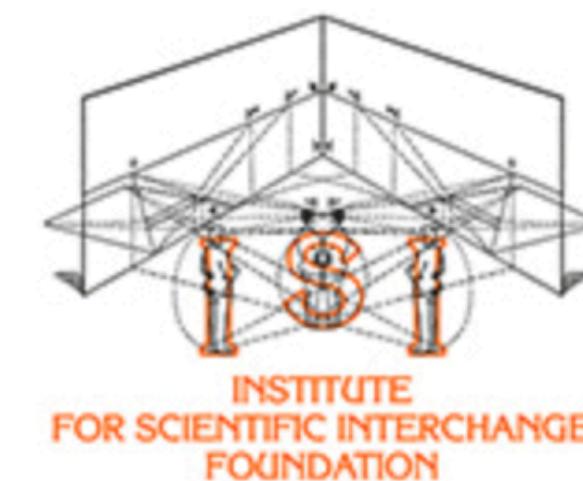


Cos'è Influweb

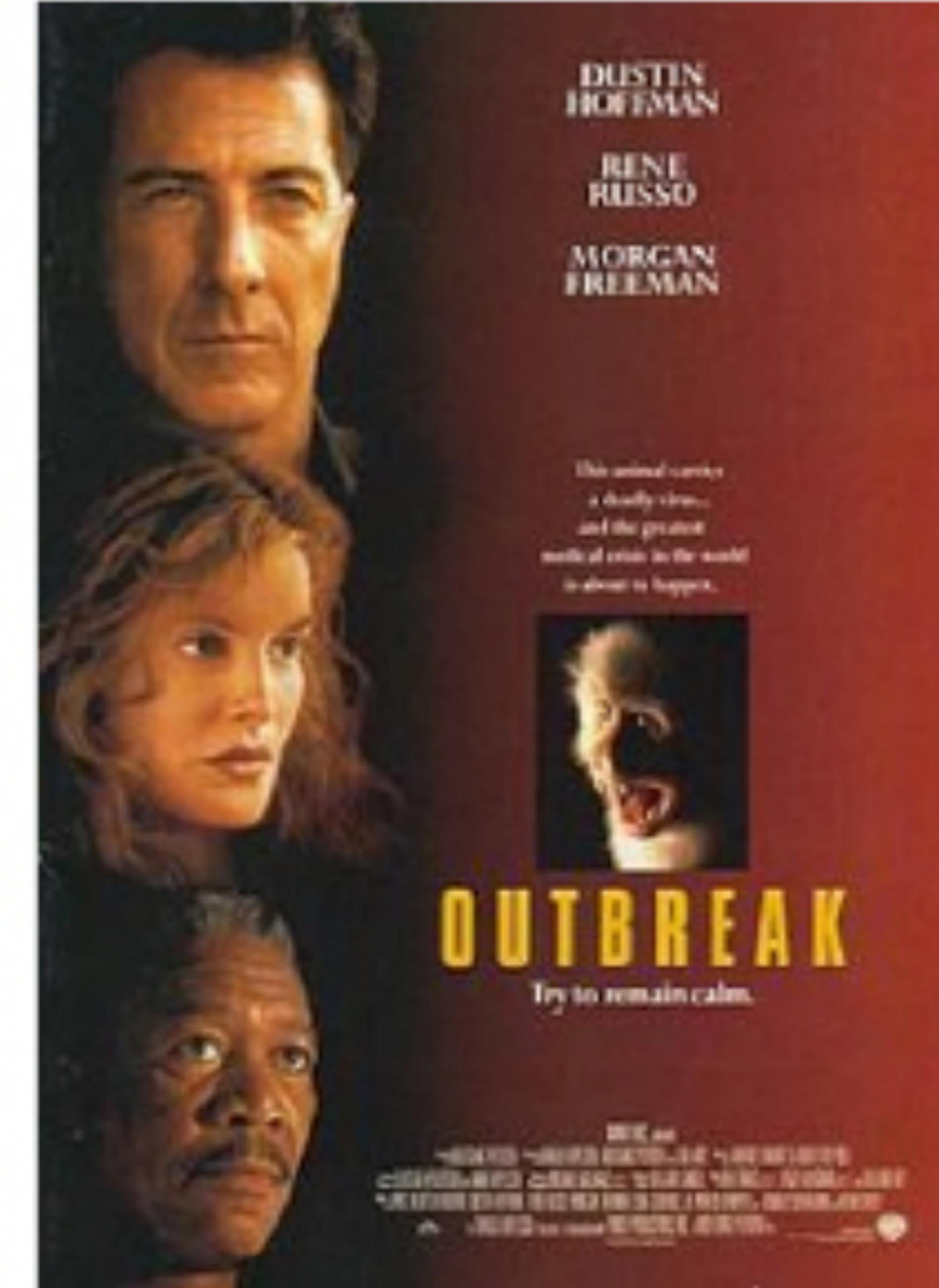
Influweb è un [progetto](#) che raccoglie informazioni sullo stato di salute delle persone in Italia. Si basa sui report che migliaia di volontari inviano ogni settimana.

A partire dal 1 Aprile, Influweb si trasforma in "[Influweb](#)

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Like



Other insights

- ❖ interview of Dr. **Daniela Paolotti** on Digital Epidemiology and InfluWeb:
<https://www.mixcloud.com/radiobandalarga/eulero-17th-january-2020/>
- ❖ EPFL epidemiologist **Marcel Salathé** gave a lecture on the 2020 spread of the COVID-19 new Coronavirus:
<https://www.youtube.com/watch?v=3RQBtA4dK9s>
- ❖ interview of **Carlo Blengino**, on science, law, ethics, rights, health and app Immuni:
<https://www.mixcloud.com/radiobandalarga/eulero-1st-may-2020/>