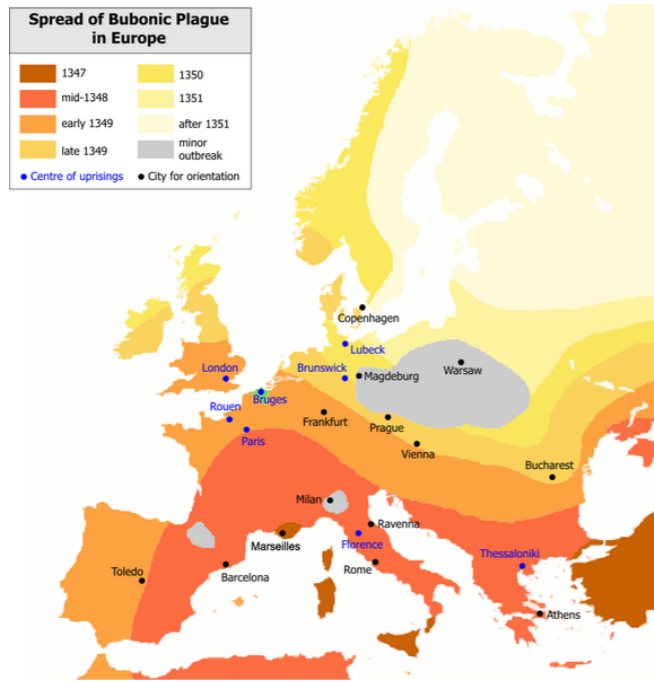


# DIFFUSION ON NETWORKS

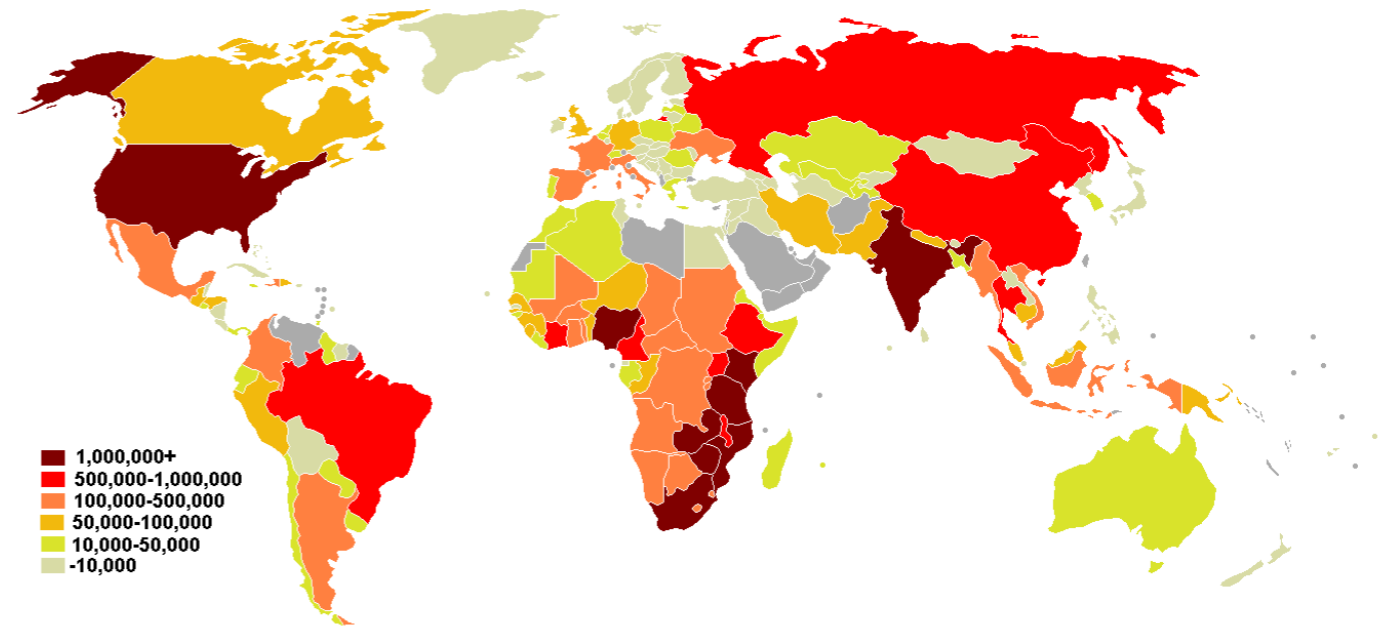
Spreading processes  
Dynamic ON networks

# Spreading processes

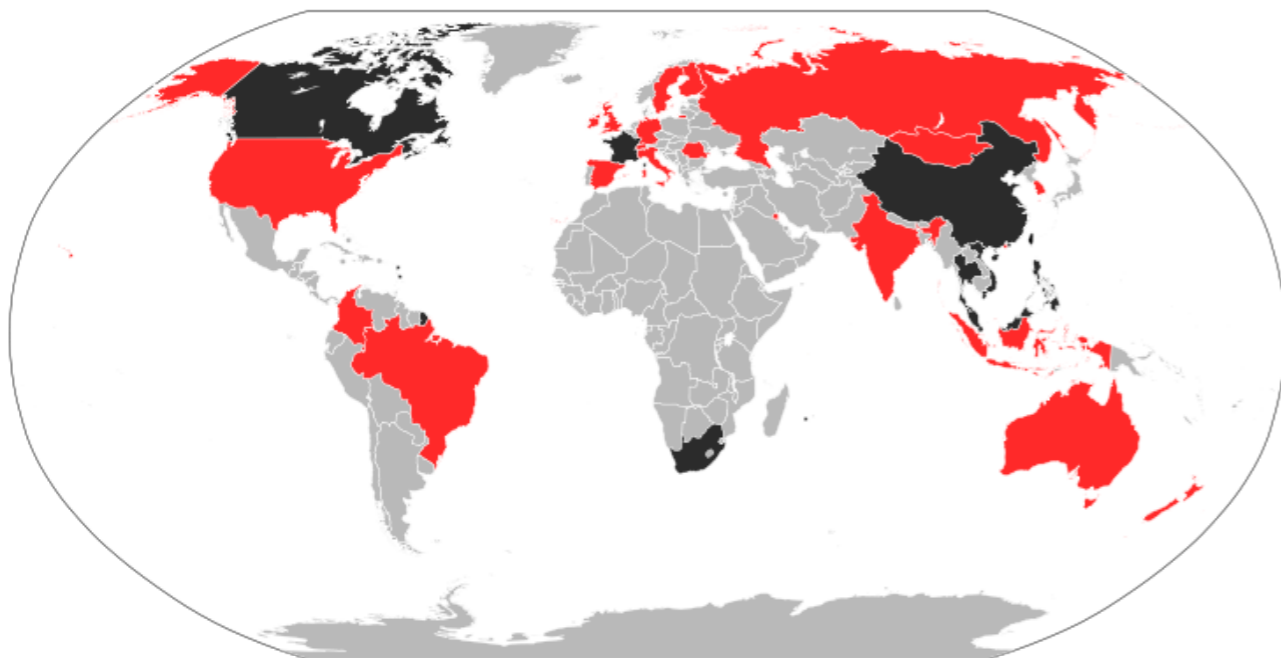
## Biological epidemic spreading



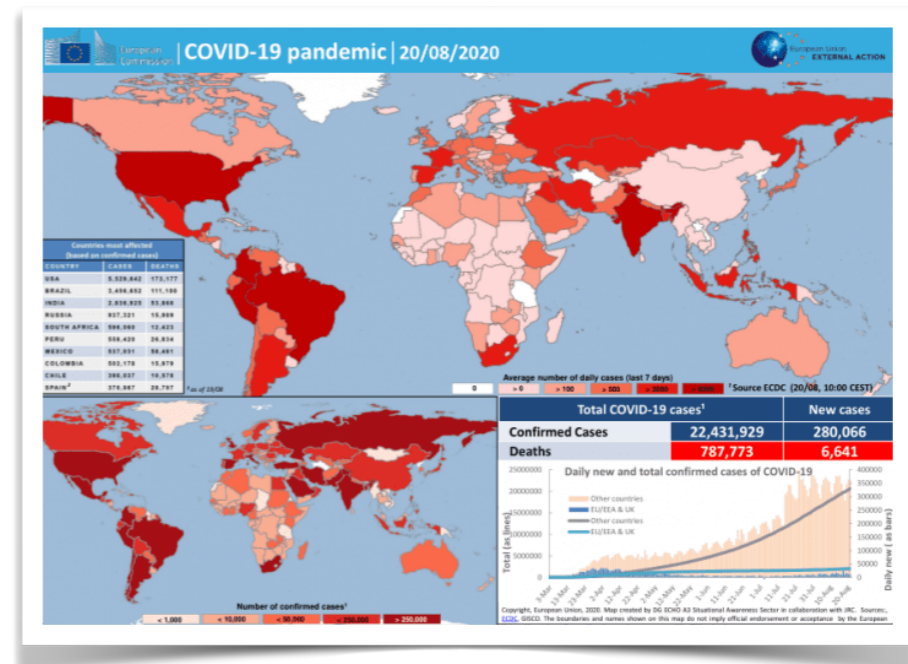
The great plague (14th century)



HIV (2008)



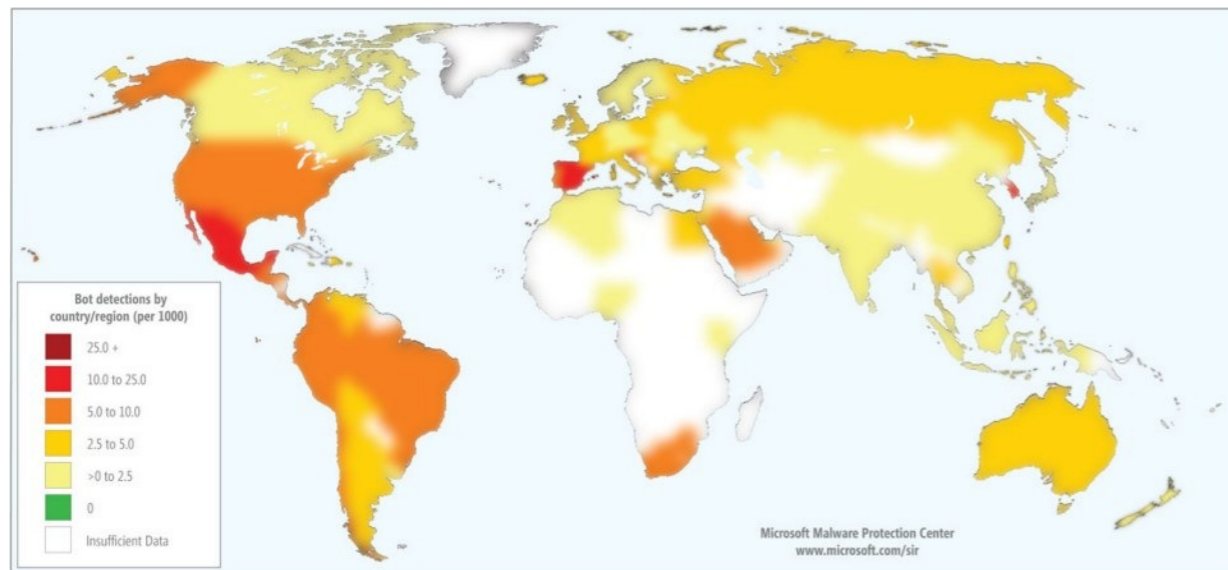
SARS (2008)



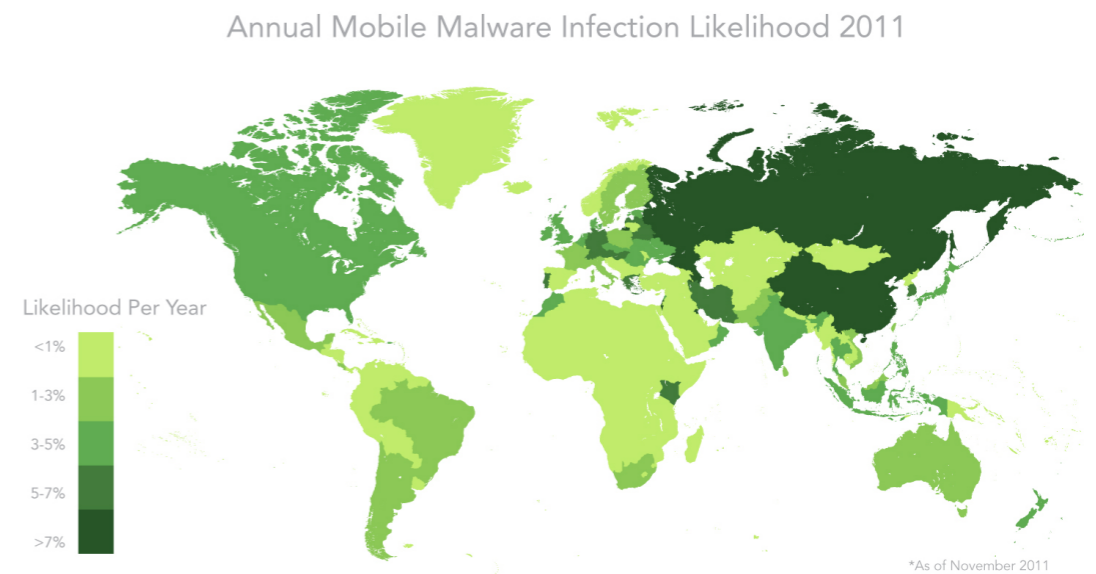
Covid-19

# Spreading processes

## Malware spreading



Botnet infections (2010)



Mobile malware (2011)

# Spreading processes

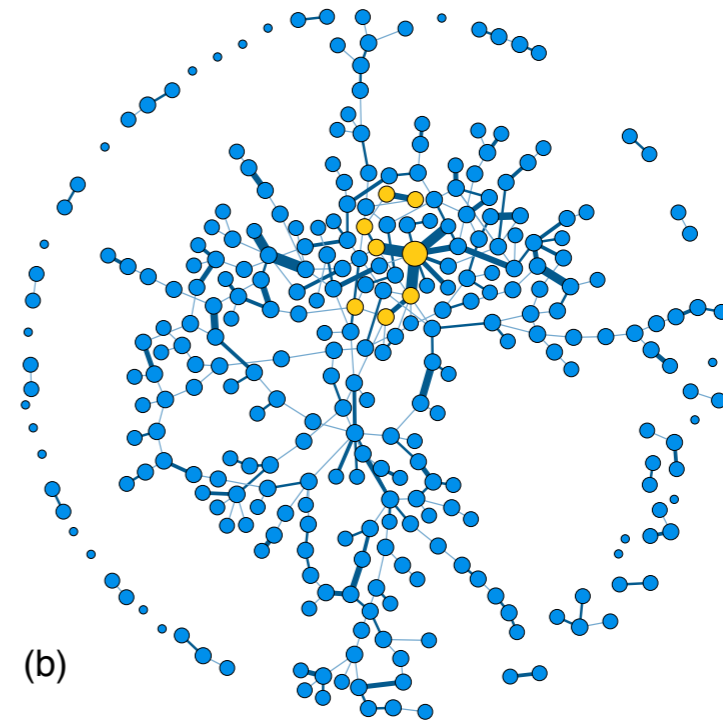
## Social contagion



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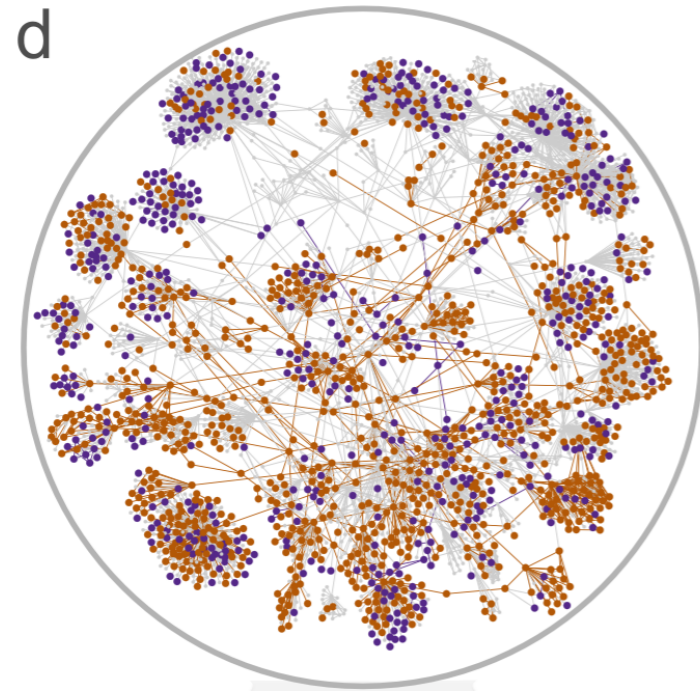
Information spreading



(b)

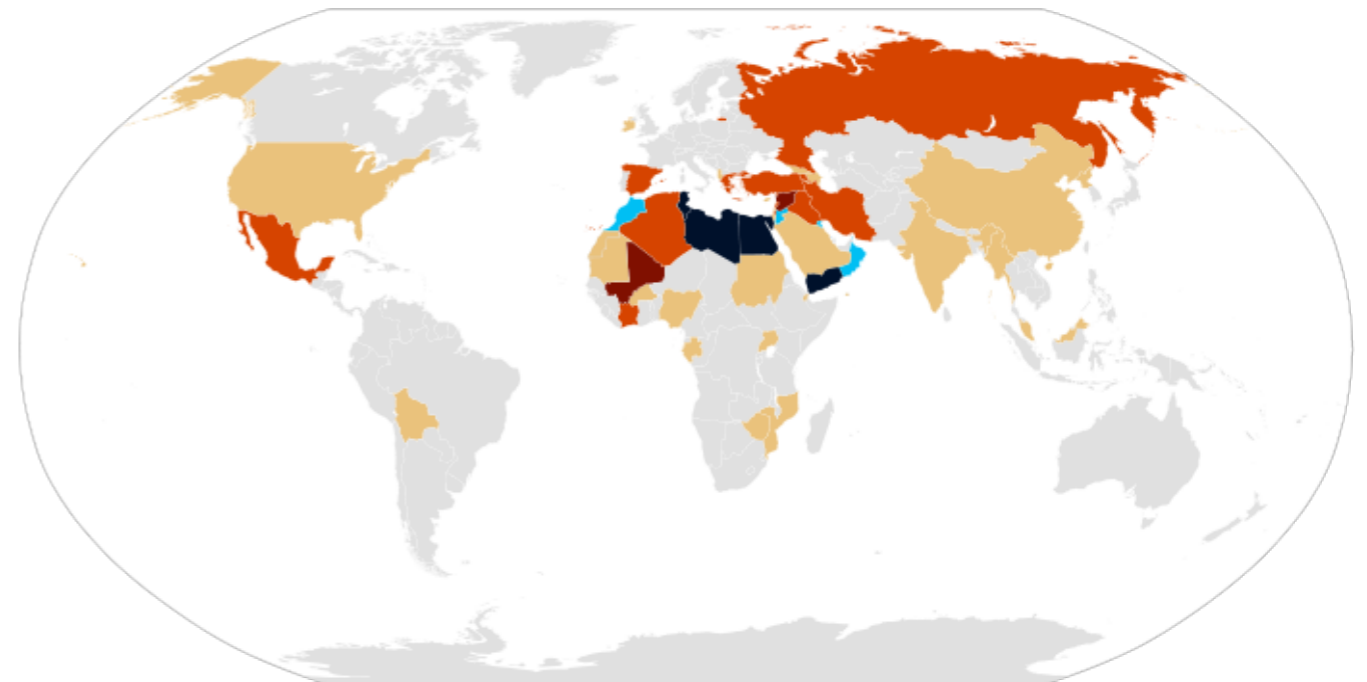
Rumour spreading

Karsai et.al. (2014)



Adoption spreading (Skype)

Karsai et.al. (2014)



Protest diffusion (Arabian spring)



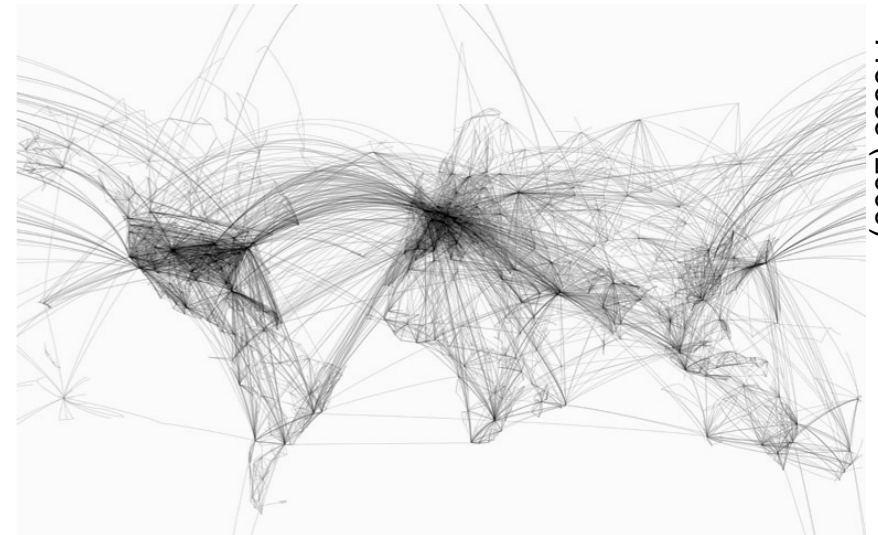
# Spreading processes

## Why on networks?

- Spreading usually happen through interactions between agents
  - Geographic vicinity
  - Physical connection
  - Social interaction
  - etc.
- Network structure critically influence the dynamics of spreading processes



PPD blog, Jooseery (2011)

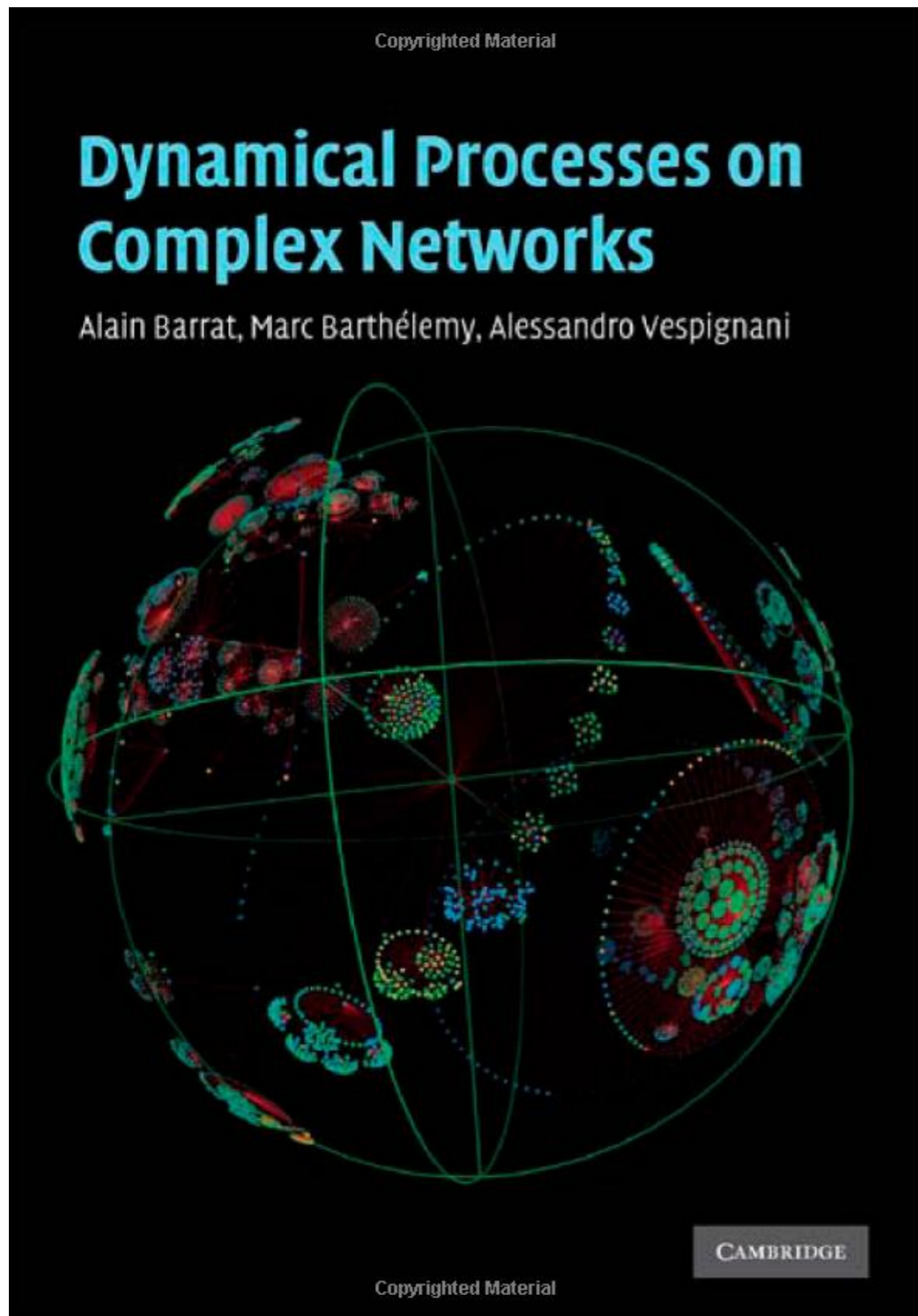


Freese (2009)



Unknown

# Literature



I'm not an epidemiologist!

Only an introduction,  
Trust the experts

Simple spreading

processes

# Spreading processes

## SI - SIR - SIS

Three of the most popular models of diffusion in epidemiology are the **SI**, **SIR** and **SIS** models. Letters correspond to the states in which individuals can be according to the model:

- **Susceptible:** Individual is not Infected
- **Infected:** Individual is Infected
- **Recovered/Removed.** Individual cannot be infected again (Considered cured or dead)

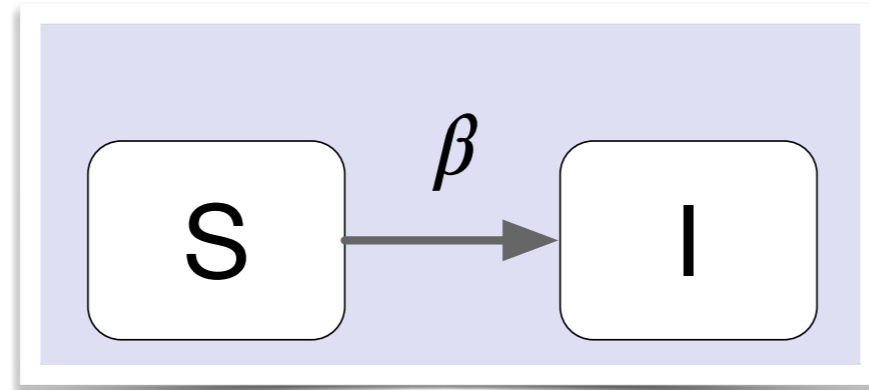
All individuals are in one of the states allowed by the model, and we define:

$s(t)$	Fraction of individuals in Susceptible state at time $t$
$i(t)$	Fraction of individuals in Infected state at time $t$
$r(t)$	Fraction of individuals in Recovered state at time $t$
$i_0$	Initial( $t = 0$ ) fraction of infected individuals

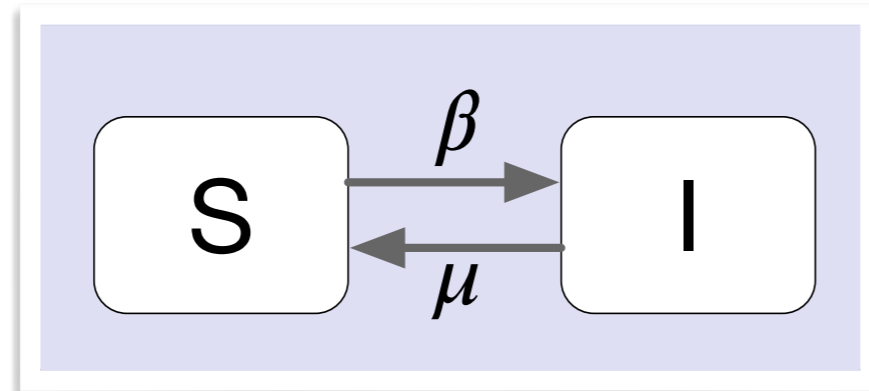


# Spreading processes

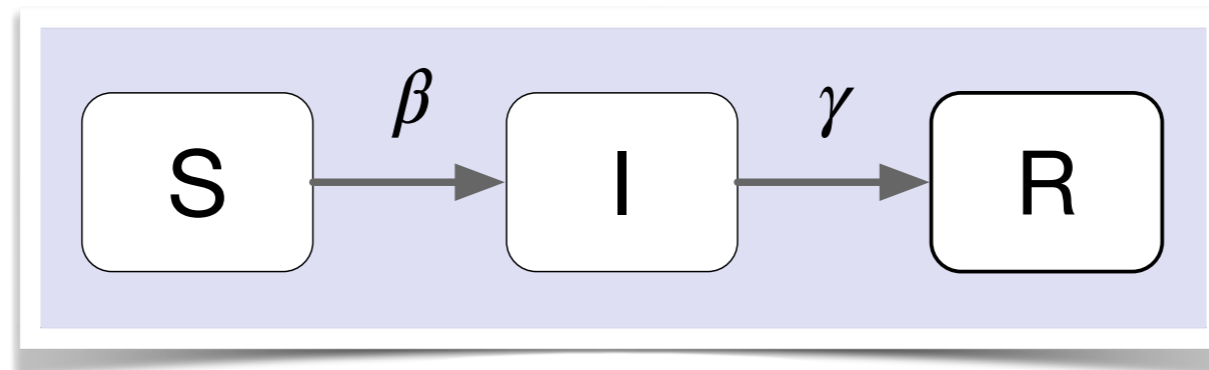
SI



SIS



SIR



# Homogeneous mixing

## Non-network approach

- Any individual can interact with any other
- The population has a finite size
- Individuals have an average number of contacts per unit of time

# SI model

$\tau$

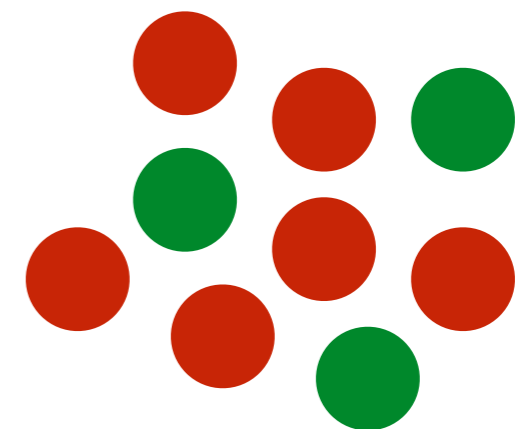
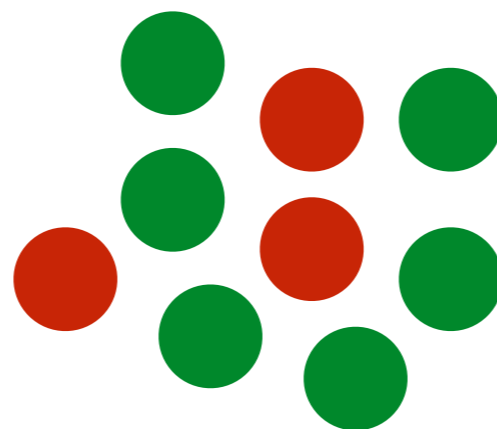
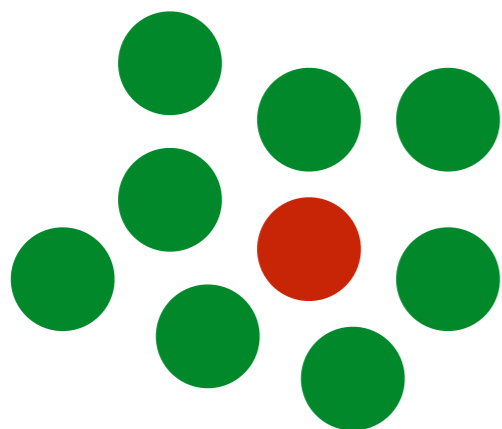
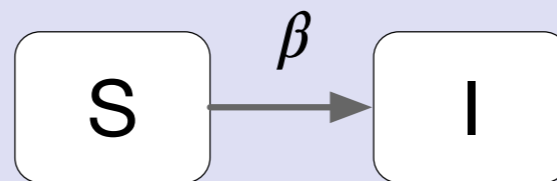
**Infectivity:** probability that the contact between an *Infected* individual and a *Susceptible* one results in the infection of the Susceptible.

$\hat{c}$

*Contact rate:* average number of contact per person per time

$\beta$

**Effective contact rate,**  $\beta = \tau \hat{c}$ , number of newly infected individuals by each infected individual in a population in which everyone else is susceptible.



# The SI model

## SI - characteristics

Each of the  $i$  infected individuals infects in average  $\beta$  contacts, but only  $s = (1 - i)$  of its contacts are indeed susceptible. More formally using differential equations:

$\frac{di}{dt}$	<b>Rate of new infection:</b> $\frac{di}{dt} = \beta i s = \beta(1 - i)i$
$i(t)$	<b>Infected fraction<sup>a</sup>:</b> $i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$
$s(t)$	<b>Susceptible fraction:</b> $1 - i(t)$



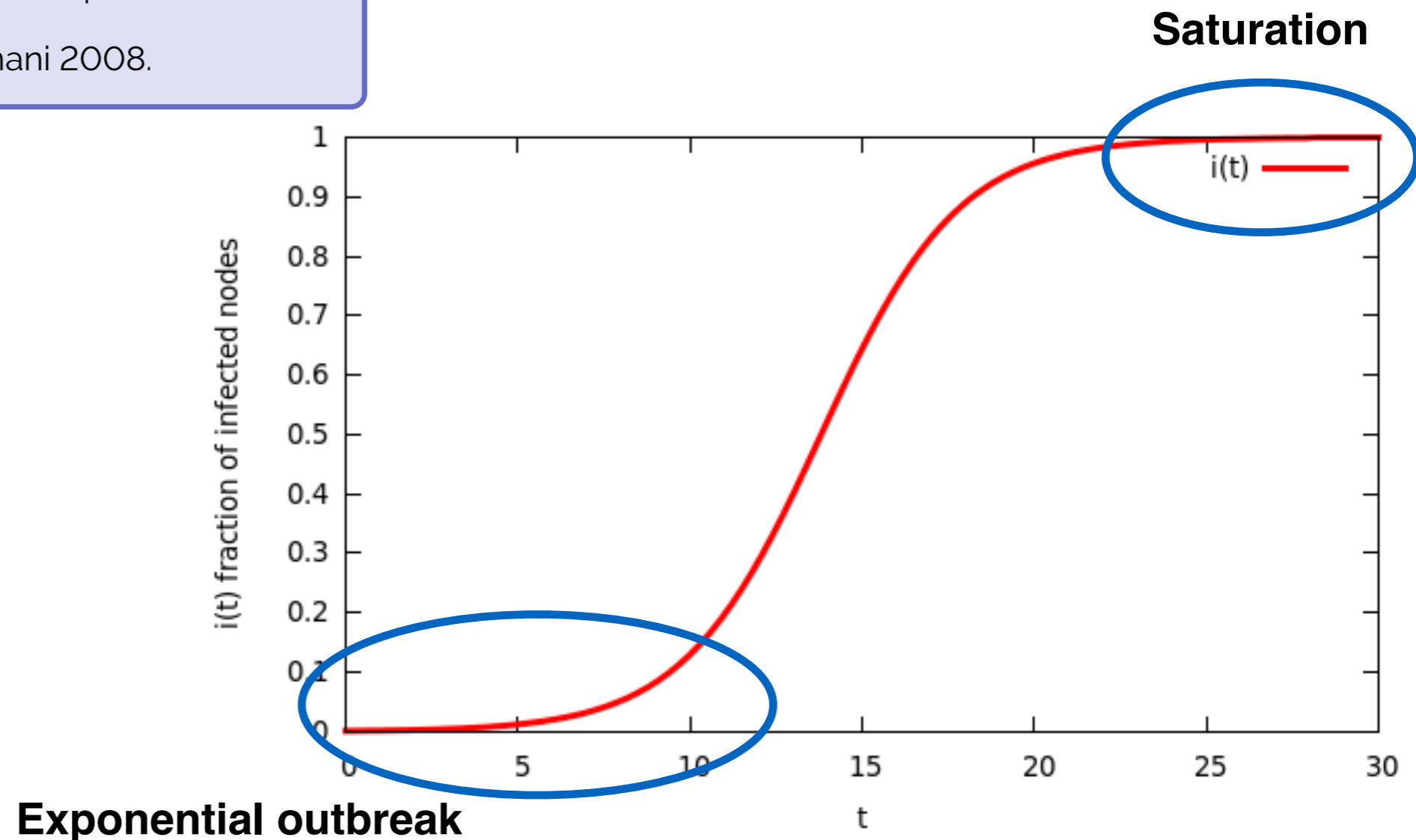
# The SI model

The process can be separated in three steps:

- At first, the fraction of infected individuals **Grows exponentially** until a large fraction of the population is infected. ( $i$  is small,  $\frac{di}{dt} \approx \beta i \Rightarrow$  exponential)
- Due to **saturation**, the infection of the last individuals is slow
- The growth is faster and faster until half the population is infected ( $\operatorname{argmax}_{x,y} (x(1-x)) : x = y = 0.5$ ).

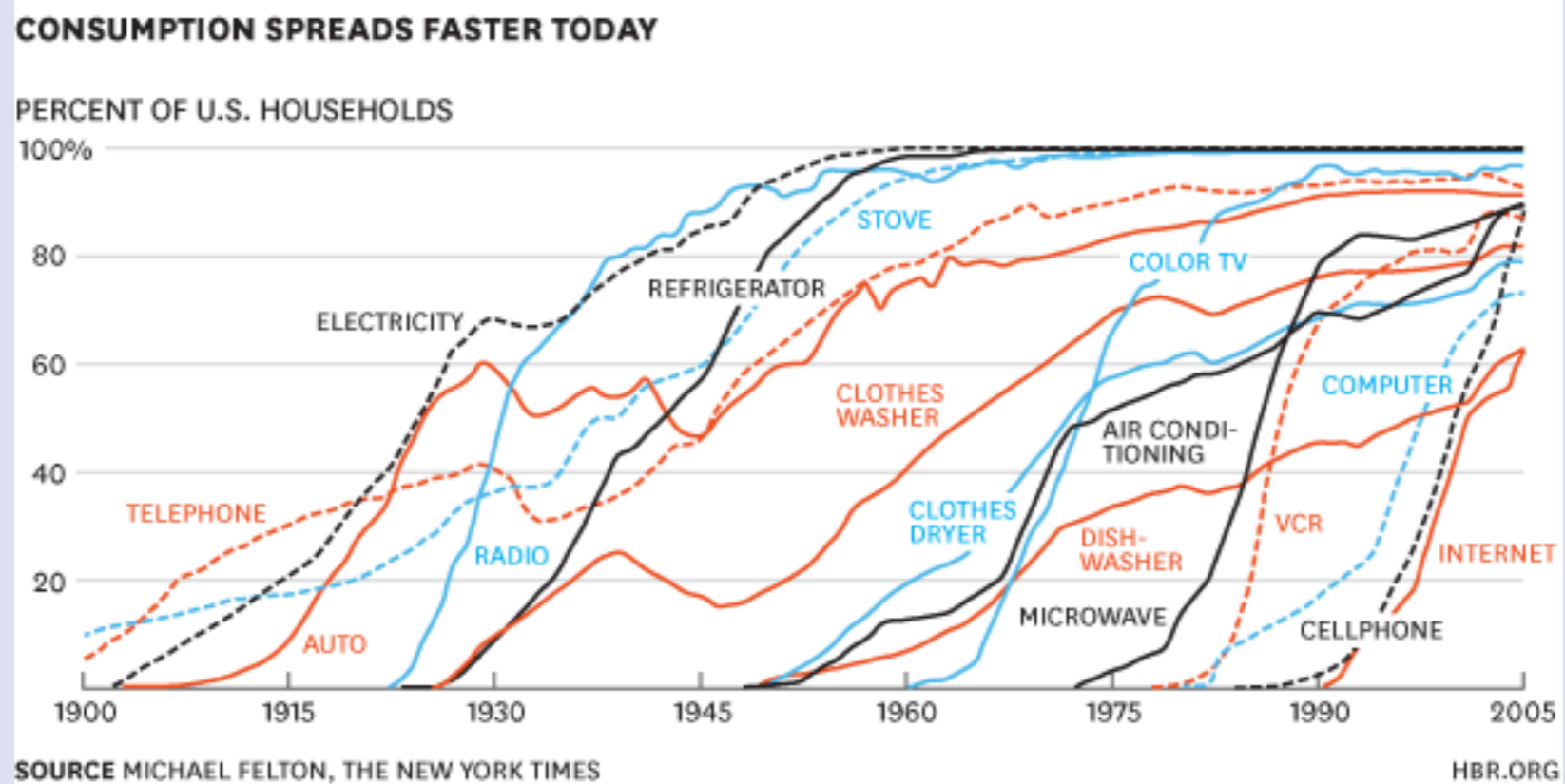
If  $\beta > 0$ , everyone is infected at the end of the process.

<sup>a</sup>Barrat, Barthelemy, and Vespignani 2008.



# The SI model

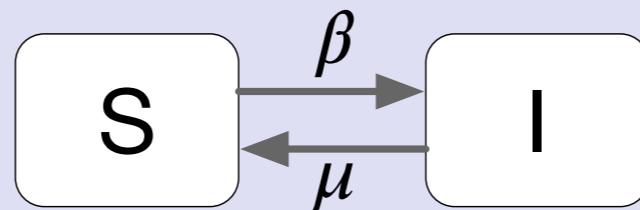
Example: technology adoption



# The SIS model

Additionally to  $\beta$ , the SIS model requires another parameter:

$\mu$  | **recovery rate**: probability that an *Infected* individual go back to the susceptible state per unit of time.



# The SIS model

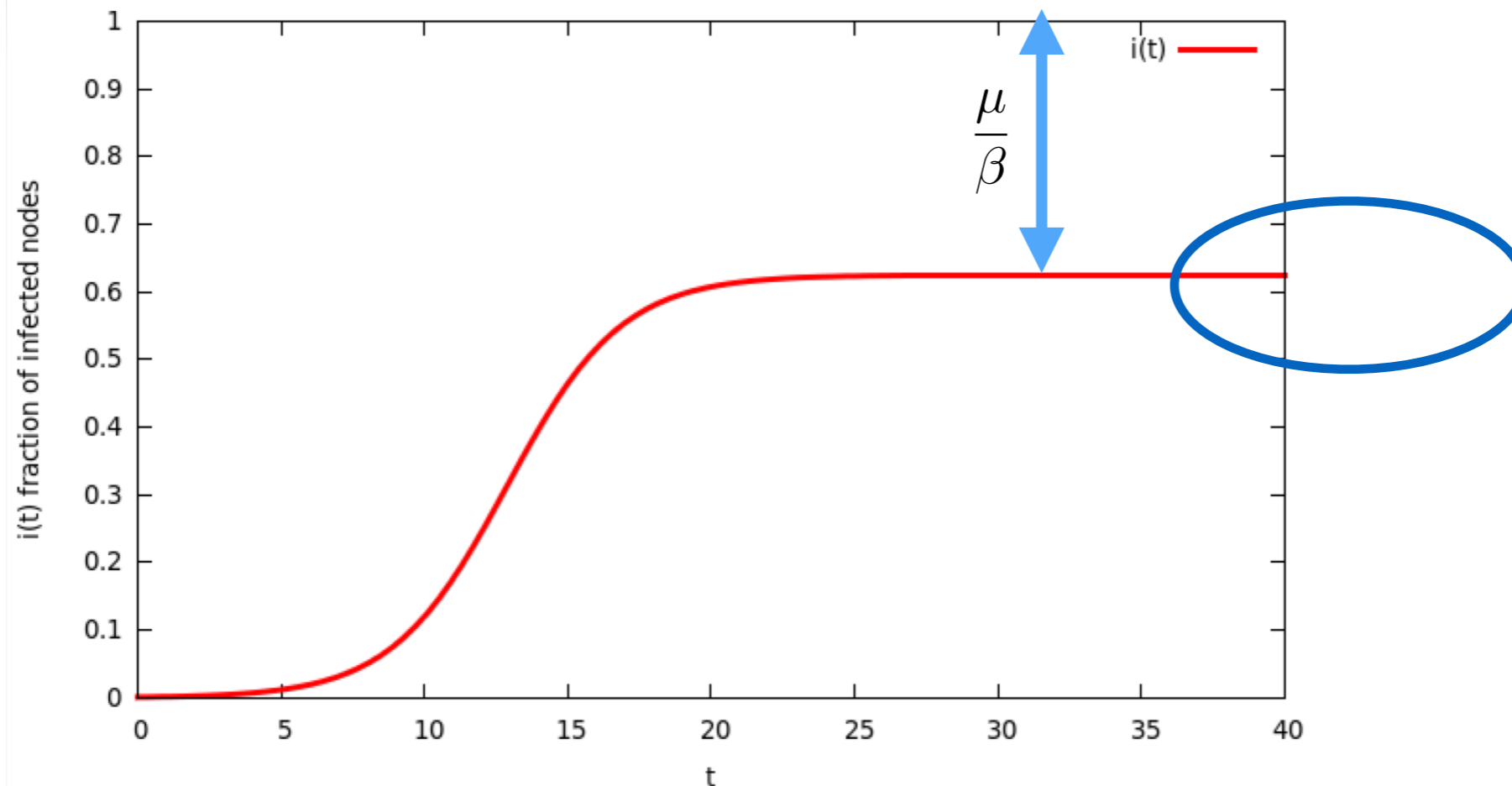
## SIS - characteristics

Intuitively, the fraction of infected individuals is now reduced by those switching to the susceptible state, more formally:

$$\begin{array}{l|l} \frac{di}{dt} & \text{Rate of new infection: } \beta i(1-i) - \mu i = i(\beta - \mu - \beta i) \\ i(t) & \text{Infected fraction}^a: \left(1 - \frac{\mu}{\beta}\right) \frac{C e^{(\beta-\mu)t}}{1 + C e^{\beta-\mu)t}} \end{array}$$

For large times,  $i(t) \rightarrow 1 - \frac{\mu}{\beta}$ , i.e., the fraction of infected individuals stabilize around a value which depends only of parameters  $\mu$  and  $\beta$ .

<sup>a</sup>Barrat, Barthelemy, and Vespignani 2008.





# The SIS model

## $\lambda$ ratio or ( $R_0$ )

In the SIS model, an important notion is the  $\lambda$  ratio, also called  $R_0$ .

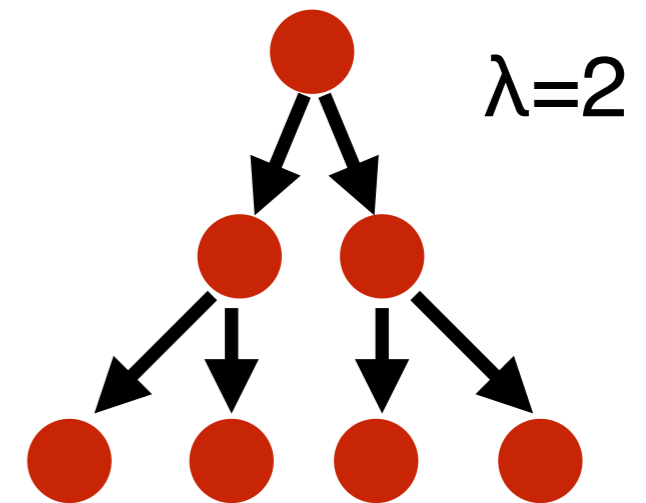
$$R_0 = \frac{\beta}{\mu}$$

$R_0$  can be understood as the average number of individuals that will be infected by an infected individual, **in a population in which all other nodes are Susceptible**.  $R_0$  is a property of the model and **do not change with time**.

Looking at the  $R_0$  is important in the early stage of the epidemic:

- if  $R_0 > 1$ , **there will be an outbreak**
- if  $R_0 < 1$ , **the epidemic will disappear naturally**.

If  $R_0$  is just above 1, the outbreak also can stop naturally by chance in the early stage.

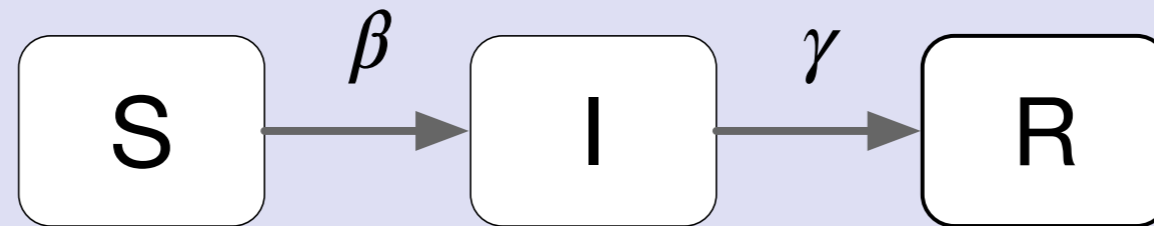


# The SIR model

Additionally to  $\beta$ , the SIR model requires another parameter:

$\gamma$

**recovery rate:** probability that an *Infected* individual switch to the Recovered state per unit of time.



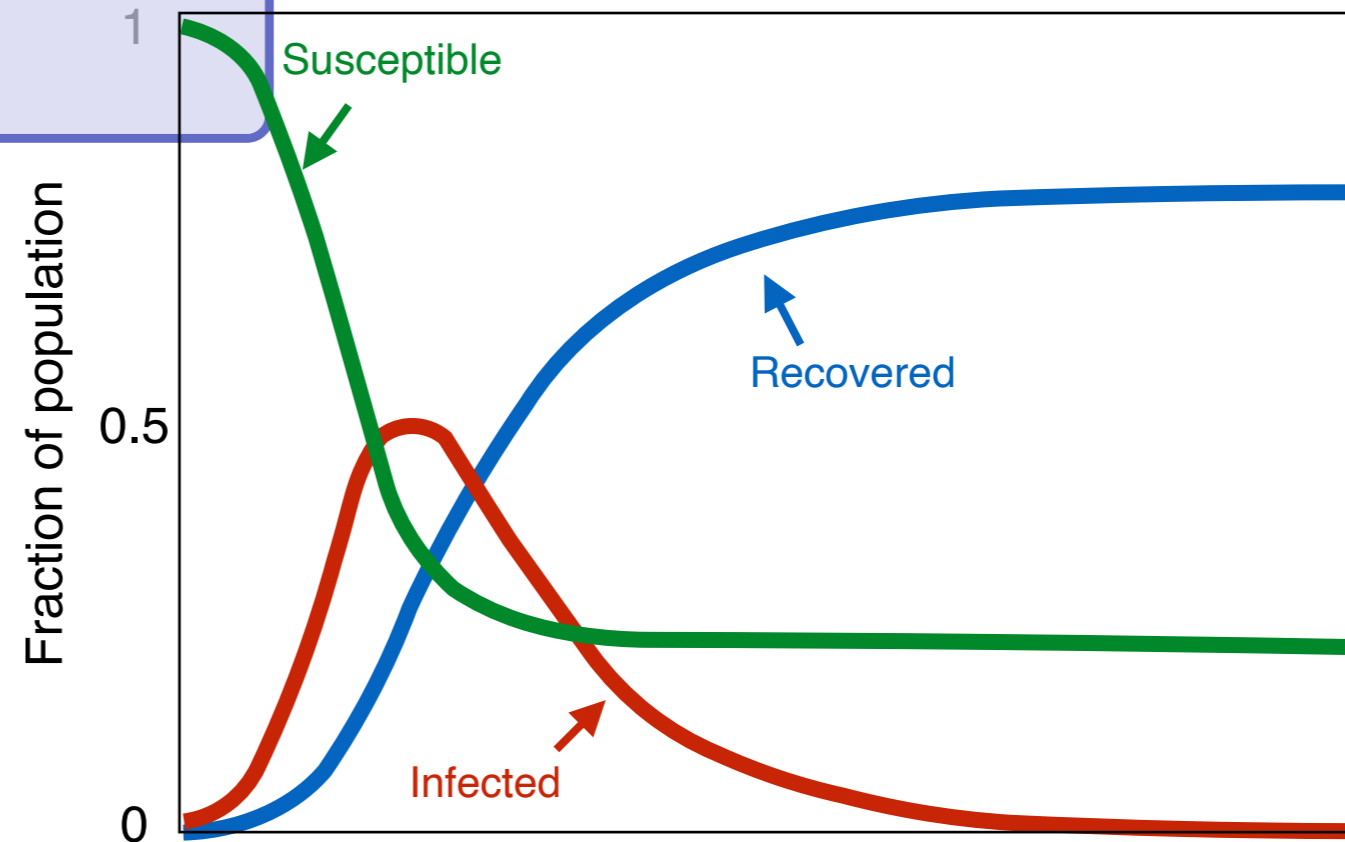
# Spreading processes

## SIR - characteristics

Intuitively, the fraction of infected individuals is now reduced by those switching to the recovered state, more formally:

$$\frac{ds}{dt} = -\beta is, \quad \frac{di}{dt} = \beta is - \gamma i, \quad \frac{dr}{dt} = \gamma i$$

- The initial steps of the outbreak still follow an exponential growth
- The fraction of infected nodes reach a peak and then decreases
- The fraction of recovered saturates below 1
- The fraction of susceptible do not necessarily reach 0
- The  $\lambda$  ratio is defined as  $\lambda = \frac{\beta}{\gamma}$



# Spreading processes

Many other models exist:

SIRD, MSIR, SEIR

SEIS, MSEIRS

Variable contact rate

Voter

Majority rule

Etc.

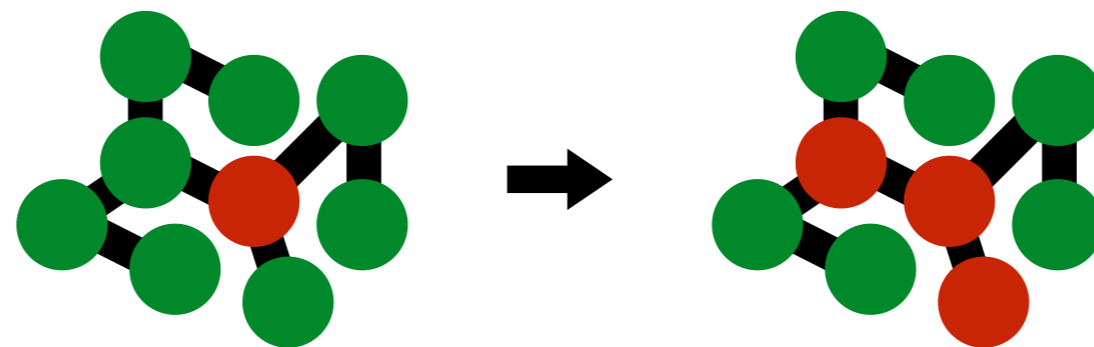
Check for instance:

<https://ndlib.readthedocs.io/en/latest/reference/reference.html#diffusion-models>

# Spreading on Networks

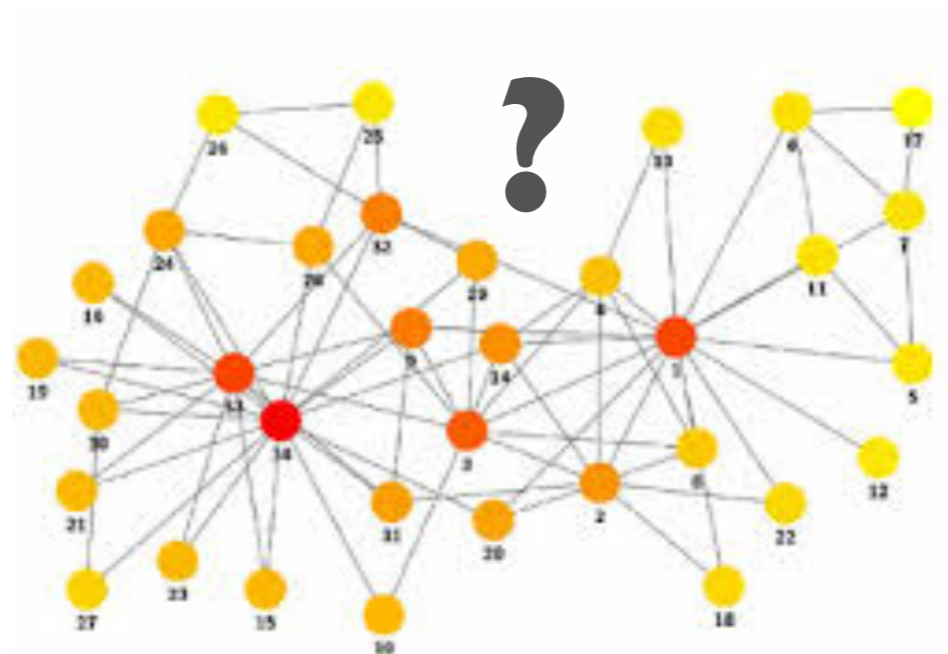
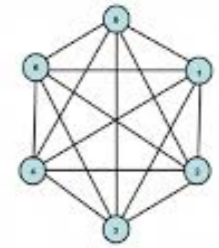
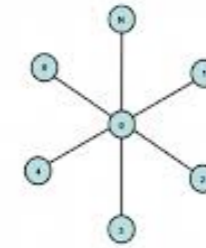
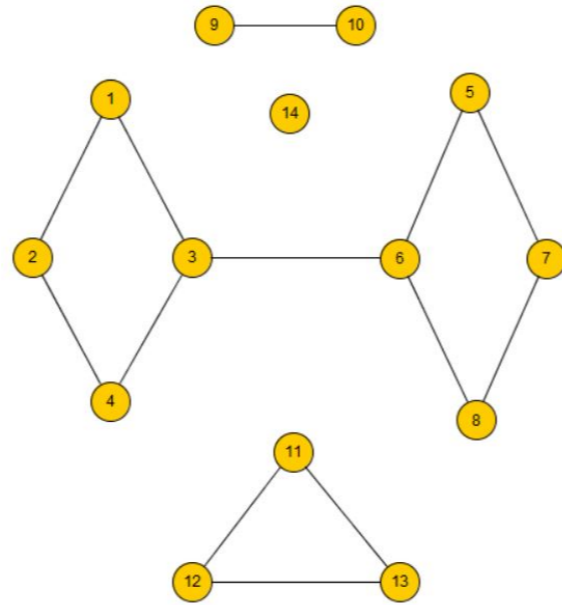
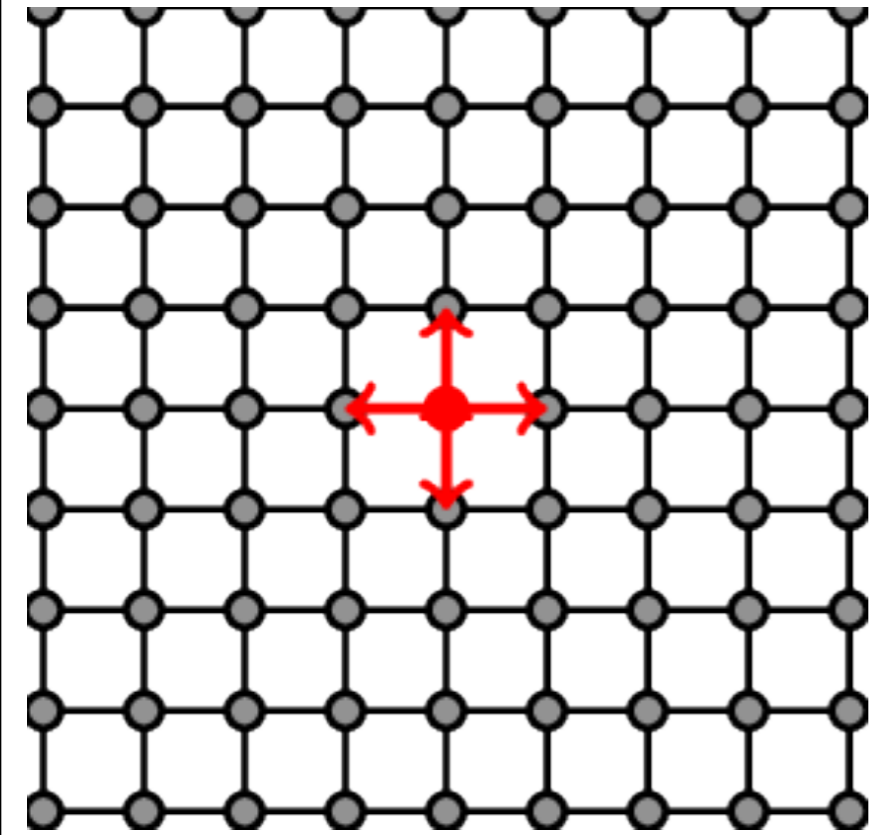
# Epidemic spreading on networks

The homogeneous mixing approach is clearly unrealistic: interactions are organized in networks



How much does it affect spreading?

# Epidemic spreading on networks





# Epidemic spreading on networks

## Notation change on networks

$\hat{c}$  has no meaning in networks (its role is played by the network structure), so by convention we use  $\beta = \tau$ : the probability for a node to infect each of its neighbor at each step.

On Networks

$$\beta = \tau$$

On homogeneous  
mixing

# Homogeneous networks

Homogeneous  
Mixing

$$\frac{di}{dt}$$

Rate of new infection:  $\frac{di}{dt} = \beta i s = \beta(1 - i)i$

## Homogeneous Networks

If we consider an **homogeneous random network** in which all nodes have degree exactly  $k$ , then we can consider the spreading on this network as similar to the non-network models, with  $\hat{c} = k$ . For instance, the SI model becomes:

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

ER random graph  $\Rightarrow$  approximation still holds,  
( $k \approx \langle k \rangle$ )

# Homogeneous networks

## $R_0$ on networks

In homogeneous or ER networks,  $R_0$  is naturally defined as  $\frac{\beta \langle k \rangle}{\mu}$   
Another way to express the same thing is that, if we define  $R_0 = \frac{\beta}{\mu}$ , then the epidemic threshold is not equal to 1 but to  $\frac{1}{\langle k \rangle}$

(Just a notation change)

# Epidemic spreading on heterogeneous networks

- In degree heterogeneous networks the  $k \approx \langle k \rangle$  approximation **does not hold**

- **Solution:** Degree Block Approximation

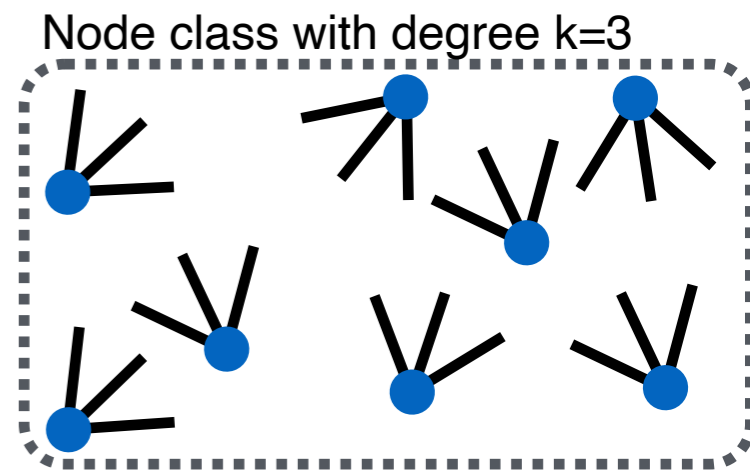
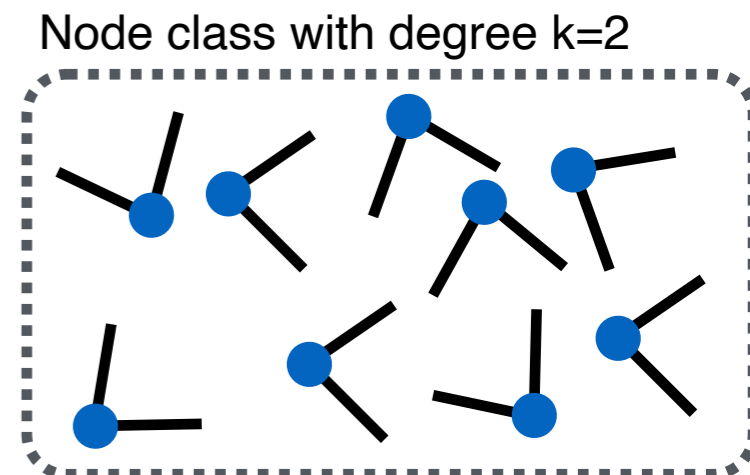
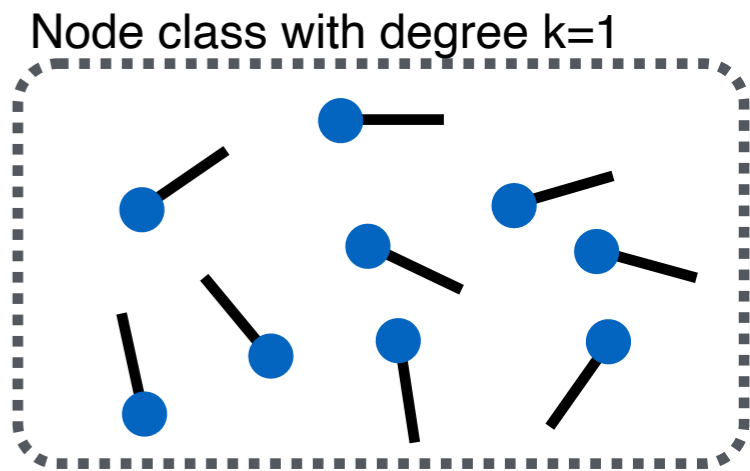
- **Assumption:** all nodes with the same degree are statistically equivalent
- Look for infection/susceptible node densities in the degree groups

$$i_k$$
$$s_k$$

- Calculate the global average by a sum considering the degree distribution

$$i = \sum_k P(k) i_k$$

$$s = \sum_k P(k) s_k$$



# Epidemic spreading on heterogeneous networks

Homogeneous  
Networks

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

## Heterogeneous Degrees - SI

For the SI model, we know that all nodes are infected in the end, but what may vary is **speed** of the process.

The speed of diffusion by degree block can be expressed as:

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

with  $\Theta_k$  being the fraction of infected neighbors of a node with degree  $k$ .

# Epidemic spreading on heterogeneous networks

- Due to the friendship paradox, nodes are more likely to be connected to large nodes than to small ones

$$\Theta_k = \sum_{k'} P(k'|k) i_{k'}$$

- Assume: **no degree-degree correlations** in the network

Number of stubs of degree  $k'$  / Total Number of stubs (normalized by nb node)

$$P(k'|k) = \frac{k' P(k')}{\sum_{k''} k'' P(k'')} = \frac{k' P(k')}{\langle k \rangle}$$

And:

$$\Theta_k = \Theta = \frac{\sum_{k'} k' P(k') i_{k'}}{\langle k \rangle}$$



# SI process on heterogeneous networks

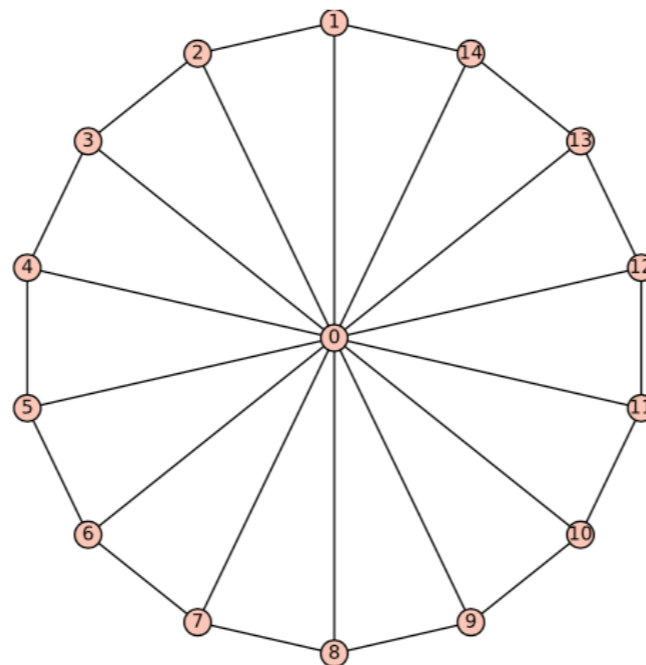
## Heterogeneous Degrees - SI - time scale

From previous equations, it can be shown<sup>a</sup> that the **time scale**  $\tau$  of the process, i.e., a measure inversely proportional to its speed,

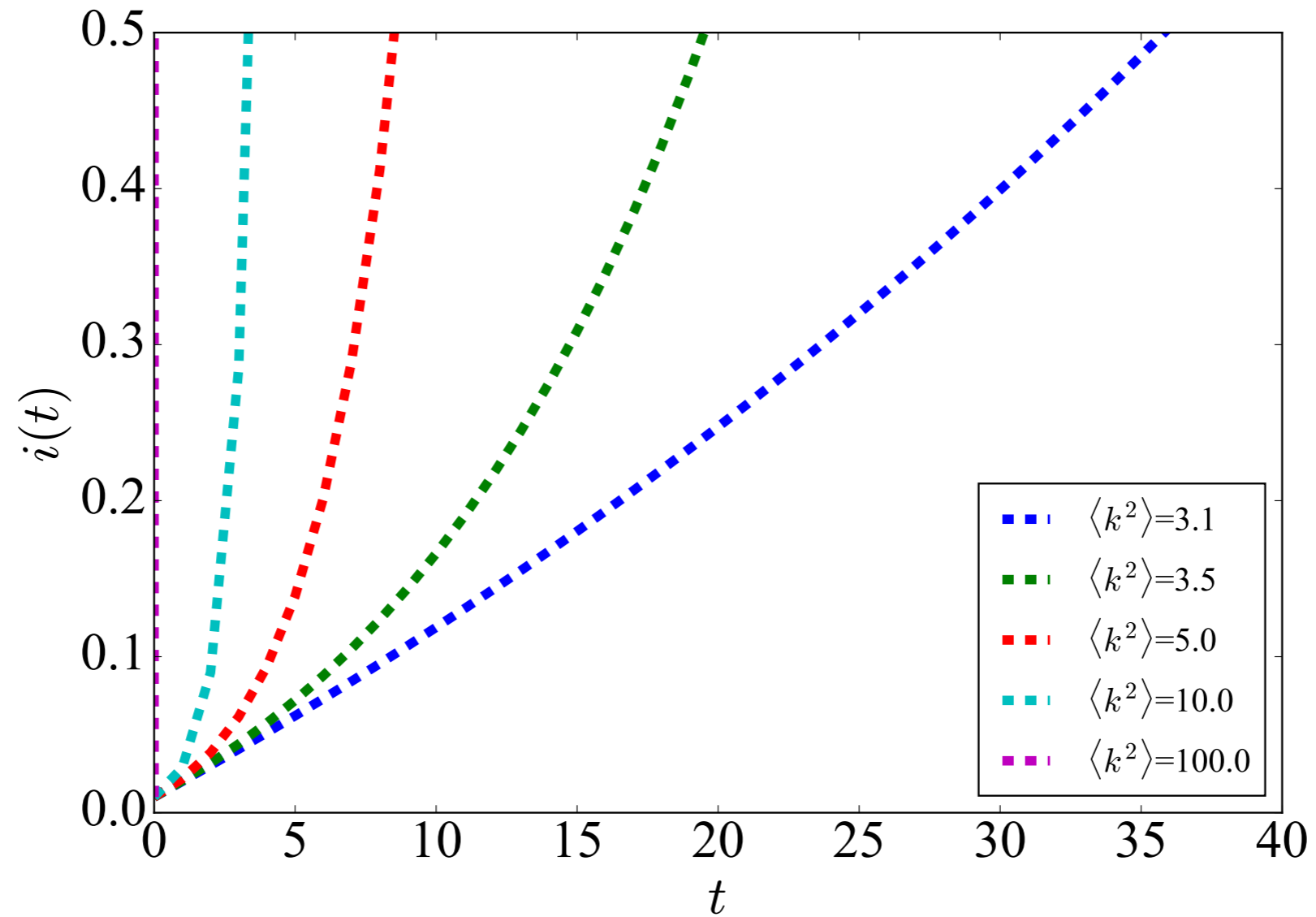
$$\text{is } \tau = \frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)}.$$

Thus, for a given average degree  $\langle k \rangle$  and a given  $\beta$ , **the more heterogeneous the degrees, the faster the diffusion.**

If the degree distribution follows a power law of exponent  $\alpha < 3$ , we have seen that  $\langle k^2 \rangle$  diverge towards infinity, thus  $\tau$  tends toward 0, thus the diffusion is nearly instantaneous.



# SI process on heterogeneous networks



# SIS process on heterogeneous networks

## Heterogeneous Degrees - $\lambda$

For SIS and SIR models, it can also be shown<sup>a</sup> that the epidemic threshold  $\lambda$  (or  $R_0$ ) is not reached when  $\lambda = \frac{\beta \langle k \rangle}{\mu} > 1$  as in homogeneous networks, but when  $\lambda > \frac{\langle k \rangle^2}{\langle k^2 \rangle}$ .

This means that in a very heterogeneous network, **an outbreak can start even if  $\lambda$  is very small, and below 1**. Intuitively, even if

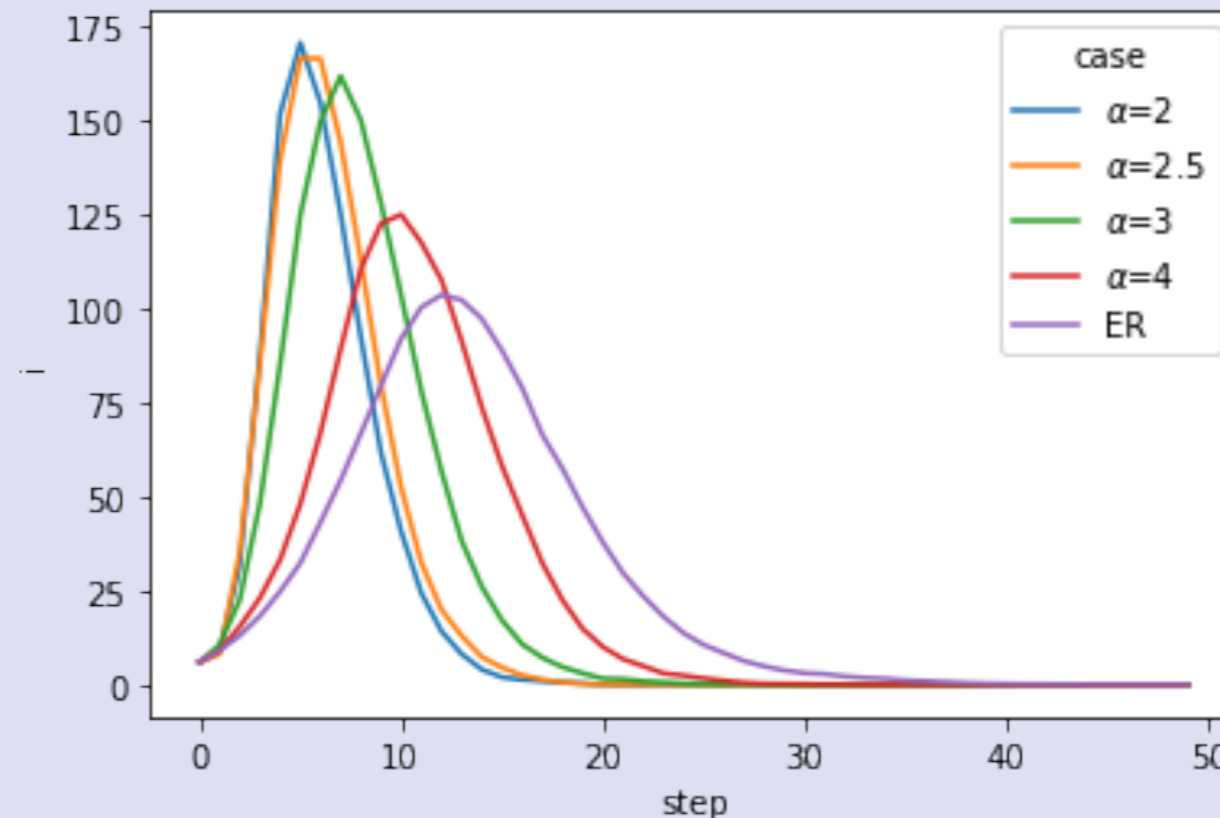
# Experiments

## SIR - Scale Free

In this experiment, we compare an ER network to Configuration Models with power law degree distributions.

**Network parameters:**  $n = 1000$ ,  $\langle k \rangle = 5$ . We vary the exponent of the distribution, while keeping  $\langle k \rangle = 5$  constant.

**SIR parameters:**  $\theta = 0.2$ ,  $\gamma = 0.5$ . The initial number of infected nodes is 5, all of them in the same community structure.



The higher the exponent of the degree distribution, the faster is the diffusion.

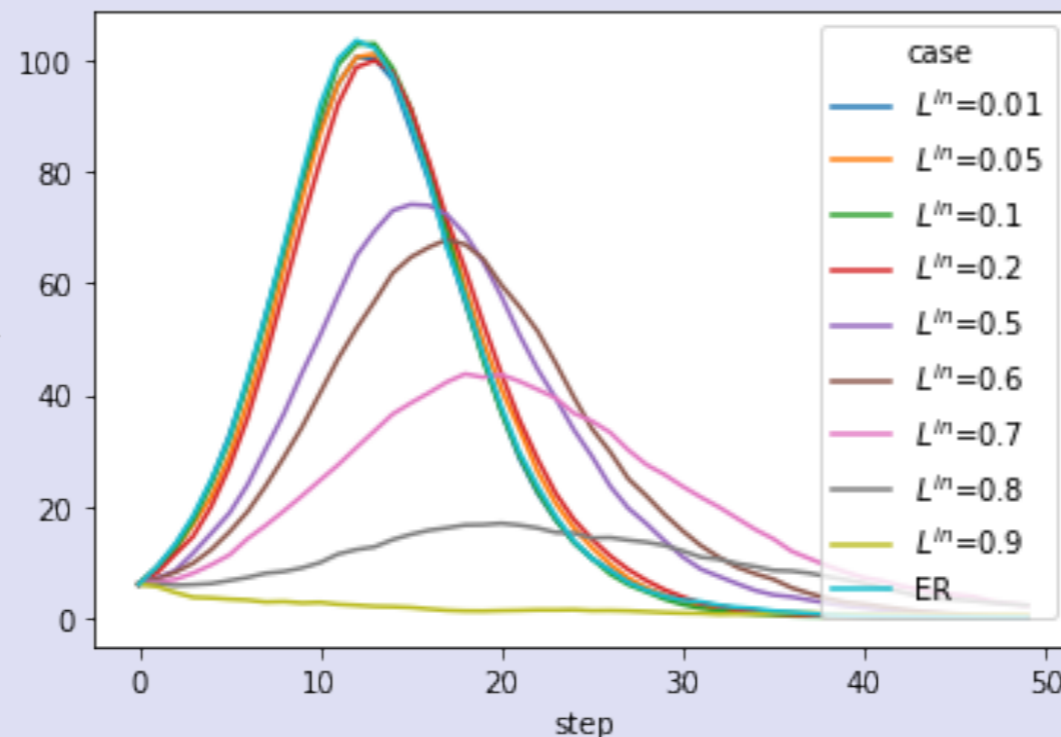
## SIR - Community Structure

In this experiment, we compare an ER network to Stochastic Block Models.

**Network parameters:**  $n = 1000$ ,  $\langle k \rangle = 5$ .

**SBM parameters** Number of blocks  $|C| = 100$ . We vary  $L^{in}$ , the fraction of all edges that are inside blocks. When  $L^{in} = 0.01$ ,  $p^{in} \approx p^{out} = 0.005$ . When  $L^{in} = 0.9$ ,  $p^{in} = 0.5$ ,  $p^{out} \approx 0.0005$

**SIR parameters:**  $\theta = 0.2$ ,  $\gamma = 0.5$ . The initial number of infected nodes is 5, all of them in the same community structure.



We observe that the more marked the communities, the less efficient the spreading process.

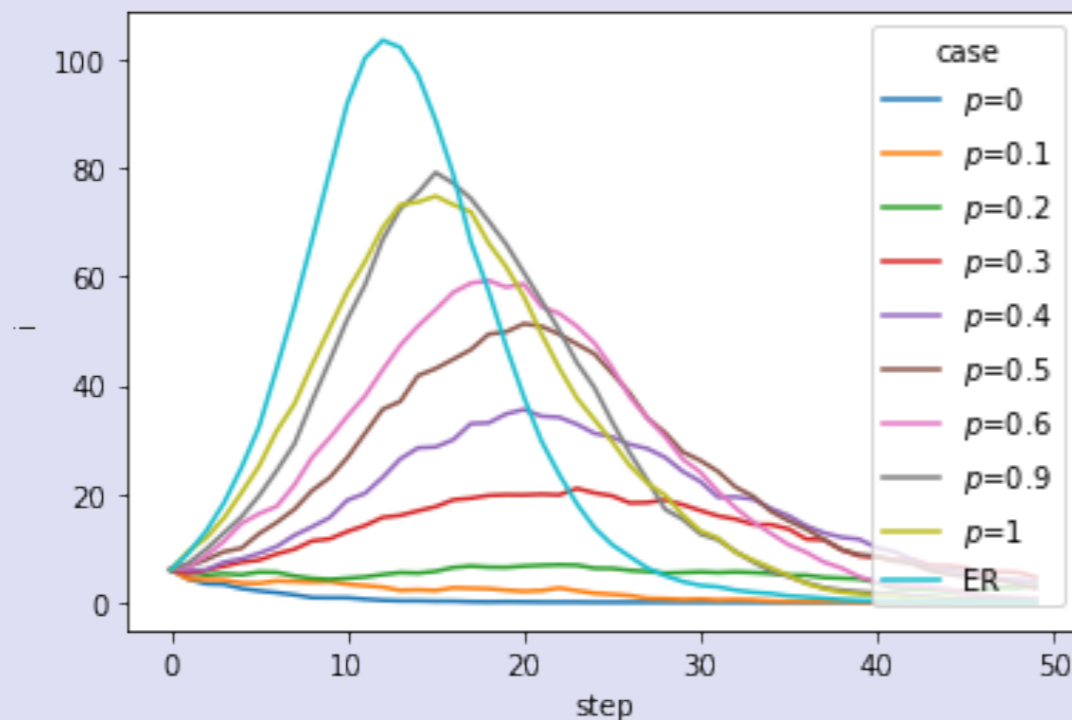


# SIR - Spatial effect - WS

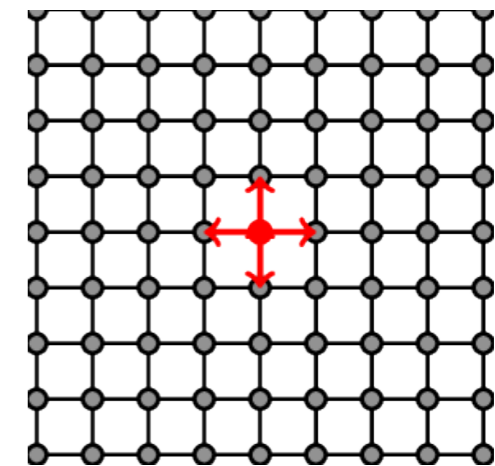
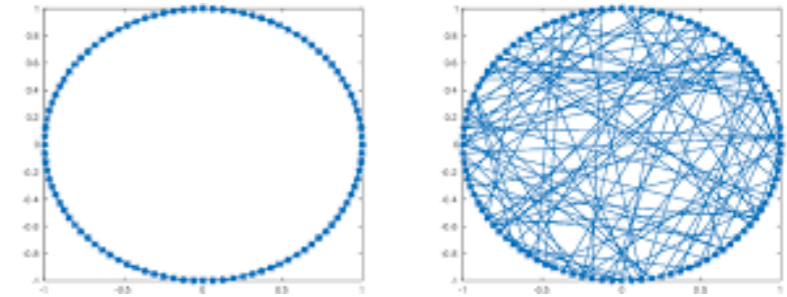
In this experiment, we compare an ER network to Watts Strogatz random graphs, varying the probability of rewiring edges. It can be understood as a model of spatial proximity: with  $p = 0$ , each node is connected only to its direct neighbors in the 1 dimensional space. If  $p = 1$ , each node is connected to exactly  $k$  random nodes.

**Network parameters:**  $n = 1000$ ,  $\langle k \rangle = 5$

**SIR parameters:**  $\theta = 0.2$ ,  $\gamma = 0.5$ . The initial number of infected nodes is 5, being 5 direct neighbors.



The more nodes tend to be connected to direct neighbors in space, the slower the diffusion.



# Applications

# Applications

- Model fitting (to better know an observed diffusion)
- Predicting future trends
- Epidemic control
  - Vaccine, etc. => Which nodes/edges to target?
- Example of strategy: friend paradox
  - Vaccine contacts of random nodes instead of random nodes

---

<sup>a</sup>Cohen, Havlin, and Ben-Avraham 2003.

# OTHER MODELS

- Many other diffusion models
  - ▶ Contagious but without symptoms state
  - ▶ Propagation of information information
  - ▶ Opinion dynamics (states correspond to opinion, e.g., red/blue), diffusion rules can vary a lot
    - Majority rule: your opinion change to the one of the majority around you
    - Repeated exposition rule: each time you are exposed to an idea, you are likely to change your opinion
    - Etc.