Lecture 10: Processes on Networks II

Current research on complex networks

• Characterisation:

average connectivity, degree distribution, clustering, diameter, network motifs, degree correlations

• Network models:

random graphs, small world networks, scale-free networks

• Dynamic processes on complex networks:

spreading phenomena: epidemics, rumours, computer viruses, information dissemination.

Epidemic models try to capture the dynamics in the spreading of a disease (or rumor, computer virus, information, etc)

Central questions:

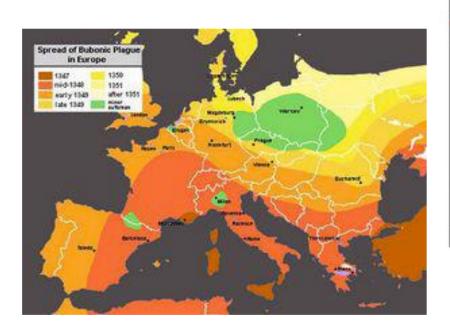
- How do contagion spread in a population?
- Will a disease become an epidemic?
- What is the best vaccination strategy?
- Classic epidemic models in mean-field (full mixing)
- Epidemic models over networks

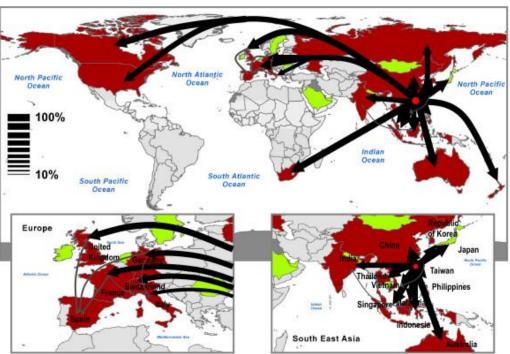


R. Pastor-Satorras, C. Castellano, P. Van Mieghem, A. Vespignani, "Epidemic processes in complex networks", Rev. Mod. Phys. 87, 925 (2015).

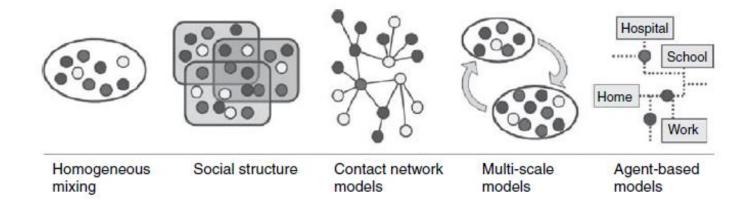
Assumptions:

- Full mixing assumption: every individual has an equal chance of coming into contact with every other individual in the population
- Network assumption: the underlying social network determines chances of coming into contact
 more realistic!





Epidemic Models:

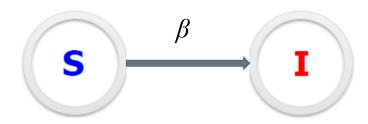


Structures at different scales used in epidemic modeling. Circles represent individuals and each shade corresponds to a specific stage of the disease. From left to right: homogeneous mixing, in which individuals are assumed to interact homogeneously with each other at random; social structure, where people are classified according to demographic information (age, gender, etc.); contact network models, in which the detailed network of social interactions between individuals provide the possible virus propagation paths; multiscale models which consider subpopulations coupled by movements of individuals, while homogeneous mixing is assumed on the lower scale; agent-based models which recreate the movements and interactions of any single individual on a very detailed scale (a schematic representation of a part of a city is shown).

The SI Model:

Individuals can be in one of two states:

- Susceptible (S)
- Infected (I)



- Contact with I causes S to become I
- I never recovers!

Parameters:

• β (infection rate): probability of contagion after contact per unit t

The SI Model:

For a population consists of N people, the average probability of a person one will meet at random being susceptible is S/N, and hence an infected person has contact with an average of $\beta S/N$ susceptible people per unit time. Since there are on average I infected individuals in total, implying the overall average rate of new infections will be $\beta SI/N$, and S+I=N. Therefore,

$$\frac{dI}{dt} = \frac{\beta SI}{N}$$

Let i=I/N, we have

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

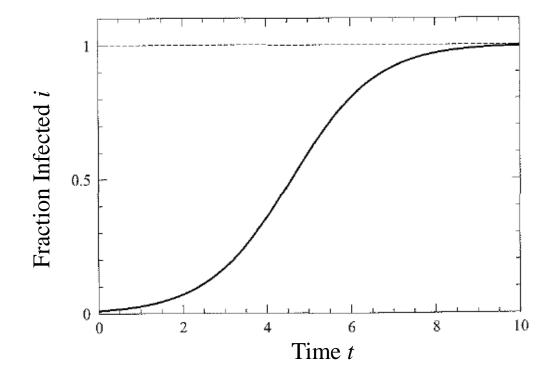
** This is a *logistic growth equation* with a standard solution,

$$i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$$

where i_0 is the value of i at t=0.

The SI Model:

The *classic logistic growth curve* of the SI epidemic model. A small initial number of infected individuals in an SI model (1% in this example) will at first grow exponentially as they infect others, but growth eventually saturates as the supply of susceptible individuals is exhausted, and the curve levels off at i = 1.



$$i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$$

The SIR Model:

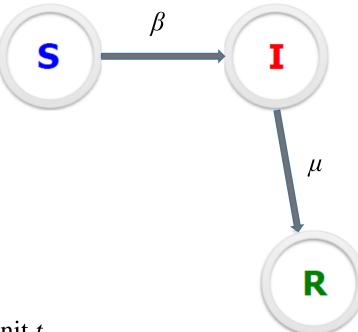
Individuals can be in one of two states:

- Susceptible (S)
- Infected (I)
- Recovered (R)
- Contact with I causes S to become I
- \triangleright I recovers after some time and becomes R

Parameters:

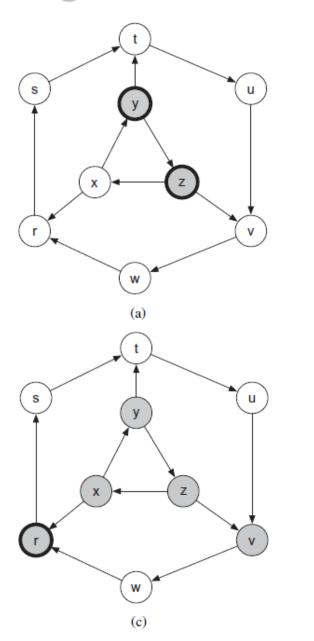
- β (infection rate): probability of contagion after contact per unit t
- μ (recovery rate): probability to recover from infection per unit t

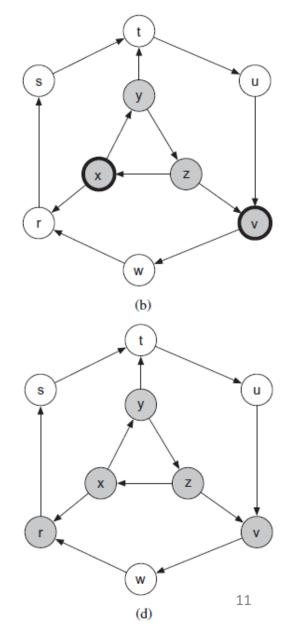
Example: chickenpox (recovered = immune), ebola (removed = dead)



The SIR Model:

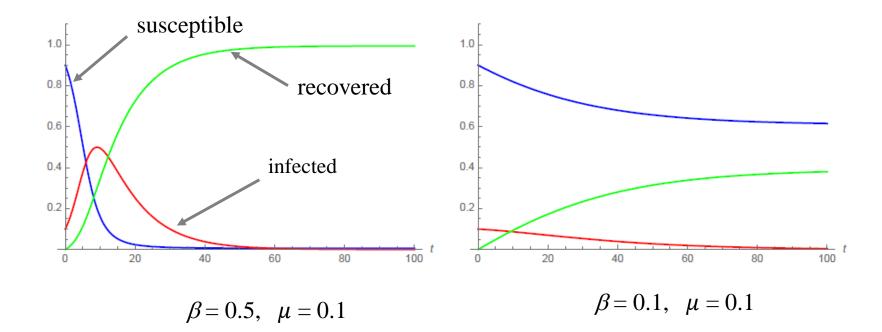
The course of an SIR epidemic in which each node remains infectious for a number of steps equal to $t_I = 1$. Starting with nodes y and z initially infected, the epidemic spreads to some but not all of the remaining nodes. In each step, shaded nodes with dark borders are in the infectious (I) state and shaded nodes with thin borders are in the removed (R) state.





The SIR Model:

$$\frac{di}{dt} = \beta i s - \mu i$$
; $\frac{ds}{dt} = -\beta i s$; $\frac{dr}{dt} = \mu i$; $i + s + r = 1$



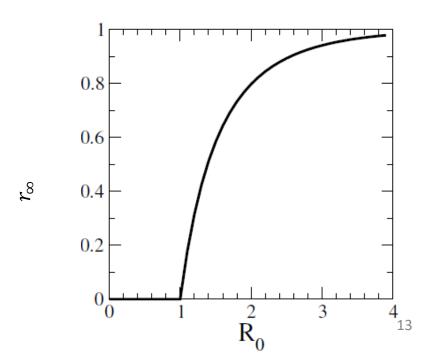
$$s_0 = 90\%$$
 , $i_0 = 10\%$, $r_0 = 0\%$

The SIR Model:

One can easily obtain, $s = s_0 e^{-\beta r/\mu}$

We are also interested to the population that will get sick (i.e., the size of the epidemic), which is basically captured by r(t) as $t \to \infty$, when $i(t) \to 0$, and we have $r_{\infty} = 1 - s_{\infty}$. We define R_0 to be the reproduction number, given by $R_0 = \beta/\mu$. Then, $r_{\infty} = (1 - e^{-R_0} r_{\infty})$

 $R_0 = 1$ is the epidemic threshold, which separates the transition between the epidemic and non-epidemic regimes

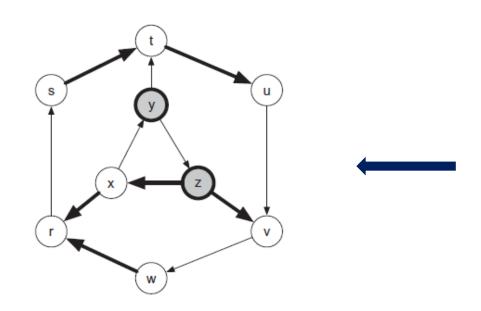


The SIR Model:

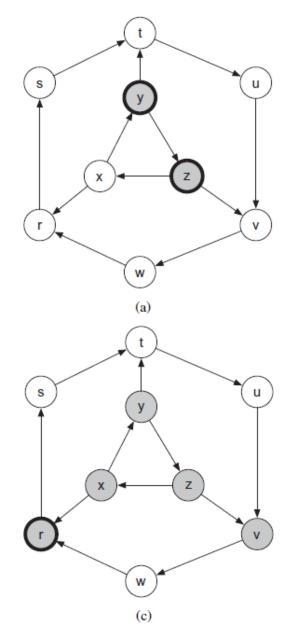
From a static viewpoint, the SIR model is equivalent to **Percolation** in physics. Consider a point in an SIR epidemic when a node v has just been infected, and has a susceptible neighbor w. Node v has one chance to infect w with probability p. Now view this random event as being determined by flipping a coin that has a probability p of "heads". From the point of view of the process, it does not matter whether the coin was flipped at the moment that node v first became infectious or was flipped at the very beginning of the whole process and is only being revealed now. Continue to flip the coin for each edge in the network, and store the result so that it can be later checked in the event that node v becomes infectious while node v is susceptible. Now the edges in the network for which the outcome is a "head" are set to be connected; and the remaining edges are declared to be blocked.

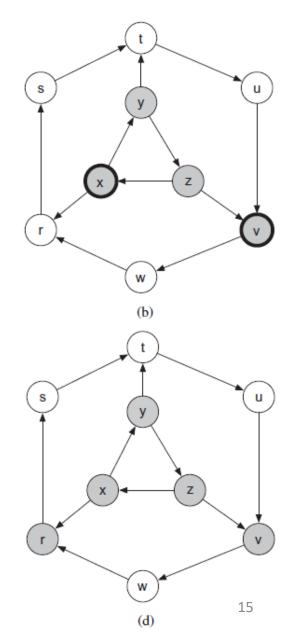
A node will become infected during the epidemic if and only if there is a path to the node from one of the initially infected nodes that consists entirely of open edges.

The SIR Model:



An equivalent way to view an SIR epidemic is in terms of percolation, where we decide in advance which edges transmit infection (should the opportunity arise) and which do not.

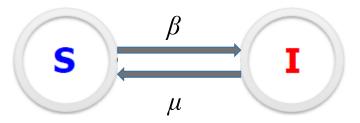




The SIS Model:

Individuals can be in one of two states:

- Susceptible (S)
- Infected (I)



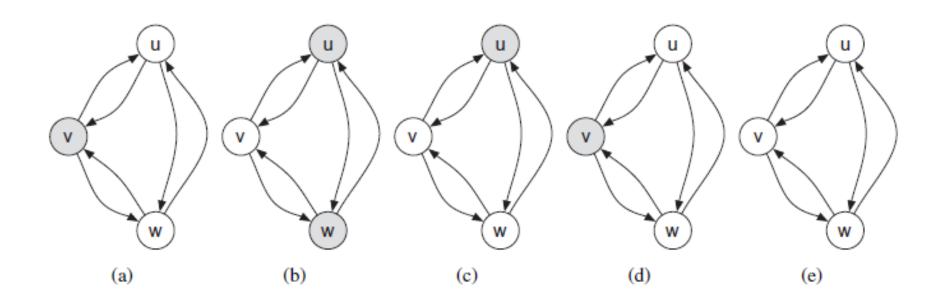
- Contact with I causes S to become I
- > I recovers after some time and becomes S!

Parameters:

- β (infection rate): probability of contagion after contact per unit t
- μ (recovery rate): probability to recover from infection per unit t

Example: common cold, influenza (no long lasting immunity)

The SIS Model:



In an SIS epidemic, nodes can be infected, recover, and then be infected again. In each step, the nodes in the infectious state are shaded.

The SIS Model: Solution

$$\frac{di}{dt} = \beta i s - \mu i \; ; \qquad \frac{ds}{dt} = \mu i - \beta i s \; ; \qquad i + s = 1 \; ; \qquad R_0 = \frac{\beta}{\mu}$$

$$\frac{ds}{dt} = \mu i - \beta i s \; ;$$

$$i+s=1 ;$$

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

People can recover now

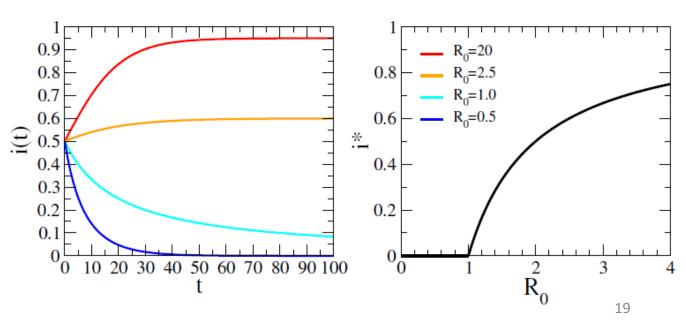
$$i(t) = \frac{i_0 e^{\mu(R_0 - 1)t}}{1 + i_0 \left[\frac{R_0}{R_0 - 1}\right] \left[e^{\mu(R_0 - 1)t} - 1\right]}$$

$$\begin{cases} R_0 \leq 1 \ \Rightarrow \qquad \qquad i(t) \approx i_0 \, e^{\mu(R_0-1)t} & \stackrel{t \to \infty}{\longrightarrow} \quad 0 \qquad \qquad \text{Exponential decay} \\ R_0 > 1 \ \Rightarrow \quad i(t) \approx \big[1 - \frac{1}{R_0} + i_0^{-1} e^{-\mu(R_0-1)t}\big]^{-1} & \stackrel{t \to \infty}{\longrightarrow} \quad 1 - 1/R_0 \qquad \text{Logistic growth} \end{cases}$$

The SIS Model: Solution

 R_0 : basic reproduction ratio = average number of additional people that a newly infected person passes the disease to before recovery

- $R_0>1$: each infected person infects more than 1 person => the epidemic grows exponentially (at least at the early stages)
- R_0 <1: the epidemic shrinks
- $R_0=1$ marks the epidemic threshold!



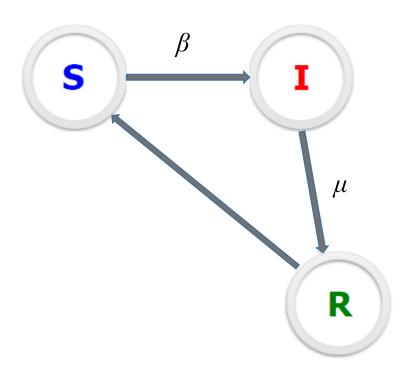
The SIRS Model:

Individuals can be in one of two states:

- Susceptible (S)
- Infected (I)
- Recovered (R)
- Contact with I causes S to become I
- > I recovers after some time and becomes R
- **R** becomes **S** again after some time

Parameters:

- β (infection rate): probability of contagion after contact per unit t
- μ (recovery rate): probability to recover from infection per unit t
- δ (immunity loss rate): probability to lose immunity per unit t



The SIRS Model:

$$\frac{di}{dt} = \beta i s - \mu i$$
; $\frac{ds}{dt} = \delta r - \beta i s$; $\frac{dr}{dt} = \mu i - \delta r$; $i + s + r = 1$

Cannot be solved analytically!

Many other epidemic models have also been proposed to model the spread of particular types of diseases. For example, extra states can be introduced such as an "exposed" state that represents people who have caught a disease but whose infection has not yet developed to the point where they can pass it on to others; An initial immune state coming before the susceptible state, often used to represent the maternally derived immunity that newborn babies possess, etc.

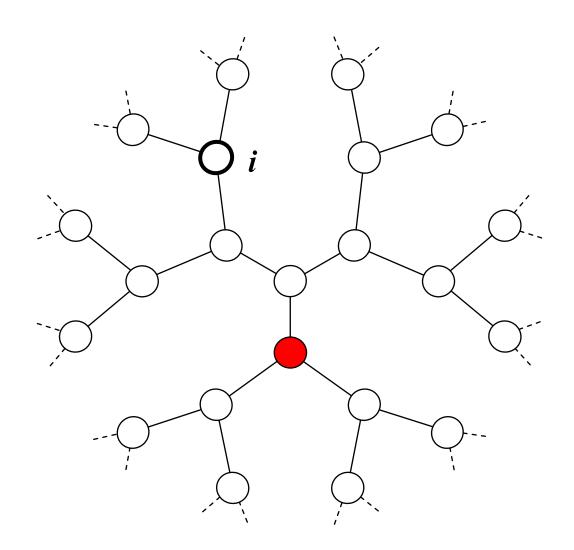
Up to now, we have assumed that each *I* meets one *S* randomly.

For a homogeneous social network with average degree < k >, each I meets on average < k > S: each link is a try to infect a neighbor => We get the same as before, but with, $R_0 = \frac{\beta}{\mu} < k >$

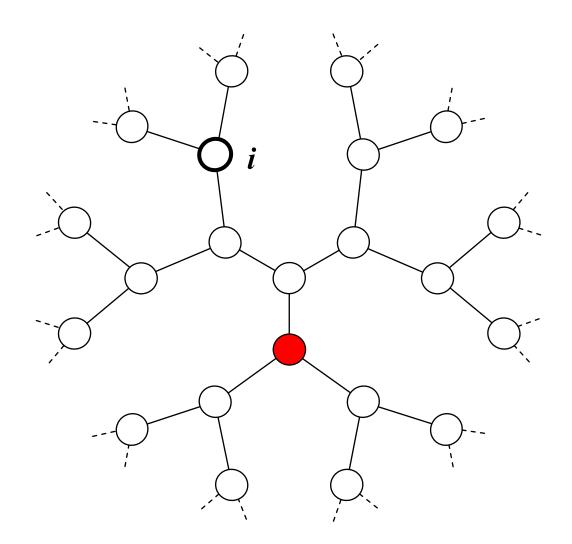
BUT: what happens if the network is heterogeneous?

Simple illustration of the SIR model on network

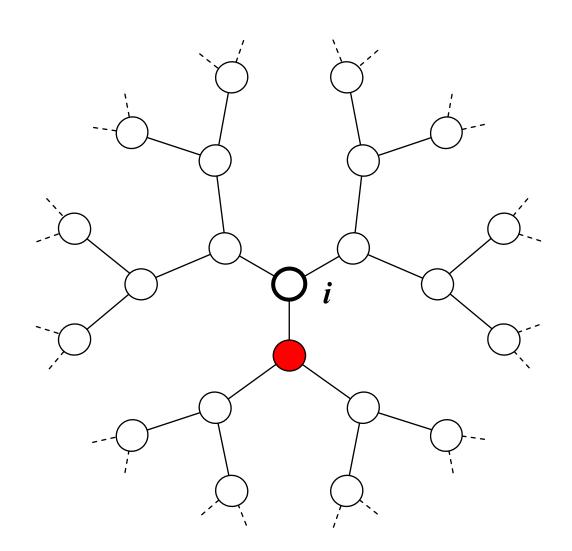
Either node *i* is *S*, but it's in a "pool" of *S*s



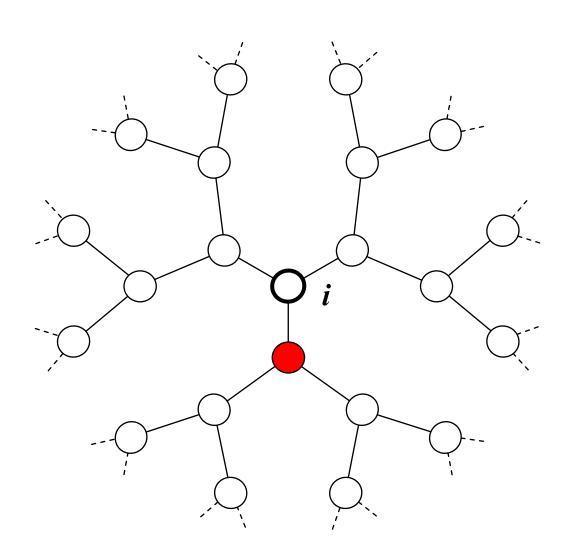
- Either node *i* is *S*, but it's in a "pool" of Ss
 - no contribution to the dynamics



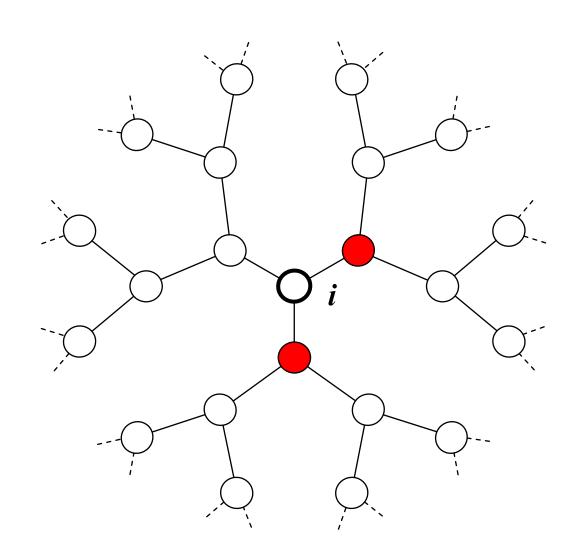
- Either node *i* is *S*, but it's in a "pool" of Ss
 - no contribution to the dynamics
- Or i is S and near an I neighbor:



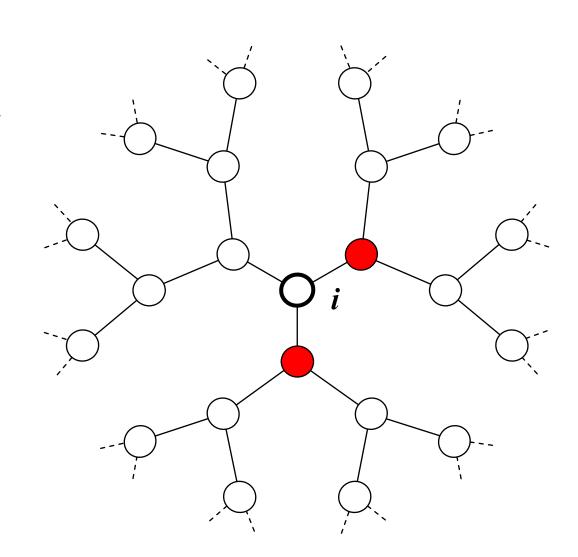
- Either node *i* is *S*, but it's in a "pool" of Ss
 - no contribution to the dynamics
- Or i is S and near an I neighbor:
 - the other 2 are susceptible



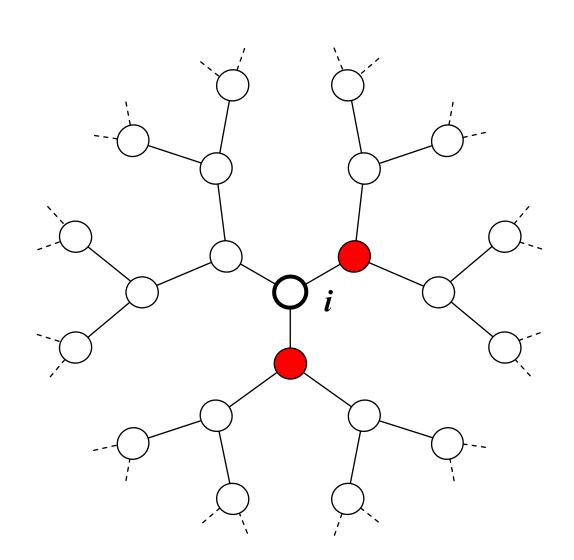
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- If the epidemic started in at least 2 places:



- Either node *i* is *S*, but it's in a "pool" of Ss
 - no contribution to the dynamics
- Or i is S and near an I neighbor:
 - the other 2 are susceptible
- If the epidemic started in at least 2 places:
 - *i* escapes infection independently

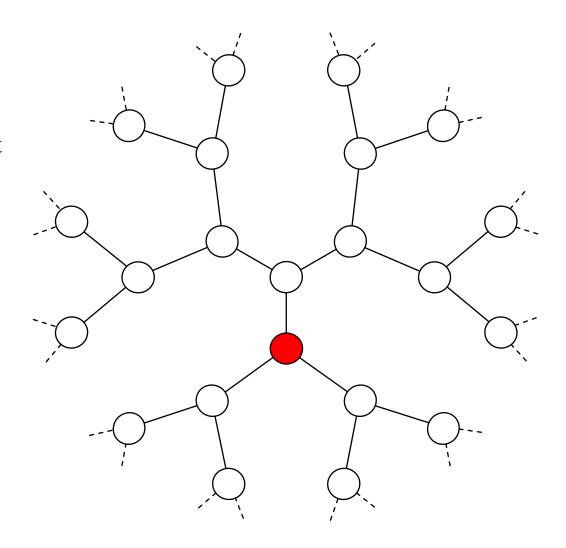


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 - no contribution to the dynamics
- Or i is S and near an I neighbor:
 - the other 2 are susceptible
- If the epidemic started in at least 2 places:
 - *i* escapes infection independently
 - S neighbor is unaffected

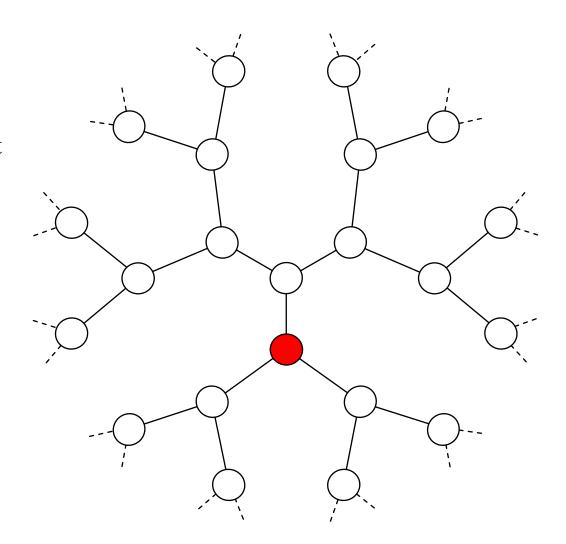


Simple illustration of the SIS model on network

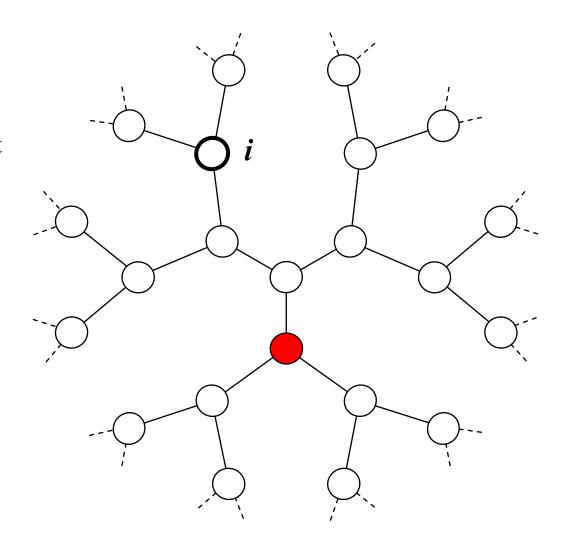
➤ Knowing that *i* is S is not enough to tell something about its surrounding



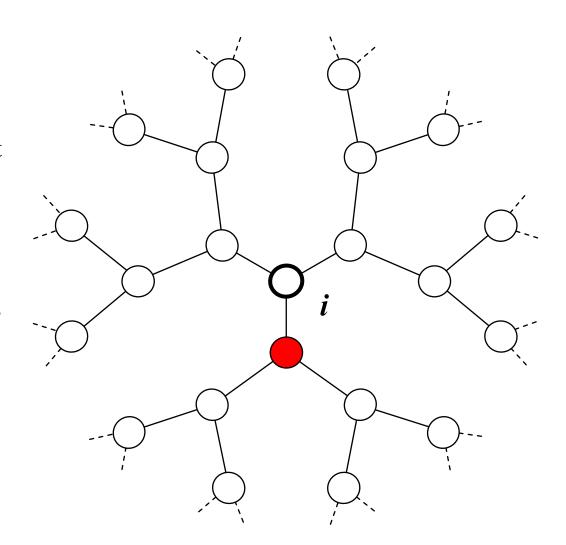
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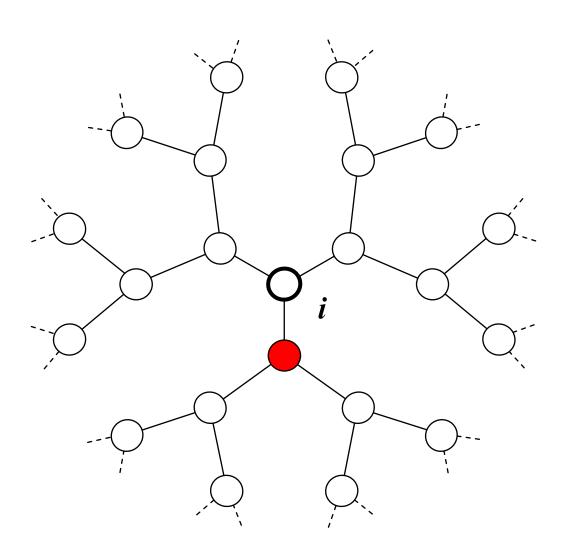
- Knowing that i is S is not enough to tell something about its surrounding
- As for SIR, if *i* has never been infected, it's:
 - either surrounded by S



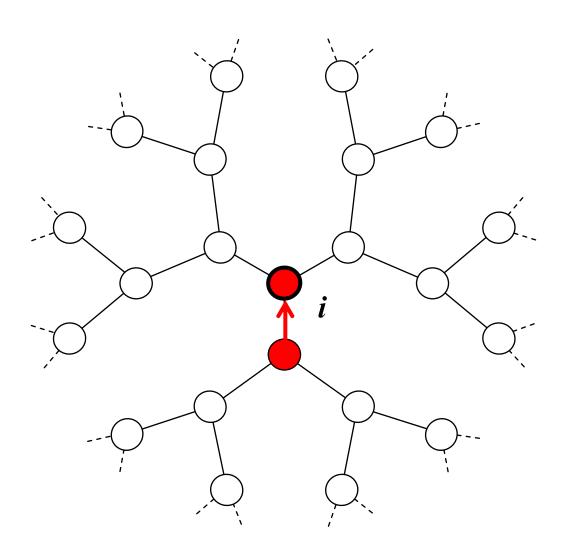
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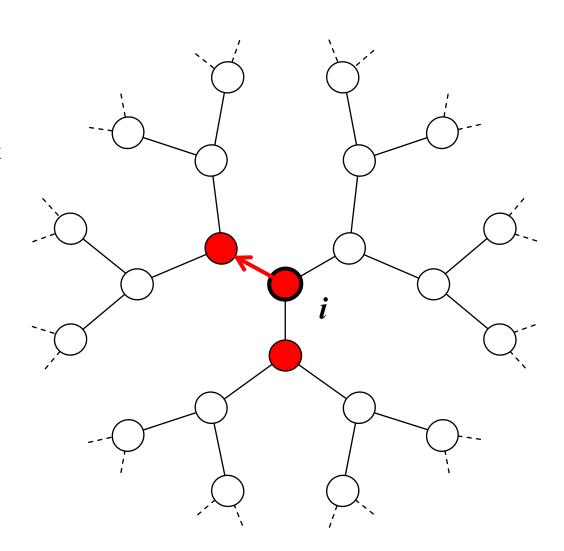
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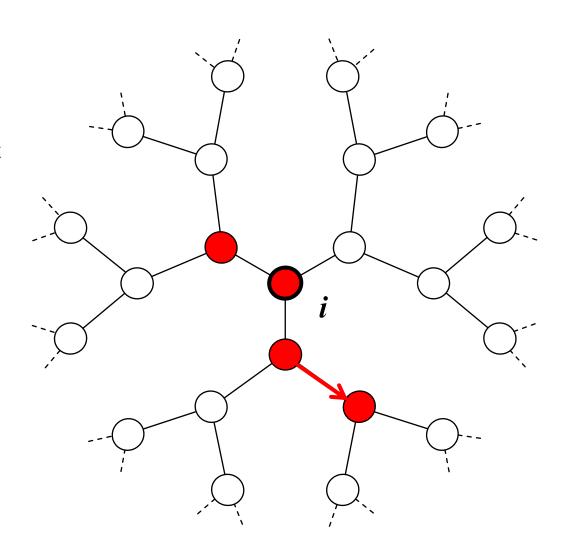
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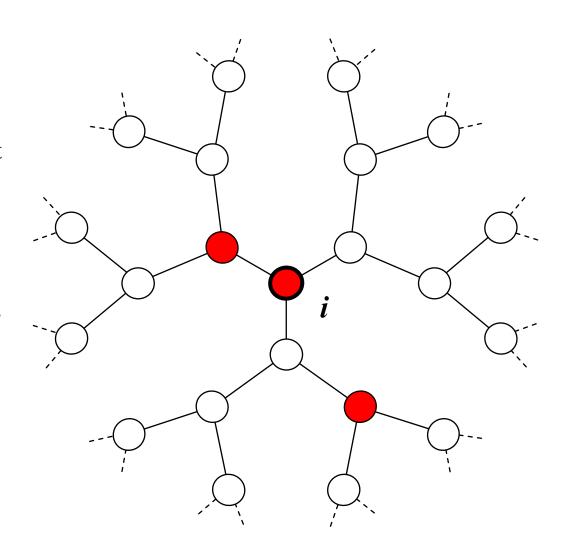
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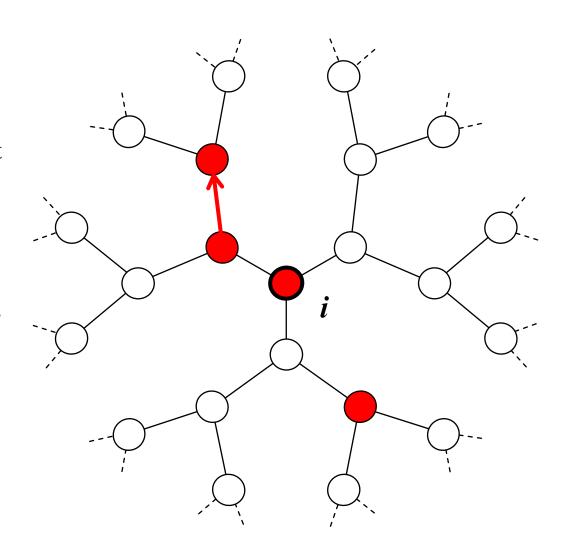
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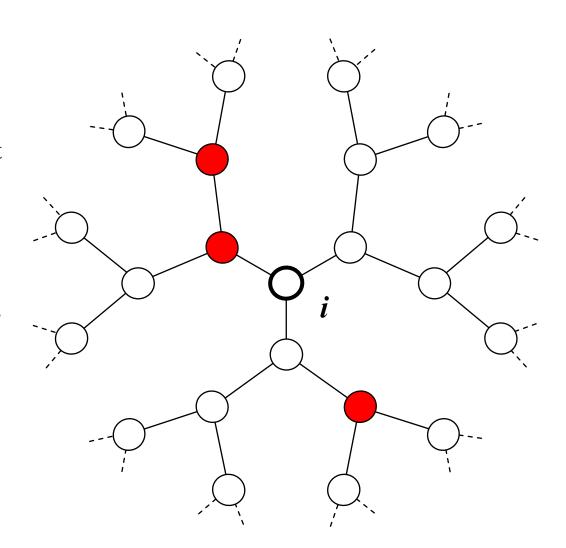
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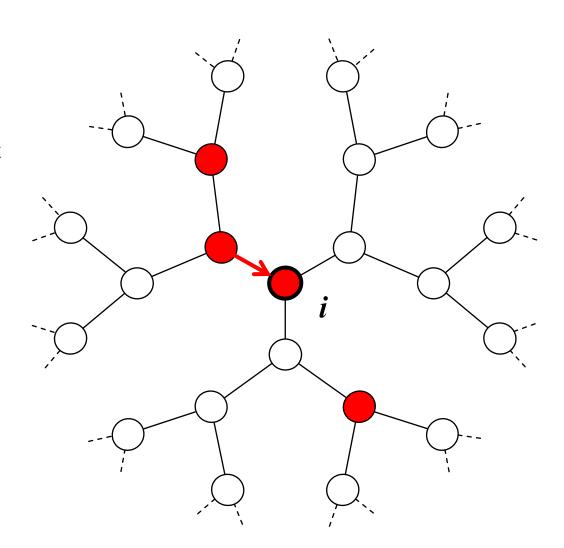
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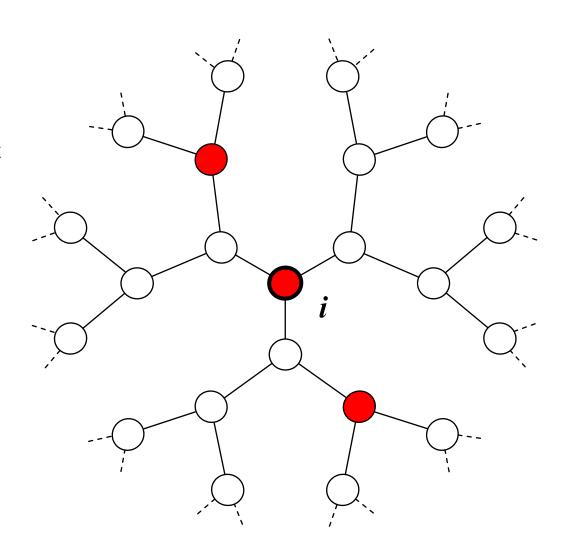
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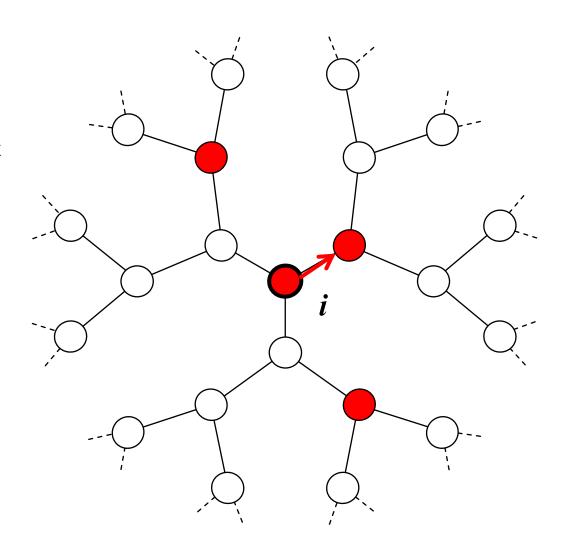
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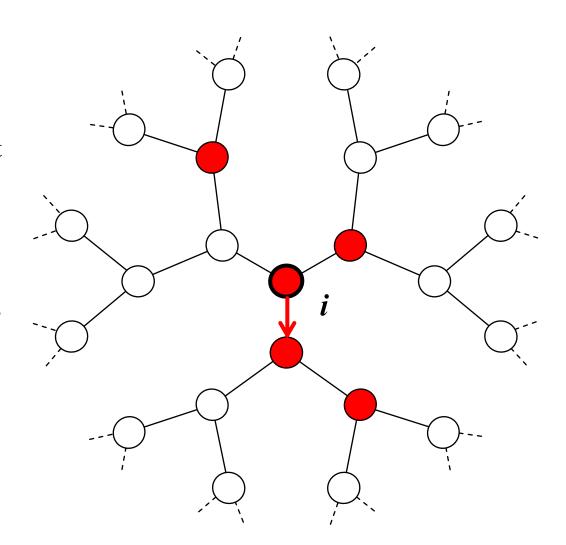
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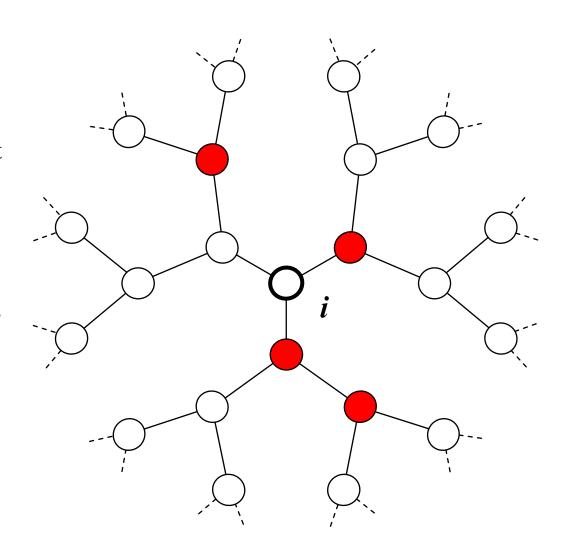
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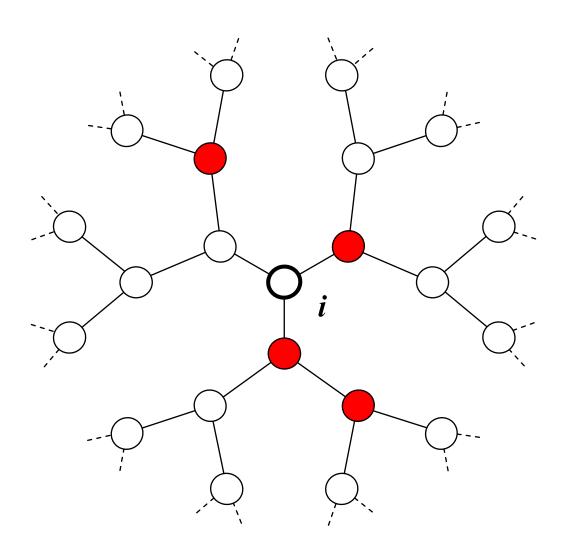
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- Unlike SIR:
 - it's surrounding depends on how many times it has recovered

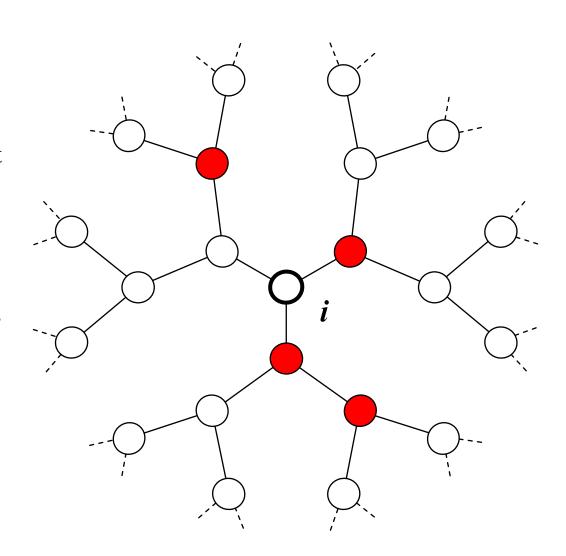


Simple illustration of the SIS model on network

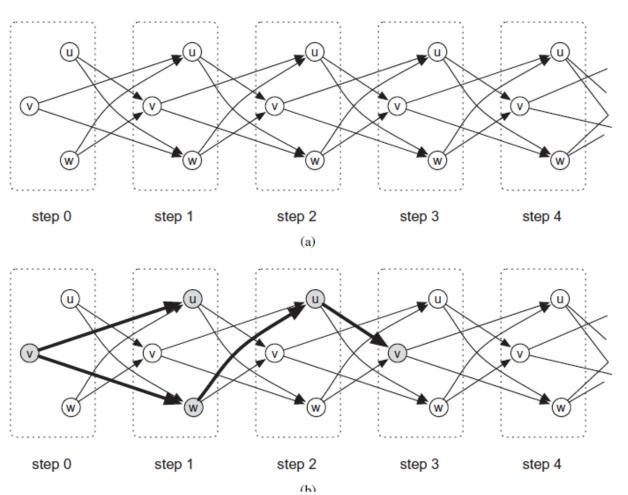
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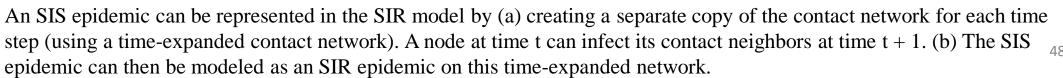


We need to count reinfections!



Connection between the SIR and SIS Model





The SI Model on Network:

$$\frac{ds_i}{dt} = -\beta s_i \sum_j A_{ij} i_j = -\beta s_i \sum_j A_{ij} (1 - s_j); \qquad i + s = 1$$

$$\frac{di_i}{dt} = \beta s_i \sum_j A_{ij} i_j = \beta (1 - i_i) \sum_j A_{ij} i_j$$

Assuming that i_i is small in the beginning, then in matrix form,

$$\frac{d\mathbf{i}}{dt} = \beta \mathbf{A}\mathbf{i} ,$$

where i is the vector with elements i_i . Write i as a linear combination of the eigenvectors of the adjacency matrix

$$\boldsymbol{i}(t) = \sum_{r=1}^{N} a_r(t) \boldsymbol{v}_r ,$$

where v_r is the eigenvector with eigenvalue κ_r .

The SI Model on Network:

$$\frac{d\mathbf{i}}{dt} = \sum_{r=1}^{N} \frac{da_r}{dt} \mathbf{v}_r = \beta \mathbf{A} \sum_{r=1}^{N} a_r(t) \mathbf{v}_r = \beta \sum_{r=1}^{N} \kappa_r a_r(t) \mathbf{v}_r$$

$$\Rightarrow \frac{da_r}{dt} = \kappa_r a_r$$

or,

$$a_r(t) = a_r(0)e^{\beta\kappa_r t}$$

$$i = \sum_{r=1}^{N} a_r(0)e^{\beta\kappa_r t}v_r$$

The fastest growing term in this expression is the term corresponding to the largest eigenvalue κ_1 . Therefore,

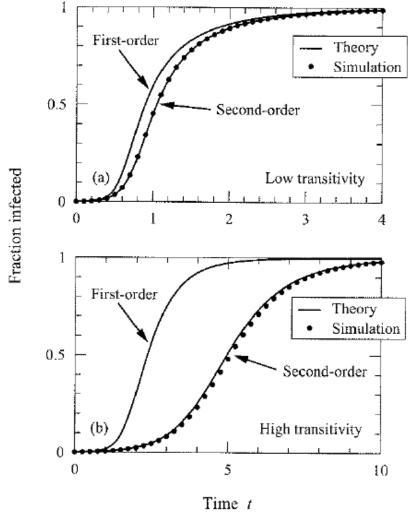
$$i(t) \sim e^{\beta \kappa_1 t} v_1$$

The number of infected individuals grows exponentially, same as in the fully mixed version.

The SI Model on Network:

Comparison of theory and simulation for the S1 model on two different networks.

(a) The fraction of infected individuals as a function of time on the giant component of a network with *low* transitivity (i.e., low clustering coefficient), calculated by numerical solution of the differential equations for the first- and second-order moment closure methods, and by direct simulation. (b) The same comparison for a network with high transitivity. The networks have one million vertices each and the transmission rate is $\beta = 1$ in all cases. Simulation results were averaged over 500 runs.



The SI Model on Network: Pair Approximation

We want to include higher order moments in the equation to get better approximation. Denote $\langle s_i \rangle$ to be the average probability that vertex i is susceptible, and $\langle s_i i_j \rangle$ the average probability that i is susceptible and j is infected at the same time. Then

$$\frac{d\langle s_i \rangle}{dt} = -\beta \sum_i A_{ij} \langle s_i i_j \rangle$$

and,

$$\frac{d\langle s_i i_j \rangle}{dt} = \beta \sum_{k(\neq i)} A_{jk} \langle s_i s_j i_k \rangle - \beta \sum_{l(\neq j)} A_{il} \langle i_l s_i i_j \rangle - \beta \langle s_i i_j \rangle$$

We now use the pair approximation method by using the Bayes theorem for probabilities

$$\langle s_i s_j i_k \rangle = P(i, j \in S, k \in I) = P(i, j \in S)P(k \in I | i, j \in S)$$

Assuming the state of k to be independent of the state of i

$$P(k \in I | i, j \in S) = P(k \in I | j \in S) = \frac{P(j \in S, k \in I)}{P(j \in S)} = \frac{\langle s_j i_k \rangle}{\langle s_i \rangle}$$

The SI Model on Network: Pair Approximation

We then have

$$\langle s_i s_j i_k \rangle = \frac{\langle s_i s_j \rangle \langle s_j i_k \rangle}{\langle s_j \rangle}$$

Similarly,

$$\langle i_{l}s_{i}i_{j}\rangle = \frac{\langle i_{l}s_{i}\rangle\langle s_{i}i_{j}\rangle}{\langle s_{i}\rangle}$$

$$\frac{d\langle s_{i}i_{j}\rangle}{dt} = \beta \frac{\langle s_{i}s_{j}\rangle}{\langle s_{j}\rangle} \sum_{k(\neq i)} A_{jk}\langle s_{j}i_{k}\rangle - \beta \frac{\langle s_{i}i_{j}\rangle}{\langle s_{i}\rangle} \sum_{l(\neq j)} A_{il}\langle s_{i}i_{l}\rangle - \beta \langle s_{i}i_{j}\rangle$$
Using $\langle s_{i}s_{i}\rangle = \langle s_{i}(1-i_{i})\rangle = \langle s_{i}\rangle - \langle s_{i}i_{i}\rangle$,

$$\frac{d\langle s_{i}i_{j}\rangle}{dt} = \beta \frac{\langle s_{i}\rangle - \langle s_{i}i_{j}\rangle}{\langle s_{j}\rangle} \sum_{k(\neq i)} A_{jk}\langle s_{j}i_{k}\rangle - \beta \frac{\langle s_{i}i_{j}\rangle}{\langle s_{i}\rangle} \sum_{l(\neq j)} A_{il}\langle s_{i}i_{l}\rangle - \beta \langle s_{i}i_{j}\rangle$$

The SI Model on Network: Pair Approximation

Define p_{ij} to be the conditional probability that j is infected given that i is not

$$p_{ij} = P(j \in I | i \in S) = \frac{P(i \in S, j \in I)}{P(i \in S)} = \frac{\langle s_i i_j \rangle}{\langle s_i \rangle}$$

$$\Rightarrow \frac{dp_{ij}}{dt} = \frac{d}{dt} \left(\frac{\langle s_i i_j \rangle}{\langle s_i \rangle} \right) = \frac{1}{\langle s_i \rangle} \frac{d\langle s_i i_j \rangle}{dt} - \frac{\langle s_i i_j \rangle}{\langle s_i \rangle^2} \frac{d\langle s_i \rangle}{dt}$$

$$= \beta \left(1 - \frac{\langle s_i i_j \rangle}{\langle s_i \rangle} \right) \sum_{k(\neq i)} A_{jk} \frac{\langle s_j i_k \rangle}{\langle s_j \rangle} - \beta \frac{\langle s_i i_j \rangle}{\langle s_i \rangle} \sum_{l(\neq j)} A_{il} \frac{\langle s_i i_l \rangle}{\langle s_i \rangle} - \beta \frac{\langle s_i i_j \rangle}{\langle s_i \rangle} + \beta \frac{\langle s_i i_j \rangle}{\langle s_i \rangle} \sum_{l} A_{il} \frac{\langle s_i i_l \rangle}{\langle s_i \rangle}$$

$$= \beta \left(1 - p_{ij} \right) \sum_{k(\neq i)} A_{jk} p_{jk} - \beta p_{ij} \sum_{l(\neq j)} A_{il} p_{il} - \beta p_{ij} + \beta p_{ij} \sum_{l} A_{il} p_{il}$$

Simplifying, we obtain

$$\frac{dp_{ij}}{dt} = \beta (1 - p_{ij}) [-p_{ij} + \sum_{k(\neq i)} A_{jk} p_{jk}]$$

Since all but one of the terms in the two sums over l cancel out and $A_{ij} = 1$ (i and j are neighbors).54

The SI Model on Network: Pair Approximation

$$\frac{d\langle s_i \rangle}{dt} = -\beta \langle s_i \rangle \sum_j A_{ij} p_{ij}$$

Giving a solution

$$\langle s_i(t)\rangle = \langle s_i(0)\rangle e^{-\beta\sum_j A_{ij}\int_0^t p_{ij}(t')dt'}$$

The SI Model on Network: Degree-based Approximation

In this approximation, we write: $i = \sum_k P(k)i_k$; $s = \sum_k P(k)s_k$. The evolution equation becomes

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

 Θ_k is the density of infected neighbors of vertices of degree k. For networks without degree correlations,

$$\Theta_k(t) = \Theta(t) = \frac{\sum_{k'} (k'-1)P(k')i_{k'}(t)}{\langle k \rangle}; \qquad \langle k \rangle = \sum_{k'} k'P(k')$$

In the initial stage, $i_k(0) = i_0 \approx 0$, neglecting $O(i^2)$

$$\frac{di_{k}(t)}{dt} = \beta k \Theta_{k}(t) ; \qquad \frac{d\Theta(t)}{dt} = \beta \left(\frac{\langle k^{2} \rangle}{\langle k \rangle} - 1\right) \Theta(t) \longrightarrow \Theta(t) = Ce^{t/\tau} ; \quad C = i_{0} \frac{\langle k \rangle - 1}{\langle k \rangle}.$$

$$\Rightarrow i_{k}(t) = i_{0} \left[1 + \frac{k(\langle k \rangle - 1)}{\langle k^{2} \rangle - \langle k \rangle} (e^{t/\tau} - 1) \right] ; \qquad \tau = \frac{\langle k \rangle}{\beta(\langle k^{2} \rangle - \langle k \rangle)}.$$

The SI Model on Network: Degree-based Approximation

Total average prevalence is given by

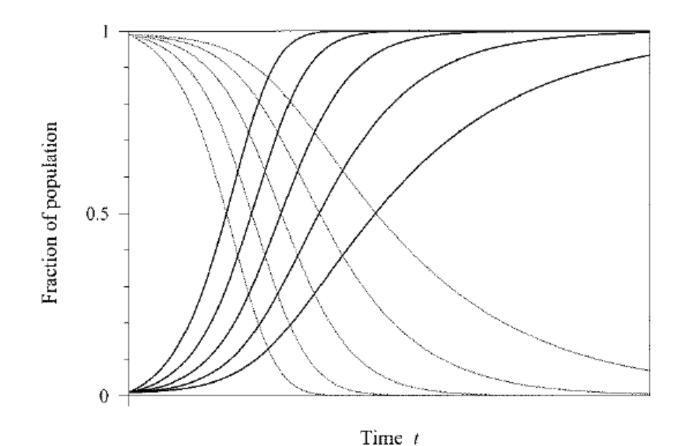
$$i(t) = \sum_{k} P(k)i_{k}(t) = i_{0} \left[1 + \frac{\langle k \rangle^{2} - \langle k \rangle}{\langle k^{2} \rangle - \langle k \rangle} (e^{t/\tau} - 1) \right]$$

The graph heterogeneity is measured by the heterogeneity ratio $\kappa = \frac{\langle k^2 \rangle}{\langle k \rangle}$. In homogeneous network with a Poisson degree distribution, $\kappa = \langle k \rangle + 1$. We recover the result $\tau = (\beta \langle k \rangle)^{-1}$, corresponding to the homogeneous mixing hypothesis. In networks with very heterogeneous connectivity patterns, κ is very large and the outbreak time scale τ is very small, signaling a very fast diffusion of the infection. For example, in scale-free networks characterized by a degree exponent $2 < \gamma \le 3$ we have that $\kappa \sim \langle k^2 \rangle \to \infty$ for networks of size $N \to \infty$.

The SI Model on Network:

Fractions of susceptible and infected vertices of various degrees in the SI model.

The various curves show the fraction of vertices of degree k that are susceptible (gray) and infected (black) as a function of time for k = 1,2,4,8, and 16. The highest values of k give the fastest changing (leftmost) curves and the lowest values the slowest changing. The curves were calculated by integrating the equation numerically with $\beta = 1$ and a Poisson degree distribution with mean degree four.



The SIR and SIS Model on Network:

For both models, the evolution equation is

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

with

$$\begin{cases} s_k(t) = 1 - i_k(t) ; & \text{(for SIS model)} \\ s_k(t) = 1 - i_k(t) - r_k(t) ; & \text{(for SIR model)} \end{cases}$$

where $r_k(t)$ is the density of removed individuals of degree k. By using the same approximation, the time scale for the SIR is

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

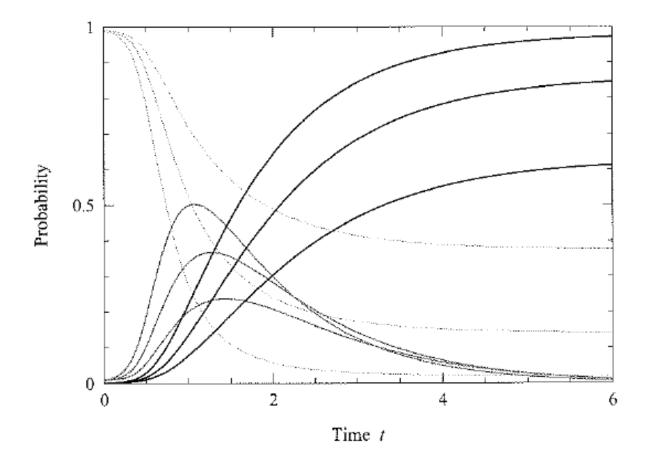
To have an epidemic outbreak, the basic condition $\tau > 0$ gives

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

The SIR and SIS Model on Network:

Fractions of susceptible, infected, and recovered vertices of various degree in the SIR model.

The fraction of vertices of degree k that are susceptible (light gray), infected (darker gray), and recovered (black) as a function of time for k = 1,2,4 and $\beta = \gamma = 1$ on a network with an exponential degree distribution with $\lambda = 0.2$. The highest values of k give the fastest growing numbers of infected and recovered vertices and the lowest values the slowest growing.



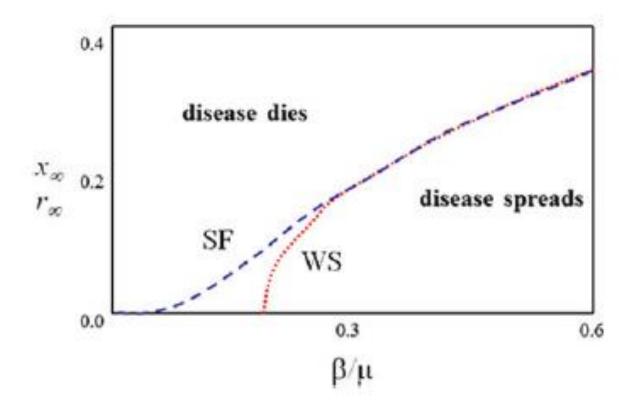


Illustration of the total prevalence of disease in both the SIR and SIS models in networks with homogeneous (Watts-Strogatz) and heterogeneous (Scale-Free) degree distributions

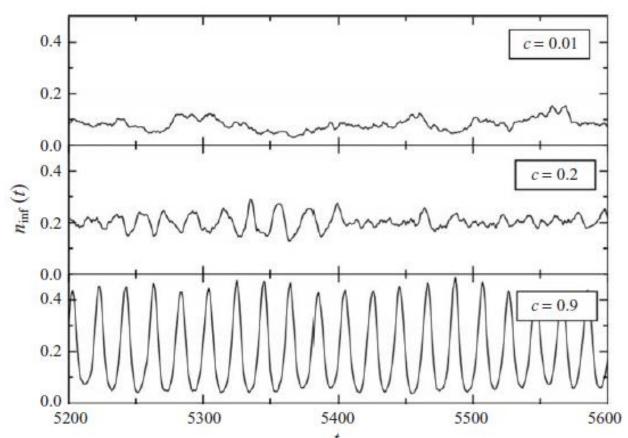
Synchronization in Epidemic Spreading (some remarks)

There is a tendency for epidemics of certain diseases to synchronize across a population, sometimes producing strong oscillations in the number of affected individuals over time. Well-known examples are diseases such as measles and syphilis. In the case of syphilis, cycles in the prevalence of syphilis across the United States over the past few decades have traditionally been attributed to large-scale societal changes, including changes in sexual moves and other forces. However, recent research has shown that oscillations and synchronization over time can in fact result largely from the contagion dynamics of the disease itself. To produce oscillation patterns, one can extend the *SIR* and *SIS* models to the so-called *SIRS* model.

Nicholas C. Grassly, Christophe Fraser, and Geoffrey P. Garnett. *Host immunity and synchronized epidemics of syphilis across the United States*. Nature, 433:417–421, January 2005.

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Synchronization in Epidemic Spreading (some remarks)



These plots depicting the number of infected people $(n_{inf}(t))$ over time by SIRS epidemics in networks with different proportions of long-range links. Here, c denotes the fraction of long-range links, we see an absence of oscillations for small c (c = 0.01), wide oscillations for large c (c = 0.9), and a transitional region (c = 0.2) where oscillations intermittently appear and then disappear. (Marcelo Kuperman and Guillermo Abramson. *Small world effect in an epidemiological model*. Physical Review Letters, 86(13):2909-2912, March 2001.