

Dynamical Processes over Networks

Héctor Corrada Bravo

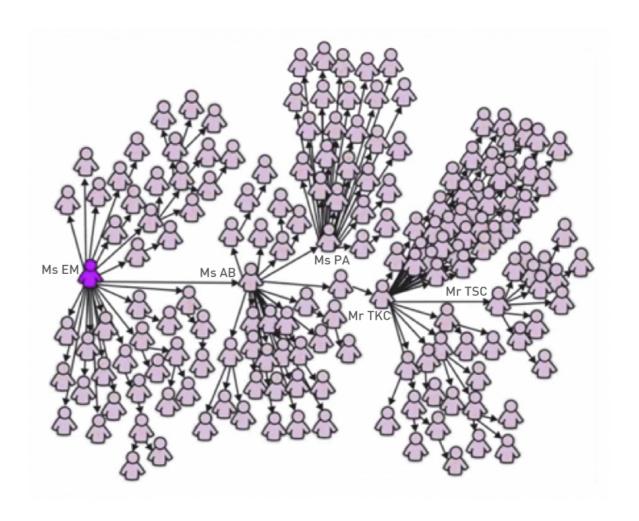
University of Maryland, College Park, USA CMSC828O 2019-10-02



Modeling epidemics over Networks

First analysis of dynamical processes over networks

Will let us exercise some of the ways of thinking about these processes



Modeling epidemics over Networks

Questions

Are there network properties that predict spread of infection?

Are certain network types more resilient to infection than others?

If we can intervene (vaccinate) are there nodes in the network that are more effective to vaccinate?

Modeling epidemics over Networks

Questions

Are there network properties that predict spread of infection?

Are certain network types more resilient to infection than others?

If we can intervene (vaccinate) are there nodes in the network that are more effective to vaccinate?

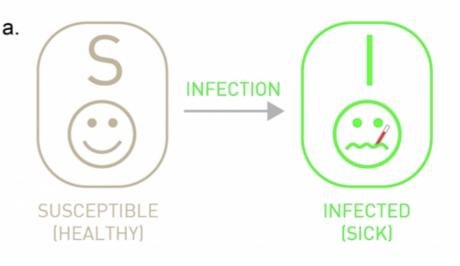
We'll start by looking at spread over non-networked populations

Susceptibility and infection (SI model)

Individuals in the population can be in two states

An infected individual can infect *any* susceptible individual they are in contact with

If we start (t=0) with some number of infected individuals (i_0). How many infected individuals are there at time t?



$$rac{d}{dt}I(t)=eta\langle k
anglerac{S(t)}{N}I(t)$$

 $\langle k
angle$, average number of contacts per individual in one time step

 β , "rate" probability an I infects an S upon contact

$$rac{d}{dt}I(t)=eta\langle k
anglerac{S(t)}{N}I(t)$$

 $\langle k
angle$, average number of contacts per individual in one time step

 β , "rate" probability an I infects an S upon contact

$$rac{di}{dt}=eta\langle k
angle i(1-i)$$

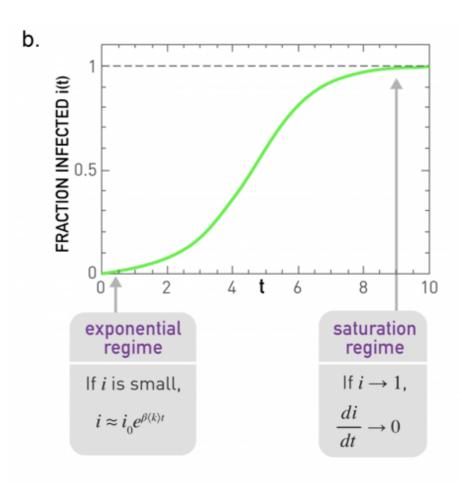
Fraction of infected individuals in population

$$i(t) = rac{i_0 e^{eta \langle k
angle t}}{1 - i_0 + i_0 e^{eta \langle k
angle t}}$$

Characteristic time (t s.t.

$$i(t) = 1/e \approx .36$$
)

$$au = rac{1}{eta \langle k
angle}$$



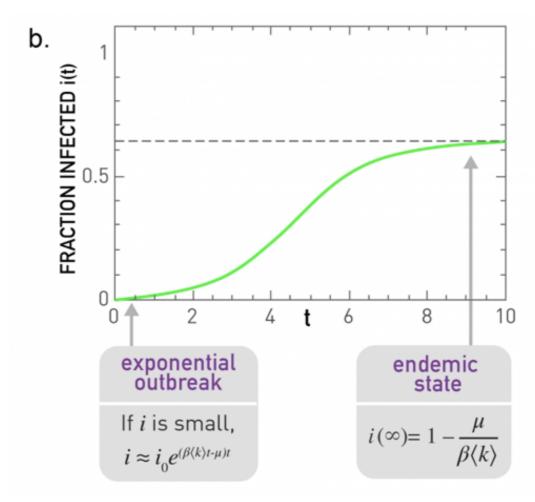
Infection ends (recovery), individual becomes susceptible again



$$rac{di}{dt} = eta \langle k
angle i (1-i) - \mu i$$

 μ - recovery rate

$$i(t) = \left(1 - rac{\mu}{eta\langle k
angle}
ight) imes rac{Ce^{(eta\langle k
angle - \mu)t}}{1 + Ce^{(eta\langle k
angle - \mu)t}}$$

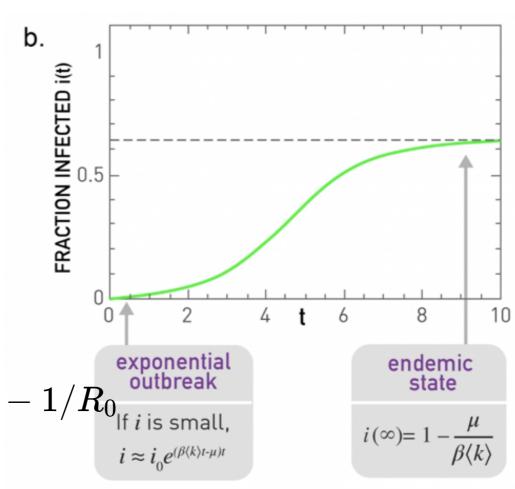


Endemic State

Pathogen persists in population after saturation

$$R_0 = rac{eta\langle k
angle}{\mu} > 1$$

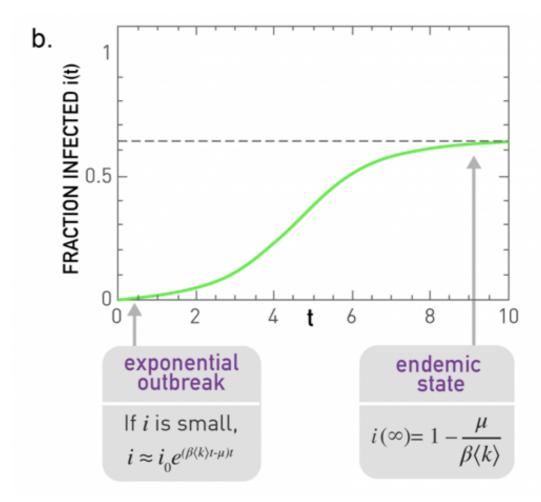
$$i(\infty)=1-rac{\mu}{eta\langle k
angle}=1$$



Disease-free State

Pathogen disappears from population

$$R_0=rac{eta\langle k
angle}{\mu}<1$$
 $i(\infty)=0$



Basic Reproductive Number

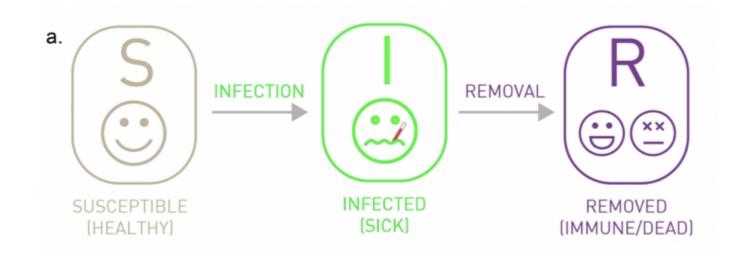
$$R_0=rac{eta\langle k
angle}{\mu}$$

Characteristic Time

$$au = rac{1}{\mu(R_0-1)}$$

SIR model

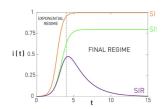
Individuals **removed** after infection (either death or immunity)

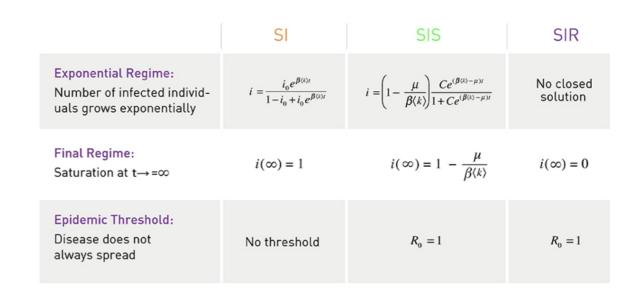


SIR model

$$rac{di}{dt}=eta\langle k
angle i(1-r-i)-\mu i$$
 and $\frac{dr}{dt}=\mu i$ and $\frac{ds}{dt}=-eta\langle k
angle i(1-r-i)$ and $\frac{ds}{dt}=-eta\langle k
angle i(1-r-i)$

Summary





Epidemic processes over networks (SI)

Consider node i in network:

 $s_i(t)$ average probability node i is *susceptible* at time t

 $x_i(t)$ average probability node i is *infected* at time t

Epidemic processes over networks (SI)

$$rac{ds_i}{dt} = -s_i eta \sum_{j=1}^N a_{ij} x_j$$

$$rac{dx_i}{dt} = s_i eta \sum_{j=1}^N a_{ij} x_j$$

Epidemic processes over networks (SI)

$$rac{ds_i}{dt} = -s_i eta \sum_{j=1}^N a_{ij} x_j$$

$$rac{dx_i}{dt} = s_i eta \sum_{j=1}^N a_{ij} x_j$$

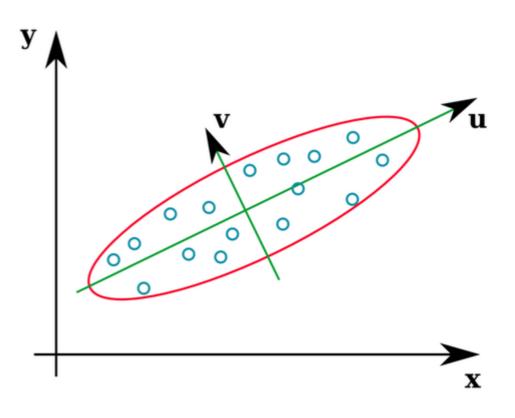
For large N, and early t

$$\frac{dx}{dt} = \beta Ax$$

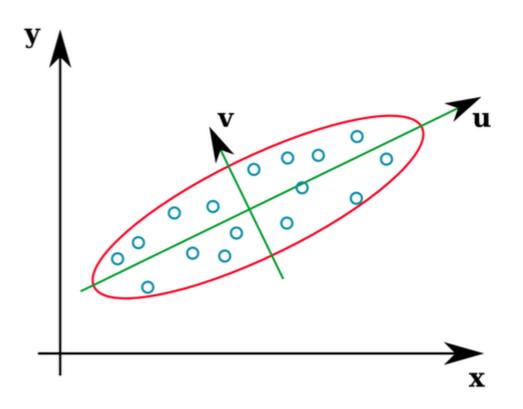
$$A = V^T \Lambda V$$

Any quantity x_i over nodes in the graph can be written as

$$\mathbf{x} = \sum_{r=1}^N c_r \mathbf{v}_r$$



$$A \mathbf{v}_r = \lambda_r \mathbf{v}_r$$
 $\lambda_1 \geq \lambda_2 \geq \cdots \geq \lambda_N$



Let's revisit centrality

$$\mathbf{x}(t) = A^t \mathbf{x}(0) = \sum_{r=1}^N c_r A^t \mathbf{v}_r$$

Then

$$\mathbf{x}(t) = \sum_{r=1}^N c_r \lambda_r^t \mathbf{v}_r = \lambda_1^t \sum_{i=1}^N c_r igg(rac{\lambda_r}{\lambda_1}igg)^t \mathbf{v}_r$$

As t grows, first term dominates

$$\mathbf{x}(t) = c_1 \lambda_1^t \mathbf{v}_1$$

So set centrality \mathbf{x} to be proportional to first *eigenvector* \mathbf{v}_1

As t grows, first term dominates

$$\mathbf{x}(t) = c_1 \lambda_1^t \mathbf{v}_1$$

So set centrality \mathbf{x} to be proportional to first *eigenvector* \mathbf{v}_1

In which case x=Ax if $\mathbf{x}=rac{1}{\lambda_1}\mathbf{v}_1$ as desired

 $\mathbf{x}(t)$ average probability each node is infected at time t

$$rac{d\mathbf{x}}{dt} = eta A \mathbf{x}$$

Can write as

$$\mathbf{x}(t) = \sum_{r=1}^N c_r(t) \mathbf{v}_r$$

$$egin{array}{lll} rac{d\mathbf{x}}{dt} &=& \sum_{r=1}^{N} rac{dc_r}{dt} \mathbf{v}_r \ &=& eta A \sum_{r=1}^{N} c_r(t) \mathbf{v}_r = eta \sum_{r=1}^{N} \lambda_r c_r(t) \mathbf{v}_r \end{array}$$

$$egin{array}{lll} rac{d\mathbf{x}}{dt} &=& \sum_{r=1}^{N} rac{dc_r}{dt} \mathbf{v}_r \ &=& eta A \sum_{r=1}^{N} c_r(t) \mathbf{v}_r = eta \sum_{r=1}^{N} \lambda_r c_r(t) \mathbf{v}_r \end{array}$$

Implying

$$rac{dc_r}{dt}=eta\lambda_r c_r$$

$$egin{array}{lll} rac{d\mathbf{x}}{dt} &=& \sum_{r=1}^{N} rac{dc_r}{dt} \mathbf{v}_r \ &=& eta A \sum_{r=1}^{N} c_r(t) \mathbf{v}_r = eta \sum_{r=1}^{N} \lambda_r c_r(t) \mathbf{v}_r \end{array}$$

Implying

$$rac{dc_r}{dt}=eta\lambda_r c_r$$

With solution

$$c_r(t) = c_r(0) e^{eta \lambda_r t}$$

As before, first term dominates so

$$\mathbf{x}(t) \sim e^{eta \lambda_1 t} \mathbf{v}_1$$

Eigen-centrality!

$$rac{d\mathbf{x}}{dt} = eta A\mathbf{x} - \mu\mathbf{x}$$

Similarily

$$\mathbf{x}(t) \sim e^{(eta \lambda_1 - \mu)t}$$

$$rac{d\mathbf{x}}{dt} = eta A\mathbf{x} - \mu\mathbf{x}$$

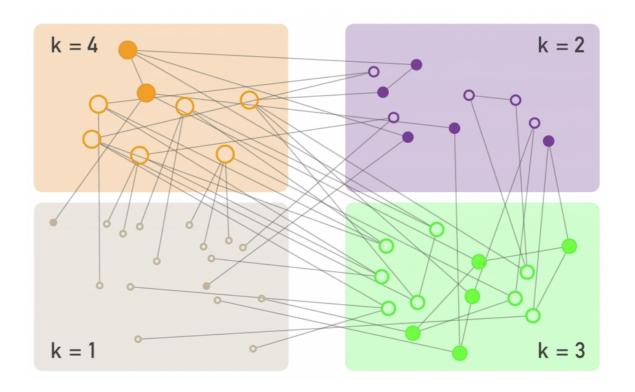
Similarily

$$\mathbf{x}(t) \sim e^{(eta \lambda_1 - \mu)t}$$

Is there an epidemic? Not if $R_0=rac{eta}{\mu}=rac{1}{\lambda_1}$

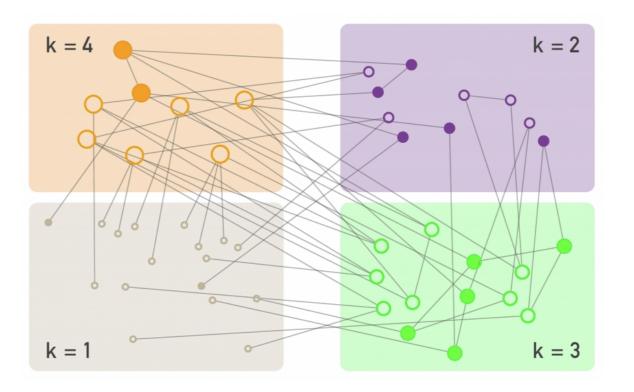
Degree Block Approximation

Assume that all nodes of the same degree are statistically equivalent



Degree Block Approximation

Assume that all nodes of the same degree are statistically equivalent



SI model

Fraction of nodes of degree k that are infected

$$i_k = rac{I_k}{N_k}$$

$$rac{di_k}{dt} = eta(1-i_k)k\Theta_k$$

With Θ_k the fraction of infected neighbors for a node of degree k

For early time and assuming no degree correlation

$$rac{di_k}{dt} = eta ki_0 rac{\langle k
angle - 1}{\langle k
angle} e^{t/ au^{SI}}$$

with

$$au^{SI} = rac{\langle k
angle}{eta(\langle k^2
angle - \langle k
angle)}$$

Characteristic time

$$au^{SI} = rac{\langle k
angle}{eta(\langle k^2
angle - \langle k
angle)}$$

For random networks $\langle k^2
angle = \langle k
angle (\langle k
angle + 1)$

$$au^{SI} = rac{1}{eta \langle k
angle}$$

Characteristic time

$$au^{SI} = rac{\langle k
angle}{eta(\langle k^2
angle - \langle k
angle)}$$

For power law network $\gamma \geq 3 \; \langle k^2
angle$ is finite, characteristic time is finite

Characteristic time

$$au^{SI} = rac{\langle k
angle}{eta(\langle k^2
angle - \langle k
angle)}$$

For power law network $\gamma \geq 3 \; \langle k^2
angle$ is finite, characteristic time is finite

For power law $\gamma < 3$, $\langle k^2 \rangle$ does not converge as $N \to \infty$ so characteristic time goes to 0

Model	Continuum Equation	τ	λ_{c}
SI	$\frac{di_k}{dt} = \beta \left[1 - i_k \right] k \theta_k$	$\frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)}$	0
SIS	$\frac{di_k}{dt} = \beta \left[1 - i_k \right] k \theta_k - \mu i_k$	$\frac{\langle k \rangle}{\beta \langle k^2 \rangle - \mu \langle k \rangle}$	$\frac{\langle k \rangle}{\langle k^2 \rangle}$
SIR	$\frac{di_k}{dt} = \beta s_k \theta_k - \mu i_k$ $s_k = 1 - i_l - r_k$	$\frac{\langle k \rangle}{\beta \langle k^2 \rangle - (\mu + \beta) \langle k \rangle}$	$\frac{\frac{1}{\langle k^2 \rangle}}{\frac{\langle k \rangle}{\langle k \rangle} - 1}$

Suppose a fraction g of nodes is immunized (i.e. resistant)

Rate of infection in SIR model changes from

$$\lambda = rac{eta}{\mu}$$

to

$$\lambda(1-g)$$

We can choose a fraction g_c such that rate of infection is below epidemic threshold

For a random network

$$g_c = 1 - rac{\mu}{eta} rac{1}{\langle k
angle + 1}$$

For a power-law network

$$g_c = 1 - rac{\mu}{eta} rac{\langle k
angle}{\langle k^2
angle}$$

For high $\langle k^2
angle$ need to immunize almost the entire population

Immunization can be more effective if performed selectively.

In power law contact networks, what is the effect of immunizing highdegree nodes?

Immunization can be more effective if performed selectively.

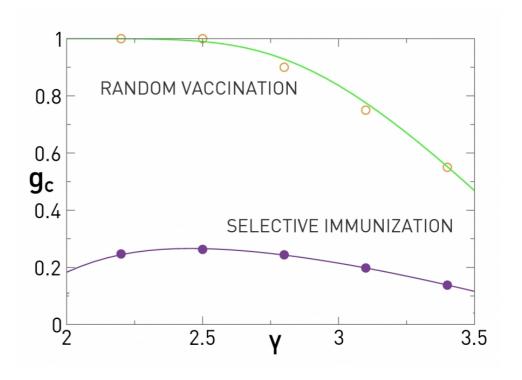
In power law contact networks, what is the effect of immunizing highdegree nodes?

First, how do you find them?

Idea: choose individuals at random, ask them to nominate a neighbor in contact graph

What is the expected degree of the nominated neighbor?

 $\propto kp_k$

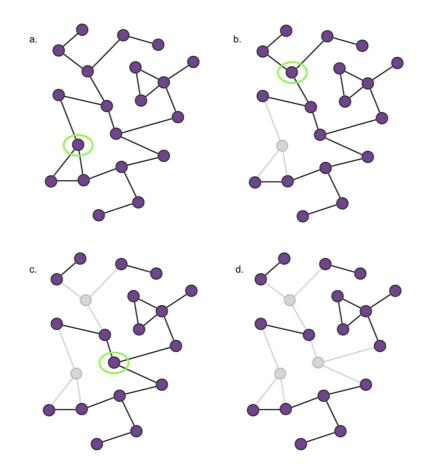


Summary

- Epidemic as first example of dynamical process over network
- Role of eigenvalue property in understanding epidemic spread
- Role of degree distribution (specifically scale) in understanding spread
- Role of high-degree nodes in robustness of networks to epidemics (immunization)

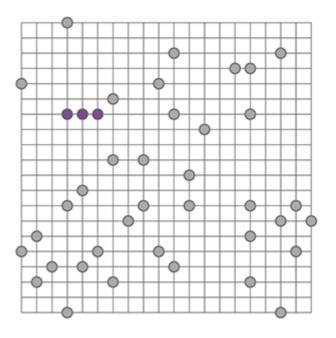
Network Robustness

What if we lose nodes in the network?



Let's look at a simplified network growth model (related to ER)

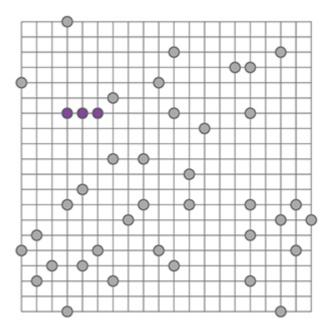
- Nodes are uniformly at random placed in the intersections of a regular grid
- Nodes in adjacent intersections are linked
- Keep track of largest component



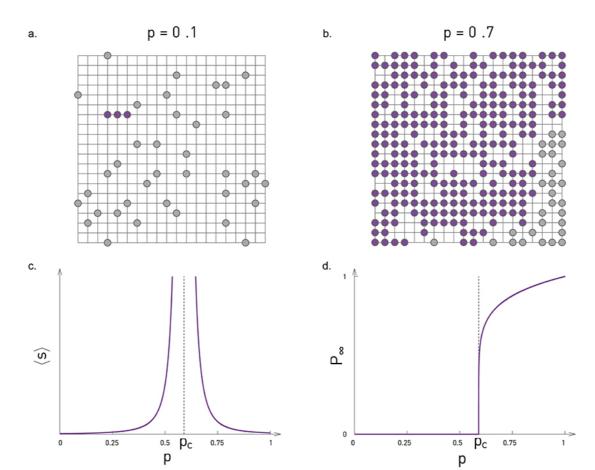
Let's look at a simplified network growth model (related to ER)

As nodes are added to the graph

- What is the expected size of the largest cluster?
- What is the average cluster size?



There is a critical threshold (percolation cluster p_c)



There is a critical threshold (percolation cluster)

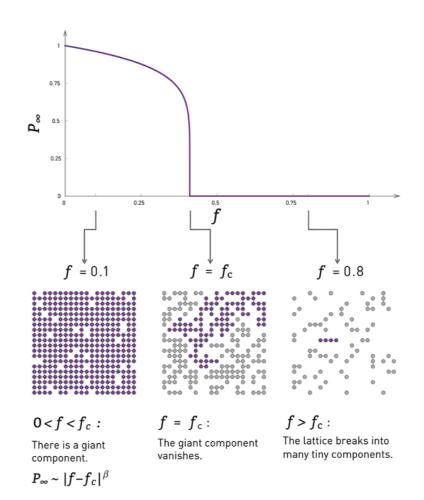
Average cluster size

$$\langle s
angle \sim \left| p - p_c
ight|^{-\gamma_p}$$

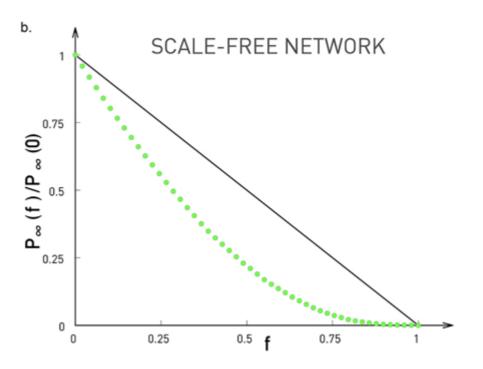
ullet Order parameter (P_{∞} probability node is in large cluster)

$$P_{\infty} \sim (p-p_c)^{eta_p}$$

Model failure as the reverse process (nodes are removed)



Similar modeling strategy applied to general networks



First question: is there a giant component?

Molloy-Reed Criterion: Yes, if

$$rac{\langle k^2
angle}{\langle k
angle}>2$$

For ER: $\langle k^2
angle = \langle k
angle (\langle k
angle + 1)$

For ER:
$$\langle k^2
angle = \langle k
angle (\langle k
angle + 1)$$

There is a large component if

$$rac{\langle k^2
angle}{\langle k
angle}=\langle k
angle>1$$

Good, coincides with what we know

Second question: if we model failure as before, when does the giant component disappear?

$$f_c = 1 - rac{1}{rac{\langle k^2
angle}{\langle k
angle} - 1}$$

Second question: if we model failure as before, when does the giant component disappear?

$$f_c = 1 - rac{1}{rac{\langle k^2
angle}{\langle k
angle} - 1}$$

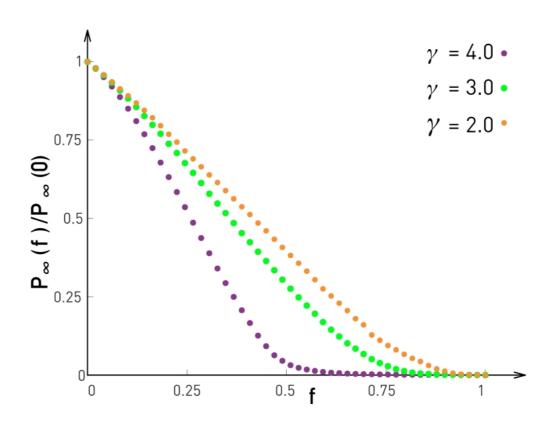
For ER:

$$f_c = 1 - rac{1}{\langle k
angle}$$

For power law networks

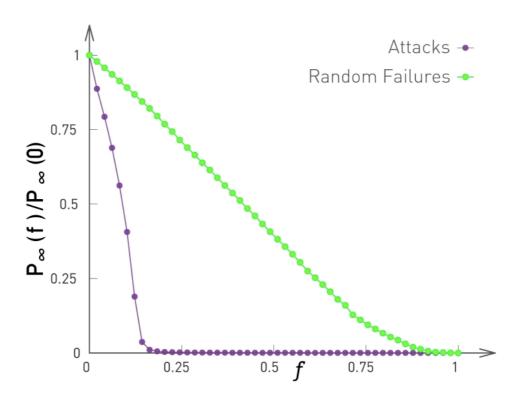
$$f_c = \begin{cases} 1 - \frac{1}{\frac{\gamma - 2}{3 - \gamma} k_{min}^{\gamma - 2} k_{max}^{3 - \gamma}} & 2 < \gamma < 3 \\ 1 - \frac{1}{\frac{\gamma - 2}{\gamma - 3} k_{min} - 1} & \gamma > 3 \end{cases}$$

For power law networks



Robustness to Attacks

What if node removal is targeted to hubs?

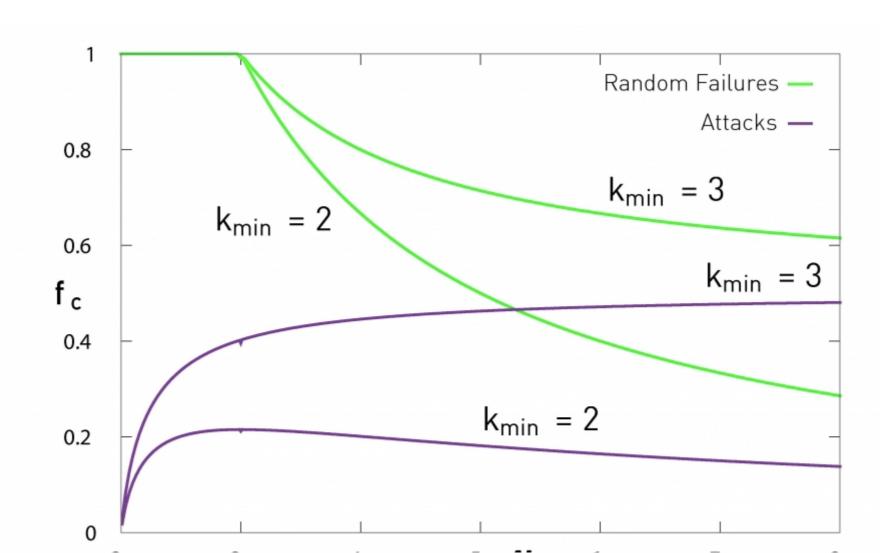


Robustness to Attacks

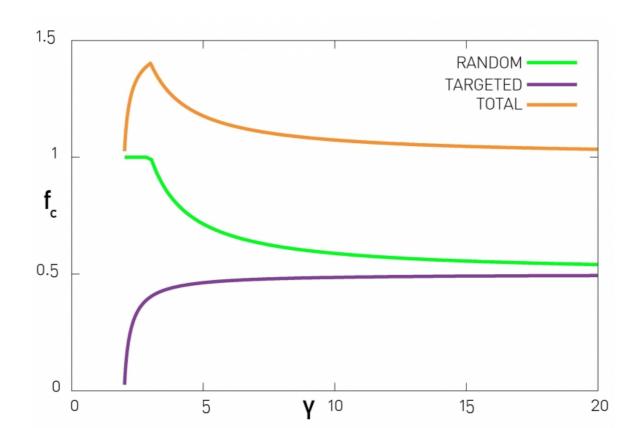
What if node removal is targeted to hubs?

$$f_c^{rac{2-\gamma}{1-\gamma}}=2+rac{2-\gamma}{3-\gamma}k_{min}(f_c^{rac{3-\gamma}{1-\gamma}}-1)$$

Robustness to Attacks



Hubs - robustness to random attacks No hubs - robustness to targeted attacks



Maximize

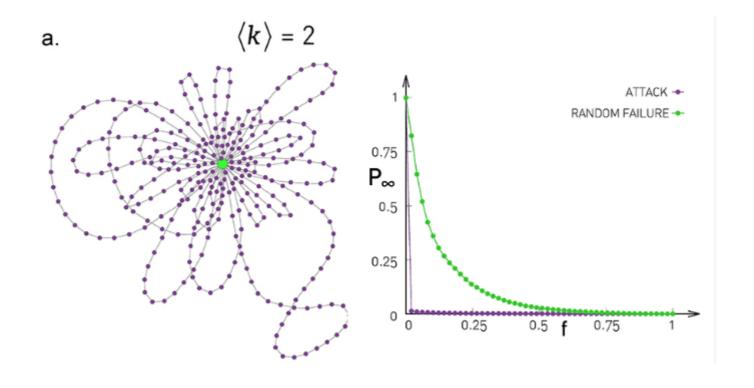
$$f_c^{tot} = f_c^{random} + f_c^{targeted}$$

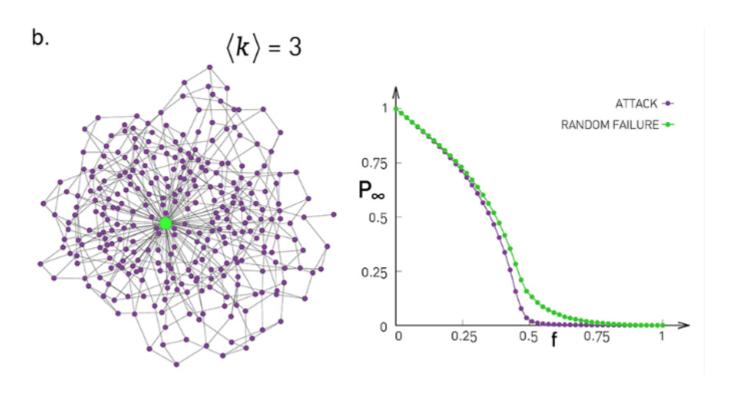
Maximize

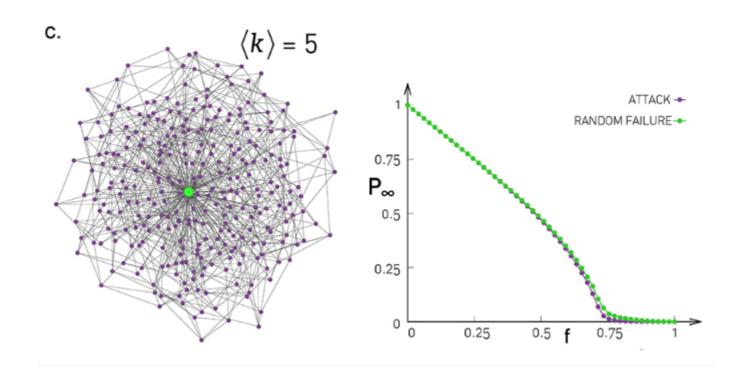
$$f_c^{tot} = f_c^{random} + f_c^{targeted}$$

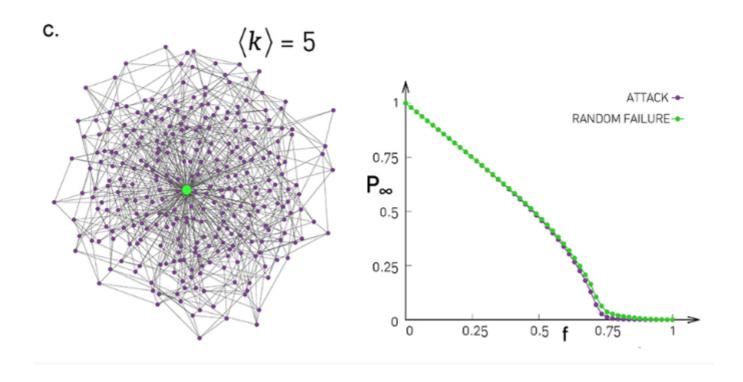
Mixture of nodes

- ullet fraction r of nodes have k_{max} degree
- ullet remaining 1-r nodes have k_{min} degree









Summary

• Percolation process to understand random failure

Summary

- Power law networks are robust to random failures
- Power law networks are susceptible to targeted failures
- Provable robustness to random and targeted failure using mixture of node degrees

Next time

Diffusion!!