

Contagion

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Principles of Complex Systems, Vols. 1 & 2
CSYS/MATH 300 and 303, 2021-2022 | @pocsvox

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Vermont Advanced Computing Core | University of Vermont



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Basic Contagion
Models

Global spreading
condition

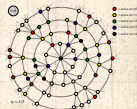
Social Contagion
Models

Network version
All-to-all networks

Theory

Spreading possibility
Spreading probability
Physical explanation
Final size

References



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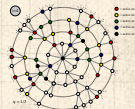
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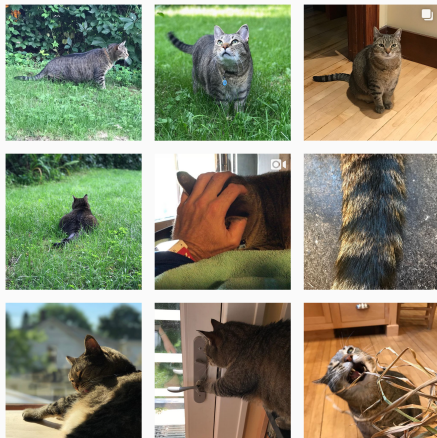
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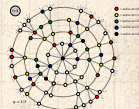
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 On Instagram at [pratchett_the_cat](https://www.instagram.com/pratchett_the_cat) 



Outline

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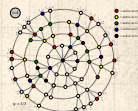
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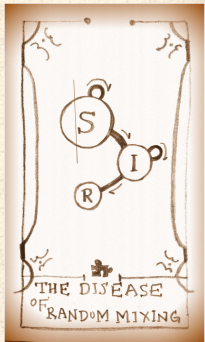
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Contagion models

Some large questions concerning network contagion:

1. For a given spreading mechanism on a given network, what's the **probability** that there will be **global spreading**?
2. If spreading does take off, how far will it go?
3. How do the **details** of the **network** affect the outcome?
4. How do the **details** of the **spreading mechanism** affect the outcome?
5. What if the **seed** is one or many nodes?



Next up: We'll look at some fundamental kinds of spreading on generalized random networks.

Basic Contagion Models

Global spreading condition

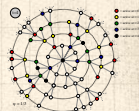
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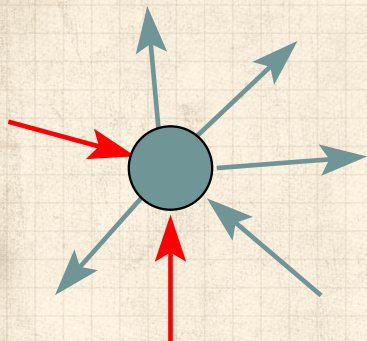
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Spreading mechanisms

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■ uninfected
■ infected



General spreading mechanism:

State of node i depends on history of i and i 's neighbors' states.



Doses of entity may be stochastic and history-dependent.



May have multiple, interacting entities spreading at once.

Basic Contagion Models

Global spreading condition

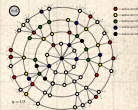
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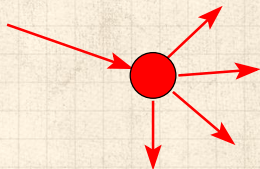


Spreading on Random Networks

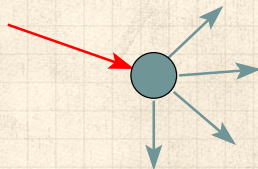
For random networks, we know local structure is pure branching.

Successful spreading is \therefore contingent on **single edges** infecting nodes.

Success



Failure:



Focus on **binary** case with edges and nodes either infected or not.

First big question: for a given network and contagion process, can global spreading from a single seed occur?

Basic Contagion Models

Global spreading condition

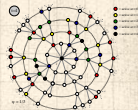
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Global spreading condition

🧱 We need to find: [5]

R = the average # of infected edges that one random infected edge brings about.

🧱 Call **R** the **gain ratio**.

🧱 Define B_{k1} as the probability that a node of degree k is infected by a single infected edge.



$$\mathbf{R} = \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet \underbrace{(k-1)}_{\substack{\# \text{ outgoing} \\ \text{infected} \\ \text{edges}}} \bullet \underbrace{B_{k1}}_{\substack{\text{Prob. of} \\ \text{infection}}} + \sum_{k=0}^{\infty} \frac{\widehat{kP_k}}{\langle k \rangle} \bullet \underbrace{0}_{\substack{\# \text{ outgoing} \\ \text{infected} \\ \text{edges}}} \bullet \underbrace{(1 - B_{k1})}_{\substack{\text{Prob. of} \\ \text{no infection}}}$$

Basic Contagion Models

Global spreading condition

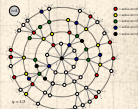
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Global spreading condition

 Our global spreading condition is then:

$$\mathbf{R} = \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet (k-1) \bullet B_{k1} > 1.$$

 **Case 1:** If $B_{k1} = 1$ then

$$\mathbf{R} = \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet (k-1) = \frac{\langle k(k-1) \rangle}{\langle k \rangle} > 1.$$

 **Good:** This is just our giant component condition again.

Basic Contagion Models

Global spreading condition

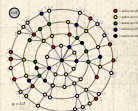
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Global spreading condition


 **Case 2:** If $B_{k1} = \beta < 1$ then

$$\mathbf{R} = \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet (k-1) \bullet \beta > 1.$$


 A fraction $(1-\beta)$ of edges do not transmit infection.


 Analogous phase transition to giant component case but **critical value** of $\langle k \rangle$ is **increased**.

 Aka bond percolation .

 Resulting degree distribution \tilde{P}_k :

$$\tilde{P}_k = \beta^k \sum_{i=k}^{\infty} \binom{i}{k} (1-\beta)^{i-k} P_i.$$

Insert question from assignment 9 

 We can show $F_{\tilde{P}}(x) = F_P(\beta x + 1 - \beta)$.

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Global spreading condition

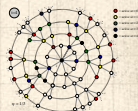
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Global spreading condition

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- Cases 3, 4, 5, ...: Now allow B_{k1} to depend on k
- Asymmetry: Transmission along an edge depends on node's degree at other end.
- Possibility: B_{k1} increases with k ... unlikely.
- Possibility: B_{k1} is not monotonic in k ... unlikely.
- Possibility: B_{k1} decreases with k ... hmmm.
- $B_{k1} \searrow$ is a plausible representation of a simple kind of social contagion.
- The story:
More well connected people are harder to influence.

Basic Contagion Models

Global spreading condition

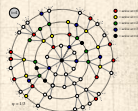
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Global spreading condition


 **Example:** $B_{k1} = 1/k$.



$$\begin{aligned}\mathbf{R} &= \sum_{k=1}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet (k-1) \bullet B_{k1} = \sum_{k=1}^{\infty} (k-1) \bullet \frac{kP_k}{\langle k \rangle} \bullet \frac{1}{k} \\ &= \sum_{k=1}^{\infty} \frac{P_k}{\langle k \rangle} \bullet (k-1) = 1 - \frac{1 - P_0}{\langle k \rangle}\end{aligned}$$

 Since \mathbf{R} is always less than 1, no spreading can occur for this mechanism.

 Decay of B_{k1} is too fast.

 Result is independent of degree distribution.

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Global spreading condition

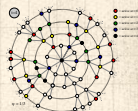
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Global spreading condition

 **Example:** $B_{k1} = H\left(\frac{1}{k} - \phi\right)$
where $0 < \phi \leq 1$ is a **threshold** and H is the
Heaviside function .

 Infection only occurs for nodes with **low** degree.

 Call these nodes **vulnerables**:
they flip when **only one** of their friends flips.



$$\mathbf{R} = \sum_{k=1}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet (k-1) \bullet B_{k1} = \sum_{k=1}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet (k-1) \bullet H\left(\frac{1}{k} - \phi\right)$$
$$= \sum_{k=1}^{\lfloor \frac{1}{\phi} \rfloor} (k-1) \bullet \frac{kP_k}{\langle k \rangle} \quad \text{where } \lfloor \cdot \rfloor \text{ means floor.}$$

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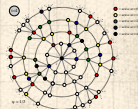
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Global spreading condition

- The uniform threshold model global spreading condition:

$$R = \sum_{k=1}^{\lfloor \frac{1}{\phi} \rfloor} (k-1) \cdot \frac{kP_k}{\langle k \rangle} > 1.$$

- As $\phi \rightarrow 1$, all nodes become resilient and $r \rightarrow 0$.
- As $\phi \rightarrow 0$, all nodes become vulnerable and the contagion condition matches up with the giant component condition.
- Key:** If we fix ϕ and then vary $\langle k \rangle$, we may see **two** phase transitions.
- Added to our standard giant component transition, we will see a cut off in spreading as nodes become more connected.

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Global spreading condition

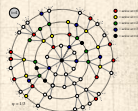
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Virtual contagion: Corrupted Blood, a 2005 virtual plague in World of Warcraft:

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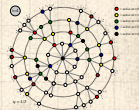
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Some important models (recap from CSYS 300)

-  Tipping models—Schelling (1971) [11, 12, 13]
 -  Simulation on checker boards.
 -  Idea of thresholds.
-  Threshold models—Granovetter (1978) [8]
-  Herding models—Bikhchandani et al. (1992) [1, 2]
 -  Social learning theory, Informational cascades,...

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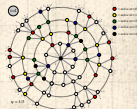
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Threshold model on a network


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Original work:



“A simple model of global cascades on random networks” 

Duncan J. Watts,
Proc. Natl. Acad. Sci., **99**, 5766–5771,
2002. ^[15]

 Mean field Granovetter model → network model

 Individuals now have a limited view of the world

Basic Contagion Models

Global spreading condition

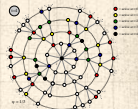
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Threshold model on a network

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- Interactions between individuals now represented by a network
- Network is **sparse**
- Individual i has k_i contacts
- Influence on each link is **reciprocal** and of **unit weight**
- Each individual i has a fixed threshold ϕ_i
- Individuals repeatedly poll contacts on network
- Synchronous, discrete time updating
- Individual i becomes active when number of active contacts $a_i \geq \phi_i k_i$
- Activation is permanent (SI)

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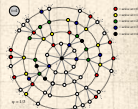
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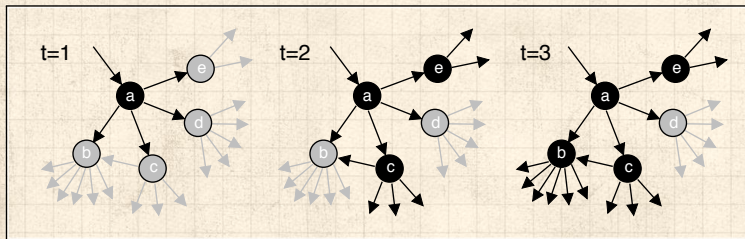
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Threshold model on a network



 All nodes have threshold $\phi = 0.2$.

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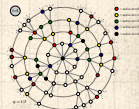
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The most gullible


Vulnerables:

 Recall definition: individuals who can be activated by just one contact being active are **vulnerables**.

 The vulnerability condition for node i : $1/k_i \geq \phi_i$.

 Means # contacts $k_i \leq \lfloor 1/\phi_i \rfloor$.

 **Key:** For global spreading events (cascades) on random networks, must have a *global component of vulnerables* ^[15]

 For a uniform threshold ϕ , our global spreading condition tells us when such a component exists:

$$\mathbf{R} = \sum_{k=1}^{\lfloor \frac{1}{\phi} \rfloor} \frac{k P_k}{\langle k \rangle} \bullet (k - 1) > 1.$$

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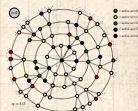
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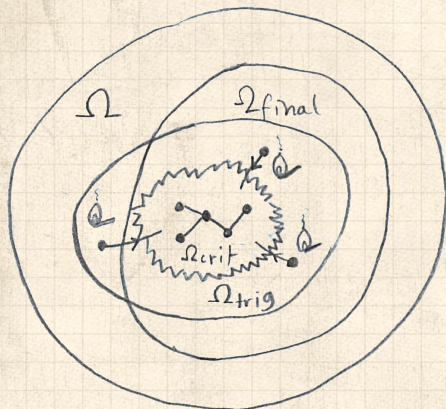
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Example random network structure:



 Ω_{crit} = critical mass = global vulnerable component

 Ω_{trig} = triggering component

 Ω_{final} = potential extent of spread

 Ω = entire network

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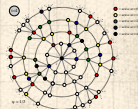
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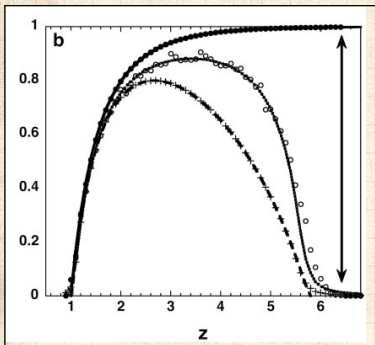
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$$\Omega_{\text{crit}} \subset \Omega_{\text{trig}}; \Omega_{\text{crit}} \subset \Omega_{\text{final}}; \text{ and } \Omega_{\text{trig}}, \Omega_{\text{final}} \subset \Omega.$$

Global spreading events on random networks ^[15]





 **Top curve:** final fraction infected if successful.


 **Middle curve:** chance of starting a global spreading event (cascade).

 **Bottom curve:** fractional size of vulnerable subcomponent. ^[15]

$$z = \langle k \rangle$$

 Global spreading events occur only if size of vulnerable subcomponent > 0 .

 System is robust-yet-fragile just below upper boundary ^[3, 4, 14]

 'Ignorance' facilitates spreading.

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Global spreading condition

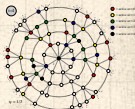
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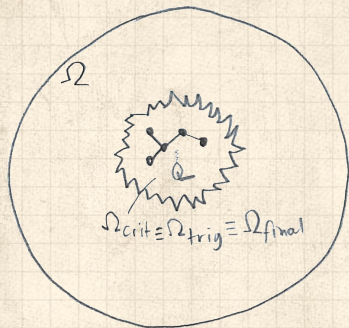
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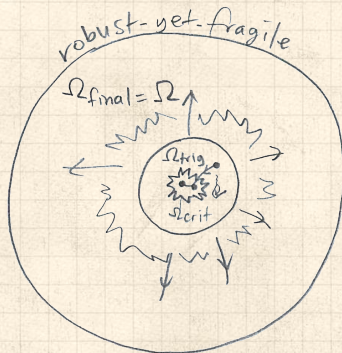
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Cascades on random networks



 Above lower phase transition



 Just below upper phase transition

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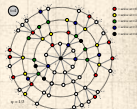
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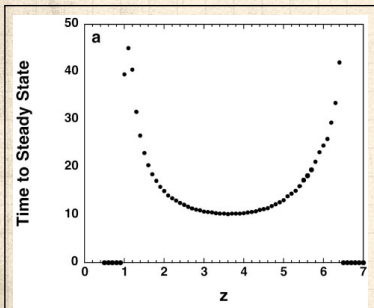
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Cascades on random networks



Time taken for cascade to spread through network. ^[15]



Two phase transitions.

(n.b., $z = \langle k \rangle$)



Largest vulnerable component = **critical mass**.



Now have endogenous mechanism for spreading from an individual to the critical mass and then beyond.

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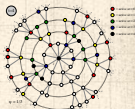
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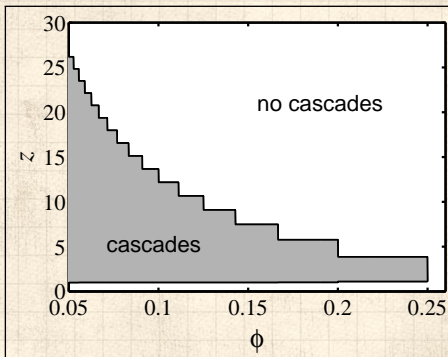
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Cascade window for random networks

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(n.b., $z = \langle k \rangle$)

 Outline of cascade window for random networks.

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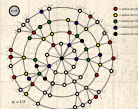
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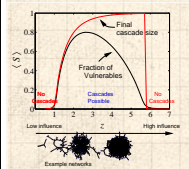
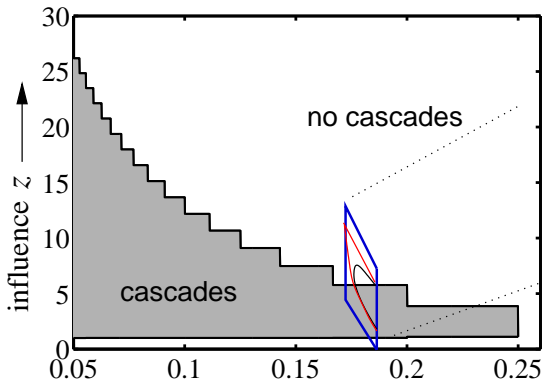
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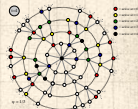
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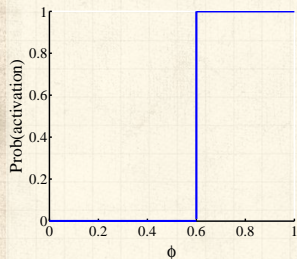
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Granovetter's Threshold model—recap



Assumes deterministic response functions



ϕ_* = threshold of an individual.



$f(\phi_*)$ = distribution of thresholds in a population.



$F(\phi_*)$ = cumulative

distribution = $\int_{\phi'_*=0}^{\phi_*} f(\phi'_*)d\phi'_*$



ϕ_t = fraction of people 'rioting' at time step t .

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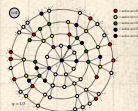
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At time $t + 1$, fraction rioting = fraction with

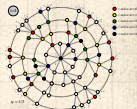
$$\phi_* \leq \phi_t.$$



$$\phi_{t+1} = \int_0^{\phi_t} f(\phi_*) d\phi_* = F(\phi_*)|_0^{\phi_t} = F(\phi_t)$$

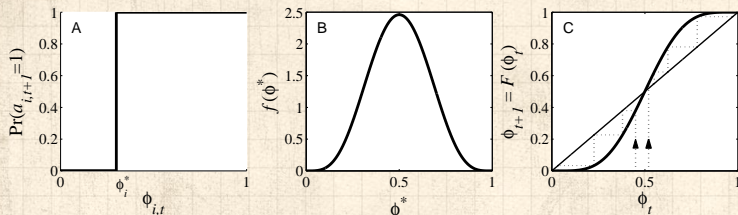


\Rightarrow Iterative maps of the unit interval $[0, 1]$.



Social Sciences—Threshold models

Action based on perceived behavior of others.



- Two states: S and I
- Recover now possible (SIS)
- ϕ = fraction of contacts 'on' (e.g., rioting)
- Discrete time, synchronous update (strong assumption!)
- This is a **Critical mass model**

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Global spreading condition

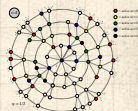
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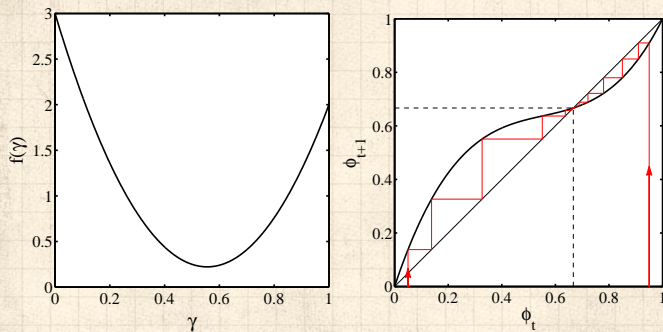
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Example of single stable state model

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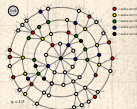
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Implications for collective action theory:

1. Collective uniformity \nRightarrow individual uniformity
2. Small individual changes \Rightarrow large global changes

Next:

-  Connect mean-field model to network model.
-  Single seed for network model: $1/N \rightarrow 0$.
-  Comparison between network and mean-field model sensible for vanishing seed size for the latter.

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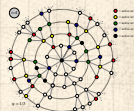
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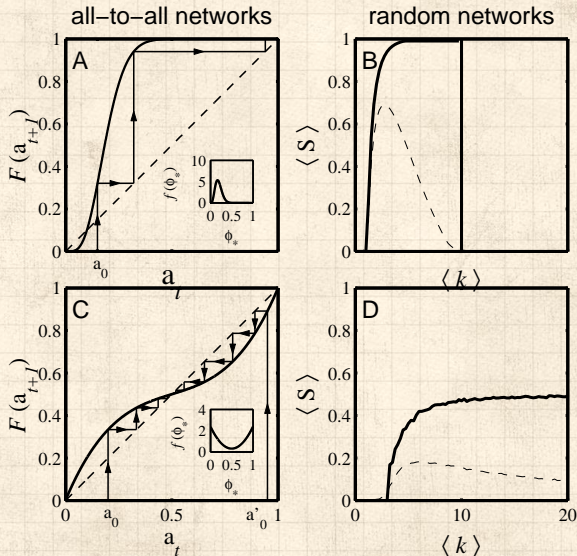
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All-to-all versus random networks



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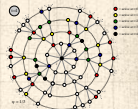
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Spreadworthiness: Cat videos

Bowling with Ragdolls:

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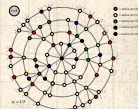
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<https://www.youtube.com/watch?v=XX-g2nmqL9Q?rel=0>

 Organic extreme outlier?

 Success did not spread  to other videos.



Threshold contagion on random networks

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Three key pieces to describe analytically:

1. The fractional size of the largest subcomponent of vulnerable nodes, S_{vuln} .
2. The chance of starting a global spreading event, $P_{\text{trig}} = S_{\text{trig}}$.
3. The expected final size of any successful spread, S .
 - ❏ n.b., the distribution of S is almost always bimodal.

Basic Contagion Models

Global spreading condition

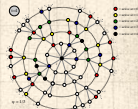
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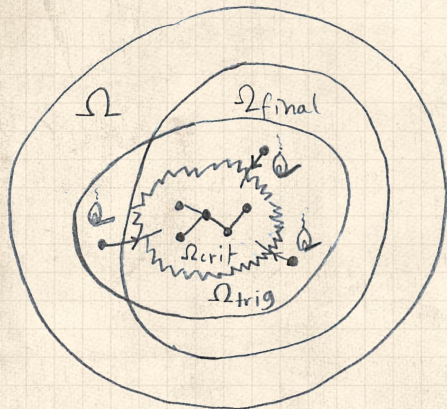
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Example random network structure:



 $\Omega_{crit} = \Omega_{vuln} =$
critical mass =
global
vulnerable
component

 $\Omega_{trig} =$
triggering
component

 $\Omega_{final} =$
potential
extent of
spread

 $\Omega =$ entire
network

$$\Omega_{crit} \subset \Omega_{trig}; \Omega_{crit} \subset \Omega_{final}; \text{ and } \Omega_{trig}, \Omega_{final} \subset \Omega.$$

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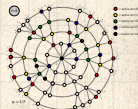
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 **First goal:** Find the largest component of vulnerable nodes.

 Recall that for finding the giant component's size, we had to solve:

$$F_{\pi}(x) = xF_P(F_{\rho}(x)) \quad \text{and} \quad F_{\rho}(x) = xF_R(F_{\rho}(x))$$

 We'll find a similar result for the subset of nodes that are vulnerable.

 This is a node-based percolation problem.

 For a general monotonic threshold distribution $f(\phi)$, a degree k node is vulnerable with probability

$$B_{k1} = \int_0^{1/k} f(\phi) d\phi.$$

Basic Contagion Models

Global spreading condition

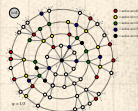
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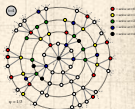
-  We now have a generating function for the probability that a randomly chosen node is vulnerable and has degree k :

$$F_P^{(\text{vuln})}(x) = \sum_{k=0}^{\infty} P_k B_{k1} x^k.$$

-  The generating function for friends-of-friends distribution is similar to before:

$$\begin{aligned} F_R^{(\text{vuln})}(x) &= \sum_{k=0}^{\infty} \frac{k P_k}{\langle k \rangle} B_{k1} x^{k-1} \\ &= \frac{\frac{d}{dx} F_P^{(\text{vuln})}(x)}{\frac{d}{dx} F_P(x)|_{x=1}} = \frac{\frac{d}{dx} F_P^{(\text{vuln})}(x)}{F_R(1)} \end{aligned}$$

-  Detail: We still have the underlying degree distribution involved in the denominator.



Threshold contagion on random networks

Functional relations for component size g.f.'s are almost the same ...

$$F_{\pi}^{(\text{vuln})}(x) = \underbrace{1 - F_P^{(\text{vuln})}(1)}_{\substack{\text{central node} \\ \text{is not} \\ \text{vulnerable}}} + x F_P^{(\text{vuln})}(F_{\rho}^{(\text{vuln})}(x))$$

$$F_{\rho}^{(\text{vuln})}(x) = \underbrace{1 - F_R^{(\text{vuln})}(1)}_{\substack{\text{first node} \\ \text{is not} \\ \text{vulnerable}}} + x F_R^{(\text{vuln})}(F_{\rho}^{(\text{vuln})}(x))$$

Can now solve as before to find

$$S_{\text{vuln}} = 1 - F_{\pi}^{(\text{vuln})}(1).$$

Basic Contagion Models

Global spreading condition

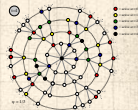
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Threshold contagion on random networks


 **Second goal:** Find probability of triggering largest vulnerable component.

 Assumption is **first node** is **randomly chosen**.

 **Same set up** as for vulnerable component except now we don't care if the initial node is vulnerable or not:

$$F_{\pi}^{(\text{trig})}(x) = xF_P \left(F_{\rho}^{(\text{vuln})}(x) \right)$$

$$F_{\rho}^{(\text{vuln})}(x) = 1 - F_R^{(\text{vuln})}(1) + xF_R^{(\text{vuln})} \left(F_{\rho}^{(\text{vuln})}(x) \right)$$

 Solve as before to find $P_{\text{trig}} = S_{\text{trig}} = 1 - F_{\pi}^{(\text{trig})}(1)$.

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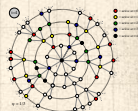
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Physical derivation of possibility and probability of global spreading:

🧱 Possibility: binary indicator of phase. Global spreading events are either possible or can never happen.

🧱 For random networks, global spreading possibility is understood as meaning a giant component of vulnerable nodes exists.

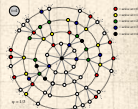
🧱 Next: what's the probability that a randomly infected node will cause a global spreading event?

🧱 Call this P_{trig} .


🧱 As usual, it's all about edges and we need to first determine the probability that an infected edge leads to a global spreading event.


🧱 Call this Q_{trig} .

🧱 Later: Generalize to more complex networks involving assortativity of all kinds.



Probability an infected edge leads to a global spreading event:

 Q_{trig} must satisfy a one-step recursion relation.


 Follow an infected edge and use three pieces:

1. Probability of reaching a degree k node is

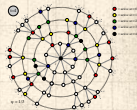
$$Q_k = \frac{k P_k}{\langle k \rangle}.$$

2. The node reached is vulnerable with probability B_{k1} .

3. At least one of the node's outgoing edges leads to a global spreading event = $1 - \text{probability no edges do so} = 1 - (1 - Q_{\text{trig}})^{k-1}$.

 Put everything together and solve for Q_{trig} :

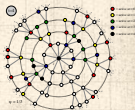
$$Q_{\text{trig}} = \sum_k \frac{k P_k}{\langle k \rangle} \cdot B_{k1} \cdot [1 - (1 - Q_{\text{trig}})^{k-1}].$$



Good things about our equation for Q_{trig} :

$$Q_{\text{trig}} = \sum_k \frac{kP_k}{\langle k \rangle} \cdot B_{k1} \cdot [1 - (1 - Q_{\text{trig}})^{k-1}] = f(Q_{\text{trig}}; P_k, B_{k1})$$

- 🧱 $Q_{\text{trig}} = 0$ is always a solution.
- 🧱 Spreading occurs if a second solution exists for which $0 < Q_{\text{trig}} \leq 1$.
- 🧱 Given P_k and B_{k1} , we can use any kind of root finder to solve for Q_{trig} , but ...
- 🧱 The function f increases monotonically with Q_{trig} .
- 🧱 We can therefore use an iterative cobwebbing approach to find the solution:
$$Q_{\text{trig}}^{(n+1)} = f(Q_{\text{trig}}^{(n)}; P_k, B_{k1}).$$
- 🧱 Start with a suitably small seed $Q_{\text{trig}}^{(1)} > 0$ and iterate while rubbing hands together.



Global spreading is possible if the fractional size S_{vuln} of the largest component of vulnerables is “giant”.

Interpret S_{vuln} as the probability a randomly chosen node is vulnerable and that infecting it leads to a global spreading event:

$$S_{\text{vuln}} = \sum_k P_k \cdot B_{k1} \cdot [1 - (1 - Q_{\text{trig}})^k] > 0.$$

Amounts to having $Q_{\text{trig}} > 0$.

Probability of global spreading differs only in that we don't care if the initial seed is vulnerable or not:

$$P_{\text{trig}} = S_{\text{trig}} = \sum_k P_k \cdot [1 - (1 - Q_{\text{trig}})^k]$$

As for S_{vuln} , P_{trig} is non-zero when $Q_{\text{trig}} > 0$.

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Global spreading condition

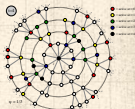
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Connection to generating function results:

- 🧱 We found that $F_\rho^{(\text{vuln})}(1)$ —the probability that a random edge leads to a finite vulnerable component—satisfies

$$F_\rho^{(\text{vuln})}(1) = 1 - F_R^{(\text{vuln})}(1) + 1 \cdot F_R^{(\text{vuln})}(F_\rho^{(\text{vuln})}(1)).$$

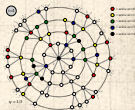
- 🧱 We set $F_\rho^{(\text{vuln})}(1) = 1 - Q_{\text{trig}}$ and deploy

$$F_R^{(\text{vuln})}(x) = \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} B_{k1} x^{k-1} \text{ to find}$$

$$1 - Q_{\text{trig}} = 1 - \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} B_{k1} + \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} B_{k1} (1 - Q_{\text{trig}})^{k-1}.$$

- 🧱 Some breathless algebra it all matches:

$$Q_{\text{trig}} = \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} \cdot B_{k1} \cdot \left[1 - (1 - Q_{\text{trig}})^{k-1} \right].$$



Fractional size of the largest vulnerable component:



The generating function approach gave

$S_{\text{vuln}} = 1 - F_{\pi}^{(\text{vuln})}(1)$ where

$$F_{\pi}^{(\text{vuln})}(1) = 1 - F_P^{(\text{vuln})}(1) + 1 \cdot F_P^{(\text{vuln})}(F_{\rho}^{(\text{vuln})}(1)).$$



Again using $F_{\rho}^{(\text{vuln})}(1) = 1 - Q_{\text{trig}}$ along with

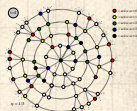
$F_P^{(\text{vuln})}(x) = \sum_{k=0}^{\infty} P_k B_{k1} x^k$, we have:

$$1 - S_{\text{vuln}} = 1 - \sum_{k=0}^{\infty} P_k B_{k1} + \sum_{k=0}^{\infty} P_k B_{k1} (1 - Q_{\text{trig}})^k.$$



Excited scabbling about gives us, as before:

$$S_{\text{vuln}} = \sum_{k=0}^{\infty} P_k B_{k1} \left[1 - (1 - Q_{\text{trig}})^k \right].$$



Triggering probability for single-seed global spreading events:

🧱 Slight adjustment to the vulnerable component calculation.

🧱 $S_{\text{trig}} = 1 - F_{\pi}^{(\text{trig})}(1)$ where

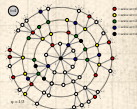
$$F_{\pi}^{(\text{trig})}(1) = 1 \cdot F_P \left(F_{\rho}^{(\text{vuln})}(1) \right).$$

🧱 We play these cards: $F_{\rho}^{(\text{vuln})}(1) = 1 - Q_{\text{trig}}$ and $F_P(x) = \sum_{k=0}^{\infty} P_k x^k$ to arrive at

$$1 - S_{\text{trig}} = 1 + \sum_{k=0}^{\infty} P_k \left(1 - Q_{\text{trig}} \right)^k.$$

🧱 More scruffing around brings happiness:

$$S_{\text{trig}} = \sum_{k=0}^{\infty} P_k \left[1 - \left(1 - Q_{\text{trig}} \right)^k \right].$$



Connection to simple gain ratio argument:

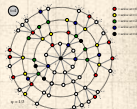
- Earlier, we showed the global spreading condition follows from the gain ratio $\mathbf{R} > 1$:

$$\mathbf{R} = \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} \cdot (k-1) \cdot B_{k1} > 1.$$

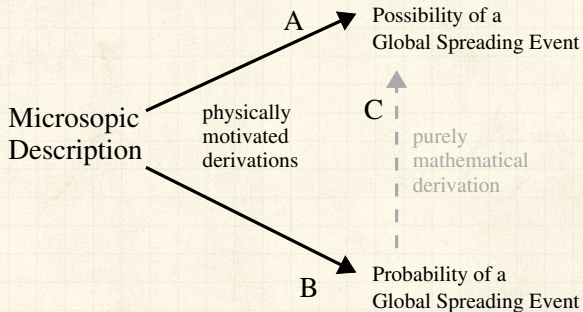
- We would very much like to see that $\mathbf{R} > 1$ matches up with $Q_{\text{trig}} > 0$.
- It really would be just so totally awesome.
- Must come from our basic edge triggering probability equation:

$$Q_{\text{trig}} = \sum_k \frac{kP_k}{\langle k \rangle} \cdot B_{k1} \cdot [1 - (1 - Q_{\text{trig}})^{k-1}].$$

- When does this equation have a solution $0 < Q_{\text{trig}} \leq 1$?
- We need to find out what happens as $Q_{\text{trig}} \rightarrow 0$. [9]



What we're doing:



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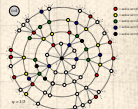
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For $Q_{\text{trig}} \rightarrow 0^+$, equation tends towards

$$Q_{\text{trig}} = \sum_k \frac{kP_k}{\langle k \rangle} \cdot B_{k1} \cdot [\lambda + (\lambda + (k-1)Q_{\text{trig}} + \dots)]$$

$$\Rightarrow Q_{\text{trig}} = \sum_k \frac{kP_k}{\langle k \rangle} \cdot B_{k1} \cdot (k-1)Q_{\text{trig}}$$

$$\Rightarrow 1 = \sum_k \frac{kP_k}{\langle k \rangle} \cdot (k-1) \cdot B_{k1}$$

Only defines the phase transition points (i.e., $\mathbf{R} = 1$).

Inequality?

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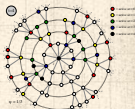
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 Again take $Q_{\text{trig}} \rightarrow 0^+$, but keep next higher order term:


$$Q_{\text{trig}} = \sum_k \frac{kP_k}{\langle k \rangle} \bullet B_{k1} \bullet \left[\lambda + \left(\lambda + (k-1)Q_{\text{trig}} - \binom{k-1}{2} Q_{\text{trig}}^2 \right) \right]$$

$$\Rightarrow Q_{\text{trig}} = \sum_k \frac{kP_k}{\langle k \rangle} \bullet B_{k1} \bullet \left[(k-1)Q_{\text{trig}} - \binom{k-1}{2} Q_{\text{trig}}^2 \right]$$

$$\Rightarrow \sum_k \frac{kP_k}{\langle k \rangle} \bullet (k-1) \bullet B_{k1} = 1 + \sum_k \frac{kP_k}{\langle k \rangle} B_{k1} \binom{k-1}{2} Q_{\text{trig}}$$

 We have $Q_{\text{trig}} > 0$ if $\sum_k \frac{kP_k}{\langle k \rangle} \bullet (k-1) \bullet B_{k1} > 1$.

 Repeat: Above is a mathematical connection between two physically derived equations.

 From this connection, we don't know anything about a gain ratio **R** or how to arrange the pieces.

Threshold contagion on random networks

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- Third goal: Find expected fractional size of spread.
- Not obvious even for uniform threshold problem.
- Difficulty is in figuring out if and when nodes that need ≥ 2 hits switch on.
- Problem solved for infinite seed case by Gleeson and Cahalane:
"Seed size strongly affects cascades on random networks," Phys. Rev. E, 2007. [7]
- Developed further by Gleeson in "Cascades on correlated and modular random networks," Phys. Rev. E, 2008. [6]

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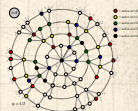
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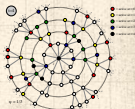
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Expected size of spread

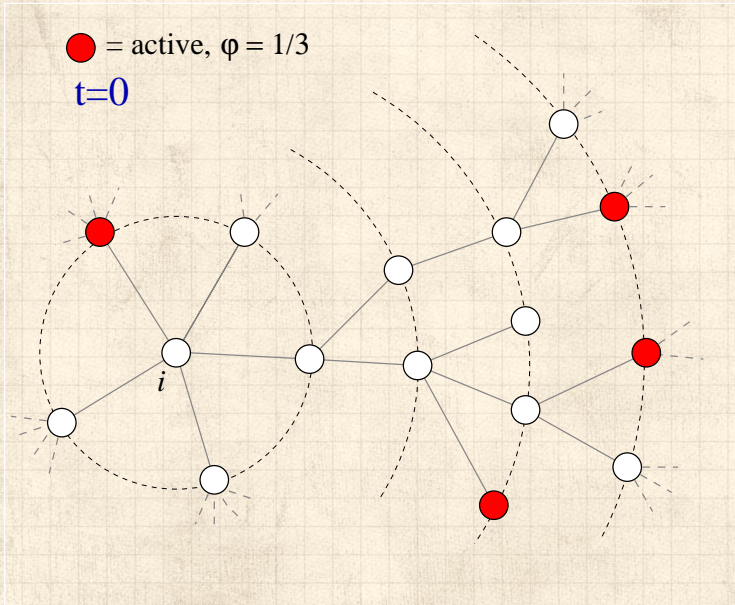
Idea:

- ☰ Randomly turn on a fraction ϕ_0 of nodes at time $t = 0$
- ☰ Capitalize on local branching network structure of random networks (again)
- ☰ Now think about what must happen for a specific node i to become active at time t :
 - $t = 0$: i is one of the seeds (prob = ϕ_0)
 - $t = 1$: i was not a seed but enough of i 's friends switched on at time $t = 0$ so that i 's threshold is now exceeded.
 - $t = 2$: enough of i 's friends and friends-of-friends switched on at time $t = 0$ so that i 's threshold is now exceeded.
 - $t = n$: enough nodes within n hops of i switched on at $t = 0$ and their effects have propagated to reach i .



Expected size of spread

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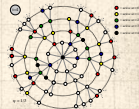
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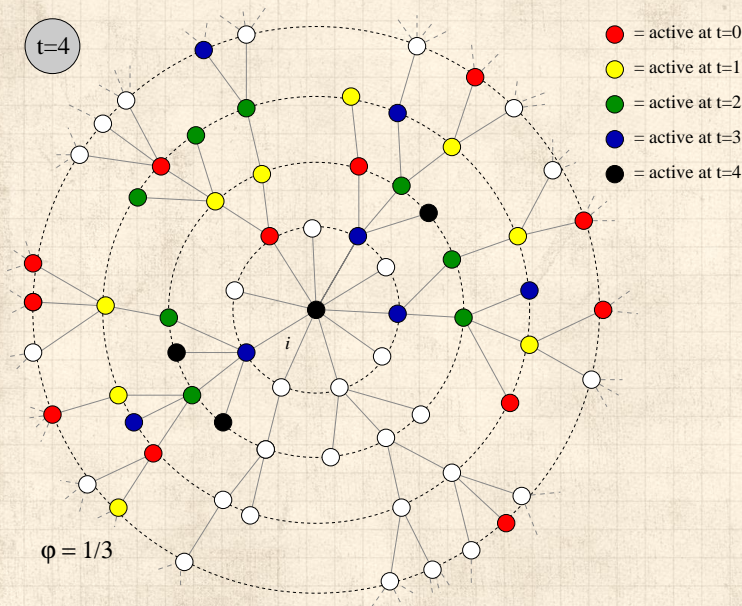
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Contagion



Basic Contagion Models

Global spreading condition

Social Contagion Models

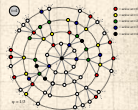
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Expected size of spread

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Notes:

- Calculations presume nodes do not become inactive (strong restriction, liftable)
- Not just for threshold model—works for a wide range of contagion processes.
- We can analytically determine the entire time evolution, not just the final size.
- We can in fact determine $\Pr(\text{node of degree } k \text{ switches on at time } t)$.
- Even more, we can compute: $\Pr(\text{specific node } i \text{ switches on at time } t)$.
- Asynchronous updating can be handled too.

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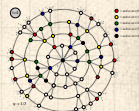
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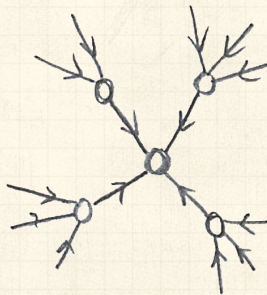
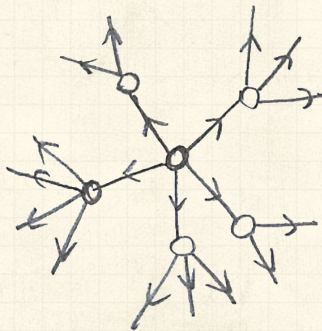
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Expected size of spread

Pleasantness:

- ✦ Taking off from a single seed story is about **expansion** away from a node.
- ✦ Extent of spreading story is about **contraction** at a node.



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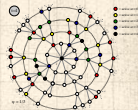
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Expected size of spread



Notation:

$\phi_{k,t} = \Pr$ (a degree k node is active at time t).



Notation: $B_{kj} = \Pr$ (a degree k node becomes active if j neighbors are active).



Our starting point: $\phi_{k,0} = \phi_0$.



$\binom{k}{j} \phi_0^j (1 - \phi_0)^{k-j} = \Pr$ (j of a degree k node's neighbors were seeded at time $t = 0$).



Probability a degree k node was a seed at $t = 0$ is ϕ_0 (as above).



Probability a degree k node was not a seed at $t = 0$ is $(1 - \phi_0)$.



Combining everything, we have:

$$\phi_{k,1} = \phi_0 + (1 - \phi_0) \sum_{j=0}^k \binom{k}{j} \phi_0^j (1 - \phi_0)^{k-j} B_{kj}.$$

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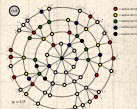
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Expected size of spread

For general t , we need to know the probability an edge coming into a degree k node at time t is active.

Notation: call this probability θ_t .

We already know $\theta_0 = \phi_0$.

Story analogous to $t = 1$ case. For specific node i :

$$\phi_{i,t+1} = \phi_0 + (1 - \phi_0) \sum_{j=0}^{k_i} \binom{k_i}{j} \theta_t^j (1 - \theta_t)^{k_i - j} B_{k_i j}.$$

Average over all nodes with degree k to obtain expression for ϕ_{t+1} :

$$\phi_{t+1} = \phi_0 + (1 - \phi_0) \sum_{k=0}^{\infty} P_k \sum_{j=0}^k \binom{k}{j} \theta_t^j (1 - \theta_t)^{k-j} B_{k j}.$$

So we need to compute θ_t ... massive excitement...

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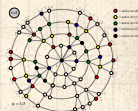
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Expected size of spread


First connect θ_0 to θ_1 :


 $\theta_1 = \phi_0 +$

$$(1 - \phi_0) \sum_{k=1}^{\infty} \frac{k P_k}{\langle k \rangle} \sum_{j=0}^{k-1} \binom{k-1}{j} \theta_0^j (1 - \theta_0)^{k-1-j} B_{kj}$$

 $\frac{k P_k}{\langle k \rangle} = Q_k = \mathbf{Pr}$ (edge connects to a degree k node).

 $\sum_{j=0}^{k-1}$ piece gives \mathbf{Pr} (degree node k activates if j of its $k - 1$ incoming neighbors are active).

 ϕ_0 and $(1 - \phi_0)$ terms account for state of node at time $t = 0$.

 See this all generalizes to give θ_{t+1} in terms of $\theta_t \dots$

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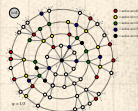
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Expected size of spread

Two pieces: edges first, and then nodes

$$1. \theta_{t+1} = \underbrace{\phi_0}_{\text{exogenous}}$$

$$+(1 - \phi_0) \underbrace{\sum_{k=1}^{\infty} \frac{k P_k}{\langle k \rangle} \sum_{j=0}^{k-1} \binom{k-1}{j} \theta_t^j (1 - \theta_t)^{k-1-j} B_{kj}}_{\text{social effects}}$$

with $\theta_0 = \phi_0$.

$$2. \phi_{t+1} =$$

$$\underbrace{\phi_0}_{\text{exogenous}} + (1 - \phi_0) \underbrace{\sum_{k=0}^{\infty} P_k \sum_{j=0}^k \binom{k}{j} \theta_t^j (1 - \theta_t)^{k-j} B_{kj}}_{\text{social effects}}$$

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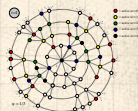
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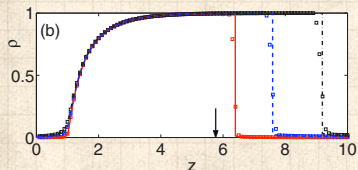
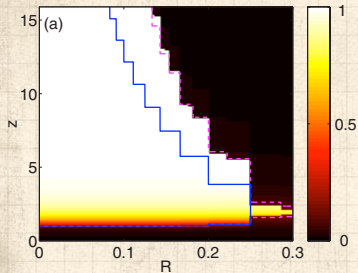
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Comparison between theory and simulations



Pure random networks with simple threshold responses



$R =$ uniform threshold (our ϕ_*); $z =$ average degree; $\rho = \phi$; $q = \theta$; $N = 10^5$.



$\phi_0 = 10^{-3}$, 0.5×10^{-2} , and 10^{-2} .



Cascade window is for $\phi_0 = 10^{-2}$ case.



Sensible expansion of cascade window as ϕ_0 increases.

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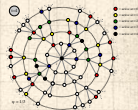
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From Gleeson and Cahalane [7]



Notes:

- Retrieve cascade condition for spreading from a single seed in limit $\phi_0 \rightarrow 0$.
- Depends on map $\theta_{t+1} = G(\theta_t; \phi_0)$.
- First: if self-starters are present, some activation is assured:

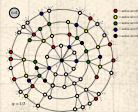
$$G(0; \phi_0) = \sum_{k=1}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet B_{k0} > 0.$$

meaning $B_{k0} > 0$ for at least one value of $k \geq 1$.

- If $\theta = 0$ is a fixed point of G (i.e., $G(0; \phi_0) = 0$) then spreading occurs for a small seed if

$$G'(0; \phi_0) = \sum_{k=0}^{\infty} \frac{kP_k}{\langle k \rangle} \bullet (k-1) \bullet B_{k1} > 1.$$

[Insert question from assignment 10](#)



In words:

- 🧱 If $G(0; \phi_0) > 0$, spreading must occur because some nodes turn on for free.
- 🧱 If G has an **unstable fixed point** at $\theta = 0$, then cascades are also always possible.

Non-vanishing seed case:

- 🧱 Cascade condition is more complicated for $\phi_0 > 0$.
- 🧱 If G has a **stable fixed point** at $\theta = 0$, and an **unstable fixed point** for some $0 < \theta_* < 1$, then for $\theta_0 > \theta_*$, spreading takes off.
- 🧱 Tricky point: G depends on ϕ_0 , so as we change ϕ_0 , we also change G .

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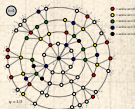
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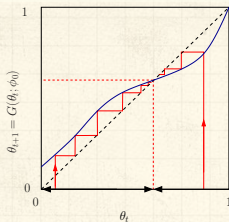
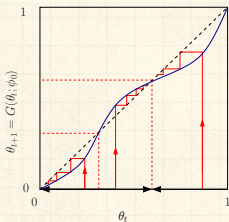
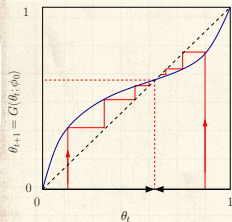
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General fixed point story:



Given $\theta_0 (= \phi_0)$, θ_∞ will be the nearest stable fixed point, either above or below.

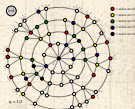
n.b., adjacent fixed points must have opposite stability types.

Important: Actual form of G depends on ϕ_0 .

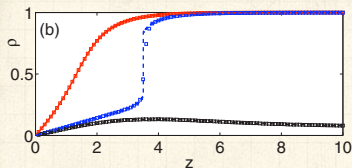
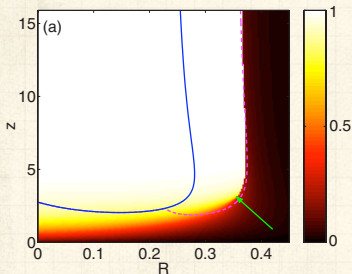
Important: ϕ_t can only increase monotonically so ϕ_0 must shape G so that ϕ_0 is at or above an unstable fixed point.

First reason: $\phi_1 \geq \phi_0$.

Second: $G'(\theta; \phi_0) \geq 0, 0 \leq \theta \leq 1$.



Interesting behavior:



Now allow thresholds to be distributed according to a Gaussian with mean R .



$R = 0.2, 0.362,$ and $0.38; \sigma = 0.2.$



$\phi_0 = 0$ but some nodes have thresholds ≤ 0 so effectively $\phi_0 > 0.$



Now see a (nasty) discontinuous phase transition for low $\langle k \rangle.$

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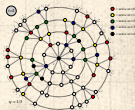
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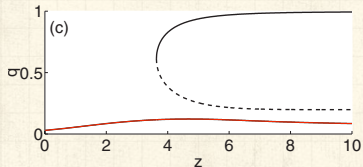
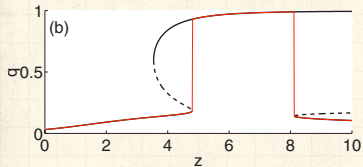
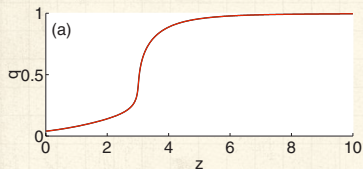
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From Gleeson and Cahalane [7]



Interesting behavior:



Plots of stability points for $\theta_{t+1} = G(\theta_t; \phi_0)$.



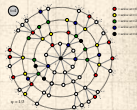
n.b.: 0 is not a fixed point here: $\theta_0 = 0$ always takes off.



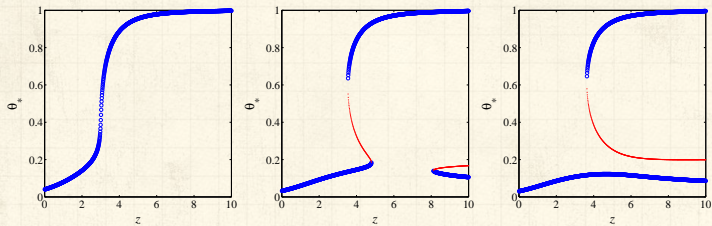
Top to bottom: $R = 0.35, 0.371, \text{ and } 0.375$.



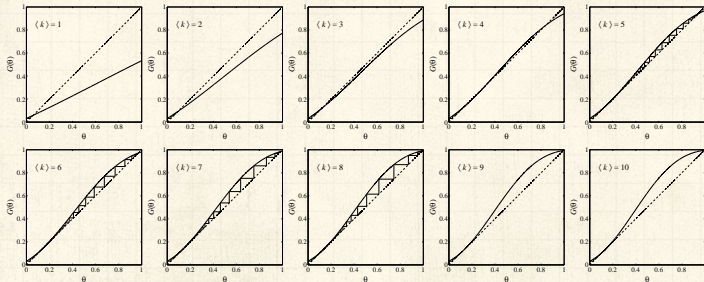
Saddle node bifurcations appear and merge (b and c).



What's happening:



Fixed points slip above and below the $\theta_{t+1} = \theta_t$ line:



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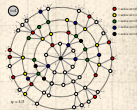
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Time-dependent solutions

Synchronous update

- Done: Evolution of ϕ_t and θ_t given exactly by the maps we have derived.

Asynchronous updates

- Update nodes with probability α .
- As $\alpha \rightarrow 0$, updates become effectively independent.
- Now can talk about $\phi(t)$ and $\theta(t)$.

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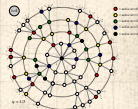
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Nutshell:

- ☰ Solid dive into understanding contagion on generalized random networks.
- ☰ Threshold model leads to idea of vulnerables and a critical mass. [16, 8]
- ☰ Generating function approaches provided first breakthroughs and gave possibility and probability of spreading. [10, 16]
- ☰ Later: A probabilistic, physical method solved the whole story for a fractional seed—final size, dynamics, ... [7, 6]
- ☰ Much can be generalized for more realistic kinds of networks: degree-correlated, modular, bipartite, ...
- ☰ The single seed contagion condition and triggering probability can be fully developed using a physical story. [5, 9]
- ☰ Many connections to other kinds of models: Voter models, Ising models, ...

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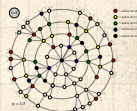
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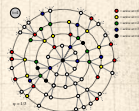
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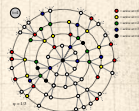
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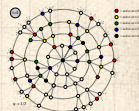
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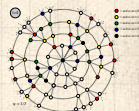
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