

Social Contagion

Principles of Complex Systems

Course 300, Fall, 2008

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Network version
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Examples abound

- ▶ fashion
- ▶ striking
- ▶ smoking (田) [6]
- ▶ residential segregation [15]
- ▶ ipods
- ▶ obesity (田) [5]
- ▶ Harry Potter
- ▶ voting
- ▶ gossip
- ▶ Rubik's cube 
- ▶ religious beliefs
- ▶ leaving lectures

SIR and SIRS contagion possible

- ▶ Classes of behavior versus specific behavior


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SIR and SIR**S** contagion possible

- ▶ Classes of behavior versus specific behavior: **dieting**

Framingham heart study:

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Evolving network stories:

- ▶ The spread of quitting smoking (田) [6]
- ▶ The spread of spreading (田) [5]

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Two focuses for us

- ▶ Widespread media influence
- ▶ Word-of-mouth influence

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We need to understand influence

- ▶ Who influences whom?
- ▶ What kinds of influence response functions are there?
- ▶ Are some individuals super influencers?
- ▶ The infectious idea of opinion leaders (Katz and Lazarsfeld) ^[12]

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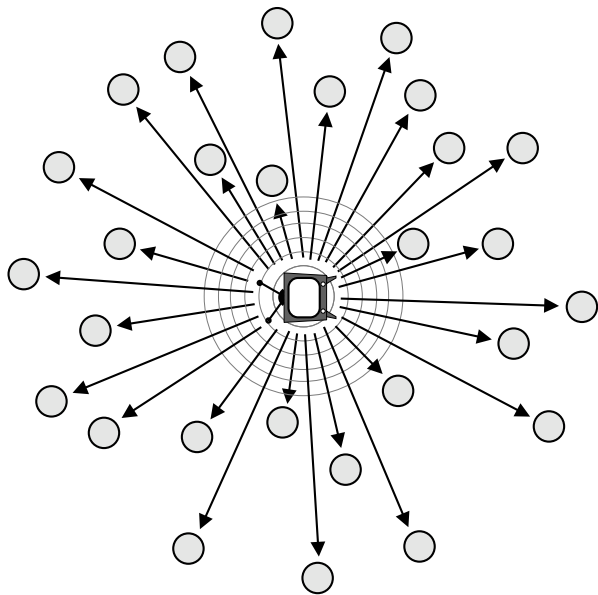
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The hypodermic model of influence

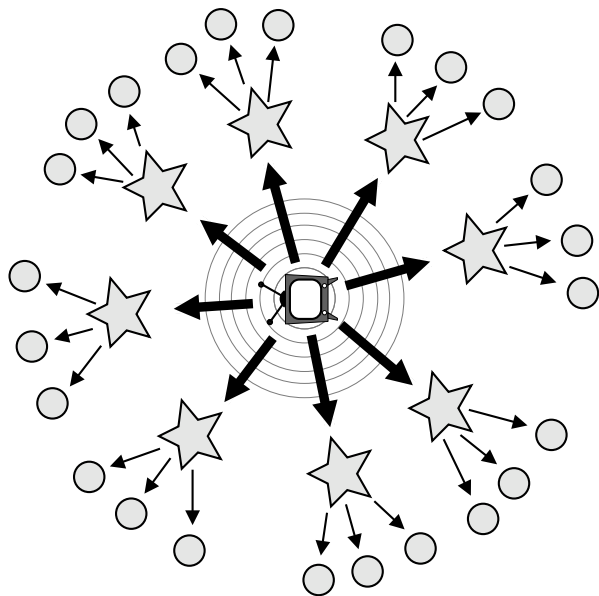


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The two step model of influence [12]

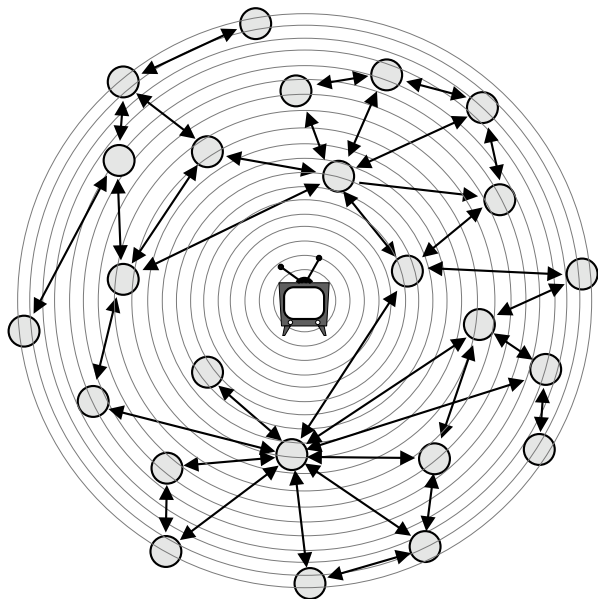


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The general model of influence



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Why do things spread?

- ▶ Because of system level properties?
- ▶ Or properties of special individuals?
- ▶ Is the match that lights the fire important?
- ▶ Yes. But only because we are narrative-making machines...
- ▶ We like to think things happened for reasons...
- ▶ System/group properties harder to understand
- ▶ Always good to examine what is said before and after the fact...

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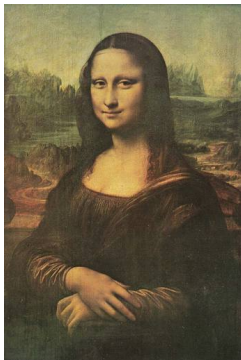
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The Mona Lisa



- ▶ “Becoming Mona Lisa: The Making of a Global Icon”—David Sassoon
- ▶ Not the world’s greatest painting from the start...
- ▶ Escalation through theft, vandalism,

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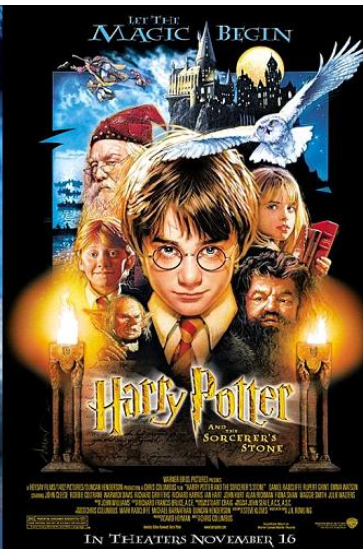
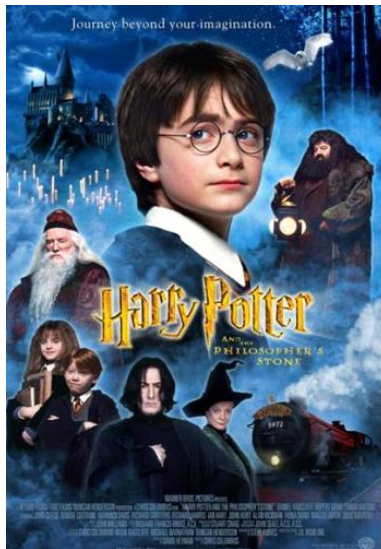
The completely unpredicted fall of Eastern Europe



Timur Kuran: ^[13, 14] “Now Out of Never: The Element of Surprise in the East European Revolution of 1989”

The dismal predictive powers of editors...

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- ▶ Buzz media
- ▶ Facebook's advertising: [Beacon](#) (田)

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Getting others to do things for you

A very good book: **'Influence'** by Robert Cialdini [7]

Six modes of influence

1. *Reciprocation: The Old Give and Take... and Take*
2. *Commitment and Consistency: Hobgoblins of the Mind*
3. *Social Proof: Truths Are Us*
4. *Liking: The Friendly Thief*
5. *Authority: Directed Deference*
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Examples

- ▶ **Reciprocation**: Free samples, Hare Krishnas
- ▶ **Commitment and Consistency**: Hazing
- ▶ **Social Proof**: Catherine Genovese, Jonestown
- ▶ **Liking**: Separation into groups is enough to cause problems.
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- ▶ Useful but can be leveraged...

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- ▶ Conspicuous Destruction (Potlatch)

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- ▶ Tipping models—Schelling (1971) ^[15, 16, 17]
 - ▶ Simulation on checker boards
 - ▶ Idea of thresholds
 - ▶ Fun with Netlogo and Schelling's model ^[20]...
- ▶ Threshold models—Granovetter (1978) ^[9]
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 - ▶ Social learning theory, Informational cascades,...

Thresholds

- ▶ Basic idea: individuals adopt a behavior when a **certain fraction of others** have adopted
- ▶ 'Others' may be everyone in a population, an individual's close friends, any reference group.
- ▶ Response can be probabilistic or deterministic.
- ▶ Individual thresholds can vary
- ▶ Assumption: order of others' adoption does not matter...
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- ▶ Assumption: level of influence per person is uniform **(unrealistic)**.

Some possible origins of thresholds:

- ▶ **Desire to coordinate**, to conform.
- ▶ **Lack of information**: impute the worth of a good or behavior based on degree of adoption (social proof)
- ▶ Economics: **Network effects** or **network externalities**
- ▶ Externalities = Effects on others not directly involved in a transaction
- ▶ Examples: telephones, fax machine, Facebook, operating systems
- ▶ An individual's utility increases with the adoption level among peers and the population in general

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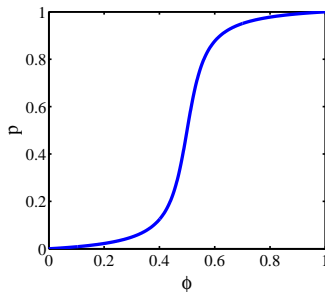
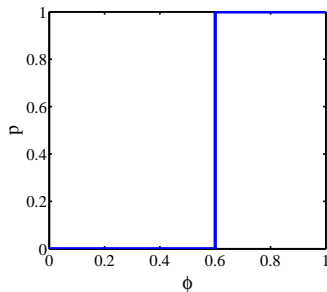
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Threshold models



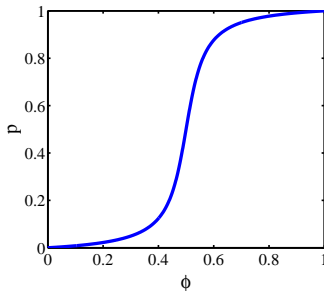
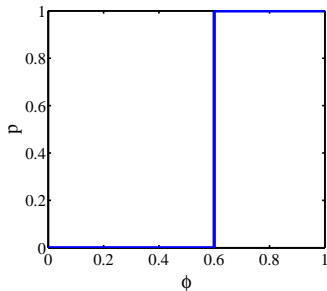
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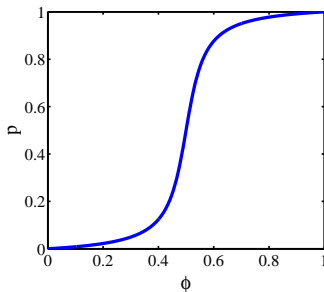
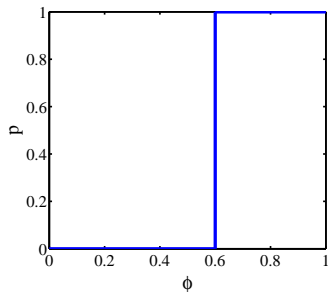
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- ▶ At time $t + 1$, fraction rioting = fraction with $\phi_* \leq \phi_t$.



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

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

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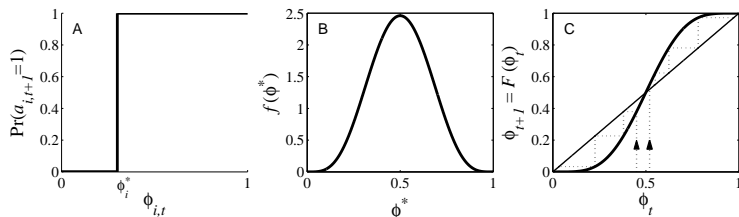
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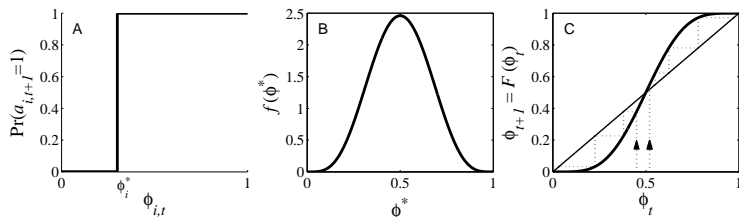
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Action based on perceived behavior of others.



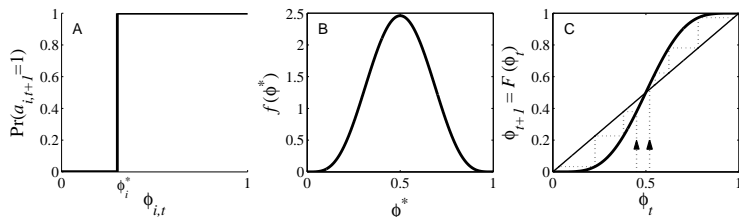
- ▶ Two states: S and I.
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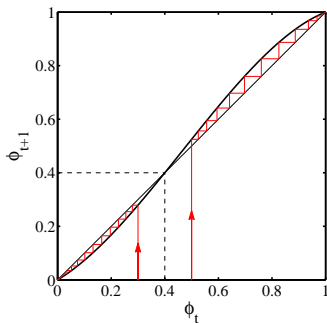
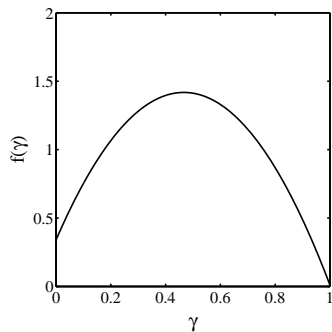
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Threshold models



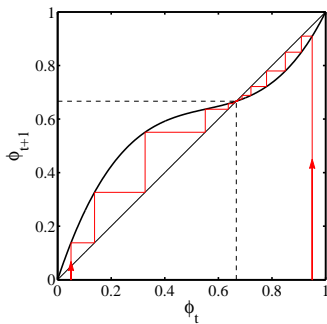
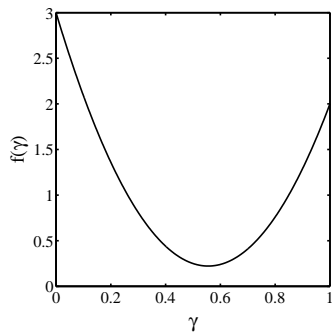
- ▶ Another example of critical mass model...

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- ▶ Example of single stable state model

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Implications for collective action theory:

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

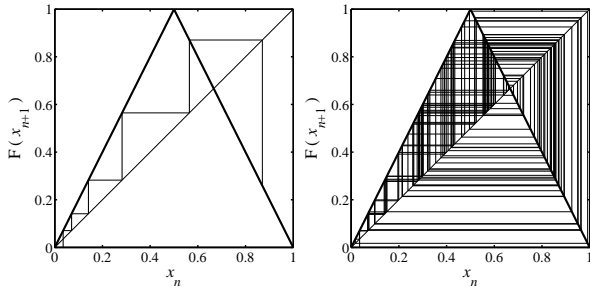
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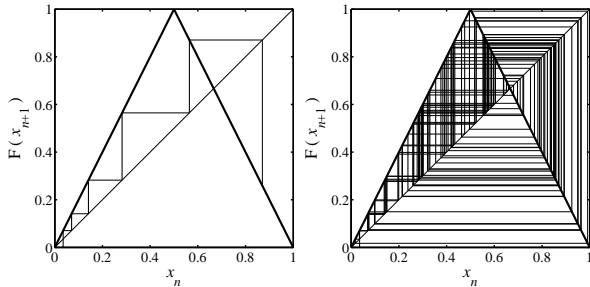
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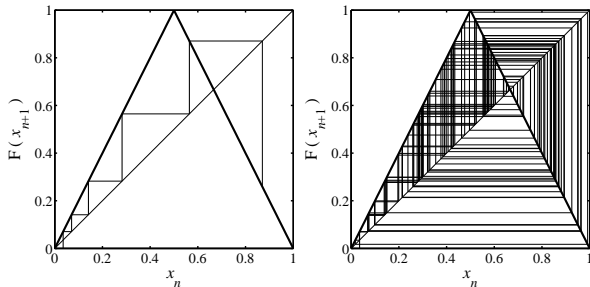
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Many years after Granovetter and Soong's work:

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Threshold model on a network

- ▶ 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
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- ▶ Individual i becomes active when fraction of active contacts $a_i \geq \phi_i k_i$
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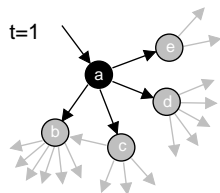
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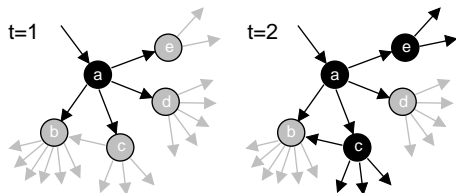
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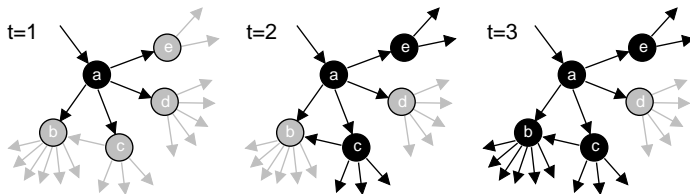
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If one individual is initially activated, what is the probability that an activation will spread over a network?

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What features of a network determine whether a cascade will occur or not?

First study random networks:

- ▶ Start with N nodes with a degree distribution p_k
- ▶ Nodes are randomly connected (carefully so)
- ▶ Aim: Figure out when activation will propagate
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Follow active links

- ▶ An active link is a link connected to an activated node.
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- ▶ We need to understand which nodes can be activated when only one of their neighbors becomes active.

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The most gullible

Vulnerables:

- ▶ We call individuals who can be activated by just one contact being active **vulnerables**
- ▶ The vulnerability condition for node i :

$$1/k_i \geq \phi_i$$

- ▶ Which means # contacts $k_i \leq \lfloor 1/\phi_i \rfloor$
- ▶ For global cascades on random networks, must have a *global cluster of vulnerables* ^[19]
- ▶ **Cluster of vulnerables = critical mass**
- ▶ Network story: 1 node \rightarrow critical mass \rightarrow everyone.

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- ▶ Follows from links being random + having k chances to connect to a node with degree k .
- ▶ Normalization:

$$\sum_{k=0}^{\infty} kP_k = \langle k \rangle = z$$

- ▶ So

$$P(\text{linked node has degree } k) = \frac{kP_k}{\langle k \rangle}$$

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Next: Vulnerability of linked node

- ▶ Linked node is **vulnerable** with probability

$$\beta_k = \int_{\phi'_*=0}^{1/k} f(\phi'_*) d\phi'_*$$

- ▶ If linked node is **vulnerable**, it produces $k - 1$ **new** outgoing active links
- ▶ If linked node is **not vulnerable**, it produces **no** active links.

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- ▶ If linked node is **not vulnerable**, it produces **no** active links.

Cascade condition

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Next: Vulnerability of linked node

- ▶ Linked node is **vulnerable** with probability

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Putting things together:

- ▶ Expected number of active edges produced by an active edge =

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So... for random networks with fixed degree distributions, cascades take off when:

$$\sum_{k=1}^{\infty} k(k-1)\beta_k P_k/z \geq 1.$$

- ▶ β_k = probability a degree k node is vulnerable.
- ▶ P_k = probability a node has degree k .

Two special cases:

- ▶ (1) Simple disease-like spreading succeeds: $\beta_k = \beta$

$$\beta \sum_{k=1}^{\infty} k(k-1)P_k/z \geq 1.$$

- ▶ (2) Giant component exists: $\beta = 1$

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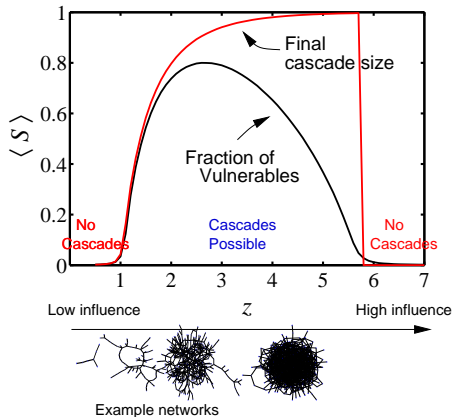
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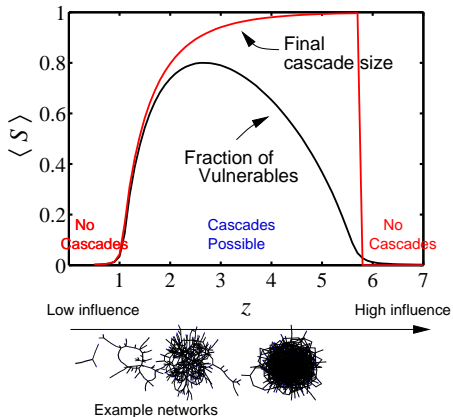
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Cascades on random networks



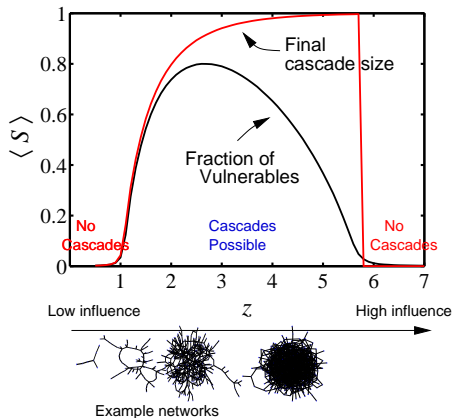
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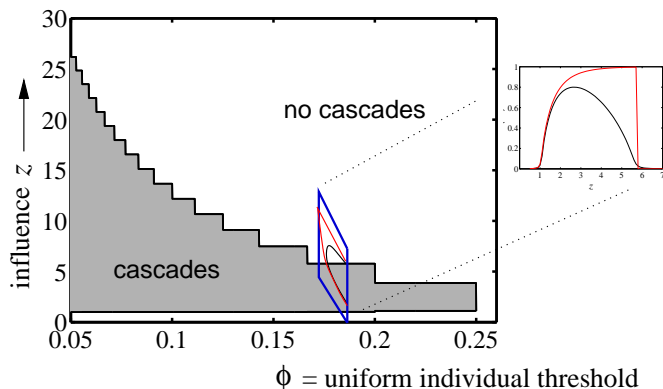
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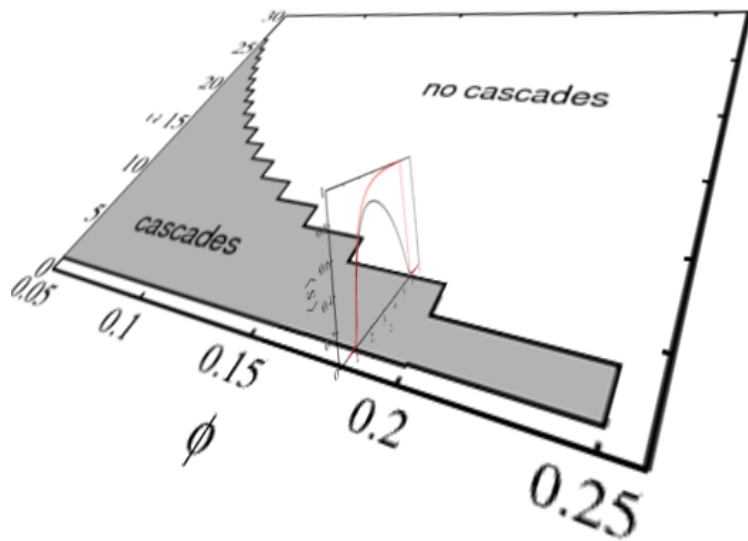
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Cascade window for random networks



- ▶ 'Cascade window' widens as threshold ϕ decreases.
- ▶ Lower thresholds enable spreading.

Cascade window for random networks



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Cascade window—summary

For our simple model of a uniform threshold:

1. **Low $\langle k \rangle$** : No cascades in poorly connected networks. No global clusters of any kind.
2. **High $\langle k \rangle$** : Giant component exists but not enough vulnerables.
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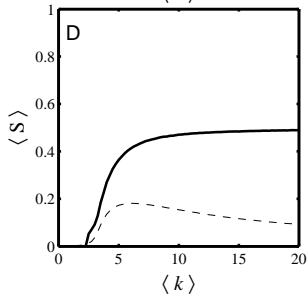
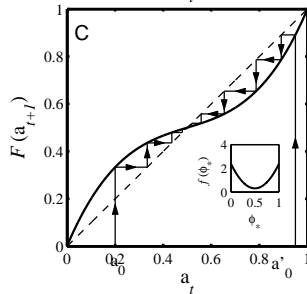
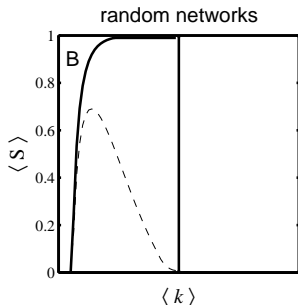
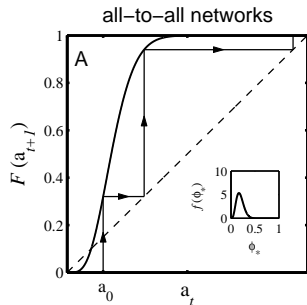
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All-to-all versus random networks

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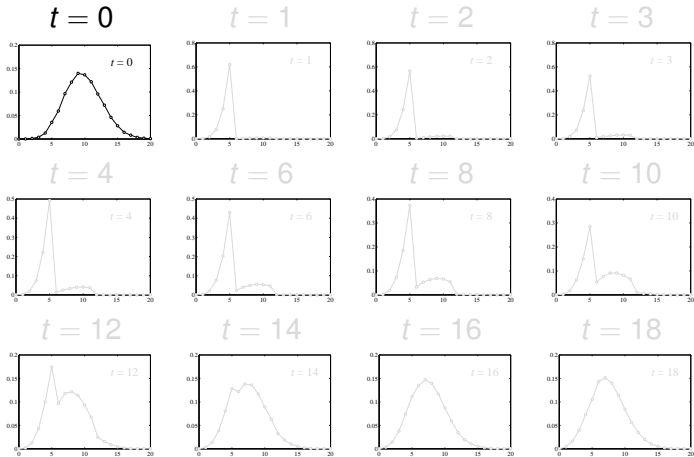


Early adopters—degree distributions

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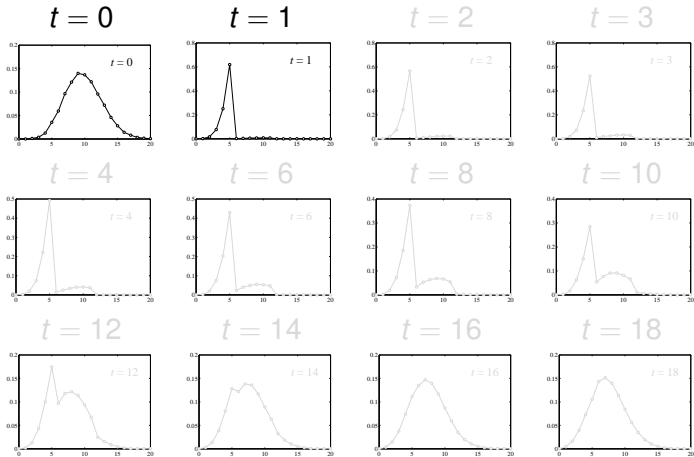
$P_{k,t}$ versus k

Early adopters—degree distributions

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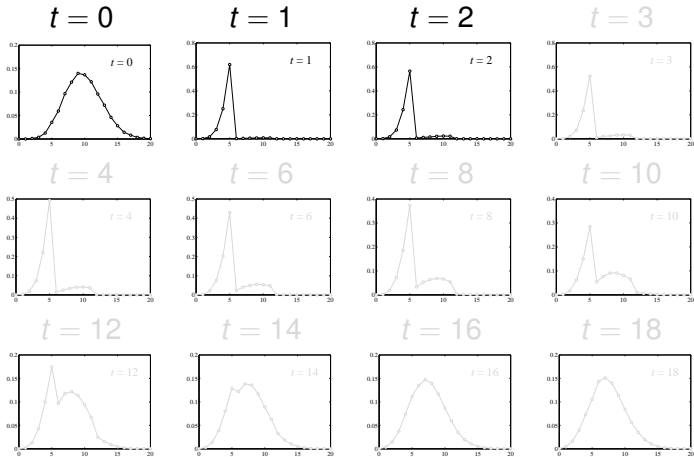
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Early adopters—degree distributions

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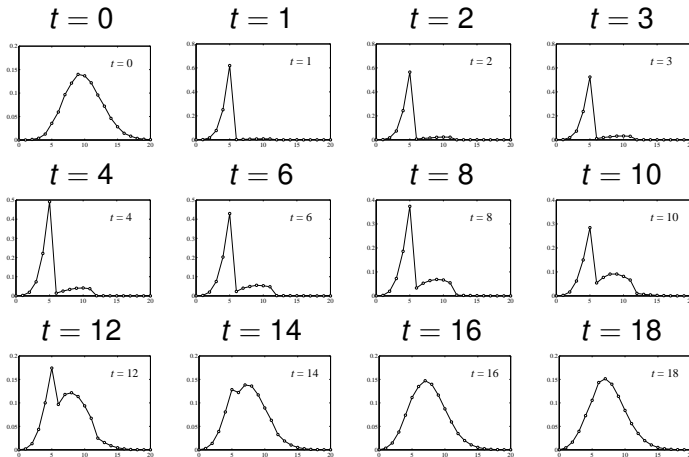
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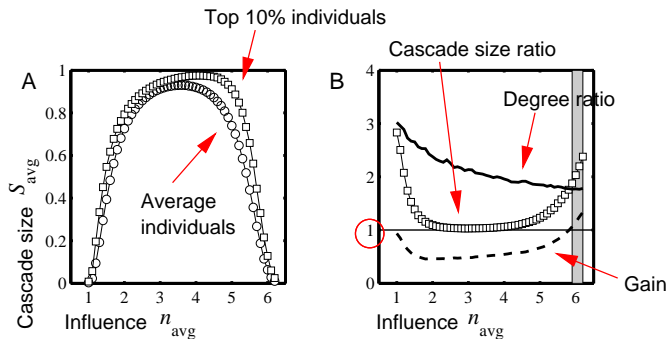
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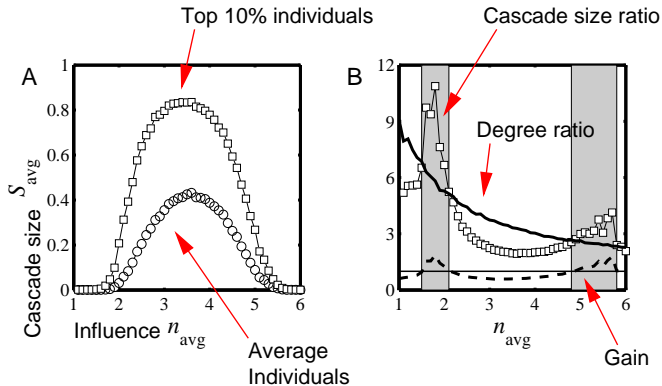
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The multiplier effect:



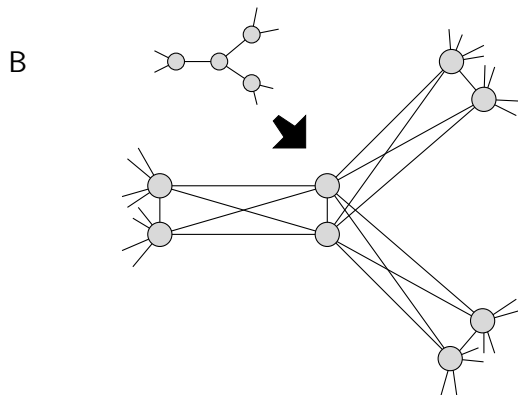
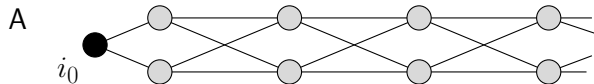
- ▶ Fairly uniform levels of individual influence.
- ▶ Multiplier effect is mostly below 1.

The multiplier effect:



- ▶ Skewed influence distribution example.

Special subnetworks can act as triggers



► $\phi = 1/3$ for all nodes

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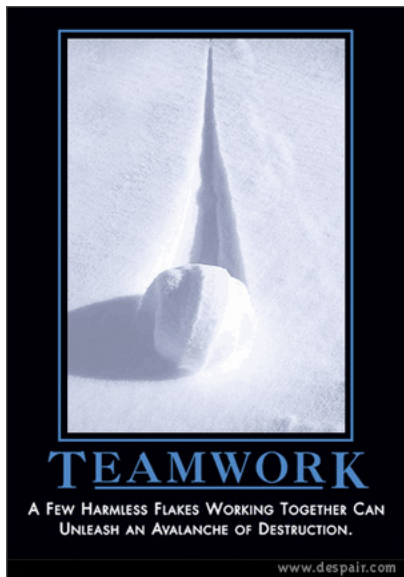
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Frame 56/88



The power of groups...



despair.com

“A few harmless flakes working together can unleash an avalanche of destruction.”

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Frame 57/88



- ▶ Assumption of sparse interactions is good
- ▶ Degree distribution is (generally) key to a network's function
- ▶ Still, random networks don't represent all networks
- ▶ Major element missing: **group structure**

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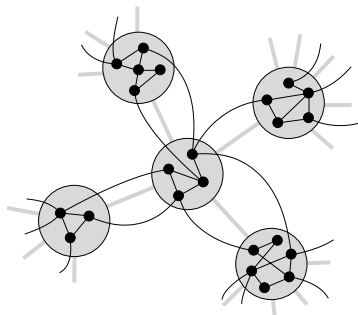
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Group structure—Ramified random networks

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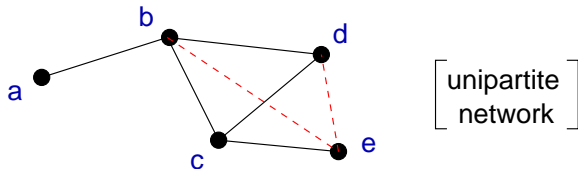
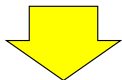
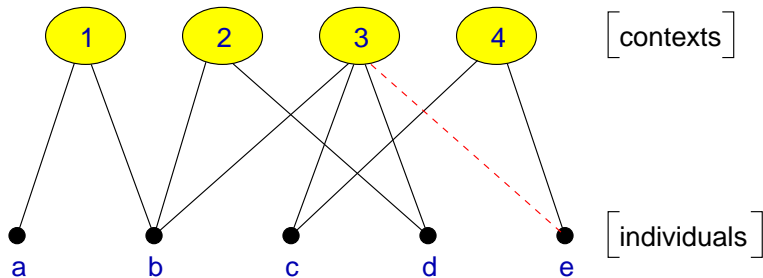
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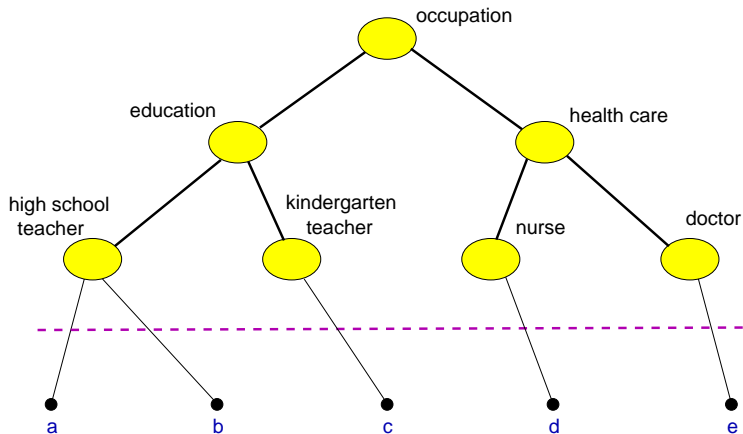


p = intergroup connection probability
 q = intragroup connection probability.

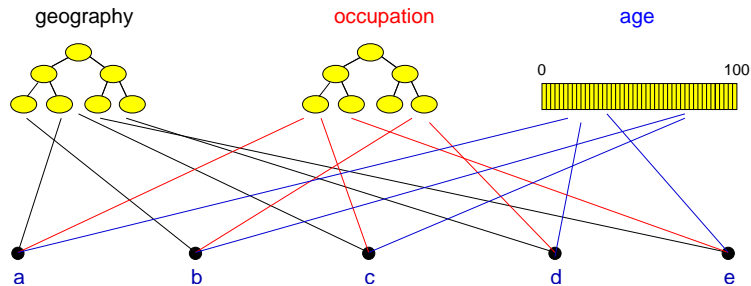
Bipartite networks



Context distance



Generalized affiliation model



(Blau & Schwartz, Simmel, Breiger)

Generalized affiliation model networks with triadic closure

- ▶ Connect nodes with probability $\propto \exp^{-\alpha d}$
where
 α = homophily parameter
and
 d = distance between nodes (height of lowest common ancestor)
- ▶ τ_1 = intergroup probability of friend-of-friend connection
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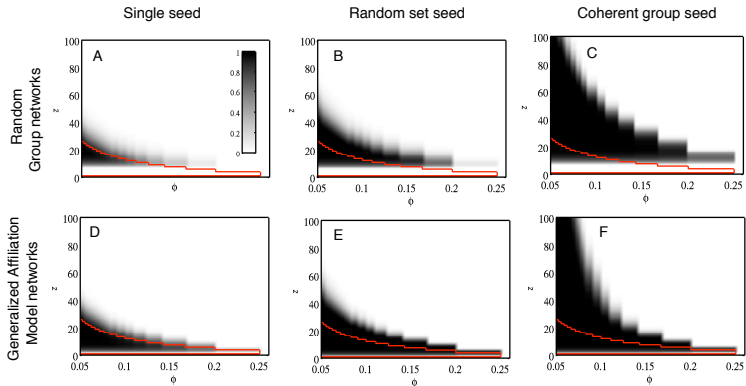
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Cascade windows for group-based networks

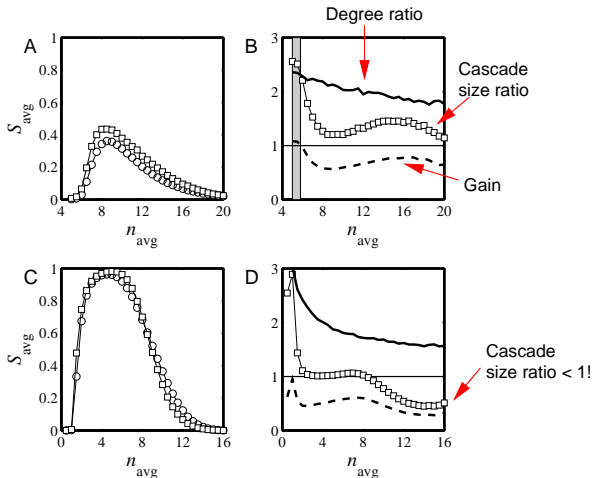
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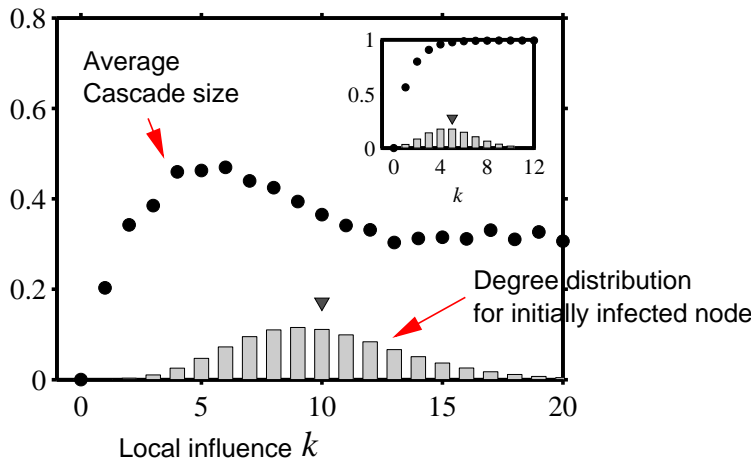


Multiplier effect for group-based networks:



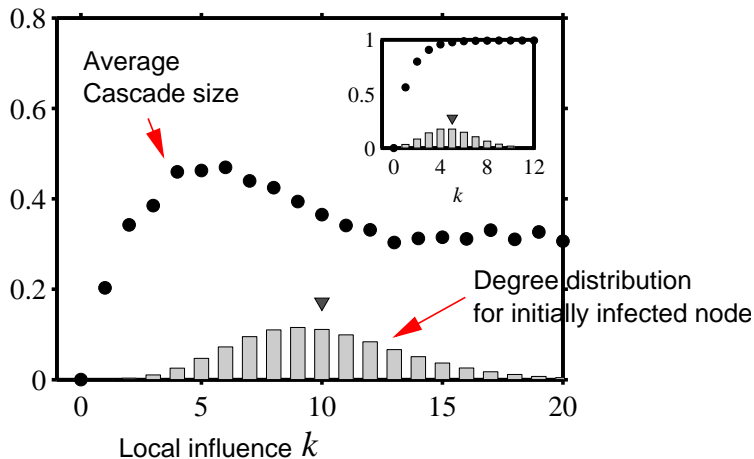
- ▶ Multiplier almost always below 1.

Assortativity in group-based networks



- ▶ The most connected nodes aren't always the most 'influential.'
- ▶ Degree assortativity is the reason.

Assortativity in group-based networks



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Summary

- ▶ **'Influential vulnerables'** are key to spread.
- ▶ Early adopters are mostly vulnerables.
- ▶ Vulnerable nodes important but not necessary.
- ▶ Groups may greatly facilitate spread.
- ▶ Seems that cascade condition is a global one.
- ▶ Most extreme/unexpected cascades occur in highly connected networks
- ▶ 'Influentials' are posterior constructs.
- ▶ Many potential influentials exist.

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Implications

- ▶ Focus on **the influential vulnerables**.
- ▶ Create entities that can be transmitted successfully through many individuals rather than broadcast from one 'influential.'
- ▶ Only **simple ideas** can spread by word-of-mouth.
(Idea of opinion leaders spreads well...)
- ▶ Want enough individuals who will adopt and display.
- ▶ Displaying can be **passive** = free (yo-yo's, fashion), or **active** = harder to achieve (political messages).
- ▶ Entities can be novel or designed to combine with others, e.g. block another one.

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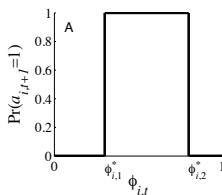
References

Chaotic contagion:

- ▶ What if individual response functions are not monotonic?
- ▶ Consider a simple deterministic version:
- ▶ Node i has an 'activation threshold' $\phi_{i,1}$
... and a 'de-activation threshold' $\phi_{i,2}$
- ▶ Nodes like to imitate but only up to a limit—they don't want to be like everyone else.

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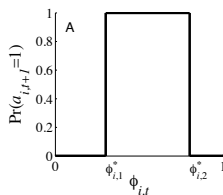
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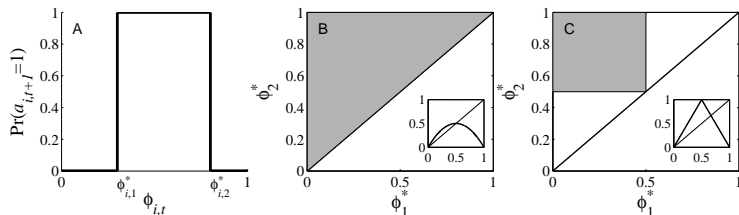


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Two population examples:



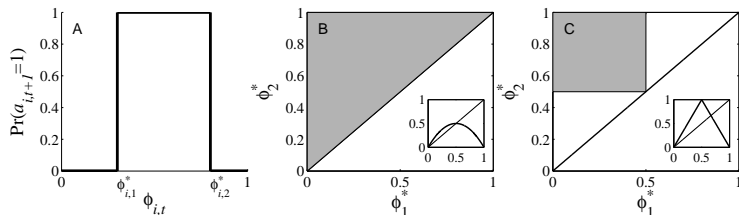
- ▶ Randomly select $(\phi_{i,1}, \phi_{i,2})$ from gray regions shown in plots B and C.
- ▶ Insets show composite response function averaged over population.
- ▶ We'll consider plot C's example: the tent map.

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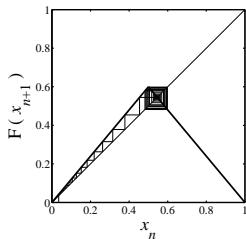
Definition of the tent map:

$$F(x) = \begin{cases} rx & \text{for } 0 \leq x \leq \frac{1}{2}, \\ r(1-x) & \text{for } \frac{1}{2} \leq x \leq 1. \end{cases}$$

- ▶ The usual business: look at how F iteratively maps the unit interval $[0, 1]$.

The tent map

Effect of increasing r from 1 to 2.



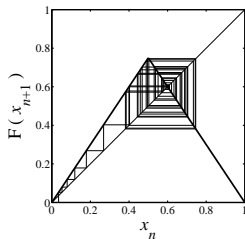
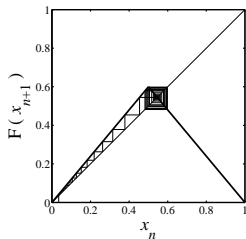
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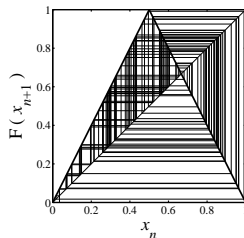
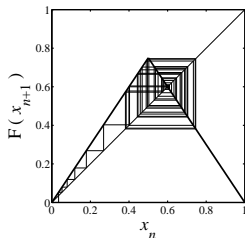
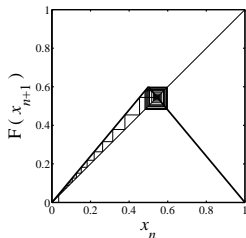
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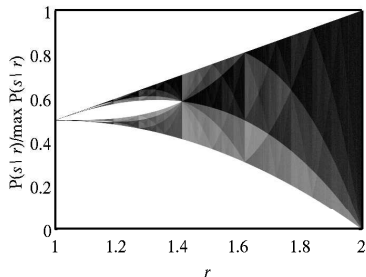
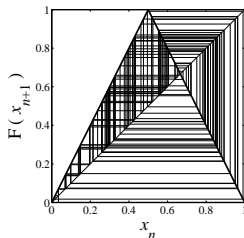
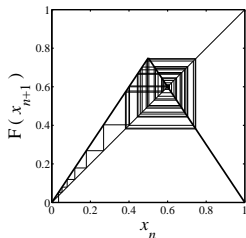
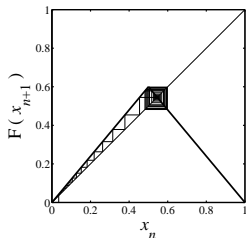
The tent map

Effect of increasing r from 1 to 2.



The tent map

Effect of increasing r from 1 to 2.



Orbit diagram:

Chaotic behavior increases as map slope r is increased.

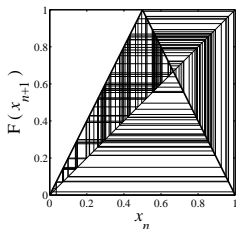
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Chaotic behavior

Take $r = 2$ case:



- ▶ What happens if nodes have limited information?
- ▶ As before, allow interactions to take place on a sparse random network.
- ▶ Vary average degree $z = \langle k \rangle$, a measure of information

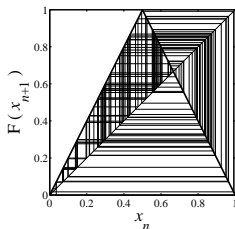
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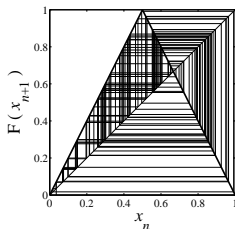
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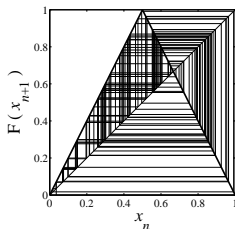
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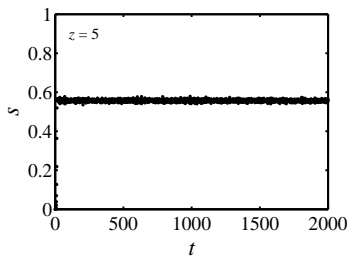
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Invariant densities—stochastic response functions

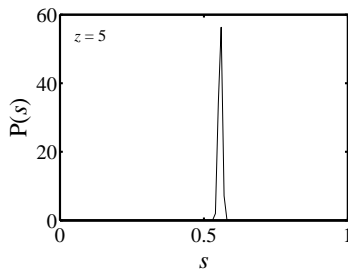
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activation time series



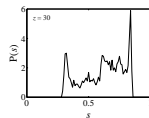
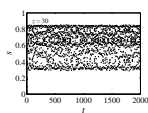
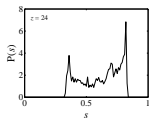
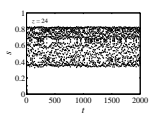
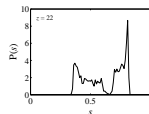
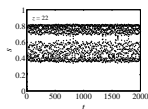
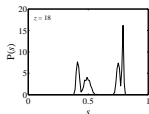
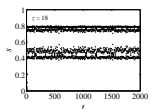
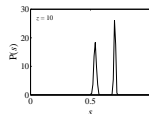
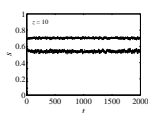
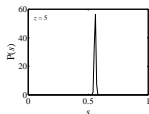
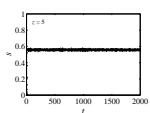
activation density

Invariant densities—stochastic response functions

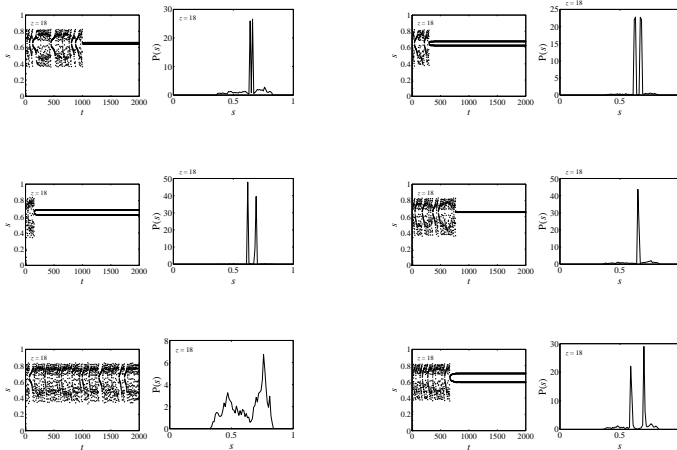
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Invariant densities—deterministic response functions for one specific network with $\langle k \rangle = 18$



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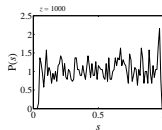
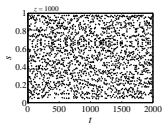
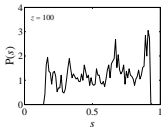
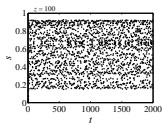
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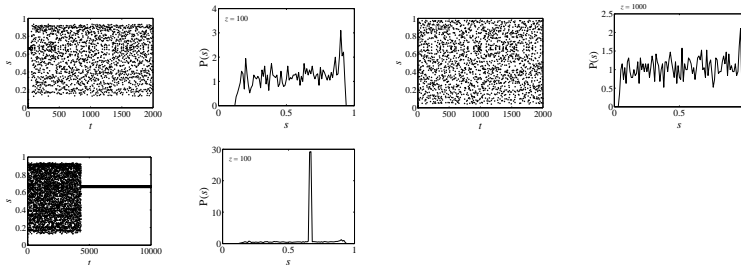
Trying out higher values of $\langle k \rangle$...

Invariant densities—deterministic response functions

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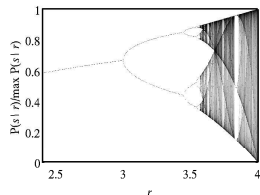
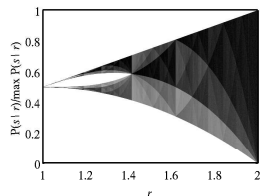
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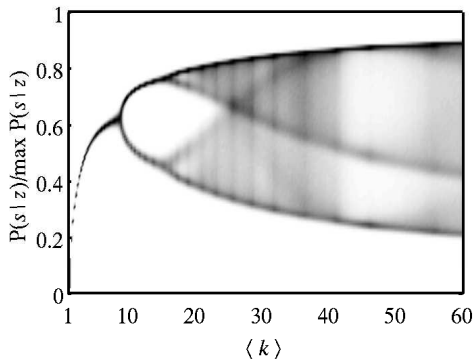


Trying out higher values of $\langle k \rangle \dots$

Connectivity leads to chaos:



Stochastic response functions:



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Chaotic behavior in coupled systems

Coupled maps are well explored
(Kaneko/Kuramoto):

$$x_{i,n+1} = f(x_{i,n}) + \sum_{j \in \mathcal{N}_i} \delta_{i,j} f(x_{j,n})$$

► \mathcal{N}_i = neighborhood of node i

1. Node states are **continuous**
2. Increase δ and neighborhood size $|\mathcal{N}_i|$
 ⇒ **synchronization**

But for contagion model:

1. Node states are **binary**
2. **Asynchrony remains** as connectivity increases

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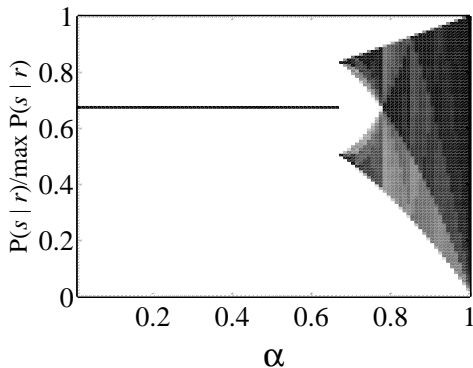
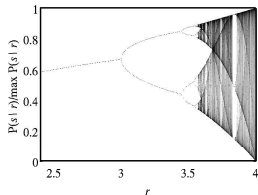
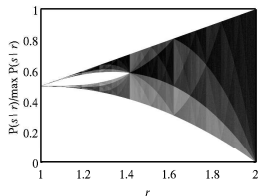
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Bifurcation diagram: Asynchronous updating






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