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Prof. Peter Dodds

Department of Mathematics & Statistics
Center for Complex Systems
Vermont Advanced Computing Center
University of Vermont



















Social Contagion Models

Background

Granovetter's model Network version

Groups





Outline

- Social Contagion Models
 - Background
 - Granovetter's model
 - Network version
 - Groups
 - Chaos

References

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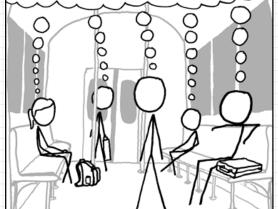
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LOOK AT THESE PEOPLE. GLASSY-EYED AUTOMATONS GOING ABOUT THEIR DAILY LIVES, NEVER STOPPING TO LOOK AROUND AND THINK! I'M THE ONLY CONSCIOUS HUMAN IN A WORLD OF SHEEP.



http://xkcd.com/610/ (⊞)

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Examples abound

- fashion
- striking
- ► smoking (⊞) [6]
- residential segregation [16]
- ipods
- ▶ obesity (⊞) ^[5]

- Harry Potter
- voting
- gossip
- Rubik's cube **
- religious beliefs
- leaving lectures

SIR and SIRS contagion possible

Classes of behavior versus specific behavior: dieting

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Evolving network stories (Christakis and Fowler):

- ► The spread of quitting smoking (⊞) [6]
- The spread of spreading (⊞) [5]
- ▶ Also: happiness (⊞) [8], loneliness, ...
- ► The book: Connected: The Surprising Power of Our Social Networks and How They Shape Our Lives (⊞)

Controversy:

- ► Are your friends making you fat? (⊞) (Clive Thomspon, NY Times, September 10, 2009).
- ► Everything is contagious (⊞)—Doubts about the social plague stir in the human superorganism (Dave Johns, Slate, April 8, 2010).

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Two focuses for us

- ► Widespread media influence
- Word-of-mouth influence



We need to understand influence

- Who influences whom? Very hard to measure...
- What kinds of influence response functions are there?
- Are some individuals super influencers? Highly popularized by Gladwell [9] as 'connectors'
- The infectious idea of opinion leaders (Katz and Lazarsfeld) [13]

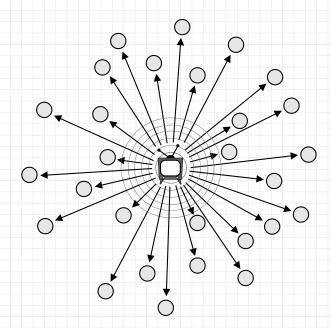
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The hypodermic model of influence



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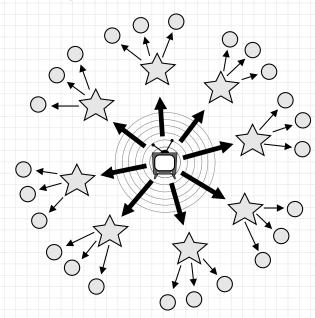
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The two step model of influence [13]



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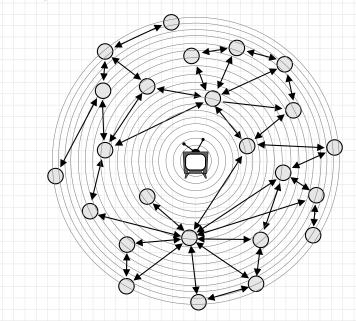
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The general model of influence



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Why do things spread?

- Because of properties of special individuals?
- Or system level properties?
- 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...
- Reasons for success are usually ascribed to intrinsic properties (e.g., Mona Lisa)
- System/group properties harder to understand
- Always good to examine what is said before and after the fact...

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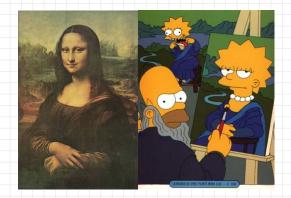
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The Mona Lisa



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

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The completely unpredicted fall of Eastern Europe



Timur Kuran: [14, 15] "Now Out of Never: The Element of Surprise in the East European Revolution of 1989"

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The dismal predictive powers of editors...



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Messing with social connections

- Ads based on message content (e.g., Google and email)
- Buzz media
- ► Facebook's advertising: Beacon (⊞)

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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
- 6. Scarcity: The Rule of the Few

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- ▶ Reciprocation: Free samples, Hare Krishnas
- Commitment and Consistency: Hazing
- ► Social Proof: Catherine Genovese, Jonestown
- Liking: Separation into groups is enough to cause problems.
- Authority: Milgram's obedience to authority experiment.
- Scarcity: Prohibition.

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- Cialdini's modes are heuristics that help up us get through life.
- Useful but can be leveraged...





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Other acts of influence

- Conspicuous Consumption (Veblen, 1912)
- Conspicuous Destruction (Potlatch)



Some important models

- ► T ipping models—Schelling (1971) [16, 17, 18]
 - Simulation on checker boards
 - Idea of thresholds
 - ▶ Explore the Netlogo (⊞) implementation [21]
- ► Threshold models—Granovetter (1978) [10]
- ► Herding models—Bikhchandani, Hirschleifer, Welch (1992) [1, 2]
 - Social learning theory, Informational cascades,...

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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... (unrealistic).
- Assumption: level of influence per person is uniform (unrealistic).

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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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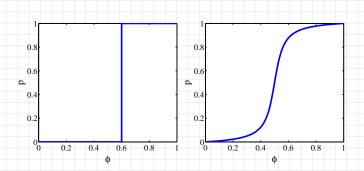
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Granovetter's Threshold model—definitions

- 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.



Threshold models



- Example threshold influence response functions: deterministic and stochastic
- ϕ = fraction of contacts 'on' (e.g., rioting)
- ► Two states: S and I.

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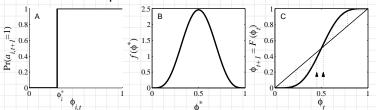
$$\phi_{t+1} = \int_0^{\phi_t} f(\phi_*) \mathrm{d}\phi_* = F(\phi_*)|_0^{\phi_t} = F(\phi_t)$$

 \rightarrow lterative maps of the unit interval [0, 1].





Action based on perceived behavior of others.



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

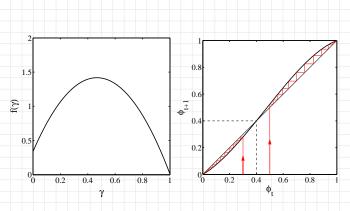
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Threshold models



Another example of critical mass model...

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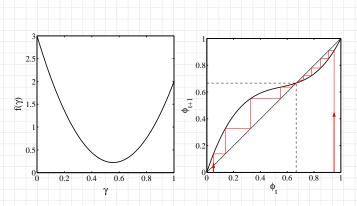
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Threshold models



Example of single stable state model

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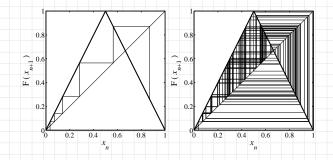
Implications for collective action theory:

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



Threshold models

Chaotic behavior possible [12, 11]



- Period doubling arises as map amplitude r is increased.
- Synchronous update assumption is crucial

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References

Many years after Granovetter and Soong's work:

"A simple model of global cascades on random networks" D. J. Watts, Proc. Natl. Acad. Sci., 2002 [20]

- Mean field model → network model
- Individuals now have a limited view of the world



 Interactions between individuals now represented by a network

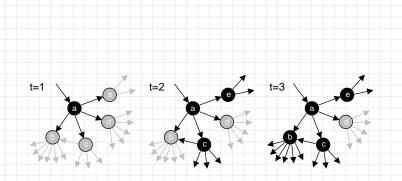
- Network is sparse
- Individual i has k_i contacts
- Influence on each link is reciprocal and of unit weight
- **Each** individual *i* has a fixed threshold ϕ_i
- Individuals repeatedly poll contacts on network
- Synchronous, discrete time updating
- Individual *i* becomes active when fraction of active contacts $\frac{a_i}{k_i} \ge \phi_i$
- Individuals remain active when switched (no recovery = SI model)

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Threshold model on a network



▶ All nodes have threshold $\phi = 0.2$.

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The Cascade Condition:

- 1. If one individual is initially activated, what is the probability that an activation will spread over a network?
- 2. 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
- Determine a cascade condition

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- An active link is a link connected to an activated node.
- If an infected link leads to at least 1 more infected link, then activation spreads.
- We need to understand which nodes can be activated when only one of their neigbors becomes active.

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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* [20]
- Cluster of vulnerables = critical mass
- Network story: 1 node → critical mass → everyone.

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Cascade condition

Back to following a link:

- A randomly chosen link, traversed in a random direction, leads to a degree k node with probability ∝ kP_k.
- ► Follows from there being *k* ways to connect to a node with degree *k*.
- Normalization:

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

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

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

- ► 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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Putting things together:

Expected number of active edges produced by an active edge:

$$R = \sum_{k=1}^{\infty} \frac{(k-1) \cdot \beta_k \cdot \frac{kP_k}{\langle k \rangle}}{\text{success}} + \underbrace{0 \cdot (1-\beta_k) \cdot \frac{kP_k}{\langle k \rangle}}_{\text{failure}}$$

$$=\sum_{k=1}^{\infty}(k-1)\cdot\beta_k\cdot\frac{kP_k}{\langle k\rangle}$$

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

$$\sum_{k=1}^{\infty} (k-1) \cdot \beta_k \cdot \frac{k P_k}{\langle k \rangle} \geq 1.$$

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



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

$$\beta \cdot \sum_{k=1}^{\infty} (k-1) \cdot \frac{kP_k}{\langle k \rangle} \geq 1.$$

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

$$1 \cdot \sum_{k=1}^{\infty} (k-1) \cdot \frac{kP_k}{\langle k \rangle} \ge 1.$$

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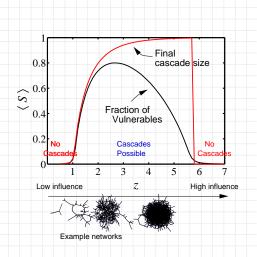
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Cascades on random networks



 Cascades occur only if size of max vulnerable cluster
 0.

- System may be 'robust-yet-fragile'.
- 'Ignorance' facilitates spreading.

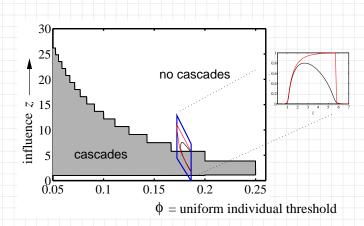
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Cascade window for random networks



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

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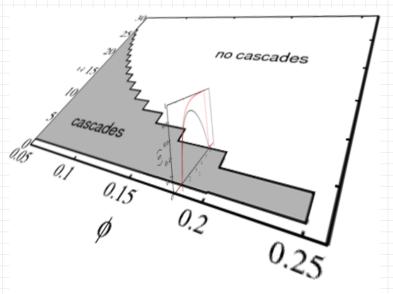
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Cascade window for random networks



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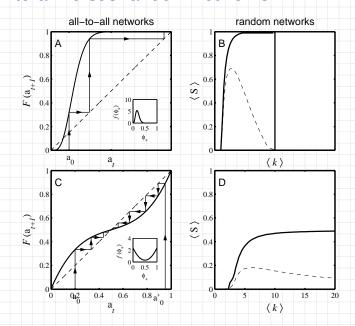
For our simple model of a uniform threshold:

- Low (k): No cascades in poorly connected networks.
 No global clusters of any kind.
- 2. High $\langle k \rangle$: Giant component exists but not enough vulnerables.
- 3. Intermediate $\langle k \rangle$: Global cluster of vulnerables exists. Cascades are possible in "Cascade window."





All-to-all versus random networks



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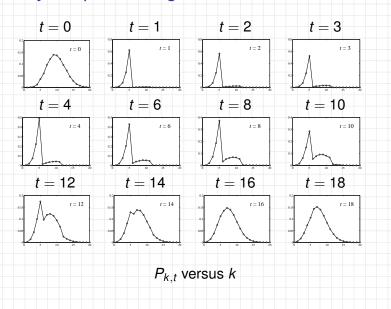
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Early adopters—degree distributions



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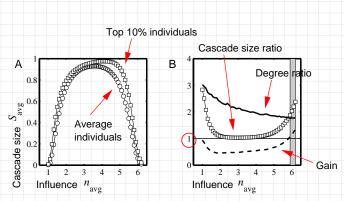
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- Fairly uniform levels of individual influence.
- Multiplier effect is mostly below 1.

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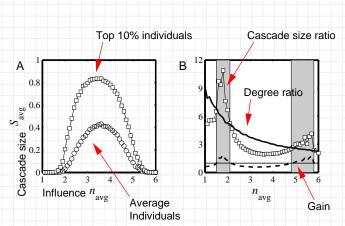
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The multiplier effect:



Skewed influence distribution example.

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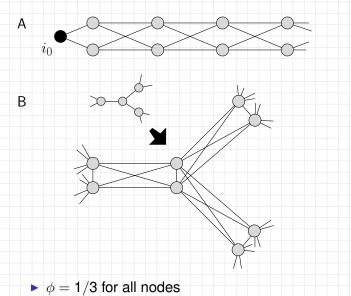
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Special subnetworks can act as triggers



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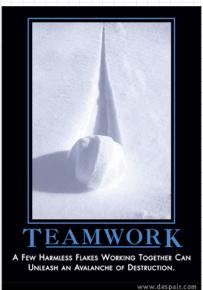
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The power of groups...



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

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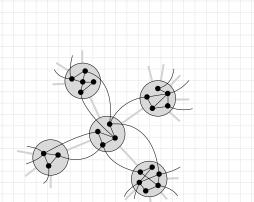




- Assumption of sparse interactions is good
- Degree distribution is (generally) key to a network's function
- Still, random networks don't represent all networks
- Major element missing: group structure



Group structure—Ramified random networks



p = intergroup connection probability q = intragroup connection probability.

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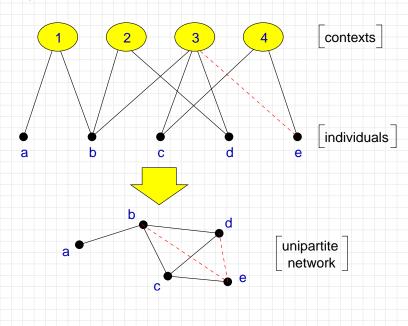
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Bipartite networks



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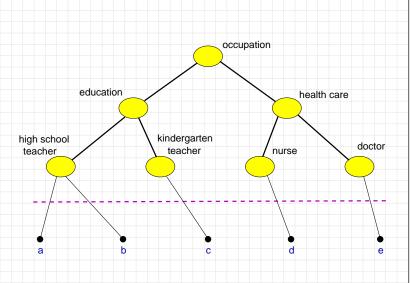
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Context distance



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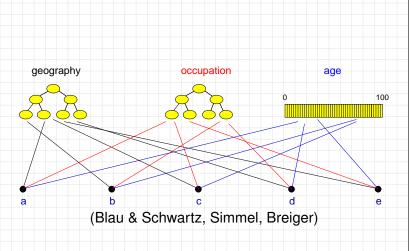
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Generalized affiliation model



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- ► 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
- au_2 = intragroup probability of friend-of-friend connection

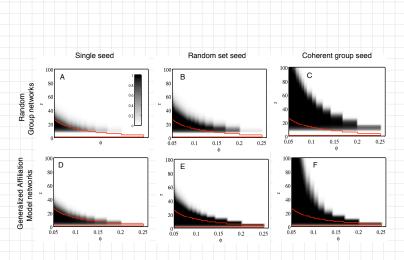
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Cascade windows for group-based networks



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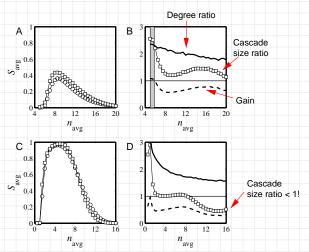
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Multiplier effect for group-based networks:



Multiplier almost always below 1.

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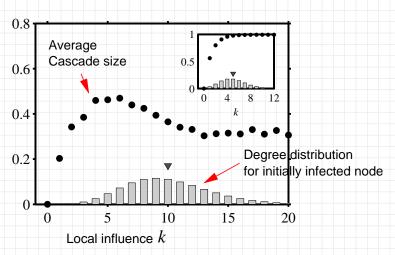
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Assortativity in group-based networks



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

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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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Social contagion

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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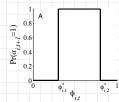




What if individual response functions are not monotonic?

Consider a simple deterministic version:

- Node i has an 'activation threshold' φ_{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.



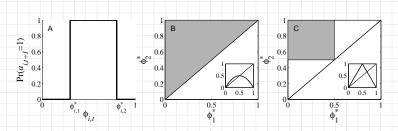
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- ▶ Randomly select $(\phi_{i,1}, \phi_{i,2})$ from gray regions shown in plots B and C.
- Insets show composite response function averaged over population.
- We'll consider plot C's example: the tent map.





Definition of the tent map:

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

► The usual business: look at how *F* iteratively maps the unit interval [0, 1].

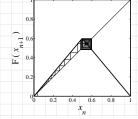




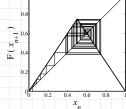


The tent map

Effect of increasing *r* from 1 to 2.

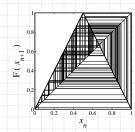


1.2



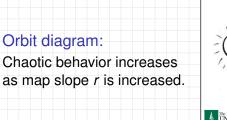
1.8

1.6





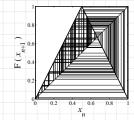
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Take r = 2 case:



- 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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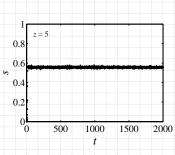
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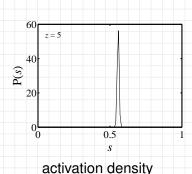




Invariant densities—stochastic response functions



activation time series



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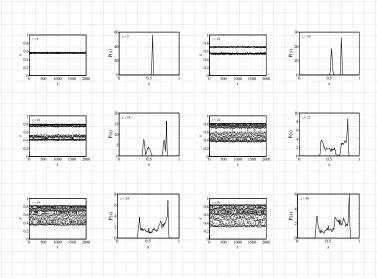
Chaos References







Invariant densities—stochastic response functions



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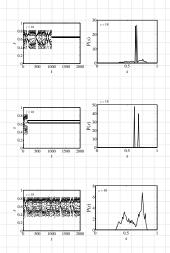


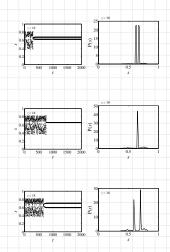




Invariant densities—deterministic response functions for one specific network with

 $\langle k \rangle = 18$





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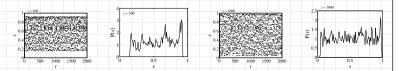
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Invariant densities—stochastic response functions



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

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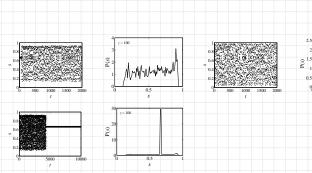
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Invariant densities—deterministic response functions



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

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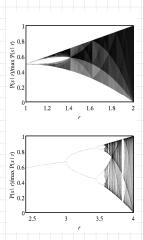
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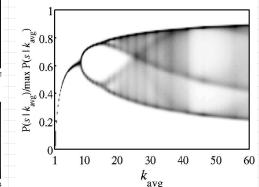




Connectivity leads to chaos:



Stochastic response functions:



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Coupled maps are well explored (Kaneko/Kuramoto):

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

- $\triangleright \mathcal{N}_i$ = neighborhood of node *i*
- Node states are continuous
- 2. Increase δ and neighborhood size $|\mathcal{N}|$

⇒ synchronization

But for contagion model:

- Node states are binary
- 2. Asynchrony remains as connectivity increases

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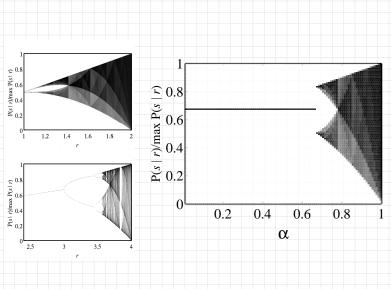
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Bifurcation diagram: Asynchronous updating



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