Social Contagion

Principles of Complex Systems
CSYS/MATH 300, Fall, 2011

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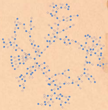
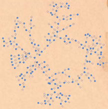
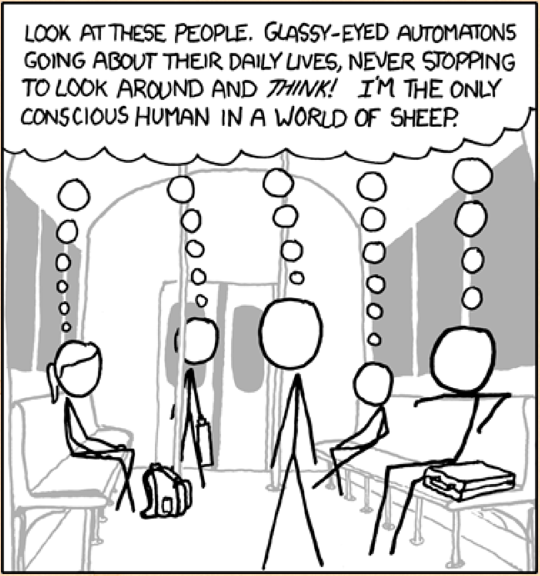


Outline



Social Contagion



http://xkcd.com/610/ (⊞)

Social Contagion

Social Contagion

Social Contagion

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

Framingham heart study:

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).

Social Contagion

Social Contagion

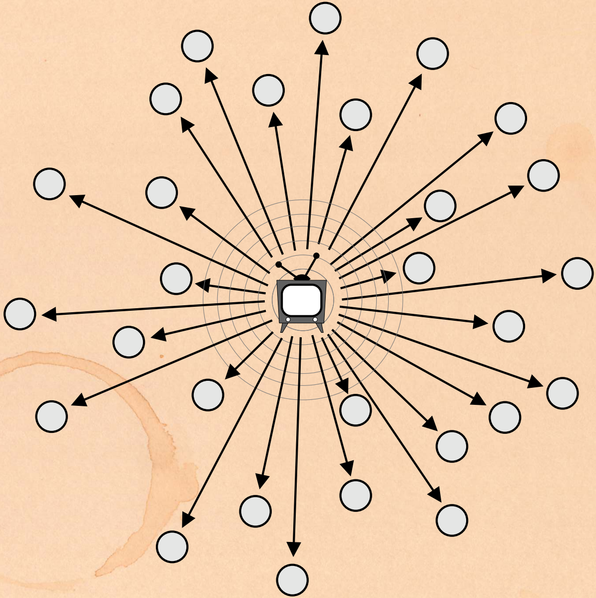
Two focuses for us

- Widespread media influence
- Word-of-mouth influence

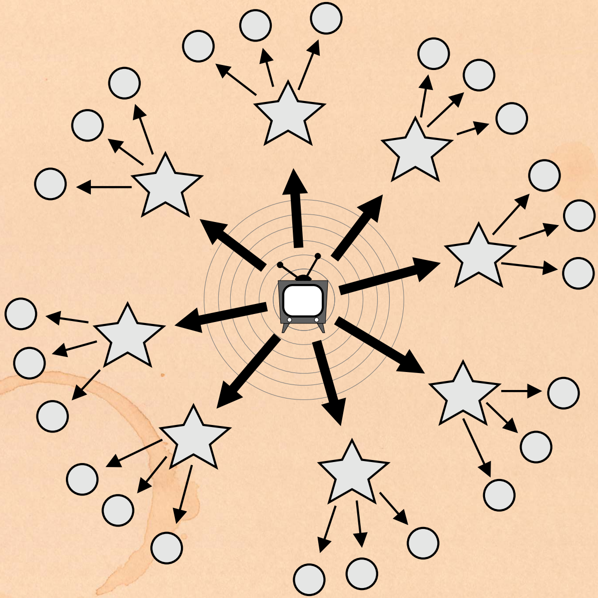
We need to understand influence

- 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]

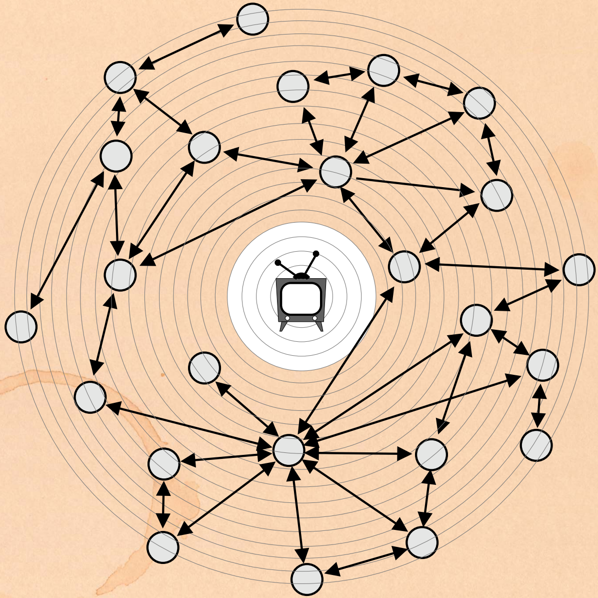
The hypodermic model of influence



The two step model of influence [13]



The general model of influence



Social Contagion

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...

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, ...

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”

The dismal predictive powers of editors...

Social Contagion

Messing with social connections

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

Getting others to do things for you

A very good book: 'Influence' [7] by Robert Cialdini (⊞)

Six modes of influence

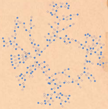
1. Reciprocation: *The Old Give and Take... and Take*
   e.g., Free samples, Hare Krishnas.
2. Commitment and Consistency: *Hobgoblins of the Mind*
   e.g., Hazing.
3. Social Proof: *Truths Are Us*
   e.g., Catherine Genovese, Jonestown
4. Liking: *The Friendly Thief*
   Separation into groups is enough to cause problems.
5. Authority: *Directed Deference*
   Milgram's obedience to authority experiment.
6. Scarcity: *The Rule of the Few*
   Prohibition.

Social contagion

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

Other acts of influence:

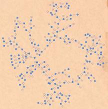
- Conspicuous Consumption (Veblen, 1912)
- Conspicuous Destruction (Potlatch)



Social Contagion

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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Social contagion models

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).

Social Contagion

Some possible origins of thresholds:

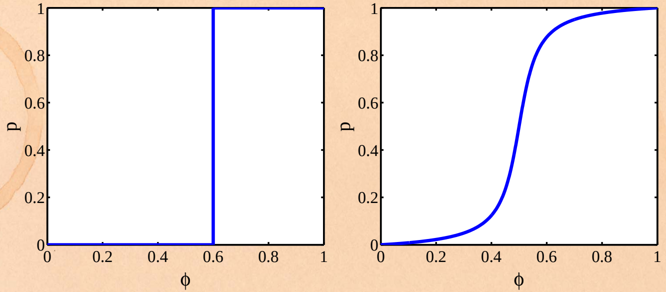
- Desire to coordinate, to conform.
- Lack of information: impute the worth of a good or behavior based on degree of adoption (social proof)
- Economics: Network effects or network externalities
- Externalities = Effects on others not directly involved in a transaction
- Examples: telephones, fax machine,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

Social Contagion

Granovetter's Threshold model—definitions

- ϕ^* = threshold of an individual.
- $f(\phi_*)$ = distribution of thresholds in a population.
- $F(\phi_*)$ = cumulative distribution = $\int_{\phi'_*=0}^{\phi_*} f(\phi'_*)\mathrm{d}\phi'_*$
- ϕ_t = fraction of people 'rioting' at time step t.

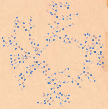
Threshold models



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

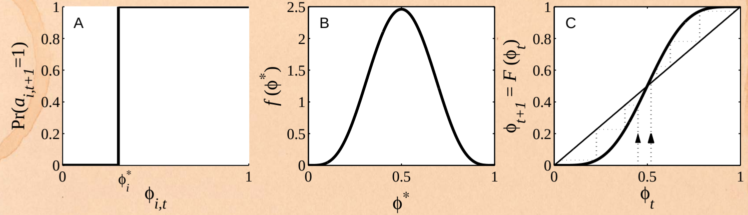
Threshold models

- At time $t + 1$, fraction rioting = fraction with $\phi_* \leq \phi_t$.
- $$\phi_{t+1} = \int_0^{\phi_t} f(\phi_*) \mathrm{d}\phi_* = F(\phi_*)|_0^{\phi_t} = F(\phi_t)$$
- $\Rightarrow$ Iterative maps of the unit interval $[0, 1]$.



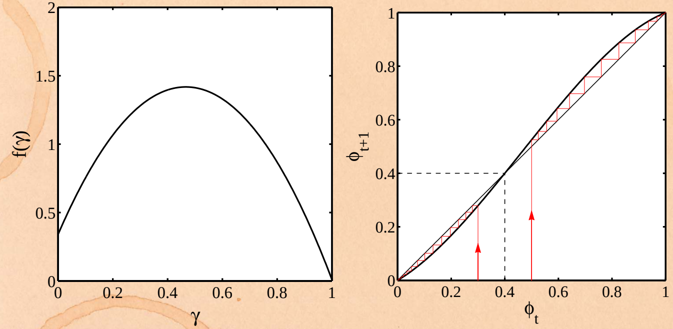
Threshold models

Action based on perceived behavior of others.



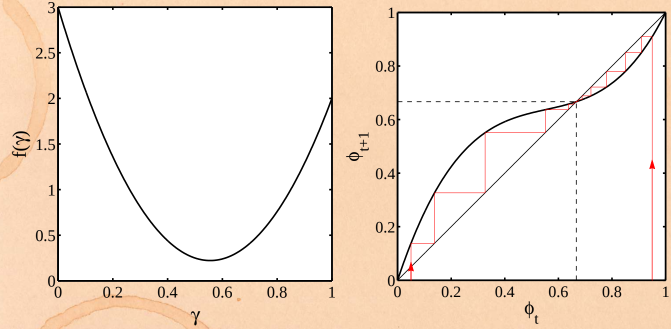
- Two states: S and I.
- ϕ = fraction of contacts 'on' (e.g., rioting)
- Discrete time update (strong assumption!)
- This is a Critical mass model

Threshold models



- Another example of critical mass model...

Threshold models

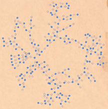


- Example of single stable state model

Threshold models

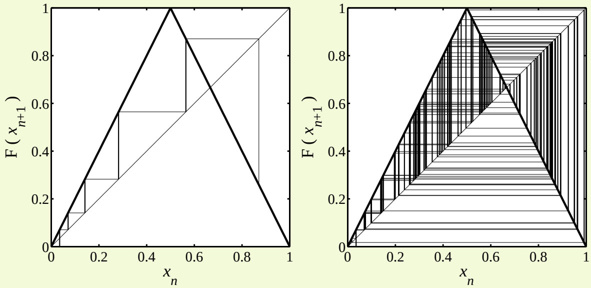
Implications for collective action theory:

1. Collective uniformity $\not\Rightarrow$ individual uniformity
2. Small individual changes $\Rightarrow$ large global changes



Threshold models

Chaotic behavior possible [12, 11]



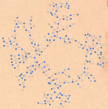
- Period doubling arises as map amplitude r is increased.
- Synchronous update assumption is crucial

Threshold model on a network

Many years after Granovetter and Soong's work:

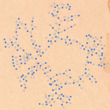
"A simple model of global cascades on random networks"
D. J. Watts. Proc. Natl. Acad. Sci., 2002 [20]

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

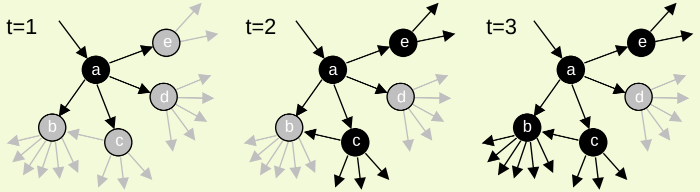


Threshold model on a network

- 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
- Synchronous, discrete time updating
- Individual i becomes active when fraction of active contacts $\frac{a_i}{k_i} \geq \phi_i$
- Individuals remain active when switched (no recovery = SI model)



Threshold model on a network



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

Snowballing

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?

Snowballing

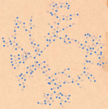
First study random networks:

- 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

Snowballing

Follow active links

- 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.



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

Cascade condition

Back to following a link:

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

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

- So

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

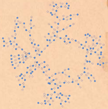
Cascade condition

Next: Vulnerability of linked node

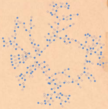
- Linked node is vulnerable with probability

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

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



Cascade condition



Putting things together:

- Expected number of active edges produced by an active edge:

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

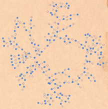
$$= \sum_{k=1}^{\infty} (k-1) \cdot \beta_k \cdot \frac{kP_k}{\langle k \rangle}$$

Cascade condition

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

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

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



Cascade condition

Two special cases:

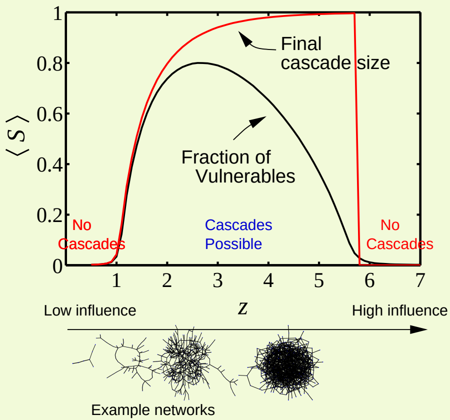
- (1) Simple disease-like spreading succeeds: $\beta_k = \beta$

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

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

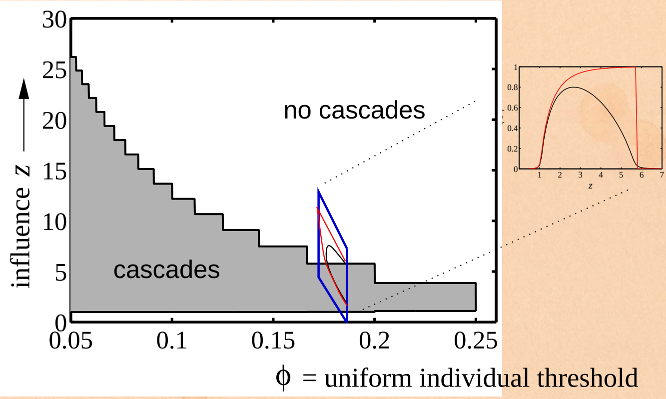
$$1 \cdot \sum_{k=1}^{\infty} (k-1) \cdot \frac{kP_k}{\langle k \rangle} \geq 1.$$

Cascades on random networks

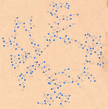


- Cascades occur only if size of max vulnerable cluster > 0.
- System may be 'robust-yet-fragile'.
- 'Ignorance' facilitates spreading.

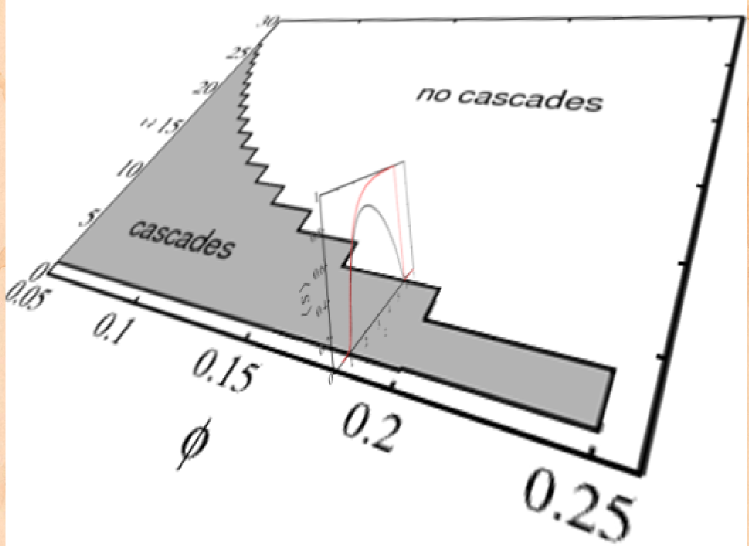
Cascade window for random networks



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



Cascade window for random networks

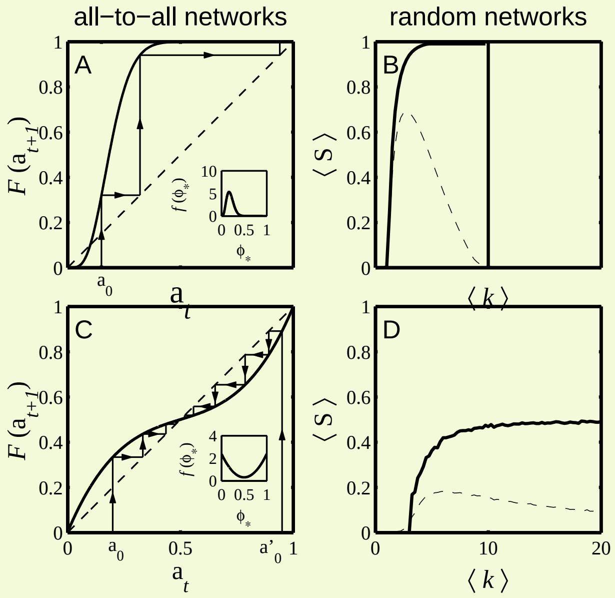


Cascade window—summary

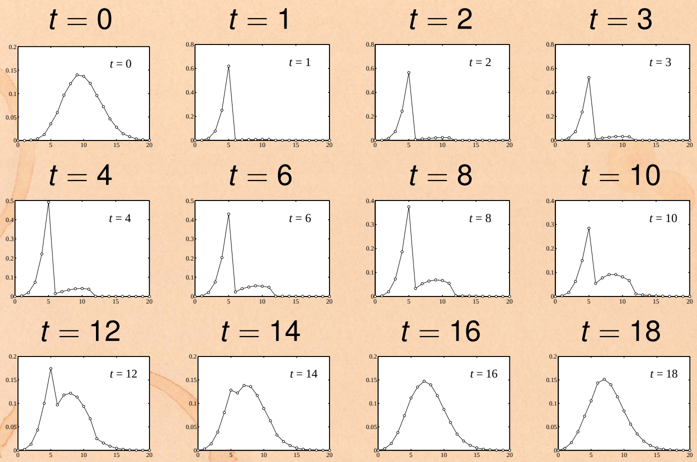
For our simple model of a uniform threshold:

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.
3. Intermediate $\langle k \rangle$: Global cluster of vulnerables exists. Cascades are possible in "Cascade window."

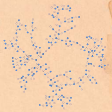
All-to-all versus random networks



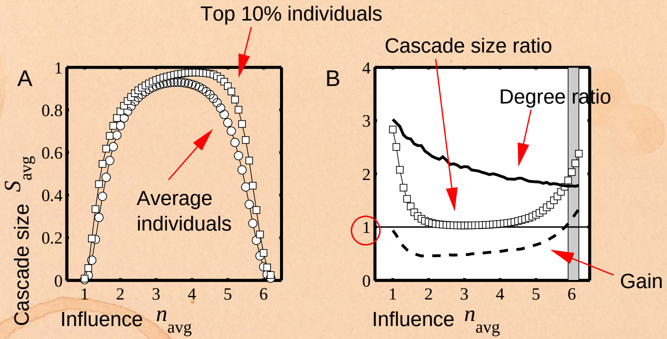
Early adopters—degree distributions



$P_{k,t}$ versus k

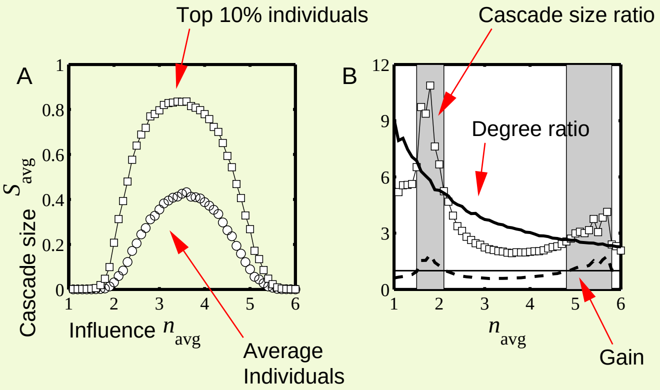


The multiplier effect:



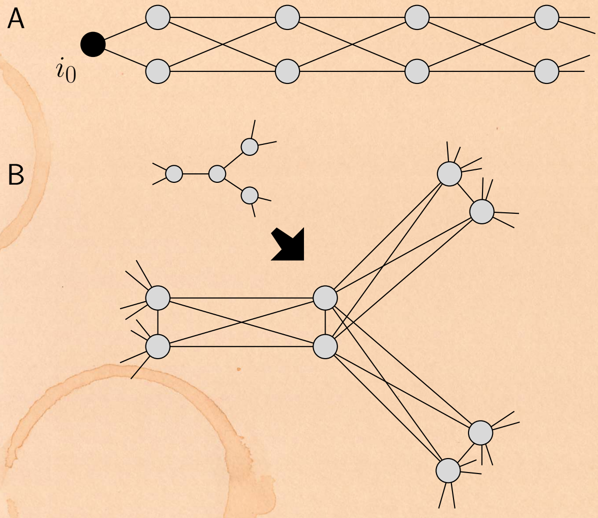
- Fairly uniform levels of individual influence.
- Multiplier effect is mostly below 1.

The multiplier effect:



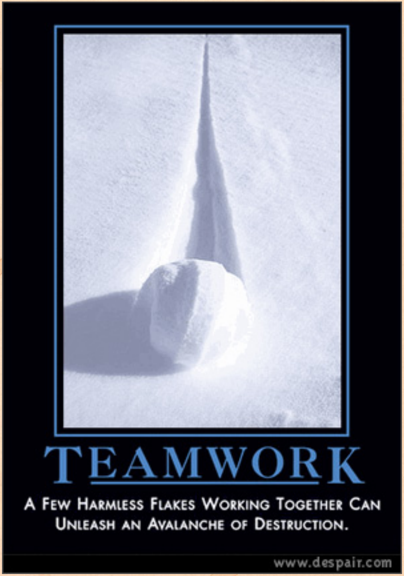
- Skewed influence distribution example.

Special subnetworks can act as triggers



- $\phi = 1/3$ for all nodes

The power of groups...



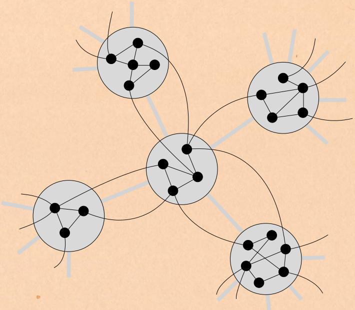
despair.com

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

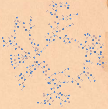
Extensions

- 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

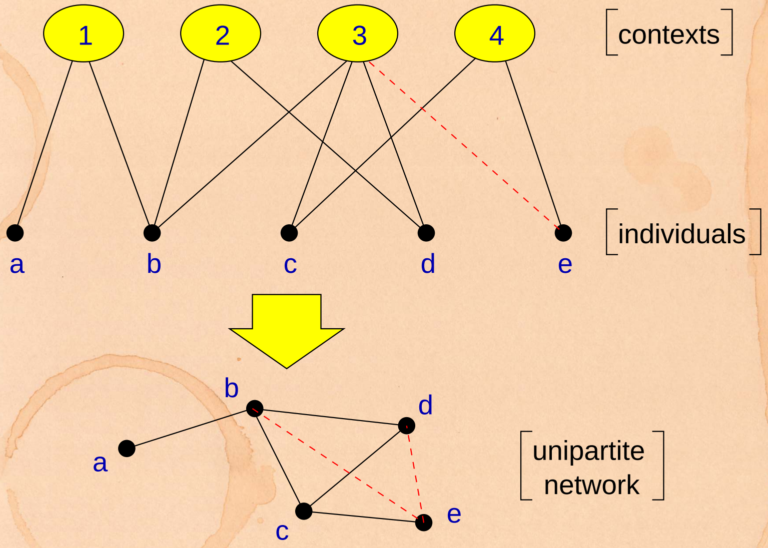
Group structure—Ramified random networks

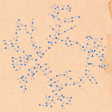


p = intergroup connection probability
q = intragroup connection probability.

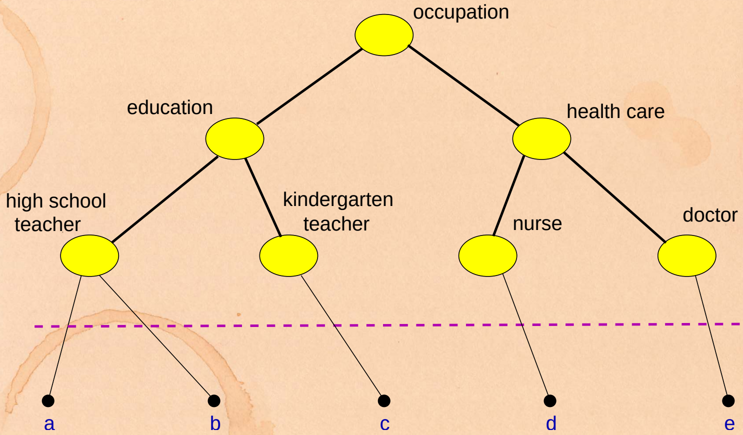


Bipartite networks

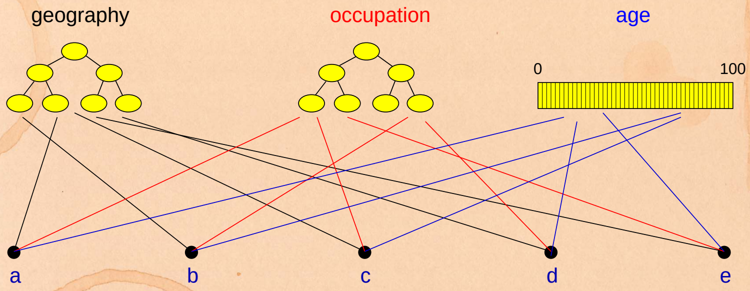




Context distance



Generalized affiliation model

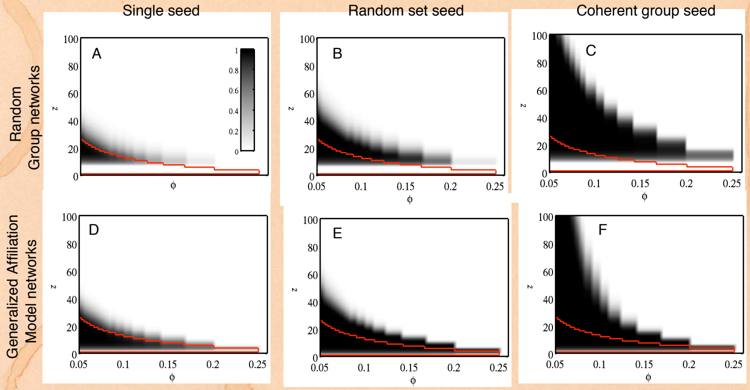


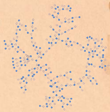
(Blau & Schwartz, Simmel, Breiger)

Generalized affiliation model networks with triadic closure

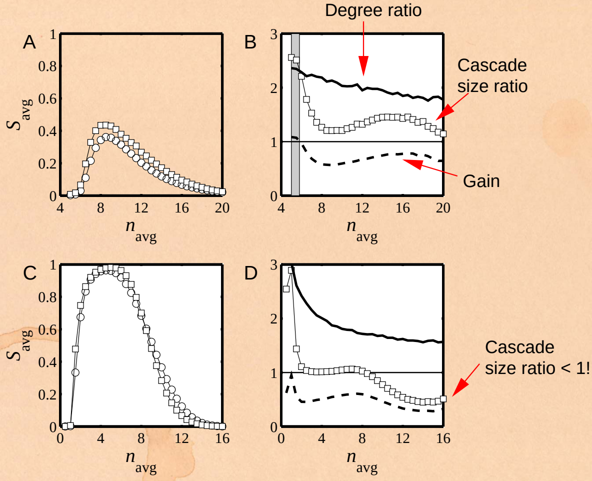
- Connect nodes with probability $\propto \exp^{-\alpha d}$ where
  α = homophily parameter
  and
  d = distance between nodes (height of lowest common ancestor)
- τ_1 = intergroup probability of friend-of-friend connection
- τ_2 = intragroup probability of friend-of-friend connection

Cascade windows for group-based networks



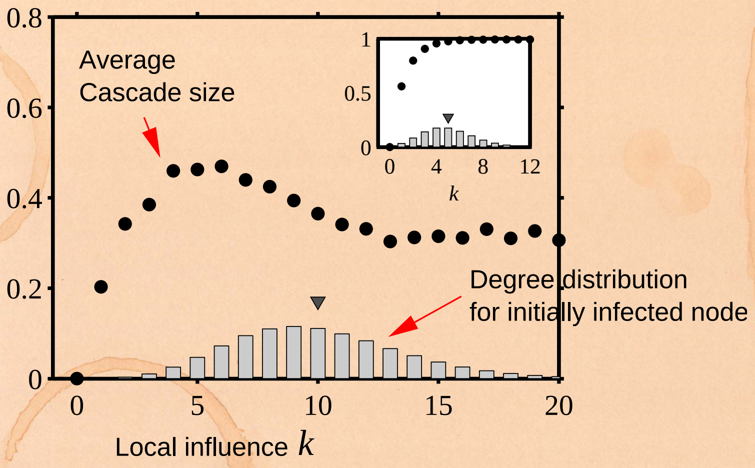


Multiplier effect for group-based networks:



- 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.

Social contagion

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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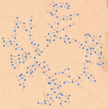
Social contagion

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.



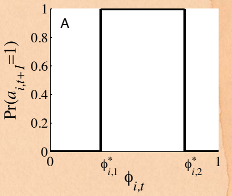
Chaotic contagion:

- What if individual response functions are not monotonic?
- Consider a simple deterministic version:

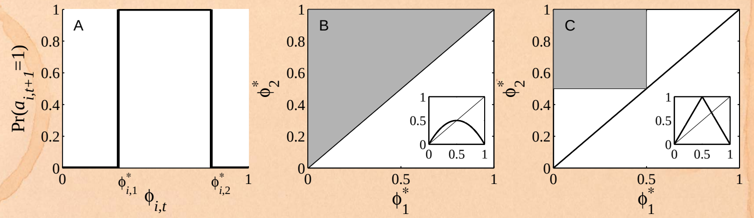
- Node i has an 'activation threshold' $\phi_{i,1}$

  ...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.



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.

Chaotic contagion

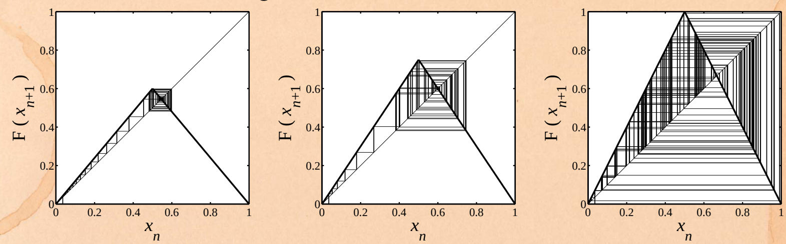
Definition of the tent map:

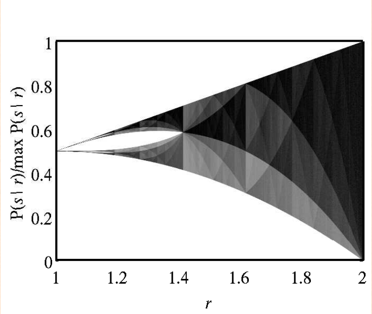
$$F(x) = \begin{cases} rx \text{ for } 0 \leq x \leq \frac{1}{2}, \\ r(1-x) \text{ for } \frac{1}{2} \leq x \leq 1. \end{cases}$$

- 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.



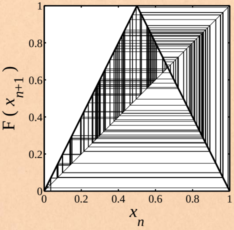


Orbit diagram:

Chaotic behavior increases as map slope *r* is increased.

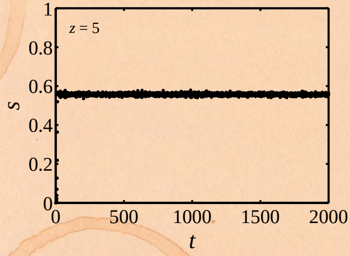
Chaotic behavior

Take $r = 2$ case:

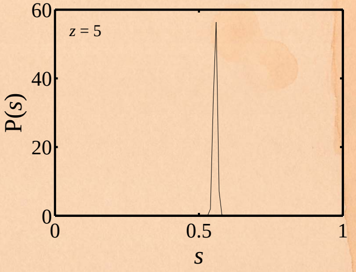


- What happens if nodes have limited information?
- As before, allow interactions to take place on a sparse random network.
- Vary average degree $z = \langle k \rangle$, a measure of information

Invariant densities—stochastic response functions

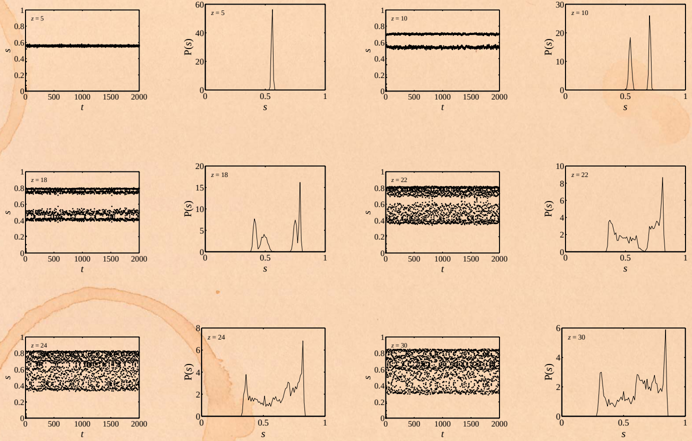


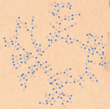
activation time series



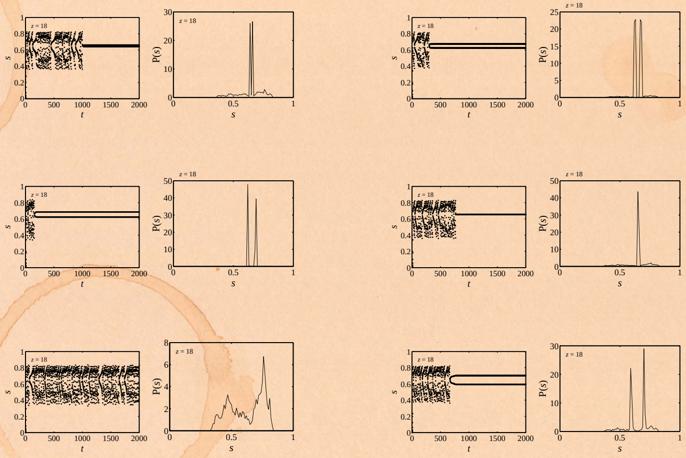
activation density

Invariant densities—stochastic response functions

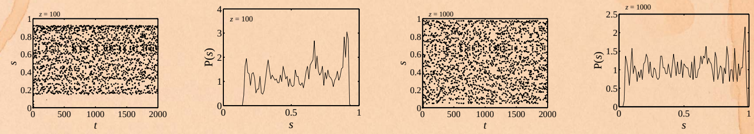




Invariant densities—deterministic response functions for one specific network with $\langle k \rangle = 18$

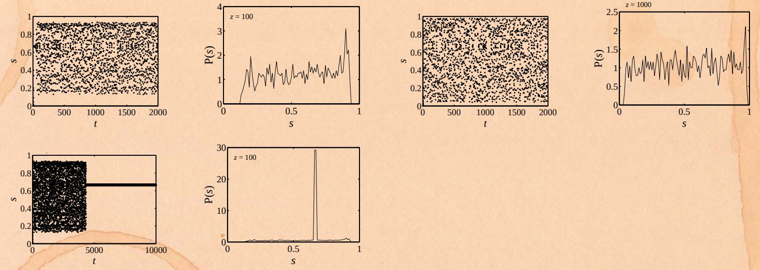


Invariant densities—stochastic response functions



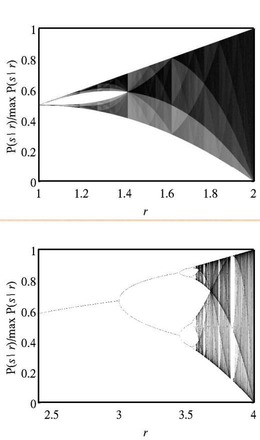
Trying out higher values of $\langle k \rangle$...

Invariant densities—deterministic response functions

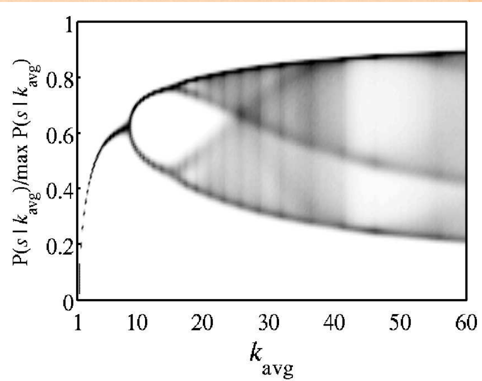


Trying out higher values of $\langle k \rangle$...

Connectivity leads to chaos:



Stochastic response functions:



Chaotic behavior in coupled systems

Coupled maps are well explored (Kaneko/Kuramoto):

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

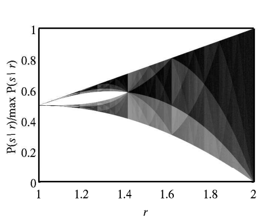
- $\mathcal{N}_i$ = neighborhood of node i

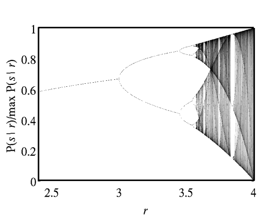
1. Node states are continuous
2. Increase δ and neighborhood size $|\mathcal{N}|$
   $\Rightarrow$ synchronization

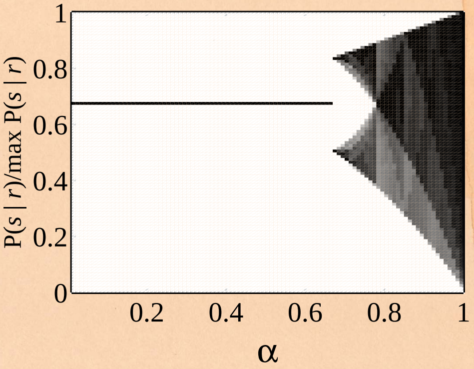
But for contagion model:

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

Bifurcation diagram: Asynchronous updating







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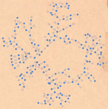

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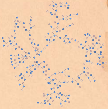

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