Social Contagion

Principles of Complex Systems
CSYS/MATH 300, Spring, 2013 | #SpringPoCS2013

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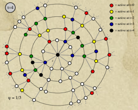

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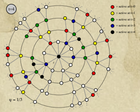
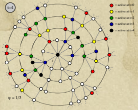


Outline

Social Contagion Models

- Background
- Granovetter's model
- Network version
- Final size
- Spreading success
- Groups

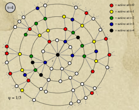
References



Things that spread well:

buzzfeed.com (⊞):

+ News ...



LOL + cute + fail + wtf:

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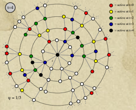
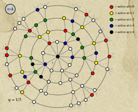
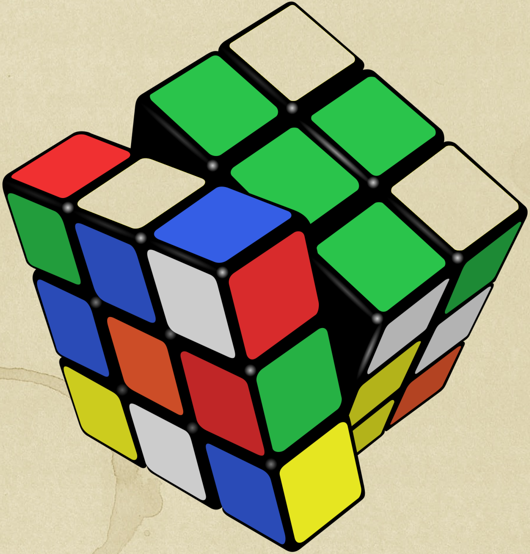


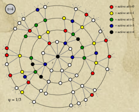
The whole lolcats thing:



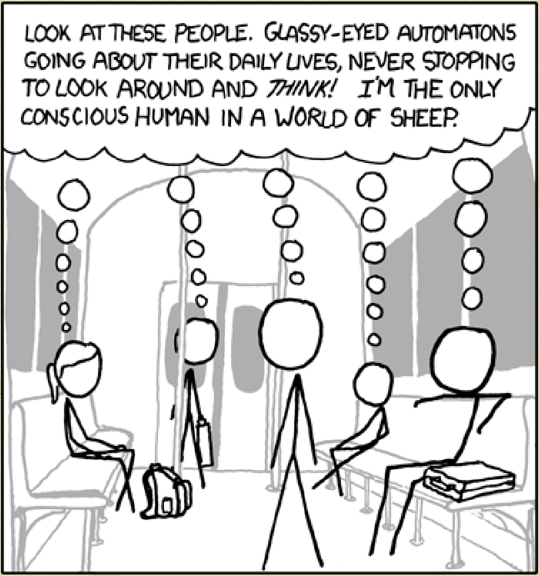
Some things really stick:

wtf + geeky + omg:

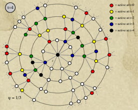




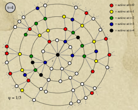
Social Contagion



http://xkcd.com/610/ (⊞)



Social Contagion



Social Contagion

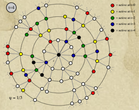
Social Contagion

Examples abound

- fashion
- striking
- smoking (⊞) [7]
- residential segregation [19]
- ipods
- obesity (⊞) [6]
- Harry Potter
- voting
- gossip
- Rubik's cube 
- religious beliefs
- leaving lectures

SIR and SIRS contagion possible

- Classes of behavior versus specific behavior: dieting



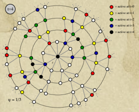
Framingham heart study:

Evolving network stories (Christakis and Fowler):

- The spread of quitting smoking (⊞) [7]
- The spread of spreading (⊞) [6]
- Also: happiness (⊞) [9], 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).



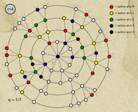
Social Contagion

Two focuses for us

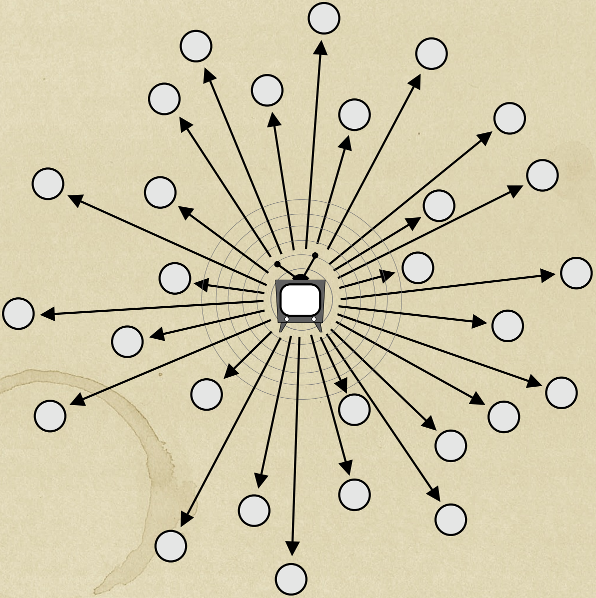
- Widespread media influence
- Word-of-mouth influence

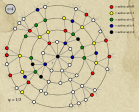
We need to understand influence

- Who influences whom? Very hard to measure...
- What kinds of influence response functions are there?
- Are some individuals super influencers?
  Highly popularized by Gladwell [10] as 'connectors'
- The infectious idea of opinion leaders (Katz and Lazarsfeld) [16]

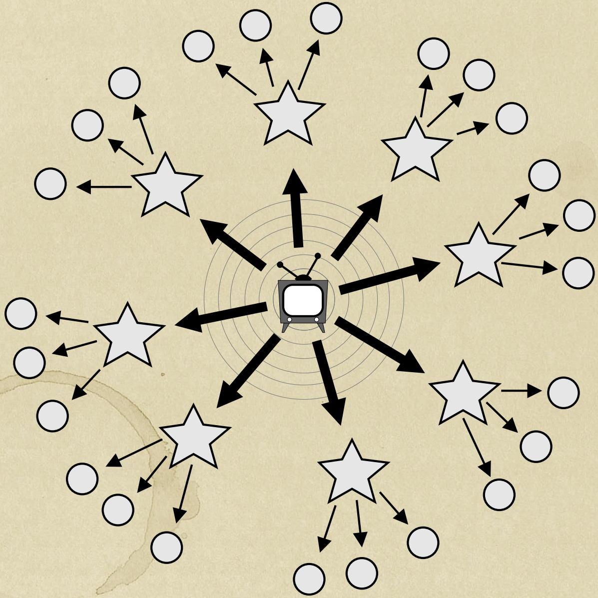


The hypodermic model of influence

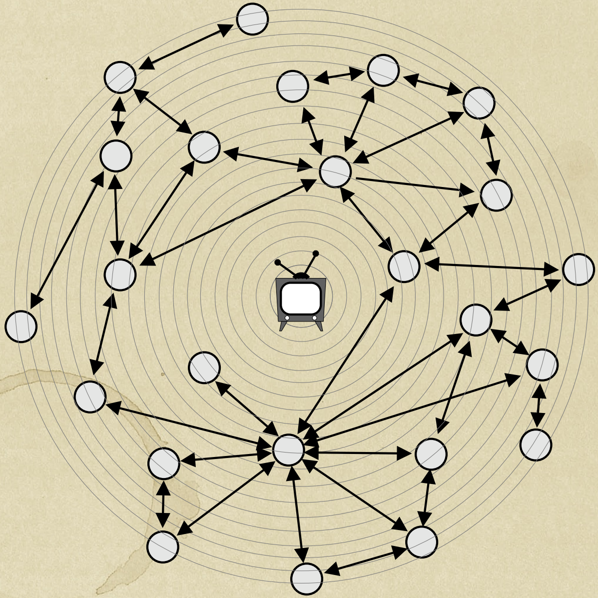




The two step model of influence [16]



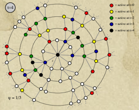
The general model of influence



Social Contagion

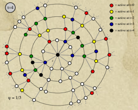
Why do things spread?

- 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...



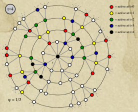
The Mona Lisa

- "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, ...

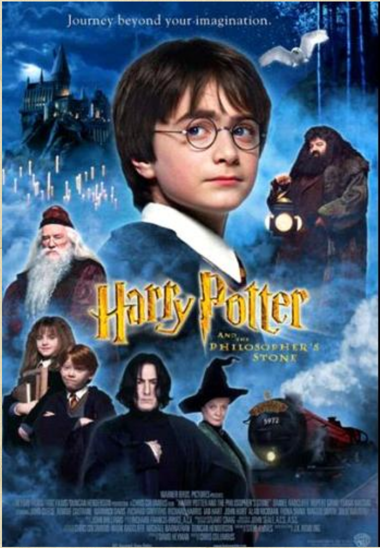


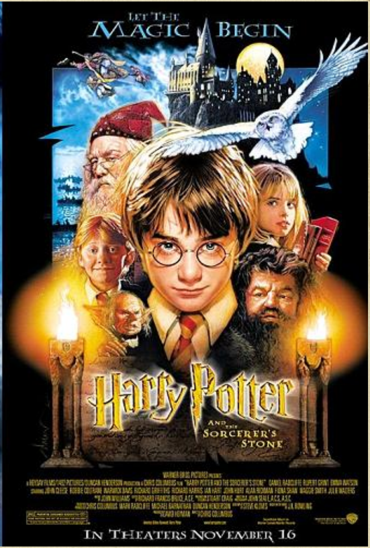
The completely unpredicted fall of Eastern Europe

Timur Kuran:[17, 18] “Now Out of Never: The Element of Surprise in the East European Revolution of 1989”



The dismal predictive powers of editors...

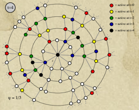




Social Contagion

Messing with social connections

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

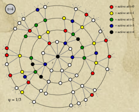


Getting others to do things for you

A very good book: 'Influence' [8] 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., Jonestown (⊞), Kitty Genovese (⊞) (contested).
4. Liking: *The Friendly Thief*; e.g., Separation into groups is enough to cause problems.
5. Authority: *Directed Deference*; e.g., Milgram's obedience to authority experiment. (⊞)
6. Scarcity: *The Rule of the Few*; e.g., Prohibition.

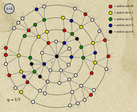


Social contagion

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

Other acts of influence:

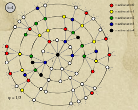
- Conspicuous Consumption (Veblen, 1912)
- Conspicuous Destruction (Potlatch)



Social Contagion

Some important models:

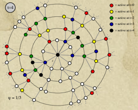
- Tipping models—Schelling (1971) [19, 20, 21]
  - Simulation on checker boards
  - Idea of thresholds
  - Explore the Netlogo (⊞) online implementation (⊞) [26]
- Threshold models—Granovetter (1978) [13]
- Herding models—Bikhchandani, Hirschleifer, Welch (1992) [2, 3]
  - Social learning theory, Informational cascades,...



Social contagion models

Thresholds

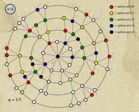
- Basic idea: individuals adopt a behavior when a certain fraction of others have adopted
- 'Others' may be everyone in a population, an individual's close friends, any reference group.
- Response can be probabilistic or deterministic.
- Individual thresholds can vary
- Assumption: order of others' adoption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



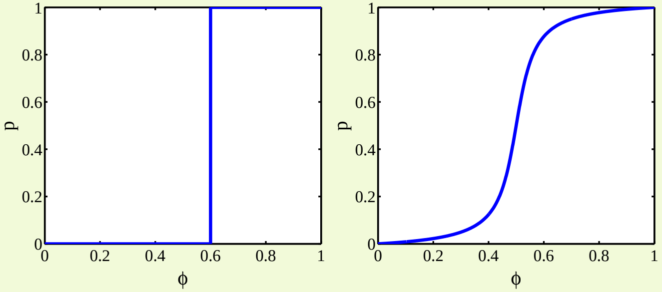
Social Contagion

Some possible origins of thresholds:

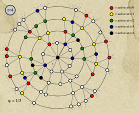
- Inherent, evolution-devised inclination to coordinate, to conform, to imitate. [1]
- Lack of information: impute the worth of a good or behavior based on degree of adoption (social proof)
- Economics: Network effects or network externalities
  - Externalities = Effects on others not directly involved 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



Threshold models—response functions



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

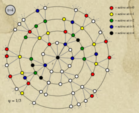


Social Contagion

Granovetter's Threshold model—definitions

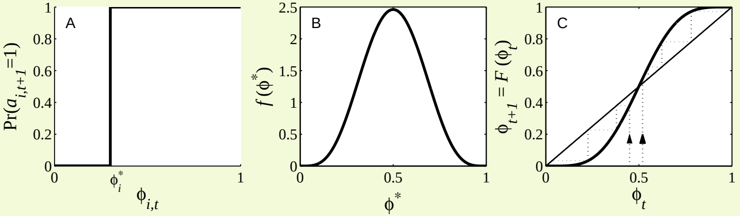
- ϕ^* = threshold of an individual.
- $f(\phi_*)$ = distribution of thresholds in a population.
- $F(\phi_*)$ = cumulative distribution = $\int_{\phi'_*=0}^{\phi_*} f(\phi'_*)\mathrm{d}\phi'_*$
- ϕ_t = fraction of people 'rioting' at time step t.

- 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 $[0, 1]$.

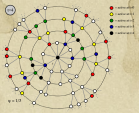


Threshold models

Action based on perceived behavior of others:

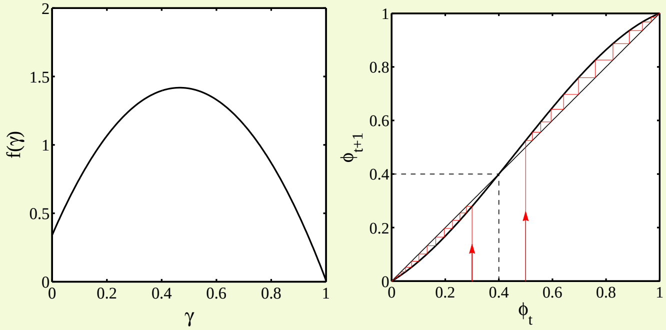


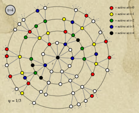
- Two states: S and I.
- ϕ = fraction of contacts 'on' (e.g., rioting)
- Discrete time update (strong assumption!)
- This is a Critical mass model



Threshold models

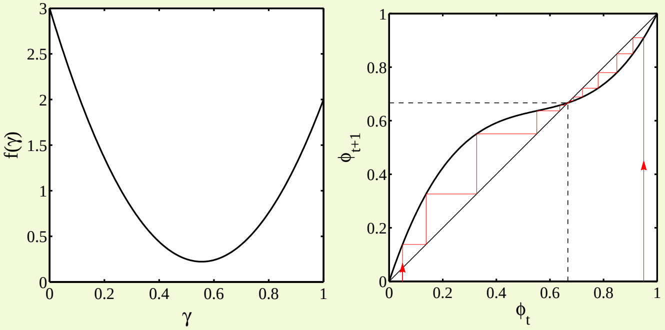
Another example of critical mass model:

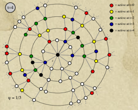




Threshold models

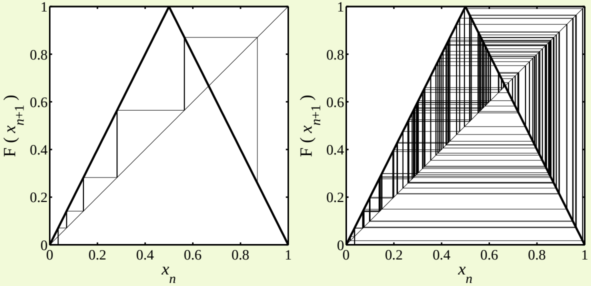
Example of single stable state model:



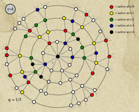


Threshold models

Chaotic behavior possible [15, 14]



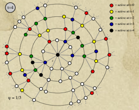
- Period doubling arises as map amplitude r is increased.
- Synchronous update assumption is crucial



Threshold models—Nutshell

Implications for collective action theory:

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

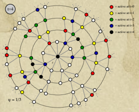


"A simple model of global cascades on random networks"

- Many years after Granovetter and Soong's work: D. J. Watts. Proc. Natl. Acad. Sci., 2002 [23]
  - Mean field model → network model
  - Individuals now have a limited view of the world

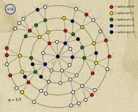
We'll also explore:

- "Seed size strongly affects cascades on random networks" [12]
  Gleeson and Cahalane, Phys. Rev. E, 2007.
- "Influentials, Networks, and Public Opinion Formation" [24]
  Watts and Dodds, J. Cons. Res., 2007.
- "Threshold models of Social Influence" [25]
  Watts and Dodds, The Oxford Handbook of Analytical Sociology, 2009.

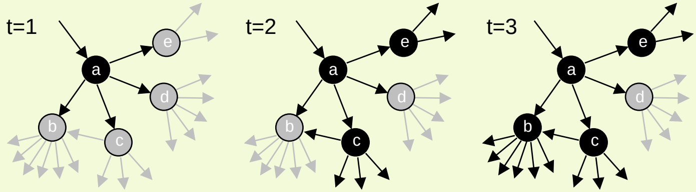


Threshold model on a network

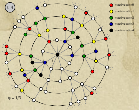
- Interactions between individuals now represented by a network
- Network is sparse
- Individual i has k_i contacts
- Influence on each link is reciprocal and of unit weight
- Each individual i has a fixed threshold ϕ_i
- Individuals repeatedly poll contacts on network
- Synchronous, discrete time updating
- 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 $\phi = 0.2$.



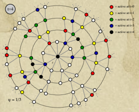
Snowballing

First study random networks:

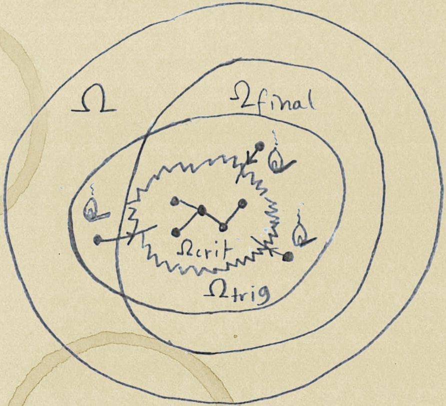
- Start with N nodes with a degree distribution p_k
- Nodes are randomly connected (carefully so)
- Aim: Figure out when activation will propagate
- Determine a cascade condition

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?

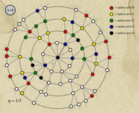


Example random network structure:



- $\Omega_{\rm crit} = \Omega_{\rm vuln}$ = critical mass = global vulnerable component
- $\Omega_{\rm trig}$ = triggering component
- $\Omega_{\rm final}$ = potential extent of spread
- Ω = entire network

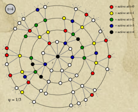
$\Omega_{\rm crit} \subset \Omega_{\rm trig}$; $\Omega_{\rm crit} \subset \Omega_{\rm final}$; and $\Omega_{\rm trig}, \Omega_{\rm final} \subset \Omega$.



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.



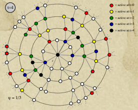
The most gullible

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* [23]
- Cluster of vulnerables = critical mass
- Network story: 1 node $\rightarrow$ critical mass $\rightarrow$ everyone.



Cascade condition

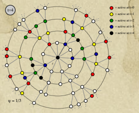
Back to following a link:

- 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

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



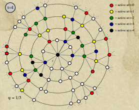
Cascade condition

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.



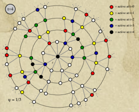
Cascade condition

Putting things together:

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

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

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

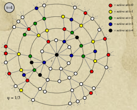


Cascade condition

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

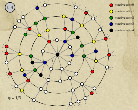
Two special cases:

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

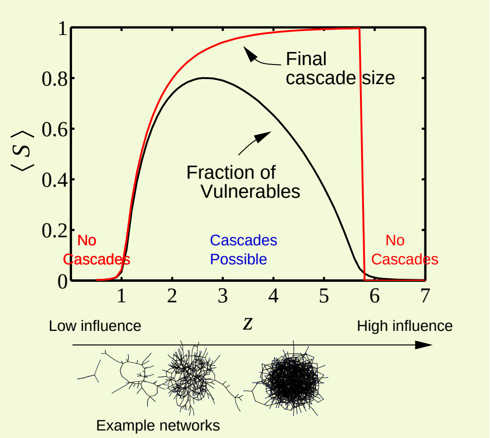
$$\beta \cdot \sum_{k=1}^{\infty} (k-1) \cdot \frac{kP_k}{\langle k \rangle} \geq 1.$$

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

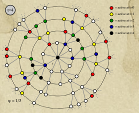
$$1 \cdot \sum_{k=1}^{\infty} (k-1) \cdot \frac{kP_k}{\langle k \rangle} \geq 1.$$



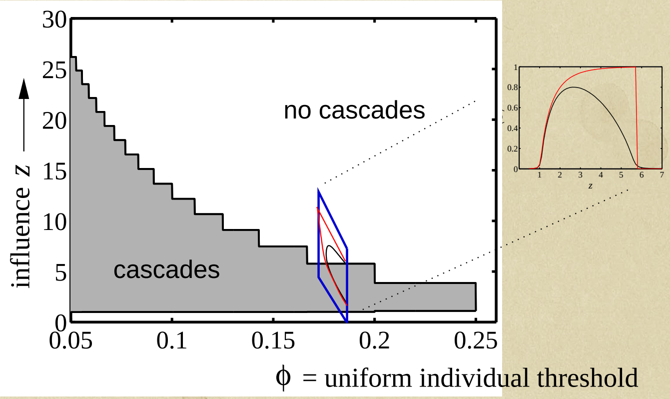
Cascades on random networks



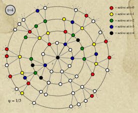
- Cascades occur only if size of max vulnerable cluster > 0.
- System may be 'robust-yet-fragile'.
- 'Ignorance' facilitates spreading.



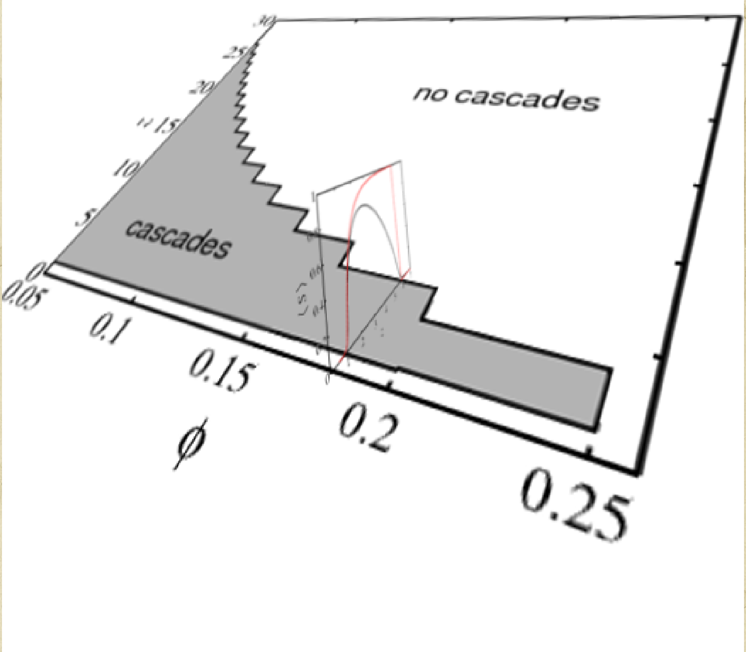
Cascade window for random networks

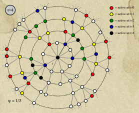


- 'Cascade window' widens as threshold ϕ decreases.
- Lower thresholds enable spreading.

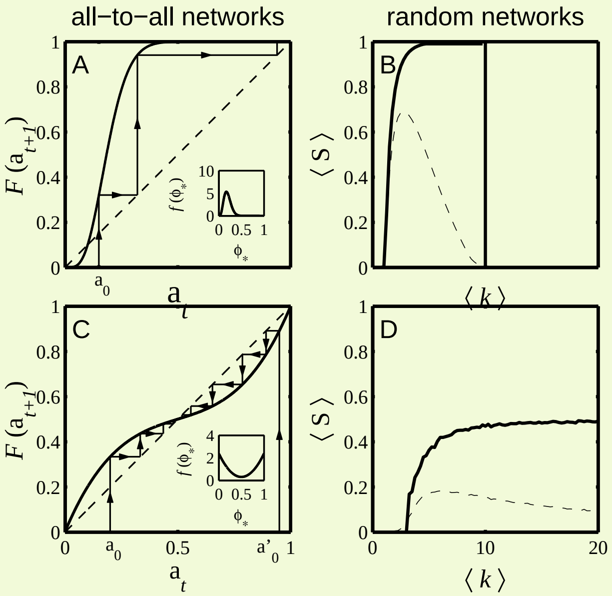


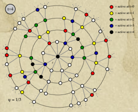
Cascade window for random networks





All-to-all versus random networks

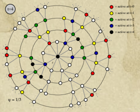




Cascade window—summary

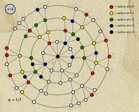
For our simple model of a uniform threshold:

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."



Threshold contagion on random networks

- Next: Find expected fractional size of spread.
- Not obvious even for uniform threshold problem.
- Difficulty is in figuring out if and when nodes that need ≥ 2 hits switch on.
- Problem solved for infinite seed case by Gleeson and Cahalane:
  "Seed size strongly affects cascades on random networks," Phys. Rev. E, 2007. [12]
- Developed further by Gleeson in "Cascades on correlated and modular random networks," Phys. Rev. E, 2008. [11]

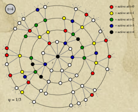


Expected size of spread

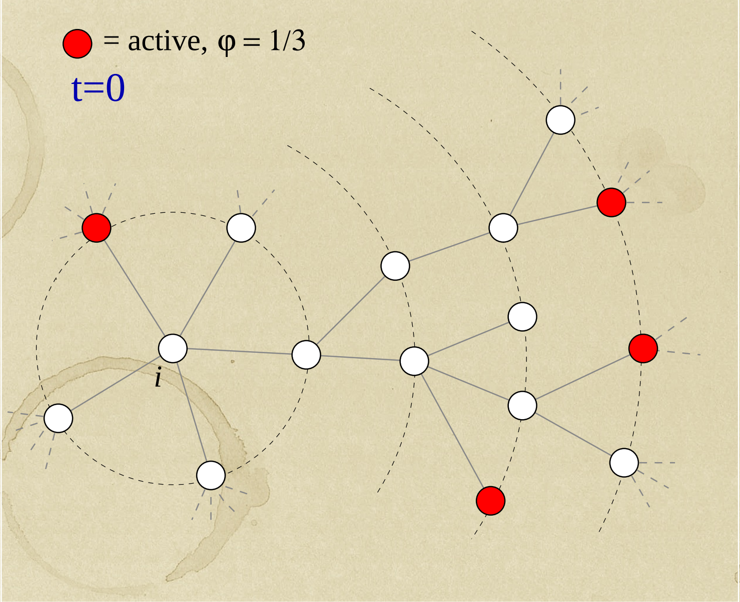
Idea:

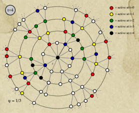
- Randomly turn on a fraction ϕ_0 of nodes at time $t = 0$
- Capitalize on local branching network structure of random networks (again)
- Now think about what must happen for a specific node *i* to become active at time *t*:

- $t = 0$: *i* is one of the seeds (prob = ϕ_0)
- $t = 1$: *i* was not a seed but enough of *i*'s friends switched on at time $t = 0$ so that *i*'s threshold is now exceeded.
- $t = 2$: enough of *i*'s friends and friends-of-friends switched on at time $t = 0$ so that *i*'s threshold is now exceeded.
- $t = n$: enough nodes within *n* hops of *i* switched on at $t = 0$ and their effects have propagated to reach *i*.

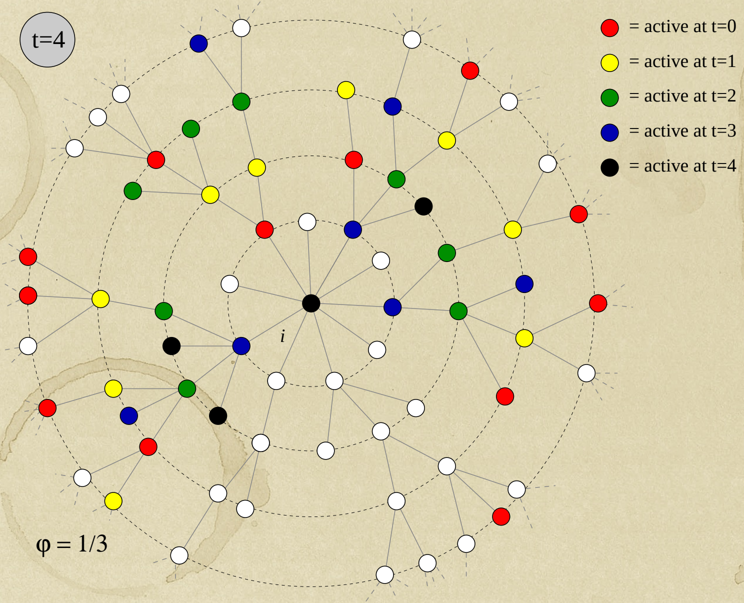


Expected size of spread





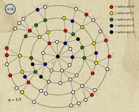
Expected size of spread



Expected size of spread

Notes:

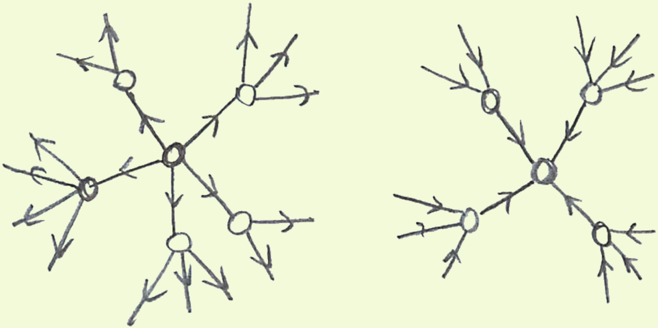
- Calculations are possible if nodes do not become inactive (strong restriction).
- Not just for threshold model—works for a wide range of contagion processes.
- We can analytically determine the entire time evolution, not just the final size.
- We can in fact determine **Pr**(node of degree k switching on at time t).
- Asynchronous updating can be handled too.



Expected size of spread

Pleasantness:

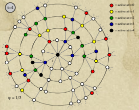
- Taking off from a single seed story is about expansion away from a node.
- Extent of spreading story is about contraction at a node.



Expected size of spread

- Notation:
  $\phi_{k,t} = \mathbf{Pr}$(a degree k node is active at time t).
- Notation: $B_{kj} = \mathbf{Pr}$ (a degree k node becomes active if j neighbors are active).
- Our starting point: $\phi_{k,0} = \phi_0$.
- $\binom{k}{j}\phi_0^j(1-\phi_0)^{k-j}$ = $\mathbf{Pr}$ (j of a degree k node's neighbors were seeded at time $t = 0$).
- Probability a degree k node was a seed at $t = 0$ is ϕ_0 (as above).
- Probability a degree k node was not a seed at $t = 0$ is $(1 - \phi_0)$.
- Combining everything, we have:

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



Expected size of spread

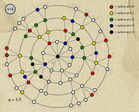
- For general t, we need to know the probability an edge coming into a degree k node at time t is active.
- Notation: call this probability θ_t.
- We already know $\theta_0 = \phi_0$.
- Story analogous to $t = 1$ case. For node i:

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

- Average over all nodes to obtain expression for ϕ_{t+1}:

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

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



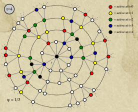
Expected size of spread

First connect θ_0 to θ_1:

- $\theta_1 = \phi_0 +$

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

- $\frac{kP_k}{\langle k\rangle} = R_k$ = **Pr** (edge connects to a degree k node).
- $\sum_{j=0}^{k-1}$ piece gives **Pr**(degree node k activates) of its neighbors $k-1$ incoming neighbors are active.
- ϕ_0 and $(1-\phi_0)$ terms account for state of node at time $t=0$.
- See this all generalizes to give θ_{t+1} in terms of θ_t...



Expected size of spread

Two pieces: edges first, and then nodes

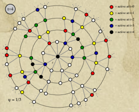
1. $$\theta_{t+1} = \underbrace{\phi_0}_{\text{exogenous}}$$

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

with $\theta_0 = \phi_0$.

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

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



Expected size of spread:

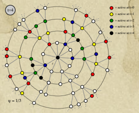
- Retrieve cascade condition for spreading from a single seed in limit $\phi_0 \rightarrow 0$.
- Depends on map $\theta_{t+1} = G(\theta_t; \phi_0)$.
- First: if self-starters are present, some activation is assured:

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

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

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

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



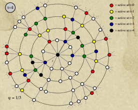
Expected size of spread:

In words:

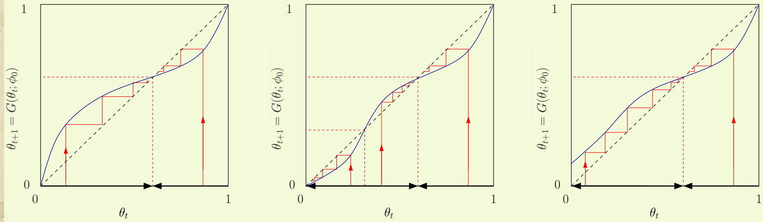
- If $G(0;\phi_0) > 0$, spreading must occur because some nodes turn on for free.
- If G has an unstable fixed point at $\theta = 0$, then cascades are also always possible.

Non-vanishing seed case:

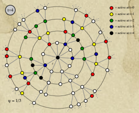
- Cascade condition is more complicated for $\phi_0 > 0$.
- If G has a stable fixed point at $\theta = 0$, and an unstable fixed point for some $0 < \theta_* < 1$, then for $\theta_0 > \theta_*$, spreading takes off.
- Tricky point: G depends on ϕ_0, so as we change ϕ_0, we also change G.



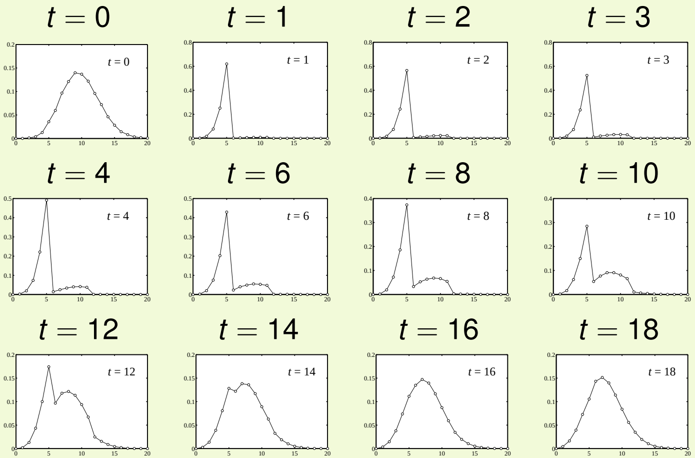
General fixed point story:



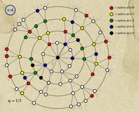
- Given $\theta_0 (= \phi_0)$, θ_∞ will be the nearest stable fixed point, either above or below.
- n.b., adjacent fixed points must have opposite stability types.
- Important: Actual form of G depends on ϕ_0.
- So choice of ϕ_0 dictates both G and starting point—can't start anywhere for a given G.



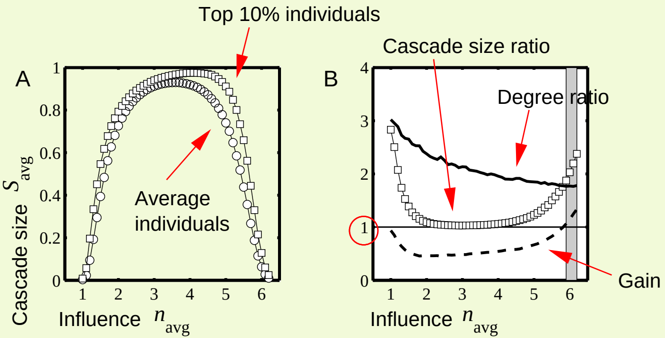
Early adopters—degree distributions



$P_{k,t}$ versus k

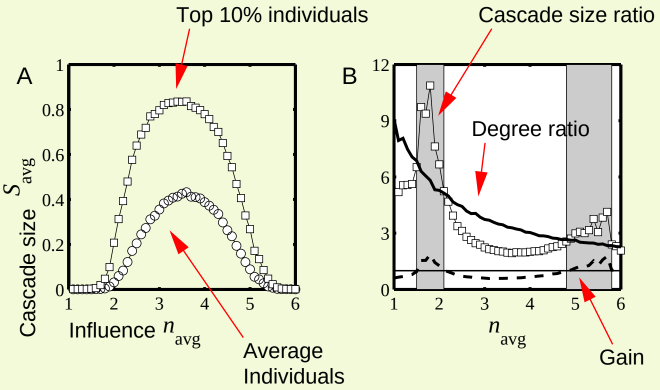


The multiplier effect:



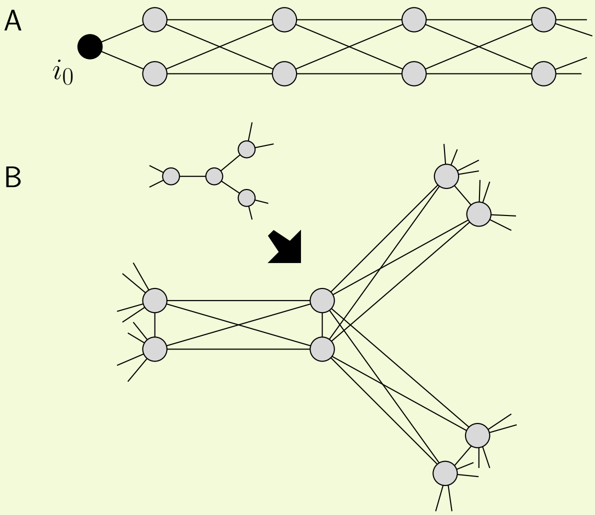
- Fairly uniform levels of individual influence.
- Multiplier effect is mostly below 1.

The multiplier effect:

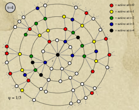


- Skewed influence distribution example.

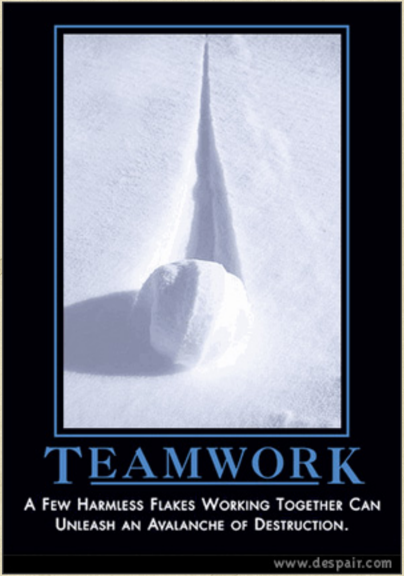
Special subnetworks can act as triggers



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

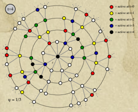


The power of groups...



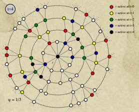
despair.com

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

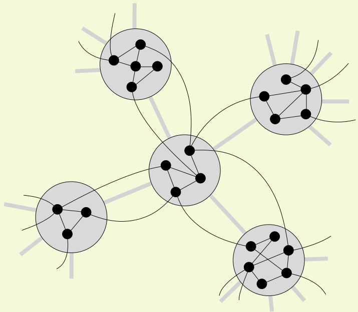


Extensions

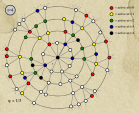
- Assumption of sparse interactions is good
- Degree distribution is (generally) key to a network's function
- Still, random networks don't represent all networks
- Major element missing: group structure



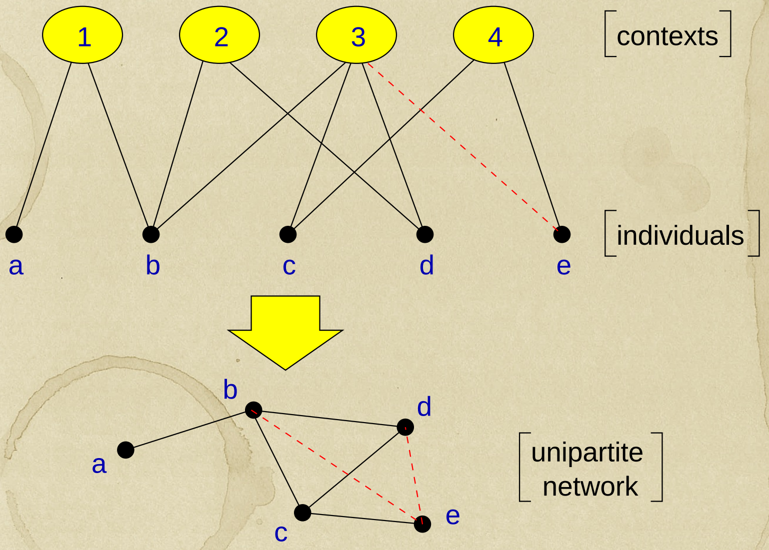
Group structure—Ramified random networks



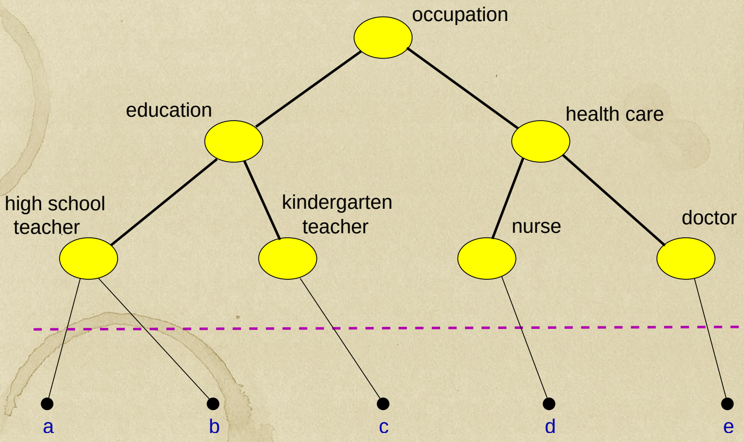
p = intergroup connection probability
q = intragroup connection probability.

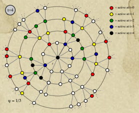


Bipartite networks

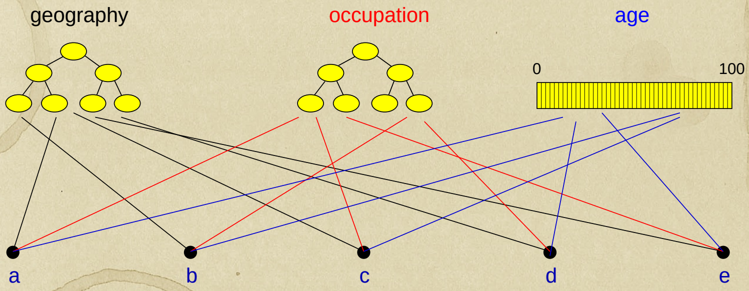


Context distance





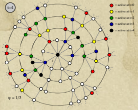
Generalized affiliation model



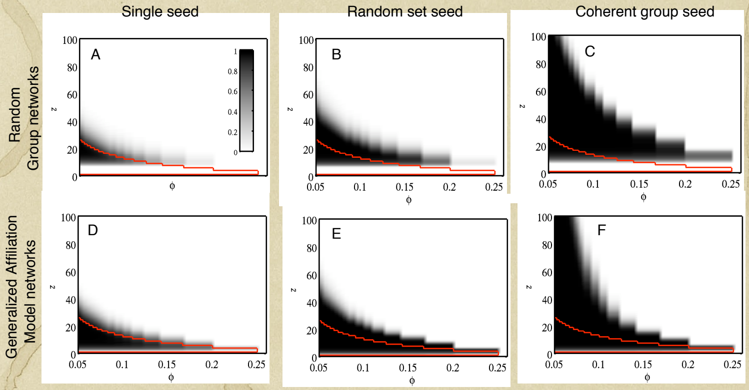
(Blau & Schwartz, Simmel, Breiger)

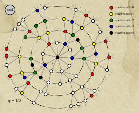
Generalized affiliation model networks with triadic closure

- 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

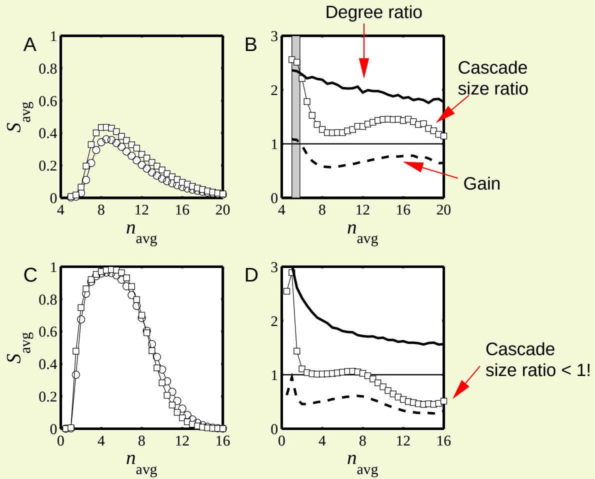


Cascade windows for group-based networks

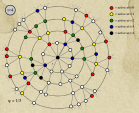




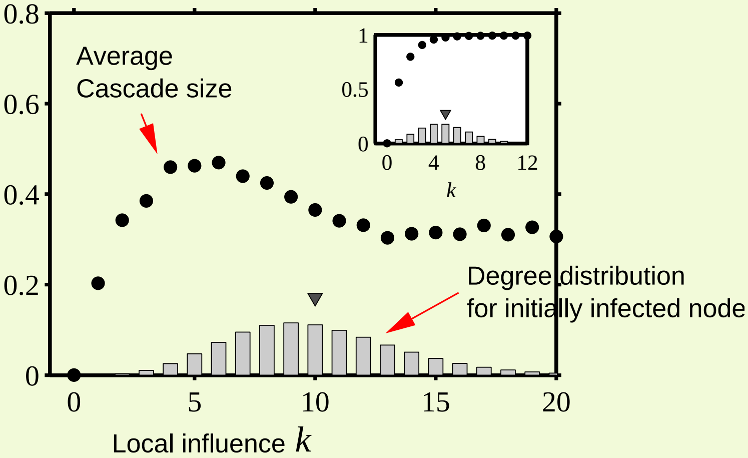
Multiplier effect for group-based networks:



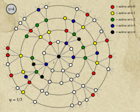
- Multiplier almost always below 1.



Assortativity in group-based networks



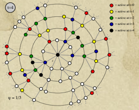
- The most connected nodes aren't always the most 'influential.'
- Degree assortativity is the reason.



Social contagion

Summary

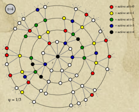
- 'Influential vulnerables' are key to spread.
- Early adopters are mostly vulnerables.
- Vulnerable nodes important but not necessary.
- Groups may greatly facilitate spread.
- Seems that cascade condition is a global one.
- Most extreme/unexpected cascades occur in highly connected networks
- 'Influentials'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



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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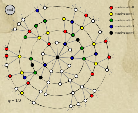

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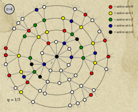

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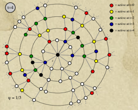

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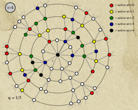

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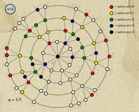

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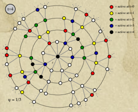

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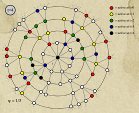

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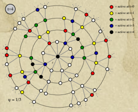

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