Social Contagion

Principles of Complex Systems | @pocsvox
CSYS/MATH 300, Fall, 2015 | #FallPoCS2015

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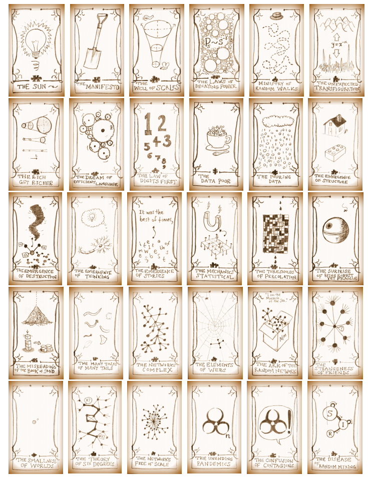
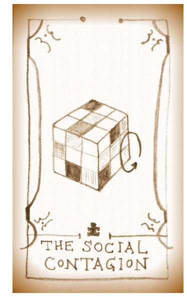
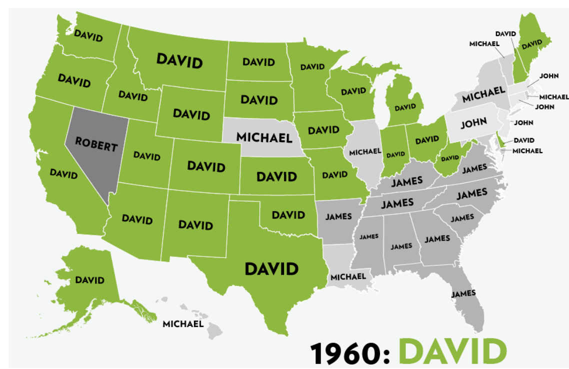
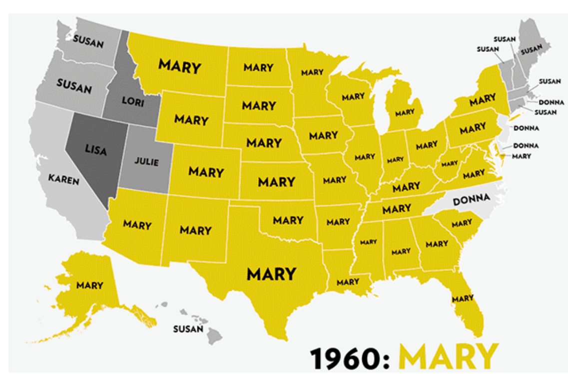
Outline







From the Atlantic



From the Atlantic

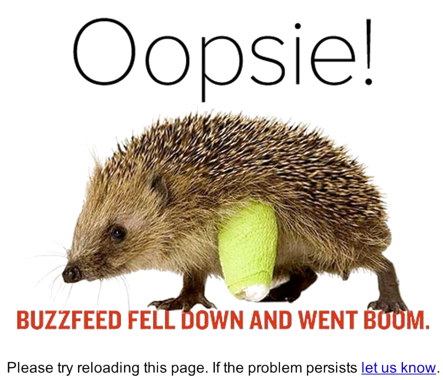
Things that spread well:

buzzfeed.com:

- Dangerously self aware: 11 Elements that make a perfect viral video.

+ News ...

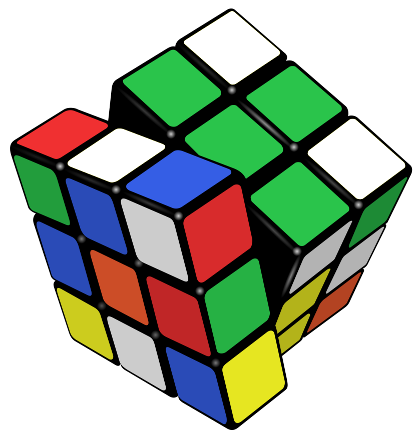
LOL + cute + fail + wtf:



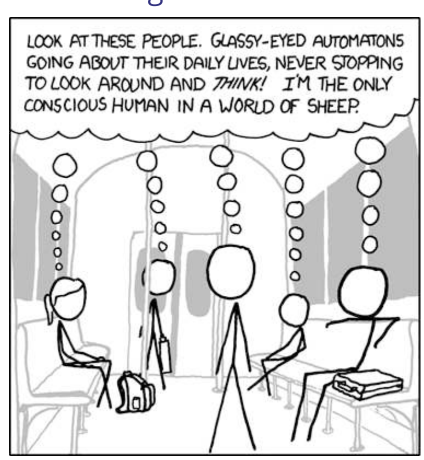
The whole lolcats thing:

Some things really stick:

wtf + geeky + omg:



Why social contagion works so well:



http://xkcd.com/610/

Social Contagion

Social Contagion

Examples abound

- fashion
- striking
- smoking [7]
- residential segregation [22]
- iPhones and iThings
- obesity [6]
- Harry Potter
- voting
- gossip
- Rubik's cube
- religious beliefs
- school shootings
- leaving lectures

SIR and SIRS type contagion possible

- Classes of behavior versus specific behavior : dieting, horror movies, getting married, invading countries, ...

Mixed messages: Please copy, but also, don't copy ...

- Cindy Harrell appeared in the (terrifying) music video for Ray Parker Jr.'s Ghostbusters.
- Misframing: Appeals only to seed on exponential growth.

Framingham heart study:

Evolving network stories (Christakis and Fowler):

- The spread of quitting smoking [7]
- The spread of spreading [6]
- Also: happiness [11], 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

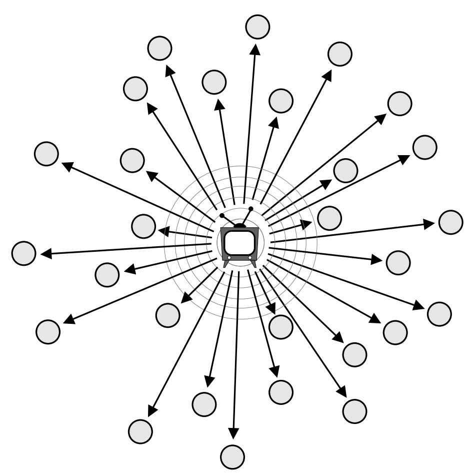
Two focuses for us

- Widespread media influence
- Word-of-mouth influence

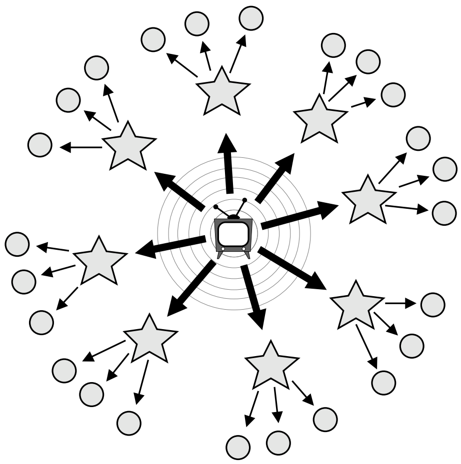
We need to understand influence

- Who influences whom? Very hard to measure...
- What kinds of influence response functions are there?
- Are some individuals super influencers? Highly popularized by Gladwell [12] as 'connectors'
- The infectious idea of opinion leaders (Katz and Lazarsfeld) [19]

The hypodermic model of influence



The two step model of influence [19]



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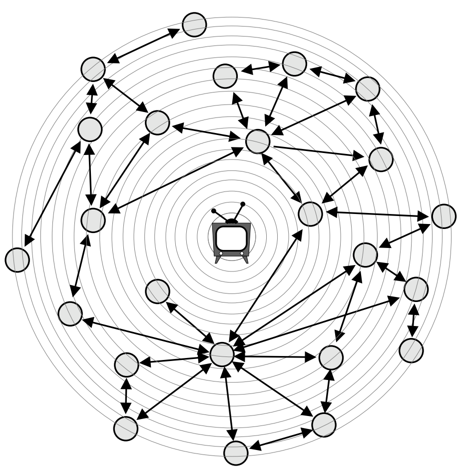
The general model of influence: the Social Wild



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Why do things spread socially?

- Because of properties of special individuals?
- Or system level properties?
- Is the match that lights the fire important?
- Yes. But only because we are storytellers: *homo narrativus*.
- We like to think things happened for reasons ...
- Reasons for success are usually ascribed to intrinsic properties (examples next).
- Teleological stories of fame are often easy to generate and believe.
- System/group dynamics harder to understand because most of our stories are built around individuals.
- Always good to examine what is said before and after the fact ...

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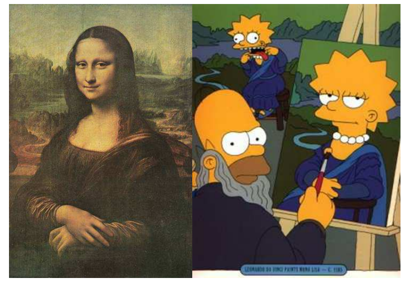
The Mona Lisa



- "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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'Tattooed Guy' Was Pivotal in Armstrong Case [nytimes]

- "... Leogrande's doping sparked a series of events ..."

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The completely unpredicted fall of Eastern Europe

Timur Kuran: [20, 21] "Now Out of Never: The Element of Surprise in the East European Revolution of 1989"

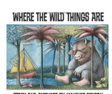
The dismal predictive powers of editors...

From a 2013 Believer Magazine interview with Maurice Sendak:

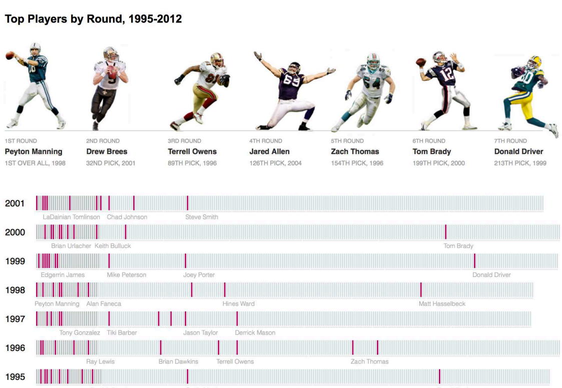
BLVR: Did the success of Where the Wild Things Are ever feel like an albatross?

MS: It's a nice book. It's perfectly nice. I can't complain about it. I remember Herman Melville said, "When I die no one is going to mention Moby-Dick. They're all going to talk about my first book, about f∗∗∗ing maidens in Tahiti." He was right. No mention of Moby-Dick then. Everyone wanted another Tahitian book, a beach book. But then he kept writing deeper and deeper and then came Moby-Dick and people hated it. The only ones who liked it were Mr. and Mrs. Nathaniel Hawthorne. Moby-Dick didn't get famous until 1930.

- Sendak named his dog Herman.
- The essential Colbert interview: Pt. 1 and Pt. 2.



Drafting success in the NFL:



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:

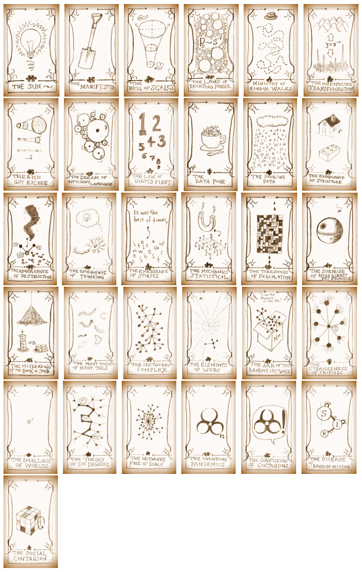
1. Reciprocation: *The Old Give and Take... and Take*; e.g., Free samples, Hare Krishnas.
2. Commitment and Consistency: *Hobgoblins of the Mind*; e.g., Hazing.
3. Social Proof: *Truths Are Us*; e.g., Jonestown, Kitty Genovese (contested).
4. Liking: *The Friendly Thief*; e.g., Separation into groups 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.

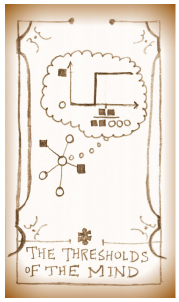
Social contagion

- 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) [22, 23, 24]
  - Simulation on checker boards
  - Idea of thresholds
  - Polygon-themed online visualization. (Includes optional diversity-seeking proclivity.)
  - Explore the Netlogo online implementation [29]
- Threshold models—Granovetter (1978) [15]
- Herding models—Bikhchandani, Hirschleifer, Welch (1992) [2, 3]
  - Social learning theory, Informational cascades,...

Social contagion models

Thresholds

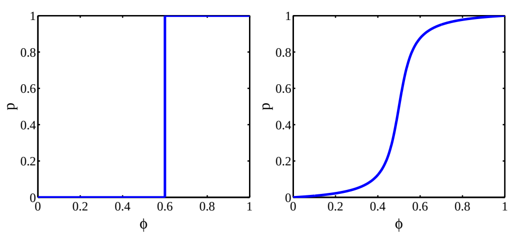
- Basic idea: individuals adopt a behavior when a certain fraction of others have adopted
- 'Others' may be everyone in a population, an individual's close friends, any reference group.
- Response can be probabilistic or deterministic.
- Individual thresholds can vary
- Assumption: order of others' adoption 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

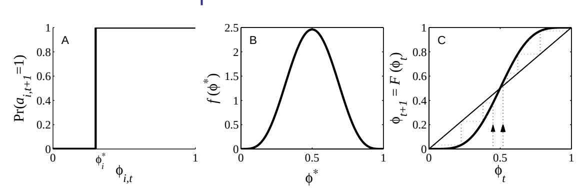
Threshold models—response functions



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

Threshold models

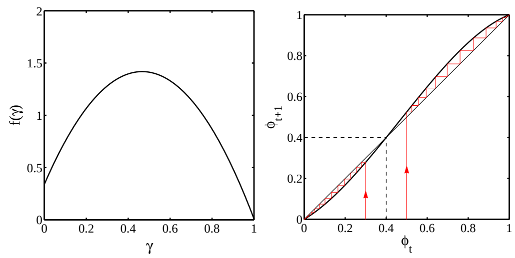
Action based on perceived behavior of others:



- 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:



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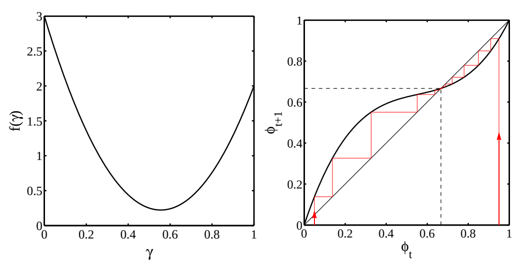
Threshold models

Example of single stable state model:



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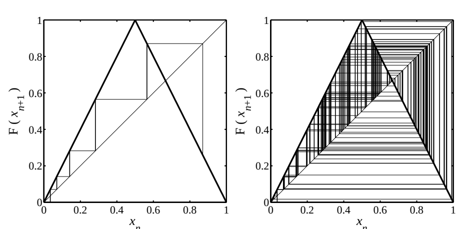
Threshold models

Chaotic behavior possible [17, 16, 9, 18]



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

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Threshold models—Nutshell

Implications for collective action theory:

1. Collective uniformity $\not\Rightarrow$ individual uniformity
2. Small individual changes $\Rightarrow$ large global changes
3. The stories/dynamics of complex systems are conceptually inaccessible for individual-centric narratives.
4. System stories live in left null space of our stories—we can't even see them.
5. But we happily impose simplistic, individual-centric stories—we can't help ourselves.

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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 [26]
  - Mean field model $\rightarrow$ network model
  - Individuals now have a limited view of the world

We'll also explore:

- "Seed size strongly affects cascades on random networks" [14]
Gleeson and Cahalane, Phys. Rev. E, 2007.
- "Direct, phyiscally motivated derivation of the contagion condition for spreading processes on generalized random networks" [10] Dodds, Harris, and Payne, Phys. Rev. E, 2011
- "Influentials, Networks, and Public Opinion Formation" [27]
Watts and Dodds, J. Cons. Res., 2007.
- "Threshold models of Social Influence" [28]
Watts and Dodds, The Oxford Handbook of Analytical Sociology, 2009.

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Threshold model on a network

- Interactions between individuals now represented by a network.
- Network is sparse.
- Individual i has k_i contacts.
- Influence on each link is reciprocal and of unit weight.
- Each individual i has a fixed threshold ϕ_i.
- Individuals repeatedly poll contacts on network.
- 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).

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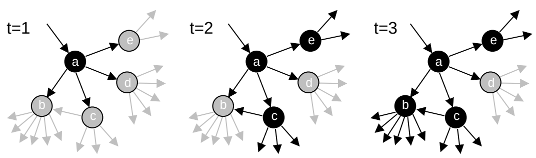
Threshold model on a network



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

Snowballing

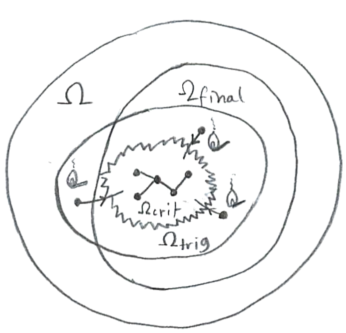
First study random networks:

- 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:



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

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

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* [26]
- 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:

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

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

Cascade condition

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

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

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

Cascade condition

Two special cases:

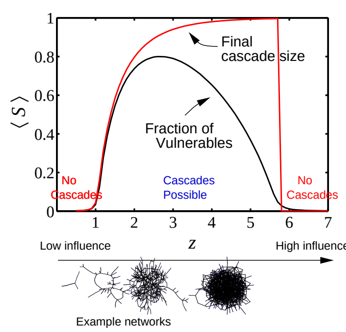
- (1) Simple disease-like spreading succeeds: $\beta_k = \beta$

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

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

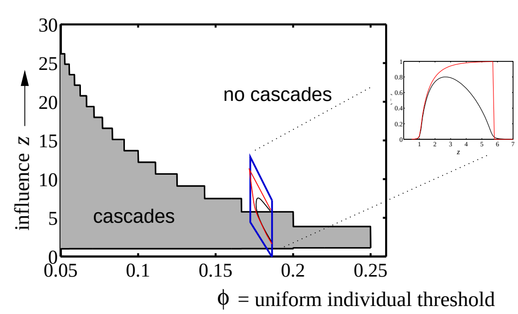
$$1 \cdot \sum_{k=1}^{\infty} (k-1) \cdot \frac{kP_k}{\langle k \rangle} > 1.$$

Cascades on random networks



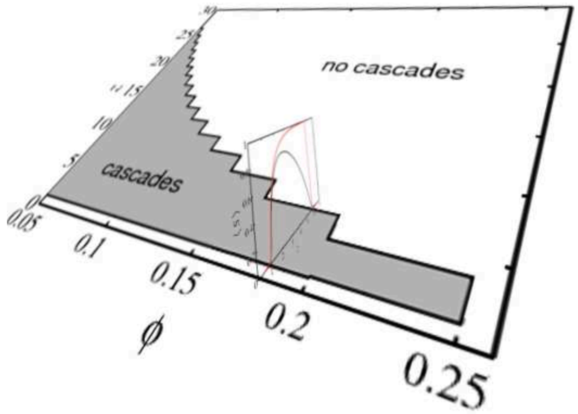
- Cascades occur only if size of max vulnerable cluster > 0.
- System may be 'robust-yet-fragile'.
- 'Ignorance' facilitates spreading.

Cascade window for random networks

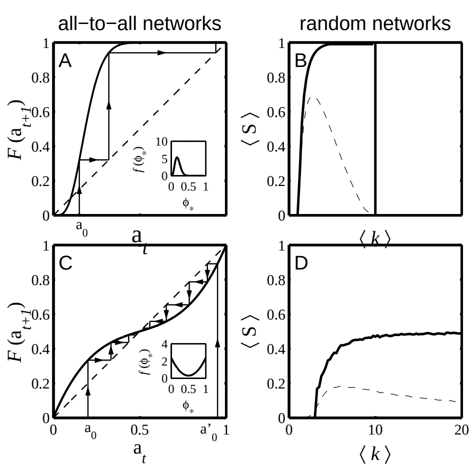


- 'Cascade window' widens as threshold ϕ decreases.
- Lower thresholds enable spreading.

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."

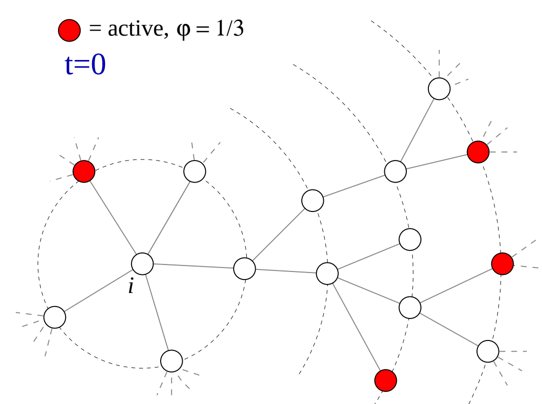
Threshold contagion on random networks

- 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 beautifully solved for infinite seed case by Gleeson and Cahalane: "Seed size strongly affects cascades on random networks," Phys. Rev. E, 2007. [14]
- Developed further by Gleeson in "Cascades on correlated and modular random networks," Phys. Rev. E, 2008. [13]

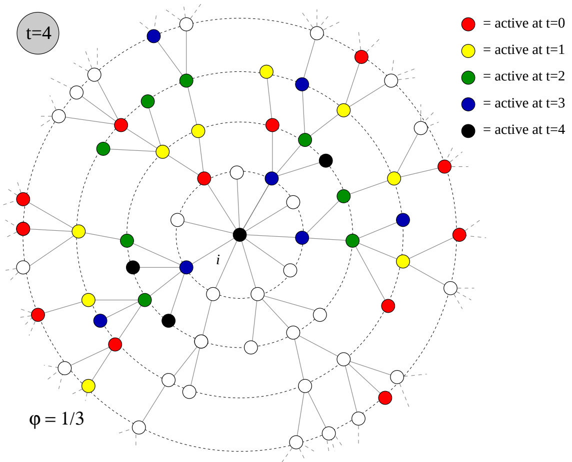
Determining expected size of spread:

- 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

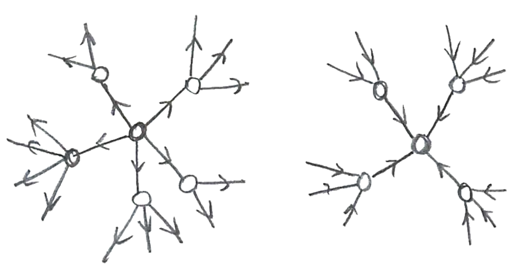
Notes:

- 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 $\mathbf{Pr}$(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:

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

- 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:

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

- 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:

- $\theta_1 = \phi_0 +$

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

- $\frac{kP_k}{\langle k\rangle} = R_k$ = $\mathbf{Pr}$ (edge connects to a degree k node).
- $\sum_{j=0}^{k-1}$ piece gives $\mathbf{Pr}$(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. $\theta_{t+1} = \underbrace{\phi_0}_{\text{exogenous}}$

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

with $\theta_0 = \phi_0$.

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

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

Expected size of spread

Iterative map for θ_t is key:

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

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

$$= G(\theta_t;\phi_0)$$

Expected size of spread:

- Retrieve cascade condition for spreading from a single seed in limit $\phi_0 \to 0$.
- 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:

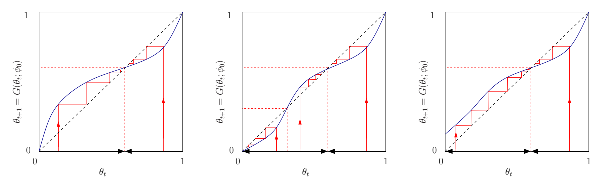
In words:

- 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:

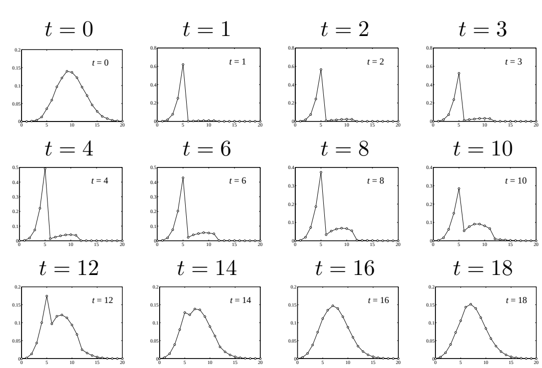
- Cascade condition is more complicated for $\phi_0 > 0$.
- If G has a stable fixed point at $\theta = 0$, and an unstable fixed point for some $0 < \theta_* < 1$, then for $\theta_0 > \theta_*$, spreading takes off.
- Tricky point: G depends on ϕ_0, so as we change ϕ_0, we also change G.
- A version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

General fixed point story:



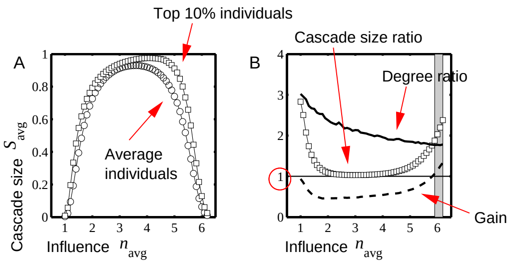
- Given $\theta_0 (= \phi_0)$, θ_∞ will be the nearest stable fixed point, either above or below.
- n.b., adjacent fixed points must have opposite stability types.
- Important: Actual form of G depends on ϕ_0.
- So choice of ϕ_0 dictates both G and starting point—can't start anywhere for a given G.

Early adopters—degree distributions



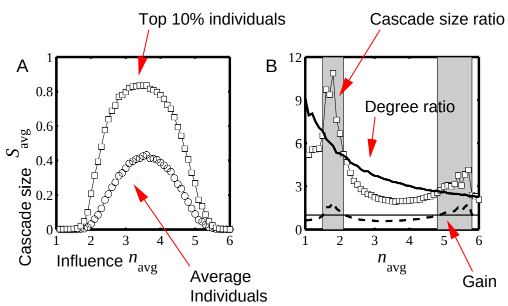
$P_{k,t}$ versus k

The multiplier effect:



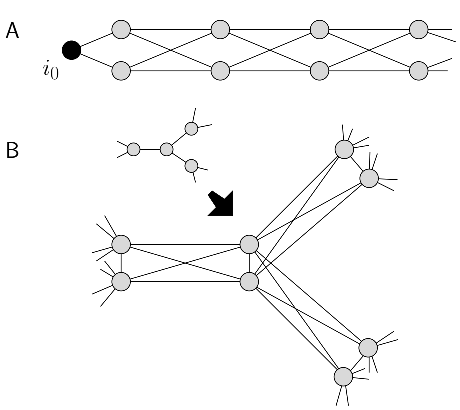
- Fairly uniform levels of individual influence.
- Multiplier effect is mostly below 1.

The multiplier effect:



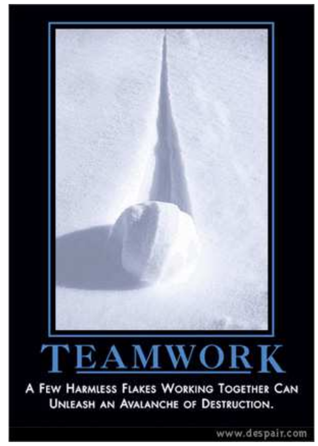
- Skewed influence distribution example.

Special subnetworks can act as triggers



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

The power of groups...



despair.com

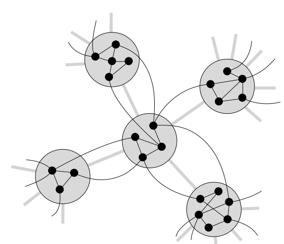
"A few harmless flakes working together can unleash an avalanche of destruction."

Extensions

"Threshold Models of Social Influence"
Watts and Dodds,
The Oxford Handbook of Analytical Sociology, , 475–497, 2009. [28]

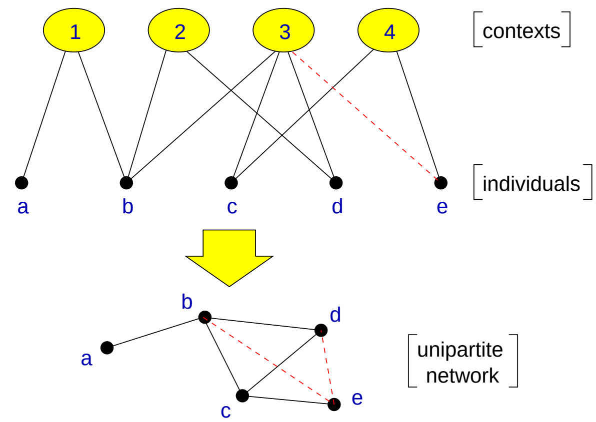
- Assumption of sparse interactions is good
- Degree distribution is (generally) key to a network's function
- Still, random networks don't represent all networks
- Major element missing: group structure

Group structure—Ramified random networks

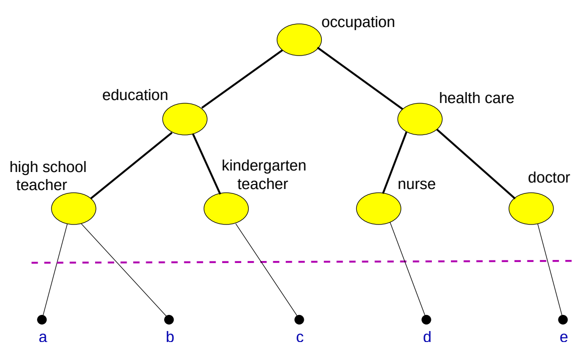


p = intergroup connection probability
q = intragroup connection probability.

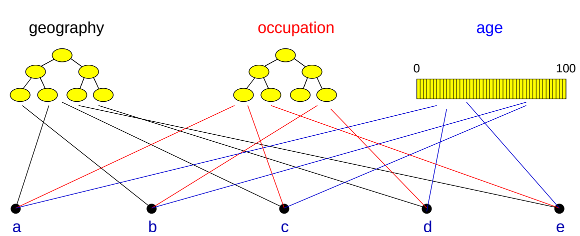
Bipartite networks



Context distance



Generalized affiliation model

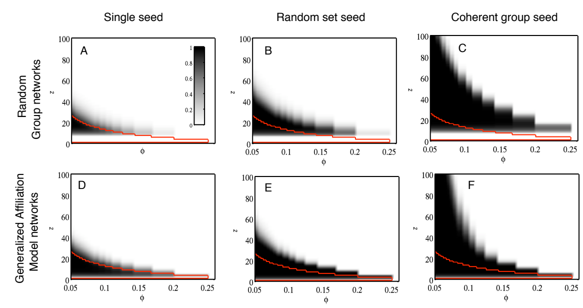


(Blau & Schwartz, Simmel, Breiger)

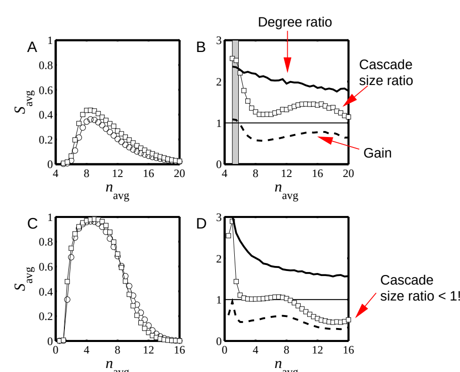
Generalized affiliation model networks with triadic closure

- 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

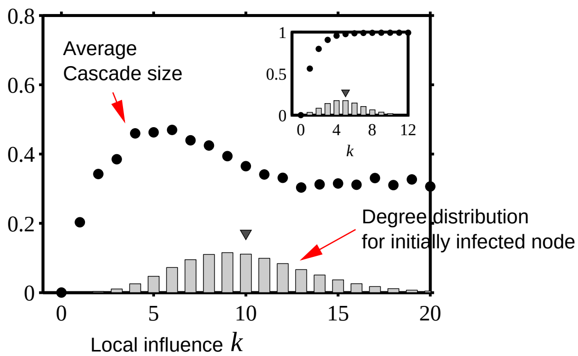


Multiplier effect for group-based networks:



- 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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