Social Contagion

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Principles of Complex Systems, Vol. 1 | @pocsvox CSYS/MATH 300, Fall, 2020

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Background Granovetter's model Network version Final size Spreading success







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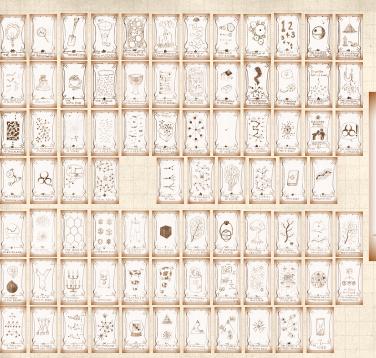
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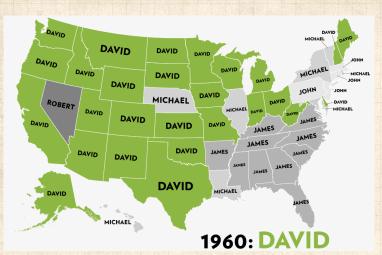












From the Atlantic 2

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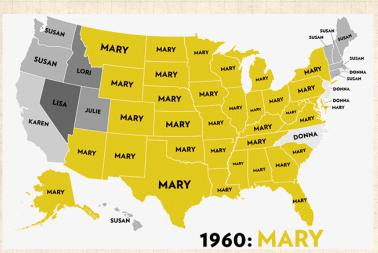
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Richard Feynmann on the Social Sciences:

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Sheldon Cooper on the Social Sciences:

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Things that spread well:

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buzzfeed.com 2:



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

+ News ...







LOL + cute + fail + wtf:

Oopsie!



BUZZFEED FELL DUWN AND WENT BUUN

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The whole lolcats thing:



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Some things really stick:



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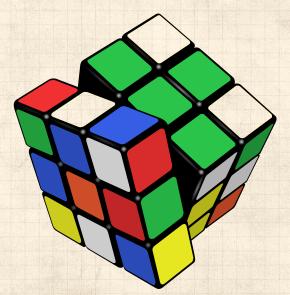






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wtf + geeky + omg:



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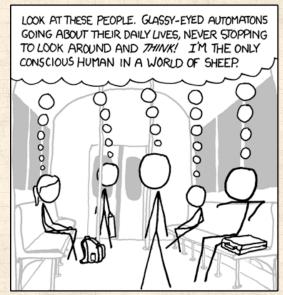




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Why social contagion works so well:



http://xkcd.com/610/

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Social Contagion

Examples abound

ቆ fashion

ቆ striking

residential segregation [22]

iPhones and iThings

obesity
 obesity

🙈 Harry Potter

<page-header> 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, ... PoCS, Vol. 1 @pocsvox Social Contagion

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

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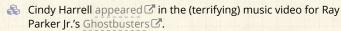
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http://www.youtube.com/watch?v=TgDxWNV4wWY?rel=0



In Stranger Things 2 , Steve Harrington reveals his Fabergé secret .







Market much?

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http://www.youtube.com/watch?v=FEaCflp9gR4?rel=0



Advertisement enjoyed during "Herstory of Dance" , Community S4E08, April 2013.







Framingham heart study:

Evolving network stories (Christakis and Fowler):

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).

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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]

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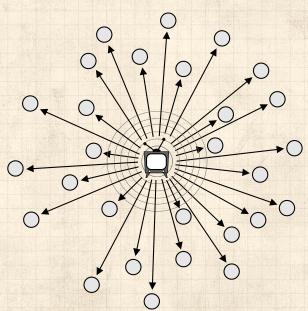
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The hypodermic model of influence



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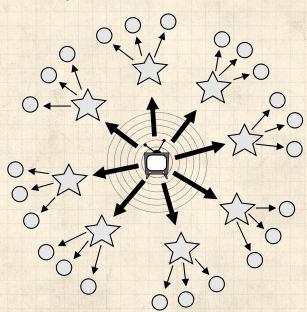
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The two step model of influence [19]



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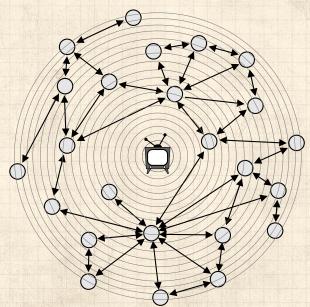
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The general model of influence: the Social Wild



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Talking about 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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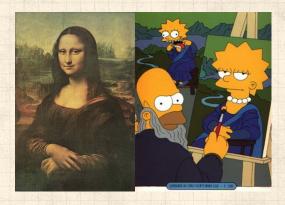
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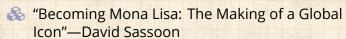
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The Mona Lisa





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:



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

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The dismal predictive powers of editors...



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From a 2013 Believer Magazine I 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 ****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.

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Drafting success in the NFL:

Terrell Owens

89TH PICK, 1998

Peyton Manning

1ST OVER ALL, 1998

Drew Brees

32ND PICK 2001

Top Players by Round, 1995-2012

Zach Thomas

154TH PICK, 1996

Tom Brady

199TH PICK, 2000

Donald Driver

213TH PICK, 1999

Jared Allen

126TH PICK, 2004



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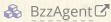


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Messing with social connections

Ads based on message content (e.g., Google and email)



- Harnessing of BzzAgents to directly market through social ties.
- Generally: BzzAgents did not reveal their BzzAgent status and did not want to be paid.
- NYT, 2004-12-05: "The Hidden (in Plain Sight) Persuaders"
- One of Facebook's early advertising attempts: Beacon
- All of Facebook's advertising attempts.
- Seriously, Facebook. What could go wrong?

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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.

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Cialdini's modes are heuristics that help up us get through life.

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Useful but can be leveraged...

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Other acts of influence:

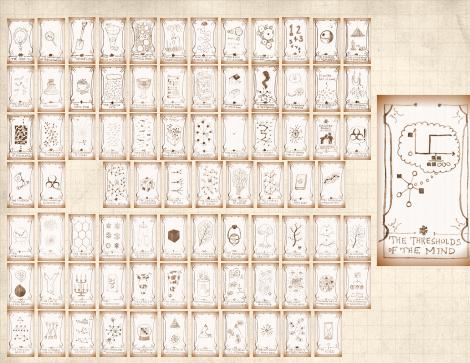
Conspicuous Consumption (Veblen, 1912)

Conspicuous Destruction (Potlatch)









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Some important models:

- Tipping models—Schelling (1971)^[22, 23, 24]
 - Simulation on checker boards
 - ldea 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,...

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Social contagion models

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Thresholds

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

Social Contagion Models

Background









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

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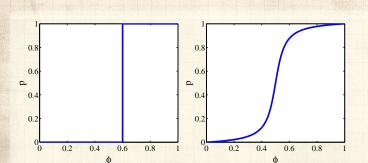
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Threshold models—response functions



Example threshold influence response functions: deterministic and stochastic

 $\Leftrightarrow \phi$ = fraction of contacts 'on' (e.g., rioting)

- Two states: S and I.

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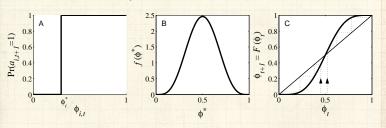






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Action based on perceived behavior of others:

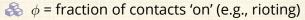


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Two states: S and I.



Discrete time update (strong assumption!)

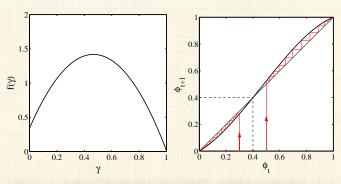
This is a Critical mass model







Another example of critical mass model:



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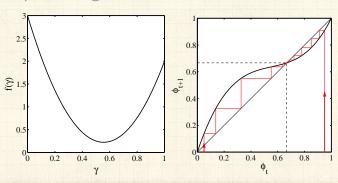
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Example of single stable state model:



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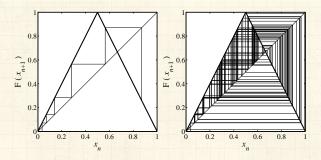
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Chaotic behavior possible [17, 16, 9, 18]



ealso Period doubling arises as map amplitude <math>r is increased.

Synchronous update assumption is crucial

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Threshold models—Nutshell

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Implications for collective action theory:

- 1. Collective uniformity ⇒ individual uniformity
- 2. Small individual changes ⇒ 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 → 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. PoCS, Vol. 1 @pocsvox Social Contagion

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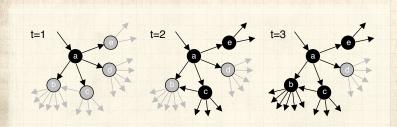
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Threshold model on a network



All nodes have threshold $\phi = 0.2$.

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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.

A Individual *i* becomes active when fraction of active contacts $\frac{a_i}{k_i} \ge \phi_i$.

Individuals remain active when switched (no recovery = SI model).

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Snowballing

First study random networks:

 $\red {\Bbb S}$ 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?

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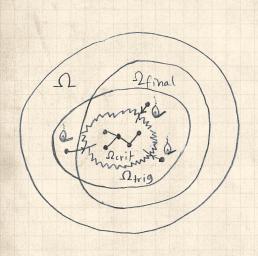
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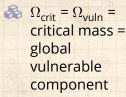
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Example random network structure:





- Ω_{trig} = triggering component
- Ω_{final} = potential extent of spread
- Ω = entire network

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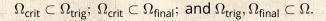
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Snowballing

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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.

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Network version







The most gullible

Vulnerables:

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

$$1/k_i \geq \phi_i$$

- $\red{\$}$ 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.

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Back to following a link:

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

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

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

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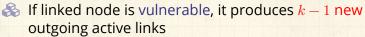


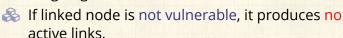
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Next: Vulnerability of linked node

Linked node is vulnerable with probability

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





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Putting things together:

Expected number of active edges produced by an active edge:

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

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

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

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

 $\beta_k = \text{probability a degree } k \text{ node is vulnerable.}$

 $P_k = \text{probability a node has degree } k.$

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Two special cases:

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

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

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

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

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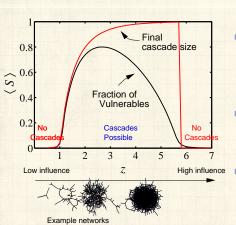
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Cascades on random networks



Cascades occur only if size of max vulnerable

8

System may be 'robust-yetfragile'.

cluster > 0.

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'lgnorance' facilitates spreading. PoCS, Vol. 1 @pocsvox Social Contagion

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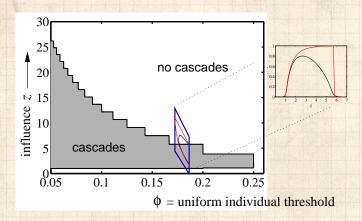
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Cascade window for random networks



 $\ref{Cascade window'}$ widens as threshold ϕ decreases.

🙈 Lower thresholds enable spreading.

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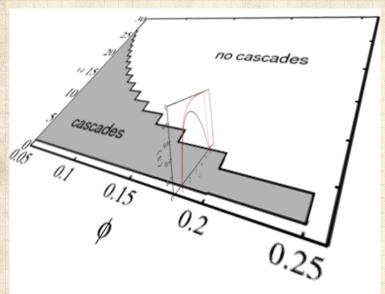
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Cascade window for random networks



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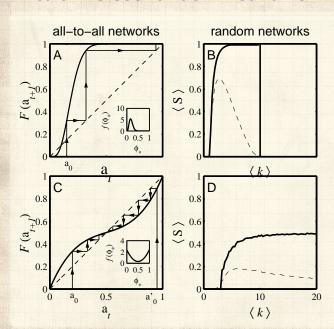






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All-to-all versus random networks



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Cascade window—summary

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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."

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Threshold contagion on random networks

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Next: Find expected fractional size of spread.



Not obvious even for uniform threshold problem.



Difficulty is in figuring out if and when nodes that $need \ge 2$ 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]

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Determining expected size of spread:

 $\ensuremath{ \mathcal{R} }$ 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.

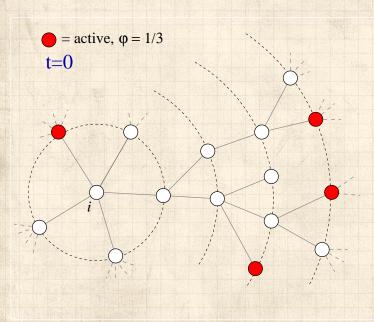
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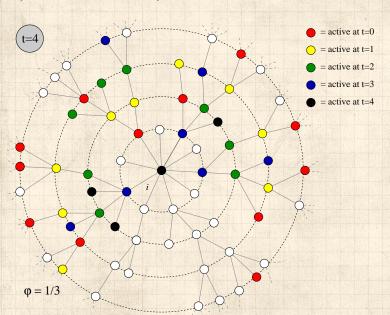
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Notes:

- 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.

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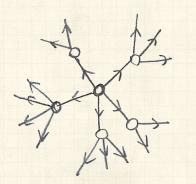


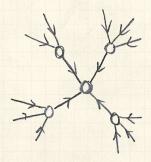


Pleasantness:

Taking off from a single seed story is about expansion away from a node.

Extent of spreading story is about contraction at a node.





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Notation:

 $\phi_{k,t} = \mathbf{Pr}(a \text{ degree } k \text{ node is active at time } t).$

- Notation: $B_{kj} = \mathbf{Pr}$ (a degree k node becomes active if j neighbors are active).
- $\mbox{\&}$ Our starting point: $\phi_{k,0} = \phi_0$.
- $(k \choose j)\phi_0^j(1-\phi_0)^{k-j}=\Pr(j \text{ of a degree } k \text{ node's neighbors were seeded at time } t=0).$
- 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:

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

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For general t, we need to know the probability an edge coming into a degree k node at time t is active.



 \mathbb{A} Notation: call this probability θ_t .



 \Leftrightarrow 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...



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First connect θ_0 to θ_1 :

$$\theta_1 = \phi_0 +$$

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

- $\stackrel{kP_k}{\cancel{(k)}}=R_k$ = **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.
- $\ \ \phi_0 \ \ {\rm and} \ (1-\phi_0)$ terms account for state of node at time t=0.
- $\ensuremath{\mathfrak{S}}$ See this all generalizes to give θ_{t+1} in terms of $\theta_t...$

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

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

social effects

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Expected size of spread

Iterative map for θ_t is key:

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

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

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

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Expected size of spread:

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

 $\red {\Bbb R}$ Depends on map $heta_{t+1} = G(heta_t;\phi_0)$.

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 $k \ge 1$.

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

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Expected size of spread:

In words:

- \Re If $G(0; \phi_0) > 0$, spreading must occur because some nodes turn on for free.

Non-vanishing seed case:

- & Cascade condition is more complicated for $\phi_0 > 0$.
- 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.

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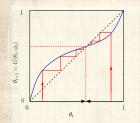


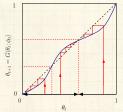


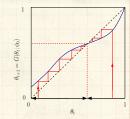


General fixed point story:











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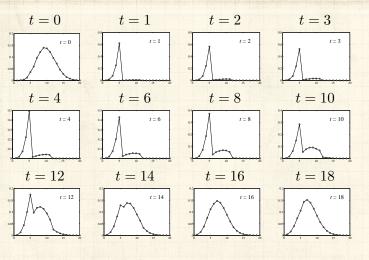
- Given $\theta_0(=\phi_0)$, θ_∞ will be the nearest stable fixed point, either above or below.
- n.b., adjacent fixed points must have opposite stability types.
- $\begin{cases} \& \end{cases}$ Important: Actual form of G depends on ϕ_0 .
- & So choice of ϕ_0 dictates both G and starting point—can't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







Early adopters—degree distributions



 $P_{k,t}$ versus k

Unpublished?

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"Influentials, Networks, and Public Opinion Formation" [7]

Watts and Dodds, J. Consum. Res., **34**, 441–458, 2007. [27]

- Exploration of threshold model of social contagion on various networks.
- 🙈 "Influentials" are limited in power.
- Connected groups of weakly influential-vulnerable" individuals are key.
- Average individuals can have more power than well connected ones.

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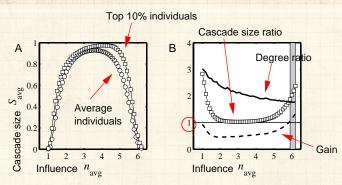
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The multiplier effect:



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Fairly uniform levels of individual influence.

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Multiplier effect is mostly below 1.

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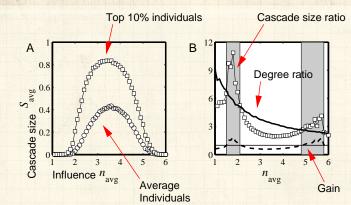
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The multiplier effect:



Skewed influence distribution example.

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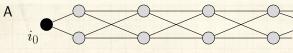
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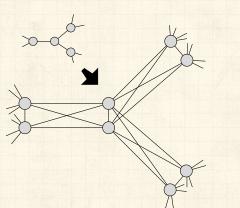




Special subnetworks can act as triggers



В





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

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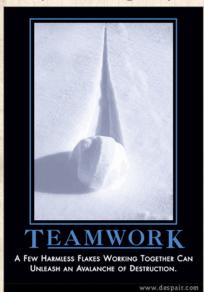
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The power of groups...



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

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Extensions



"Threshold Models of Social Influence"

Watts and Dodds, The Oxford Handbook of Analytical Sociology, 34, 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

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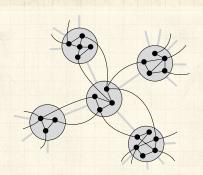
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Group structure—Ramified random networks



p = intergroup connection probability q = intragroup connection probability.

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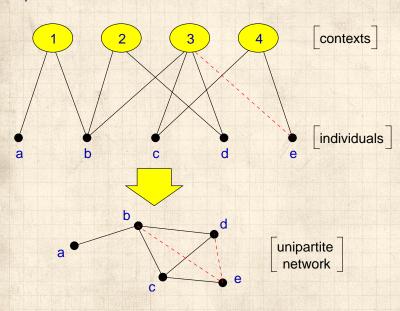
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Bipartite networks



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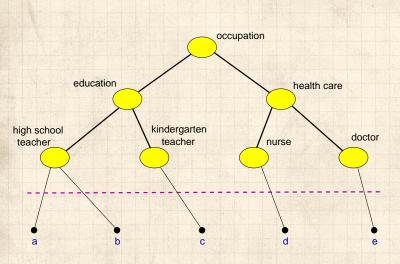
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Context distance



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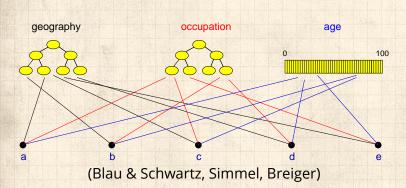
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Generalized affiliation model



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Generalized affiliation model networks with triadic closure



 Connect nodes with probability $\propto e^{-\alpha d}$ where

 α = homophily parameter and

d = distance between nodes (height of lowest common ancestor)



 $\underset{\tau_1}{\&}$ = intergroup probability of friend-of-friend connection



 \mathcal{L}_2 = intragroup probability of friend-of-friend connection

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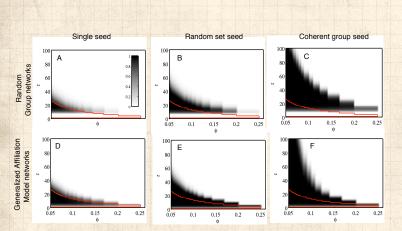
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Cascade windows for group-based networks



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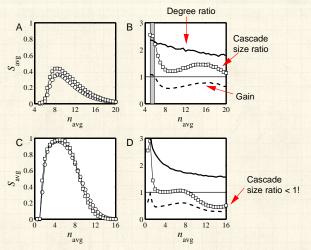






Multiplier effect for group-based networks:





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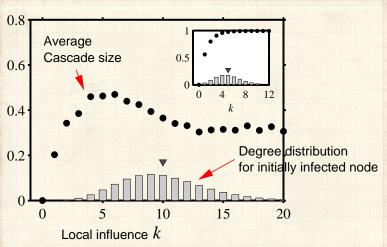






Multiplier almost always below 1.

Assortativity in group-based networks



The most connected nodes aren't always the most 'influential.'

Degree assortativity is the reason.

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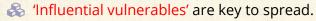




Social contagion

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Summary



- 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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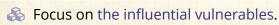


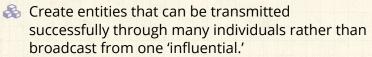


Social contagion

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Implications





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. Social Contagion Models Background

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