# Social Contagion

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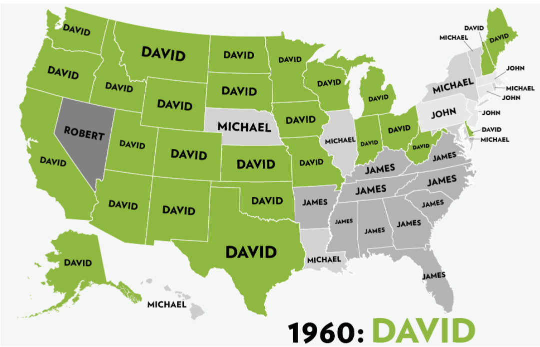
## Outline

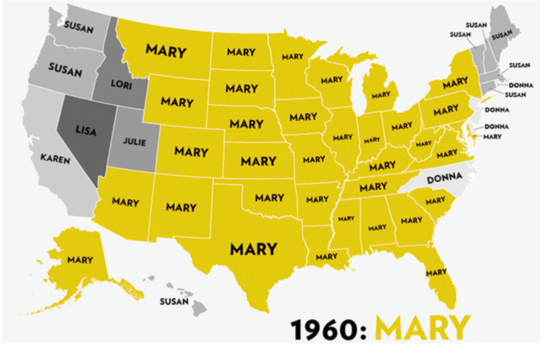







From the Atlantic






From the Atlantic



Richard Feynmann on the Social Sciences:



Sheldon Cooper on the Social Sciences:



## 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:

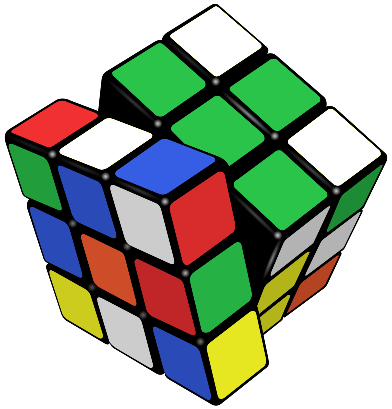
## Some things really stick:







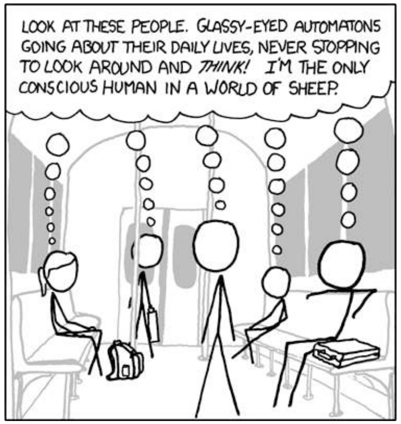
## wtf + geeky + omg:







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





### Market much?

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





## 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

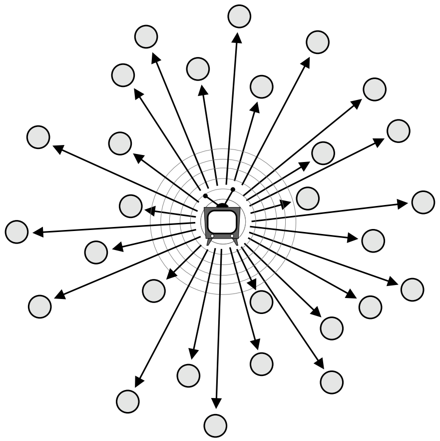
### Two focuses for us

- 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]

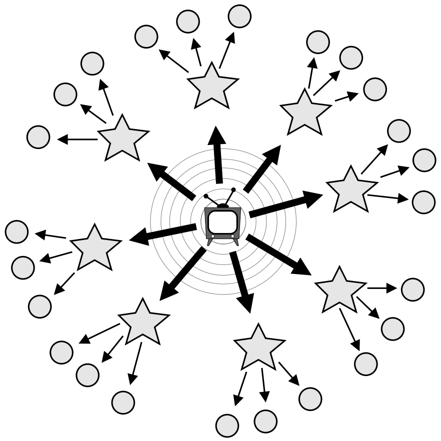
## The hypodermic model of influence







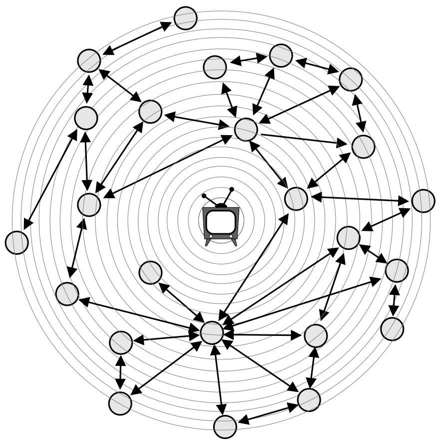
## The two step model of influence [19]







## The general model of influence: the Social Wild







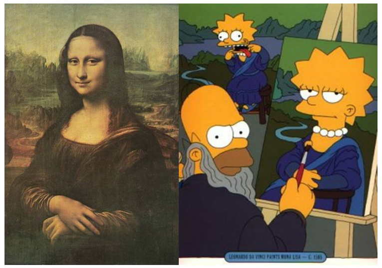
## Why do things spread socially?

- 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 ...





## 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, ...





## 'Tattooed Guy' Was Pivotal in Armstrong Case [nytimes]

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





## 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"





## The dismal predictive powers of editors...





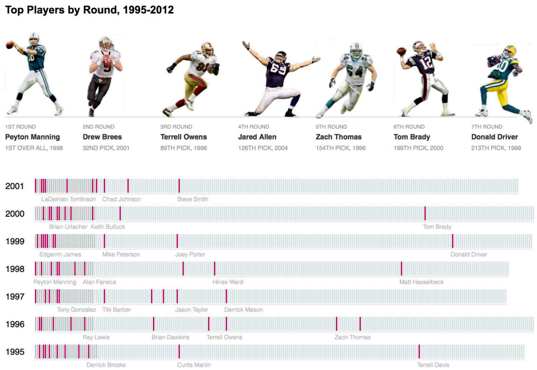

### From a 2013 Believer Magazine interview with Maurice Sendak:

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 ****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
  - 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?





## 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

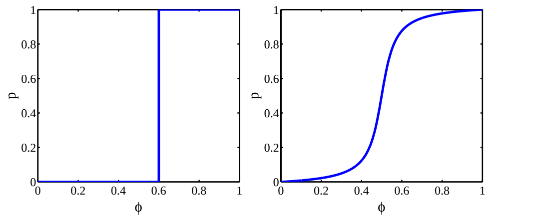
### Some possible origins of thresholds:

- 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



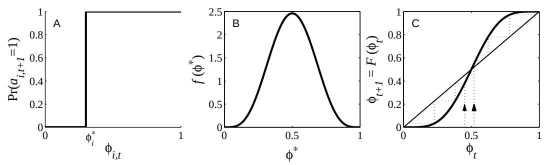

- Example threshold influence response functions: deterministic and stochastic
- $\phi$ = fraction of contacts 'on' (e.g., rioting)
- Two states: S and I.





## Threshold models

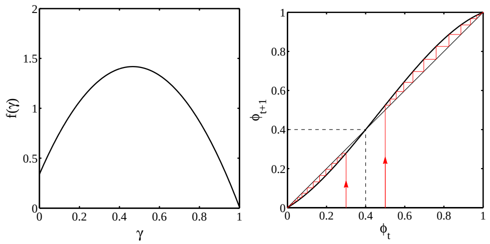
### Action based on perceived behavior of others:




- Two states: S and I.
- $\phi$ = 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:



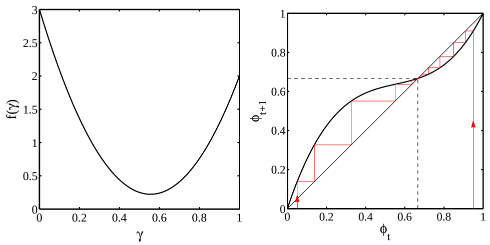







# Threshold models

## Example of single stable state model:



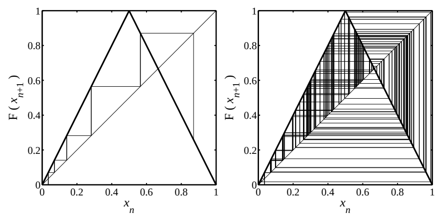







# Threshold models

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




- 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
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.







## 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:

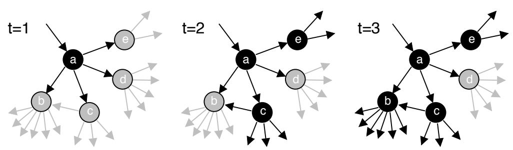
- "Seed size strongly affects cascades on random networks" [14]
  Gleeson and Cahalane, Phys. Rev. E, 2007.
- "Direct, phyiscally motivated derivation of the contagion condition for spreading processes on generalized random networks" [10] Dodds, Harris, and Payne, Phys. Rev. E, 2011
- "Influentials,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







# Threshold model on a network




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







# 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 $\phi_i$.
- 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).







# 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:

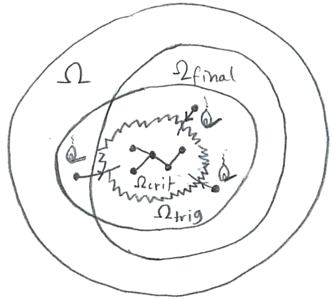
1. If one individual is initially activated, what is the probability that an activation will spread over a network?
2. What features of a network determine whether a cascade will occur or not?







# Example random network structure:




- $\Omega_{\text{crit}} = \Omega_{\text{vuln}}$ = critical mass = global vulnerable component
- $\Omega_{\text{trig}}$ = triggering component
- $\Omega_{\text{final}}$ = potential extent of spread
- $\Omega$ = 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 → critical mass → 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:

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

- $\beta_k$ = 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: $\beta_k = \beta$

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

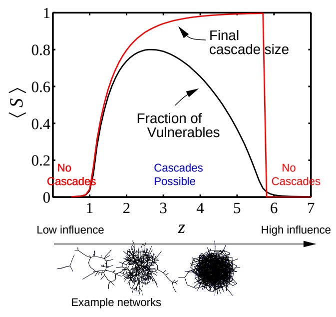
- (2) Giant component exists: $\beta = 1$

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



# Cascades on random networks



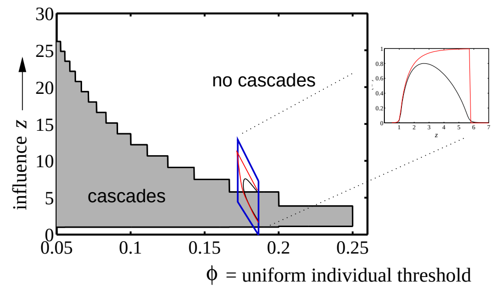



- Cascades occur only if size of max vulnerable cluster $> 0$.
- System may be 'robust-yet-fragile'.
- 'Ignorance' facilitates spreading.



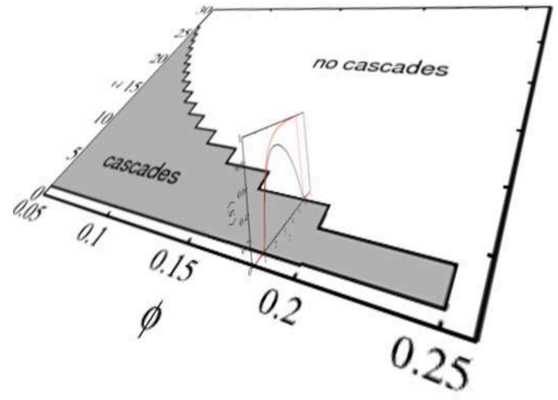
# Cascade window for random networks






- 'Cascade window' widens as threshold $\phi$ decreases.
- Lower thresholds enable spreading.

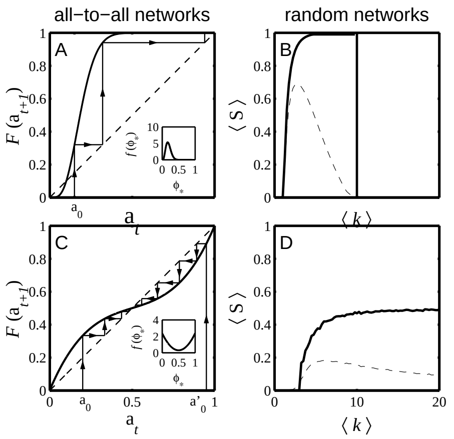
# Cascade window for random networks








# 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 $\geq 2$ 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]





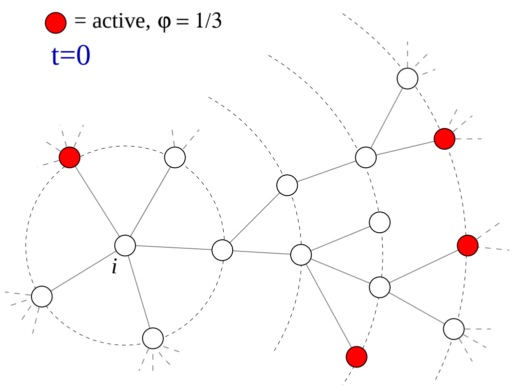
# Determining expected size of spread:

- Randomly turn on a fraction $\phi_0$ 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 = $\phi_0$)
  - $t = 1$: $i$ 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 $i$.





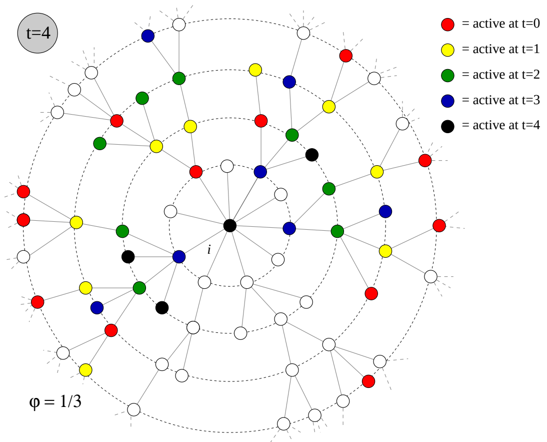
# Expected size of spread








# 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.

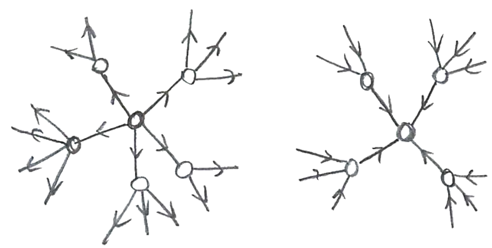




# Expected size of spread

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:
$\phi_{k,t}$ = **Pr**(a degree $k$ node is active at time $t$).
- Notation: $B_{kj}$ = **Pr** (a degree $k$ node becomes active if $j$ neighbors are active).
- Our starting point: $\phi_{k,0} = \phi_0$.
- $\binom{k}{j}\phi_0^j(1-\phi_0)^{k-j}$ = **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 $\phi_0$ (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 $\theta_t$.
- 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 $\phi_{t+1}$:

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

- So we need to compute $\theta_t$... massive excitement...



## Expected size of spread

First connect $\theta_0$ to $\theta_1$:

- $\theta_1 = \phi_0 +$

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

- $\frac{kP_k}{\langle k\rangle} = R_k$ = **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.
- $\phi_0$ and $(1-\phi_0)$ terms account for state of node at time $t=0$.
- See this all generalizes to give $\theta_{t+1}$ in terms of $\theta_t$...



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

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



## Expected size of spread

Iterative map for $\theta_t$ is key:

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

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

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



## 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:

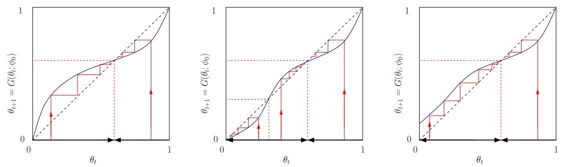
- If $G(0;\phi_0) > 0$, spreading must occur because some nodes turn on for free.
- If $G$ has an unstable fixed point at $\theta = 0$, then cascades are also always possible.

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 $\phi_0$, so as we change $\phi_0$, we also change $G$.
- A version of a critical mass model again.



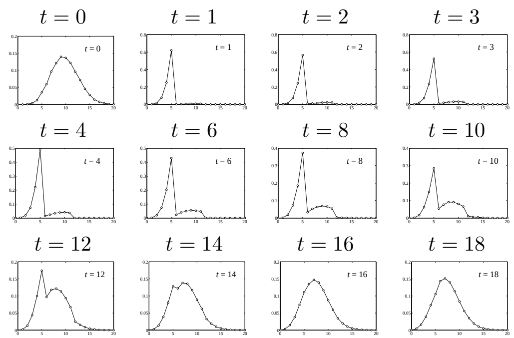
## General fixed point story:



- Given $\theta_0(=\phi_0)$, $\theta_\infty$ 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 $\phi_0$.
- So choice of $\phi_0$ dictates both $G$ and starting point—can't start anywhere for a given $G$.

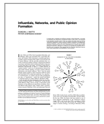


## Early adopters—degree distributions




$P_{k,t}$ versus $k$

Unpublished?



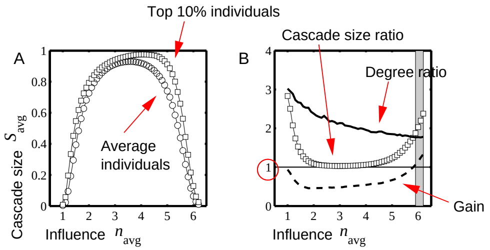
"Influentials, Networks, and Public Opinion Formation"
Watts and Dodds,
J. Consum. Res., **34**, 441–458, 2007. [27]

- 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.





## The multiplier effect:

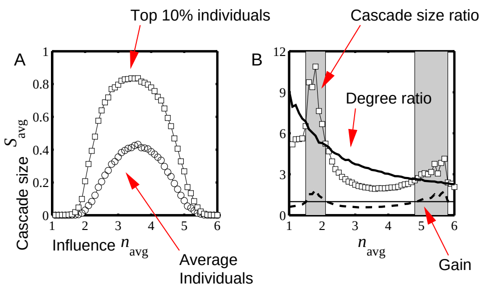



- Fairly uniform levels of individual influence.
- Multiplier effect is mostly below 1.





## The multiplier effect:

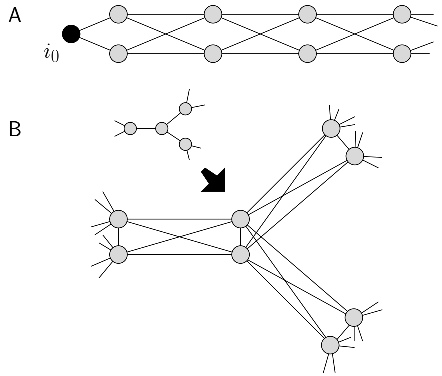



- Skewed influence distribution example.





## Special subnetworks can act as triggers

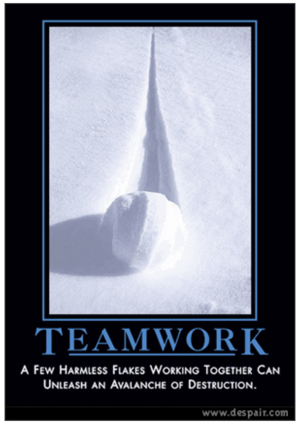



- $\phi = 1/3$ for all nodes





## The power of groups...




despair.com

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





## Extensions

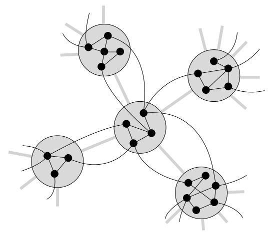
"Threshold Models of Social Influence"
Watts and Dodds,
The Oxford Handbook of Analytical Sociology, **34**, 475–497, 2009. [28]

- 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





## Group structure—Ramified random networks

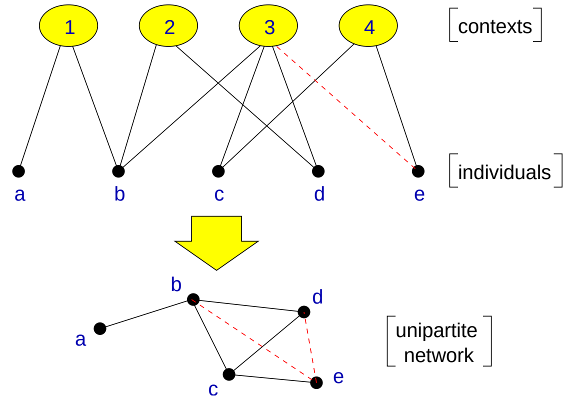


$p$ = intergroup connection probability
$q$ = intragroup connection probability.





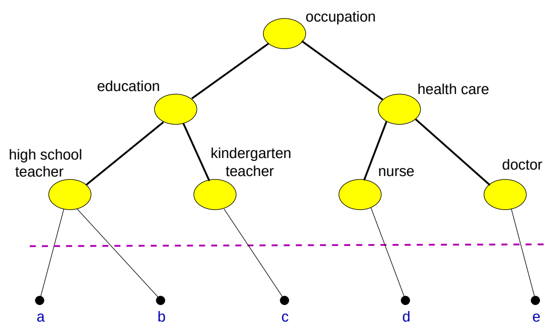
## Bipartite networks





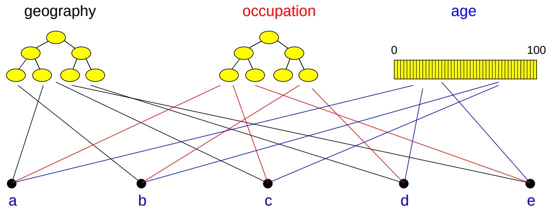



## Context distance



## Generalized affiliation model






(Blau & Schwartz, Simmel, Breiger)



## Generalized affiliation model networks with triadic closure

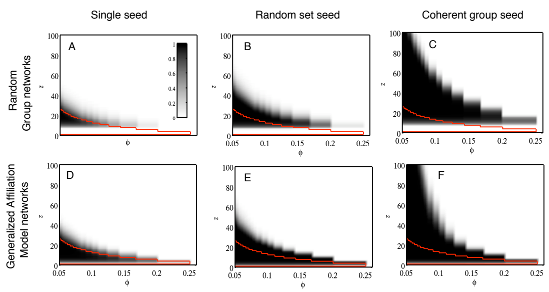


- Connect nodes with probability $\propto e^{-\alpha d}$ where
  $\alpha$ = homophily parameter
  and
  $d$ = distance between nodes (height of lowest common ancestor)
- $\tau_1$ = intergroup probability of friend-of-friend connection
- $\tau_2$ = intragroup probability of friend-of-friend connection



## Cascade windows for group-based networks

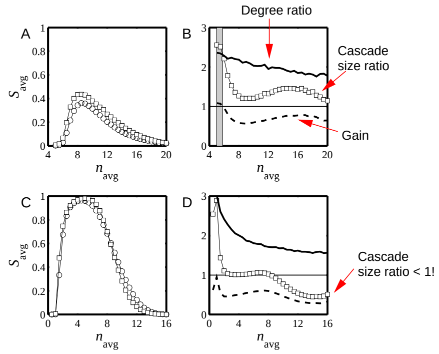







## Multiplier effect for group-based networks:



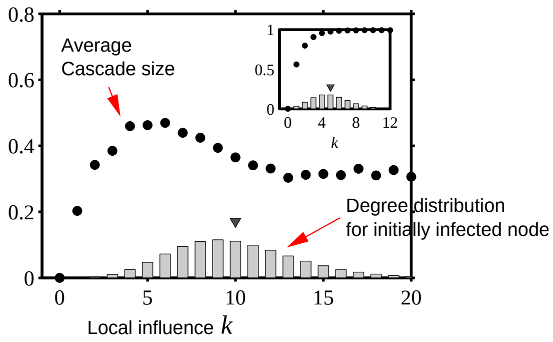



- Multiplier almost always below 1.



## 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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