

# Social Contagion

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Principles of Complex Systems, Vols. 1, 2, & 3D  
CSYS/MATH 6701, 6713, & a pretend number,  
2023–2024 | @pocsvox

Prof. Peter Sheridan Dodds | @peterdodds

Computational Story Lab | Vermont Complex Systems Center  
Santa Fe Institute | University of Vermont

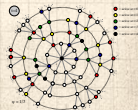


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References



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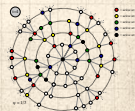
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Sealie & Lambie  
Productions

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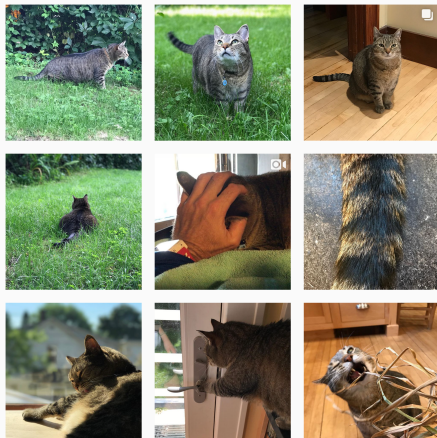
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## Special Guest Executive Producer



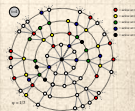
 On Instagram at [pratchett\\_the\\_cat](https://www.instagram.com/pratchett_the_cat) 

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# Outline

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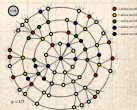
Final size

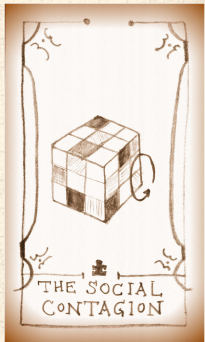
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## References



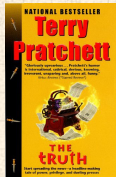


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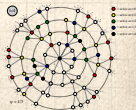
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'The rumor spread through the city like wildfire which had quite often spread through Ankh-Morpork since its citizens had learned the words "fire insurance").'



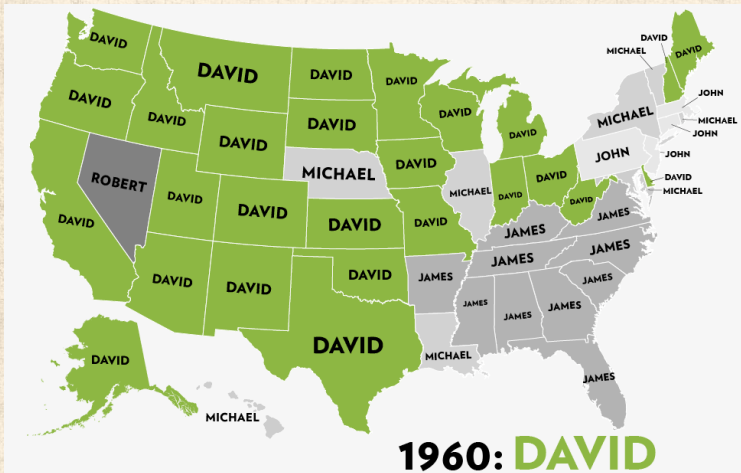
"The Truth" [a](#)   
by Terry Pratchett (2000). [22]



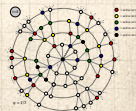
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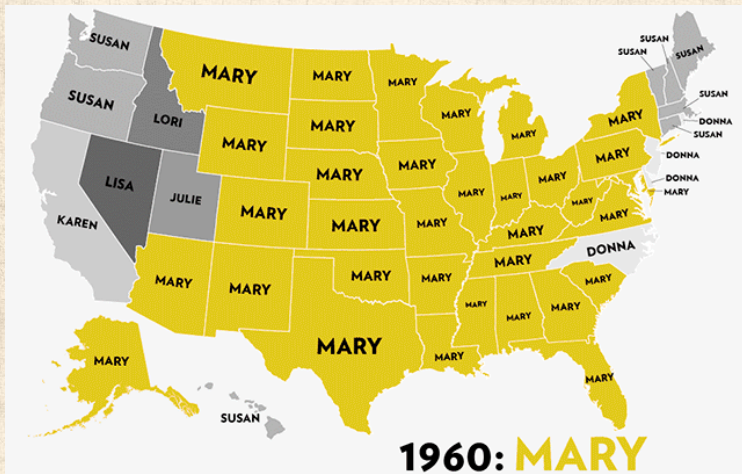
From the Atlantic ↗



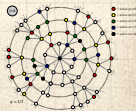
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From the Atlantic 







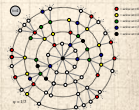
# Things that spread well:

[buzzfeed.com](http://buzzfeed.com) 



 Dangerously self aware: [11 Elements that make a perfect viral video.](#) 

+ News ...



LOL + cute + fail + wtf:

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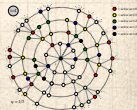
References

# Oopsie!



**BUZZFEED FELL DOWN AND WENT BOOM.**

Please try reloading this page. If the problem persists [let us know](#).



# The whole lolcats thing:

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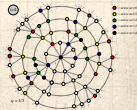
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:-p



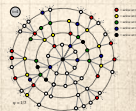
# Some things really stick:

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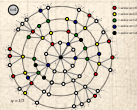
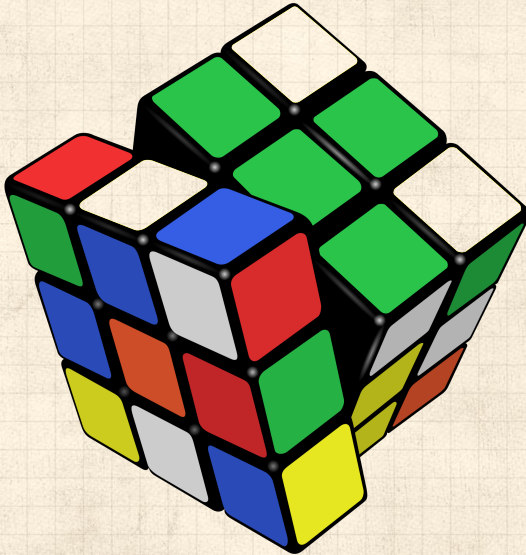
wtf + geeky + omg:

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# Why social contagion works so well:

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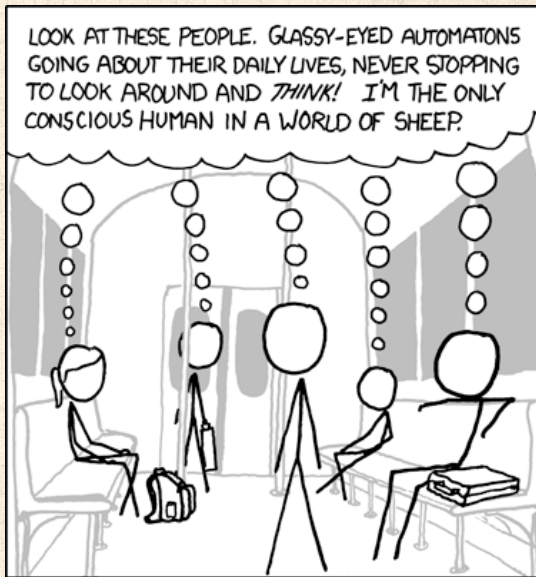
Network version

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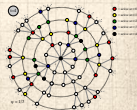
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<http://xkcd.com/610/>



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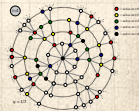
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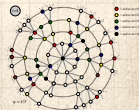
# Social Contagion

Examples are claimed to abound:

-  Fashion
-  Striking
-  smoking  [7]
-  Residential segregation [23]
-  iPhones and iThings
-  obesity  [6]
-  Stupidity
-  Harry Potter
-  voting
-  gossip
-  Rubik's cube 
-  religious beliefs
-  school shootings
-  yawning 
-  **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 ...

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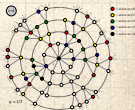
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## References

<https://www.youtube.com/watch?v=TgDxWNV4wWY?rel=0>

 [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?

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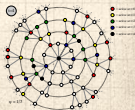
Groups

## References

<https://www.youtube.com/watch?v=FEaCflp9qR4?rel=0>











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



# Framingham heart study:

## Evolving network stories (Christakis and Fowler):

-  The spread of quitting smoking  <sup>[7]</sup>
-  The spread of spreading  <sup>[6]</sup>
-  Also: happiness  <sup>[11]</sup>, loneliness, ...
-  The book: Connected: The Surprising Power of Our Social Networks and How They Shape Our Lives 

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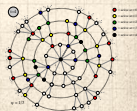
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## Controversy:

-  Are your friends making you fat?  (Clive Thomson, 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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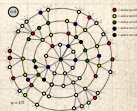
References

## 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<sup>[12]</sup> as 'connectors'
- The infectious idea of opinion leaders (Katz and Lazarsfeld)<sup>[19]</sup>

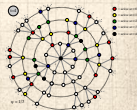
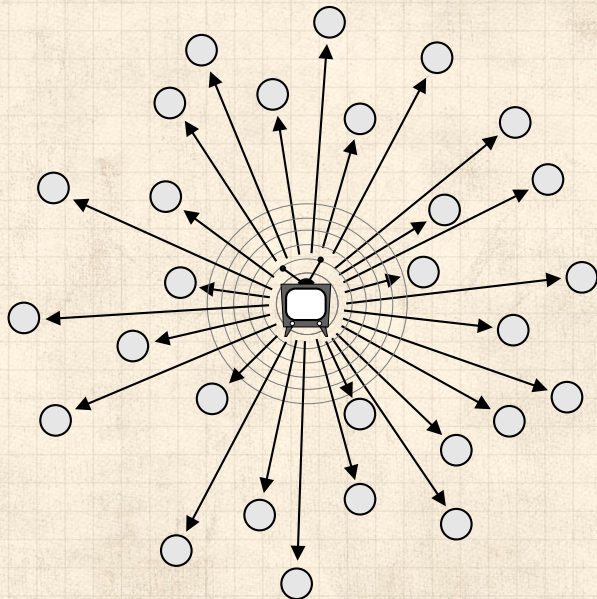


# The hypodermic model of influence

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# The two step model of influence <sup>[19]</sup>

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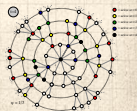
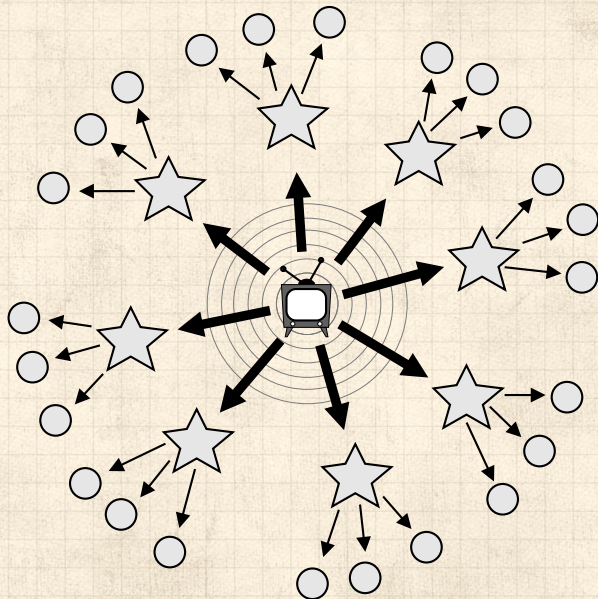
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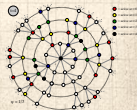
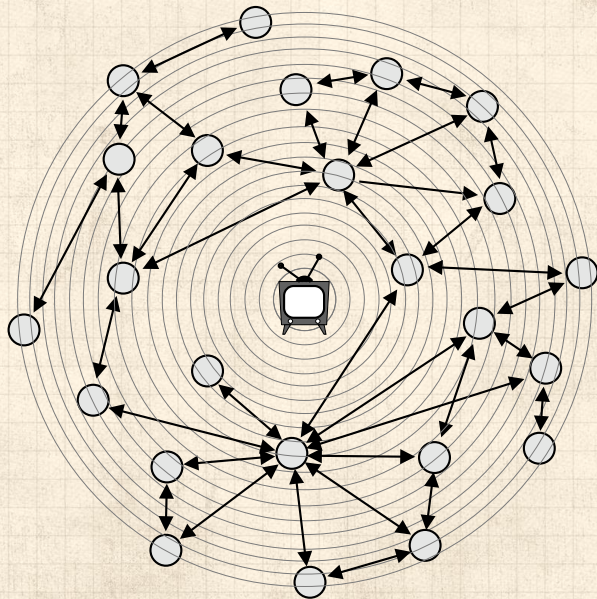
# The general model of influence: the Social Wild

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## Talking about the social wild:

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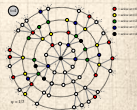
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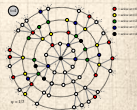
### References





## 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

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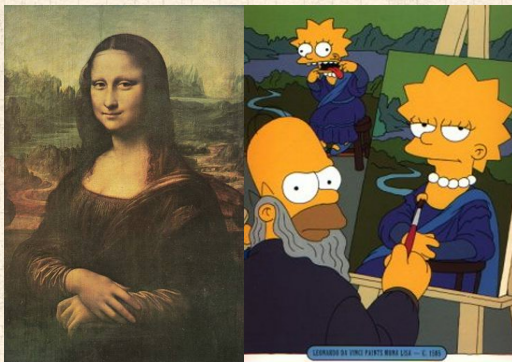
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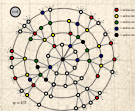
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- 🧱 “Becoming Mona Lisa: The Making of a Global Icon”—David Sassoon
- 🧱 Not the world’s greatest painting from the start...
- 🧱 Escalation through theft, vandalism, **parody**, ...



# 'Tattooed Guy' Was Pivotal in Armstrong Case

[nytimes] 



“... Leogrande’s doping sparked a series of events  
...”

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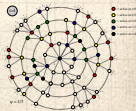
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# The completely unpredicted fall of Eastern Europe:



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

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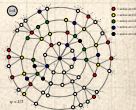
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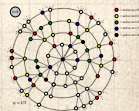
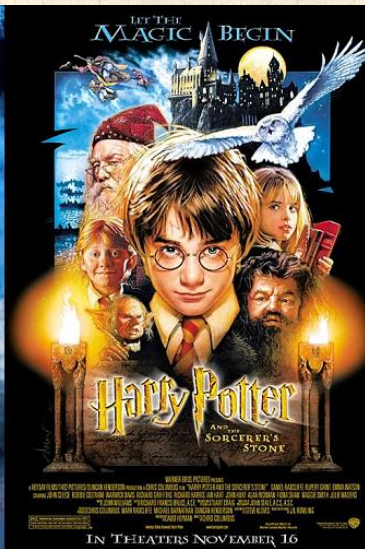
# The dismal predictive powers of editors...

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## 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](#) [↗](#).

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# Drafting success in the NFL: ↗

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### Top Players by Round, 1995-2012



1ST ROUND  
**Peyton Manning**  
1ST OVER ALL, 1998



2ND ROUND  
**Drew Brees**  
32ND PICK, 2001



3RD ROUND  
**Terrell Owens**  
89TH PICK, 1996



4TH ROUND  
**Jared Allen**  
126TH PICK, 2004



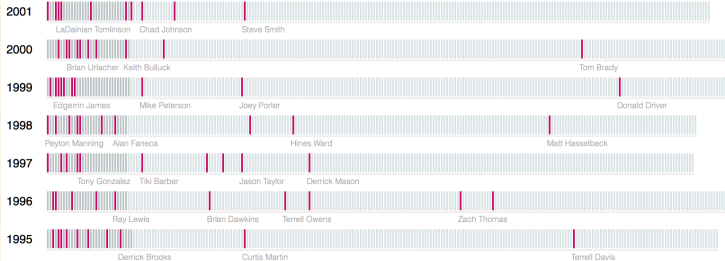
5TH ROUND  
**Zach Thomas**  
154TH PICK, 1996






6TH ROUND  
**Tom Brady**  
199TH PICK, 2000



7TH ROUND  
**Donald Driver**  
213TH PICK, 1999



## Messing with social connections

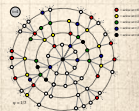
- 📦 Ads based on message content (e.g., Google and email)
- 📦 BzzAgent 
  - 📦 Harnessing of BzzAgents to directly market through social ties.
  - 📦 Generally: BzzAgents did not reveal their BzzAgent status and did not want to be paid.
  - 📦 NYT, 2004-12-05: "The Hidden (in Plain Sight) Persuaders" 
-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

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# Getting others to do things for you

A very good book: 'Influence'<sup>[8]</sup> 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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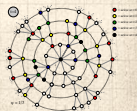
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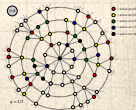
 Cialdini's modes are heuristics that help up us get through life.

 Useful but can be leveraged...

Other acts of influence:









 Conspicuous Consumption (Veblen, 1912)

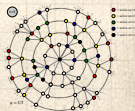
 Conspicuous Destruction (Potlatch)





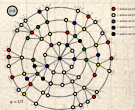
## Some important models:

-  Tipping models—Schelling (1971) [23, 24, 25]
  -  Simulation on checker boards
  -  Idea of thresholds
  -  Polygon-themed online visualization. (Includes optional diversity-seeking proclivity.) 
-  Threshold models—Granovetter (1978) [15]
-  Herding models—Bikhchandani, Hirschleifer, Welch (1992) [2, 3]
  -  Social learning theory, Informational cascades,...









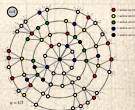
## Thresholds

- 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)**.

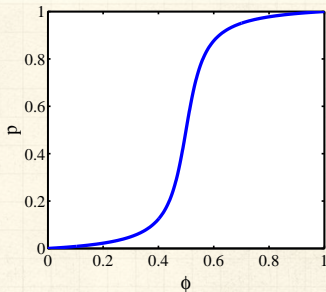
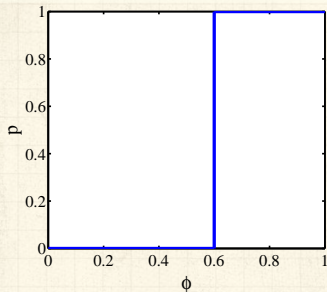



## Some possible origins of thresholds:


-  Inherent, evolution-devised inclination to coordinate, to conform, to imitate. <sup>[1]</sup>
-  **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, TikTok, operating systems
  -  An individual's utility increases with the adoption level among peers and the population in general




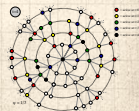
# Threshold models—response functions



 Example threshold influence response functions:  
**deterministic** and **stochastic**

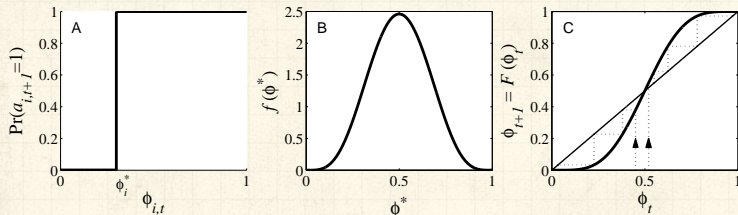
  $\phi$  = fraction of contacts 'on' (e.g., rioting)

 Two states: S and I.



# Threshold models

Action based on perceived behavior of others:



Two states: S and I.



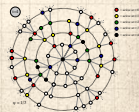
$\phi$  = fraction of contacts 'on' (e.g., rioting)



Discrete time update (strong assumption!)

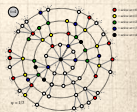
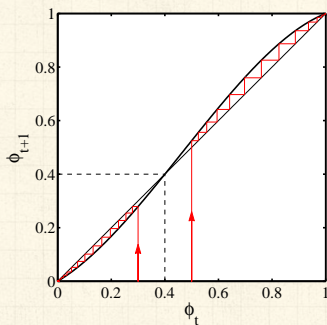
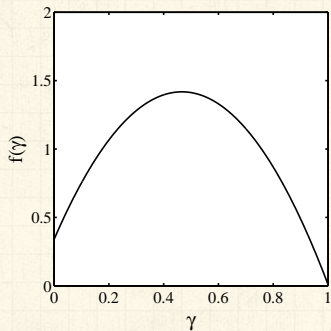


This is a **Critical mass model**





Another example of critical mass model:



# Threshold models

## Social Contagion Models

Background

Granovetter's model

Network version

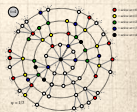
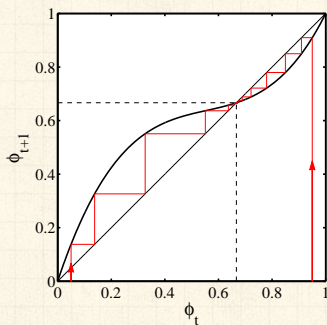
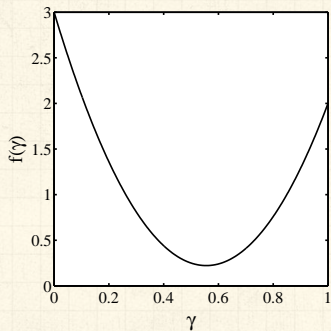
Final size

Spreading success

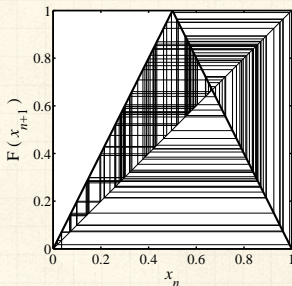
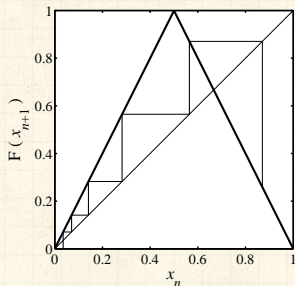
Groups


References


Example of single stable state model:

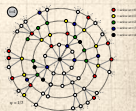


## Chaotic behavior possible [17, 16, 9, 18]



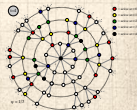
 Period doubling arises as map amplitude  $r$  is increased.

 Synchronous update assumption is crucial



## Implications for collective action theory:

1. Collective uniformity  $\nRightarrow$  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 [27]



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.

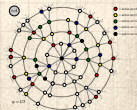


"Direct, physically motivated derivation of the contagion condition for spreading processes on generalized random networks" [10] Dodds, Harris, and Payne, Phys. Rev. E, 2011



"Influentials, Networks, and Public Opinion Formation" [28]

Watts and Dodds, J. Cons. Res., 2007.



# Threshold model on a network

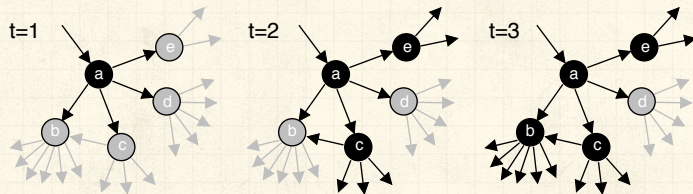
## Social Contagion Models


Background  
Granovetter's model

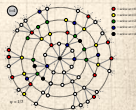
Network version  
Final size

Spreading success  
Groups

References

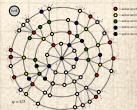


 All nodes have threshold  $\phi = 0.2$ .



# 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).



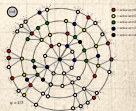
# Snowballing

## 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**

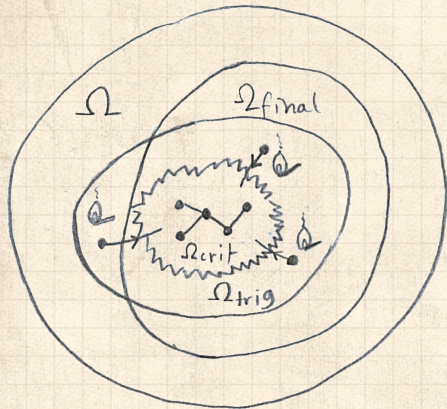
## The Cascade Condition:


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:



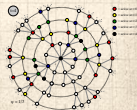
  $\Omega_{crit} = \Omega_{vuln} =$   
critical mass =  
global  
vulnerable  
component

  $\Omega_{trig} =$   
triggering  
component

  $\Omega_{final} =$   
potential  
extent of  
spread

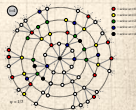
  $\Omega =$  entire  
network

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




## 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 neighbors 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  $i$ :

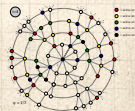
$$1/k_i \geq \phi_i$$

 Which means # contacts  $k_i \leq \lfloor 1/\phi_i \rfloor$

 For global cascades on random networks, must have a *global cluster of vulnerables* <sup>[27]</sup>


 **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

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

## Social Contagion Models

Background

Granovetter's model

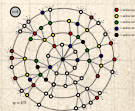
Network version

Final size


Spreading success

Groups


## References




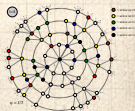
## Next: Vulnerability of linked node

 Linked node is **vulnerable** with probability

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

 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


## Social Contagion Models

Background  
Granovetter's model  
Network version  
Final size

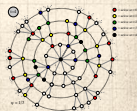
Spreading success  
Groups

References

Putting things together:

 Expected number of active edges produced by an active edge:


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




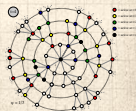
# Cascade condition

So... for random networks with fixed degree distributions, cascades 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  $k$ .



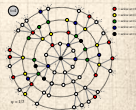
Two special cases:

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

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

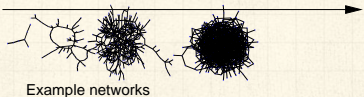
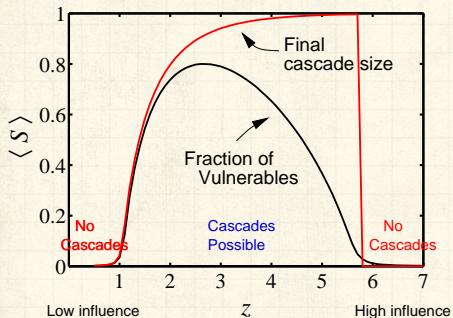
 (2) Giant component exists:  $\beta = 1$

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





# Cascades on random networks



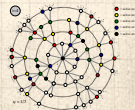
Cascades occur only if size of max vulnerable cluster  $> 0$ .



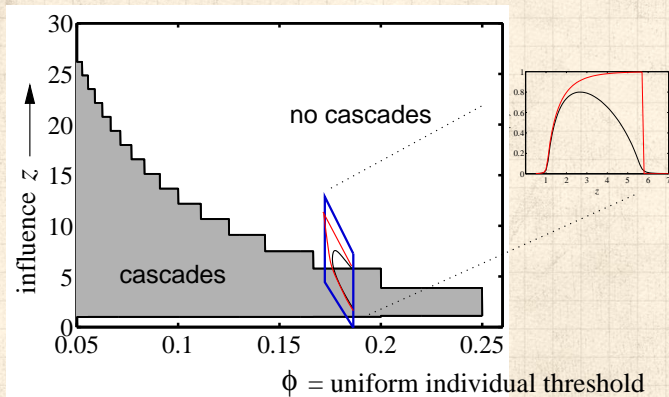
System may be 'robust-yet-fragile'.





'Ignorance' facilitates spreading.

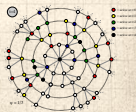


# Cascade window for random networks

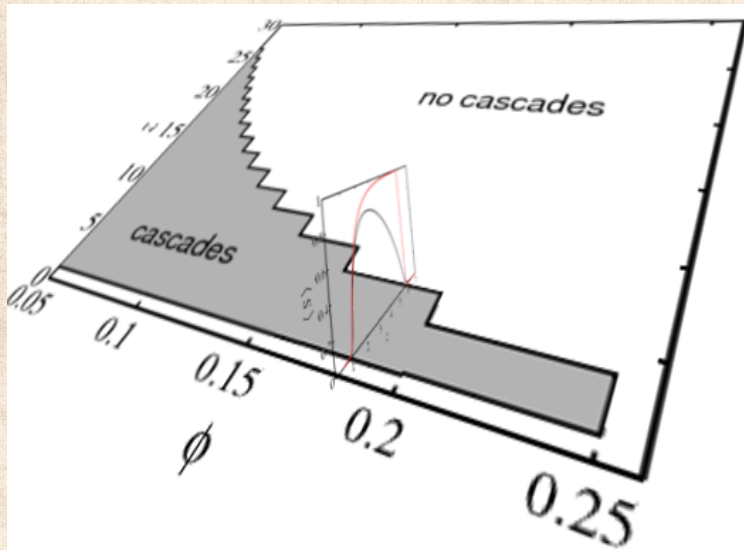


 'Cascade window' widens as threshold  $\phi$  decreases.

 Lower thresholds enable spreading.



# Cascade window for random networks



The PoCSverse  
Social Contagion  
65 of 112

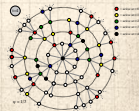
Social Contagion  
Models

Background  
Granovetter's model

**Network version**  
Final size

Spreading success  
Groups

References



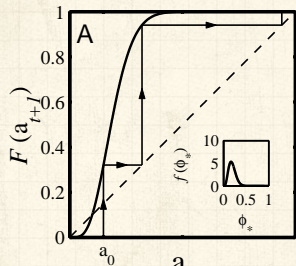
# All-to-all versus random networks

## Social Contagion Models

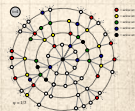
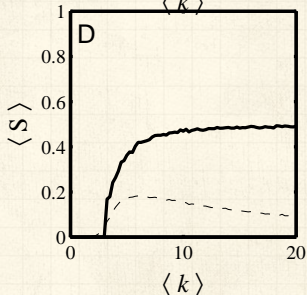
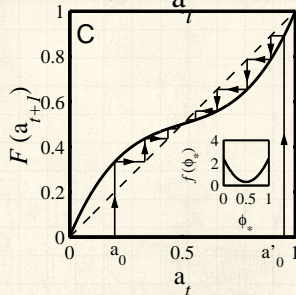
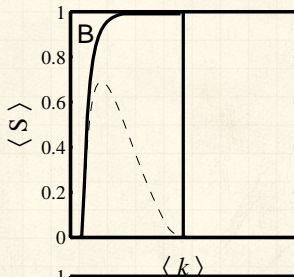
- Background
- Granovetter's model
- Network version**
- Final size
- Spreading success
- Groups

## References

all-to-all networks



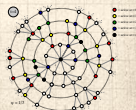
random networks



# Cascade window—summary

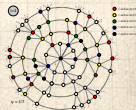
For our simple model of a uniform threshold:

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."**



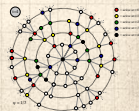
# Threshold contagion on random networks

- 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. <sup>[14]</sup>
- Developed further by Gleeson in "Cascades on correlated and modular random networks," Phys. Rev. E, 2008. <sup>[13]</sup>



# 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  $i$ .

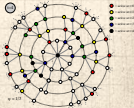
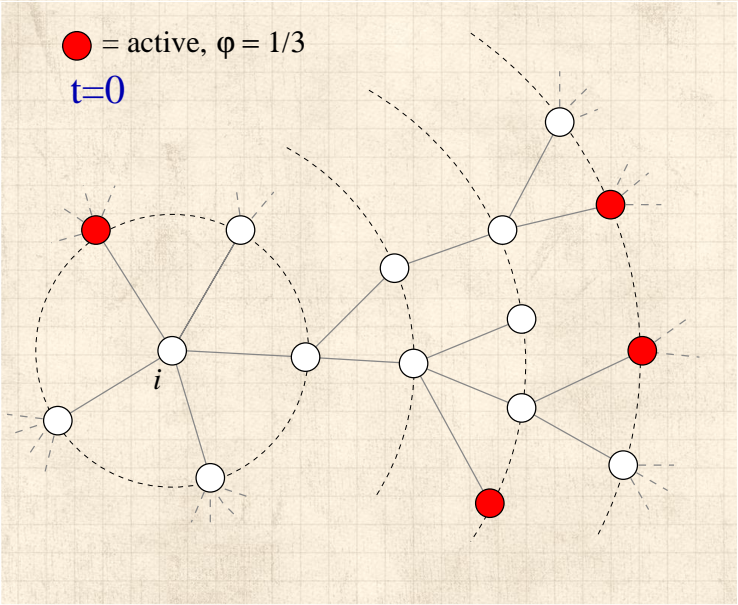


# Expected size of spread

## Social Contagion Models

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

## References



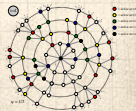
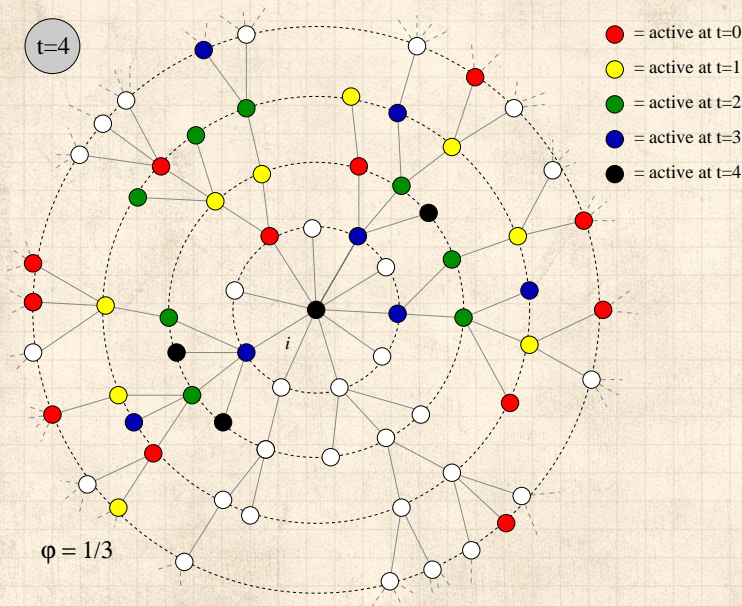


# Expected size of spread

## Social Contagion Models

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# Expected size of spread

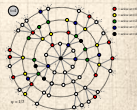
## Social Contagion Models

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### Notes:

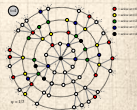
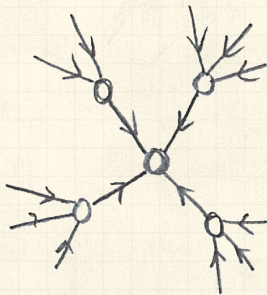
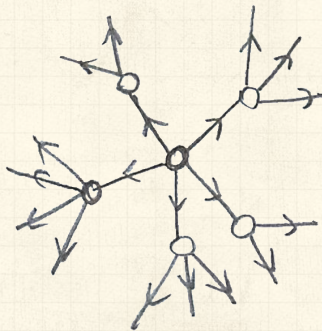
- Calculations are possible if nodes do not become inactive (strong restriction).
- Not just for threshold model—works for a wide range of contagion processes.
- We can analytically determine the entire time evolution, not just the final size.
- We can in fact determine **Pr**(node of degree  $k$  switching 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  $\phi_0$  (as above).



Probability a degree  $k$  node was not a seed at  $t = 0$  is  $(1 - \phi_0)$ .



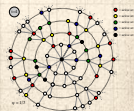
Combining everything, we have:

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

## Social Contagion Models

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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.

**Notation:** call this probability  $\theta_t$ .

We already know  $\theta_0 = \phi_0$ .

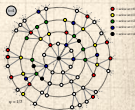
Story analogous to  $t = 1$  case. For node  $i$ :

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

Average over all nodes to obtain expression for  $\phi_{t+1}$ :


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

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





# Expected size of spread


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


  $\theta_1 = \phi_0 +$

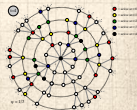
$$(1 - \phi_0) \sum_{k=1}^{\infty} \frac{k P_k}{\langle k \rangle} \sum_{j=0}^{k-1} \binom{k-1}{j} \theta_0^j (1 - \theta_0)^{k-1-j} B_{kj}$$

  $\frac{k P_k}{\langle k \rangle} = R_k = \mathbf{Pr}$  (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.

  $\phi_0$  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  $\theta_t \dots$



# Expected size of spread

Two pieces: edges first, and then nodes

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

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

with  $\theta_0 = \phi_0$ .

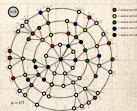
$$2. \phi_{t+1} =$$

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

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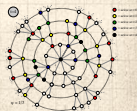
# Expected size of spread

Iterative map for  $\theta_t$  is key:

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

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

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





# Expected size of spread:

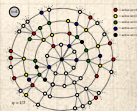
- Retrieve cascade condition for spreading from a single seed in limit  $\phi_0 \rightarrow 0$ .
- Depends on map  $\theta_{t+1} = G(\theta_t; \phi_0)$ .
- First: if self-starters are present, some activation is assured:

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

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

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

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



# Expected size of spread:

## In words:

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

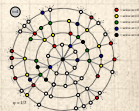
## Non-vanishing seed case:

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

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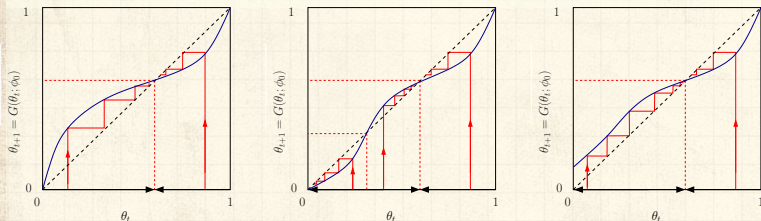



# General fixed point story:


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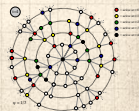


 Given  $\theta_0 (= \phi_0)$ ,  $\theta_\infty$  will be the nearest stable fixed point, either above or below.

 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  $G$ .

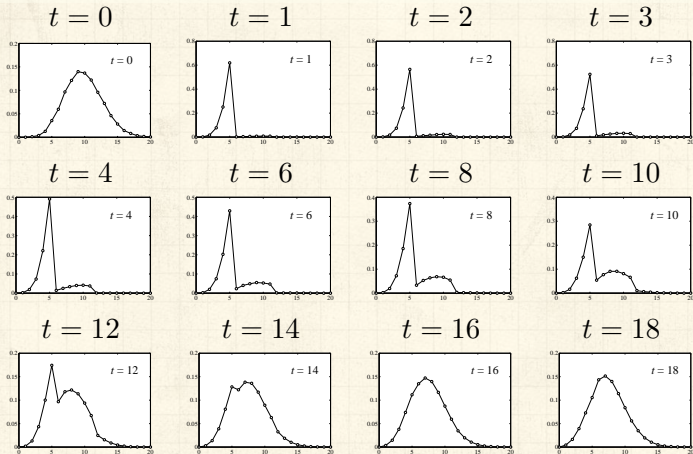


# Early adopters—degree distributions

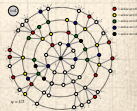
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$P_{k,t}$  versus  $k$

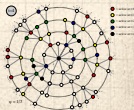




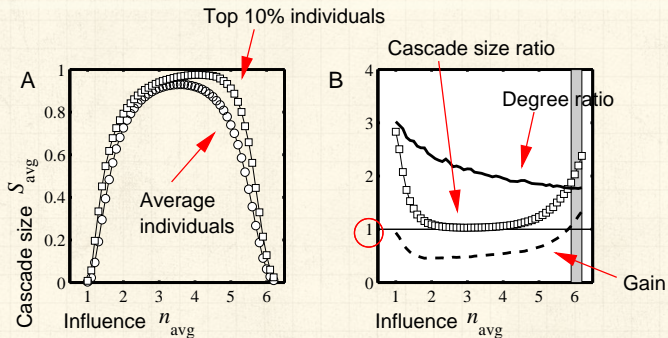
## "Influentials, Networks, and Public Opinion Formation"

Watts and Dodds,  
J. Consum. Res., **34**, 441–458, 2007. [28]

-  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.



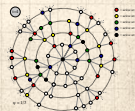
# The multiplier effect:



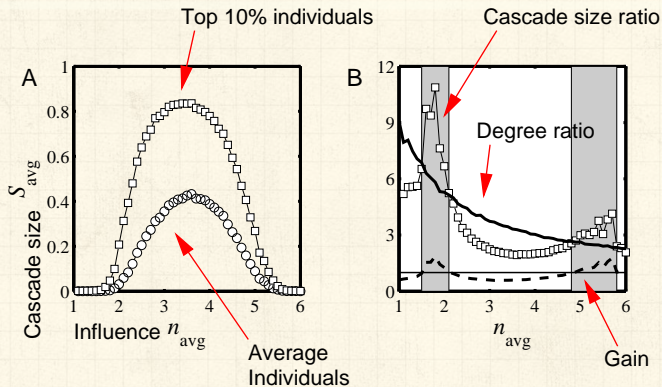
Fairly uniform levels of individual influence.



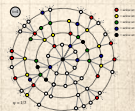
Multiplier effect is mostly below 1.



# The multiplier effect:



Skewed influence distribution example.

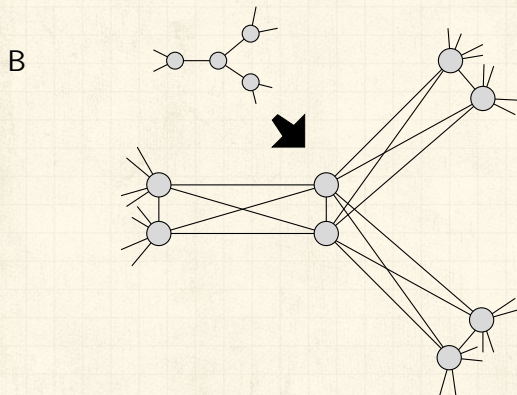
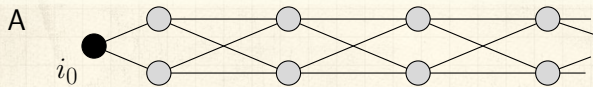



# Special subnetworks can act as triggers

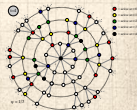
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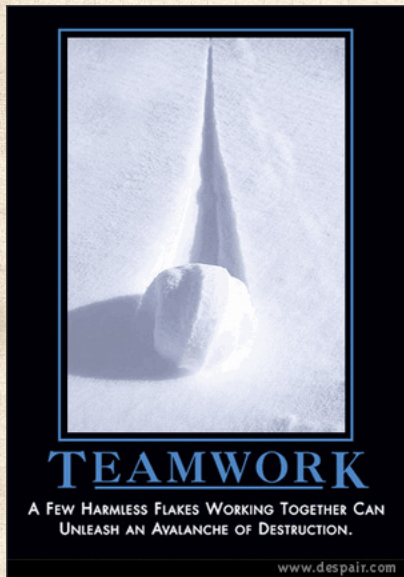


  $\phi = 1/3$  for all nodes





# The power of groups...



[despair.com](http://despair.com)

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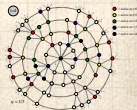
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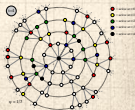
"A few harmless flakes working together can unleash an avalanche of destruction."





"Threshold Models of Social Influence"   
Watts and Dodds,  
The Oxford Handbook of Analytical  
Sociology, **34**, 475–497, 2009. <sup>[29]</sup>

-  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**



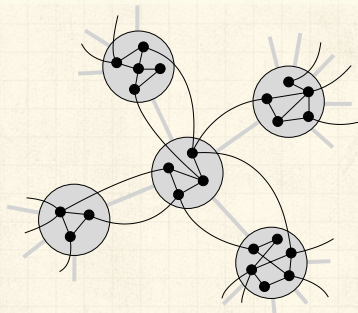
# Group structure—Ramified random networks

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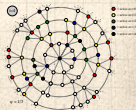
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$p$  = intergroup connection probability  
 $q$  = intragroup connection probability.

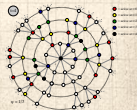
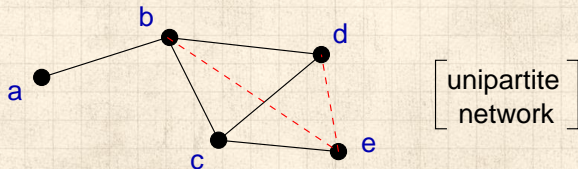
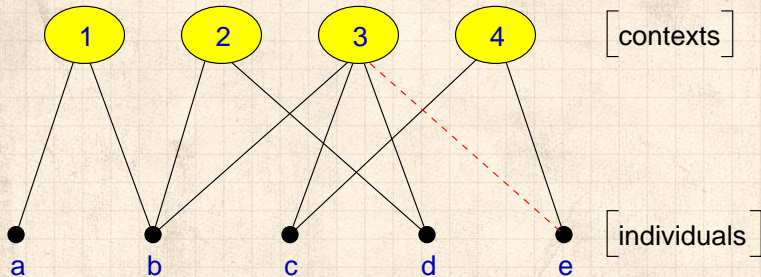


# Bipartite networks

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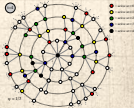
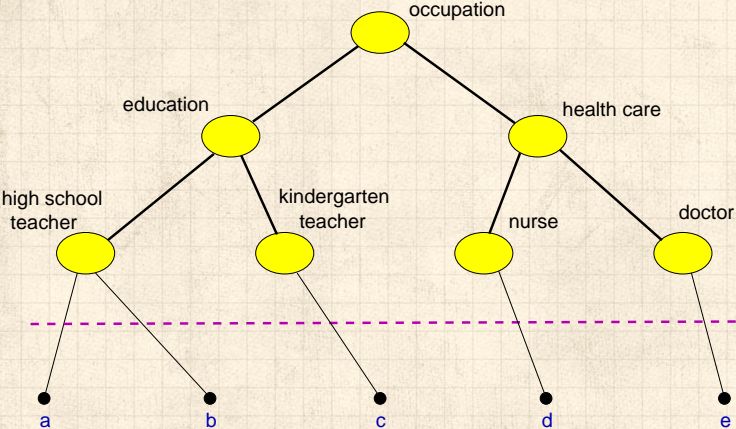


# Context distance

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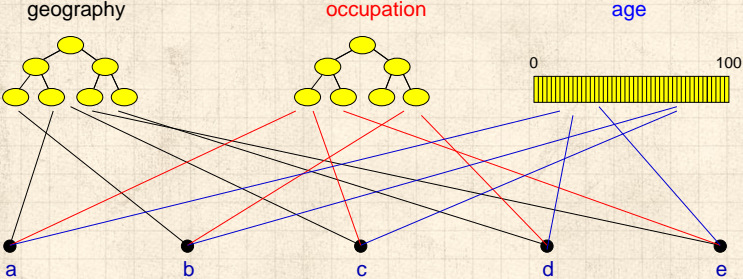


# Generalized affiliation model

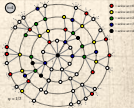
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(Blau & Schwartz, Simmel, Breiger)



# Generalized affiliation model networks with triadic closure



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

where

$\alpha$  = homophily parameter

and

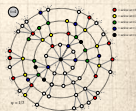
$d$  = distance between nodes (height of lowest common ancestor)



$\tau_1$  = intergroup probability of friend-of-friend connection



$\tau_2$  = intragroup probability of friend-of-friend connection



# Cascade windows for group-based networks

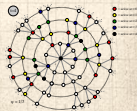
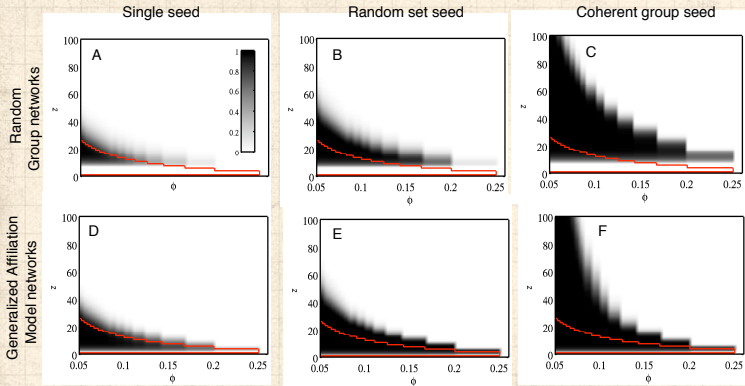
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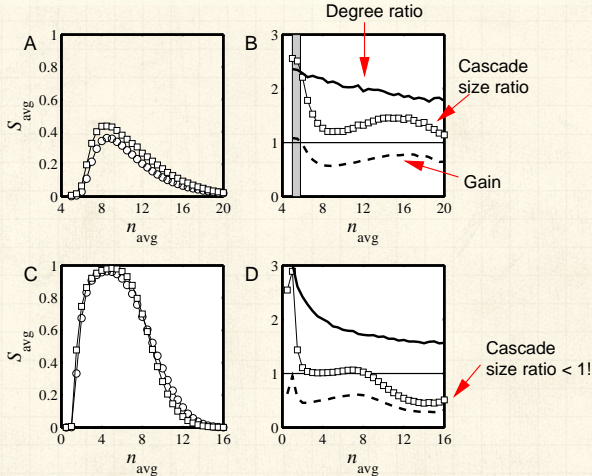


# Multiplier effect for group-based networks:

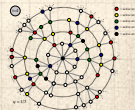
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Multiplier almost always below 1.

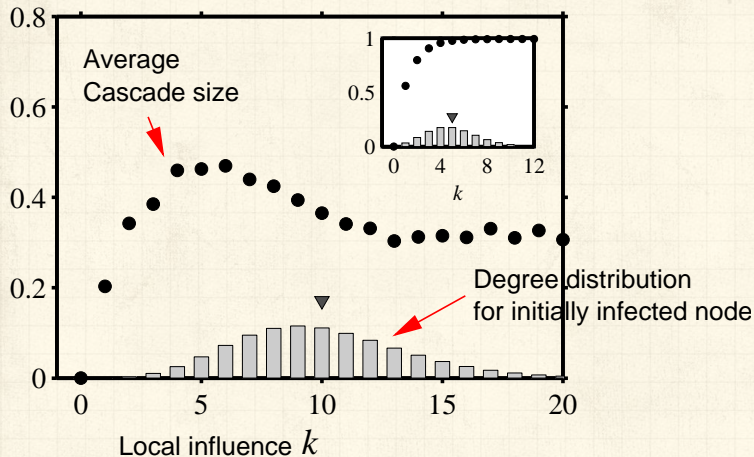



# Assortativity in group-based networks


## Social Contagion Models

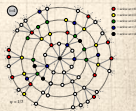
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 The most connected nodes aren't always the most 'influential.'

 **Degree assortativity** is the reason.



# Social contagion

“Without followers, evil cannot spread.” –Leonard Nimoy

## Summary

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

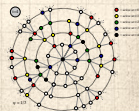
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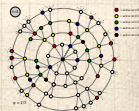
## Implications

- 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 (fashion), or **active** = harder to achieve (political messages; even so: buttons and hats).
- Entities can be novel or designed to combine with others, e.g. block another one.


## Social Contagion Models

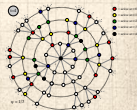
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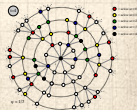




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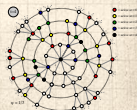
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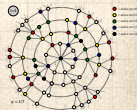
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[BMJ, 337:article #2338, 2008.](#) pdf 

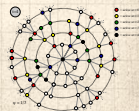
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


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restaurants, residential segregation, and the  
spiral of silence.

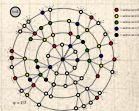
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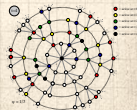
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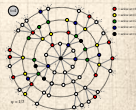
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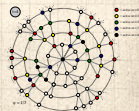
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