Social Contagion
Principles of Complex Systems
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Outline

Social Contagion Models
Background
Granovetter's model
Network version
Groups
Chaos

References
Examples abound
- fashion
- striking
- smoking (hesion)[6]
- residential segregation[15]
- ipods
- obesity (hesion)[5]

SIR and SIRS contagion possible
- Classes of behavior versus specific behavior: dieting

Two focuses for us
- Widespread media influence
- Word-of-mouth influence

Framingham heart study:

Evolving network stories:
- The spread of quitting smoking (hesion)[6]
- The spread of spreading (cession)[5]

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[8] as ‘connectors’
- The infectious idea of opinion leaders (Katz and Lazarsfeld)[12]
Why do things spread?

- Because of system level properties?
- Or properties of special individuals?
- Is the match that lights the fire important?
- Yes. But only because we are narrative-making machines...
- We like to think things happened for reasons...
- System/group properties harder to understand
- Always good to examine what is said before and after the fact...
The Mona Lisa

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

The completely unpredicted fall of Eastern Europe


The dismal predictive powers of editors...

Social Contagion

Messing with social connections

▶ Ads based on message content (e.g., Google and email)
▶ Buzz media
▶ Facebook’s advertising: Beacon (ע)

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Getting others to do things for you


Six modes of influence
1. Reciprocation: *The Old Give and Take... and Take*
2. Commitment and Consistency: *Hobgoblins of the Mind*
3. Social Proof: *Truths Are Us*
4. Liking: *The Friendly Thief*
5. Authority: *Directed Reference*
6. Scarcity: *The Rule of the Few*

Examples

- Reciprocation: Free samples, Hare Krishnas
- Commitment and Consistency: Hazing
- Social Proof: Catherine Genovese, Jonestown
- Liking: Separation into groups is enough to cause problems.
- Authority: Milgram's obedience to authority experiment.
- Scarcity: Prohibition.

Getting others to do things for you

- Cialdini’s modes are heuristics that help 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)\(^{15, 16, 17}\)
  - Simulation on checker boards
  - Idea of thresholds
  - Fun with Netlogo and Schelling’s model\(^{20}\)...
- Threshold models—Granovetter (1978)\(^ {9}\)
- Herding models—Bikhchandani, Hirschleifer, Welch (1992)\(^ {1, 2}\)
  - Social learning theory, Informational cascades,...

Social Contagion

Some possible origins of thresholds:

- Desire to coordinate, to conform.
- 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

Social Contagion

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

Granovetter’s Threshold model—definitions

- \( \phi^* \) = threshold of an individual.
- \( f(\phi^*) \) = distribution of thresholds in a population.
- \( F(\phi^*) = \) cumulative distribution = \( \int_{\phi^*}^{\phi'} f(\phi') d\phi' \)
- \( \phi_t \) = fraction of people ‘rioting’ at time step \( t \).
Threshold models

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

At time \( t + 1 \), fraction rioting = fraction with \( \phi_s \leq \phi_t \).
\[
\phi_{t+1} = \int_0^{\phi_t} f(\phi) d\phi_s = F(\phi_s)|_{0}^{\phi_t} = F(\phi_t)
\]

⇒ Iterative maps of the unit interval \([0, 1]\).

Threshold models

Another example of critical mass model...
Threshold models

Example of single stable state model

Threshold models

Chaotic behavior possible

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

Synchronous update assumption is crucial

Threshold models

Implications for collective action theory:
1. Collective uniformity $\neq$ individual uniformity
2. Small individual changes $\Rightarrow$ large global changes

Threshold model on a network

Many years after Granovetter and Soong's work:

"A simple model of global cascades on random networks"

Mean field model $\rightarrow$ network model

Individuals now have a limited view of the world
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 $a_i \geq \phi_i k_i$
- Individuals remain active when switched (no recovery = SI model)

Snowballing

The Cascade Condition:
If one individual is initially activated, what is the probability that an activation will spread over a network?

What features of a network determine whether a cascade will occur or not?

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
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 neighbors becomes active.

Cascade condition

Back to following a link:
- Link from leads to a node with probability $\propto kP_k$.
- Follows from links being random + having $k$ chances to connect to a node with degree $k$.
- Normalization:
  $$\sum_{k=0}^{\infty} kP_k = \langle k \rangle = z$$
- So
  $$P(\text{linked node has degree } k) = \frac{kP_k}{\langle k \rangle}$$

The most gullible

Vulnerables:
- We call individuals who can be activated by just one contact being active vulnerables
- The vulnerability condition for node $i$:
  $$\frac{1}{k_i} \geq \phi_i$$
- Which means # contacts $k_i \leq \lfloor 1/\phi_i \rfloor$
- For global cascades on random networks, must have a global cluster of vulnerables
- Cluster of vulnerables = critical mass
- Network story: 1 node $\rightarrow$ critical mass $\rightarrow$ everyone.

Cascade condition

Next: Vulnerability of linked node
- Linked node is vulnerable with probability
  $$\beta_k = \int_{\phi_*}^{1/k} f(\phi_*) d\phi_*$$
- 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

Putting things together:

- Expected number of active edges produced by an active edge =
  \[ \sum_{k=1}^{\infty} (k-1) \beta_k \frac{kP_k}{z} + 0 \frac{(1-\beta_k)kP_k}{z} \]

- \[ = \sum_{k=1}^{\infty} (k-1)k\beta_k P_k / z \]

Two special cases:

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

- \[ \beta \sum_{k=1}^{\infty} k(k-1)P_k / z \geq 1. \]

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

- \[ \sum_{k=1}^{\infty} k(k-1)P_k / z \geq 1. \]

Cascade condition

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

\[ \sum_{k=1}^{\infty} k(k-1)\beta_k P_k / z \geq 1. \]

- \( \beta_k = \) probability a degree \( k \) node is vulnerable.

- \( P_k = \) probability a node has degree \( k \).

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—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.”
Early adopters—degree distributions

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

The multiplier effect:

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

Skewed influence distribution example.

Gain

Special subnetworks can act as triggers

$\phi = 1/3$ for all nodes
The power of groups...

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

Group structure—Ramified random networks

\[
p = \text{intergroup connection probability} \\
q = \text{intragroup connection probability.}
\]

Extensions

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

Bipartite networks

\[
\begin{array}{c}
\text{contexts} \\
1 \\
2 \\
3 \\
4 \\
\end{array} \quad \begin{array}{c}
\text{individuals} \\
an \\
b \\
c \\
d \\
e \\
\end{array} \quad \begin{array}{c}
\text{unipartite network} \\
a \\
b \\
c \\
d \\
e \\
\end{array}
\]

\[
\begin{array}{c}
1 \\
2 \\
3 \\
4 \\
\end{array}
\]

\[
\begin{array}{c}
an \\
b \\
c \\
d \\
e \\
\end{array}
\]

\[
\begin{array}{c}
a \\
b \\
c \\
d \\
e \\
\end{array}
\]

\[
\begin{array}{c}
a \\
b \\
c \\
d \\
e \\
\end{array}
\]
Generalized affiliation model networks with triadic closure

- Connect nodes with probability $\propto \exp^{-\alpha d}$
  - $\alpha$ = homophily parameter
  - $d$ = 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

(Blau & Schwartz, Simmel, Breiger)
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.

#### 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.
Chaotic contagion:

- What if individual response functions are not monotonic?
- Consider a simple deterministic version:
  - Node / has an ‘activation threshold’ $\phi_{i,1}$
  - ... and a ‘de-activation threshold’ $\phi_{i,2}$
- Nodes like to imitate but only up to a limit—they don’t want to be like everyone else.

Definition of the tent map:

$$F(x) = \begin{cases} 
  rx & \text{for } 0 \leq x \leq \frac{1}{2}, \\
  r(1 - x) & \text{for } \frac{1}{2} \leq x \leq 1.
\end{cases}$$

- The usual business: look at how $F$ iteratively maps the unit interval $[0, 1]$.

The tent map

Effect of increasing $r$ from 1 to 2.

Orbit diagram:
Chaotic behavior increases as map slope $r$ is increased.
Chaotic behavior

Take \( r = 2 \) case:

- What happens if nodes have limited information?
- As before, allow interactions to take place on a sparse random network.
- Vary average degree \( z = \langle k \rangle \), a measure of information

Invariant densities—stochastic response functions

Invariant densities—deterministic response functions for one specific network with \( \langle k \rangle = 18 \)
Invariant densities—stochastic response functions

Trying out higher values of $\langle k \rangle$...

Connectivity leads to chaos:

Stochastic response functions:

Chaotic behavior in coupled systems

Coupled maps are well explored (Kaneko/Kuramoto):

$$x_{i,n+1} = f(x_{i,n}) + \sum_{j \in \mathcal{N}_i} \delta_{ij} f(x_{j,n})$$

- $\mathcal{N}_i =$ neighborhood of node $i$

1. Node states are continuous
2. Increase $\delta$ and neighborhood size $|\mathcal{N}|$ ⇒ synchronization

But for contagion model:

1. Node states are binary
2. Asynchrony remains as connectivity increases
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