Contagion

Complex Networks, Course 303A, Spring, 2009

Prof. Peter Dodds

Department of Mathematics & Statistics
University of Vermont

Outline

Contagion models

Some large questions concerning network contagion:

1. For a given spreading mechanism on a given network, what's the probability that there will be global spreading?
2. If spreading does take off, how far will it go?
3. How do the details of the network affect the outcome?
4. How do the details of the spreading mechanism affect the outcome?
5. What if the seed is one or many nodes?

- Next up: We'll look at some fundamental kinds of spreading on generalized random networks.

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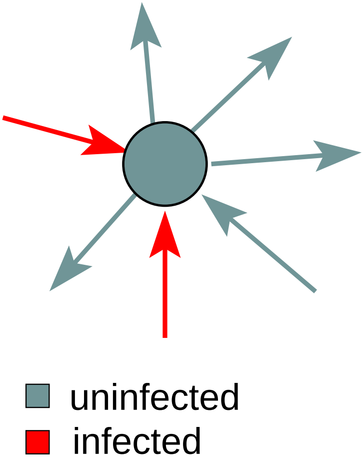

- General spreading mechanism:
  State of node *i* depends on history of *i* and *i*'s neighbors' states.
- Doses of entity may be stochastic and history-dependent.
- May have multiple, interacting entities spreading at once.

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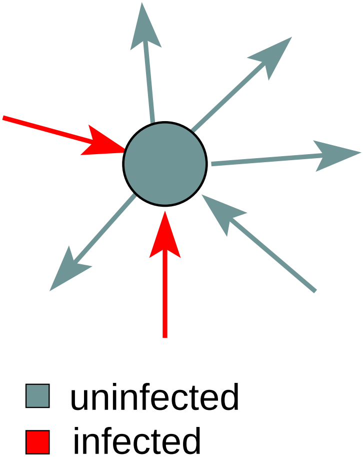

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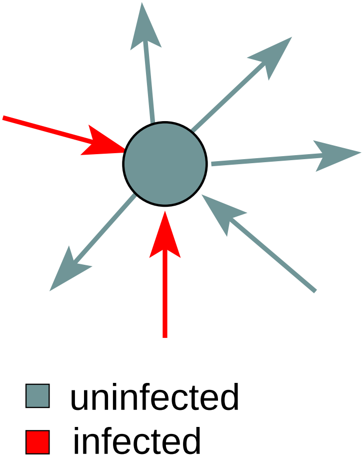

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- For random networks, we know local structure is pure branching.
- Successful spreading is ∴ contingent on single edges infecting nodes.
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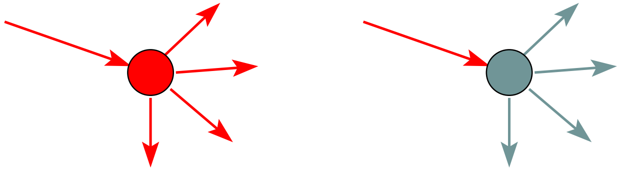

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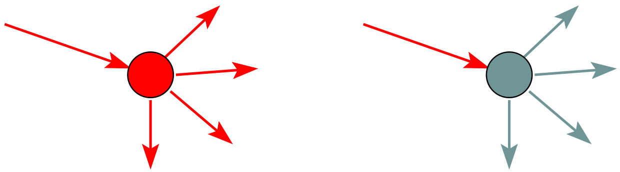

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Contagion condition

- We need to find:
  r = the average # of infected edges that one random infected edge brings about.
- Define β_k as the probability that a node of degree k is infected by a single infected edge.
- $$r = \sum_{k=0}^{\infty} \underbrace{\frac{kP_k}{\langle k \rangle}}_{\substack{\text{prob. of}\\\text{connecting to}\\\text{a degree } k \text{ node}}} \cdot \underbrace{\beta_k}_{\substack{\text{Prob. of}\\\text{infection}}} \cdot \underbrace{(k-1)}_{\substack{\text{\# outgoing}\\\text{infected}\\\text{edges}}}$$

$$+ \sum_{k=0}^{\infty} \overbrace{\frac{kP_k}{\langle k \rangle}} \cdot \underbrace{(1-\beta_k)}_{\substack{\text{Prob. of}\\\text{no infection}}} \cdot \underbrace{0}_{\substack{\text{\# outgoing}\\\text{infected}\\\text{edges}}}$$

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Contagion condition

- Our contagion condition is then:

$$\boxed{r = \sum_{k=0}^{\infty} \frac{(k-1)kP_k}{\langle k \rangle} \beta_k > 1.}$$

- Case 1: If $\beta_k = 1$ then

$$r = \frac{\langle k(k-1) \rangle}{\langle k \rangle} > 1.$$

- Good: This is just our giant component condition again.

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- **Case 2:** If $\beta_k = \beta < 1$ then

$$r = \beta \frac{\langle k(k-1)\rangle}{\langle k\rangle} > 1.$$

- A fraction (1-β) of edges do not transmit infection.
- Analogous phase transition to giant component case but critical value of $\langle k\rangle$ is increased.
- Aka bond percolation.
- Resulting degree distribution P'_k:

$$P'_k = \beta^k \sum_{i=k}^{\infty} \binom{i}{k} (1-\beta)^{i-k} P_i.$$

- We can show $F_{P'}(x) = F_P(\beta x + 1 - \beta)$.

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- Cases 3, 4, 5, ...: Now allow β_k to depend on k
- Asymmetry: Transmission along an edge depends on node's degree at other end.
- Possibility: β_k increases with k... unlikely.
- Possibility: β_k is not monotonic in k... unlikely.
- Possibility: β_k decreases with k... hmmm.
- $\beta_k \searrow$ is a plausible representation of a simple kind of social contagion.
- The story:
  More well connected people are harder to influence.

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- Example: $\beta_k = 1/k$.
- $$r = \sum_{k=1}^{\infty} \frac{(k-1)kP_k}{\langle k \rangle} \beta_k = \sum_{k=1}^{\infty} \frac{(k-1)kP_k}{\langle k \rangle k}$$
$$= \sum_{k=1}^{\infty} \frac{(k-1)P_k}{\langle k \rangle} = \frac{\langle k \rangle - 1}{\langle k \rangle} = 1 - \frac{1}{\langle k \rangle}$$
- Since r is always less than 1, no spreading can occur for this mechanism.
- Decay of β_k is too fast.
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- Example: $\beta_k = 1/k$.
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- Example: $\beta_k = H(\frac{1}{k} - \phi)$
  where $0 < \phi \leq 1$ is a threshold and H is the Heaviside function.
- Infection only occurs for nodes with low degree.
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  they flip when only one of their friends flips.
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- The contagion condition:

$$r = \sum_{k=1}^{\lfloor \frac{1}{\phi} \rfloor} \frac{(k-1)kP_k}{\langle k \rangle} > 1.$$

- As $\phi \to 1$, all nodes become resilient and $r \to 0$.
- As $\phi \to 0$, all nodes become vulnerable and the contagion condition matches up with the giant component condition.
- Key: If we fix ϕ and then vary $\langle k \rangle$, we may see two phase transitions.
- Added to our standard giant component transition, we will see a cut off in spreading as nodes become more connected.

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Outline

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Some important models (recap from CSYS 300)

- Tipping models—Schelling (1971) [8, 9, 10]
  - Simulation on checker boards.
  - Idea of thresholds.
- Threshold models—Granovetter (1978) [7]
- Herding models—Bikhchandani et al. (1992) [1, 2]
  - Social learning theory, Informational cascades,...

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Original work:

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Threshold model on a network

- Interactions between individuals now represented by a network
- Network is sparse
- Individual i has k_i contacts
- Influence on each link is reciprocal and of unit weight
- Each individual i has a fixed threshold ϕ_i
- Individuals repeatedly poll contacts on network
- Synchronous, discrete time updating
- Individual i becomes active when fraction of active contacts $a_i \geq \phi_i k_i$
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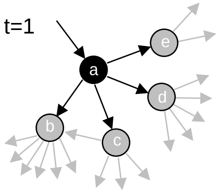

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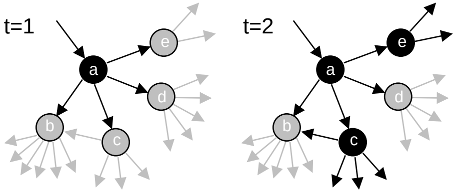

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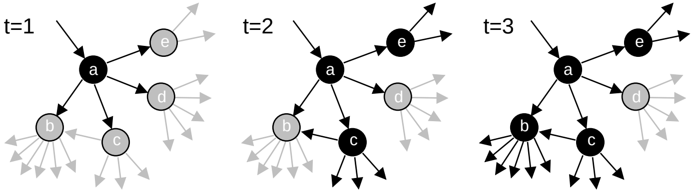

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The most gullible

Vulnerables:

- Recall definition: individuals who can be activated by just one contact being active are vulnerables.
- The vulnerability condition for node i: $1/k_i \geq \phi_i$.
- Means # contacts $k_i \leq \lfloor 1/\phi_i \rfloor$.
- Key: For global cascades on random networks, must have a *global component of vulnerables* [12]
- For a uniform threshold ϕ, our contagion condition tells us when such a component exists:

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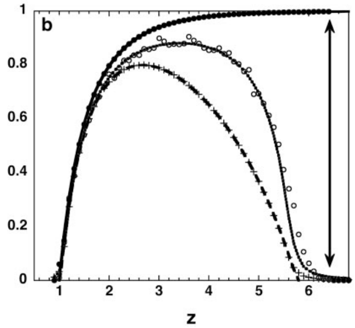

(n.b., $z = \langle k \rangle$)

- Top curve: final fraction infected if successful.
- Middle curve: chance of starting a global spreading event (cascade).
- Bottom curve: fractional size of vulnerable subcomponent.[12]
- Cascades occur only if size of vulnerable subcomponent > 0.
- System is robust-yet-fragile just below upper boundary [3, 4, 11]
- 'Ignorance' facilitates spreading.

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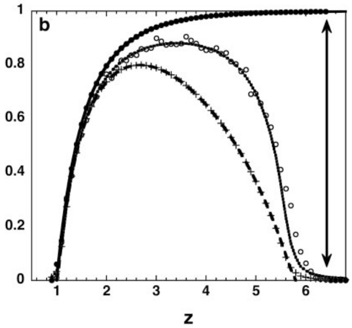

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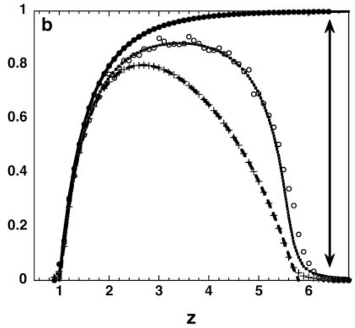

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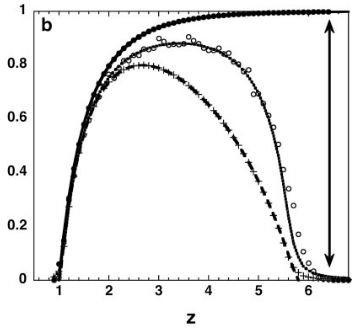

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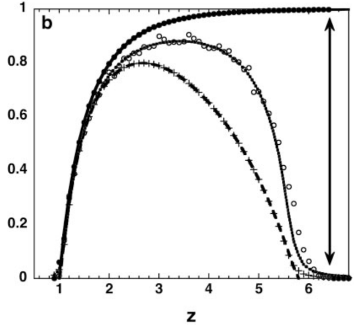

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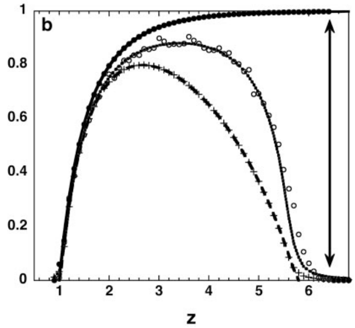

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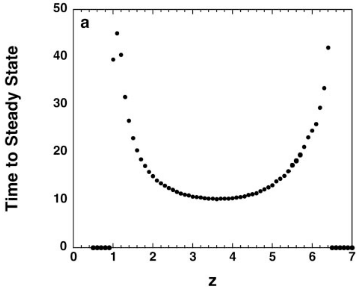

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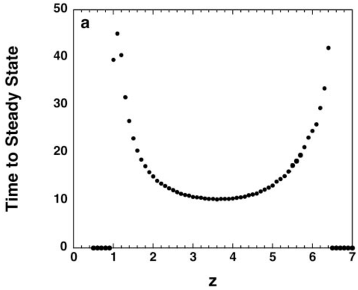

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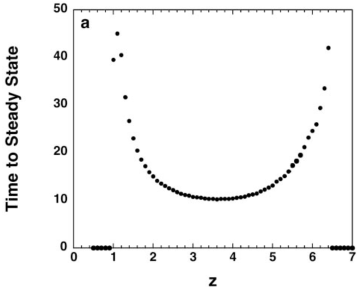

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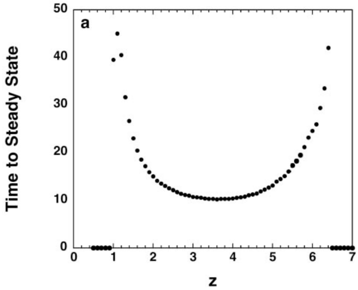

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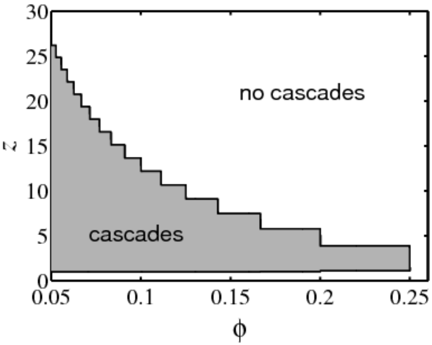
- Now have endogenous mechanism for spreading from an individual to the critical mass and then beyond.

Extreme shepherding:

Non-spontaneous collective sheep phenomena (⊞)

Cascade window for random networks



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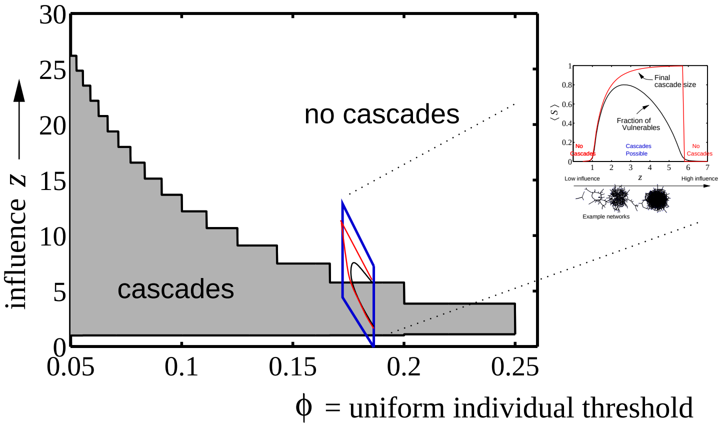
- Outline of cascade window for random networks.

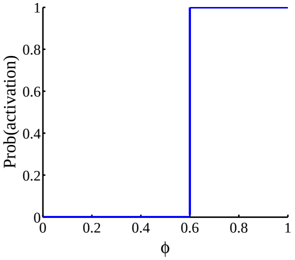
Cascade window for random networks



Outline

Social Contagion

Granovetter's Threshold model—recap



- Assumes deterministic response functions
- ϕ_* = threshold of an individual.
- $f(\phi_*)$ = distribution of thresholds in a population.
- $F(\phi_*)$ = cumulative distribution = $\int_{\phi'_*=0}^{\phi_*} f(\phi'_*)\mathrm{d}\phi'_*$
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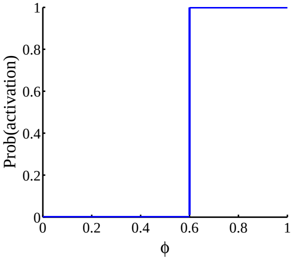

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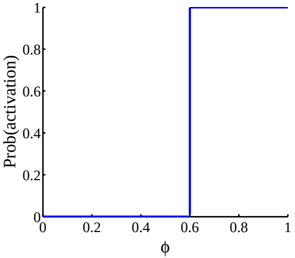

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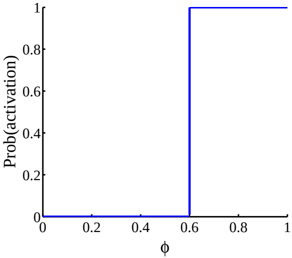

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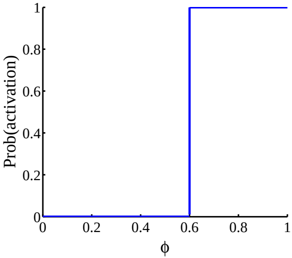

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Social Sciences—Threshold models

- At time $t+1$, fraction rioting = fraction with $\phi_* \leq \phi_t$.
- $$\phi_{t+1} = \int_0^{\phi_t} f(\phi_*)\mathrm{d}\phi_* = F(\phi_*)|_0^{\phi_t} = F(\phi_t)$$
- $\Rightarrow$ Iterative maps of the unit interval $[0, 1]$.

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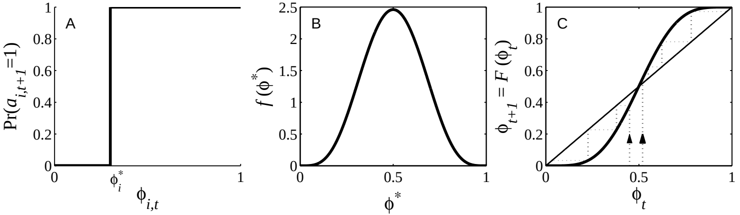
Action based on perceived behavior of others.



- Two states: S and I
- Recover now possible (SIS)
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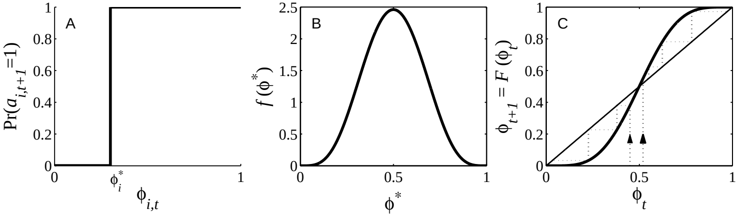

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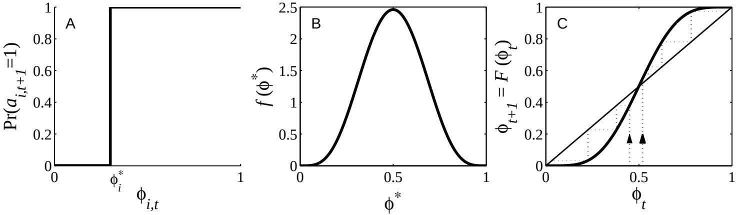

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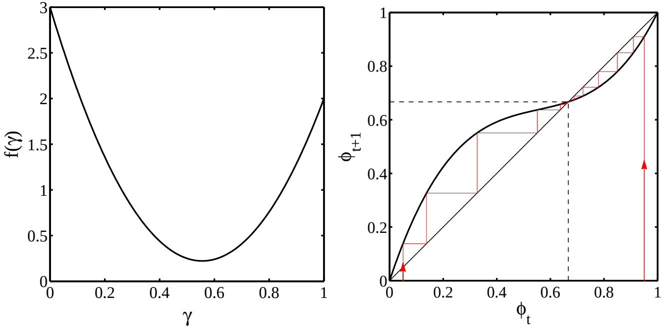

- Example of single stable state model

Social Sciences—Threshold models

Implications for collective action theory:

1. Collective uniformity $\not\Rightarrow$ individual uniformity
2. Small individual changes $\Rightarrow$ large global changes

Next:

- Connect mean-field model to network model.
- Single seed for network model: $1/N \to 0$.
- Comparison between network and mean-field model sensible for vanishing seed size for the latter.

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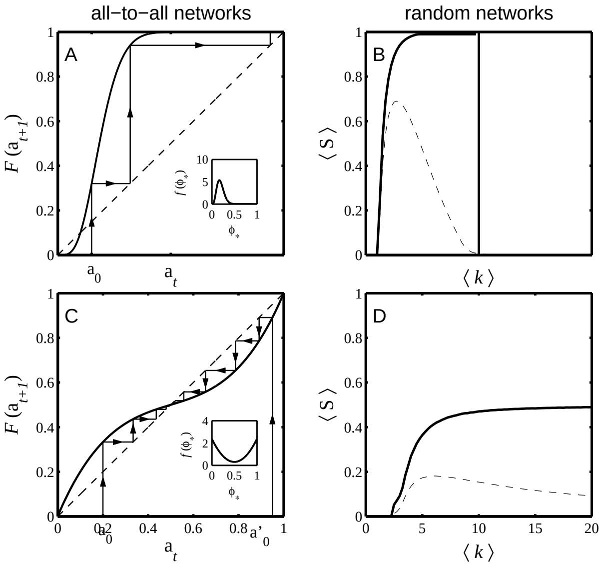
All-to-all versus random networks



Outline

Threshold contagion on random networks

Three key pieces to describe analytically:

1. The fractional size of the largest subcomponent of vulnerable nodes, S_{vuln}.
2. The chance of starting a global spreading event, $P_{\text{trig}} = S_{\text{trig}}$.
3. The expected final size of any successful spread, S.
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- First goal: Find the largest component of vulnerable nodes.
- Recall that for finding the giant component's size, we had to solve:

$$F_\pi(x) = xF_P\left(F_\rho(x)\right) \text{ and } F_\rho(x) = xF_R\left(F_\rho(x)\right)$$

- We'll find a similar result for the subset of nodes that are vulnerable.
- This is a node-based percolation problem.
- For a general monotonic threshold distribution $f(\phi)$, a degree k node is vulnerable with probability

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- Generating function for friends-of-friends distribution is related in same way as before:

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- Functional relations for component size g.f.'s are almost the same...

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- Assumption is first node is randomly chosen.
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- Now think about what must happen for a specific node *i* to become active at time *t*:
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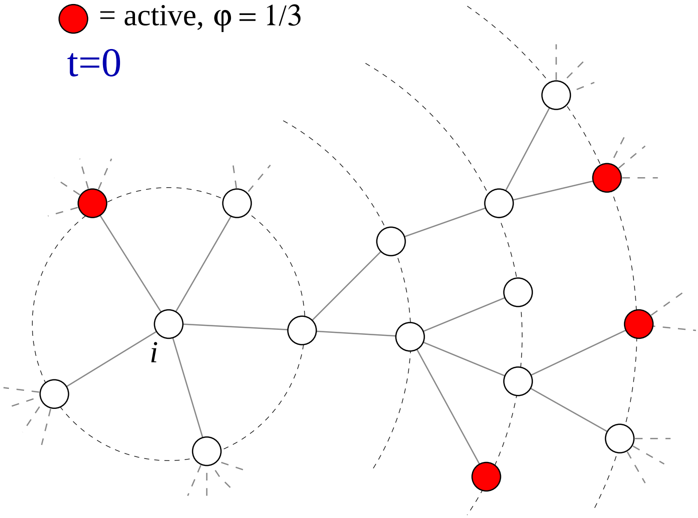

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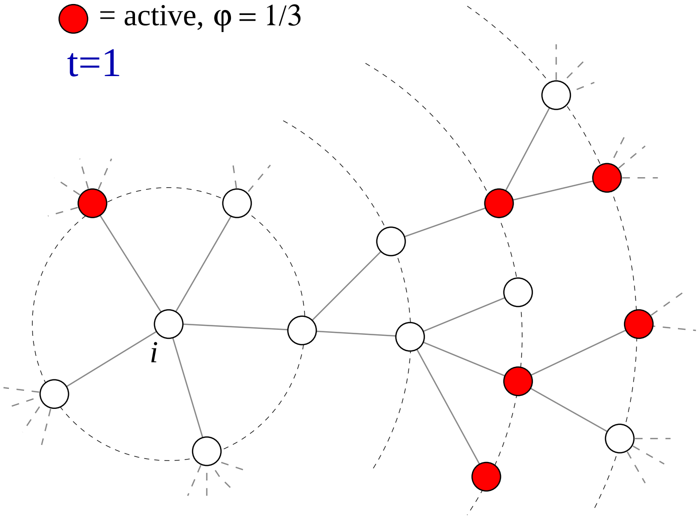

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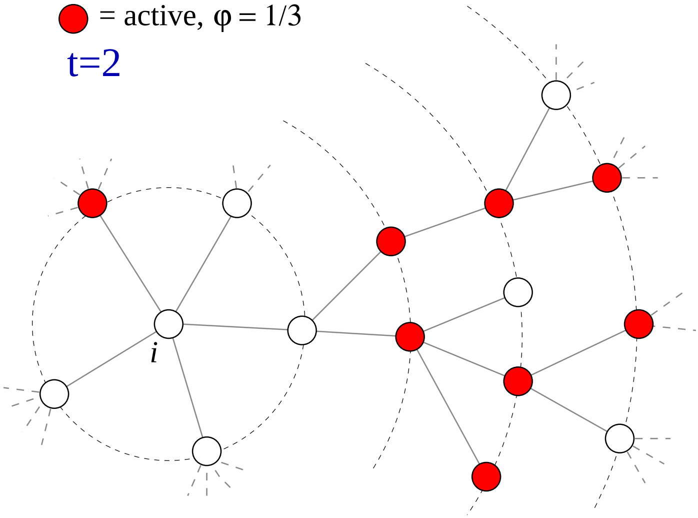

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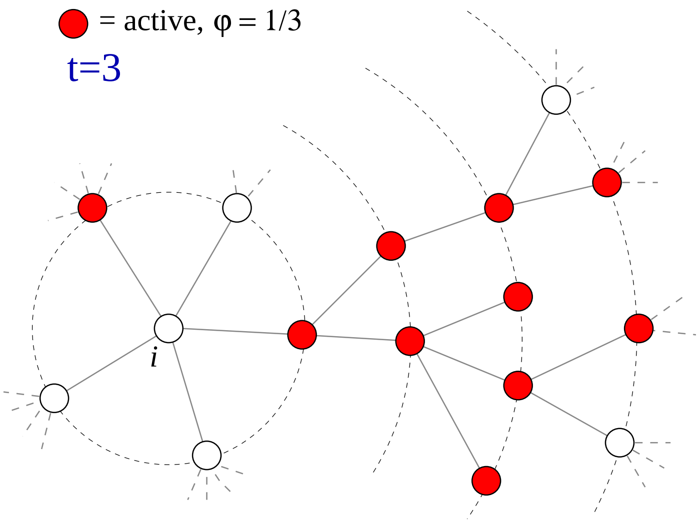

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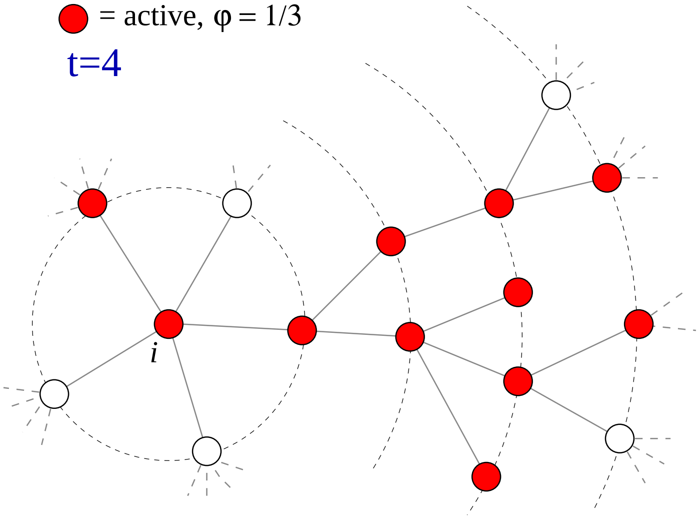

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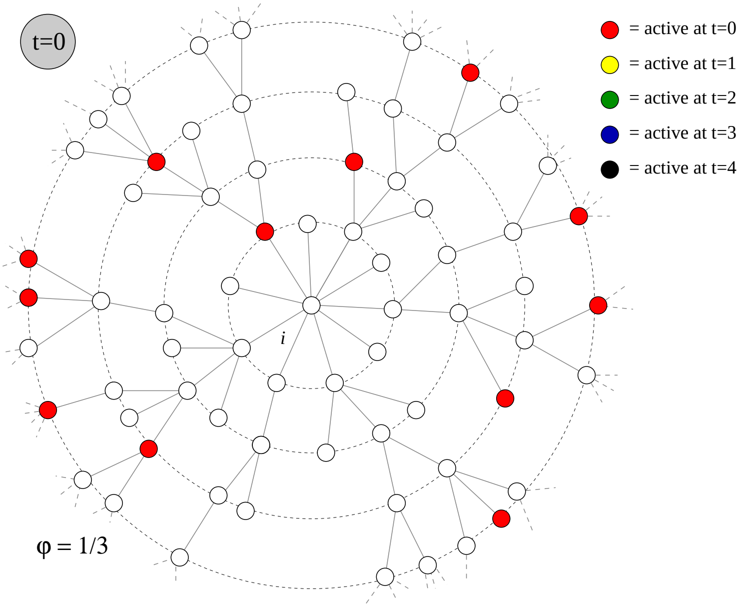

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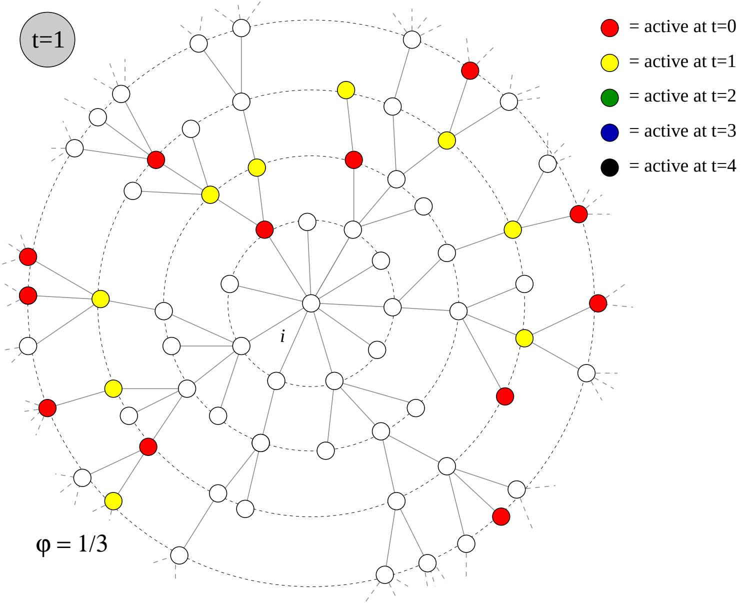

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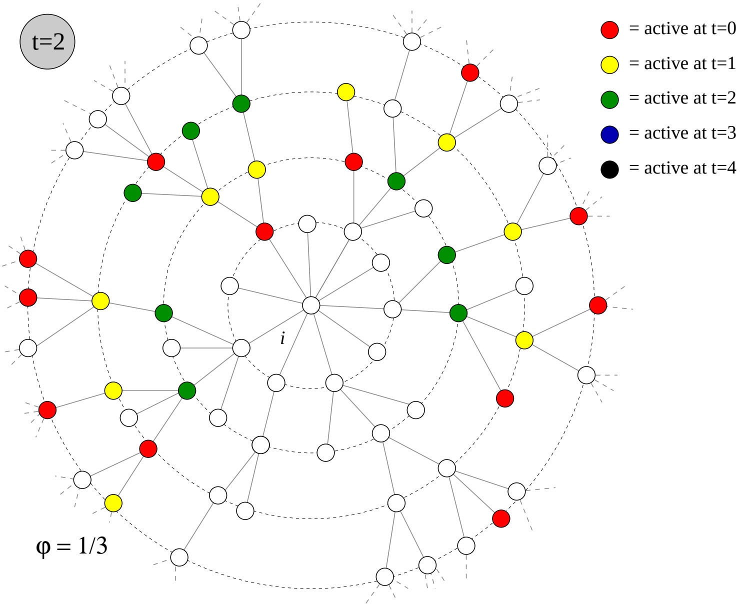

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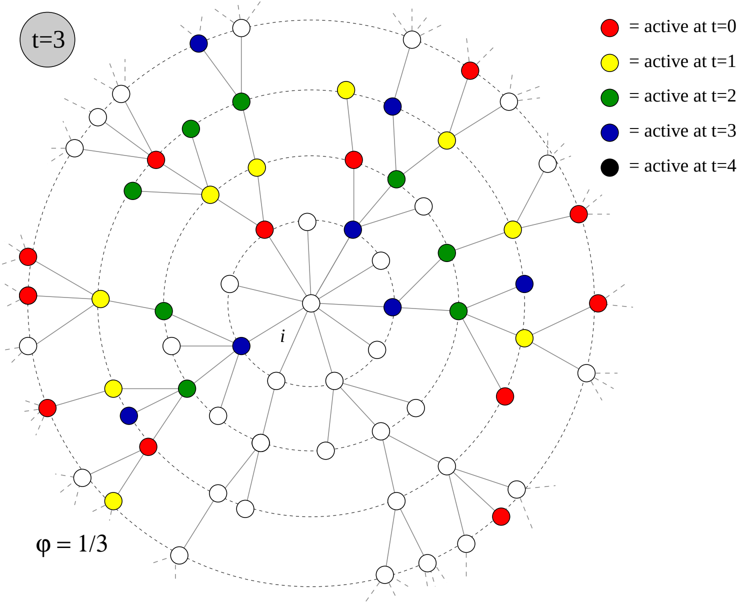

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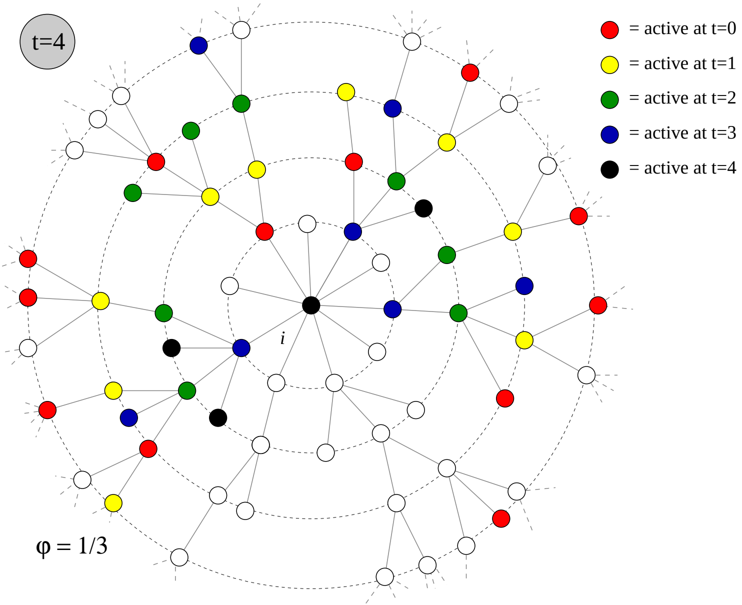

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- We can analytically determine the entire time evolution, not just the final size.
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- Story analogous to $t = 1$ case:

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First connect θ_0 to θ_1:

- $\theta_1 = \phi_0 +$

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Two pieces:

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2. $\phi_{t+1} = \phi_0 +$

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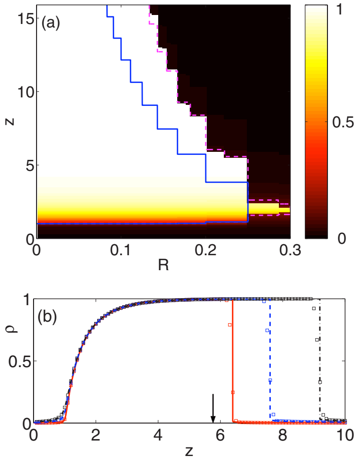
Comparison between theory and simulations



From Gleeson and Cahalane [6]

- Pure random networks with simple threshold responses
- R = uniform threshold (our ϕ_*); z = average degree; $\rho = \phi$; $q = \theta$; $N = 10^5$.
- $\phi_0 = 10^{-3}$, 0.5×10^{-2}, and 10^{-2}.
- Cascade window is for $\phi = 10^{-2}$ case.
- Sensible expansion of cascade window as ϕ_0 increases.

Notes:

- 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:

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

meaning $\beta_{k0} > 0$ 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

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In words:

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

Non-vanishing seed case:

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

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- If G has a stable fixed point at $\theta = 0$, and an unstable fixed point for some $0 < \theta_* < 1$, then for $\theta_0 > \theta_*$, spreading takes off.
- Tricky point: G depends on ϕ_0, so as we change ϕ_0, we also change G.

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In words:

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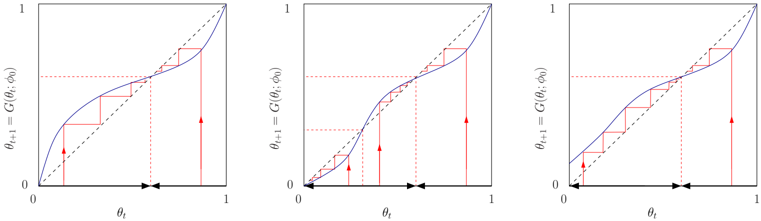

- Given $\theta_0 (= \phi_0)$, θ_∞ will be the nearest stable fixed point, either above or below.
- n.b., adjacent fixed points must have opposite stability types.
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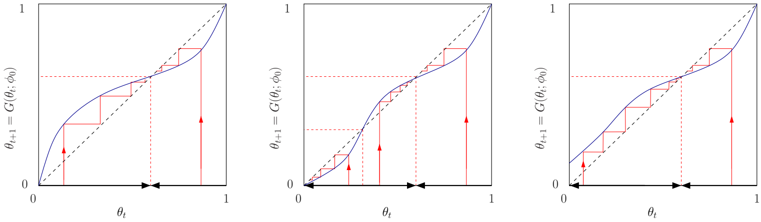

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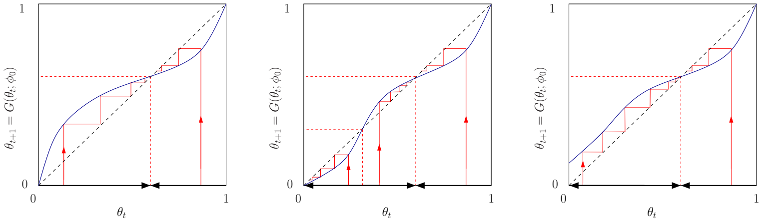

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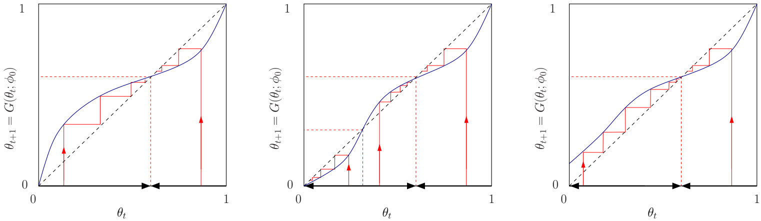

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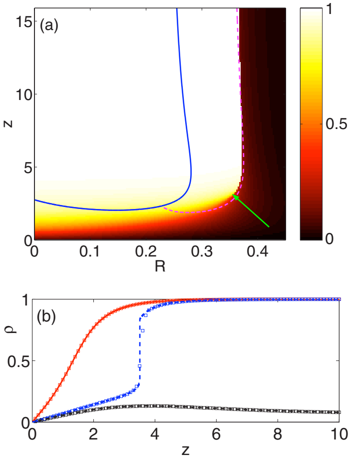
Comparison between theory and simulations



From Gleeson and Cahalane [6]

- Now allow thresholds to be distributed according to a Gaussian with mean R.
- R = 0.2, 0.362, and 0.38; $\sigma = 0.2$.
- $\phi_0 = 0$ but some nodes have thresholds ≤ 0 so effectively $\phi_0 > 0$.
- Now see a (nasty) discontinuous phase transition for low $\langle k \rangle$.

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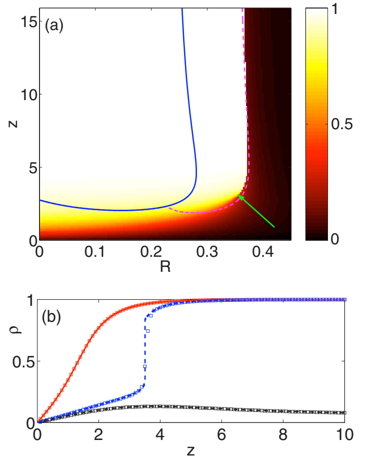

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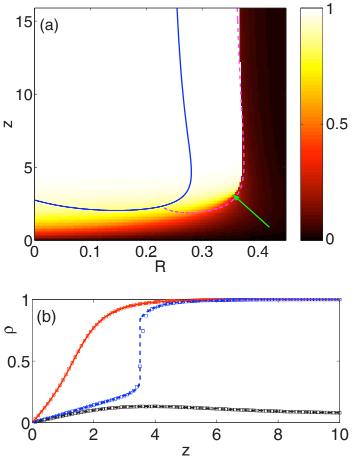

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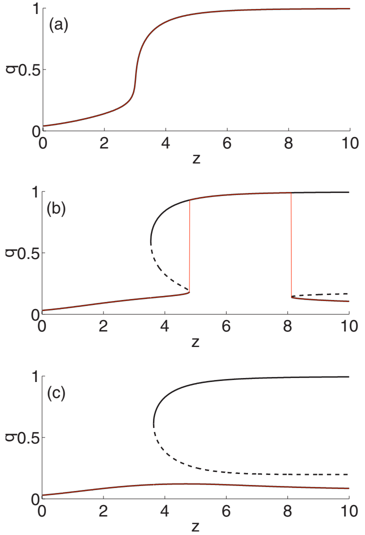

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- Plots of stability points for $\theta_{t+1} = G(\theta_t; \phi_0)$.
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- Top to bottom: R = 0.35, 0.371, and 0.375.
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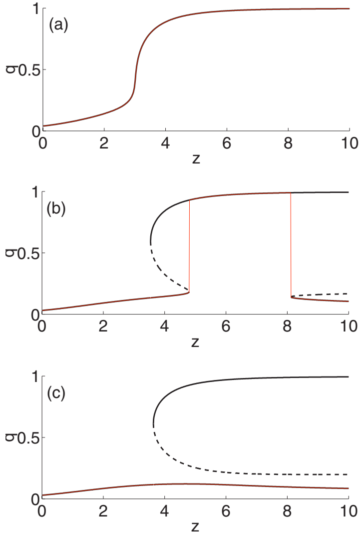

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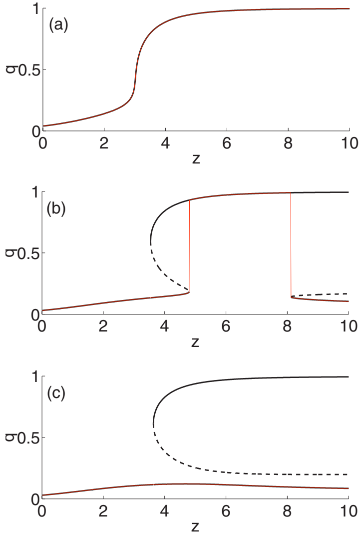

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Spreadarama

Bridging to single seed case:

- Consider largest vulnerable component as initial set of seeds.
- Not quite right as spreading must move through vulnerables.
- But we can usefully think of the vulnerable component as activating at time $t = 0$ because order doesn't matter.
- Rebuild ϕ_t and θ_t expressions...

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Two pieces modified for single seed:

1. $\theta_{t+1} = \theta_{\text{vuln}} +$

$$(1-\theta_{\text{vuln}})\sum_{k=1}^{\infty}\frac{kP_k}{\langle k\rangle}\sum_{j=0}^{k-1}\binom{k-1}{j}\theta_t^{\,j}(1-\theta_t)^{k-1-j}\beta_{kj}$$

   with $\theta_0 = \theta_{\text{vuln}}$ = **Pr** an edge leads to the giant vulnerable component (if it exists).

2. $\phi_{t+1} = S_{\text{vuln}} +$

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Time-dependent solutions

Synchronous update

- Done: Evolution of ϕ_t and θ_t given exactly by the maps we have derived.

Asynchronous updates

- Update nodes with probability α.
- As $\alpha \rightarrow 0$, updates become effectively independent.
- Now can talk about $\phi(t)$ and $\theta(t)$.
- More on this later...

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