Social and Information Networks

University of Toronto CSC303
Winter/Spring 2024

Week 8: Mar 4-8

This week's high-level learning goals

- Explain the initial adopter problem, and recall the conditions seen in class for efficient approximation
  - Define the linear threshold model
  - Define the independent cascade model
  - Prove these models meet the required conditions for efficient approximation
- Define and execute a simple collective action model, and explain how it illustrates the impact of local vs. global knowledge
- Define and execute the wave propagation process for competitive influence spread
- Model disease contagion by determining the relevant properties of a disease
  - Define the basic reproductive number (R_0)
  - Define, execute, and calculate probabilities under the SIR, SIS, and SIRS models
    - ⋆ Explain the interpretation of the SIR model as percolation
    - ⋆ Explain why the graph structure of contacts is important, even given R_0
  - Modify SIR/SIS/SIRS to reflect transient contacts

Key terms & concepts

- Choosing influential adopters
  - Linear threshold model
  - Independent cascade model
- Local vs. global knowledge
- Competitive influence spread
- Modelling disease contagion
  - Basic reproductive number (R_0)
  - SIR model
    - ★ SIR as percolation
    - ★ Structure vs. R_0
    - ★ SIS & SIRS models
  - Modelling transient contacts

Choosing influential adopters

- Suppose we wish to spread a new technology and to do so we have money to influence some "small" set of initial adopters (e.g. by giving away the product or even paying people to adopt it).

- Even in this simple model of (non-competitive) influence spread, and even if we have complete knowledge of the social network, it is not at all clear how to chose an initial set of adopters so as to achieve the largest spread.

- Furthermore the spread process could be much more sophisticated.
  - For example, adoption by a node might be a more random process (say adopting with some probability relative to the nodes threshold) and maybe the influence of neighbors first increases and then decreases over time. And maybe u can have a negative influence on v in say signed networks.

Choosing influential adopters continued

- Suppose we have funds/ability to influence k nodes to become initial adopters.
  - We can try all possible subsets of the entire $n = |V|$ nodes and for each such subset simulate the spread process.
  - But clearly as k gets larger, this "brute force" becomes prohibitive for large (and not even massive) networks.

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  - We can try all possible subsets of the entire $n = |V|$ nodes and for each such subset simulate the spread process.
  - But clearly as k gets larger, this "brute force" becomes prohibitive for large (and not even massive) networks.
- It turns out that the problem of the optimum set of initial adopters in many settings is an NP-hard problem.

Can we determine a "good" set of initial adopters?

- For even simple models of information spread similar to those discussed last week, it can be computationally difficult (NP-Hard) to obtain an approximation within a factor n^c for any $c < 1$.

- Instead we will identify properties of a spread process that will allow a good approximation: a good set of initial adopters that will do "almost as well" as the best set.

Note: What follows is a discussion as to how to choose a set of initial adopters by a relatively efficient approximation algorithm when making some assumptions on the spread process. However, we would need much more efficient methods for massive networks.

Influence maximization models; monotone submodular set functions

- Some spread models have the following nice properties.

For any initial set of adopters, S, let $f(S)$ be size (or more generally a real value benefit since some nodes may be more valuable) of the final set of adopters. Furthermore, let f satisfy:

1. Monotonicity: $f(S) \leq f(T)$ if S is a subset of T
2. Submodularity: $f(S+v) - f(S) \geq f(T+v) - f(T)$ if S is a subset of T

- We also usually assume that $f(\varnothing) = 0$. Such normalized, monotone, submodular functions arise in many applications.
- The simple threshold examples considered thus far are monotone processes but are not submodular in general. Are these contrived worst case network examples?
- But some variants of the threshold model and related models do satisfy these properties. We consider two such stochastic models.

Linear threshold model

- We have an edge weighted (undirected or directed) network where weight $w(u, v)$ represents the relative influence of node u on node v (e.g., a quantitative version of weak and strong ties and possibly also dependent on the "reputation" of node u).

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- Now each nodes threshold $q(v)$ is chosen randomly in $[0, 1]$ to model lack of knowledge as to how easy it is to influence a given individual.
- A node v adopts A if the sum of all edge weights into v (from nodes using A) exceeds the randomly chosen $q(v)$.

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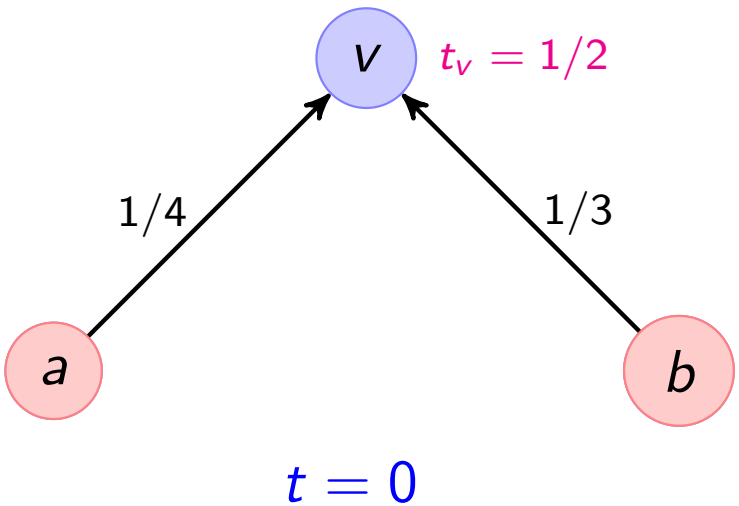
- A node v adopts A if the sum of all edge weights into v (from nodes using A) exceeds the randomly chosen $q(v)$.
- **Goal:** find an initial set of k adopters so as to maximize the expected number (or benefit) of eventual adopters. (This is a stochastic process so that we are trying to optimize the expected value of the process.) At time $t = 0$, only the initial adopters are influenced.
- **Aside:** We often use the language of disease spread and say "infected nodes" rather than "already influenced nodes".

The linear threshold model continued

- Each node v chooses a threshold t_v randomly from $[0, 1]$.
- Each edge (u, v) has assigned weight w_{uv} from $[0, 1]$ such that for any fixed node v:

$$\sum_{u \to v} w_{uv} \leq 1.$$

- In each step t, a node v is infected if the weighted sum of incident edges coming from infected neighbors exceeds threshold.



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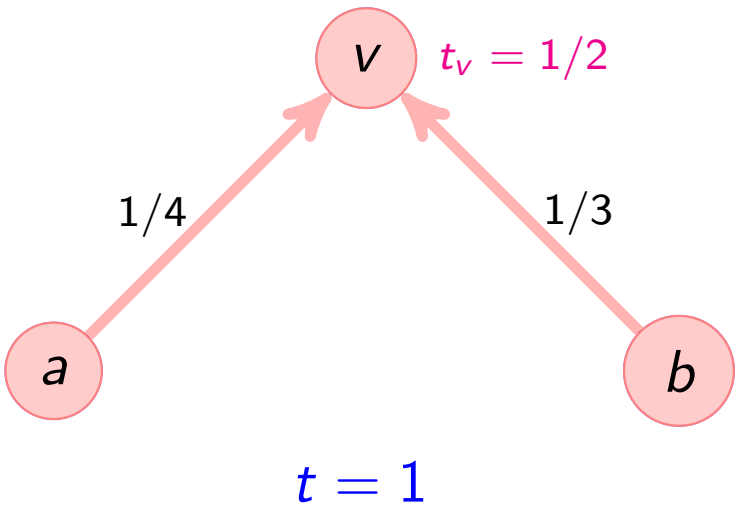

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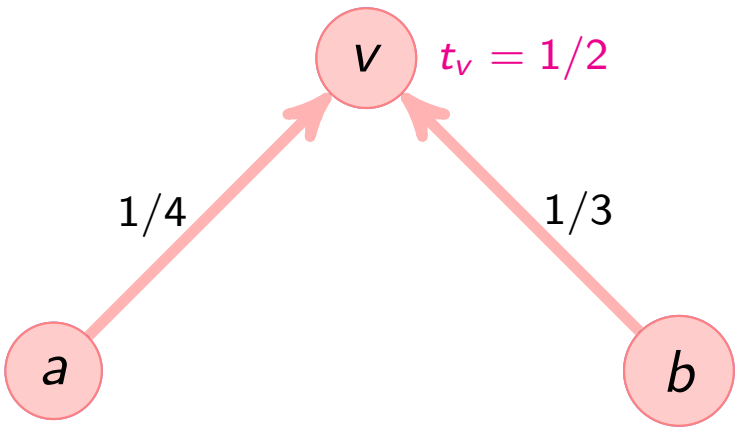

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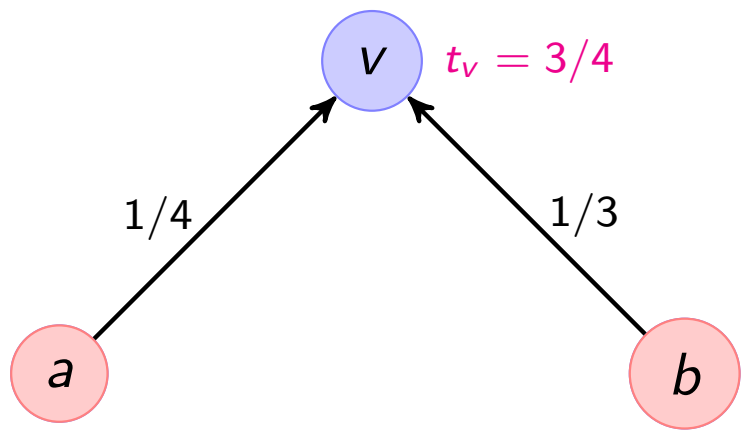

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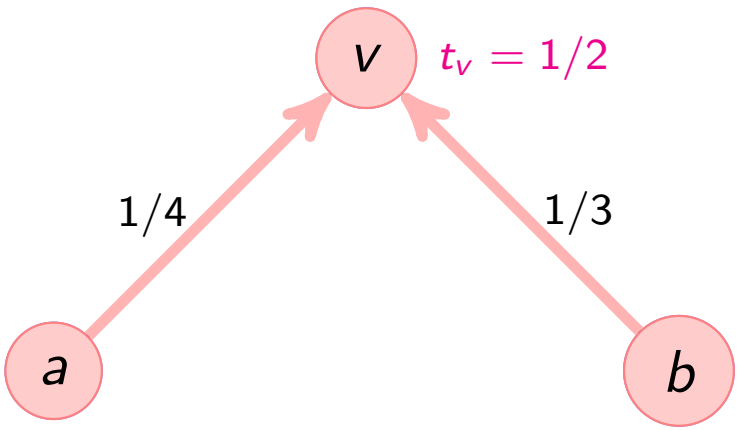

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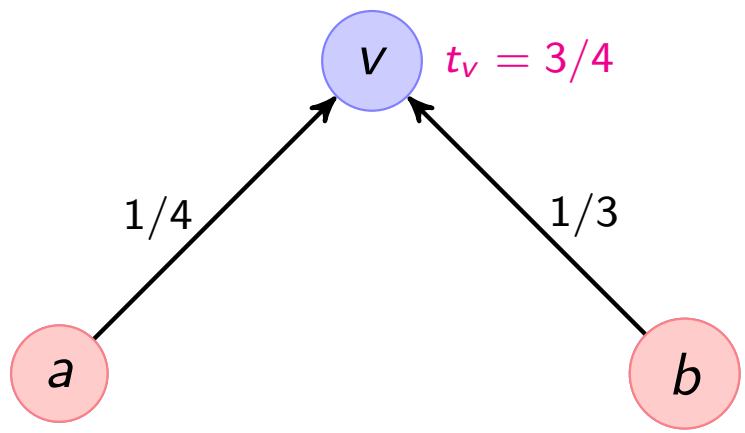

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Independent cascade influence model

- We again have an edge weighted network (as in threshold model) but now the weights $p(u, v) \leq 1$ represent the probability that node u will influence node v given one and only one chance to do so.

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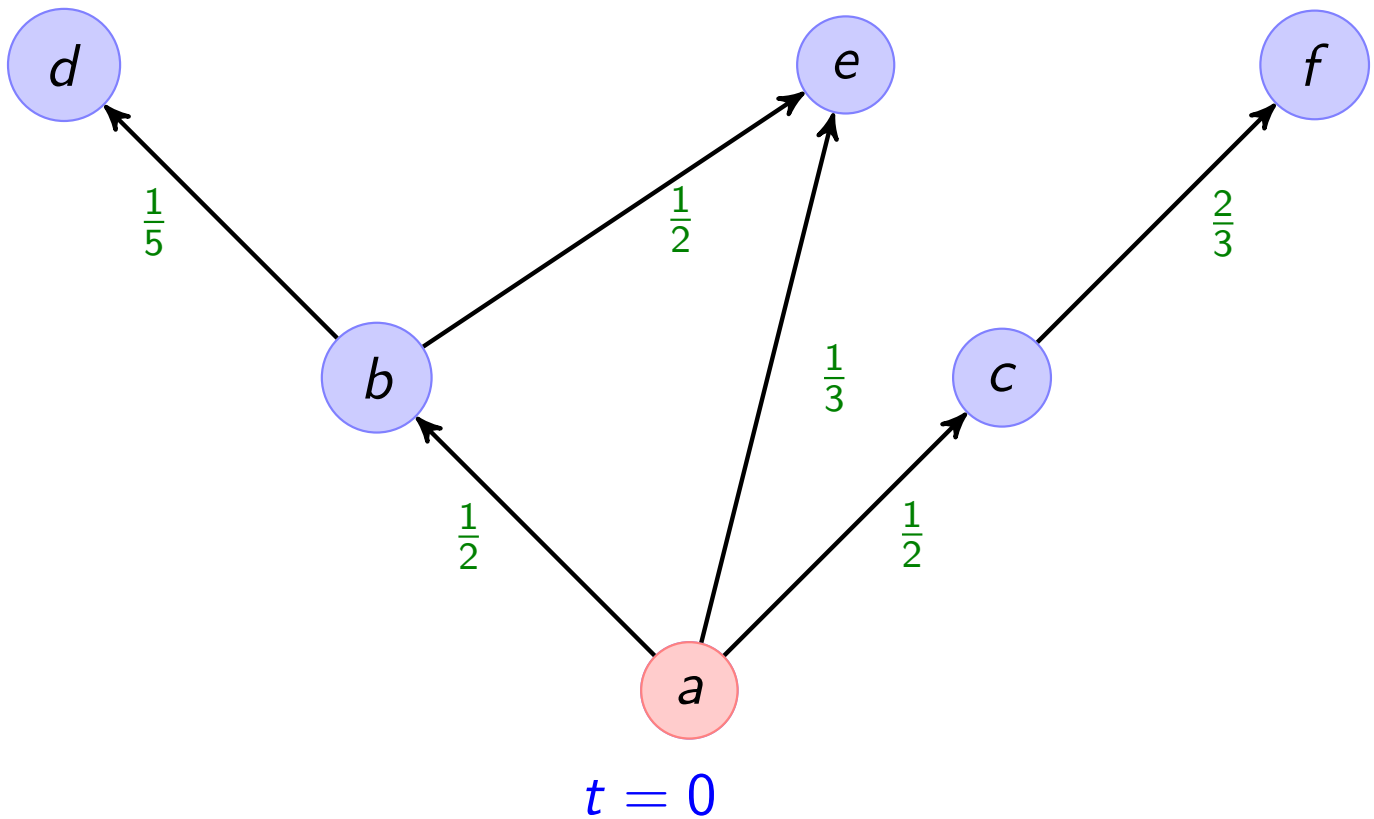
- After this, node u will *not* have another opportunity to influence v.
- **Goal for both threshold and cascade models:** to find initial set of adopters to maximize the expected number of eventual adopters.

The Independent Cascade Process

- Each edge (u, v) has an associated probability p_{uv}.
- In each step t, nodes that adopted technology at step $t-1$ "infect" each of their uninfected neighbors independently with probability p_{uv}.



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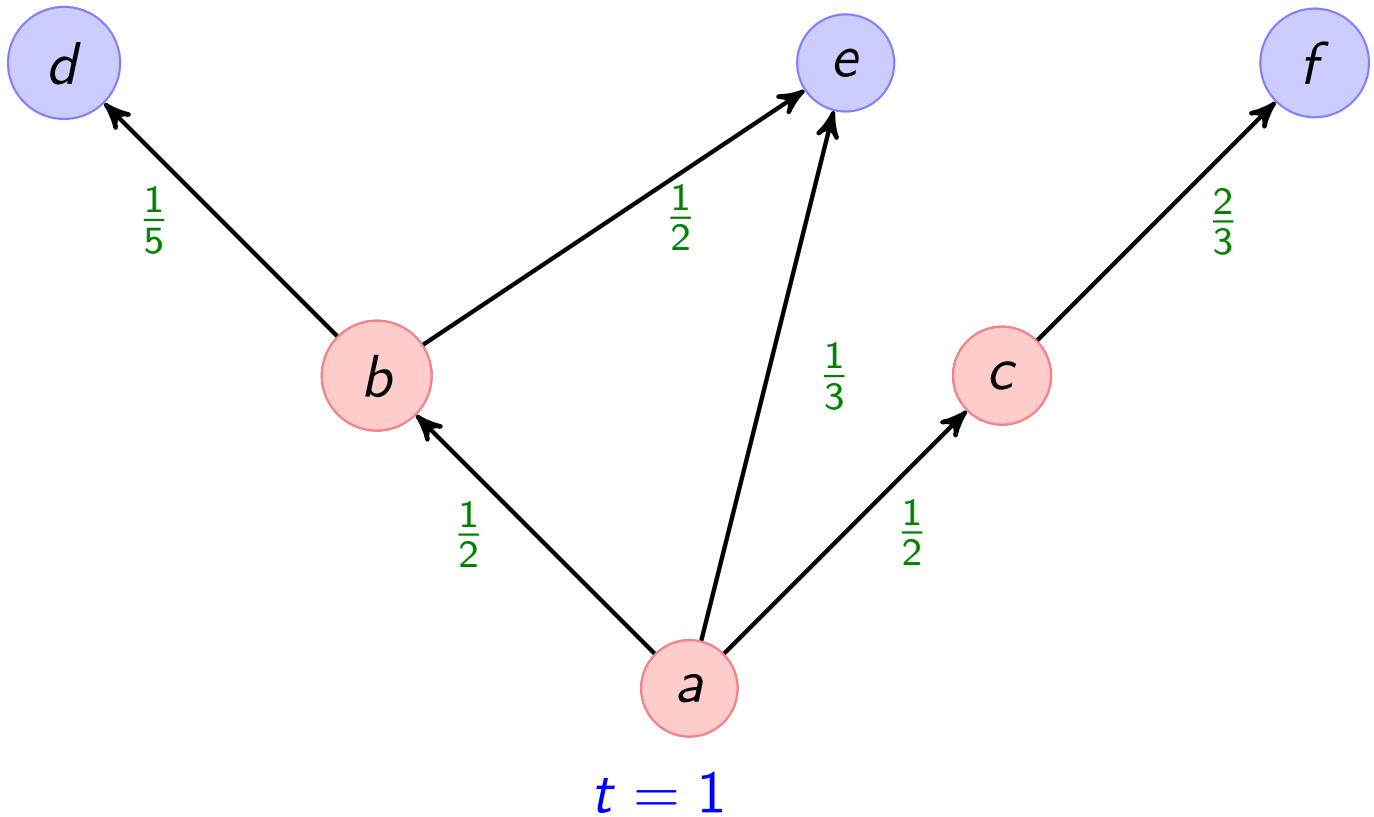

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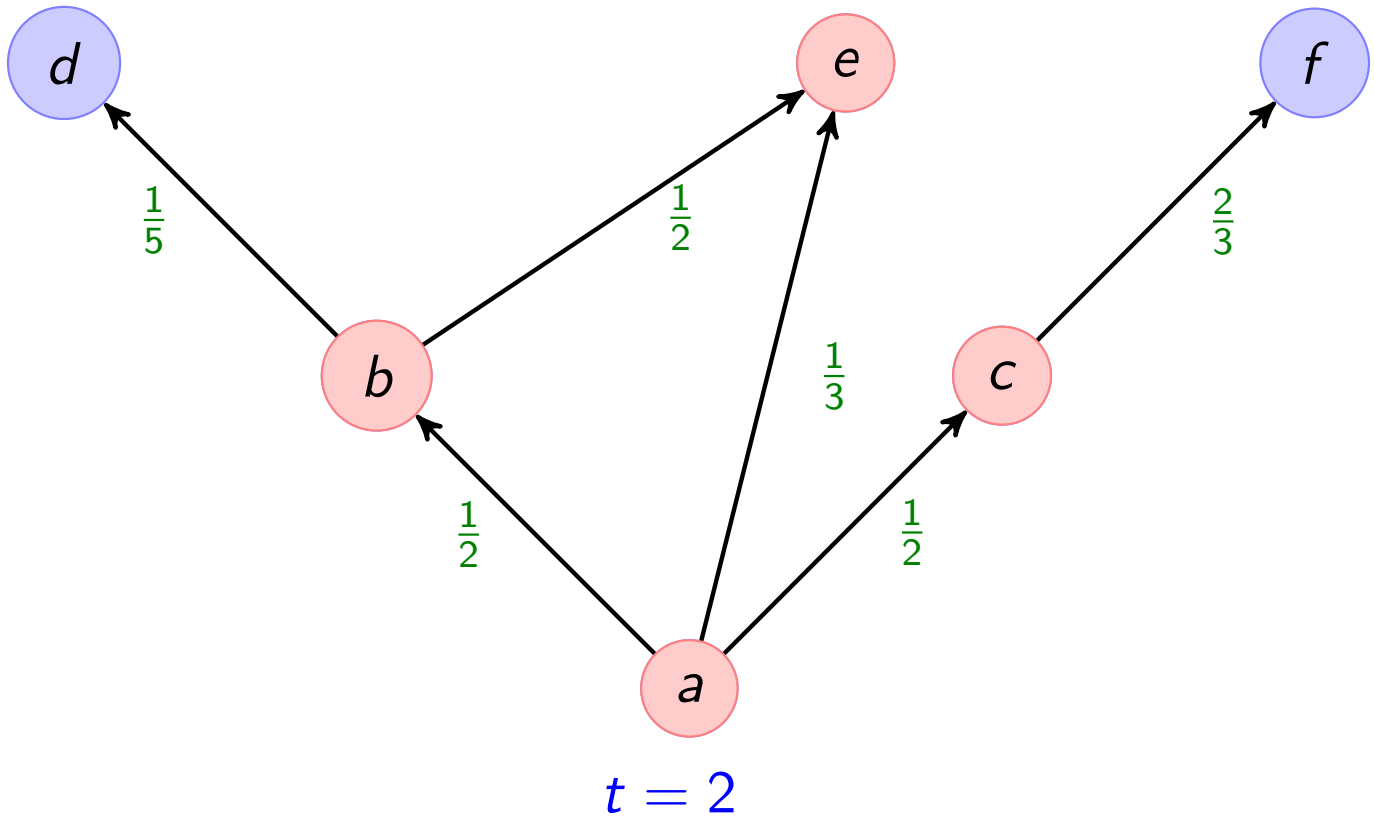

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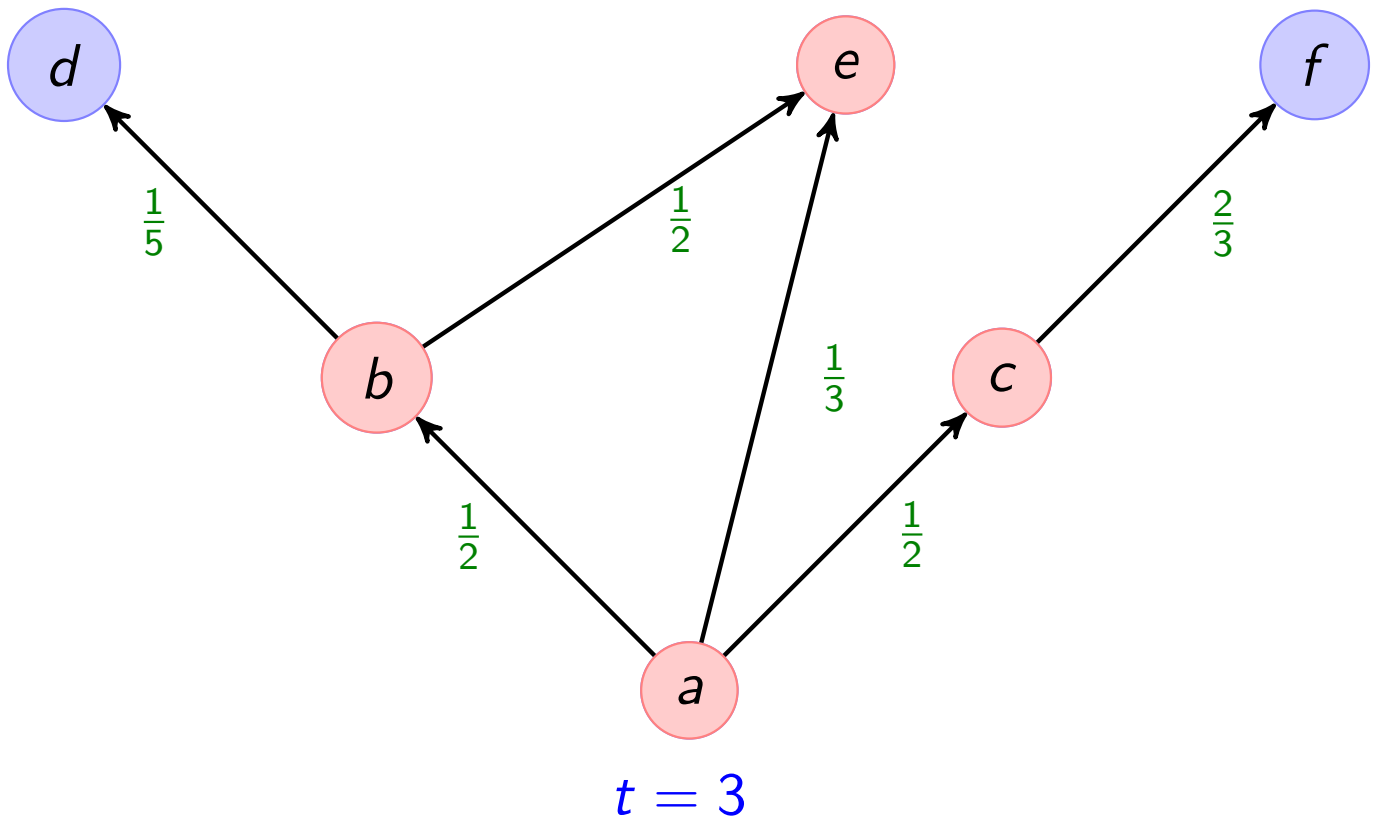

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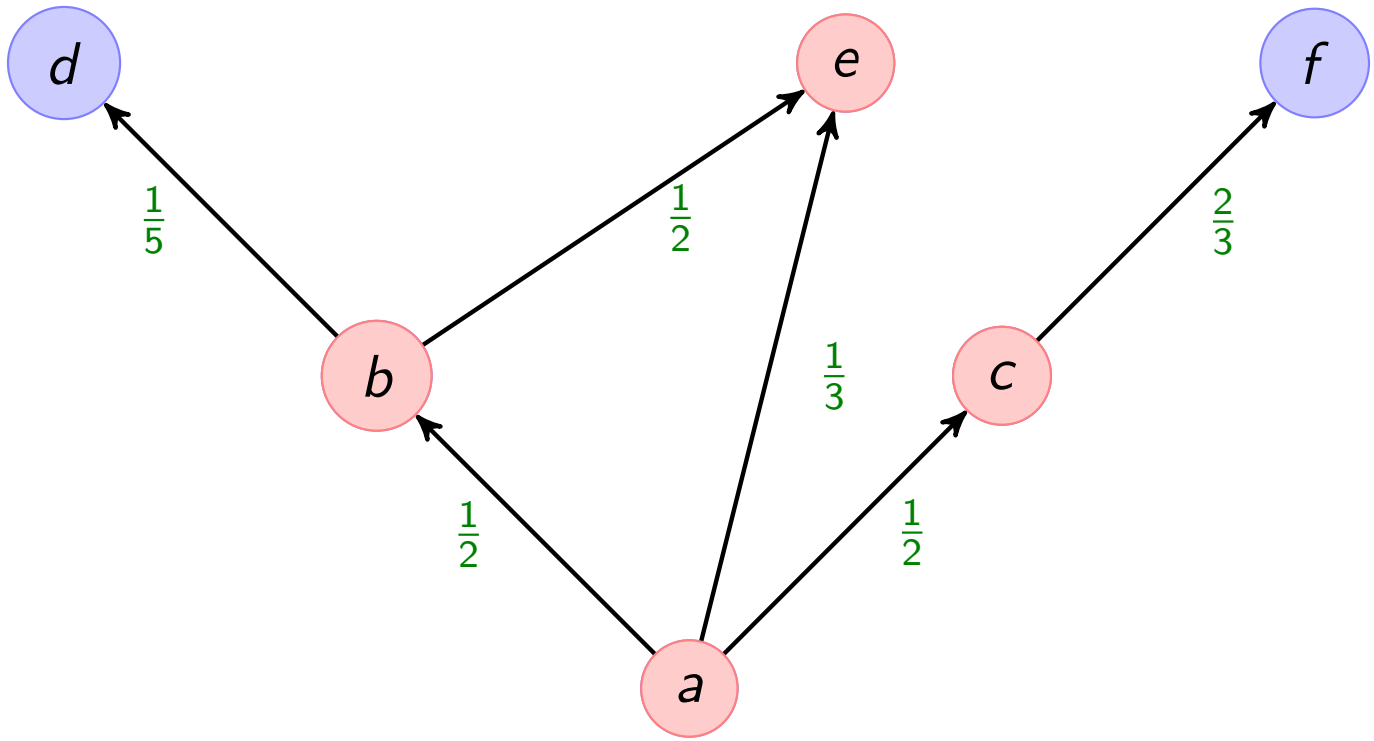

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Look familiar?

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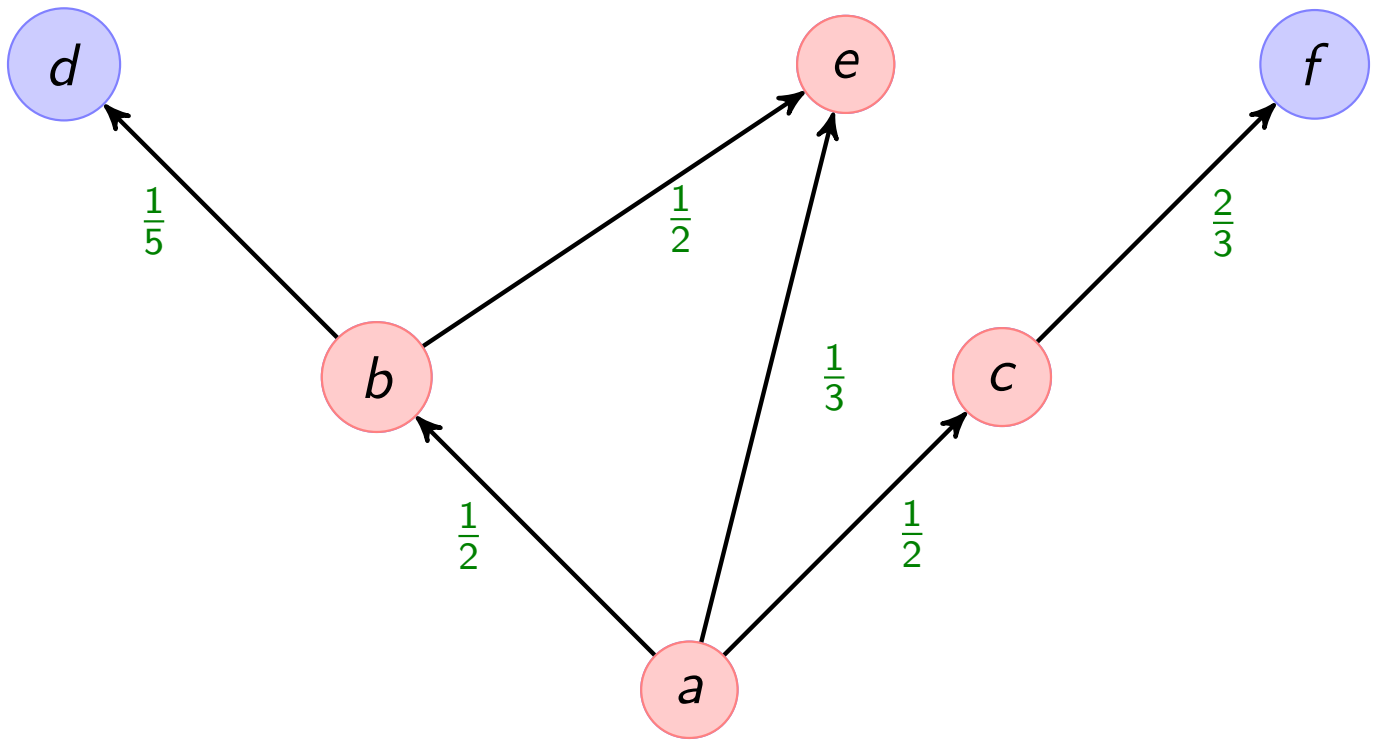

$t = 3$

Look familiar? News spread in Twitter!

How to select a good set of initial adopters

- For an initial set S of adopters, let $f(S)$ be the expected number of eventual adopters. While in general it is computationally hard to find an optimal set S of initial adopters, for the stochastic linear threshold and independent cascade models, $f(S)$ is a normalized, monotone, submodular function.

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- This allows for a very simple "greedy" algorithm that (provably) selects a set S such that $f(S)$ is at least within a factor $(1 - \frac{1}{e}) \sim .63$ of optimality.

- The greedy strategy is to iteratively add (to whatever nodes S have already been selected) one new initial adopter v so as to maximize the expected *marginal gain* $f(S + v) - f(S)$.

- We need to simulate the stochastic process for sufficiently many trials to determine the next node to add. (When different nodes have different utility values, accurate simulation requires that the ratio of such values is reasonably bounded.)

An experimental study comparing methods: Kempe, Kleinberg, Tardos

- To test the usefulness of the models being studied, Kempe et al. compare the greedy by best expected marginal gain algorithm with three other simple (all adding one initial node at a time) methods that do not require simulating the process.
- Namely, they compare against:
  - Greedy by highest degree first
  - Greedy by centrality, i.e. by best average path length
  - Random choice of adopters
- The experimental data set is an undirected multi-graph based on jointly authored papers by physicists.
- Here we have r edges between u and v if they have been co-authors on r papers.
  - In the threshold model, weights $w(u, v)$ are chosen proportional to the multiplicity of edges between u and v.
  - In the weighted cascade model, probabilities are set proportionally.

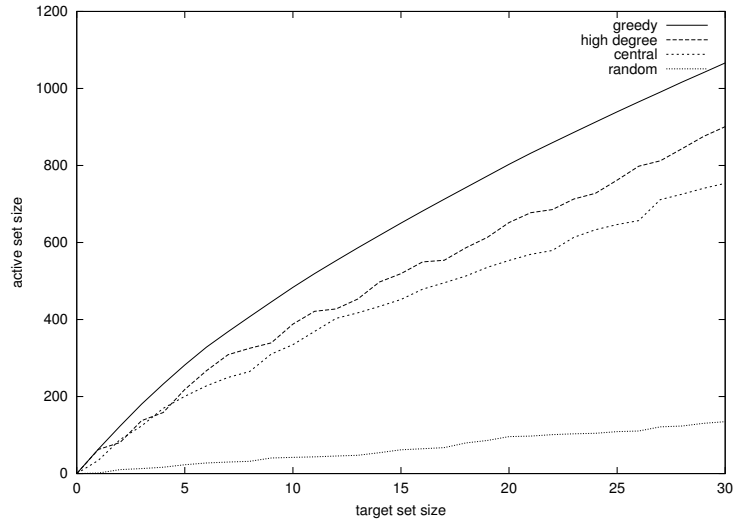

Figure 1: Results for the linear threshold model

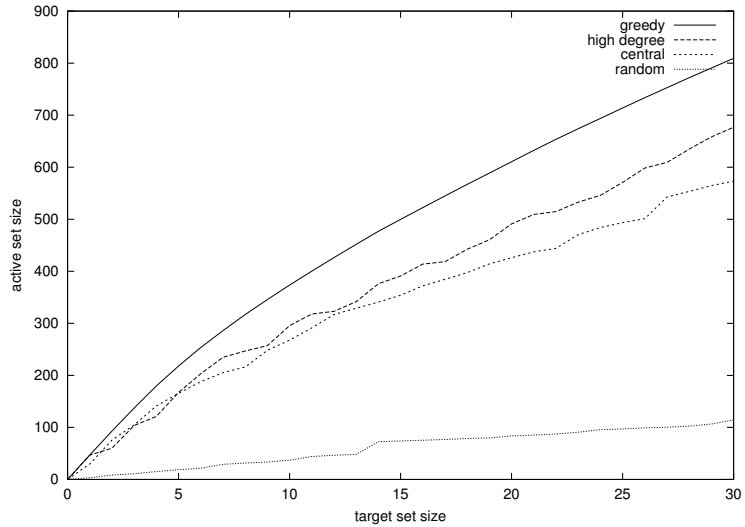

Figure 2: Results for the weighted cascade model

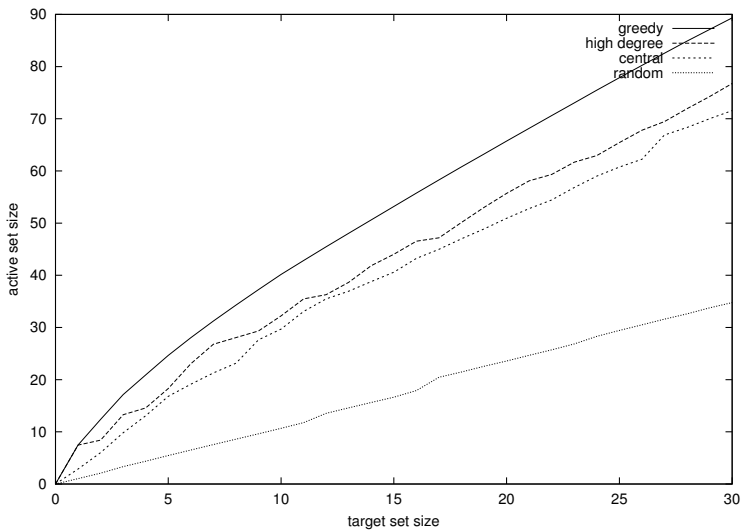

Figure 3: Independent cascade model with probability 1%

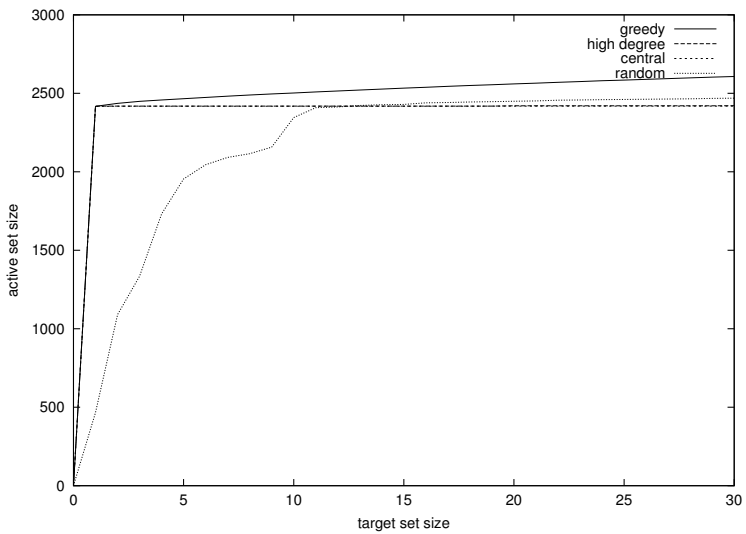

Figure 4: Independent cascade model with probability 10%

Experimental Results from Kempe, Kleinberg, Tardos (2003): "Maximizing the spread of influence through a social network," KDD-03.

Recap

- Choosing influential adopters
  - Linear threshold model
  - Independent cascade model

Aside: The importance of assumptions

- Threshold and (especially) cascade processes are motivated by models for the contagious spread of disease. Should disease spread and influence spread should be governed by similar processes?
  - See `http://www.economist.com/blogs/babbage/2012/04/social-contagion`

Comment from Economist article

- Epidemiology-based models suggest that the most important factor in determining whether an idea (in this case, to join Facebook) will spread to a given individual is how many other people the individual knows who have already been exposed to it
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- Remember! Just because we can abstract two processes (e.g., Facebook adoption & disease spread) to a graph, it doesn't mean that the same assumptions (and therefore, the same models) hold!

Some lessons to be learned about influence in a social network (Chapter 19)

- As seen with the models of popularity, in population-level effects, it can be relatively difficult for a new technology/product/idea to get past a tipping point

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- But tightly knit communities (clusters) can stall the spread.

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- In contrast in social networks, new products/ideas (rumours) can spread extensively and quickly.
- But tightly knit communities (clusters) can stall the spread.
- We saw in the early part of the course that weak ties are often bridges or local bridges between different communities.
- Hence such weak ties may convey some degree of awareness to another community but not likely to change behaviour especially if that change has risks as in political movements and high stakes economic decisions.

Further considerations (collective action)

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- The discussion here begins to combine aspects of social network interaction (e.g. transmitting information) with direct benefit population effects (being part of a large demonstration).
- In particular, the organization for demonstrations against a regime can begin with discussions within a community but for someone to participate, it usually takes some knowledge that there will be a sufficiently large population wide participation.
- On a smaller scale, when challenging a mayor or a CEO, the same phenomena may be operating.

Knowledge and common knowledge

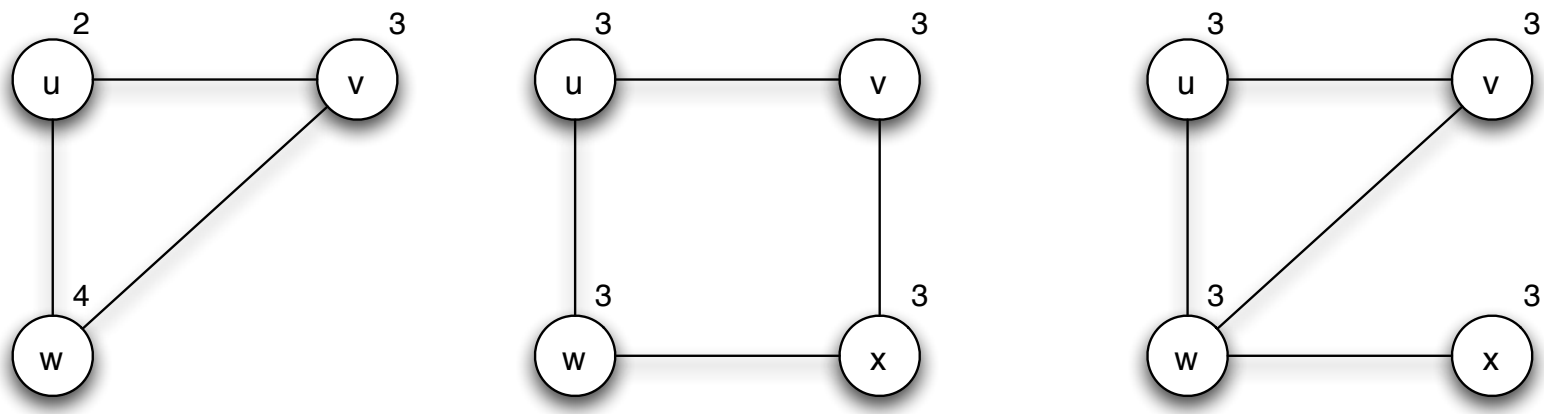
- Our first example of a tightly knit community blocking a complete cascade occurred even when everyone knew the common threshold q.
- A uniform threshold is not realistic in any reasonable size social network.
  - We might have a sense of the thresholds for our friends but not of all their friends (and their friends friends, etc.)

The impact of limited knowledge continued

- As a toy example, consider a company with 4 vice presidents who all wish the Board of Directors would remove the CEO
- However, each VP wants to act as part of a larger group

The impact of limited knowledge continued

- As a toy example, consider a company with 4 vice presidents who all wish the Board of Directors would remove the CEO
- However, each VP wants to act as part of a larger group
- Here threshold k means that the node (being me) will participate if at least k people (including myself) will do so. So in the case of the 4 vice presidents, when will an "uprising" (i.e., calling for the Board to remove the CEO)? The reasoning why an uprising in Figure 19.14 (b) does not occur is perhaps somewhat subtle.



(a) *An uprising will not occur* (b) *An uprising will not occur* (c) *An uprising can occur*

[Fig 19.14, E&K]

Knowledge and common knowledge

- The 3 and 4 node examples in Figure 19.14 illustrate the impact of limited knowledge even when everyone knows the entire network but only knows their friends and their own absolute (i.e. not fractional in this example) thresholds.

Wed. Mar 6: Announcements and Corrections

- Midterm this Friday in tutorial
  - 5 questions, the cover page is on the course website
  - Makeup test is 1 week later (location to be announced)
    - ★ Anyone is free to write the makeup *instead* (you'll miss the tutorial)
    - ★ My **strong** recommendation is to only write the makeup in the case of unexpected & significantly impacting situations, or immovable prior commitments
    - ★ The make up is **NOT** an extra week to study. There is no makeup makeup test, and tutorials, course material, and assignments will continue to advance without you

Further considerations: competitive influence spread

- In many economic, social, and political settings the spread of influence is a competitive process.
- It may be that both technologies (political factions, etc.) A and B are competing for new adopters in a social network by promotion via an initial set of adopters (people with vested interests, etc.).
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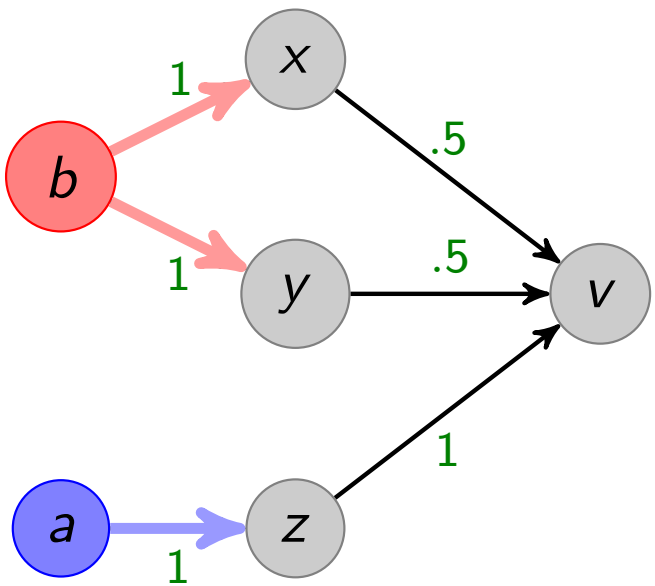
- There are many models for how such competition is resolved.
- One possibility is to use the stochastic independent cascade model and then the first technology (political faction, etc.) to have a "path of adoption" succeeds (breaking ties in some manner).
- Equivalently, we can view edges as being open or closed with some probability, and consider the shortest open paths to a node (if any exist) from the initial adopters (party faithful, etc.) to the initially uncommitted.

The Wave Propagation Process

- Two technologies A and B with their sets of initial adopters I_A and I_B.
- Technology spreads according to the Independent Cascade process.
- If a node is successfully infected at the same step t by both
  - a set of nodes V_A using technology A
  - a set of nodes V_B using technology B

  it will adopt technology A with probability $\frac{|V_A|}{|V_A| + |V_B|}$



Example

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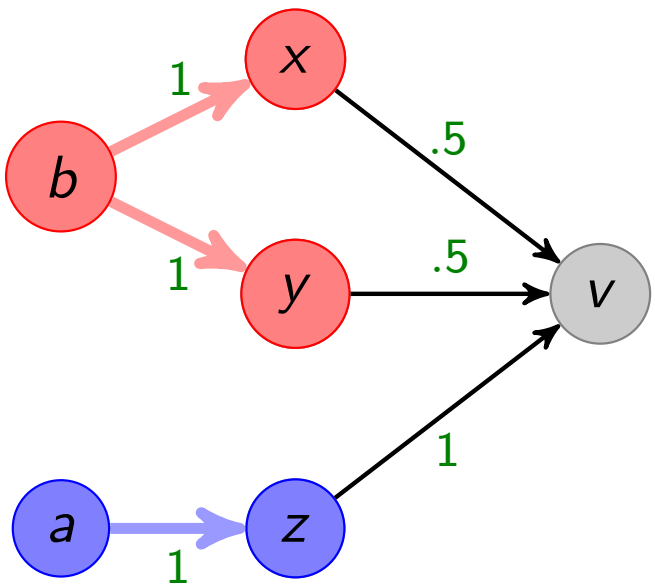

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The Wave Propagation Process

- Two technologies A and B with their sets of initial adopters I_A and I_B.
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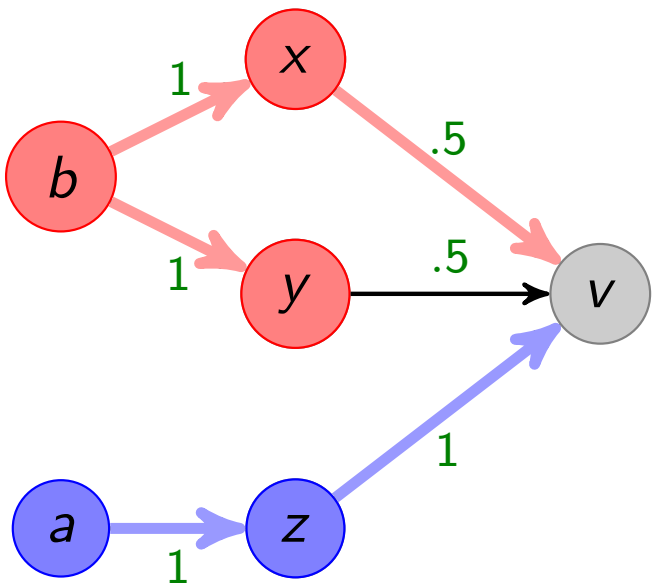

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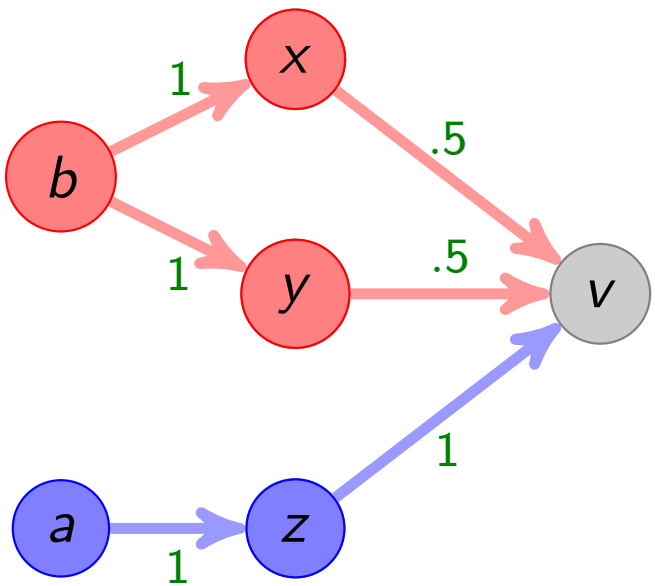

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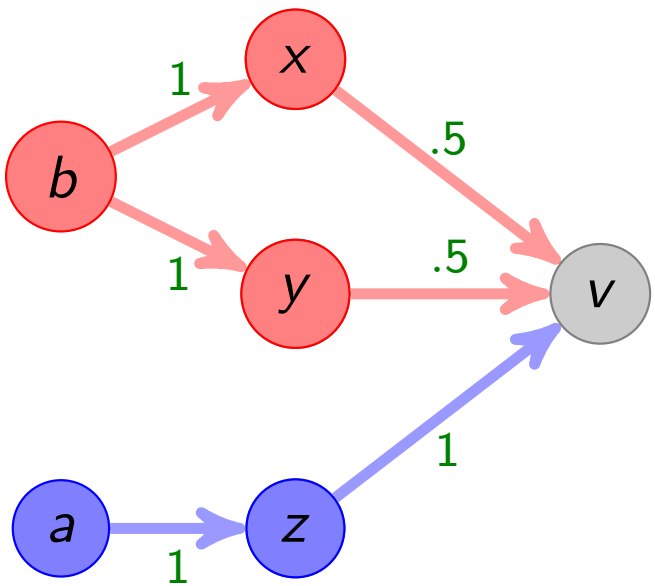

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- What is $Pr[v \text{ adopts } A]$?

A concluding comment for chapter 19

- The last sentence of the chapter makes the final comment:
  Even small extensions [...] can introduce significant new sources of complexity, and the development of even richer extensions is an open area of research.

- Indeed, as we have already suggested, analytic and empirical studies of influence spread in social networks is a field of significant research interest impacting computer science, sociology, economics, and political science.

Recap

- Choosing influential adopters
  - Linear threshold model
  - Independent cascade model
- Local vs. global knowledge
- Competitive influence spread

Chapter 21: Epidemics and the spread of disease in a contact network

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- The spread of a disease and the dynamics of an epidemic clearly depend on the nature of the disease (e.g. how infectious, periods of incubation, periods of contagion, one-time vs recurring infection).
- But the spread process also depends on the contact network within which the process is unfolding. Of course, our interest here is in the way in which we model these dynamics and how the network characteristics impact the process.

How does social/information spread differ from disease contagion?

- Both disease spread and social contagion are often best viewed as a stochastic process
  - However, as mentioned before, disease spread is general determined by the total amount of exposure, which may or may not be the case for social contagion (e.g., Facebook spread is better determined by number of social groups rather than people)

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- A more intrinsic difference in these studies is that in contact networks (for disease spread), the links are often considered to be transient (i.e. only lasting for some period of time) whereas our study of social spread, small worlds and decentralized search were discussed in the context of permanent relationships (i.e.a static network).

Pure branching processes

- For simplicity (as we did in Chapter 20 and the study of decentralized search), we start off with a tree network (i.e. assuming no triadic closure)
  - assume that every individual v at time t comes in contact with k new individuals
  - if v is infectious at time t, then with probability p, v will independently pass on the disease to each of these new contacts by time $t + 1$

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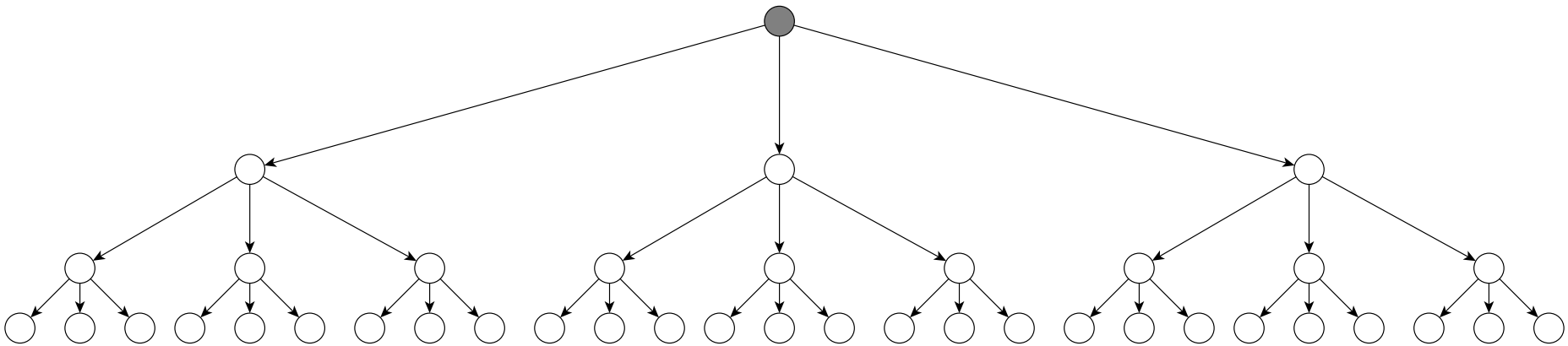
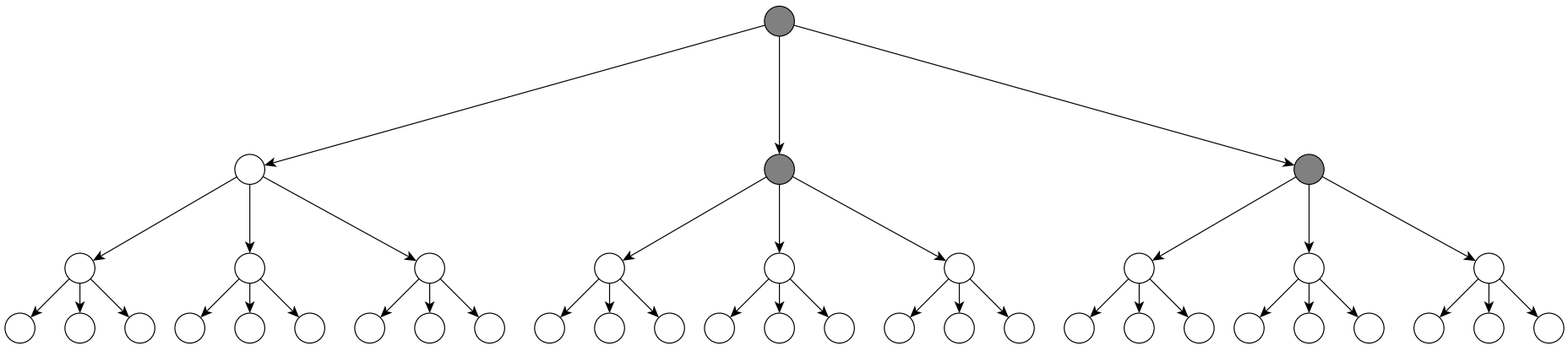
  - if v is infectious at time t, then with probability p, v will independently pass on the disease to each of these new contacts by time $t+1$
- That is, if a given (root) individual initially (at time $t=0$) is infectious, then at time 1, there will be k people, each of which will independently contract the disease with probability p and become infectious
  - Any of these (say k') newly infected individuals are potentially passing on the disease to some of the $k \times k'$ individuals who have indirectly come in contact with the root by time 2, etc...

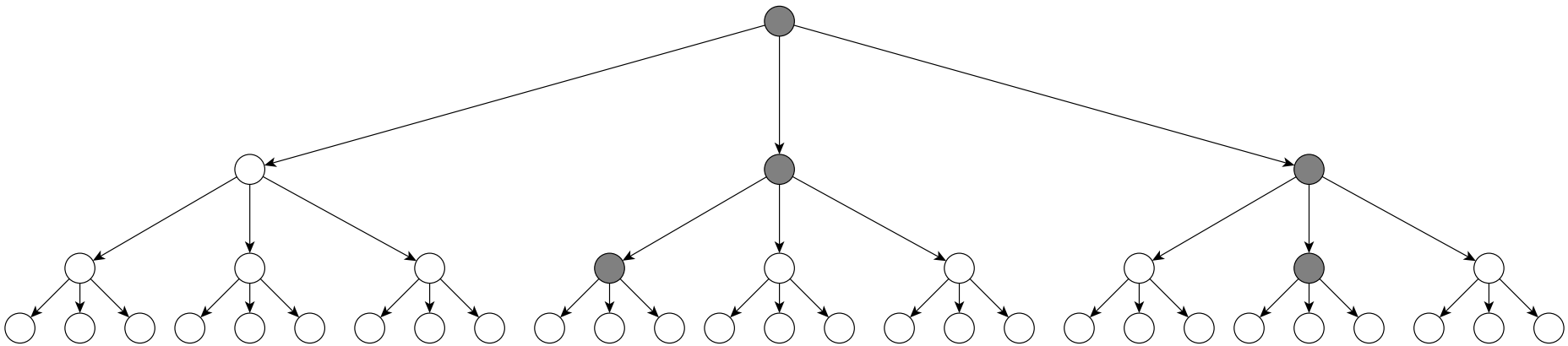
The tree network at time $t = 0$



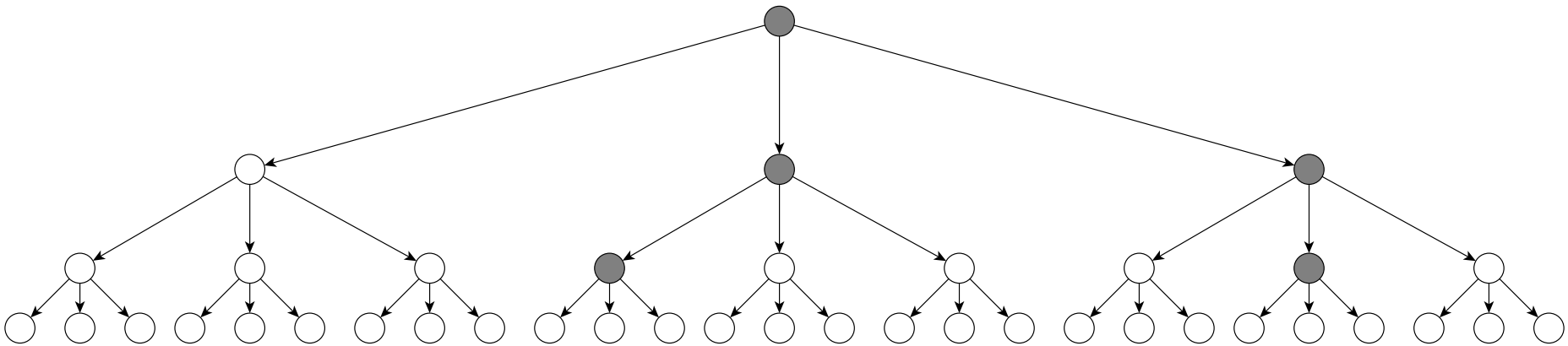
The tree network at time $t = 1$



The tree network at time $t = 2$



The tree network at time $t = 3$



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  - What is R_0 in this simple branching process? $R_0 = p \cdot k$
- When $R_0 < 1$, the disease will eventually die out
  - Intuitively, each individual is not able to sufficiently replenish the disease (even if by the randomization of the process the number of new infections fluctuates for a while)
- When $R_0 > 1$, unless the disease gets unlucky (and society gets lucky), the disease is likely to persist and continue to witness new infections at every time step and indeed the infection will likely be wide spread

$R_0 > 1$: likely that disease spreads widely

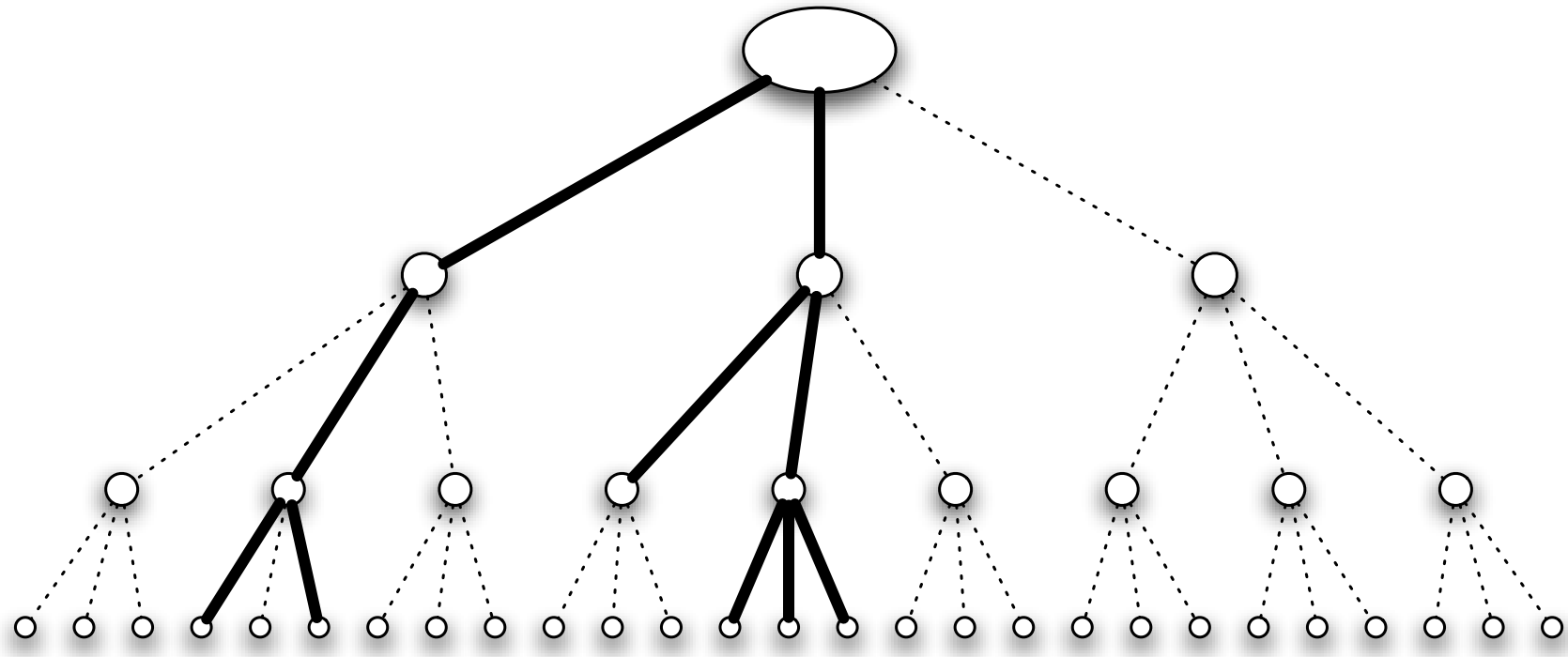

Figure: High reproductive number. [Fig 21.1(b), E&K]

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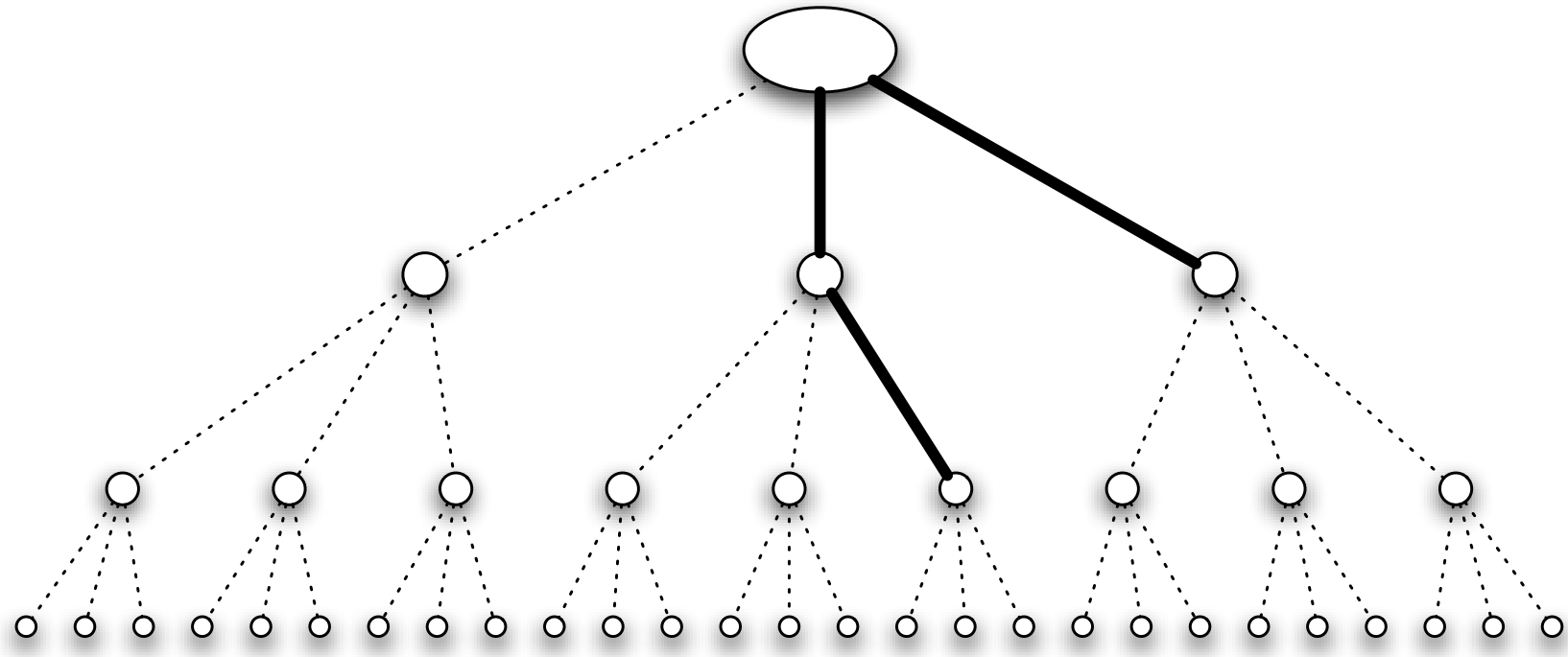

Figure: Low reproductive number. [Fig 21.1(c), E&K]

A simple conclusion from a simple model

Given that we are starting with such a simple model, we can't expect to draw many conclusions. But one conclusion is as follows. When the basic reproductive number R_0 exceeds 1, there is a huge societal benefit in trying to reduce k or p so as to lower R_0. How?

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Aside: The above is a misuse of the term R_0, strictly speaking the basic reproductive number is in the absence of public health measures. Only the *effective reproductive number at time t*, R_t, can be reduced by the measures above, but that's beyond the scope of the course.

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- **S:** The *susceptible state* where the individual can contract the disease
- **I:** The *infectious state* when the individual has caught the disease and now is infectious with some probability of spreading the disease
- **R:**The *removed state* when the individual is no longer infectious and is "removed" from further consideration
  - Clearly there are good (recovered and living) and bad ways to be removed
  - Regardless, once someone has had the disease, under the SIR model we assume that they are immune in the future

The SIR Process

- Initially, some nodes are in the infectious state I, and all others are in the susceptible state S

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- The infectious state can be partitioned in sub-stages (e.g. early, middle, late stages of infection) with different contagion probabilities
- The disease itself mutates during an outbreak or epidemic which then continues to dynamically change the process

The course of an SIR contagion spread with $t_I = 1$

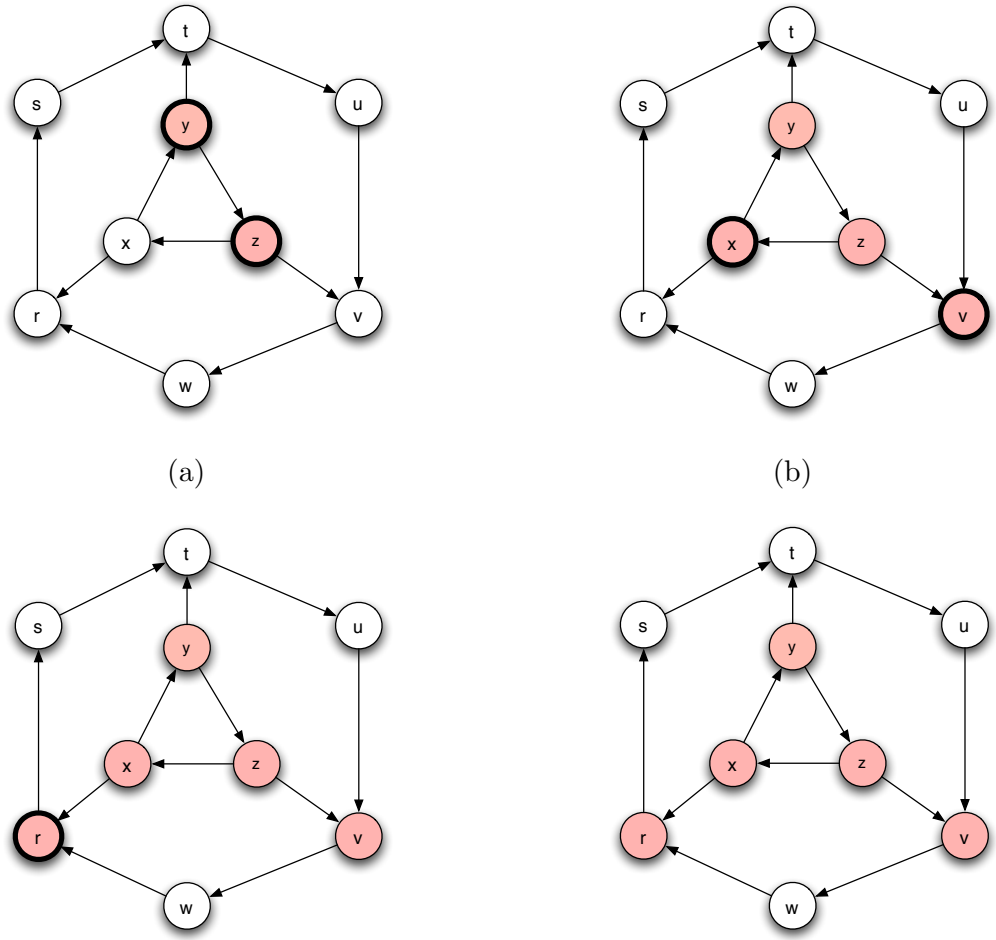

Figure 21.2: The course of an SIR epidemic in which each node remains infectious for a number of steps equal to $t_I = 1$. Starting with nodes y and z initially infected, the epidemic spreads to some but not all of the remaining nodes. In each step, shaded nodes with dark borders are in the Infectious (I) state and shaded nodes with thin borders are in the Removed (R) state.

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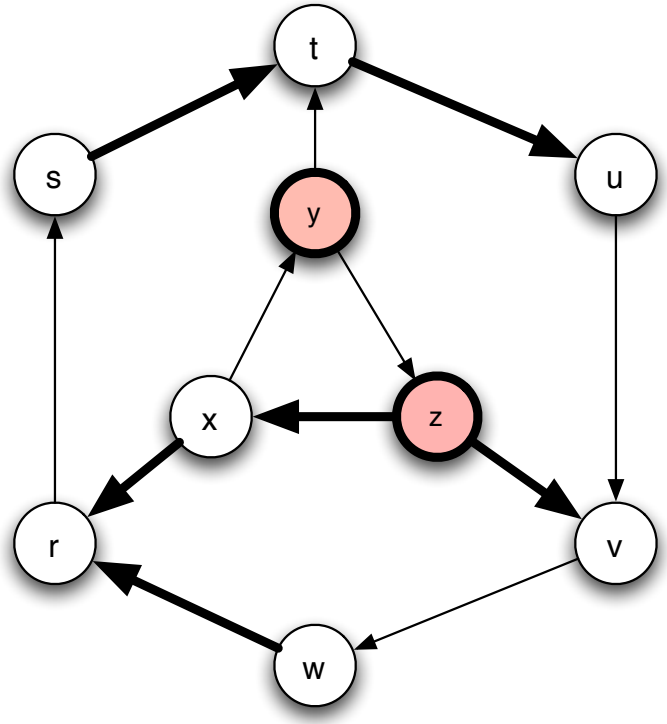
- In the following figure, nodes s,t,u,w will not become infected in the instantiation depicted by the bold open edges. The other nodes will become infected at some time.

Alternative view of the previous specific instantiation



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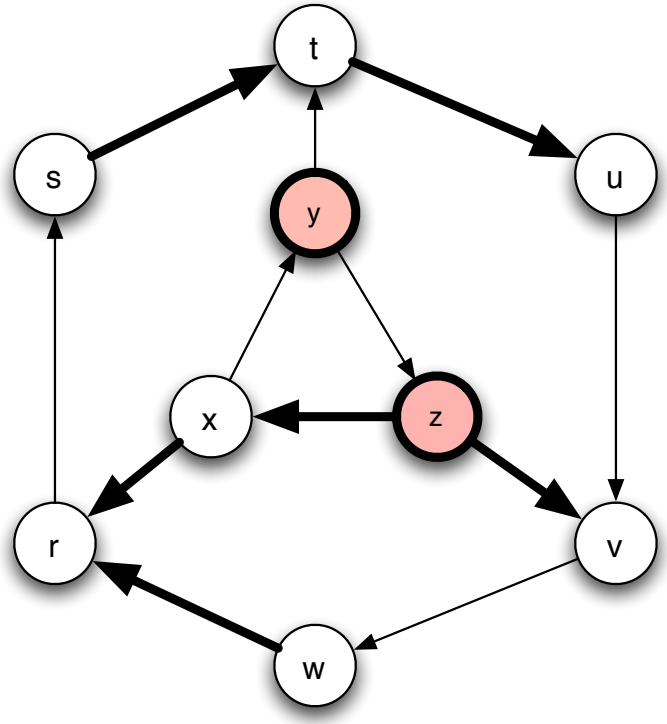

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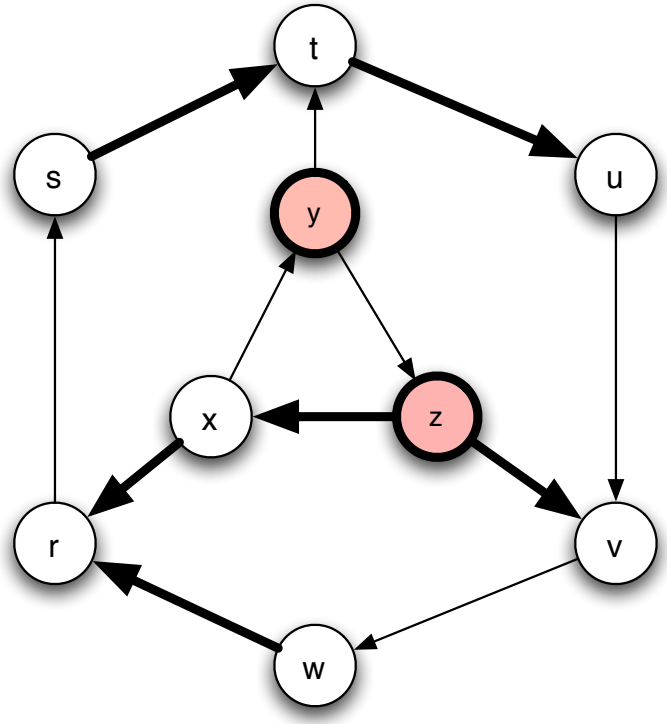

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- When $t_I = 1$ we can get away with labelling edges as open or closed
- when $t_I > 1$ we need to label edges with when they are open

Roadblocks to contagion spread

- In the context of social influence spread, we saw that tightly knit communities can be isolated against the adoption of a new technology
- Similarly, once we move away from the pure branching process, the basic reproductive number R_0 no longer completely determines the extent of contagion

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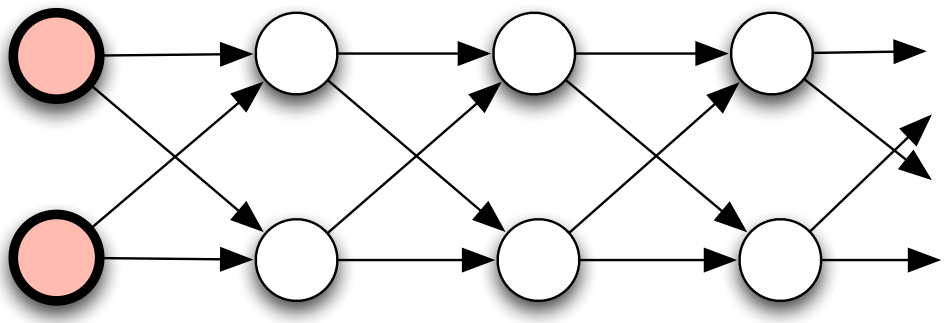

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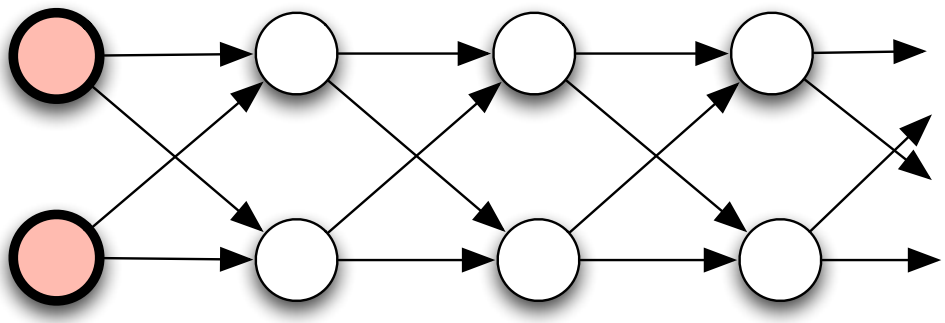

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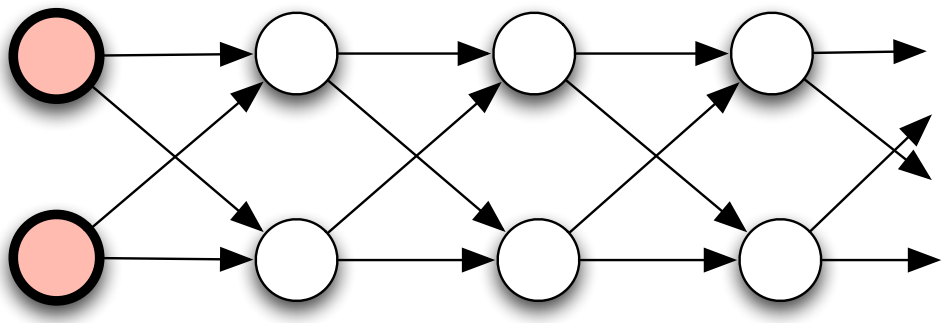

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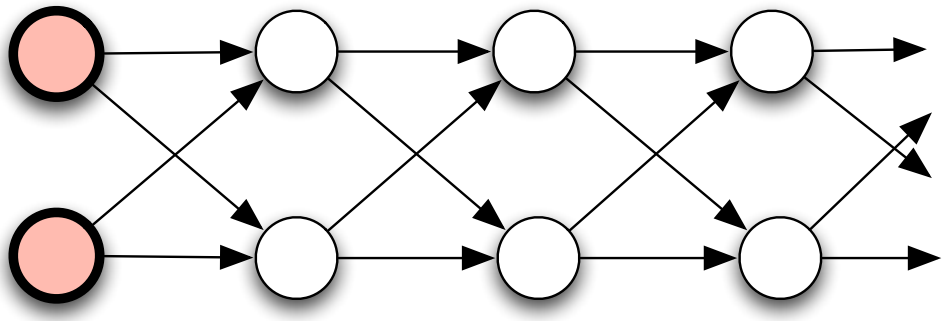

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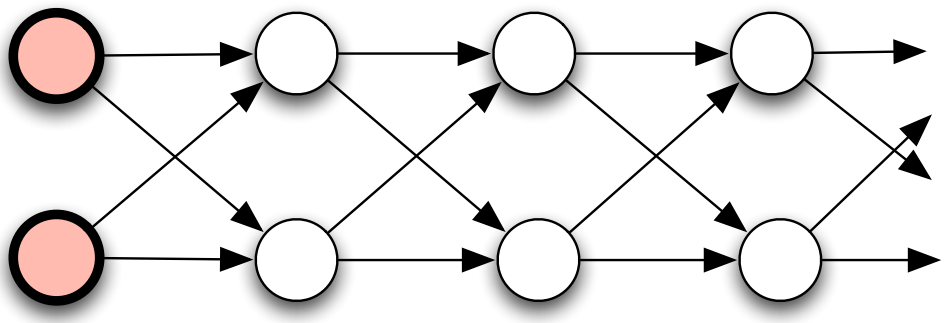

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  - What's the long term behaviour? The disease would have to continue to pass through a narrow channel where there is a probability of $q = (\frac{1}{3})^4$ that all four edges in some stage of this network will fail to transmit and hence the disease will be wiped out

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- After t_I steps, node v is no longer infectious and returns to the susceptible state S

Representing an SIS process as a sequence of SIR iterations

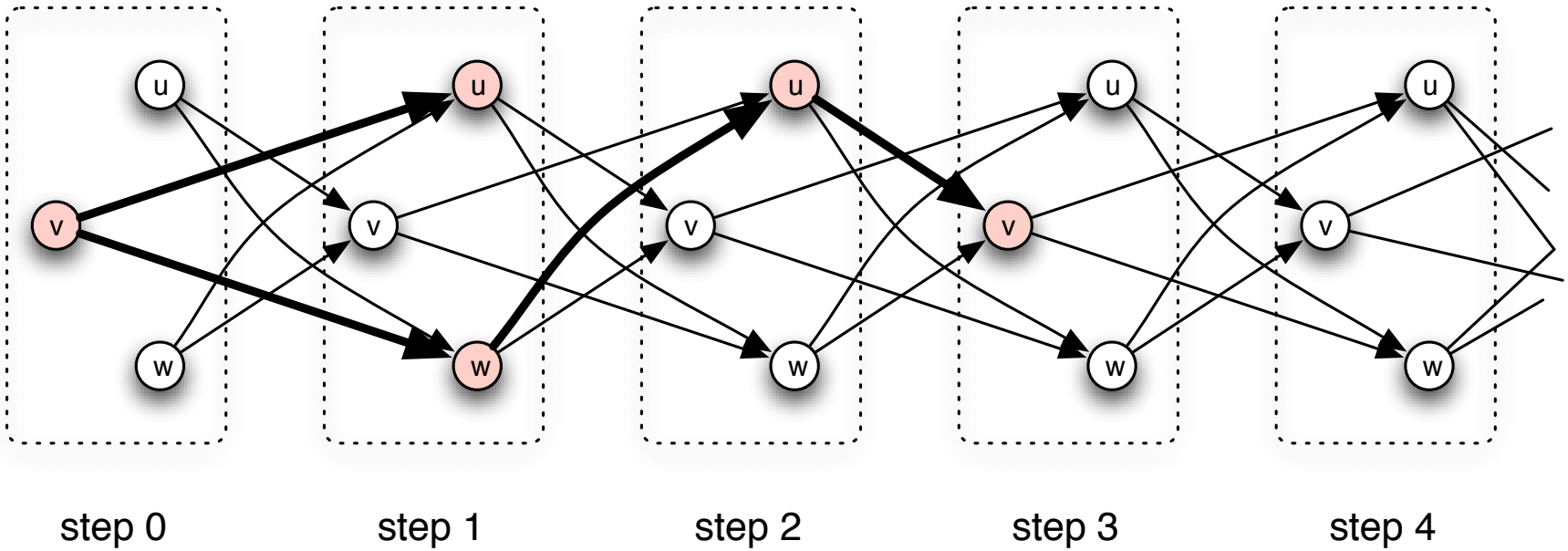

Figure: A **SIS** process (with $t_I = 1$) depicted as a sequence of **SIR** steps. [Fig 21-6(b), E&K]

Extensions of the SIS model

- The basic **SIS** model can be extended in many ways. For example:
  - As in the **SIR** model, there can be different probabilities $p_{(u,v)}$ associated with each network edge (u, v)
  - An individual only returns to the susceptible state S with some probability q
  - There can be multiple stages of an infection with each stage having different contagion properties
- An interesting modification is the following **SIRS** model which provides insight into why some diseases seem to show a time oscillating behaviour in terms of the extent of infection in given populations

The SIRS model

- As in the previous models, initially some nodes are in the infectious I state; all others are in the susceptible S state
- Each node v that enters the infectious state stays infectious for some t_I steps
- During each of these t_I steps, each infectious v has a probability p of infecting each of its susceptible neighbours

The SIRS model

- As in the previous models, initially some nodes are in the infectious I state; all others are in the susceptible S state
- Each node v that enters the infectious state stays infectious for some t_I steps
- During each of these t_I steps, each infectious v has a probability p of infecting each of its susceptible neighbours
- After t_I steps, the infectious node v enters the R (i.e., a period of immunity) state for some t_R steps
- After these t_R steps, the node returns to the S state
  - Either or both t_I and t_R can be random variables

Disease oscillations

- The presence of periods of immunity in the **SIRS** model induced by the t_R parameter can produce oscillations in localized parts of a network
- It can also cause seemingly coordinated outbreaks of a disease in different parts of the network

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- To explain how this can occur, consider a network that has long range edges in addition to edges within small neighbourhoods.
  - This is, of course, reminiscent of the network structure that provided an explanation for the small world phenomena
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  - Indeed, Kuperman and Abrahamson [2001] consider a network model following the original network model of Watts and Strogatz
- More specifically, we have a network with edges connecting (graph theoretically) nearby nodes augmented with some edges chosen uniformly at random
  - Note that here the random edges do not depend on distance, unlike the model used to explain decentralized search in Chapter 20

The Kuperman and Abrahamson model

- Specifically, Kuperman and Abrahamson consder a one dimensional model contructed as follows:
  - Nodes are arranged in a ring (i.e. a cycle) with edges between nodes within some small distance of each other
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  - Then with some probability c, an edge is redirected randomly to a node chosen uniformly at random
- They then study the **SIRS** contagion model for such a stochastic network
- As we might expect the behaviour of disease occurrence in such a network will depend on the probability c of redirecting an edge even when fixing p (the probability of transmitting the disease), t_i (the duration for being infectious, and t_R (the period of immunity)

Simulations from Kuperman and Abrahamson

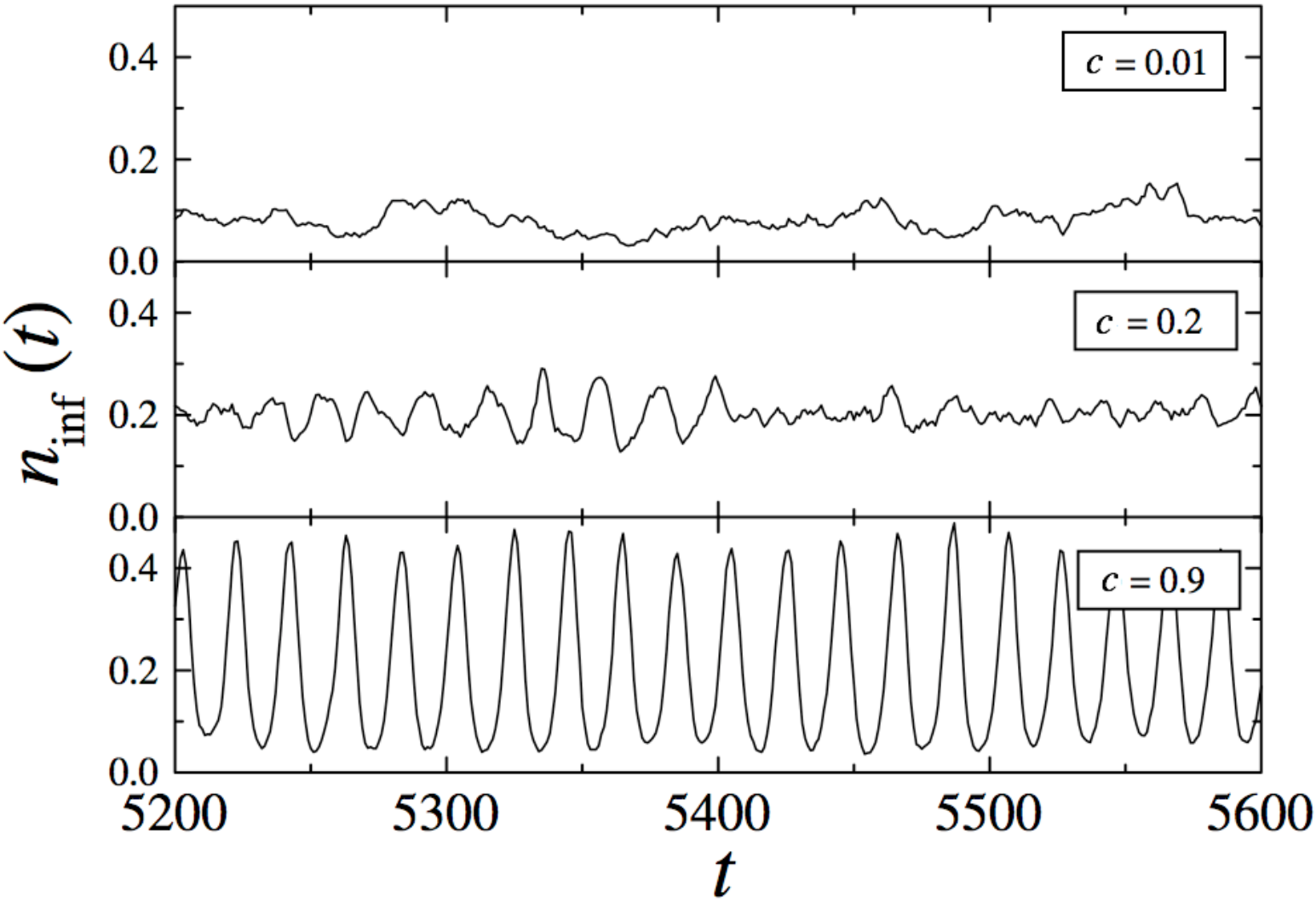

Figure: The plots depict the number $n_{inf}(t)$ (at time t) of infected people in an **SIRS** contagion spread. Figure and results are due to Kuperman and Abrahamson.

Reflections on the Kuperman and Abrahamson study for a syntactic network, and empirical findings

As always the text cautions us about the significance of models, and in this case, the simplified network model. Still, it is interesting to observe how different the results are for different settings of the random redirection probability c

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However, there is some real world findings for which the **SIRS** model provides some insight (into observed oscillations in disease outbreaks)

- Grassly, Fraser and Garnett [2005] compared the differences in the occurrence of two STIs, namely syphilis and gonorrhea. Namely syphilis exhibits oscillations on an 8-11 year cycle whereas gonorrhea does not exhibit any substantial periodic behavior

How to explain the differences in the spread of two different STIs?

This difference in oscillating behaviour is, at first thought, surprising since the method of contagion spread is the same and the underlying network for social relations is also the same. What is a plausible explanation?

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This difference in oscillating behaviour is, at first thought, surprising since the method of contagion spread is the same and the underlying network for social relations is also the same. What is a plausible explanation?

- Syphilis has limited periods of temporary immunity after infection whereas gonorrhea does not
  - The oscillation periods for syphilis seem to correlate well with the timing of immunity (i.e., the t_R parameter)
- The extent to which the outbreaks of syphilis are synchronized in the U.S. increased over the second half of the 20th century
  - This can be explained by increasing levels (i.e. the redirection parameter c) of cross-country contacts

Mon. Mar 11: Announcements and Corrections

- Makeup Midterm this Friday during tutorial time, **in the lecture hall (WB116)**
- Initial draft of critical review, and assignment 2, are both due in about a week and a half

The transient nature of contacts

In our introduction of contact networks and models for disease spread, we noted that there is a dynamic aspect to such models. This manifested itself in the duration for being contagious. However, the underlying network itself was static. This is not a bad assumption for infections that spread quickly at a faster pace than the creation and ending of contacts.

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We can extend the contact network models to reflect very transient contacts, by specifying (on the edges) the time period when individuals are in contact with each other and can transmit the disease.

The transient nature of contacts continued: exact timing maters

It should not be surprising that the exact timing matters, the order of contact determines how the disease can travel through a node.

And as the text points out, this transient behaviour of contacts can apply to settings outside of disease spread such as information spread.

The transient nature of contacts continued: exact timing maters

The following example illustrates the impact of order while keeping the duration t_I of infection fixed

- We fix $t_I = 5$
- Each edge $e = (k, \ell)$ is labelled by an interval $[s_e, f_e]$ indicating that individuals k and ℓ were in contact starting at time s_e and ending at time f_e
- In these examples, the number n_e of time steps of contact has been set to $n_e = 5$ for all edges. It is an unfortunate coincidence that $n_e = t_I = 5$, as this is not mandated by the model

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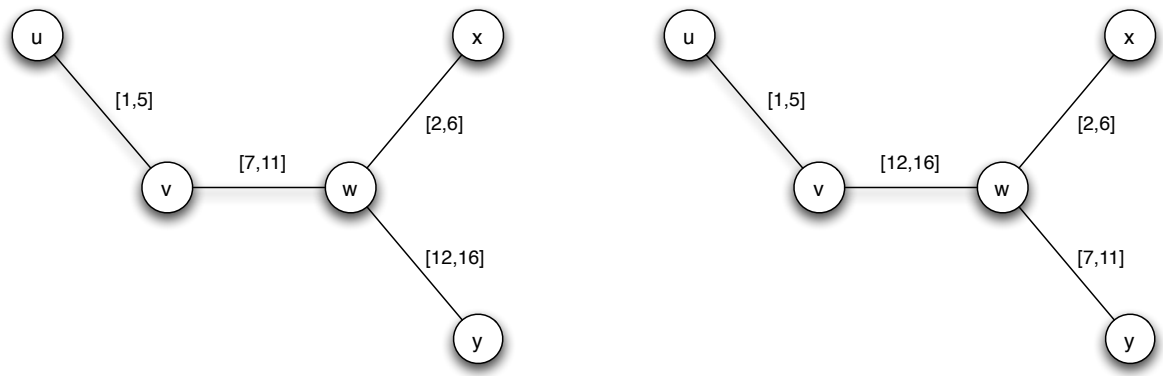
The assumption is that if individual k becomes infectious at some time $t \in [s_e, f_e]$, then ℓ can possibly be infected at some time step t' with $t + 1 \leq t' \leq \min\{f_e + 1, t + t_I + 1\}$

The impact of timing

- The only change in the networks is that the period of contact between v and w has been switched with the period of contact between w and y
- Assume node u is initially infected (at some time $t \in [1, 5]$). Let's ignoring the probability of becoming infecting and just looking at what is possible:
  - In the network on the left, it is the possible that the disease could pass to all nodes except node x
  - In the network on the right, only node v can become infected



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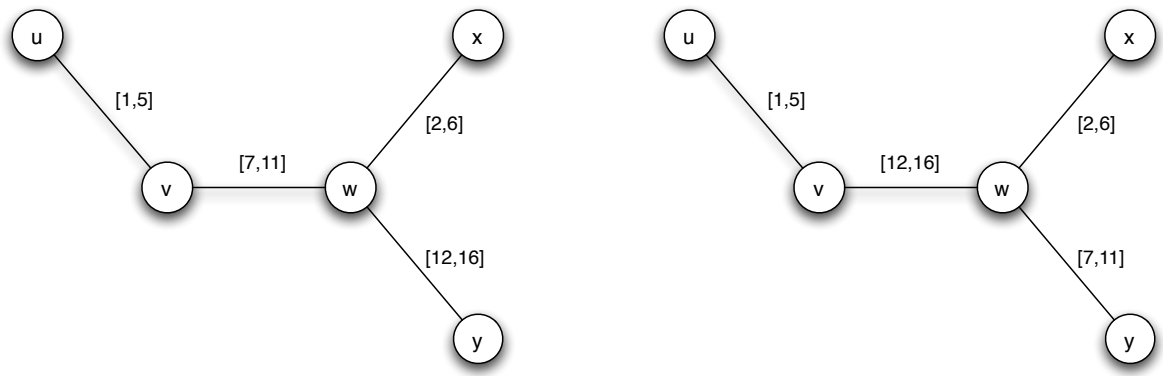
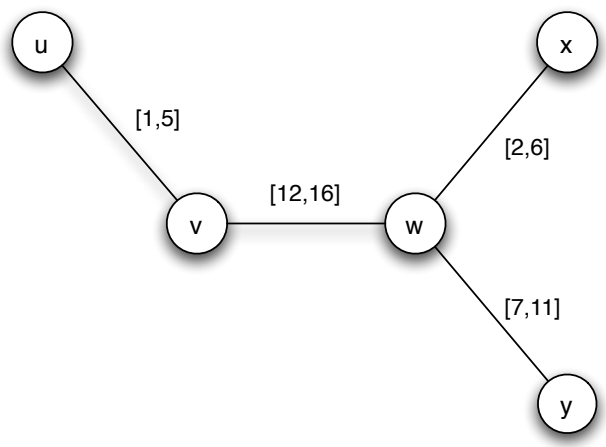
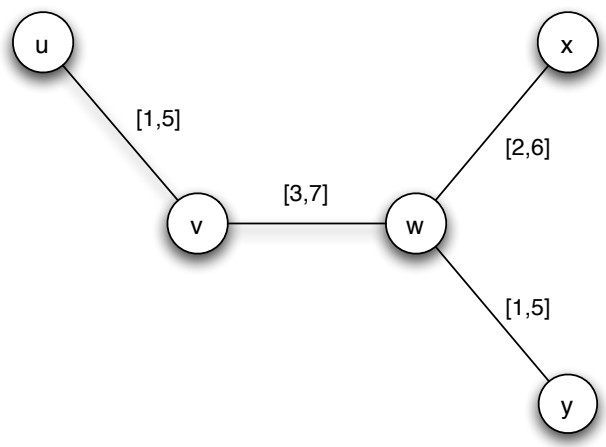


We can see that the timing on the edges of w allows for infection to move in only one direction between v and y through w

The impact of concurrency

- It should not be surprising that the more contacts occur simultaneously, the more extensive will be the spread of a disease
- Again assume $t_I = 5$
- Here we have the same underlying network as in Figure 12.8 and . But now the times for concurrent contact have been significantly altered
  - In the figure on the left, there are no concurrent times of contact between any two individuals
    - ★ In this case, no individual can spread the disease to everyone else
  - In the figure on the right, any single individual can possibly spread the disease to everyone in the network





Recap

With practice & review, you'll be able to:

- Explain the initial adopter problem, and recall the conditions seen in class for efficient approximation
  - Define the linear threshold model
  - Define the independent cascade model
  - Prove these models meet the required conditions for efficient approximation
- Define and execute a simple collective action model, and explain how it illustrates the impact of local vs. global knowledge
- Define and execute the wave propagation process for competitive influence spread
- Model disease contagion by determining the relevant properties of a disease
  - Define the basic reproductive number (R_0)
  - Define, execute, and calculate probabilities under the SIR, SIS, and SIRS models
    - ⋆ Explain the interpretation of the SIR model as percolation
    - ⋆ Explain the possible impact of graph structure on R_0
  - Modify SIR/SIS/SIRS to reflect transient contacts