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INSTITUT DE HAUTES
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GRADUATE INSTITUTE
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Diffusion

Introduction to Social Networks

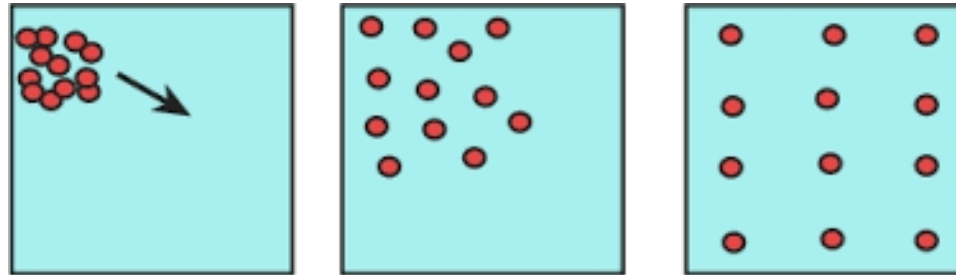
James Hollway

Networks and Diffusion

- Networks not just abstract structures, also
 - conduits for influence (pipes)
 - equivalencies for emulation (prisms)
- Network ties and structures provide **opportunities**
- Seek to apply basic ideas of social networks to **diffusion**

Diffusion

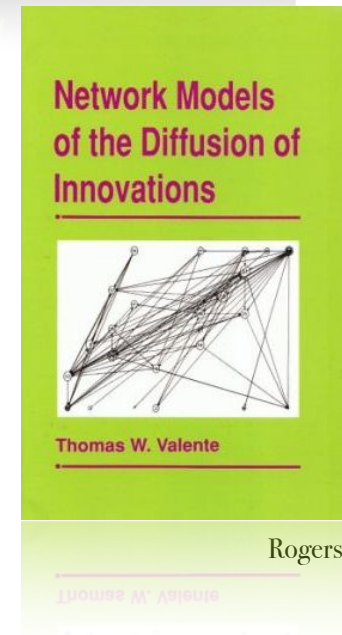
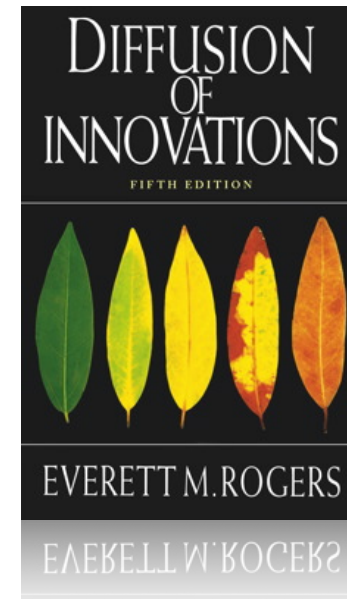
- According to *Dictionary of the History of Science*, the term ‘diffusion’
 - originated in the 19thC with Graham and Maxwell
 - Latin etymology, *diffundere*, means “to spread out”



- Compare with adjacent terms “contagion”, “adoption”, etc.

Social Diffusion

- Pre-paradigmatic heritage from Galton (cultural traits), Pemberton (postage stamps), and Stuart Chapin (sociology)
- Early diffusion in **rural sociology**
 - 2 rural sociologists at Iowa State University, Ryan and Gross
 - “Diffused” to other midwestern rural sociological researchers in ‘50s and ‘60s, and then larger, interdisciplinary field
- By late 1960s, rural sociologists lost interest
 - Not because ineffective scientifically
 - But lack of support during era of overproduction
 - And most interesting questions thought to be answered...
- Now most prevalent in biology: **epidemiology**



Rogers 1995, Valente 1996

What diffuses? Examples

Diseases

Fires

Rumours

Purchases

Innovations

Knowledge

Behaviour

Beliefs

Norms

Perceptions

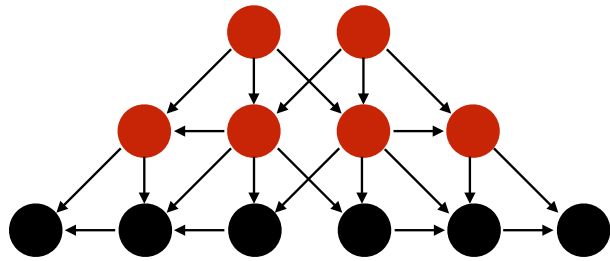
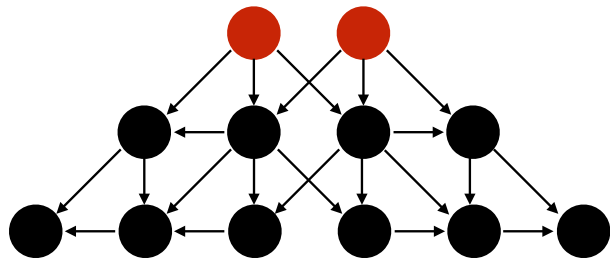


Compartmental
Models

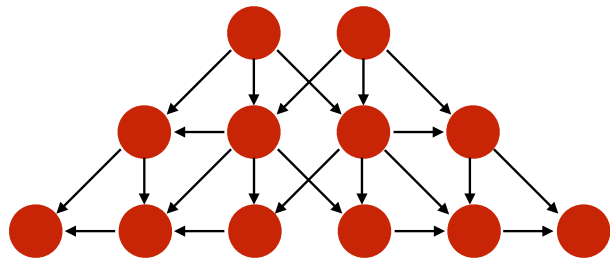
Epidemiological Models

- Simulating diffusion involves a basic process:
 - Select n nodes as seeds
 - At each step, each infected node may diffuse/transmit its (network) neighbours with probability p (transmission rate β)
- Most simple (SI) model moves nodes between two compartments:
 - Susceptible (S)
 - Infected (I)

Simple diffusion



Exposure \rightarrow infection/adoption

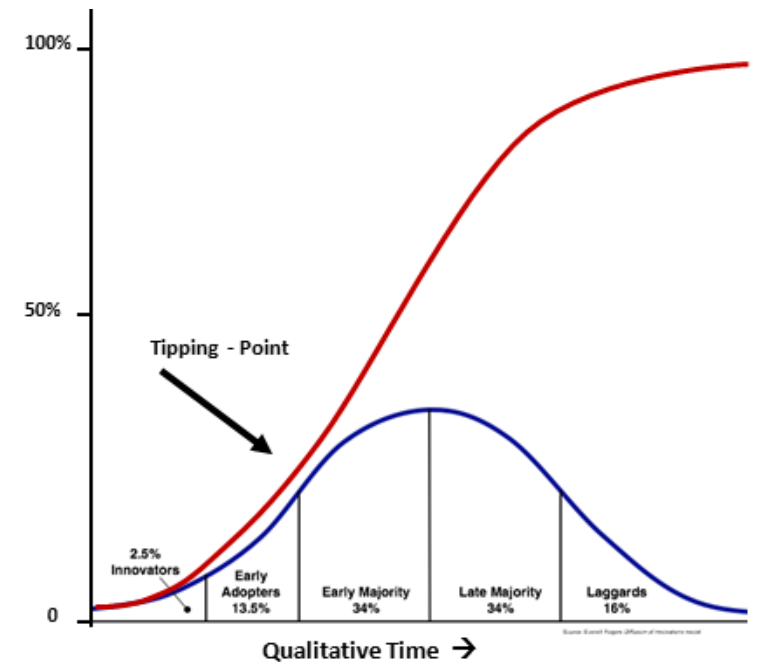


Diffusion and Adoption

- Given the transmission rate β , SI model can be described with the differential equations:

$$\frac{dS}{dt} = -\beta SI \quad \frac{dI}{dt} = \beta SI$$

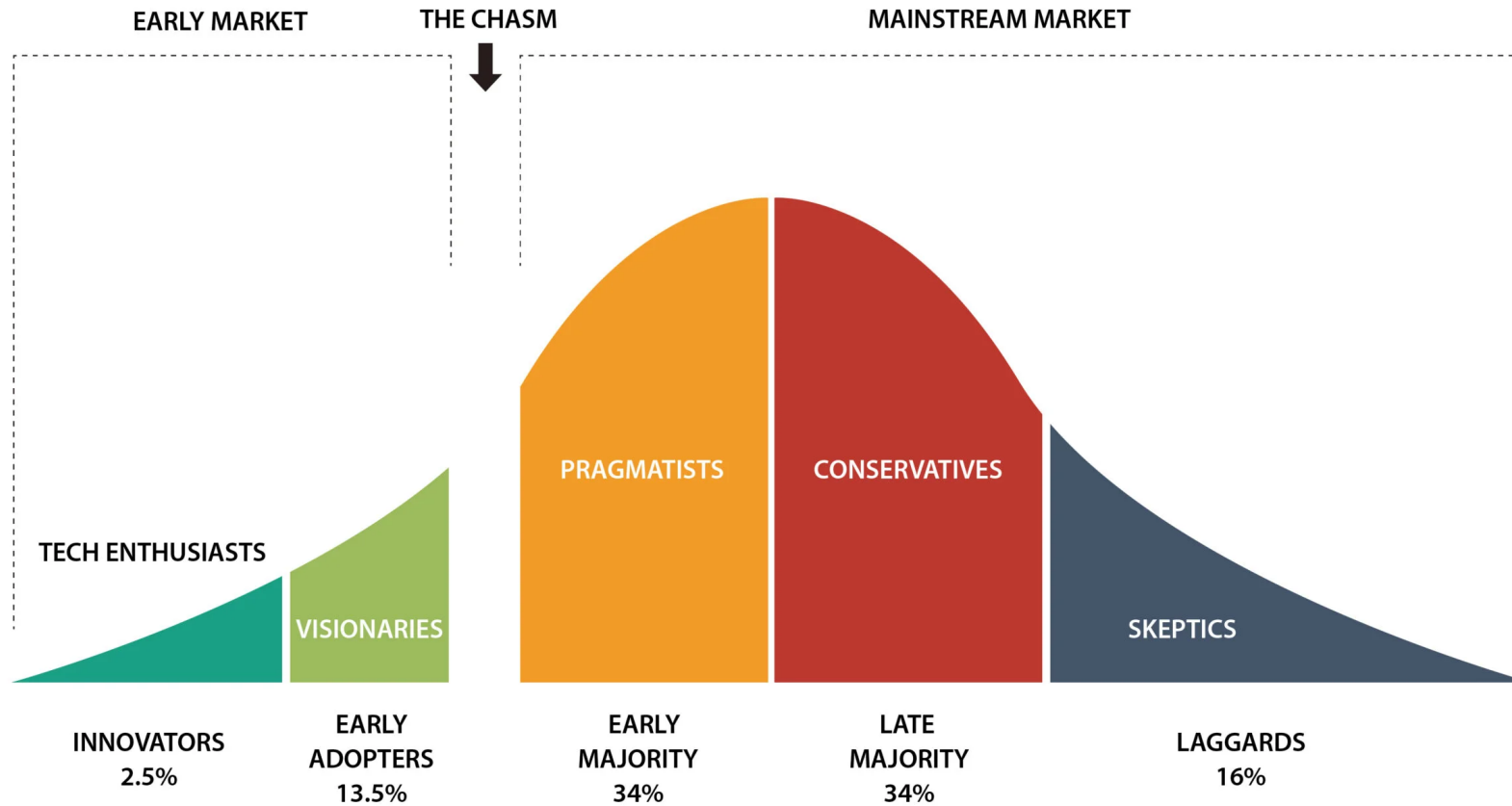
- where $I + S = 1$
- Solving this equation gives us a logistic growth function (an S-shaped curve)
- This curve increases fast after it crosses the critical/**tipping point**, and grows much slower in the later stage



Interested in...

- Growth curve form
- Total number of infected
- Final prevalence
- Duration of an epidemic
- Peak infection
- Tipping point
- Reproductive number
- Adopter class
- Intervention strategies
- Seed strategies

Classifying adopters

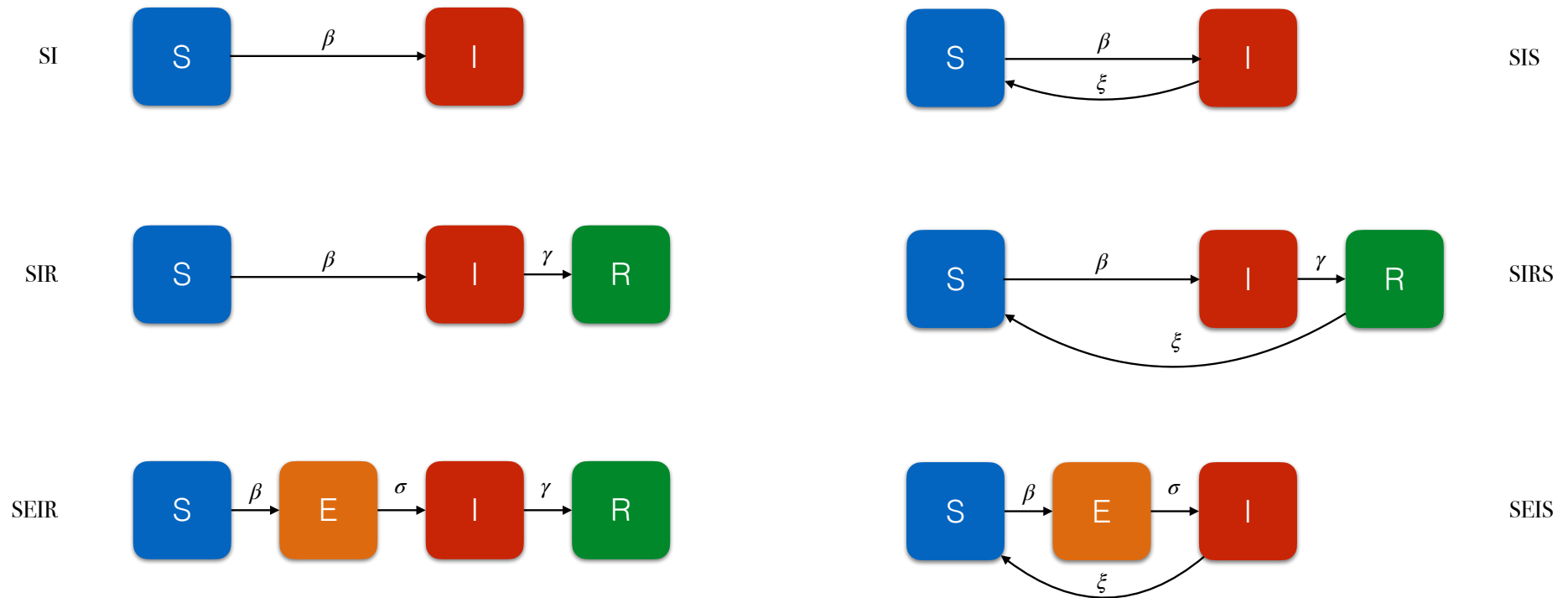


Ryan and Gross 1943; Rogers 1995; Valente 1996

Naiiveté of the SI Model

- For more realistic epidemic spreading, we should consider how the status of infected change...
 - They might recover but become susceptible again (**SIS model**, like gonorrea)
 - They might recover and then be immune (**SIR model**, like chicken pox)
 - They might be removed the network (like death)
- Recovery or other rates can be captured with additional parameters, e.g. γ

Range of compartmental models



See also SIRD, SIRV, SICR, and MSIR models

R_0

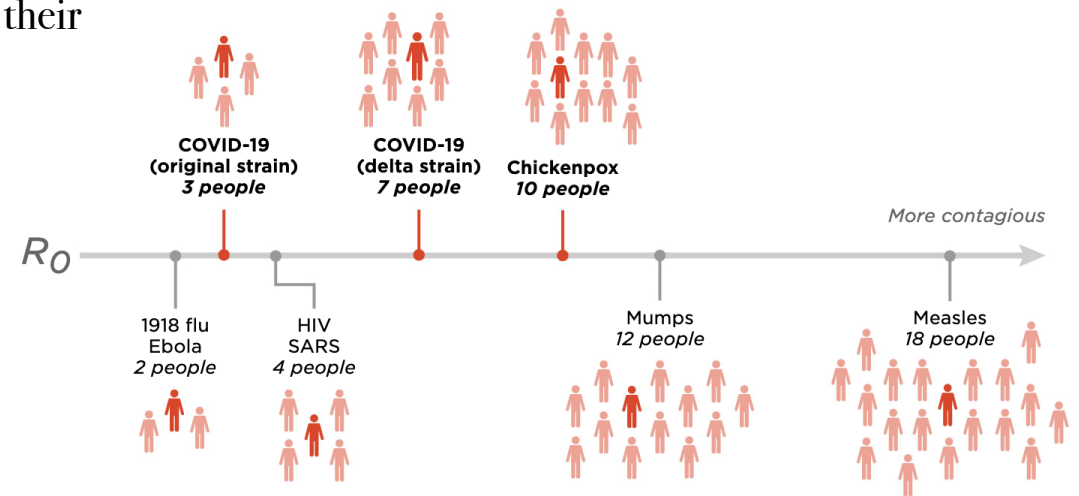
- The **basic reproductive number** is defined as the expected number of people infected from a contagious person over the length of their contagiousness (in a fully susceptible population).

- $R_0 = \frac{\beta}{\gamma}$, i.e.

- expected number of close contacts per day (β)

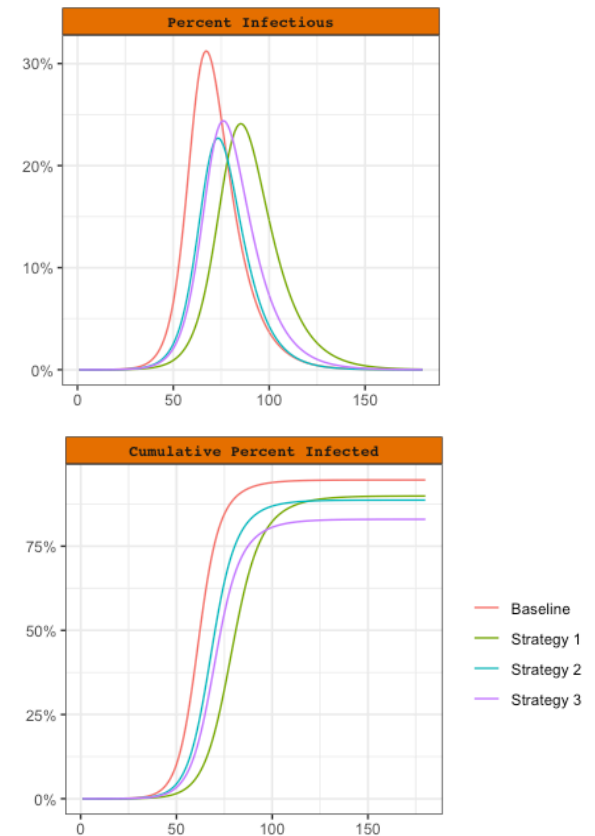
- multiplied by average number of days contagious ($\frac{1}{\gamma}$)

- If $R_0 > 1$ or, equivalently $\beta > \gamma$, likely to have an epidemic on our hands as each infected person infects more than one other on average, who will infect more than one other, and so on (geometric growth) until herd immunity



So what can we do?

- Reduce the transmission rate (decrease β)
 - Pump resources into supporting better practices, such as social distancing, limitations on group sizes, and quarantines to flatten the curve and push the peak down (but really just delays)
- Reduce the length of time that someone is contagious (increase γ)
 - Pump resources into supporting recovery, such as medications (e.g. Remdesivir), supplements, nutrition, exercise and so on, and can have an important effect on both peak infections and total number infected
- Reduce the number of susceptible people (decrease S_1)
 - Pump resources into supporting vaccination, which reduces peak infections but also results in largest reduction in total infections



Compartmental Models

- This model assumes:
 - **Infinite time**: Every individual will be infected eventually
 - **Homogenous nodes**: Each node has same probability to transfer/be transferred the e.g. disease, innovation, or information to neighbours
 - **Homogenous mixing**: Or at least random distribution
 - **Closed population**: No birth, death, or mobility

But what about networks?

- Homogenous mixing an important assumption in compartment modelling...
- But real-life networks rarely uniform topology!
- TBD:
 - **Vital dynamics**: new nodes may enter, with fewer ties and older nodes may perish, taking ties with them
 - **Serosorting**: process by which tie probability is linked to disease status



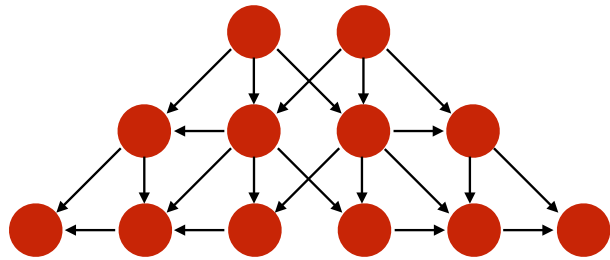
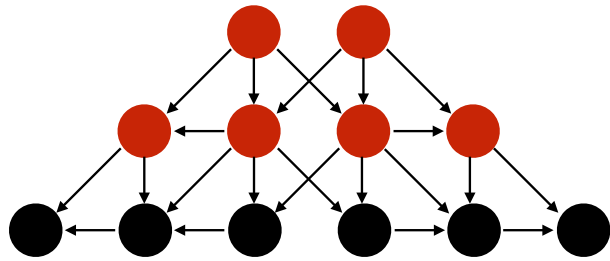
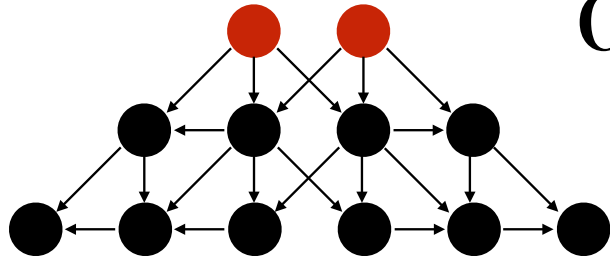
Lesson #2

Networks can spread bad stuff like disease, but also (and simultaneously) good stuff like practices that reduce disease

“Ideas and products and messages and behaviors spread like viruses do.”

–Malcolm Gladwell, *The Tipping Point*

Simple vs complex diffusion

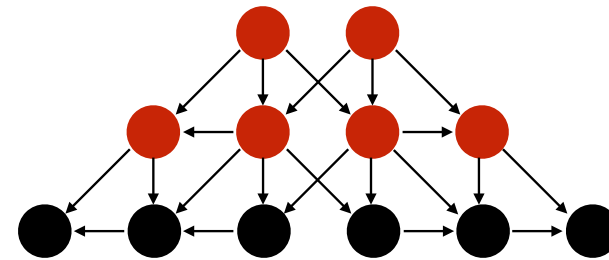
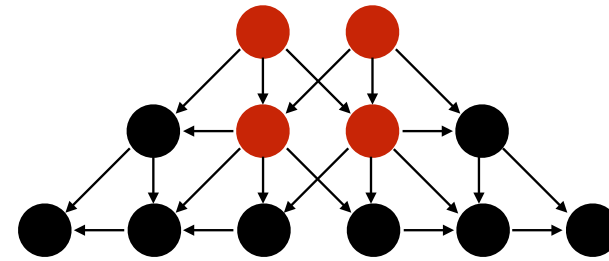
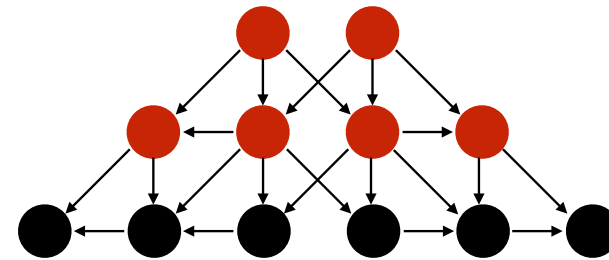
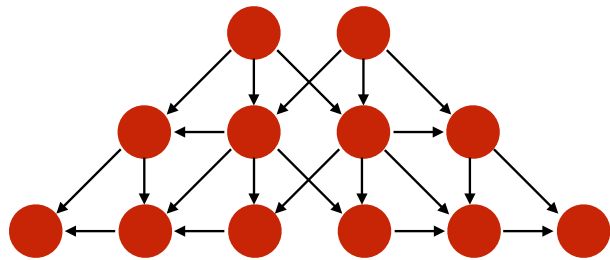
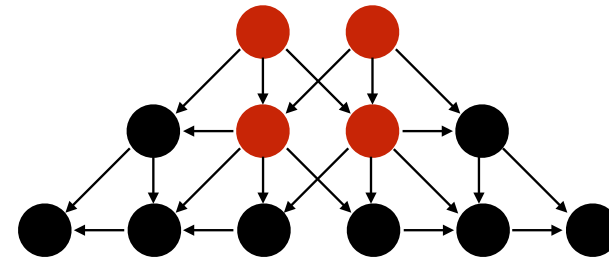
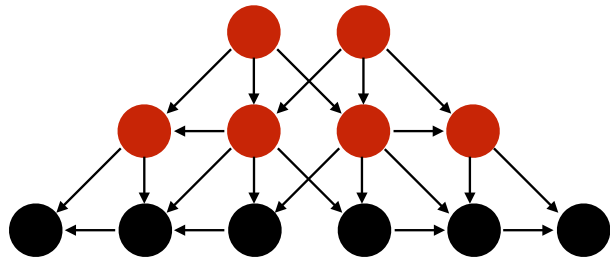
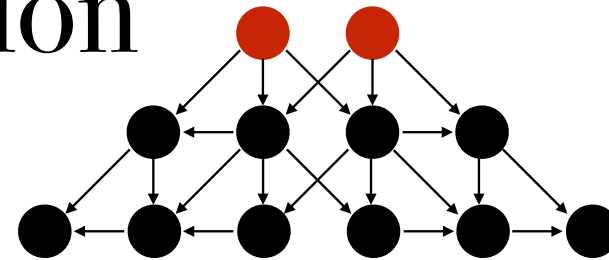
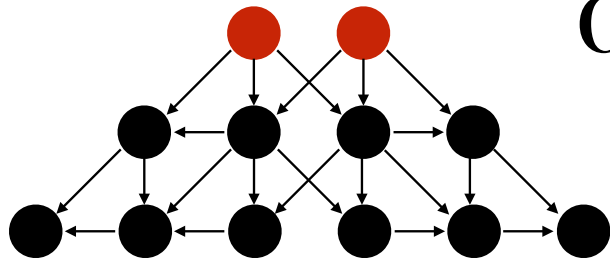


Exposure \rightarrow infection/adoption



But thresholds?

Simple vs complex diffusion

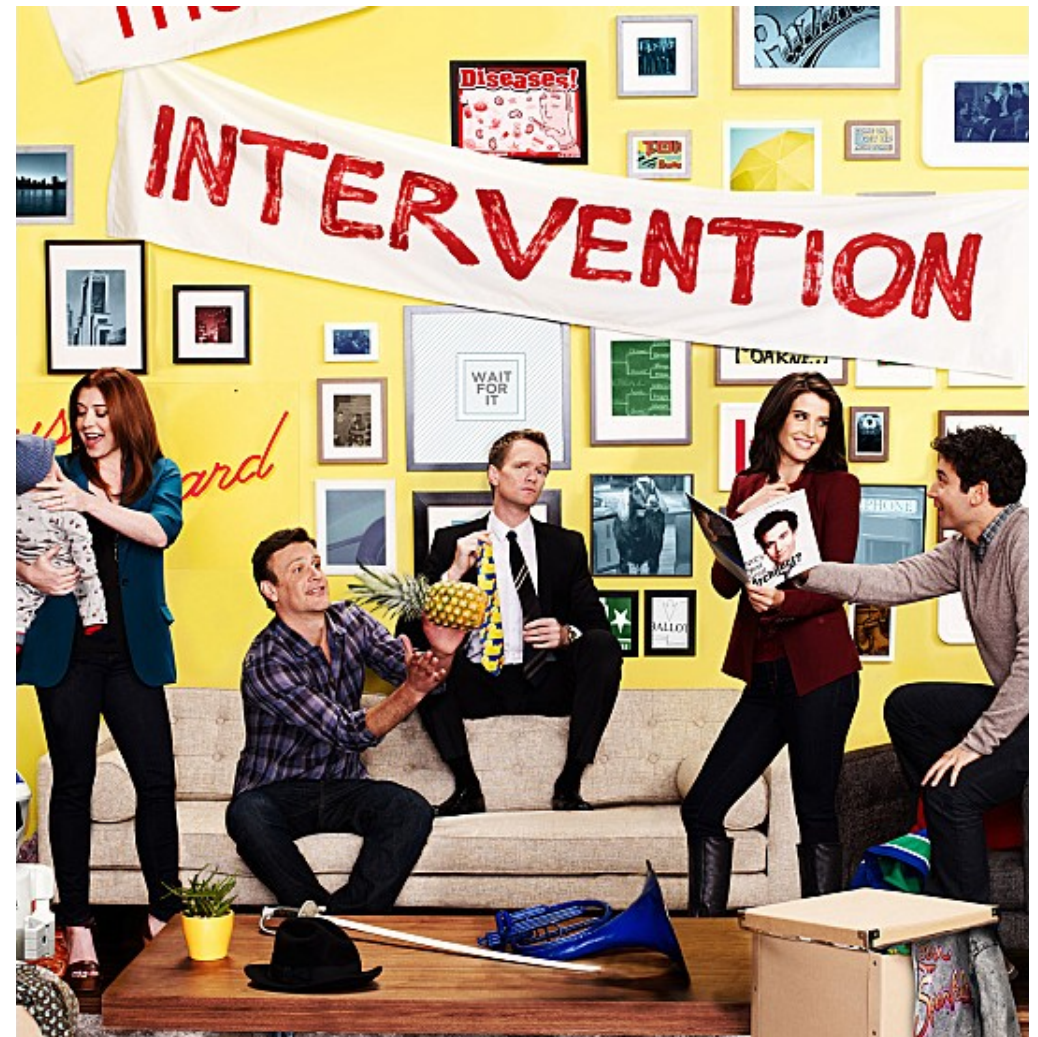


Thresholds

- This transmission rate can be counteracted by a **threshold**
 - When a balance between acting and not acting is overcome in response to a combination of internal propensities and the influence of external events, such as the perception that “everyone is doing it”
 - Can mean various things in different networks
- These thresholds could:
 - Be uniform, or the same across all nodes
 - Vary, perhaps based on some salient attribute
 - Be structural, or be based on their local neighbourhood
 - Vary over time, or change based on overall prevalence (tbi)

Network

Network interventions literature uses network data to assist behavioural change programs by identifying specific nodes to deliver or receive a treatment so as to accelerate or decelerate behaviour change or improve performance



So seed clusters?

- Targeting individuals in specific subgroups may amplify the initial effect rather than spreading resources more broadly, but:
 - How many is enough?
 - Could such a strategy lead to polarisation due to group identities being associated with technology adoption?
 - Which groups should be selected for targeting?

Naïveté of Threshold Models

- *Structurally diverse social reinforcement*
 - Exposure to information from multiple sources has a larger effect when sources independent (unconnected)
 - People more likely to join Facebook when invited from friends that are themselves *not connected* (possibly different groups of friends, colleagues, etc) (Ugander et al. 2012)
 - Similarly, people more likely to donate to a political campaign when exposed to earlier donors from different groups, rather than from a single group (Traag 2016)

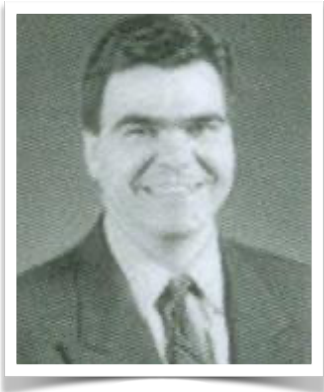
Lesson # 1

Not all nodes are equal, and
not all nodes are positioned
equally



Learning



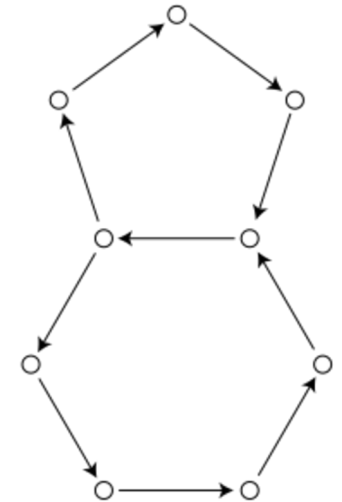


DeGroot Learning Model

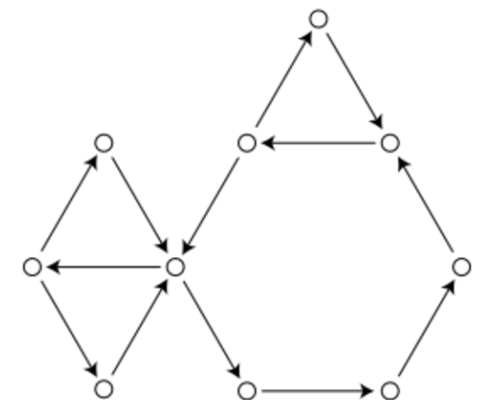
- Developed by John French and Frank Harary, but formalised by Morris De Groot
 - Compared to Bayesian learning model
- Initialisation
 - n nodes have beliefs with respect to some subject, represented as a vector of probabilities e.g. $\{p_1(0), \dots, p_n(0)\}$
 - No new information is introduced, but they communicate/update based on how they 'trust' other actors
 - A 'trust matrix' T is a type of Markov matrix that indicates the weight each i actor puts on j 's opinion
 - directed (typically)
 - weighted
 - (right) stochastic, i.e. non-negative cells as probabilities that (row) sum to 1
 - Nodes can also trust themselves such that $T_{ii} > 0$, or even not listen to anyone else such that $T_{ii} = 1$
- At each step, each node's beliefs are updated
$$p_i(t) = \sum_j T_{ij} p_j(t-1)$$
- What happens? Do beliefs converge? To a single value (consensus)?

Whether beliefs will converge

- An important question is whether varying beliefs will converge over the long run
- Since trust matrix *stochastic*, Markov chain theory (see e.g. Jackson 2008; Golub and Jackson 2010) means that belief convergence will occur where:
 - The network is *strongly connected*
 - The network is *aperiodic*
 - There is a unique eigenvector corresponding to eigenvalue 1
- A network that isn't strongly connected but where each component is strongly connected and aperiodic will still get belief convergence and consensus within each group

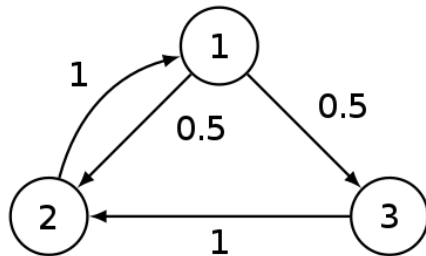


Strongly connected, aperiodic graph
(Cycles of 5 and 6 have no common divisor greater than 1)

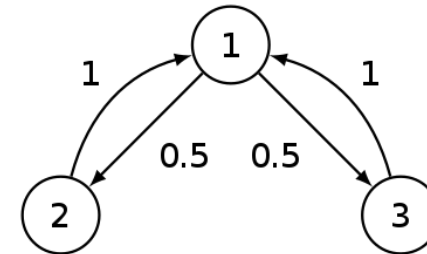


Strongly connected, but with period = 3

Examples



- Here node 1 weights beliefs of other two equally, node 2 only listens to node 1, and node 3 only listens to node 2
- The influence vector (eigenvector) is $(2/5, 2/5, 1/5)$
- So, independent of initial beliefs, individuals reach a consensus where initial beliefs of node 1 and node 2 (whatever they might be) have twice as much influence as node 3



- Here the same structure except that node 3 now only listens to node 1 (instead of node 2, and similarly to node 2)
- The limit does not exist because the network is *periodic*
- Node 1 is updating based on node 2 and node 3's beliefs, but nodes 2 and 3 update based on node 1's belief
- So unless they all have the same initial beliefs, they will just interchange their beliefs at each iteration

Convergent conclusions

- So both the structure of the network and the distribution of the initial beliefs are important for what the convergence/consensus is
- Weighted averaging provides a nice linear system
- Can identify those nodes with most influence over the final result...
- Can identify the speed of convergence
- Extensions from beliefs to actions, multinomial beliefs, etc.
- Constraints e.g. on listening to neighbours with values not too dissimilar from own

Lesson #2

Galton's problem: difficulty of distinguishing contagion and heterogeneity in most data...

