

Social and Technological Network Analysis

Lecture 8: Epidemics Spreading

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In This Lecture

- In this lecture we introduce the process of spreading epidemics in networks.
 - This has been studied widely in biology.
 - But it also has important parallels in information/ idea diffusion in networks.





Epidemics vs Cascade Spreading

- In cascade spreading nodes make decisions based on pay-off benefits of adopting one strategy or the other.
- In epidemic spreading
 - Lack of decision making.
 - Process of contagion is complex and unobservable
 - In some cases it involves (or can be modeled as randomness).





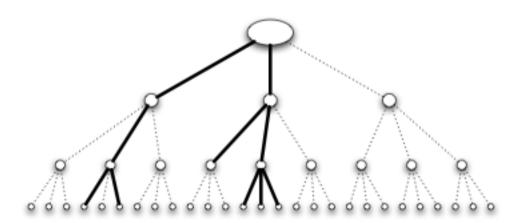
Branching Process

- Simple model.
- **First wave:** A person carrying a disease enters the population and transmit to all he meets with probability p. He meets k people: a portion of which will be infected.
- Second wave: each of the k people goes and meet k different people. So we have a second wave of kxk=k² people.
- Subsequent waves: same process.





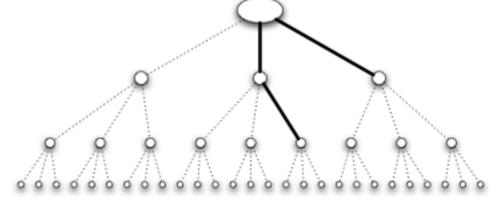
Example with k=3



High contagion probability: The disease spreads

Low contagion probability: The disease dies out







Basic Reproductive Number

- Basic Reproductive Number R₀=p*k
 - It determines it the disease will spread or die out.
- In the branching process model, if R_0 <1 the disease will die out after a finite number of waves. If R_0 >1, with probability >0, the disease will persist by infecting at least one person in each wave.







- When R₀ is close 1, slightly changing p or k can result in epidemics dying out or happening.
 - Quarantining people/nodes reduces k.
 - Encouraging better sanitary practices reduces germs spreading [reducing p].
- Limitations of this model:
 - No realistic contact networks: no triangles!
 - Nodes can infect only once.
 - No nodes recover.

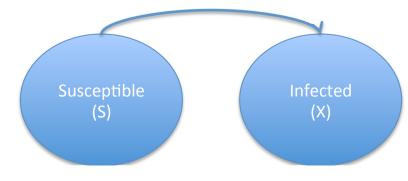


Formal Epidemics Models The SI Model



- S: susceptible individuals.
- X: infected individuals, when infected they can infect others continuously.
- n: total population.
- <k> average contacts per individual
- $\beta=\lambda< k>$ is the infection rate per individual ($0\leq\lambda\leq 1$)
- Susceptible contacts per unit of time βS/n.
- Overall rate of infection XβS/n.





SI Model



$$\frac{dX}{dt} = \beta \frac{SX}{n}$$

$$\frac{dS}{dt} = -\beta \frac{SX}{n}$$

$$s = \frac{S}{n} \qquad x = \frac{X}{n}$$

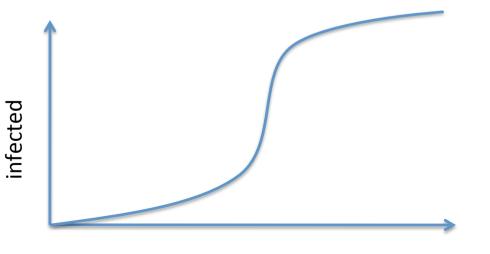
$$s = 1 - x$$

$$\frac{dx}{dt} = \beta x (1 - x)$$



$$x(t) = \frac{x_0 e^{\beta t}}{1 - x_0 + x_0 e^{\beta t}}$$

Logistic Growth Equation



SIR Model



- Infected nodes recover at a rate γ.
- A node stays infected for τ time.
- Branching process is SIR with τ =1. $\frac{ds}{dt} = -\beta sx$

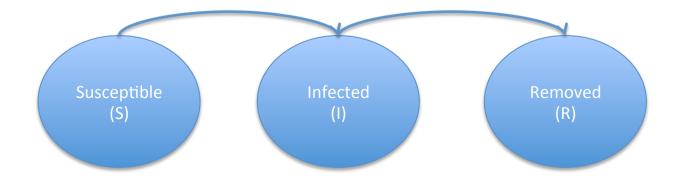
$$\frac{ds}{dt} = -\beta sx$$

$$\frac{dx}{dt} = \beta sx - \gamma x$$

$$\frac{dr}{dt} = \gamma x$$

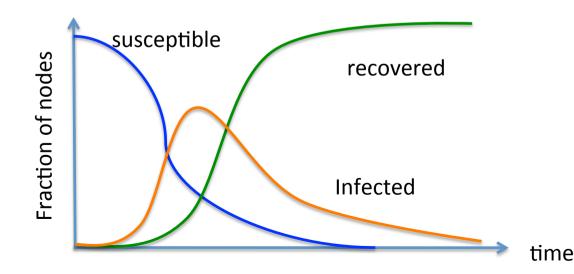
$$s + x + r = 1$$





Example

- Numerical examples of solution:
- β=1, γ=0.4, s(at start)=0.99, x(at start)=0.01, r (at start)=0









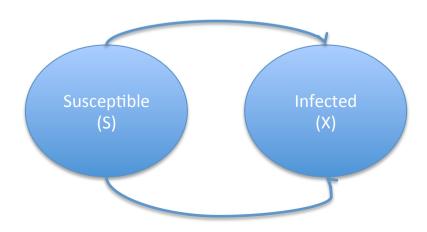
$$\frac{ds}{dt} = \gamma x - \beta sx$$

$$\frac{dx}{dt} = \beta sx - \gamma x$$

$$s + x = 1$$

- •If $\beta > \gamma$ growth curve like in SI but never reaching all population infected. The fraction of infected->0 as β approaches γ .
- •If β < γ the infection will die out exponentially.
- •SIS has the same R_0 as SIR.







Epidemic Threshold

- When would the epidemic develop and when would it die out?
- It depends on the relationship of β and γ :
 - Basic Reproductive Number $R_0 = \beta/\gamma$
 - If the infection rate [per unit of time] is higher than the removal rate the infection will survive otherwise it will die out.
 - In SI, γ =0 so the epidemics always happen.





Limitations of SIR

- Contagion probability is uniform and "on-off"
- Extensions
 - Probability q of recovering in each step.
 - Infected state divided into intermediate states (early, middle and final infection times) with varying probability during each.
 - We have assumed homogenous mixing: assumes all nodes encounter each others with same probability: we could assume different probability per encounter.





Relaxing Assumptions

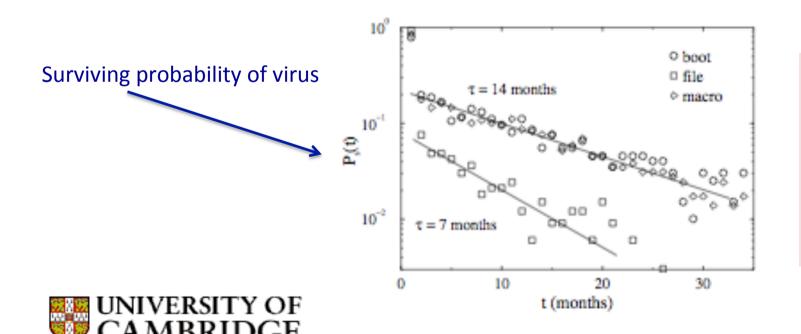
- Homogeneous Mixing: a node connects to the same average number of other nodes as any other.
- Most real networks are not Erdos-Renyi random networks (for which the homogeneous mixing assumption holds).
- Most networks have heterogeneous degree distributions.
 - Scale free networks!





Would the model apply to SF?

 Pastor-Satorras and Vespignani [2001] have considered the life of computer viruses over time on the Internet:



Virus survived on average 6-9/14 months depending on type



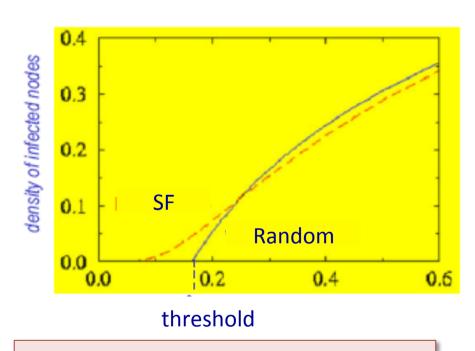


- The virus survival time is considerably high with respect to the results of epidemic models of spreading/recovering:
 - Something wrong with the epidemic threshold!
- Experiment: SIS over a generated Scale Free network (exponent -3).

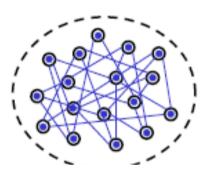


No Epidemic Threshold for SF!

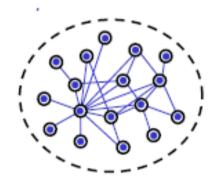




Infections proliferate in SF networks independently of their spreading rates!



Random Network



Scale Free Network



Following result on Immunization

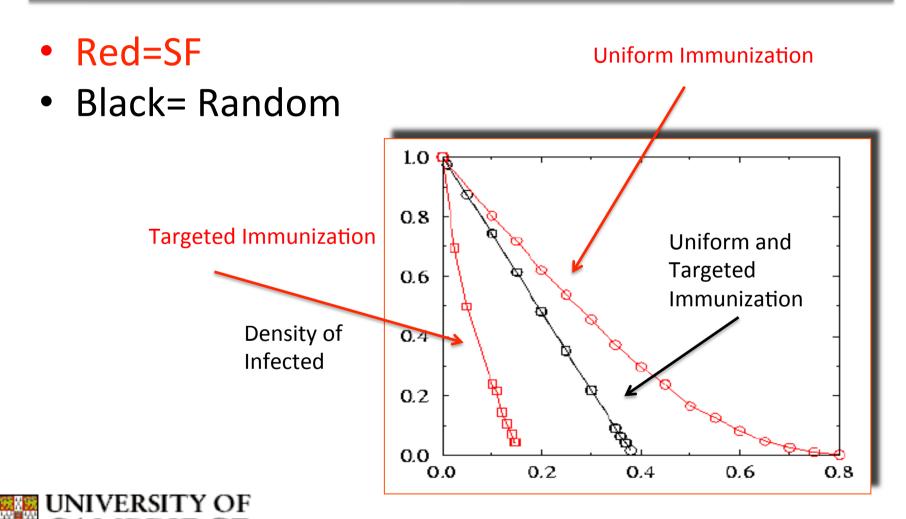


- Random network can be immunized with some sort of uniform immunization process [oblivious of the characteristics of nodes].
- This does not work in SF networks no matter how many nodes are immunized [unless it is all of them].
- Targeted immunization needs to be applied
 - Keeping into account degree!



Immunization on SF Networks





Fraction of immunized nodes



Local Immunization

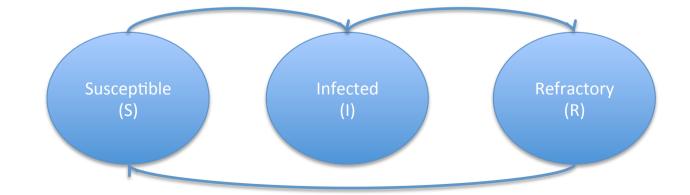
- Global knowledge on the network structure is rarely (or never) available
- Local immunization strategy:
 - Select g nodes at random
 - Ask to each of them to pass over the vaccine to one of their neighbors
 - As a result, a node with degree k is immunized with a probability kP(k) (hubs are immunized with higher probability!)





SIRS Model

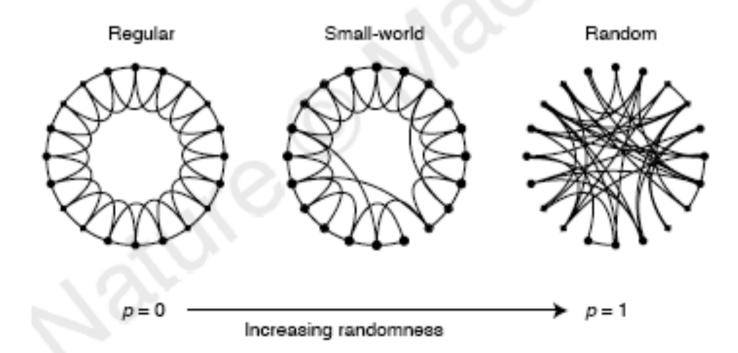
- SIR but after some time an R node can become susceptible again.
- A number of epidemics spread in this manner (remaining latent for a while and having bursts).





Application of SIRS to Small World Models



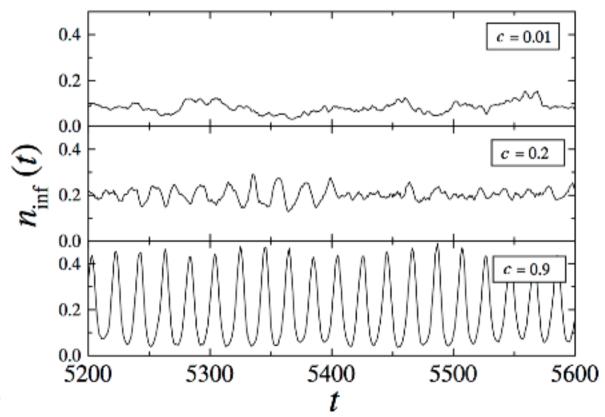






Numerical Results

c is the rewiring probability







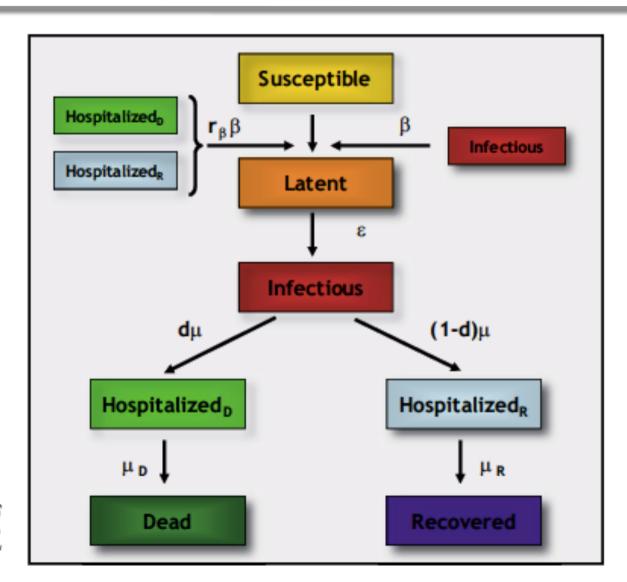
SARS Prediction

- SARS: severe acute respiratory syndrome
- SIR like model with more parameters and homogenous mixing
- Travel data and census data
- WHO data about SARS spreading to evaluate the model
- Outbreak starts in Hong Kong





Epidemics Flow









• Parameters used:

Parameter	Description		Baseline value
T ₀	Initial offset from 21 February (days)		3*
β	Rate of transmission		0.57*
L(t = 0)	Number of initial latent individuals		10*
		21 February + T ₀ -20 March	1.00
s _f (t)	Scaling factor for the rate of transmission	21 March – 9 April	0.37
	· ·	10 April – 11 July	0.06
r _β	Relative infectiousness of patients at the hospital		0.2
اَجَ	Average latency period (days)		4.6
		21 February + T ₀ -25 March	4.84
μ ⁻¹ (t)	Average period from onset of symptoms to admission (days)	25 March – I April	3.83
		2 April – I I July	3.67
μ_{R}^{-1}	Average period from admission to recovery (days)		23.5
μ_{D}^{-I}	Average period from admission to death (days)		35.9
d	Case fatality rate		0.2





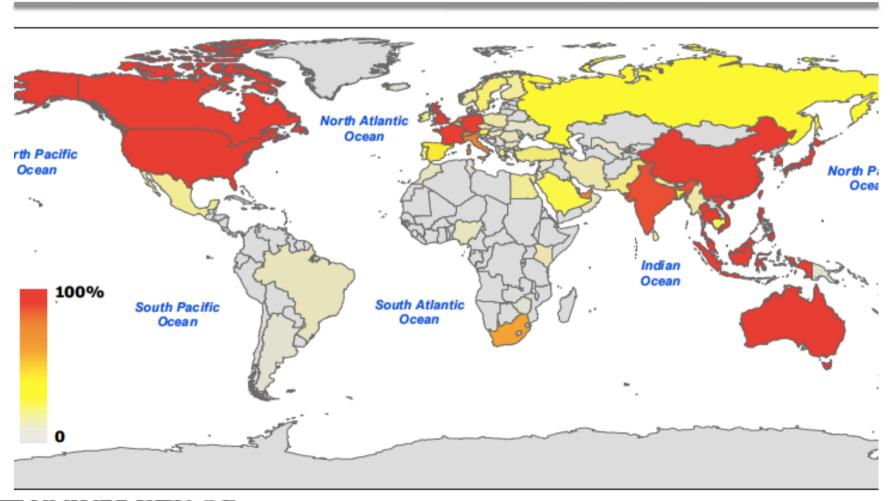


- A model per city
- Probabilities of an individual of moving from one city to the next follows proportions of traffic observed in the air travel data



Predicted Outbreak Likelihood

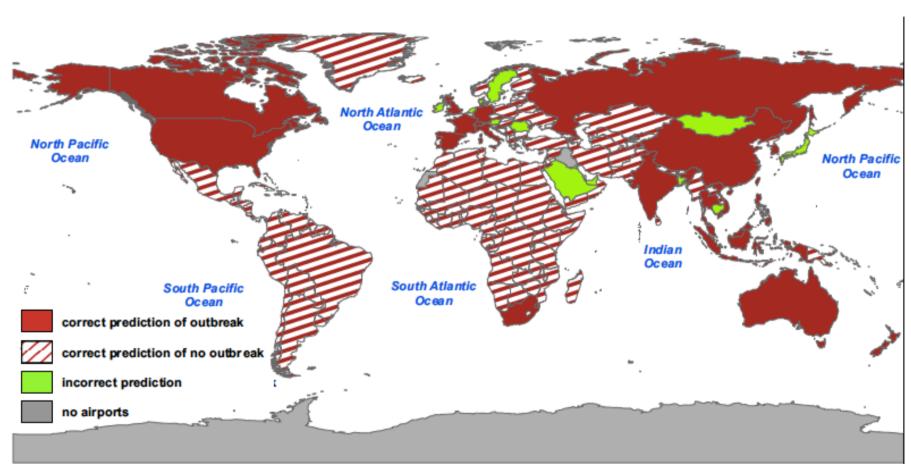








Comparison with Data





SI and Weak Ties (Onnela 2007)

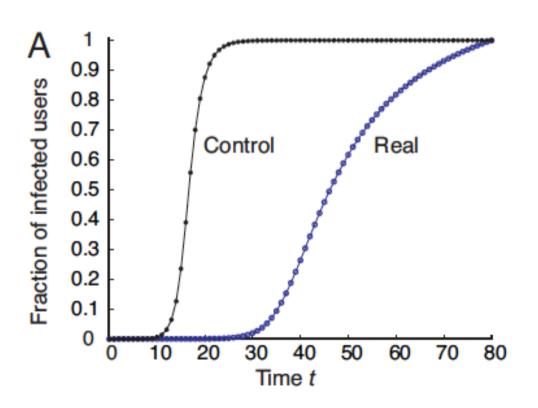


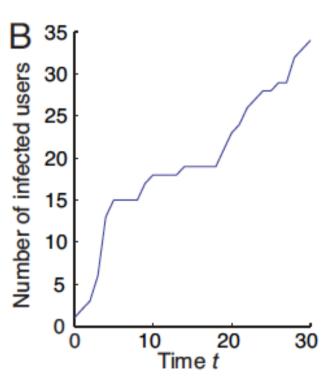
- What is the role of tie strength in information diffusion/epidemic spreading?
- Let us remember the findings in Lecture 2...



SI over Communication Network









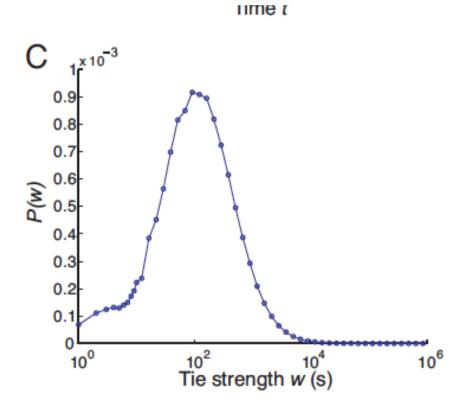


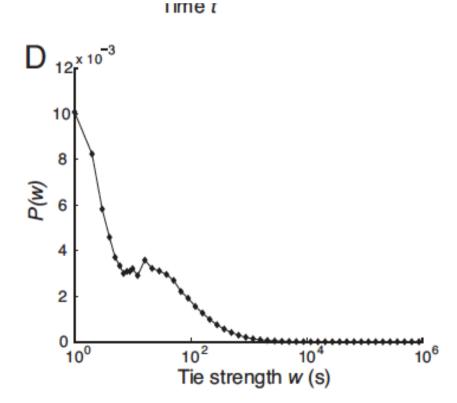
SI and tie strength

Medium ties are the most used:

Weak ties: not enough time to communicate

Strong ties: stuck inside a community





Summary

- Epidemics are very complex processes.
- Existing models have been increasingly capable of capturing their essence.
- However there are still a number of open issues related to the modelling of real disease spreading or information dissemination.
- Mixing geographic factors with epidemics model is very relevant and important.
- Tie strength add another perspective.



References



- Chapter 21
- Pastor-Satorras, R. and Vespignani, A. Epidemic Spreading in Scale-Free Networks. Phys. Rev. Lett.(86), n.14. Pages = 3200--3203. 2001.
- Pastor-Satorras, R. and Vespignani, A. Immunization of Complex Networks. Physical Review E 65. 2002.
- Marcelo Kuperman and Guillermo Abramson. Small world effect in an epidemiological model. Physical Review Letters, 86(13):2909–2912, March 2001.
- V. Colizza, A. Barrat, M. Barthélemy and A. Vespignani. Predictability and epidemic pathways in global outbreaks of infectious diseases: the SARS case study. BMC Medicine 2007, 5:34

