



Social and Technological Network Analysis

Lecture 7: 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



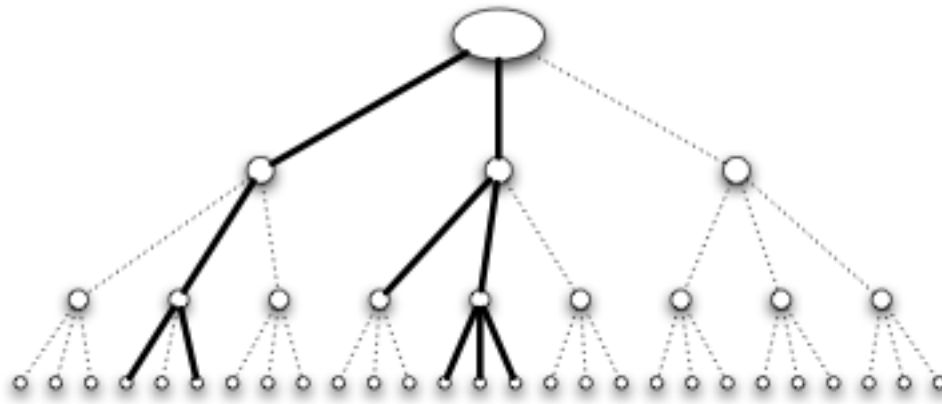
- In cascade spreading nodes make decisions based on pay-off benefits of adopting one strategy or the other.
- In epidemic spreading
 - Lack of decision making.
 - Process of contagion is complex and unobservable
 - In some cases it involves (or can be modeled as randomness).

Branching Process



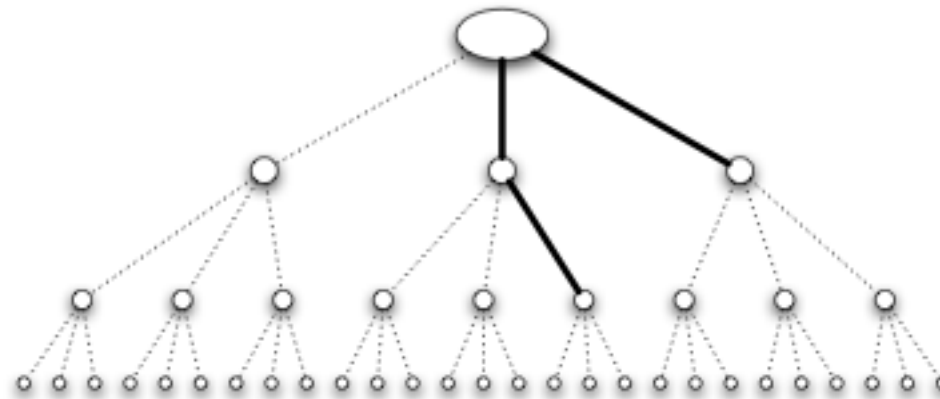
- Simple model.
- **First wave:** A person carrying a disease enters the population and transmits to all he meets with probability p . He meets k people: a portion of which will be infected.
- **Second wave:** each of the k people goes and meets k different people. So we have a second wave of $k \times k = k^2$ 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 $R_0 = p * k$
 - It determines if 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.

Measures to limit the spreading



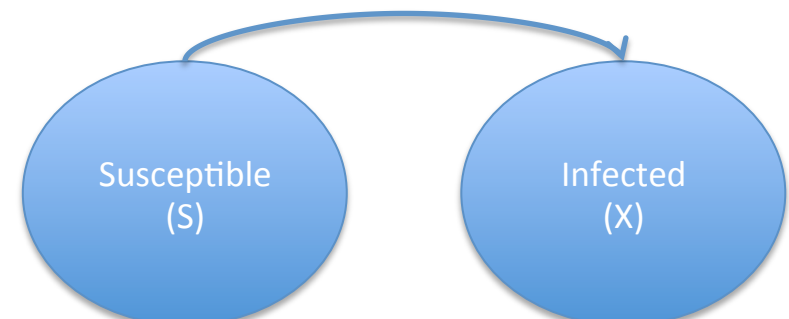
- When R_0 is close 1, slightly changing p or k can result in epidemics dying out or happening.
 - Quarantining people/nodes reduces k .
 - Encouraging better sanitary practices reduces germs spreading [reducing p].
- Limitations of this model:
 - No realistic contact networks: no triangles!
 - Nodes 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 (different from before).
- n: total population.
- β (called k before) is the number of contacts per unit of time of an individual.
- Susceptible contacts per unit of time $\beta S/n$.
- Overall rate of infection $X\beta S/n$.





SI Model

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

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

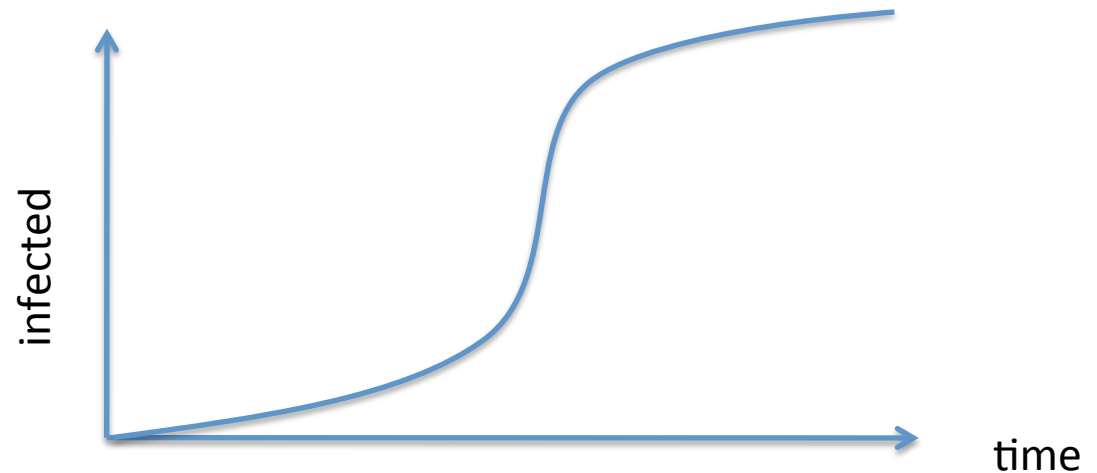
$$s = \frac{S}{n} \quad x = \frac{X}{n}$$

$$s = 1 - x$$

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

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

Logistic Growth Equation





SIR Model

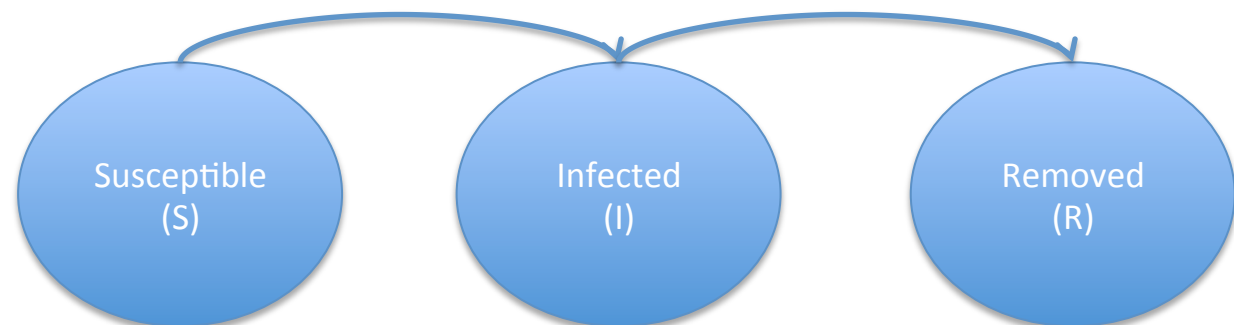
- Infected nodes recover at a rate γ .
- A node stays infected for τ time.
- Branching process is SIR with $\tau=1$.

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

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

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

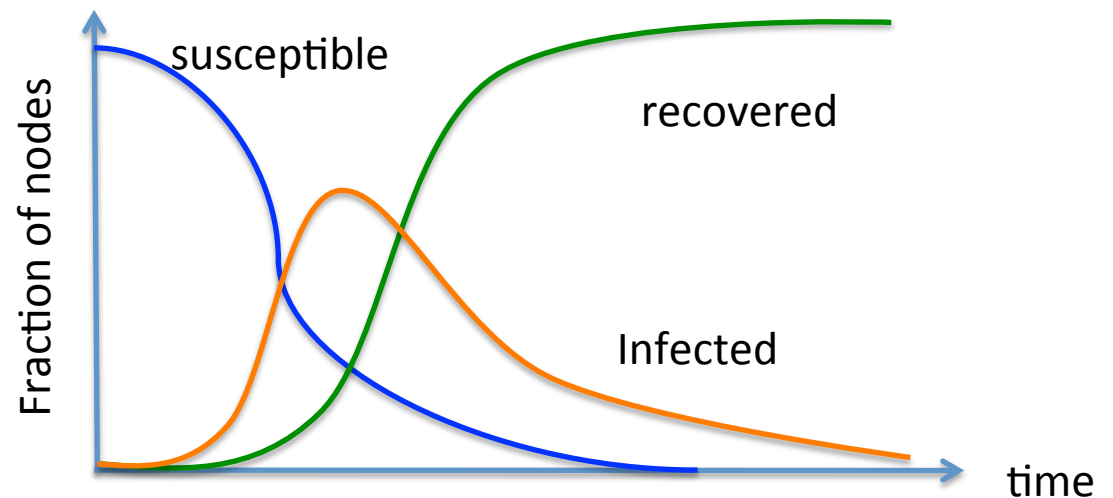
$$s + x + r = 1$$



Example



- The solution to the system is complex
- Numerical examples of solution:
- $\beta=1$, $\gamma=0.4$, $s(\text{at start})=0.99$, $x(\text{at start})=0.01$, r
(at start)=0



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, $\gamma=0$ 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.



SIS Model

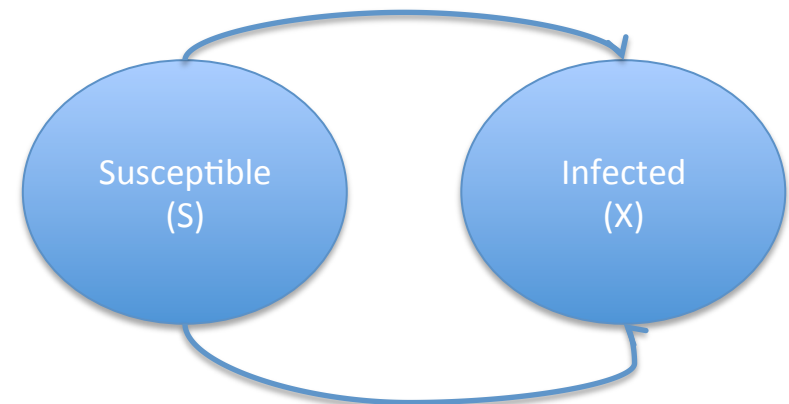
$$\frac{ds}{dt} = \gamma x - \beta sx$$

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

$$s + x = 1$$

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

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





Relaxing Assumptions

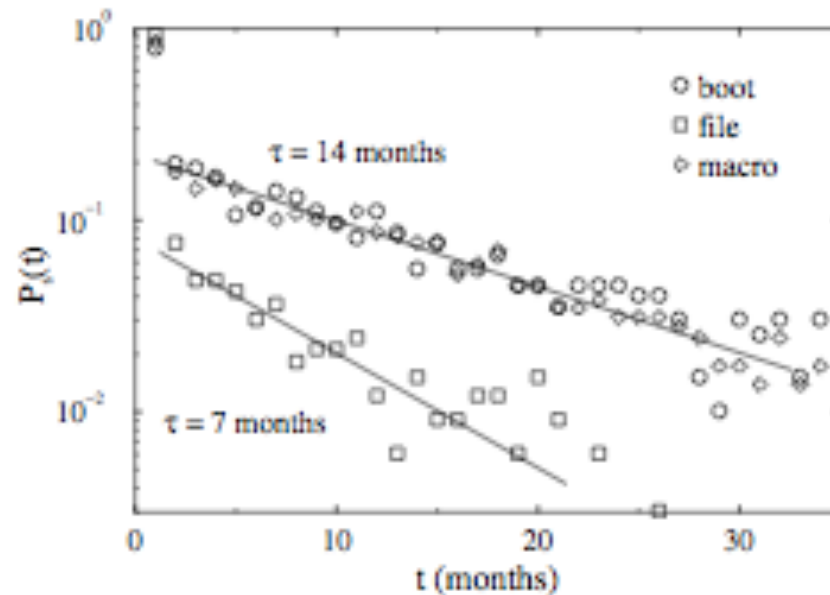
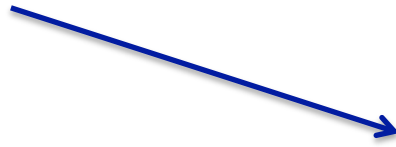
- Homogeneous Mixing: a node connects to the same average number of other nodes as any other.
- Most real networks are not random networks where the homogeneous mixing assumption holds.
- Most networks have different degree distributions.
 - Scale free networks!

Would the model apply to SF?



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

Surviving probability of virus



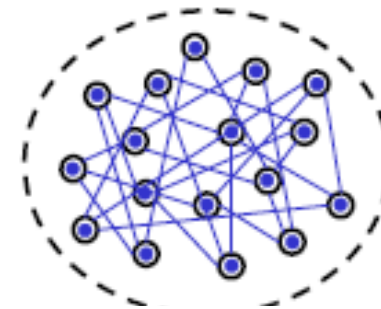
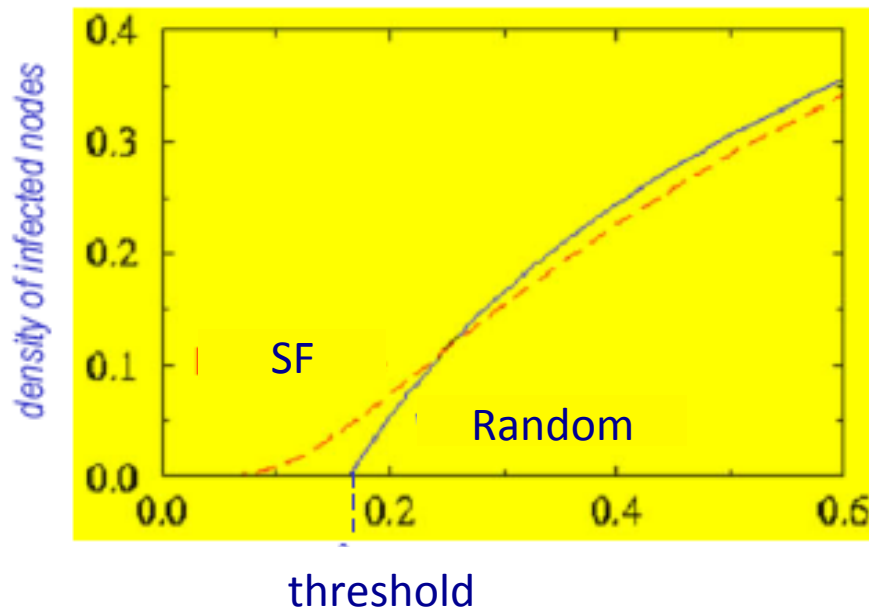
Virus survived on average 6-9/14 months depending on type

How to justify this survival time?

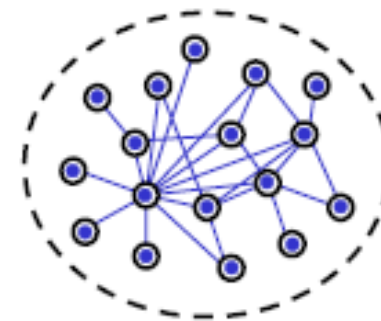


- The virus survival time is considerably high with respect to the results of epidemic models of spreading/recovering:
 - Something wrong with the epidemic threshold!
- Experiment: SIS over a generated Scale Free network (exponent -3).

No Epidemic Threshold for SF!



Random Network



Scale Free Network

Infections proliferate in SF networks independently of their spreading rates!

Following result on Immunization

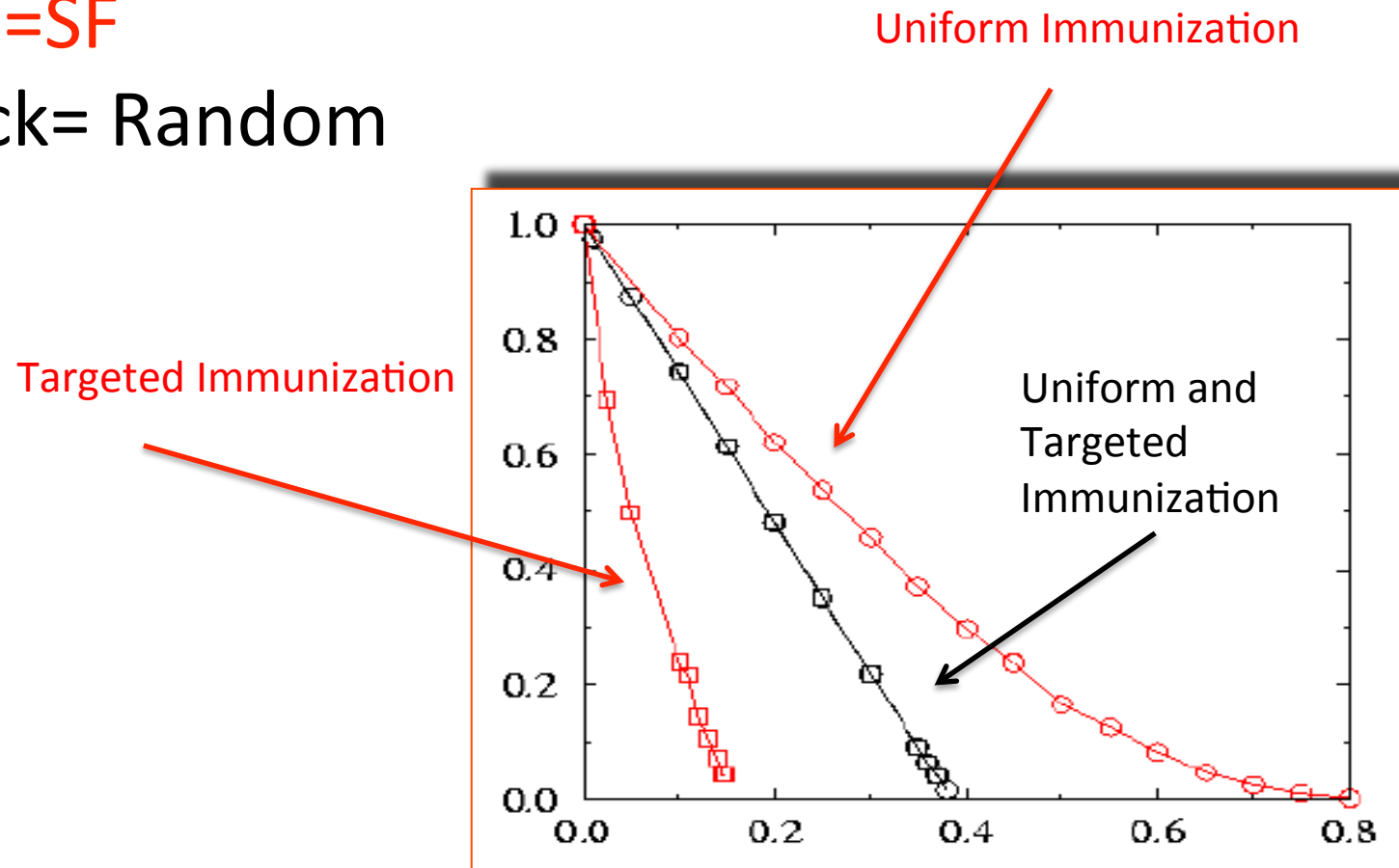


- Random network can be immunized with some sort of uniform immunization process [oblivious of the characteristics of nodes].
- **This does not work in SF networks** no matter how many nodes are immunized [unless it is all of them].
- Targeted immunization needs to be applied
 - Keeping into account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

Immunization on SF Networks



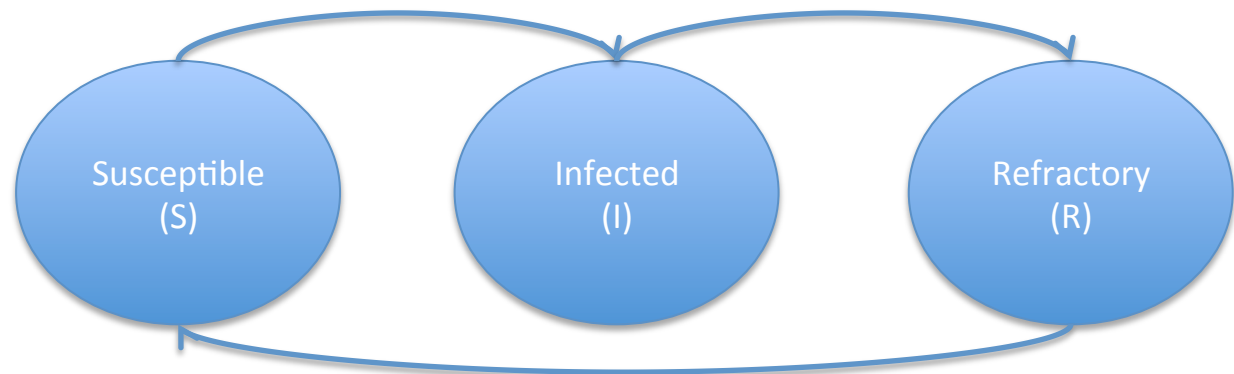
- Red=SF
- Black= Random



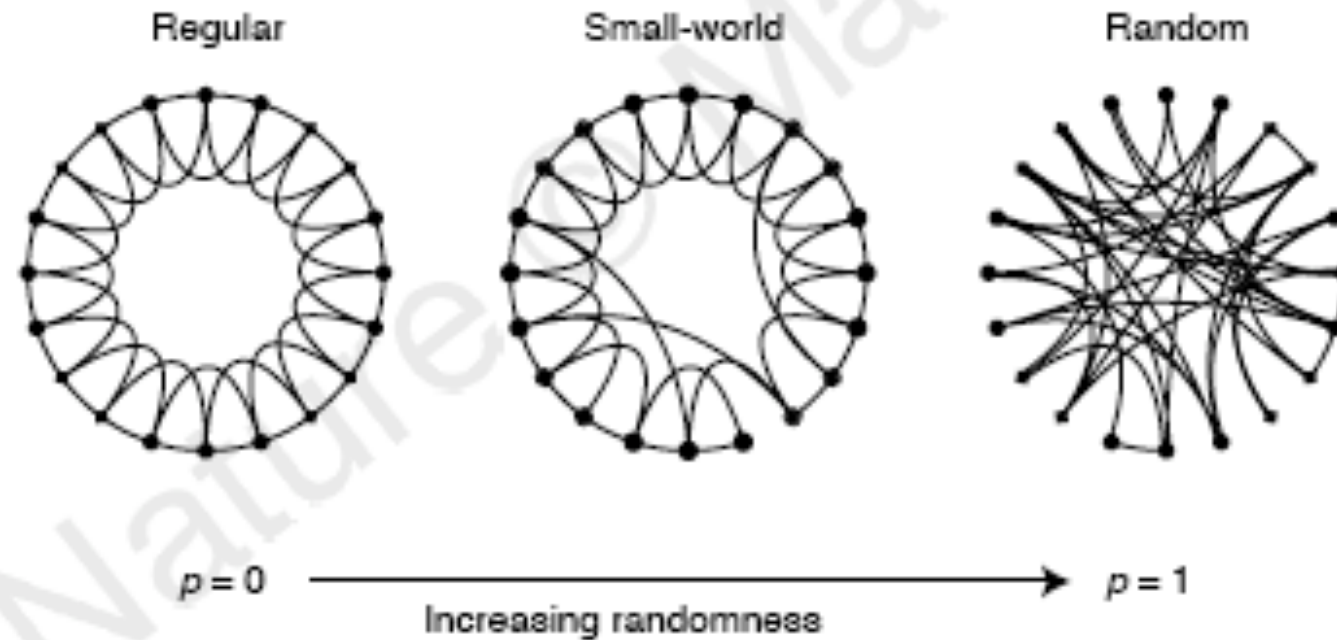


SIRS Model

- 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).



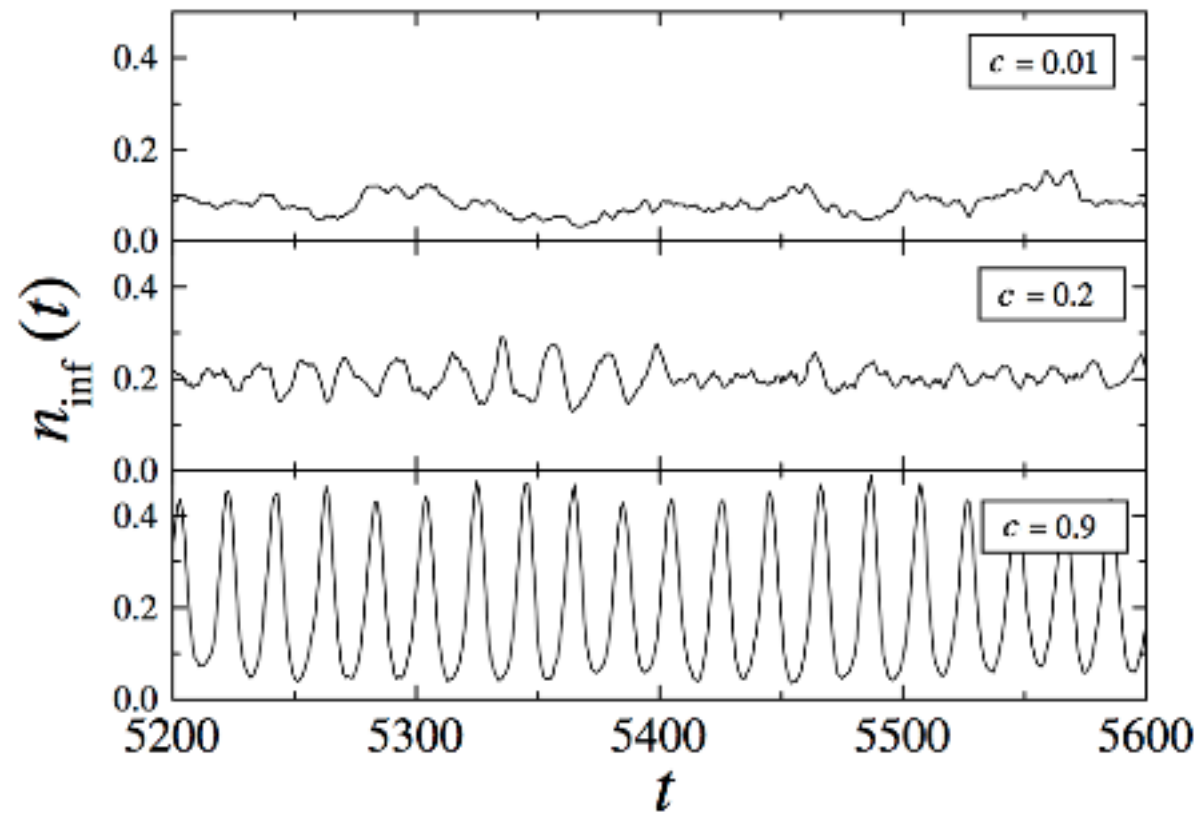
Application of SIRS to Small World Models



Numerical Results



- c is the jumping probability



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.

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.