

Spreading Phenomenon and Epidemic models

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Some slides are taken from Prof. Barabási's class on Network Science (www.BarabasiLab.com)



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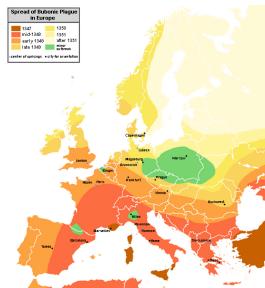
Outline

- Spreading phenomena
- Classical epidemic models
- Network epidemics
- Immunization

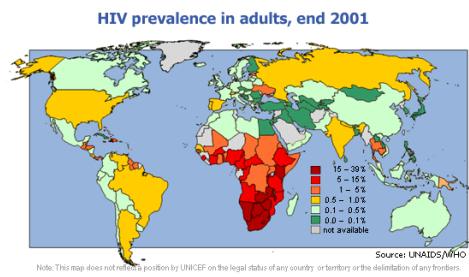


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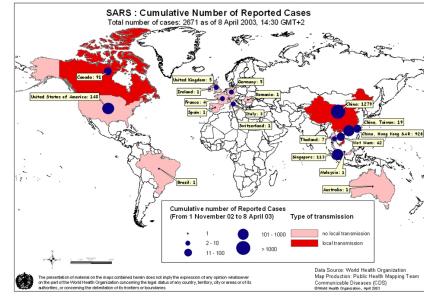
Epidemic outbreaks



The Great Plague



HIV



SARS



1918 Spanish flu



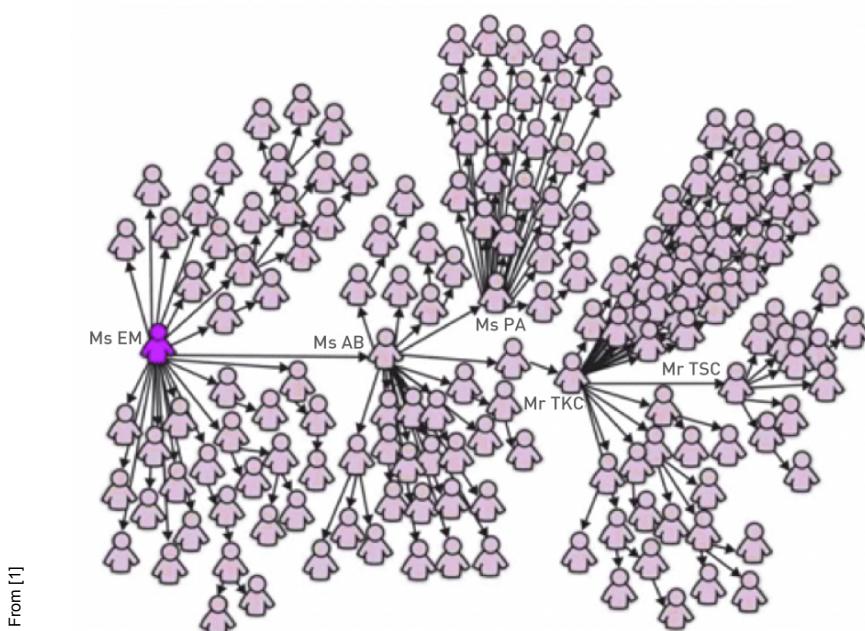
H1N1 flu

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Network influence: SRAS example

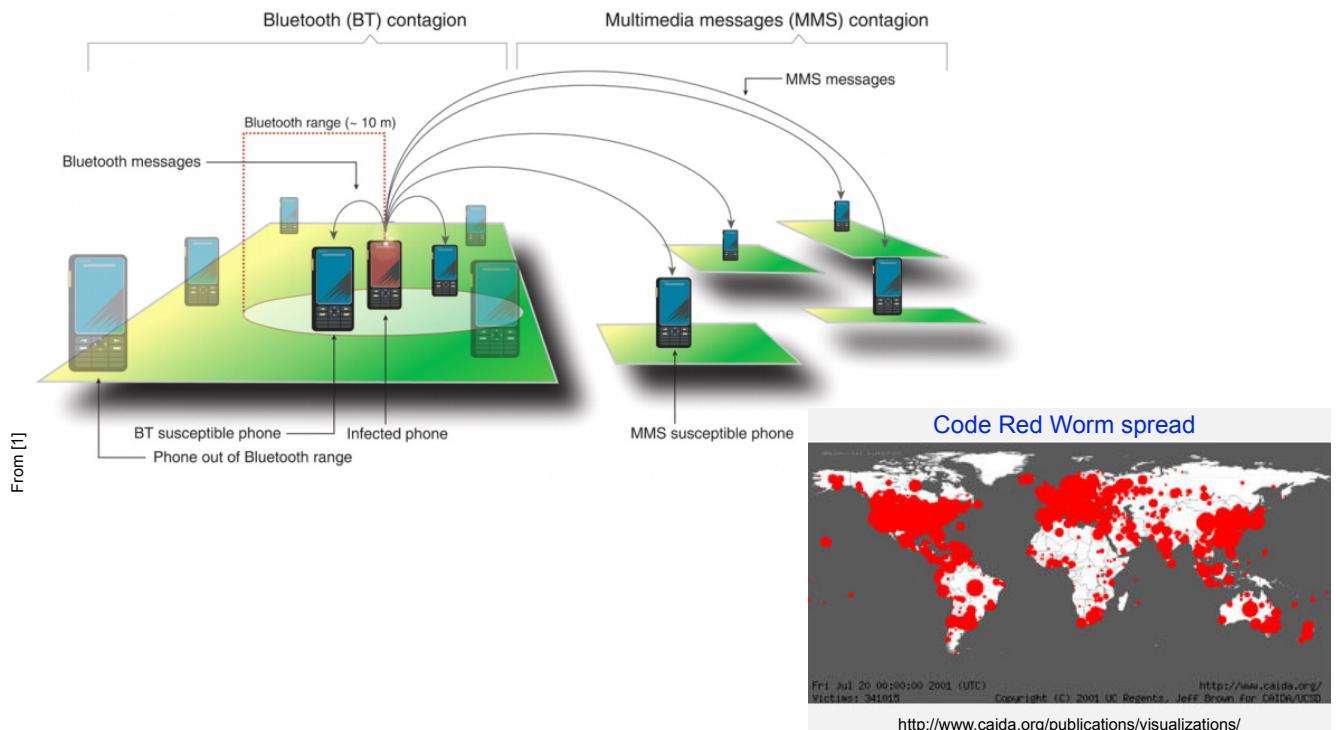


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Digital viruses

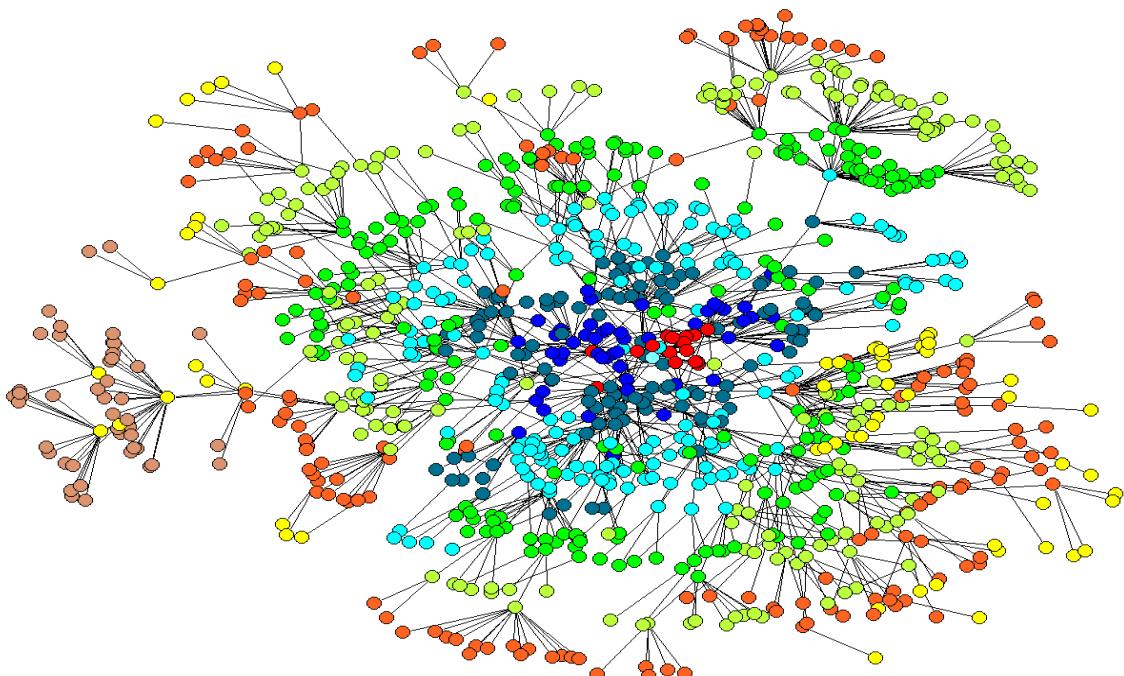


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Information spreading



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Networks and Agents

Phenomena	Agent	Network
Venereal Disease	Pathogens	Sexual Network
Rumor Spreading	Information, Memes	Communication Network
Diffusion of Innovations	Ideas, Knowledge	Communication Network
Computer Viruses	Malwares, Digital viruses	Internet
Mobile Phone Virus	Mobile Viruses	Social Network/Proximity Network
Bedbugs	Parasitic Insects	Hotel - Traveler Network
Malaria	Plasmodium	Mosquito - Human network

From [1]

Epidemic spreading always implies network structure!

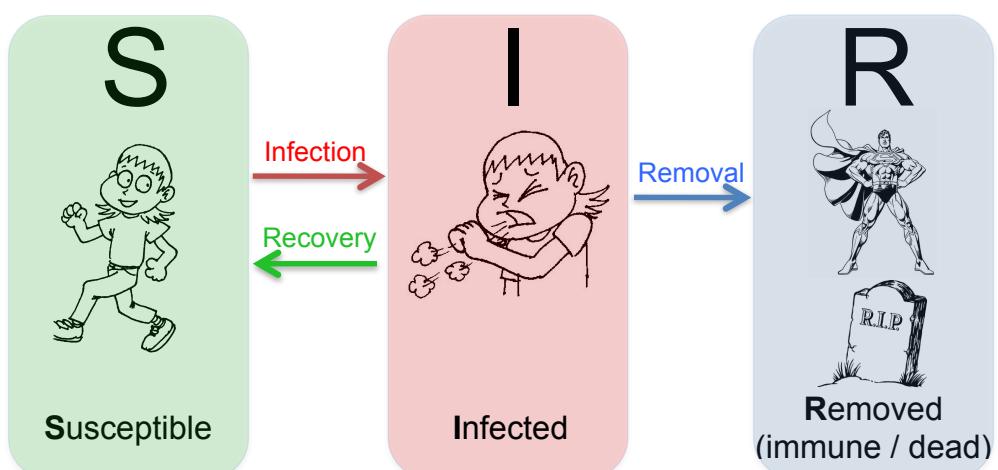


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Classical Epidemic Models

- Epidemiology relies on two fundamental hypotheses
 - Compartmentalization: individuals are in one of 3 states



some diseases require additional states (immune, latent, etc)

- Homogenous Mixing: each individual has the same chance of coming into contact with an infected individual

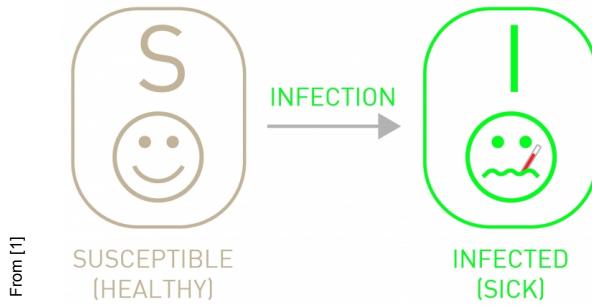
no need to know precise contact network



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SI model



- An individual can be in one of two states: susceptible (healthy) or infected (sick).
 - if a susceptible individual comes into contact with an infected individual, it becomes infected at rate β
 - Once an individual becomes infected, it stays infected, hence it cannot recover.

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SI model analysis

- Number of susceptible and infected individuals at time t : $S(t)$ resp. $I(t)$
- With homogeneous mixing
 - probability for an I individual to meet a S one: $\langle k \rangle S(t)/N$ degree of a typical individual: $\langle k \rangle$
 - average number of new infections in a time interval: $dI(t) = \beta \langle k \rangle \frac{S(t)I(t)}{N} dt$ infection rate: β
- or an equivalent rate of $\frac{dI(t)}{dt} = \beta \langle k \rangle \frac{S(t)I(t)}{N}$
- or $\frac{di}{dt} = \beta \langle k \rangle si = \beta \langle k \rangle i(1 - i)$ transmission rate $s(t) = S(t)/N$ $i(t) = I(t)/N$
- or $\frac{di}{i} + \frac{di}{(1 - i)} = \beta \langle k \rangle dt$
- integrating, we have $\ln i - \ln(1 - i) + C = \beta \langle k \rangle t$
- solving it, we finally have
$$i = \frac{i_0 e^{\beta \langle k \rangle t}}{1 - i_0 + i_0 e^{\beta \langle k \rangle t}}$$

$$\begin{aligned}i_0 &= i(t=0) \\e^C &= (1 - i_0)/i_0\end{aligned}$$

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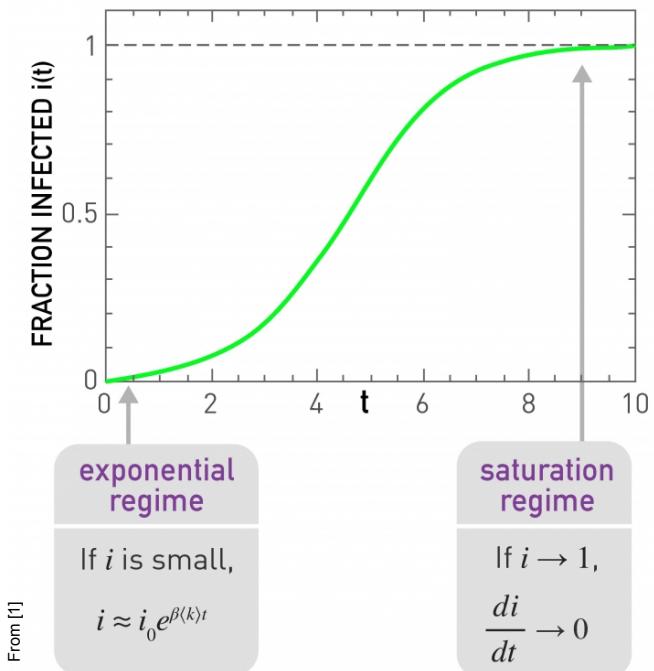
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Infection in SI

- Fraction of infected individuals increased exponentially at the beginning
 - I's meets only S's: fast spread
- Characteristic time

$$\tau = \frac{1}{\beta \langle k \rangle}$$

- Growth slower for large t
 - Less and less S's to be infected
- Infection stops spreading when everyone is infected

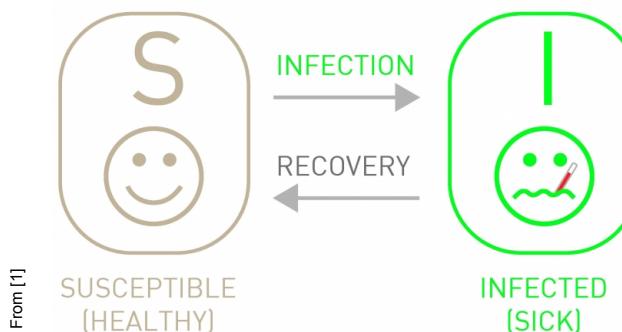


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SIS model



- Individuals can recover at fixed rate μ

$$\frac{di}{dt} = \beta \langle k \rangle i(1 - i) - \boxed{\mu i} \text{ recovery 'rate'}$$

- Again, by integration, the fraction of infected individuals is written as

$$i = \left(1 - \frac{\mu}{\beta \langle k \rangle}\right) \frac{Ce^{(\beta \langle k \rangle - \mu)t}}{1 + Ce^{(\beta \langle k \rangle - \mu)t}}$$

$$i_0 = i(t=0)$$

$$C = i_0 / (1 - i_0 - \mu / \beta \langle k \rangle)$$

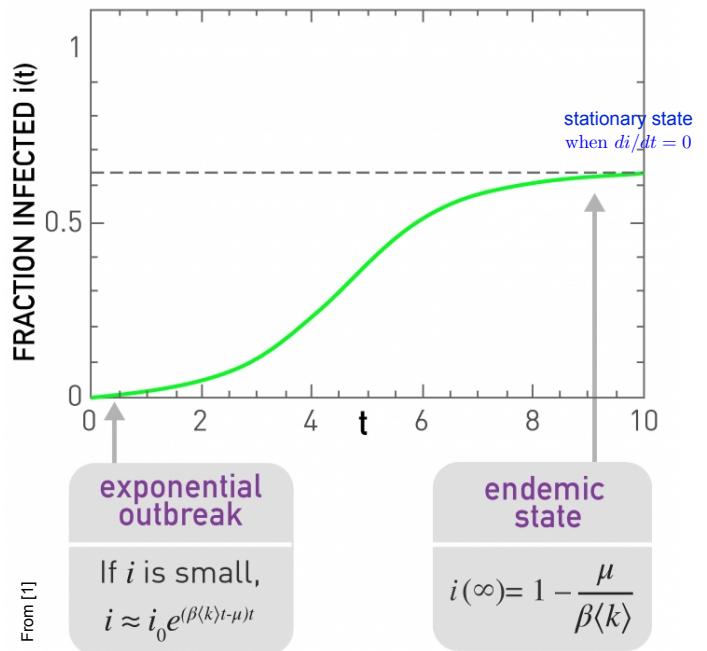
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Infection in SIS

- Recovery becomes possible
 - individuals are cured and become susceptible
 - at large t , endemic state, with constant fraction of infected individuals
 - for high recovery rate μ , the disease dies out



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Epidemic outcomes in SIS

- Endemic state: $\mu < \beta \langle k \rangle$
 - for low recovery rate, the ratio of infected individuals is similar to the SI model
 - in the endemic or stationary state, infection is equal to recovery

$$i(\infty) = 1 - \frac{\mu}{\beta \langle k \rangle} \quad \text{when } di/dt = 0$$

- Disease-free state: $\mu > \beta \langle k \rangle$
 - ratio of infected individuals decays exponentially, disease dies out
 - number of cured individuals larger than the number of infected ones

- Difference between above outcomes is driven by characteristic time τ of the pathogen

$$\tau = \frac{1}{\mu(R_0 - 1)} \quad \text{with} \quad R_0 = \frac{\beta \langle k \rangle}{\mu} \quad (\text{basic reproductive number})$$

- $R_0 > 1$: endemic state, while $R_0 < 1$: disease-free state

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Reproductive numbers

Disease	Transmission	R_0
Measles	Airborne	12-18
Pertussis	Airborne droplet	12-17
Diphtheria	Saliva	6-7
Smallpox	Social contact	5-7
Polio	Fecal-oral route	5-7
Rubella	Airborne droplet	5-7
Mumps	Airborne droplet	4-7
HIV/AIDS	Sexual contact	2-5
SARS	Airborne droplet	2-5
Influenza (1918 strain)	Airborne droplet	2-3

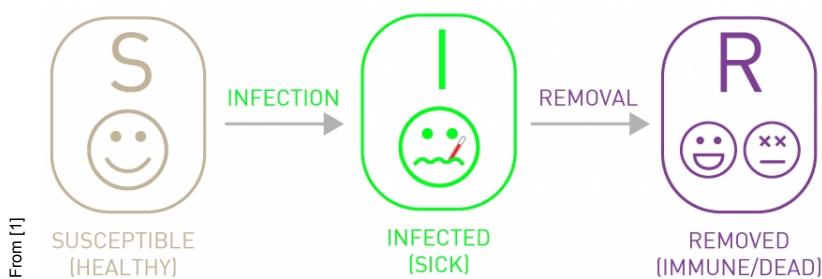
From [1]



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SIR model



- Individuals can develop immunity after recovery with rate μ
 - they cannot be infected nor infect anymore - they do not count any longer
 - time evolution in different states reflects this change:

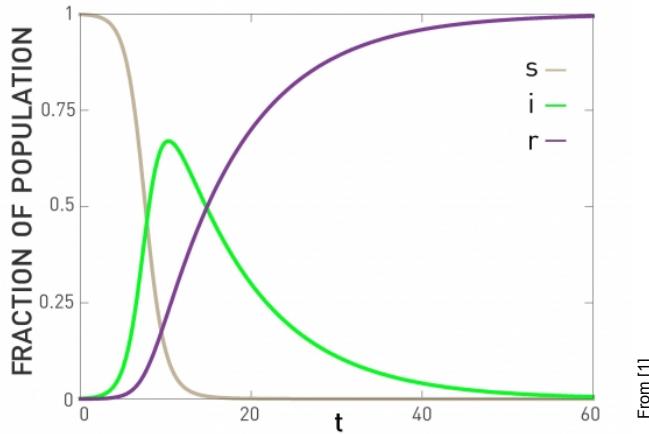
$$\frac{ds}{dt} = -\beta \langle k \rangle i [1 - r - i] \quad \text{\#susceptible}$$
$$\frac{di}{dt} = -\mu i + \beta \langle k \rangle i [1 - r - i]$$
$$\frac{dr}{dt} = \mu i$$



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Infection in SIR



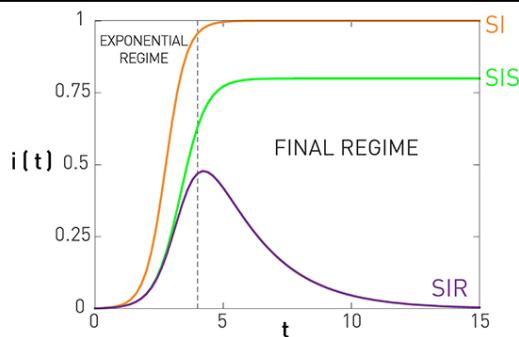
- All individuals transition from a susceptible (healthy) state to the infected (sick) state and then to the recovered (immune) state.

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Regime comparisons



	SI	SIS	SIR
Exponential Regime: Number of infected individuals grows exponentially	$i = \frac{i_0 e^{\beta \langle i \rangle t}}{1 - i_0 + i_0 e^{\beta \langle i \rangle t}}$	$i = \left(1 - \frac{\mu}{\beta \langle k \rangle}\right) \frac{C e^{(\beta \langle i \rangle - \mu)t}}{1 + C e^{(\beta \langle i \rangle - \mu)t}}$	No closed solution

Final Regime: Saturation at $t \rightarrow \infty$	$i(\infty) = 1$	$i(\infty) = 1 - \frac{\mu}{\beta \langle k \rangle}$	$i(\infty) = 0$
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Epidemic Threshold: Disease does not always spread	No threshold	$R_0 = 1$	$R_0 = 1$
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From [1]

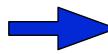
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Network epidemics

- The classical epidemic models do not incorporate the network structure
 - rather based on an homogenous mixing hypothesis and $\langle k \rangle$ for all individuals
- However, the network is important
 - pathogens can spread only on a complex contact network (= through actual contact)
 - many networks are scale-free, hence $\langle k \rangle$ does not provide sufficient information



Network epidemics

R. Pastor-Satorras and A. Vespignani. Epidemic spreading in scalefree networks. Physical Review Letters, 86:3200–3203, 2001.

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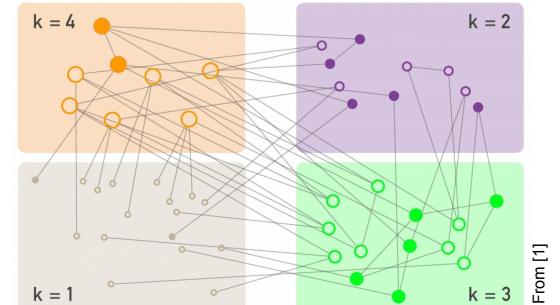
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(Network) SI analysis

- Individual with more links more likely to get in contact with infected individual
 - node degree should be an implicit variable
 - *degree block approximation*: nodes with same degree are statistically equivalent
- Epidemic evolution changes according to degrees
 - fraction of nodes of degree k infected: $i_k = \frac{I_k}{N_k}$
 - total fraction of infected nodes: $i = \sum_k p_k i_k$
 - infection evolution for each degree separately:

$$\frac{di_k}{dt} = \beta(1 - i_k)k\Theta_k$$

non-infected degree- k nodes



Susceptible node with k neighbours, with $\Theta_k(t)$ of the neighbors infected.

dependence on actual node's degree
 Θ_k : fraction of infected neighbours of a susceptible node (depends on k and t)
system of k_{\max} equations

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Density function

- Density function Θ_k : fraction of infected nodes in the neighbourhood of a susceptible node with degree k
- Probability of having a node of degree k' at the end of a randomly chosen link

$$q_{k'} = \frac{k' p_{k'}}{\langle k \rangle} \quad (\text{excess degree})$$

- probability that a link points from a node with degree k to a node with degree k' is independent of k
- The density function becomes

$$\Theta_k = \frac{\sum_{k'} (k' - 1) p_{k'} i_{k'}}{\langle k \rangle} = \Theta \quad \text{w/o degree correlations } \Theta_k \text{ is independent of } k$$

- number of links available for future transmission is $(k'-1)$

- Differentiating, we have

$$\frac{d\Theta}{dt} = \sum_k \frac{(k - 1)p_k}{\langle k \rangle} \frac{di_k}{dt}$$

For SI - actual form changes with epidemic model



Density function in SI

Combining $\frac{di_k}{dt} = \beta(1 - i_k)k\Theta_k$ and $\frac{d\Theta}{dt} = \sum_k \frac{(k - 1)p_k}{\langle k \rangle} \frac{di_k}{dt}$

we can write $\frac{d\Theta}{dt} = \beta \sum_k \frac{(k^2 - k)p_k}{\langle k \rangle} [1 - i_k] \Theta$

or, for early behaviour $\frac{d\Theta}{dt} = \beta \left(\frac{\langle k^2 \rangle}{\langle k \rangle} - 1 \right) \Theta$ $i_k \ll 1$ for small t

Its solution is $\Theta(t) = C e^{t/\tau^{SI}}$ with $\tau^{SI} = \frac{\langle k \rangle}{\beta (\langle k^2 \rangle - \langle k \rangle)}$

characteristic time for SI

With initial conditions $\Theta(t=0) = C = i_0 \frac{\langle k \rangle - 1}{\langle k \rangle}$

We finally have

$$\Theta(t) = i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} e^{t/\tau^{SI}}$$



(Network) SI epidemic analysis

$$\frac{di_k}{dt} = \beta(1 - i_k)k\Theta_k \quad \text{approximated as} \quad \frac{di_k}{dt} \approx \beta k\Theta_k \quad \begin{matrix} & \\ & i_k \text{ small} \end{matrix}$$

With explicit density function: $\frac{di_k}{dt} \approx \beta k i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} e^{t/\tau^{SI}}$

$$\tau^{SI} = \frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)}$$

By integrating, we have $i_k = i_0 \left(1 + \frac{k(\langle k \rangle - 1)}{\langle k^2 \rangle - \langle k \rangle} (e^{t/\tau^{SI}} - 1) \right)$

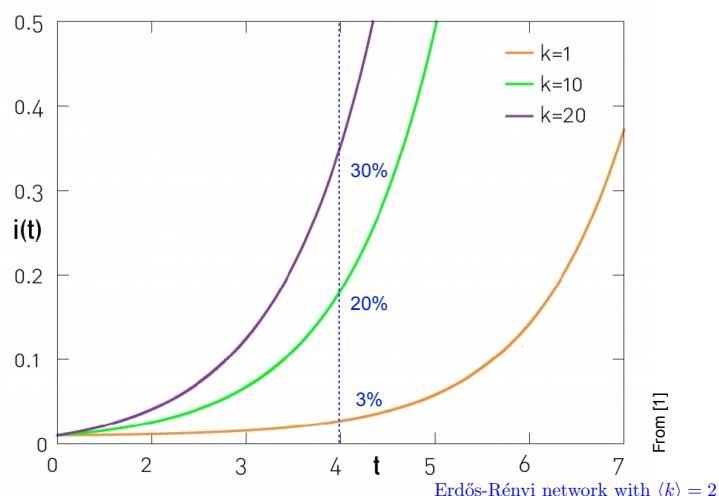
fraction of infected nodes with degree k

- We can see that
 - higher degree k leads to higher infection probability $i_k = g(t) + kf(t)$
 - total fraction of infected nodes grows with time

$$i = \int_0^{k_{\max}} i_k p_k dk = i_0 \left(1 + \frac{\langle k \rangle^2 - \langle k \rangle}{\langle k^2 \rangle - \langle k \rangle} (e^{t/\tau^{SI}} - 1) \right)$$



Infection in (Network) SI



- At any time, the fraction of high degree nodes that are infected is higher than the fraction of low degree nodes.
 - at any time virtually all hubs are infected, but small-degree nodes tend to be disease free
 - disease maintained in the hubs, which in turn broadcast it to the rest of the network



Network influence in SI

- Characteristic time depends on $\langle k^2 \rangle$
- Random network: $\langle k^2 \rangle = \langle k \rangle(\langle k \rangle + 1)$ and $\tau_{ER}^{SI} = \frac{1}{\beta \langle k \rangle}$ similar to earlier homogeneous network result
- Scale-free Network with $\gamma > 3$
 - both $\langle k \rangle$ and $\langle k^2 \rangle$ are finite, so that τ^{SI} is also finite: behaviour similar random network, with altered τ^{SI}
- Scale-free Network with $\gamma \leq 3$
 - $\langle k^2 \rangle$ becomes unbounded for large N , so that $\tau^{SI} \rightarrow 0$
 - the spread of a pathogen on a scale-free network is instantaneous! 
 - the hubs are the first to be infected, and broadcast disease through their numerous links: *super-spreader*
- Inhomogenous Networks
 - scale-free property is not the only structure characteristic that has an impact on spreading
 - as long as $\langle k^2 \rangle > \langle k \rangle(\langle k \rangle + 1)$ then τ^{SI} is reduced
 - heterogenous networks enhance the speed of any pathogen

Density function in SIR

Density of infected nodes in SIR

$$\frac{di_k}{dt} = \beta(1 - i_k - r_k)k\Theta - \mu i_k$$

r_k : fraction of degree-k recovered node

or, for early behaviour

$$\frac{di_k}{dt} = \beta k\Theta - \mu i_k$$

$i_k \ll 1$ and $r_k \ll 1$ for small t

Equivalently, we can write

$$\frac{d\Theta}{dt} = \left(\beta \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle} - \mu \right) \Theta$$

multiplying by $(k-1)p_k/\langle k \rangle$
and summing over k to get

$$\frac{d\Theta}{dt} = \sum_k \frac{(k-1)p_k}{\langle k \rangle} \frac{di_k}{dt}$$

Its solution is

$$\Theta(t) = C e^{t/\tau^{SIR}} \quad \text{with} \quad \tau^{SIR} = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - \langle k \rangle (\beta + \mu)}$$

characteristic time for SIR

Outbreak only if $\tau^{SIR} > 0$ where number of infected nodes grows exponentially

$$\text{in other words, outbreak happens if } \lambda = \frac{\beta}{\mu} > \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}$$

Hence, the epidemic threshold for SIR is

$$\lambda_c = \frac{1}{\frac{\langle k^2 \rangle}{\langle k \rangle} - 1}$$

Density function in SIS

Density of infected nodes in SIS

$$\frac{di_k}{dt} = \beta(1 - i_k)k\Theta - \mu i_k$$

Note that, for SIS, we have

$$\Theta_k = \frac{\sum_{k'} k' p_{k'} i_{k'}}{\langle k \rangle} = \Theta$$

all k links can spread disease
(differently than SI)

For early behaviour, we have

$$\frac{di_k}{dt} = \beta k \Theta - \mu i_k$$

$i_k \ll 1$ for small t

equivalently, we can write

$$\frac{d\Theta}{dt} = \left(\beta \frac{\langle k^2 \rangle}{\langle k \rangle} - \mu \right) \Theta$$

multiplying by $k p_k / \langle k \rangle$
and summing over k to get
 $\frac{d\Theta}{dt} = \sum_k \frac{k p_k}{\langle k \rangle} \frac{di_k}{dt}$

Its solution is $\Theta(t) = C e^{t/\tau^{SIS}}$ with

$$\tau^{SIS} = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - \langle k \rangle \mu}$$

characteristic time for SIS

Outbreak only if $\tau^{SIS} > 0$ where number of infected nodes grows exponentially

$$\text{in other words, outbreak happens if } \lambda = \frac{\beta}{\mu} > \frac{\langle k \rangle}{\langle k^2 \rangle}$$

Hence, the epidemic threshold for SIS is

$$\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}$$

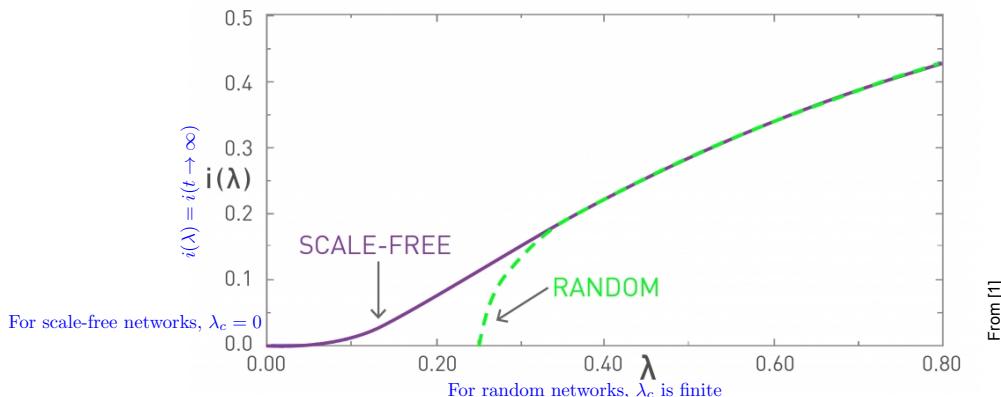


(Network) SIS epidemic analysis

- Dynamics of SIS: $\frac{di_k}{dt} = \beta(1 - i_k)k\Theta_k(t) - \mu i_k$ recovery term simple extension of SI model
- Characteristic time: $\tau^{SIS} = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - \langle k \rangle \mu}$ negative for large μ
 - depends on network heterogeneity, $\langle k^2 \rangle$
 - spreading rate $\lambda = \frac{\beta}{\mu}$ depends on the biological characteristics, but does not control spreading alone
 - spreading only if the spreading rate is above a threshold, i.e., $\lambda > \lambda_c$
- Random network: with $\langle k^2 \rangle = \langle k \rangle(\langle k \rangle + 1)$ we have $\tau_{ER}^{SIS} = \frac{1}{\beta(\langle k \rangle + 1) - \mu} > 0$
 - with $\lambda = \beta/\mu$ the condition leads to $\lambda_c = \frac{1}{\langle k \rangle + 1}$ non-zero epidemic threshold
 - if the spreading rate is larger than the threshold: endemic state - otherwise, pathogen dies out
- Scale-free Network: $\tau^{SIS} = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - \langle k \rangle \mu}$
 - with $\lambda = \beta/\mu$, the condition $\tau^{SIS} > 0$ can be written as $\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}$ vanishing threshold for large N
 - even viruses that are hard to pass from individual to individual can spread successfully



Network influence in SIS



- Endemic state depends on λ and λ_c
- Accounting for the network topology alters the predictive power of epidemic models!
 - in scale-free networks, a virus can instantaneously reach most nodes
 - in scale-free networks, even virus with small spreading rate can persist
 - this is a consequence of hubs' ability to broadcast a pathogen to a large number of other nodes

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Asymptotic Behavior of the SIS Model

$$\lambda_c = 0$$

$$2 < \gamma < 3 \quad \Theta(\lambda) \sim (k_{\min} \lambda)^{(\gamma-2)/(3-\gamma)}$$

$$i(\lambda) \sim \lambda^{1/(3-\gamma)}$$

$$\lambda_c = 0$$

$$\gamma = 3 \quad \Theta(\lambda) \approx \frac{e^{-1/k_{\min} \lambda}}{\lambda k_{\min}} (1 - e^{-1/k_{\min} \lambda})^{-1}$$

$$i(\lambda) \sim 2e^{-1/k_{\min} \lambda}$$

$$\lambda_c > 0$$

$$3 < \gamma < 4 \quad i(\lambda) \sim \left(\lambda - \frac{\gamma-3}{k_{\min}(\gamma-2)} \right)^{1/(\gamma-3)}$$

$$\lambda_c > 0$$

$$\gamma > 4 \quad i(\lambda) \sim \lambda - \frac{\gamma-3}{k_{\min}(\gamma-2)}$$

- The fraction of individual infected in endemic state depends on
 - structure of the underlying network
 - disease transmission and recovery rates β and μ
- Only for large γ , the epidemics on scale-free networks converge to the results of the classical epidemic models

R. Pastor-Satorras and A. Vespignani. Epidemic dynamics and endemic states in complex networks. Physical Review E, 63:066117, 2001.

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Epidemic models on networks

Model	Continuum Equation	τ	λ_c
SI	$\frac{di_k}{dt} = \beta [1 - i_k] k \theta_k$	$\frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)}$	0
SIS	$\frac{di_k}{dt} = \beta [1 - i_k] k \theta_k - \mu i_k$	$\frac{\langle k \rangle}{\beta \langle k^2 \rangle - \mu \langle k \rangle}$	$\frac{\langle k \rangle}{\langle k^2 \rangle}$
SIR	$\frac{di_k}{dt} = \beta s_k \theta_k - \mu i_k$ $s_k = 1 - i_k - r_k$	$\frac{\langle k \rangle}{\beta \langle k^2 \rangle - (\mu + \beta) \langle k \rangle}$	$\frac{1}{\langle k^2 \rangle - 1}$

- Rate equations for the three basic epidemic models (SI, SIS, SIR)
 - not limited to scale-free networks
- Limitations:
 - with exact formulation (w/o the degree-block approximation), it can be shown that hubs play an even bigger role
 - some characteristics are not captured by our simple model, which mostly considers the degree distribution

Y. Wang, D. Chakrabarti, C. Wang, and C. Faloutsos. Epidemic spreading in real networks: an eigenvalue viewpoint. Proceedings of 22nd International Symposium on Reliable Distributed Systems, pg. 25-34, 2003.

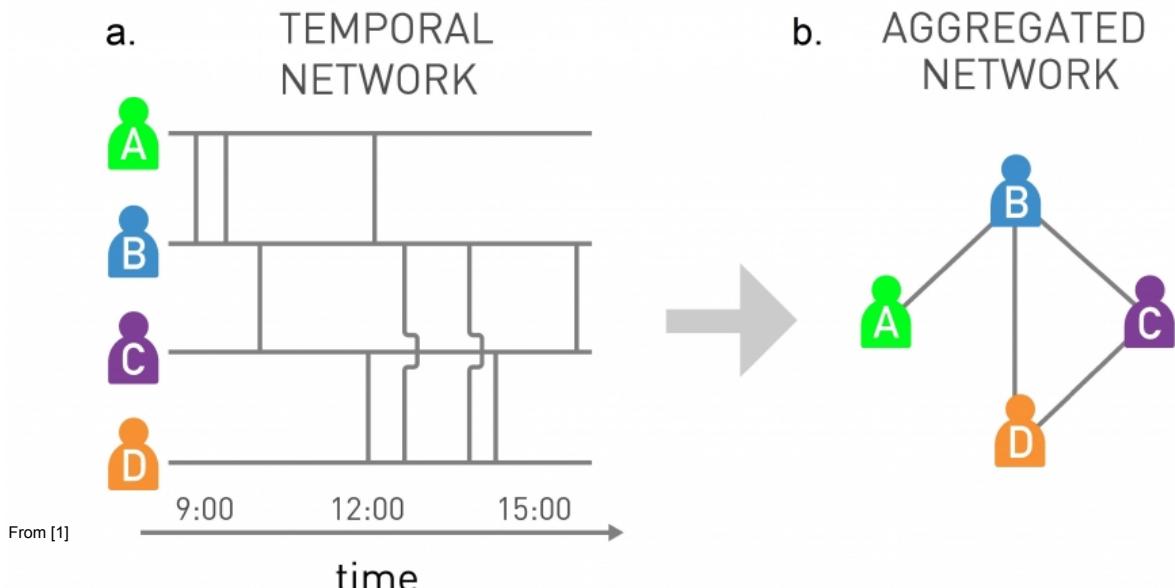
R. Durrett. Some features of the spread of epidemics and information on a random graph. PNAS, 107:4491-4498, 2010.

S. Chatterjee and R. Durrett. Contact processes on random graphs with power law degree distributions have critical value 0. Ann. Probab., 37: 2332-2356, 2009.

C Castellano, and R Pastor-Satorras. Thresholds for epidemic spreading in networks. Physical Review Letters, 105:218701, 2010



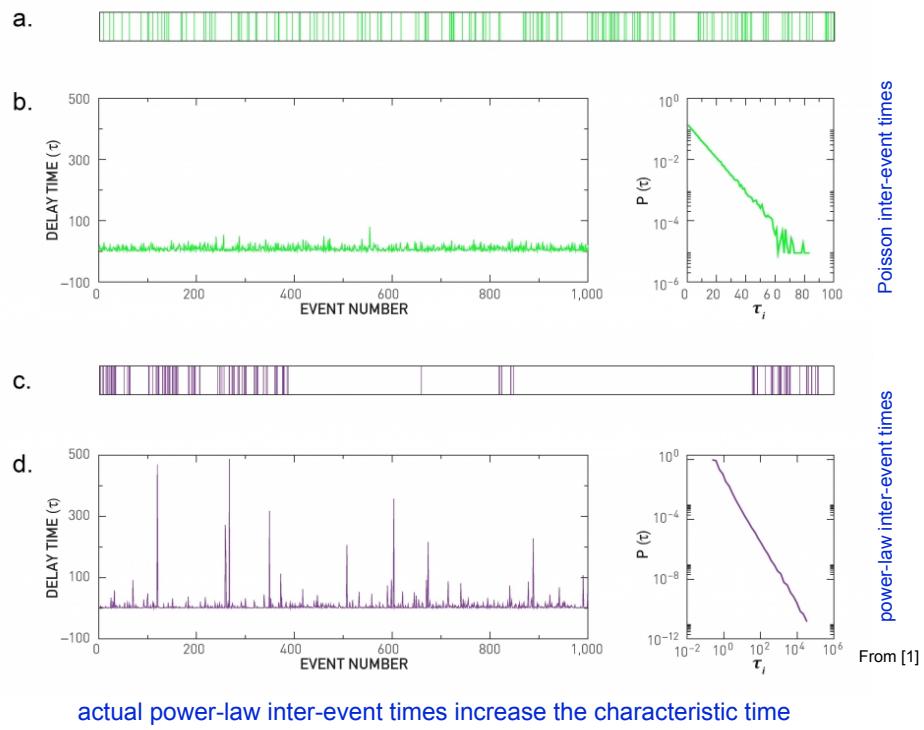
Ext. 1: Temporal networks



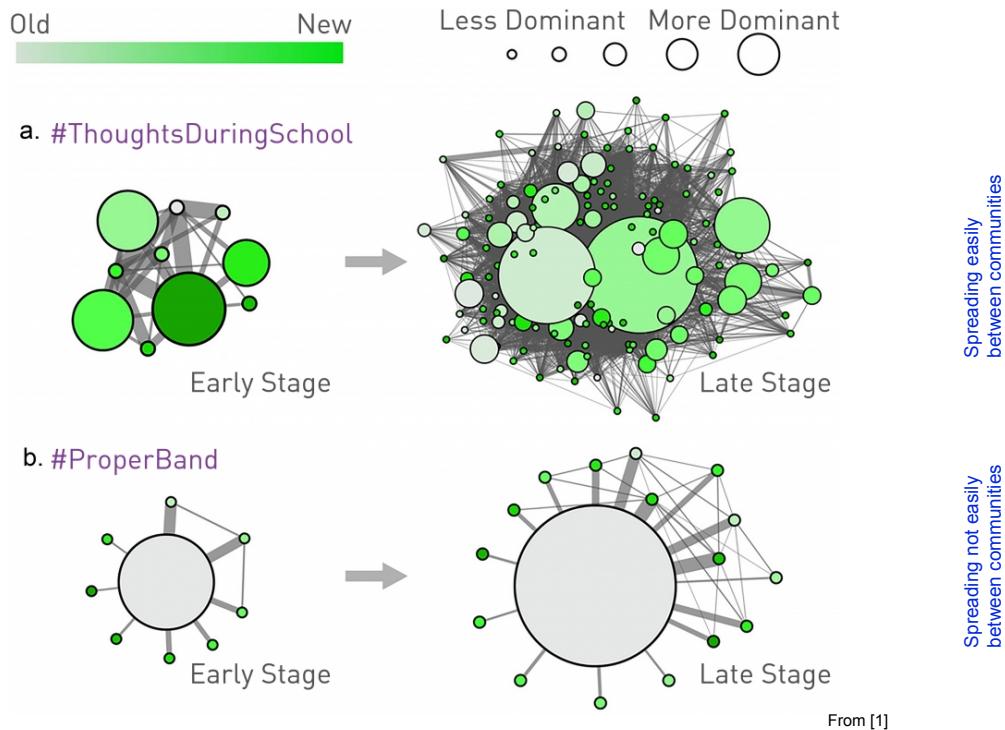
not equivalent: e.g. disease cannot spread from D to A in a), while it could in b)



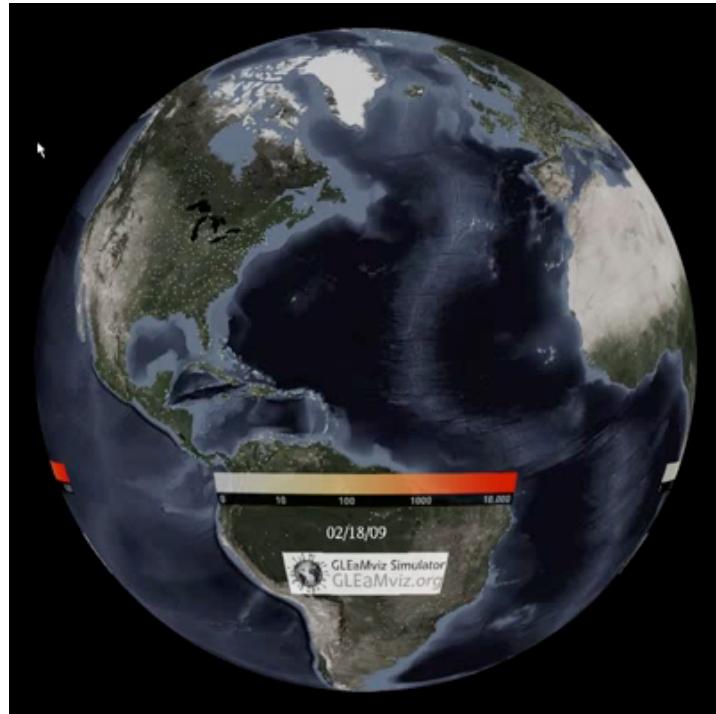
Ext. 2 : Bursty interactions



Ext. 3 :Simple vs complex contagion



Speed of a pandemic



Immunisation

- Ideally, a treatment should be given to every infected individual, or those at risk
 - not always possible (costs, reach...)
- It is therefore important to design strategies to distribute effectively the available treatment
 - network structure plays a role
- Ideally, we should try to bring the critical threshold λ_c beyond the spreading rate λ of the pathogens
 - random immunisation
 - selective immunisation

Random immunisation

- Immunisation protects from infection, but also slows down spreading
- Fraction of immunised individuals: g
- With SIS model:
 - only $(1 - g)$ individuals can contract and spread disease
 - effective degree of nodes is changed from $\langle k \rangle$ to $\langle k \rangle(1 - g)$
 - spreading rate gets affected too; it changes from $\lambda = \beta/\mu$ to $\lambda' = \lambda(1 - g)$
 - objective: find g such that λ' falls below λ_c
- Random networks:

$$\frac{(1 - g_c)\beta}{\mu} = \frac{1}{\langle k \rangle + 1} \quad \Rightarrow \quad g_c = 1 - \frac{\mu}{\beta} \frac{1}{\langle k \rangle + 1}$$

- Heterogenous networks (high $\langle k^2 \rangle$)

$$\frac{\beta}{\mu}(1 - g_c) = \frac{\langle k \rangle}{\langle k^2 \rangle} \quad \Rightarrow \quad g_c = 1 - \frac{\mu}{\beta} \frac{\langle k \rangle}{\langle k^2 \rangle}$$

For a scale-free network with $\gamma < 3$, $\langle k^2 \rangle$ is unbounded and $g_c \rightarrow 1$

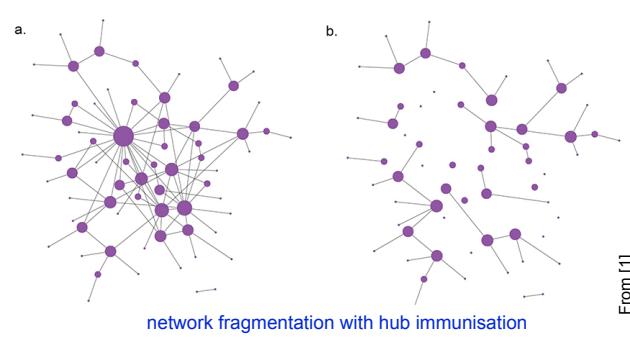
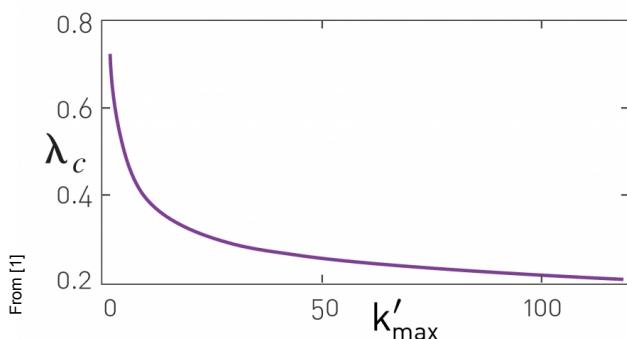


Selective immunisation

- Random immunisation ineffective in scale-free networks due to vanishing threshold
 - the threshold needs to be increased, by altering the structure of the network (e.g., $\langle k^2 \rangle$)
- *Immunising the hubs* (= immunising all nodes with degree larger than k'_{\max})
 - degree variance decreases, and epidemic threshold increases as $\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}$
 - the epidemic threshold changes to

$$\lambda'_c \approx \frac{\gamma - 2}{3 - \gamma} \frac{k_{\min}^{2-\gamma}}{(k'_{\max})^{\gamma-3}}$$

For $\gamma < 3$ the smaller k'_{\max} the larger λ'_c

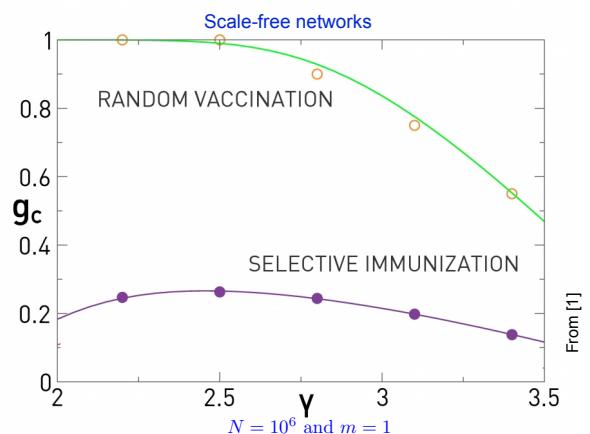


Z. Dezső and A-L. Barabási. Halting viruses in scale-free networks. Physical Review E, 65:055103, 2002.
 R. Pastor-Satorras and A. Vespignani. Immunization of complex networks. Physical Review E, 65:036104, 2002.



Selective immunisation strategy

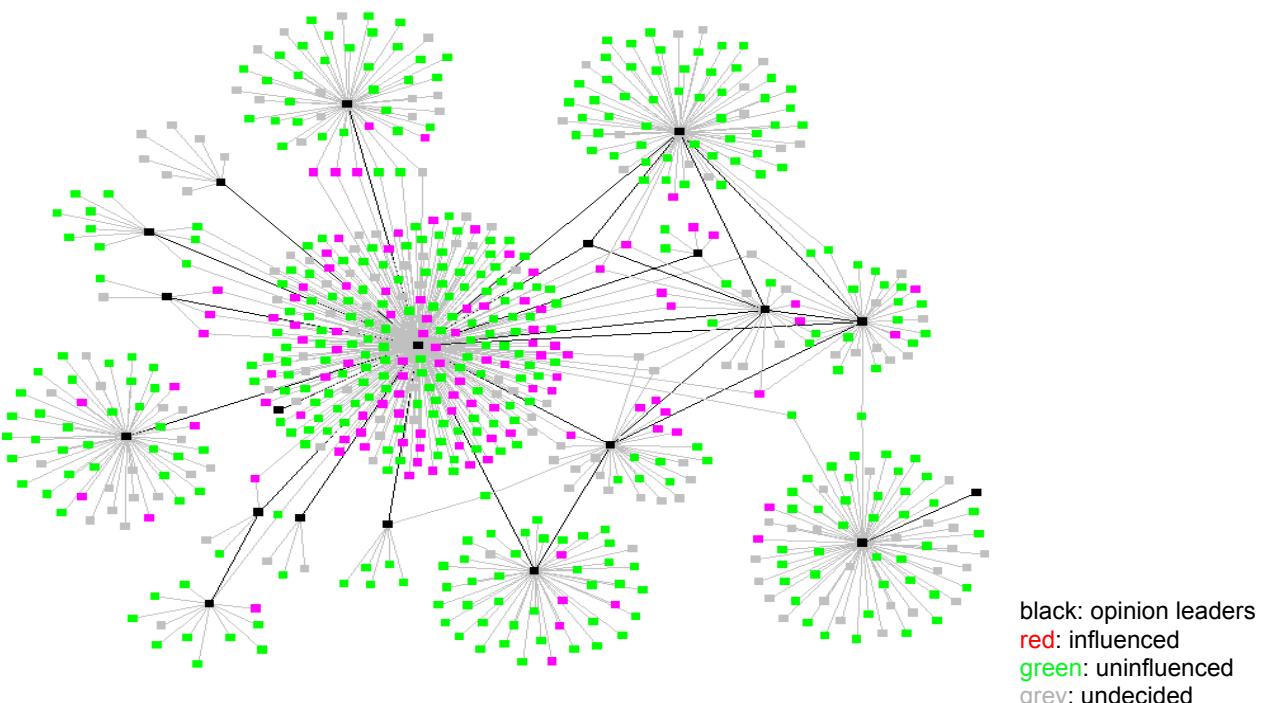
- If we lack a detailed map of the network, we cannot identify hubs
 - rely on friendship paradox (on average, neighbours have higher degree than node itself)
 - immunising neighbours, we can target hubs without identifying them precisely
- Selective immunisation strategy:
 1. Choose randomly a p fraction of nodes (Group 0).
 2. Select randomly a link for each node in Group 0 (connected nodes form Group 1, with $p'_k \sim kp_k$)
 3. Immunise the Group 1 individuals.
- Critical threshold required to eradicate a pathogen for a scale-free network
 - Random Immunisation: as degree exponent γ approaches 3, the network develops a finite epidemic threshold, and the critical immunisation rate drops
 - Selective Immunisation: critical immunisation rate always below 30%. Always more efficient than random immunisation



R. Cohen, S. Havlin, and D. ben-Avraham. Efficient Immunization Strategies for Computer Networks and Populations. Physical Review Letters, 91:247901, 2003.



Viral marketing



Summary - Spreading

- Most networks facilitate transfer along their links
 - network topology affects these dynamical processes.
 - interplay between dynamical phenomena and network topology
 - models of the spread of pathogens have important practical applications
- Analytical framework of network epidemics
 - vanishing characteristic spreading time and epidemic threshold in heterogeneous networks.
- Insights offered by network epidemiology
 - selective immunisation strategies are the most effective in scale-free networks

At a glance: Network Epidemics

- Infection rate: β
- Recovery rate: μ
- Spreading rate: $\lambda = \frac{\beta}{\mu}$
- Reproductive number: $R_0 = \frac{\beta \langle k \rangle}{\mu}$
- SI model $i(t) = \frac{i_0 e^{\beta \langle k \rangle t}}{1 - i_0 + i_0 e^{\beta \langle k \rangle t}}$
- SIS model $i(t) = \left(1 - \frac{\mu}{\beta \langle k \rangle}\right) \frac{C e^{(\beta \langle k \rangle - \mu)t}}{1 - C e^{(\beta \langle k \rangle - \mu)t}}$
- Characteristic time: $\tau^{SI} = \frac{\langle k \rangle}{\beta (\langle k^2 \rangle - \langle k \rangle)}$
 $\tau^{SIS} = \frac{\langle k \rangle}{\beta (\langle k^2 \rangle - \mu \langle k \rangle)}$
 $\tau^{SIR} = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - (\mu + \beta) \langle k \rangle}$
- Epidemic threshold: $\lambda_c^{SIS} = \frac{\langle k \rangle}{\langle k^2 \rangle}$
 $\lambda_c^{SIR} = \frac{1}{\frac{\langle k^2 \rangle}{\langle k \rangle} - 1}$
- Immunization threshold (SIS): $g_c = 1 - \frac{\mu}{\beta} \frac{\langle k \rangle}{\langle k^2 \rangle}$



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