

Epidemic spreading processes

Wout Merbis

Dutch Institute for Emergent Phenomena and Institute for Theoretical Physics, UvA

May 13, 2024



Introduction

- ▶ Spreading processes are plentiful in Nature and society
 - Epidemic spreading
 - Social contagions (rumor spreading, opinion and voter models)
 - Diffusion on networks: traffic and transportation
 - Population dynamics and ecology
 - Forest fires
 - ...

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 - Epidemiology (public health)
 - Computer science
 - Theoretical ecology
 - Statistical mechanics (physics)

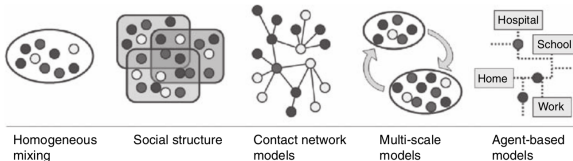
Each discipline has their own favorite modeling schemes and literature, however the most basic models are quite similar

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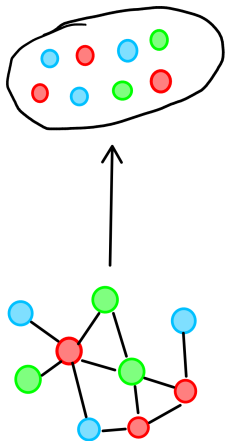
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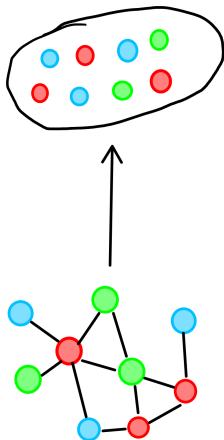
- ▶ Different modeling scenarios:



Plan for today:



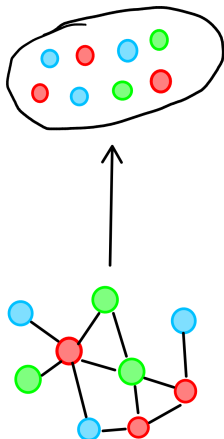
Plan for today:



► Compartmental models of epidemiology (deterministic)

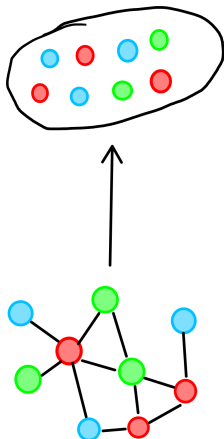
- Susceptible-Infected (SI) model
- Susceptible-Infected-Susceptible (SIS) model
- Susceptible-Infected-Recovered (SIR) model
- Epidemic thresholds

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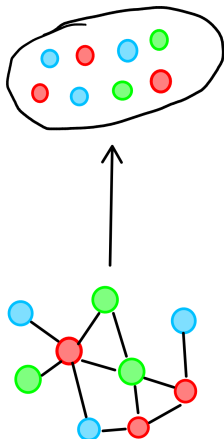
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- ▶ 'Microscopic' network models (stochastic)
 - Epidemic spreading on networks
 - Mean-field approximation in the context of epidemic models
 - Homogeneous vs. heterogeneous network models

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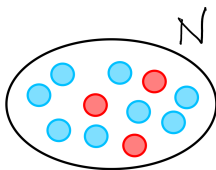


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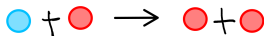
Key concepts:

- Dynamical process (out-of-equilibrium)
- Dynamical (continuous) phase transition
- Mean-field approximations
- Network heterogeneity

Compartmental models of epidemiology

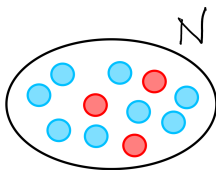


SI-model

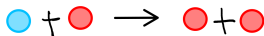


● Susceptible
● Infected

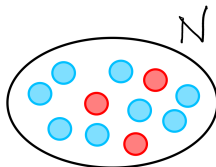
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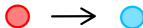
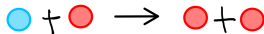
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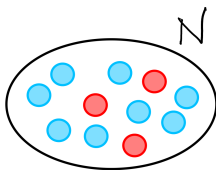
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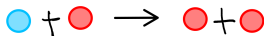
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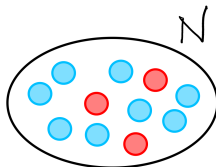
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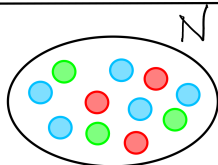
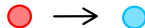
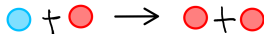
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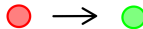
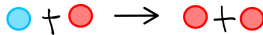
● Susceptible
● Infected



SIS-model



SIR-model



● Recovered

Compartmental models of epidemiology

- ▶ Divide a population of N individuals into compartments $[m]$ each containing a certain number of individuals $X^{[m]}(t)$ at time t .

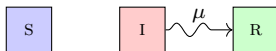
Conservation of the total number of individuals implies $\sum_{[m]} X^{[m]}(t) = N$



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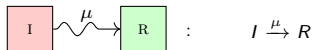
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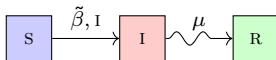
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Compartmental models of epidemiology

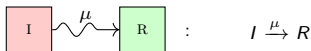
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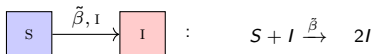


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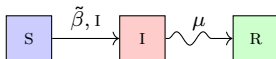
- Binary interaction transitions among individuals of different compartments



Compartmental models of epidemiology

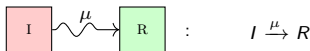
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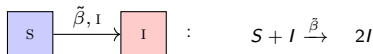


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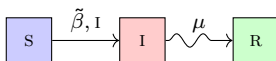


- ▶ The population is assumed to be homogeneously mixed: all individuals have equal probability of running into each other

Compartmental models of epidemiology

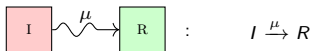
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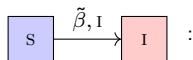


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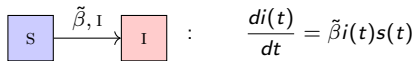


- ▶ The population is assumed to be homogeneously mixed: all individuals have equal probability of running into each other
- ▶ Interested in the rate of change of the fraction of infected $i(t) = \frac{1}{N} X^{[I]}(t)$

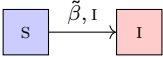
Susceptible-Infected (SI) model



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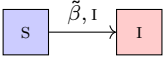
Susceptible-Infected (SI) model



The diagram shows a blue box labeled 'S' (Susceptible) on the left and a red box labeled 'I' (Infected) on the right. An arrow points from 'S' to 'I' with the label $\tilde{\beta}, I$ above it. To the right of the arrow is a colon ':'.

$$\frac{di(t)}{dt} = \tilde{\beta} i(t) s(t) = \tilde{\beta} i(t) (1 - i(t))$$

Susceptible-Infected (SI) model



The diagram shows a blue box labeled 'S' (Susceptible) with an arrow pointing to a red box labeled 'I' (Infected). Above the arrow is the label $\tilde{\beta}, I$. To the right of the boxes is a colon followed by the differential equation.

$$\frac{di(t)}{dt} = \tilde{\beta}i(t)s(t) = \tilde{\beta}i(t)(1 - i(t))$$

This is the logistic equation (Verhulst, 1840s), originally used to model population growth.

Susceptible-Infected (SI) model

$$\begin{array}{|c|} \hline S \\ \hline \end{array} \xrightarrow{\tilde{\beta}, I} \begin{array}{|c|} \hline I \\ \hline \end{array} : \quad \frac{di(t)}{dt} = \tilde{\beta}i(t)s(t) = \tilde{\beta}i(t)(1 - i(t))$$

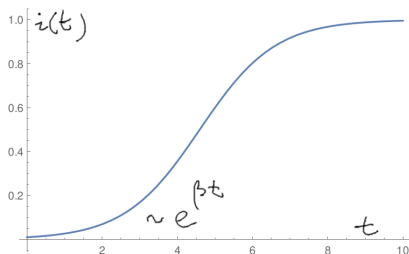
This is the logistic equation (Verhulst, 1840s), originally used to model population growth. It can be solved analytically as

$$i(t) = \frac{i(0)e^{\tilde{\beta}t}}{1 + i(0)(e^{\tilde{\beta}t} - 1)}$$

► At early times, when $i(t) \ll 1$:

$$\frac{di(t)}{dt} = \tilde{\beta}i(t)$$

And $i(t) \sim e^{\tilde{\beta}t}$.



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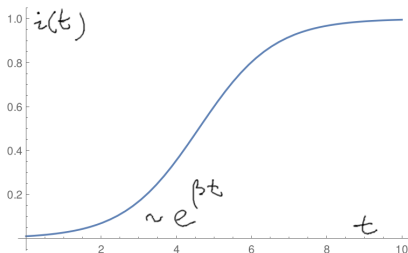
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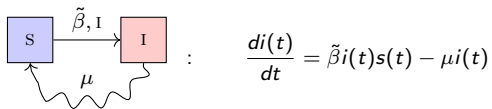
- When $i(t) = 1$:

$$\frac{di(t)}{dt} = 0$$

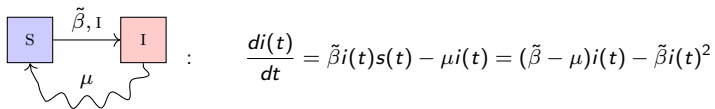
So $i(t) = 1$ is a steady-state



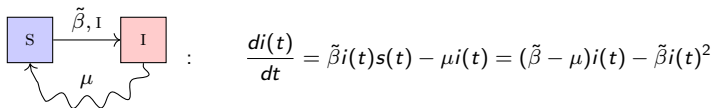
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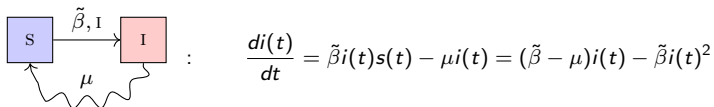


Susceptible-Infected-Susceptible (SIS) model



This is still the Verhulst logistic equation, as long as $R_0 = \frac{\tilde{\beta}}{\mu} > 1$.

Susceptible-Infected-Susceptible (SIS) model



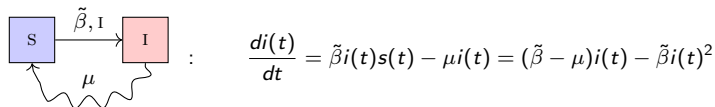
This is still the Verhulst logistic equation, as long as $R_0 = \frac{\tilde{\beta}}{\mu} > 1$.

For small $i(t)$, we now have:

$$i(t) \sim e^{t/\tau} \quad \tau^{-1} = (\tilde{\beta} - \mu) \equiv \mu(R_0 - 1)$$

Here $R_0 = \frac{\tilde{\beta}}{\mu}$ is the **basic reproductive number**.

Susceptible-Infected-Susceptible (SIS) model



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Dynamical (continuous) phase transition between:

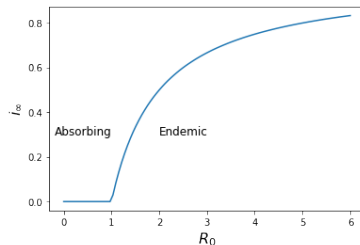
$R_0 \leq 1$: Absorbing phase: epidemic eventually dies out

$$i_{\infty} = 0$$

$R_0 > 1$: Endemic phase: logistic growth to a stable 'endemic' state

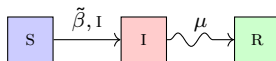
$$i_{\infty} = 1 - \frac{1}{R_0}$$

$R_c = 1$ is the **Epidemic threshold**



Susceptible-Infected-Recovered (SIR) model

The SIR model adds recovery to a new R compartment for recovered (or removed)



The rate of equations are now:

$$\frac{ds(t)}{dt} = -\tilde{\beta}i(t)s(t), \quad \frac{di(t)}{dt} = \tilde{\beta}i(t)s(t) - \mu i(t), \quad \frac{dr(t)}{dt} = \mu i(t)$$

Now the epidemic will always die out eventually, but still two types of behaviour can be distinguished depending on $R_0 = \frac{\beta}{\mu}$

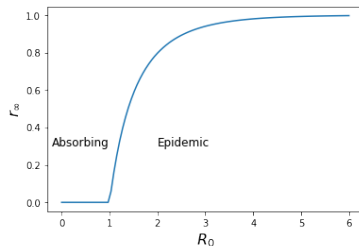
$R_0 \leq 1$: Absorbing regime: infection does not reach a finite fraction of the population $r_\infty = 0$.

$R_0 > 1$: Epidemic regime: infection reaches a finite fraction of the population r_∞

At late time:

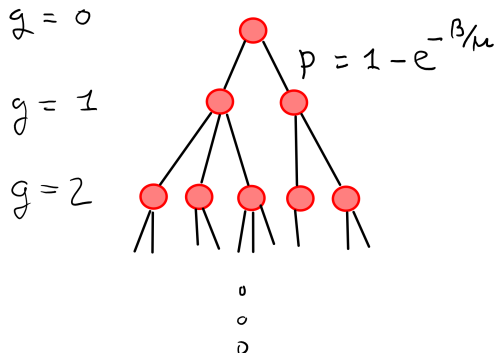
$$1 - r_\infty = e^{-R_0 r_\infty}$$

Exercise: derive this equation.



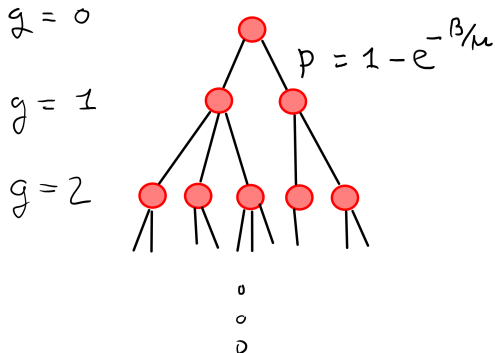
Map to directed percolation

Constructing r_∞ as a percolation problem: start with the initial infected and trace the edges along which the infection has spread



Map to directed percolation

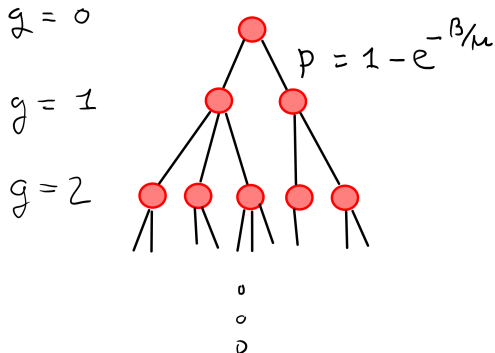
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Exactly like a percolation problem, where an edge is present with probability p and absent with probability $1 - p$.

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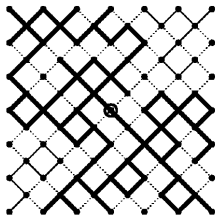


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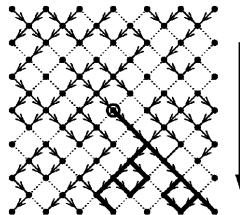
The **percolation threshold**, above which a giant connected component appears, is equal to the epidemic threshold.

Directed percolation

Difference with bond percolation: now the process is *directed*, as the infection cannot travel backwards in time.



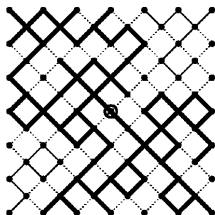
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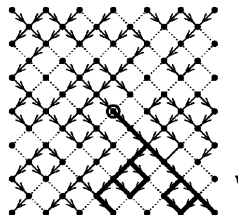
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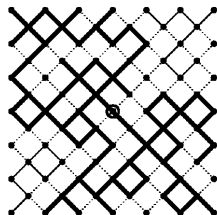


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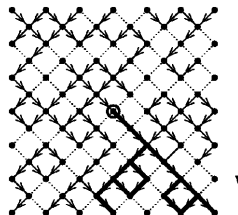
SIS model is equivalent to the mean-field approximation of the directed percolation problem.

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isotropic bond percolation



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Universality of critical exponents: close to the critical threshold, the density of infected scales as

$$\rho \sim (p - p_c)^\beta$$

The value of p_c may differ between different models with absorbing state transition, but the critical exponent β is the same for many different models.

Directed percolation critical exponents

Critical exponents for directed percolation: β for the density in the percolating cluster

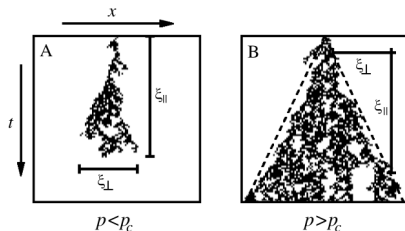
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Directed percolation critical exponents

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Two types of correlation lengths: spatial ξ_\perp and temporal correlation length ξ_\parallel



$$\xi_\perp \sim |p - p_c|^{-\nu_\perp}$$

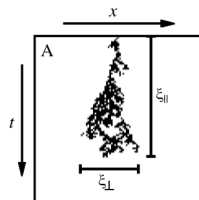
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Directed percolation critical exponents

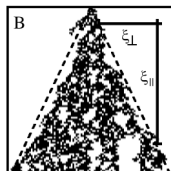
Critical exponents for directed percolation: β for the density in the percolating cluster

$$\rho \sim (p - p_c)^\beta$$

Two types of correlation lengths: spatial ξ_\perp and temporal correlation length ξ_\parallel



$p < p_c$



$p > p_c$

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critical exponent	MF	$d = 1$ [168]	$d = 2$ [125]	$d = 3$ [170]
β	1	0.276 486 (8)	0.584(4)	0.81(1)
ν_\perp	1/2	1.096 854 (4)	0.734(4)	0.581(5)
ν_\parallel	1	1.733 847 (6)	1.295(6)	1.105(5)

$d_c = 4$ is the upper critical dimension for DP

Deterministic vs. stochastic models

The SIS/SIR models are often extended to include more compartments, for instance

- ▶ Exposed individuals who are not yet infectious
- ▶ Asymptomatic infectious individuals
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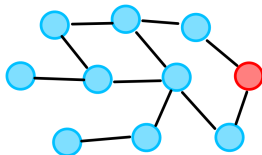
Ultimately, a population is a complex system composed out of many individuals. The deterministic compartmental models should emerge from a stochastic microscopic model detailing the interactions of individuals.

Stochastic spreading models on networks

Let's take a microscopic and stochastic perspective to the spreading process.

● : $S = 0$

● : $I = 1$



Transmission: ● — ● $\xrightarrow{\beta}$ ● — ●

Recovery: ● $\xrightarrow{\mu}$ ● SIS

● \rightarrow ● SIR

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Here A^{ij} is the adjacency matrix of a contact network: $A^{ij} = 1$ if i and j are connected, $A^{ij} = 0$ otherwise

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This gives up to order dt :

$$\mathbb{P}[X_i(t + dt) = 1] = \mathbb{P}[X_i(t) = 1](1 - \mu dt) + \sum_j A^{ij} \mathbb{P}[X_i(t) = 0; X_j(t) = 1] \beta dt .$$

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After some rearrangement and taking the limit $dt \rightarrow 0$:

$$\frac{d\mathbb{P}[X_i(t) = 1]}{dt} = -\mu \mathbb{P}[X_i(t) = 1] + \beta \sum_j A^{ij} \mathbb{P}[X_i(t) = 0; X_j(t) = 1].$$

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The probability of any i, j forming an SI pair in turn depends on II and SI pairs and triples of the form SSI and ISI. (exercise: derive the eqn for SI pairs)

$$\begin{aligned} \frac{d}{dt} \text{SI}(i) = & -\beta \text{SI}(i) - \mu \text{SI}(i) + \mu \text{SI}(i) \\ & + \beta \sum_k \text{SI}(k) - \beta \sum_k \text{SI}(k) \end{aligned}$$

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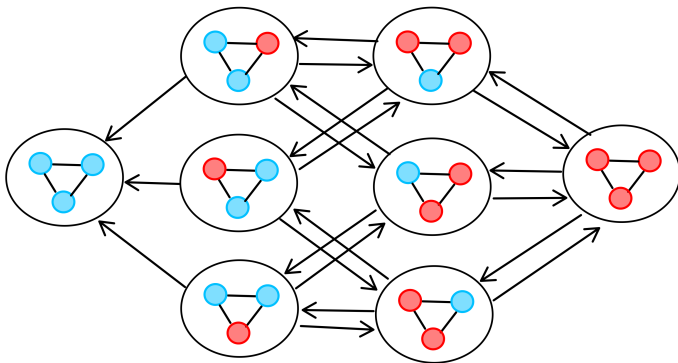
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The chain of dependencies can be closed by mean-field approximations, as we will see shortly.

Exact Markovian description

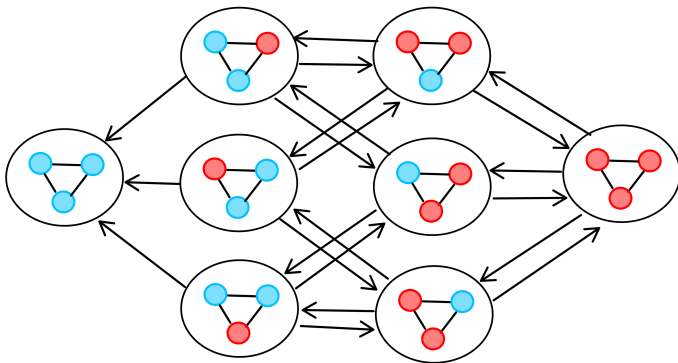
The exact problem is a Markov chain on the space of all possible network configurations. The SIS model on a graph with $N = 3$:



Transitions to the right have rate $\sim \beta$, transitions to the left have rate μ .
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Hard problem: number of configurations grows $\sim 2^N$.

Mean-field approximation: limit to compartmental models

Using the mean-field approximation, we can bridge the stochastic network models to the deterministic compartmental models:

- ▶ Define the density of infected $i(t)$:

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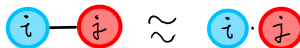
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Human beings typically do not interact randomly. For instance, we fly around the world using airplanes. All airports and the flights connecting them form a complex network



Heterogeneous network models

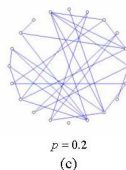
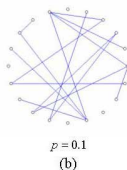
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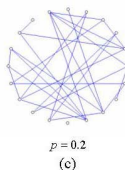
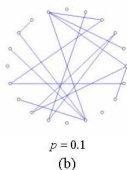


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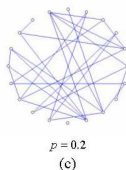
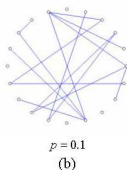
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- There are $\binom{N-1}{k}$ different ways to select k distinct neighbors
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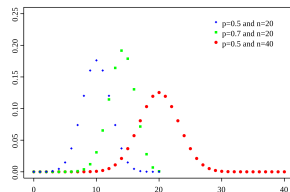
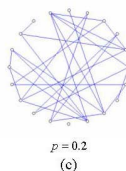
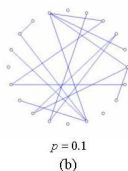
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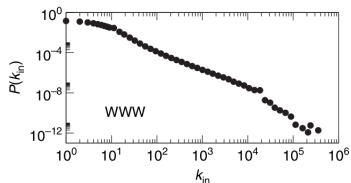
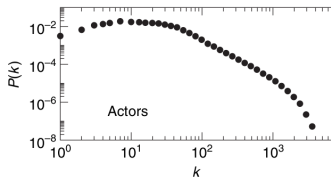
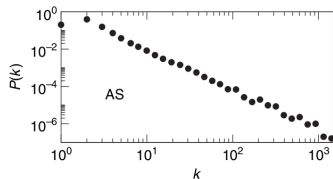
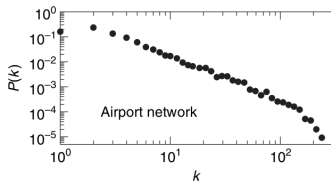
$$P(k) = \binom{N-1}{k} p^k (1-p)^{N-1-k}$$

Heterogeneous network models

Do real-world networks have a similar degree distribution?

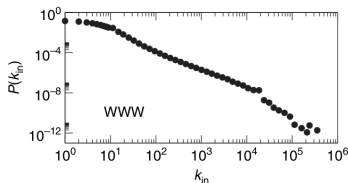
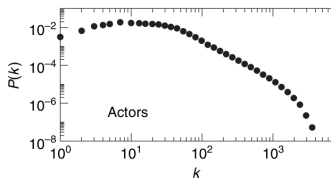
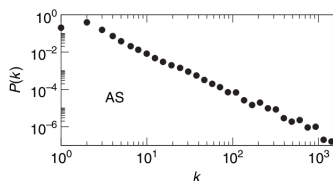
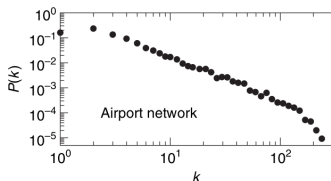
Heterogeneous network models

Do real-world networks have a similar degree distribution?



Heterogeneous network models

Do real-world networks have a similar degree distribution?



Real-world networks are often **fat-tailed**. For a range of intermediate k values the distribution follows a power-law

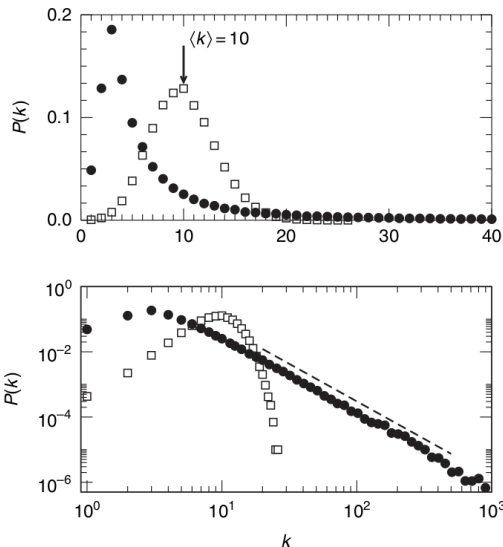
$$P(k) \sim k^{-\gamma}, \quad \text{with: } 2 < \gamma < 3$$

These networks are called **scale-free networks**.

Heterogeneous network models

Comparing binomial distribution (\square)
vs power-law distribution (\bullet) with
 $\gamma = 2.3$

Both networks have the same
average degree $\langle k \rangle = 10$



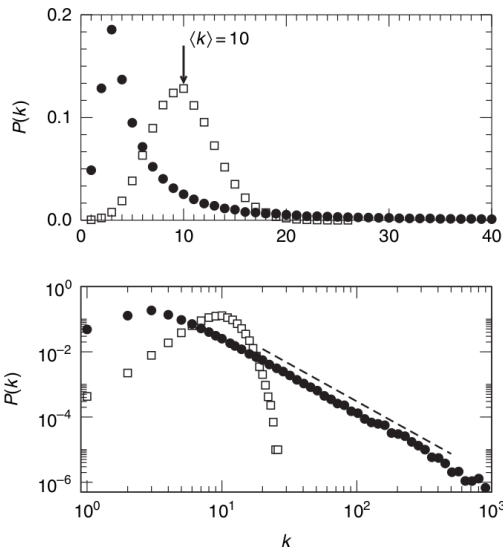
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Scale-free networks properties

- Overabundance of *hubs*: nodes of high degree



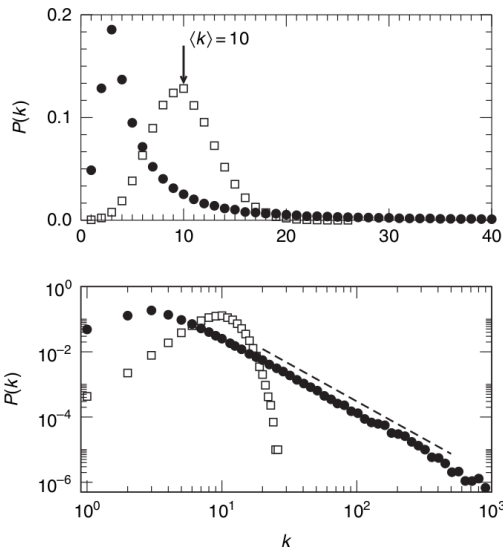
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- ▶ Scale-free: self-similarity across different length-scales



Heterogeneous network models

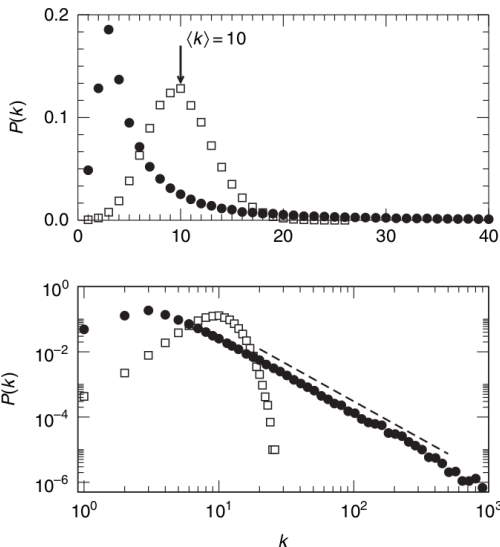
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Scale-free networks properties

- ▶ Overabundance of *hubs*: nodes of high degree
- ▶ Scale-free: self-similarity across different length-scales
- ▶ For $2 < \gamma < 3$, the fluctuations in degree depend on the system size and diverge as $N \rightarrow \infty$:

$$\langle k^2 \rangle = \sum_k k^2 P(k) \rightarrow \infty$$



Heterogeneous network models

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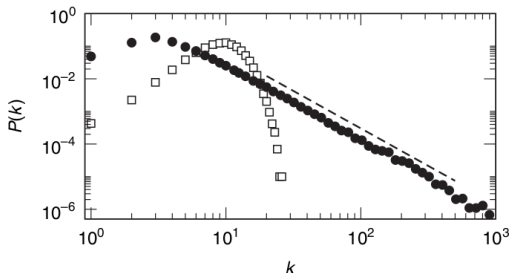
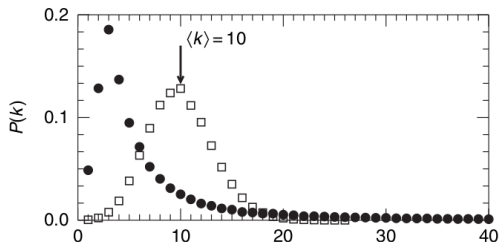
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Scale-free networks properties

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- ▶ This means the average degree is not a characteristic scale



Heterogeneous mean-field approximation

Degree block approximation: Assume all nodes with the same degree are statistically equivalent

Group all nodes of degree k in degree classes

$$i_k = \frac{I_k}{N_k}, \quad s_k = \frac{S_k}{N_k}, \quad \text{s.t. } i(t) = \sum_k i_k(t)P(k)$$

I_k, S_k : number of infected (resp. susceptible) nodes of degree k . $N_k = I_k + S_k$

Heterogeneous mean-field approximation

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How does $i_k(t)$ change in time?

Heterogeneous mean-field approximation

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Recovery is a Poisson process

$$\frac{di_k(t)}{dt} = -\mu i_k(t)$$

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Transmission contains a new quantity $\Theta_k(t)$:

$$\frac{di_k(t)}{dt} = -\mu i_k(t) + \beta k s_k(t) \Theta_k(t)$$

Where $\Theta_k(t)$ is the probability that a neighbor of a node of degree k is infected.

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Where $\Theta_k(t)$ is the probability that a neighbor of a node of degree k is infected. We need further simplifying assumptions to get $\Theta_k(t)$

Assume lack of degree correlations: Prob of arriving at a node with degree k' when following an edge starting from a node of degree k is independent of k

Heterogeneous mean-field approximation

We want the probability that a neighbor of a node with degree k is infected $\Theta_k(t)$. First break it up in two parts:

$$\Theta_k(t) = \sum_{k'} P(k'|k) i_{k'}(t)$$

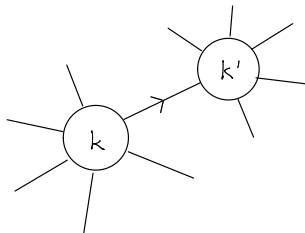
$P(k'|k)$: Probability that an edge starting from a degree k node ends in a node of degree k'

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Generic neighbor of degree k'

No degree correlations: Assume $P(k'|k)$ is independent of k

- ▶ prop. to probability $P(k')$ of node having degree k'
- ▶ prop. to k' : the no. of ways the neighboring node can be reached

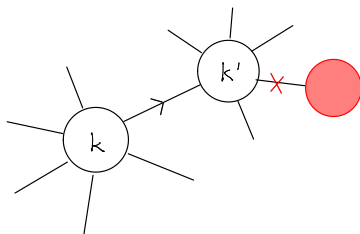
$$P(k'|k) = \frac{k'}{\langle k \rangle} P(k')$$

Heterogeneous mean-field approximation

We want the probability that a neighbor of a node with degree k is infected $\Theta_k(t)$. First break it up in two parts:

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Infected neighbor of degree k'

No degree correlations: Assume $P(k'|k)$ is independent of k

- ▶ prop. to probability $P(k')$ of node having degree k'
- ▶ prop. to $k' - 1$: cannot reach neighbor through the edge which transmitted the infection

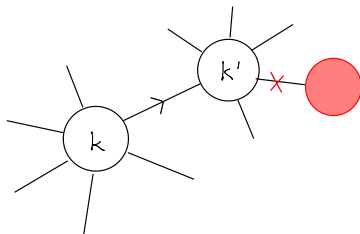
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Putting it all together, we have:

$$\Theta_k(t) = \Theta(t) = \sum_{k'} \frac{k' - 1}{\langle k \rangle} P(k') i_{k'}(t)$$

Heterogeneous mean-field approximation

Let's see how the degree fluctuations influence the epidemic threshold. We have:

$$\frac{di_k(t)}{dt} = -\mu i_k(t) + \beta k(1 - i_k(t))\Theta(t), \quad \text{with: } \Theta(t) = \sum_{k'} \frac{k' - 1}{\langle k \rangle} P(k') i_{k'}(t)$$

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Assuming that in the early stage of the outbreak, $i_k(t) \ll 1$, so we neglect $\mathcal{O}(i_k^2)$ terms, such that:

$$\begin{aligned} \frac{di_k(t)}{dt} &= -\mu i_k(t) + \beta k \Theta(t), \\ \frac{d\Theta(t)}{dt} &= \left(\beta \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle} - \mu \right) \Theta(t) \end{aligned}$$

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So at early times:

$$\Theta(t) \sim e^{t/\tau}, \quad \text{with: } \tau^{-1} = \mu \left(\frac{\beta}{\mu} \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle} - 1 \right) \equiv \mu(R_0 - 1)$$

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So the basic reproductive ratio R_0 increases with larger the degree fluctuation $\langle k^2 \rangle$

$$R_0 = \frac{\beta}{\mu} \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle}. \quad \text{at the epidemic threshold: } \frac{\beta}{\mu} = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}$$

Heterogeneous mean-field approximation

Bottom line: large degree fluctuations lower the epidemic threshold!

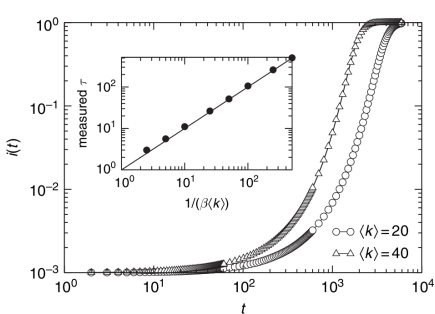
Discussion: why do you think this is true?

Heterogeneous mean-field approximation

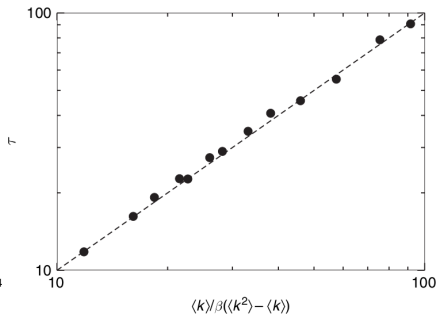
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Numerical simulations confirm initial outbreak rate



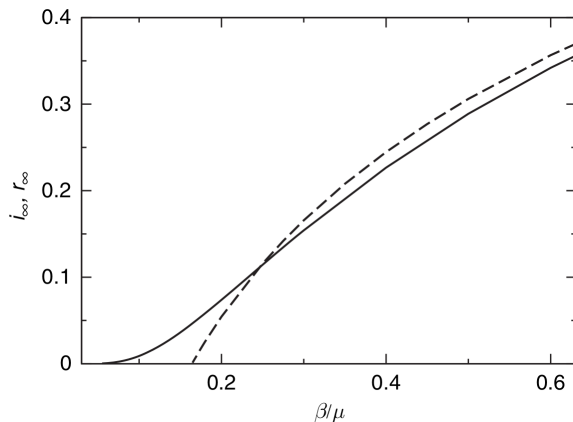
SI outbreaks on Erdős-Rényi random graphs



On (approximate) scale-free networks

Phase diagram

The phase diagram for the epidemic models with i_∞, r_∞ as order parameter and β/μ as control parameter



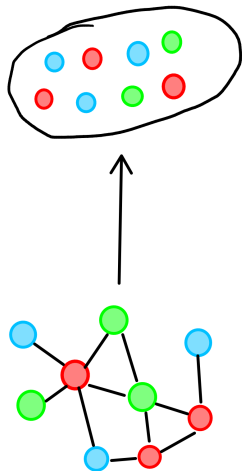
Dashed: homogeneous mixing

Solid: heterogeneous (scale-free) networks

Overview

Today we looked at epidemic models from a microscopic perspective

- ▶ Deterministic compartmental models describe the dynamics of well-mixed populations
- ▶ Stochastic network models can incorporate structure within the population
- ▶ Mean-field approximations relate the two modeling frameworks



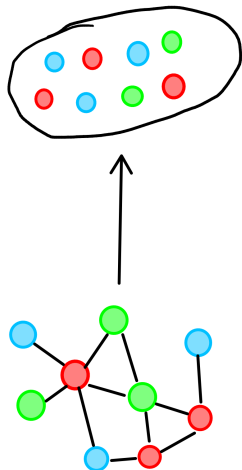
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- ▶ Threshold for a dynamical phase transition
- ▶ Markov process on networks
- ▶ Heterogeneity in contact networks (scale-free property)
- ▶ Universality of critical exponents



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Many more refined models can and have been proposed and studied! (see for instance this good [\[Review paper\]](#))

