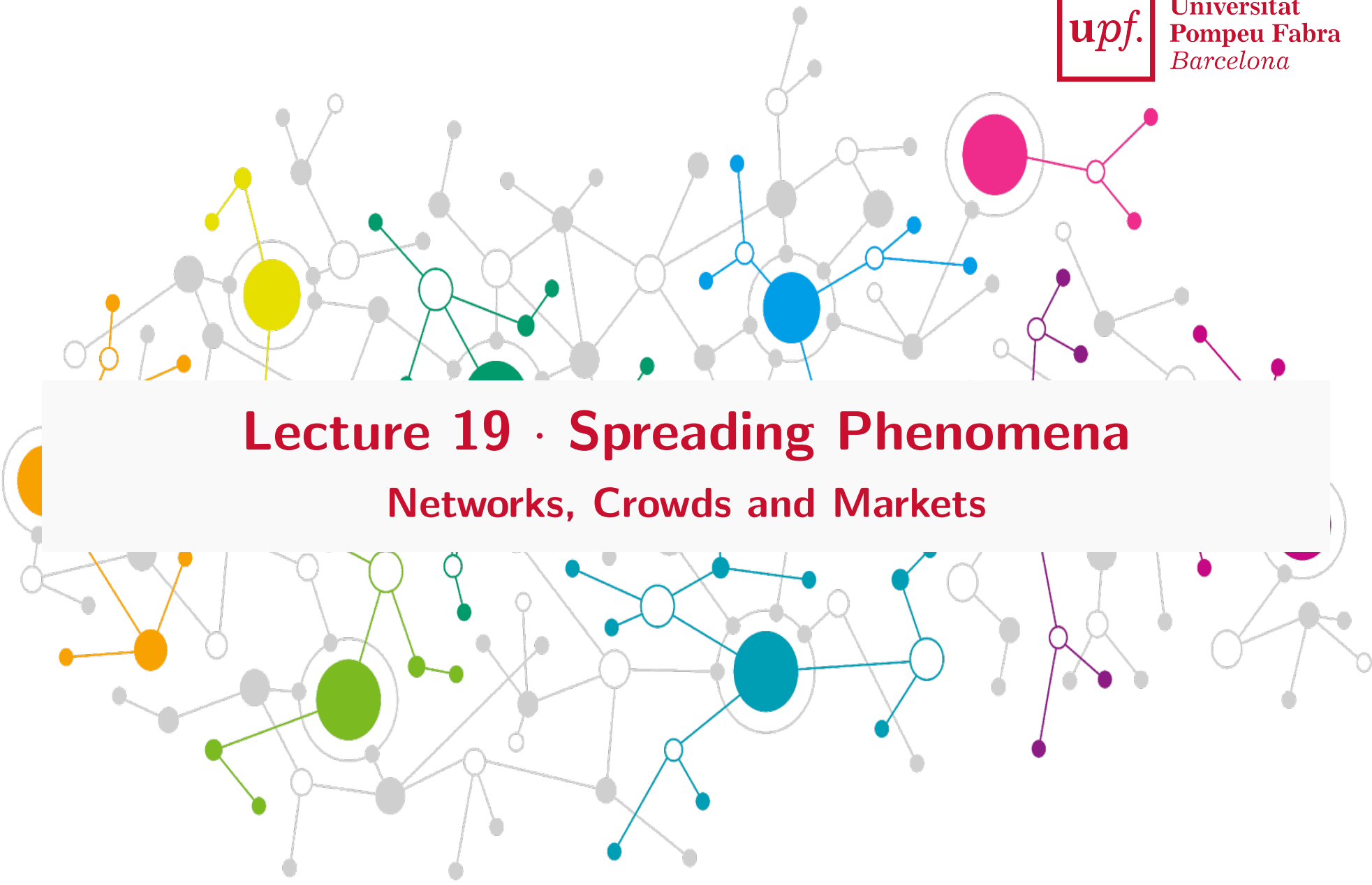


Universitat Pompeu Fabra Barcelona

Lecture 19 · Spreading Phenomena

Networks, Crowds and Markets

Reports feedback

Generally, presentations much better than the reports.

Final grading: 60% project, 20% group presentation, 20% individual.

My main complaints:

- Impersonal, monotone tone. Developing simple examples is critical.
- Often read like 4 seperate reports. No consistent terminology.
- Detached from the lecture. Things are redefined, often with different notation.
- Suggested references were just the starting point.
- Few reports: Too little focus on the underlying modelling.

Summary for the lecture

Last week we started discussing:

1. Spreading phenomena

2. Modelling hypotheses

3. Epidemic modelling
   - 3.1 Susceptible-Infected (SI)
   - 3.2 Susceptible-Infected-Susceptible (SIS)

Today we continue with this topic.

3. Epidemic modelling
   - 3.3 Susceptible-Infected-Recovered (SIR)

4. Epidemics on networks (rumor spread)

Susceptible-Infected-Recovered Model (SIR)

$$\mathbf{S} \xrightarrow{\beta} \mathbf{I} \xrightarrow{\mu} \mathbf{R}.$$

Susceptible-Infected-Recovered Model (SIR)

For many pathogens individuals develop immunity after they recover.

Thus, they do not immediately return to the susceptible state.

- These recovered individuals no longer matter from the perspective of the pathogen: they cannot be infected and they cannot infect others.
- The SIR model captures the dynamics of such infections.

Susceptible-Infected-Recovered Model (SIR)

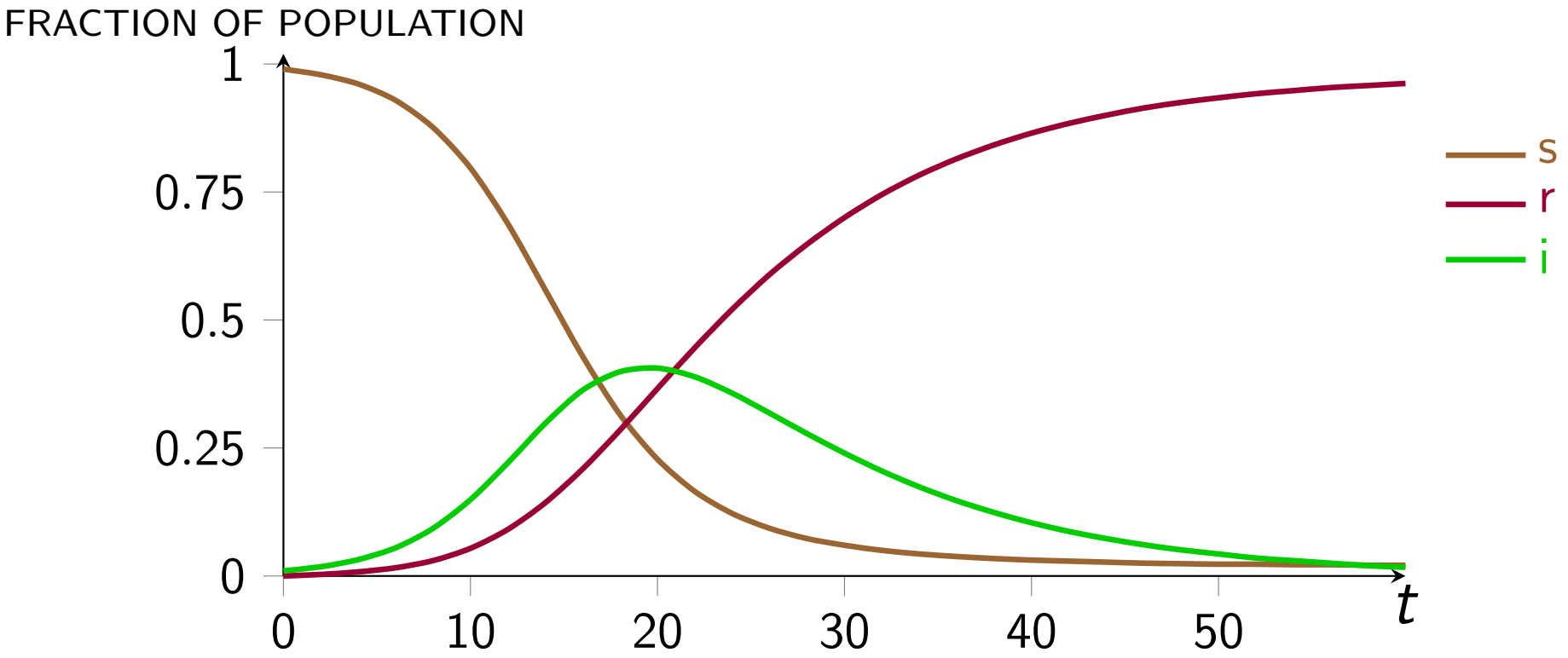
In this model $s(t) + i(t) + r(t) = 1$, where:

$$\frac{\mathrm{d}s}{\mathrm{d}t} = -\beta c\, i(1 - r - i), \quad \frac{\mathrm{d}i}{\mathrm{d}t} = -\mu i + \beta c\, i(1 - r - i), \quad \frac{\mathrm{d}r}{\mathrm{d}t} = \mu i.$$

- The SIR system describes infections where recovered individuals acquire immunity and do not become susceptible again.
- Unlike the SI and SIS models, the SIR equations do **not** admit a simple closed-form solution for $s(t)$, $i(t)$, or $r(t)$.
- Still, several analytical facts allow us to understand the qualitative behaviour of the model (as illustrated by plots).

Behavior of s, i, r in the SIR model

Recall $s(t) + i(t) + r(t) = 1$ for all $t \geq 0$.



Epidemics on Networks

What These Models Miss

The SI/SIS/SIR models assume:

- **homogeneous mixing** – everyone meets everyone else,
- identical contact rates c for all individuals,
- no information on who interacts with whom.

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- **homogeneous mixing** – everyone meets everyone else,
- identical contact rates c for all individuals,
- no information on who interacts with whom.

These assumptions make the equations simple, but they ignore a key fact:

Real spreading happens on networks.

(and many real networks are scale free)

Why Networks Matter

In reality:

- people meet their friends, colleagues, family;
- ideas and rumours spread along social links;
- computer viruses spread along digital connections;
- failures propagate in supply or infrastructure networks.

Network structure changes:

- who gets infected,
- how fast things spread,
- whether an epidemic can be stopped.

Discrete-Time SI on a Network

For simplicity we consider a discrete time of the earlier models.

Network SI (discrete time)

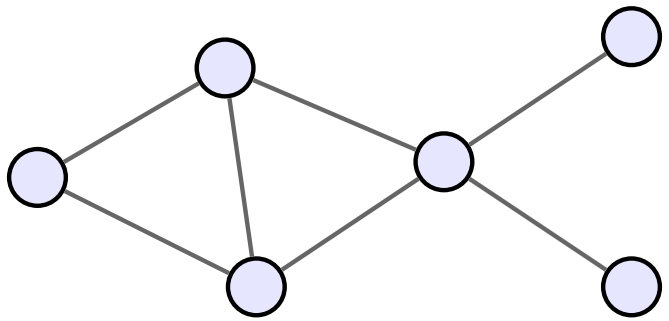
- Nodes = individuals; edges = possible contacts.
- Each node is Susceptible (S) or Infected (I).
- Time steps $t = 0, 1, 2, \ldots$
- At each step, every infected node infects each susceptible neighbour with probability p.
- Once infected, a node never recovers.

"Probability p per step" plays the role of "rate β" in continuous time.

Susceptible-Infected (SI) Model on a Network

- Start at $t = 0$ with a single infected node.
- For each t, look at every edge connecting an **infected** node to a **susceptible** neighbour.
- Along each such edge, independently, infection is transmitted with probability p (coin: $1 =$ infection, $0 =$ no infection).
- All new infections become active at the next step.

(nobody infected yet)



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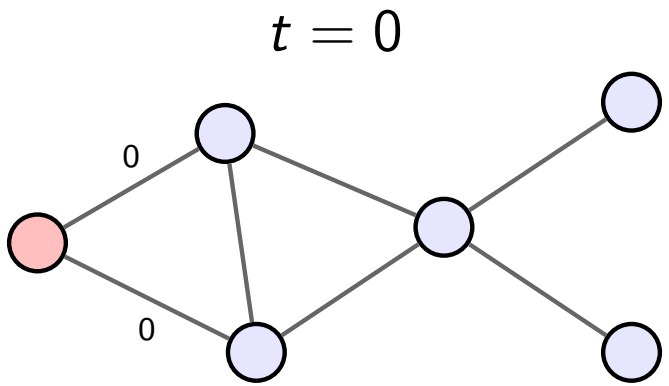

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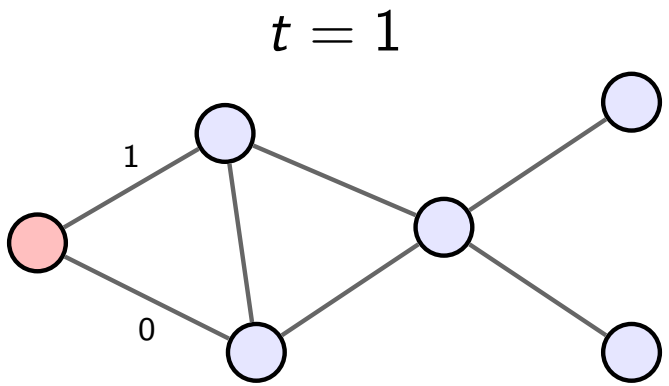

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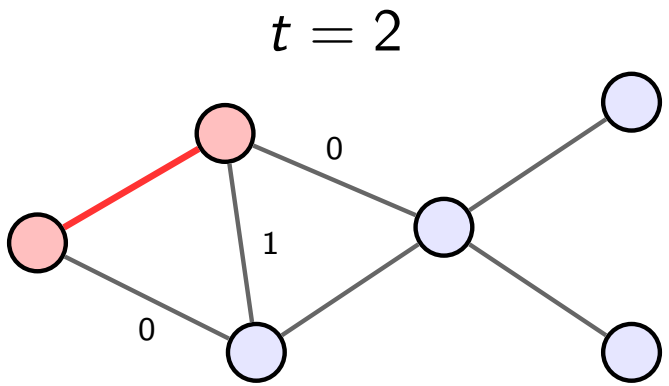

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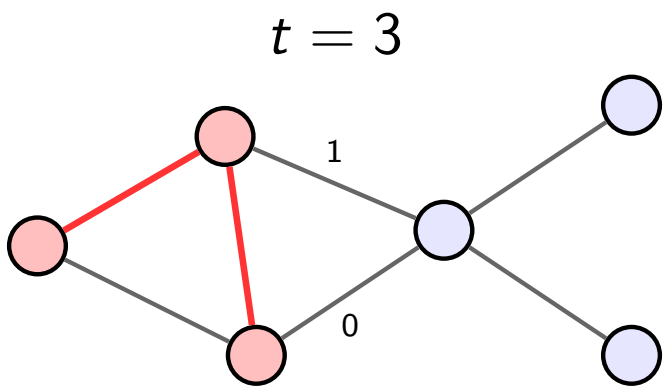

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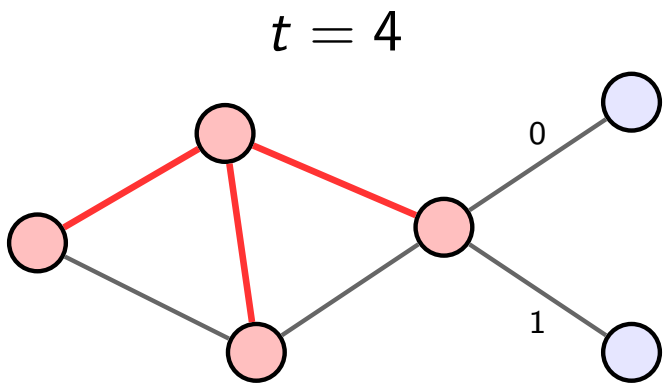

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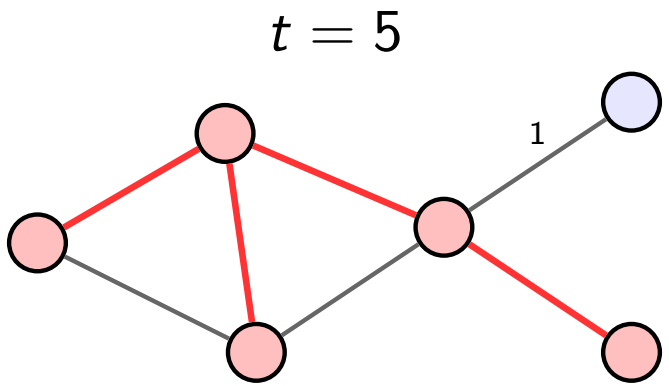

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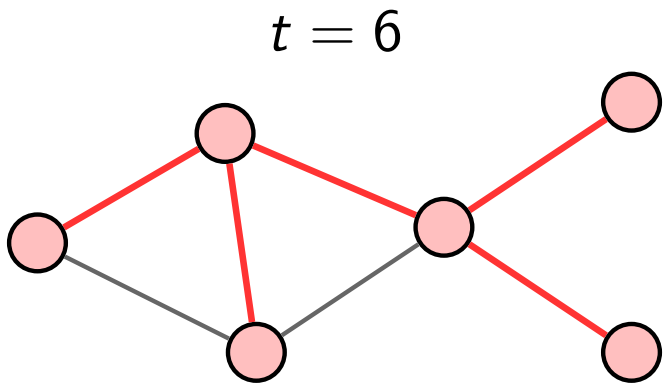

Why Degrees Matter

High-degree nodes ("hubs") tend to get infected early and then infect many neighbours quickly.

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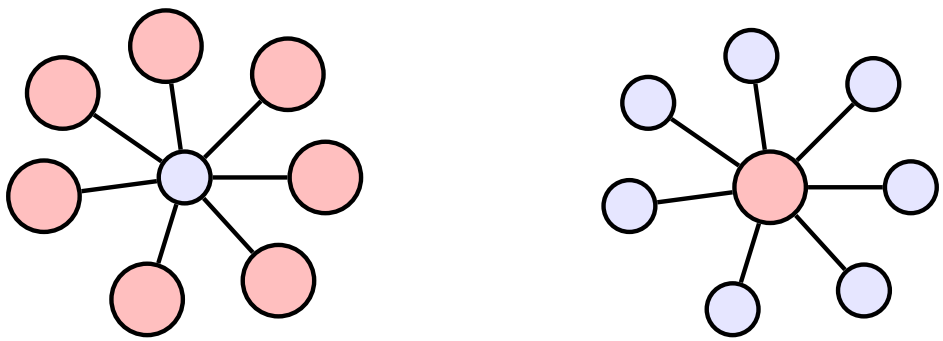
Consider a degree k node:

- If its k neighbours are infected, in the next step it gets infected with probability $1 - (1 - p)^k \approx kp$ (for small p).
- If it is infected, it will infect about kp neighbours in the next step.



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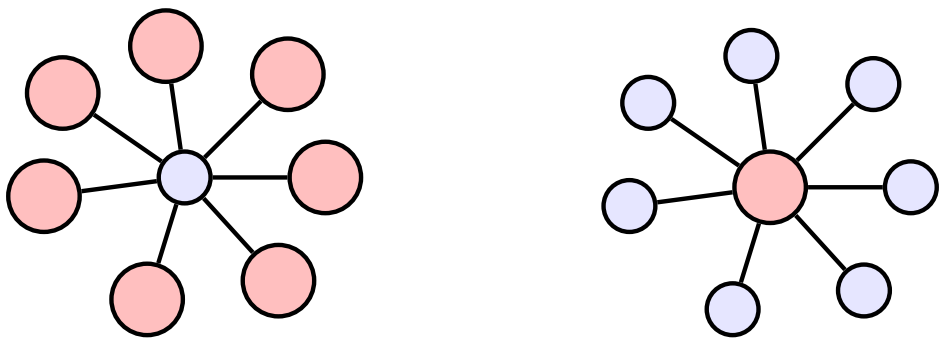


Hubs act as super-spreaders: many chances to receive/transmit infection.

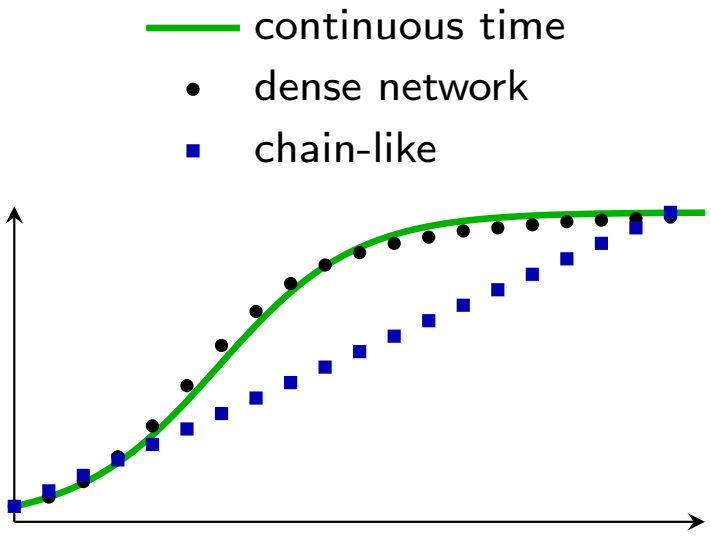
SI: Homogeneous mixing vs Networked Spread

Continuous-time SI, homogeneous mixing

$$\frac{\mathrm{d}i}{\mathrm{d}t} = \beta c\, i(t)\big(1 - i(t)\big)$$

Network SI (discrete time)

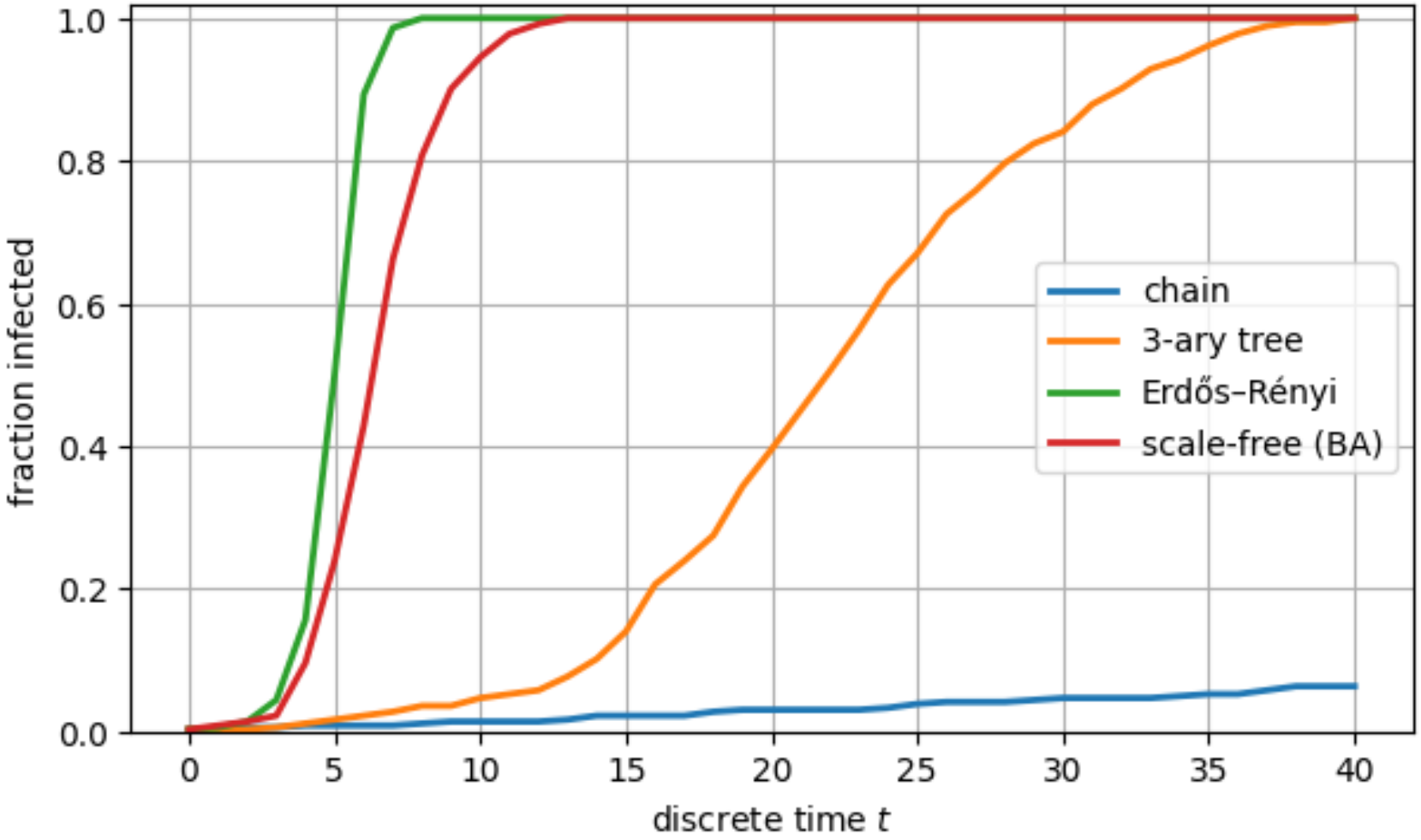
$$i_{t+1} = f(i_t, \text{network structure}, p)$$



Green: homogeneous mixing
Black: SI on a dense network
Blue: SI on a sparse / chain-like network

Epidemic spread depends on topology

Consider four different graphs on 364 nodes.



Note that the spread on the ER graph is faster than on the BA graph! In the colab you can play with different parameters.

Rumor spreading and network SIR

SIR on a Network (Discrete Time)

Network SIR (discrete time). At each time step:

- each infected node infects a susceptible neighbour with probability p;
- each infected node recovers with probability r;
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The dynamics depends strongly on the network:

- on dense networks the infection can reach many nodes before recovery happens;
- on sparse or tree-like networks the infection may die out quickly simply because it does not reach enough new nodes.

This is the network analogue of SIR: infections travel along edges, while recovery removes nodes from the process permanently.

Rumor Spreading on a Network

Rumor states (Maki–Thompson model)

- **Ignorant (I)** – has not heard the rumor.
- **Spreader (S)** – knows it and wants to share it.
- **Stifler (R)** – knows it but has lost interest.

Rumors behave differently from diseases:

- People do not "recover" automatically.
- They stop spreading only when the rumor is no longer exciting.
- Losing interest is triggered by meeting someone who already knows it.

Interpretation: if I am excitedly tell you a rumor and you say "yes, I've heard," I immediately feel less motivated to repeat it further – I become a stifler.

Rumor Contact Rule

At each discrete step, each spreader S selects one neighbour:

- If the neighbour is **Ignorant (I)**:

$$I \longrightarrow S \quad \text{(rumor spreads)}$$

- If the neighbour is **Spreader (S)** or **Stifler (R)**:

$$S \longrightarrow R \quad \text{(caller loses interest)}$$

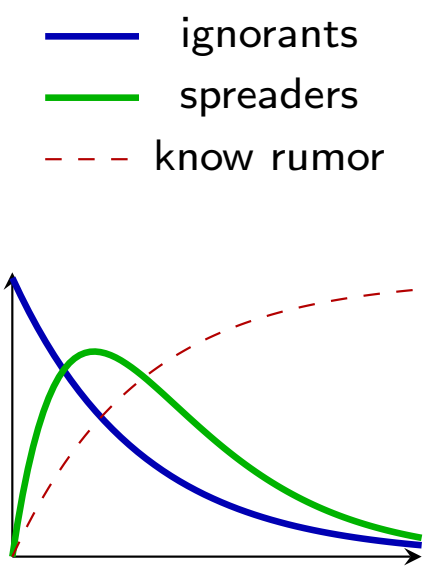
This makes the model qualitatively different from SIR.

Rumor eventually dies because all spreaders become stiflers.

Rumor Spreading: Typical Behaviour

1. Initially: one spreader, many ignorants.
2. Number of spreaders increases rapidly.
3. Eventually most nodes know the rumor (spreaders + stiflers).
4. Spreaders disappear as they repeatedly meet people who already know it.

At the end: almost all are informed, but no active spreaders remain.

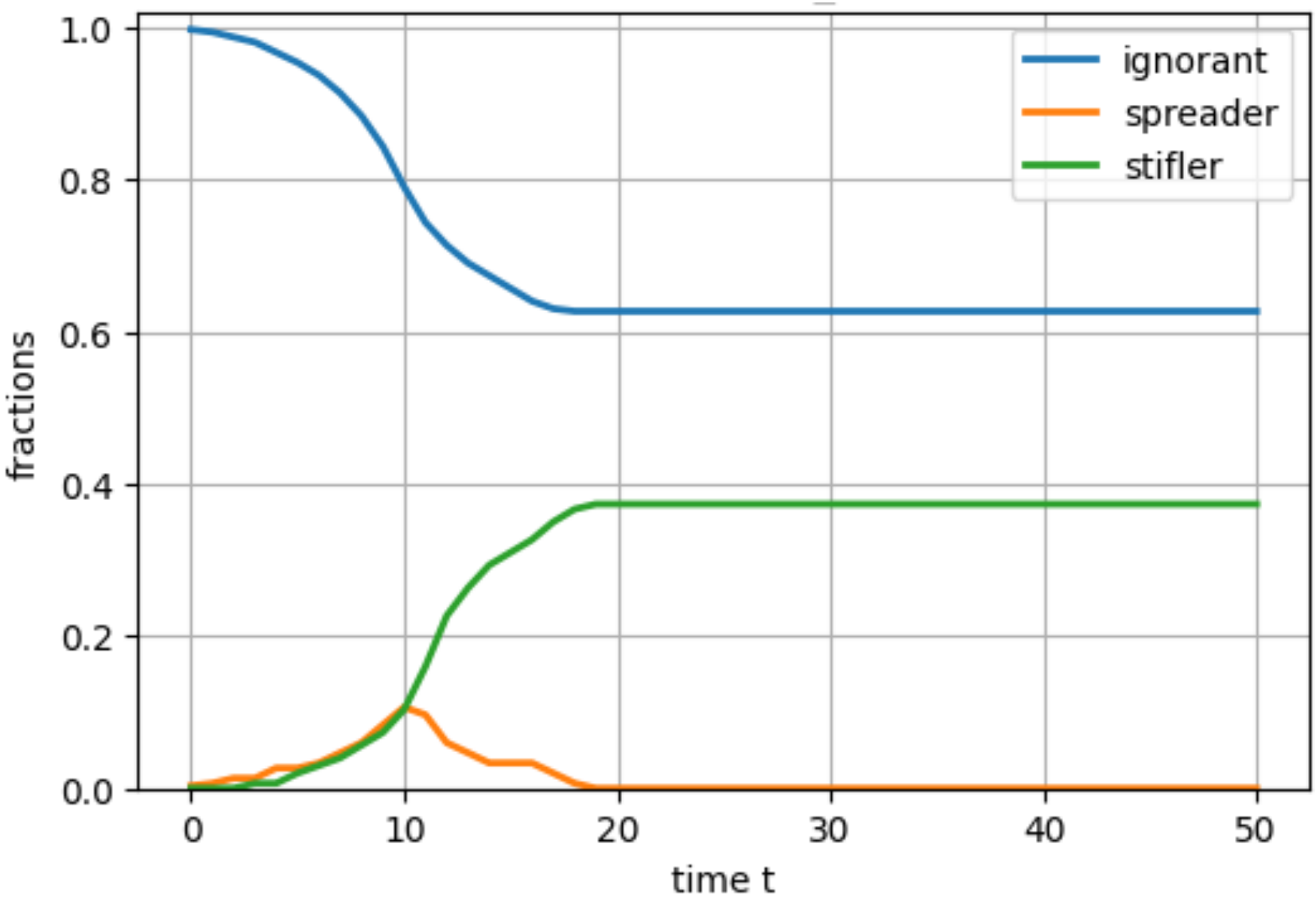


Stylised curves from discrete-time simulations.

We will look at some Colab simulations.

Simulated plot

Rumor spread on $\mathrm{ER}(n = 300, p = 0.05)$.



Summary: Epidemics on Networks

- Classical SI/SIS/SIR models are simple but assume homogeneous mixing.
- On networks, infection spreads via discrete updates along edges.
- Hubs accelerate SI/SIR spreading.
- Rumor spreading is similar to SIR but with "loss of interest."
- The final epidemic size depends strongly on network topology.

One infection rule + different networks $\Rightarrow$ very different speeds, peaks, and outcomes.