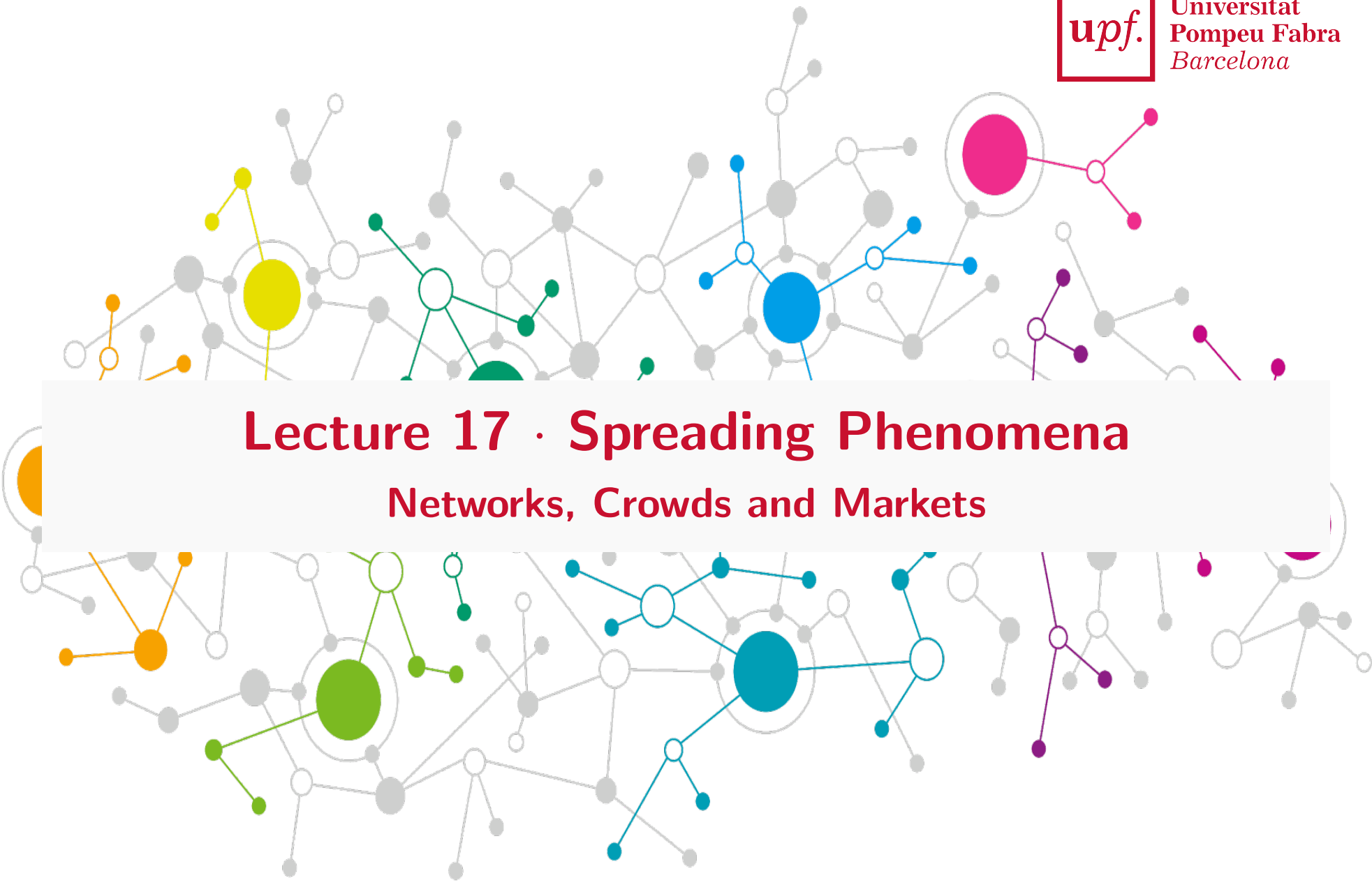


upf. Universitat Pompeu Fabra Barcelona

Lecture 17 · Spreading Phenomena

Networks, Crowds and Markets

Today's Lecture

1. Spreading phenomena
2. Modelling hypotheses
3. Epidemic modelling
   - 3.1 Susceptible-Infected (SI)
   - 3.2 Susceptible-Infected-Susceptible (SIS)
   - 3.3 Susceptible-Infected-Recovered (SIR)
4. Epidemics on networks (rumor spread)

Spreading Phenomena

Despite their differences, many spreading processes follow similar patterns and can be described within a common framework.

PHENOMENA	AGENT	NETWORK
Venereal Disease	Pathogens	Sexual Network
Rumor Spreading	Information, Memes	Communication Network
Diffusion of Innovations	Ideas, Knowledge	Communication Network
Computer Viruses	Malware, digital viruses	Internet
Mobile Phone Virus	Mobile Viruses	Social / Proximity Network
Bedbugs	Parasitic Insects	Hotel–Traveler Network
Malaria	*Plasmodium*	Mosquito–Human Network

In the first part of this lecture the network will not appear explicitly.

Modelling hypotheses

Compartmentalization Hypothesis

Epidemic models classify each individual according to the stage of the disease. A simple classification is:

- **Susceptible (S)** – can become infected,
- **Infectious (I)** – currently contagious,
- **Recovered (R)** – has cleared the infection and is immune.

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

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

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

More detailed models add states such as **latent** individuals, who have been exposed but are not yet contagious.

Homogeneous Mixing Hypothesis

Homogeneous mixing assumes that each individual has the same chance of coming into contact with any given infected individual.

This hypothesis removes the need to know the precise contact network on which the disease spreads. Instead, we assume that anyone can infect anyone else (as if the population formed a complete graph).

Susceptible-Infected Model (SI)

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

Susceptible-Infected Model (SI)

Consider a disease that spreads in a population of N individuals.

- $S(t)$ is the number of individuals who are susceptible at time t.
- $I(t)$ is the number of individuals that have been infected by time t.

Susceptible-Infected Model (SI)

Consider a disease that spreads in a population of N individuals.

- $S(t)$ is the number of individuals who are susceptible at time t.
- $I(t)$ is the number of individuals that have been infected by time t.

The question we ask is:

If a single individual becomes infected at time $t = 0$ (i.e. $I(0) = 1$), how many individuals will be infected at some later time t?

Susceptible-Infected Model (SI)

Consider a disease that spreads in a population of N individuals.

- $S(t)$ is the number of individuals who are susceptible at time t.
- $I(t)$ is the number of individuals that have been infected by time t.

The question we ask is:

If a single individual becomes infected at time $t = 0$ (i.e. $I(0) = 1$), how many individuals will be infected at some later time t?

Assume:

- Each individual makes on average c contacts per unit time, $c > 0$.
- The probability that the disease is transmitted from an infected to a susceptible individual in a unit time is β.
  - Over a small time interval Δt this probability is $\beta \Delta t + o(\Delta t)$.

Susceptible-Infected Model (SI)

At each time t there are $I(t)$ infected individuals.

Each of them gets in touch with $c\frac{S(t)}{N}$ susceptible individuals.

Each new person gets infected in time Δt with probability $\beta\Delta t + o(\Delta t)$.

Susceptible-Infected Model (SI)

At each time t there are $I(t)$ infected individuals.

Each of them gets in touch with $c\frac{S(t)}{N}$ susceptible individuals.

Each new person gets infected in time Δt with probability $\beta\Delta t + o(\Delta t)$.

We conclude:

$$I(t+\Delta t) - I(t) \;=\; I(t)c\frac{S(t)}{N}(\beta\Delta t + o(\Delta t)) \;=\; I(t)c\frac{S(t)}{N}\beta\Delta t + o(\Delta t).$$

Taking the limit $\Delta t \to 0$, we get the expression for the derivative.

$$\frac{\mathrm{d}I(t)}{\mathrm{d}t} \;=\; I(t)c\,\frac{S(t)}{N}\beta\,.$$

Susceptible-Infected Model (SI)

Recall: $\frac{\mathrm{d}I(t)}{\mathrm{d}t} = \beta c \frac{S(t)I(t)}{N}$. Denote $s(t) := S(t)/N$ and $i(t) := I(t)/N$.

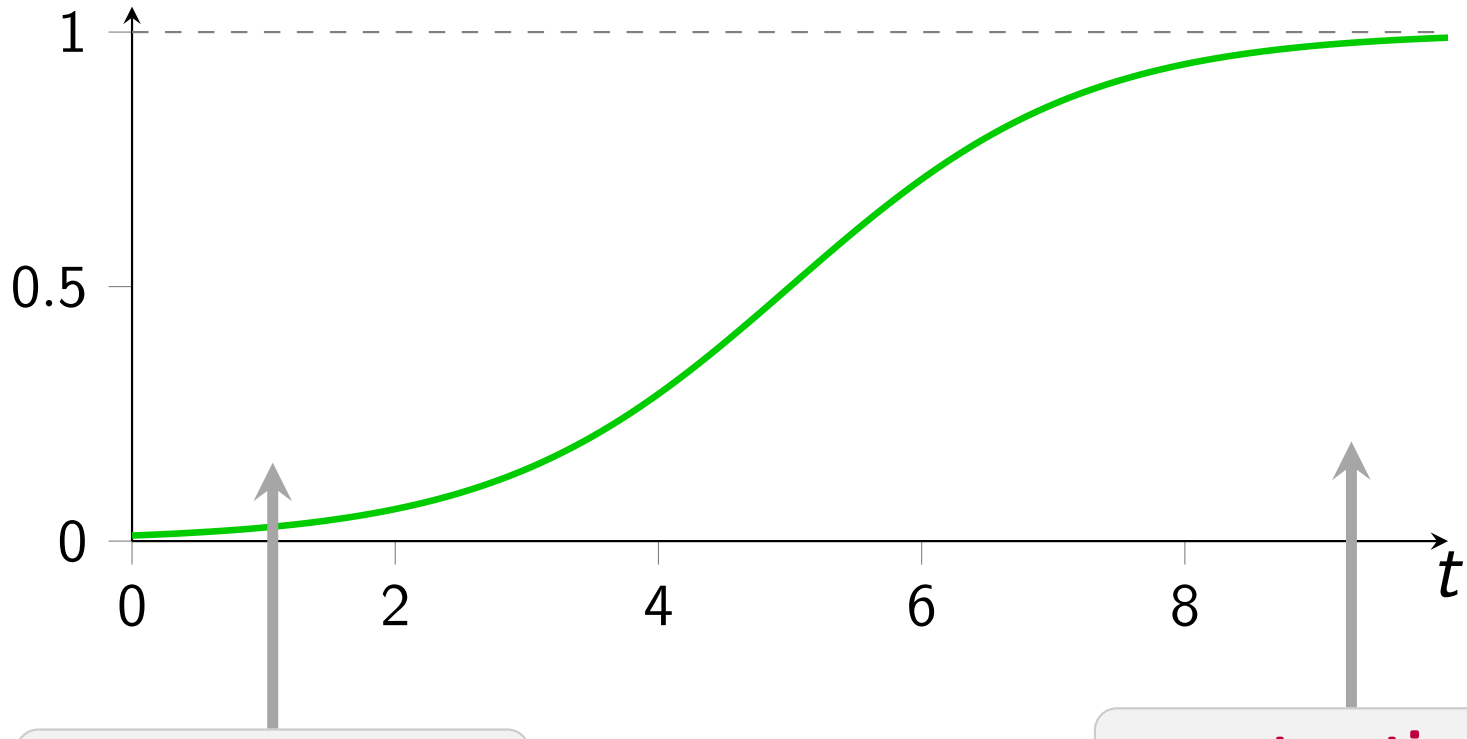
The equation becomes

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

with solution

$$i(t) \;=\; \frac{i_0 e^{\beta c t}}{1 - i_0 + i_0 e^{\beta c t}},$$

where $i_0 = i(0)$. Verify this!



exponential regime

If i is small,

$$i \approx i_0 e^{\beta c t}$$

saturation regime

As $i \to 1$,

$$\frac{\mathrm{d}i}{\mathrm{d}t} \to 0$$

In the SI model the epidemic ends only when everyone is infected.

Susceptible-Infected-Susceptible Model (SIS)

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

Motivation for a different model

Most pathogens are eventually defeated by the immune system or by treatment. We therefore allow individuals to recover at rate μ.

- The probability of recovering in time Δt is $\mu \Delta t + o(\Delta t)$.

Once an individual recovers:

- they become susceptible again;
- they cease to spread the disease.

Susceptible-Infected-Susceptible Model (SIS)

The equation to be solved is now

$$\frac{\mathrm{d}i}{\mathrm{d}t} = \beta c\big(1 - i(t)\big)i(t) - \mu\, i(t) = (\beta c - \mu)\, i(t) - \beta c\, i^2(t).$$

Set

$$r := \beta c - \mu, \qquad i^* := 1 - \frac{\mu}{\beta c}.$$

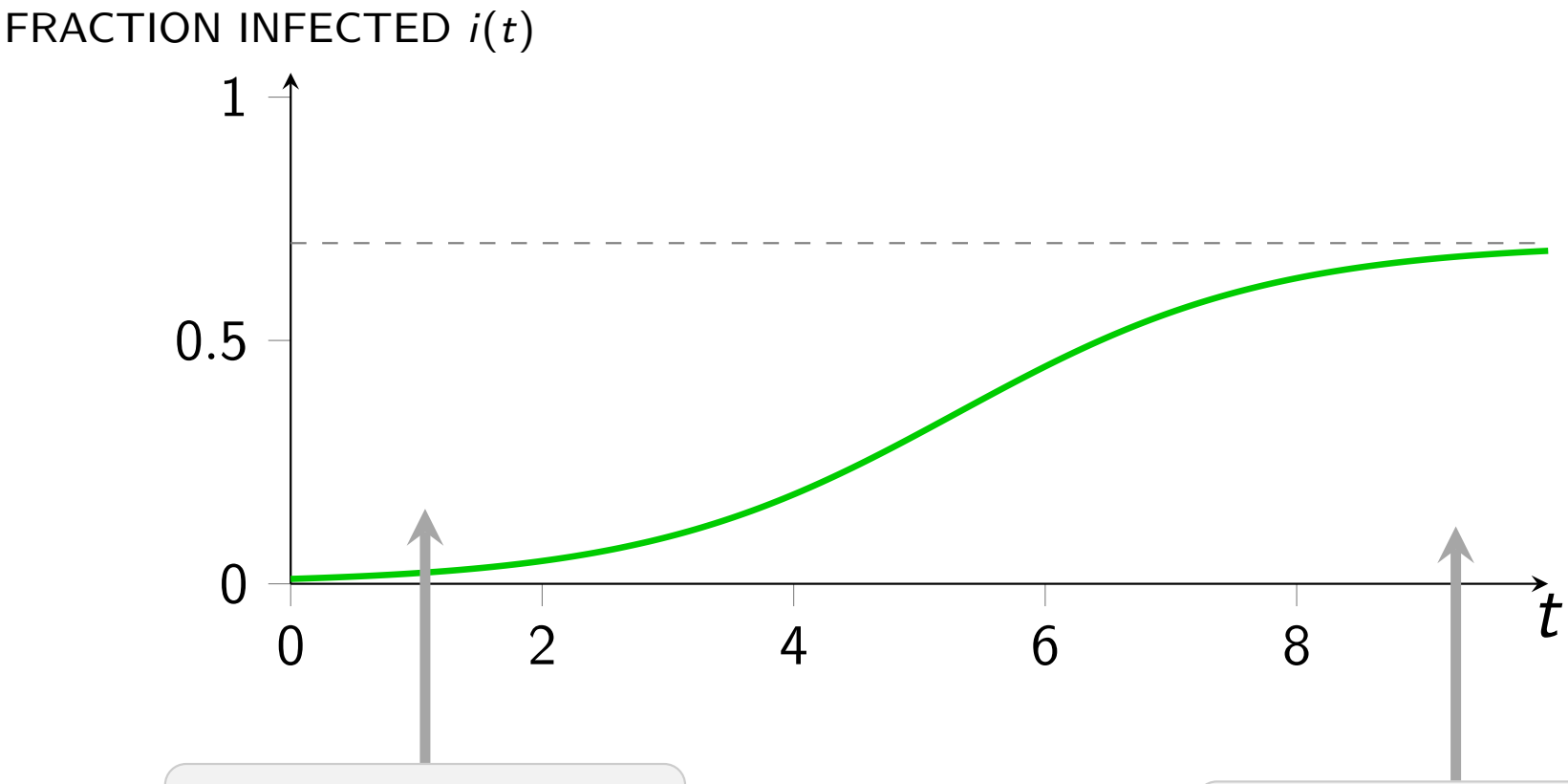
Case $\beta c > \mu$ (endemic regime). Then $r > 0$ and $i^* \in (0, 1)$. For $i(0) = i_0 \in (0, 1)$ the solution is

$$i(t) = \frac{i^*}{1 + \left(\frac{i^* - i_0}{i_0}\right) e^{-rt}},$$

which increases monotonically to the equilibrium i^*.

Case $\beta c \leq \mu$ (disease-free regime). The only equilibrium in $[0, 1]$ is $i = 0$, and every solution with $0 < i_0 < 1$ decreases monotonically to 0.

Endemic state: $\mu < \beta c$



exponential outbreak

If i is small,

$i \approx i_0 e^{(\beta c - \mu)t}$

endemic state

$i(\infty) = 1 - \frac{\mu}{\beta c}$

The Basic Reproductive Number

Basic reproductive number

$$R_0 := \frac{\beta c}{\mu}$$

is the average number of new infections caused by one infected individual in an otherwise susceptible population.

- We can show that $1/\mu$ is the average infectious period.

The Basic Reproductive Number

Basic reproductive number

$$R_0 := \frac{\beta c}{\mu}$$

is the average number of new infections caused by one infected individual in an otherwise susceptible population.

- We can show that $1/\mu$ is the average infectious period.

If $R_0 > 1$, the epidemic reaches an endemic state.

- If each infected individual infects on average more than one healthy person, the pathogen is poised to spread and persist.

If $R_0 < 1$ the epidemic dies out.

- If each infected individual infects on average fewer than one additional person, the pathogen cannot persist in the population.

Examples of R_0 for some diseases

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

Likely, we will never about any pathogen with $R_0 < 1$.

Exercise

Given the following function in the SIS model of an epidemic that affects 50 million people:

$$i(t) = \frac{0.125e^{0.35t}}{1 + 0.25e^{0.35t}},$$

where t is time in years.

a) What is the state of the epidemic (endemic or disease-free)?

b) How many people were infected at the beginning of the disease?

c) At what time will more than 20 million people be infected?

d) What is the stationary state? How long will it take to get close to the stationary state?

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)

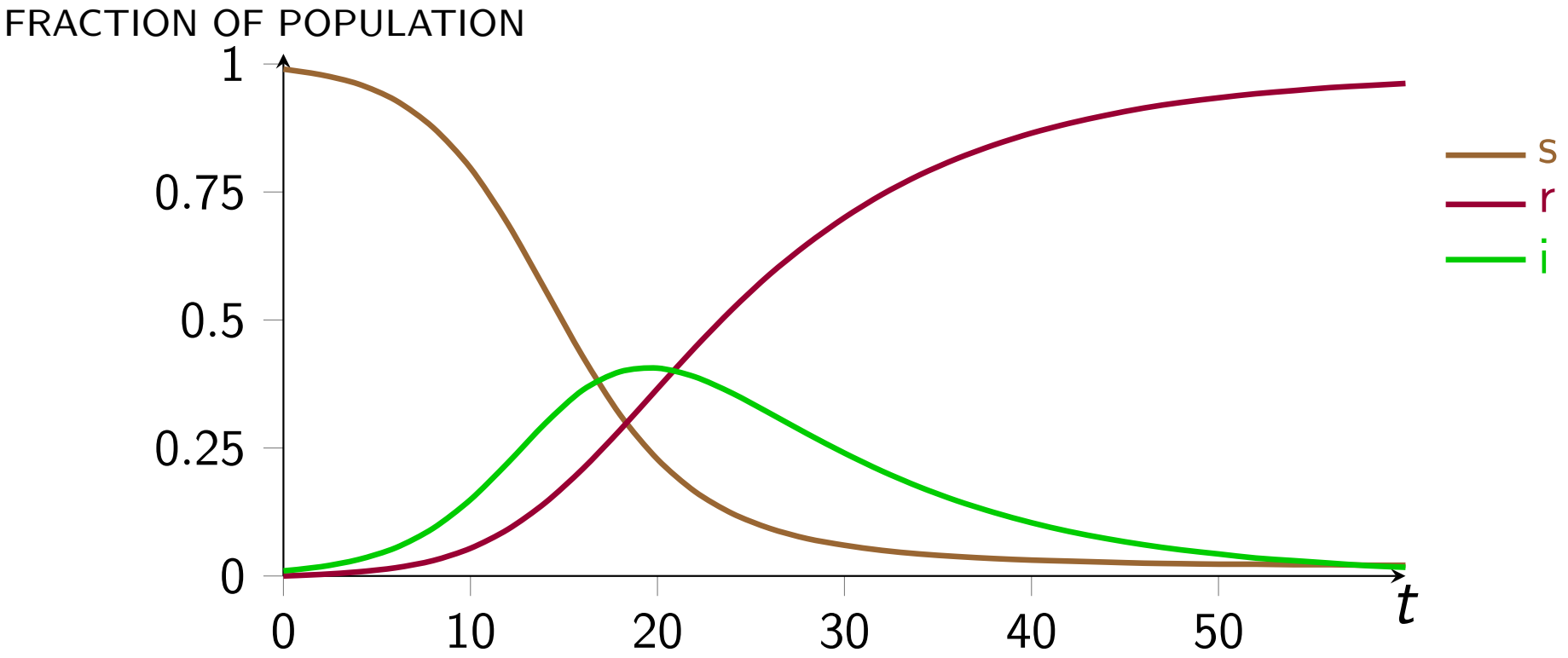
In this model $s(t) + i(t) + r(t) = 1$, where:

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

- 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.

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.

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)

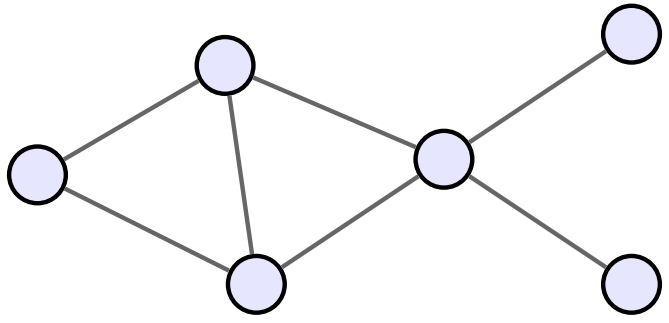
- Nodes = individuals; edges = possible contacts.
- Each node is Susceptible (S) or Infected (I).
- Time steps $t = 0, 1, 2, \ldots$
- At each step, every infected node infects each susceptible neighbour with probability p.
- Once infected, a node never recovers.

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

Susceptible-Infected (SI) Model on a Network

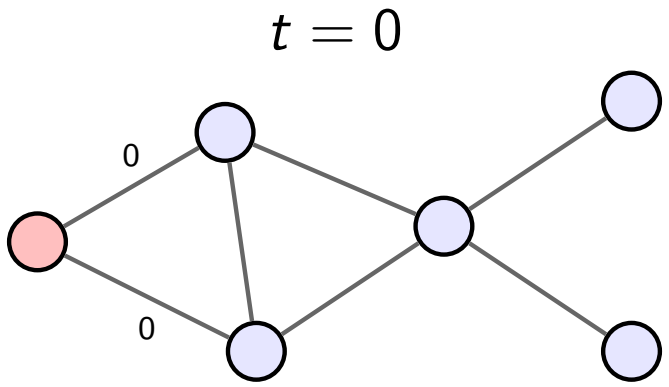
- Start at $t = 0$ with a single infected node.
- For each t, look at every edge connecting an **infected** node to a **susceptible** neighbour.
- Along each such edge, independently, infection is transmitted with probability p (coin: 1 = infection, 0 = no infection).
- All new infections become active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

(nobody infected yet)



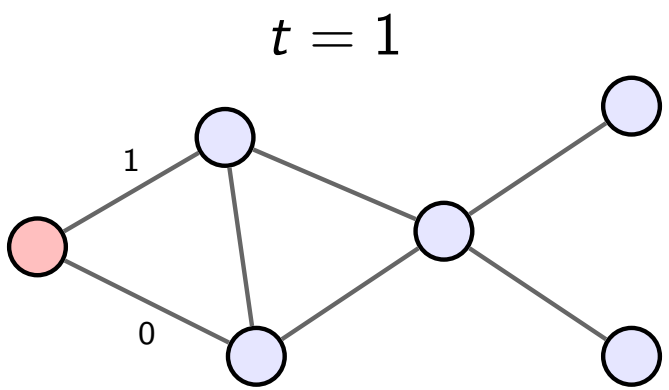
Susceptible-Infected (SI) Model on a Network

- 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.



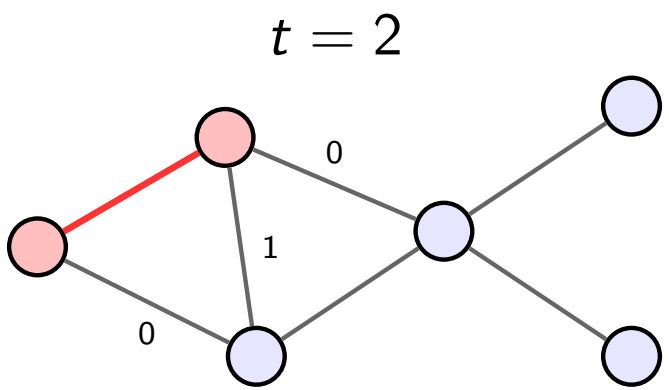
Susceptible-Infected (SI) Model on a Network

- 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.



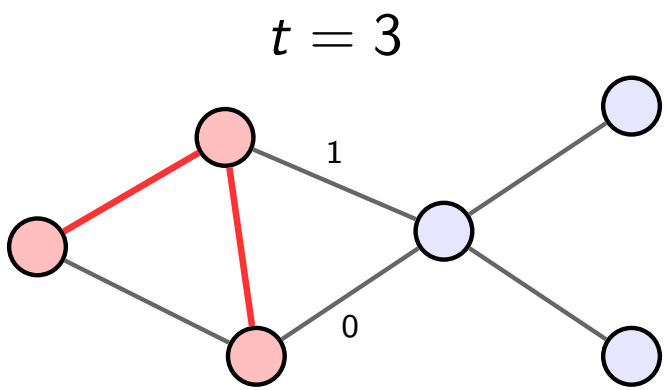
Susceptible-Infected (SI) Model on a Network

- 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.



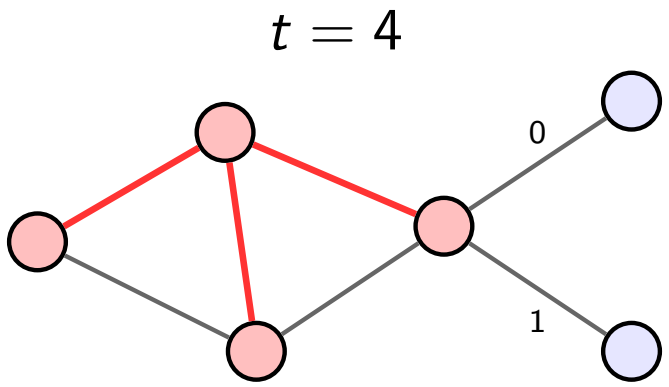
Susceptible-Infected (SI) Model on a Network

- 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.



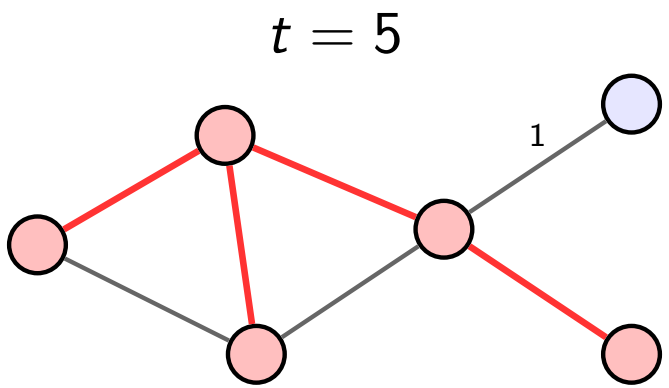
Susceptible-Infected (SI) Model on a Network

- 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.



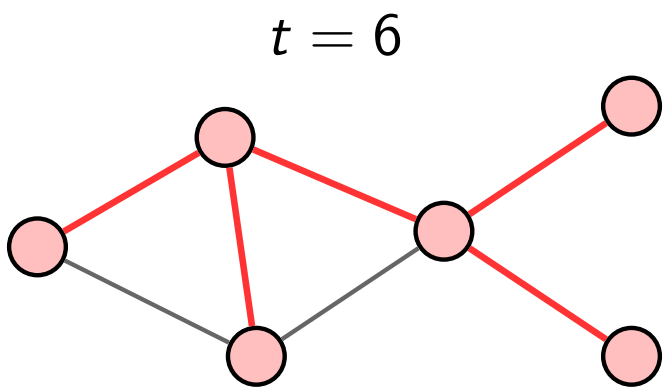
Susceptible-Infected (SI) Model on a Network

- 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.



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.



Why Degrees Matter

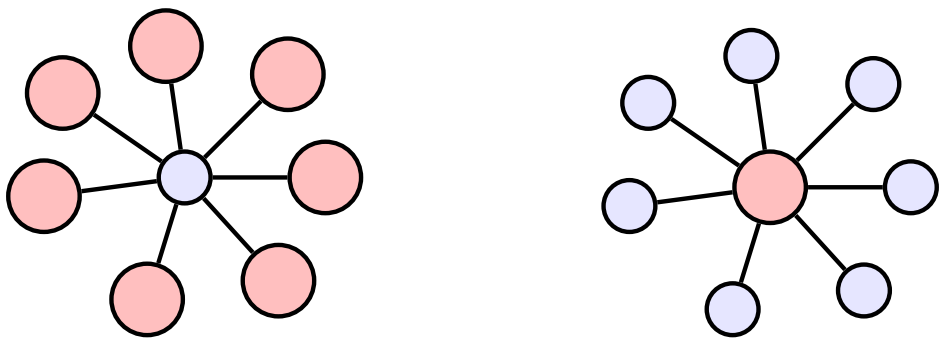
High-degree nodes ("hubs") tend to get infected early and then infect many neighbours quickly.

Why Degrees Matter

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

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.

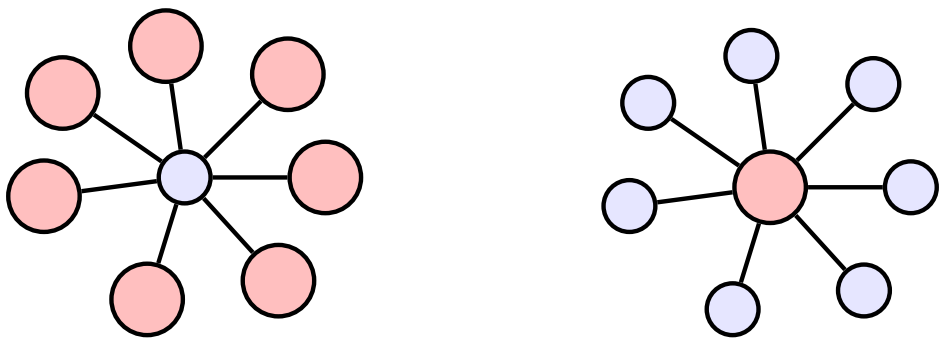


Why Degrees Matter

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

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.



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

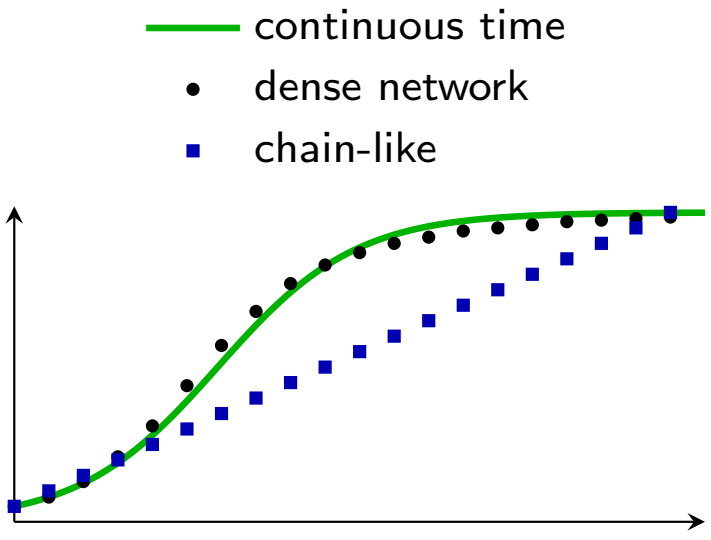
SI: Homogeneous mixing vs Networked Spread

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)

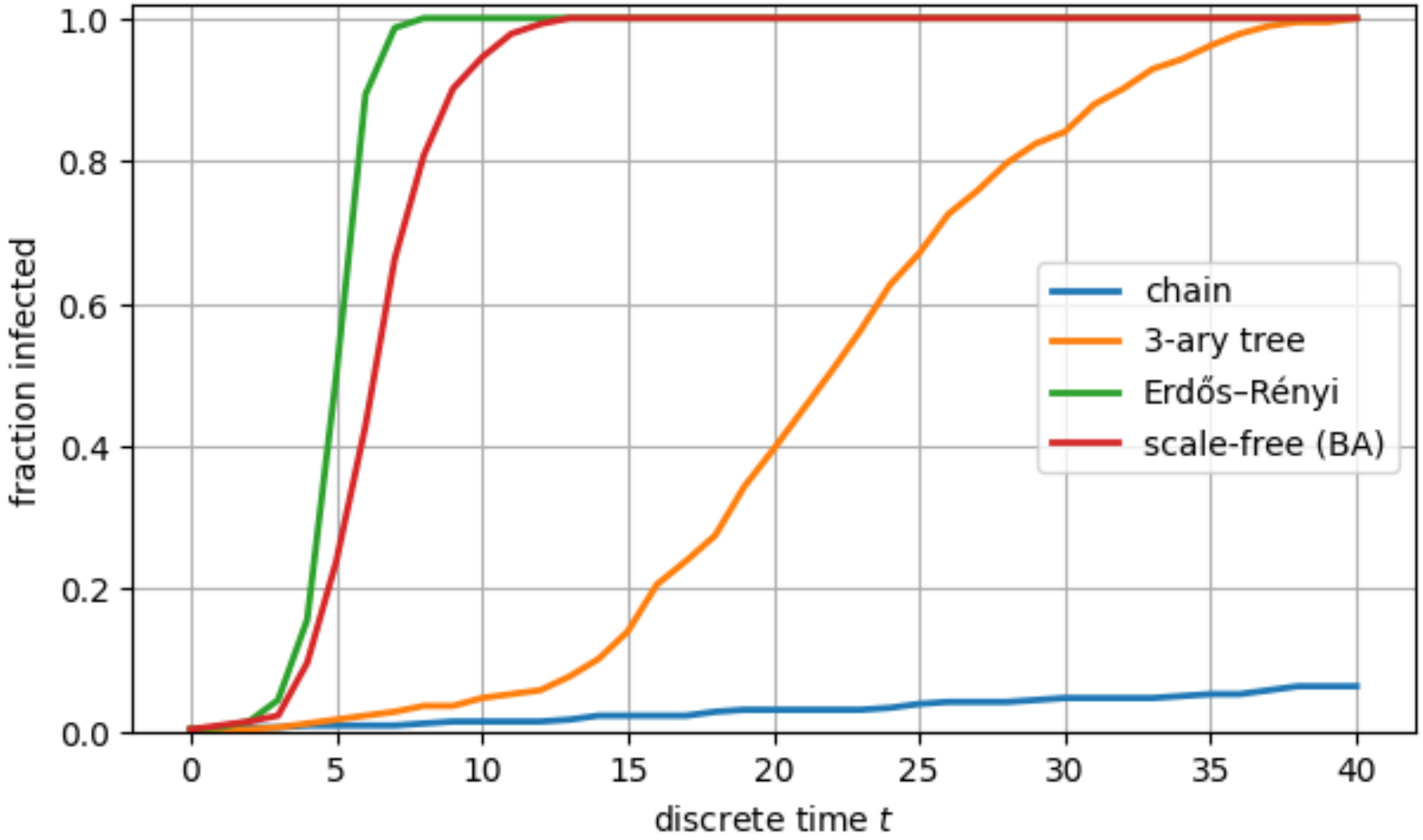
$$i_{t+1} = f(i_t, \text{network structure}, p)$$



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;
- recovered nodes never get infected again.

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;
- recovered nodes never get infected again.

The dynamics depend 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 $\rightarrow$ 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)**:

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

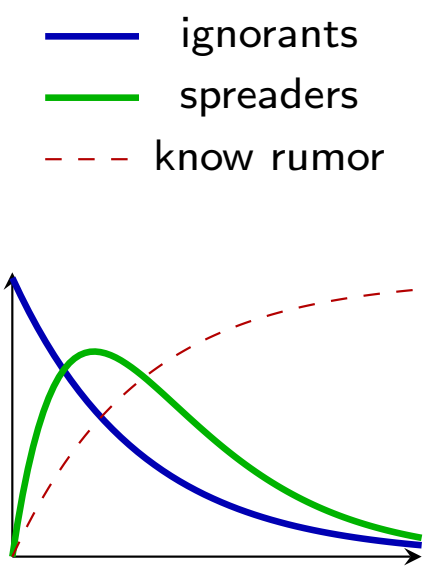
This makes the model qualitatively different from SIR.

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.

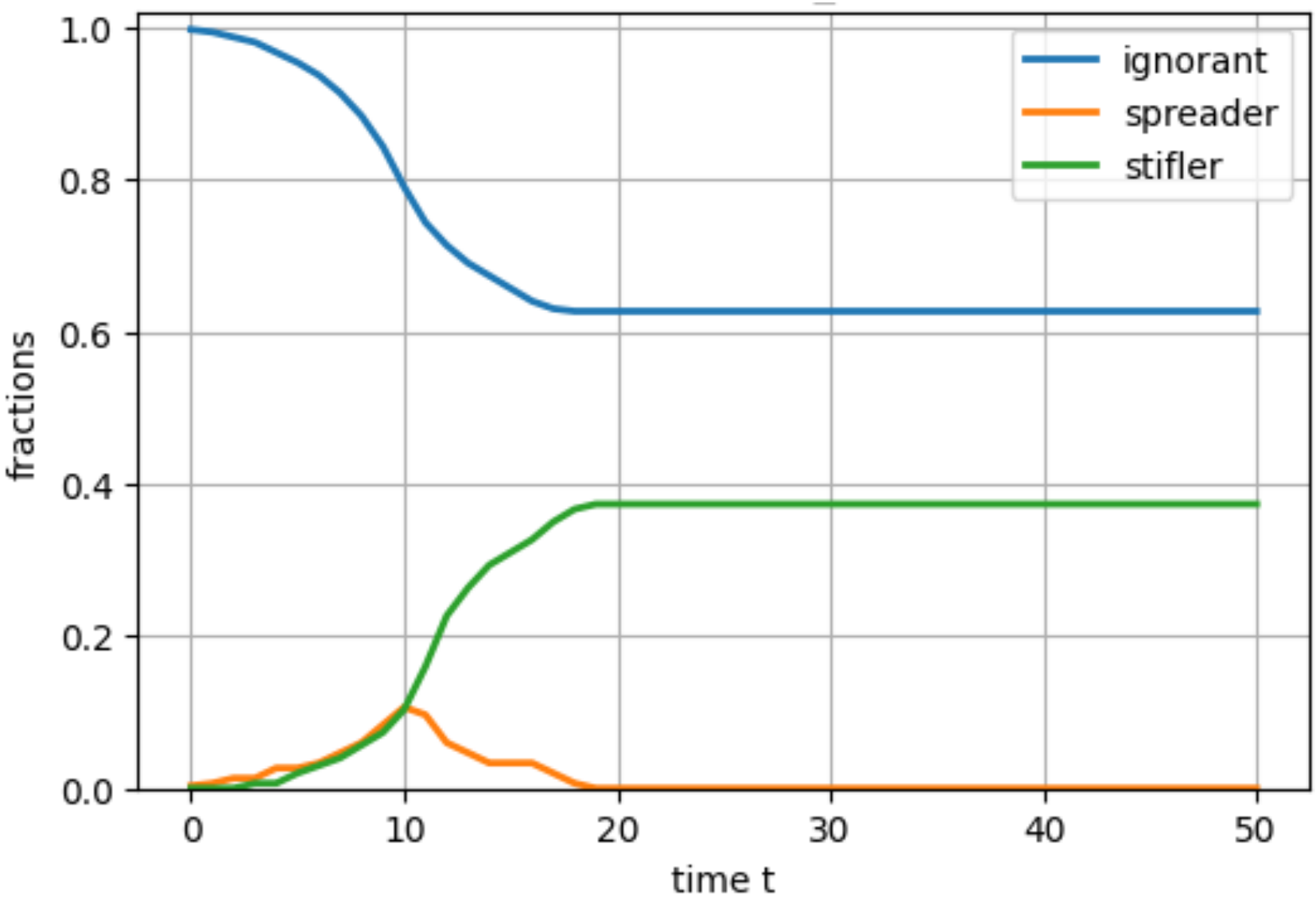


Stylised curves from discrete-time simulations.

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.