

Michele Tizzani - Università degli Studi di Torino (Italy)

Lecture 13.ns09

Course: Complex Networks Analysis and Visualization
Sub-Module: NetSci



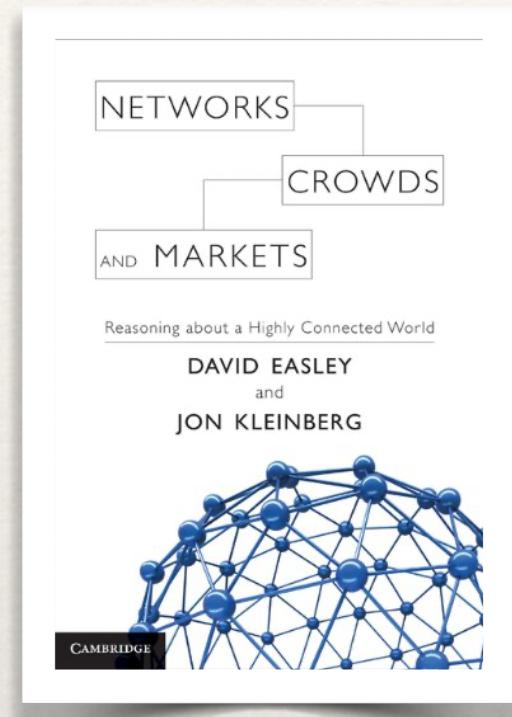
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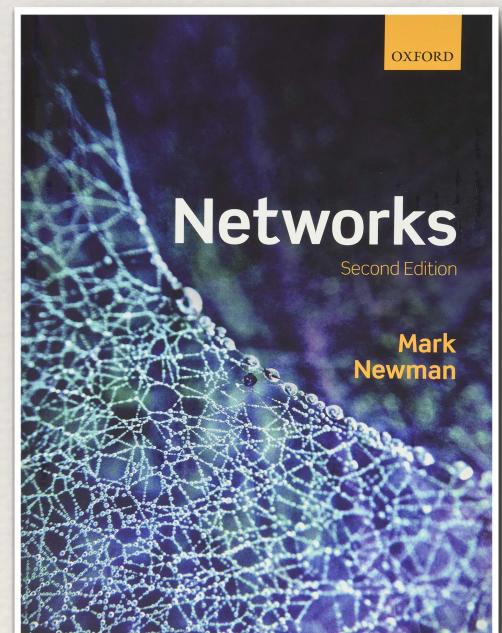
Epidemics

References



[ns2] Chapter 21 (21.1 - 21.5, 21.8A)

"Epidemics" —-> <https://www.cs.cornell.edu/home/kleinber/networks-book/networks-book-ch21.pdf>



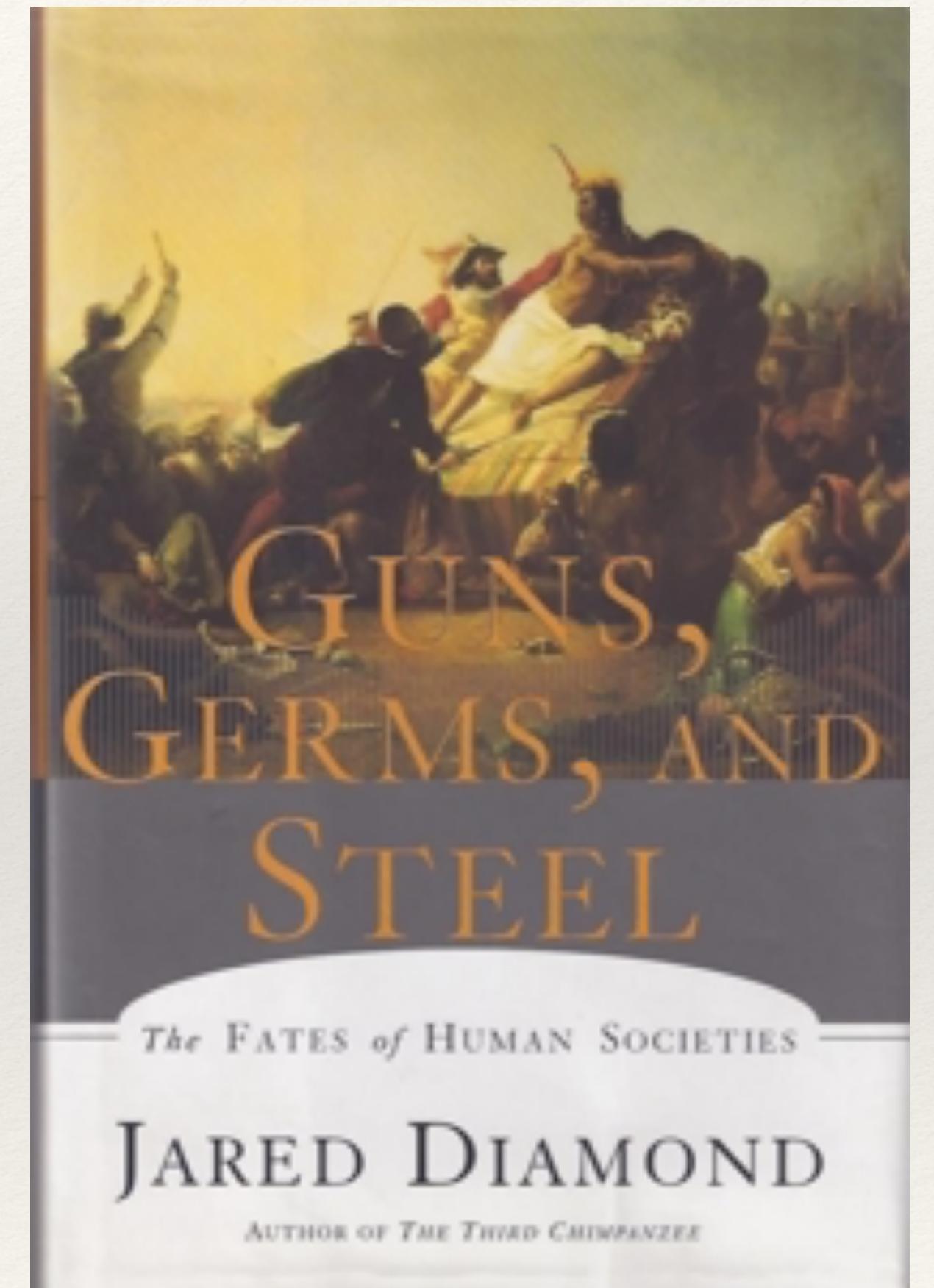
[ns4] Chapter 16 (16.1, 16.2, 16.3)

"Epidemics on Networks"

Please, check your general understanding with exercises at the end of the chapter!

Contagion caused by biological pathogens

- ❖ Contagious diseases had always changed the shape of human history
- ❖ Models that aim to predict their spreading can be of vital importance



Agenda

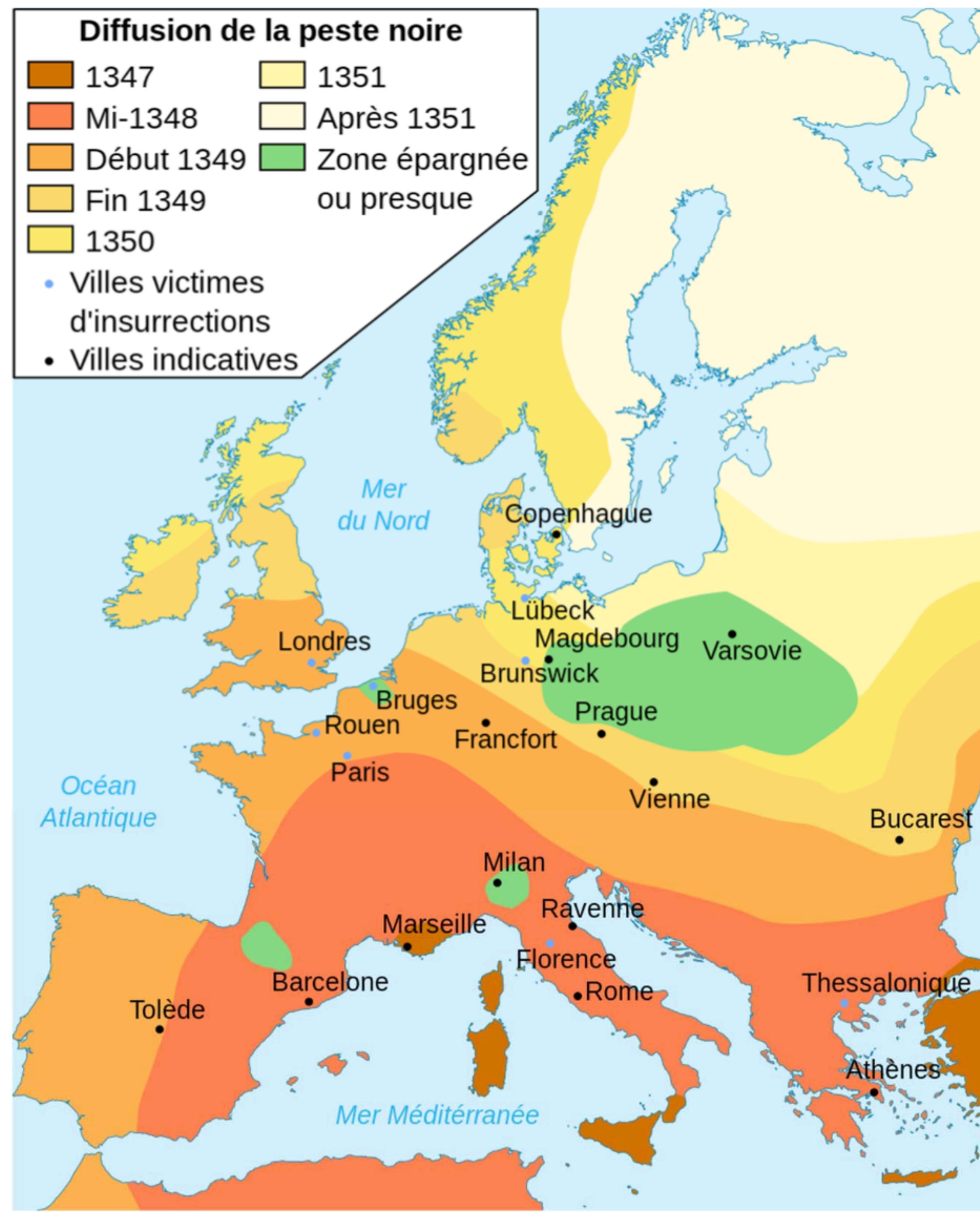
- ❖ Diseases and the Networks that Transmit Them
- ❖ Branching Processes
- ❖ The SIR Epidemic Model
- ❖ The SIS Epidemic Model
- ❖ Synchronization
- ❖ (Transient Contacts and the Dangers of Concurrency) → READ
- ❖ (Genealogy, Genetic Inheritance, and Mitochondrial Eve) → READ
- ❖ A. Analysis of Branching Process
- ❖ (B. Analysis of the Coalescent Process) → READ

Diseases and the Networks that Transmit Them

Pre-1500

[edit]

Death toll (estimate) ▲	Location ▲	Date ▲	Article ▲	Disease ▲	Ref. ▲
25–50 million; 40% of population	Europe, Egypt, West Asia	541–542	Plague of Justinian	plague	[4]
100,000+	Ctesiphon, Persia	627		plague	[5]
	British Isles	664–668	Plague of 664	plague	[6][page needed]
	British Isles	680–686		plague	[6][page needed]
	Byzantine Empire, West Asia, Africa	746–747		plague	[7]
75–200 million; 30–60% of population	Europe, Asia and North Africa	1346–1350	Black Death	plague  Yersinia pestis	[8]
	Europe	250–266	Plague of Cyprian	unknown, possibly smallpox	[3]
75,000–100,000	Greece	429–426 BC	Plague of Athens	unknown, possibly typhus	[1]
5 million; 30% of population in some areas	Europe, Western Asia, Northern Africa	165–180	Antonine Plague	unknown, symptoms similar to smallpox	[2]



WHO officially declared COVID-19 a pandemic on Mar 11, 2020.

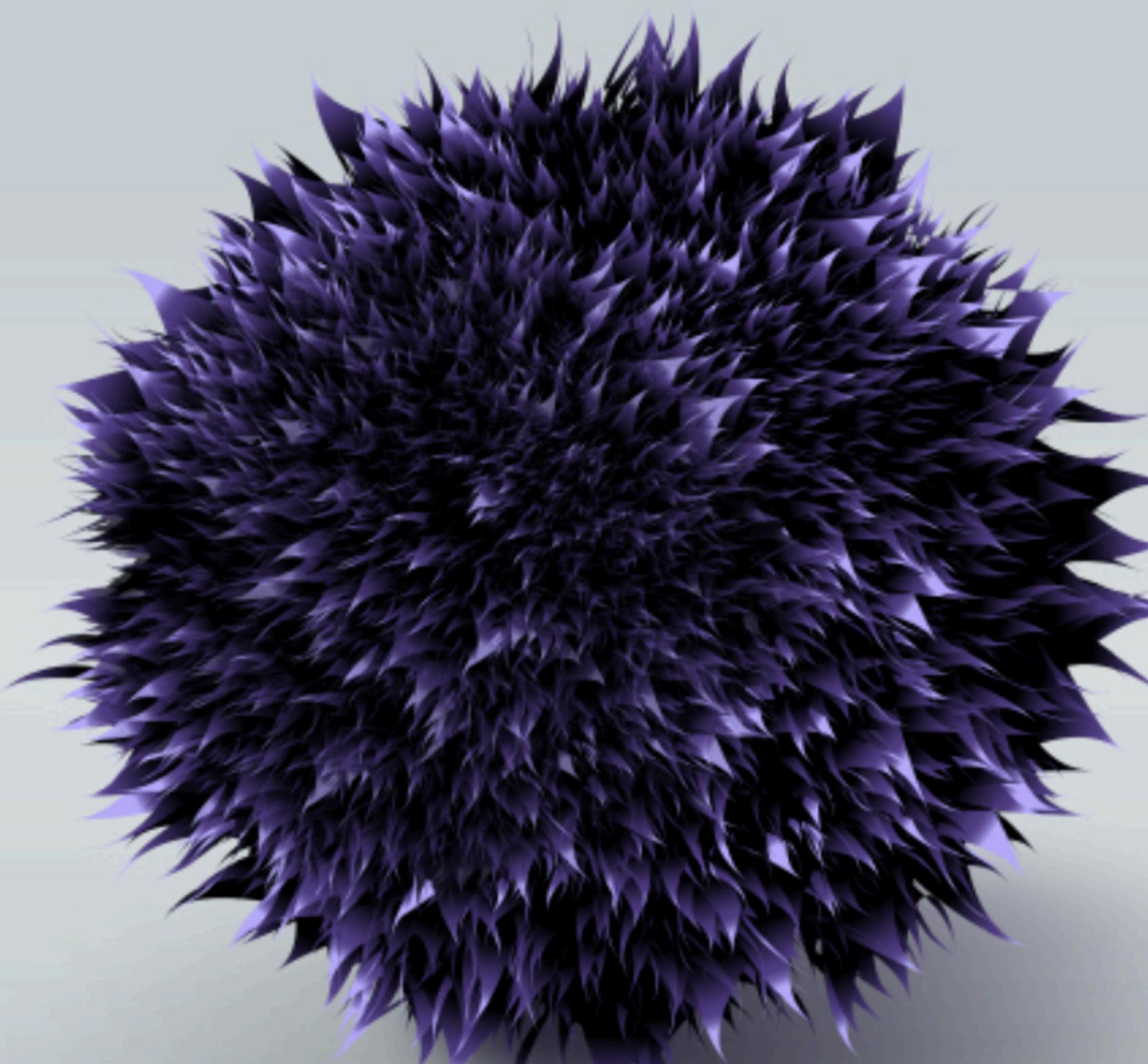
It is hard to calculate and forecast the impact of COVID-19 because the disease is new to medicine, and data is still coming in.

*Johns Hopkins University estimates

DEATH TOLL [HIGHEST TO LOWEST]

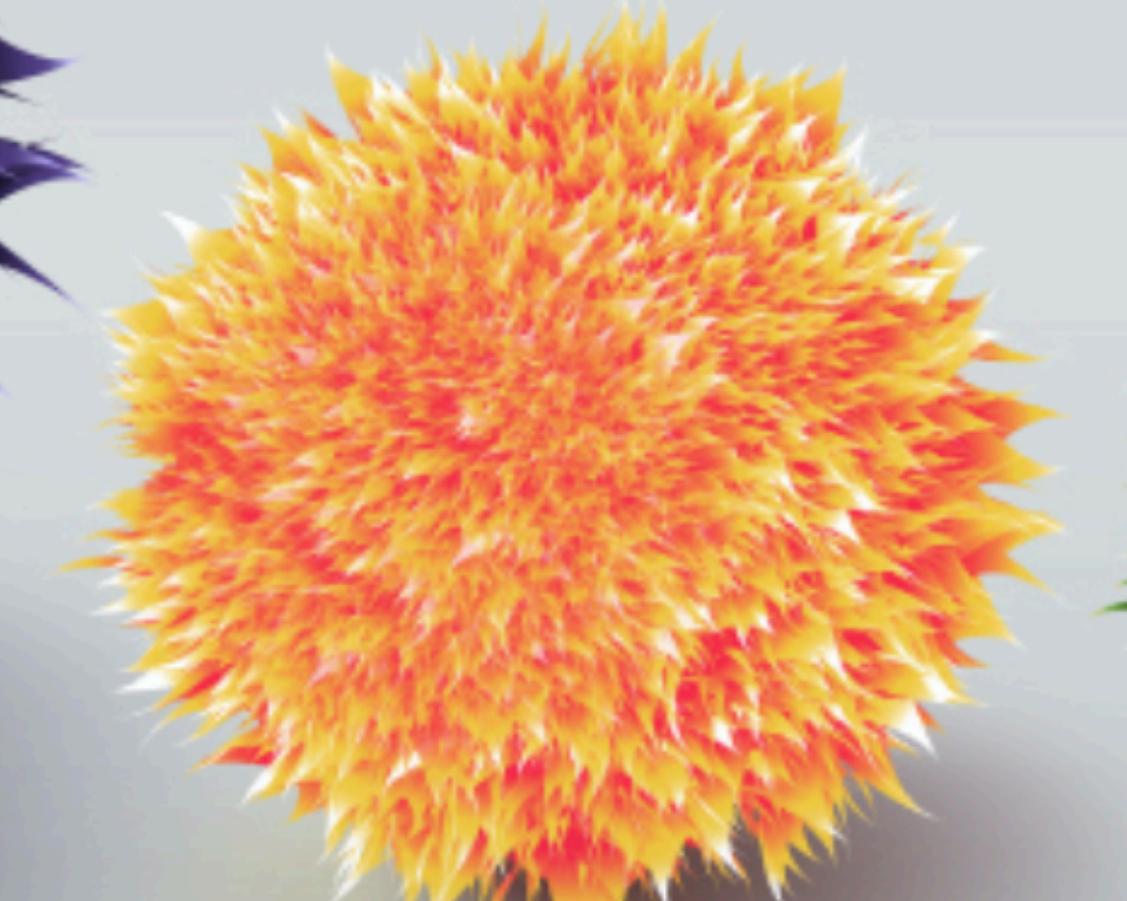
200M

Black Death (Bubonic Plague)
1347-1351



56M

Smallpox
1520



40-50M

Spanish Flu
1918-1919



30-50M

Plague of Justinian
541-542





World Map



NEW



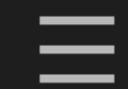
U.S. Map



Critical Trends



COVID-19 Dashboard by the Center for Systems Science and Engineering (CSSE) at Johns Hopki...



Total Confirmed

5,360,841Confirmed Cases by
Country/Region/Sovereign

y

1,632,721 US

347,398 Brazil

344,481 Russia

260,916 United
Kingdom

235,772 Spain

229,858 Italy

182,102 France

Admin0

Last Updated at (M/D/YYYY)

5/24/2020, 7:32:44 PM

188
countries/regions[Lancet Inf Dis Article: Here.](#) [Mobile Version: Here.](#)Lead by JHU CSSE. Technical Support: Esri Living Atlas team and JHU APL. Financial
Support: Bill and Melinda Gates Foundation, Esri, and the University of Maryland.

Global Deaths

343,36497,424 deaths
US36,875 deaths
United Kingdom32,785 deaths
Italy28,752 deaths
Spain

Global Dea...

Global Dea...

US State Level
Deaths, Recovered29,141
deaths, **63,292**
recovered

New York US

11,133
deaths, **24,762**
recovered

New Jersey US

6,304 deaths,
recovered

US Deaths...



Tracking Home

Critical Trends ▾

Global Map

U.S. Map

Data in Motion

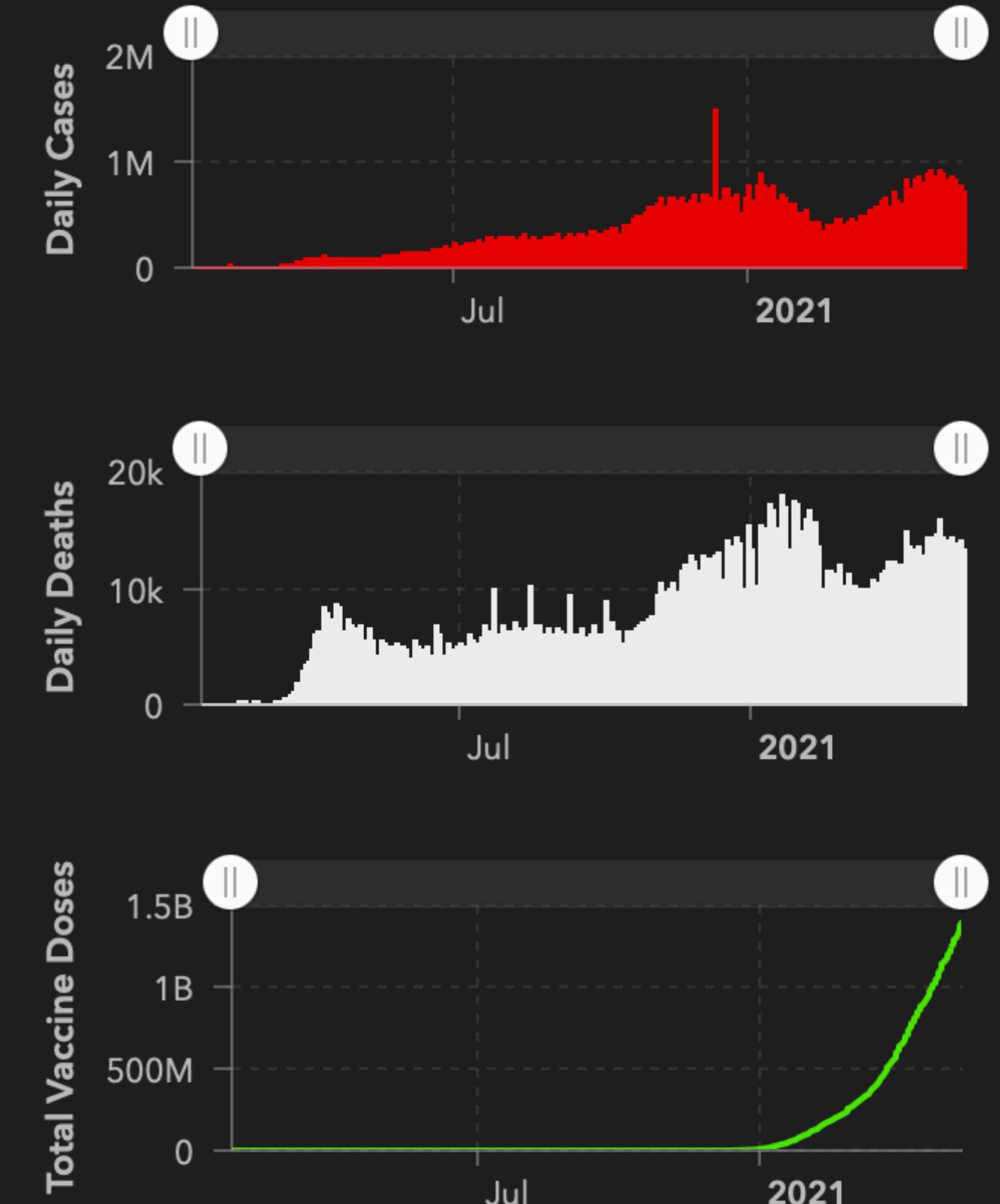
Tracking FAQ

Last Updated at 5/14/2021 3:21 PM

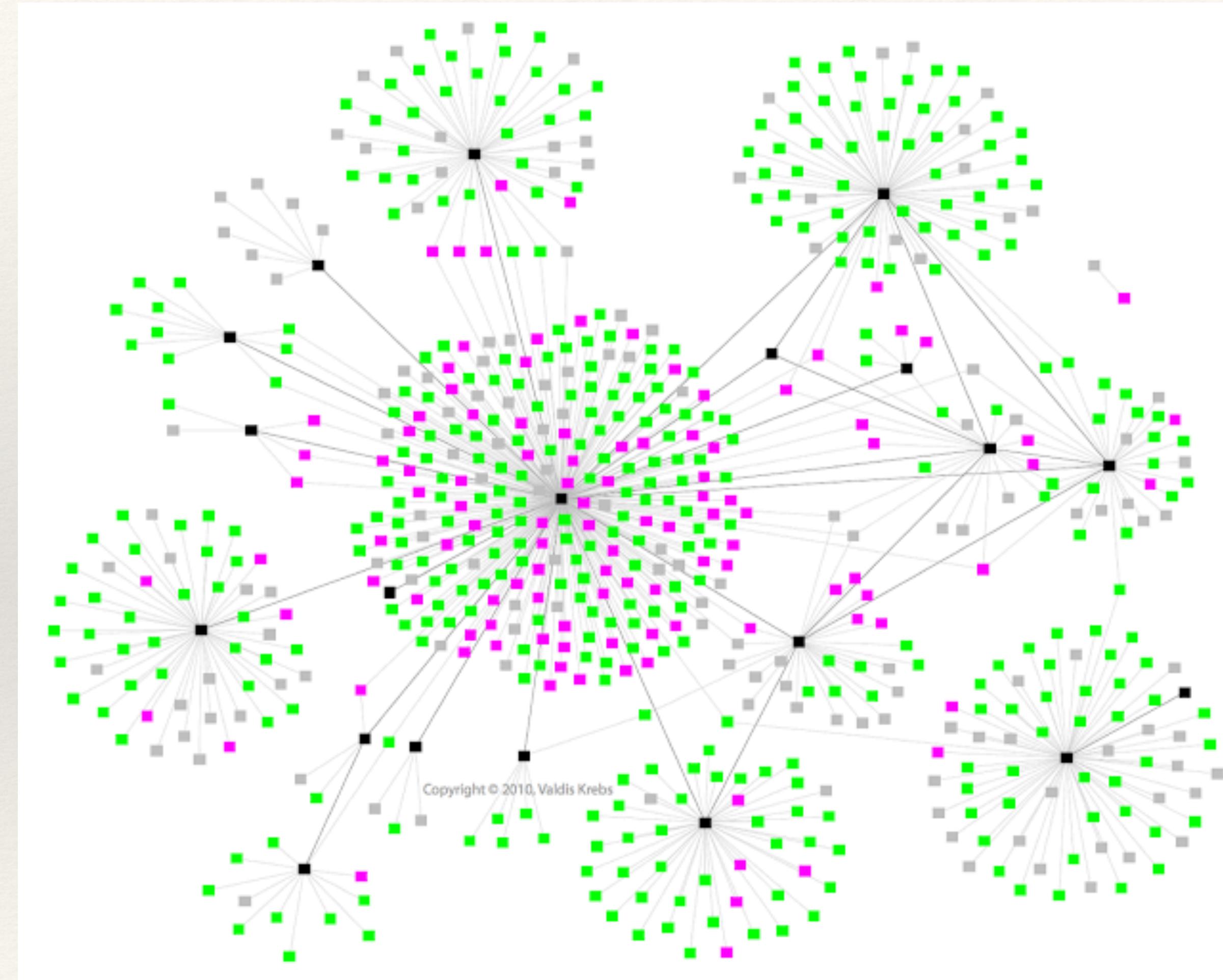
Cases
161,288,384**Deaths**
3,347,154**Vaccine Doses Administered**
1,405,665,427

Cases and Deaths by Country/Region/Sovereignty

32,853,960 584,510	US
24,046,809 262,317	India
15,433,989 430,417	Brazil
5,902,899 107,413	France



Contact networks



Accurately modeling the underlying network is crucial to understanding the spread of an epidemic

CONNECTIONS

3.200 AIRPORTS

martingrandjean.ch



<http://www.martingrandjean.ch/connected-world-air-traffic-network/>

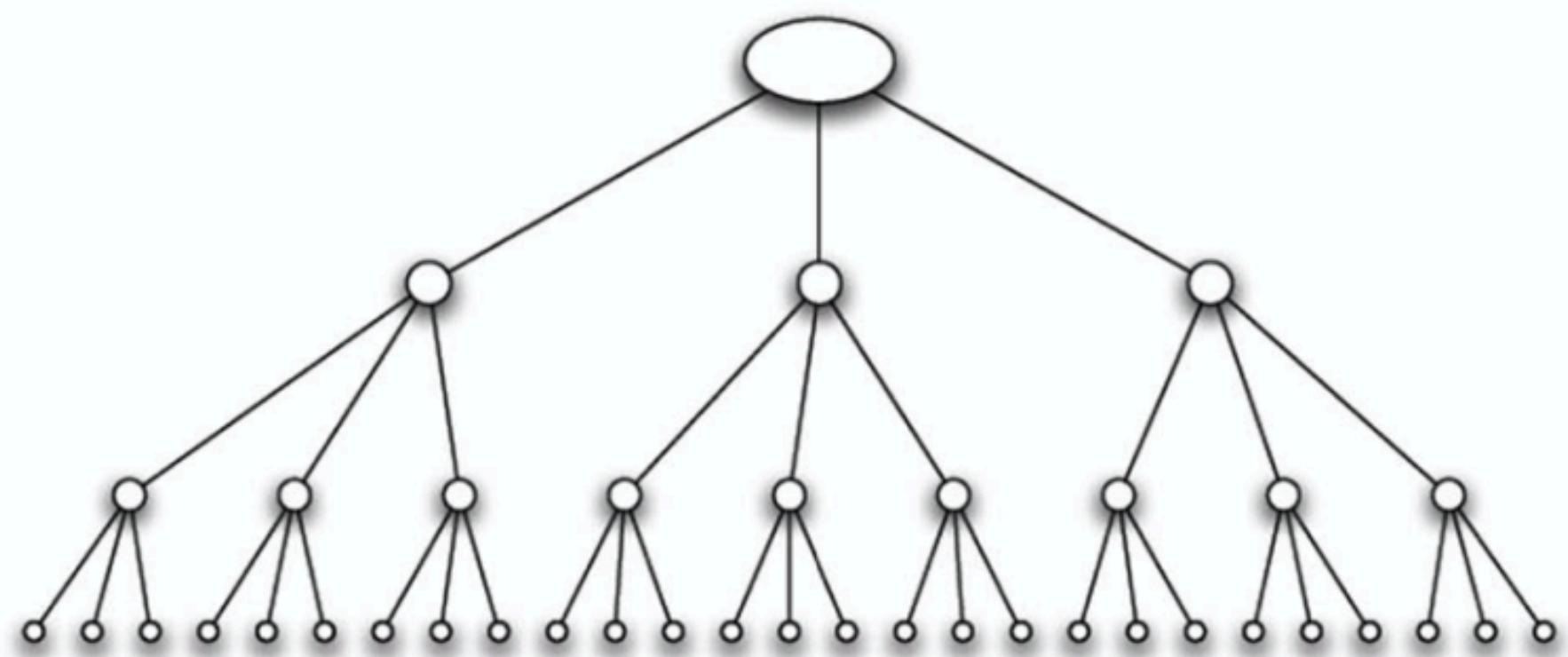
Simple vs complex contagion models

- ❖ Idea, opinions, and behaviors and infectious diseases: they all spread in very profoundly different ways
- ❖ We have already classified contagion between **simple** and **complex**:
 - ❖ both types spread from person to person
 - ❖ with **social contagion**: people makes decision to adopt a new idea or an innovation
 - ❖ with **biological contagion**: it is not dependent on the individual's will, and moreover it is not easily observable at the person-to-person level
- ❖ Infection diseases can be seen as a simple contagion processes; however, due to not trivial observability, it is usually better to adopt **non deterministic models** - i.e., based on *randomness* and *probabilities*

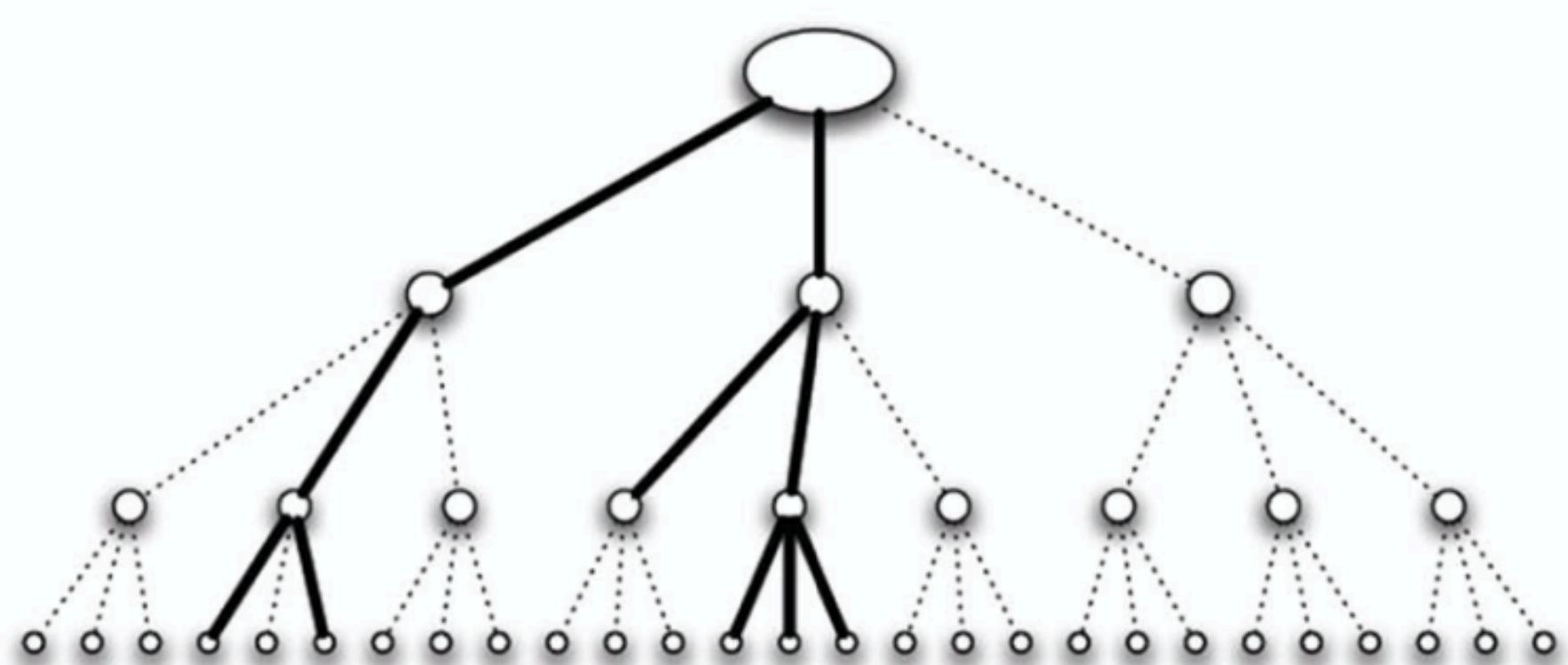
Branching Processes

Randomness and trees

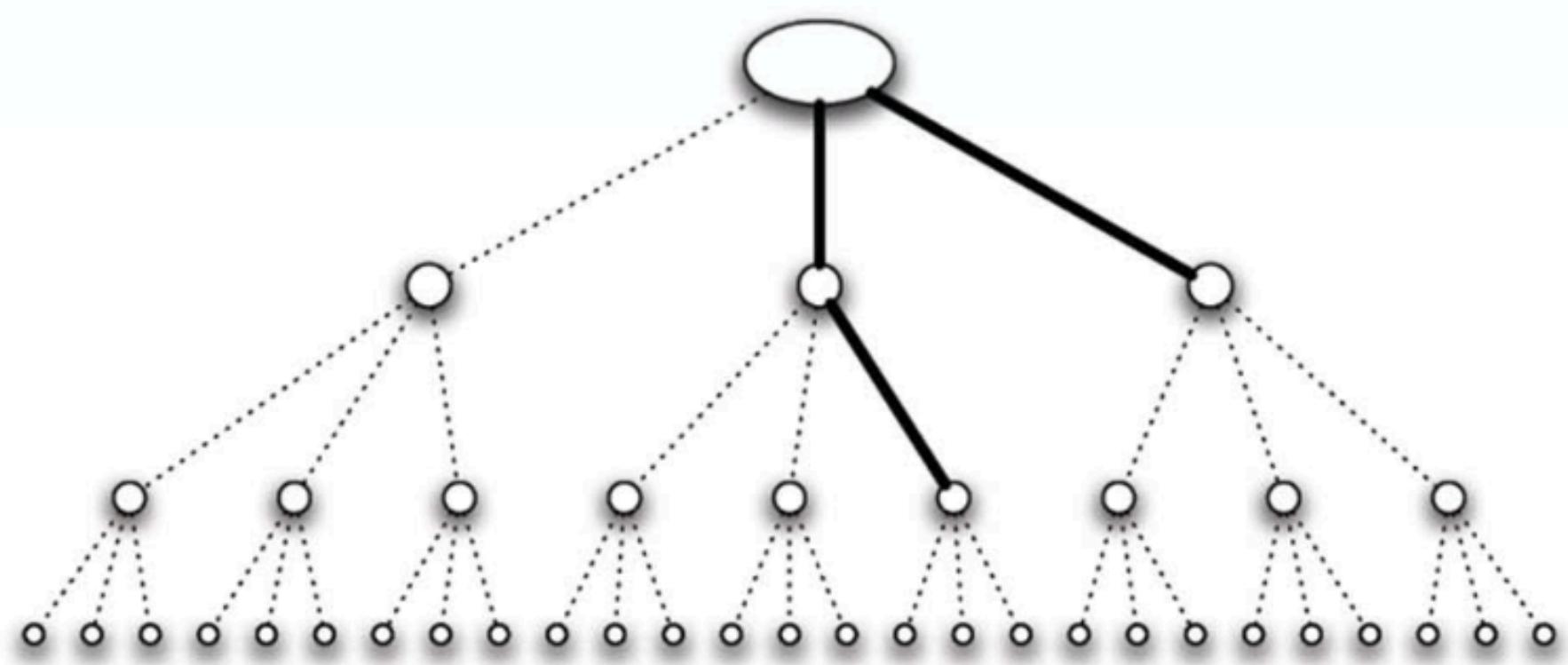
- ❖ **Branching process:** the simplest form of contagion
- ❖ Assumption: the underlying network is a (infinite) tree
 - ❖ Patient 0: the **root**
 - ❖ a node at **level n** (representing a *wave* of the epidemic) is a person that meets other k people
 - ❖ every person passes the disease with **prob. β**
- ❖ Question: *can we predict if and when the diseases vanishes?*



(a) *The contact network for a branching process*



(b) *With high contagion probability, the infection spreads widely*



(c) With low contagion probability, the infection is likely to die out quickly

Figure from: Easley, Kleinberg [ns2]

The basic reproductive number R_0

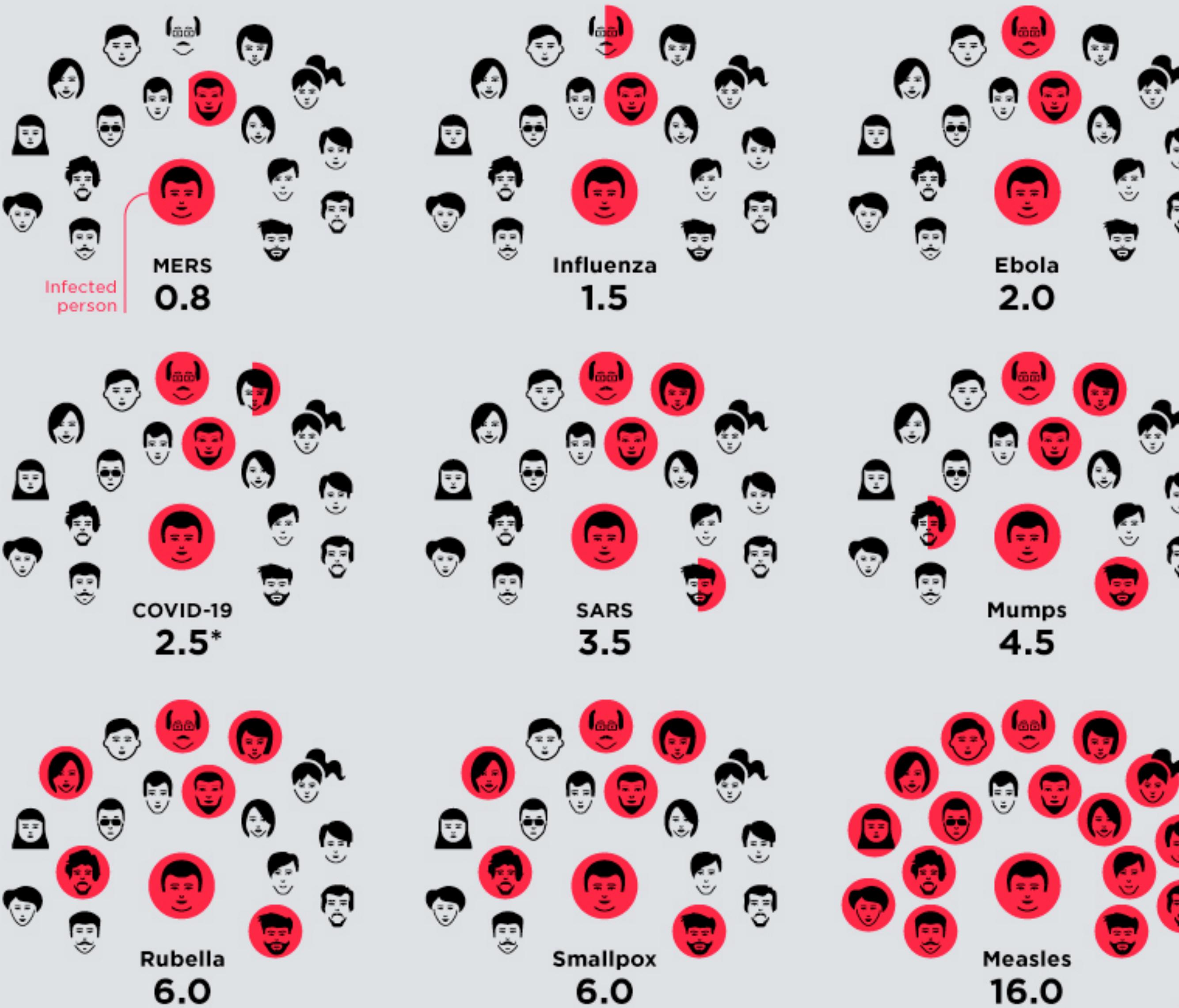
- ❖ Main characteristic in a branching process: if all the individuals in wave n fail to infect any other individuals, the infection vanishes from waves $\geq (n + 1)$
- ❖ R_0 : the **basic reproductive number** of the disease is *the expected number of new cases of the disease caused by a single individual*
 - ❖ in a branching process: $R_0 = \beta k$
 - ❖ We will prove (at the end of this lecture) the following:
Claim: *if $R_0 < 1$, then with probability 1, the disease dies out after a finite number of waves. If $R_0 > 1$, then with probability greater than 0 the disease persists by infecting at least one person in each wave*

Consequences of the dichotomy

- ❖ R_0 can be applied as a *rule of thumb*, but with some precautions:
 - ❖ It can be estimated empirically, and it is a useful *approximate indication* of the spreading power of the disease, when epidemiologists do not have other precise conditions governing the epidemic
 - ❖ Like in our previous lecture, remember that the tree is far from being representative of a real contact network
- ❖ Importantly: when R_0 is close to 1 (slightly below or above), and the contagion probability increases or decreases of a little bit: the epidemics can have the opportunity to become an enormous outbreak or, conversely, to reduce consistently its danger.
- ❖ Remember that $R_0 = \beta k$, so also small changes in k can have a large effect on R_0

RO (basic reproduction number) of diseases

A measure of how many people each sick person will infect on average



Compartmental models

Compartmental model of diseases

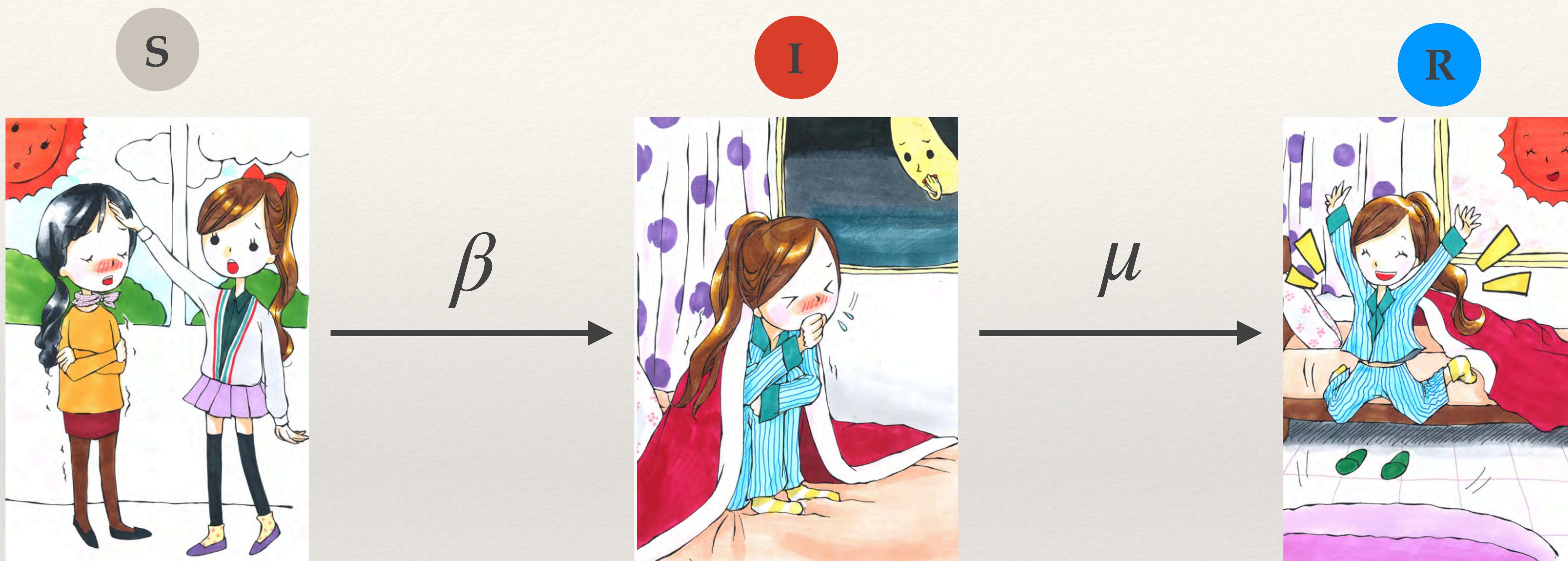
- ❖ In the mathematical representation of an epidemic the host dynamics is reduced to few basic disease *states* or *compartments*
- ❖ Traditional approach doesn't consider contact networks at all.
 - ❖ Assumption: "every individual has an equal chance, per unit time, of coming into contact with every other", i.e. *fully mixed* or *mass-action* approximation

The SIR Epidemic Model

From trees to networks

- ❖ Branching processes are a good starting point when we do not know anything about the network structure
- ❖ However, we need models that can be applied to any network
- ❖ First of all, let's introduce three *states* or *compartments*:
 - ❖ **Susceptible**: the node has not caught the disease yet
 - ❖ **Infectious**: the node has caught the diseases and has some prob of infecting others
 - ❖ **Removed**: after a infectious period, the node has recovered, and it will no longer infect anyone else or being infected again

SIR compartmental model



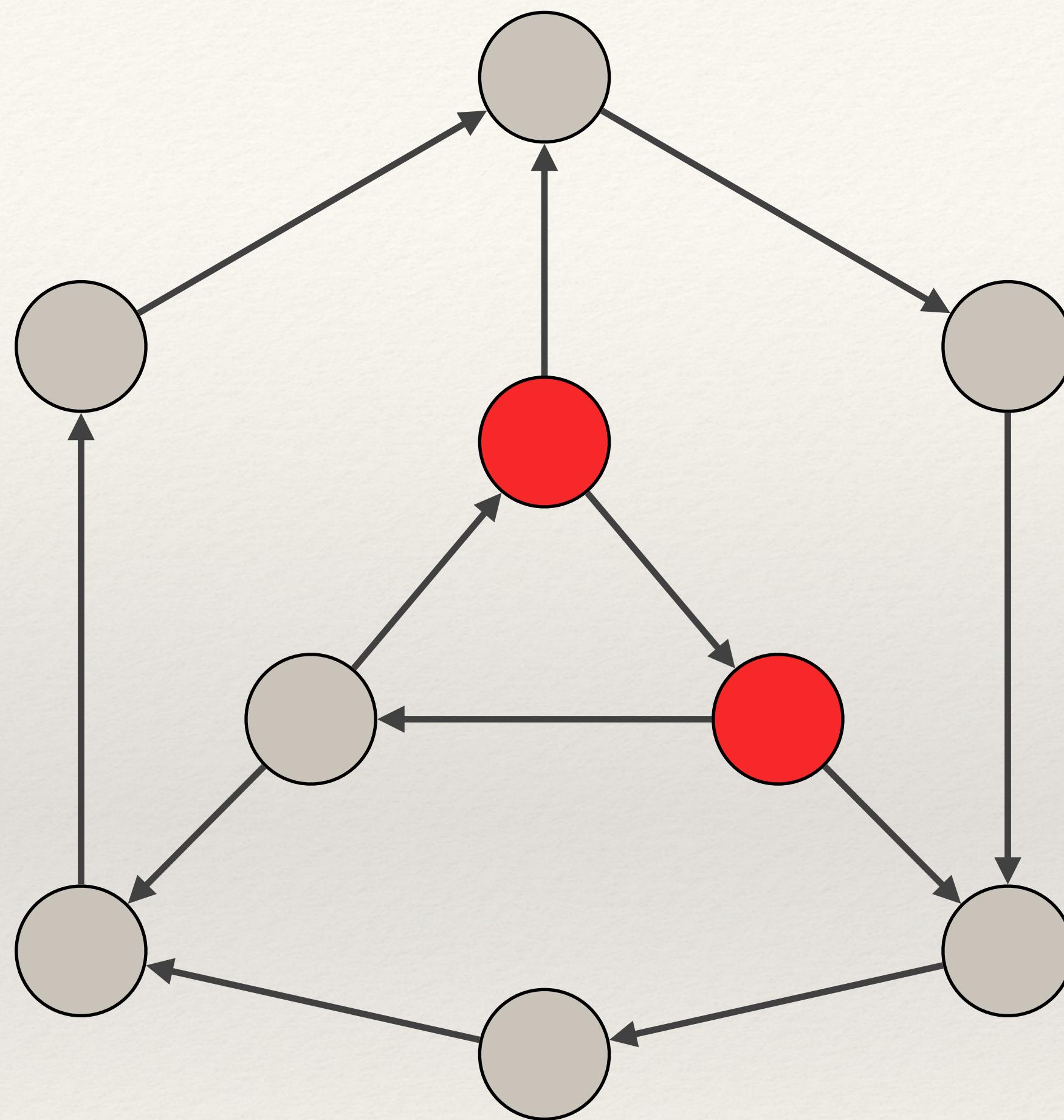
β : prob. of contagion

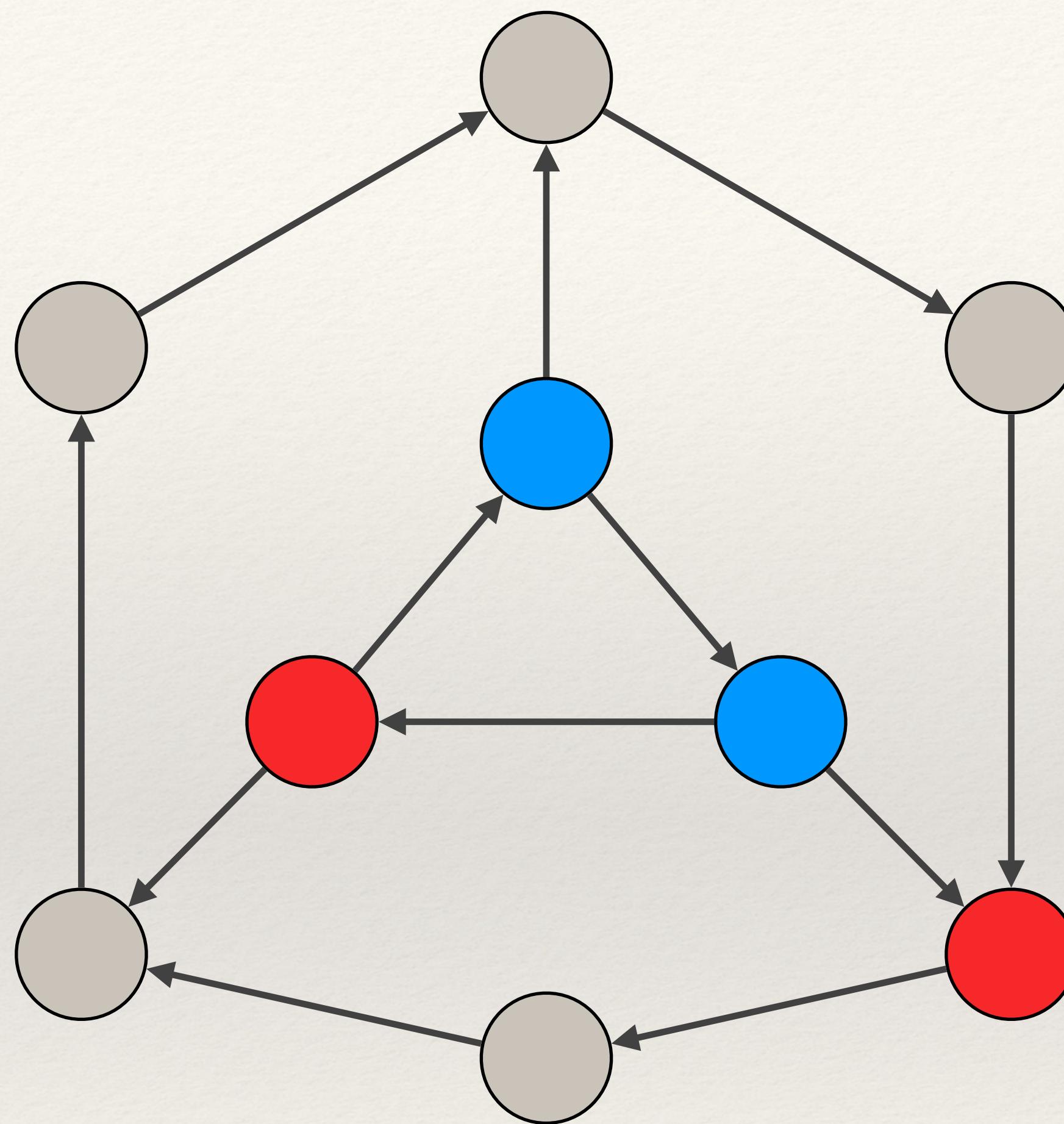
μ : recovery rate

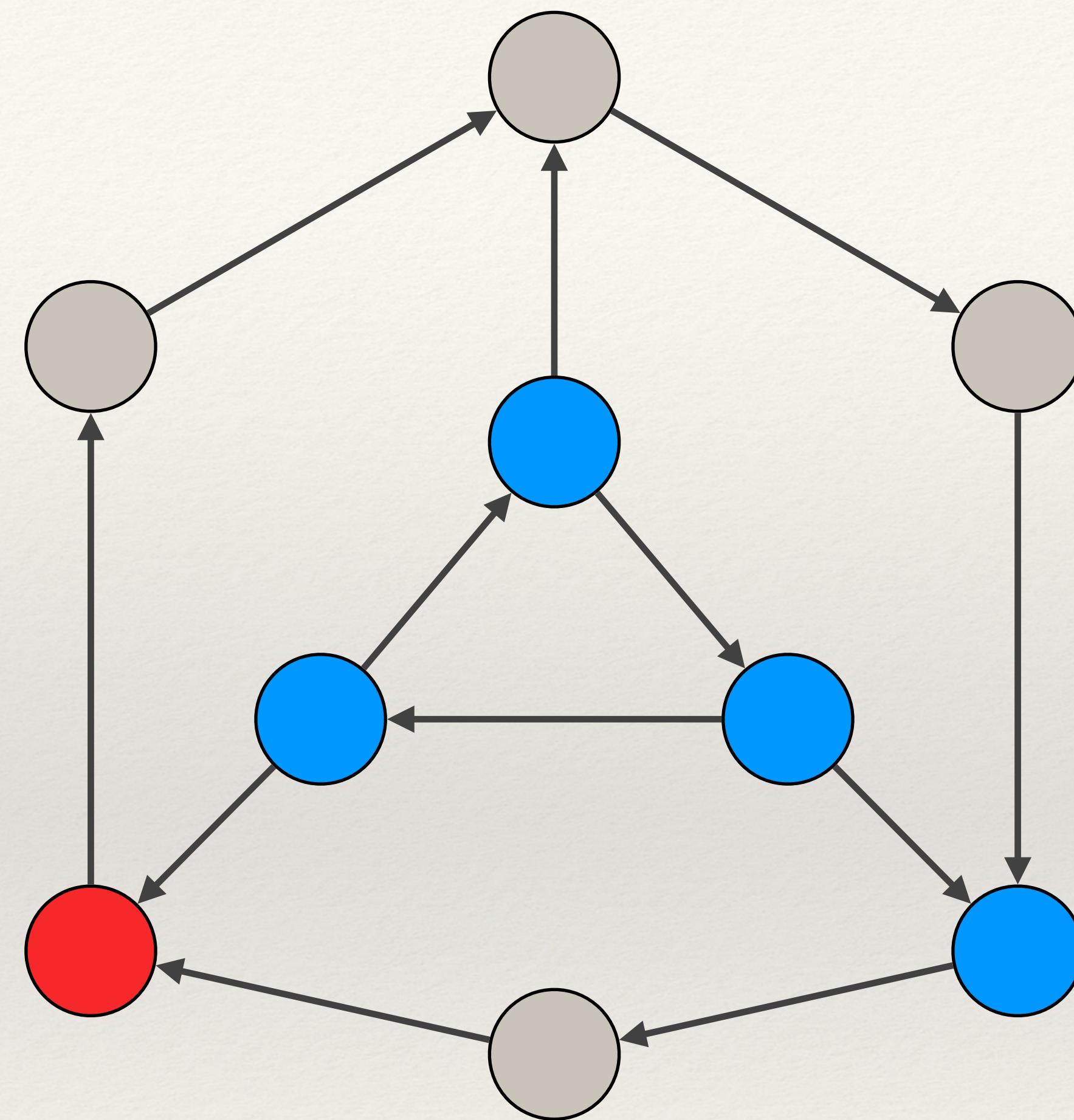
for the sake of simplicity, let's think (temporarily) to μ as a fixed temporal length of the infection

SIR infections in networks

- ❖ Directed graph: contact network
- ❖ Initialization: some nodes are in the I state and all the others on the S state
- ❖ Infection: each node that enters the I state remains **infected** for a fixed number of steps μ , and it passes the disease to each of its **susceptible** neighbors with prob. β
- ❖ Recovering: after μ steps, the infected node is no longer infectious or susceptible, and we describe it as **removed** (R), since it is now an *inert* node in the contact network

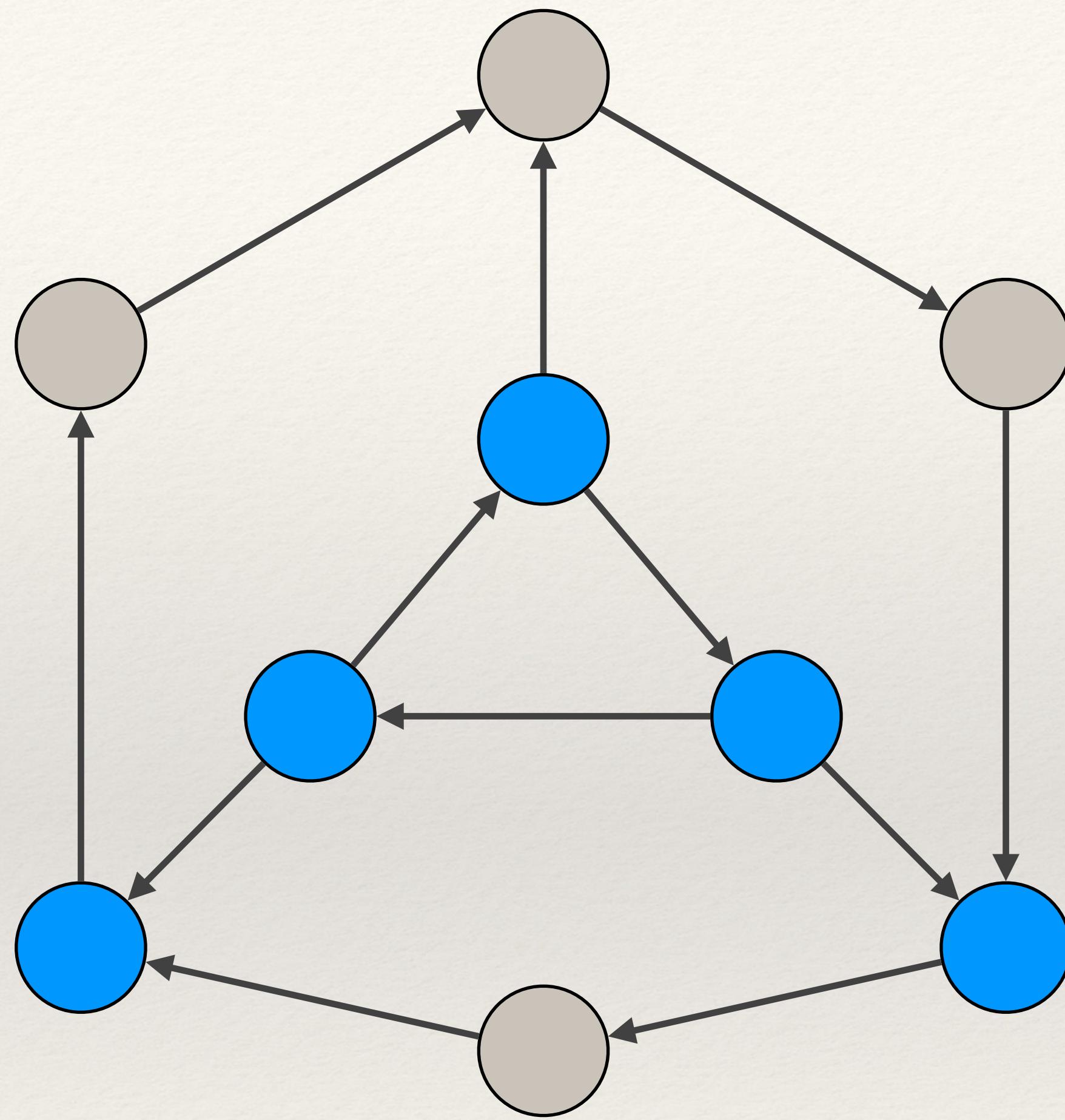
$\mu = 1$ $t = 0$ 

$\mu = 1$ $t = 1$ 

$\mu = 1$ $t = 2$ 

$$\mu = 1$$

$$t = 3$$



Observations

- ❖ The branching process is a **special case** of the SIR model, where:
 - ❖ the contact network is an infinite tree, with each node connected to a fixed number of child nodes
 - ❖ and $\mu = 1$
- ❖ To study the general dynamics:
 - ❖ execute different **agent-based simulations** (each simulation is a realization of the model)
 - ❖ observe what happens to $\frac{S(t)}{N}$, $\frac{I(t)}{N}$ and $\frac{R(t)}{N}$ when $t \rightarrow \infty$
 - ❖ otherwise, solve the problem mathematically (next lecture)

Mode: Interactive Commands and Code: Bottom

model speed

ticks: 26

number-of-nodes 150

average-node-degree 6

initial-outbreak-size 3

setup

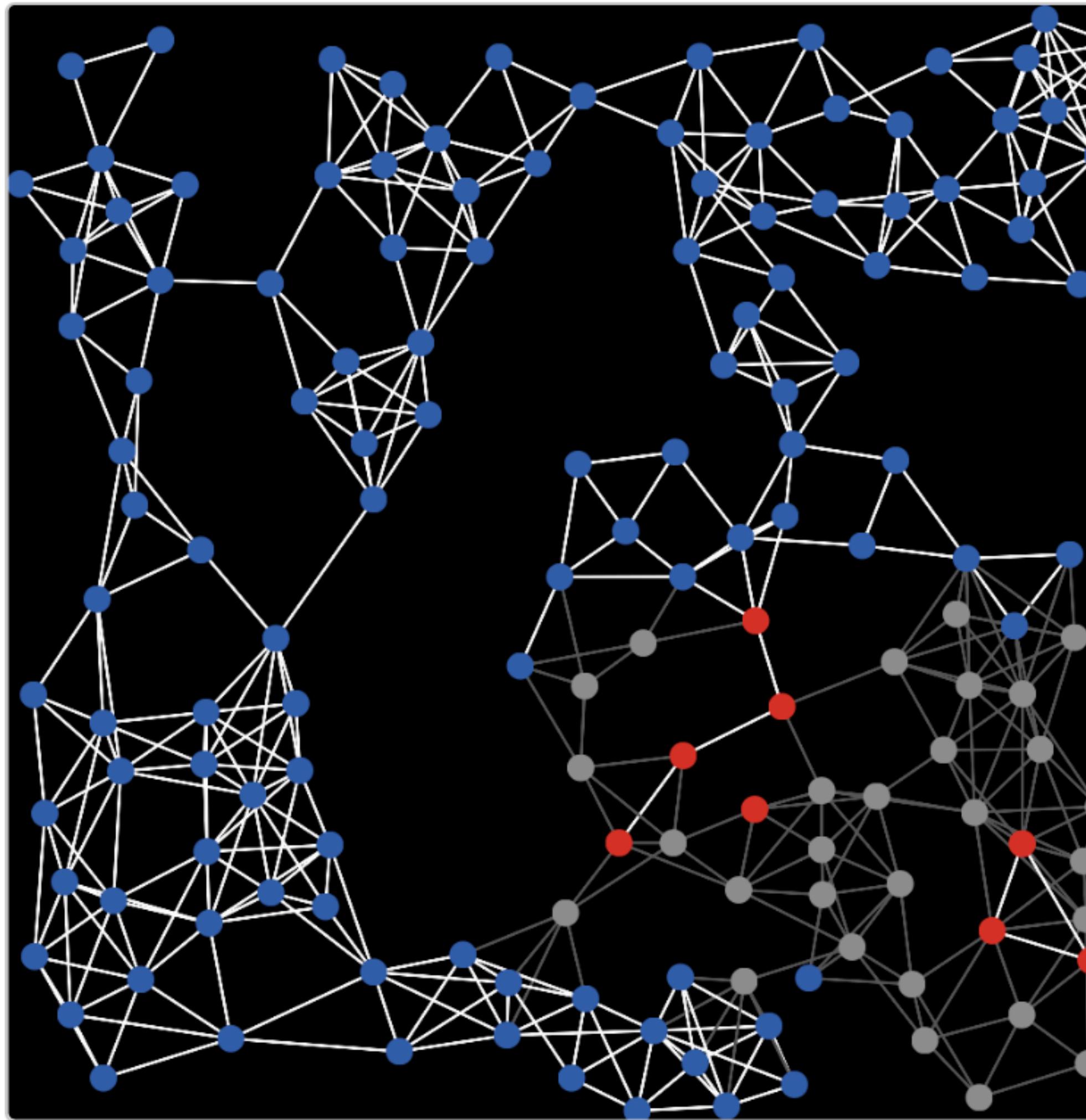
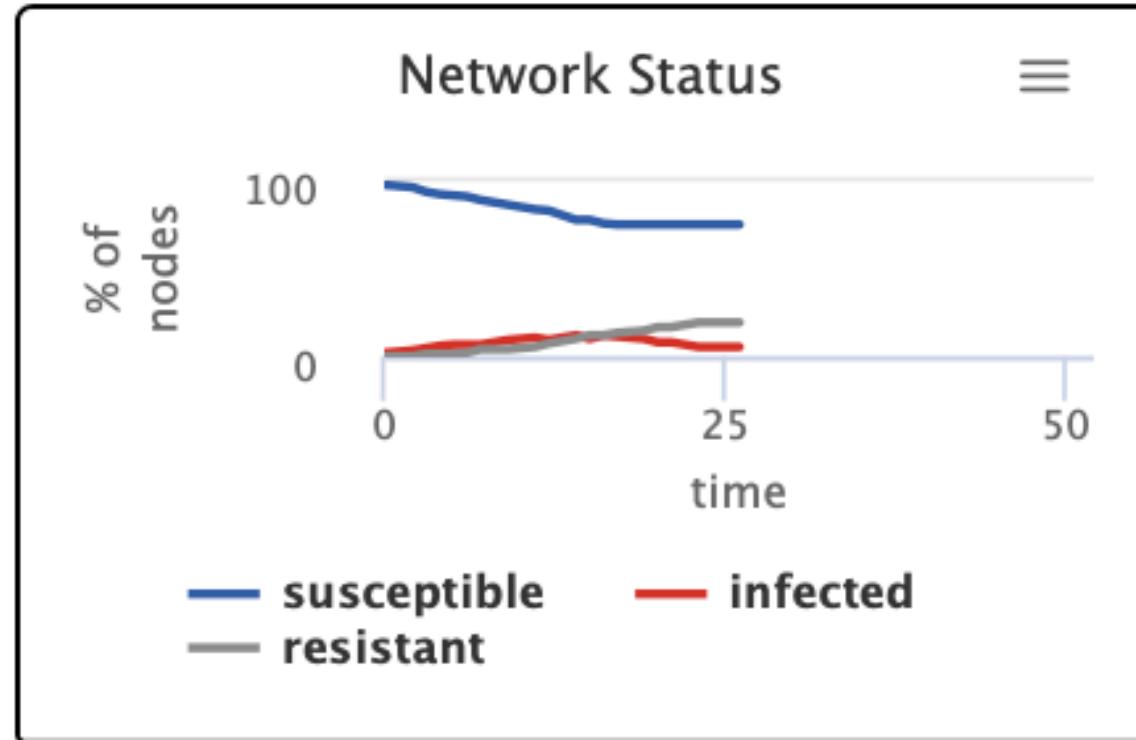
go

virus-spread-chance 7.4 %

virus-check-frequency 1 ticks

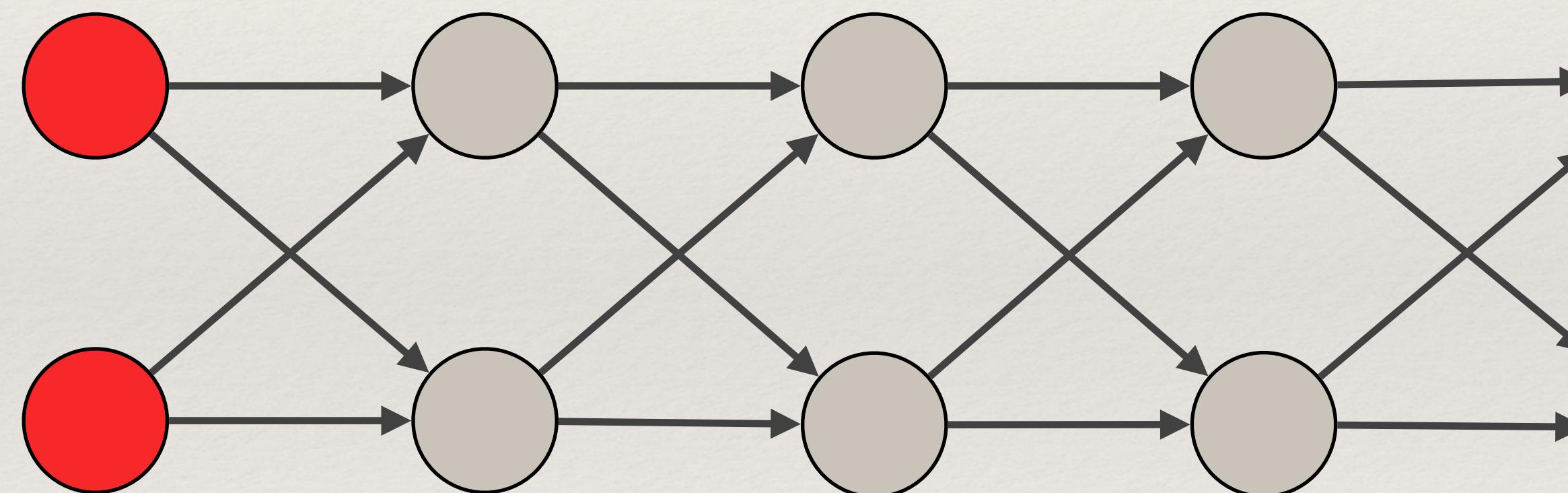
recovery-chance 9.8 %

gain-resistance-chance 100 %



The role of R_0

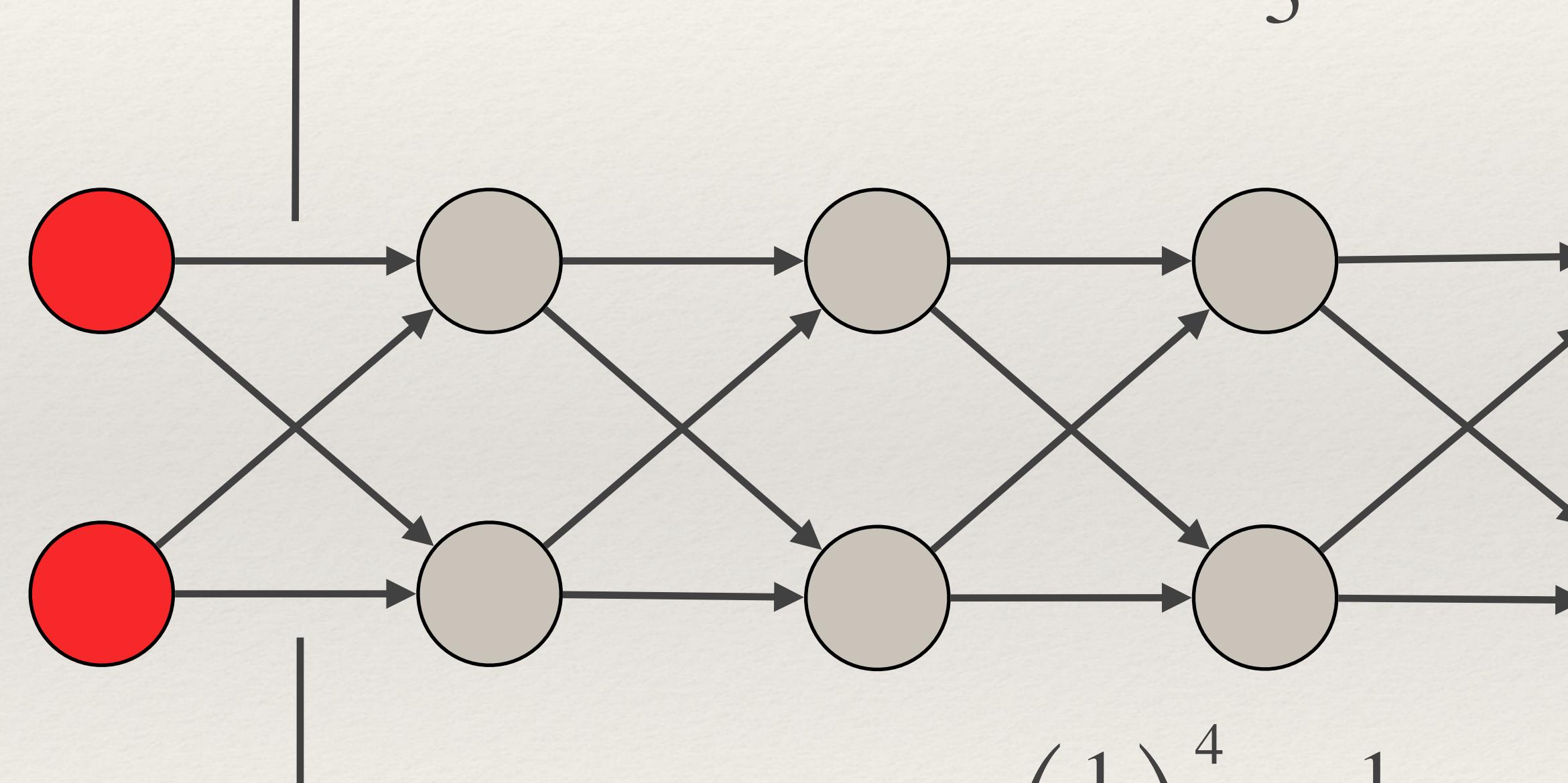
- ❖ With an arbitrary network structure, the dichotomy in epidemic behavior determined by R_0 does not necessarily hold (\rightarrow next lecture)
 - ❖ example: $\beta = 2/3, \mu = 1$



- ❖ $R_0 = 2 \cdot \frac{2}{3} = \frac{4}{3} > 1$; nevertheless, the disease will vanish from the above network.

The role of R_0

each edge fails to transmit with prob. $(1 - \beta) = \frac{1}{3}$



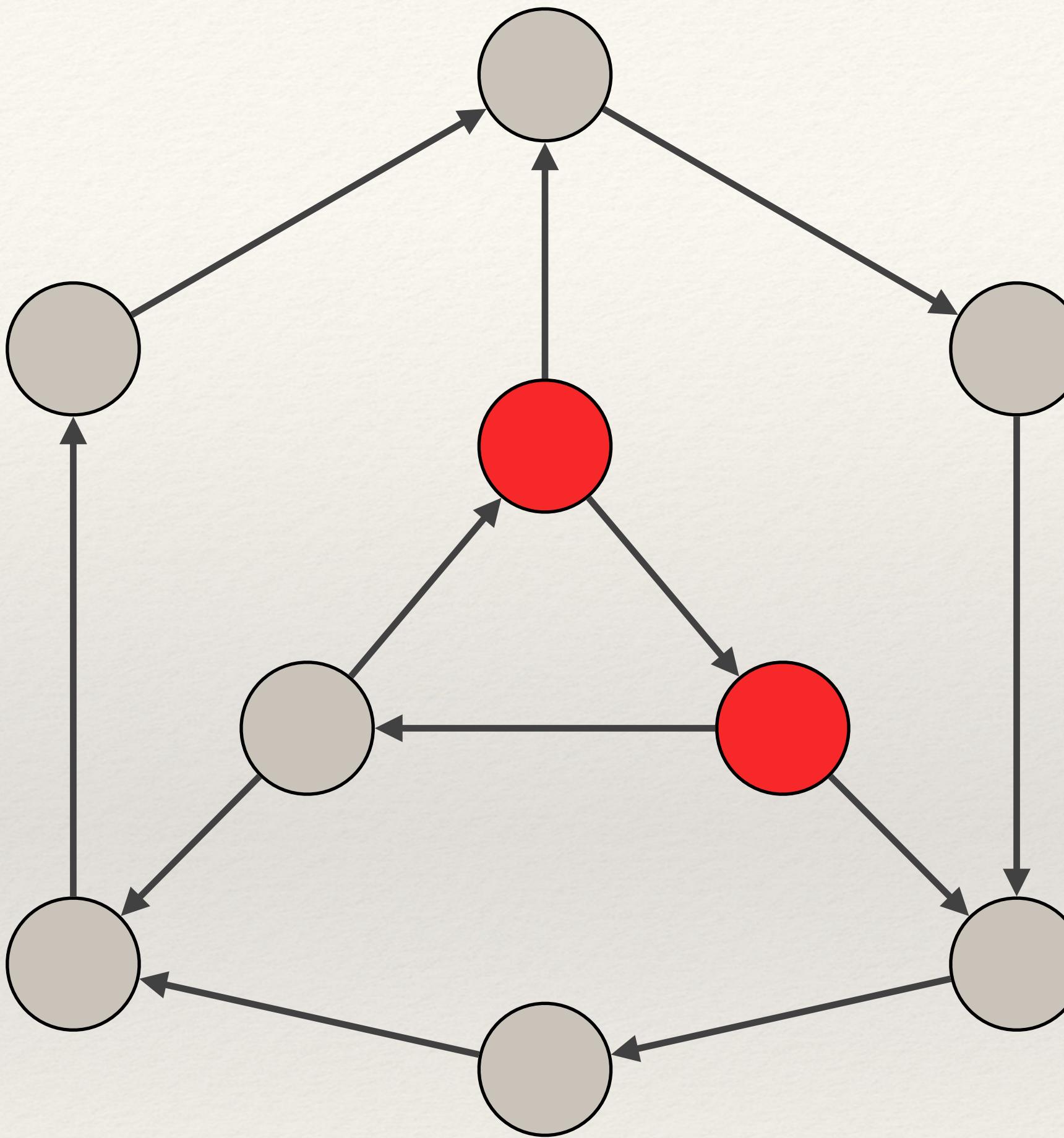
All four edges fail to transmit with prob. $\left(\frac{1}{3}\right)^4 = \frac{1}{81}$

Sooner or later this will happen in a number of finite steps: with prob. 1 the disease will die out!

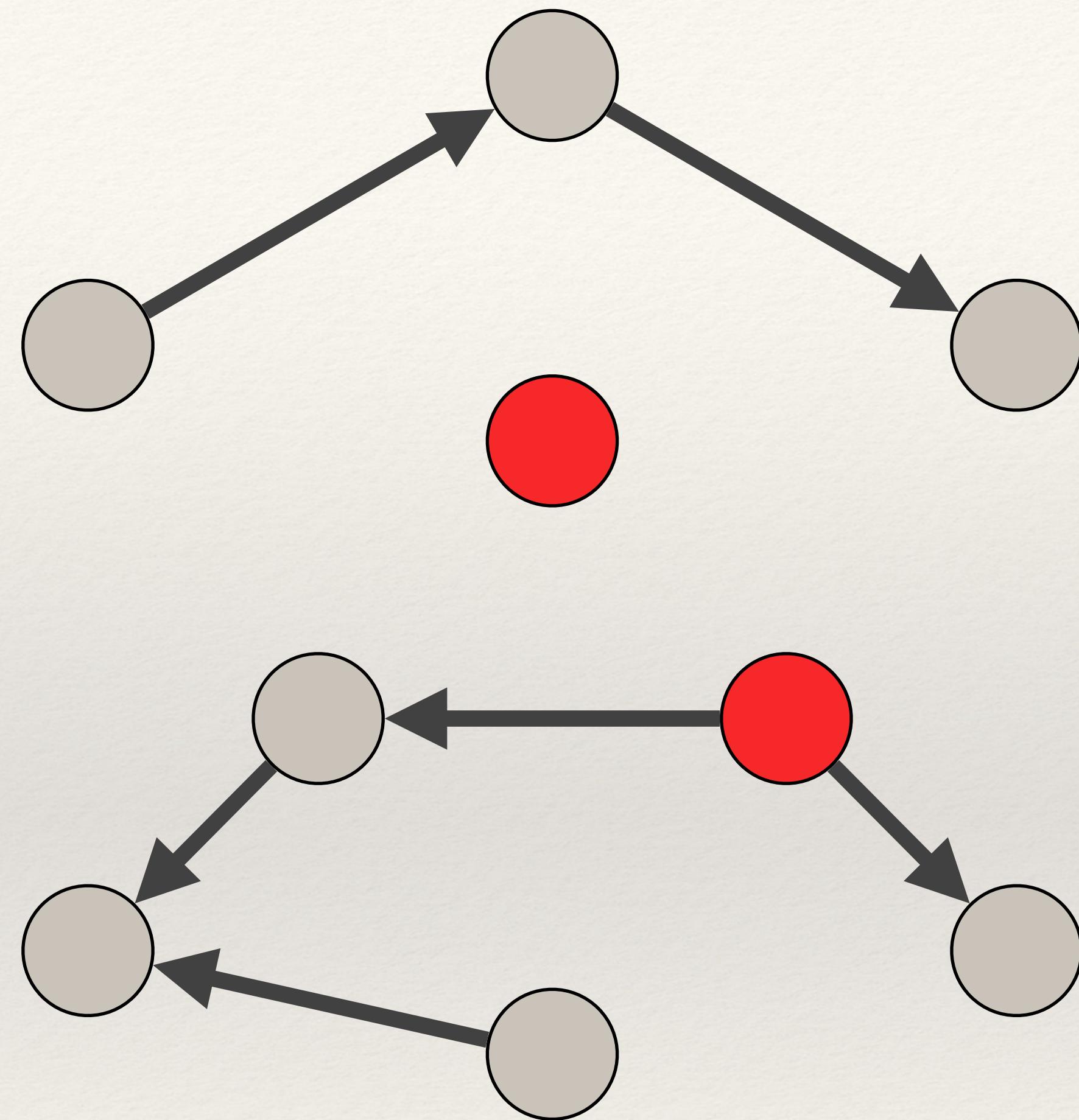
SIR epidemics and percolation

- ❖ A **static view of the process**, again focusing on the basic SIR model in which $\mu = 1$:
 - ❖ Instead of deciding with probability β at time t if the edge will transmit the disease, we can run our probabilities at the beginning; this will *open* or *block* the edges
 - ❖ then, we can just check if there are paths from the infected nodes to other nodes in the network to understand which nodes will be infected: it is like observing **fluid percolation** in a system of pipes

$$\mu = 1$$



$$\mu = 1$$

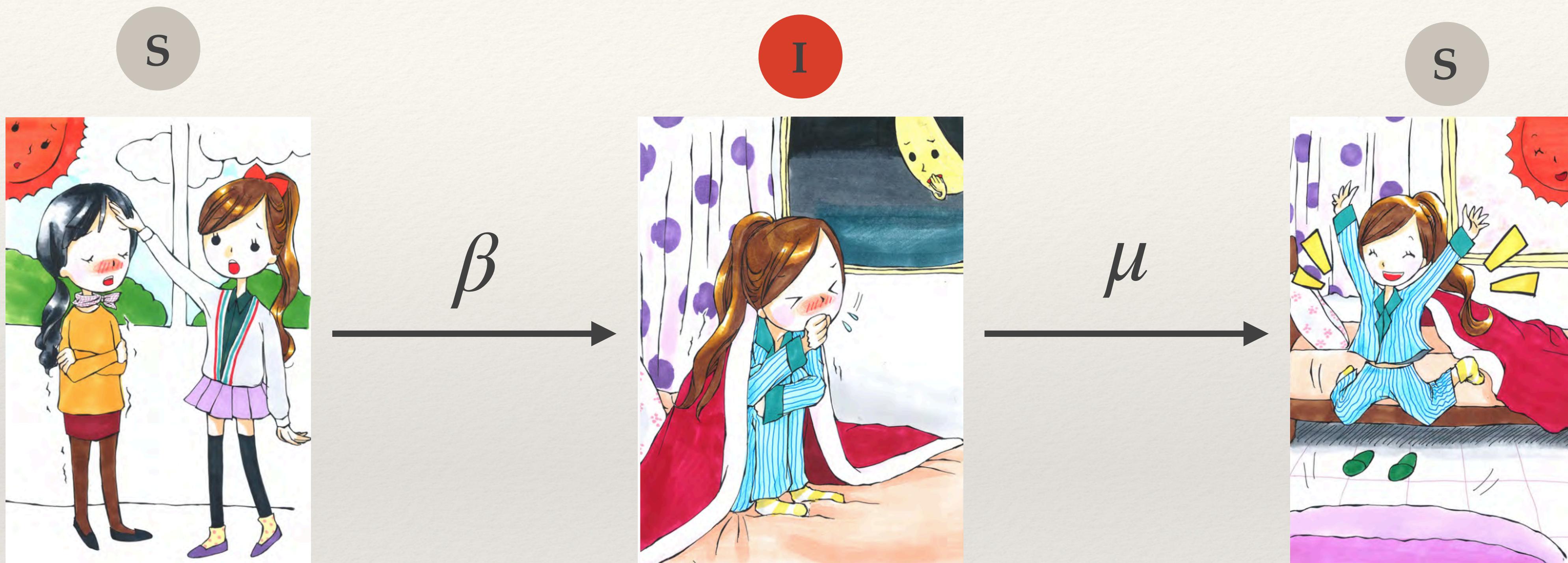


The SIS Epidemic Model

From Infected to Susceptible

- ❖ If we want to focus on epidemics in which individual can be reinfected multiple times, we can use a SIS model
- ❖ Now we have only **Susceptible** and **Infectious** states
- ❖ Initialization: some nodes are in the I state and all the others on the S state
- ❖ Infection: each node that enters the I state remains **infected** for a fixed number of steps μ , and it passes the disease to each of its **susceptible** neighbors with prob. β
- ❖ Recovering: after μ steps, the infected node is no longer infectious returns to the **S** state

SIS compartmental model



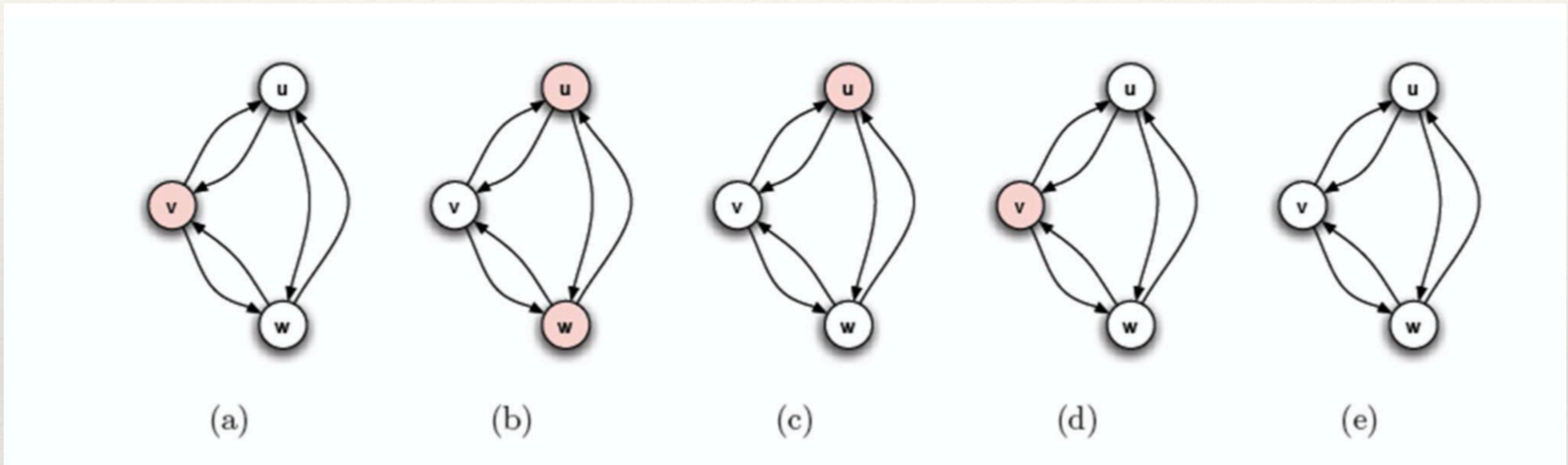
β : prob. of contagion

μ : recovery rate

for the sake of simplicity, let's think (temporarily) to μ as a fixed temporal length of the infection

An example

$$\mu = 1$$



Connections between SIR and SIS

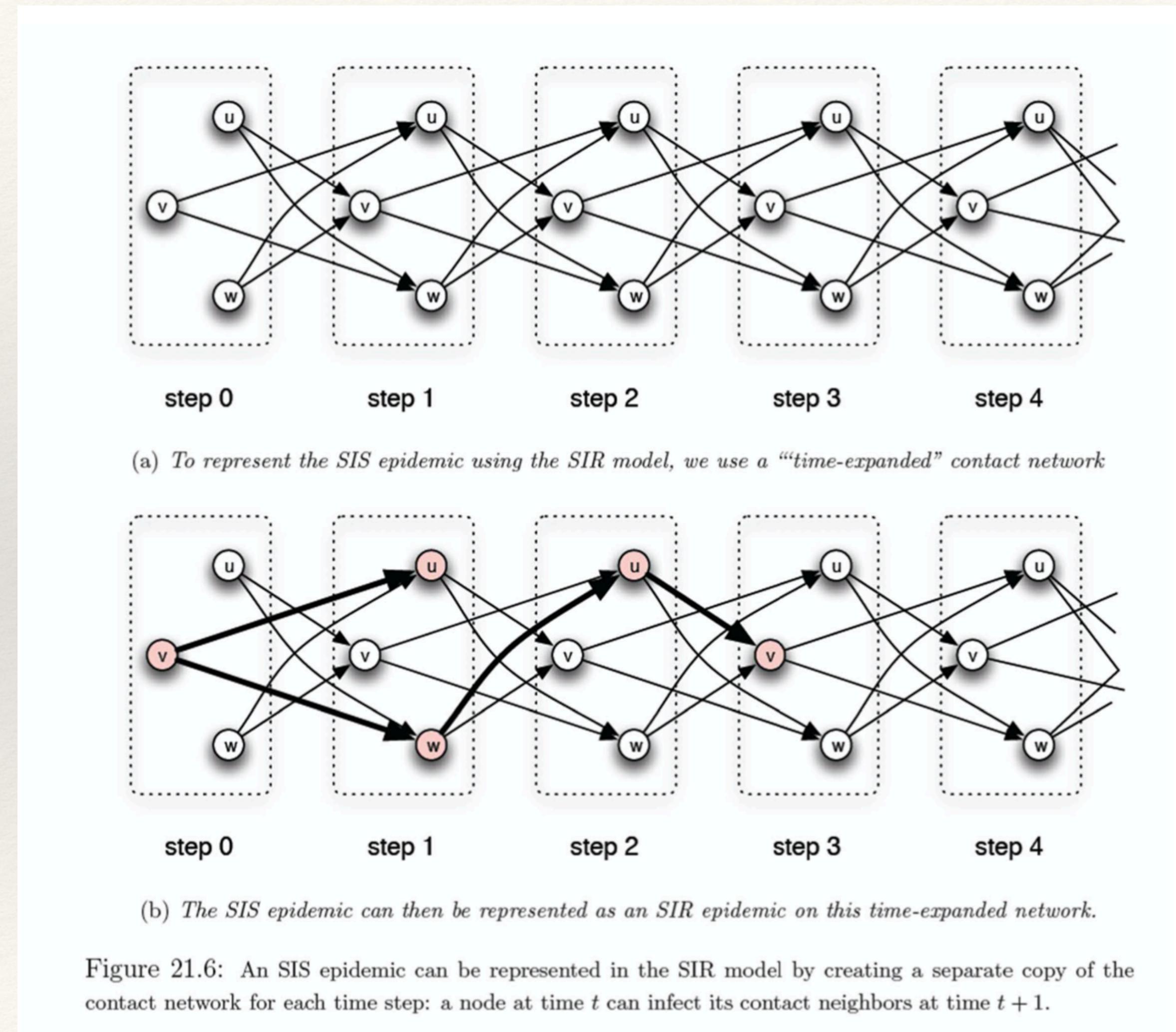


Figure from: Easley, Kleinberg [ns2]

Synchronization

Synchronization and oscillations

- ❖ Global dynamics of a disease: the tendency of epidemics for certain diseases to **synchronize** across a population, sometimes producing strong **oscillations** in the number of affected individuals over time
- ❖ If you look at public-health data, it is natural to look at periodic oscillations
- ❖ Why this happens?
 - ❖ large-scale societal changes, or also
 - ❖ contagion dynamics of the disease itself
- ❖ How can we model this?
 - ❖ let's embed in our models a combination of **temporary immunity** and **long-range links** in the contact network

The SIRS epidemic model

- ❖ We combine elements of the SIR and SIS models to confer temporary immunity on infected individuals
- ❖ Initialization: some nodes are in the I state and all the others on the S state
- ❖ Infection: each node that enters the I state remains **infected** for a fixed number of steps μ_I , and it passes the disease to each of its **susceptible** neighbors with prob. β
- ❖ New feature: after μ_I steps, the infected node is no longer infectious. It enters the **R** state for a fixed number of steps μ_R , then it returns to the **S** state.

Small-world contact networks

- ❖ Temporary immunity can produce oscillations in very localized parts of the network, with patches of immunity following large numbers of infections in a concentrated area
- ❖ If we have many long-range connections (recall previous lecture), we introduce *coordination* with many different places
- ❖ We can build Small-World networks following the Watts-Strogatz model: nodes arranged in a ring, with many homophilous links, and some 'shortcuts';
- ❖ We add a probability c that controls the fraction of long-range links in the network

SIRS epidemics with synchronization

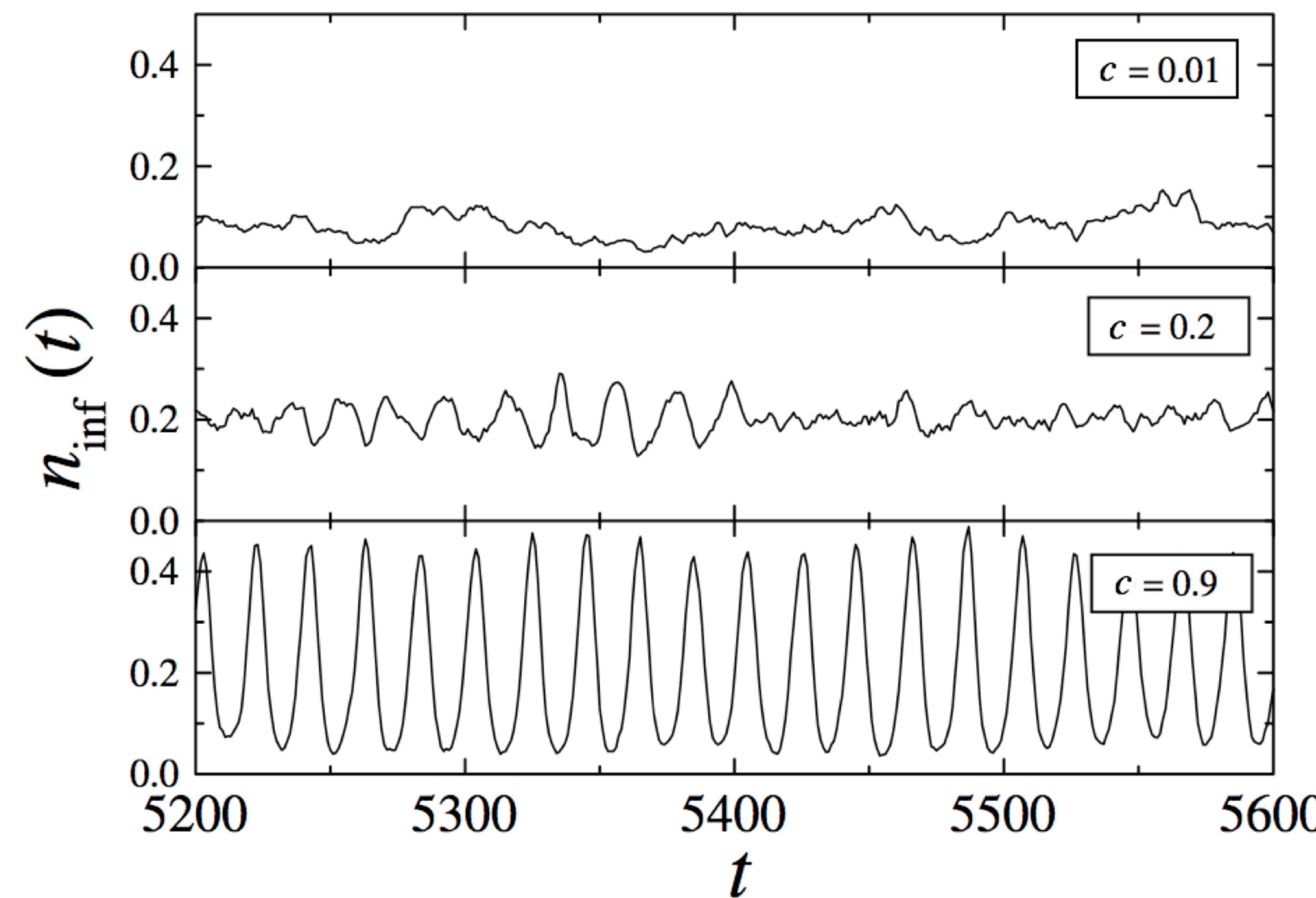


Figure from: M. Kuperman and G. Abramson. Small world effect in an epidemiological model. PRL, 86(13):2909–2912, March 2001.

Note on R₀ ad R_t

The **basic reproductive number (R₀)**, or the more ‘real-life’ **effective reproductive number (R_t)** for a given population are different:

R₀ is the number of secondary cases generated by the presence of one infected individual in an otherwise fully susceptible, well-mixed population.

R_t is a more practical real-life version of this, which uses real-life data (from diagnostic testing and /or clinical surveillance) to estimate the reproductive number for an ongoing epidemic.