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# Epidemic Spreading

*Network Science '21: Session 10.2*

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## Lecture objectives

- Learn the simplest (and more used!) modelling framework to study the spreading of epidemics and other social processes
- Get more familiar to key concepts in ABM: Control Parameters/Order Parameters



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## Short recap



## Components of a model: Agents

### State variables

Each agent is characterised by a set of variables which identify their state

- + ***Binary***
  - Whether it adopted a product or not
  - In favour or against a legislation
- + ***Categorical***
  - Status with regard to an illness
- + ***Real***
  - Knowledge level
  - Investment



## Subject of study: (ii) The role of a mechanism

### Dynamics

Agents change their state(s) because of the interaction with others or information from the environment. These are **microscopic rules**, or mechanisms under study

- + *Imitation*
- + *Herding*
- + *Diffusion*
- + *Homophily*



## Components of a model: Interactions

### Network

Agents do not act in isolation, but are affected by (or affect) the behaviour of peers

- + *All-to-all*
- + *Local neighbours*
- + *Social network*



## Control Parameters

### Definition of Control Parameters

The rules that specify the mechanism, the agents, etc. are endowed with parameters that can be adjusted to understand their effect on emergent properties



# Order Parameters

## Definition of Order Parameters

During the simulation, we will measure large scale properties of the system. They quantify the emergent state of the system



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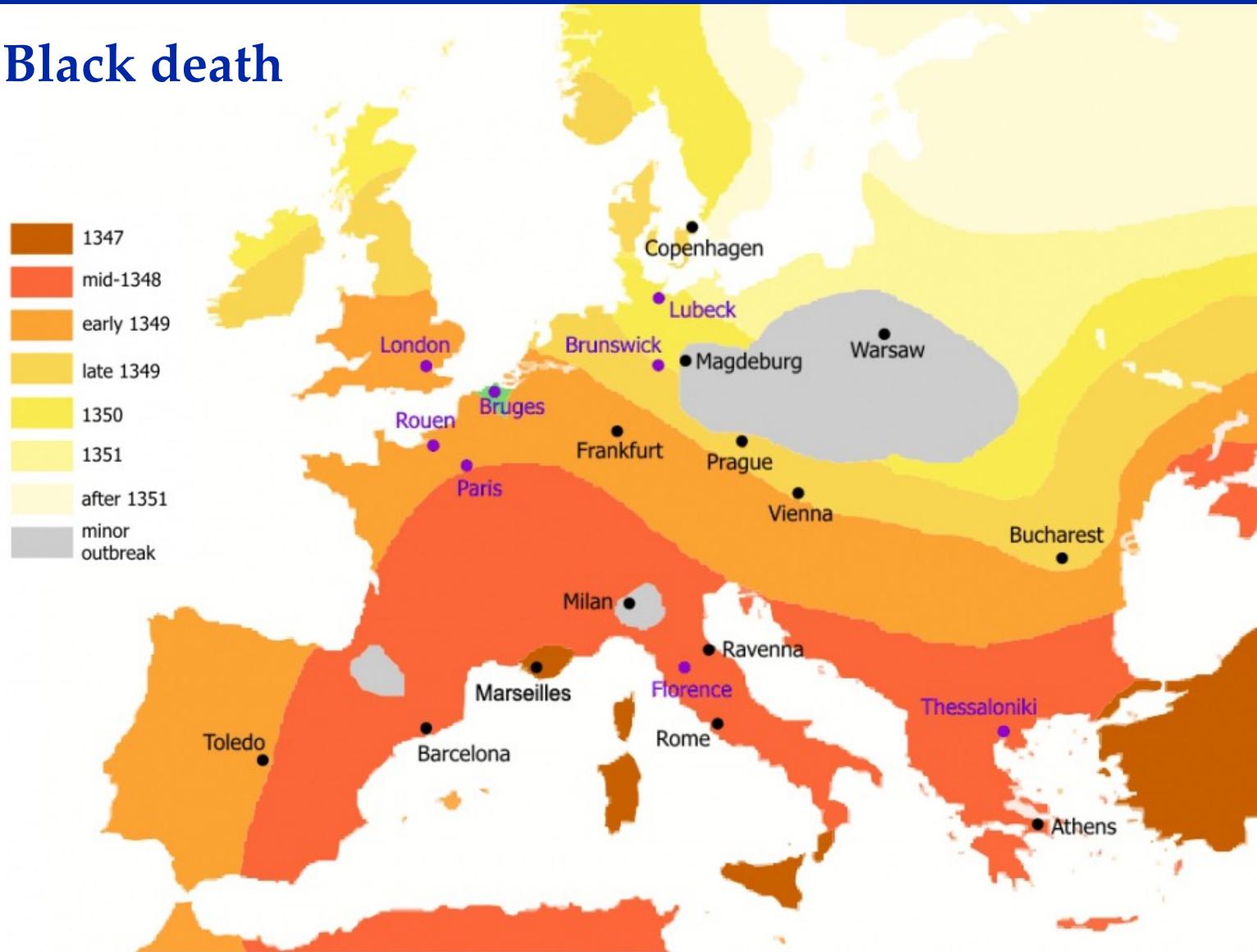
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# Introduction to compartmental models



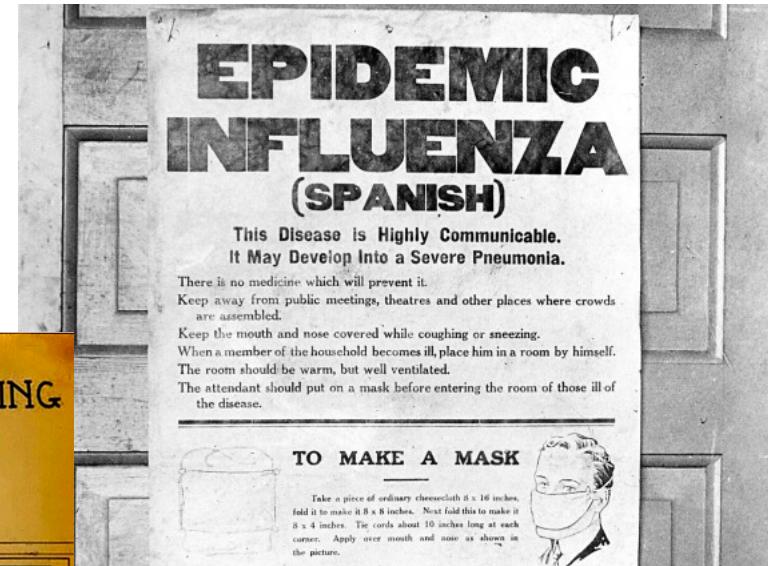
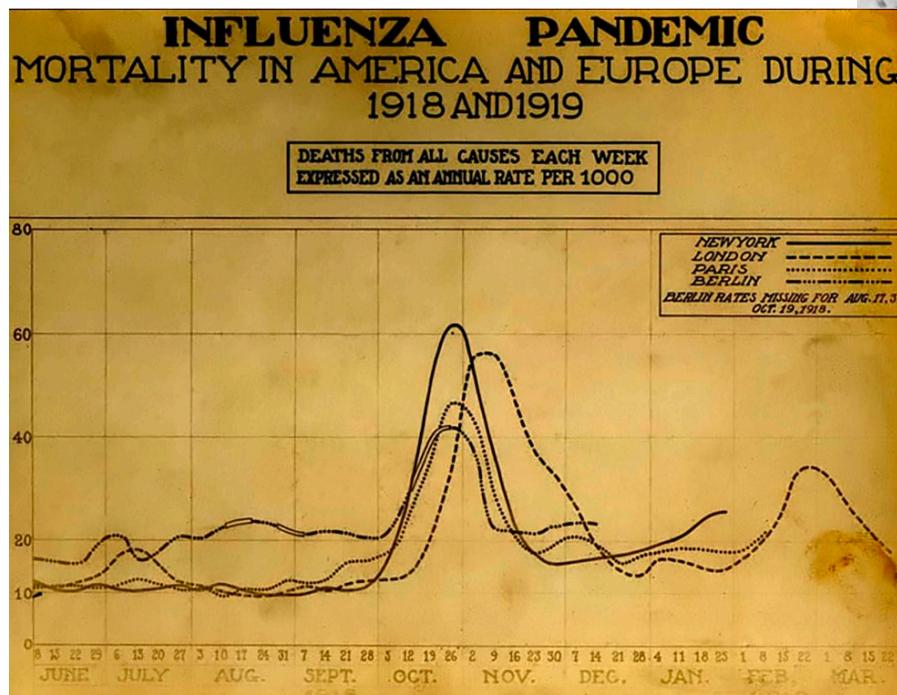
*The Tirumph of Death*, Pieter Bruegel de Oude [from Wikipedia]

## Black death





## Spanish Flu (H1N1)



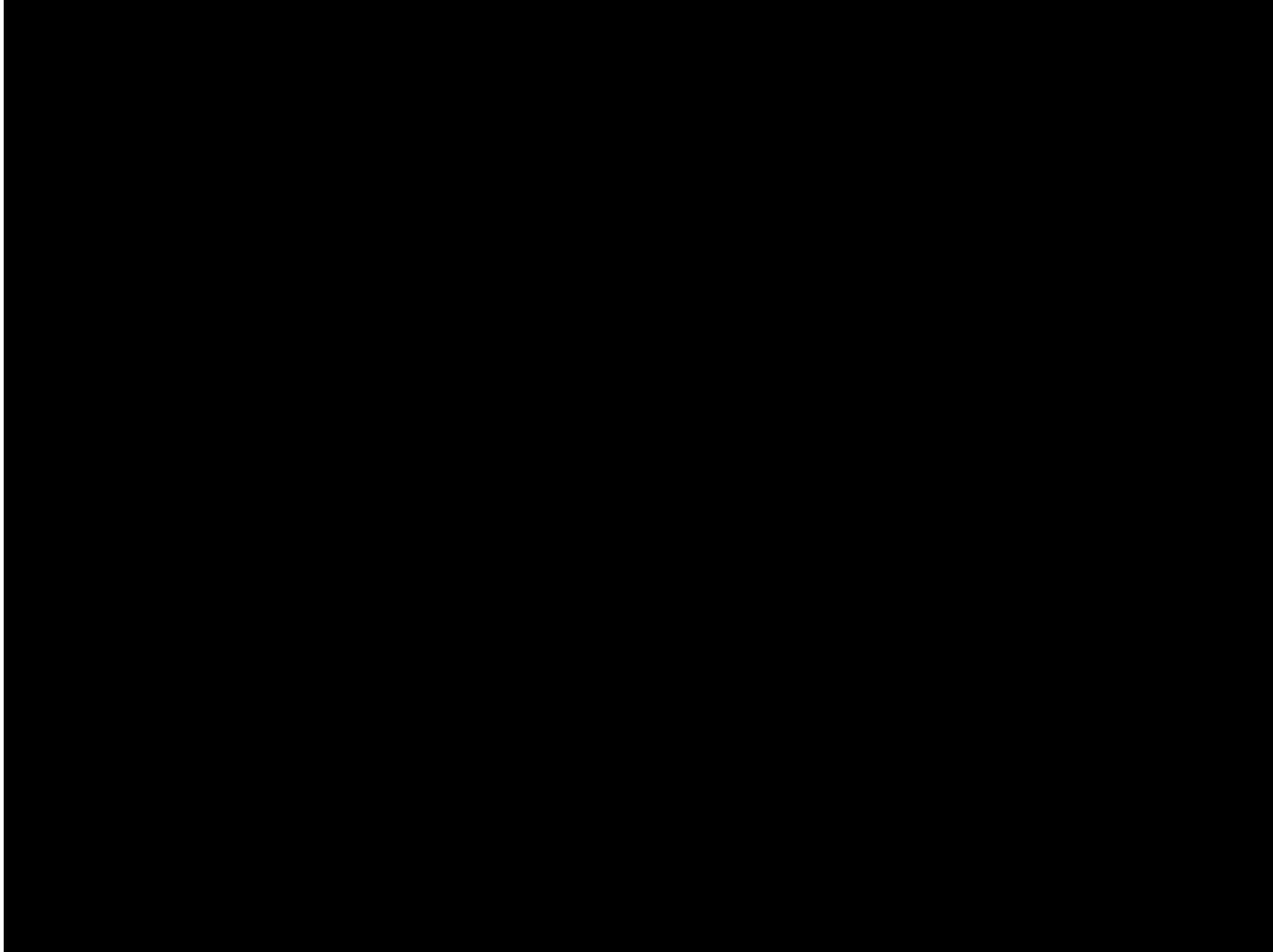


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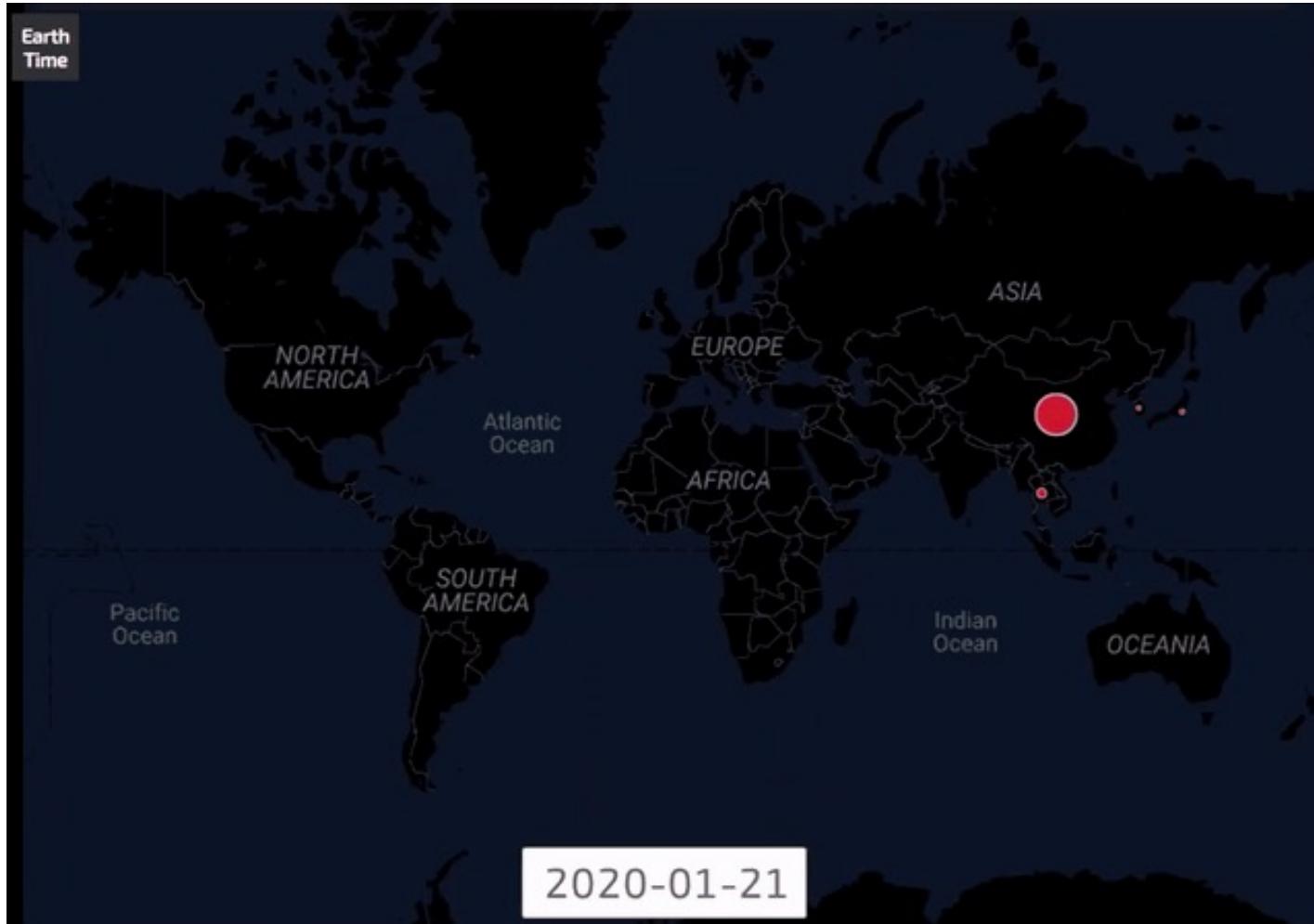
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## H1N1



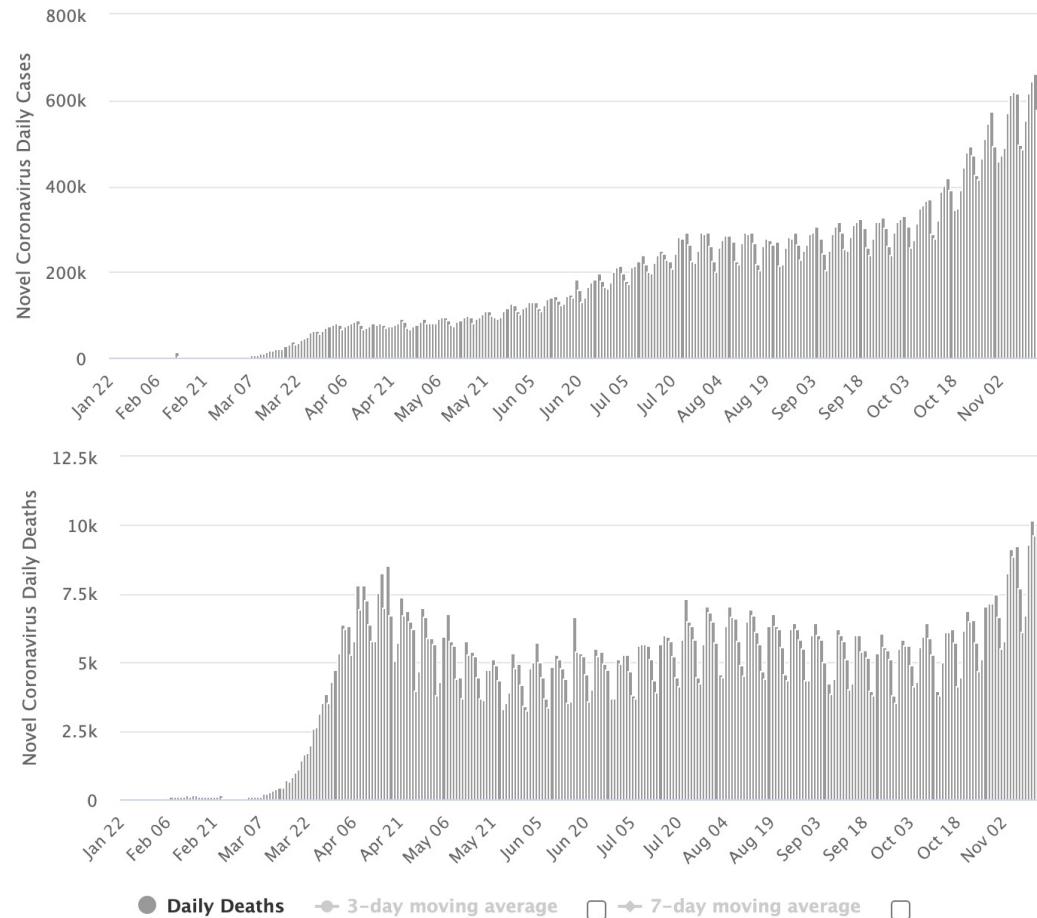


## Covid-19





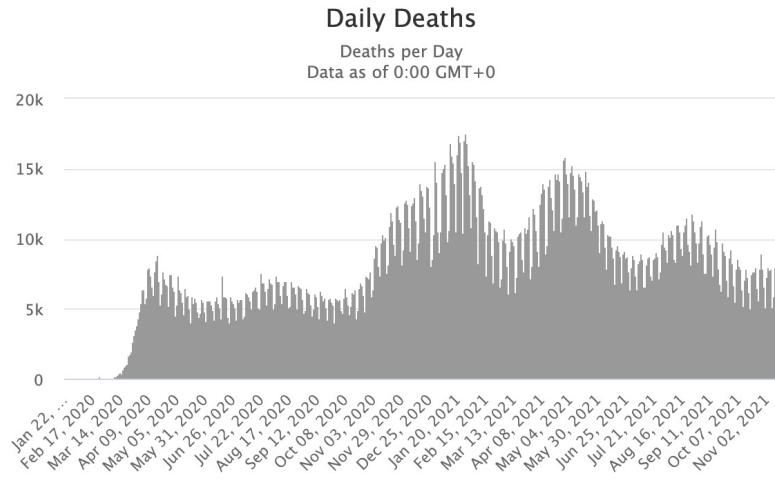
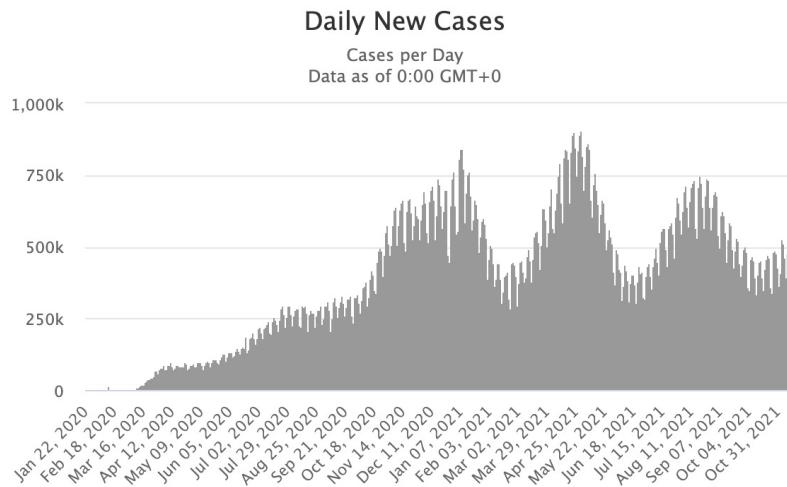
## Covid-19



Source: Worldometer - [www.worldometers.info](http://www.worldometers.info)

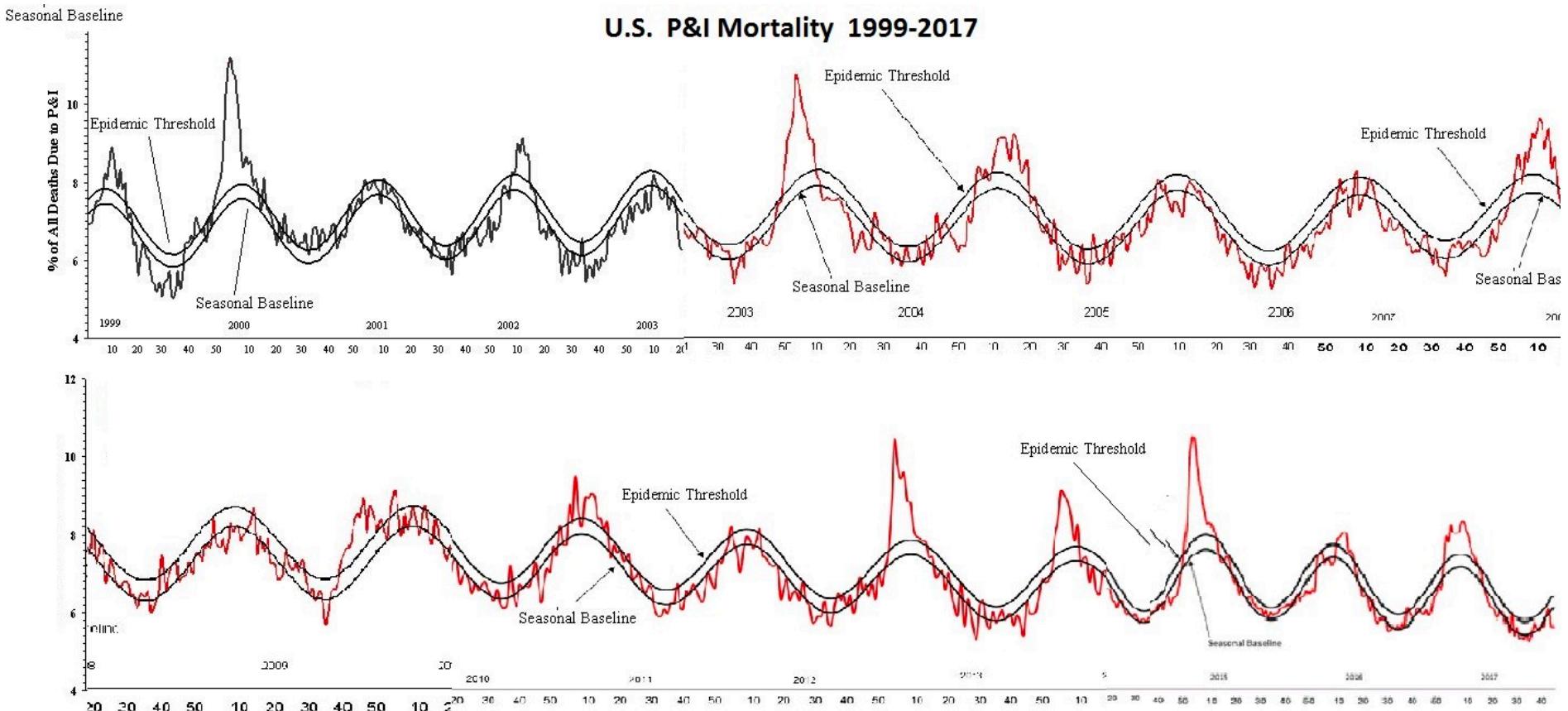


## Covid-19





## Seasonal flu





*Studying dynamical processes is useful to understand the effects of mechanisms on the spread of states: disease / information diffusion / product adoption.*

*The idea is to forecast, control (or foster) specific properties*



## Examples of spreading dynamics

One of the most important examples is epidemic spreading, i.e. how diseases spread over networks representing contacts between individuals.

- Airborne diseases like covid, influenza or tuberculosis are transmitted when two people breathe the air in the same room.
- Other contagious diseases (e.g. HIV) and parasites are transmitted through human contact.

These are not the only kind spreading phenomena following the same kind of dynamics

- Rumour spreading
- Computer viruses

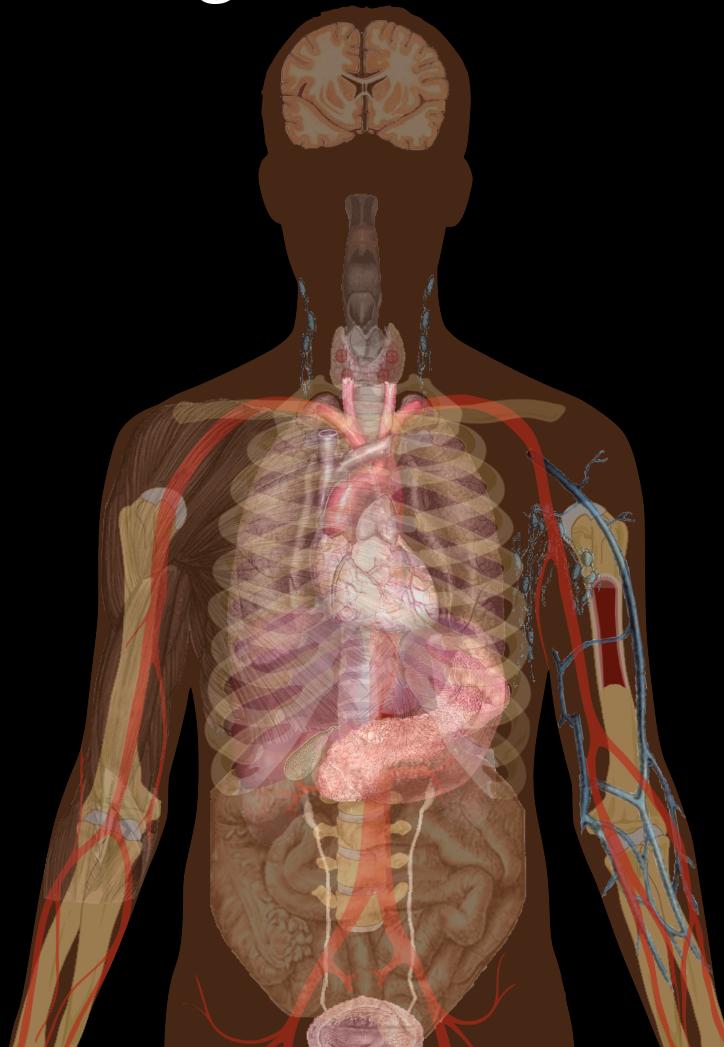


*How to model the an epidemic outbreak?*

*There are too many aspects involved*



# The agents



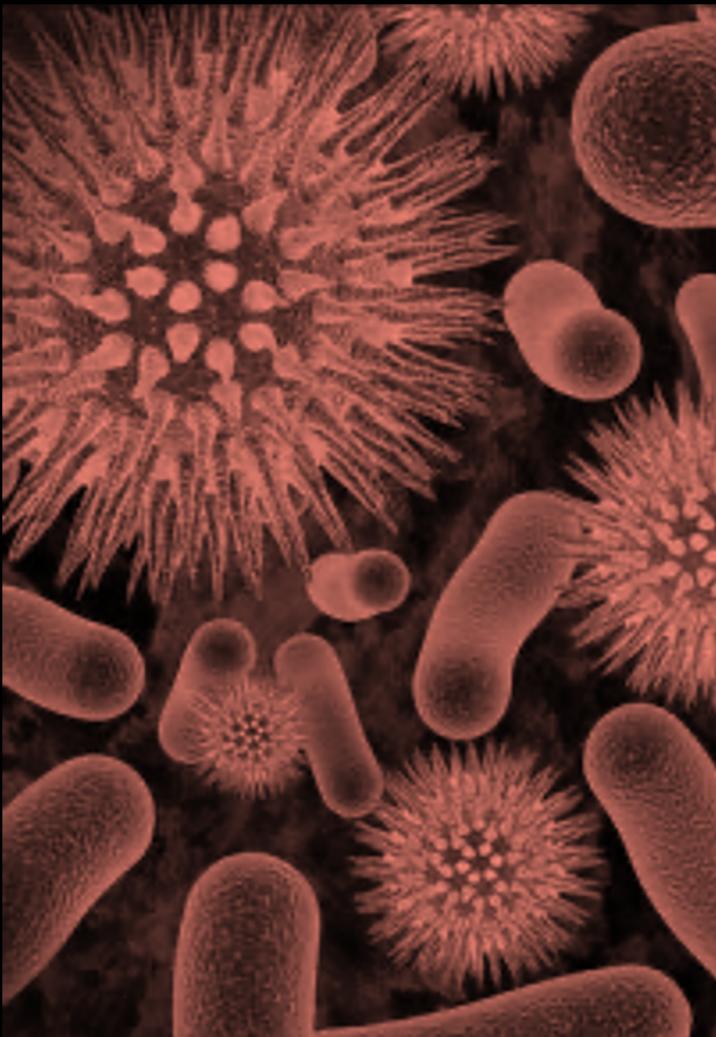
- Should the dynamics of clinical measurements be included?
  - Body Temperature
  - Oxygen in blood
  - Red blood cells count
  - White blood cells count

Should we include the organs?

How general would be the  
modelling approach?



# The disease



- Which microscopic description should we make of the disease?
- To which level would it interact with the agent?
- Viruses and bacteria work differently, how to cope with them?

How general would be the modelling approach?



# The transmission

- To which level should the mechanisms of transmission be implemented
- At which level of detail should we include the dynamics by which one person gets infected? Should we count how many pathogens has this person in his/her body?

**How general would be the modelling approach?**

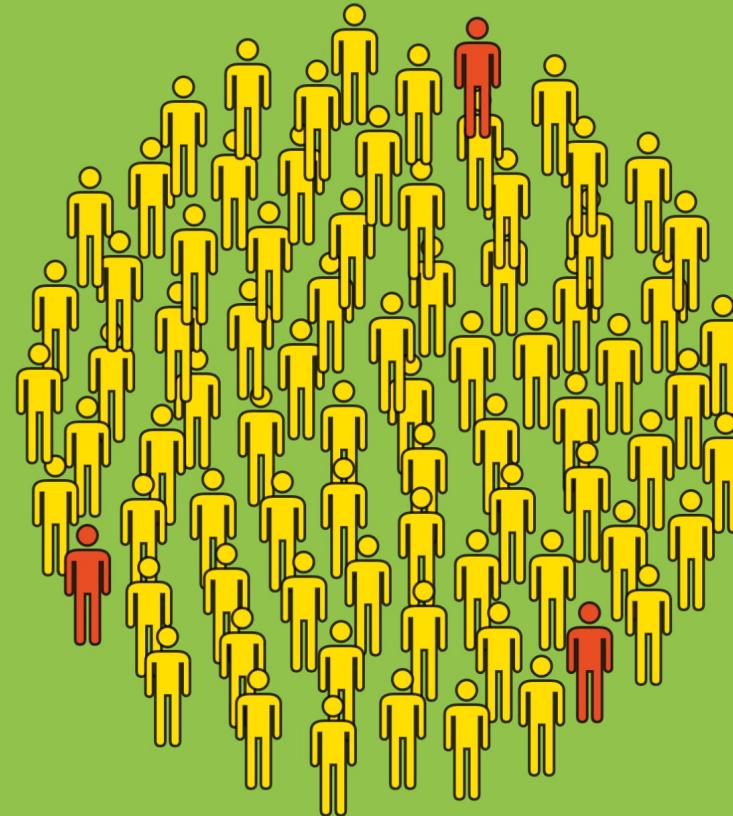


*ABMs are abstract, limited, idealised, description of reality that still captures a specific phenomenon.*



## *Model Interactions:* Well-mixed populations

## Well-mixed population



*All agents can interact with any other. All encounters between pairs of agents is equally likely*



# Susceptible-Infected Model

introduced by Kermack-McKendrick in 1927



*Compartment models: The internal details  
describing the complete state of an agent are  
unimportant*



## An agent is fully characterised by its *state*



*A susceptible state describes those agents who are in a healthy state, but are prone to become ill*



*An infected state describes those agents who are ill*

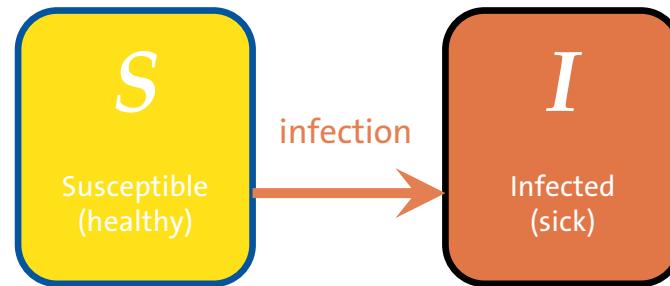


*Agents of the model:*  
**Represented by a categorical state variable**



## Susceptible-Infected Model

The agents can be in one of two states



A susceptible individual may become infected because of being in contact with an infected agent

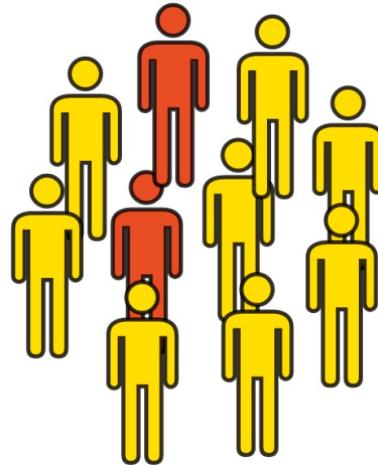
Infected individuals can infect susceptible ones when they get in contact



## *Model mechanism:* Infection



## Infection Dynamics



We consider a **small time-interval  $dt$**

*The probability that a susceptible agent gets infected is given by*

*Infection rate  
per social contact*



*Number of social contacts  
per time unit*

$\times dt$



## Assumptions of this approach

- All contacts have the same probability of infecting an agent
- All contacts are independent processes
- What happens in a time interval is independent of what happens in the next time interval
- $dt$  the time interval is small



## *Control Parameter: Infection Rate*

*Infection rate*

$$\beta$$



*Order Parameters:*  
**Global System properties**  
Number of infected and susceptible agents

$$N_I \ N_S$$



## Algebraic description of the SI model

- $N_S(t)$  is the number of individuals not yet infected by the disease (or those susceptible to the disease) at time t.
- $N_I(t)$  is the number of individuals who have been infected with the disease and are able to spread it to the susceptible ones.



## Infection Dynamics in a well-mixed population

*Infection rate*

$\beta$

*Number of social contacts  
per time unit*

$N_I$

*Infection rate  
per social contact*

$\times$

*Number of social contacts  
per time unit*

$\times dt$

$$\beta \times N_I dt$$



## The SI model: properties

- The probability that an agent gets infected is given by

$$p(S \rightarrow I) = \beta N_I dt$$

Where  $N_I$  is the number of infected individuals the agent had contact with in the time interval  $dt$

- How many infections will occur in a time interval  $dt$ ?

$$dN_I = \beta N_I N_S dt$$

- Equivalently

$$dN_S = -\beta N_I N_S dt$$



## The SI model: mathematical representation

- This creates the simple set of differential equations for the change in the number of infected and susceptible individuals

$$\begin{aligned}\frac{dN_S}{dt} &= -\beta N_S N_I \\ \frac{dN_I}{dt} &= \beta N_S N_I\end{aligned}$$

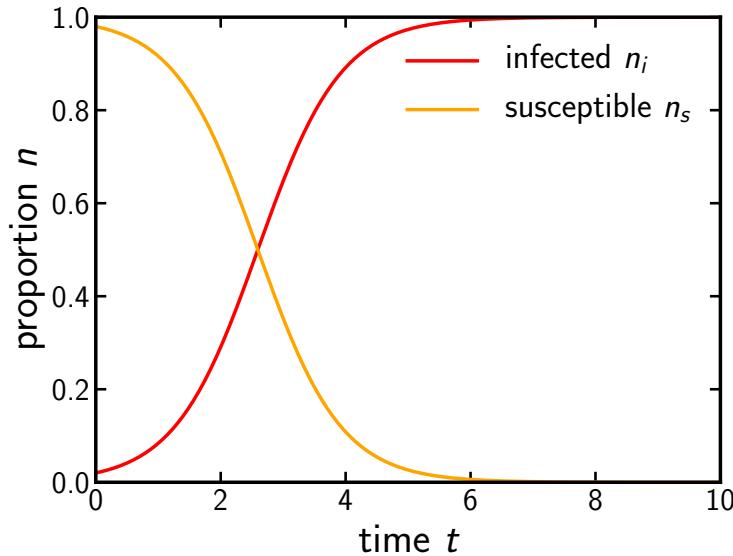
- By recalling that agents are either susceptible or infected and normalising

$$N_S + N_I = N \Rightarrow n_s + n_i = 1$$

- We get

$$\frac{dn_i}{dt} = \beta(1 - n_i)n_i$$

## The SI model: results



$$\frac{dn_i}{dt} = \beta(1 - n_i)n_i$$

The infection eventually takes over the complete population



## Overview

Emergent property

Time it takes for a disease to spread throughout the population

- Control parameter (exogenous): Infection rate  $\beta$
- Order parameter (endogenous):  $N_i$



## Elements – 4.1 Synchronous update rule

All agents have two states: susceptible and infected.

Randomly select one of the agents being infected initially and all other nodes are set to susceptible.

At each time step  $t$ ,

- if agent  $i$  is in the susceptible state and it has  $v$  infected neighbors, then agent  $i$  remains susceptible with probability  $(1 - \beta)^v$ , otherwise  $i$  gets infected at time  $t + 1$ .

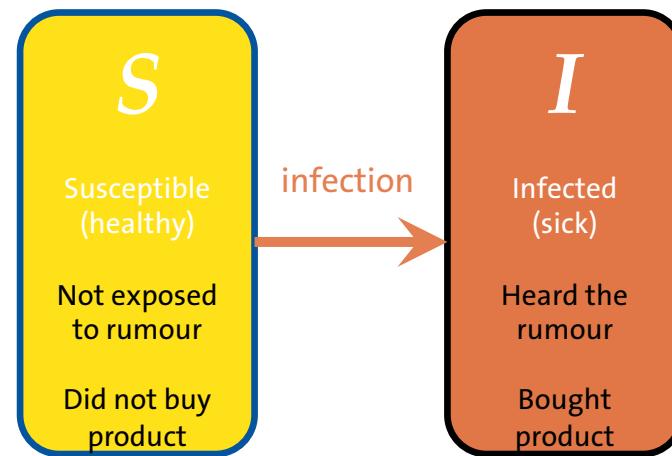
Run until the system reaches a steady state.



*When  $\beta$  is small, the dynamics is events are rare: most of the times there is NO dynamics, and the simulation is slow*



## Alternative interpretations of the states





## Interpreting the infection rate

$$dN_I = \beta N_I N_S dt$$

$\beta$  can represent the infection rate (i.e. a measure for the transmissibility)

it can also include the fraction  $\pi$  of the population with whom an individual has contacts

$$\beta_{eff} = \beta \times \pi$$



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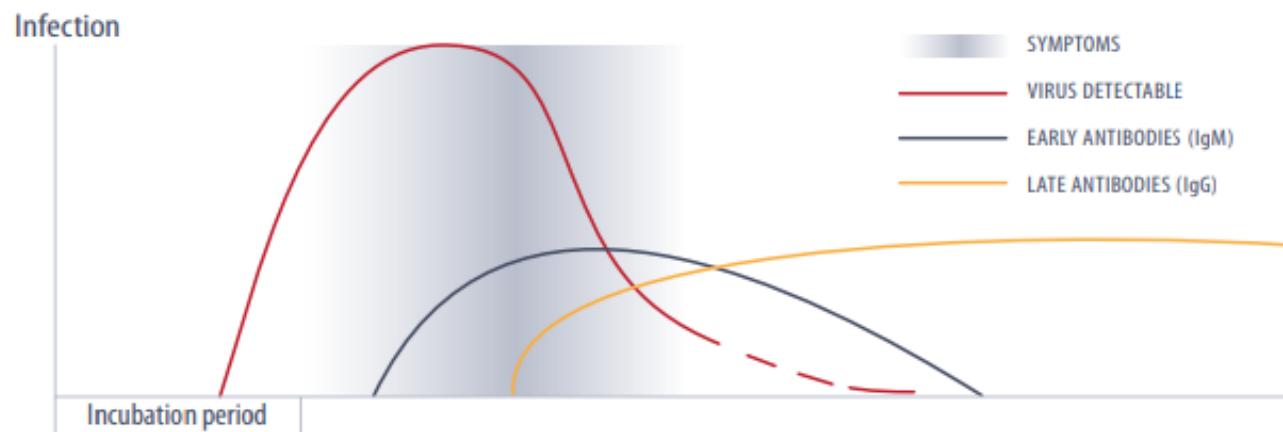
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# Susceptible-Infected-Recovered Model

## But exposed people become immune...

### Immune response

Antibodies to the COVID-19 virus (both IgM and IgG) start to appear between 1 to 2 weeks after symptom onset after which there is a slow, steady decline in viral load. Some patients may still be infectious after improving clinically & may require further isolation after hospital.





## An agent is fully characterised by its *state*



*A susceptible state describes those agents who are in a healthy state, but are prone to become ill*



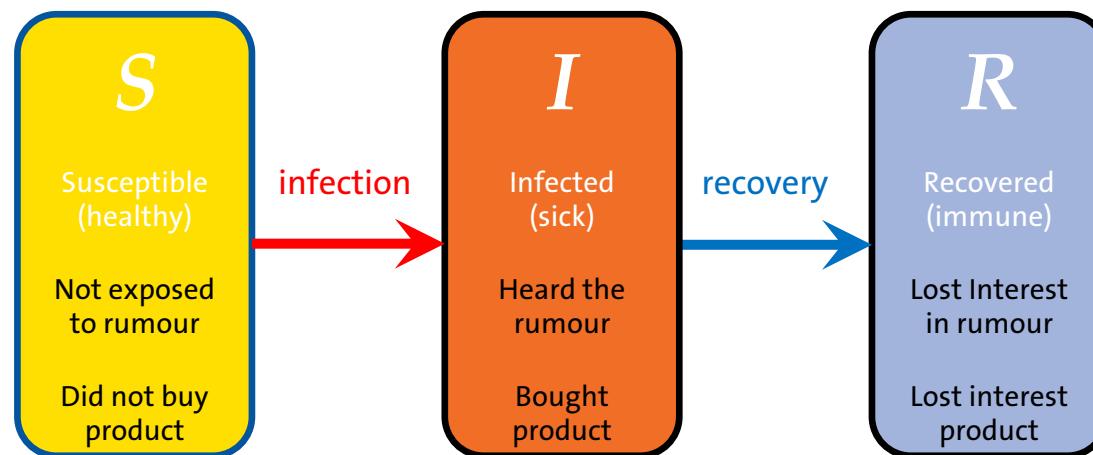
*An infected state describes those agents who are ill*



*A recovered or removed state describes those agents who are got the disease and cannot transmit it: e.g. because they recovered, or because they are dead*

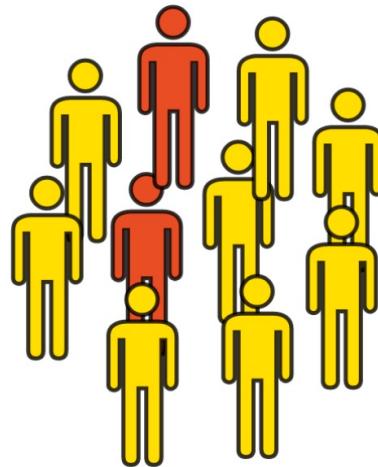
## The SIR model

The SIR model was proposed by Kermack and Mckendick in 1927. The aim was to model the spread of infectious diseases with recovery





## Infection Dynamics



We consider a **small time-interval  $dt$**

*The probability that a susceptible agent gets infected is given by*

*Infection rate  
per social contact*

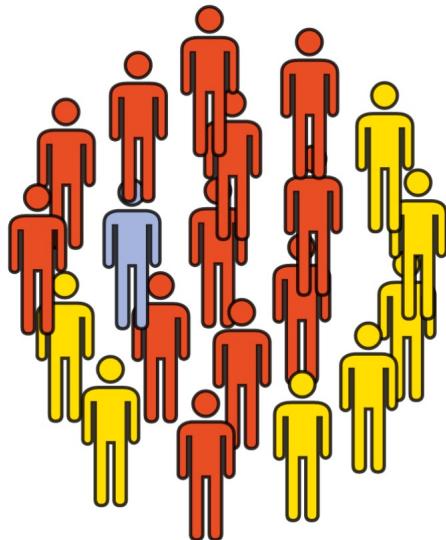


*Number of social contacts  
per time unit*

$\times dt$



## Recovery Dynamics



After some time, the agents recover – or die – and cannot propagate the disease

*Recovery rate*

$\gamma$



## Details of SIR model

- $N_I$  is the number of individuals not yet infected with the disease (or those susceptible to the disease) at time t.
- $N_S$  is the number of individuals who have been infected with the disease and are able to spread the disease to the susceptible ones.
- $N_R$  is the number of individuals who have been infected and cannot propagate anymore the disease, either due to immunisation or due to death.



## The SIR model: properties

- Infections occur like in the SI model
- Agents recover at a rate  $\gamma$

$$p(S \rightarrow I) = \beta N_I dt$$

$$p(I \rightarrow R) = \gamma dt$$

- How many infections will occur in a time interval  $dt$ ?

$$dN_I = \beta N_S N_I dt$$

$$dN_S = -\beta N_S N_I dt$$

- How many recovered individuals appear in a time interval  $dt$ ?

$$dN_R = \gamma N_I dt$$



*Infection grows with the number of contacts  
between healthy and infected agents*

*Recovery is proportional to the number of  
infected individuals*



## The SIR model: mathematical representation

- Once again recalling that

$$N_S + N_I + N_R = N \Rightarrow n_s + n_i + n_r = 1$$

- We get a set of differential equations

$$\begin{aligned}\frac{dn_S}{dt} &= -\beta n_S n_I \\ \frac{dn_I}{dt} &= \beta n_S n_I - \gamma n_I \\ \frac{dn_R}{dt} &= \gamma n_I\end{aligned}$$

[applet]



## The SIR model: implications

The time evolution is given by

$$n_I = \left(1 - \frac{\gamma}{\beta}\right) \frac{Ce^{(\beta-\gamma)t}}{1 + Ce^{(\beta-\gamma)t}}$$

By defining the reproductive number  $R_0 = \frac{\beta}{\gamma}$

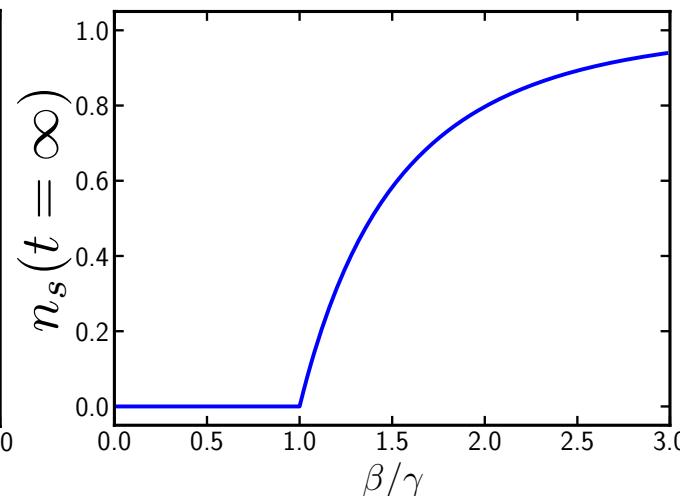
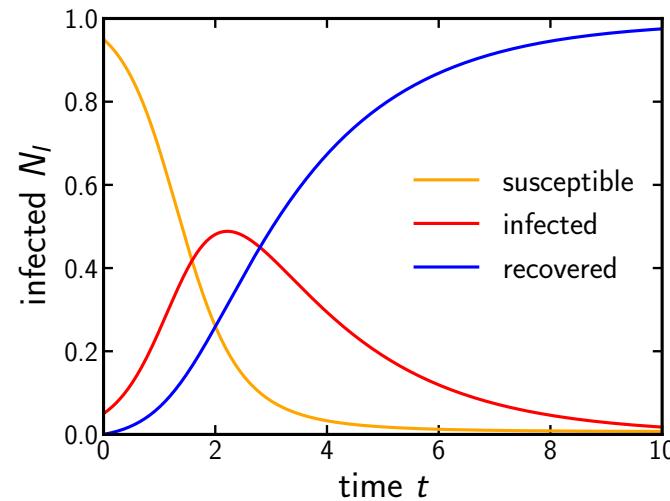
Whenever  $\left(1 - \frac{1}{R_0}\right) < 0$       or       $R_0 < 1$

The infection dies out

## The SIR model: results

$$n_s(t = \infty)$$

- The total number of recovered individuals at large times, is equal to all those that were infected





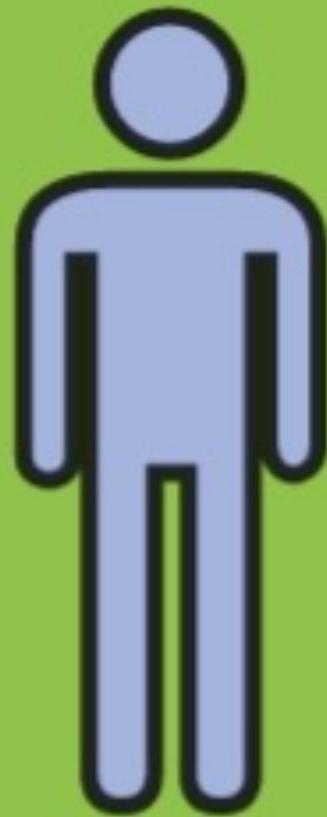
## The SIR model: implications

***Recovery rate:*** Cannot be changed in absence of medication

***Infection rate:*** Can effectively be changed by reducing social contacts (reduced mobility), or reducing the effectiveness of disease spreading (wearing masks)



Recovered individuals...



*also represent vaccinated agents*



## Elements – 4.1 Synchronous update rule

All agents have one of three states: susceptible, infected, and recovered.

Randomly select one of the agents being infected initially and all other nodes are set to susceptible.

At each time step  $t$ ,

- ⊕ if agent  $i$  is in the susceptible state and it has  $v$  infected neighbors, then agent  $i$  remains susceptible with probability  $(1 - \beta)^v$  , otherwise  $i$  gets infected at time  $t + 1$ .
- ⊕ If agent  $i$  is in the infected state at time  $t$  then  $i$  will be recovered with probability  $\gamma$  at time  $t + 1$ .

Run until the system does not have infected individuals



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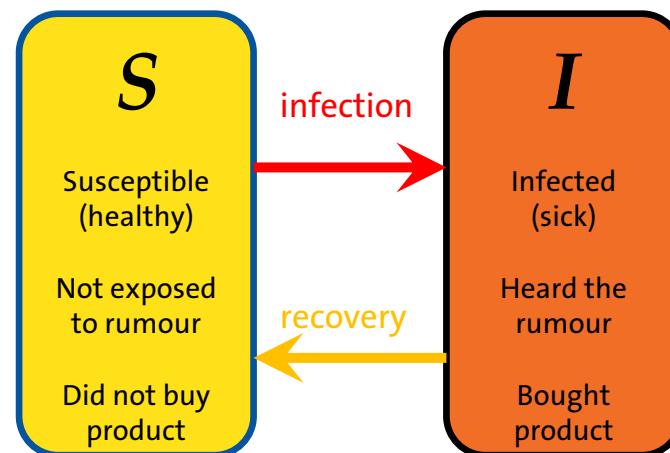
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# Susceptible-Infected-Susceptible Model

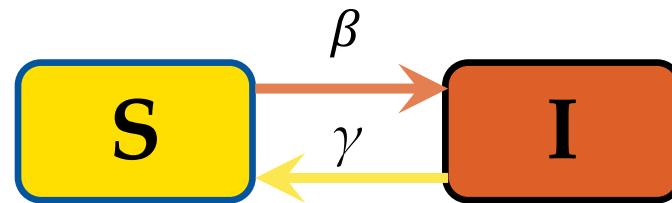
## The SIS model is a variant of the SIR model

- The SIS model can be easily derived from the SIR model by assuming that individuals recover with no immunity to the disease.
- This implies that the individuals “removed” from the infected group will enter the “susceptible” group immediately (reinfection).





## Susceptible-Infected Model



- A susceptible individual becomes infected because of being in contact with an infected at a constant rate  $\beta$
- Infected individuals recover - but become susceptible again at a rate  $\gamma$



## Mathematical representation of the SIS model

- Similar to the SIR model, the number of susceptible individuals decreases as

$$dN_s = -\beta \frac{N_S N_I}{N}$$

- Besides, individuals recovered from infection will enter the susceptible group. We will then have:
- The change of infected population remains the same as the SIR model:

$$\begin{aligned}\frac{dn_S}{dt} &= -\beta n_S n_I + \gamma n_I \\ \frac{dn_I}{dt} &= \beta n_S n_I - \gamma n_I\end{aligned}$$



## The Reproduction Number

- Both, in the SIS and SIR model the fraction of infected individuals follows

$$\frac{dn_I}{dt} = (\beta n_S - \gamma) n_I$$

- Let us concentrate on the case  $n_S \approx 1$  (i.e. just after the disease outbreak started)

$$\frac{dn_I}{dt} \approx (\beta - \gamma) n_I$$



# The Reproduction Number

- The solution is

$$n_I(t) = n_I(t=0) e^{(\beta - \gamma)t}$$

- So, when the population is composed by susceptible individuals, the evolution is exponential

- $\beta - \gamma > 0$  exponential growth
  - $\beta - \gamma < 0$  exponential decay



## The Reproduction Number

$$\beta - \gamma < 0 \quad \text{implies} \quad R_0 \equiv \frac{\beta}{\gamma} < 1$$



# *Will an epidemic outbreak occur or will die out?*

It depends on the *Basic Reproduction Rate*, the average number of susceptible individuals an infected person will infect

$$R_0 = \frac{\beta}{\gamma}$$

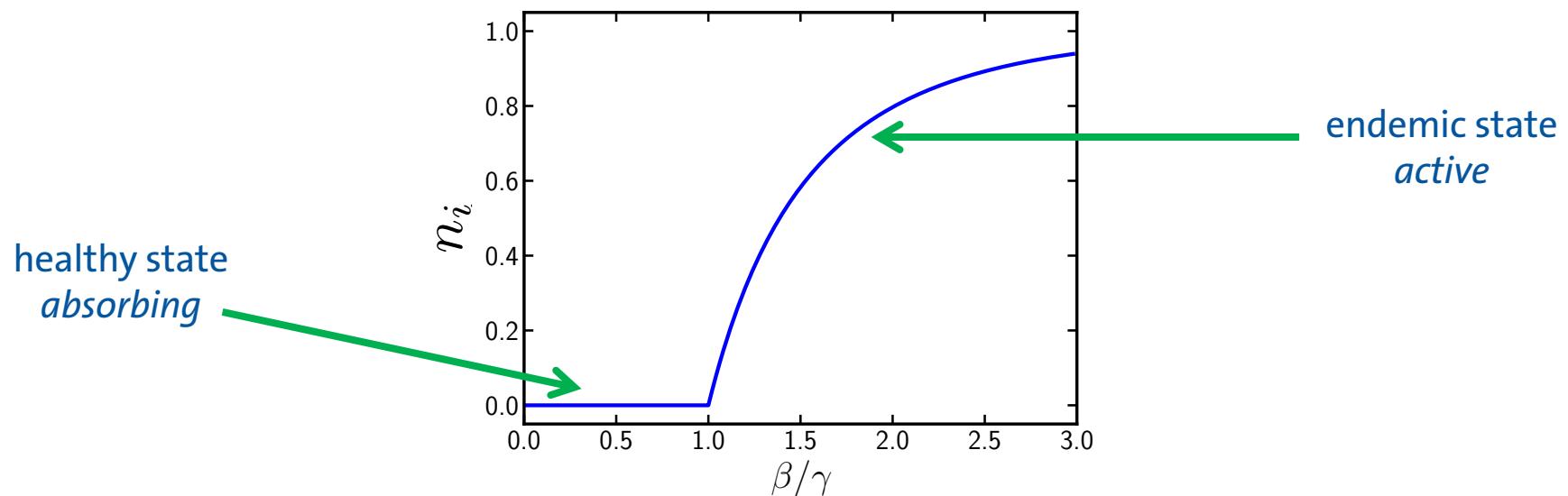


## Some real-world values

Disease	Transmission	R <sub>0</sub>
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
Covid-19	Airborne droplets	2-4

## Stationary state

This model converges to a stationary state that is dynamic



**Absorbing (healthy) state:** the fraction of agents in the infected state vanishes in the long run.

**Active (endemic) state:** a finite fraction of the population is always in the infected state. The disease becomes endemic



*There is a phase transition as a function of  
the order parameters.*

*The global state changes completely  
depending on whether  $R_0$   
is above or below 1*



## Elements – 1. Agents

The state of an agent is a three-state variable:  $(0, 1)$  /  
(Susceptible/Infected).

There are  $N$  agents in the system

- ✚ *How to represent it?* An array  $S$  with the states, of dimension  $N$  holding the values
- ✚  $S[i]$  is the state of agent  $i$

Initialisation: a set of  $N_0$  agents is set to state Infected



## Elements – 2. Network

### Who are the neighbours?

Original SIR model considers a fully connected network, i.e. all agents influence all others

A more realistic approach considers that each agent has a finite set of social interactions, and there is - therefore - an underlying network



## Elements – 4.1 Synchronous update rule

All agents have two states: susceptible and infected.

Randomly select one of the agents being infected initially and all other nodes are set to susceptible.

At each time step  $t$ ,

- ⊕ if agent  $i$  is in the susceptible state and it has  $v$  infected neighbors, then agent  $i$  remains susceptible with probability  $(1 - \beta)^v$  , otherwise  $i$  gets infected at time  $t + 1$ .
- ⊕ If agent  $i$  is in the infected state at time  $t$  then  $i$  will be susceptible with probability  $\gamma$  at time  $t + 1$ .

Run until the system reaches a steady state.



## Overview

Emergent property

Whether a disease becomes endemic or not

- **Control parameter (exogenous):** Infection rate  $\beta$ , recovery rate  $\gamma$
- **Order parameter (endogenous):**  $N_i$



## Epidemic Threshold

The basic reproductive number  $R_0$  is defined as the average number of secondary infections caused by a primary case introduced in a fully susceptible population In homogeneous mixing it is equal to the control parameter  $R_0 = \lambda = \mu\beta$



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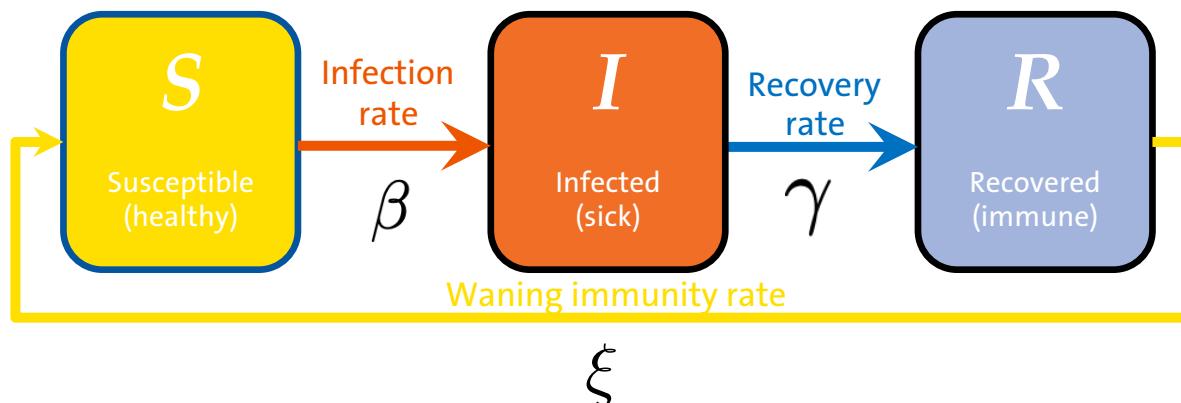
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# Susceptible-Infected-Recovered-Susceptible Model

## Susceptible-Infected-Recovered-Susceptible Model

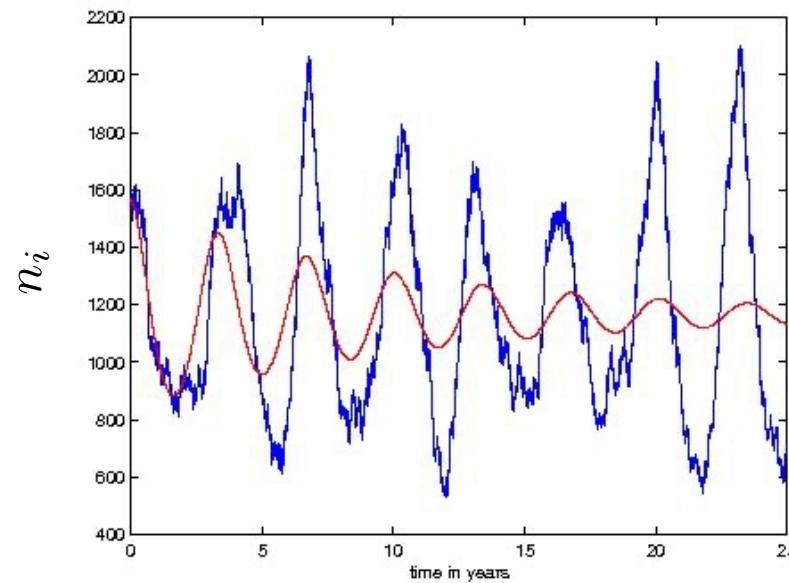
The SIRS model was proposed by Kermack and Mckendick in 1927. The aim was to model the spread of infectious diseases with recovery



- After recovering of the disease, agents' immune response decreases and the agents become susceptible again

## The SIRS model: results

- There are self-sustained oscillations. The results are different from numerical integration of the equations





*These results with respect to the epidemic threshold will change when we consider spreading in networks*



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