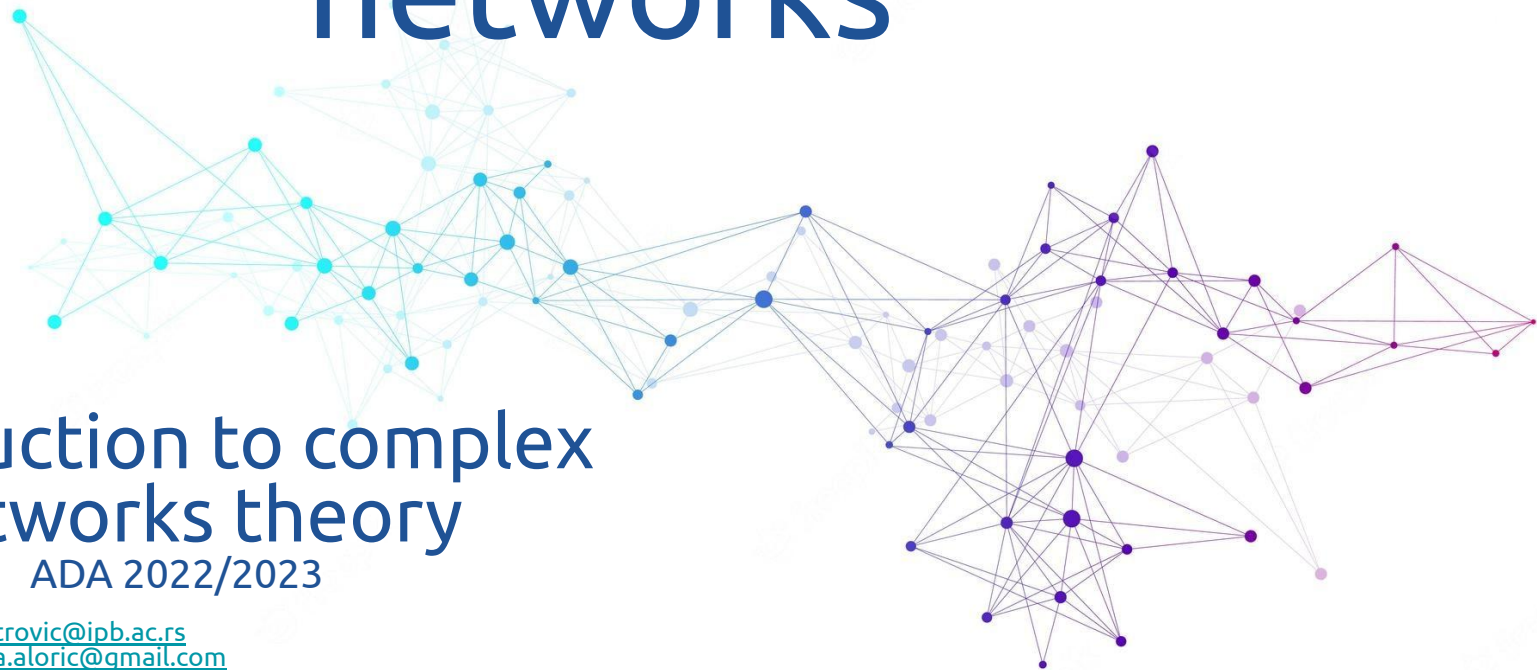


# Spreading processes on networks



## Introduction to complex networks theory

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# Aim

- Gain intuition about spreading processes and particularly how network structure affect the spreading process
- Create simulations in python and use them to investigate different epidemic spreading processes

# So far we have:

- Learned how to analyse structure of networks:
  - Micro: Node centralities
  - Mezo: Community detection
  - Macro: Network connectedness
- We shown some examples where these structural features can reveal tell us something about underlying systems, e.g.:
  - Degree distributions
  - Communities
- That way we have analysed networks that are trace of some sort of activity in the system we analyse:
  - Friendships, romantic relationships, collaborations...

# But networks are rarely static and isolated objects

- To overcome this, we discussed temporal networks and their application as a way to observe changes in the studied system via tracking changes in the network structure
- But more commonly, we think about a network together with some process that happens on top of it and potentially modifies it along the way
- Most typically, we look at the spreading processes
  - Infectious disease
  - Rumor spreading
  - Computer malware
- For these processes, a structure of connections between individual objects that are affected by the spread (people or animals in case of disease, computers or other devices in case of malware) - network - has a strong effect on the speed and effects of the spreading process

# Spreading processes on a network

- State: Each element (node) can be in one state (knows gossip, infected by a virus)
- Update rule: How a node changes state, what needs to happen for a given node to switch its state among possible options
- Key message is that we treat network edges as transmission paths -> node changes its state depending on the states of its neighbours, e.g. edges transmit information/viruses
  - This is why structure of the contact network plays an important role in the spreading process

# Epidemic modelling

# SI-X models



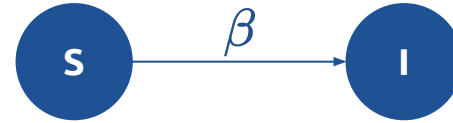
A modelling framework where we neglect various complexities of an personalized experience of the disease, different medical and biological mechanisms that occur within a body of infected individual and instead focus only on the global state of the individual, is it:



- **Susceptible** (S) to the disease, or it is already
- **Infected** (I) by the disease
- In the simplest case, when using the **compartmental model**, we do not care about the structure of interactions within the population (everyone can infect everyone) and track only the total number of subpopulations (compartments) of people who are still susceptible or infected
  - This basically means we study a fully connected network (in terms of transmission paths) and track only the total number of nodes in each given state instead of caring about which node is in which state
- The way we define update rule (and possible additional states) differentiate between different families of SI models, e.g. SI, SIS, SIR, SEIR models
- We often study fixed size populations in these models, e.g. we neglect the births/deaths that are not related to the modelled disease

# SI model

- Only two states:
  - Susceptible
  - Infected
- Update rule:

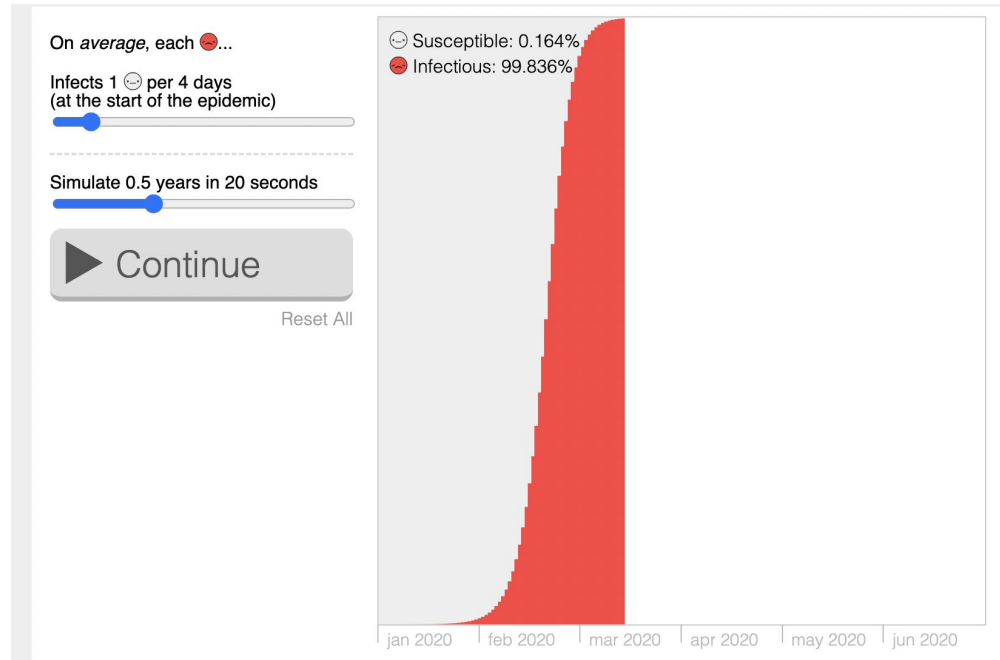


- Infected person infects one susceptible person with probability  $\beta$  within unit time
- In this scenario, the only allowed flow is from susceptible to infected individuals, once infected, person stays in that state
- This model although very simplistic is a decent approximation of the behavior of diseases like those caused by herpes that has lifelong presence within the infected organism



# SI model animation

Use animation to build intuition -> what happens when model parameter changes



# A bit of math

The process can be described by the following two differential equations:

$$\frac{dS}{dt} = -\beta S \frac{I}{N}$$

$$\frac{dI}{dt} = \beta S \frac{I}{N}$$

Using the fact that the total size of population  $N$  does not change and it is at any time  $S+I$ , this equation describes the change of infected users in time:

$$\frac{dI}{dt} = \beta I \left(1 - \frac{I}{N}\right)$$

From this equation, we see that the only two stable states (when the derivative will be 0, e.g. no more changes in the number of infected individuals) is **either** when  $I = 0$  (**nobody is infected**), **or** when  $I = N$  (**everybody is infected**). This agrees with our conclusion from playing around with previous SI implementation - independent of beta, everyone will be infected at some point in time, beta only affects the time it takes the system to get there.

# SIS model

- Still only two states:

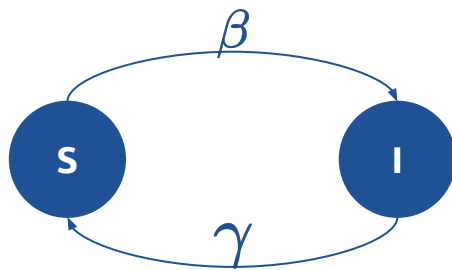
- Susceptible
- Infected

- But now infected individual can become susceptible again, so the new update rules:

$$S + I \xrightarrow{\beta} 2I$$

$$I \xrightarrow{\gamma} S$$

- This model makes more sense when we model diseases where a person can recover, but does not gain long term immunity, common cold, influenza, many STDs



# How math changes

The SI equations are now slightly altered:

$$\begin{aligned}\frac{dS}{dt} &= -\beta S \frac{I}{N} + \gamma I \\ \frac{dI}{dt} &= \beta S \frac{I}{N} - \gamma I\end{aligned}$$

As well as the single equation that describes changes in the number of infected individuals (I) where we used that S and I are always summed to N:

$$\frac{dI}{dt} = \beta I \left(1 - \frac{\gamma}{\beta} - \frac{I}{N}\right)$$

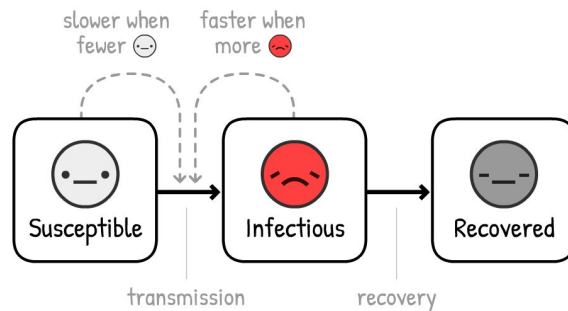
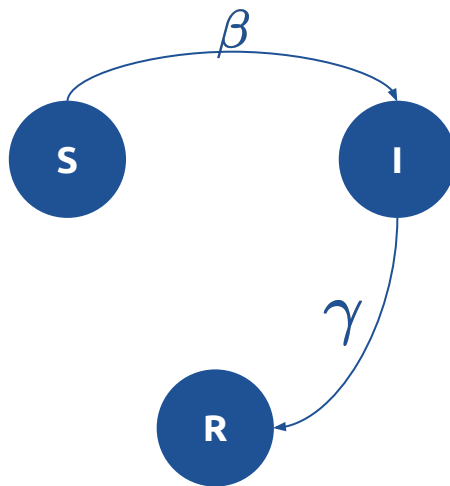
Here, too, we see that  $I = 0$  is one stable state, e.g. whenever there's no infected individuals, the total number of infected individuals don't change (the disease is eradicated), but depending on beta and gamma, the final state does not need to be that everyone is infected.

# SIR model

- Possible states:
  - Susceptible
  - Infected
  - Recovered (sometimes also Removed)
- Update rules:



- This model is a decent approximation of diseases like measles or similar where recovery gives you immunity



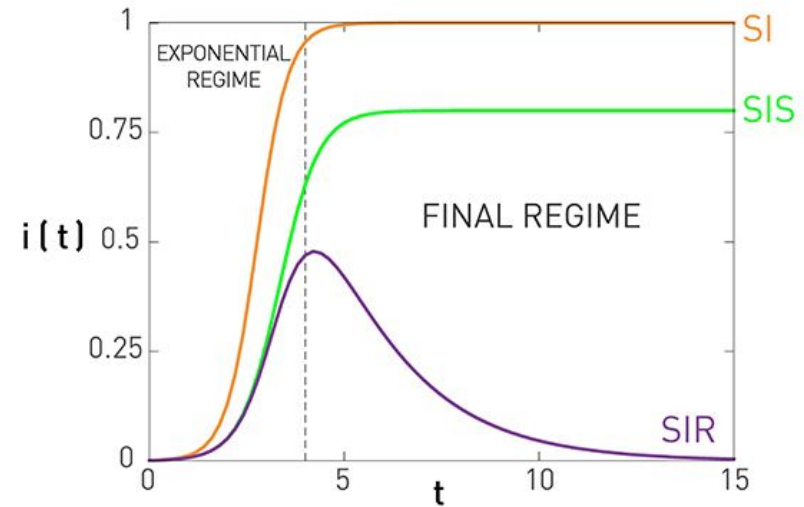
# SIR model animation

Use animation to build intuition -> what happens with parameter changes



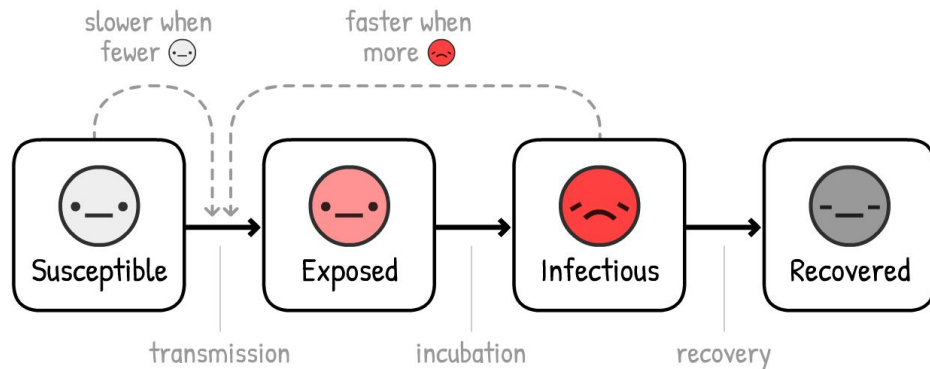
# SI/SIS/SIR solution comparison

- Depending on the characteristics of the disease different types of models will be more suitable for its modelling
- Early stages of the epidemic are similar - exponential regime
- Long term outcomes differ
  - SI - everyone become infected
  - SIS - the disease is either dies out, or reaches endemic state with finite fraction of always infected individuals
  - SIR - everyone recovers at the end



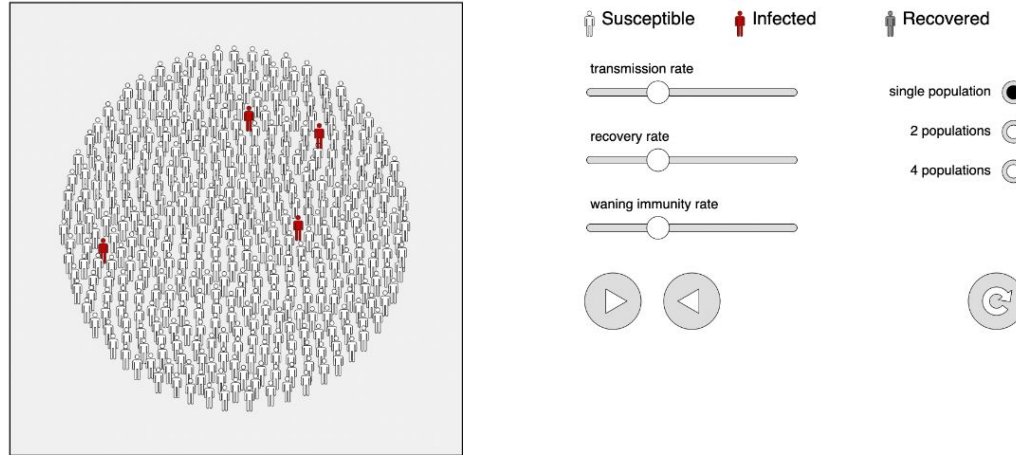
# Other SI models

- SIRS - recovery does not grant lifelong immunity, but once recovered, an individual can become susceptible again
- SIRD/SIRV - Diseased/Vaccinated, when your main goal is to track total number of infected individuals at any given time, these categories can be within R (then it's called removed rather than recovered), but when you want to model the deaths or vaccinations, you want that state explicitly
- SEIR(S) - Exposed, when you want to take into account the delay from exposure to the virus until the infectious stage
- Even more models you can find for example on [Wikipedia](#)





# The SIRS epidemic model



based on Complexity Explorable:

*" **Epidemonic** - An example for contagion dynamics in a population"*

# Network epidemics

# What when network is not fully connected?

- So far, we discussed different possible states and update rules, but always assuming everyone can infect anybody in the population
- That is not a realistic assumption, as we have seen in previous lectures, networks are rarely fully connected or regular, but there is significant structure in their degrees and other properties
- We need to move away from the fully connected network and instead look at the **appropriate** contact networks between individuals
- In networks, the initial state (where in network is the patient zero) is also going to be of great importance for the speed and final state of the epidemic

# Virus on a network playable animations

powered by NetLogo

Virus on a Network

File: New

Export: NetLogo HTML

Mode: Interactive Commands and Code: Bottom

model speed

ticks: 200

number-of-nodes 150

average-node-degree 6

initial-outbreak-size 3

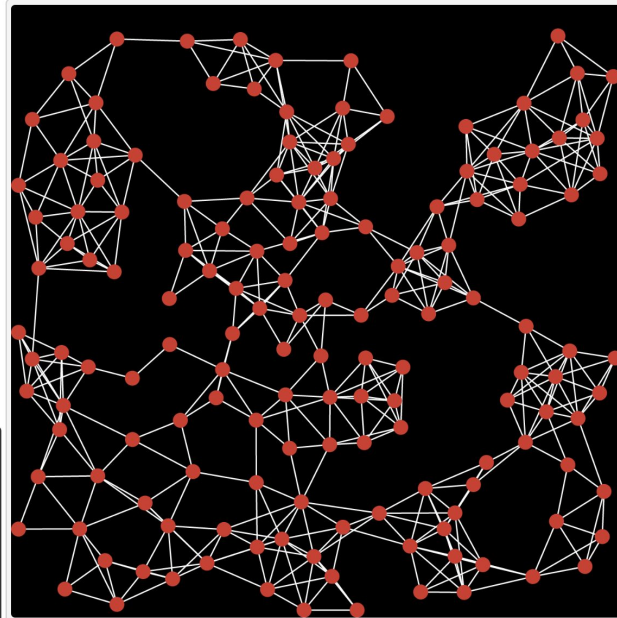
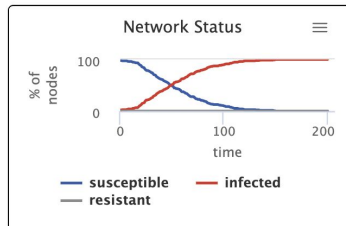
setup go

virus-spread-chance 2,5 %

virus-check-frequency 1 ticks

recovery-chance 0 %

gain-resistance-chance 0 %



python practice time

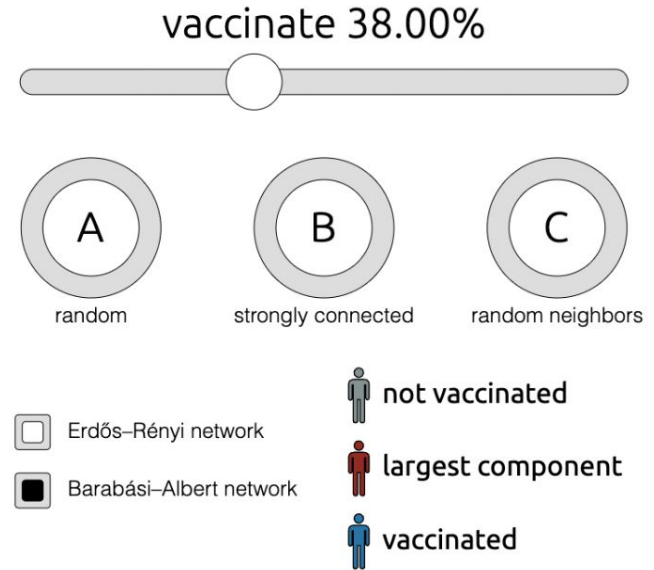
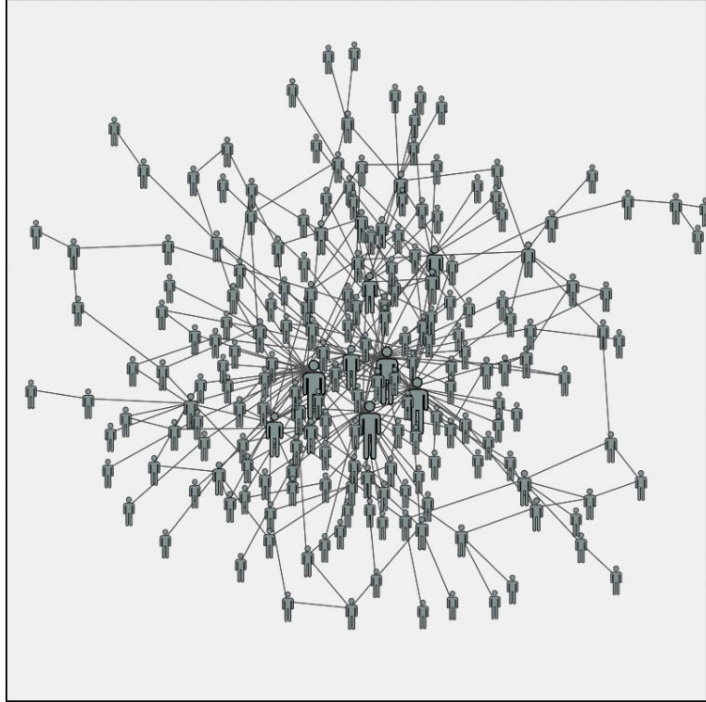
# What's different

- Depending on the network connectivity, now even in the SI model, it is not necessary for everyone to get infected
- The speed of disease spread is not only dependent on the beta/gamma parameters but the initial state and network characteristics in general

# Contact network

- The big challenge is collect the adequate contact network
- It depends on the type of disease we study:
  - Who spent time with whom - for airborne diseases like common cold
  - Who was in physical contact with whom - for diseases like Conjunctivitis, Ebola...
  - Network of sexual interactions - for sexually transmitted diseases
  - Bipartite network between humans and animals (e.g. mosquito) for malaria
- Many of these networks are very difficult to collect directly, but we can try to collect some or model them based on other indirect data that is more easy to collect

# Network robustness exploration can help us in designing effective vaccination strategies - try out!







[Tour >](#)

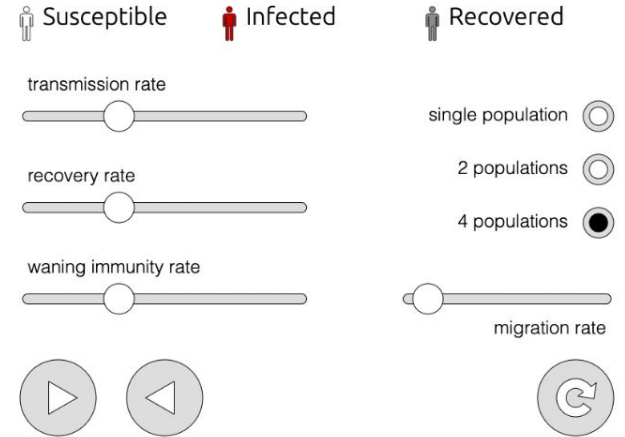
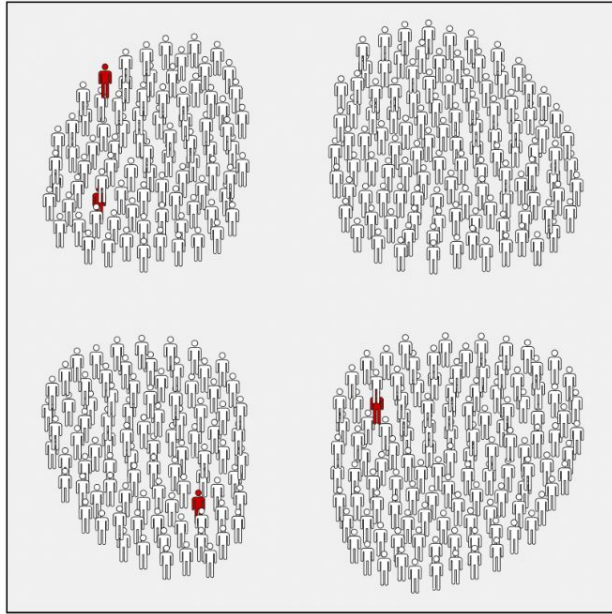
[Full Game >](#)

[Herd Immunity >](#)

Salathé Group | 2014

# Here you can investigate spread with communities affect spread across different communities:

This explorable illustrates the dynamics of the SIRS epidemic model, a generic model that captures disease dynamics in a populations or related contagion phenomena.



# Conclusions

- Network structure affects processes that occur in population
- Edges represent transmission paths and thus networks between the same individuals are different for different diseases
- Same dynamical rules, e.g. same transition rates from susceptible to infected individuals can have dramatically different effects based on the population network structure and position of the patient zero

# Further reading

- Explore further animation mentioned:
  - Here for compartmental models: <https://ncase.me/covid-19/>
  - Here for compartmental and network models:  
<https://www.complexity-explorables.org/slides/epidemonic/>
- Read [chapter 10](#)