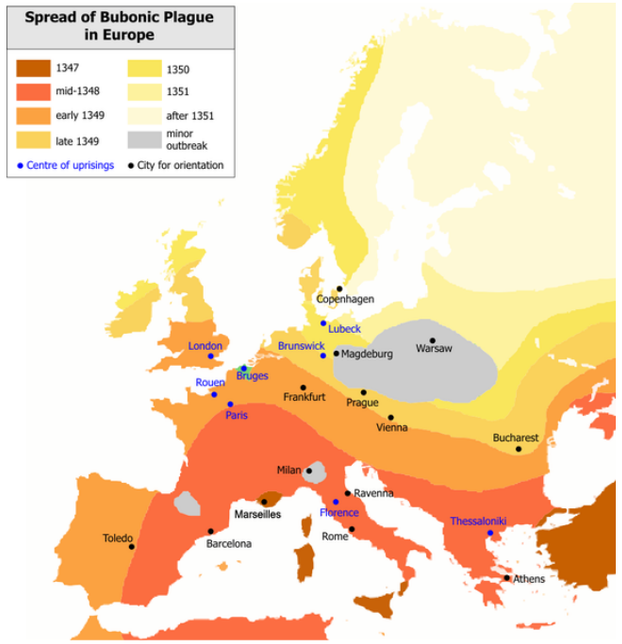
NETWORK DIFFUSION

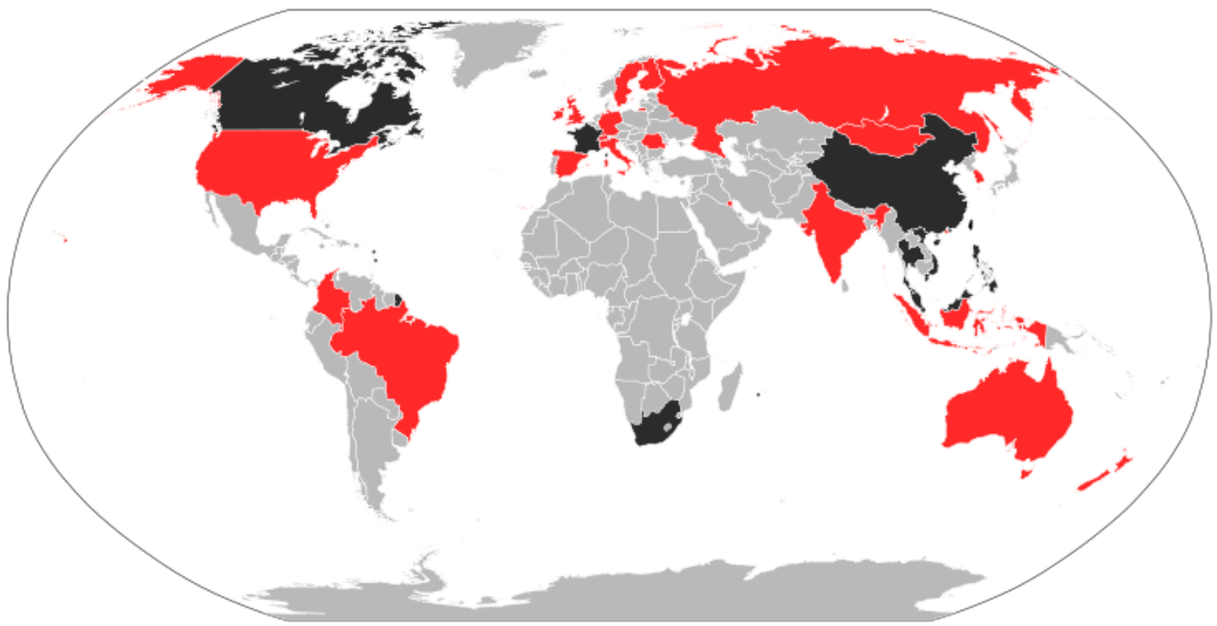
Spreading processes
Dynamic ON networks

Spreading processes

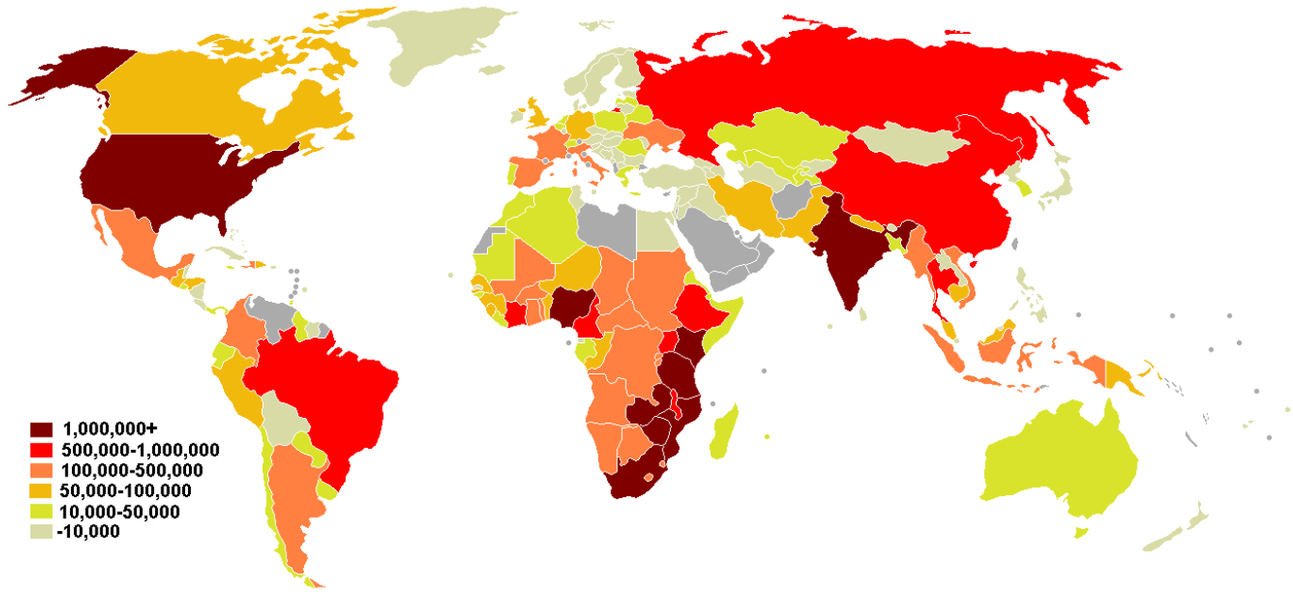
Biological epidemic spreading



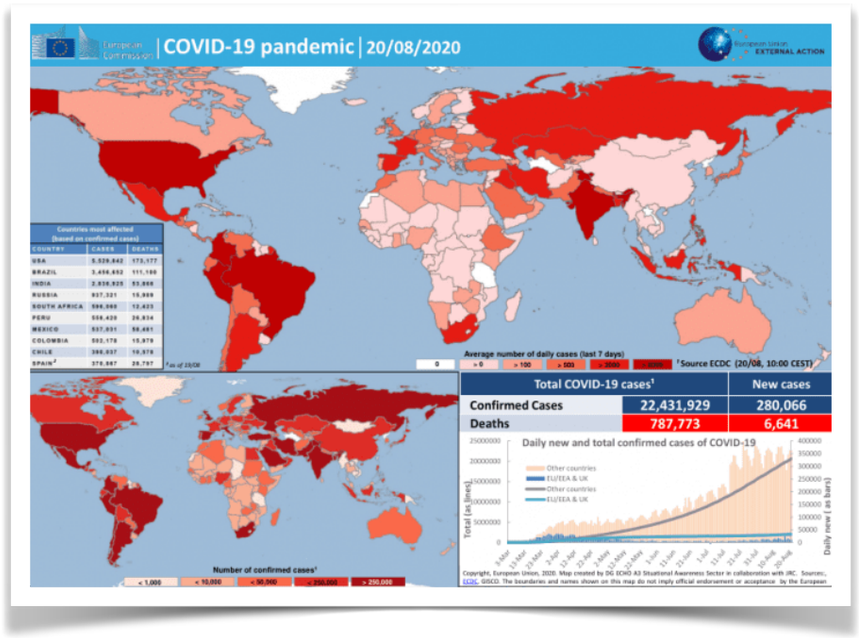
The great plague (14th century)



SARS (2008)



HIV (2008)

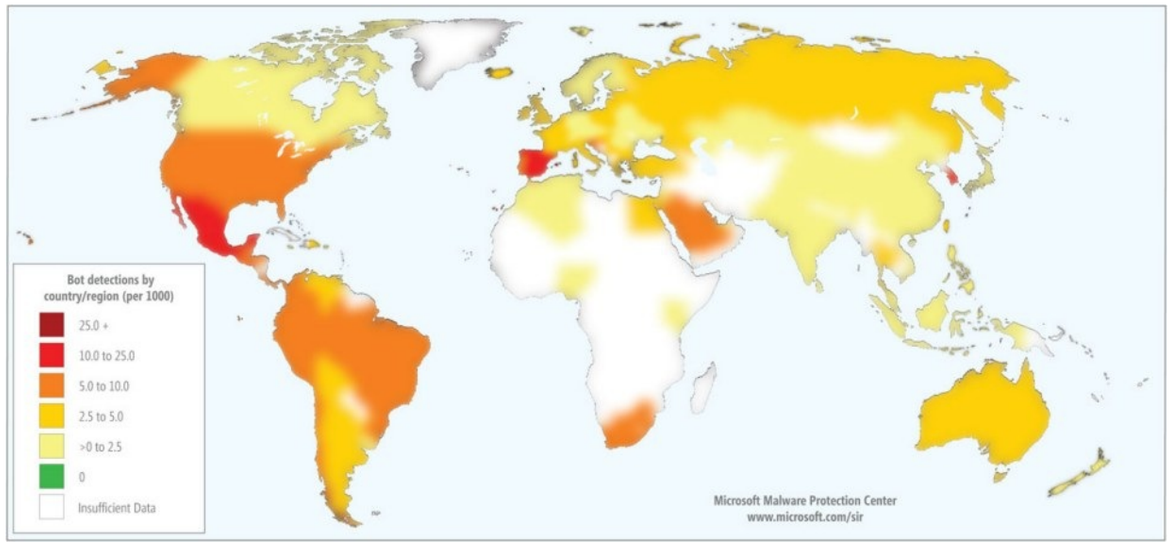


Covid-19

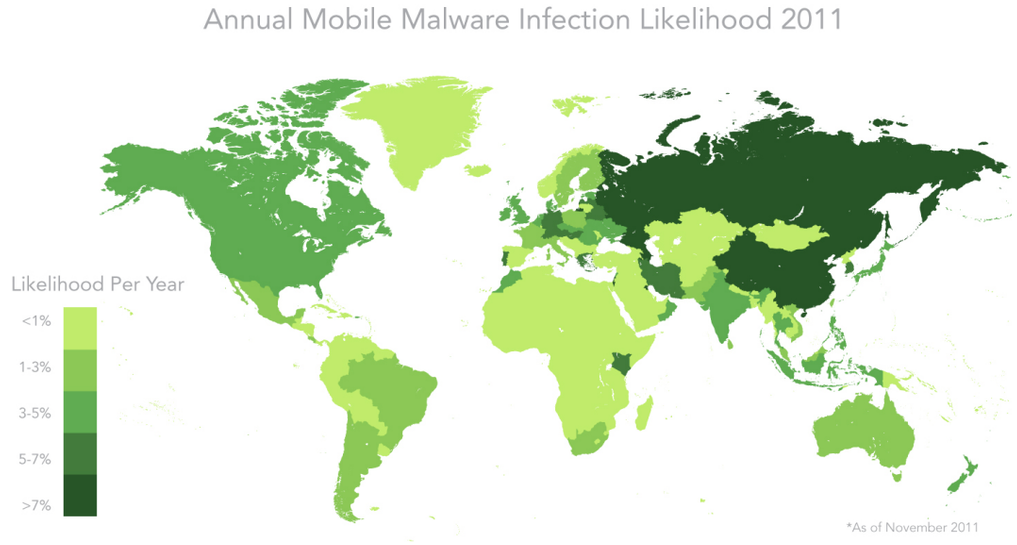
Wikipedia

Spreading processes

Malware spreading



Botnet infections (2010)

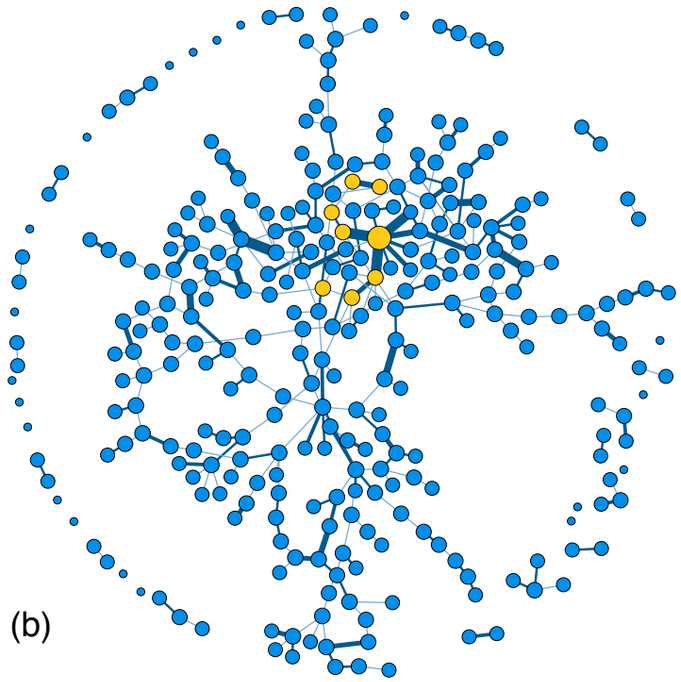


Mobile malware (2011)

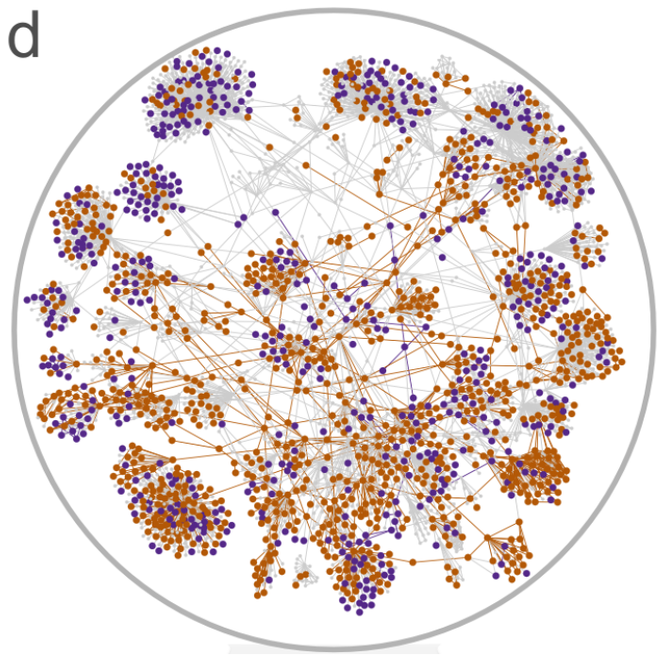
Spreading processes

Social contagion

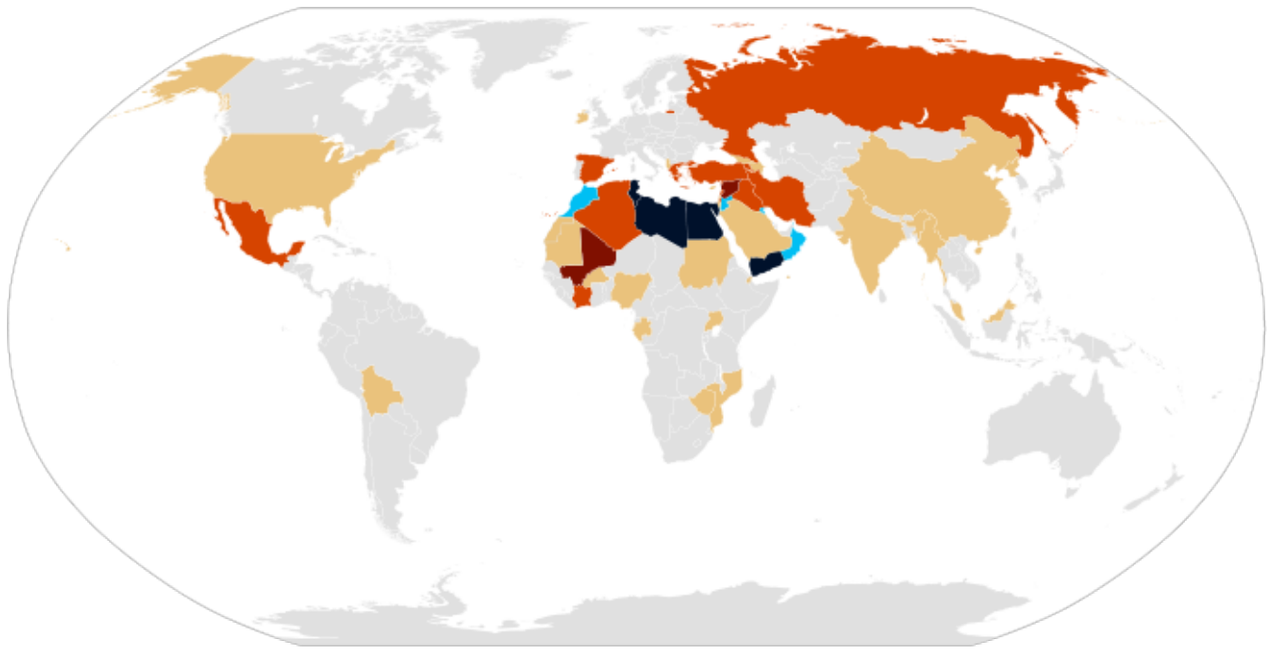
Information spreading



Rumour spreading
Karsai et.al. (2014)



Adoption spreading (Skype)
Karsai et.al. (2014)



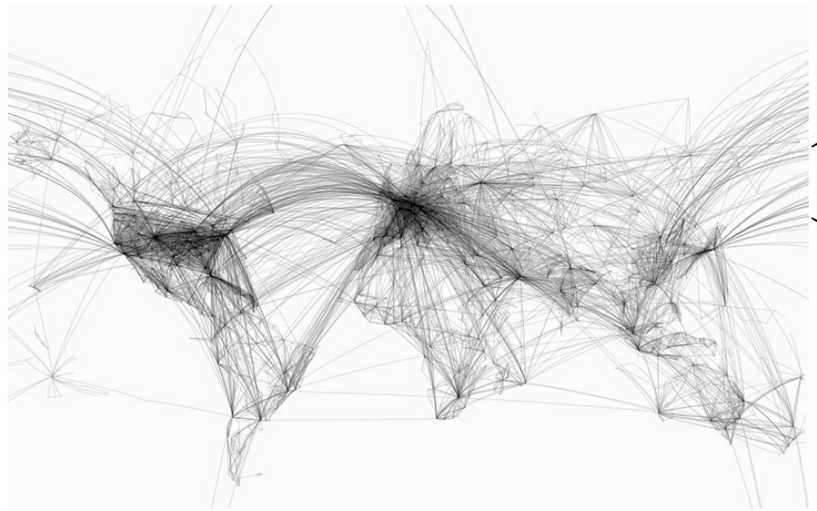
Protest diffusion (Arabian spring)

Spreading processes

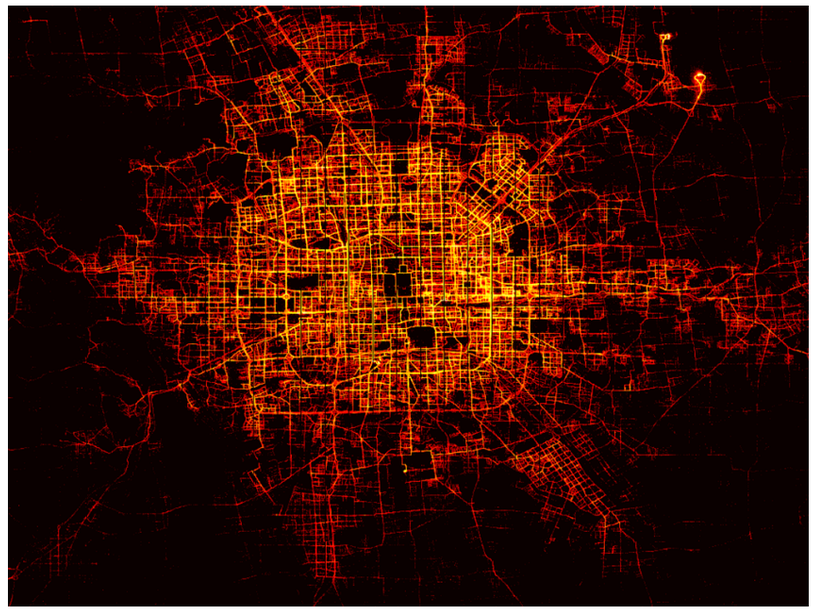
Why on networks?

- Spreading usually happen through interactions between agents
  - Geographic vicinity
  - Physical connection
  - Social interaction
  - etc.
- Network structure critically influence the dynamics of spreading processes

PPD blog, Jooseery (2011)

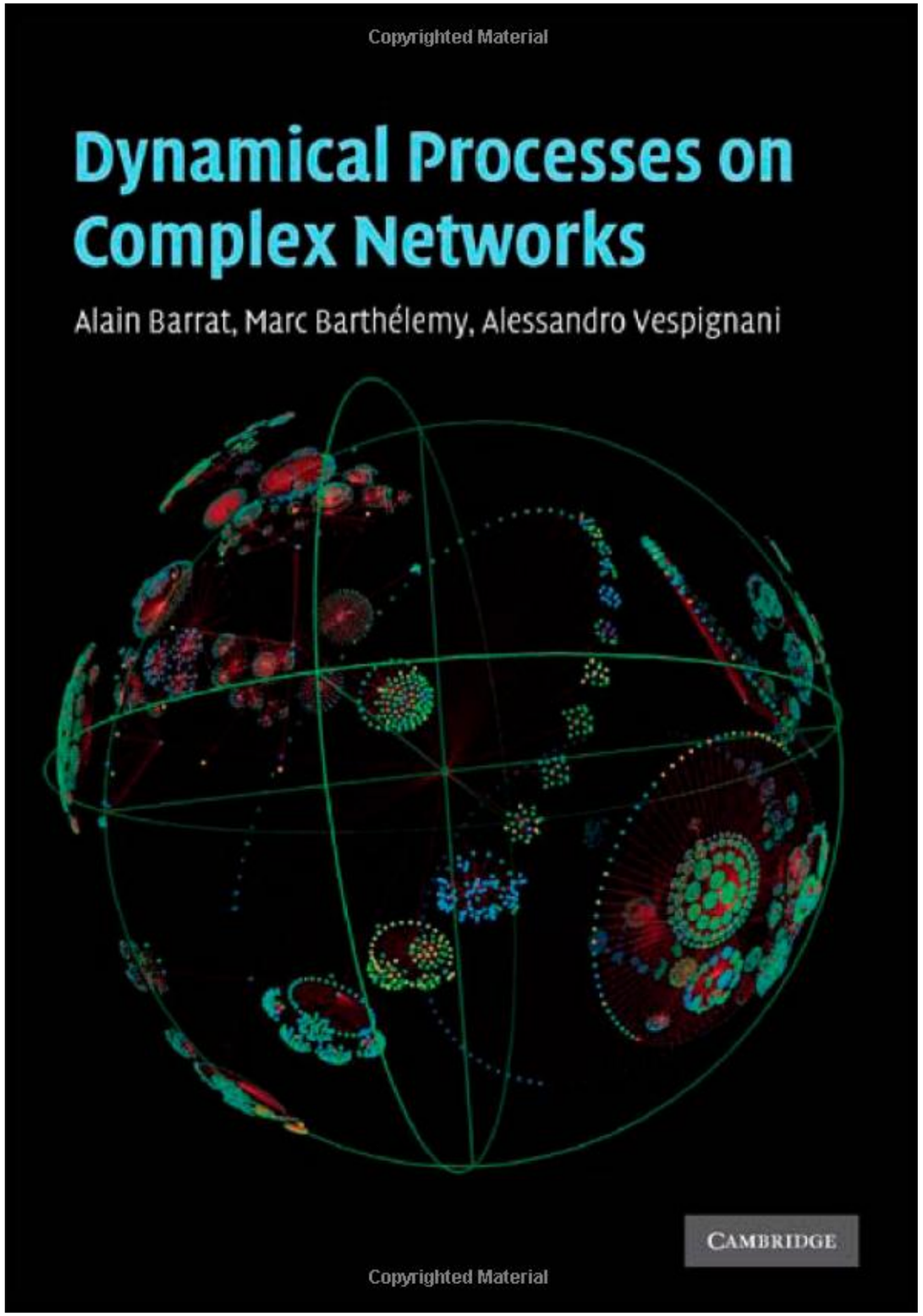


Freese (2009)



Unknown

Literature



I'm not an epidemiologist!

Only an introduction,
Trust the experts

Simple spreading processes

Spreading processes

SI - SIR - SIS

Three of the most popular models of diffusion in epidemiology are the **SI**, **SIR** and **SIS** models. Letters correspond to the states in which individuals can be according to the model:

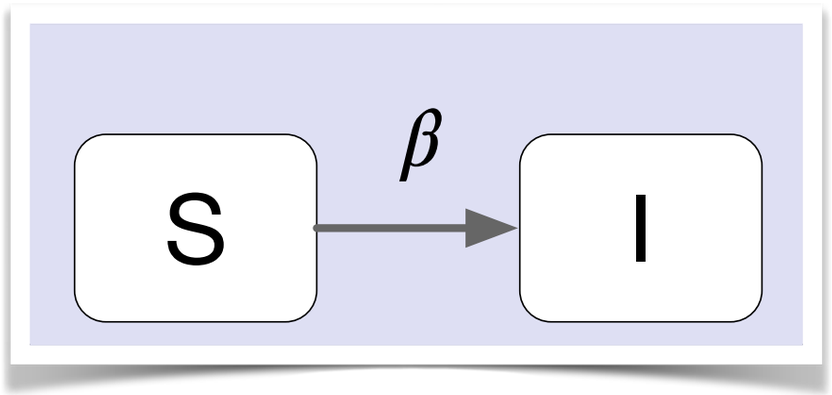
- **Susceptible**: Individual is not Infected
- **Infected**: Individual is Infected
- **Recovered/Removed**. Individual cannot be infected again (Considered cured or dead)

All individuals are in one of the states allowed by the model, and we define:

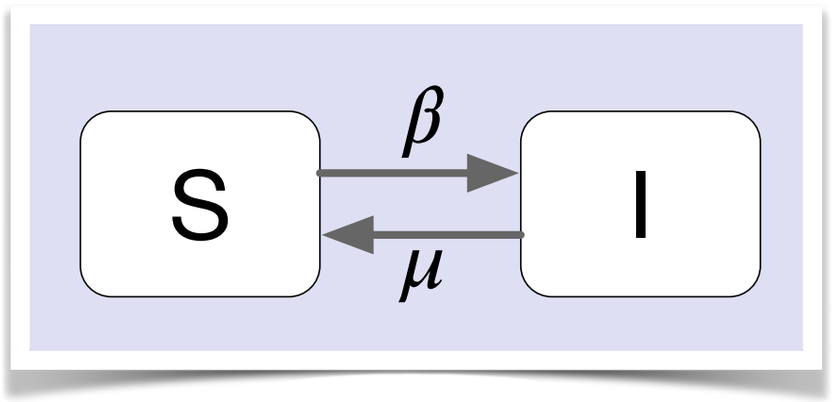
$s(t)$	Fraction of individuals in Susceptible state at time t
$i(t)$	Fraction of individuals in Infected state at time t
$r(t)$	Fraction of individuals in Recovered state at time t
i_0	Initial($t = 0$) fraction of infected individuals

Spreading processes

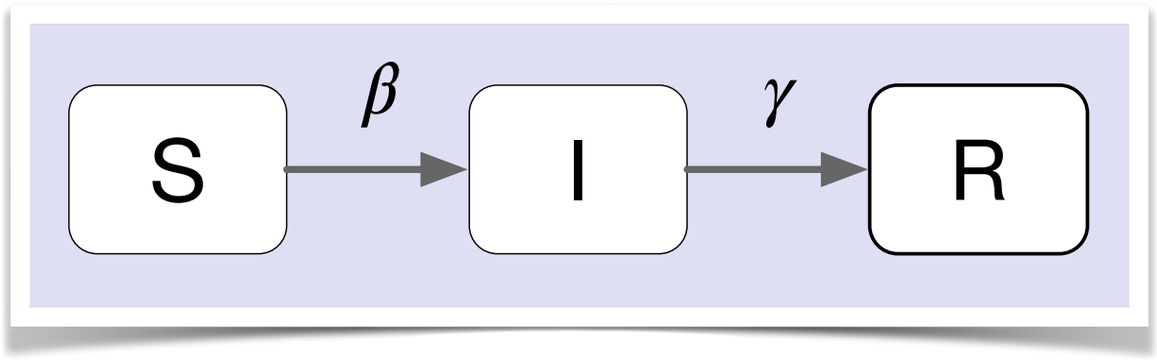
SI



SIS



SIR



Homogeneous mixing

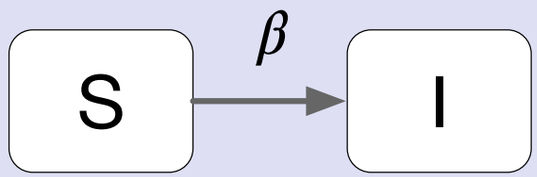
Non-network approach

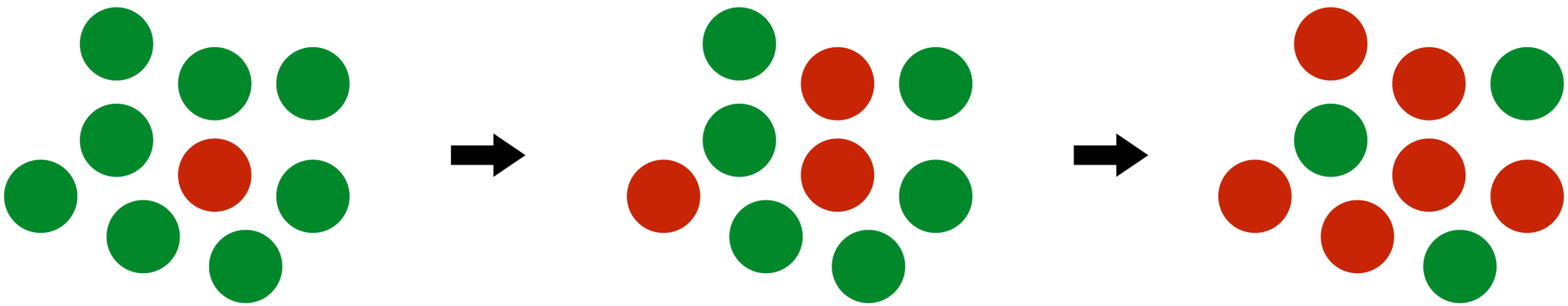
- Any individual can interact with any other
- The population has a finite size
- Individuals have an average number of contacts per unit of time

Kermack, W. O., & McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, *115*(772), 700-721.

SI model

τ	**Infectivity**: probability that the contact between an *Infected* individual and a *Susceptible* one results in the infection of the Susceptible.
$\hat{c}$	*Contact rate*: average number of contact per person per time
β	**Effective contact rate**, $\beta = \tau\hat{c}$, number of newly infected individuals by each infected individual in a population in which everyone else is susceptible.





The SI model

SI - characteristics

Each of the i infected individuals infects in average β contacts, but only $s = (1 - i)$ of its contacts are indeed susceptible. More formally using differential equations:

$\frac{di}{dt}$	**Rate of new infection**: $\frac{di}{dt} = \beta i s = \beta(1 - i)i$
$i(t)$	**Infected fraction**[a]: $i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$
$s(t)$	**Susceptible fraction**: $1 - i(t)$

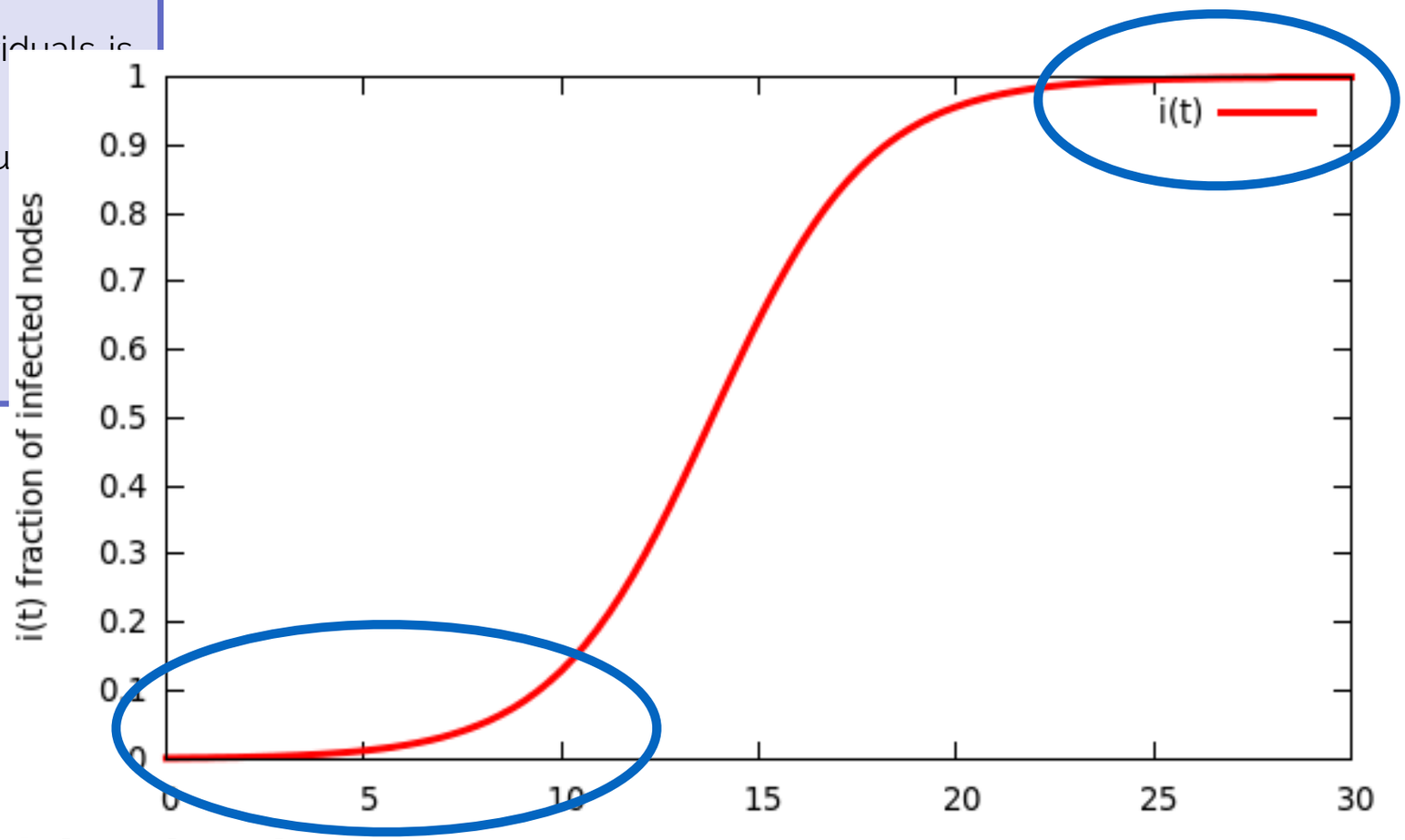
The process can be separated in three steps:

- At first, the fraction of infected individuals **Grows exponentially** until a large fraction of the population is infected. (i is small, $\frac{di}{dt} \approx \beta i \Rightarrow$ exponential)
- Due to **saturation**, the infection of the last individuals is slow
- The growth is faster and faster until half the popu infected ($\text{argmax}_{x,y}(x(1 - x)) : x = y = 0.5$).

If $\beta > 0$, everyone is infected at the end of the process.

[a] Barrat, Barthelemy, and Vespignani 2008.

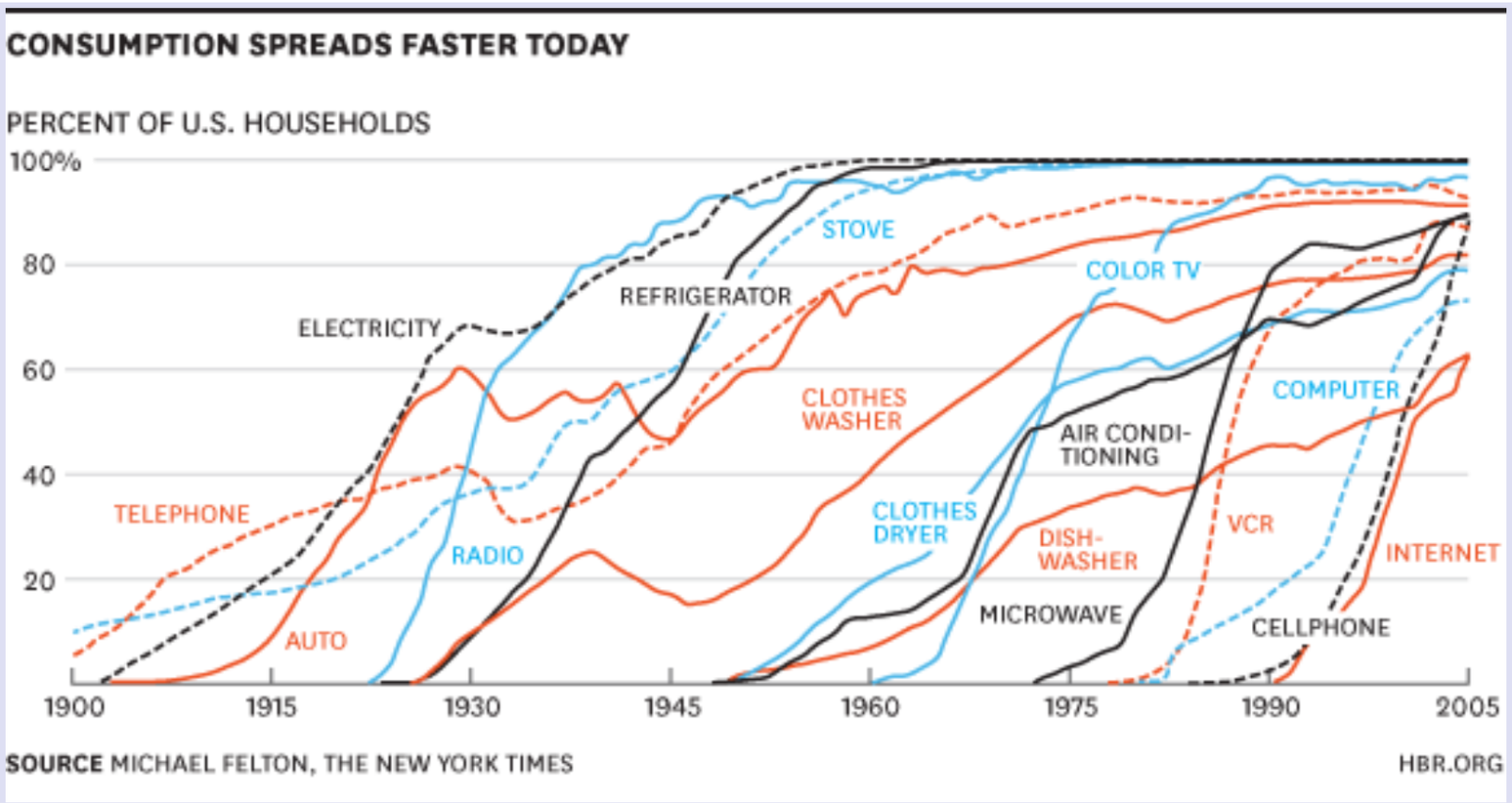
Saturation



Exponential outbreak

The SI model

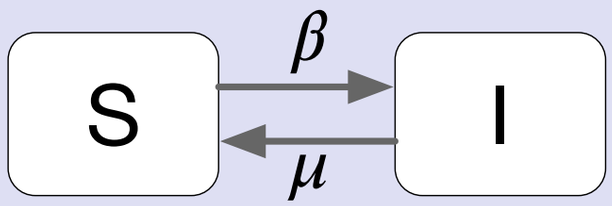
Example: technology adoption



The SIS model

Additionally to β, the SIS model requires another parameter:

μ	**recovery rate**: probability that an *Infected* individual go back to the susceptible state per unit of time.



The SIS model

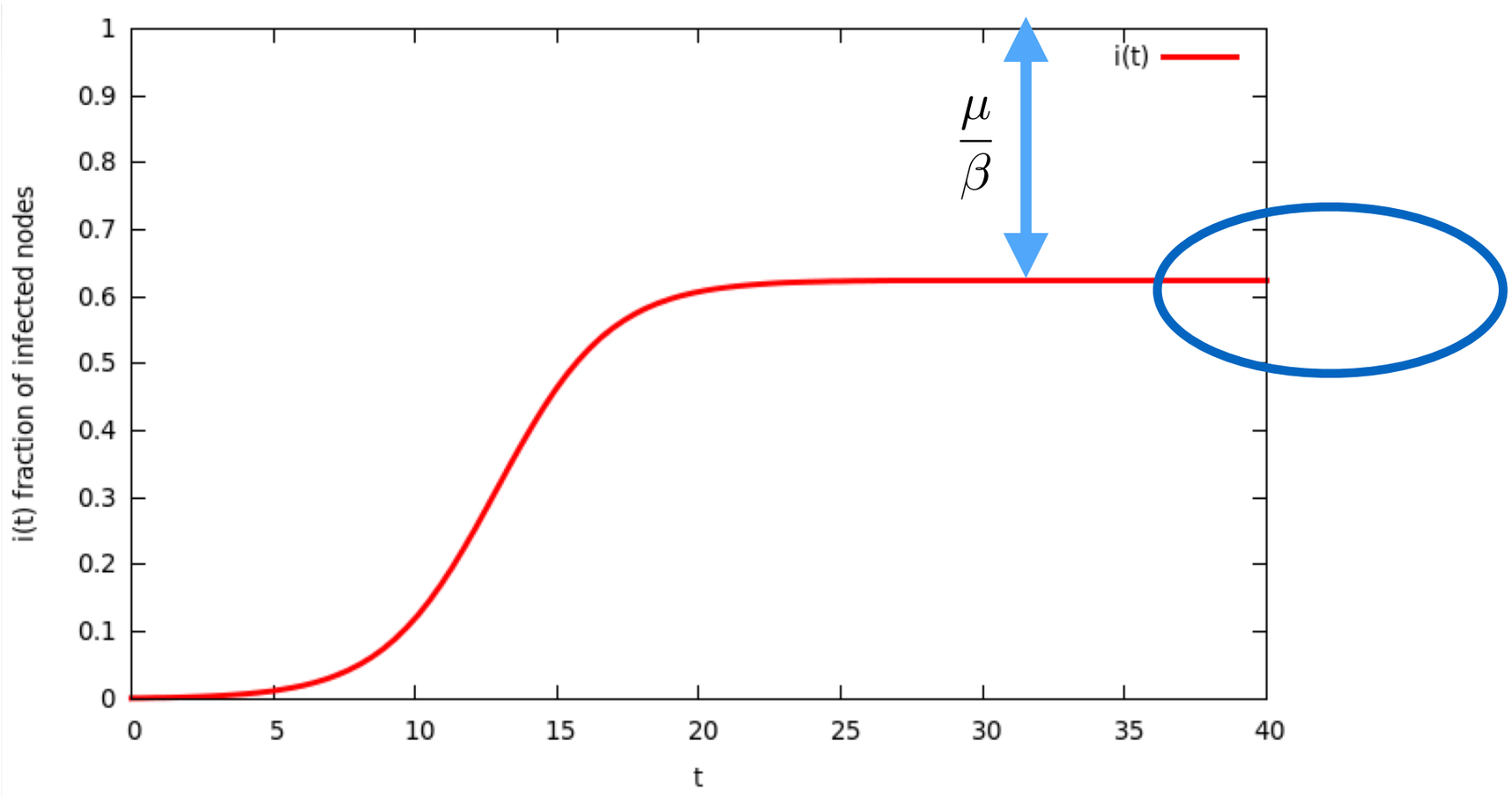
SIS - characteristics

Intuitively, the fraction of infected individuals is now reduced by those switching to the susceptible state, more formally:

$\frac{di}{dt}$	**Rate of new infection**: $\beta i(1-i) - \mu i = i(\beta - \mu - \beta i)$
$i(t)$	**Infected fraction**[a]: $\left(1 - \frac{\mu}{\beta}\right) \frac{Ce^{(\beta-\mu)t}}{1+Ce^{\beta-\mu)t}}$

For large times, $i(t) \to 1 - \frac{\mu}{\beta}$, i.e., the fraction of infected individuals stabilize around a value which depends only of parameters μ and β.

[a] Barrat, Barthelemy, and Vespignani 2008.



The SIS model

λ ratio or (R_0)

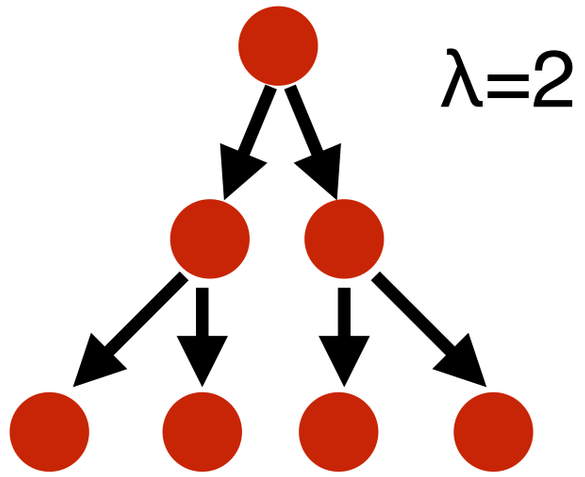
In the SIS model, an important notion is the λ ratio, also called R_0.

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

R_0 can be understood as the average number of individuals that will be infected by an infected individual, **in a population in which all other nodes are Susceptible**. R_0 is a property of the model and **do not change with time**.
Looking at the R_0 is important in the early stage of the epidemic:

- if $R_0 > 1$, **there will be an outbreak**
- if $R_0 < 1$, **the epidemic will disappear naturally**.

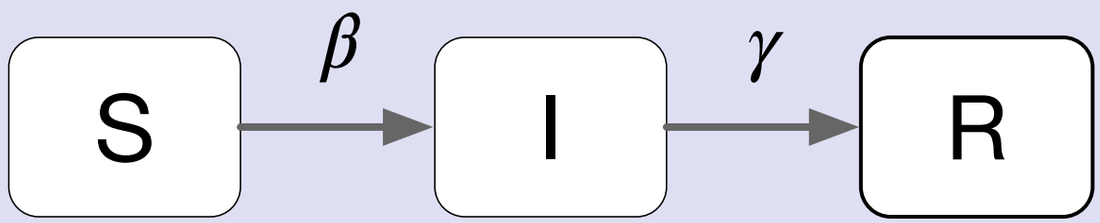
If R_0 is just above 1, the outbreak also can stop naturally by chance in the early stage.



The SIR model

Additionally to β, the SIR model requires another parameter:

γ	**recovery rate**: probability that an *Infected* individual switch to the Recovered state per unit of time.



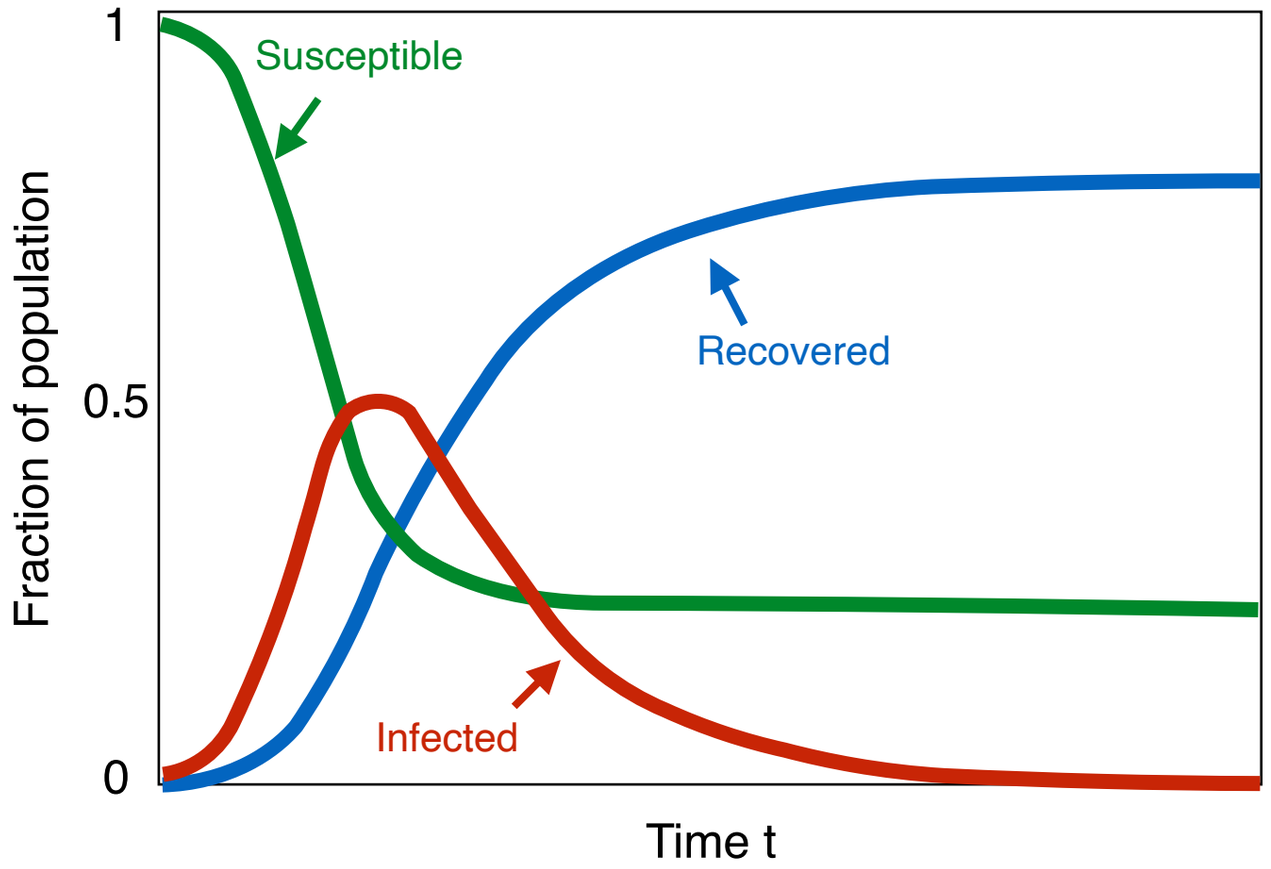
Spreading processes

SIR - characteristics

Intuitively, the fraction of infected individuals is now reduced by those switching to the recoved state, more formally:

$$\frac{ds}{dt} = -\beta is, \frac{di}{dt} = \beta is - \gamma i, \frac{dr}{dt} = \gamma i$$

- The initial steps of the outbreak still follow an exponential growth
- The fraction of infected nodes reach a peak and then decreases
- The fraction of recovered saturates below 1
- The fraction of susceptible do not necessarily reach 0
- The λ ratio is defined as $\lambda = \frac{\beta}{\gamma}$



Spreading processes

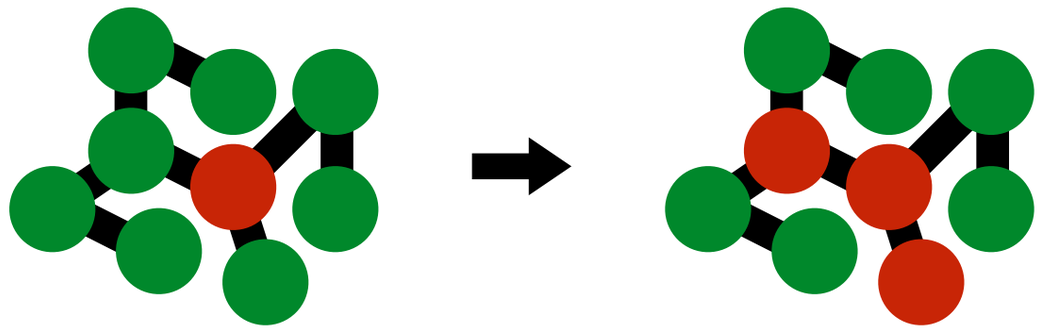
Many other models exist:
SIRD, MSIR, SEIR
SEIS, MSEIRS
Variable contact rate
Voter
Majority rule
Etc.

Check for instance:
https://ndlib.readthedocs.io/en/latest/reference/reference.html#diffusion-models

Spreading on Networks

Epidemic spreading on networks

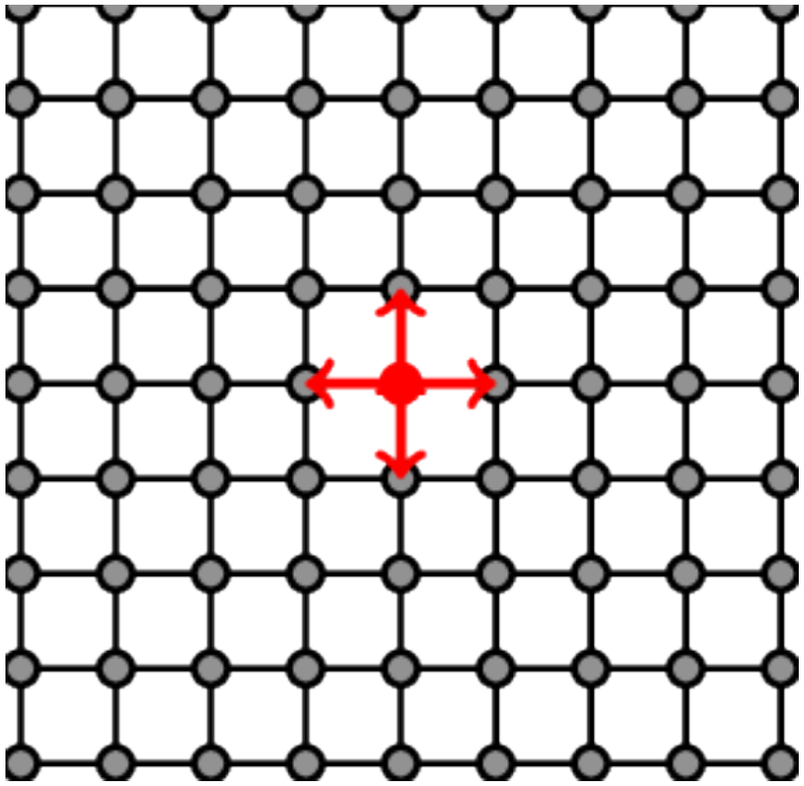
The homogeneous mixing approach is clearly unrealistic: interactions are organized in networks

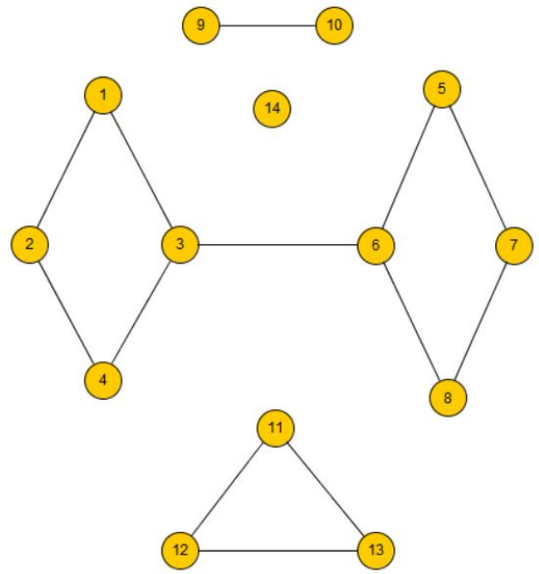


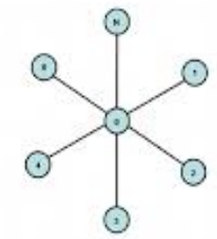
How much does it affect spreading?

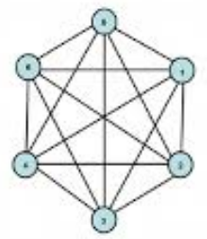
$\hat{c}$ becomes useless: $\beta = \tau$

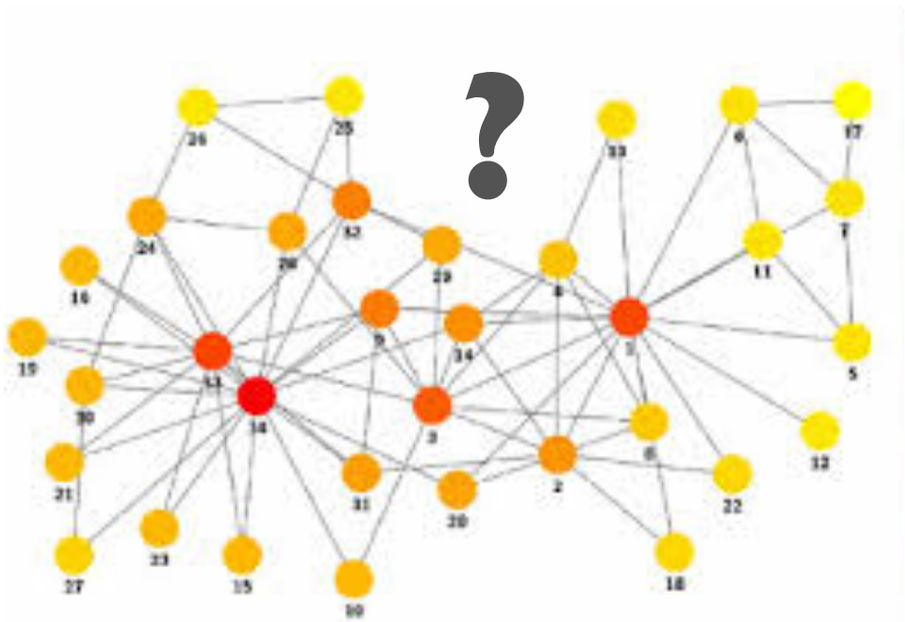
Epidemic spreading on networks











Homogeneous networks

Homogeneous Mixing

$\frac{di}{dt}$	**Rate of new infection**: $\frac{di}{dt} = \beta i s = \beta(1-i)i$

Homogeneous Networks

If we consider an **homogeneous random network** in which all nodes have degree exactly k, then we can consider the spreading on this network as similar to the non-network models, with $\hat{c} = k$. For instance, the SI model becomes:

$$\frac{di}{dt} = \beta\langle k\rangle(1-i)i$$

Note that in practice, there are a few differences, such as a switch from a continuous to a discrete setting, but they have no consequences on large graphs.

ER random graph =>approximation still holds,

$(k \approx \langle k\rangle)$

Epidemic spreading on heterogeneous networks

- In degree heterogeneous networks the $k \simeq \langle k \rangle$ approximation does not hold

- Solution: Degree Block Approximation
  - Assumption: all nodes with the same degree are statistically equivalent
  - Look for infection/susceptible node densities in the degree groups

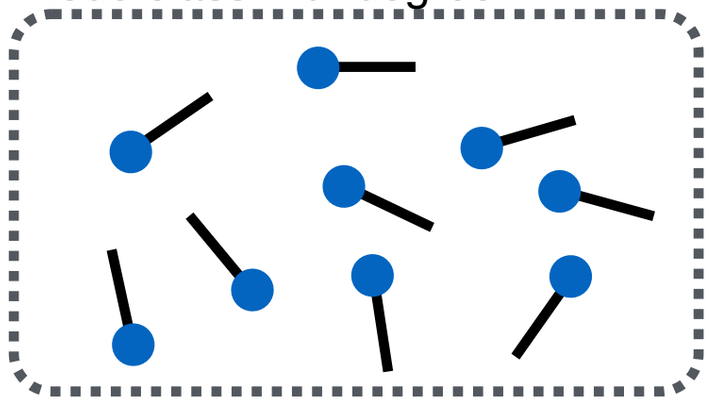
$$i_k$$

$$s_k$$

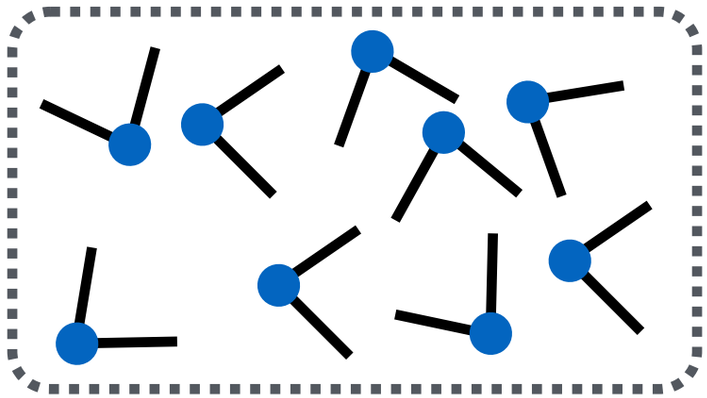
- Calculate the global average by a sum considering the degree distribution

$$i = \sum_k P(k) i_k \qquad s = \sum_k P(k) s_k$$

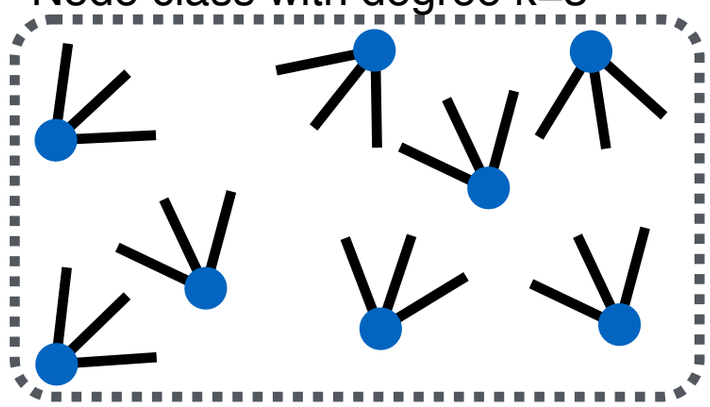
Node class with degree k=1



Node class with degree k=2



Node class with degree k=3



Epidemic spreading on heterogeneous networks

Homogeneous Networks

$$\frac{di}{dt} = \beta\langle k\rangle(1-i)i$$

Heterogeneous Degrees - SI

For the SI model, we know that all nodes are infected in the end, but what may vary is **speed** of the process.
The speed of diffusion by degree block can be expressed as:

$$\frac{di_k}{dt} = \beta k(1-i_k)\Theta_k$$

with Θ_k being the fraction of infected neighbors of a node with degree k.

Epidemic spreading on heterogeneous networks

- Due to the friendship paradox, nodes are more likely to be connected to large nodes than to small ones
- In real networks, we have seen that there is often a degree assortativity, thus nodes of a given degree have different degrees in their neighborhood.

$$\Theta_k = \sum_{k'} P(k'|k) i_{k'}$$

- Assume: no degree-degree correlations in the network

$$P(k'|k) = \frac{k' P(k')}{\sum_{k''} k'' P(k'')} = \frac{k' P(k')}{\langle k \rangle}$$

And:

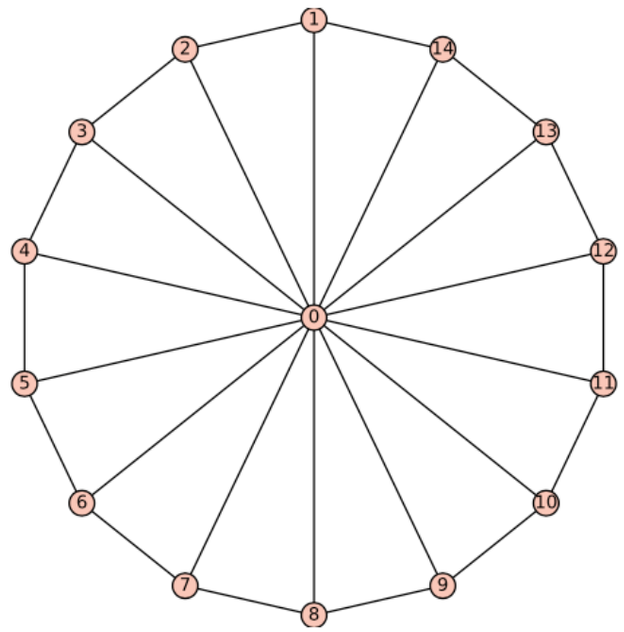
$$\Theta_k = \Theta = \frac{\sum_{k'} k' P(k') i'_k}{\langle k \rangle}$$

SI process on heterogeneous networks

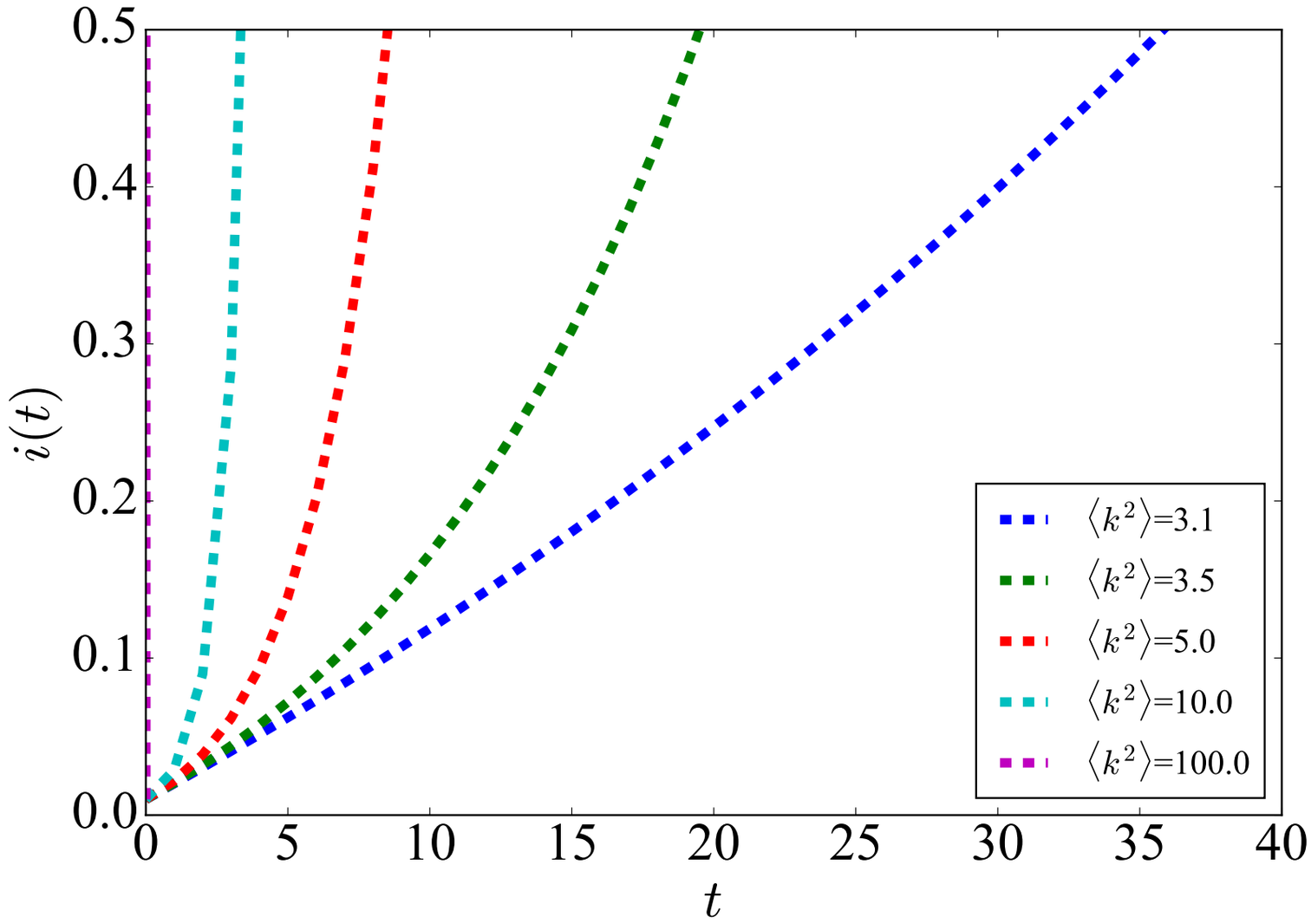
Heterogeneous Degrees - SI - time scale

From previous equations, it can be shown[a] that the **time scale** τ of the process, i.e., a measure inversely proportional to its speed, is $\tau = \frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)}$.
Thus, for a given average degree $\langle k \rangle$ and a given β, **the more heterogeneous the degrees, the faster the diffusion**.

If the degree distribution follows a power law of exponent $\alpha < 3$, we have seen that $\langle k^2 \rangle$ diverge towards infinity, thus τ tends toward 0, thus the diffusion is nearly instantaneous.



SI process on heterogeneous networks



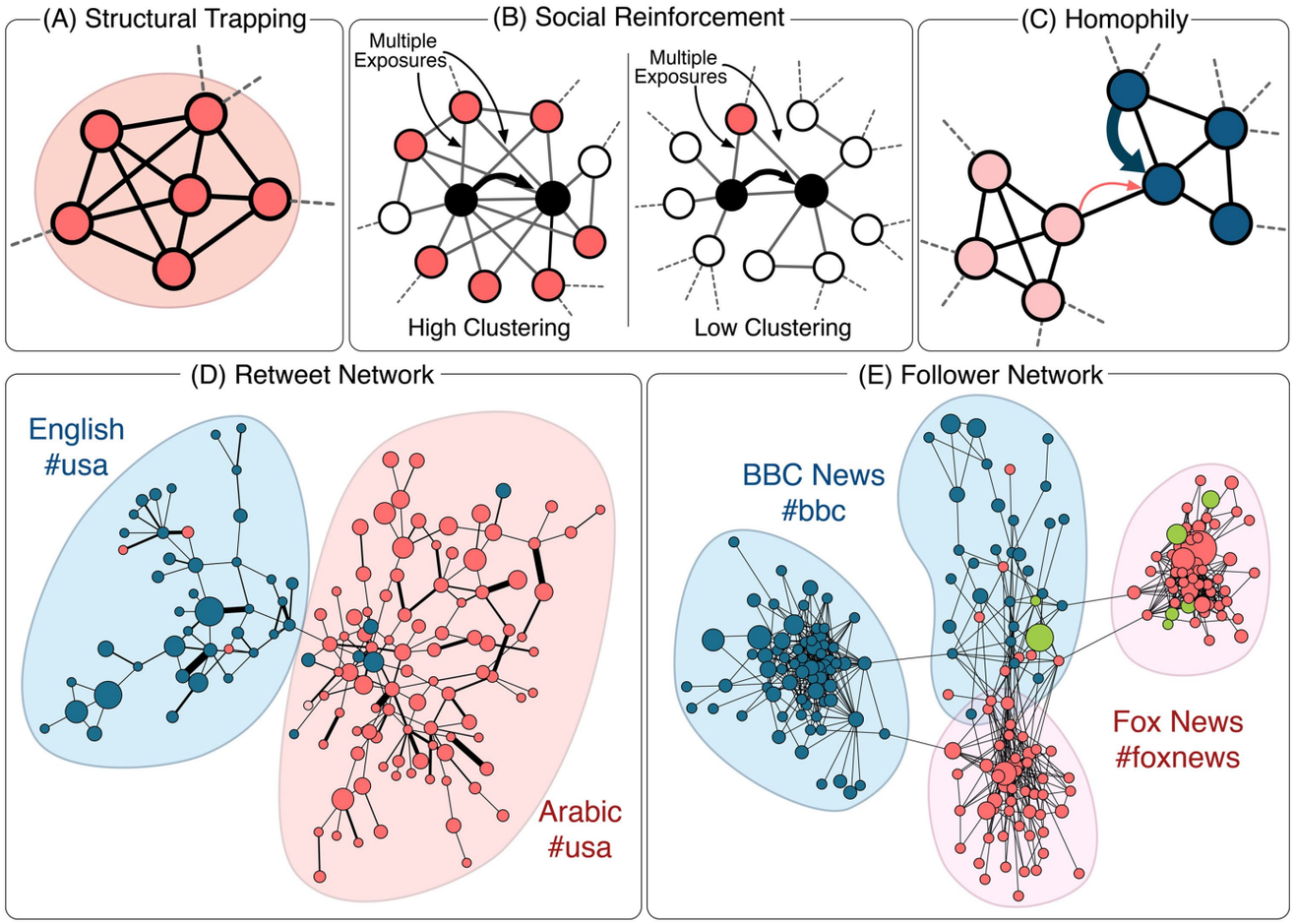
SIS process on heterogeneous networks

Heterogeneous Degrees - λ

For SIS and SIR models, it can also be shown[a] that the epidemic threshold λ (or R_0) is not reached when $\lambda = \frac{\beta\langle k\rangle}{\mu} > 1$ as in homogeneous networks, but when $\lambda > \frac{\langle k\rangle}{\langle k^2\rangle}$.

This means that in a very heterogeneous network, **an outbreak can start even if λ is very small, and below 1**. Intuitively, even if

Community structure and spreading



Example: Opinion diffusion

(Competing diffusion processes)

(A) ***Structural trapping*****:** dense communities with few outgoing links naturally trap information flow. (B) ***Social reinforcement***: people who have adopted a meme (black nodes) trigger multiple exposures to others (red nodes). In the presence of high clustering, any additional adoption is likely to produce more multiple exposures than in the case of low clustering, inducing cascades of additional adoptions. (C) ***Homophily*****:** people in the same community (same color nodes) are more likely to be similar and to adopt the same ideas.

Stegehuis, C., Van Der Hofstad, R., & Van Leeuwaarden, J. S. (2016). Epidemic spreading on complex networks with community structures. *Scientific reports*, *6*, 29748.

Experiments

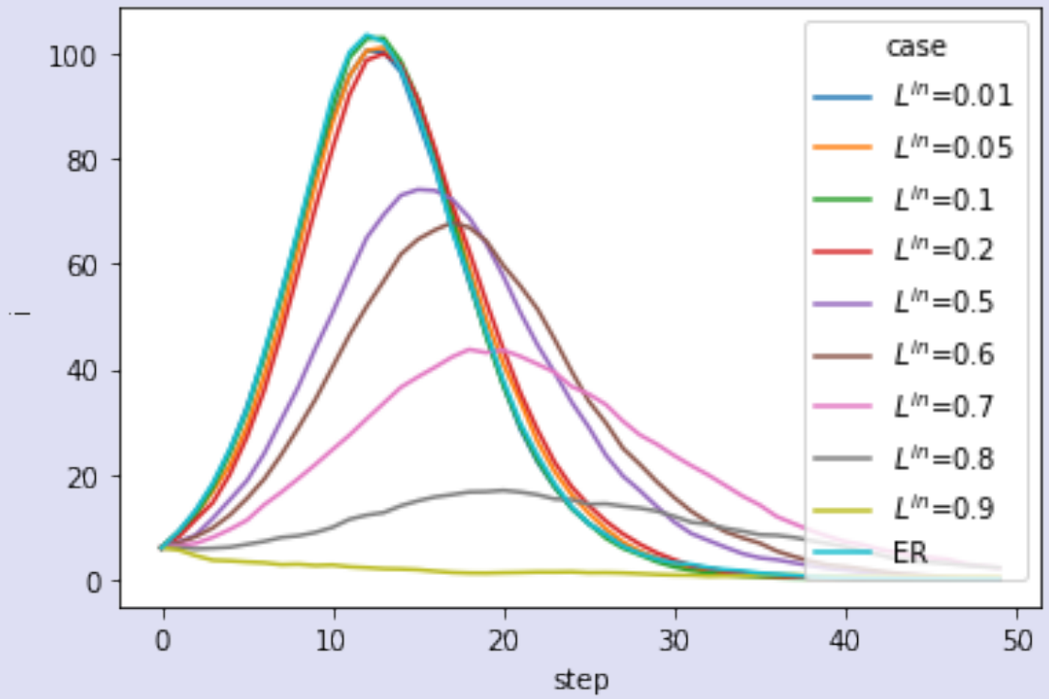
SIR - Community Structure

In this experiment, we compare an ER network to Stochastic Block Models.

Network parameters: $n = 1000, \langle k \rangle = 5$.

SBM parameters Number of blocks $|C| = 100$. We vary L^{in}, the fraction of all edges that are inside blocks. When $L^{in} = 0.01, p^{in} \approx p^{out} = 0.005$. When $L^{in} = 0.9, p^{in} = 0.5, p^{out} \approx 0.0005$

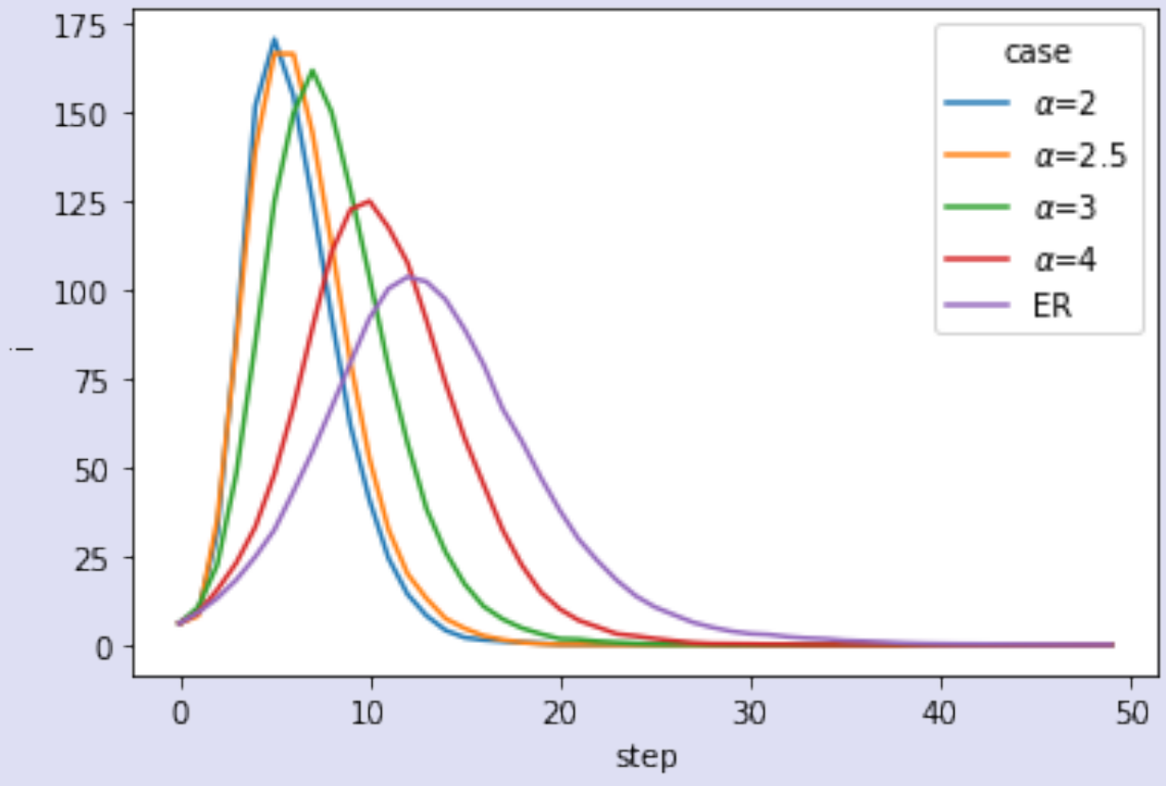
SIR parameters: $\theta = 0.2$,$\gamma = 0.5$. The initial number of infected nodes is 5, all of them in the same community structure.



We observe that the more marked the communities, the less efficient the spreading process.

SIR - Scale Free

In this experiment, we compare an ER network to Configuration Models with power law degree distributions.
Network parameters:$n = 1000, \langle k \rangle = 5$. We vary the exponent of the distribution, while keeping $\langle k \rangle = 5$ constant.
SIR parameters: $\theta = 0.2$,$\gamma = 0.5$. The initial number of infected nodes is 5, all of them in the same community structure.



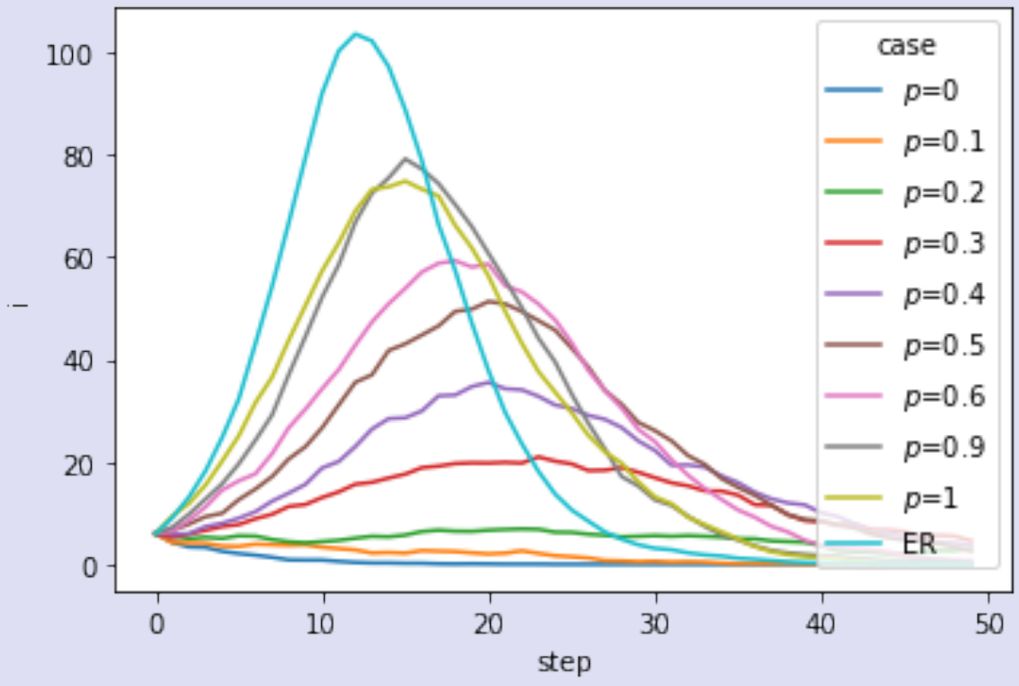
The highest the exponent of the degree distribution, the faster is the diffusion.

SIR - Spatial effect - WS

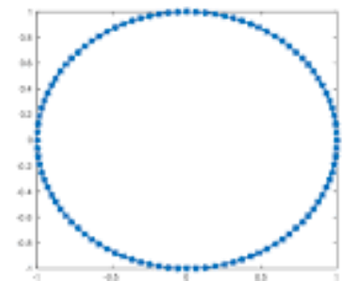
In this experiment, we compare an ER network to Watts Strogatz random graphs, varying the probability of rewiring edges. It can be understood as a model of spatial proximity: with $p = 0$, each node is connected only to its direct neighbors in the 1 dimensional space. If $p = 1$, each node is connected to exactly k random nodes.

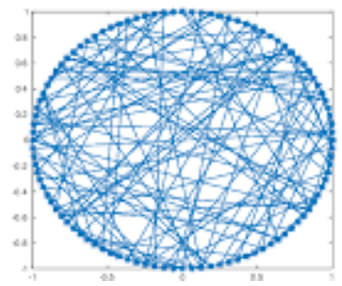
Network parameters:$n = 1000, \langle k \rangle = 5$

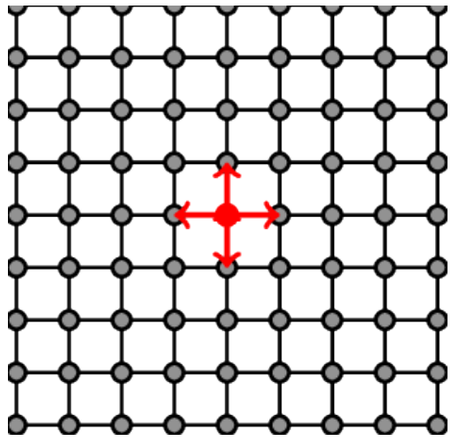
SIR parameters: $\theta = 0.2$,$\gamma = 0.5$. The initial number of infected nodes is 5, being 5 direct neighbors.



The more nodes tend to be connected to direct neighbors in space, the slower the diffusion.







Applications

Applications

- Model fitting (to better know an observed diffusion)
- Predicting future trends
- Epidemic control
  - Vaccine, etc. => Which nodes/edges to target?

- Example of strategy: friend paradox
  - Vaccine contacts of random nodes instead of random nodes

[a] Cohen, Havlin, and Ben-Avraham 2003.

Recent example

Preprint

Mobility network models of COVID-19 explain inequities and inform reopening

Chang, S., Pierson, E., Koh, P. W., Gerardin, J., Redbird, B., Grusky, D., & Leskovec, J. (2020). Mobility network models of COVID-19 explain inequities and inform reopening. *Nature*, 1-8.

Serina Chang[1,9], Emma Pierson[1,2,9], Pang Wei Koh[1,9], Jaline Gerardin[3], Beth Redbird[4,5], David Grusky[6,7] & Jure Leskovec[1,8] ✉

[1]Department of Computer Science, Stanford University, Stanford, CA, 94305, USA. [2]Microsoft Research, Cambridge, MA, 02142, USA. [3]Department of Preventive Medicine, Northwestern University, Chicago, IL, 60611, USA. [4]Department of Sociology, Northwestern University, Evanston, IL, 60208, USA. [5]Institute for Policy Research, Northwestern University, Evanston, IL, 60208, USA. [6]Department of Sociology, Stanford University, Stanford, CA, 94305, USA. [7]Center on Poverty and Inequality, Stanford University, Stanford, CA, 94305, USA. [8]Chan Zuckerberg Biohub, San Francisco, CA, 94158, USA. [9]These authors contributed equally: Serina Chang, Emma Pierson, Pang Wei Koh. ✉e-mail: jure@cs.stanford.edu

Mobility network models of COVID-19 explain inequities and inform reopening

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Applications

Data: SafeGraph, a company that aggregates anonymized location data from mobile applications

Period: **March 1**- **May 2** - 2020

Time scale: Hourly movements

Applications

Data type: **CBG** -> **POI**

CBG: Census Block Group (small Area, 600-3000 people)

POI: Point of Interest

Access to location and type

- Restaurant
- Grocery store
- Religious Establishment
- etc.

Applications

Population: 98 Million people (anonymized)

CBG: 59,945

POI: 552,758

Hourly Edges: 5.4 Billion

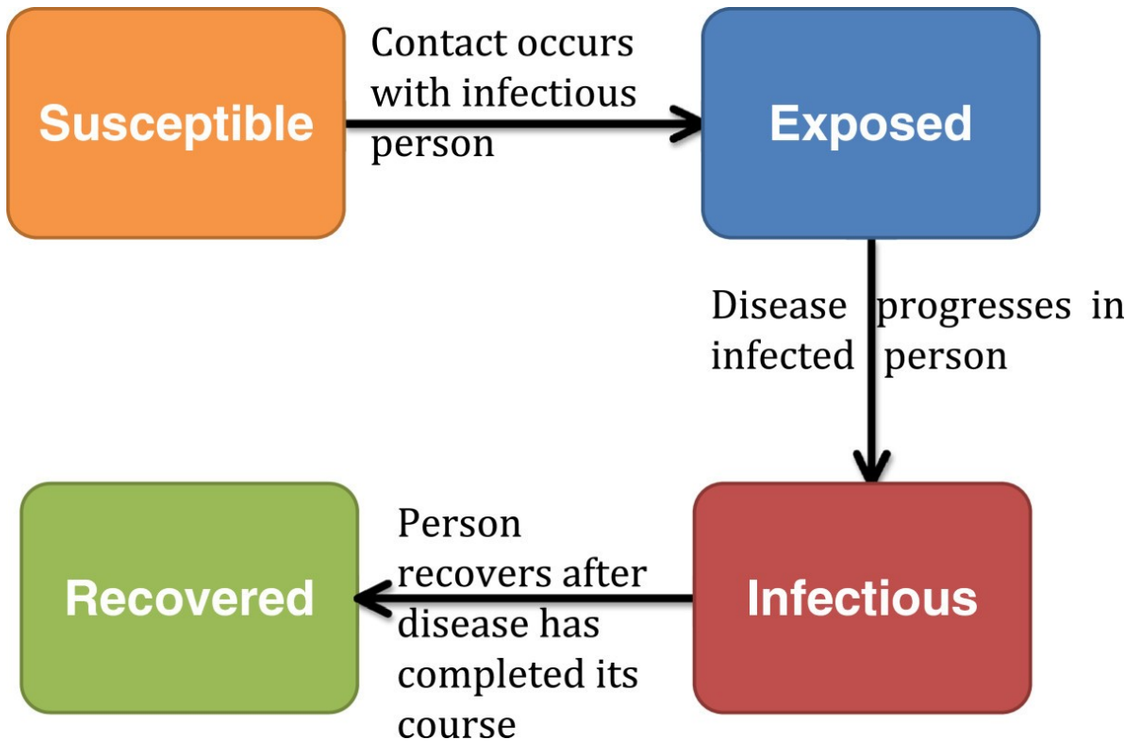
Split my metropolitan Erea

Chicago, New York, etc.

Each an independent model

Applications

Model: SEIR



Exposed:
Infected but not aware and not contagious

Applications

Model: SEIR

$$N^{(t)}_{S_{c_i}\to E_{c_i}} \sim \text{Pois}\left(\frac{S^{(t)}_{c_i}}{N_{c_i}} \sum_{j=1}^{n} \lambda^{(t)}_{p_j} w^{(t)}_{ij}\right) + \text{Binom}(S^{(t)}_{c_i}, \lambda^{(t)}_{c_i}) \quad (1)$$

$$N^{(t)}_{E_{c_i}\to I_{c_i}} \sim \text{Binom}(E^{(t)}_{c_i}, 1/\delta_E) \quad (2)$$

$$N^{(t)}_{I_{c_i}\to R_{c_i}} \sim \text{Binom}(I^{(t)}_{c_i}, 1/\delta_I), \quad (3)$$

where $\lambda^{(t)}_{p_j}$ is the rate of infection at POI p_j at time t; $w^{(t)}_{ij}$, the ij-th entry of the visit matrix from the mobility network (Methods M2), is the number of visitors from CBG c_i to POI p_j at time t; $\lambda^{(t)}_{c_i}$ is the base rate of infection that is independent of visiting POIs; δ_E is the mean latency period; and δ_I is the mean infectious period.

Param.	Description	Value (Source)
δ_E	mean latency period	96 hours[24,63]
δ_I	mean infectious period	84 hours[24]

Applications

For a particular POI:

Infection rate at *t* depends on:

-**Fraction of infected visitors** at *t*

-**Transmission rate** β

-**Transmission rate** β at *t* depends on:

-**Size** of the POI

-**Number of visitors** at *t*

-Shared **learnt parameter**

We model the infection rate at POI p_j at time t, $\lambda_{p_j}^{(t)} := \beta_{p_j}^{(t)} \cdot I_{p_j}^{(t)} / V_{p_j}^{(t)}$, as the product of its transmission rate $\beta_{p_j}^{(t)}$ and proportion of infectious individuals $I_{p_j}^{(t)} / V_{p_j}^{(t)}$, where $V_{p_j}^{(t)} := \sum_{i=1}^{m} w_{ij}^{(t)}$ is the total number of visitors to p_j at time t. We model the transmission rate at POI p_j at time t as

$$\beta_{p_j}^{(t)} := \psi \cdot d_{p_j}^2 \cdot \frac{V_{p_j}^{(t)}}{a_{p_j}}, \tag{8}$$

where a_{p_j} is the physical area of p_j, and ψ is a transmission constant (shared across all POIs) that we fit to data. The inverse scaling of transmission rate with area a_{p_j} is a standard simplifying assumption[57]. The

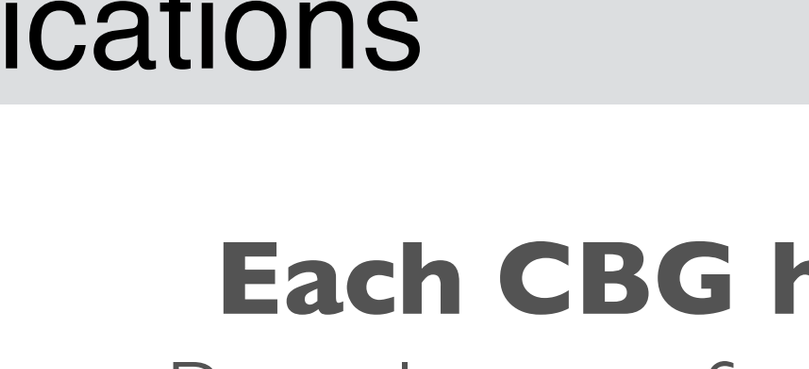
Applications

Each CBG has its own model

Prevalence of each state varies by SBG,
c.f. degree blocks

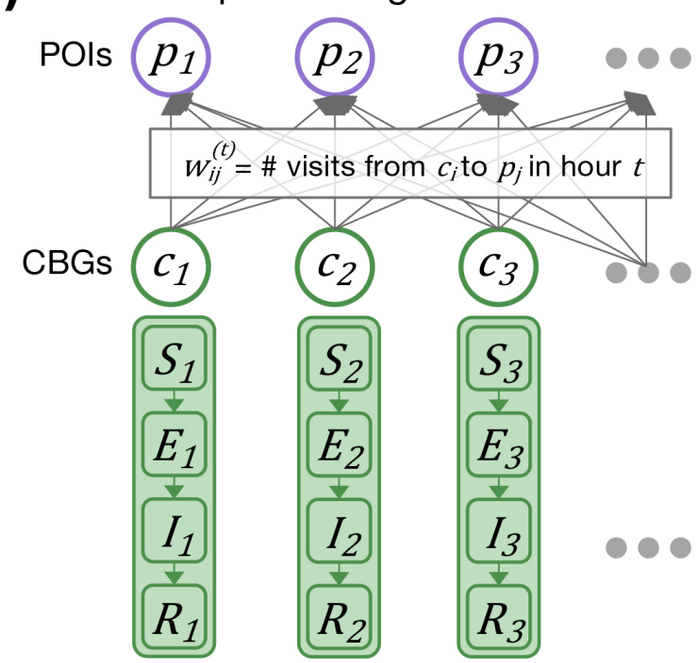
(a) Mobility networks in Chicago metro area

March 2, 2020 (Monday), 1pm



April 6, 2020 (Monday), 1pm

(b) Epidemiological model



Applications

Only 3 parameters to learn, constant over time:

-Scaling in POI (size*visitors*?) (proxy β)

-CBG transmission rate (β)

-Initial proportion of exposed individuals

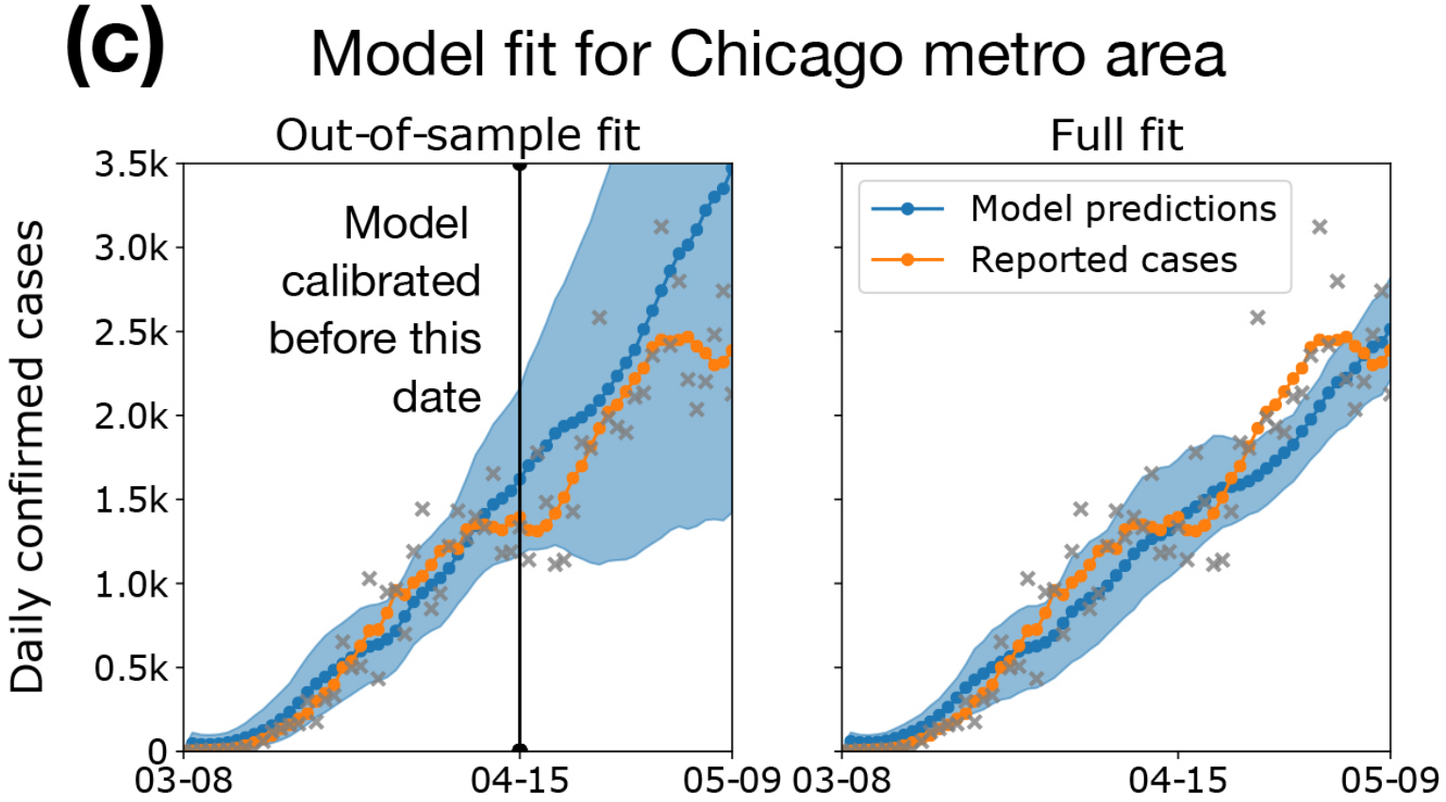
Fit using RMSE

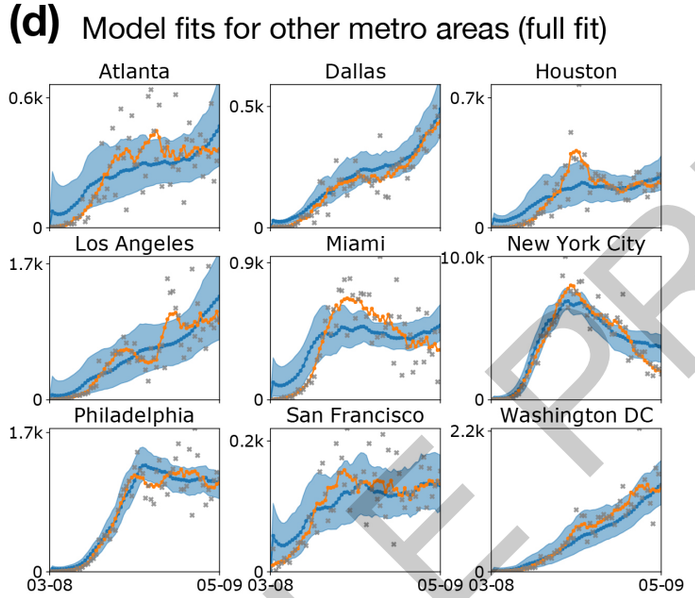
(minimizing the root of the mean square error)

=>Run various simulations with different sets of parameters to find those generating a diffusion as close as possible from observed data.

Applications

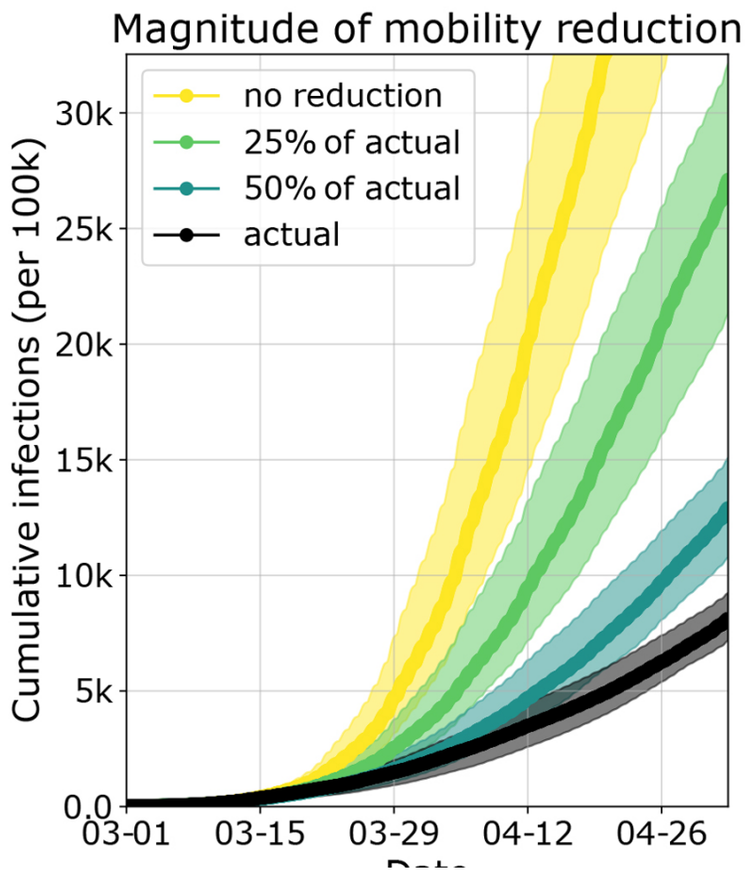
Prediction of future trends

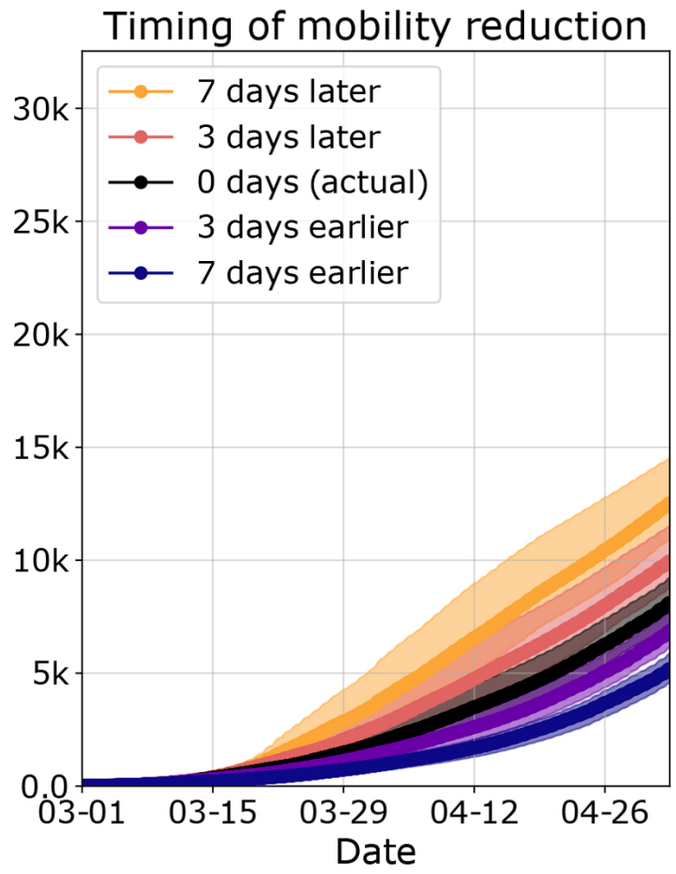


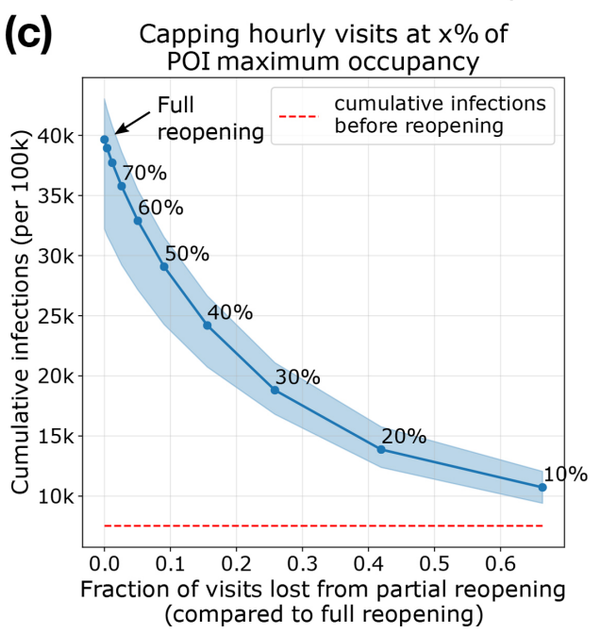


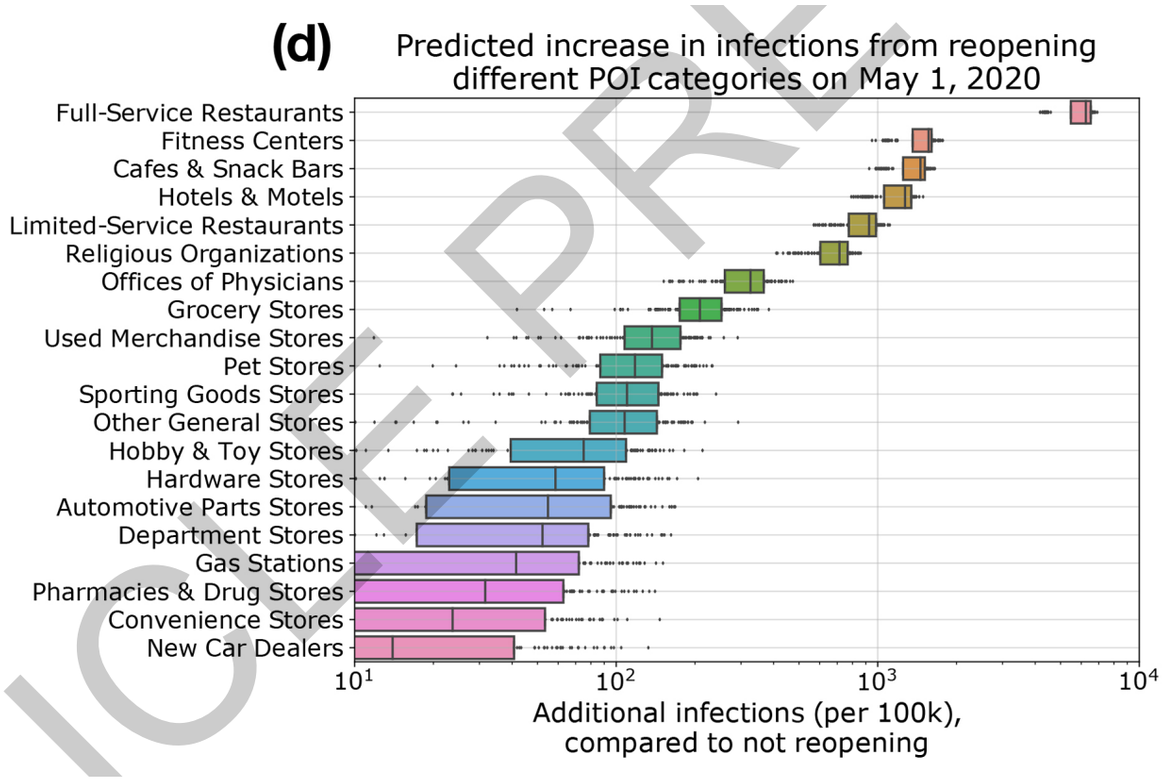
Applications

What if Scenarios





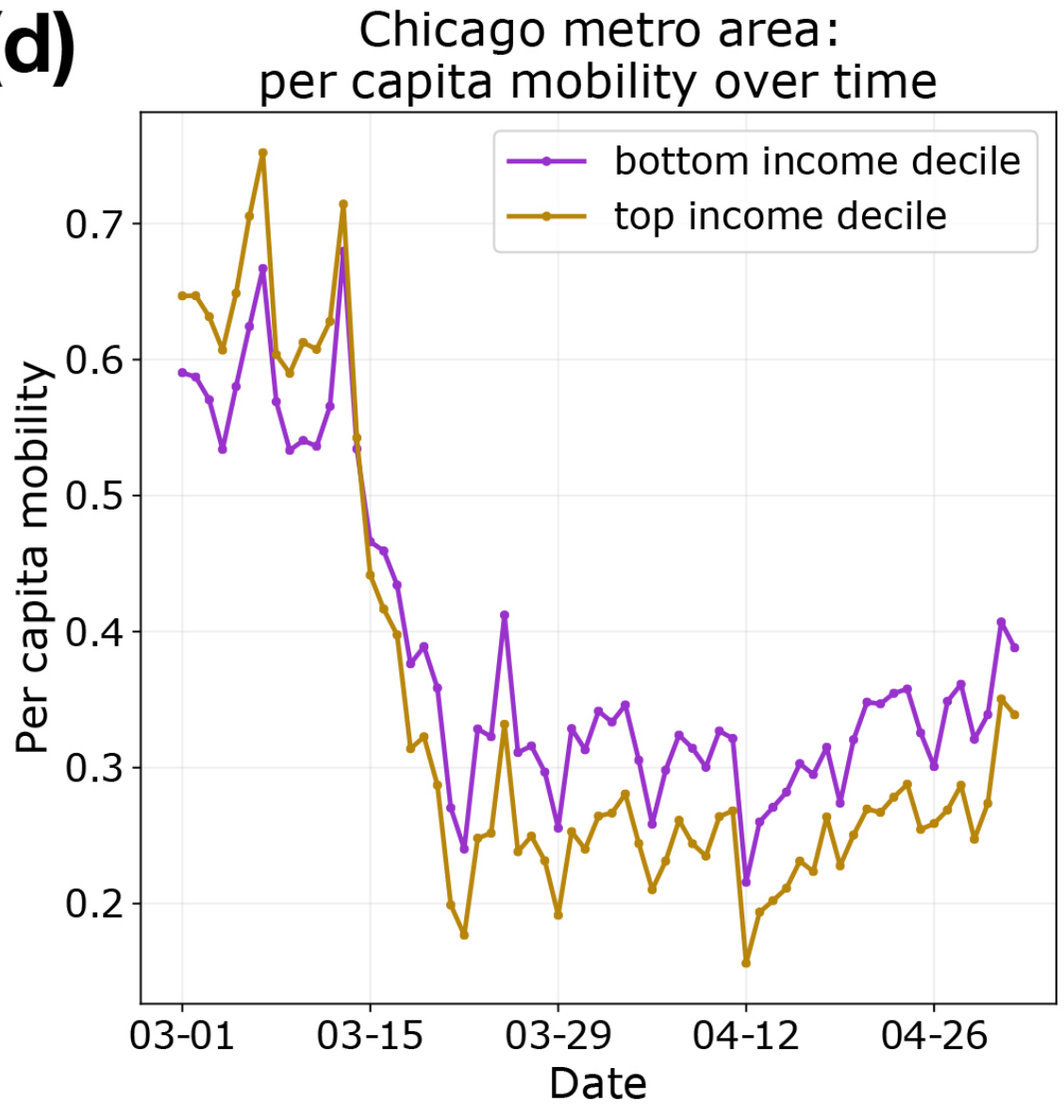




Applications

Explaining social phenomenon:
Bottom income population
more affected than
top Income population

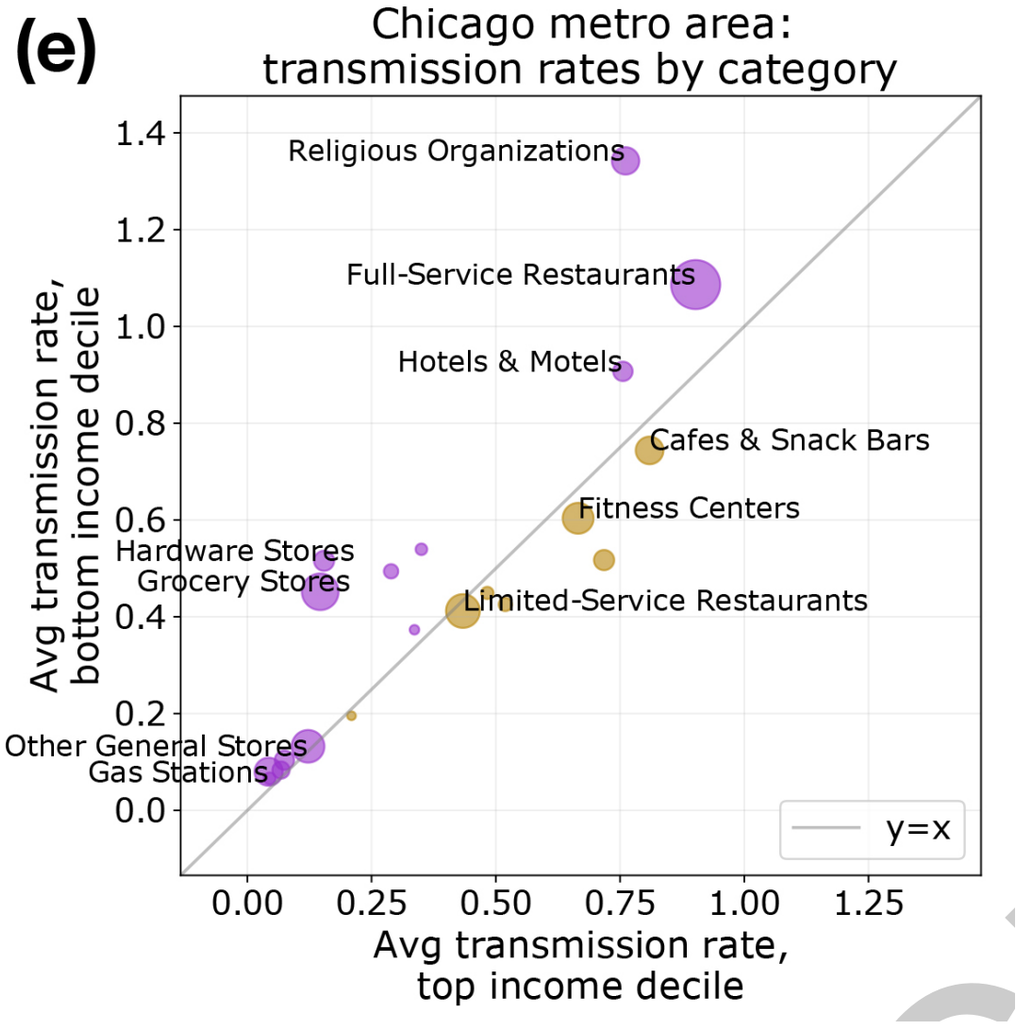
(d)



Mobility is less reduced by lockdown
=>More essential workers

Applications

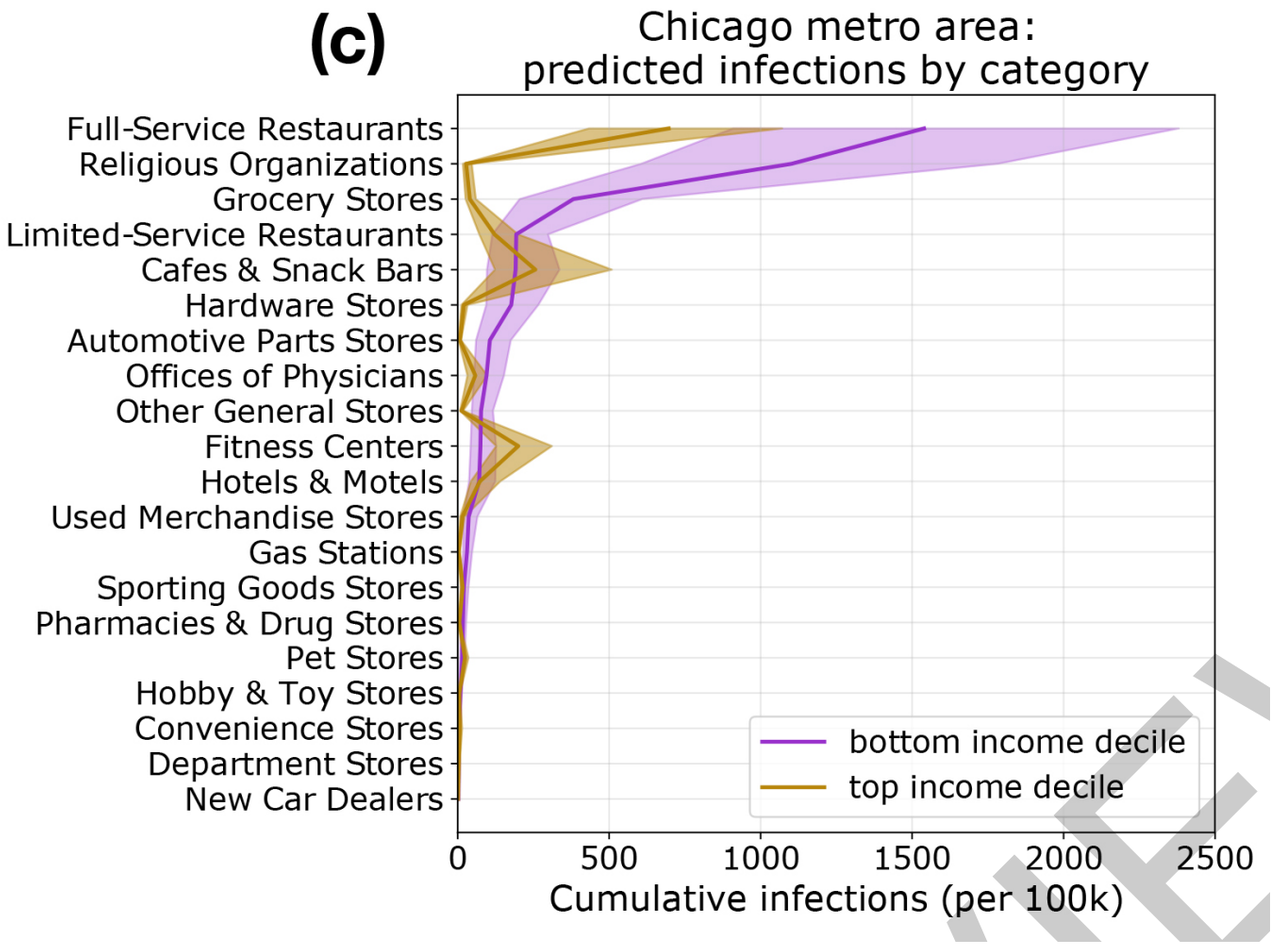
Explaining social phenomenon:
Bottom income population
more affected than
top Income population



More risks in essential stores,
notably due to smaller, more crowded local stores

Applications

Policies guidance?



Risks come from different
Sources for different populations