Life Data Epidemiology

Behavioural adaption to epidemics

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Introduction



- Human behavior plays an important role in the spread of infectious diseases
- Goal: Incorporate human behavior into disease models
- Individuals base their behaviour on **type** and **source** of information
- Assume effects on such behavior (fear/risk perception)

Source and type of information



Source:

• Global (govt, media)

Local (social neighborhood)

Type:

prevalence-based

belief-based

Behavioral changes



there is a multitude of behavioral changes that can influence the spread of infectious diseases

Two main classes of behavioral changes:

- change disease parameters (wearing of face masks or practice of better hygiene)
- change networks **topology** (reductions in the number of potentially infectious contacts)

Models considered



Change disease parameters:

- SIS with risk perception
- Multilayer SEIRD

Change networks **topology**:

- SIS on an adaptive network
- SIR on an adaptive network

SIS with risk perception



• The social network is not affected by the infection level (reasonable assumption for **mild diseases**, such as computer viruses)

 Assume the effects of the infection to be immediately visible (no latency and hidden infectivity)

• Immediate recovery ($\mu = 1$) from illness without immunization (SIS)

Risk perception model



Replace the bare infectivity rate β with $\tau = \beta^*I(s,k)$, where:

$$I(s,k) = \exp\left\{-\left[H + J\left(\frac{s}{k}\right)^{\alpha}\right]\right\}$$

- H: global influence over the population (H=0)
- s/k: fraction of infected individuals among the neighbors
- J: level of precaution measures adopted (linear response)
- α : special prophylaxis ($\alpha \le 1$) (non-linear response)

Networks

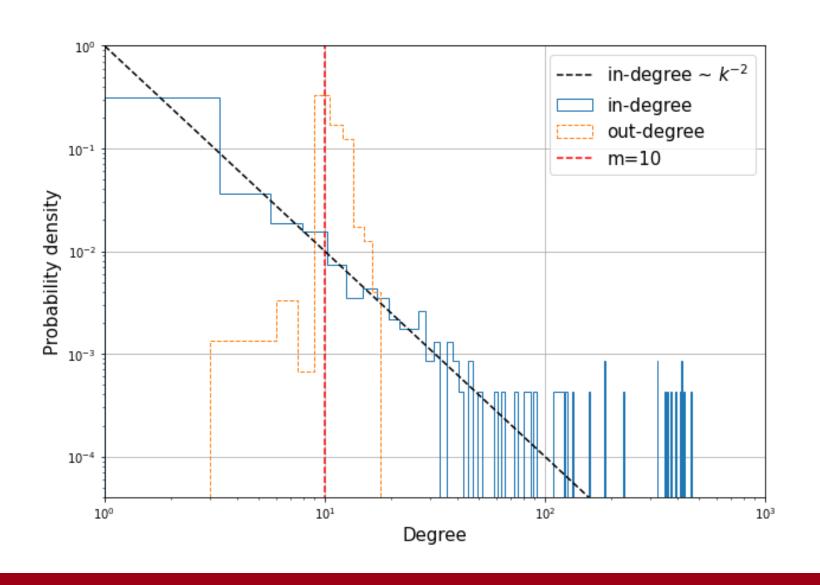


- Regular one-dimensional lattice (z = 10)
- Fully rewired lattice (Watts-Strogatz net., p=1, z=10)
- Erdős–Rényi (Poissonian degree distribution)
- Scale-free network

For each of them we considered N=1000 nodes.

Scale-free network





Mean-Field analysis



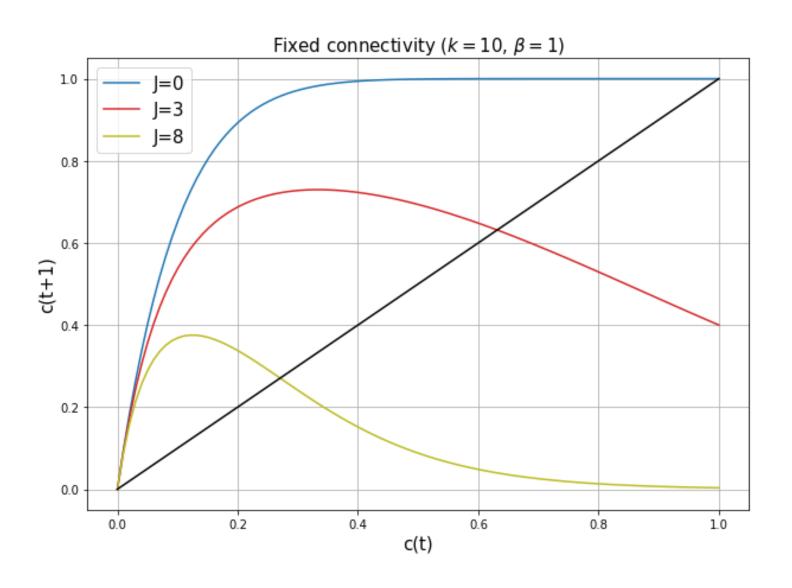
Assumption:

• Non-assortative network: $P_L(n|k) = \frac{nP(n)}{2}$

$$c_k' = 1 - \left(1 - \tau \sum_n c_n P_L(n|k)\right)^k$$

Mean-Field return map





Critical precaution level



$$c'_{k} = 1 - \left(1 - \tau \sum_{n} c_{n} P_{L}(n|k)\right)^{k} \qquad \stackrel{c \sim 0}{\longrightarrow} \qquad c'_{k} \simeq k\tau \sum_{n} c_{n} P(n|k)$$

$$\tau = \beta I(s, k)$$

$$P_L(n|k) = \frac{nP(n)}{z}$$

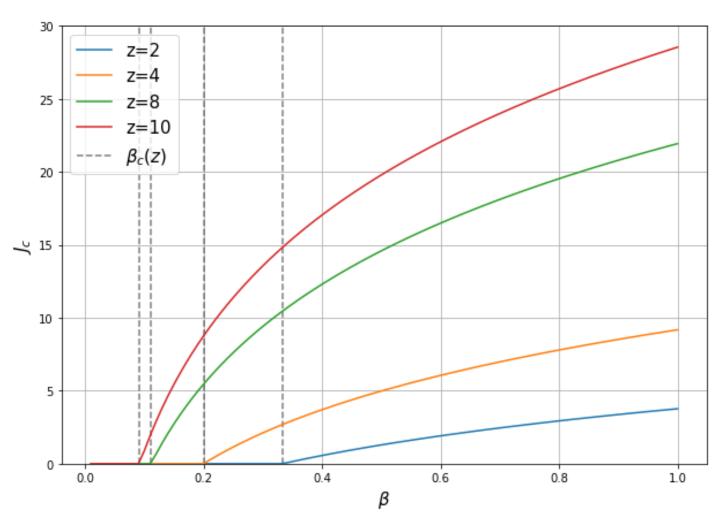
$$c_k(t+1) = c_k(t)\frac{\beta}{z} \sum_{n} \exp\left(-\frac{J}{n^{\alpha}}\right) n^2 P(n)$$

$$\sum_{k} \exp\left(-\frac{J_c}{k^{\alpha}}\right) k^2 P(k) = \frac{z}{\beta}$$

Critical precaution level



(scipy.optimize.fsolve)

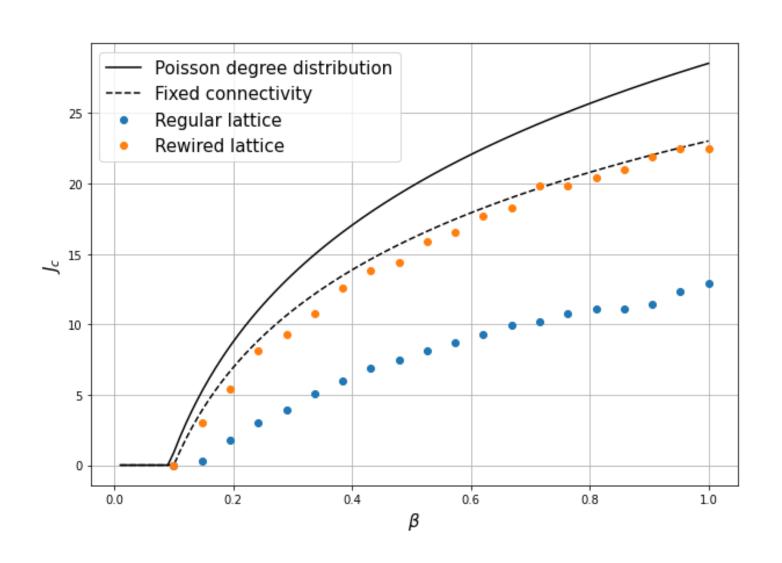


$$P(k) \simeq \frac{z^k e^{-z}}{k!}$$

$$\beta_c(z) = \frac{\langle k \rangle}{\langle k^2 \rangle} = \frac{z}{z + z^2}$$
 (J = 0)

Critical precaution level





$$\alpha = 1; z=10$$

• Fixed connectivity:

$$P(k) = \delta_{kz}$$
 $J_c = z \log(\beta z)$

Numerical simulations:

Initial infected: m=10

Critical precaution level (sf)



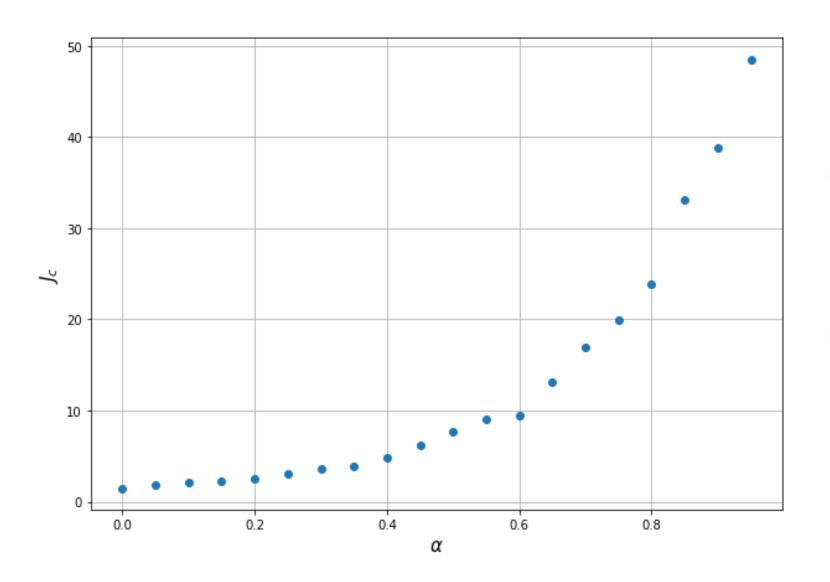
In the case of **scale-free network**, for $\alpha = 1$ there is no value of J for which the infection may be stopped for any value of β .

In real life, public service workers who are exposed to many contacts use additionally measures.

 α : increases the perception of the risk (or the safety measures) for nodes with higher connectivity (hubs).

Critical precaution level (sf)





- Hubs may fail to be infected due to their increased risk perception and this stops efficiently the spreading.
- This transition may be a finite-size effect (cut-off on degree distribution)

Conclusions



- For networks with fixed or peaked connectivity there is always a finite critical value of J.
- In the "worst case" social network (scale-free input connectivity), a linear perception cannot stop the disease.
- If the perception is increased in a non-linear way, the epidemics may get extinct.



- Separate social and physical contacts networks
- Model fear as a continuos variable
- Model epidemic as SEIRD
- Couple fear to number of infected and deaths neighbors

Each node j has a fear level given by $\left.fear_j = exp\left\{-\left(arac{i}{k} + brac{d}{k}
ight)
ight\}$

Each node infects with prob beta*fear_j

Social Net:

Facebook network

Edges: ~ 40K

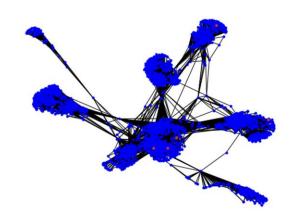
Nodes: ~ 4K

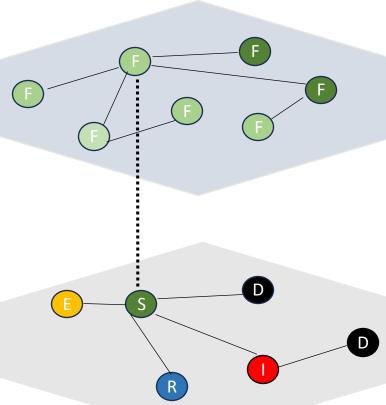
Average degree: ~ 43

Epidemic net:

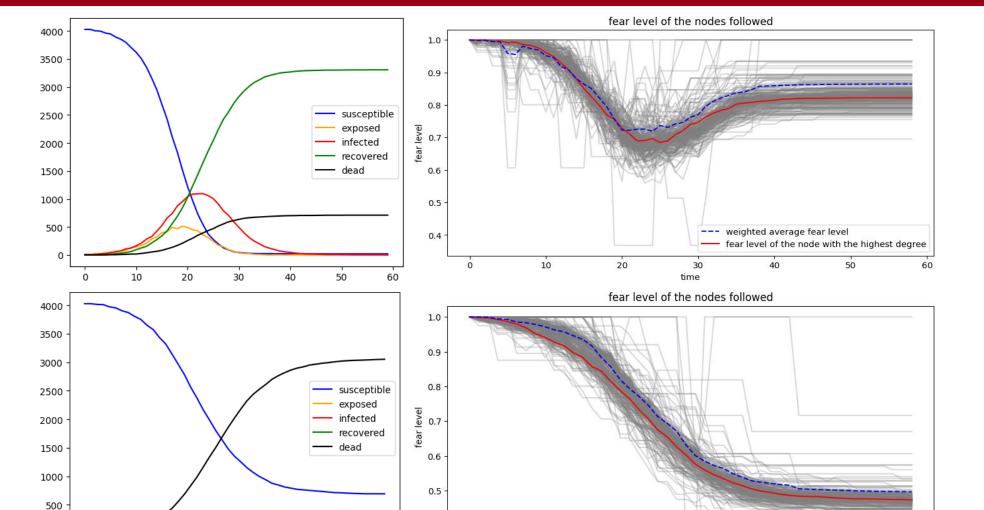
Watts Strogatz (k=5,p=0.5)

Average degree: ~ 4









--- weighted average fear level

10

20

time

fear level of the node with the highest degree

20

time

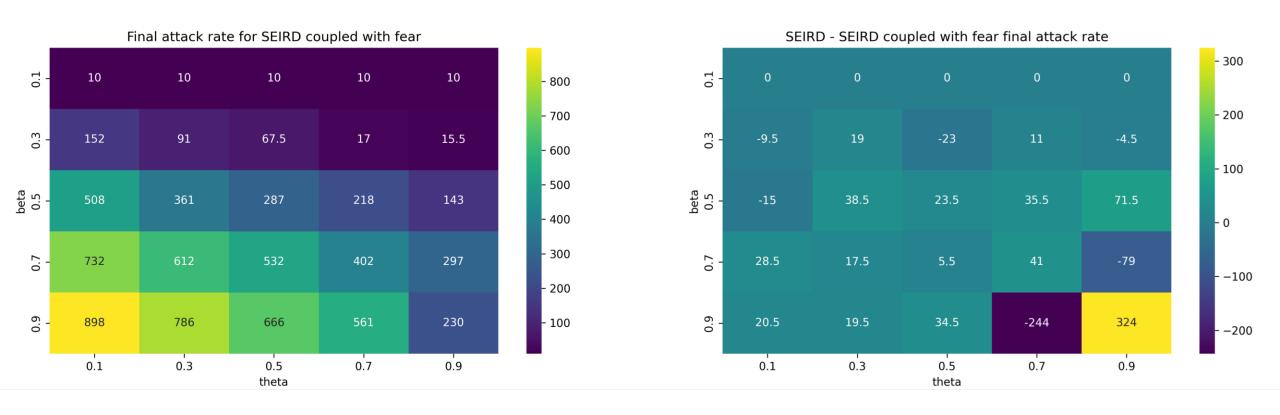
30

10

Sigma = 0.6 E->I Beta = 0.7 S->E Mu = 0.2 I->R Theta = 0.05 I->D a=1 b=1

Sigma = 0.6 E->I Beta = 0.7 S->E Mu = 0.2 I->R Theta = 0.7 I->D a=1 b=1





Number of maximum infected nodes decreases for higher theta as the fear level increases lowering the infectivity







Conclusions



- We have modeled fear in a more realistic way coupling it to the number of infected/deaths neighbors
- Separating social and contacts network lead to complex dynamics (higher number of parameters)

Adaptive network



Humans tend to respond to the emergence of an epidemic by avoiding contacts with infected individuals.

- Susceptible can avoid contact with infected by rewiring their network connections with a given probability.
- Static infection rate β.
- Both dynamics on network and dynamics of network.

SIS on adaptive network



We are interested to study the consequences of this kind of fear model in terms of epidemic threshold.

- Network: Erdős–Rényi (N = 1000, p = 0.02)
- **Dynamics on network**: SIS model with β infection rate, μ = 0.02 recovery rate.
- **Dynamics of network**: with probability w for every SI-link, the susceptible breaks the link to the infected and forms a new link to another randomly selected susceptible. Double and self-connections are not allowed.

Epidemic threshold



Epidemic threshold without rewiring (w=0):

$$\beta_c = \frac{\mu}{\langle k \rangle} = \frac{\mu}{p(N-1)}$$

• By considering rewiring, the mean degree of an infected node is (on average):

$$k(t) = \langle k \rangle e^{-wt}$$

then, by averaging over the typical lifetime $1/\mu$ of an infected node:

$$\beta_c = \frac{w}{\langle k \rangle (1 - \exp(-w/\mu))}$$

Mean field approximation

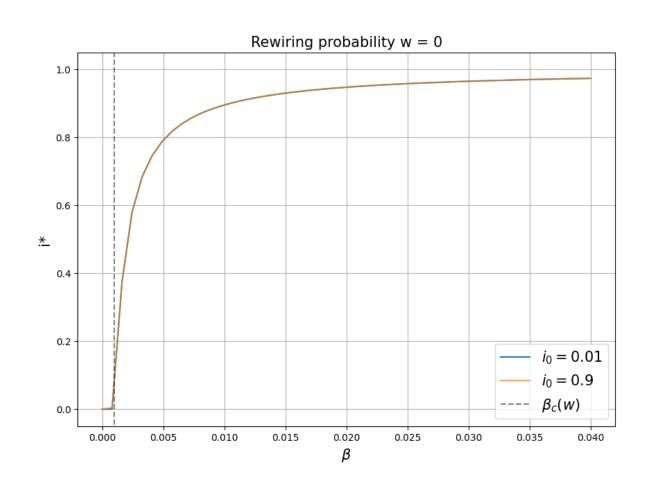


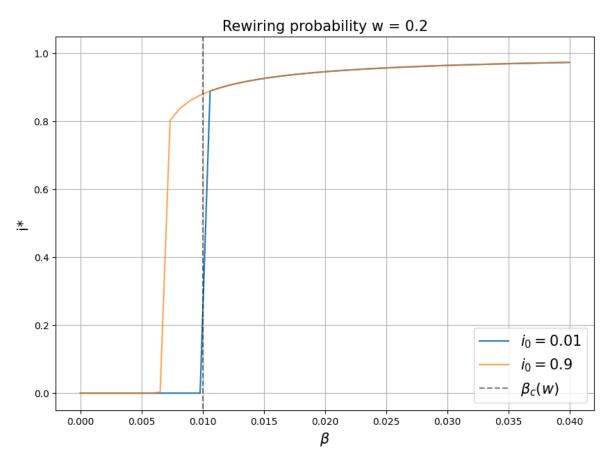
$$\begin{cases} \frac{d}{dt}i = \beta l_{SI} - \mu i \\ \\ \frac{d}{dt}l_{II} = \beta l_{SI} \left(\frac{l_{SI}}{s} + 1\right) - 2\mu l_{II} \\ \\ \frac{d}{dt}l_{SS} = (\mu + w)l_{SI} - 2\frac{\beta l_{SI}l_{SS}}{s} \end{cases}$$

$$l_{SI} = \langle k \rangle / 2 - l_{SS} - l_{II}$$

Mean field approximation

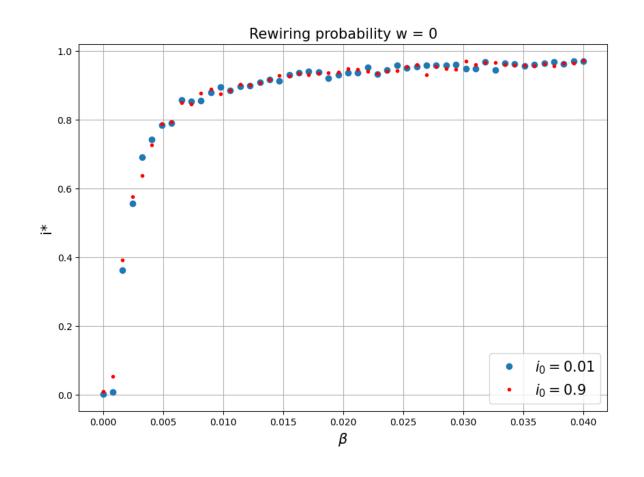


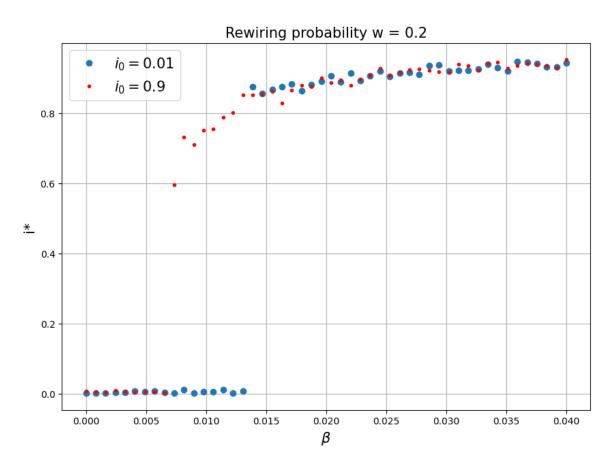




Simulations







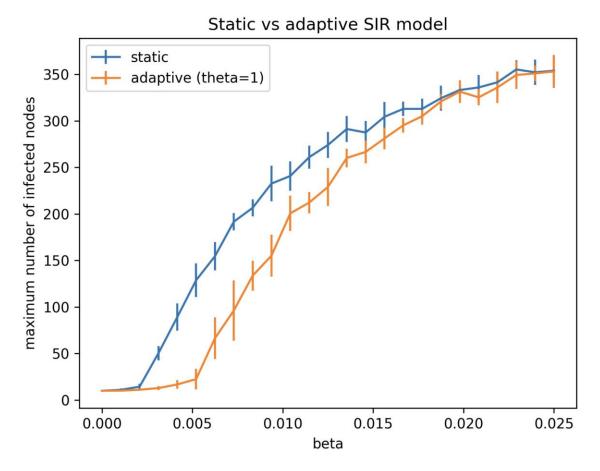
Conclusions



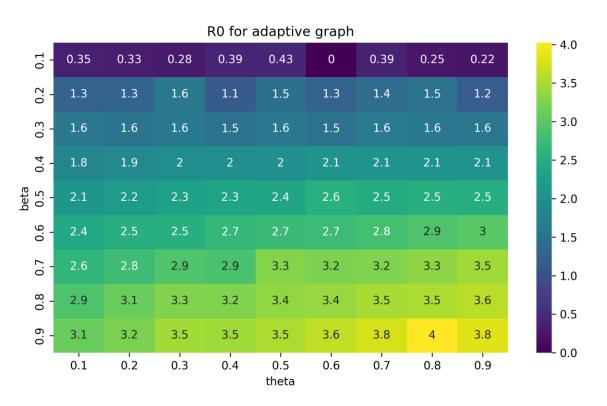
- Adaptive rewiring can increase the epidemic threshold
- It promotes the isolation of infected individuals...
 ...but it leads to the formation of highly connected susceptible cluster
- This fact can enable the persistence of diseases which would not be able to persist at low infection densities (bistability)

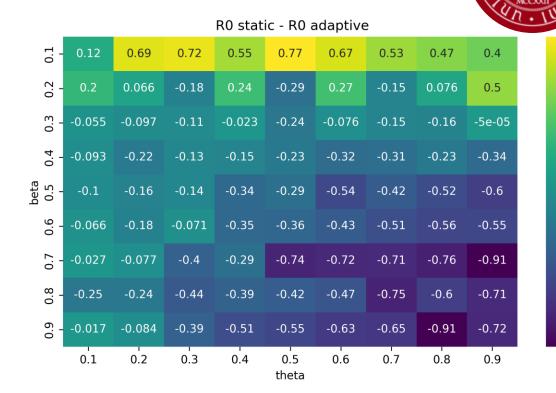


- Same rewiring rule as SIS
- For lower betas the rewiring helps the epidemic to slow down
- Increasing the infectivity vanish this effect



Watts Strogatz (N=500,k=5,p=0.15) Mu=0.1





Increasing further the infection rate has the opposite effect:

- It helps the epidemic to spread more aggressively
- Explanable by formation of hubs

- 0.6

- 0.4

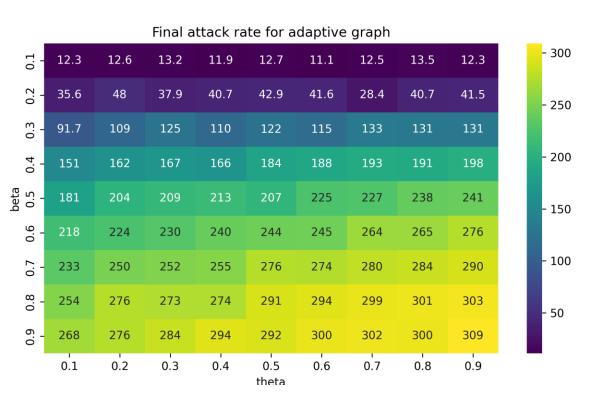
- 0.2

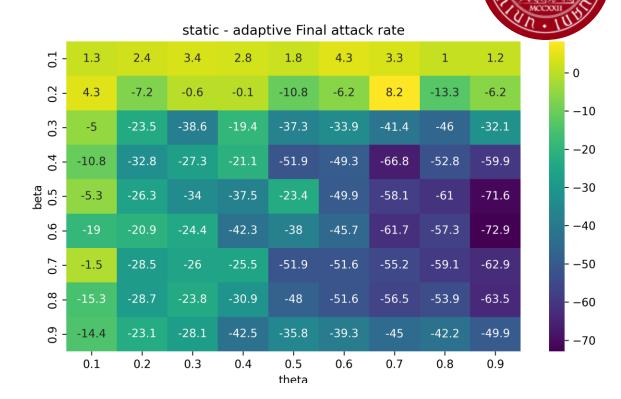
- 0.0

- -0.2

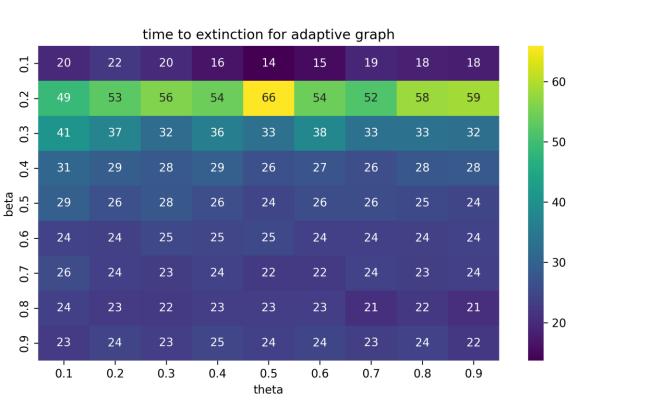
- -0.4

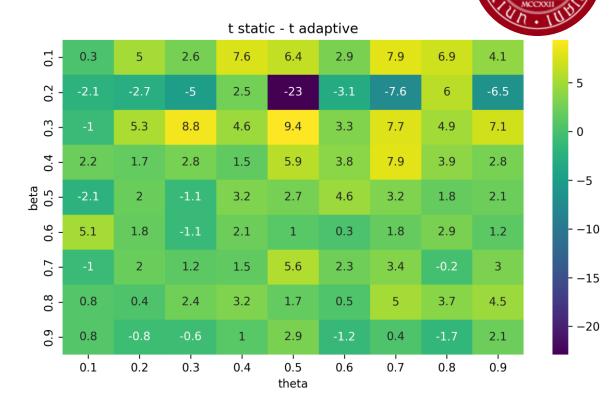
- -0.6





Indeed the maximum number of infected nodes for the adaptive case is higher





The time to extinction for the adaptive case is lower so the epidemic dies faster but leads to more infected

Conclusions



- Rewing for lower betas make the epidemic less aggresive as expected
- For higher betas the rewiring help the epidemic to spread faster because of the formation of hubs

Bibliography



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