



Northeastern

EPIDEMIC SPREADING

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 **MOBS LAB**
LABORATORY FOR THE MODELING OF BIOLOGICAL
AND SOCIO-TECHNICAL SYSTEMS

MODELING EPIDEMIC

Modeling such processes is an old enterprise

- Bernoulli in 1760 studied the effectiveness of inoculation against smallpox
- Long tradition in mathematical sciences
- Let us focus on human-to-human interactions

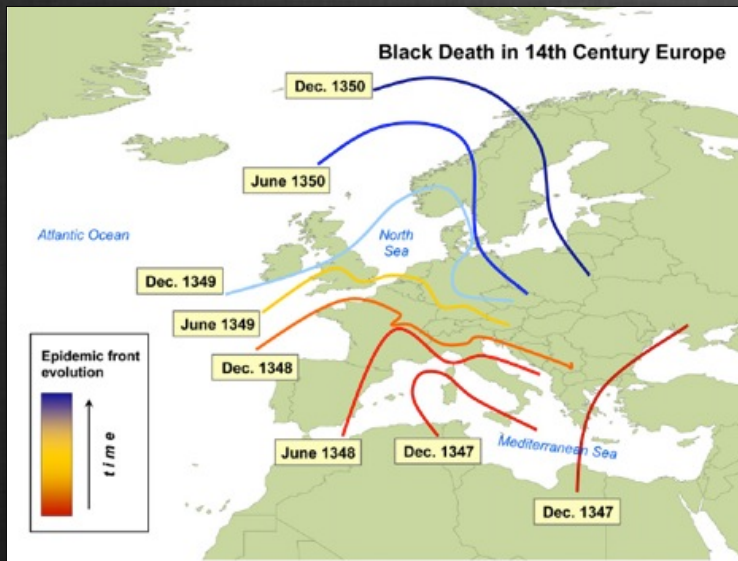
MODELING EPIDEMIC

Modeling epidemic spreading

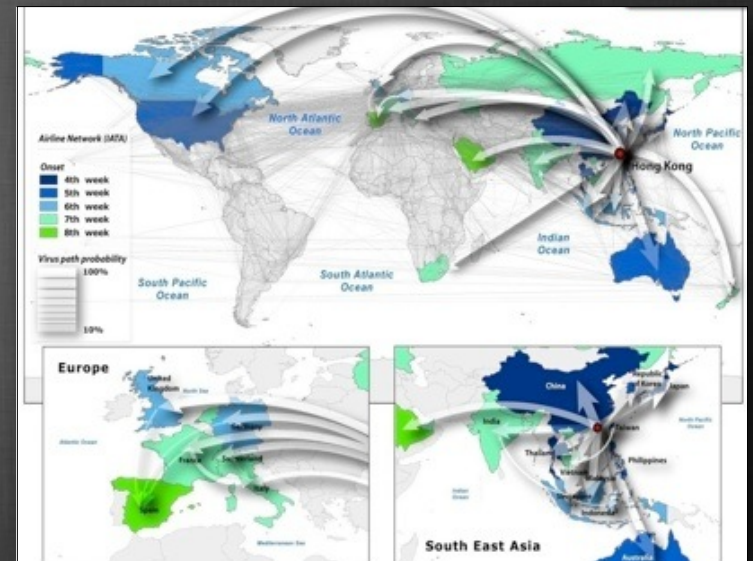
- We have an arsenal of models
- Each one is suited to specific diseases
- ...and specific geographical scales
- Data is the big limiting factor

EPIDEMICS AND HUMAN DYNAMICS

Our mobility and contacts are crucial ingredients



Black death



SARS

EPIDEMICS AND HUMAN DYNAMICS

Feb 18 2009

Chicago
New York
Los Angeles
Houston
Toronto
Vancouver
Calgary
Indianapolis

La Gloria

Sao Paulo
Mexico City
Rio De Janeiro
San Juan
Bogota

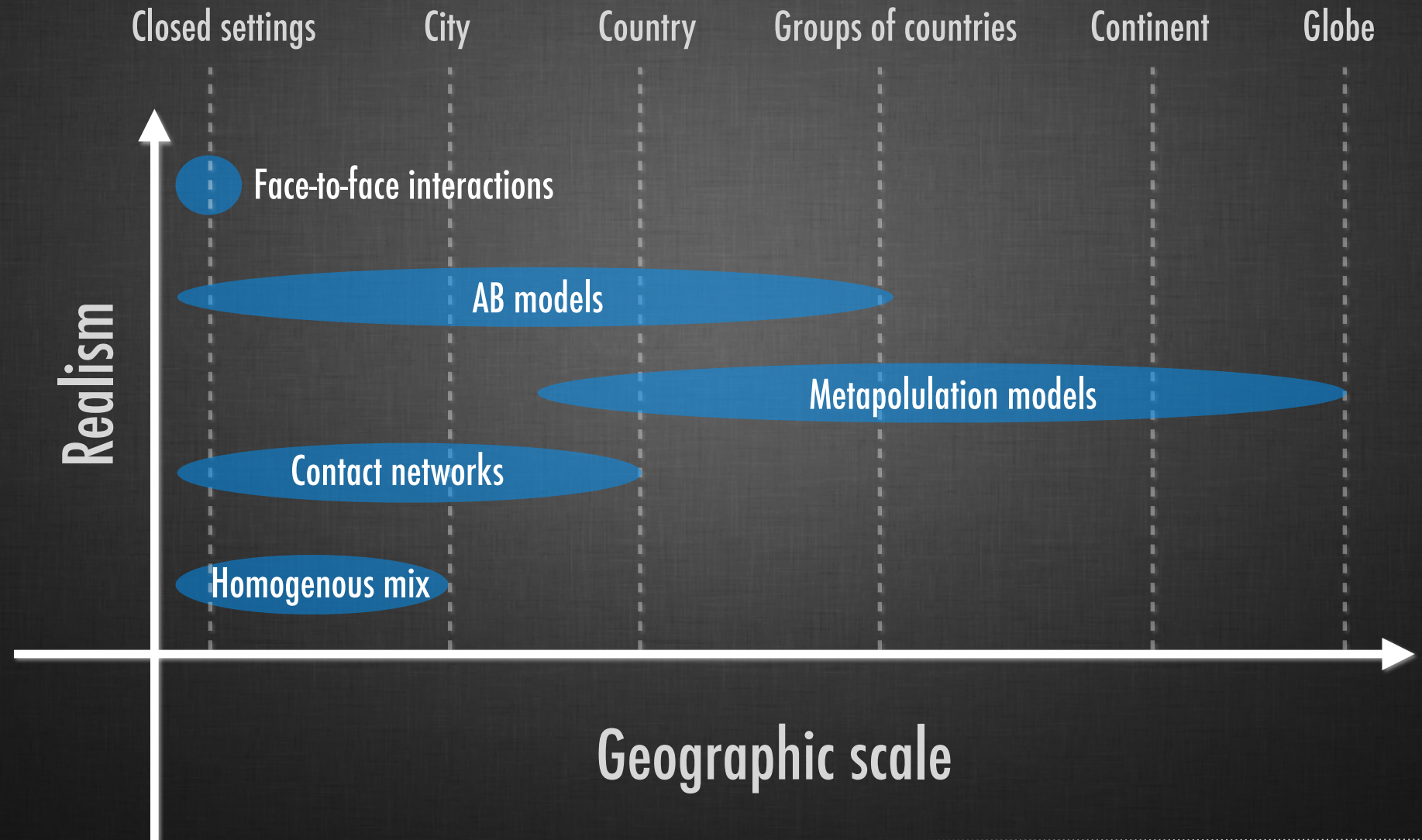
Johannesburg
Cairo
Cape Town
Nairobi

London
Paris
Frankfurt
Amsterdam
Rome
Milan
Moscow
Dublin

Hong Kong
Tokyo Narita
Bangkok
Singapore
Beijing
Manila

Sydney
Brisbane
Auckland
Perth

GEOGRAPHIC SCALE



MODELING EPIDEMIC

Basic concepts

- Population divided in compartments according to the disease status
- Susceptible (S)
- Latent (L)
- Infectious (I)
- Recovered (R)

MODELING EPIDEMIC

Natural history of the disease

- Describe the possible steps, and sequence of transitions between compartments
- There three main classes of diseases
 - SI
 - SIS
 - SIR

MODELING EPIDEMIC

Modeling transition between compartments

Infection process



Recovery process

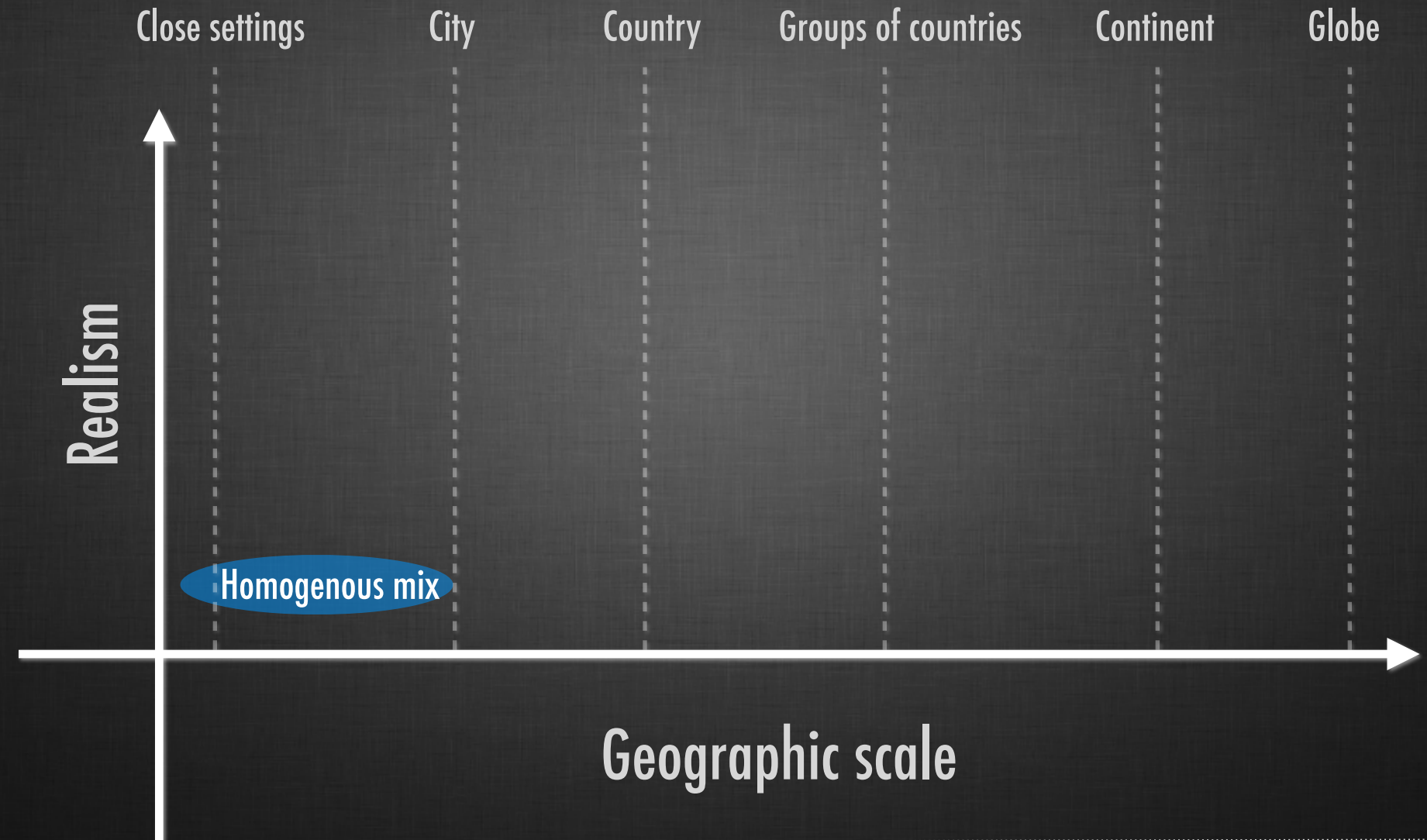


MODELING EPIDEMIC

Modeling the infection process

- Intuitively it should be function of :
 - the number of infected individuals in the population
 - the probability of infection given a contact with an infected
 - the number of such contacts

HOMOGENOUS MIXING



HOMOGENOUS MIXING

Force of infection

- Per capita rate at which susceptibles contract the disease
- mass-action law

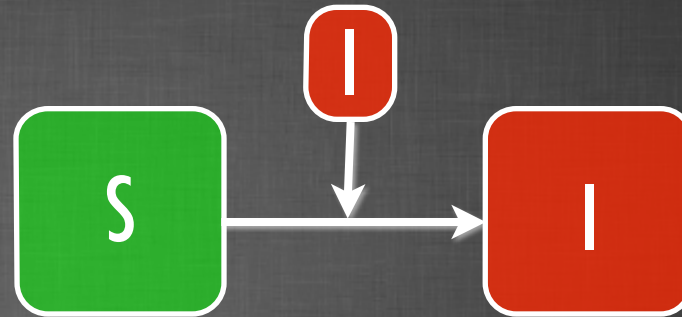
$$\lambda = \beta \frac{I}{N}$$

β : transmission rate

HOMOGENOUS MIXING

SI model

- Simplest model
- Infection is permanent
- Examples: HIV, HBV,



$$S(t + dt) = S(t) - \beta S(t) \frac{I(t)}{N} dt$$

$$I(t + dt) = I(t) + \beta S(t) \frac{I(t)}{N} dt$$

HOMOGENOUS MIXING

In the

$$d_t S = -\beta S \frac{I}{N}$$

$$d_t I = \beta S \frac{I}{N}$$

Often convenient using densities

$$d_t s = -\beta s i \quad s \equiv \frac{S}{N}$$

$$d_t i = \beta s i \quad i \equiv \frac{I}{N}$$

HOMOGENOUS MIXING

Python code

```
population=np.zeros(2,int)
# 0, will be referring to S
# 1, will be referring to I
```

```
def SI(population,beta,N):
    # the force of infection is given by beta*I/N
    prob_of_infection=beta*population[1]/N
    # given this prob. we can evaluate the number of people
    # going from S to I
    delta_pop=np.random.binomial(population[0],prob_of_infection)
    # update the population status
    population[0]-=delta_pop
    population[1]+=delta_pop
```

MATHEMATICAL

Exact

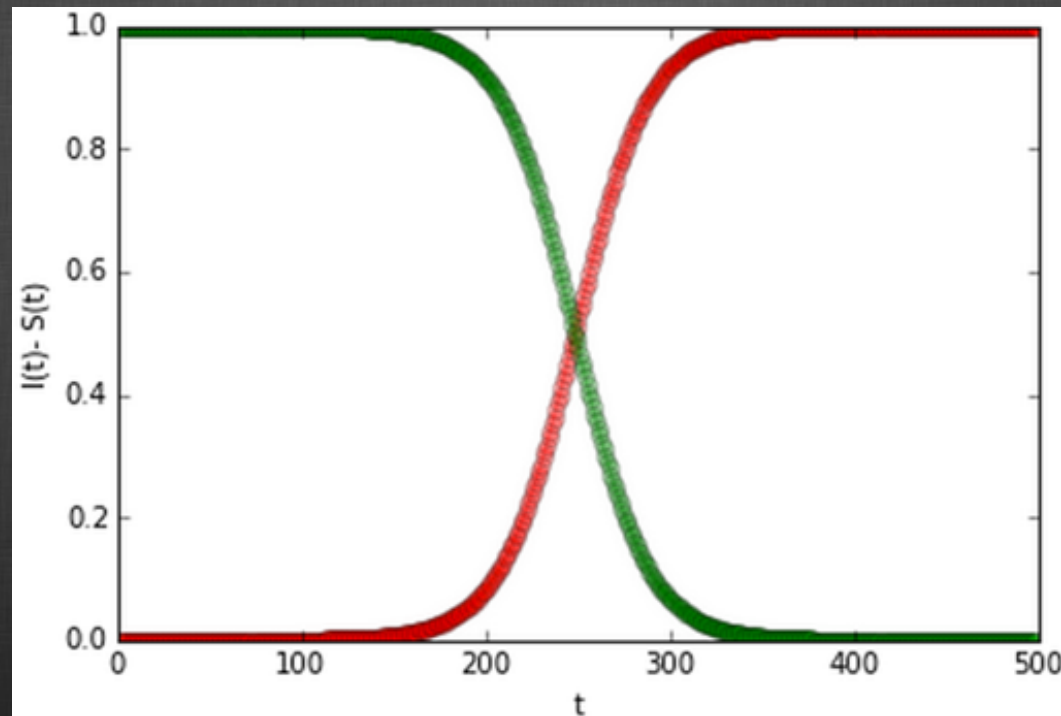
$$i(t) = \frac{1}{1 + \frac{a}{b}e^{-\beta t}}$$

$$s(t) = 1 - i(t)$$

$$s(\infty) = 0, \quad i(\infty) = 1$$

HOMOGENOUS MIXING

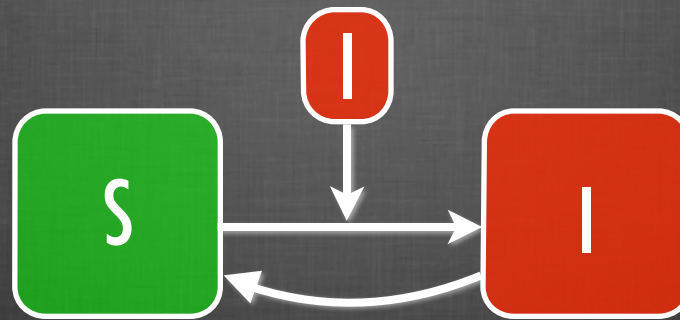
Numerical



HOMOGENOUS MIXING

SIS model

- Infection is not permanent, there is a recovery process
- Individuals after recovery are susceptible again



$$d_t s = -\beta si + \mu i \quad d_t i = \beta si - \mu i$$

HOMOGENOUS MIXING

Early time

- The number of infectious is small respect to the population size ($s \sim 1, i \sim 0$)

$$d_t i = \beta i - \mu i = (\beta - \mu) i$$

- Epidemic threshold:

$$\frac{\beta}{\mu} > 1$$

HOMOGENOUS MIXING

Basic reproductive number

- Central concept in epidemiology
- Definition: *average number of secondary infections generated by a initial seed in a fully susceptible population*
- Its expression depends on the details of the disease

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

HOMOGENOUS MIXING

Python code

```
def SIS(population,beta,mu,N):  
    # the force of infection is given by beta*I/N  
    prob_of_infection=beta*population[1]/N  
    # given this prob. we can evaluate the number of people  
    # going from S to I  
    delta_I=np.random.binomial(population[0],prob_of_infection)  
  
    # now we have another transition I->S  
    prob_of_recovery=mu  
    delta_S=np.random.binomial(population[1],prob_of_recovery)  
  
    # update the population status  
    population[0]=population[0]-delta_I+delta_S  
    population[1]=population[1]+delta_I-delta_S
```

MODELING EPIDEMIC

Exact solution

$$i(t) = \frac{\beta - \mu}{\beta + ae^{-\mu(R_0 - 1)t}}$$

Disease-free equilibrium

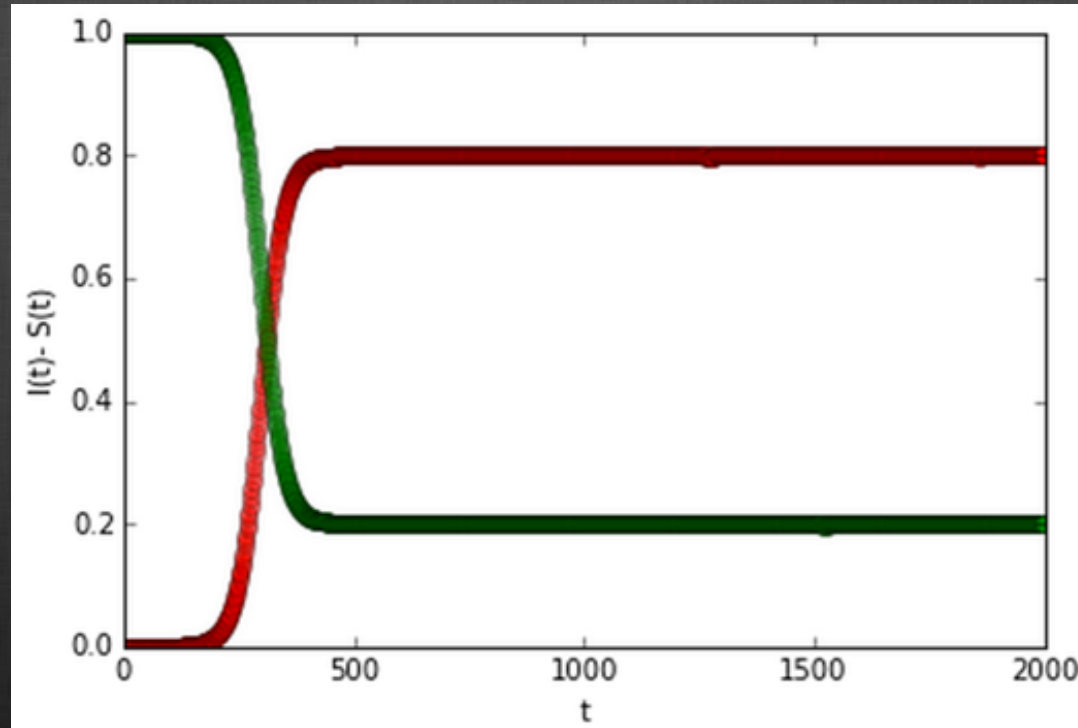
$$\text{if } R_0 < 1 \quad i(\infty) = 0$$

Endemic state

$$\text{if } R_0 \geq 1 \quad i(\infty) = 1 - \frac{1}{R_0}$$

HOMOGENOUS MIXING

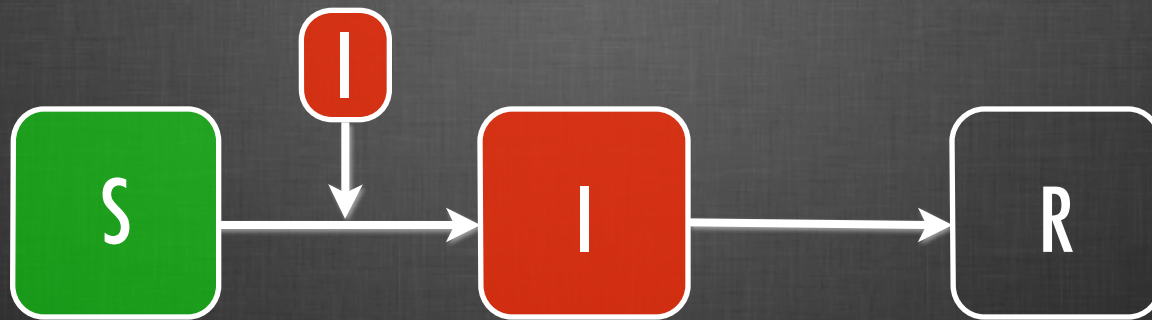
Numerical



HOMOGENOUS MIXING

SIR model

- Infection is not permanent, there is a recovery process
- Individuals after recovery are not susceptible again
- Examples: Influenza like illnesses (ILIs)



$$d_t s = -\beta si$$

$$d_t i = \beta si - \mu i$$

$$d_t r = \mu i$$

HOMOGENOUS MIXING

Early time

- Easy to prove that the same results for SIS hold
- Same epidemic threshold!

HOMOGENOUS MIXING

Disease free equilibrium

- The disease will eventually die off

$$s(\infty) = s_0 e^{-R_0 r(\infty)}$$

There will always be some individuals not affected!

HOMOGENOUS MIXING

Python code

```
population=np.zeros(3,int)
# 0, will be referring to S
# 1, will be referring to I
# 2, will be referring to R
```

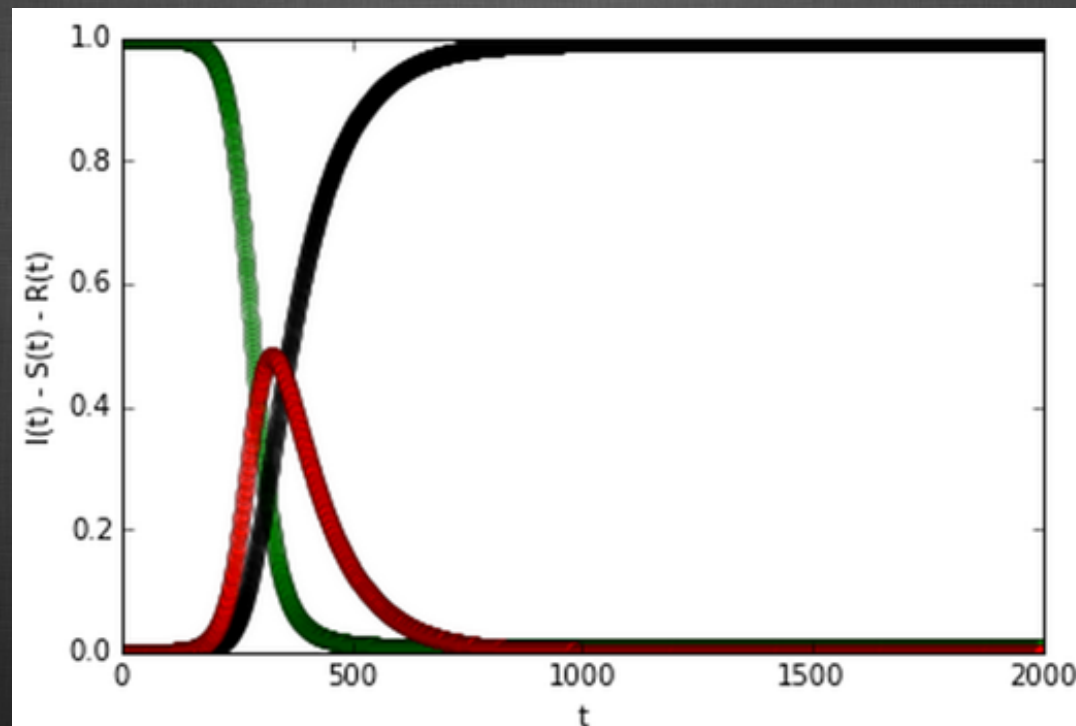
```
def SIR(population,beta,mu,N):
    # the force of infection is given by beta*I/N
    prob_of_infection=beta*population[1]/N
    # given this prob. we can evaluate the number of people
    # going from S to I
    delta_I=np.random.binomial(population[0],prob_of_infection)

    # now we have another transition I->R
    prob_of_recovery=mu
    delta_R=np.random.binomial(population[1],prob_of_recovery)

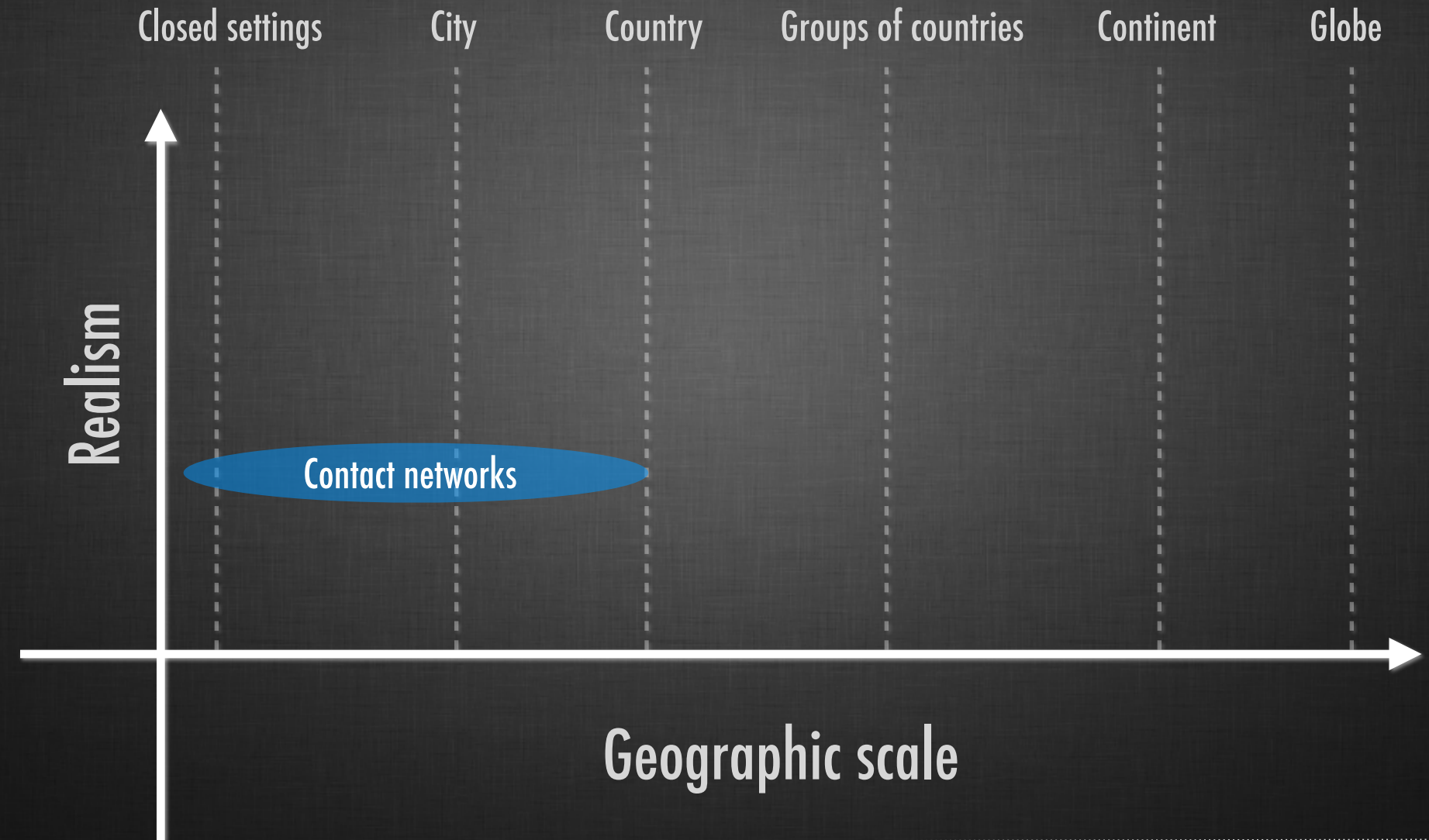
    # update the population status
    population[0]=population[0]-delta_I
    population[1]=population[1]+delta_I-delta_R
    population[2]=population[2]+delta_R
```

HOMOGENOUS MIXING

Numerical



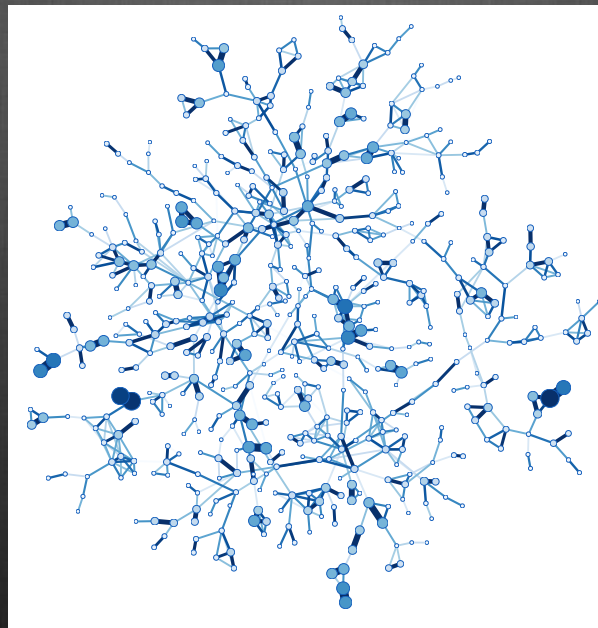
CONTACT NETWORKS



CONTACT NETWORKS

Epidemics in contact networks

- We relax the well mixed approximation
- We consider explicitly a connectivity network G
- Each node is person, and each link is an interaction (phone



CONTACT NETWORKS

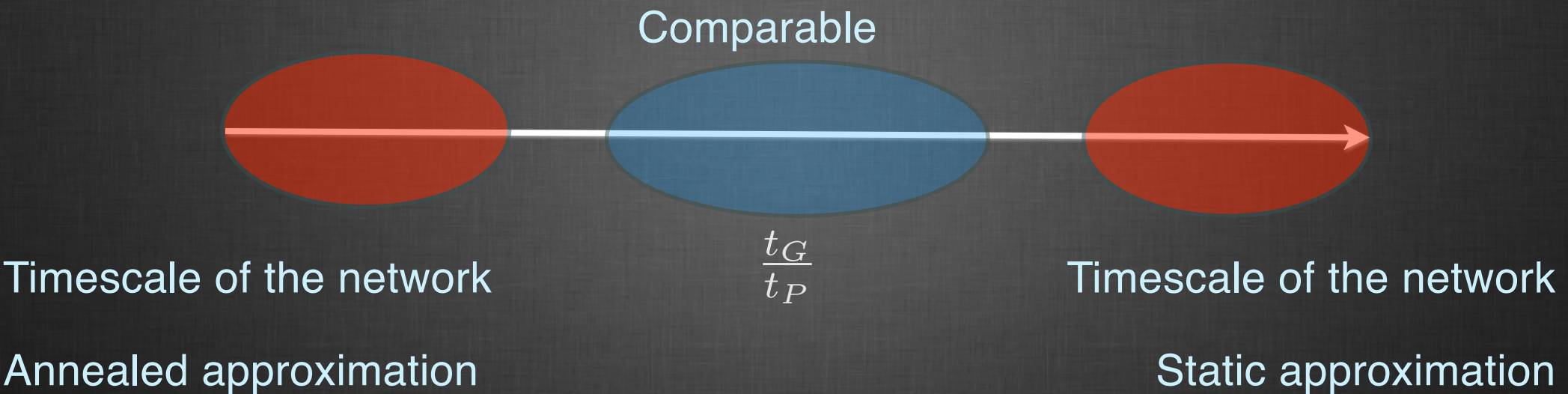
Epidemics in contact networks

- According to the data available different type of network representation can be used
- Weighted, unweighted, and temporal

In general we have two timescales:

- t_P describes the evolution of the process
- t_G describes the evolution of the network

TIMESCALES PROBLEM



CONTACT NETWORKS

Important note

- Infectious diseases spread through real interactions!
- Phone data could serve as proxies of social circles

CONTACT NETWORKS

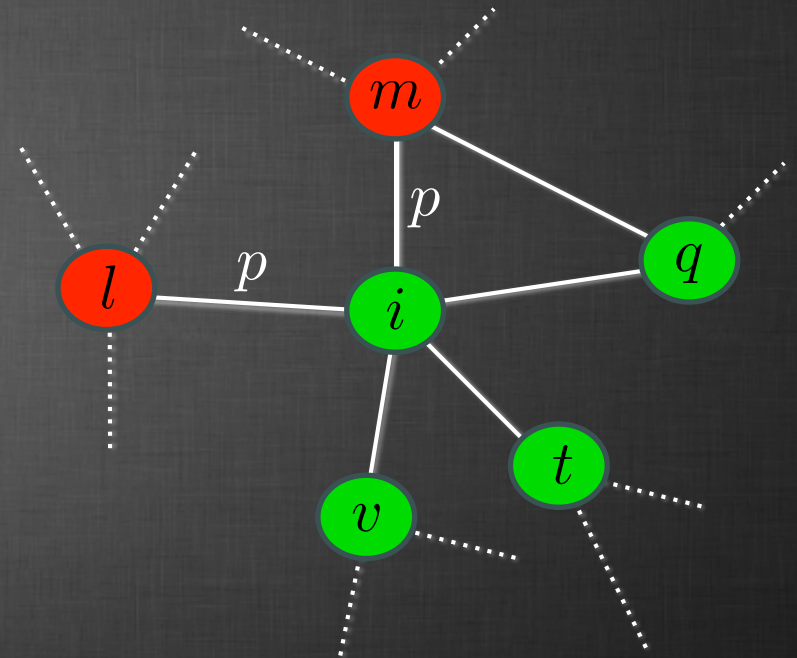
Modeling the contagion in contact

$$\lambda_i = p \sum_j A_{ij} X_j$$

$$X_i = \begin{cases} 1 & \text{if } i \text{ is infected} \\ 0 & \text{otherwise} \end{cases}$$

$$A_{ij} = \begin{cases} 1 & \text{if } i \text{ is connected to } j \\ 0 & \text{otherwise} \end{cases}$$

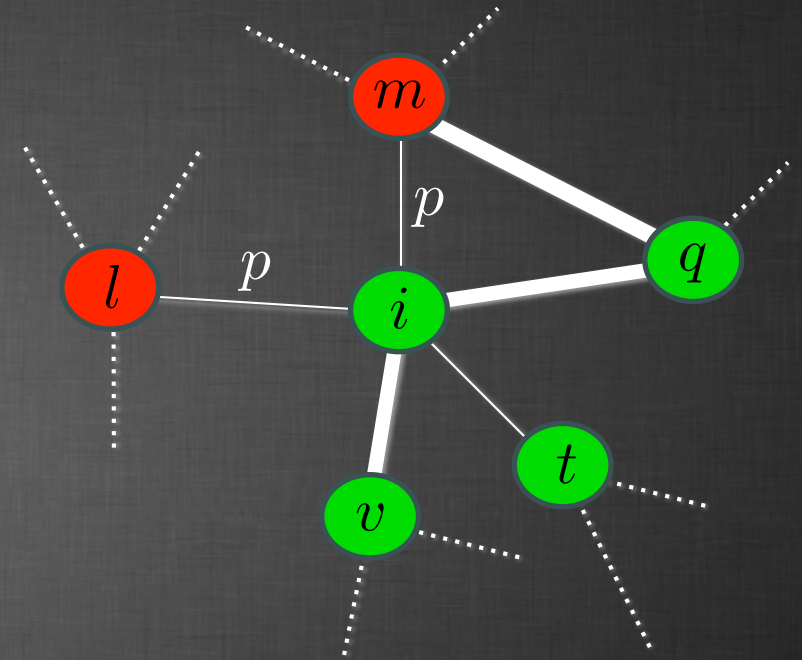
p : probability of infection per contact



CONTACT NETWORKS

- In the case of weighted

$$\lambda_i = p \sum_j W_{ij} X_j$$



$$X_i = \begin{cases} 1 & \text{if } i \text{ is infected} \\ 0 & \text{otherwise} \end{cases}$$

$$W_{ij} = \begin{cases} w & \text{if } i \text{ was connected to } j \text{ } w \text{ times} \\ 0 & \text{otherwise} \end{cases}$$

p : probability of infection per contact

CONTACT NETWORKS

Modeling the recovery in contact networks

- The same as before!

CONTACT NETWORKS

Python code (SIS model)

```
status=np.zeros(N,int)
```

```
def SIS_net(G,p,mu,status):  
    # we make the process synchronous  
    # at each time step all infected individuals can infect their peers  
    # status is a vector that takes two values for each node  
    # 0 -> S  
    # 1 -> I  
    temp_inf=set()  
    temp_rec=set()  
    for i in G.nodes():  
        # if the node is infected  
        if status[i]==1:  
            # we go through her neighbors and we try to infect the S  
            for j in G.neighbors(i):  
                if status[j]==0 and uniform(0,1)<p:  
                    temp_inf.add(j) # this node will be infected next  
  
            # recovery process  
            if uniform(0,1)<mu:  
                temp_rec.add(i)  
  
    # update the status  
    for i in temp_inf:  
        status[i]=1  
    for i in temp_rec:  
        status[i]=0
```

CONTACT NETWORKS

Effects of network structure

- Social networks are characterized by several important features that affect spreading processes:
 - the number of contacts is typically heterogeneous (**facilitates the spreading**)
 - the intensity of contacts is typically heterogeneous (**slows down the spreading**)

CONTACT NETWORKS

Understanding the effect of heterogenous number of contacts

- Let us consider a network $G(N,E)$ described by a degree distribution $P(k)$

$$d_t i_k = -\mu i_k + pk(1 - i_k)\Theta_k$$

Θ_k : density of infected neighbors

CONTACT NETWORKS

In the case of uncorrelated networks
the epidemic thresholds reads

$$\frac{p}{\mu} \geq \frac{\langle k \rangle}{\langle k^2 \rangle}$$

R. Pastor-Satorras, et al, PRL,
86,14,2001

Considering realistic degree

$$\langle k \rangle \ll \langle k^2 \rangle$$

The heterogeneity in the distribution of contacts

CONTACT NETWORKS

This is a worrisome scenario

- The degree distribution of real networks tends to facilitate the spreading
- Not all the nodes play the same role in the spreading
- Some, the most central, are crucial in sustaining the process
- If we can find them, we can efficiently protect the network

CONTACT NETWORKS

Two main classes

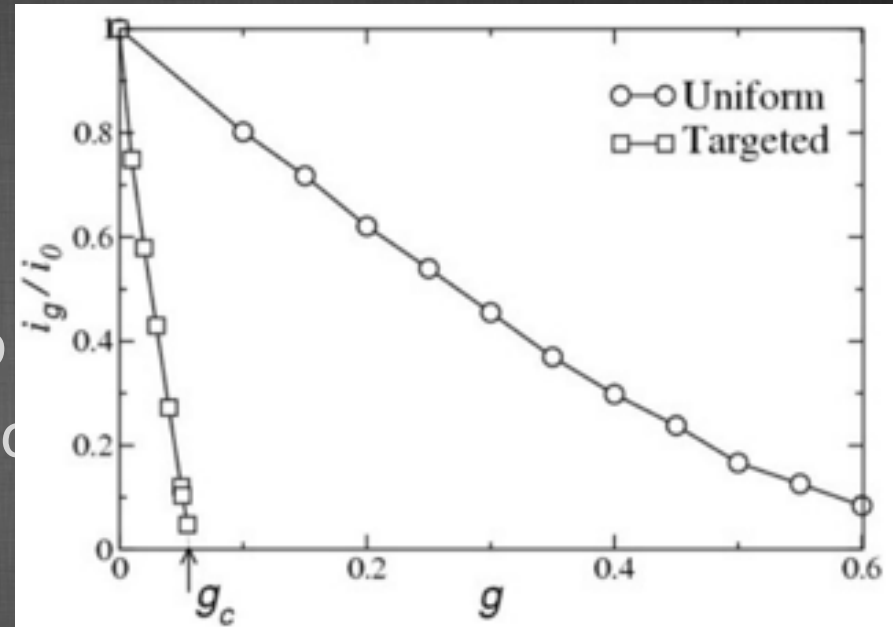
- Global knowledge is required
 - nodes can be selected considering their degree, betweenness, pagerank, closeness, k-core, etc., centrality
- Just partial access to the network is necessary
 - nodes can be selected through sampling processes

CONTACT NETWORKS

Pastor-Satorras, PRE, 65, 036

Problem formulation

- We have a fraction g of vaccines to
- Each node vaccinated is fully protected
- Let us see two different cases
 - vaccines randomly distributed
 - vaccines assigned proportionally to the degree of each node

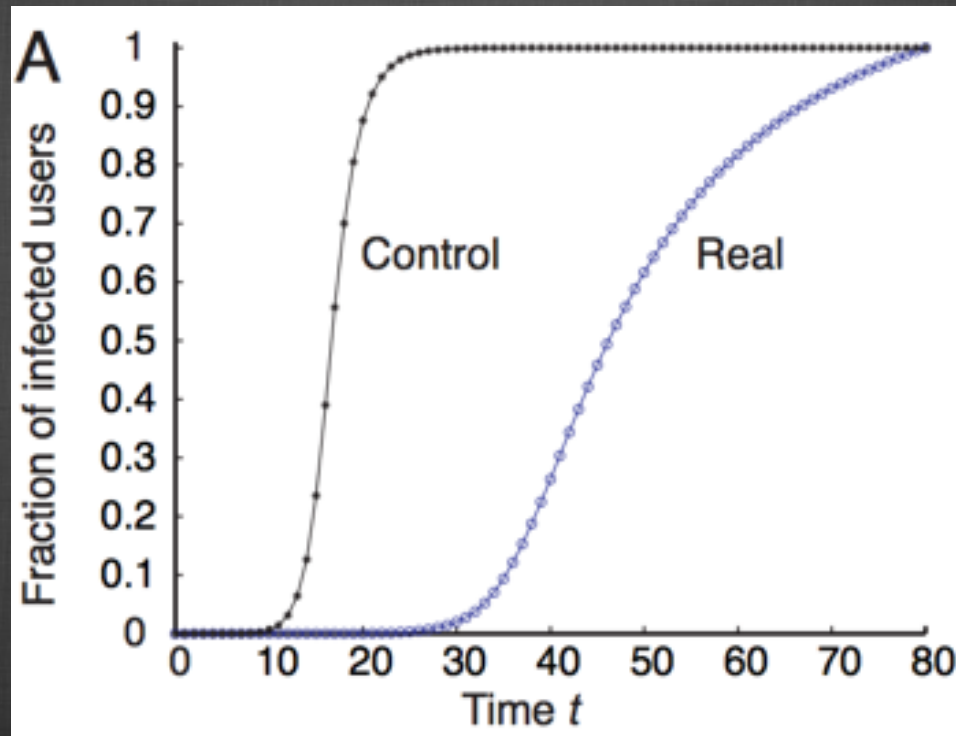


CONTACT NETWORKS

Understanding the effect of heterogenous intensity of contacts

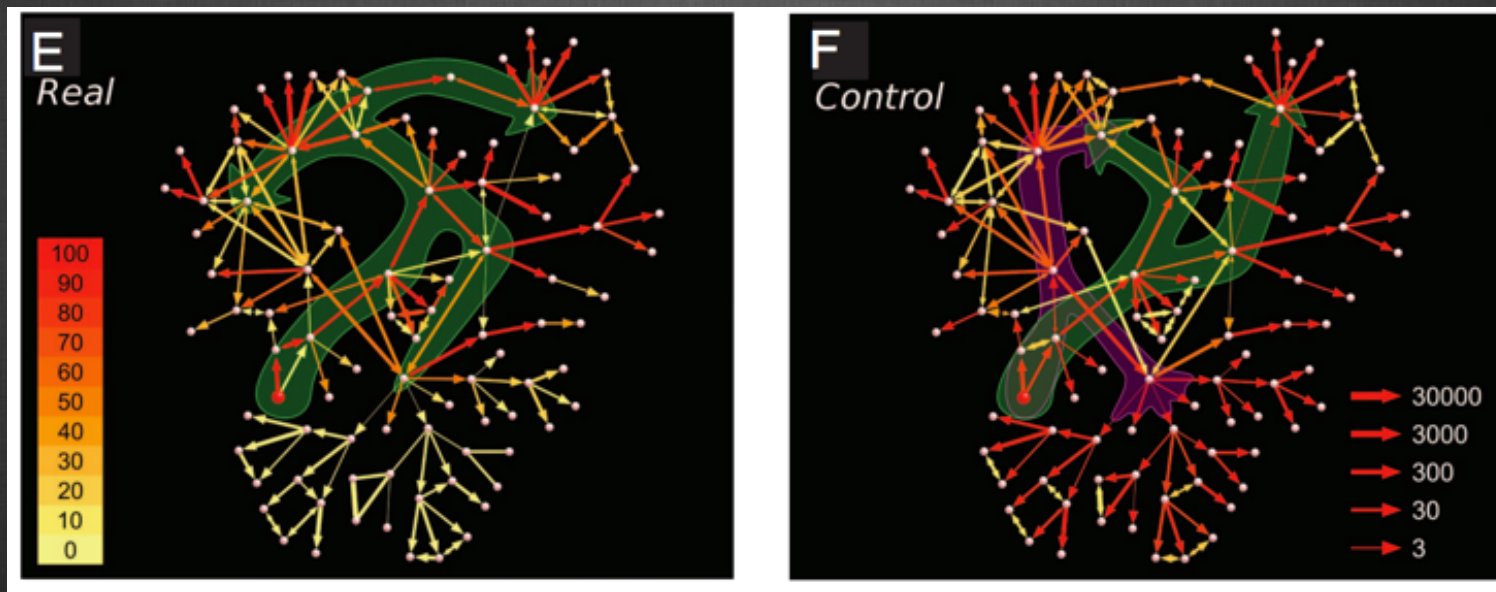
- Let us consider a real mobile phone datasets (Onnela et al, PNAS, 104, 18, 2007)
- Let us study the spread of a SI process on top
- As control we consider a network with the same degree

CONTACT NETWORKS



Onnela et al, PNAS, 104, 18, 2007

CONTACT NETWORKS



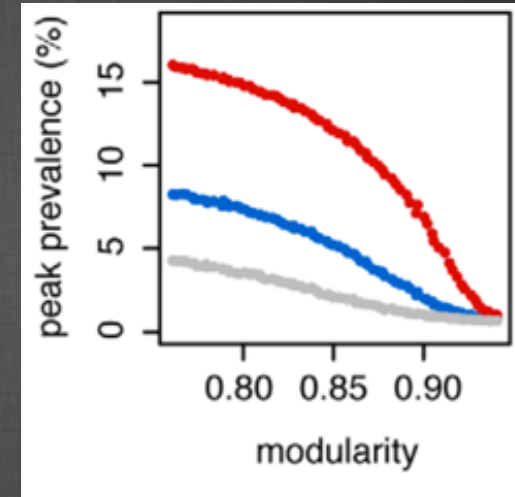
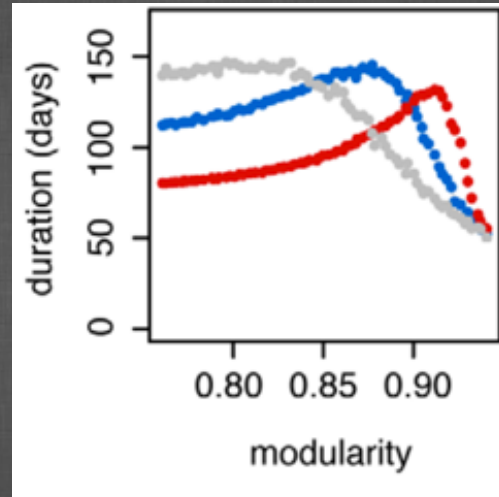
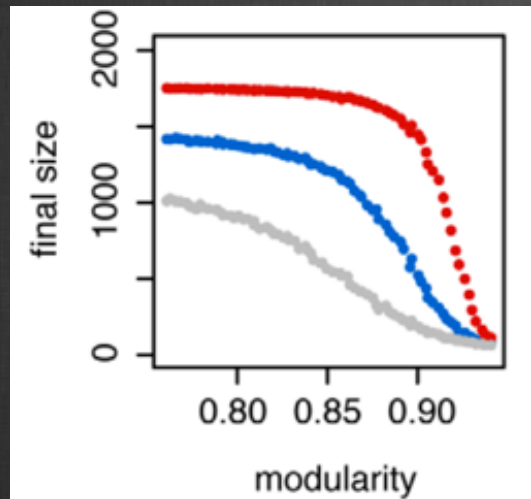
Onnela et al, PNAS, 104, 18, 2007

CONTACT NETWORKS

Understanding the effect of community structure

- Let us consider a set of synthetic networks with different modularity (Salathe et al., PLoS Comp. Bio., 6, 4, 2010)

CONTACT NETWORKS



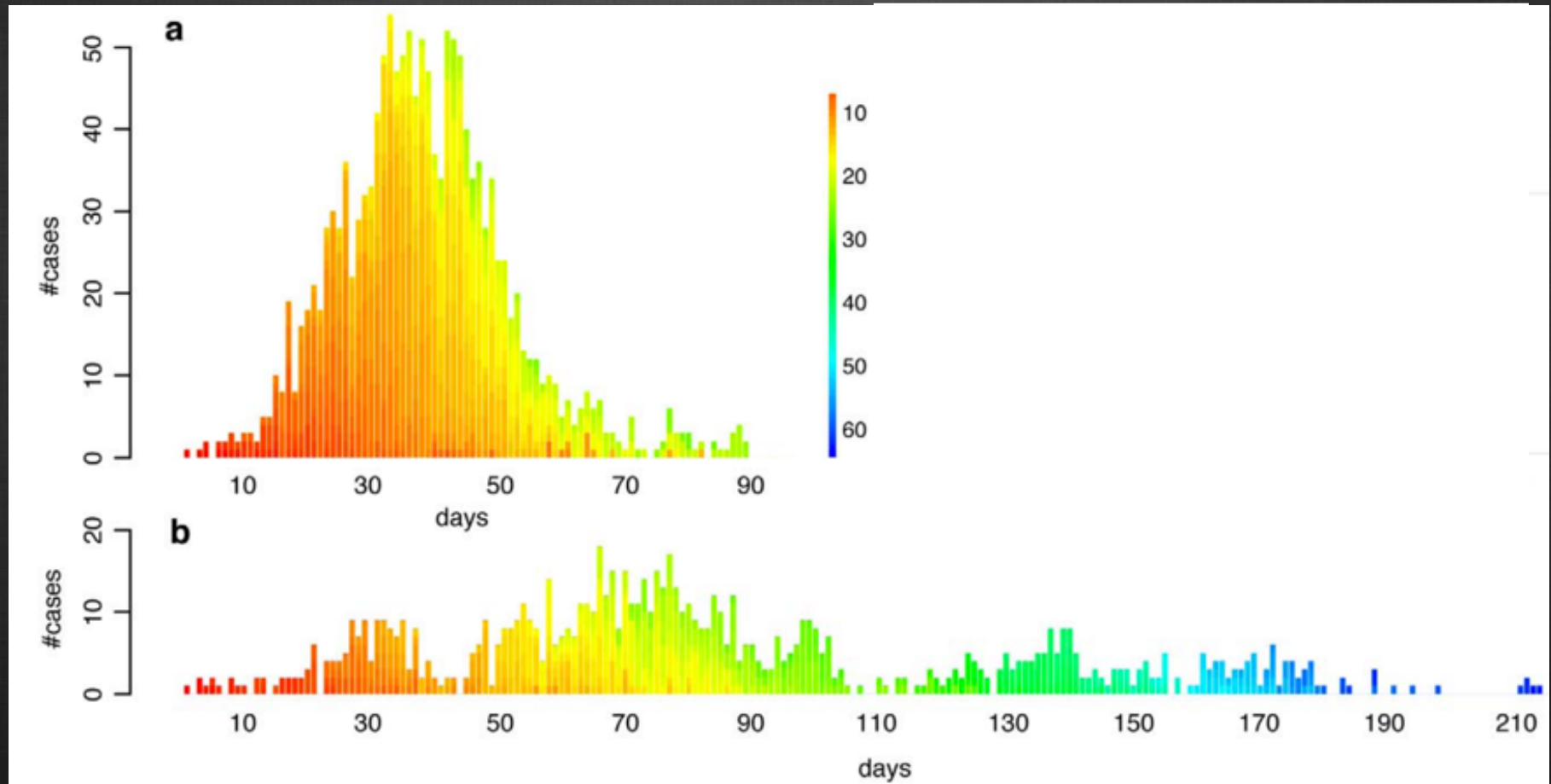
Salathe et al., PLoS Comp. Bio., 6, 4, 20

Grey $R_0=2.5$

Blue $R_0=3$

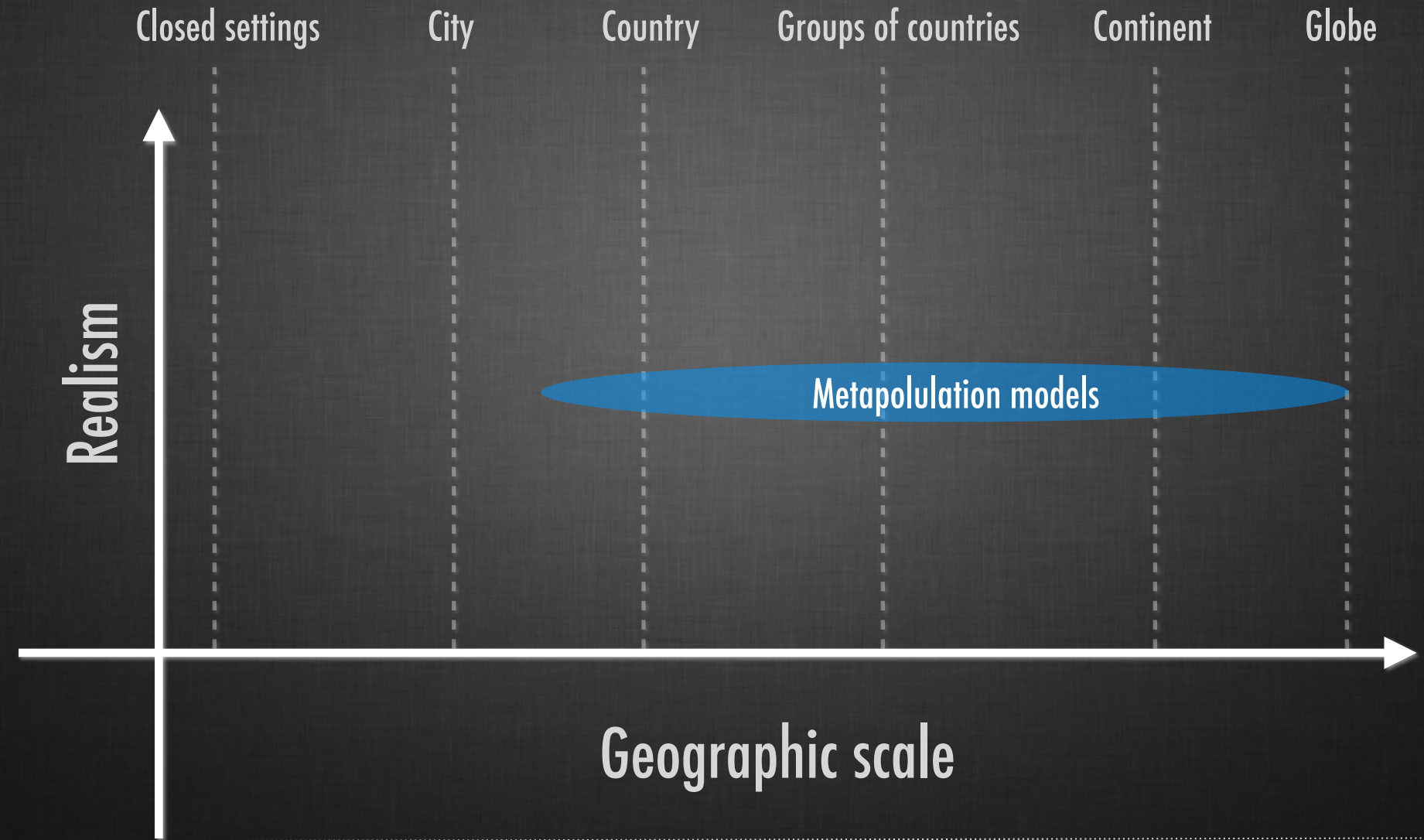
Red $R_0=4$

CONTACT NETWORKS



Salathe et al., PLoS Comp. Bio., 6, 4

METAPOPOPULATION



METAPOPOPULATION

Typically used to model patchy systems coupled by mobility

- Each patch is a geographical unit
- Patches are connected by mobility



METAPOPOPULATION

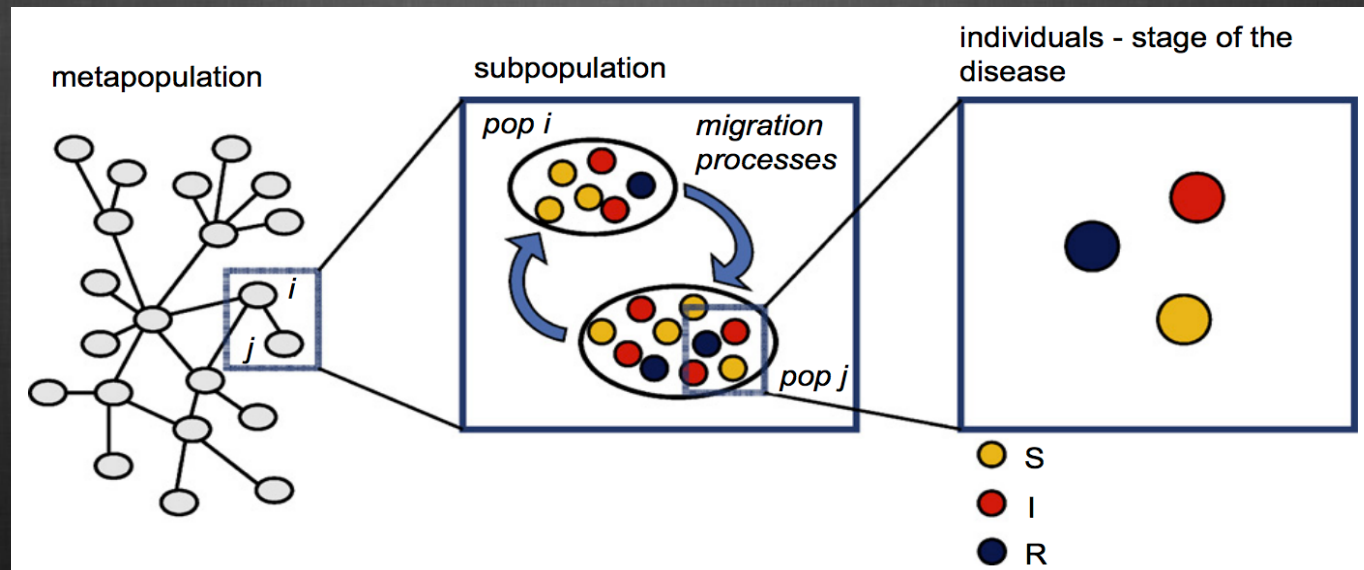
Used in many disciplines, plus..

- Extremely useful to reduce the level data necessary at large geographical scales
- Mobility data is available at many different scales

METAPOPOPULATION MODELS

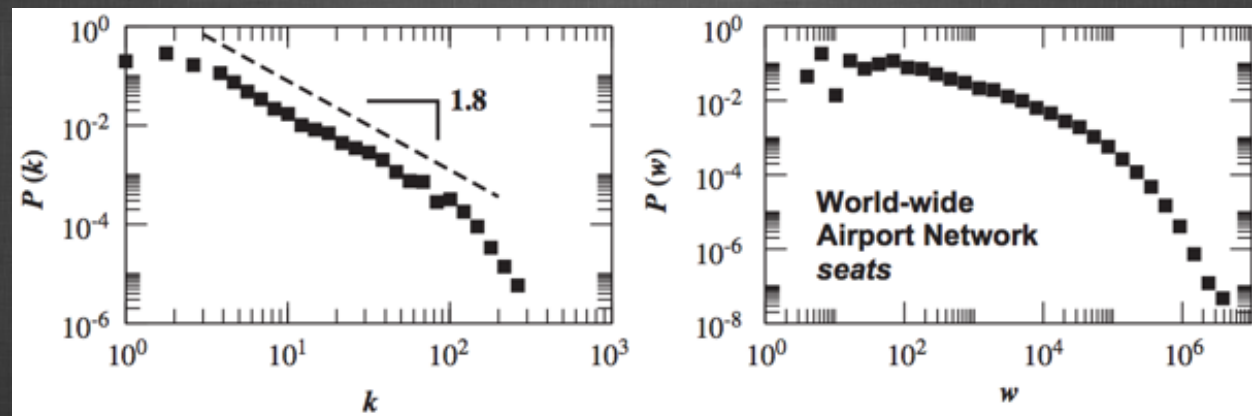
Reaction-Diffusion framework

- Considering the general lack on information about contacts inside each patch we can use the homogenous assumption inside each node



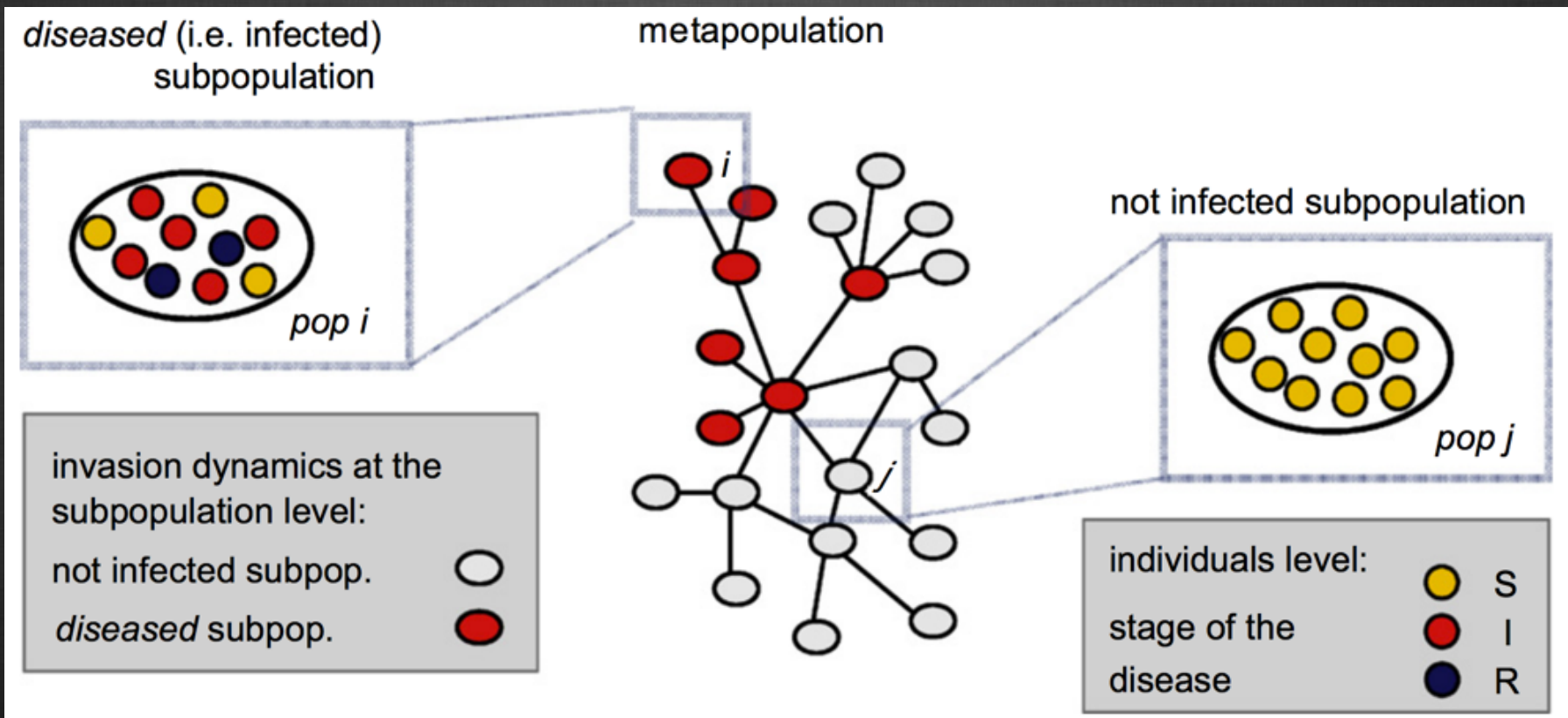
METAPOPOPULATION MODELS

Real networks

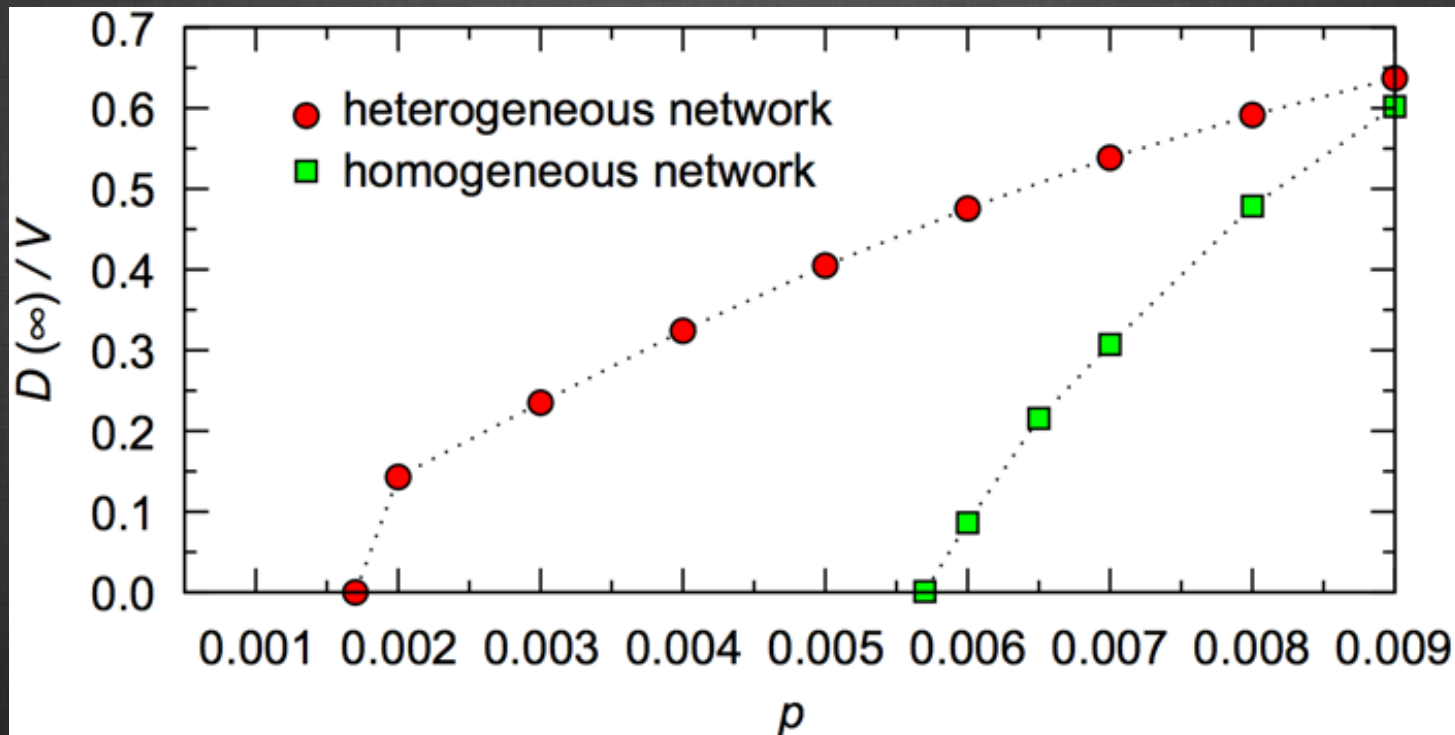


Also in this case degree and weight are heterogenous!

METAPOPULATION MODELS



METAPOPOPULATION MODELS



Critical effects introduced by heterogeneous degree distributions!

METAPOPULATION MODELS

Codes available at

<http://www.nicolaperra.com/teaching.html>

