### Chapter 13

## **Diffusion: Epidemics**

### Summary

- Probabilistic Epidemic Models
  - SI/SIS/SIR
- Mean Field Formulation
- Epidemics on Networks

### Reading

- Chapter 21 of Kleinberg's book
- Chapter 10 of Barabasi's book



# **Epidemic**

### Biological:

- Airborne diseases (flu, SARS, ...)
- Venereal diseases (HIV, ...)
- Other infectious diseases (HPV, ...)
- Parasites (bedbugs, malaria, ...)

### Digital:

- Computer viruses, worms
- Mobile phone viruses

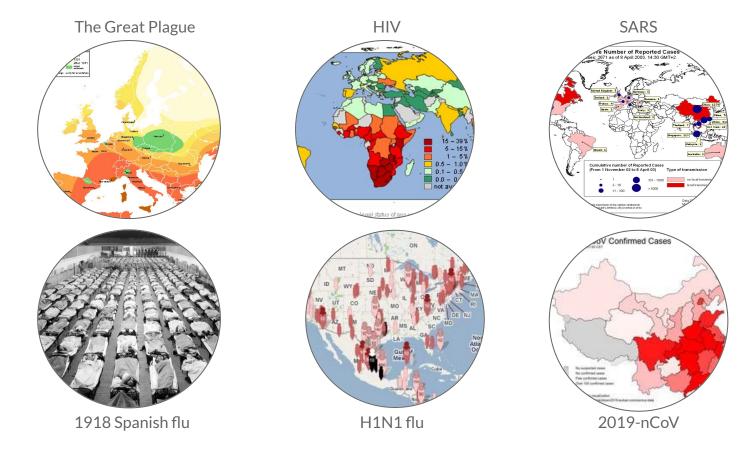
### Conceptual/Intellectual:

- Diffusion of innovations
- Rumors
- Memes
- Business practices

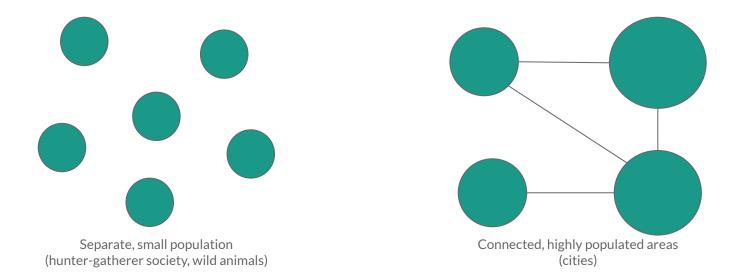
### Epi + demos

upon people





**Biological: Notable Epidemic Outbreaks** 



Human societies have "**crowd diseases**", which are the consequences of large, interconnected populations (Measles, tuberculosis, smallpox, influenza, common cold, ...)

### Large population can provide the "fuel"

# **Probabilistic Epidemic Models**



# Compartmental Models

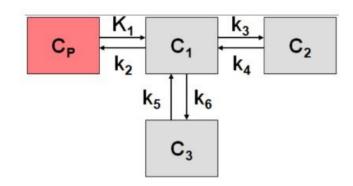
The framework is based on two hypotheses:

### Compartmentalization:

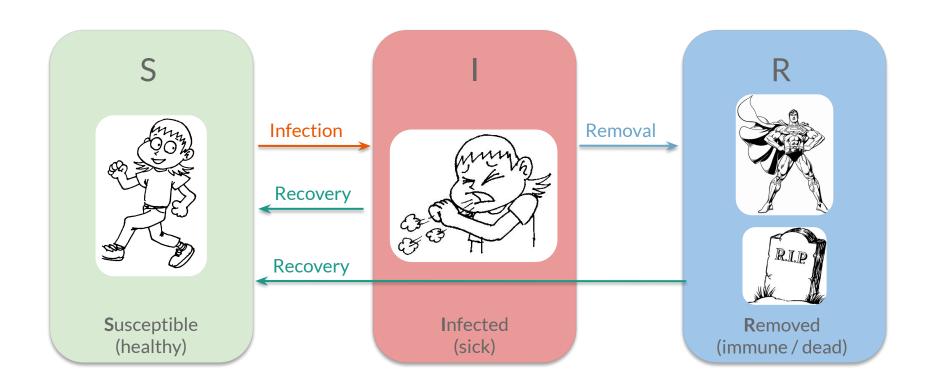
each individual is classified into distinct states. The simplest classification assumes that an individual can be in one of the states.

### Homogeneous Mixing:

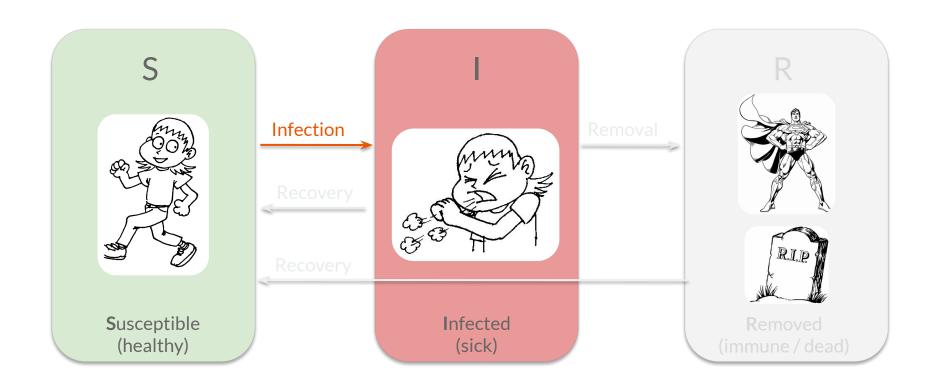
each individual has the same chance of coming into contact with an infected individual.



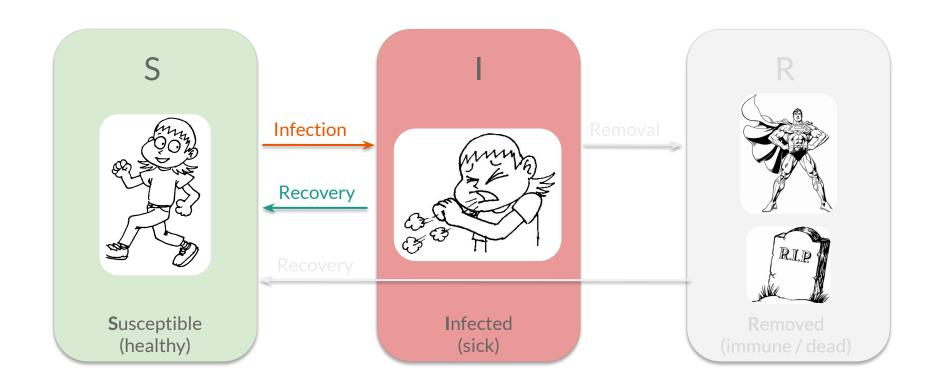
W. O. Kermack and Ag McKendrick. A Contribution to the Mathematical Theory of Epidemics. 1927



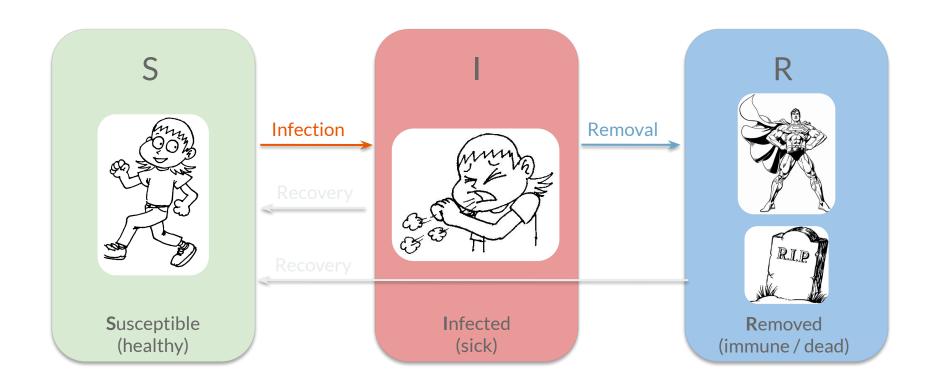
Classic Models Compartments



**SI**: The simplest model



SIS: Common Cold



SIR: Flu, SARS, Plague

## **Mean Field formulation**

(Homogeneous mixing)



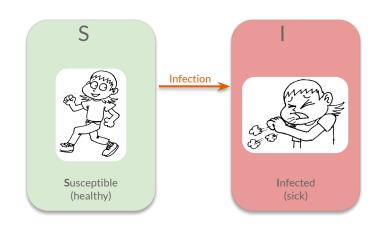
## SI model

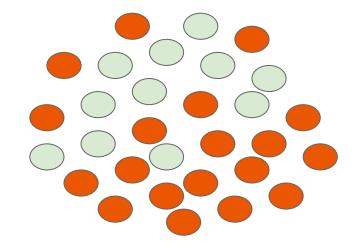
Each individual has  $\beta$  contacts with randomly chosen others individuals per unit time.

If there are I infected individual and S susceptible individuals, the average rate of new infection is  $\beta si/N$ 

with 
$$s = S/N$$
,  $i = I/N$ 

$$\frac{di}{dt} = \beta si = \beta i(1-i)$$





## SI model

# **Dynamics**

Logistic equation: a basic model of population growth.

$$\frac{di}{dt} = \frac{\beta i (1-i)}{S}$$

$$\frac{di}{i} + \frac{di}{(1-i)} = \beta dt \qquad \ln i - \ln(1-i) + c = \beta t$$

$$\frac{i}{1-i} = C \exp(\beta t) \qquad C = \frac{i_0}{1-i_0}$$

$$\ln \frac{i}{1-i} = c + \beta t$$

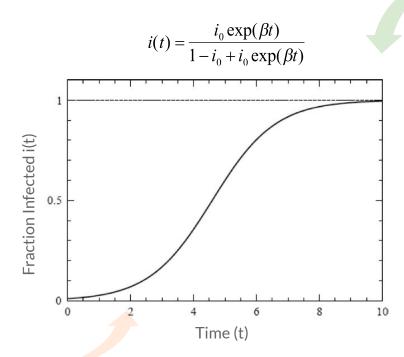
$$\therefore i(t) = \frac{i_0 \exp(\beta t)}{1 - i_0 + i_0 \exp(\beta t)}$$

# SI model **Behaviour**

If *i*(*t*) is small,

 $\frac{di}{dt} \approx \beta i$  $i \approx i_0 \exp(\beta t)$ 

exponential outbreak



As  $i(t) \sim 1$ .

$$\frac{di}{dt} \rightarrow 0$$

saturation

#### SI model:

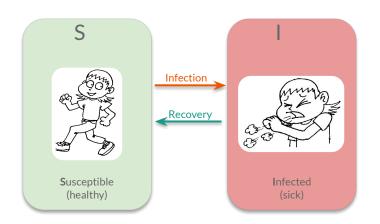
the fraction infected increases until everyone is infected.

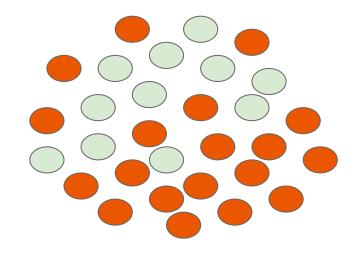


### **Modeling Common Cold**

Each individual has  $\beta$  contacts with randomly chosen others individuals per unit time.

Each infected individual has  $\mu$  probability of revert its status to susceptible





## SIS model

## **Behaviour**

$$\frac{di}{dt} = \frac{\beta i(1-i) - \mu i = i(\beta - \mu - \beta i)}{\frac{\beta}{1-\beta}}$$

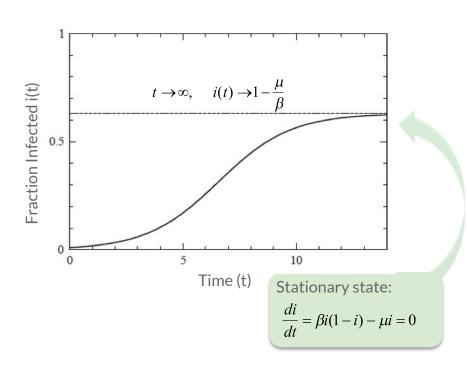
$$\frac{di}{i} + \frac{di}{1 - \mu/\beta - i} = (\beta - \mu)dt$$

$$\ln(i) - \ln(1 - \mu/\beta - i) = (\beta - \mu)t + c$$

$$\frac{i}{1 - \mu/\beta - i} = Ce^{(\beta - \mu)t}$$

$$\therefore i(t) = \left(1 - \frac{\mu}{\beta}\right) \frac{Ce^{(\beta - \mu)t}}{1 + Ce^{(\beta - \mu)t}}$$

# SIS model **Dynamics**



$$\therefore i(t) = \left(1 - \frac{\mu}{\beta}\right) \frac{Ce^{(\beta - \mu)t}}{1 + Ce^{(\beta - \mu)t}}$$

### SIS model:

the fraction of infected individual saturates below 1

## SIS model

# **Basic Reproductive**Number

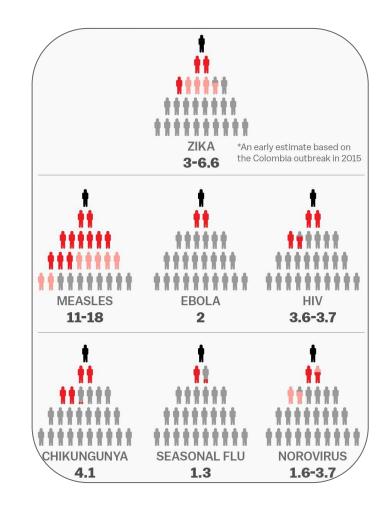
 $\hbar$  (also identified with R<sub>0</sub>): average # of infectious individuals generated by one infected in a fully susceptible population.

$$\lambda \equiv \frac{\beta}{\mu}$$

እ > 1: Outbreack

**1** < **1**: Die Out

Epidemic Threshold if  $\mu \approx \Box$  then  $i \rightarrow 0$ 

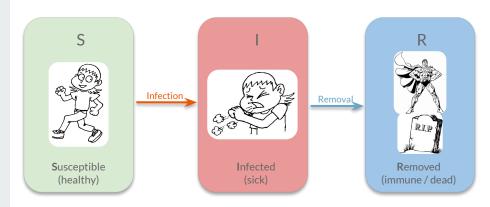


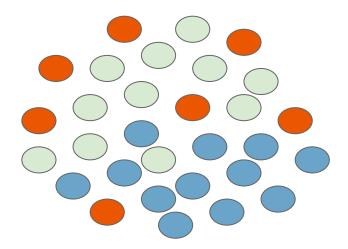
## SIR model

### Modeling Flu-like disease

Each individual has  $\beta$  contacts with randomly chosen others individuals per unit time.

Each infected individual has  $\mu$  probability of becoming immune after being infected





## SIS model

## **Behaviour**

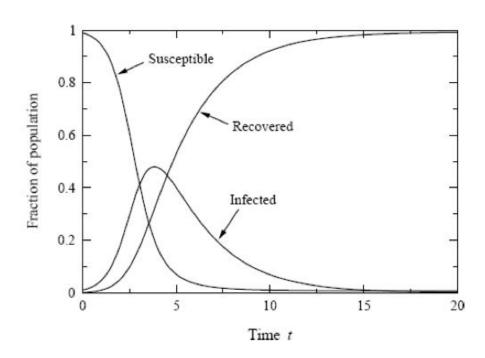
$$\frac{\mathrm{d}s(t)}{\mathrm{d}t} = \beta \langle k \rangle i(t) \left[ 1 - r(t) - i(t) \right]$$

$$\frac{\mathrm{d}i(t)}{\mathrm{d}t} = -\mu i(t) + \beta \langle k \rangle i(t) \left[ 1 - r(t) - i(t) \right]$$

$$\frac{\mathrm{d}r(t)}{\mathrm{d}t} = \mu i(t).$$

### SIR model:

the fraction infected peaks and the fraction recovered saturates.



		SI	SIS
1	Early Behaviour Exponential growth of infected individuals	$i(t) = \left(1 - \frac{\mu}{\beta}\right) \frac{Ce^{(\beta - \mu)t}}{1 + Ce^{(\beta - \mu)t}}$	$i(t) = \frac{i_0 \exp(\beta t)}{1 - i_0 + i_0 \exp(\beta t)}$
2	<b>Late Behaviour</b> Saturation at t → ∞	$i(t) \rightarrow 1$	$i(t) \rightarrow 1 - \frac{\mu}{\beta}$
3	<b>Epidemic Threshold</b> Disease not always spread	No Threshold	$\lambda_c = 1$

Recap: Basic Features of Epidemic Models

# **Epidemics on Networks**



# **Topology matters**

The described approaches assumed *homogenous mixing*, which means that each individual can infect *any* other individual.

In reality, epidemics spread along *links in a* network: we need to explicitly account for the role of the network in the epidemic process.



## **Modeling choices**

### Degree based representation:

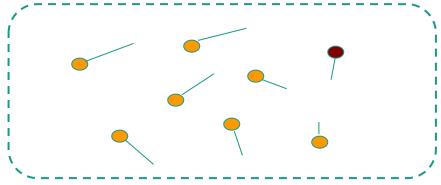
split nodes by degree

$$i_k = \frac{I_k}{N_k}, \quad i = \sum_k P(k)i_k$$

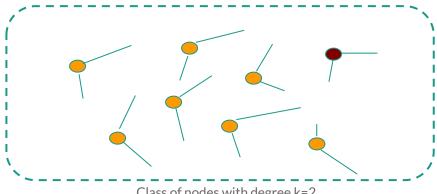
### **Example SIS:**

I am susceptible with k neighbors, and  $\Theta_{k}(t)$  of my neighbors are infected.

$$rac{di_k(t)}{dt} = eta(1-i_k(t))k\Theta_k(t) - \mu i_k(t)$$
Proportional to k Density of infected neighbors of nodes with degree  $k$ 



Class of nodes with degree k=1



Class of nodes with degree k=2

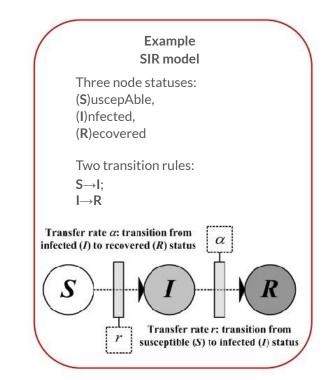
## **Modeling choices**

### Agent based representation:

Each node is an agent having a current status (S/I/R...) and subject to probabilistic transition rules

### **Example SIR:**

- Current node status S:
  Applicable rules: S→I
  If at least one of my neighbors is infected, with probability β change my status to infected.
- Current node status I:
   Applicable rules: I→R
   With probability μ turn my status to removed.



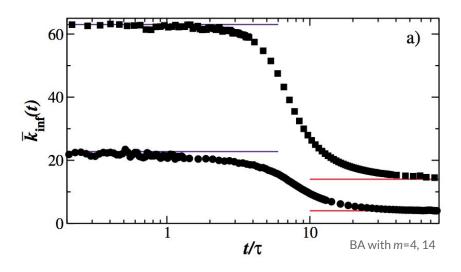
### SI Model

## Early time behaviour

$$au = rac{\langle k 
angle}{etaig(ig\langle k^2ig
angle - \langle k 
angleig)}$$

The timescale it takes for an epidemics to grow. The smaller is T, the faster it grows.

ER model	BA model
<k<sup>2&gt;=<k>(<k>-1)</k></k></k<sup>	<k²> □ ∞ for N □ ∞ □ τ □ 0</k²>
The more connected the network is, the faster does the epidemic spread.	The characteristic time vanishes: the epidemic becomes instantaneous.
	Reason: the hubs get infected first, which then rapidly reach most nodes.



Numerical Test:

The average degree of newly infected nodes at

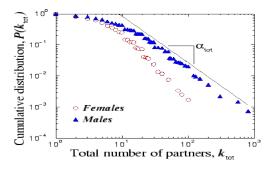
$$\bar{k}_{\inf}(t) = \frac{\sum_{k} k (I_k(t) - I_k(t-1))}{I(t) - I(t-1)}$$

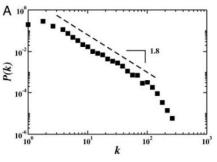
## SIS Model

## No Epidemic Threshold

Many networks will have small or vanishing epidemic threshold.

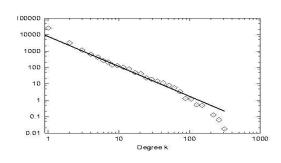
Diffusion will not die out.







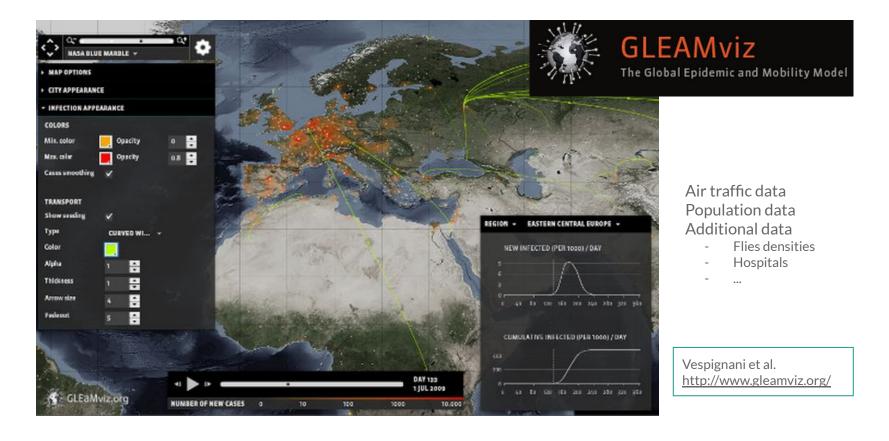
Air Transportation network



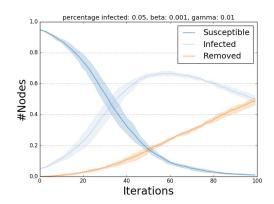
Email network

# Summarizing

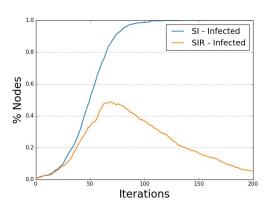




GleamViz: leveraging mobility networks







**Diffusion Models** 

12+5

**Epidemics & Opinion Dynamics** 

pip install ndlib

User Base

~20.000

Installations (2019-Q1 only)

Research impact

20

Publications citing NDlib since 2018 (First release 12/2017)





### Chapter 13

## Conclusion

### **Take Away Messages**

- 1. Viruses spread over a population
- 2. Populations can be modeled with social networks topologies
- Stochastic epidemic models can be leveraged to simulate and reason upon real world viruses diffusion

### **Suggested Readings**

- Chapter 21 of Kleinberg's book
- Chapter 10 of Barabasi's book

### What's Next

Chapter 14:

**Diffusion: Opinion Dynamics** 

