Chapter 14

Diffusion: Epidemics

Summary

- Probabilistic Epidemic Models
 - o 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

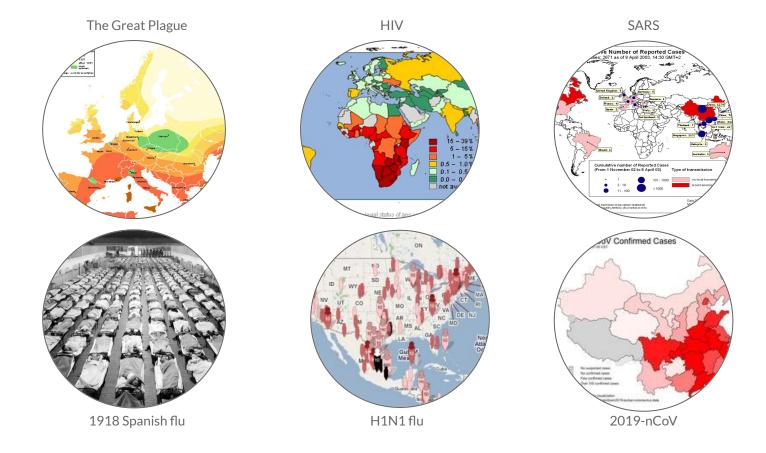
Conceptual/Intellectual:

- Diffusion of innovations
- Rumors
- Memes
- Business practices

Epi + demos

upon people

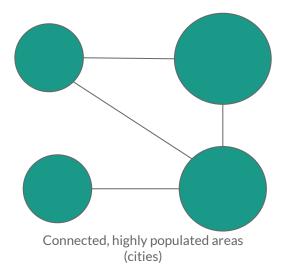




Biological: Notable Epidemic Outbreaks



Separate, small population (hunter-gatherer society, wild animals)



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

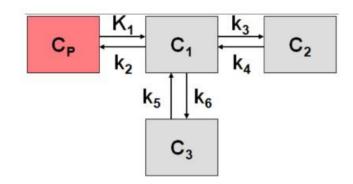
The framework is based on two hypotheses:

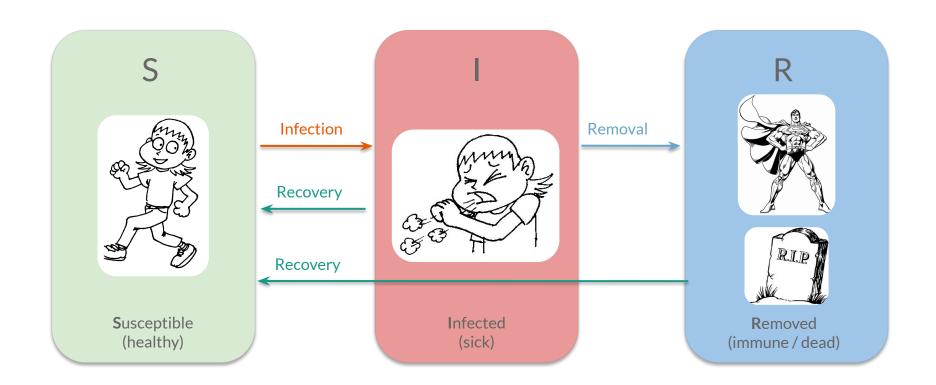
Compartmentalization:

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

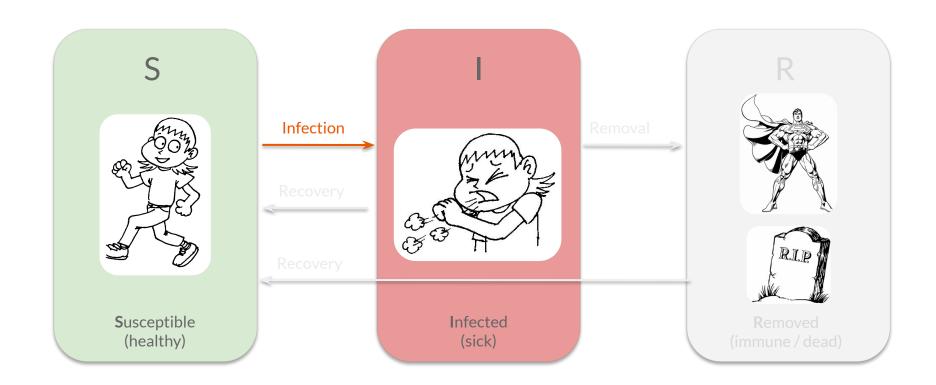
Homogeneous Mixing:

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

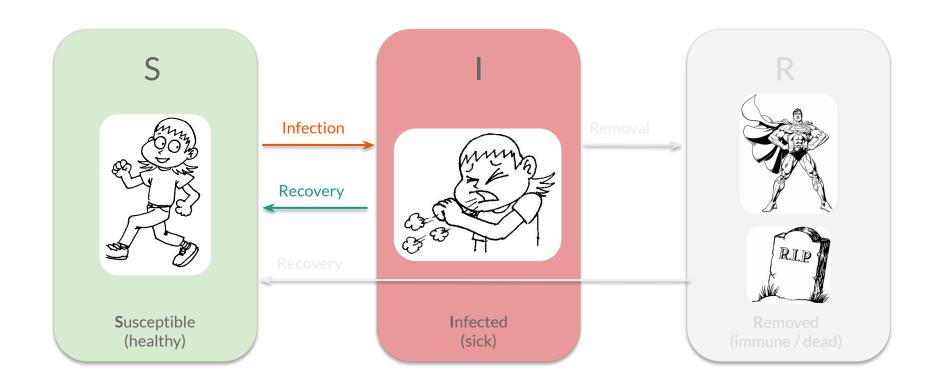




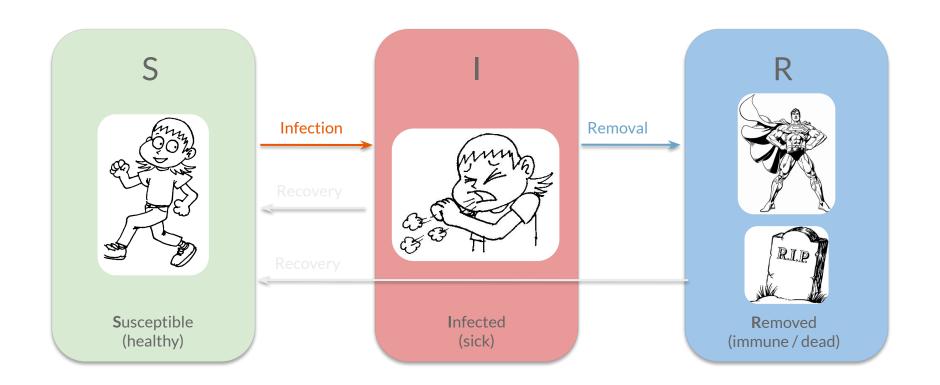
Classic Models Compartments



SI: The simplest model



SIS: Common Cold



SIR: Flu, SARS, Plague

Mean Field formulation

(Homogeneous mixing)

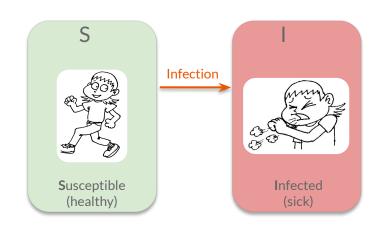


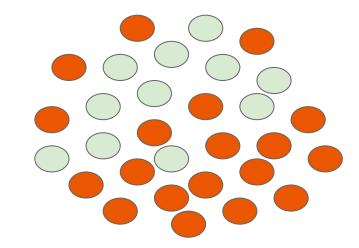
Each individual has β contacts with randomly chosen others individuals per unit time.

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

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

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





Dynamics

Logistic equation: a basic model of population growth.

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

http://en.wikipedia.org/wiki/Logistic_function http://mathworld.wolfram.com/LogisticEquation.html

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

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

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

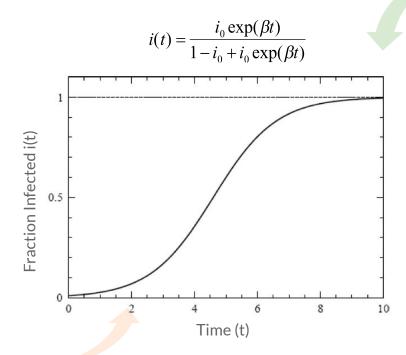
$$\therefore i(t) = \frac{i_0 \exp(\beta t)}{1 - i_0 + i_0 \exp(\beta t)}$$

Behaviour

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

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

exponential outbreak



As i(t)~ 1.

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

saturation

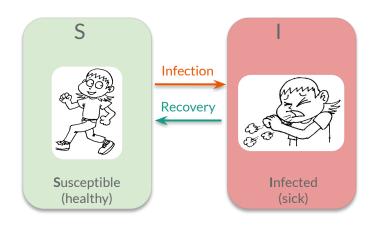
SI model:

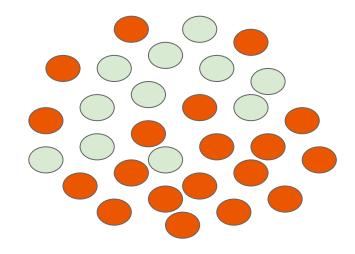
the fraction infected increases until everyone is infected.

Modeling Common Cold

Each individual has β contacts with randomly chosen others individuals per unit time.

Each infected individual has μ probability of revert its status to susceptible





Behaviour

$$\frac{di}{dt} = \frac{\beta i(1-i) - \mu i = i(\beta - \mu - \beta i)}{\frac{1}{100}}$$

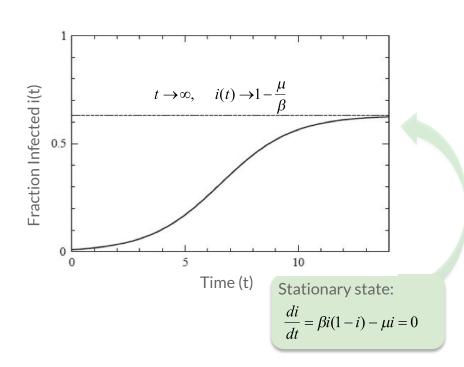
$$\frac{di}{i} + \frac{di}{1 - \mu/\beta - i} = (\beta - \mu)dt$$

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

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

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

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

Basic ReproductiveNumber

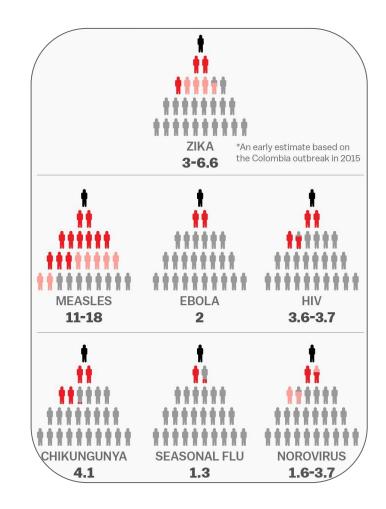
 \hbar (also identified with R₀): average # of infectious individuals generated by one infected in a fully susceptible population.

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

እ > 1: Outbreack

አ < 1: Die Out

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

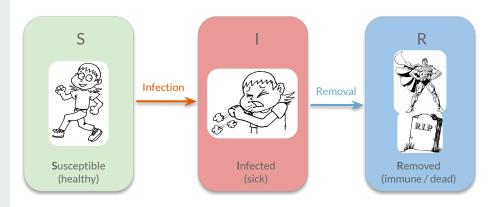


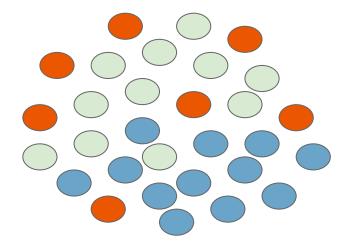
SIR model

Modeling Flu-like disease

Each individual has β contacts with randomly chosen others individuals per unit time.

Each infected individual has μ probability of becoming immune after being infected





Behaviour

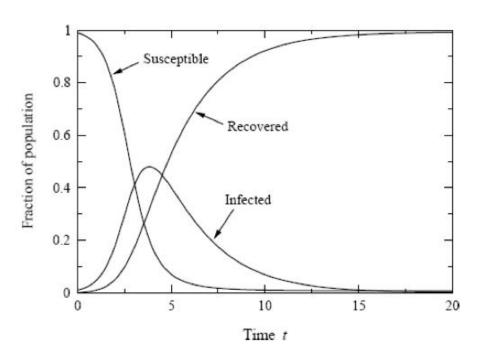
$$\frac{\mathrm{d}s(t)}{\mathrm{d}t} = \beta \langle k \rangle i(t) \left[1 - r(t) - i(t) \right]$$

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

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

SIR model:

the fraction infected peaks and the fraction recovered saturates.



		SI	SIS
1	Early Behaviour Exponential growth of infected individuals	$i(t) = \frac{i_0 \exp(\beta t)}{1 - i_0 + i_0 \exp(\beta t)}$	$i(t) = \left(1 - \frac{\mu}{\beta}\right) \frac{Ce^{(\beta - \mu)t}}{1 + Ce^{(\beta - \mu)t}}$
2	Late Behaviour Saturation at t → ∞	$i(t) \rightarrow 1$	$i(t) \rightarrow 1 - \frac{\mu}{\beta}$
3	Epidemic Threshold 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:

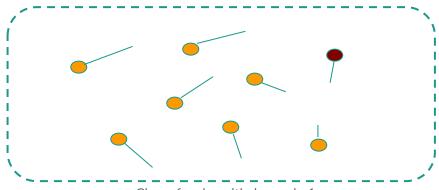
split nodes by degree

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

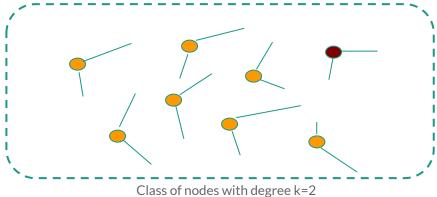
Example SIS:

I am susceptible with k neighbors, and $\Theta_{l}(t)$ 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



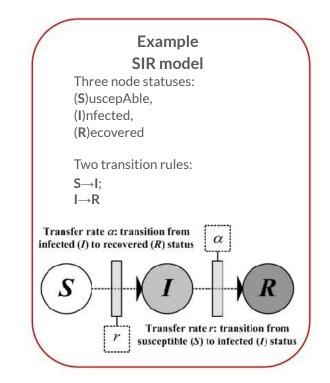
Modeling choices

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.



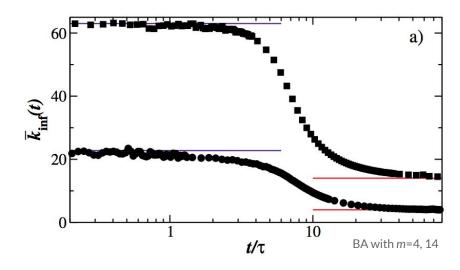
SI Model

Early time behaviour

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

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

ER model	BA model
<k<sup>2>=<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

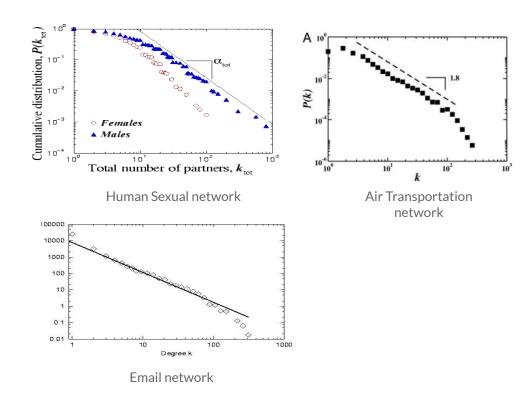
time t:
$$\overline{k}_{\inf}(t) = \frac{\sum_{k} k \left(I_k(t) - I_k(t-1)\right)}{I(t) - I(t-1)}$$

SIS Model

No Epidemic Threshold

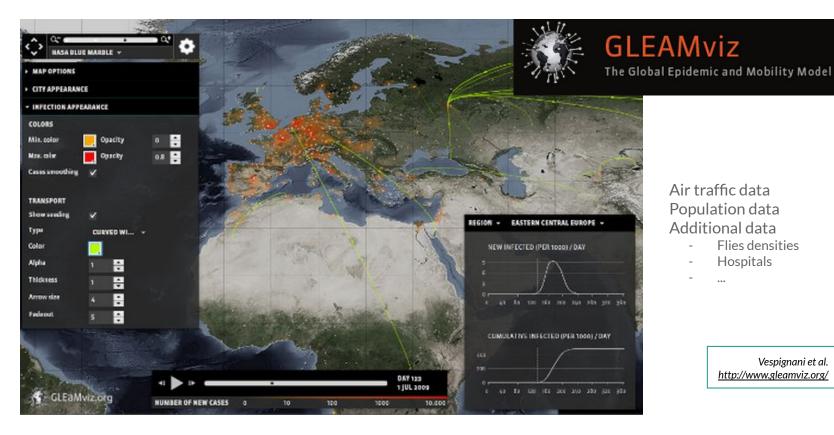
Many networks will have small or vanishing epidemic threshold.

Diffusion will not die out.



Summarizing



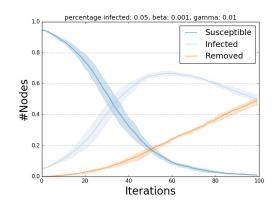


Air traffic data Population data Additional data

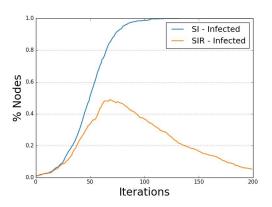
- Flies densities
- Hospitals

Vespignani et al. http://www.gleamviz.org/

GleamViz: leveraging mobility networks







Diffusion Models

12+5

Epidemics & Opinion
Dynamics

pip install ndlib

User Base

~60k

Installations (2021-Q1 only)

Research impact

33

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
- 3. 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 15: **Diffusion: Opinion Dynamics**

