1.022 Introduction to Network Models

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Lecture 20
Models of diffusion with network structure

- Networked Susceptible-Infected-Susceptible (SIS) Model: In a general network, nodes can become infected and then recover in such a way that they become susceptible again.

Models of diffusion without network structure

- The classic SIS Epidemic Model: Individuals can become infected and then recover in such a way that they become susceptible again (the underlying structure is a complete graph).
- The classic Susceptible-Infected-Removed Epidemic (SIR) Model: the diffusion takes place between infected nodes and susceptible nodes over a complete graph. Once a node reaches the “removed” state, it has either recovered and is no longer susceptible or contagious, or it has died.

Optional Readings:
Ch. 21 [Easley-Kleinberg]
C. 17 [M.E.J. Newman]
Ch. 7.1 and 7.2 [Matthew O. Jackson]
We are interested in the following questions:

- Under what conditions will an initial outbreak spread to a nontrivial portion of the population?
- What percentage of the population will eventually become infected?
- What is the effect of immunization policies?
- How do contagions spread in populations?
- Will a disease become an epidemic?
- Who are the best people to vaccinate?
- Will a given YouTube video go viral?
- What individuals should we market to for maximizing product penetration?
Epidemics

- Epidemiology is where Biology meets Social science
  - McKendrick & Kermack (1927): “Mathematical Theory of Epidemics”
- Spread of an epidemic depends on the pathogen carrying it
  - As well as the “contagion” network structure
- Goal of studying epidemics
  - Understand how to outbreaks happen
  - Use it to design intervention to curb / prevent the outbreak
- Similar to “information spreading”
  - Spread of opinions in society
  - Adoption of new technology
A Networked Susceptible-Infected-Susceptible Model

- SIS epidemic model over a network with $n$ agents
  - the network of contacts is given by adjacency matrix $A = [a_{ij}]$
  - the set of the neighbors of node $i$: $N_i = \{j \mid a_{ij} \neq 0\}$
  - probability of contact between individuals $i$ and $j$ during the time interval $[t, t + \Delta)$: $\Delta a_{ij}$.

- Each individual (or node) is in one of the two states $X_i(t) \in \{0, 1\}$
  - **Susceptible**: the state $X_i(t) = 0$ means that node $i$ doesn’t have the disease at time $t$, but could catch it via contact
  - **Infected**: the state $X_i(t) = 1$ means that node $i$ has the disease at time $t$; can pass it on to susceptible via contact
A Networked SIS Model (continued)

- Dynamics
  - a susceptible individual comes in contact with his/her neighbor
  - if the neighbor is infected, susceptible becomes infected
  - infected nodes recover in such a way that they become susceptible again

- Each node can switch to the infected state during the time interval \([t, t + \Delta]\) with a probability that depends on:
  - an infection rate \(\beta\)
  - the probability of contact with a neighbor in this interval \((\Delta a_{ij})\)
  - their states
Probability of transition from susceptible to infected:
\[
\Pr[X_i(t + \Delta) = 1|X_j(t) = 1, X_i(t) = 0, X_k(t) = 0 \text{ for all } k \neq j] = \beta \Delta a_{ij}.
\]

Hence,
\[
\Pr[X_i(t + \Delta) = 0|X_j(t) = 1, X_i(t) = 0, X_k(t) = 0 \text{ for all } k \neq j] = 1 - \beta \Delta a_{ij}
\]

Let \(X(t) := (X_1(t), \ldots, X_n(t))^\top\) be the state vector:
\[
\Pr[X_i(t + \Delta) = 0|X_i(t) = 0, X(t)] = \prod_{j \in \{j|X_j(t) = 1\}} (1 - \beta \Delta a_{ij})
\]

Finally
\[
\Rightarrow \Pr[X_i(t + \Delta) = 1|X_i(t) = 0, X(t)] = 1 - \prod_{j \in \{j|X_j(t) = 1\}} (1 - \beta \Delta a_{ij})
\]
Using the first-order approximation ( $\Delta \ll 1$)

$$\Pr[X_i(t + \Delta) = 1|X_i(t) = 0, X(t)] = \sum_{j \in N_i} \beta \Delta a_{ij} X_j(t)$$

Will see in your project!

Assume node $i$ is infected, the probability of $i$ recovering back to the susceptible state in the time interval $[t, t + \Delta)$ is given by

$$\Pr[X_i(t + \Delta) = 0|X_i(t) = 1] = \Delta \gamma$$

$0 \leq \gamma \leq 1$ is the curing rate.
A Networked SIS Model (continued)

This spread model is still hard to analyze for large-scale networks (it has \(2^n\) states). One standard approach is to use a mean-field approximation.

- Transitional probabilities:

\[
\Pr[X_i(t + \Delta) = 1 | X_i(t) = 0, X(t)] = \sum_{j \in N_i} \beta \Delta a_{ij} X_j(t)
\]

\[
\Pr[X_i(t + \Delta) = 1 | X_i(t) = 1] = 1 - \Delta \gamma
\]

- Define \(p_i(t) = \Pr[X_i(t) = 1] = \mathbb{E}[X_i(t)]\). Then, applying Bayes rule

\[
p_i(t + \Delta) \approx p_i(t)(1 - \Delta \gamma) + (1 - p_i(t)) \sum_{j \in N_i} \beta \Delta a_{ij} p_j(t)
\]

- Shifting \(\Delta \to 0\), the dynamics of \(p_i(t)\) can be written as

\[
\frac{dp_i(t)}{dt} = \beta (1 - p_i(t)) \sum_{j=1}^{n} a_{ij} p_j(t) - \gamma p_i(t)
\]

This approximation is widely used in the field of epidemic analysis and control, since it performs numerically well for many realistic network topologies.
In this example, we consider the epidemic model on a 2-d square lattice; each cell can infect any of the eight immediate neighbors.

The color reflects $p_i(t)$. 

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Example: Spatial Epidemic Model Simulation
In this example, we consider the epidemic model on a 2-d square lattice; each cell can infect any of the eight immediate neighbors.

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Full mixing in classic Epidemiological models

- **Full mixing assumption**

  In classic epidemiology, it is assumed that each individual can potentially have contact with any other in the population (= complete underlying graph).

Image by David Benbennick. Source: [Wikimedia Commons](https://commons.wikimedia.org). This image is in the public domain.
Traditional approach in mathematical epidemiology: *no contact network*

- *fully mixed*: each individual contacts any other random individual

That is, contact graph is *complete*

- due to symmetry, we can study it more carefully

Using the full mixing assumption \( a_{ij} = 1/(n-1) \) for \( i \neq j \) and the dynamics:

\[
\frac{dp_i(t)}{dt} = \beta (1 - p_i(t)) \sum_{j=1}^{n} a_{ij} p_j(t) - \gamma p_i(t)
\]

it follows that

\[
\frac{dp_i(t)}{dt} = \beta (1 - p_i(t)) p_i(t) - \gamma p_i(t)
\]

Because of the full mixing assumption and symmetry, all \( p_i \)'s are the same.
\[
\frac{dp_i(t)}{dt} = \beta (1 - p_i(t)) p_i(t) - \gamma p_i(t)
\]

- \(p_i(t)\) and \(1 - p_i(t)\) are the fraction of susceptible and infected individuals

- Let’s define \(x(t) := 1 - p_i(t)\) and \(s(t) := p_i(t)\)

- Then, we get the exact classic SIS model

\[
\frac{ds(t)}{dt} = \gamma x(t) - \beta s(t)x(t),
\]
\[
\frac{dx(t)}{dt} = \beta s(t)x(t) - \gamma x(t), \quad s(t) + x(t) = 1.
\]
The size of infection is given by

$$\frac{dx(t)}{dt} = \beta(1 - x(t))x(t) - \gamma x(t).$$

Rearranging the terms we get

$$\frac{dx(t)}{\beta(1 - x(t))x(t) - \gamma x(t)} = dt.$$

Integrating both sides and after some simplifications we arrive at

$$x(t) = \frac{\beta - \gamma}{\beta + (\beta - \gamma)Ce^{-(\beta-\gamma)t}}$$

where

$$C = \frac{(\beta - \gamma) - \beta x(0)}{(\beta - \gamma)x(0)}$$
The size of infection \( x(t) \) is thus

\[
x(t) = x(0) \frac{(\beta - \gamma)e^{(\beta-\gamma)t}}{\beta - \gamma + \beta x(0)(e^{(\beta-\gamma)t} - 1)}
\]

Assuming \( \beta > \gamma \), the steady state value of \( x(t) \) is \( (\beta - \gamma)/\beta \), which is called an endemic state.

The fraction of infected individuals in the SIS model grows with time following a logistic curve. The fraction infected never reaches unity, tending instead to an intermediate value at which the rates of infection and recovery are balanced.
In the SI model, individuals once infected are infected forever ($\gamma = 0$).

An infected never recovers and stays infected and infectious to others.

The parameters of the SI model are

- $\beta$ infection rate: probability of contagion after contact per unit time
- Zero recovery rate ($\gamma = 0$)!

Dynamics: $s(t)$ and $x(t)$ be the fraction of individuals in susceptible, and infected state

\[
\begin{align*}
\frac{ds(t)}{dt} &= -\beta s(t)x(t), \\
\frac{dx(t)}{dt} &= \beta s(t)x(t)
\end{align*}
\]

where $s(t) + x(t) = 1$ for all $t$. 

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For many real diseases, people recover from infection

Moreover, they retain immunity to the disease after recovery (e.g., chickenpox).

This motivates SIR model

Three states: susceptible, infected, and recovered.

Dynamics: $s(t)$, $x(t)$ and $r(t)$ be the fraction of individuals in susceptible, infected and recovered state

\[
\frac{ds(t)}{dt} = -\beta s(t)x(t),
\]
\[
\frac{dx(t)}{dt} = \beta s(t)x(t) - \gamma x(t),
\]
\[
\frac{dr(t)}{dt} = \gamma x(t),
\]

where $\gamma$ is the recovery rate and $s(t) + x(t) + r(t) = 1$ for all $t$. 


- Solving these equations, we obtain \( \frac{dr(t)}{dt} = \gamma (1 - r - s(0)e^{-\beta r(t)/\gamma}) \).

- Assume \( s(0) \approx 1 \). The steady state value of \( r(t) \) satisfies

\[
r = 1 - e^{-\beta r/\gamma}.
\]

- For \( \beta/\gamma < 1 \), this has a unique solution at \( r = 0 \): there is no epidemic (infected individuals recover faster than susceptible ones become infected).

- The transition between epidemic and non-epidemic regimes happen at the point \( \beta = \gamma \), called the epidemic threshold or transition.