Epidemics on Networks

Analysis of infection spread:

- In theory we should consider all biological processes
- In practice we can only model them because of the complexity of the problem

S(usceptible)-I(nfected) Model

Someone who doesn't have the disease but potentially could catch one if comes into contact with infected individual

Someone who has the disease and can potentially pass it to susceptible individual if they come into contact

Infected

Susceptible

Model 1: mass-action approximation (any individual has equal chances to come into contact with every other) Model 2: network-based (limited possible number of contacts)

Anderson McKendrick 1876-1943 Pioneered mathematical methods in epidemiology



number of individuals in the system S(t)number of susceptible individuals at time tX(t)number of infected individuals at time t $s = S/n, \quad x = X/n$ S, X expectations of S(t) and X(t) if we run the same process many times contact rate per-individual number of susceptible *s+x* = 1 people got infected from one person average rate of new infections is $X \left(\beta \frac{S}{n} \right) \Rightarrow \frac{dX}{dt} = X \beta \frac{S}{n}$ or $\frac{dx}{dt} = \beta (1 - x)x$ New Cases of AIDS in The United States 60000 1 40000 with solution $x(t) = \frac{x_0 e^{\beta t}}{1 - x_0 + x_0 e^{\beta t}}$ logistic growth 20000 curve 1986 1989 1992 1980 1983 t

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n

 β

Year http://www.nlreg.com/aids.htm

SIR Assumption



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SIR Model

Susceptible

Infected

Recovered

number of individuals in the system nS(t)number of susceptible individuals at time tX(t)number of infected individuals at time tR(t)number of recovered individuals at time texpectations of S(t), X(t), R(t)S, X, Rs = S/n,x = X/n, r = R/neliminate x and β contact rate per-individual integrate both recovery rate per-individual parts number of susceptible decreases $\frac{ds}{dt} = -\beta sx$ $\frac{dx}{dt} = \beta sx - \gamma x$ $\frac{1}{s}\frac{ds}{dt} = -\frac{\beta}{\gamma}\frac{dr}{dt} \implies s = s_0 e^{-\frac{\beta r}{\gamma}}$ $x = 1 - s - r \Rightarrow \frac{dr}{dt} = \gamma \left(1 - r - s_0 e^{-\frac{\beta r}{\gamma}} \right)$ $= \gamma x$ $\frac{dr}{dt}$ $r = 1 - s_0 e^{-\frac{\beta r}{\gamma}}$ Remember the size of giant component in Poisson graph? Note: s + x + r = 1in the limit of *n* $S = 1 - e^{-cS}$

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4

SIR Model



Figure 17.2: Time evolution of the SIR model. The three curves in this figure show the fractions of the population in the susceptible, infected, and recovered states as a function of time. The parameters are $\beta = 1$, $\gamma = 0.4$, $s_0 = 0.99$, $x_0 = 0.01$, and $r_0 = 0$.

demo in matlab

SIR Model

Susceptible

The transition between epidemic and non-epidemic regimes happens when

Basic reproduction number is the number of cases one case generates on average over the course of its infectious period

 $\beta = \gamma$

$$R_0 = \beta / \gamma$$

SIS Model

Infected

Recovered

Networks (not full mixing), SIR

Transmission/infection rates have the same meaning but they will work via edges only

Probability (in full mixing) that the individual is still infected after time τ is

$$\lim_{\delta\tau\to 0} (1 - \gamma\delta\tau)^{\tau/\delta\tau} = e^{-\gamma\tau}$$

In the same way the probability that the disease is transmitted

$$\phi = 1 - e^{-\beta\tau}$$

Assumption for networks: every infected individual remains infected for the same length of time, i.e., ϕ is constant.

Consider edge percolation with occupation probability ϕ , i.e., leave edges for disease transmission.

Figure 17.4: Bond percolation. In bond percolation, a fraction ϕ of the edges in a network are filled in or "occupied" at random to create connected clusters of vertices. (a) For small occupation probability ϕ the clusters are small. (b) Above the percolation threshold a large cluster forms, though there are usually still some small clusters as well. (c) When $\phi = 1$ all edges are occupied but the large cluster may still not fill the whole network: at $\phi = 1$ the largest cluster corresponds to the largest component of the network, which is often just a subset of the whole network.

Consider SIR on network with degree distribution p_k

• u - average prob that $i \in V$ is not connected to gc via one particular edge ij, i.e., either ij is unoccupied $(1 - \phi)$ or $j \notin \text{gc}(u^k \text{ is } j \text{ has } k \text{ neighbors})$

$$u = 1 - \phi + \phi \sum_{k=0}^{\infty} q_k u^k = 1 - \phi + \phi g_1(u)$$

gen function of excess deg distribution

• Thus, gc fraction is $S = 1 - \sum_{k=0}^{\infty} p_k u^k = 1 - g_0(u) \bigoplus \phi_c = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}$ in node percolation it was $S = \phi(1 - g_0(u))$

$$\phi = 1 - e^{-\beta\tau} \Rightarrow \beta\tau = -\ln(1 - \phi_c) = \ln\frac{\langle k^2 \rangle - \langle k \rangle}{\langle k^2 \rangle - 2\langle k \rangle}$$

Epidemic threshold if >= then possible epidemic

Epidemic Thresholds in Real Networks

see paper by Chakrabarti, Wang, Wang, Leskovec, Faloutsos (will follow their notation)

Q: How to model viral propagation on arbitrary network? Which node is best to immunize? When is the outbreak?

Homogeneous model – everybody has equal contact to others (rate of infection depends on the density of population). *Nonhomogeneous model* – otherwise.

Kephart-White model: directed graph; $i \to j \in E$ if i can infect j; β is rate of infection (virus birth rate); δ is virus death rate (node-curing). η_t - size of infected population at time t

$$\frac{d\eta_t}{dt} = \beta \langle k \rangle \eta_t \left(1 - \frac{\eta_t}{N} \right) - \delta \eta_t \quad \left(\text{ its steady state } \eta = N \left(1 - \frac{\delta}{\beta \langle k \rangle} \right) \right)$$

Intuition: epidemic threshold τ is a value such that a viral outbreak dies out quickly

$$\beta/\delta < \tau, \ \tau_{KW} = 1/\langle k \rangle$$

good approximation for homogeneous networks

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Consider small time steps $\Delta t \to 0$; the system is a Markov chain with 2^N configurations of nodes S/I.

 $p_{i,t}$ - probability *i* is infected at time *t*

 $\zeta_{i,t}$ - probability that i will not receive infections from N(i) in the next step

$$\zeta_{i,t} = \prod_{j \in N(i)} (p_{j,t-1}(1-\beta) + (1-p_{j,t-1})) = \prod_{j \in N(i)} (1-\beta p_{j,t-1})$$

Non-linear dynamical system

Fig. 2. Experiments on the real-world Oregon graph. The plots show the time evolution of infection in the *Oregon* network. Both simulations were performed with fixed β , but varying δ . In both cases, our model conforms more precisely to the simulation results than the MFA model.

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router views.

Definition (NLDS Epidemic Threshold). The epidemic threshold τ for NLDS is a value such that

 $\beta/\delta < \tau \Rightarrow$ infection dies out over time

 $\beta/\delta > \tau \implies$ infection survives and becomes an epidemic

Theorem 1. In NLDS, the epidemic threshold τ for an undirected graph is

$$\tau = 1/\lambda_{1,A},$$

where $\lambda_{1,A}$ is the largest eigenvalue of the adjacency matrix A. $s = \beta / \delta \cdot \lambda_{1,A}$ is a score of infection.

Theorem 2. When an epidemic is diminishing, i.e., $\beta/\delta < 1/\lambda_{1,A}$, the probability of infection decays at least exponentially over time.

*s>1 - i*nfection survives

Enron database graph (email exchanges) 33K nodes, 361K edges

Immunization

Q: We have a budget of k nodes that can be immunized. How to choose these nodes?

Theorem 1 \rightarrow We need to decrease the maximal eigenvalue of the adjacency matrix.

Observation: Popular "targeted" immunization strategy (immunization of high-degree nodes) does not agree with maximum eigenvalue decrease! Example:

Fig. 8. The "bar-bell" graph. Two cliques of the same size connected with a bridge.

Matlab immunization demo

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Example of a Model: Response to Epidemics and Cyber Attacks

Open Science Grid: collaboration network example

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Multiscale Methods for Networks Ilya Safro, Clemson University