Lecture Notes: Social Networks: Models, Algorithms, and Applications
Lecture 1: Mar 27, 2012
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1 Plan for today

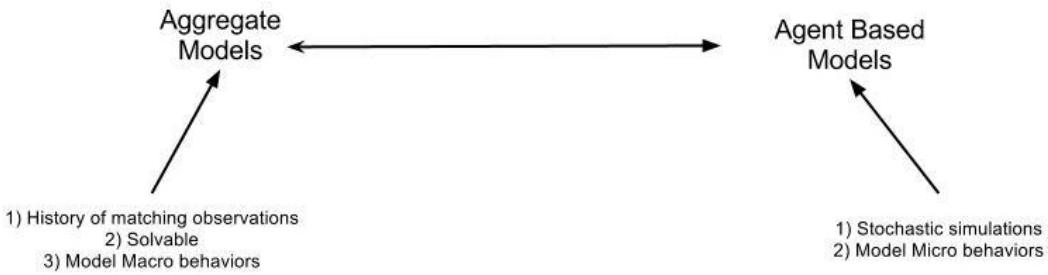
Network Models:

- Agent Based Models.
- Differential Equations Models - DE Models

If we look on a real epidemic data, we will see that these model are fairly good in showing of shapes of epidemic curves. They seem to correspond reality.

Agent Based Models describe the method with which the most network models are used. In other words we run a stochastic simulations and see what happens. And we run this model over and over again, so we get an aggregate of different unfolding of the epidemic. These techniques are usually called Agent Based Models or Stochastic Simulations.

If we think about Homogeneous Models and Differential Equations Models disease can be solved or numerically approximated.
Advantages of Network Models or in other words Agent Based Models
Explicit contact model that reflects our ideas about disease diffusion. Differential Equations Models



What do we need to know for a network model?

1. Content Structure
   (a) Is it time dependent? (This depends on pathogen and any intervention)
   (b) Is it symmetric?
2. Disease Parameters
   (a) How long does it last?
   (b) How long is it infectious?

(c) What is the probability of transmission?

i. Susceptibility

ii. Does it depend on time?

3. Run Simulations. Good experimental design

(a) How many replicas to run?

(b) What to report?

The idea that we are going to work today is that both sides are working towards the middle.

2 Building epidemiological models from R_0: an implicit treatment of transmission in networks.[1]

Abstract:
Simple deterministic models are still at the core of theoretical epidemiology despite the increasing evidence for the importance of contact networks underlying transmission at the individual level. These mean-field or 'compartmental' models based on homogeneous mixing have made, and continue to make, important contributions to the epidemiology and the ecology of infectious diseases but fail to reproduce many of the features observed for disease spread in contact networks. In this work, we show that it is possible to incorporate the important effects of network structure on disease spread with a mean-field model derived from individual level considerations. We propose that the fundamental number known as the basic reproductive number of the disease, R_0, which is typically derived as a threshold quantity, be used instead as a central parameter to construct the model from. We show that reliable estimates of individual level parameters can replace a detailed knowledge of network structure, which in general may be difficult to obtain. We illustrate the proposed model with small world networks and the classical example of susceptible-infected-recovered (SIR) epidemics

Their approach is to make DE model less dependent on homogeneity. There were particularly interested in showing how DE models could approximate results of network-explicit models.

And to do that they have looked on two graphs:

1. Erdos-Renyi graph.[1]

2. Watts-Strogatz graph.[2]

They have realized that R_0 is the key parameter in DE models and recall that R_0 "falls out" of the model.
So, they have used different approach:

[1] From now ER Graph.
[2] From now WS Graph.

Instead of letting R_0 be the parameter that "falls out" this model, what happens if we instead run stochastic simulations and then try to fit R_0 into stochastic simulations to get the DE Model. So they started with a network model on random graphs of different types, the run the simulations, got values and fit DE model to the output of the Agent Based Model and looked what that tells.

From the paper R_0 is the 1^{st} level infection produced. In fact as the epidemic progresses $\frac{s}{n}$ gets smaller (s: susceptible, n: population)[3]. Would you expect this number to be fixed? You have to see it as necessarily getting smaller. And this is the phenomena they[4] called - "saturation".

Now let us think for a minute what happens in Watts-Strogatz model: In Watts-Strogatz model we have a local connected regions with a few long distance edges. In this model we can get an effect that is called - "local saturation". In other words, the chance to infect somebody is going to depend from the saturation of the local area.
Therefore, R_0 becomes a "mixture model" that depends whether saturation is a local region or there is another pool of people that can be infected.

For example if you look on Watt-Strogatz "disorder parameter", where the value is between 0 and 1.[5] If this parameter ≈ 0, then there is no much difference than Erdos-Renyi graph epidemics, but if it is ≈ 1, it doesn't get the initial exponential growth as in Erdos-Renyi model.
Please recall that in Erdos-Renyi model epidemics growth exponentially.

In this paper they have spent a lot of time trying to derive a DE model using R_0 function fir to data. This is an alternative approach to get around not having good contact data, but incorporating some network effects.

[3] This is assuming SI model.
[4] Aparicio and Pascual.
[5] Probability that an edge is rewired.

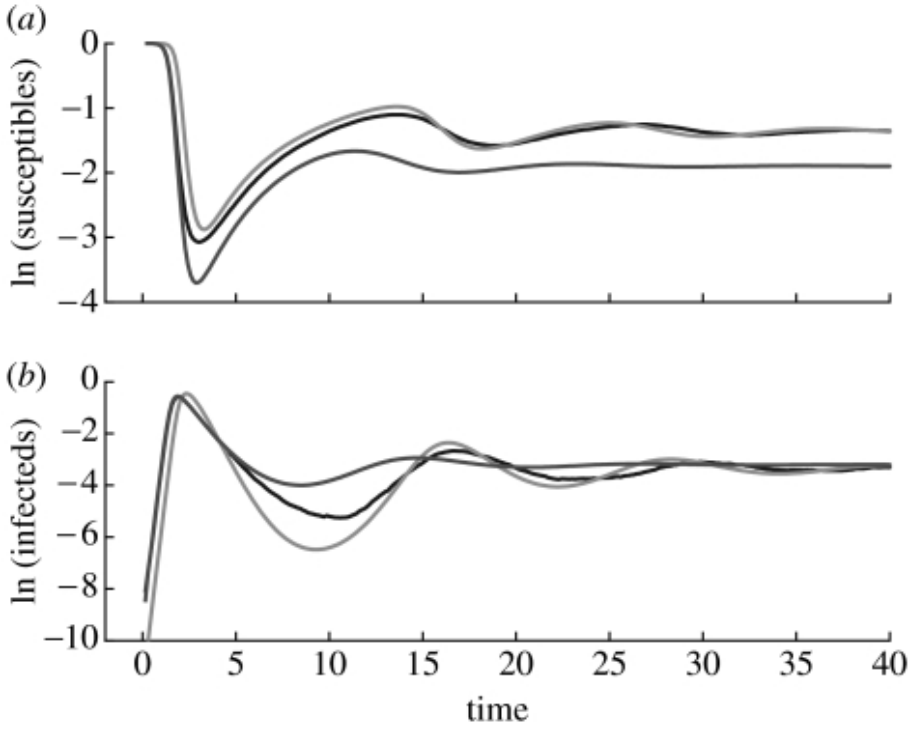


Figure 1: Comparison of the population dynamics of (a) susceptible and (b) infected proportions for the stochastic model of transmission in a Poisson random network and two mean-field models. The blue lines correspond to a stochastic simulation (with a total number of individuals N=90 000, a probability of transmission per contact per unit of time τ=1, and a mortality rate μ=0.05).

3 When individual behavior matters: homogeneous and network models in epidemiology[2]

Abstract:
Heterogeneity in host contact patterns profoundly shapes population-level disease dynamics. Many epidemiological models make simplifying assumptions about the patterns of disease-causing interactions among hosts. In particular, homogeneous-mixing models assume that all hosts have identical rates of disease-causing contacts. In recent years, several network-based approaches have been developed to explicitly model heterogeneity in host contact patterns. Here, we use a network perspective to quantify the extent to which real populations depart from the homogeneous-mixing assumption, in terms of both the underlying network structure and the resulting epidemiological dynamics. We find that human contact patterns are indeed more heterogeneous than assumed by homogeneous-mixing models, but are not as variable as some have speculated. We then evaluate a variety of methodologies for incorporating contact heterogeneity, including network-based models and several modifications to the simple SIR compartmental model. We conclude that the homogeneous-mixing compartmental model is appropriate when host populations are nearly homogeneous, and can be modified effectively for a few classes of non-homogeneous networks. In

general, however, network models are more intuitive and accurate for predicting disease spread through heterogeneous host populations.

Bansal, S., B.T. Grenfell, L.A. Meyers in their research asked a very interesting question: "How far can I go before individual behaviour matters?"
And by how far can I go it means how complicated, and how much change can I make to DE model that we have, until there is nothing I can do to make it keep up with the observed behaviour of the Agent Based model?

For their research they have used 3 types of graphs

- Regular lattices with uniform degree.
- ER .
- WS.

And they tried to map DE models to these 3 types of graphs. And they have find out that it is hard to model individual diversity with population-based models.

In their research they introduced also a contact-network world and pointed that in the real world Agent Based models use "proxy networks".

And they gave some examples.

- It is possible to trace individual infection post-hoc[6]
- Use survey and demographic data.
- Use census data or other sociometric data.

Their methodology was to:

- Map DE to random graphs with some degree K.
- Map DE to complete graph with degree N-1.

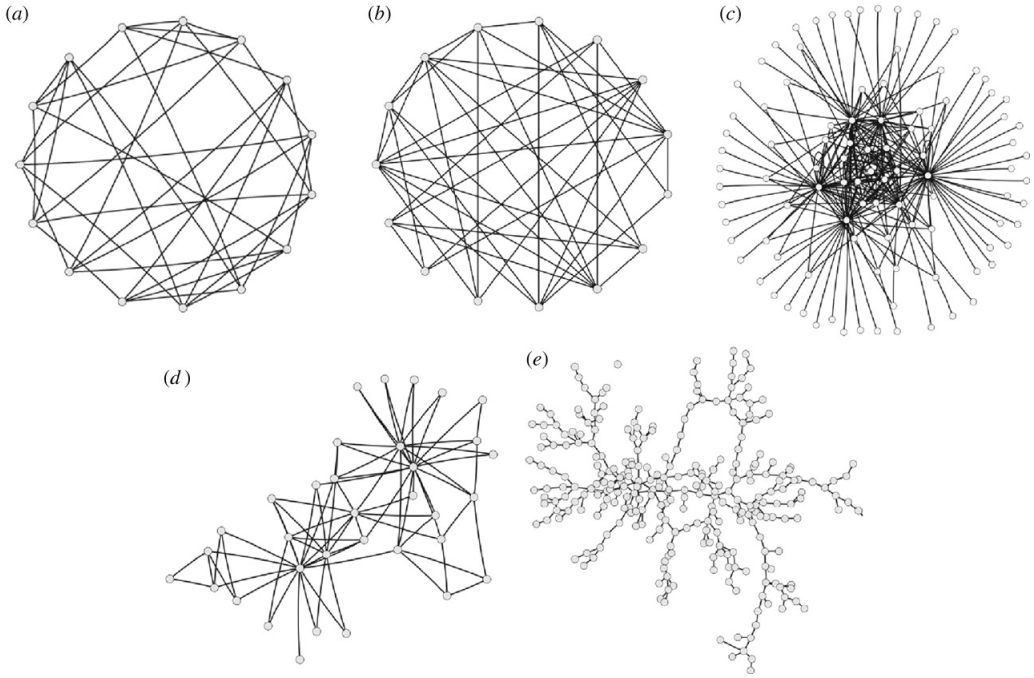
4 Percolation models

Next thing they did in the paper: They looked at 6 real networks and examined how these different models approximated the Agent Based simulations.
6 Data networks:

- Vancouver, BC - Demographic network.
- Portland, OR - Demographic network AND survey info.
- Karate club - small network of social interaction.

[6]This is also called contact-tracing.

- 4-6 Different sexual contact networks.



Examples of
(a) a regular random network with 15 nodes and mean=5
(b) a Poisson random graph with 15 nodes and mean=5,
(c) a scale-free random graph with 100 nodes and mean=5,
(d) the Zachary Karate Club contact network (Zachary 1977) with 34 nodes and mean≈5 and
(e) the sexual network for adolescents in a Midwestern US town, with 287 nodes and mean≈2.
They have realized that the real contact network tend to have an exponential degree distribution.

Another thing that they wanted to know is: "How far can I "push" the network structure to get behaviour to deviate from that of random mixing?".

They came up with a nice idea.They wanted to quantify the distance[7] between the behaviour of the exponential network and homogeneous network model. So, they have started with a random network and they used the rewiring procedure[8] and turned the random network to an exponential network. They decreased the degree of the original node and increased the degree of the destination node.
They defined a parameter that they called a Coefficient of Variation - CV

$$\text{CV}=\frac{Std\ dev\ degree}{\mu\ degree}$$

So, as CV$\rightarrow$ 1, we get an exponential distribution.
The CV and this measures how much work they have to do to rewire the network. Basically, CV=1 means that every one has been rewired.

[7] difference
[8] Like in Watts-Strogatz Model

References

[1] Aparicio JP, Pascual M.. *Building epidemiological models from R_0: an implicit treatment of transmission in networks.* 2007: Proc Biol Sci. 2007 505-12.

[2] Bansal, S., B.T. Grenfell, L.A. Meyers *MWhen individual behavior matters: homogeneous and network models in epidemiology.* 2007: Journal of the Royal Society Interface 4: 879-891