CSE 8803 EPI: Data Science for Epidemiology, Fall 2022

Lecturer: B. Aditya Prakash
Scribe: MH Seabolt, RG Bauer

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Lecture 5 : Other Contagion Models

1 Summary : Contagions Everywhere!

This lecture covers extensions to classic SIR epidemic modles, such as SIS, SIRS, and SEIR, as well as a range of other models which have been developed to capture additional layers of complexity which traditional SIR is unable to capture or makes simplifying assumptions that lead to unsuitable modelling predictions. Examples of these other models include the Independent Cascade model, SpikeM models, and the classic Lotka-Volterra (Predator-Prey) model. Scenarios in which each of these models may be most suited for use are briefly discussed.

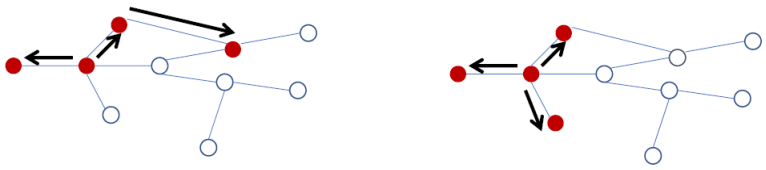

Figure 1: Contagions everywhere! Image from Slide 3 in Lecture 5

2 Continuation from previous lecture: SIR models and Extensions to SIR

SIR models are simple and intuitive in nature, but often make simplifying assumptions about the model characteristics that do not capture the range of potential factors influencing real world behaviors. To better capture these elements while retaining the intuitiveness of SIR, a substantial number of extended models based on SIR have been developed. Examples of these include SIS (Susceptible-Infected-Susceptible), SEIR (Susceptible-Exposed-Infected-Recovered), and SIRS (Susceptible-Infected-Recovered-Susceptible). The primary differences between these variations are how the compartments are initialized, how the transitions between states occur, and the additional layers of complexity used as input to compute the reproductive number R_0. For example, in the SIRS and SIS models people who have been infected can get better and be infected again. In the SEIR model, a new compartment E is introduced to capture an exposure or incubation period prior to being recognized as infected – potential or probably infections do not immediately transition from the S to I compartments.

2.1 Calibrating SIR network models

Obtaining a suitably appropriate network is the primary importance for network-based models, since incorrect networks will always produce incorrect SIR curves. In the case of differential equation-based (ODE) models, only the hyperparameters β and γ need to

be inferred, whereas networks require both β and γ hyperparameters plus the correct construction of the network itself. One example of network construction might be the boroughs of London example where the geographical proximity of neighboring boroughs is used to model an SIR network. ODE-based SIR/SIS models make the simplifying assumption of constant homogeneous population mixing, which can be approximated in network models as "cliques", which are complete graphs in which all nodes share an edge with all other nodes in the graph. More complex model heterogeneity can be achieved as the connectivity, betweenness, and centrality of the network changes.

3 Other Types of Epidemic Models

3.1 Metapopulation Models

All models have trade-offs as the complexity (i.e. the number of model paramters) increases, particularly in relation to the model's transparency and accuracy. Epidemiological models like SIR et al. have found many uses in other realms such as social collaboration initiatives, information diffusion, viral marketing strategies, cybersecurity, gaming, and in the life sciences such as public health, personalized medicine, and ecology.

3.2 Independent Cascade (IC) Models

A special case of the network-based SIR model that is particularly useful for modeling "viral" spread (in the context of social media, not *sensu stricto* public health). Independent Cascade models a social network with directed, weighted edges which reflect, for example, individual pairwise probabilities of 'infection' spread from node u to node v. This model is reminiscent of the Twitter "following" system where connections are directed, i.e. Person A may "follow" Person B while Person B may not follow Person A. The IC model aims to capture the "spread of infection", here referring to the amount of interactions, precipitated when a node becomes active and fires an event. The active node tries to "infect" each of its out-neighbors, thus cascading the infection forward in time. The odds that this peer-to-peer activation is successful is based upon the probability of the edge weight from (u, v). As each event is a single discrete action, the activated node tries to activate its nodes only once before reverting to an inactive state. In relation to ODE-based SIR models, network-based IC models extend the β rate of transmission from a single universal rate to a pairwise rate along the edge (u, v). Similarly, the recovery hyperparameter δ is understood to be 1.0.

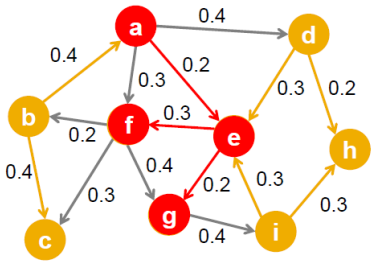

Figure 2: Example SpikeM model diagram. Image from Slide 4 of Lecture 5

One key benefit of the IC model is that it is very intuitive for information flow, however

this also brings with it some drawbacks: the computational complexity increases exponentially as the number of edges in model grows, and the significant number of parameters (e.g. pairwise β, to estimate. These can make it difficult to calibrate IC models accurately.

One feature of this model is that it is that popularity drops exponentially over time. This is since it is based on an SIR model which is inherently exponential. In some real life examples of the spread of knowledge or trends like blog post sharing the drop off is not as steep. In these real life datasets the drop off actually follows a power law with the power -1.6. This allows for rare events and rebounds that are not possible in the exponential decay of the SIR and IC models.

3.3 SpikeM Models

A SpikeM Model is a variation of the classic SIR model that uses the Power Law to calculate β, making it not exponential in the same way as standard SIR. This model works by having a series of 'uninformed' nodes, followed by a random "disturbance" such as -for example- Operation London Bridge, the surprise news report covering the death of Queen Elizabeth II. Following the perturbation event, "infection" can spread in the form of word-of-mouth transmission. This model allows for a resurgent or periodic spread after the initial disturbance event and initial spread of infection subsides. This is because Power Law distributions that have mathematical properties making the subsequent "unlikely" disturbance events in a standard SIR model more likely to occur.

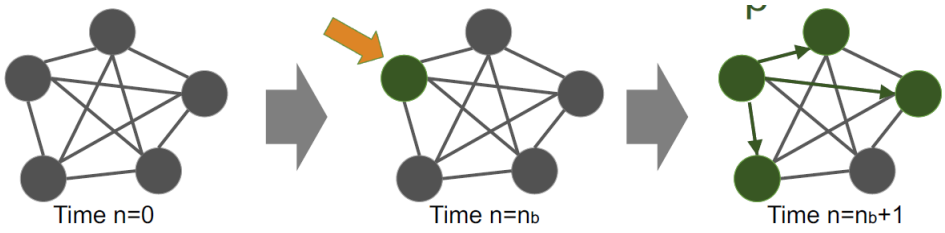

Figure 3: Example SpikeM model diagram. Image from Slide 12 of Lecture 5

3.4 Complex contagions

The idea behind a complex contagion models is that the neighbors of a node expose that node to a transmissible 'contagion'. In a traditional SIR model, the probability of a node becoming infected is $p = 1-(1-\beta)^k$, where k is the number of infected adjacent neighbors. This results in a curve that resembles a Logarithmic or Negative Binomial growth curve and reflects the probability of becoming infected based on the number of infectious neighbors. In effect, this suggests an effect of diminishing returns from the amount of infected neighbors (i.e. the Power Law decay parameter α is less than 0).

Complex contagions may model different behaviors. For example, a sharp rise in the probability of becoming infected once a certain minimum threshold of infectious neighbors is reached, or conversely, a steep drop representing an over-exposure effect such as an individual being "over-advertised" to, which reduces the likelihood of being interested in a product. This type of model may not be suitable for application to many epidemiological scenarios but may be useful for related fields.

3.5 Other types of complex (single contagion) models

Other types of structural models exist which take a different approach to modeling single contagion infection spread based not on individual nodes, but on different levels of granularity, such as a person's social circle, local community, or familial relationships.

Briefly, examples of these models include Decision models which have nodes that each have an activation threshold before the node will activate. An example of this might be at the start of a riot, each person in a crowd may have an activation threshold (e.g. number of disgruntled neighbors beginning to riot) before they join in. Thus, the centrality of nodes in this type of network is a critical factor in infection spread.

Other examples of complex contagion models are voter models where nodes pick an 'ideology' based on their neighbors and it shows how beliefs are picked, or hybrid models where not every node is modeling the same thing. For example one model could show both how things are pick and also how they spread all at once.

3.6 Multiple contagion models

These are extensibly models like the Lotka-Volterra Model, sometimes known as the Predator-Prey model. This model consists of two populations: one of a predator and the other being a prey species. Each of these populations experience fluctiations in population size of time, influenced both by birth/death parameters of their own population (part of each species' carrying capacity) and by the population of the other species. The expected population sizes can be modeled using ODEs and are extensible to any number of competitive species.

The classic Lotka-Volterra model contains 2 species, X and Y, which interact with one another using the following system of equations:

$$\frac{dx}{dt} = \alpha x - \beta xy \tag{1}$$

$$\frac{dx}{dt} = \delta xy - \gamma y \tag{2}$$

where αx and δxy represent the birth rated of Species X and Species Y respectively, and the βxy and γy terms represent the mortality rate of each population. There are two stable equilibria points: one is trivial – the extinction point, where $x = y = 0$. The second one is the co-occurrence point at $x = \frac{\gamma}{\delta}$ and $y = \frac{\alpha}{\beta}$.

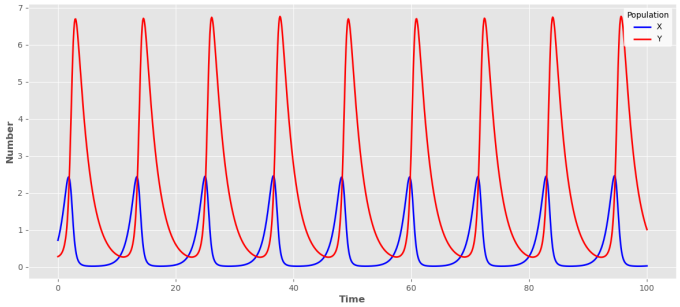

Figure 4: Example Lotka-Volterra model.

3.7 Modelling Extensions

There can also be two competing contagions fighting for the same population in one model. This could be for real world diseases like influenza and SARS-CoV-2 or this could be used for non-epidemiological fields like modeling the spread of Apple vs Android devices. This requires the model to have two β and two γ parameters to model the traits of the two different contagions.

There can also be an ϵ (epsilon) value to model the interaction. This is because sometimes one contagion can block off people from getting another, like getting Cowpox preventing people from getting Smallpox. In such a case, $\epsilon = 0$. Sometimes they only grant partial immunity so getting one makes people less likely to get the other but it is still possible. This is like getting one variant of COVID making it less likely someone gets a different variant but it is still possible to get. This is represented by $0 <= \epsilon <= 1$. There could also be two diseases that are unrelated and do not limit people from getting the other if they already have one. This scenario occurs when $\epsilon = 1$.

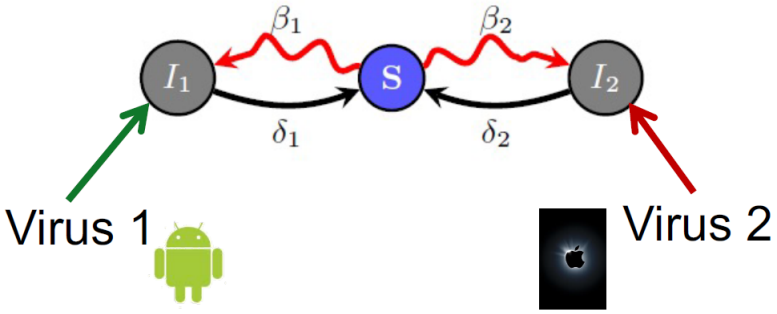

Figure 5: A two-contagion model diagram. Image from Slide 38 of Lecture 5