1 Introduction

Scientific experiments are usually designed to obtain information and to test hypothesis – but how could it be possible to experiment an epidemic? Therefore, mathematical epidemiology has been playing a crucial role in studying the spread and management of diseases.

The basic compartmental models to describe the transmission of communicable diseases are first defined in a sequence of three papers by W.O. Kermack and A.G. McKendrick in 1927, 1932, and 1933 [1]. These models assume that each individual has an equal likelihood of coming into contact with any other individual. However, in reality the patterns of contacts between individuals are highly heterogeneous, and this heterogeneity can be a significant factor in the early spread of a disease. When this is the case, the compartmental model can be insufficient.

In the case of the 2002 SARS outbreak, the basic reproduction number $R_0$ of the disease, or the average number of secondary infections caused by an infected individual, was estimated to be between 2.2 and 3.6. With these values, compartmental models predicted that there should have been approximately between 30,000 and 10 million cases in the first 120 days of transmission in China. However, these predictions turned out to be inaccurate, and only 782 case were reported during the initial three months [5].

In [5], L.A. Meyers and her team attribute this inaccuracy to the assumption of homogeneity of contacts between individuals in compartmental models. They point out that there is an enormous epidemiological difference between a situation where all individuals share similar contact patterns and a situation where most infected individuals pass the disease to only a few others but a small number pass it to dozens or even hundreds, even though mean value of $R_0$ can be the same in both cases.

For this project, we will look into the influence of contact networks on epidemics by building up network models for a past epidemic, and comparing the results with those of compartmental models.

2 Background

2.1 Compartmental Models

Compartmental models are perhaps the most well-known tool in mathematical epidemiology. A compartmental model analyzes an epidemic by dividing the host population into several
compartments, each containing individuals that are identical in terms of their status with respect to the disease in question [1]. The most basic compartmental model is the SIR model, where S stands for the susceptible, I the infectious, and R the removed. Their relationship can be described by the following system of differential equations,

\[
\frac{dS}{dt} = -\beta SI,
\frac{dI}{dt} = \beta SI - \alpha I,
\]

where the transmission rate is $\beta$ and the recovery rate is $\alpha$. The basic SIR model is used when the disease in question confers immunity against re-infection. Many adjustments can be made to adapt the basic SIR model to a specific disease. For example, we can add an $E$ class between the $S$ class and the $I$ class, forming a SEIR model which describes a disease with an exposed period between becoming infected and becoming infectious, or able to transmit the disease.

Notice that $S' < 0$ at any time and $I' > 0$ if and only if $S > \alpha/\beta$. If $S(0) < \alpha/\beta$, $I$ decreases to zero and there is no epidemic. On the other hand, if $S(0) > \alpha/\beta$, $I$ first increases to a maximum value and then decreases to zero, indicating the occurrence of an epidemic. Therefore, the quantity $\beta S(0)/\alpha$, called the basic reproduction number and denoted by $R_0$, is a crucial threshold quantity used to predict whether an epidemic will occur.

The compartmental models simplify the complicated reality by making assumptions that allow us to examine an epidemic macroscopically. However, they don’t perform the best in all the cases. The fact that compartmental models focus on compartments instead of individuals indicates their assumption of random mixing of the population, meaning each individual has an equal likelihood of coming into contact with any other individual. This is obviously not the case in reality, and the error caused by this assumption could be non-negligible, especially at the beginning stage of an epidemic where there are only a few infectives. Thus, network models are introduced to solve this problem.
2.2 Networks

In network models, we refer to individuals of a population as nodes and their connections with others as edges through which disease can spread. Also, the set of contacts of an individual is called its neighborhood, and the size of this neighborhood is called its degree. There exist many different types of network models, and we are going to focus on three of them – random networks, lattices, and small-world networks.

![Random Network, Small-World Network, Lattice](image)

**Figure 2:** Visualization of random network, small-world network, and lattice [3]

2.2.1 Random Networks

In random networks, each individual has a fixed neighborhood of a fixed size and the connections are randomly generated. Due to their randomness, there are no obvious clusters in which individuals are mostly connected but isolated from the rest of the network. In terms of connections, individuals in a random network are no different from each other (homogeneity of individual-level network properties). And yet, however random they are, these networks are still less random than the random-mixing models (compartmental models) because the neighborhood of an individual in a random network is fixed once the edges have been assigned. The result of this is that both the early growth rate of the disease and the final epidemic size are reduced.

2.2.2 Lattices

Contrary to the random networks, the lattices are highly ordered based on different assumptions. In lattice models, connections exist only between adjacent individuals, emphasizing the spatial position of individuals of a population. Thus, contacts are spatially localized, and it requires a long chain of contacts for a disease to spread between two distant individuals. As a result, more clustering is present and the spread of the disease described by lattice models is further slowed down.

2.2.3 Small-World Networks

In reality, both clustering of connections (featured in the lattices) and long-range transmission events (featured in the random networks) are likely to be significant factors in an
epidemic. It is thus natural for us to look for a model in between complete order and complete randomness. This is where small-world networks came into play.

Small-world networks can be formed by adding a small number of random connections to a lattice in such a way that we may tune the degree of randomness of our model. In order to determine whether an epidemic will occur, percolation theory is applied to calculate the threshold parameter values. Assuming each individual is connected with its two nearest neighbors and on average with another \( \phi \) random individuals, then the critical bond percolation probability is \([4]\)

\[
p_c = \frac{\sqrt{1 + 12\phi + 4\phi^2} - 1 - 2\phi}{4\phi} = 1 - 4\phi + O(\phi^2).
\]

If the transmission rate \( \beta \) of the disease exceeds \( p_c \), then an epidemic will occur.

For this project, we are going to utilize the flexibility of the small-world networks to see if we can build up a network model to better simulate what really happened during an epidemic.

3 Proposed Methodology

We will first delve into the “Small-World” Networks, following chapters 2, 3, and 6 of \([6]\). Then we will build up a small-world model for the beginning stage of an infectious disease case which has been well-studied by compartmental models. Potential candidates include SARS and Influenza since they are classic epidemic cases and thus reliable data and parameters are available. During the process, we will experiment with different small-world network models, making adjustments and trying out different parameters according to particular properties of the case being studied. Finally, we will compare our results derived by small-world network models to those by compartmental models in order to gain insights into under what circumstances the small-world network models perform better than the traditional compartmental models.

References


