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Epidemic Spread and Network Connectivity

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ABSTRACT
This paper presents selected historical mortality statistics and analyse their characteristics and trends. Statistics are collated particularly for the pandemics related with cholera, plague, Spanish flu and covid-19 between 1720 -2020. We introduce epidemic and epidemic spreading. Then the dissertation continues with brief introduction and discussion of Epidemic models such as SI, SIS and SIR Epidemic pass-through populations and persists over long time periods. Thus, efficient modeling of the understanding network plays a crucial role in understanding the spread and prevention of an epidemic. Further the mathematical modelling of infectious disease epidemics on network, starting from the simplest Erdos-Renyi random graphs, Percolation on graph and epidemics is studied. We also show empirical results of applying the models to calculate the spread of contagion and information connectivity on two complex networks suitable for the models. Based on the results, we calculate centrality metrics reflecting the outcome of the application, highlighting its important properties. We observed that the centrality values obtained by running the epidemic model and the connectivity model turn out to be mutually equivalent, as predicted by their similar fashions of calculation. Here we study about the network Modelling of Epidemics and its similarities with the probabilistic model.

Keywords: Epidemic spreading, Epidemic modelling, Complex network, Network Connectivity, Network theory.

AMS Mathematical Subject Classification: 00A05, 00A06, 58Z05, 62-02, 62-11,62P10.

CHAPTER 1
INTRODUCTION

Mathematical functions can be applied as tools to describe the dynamics of how infectious diseases propagate among people. Mathematical modelling generates a picture or a 'model' of the dynamics of the disease, which can be visually represented by graphs, charts and comparative tables.

Models provide valuable inputs to visualise how diseases affect people. Hence, epidemiologists — public health experts – use them extensively to assess risk or to analyse intervention strategies to control or prevent diseases. Insights available from models facilitate disease management protocols like mass vaccination drives, treatment patterns, and precautionary procedures.

1.1 Epidemic

An epidemic is the rapid spread of an infectious disease to many people in a given population within a short period of time.

An epidemic is a sudden disease outbreak that affects a large number of people in a particular region, community or population. In an epidemic, the number of people affected by the disease can happen if

the virus, bacteria, or other cause of the disease has recently grown stronger, is introduced somewhere it has never been before, or finds new ways to enter the bodies of those it is affecting. It also can happen if people somehow grow more susceptible to the cause of the disease or have greater exposure to it [14].

Examples:

- Some well-known examples of epidemic include the Spanish flue pandemic in 1918, the HIV/AIDS Epidemic, and the recent COVID-19 pandemic.
- Epidemic can occur on a local, national, or global scale, depending on the infectious disease and its transmission patterns.

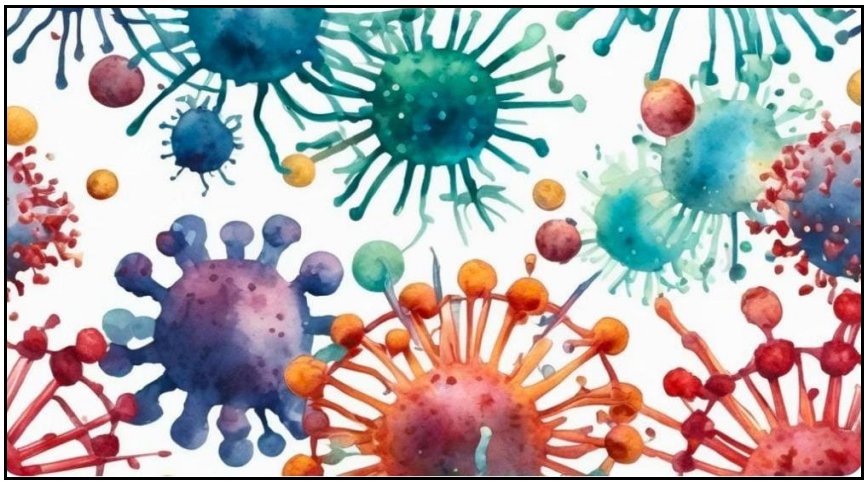


Figure 1.1 Illustration of virus

1.2 Historical background of epidemics

Many such worst epidemics and pandemic have arisen in the world, which led to the death of many people. Here we will talk about the last four deadly diseases, which have threatened humanity after every 100 years and humans have to find new pandemic medicines every time and this has been happening for the last 400 years. From 1720 to 2020, four disease outbreaks have devastated humanity, these are the plague in 1720, the cholera outbreak in 1820, the Spanish flu in 1920, and now the coronavirus in 2020 [18].

Figure 1.2 Representation of history repeats

1.2.1 Great Plague of Marseille (1720)

The Great plague of Marseille hit the world between 25 May 1720-1722. It was the last of the significant European outbreaks of bubonic plague, which is one of three types of plagues. The disease that originated in Marseille area of France in 1720, killed a total of 1,00,000 people. 50,000 in the city during the next two years and another 50,000 to the north in surrounding provinces and towns [18].

Figure 1.3 Death rate of Great Plague

1.2.2 Cholera Pandemic (1820)

The first pandemic hit the world during 1817 to 1920. The Cholera outbreak, which is also known as the first Asiatic cholera pandemic or Asiatic cholera, began near Kolkata and spread throughout southeast Asia to the middle east, eastern Africa and the Mediterranean coast [12]. Each year there are 1.3 to 4.0 million cases of cholera and 21000 and 143000 deaths worldwide due to cholera [17].

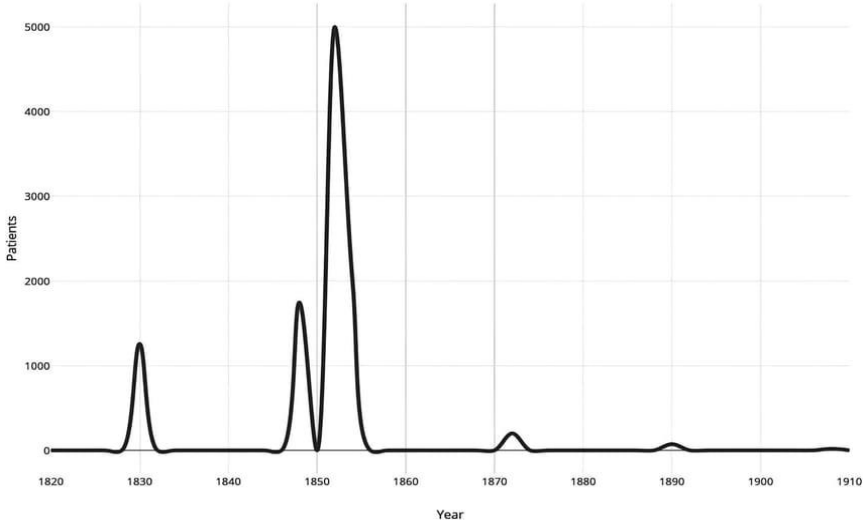


Fig 1.4 Cholera death rate per year

1.2.3 Spanish Flu Pandemic (1920)

The Spanish flu pandemic hit the world for two years between January 1918 to December 1920. 500 million people infected were infected with the Spanish flu worldwide and more than 50 million people died. The fatality as a percentage of population during Spanish flu was approximately 2.7 percent [15].

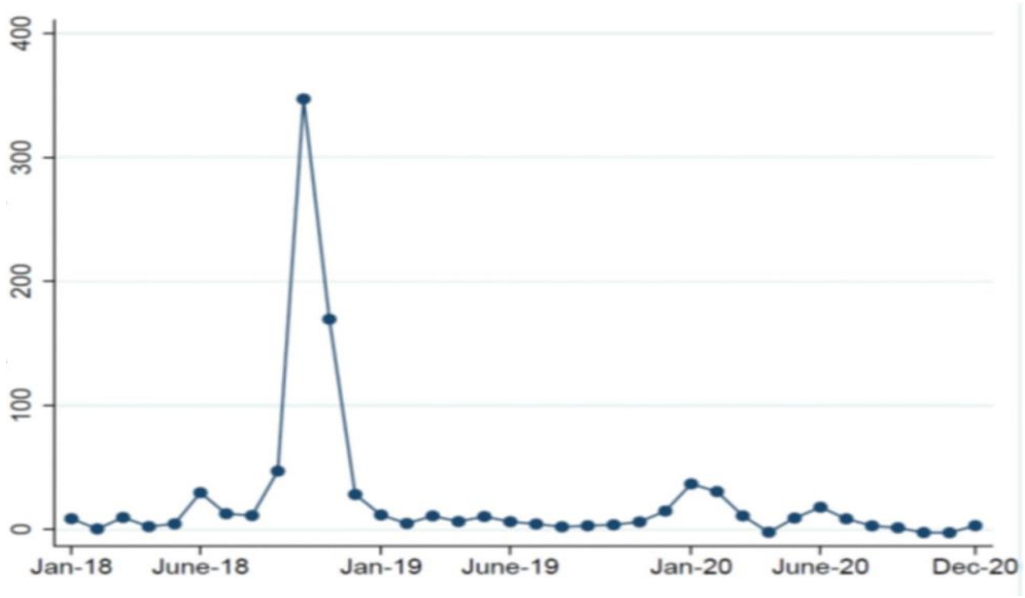


Fig 1.5 Spanish Flu death rate

1.2.4 Coronavirus Pandemic (2020)

The coronavirus disease (COVID-19) pandemic, which originated in the city of Wuhan, China, has quickly spread to various countries, with many cases having been reported worldwide. COVID-19 pandemic has caused a significant global crisis, with over 663 million confirmed cases and 6.68 million deaths worldwide, as of December 2022. The virus, caused by the severse acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [20].

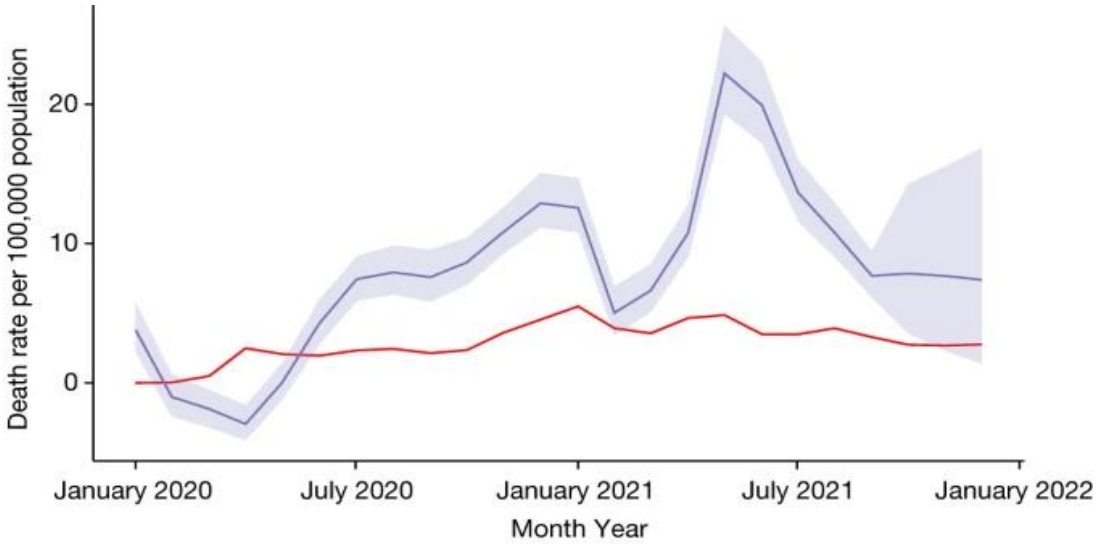


Fig 1.6 Global excess death rate per 1,00,000 population

Reports COVID-19 death rate per 1,00,000 Excess death rate per 1,00,000

1.3 Epidemic spread

The study of epidemics has always been of interest in areas where biological applications coincide with social issues. For instance, epidemics like influenza, measles, and STDs, can pass through large group of individuals, populations, and/or persist over longer timescales at low levels. These might even experience sudden changes of increasing and decreasing prevalence. Furthermore, in some cases, single infection outbreaks may have significant effects on a complete population group.

Epidemic spreading can also occur on complex networks with vertices representing individuals and the links representing interactions among individuals. Thus, spreading of diseases can occur over the network of individuals as spreading of computer viruses occur over the world-wide-web. The underlying network in epidemic models is considered to be static while the individual state vary from infected to non-infected individual according to certain probabilistic rules Furthermore, the evolution of an infected group of individuals in time can be studied by focusing on the average density of infected individuals in steady state.

Lastly, the spread as well as growth of epidemics can also be monitored by studying the architecture of the network of individuals as well as its statistical properties [9].

1.3.1 Branching

One of the essential properties of epidemic spread is its branching pattern, thereby infecting healthy individuals over a time period. This branching pattern of epidemic progression can be classified on the basis of their infection initiation, spread and further spread.

1. Infection initiation: If an infected individual comes in contact with a group of individuals, the infection is transmitted to each with a probability p, independent of one another. Furthermore, if the same individual meets k others while being infected, these k individuals form the infected set. Due to this random disease transmission from the initially infected individual, those directly connected to it get infected.
2. Spread: Every individual in the original infected set meets k other individuals, which results in k^2 individuals.
3. Further spread: The infection spreads further with each individual in the present infected set connecting to k healthy individuals with a probability [9].

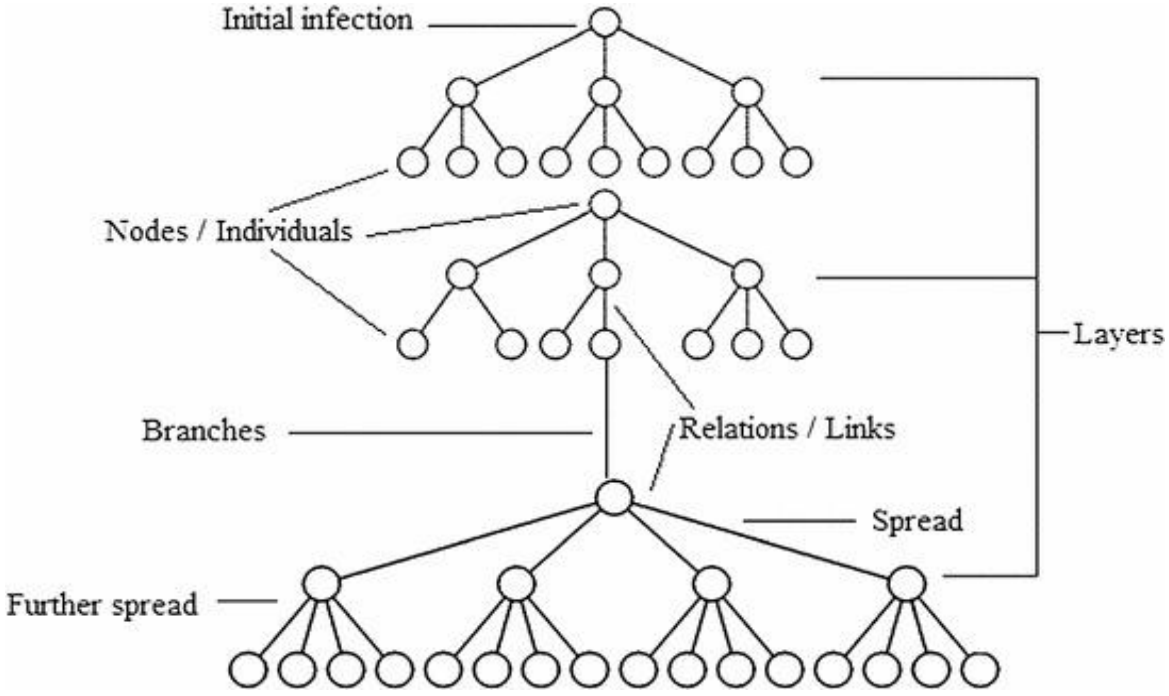


Figure 1.7 Branching modes and patterns in epidemic progression

1.4 Network

Network (or graph) are extremely flexible tools for representing complex systems of interacting components. Each component is represented by a node (or vertex) and each link (or edge) between

nodes describes some sort of interaction between them. Here, we focus on the specific application of network in the field of infectious disease modelling. Because of their flexibility, networks have been used to model infection spread in different forms. Nodes can describe single individuals, groups of individuals or locations to which individuals are connected. Links can represent infectious attempts or transmission events or simply acquaintances between them, movements of animals between farms, flight routes, etc., [7].

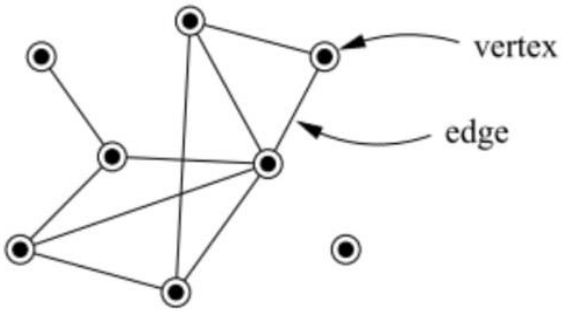


Figure 1.8 Representation of Network

1.4.1 Basic Characteristics of Network

Let G (V, E) be an unweighted, undirected network with V representing the set of nodes and E∈V×V representing the set links between node in the network. For a finite network with N nodes, the set E can be encoded in the adjacency matrix whose entry g_{ij} is 1 if there is a link between i and j, and 0 otherwise. A finite network is determined uniquely by its adjacency matrix.

Degree distribution is the most important characteristic of the network. The degree of one node is the number of links connecting it. Then, the degree distribution is given by $P_k = \frac{N_k}{N}$, where N_k is the number of nodes with degree k. Some widely used network include regular random, poisson, bimodal, and scale-free networks.

Clustering, one frequently used statistical property of characterizing networks, measures the probability that two neighbors of a randomly chosen node share a link to form a triangle. Specifically, the clustering coefficient of node i is defined as the ratio of the number of triangles and triples:

$$c_i = \frac{2T_i}{K_i(K_i - 1)},$$

where T_i and $\frac{K_i(K_i-1)}{2}$ are the total number of existing links and the number of all possible links between the neighbors of node, respectively. Furthermore, the degree-dependent clustering coefficient of node with degree k can be defined as the average clustering coefficient of nodes with degree k, i.e.,

$$c(k) = \frac{1}{N_k} \sum_{i \in y(k)} ci$$

where $y(k)$ is the set of nodes with degree k . Then, the clustering coefficient of the network is defined as the weighted average of degree-dependent clustering coefficient, which is also the average of clustering coefficient of nodes: $\bar{c} = \frac{1}{N}$

where k_{max} is the maximum degree of nodes [4].

2. NETWORK THEORY

2.1 Fundamental Concepts

A Network is made up of two objects nodes, and links. These are abstract terms for the individuals we want to consider, and the relationship between them respectively. In the current context, these could be individuals who can be infected and contacts that can lead to the transmission of disease. Suppose we have N nodes labelled by indices I, j=1,2…., N. Then a common way to represent network structure is through the adjacency matrix $G = G_{ij}$, where

$$G_{ij} = G_{ji} = \begin{cases} 1 & if\ i\ and\ j\ are\ linked, \\ 0 & otherwise. \end{cases} \quad (1)$$

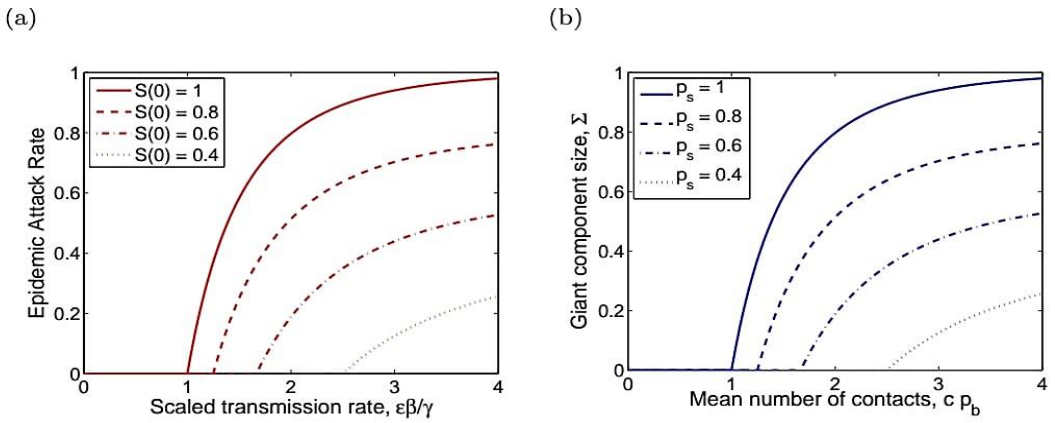


Figure 2.1 (a) shows the final size of an SIR epidemic
(b) shows the giant component size of an ER random graph

It is possible to define generalizations of this, where the matrix is not equal to its transpose or takes general values. There is also the question of whether a node can be connected to itself but in the context of infectious disease, it makes most sense to assume that G_{ii}=0 so that nodes do not link to themselves. There are many different properties of a network that can be defined, the degree of node 'i' is,

$$k_i = \sum_j G_{ij} \quad (2)$$

We will also write N_k for the number of nodes of degree k, so that, $d_k = \frac{N_k}{N}$ is a discrete distribution known as the network's degree distribution.

Another particularly important concept for epidemic network is that of a component- asset of nodes for which any pair is linked to each other through a finite-length path the network. By labelling the nodes correctly, it is possible to write the adjacency matrix in block diagonal form:

$$G = \begin{pmatrix} G_1 & 0 & 0 & \cdots \\ 0 & G_2 & 0 & \cdots \\ O & 0 & G_3 & \cdots \\ \vdots & \vdots & \vdots & \ddots \end{pmatrix} \quad (3)$$

So that $G_{(C)}$ is the adjacency matrix for component C. There is clearly a qualitative difference between a network in which a significant number of the nodes are in one component, and a network made up of many small components. We will now turn to how network can move between one regime and the other [5].

2.2 Erdos-Renyi random graphs

Stochastic processes that produce network are called random graphs models. The word 'graph' is essentially synonymous with the word 'network' in this context, although some authors do make a distinction. These models are useful a variety of reasons. It may be that the family of networks produced by a random graph model has interesting properties or the random graph model might be used as a null model i.e., something to test against real data-in statistical work.

The Erdos-Renyi (ER) random graph model involves taking N individuals, and putting a link between each of the $\frac{N(N-1)}{2}$ pairs of individuals with independent probability π. While a highly mathematical treatment of this model is possible, we will argue heuristically here. Of particular interest is the size of components the network produced. The largest components in a network is called the giant component the key qualitative difference of network types is whether the giant component size as a proportion of the nodes S tends to 0 as $N \to \infty$, or whether it tends to some finite value between 0 and 1.

Let us suppose we are in the latter situation, and pick a random node in the graph. The probability that this node is not in the giant component is x, which is the same for all nodes since they are not differentiated and we have picked randomly. Now consider all other nodes in the network if the initial node, or connected to the initial node and not in the giant component themselves. We can write this statement mathematically as

$$x = ((1-\pi) + \pi x)^{N-1} \quad (4)$$

Which is a polynomial in x with no simple analytic solution. As N increases, even numerical solution of (2) becomes difficult, and it is necessary to take the limit $N \to \infty$, constant the mean number of contacts per node $c = (N-1)\pi$, so that

$$x = e^{(x-1)c} \quad (5)$$

is the appropriate equation for the probability that a node is not in the giant component of a very large ER graph. Before doing this, note that c is the mean node degree, and the networks degree distribution will be Poisson with parameter c in the limit $N \to \infty$. One important qualitative feature of the ER random graph model is that it undergoes a phase transition at c=1.

Below this critical value, the equation (5) does not have a solution such that $0 \leq x < 1$ and there is not sizeable giant component the network is made of lots of small components. For $c > 1$, one component dominates meaning that a disease spreading around the population can reach a significant proportion of individuals.

2.3 Percolation on graph and epidemics

Percolation is a standard tool in statistical physics in the context of networks, there are two ways that this method is used to modify an existing network. In site percolation, each original node is present in the modified network with independent probability p_s. In bond percolation, each original link is present in the modified network with independent probability p_b.

Suppose we have applied both node and link removal to an ER random graph, and make the same argument about picking a node at random.

If the node is not in the giant component, then either it is not present following site percolation, or else all other nodes in the network that survive site percolation must either (i) not be linked in the original ER graph, or be linked in the original ER graph and have the link deleted during bond percolation or (ii) be linked in the original ER graph, have the link remain during bond percolation, and not be in the giant component. After some mathematical manipulations along the lines of those used to derive (5), tacking the $N \to \infty$ limit for constant c gives

$$x = (1 - p_s) + p_s e^{(x+1-2p_s)cp_b}$$

Then the giant component size is given by $\sum = 1 - x,$ once we have solved for x. Figure 2.1(b) shows the results of doing this having gone through all the math's above, it is not surprising both plots in Figure 2 are identical since the equations used to generate them are mathematically isomorphic. In fact, we can write this equivalence out explicitly as shown in Table 2.1.

EPIDEMIC	NETWORK
Basic reproductive ratio, R_0	Mean node degree, c
Leaky vaccine scaling, ε	Remaining fraction of links after bond percolation, p_b
All-or-nothing vaccination level, p_V	Fraction of nodes removed by side percolation, 1-p_s
Attack rate, R_∞-p_V	Giant component size, $\sum$

Table 2.1 Parallels between SIR epidemics and ER random graphs

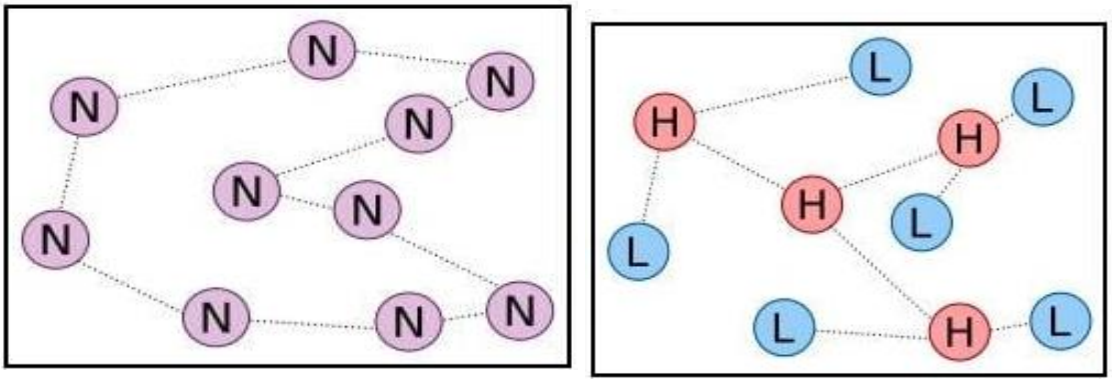
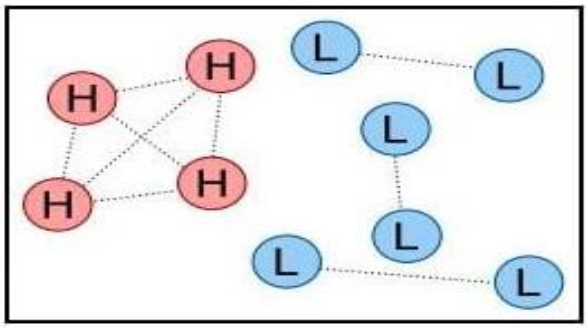


Figure 2.2

(a) Regular graph with degree 2 **(b)Heterogeneous network with high degree**



(c) Highly degree-assortative heterogeneous network

Figure 2.2 (a) shows a regular graph in which every Node has degree 2. (b) shows a heterogeneous network where some nodes are High- degree with 3 links and others are Low-degree with 1 link, but there are connections between these two types of nodes. (c) shows a highly degree-assortative heterogeneous network where high-degree and low-degree nodes connect only to other nodes of the same type.

Even for the simpler SIR case where immunity to further infection is long-lasting, side and bound percolation do not work as calculational tools where short, closed loops are present in the network in appreciable numbers.

But where percolation works, it is very useful, since there are few other analytic approaches to epidemics on networks. Generally applicable Monti-Carlo methods, where a computer pics random numbers to simulate the epidemic process, can be highly computationally intensive [3].

2.4 Epidemic Models

The process modeling of infectious disease has been a means of study disease spread and predicting of an outbreak as well as evaluating strategies for the control and possibly prevention of the epidemic.

An epidemic model is a simplified means of describing the transmission of a disease through individuals. There are three main aims:

- Understand the spreading mechanism of the disease
- Predict the future course of epidemic
- Understand how to control the spread

There are different types of models such as Stochastic and Deterministic alongside with different variations of each model.

Stochastic models rely on a chance variation in risks of exposure, disease and other factors, and provide information in small or isolated populations. Having advantages, however, they are very difficult to test, set simulations to yield any useful data. Apart from that, these models can be mathematically complex and contribute nothing to the explanation of the dynamics.

Deterministic models, also known as compartmental models, try to summaries, describe and explain what happens, on the average, to the population during and after epidemics. A compartmental model is one where each individual within a population can be in one of a number of different categories, and that transitions occur between categories. They are deterministic because they do not require a lot of data, they are set up relatively easily and computer software can be used to run them. Examples of such models include SI, SIS, SIR and SEIR models, in addition to these, there are other more complex deterministic models that combine elements from stochastic models.

These letters of the models such as SI or SIR have the following names:

- S=Susceptible
- E=Exposed
- I=Infections
- R=removed

SI model involving, S and I compartments, although general outlook of other common models such as SIS and SIR will also be given later.

We define S(t), I(t) and R(t) as number of susceptible, infective and removed individuals at a certain times t.

2.4.1 SI Model

For epidemic modelling, SI model, which stands for Susceptible-Infective, is one of the simplest. The population is divided in two compartments, where an individual with a disease is infective and an individual without the disease, but who can catch the disease, is called a susceptible.

Susceptible ⟹ Infected

For this model, there are some assumptions that we consider:

- Initially, there is at least one per
- son with the disease
- Disease can be passed on with certain probability upon contact
- Infectives remain in contact with susceptible for t times
- There is no death caused by the disease, it is assumed that once an individual is infected, they stay infected
- Newborns and the natural death of the population are not considered, so we take population size to be constant.

For the SI model, we name this the equation of constraint:

S(t)+I(t)=N

Where,

S(t) →represents number of susceptible populations at time t

I(t) → represents number of infected populations at time t

N → represents the population size

We can further elaborate that not everyone has the same likelihood of getting a disease, for example: comparing public workers and general public, where public workers of departments such as healthcare are much more likely to catch a disease according to the studies. However, we won't look into those cases here.

Just like any other deterministic model, SI requires some equations to be solved. In our case, we have a pair of ordinary differential equations:

$$\frac{dS}{dt} = -\frac{\beta}{N} IS + gI$$

$$\frac{dI}{dt} = \frac{\beta}{N} IS - g\mathrm{I}$$

For further calculations, we also need to define few terms:

b → represents the population birth rate

p → probability of disease transmission

$\frac{\beta}{N}$ → the rate of infection from a given susceptible to a given infective

g → the rate of recovery (Note that this quantity is also widely used as γ)

we can use fraction of population (or proportions) instead of population values, such that $s(t) = \frac{S(t)}{N}$ and $i(t) = \frac{I(t)}{N}$. Combined with constraint expression, we get:

s(t) + i (t)=1

Equations now can be rewritten as

$$\frac{di(t)}{dt} = \beta s(t) i(t)$$

$$\frac{ds(t)}{dt} = -\beta s(t) i(t)$$

In both equations, β is the rate of infection; the top equation controls the number of infected people, growing with time and the bottom one controls number of susceptible people, decreasing with time t.

Plugging in $\frac{dS}{dt}$ into $\frac{dI(t)}{dt} di(t)$ yields.

$$\frac{di(t)}{dt} = \beta\big(1 - i(t)\big) i(t)$$

With initial conditions of i (t = 0) =i_0 such as, at the time t=0 we have i_0 infected population.

Hence, the solution to $\frac{di(t)}{dt}$ is given by the following:

$$i(t) = \frac{i_0}{i_0 + (1 - i_0) e^{-\beta t}}$$

of course, right away it is clear that if $i_0 = 0$, there will be no infection, so we need initial number of infected populations to be greater than 0. Since t→ ∞ and $\beta > 0$, according to this solution, everyone will be infected, illustrated in the graph below. For the limit when t→ ∞,we get

i(t)→1 s(t)→ 0

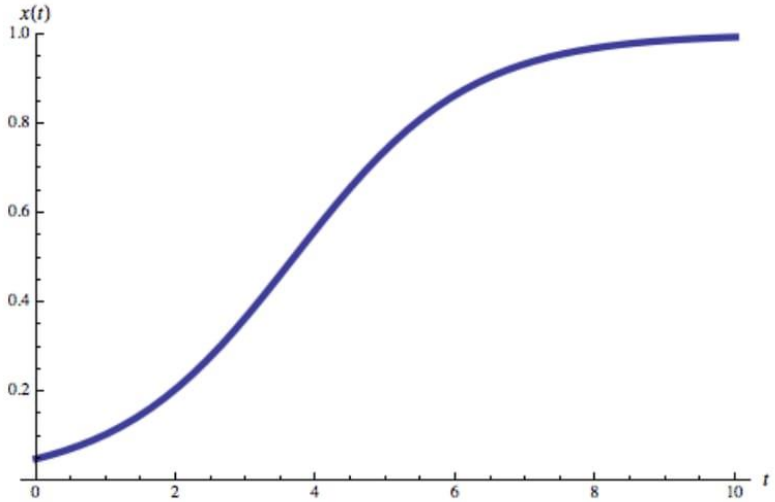


Figure 2.3 Logistic growth function graph

2.4.2 SIR model

There are two models of SIR, one describing the epidemic and another – the epidemic, a disease present in the population for a long period of time where the class of susceptible is being nourished by new income from births or recovered individuals who lost their temporal immunity.

We'll look at the epidemic model by, generally, keeping almost all assumptions that we had for SI model. The big difference here is the addition of R(t), the compartment of Recovered (or removed) population. This, consequently, changes the differential equations system, while the population formula being now S(t)+I(t)+R(t)=N.

The stages of SIR follow accordingly Susceptible $\Longrightarrow$ Infected $\Longrightarrow$ Removed

The "endgames" of SIR model are the following scenarios: (1) an epidemic happens and everyone is Removed;(2) whoever got infected is then recovered, meaning that the population becomes divided between Susceptible who never got a disease in the first place, and recovered, who were infected before.

The rate of recovery, g, comes into the expression alongside with β which is rate of infection.

Hence, for SIR model, we have the following equations:

$$\frac{dS}{dt} = bN - \frac{\beta}{N} IS - dS$$

$$\frac{dI}{dt} = \frac{\beta}{N} IS - gI - dI$$

$$\frac{dR}{dt} = gI - dR$$

Here, we may choose to continue our calculations by using compartment ratios and setting them such that:

$$\frac{ds(t)}{dt(t)} = \frac{ds}{dt} \quad \frac{di(t)}{dt(t)} = \frac{di}{dt} \quad \frac{dr(t)}{dt(t)} = \frac{dr}{dt}$$

Hence, the equations become

$$\frac{ds}{dt} = -\beta si$$

$$\frac{di}{dt} = \beta si - gi$$

$$\frac{dr}{dt} = gi$$

With $s + i + r = 1$.Here, equations are a bit more difficult to solve outright, so let's plug in $\frac{dr}{dt}$ into ds/dt into $\frac{ds}{dt}$. The expression for $\frac{ds}{dt}$ now becomes

$$\frac{ds}{dt} = -\beta s \frac{dr}{dt} g^{-1}$$

$$s = s_\circ e^{-\frac{\beta}{g}r}$$

this gives connection between number of susceptible and recovered population. And now using the constraint $s + i + r = 1$, we get

$$\frac{dr}{dt} = g(1 - r - s_\circ^{-\frac{\beta}{g}r})$$

Solution can be found by solving the system of all 3 differential equations of compartments $\frac{di}{dt}$, $\frac{ds}{dt}$ and $\frac{dr}{dt}$, which gives $t = g^{-1}\int_0^r \frac{dr}{1-r-s_\circ^{-\frac{\beta}{g}r}}$

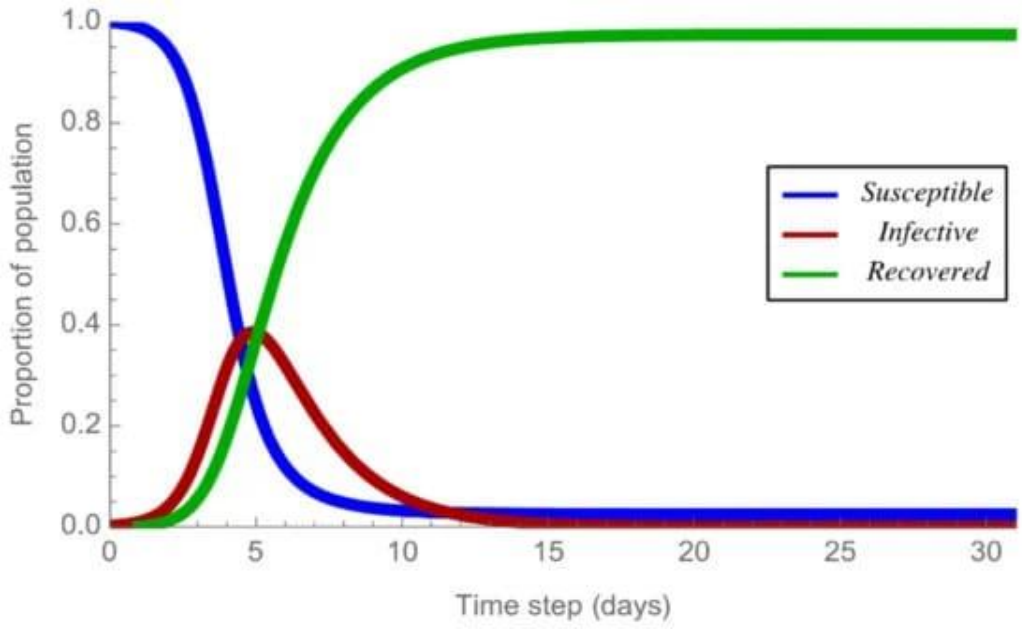


Figure 2.4 Three Compartment graph

Figure 2.4 above shows graphs of 3 compartments, susceptible as green, infected as red and removed as blue. This SIR model starts with some number of Infective, a lot susceptible and no (yet) Removed population. Notice that there is a difference between blue line (Removed) approaching the "ceiling" of the graph and indicator of the whole population, i.e., 1.0 on the vertical axis. As clearly shown in the graph on the left, when we have a case of $\beta > g$, as t→ ∞, Removed compartment dominates and there are very few susceptible left who did not get infection at all.

2.4.3 SIS model

Infections that cannot be stopped with long lasting immunity such as common flu, can be modelled using SIS model. The idea behind is that after susceptible become infected, they can be recovered but since they are still vulnerable – they join the rest of susceptible compartment.

Susceptible ⇔ Infectious

Susceptible ⇒ Infectious⇒ Susceptible

The differential equations now become

$$\frac{dS}{dt} = bN - \frac{\beta}{N}IS + gI - dS$$

$$\frac{dI}{dt} = \frac{\beta}{N}IS - gI - dI$$

Where

b → population birth rate

d → death rate

g → recovery rate

$\frac{\beta}{N}$ → rate of infection from a given susceptible to a given infective.

Combining these two equations above gives the size of the population

$$\frac{dN}{dt} = \frac{d(S+I)}{dt} = (b-d)N$$

With

$$\frac{dS}{dt} + \frac{dI}{dt} = 0 \iff S(t) + I(t) = N$$

From here, just like before, we may choose to continue our calculations by setting $\frac{ds(t)}{dt(t)} = \frac{ds}{dt}$ and $\frac{di(t)}{dt(t)} = \frac{di}{dt}$ as well as using β infection rate (on contact) and g as recovery rate to keep our equations clear and simple.

Now, differential equations for SIS model are

$$\frac{ds}{dt} = -\beta si + gi$$

$$\frac{di}{dt} = \beta si - gi$$

$$s + i = 1$$

Since there is recovery in this model at rate of g, the proportions of infected people and susceptible change accordingly and based on the ratios of $\frac{\beta}{g}$, we will get an equilibrium. Actually, two type of equilibrium cases, one based on a certain number of susceptible and infected people when balanced is preserved over time, and the other one is a possibility that the disease vanishes, susceptible will then occupy the whole population.

Setting i (t = 0) = i_0 yields

$$\frac{di}{dt} = (\beta - g - i)i$$

With the solution

$$i(t) = (1 - \frac{g}{\beta})\frac{C}{C + e^{-(\beta - g)t}}$$

And the constant being

$$\frac{\beta i_\circ}{\beta - g - \beta i_\circ}$$

Now, as limit of t tends to ∞, we have 2 cases:

1. $\beta > g, i(t) \rightarrow (1 - \frac{g}{\beta})$

2. $\beta < g, i(t) = i_\circ e^{(\beta - g)t} \rightarrow 0$

This is exactly what is mentioned few lines above. When. $\beta > g$, the population will eventually be in equilibrium with some number of infected and susceptible people, depending on the fraction of infection rate and recovery rate g. Otherwise, if $\beta < g$,infected number of people goes to 0, i.e., infection vanishes.

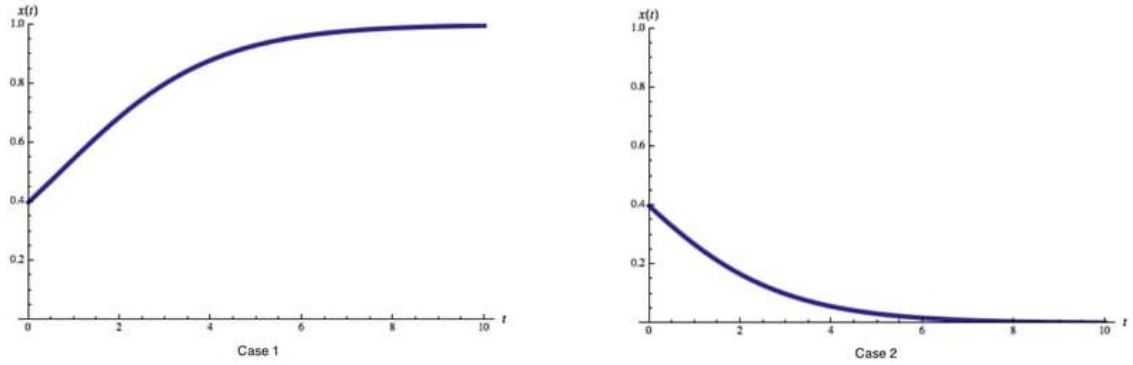


Figure 2.5 Logistic growth functions for case (1) and case (2)

Figure 2.5 shows that in the case (1) there will always be population of susceptible people (or nodes in network) and this value become susceptible again. in the case (2), infection rate is weaker and so epidemic never happens and disease vanishes [19].

3.NETWORK MODELLING ON EPIDEMIC

There are a number of different families of models in use today, each with different advantages and disadvantages. This commentary argues that adopting a network perspective that explicitly accounts for the structure of interactions among individuals can provide important insights regarding both the spread of a disease and the best way to tackle it, in particular when compared to the class of SIR models, one of the most widely used modeling approaches in epidemiology.

Network models have successfully been employed in many fields to study phenomena for which interrelationships matter. In economics, these included job referrals in labor markets patterns of international trade, the diffusion of technology and contagion in financial markets. In all of these areas, adopting network models has led to new perspective and novel insights, and we argue that network models can do the same to advanced our understanding of epidemics.

3.1 The Evolving Network Model

The local-world evolving network was first proposed by Li and Chen. It captures the localization of real-life networks. Then, Zhang et al. presented expanded local-world evolving network with tunable

clustering by including a triad formation (TF) step. Considering the local-world structure and clustering of networks, we propose the generating algorithm of the dynamic clustered network with demographics. It is summarized as follows:

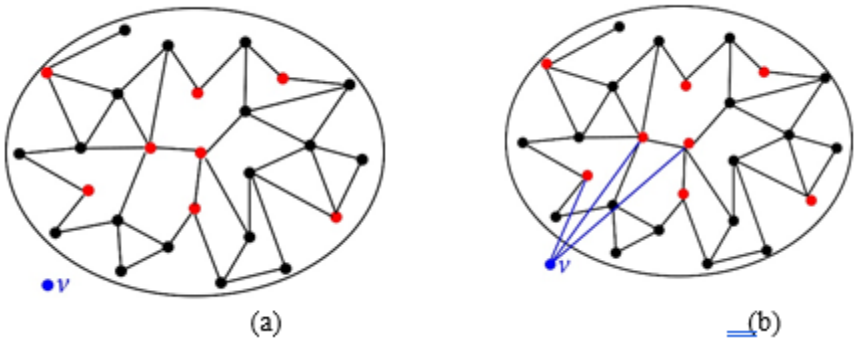
1. *Initial condition:* the initial network consists of N nodes and has a Poisson degree distribution with average degree (K).
2. *Network evolving:* at each time step t, we fist add a new node v to the network and then delete a randomly selected node as well as its links from the network. the attachment mechanism between the new node v and existing node is as follows:
3. *Local-world establishment:* randomly select M nodes from the network, referred to as the "local world" of v.
4. *Local preferential attachment (LPA):* the new node v connects to m different nodes in its local world according to the preferential attachment. Thus, the probability $\pi_{Local\,(k_i)}$ that node v is connected to an existing node i in the local world of node v depends on the degree k_i of node i:

$$\pi_{Local\,(k_i)} = \frac{M}{N}\frac{k_i}{\sum_{l\in Local} k_i}$$

i. *Triad formation (TF):* when the new node v connects an existing node i, we then implement a TF step with probability p. Specifically, if a link between v and i was added in the previous LPA step, then one more link between v and a randomly chosen neighbor of I is added. If all neighbors of i have already been connected to v, do one more LPA step instead.

A sketch of the network evolving process is shown in figure 1. After T steps (the designed total iteration steps which are large enough to reach a stationary network), the algorithm results in a connected network with N nodes and average degree is (k) = m(1+p).

obviously, the network size keeps a constant and it has $M \le N$ and $0 \le p \le 1$. There are two limiting cases for the evolving network: M=m, p=0 and M=N, p=0. When M=m, p=0, the stationary degree distribution is Poisson with mean m, while when M=N, p=0, the stationary degree distribution is "stretched exponential," namely, p_k . Besides, the parameter p allows us to include the clustering effect into network by building triads.



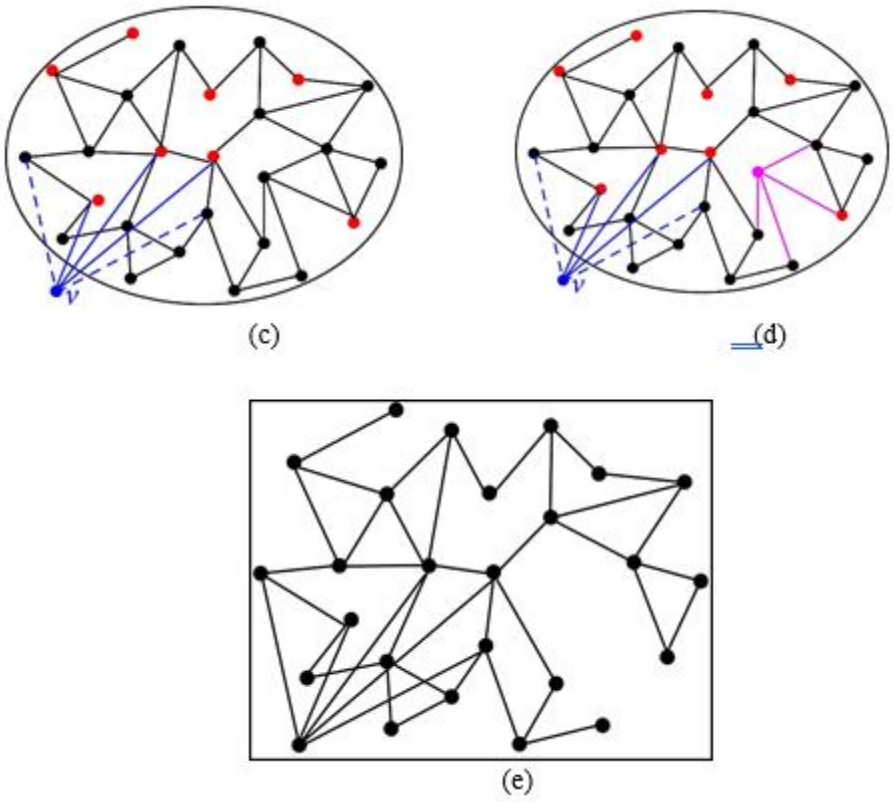


Figure 3.1 The network evolving process

Figure 3.1: Sketch of the network evolving process with the local-world size M=8 and the value m=3. In (a), M nodes (the red nodes) are randomly selected as the local world of the new node v (the blue node). In (b), m nodes in the local world are chosen to be connected with v by blue lines according to the preferential attachment. In (c), for the m nodes in (b), a randomly chosen neighbors is linked to v (by blue dashed line) with probability p. In (d), one randomly selected node as well as its links (denoted by pink color) is deleted from the network. Then, the network after one step, i.e., adding one node and then deleting one node, is shown in (e).

3.2 Epidemic Spreading on Evolving Networks

We consider an SIS epidemic spreading on evolving networks, where the initial network is the stationary network obtained by the generating algorithm. During the epidemic, nodes can neither be susceptible (S) or infectious (I). The susceptible nodes can be infected by their infectious neighbors with a per-contact transmission rate λ if they are connected to one or more infected nodes, while the infectious one can be cured at a recovery rate γ . when an infectious node recovers, it turns to susceptible and can be infected again. Meanwhile existing nodes together with all their links can leave the network due to death, and new node enter or leave the network is identical to that in the evolving network model. thus, the population size is a constant. Given a shorter time interval δ_t, the algorithm of epidemic spreading on evolving networks can be summarized as follows:

1. *Initialization*: an undirected and unweighted network with N nodes is created and set to the initial network by performing the network generating algorithm in the evolving network model. then, I (0) randomly selected nodes in the network are initially infected and the other nodes are susceptible.

2. *Epidemic process*: Given the state of each node at time t, the states of nodes at time t+ δt are updated as follows. Each infected node recovers and becomes susceptible again with probability $\gamma\delta_t$. Each susceptible node, such as node i, turns into infectious with probability λn_i, where n_i is the number of infectious neighbours of i.

3. *Network evolving*: At time t, D_t (extracted from the binomial distribution B (N, $\mu\delta_t$)) randomly selected nodes as well as all their links are eliminated from the network sequentially where are created according to the mechanism in section 3.1.
   To sum up, we can simulate the epidemic spreading on dynamic clustered networks by iterating the epidemic process and networking evolving process until there is no infectious node in the network or the number of infectious nodes reaches a steady state. During the iteration, record the number of infectious nodes in each time step [4].

3.3 The baseline SIR Model

There are many models that track the behavior of an epidemic as it infects a population. Most of these are categorical models in which individuals are categorized by disease status. A particularly prominent example of such a model is the SIR model introduced by Kermack and McKendrick (1927). In this model individuals are either susceptible (S), infected (I), or removed(R). individuals with already contagious individuals and the characteristics of the disease (such as how contagious it is), proceed to I and then R, which includes individuals who have either recovered (or are otherwise immune) or died.

Other categorical models include the SIS ('susceptible-infected-susceptible") model, in which recovered individuals can be reinfected and thus re-enter the pool of the susceptible, and the SEIR ("susceptible-exposed-infected-removed") model, which incorporates a new health status for individuals that have been exposed to the virus and are contagious but are as yet asymptomatic. The

key parameters describing the epidemic in the SIR model are the infection parameter, called reproduction number, which describes how quickly infected individuals infect others, and the

recovery rate, which describes how quickly infected individuals recover and there by stop being infectious. Once the parameters are fixed, the flow of newly infected individuals in these models depends on both the number of already infected individuals and the number of susceptible individuals at a given point in time. In the standard SIR model, the flow from susceptible to infected is proportional to the total numbers of both the susceptible and the infected. This approach seeks to capture the idea that for a given transmissibility of a virus the likelihood of infection depends on how frequently already infected individuals interact with those who are still susceptible.

In the SIR model, a disease to which no individual has immunity (as is likely the case with COVID-19) starts with a small number of infected and a large pool of susceptible individuals. As the infected individuals increases, the rate of new infections increases rapidly while the rate of removal remains constant, causing the number of those infected to increase more rapidly. Without any interventions or changes in behavior, the number of new infections pr day increases up to a peak and declines from then on.

The decline arises from the fact that fewer and fewer susceptible individuals are found in the population, limiting opportunities for further transmission. As the number of susceptible individuals declines further and a large share of the population in immune by having recovered, the spread of the disease will slow

down and at some point begin to peter out. The large number of immune individuals then provides protection to the population, resulting in herd immunity.

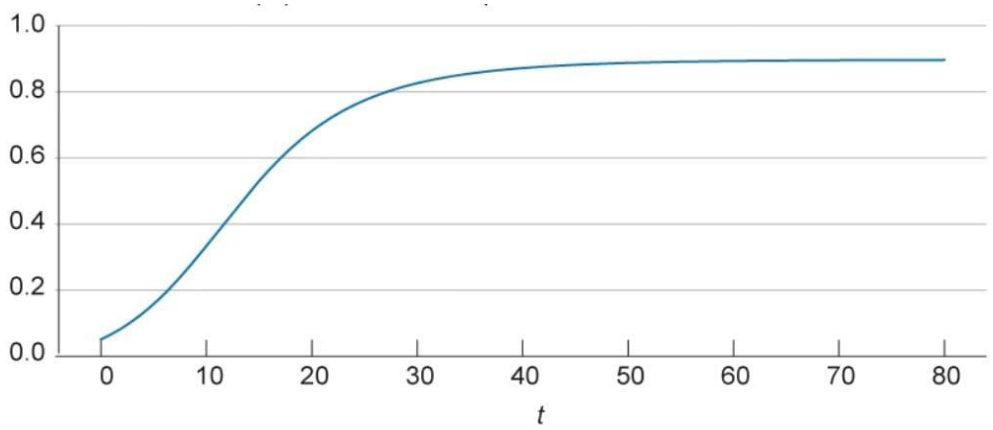


Figure 3.2 (a) Share of the population infected at time

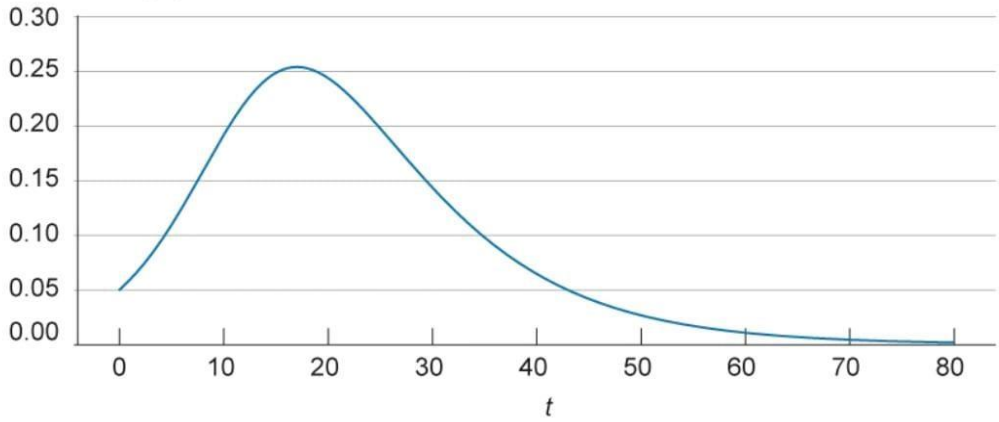


Figure 3.2 (b) Cumulative share of the population infected up to time

Figure 3.2: illustrates these infection dynamics for a simple setting with parameters based on the early COVID-19 experience. The top panel shows for any point in time the share of the population that is currently infected. The bottom panel shows for any point in time the share of the population that has ever been infected up that point; that is, it includes both those currently infected as well as those previously infected but now recovered.

Vaccines work in an SIR setting by protecting vaccinated individuals themselves, and by also increasing the share of immune individuals, there by solving down the spread of disease. If sufficient numbers are vaccinated, the population will reach herd immunity without large portion of the population ever becoming infected.

The SIR model has been a standard tool used in epidemiology to study the spread of infectious disease. Because both infection rates and mitigation efforts such as social distancing have a large impact on economic behavior, economists are now working with these models to better understand the interrelationships between public health measures and economics activity. However, like any model, it is not without shortcomings. The key limitation of the SIR model for the purposes of this commentary is the assumption it makes regarding the frequency at which infected individuals meet with those who are susceptible, there by generating opportunities for disease transmission.

The SIR model assumes uniform mixing across the entire population, meaning that infections evolve as if any susceptible individual interacts with and could be infected by any infected individual across the

population with equal probability. However, it is well documented that social interactions are not organized in this stylized way. Instead, individuals interact mostly within much narrower groups, shaped, for example, by family ties, work and social environments, and geography. Network models provide a route into analyzing epidemics in a way that takes these patterns of interaction into account.

3.4 SIR Model of Epidemics Networks

The key component of adopting the network approach to modelling an epidemic is the description of patterns of interaction using a network, consisting of nodes and links. Nodes represent individuals or households, and the links describe the interactions that potentially spread disease. The existing of a link could indicate, for example, that two individuals work in the same plant or attend the same school,
and a disease could be transmitted between them in that environment. Importantly, in the absence of a link, for example, because two individuals live at opposite ends of the country, the disease does not pass directly from one to the other.

Figures 3.3 and 3.4 illustrate two sample networks that will be useful in the discussion below. Nodes are represented by numerable blue circles and the lines between them are links.

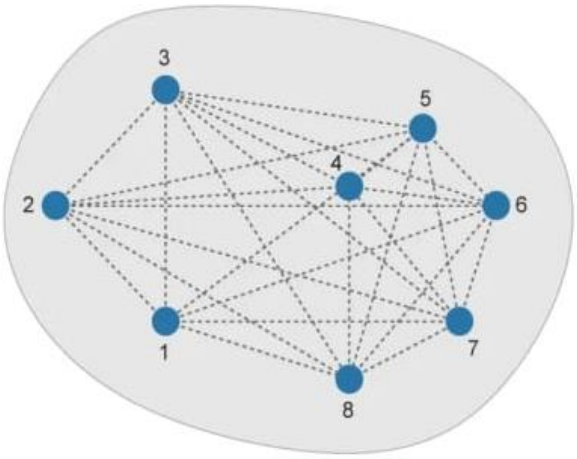


Figure 3.3 The Complete Network in the SIR Model

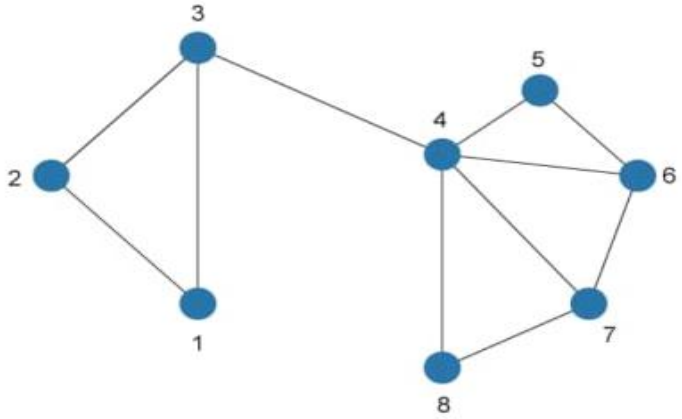


Figure 3.4 An Incomplete Network with Link Heterogeneity

The elements of the baseline SIR model can be accommodated within this network framework. Individual nodes are either "susceptible," "infected," or "removed," and the transitions between these states work as before: A susceptible individual interacting with many infected contacts will be more likely to become infected than an individual with only few or no infected contacts in their network. Just

as in the SIR model, the epidemic is then modeled by simulating the spread of the virus among individuals that interact. The difference is that the interactions are explicitly described by a network that permits more complex patterns of interaction than the baseline SIR model.

Within the framework of a network model, the baseline SIR model is a special case with two properties: First, each and every pair of individuals across the entire population is potentially linked (such a network is said to be complete). Second, each and every individual has the same number of links as all other individuals (such a network is homogeneous in its structure). Figure 2 illustrates these assumptions in a simple setting with eight nodes. Every pair of nodes is linked by a dashed line, indicating that in the SIR setting the disease can potentially be transferred between every pair of individuals in the population.

This is an extreme case, and in most settings the assumptions of completeness and homogeneity are not satisfied. Figure 3 shows a different network using the same eight nodes but adding heterogeneity to the link patterns-not all nodes are directly connected by a link and there is variation in the number of links that each node has. We will argue that ignoring such variation hides some important aspects of disease spread that a more flexible network model can uncover.

The greater granularity of the network model does not come without costs. For example, the data required to map out a given interaction network fully may be prohibitively costly to acquire. In addition, network models can be harder to work with than simpler classes of models and often can only be solved using simulations. However, we argue that the useful insights such models can offer about the spread of disease may make it worthwhile to incur the costs [2].

4.SIMILARITY OF EPIDEMIC SPREAD AND NETWORK CONNECTIVITY WITH TWO PROBABILISTIC MODEL

4.1 Methods

We compare two simulation models built for two use cases: the epidemic spreading simulation model, and the connection reliability model. Both models consider a network as a set of weighted nodes and weighted directed edges between them. Undirected edges are modelled as two identical edges with the endpoints swapped. The output of the models is the two-dimensional probability matrix C: $V \times V \rightarrow [0, 1]$, where C (s, t) marks the conditional probability of influence spreading from node s to node t, given that spreading starts from node s and V is the set of nodes of the network. Even though both models work by simulation, their approaches differ. Additionally, we present applications of our models for calculating centrality measures for nodes in the network. The probability matrix C (s, t) produced by both of the models gives the conditional probabilities of influence spreading from node s to node t given that influence starts to spread from node s. If the probabilities, at which the nodes initially start to spread influence are known, the conditional probabilities of the probability matrix can be multiplied by them, producing unconditional probabilities.

4.1.1 Models

The Spreading model works by simulating the spreading of influence from each node to the rest of the network separately. For each node as the source, the simulation is carried out a certain number of times: this is called the number of iterations. The probability of influence spreading to another node is calculated as the number of iterations where the node was influenced divided by the total number of iterations. The simulation itself works in steps, on which all newly influenced nodes (that is, nodes that became influenced on the step right before the current one) attempt to spread influence to all of their neighbouring nodes. This attempt automatically fails if the neighbouring node has been previously

influenced. Otherwise, the spreading will succeed with a probability of $w_e . W_t$, where w_e is the weight of the edge connecting the nodes and W_t is the weight of the target node. If the spreading succeeds, the target node will be marked as influenced and will attempt to spread influence in the subsequent step.

Another parameter, L_{max}, is used to limit the maximum spreading path length: influence will only spread along paths at maximum, L_{max} edges long. Limiting, L_{max} can be used to shorten the execution times of the model at the cost of less precise results or to model a situation where spreading paths are limited by some factor, such as cutting spreading chains as a preventive measure against an epidemic. A more detailed description and pseudocode for the Simulation model are provided in our earlier study.

The calculation in the Connectivity model works similarly in that it simulates the connectivity multiple times, taking the average results. Instead of simulating the influence spreading from each node separately, for each iteration, the set of active nodes and edges is randomly determined: the probability at which each node and edge is active is its weight. For each active node, the nodes that can be reached by paths consisting of active nodes and edges are considered connected to it. C (s, t) is then the number of iterations where the active node s had an active path to node t divided by the total number of iterations. Unlike the Spreading model, the Connectivity model does not have the, L_{max} parameter, as the connectivity is not determined by stepping along paths. A more detailed description and pseudocode for the Connectivity model is provided in our earlier study.

4.1.2 Theoretical equivalence of the models

For both models, the probability of influence spreading through a path with a specified starting node is the product of the weights of each edge and node on the path, excluding that of the starting node.In other words,

$$P(L)=\prod_{i=1}^{m} w_{ei} W_{ni}, \qquad (4.1)$$

where P(L) is the probability of spreading through the path L of m edges and nodes excluding the starting node, and w_{ei} and W_{ni} are the weight of the ith edge and node on the path, respectively, excluding the starting node. This is clear in the Spreading model, as all attempts to spread along the path must be successful, which happens with the probability of the product of the individual spreading probabilities, corresponding to the weights of the edges and nodes along the path. Here, the weight of the starting node is not included in the product, as the spreading is assumed to start from there. Similarly, in the Connectivity model, for two nodes to be connected via a certain path, all edges and nodes of that path must be active. The probability at which the whole path is active is then the product of the weights of the edges and the nodes on the path. The starting node's weight is left out of the product, as an active path from it can only exist if it is active in the first place.

As the probability of influence spreading through a path is given by the same equation for both of the models, the models' results should theoretically be equivalent. This equivalence only holds, however, when L_{max} is not capped to some number in the Spreading model, in order to take into account all possible paths in the network as the Connectivity model does.

4.1.3 Applications

The probability matrix that both of the models generate can be used for various applications. At the core of these applications are different centrality measures that reflect how central each edge and node in the network is. These measures give insight into the structure of the network, helping to understand patterns and phenomena present therein. We give three examples of centrality measures: the in- and out-centralities and the betweenness centrality [5].

4.1.3.1 In- and out-centrality

The in- and out-centrality measures are a natural way to approach node centrality [10]. They represent how much influence flows in and out of the node, respectively. We define the in-centrality of node 't' as

$$C^{(in)}(t) = \sum_{\substack{s \in V \\ s \neq t}} C(s,t) \qquad (4.2)$$

and, similarly, the out-centrality of node s as

$$C^{(out)}(t) = \sum_{\substack{t \in V \\ s \neq t}} C(s,t) \qquad (4.3)$$

where V is the set of nodes in the network.

In other words, the in-centrality of a node is the sum of the probabilities of spreading from all other nodes to the node in question, and the out-centrality of a node is the sum of the probabilities of spreading from the node in question to all other nodes. The in- and out-centralities directly translate to physical quantities of the network. As C (s, t) is the probability of influence spreading from node s to node t, the sum can be thought of as an expected value. In the case of in-centrality (Eq.2), the sum represents the expected number of nodes that will spread influence to the specified node, and, for the out-centrality (Eq.3), the sum represents the expected number of nodes that influence from the specified node will spread to. As a concrete example, the out-centrality of a node represents the expected number of infected people in a social network, when a contagious infection begins to spread from the starting node, and the in-centrality of an individual's vulnerability to being infected by different sources.

4.1.3.2 Betweenness centrality

The betweenness centrality measure is another approach to studying node centrality. It represents the significance of a node in transmitting influence between different parts of the network. Betweenness centrality can be easily defined for a set of nodes S, where the betweenness centrality of a single node s can be expressed as that for the set {s}. We first define the cohesion of a network as

$$C = \sum_{\substack{s,t \in V \\ s \neq t}} C(s,t) \qquad (4.4)$$

And the partial cohesion of the network without the set of nodes S as

$$C_s = \sum_{\substack{s,t \in V \setminus S \\ s \neq t}} C_s(s,t), \qquad (4.5)$$

where V is the set of nodes in the network and C_s is the probability matrix calculated with only nodes and edges between nodes in S taken into account. The probability matrix for partial cohesion has to be calculated independently from that for total cohesion, as the effects of removing nodes and edges can cut off paths between parts of the network and change the spreading probabilities.

With equations 4.4 and 4.5, we define the betweenness centrality as the relative difference between the total and partial cohesion:

$$b_s = \frac{C - C_s}{C} = 1 - \frac{C_s}{C} \qquad (4.6)$$

The cohesion portrays the total interconnectivity of the network. As the betweenness centrality is a relative difference in the cohesion (Eq.4.6), the larger the betweenness centrality, the greater the effect of removing the specified nodes is on the interconnectivity. The betweenness centrality can be used to

spot individuals who act in bridging roles between parts of the network, the isolation of which can help to contain the contagion to only a small part of the network.

4.2 Results and Discussion

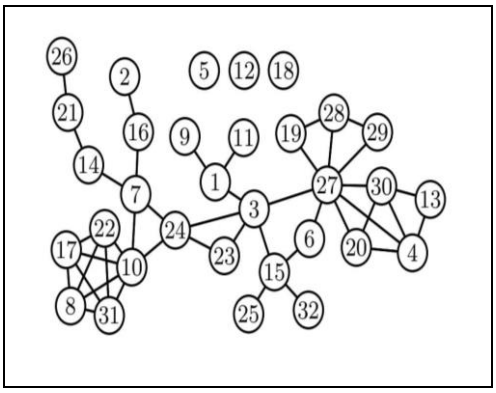
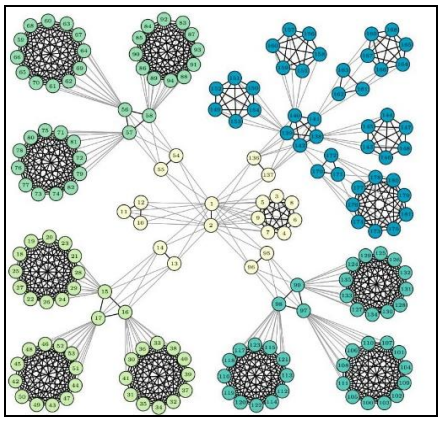


Figure 4.1 (a) A visualisation of the student network



(b) A visualisation of the Organization network

In the above figure visualisation of the networks used in the demonstration of the models. (a) A visualisation of the student network. (b) A visualisation of the Organisation network. Edges drawn in black represent relations within a group and have a weight of 0.5. Edges drawn in grey represent leadership relations and have a weight of 0.3. No other edges exist in the network. The colours represent the network's division into departments.

To compare the models, we run them on two networks, the student network and the Organisation network (Figure 4.1a, b). Precise descriptions of how the calculations in the models are carried out are presented in our earlier studies. The student network is a small, 32-node network composed of empirical data on the relationships of Dutch university students. We consider the network with all edge weights set to 0.5. The Organisation network, on the other hand, is a larger, 181-node network that represents a real-world organization structure. The network consists of five departments with multiple groups forming each of them as well as hierarchical leadership relations. The Organisation network was first introduced in, along with different classes of preventive measures that simulate epidemic prevention by decreasing the weights of certain edges.

In this paper, we study the case where preventive measures are in use on all edges except on those representing leadership relations, which means that only edges representing leadership and group relations are present. Edges representing group relations have a weight of 0.5, and edges representing

leadership relations have a weight of 0.3. Both of the networks are considered to be undirected with node weights equal to 1. The models work and give equivalent results for directed networks with varying node weights as well.

As both of the networks are real-life social networks, they are well suited for modelling epidemic spreading. Using the two networks, we can compare the models on networks exhibiting different properties: the student network is small and sparse, whereas the Organisation network is larger and much denser. Together, the networks represent a multitude of different situations for our models to perform on. It is worth noting, however, that as the edges of the networks are undirected, the in- and out-centrality (Eqs.4.2 and 4.3) for a node will always be equal due to the symmetry of spreading from and to the node.

In practice, the probability of an infection spreading from one person to another is often different than the probability of spreading in the other direction, represented by different weights in the directed edges between them.

The models calculate the probabilities of spreading between all pairs of nodes given the layout and weight parameters of the network. The results can be used to calculate important quantities, specific to the network and its weights. One example of such quantity is the basic reproduction number for epidemics, which is a measure of contagiousness defined as the number of new infections a single infection on average leads. We have calculated the basic reproduction number for simulations run on the Organisation network as a function of edge weights in our previous study. It is important to note that quantities such as the basic reproduction number are not specific to the models and must be independently calculated for any network and parameters that the models are run on. As each set of parameters and the input network define a unique spreading scenario, measures such as the basic reproduction number can always be calculated for any combination thereof.

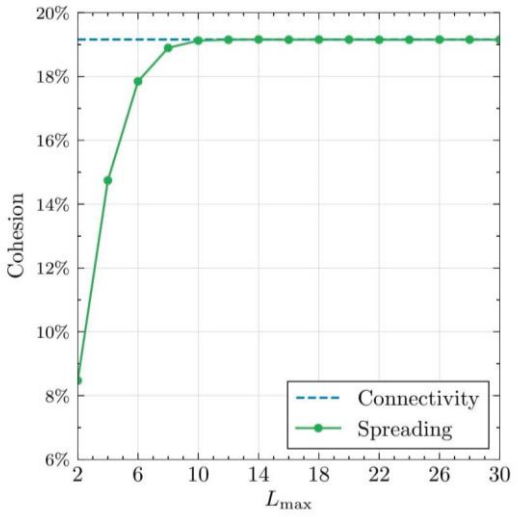
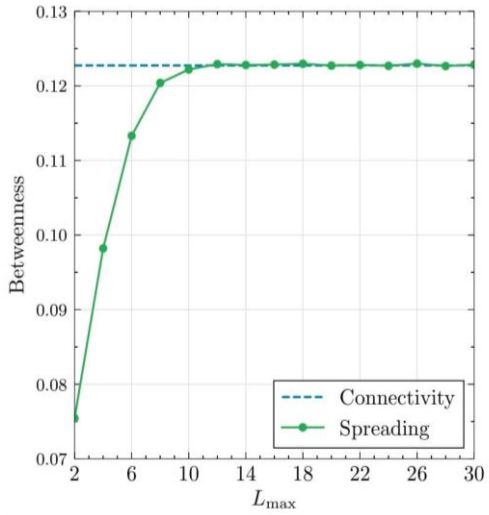


Figure 4.2 (a) Student network cohesion



(b) Student network betweenness centrality

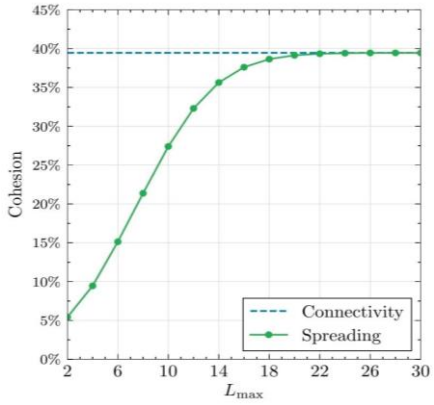
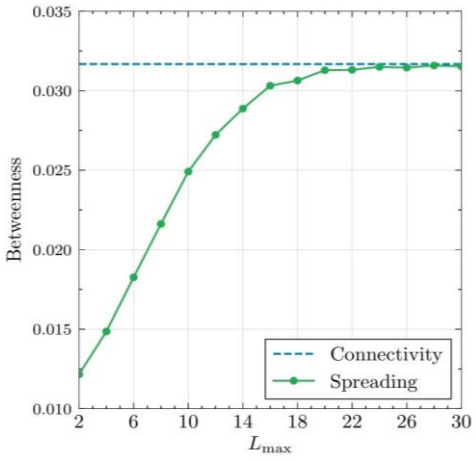


Figure 4.3 (a) Organisation Network Cohesion



(b) Organisation Network Betweenness Centrality

Figure 4.2:The normalized cohesion and betweenness centrality for the student network as a function of L_{max} calculated with 10, 00,000 iterations in the Connectivity and Spreading models.

Figure 4.3: The normalized cohesion and betweenness centrality for the Organisation network as a function of L_{max} calculated with 10,00,000 iterations in the Connectivity and Spreading models.

For both networks, we calculate the total cohesion (Eqs.4.4) and mean betweenness centrality (Eqs.4.6) using both models and varying L_{max} for the Spreading model. We normalise the total cohesion by the number of values constituting its sum, N (N − 1), where N is the number of nodes in the network, to get the node-wise average in- and out-centrality. This scaling factor is attained from the probability matrix being of shape N × N with the diagonal consisting of N missing values. The sum of the in- and out-centralities (Eqs.4.2 and 4.3) are equal to the cohesion:

$$\sum_{t\in V} C^{(in)}(t) = \sum_{t\in V}\sum_{\substack{s\in V\\ s\neq t}} C(s,t) = C = \sum_{s\in V}\sum_{\substack{t\in V\\ s\neq t}} C(s,t) = \sum_{s\in V} C^{(out)}(s) \qquad (4.7)$$

From equation (4.7), the averages of the in- and out-centralities are also equal. This means that the normalized cohesion represents both the average node-wise in- and out-centralities. The mean betweenness centrality is calculated as the average of the node-wise betweenness centralities. The minimum L_{max} value, for which the results of the models are ideally equal (assuming arbitrary precision), is the maximum length, for which a self-avoiding path exists in the network, since then the Spreading model is able to simulate spreading throughout the whole network. A self-avoiding path is never longer than the number of nodes in the network, which gives a trivial upper bound. Thus, as L_{max} increases, the results of the Spreading model approach those of the Connectivity model. In practice, this happens with L_{max} much lower than N (Figures 4.2 and 4.3). This is due to the probability of spreading through a path decreasing exponentially with the path length.

The cohesions of the Student and Organisation networks are around 19% and 40%, respectively (Figures 4.2a and 4.3a). The Organisation network achieves a higher cohesion due to its much denser nature. The difference in density can also be seen in Figures 4.2b and 4.3b, where the Student and Organisation networks' betweenness centralities (Eq. 4.6) are around 0.12 and 0.03, respectively. Nodes in the Student network are connected by much fewer paths than in the Organisation network, and therefore each node has a more prominent role in allowing connections between other nodes.

As the results given by the Spreading model converge already with lower values of L_{max}, epidemic simulation can be performed more efficiently by not taking longer spreading paths into account. With a lowL_{max}, however, the results differ, which allows the simulation of scenarios where spreading paths are limited by factors such as consistent isolation of patients. Since the two models produce identical results with high L_{max}, both models can be used for the intended purpose of the other.

The models can be run on any network with any edge and node weights but are capable of modelling only scenarios where any one node is influenced at most once. In our earlier studies, we have also presented a model where all nodes can get influenced any number of times. We will present a model capable of dealing with situations where getting influenced decreases a node's probability of getting influenced again by a given breakthrough probability in our future studies. [1].

5. EXAMPLES OF PRACTICAL APPLICATIONS

5.1 Health

5.1.1 Influenza

Consider an epidemic of influenza in a British boarding school. Three boys were reported to the school infirmary with the typical symptoms of influenza. Over the next few days, a very large fraction of the 763 boys in the school had contact with the infection. Within two weeks, the infection had become extinguished. The best fit parameters yield an estimated active infectious period of $\frac{1}{\gamma} = 2.2$ days and a mean transmission rate $\beta = 1.66$ per day. Therefore, the estimated R_o is 3.652. Figure 1 represents the dynamics of the three state variables. It can be observed that the curve of susceptible is decreasing all over the time, because the birth was no considered, and once become infected never returns to the state of susceptible. The curve of infected reaches to a peak of the disease beyond 5 weeks. This information could be very useful for health authorities to ensure that all resources are available - medicines, doctors, hospitalization resources – to provide a good health care if necessary. Depending of flatness of the curve the response should be adaptive. The curve related to the recovered compartment is important because accumulates the number of individuals that have been seek in that outbreak.

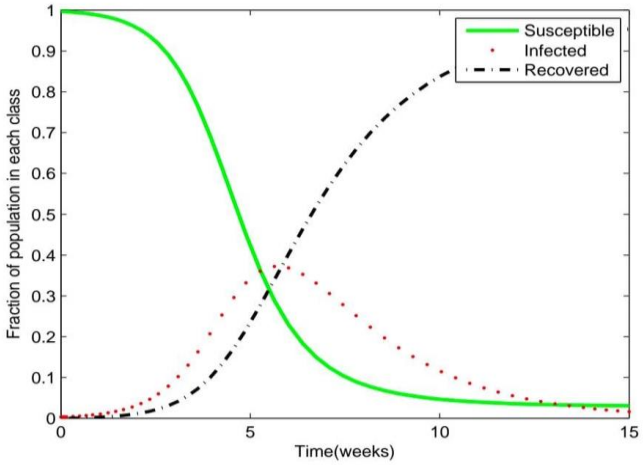


Figure 5.1 The time involving of influenza over 15 days

In other example the curves do not be shown, instead the only thing that are changed are the parameters values. The main goal of this paper is not to show all the graphics related to SIR model, but to present a set of applications in several fields.

5.1.2 Dengue Fever

Dengue is a vector-borne disease transmitted from an infected human to a female Aedes mosquito by a bite. Then, the mosquito, that needs regular meals of blood to feed their eggs, bites a potential healthy human and transmits the disease making it a cycle. Nowadays, Dengue is the mosquito-borne infection that has become a major international public health concern. According to the World Health Organization (WHO), 50 to 100 million Dengue Fever infections occur yearly, including 5,00,000 Dengue Hemorrhagic Fever cases and 22,000 deaths, mostly among children.

This global pandemic is attributed to the unprecedented population growth, the rising level of urbanization without adequate domestic water supplies, increasing movement of the virus between humans (due to tourism, migration, or international trade), and lack of effective mosquito control. Dengue virus is transmitted to humans through the bite of infected Aedes mosquitoes, specially Aedes Aegypti. Once infected, a mosquito remains infected for life, transmitting the virus to susceptible

individuals during feed. Without a vaccine, vector control remains the only available strategy against dengue. Appropriate mathematical models can give a deeper insight into the mechanism of disease transmission. In this particular disease, the SIR model associated to the human population, usually is coupled to a SI model for the mosquito, due to the vector transmission process.

5.1.3 SARS

The Severe Acute Respiratory Syndrome (SARS) was the first epidemic of the 21st century. It emerged in China late 2002 and quickly spread to 32 countries causing more than 774 deaths and 8098 infections worldwide.

SARS is a highly contagious respiratory disease which is caused by the SARS Coronavirus. It is a serious form of pneumonia, resulting in acute respiratory distress and sometimes death. The SARS epidemic originated in China, in late 2002. Although the Chinese government tried to control the outbreak of the SARS epidemic without the awareness of the World Health Organization (WHO), it continued to spread.

5.2 Networks

5.2.1 Online social networks

The last decade has rise a huge number of online social networks. Several papers have studied, under epidemiological models, the adoption or abandonment of online social networks. Cannarella and Spechler studied the information diffusion on Twitter, in order understand the properties of underlying media and model communication patterns; with the popularity of Twitter it become a venue to broadcast rumors and misinformation.

Wang and Wang investigate a SIR model to study rumor spreading. With the development of microblogging technology, it become easy to publish several messages on the network websites, and also for other people to be able to visit these websites to search for messages according to their own needs, increasing rapidly the social network.

5.2.2 Viral Marketing

Viral marketing (VM) is a recent approaching to markets and can potentially reach a large and fast audience, through a cheap communication campaigns. VM exploits existing social networks by encouraging people to share product information and campaigns with their friends, through email or networks medium. This type of communication has more impact in the customer, because the information was recommended by friends and peer networks that knows the personal interests, instead of standard companies; this kind of communication have more impact because is directly targeted. Besides When a marketing message goes viral, it is analogous to an epidemic, since involves a person-to-person transmission, spreading within a population. Rodrigues and Fonseca explored a set of simulation experiments to explore the influence of several controlled and external factors that could influence viral campaigns.

Also known as internet worth of mouth marketing, VM has been gaining more fans, from professionals to researchers, as an alternative strategy to traditional communication, transferring funds from companies to online marketing actions and exploring these spreading phenomena.

5.2.3 Audience applause

The social identity and crowd psychology study how and why an individual change their behavior in response to others; within a group, a distinct attitude can arise in a few persons and then spread quickly to all other members.

According to Mann et al. individuals' probability of starting clapping increased in proportion to the number of other audience members already affected by this social contagion. In this paper, the authors apply a Bayesian model selection approach to determine the dynamics of how some details or social cues can provoke the spread of social behavior in a group of people. They reach to the conclusion that the audience clapping can vary, even when the quality of the presentations are identical, changing according to the set of infected people.

5.2.4 Diffusion of ideas

The population dynamics underlying the diffusion of ideas hold many qualitative similarities to those involved in the spread of infections. Bettencourt et al. explore this point of view as a tool to quantify sociological and behavioral patterns. They explore the spreading of Feynaman diagram through the theoretical physics communities of the USA, Japan, and the USSR in the period immediately after World War II; having this in mind they investigate the effectiveness of the adoption of an idea, finding values for parameters that describe intentional social organization and long lifetimes for the idea.

By other hand, Funk, explore the concept of epidemiology in the human behavior when public campaigns and mass media reports are diffused. The spread of awareness is crucial in this model to describe the susceptible person to become convinced or informed to the disease and have additional precautions related to the disease transmission process.

5.3 Informatics

5.3.1 Peer-to-peer (P2P) networks

Understanding the spread of information on complex networks is crucial from a theoretical and applied perspective. To evaluate them with large-scale real-world data remains an important challenge.

During the downloading process, the peer shares the downloaded parts of the file and, thus, contributes to distributing it in the network. The authors consider a file sharing application similar to eDonkey which belongs to the class of hybrid P2P architectures and apply the SIR model, that corresponds to the populations of idle peers, peers currently downloading the file, and those sharing it. Bernardes et al. asses the relevance of the SIR model to mimic key properties of spreading cascade of a file sharing.

5.3.2 Spread of computer virus

Nowadays, with the rapid development of network information technology, information networks security has become a very critical issue in our work and daily life. The computer virus is being developed simultaneously with the computer systems and the use of internet facilities increases the number of damaging virus incidents, producing serious problems for individuals and corporate computer systems. Antivirus software is the major means of defending against viruses. Although, antivirus technique cannot predict the evolution trend of viruses and, hence, cannot provide global suggestions for their prevention and control. The strong desire to understand the spread mechanism of computer viruses has motivated the proposal of a variety of epidemic models that are based on fully connected networks, that is, networks where each computer is equally likely to be accessed by any other computer.

Computer virus is considered as one of the most important weapon in the internet, and their emergence and spread may have great effect on the computer world. Different codes have different ways to spread in the internet. Virus mainly attack the file system and worm uses system vulnerability to search and attack the computer. And for trojan horses, they camouflage themselves and thus induce the users to download them. There are a variety of computer virus, but they all have infectivity, invisibility, latent,

destructibility and unpredictability. The word latent means that the virus hides themselves in the computer and spread them in the internet while the users can not notice them.

5.4 Economics and Finance

5.4.1 Rational expectations

The economic epidemiology merges the epidemiological models with economic choice, translating a rational decision making. Economic research in this area began in response to the AIDS epidemic and has led to an improved understanding of the thought/decisions towards a infectious disease, by anonymous individuals or policymakers. the power to eradicate an infectious disease is not only in the hands of policymakers or health authorities: it is also important that rational individuals made their own response to lower the prevalence of a disease, by increasing protection. Economic epidemiology has made significant advances in educating health officials about the behavioral implications of public policies. Aadland et al. explored the nature of the short-run equilibrium dynamics for rational expectations economic epidemiological systems. They show that well-intentioned policy has the potential to create instability when people behave rationally and in a self-interested manner.

5.4.2 Financial network contagion

The financial sector is always a theme of interest, due to its importance in economy in general, and our daily lives in particular. analyze the importance of individual bank-specific factors on financial stability. The spreading of the contagion in the interbank network can be seen as an epidemiological model. The authors investigate the systemic risk and how this risk can propagate in different bank and countries within the euro area. Fisher makes counterfactual simulations to propagate shocks emerging from three sources of systemic risk: interbank, asset price, and sovereign credit risk markets. when the conditions deteriorate, these channels trigger severe direct and indirect losses and cascades of defaults, whilst the dominance of the sovereign credit risk channel amplifies, as the primary source of financial contagion in the banking network.

5.5 Science Fiction: Zombie's attack

In 2009, the first mathematical investigation of the zombie community appears. Taking their cues from traditional zombie movies. hypothesized the effect of a zombie attack and its impact on human civilization. According to their mathematical model, "a zombie outbreak is likely to lead to the collapse of civilization, unless it is dealt with quickly. While aggressive quarantine may contain the epidemic, or a cure may lead to coexistence of humans and zombies, the most effective way to contain the rise of the undead is to hit hard and hit often." The model showed two equilibria: the disease-free equilibrium (with no zombies) and the doomsday equilibrium (where everyone is a zombie). The application of a linear stability analysis showed that - in the absence of further interventions - the disease-free equilibrium was unstable and the doomsday equilibrium was stable. Since this paper, other authors follow this area with a careful attention not only motivated by the tv series, but as a way to motivate young students for the epidemiology issues [8].

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