Modelling Infectious Diseases on Small World Network

By

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June 2017

AIMS | African Institute for Mathematical Sciences
RWANDA

DECLARATION

This work was carried out at AIMS Rwanda in partial fulfilment of the requirements for a Master of Science Degree.

I hereby declare that except where due acknowledgement is made, this work has never been presented wholly or in part for the award of a degree at AIMS Rwanda or any other University.

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ACKNOWLEDGEMENTS

DEDICATION

This is optional.

Abstract

A short, abstracted description of your essay goes here. It should be about 100 words long. But write it last.

An abstract is not a summary of your essay: it's an abstraction of that. It tells the readers why they should be interested in your essay but summarises all they need to know if they read no further.

The writing style used in an abstract is like the style used in the rest of your essay: concise, clear and direct. In the rest of the essay, however, you will introduce and use technical terms. In the abstract you should avoid them in order to make the result comprehensible to all.

You may like to repeat the abstract in your mother tongue.

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2.1   Zika Virus

Zika virus is a member of the Flaviridae family and the Flavivirus genus. It is related to other mosquito borne viruses such as Dengue virus, yellow-fever virus (YFV) and West Nile virus (Dick et al., 1952). The virus originates from the Zika forest of Uganda and the first case was isolated in 1947 from a rhesus monkey in the forest. Then later in 1954 a human was diagnosed with the virus in Nigeria (Junior et al., 2015). Since then the virus has spread to different parts of the world.
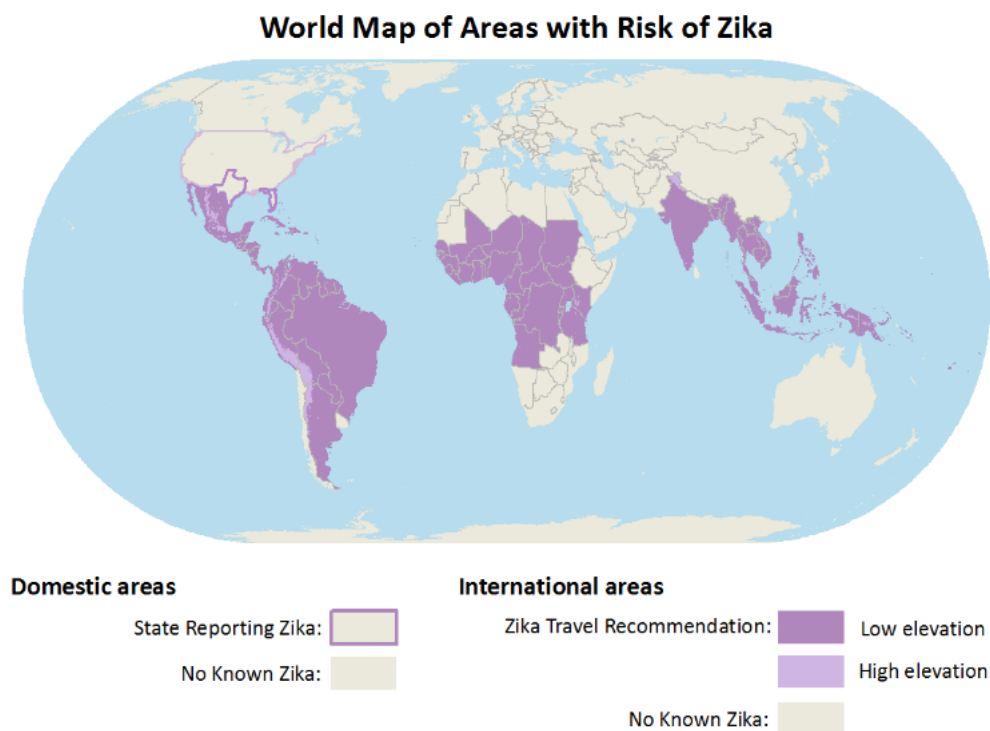

Figure 2.1: Countries with Zika Virus: Source CDC

Zika Virus is mainly spread by Aedes mosquitoes. When a pathogen carrying mosquito bites an uninfected individual it infects them with the virus. Other ways by which the virus spreads include, blood transfusion, unprotected sex with an infected person and from mother to child, infected mothers can pass on the virus to their unborn children (Musso et al., 2014).

Some of the symptoms of Zika virus are papular rash,fever, arthritis or arthralgia Musso et al. (2015).Papular rash is characterized by a flat, red area on the skin that is covered with small confluent bumps and arthralgia is characterised by pain or aching in the joints without pain.

In addition, headache and red eyes are common symptoms of Zika virus. Simões et al. (2016) adds that the extent of the risk that Zika infection will result in birth defects still remains unknown. For expecting mothers, Zika virus affects their foetus and development of the baby. Babies can face a range of neurologic sequelae such as intellectual disability, hearing loss, vision loss, and seizures. These problems can range from mild to severe and are often life-long (Rasmussen et al., 2016).

There is no known vaccination to prevent or treat the Zika virus. Prevention measures can be taken to prevent the spread of the virus. This done by preventing mosquito bites. Measures such as sleeping under a mosquito net, using mosquito repellent and fumigating mosquito breeding areas in the vicinity among others can be taken. Another measure of prevention of Zika virus is practising safe sex and avoiding travel to areas with high prevalence of Zika.

Drugs for the symptoms of Zika are administered to patients as a way of treating Zika patients because of the lack of a vaccine for the virus.

The spread of Zika virus has resulted in Zika epidemics in some part of the world as can be seen in figure 2.1 above as of April 2017. This causes a worry as the effects of the epidemic are more devastating and if not controlled can affect the whole country, region and World at large.

2.2   Epidemiology

Epidemiology is the study of the origin and course of diseases in a community. The goal of epidemiologist is to understand the cause of a disease, then to predict its course, and come up with methods to control the disease. This involves collaborative work of statisticians, mathematicians, physicians and various health specialists (Brauer, 2017).

The knowledge about infectious diseases has been built up by the method of experience, by observation and analysis of particular conditions associated with occurrence of the disease in nature (Frost, 1923).

The first step in epidemiology was the collection and analysis of data on causes of death in London parishes in the early 1660s by Graunt. He gave a method of estimating the comparative risks of dying from various diseases, giving the first approach to a theory of competing risks (Brauer, 2017).

Mathematical models of disease transmission have been used to link biological processes of disease transmission and emergence of dynamics of infections at the population level. Researchers try to

understand the environmental, biological and behavioural infectiousness of a disease.

Environmental infectiousness depends on geographical factors of an infected person. Some pathogens cannot survive inside or outside a host in given geographical conditions. Thus, some diseases or infections spread faster in certain weather conditions (Grassly and Fraser, 2006). Understanding the timing and causes of seasonality offers important insights on how parasite–host systems interact. How and when parasite control measures can be applied, and how disease risks will respond to anthropogenic climate change and altered patterns of seasonality (Altizer et al., 2006). These factors must be captured in the models.

Biological infectiousness depends on the pathogen's life cycle and the individual's or host's immune system. Some individuals have strong immunity against certain infections, this may slow down the propagation the infection. On the other hand the life cycle of pathogens also affects the transmission dynamics of the infection.Some pathogens can only survive in the host while the other can survive outside the host, this will play a major role in the spread of the infection. The interaction of the genetic determinant of disease propagation in the pathogen and host is important in building models for the transmission dynamics of infectious diseases.

Behavioural infectiousness depends on the interaction behaviour of an individual. The contact pattern of the person affects how the individual is likely to propagate the disease. Depending on the nature of disease transmission, a person who has a lot of contacts is are more likely to spread the disease to more people compared to one who has fewer contacts (Johnson et al., 2001). Contact in this context implies any interaction likely to result in transmission of an infection.

The susceptibility of an individual largely depends on the biological, environmental and behavioural factors of an individual. For example, one's contact pattern, immunity and the environmental conditions will highly affect the probability of contracting an infection.

Over a century, Mathematical representation and analysis of infectious diseases has been the centre of infectious disease epidemiology (Beisner, 2005).

Mathematical modelling of infections diseases, started by the works of Daniel Bernoulli in Bernoulli (1760),in the quest to model the spread of small box and possible eradication. A century later the modelling become well established. The modelling of infectious disease dynamics is important for science and public policy among others. There are three main aims of infectious disease modelling; to is to understand the how the spreading mechanism of the disease, to predict how the disease will progress among the population and to understand how the disease can be controlled. They provide tools for investigating and quantifying the spread of disease dynamics. Conducting experimental research on the spread of infectious disease raises a lot of ethical issue and therefore can not be conducted on humans. Mathematical simulations and modelling the disease has helped in providing understanding the impact of the infectious disease on the population and give a guide to new control measures (Ming et al., 2016).

2.3   Modelling with Compartmental Models

Differential equations have been used in the modelling of the dynamics of the spread of infectious diseases. They are based on the assumption of uniform mixing, that is, everyone in the population has an equal probability of contracting an infectious disease (Kaplan et al., 2002). Compartmental Mathematical models have been used to describe the transmission dynamic of Zika Virus (Gao et al., 2016). Infectious diseases are transmitted indirectly or directly by contact between the infected and those who are not infected thus these models try to capture these interactions (Sattenspiel, 1990).

In compartmental models of infectious disease individuals are divided into several compartments such as; Susceptible (S) , latent (E) , infected (I), vaccinated (V) and recovered or removed (R). Depending on the on the propagation of the disease, compartment models are built by combining these different classes or creating new ones (Li and Muldowney, 1995).

Deterministic models also know as compartmental describe and explains what happens on average of the population. They assume that the population is homogeneous, that is, everyone in the population reacts the same risks of exposure and infection. This assumption in some cases has proven not to be realistic and hence the introduction of stochastic models. Stochastic models introduce the idea of randomness in the reaction to risk and infection by individuals in the population (Ming et al., 2016). The main advantage of the stochastic model is they take into consideration each individual, but the major drawback is that it is laborious to model them as they require a lot of stimulation and sometimes become mathematically complex.

Deterministic models are studied either by theory or methods.   [Jan: I don't understand sentence above.]   Ordinary differential equations, partial differential equations and difference equations   !   (Keeling and Rohani, 2008). The trends in these research areas are for higher model dimension and deeper and more refined analysis. Unlike for stochastic models where the trends of research in these models are toward specific diseases and toward deterministic and stochastic mixed models (Fu et al., 2013).

To build models that incorporate contact patterns of the individuals, Mathematicians have resolved to use results from the work of Moreno (1945) where he analysed contact patterns of prisoners. This work has given basis for understanding or building models based on the contact patterns of individuals in the population (Sattenspiel, 1990). Freeman (2004) characterizes the analysis of social networks by four properties. First, it involves the intuition that links between social actors are important. Second, it is based on the data collection and analysis of data about social relations that link actors. Third, it draws heavily on graphic imagery to reveal and display the patterning of those links and lastly, it develops mathematical and computational models to describe and explain those patterns. A number of disease propagation models have been built for various infectious diseases among others Malaria, Zika, HIV, Small pox and Chicken pox (Ding et al., 2016)

2.4 Modelling with Graph

Graph theory has over the years grown and has found its application in many fields. A graph also known as a network can be defined as couple $G = (V, E)$ where V is a finite set of nodes $E \subset V \oplus V = \{e_1, e_2, \ldots, e_m\}$ is a set (Estrada, 2012).

Over the years contemporary science, has had challenges in describing complex networks. This posed limits in the advancement of many disciplines. However, with the advancement of computerization, there is a raise in the possibility of understanding the stability of large networks Barabási and Albert (1999).

For many complex networks vertices are described as elements of the system and edges represent the interaction between them. Similarly, in modelling the spread of infectious diseases on networks, individuals or populations are represented by nodes of the network, contacts likely to result in the transmission of disease are represented by edges. Modelling of infectious disease on networks give better models for heterogeneous populations (Ming et al., 2016). One of the major challenges in modelling the spread of infectious diseases on networks, is capturing the contact patterns of individuals. The non availability of such data has lead to mathematicians modelling the spread of infectious diseases on various simulated network structures (Pastor-Satorras and Vespignani, 2001).

Random network models of infectious disease do not take into account spatial position of individuals and connections are made at random (Keeling and Eames, 2005). The growth rate and and final epidemic size of a disease on a random network are reduced compared with a random mix model. Growth rate in random network is $\tau(n-2) - g$ and the growth rate with random mixing is $\beta - g = \tau \widehat{n} - g$, where τ is the transmission rate across a contact, n and $\widehat{n}$, is the number of contacts in a network and the unit number of contact per unit time in a random mixing model respectively. g is the probability of recovery.

The reduction in the growth rate is due to two reasons; each infectious individual has been infected by one of its contacts, reducing the number of susceptible $n - 1$ and as an infectious individual starts to infect its susceptible contacts it depletes its local environment, regardless of the population prevalence rate, hence limits the rate of disease spread (Keeling and Eames, 2005).

Lattice based epidemic models are used to study the spatial and temporal rates of the disease spread in a spatially distributed host populations (Rhodes and Anderson, 1997). Models built on latices assume that individuals are located as nodes on a regular lattice and connections are made to a collection of near neighbours or each node. For example people may be spread out such that connections are made to their four nearest neighbours, one on the left,right, up and down. (Lloyd et al., 2006).To avoid the effect of the nodes at the end not being connected the last and first neighbours are made neighbours. The spread of influenza is on of the infectious disease that have been modelled based on lattice models Liccardo and Fierro (2013).

Another kind of network epidemic model has been the small world network. Disease spread through small-world networks has received considerable attention from both a theoretical and more applied context. The high level of clustering means that most infection occurs locally, but short path lengths mean that epidemic spread through the net-work is rapid and disease is unlikely

to be contained within small regions of the population (Watts and Strogatz, 1998).

The spread of infectious diseases on network has also has also been modelled on scale free networks. Infectious disease like Ebola,SARS and HIV-AIDS have been modelled on scale free networks. A scale free network is a network whose degree distribution follows a the power that is $P(k) \approx ck^{-\lambda}$ (Morita, 2016)

Simulated network models have been used to model the spread of disease, whose network data is difficult to collect. Various infectious diseases have been modelled on simulated networks (Keeling and Eames, 2005). Networks that are based on simulation are limited because they are no ways to test the sensitivity of the epidemiological results to the details of the network structure. Nevertheless, a range of idealized networks and analytical tools that can reveal elements of network structure that are important determinants of epidemic dynamics, have been developed (Keeling and Eames, 2005).

A number of infectious disease models have been built on various network structures. This is so because networks capture the contact patterns in a community. The networks are either social networks or simulated networks. Many real world and social networks in which infectious diseases propagate is either small world or scale free networks and not random or regular as earlier assumed Watts and Strogatz (1998). The underlying structure of a network influences the effect of that the dynamics of epidemics will have on a population. For example in a small world network, where the network has a high clustering coefficient a shorter average distance. A disease is more likely to spread faster than in a random network or a regular network (Watts and Strogatz, 1998).

3. Background

3.1 Definitions

3.1.1 Epidemic and Pandemic . An infectious disease is a disease that is caused by pathogenic microorganisms, such as bacteria, viruses, parasites or fungi; these diseases can be spread, directly or indirectly, from one person to another. An epidemic is a situation where an infectious disease is affecting many people at a particular time and spread at a very high rate. A pandemic on the other [Jan: hand] is an epidemic over a large area (Morens et al., 2009). !

3.1.2 deterministic and stochastic infectious disease models. [Jan: Fix capitalization for subsection titles.] The dynamics of infectious disease propagation are modelled as a dynamical ! system. A dynamical system is a system that evolves with time over a state space according to a fixed rule. Thus, let $\mathbb{X}$ be a state space $\mathbb{T}$ set of times and $\mathbb{R}$ rule that specifies how the state evolves with time. the rule is a function whose domain is $\mathbb{X} \times \mathbb{T}$ and co-domain $\mathbb{X}$ that is,

$$\mathbb{R} : \mathbb{X} \times \mathbb{T} \longrightarrow \mathbb{X}.$$

This means that $\mathbb{R}$ takes two arguments $(\mathbf{x}, t)$ where $\mathbf{x} \in \mathbb{X}$ is the initial state and $t \in \mathbb{T}$. That is $\mathbb{R}(\mathbf{x}, t)$ give [Jan: s/give/gives] the state of the system at time t given the initial state of the ! system was $\mathbf{x}$ (Nykamp, 2017).

The population can be characterized as $S(t)$, $E(t)$, $I(t)$ and $R(t)$. Where $S(t)$ is the number of individuals susceptible but not infected at time t, $E(t)$ is the number of people exposed or infected, but not infectious at time t, $I(t)$ is the number of infected and infectious people at time t and $R(t)$ is the number of people whose ability to be infected is removed, by either immunization, death or recovery from the disease. The epidemiological models can be classified as Susceptible, Infected and Recovered (SIR), Susceptible Infected (SIS or SIS), Susceptible -Exposed - Infectious and Removed (SEIR) [Jan: Make use of dashes consistent.] and by any other states the population ! may be partitioned into. [Jan: It feels to me you are contradicting yourself. If you assume continuous time, I don't think it is accurate to represent the disease evolution as "rule" $\mathbb{R}$. may need your help in this] !

[Jan: Previous para: First introduce different compartment schemes, then describe SEIR as example.] !

The SIS model assumes that there is no immunity after recovery. It is used to model infections were once a person recovers, they become susceptible again. For example infections like flu. [Jan: Change to: "An example of such an infection is flu."] The SIR model assumes that once a person ! recovers, they become immune to the infection for example chicken pox. [Jan: Comma before "for example". "Chickenpox" is spelled together.] The SEIR assumes that once a person becomes ! infected, they do not become infectious immediately hence the intermediate compartment for exposed.

The independent variable in the compartmental model is the time t.The rates of transfer between compartments are expressed mathematically as a result models are formulated initially as differential equations. Most epidemic models are built on the SIR model (M'Kendrick, 1925). The

8

268 system can be written as;

$$
\begin{array}{rcl}
\frac{dS}{dt} & = & -\alpha S(t)I(t), \\
\frac{dI}{dt} & = & \alpha S(t)I(t) - \gamma I(t), \\
\frac{dR}{dt} & = & \gamma I(t),
\end{array}
\tag{3.1.1}
$$

$$
\frac{dS}{dt} = -\alpha S(t)I(t),
$$
$$
\frac{dI}{dt} = \alpha S(t)I(t) - \gamma I(t),
$$
$$
\frac{dR}{dt} = \gamma I(t),
$$
(3.1.2)

269   [Jan: Use align instead of array for multiline equations. See my code above how to do that.]   **!**

270   [Jan: Say that α and γ are parameters of the model.] with assumptions that there is homogeneous   **!**
271   mixing in the population. That is the rate of new infections is proportional to the current
272   numbers of susceptibles and infectives in the population. Which [Jan: s/Which/This is] the main   **!**
273   assumption deterministic models are built on. Deterministic population models are models where
274   the behaviour of the population of determined completely by history and the rules which govern
275   the model. In formulating these models, in terms of derivatives of the sizes of the compartments
276   and it is assumed that the number of members in each compartment is differentiable with time.
277   This assumption is tenable only when the disease outbreak has been established, but not valid at
278   the beginning of a disease outbreak, when they are few infectives. When they are a few infectives,
279   the number of infectious depends on random contacts of between a small number of individuals
280   (Brauer and Castillo-Chavez, 2012). [Jan: There are multiple thoughts in this para, split it.]   **!**

281   On the other hand, life phenomena are in general stochastic in nature and the dynamics cannot
282   be well captured by deterministic models hence a resolve [Jan: s/resolve/need] for stochastic   **!**
283   models. Stochastic models take into account random variations associated with environment and
284   biological fluctuations of the factors that affect disease propagation. These random fluctuations
285   may impact the evolution of the infection. Unlike deterministic model which assume homogeneous
286   mix, an assumption which only holds in small populations. It is quite unlikely that all people will
287   be equally susceptible to the disease and and effective in spreading it (Ball, 1985).

288   There are a number of different stochastic modelling processes, such as discrete time Markov
289   chain model, continuous time Markov chain models and stochastic differential equations model.
290   These models differ in underlying assumptions regarding the time and variables.

For example, lets [Jan: s/lets/let us. There are more typos in this para.] take an SIR compartmental   **!**
model. S, I and R repents compartments as well as the number of individuals in each compartment
and we assume that $S_t + I_t + R_t = N$ [Jan: Be consistent with notation: $S(t)$ etc.] is constant.   **!**
Time $t > 0$ is continuous for each state S_t, I_t and R_t. Let β to be the average number of
contacts an infectious person makes per unit of time that take leads to infection. The probability

of a susceptible individual moving from compartment S to compartment I in the time interval $[t, \triangle t]$ that is $S \to S - 1$ and $I \to I + 1$ is β S I $\triangle t + o(\triangle t)$. [Jan: I think you should normalize by N here.] If it is assumed the an infected person recovers at the rate γ hence the probability of an infected person moving from infected to recover over an interval $[t, \triangle t]$ given by $\gamma I_{t+\triangle t} - o(\triangle t)$. It is known that, [Jan: s/It is known that,/Since] [Jan: Write just the second equation.]

$$N_t = S_t + I_t + R_t$$
$$\Rightarrow R_t = N_t - S_t - I_t,$$

which implies that knowing S_t, I_t is knowing R_t. Hence the model becomes an S_t, I_t and thus the stochastic dynamical system can be written as;

$$P((S_{t+\triangle t}, I_{t+\triangle t} - (S_t, I_t) = (-1, 1)) = \beta S I \triangle t + o(\triangle t). \qquad (3.1.3)$$
$$P(((S_{t+\triangle t}, I_{t+\triangle t} - (S_t, I_t) = (0, -1)) = \gamma I_{t+\triangle t} - o(\triangle t). \qquad (3.1.4)$$

291   With a complementary equation,

$$P((S_t + \triangle t, I_t + \triangle t) - S_t, I_t) = (0,)) = 1 - \left(\beta \frac{S_t}{N} \right) I_t \triangle t + o \triangle t \qquad (3.1.5)$$

292   [Jan: Fix typos in equation above.] which is refereed to as the general stochastic epidemic model
293   Greenwood and Gordillo (2009). [Jan: Use \citep above.]

294   **3.1.3 Network.** A graph also known as a network can be defined as couple $G = (V, E)$ where
295   V is a finite set of nodes $E \subset V \oplus V = \{e_1, e_2, \dots, e_m\}$ is a set (Estrada, 2012). Nodes can be
296   human beings, cities or houses while edges could be any connection such as friendship, physical
297   connection or road.

298   A network is said to be connected if there exists a path between any two nodes in the network.
299   Distance between any two nodes in a network is defined as the length of the shortest path by
300   which each a node can be reached. [Jan: s/by which each a node can be reached/between them]
301   This can be summed up as the average distance taken over all pairs of vertices, which give the
302   idea of the typical distance between nodes in a network. The diameter of the graph is the largest
303   distance taken over all pairs.

304   **3.1.4 Statistical Characterization.** Networks can be characterized by the following statistical
305   properties.

306   i **Degree distribution** The degree of a node is the number of connections to other nodes, a
307   particular node has and is denoted as k and the average distribution [Jan: What is "average
308   distribution"?] of a network is denoted by $\langle k \rangle$.

309   Looking at the entire space or network one can obtain a distribution for the degree. Let $n(k)$
310   be the number of nodes of degree k in a network of size n, $p(k) = \dfrac{n(k)}{n}$. $p(k)$ represents the
311   probability that a node selected uniformly at random has degree k. the degree distribution is
312   obtained by plotting $p(k)$ against k(Estrada et al., 2015). [Jan: Space before parenthesis...]
313   The common distribution found in the network are normal distribution, exponential, power
314   law distribution(Chung and Lu, 2002).

ii **Clustering** A cluster in a network is a collection of nodes which are similar among them and are dissimilar to other nodes belonging to other clusters. Clustering in friendship network may mean [Jan: s/mean/signify] friends people have in common. Local clustering ! in a network is measured by the Watts -stoggatz [Jan: Correct spelling, capitalization and spaces...] coefficient and the global clustering by Newman clustering coefficients. !

Watts -strogatz average clustering coefficient is given by

$$\overline{C} = \frac{1}{n} \sum_i c_i \tag{3.1.6}$$

$$c_i = \frac{2t_i}{k_i(k_i - 1)}$$

where t_i the number of triangles attached to node i of degree k_i. The Watts-Strogatz clustering coefficient of a node quantifies how close its neighbours are close to making a clique. In terms of friends it quantifies how one's friends are friends with each other. The clustering coefficient lies between 0 and 1, if its zero, then no two nodes of a node's neighbours are connected and if it is 1 then all the neighbours of a node are connected to connected to each other.

The Newman clustering coefficient is given by

$$c = \frac{3t}{p_2} = \frac{3|c_3|}{p_2} \tag{3.1.7}$$

where $t = c_3$ number of triangle in the network and $|p_2|$ the number of closed paths of length 2.

The Newman clustering coefficient or so know as the network clustering coefficient, gives the average clustering for the whole network. Node with less than two neighbours are given 0 as the clustering coefficient. [Jan: I don't get previous sentence.] It looks at the clustering over the ! whole network. [Jan: Put this para inside itemize.] !

In a social network if a person A is friends with person B and B friends with C most likely [Jan: ! s/most likely/it is likely that] A will be introduced to C and the two will know each other. This ! will result in the three forming a triangle. The clustering coefficient either global or local gives a proportion of how many such triangles are there and how many are likely to exist. The local clustering coefficient will give this value in relation to a particular node while the global clustering coefficient will give the value over the entire network (Estrada et al., 2015).

A network is said to be small world or to exhibit small world properties if its Newman clustering coefficient is greater than the Watts- strogatz clustering coefficient. That is a small world network has a high clustering coefficient and a low average distance (Estrada, 2012). A small world property can be defined as let D be the average distance between any pair of vertices in a network, if D increase proportionally to the logarithm of the size of the network N (Newman and Watts, 1999). there are multiple definition That is,

$$D \propto \log N \tag{3.1.8}$$

[Jan: The problem with this paragraph is that actually our network does not have some of those ! properties. For example, I'm pretty sure that for us both clustering coefficient will be close to 0.]

347 **3.1.5 Random Graphs.** A random graph $G(n, p)$ can be defined as , given N [Jan: Be consistent
348 with capitalization of n.] number of vertices, edges between them are drown such that between [!]
349 any pair i, j there is an edge with uncorrelated probability p.

350 Taking z to be the average degree. [Jan: Put a verb in this sentence.] The probability p of an edge [!]
351 being present between any two vertices is given by $p = \frac{z}{n-1}$, for large n can be approximated by
352 $\frac{z}{n}$ (Newman et al., 2002). The number of nodes connected to any particular vertex, [Jan: Delete
353 first part of this sentence.] the degree k of that vertex has a probability distribution p_k given by; [!]

$$p_k = \binom{n}{k} p^k (1 - p)^{n-k} \approx \frac{z^k e^{-z}}{k!}. \tag{3.1.9}$$

[Jan: This is only for constant k and n going to infinity.] For example, let us take 20 vertices [!]
draw and an edge between any vertices with probability $p = 0.2$ We can get a random network
presented in [Jan: Figure] 3.1 below. A random network model can exhibit the small world network [!]
property. It can be shown that a random graph can exhibit small world effects. Assuming that
person A, represents a node on a network such as figure 3.1. A has z neighbours and about z^2,
z^3 second and third neighbours respectively, and so on. Then the diameter of the network D is
given by $z^D = n$. Thus

$$D = \frac{\log n}{\log z}$$

354 The logarithmic increase in the diameter of the network and the distance between node is typical
355 of a small world effect. Since $\log n$ increases slowly with n it allows the distance to be quite small
356 in very large systems (Newman, 2000). A random graph has an average degree $z = pN$. [Jan:
357 You are repeating yourself.] Random networks have a low clustering coefficient $c = \frac{z}{N}$ (Newman, [!]
2003).

Figure 3.1: Random Graph with n =20 and p $= 0.2$

358
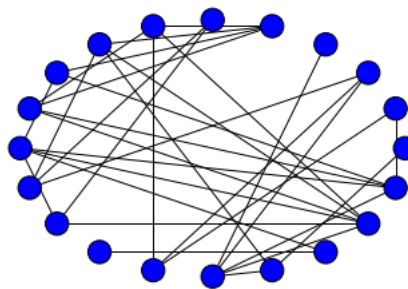
359 However, random networks are not a good model of social networks. People's circles of acquain-
360 tances tend to overlap to a great extent. Random models have a very low clustering coefficient.

361 **3.1.6 Odered Lattice.** [Jan: s/odered/ordered] In order to deal with real world networks, graphs [!]
362 must have both low clustering and small work effect properties. [Jan: Low clustering (you mean

363 high)? Small work?] Random graphs as discussed earlier show a small work effect. The average   !
364 vertex to vertex distances increase only logarithmically with n but does not show low clustering
365 (Newman, 2000). This leads us to another graph model which is an ordered lattice.

366 The opposite of a random graph is a completely ordered lattice. An ordered lattice is a graph
367 where each vertex is connected to its z neighbours. A lattice can be drawn in many dimensions.
     In the example figure 3.2 shows two lattices drawn in different dimensions.

(a) A square lattice, with $z = 4$ (b) A ring lattice, with $z = 3$

Figure 3.2: Different types of regular lattices

368

369 Regular lattices and random graphs have a long history of use in network theory and to model
370 population structures, gives an example of classic lattice (Harris, 1974).

371 The clustering coefficient for a lattice is given as

$$C = \frac{3(z - d)}{4(2 - z)} \tag{3.1.10}$$

372   [Jan: Fix typo in equation above.]   [Jan: What is C for as z goes to infinity?]   where d is the   !
373 dimension of the lattice.   !

374 However, regular lattices do not show the small world effect of vertex to vertex distances which
375 increase slowly with size. For a regular lattice of dimensions with the shape of a square or
376 hypercube of size L with $n = L^d$ vertices, the average vertex to vertex distance increases linearly
377 with the system size, which is not typical of the small world behaviour.

378 Models built on latices assume that individuals are nodes on a regular lattice and connections are
379 made of some collection of near neighbours or each node. For example, people may be spread
380 out such that connections are made to their four nearest neighbours, one on the left, right,
381 up and down or eight neighbours including the four diagonal elements are added to the (Lloyd
382 et al., 2006).To avoid the effect of the nodes at the end not being connected the last and first
383 neighbours are made neighbours.

384 The main difference between a random graph and lattices is that interactions are local, that is
385 individuals are only related to their neighbours. Where as in random networks the connections are

made are global, that is, connections are made without taking spatial locations of an individual into consideration.
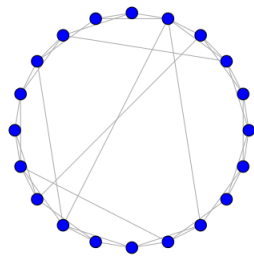
3.1.7 Watts - Strogats Small World Networks. We have shown that lattices are characterised by high clustering coefficients but long path lengths or vertex to vertex distances. That is, it takes many steps to move between any two randomly selected vertices, whereas random networks have shorter vertex to vertex distances, since there are many long range links, but low clustering (Keeling and Eames, 2005).

Small world networks were first introduced by Watts and Strogatz as an intermediate between the regular lattice and randomly rewiring certain proportions p of the network links(Watts and Strogatz, 1998). The small world networks allow for random contacts across the network. That is in addition to near neighbours as a regular lattice, each node has a random distant neighbour connected to it (Watts and Strogatz, 1998).
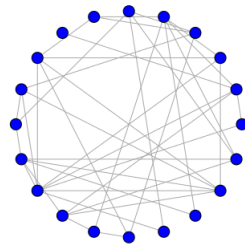
The Watts-Strogatz network is essentially a regular lattice with some degree of randomness in the connectivity of vertices. Take for example figure 3.2b above and we rewire some edges with a some probability p, that is one of its ends is moved into a randomly chosen position in the lattice. For a small p this produces mostly a regular graph, but with a few connections stretch along distances across the lattice and for $p1$ it produced a complete graph. Figure **??**elow shows Watts- Strogats networks with different probabilities.

[Jan: There are lots of typos in the graph sections. Please re-read.]   !
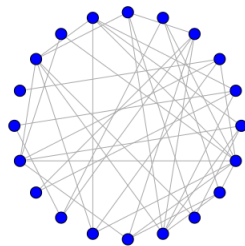
(a) A Small world, with $p = 4$ (b) A Small world network, with $p = 0.5$

(c) A Small world network, with $p = 0.8$

Figure 3.3: Watts- Strogatz

# 4. Compartmental and Stochastic Models

4.1 Deterministic Models

4.2 SIR Model

In the model the population is partitioned into three compartments. Susceptible, Infected and Recovered this is the basis for most epidemiological models. (M'Kendrick, 1925). In building the model the number of individuals Susceptible, Infected and Recovered is assumed to be differentiable over time. The simple epidemic model is given by.

$$
\begin{aligned}
\frac{dS}{dt} &= -\beta SI, \\
\frac{dI}{dt} &= \beta SI - \gamma I, \\
\frac{dR}{dt} &= \gamma I,
\end{aligned}
\tag{4.2.1}
$$

$N = S + I + R$ The model is based on the assumption that susceptible individuals become infected at a rate β proportional to the number of people infected and susceptible at time t and Infected people recover at γ rate. The reciprocal $\frac{1}{\gamma}$ is referred to as the average infectious period . Another assumption in this model is that the population remains constant, thus it does not take int account the demographic changes of the population. Figure 4.1 shows the compartmental diagram for an SIR model without demographic dynamics.

Figure 4.1: SIR compartmental diagram
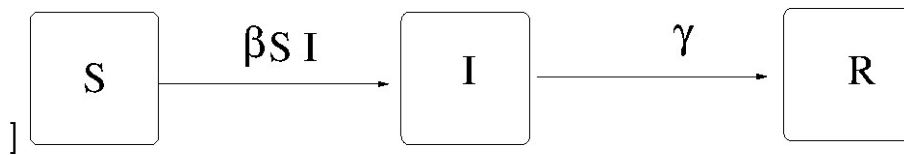
4.2.1 Model Analysis. We determine the equilibrium and the stability of 4.2.1, but since $N = S + I + R$ knowing S and I implies that we can solve for R. Hence our system of equations can be reduced to

$$
\frac{dS}{dt} = -\beta SI.
\tag{4.2.2}
$$

$$
\frac{dI}{dt} = \beta SI - \gamma I.
\tag{4.2.3}
$$

With $S(0) > 0$, $I(0) > 0$ and $R(0) = 0$ as the initial conditions for the model. We now calculate the disease free equilibrium and endemic equilibrium by equating 4.2.2 and 4.2.3 to zero then

solving them. Despite its extreme simplicity, this model 4.2.1 cannot be solved explicitly. That is, we cannot obtain an exact analytical expression for the dynamics of S and I though time, instead the model has to be solved numerically.

The equation 4.2.2 gives two import insights in understanding the spread of disease and has since been used in infectious disease modelling for along time.

4.2.2 Threshold Phenomenon. Its is important to determine whether the infection will result into an epidemic or not and what factors could determine this. Consider the initial stage after $I(0)$ individuals have been infected in a population with $S(0)$ susceptible. Equation 4.2.3 can be rewritten as,

$$\frac{dI}{dt} = I\left(\beta S - \gamma\right) \tag{4.2.4}$$

In equation 4.2.4 if the initial susceptible (S(0)) is less than $\frac{\gamma}{\beta}$. Then $\frac{dI}{dt} < 0$. This means that there will be no epidemic in this case.

This result was coined by M'Kendrick (1925) and is what is refereed to as the threshold phenomenon. The initial S(0) must exceed the threshold $\frac{\gamma}{\beta}$ for an epidemic occur. In other words the relative removal rate $\frac{\gamma}{\beta}$ must be small enough to allow the occurrence of the epidemic.

The reciprocal of the of relative removal rate is called the basic reproductive ratio and is one of the most important quantities in epidemiology . Basic reproduction ratio is defined as the average number of secondary cases arising from an average primary case in an entirely susceptible population. It measures measures the maximum reproductive potential for an infection. For the our SIR model in equation 4.2.1 it is given by:

$$R_0 = \frac{\beta}{\gamma} \tag{4.2.5}$$

For initial susceptible $S(0) = 1$, if $R_0 > 1$ then there will be an outbreak if $R_0 < 1$ the will be no outbreak. It can be noted that every disease has a different R_0 value and also depending on the population's contact pattern the R_0 value will differ.

4.2.3 Epidemic Burnout. The threshold phenomena give a description of what happens in the initial stages after introduction of an infection. Another important quantity we get from the SIR model is the long term state infection. From they system in equation 4.2.1 we take

$$\frac{dS}{dt} = -\beta SI \tag{4.2.6}$$

$$\frac{dR}{dt} = \gamma I \tag{4.2.7}$$

dividing equation 4.2.4 by equation 4.2.7 we get

$$\frac{dS}{dR} = \frac{-\beta S}{\gamma} = R_0 S \tag{4.2.8}$$

446   Integrating equation 4.2.8 with respect to R, we get;

$$\int \frac{dS}{S} = \int R_o dR \tag{4.2.9}$$

$$lnS = -R_0 R + k \tag{4.2.10}$$

$$e^{lnS} = e^{-R_0 R + k} \tag{4.2.11}$$

$$S(t) = e^{-R_0 R(t)} e^k \tag{4.2.12}$$

assuming R(0) = 0

$$S(t) = S(0) e^{-R_0 R(t)} \tag{4.2.13}$$

447   Hence as the epidemic develops the number of susceptible reduce, taking into consideration the
448   infectious period there is a lag but eventually the number of recovered start to increase. There
449   number of susceptible in the population will always be above zero as can be seen in equation
450   4.2.13.

451   From equation 4.2.13, $s(t) \geq e^{-R_o}$ since $R(t) < 1$. Thus there will always be a proportion of
452   susceptibles in the population. The epidemic burnout gives the intuitive idea that the chain of
453   transmission eventually breaks due to the decline in infectives not due to lack of susceptibles
454   (Haran, 2009).

455   **4.2.4 Disease free equilibrium.** Adding demographic parameters to 4.2.1 we get a new system
456   of equations. That is adding a parameter for birth rate and death rate, hence the population is
457   no longer closed in this case.

$$\frac{dS}{dt} = \mu - \beta SI - \mu S \tag{4.2.14}$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \tag{4.2.15}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{4.2.16}$$

458   Using the procedure we used to get equation 4.2.5 it can be shown that the R_0 for this model is

$$R_0 = \frac{\beta}{\mu + \gamma} \tag{4.2.17}$$

Now we calculate the equilibria of the model by setting equation 4.2.14, 4.2.15 and 4.2.16 to
zero, that is $\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$ and denote by $S^*, I^* and R^*$ values of I,S and R that satisfy

the condition ,that this satisfy the condition. From equation 4.2.14 we get

$$\mu - \beta SI - \mu S = 0 \tag{4.2.18}$$
$$\mu - S(\beta I + \mu) = 0 \tag{4.2.19}$$
$$S = \frac{\mu}{\beta I + \mu} \tag{4.2.20}$$

It can be seen that $S^*I^*R^* = (1, 0, 0)$ is the epidemic free equilibrium.

To establish the endemic equilibrium , we factorize I in equation 4.2.15 and we get,

$$I(\beta S - (\gamma + \mu)) = 0 \tag{4.2.21}$$

thus we get

$$I = 0 \rightarrow S = \frac{\gamma + \mu}{\beta} \tag{4.2.22}$$

Therefore, $I^* = 0$ and $S^* = \frac{\gamma + \mu}{\beta}$, but since $I^* = 0$ is a disease free equilibrium. We concentrate on $S^* = \frac{\gamma + \mu}{\beta} = \frac{1}{R_0}$ see 4.2.17

Now, we take $I \neq 0$ and solve (4.2.16). Since $S + R + I = 1$

$$\gamma I - \mu(1 - S - I) = 0 \tag{4.2.23}$$
$$\gamma I - \mu I - \mu(1 - S) = 0 \tag{4.2.24}$$
$$I = \frac{\mu}{\beta}R_0\left(1 - \frac{1}{R_0}\right) \tag{4.2.25}$$
$$I = \frac{\mu}{\beta}(R_0 - 1) \tag{4.2.26}$$

Thus the endemic equilibrium point (S^*, I^*, R^*) is $\left(\frac{1}{R_0}, \frac{\mu}{\beta}(R_0 - 1), 1 - \frac{1}{R_0}\frac{\mu}{\beta}(R_0 - 1) - \frac{1}{R_0}\right)$

4.2.5 stability of the model. Once an out break occurs its important to understand the long term behaviour of the out break and finding the stability of the model gives an insight on this. In other words calculating the stability of the model is establishing at which point the epedemic burn out will occur.

4.3   SEIR Model

The susceptible, Exposed, Infected and Recovered models add a new comportment to the previously discussed SIR Model. The earlier models assume that once a person is infected they become infectious immediately. In this model an assumption is made that one a person is exposed there is an intermediate stage between the time of infection and when they become infectious, this maybe refereed to as the latent or incubation period of the infection. The will system of equations will be;

$$S' = \mu - \beta SI - \mu S \tag{4.3.1}$$
$$E' = \beta SI - (\mu + \gamma)E \tag{4.3.2}$$
$$I' = \gamma E - (\alpha + \mu I) \tag{4.3.3}$$
$$R' = \alpha I - \mu R \tag{4.3.4}$$

where β is the rate at which susceptible individuals become infectious, γ the rate at which exposed people become infection. The quantity $\frac{1}{\gamma}$ is called the latent period of the infection. α is the recovery rate. In this model the total infected individuals is given by $E + I$ and we assume that our system is density dependant thus $S + E + I + R = 1$ and that the population is constant implying that birth rate (μ) = death rate (μ). Figure 4.2 shows the compartmental diagram of an SEIR model.

Figure 4.2: SEIR compartmental model
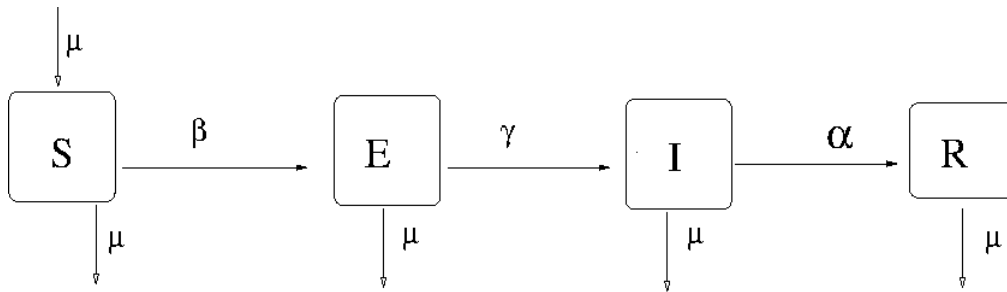
Since $R = 1 - S - E - I$ we can be drop drop equation 4.3.4 from the system and calculate equilibrium point by equating equations 4.3.1 to 4.3.3 to zero and solving the system of equations.

From equation (4.3.1) we get $S = \dfrac{\mu}{\beta I - \mu}$ and from equation 4.3.3 we get $I = \frac{\gamma E}{(\alpha + \mu)}$ and from

equation 4.3.3 we get $E = \dfrac{\beta SI}{(\mu + \gamma)}$. Thus, for I = 0, E =0 and S = 1 hence, the disease free

equilibrium of the system $S^*, E^*, I^* = (1, 0, 0)$.

When $I^* \neq 0$ we find the disease pandemic equilibrium, which is given by

$$S^*, E^*, I^* = \left(\frac{(\alpha + \mu)(\gamma + \mu)}{\beta \gamma}, \frac{\alpha + \mu}{\gamma} I^*, \frac{\mu}{\beta S*} \frac{\mu}{\beta} \right).$$

The reproductive number R_0 will be calculated using the new generation matrix method. Let F and V be non negative matrices,

$$F = \left[\frac{\partial F_i(x^*)}{\partial j} \right] \tag{4.3.5}$$

Where $F_i(x^*)$ are the rates of new infections in compartment i and

$$V = \left[\frac{\partial V_i(x*)}{\partial j} \right] \tag{4.3.6}$$

488 Where $V_i(x^*)$ are the rates of transfer of infection from one compartment to another (Van den

489 Driessche and Watmough, 2002). $F = \begin{pmatrix} 0 & 0 \\ \beta & 0 \end{pmatrix}$ and $V = \begin{pmatrix} \gamma + \mu & \gamma \\ 0 & \alpha + \mu \end{pmatrix}$

490 Therefore,

$$FV^{-1} = \begin{pmatrix} 0 & 0 \\ \beta & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{(\gamma+\mu)} & \frac{-\gamma}{(\alpha+\mu)+(\gamma+\mu)} \\ 0 & \frac{1}{\alpha+\mu} \end{pmatrix} = \begin{pmatrix} 0 & 0 \\ \frac{\beta}{(\gamma+\mu)} & \frac{-\beta\gamma}{(\alpha+\mu)+(\gamma+\mu)} \end{pmatrix} \tag{4.3.7}$$

491 The equation 4.3.7 has eigenvalue values λ_1 , λ_2 as 0 and $\frac{-\beta\gamma}{(\alpha+\mu)+(\gamma+\mu)}$ respectively.

$$R_0 = max|\lambda_1||\lambda_2| \tag{4.3.8}$$

492 Thus the R_0 for the system will be $\frac{\beta\gamma}{(\alpha+\mu)+(\gamma+\mu)}$.

493 For the disease free equilibrium (1,0,0)is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$
494 (Van den Driessche and Watmough, 2002). That is the solutions of the systems of equations
495 4.3.1 4.3.3 move towards the disease free equilibrium when $R_0 < 1$.

496 **4.3.1 Other Compartmental Models..** There are several other types of compartmental model
497 and epidemiologist tend to include additional components. Here are some of the models that can
498 be used to model infectious diseases.

499 **4.3.2 SI.** The susceptible infected model assumes that once someone is infected there is no
500 recovery. Hence it has only two components and it is used to model infectious like HIV and other
501 incurable infectious diseases. The description of the short term and long term behaviour of the
502 model can be calculated similar to the SIR model.

503 4.4   Stochastic Models

4.4.1 SIR Model. We will consider an SIR model only for the stochastic models. The total
population $N(t) = S(t) + I(t) + R(t)$ just as in deterministic models. Where

$$S(t), I(t), R(t) \in \{0, 1, 2, \ldots, N\} \tag{4.4.1}$$

504 and $t \in \{0, \triangle t, 2 \triangle t, \ldots\}$. There are two independent discrete random variables S, E and I
505 because $R(t) = N(t) - S(t) - I(t)$. Therefore the stochastic process for the SIR model is a
506 bivariate process $\{S(t), I(t)\}_{t=0}^{\inf}$ ha has a joint probability

$$p_{(s+k,i+j),(s,i)}(\triangle t) = Prob\{(\triangle S, \triangle I) = (k, j) \mid (S(t), I(t)) = (s, i)\} \tag{4.4.2}$$

where $\triangle S = S(t + \triangle t) - S(t)$. Hence the transition probability of the SIR model is a follows;

$$p_{(s+k,i+j),(s,i)}(\triangle t) = \begin{cases} \beta is/N \triangle t & (k,j) = (-1,1) \\ \gamma i \triangle t, & (k,j) = (0,-1) \\ b(N-s-i) \triangle t & (k,j) = (1,-1) \\ 1 - \beta is/N \triangle t - [\gamma i + b(N-s)] n \triangle t, & (k,j) = (0,0) \\ 0, otherwise \end{cases} \tag{4.4.3}$$

The time step $\triangle t t$ must be chosen small enough such that each transition probabilities lie in the interval [0,1]. Applying the Markov property, the difference equation satisfied by the probability $p_{(s,i)}(t + \triangle t)$ can be expressed in terms of the transition probabilities.

$$p_{(s,i)}(t + \triangle t) = p_{(s+1,i-1)}(t)(t) + \triangle t) rac\beta N (i - 1)(s + 1) \triangle t + p_{(s,i+1)}(t)\gamma(i + 1) \triangle t$$
$$+ p_{(s-1,i+1)}(t)b(i + 1) \triangle t + p_{(s-1,i)}(t)b(N - s + 1 - i) \triangle t$$
$$+ p_{(}s,i)(t) \left(1 - \left[\frac{\beta}{N} is + \gamma i + b(N - s) \right] \triangle t \right)$$

The difference equations can be written as in matrix form as

$$p_{(s,i)}(t + \triangle t) = P(\triangle t)p(t),$$

where $P(\triangle t)$ is the transition matrix and $p(t)$ is the probability distribution vector for the stochastic process. The state set is divided into two classes; the current and the transient.(N,0) is an adsorbing recurrent state while all other states are transient. The probability of an out break is given given as $1 - \frac{1}{R_0}$ when $R_0 > 1$ (Brauer, 2017).

One the main aspects in which deterministic and stochastic models differ is extinction. In deterministic SIR model an epidemic never goes to extinction in a limited time frame because the number of infectives declines exponentially and only reaches zero at infinity. In a deterministic framework an epidemic is said to go into extinction if it has a negative growth rate. On the hand in the stochastic SIR model an epidemic is the epidemic becomes extinct in a more direct sense, the number of infects can go to zero without waiting forever.

5. Spread of Zika virus on a small world network

The deterministic models discussed in the chapters above assume that all individuals have an equally small probability of being infected. In this section we build a model for the propagation of Zika virus based on a small world network.

Traditional models of infectious disease dynamics have a long, successful history of describing and modelling infectious disease spread of many diseases. They are quiet simple and tractable (Fu et al., 2013).

They [Jan: s/They/There] are certain specific and common situations when the structure of social ! connectivity is at least as important as the inactivity of the underlying infectious agents for the study of transmission of infection and control. This is one among the major reasons that has motivated the modelling of infectious diseases on social networks Fu et al. (2013).

5.1   Vector bone disease propagation on a small world network

We assume that there is a lattice with two layers. One for mosquitoes, another for human beings. A mosquito M_1 bites a Zika infected person h_1 with probability α_1. Then transmits the virus to person h_2, with a probability of α_2. Thus, person h_1 is connected to person h_2 through M_1. It can then be said that h_2 can be infected by h_1 with probability p_1, where $p_1 = \alpha_1\alpha_2$. Owing to the fact [Jan: It is not a fact, but a simplifying assumption we are making.] that α_1 and α_2 are ! independent. h_1 and h_2 are said to be near neighbours.

[Jan: Change order here. First say that we assume mosquitoes are stationary and that people have close and remote links. Only then discuss probabilities.] !

Mosquitoes do not travel long distances, but humans on the other hand travel long distances. If person h_3 travels to the place where h_1 live or gets close enough that he gets bitten by mosquito M_1 and is infected with probability α_3. Thus, α_3 is the probability that person h_3 travels and get bitten by mosquito M_1. It can be said that h_1 infects h_3 with probability p_2, because h_1 is connected to h_3 through M_1. Since α_1 and α_3 are independent, $p_2 = \alpha_1\alpha_3$. h_3 is said to be a distant neighbour to h_1. The phenomenon of infecting a distant neighbour can be expressed in 3 cases. [Jan: Don't you need 4 cases, also for local-local?] !

    i. h_4 may travel to a place near enough to get the infection from h_1 through M_1. In this case h_3 is still referred to as a distant neighbour.

    ii. h_1 may travel to a place near enough to infect h_4 through another mosquito in that vicinity.In this case h_3 is still referred to as a distant neighbour.
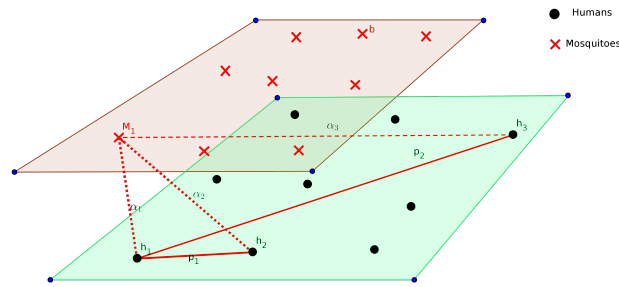
Figure 5.1: Disease transmission through a vector

iii. h_1 and h_3 may both travel to some place at the same time and h_1 transmits the infection to h_3. In this case h_3 is still referred to as a distant neighbour.   [Jan: No, we neglect this case.]   !

In all the case we assume α_3 is the same. Hence the probability of affecting a remote any distant neighbour neighbour is the same.   [Jan: Also mention that we assume a single mosquito transmits at most one infection.]   !

The existence of near and distant neighbours in the disease infection dynamics of Zika virus on a lattice with two layers in 5.1 makes it possible to represent the dynamics of disease spread on a small world network in figure 5.3.Thus, in modelling the spread of Zika virus on a small world network, the dynamics of transmission through mosquitoes are represented by the edges of the graph. An edge is drawn between two vertices, whenever there is a likelihood of transmission from one to another via mosquito bite as can be seen in figure 5.3.

5.2   Small world methodology

We can now suppose that the population is arranged in a regular   [Jan: 2-dimensional square]   !
grid. Where each vertex can infect its 4 nearest neighbours and a number of distant neighbours. Near neighbours in this case refers to individuals that one spends most of their time could be colleagues at work or school, people in the same house and distant neighbours refers to random individuals that one is likely to transmit the infection to.

 [Jan: I never liked this picture. If you had time, you could make another one that makes clear that all vertices lie on a single grid.]   !

Figure 5.2 shows the arrangement of nodes in a small world network and figure 5.3 show the transmission state diagram: S to E based on the small world network structure and the infection probabilities $p_{1,2}$: E to I with probability r_0 and I to R with probability r_1. in figure 5.2, the
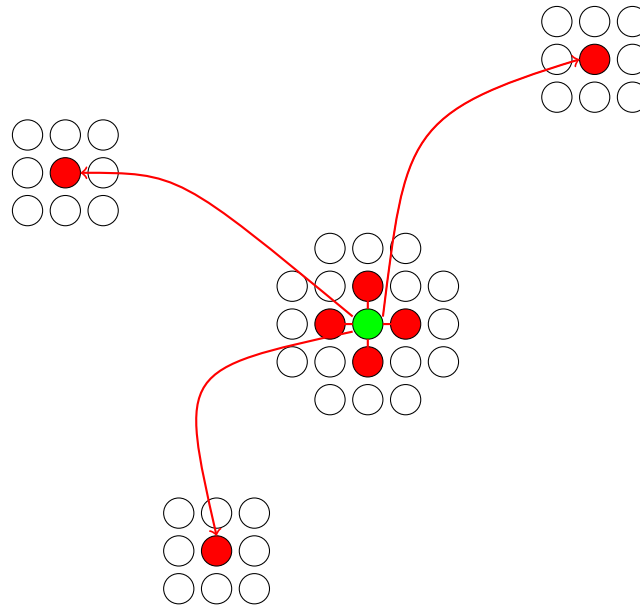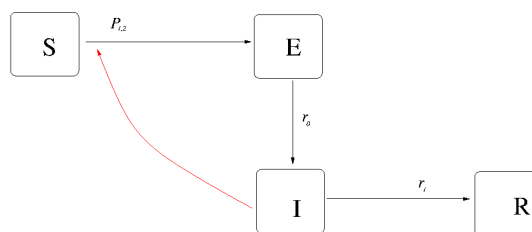
Figure 5.2: Smallworld network structure

Figure 5.3: State transition diagram

infected green node may infect its four red near neighbours with probability p_1 and its three remote neighbours with probability p_2. By infection we mean transition from Susceptible to exposed state.

Let p_1 be the probability of an infected individual causing their susceptible near neighbours to become infected and p_2 the probability of their distant or remote neighbours to become infected. Exposed people become infectious with probability r_0 and recover or get removed with probability r_1. In addition n_1 and n_2 is number of close neighbours and near neighbours respectively. [Jan: Mention that all probabilities are per day.] [Jan: Make clear that $n1 = 4$ (or less on the borders).] !

The number of distant neighbours n_2 if fixed and random for each node. [Jan: Better say: it is ! i.i.d. for each node.] That is for node I [Jan: Don't use I, it means infected. It is typical to use u or ! v.] there are $n_2^{(I)}$ distant neighbours. $n_2^{(I)}$ is chosen to follow a discrete exponentially decaying ! distribution !

$$f_c(x) = \frac{1}{c} e^{\frac{-x}{\mu}} \tag{5.2.1}$$

[Jan: Write it as $\Pr[n_2^{(v)} = k] = c \cdot \exp(-\mu x).$] with parameter μ proportional to the expected ! number of links [Jan: I don't think μ is proportional to that.] to remote nodes and $c = \frac{1}{1 - e^{frac-1\mu}}$!

ensures that f_c is a probability distribution function (Fu et al., 2013). The degree distribution in most social networks is exponentially distributed because of the celebrity effect (Estrada et al., 2015). In social networks, there are few people who have a high number of connections and many others with an average number of connections. In modelling infectious diseases these individuals are referred to as supper spreaders.

The transition probability r_0, the number of days an individual is in the exposed state is as a result of Bernoulli trials with mean $\frac{1}{r_0}$, follows a geometric distribution $f_X(x) = (1-p)^{x-1}p$. Similarly the infectious period follows a geometric distribution with mean $\frac{1}{r_o}$ (Fu et al., 2013).

5.3 Model

The model has seven parameters, they are $N, p_1, p_2, n_1, n_2, r_0$ and r_1. We let N be the population size of a city or country and is arranged in a regular grid of side length l such that $l^2 = N$. The rest of the parameters have been described above.   [Jan: I wouldn't say n_1 is a parameter, it is always 4 for us.]   !

A thorough review of literature in Lessler et al. (2016) indicates that 95% of patients [Jan: What do you mean by 95% of patients? I think you are confused here.] begin to exhibit symptoms of   !
Zika infection after 11.2 days of infection with a 95% confidence interval of $7.6 - 18$. Further the center for disease control and prevention (CDC) indicate that the incubation period for the Zika virus ranges from 3 days to 14 days from infection (Krow-Lucal et al., 2017). Therefore we estimate r_o with $\frac{1}{11.2}$ [Jan: These are the values before you exhibit symptoms. Are they the same for becoming infective?]   [Jan: Consider adding some days to allow time for a mosquito to transmit the   !
virus.]   !

95% of the cases will still have detectable virus infectiousness 18.9 days after infection with a confidence interval of 13.6 -79.4 (Lessler et al., 2016). The infectiousness in Zika infection ends 1.5 - 2 days before the virus becomes undetectable (Funk et al., 2016). Thus the chosen value of the infectious period is $18.9 - 1.5 = 17.4$ days. Therefore r_1 is estimated to be $\frac{1}{17.4}$.   [Jan: If you have time, please try different $r1$ since the confidence interval is large.]   !

Hence we have n_1, μ, p_1 and p_2 free parameters. Without control. Since the average number of secondary infections resulting from a primary Zika virus infectious is between 3 and 6, therefore we choose 4.5 as the R_0. Since the number of remote neighbours is random and fixed for each, we estimate $E(n_2^{(I)}) = \mu$.

[Jan: Please explain equations below.]   !

$$n_1 p_1 + \mu p_2 \approx \frac{R_0}{r_1} \tag{5.3.1}$$

$$n_1 p_1 + \mu p_2 \approx 0.2586 \tag{5.3.2}$$

thus, $p_1 \approx 0.0645 - 0.25\mu p_2$

We can summarize the parameters of the models as;

$$n_1 = 4 \tag{5.3.3}$$

$$\mu = 8 \tag{5.3.4}$$

$$r_0 = \frac{1}{11.2} \approx 0.089 \tag{5.3.5}$$

$$r_1 = \frac{1}{17.4} \approx 0.057 \tag{5.3.6}$$

$$p_1 = 0.0645 - 2p_2 \tag{5.3.7}$$

Now we have one free parameter. We can now estimate the number of new infections by;

$$E(- \triangle S) = (n_1 k p_1 + \mu p_2 - r_1)I \tag{5.3.8}$$

Where k is the average number of near neighbours' links that support possible infection and near neighbours are arranged in clusters, therefore $0.5k1$. In our computation, we will take $k = 0.5$. From equation 5.3.8 we can estimate the number of new infections as;

$$E(- \triangle S) = (2p_1 + 8p_2 - 0.057)I \tag{5.3.9}$$

From equation 5.3.2, it can be shown that p_1 and p_2 have natural bounds. That is taking $p_2 = 0$, implies that $p_1 \leq 0.1293$ for $k = 0.5$. For $p_1 = 0$, we get $p_2 \leq 0.3232$.

5.4   Simulation

To investigate how disease propagation varies depending on p_1, p_2, and r_1 we ran a couple of simulation on a small work network.

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