Stability Properties of Disease Models under Economic Expectations

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Abstract

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Comprehending the dynamics of infectious diseases is very important in formulating public health policies to tackling their prevalence. Mathematical epidemiology (ME) has played a very vital role in achieving the above. Nevertheless, classical mathematical epidemiological models do not explicitly model the behavioural responses of individuals in the presence of prevalence of these diseases. Economic epidemiology (EE) as a field has stepped in to fill this gap by integrating economic and mathematical concepts within one framework. This thesis investigated two issues in this area. The methods employed are the standard linear analysis of stability of dynamical systems and numerical simulation. Below are the investigations and the findings of this thesis:

Firstly, an investigation into the stability properties of the equilibria of EE models is carried out. We investigated the stability properties of modified EE systems studied by Aadland et al. [6] by introducing a parametric quadratic utility function into the model, thus making it possible to model the maximum number of contacts made by rational individuals to be determined by a parameter. This parameter in particular influences the level of utility of rational individuals. We have shown that if rational individuals have a range of possible contacts to choose from, with the maximum of the number of contacts allowable for these individuals being dependent on a parameter, the variation in this parameter tends to affect the stability properties

of the system. We also showed that under the assumption of permanent recovery for disease coupled with individuals observing or not observing their immunity, death and birth rates can affect the stability of the system. These parameters also have effect on the dynamics of the EE SIS system.

Secondly, an EE model of syphilis infectivity among "men who have sex with men" (MSM) in detention centres is developed in an attempt at looking at the effect of behavioural responses on the disease dynamics among MSM. This was done by explicitly incorporating the interplay of the biology of the disease and the behaviour of the inmates. We investigated the stability properties of the system under rational expectations where we showed that: (1) Behavioural responses to the prevalence of the disease affect the stability of the system. Therefore, public health policies have the tendency of putting the system on indeterminate paths if rational MSM have complete knowledge of the laws governing the motion of the disease states as well as a complete understanding on how others behave in the system when faced with risk-benefit trade-offs. (2) The prevalence of the disease in the long run is influenced by incentives that drive the utility of the MSM inmates. (3) The interplay between the dynamics of the biology of the disease and the behavioural responses of rational MSM tends to put the system at equilibrium quickly as compared to its counterpart (that is when the system is solely dependent on the biology of the disease) when subjected to small perturbation.

Keywords: economic and mathematical epidemiology models, syphilis, Saddle-path stability, explosive path, indeterminate-path stability, numerical solution, health gap.

Dedicated to my lovely mother, Victoria Korkoe.

You have proven to me that mother is supreme.

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Chapter 1

Introduction

1.1 Background of Study

The rise of infectious or communicable diseases is a threat to the existence of humanity. This is a challenge to public health officials. The epidemics of cholera, malaria, tuberculosis, sexually transmitted diseases and other infectious diseases in some jurisdictions is a concern. For instance, cholera claims about 100,000 to 120,000 lives each year out of an estimated number of 3-5 million cases; malaria and tuberculosis related death is estimated at 660,000 out of about 219 million and 1.4 million out of about 8.7 million respectively [48]. The World Health Organization has reported that in 2011 that about 34 million people were living with HIV/AIDS with adults constituting about 0.8% of the figure [49]. Understanding, predicting and tracking the development of these diseases is very vital if we are to make head way in formulating the right policies to effectively control the spread of these diseases. Thus, there exists the need to develop a method to tackle this.

Mathematical modelling has not only become an effective tool in understanding the dynamics of the epidemiology of diseases, it has also shed more light on the various control mechanisms that can be employed to tackle disease spread. Documentation of the above can be found in [17],[18],[22]. Even though, this approach has been helpful, it comes with its own limitations, in that, the behavioural aspect of the population is not explicitly modelled. Thus, in the presence of a disease prevalence, it is assumed that individuals behave in the same fashion. We are aware that in reality this is not applicable. Therefore, a different approach is needed. This has led to a new area in disease modelling – the Economic Epidemiology (EE) . This area incorporates economic choices in epidemic models, hence accounting for the effect(s) of behavioural responses on disease prevalence. The EE models take into account the role of externalities. That is, given a choice made by a rational individual, what bearing does it have on the total population? Will it be positive or negative? In the works of Aadland et al. [5], they pointed out that, given a disease prevalence, the choice to engage in a risky behaviour while infected imposes negative externalities on the rest of the population, in that, it forces the susceptible group in the population to choose a suboptimal number of contacts since the decision by the infected will promote the spread of the disease rapidly. Kaplan [33] investigated how the number of sexual contacts by individuals affects HIV infection rates. Philipson et al. also investigated the effects of private choices on public policy [40]. This provided good understanding to how infectious diseases spread given behavioural choices as well as their effect(s) on public health policy. An individual's ability to access quality information about disease prevalence also drives disease dynamics; it determines whether the disease prevalence will be stable or cyclical over time, since behavioural changes are dependent on the availability of information to individuals [19]. Therefore, we can not rule out the need to understand behavioural influence on disease spread.

It must be pointed out that apart from the method we employed in incorporating

behavioural responses into the models studied in this thesis, one can employ game theory. For instance, Schroeder et al. [42] made use of this method in looking at the influence of behaviour on STDs epidemics. They look at the contribution of game theory to sexual behaviour and the dynamics of the infection rate. Also they investigated as to whether or not a mere looking at the behavioural patterns of a potential sexual partner, one can tell his or her HIV status.

1.2 Method of Investigation

Various approaches can be used to study disease epidemiology; we can employ mathematical method (modelling) and statistical method (which we will not throw light on). With the mathematical method, we can develop a model for a particular infectious disease either by developing the model in discrete or continuous time interval. The thesis employs discrete dynamical systems.

1.2.1 Discrete modelling

Discrete models describe the time evolution of a system expressed by difference equations. In other words, we have a discrete system when time, $t \in \mathbb{Z}$ acts on the system. This approach of modelling sets the relationship between the future state of the system on the present state of the system at discrete intervals. An example is the study by Zhang [52], which is on the stability properties for innovation diffusion systems. There are other examples found in [21]. Discrete models can also depend on history [30], [47],. Discrete models can take stochastic and deterministic forms.

1.3 Motivation of Research

1.3.1 A modified EE model studied by Aadland et al.

Aadland et al. [6], investigated the dynamics of economic epidemiology equilibria in which they established rich properties of the EE versions of the compartmental disease models and various underlying behavioural implications on public policies were pointed out. Their analysis digs into the behavioural influences on dynamic properties of the system they considered. They further showed that a well-intentioned policy can create instability and indeterminacy when individuals behave rationally and in selfish manner. In their model, they made the following assumptions: a constant population was assumed, a utility function that is logarithmic in nature and others that will be outlined in the body of the thesis. Based on the assumptions that they made, the following questions come to mind: What happens if (1) population is not constant? That is to say birth and death rate are not the same? (2) The utility function they considered implies that there is no limit on the maximum number of contacts. If the maximum number of contacts depends on a parameter, how will this parameter affect the dynamics of the system?

This thesis attempts to look into the above questions. Hence, the thesis considered a model that has a population that is not constant and a parametric utility function that is quadratic. The choice of the the utility function is to make the maximum number of contacts dependent on a parameter which may influence the dynamics of the system.

1.3.2 Syphilis infectivity among "men who have sex with men"(MSM) inmates

STDs have become part and parcel of our human development. Almost every country is saddled with this problem. It has been estimated in 2011 that about 34 million people were living with HIV/AIDS with adults constituting about 0.8% of the figure [49]. How to address this issue is a challenge to public health policy makers. The increase in the incidence of the STDs is not limited to HIV/AIDS but to other STDs such as syphilis, chlamydia, genital warts and others. For instance, records have it that the prevalence of syphilis was reduced drastically after the introduction of penicillin, but the disease has resurfaced in recent years [5]. Recent surveys show that an estimated 10 million new infections occur every year [50].

These phenomena have struck detention centres (prisons), thus posing very challenging problems to the authorities in charge of these facilities. For instance, statistics show that the HIV prevalence among prisoners is more than fifty times higher than that of the general adult population [39]. In the USA for instance, it is believed that the ratio is as 6 to 1; in France, 10 to 1; and in Mauritius, 50 to 1 [46]. As the world's population is growing rapidly, so is the prison population . For instance, the incarceration rate for USA is about 756 per 100,000 of the national population [44]. In Sub-Sahara Africa, about 668,000 men and women are imprisoned with South Africa recording the highest – 157,402 prisoners with 335 per 100,000 imprisoned from the national population [39].

With high levels of incarceration rates, it is undoubted that the detention centres are becoming overcrowded. It is evident that some of these detention centres are in poor shape to accommodate this number of prisoners. This poses a serious threat to the health of these inmates, as authorities responsible in managing these facilities

will find it difficult to put in place effective control mechanisms to control the spread of communicable diseases among them.

The spread of STDs in detention centres is not limited to HIV/AIDS but also to other STDs such as syphilis, gonorrhoea etc.. In fact there are researches that confirm the high level of syphilis prevalence among detainees in prisons. Vaz et al. pointed out that syphilis and HIV are common among the inmates in prisons in Maputo [45]. Work by other researchers in the field also confirms this [8],[27],[51].

Most prisons are dominated by men. Some of these detainees become involved in sexual activities [39]. These activities could take the form of prisoner to prisoner or prison-guard to prisoner [39]. It is also pointed out in [39] that about 10 to 60 percent of prisoners in Zomba prison in Malawi have had at least one anal sex encounter. Sexual activities among theses individuals could be by consent or by force. In 2001, Human Rights Watch reported that those inmates who are involved in this act appeared to be in agreement with the supposed partners [2]. In some jurisdictions, MSM is regarded as illegal. This may be one of the contributing factors to the rapid spread of these diseases since protection is not given to this section of people in the population. Prisoners in Africa are sometimes seen as outcasts and are left without protection. The prison guards sometimes take advantage of this situation and sexually abuse these inmates with impunity : Sexual abuse is one of the means by which inmates are disciplined [39]. This situation is not only applicable to prisons in Africa but to other parts of the world. For instance, there are incidence of rape in USA prisons [46]. Thus, anal intercourse either by rape or consent has remained one of the key transmission media of STDs and for that matter syphilis among male inmates.

There have been models on the spread of HIV among inmates in prisons [23],[39].

Since at times there is transfer or movement of prisoners to and from within the system of prisons; there has been work on the epidemiology of diseases within this framework. For instance, Ching et al. looked into this issue [20]. Nevertheless, none of these works have looked into the stability properties of syphilis among inmates and its possible relation to public health policy by combining mathematical and economic concepts in formulating an Economic Epidemiology (EE) model where there is the interplay of the biology of the disease and behavioural choices. This thesis attempts to address this issue. To the best of our knowledge, this is the first attempt made to understand this phenomenon among inmates.

1.4 Thesis Outline

This thesis studied the economic version of mathematical compartmental epidemiology models. Two studies were carried out: a modified model of the earlier works by Aadland et al. [6] as well as a model on syphilis infectivity among MSM inmates. We apply the standard linearization method to investigate the dynamic paths of the system around the endemic steady state under economic expectations. In order to compare the results from the former model to that of Aadland et al., we used the same parameter values they used in their studies and then vary them to check whether our model will yield a different result. Below is the outline of the thesis:

Chapter 2 discusses the background of the concepts we applied in our study. It gives some insight into epidemiology, mathematical epidemic and economic epidemiological models, and dynamical models, where discussion is held on the stability conditions for both deterministic and stochastic discrete models. Also the notion of utility functions and dynamic programming was visited.

Chapter 3 is on the development of the modified model on Aadland et al. works and its analysis. We first provided the underlying framework for the model studied by Aadland et al. and then modified it to the one of interest. We made use of a quadratic utility function. Our choice for this utility function is motivated by the fact that it provides the necessary limit on the number of contacts rational individuals can make unlike the utility function considered by Aadland et al. which is a logarithmic utility function. It also adds a new dimension to the behaviour of the system. In that, the maximum number of contacts made by individuals is determined by a parameter. We talked about the economic versions of the compartmental disease models where we considered the following cases: When individuals observed their immunity and when they did not observe their immunity. We carried out stability analysis of our model. We derived a matrix system of equations by linearizing the system around the endemic steady states and then carried out simulations. We assumed that individuals make choices based on rational expectations by imposing a perfect foresight on the system. Due to the complex nature of the system, there were no analytical solution provided. Thus, we carried out numerical simulation and based our discussion on the results. We showed that (1) if individuals behave in a self centred manner but have their maximum number contacts dependent on a parameter, this parameter influences the dynamics of the system. (2) Public policy has the tendency to drive the dynamics of the system.

Chapter 4 is on a model on syphilis infectivity among MSM inmates. We developed a compartmental disease model. Economic concepts are introduced into the analysis of the system where MSM inmates are assumed to behave in a self centred manner without taking into consideration the welfare of the other inmates. We employed the same approach outlined in chapter 3 in the analysis of the system. We investigated the dynamic properties of the model under rational expectations. We have shown that if rational individual inmates behave in a self-centred manner, their

behavioural responses to disease prevalence affect the dynamic properties of the system. This result holds for the case under rational expectations where individuals are assumed to have perfect knowledge of the system and are aware of the risk and benefits faced by the other inmates as they make a choice. On the other hand, under naive expectations, public policy has no effect on the stability properties of the system. It is also shown that the prevalence of the disease in the long run is influenced by incentives that drive the utility of the MSM inmates. Furthermore, we compared the dynamics of the EE and the ME systems. We found out that the EE system gravitates towards the equilibrium more quickly than its counterpart.

Chapter 5 summarizes our results as well as explains the contribution of the thesis to this area of study. Future work is also outlined.

Chapter 2

Literature Review

This chapter talks about several basic concepts related to this thesis which includes (just to mention a few): epidemic models, stability analysis of dynamical systems, utility and objective functions and Bellman equations, etc.. Specific examples are given to buttress the discussion on these concepts.

2.1 Epidemiology

Epidemiology has played a vital role in health policy formulation, as it has helped provide an informed public decision in disease outbreak. Through the development of methodologies, epidemiology as a field has helped in identifying the manner in which infectious diseases are contracted [4].

In literal terms, epidemiology is coined from the Greek words *epi*, meaning "on or upon," *demos*, meaning "the common people," and *logy*, meaning "study." By putting these words together one can define epidemiology as "the study of that which

falls upon the common people" [4]. The earth bears records of infectious diseases that suddenly infect a particular population and suddenly disappear and then resurface again. Some (infectious diseases) also reside within a particular population. An example includes, the spread of smallpox across many parts of Europe that resulted in the death of about 10% of young children [22]. There are biblical accounts of plaques suffered by a particular population. An example is the plagues of Egypt [1]. One can argue that early marriages coupled with technological advancements that helped to improve the supply and distribution of food contributed to the sharp increase in the population in the eighteenth century (China's population increased from 150 million to 313 million between 1760 and 1794 and in Europe; in increased from 118 million to 187 million between 1700 and 1800 [18]). However, one can not rule out the fact that the reduction in communicable diseases contributed to a lower death rate. As well, the improvement in medicine and the development of a strong immunity against these diseases also contributed to the trend in rising population rates.

An epidemic, which acts on a short duration, can be described as a sudden outbreak of disease that infects a great proportion of the population in an area (or region) before disappearing. Epidemic attacks are of a recurrent nature with intervals of several years between outbreaks.

Thus, epidemiology can be regarded as a chain of reasoning concerned with biological inferences stemming from the observations of disease(s) occurrence and related phenomena in human population groups [36]. Therefore, one can view epidemiology as an integrated field deriving and borrowing its concepts and methods from other disciplines such as mathematics, statistics, sociology and others for the study of disease(s) in a population. For instance, mathematical methods were introduced to this effect in the twentieth century. The works by McKendrick et al. relates to that

[34], where a disease model was developed to look at the behaviour of outbreaks of infectious diseases.

Epidemiologists are primarily concerned about the occurrence of disease as grouped by time, place, and persons. They endeavour to determine whether or not there has been a reduction or an increment in the prevalence of a disease over the years, whether one geographical area has a higher prevalence of the disease than the other, as well as whether the characteristics of person(s) with a particular disease or condition differentiate them from those without it [36]. The next section talks about the mathematical methods employed in epidemiology.

2.2 Mathematical Epidemic Models

An epidemic model is a simplified way of explaining the transmission of communicable disease(s) such as Sexually Transmitted Diseases (STD s) through individuals. Being able to know or to predict the behaviour of a disease could aid scientists in evaluating inoculation (the process of artificial induction of immunity against infectious disease) or isolation plans. This could have a tremendous effect on the death rate associated with a particular epidemic [14]. The modelling of infectious disease can be seen as a tool which has been used to study the mechanisms by which disease spread, to predict the future course of an outbreak and to evaluate strategies to control an epidemic [22]. The first record of mathematical modelling of infectious disease was carried out by Daniel Bernoulli in 1766 [22]. He defended the practice of inoculating against smallpox by using a mathematical model to evaluate the efficacy of the method of variolation [31]. During those times, smallpox spread across many parts of Europe where a large part of the population was affected. The disease related death was about 10% among young children [22].

In order to put in place better public policies to curb the spread of these diseases, there is the need to know the intensity of the epidemic of the disease in question. In order to achieve the above, questions such as the following need to be answered: how many people will suffer from the disease outbreak? What is the peak number of people to be infected? and others. For instance, in [34], the behaviour of the outbreak of infectious disease was investigated and a prediction was made which is in line with the above questions.

In disease modelling, there are cases that affect the nature and the underlying assumptions of the model. For instance, if one is dealing with a large population, a deterministic mathematical model is made use of. An example is the studying of the epidemics of whooping cough (pertussis) which is highly contagious. Also, the manner in which disease(s) spread is random. This is due to the unpredictable nature of the interaction(s) between individuals. Therefore, there is the need for this random process to be accounted for in the model. For instance, if a new infectious disease hits a population, at the early stage of the outbreak (because there may be a small number of cases recorded), one cannot predict the very dynamic nature of the disease. It may or may not affect a large portion of the total population before dying out. To account for this, one needs to specify the model "stochastically". Thus, in mathematical epidemiology modelling, we have two types of models to consider: deterministic and stochastic mathematical epidemiology models. The next sections will delve briefly into these types of models.

2.2.1 Deterministic mathematical epidemiology models

In deterministic models, the formulation is done by first dividing the population under study into mutually exclusive subgroups(compartments) such that the

transition rate from moving from one compartment to another is specified. This transition rate is done via differential or difference equations. Because of the groupings of the population into various subgroups, the deterministic models are also known as compartmental models. Each of the subgroups describes a disease category. The assumption is that the compartmental size of each disease category changes with respect to time. There is a different compartmental structure for studying the epidemiology of every disease. For instance, the diseases that no immunity can be built against have a structure different from the one in which an immunity can be built against and also from those that are transmitted by vectors [17]. In [11] for example, models that involve vector transmissions are considered. These are diseases that are transmitted from human to human indirectly as malaria which is spread by mosquitoes. In this case, the model must include both humans and mosquitoes which mostly requires creation of a lot of compartments. Below is an example of a simple epidemic model .

Example 1

This particular example is a model that describes the transmission of communicable diseases. It is a special case of those studied by Kermack and Mckendrick (1927, 1932, 1933) [18]. The model describes three mutually exclusive disease compartments in a population: the susceptible compartment (S), where individuals in the population are susceptible to the disease, the infective compartment (I), where those infected with the disease in question belong and the recovered compartment (R), where those who have recovered from the disease are grouped. Terminologically, we call this model the SIR model. We have other cases which we can also term the $SIRS$, SIS, and the SI. These models are considered in [17], [18]. Also, there are many extensions of the SIR. Examples are SEIR and SEIRS. The E is the category for individuals exposed to the disease but not yet infectious themselves.

Suppose the probability that an infected person coming in contact with a susceptible person who can transmit the infection at period t is $\frac{S_t}{N_t}$. Let the contacts enough to make transmission of infection by the average member of the population with others be cN_t per unit time. Then we have the number of new infections in unit time to be $(cN_t)(\frac{S_t}{N_t})I_t = cS_tI_t$, where c and N are the contact rate and population size respectively. Let the rate at which the infected leaves the infective class be b and finally, we suppose that the population is constant with no entry or departure except where there is disease related death. What this means is that, the duration of the disease is so short that the effect of birth and death rate on the demographics of the population can be ignored. Based on this assumption we have the following difference equations explaining the model:

$$\begin{cases} S_{t+1} = S_t - cS_tI_t, \\ I_{t+1} = I_t + cS_tI_t - bI_t, \\ R_{t+1} = R_t + bI_t. \end{cases} \tag{2.1}$$

To make biological sense, at $t = 0$, $S_0, I_0, R_0 > 0$. An attempt will not be made at analyzing the model. The main idea is to give an example.

2.2.2 Stochastic mathematical epidemiological models

As we hinted earlier, looking at the random nature of disease transmission, it is imperative to include the stochastic behaviour in our model. Stochastic modelling involves the incorporation of the variation of chance into the disease transmission process such that a provision is made for the possible ranges of outcomes which are based on probability. When one is considering a small or isolated population in disease modelling and the chance of fluctuations is needed to be accounted for, then

this type of modelling is very useful [43]. Stochastic models preserve unique properties from the deterministic models: probability of disease extinction, probability of disease outbreak, quasi stationary probability distribution, final size distribution and expected duration of an endemic. These properties are well documented in [18]. For the purpose of my thesis we will not throw more light on stochastic models.

2.3 Economic Epidemiology (EE)

The field of economic epidemiology incorporates economic choices in epidemiology models. The main theme of this field is to study the interaction of how the health state of an individual and the way he or she responds to a disease's outbreak in the epidemiological context. The notion that the way individuals behave in the presence of an infectious disease is dependent on the disease prevalence gave rise to this field. For instance, in the emergence of the AIDS epidemic, there has been a lot of economic research in order to understand how the behavioural pattern affects the spread of the disease [40]. EE models also take into account other factors such as externalities, global diseases such as AIDS, syphilis, malaria and cholera, the effects of individuals' incentives on the epidemiology of disease(s) and the cost of curbing it. Disease treatment and prevention depend heavily on the volatility in the behaviour of individuals in the presence of disease outbreak(s). Their decisions also have tremendous bearing on the entire population. For instance, Kaplan showed that the number of sexual contacts by individuals affects HIV infectivity [33].

Behavioural responses have the potential of affecting the prevalence of an infectious disease. An example is shown in the works by Blower et al., where they demonstrated that the reduction in the risk of contracting HIV as a result of the introduction of vaccine could lead to an increase in the incidence of the disease [16].

Timing of public policies in the presence of a disease outbreak is very crucial. For instance, in the works of Geoffard et al. [28], they found out that, due to competition between public policies and behaviours on the part of individuals in a population hit by an infectious disease, it becomes futile for a public policy directed at providing subsidies if the the prevalence of the disease makes individuals to take protective measures. Thus, the policy has no effect. Behavioural response does not only sometimes render public policy futile, but also may have bearing on the vaccine policy formulation. In [10], the authors pointed out that the manner in which individuals respond to the introduction of vaccine to control the HIV epidemic will rather lead to an increase in the incidence of the disease, in that, the introduction of the vaccine will promote risky behaviour on the part of individuals. Since decision making is based on individual choices, vaccination policy may contribute less than maximum social outcome that the policy sought to achieve. The reason is that, individuals are mostly concerned about their immediate benefit not the benefit of the society as a whole. Availability of information on the prevalence level of a disease either in the present or in the future affects the behavioural pattern of individuals, in that, if individuals perceive that the prevalence level of a disease is very high, the likelihood that they will reduce their contact rate is high. If the converse happens, they involve in risky behaviour which may even lead to a high prevalence level in the long-run. Thus, a myopic behaviour on the part of individuals may trigger a disease outbreak.

The above and others are some of the issues EE seeks to address. What we ought to know is that "economic and biological epidemiology can make different predictions about diseases occurrence mainly due to their different predictions about the relationship between prevention and prevalence"[40].

2.4 The Notion of Utility Functions

This section is devoted to a brief discussion on utility theory and expected utility. Specific examples are given for illustration purposes.

2.4.1 Utility theory

Firms, households and governments make decisions and choices. There are rewards associated with these decisions or choices. These decision makers make choices or decisions such that the possible optimal reward is ascertained. These rewards are what is termed utility or satisfaction. Thus, utility theory gives a framework for evaluating the associated utility with a particular choice. The underlying assumption is that, every choice or decision made is based on the principle of utility maximization. Therefore, decision makers should be concerned with making the best possible choice(s) that will yield the optimal utility. Utility theory is applicable in many fields in economics, such as finance and behavioural economics.

Utility is measured by a function called the utility function. This function measures the associated reward with a specific choice or decision made by a decision maker. For instance, the reward can be measured in monetary terms, services or quantity of goods. In disease modelling, utility can be measured in terms of the number of contacts by decision makers (in this case individuals).

Utility functions

A continuous function that satisfies the following conditions is a utility function:

$$U'(x) > 0 \text{ and } U''(x) < 0,$$

where x is a variable the decision maker is interested in. For instance, it could be a monetary amount, or the number of contacts etc.. The mathematical interpretation of the above condition is that the function U is a concave function on x. For economic interpretation, the utility of a decision maker increases with respect to x. But as his utility increases, it will get to a point where an additional demand for x will yield a decrease in the additional level of his utility. $U'(x) > 0$ also means the representative agents will always prefer more to less. As an example, let us consider an individual who is very thirsty. His utility will increase as he consumes more cups of water but will get to a point where an additional consumption of a cup of water will yield a decrease in his utility. The additional utility derived as a result of consuming an additional unit of x is what is termed as marginal utility. Utility functions can be expressed in logarithmic, quadratic, exponential, power form etc., depending on the objective of study.

2.4.2 Expected utility

We are always faced with uncertainties when it comes to choice making. The way a decision is valued in a particular instance is different from another instance. Therefore, if one places a value on a particular decision in a particular state as compared to another possible state, then there must be a probability that the state of interest will actually occur. Therefore, in economic theory, in the presence of uncer-

tainty, the decision process should be based on expected utility [32]. For instance, in the presence of a disease epidemic, say HIV, a representative agent may decide to have an incident of unprotected sex with a random partner; since this agent is not certain about his/her decision (whether he will be infected with HIV or not after the act), the utility should be expressed as an expected utility, taken into account the average of utility he/she may get when remaining uninfected and when infected.

Constructing expected utility

For the purpose of my work, we will discuss two states.

Let us consider two mutually exclusive states, such that the probability of being in state A and B is denoted by p_1 and p_2 respectively. Let the associated utility in A and B be $u_1(x)$ and $u_2(x)$ respectively. Then our expected utility $U(x)$ will be

$$U(x) = p_1 u_1(x) + (1 - p_1)u_2(x),$$

where $p_2 = 1 - p_1$. It must be noted here that, the probabilities could be dependent on other variables in the model under study. What the above relation means is that, the expected utility will be a function of the utility in each state, and the probabilities. Therefore, in our example in 2.4.2, if we let $u_1(x)$ and $u_2(x)$ denote the associated utility for the individual being infected and not being infected after making x number of contacts respectively, then we expect the individual's expected utility to be as the above equation with it respective probabilities.

2.5 Dynamic Programming and Bellman's Equation

This section briefly discusses some concepts on dynamic programming and Bellman equation as related to this thesis. Before we touch on the Bellman's equation, let us first talk about the following analytical concepts as pertaining to dynamic programming.

2.5.1 The underlying concepts

Firms, households or governments are faced with many problems in which decisions have to be made to address them. For instance, households will have to decide how much they spend on their livelihood and how much they must save for their future. Another instance could be an individual who in the prevalence of HIV may decide to have unprotected sex or not. Given a resource constraint, a government may decide to make education more affordable by giving free or subsidized eduction to its citizens or making education more accessible by investing in educational infrastructure close to the citizens. All these choices involve decision processes that involve cost-benefit trade off. These decisions can be broken down in such a way that each is tackled over time. The process of doing this is what is referred to as dynamic programming or optimization. This presupposes that time plays a crucial role.

In solving an optimization problem, there are objectives that we seek to meet. The goal of a firm, for instance, is how to combine resources (for example, capital and labour) to achieve the possible maximum production level so as to maximize profit. In general, minimizing cost and maximizing utility (satisfaction), are the objectives

of any representative agent(s). These objectives can be described mathematically. The function describing these objectives is what is termed the objective function.

In dynamic optimization, one needs to know the information for the period in question before a right decision can be arrived at. In other words, the state of the system must be known. To illustrate this, let us consider a representative agent, say a firm, to give dividends to its shareholders, must have information, for instance, about its profit margin at the current period (of course, other factors also play a crucial role in determining dividend). The profit margin is therefore one of the state variables and the amount of dividends to issue out is the control variable. The control variables are therefore those variables chosen at any point in time. Thus, the rule that helps determine the control variables is the policy function. In our case, if we assumed that the amount of dividends to be issued is only determined by the profit margin of the firm, then the function $D(p)$ is the policy function which the firm seeks to find. D is the amount of dividend and p is the profit margin.

By definition, optimal policy is "a policy which maximizes a preassigned function of the final state variables" [13].

2.5.2 Derivation of the Bellman's equation

Let us suppose a representative agent makes a decision at discrete points in time such that time $t \in \Gamma$, where

$$\begin{cases} \Gamma = \{0, 1, ..., T\}, \\ T \leq \infty. \end{cases}$$

The above expression means that, the decision process could have an infinite or finite horizon. Suppose an initial state s_0 is given. The representative agent chooses an action a_0 which is dependent upon the current state of the system (that is $a_0 \in \eta(s_0)$). $\eta(s_0)$ is the possible set of feasible actions available to the agent. In general, an action taken in time t can be related to the state by $a_t \in \eta(s_t)$. Therefore, an action a_t taken at state s_t yields a one-period reward to the agent. Let $U_t(a_t, s_t)$ denote the reward. Note that, if the agent's decision process assumes a finite horizon, then it is assumed that the agent will not take any action at the last period. Therefore, the reward he or she may get at the terminal period will be dependent on only the state in that period.

Now, from the above, given the initial state s_0 with the corresponding action a_0 taken by the agent, state s_1 will be dependent upon s_0 and a_0. The agent will then take an action a_1 dependent on s_1. Therefore, action a_t will be dependent on s_t which is also dependent on previous actions and states. These actions must satisfy some optimality criteria. Actions chosen at any period may be randomly or deterministically chosen.

Let us now define a function called the value function, $v(s_o)$ which is defined as the optimal value of the objective function defined as the discounted value of the sum of all future values given an initial state at time $t = 0$. Let us also assume the decision process has an infinite horizon. Therefore, we have the following:

$$v(s_0) = \underset{\{a_t\}_{t=0}^{\infty}}{max} \sum_{t=0}^{\infty} \beta^t U(s_t, a_t), \tag{2.2}$$

subject to

$$\begin{cases} a_t \in \eta(s_t), \\ s_{t+1} = \top(s_t, a_t), \text{ for } t \geq 0. \end{cases}$$

$\beta \in (0,1)$ is the discount factor that satisfies the assumption that representative agents place less value on future values of the action taken today. This means that at $t = 1$, the optimal value of the objective function subjected to the constraint will be given as $v(s_1)$ and $\beta v(s_1)$ is the value $v(s_1)$ discounted back to period $t = 0$.

The above problem can be solved by breaking the problem into sub ones such that we have the following :

$$v(s_0) = \underset{a_0}{max}\left[U(s_0, a_0) + \underset{\{a_t\}_{t=1}^{\infty}}{max} \sum_{t=1}^{\infty} \beta^t U(s_t, a_t)\right], \tag{2.3}$$

subject to

$$\begin{cases} a_t \in \eta(s_t), \\ s_{t+1} = \top(s_t, a_t) \text{ for } t = 1, 2...T. \end{cases}$$

Also, Eq. (2.3) is subjected to the constraint

$$\begin{cases} a_0 \in \eta(s_0), \\ s_1 = \top(s_0, a_0). \end{cases}$$

The decomposition of (2.2) into (2.3) is governed by the principle of optimality which states that "An optimal policy has the property that whatever the initial state and initial decision are, the remaining decisions must constitute an optimal policy with regard to the state resulting from the first decision" [13]. Thus, equation (2.3) can be further written into the form:

$$v(s_0) = max\{U(s_0, a_0) + \beta\nu(s_1)\}, \tag{2.4}$$

subject to

$$\begin{cases} a_0 \in \eta(s_0), \\ s_1 = \top(a_0, s_0). \end{cases}$$

The above equation is what is termed Bellman's equation. In general we have

$$v(s_t) = max\{U(s_t, a_t) + \beta v(s_{t+1})\}, \tag{2.5}$$

subject to

$$\begin{cases} a_t \in \eta(s_t), \\ s_{t+1} = \top(a_t, s_t). \end{cases}$$

Deriving the first order condition

We demonstrate how to derive the first order condition with this specific problem in economics. Suppose a representative agent has the following problem to solve:

$$\underset{\{a_t\}}{max} \sum_{t=0}^{\infty} \beta^t U(a_t), \tag{2.6}$$

subject to

$$a_t + s_{t+1} = f(s_t).$$

Where $U(a_t)$ denotes a utility function, a and s are variables to be determined. The state variable is s_t . The control variable is a_t. Therefore, we have the following as the Bellman's equation:

$$v(s_t) = \underset{\{a_t\}}{max}\{U(a_t) + \beta v(s_{t+1})\}, \tag{2.7}$$

subject to

$$a_t + s_{t+1} = f(s_t).$$

Assume the representative agent lives in two periods; period t and $t+1$. To get the optimal behaviour of the agent, we make use of the Lagrangian principle. Therefore, let λ_t be the associated multiplier. Thus, we have

$$v(s_t) = \underset{\{a_t\}}{max}\{U(a_t) + \beta v(s_{t+1})\} + \lambda_t(f(s_t) - s_{t+1} - a_t)\}. \tag{2.8}$$

For the first order conditions involving a_t and s_{t+1} associated with the above equation, we have:

$$\begin{cases} U'(a_t) = \lambda_t, \\ \beta v'(s_{t+1}) = \lambda_t. \end{cases} \tag{2.9}$$

Where

$$\begin{cases} U'(a_t) = \frac{dU}{da_t}, \\ v'(s_{t+1}) = \frac{dv}{ds_{t+1}}. \end{cases} \tag{2.10}$$

Thus, we have

$$U'(a_t) = \beta v'(s_{t+1}). \tag{2.11}$$

Also, from Eq. (2.8), we have

$$v'(s_t) = \lambda_t f'(s_t).$$

From $U'(a_t) = \lambda_t$, we therefore have

$$v'(s_t) = U'(a_t) f'(s_t). \tag{2.12}$$

Thus, moving Eq. (2.12) one period forward and making use of Eq. (2.11), we have the following equation:

$$U'(a_t) = \beta[U'(a_{t+1})f'(s_{t+1})].$$

The above equation is the first order necessary condition. This equation coupled with the constraint depicts the optimal behaviour of the agent. This method can be extended to problems involving stochastic processes.

2.6 Dynamical Systems

A dynamical system can be seen as any system that evolves over time. It is a system that is specified by points in geometrical space, say $X \in \mathbb{R}^n$ and a rule or a function , say $F : \mathbb{R}^n \to \mathbb{R}^n$ that conveys the behaviour of a system as it evolves over time. Therefore, one can say that, a dynamical system is basically made up of two parts: the state vector which gives the exact description of the state of the system in question and a rule that describes the future state of the system given its initial state. To some extent, knowing the state vector of any system explaining a phenomenon (be it a physical, biological, economical etc. system) is enough to determine the future state of that system in question. Thus, one can say that "the state vector is the numerical description of the current configuration of a system" [41].

Theories on dynamical systems are employed in the analysis of phenomena in several fields outside of mathematics: one can talk of disease modelling, dynamic optimization as pertaining to mathematical economics and others. An interval for data acquisition takes primarily two forms: discrete and continuous. For instance, a report on the price of gold every two weeks only is an example of data acquisition at discrete intervals. On the other hand, a ball thrown in the air has no time interval.

The time the ball is in motion is continuous. Thus, dynamical systems are categorized into continuous and discrete systems. Due to the scope of this thesis, focus would be made only on discrete dynamical systems.

2.6.1 Discrete dynamical systems

A discrete dynamical system describes the time evolution of a system expressed by difference equations. In other words, we have a discrete system when time, $t \in \mathbb{Z}$ acts on the system . A discrete dynamical system can be written in the following form:

$$X_{t+1} = AX_t + B,$$

where X is an $(n \times 1)$ vector of variables, and $X(0) = X_0$, A is an $n \times n$ matrix of parameters which can be constant or vary across time, and B is an n-dimension column vector of parameters which can be constant or vary with respect to time.

In [21] there are models studied in which difference equations are applied to practical problems: we have the model for beetle population, disease epidemiology and others. Discrete dynamical systems can be studied in two forms: the stochastic and the deterministic form. Below is the stability analysis of difference equations of each respective type.

2.6.2 Deterministic difference (discrete) equations

This section is devoted to discussing the stability conditions of the above type of systems. We will only be dwelling on First-order Linear systems.

Stability analysis

Suppose a general system takes the following form:

$$AX_{t+1} = BX_t + CZ_t, \qquad X_{t=0} = X_0. \tag{2.13}$$

Where X is an $(n \times 1)$ vector of endogenous variables and Z_t is an $(k \times 1)$ vector of exogenous variables. A, B and C are conformable matrices. Let us suppose the following:

Assumption 1 *There exists α such that $det(A\alpha - B) \neq 0$.*

Assumption 2 *There exists $T > 0$ such that $Z_t = Z^*$ for all $t \geq T$.*

Assumption 3 *$\{Z_t\}$ is a stable sequence.*

Let us also consider the following definitions:

Definition 1 *A steady state point, X^* of a sequence $\{X_t\}$ is a point such that $X_t = X^*$, then $X_s = X^*$ for all $s > t$.*

Definition 2 *A sequence $\{X_t\}$ is stable if there exists $M > 0$ such that $\|X_t\|_{max} < M$ for all t, where $\|X_t\|_{max} = max|X_j|$ for all $X \in \mathbb{R}^n$.*

Definition 3 *A point X^* is said to be stable asymptotically if the sequence $\{X_t\}$ is such that $lim_{t\to\infty} X_t = X^*$ for some initial value of X (that is $X_{t=0} = X_0$).*

Definition 4 *A point X^* is said to be stable globally if the sequence $\{X_t\}$ is such that*

$$lim_{t\to\infty} X_t = X^* \tag{2.14}$$

for all initial values of X.

The next section is for discussion on the stability conditions of Eq.(2.13).

Let us employ the method proposed by Blanchard and Khan to analyse the above system. [15]. Suppose A is a non-singular matrix. Then Eq. (2.13) can be rewritten in the form

$$X_{t+1} = A^{-1}BX_t + A^{-1}CZ_t. \tag{2.15}$$

Let us suppose assumption 1 is satisfied. Also let $W = A^{-1}B$ such that by transforming W into Jordan canonical form , we have $W = P^{-1}JP$, where J is the Jordan matrix having its diagonal element being the eigenvalues λ of W. P is an invertible matrix which consists of the eigenvectors of W. J can be decomposed into the following:

$$J = \begin{bmatrix} J_1 & & & \\ & J_2 & & \\ & & ... & \\ & & & J_k \end{bmatrix},$$

where

$$J_i = \begin{bmatrix} \lambda_i & 1 & & \\ & \lambda_i & 1 & \\ & & ... & 1 \\ & & & \lambda_i \end{bmatrix}_{n_i \times n_i},$$

such that for each λ, $\det(W - \lambda I) = 0$ is satisfied. Also for all $i = 1, ...k$, λ_i has n_i repeated values.

Definition 5 *If $|\lambda| < 1$ then we say λ is stable. If $|\lambda| > 1$ then we say λ is unstable. We have a stable matrix if all the eigenvalues of that matrix have a modulus less than one. In other words, we have a stable (unstable) matrix if all its eigenvalues are within (outside) the unit circle.*

Theorem 1 *Let A and $I-W$ be non-singular matrices. Suppose assumption 2 holds and $(A-B)^{-1}$ exists then by definition 1, X^* can be expressed as*

$$X^* = (A-B)^{-1}CZ^*,$$

such that for all X_0, there exists a solution $\{X_t\}$ such that

$$lim_{t\to\infty}X_t = X^*$$

if and only if all λ of W are stable.

See [38] for the proof.

If the above theorem holds, then the steady state is referred to as a sink or indeterminate. If all λ of W is unstable, then the steady state is referred to as a source (explosion).

Theorem 1 was ascertained by deriving a backward-looking solution for Eq. (2.13) by assuming that all the initial values X_0 are given. At times not all the components of the initial value X_0 are given. In this case, the above theorem is not applicable. The reason is that, we can not solve Eq. (2.13) using the backward-looking solution in this case. Thus, we employed a different approach. This takes us to our next subtopic.

The notion of predetermined and non-predetermined variables

For deterministic systems, predetermined variables are variables whose initial values are exogenously given whilst for non-predetermined (jump) variables, their initial values are not exogenously given.

Now, let us rewrite Eq. (2.15) in the following form :

$$\begin{bmatrix} X_{t+1} \\ Y_{t+1} \end{bmatrix} = W \begin{bmatrix} X_t \\ Y_t \end{bmatrix} + \gamma Z_t, \tag{2.16}$$

where

$$\begin{cases} W = A^{-1}B, \\ \gamma = A^{-1}C. \end{cases}$$

X is an $(n \times 1)$ vector of predetermined variables, Y is an $(m \times 1)$ vector of non-predetermined variables. Let us partition the Jordan matrix as follows:

$$J = \begin{bmatrix} J_1 & \\ & J_2 \end{bmatrix}, \tag{2.17}$$

where J_1 is an $n_1 \times n_1$ matrix having all its eigenvalues as stable and J_2 is an $n_2 \times n_2$ matrix having all its eigenvalues as unstable. Let us consider the following theorem. See [15] for the proof.

Theorem 2 *If the number of unstable eigenvalues is equal to the number of non-predetermined variables, then given X_0, there exists a unique stable solution for all stable sequence $\{Z_t\}$ satisfying Eq. (2.16).*[1]

It follows that if theorem 2 and assumption 2 are satisfied, then it is sufficient to say the solution to Eq. (2.16) satisfies

$$\lim_{t\to\infty} X_t = X^*,$$

where X^* is defined in theorem 1. In this case, the steady state is referred to as saddle [38].

[1]The proof of the above is well detailed in [38] by Miao

There is no solution to Eq.(2.16) if the number of unstable eigenvalues is more than the number of non-predetermined variables. If the number of unstable eigenvalues is less than the number of non-predetermined variables, then there are infinitely many solutions to Eq.(2.16).

Example 3

Let us consider the neoclassical growth model with constant relative risk aversion preferences. Suppose a single agent who lives within an infinite time period chooses a control variable c (level of consumption) at a time period t, such that his/her choice is to maximize an objective function of the form:

$$\underset{\{c_t\}}{max} \sum_{t=\infty}^{\infty} \beta^t \Big(\frac{c_t^{1-\sigma}}{1-\sigma} \Big), \tag{2.18}$$

subject to

$$c_t + k_t = k_{t-1}^{\alpha} + (1-\delta)k_{t-1}. \tag{2.19}$$

Where $\beta, \delta \in (0,1)$ denotes a discount factor and depreciation rate of capital respectively. σ is the coefficient of relative risk aversion. k denotes capital. Let us further assume that utility is time-separable (that is to mean marginal utility of consumption today depends only on the level of consumption today). We seek for the stability of the equilibrium of the agent's consumption and capital as time passes by. The first order condition of the objective function is as follows:

$$\begin{cases} c_t^{-\sigma} = \beta c_{t+1}^{-\sigma}(\alpha k_{t+1}^{\alpha-1} + (1-\delta)), \\ k_{t+1} = k_t^{\alpha} - c_t + (1-\delta)k_t. \end{cases} \tag{2.20}$$

Below is the time-invariant version of the above equations in c and k, which represent the steady state value for consumption level of the representative agent and capital

respectively which ought to be solved for.

$$\begin{cases} c^{-\sigma} = \beta c^{-\sigma}(\alpha k^{\alpha-1} + (1-\delta)), \\ k = k^{\alpha} - c + (1-\delta)k, \end{cases}$$

By log-linearizing[2] Eq. (2.20) about the steady state, we have the following:

$$\begin{cases} -\sigma \hat{c}_t = \sigma \hat{c}_{t+1} + \beta(\alpha-1)F\hat{k}_{t+1}, \\ \hat{k}_{t+1} = \frac{1}{\beta}\hat{k}_t - \frac{c}{k}\hat{c}_t. \end{cases}$$

where $\frac{c}{k}$ and $F = \alpha k^{(\alpha-1)}$ are the ratio of consumption to capital and marginal product of capital respectively. Hat, $\wedge$ on top of the variables indicates the deviation of the variables from the steady state value of the variables. We therefore have the following as the linearized matrix system from the above equation:

$$\begin{bmatrix} -\sigma & \beta(\alpha-1) \\ 0 & 1 \end{bmatrix} \begin{bmatrix} \hat{c}_{t+1} \\ \hat{k}_{t+1} \end{bmatrix} = \begin{bmatrix} -\sigma & 0 \\ -\frac{c}{k} & \frac{1}{\beta} \end{bmatrix} \begin{bmatrix} \hat{c}_t \\ \hat{k}_t \end{bmatrix}.$$

So that we have

$$\begin{bmatrix} \hat{c}_{t+1} \\ \hat{k}_{t+1} \end{bmatrix} = M \begin{bmatrix} \hat{c}_t \\ \hat{k}_t \end{bmatrix}, \tag{2.21}$$

where

$$M = \begin{bmatrix} 1 - \frac{c\beta(\alpha-1)F}{k\sigma} & \frac{(\alpha-1)F}{\sigma} \\ -\frac{c}{k} & \frac{1}{\beta} \end{bmatrix}.$$

We have one predetermined variable $\{k\}$ and one non-predetermined variable $\{c\}$. The interpretation is that, if the eigenvalues of M is such a way that, one is

[2]The concept of log-linearization is well explained in [38].

stable and the other one is unstable then we have a saddle path stability. If all the two eigenvalues are not stable then we have a source and if all the eigenvalues are stable, then we have an indeterminate path stability.

Stochastic discrete models: Rational expectation

There are several definitions of rational expectation. We have the broad, mid and weak definition. The weak definition is what is mostly accepted. Therefore, the discussion on this section will be based on the definition below.

Definition 6 *"Agents formulate expectations in such a way that their subjective probability distribution of economic variables (conditional on the available information) coincides with the objective probability distribution of the same variable(the state of nature) in an equilibrium."[3]*

The above definition can be expressed mathematically as follows:

$$Y_{t+1}^{e} = E(Y_{t+1}|\Omega_t), \tag{2.22}$$

where $E(.)$ is the mathematical expectation operator, Ω_t is the information set available to the agents at time t and Y_{t+1}^{e} is the equilibrium at time, $t+1$. In this thesis we will use the following notation:

$$E_t(Y_{t+1}) = E(Y_{t+1}|\Omega_t).$$

From the above definition, it is imperative we consider the following proposition:

Proposition 1 *Suppose* $\hat{Y}_t = Y_t - Y_t^e$*, which is the expectation error. The following holds:*

$$E_t(\hat{Y}_{t+1}) = 0.$$

The above proposition means, individuals do not make systemic error when forming rational expectations.

Proposition 2 *Expectation errors do not exhibit any serial correlation.*

Proposition 3 *Let us consider the following information sets:* Ω_t *and* Ω_{t-1} *such that* $\Omega_{t-1} \supset \Omega_t$*, then the following holds:*

$$E_t Y_{t+1} = E_t(E_{t-1} Y_{t+1}).$$

The following section discusses the solution to linear difference (discrete) models under rational expectations.

The general model

Let us recall Eq. (2.16):

$$\begin{bmatrix} X_{t+1} \\ E_t Y_{t+1} \end{bmatrix} = W \begin{bmatrix} X_t \\ Y_t \end{bmatrix} + \gamma Z_t, \qquad X_{t=0} = X_0,$$

where X is an $(n \times 1)$ vector of predetermined variables, Y is an $(m \times 1)$ vector of non-predetermined variables. Here, because we are dealing with the stochastic case of the model, predetermined and non-predetermined is defined differently. According to Blanchard and Kahn [15], a stochastic variable is predetermined if $E_t X_{t+1} = X_{t+1}$ whatever the realization of the variables (actual observed value) in the information

set at time $t+1$. Also the initial value, X_0 is given exogenously. On the other hand, a stochastic variable is said to be non-predetermined if $E_t Y_{t+1} = Y_{t+1}$ such that only the realizations of all variables in Ω_{t+1} are the same as their expectations conditional on Ω_t [15]. Note that Ω_t is made up of current and past values of X, Y and Z. To rule out the possibility of the exogenous variable Z from exploding too fast, let us impose the following restriction on its value(s)[3]:

For all t there exist $\bar{Z}_t \in \mathbb{R}^k, \theta_t \in \mathbb{R}$ such that

$$-(1+i)^{\theta_t}\bar{Z}_t \leq E(Z_{t+1}|\Omega_t) \leq (1+i)^{\theta_t}\bar{Z}_t \tag{2.23}$$

for all $i \geq 0$ [15]. As in the deterministic case, the same conditions for the stability of the system are applicable to the stochastic case.

Example 4 .

Let us consider the following log-linearized systems of equation:

$$\begin{cases} \pi_t = kx_t + \beta E_t \pi_{t+1}, \\ x_t = E_t x_{t+1} - \frac{1}{\sigma}(i_t - E_t \pi_{t+1}) + e_t, \\ i_t = \rho_r i_{t-1} + \epsilon_t, \end{cases} \tag{2.24}$$

where $k > 0, \beta > 0,$ and $\sigma > 0$ are constants and $|\rho_r| < 1$ and ϵ_t are random numbers that are independent and identically distributed (i.i.d). e_t is an exogenous i.i.d shocks.

[3]For a detailed explanation on the above concept, visit [15].

Writing the above system of equations in matrix form yields the following:

$$\begin{bmatrix} 1 & 0 & 0 \\ -\sigma^{-1} & 1 & \sigma^{-1} \\ 0 & 0 & \beta \end{bmatrix} \begin{bmatrix} i_t \\ E_t x_{t+1} \\ E_t \pi_{t+1} \end{bmatrix} = \begin{bmatrix} \rho_r & 0 & 0 \\ 0 & 1 & 0 \\ 0 & -k & 1 \end{bmatrix} \begin{bmatrix} i_{t-1} \\ x_t \\ \pi_t \end{bmatrix} + \begin{bmatrix} \epsilon_t \\ -e_t \\ 0 \end{bmatrix}.$$

The above matrix can be rewritten into the form

$$\begin{bmatrix} i_t \\ E_t x_{t+1} \\ E_t \pi_{t+1} \end{bmatrix} = W \begin{bmatrix} i_{t-1} \\ x_t \\ \pi_t \end{bmatrix} + M \begin{bmatrix} \epsilon_t \\ -e_t \\ 0 \end{bmatrix},$$

where

$$W = \begin{bmatrix} \rho_r & 0 & 0 \\ \frac{1}{\sigma} & 1 + \frac{k}{\beta\sigma} & -\frac{1}{\beta\sigma} \\ 0 & -\frac{k}{\beta} & \frac{1}{\beta} \end{bmatrix} \tag{2.25}$$

and

$$M = \begin{bmatrix} 1 & 0 & 0 \\ -\sigma^{-1} & 1 & \sigma^{-1} \\ 0 & 0 & \beta \end{bmatrix}^{-1}. \tag{2.26}$$

If the number of unstable eigenvalues for W is equal to the number of non-predetermined variables (in this case are x and π) the above system will have a unique stable solution. On the other hand, there will be an indeterminate or infinite solution if the number of unstable eigenvalues is less than the number of non-predetermined variables and an explosion when we have unstable eigenvalues more than the number of non-predetermined variables. Now, in our case, when we find the expression for the eigenvalues (not included), we will have only one unstable eigenvalue, since we assumed that $|\rho_r| < 1$. Thus, the system's equilibrium path is indeterminate.

Chapter 3

The Stability Properties of SIR(S) and SI(S) Economic Epidemiological Models

This chapter discusses the model investigated by Aadland et al [6]. Description of the modified model is presented and its stability properties investigated by employing numerical analysis.

3.1 The Model considered by Aadland et al.

3.1.1 Epidemiology

The epidemiology part of the model considered three mutually exclusive disease categories: Susceptible(S), infected(I) and recovered with immunity(R). The mech-

anism involved in transitioning from one disease category to the other is as follows: an individual infected by a disease will migrate from the susceptible category to the infected category and then when treated and immune against the disease, will migrate from the infected category to the recovered category and then back to the susceptible category when he or she becomes prone to the disease again. This explains the classical SIRS model. If there is permanent recovery such that the individual will not be prone to disease, then we have the classical SIR model [11].

Let p_t be the probability that susceptible individuals become infected after coming into contact with infected individual(s) so that $(1 - p_t)$ is the probability of susceptible individuals remaining susceptible after coming into contact with infected individual(s). Suppose that individuals in the recovered category have recovery rate as ν, then there will be recovery of νI individuals from the infected category and $(1 - \nu)I$ individuals remaining in the infected category. Also, let γ be the rate at which individuals in the recovered category become susceptible so that $\frac{1}{\gamma}$ is the average duration of immunity (the assumption is that the immunity period is exponentially distributed) and $(1 - \gamma)$ is the rate at which individuals remain in the recovered category. In this case, the number of individuals entering the susceptible category is γR and those remaining in the recovered category is $(1-\gamma)R$. Finally, let us assume that the population is constant such that the birth and death rate are the same and are given as μ and μ respectively. The epidemiological model is described by Fig. 3.1.

Therefore, we have the following discrete equations explaining the model:

$$\begin{cases} S_{t+1} = \mu N_t + (1 - p_t - \mu)S_t + \gamma R_t, \\ I_{t+1} = (1 - \nu - \mu)I_t + p_t S_t, \\ R_{t+1} = (1 - \gamma - \mu)R_t + \nu I_t. \end{cases} \tag{3.1}$$

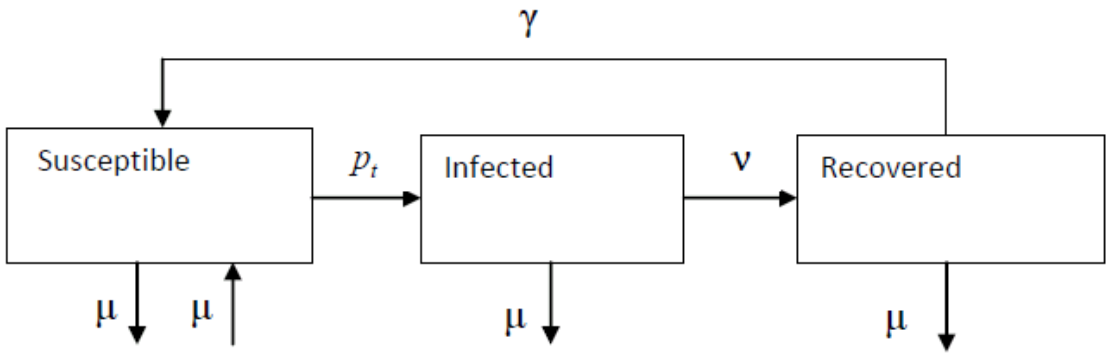


Figure 3.1: The flow chart for the SIRS model.

Let $N_{t+1} = S_{t+1} + I_{t+1} + R_{t+1}$. Then we have $N_{t+1} = N_t$. Writing Eq. (3.1) as a proportion of N_{t+1}, we have the following:

$$\begin{cases} s_{t+1} = \mu + (1 - p_t - \mu)s_t + \gamma r_t, \\ i_{t+1} = (1 - \nu - \mu)i_t + p_t s_t, \\ r_{t+1} = (1 - \mu - \gamma)r_t + \nu i_t, \end{cases} \tag{3.2}$$

where

$$\begin{cases} s_{t+1} = \frac{S_{t+1}}{N_{t+1}}, \\ s_t = \frac{S_t}{N_t}, \\ i_{t+1} = \frac{I_{t+1}}{N_{t+1}}, \\ i_t = \frac{I_t}{N_t}, \\ r_{t+1} = \frac{R_{t+1}}{N_{t+1}}, \\ r_t = \frac{R_t}{N_t}. \end{cases}$$

Suppose individuals independently choose x_t contacts and that the probability of an uninfected individual becoming infected follows Bernoulli process. Let λ_p be the chance of becoming infected with each contact. Then the probability of a susceptible

individual becoming infected is

$$p_t = 1 - (1 - \lambda_p i_t)^{x_t} \,, \tag{3.3}$$

where

$$\lambda_p = 1 - (1 - \lambda_a)^a$$

denotes the probability of contracting the disease from a single infected contact. λ_a denotes the probability of an individual contracting the disease from a single interaction with an infected contact.

3.2 Introducing Economics into the Model

Let the representative agent (individual) n utility at time t be $u(x_{n,t}, h_{n,t})$, where $h_{n,t}$ is a parameter that captures the agent's health status at time t. This parameter plays a very important role in the individual's choice of a number of contacts, in that, if the individual is infected with the infectious disease in question, the individual experiences a low value for h. Because the additional contacts made by an individual bring immediate satisfaction or a risk of getting infected by the disease in question, any additional contact(s) the individual makes either affect(s) the level of utility positively or negatively. For instance, a contact made by an individual that resulted in contracting the disease will cause a deterioration in the individual's health, thus reducing the value of the parameter h at the given period, hence affecting the utility of the individual inversely. Utility is assumed to be concave. Aadland et al. [6] considered a logarithmic utility function. Thus, if we suppose that the representative agent n maximizes expected lifetime utility by choosing the number of contacts, $x_{n,t}$,

then we have the following as the objective function of the agent :

$$E_t \sum_{j=0}^{\infty} \beta^j [\ln(x_{n,t+j}) + h_{n,t+j}], \tag{3.4}$$

where $0 < \beta < 1$ is the discount factor, E_t is the expectation operator at t.

The above is the description of the model studied by Aadland et al. [6]. The next section is on the discussion of my modification to their model.

3.3 The Model of Interest: The SIR(S) Model

This model considers a population that has a birth and death rate not necessarily equal. It also assumes a quadratic utility function.

The epidemiological model

As in the case of Aadland et al. [6], we followed the same chain of reasoning. Below are the equations explaining the model:

$$\begin{cases} S_{t+1} = \omega N_t + (1 - p_t - \mu) S_t + \gamma R_t, \\ I_{t+1} = (1 - \nu - \mu) I_t + p_t S_t, \\ R_{t+1} = (1 - \gamma - \mu) R_t + \nu I_t, \end{cases} \tag{3.5}$$

where ω and μ are birth and death rate respectively. Again, let $N_{t+1} = S_{t+1} + I_{t+1} + R_{t+1}$, so that we have

$$N_{t+1} = (1 + \omega - \mu) N_t. \tag{3.6}$$

If $\omega = \mu$ in the above equation, then the model reduces to the one studied by Aadland et al. [6]. That is, we have a constant population. Writing model (3.5) as a proportion of N_{t+1}, gives us

$$\begin{cases} s_{t+1} = A\omega + A(1 - p_t - \mu)s_t + A\gamma r_t, \\ i_{t+1} = A(1 - \nu - \mu)i_t + Ap_t s_t, \\ r_{t+1} = A(1 - \mu - \gamma)r_t + A\nu i_t, \end{cases} \tag{3.7}$$

where

$$\begin{cases} s_{t+1} = \frac{S_{t+1}}{N_{t+1}}, \\ s_t = \frac{S_t}{N_t}, \\ i_{t+1} = \frac{I_{t+1}}{N_{t+1}}, \\ i_t = \frac{I_t}{N_t}, \\ r_{t+1} = \frac{R_{t+1}}{N_{t+1}}, \\ r_t = \frac{R_t}{N_t} \\ A = \frac{1}{1+\omega-\mu}. \end{cases}$$

Let α be the chance of becoming infected with each contact. Then the probability of a susceptible individual becoming infected is

$$p_t = \Pr(\text{infection}) = 1 - (1 - \alpha i_t)^{x_t} . \tag{3.8}$$

The dependence of the probability of infection on the chosen number of contacts differentiates the analysis from the standard (classical) mathematical epidemiology (ME) [5]. For instance, if individuals under study do not take into account the health consequences of their risky behaviour, thus going for the maximum number of contacts $\bar{x}$ in any given period, then we have the EE model collapsing to the standard ME

with infection probability being

$$p_t = 1 - (1 - \alpha i_t)^{\bar{x}} \, . \tag{3.9}$$

Because individuals in this case do not value their health, the ME model sets $h = 0$, hence depicting a constant infection parameter. Even if the EE model has $h = 0$, the infection parameter varies with i_t [5].

3.3.1 Introducing economics into the model

With regards to the objective function, we considered a utility function that is quadratic in nature so that there will be a restriction on the possible number of contacts individuals can make. In our work we used

$$u(x_{n,t}, h_{n,t}) = x_{n,t} - \delta x_{n,t}^2 + h_{n,t}, \tag{3.10}$$

where $0 < \delta < 1$ is a fixed parameter. The introduction of δ into the utility function plays very crucial role, in that, the availability of drugs and vaccination will cause δ to assume a small value. The converse holds when there is unavailability of vaccination or treatments. Therefore, public policy direction can influence the dynamics of the system via δ. As you will notice later, the maximum number of contacts is dependent on this parameter: The low values this parameter takes, the higher the possible number of maximum contacts. This implies that, as δ decreases, the utility of individuals increases. If one thinks his choice of contacts have no bearing on his probability of transitioning to another health state, then he has a static optimization

problem such that his optimality condition becomes

$$\frac{\partial u}{\partial x_{n,t}} = 0. \tag{3.11}$$

In such a case, the utility maximizing number of contacts is $x_{n,t} = \frac{1}{2\delta}$. This is the optimal choice of number of contacts for an individual in either the infected or recovered category [24]. Clearly, you will notice that $x_{n,t}$ is dependent on δ. Thus, a decrease in δ means an increase in $x_{n,t}$ and an increase in δ means a decrease in $x_{n,t}$.

Suppose the representative agent n maximizes expected lifetime utility by choosing the number of contacts, $x_{n,t}$. We have the following as the objective function of the agent:

$$E_t \sum_{j=0}^{\infty} \beta^j [(x_{n,t+j} - \delta x_{n,t+j}^2) + h_{n,t+j}], \tag{3.12}$$

where $0 < \beta < 1$ is the discount factor, E_t is the individual's expectation operator at time t

Considering our categories S, I and R, at any given time t, an individual can only be in S, I or R. Averaging over all n, we have the proportions of susceptible, infected and recovered individuals in the entire population. Also, we assumed that all individuals are identical with the exception of having a different disease state and health level. We analysed the model in terms of a single individual in each of the disease categories. Therefore, let us drop the n subscript.

An individual belonging to the susceptible group makes a choice about contacts on the basis of his single-period utility function and expected future utility which depends on infection expectations. This susceptible individual's decision will satisfy

the Bellman's equation

$$V_t^s = \underset{x \in X}{max}\{x_t - \delta x_t^2 + h^s + \beta E_t[p_t V_{t+1}^i + (1 - p_t)V_{t+1}^s]\} \,. \tag{3.13}$$

V_t^s in the equation above is the value function associated with being susceptible at time,t. The term in the bracket is the expected future utility which depends on expected future infection levels. The present value of the expected future utility is the V_{t+1}^s if the individual remains susceptible and V_{t+1}^i is the present value of expected future utility if the individual becomes infected after making a choice in period t [24]. X is the range of possible contacts. In our case we have $X = [0, \frac{1}{2\delta}]$. From the same reasoning we have the value function for the infected and recovered groups as follows:

$$V_t^i = \bar{x} - \delta \bar{x}^2 + h^i + \beta E_t[\nu V_{t+1}^r + (1 - \nu)V_{t+1}^i], \tag{3.14}$$

$$V_t^r = \bar{x} - \delta \bar{x}^2 + h^s + \beta E_t[\gamma V_{t+1}^s + (1 - \gamma)V_{t+1}^r] \,, \tag{3.15}$$

where $h^s > h^i$. $h^s$and h^i are the health status associated with an individual in the susceptible (or recovered) and infected groups respectively.

All individuals regardless of infection status maximize Eq.(3.4) without the concern for the general population. Infected and recovered individuals with immunity therefore choose the maximum number of contacts, $\bar{x}$ because they do not stand any risk of immediate infection [6]. The implication of this is that, an infected individual who is involved in the maximum possible amount of risky behaviour will spread the disease in the population, thus causing the susceptible group to make the number of contacts that is suboptimal [5]. The converse holds if one is dealing with an altruistic population (In [5], the study of syphilis cycles was studied based on this assumption). Suppose an individual in the susceptible group chooses a number of contacts, such

that this number of contacts satisfies the first order necessary condition

$$(2\delta x_t - 1) = -\beta p_{x,t} E_t[V^s_{t+1} - V^i_{t+1}], \tag{3.16}$$

where

$$p_{x,t} = \frac{\partial p_t}{\partial x_t} = -\frac{(1-p_t)}{x_t}\ln(1-p_t).$$

The term on the right hand of Eq.(3.16) depicts the expected marginal damage costs of increasing current contacts in terms of the discounted expected reduction in future utility due to infection. On the other hand, the left hand term represents the current period benefit as the individual increases contacts. What this means is that, an individual who is in the susceptible group chooses x_t such that his or her marginal benefits and expected marginal cost are equal. Also, the contact level decision influences the probability of becoming infected. Furthermore, Eq.(3.16) shows that the contact rate in EE model is based on behavioural responses to changes in disease risk as opposed to the mathematical (classical) epidemiology models where the contact rate is considered as being constant or can be varied deterministically. This is exhibited by the expression connecting $p_{x,t}$. Eq.(3.16) is also known as Euler's equation, and henceforth will be referred to as such. In the analysis of the model, we considered two cases (following the works by Aadland et al. [6]) depending on the agent's observance of their own immunity.

Unobservable Host Immunity

Suppose an individual who has recovered with immunity from the disease in question believes he is still susceptible to the disease. Then we can ignore Eq. (3.15)

and obtain from Eq. (3.14) that

$$V_t^i = x_t - \delta x_t^2 + h^i + \beta E_t[\nu V_{t+1}^s + (1-\nu)V_{t+1}^i]. \tag{3.17}$$

From Eq. (3.13) and (3.17) we have

$$\begin{aligned} V_t^s - V_t^i &= (x_t - \delta x_t^2 + h^s) - [(\bar{x} - \delta \bar{x}^2) + h^i] + \beta E_t[p_t V_{t+1}^i \\ &\quad + (1-p_t)V_{t+1}^s - \nu V_{t+1}^s - (1-\nu)V_{t+1}^i] \\ &= [(x_t - \delta x_t^2) - (\bar{x} - \delta \bar{x}^2)] + h^s - h^i + \beta E_t[-p_t(V_{t+1}^s + \\ &\quad - V_{t+1}^i) + V_{t+1}^s - V_{t+1}^i - \nu(V_{t+1}^s - V_{t+1}^i)] \\ &= \psi(x_t, \bar{x}) + h + \beta E_t[(1-p_t)(V_{t+1}^s - V_{t+1}^i) - \nu(V_{t+1}^s - V_{t+1}^i)]. \end{aligned}$$

This implies

$$V_t^s - V_t^i = \psi(x_t, \bar{x}) + h + \beta(1 - \nu - p_t)E_t[V_{t+1}^s - V_{t+1}^i]. \tag{3.18}$$

Therefore, from Eq. (3.16), Eq. (3.18) becomes

$$V_t^s - V_t^i = \psi(x_t, \bar{x}) + h - \frac{(1-\nu-p_t)}{p_{x,t}}[2\delta x_t - 1]. \tag{3.19}$$

Let us move Eq. (3.19) one step ahead and then take E_t on both sides and multiply through by β so that we have

$$\beta E_t[V_{t+1}^s - V_{t+1}^i] = \beta E_t\left[\psi(x_{t+1}, \bar{x}) + h - \frac{(1-\nu-p_{t+1})}{p_{x,t+1}}[2\delta x_{t+1} - 1]\right]. \tag{3.20}$$

Substituting out V^s_{t+1} and V^i_{t+1} from the Eq. (3.20) by making use of Eq. (3.16), we have

$$(2\delta x_t - 1) = p_{x,t}\beta E_t\Bigg[-[\psi(x_{t+1},\bar{x}) + h] + \frac{(1-\nu-p_{t+1})}{p_{x,t+1}}[2\delta x_{t+1} - 1]\Bigg], \tag{3.21}$$

where

$$\begin{cases} \psi(x_t,\bar{x}) = (x_t - \delta x_t^2) - (\bar{x} - \delta\bar{x}^2), \\ \psi(x_{t+1},\bar{x}) = (x_{t+1} - \delta x_{t+1}^2) - (\bar{x} - \delta\bar{x}^2), \\ h = h^s - h^i. \end{cases}$$

Observable Host Immunity

In this case, let us suppose individuals who recovered with immunity from the disease in question, observe their own immunity and thus rationally choose the maximum number of contacts $\bar{x}$ and have health level h^s. In this case Eq. (3.13), (3.14) and (3.15) become relevant. Therefore, from Eq. (3.14) and (3.15) we have

$$\begin{aligned} V^r_t - V^i_t &= h^s - h^i + \beta E_t[\gamma V^s_{t+1} + (1-\gamma)V^r_{t+1} - \nu V^r_{t+1} - (1-\nu)V^i_{t+1}] \\ &= h + \beta E_t[\gamma V^s_{t+1} - \gamma V^r_{t+1} - \nu V^r_{t+1} + \nu V^i_{t+1} + V^r_{t+1} - V^i_{t+1}] \\ &= h + \beta E_t[\gamma V^s_{t+1} - \gamma V^r_{t+1} + \gamma V^i_{t+1} - \gamma V^i_{t+1} - \nu V^r_{t+1} + \nu V^i_{t+1} + V^r_{t+1} - V^i_{t+1}] \\ &= h + \beta E_t[\gamma(V^s_{t+1} - V^i_{t+1}) - \gamma(V^r_{t+1} - V^i_{t+1}) + (1-\nu)(V^r_{t+1} - V^i_{t+1})] \\ &= h + \beta E_t[\gamma(V^s_{t+1} - V^i_{t+1}) + (1-\nu-\gamma)(V^r_{t+1} - V^i_{t+1})] \\ &= h + \beta E_t[\gamma(V^s_{t+1} - V^i_{t+1})] + (1-\nu-\gamma)\beta E_t[(V^r_{t+1} - V^i_{t+1})]. \end{aligned}$$

From Eq. (3.16) we have

$$V^r_t - V^i_t = h - \gamma\frac{(2\delta x_t - 1)}{p_{x,t}} + (1-\nu-\gamma)\beta E_t(V^r_{t+1} - V^i_{t+1}). \tag{3.22}$$

Moving Eq. (3.22) one period ahead and then taking E_{t-1} on both sides gives

$$E_{t-1}[V^r_{t+1}-V^i_{t+1}] = h-\gamma E_{t-1}\left[\frac{(2\delta x_{t+1}-1)}{p_{x,t+1}}\right]+(1-\nu-\gamma)\beta E_{t-1}[(V^r_{t+2}-V^i_{t+2})]. \quad (3.23)$$

From Eq. (3.13), (3.14) and (3.16) we have

$$\begin{aligned} V^s_t - V^i_t &= \psi(x_t,\bar{x}) + h + \beta E_t[p_t V^i_{t+1} + (1-p_t)V^s_{t+1} - \nu V^r_{t+1} - (1-\nu)V^i_{t+1}] \\ &= \psi(x_t,\bar{x}) + h + \beta E_t[(1-p_t)(V^s_{t+1} - V^i_{t+1}) - \nu(V^r_{t+1} - V^i_{t+1})] \\ &= \psi(x_t,\bar{x}) + h + \beta(1-p_t)E_t[(V^s_{t+1} - V^i_{t+1})] - \nu\beta E_t[(V^r_{t+1} - V^i_{t+1})]. \end{aligned} \quad (3.24)$$

Therefore,

$$V^s_t - V^i_t = \psi(x_t,\bar{x}) + h - (1-p_t)\frac{(2\delta x_t - 1)}{p_{x,t}} - \nu\beta E_t[(V^r_{t+1} - V^i_{t+1})]. \quad (3.25)$$

Eq. (3.16) can be re-written as

$$E_{t-1}[V^s_t - V^i_t] = -\frac{(2\delta x_{t-1} - 1)}{\beta p_{x,t-1}}. \quad (3.26)$$

Therefore, taking E_{t-1} on both sides of Eq. (3.25) and making use of Eq. (3.26) and rearranging, we have

$$\begin{aligned} \beta\nu E_{t-1}[V^r_{t+1} - V^i_{t+1}] &= E_{t-1}\left[\psi(x_t,\bar{x}) + h - (1-p_t)\frac{(2\delta x_t - 1)}{p_{x,t}} - (V^s_t - V^i_t)\right] \\ &= E_{t-1}\left[\psi(x_t,\bar{x}) + h - (1-p_t)\frac{(2\delta x_t - 1)}{p_{x,t}}\right] - E_{t-1}[V^s_t - V^i_t]. \end{aligned} \quad (3.27)$$

This implies that

$$E_{t-1}[V^r_{t+1} - V^i_{t+1}] = \frac{1}{\beta\nu}E_{t-1}\left[\psi(x_t, \bar{x}) + h - (1-p_t)\frac{(2\delta x_t - 1)}{p_{x,t}}\right] + \frac{1}{\beta^2\nu}\left[\frac{(2\delta x_{t-1} - 1)}{p_{x,t-1}}\right]. \tag{3.28}$$

Imposing perfect foresight (that is $E_t\hat{x}_{t+1} = \hat{x}_{t+1}$) and moving one period ahead and then taking E_{t-1} on both sides of Eq. (3.28) gives

$$E_{t-1}[V^r_{t+2} - V^i_{t+2}] = \frac{1}{\beta\nu}E_{t-1}\left[\psi(x_{t+1}, \bar{x}) + h - (1-p_{t+1})\frac{(2\delta x_{t+1} - 1)}{p_{x,t+1}}\right] + \frac{1}{\beta^2\nu}\left[\frac{(2\delta x_t - 1)}{p_{x,t}}\right]. \tag{3.29}$$

Thus, from Eq. (3.22), (3.28) and (3.29) we have

$$\begin{aligned}
&\frac{1}{\beta\nu}E_{t-1}\left[\psi(x_t, \bar{x}) + h - (1-p_t)\frac{(2\delta x_t - 1)}{p_{x,t}}\right] - \frac{1}{\beta^2\nu}\left[\frac{(2\delta x_{t-1} - 1)}{p_{x,t-1}}\right] \\
&= h - \gamma E_{t-1}\left[\frac{(2\delta x_{t+1} - 1)}{p_{x,t+1}}\right] + (1-\nu-\gamma)\beta\frac{1}{\beta\nu}E_{t-1}\left[\psi(x_{t+1}, \bar{x})\right. \\
&\qquad \left. + h - (1-p_{t+1})\frac{(2\delta x_{t+1} - 1)}{p_{x,t+1}}\right] + \frac{1}{\beta}\left[\frac{(2\delta x_t - 1)}{p_{x,t}}\right]\Bigg].
\end{aligned} \tag{3.30}$$

By imposing perfect foresight on both sides of Eq. (3.30) and multiplying through by $\beta\nu$ gives

$$\begin{aligned}
&\left[\psi(x_t, \bar{x}) + h - (1-p_t)\frac{(2\delta x_t - 1)}{p_{x,t}}\right] - \frac{1}{\beta}\left[\frac{(2\delta x_{t-1} - 1)}{p_{x,t-1}}\right] \\
&= \beta\nu h - \beta\nu\gamma\left[\frac{(2\delta x_{t+1} - 1)}{p_{x,t+1}}\right] + (1-\nu-\gamma)\beta\left[\psi(x_{t+1}, \bar{x})\right. \\
&\qquad \left. + h - (1-p_{t+1})\frac{(2\delta x_{t+1} - 1)}{p_{x,t+1}} + \frac{1}{\beta}\left[\frac{(2\delta x_t - 1)}{p_{x,t}}\right]\right].
\end{aligned}$$

Hence,

$$\begin{aligned}
&\psi(x_t,\bar{x})+h-(1-\nu-p_t)\frac{(2\delta x_t-1)}{p_{x,t}}+\frac{1}{\beta}\left[\frac{(2\delta x_{t-1}-1)}{p_{x,t-1}}\right]\\
&=(1-\nu-\gamma)\beta\left[\psi(x_{t+1},\bar{x})-(1-p_{t+1})\frac{(2\delta x_{t+1}-1)}{p_{x,t+1}}\right]\\
&+(1-\gamma)\beta\left[h+\frac{(2\delta x_t-1)}{\beta p_{x,t}}\right]-\beta\nu\gamma\left[\frac{(2\delta x_{t+1}-1)}{p_{x,t+1}}\right].
\end{aligned}$$

Move one period ahead, then rearrange and take E_t on both sides of the above equation so that we have

$$\begin{aligned}
E_t\left[\frac{(2\delta x_t-1)}{\beta p_{x,t}}\right]=E_t\Bigg[&-\psi(x_{t+1},\bar{x})-h+(1-\nu-p_{t+1})\frac{(2\delta x_{t+1}-1)}{p_{x,t+1}}\\
&+(1-\nu-\gamma)\beta\left[\psi(x_{t+2},\bar{x})-(1-p_{t+2})\frac{(2\delta x_{t+2}-1)}{p_{x,t+2}}\right]\\
&+(1-\gamma)\beta\left[h+\frac{(2\delta x_{t+1}-1)}{\beta p_{x,t+1}}\right]-\beta\nu\gamma\left[\frac{(2\delta x_{t+2}-1)}{p_{x,t+2}}\right]\Bigg].
\end{aligned}\tag{3.31}$$

Therefore,

$$(2\delta x_t-1)=\beta p_{x,t}E_t\left[-[\psi(x_{t+1},\bar{x})+h]+(1-\nu-p_{t+1})\frac{(2\delta x_{t+1}-1)}{p_{x,t+1}}+\beta\tau_{t+2}\right].$$

where

$$\begin{aligned}
\tau_{t+2}=&(1-\nu-\gamma)\left[\psi(x_{t+2},\bar{x})-\frac{(1-p_{t+2})(2\delta x_{t+2}-1)}{p_{x,t+2}}\right]\\
&+(1-\gamma)\left[h+\frac{(2\delta x_{t+1}-1)}{\beta p_{x,t+1}}\right]-\nu\gamma\left[\frac{(2\delta x_{t+2}-1)}{p_{x,t+2}}\right]
\end{aligned}$$

and

$$\psi(x_{t+2},\bar{x})=(x_{t+2}-\delta x_{t+2}^2)-(\bar{x}-\delta\bar{x}^2).$$

Hence, susceptible individuals will choose x_t to satisfy the Euler's equation

$$(2\delta x_t - 1) = \beta p_{x,t} E_t \Big[-[\psi(x_{t+1}, \bar{x}) + h] + (1 - \nu - p_{t+1}) \frac{(2\delta x_{t+1} - 1)}{p_{x,t+1}} + \beta \tau_{t+2} \Big]. \quad (3.32)$$

Eq. (3.21) and (3.32) are identical except for τ_{t+2} in Eq.(3.32). The term τ_{t+2} captures the expected future "costs" of an individual infected but can observe acquired immunity [6]. The term, τ_{t+2} captures the expected future "costs" of infection associated with observed acquired immunity. If τ_{t+2} happens to be less than zero ($\tau_{t+2} < 0$), the possibility of future immunity will be a benefit of becoming infected since it will have an adverse effect on the marginal cost. On the other hand, if τ_{t+1} is positive (i.e $\tau_{t+2} > 0$), becoming infected will be a cost even under the possibility of future immunity.

3.4 The SI(S) model

The SIS and the SI model consider two mutually exclusive disease categories: susceptible (S) and Infected (I). An individual in the susceptible category makes a transition to the infected category when he or she becomes infected and then back to the susceptible category immediately after recovering, the reason being there is no recovery region. That is, the disease in question does not confer any long lasting immunity so there is no need to create the recovered region. An example is the common cold. Fig. 3.2 describes the model. ν in this case is the rate of migrating from the infected group to the susceptible. The other parameters in the model denote those that we explained earlier on. That is, the birth and death rate, probability of infection and the chance of becoming infected with each contact. We therefore have

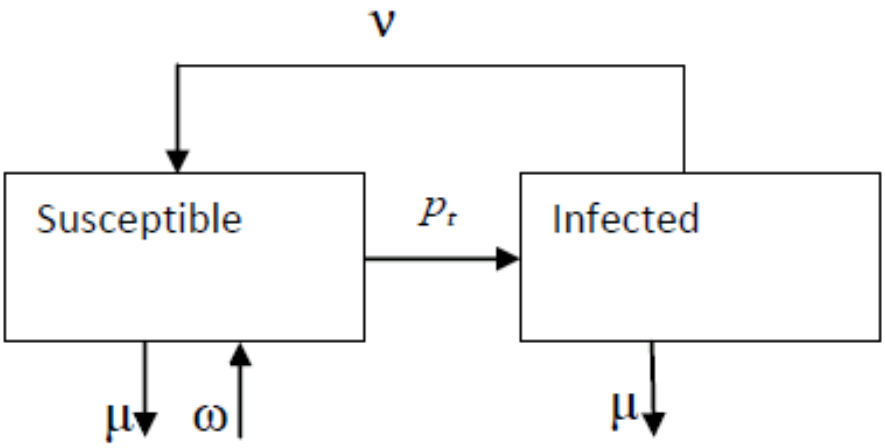


Figure 3.2: The flow chart for the SIS model.

the following equations explaining the model:

$$\begin{cases} s_{t+1} = A\omega + A(1 - p_t - \mu)s_t + A\nu I_t, \\ i_{t+1} = A(1 - \nu - \mu)i_t + Ap_t s_t. \end{cases} \tag{3.33}$$

Where

$$\begin{cases} s_{t+1} = \frac{S_{t+1}}{N_{t+1}}, \\ s_t = \frac{S_t}{N_t}, \\ i_{t+1} = \frac{I_{t+1}}{N_{t+1}}, \\ i_t = \frac{I_t}{N_t}, \\ A = \frac{1}{1+\omega-\mu}, \end{cases} \tag{3.34}$$

and

$$N_{t+1} = (1 + \omega - \mu)N_t,$$

which is explained in the SIR(S) model. If $\nu = 0$ we have the SI model. The SI model is used to study diseases that impose permanent infections. An example is HIV/AIDS. The economic part of the model follows the same reasoning as from the

earlier model. In this model we have the Euler's equation as follows

$$(2\delta x_t - 1) = p_{x,t}\beta E_t\left[-[\psi(x_{t+1}, \bar{x}) + h] + \frac{(1 - \nu - p_{t+1})}{p_{x,t+1}}[2\delta x_{t+1} - 1]\right], \tag{3.35}$$

where

$$\begin{cases} \psi(x_t, \bar{x}) = (x_t - \delta x_t^2) - (\bar{x} - \delta \bar{x}^2), \\ \psi(x_{t+1}, \bar{x}) = (x_{t+1} - \delta x_{t+1}^2) - (\bar{x} - \delta \bar{x}^2), \\ h = h^s - h^i, \end{cases} \tag{3.36}$$

which is similar to the SIR(S) model when individuals do not observe their immunity.

3.5 Analysis Under Rational Expectations

We assumed that the agents or individuals have complete information about the laws of motion for disease states and how their risky activities influence the behaviour of others.[1] Thus, they understand the effect of their risky behaviour on their health status and that of others. In other words, the risk-benefit trade-off faced by others as a result of their behaviour is completely understood. For instance, in the outbreak of HIV, an individual is fully knowledgeable of the risk and the benefit associated with others who are involved in an unprotected sexual act.

3.6 Equilibria

Due to the complex nature of the EE system, we can not have a closed form solution. Therefore, we examined the stability of the system by solving for the steady

[1]The law of motion tells us how the state variable changes over time.

state values of the variables and then linearized around each steady state to evaluate the stability and transition dynamics of the system. The sequence of values $\{s_t, i_t, r_t, x_t\}_{t=0}^{\infty}$ that solve the representative individuals optimization problem and satisfy models (3.7) and (3.33) for all t given the initial values s_0, i_0 and r_0 constitute the equilibrium for the EE systems. The next section is an examination of the long-run equilibrium.

3.6.1 Long-run equilibrium

In the long-run, the EE system has no disturbances and is allowed to converge to its steady state. Therefore, the equilibrium is when changes in time do not have an effect on the values of the variables in the model. Since an infectious disease can be endemic in or may be eradicated from a population, generally there are two possible steady state equilibria: the endemic equilibrium and the eradication equilibrium. For the eradication steady state equilibrium, $s = 1$, $i = r = 0$, and $x = \bar{x}$. Below are the equations relating to the endemic steady state equilibrium for the SIR(S) and the SI(S) [2]:

[2] Aadland et al. pointed out in [6] that the disease eradication steady state for the EE system is locally unstable since individuals do not have any incentive to curb their number of contacts. They choose their number of contacts without any fear of contracting the disease.

The endemic steady state for the EE SIR(S) model

At the endemic steady state, we assume time is invariant. Therefore, we have the following system of equations in four unknown variables (s, i, r, x):

$$\begin{cases} s &= \frac{A(\omega+\gamma r)}{1-A(1-p-\mu)}, \\ i &= \frac{Asp}{1-A(1-\nu-\mu)}, \\ r &= \frac{A\nu i}{1-A(1-\mu-\gamma)}, \\ x &= \frac{\beta}{2\delta}\Big[p_x[\phi\beta\tau - (\psi(x,\bar{x})+h)] + (1-\nu-p)(2\delta x-1)\Big] + \frac{1}{2\delta}. \end{cases} \quad . \tag{3.37}$$

where the Euler Equation either takes the form (3.21) when the indicator variable $\phi = 0$ or the form (3.32), when $\phi = 1$.

$$\tau = \frac{1}{p_x}\Big[(2\delta x-1)\big[\frac{(1-\gamma)}{\beta} - (1-\nu-\gamma)(1-p) - \nu\gamma\big]\Big] + (1-\gamma)h + (1-\nu-\gamma)\psi(x,\bar{x}),$$

where $\psi(x,\bar{x}) = (x-\delta x^2) - (\bar{x}-\delta\bar{x}^2)$.

The endemic steady state for the EE SI(S) model

From the same reasoning as in the case of the E-SIR(S), we have

$$\begin{cases} s &= \frac{A(\omega+\nu i)}{1-A(1-p-\mu)}, \\ i &= \frac{Asp}{1-A(1-\nu-\mu)}, \\ x &= \frac{\beta}{2\delta}\Big[p_x[-(\psi(x,\bar{x})+h)] + (1-\nu-p)(2\delta x-1)\Big] + \frac{1}{2\delta}, \end{cases} \tag{3.38}$$

to solve for $\{s, i, x\}$.

3.6.2 Short-run equilibrium and transition dynamics for the EE SIR(S)

To investigate the stability or the transition paths of the system, we linearized around the endemic steady state by employing first-order Taylor series approximation. Therefore, we have

$$\hat{s}_{t+1} = A(1-p-\mu)\hat{s}_t + A\gamma\hat{r}_t - As\hat{p}_t, \tag{3.39}$$

$$\hat{i}_{t+1} = A(1-\nu-\mu)\hat{i}_t + As\hat{p}_t + Ap\hat{s}_t, \tag{3.40}$$

$$\hat{r}_{t+1} = A(1-\mu-\gamma)\hat{r}_t + A\nu\hat{t}_t\,. \tag{3.41}$$

The hat ($\wedge$) over the variables denotes deviation from the endemic steady state. The linearized Euler equation is below:

$$\begin{aligned} 2\delta p_x\hat{x}_t - (2\delta x-1)\hat{p}_{x,t} &= \beta p_x[p_x(2\delta x-1) + 2\delta(1-\nu-p)]E_t\hat{x}_{t+1} \\ &\quad -\beta(1-\nu-p)(2\delta x-1)E_t\hat{p}_{x,t+1} \\ &\quad -\beta p_x(2\delta x-1)E_t\hat{p}_{t+1} \\ &\quad +\phi\beta^2\Big[\frac{2\delta p_x(1-\gamma)}{\beta}E_t\hat{x}_{t+1} - \frac{(1-\gamma)(2\delta x-1)}{\beta}E_t\hat{p}_{x,t+1} \\ &\quad +p_x[(1-\nu-\gamma)[p_x(1-2\delta x) - 2\delta(1-p)] - 2\delta\nu\gamma]E_t\hat{x}_{t+2} \\ &\quad +[(1-\nu-\gamma)(1-p)+\nu\gamma](2\delta x-1)E_t\hat{p}_{x,t+2} \\ &\quad +p_x(2\delta x-1)(1-\nu-\gamma)E_t\hat{p}_{t+2}\Big], \end{aligned} \tag{3.42}$$

where

$$\hat{p}_t = p_i\hat{i}_t + p_x\hat{x}_t, \tag{3.43}$$

$$\hat{p}_{x,t} = \frac{[1+\ln[1-p]}{x}\hat{p}_t - \frac{p_x}{x}\hat{x}_t, \tag{3.44}$$

and

$$p_i = \frac{\partial p}{\partial i} = x\alpha(1-\alpha i)^{x-1}, \tag{3.45}$$

$$p_x = -\frac{(1-p)}{x}\ln(1-p). \tag{3.46}$$

For the case of unobservable immunity, we set $\phi = 0$. Coupled with the relation $\hat{s}_t = -\hat{r}_t - \hat{i}_t$, and imposing perfect foresight (that is $E_t\hat{x}_{t+1} = \hat{x}_{t+1}$) we have the following EE matrix systems:

$$\begin{aligned}
&\underbrace{\begin{bmatrix} 0 & A(1-\nu-\mu-p) & -Ap \\ 0 & A\nu & A(1-\mu-\gamma) \\ 2\delta p_x & 0 & 0 \end{bmatrix}}_{M_1} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \\ \hat{r}_t \end{bmatrix} \\
&\qquad + \underbrace{\begin{bmatrix} As & 0 \\ 0 & 0 \\ 0 & -(2\delta x - 1) \end{bmatrix}}_{M_2} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} = \\
&\underbrace{\begin{bmatrix} 0 & 1 & 0 \\ 0 & 0 & 1 \\ \beta p_x[p_x(2\delta x - 1) + 2\delta(1-\nu-p)] & 0 & 0 \end{bmatrix}}_{M_3} \begin{bmatrix} \hat{x}_{t+1} \\ \hat{i}_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} \\
&+ \underbrace{\begin{bmatrix} 0 & 0 \\ 0 & 0 \\ -\beta p_x(2\delta x - 1) & -\beta(1-\nu-p)(2\delta x - 1) \end{bmatrix}}_{M_4} \begin{bmatrix} \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix}
\end{aligned} \tag{3.47}$$

and

$$\underbrace{\begin{bmatrix} 1 & 0 \\ -\frac{\left[1+\ln(1-p)\right]}{x} & 1 \end{bmatrix}}_{M_5} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} = \underbrace{\begin{bmatrix} p_x & p_i & 0 \\ -\frac{p_x}{x} & 0 & 0 \end{bmatrix}}_{M_6} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \\ \hat{r}_t \end{bmatrix}. \tag{3.48}$$

Similarly, for observable immunity we set $\phi = 1$. Eq.(3.42) after simplification reduces to

$$\begin{aligned} 2\delta p_x \hat{x}_t - (2\delta x - 1)\hat{p}_{x,t} &= \beta p_x[p_x(2\delta x - 1) + 2\delta(2 - \nu - p - \gamma)]E_t\hat{x}_{t+1} + \\ &\quad \beta^2 p_x[(1 - \nu - \gamma)[p_x(1 - 2\delta x) - 2\delta(1 - p)] - 2\delta\nu\gamma]E_t\hat{x}_{t+2} \\ &\quad -\beta p_x(2\delta x - 1)E_t\hat{p}_{t+1} - \beta(2 - \nu - p - \gamma)(2\delta x - 1)E_t\hat{p}_{x,t+1} \\ &\quad +\beta^2 p_x(2\delta x - 1)(1 - \nu - \gamma)E_t\hat{p}_{t+2} + \beta^2[(1 - \nu - \gamma)(1 - p) \\ &\quad +\nu\gamma](2\delta x - 1)E_t\hat{p}_{x,t+2}. \end{aligned} \tag{3.49}$$

Therefore, imposing perfect foresight (that is $E_t x_{t+1} = x_{t+1}$), we have the following as the linearized EE matrix system:

$$\underbrace{\begin{bmatrix} 0 & A(1-\nu-\mu-p) & -Ap & 0 & 0 \\ 0 & A\nu & A(1-\mu-\gamma) & 0 & 0 \\ 2\delta p_x & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix}}_{M_1} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \\ \hat{r}_t \\ \hat{x}_{t+1} \\ \hat{i}_{t+1} \end{bmatrix} + \underbrace{\begin{bmatrix} As & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & -(2\delta x-1) & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}}_{M_2} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \\ \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix} =$$

$$\underbrace{\begin{bmatrix} 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 \\ \beta p_x[p_x(2\delta x-1)+2\delta(2-\nu-p-\gamma)] & 0 & 0 & \beta^2 p_x[(1-\nu-\gamma)[p_x(1-2\delta x)-2\delta(1-p)]-2\delta\nu\gamma] & 0 \\ 1 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 \end{bmatrix}}_{M_3} \begin{bmatrix} \hat{x}_{t+1} \\ \hat{i}_{t+1} \\ \hat{r}_{t+1} \\ \hat{x}_{t+2} \\ \hat{i}_{t+2} \end{bmatrix} +$$

$$(2\delta x-1) \underbrace{\begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -\beta p_x & -\beta(2-\nu-p-\gamma) & \beta^2 p_x(1-\nu-\gamma) & \beta^2[(1-\nu-\gamma)(1-p)+\nu\gamma] \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}}_{M_4} \begin{bmatrix} \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \\ \hat{p}_{t+2} \\ \hat{p}_{x,t+2} \end{bmatrix} \quad (3.50)$$

and

$$\underbrace{\begin{bmatrix} 1 & 0 & 0 & 0 \\ -\frac{(1+\ln(1-p)}{x} & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & -\frac{(1+\ln(1-p)}{x} & 1 \end{bmatrix}}_{M_5} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \\ \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix} = \underbrace{\begin{bmatrix} p_x & p_i & 0 & 0 & 0 \\ -\frac{p_x}{x} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & p_x & p_i \\ 0 & 0 & 0 & -\frac{p_x}{x} & 0 \end{bmatrix}}_{M_6} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \\ \hat{r}_t \\ \hat{x}_{t+1} \\ \hat{i}_{t+1} \end{bmatrix}. \quad (3.51)$$

If we let $\hat{Z}_t = (\hat{x}_t, \hat{i}_t, \hat{r_t})^T$ or $\hat{Z}_t = (\hat{x}_t, \hat{i}_t, \hat{r}_t, \hat{x}_{t+1}, \hat{i}_{t+1})^T$ and $\hat{P}_t = (\hat{p}_t, \hat{p}_{x,t})^T$ or $\hat{P}_t = (\hat{p}_t, \hat{p}_{x,t}, \hat{p}_{t+1}, \hat{p}_{x,t+1})^T$then we have

$$M_1\hat{Z}_t + M_2\hat{P}_t = M_3\hat{Z}_{t+1} + M_4\hat{P}_{t+1} \quad (3.52)$$

and

$$M_5\hat{P}_t = M_6\hat{Z}_t. \tag{3.53}$$

Therefore, the EE system reduces to

$$\hat{Z}_t = J\hat{Z}_{t+1}, \tag{3.54}$$

where

$$J = (M_1 + M_2M_5^{-1}M_6)^{-1}(M_3 + M_4M_5^{-1}M_6).$$

The method proposed by Blanchard and Kahn [15] was employed in the analysis of the stability of the linearized EE system around the endemic steady state. Considering the three-variable system, Eq.(3.54), we have one non-predetermined, $\hat{x}_t$ and two predetermined ($\hat{i}_t$ and $\hat{r}_t$) variables. If we have exactly two eigenvalues of J outside the unit circle, the system exhibits a stable saddle-path. On the other hand, the system will exhibit indeterminate multiple stable paths or a sink if all the eigenvalues of J are unstable (that is, are more than one) and explosive paths if the forward stable eigenvalues (eigenvalues less than the unit circle) of J are more than one. The five-variable system has three non-predetermined ($\hat{x}_t, \hat{x}_{t+1},$ and $\hat{i}_{t+1}$) and two predetermined (i_t and $\hat{r}_t$) variables. Following the same chain of analysis: the system will exhibit a saddle-path stability if we have exactly two eigenvalues outside the unit circle, indeterminate multiple path stability if we have more than two unstable eigenvalues, and explosive paths if we have less than two unstable eigenvalues.

We must note here that Eq.(3.54) could take the following form:

$$Z_{t+1} = J^{-1}Z_t\,, \tag{3.55}$$

where

$$J^{-1} = (M_3 + M_4 M_5^{-1} M_6)^{-1}(M_1 + M_2 M_5^{-1} M_6).$$

In this case, the eigenvalues for the matrix J will be the reciprocal of the one considered in Eq.(3.54). The stability condition will be as follows: For the three variables system, if the number of the unstable eigenvalue is one, then we have a stable-path stability. If we have no unstable eigenvalue, then we have indeterminate multiple path stability, and an explosive path if we have more than one unstable eigenvalue. With the five variable systems, if we have exactly three unstable eigenvalues, then we have a saddle-path stability. If we have less than three unstable eigenvalues then we have indeterminate multiple paths stability and if we have more than three unstable eigenvalues, then we have explosive paths.

3.6.3 Short-Run Equilibrium and Transition Dynamics for the EE SI(S)

Similarly, the linearized system around the endemic steady state is as follows:

$$\hat{s}_{t+1} = A(1-\mu-p)\hat{s}_t + A\nu\hat{i}_t - As\hat{p}_t, \tag{3.56}$$

$$\hat{i}_{t+1} = A(1-\nu-\mu)\hat{i}_t + As\hat{p}_t + Ap\hat{s}_t. \tag{3.57}$$

The following is the linearized Euler equation :

$$\begin{aligned} 2\delta p_x \hat{x}_t - (2\delta x - 1)\hat{p}_{x,t} = {} & \beta p_x[p_x(2\delta x - 1) + 2\delta(1-\nu-p)]E_t\hat{x}_{t+1} \\ & - \beta(1-\nu-p)(2\delta x - 1)E_t\hat{p}_{x,t+1} \\ & - \beta p_x(2\delta x - 1)E_t\hat{p}_{t+1}. \end{aligned} \tag{3.58}$$

Using the relation $\hat{s}_t + \hat{i}_t = 0$ we have Eq.(14) as

$$\hat{i}_{t+1} = A(1-\nu-\mu-p)\hat{i}_t + A(1-i)\hat{p}_t, \text{ from } s+i=1. \tag{3.59}$$

For the same reason as from earlier discussions we have the following as the EE matrices:

$$\underbrace{\begin{bmatrix} 0 & A(1-\nu-\mu-p) \\ 2\delta p_x & 0 \end{bmatrix}}_{N_1} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \end{bmatrix} + \underbrace{\begin{bmatrix} A(1-i) & 0 \\ 0 & -(2\delta x - 1) \end{bmatrix}}_{N_2} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix}$$

$$\underbrace{\begin{bmatrix} 0 & 1 \\ \beta p_x[p_x(2\delta x - 1) + 2\delta(1-\nu-p)] & 0 \end{bmatrix}}_{N_3} \begin{bmatrix} \hat{x}_{t+1} \\ \hat{i}_{t+1} \end{bmatrix} + \underbrace{\begin{bmatrix} 0 & 0 \\ -\beta p_x(2\delta x - 1) & -\beta(1-\nu-p)(2\delta x - 1) \end{bmatrix}}_{N_4} \begin{bmatrix} \hat{p_{t+1}} \\ \hat{p}_{x,t+1} \end{bmatrix} \tag{3.60}$$

and

$$\underbrace{\begin{bmatrix} 1 & 0 \\ -\frac{(1-\ln(1-p)}{x} & 1 \end{bmatrix}}_{N_5} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} = \underbrace{\begin{bmatrix} p_x & p_i \\ -\frac{p_x}{x} & 0 \end{bmatrix}}_{N_6} \begin{bmatrix} \hat{x}_t \\ \hat{i}_t \end{bmatrix}. \tag{3.61}$$

Let $\hat{Z}_t = (\hat{x}_t, \hat{i}_t)^T$ and $\hat{Q}_t = (\hat{p}_t, \hat{p}_{x,t})^T$ so that the system reduces to

$$\hat{Z}_t = J\hat{Z}_{t+1}. \tag{3.62}$$

where

$$J = (N_1 + N_2 N_5^{-1} N_6)^{-1}(N_3 + N_4 N_5^{-1} N_6).$$

From the above equation, we have one non-predetermined $\{\hat{x}_t\}$and predetermined$\{\hat{i}_t\}$ variable. If we have exactly one unstable eigenvalue, then we have saddle-path stability. If there are two unstable eigenvalues, then the system will exhibit indeterminate multiple paths stability. And if there is no unstable eigenvalue, then we have explosive

paths.

In the same way, Eq. (3.62) could be written as Eq. (3.55). Also, in this case, if we have exactly one eigenvalue outside the unit circle we have stable saddle-path. If there is no eigenvalue outside the unit circle then we have indeterminate multiple stable paths. Finally, if the two eigenvalues are unstable, then we have a source (explosive paths). In other words there is no solution.

3.7 Numerical Solution

We employed Maple in our simulation for the various types of the EE models. The simulation carried out is to find out the effects of the health gap ($h = h^s - h^i$) and the infection parameter (α) on the dynamics of the system. We simulate for the various values for δ (determinant of maximum contacts) coupled with the birth and death rate, ω and μ respectively. What we did was to vary δ whilst we hold μ and ω constant. We did the converse.

We found the steady state values for the disease categories for the range of parameter values for α and h and then determined their dynamic paths by finding the eigenvalues for J. We then plotted the point (α, h).

These two parameters mentioned above are the possible public health policy targets, in that, a high h means the health gap of individuals within the population is high. This raises concern. Therefore, for h to be maintained at a low level, investment could be made into drugs or medication. Also, α can also be maintained low by the introduction of vaccines or a new way of protecting the population from being infected [26]. A high α means the disease can spread quickly among the population.

This section discusses the dynamic paths of the EE models around the endemic steady state. As we said earlier on that, if $i = r = 0,$ then we have $s = 1$. This case demonstrates unstable steady path dynamics, indicating that individuals do not have any incentive to curb their number of contacts as they face no risk.

The red, green, yellow and black color in the figures indicate the region for saddle-path equilibria (stability), indeterminate multiple paths stability, explosive paths and where individuals are going for maximum contacts. The selection of the values for β, ν and γ (see Table 3.1) implies that the annual discount rate is 4%, 100% recovery rate within a year of infection, and an expected 5-year immunity duration respectively. β is found by using the following relation:

$$\beta = \frac{1}{(1+r)^t}, \tag{3.63}$$

where r is the annual discount rate. Based on the assumption that individuals live within a single period ,we can set time t to one.

Table 3.1: Parameter values for numerical analysis

Parameters	β	ν	γ
Values	0.96	1	$0.2yr^{-1}$

3.7.1 Unobservable host immunity

Simulations and policy implications for the EE SIR model

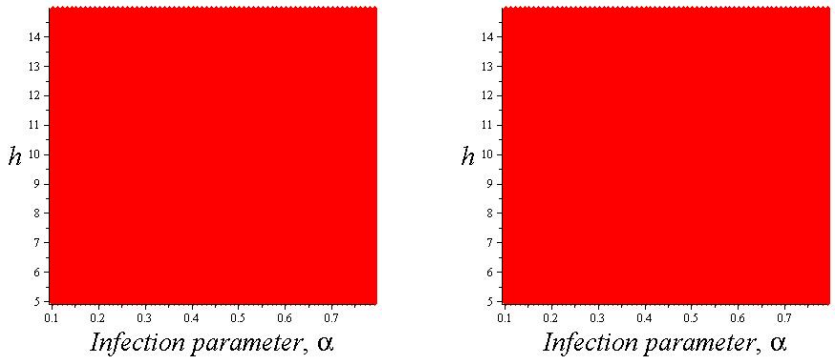
The EE SIR model is a special case of the EE SIRS when $\gamma = 0$ (confirming permanent immunity). We simulate for various combinations for birth rate ω and death rate μ.

Figs. 3.3a and 3.3b show the dynamic paths for $\omega > \mu$ and $\omega = \mu$ at $\delta = 0.025$ respectively. The system exhibits saddle-paths equilibria (red region) for all combinations for h and α. It implies that individuals have contact levels less than the maximum allowable, $\bar{x}$. Therefore, public policy targeted at reducing the health gap (or improving the health of infected individuals) would not affect the stability of the system.

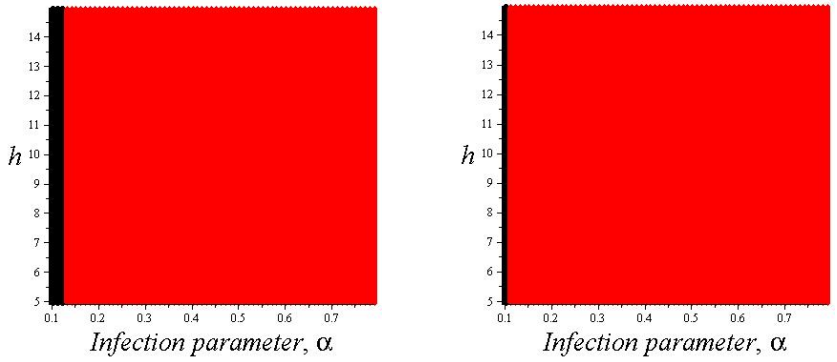
Figs. 3.3c and 3.3d shows the dynamic paths for $\omega > \mu$ and $\omega = \mu$ at $\delta = 0.05$ respectively. For $\omega > \mu$, at very low values for α, individuals are going for the maximum number of contacts (black region). This indicates that because there is permanent cure or immunity for disease, coupled with low probability of infection, individuals are involving in risky behaviour. This is also attributed to the fact that individuals are aware that future cost of contracting the disease is low. The rest of the region exhibits saddle-path equilibria (red region). For $\omega = \mu$ (Fig. 3.3d), the system shows similar pattern, but with a smaller maximum–contact region. Therefore, policy direction towards the reduction of the level of contacts may not be effective as individuals will place much importance on the benefit associated with going for maximum number of contacts.

These results show that δ plays a significant role in determining the stability of the system. The only case for which μ and ω has a somewhat significant effect on the EE SIR system is when $\delta = 0.05$. This shows that further increase in δ will produce maximum contact[3] region for wide parameter values for h and α.

[3]We simulated for $\delta = 0.04$ and the pattern shows that as we increase the values for δ, the region for individuals going for $\bar{x}$ becomes wider.



(a) $\delta = 0.025, \omega = 0.06, \mu = 0.05$ (b) $\delta = 0.025, \omega = 0.05, \mu = 0.05$



(c) $\delta = 0.05, \omega = 0.06, \mu = 0.05$ (d) $\delta = 0.05, \omega = 0.05, \mu = 0.05$

Figure 3.3: Dynamic paths for the EE SIR model for unobservable host immunity.

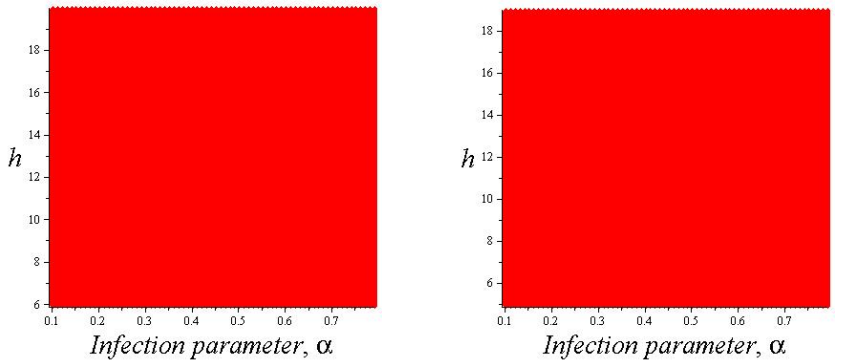
Simulation and policy implication for EE SIRS model

Assume an average duration of immunity is five years ($\gamma = 0.2$). Figs. 3.4a and 3.4b shows simulation for the dynamic paths for $\omega > \mu$ and $\omega = \mu$ at $\delta = 0.025$ respectively. As in the case for the EE SIR system discussed above, the system exhibits saddle-paths equilibria (red region) for the entire parameter combination for h and α, indicating that when individuals behave rationally, they will always go for contact levels less than $\bar{x}$.

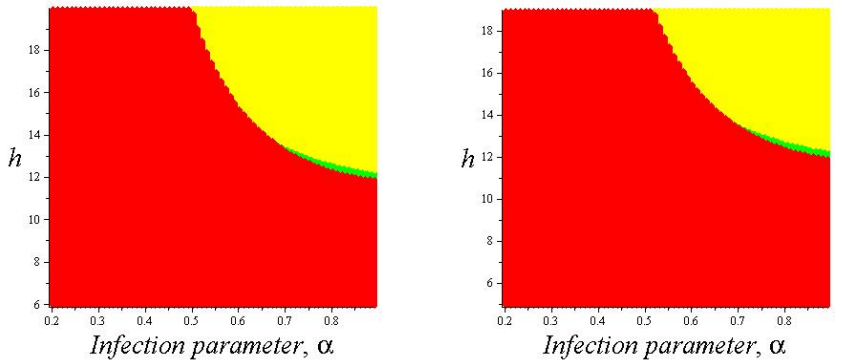
Figs. 3.4c and 3.4d show simulation for the dynamic paths for $\omega > \mu$ and $\omega = \mu$ at $\delta = 0.05$ respectively. It is noticed that the system exhibited saddle-path stability (red region) for all values of h and low range of values for α and low values for h and

the entire range of values for α. It also exhibits some indeterminate path dynamics (green region) for parameter values for high values for α and moderate values for h. Finally, when $\omega \geq \mu$ and both α and h are high, explosive paths (yellow region) are expressed. It is noticed that, since there is no permanent immunity, rational individuals are going for a number of contacts less than the maximum.

The above discussion implies that the birth and death rate do not have an effect on the dynamic paths of this system. On the other hand, it is evident that δ does have significant effect, thus the assumption of $\overline{x}$ depending on a parameter led a different outlook for the dynamic paths.



(a) $\delta = 0.025, \omega = 0.06, \mu = 0.05$ (b) $\delta = 0.025, \omega = 0.05, \mu = 0.05$



(c) $\delta = 0.05, \omega = 0.06, \mu = 0.05$ (d) $\delta = 0.05, \omega = 0.05, \mu = 0.05$

Figure 3.4: Dynamic paths for the EE SIRS model for unobservable host immunity

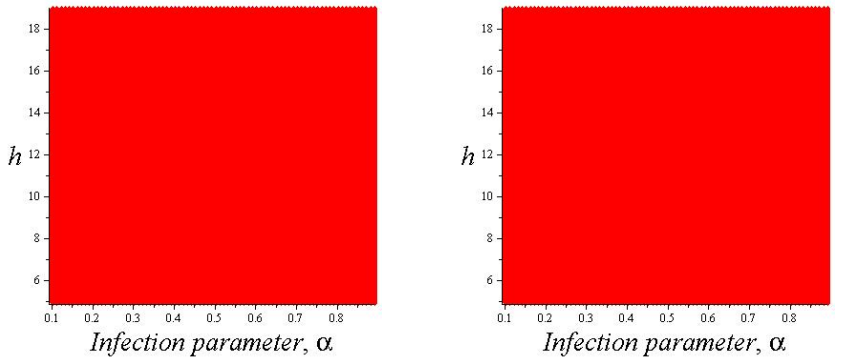
3.7.2 Observable host immunity

Just as in the case for unobservable host immunity, we carried out simulations for EE SIR(S) under the assumption that individuals observe their immunity.

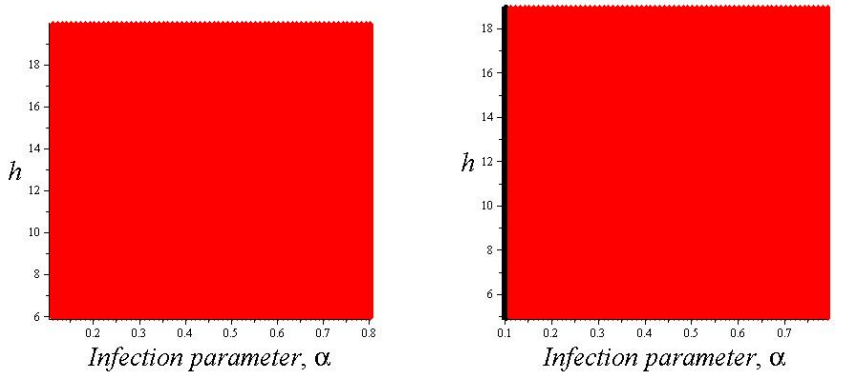
Simulations for EE SIR(S) model and policy implication

Figs. 3.5c–3.5d show the dynamic paths for the EE SIR system. For unequal birth and death rate and δ set at 0.05, the system exhibits saddle-paths stability (red region) for all the parameter combinations of h and α. The same pattern holds when we simulated for equal birth and death rates. But it is observed that individuals are going for $\bar{x}$ at very low values for α. Both cases (that is $\omega \geq \mu$) indicate that as rational individuals observe their immunity against a particular infectious disease, they are still conscious of the health status of others and themselves and thus will opt to go for a number of contact less than the maximum allowable. Figs. 3.5c and 3.5d indicate these dynamics. Figs. 3.5a and 3.5b exhibit the same dynamic paths as in the case for $\omega > \mu$ with δ set at 0.05.

Figs. 3.6a-3.6d show the dynamic paths for the EE SIRS system. All the parameter combinations yielded the same dynamic paths. That is, they exhibited saddle-paths equilibria (red region) for all the possible combinations of h and α given the respective values for ω and μ. These results mean that public policy direction (whether to reduce h or α or both) will not have any bearing on the stability properties of the system.



(a) $\delta = 0.025, \omega = 0.06, \mu = 0.05$ (b) $\delta = 0.025, \omega = 0.05, \mu = 0.05$



(c) $\delta = 0.05, \omega = 0.06, \mu = 0.05$ (d) $\delta = 0.05, \omega = 0.05, \mu = 0.05$

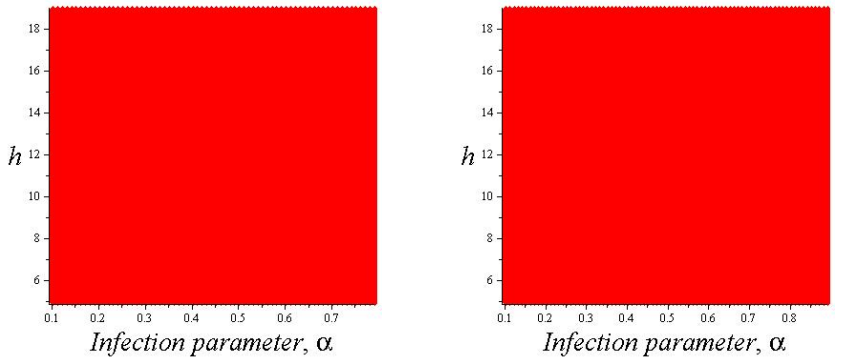
Figure 3.5: Dynamic paths for the EE SIR model for observable host immunity

3.7.3 Simulation for the SI(S) model

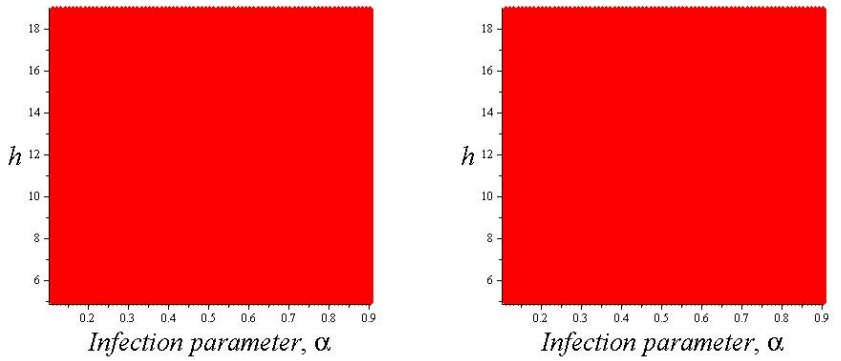
Simulation for the EE SI model

For the simulation for the EE SI system, we set $\nu = 0$, which is indicative of no treatment being available for the disease. We can talk of HIV as an example in this case. Figs. 3.7a and 3.7b show the dynamic paths of this system at $\delta = 0.025$ with $\omega > \mu$ and $\omega = \mu$ respectively. Both cases show that rational individuals will go for $\bar{x}$ irrespective of the level of infection parameter and health gap. Thus, public health policy will have no effect on the number of contacts made by these individuals.

Figs. 3.7c and 3.7d have the parameters $\delta = 0.05$ with $\omega > \mu$ and $\omega = \mu$



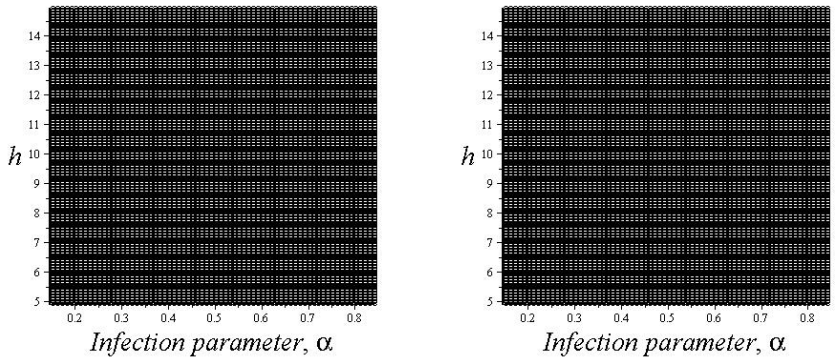
(a) $\delta = 0.025, \omega = 0.06, \mu = 0.05$ (b) $\delta = 0.025, \omega = 0.05, \mu = 0.05$



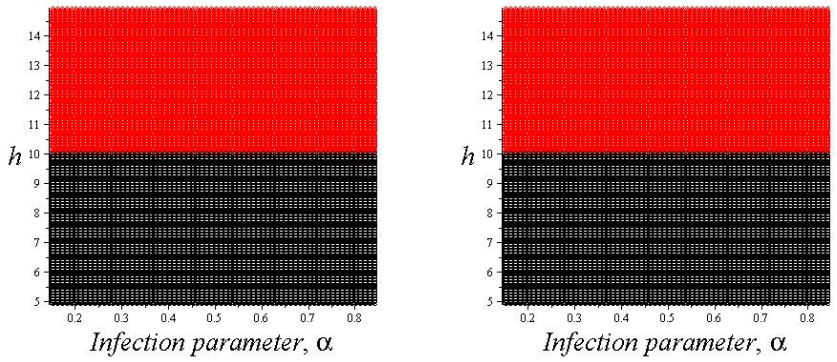
(c) $\delta = 0.05, \omega = 0.06, \mu = 0.05$ (d) $\delta = 0.05, \omega = 0.05, \mu = 0.05$

Figure 3.6: Dynamic paths for the EE SIRS model for observable host immunity

respectively. Both cases show saddle-path stability (red region) for high values of h given the entire range of values for α. This demonstrates that, at a high level of health gap, rational individuals are willing to choose a number of contacts less than $\bar{x}$. On the other hand, low values of h yield a case where rational individuals are choosing $\bar{x}$ (black region), indicating that, irrespective of the level of infection parameter they are willing to involve in risky behaviour by going for the $\bar{x}$. It also shows that δ has a significant effect on the properties of the system.



(a) $\delta = 0.025, \omega = 0.06, \mu = 0.05$ (b) $\delta = 0.025, \omega = 0.05, \mu = 0.05$



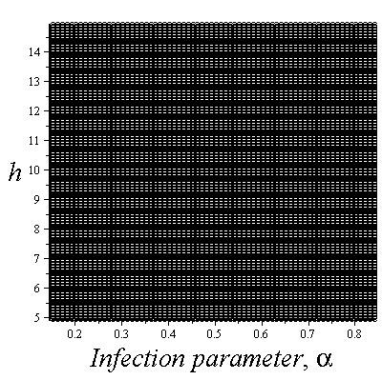
(c) $\delta = 0.05, \omega = 0.06, \mu = 0.05$ (d) $\delta = 0.05, \omega = 0.05, \mu = 0.05$

Figure 3.7: Dynamic paths for the EE SI model

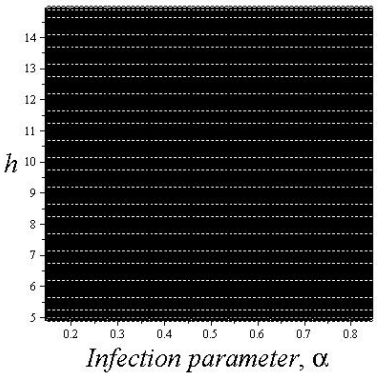
Simulation for the EE SIS model

As in the case for the EE SI system, the EE SIS system shows the same property for the parameter combination of $\omega > \mu$ and $\omega = \mu$ for $\delta = 0.025$, in that, irrespective of the levels of infection and health gap, rational individuals are going for the maximum number of contacts. Fig. 3.8a and 3.8b demonstrate the results.

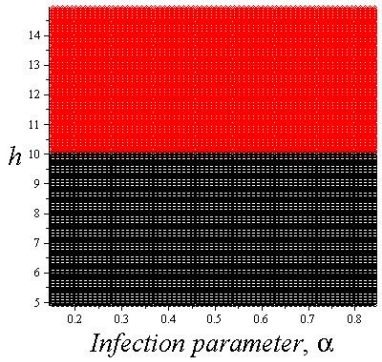
Fig. 3.8c and 3.8d show the dynamic paths for the system for $\omega > \mu$ and $\omega = \mu$ for $\delta = 0.025$ respectively. For $\omega > \mu$, the system exhibited the same property for the EE SI system. On the other hand, for $\omega = \mu$ the property is different, in that, the system exhibited explosive paths for parameter combinations of high values of h and α.



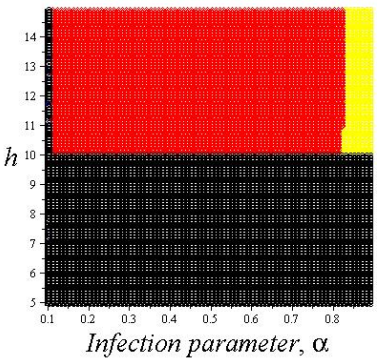
(a) $\delta = 0.025, \omega = 0.06, \mu = 0.05$



(b) $\delta = 0.025, \omega = 0.05, \mu = 0.05$



(c) $\delta = 0.05, \omega = 0.06, \mu = 0.05$



(d) $\delta = 0.05, \omega = 0.05, \mu = 0.05$

Figure 3.8: Dynamic paths for the EE SIS model

Chapter 4

Stability Properties of an Economic Epidemiology Model of Syphilis Infectivity among Male Inmates

4.1 Syphilis

Treponema pallidun is the bacterium that causes syphilis [5]. When one is infected with the disease it shows mild symptoms. The disease manifests itself in four main stages: The primary, secondary, latent and tertiary(late) stage.

First, when one comes into contact with a person with the infectious lesions (typically through a sexual act), the point of contact becomes infected [25]. There is the appearance of a skin lesion called ulceratic chancre at the point of contact.

This is the primary stage of the disease. If the disease is left untreated, it then progresses to the secondary stage where the infected person experiences symptoms such as fatigue and loss of appetite, swollen glands, a non-itchy rash covering the entire body and other symptoms. It must be pointed out that the disease is quite infectious at these two stages. If the disease progresses to the secondary stage and it is left untreated it then progresses to the latent stage where the person does not experience any symptoms of the disease. The disease is not contagious at this stage. The disease then progresses to the stage where it causes a lot of damage to the internal organs of the infected person. It can affect the heart and the nervous system. This stage is what is termed the tertiary(late) stage [25]. Syphilis can be treated.

4.2 The Epidemiological Model

We start the modelling process by first modelling the epidemiology of the disease among the inmates. Let N be the entire population of the male inmates. We divided the population into the following disease categories:

S^{nh} : Susceptible non-MSM,

S^{h} : Susceptible MSM,

I^{nh} : Non-MSM syphilis infectives,

I^{h} : MSM syphilis infectives,

R^{nh} : Non-MSM who attained recovery from the disease,

R^{h} : MSM who attained recovery from the disease.

Therefore,

$$N = S^{nh} + S^h + I^{nh} + I^h + R^{nh} + R^h. \tag{4.1}$$

Suppose the rate at which individuals are recruited into prison is fixed and is given as Λ. Λ here denotes the number of recruits into prison per the total population of a jurisdiction. The unit for prison incarceration rate is given as the number of prisoners per 100,000 population of a jurisdiction. For example, the incarceration rate of the USA in 2008 is 756 per 100,000 [44]. Let π_0, π_1, π_2, and π_3 be the proportion of individuals who are recruited into S^{nh}, S^h, I^{nh}, and I^h. This implies that new recruits either enter the susceptible or the infected disease categories. Let ω be the rate at which individuals are released from the prison so that $\omega S^{nh}, \omega S^h, \omega I^{nh}, \omega I^h, \omega R^{nh}$ and ωR^h individuals leave the prison from the respective disease category (here, we assumed that, the rate ω is fixed throughout the disease category). We assumed that the natural death rate μ is fixed throughout the disease category. Let σ denotes the recovery rate of syphilis for infected non-MSM I^{nh} and MSM I^h respectively. Furthermore, let us suppose that the rate at which individuals who are not MSM become MSM is fixed and is denoted as α. Also, let γ denotes the rate at which recovered MSM become susceptible to the disease again (we assumed permanent recovery for non-MSM since they will not involve in sexual activity). There is no disease related death. Finally, the probability of susceptible MSM who after being involved in a risky behaviour (having unprotected sex: we assumed throughout the analysis of the model on this basis) become infected is given as p_t and if remain uninfected is given as $(1 - p_t)$. Fig. 4.1 shows the flow chart for the model. Based on the discussion above, the model can

be explained by the following discrete equations:

$$S_{t+1}^{nh} = \Lambda\pi_0 + (1-\omega-\mu-\alpha)S_t^{nh}, \tag{4.2}$$

$$S_{t+1}^{h} = \Lambda\pi_1 + (1-\omega-\mu-p_t)S_t^{h} + \alpha S_t^{nh} + \gamma R_t^{h}, \tag{4.3}$$

$$I_{t+1}^{nh} = \Lambda\pi_2 + (1-\omega-\mu-\sigma-\alpha)I_t^{nh}, \tag{4.4}$$

$$I_{t+1}^{h} = \Lambda\pi_3 + p_t S_t^{h} + (1-\omega-\mu-\sigma)I_t^{h} + \alpha I_t^{nh} \tag{4.5}$$

$$R_{t+1}^{nh} = \sigma I_t^{nh} + (1-\omega-\mu)R_t^{nh}, \tag{4.6}$$

$$R_{t+1}^{h} = \sigma I_t^{h} + (1-\omega-\mu-\gamma)R_t^{h}. \tag{4.7}$$

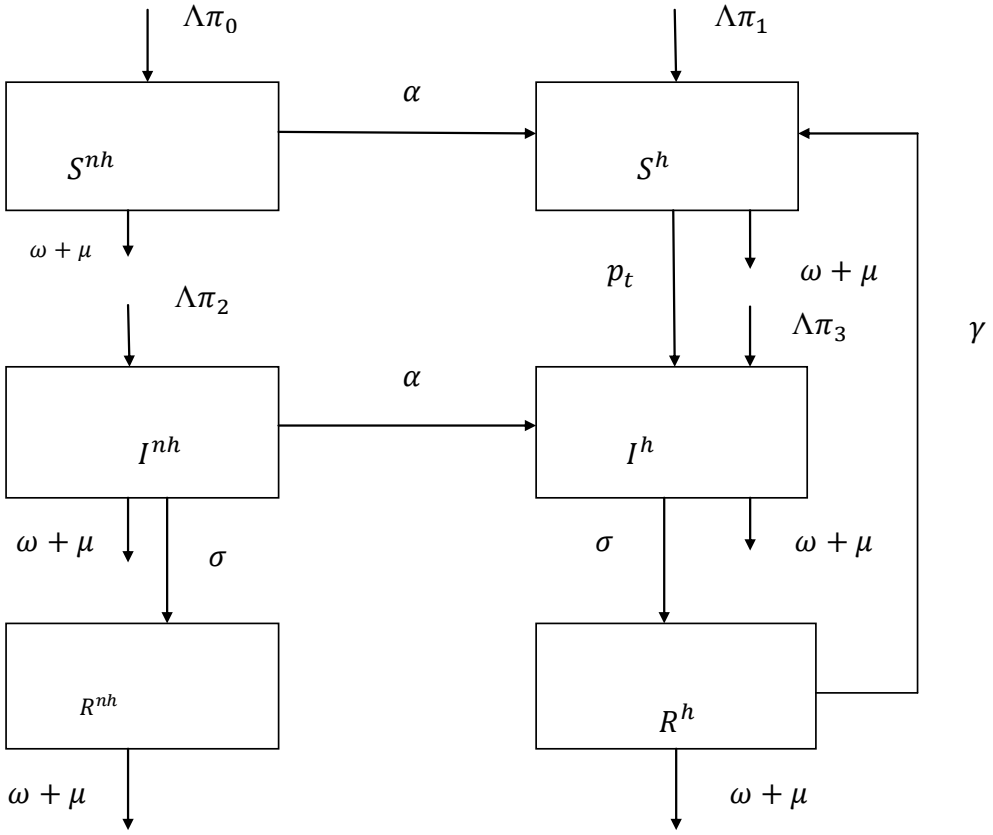


Figure 4.1: Model flow chart

The summation of Eq. (4.2)–(4.7) gives the number of the host population in the prison as follows:

$$N_{t+1} = N_t + \Lambda - (\omega + \mu)N_t. \tag{4.8}$$

Eq. (4.8) has the following solution:

$$N_t = \frac{\Lambda(1 - (1 - \omega - \mu)^t)}{\omega + \mu} + (1 - \omega - \mu)^t N_0,$$

given that $\omega + \mu < 1$ and the initial value of N is N_0. Based on the solution for the number of inmates, we have

$$\lim_{t\to\infty} N_t = \frac{\Lambda}{\omega + \mu},$$

which is independent of the initial number of the inmates. Hence, the steady state of the population of inmates is globally stable.

Since Eq.(4.2) and (4.4) are independent of the other states variables (that is S_t^h, I_t^h, R_t^{nh} and R_t^h), we can analyse the model in terms of Eq. (4.3), (4.5), (4.6) and (4.7). Therefore, we have the following:

$$\begin{cases} S_{t+1}^h = \Lambda\pi_1 + (1 - \omega - \mu - p_t)S_t^h + \alpha S^{nh} + \gamma R_t^h, \\ I_{t+1}^h = \Lambda\pi_3 + p_t S_t^h + (1 - \omega - \mu - \sigma)I_t^h + \alpha I^{nh}, \\ R_{t+1}^{nh} = \sigma I^{nh} + (1 - \omega - \mu)R_t^{nh}, \\ R_{t+1}^h = \sigma I_t^h + (1 - \omega - \mu - \gamma)R_t^h, \end{cases} \tag{4.9}$$

where

$$\begin{cases} S^{nh} = \frac{\Lambda\pi_0}{\omega+\mu+\alpha} & \text{and} \\ I^{nh} = \frac{\Lambda\pi_2}{\omega+\mu+\sigma+\alpha} \end{cases}$$

are the steady states for the susceptible and infected non-MSM population.

In modelling the probability of infection, we followed the approach employed in [6]. Let λ_p and λ_a be the probability of contracting syphilis from a single infected partner and a single sexual act respectively. We can model λ_p as follows:

$$\lambda_p = 1 - (1 - \lambda_a)^a. \tag{4.10}$$

λ_p and λ_a are noted as natural probabilities [6]. a is the number of fixed sexual acts by individuals. Suppose individuals (n) choose x_t number of contacts at time t. Then we have the probability of infection as

$$\Pr(\text{infection}) = p_t = 1 - \left(1 - \lambda_p \frac{I_t^h}{N_t^h}\right)^{x_t}, \tag{4.11}$$

where $N_t^h = I_t^h + S_t^h + R_t^h$ is the total number of inmates who are MSM at time t. The implication of the above expression for p_t is that, unlike the classical mathematical epidemiological models (examples are those studied in [11],[37],[39],[35]), the probability of a susceptible MSM becoming infected is endogenous and is dependent on the natural probabilities of infection from one sexual act and from a single infected partner as well as his number of sexual partners. A susceptible MSM who involved in a risky behaviour and is not infected with the disease has the probability of not being infected as follows:

$$\Pr(\text{remaining uninfected}) = \left(1 - \lambda_p \frac{I_t^h}{N_t^h}\right)^{x_t}. \tag{4.12}$$

The next section talks about the economic analysis and the optimal choice of partners a MSM in any of the disease category would be making.

4.3 Introducing Economics

Suppose that an individual (n) MSM goes for a number of partners $x_{n,t}$ such that his expected lifetime utility is maximized. We have the following as the individual's objective function:

$$E\sum_{j=0}^{\infty}\beta^{j}\left[(x_{n,t+j}-\delta x_{n,t+j}^{2})+h_{n,t+j}\right], \tag{4.13}$$

where $0<\beta<1$ is the discount factor, E is the mathematical expectation operator of future outcomes. h in the objective function captures the health status of a MSM in any of the disease categories with high and low values of h associated with the health status for a susceptible and infected MSM respectively. The underlying trade-off in the model is that there is an immediate satisfaction associated with any additional number of sexual partners a MSM chooses, but associated with this choice is a risk of future infection. Therefore, if one becomes infected, the infection deteriorates his health. A MSM can only be in one disease category at a time. That is, either the individual is in the susceptible, infected, recovered MSM or recovered non-MSM category. Suppose that individual inmates are identical with the exception of their sexual orientation and health status. Also, since the infected non-MSM receive permanent recovery and thus will not be involving in any sexual activity in the prison, we formulate the value function for only the MSM population for each disease category. Furthermore, it is pointed out in [6] that it is difficult for one to observe syphilis immunity and that, individuals in the recovered category can not tell whether they are immune to syphilis. Therefore, we ignore the recovered category for the MSM. Based on the assumption that individuals are identical with the exception of their health status and sexual orientation, the analysis is conducted in terms of a single individual so the index n is dropped. The value functions for the category for the

susceptible and infected MSM are given below:

$$V_t^{S^h} = x_t - \delta x_t^2 + h^{S^h} + \beta E\left[p_t V_{t+1}^{I^h} + (1-p_t)V_{t+1}^{S^h}\right], \tag{4.14}$$

$$V_t^{I^h} = \bar{x} - \delta \bar{x}^2 + h^{I^h} + \beta E\left[\sigma V_{t+1}^{S^h} + (1-\sigma)V_{t+1}^{I^h}\right], \tag{4.15}$$

where h^{S^h} and h^{I^h} are the health parameters for a susceptible and a syphilis–infected MSM respectively, $h^{S^h} > h^{I^h}$ and $\bar{x} = 1/2\delta$ is the maximum number of partners. We assume that MSM in the prison(s) do not consider the general welfare of their fellow inmates and thus behave in a self centred manner irrespective of their health status (that is, whether they are infected or not). Therefore, individuals in the infected category will go for the maximum number of partners because they do not face immediate risk of infection [29]. Eq. (4.14) and (4.15) are the value functions associated with the optimal decision made by an individual susceptible and infected MSM respectively. The term in Eq. (4.14) is the expected future utility which is dependent on the expected future infection levels. The present value of the expected future utility is $V_{t+1}^{S^h}$ if the individual remains susceptible and $V_{t+1}^{I^h}$ is the present value of expected future utility if an individual becomes infected after choosing a number of sexual partners at time t. On the other hand, the term in the bracket for the value function for the infected MSM is the present value for expected future utility for a syphilis infected individual: with $V_{t+1}^{S^h}$ or $V_{t+1}^{I^h}$ being the expected future utility in case the individual recovers or the individual remains infected in the next period.

Suppose a susceptible MSM chooses an optimal number of partners such that the first order necessary condition is satisfied. Then we have

$$2\delta x_t - 1 = -\beta p_{x,t} E\left[V_{t+1}^{S^h} - V_{t+1}^{I^h}\right], \tag{4.16}$$

where

$$p_{x,t} = \frac{\partial p_t}{\partial x_t} = -\frac{1-p_t}{x_t}\ln(1-p_t). \tag{4.17}$$

Eq. (4.16) measures the expected marginal damage cost and benefit of a susceptible MSM any time he increases his current number of partners. The terms on the right and left hand side of the equation are the expected marginal damage cost and marginal benefit respectively. The implication of this is that a susceptible MSM will choose a number of sexual partners such that his marginal benefits and expected marginal cost are equal. Because individuals determine the number of partners independently, the probability of infection is dependent on their choice of number of partners. From Eq. (4.14) and Eq. (4.15), we have

$$\begin{aligned} V_t^{S^h} - V_t^{I^h} = \left(x_t - \delta x_t^2 + h^{S^h}\right) - \left(\bar{x} - \delta\bar{x}^2 + h^{I^h}\right) + \\ \beta E\left[\left(p_t V_{t+1}^{I^h} + (1-p_t)V_{t+1}^{S^h}\right) - \left(\sigma V_{t+1}^{S^h} + (1-\sigma)V_{t+1}^{I^h}\right)\right]. \end{aligned}$$

This implies that

$$V_t^{S^h} - V_t^{I^h} = \left(x_t - \delta x_t^2\right) - \left(\bar{x} - \delta\bar{x}^2\right) + h + \beta E\left[(1-\sigma-p_t)\left(V_{t+1}^{S^h} - V_{t+1}^{I^h}\right)\right], \tag{4.18}$$

where $h = h^{S^h} - h^{I^h}$. Therefore, making use of Eq. (4.16), Eq. (4.18) becomes

$$V_t^{S^h} - V_t^{I^h} = \left(x_t - \delta x_t^2\right) - \left(\bar{x} - \delta\bar{x}^2\right) + h - \frac{1-\sigma-p_t}{p_{x,t}}(2\delta x_t - 1). \tag{4.19}$$

Let move Eq. (4.19) one step ahead and take E on both sides and multiply through by β so that we have

$$\beta E\left[V_{t+1}^{S^h} - V_{t+1}^{I^h}\right] = \beta E\left[\left(x_{t+1} - \delta x_{t+1}^2\right) - \left(\bar{x} - \delta\bar{x}^2\right) + h - \frac{1-\sigma-p_{t+1}}{p_x, t+1}(2\delta x_{t+1} - 1)\right]. \tag{4.20}$$

Substituting out $V_{t+1}^{S^h}$ and $V_{t+1}^{I^h}$ from Eq. (4.20) by making use of Eq. (4.16), we have the following as the Euler equation:

$$2\delta x_t - 1 = p_{x,t}\beta E\left[(\bar{x} - \delta\bar{x}^2) - (x_{t+1} - \delta x_{t+1}^2) - h + \frac{1-\sigma-p_{t+1}}{p_{x,t+1}}(2\delta x_{t+1} - 1)\right]. \tag{4.21}$$

4.4 Stability Analysis of the Model

We assumed that individuals are forward–looking and concerned about future benefits and risks [5]. The analysis of the model is carried out under two types of economic expectations: the rational expectation under perfect foresight and naive expectations. By perfect foresight, we mean individuals have complete information about the laws of motion governing the disease states and how their risky activities influence the behaviour of others. Therefore, they have complete knowledge of the effect of their risky behaviour on their health status and that of others. On the other hand, by naive expectation we mean individuals expect future risks of infection and benefits to remain at their current level.

We proceed with the analysis by looking at the equilibria of the model.

4.4.1 Equilibria

The system does not have a disease–free steady state (that is a case where there is no syphilis infection among the inmates). Thus, we proceed to finding the endemic steady state. At the endemic steady state, we solve for the time–invariant of system (4.9) and the Euler equation. The following equations in terms of S^h, I^h, R^h, R^{nh} and

x can be obtained:

$$\begin{cases} S^h = \frac{\Lambda\pi_1+\alpha S^{nh}+\gamma R^h}{\omega+\mu+p}, \\ I^h = \frac{\Lambda\pi_3+pS^h+\alpha I^{nh}}{\omega+\mu+\sigma}, \\ R^h = \frac{\sigma I^h}{\omega+\mu+\gamma}, \\ R^{nh} = \frac{\nu I^{nh}}{\omega+\mu}, \\ 2\delta x - 1 = \beta(p_x((\bar{x} - \delta\bar{x}^2) - (x - \delta x^2) - h) + (1 - \sigma - p)(2\delta x - 1)), \end{cases} \tag{4.22}$$

where

$$\begin{cases} p = 1 - \left(1 - \lambda_p \frac{I^h}{N^h}\right)^x, \\ p_x = -\frac{(1-p)}{x}\ln(1-p), \\ N^h = R^h + I^h + S^h. \end{cases}$$

4.4.2 Transition dynamics

To investigate the Transition dynamics of the model, the system is linearized around the endemic steady state using first-order Taylor series approximation. Again, the hat $\wedge$ over the variables denotes the deviation from the endemic steady state. Below is the linearized system:

$$\begin{cases} \hat{S}^h_{t+1} = (1 - \omega - \mu - p)\hat{S}^h_t - S^h\hat{p}_t + \gamma\hat{R}^h_t, \\ \hat{I}^h_{t+1} = p\hat{S}^h_t + S^h\hat{p}_t + (1 - \omega - \mu - \sigma)\hat{I}^h_t, \\ \hat{R}^h_{t+1} = \sigma\hat{I}^h_t + (1 - \omega - \mu - \gamma)\hat{R}^h_t, \\ \hat{R}^{nh}_{t+1} = (1 - \omega - \mu)\hat{R}^{nh}_t. \end{cases} \tag{4.23}$$

The linearized Euler equation is as follows:

$$\begin{aligned} 2\delta p_x \hat{x}_t - (2\delta x - 1)\hat{p}_{x,t} = \beta p_x \left[p_x(2\delta x - 1) + 2\delta(1 - \sigma - p) \right] E\hat{x}_{t+1} \\ - \beta(1 - \sigma - p)(2\delta x - 1) E\hat{p}_{x,t+1} \\ - \beta p_x (2\delta x - 1) E\hat{p}_{t+1}, \end{aligned} \tag{4.24}$$

where

$$\begin{cases} \hat{p}_t = (p_{I^h} + p_{N^h})\hat{I}_t^h + p_x \hat{x}_t + p_{N^h}(\hat{S}_t^h + \hat{R}_t^h), \\ \hat{p}_{x,t} = \left[\frac{1+\ln(1-p)}{x} \right] \hat{p}_t - \frac{p_x}{x} \hat{x}_t, \end{cases} \tag{4.25}$$

and

$$\begin{cases} p_{I^h} = \frac{\partial p}{\partial I^h} = \frac{x\lambda_p}{N^h} \left[1 - \lambda_p \frac{I^h}{N^h} \right]^{x-1}, \\ p_{N^h} = \frac{\partial p}{\partial N^h} = -x\lambda_p \frac{I^h}{N^{h2}} \left[1 - \lambda_p \frac{I^h}{N^h} \right]^{x-1}, \\ p_x = -\frac{1-p}{x} \ln(1-p). \end{cases} \tag{4.26}$$

Stability analysis under perfect foresight

We carry out the analysis of the system under rational expectations by imposing perfect foresight and making use of (4.23)–(4.25). That is setting $Ex_{t+1} = x_{t+1}$. Thus,

we have the EE system in matrix form as follows:

$$\underbrace{\begin{bmatrix} 0 & (1-\omega-\mu-p) & 0 & \gamma & 0 \\ 0 & p & (1-\omega-\mu-\sigma) & 0 & 0 \\ 0 & 0 & \sigma & (1-\omega-\mu-\gamma) & 0 \\ 0 & 0 & 0 & 0 & (1-\omega-\mu) \\ 2\delta p_x & 0 & 0 & 0 & 0 \end{bmatrix}}_{\text{A}} \begin{bmatrix} \hat{x}_t \\ \hat{S}_t^h \\ \hat{I}_t^h \\ \hat{R}_t^h \\ \hat{R}_t^{nh} \end{bmatrix}$$

$$+ \underbrace{\begin{bmatrix} -S^h & 0 \\ S^h & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & -(2\delta x - 1) \end{bmatrix}}_{\text{B}} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} =$$

$$\underbrace{\begin{bmatrix} 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \\ \beta p_x[p_x(2\delta x - 1) + 2\delta(1-\sigma-p)] & 0 & 0 & 0 & 0 \end{bmatrix}}_{\text{C}} \begin{bmatrix} \hat{x}_{t+1} \\ \hat{S}_{t+1}^h \\ \hat{I}_{t+1}^h \\ \hat{R}_{t+1}^h \\ \hat{R}_{t+1}^{nh} \end{bmatrix}$$

$$+ \underbrace{\begin{bmatrix} 0 & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \\ -\beta p_x(2\delta x - 1) & -\beta(1-\sigma-p)(2\delta x - 1) \end{bmatrix}}_{\text{D}} \begin{bmatrix} \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix} \tag{4.27}$$

and

$$\underbrace{\begin{bmatrix} 1 & 0 \\ -\left[\frac{1+\ln(1-p)}{x}\right] & 1 \end{bmatrix}}_{\mathrm{E}} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} = \underbrace{\begin{bmatrix} p_x & p_{N^h} & (p_{I^h}+p_{N^h}) & p_{N^h} & 0 \\ -\frac{p_x}{x} & 0 & 0 & 0 & 0 \end{bmatrix}}_{\mathrm{F}} \begin{bmatrix} \hat{x}_t \\ \hat{S}_t^h \\ \hat{I}_t^h \\ \hat{R}_t^h \\ \hat{R}_t^{nh} \end{bmatrix}. \quad (4.28)$$

Based on Eq.(4.27) and (4.28) we have the following:

$$\hat{Z}_t = J\hat{Z}_{t+1}, \quad (4.29)$$

where

$$J = (A + BE^{-1}F)^{-1}(C + DE^{-1}F),$$

and

$$\begin{cases} \hat{Z}_t = (\hat{x}_t, \hat{S}_t^h, \hat{I}_t^h, \hat{R}_t^h, \hat{R}_t^{nh})^T, \\ \hat{Q}_t = (\hat{p}_t, \hat{p}_{x,t})^T. \end{cases}$$

The stability of the above system is analysed by employing the Blanchard and Kahn condition [15]. The system has one non-predetermined variable ($\hat{x}_t$) and four predetermined variables ($\hat{S}_t^h, \hat{I}_t^h, \hat{R}_t^h, \hat{R}_t^{nh}$). Therefore, if we have exactly four eigenvalues of J outside the unit circle, the system exhibits saddle-path stability. If all the eigenvalues of J are outside the unit circle, we have indeterminate multiple stable paths and explosive paths if there is more than one eigenvalues inside the unit circle.

Stability analysis under naive expectations

Under naive expectations, we set $Ex_{t+1} = x_t$. Making use of Eq. (4.24) and (4.25) we have the linearized Euler equation under naive expectation as

$$\hat{x}_t = \frac{C_2}{C_1}\hat{I}_t^h + \frac{C_3}{C_1}\left(\hat{S}_t^h + \hat{R}_t^h\right), \tag{4.30}$$

where

$$\begin{cases} C_1 = \frac{p_x}{x}(1-\beta(1-\sigma-p))\left[2\delta x - (2\delta x - 1)\ln(1-p)\right], \\ C_2 = (2\delta x - 1)\left[\frac{1+\ln(1-p)}{x}(1-\beta(1-\sigma-p) - \beta p_x\right](p_{I^h} + p_{N^h}), \\ C_3 = (2\delta x - 1)\left[\frac{1+\ln(1-p)}{x}(1-\beta(1-\sigma-p) - \beta p_x\right] p_{N^h}. \end{cases} \tag{4.31}$$

Therefore, we have the linearized EE system as follows:

$$\begin{cases} \hat{S}_{t+1}^h = (1-\omega-\mu-p-S_h\theta_1)\hat{S}_t^h - S^h\theta_2\hat{I}_t^h + (\gamma - S^h\theta_1)\hat{R}_t^h, \\ \hat{I}_{t+1}^h = (p + S^h\theta_1)\hat{S}_t^h + (1-\omega-\mu-\sigma+S^h\theta_2)\hat{I}_t^h + S^h\theta_1\hat{R}_t^h, \\ \hat{R}_{t+1}^h = \sigma\hat{I}_t^h + (1-\omega-\mu-\gamma)\hat{R}_t^h, \\ \hat{R}_{t+1}^{nh} = (1-\omega-\mu)R_t^{nh}, \end{cases} \tag{4.32}$$

where

$$\begin{cases} \theta_1 = p_{N^h} + \frac{p_x C_3}{C_1}, \\ \theta_2 = p_{I^h} + p_{N^h} + \frac{p_x C_2}{C_1}. \end{cases} \tag{4.33}$$

We have Eq. (4.32) in Matrix form as

$$\begin{bmatrix} \hat{S}^h_{t+1} \\ \hat{I}^h_{t+1} \\ \hat{R}^h_{t+1} \\ \hat{R}^{nh}_{t+1} \end{bmatrix} = \underbrace{\begin{bmatrix} 1-\omega-\mu-p-S^h\theta_1 & -S^h\theta_2 & \gamma-S^h\theta_1 & 0 \\ p+S^h\theta_1 & 1-\omega-\mu-\sigma+S^h\theta_2 & S^h\theta_1 & 0 \\ 0 & \sigma & 1-\omega-\mu-\gamma & 0 \\ 0 & 0 & 0 & 1-\omega-\mu \end{bmatrix}}_{\mathrm{Y}} \begin{bmatrix} \hat{S}^h_t \\ \hat{I}^h_t \\ \hat{R}^h_t \\ \hat{R}^{nh}_t \end{bmatrix}. \tag{4.34}$$

The condition for stability for the above system is as follows: If all the eigenvalues for Y are inside the unit circle, we have the system to be stable. The system returns to the endemic steady state after small disturbances. If one of the eigenvalues is outside the unit circle then the system is unstable; the system does not return to the endemic steady state after small perturbation.

4.4.3 Stability analysis of the ME system

In the ME system, individuals do not have control over the number of sexual partners. Therefore, the dynamics of the system is dependent only on the biology of the disease. We fixed the number of sexual partners for individuals at $\bar{x}$. Below is the linearized Matrix for the ME system:

$$\begin{bmatrix} \hat{S}^h_{t+1} \\ \hat{I}^h_{t+1} \\ \hat{R}^h_{t+1} \\ \hat{R}^{nh}_{t+1} \end{bmatrix} = \underbrace{\begin{bmatrix} 1-\omega-\mu-p-S^h p_{N^h} & -S^h(p_{I^h}+p_{N^h}) & \gamma-S^h p_{N^h} & 0 \\ p+S^h p_{N^h} & 1-\omega-\mu-\sigma+S^h(p_{I^h}+p_{N^h}) & S^h p_{N^h} & 0 \\ 0 & \sigma & 1-\omega-\mu-\gamma & 0 \\ 0 & 0 & 0 & 1-\omega-\mu \end{bmatrix}}_{\mathrm{Y}} \begin{bmatrix} \hat{S}^h_t \\ \hat{I}^h_t \\ \hat{R}^h_t \\ \hat{R}^{nh}_t \end{bmatrix}. \tag{4.35}$$

The stability condition for the above system is the same as the one given for system (4.34). Let us proceed to the numerical analysis of the systems.

4.5 Numerical Solution

Due to the complex nature of the system, there is no analytical solution for it. Thus, we carried out the analysis of the system numerically. The simulation carried out is to look at the effect of the parameter combination of the health gap, $(h = h^{s_h} - h^{I_h})$ and the infection parameter, λ_p. The variation in λ_p could be due to a variation in the natural rate of infection λ_a of the disease or the number of sexual acts per partner a. Thus, given a fixed λ_a and an increasing(decreasing) number of sexual acts per partner will cause λ_p to increase(decrease). The converse holds for a fixed a and a varying λ_a. These parameters are the possible policy targets. A high h can be lowered between the inmates by administering treatments to the infected MSM while λ_p can be lowered by educating the inmates about the consequences of their risky behaviour. Perhaps, the introduction of condom usage can be an effective way to reduce this parameter. Below are the parameter values used for the numerical analysis. It must be pointed out that these values are not calibrated to a particular detention center in any jurisdiction.

The value for Λ indicates the fixed rate at which law offenders are recruited into prison. We have 5500 per total population of recruits. The value for β implies that the annual discount rate is 4% whilst the value for σ implies 100% recovery rate of syphilis. γ implies the average duration of immunity against syphilis is 5-years. The value for α indicates that on the average, 4% of non-MSM become MSM from the non-MSM susceptible and infected disease category respectively, ω and μ gives 4% and 2% rate at which inmates are released and the natural mortality rate respectively.

Table 4.1: Fixed Parameter values for the numerical analysis

Parameter	Symbol	Value	Source
Recruitment rate	Λ	5500	Assumed
Discount factor	β	0.96	[6]
Recovery rate	σ	1	[6]
Rate of a R^h becoming susceptible	γ	$0.2yr^{-1}$	[6]
Proportion of a non-MSM becoming a MSM	α	$0.04yr^{-1}$	Assumed
Rate of release from prison	ω	$0.04yr^{-1}$	Assumed
Natural mortality rate	μ	$0.02yr^{-1}$	Assumed
Proportion recruited into S^{nh} categories	π_0	0.6	Assumed
Proportion recruited into S^h categories	π_1	0.2	Assumed
Proportion recruited into I^{nh} categories	π_2	0.1	Assumed
Proportion recruited into I^h categories	π_3	0.1	Assumed
Determinant of maximum contacts	δ	0.05	Assumed

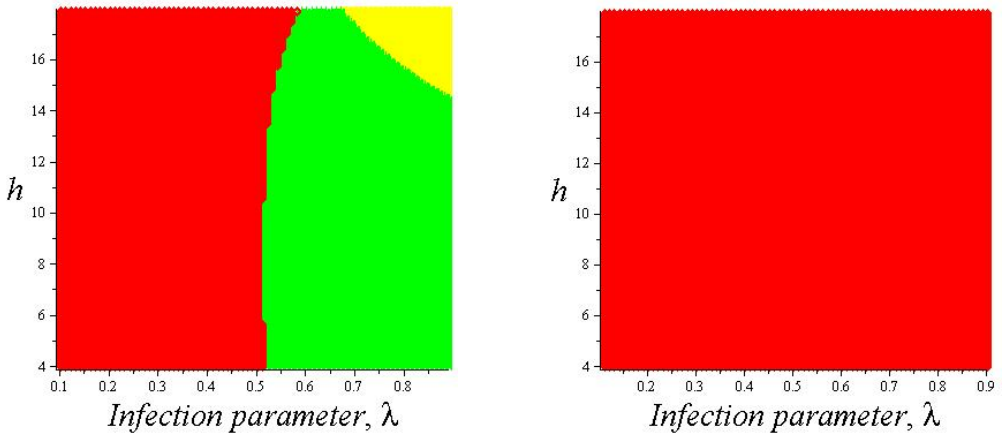
60%, 20%, 10% and 10% for π_0, π_1, π_2, and π_3 respectively are the percentages of recruits that enter the respective disease categories. Finally, the value for δ gives the maximum number of partners to be 10.

4.6 Result and Discussion

Fig. 4.2a shows the dynamic paths for the EE system (4.29) under perfect foresight. The regions indicated by color red, green and yellow denote the region for saddle-path stability, indeterminate paths and explosive paths respectively. The system exhibits saddle-paths stability (red) for low values of λ_p and indeterminate multiple stable paths (green) for high values of λ_p. Also, for very high values of h and λ_p, the system exhibit explosive path (yellow region). In carrying out the simulation, we investigated whether there would be a situation where individuals will be going for the maximum number of partners. As it is shown in the figure, such a case did not happen, thus indicating that rational individuals are going for a number of

partners less than the maximum allowable partners. Thus, policy direction towards the reduction of the infection parameter would lead to a saddle–path stability whilst poor policy direction that would lead to an increase in the infection parameter would cause the system to exhibit indeterminate multiple stable paths and explosive paths.

Fig. 4.2b shows the dynamic paths for the system under naive expectations (4.34). The red region indicates that the system is stable for all the combinations of the parameter range for h and the infection parameter. This means that a small perturbation in the system would bring the system back to the equilibrium paths. The possible policy implication of the result is that, a policy direction at improving the health status of the inmates or reducing the infection parameter of syphilis will not affect the stability of the system. In other words, given the parameter range, health policies have no potential of putting the system on unstable paths if individuals formulate expectations in a naive manner. It must also be noted that, individuals are going for the number of partners less than the possible maximum number.

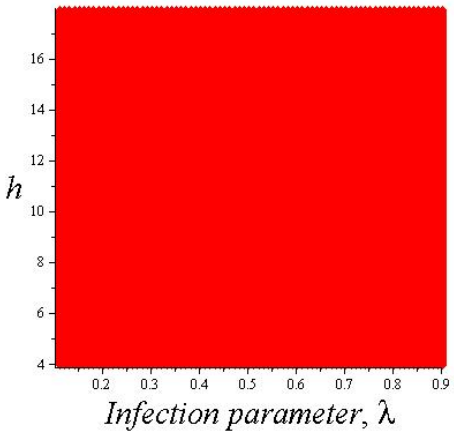


(a) Perfect Foresight (b) Naive Expectations

Figure 4.2: Dynamic paths for the EE system

Fig. 4.3a shows the stability region for the ME system (4.35). We obtained this by setting a fixed value for the number of partners x. We set x to be equal to the maximum number of contacts (that is, $x = \frac{1}{2\delta}$). In this case, we have $x = 10$. The

simulation shows stability of the system for all the parameter combinations of h and the infection parameter. The ME system is independent of h.



(a) Number of contacts $\bar{x} = 10$

Figure 4.3: The dynamic paths for the ME system

4.7 Discussions on the Stability Region

We compared the dynamics of the ME and EE systems ((4.35) and (4.29) and (4.34)) in the stability region by making use of the values for the parameters in table 4.1. We chose the value for the infection parameter and the health gap h that gave the stability of the system after a small perturbation. We assumed that there is a one time exogenous increase in the number of MSM infected with syphilis by 5%. Below are the baseline parameters and the corresponding endemic steady state values:

Table 4.2: Parameter values

Parameters	a	λ_a	λ_p
Values	$20yr^{-1}$	0.0448	0.6002

Table 4.3: Endemic steady states for EE when $h = 12$ and $\delta = 0.05$

Sh	Ih	Rh	x
16545.6	6868.9	26418.8	6.02469

Table 4.4: Endemic steady states for EE when $h = 6$ and $\delta = 0.05$

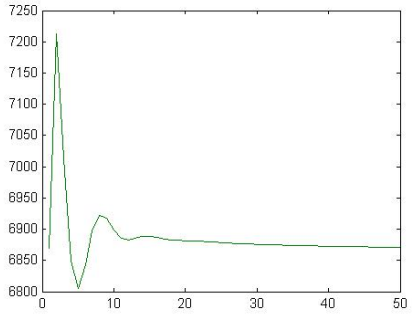
Sh	Ih	Rh	x
13435.3	7510.7	28887.3	8.42558

Figs. 4.4 and 4.5 show the graph for $h = 12$ and $h = 6$ respectively. The respective Figures show that an initial increase in disease prevalence [1] coupled with a high value of h causes susceptible individuals to reduce the number of partners. The reason is that, with the above condition there is a high risk of one becoming infected with the disease. Due to this, the endemic steady state is characterized with a low prevalence rate and number of sexual partners. Specifically, considering our values for h in our simulation, we have $h = 6$ giving the number of partners and its associated disease prevalence higher than that of $h = 12$. Thus, policy directed at reducing the health gap will, in the long run, cause the disease to shoot up as rational individuals, knowing that the health gap has decreased (implying treatment for the infected population) and thereby lowering the risk of infection, will increase their number of sexual partners. This result shows some of the reasons why it will be very difficult to eradicate STDs in a population. The behavioural factor is significant in understanding disease dynamics.

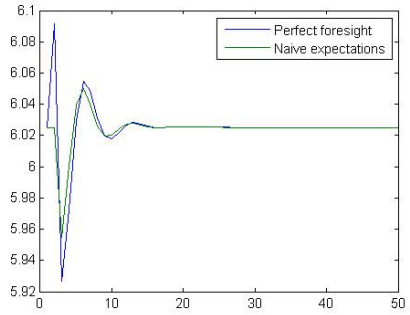
The simulation also shows that, as the health gap increases, the system gravitates toward the equilibrium paths quickly. This is shown by comparing Figs. 4.5 and 4.4. Thus, if the system is subjected to a small perturbation it reaches its equilibrium quickly for high values of h.

Furthermore, we simulated for $\delta = 0.05$ and $\delta = 0.025$, coupled with $h = 6$.

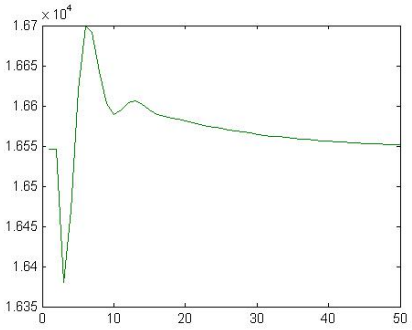
[1] Prevalence is the percentage or proportion of the inmates infected with syphilis at a given period.



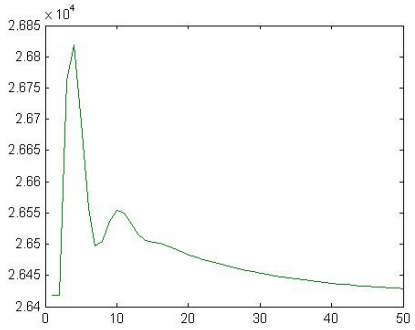
(a) Number of infected MSM



(b) Number of sexual partners



(c) Number of susceptible MSM



(d) Number of recovered MSM

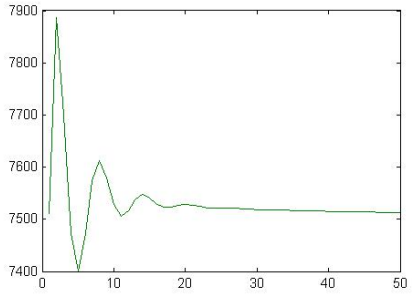
Figure 4.4: The graph for the EE system when $h = 12$ and $\delta = 0.05$

In the simulation result, it is noticed that, in the long run, MSM inmates have a number of sexual partners for $\delta = 0.025$ more than when $\delta = 0.05$. This is exhibited by Figs. 4.5 and 4.6. As pointed out in chapter 3 the role δ plays, the low value for δ implies high level of utility. Thus, MSM inmates have the incentive to increase their number of sexual partners even though this decision also comes with its side effect. In that, as a result of the high level of partners, the prevalence of the disease will also increase. Which is why the simulation result gives a high steady state value for the infective class for $\delta = 0.025$ as compared to $\delta = 0.05$. This is shown in Tabs. 4.5 and 4.4 respectively. This shows that incentives that can influence the utilities of MSM inmates will in the long run influence the prevalence of the disease in prisons.

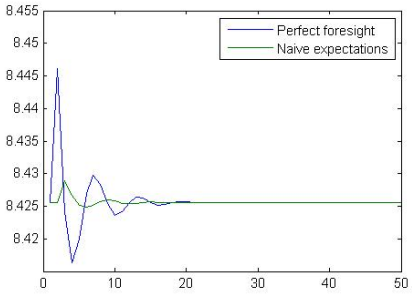
For the comparison of the dynamics of the EE and the ME systems consider

Table 4.5: Endemic steady states for EE when $h = 6$ and $\delta = 0.025$

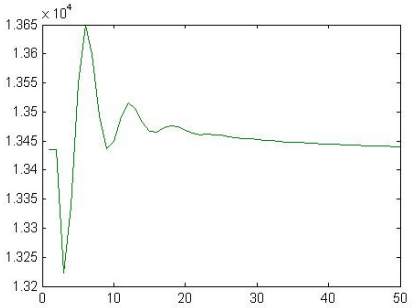
Sh	Ih	Rh	x
9512.6	8320.15	32000.6	19.1214



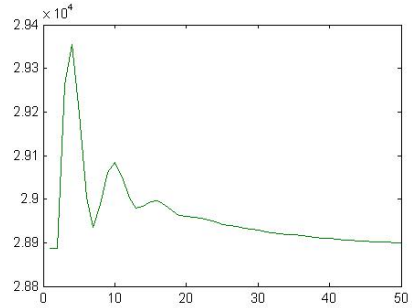
(a) Number of infected MSM



(b) Number of sexual partners



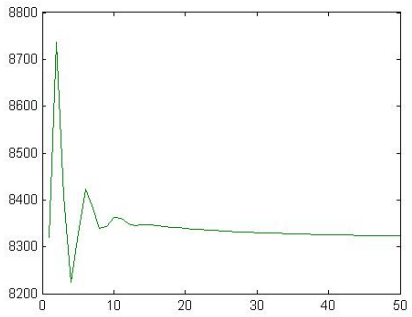
(c) Number of susceptible MSM



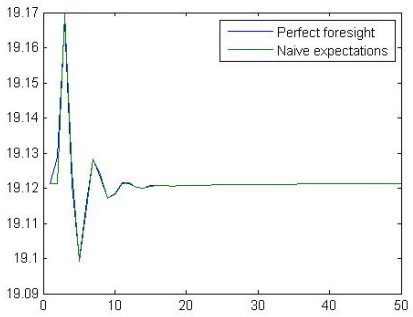
(d) Number of recovered MSM

Figure 4.5: The graph for the EE system when $h = 6$ and $\delta = 0.05$

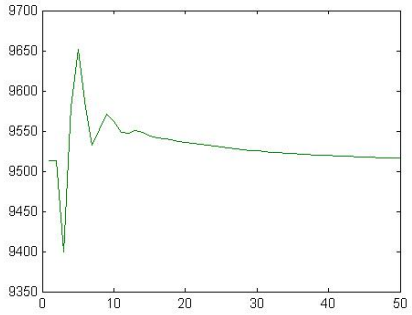
Figs. 4.4 and 4.7. You will notice that the EE system gravitates towards equilibrium quickly as compared to the ME system. This shows that if the number of partners is fixed (implying that the system is dependent only on the biology dynamics), a small perturbation of the system will take a longer period before the system comes back to the steady state. On the other hand, if MSM can freely choose the number of partners they desire, the system reaches stability quickly. This is due to the fact that an initial increase in disease prevalence causes rational susceptible MSM to reduce their number of partners since the associated probability of being infected with the disease is high at that state. This action imposes downward pressure on the rising prevalence of the disease among the inmates. As the newly infected inmates subject themselves



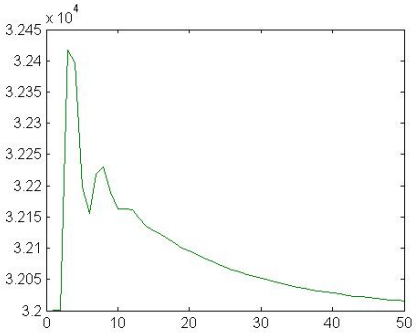
(a) Number of infected MSM



(b) Number of sexual partners



(c) Number of susceptible MSM



(d) Number of recovered MSM

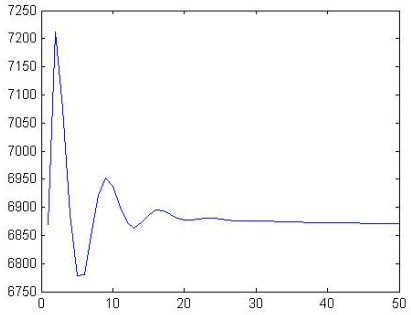
Figure 4.6: The graph for the EE system when $h = 6$ and $\delta = 0.025$

to treatment, they migrate to the recovered category, thus causing the number of infected MSM to decrease. As this happens the risk of infection reduces. This will cause a rational susceptible individual to increase his number of partners. This action will then cause the number of infections to shoot up. This cycle will continue till the system reaches equilibrium[2]. This behaviour puts the system at equilibrium quickly as compared to when the system is solely dependent on the biological dynamics of the disease.

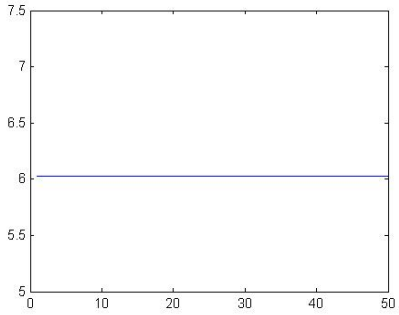
Table 4.6: Endemic steady states for ME when $h = 12$ and $\delta = 0.05$

Sh	Ih	Rh	x
16545.6	6868.9	26418.8	6.02469

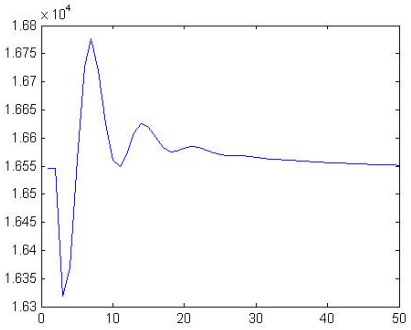
[2]In [5] it is shown that syphilis cycles are smaller and less persistent when there is an interplay between human responses and biological dynamics.



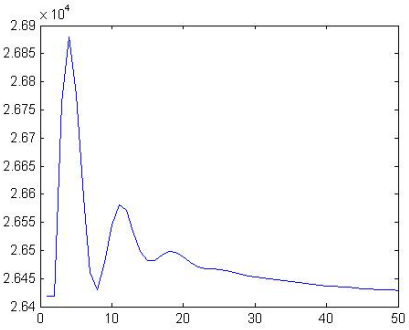
(a) Number of infected MSM



(b) Number of sexual partners



(c) Number of susceptible MSM



(d) Number of recovered MSM

Figure 4.7: The graph for the ME system when $h = 12$ and $\delta = 0.05$

Chapter 5

Conclusion and Future Work

As the emergence of infectious diseases has become a thorn in the flesh of humanity, it is imperative to understand the mechanisms involved in the transmission of these diseases so that health policies targeted at controlling their spread are effective. Classical mathematical epidemiological models provide a fair framework to achieving this purpose [18]. However, it has the limitation of not explicitly modelling the behavioural influence of individuals on the spread of the diseases. Economic Epidemiology aims to fill this gap since disease treatment and prevention depends heavily on the behaviour of individuals [24].

This thesis studied a modified version of the EE models that was recently introduced in [7] within an optimization framework. As a new space variable, the maximum number of contacts is introduced. In addition, we extended the previous model by considering the case of dynamic population (different birth and death rates). Our assumption in particular, is practical at the beginning of the spread of a disease since the maximum number of contacts can be controlled by isolation or limiting contacts.

Furthermore, an EE model on syphilis infectivity among MSM in detention centres was proposed and an investigation on the stability properties was carried out. We employed numerical simulation to get an insight into the various types of dynamic paths of the system; the saddle-paths stability, indeterminate paths stability and explosive paths.

We investigated the stability properties of the EE models by assuming that individual agents have perfect understanding of the system and thus know completely the disease dynamics and the risk and benefits faced by other individuals in the system. Naive expectations is also assumed, where individuals expect future benefit and risk to remain at their current level.

This chapter summarizes the contribution of the thesis to this area. Some directions and future work are proposed.

5.1 The Modified EE Model Studied by Aadland et al.

5.1.1 Conclusion

We investigated the dynamic paths of the EE systems when the maximum number of contacts is dependent on a parameter (δ). Thus, variation in this parameter affected the dynamics of the system. We made use of a quadratic utility function in formulating the model. We further assumed that birth and death rates were not necessarily equal. We demonstrated that

- If the maximum contacts are dependent on a parameter, this parameter has the potential to drive the dynamics of the system.
- The birth and death rates do not have significant effects on the system with exception of some extreme cases where we have high levels of the health gap and infection parameter.
- Public policy has the potential to affect the stability(dynamic paths) of the system.

5.1.2 Future work

The simulation carried out in the work focuses on abstract description of the model. It will be interesting if future research could be tailored to applying real world data on a specific disease to the model so as to ascertain precise policy recommendations.

The model studied assumed that every disease category has equal death rate and there is no death related disease, thus oversimplifying the model. Future research could be look at a more general model where these parameters are considered.

5.2 EE Model on Syphilis Infectivity among MSM Inmates

5.2.1 Conclusion

In this thesis, we have developed a model that enabled us to investigate the stability properties of an Economic Epidemiology(EE) disease model for syphilis infectivity among male inmates. The investigation was carried out under perfect foresight and naive expectations. We have shown that

- Under perfect foresight, if individual inmates behave in a self–centred manner, their behaviours have the tendency of affecting the stability of the system. This result shows that public health policy direction contributes to the dynamic properties of the system.
- If we assume a fixed number of partners for individuals (that is if the system is independent of the number of partners), policy direction has no effect on the stability properties of the system.
- The health gap has effect on the prevalence of syphilis among MSM inmates.
- The prevalence of the disease in the long run is influenced by incentives that drive the utility of the MSM inmates.
- Behavioural responses affect the duration by which the system converges back to its equilibrium position when subjected to a small perturbation. Therefore, it is imperative for policy makers to formulate policies that take into consideration the behavioural factor of MSM inmates.

5.2.2 Future work

Since this study did not consider a particular detention centre, future research should be tailored to making use of data from a particular detention center, where data could be collected on the key parameters so as to provide a precise policy recommendation.

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Appendix A

Simulation Code for Stability Analysis

A.1 Simulation Code for Stability Analysis for SIR EE system

We employed Maple in studying the stability of the system by solving for the endemic steady state values for the variables $x, r, s,$ and i respectively. We then evaluated the corresponding matrices gotten from the linearized system at the endemic steady state. After this we found the eigenvalues of the matrices at these values and then categorized the dynamic paths of the system by employing the stability conditions outlined in chapter 3. The idea is to investigate the effects of the health gap h and the infection parameter α on the stability of the system when subjected to variations. The final step is to plot the point (α, h). For illustration purposes the Maple code below gives the dynamic paths or stability properties for the SIR EE

system:

$restart$:

General Inputs:

$beta := .96 : mu := 0.5e-1 : omega := 0.5e-1 :$

$delta := 0.5e-1 : nu := 1 : g := 0 : phi := 0 :$

Specific Inputs:

$hx := 0.02 : hp := 0.01 : np := 70 : nh := 600 :$

$lp0 := 0.1 : h0 := 5 : NHP := np * nh :$

$XL := array(0..np, 0..nh) :$

$IL := array(0..np, 0..nh) :$

$RL := array(0..np, 0..nh) :$

$INDETERMINATE := array(0..NHP);$

$SADDLE := array(0..NHP);$

$EXPLOSIVE := array(0..NHP);$

$XLM := array(0..np, 0..nh) :$

$ILM := array(0..np, 0..nh) :$

$RLM := array(0..np, 0..nh) :$

$alpha := lp0 : Xb := 1/(2 * delta) : A := 1/(1 + omega - mu) :$

$h := h0 :$

Initial endemic steady state values

$p1 := 1 - (1 - alpha * i1)^x 1 :$

$px := -(1 - p1) * ln(1 - p1)/x1 :$

$psi := x1 - delta * x1^2 - Xb + delta * Xb^2 + h :$

$tor := (2 * delta * x1 - 1) * ((1 - g)/beta - (1 - nu - g) * (1 - p) - nu * g)/px + (1 -$

$nu - g) * (psi - h) + h * (1 - g) :$

$eqn1 := s1 = A * (omega + g * r1)/(1 - A * (1 - p1 - mu)) :$

$eqn2 := i1 - A * s1 * p1/(1 - A * (1 - nu - mu)) = 0 :$

$eqn3 := r1 = A * nu * i1/(1 - A * (1 - mu - g)) :$

$eqnforx := beta * (px * (phi * beta * tor - psi) + (1 - nu - p1) * (2 * delta * x1 - 1))/(2 *$

$delta) + 1/(2 * delta) :$

$eqn4 := x1 - eqnforx = 0 :$

$steadystates := fsolve(eqn1, eqn2, eqn3, eqn4, i1 = .1, r1 = .8, s1 = .1, x1 = 19) :$

$i := eval(i1, steadystates) : x := eval(x1, steadystates) :$

$s := eval(s1, steadystates) : r := eval(r1, steadystates) :$

Eigenvalues calculation and stability analysis

$with(linalg) :$

$with(plots) :$

$printlevel := 6 :$

$KI := 0; KE := 0; KS := 0; KM := 0; h := h0; KN := 0;$

$for \quad jp \quad from \quad 0 \quad to \quad np - 1 \quad do$

$alpha := lp0 + jp * hp;$

$if \quad (h0 < h) then \quad hsign := -1; \quad else \quad hsign := 1; end \quad if;$

$h0 := h;$

$for \quad jh \quad from \quad 0 \quad to \quad nh - 1 \quad do$

$h := h0 + jh * hx * hsign;$

$p1 := 1 - (1 - alpha * i1)^x 1 :$

$px := -(1 - p1) * ln(1 - p1)/x1 :$

$psi := x1 - delta * x1^2 - Xb + delta * Xb^2 + h :$

$tor := (2 * delta * x1 - 1) * ((1 - g)/beta - (1 - nu - g) * (1 - p) - nu * g)/px + (1 -$

$nu - g) * (psi - h) + h * (1 - g) :$

$eqn1 := s1 = A * (omega + g * r1)/(1 - A * (1 - p1 - mu)) :$

$eqn2 := i1 - A * s1 * p1/(1 - A * (1 - nu - mu)) = 0 :$

$eqn3 := r1 = A * nu * i1/(1 - A * (1 - mu - g)) :$

$eqnforx := beta * (px * (phi * beta * tor - psi) + (1 - nu - p1) * (2 * delta * x1 - 1))/(2 *$

$delta) + 1/(2 * delta)$:

$eqn4 := x1 - eqnforx = 0$:

$steadystates := fsolve(eqn1, eqn2, eqn3, eqn4, i1 = 0.1, r1 = 0.8, s1 = 0.1, x1 = 5)$:

$i := eval(i1, steadystates); x := eval(x1, steadystates);$

$s := eval(s1, steadystates); r := eval(r1, steadystates);$

$if \quad (x < Xb \quad and \quad i > 0 \quad and \quad s > 0 \quad and \quad r > 0 \quad and \quad x > 0) \quad then$

$p := 1 - (1 - alpha * i)^x$:

$px := -(1 - p) * ln(1 - p)/x$:

$pin := x * alpha * (1 - alpha * i)^(x - 1)$:

$M1 :=<< 0|A * (1 - nu - mu - p)| - A * p >, < 0|A * nu|A * (1 - g - mu) >, <$

$2 * delta * px|0|0 >>$:

$M2 :=<< A * s|0 >, < 0|0 >, < 0| - (2 * delta * x - 1) >>$:

$M3 :=<< 0|1|0 >, < 0|0|1 >, < beta * px * (px * (2 * delta * x - 1) + 2 * delta * (1 -$

$nu - p))|0|0 >>$:

$M4 :=<< 0|0 >, < 0|0 >, < -beta * px * (2 * delta * x - 1)| - beta * (1 - nu - p) * (2 *$

$delta * x - 1) >>$:

$M5 :=<< 1|0 >, < -(1 + ln(1 - p))/x|1 >>$:

$M6 :=<< px|pin|0 >, < -px/x|0|0 >>$:

$J1 := inverse(M1 + M2.inverse(M5).M6)$:

$J2 := (M3 + M4.inverse(M5).M6)$:

$J := J1.J2$:

$lambda := eigenvalues(J)$:

$EIGEN[jp, jh] := lambda;$

$XL[jp, jh] := lambda[1]; IL[jp, jh] := lambda[2]; RL[jp, jh] := lambda[3];$

$XLM[jp, jh] := abs(lambda[1]); ILM[jp, jh] := abs(lambda[2]); RLM[jp, jh] := abs(lambda[3]);$

Stability conditions

$if \quad ((XLM[jp, jh] > 1) \quad and \quad (ILM[jp, jh] > 1) \quad and \quad (RLM[jp, jh] > 1))$

$then \quad KI := KI + 1; \quad INDETERMINATE[KI] := [alpha, h];$

$elif \quad (((XLM[jp, jh] < 1) \quad and \quad ((ILM[jp, jh] > 1) \quad and$

$(RLM[jp, jh] > 1))) \quad or((ILM[jp, jh] < 1) \quad and$

$((XLM[jp, jh] > 1) \quad and \quad (RLM[jp, jh] > 1))) \quad or \quad ((RLM[jp, jh] < 1)$

$and \quad ((ILM[jp, jh] > 1) \quad and$

$(XLM[jp, jh] > 1)))) \quad then \quad KS := KS + 1; \quad SADDLE[KS] := [alpha, h];$

$else \quad KE := KE + 1; \quad EXPLOSIVE[KE] := [alpha, h];$

$end \quad if;$

$elif \quad (i < 0 \quad or \quad s < 0 \quad or \quad r < 0 \quad or \quad x < 0)$

$then \quad KN := KN + 1; \quad NEGATIVECASE[KN] := [alpha, h] :$

$else \quad KM := KM + 1; \quad MAXCONTACT[KM] := [alpha, h];$

$end \quad if;$

$end \quad do :$

$end \quad do :$

$ptssaddle := [seq(SADDLE[j1], j1 = 1..KS)] :$

$Plotsaddle := pointplot(ptssaddle, thickness = 5, color = red,$

$symbol = diamond, symbolsize = 15, gridlines = true) :$

$ptsintermediate := [seq(INDETERMINATE[j1], j1 = 1..KI)] :$

$Plotindeterminate := pointplot(ptsintermediate, thickness = 5,$

$color = green, symbol = asterisk, symbolsize = 15, gridlines = true) :$

$ptsexplosive := [seq(EXPLOSIVE[j1], j1 = 1..KE)] :$

$Plotexplosive := pointplot(ptsexplosive, thickness = 5,$

$color = yellow, symbol = circle, symbolsize = 15, gridlines = true) :$

$ptsmaxcontact := [seq(MAXCONTACT[j1], j1 = 1..KM)] :$

$Plotmaxcontact := pointplot(ptsmaxcontact, thickness = 5, color = black, symbol =$

$circle,$

$symbolsize = 15, gridlines = true) :$

$ptsnegative := [seq(NEGATIVECASE[j1], j1 = 1..KN)] :$

$Plotnegative := pointplot(ptsnegative, thickness = 5, color = blue,$

$symbol = diamond, symbolsize = 15, gridlines = true) :$

$display(Plotsaddle, Plotindeterminate, Plotexplosive, Plotmaxcontact, Plotnegative);$

Appendix B

Numerical Solution with Dynare

In investigating the effects of small perturbation on the systems, we employed Dynare. Dynare is a software that provides the platform for solving or simulating economic models such as Dynamic Stochastic General Equilibrium models and Overlapping Generation Models (OGM), where economic agents such as productive firms, consumers, governments, monetary authorities and others are assumed to formulate expectations about future outcome consistent with the model. In short, the software solves models that are based on the rational expectations hypothesis [9]. It can also solve both linear and non-linear deterministic models.

The software employs applied mathematics and computer science concepts such as multivariate nonlinear solving and optimization, matrix factorizations, local functional approximation, MCMC techniques for Bayesian estimation, graph algorithms, optimal control, Kalman filters and smoothers and others [9].

The program is made up of five blocks:

- The preamble block where the variables in the models are declared. The distinction is made between predetermined and non-predetermined and exogenous variables. The parameters are also declared, and the parameters in the model are also declared.

- The model block: Here the model is specified. The dynamic equations linking the variables and the parameters are specified.

- The steady state or initial values block: The initial values of the variables are specified in this block.

- Shocks: If one wants to study the impacts of a temporary shock to the model, it is specified in this block.

- Computation block: Here the number of simulation periods for the model is specified.

Due to the scope of this thesis, we would only discuss briefly, the basic idea behind the software and then provide the codes for the models studied in this thesis.

B.1 Deterministic Models and Dynare

Suppose we have the following non-linear equation to solve:

$$f(y_{t+1}, y_t, e_t) = 0, \tag{B.1}$$

where the function f is defined as follows:

$$f : \mathbb{R}^{2n} \times \mathbb{R}^n \to \mathbb{R}^n.$$

$y_t \in \mathbb{R}^n$ and $e_t \in \mathbb{R}^{n_e}$ are vectors of endogenous and exogenous variables respectively. Furthermore, let $y_t = \begin{bmatrix} C_t \\ X_t \end{bmatrix}$, where C_t is a vector of $(n \times 1)$ predetermined variables at time t. Therefore, we can assume that the initial value for C is given. We have $C_{t=0} = C_0$. X_t is a vector of $(m \times 1)$ non-predetermined variables at time t. Let suppose that the terminal value for X is known. Then we have the following unknown variables to solve for: $C_0, C_1, ..., C_{T+1}; X_0, ..., X_T$. Where $T+1$ is the terminal time. This means that we have a total number of $(T+1) \times (n+m)$ non-linear equations to worry about. We can employ the Newton-type root finding method to solve for these variables [38]. If one is dealing with an infinite horizon problem, then we have to write the terminal condition as a transversality conditions[1].

Based on the above information, let specify an equation of the following form:

$$F(C_t, X_t, C_{t+1}, X_{t+1}, e_t) = 0, t = 0, 1, 2, ..., T. \tag{B.2}$$

T can be finite or infinite. The next step is to solve for the stable solution by employing the shooting algorithm [38]. This is done by specifying a time $T+1$ which is indicative that the system is at the steady state. In this case we have

$$F(\bar{C}, \bar{X}, \bar{C}, \bar{X}, \bar{e}) = 0. \tag{B.3}$$

We then set $X_{T+1} = \bar{X}$ and then make use of the shooting algorithm [38]. The last step is by adjusting T till (C_T, X_T, C_{T+1}) is very close to the steady state values $(\bar{k}, \bar{y}, \bar{k})$. Here, X_{T+1} is set at $\bar{X}$. For the algorithm to find a stable solution, the Blanchard and Kahn condition ought to be satisfied.

[1]The requirement for the tranversality condition is that the present value of the state variables should converge to zero as time approaches infinite [12].

In a more general form, Eq. (B.1) can take the following form:

$$E_t F(y_{t+1}^+, y_t, y_{t-1}^-, u_t) = 0, \tag{B.4}$$

where y_t, and y_{t-1}^- are vectors of endogenous variables and a subset of predetermined variables. y_{t+1} is a subset of variables with a lead and u_t is a vector of exogenous variables. Dynare can solve a model of the above form.

B.2 Dynare Code for Simulating the Model on Syphilis

We assumed a 5% exogenous increase in the infected MSM population. This is denoted by z. Below are the codes for the EE and ME systems respectively.

B.2.1 The EE system

The following Dynare code gives the simulation results for the EE system under both perfect foresight and naive expectations:

Declare variables

```
var     Snh     Sh     Inh     Ih     Rnh     Rh
      x     N     Snh1     Sh1     Inh1     Ih1     Rnh1     Rh1 x1     N1;
```

Declare predetermined variables

```
predetermined_variables     Snh     Sh     Inh     Ih     Rnh     Rh     N
          Snh1     Sh1     Inh1     Ih1     Rnh1     Rh1     N1;
```

Declare exogenous variables

varexo z;

Declare parameters

parameters quad Lam cbeta nu sig gamma alp ome mu pi0 pi1

pi2 pi3 del lam xbar h a lama;

Lam=5500;

cbeta=0.96;

nu=1;

sig=1;

gamma=0.2;

alp=0.04;

ome=0.04;

mu=0.02;

pi0=0.6;

pi1=0.2;

pi2=0.1;

pi3=0.1;

del=0.05;

a=20;

lama=0.0448;

lam=1-(1-lama) ∧ (a);

xbar=1/(2*del);

h=12;

model;

The model for the EE system under naive expectations

N(+1)=Lam+(1-ome-mu)*N;

Snh(+1)=Lam*pi0+(1-ome-mu-alp)*Snh;

Sh(+1)=Lam*pi1+(1-ome-mu-(

1-(1-lam*Ih/(Sh+Rh+Ih))∧(x)))*Sh+alp*Snh+gamma*Rh;

Inh(+1)=Lam*pi2+(1-ome-mu-nu-alp)*Inh;

Ih(+1)=Lam*pi3+(1

-(1-lam*Ih/(Sh+Rh+Ih))∧(x))*Sh+(1+z-ome-mu-sig)*Ih+alp*Inh;

Rnh(+1)=nu*Inh+(1-ome-mu)*Rnh;

Rh(+1)=sig*Ih+(1-ome-mu-gamma)*Rh;

2*del*x-1=(-(1-(1-(1-lam*Ih/(Sh+Rh+Ih))

∧(x)))*(ln(1-(1-(1-lam*Ih/(Sh+Rh+Ih))∧(x))))/x)*

cbeta*((xbar-del*(xbar)∧ 2)-(x-del*(x)∧ 2)-h+

(1-sig-(1-(1-lam*Ih/(Sh+Rh+Ih))∧ (x)))*(2*del*x-1)/(-(1-(1-(1-lam*Ih/(Sh+Rh

+ Ih))∧(x)))*(ln(1-(1-(1-lam*Ih/(Sh+Rh+Ih))∧ (x))))/x));

The model for the EE system under perfect foresight

N1(+1)=Lam+(1-ome-mu)*N1;

Snh1(+1)=Lam*pi0+(1-ome-mu-alp)*Snh1;

Sh1(+1)=Lam*pi1+(1-ome-mu-(

1-(1-lam*Ih1/(Sh1+Rh1+Ih1))∧(x)))*Sh1+alp*Snh1+gamma*Rh1;

Inh1(+1)=Lam*pi2+(1-ome-mu-nu-alp)*Inh1;

Ih1(+1)=Lam*pi3+(1

-(1-lam*Ih1/(Sh1+Rh1+Ih1))∧(x))*Sh1+(1+z-ome-mu-sig)*Ih1+alp*Inh1;

Rnh1(+1)=nu*Inh1+(1-ome-mu)*Rnh1;

Rh1(+1)=sig*Ih1+(1-ome-mu-gamma)*Rh1;

2*del*x1-1=(-(1-(1-(1-lam*Ih1/(Sh1+Rh1+Ih1))∧(x1)))*(ln(1-(1-(1-

lam*Ih1/(Sh1+Rh1+Ih1))∧(x1))))/ x1)*cbeta*((xbar-del*(xbar)∧

2)-(x1(+1)-del*(x1(+1))∧ 2)-h+

(1-sig-(1-(1-lam*Ih1(+1)/(Sh1(+1)+Rh1(+1)+Ih1(+1)))∧ (x1(+1))))*

```
      (2*del*x1(+1)-1)/(-(1-(1-(1-lam*Ih1(+1)/(Sh1(+1)+Rh1(+1)+Ih1(+1)))^
(x1(+1))))*
      (ln(1-(1-(1-lam*Ih1(+1)/(Sh1(+1)+Rh1(+1)+Ih1(+1)))^
(x1(+1)))))/x1(+1)));
end;
initval;
x=9.19958;
N=91666.7;
Snh=33000;
Sh=27090.8;
Inh=500;
Ih=4692.91;
Rnh=8333.33;
Rh=18049.7;
x1=9.19958;
N1=91666.7;
Snh1=33000;
Sh1=27090.8;
Inh1=500;
Ih1=4692.91;
Rnh1=8333.33;
Rh1=18049.7;
end;
steady;
check;
shocks;
var z;
```

```
periods 1;
values 0.05;
end;
simul(periods=60);
```

B.2.2 ME system

Declare variables

```
var Snh Sh Inh Ih Rnh Rh N ;
```

Declare predetermined variables

```
predetermined_variables Snh Sh Inh Ih Rnh Rh N ;
```

Declare exogeneous variables

```
varexo z;
```

Declare parameters

```
parameters Lam cbeta nu sig gamma alp
        ome mu pi0 pi1 pi2 pi3 del lam xbar x;
Lam=5500;
cbeta=0.96;
nu=1;
sig=1;
gamma=0.2;
alp=0.04;
ome=0.04;
mu=0.02;
pi0=0.6;
pi1=0.2;
```

pi2=0.1;

pi3=0.1;

del=0.05;

a=20;

lama=0.0448;

lam=1-(1-lama)∧(a);

x=6.02469;

model;

N(+1)=Lam+(1-ome-mu)*N;

Snh(+1)=Lam*pi0+(1-ome-mu-alp)*Snh;

Sh(+1)=Lam*pi1+(1-ome-mu-(

1-(1-lam*Ih/(Sh+Rh+Ih))∧(x)))*Sh+alp*Snh+gamma*Rh;

Inh(+1)=Lam*pi2+(1-ome-mu-nu-alp)*Inh;

Ih(+1)=Lam*pi3+(1 -(1-lam*Ih/(Sh+Rh+Ih))∧

(x))*Sh+(1+z-ome-mu-sig)*Ih+alp*Inh;

Rnh(+1)=nu*Inh+(1-ome-mu)*Rnh;

Rh(+1)=sig*Ih+(1-ome-mu-gamma)*Rh;

end;

Declare initial values for the variables

initval;

N=91666.6;

Snh=22000;

Sh=20753;

Inh=750;

Ih=2385.953729;

Rnh=12500;

Rh=28304;

```
end;
steady;
check;
shocks;
var z;
periods 1;
values 0.05;
end;
simul(periods=50);
```

Appendix C

Abbreviations and Definitions

AIDS Acquired Immune Deficiency Syndrome.

EE Economic Epidemiology.

ME Mathematical Epidemiology.

MSM Men who have Sex with men (can be interpreted as Man who have sex with men).

SI Susceptible Infected.

SIR Susceptible Infected Recovered.

SIS Susceptible Infected Susceptible.

SIRS Susceptible Infected Recovered Susceptible.

STDs Sexually Transmitted Diseases.

Birth rate The number of live births per year for every thousand of population.

Disease prevalence The percentage of of individuals infected by the disease at a given period.

Death rate The number of deaths per year for every thousand of population.

Health gap The difference between the health status of susceptible and infected individuals.

Probability of infection The probability of individuals becoming infected by an infectious disease after coming in contact with infected person(s).

Marginal damage cost The damage incurred in terms of health as a result of an individual going for an additional contact.

Marginal utility It is the additional satisfaction or utility an individual get as a result of making one more contact.

Naive expectations Individuals are assumed not to have perfect knowledge about the system.

Perfect foresight Individuals are assumed to have perfect knowledge of the system.

Recovery rate The rate at which infected individuals become recovered.

Syphilis A STD caused by a bacterium called treponema pallidum.

Utility function The function that measures the level of utility associated with a specific choice by a decision maker.

Value function It measures the best possible objective written as a function of the state of the system.

$\boldsymbol{\delta}$ Determinant of maximum number of contacts.

$\boldsymbol{\nu}$ Recovery rate.

$\boldsymbol{\gamma}$ Rate of recovered individuals becoming susceptible.

$\boldsymbol{\lambda_p}$ The natural rate of becoming infected by an infected contact.

$\boldsymbol{\lambda_a}$ The natural rate of becoming infected by a single interaction.

$\boldsymbol{a}$ Fixed number of interaction.