Network Modelling for Sexually Transmitted Diseases

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Abstract

The aim of this thesis is to propose suitable mathematical models for the analysis of sexually transmitted disease epidemics. We are interested in a closed population, where infections are only transmitted through heterosexual contacts. The population is hence divided into two groups: male and female. Individuals are classified according to gender, relationship and disease status. Both stochastic and deterministic SIS models are employed. The stochastic models are formulated in terms of a Markov process with a finite state space. Two main models are constructed and quantities of interest such as the basic reproduction number and endemic level of the sexually transmitted disease (STD) are obtained.

The first model is formulated to describe dynamics of STDs, where the sexual behaviour is considered "faithful". By being faithful, we mean individuals are monogamous, and there are no casual sexual contacts (one-night stands). The early stages of the epidemic are approximated by a 2-type branching process. This allows us to compute the following quantities of interest, the threshold parameter (R_0) and the probability of extinction. In order to study the endemic level, it is helpful to use the deterministic (ODE) approximation of the stochastic SIS epidemic. The behaviour about the endemic equilibrium is studied using an Ornstein-Uhlenbeck process. Stochastic simulations are utilised to obtain the mean time to extinction.

The second model is an extension of the first model, where casual sexual contacts (one - night stands) are included in the model. The model is again a Markov process but its analysis is more involved. A key difference is now a 5 type branch-

ing process is used to approximate the initial stages of the epidemic, to determine the threshold parameter (R_0) and the probability of extinction. Other quantities of interest are studied through similar approaches.

Medication use is studied as a control measure in this thesis. We introduce a new parameter (v) governing the medication use into both models. Throughout we study the effect of the control strategies on the key quantities of interest highlighted above.

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Last but not least, I would like to thank my family for giving me such unconditional love, and that is the greatest thing in the world for me.

Declaration

I declare that the work in this thesis has been done by myself and has not been submitted elsewhere for the award of any other degree. The numerical analysis within this thesis has been performing by using the statistical programming language R and Matlab.

Thitiya Theparod

To *my beautiful mother*

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Chapter 1

Introduction

1.1 Motivation and objective

Sexually transmitted diseases (STDs) are infectious diseases that can be acquired and passed on from one person to another through sexual contact. STDs have gained considerable attention from researchers in various fields, including mathematicians and statisticians with the aim of identifying effective intervention and control strategies to reduce the impact of the diseases. Mathematical modelling has proved to be a very useful tool in providing insights into understanding and for analysing the spread and control of infectious diseases (Hethcote (2000)). The transmission dynamics and epidemiology of STDs rely mainly on sexual contacts based on human behaviours, in which each contact generates a route of infection. Various mathematical models in the area of sexually transmitted diseases have been extensively studied in the past three decades. In Hethcote & Yorke (1984), pioneering work was done into the study of gonorrhoea. Summaries of various mathematical models in STD research can be found in Anderson et al. (1986), Anderson & May (1991), Dietz & Hadeler (1988), Kretzschmar et al. (1996) and Ferguson & Garnett (2000).

The objective of this thesis is to propose suitable mathematical models describ-

ing dynamics of sexually transmitted diseases with an ability to answer 'what if' questions regarding epidemic behaviour: is a major epidemic outbreak possible? If so, what is the probability of a major epidemic outbreak? If the epidemic takes off, what is the endemic level? What is the probability of the disease going extinct, and what is the time to extinction? Mathematical and computational tools will be key to answering these questions. In addition, the models should be able to assist the identification of successful interventions or control strategies to deliver public health benefits. To provide some background understanding, we start by giving a brief overview of classical epidemic models.

1.2 Homogeneous - mixing models in a closed population

In general, infectious disease models implicitly assume that contact patterns are highly homogeneous, i.e. interaction between individuals is assumed to be uniformly at random (Anderson & May (1991)). The first epidemic model was the SIR epidemic model (Kermack & McKendrick (1927)) where individuals are in one of three states: susceptible (S), infectious (I) or removed (R). A susceptible individual may become infected at some point in time when the contact is made with an infected individual. At the end of the infectious period, the individual recovers and becomes completely immune. As a consequence, there are only two transitions occurring between states: from S to I and from I to R, see figure 1.1.

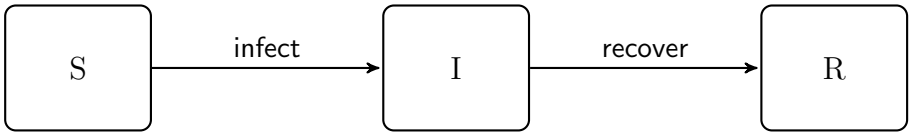


Figure 1.1: State diagram for the SIR model

Other types of models such as SI, SIS and SIRS models are modifications of the

basic SIR model. If individuals become susceptible immediately after recovering, we can say that the model has no immune state, and such a model is called an SIS epidemic model. Since very few sexually transmitted diseases confer immunity after infection, the SIS model is relevant to many STDs. Therefore, throughout this thesis, we focus our attention on SIS models, see Figure 1.2.

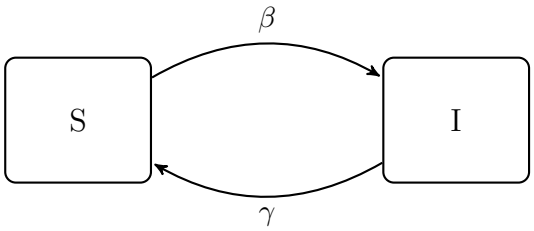


Figure 1.2: State diagram for the SIS model

The dynamics of the model are described as follows. Infectious individuals make infectious contacts with other individuals at the points of a homogeneous Poisson point process with rate β. Each infectious contact is with a randomly selected individual, and all contacts made by infectives are mutually independent. If the selected individual is susceptible then that individual is infected, otherwise the infectious contact has no effect. Individuals have independent and identically distributed infectious period, at the end of the period, individuals are recovered and immediately become susceptible to reinfection. If the infectious period is chosen to be exponentially distributed, then the model is Markovian (Bailey (1975), Ch. 6.3).

We now define the standard stochastic SIS epidemic model. The population is assume to be closed of size n. For $t \geq 0$, let $S(t)$ and $I(t)$ denote the total number of susceptible and infectives at time t. Hence, $S(t) + I(t) = n$, for all $t \geq 0$. We can see that in SIS model, there is only one independent variable, $I(t)$, as $S(t) = n - I(t)$. The stochastic process $\{I(t) : t \geq 0\}$ is described by the

following transition probabilities:

$$Pr(I(t+\Delta t)=i+1|I(t)=i)=\beta i\frac{(n-i)}{n}\Delta t+o(\Delta t), \tag{1.1}$$

$$Pr(I(t+\Delta t)=i-1|I(t)=i)=\gamma i\Delta t+o(\Delta t). \tag{1.2}$$

β is the rate at which an individual make an "infectious" contact. At time t, each infectious contact has probability $\frac{n-I(t)}{n}$ of being with a susceptible, and γ is the recovery rate for infected individuals. Note that, the constant γ yields an exponential distribution for the infectious periods (Giesecke (1994)). In other words, $D \sim Exp(\gamma)$ with $E[D]=\frac{1}{\gamma}$.

The stochastic model described above also has a deterministic counterpart. For $n \to \infty$, suppose that $\lim_{n\to\infty}\frac{I(0)}{n}=x(0)$. Then, the process density of infective, $\{\frac{I(t)}{n}; t \geq 0\}$, converges to a deterministic limit described by a differential equation (Kurtz (1970)). For convenience, let $\dot{x}(t)=\frac{dx(t)}{dt}$, then we have

$$\dot{x}(t)=\beta x(t)(1-x(t))-\gamma x(t).$$

A key quantity of interest in epidemiology is the basic reproduction number (R_0). In a homogeneously mixing population R_0 is simply the mean number of susceptibles infected by an infective in a totally susceptible population (Anderson & May (1991)). The basic reproduction number is defined as follows:

$$R_0=\frac{\beta}{\gamma} \tag{1.3}$$

From (1.3), $\dot{x}(t)$ can be rewritten as $\dot{x}(t)=\gamma(R_0(1-x(t))-1)x(t)$. We can immediately see that $R_0=1$ is a critical point. If $R_0 \leq 1$, we can see that $\dot{x}(t)<0$, for all $x(t)>0$. Therefore, $x(t)$ decreases monotonically as $t \to \infty$ to the disease-free equilibrium $x^*=0$. If the epidemic starts with one initial infective, $x(0) \approx 0$, then during the early stages, $1-x(t) \approx 1$, giving $R_0(1-x(t))>1$, if

$R_0 > 1$.

Then if $R_0 > 1$, $\dot{x}(t) > 0$ with $x(t)$ increasing. The endemic equilibrium occurs when $\dot{x}(t) = 0$, this gives the equation $R_0(1 - x(t)) - 1 = 0$. Hence, the endemic level is at $x(t) = 1 - 1/R_0$. As a result, $1/R_0$ is the fraction of the population susceptible at equilibrium. In conclusion, if $R_0 \leq 1$, $x(t)$ decreases and converges to 0, in other words, the disease dies out. If $R_0 > 1$, $x(t)$ increases and converges to $1 - 1/R_0$, which is the fraction of the population infected at the endemic equilibrium, in other words, the disease takes off.

We now return to the stochastic SIS model. $R_0 = \frac{\beta}{\gamma}$ is the mean number of contacts by an individual in the epidemic. Therefore if $R_0 < 1$ each individual is making on average less than one contact and the size of the epidemic will be decreasing.

One property of the stochastic SIS model is that it has an absorbing state at the origin (the number of infectives is 0). When the epidemic reaches the absorbing state, we can say that the epidemic has gone extinct. Starting the epidemic with one infected individual, during the early stages, all infectious contacts are likely to be with susceptibles. Equations (1.1) and (1.2) define a finite state space Markov process, where each state refers to number of infectives. When we start the process with a positive number of infectives, from any state i we can reach any state j with positive probability. All states other than 0 infectives are transient, that is, for fixed n we only return to each state finitely often. Therefore, the process will eventually reach the absorbing state. As a consequence, there are two possibilities either the absorbing state is visited quickly or not, but the probability of the epidemic going extinct eventually (enter the absorbing state of 0 infectives) is one. As such, the probability of *early extinction* is another quantity of interest in epidemiology. We are interested in whether or not the epidemic takes off. When $R_0 \leq 1$, the probability of early extinction is 1. When $R_0 > 1$, there is possibility that the disease will either go extinct early or take off in the population, therefore,

the probability of early extinction is positive but less than one.

To make the above statement more concrete, "early stages" and "early extinction" are described as the following. Let $b_n = \log(n)$. We define early stages up until the total number of the population infectious first reaches b_n. Then early extinction is defined as extinction during the early stages. That is the total number infected never reaches b_n, where $b_n \to \infty$ as $n \to \infty$. The probability of early extinction converges to the extinction probability of the approximating branching process, which is studied in Section 1.3.

1.3 Branching process approximation

Many studies have been devoted to prediction of the incidence of epidemics. In general, epidemics have non-linear dynamics which makes them potentially difficult to work with. However, in the initial stages, a linear approximation can be made if all infectious contacts are assumed to be with susceptibles (Whittle (1955)).

The basic framework of the branching process is that the process starts with a single individual. The initial individual produces a random number of offspring. These offspring reproduce independently of each other. In the context of epidemics, infections correspond to births of offspring in the branching process terminology. Ball (1983) and Ball & Donnelly (1995) use a coupling argument to link the epidemic process with an approximate branching process, saying that the two processes agree until an infectious contact in the epidemic is with a previously infected individual.

Branching processes can be categorised into two types: discrete time branching processes and continuous time branching processes. Discrete time branching processes are easier to work with in order to answer our key questions of interest. Even though, the time scale in the epidemic process is continuous, it is possible to embed the process in a discrete time branching process based on successive gen-

erations of the epidemic process. The initial individuals in the branching process form generation 0. Then for $k \geq 0$, the offspring of the individuals in generation k form generation k+1 of the branching process. Individuals in different generations are alive at the same time. We focus on the successive generations rather than the time at which infected individuals are produced. Therefore, discrete time branching processes, known as Galton-Watson (GW) branching processes, are employed in this thesis, and are the focus on a discrete time branching process in this section. Useful branching process references are Harris (1963), Jagers (1975) and Haccou et al. (2005).

1.3.1 A Galton-Watson (GW) process

In the context of epidemiology, a key question is whether or not an infected individual introduced into a large population can cause a major outbreak. In answering this question the offspring distributions are important. The mean number of offspring of the approximating GW process corresponds to the basic reproduction number (R_0) in the epidemiology context.

The basic branching processes are based on the following concepts. The process starts with a single individual. Consider a sequence of random variables $\{Z_n, n \in \mathbb{N}_0\}$, where Z_n denotes the number of individuals in the n^{th} generation. Let $\xi_{n,i}, n, i \in \mathbb{N}_0$ be independent and identically distributed random variables according to ξ with distribution $\{p_k, k \in \mathbb{N}_0\}$, where $\xi_{n,i}$ denotes the number of offspring of the i^{th} individual in generation n. In other words, $P(\xi = k) = p_k$. The process starts at time zero (generation 0) with 1 initial individual, $Z_0 = 1$, in which the individual produces a random number of offspring, $Z_1 = \xi_{0,1}$. These individuals will reproduce independently of each other according to the same distribution. Therefore, the number of individuals in the n^{th} generation satisfies $Z_n = \sum_{i=1}^{Z_{n-1}} \xi_{n-1,i}, n \geq 1$.

One of the most interesting results of branching processes is the probability of extinction. Its applications give answers to the question of what is the probability that the disease dies out before a major outbreak occurs. If $Z_n = 0$, for any n, the branching process is said to have gone extinct. Since $Z_n = 0$ implies $Z_{n+k} = 0$ for all $k > 0$, the extinction time denoted by τ satisfies $\tau = \min\{n : Z_n = 0\}$. If there is no n such that $Z_n = 0$, then $\tau = \infty$. Therefore, the probability of extinction can be represented as $Pr(\tau < \infty)$. An important tool in determining the extinction probability is the probability generating function (p.g.f.) of the offspring distribution. Recall that ξ_n is a discrete random variable denoting the number of individual in the n^{th}generation, taking values in $\{0, 1, 2, ...\}$ with associated probabilities,

$$P(\xi = k) = p_k, \quad k = 0, 1, 2, ...,$$

where $\sum_{k=0}^{\infty} p_k = 1$.
Then $\mathbb{E}[\xi_n]$ is the expectation of ξ_n,

$$\mathbb{E}[\xi_n] = p_1 + 2p_2 + 3p_3 + ... = \sum_{k}^{\infty} kp_k = m.$$

The random variable ξ_n has the p.g.f given by

$$g(s) = \mathbb{E}[s^{\xi_n}] = p_0 + p_1 s + p_2 s^2 + ... = \sum_{k=0}^{\infty} p_k s^k, \quad 0 \leq s \leq 1. \tag{1.4}$$

As mentioned earlier, the mean offspring number $m = \sum_{k}^{\infty} kp_k$ is the quantity of interest, $R_0 = m$. The process is said to be *supercritical* if $R_0 > 1$, *critical* if $R_0 = 1$, *subcritical* if $R_0 < 1$. Note that $\sum_{k=0}^{\infty} p_k = 1$. Assume that there is no k such that $p_k = 1$, and that $p_0 + p_1 < 1$, then we have that $g(s)$ is strictly convex with strictly increasing first derivative for $0 \leq s \leq 1$. Hence, $g(s) = s$ has either

one or two roots, one of which must be 1 ($g(1) = 1$). Also, $g(0)$ is the probability of having no offspring. Therefore, $g^{'}(1) = m > 1, g(1) = 1$ and $g(0) > 0$. This implies a second solution of $g(s) = s$ on $[0, 1)$, see Figure 1.3 for more graphical explanation.

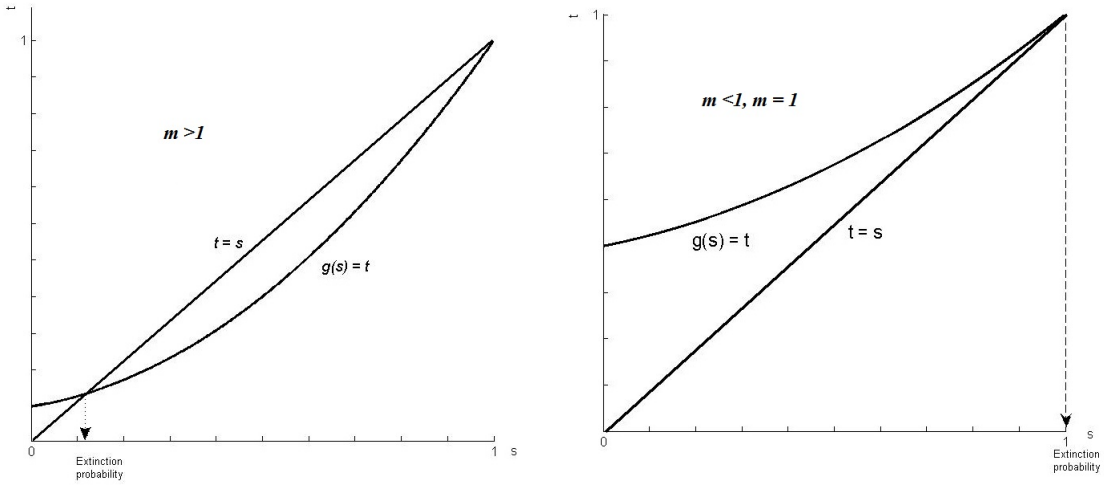


Figure 1.3: Graphs showing extinction probabilities for supercritical, subcritical and critical cases.

As shown Figure 1.3, we can see from the graph that when $m > 1$, except at $t = 1$, the curve $g(s)$ also crosses the line $t = s$ at $t < 1$. Thus there exist a solution to the equation $g(s) = s$ between 0 and 1. Then, this solution will be the extinction probability since the quantity is the smallest non- negative root. In the subcritical case ($m < 1$) and the critical case ($m = 1$), there is no possibility for the curve $g(s)$ to cross the line apart from at $t = 1$. Therefore, there is no other solution to the equation $g(s) = s$ except $t = 1$. The following theorem is hence established (see Haccou et al. (2005) for more details).

Theorem 1.3.1 *The probability of extinction of a branching process with one initial infective is the smallest non-negative root of the equation $g(s) = s$. The solution is less then 1 if and only if $m > 1$, and equals to 1 if $m \leq 1$.*

1.3.2 The large population limit

The stochastic model is a suitable starting point for a model of STDs dynamics and will be applied in this thesis. For large populations, the dynamics once the epidemic has taken off are well approximated by a deterministic approximation and studying this is informative, especially in identifying the endemic equilibrium. The deterministic model is often a system of ordinary differential equations (ODE). For large populations, Kurtz (1970) and Kurtz (1971) tie together the deterministic and stochastic models. Kurtz's theorem states that the deterministic dynamic is a good approximation of the stochastic process as n becomes large (Kurtz (1970), Theorem 3.1). More specifically, for large n, the stochastic process scaled by the population size n, $\left\{\frac{X_n(t)}{n}, t \geq 0\right\}$, converges to a deterministic process $x(t)$ that is the solution of a system of ordinary differential equations, with initial value $x(0) = x_0$, i.e. $\lim_{n\to\infty} \sup_{s\leq t} \left|\frac{X_n(s)}{n} - x(s)\right| = 0$. The results are stated in Theorem 1.3.2. Another useful result is stated in Theorem 1.3.3.

Theorem 1.3.2 *(Kurtz (1970), Theorem 3.1)*

Let $X_n(t)$ be a one parameter family of time-homogeneous Markov processes with state space $E_n \subset \mathbb{Z}^K$, where $\mathbb{Z}^K$ denotes the set of K-vectors with integer components. Define $q^n_{k,k+l} = \lim_{\Delta t \longrightarrow 0} Pr\{X_n(\Delta t) = k + l | X_n(0) = k\}/\Delta t$. Suppose there exists a function $f(x, l), x \in \mathbb{R}^K, l \in \mathbb{Z}^K$ that satisfies

$$q^n_{k,k+l} = nf\left(\frac{1}{n}k, l\right), l \neq 0$$

Define $F : \mathbb{R}^K \to \mathbb{R}^K$ by

$$F(x) = \sum_l lf(x, l)$$

Suppose there exists an open set E in $\mathbb{R}^K$ and a constant M such that

$$1. |F(x) - F(y)| \leq M|x - y|, x, y \in E,$$

$$2. \sup_{x \in E} |l| f(x, l) < \infty,$$

$$3. \lim_{d \to \infty} \sup_{x \in E} \sum_{|l| > d} |l| f(x, l) = 0.$$

Let $X(s; x_0)$ satisfy $\frac{\partial}{\partial s} X(s; x_0) = F(X(s; x_0))$ with initial value $X(0, x_0) = x_0$, where $X(s; x_0) \in E, 0 \leq s \leq t$. Let $\lim_{n \to \infty} \frac{X_n(0)}{n} = x_0$ for the original Markov processes. For every $\epsilon > 0$, we have

$$\lim_{n \to \infty} Pr \left(\sup_{t \leq T} \left| \frac{1}{n} X_n(t) - X(t; x_0) \right| > \epsilon \right) = 0.$$

In an SIS epidemic, let $X_n(t)$ be the number of infectives at time t, β be the infection rate, and γ be the recovery rate. Following the notation in Theorem 1.3.2, let

$$q_{i,j}^n = \lim_{\Delta t \longrightarrow 0} Pr\{X_n(\Delta t) = j | X_n(0) = i\} / \Delta t.$$

It is then straightforward to show that

$$\begin{aligned} q_{i,i-1}^n &= n \left(\gamma \frac{i}{n} \right) = nf \left(\frac{i}{n}, -1 \right) \\ q_{i,i+1}^n &= n \left(\beta \frac{i}{n} \frac{(n-i)}{n} \right) = nf \left(\frac{i}{n}, +1 \right) \\ q_{i,k}^n &= 0, \quad k \neq i+1, i-1. \end{aligned}$$

where the corresponding density family functions are

$$f(x,1) = \beta x(1-x) \tag{1.5}$$

$$f(x,-1) = \gamma x \tag{1.6}$$

$$f(x,k) = 0, \quad k \neq 1,-1. \tag{1.7}$$

Therefore, $F(x) = \beta x(1-x) - \gamma x$. For $x \in (0,1]$, we have

$$\begin{aligned} F(x) - F(y) &= \beta x(1-x) - \gamma x - \beta y(1-y) + \gamma y \\ &= \beta x(1-x) - \beta y(1-x) + \beta y(1-x) - \beta y(1-y) + \gamma(y-x) \\ &= \beta(x-y)(1-x) + \beta y\left((1-x) - (1-y)\right) + \gamma(y-x) \\ &= \beta(x-y)(1-x) - \beta y(x-y) + \gamma(y-x) \end{aligned}$$

Since $0 < x, y \leq 1$, then $0 < (1-x) \leq 1$. Therefore, $\beta|(x-y)(1-x)| < \beta|(x-y)|$ and $\beta|y(x-y)| < \beta|(x-y)|$. That is,

$$\begin{aligned} |F(x) - F(y)| &= |\beta(x-y)(1-x) - \beta y(x-y) + \gamma(y-x)| \\ &< |\beta(x-y)| + |\beta(x-y)| + |\gamma(y-x)| \end{aligned}$$

Hence, $|F(x) - F(y)| < (2\beta + \gamma)|(x-y)|$. We also have that

$$\sup_{x \in E} |l| f(x,l) = \sup_{x \in (0,1]} \{\beta x(1-x) + \gamma x\} \leq \beta + \gamma < \infty.$$

According to (1.7), it immediately follows that

$$\lim_{d \to \infty} \sup_{x \in (0,1]} \sum_{|k|>d} |k| f(x,k) = 0.$$

If $\lim_{n\to\infty} \frac{X(0)}{n} = x(0)$, then it follows that for every $\epsilon > 0$,

$$\lim_{n\to\infty} Pr\left(\sup_{s\leq t}\left|\frac{X_n(s)}{n} - x(s)\right| > \epsilon\right) = 0,$$

where $x(s)$ solves $\dot{x}(t) = F(x)$, where $F(x) = \beta x(1-x) - \gamma x$ with initial condition $x(0)$.

Now, consider the differences between $\frac{X_n(s)}{n}$ and $x(s)$, for $s \leq t$. Theorem 1.3.3 states that $\sqrt{n}\left|\frac{X_n(s)}{n} - x(s)\right|$ converges to a diffusion process. The diffusion process describes the fluctuations of the stochastic process about its deterministic approximation and is particularly useful for studying the behaviour about the endemic equilibrium.

Theorem 1.3.3 *Suppose that F and G are uniformly continuous on E, where F is defined as in Theorem 1.3.2. Suppose also that $G(x) = \sum_l l^2 f(x,l)$ where $sup_x G(x) < \infty$. We have that*

$$\lim_{n\to\infty} \sqrt{n}(X_n(0) - x) = v$$

implies that $V_n(t) = \sqrt{n}(X_n(t) - X(t,x))$ converges weakly to the diffusion $V(t)$ with $V(0) = v$ and $V(t) = V(0) + \frac{1}{\sqrt{n}}\int_0^t \sqrt{G(X(s))}dB_s + \int_0^t F(X(s))ds$.

Note that Theorem 1.3.3 is simplified from Theorem 2.2 in Pollett (2001), and that dB_s denotes integration with respect to K-dimensional Brownian motion. For studies of the behaviour at equilibrium, the appropriate diffusion process approximation is Ornstein - Uhlenbeck (OU) process (Barbour (1976)). If the initial value of the process is chosen close to the equilibrium point then the limiting process is an OU process (Pollett (2001)).

1.4 Multi-type branching processes

An important extension of the Galton-Watson process which will be used extensively in this thesis is the multi-type branching process. For multi-type branching processes, we allow for different types of individuals. It is worthwhile to give an example of the concept of types. For sexually transmitted diseases, suppose that infections are transmitted only through heterosexual contacts. Then, individuals can be distinguished into two types: male and female. If an infected male makes an infectious contact with a susceptible female, then an infection is said to be of type 1, or of type 2 if an infected female makes an infectious contact with a susceptible male. Therefore an infectious male produces a random number of female offspring (type 2 offspring), an infectious female produces a random number of male offspring (type 1 offspring). Then the number of offspring of each type is the key quantity for determining the basic reproduction number. This formulates the framework of a 2-type branching process. More generally, let us define a set of type $K = \{1, 2, ...k\}$, for integer k, such that, $2 \leq k < \infty$. Let $\mathbf{Z}_0, \mathbf{Z}_1, \mathbf{Z}_2, ...$ be $|k|$-dimensional vectors, where $\mathbf{Z}_n$ is a random vector representing the number of individuals in n^{th} generation, $\mathbf{Z}_n = (Z_n^{(1)}, Z_n^{(2)}, ..., Z_n^{(k)})$. Let $Z_n^{(r)}$ be the number of individuals of type r in generation n, where $Z_n^{(r)}$ is the r^{th} component in vector $\mathbf{Z}_n$. Let $\xi_r = (\xi_r^1, \xi_r^2, ..., \xi_r^k)$ be an associated offspring random vector of type $r \in K$, where ξ_r^k is a random variable representing the number of offspring of type k born

from a parent of type r individual. For $i, j = 1, 2, ..., k$, we are interested in the number of type j offspring that a type i individual has. The mean number of offspring of k different types can be presented in a $k \times k$ matrix. This matrix is called the mean offspring matrix or the next-generation matrix, denoted by M. Each element m_{ij} denotes the mean number of type j offspring born from a type i parent, $m_{ij} = E[\xi_i^j]$. The next-generation matrix can be constructed as follows:

$$M = \begin{bmatrix} m_{11} & m_{12} & m_{13} & \cdots & m_{1k} \\ m_{21} & m_{22} & m_{23} & \cdots & m_{2k} \\ m_{31} & m_{32} & m_{33} & \cdots & m_{3k} \\ \vdots & \vdots & \vdots & \vdots & \vdots \\ m_{k1} & m_{k2} & m_{k3} & \cdots & m_{kk} \end{bmatrix} \tag{1.8}$$

The next-generation matrix is the basis for the calculation of the important threshold parameter, R_0. The basic reproduction number, R_0, is the spectral radius or dominant eigenvalue of the next-generation matrix (Diekmann, Heesterbeek & Metz (1990)).

The probability generating function (p.g.f) is also the key to determining the probability of extinction for multi-type branching processes. Assuming that initially we have $Z_0^{(r)} = 1$, $Z_0^{(j)} = 0, j \neq r \in K$, then the p.g.f of $\mathbf{Z}_1$ is denoted by $g^{(r)}(s_1, s_2, ..., s_k)$.

$$g^r(s_1, s_2, ..., s_k) = \sum_{\xi_r^{(1)}, ..., \xi_r^{(k)}}^{\infty} p^r(\xi_r^{(1)}, ..., \xi_r^{(k)}) s_1^{\xi_r^{(1)}} ... s_k^{\xi_r^{(k)}}, \qquad |s_1|, ..., |s_k| \leq 1, \tag{1.9}$$

where $p^r(\xi_r^{(1)}, ..., \xi_r^{(k)})$ is the probability that an individual of type r has ξ offspring

of type 1, 2, ..., k. Let

$$\mathbf{s} = (s_1, s_2, ...s_k), \quad s_1, s_2, ...s_k \in [0, 1],$$
$$\mathbf{g}(\mathbf{s}) = (g^1(\mathbf{s}), ..., g^k(\mathbf{s})).$$

Then the probability of extinction is the smallest non-negative root of equation $\mathbf{g}(\mathbf{s}) = \mathbf{s}$. Note that s_i is the probability of extinction starting from a type i individual.

1.5 The standard model for sexually transmitted diseases

We can see that homogeneously mixing SIS epidemic models have been intensively studied and many results are obtained. However, for sexually transmitted diseases, random interaction between individuals is not appropriate for describing contacts within sexual relationships, as sexual activity is simply not random (Laumann et al. (1994)). One can include heterogeneity into the models by stratifying the population into subpopulations defined by their dynamic nature or the heterogeneity in sexual activity. Anderson et al. (1986) and Knox (1986) have developed this idea by dividing the total population into subpopulations according to their sexual preferences and their rate of partner change whilst ignoring the aspect of pair formation. Dietz & Hadeler (1988) then developed the basic model for STDs by generalising the classical SIS model with the pair formation taken into account. Despite the disease status, an individual is also described as being single or in a relationship with another individual (see Figure 1.4).

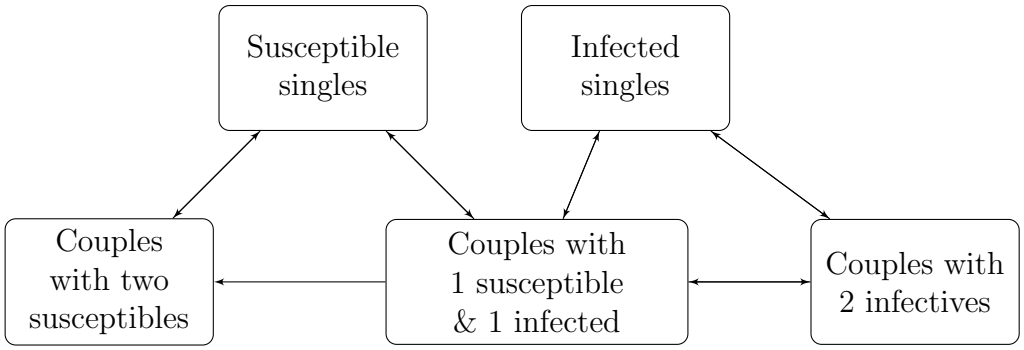


Figure 1.4: A pair formation and separation model

Disease transmission can only take place within a relationship. Therefore, the relationship consisting of two susceptibles can be considered temporarily immune as there is no transmission occurring within the relationship. This aspect can affect the spread of an infection especially in the initial stage of an epidemic as the majority of existing couples consist of susceptibles. Dietz (1988) made a comparison between two models for HIV; one model with pair formation and one model without pair formation. He observed that with pair formation, the rise of incidence of HIV towards the endemic equilibrium is much slower and the endemic level is smaller than estimated by the model without pair formation.

In addition to being single and in a relationship, Anderson et al. (1986) also included gender classification in the model. By incorporating these fundamental factors into a deterministic model and considering the transition between states in terms of pair formation, separation rate, recovery and infection rates, the model can be represented by a system of differential equations. In particular, if we divide the population into two groups: male and female. For heterogeneous populations, especially for sexually transmitted disease models, individuals are assumed to mix between groups, allowing the distinct subgroups to have different contact rates, α_i, where i represents group index, in this case $i = 1, 2$. Other rate parameters, such as recovery and infectious rates are also set accordingly to the distinct subgroups. Moreover, we can further subdivide each group by disease status: susceptible,

infective and by relationship status: single and non-single. Then, the overall framework is essentially that of the SIS model but with a larger state space. In this case, the state space will be 8 dimensional:

$$\{M_I, F_I, M_S, F_S, C_{M_I F_S}, C_{M_S F_I}, C_{M_S F_S}, C_{M_I F_I}\}$$

Where M and F represent state of single male and single female, respectively. Index I and S denote disease status: infectious and susceptible. C represents a couple state, and its index indicates the disease status of male and female within the relationship. For instance, $C_{M_I F_S}$ denotes a couple state within which the male is infected and the female is susceptible.

However, as the dimension of the differential equations as well as the stochastic process increases, it becomes in general more difficult to solve. Therefore, it is important that whilst we look to make the model realistic we ensure that it remains amenable to mathematical analysis.

1.6 The threshold parameter for sexually transmitted diseases

The basic reproduction number, R_0, has an intuitive definition for the homogeneously mixing model as the mean number of infectious contacts made by an infective in an otherwise susceptible population (Anderson & May (1991)). This threshold behaviour has received special attention by practitioners. In practice, it provides a quantitative guide to both invasion and control of an infectious disease (Heesterbeek (2002)). In an endemic infection, ultimately, we are looking for control measures which bring R_0 below 1. An important interest is how to generalise the concept of R_0 for general heterogeneous and structured populations.

In a homogeneous population, R_0 is the product of the contact rate per unit of

time (λ) and the mean infectious period ($E[D]$), $R_0 = \lambda E[D]$. In a heterogeneous population, R_0 is less straightforward. It is no longer equal to the average number of infectious contacts produced by an infected person (Liljeros et al. (2003)). However, we can still define a quantity R_0 as an informative threshold condition for whether or not the epidemic will take off. Diekmann, Dietz & Heesterbeek (1990) showed that, for sexually transmitted diseases where the heterogeneity is taken into account, we can make use of the ideas of the underlying R_0 for homogeneous populations. For example, if the host population is partitioned into 2 subgroups. R_0 for an infected individual in group 1 or 2 (R_0^1 and R_0^2) can be derived using a similar idea, but now the infection rate depends upon who acquires infection from whom. Let N_1 and N_2 be the population size of group 1 and 2, where everyone in the population is susceptible. $\lambda_{i,j}$ denotes the infectious rate at which a susceptible individual in group j acquires the disease from an infected individual in group i, where $i, j \in \{1, 2\}$. According to Rock et al. (2014), R_0^1 and R_0^2 can be expressed as the following.

$$R_0^1 = E[D_1](\lambda_{1,1}N_1 + \lambda_{2,1}N_2) \tag{1.10}$$

$$R_0^2 = E[D_2](\lambda_{1,2}N_1 + \lambda_{2,2}N_2), \tag{1.11}$$

where $E[D_1]$ and $E[D_2]$ are the mean of infectious period of infected individuals in group 1 and 2 respectively. R_0 for the population level is calculated by averaging these two values :

$$R_0 = w_1 R_0^1 + w_2 R_0^2 \tag{1.12}$$

where w_1 and w_2 are weighting average corresponding to the ratio of early infection of group 1 and group 2. One way of determining values of w_1 and w_2 is to calculate

the eigenvector associated with the dominant eigenvalue of the Jacobian at the disease - free equilibrium.

In summary, the basic reproduction number, R_0, can be derived either from stochastic model or from its deterministic counterpart. For deterministic models, R_0 can be derived by directly solving the system of differential equations in order to find a critical point (see Section 1.2). Stochastically, for higher dimensional problems, R_0 can be determined using multi-type branching processes to find the dominant eigenvalues of the *next generation matrix* as described earlier in Section 1.4. A key motivation for using the stochastic model is that only the stochastic model can be used to answer questions regarding the diseases extinction (Harris (1963), Ch. 1). Examples of derivation of R_0 for sexually transmitted diseases using stochastic models can be found in (Mode (1997) and Diekmann, Dietz & Heesterbeek (1990)).

1.7 Outline of the thesis

In Chapter 2, the sexual network modelling describing dynamics of STDs is introduced. We first focus on a simple case where there are no concurrent partnerships and the disease can only be spread between partners. We employ a stochastic SIS model along with a deterministic approximation. A 2-type branching process approximation for the early stages of the epidemic is obtained. This is used to derive the threshold parameter R_0 and the probability that the disease goes extinct in Sections 2.3 and 2.4, respectively. In Section 2.5, we move away from the early stages of the epidemic, and we study the behaviour at equilibrium assuming that there is a major outbreak ($R_0 > 1$). In this case, we employ a deterministic approximation of the Markov model to determine the endemic level, according to Theorem 1.3.2. In terms of the dynamic behaviour about the equilibrium, the limiting diffusion (Ornstein-Uhlenbeck) process approximation is exploited. In a

finite population, the epidemic will eventually go extinct with probability 1. In Section 2.6, we use stochastic simulations to explore the mean time to extinction, started at the endemic equilibrium. Finally, we present numerical results in Section 2.7. Throughout this thesis, we refer to the model constructed in this chapter as Model 1.

In Chapter 3, we extend the model presented in Chapter 2 by allowing individuals to have "one-night stands". In other words, there are sexual contacts outside partnerships, leading to the disease transmission occurring outside relationship stages. Similar studies to the model in Chapter 2, but now using a 5-type branching process, the derivation of R_0 and the probability of extinction are obtained in Sections 3.3 and 3.4, respectively. In order to obtain the endemic level, the deterministic counterpart of the stochastic SIS model is then derived in Section 3.5. The mean time to extinction is studied using stochastic simulations formulating the Markov process, discussed in Section 3.6. The numerical results are presented in Section 3.7. Throughout this thesis, we refer to the model constructed in this chapter as Model 2.

Chapter 4 discusses control strategies focussing on incorporating medication use, in which medications can be given to both single and non-single individuals. If the medications are given to individuals in a relationship, they can also pass on the medications to their partners. By having this control measure, individuals within relationships are now able to recover simultaneously with their partners. This is applied to both the models of Chapters 2 and 3. A new parameter, v, describing the probability of an infected individual taking drugs and recovering is introduced. The models are adjusted accordingly and the threshold parameter is recomputed. The construction of Model 1 accommodating a control measure is discussed in Section 4.2. The endemic level of the underlying model is also studied in this section. Similar studies for model 2 with a control measure are presented in Section 4.3. Section 4.4 illustrates their numerical results. In addition

to the control strategies being addressed in this chapter, we also give some case studies that illustrate the applications of our models to a particular disease which is gonorrhoea in section 4.5.

Finally in Chapter 5, we make some conclusions and suggest some directions for future work to extend the findings of this thesis.

Chapter 2

Sexual network modelling

2.1 Introduction

The structure of sexual networks varies, as each society has its own social interactions depending upon their culture and other social factors. It is not possible to construct a network by taking into account all possible details due to the complexity of the network and the many unmeasurable components. However, we can gain an insight into the spread of the disease over the network by simplifying the sexual contact network. Therefore, we develop an SIS model with the assumption that men and women are faithful. Namely, each man and woman has either one or no partner (single) at any point in time. We establish a branching process approximation for the early stages of the epidemic whilst a deterministic model is employed to study the endemic equilibrium.

In Section 2.2 we give a description of the model formulation and assumptions. Section 2.3 discusses a two-type branching process approximation and the derivation of an expression of the threshold parameter including some interesting mathematical findings. The question of extinction is addressed in Section 2.4. Then we discuss the behaviour of the epidemic at the endemic equilibrium, in particular, the endemic level and fluctuations about the endemic level in Section

2.5. The time to extinction is studied in Section 2.6, and the numerical results are presented in Section 2.7.

2.2 Model formulation

In our model we focus on a heterosexual population in which individuals are characterized by gender and disease status. We use a Markov model to describe the disease and relationship dynamics. Therefore, the future evolution of the disease and population depends only upon the current disease status of the population. Our model assumptions are as follows.

Assumption 1. The population is large and finite, consisting of two types of individuals: males (type 1 individuals) and females (type 2 individuals). We assume that the population has n individuals of each type, i.e. the numbers of males and females are equal.

Assumption 2. The epidemic starts with one infected individual in an otherwise susceptible population. Throughout this research, we assume that the initial infective is of type 1 (male), with identical arguments holding for a female initial infective.

Assumption 3. An individual can only have one partner at a time. Therefore there are no concurrent relationships involving the same individual.

Assumption 4. A single male attempts to form a relationship with a female at the points of a homogeneous Poisson point process with rate α. The female is chosen uniformly at random from the entire population. If the female is single, a relationship is formed, otherwise nothing happens.

Assumption 5. The relationship length follows an exponential distribution with mean $\frac{1}{\delta}$. At the end of the relationship, the relationship breaks up and both individuals will return to the single state and will be able to form a relationship

with another single individual of the opposite sex.

Assumption 6. Whilst infectious, an individual of type i makes infectious contact with their partner at the points of a homogeneous Poisson point process with rate β_i. If the partner is susceptible, they become infected when contacted. Otherwise the contact has no effect. Note that single infectious individuals can not make infections.

Assumption 7. Type i individuals have infectious periods that are independently exponentially distributed with rate γ_i.

Assumption 8. The model is a stochastic SIS epidemic model. Namely, an individual can be infected and after recovery the individual immediately becomes susceptible to reinfection.

Assumption 9. We assume that at the start of the epidemic, the population relationship structure is in equilibrium. Thus the proportion of the population in relationships (and single) remains fairly constant throughout the course of the epidemic. Let σ denote the proportion of the population single in equilibrium.

Our main focus here is the incidence of infection in the defined population. The question arises whether we can determine the disease behaviour in the population, such as, what is the probability that the disease becomes endemic in the population? Given that the disease becomes endemic, what is the endemic level and how long does the disease persist in the population? However, these population level questions are determined by individual behaviour. Important insights into the disease propagation on the network can be gained by studying the early stages of the epidemic. The ability of an infectious disease, starting with a single infected individual, to invade a susceptible population is the initial key question. As discussed in Section 1.2, the basic reproduction number (R_0) plays a key role as the threshold parameter in understanding an emerging infectious disease. R_0 is defined as the expected number of secondary infections caused by a typical in-

fected individual in an otherwise entirely susceptible population during its entire period of infectiousness. In the literature, R_0 is often derived under the assumption that individuals make no repeated infectious contacts with the same individuals. However, for sexually transmitted diseases, by definition the disease is being transmitted through sexual contacts in which the sexual contacts are usually repeated within relationships. Therefore, the same individual can potentially be re-infected by an infective partner multiple times. In the literature, the basic reproduction number R_0 based upon the models that take into account relationship formation and breakup have been studied which shows that the term basic reproduction number is still applicable here (Britton et al. (2007); Diekmann, Dietz & Heesterbeek (1990); Kretzschmar et al. (1996)). Throughout the thesis, in order to account for an infection in which an infective can infect a partner more than once and distinguish the difference between the number of infections made by an infective and the number of secondary cases arising from an infective, we simply term R_0, *the reproduction number* with the definition being model specific. First, we construct a sexual network model describing the transmission dynamics based on individual behaviour.

Figure 2.1 displays the relationship network between individuals. Each node represents a relationship stage and is labelled with disease status, namely 1 represents an infected single individual and 0 represents a susceptible single individual. In a relationship, note that the first digit represents the individual of type 1 (male) whilst the second digit represents the individual of type 2 (female). For example, node (10) represents a partnership formation state with an infected male and a susceptible female. Moreover, a comma between 2 digits illustrates the break up of a relationship. For example (1,0) represents a relationship dissolution resulting in a single infected male and a single susceptible female. The transition rates are represented by parameters on arrows between states. We start the process with one infected individual, say type 1(male).

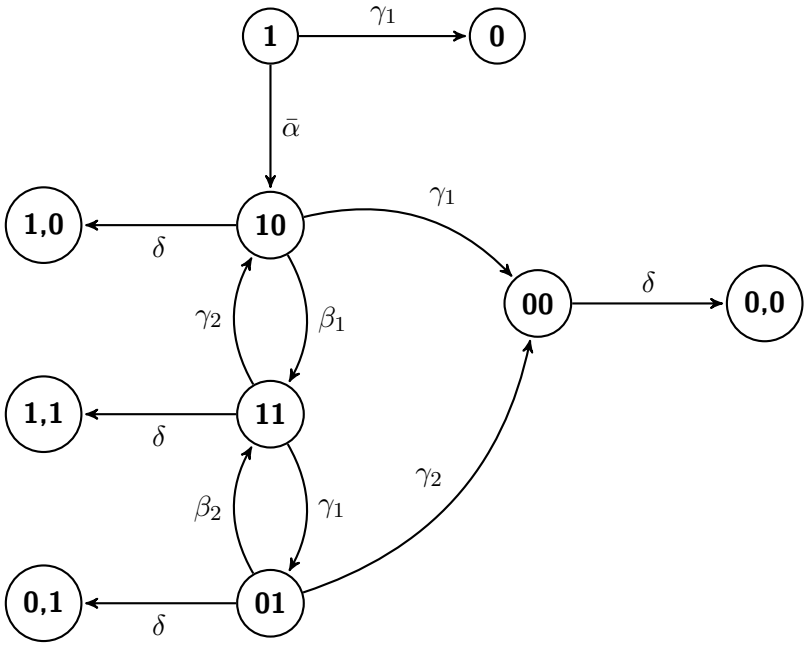


Figure 2.1: Sexual network diagram

As the figure 2.1 shows, an infected individual will either form a relationship or recover from the disease. During the initial stages of the epidemic, an infective forms a relationship with high probability with a susceptible individual as almost the entire population is susceptible. Moreover, the single male can only choose to form a relationship with a single female in the population. Therefore, the proportion of the population who are single females at equilibrium is taken into account, namely, $\bar{\alpha} = \alpha(\sigma/2)$. Hence, the proportion of the population single in equilibrium as defined in *Assumption 9* is $\sigma = \frac{\delta}{\bar{\alpha}+\delta}$, which can be expressed in terms of α and δ as $\sigma = \frac{-2\delta+\sqrt{4\delta^2+8\alpha\delta}}{2\alpha}$.

If the initial individual recovers before he forms a relationship, he becomes susceptible and the process is terminated, we obtain one susceptible male as a result (state (0)). If he enters the relationship state (10), he can either transmit the disease to his partner with rate β_1 (move to state (11)), recover before any transmission occurs with rate γ_1 (move to state (00)), or break up with his partner with rate δ (move to state (1,0)). The relationship breakups can occur from any

disease state of the relationship, and when a breakup occurs we obtain single individuals with disease status corresponding to the state prior to break up.

Figure 2.1 forms the basis for the branching process approximation introduced in Section 2.3. Our key unit is single infected individuals. In particular, for a single infected individual we consider how many infected individuals of each type will result from the formation and breakup of their next relationship. The process can be constructed on a generation basis by considering successive relationships. At the end of each relationship, as a result, this process leads us to the following 4 possible outcomes:

- (1,1) - infected male and female,
- (1,0) - infected male and susceptible female,
- (0,1) - susceptible male and infected female,
- (0,0) - susceptible male and female.

These 4 outcomes are the secondary cases produced from an infected male (or infected female). Throughout, we call infectious individuals resulting from the formation and breakup of the relationship as "offspring" of the infected individual at the start of the relationship. This forms the basis for the branching process approximation and the derivation of the threshold parameter, R_0. We can see that the offspring are produced once the relationship has finished, and we refer to this as a relationship reproduction number. The branching process approximation will be discussed in Section 2.3.

2.3 Branching process approximation

In this section, we will describe the basic concept of the branching process, the derivation of the reproduction number as well as the probability of extinction.

As we categorised our population into two types of individuals, we will employ a 2-type branching process in order to determine the threshold parameter as well as the probability of extinction. The basic concepts of the process are as follows. The process starts with one infected individual of either type 1 or type 2. The initial infective will produce a random number of offspring. Moreover, in each generation, the offspring of different individuals are independent. The expected number of offspring of each individual of each type is the key to identifying whether or not it is possible for the disease to invade the population.

As described in the previous section, starting with one infected individual, at the end of generation 1, we obtain 4 possible cases in which either 0 or 1 offspring of each type are produced. To determine the expected number of offspring of each type generated by a single infected individual of a given type, it is necessary to calculate the probability of obtaining each of the 4 outcomes. To begin with, in order to make the calculations easier, we consider the process starting from a relationship state.

Let $Q_{i,j}$ denote the probability of having i male and j female offspring from a relationship starting with an infected male and infected female. Let $P_{i,j}$ denote the probability of having i male and j female offspring from a relationship starting with an infected male and a susceptible female. Let $F_{i,j}$ denote the probability of having i male and j female offspring from a relationship starting with an infected female and a susceptible male, where $i, j \in \{0, 1\}$. Then, let $\mathbf{Q} = (Q_{0,0}, Q_{1,0}, Q_{0,1}, Q_{1,1})$, $\mathbf{P} = (P_{0,0}, P_{1,0}, P_{0,1}, P_{1,1})$, and $\mathbf{F} = (F_{0,0}, F_{1,0}, F_{0,1}, F_{1,1})$.

According to the network diagram displayed in Figure 2.1, state (10) can either jump to state (00), (11) or (1,0) with rates γ_1, β_1 and δ, respectively. Hence, the transition probability from state (10) to state (00) is $\frac{\gamma_1}{\delta+\gamma_1+\beta_1}$, from state (10) to state (11) is $\frac{\beta_1}{\delta+\gamma_1+\beta_1}$ and from state (10) to (1,0) is $\frac{\delta}{\delta+\gamma_1+\beta_1}$.

The following formulae gives $\mathbf{P}$ in terms of $\mathbf{Q}$.

$$
\begin{aligned}
P_{1,0} &= \frac{\delta}{\delta+\beta_1+\gamma_1}\cdot 1 + \frac{\beta_1}{\delta+\beta_1+\gamma_1}\cdot Q_{1,0} + \frac{\gamma_1}{\delta+\beta_1+\gamma_1}\cdot 0 \\
P_{1,1} &= \frac{\delta}{\delta+\beta_1+\gamma_1}\cdot 0 + \frac{\beta_1}{\delta+\beta_1+\gamma_1}\cdot Q_{1,1} + \frac{\gamma_1}{\delta+\beta_1+\gamma_1}\cdot 0 \\
P_{0,1} &= \frac{\delta}{\delta+\beta_1+\gamma_1}\cdot 0 + \frac{\beta_1}{\delta+\beta_1+\gamma_1}\cdot Q_{0,1} + \frac{\gamma_1}{\delta+\beta_1+\gamma_1}\cdot 0 \\
P_{0,0} &= \frac{\delta}{\delta+\beta_1+\gamma_1}\cdot 0 + \frac{\beta_1}{\delta+\beta_1+\gamma_1}\cdot Q_{0,0} + \frac{\gamma_1}{\delta+\beta_1+\gamma_1}\cdot 1.
\end{aligned} \tag{2.1}
$$

Similarly, we can express $\mathbf{Q}$ in terms of $\mathbf{P}$ and $\mathbf{F}$, and $\mathbf{F}$ in terms of $\mathbf{Q}$ as follows.

$$
\begin{aligned}
Q_{1,0} &= \frac{\delta}{\delta+\gamma_1+\gamma_2}\cdot 0 + \frac{\gamma_2}{\delta+\gamma_1+\gamma_2}\cdot P_{1,0} + \frac{\gamma_1}{\delta+\gamma_1+\gamma_2}\cdot F_{1,0} \\
Q_{1,1} &= \frac{\delta}{\delta+\gamma_1+\gamma_2}\cdot 1 + \frac{\gamma_2}{\delta+\gamma_1+\gamma_2}\cdot P_{1,1} + \frac{\gamma_1}{\delta+\gamma_1+\gamma_2}\cdot F_{1,1} \\
Q_{0,1} &= \frac{\delta}{\delta+\gamma_1+\gamma_2}\cdot 0 + \frac{\gamma_2}{\delta+\gamma_1+\gamma_2}\cdot P_{0,1} + \frac{\gamma_1}{\delta+\gamma_1+\gamma_2}\cdot F_{0,1} \\
Q_{0,0} &= \frac{\delta}{\delta+\gamma_1+\gamma_2}\cdot 0 + \frac{\gamma_2}{\delta+\gamma_1+\gamma_2}\cdot P_{0,0} + \frac{\gamma_1}{\delta+\gamma_1+\gamma_2}\cdot F_{0,0}
\end{aligned} \tag{2.2}
$$

and

$$
\begin{aligned}
F_{1,0} &= \frac{\delta}{\delta+\beta_2+\gamma_2}\cdot 0 + \frac{\beta_2}{\delta+\beta_2+\gamma_2}\cdot Q_{1,0} + \frac{\gamma_2}{\delta+\beta_2+\gamma_2}\cdot 0 \\
F_{1,1} &= \frac{\delta}{\delta+\beta_2+\gamma_2}\cdot 0 + \frac{\beta_2}{\delta+\beta_2+\gamma_2}\cdot Q_{1,1} + \frac{\gamma_2}{\delta+\beta_2+\gamma_2}\cdot 0 \\
F_{0,1} &= \frac{\delta}{\delta+\beta_2+\gamma_2}\cdot 1 + \frac{\beta_2}{\delta+\beta_2+\gamma_2}\cdot Q_{0,1} + \frac{\gamma_2}{\delta+\beta_2+\gamma_2}\cdot 0 \\
F_{0,0} &= \frac{\delta}{\delta+\beta_2+\gamma_2}\cdot 0 + \frac{\beta_2}{\delta+\beta_2+\gamma_2}\cdot Q_{0,0} + \frac{\gamma_2}{\delta+\beta_2+\gamma_2}\cdot 1.
\end{aligned} \tag{2.3}
$$

We can then substitute (2.1) and (2.3) into (2.2) to solve for $\mathbf{Q}$. It is then

trivial to obtain $\mathbf{P}$ and $\mathbf{F}$. The solution for $\mathbf{P}$ is

$$
\begin{aligned}
P_{1,0} &= \frac{(\delta(\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\delta\gamma_1\beta_2)}{(\delta+\beta_1+\gamma_1)((\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\gamma_1\beta_2)-\gamma_2\beta_1(\beta_2+\delta+\gamma_2))} \\
P_{1,1} &= \frac{\beta_1\delta(\delta+\beta_2+\gamma_2)}{(\delta+\beta_1+\gamma_1)((\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\gamma_1\beta_2)-\gamma_2\beta_1(\beta_2+\delta+\gamma_2))} \\
P_{0,1} &= \frac{\gamma_1\delta\beta_1}{(\delta+\beta_1+\gamma_1)((\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\gamma_1\beta_2)-\gamma_2\beta_1(\beta_2+\delta+\gamma_2))} \\
P_{0,0} &= \frac{\gamma_1\gamma_2\beta_1\bar{\alpha}+\gamma_1((\delta+\gamma_1+\gamma_2)(\delta+\beta_2+\gamma_2)-\gamma_1\beta_2)}{(\delta+\beta_1+\gamma_1)((\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\gamma_1\beta_2)-\gamma_2\beta_1(\beta_2+\delta+\gamma_2))}.
\end{aligned}
$$

We now obtain the probabilities of having the 4 resulting outcomes starting from a single infected male. Therefore, the probabilities of the events occurring before going to the relationship stage need to be considered. Let $p_{i,j}^{(k)}$ define the probability of having i infected male individuals and j infected female individuals end of relationships, starting from 1 infected individual of type k, where $i,j \in \{0,1\}$ and $k \in \{1,2\}$. Thus, $p_{0,0}^{(1)} = \frac{\gamma_1}{\gamma_1+\bar{\alpha}} + \frac{\bar{\alpha}}{\bar{\alpha}+\gamma_1}P_{0,0}$, $p_{1,0}^{(1)} = \frac{\bar{\alpha}}{\bar{\alpha}+\gamma_1}P_{1,0}$, $p_{0,1}^{(1)} = \frac{\bar{\alpha}}{\bar{\alpha}+\gamma_1}P_{0,1}$, and $p_{1,1}^{(1)} = \frac{\bar{\alpha}}{\bar{\alpha}+\gamma_1}P_{1,1}$. As a result, we derive the probabilities for each of the 4 outcomes starting with an infected male as follows.

$$
\begin{aligned}
p_{0,0}^{(1)} &= \frac{\gamma_1}{\gamma_1+\bar{\alpha}} + \frac{\gamma_1\gamma_2\beta_1\bar{\alpha}+\gamma_1\{(\delta+\gamma_1+\gamma_2)(\delta+\beta_2+\gamma_2)-\gamma_1\beta_2\}\bar{\alpha}}{(\delta+\beta_1+\gamma_1)((\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\gamma_1\beta_2)-\gamma_2\beta_1(\beta_2+\delta+\gamma_2))(\gamma_1+\bar{\alpha})} \\
p_{1,0}^{(1)} &= \frac{(\delta(\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\delta\gamma_1\beta_2)\bar{\alpha}}{(\delta+\beta_1+\gamma_1)((\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\gamma_1\beta_2)-\gamma_2\beta_1(\beta_2+\delta+\gamma_2))(\gamma_1+\bar{\alpha})} \\
p_{0,1}^{(1)} &= \frac{\gamma_1\delta\beta_1\bar{\alpha}}{(\delta+\beta_1+\gamma_1)((\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\gamma_1\beta_2)-\gamma_2\beta_1(\beta_2+\delta+\gamma_2))(\gamma_1+\bar{\alpha})} \\
p_{1,1}^{(1)} &= \frac{\beta_1\delta(\delta+\beta_2+\gamma_2)\bar{\alpha}}{(\delta+\beta_1+\gamma_1)((\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-\gamma_1\beta_2)-\gamma_2\beta_1(\beta_2+\delta+\gamma_2))(\gamma_1+\bar{\alpha})}.
\end{aligned}
\tag{2.4}
$$

Similarly, the probabilities for each of the 4 outcomes starting with an infected

female are

$$
\begin{aligned}
p_{0,0}^{(2)} &= \frac{\gamma_2}{\gamma_2+\bar{\alpha}} + \frac{\gamma_1\gamma_2\beta_2\bar{\alpha} + \gamma_2\{(\delta+\gamma_1+\gamma_2)(\delta+\beta_1+\gamma_1) - \gamma_2\beta_1\}\bar{\alpha}}{(\delta+\beta_2+\gamma_2)((\delta+\gamma_1+\gamma_2)(\beta_1+\delta+\gamma_1) - \gamma_2\beta_1) - \gamma_1\beta_2(\beta_1+\delta+\gamma_1))(\gamma_2+\bar{\alpha})} \\
p_{1,0}^{(2)} &= \frac{\gamma_2\delta\beta_2\bar{\alpha}}{(\delta+\beta_2+\gamma_2)((\delta+\gamma_1+\gamma_2)(\beta_1+\delta+\gamma_1) - \gamma_2\beta_1) - \gamma_1\beta_2(\beta_1+\delta+\gamma_1))(\gamma_2+\bar{\alpha})} \\
p_{0,1}^{(2)} &= \frac{(\delta(\delta+\gamma_1+\gamma_2)(\beta_1+\delta+\gamma_1) - \delta\gamma_2\beta_1)\bar{\alpha}}{(\delta+\beta_2+\gamma_2)((\delta+\gamma_1+\gamma_2)(\beta_1+\delta+\gamma_1) - \gamma_2\beta_1) - \gamma_1\beta_2(\beta_1+\delta+\gamma_1))(\gamma_2+\bar{\alpha})} \\
p_{1,1}^{(2)} &= \frac{\beta_2\delta(\delta+\beta_1+\gamma_1)\bar{\alpha}}{(\delta+\beta_2+\gamma_2)((\delta+\gamma_1+\gamma_2)(\beta_1+\delta+\gamma_1) - \gamma_2\beta_1) - \gamma_1\beta_2(\beta_1+\delta+\gamma_1))(\gamma_2+\bar{\alpha})}.
\end{aligned}
\tag{2.5}
$$

We now have explicit formulae for the necessary ingredients for determining an expression for R_0. It was shown in Diekmann, Heesterbeek & Metz (1990) the threshold parameter is equivalent to the dominant eigenvalue of the "next-generation matrix". Now, let us denote the next-generation matrix (NGM) by $K = (k_{ij})$, where k_{ij} represents expected number of infected individuals of type j caused by a single infected individual of type i, where $i, j \in \{1, 2\}$.

$$
K = \begin{pmatrix} k_{11} & k_{12} \\ k_{21} & k_{22} \end{pmatrix} = \begin{pmatrix} p_{1,0}^{(1)} + p_{1,1}^{(1)} & p_{0,1}^{(1)} + p_{1,1}^{(1)} \\ p_{1,0}^{(2)} + p_{1,1}^{(2)} & p_{0,1}^{(2)} + p_{1,1}^{(2)} \end{pmatrix}. \tag{2.6}
$$

Since R_0 is the dominant eigenvalue of the next generation matrix, we obtain that

$$
R_0 = \frac{T + \sqrt{T^2 - 4D}}{2}, \tag{2.7}
$$

where

$$
\begin{aligned}
T &= p_{1,0}^{(1)} + p_{1,1}^{(1)} + p_{0,1}^{(2)} + p_{1,1}^{(2)} \text{ and} \\
D &= (p_{1,0}^{(1)} + p_{1,1}^{(1)})(p_{0,1}^{(2)} + p_{1,1}^{(2)}) - (p_{0,1}^{(1)} + p_{1,1}^{(1)})(p_{1,0}^{(2)} + p_{1,1}^{(2)}).
\end{aligned}
$$

We analyse the expression of R_0 and obtain some interesting results. In the simplest case where there is no difference in infection and recovery rates between the

sexes, i.e. $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$, a simple expression for R_0 is given in Lemma 2.3.1.

Lemma 2.3.1 *For $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$, the reproduction number is*

$$R_0 = p_{1,0}^{(1)} + p_{0,1}^{(1)} + 2p_{1,1}^{(1)} \tag{2.8}$$

Proof Since $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$, we have that

$$p_{1,0}^{(1)} = p_{0,1}^{(2)},\ p_{1,0}^{(2)} = p_{0,1}^{(1)},\ p_{1,1}^{(1)} = p_{1,1}^{(2)}.$$

From (2.7),

$$\begin{aligned}
T^2 - 4D &= (p_{1,0}^{(1)} + p_{1,1}^{(1)})^2 + 2(p_{1,0}^{(1)} + p_{1,1}^{(1)})(p_{0,1}^{(2)} + p_{1,1}^{(2)}) + (p_{0,1}^{(2)} + p_{1,1}^{(2)})^2 \\
&\quad - 4\{(p_{1,0}^{(1)} + p_{1,1}^{(1)})(p_{0,1}^{(2)} + p_{1,1}^{(2)}) - (p_{0,1}^{(1)} + p_{1,1}^{(1)})(p_{1,0}^{(2)} + p_{1,1}^{(2)})\} \\
&= (p_{1,0}^{(1)} + p_{1,1}^{(1)} - p_{0,1}^{(2)} - p_{1,1}^{(2)})^2 + 4(p_{0,1}^{(1)} + p_{1,1}^{(1)})(p_{1,0}^{(2)} + p_{1,1}^{(2)}) \\
T^2 - 4D &= 4(p_{0,1}^{(1)} + p_{1,1}^{(1)})^2 \quad (\because p_{1,0}^{(1)} = p_{0,1}^{(2)},\ p_{1,0}^{(2)} = p_{0,1}^{(1)},\ p_{1,1}^{(1)} = p_{1,1}^{(2)}).
\end{aligned}$$

From equation (2.7), we have

$$\begin{aligned}
R_0 &= \frac{(p_{1,0}^{(1)} + p_{1,1}^{(1)} + p_{1,0}^{(1)} + p_{1,1}^{(1)}) + 2(p_{0,1}^{(1)} + p_{1,1}^{(1)})}{2} \\
&= \frac{2(p_{1,0}^{(1)} + p_{1,1}^{(1)}) + 2(p_{0,1}^{(1)} + p_{1,1}^{(1)})}{2} \\
&= p_{1,0}^{(1)} + p_{1,1}^{(1)} + p_{0,1}^{(1)} + p_{1,1}^{(1)} \\
&= p_{1,0}^{(1)} + p_{0,1}^{(1)} + 2p_{1,1}^{(1)}.
\end{aligned}$$

Thus, Lemma 2.3.1 has been proved.

Lemma 2.3.2 *1. If $p_{1,1}^{(1)} < p_{0,0}^{(1)}$ and $p_{1,1}^{(2)} < p_{0,0}^{(2)}$, then $R_0 < 1$.*
2. If $p_{1,1}^{(1)} > p_{0,0}^{(1)}$ and $p_{1,1}^{(2)} > p_{0,0}^{(2)}$, then $R_0 > 1$.

Proof Consider the case $R_0 < 1$, i.e. $R_0 = \frac{T+\sqrt{T^2-4D}}{2} < 1$. We have that $T^2 - 4D < (2-T)^2$, following by $T^2 - 4D < 4 - 4T + T^2$. As a result, we have that $R_0 < 1$ if and only if $T - D < 1$.

Substitute T and D and after simplification we thus obtain the following conditions,

$$R_0 < 1 \text{ if } p_{1,1}^{(1)}p_{1,1}^{(2)} - p_{0,0}^{(1)}p_{0,0}^{(2)} + p_{1,0}^{(2)}(p_{1,1}^{(1)} - p_{0,0}^{(1)}) + p_{0,1}^{(1)}(p_{1,1}^{(2)} - p_{0,0}^{(2)}) < 0 \tag{2.9}$$

$$R_0 > 1 \text{ if } p_{1,1}^{(1)}p_{1,1}^{(2)} - p_{0,0}^{(1)}p_{0,0}^{(2)} + p_{1,0}^{(2)}(p_{1,1}^{(1)} - p_{0,0}^{(1)}) + p_{0,1}^{(1)}(p_{1,1}^{(2)} - p_{0,0}^{(2)}) > 0. \tag{2.10}$$

It is obvious that condition (2.9) is satisfied if $p_{1,1}^{(1)} < p_{0,0}^{(1)}$ and $p_{1,1}^{(2)} < p_{0,0}^{(2)}$, whilst $p_{1,1}^{(1)} > p_{0,0}^{(1)}$ and $p_{1,1}^{(2)} > p_{0,0}^{(2)}$ will ensure that (2.10) is satisfied. The lemma is thus proved.

Lemma 2.3.2 has an intuitive interpretation that if for both males and females the probability of having no offspring is greater than the probability of having two offspring, the disease will die out. In contrast, we have the following surprising result.

Theorem 2.3.3 *The reproduction number remains unchanged when swapping the values of β_1 and β_2, whilst keeping the other parameters fixed.*

Proof Let $a, b \in \mathbb{R}^+$. Consider two epidemic processes. Fix α, δ, γ_1 and γ_2 to be the same in both epidemic processes.

For epidemic model 1, set parameters $\beta_1 = a, \beta_2 = b$.

For epidemic model 2, set parameters $\beta_1 = b, \beta_2 = a$.

Let M be the next generation matrix for epidemic model 1 and let M^* be the next generation matrix for epidemic model 2. Then

$$M = \begin{pmatrix} p_{1,0}^{(1)} + p_{1,1}^{(1)} & p_{0,1}^{(1)} + p_{1,1}^{(1)} \\ p_{1,0}^{(2)} + p_{1,1}^{(2)} & p_{0,1}^{(2)} + p_{1,1}^{(2)} \end{pmatrix}, \quad M^* = \begin{pmatrix} q_{1,0}^{(1)} + q_{1,1}^{(1)} & q_{0,1}^{(1)} + q_{1,1}^{(1)} \\ q_{1,0}^{(2)} + q_{1,1}^{(2)} & q_{0,1}^{(2)} + q_{1,1}^{(2)} \end{pmatrix},$$

Note that $q_{i,j}^{(k)}$ is the probability of having i male offspring and j female offspring using the parameter set from epidemic model 2.

Recall that if we have two matrices $A_2 = (a_{ij})$ and $B_2 = (b_{ij})$ such that $a_{11} = b_{11}$, $a_{22} = b_{22}$ and $a_{12}a_{21} = b_{12}b_{21}$, then

$$\begin{aligned} det(A - \lambda I) &= (a_{11} - \lambda)(a_{22} - \lambda) - a_{12}a_{21} \\ &= (b_{11} - \lambda)(b_{22} - \lambda) - b_{12}b_{21} \\ &= det(B - \lambda I). \end{aligned} \tag{2.11}$$

That is, A and B have the same eigenvalues and hence, the same maximal eigenvalue.

Let R and R^* denote the maximal eigenvalues of matrices M and M^*, respectively. To show that $R = R^*$, we show that

$$p_{1,0}^{(1)} + p_{1,1}^{(1)} = q_{1,0}^{(1)} + q_{1,1}^{(1)} \tag{2.12}$$

$$p_{1,0}^{(2)} + p_{1,1}^{(2)} = q_{1,0}^{(2)} + q_{1,1}^{(2)} \tag{2.13}$$

$$(p_{0,1}^{(1)} + p_{1,1}^{(1)})(p_{1,0}^{(2)} + p_{1,1}^{(2)}) = (q_{1,0}^{(1)} + q_{1,1}^{(1)})(q_{1,0}^{(2)} + q_{1,1}^{(2)}). \tag{2.14}$$

The denominator of $p_{i,j}^{(1)}$ for $i, j \in \{0, 1\}$ is

$((\delta + \gamma_1 + \gamma_2)(b + \delta + \gamma_2)(\delta + a + \gamma_1) - \gamma_1 b(\delta + a + \gamma_1) - \gamma_2 a(b + \delta + \gamma_2))(\gamma_1 + \bar{\alpha})$.

The denominator of $q_{i,j}^{(1)}$ for $i, j \in \{0, 1\}$ is

$((\delta + \gamma_1 + \gamma_2)(a + \delta + \gamma_2)(\delta + b + \gamma_1) - \gamma_1 a(\delta + b + \gamma_1) - \gamma_2 b(a + \delta + \gamma_2))(\gamma_1 + \bar{\alpha})$.

It is straightforward to show that these two denominators are the same. Let us denote the denominator of $p_{i,j}^{(1)}$ and $q_{i,j}^{(1)}$ by L_1. Then

$$\begin{aligned}
\left(\frac{L_1}{\bar{\alpha}}\right)(p_{1,0}^{(1)} + p_{1,1}^{(1)}) &= \delta(\delta+\gamma_1+\gamma_2)(b+\delta+\gamma_2) - \delta\gamma_1 b + a\delta(\delta+b+\gamma_2) \\
\left(\frac{L_1}{\bar{\alpha}}\right)(q_{1,0}^{(1)} + q_{1,1}^{(1)}) &= \delta(\delta+\gamma_1+\gamma_2)(a+\delta+\gamma_2) - \delta\gamma_1 a + b\delta(\delta+a+\gamma_2) \\
\left(\frac{L_1}{\bar{\alpha}}\right)(p_{1,0}^{(1)} + p_{1,1}^{(1)} - (q_{1,0}^{(1)} + q_{1,1}^{(1)})) &= \delta(\delta+\gamma_1+\gamma_2)(b-a) - \delta\gamma_1(b-a) + a\delta(\delta+b+\gamma_2) \\
&\quad - b\delta(\delta+a+\gamma_2) \\
&= (\delta^2 b - \delta^2 b) + (\delta^2 a - \delta^2 a) + (\delta\gamma_1 b - \delta\gamma_1 b) \\
&\quad + (\delta\gamma_2 b - \delta\gamma_2 b) + (\delta\gamma_2 a - \delta\gamma_2 a) + (\delta\gamma_1 a - \delta\gamma_1 a) \\
&\quad + (ab\delta - ab\delta) = 0.
\end{aligned}$$

Hence, $p_{1,0}^{(1)} + p_{1,1}^{(1)} = q_{1,0}^{(1)} + q_{1,1}^{(1)}$. Similarly, we have

$$p_{1,0}^{(2)} + p_{1,1}^{(2)} = q_{1,0}^{(2)} + q_{1,1}^{(2)}.$$

Let L_2 denote the denominator of $p_{i,j}^{(2)}$ and $q_{i,j}^{(2)}$, thus

$$\begin{aligned}
\frac{L_1 L_2}{\bar{\alpha}^2}(p_{0,1}^{(1)} + p_{1,1}^{(1)})(p_{1,0}^{(2)} + p_{1,1}^{(2)}) &= (\delta\gamma_1 a + a\delta(\delta+b+\gamma_2))(\delta\gamma_2 b + b\delta(\delta+a+\gamma_1)) \\
&= \delta^2 a(\gamma_1+\delta+b+\gamma_2)b(\gamma_2+a+\delta+\gamma_1) \\
\frac{L_1 L_2}{\bar{\alpha}^2}(q_{10}^{(1)} + q_{1,1}^{(1)})(q_{1,0}^{(2)} + q_{1,1}^{(2)}) &= (\delta\gamma_1 b + b\delta(\delta+a+\gamma_2))(\delta\gamma_2 a + a\delta(\delta+b+\gamma_1)) \\
&= \delta^2 a(\gamma_1+\delta+b+\gamma_2)b(\gamma_2+a+\delta+\gamma_1).
\end{aligned}$$

Hence $(p_{0,1}^{(1)} + p_{1,1}^{(1)})(p_{1,0}^{(2)} + p_{1,1}^{(2)}) = (q_{10}^{(1)} + q_{1,1}^{(1)})(q_{1,0}^{(2)} + q_{1,1}^{(2)})$, as required.

The theorem then follows by (2.11).

We also obtain the same result from swapping the values of γ_1 and γ_2, keeping the other parameters fixed, which can be proved in a similar manner to Theorem

2.3.3.

We have studied the threshold parameter, R_0, which indicates whether or not the disease can persist in the population. If $R_0 \leq 1$, then the disease will go extinct with probability 1. If $R_0 > 1$, the probability that the disease will go extinct is positive but less than one. We are interested in determining this probability of extinction in Section 2.4.

2.4 The probability of extinction

The behaviour of the disease in terms of extinction follows branching processes characteristic, in which it will either grow large or quickly become extinct. In a finite population, the epidemic will always go extinct, but here, we are interested in determining the probability of the disease taking off first before going extinct. Therefore, in this section, we are interested in studying the probability of extinction, since then, the probability of a major epidemic outbreak occurring is given by 1 - the probability of extinction. Then, we have that the probability of a major outbreak is 0 if $R_0 \leq 1$, and if $R_0 > 1$, it is greater than 0 and is equal to 1 - the probability of extinction.

In order to determine the probability of extinction, we employ 2-type branching processes. The expected numbers of offspring produced in each generation calculated in the previous section, are parameters used in forming the probability generating functions. According to Theorem 7.1, page 41 in Harris (1963), the probability of extinction is the unique non-negative solution less than 1 of equations of the probability generating functions. In particular, recall that $p_{i,j}^{(k)}$ is the probability of having i infected male individuals and j infected female individuals, starting from 1 infected individual of type k. We define the probability generating

functions for the number of offspring of a type k individual as

$$g_k(s_1, s_2) = \sum_{j,i} p_{i,j}^{(k)} s_1^i s_2^j, \quad k = 1, 2, \tag{2.15}$$

where

$$g_1(s_1, s_2) = p_{0,0}^{(1)} + p_{1,0}^{(1)} s_1 + p_{0,1}^{(1)} s_2 + p_{1,1}^{(1)} s_1 s_2$$
$$g_2(s_1, s_2) = p_{0,0}^{(2)} + p_{1,0}^{(2)} s_1 + p_{0,1}^{(2)} s_2 + p_{1,1}^{(2)} s_1 s_2.$$

Let π_i denote the probability of extinction given that a single infected individual of type i is introduced into the population. The probabilities of extinction, π_1 and π_2, are the smallest non-negative root of the equation

$$(g_1(\pi_1, \pi_2), g_2(\pi_1, \pi_2)) = (\pi_1, \pi_2). \tag{2.16}$$

Due to the complex formulae for $p_{ij}^{(k)}$, it is not possible to obtain explicit expressions for π_1 and π_2. Therefore, we focus on a numerical analysis. However, analytical progress can be made in the case, $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$. We obtain the following lemma.

Lemma 2.4.1 *If $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$, then $\pi_1 = \pi_2$ and* $\pi_1 = \min\left\{1, \dfrac{p_{0,0}^{(1)}}{p_{1,1}^{(1)}}\right\}$

Proof From equation (2.16), we have

$$\pi_1 = p_{0,0}^{(1)} + p_{1,0}^{(1)} \pi_1 + p_{0,1}^{(1)} \pi_2 + p_{1,1}^{(1)} \pi_1 \pi_2, \tag{2.17}$$
$$\pi_2 = p_{0,0}^{(2)} + p_{1,0}^{(2)} \pi_1 + p_{0,1}^{(2)} \pi_2 + p_{1,1}^{(2)} \pi_1 \pi_2. \tag{2.18}$$

Since $p_{0,0}^{(1)} = p_{0,0}^{(2)},\ p_{1,0}^{(1)} = p_{0,1}^{(2)},\ p_{1,0}^{(2)} = p_{0,1}^{(1)},\ p_{1,1}^{(1)} = p_{1,1}^{(2)}$ (because $\beta_1 = \beta_2$, $\gamma_1 = \gamma_2$)

and by subtracting equation (2.17) and (2.18), we obtain

$$\pi_1 - \pi_2 = p_{1,0}^{(1)}\pi_1 - p_{1,0}^{(1)}\pi_2 + p_{0,1}^{(1)}\pi_2 - p_{0,1}^{(1)}\pi_1 \tag{2.19}$$

$$(1 - p_{1,0}^{(1)} + p_{0,1}^{(1)})\pi_1 = (p_{0,1}^{(1)} - p_{1,0}^{(1)} + 1)\pi_2. \tag{2.20}$$

$$\text{Hence,} \qquad \pi_1 = \pi_2. \tag{2.21}$$

Now, we want to show that $\pi_1 = \min\left\{1, \dfrac{p_{0,0}^{(1)}}{p_{1,1}^{(1)}}\right\}$. Let $\pi = \pi_1 = \pi_2$. From (2.17), we have that

$$p_{0,0}^{(1)} + (p_{1,0}^{(1)} + p_{0,1}^{(1)} - 1)\pi + p_{1,1}^{(1)}\pi^2 = 0. \tag{2.22}$$

Recall that $p_{0,0}^{(1)} + p_{1,0}^{(1)} + p_{0,1}^{(1)} + p_{1,1}^{(1)} = 1$, then $p_{1,0}^{(1)} + p_{0,1}^{(1)} - 1 = -(p_{0,0}^{(1)} + p_{1,1}^{(1)})$.

Hence, equation (2.22) becomes

$$p_{0,0}^{(1)} - (p_{0,0}^{(1)} + p_{1,1}^{(1)})\pi + p_{1,1}^{(1)}\pi^2 = 0$$

$$(\pi - 1)(p_{1,1}^{(1)}\pi - p_{0,0}^{(1)}) = 0.$$

As a result, $\pi = 1$ or $\pi = \dfrac{p_{0,0}^{(1)}}{p_{1,1}^{(1)}}$. Since π is the smallest non-negative root, then $\pi = \min\left\{1, \dfrac{p_{0,0}^{(1)}}{p_{1,1}^{(1)}}\right\} = \pi_1$. Thus, the Lemma is proved.

What if there is a difference in either infection or recovery rate between males and females? As we observed, a higher infection rate will increase the expected number of offspring, giving a smaller probability of extinction. Whilst, a higher recovery rate will reduce the expected number of offspring, giving a higher probability of extinction. As such, we have the following lemma which supports the above intuition.

Lemma 2.4.2 *(i) If $\beta_1 = \beta_2$ and $\gamma_1 < \gamma_2$, then $\pi_1 < \pi_2$.*

(ii) If $\beta_1 > \beta_2$ and $\gamma_1 = \gamma_2$, then $\pi_1 < \pi_2$.

Proof (i) Let ϕ denote the probability of extinction starting with a relationship containing both an infected male and an infected female. Let q_i denote the probability of extinction starting with an individual of type i before infecting an individual of the opposite sex. Let $M_I(F_I)$, $M_S(F_S)$ represent an infected and a susceptible male(female), respectively.

Then Figure 2.2 shows the initial stages of a sexual network starting from a single infected male.

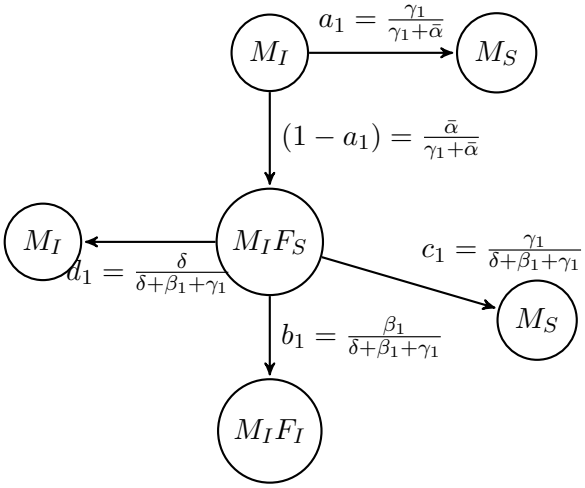


Figure 2.2: Sexual network diagram

From the diagram, we have

$$q_1 = a_1 + (1-a_1)(c_1 + d_1 q_1) = \frac{\gamma_1\delta + \beta_1\gamma_1 + \bar{\alpha}\gamma_1 + \gamma_1^2}{\bar{\alpha}\beta_1 + \delta\gamma_1 + \beta_1\gamma_1 + \bar{\alpha}\gamma_1 + \gamma_1^2} \tag{2.23}$$

$$1 - q_1 = (1-a_1)(b_1 + d_1(1-q_1)) = \frac{\bar{\alpha}\beta_1}{\bar{\alpha}\beta_1 + \delta\gamma_1 + \beta_1\gamma_1 + \bar{\alpha}\gamma_1 + \gamma_1^2}. \tag{2.24}$$

Similarly, we have

$$q_2 = \frac{\gamma_2\delta + \beta_2\gamma_2 + \bar{\alpha}\gamma_2 + \gamma_2^2}{\bar{\alpha}\beta_2 + \delta\gamma_2 + \beta_2\gamma_2 + \bar{\alpha}\gamma_2 + \gamma_2^2} \tag{2.25}$$

$$1 - q_2 = \frac{\bar{\alpha}\beta_2}{\bar{\alpha}\beta_2 + \delta\gamma_2 + \beta_2\gamma_2 + \bar{\alpha}\gamma_2 + \gamma_2^2} \tag{2.26}$$

We can therefore write the probability of extinction as

$$\pi_1 = q_1 + (1 - q_1)\phi \tag{2.27}$$

$$\pi_2 = q_2 + (1 - q_2)\phi. \tag{2.28}$$

Let

$$f(\beta, \gamma) = \frac{\gamma\delta + \beta\gamma + \bar{\alpha}\gamma + \gamma^2}{\bar{\alpha}\beta + \delta\gamma + \beta\gamma + \bar{\alpha}\gamma + \gamma^2}, \tag{2.29}$$

where $\beta > 0, \gamma > 0$, and $\pi(\beta, \gamma) = f(\beta, \gamma) + (1 - f(\beta, \gamma))\phi$. Then $q_1 = f(\beta_1, \gamma_1)$ and $q_2 = f(\beta_2, \gamma_2)$. We can write the probabilities of extinction as

$$\pi_1 = \pi(\beta_1, \gamma_1) \text{ and } \pi_2 = \pi(\beta_2, \gamma_2).$$

By (2.29), we have

$$\frac{\partial f(\beta, \gamma)}{\partial \gamma} = \frac{\bar{\alpha}\beta(\bar{\alpha} + \delta + \beta + 2\gamma)}{(\bar{\alpha}\beta + \delta\gamma + \beta\gamma + \bar{\alpha}\gamma + \gamma^2)^2} > 0,$$

for every $\beta > 0$. Therefore, $f(\beta, \gamma)$ is an increasing function in γ. If $\gamma_1 < \gamma_2$, and $\beta_1 = \beta_2 = \beta$ then $f(\beta, \gamma_1) < f(\beta, \gamma_2)$. Thus,

$$\pi(\beta, \gamma_1) = f(\beta, \gamma_1) + (1 - f(\beta, \gamma_1))\phi < f(\beta, \gamma_2) + (1 - f(\beta, \gamma_2))\phi = \pi(\beta, \gamma_2). \tag{2.30}$$

That is, $\pi_1 = \pi(\beta_1, \gamma_1) < \pi(\beta_2, \gamma_2) = \pi_2$ as required. Note that $0 < \phi < 1$.

(ii) Similarly,

$$\frac{\partial f(\beta, \gamma)}{\partial \beta} = -\frac{\gamma\bar{\alpha}(\gamma + \delta + \bar{\alpha})}{(\bar{\alpha}\beta + \gamma\delta + \beta\gamma + \bar{\alpha}\gamma + \gamma^2)^2} < 0.$$

Hence, $f(\beta_1, \gamma) < f(\beta_2, \gamma)$ for every $\beta_1 > \beta_2$ and $\gamma > 0$. Thus,

$$\pi(\beta_1, \gamma) = f(\beta_1, \gamma) + (1 - f(\beta_1, \gamma))\phi > f(\beta_2, \gamma) + (1 - f(\beta_2, \gamma))\phi = \pi(\beta_2, \gamma), \tag{2.31}$$

for every $\beta_1 > \beta_2$ and $\gamma > 0$. Therefore, if $\beta_1 > \beta_2$ and $\gamma_1 = \gamma_2$, then $\pi_1 = \pi(\beta_1, \gamma_1) < \pi(\beta_2, \gamma_2) = \pi_2$ as required. Following Lemma 2.4.2, we have Corollary 2.4.3.

Corollary 2.4.3 *If $\beta_1 \geq \beta_2$ and $\gamma_1 \leq \gamma_2$, then $\pi_1 \leq \pi_2$.*

Proof From Lemma 2.4.1, we have that $\pi_1 = \pi_2$ if $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$. Now, assume that $\beta_1 > \beta_2$ and $\gamma_1 < \gamma_2$. Then,

$$\begin{aligned} \pi_2 = \pi(\beta_2, \gamma_2) &> \pi(\beta_2, \gamma_1) && \text{(From (2.30) and } \gamma_1 < \gamma_2) \\ &> \pi(\beta_1, \gamma_1) = \pi_1 && \text{(From (2.31) and } \beta_1 > \beta_2) \end{aligned}$$

Therefore, if $\beta_1 \geq \beta_2$ and $\gamma_1 \leq \gamma_2$, then $\pi_1 \leq \pi_2$. The corollary is then proved.

We have explored the relationship between infection and recovery rates and the probability of extinction. However, infection is also controlled by the relationship formation rate, $\bar{\alpha}$. If the rate at which relationships are formed is low, it will lower the chance that the disease is transmitted even if the infection rate is high. This is because the infection occurs only within a relationship. At the same time, if the relationship formation rate is high, but the breakup rate is very small, the chance that an infected individual will infect new partners is small. With high breakup rate and low relationship formation rate, individuals tend to stay single rather than forming relationships, leading to less chance of individuals infecting each other. For this reason, to observe the effect of relationship dynamics on the probability of extinction, we consider reparameterising the model in terms of δ and $F = \frac{\bar{\alpha}}{\delta}$, the relative rate of formation to breakup. Lemma 2.4.4 states

the relationship between the relationship breakup rate (δ) and the probability of extinction.

Lemma 2.4.4 *If $\beta_1 = \beta_2 = \beta$ and $\gamma_1 = \gamma_2 = \gamma$, for fixed $F = \frac{\bar{\alpha}}{\delta}$, the probability of extinction is decreasing as δ increases.*

Proof Since $\beta_1 = \beta_2 = \beta$ and $\gamma_1 = \gamma_2 = \gamma$, it suffices to look at the final outcomes without considering the type of individuals. In other words, we consider the number of offspring produced from 1 infected individual.
Let H_i be the probability of having i offspring starting at the relationship stage with 1 infected individual. Let P_i be the probability of having i offspring starting with 1 single infected individual.

The probabilities of having 0, 1 and 2 offspring can be expressed as

$$P_0 = \frac{\gamma}{\bar{\alpha}+\gamma} + \frac{\bar{\alpha}}{\bar{\alpha}+\gamma}H_0, \qquad P_1 = \frac{\bar{\alpha}}{\bar{\alpha}+\gamma}H_1, \qquad P_2 = \frac{\bar{\alpha}}{\bar{\alpha}+\gamma}H_2 \tag{2.32}$$

Consider the following diagram, Figure 2.3

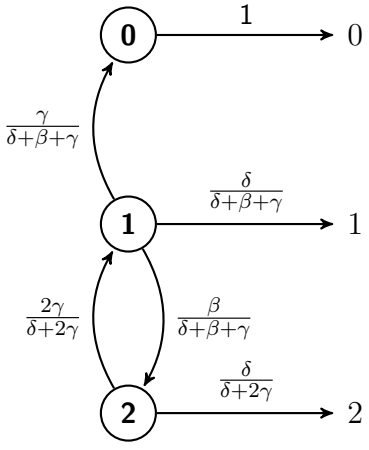


Figure 2.3: Network diagram with nodes representing the number of infected offspring in a relationship, and arrows representing the transition probabilities.

From Figure 2.3, we have an expression of H_i as follows

$$\begin{aligned} H_0 &= \frac{\gamma}{\delta+\beta+\gamma} + (\frac{\beta}{\delta+\beta+\gamma})(\frac{2\gamma}{2\gamma+\delta})H_0 \\ H_1 &= \frac{\delta}{\delta+\beta+\gamma} + (\frac{\beta}{\delta+\beta+\gamma})(\frac{2\gamma}{2\gamma+\delta})H_1 \\ H_2 &= \frac{\beta}{\delta+\beta+\gamma}\frac{\delta}{2\gamma+\delta} + (\frac{\beta}{\delta+\beta+\gamma})(\frac{2\gamma}{2\gamma+\delta})H_2. \end{aligned} \tag{2.33}$$

Hence,

$$\begin{aligned} H_0 &= \frac{\gamma(\delta+2\gamma)}{\delta^2+\delta\beta+3\delta\gamma+2\gamma^2} \\ H_1 &= \frac{\delta(\delta+2\gamma)}{\delta^2+\delta\beta+3\delta\gamma+2\gamma^2} \\ H_2 &= \frac{\beta\delta}{\delta^2+\delta\beta+3\delta\gamma+2\gamma^2}. \end{aligned} \tag{2.34}$$

The probability of extinction π satisfies

$$\pi = P_0 + P_1\pi + P_2\pi^2, \tag{2.35}$$

$$\pi = \min\left\{1, \frac{P_0}{P_2}\right\}. \tag{2.36}$$

From (2.32), (2.34) and substituting in $F\delta$ for $\bar{\alpha}$, we have

$$P_0 = \frac{\gamma}{\delta F+\gamma} + \frac{(\delta F)\gamma(\delta+2\gamma)}{(\delta F+\gamma)(\delta^2+\delta\beta+3\delta\gamma+2\gamma^2)} \tag{2.37}$$

$$P_2 = \left(\frac{\delta F}{\delta F+\gamma}\right)\left(\frac{\beta\delta}{\delta^2+\delta\beta+3\delta\gamma+2\gamma^2}\right). \tag{2.38}$$

Hence, if $\pi = \frac{P_0}{P_2}$, we have

$$\pi = \frac{P_0}{P_2} = \frac{\gamma((1+F)\delta^2+(\beta+3\gamma+2\gamma F)\delta+2\gamma^2)}{\delta^2 F\beta} = \frac{A\delta^2+B\delta+C}{\delta^2 F\beta}, \tag{2.39}$$

where A, B and C are constants in β, γ and F. Thus the gradient of π with respect

to δ is

$$\frac{\partial \pi}{\partial \delta} = -\frac{B\delta + 2C}{\delta^3 F\beta} < 0. \tag{2.40}$$

Therefore π is a decreasing function in δ. In other words, the probability of extinction is decreasing in δ, for fixed $F = \frac{\bar{\alpha}}{\delta}$.

We have now studied the behaviour of the epidemic in its early stages. Now, we are interested in the case moving away from the early stages to the case where the disease persists. The key quantity of interest here is the endemic level.

2.5 Endemic Level

The behaviour of the epidemic in its initial stages has been described using the branching process approximation, in which it indicates the ability of the epidemic taking off in the population; whether or not a major outbreak of the disease can occur and the probability such an outbreak occurs. When an outbreak of the disease occurs, there are two possible scenarios. The first scenario being that a few susceptible individuals become infected and the disease dies out quickly (time to extinction is short), which is covered by branching process approximation. The second scenario is that there is a large outbreak, namely, many individuals become infected, and the epidemic persists in the population for some period of time before dying out (time to extinction is long). When the second scenario occurs, the disease has become endemic in the population. The outbreak of the disease when it is endemic is defined by the proportion of infected individuals at equilibrium, the so-called *endemic level*. Here, we are interested in studying and answering questions arising in the second scenario. What is the proportion of infected individuals at the endemic, in other words, what is the endemic level? What is the expected time to extinction of the epidemic given that the epidemic starts at the endemic level?

Since we are interested in the case where there is an epidemic outbreak, throughout this section, we assume that the epidemic is supercritical ($R_0 > 1$). When the population size is sufficiently large, especially for multi-dimensional Markov processes, the stochastic model becomes difficult to work with, from both an analytical and a computational point of view. However, according to Theorem 1.3.2 in Chapter 1, for large N, sequences of Markov processes converge to solutions of ordinary differential equations. Therefore, it is useful to approximate the stochastic model by a deterministic approximation, and to study the behaviour of the deterministic approximation. This will be described in detail in Section 2.5.1.

Another question arising here is what are the fluctuations about the endemic level? An Ornstein-Uhlenbeck (OU) approximation is constructed to describe fluctuations about a mean. The Ornstein-Uhlenbeck process is stationary, Gaussian and Markovian. As Theorem 1.3.3 stated, using the central limit theorem, for large N, the fluctuations of the stochastic process about the deterministic limit converges weakly to a diffusion process. If an equilibrium point is chosen as the initial value of the deterministic approximation, the fluctuations of the stochastic process can be approximated by an Ornstein-Uhlenbeck process (Pollett (2001)). The construction of the Ornstein-Uhlenbeck process will be described in detail in Section 2.5.2.

For epidemic models in general, typical behaviour is that the model will converge towards a disease free equilibrium at 0 in the case that $R_0 \leq 1$, or some non-zero fixed point steady state when $R_0 > 1$. This is according to the existence and uniqueness properties of the ordinary differential equations (Atkinson et al. (2009), Chapter 1). Whereas, in terms of stochastic behaviour, even if $R_0 > 1$ but the process starts with only one (or a few) initial infectives in a large population, it is possible that the epidemic never takes off. The deterministic models can not capture this behaviour. Therefore, the deterministic model is not informative for determining the mean time to extinction. In Section 2.6, we estimate the mean

time to extinction using simulations of the stochastic process.

2.5.1 Deterministic Model

The population is classified into 8 disjoint classes according to sex, disease status and relationship status. The parameters of the model are as above. Let $\boldsymbol{X(t)}, \boldsymbol{Y(t)}$ and $\boldsymbol{Z(t)}$ represent the number of males, females and couples at time t, respectively, namely,

$$\boldsymbol{X(t)} = (X_0(t), X_1(t)), \quad \boldsymbol{Y(t)} = (Y_0(t), Y_1(t)), \quad \boldsymbol{Z(t)} = (Z_{00}(t), Z_{10}(t), Z_{01}(t), Z_{11}(t)),$$

where

$X_0(t)$ is the number of susceptible males at time t

$X_1(t)$ is the number of infected males at time t,

$Y_0(t)$ is the number of susceptible females at time t,

$Y_1(t)$ is the number of infected females at time t,

$Z_{00}(t)$ is the number of uninfected couples at time t,

$Z_{10}(t)$ is the number of couples with an infected male only at time t,

$Z_{01}(t)$ is the number of couples with an infected female only at time t,

$Z_{11}(t)$ is the number of infected couples at time t.

Also, $X_0(t) + X_1(t) + Y_0(t) + Y_1(t) + 2Z_{00}(t) + 2Z_{10}(t) + 2Z_{01}(t) + 2Z_{11}(t) = N$, where N is the total population. Let $I(t)$ denote the number of infected individuals at time t, i.e. $I(t) = X_1(t) + Y_1(t) + Z_{10}(t) + Z_{01}(t) + 2Z_{11}(t)$.

Now we have a jump Markov process with state space $(\boldsymbol{X(t)}, \boldsymbol{Y(t)}, \boldsymbol{Z(t)})$. To apply the functional law of large number, we define for $t \geq 0$

$$(\boldsymbol{X_N(t)}, \boldsymbol{Y_N(t)}, \boldsymbol{Z_N(t)}) = \left(\frac{\mathbf{X(t)}}{N}, \frac{\mathbf{Y(t)}}{N}, \frac{\mathbf{Z(t)}}{N}\right),$$
$$\boldsymbol{I}_N(t) = \frac{I(t)}{N}.$$

According to Kurtz (1970), the stochastic model converges to the deterministic model, for $N \to \infty$, namely,

$$\lim_{N \longrightarrow \infty} (\boldsymbol{X_N(t)}, \boldsymbol{Y_N(t)}, \boldsymbol{Z_N(t)}) = (\boldsymbol{x(t)}, \boldsymbol{y(t)}, \boldsymbol{z(t)}),$$

where $(\boldsymbol{x(t)}, \boldsymbol{y(t)}, \boldsymbol{z(t)}) = (x_0(t), x_1(t), y_0(t), y_1(t), z_{00}(t), z_{01}(t), z_{10}(t), z_{11}(t))$ is a deterministic counterpart to $(\boldsymbol{X}_N(t), \boldsymbol{Y}_N(t), \boldsymbol{Z}_N(t))$. The deterministic model satisfies the following ordinary differential equations (ODEs),

$$\begin{aligned}
\frac{dx_0(t)}{dt} &= \gamma_1 x_1(t) + \delta(z_{01}(t) + z_{00}(t)) - \alpha x_0(t)(y_1(t) + y_0(t)),\\
\frac{dx_1(t)}{dt} &= -\gamma_1 x_1(t) + \delta(z_{11}(t) + z_{10}(t)) - \alpha x_1(t)(y_1(t) + y_0(t)),\\
\frac{dy_0(t)}{dt} &= \gamma_2 y_1(t) + \delta(z_{10}(t) + z_{00}(t)) - \alpha y_0(t)(x_1(t) + x_0(t)),\\
\frac{dy_1(t)}{dt} &= -\gamma_2 y_1(t) + \delta(z_{11}(t) + z_{01}(t)) - \alpha y_1(t)(x_1(t) + x_0(t)),\\
\frac{dz_{00}(t)}{dt} &= \gamma_1 z_{10}(t) + \gamma_2 z_{01}(t) + \alpha y_0(t) x_0(t) - \delta z_{00}(t),\\
\frac{dz_{10}(t)}{dt} &= \gamma_2 z_{11}(t) - \beta_1 z_{10}(t) - \gamma_1 z_{10}(t) + \alpha x_1(t) y_0(t) - \delta z_{10}(t),\\
\frac{dz_{01}(t)}{dt} &= \gamma_1 z_{11}(t) - \gamma_2 z_{01}(t) - \beta_2 z_{01}(t) + \alpha x_0(t) y_1(t) - \delta z_{01}(t),\\
\frac{dz_{11}(t)}{dt} &= \beta_1 z_{10}(t) + \beta_2 z_{01}(t) - \gamma_1 z_{11}(t) - \gamma_2 z_{11}(t) + \alpha y_1(t) x_1(t) - \delta z_{11}(t),
\end{aligned} \tag{2.41}$$

and,

$$n(t) = x_0(t) + x_1(t) + y_0(t) + y_1(t) + 2z_{00}(t) + 2z_{01}(t) + 2z_{10}(t) + 2z_{11}(t) = 1. \tag{2.42}$$

Note that, the numbers of males and females are assumed to be equal. Therefore,

we have the following condition

$$x_0(t) + x_1(t) = y_0(t) + y_1(t). \tag{2.43}$$

The endemic level is a solution of the simultaneous non-linear equations obtained by finding the stationary points to the system of differential equations defined above, with initial values $\boldsymbol{x(0)}, \boldsymbol{y(0)}, \boldsymbol{z(0)}$ where $0 \leq x_i(0), y_i(0), z_{ij}(0) \leq 1$ and $i, j \in \{0, 1\}$. We can reduce the system (2.41) to 6 equations. From (2.42) and (2.43), we have that

$$x_0(t) = 0.5 - (z_{00}(t) + z_{10}(t) + z_{01}(t) + z_{11}(t) + x_1(t)), \tag{2.44}$$

$$y_0(t) = 0.5 - (z_{00}(t) + z_{10}(t) + z_{01}(t) + z_{11}(t) + y_1(t)). \tag{2.45}$$

Substituting (2.44) and (2.45) in (2.41), we have the following system of equations

$$\begin{aligned}
\frac{dx_1(t)}{dt} &= -\gamma_1 x_1(t) + \delta(z_{11}(t) + z_{10}(t)) - \alpha x_1(t) G_3(t), \\
\frac{dy_1(t)}{dt} &= -\gamma_2 y_1(t) + \delta(z_{11}(t) + z_{01}(t)) - \alpha y_1(t) G_3(t), \\
\frac{dz_{00}(t)}{dt} &= \gamma_1 z_{10}(t) + \gamma_2 z_{01}(t) + \alpha G_1(t) G_2(t) - \delta z_{00}(t), \\
\frac{dz_{10}(t)}{dt} &= \gamma_2 z_{11}(t) - \beta_1 z_{10}(t) - \gamma_1 z_{10}(t) + \alpha x_1(t) G_2(t) - \delta z_{10}(t), \\
\frac{dz_{01}(t)}{dt} &= \gamma_1 z_{11}(t) - \gamma_2 z_{01}(t) - \beta_2 z_{01}(t) + \alpha G_1(t) y_1(t) - \delta z_{01}(t), \\
\frac{dz_{11}(t)}{dt} &= \beta_1 z_{10}(t) + \beta_2 z_{01}(t) - \gamma_1 z_{11}(t) - \gamma_2 z_{11}(t) + \alpha y_1(t) x_1(t) - \delta z_{11}(t)
\end{aligned} \tag{2.46}$$

where

$$G_1(t) = 0.5 - (z_{00}(t) + z_{10}(t) + z_{01}(t) + z_{11}(t) + x_1(t)), \tag{2.47}$$

$$G_2(t) = 0.5 - (z_{00}(t) + z_{10}(t) + z_{01}(t) + z_{11}(t) + y_1(t)), \tag{2.48}$$

$$G_3(t) = 0.5 - (z_{00}(t) + z_{10}(t) + z_{01}(t) + z_{11}(t)). \tag{2.49}$$

Equation (2.46) will be studied in detail to explore the endemic equilibrium and fluctuations about the endemic equilibrium.

2.5.1.1 Existence of disease-free equilibrium state (E_f)

Theorem 2.5.1 *A disease-free equilibrium state of the model in (2.41) exists at the point*

$$E_f = (x_0^*, x_1^*, y_0^*, y_1^*, z_{00}^*, z_{10}^*, z_{01}^*, z_{11}^*) = (h, 0, h, 0, \frac{\alpha h^2}{\delta}, 0, 0, 0),$$
$$\textit{where} \quad h = \frac{-\delta + \sqrt{\delta(\delta + 2\alpha)}}{2\alpha}.$$

Proof Assume that $(x_0^*, x_1^*, y_0^*, y_1^*, z_{00}^*, z_{10}^*, z_{01}^*, z_{11}^*)$ is the solution to the system in (2.41). At the disease-free equilibrium, we have that

$$x_1^* = y_1^* = z_{10}^* = z_{01}^* = z_{11}^* = 0.$$

Then, the system of equations in (2.41) has only one equation remaining, which is

$$\delta z_{00}^* - \alpha x_0^* y_0^* = 0. \tag{2.50}$$

Equation (2.42) becomes

$$2z_{00}^* + x_0^* + y_0^* = 1. \tag{2.51}$$

From (2.50), we immediately have that $z_{00}^* = \frac{\alpha}{\delta} x_0^* y_0^*$. Recall that we assume at the start the number of males and females are equal, the proportion of the population in relationships (and single) remain constant. Therefore, at equilibrium, $x_0^* = y_0^*$. Hence, the disease - free equilibrium exists at $(x_0^*, 0, x_0^*, 0, \frac{\alpha}{\delta} x_0^{*2}, 0, 0, 0)$.

Substituting $z_{00}^* = \frac{\alpha}{\delta} x_0^{*2}$ and $x_0^* = y_0^*$ in (2.51), we have

$$2\frac{\alpha}{\delta}x_0^{*2} + 2x_0^* = 1$$
$$2\frac{\alpha}{\delta}x_0^{*2} + 2x_0^* - 1 = 0.$$

Hence, $x_0^* = \dfrac{-\delta \pm \sqrt{\delta(\delta + 2\alpha)}}{2\alpha}$. Choose $h = x_0^*$. Hence, the theorem is proved.

We now know that the disease-free equilibrium exists. Its stability is also interesting. In general epidemiological models, the reproduction number, R_0, is also a threshold parameter for the stability of disease free equilibrium, such that if $R_0 < 1$, then the disease-free equilibrium is locally asymptotically stable; whereas unstable if $R_0 > 1$, (Hethcote (2000)). However, the stability may not hold for every model. It could depend on state variables and parameters. Therefore, for our model, we are also interested in investigating whether or not the resulting disease-free equilibrium is stable. This will be studied in Subsection 2.5.1.2.

2.5.1.2 Local stability of disease free equilibrium (E_f)

We will start this subsection with some background knowledge and theorems that are useful for our studies in this subsection. We begin with the stability of the equilibrium.

Theorem 2.5.2 *(Kahoui & Otto (2001)) Suppose that we have a system of ordinary differential equations written in a vector form*

$$\dot{\boldsymbol{x}} = \boldsymbol{f}(\boldsymbol{x}) \tag{2.52}$$

and $\boldsymbol{x}^*$ satisfies $\boldsymbol{f}(\boldsymbol{x}) = 0$. $\boldsymbol{J}^*$ is the Jacobian matrix of $\boldsymbol{f}(\boldsymbol{x})$ evaluated at $\boldsymbol{x}^*$. The equilibrium point $\boldsymbol{x}^*$ is locally asymptotically stable if all of the eigenvalues of $\boldsymbol{J}^*$ have negative real parts and unstable if at least one of the eigenvalues has a positive real part.

The process of finding eigenvalues for high dimensional matrices (higher than 4) can be very difficult. Usually, the calculation of eigenvalues of matrices containing state variables and parameters involves polynomials. When polynomials contain many unknown parameters, it is important to avoid computation of the coefficients. An alternative way of determining the stability is to replace the eigenvalue equation, $A\boldsymbol{x} = \lambda\boldsymbol{x}$, by the characteristic equation $det(\lambda I - A) = 0$ and determine the stability of the corresponding polynomials instead of calculate the exact eigenvalues. One useful method is the Routh-Hurwitz criteria which is a method of determining stability of a polynomial with real coefficients. The Routh-Hurwitz criteria are often used to determine local asymptotic stability of an equilibrium for non-linear system of differential equations. The criterion is stated as follows.

Theorem 2.5.3 *(Gantmakher (2000), page 221)* ***(Routh-Hurwitz criteria)*** *Given the polynomial,*

$$P(\lambda) = \lambda^n + a_1\lambda^{n-1} + \cdots + a_{n-1}\lambda + a_n,$$

where the coefficients a_i are real and non-zero, i = 1, 2, 3,..., n. The Routh-Hurwitz criteria for n = 2, 3, 4 are

$n = 2; a_1 > 0$ *and* $a_2 > 0$

$n = 3; a_1 > 0, a_3 > 0$ *and* $a_1a_2 > a_3$

$n = 4; a_1 > 0, a_3 > 0, a_4 > 0$ *and* $a_1a_2a_3 > a_3^2 + a_1^2a_4$

Note that polynomials being stable is equivalent to the roots lying in the left half of the complex plane. In other words, the solutions of polynomials have negative real parts. Following Theorem 2.5.3, we have Corollary 2.5.4 stating a necessary condition for the roots of the polynomial $P(\lambda)$ to have negative real parts.

Corollary 2.5.4 *(Gantmakher (2000), page 220) Suppose the coefficients a_i of the*

characteristic polynomial are real. If all of the roots of the following polynomial

$$P(\lambda) = \lambda^n + a_1\lambda^{n-1} + \cdots + a_{n-1}\lambda + a_n,$$

are negative or have negative real parts, then the coefficient a_i must be strictly positive, that is, $a_i > 0$ for i = 1, 2, ..., n.

The following states some basic properties which are important for computing determinants of matrices (Chapter 4, Cheney & Kincaid (2010)).

Theorem 2.5.5 *Let A and B be $n \times n$ matrix.*

1. If B is obtained by adding a multiple of one row of A to another row, $(R_i \leftarrow R_i + kR_j)$, then det B = det A.

2. If B is obtained by multiplying a row of A by a non zero constant c, $(R_i \leftarrow cR_i)$, then det B = c(det A).

3. If B is obtained by interchanging two rows of A, $(R_i \leftrightarrow R_j)$, then det B = -det A.

Now, we will start the study of the stability of the disease-free equilibrium for our model. Define $\partial_x f(x^*)$ as the Jacobian matrix with respect to x of f at x^* and note that E_f is the resulting disease-free equilibrium as in Theorem 2.5.1. Let $J(E_f) = \partial_{E_f} f(E_f)$, we have

$$J(E_f) = \begin{pmatrix} -\alpha F - \gamma_1 & 0 & 0 & \delta & 0 & \delta \\ 0 & -\alpha F - \gamma_2 & 0 & 0 & \delta & \delta \\ -\alpha F & -\alpha F & -2\alpha F - \delta & \gamma_1 - 2\alpha F & \gamma_2 - 2\alpha F & -2\alpha F \\ \alpha F & 0 & 0 & -\beta_1 - \delta - \gamma_1 & 0 & \gamma_2 \\ 0 & \alpha F & 0 & 0 & -\beta_2 - \delta - \gamma_2 & \gamma_1 \\ 0 & 0 & 0 & \beta_1 & \beta_2 & -\delta - \gamma_1 - \gamma_2 \end{pmatrix}$$

where $F = 0.5 - \frac{x_0^* y_0^* \alpha}{\delta}$. To find the eigenvalues of the Jacobian matrix $J(E_f)$, we solve $\det(J(E_f) - \lambda I_6) = 0$. Note that, the determinant of a triangular matrix

is the products of the entries on its diagonal. Therefore, in order to find the determinant of matrix $(J(E_f)-\lambda I_6)$, we use elementary row operations to obtain an upper triangular matrix. Let T_{ij} represent row-switching transformations between row i and row j. $T_{ij}(m)$ represents row-addition transformations, adding row j multiplied by a scalar m to row i, where m is a non-zero scalar. Let $M = (J(E_f) - \lambda I_6)$, and $M^{(n)}$ denotes the matrix M after the n^{th} row operation. $M^{'}$ is the final matrix after row operations, an upper triangular matrix. The sequence of the row operations is hence written as

$$T_{13}T_{41}(1)T_{31}\left(-\frac{M^{(2)}_{3,1}}{M^{(2)}_{1,1}}\right)T_{24}T_{32}\left(-\frac{M^{(4)}_{3,2}}{M^{(4)}_{2,2}}\right)T_{42}\left(-\frac{M^{(5)}_{4,2}}{M^{(5)}_{2,2}}\right)$$
$$T_{52}\left(-\frac{M^{(6)}_{5,2}}{M^{(6)}_{2,2}}\right)T_{35}T_{43}\left(-\frac{M^{(8)}_{4,3}}{M^{(8)}_{3,3}}\right)T_{53}\left(-\frac{M^{(9)}_{5,3}}{M^{(9)}_{5,3}}\right)T_{45}T_{64}$$
$$\left(-\frac{M^{(10)}_{6,4}}{M^{(10)}_{4,4}}\right)T_{65}\left(-\frac{M^{(11)}_{6,5}}{M^{(11)}_{5,5}}\right).$$

From the sequence of the row operations, we can see that there are row-interchanging 4 times and there are no elementary products. According to Theorem 2.5.5, the determinant changes sign 4 times, but was otherwise unchanged, i.e. $det(M) = (-1)(-1)(-1)(-1)det(M^{'}) = det(M^{'})$. Therefore, the determinant of M is the product of the entries on the main diagonal of $M^{'}$, which is

$$(-2F\alpha - \delta - \lambda)A_1A_2A_3 = 0 \tag{2.53}$$

where

$$A_1 = F\alpha\beta_1 + F\alpha\lambda + F\alpha\gamma_1 + \beta_1\lambda + \beta_1\gamma_1 + \delta\lambda + \delta\gamma_1 + \lambda^2 + 2\lambda\gamma_1 + \gamma_1^2.$$

$$A_2 = F\alpha\beta_2 + F\alpha\lambda + F\alpha\gamma_2 + \beta_2\lambda + \beta_2\gamma_2 + \delta\lambda + \delta\gamma_2 + \lambda^2 + 2\lambda\gamma_2 + \gamma_2^2.$$

$$A_3 = -\delta - \gamma_1 - \gamma_2 - \lambda + \frac{\beta_1(F\alpha\delta + F\alpha\gamma_2 + \lambda\gamma_2 + \gamma_1\gamma_2)}{F\alpha\beta_1 + F\alpha\lambda + F\alpha\gamma_1 + \beta_1\lambda + \beta_1\gamma_1 + \delta\lambda + \delta\gamma_1 + \lambda^2 + 2\lambda\gamma_1 + \gamma_1^2} + \frac{\beta_2(F\alpha\delta + F\alpha\gamma_1 + \lambda\gamma_1 + \gamma_1\gamma_2)}{F\alpha\beta_2 + F\alpha\lambda + F\alpha\gamma_2 + \beta_2\lambda + \beta_2\gamma_2 + \delta\lambda + \delta\gamma_2 + \lambda^2 + 2\lambda\gamma_2 + \gamma_2^2}.$$

Consider type 1 as an initial individual. Then we have that $\bar{\alpha} = \alpha(y_0^* + y_1^*)$. Since we are looking at the disease-free equilibrium, $x_1^* = y_1^* = z_{10}^* = z_{01}^* = z_{11}^* = 0$, and $x_0^* = y_0^*$. Hence, $z_{00}^* = 0.5 - y_0^*$. At equilibrium, we have $0 = -\alpha y_0^{*2} + \delta(z_{00}^*) = -\alpha y_0^{*2} + \delta(0.5 - y_0^*)$, then $y_0^* = 0.5 - \frac{\alpha y_0^{*2}}{\delta} = F$. Therefore, we have that

$$\bar{\alpha} = \alpha y_0^* = \alpha F \tag{2.54}$$

Now, we first consider the stability of the (E_f) for the special case where $\gamma_1 = \gamma_2 = \gamma$ and $\beta_1 = \beta_2 = \beta$.

2.5.1.3 The stability of the disease-free equilibrium (E_f), for the special case $\gamma_1 = \gamma_2, \beta_1 = \beta_2$, when $R_0 > 1$.

For the case $\gamma_1 = \gamma_2$ and $\beta_1 = \beta_2$, from Lemma 2.3.1, we have that $R_0 = p_{1,0}^{(1)} + p_{0,1}^{(1)} + 2p_{1,1}^{(1)}$. Substituting in the values of $p_{1,0}^{(1)}, p_{0,1}^{(1)}$, and $2p_{1,1}^{(1)}$, recalling the values from (2.4), we have that

$$R_0 = \frac{\bar{\alpha}\delta(\delta + 2\gamma + 2\beta)}{(\gamma + \bar{\alpha})(\beta\delta + \delta^2 + 3\delta\gamma + 2\gamma^2)} \tag{2.55}$$

Assuming $R_0 > 1$, by rearranging (2.55) we obtain that

$$\bar{\alpha}\beta\delta > \gamma\beta\delta + \gamma\delta^2 + 3\delta\gamma^2 + 2\gamma^3 + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 \tag{2.56}$$

From (2.53), for $\gamma_1 = \gamma_2 = \gamma$ and $\beta_1 = \beta_2 = \beta$, we have $A_1 = A_2$, and by (2.54), $\det(M) = 0$ is rewritten as follows.

$$(-2\bar{\alpha} - \delta - \lambda)A_1^2 A_3 = 0 \tag{2.57}$$

where

$$A_1 = \bar{\alpha}\beta + \bar{\alpha}\lambda + \bar{\alpha}\gamma + \beta\lambda + \beta\gamma + \delta\lambda + \delta\gamma + \lambda^2 + 2\lambda\gamma + \gamma^2.$$
$$A_3 = -\delta - 2\gamma - \lambda + \frac{2\beta(\bar{\alpha}\delta + \bar{\alpha}\gamma + \lambda\gamma + \gamma^2)}{\bar{\alpha}\beta + \bar{\alpha}\lambda + \bar{\alpha}\gamma + \beta\lambda + \beta\gamma + \delta\lambda + \delta\gamma + \lambda^2 + 2\lambda\gamma + \gamma^2}.$$

Equation (2.57) holds if $(-2\bar{\alpha}-\delta-\lambda) = 0$, $A_1 = 0$ or $A_3 = 0$. Thus, $\lambda = -(2\bar{\alpha}+\delta)$ is a root, which is negative. Consider $A_1 = 0$, it can be written in a standard from of a quadratic function as $\lambda^2+(\bar{\alpha}+\beta+\delta+2\gamma)\lambda+\bar{\alpha}\beta+\bar{\alpha}\gamma+\beta\gamma+\delta\gamma+\gamma^2 = 0$. We can see that $(\bar{\alpha}+\beta+\delta+2\gamma) > 0$ and $(\bar{\alpha}\beta+\bar{\alpha}\gamma+\beta\gamma+\delta\gamma+\gamma^2) > 0$. Therefore, according to the Routh-Hurwitz criteria for n = 2, A_1 has negative roots or the roots have negative real parts.

Now, consider $A_3 = 0$,

$$\begin{aligned}
0 &= -\delta - 2\gamma - \lambda + \frac{2\beta(\bar{\alpha}\delta + \bar{\alpha}\gamma + \lambda\gamma + \gamma^2)}{\bar{\alpha}\beta + \bar{\alpha}\lambda + \bar{\alpha}\gamma + \beta\lambda + \beta\gamma + \delta\lambda + \delta\gamma + \lambda^2 + 2\lambda\gamma + \gamma^2} \\
&= \bar{\alpha}\beta\delta - \bar{\alpha}\beta\lambda - \bar{\alpha}\delta\lambda - \bar{\alpha}\delta\gamma - \bar{\alpha}\lambda^2 - 3\bar{\alpha}\lambda\gamma - 2\bar{\alpha}\gamma^2 - \beta\delta\lambda - \beta\delta\gamma - \beta\lambda^2 - \beta\lambda\gamma \\
&\quad - \delta^2\lambda - \delta^2\gamma - 2\delta\lambda^2 - 5\delta\lambda\gamma - 3\delta\gamma^2 - \lambda^3 - 4\lambda^2\gamma - 5\lambda\gamma^2 - 2\gamma^3. \\
&= \lambda^3 + (\bar{\alpha} + \beta + 2\delta + 4\gamma)\lambda^2 + (\bar{\alpha}\beta + \bar{\alpha}\delta + \beta\delta + 3\bar{\alpha}\gamma + \beta\gamma + \delta^2 + 5\delta\gamma + 5\gamma^2)\lambda \\
&\quad + (-\bar{\alpha}\beta\delta + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3).
\end{aligned}$$

Since $R_0 > 1$, by (2.56), we have that

$$\begin{aligned} d &= -\bar{\alpha}\beta\delta + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3 \\ &< -(\gamma\beta\delta + \gamma\delta^2 + 3\delta\gamma^2 + 2\gamma^3 + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2) + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3 \\ &= 0. \end{aligned}$$

Hence $d < 0$. Since there is a negative coefficient, Corollary 2.5.4 guarantees a positive root or a root with positive real part. We can conclude that there exists at least one positive eigenvalue in this case. We thus establish that the disease-free equilibrium, E_f, is unstable if $R_0 > 1$.

2.5.1.4 The stability of the disease-free equilibrium (E_f), for the special case $\gamma_1 = \gamma_2, \beta_1 = \beta_2$, when $R_0 < 1$.

$$\text{If } \; R_0 \leq 1, \;\; \text{then } \; \bar{\alpha}\beta\delta \leq \gamma\beta\delta + \gamma\delta^2 + 3\delta\gamma^2 + 2\gamma^3 + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 \tag{2.58}$$

(2.57) still holds and thus we only need to consider $A_3 = 0$ as $A_1 = 0$ and $(-2\bar{\alpha} - \delta - \lambda) = 0$ correspond to $\lambda < 0$. As described earlier, we have

$$\begin{aligned} A_3 = \lambda^3 &+ (\bar{\alpha} + \beta + 2\delta + 4\gamma)\lambda^2 + (\bar{\alpha}\beta + \bar{\alpha}\delta + \beta\delta + 3\bar{\alpha}\gamma + \beta\gamma + \delta^2 + 5\delta\gamma + 5\gamma^2)\lambda \\ &+ (-\bar{\alpha}\beta\delta + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3) = 0. \end{aligned}$$

We can express A_3 in terms of a characteristic polynomial as $\lambda^3 + b\lambda^2 + c\lambda + d$. In this case, $d > 0$, as a result all coefficients are positive. According to Routh-Hurwitz criteria for $n = 3$, we need to check if $bc > d$ (see Theorem 2.5.3).

$$\begin{aligned} bc &= (\bar{\alpha}+\beta+2\delta+4\gamma)(\bar{\alpha}\beta+\bar{\alpha}\delta+\beta\delta+3\bar{\alpha}\gamma+\beta\gamma+\delta^2+5\delta\gamma+5\gamma^2) \\ &= \bar{\alpha}^2\beta+\bar{\alpha}^2\delta+3\bar{\alpha}^2\gamma+\bar{\alpha}\beta^2+4\bar{\alpha}\beta\delta+8\bar{\alpha}\beta\gamma+3\bar{\alpha}\delta^2+15\bar{\alpha}\delta\gamma+17\bar{\alpha}\gamma^2 \\ &+ \beta^2\delta+\beta^2\gamma+3\beta\delta^2+11\beta\delta\gamma+9\beta\gamma^2+2\delta^3+14\delta^2\gamma+30\delta\gamma^2+20\gamma^3. \end{aligned}$$

Then

$$\begin{aligned} bc-d &= \bar{\alpha}^2\beta+\bar{\alpha}^2\delta+3\bar{\alpha}^2\gamma+\bar{\alpha}\beta^2+5\bar{\alpha}\beta\delta+8\bar{\alpha}\beta\gamma+3\bar{\alpha}\delta^2+14\bar{\alpha}\delta\gamma+15\bar{\alpha}\gamma^2 \\ &+ \beta^2\delta+\beta^2\gamma+3\beta\delta^2+10\beta\delta\gamma+9\beta\gamma^2+2\delta^3+13\delta^2\gamma+27\delta\gamma^2+18\gamma^3 > 0. \end{aligned}$$

As a result, $bc > d$. Routh-Hurwitz criteria confirms that the polynomial is stable, i.e. roots of A_3 have negative real parts. Therefore, E_f is stable. We then establish Theorem 2.5.6.

Theorem 2.5.6 *If $\gamma_1 = \gamma_2$ and $\beta_1 = \beta_2$, then the disease-free equilibrium $E_f = (h_1, 0, h_2, 0, \frac{\alpha h^2}{\delta}, 0, 0, 0)$, where $h = \dfrac{-\delta+\sqrt{\delta(\delta+2\alpha)}}{2\alpha}$ is locally asymptotically stable if $R_0 \leq 1$ and unstable if $R_0 > 1$.*

We have proved the local stability of the disease-free equilibrium which is non-trivial. We would expect much more difficulty with algebra for proving global stability due to the Jacobian matrix not being sparse in this case. In terms of the stability of the endemic equilibrium, we need an alternative approach. Lyapunov 's direct method has been a popular technique to study global stability of epidemic models (Salle & Lefschetz (1961); Vargas-De-Leon (2011); Egbetade & Ibrahim (2012) ; Korobeinikov & Wake (2012)). However, a suitable Lyapunov function specifically for our model is not yet known to us, and to find one could require considerably more research. Therefore, we will leave the stability of the endemic equilibrium of both Model 1 and Model 2 for future work. Note that, the stability of the disease-free and the endemic equilibrium for Model 2 could also be proved

using the same approach but would be more complicated due to more parameters (one-night stands) being included in the model.

2.5.2 Limiting Diffusion Process

According to Section 1.3.2 in Chapter 1, for $N \to \infty$, the stochastic process converges to the solution of the ODEs, i.e. $\lim_{N\to\infty}(\boldsymbol{X}_N(t), \boldsymbol{Y}_N(t), \boldsymbol{Z}_N(t)) = (\boldsymbol{x}(t), \boldsymbol{y}(t), \boldsymbol{z}(t))$. In the deterministic process, the endemic level is steady, but we can study the fluctuations of the stochastic process about the deterministic process. The fluctuations of the stochastic process about the deterministic limits scaled by $\sqrt{N}$, $\sqrt{N}((\boldsymbol{X}_N(t), \boldsymbol{Y}_N(t), \boldsymbol{Z}_N(t)) - (\boldsymbol{x}(t), \boldsymbol{y}(t), \boldsymbol{z}(t)))$, converges to a diffusion process. More importantly, if we choose an equilibrium point as an initial value of the deterministic approximation, $\sqrt{N}((\boldsymbol{X}_N(t), \boldsymbol{Y}_N(t), \boldsymbol{Z}_N(t)) - (\boldsymbol{x}(t), \boldsymbol{y}(t), \boldsymbol{z}(t)))$ will converge to the Ornstein-Uhlenbeck process (Pollett (2001)). This Ornstein-Uhlenbeck has a Gaussian stationary distribution with mean-zero and covariance matrix $\boldsymbol{\Sigma}$ defined by

$$\boldsymbol{B\Sigma} + \boldsymbol{\Sigma B^T} = -\boldsymbol{U} \tag{2.59}$$

with

$$\boldsymbol{B} = (b)_{i,j}, i, j \in \{1, 2, ..., 6\}$$

where,

$$
\begin{array}{lll}
b_{1,1} = -\gamma_1 - \alpha G_3^* & b_{1,2} = 0 & b_{1,3} = \alpha x_1^* \\
b_{1,4} = \alpha x_1^* + \delta & b_{1,5} = \alpha x_1^* & b_{1,6} = \alpha x_1^* + \delta \\
b_{2,1} = 0 & b_{2,2} = -\gamma_2 - \alpha G_3^* & b_{2,3} = \alpha y_1^* \\
b_{2,4} = \alpha y_1^* & b_{2,5} = \alpha y_1^* + \delta & b_{2,6} = \alpha y_1^* + \delta \\
b_{3,1} = -G_2^* \alpha & b_{3,2} = -\alpha G_1^* & b_{3,3} = -G_1^* \alpha - G_2^* \alpha - \delta \\
b_{3,4} = -G_1^* \alpha - G_2^* \alpha + \gamma_1 & b_{3,5} = -G_1^* \alpha - G_2^* \alpha + \gamma_2 & b_{3,6} = -G_1^* \alpha \\
b_{4,1} = \alpha G_2^* & b_{4,2} = -\alpha x_1^* & b_{4,3} = -\alpha x_1^* \\
b_{4,4} = -\alpha x_1^* - \beta_1 - \delta - \gamma_1 & b_{4,5} = -\alpha x_1^* & b_{4,6} = -\alpha x_1^* + \gamma_2 \\
b_{5,1} = -\alpha y_1^* & b_{5,2} = \alpha G_1^* & b_{5,3} = -\alpha y_1^* \\
b_{5,4} = -\alpha y_1^* & b_{5,5} = -\alpha y_1^* - \beta_2 - \delta - \gamma_2 & b_{5,6} = \gamma_1 - \alpha y_1^* \\
b_{6,1} = \alpha y_1^* & b_{6,2} = \alpha x_1^* & b_{6,3} = 0 \\
b_{6,4} = \beta_1 & b_{6,5} = \beta_2 & b_{6,6} = -(\gamma_1 + \gamma_2 + \delta)
\end{array}
$$

and

$$\boldsymbol{U} = (u)_{i,j}, i, j \in \{1, 2, ..., 6\}$$

where,

$$
\begin{array}{ll}
u_{11} = \alpha x_1^* G_2 + \alpha x_1^* y_1^* + \delta z_{10}^* + \delta z_{11}^* + \gamma_1 x_1^*, & u_{1,2} = \alpha x_1^* y_1^* + \delta z_{11}^* \\
u_{1,4} = -\delta z_{10}^* - \alpha x_1^* G_2^* & u_{1,6} = -\alpha x_1^* y_1^* - \delta z_{11}^* \\
u_{2,2} = \alpha G_1^* y_1^* + \alpha x_1^* y_1^* + \delta z_{01}^* + \delta z_{11}^* + \gamma_2 y_1^* & u_{2,5} = -\alpha G_1^* y_1^* - \delta z_{01}^* \\
u_{2,6} = -\alpha x_1^* y_1^* - \delta z_{11}^* & u_{3,3} = \alpha G_1^* G_2^* + \delta z_{00}^* + \gamma_1 z_{10}^* + \gamma_2 z_{01}^* \\
u_{3,4} = -\gamma_1 z_{10}^* & u_{3,5} = -\gamma_2 z_{01}^* \\
u_{4,4} = \alpha x_1^* G_2^* + \delta z_{10}^* + \beta_1 z_{10}^* + \gamma_2 z_{11}^* + \gamma_1 z_{10}^* & u_{4,6} = -\beta_1 z_{10}^* - \gamma_2 z_{11}^* \\
u_{5,5} = \alpha G_1^* y_1^* + \delta z_{01}^* + \beta_2 z_{01}^* + \gamma_2 z_{01}^* + \gamma_1 z_{11}^* & u_{5,6} = -\beta_2 z_{01}^* - \gamma_1 z_{11}^* \\
u_{6,6} = \alpha x_1^* y_1^* + \delta z_{11}^* + \beta_1 z_{10}^* + \beta_2 z_{01}^* + \gamma_1 z_{11}^* + \gamma_2 z_{11}^* &
\end{array}
$$

The remaining covariance terms are 0, and $\boldsymbol{U}$ is a symmetric matrix. Note that matrices $\boldsymbol{B}$ and $\boldsymbol{U}$ are local drift and covariance matrices corresponding to the system (2.46), and G_1, G_2 and G_3 are as defined in (2.47), (2.48) and (2.49) with equilibrium values $\boldsymbol{x^*}, \boldsymbol{y^*}, \boldsymbol{z^*}$.

It is not possible to give an algebraic solution for $\mathbf{\Sigma}$ in (2.59), but it is quite straightforward to numerically evaluate $\mathbf{\Sigma}$, for example, using Matlab. Note that we are interested in studying fluctuations of infectives about the endemic level. In particular, we are interested in mean and variance of the total number of infected individuals. Recall that $I(t)$ represents the total number of infectives and i is the proportion of infectives in population size N. Let i^*be the proportion of infectives in equilibrium. Note that $i^* = x_1^* + y_1^* + z_{10}^* + z_{01}^* + 2z_{11}^*$. Let $\hat{I}$ denote the quasi-stationary distribution of the epidemic process. Then $\hat{I}$ is the limiting distribution as $t \to \infty$ of $I(t)|I(t) > 0$. As $N \to \infty$, $\sqrt{N}(\hat{I}/N - i^*)$ converges in distribution to a zero mean Gaussian distribution with variance Σ_i, where $\mathbf{\Sigma}_i$ represents the variance of the proportion of infectives obtained in the Ornstein-Uhlenbeck process and note that ς_{ij} are elements of the covariance matrix $\mathbf{\Sigma}$ which is a solution to the equation (2.59). Therefore, for large N the evolution of $I(t)$ is well approximated by an Ornstein-Uhlenbeck process with the following mean and variance

$$
\begin{aligned}
&E[\hat{I}] = Ni^* = N(x_1^* + y_1^* + z_{10}^* + z_{01}^* + 2z_{11}^*),\\
&Var(\hat{I}) = N\mathbf{\Sigma_i} = N(\varsigma_{11} + \varsigma_{22} + \varsigma_{44} + \varsigma_{55} + 4\varsigma_{66} + 2\varsigma_{12} + 2\varsigma_{14}\\
&+ 2\varsigma_{15} + 4\varsigma_{16} + 2\varsigma_{24} + 2\varsigma_{25} + 4\varsigma_{26} + 2\varsigma_{45} + 4\varsigma_{46} + 4\varsigma_{56}),
\end{aligned}
$$

Since the standard deviation determines the size of fluctuation about the mean, a smaller standard deviation of infectives indicates that the number of infected individuals fluctuates closer to its mean. When the epidemic makes larger fluctuations, it implies that the number of infected individuals is likely to hit zero faster. As a result, the time to extinction is expected to be shorter. To get an idea of variation relative to the mean, we consider the coefficient of variation (C_v) which is defined as the ratio of the standard deviation to the mean, $C_v = \frac{\sigma_i}{\mu_i}$. Therefore, a higher coefficient of variation means that the epidemic is likely to reach the disease free state faster. The numerical studies regarding fluctuations about endemic level

will be discussed in Section 2.7.

Next, our key interest is the time to extinction starting from the endemic equilibrium, which will be discussed in Section 2.6.

2.6 Time to Extinction

Our aim in this section is to study the expected time to extinction of the epidemic given the process is started at the endemic level. We use stochastic simulations to study this phenomenon. The simulation can be done using the well-known Gillespie algorithm (Gillespie (1976)), which is discussed below. To start, we define the state space as a set of number of individuals of each class defined in Subsection 2.5.1, namely,

$$S = \{X_0(t), X_1(t), Y_0(t), Y_1(t), Z_{00}(t), Z_{10}(t), Z_{01}(t), Z_{11}(t)\},$$

where S represents the state space. Let a_i represent a rate with respect to process i, where $i \in \{1, 2, ...16\}$. We have the following table.

Table 2.1: Processes and their transition rates

a_i	Rate	Process
a_1	$\gamma_1 X_1$	Recovery of a single male.
a_2	$\gamma_2 Y_1$	Recovery of a single female.
a_3	$\gamma_1 Z_{10}$	Recovery of a male whose partner is susceptible
a_4	$\gamma_2 Z_{01}$	Recovery of a female whose partner is susceptible
a_5	$\gamma_1 Z_{11}$	Recovery of a male whose partner is infectious
a_6	$\gamma_2 Z_{11}$	Recovery of a female whose partner is infectious
a_7	$\alpha X_0 Y_0$	Relationship formation between a susceptible male and female.
a_8	$\alpha X_1 Y_0$	Relationship formation between an infectious male and a susceptible female.
a_9	$\alpha X_0 Y_1$	Relationship formation between a susceptible male and an infectious female.
a_{10}	$\alpha X_1 Y_1$	Relationship formation between an infectious male and female.
a_{11}	δZ_{00}	Relationship dissolution between a susceptible male and female.
a_{12}	δZ_{10}	Relationship dissolution between an infectious male and a susceptible female.
a_{13}	δZ_{01}	Relationship dissolution between a susceptible male and an infectious female.
a_{14}	δZ_{11}	Relationship dissolution between an infectious male and female.
a_{15}	$\beta_1 Z_{10}$	Infection from male to female
a_{16}	$\beta_2 Z_{01}$	Infection from female to male

According to the table above, 16 transitions together with their respective rates are defined. First, we determine which event happens next using random number generators. The waiting time until the next event occurs is exponentially distributed. Once the event occurs, the state variables are updated according to their transitions. We repeat the algorithm until the total number of infected individuals equals 0. Namely, the algorithm is terminated when $I(t) = 0$.

The algorithm can be summed up in the following pseudo code.

Algorithm 2.6.1: STOCHASTIC$(parameters, initial, t_{end})$

1 : **if** $(I(t) > 0)$ **then**

2 : **for** $i = 1..16$ **do**

$Calculate \;\; a_i \;\; and \;\; \lambda_i = \sum_{k=1}^{i} a_i$

end for

3 : $\tau \sim Exp(\lambda_{16})$

4 : *Generate a uniform random variable* $r, 0 \leq r \leq 1$

$r* = r\lambda_{16}$

5 : *Find i such that* $\lambda_{i-1} < r^* \leq \lambda_i$

6 : *Set* $t = t + \tau$

7 : *Update the current state space S corresponding to the event i found in* 5.

The stochastic simulations based on the algorithm presented above are implemented in Matlab. The numerical results and analysis will be shown in Section 2.7.

2.7 Numerical Results and analysis

In this section, numerical calculations of the reproduction number (R_0), the probability of extinction, and the endemic equilibrium level were performed for a range of parameter values, using the R programming language. Time to extinction was studied using stochastic simulations, implemented in Matlab. For the mean time to extinction, we present no simulations for $R_0 \leq 1$. For $R_0 > 1$, the mean time to extinction results were calculated from 1000 simulations.

2.7.1 Effects of each parameter on R_0 and probability of extinction

In this Section, we first consider a simplification of the model where there is no difference in rates between male and female, i.e. $\beta_1 = \beta_2 = \beta$ and $\gamma_1 = \gamma_2 = \gamma$. In order to gain more insight into the behaviour of the epidemic, it is worthwhile exploring the role of each parameter on R_0. Intuitively, increasing infection rates will increase the likelihood of infection, and increase R_0. If the rates of recovery from the disease increase, it is obvious that the incidence of the disease reduces, resulting in decreasing R_0. Figure 2.4 shows the effect of each parameter on R_0, for fixed values of the other parameters. For each case, we choose 4 different sets of parameters such that, for example, while increasing each parameter of interest other parameters are set either high or low governing varied possible behaviours. The parameter values can be found in Table 2.2 - 2.5. Note that, in this section, we only want to study the effect on the behaviour of R_0 of each parameter, therefore, there is no link from the chosen parameters to any real data. The results corresponding to each parameter set are represented in colour.

Table 2.2: Parameters chosen for Figure 2.4(a)

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$
1	9	9	2	2	1:100	1:100
2	30	50	1	1	1:100	1:100
3	0.3	0.8	0.1	0.1	1:100	1:100
4	1000	1000	5	5	1:100	1:100

Table 2.3: Parameters chosen for Figure 2.4(b)

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$
1	3	6	1:100	1:100	100	100
2	1000	1000	1:100	1:100	20	20
3	30	0.1	1:100	1:100	500	500
4	10	100	1:100	1:100	300	300

Table 2.4: Parameters chosen for Figure 2.4(c)

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$
1	1:100	6	2	2	20	20
2	1:100	1000	1	1	50	50
3	1:100	1	0.1	0.1	0.5	0.5
4	1:100	0.1	1	1	80	80

Table 2.5: Parameters chosen for Figure 2.4(d)

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$
1	5	1:100	1	1	100	100
2	0.8	1:100	0.02	0.02	0.5	0.5
3	100	1:100	0.1	0.1	0.2	0.2
4	200	1:100	3	3	40	40

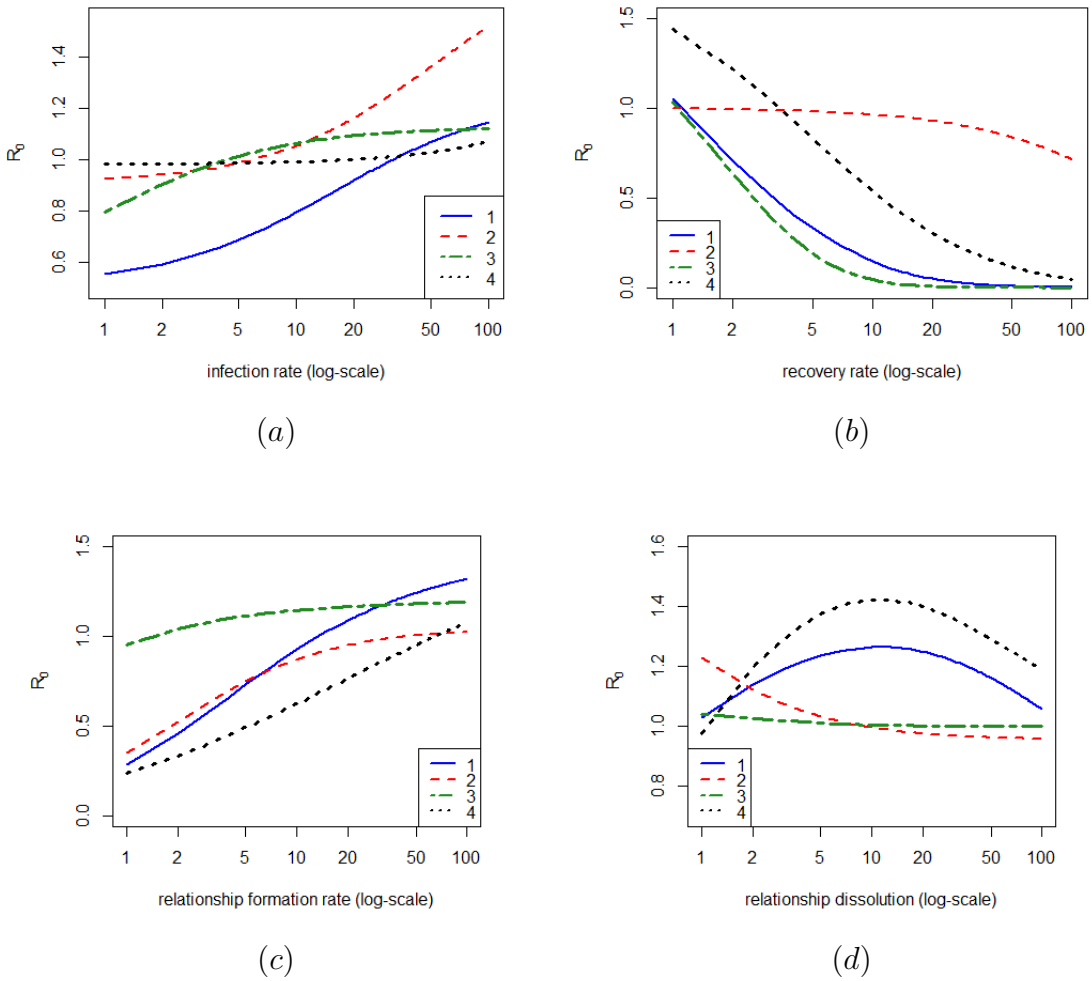


Figure 2.4: (a) Effect of an infection rate on R_0, varying β: 1, 2 ,..,100.
(b) Effect of a recovery rate on R_0, varying γ: 1, 2 ,..,100.
(c) Effect of a relationship formation rate on R_0, varying α: 1, 2 ,..,100.
(d) Effect of a relationship dissolution (break up) rate on R_0, varying δ: 1, 2 ,..,100.

We can see in Figure 2.4 (a) and (b) that whilst fixing other parameters, increasing β and γ will respectively increase and decrease R_0 with different slope for different parameter sets. The results for parameter set 2 (red dash line) in Figure 2.4 (b) show a significant difference in behaviour of R_0 in comparison to those with other parameter sets. From Table 2.3, the parameters chosen for set 2 are $\beta = 20, \alpha = 1000, \delta = 1000$, and γ varies from 1 to 100. We can see that relationship formation and break up rates are very high showing that there are a lot of

turnover of relationships with very short relationship period. Since the infection rate is small, the disease has very low chance to be transmitted before the break up occurs. This leads to much slower decreasing in R_0. In Figure 2.4 (c), we can see an expected behaviour of R_0 such that it increases for an increasing α, but what is worth noting is the behaviour of R_0 for increasing δ in Figure 2.4 (d). We can see that R_0 can be both increasing and decreasing depending on which parameter set. This is possibly because, in this model, the transmission only occurs within the relationship stages. Small δ means people stay in a relationship for a long time and eventually the disease dies out (both infectives in a relationship eventually recover). Whereas, large δ means people spend relatively little time in relationships and thus they have limited chance to pass on the infection. For moderate δ, it means infected individuals are able to transmit the disease to their partner and then break up and find a new partner to infect. Therefore, it is interesting to see what happens if we keep the proportion in relationship fixed, namely, we set $F = \frac{\bar{\alpha}}{\delta}$. The remaining parameters are kept constant. Figure 2.5 illustrates the behaviour of R_0 with respect to increasing values of δ from 1 to 2000. In this study, we consider 6 different proportions of relationships, i.e $F = 1, 5, 50, 500, 1000, 2000$. There is no difference in gender, i.e. $\gamma_1 = \gamma_2, \beta_1 = \beta_2$. We study 4 cases where γ and β are set as follows :

1) $\gamma = 0.05, \beta = 0.8$ 2) $\gamma = 1, \beta = 200$ 3) $\gamma = 1.5, \beta = 0.5$ 4) $\gamma = 5, \beta = 30$

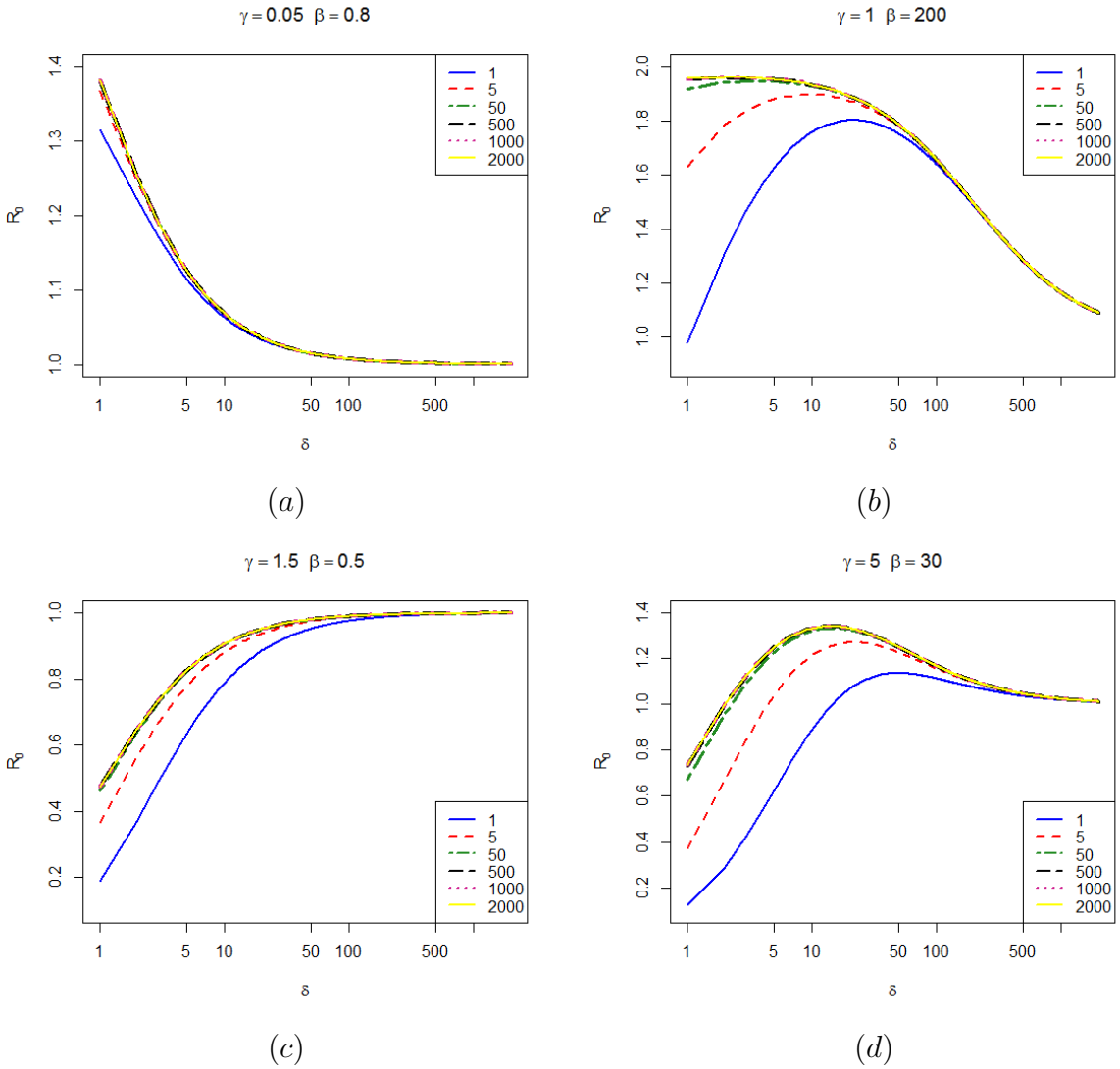


Figure 2.5: (a) Plot of R_0 against δ corresponding to each fixed F for $\gamma = 0.05, \beta = 0.8$
(b) Plot of R_0 against δ corresponding to each fixed F for $\gamma = 1, \beta = 200$
(c) Plot of R_0 against δ corresponding to each fixed F for $\gamma = 1.5, \beta = 0.5$
(d) Plot of R_0 against δ corresponding to each fixed F for $\gamma = 5, \beta = 30$

Figure 2.5 shows that, as δ increases, the behaviour of R_0 is not always monotonic. We also observe that R_0 converges to 1 when $\delta \longrightarrow \infty$ and this can be seen in every case. It is quite straightforward to show this mathematically. Recalling an expression of R_0 when $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$ as in (2.55) and substituting $\bar{\alpha} = \delta F$,

we have

$$\begin{aligned} R_0 &= \frac{(\delta^2 F)(\delta + 2\gamma + 2\beta)}{(\gamma + (\delta F))(\beta\delta + \delta^2 + 3\delta\gamma + 2\gamma^2)} \\ &= \frac{(\delta^3 F) + 2\gamma(\delta^2 F) + 2\beta\delta^2 F}{(\delta^3 F) + \delta^2 F(\beta + 3\gamma) + 2\gamma^2\delta F + \delta\beta\gamma + \gamma\delta^2 + 3\delta\gamma^2 + 2\gamma^3.} \end{aligned}$$

It is straightforward to see that $\lim_{\delta\to\infty} R_0 = 1$. Moreover, we observe from the graphs that when F increases, R_0 converges to a limit. We can easily find the limiting value :

$$\lim_{F\to\infty} R_0 = \frac{\delta(\delta + 2\gamma + 2\beta)}{\beta\delta + \delta^2 + 3\delta\gamma + 2\gamma^2}.$$

Also, note that, F is the relative rate of formation and breakup, therefore, as $F = \frac{\bar{\alpha}}{\delta} \to \infty$, $\sigma = \frac{\delta}{\bar{\alpha}+\delta} \to 0$. Hence, $1 - \sigma \to 1$ as $F \to \infty$, where $1 - \sigma$ is the proportion of relationships in equilibrium. In other words, we could say that as F increases and tends to infinity, we will eventually end up having only couples in the population (nobody stays single). With both increasing $\bar{\alpha}$ and δ, this illustrates a scenario such that there is a large turnover of relationships. Namely, individuals break up and form a new relationship almost immediately. Therefore, the chance of an individual trying to infect the same individual twice converges to 0. Consequently, the model increasingly resembles a host-vector epidemic model with infection rate $\beta(1 - \sigma)$ and recovery rate γ for both vectors and hosts.

Next, we are interested in studying the behaviour of the corresponding extinction probabilities. Figure 2.6 illustrates the behaviour of π_1 corresponding to parameter sets and fixed F in Figure 2.5. Note that the probability of extinctions of Figure 2.5(c) will always be 1 as $R_0 < 1$. Since $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$, will have that $\pi_1 = \pi_2$.

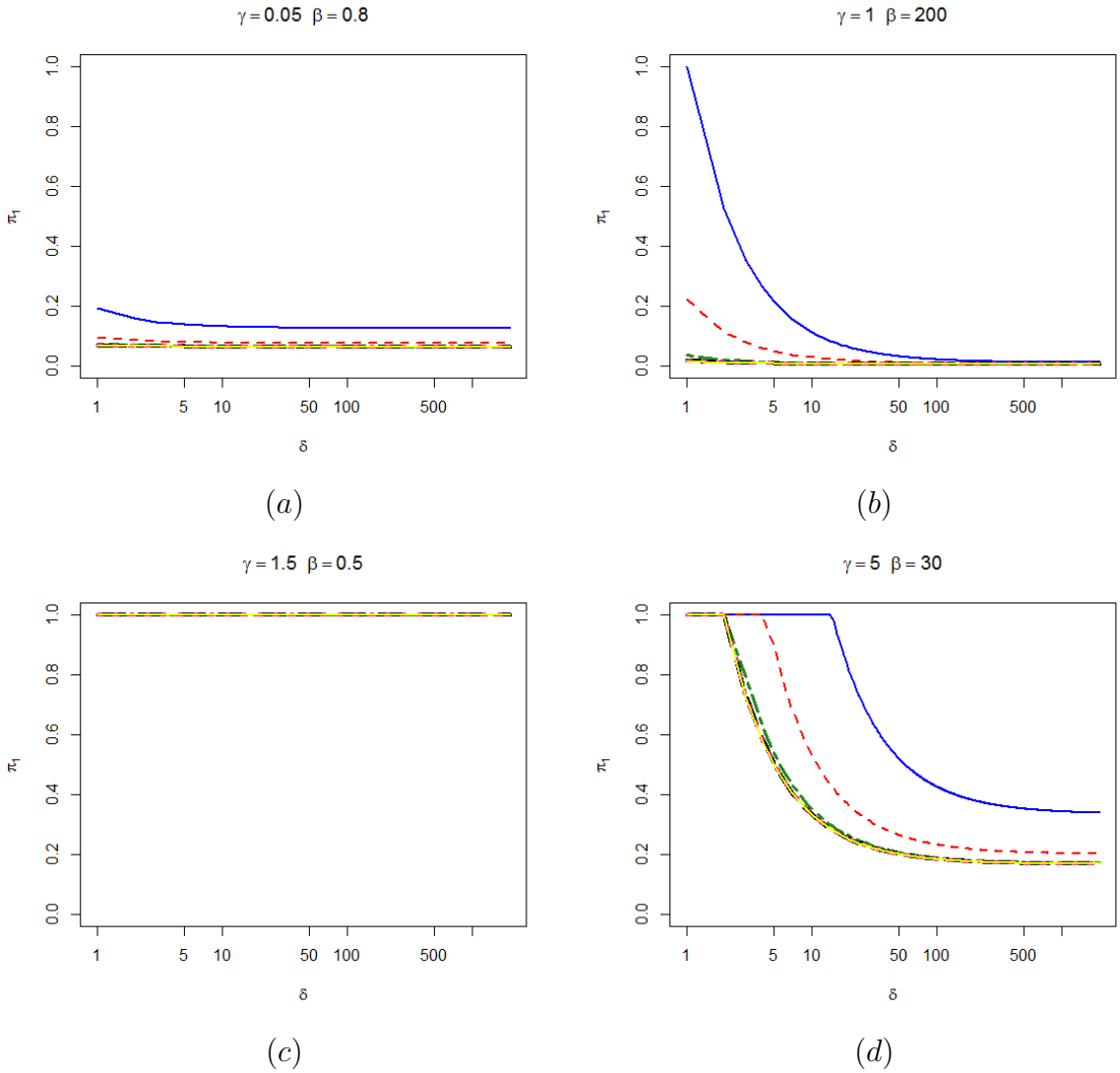


Figure 2.6: (a) Probability of extinction with respect to Figure 2.5(a)
(b) Probability of extinction with respect to Figure 2.5(b)
(c) Probability of extinction with respect to Figure 2.5(c)
(d) Probability of extinction with respect to Figure 2.5(d)

We note that π_1, in contrast to R_0, is monotonically decreasing as δ increases. As F increases, π_1 converges to a limit. Moreover, we can see in Figure 2.6(b) and 2.6(d) that π_1 is monotonically decreasing in δ and appears to be converging as δ tends to infinity. This limiting value can be found by taking limit of π as $\delta \to \infty$. We have proved that π is decreasing in δ in Lemma 2.4.4. Therefore, we can say that for fixed $F = \frac{\bar{\alpha}}{\delta}$, π is monotonically decreasing in δ and converging to a limiting value θ. According to Lemma 2.4.4, we know that

$\pi = \frac{p_0}{p_2} = \frac{\gamma((1+F)\delta^2+(\beta+3\gamma+2\gamma F)\delta+2\gamma^2)}{\delta^2 F\beta}$. Then,

$$\lim_{\delta\to\infty} \frac{p_0}{p_2} = \frac{\gamma(1+F)}{F\beta} = \theta.$$

Substituting $F = \frac{\bar{\alpha}}{\delta}$, we have $\theta = \frac{\gamma(\bar{\alpha}+\delta)}{\beta\bar{\alpha}}$.

Moreover, if we take limit of π as $F \to \infty$, we have

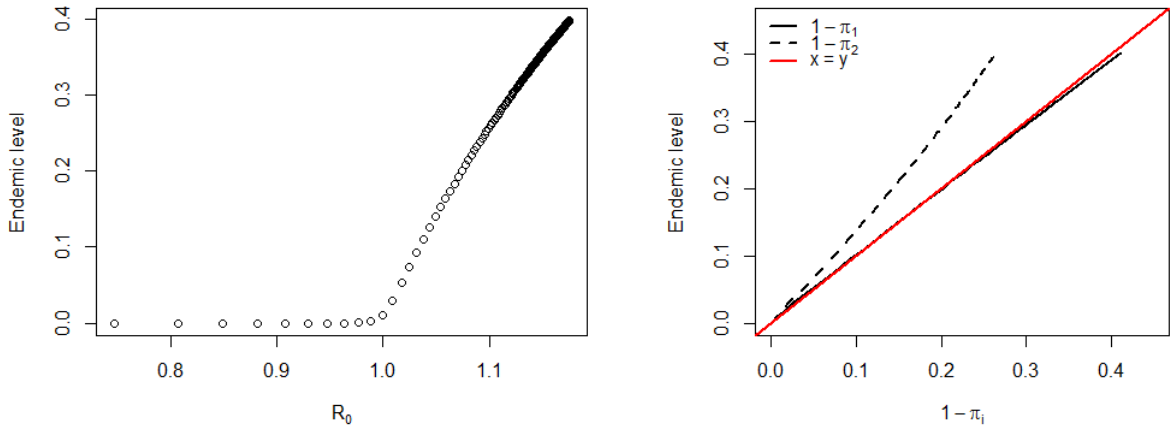
$$\lim_{F\to\infty} \frac{p_0}{p_2} = \frac{\gamma}{\beta}.$$

Note that, for the homogeneously mixing epidemic model, the reproduction number is $R_0 = \frac{\beta}{\gamma}$ and the endemic level is $1-(1/R_0)$ (see Section 1.2). For our model, we have that as $F \to \infty$, $\pi \to \frac{\gamma}{\beta}$ which is $1/R_0$ in a homogeneously mixing model. Also, $1-\pi = 1-(1/R_0)$, which is the endemic level. Therefore, we can say that when $\beta_1 = \beta_2$ and $\gamma_1 = \gamma_2$, as $F \to \infty$, the model increasingly behaves like a host-vector model (hosts infect vectors and visa-versa), with infection rate β and recovery rate γ for both hosts and vectors.

2.7.2 R_0, the probability of extinction and the endemic level

As we mentioned earlier, R_0 determines if a major outbreak is possible. If there is an outbreak, the epidemic will reach an endemic equilibrium and spend a long time close to this level before eventually going extinct. The positive proportion infected in equilibrium is called the endemic level. In this subsection, we focus on the relationship between R_0, the probability of extinction and the endemic level. In general, we know that an epidemic has behaviour such that when $R_0 \leq 1$, there is no outbreak of the epidemic, therefore the endemic level is 0, whilst for $R_0 > 1$, the endemic level is greater than 0. In terms of the relationship between the endemic level and π_1, as the endemic level increases, π_1 decreases and goes to

1 when the endemic level is 0, meaning that the probability of the disease dying out is 1. In our model, as we studied in Subsection 2.7.1, R_0 is monotonically increasing and decreasing in infection and recovery rates, respectively. This is not surprising as we can see such relationship in general epidemic models. We have looked at the case where there is no difference in disease dynamics between the two sexes and now we will look at the case where there is a difference. This will be illustrated in the Figure 2.7.

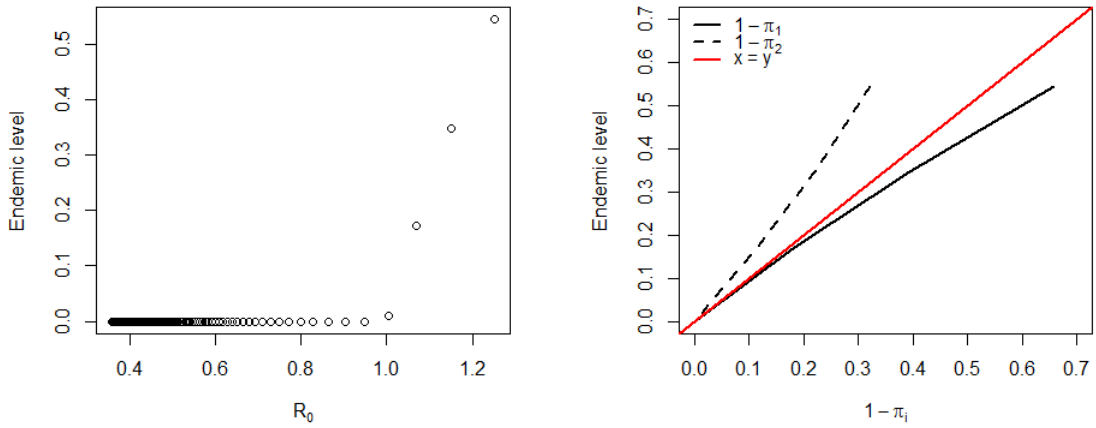


(a) (b)

Figure 2.7: (a) Plot of endemic level against R_0 and (b) Plot of endemic level against probability of the disease not going extinct when $\gamma_1 = 1, \gamma_2 = 2, \delta = 6, \alpha = 9, \beta_2 = 18$ and β_1 varying from $1, 2, 3, \ldots, 2000$.

Figure 2.7(a) shows the expected behaviour between endemic level and R_0 as R_0 increases in β. In terms of the probability of extinction, we plot endemic level against $1 - \pi_1$ and $1 - \pi_2$, as well as the line $x = y$ (red line). We can see from Figure 2.7(b) that the endemic level has linear relationship with $1 - \pi_1$ close to $x = y$, where as it has non-linear relationship with $1 - \pi_2$.

Similarly, we consider the case where γ_1 is varied. Parameter values are $\gamma_2 = 8, \beta_1 = 300, \beta_2 = 200, \delta = 25, \alpha = 18$. γ_1 varies from 1, 2, ..., 1000. See Figure 2.8.



(a) (b)

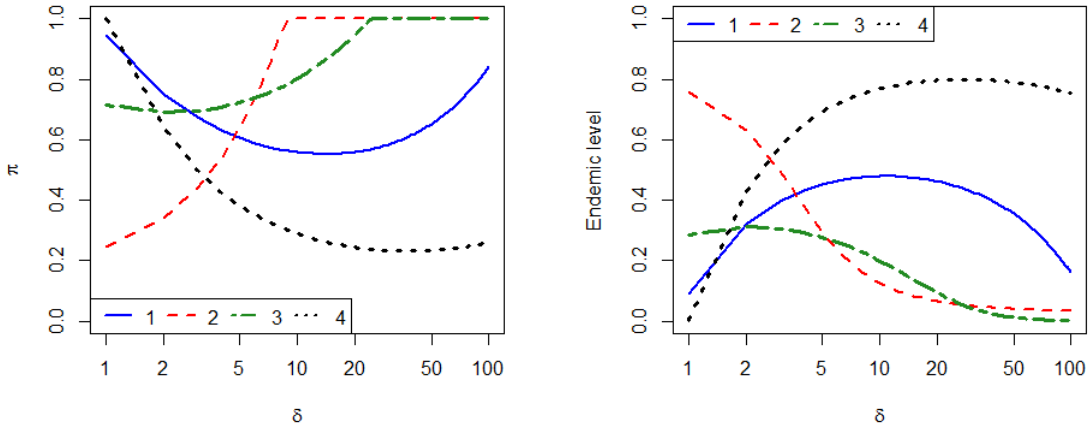
Figure 2.8: (a) Plot of endemic level against R_0 and (b) Plot of endemic level against $1-\pi_i, i = 1, 2$ when $\gamma_2 = 8, \delta = 25, \alpha = 18, \beta_1 = 300, \beta_2 = 200$ and γ_1 varying from $1, 2, 3, \ldots, 1000$.

From Figure 2.8(b), the endemic level looks non-linearly related to both $1-\pi_1$ and $1-\pi_2$. We can also see that, $1-\pi_1$ goes away from the straight line $x = y$ in this case.

Now, what is more interesting here is the case with increasing δ. As we can see that for some parameter sets with reasonably large δ, R_0 becomes non-monotonic. It is interesting to investigate the behaviours of the endemic level and the probability of extinction corresponding to such R_0. Note from our studies in the previous subsection that π has monotonic behaviour for every case as δ increases if $F = \frac{\bar{\alpha}}{\delta}$ is fixed, whereas this is not the case for R_0. What if $F = \frac{\bar{\alpha}}{\delta}$ is not fixed? What is the behaviour of the probability of extinction for the super-critical case ($R_0 > 1$) corresponding to an increasing δ? For the sub-critical case, the probability of extinction is not interesting as it will always be 1. We choose to study the parameter sets in Table 2.5 as we have a range of behaviours of R_0 for these parameter sets, both monotonic and non-monotonic (see Figure 2.4(d)). The studies are illustrated in Figure 2.9(a) and (b). We also recall Table 2.5 here

Table 2.6: Parameters chosen

set	α	δ	γ_1	γ_2	β_1	β_2
1	5	1:100	1	1	100	100
2	0.8	1:100	0.02	0.02	0.5	0.5
3	100	1:100	0.1	0.1	0.2	0.2
4	200	1:100	3	3	40	40



(a) (b)

Figure 2.9: (a) Plot of the probability of extinction against δ (b) Plot of the endemic level against δ, with respect to parameter sets in Table 2.5

Note that in Figure 2.4(d), R_0 for parameter sets 1 and 4 have non-monotonic behaviour. Figure 2.9 shows that π and the endemic level can also have both monotonic and non-monotonic behaviours as δ increases.

Let us now turn our attention to the case where F is fixed. In Subsection 2.7.1, we studied behaviours of R_0 and π_1 with respect to increasing F. We have proved in previous section that as $F \to \infty$, the model behaves like a two type homogeneously mixing model (an individual can only infect someone of the opposite sex). Therefore, numerically, we expect to see similar behaviour for the endemic level, when $F \to \infty$, see Figure 2.10.

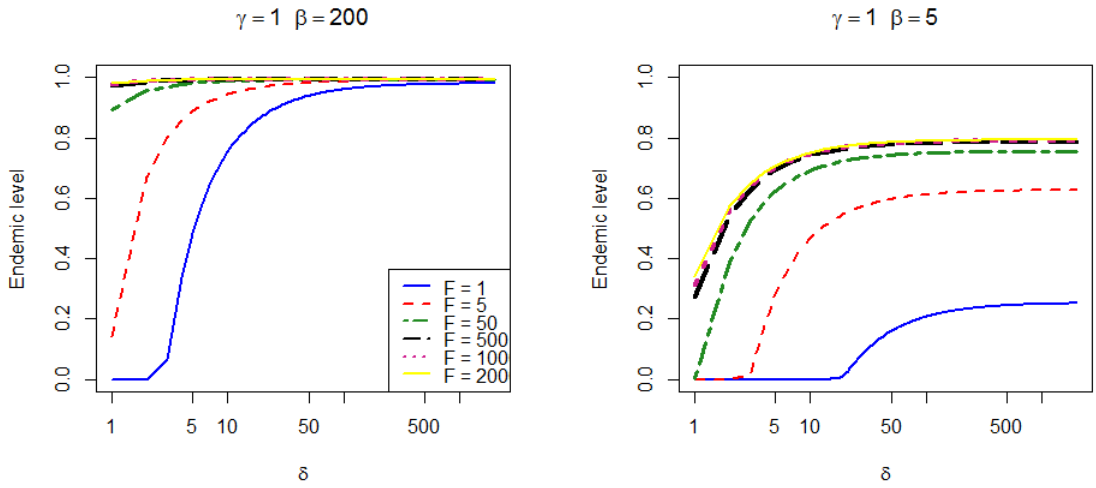


Figure 2.10: Plot of endemic level against δ

2.7.3 Mean time to extinction and the fluctuations about the endemic level

To study the mean time to extinction, the stochastic SIS simulations are utilised and the results are calculated from 1000 simulations. We vary β_1 from 13 to 33, whilst keeping other parameter fixed; $\gamma_1 = 1, \gamma_2 = 2, \delta = 6, \alpha = 9, \beta_2 = 18$. These parameter sets are illustrative.

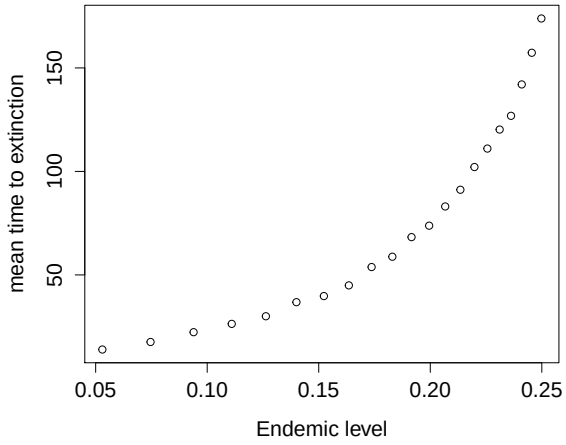


Figure 2.11: Plot of mean time to extinction against endemic level when $\gamma_1 = 1, \gamma_2 = 2, \delta = 6, \alpha = 9, \beta_2 = 18$ and β_1 varying by 1 from 13 to 33.

As shown in Figure 2.11, we can see that the mean time to extinction is 0 when initially the population size (endemic level) is 0 and increases with the endemic level. It has been shown that, the time to extinction when the epidemic is started from the endemic level is exponentially distributed, see Andersson & Djehiche (1998), Hakoyama & Iwasa (2000).

What if the endemic level is fixed? It is interesting to study effects of the model parameters and how the mean time to extinction behaves in this case. Assume that the population size is 100, our fixed endemic level is 14 infectives (endemic level = 0.14), for each set of parameters. First, we vary the infection rates β_1 and β_2, whilst keeping other parameters fixed. In the second case of Table 2.7, we also vary β_1 and β_2, but in this case, $\gamma_1 \neq \gamma_2$. Table 2.8 presents the same phenomenon, but with parameter sets, where α and δ are varied. Note that the parameters used are rounded off to 2 decimal places whilst the results are rounded off to 4 decimal places for presentation.

Table 2.7: Time to extinction and the standard deviation (SD) of the total number of infectives with respect to each parameter set, with 14 initial infectives (endemic equilibrium) and population size 100, varying β_1 and β_2.

α	δ	γ_1	γ_2	β_1	β_2	SD	time to extinction
9	6	1.5	1.5	100	10	10.1342	30.3325
9	6	1.5	1.5	80.02	10.45	10.1207	29.7310
9	6	1.5	1.5	10.18	90.72	10.1285	30.2474
9	6	1.5	1.5	50.22	11.90	10.0880	30.9383
9	6	1	5	103.94	120.08	10.0313	32.7467
9	6	1	5	79.90	182.09	9.9796	33.9617
9	6	1	5	181.60	78.90	10.1128	31.8276
9	6	1	5	120.90	102.99	10.0570	32.4688
9	6	1	5	69.18	270.93	9.9474	33.2564
9	6	1	5	269.60	68.00	10.1517	32.4353
9	6	1	5	111.12	111.62	10.0430	33.1308

Table 2.7 shows that when we fix other parameters apart from β_1 and β_2, we can see that values of β_1 and β_2 do not have much effect on the fluctuation. This is because, in this model, the transmission can only occur within relationships. Therefore, high infection rates will affect the epidemic in such a way individuals

keep infecting each other within relationships. Since other parameters are fixed, we have similar end results of relationships (break ups). As a result, there is not much variation in behaviour of the epidemic.

Table 2.8: Time to extinction and the standard deviation (SD) of the total number of infectives with respect to each parameter set, with 14 initial infectives (endemic equilibrium) and population size 100, varying α and δ.

α	δ	γ_1	γ_2	β_1	β_2	SD	mean time to extinction
8.90	5.00	1	2	18	20	10.0274	36.0071
8.45	8.45	1	2	18	20	9.7201	33.1603
14.01	2.00	1	2	18	20	10.8262	48.7645
8.59	6.14	1	2	18	20	9.8819	35.1701
31.58	1.00	1	2	18	20	11.3977	59.7104
8.53	10.00	1	2	18	20	9.6051	34.2544
9	4.77	1	2	18	20	10.0632	36.1874
9.61	8.04	1.5	1.5	18	18	9.8472	29.3195
11.00	4.00	1.5	1.5	18	18	10.3277	35.3000
9.76	11.00	1.5	1.5	18	18	9.6923	26.4575
17.03	2.00	1.5	1.5	18	18	10.9338	41.7632

For the case where α and δ are varied, Table 2.8 shows a clearer result that, for fixed endemic level, the variance and the mean time to extinction could vary. As a result, we can say that an estimation of the persistence of the epidemic is not only relying upon the endemic level, but also the model parameters. For example, for high α and low δ, it means individuals stay in a relationship longer and quickly reform relationships when they do break up. Hence, individuals keep infecting each other within the relationship, and slowly move on to other susceptible individuals when they break up. This gives a slower move to the disease-free states. It seems to cause persistence of relationships where the epidemic is maintained.

What if both α and δ are really high? Table 2.9 presents the result for this case. Given α and δ are large, we require β_1 and β_2 to be high. We know that in this case, individuals are very active in terms of relationship formation and breaking up. They tend to have a very high chance of meeting new susceptible individuals, and with reasonably high β_1 and β_2, the epidemic becomes very active. This is an interesting behaviour as at the same endemic level, we obtain smaller standard

deviation and also shorter time to extinction compared to the results presented in Table 2.7 and 2.8.

Table 2.9: Time to extinction and the standard deviation of the total number of infectives with respect to each parameter set, with endemic level at 14 and population size 100, varying α and δ, for large α, δ.

α	δ	γ_1	γ_2	β_1	β_2	SD	time to extinction
1000	10000	1.5	1.5	38.4493	38.4493	9.2528	20.9236
800	10100	1.5	1.5	47.4227	47.4227	9.2731	21.2135
10000	1000	1.5	1.5	2.7299	2.7299	9.2558	23.7921

Now, we know that β does not have much effect on the mean time to extinction. Also, when α and δ are varied, the mean time to extinction is more affected but not in a substantive way. Next, we will consider the case where other parameters including γ_2 are allowed to be varied, whilst γ_1 is fixed to be 1.

Table 2.10: Mean to extinction and SD of the total number of infectives with respect to each parameter set, for fixed endemic level at 14 and population size 100, fix $\gamma_1 = 1$, allowing other parameters to vary.

γ_2	β_1	β_2	α	δ	SD	mean time to extinction
124.0645	5000	622.162	40	50	8.4697	30.9922
5.4542	15	8.2952	100	5	10.139	27.3974
0.0401	41.0168	1000	1	99	10.5436	2250
2	18.54479	20	8.59	6	9.8699	35.633
3.3	6.414945	3	264	10	9.6216	25.7637
1.13	124.0224	15	9	1	11.3229	78.1938
1	3.4758	50	14	2	10.6296	51.8529
5.4542	15	8.2952	100	5	10.1444	27.3974
0.7498	1	10.4524	150	100	10.2567	27.5222
15	100	200	243.6944	1	11.7499	57.0261
8	200	211	11	7.1065	9.8842	31.1288
2.1464	4	52	30	3	10.1384	38.0144
0.05	0.1540	30	9	10	10.9911	221.5650
0.01	1.68606	9	0.5	0.8	8.0655	2920

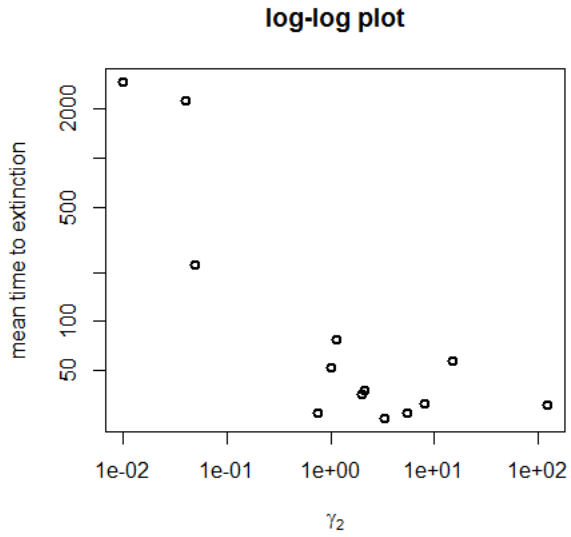


Figure 2.12: Plot of the mean time to extinction against γ_2 corresponding to results in Table 2.10

According to Figure 2.12, when we fix $\gamma_1 = 1$ whilst varying other parameters, the mean time to extinction does not depend upon any other parameters in any obvious way apart from γ_2. We can clearly see a decreasing trend as γ_2 increases. This shows that γ_2 is a dominant influence in the mean time to extinction.

Now, in order to study a wider range of parameters, we consider an endemic level of 25% of the population infected. The study is similar to the previous study for 14% endemic level. First, we fix $\gamma_1 = 1$ and consider varying (β_1, β_2) whilst keeping the other parameters fixed.

Table 2.11: the mean time to extinction and the standard deviation (SD) of the total number of infectives with respect to each parameter set, for fixed endemic level at 25 and population size 100, varying β_1 and β_2.

γ_1	γ_2	β_1	β_2	α	δ	SD	mean time to extinction
1	0.1	20.74605	15	9	0.1	11.7712	1078.20
1	0.1	13.37569	25	9	0.1	12.0745	1036.80
1	0.1	40	10.94915	9	0.1	11.6239	1088.40
1	0.1	100	9.2585	9	0.1	11.4729	1010.10
1	0.1	8.57131	1000	9	0.1	12.5209	1078.10

According to Table 2.11, there are small changes in the mean time to extinction

and the standard deviation even when there is a high level of variation of the infection parameter which is similar to the results found in Table 2.7. We now also consider varying γ_2. The results in Table 2.12 show high variation in variance and the mean time to extinction. For visualisation, we plot variance and the mean time to extinction against γ_2 on the log scale.

Table 2.12: The mean time to extinction and the SD of the total number of infectives with respect to each parameter set, for fixed endemic level at 25 and population size 100, fix $\gamma_1 = 1$ and $\alpha = 9$, allowing other parameters to vary

γ_2	β_1	β_2	α	δ	SD	mean time to extinction
0.1089641	20	20	9	0.1	11.9053	2478.05
0.2723315	1000	1000	9	0.1	11.7507	1946.05
0.01649724	3	3	9	0.1	18.5380	9968.30
0.1	5.162857	1	9	1	8.1803	2214.80
0.5	34.70589	5	9	1	10.0074	380.82
1	100000000	14.05631	9	1	10.5936	138.8699
1.136624	40	40	9	1	10.6438	270.3304
0.4777241	4	100	9	1	10.4552	319.6362
0.4684035	100	4	9	1	9.8989	390.7427
0.1	5.598458	1	9	10	7.3847	1921.40
0.5	1.787737	100	9	10	9.5383	170.8075
1	3.54577	10000	9	10	9.2636	143.5572
2.295557	30	25	9	10	9.0093	164.802
3.134849	30	80	9	10	8.8927	157.9564
1.29343	15	13	9	10	8.9874	187.0062

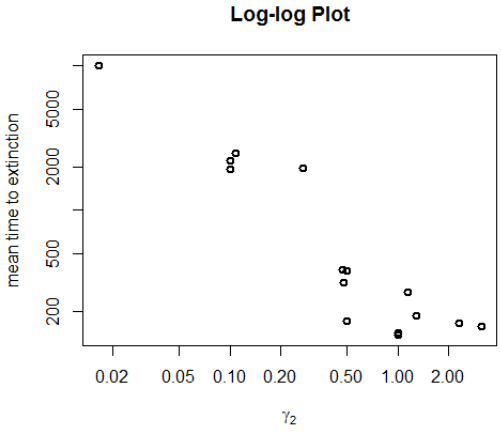


Figure 2.13: Plot the mean time to extinction against γ_2 corresponding to results in Table 2.12

As we can see in Figure 2.12 and 2.13 that the mean time to extinction has a clear decreasing trend with respect to γ_2, showing that γ_2 has an effect on the time to extinction. In terms of the time to extinction and the variance about the endemic level, we can see that they are not correlated in a simple manner. In other words, the variance does not give us any information on how long we would expect to wait on the mean for the epidemic to go extinct. Now, we will allow α to vary in order to observe whether or not α has a major influence on the mean time to extinction.

Table 2.13: Time to extinction and the SD of the total number of infectives with respect to each parameter set, for fixed endemic level at 25 and population size 100, fix $\gamma_1 = 1$, allowing other parameters to vary

γ_2	β_1	β_2	α	δ	SD	mean time to extinction
0.5	50	50	31.5250	0.1	11.1162	664.0552
0.0165	3	3	9	0.1	18.5370	9968.30
0.3	15	13	161.777	0.1	10.7652	244.4789
0.1	8.5713	1000	9	0.1	12.5209	1078.10
1	6.2338	6.2338	100	1	10.1185	165.4944
3	34	80.65	29.8408	1	10.8583	199.8821
0.4	100	4	7.2991	1	9.7729	469.7782
1.2	800	3	112.2586	1	10.5028	128.2327
1.1366	40	40	9	1	10.6438	270.3304
2	50	30	6.8848	10	9.0505	264.4116
2.28	150	120	5.3920	10	9.0575	152.4373
4.48	8	5	264.3126	10	8.9451	123.6751
0.2	1000	1000	1.2690	10	8.2679	1054.3
0.77	99	2	12.2224	10	9.1904	163.1799
1.2934	15	13	9	10	8.9874	187.0062

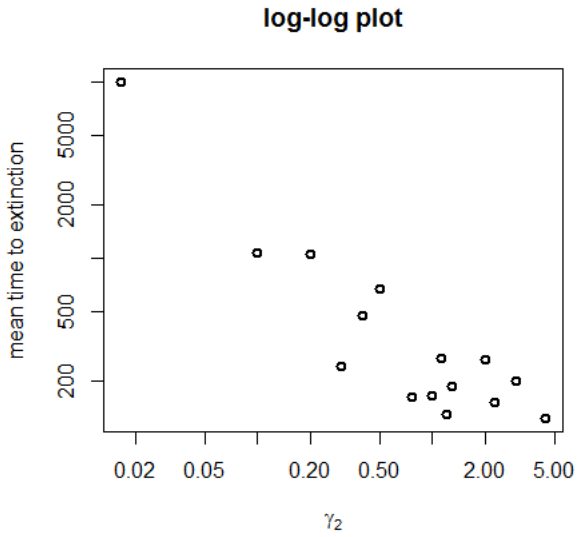


Figure 2.14: Plot of the mean time to extinction against γ_2 corresponding to results in Table 2.13

We can see from Figure 2.14 that the mean time to extinction has a decreasing trend in γ_2 showing that α does not have a major influence on the mean time to extinction. Precisely, for fixed γ_1, other parameters except γ_2 do not affect the mean time to extinction in a predictable way, in which it supports the previous studies for the endemic level 0.14 in Figure 2.12.

Chapter 3

Sexual network modelling with one-night stand condition.

3.1 Introduction

In Chapter 2, individuals in the population are assumed to be faithful (transmission of the STD within partnerships only). However, this is an unrealistic assumption and disregards an important disease dynamic that includes transmission of the disease outside partnerships. This could lead to a severe misunderstanding of STD dynamics. In this Chapter, we aim to achieve a more realistic model by extending the sexual network model formulated in Chapter 2, allowing for individuals in the population to be unfaithful (transmission of the STD outside partnerships). This includes allowing the transmission of the disease to occur between singles. Therefore, we need to modify the model described in Chapter 2.

The model formulation will be described in detail in Section 3.2. In Section 3.3, the reproduction number (R_0) is derived using a 5-type branching process approximation for the early stages of the epidemic instead of a 2-type branching process. The branching process approximation also gives us formulae for the probability that the disease goes extinct, which will be discussed in Section 3.4. Then, in

Section 3.5, we move away from the early stages of the epidemic and consider the epidemic at the endemic equilibrium. This will give us information about the endemic level including the proportion of infected individuals and fluctuations about the endemic level. The mean time to extinction is studied in Section 3.6 using stochastic modelling. Lastly, Section 3.7 discusses numerical results and comparison between the model presented in this Chapter with the model of Chapter 2. Note that the model given in Chapter 2 is a special case of the model considered in this Chapter with the parameter for one-night stands set equal to 0.

3.2 Model formulation

Assuming that everyone in the population is faithful is unrealistic. To achieve a more realistic model, we extend our previous model by adding one more condition in which the possibility of unfaithfulness is now taken into account. An individual is allowed to have casual one-off sexual experiences, which we shall term "one-night stands", with any random individual of the opposite sex regardless of whether the individual is in a relationship or not. Specifically, there are 3 possible events occurring in terms of one-night stands.

- A single individual has a one-night stand with another single individual of the opposite sex.
- A single individual has a one-night stand with an individual of the opposite sex in a relationship.
- Two individuals of the opposite sex in different relationships have one-night stands with each other.

The model is an extension of the model in Chapter 2 so many of the assumptions which were made in Chapter 2 still hold. However, there are new assumptions as well and we give full details of the assumptions of the model below.

1. The population is large and finite, consisting of two types of individuals: males (type 1 individuals) and females (type 2 individuals). We assume that the population has n individuals of each type, i.e. the numbers of males and females are equal.

2. The epidemic starts with one infected individual in an otherwise susceptible population.

3. A single male attempts to form a relationship with a female at the points of a homogeneous Poission point process with rate α. The female is chosen uniformly at random from the entire population. If the female individual is single, a relationship is formed, otherwise nothing happens.

4. The relationship length follows an exponential distribution with mean $\frac{1}{\delta}$. At the end of the relationship, the relationship breaks up and both individuals will return to the single state and will be able to form relationships with other single individuals of the opposite sex.

5. Whilst infectious, an individual of type i makes infectious contact with their partner at the points of a homogeneous Poisson point process with rate β_i. If the partner is susceptible, they become infected when contacted. Otherwise the contact has no effect. Note that single infectious individuals can not make such infectious contacts.

6. Type i individuals have infectious periods that are independently exponentially distributed with rate γ_i.

7. The model is a stochastic SIS epidemic model. Namely, an individual can be infected and after recovery the individual immediately becomes susceptible to reinfection.

8. We assume that at the start of the epidemic, the population relationship

structure is in equilibrium. Thus the proportion of the population in relationships (or single) remains fairly constant throughout the course of the epidemic. Let σ denote the proportion of the population single in equilibrium.

9. Individuals are allowed to have one-off sexual contacts with any individuals of the opposite sex. However, we are only interested in infectious one-night stands, in other words, one-night stands which result in an infectious contact. Note that a one-night stand between two infectives will result in infectious contacts which have no effect.

10. Infectious one-night stands attempted at rate ω_i, where i is the type of the individual who is infectious and attempting to have a one-night stand.

11. When an individual attempts to have a one-night stand, he/she attempts to have a one-night stand with an opposite sex who is either single or in a relationship. We let p represent the probability of an individual trying to have a one-night stand with someone who is in a relationship. Thus $p = (1 - \sigma)/2$, where $\sigma = \frac{\delta}{\bar{\alpha}+\delta}$. On the other hand, q is the probability of an individual trying to have a one-night stand with a single person, hence, $q = \sigma/2$.

12. Not every individual would agree to have a one-night stand, especially those who are in a relationship. Therefore, we define r as the willingness of someone in a relationship to have a one-night stand ($0 \leq r \leq 1$). Consequently, the probability of two individuals in a relationship agreeing to have a one-night stand is r^2. Note that $1 - r$ represents a reticence of individuals in relationships to have one-night stands. Note that, $r = 0$ means only single individuals partake in one-night stands. Whilst, $r = 1$ means everybody is equally likely to have a one night stand.

Using the model with a one-night stand condition, we aim to be able to answer various questions similar to those posed in Chapter 2. For example, the question arises as to how one-night stands have an effect on the incidence of the disease, the time to extinction if the disease appears to persist in the population before it goes extinct, and more importantly, how likely is the epidemic to take off? As such, the comparisons between the two models are investigated. The structure of the model will be described in Section 3.3.

3.3 5 - type branching process approximation

In terms of the mathematical structure, in this case, it is easier to express the process in terms of 5-types of infection units. Therefore instead of dividing individuals in the population into 2 types, we categorise them into 5 types corresponding to their disease and relationship status. Namely, we consider each of infected male, infected female, a couple within which the male is infected, a couple within which the female is infected, and a couple within which both are infected, as infective units. These types form the basis of our branching process approximation. The 5 types of individuals are defined as follows.

1. Type 1 - A single infected male, represented by M_I.
2. Type 2 - A couple within which only the male is infected, represented by $M_I F_S$.
3. Type 3 - A couple within which both the male and the female are infected, represented by $M_I F_I$.
4. Type 4 - A couple within which only the female is infected, represented by $M_S F_I$.
5. Type 5 - A single infected female, represented by F_I

Now, instead of starting the process with either an infected male or female, we can start the process with one of the 5 types defined above. For instance, if the process starts with an individual of type 2, $M_I F_S$, a couple within which the male is infected. Then, one of the following events could happen.

- The male recovers from the disease, the couple then becomes susceptible ($M_S F_S$). In this case, in the branching process approximation formulation we have no offspring.

- The infected male infects his partner, the couple then becomes infected (type 3, $M_I F_I$). In this case, we have a type 3 offspring.

- The infected male has a one-night stand with a single susceptible female, giving a single infected female (type 5, F_I) as a result. Note that the status of the couple remains unchanged ($M_I F_S$). In this case, we have a type 5 and a type 2 offspring.

- The infected male has a one-night stand with a non-single susceptible female, and she becomes infected, then we have a couple where the female is infected (type 4, $M_S F_I$), with the original couple unchanged. In this case, we have a type 4 and a type 2 offspring. Note that in the early stages, the number of infectives is small so that the probability of having a relationship with somebody whose partner is infected is close to 0.

- The relationship breaks up, giving a single infected male (type 1, M_I) and a single susceptible female. In this case, we have a type 1 offspring.

Note that in the early stages, with high probability those individuals contacted by an infective will be susceptible. From the possible events described above, we can see that the possible outcomes consist of single and non-single susceptible individuals (M_S, F_S, and $M_S F_S$), as well as individuals of type 1, 2, 3, 4 and 5. However, we are interested in the infected individuals only. A couple within which

either male or female is infected, or both are infected, is capable of infecting other individuals if one-night stands occur. Note that types 2, 3, and 4 correspond to couples in the epidemic process. Therefore, these 5 types are the secondary cases produced from the initial infective of each type, leading to a 5×5 next-generation matrix.

Throughout, we call infected individuals produced from the initial infective of the 5 types individuals as "offspring". Now, if we start the process with an initial infective of type 1 (male), the infected male will either form a relationship, recover from the disease, or have a one-night stand with either a single or non-single susceptible female. Figure 3.1 illustrates this situation. Each node represents offspring obtained from each activity and transition rates are represented by arrows.

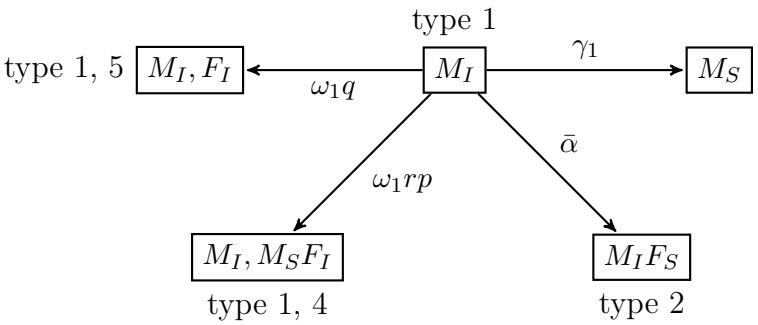


Figure 3.1: The 5- type branching process starting with an individual of type 1.

From Figure 3.1, if he has a one-night stand with a single susceptible female and she becomes infected, a type 1 and a type 5 offspring will be produced with rate $\omega_1 q$. With rate $\omega_1 rp$, he has a one night stand with a non-single susceptible female and she becomes infected, we have offspring of types 1 and 4. At the same time, he could form a relationship with rate $\bar{\alpha}$, having a type 2 offspring, or he could recover before anything happens with rate γ_1, and consequently having no offspring. We can see that there are 4 types of offspring produced from an infected male (type 1), from 4 possible events. Therefore, the probability of having a type 1 offspring from a type 1 initial infective is the probability of having a one-night

stand with a single or non-single female, that is $\frac{\omega_1 q}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp} + \frac{\omega_1 rp}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$. The probabilities of having offspring of other types are determined similarly. Hence, the secondary cases produced from an initial infective of type 1 and their probabilities are summarised as follows.

- type 1 offspring with probability $\frac{\omega_1 q+\omega_1 rp}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$.
- type 2 offspring with probability $\frac{\bar{\alpha}}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$.
- type 4 offspring with probability $\frac{\omega_1 rp}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$.
- type 5 offspring with probability $\frac{\omega_1 q}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$.

Another example is shown in Figure 3.2 for a couple with both infected (type 3).

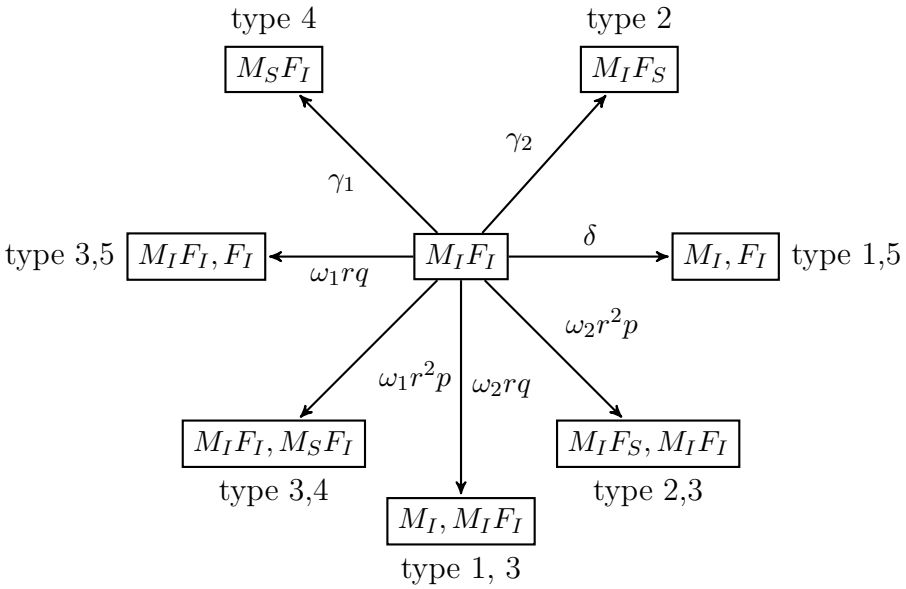


Figure 3.2: The 5-type branching process starting with an individual of type 3.

Figure 3.2 shows an example where the process starts with an individual of type 3, i.e. an infected couple. In this case, there are 7 possible events, which are the relationship breakups, the infected male recovers, the infected male has a one-night stand with a single susceptible female or has a one night stand with a

non-single susceptible female. These last three situations could also happen with the infected female with the roles of males and females reversed. As such, all 5 types of offspring can be produced. In Table 3.1, we summarise the process starting with each type of individual, together with the probabilities of having each offspring according to each event.

Initial infective$^{(type)}$	Probability	Offspring$^{(type)}$
$M_I^{(1)}$	$\frac{\gamma_1}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$	0
	$\frac{\bar{\alpha}}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$	$M_I F_S{}^{(2)}$
	$\frac{\omega_1 q}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$	$M_I{}^{(1)}$,$\mathrm{F}_I{}^{(5)}$
	$\frac{\omega_1 rp}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}$	$M_I{}^{(1)}$,$\mathrm{M}_S F_I{}^{(4)}$
$M_I F_S^{(2)}$	$\frac{\delta}{\delta+\gamma_1+\beta_1+\omega_1 rq+\omega_1 r^2 p}$	$M_I{}^{(1)}$
	$\frac{\gamma_1}{\delta+\gamma_1+\beta_1+\omega_1 rq+\omega_1 r^2 p}$	0
	$\frac{\beta_1}{\delta+\gamma_1+\beta_1+\omega_1 rq+\omega_1 r^2 p}$	$M_I F_I{}^{(3)}$
	$\frac{\omega_1 rq}{\delta+\gamma_1+\beta_1+\omega_1 rq+\omega_1 r^2 p}$	$F_I{}^{(5)}$,$\mathrm{M}_I F_S^{(2)}$
	$\frac{\omega_1 r^2 p}{\delta+\gamma_1+\beta_1+\omega_1 rq+\omega_1 r^2 p}$	$M_I F_S{}^{(2)}$,$\mathrm{M}_S F_I{}^{(4)}$
$M_I F_I^{(3)}$	$\frac{\delta}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$	$M_I{}^{(1)}$,$\mathrm{F}_I{}^{(5)}$
	$\frac{\gamma_1}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$	$M_S F_I{}^{(4)}$
	$\frac{\gamma_2}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$	$M_I F_S{}^{(2)}$
	$\frac{\omega_1 rq}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$	$M_I F_I{}^{(3)}$,$\mathrm{F}_I{}^{(5)}$
	$\frac{\omega_1 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$	$M_S F_I{}^{(4)}$,$\mathrm{M}_I F_I{}^{(3)}$
	$\frac{\omega_2 rq}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$	$M_I{}^{(1)}$,$\mathrm{M}_I F_I{}^{(3)}$
	$\frac{\omega_2 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$	$M_I F_S{}^{(2)}$,$\mathrm{M}_I F_I{}^{(3)}$

Table 3.1: Probabilities of having each type of offspring produced from an initial infective of each type.

Initial infective$^{(type)}$	Probability	Offspring$^{(type)}$
$M_S F_I^{(4)}$	$\frac{\delta}{\delta+\beta_2+\gamma_2+\omega_2 rq+\omega_2 r^2 p}$	$F_I^{(5)}$
	$\frac{\beta_2}{\delta+\beta_2+\gamma_2+\omega_2 rq+\omega_2 r^2 p}$	$M_I S_I^{(3)}$
	$\frac{\gamma_2}{\delta+\beta_2+\gamma_2+\omega_2 rq+\omega_2 r^2 p}$	0
	$\frac{\omega_2 rq}{\delta+\beta_2+\gamma_2+\omega_2 rq+\omega_2 r^2 p}$	$M_S F_I^{(4)}$, $M_I^{(1)}$
	$\frac{\omega_2 r^2 p}{\delta+\beta_2+\gamma_2+\omega_2 rq+\omega_2 r^2 p}$	$M_S F_I^{(4)}$, $M_I F_S^{(2)}$
$F_I^{(5)}$	$\frac{\gamma_2}{\bar{\alpha}+\gamma_2+\omega_2 q+\omega_2 rp}$	0
	$\frac{\bar{\alpha}}{\bar{\alpha}+\gamma_2+\omega_2 q+\omega_2 rp}$	$M_S F_I^{(4)}$
	$\frac{\omega_2 q}{\bar{\alpha}+\gamma_2+\omega_2 q+\omega_2 rp}$	$M_I^{(1)}$, $F_I^{(5)}$
	$\frac{\omega_2 rp}{\bar{\alpha}+\gamma_2+\omega_2 q+\omega_2 rp}$	$M_I F_S^{(2)}$, $F_I^{(5)}$

We now have the probabilities of having offspring produced from each type of an initial infective. The next-generation matrix can now be constructed as a 5×5 matrix, such that each element is the probability of having each type of offspring produced from each type of initial infective. The branching process is based upon infectious units in the epidemic which changes (produces offspring) each time an event occurs.

Now, we let M denote the next- generation matrix, therefore, m_{ij} represents the probability of offspring of type j produced from an initial infective of type i. Note that individuals produce only 0 or 1 offspring of a given type so the mean is equal to the probability of having an offspring of the given type. According to Table 3.1, Matrix M is constructed as follows.

The next-generation matrix

$$M = \begin{bmatrix} m_{1,1} & m_{1,2} & 0 & m_{1,4} & m_{1,5} \\ m_{2,1} & m_{2,2} & m_{2,3} & m_{2,4} & m_{2,5} \\ m_{3,1} & m_{3,2} & m_{3,3} & m_{3,4} & m_{3,5} \\ m_{4,1} & m_{4,2} & m_{4,3} & m_{4,4} & m_{4,5} \\ m_{5,1} & m_{5,2} & 0 & m_{5,4} & m_{5,5} \end{bmatrix}$$

where

$$m_{1,1} = \frac{\omega_1 q+\omega_1 rp}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}. \qquad m_{1,2} = \frac{\bar{\alpha}}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}.$$

$$m_{1,3} = 0. \qquad m_{1,4} = \frac{\omega_1 rp}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}.$$

$$m_{1,5} = \frac{\omega_1 q}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}. \qquad m_{2,1} = \frac{\delta}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p}.$$

$$m_{2,2} = \frac{\omega_1 rq+\omega_1 r^2 p}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p} \qquad m_{2,3} = \frac{\beta_1}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p}$$

$$m_{2,4} = \frac{\omega_1 r^2 p}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p} \qquad m_{2,5} = \frac{\omega_1 rq}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p}$$

$$m_{3,1} = \frac{\delta+\omega_2 rq}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)} \qquad m_{3,2} = \frac{\gamma_2+\omega_2 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$$

$$m_{3,3} = \frac{\omega_1 rq+\omega_1 r^2 p+\omega_2 rq+\omega_2 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)} \qquad m_{3,4} = \frac{\gamma_1+\omega_1 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$$

$$m_{3,5} = \frac{\delta+\omega_1 rq}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)} \qquad m_{4,1} = \frac{\omega_2 rq}{\gamma_2+\beta_2+\delta+\omega_2 rq+\omega_2 r^2 p}$$

$$m_{4,2} = \frac{\omega_2 r^2 p}{\gamma_2+\beta_2+\delta+\omega_2 rq+\omega_2 r^2 p} \qquad m_{4,3} = \frac{\beta_2}{\gamma_2+\beta_2+\delta+\omega_2 rq+\omega_2 r^2 p}$$

$$m_{4,4} = \frac{\omega_2 rq+\omega_2 r^2 p}{\gamma_2+\beta_2+\delta+\omega_2 q+\omega_2 rp} \qquad m_{4,5} = \frac{\delta}{\gamma_2+\beta_2+\delta+\omega_2 rq+\omega_2 r^2 p}$$

$$m_{5,1} = \frac{\omega_2 q}{\gamma_2+\bar{\alpha}+\omega_2 q+\omega_2 rp} \qquad m_{5,2} = \frac{\omega_2 rp}{\gamma_2+\bar{\alpha}+\omega_2 q+\omega_2 rp}$$

$$m_{5,3} = 0 \qquad m_{5,4} = \frac{\bar{\alpha}}{\gamma_2+\bar{\alpha}+\omega_2 q+\omega_2 rp}$$

$$m_{5,5} = \frac{\omega_2 q+\omega_2 rp}{\gamma_2+\bar{\alpha}+\omega_2 q+\omega_2 rp}.$$

Recall that the reproduction number R_0 is the dominant eigenvalue of the matrix M. However, solving R_0 algebraically for this case is complicated. Hence, for this model, we will calculate R_0 numerically using the R statistical language and the results will be presented in section 3.7.

The probability of having offspring produced from each type of individuals is not only useful for determining R_0, but also for determining the probability of extinction. In the special case where there are no gender differences in rates, $\beta_1 = \beta_2$, $\gamma_1 = \gamma_2$, $\omega_1 = \omega_2$, the transmission from male to female and from female to male are the same, the branching process can be considered as 3 types of individuals. Namely, the infective units consist of a single infective, a couple within which only one person is infected, a couple within both are infected. As a result, the 5-type branching process can be reduced to 3 types: a single infective (type1), a couple with one infective (type 2), a couple with both infectives (type

3). Hence, the next-generation matrix defined by K is a 3×3 matrix, as follows.

$$K = \begin{bmatrix} k_{1,1} & k_{1,2} & 0 \\ k_{2,1} & k_{2,2} & k_{2,3} \\ k_{3,1} & k_{3,2} & k_{3,3} \end{bmatrix} \tag{3.1}$$

where

$$\begin{aligned} k_{1,1} &= \tfrac{2\omega q+\omega rp}{\gamma+\bar{\alpha}+\omega q+\omega rp}. & k_{1,2} &= \tfrac{\bar{\alpha}+\omega rp}{\gamma+\bar{\alpha}+\omega q+\omega rp}. \\ k_{1,3} &= 0. & k_{2,1} &= \tfrac{\delta+\omega rq}{\beta+\gamma+\delta+\omega rq+\omega r^2p}. \\ k_{2,2} &= \tfrac{\omega rq+2\omega r^2p}{\beta+\gamma+\delta+\omega rq+\omega r^2p} & k_{2,3} &= \tfrac{\beta}{\beta+\gamma+\delta+\omega rq+\omega r^2p} \\ k_{3,1} &= \tfrac{2\delta+2\omega rq}{\delta+2\gamma+2\omega r^2p+2\omega qr} & k_{3,2} &= \tfrac{2\gamma+2\omega r^2p}{\delta+2\gamma+2\omega r^2p+2\omega qr} \\ k_{3,3} &= \tfrac{2\omega rq+2\omega r^2p}{\delta+2\gamma+2\omega r^2p+2\omega qr} \end{aligned}$$

Then, R_0 is the largest positive solution of the following cubic equation, $\lambda^3 - (k_{3,3}+k_{2,2}+k_{1,1})\lambda^2-(-k_{2,3}k_{3,2}+k_{2,2}k_{3,3}-k_{1,2}k_{2,1}+k_{1,1}k_{3,3}+k_{1,1}k_{2,2})\lambda-k_{1,1}k_{2,2}k_{3,3}+k_{1,1}k_{2,3}k_{3,2}+k_{1,2}k_{2,1}k_{3,3}-k_{1,2}k_{2,3}k_{3,1}=0$. This is useful in terms of mathematical analysis. If we set $\omega = 0$, Model 2 represents the same scenario as Model 1 in which there is no one-night stands. We are interested in the relationship between R_0 obtained from Model 1 and Model 2. Throughout this thesis where conducting comparison between models we define R_0^1 and R_0^2 as the reproduction number obtained from Model 1 and Model 2, respectively.

3.3.1 Relationship between R_0^1 and R_0^2

In this subsection, we study the relationship between the reproduction number obtained from Model 1 (R_0^1) and the reproduction number obtained from Model 2 (R_0^2). We focus on the special case where there is no distinction between the sexes, $\beta_1 = \beta_2$, $\gamma_1 = \gamma_2$, $\omega_1 = \omega_2$. As mentioned earlier, Model 1 is a special case

of Model 2 when $\omega = 0$. Now, we set $\omega = 0$, the next generation matrix, K, in (3.1) becomes

$$K = \begin{bmatrix} 0 & \frac{\bar{\alpha}}{\gamma+\bar{\alpha}} & 0 \\ \frac{\delta}{\delta+\gamma+\beta} & 0 & \frac{\beta}{\delta+\beta+\gamma} \\ \frac{2\delta}{\delta+2\gamma} & \frac{2\gamma}{\delta+2\gamma} & 0 \end{bmatrix} \tag{3.2}$$

To find eigenvalues of matrix K, we solve $det(K - \lambda I) = 0$, which is

$$\{(\gamma+\bar{\alpha})(\delta+\beta+\gamma)(\delta+2\gamma)\}\lambda^3 - \{2\gamma\beta(\gamma+\bar{\alpha}) + \delta\bar{\alpha}(\delta+2\gamma)\}\lambda - \bar{\alpha}\beta(2\delta) = 0. \tag{3.3}$$

Recall from (2.55), we have that

$$R_0^1 = \frac{\bar{\alpha}\delta(\delta+2\gamma+2\beta)}{(\gamma+\bar{\alpha})(\beta\delta+\delta^2+3\delta\gamma+2\gamma^2)} \tag{3.4}$$

It follows that

$$R_0^1 = 1 \iff \bar{\alpha}\beta\delta = \gamma\beta\delta + \gamma\delta^2 + 3\delta\gamma^2 + 2\gamma^3 + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 \tag{3.5}$$

$$R_0^1 > 1 \iff \bar{\alpha}\beta\delta > \gamma\beta\delta + \gamma\delta^2 + 3\delta\gamma^2 + 2\gamma^3 + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 \tag{3.6}$$

$$R_0^1 < 1 \iff \bar{\alpha}\beta\delta < \gamma\beta\delta + \gamma\delta^2 + 3\delta\gamma^2 + 2\gamma^3 + \bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 \tag{3.7}$$

Rewriting equation (3.3), we have

$$A\lambda^3 - B\lambda - C = 0, \tag{3.8}$$

where,

$$A = (\gamma + \bar{\alpha})(\delta + \beta + \gamma)(\delta + 2\gamma) > 0$$
$$B = 2\gamma\beta(\gamma + \bar{\alpha}) + \delta\bar{\alpha}(\delta + 2\gamma) > 0$$
$$C = \bar{\alpha}\beta(2\delta) > 0.$$

Let k be the largest solution to equation (3.8). Therefore, the following equation holds.

$$Ak^3 - Bk = C \tag{3.9}$$

Because

$$\begin{aligned}
Ak^3 &= (\gamma + \bar{\alpha})(\delta + \beta + \gamma)(\delta + 2\gamma)k^3 \\
&= (2\bar{\alpha}\beta\gamma + \bar{\alpha}\delta^2 + 3\bar{\alpha}\delta\gamma + 2\beta\gamma^2 + \bar{\alpha}\beta\delta + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3)k^3 \\
&= (2\bar{\alpha}\beta\gamma + \bar{\alpha}\delta^2 + 2\bar{\alpha}\delta\gamma + 2\beta\gamma^2)k^3 + (\bar{\alpha}\delta\gamma + \bar{\alpha}\beta\delta + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3)k^3 \\
Bk &= (2\gamma\beta(\gamma + \bar{\alpha}) + \delta\bar{\alpha}(\delta + 2\gamma))k \\
&= (2\bar{\alpha}\beta\gamma + \bar{\alpha}\delta^2 + 2\bar{\alpha}\delta\gamma + 2\beta\gamma^2)k \\
C &= \bar{\alpha}\beta(2\delta).
\end{aligned}$$

Then by (3.9) we have

$$\begin{aligned}
2\bar{\alpha}\beta\delta &= (2\bar{\alpha}\beta\gamma + \bar{\alpha}\delta^2 + 2\bar{\alpha}\delta\gamma + 2\beta\gamma^2)k^3 + (\bar{\alpha}\delta\gamma + \bar{\alpha}\beta\delta + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3)k^3 \\
&\quad - (2\bar{\alpha}\beta\gamma + \bar{\alpha}\delta^2 + 2\bar{\alpha}\delta\gamma + 2\beta\gamma^2)k \\
&\quad = (2\bar{\alpha}\beta\gamma + \bar{\alpha}\delta^2 + 2\bar{\alpha}\delta\gamma + 2\beta\gamma)(k^3 - k) + (\bar{\alpha}\beta\delta)k^3 \\
&\quad + (\bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3)k^3
\end{aligned}$$

Rearranging to get $(\bar{\alpha}\beta\delta)k^3$ on the left hand side and using the fact that $(\bar{\alpha}\beta\delta)k^3 = 2(\bar{\alpha}\beta\delta)k^3 - (\bar{\alpha}\beta\delta)k^3$, we have

$$\begin{aligned}(\bar{\alpha}\beta\delta)k^3 = (2\bar{\alpha}\beta\gamma + \bar{\alpha}\delta^2 + 2\bar{\alpha}\delta\gamma + 2\beta\gamma)k(k^2-1) + 2(\bar{\alpha}\beta\delta)(k^3-1) \\ + (\bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3)k^3 \qquad (3.10)\end{aligned}$$

Now, we consider 3 possible k situations, $k < 1$, $k > 1$ and $k = 1$.
If $k = 1$, (3.10) is satisfied if and only if

$$\begin{aligned}&(\bar{\alpha}\beta\delta) = (\bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3) \\ \iff \quad &R_0^1 = 1 \qquad \text{by} \quad (3.5). \qquad (3.11)\end{aligned}$$

If $k < 1$, we can see that the first and the second term on the right hand side of equation (3.10) are negative. This gives

$$\begin{aligned}&(\bar{\alpha}\beta\delta)k^3 < (\bar{\alpha}\beta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3)k^3 \\ &(\bar{\alpha}\beta\delta) < (\bar{\alpha}\delta\gamma + +2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3) \\ \iff \quad &R_0^1 < 1 \qquad \text{by} \quad (3.7). \qquad (3.12)\end{aligned}$$

Similarly, if $k > 1$, the first and the second term on the right hand side are positive. We have

$$\begin{aligned}&(\bar{\alpha}\beta\delta)k^3 > (\bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3)k^3 \\ &(\bar{\alpha}\beta\delta) > (\bar{\alpha}\delta\gamma + 2\bar{\alpha}\gamma^2 + \beta\delta\gamma + \delta^2\gamma + 3\delta\gamma^2 + 2\gamma^3) \\ \iff \quad &R_0^1 > 1 \qquad \text{by} \quad (3.6). \qquad (3.13)\end{aligned}$$

Since k is the largest solution that solves equation (3.3), it is a dominant eigenvalue of Matrix K which is equivalent to the reproduction numberof Model 2, R_0^2. As a

result, we conclude that $R_0^1 \leq 1$ if and only if $R_0^2 \leq 1$.

3.4 Extinction probability

We now turn our attention to the extinction probability of the approximating branching process. Let x_i be the number of offspring of type i, where $i \in \{1, 2, 3, 4, 5\}$, $x_i \in \{0, 1\}$. Let $\mathbf{s} = (s_1, s_2, s_3, s_4, s_5)$, where $0 \leq s_1, s_2, s_3, s_4, s_5 \leq 1$. Let $p_{\mathbf{x}}^{(k)}$ is the probability of having $\mathbf{x}$ offspring produced from an initial infected individual of type k, where $\mathbf{x} = (x_1, x_2, x_3, x_4, x_5)$. The probability generating function for the number of offspring of a type k individual can be written as,

$$g_k(\boldsymbol{s}) = \sum_{x_1,x_2,x_3,x_4,x_5} p^{(k)}_{x_1,x_2,x_3,x_4,x_5} s_1^{x_1} s_2^{x_2} s_3^{x_3} s_4^{x_4} s_5^{x_5}. \tag{3.14}$$

Note that if vector $\mathbf{x} = \mathbf{0}$, it means that the individual has no offspring of any type. Therefore, $p_{\mathbf{0}}^{(k)}$ represents the probability of having 0 offspring produced from an initial infective of type k. For convenience, if there exists $i, j \in \{1, 2, ..., 5\}$ such that for $k \neq i$ or j, $x_k = 0$ we write $\mathbf{x} = (x_i, x_j)$. For example, $(0, 0, x_3, 0, x_5)$ is abbreviated as (x_3, x_5). Thus, we express the probability generating functions as follows.

$$\begin{aligned}
g_1(\mathbf{s}) &= p_{\mathbf{0}}^{(1)} + p_{(x_2)}^{(1)} s_2 + p_{(x_1,x_5)}^{(1)} s_1 s_5 + p_{(x_1,x_4)}^{(1)} s_1 s_4. \\
g_2(\mathbf{s}) &= p_{\mathbf{0}}^{(2)} + p_{(x_1)}^{(2)} s_1 + p_{(x_3)}^{(2)} s_3 + p_{(x_2,x_5)}^{(2)} s_2 s_5 + p_{(x_2,x_4)}^{(2)} s_2 s_4. \\
g_3(\mathbf{s}) &= p_{(x_1,x_5)}^{(3)} s_1 s_5 + p_{(x_4)}^{(3)} s_4 + p_{(x_2)}^{(3)} s_2 + p_{(x_3,x_5)}^{(3)} s_3 s_5 + p_{(x_3,x_4)}^{(3)} s_3 s_4 \\
&\quad + p_{(x_1,x_3)}^{(3)} s_1 s_3 + p_{(x_2,x_3)}^{(3)} s_2 s_3. \\
g_4(\mathbf{s}) &= p_{\mathbf{0}}^{(4)} + p_{(x_5)}^{(4)} s_5 + p_{(x_3)}^{(4)} s_3 + p_{(x_1,x_4)}^{(4)} s_1 s_4 + p_{(x_2,x_4)}^{(4)} s_2 s_4. \\
g_5(\mathbf{s}) &= p_{\mathbf{0}}^{(5)} + p_{(x_4)}^{(5)} s_4 + p_{(x_1,x_5)}^{(5)} s_1 s_5 + p_{(x_2,x_5)}^{(5)} s_2 s_5.
\end{aligned} \tag{3.15}$$

Let π_i denote the probability of extinction given that an infected individual

of type i is introduced into the population. Let $\boldsymbol{\pi} = (\pi_1, \pi_2, \pi_3, \pi_4, \pi_5)$. The probability of extinction is then the non-negative root of the equations

$$\boldsymbol{g}(\boldsymbol{\pi}) = (g_1(\pi), g_2(\pi), g_3(\pi), g_4(\pi), g_5(\pi)) = \boldsymbol{\pi}. \tag{3.16}$$

It is not possible to obtain a general analytical solution. Therefore, it will be solved numerically using R programming language. In the special case where there is no differences in rates between male and female the probability of extinction can be found using a 3-type branching process.

3.4.1 Probability of extinction for the special case $\beta_1 = \beta_2, \gamma_1 = \gamma_2, \omega_1 = \omega_2$

Considering the case where $\beta_1 = \beta_2, \gamma_1 = \gamma_2, \omega_1 = \omega_2$, then male offspring and female offspring have identical probability distribution. As a result, the mean number of offspring of the following types are equal, $M_I = F_I$, and $M_I F_S = M_S F_I$. Therefore, it is clear to see that $\pi_1 = \pi_5$, and $\pi_2 = \pi_4$. In other words, there is no gender distinction, therefore we can reduce types of singles and couples. Hence, we have 3 types of offspring: a single infective represented by S_I, a couple within which one person is infected represented by C_{1I}, a couple within which two persons are infected represented by C_{2I}. As such, in terms of branching process approximation, we can reduce a 5-type branching process into 3-type branching process in this case. The corresponding probability generating functions are expressed as in 3.17.

$$\begin{aligned}
g_1(\mathbf{s}) &= p^{(1)}_{\mathbf{0}} + p^{(1)}_{(x_2)} s_2 + p^{(1)}_{(x_1,x_5)} s_1^2 + p^{(1)}_{(x_1,x_4)} s_1 s_2. \\
g_2(\mathbf{s}) &= p^{(2)}_{\mathbf{0}} + p^{(2)}_{(x_1)} s_1 + p^{(2)}_{(x_3)} s_3 + p^{(2)}_{(x_2,x_5)} s_2 s_1 + p^{(2)}_{(x_2,x_4)} s_2^2. \\
g_3(\mathbf{s}) &= p^{(3)}_{(x_1,x_5)} s_1^2 + 2p^{(3)}_{(x_2)} s_2 + p^{(3)}_{(x_3,x_5)} s_3 s_1 + p^{(3)}_{(x_3,x_2)} s_3 s_2 + p^{(3)}_{(x_1,x_3)} s_1 s_3 \\
&\quad + p^{(3)}_{(x_3,x_2)} s_3 s_2.
\end{aligned} \tag{3.17}$$

Giving,

$$(g_1(\pi), g_2(\pi), g_3(\pi)) = \boldsymbol{\pi}, \quad \text{where} \quad \boldsymbol{\pi} = (\pi_1, \pi_2, \pi_3). \tag{3.18}$$

To seek the solutions of equations (3.16) and (3.18), deriving them algebraically appears to be too complex. However, we know that if $R_0 \leq 1$, then $\boldsymbol{\pi} = 1$. If $R_0 > 1$, then $\boldsymbol{\pi} < 1$. We use R software to determine the numerical results, see Section 3.7.

3.5 Endemic Level

As in Section 2.5, we explore the endemic level of the disease. To study the endemic level, we employ the deterministic model as shown in Section 3.5.1. To study the fluctuations about endemic level, an Ornstein-Uhlenbeck approximation is utilised, see Section 3.5.2. The mean time to extinction is investigated using stochastic simulations described in Section 3.6.

3.5.1 Deterministic Model

Similar to Section 2.5.1, the population is classified into 8 disjoint classes according to sex, disease status and relationship status. Recall the definition of each class as follows.

$x_0(t)$ denotes the proportion of non-infected single males at time t,

$x_1(t)$ denotes the proportion of infected single males at time t,

$y_0(t)$ denotes the proportion of non-infected single females at time t,

$y_1(t)$ denotes the proportion of infected single females at time t,

$z_{10}(t)$ denotes the proportion of couples with an infected male only at time t,

$z_{01}(t)$ denotes the proportion of couples with an infected female only at time t,

$z_{11}(t)$ denotes the proportion of couples with both an infected male and female

at time t,

$z_{00}(t)$ denotes the proportion of couples with no infected at time t,

Then, the deterministic counterpart to the stochastic model including the one-night stand condition is described by the following system of differential equations:

$$
\begin{aligned}
\frac{dx_0(t)}{dt} &= \gamma_1 x_1(t) + \delta(z_{01}(t) + z_{00}(t)) - \alpha x_0(t)(y_1(t) + y_0(t)) - \omega_2 x_0(t) y_1(t) \\
&\quad - \omega_2 r x_0(t)(z_{01}(t) + z_{11}(t)) \\
\frac{dx_1(t)}{dt} &= -\gamma_1 x_1(t) + \delta(z_{11}(t) + z_{10}(t)) - \alpha x_1(t)(y_1(t) + y_0(t)) + \omega_2 x_0(t) y_1(t) \\
&\quad + \omega_2 r x_0(t)(z_{01}(t) + z_{11}(t)) \\
\frac{dy_0(t)}{dt} &= \gamma_2 y_1(t) + \delta(z_{10}(t) + z_{00}(t)) - \alpha y_0(t)(x_1(t) + x_0(t)) - \omega_1 x_1(t) y_0(t) \\
&\quad - \omega_1 r y_0(t)(z_{10}(t) + z_{11}(t)) \\
\frac{dy_1(t)}{dt} &= -\gamma_2 y_1(t) + \delta(z_{11}(t) + z_{01}(t)) - \alpha y_1(t)(x_1(t) + x_0(t)) + \omega_1 x_1(t) y_0(t) \\
&\quad + \omega_1 r y_0(t)(z_{10}(t) + z_{11}(t)) \\
\frac{dz_{00}(t)}{dt} &= \gamma_1 z_{10}(t) + \gamma_2 z_{01}(t) + \alpha y_0(t) x_0(t) - \delta z_{00}(t) - \omega_2 r y_1(t) z_{00}(t) - \omega_1 r x_1(t) z_{00}(t) \\
&\quad - \omega_2 r^2 p z_{00}(t)(z_{01}(t) + z_{11}(t)) - \omega_1 r^2 z_{00}(t)(z_{10}(t) + z_{11}(t)) \\
\frac{dz_{10}(t)}{dt} &= \gamma_2 z_{11}(t) - \beta_1 z_{10}(t) - \gamma_1 z_{10}(t) + \alpha x_1(t) y_0(t) - \delta z_{10}(t) + \omega_2 r y_1(t) z_{00}(t) \\
&\quad + \omega_2 r^2 z_{00}(t)(z_{01}(t) + z_{11}(t)) - \omega_1 r x_1(t) z_{10}(t) - \omega_1 r^2 z_{10}(t)(z_{10}(t) + z_{11}(t)) \\
\frac{dz_{01}(t)}{dt} &= \gamma_1 z_{11}(t) - \gamma_2 z_{01}(t) - \beta_2 z_{01}(t) + \alpha x_0(t) y_1(t) - \delta z_{01}(t) + \omega_1 r x_1(t) z_{00}(t) \\
&\quad + \omega_1 r^2 z_{00}(t)(z_{10}(t) + z_{11}(t)) - \omega_2 r y_1(t) z_{01}(t) - \omega_2 r^2 z_{01}(t)(z_{11}(t) + z_{01}(t)) \\
\frac{dz_{11}(t)}{dt} &= \beta_1 z_{10}(t) + \beta_2 z_{01}(t) - \gamma_1 z_{11}(t) - \gamma_2 z_{11}(t) + \alpha y_1(t) x_1(t) - \delta z_{11}(t) \\
&\quad + \omega_1 r x_1(t) z_{10}(t) + \omega_1 r^2 z_{10}(t)(z_{10}(t) + z_{11}(t)) + \omega_2 r y_1(t) z_{01}(t) \\
&\quad + \omega_2 r^2 z_{01}(t)(z_{10}(t) + z_{11}(t)) \\
n(t) &= x_1(t) + x_0(t) + y_1(t) + y_0(t) + 2z_{00}(t) + 2z_{10}(t) + 2z_{01}(t) + 2z_{11}(t) = 1.
\end{aligned}
\tag{3.19}
$$

An equilibrium point $(\tilde{\mathbf{x}}, \tilde{\mathbf{y}}, \tilde{\mathbf{z}}) = (\tilde{x}_0, \tilde{x}_1, \tilde{y}_0, \tilde{y}_1, \tilde{z}_{00}, \tilde{z}_{10}, \tilde{z}_{01}, \tilde{z}_{11})$ is a solution of the simultaneous non-linear equations obtained by finding the stationary points to the system of differential equations defined above. Solving the system of differential equations of 8 dimensions is difficult, therefore, it will be solved numerically using R language and will be presented in Section 3.7.

3.5.2 Limiting Diffusion Process

As in Section 2.5.2, we study the fluctuations about endemic level using Ornstein-Uhlenbeck process. We seek the covariance matrix $\Sigma = (\sigma_{ij})$ such that it solves the matrix equation

$$\boldsymbol{D\Sigma} + \boldsymbol{\Sigma D^T} = -\boldsymbol{C} \tag{3.20}$$

where matrices $\boldsymbol{D}$ and $\boldsymbol{C}$ are the local drift and covariance matrix. Note that, the system of linear equations in (3.20) has a unique solution if and only if D is of full rank. We can see that the system of equations in (3.19) are not linearly independent, giving the rank of D less than 8 which is not of full rank. Therefore, to solve (3.20), with our assumption that the proportions of males and females are equal, i.e. $x_0(t) + x_1(t) = y_0(t) + y_1(t)$, similarly to Section 2.5.1, we can reduce the number of equations to 6.

$$Q_1(t) = x_0(t) = 0.5 - z_{10}(t) - z_{00}(t) - z_{01}(t) - z_{11}(t) - x_1(t) \tag{3.21}$$

$$Q_2(t) = y_0(t) = 0.5 - z_{10}(t) - z_{00}(t) - z_{01}(t) - z_{11}(t) - y_1(t) \tag{3.22}$$

Substituting (3.21) and (3.22) in (3.19), we have 6 equations in 6 variables, giving the local drift matrix D as 6×6 matrix, defined as follows. For convenience, let

$$Q_3(t) = 0.5 - z_{10}(t) - z_{00}(t) - z_{01}(t) - z_{11}(t) \tag{3.23}$$

Therefore, (3.19) can be reduced to 6 differential equations as follows

$$
\begin{aligned}
\frac{dx_1(t)}{dt} &= -\gamma_1 x_1(t) + \delta(z_{11}(t) + z_{10}(t)) - \alpha x_1(t) Q_3(t) + \omega_2 Q_1(t) y_1(t) \\
&\quad + \omega_2 r Q_1(t)(z_{01}(t) + z_{11}(t)) \\
\frac{dy_1(t)}{dt} &= -\gamma_2 y_1(t) + \delta(z_{11}(t) + z_{01}(t)) - \alpha y_1(t) Q_3(t) + \omega_1 x_1(t) Q_2(t) \\
&\quad + \omega_1 r Q_2(t)(z_{10}(t) + z_{11}(t)) \\
\frac{dz_{00}(t)}{dt} &= \gamma_1 z_{10}(t) + \gamma_2 z_{01}(t) + \alpha Q_1(t) Q_2(t) - \delta z_{00}(t) - \omega_2 r y_1(t) z_{00}(t) - \omega_1 r x_1(t) z_{00}(t) \\
&\quad - \omega_2 r^2 z_{00}(t)(z_{01}(t) + z_{11}(t)) - \omega_1 r^2 z_{00}(t)(z_{10}(t) + z_{11}(t)) \\
\frac{dz_{10}(t)}{dt} &= \gamma_2 z_{11}(t) - \beta_1 z_{10}(t) - \gamma_1 z_{10}(t) + \alpha x_1(t) Q_2(t) - \delta z_{10}(t) + \omega_2 r y_1(t) z_{00}(t) \\
&\quad + \omega_2 r^2 z_{00}(t)(z_{01}(t) + z_{11}(t)) - \omega_1 r x_1(t) z_{10}(t) - \omega_1 r^2 z_{10}(t)(z_{10}(t) + z_{11}(t)) \\
\frac{dz_{01}(t)}{dt} &= \gamma_1 z_{11}(t) - \gamma_2 z_{01}(t) - \beta_2 z_{01}(t) + \alpha Q_1(t) y_1(t) - \delta z_{01}(t) + \omega_1 r x_1(t) z_{00}(t) \\
&\quad + \omega_1 r^2 z_{00}(t)(z_{10}(t) + z_{11}(t)) - \omega_2 r y_1(t) z_{01}(t) - \omega_2 r^2 z_{01}(t)(z_{11}(t) + z_{01}(t)) \\
\frac{dz_{11}(t)}{dt} &= \beta_1 z_{10}(t) + \beta_2 z_{01}(t) - \gamma_1 z_{11}(t) - \gamma_2 z_{11}(t) + \alpha y_1(t) x_1(t) - \delta z_{11}(t) \\
&\quad + \omega_1 r x_1(t) z_{10}(t) + \omega_2 r y_1(t) z_{01}(t) + \omega_2 r^2 z_{01}(t)(z_{10}(t) + z_{11}(t)) \\
&\quad + \omega_1 r^2 z_{10}(t)(z_{10}(t) + z_{11}(t))
\end{aligned}
\tag{3.24}
$$

The local drift matrix ($\boldsymbol{D}$) and the covariance matrix ($\boldsymbol{C}$) corresponding to (3.20) are defined as follows.

$$\boldsymbol{D} = (d)_{i,j}, i, j \in \{1, 2, ..., 6\}$$

where,

$$
\begin{aligned}
d_{11} &= \frac{\partial}{\partial x_1}\frac{(dx_1)}{(dt)} = -\gamma_1 - \alpha Q_3^* - \omega_2 y_1^* - \omega_2 r(z_{01}^* + z_{11}^*) \\
d_{12} &= \frac{\partial}{\partial y_1}\frac{(dx_1)}{(dt)} = \omega_2 Q_1^* \\
d_{13} &= \frac{\partial}{\partial z_{00}}\frac{(dx_1)}{(dt)} = \alpha x_1^* - \omega_2 y_1^* - \omega_2 r(z_{11}^* + z_{01}^*) \\
d_{14} &= \frac{\partial}{\partial z_{10}}\frac{(dx_1)}{(dt)} = \delta + \alpha x_1^* - \omega_2 y_1^* - \omega_2 r(z_{11}^* + z_{01}^*) \\
d_{15} &= \frac{\partial}{\partial z_{01}}\frac{(dx_1)}{(dt)} = \alpha x_1^* - \omega_2 y_1^* - \omega_2 r(z_{11}^* + z_{01}^*) + \omega_2 r Q_1^* \\
d_{16} &= \frac{\partial}{\partial z_{11}}\frac{(dx_1)}{(dt)} = \delta + \alpha x_1^* - \omega_2 y_1^* - \omega_2 r(z_{11}^* + z_{01}^*) + \omega_2 r Q_1^*
\end{aligned}
$$

$$
\begin{aligned}
d_{21} &= \frac{\partial}{\partial x_1}\frac{(dy_1)}{(dt)} = \omega_1 Q_2^* \\
d_{22} &= \frac{\partial}{\partial y_1}\frac{(dy_1)}{(dt)} = -\gamma_2 - \alpha Q_3^* - \omega_1 x_1^* - \omega_1 r(z_{10}^* + z_{11}^*) \\
d_{23} &= \frac{\partial}{\partial z_{00}}\frac{(dy_1)}{(dt)} = \alpha y_1^* - \omega_1 x_1^* - \omega_1 r(z_{10}^* + z_{11}^*) \\
d_{24} &= \frac{\partial}{\partial z_{10}}\frac{(dy_1)}{(dt)} = \alpha y_1^* - \omega_1 x_1^* - \omega_1 r(z_{10}^* + z_{11}^*) + \omega_1 r Q_2^* \\
d_{25} &= \frac{\partial}{\partial z_{01}}\frac{(dy_1)}{(dt)} = \delta + \alpha y_1^* - \omega_1 x_1^* - \omega_1 r(z_{10}^* + z_{11}^*) \\
d_{26} &= \frac{\partial}{\partial z_{11}}\frac{(dy_1)}{(dt)} = \delta + \alpha y_1^* - \omega_1 x_1^* - \omega_1 r(z_{10}^* + z_{11}^*) + \omega_1 r Q_2^*
\end{aligned}
$$

$$d_{31} = \frac{\partial}{\partial x_1}\frac{(dz_{00})}{(dt)} = -\omega_1 r z^*_{00} - \alpha Q^*_2$$
$$d_{32} = \frac{\partial}{\partial y_1}\frac{(dz_{00})}{(dt)} = -\omega_2 r z^*_{00} - \alpha Q^*_1$$
$$d_{33} = \frac{\partial}{\partial z_{00}}\frac{(dz_{00})}{(dt)} = -\delta - \omega_2 r y^*_1 - \omega_1 r x^*_1 - \omega_2 r^2(z^*_{11} + z^*_{01}) - \omega_1 r^2(z^*_{10} + z^*_{11}) - \alpha(Q^*_1 + Q^*_2)$$
$$d_{34} = \frac{\partial}{\partial z_{10}}\frac{(dz_{00})}{(dt)} = -r^2\omega_1 z^*_{00} + \gamma_1 - \alpha(Q^*_1 + Q^*_2)$$
$$d_{35} = \frac{\partial}{\partial z_{01}}\frac{(dz_{00})}{(dt)} = -r^2\omega_2 z^*_{00} + \gamma_2 - \alpha(Q^*_1 + Q^*_2)$$
$$d_{36} = \frac{\partial}{\partial z_{11}}\frac{(dz_{00})}{(dt)} = -r^2\omega_1 z^*_{00} - r^2\omega_2 z^*_{00} - \alpha(Q^*_1 + Q^*_2)$$

$$d_{41} = \frac{\partial}{\partial x_1}\frac{(dz_{01})}{(dt)} = \alpha Q^*_2 - \omega_1 r z^*_{10}$$
$$d_{42} = \frac{\partial}{\partial y_1}\frac{(dz_{01})}{(dt)} = r\omega_2 z^*_{00} - \alpha x^*_1$$
$$d_{43} = \frac{\partial}{\partial z_{00}}\frac{(dz_{01})}{(dt)} = -\alpha x^*_1 + \omega_2 r y^*_1 + \omega_2 r^2(z^*_{11} + z^*_{01})$$
$$d_{44} = \frac{\partial}{\partial z_{10}}\frac{(dz_{01})}{(dt)} = -\beta_1 - \gamma_1 - \alpha x^*_1 - \delta - \omega_1 r x^*_1 - \omega_1 r^2(z^*_{10} + z^*_{11}) - \omega_1 r^2 z^*_{10}$$
$$d_{45} = \frac{\partial}{\partial z_{01}}\frac{(dz_{01})}{(dt)} = \omega_2 r^2 z^*_{00} - \alpha x^*_1$$
$$d_{46} = \frac{\partial}{\partial z_{11}}\frac{(dz_{01})}{(dt)} = -r^2\omega_1 z^*_{10} + r^2\omega_2 z^*_{00} - \alpha x^*_1 + \gamma_2$$

$$d_{51} = \frac{\partial}{\partial x_1}\frac{(dz_{10})}{(dt)} = r\omega_1 z^*_{00} - \alpha y^*_1$$
$$d_{52} = \frac{\partial}{\partial y_1}\frac{(dz_{10})}{(dt)} = \alpha Q^*_1 - \omega_2 r z^*_{01}$$
$$d_{53} = \frac{\partial}{\partial z_{00}}\frac{(dz_{10})}{(dt)} = -\alpha y^*_1 + \omega_1 r x^*_1 + \omega_1 r^2(z^*_{10} + z^*_{11})$$
$$d_{54} = \frac{\partial}{\partial z_{10}}\frac{(dz_{10})}{(dt)} = r^2\omega_1 z^*_{00} - \alpha y^*_1$$
$$d_{54} = \frac{\partial}{\partial z_{01}}\frac{(dz_{10})}{(dt)} = -\gamma_2 - \beta_2 - \alpha y^*_1 - \delta - \omega_2 r y^*_1 - \omega_2 r^2(z^*_{11} + z^*_{01}) - \omega_2 r^2 z^*_{01}$$
$$d_{56} = \frac{\partial}{\partial z_{11}}\frac{(dz_{10})}{(dt)} = r^2\omega_1 z^*_{00} - r^2\omega_2 z^*_{01} - \alpha y^*_1 + \gamma_1$$
$$d_{61} = \frac{\partial}{\partial x_1}\frac{(dz_{11})}{(dt)} = r\omega_1 z^*_{10} + \alpha y^*_1$$
$$d_{62} = \frac{\partial}{\partial y_1}\frac{(dz_{11})}{(dt)} = \alpha x^*_1 + r\omega_2 z^*_{01}$$
$$d_{63} = \frac{\partial}{\partial z_{00}}\frac{(dz_{11})}{(dt)} = 0$$
$$d_{64} = \frac{\partial}{\partial z_{10}}\frac{(dz_{11})}{(dt)} = \beta_1 + \omega_1 r x^*_1 + \omega_1 r^2(z^*_{10} + z^*_{11}) + \omega_1 r^2 z^*_{10}$$
$$d_{65} = \frac{\partial}{\partial z_{01}}\frac{(dz_{11})}{(dt)} = \beta_2 + \omega_2 r y^*_1 + \omega_2 r^2(z^*_{01} + z^*_{11}) + \omega_2 r^2 z^*_{01}$$
$$d_{66} = \frac{\partial}{\partial z_{11}}\frac{(dz_{11})}{(dt)} = r^2\omega_1 z^*_{10} - \delta - \gamma_1 - \gamma_2 + r^2\omega_1 z^*_{10}$$

and the covariance matrix,

$$\boldsymbol{C} = (c)_{i,j}, i, j \in \{1, 2, ..., 6\}$$

where,

$$c_{1,1} = \alpha x^*_1 Q^*_2 + \alpha x^*_1 y^*_1 + \delta z^*_{10} + \delta z^*_{11} + \gamma_1 x^*_1 \quad c_{1,2} = \alpha x^*_1 y^*_1 + \delta z^*_{11}$$
$$c_{1,3} = 0 \quad c_{1,4} = -\delta z^*_{10} - \alpha x^*_1 Q^*_2$$
$$c_{1,5} = 0 \quad c_{1,6} = -\alpha x^*_1 y^*_1 - \delta z^*_{11}$$
$$c_{2,2} = \alpha Q^*_1 y^*_1 + \alpha x^*_1 y^*_1 + \delta z^*_{01} + \delta z^*_{11} + \gamma_2 y^*_1 \quad c_{2,3} = 0$$
$$c_{2,4} = 0 \quad c_{2,5} = -\alpha Q^*_1 y^*_1 - \delta z^*_{01}$$
$$c_{2,6} = -\alpha x^*_1 y^*_1 - \delta z^*_{11}$$

$$c_{3,3} = \alpha Q_1^* Q_2^* + \delta z_{00}^* + \gamma_1 z_{10}^* + \gamma_2 z_{01}^* \qquad c_{3,4} = -\gamma_1 z_{10}^*$$
$$c_{3,5} = -\gamma_2 z_{01}^* \qquad c_{3,6} = 0$$
$$c_{4,4} = \alpha x_1^* Q_2^* + \delta z_{10}^* + \beta_1 z_{10}^* + \gamma_2 z_{11}^* + \gamma_1 z_{10}^* \qquad c_{4,5} = 0$$
$$c_{4,6} = -\beta_1 z_{10}^* - \gamma_2 z_{11}^*$$
$$c_{5,5} = \alpha Q_1^* y_1^* + \delta z_{01}^* + \beta_2 z_{01}^* + \gamma_2 z_{01}^* + \gamma_1 z_{11}^* \qquad c_{5,6} = -\beta_2 z_{01}^* - \gamma_1 z_{11}^*$$
$$c_{6,6} = \alpha x_1^* y_1^* + \delta z_{11}^* + \beta_1 z_{10}^* + \beta_2 z_{01}^* + \gamma_1 z_{11}^* + \gamma_2 z_{11}^*$$

$\boldsymbol{C}$ is a symmetric matrix and the remaining covariance terms are 0. Note that, Q_1^*, Q_2^* and Q_3^* are defined in (3.21), (3.22) and (3.23) with equilibrium values $\boldsymbol{x^*}, \boldsymbol{y^*}, \boldsymbol{z^*}$. According to Section 1.3.2 in Chapter 1, if we choose an equilibrium point as an initial value of the deterministic approximation, for large N, the process $\sqrt{N}((X_N, Y_N, Z_N) - (\mathbf{x}^*, \mathbf{y}^*, \mathbf{z}^*))$ is approximated by the Ornstein-Uhlenbeck process, which has a Gaussian stationary distribution with mean-zero and covariance matrix Σ defined in (3.20). Similar to Chapter 2, with the same definition of the variables, we have the following

$$E\left[\hat{I}\right] = Ni^* = N(x_1^* + y_1^* + z_{10}^* + z_{01}^* + 2z_{11}^*),$$
$$Var(\hat{I}) = N\boldsymbol{\Sigma_i} = N(\varsigma_{11} + \varsigma_{22} + \varsigma_{44} + \varsigma_{55} + 4\varsigma_{66} + 2\varsigma_{12} + 2\varsigma_{14}$$
$$+ 2\varsigma_{15} + 4\varsigma_{16} + 2\varsigma_{24} + 2\varsigma_{25} + 4\varsigma_{26} + 2\varsigma_{45} + 4\varsigma_{46} + 4\varsigma_{56}),$$

The coefficient of variation is $C_v = \frac{\sqrt{N\Sigma_i}}{i^*}$. We are interested in how the coefficient of variation (C_v) tell us about the fluctuations about the endemic level by looking at the relationship between the C_v and the endemic level as well as the mean time to extinction for various parameter sets. The experiments will be done numerically in Section 3.7.

3.6 Time to extinction

To study the expected time to extinction of the epidemic given that the process is started in the endemic level, we use Stochastic simulation as in Section 2.6. The set of the state space is again

$$S = \{X_0(t), X_1(t), Y_0(t), Y_1(t), Z_{00}(t), Z_{10}(t), Z_{01}(t), Z_{11}(t)\}$$

As shown in Table 3.2, we have 34 events instead of 16. Recall that a_i represent a rate with respect to process i, where $i \in \{1, 2, 3, ..., 34\}$. Table 3.2 presents the rate corresponding to each event in the epidemic process.

Table 3.2: Events and their transition rates

a_i	Rate	Event
a_1	$\gamma_1 X_1$	Recovery of a single male.
a_2	$\gamma_2 Y_1$	Recovery of a single female.
a_3	$\gamma_1 Z_{10}$	Recovery of a male whose partner is susceptible
a_4	$\gamma_2 Z_{01}$	Recovery of a female whose partner is susceptible
a_5	$\gamma_1 Z_{11}$	Recovery of a male whose partner is infectious
a_6	$\gamma_2 Z_{11}$	Recovery of a female whose partner is infectious
a_7	$\alpha X_0 Y_0$	Relationship formation between a susceptible male and female.
a_8	$\alpha X_1 Y_0$	Relationship formation between an infectious male and a susceptible female.
a_9	$\alpha X_0 Y_1$	Relationship formation between a susceptible male and an infectious female.
a_{10}	$\alpha X_1 Y_1$	Relationship formation between an infectious male and female.
a_{11}	δZ_{00}	Relationship dissolution between a susceptible male and female.
a_{12}	δZ_{10}	Relationship dissolution between an infectious male and a susceptible female.
a_{13}	δZ_{01}	Relationship dissolution between a susceptible male and an infectious female.
a_{14}	δZ_{11}	Relationship dissolution between an infectious male and female.
a_{15}	$\beta_1 Z_{10}$	Infection from male to female
a_{16}	$\beta_2 Z_{01}$	Infection from female to male

a_i	Rate	Process
a_{17}	$\omega_1 X_1 Y_0$	A single infected male has a one-night stand with a single susceptible female
a_{18}	$\omega_1 r X_1 Z_{10}$	A sing infected male has a one-night stand with a non-single susceptible female with an infected partner.
a_{19}	$\omega_1 r X_1 Z_{00}$	A single infected male has a one night stand with a non-single susceptible female with a susceptible partner
a_{20}	$\omega_2 X_0 Y_1$	A single infected female has a non-night stand with a single susceptible male.
a_{21}	$\omega_2 r Y_1 Z_{01}$	A single infected female has a one-night stand with a non-single susceptible male with an infected partner
a_{22}	$\omega_2 r Y_1 Z_{00}$	A single infected female has a one-night stand with a non-single susceptible male with a susceptible partner
a_{23}	$\omega_1 r Z_{10} Y_0$	A non-single infected male has a one-night stand with a single susceptible female.
a_{24}	$\omega_1 r^2 Z_{10} Z_{10}$	A non-single infected male has a one-night stand with a non-single female with an infected partner.
a_{25}	$\omega_1 r^2 Z_{10} Z_{00}$	A non-single infected male has a one-night stand with a non-single female with a susceptible partner.
a_{26}	$\omega_1 r Z_{11} Y_0$	A non-single infected male with an infected partner has a one-night stand with a single susceptible female.
a_{27}	$\omega_1 r^2 Z_{11} Z_{10}$	A non-single infected male with an infected partner has a one-night stand with a single susceptible female.
a_{28}	$\omega_1 Z_{11} Z_{00}$	A non-single infected male with an infected partner has a one-night stand with a non-single with a susceptible partner.
a_{29}	$\omega_2 r Z_{01} X_0$	A non-single infected female has a one-night stand with a single susceptible male.
a_{30}	$\omega_2 r^2 Z_{01} Z_{01}$	A non-single infected female has a one-night stand with a non-single susceptible male with an infected partner.
a_{31}	$\omega_2 r^2 Z_{01} Z_{00}$	A non-single infected female has a one-night stand with a non-single susceptible male having an infected partner.
a_{32}	$\omega_2 r Z_{11} X_0$	A non-single infected female has a one-night stand with a single susceptible male.
a_{33}	$\omega_2 r^2 Z_{11} Z_{01}$	A non-single infected female with an infected partner has a one-night stand with a non-single male having an infected partner.
a_{34}	$\omega_2 r^2 Z_{11} Z_{00}$	A non-single infected female with an infected partner has a one-night stand with a non-single male having a susceptible partner.

The simulation is based on Gillespie algorithm, which can be summed up in the following pseudocode.

Algorithm 3.6.1: STOCHASTIC$(parameters, initial, t_{end})$

1 : **if** $(t < t_{end})$ *and* $(X_1(t) + Y_1(t) + Z_{10}(t) + Z_{01}(t) + Z_{11}(t)) \neq 0$ **then**

2 : **for** $i = 1..34$ **do**

$Calculate \;\; a_i \;\; and \;\; \lambda_i = \sum_{k=1}^{i} a_i$

end for

3 : $\tau \sim Exp(\lambda_{34})$

4 : *Generate a uniform random variable* r

$r* = r\lambda_{34}$

5 : *Find* i *such that*$\lambda_{i-1} < r^* \leq \lambda_i$

6 : *Set* $t = t + \tau$

7 : *Update the current state* S

3.7 Numerical Results and analysis

The objective of the numerical studies in this Section is to explore the model behaviour based on various set of parameters, as well as illustrating some of the theoretical results obtained in this Chapter. In particular evaluating the reproduction number, the probability of extinction and the endemic level for which no explicit formulae exist. For convenience, we label the model without one-night stands in Chapter 2 as Model 1, and the model with one-night stands in Chapter 3 as Model 2. In this subsection, similar to Model 1, we study the behaviour of the epidemic based on the model parameters.

Firstly, Model 1 is a special case of Model 2 when the rate at which an individual attempts to have a one-night stand is zero ($\omega = 0$). Different branching process approximations are used for the early stages of the two models. For Models 1 and 2, 2-type and 5-type branching process approximations are used, respectively. It is interesting to compare the results from the branching processes, the reproduction number and the probability of extinction, between both models for which $\omega = 0$. This will be done in subsection 3.7.1.

In subsection 3.7.2, we are interested in exploring the general behaviour of Model 2 focusing on the case that one-night stands play a part in the model ($\omega > 0$). We first look at the relationship between the endemic level, R_0 and the probability of extinction, then followed by the study of varying δ based on various sets of parameters, in which we know from Model 1 that moderate δ assists the epidemic. The study of $F = \frac{\bar{\alpha}}{\delta}$ is also included to see if the model converges to the two type homogeneously mixing model when $F \to \infty$. Lastly in this subsection, there is a comparison between the two extremal cases: the disease transmission occurs only within relationships ($\omega = 0$) and the disease is transmitted only outside relationships ($\beta = 0$) in order to investigate which kind of infection makes higher impact on the spread of the disease.

In subsection 3.7.3, we emphasise the effects of one-night stands on the model in the case that one-night stands can take place only between singles. As a result, the proportion of singles in equilibrium and the amount of time spent whilst being single are important factors. We explore a range of behaviours by looking at R_0, the probability of extinction and the endemic level based on different sizes of proportion of single individuals.

In terms of infections outside relationships, an individual can only be infected through a successful one-night stand. Someone attempting a one-night stand will not pass on the disease without another person agreeing to have one-night stand. In other words, an infectious one-night stand does not only depend upon the rate at which someone attempting one-night stands (ω), but also the probability of someone agreeing to have a one night stand (r). Therefore, in subsection 3.7.4, the constant rate of one-night stands for individuals is of our interest. R_0, the probability of extinction, as well as the endemic level are subsequently explored.

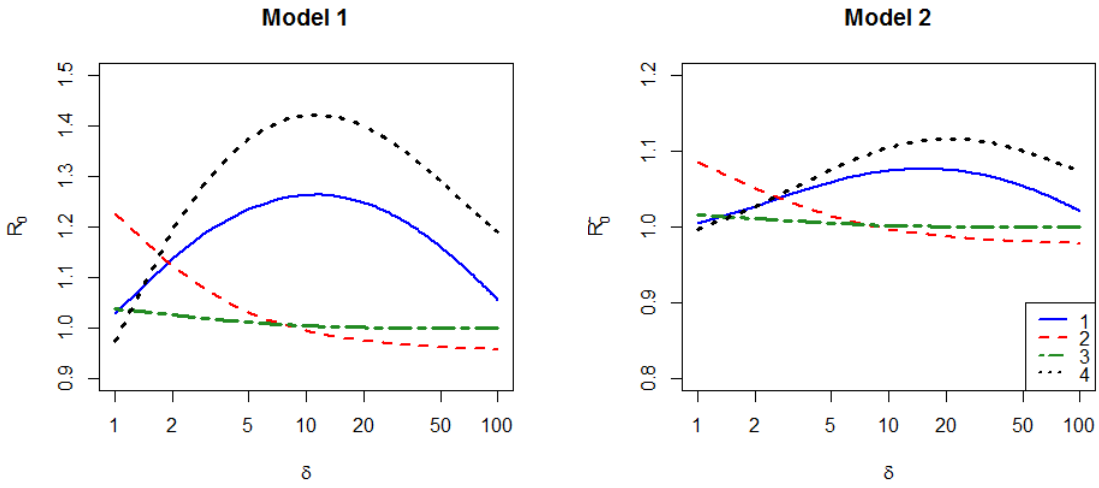
Lastly, in subsection 3.7.5, we study the mean time to extinction and the fluctuations about the endemic level based on a range of parameters for fixed endemic level. We look at the endemic level of 14% and 25% to be consistent with the study in Model 1 with the objective of investigating the effect of each model parameter on the mean time to extinction. In particular, in Model 1, γ_2 is the main influence on the mean time to extinction, we are interested in investigating similar behaviours.

3.7.1 Relationship between Model 1 and Model 2 ($\omega = 0$)

In this subsection, we explore the relationship between Model 1 and Model 2 by comparing the behaviour of R_0 between both models. For Model 2, we consider the case without one-night stands ($\omega = 0$). Again, we recall parameter sets from Table 2.5 here.

Table 3.3: Parameters chosen

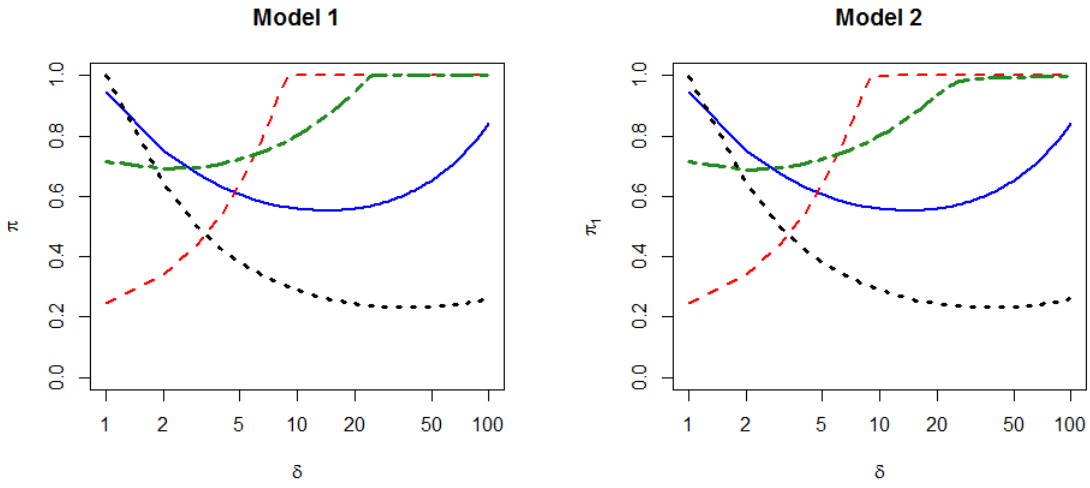
set	α	δ	γ_1	γ_2	β_1	β_2
1	5	1:100	1	1	100	100
2	0.8	1:100	0.02	0.02	0.5	0.5
3	100	1:100	0.1	0.1	0.2	0.2
4	200	1:100	3	3	40	40



(a) (b)

Figure 3.3: Plots of R_0 of Model 1 and Model 2 against δ using parameter sets in Table 3.3

Note from Figure 3.3(a) that each colour represents the same set of results as labelled in Figure 3.3(b). The 4 numbers in the label correspond to the sets of parameters illustrated in Table 3.3. Note also that, R_0 obtained from Model 1 in Figure 3.3(a) is R_0^1 and R_0 obtained from Model 2 is R_0^2. As we can see from Figure 3.3, R_0^1 and R_0^2 display similar behaviour as δ varies. Also, when $R_0^1 \leq 1$, $R_0^2 \leq 1$ and $R_0^1 > 1$, $R_0^2 > 1$ even though R_0^1 and R_0^2 have different values.



(a) (b)

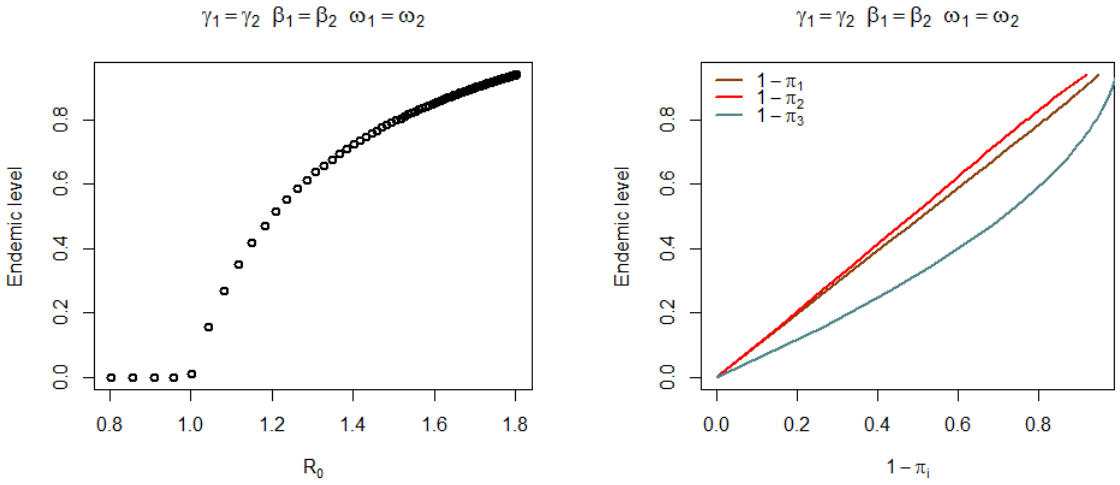
Figure 3.4: Plots of the probability of extinction (π_1) of Model 1 and Model 2 corresponding the parameter sets in Table 3.3

In terms of probabilities of extinction related to R_0^1 and R_0^2 in Figure 3.3, Figure 3.4 shows that that the probability of extinction obtained from Model 1 and Model 2 are the same.

3.7.2 General behaviour of the model with one-night stands (Model 2 : $\omega_1, \omega_2 \neq 0$)

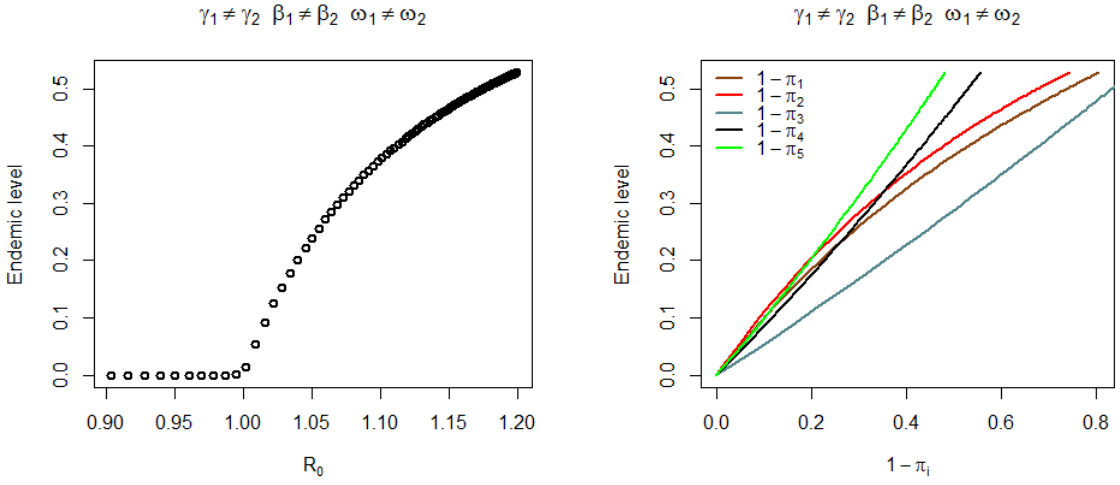
In Section 2.7, we studied the effects of each parameter on the threshold parameter R_0, such that R_0 increases when the infection rate increases, whereas R_0 decreases if there is an increasing of the recovery rates. For increasing δ, R_0 can either increase or decrease both monotonically and non-monotonically. In this section, it is interesting to see what happens in the model with one-night stands as the infection is allowed to be transmitted outside relationships in this case. We will restrict our attention to the rate at which one-night stand occurs, i.e. $\omega_1, \omega_2 \neq 0$. Generally, a one-night stand should increase the chance of infection between individuals of the opposite sex, resulting in an increasing in number of infected individuals, leading to an increasing of R_0. We investigate the relationship between

R_0, the endemic level and the probability of extinction of two cases : no difference in parameters between the two sexes (Figure 3.5) and there is the difference (Figure 3.6). The parameters are chosen as labelled in the Figures.



(a) (b)

Figure 3.5: (a) Plot of endemic level against R_0 (b) Endemic level against probability of the disease not going extinct, for varying ω_1, ω_2 : 1, 2,...,100. Other parameters are fixed : $\gamma = 2, \beta = 1, \alpha = 9, \delta = 6, \bar{\alpha} = 3, p = 0.1667, q = 0.3333, r = 0.5$.



(a) (b)

Figure 3.6: (a) Plot of endemic level against R_0 (b) Endemic level against probability of the disease not going extinct, for varying ω_1: 1, 2,...,100. Other parameters are fixed : $\omega_2 = 1, \gamma_1 = 5, \gamma_2 = 1, \beta_1 = 0.1, \beta_2 = 5, \delta = 15, \alpha = 10, \bar{\alpha} = 3.9564, p = 0.1044, q = 0.3956, r = 0.5$.

We can see in Figure 3.5 and 3.6 that R_0 and the endemic level are increasing while ω increases. The endemic level increases as R_0 increases in a monotonic manner. Note that in Figure 3.5(b) only $1-\pi_1$, $1-\pi_2$ and $1-\pi_3$ are plotted as when $\beta_1 = \beta_2, \gamma_1 = \gamma_2, \omega_1 = \omega_2$, we have that $\pi_1 = \pi_5$ and $\pi_2 = \pi_4$. We can see that the endemic level has a non-linear relationship with $1-\pi_1$, $1-\pi_2$ and $1-\pi_3$, even though it looks very close to linear with $1-\pi_1$ and $1-\pi_2$. For the case where $\beta_1 \neq \beta_2, \gamma_1 \neq \gamma_2$ and $\omega_1 \neq \omega_2$, Figure 3.6 shows similar relationship between R_0 and endemic level. The endemic level also has non-linear relationship with $1-\pi_i, i = 1, 2, 3, 4, 5$.

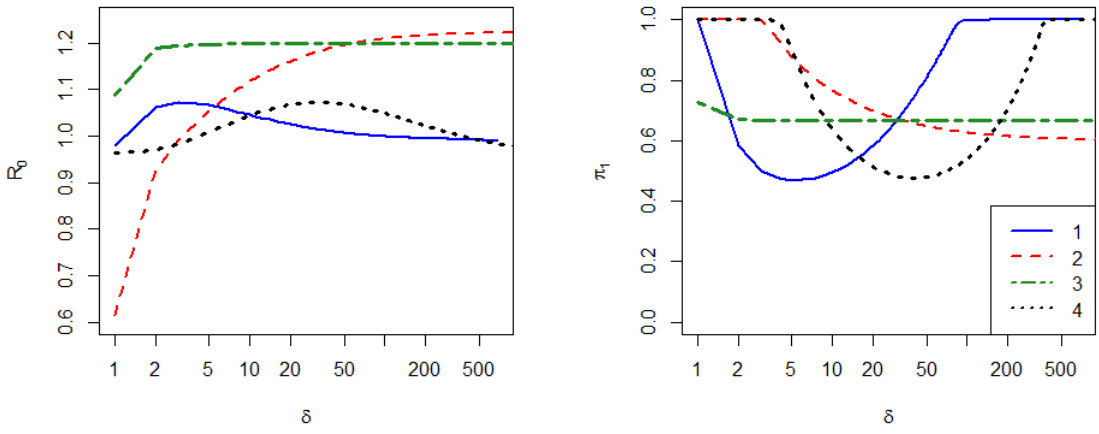
The above relationships with varying ω are as we would expect. Given the observations in Chapter 2 and Section 3.7.1 it is interesting to look at how the quantities of interest vary as δ varies. The key point is that, in Model 1, the disease is allowed to be transmitted only within relationships whereas in this model the transmission can occur outside relationships. Therefore, it is interesting to investigate R_0, the endemic level, and the probability of extinction with regards to increasing δ into the model incorporating one-night stands. Parameter sets are chosen as illustrated in Table 3.4.

Table 3.4: parameters chosen

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$	$\boldsymbol{\omega_1}$	$\boldsymbol{\omega_2}$	$\boldsymbol{r}$
1	5	0.001:2000	0.1	0.1	0.5	0.5	0.1	0.1	0.5
2	10	0.001:2000	30	30	1	1	100	100	0.5
3	0.1	0.001:2000	10	10	50	50	30	30	0.5
4	100	0.001:2000	5	5	50	50	0.8	0.8	0.5

Figure 3.7 shows the behaviours of R_0 as δ increases corresponding to each parameter set in Table 3.4. Note that there is no difference between parameters of both sexes, i.e. $\beta_1 = \beta_2, \gamma_1 = \gamma_2$ and $\omega_1 = \omega_2$. Recall our model assumption that we start with 1 infective individual in a susceptible population, assuming that it is of type 1. Therefore, throughout our numerical studies in this Chapter, we are interested in the probability of extinction starting from a type 1 individual. As a

result, the probability of extinction in Figure 3.7 is π_1.



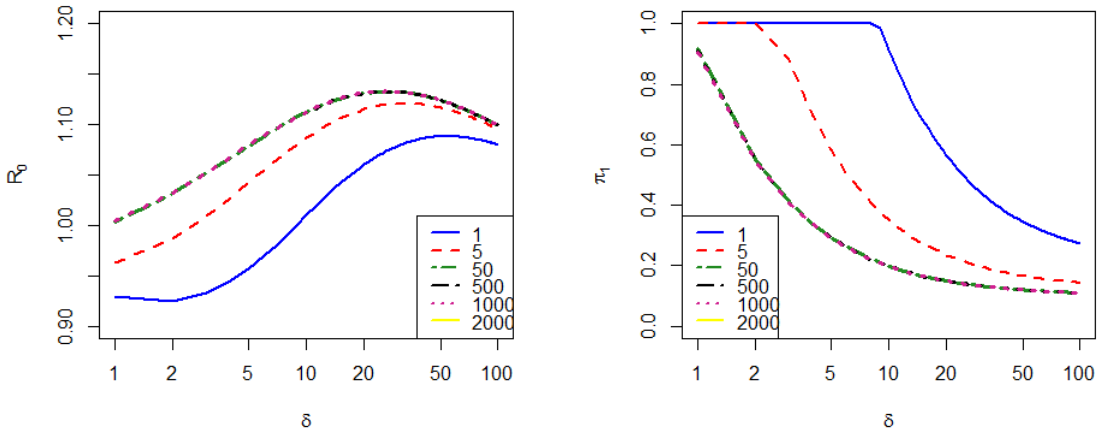
(a) (b)

Figure 3.7: Plots of R_0 and probability of extinction against δ using parameter sets in Table 3.4

According to Figure 3.7, we can see that R_0 exhibits a range of behaviour. For parameter set 1 and 4, R_0 is largest when the level of δ relative to α is moderate. Note that β is considerably higher than ω meaning that infections occur mainly within relationships. Therefore, when the epidemic is driven by β, the spread of the epidemic is maximum when the time spent in a relationship is moderate. This coincides with our results for Model 1 (see Figure 2.4(d)). At the same time, when the disease transmission outside relationships is substantial, R_0 will increase as δ increases (parameter set 2 and 4). This is because the chance that transmission outside relationships will occur is higher when individuals are single. Therefore, increasing the level of individuals being single will also increase the spread of the disease.

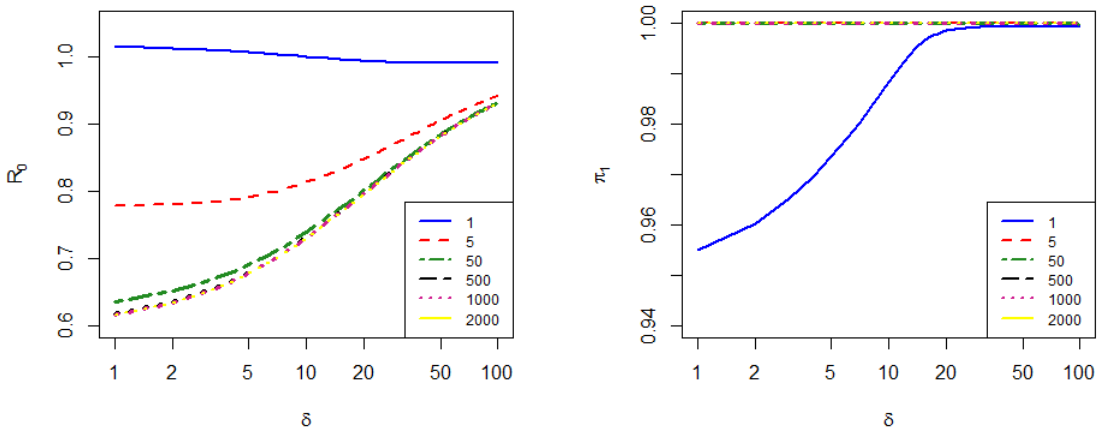
Fixing $F = \frac{\bar{\alpha}}{\delta}$ and letting δ increase, is also interesting for Model 2, especially when $F \to \infty$. Intuitively, we know that $\alpha = \bar{\alpha}(\frac{\sigma}{2})$ and $\sigma = \frac{\delta}{\bar{\alpha}+\delta} = \frac{1}{F+1}$. Therefore, as $F \to \infty$, we have that $\sigma \to 0$ and $\alpha \to \infty$. The key point is as δ and F increase then an individual spends most of their time in a relationship but moves

very quickly from one relationship to another. Therefore every within relationship infection is almost certainly with somebody different. As such, the model increasingly behaves like a two type homogeneously mixing model. This agrees with our numerical study which is illustrated in Figure 3.8. Parameter sets are chosen from parameter set 4 in Table 3.4, which are $\gamma = 5, \beta = 50, \omega = 0.8, r = 0.5$. F is fixed at $F = 1, 5, 50, 500, 1000, 2000$ and δ increases by 1 from 1 to 100. Note that $\bar{\alpha} = dF$.



(a) (b)

Figure 3.8: Plots of R_0 and π_1 against δ for Fixed F, where $\gamma = 5, \beta = 50, \omega = 0.8, r = 0.5, \delta = 1, 2, ..., 100$ and $\bar{\alpha} = dF$



(a) (b)

Figure 3.9: Plots of R_0 and π_1 against δ for Fixed F, where $\gamma = 30$, $\beta = 1$, $\omega = 100$, $\delta = 1, 2, ..., 100$ and $\bar{\alpha} = dF$.

Figure 3.9 shows that as F increases, the level of R_0 is decreasing in contrast with the behaviour of R_0 for the case that β is driving in which it is increasing, as seen in Figure 3.8. However, as F tends to infinity, the model still increasingly mimics the two type homogeneously mixing model.

Now, we have noticed from our previous studies in Figure 3.7 that with high ω, there is a rapid increase in R_0. It is interesting to compare and contrast two extremal cases. Case 1 is $\omega = 0$, no one-night stands so infection only takes place in relationships. Case 2 is $\beta = 0$ where the disease can only be transmitted outside relationships. Parameters chosen for both cases are $\gamma_1 = 3, \gamma_2 = 3, \delta = 11, \bar{\alpha} = 15, r = 0.5$. For the first case (red line), we fix $\omega = 0$ and increase β from 1, 2, ...,2000. On the other hand, we fix $\beta = 0$ for the second case (black line) and increase ω from 1 to 2000. Note that $\beta_1 = \beta_2 = \beta$ and $\omega_1 = \omega_2 = \omega$. In Figure 3.10, R_0 corresponding to the two cases mentioned above are plotted against ω for the first case, and β for the second case.

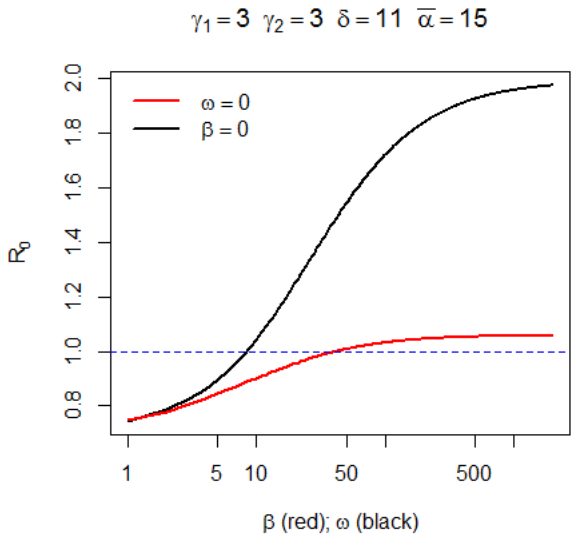


Figure 3.10: R_0 corresponding to the case where $\beta = 0$ and $\omega = 0$.

As we can see from Figure 3.10 the starting points of R_0 from both cases are nearly the same before increasing in a different speed. Therefore, it clearly to see that ω makes a greater impact on R_0 as compared to β. Note that, in this case, if a single person attempts to have a one night stand, it is 50% chances the one-night stand with a non-single person is successful ($r = 0.5$). In other words, 50% of individuals in relationships who have attempted one-night stands with a single person will eventually agree to have a one-night stand. Therefore, in general, it makes sense that a higher chance of individuals having one-night stands will increase the incidence of the disease.

As such, for the model with one-night stands, we could see that not only the rate at which individuals attempt to have one-night stand plays an important role in the model, but also the probability of such a one-night stand being successful. Note that by the model assumptions, a one-night stand between singles is 100% successful. In an extreme case if $r = 1$, this means whoever attempts to have a one-night stand, it is 100% successful. If $r = 0$, it means nobody in relationships agrees to have a one-night stand (one-night stands only occur between singles).

The behaviour of R_0 for which $r = 0$, one-night stands occur only among single individuals, is interesting. This is because, when one-night stands only occur between singles, the proportion of single individuals in the population at that time matters. If the population only consists of couples (when $\sigma \approx 0$), even though the rate of attempting one-night stands is high, there is no chance that the disease is transmitted as nobody is single. Namely, a high value of ω will have no effect on R_0. For that reason, in Subsection 3.7.3, we investigate the behaviour of R_0, the probability of extinction, as well as the endemic level for which $r = 0$.

3.7.3 One-night stands occur only between singles ($r = 0$)

In this subsection, the objective is to emphasise the epidemic behaviours driven by infections through one-night stands given that only singles can have one-night stands. We explore behaviours corresponding to varied sizes of the proportion of singles (σ) in the population and the time one spends being single between relationships. It is interesting to study the following 3 different scenarios: large proportion of singles (close to 1), moderate proportion of singles, and extremely small proportion of singles (close to 0). Since σ is a function of $\bar{\alpha}$ and δ, the experiments will be carried out through a ratio of $\bar{\alpha}$ to δ, i.e. $F = \frac{\bar{\alpha}}{\delta}$. Three different situations are thus considered : extremely small F ($F \approx 0$), moderate F ($F = 1$ and $F = 10$), and extremely large F ($F \approx \infty$). Note that each F results in a different value of σ, such that $F = 0.0001 \approx 0$ gives $\sigma = 0.9999 \approx 1$, $F = 1$ and $F = 10$ give $\sigma = 0.5$ and 0.09 respectively, and $F = 10000 \approx \infty$ gives $\sigma = 0.00009 \approx 0$. Also, how an individual spends time inside or outside relationships are represented through high values or low values of $\bar{\alpha}$ and δ. Therefore, in each case, we study two different sets as labelled on the graphs. The fixed parameters are $\gamma_1 = 8, \gamma_2 = 8, \beta_1 = 5, \beta_2 = 5$. In each case, ω_1 and ω_2 increase from 1,2, ..., 100. Figure 3.11 shows the behaviour of R_0 corresponding to each case.

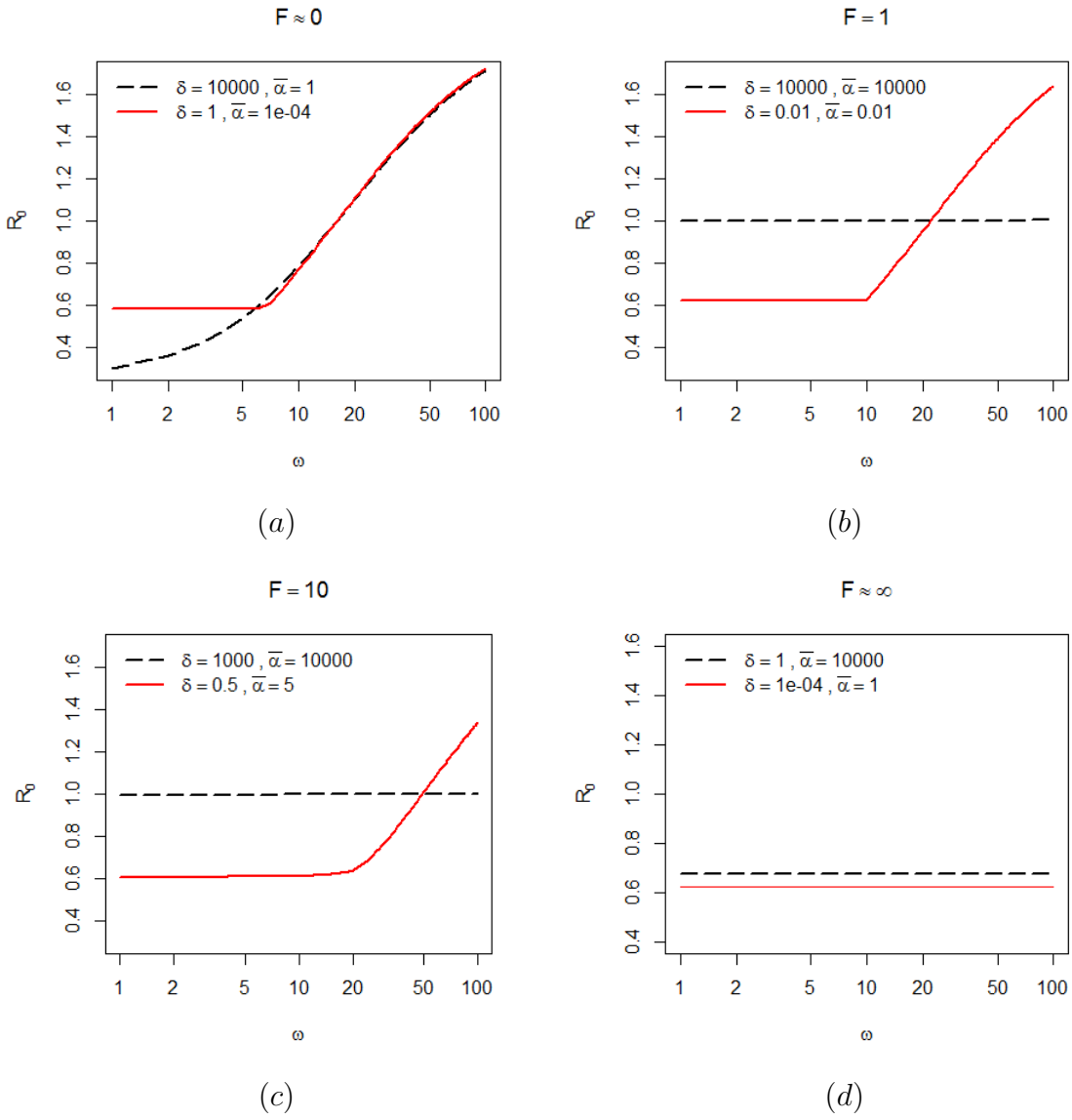
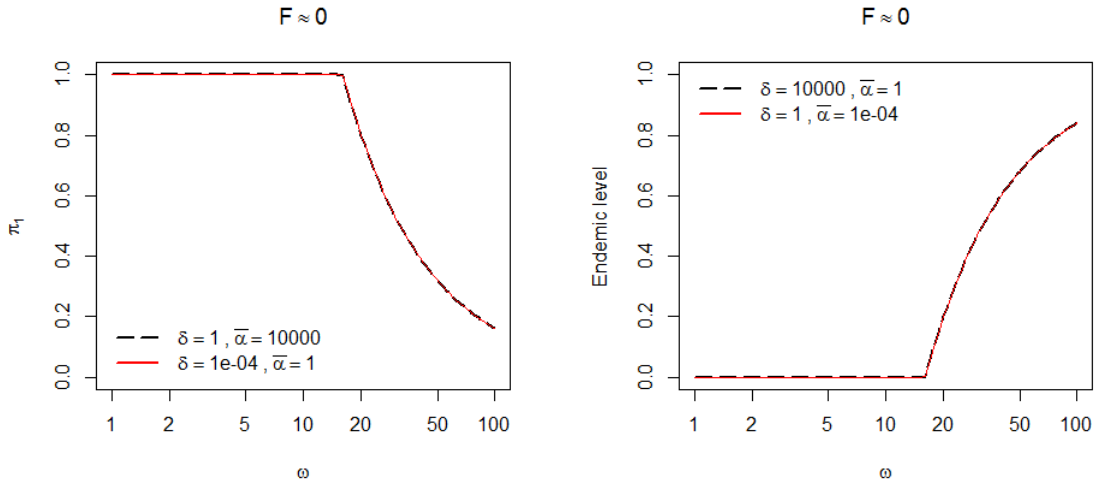


Figure 3.11: (a) Plot of R_0 corresponding to $F \approx 0, \sigma \approx 1$
(b) Plot of R_0 corresponding to $F = 1, \sigma = 0.5$
(c) Plot of R_0 corresponding to $F = 10, \sigma = 0.09$
(d) Plot of R_0 corresponding to $F \approx \infty, \sigma \approx 0$

From Figure 3.11, in the last case ($F \approx \infty$), it is seen that ω has almost no effect whilst it increases in which such behaviour has been described before we start this subsection. Considering the other cases ($F \approx 0, F = 1, F = 10$), when there is a slow rate in changing partners (the red line), R_0 starts to dramatically increase when ω reaches a large enough value. As a consequence, we could say that when ω is substantial, a high breakup rate drives the epidemic since the disease is

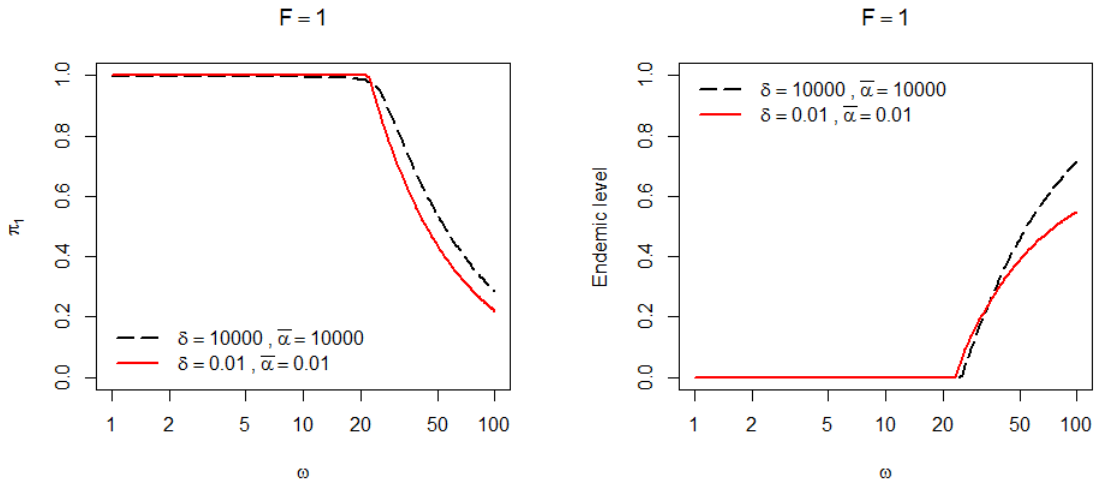
being transmitted mostly among singles. In the case when an individual spends very short time being single leading to a very small or no chance a single person is infected by a one-night stand, an increasing ω has almost no effect on R_0 even though the proportion of singles are moderate ($\sigma = 0.5$ and 0.09), as seen as the black dash line in the cases $F = 1$ and $F = 10$. In contrast, the black dash line in $F \approx 0$ shows a dramatic increases in ω. This case represents the scenario as such an individual spends a lot of time being single increasing the chance of the disease being transmitted.

Next, we are interesting in their corresponding probabilities of extinction and the endemic level. The case that $F \approx \infty$ will not be considered as we know that the probability of extinction will always be 1 and the corresponding endemic level will be 0. The results are plotted in Figures 3.12, 3.13 and 3.14.



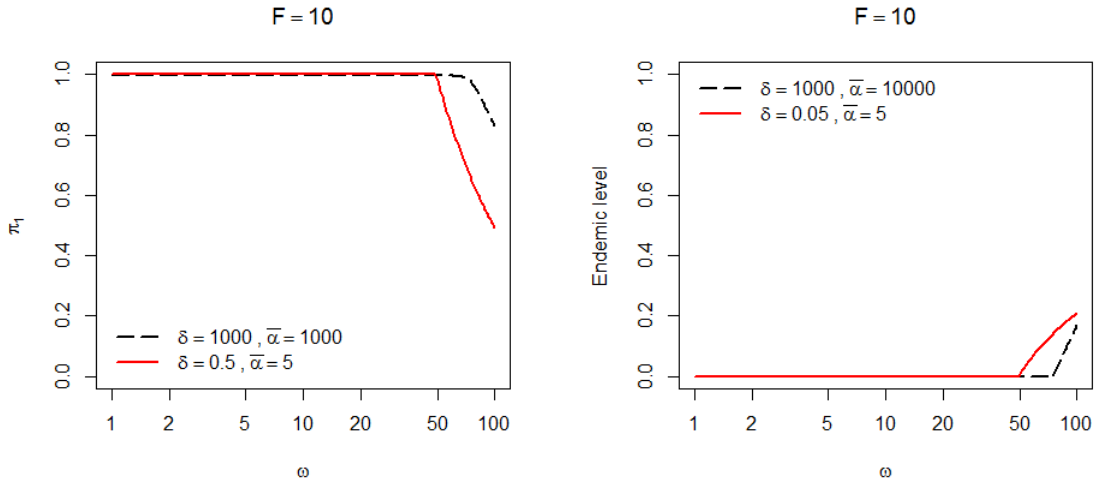
(a) (b)

Figure 3.12: Plots of the probability of extinction and the endemic level against r corresponding to $F \approx 0$



(a) (b)

Figure 3.13: Plots of probability of extinction and the endemic level against r corresponding to $F = 1$



(a) (b)

Figure 3.14: Plots of probability of extinction and the endemic level against r corresponding to $F = 10$

In Figure 3.12, the probability of extinction and the endemic level corresponding to $F \approx 0$ are nearly the same for the two sets of $\bar{\alpha}$ and δ. Note that in the case that R_0 looks steady around 1, it might have some effect and even cross 1 at some point, in which the effect is too little to be seen in the graph. The probability

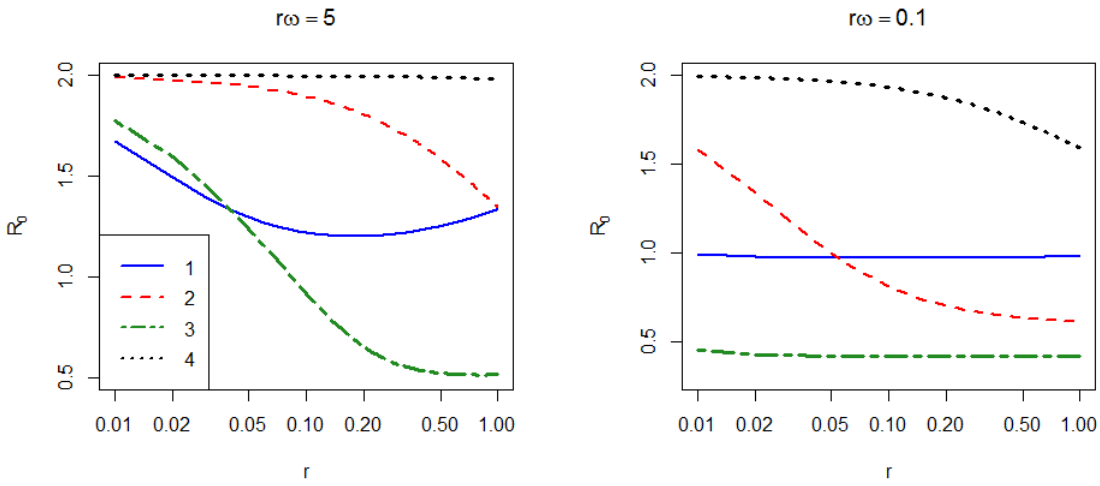
of extinction and the endemic level indicate clearer behaviours as we can see in Figures 3.13 and 3.14 that the black dash line deviates from 0 ($R_0 > 1$) after a certain value of ω.

3.7.4 Constant rate of successful one-night stands ($r\omega$)

We know from the previous studies that R_0 increases in ω. As ω increases whereas the probability that an individual in a relationship agrees to have a one-night stand (r) is fixed, we will have an increasing rate of successful one-night stands. Therefore, it makes sense that the incidence of the disease will also increase. We also know that as r increases, there is a higher chance that non-singles will agree to have one-night stands, R_0 should also increase. What if we have the rate at which one-night stands being successful ($r\omega$) fixed? We know that in order to fix $r\omega$, ω will be decreasing as r increases and vice versa. Therefore, in this subsection, we are looking at the case for which r increases from 0 to 1 by 0.01 and ω consequently decreases. Different sets of parameters are chosen as illustrated in Table 3.5. We deliberately chose the underlying parameter sets to cover a range of R_0 behaviours. $r\omega$ is fixed for the following two cases : $r\omega = 5$ and $r\omega = 0.1$. Note that there is no distinction between parameters of the two sexes in this case.

Table 3.5: Parameters chosen

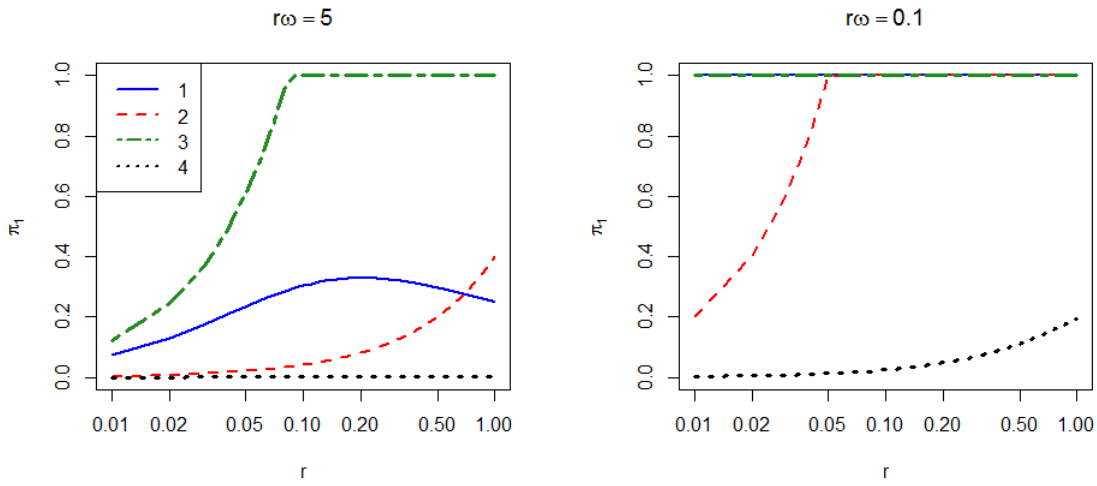
set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma}$	$\boldsymbol{\beta}$
1	100	1	1	3
2	1	100	1	2
3	6	5	20	5
4	0.01	0.02	0.01	0.02



(a) (b)

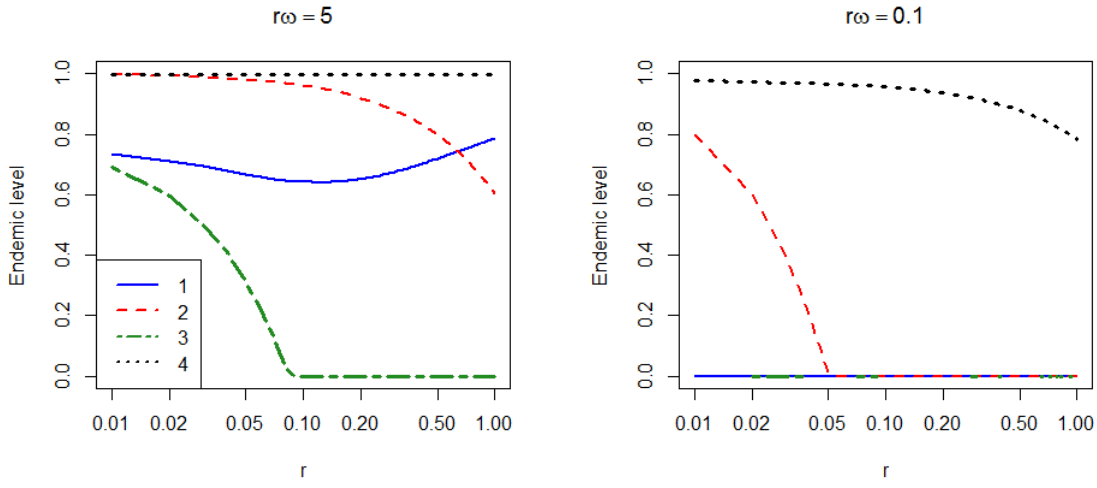
Figure 3.15: Plot of R_0 against r corresponding to parameter sets in Table 3.5

We can see from Figure 3.15, R_0 is not always monotonic in which we can clearly see in Figure 3.15(a) parameter set 1. In Figure 3.15(b), parameter set 1, R_0 also displays a non-monotonic behaviour even though it is not as clear. Corresponding to the same parameter sets in Table 3.5, we are interested in how the endemic level and the probability of extinction would look like. Figure 3.16 and 3.17 show the plots of the underlying endemic level and the probability of extinction.



(a) (b)

Figure 3.16: Plot of the probability of extinction (π_1) against r corresponding to parameter sets in Table 3.5



(a) (b)

Figure 3.17: Plot of the endemic level against r corresponding to parameter sets in Table 3.5

The results shown in Figure 3.16 and 3.17 are as expected. It is worth drawing attention to the parameter set 1 where an individual spends a long time in a relationship due to a high value of α in which it shows the minimum incidence of the disease at moderate ω and r in contrast with the other parameter sets in the same table that R_0 is minimum at highest r, in other words, smallest ω.

3.7.5 Fluctuations about the endemic level and the mean time to extinction

In subsection 2.7.3, we have studied the fluctuations about the endemic level of Model 1 and the effect of each parameter on the mean time to extinction given that the endemic level is fixed. The results of Model 1 show that the standard deviation of the total number of infectives is not informative for the mean time to extinction. In terms of the effect of the model parameters, we found that β does not have much effect. It is interesting to see if such behaviour also holds for ω as both are parameters subject to infection. Moreover, in Model 1 with γ_1 fixed equal to 1, we found that the mean time to extinction decreases with γ_2 and that the other parameters had little effect for a fixed endemic level. It is worthwhile to investigate whether or not such similar behaviour holds for Model 2. In this subsection, we will carry out similar studies focusing on ω. First, in order to see whether or not ω has a significant effect on the mean time to extinction, we fix the other parameters and vary ω_1 and ω_2. For convenience, throughout this subsection, we define T the time to extinction and thus $E[T]$ denotes the mean time to extinction.

Table 3.6: Mean time to extinction and the standard deviation (SD) of the total number of infectives about the endemic level 0.14 and population size 100, varying ω_1 and ω_2.

α	δ	γ_1	γ_2	β_1	β_2	ω_1	ω_2	SD	$E[T]$
9	6	1	2	3	4	5.8066	1	9.1887	24.5115
9	6	1	2	3	4	0.3501	10	9.4199	21.6592
9	6	1	2	3	4	9.9643	0.1	9.8439	21.2822
9	6	1	2	3	4	10.7483	0.0010	9.9775	21.0696
9	6	1	2	3	4	2.6645	3	8.8380	25.8982
9	6	1	2	0.1	0.5	52.2169	0.1	14.4172	8.1118
9	6	1	2	0.1	0.5	0.1239	100	17.6199	5.5126
9	6	1	2	0.1	0.5	0.5139	30	11.3493	11.9459
9	6	1	2	0.1	0.5	5.1136	3	8.7451	24.9835
9	6	1	2	0.1	0.5	13.5998	1	9.8771	17.2025
9	6	1	2	0.1	0.5	0.0010	363.0310	31.3905	4.0084
9	6	1	2	0.1	0.5	4	3.8898	8.6498	25.1970

In Table 3.6, two cases are carried out ; $\beta_1 = 3, \beta_2 = 4$, and $\beta_1 = 0.1, \beta_2 = 0.5$. The second case was chosen so that the epidemic is driven by one-night stands. In the first case when the epidemic is driven by both infections within and outside relationships, we can see that varying ω_1 and ω_2 does not have much effect on the mean time to extinction in which it agrees with the results of varying β_1 and β_2 in Model 1. In the second case when the one-night stand infection is the main spreader, we can see variation in the mean time to extinction varying from 4 - 25. Moreover, in Table 3.6, we observe that there is a clear decreasing trend of the mean time to extinction in the standard deviation of the total number of infectives, see Figure 3.18.

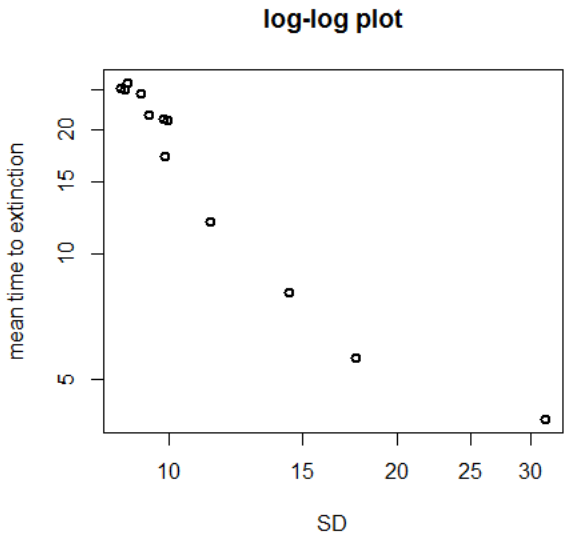


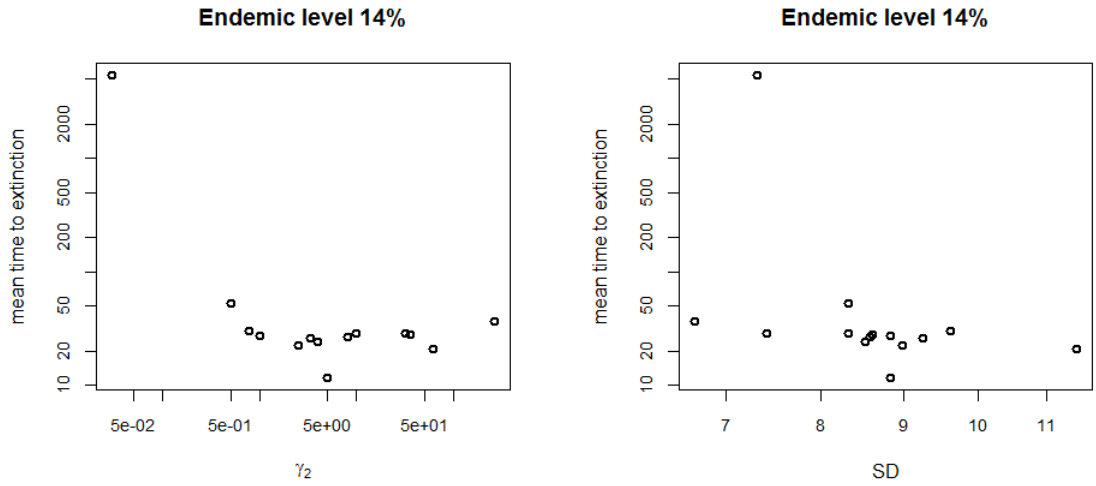
Figure 3.18: Plot of the mean time to extinction against standard deviation of the results in Table 3.6

Next, we want to explore the relationship between the mean time to extinction and γ_2 where ω_1 and ω_2 are included in the model. Similar to the study for Model 1 in subsection 2.7.3, we consider the endemic level of 14% and fix $\gamma_1 = 1$ whilst allowing other parameters to vary.

Table 3.7: Mean time to extinction and the standard deviation (SD) of the total number of infectives about the endemic level 0.14 and population size 100, fixing $\gamma_1 = 1$

set	α	δ	γ_2	β_1	β_2	ω_1	ω_2	$E[T]$	SD
1	6	9	8.0261	30	40	3	4	26.7766	8.5825
2	40	30	62.6539	8	9	100	1	21.1299	11.4899
3	1	10	0.7788	0.1	8	5	0.5	30.2420	9.6062
4	100	2	35.4244	30	80	5	6	28.2738	8.6099
5	1.5	100	269.4014	3	8	50	30	36.4013	6.6987
6	15	9	1	1.9810	3	2	1	27.4466	8.8231
7	5	25	2.5	1.4942	1	2	7	22.4006	8.9796
8	20	10	3.3	12.3853	5	1.5	1.5	26.2574	9.2365
9	10	11	0.5	1.2454	1.5	1.5	1.25	52.8581	8.3249
10	10	1	10	7.9699	30	5	6	28.8461	8.3202
11	3	1	0.0297	0.1	0.8	0.5	0.2	5326.4	7.3143
12	105	8	5	0.0033	0.1	2	70	11.8250	8.8367
13	5	95	31.7168	15	11	11.2	15	28.5174	7.4212
14	9	6	3.9465	3	2	5.55	4.48	24.5216	8.5202

As we can see in Table 3.7, a high variation in γ_2, ranging from 0.0297 -269.4014, only has a small influence on the mean time to extinction with the exception of parameter set 11. In parameter set 11, small γ_2 gives rise to a very large mean time to extinction. This is probably because other parameters in this set are also small so that the epidemic moves with a very slow rate to the disease-free stages. In Figure 3.19, we plot the mean time to extinction against γ_2 and the standard deviation in order to see clearer relationships.



(a) (b)

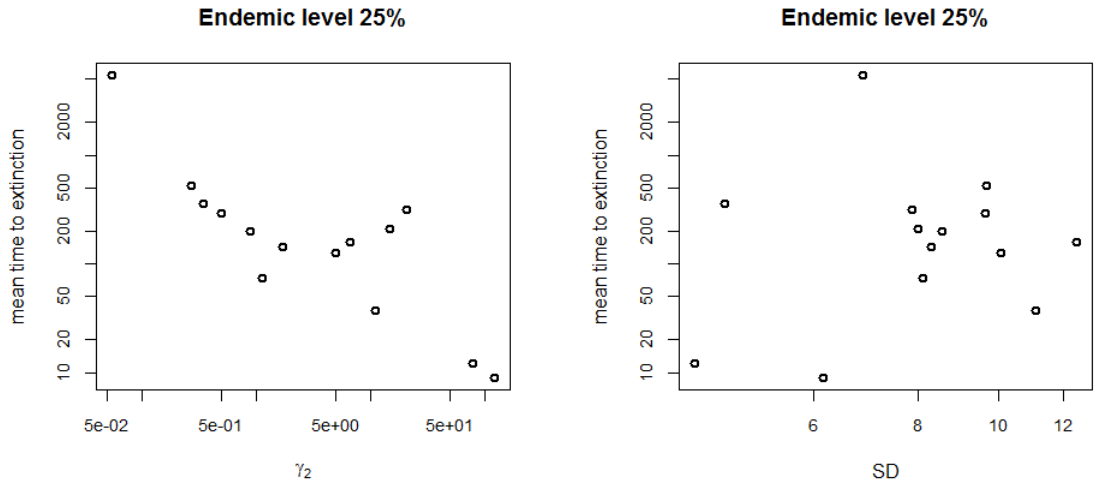
Figure 3.19: Plot of the mean time to extinction against γ_2 and against standard deviation (SD) of the results in Table 3.7

According to Figure 3.19(a), apart from a very high mean time to extinction for $\gamma_2 = 0.02968$, we can see small fluctuations in the mean time to extinction with no clear pattern. Also, in Figure 3.19(b), there is no obvious relationship between the mean time to extinction and the standard deviation.

Now, we will explore a higher range of the parameters by considering the higher endemic level which is 25%. In this case, we reduce the range of δ and keep it fixed to be 0.1, 1, 10 and 15 in order to see the behaviour of the epidemic as such the breakup rate is not too high. α is varied freely and can be as high as 100. Other parameters are varied accordingly whilst keeping $\gamma_1 = 1$ fixed. Note that, the parameters are chosen to cover a range of epidemic behaviours, such as the epidemic is driven either by infections within relationships or outside relationships or by both.

Table 3.8: Mean time to extinction and the standard deviation (SD) of the total number of infectives about the endemic level 0.25 and population size 100, fixing $\gamma_1 = 1$

set	γ_2	β_1	β_2	α	δ	ω_1	ω_2	E[T]	SD
1	0.5	3	10	30	0.1	0.1412	2	294.1279	9.6579
2	124	762.6021	100	9	0.1	2	3	9.0710	6.1588
3	6.7121	0.1	0.2	1	0.1	10	20	159.3167	12.4487
4	11	1	0.1	3.1917	1	100	2	37.6065	11.0856
5	0.2729	4	5	9	1	0.01	0.02	520.1180	9.6904
6	1.6998	0.1	0.2	1	1	5	4	142.9227	8.2957
7	5	8.5388	2	5	1	8	6	124.6031	10.0900
8	15	32.5414	7	0.3	1	16	6.5	210.4932	7.9944
9	0.8912	5.8075	5	1.27	10	2.5	2	197.8008	8.5498
10	0.35	0.4274	1.98	100	10	1	1	357.9315	4.6859
11	0.0549	0.5	5	0.1	10	1.5	0.25	5396.7	6.8668
12	20.8775	11	9	9	10	15	10	311.4344	7.8752
13	1.1354	9	11	9	10	0.25	0.1	74.8351	8.1172
14	80	5.3865	50	10	15	1	100	12.1832	4.3081



(a) (b)

Figure 3.20: Plot of the mean time to extinction against γ_2 and against standard deviation (SD) of the results in Table 3.7

In Figure 3.20(a), we can see a clearer decreasing behaviour of the mean time to extinction in γ_2. Whereas, the mean time to extinction and the standard deviation still do not display any clear relationship. We know that each model parameter has some impact on the mean time to extinction, especially, γ_2 is a dominant influence

on the mean time to extinction for Model 1. However, in Model 2, γ_2 still plays an important role in influencing the mean to extinction but it seems that it is not as dominant as it is in Model 1, depending on how the model parameters are set. In particular, γ_2 is a within relationship recovery and thus an infective despite how she becomes infectious (either by infections inside or outside relationships) will be recovering only after an episode of a relationship. Therefore, if there is a sizeable infections from outside relationships (ω) taking part in the model, we can intuitively see that γ_2 will have less impact.

Chapter 4

Control strategies and case studies

4.1 Introduction

We have studied mathematical models describing the dynamical behaviour of STDs and their impact in Chapters 2 and 3. However, another question arises as to what can we do if there is a major outbreak of the disease? How can we control the diseases? To answer these questions, in this Chapter, we focus our studies on control strategies.

For sexually transmitted diseases, various control strategies such as vaccination, condom use, and antiviral drugs, have been used. To study their impacts, we apply these strategies into our models to intervene in the spread of the disease. Regarding vaccination and condom use, it will reduce the chances that a susceptible becomes infected. In other words, it reduces the infection rate. For example, if the rate at which males use condoms is v, then the infection rate becomes $(1-v)\beta_1$. Thus, to investigate these strategies, we only need to adjust the model parameters without changing the structure. Therefore, in this Chapter, we focus primarily on incorporating medication use as a control measure leading to a change in the

model structure.

In Section 4.2 we incorporate a control strategy in Model 1, and study its impact on the reproduction number, the probability of extinction, and the endemic level. Similarly, the control strategy is applied to Model 2 with the results discussed in Section 4.3. Numerical results and discussion will be presented in Section 4.4. Moreover, the applications of Model 1 and Model 2 to a specific disease, gonorrhoea are also illustrated in Section 4.5.

4.2 Model 1 with a control measure

Recall that Model 1 is a sexual model without one-night stands (Chapter 2). We studied its early stages using a two-type branching process to determine the reproduction number and probability of extinction. In this Section, we use a similar approach such that the two-type branching process can still be considered. We introduce additional assumptions to those made in Chapter 2 (Section 2.2). The additional assumptions are:

1. Within a relationship where both the male and the female are infected, either or both of them could go to a doctor and be diagnosed with an STD. The male is diagnosed and recovers with rate γ_1, whilst the female is diagnosed and recovers with rate γ_2.

2. An infected individual is given two treatment prescriptions, one for themselves and one to pass onto their partner. When an infected individual is given extra drugs, he/she can either pass it on to his/her partner or not. If they pass the drugs on to their partner, their partner may take the drugs and also recover. Hence, the individuals in a relationship can recover simultaneously.

3. Let v_1 be the probability that a male on diagnosis passes the drugs onto his

partner and the female takes them and recovers. Otherwise with probability $1 - v_1$ the female does not recover. Let v_2 be the corresponding probability for a diagnosed female.

From the above assumptions, we can construct a sexual network model focusing on the relationship stage using a two-type branching process as in Chapter 2. The explanation of the model incorporating the control measure and the derivation of the reproduction number, R_0, as well as the probability of extinction are discussed in section 4.2.1.

4.2.1 The reproduction number (R_0) and the probability of extinction

According to the assumptions stated above, we can see that the original model in Chapter 2 is unchanged apart from the relationship stages. Figure 4.1 illustrates scenarios occurring among those relationships within which both are infected, only one person is infected, and nobody is infected. All parameters and variables are as defined in Chapter 2.

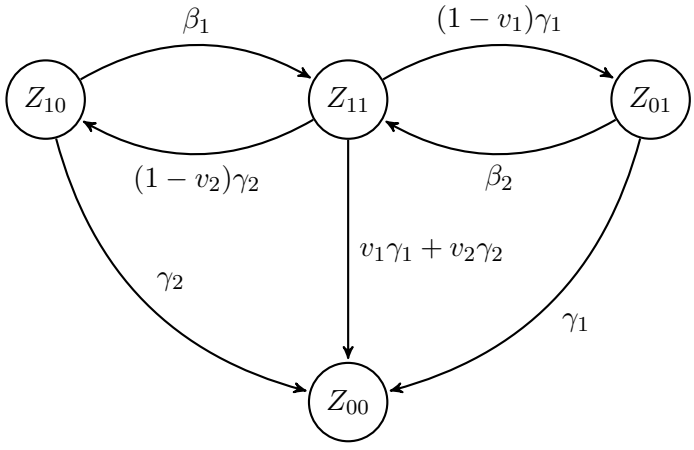


Figure 4.1: Sexual network diagram within a relationship stage when there is a control measure.

Note that Figure 4.1 shows only the process within the relationship stages,

whilst the rest of the process has not changed. Starting from the stage at which both are infected, the following possible events could happen.

- A male is diagnosed and recovers with rate γ_1, he is given extra drugs and passes them on to his partner. His partner takes the drugs and recovers with probability v_1. The infected relationship will become an uninfected relationship with rate $v_1\gamma_1$. At the same time, if a female is diagnosed, she will pass on the extra drugs to her partner, the relationship will become uninfected with rate $v_2\gamma_2$. Therefore, in this case, the rate of moving from state Z_{11} to Z_{00} is $v_1\gamma_1 + v_2\gamma_2$.

- A male is diagnosed but the extra drugs have no effect on his partner (either he does not pass them or she does not take them). Therefore, only the male recovers in this case with rate $(1 - v_1)\gamma_1$ of moving from state Z_{11} to Z_{01}.

- A female is diagnosed but the extra drugs have no effect on her partner. Only the female recovers in this case and with rate $(1 - v_2)\gamma_2$ of moving from state Z_{11} to Z_{10}.

When there is only one infected person, they will be diagnosed and recover with rate γ_1 if it is male, and γ_2 if it is female. Since the extra drugs given to such an individual will have no effect on their susceptible partner, the recovery rates are unchanged in this case. Therefore, the process moves from state Z_{10} to state Z_{00} with rate γ_1, and from state Z_{01} to Z_{00} with rate γ_2. The remaining events and their probabilities occurring in the original model are unchanged. Additionally, the process still gives the same outcomes when the relationship ends (male and female infected, male infected, female infected and nobody infected) but the probabilities have changed. Hence, we obtain a formulation amenable to study. Using the same approach as in Section 2.3, we obtain the formulae for $h_{i,j}^{(k)}$, the probability of having i infected males j infected females, where $i, j \in \{0, 1\}$, starting from an

infected individual of type k, such that type 1 is male and type 2 is female, as follows. Let

$$D_1 = (\delta+\beta_1+\gamma_1)\left[(\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-(1-v_1)\gamma_1\beta_2\right]-(1-v_2)\gamma_2\beta_1(\beta_2+\delta+\gamma_2)$$
$$D_2 = (\delta+\beta_2+\gamma_2)\left[(\delta+\gamma_1+\gamma_2)(\beta_1+\delta+\gamma_1)-(1-v_2)\gamma_2\beta_1\right]-(1-v_1)\gamma_1\beta_2(\beta_1+\delta+\gamma_1).$$

Then

$$h_{0,0}^{(1)} = \frac{\gamma_1}{\gamma_1+\bar{\alpha}} + \frac{\left[(1-v_1)\gamma_1\gamma_2\beta_1+\beta_1(v_1\gamma_1+v_2\gamma_2)(\delta+\beta_2+\gamma_2)+\gamma_1((\delta+\gamma_1+\gamma_2)(\delta+\beta_2+\gamma_2)-(1-v_1)\gamma_1\beta_2)\right]\bar{\alpha}}{D_1(\gamma_1+\bar{\alpha})}$$
$$h_{1,0}^{(1)} = \frac{(\delta(\delta+\gamma_1+\gamma_2)(\beta_2+\delta+\gamma_2)-(1-v_1)\delta\gamma_1\beta_2)\bar{\alpha}}{D_1(\gamma_1+\bar{\alpha})}$$
$$h_{0,1}^{(1)} = \frac{(1-v_1)\gamma_1\delta\beta_1\bar{\alpha}}{D_1(\gamma_1+\bar{\alpha})}$$
$$h_{1,1}^{(1)} = \frac{\beta_1\delta(\delta+\beta_2+\gamma_2)\bar{\alpha}}{D_1(\gamma_1+\bar{\alpha})}$$

Similarly, the probabilities for each of the 4 outcomes starting with an infected female are

$$h_{0,0}^{(2)} = \frac{\gamma_2}{\gamma_2+\bar{\alpha}} + \frac{\left[(1-v_2)\gamma_1\gamma_2\beta_2+\beta_2(v_1\gamma_1+v_2\gamma_2)(\delta+\beta_1+\gamma_1)+\gamma_2((\delta+\gamma_1+\gamma_2)(\delta+\beta_1+\gamma_1)-(1-v_2)\gamma_2\beta_1)\right]\bar{\alpha}}{D_2(\gamma_2+\bar{\alpha})}$$
$$h_{1,0}^{(2)} = \frac{(1-v_2)\gamma_2\delta\beta_2\bar{\alpha}}{D_2(\gamma_2+\bar{\alpha})}$$
$$h_{0,1}^{(2)} = \frac{(\delta(\delta+\gamma_1+\gamma_2)(\beta_1+\delta+\gamma_1)-(1-v_2)\delta\gamma_2\beta_1)\bar{\alpha}}{D_2(\gamma_2+\bar{\alpha})}$$
$$h_{1,1}^{(2)} = \frac{\beta_2\delta(\delta+\beta_1+\gamma_1)\bar{\alpha}}{D_2(\gamma_2+\bar{\alpha})}.$$

Recall that R_0 is the dominant eigenvalue of the next generation matrix. Therefore, we have the same expression as in (2.7), that is

$$R_0^1 = \frac{T+\sqrt{T^2-4D}}{2}, \tag{4.1}$$

where

$$T = h_{10}^{(1)} + h_{11}^{(1)} + h_{01}^{(2)} + h_{11}^{(2)} \text{ and}$$
$$D = (h_{10}^{(1)} + h_{11}^{(1)})(h_{01}^{(2)} + h_{11}^{(2)}) - (h_{01}^{(1)} + h_{11}^{(1)})(h_{10}^{(2)} + h_{11}^{(2)}).$$

We have an expression for R_0^1 and its value will be computed numerically. The probability of extinction is the smallest non-negative fixed point of the probability generating functions as discussed in Section 2.4, Chapter 2, but with probabilities $h_{i,j}^{(k)}$.

$$g_k(s_1, s_2) = \sum_{j,i} h_{i,j}^{(k)} s_1^i s_2^j, \quad k = 1, 2. \tag{4.2}$$

Now, consider the case where there are no differences in parameters for both genders. According to Lemma 2.3.1 and Lemma 2.4.1 in Chapter 2, we can reduce the model to a single type model with

$$R_0 = h_{1,0}^{(1)} + h_{0,1}^{(1)} + 2h_{1,1}^{(1)} = 1 + h_{1,1} - h_{0,0}$$
$$z = \min\left\{1, \frac{h_{0,0}}{h_{1,1}}\right\}.$$

where z is the probability of extinction. For equal probability for both genders, we have the following

$$D_1 = (\delta + \beta + \gamma)\{(\delta + 2\gamma)(\delta + \beta + \gamma) - 2(1 - v)\beta\gamma\}$$

We can see that D_1 can be rewritten in the form $A + Bv$ where A and B are

functions of parameters other than v.

$$h_{0,0} = \frac{\gamma}{\gamma+\bar{\alpha}} + \frac{(1-v)\beta\gamma(\gamma-1)\bar{\alpha} + \beta(2v\gamma)(\delta+\beta+\gamma)\bar{\alpha} + \gamma\bar{\alpha}(\delta+2\gamma)(\delta+\beta+\gamma)\bar{\alpha}}{(\gamma+\bar{\alpha})D_1}$$
$$h_{1,1} = \frac{\beta\delta(\delta+\beta+\gamma)\bar{\alpha}}{D_1(\gamma+\bar{\alpha})}.$$

Similarly, $h_{0,0}$ can be rewritten as

$$h_{0,0} = \frac{\gamma D_1 + (C+Ev)}{(\gamma+\bar{\alpha})D_1}.$$

where C and E are functions of parameters other than v. Therefore,

$$\frac{h_{0,0}}{h_{1,1}} = \frac{\gamma D_1 + (C+Ev)}{\beta\delta(\delta+\beta+\gamma)\bar{\alpha}}.$$

Now we can note that v also features in D_1 which is a linear function in v. Therefore $h_{0,0}/h_{1,1}$ is of the form $(a+bv)/c$ where a, b and c are functions of parameters other than v, so the probability of extinction, z, is linear in v until it hits 1. Similarly we have that R_0 is of the form $1+(a+bv)/(c+dv)$ with $b<0$ and $d>0$. Thus we can show that R_0 is decreasing as v increases.

4.2.2 Endemic level

In this subsection, we study effects on the endemic level when the control measure is incorporated. With the control parameter $v>0$, we should expect to see a lower endemic level in this case. In terms of the model development, according to our assumptions in Subsection 4.2.1, we have that only the relationship states are adjusted, while the remaining equations in the deterministic model in 2.5.1 are

unchanged. Therefore, we have

$$\begin{aligned}
\frac{dx_0(t)}{dt} &= \gamma_1 x_1(t) + \delta(z_{01}(t) + z_{00}(t)) - \alpha x_0(t)(y_1(t) + y_0(t)),\\
\frac{dx_1(t)}{dt} &= -\gamma_1 x_1(t) + \delta(z_{11}(t) + z_{10}(t)) - \alpha x_1(t)(y_1(t) + y_0(t)),\\
\frac{dy_0(t)}{dt} &= \gamma_2 y_1(t) + \delta(z_{10}(t) + z_{00}(t)) - \alpha y_0(t)(x_1(t) + x_0(t)),\\
\frac{dy_1(t)}{dt} &= -\gamma_2 y_1(t) + \delta(z_{11}(t) + z_{01}(t)) - \alpha y_1(t)(x_1(t) + x_0(t)),\\
\frac{dz_{00}(t)}{dt} &= \gamma_1 z_{10}(t) + \gamma_2 z_{01}(t) + \alpha y_0(t)x_0(t) - \delta z_{00}(t) + (v_1\gamma_1 + v_2\gamma_2)z_{11}(t), \quad (4.3)\\
\frac{dz_{10}(t)}{dt} &= -\beta_1 z_{10}(t) - \gamma_1 z_{10}(t) + \alpha x_1(t)y_0(t) - \delta z_{10}(t) + (1 - v_2)\gamma_2 z_{11}(t),\\
\frac{dz_{01}(t)}{dt} &= -\gamma_2 z_{01}(t) - \beta_2 z_{01}(t) + \alpha x_0(t)y_1(t) - \delta z_{01}(t) + (1 - v_1)\gamma_1 z_{11}(t),\\
\frac{dz_{11}(t)}{dt} &= \beta_1 z_{10}(t) + \beta_2 z_{01}(t) - (\gamma_1 + \gamma_2)z_{11}(t) + \alpha y_1(t)x_1(t) - \delta z_{11}(t).\\
n(t) &= x_1(t) + x_0(t) + y_1(t) + y_0(t) + z_{00}(t) + z_{10}(t) + z_{01}(t) + z_{11}(t) = 1.
\end{aligned}$$

Note that, similar alterations are applied in the stochastic analogue of the deterministic model.

4.3 Model 2 with a control measure

Model 2 is a sexual model with one-night stands in Chapter 3. Similar to Section 4.2, we are looking at incorporating medication use as a control measure into the model. Recall that, for model 2, we constructed a 5-type branching process approximation for determining and the probability of extinction.

4.3.1 The reproduction number and probability of extinction

The 5 types of individuals are composed of a single infected male, a couple within which only the male is infected, a couple within which both male and female are

infected, a couple within which only the female is infected, and a single infected female. They are classified as individuals of type 1, 2, 3, 4 and 5, respectively. From previous studies, to incorporate the control measure, we observe that only the relationship state within which both male and female are infected is adjusted (type 3). Table 4.1 shows the probabilities of events occurring when the process starts from an individual of type 3. Note that other events with their probabilities in Table 3.1 remain the same.

Table 4.1: Events and their corresponding probabilities when the process starts with an individual of type 3.

Initial infective$^{(type)}$	Probability	Offspring$^{(type)}$
$M_I F_I^{(3)}$	$\frac{\delta}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2p)}$	$M_I{}^{(1)}$,$\mathrm{F}_I{}^{(5)}$
	$\frac{\omega_1 rq}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^p)}$	$M_I F_I{}^{(3)}$,$\mathrm{F}_I{}^{(5)}$
	$\frac{\omega_1 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2p)}$	$M_S F_I{}^{(4)}$,$\mathrm{M}_I F_I^{(3)}$
	$\frac{\omega_2 rq}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2p)}$	$M_I{}^{(1)}$,$\mathrm{M}_I F_I{}^{(3)}$
	$\frac{\omega_2 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2p)}$	$M_I F_S{}^{(2)}$,$\mathrm{M}_I F_I^{(3)}$
	$\frac{(1-v_1)\gamma_1}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2p)}$	$M_S F_I^{(4)}$
	$\frac{(1-v_2)\gamma_2}{\delta+\gamma_1+\gamma_2+r(\omega_1+\omega_2)(rq+r^2p)}$	$M_I F_S^{(2)}$
	$\frac{v_1\gamma_1+v_2\gamma_2}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2p)}$	$M_S F_S^{(\emptyset)}$

To seek the reproduction number, we generate a 5×5 next-generation matrix such that an element $m_{i,j}$ denotes the probability of having 1 offspring of type j from an initial offspring of type i. Using the information from Table 3.1 and 4.1, we have the next-generation matrix as follows.

The next-generation matrix

$$M = \begin{bmatrix} m_{1,1} & m_{1,2} & 0 & m_{1,4} & m_{1,5} \\ m_{2,1} & m_{2,2} & m_{2,3} & m_{2,4} & m_{2,5} \\ m_{3,1} & m_{3,2} & m_{3,3} & m_{3,4} & m_{3,5} \\ m_{4,1} & m_{4,2} & m_{4,3} & m_{4,4} & m_{4,5} \\ m_{5,1} & m_{5,2} & 0 & m_{5,4} & m_{5,5} \end{bmatrix} \tag{4.4}$$

$m_{1,1} = \frac{\omega_1 q+\omega_1 rp}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}.$ $m_{1,2} = \frac{\bar{\alpha}}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}.$

$m_{1,3} = 0.$ $m_{1,4} = \frac{\omega_1 rp}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}.$

$m_{1,5} = \frac{\omega_1 q}{\gamma_1+\bar{\alpha}+\omega_1 q+\omega_1 rp}.$ $m_{2,1} = \frac{\delta}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p}.$

$m_{2,2} = \frac{\omega_1 rq+\omega_1 r^2 p}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p}$ $m_{2,3} = \frac{\beta_1}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p}$

$m_{2,4} = \frac{\omega_1 r^2 p}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p}$ $m_{2,5} = \frac{\omega_1 rq}{\beta_1+\gamma_1+\delta+\omega_1 rq+\omega_1 r^2 p}$

$m_{3,1} = \frac{\delta+\omega_2 rq}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$ $m_{3,2} = \frac{(1-v_2)\gamma_2+\omega_2 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$

$m_{3,3} = \frac{\omega_1 rq+\omega_1 r^2 p+\omega_2 rq+\omega_2 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$ $m_{3,4} = \frac{(1-v_1)\gamma_1+\omega_1 r^2 p}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$

$m_{3,5} = \frac{\delta+\omega_1 rq}{\delta+\gamma_1+\gamma_2+(\omega_1+\omega_2)(rq+r^2 p)}$ $m_{4,1} = \frac{\omega_2 rq}{\gamma_2+\beta_2+\delta+\omega_2 rq+\omega_2 r^2 p}$

$m_{4,2} = \frac{\omega_2 r^2 p}{\gamma_2+\beta_2+\delta+\omega_2 rq+\omega_2 r^2 p}$ $m_{4,3} = \frac{\beta_2}{\gamma_2+\beta_2+\delta+\omega_2 rq+\omega_2 r^2 p}$

$m_{4,4} = \frac{\omega_2 rq+\omega_2 r^2 p}{\gamma_2+\beta_2+\delta+\omega_2 q+\omega_2 rp}$ $m_{4,5} = \frac{\delta}{\gamma_2+\beta_2+\delta+\omega_2 rq+\omega_2 r^2 p}$

$m_{5,1} = \frac{\omega_2 q}{\gamma_2+\bar{\alpha}+\omega_2 q+\omega_2 rp}$ $m_{5,2} = \frac{\omega_2 rp}{\gamma_2+\bar{\alpha}+\omega_2 q+\omega_2 rp}$

$m_{5,3} = 0$ $m_{5,4} = \frac{\bar{\alpha}}{\gamma_2+\bar{\alpha}+\omega_2 q+\omega_2 rp}$

$m_{5,5} = \frac{\omega_2 q+\omega_2 rp}{\gamma_2+\bar{\alpha}+\omega_2 q+\omega_2 rp}.$

Again, R_0 is the dominant eigenvalue of matrix M above. The probability of extinction is the smallest non-negative root of the system of equations of the probability generating functions defined similarly to (3.14), Chapter 3, which is

$$g_k(\mathbf{s}) = \sum_{x_1,x_2,x_3,x_4,x_5} h^{(k)}_{x_1,x_2,x_3,x_4,x_5} s_1^{x_1} s_2^{x_2} s_3^{x_3} s_4^{x_4} s_5^{x_5}. \tag{4.5}$$

We have proved in Section 4.2 that R_0 is a decreasing function in v for Model 1. We are also interested in the relationship between R_0 and the control parameter v for Model 2 but this is more difficult to analyse. However R_0 is only one indicator of the likelihood of a major epidemic outbreak with the probability of extinction being more informative. We analyse the probability of extinction for Model 2 in the case where there is no distinction between the sexes and can reduce the analysis to a 3-type branching process approximation. Specifically we consider a 3 type branching process, consisting of type 1 - a single infective, type 2 - a couple within which only 1 infective, and type 3 - a couple with 2 infectives. These correspond to the infectious units in the branching process. Therefore, we will refer them as types 1, 2 and 3 infective, respectively. Each type of infective will produce at most 2 offspring. Type 1 infectives produce either 2 offspring of type 1, 1 offspring of type 1 and 1 offspring of type 2, or 1 offspring of type 2. Type 2 infectives produce either 1 offspring of type 1 and 1 offspring of type 2, 2 offspring of type 2, 1 offspring of type 3, or 1 offspring of type 1. Type 3 infectives produce either 1 offspring of type 1 and 1 offspring of type 3, 1 offspring of type 2 and 1 offspring of type 3, 1 offspring of type 2, or 2 offspring of type 1. We then construct 2 branching processes.

Let $\mathcal{B}_k$ represent the branching process of index k, for $k = 1, 2$, and v_k be a control parameter used in the branching process $\mathcal{B}_k$. Let $p^k_{n_i m_j}(l)$ denote the probability of n offspring of type i and m offspring of type j produced from a type l infective in the branching process $\mathcal{B}_k$, where $m, n = \{0, 1, 2\}$ and $l, i, j = \{1, 2, 3\}$. For example, $p^1_{1_2}(3)$ denote the probability of 1 offspring of type 2 produced from a type 3 infective in the branching process $\mathcal{B}_1$. According to the construction of the branching processes, we have that for $\mathcal{B}_1$ and $\mathcal{B}_2$, $p^1_{n_i m_j}(l) = p^2_{n_i m_j}(l)$ except $p^1_{1_2}(3) \neq p^2_{1_2}(3)$. Suppose $v_1 > v_2$. Then, in $\mathcal{B}_2$, let

$$p^2_{1_2}(3) = p^1_{1_2}(3) + p^*, \quad p^* > 0 \tag{4.6}$$

We couple the two branching processes such that every individual in $\mathcal{B}_1$ has a corresponding individual in $\mathcal{B}_2$ and that the corresponding individuals have the same offspring except that there is probability p^* that there will be a type 2 individual born in $\mathcal{B}_2$ from a type 3 individual in cases where the corresponding individual in $\mathcal{B}_1$ has no offspring. The type 2 individual and their offspring have no corresponding individuals in the branching process $\mathcal{B}_1$. Define

$$p^k_{1_1 1_3}(3) + p^k_{1_2 1_3}(3) + p^k_{1_2}(3) + p^k_{2_1}(3) = a^k$$

According to (4.6), we have that $p^2_{1_2}(3) = p^1_{1_2}(3) + p^*$. Therefore, in $\mathcal{B}_2$, we have

$$p^1_{1_2}(3) + (1 - a^1)\frac{p^*}{1 - a^1} = p^1_{1_2}(3) + p^* = p^2_{1_2}(3) \tag{4.7}$$

Let π^k_j denote the probability of a branching process $\mathcal{B}_k$ going extinct starting from a type j infective. The above statement immediately implies that $\pi^2_j \leq \pi^1_j$. It also follows that if $\pi^1_j = 0$, then $\pi^2_j = 0$. Therefore, we want to ensure that this also holds for the case that the branching process $\mathcal{B}_1$ does go extinct, i.e. $\pi^1_j > 0$. Assume that $\pi^1_j > 0$. Again, we consider a type 3 infective. For convenience, we drop the index l of type 3 here. In $\mathcal{B}_2$, we have

$$\begin{aligned}
\pi^2_3 &= p^2_{1_1 1_3}\pi^2_1\pi^2_3 + p^2_{1_3 1_2}\pi^2_3\pi^2_2 + p^2_{1_2}\pi^2_2 + p^2_{2_1}(\pi^2_1)^2 + (1 - a^2) \\
&= p^1_{1_1 1_3}\pi^2_1\pi^2_3 + p^1_{1_3 1_2}\pi^2_3\pi^2_2 + p^1_{1_2}\pi^2_2 + p^*(\pi^2_2 - 1) && \text{(using (4.7))} \\
&\quad + p^1_{2_1}(\pi^2_1)^2 + (1 - a^1) \\
&< p^1_{1_1 1_3}\pi^2_1\pi^2_3 + p^1_{1_3 1_2}\pi^2_3\pi^2_2 + p^1_{1_2}\pi^2_2 + p^1_{2_1}(\pi^2_1)^2 + (1 - a^1) && (iff \quad \pi^2_2 < 1) \\
&\leq p^1_{1_1 1_3}\pi^1_1\pi^1_3 + p^1_{1_3 1_2}\pi^1_2\pi^1_3 + p^1_{1_2}\pi^1_2 + p^1_{2_1}(\pi^1_1)^2 + (1 - a^1) && (\because \pi^2_j \leq \pi^1_j) \\
&= \pi^1_3
\end{aligned}$$

Therefore, $\pi^2_3 < \pi^1_3$. This argument has shown that if $v_2 < v_1$, then $\pi^2_j < \pi^1_j$,

implying that the probability of extinction strictly increases as v increases. This is informative in terms of the epidemic behaviour for which the probability of extinction less than 1, especially the behaviour of R_0 with respect to v is not achieved in this case. The probability of extinction indicates that v decreases the incidence of the disease since the disease dies out faster. Based upon our previous studies, we would not expect different behaviour for R_0. Our conjecture can be further supported in the numerical studies in Subsection 4.4.2.

4.3.2 Endemic level

In this Subsection, similarly, the relationship states are adjusted to take into account of medication whilst the remaining states are unchanged. The equations are expressed as follows.

$$
\begin{aligned}
\frac{dx_0(t)}{dt} &= \gamma_1 x_1(t) + \delta(z_{01}(t) + z_{00}(t)) - \alpha x_0(t)(y_1(t) + y_0(t)) - \omega_2 x_0(t) y_1(t) \\
&\quad - \omega_2 r x_0(t)(z_{01}(t) + z_{11}(t)), \\
\frac{dx_1(t)}{dt} &= -\gamma_1 x_1(t) + \delta(z_{11}(t) + z_{10}(t)) - \alpha x_1(t)(y_1(t) + y_0(t)) + \omega_2 x_0(t) y_1(t) \\
&\quad + \omega_2 r x_0(t)(z_{01}(t) + z_{11}(t)), \\
\frac{dy_0(t)}{dt} &= \gamma_2 y_1(t) + \delta(z_{10}(t) + z_{00}(t)) - \alpha y_0(t)(x_1(t) + x_0(t)) - \omega_1 x_1(t) y_0(t) \\
&\quad - \omega_1 r y_0(t)(z_{10}(t) + z_{11}(t)), \\
\frac{dy_1(t)}{dt} &= -\gamma_2 y_1(t) + \delta(z_{11}(t) + z_{01}(t)) - \alpha y_1(t)(x_1(t) + x_0(t)) + \omega_1 x_1(t) y_0(t) \\
&\quad + \omega_1 r y_0(t)(z_{10}(t) + z_{11}(t)), \qquad (4.8) \\
\frac{dz_{00}(t)}{dt} &= \gamma_1 z_{10}(t) + \gamma_2 z_{01}(t) + \alpha x_0(t) y_0(t) - \delta z_{00}(t) - \omega_2 r y_1(t) z_{00}(t) - \omega_1 r x_1(t) z_{00}(t) \\
&\quad - \omega_2 r^2 z_{00}(t)(z_{01}(t) + z_{11}(t)) - \omega_1 r^2 z_{00}(t)(z_{10}(t) + z_{11}(t)) + (v_1\gamma_1 + v_2\gamma_2) z_{11}(t), \\
\frac{dz_{10}(t)}{dt} &= (1 - v_2)\gamma_2 z_{11}(t) - \beta_1 z_{10}(t) - \gamma_1 z_{10}(t) + \alpha x_1(t) y_0(t) - \delta z_{10}(t) + \omega_2 r y_1(t) z_{00}(t) \\
&\quad + \omega_2 r^2 z_{00}(t)(z_{01}(t) + z_{11}(t)) - \omega_1 r x_1(t) z_{10}(t) - \omega_1 r^2 z_{10}(t)(z_{10}(t) + z_{11}(t)), \\
\frac{dz_{01}(t)}{dt} &= (1 - v_1)\gamma_1 z_{11}(t) - \gamma_2 z_{01}(t) - \beta_2 z_{01}(t) + \alpha x_0(t) y_1(t) - \delta z_{01}(t) + \omega_1 r x_1(t) z_{00}(t) \\
&\quad + \omega_1 r^2 z_{00}(t)(z_{10}(t) + z_{11}(t)) - \omega_2 r y_1(t) z_{01}(t) - \omega_2 r^2 z_{01}(t)(z_{11}(t) + z_{01}(t)), \\
\frac{dz_{11}(t)}{dt} &= \beta_1 z_{10}(t) + \beta_2 z_{01}(t) - (1 - v_1)\gamma_1 z_{11}(t) - (1 - v_2)\gamma_2 z_{11}(t) + \alpha y_1(t) x_1(t) - \delta z_{11}(t) \\
&\quad + \omega_1 r x_1(t) z_{10}(t) + \omega_1 r^2 z_{10}(t)(z_{10}(t) + z_{11}(t)) - (v_1\gamma_1 + v_2\gamma_2) z_{11}(t)
\end{aligned}
$$

We now have the ingredients for analysing the effect of the control measure. We will be looking at R_0, the probability of extinction, and the endemic level. This will be done numerically in Section 4.4.

4.4 Numerical Results

We implemented the models derived in Section 4.2 and 4.3 in R. The numerical results are presented in this Section. We first study Model 1 in Section 4.4.1.

4.4.1 Model 1 with a control measure

Our aim in this subsection is to investigate the effect of v on R_0 and the endemic level. First, we start with the case where there is no difference in rates between sexes, that is $\gamma_1 = \gamma_2$, $\beta_1 = \beta_2$, and $v_1 = v_2$. As v increases, there is a higher chance that a diagnosed individual and his/her partner will be recovering simultaneously with rate $2v\gamma$, and with rate $(1 - v)\gamma$ if only one person in a relationship is recovering. The chance that infected individuals will be simultaneously recovering also depends upon the proportion of individuals in relationships and how fast the turnover of relationships. Therefore, it is worthwhile to explore the correlation between v and the model parameters. We know from Section 4.2 that R_0 decreases as v increases. Therefore, in this case, we choose the most effective control measure which is $v = 1$ and explore how v influences the model whilst varying each of the model parameters. We choose the study in Subsection 2.7.1 as the basis for this case. Therefore, our parameters are chosen as the same as in Table 2.2 -2.5 which are recalled here.

Table 4.2: Parameters chosen for varying infectious rate

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$
1	9	9	2	2	1:100	1:100
2	30	50	1	1	1:100	1:100
3	0.3	0.8	0.1	0.1	1:100	1:100
4	1000	1000	5	5	1:100	1:100

Table 4.3: Parameters chosen for varying recovery rate

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$
1	3	6	1:100	1:100	100	100
2	1000	1000	1:100	1:100	20	20
3	30	0.1	1:100	1:100	500	500
4	10	100	1:100	1:100	300	300

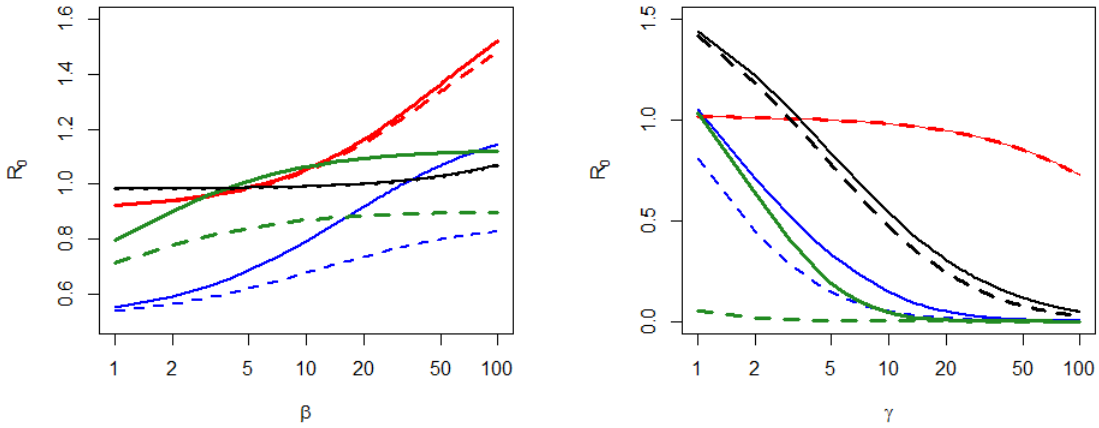
Table 4.4: Parameters chosen for varying rationship formation rate

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$
1	1:100	6	2	2	20	20
2	1:100	1000	1	1	50	50
3	1:100	1	0.1	0.1	0.5	0.5
4	1:100	0.1	1	1	80	80

Table 4.5: Parameters chosen for varying breakup rate

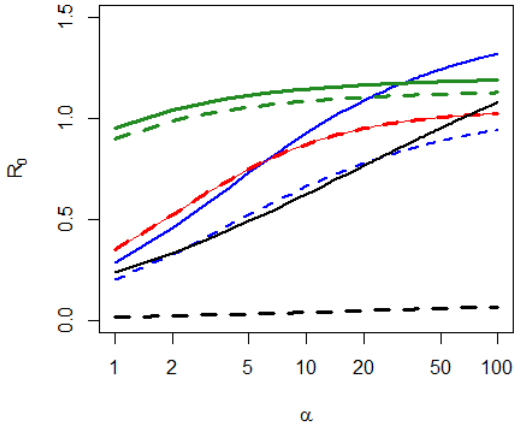
set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$
1	5	1:100	1	1	100	100
2	0.8	1:100	0.02	0.02	0.5	0.5
3	100	1:100	0.1	0.1	0.2	0.2
4	200	1:100	3	3	40	40

Note that, we are interested only in the behaviour of R_0 as each parameter increases, therefore, the results are plotted on the log scale in Figure 4.2. Each colour in Figure 4.2 represents the following : blue - set 1, red - set 2, green - set 3 and black - set 4. There are two lines for each colour. The normal line represents R_0 for the original model without control ($v = 0$) and the dash line represents R_0 for the model with control ($v = 1$). The distance between the normal line and the dash line of the same colour shows the effectiveness of v.

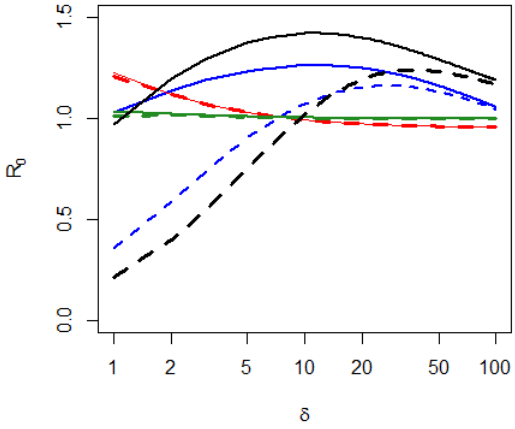


(a)

(b)



(c)



(d)

Figure 4.2: (a) Plot of R_0 for the model with and without control measure, whilst varying β based on parameter sets in Table 4.2
(b) Plot of R_0 for the model with and without control measure, whilst varying γ based on parameter sets in Table 4.3
(c) Plot of R_0 for the model with and without control measure, whilst varying α based on parameter sets in Table 4.4
(d) Plot of R_0 for the model with and without control measure, whilst varying δ based on parameter sets in Table 4.5

According to Figure 4.2, v has no effect on R_0 when δ is large as we can see from parameter set 4 (black) in Figure 4.2(a) and parameter set 2 (red) in Figure 4.2(b), 4.2(c) and 4.2(d). In the case where the model parameters are

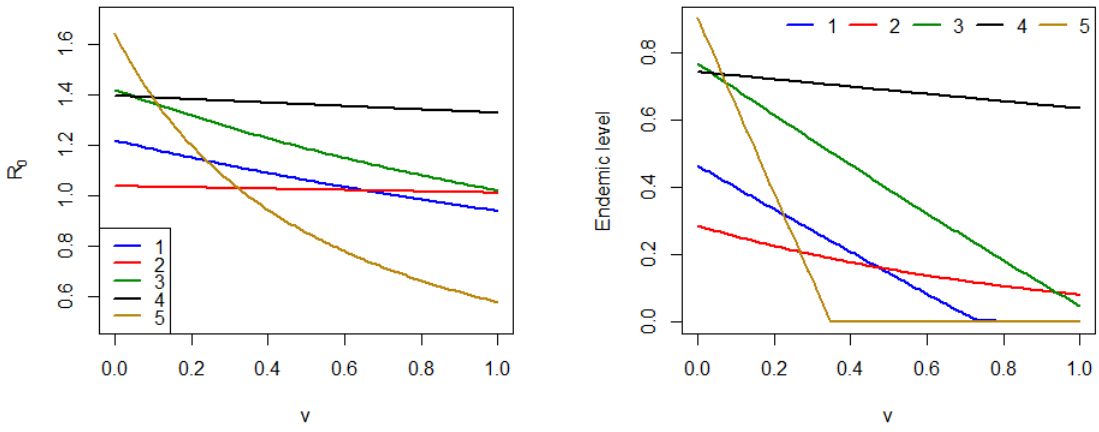
moderate, we observe that as β and α increase, v is increasingly effective and decreasingly effective as γ and δ increase. Moreover, parameter sets 3 and 4 in Figure 4.2(b) and 4.2(c) are also very interesting as v has a dramatic effect on R_0. δ in both sets are very small in comparison to α. This describes that the model has large proportion of couples and small relationship turnovers. In other words, individuals spend some time in relationships. We note that the control measure v takes place within relationships. Therefore, as individuals tend to stay in a relationship, the probability that the recovery takes place within relationships increases. However, even given high α, if δ is high as well, this means there is large relationship turnovers in which the breakup and forming relationships happen too fast before other events could happen. We see that v has no effect on R_0 in this case (set 4 and 2 in Figure 4.2(a) and (b)).

Now, we consider investigating effect of v on R_0 for varied set of parameters. In this case, we vary v from 0 to 1 by 0.01. We know that $v = 1$ is the most effective strategy. However, in some cases, choosing $v = 1$ is unnecessary if $v < 1$ has a significant effect and is able to bring R_0 below 1. As a result, we are interested in investigating how v affects the model as v increases, so as to provide a guide to the required level of the control measure v. The parameters are chosen as in Table 4.6.

Table 4.6: Parameters chosen

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma}$	$\boldsymbol{\beta}$	$\boldsymbol{R_0}$
1	15	10	2	50	1.2190
2	100	1	0.1	0.2	1.0369
3	200	10	3	40	1.4211
4	30	50	2	100	1.3955
5	1	0.01	0.01	0.8	1.6456

Figure 4.3 shows the behaviour of R_0 and the endemic level, when v increases with respect to 5 sets of parameters in Table 4.6. As we are interested in the actual effect of v, the results plotted in Figure 4.3 are on a linear scale. Each colour represents R_0 with respect to each parameter set as labelled in the figure.



(a) (b)

Figure 4.3: Plot of R_0 and the endemic level corresponding to parameter sets in Table 4.6

We can see in Figure 4.3(a) that R_0 decreases as v increases. We can also see that v has different impact on R_0 for different parameter sets. However, v has small effect on R_0 for parameter set 2 and 4, whereas, there is a significant effect on R_0 for parameter set 5.

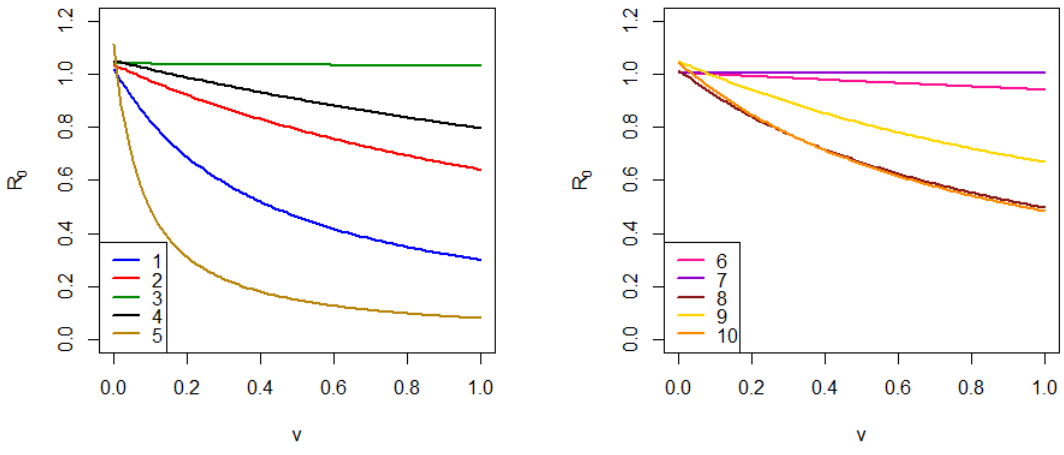
Note that, our aim in reducing the spread of the disease is to be able to reduce the number of infected individuals in the population. R_0 is an indicator whether or not the disease is severe, but the effect on the endemic level is important. Once we reduce R_0 below 1 the endemic level is 0 and stays there as v increases. Therefore, we are also interested to see how v makes an impact on the endemic level corresponding to the same parameter sets as in Table 4.6, see Figure 4.3(b). We can see from the figure that the endemic level becomes 0 at $v = 0.35$ for parameter set 5. For parameter set 1, the endemic level becomes 0 at $v = 0.79$. In the case that R_0 is not reduced below 1, we would choose to reduce the endemic level as much as possible. Also, we can see that a dramatic decrease in R_0 tends to coincide with a dramatic decrease in the endemic level.

We know that v affects R_0 and the endemic level in a different manner de-

pending upon how the model parameters are varied. Now, we are interested to see how v affects R_0 if the chosen sets of parameters give the same endemic level. We investigate this by studying different sets of parameters which all have an endemic level of 14% when $v = 0$. We study how R_0 changes as v varies from 0 to 1 whilst keeping the other parameters constant. In particular, we are interested in the value of v, if any, for which $R_0 = 1$ as this can be used to identify the critical vaccination coverage beyond which the disease can not become endemic. Note that, the sets of parameters are chosen from Table 2.10 and the rates between the two sexes are different in this case.

Table 4.7: Parameters chosen for fixed endemic level at 0.14

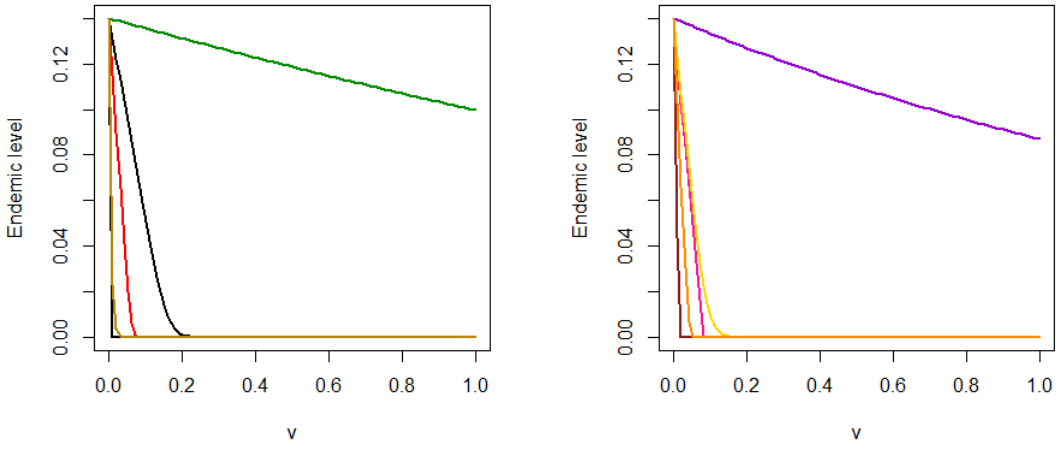
set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma_1}$	$\boldsymbol{\gamma_2}$	$\boldsymbol{\beta_1}$	$\boldsymbol{\beta_2}$	$\boldsymbol{R_0}$
1	40	50	1	124.0645	5000	622.162	1.0174
2	100	5	1	5.4542	15	8.2952	1.0349
3	1	99	1	0.0401	41.0168	1000	1.0396
4	8.59	6	1	2	18.5448	20	1.0498
5	243.6944	1	1	15	100	200	1.1126
6	9	10	1	0.05	0.1540	30	1.0056
7	150	100	1	0.7498	1	10.4524	1.0041
8	0.5	0.8	1	0.01	1.6861	9	1.0152
9	14	2	1	1	3.4758	50	1.0483
10	11	7.1065	1	8	200	211	1.0436



(a) (b)

Figure 4.4: Plots of R_0 against v corresponding to parameter sets in Table 4.7

From Figure 4.4, we can see that v affects R_0 in a different manner for different sets of parameters. However, for parameter sets 3 and 7, we can see that v makes too small effect on R_0 so we can not see the decrease on the scale of the graph. Whereas, R_0 for other parameter sets are brought below 1 very quickly. As soon as R_0 is below 1, the effect of v on R_0 is no longer interesting. We also investigate how v affects the endemic level based on the same parameter sets in Figure 4.5.



(a) (b)

Figure 4.5: Plots of the endemic level against v corresponding to parameter sets in Table 4.7

In Figure 4.5, the behaviour of endemic level is consistent with the behaviour of R_0. What we can see clearer is the parameter sets 3 and 7 that the endemic level shows clearer decreasing behaviour. In the next subsection, we will study the effect of v on Model 2.

4.4.2 Model 2 with a control measure

Similar to Subsection 4.4.1, we study the effect of v on Model 2. In Subsection 4.4.1, we explored the relationship between the effectiveness of v and each parameter, β, γ, α and δ, by comparing R_0 between the model without control ($v = 0$) and with control ($v = 1$). In this subsection, we are also interested in ω. We also start our studies with no difference in rates between the two sexes. 4 different sets of parameters covering a range of behaviours, such as large α and small δ and vice versa. ω is increasing from 1 to 100 and $v = 1$. The colours representing each set of parameters are as labelled in Figure 4.6. The normal line represents the model without control ($v = 0$) and the dash line represents the model with control ($v = 1$). The distance between the two lines shows how much R_0 is reduced by $v = 1$, in other words, the effectiveness of v. Parameters are chosen as shown in Table 4.8.

Table 4.8: Parameters chosen

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma}$	$\boldsymbol{\beta}$
1	100	0.1	1	80
2	9	6	30	2
3	0.1	100	1	2
4	1000	1000	3	8

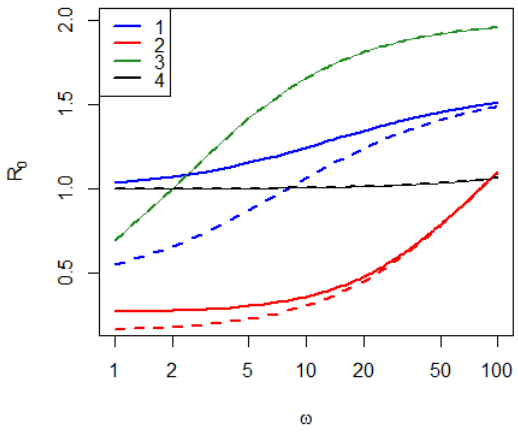


Figure 4.6: Plots of the R_0 against v corresponding to parameter sets in Table 4.8

In Figure 4.6, we can see that v almost has no effect on R_0 for parameter set 3 and 4 in which δ is large for both sets. This supports our studies for Model 1. For parameter set 1, v has significant effect on R_0 and is increasingly less effective as ω increases. Similarly, for parameter set 2, v is less effective as ω increases since we can see for both sets that the normal line and the dash line converges. As a result, we can say that when ω is large, v has no effect on the model. This makes sense as when ω is reasonably large, the infection is driven by one-night stands which can occur outside relationships, whereas, the control measure v helps increasing the recovery only within relationships.

In terms of the effect of v on R_0 whilst v increases from 0 to 1, we should see a decreasing behaviour as it has been addressed in Section 4.3 that R_0 decreases in v. However, for different sets of parameters, we expect to see v affect R_0 in a different manner. It is worth exploring what kind of behaviours we could see in this model. From our previous studies, we know that for very large ω or δ, v has no effect on R_0 so it is no longer interesting to explore the effect of v in these cases. Therefore, we choose different sets of parameters ensuring there is an effect on R_0 in order to study how v affects R_0 as v increases. Parameters are as the following. Plots in Figure 4.7 are on a linear scale.

Table 4.9: Parameters chosen

set	$\boldsymbol{\alpha}$	$\boldsymbol{\delta}$	$\boldsymbol{\gamma}$	$\boldsymbol{\beta}$	ω
1	5	1	1	100	10
2	6	9	1	2	2
3	100	0.1	2	50	3
4	0.1	0.01	0.1	0.2	0.5

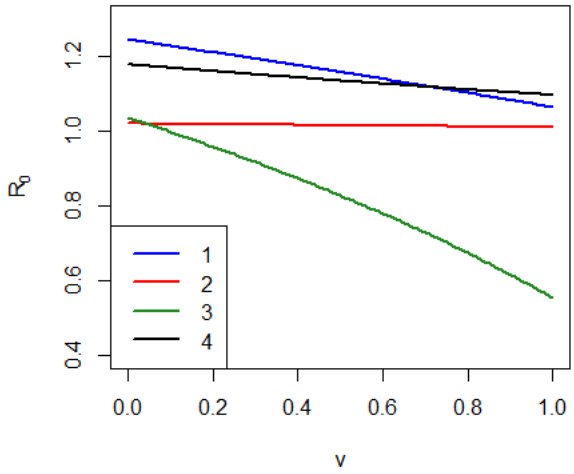


Figure 4.7: Plots of R_0 against v corresponding to parameter sets in Table 4.9

As expected, we see a decreasing behaviour of R_0 as v increases but with a different slope for different parameter sets. We can see that v is most effective on parameter set 1 and 3, whereas for other parameter sets we only see slight effect. Next, we plot the corresponding endemic level to the R_0 in Figure 4.7 using the same parameters in Table 4.9.

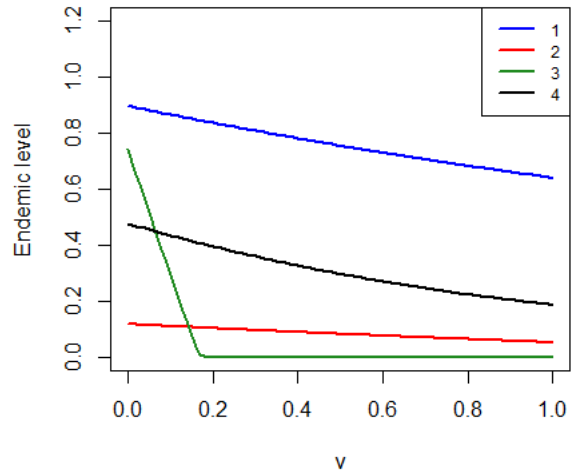


Figure 4.8: Plots of R_0 against v corresponding to parameter sets in Table 4.9

As we can see in Figure 4.8, the endemic level has a consistent behaviour corresponding to R_0. In other words, $R_0 < 1$, the endemic level is 0 and greater than 0 if $R_0 > 1$. Also, we can see that a large decrease in R_0 results in a large decrease in the endemic level as well.

In reality, it is hard to control v as there are several factors influencing the value of v. However, studying how v affects the quantities of interest is useful in terms of investigating whether or not it is worthwhile to invest for higher v.

4.5 Applications to gonorrhoea

We have constructed a mathematical model describing sexually transmitted disease dynamics with the theoretical and numerical results being extensively studied. In this section, we are interested in seeing how our model fits to real world situations by considering how it can be used to describe a particular disease. One of the most common STDs is gonorrhoea and it has received a lot of attention in the literature (Hethcote & Yorke (1984); Kretzschmar et al. (1996); Lloyd-Smith et al. (2003); Garnett et al. (1999)). Gonorrhoea is an STD caused by bacteria that has the 3 following epidemiological characteristics (Hethcote & Yorke (1984)) :

- The infection does not confer protective immunity, which means individuals will be susceptible again as soon as they recover from infection.
- The latent period is much shorter than the infectious period.
- It has relatively small seasonal oscillations resulting in good approximation by a model with constant parameter values.

Based upon the above characteristics of gonorrhoea the SIS epidemic model gives a reasonable approximation of the disease dynamics. Since gonorrhoea is the most frequently reported STD, almost 700,000 cases were reported in 1990 (Tanfer et al. (1995)), we can obtain parameter estimates from the literature for our case

study. In this section, we will look at 3 case studies : Case study 1, there is no difference in parameters for males and females ; Case study 2, the parameters are different for both sexes; and Case study 3, the inclusion of one-night stands into the model. Note that one-night stands are not taken into account in Case study 1 and Case study 2.

Case study 1 : In this study, our parameter values will be chosen based on the parameter estimates available in Stigum et al. (1997). The paper focuses on the study of 3 different sexually transmitted diseases; gonorrhoea, chlamydia, and human immunodeficiency virus (HIV) of which gonorrhoea will be our focus here. The data were collected from 8,477 heterosexual Norwegians aged 18-52 with 3,060 people subject to potential STD risk behaviour. The authors considered two levels of sexual activity, high and low. The population with high level of sexual activity is referred to as the "core group" and the other group is referred to as the "non-core group". The effect of migration from the core group to the non-core group in which there is a partner mixing between the two groups was also considered. In our case, we will consider the case under the assumption of no partner mixing between the two groups (0% migration) and compare our results with the literature. Our chosen time unit is one year. The relevant parameter values provided in the literature are summarised in Table 4.10. Note that, the data is a per year average.

Table 4.10: Parameter values taken from Stigum et al. (1997)

	value	
parameter (symbol)	core	noncore
Proportion of the population (θ)	0.025	0.975
Partner frequencies (ν)	7.6	0.77
Unprotected intercourses (λ)	109.6	55.9
Transmission rate per episode of sexual intercourse (κ)	0.3	0.3
Duration of infectiousness (D)	0.3	0.3

The parameters in Table 4.10 will be recalculated to fit with our model as-

sumptions. The calculations for the infection rate (β) and the recovery rate (γ) appear to be straightforward and will be carried out according to the following formulae, $\beta = \lambda\kappa$, $\gamma = 1/D$. In order to determine δ and α, we employ the assumption that healthy individuals will divide their time spent approximately equally between being single and in a relationship, see for example, Lloyd-Smith et al. (2003). Therefore, $\delta = \bar{\alpha}$. Then δ and α can be calculated in terms of the mean number of partners: $\nu = \frac{1}{\bar{\alpha}} + \frac{1}{\delta}$. Also, since $\bar{\alpha} = \alpha(\sigma/2)$, then δ and α can be determined accordingly. The results of the calculations are expressed in Table 4.11. The results based on the parameters in Table 4.12 are illustrated in Table 4.11.

Table 4.11: parameter values used in the simulations

	value	
Parameter	core	non-core
Relationship breakup rate (δ)	15.20	1.54
Relationship formation rate (α)	60.80	6.16
Infection rate (β)	32.70	16.77
Recovery rate (γ)	3.33	3.33

Table 4.12: Result from Model 1 using parameters in Table 4.11

	Result	
Quantity of interest	core	non-core
R_0	1.2061	0.3091
Probability of extinction	0.5441	1
Endemic level	0.5059	0

The results in Table 4.12 show that infection can persist in the core group but not in the non-core group alone ($R_0 > 1$ for core group and $R_0 < 1$ for non-core group). This agrees with the results in the literature for the case that there is no migration from the core group. In the case where there is migration, our model could also be extended to two groups of individuals with different level of sexual activities. When there is migration, an individual from the core group can also choose to form a relationship with individual of the opposite sex in the non- group

and that there is the proportional mixing between the two groups. The rate of mixing between the two groups would then need to be defined.

In terms of the mean time to extinction, due to the large proportion infected with the endemic level slightly greater than 50% in the core group, the disease would persist in the population for a very long time before going extinct. The study in Britton & Neal (2010) suggested that the mean time to extinction should decrease in the infection rate within household, but the actual analysis could not be presented in the case that the endemic level is greater than half of the population with the reason being that the mean time to extinction is very large even for a small population. As a result, in our case, we would also not expect to be able to achieve the mean time to extinction from the simulation.

Case study 2: Similar to Case study 1, we employ parameter estimates existing in the literature and in this case the rates between genders are different (Over & Piot (1993)). The authors studied the transmission dynamic of HIV as well as other types of sexually transmitted diseases including gonorrhoea. Core and non-core groups which have different sizes and different rates of sexual activity are also considered. The size of core and non-core group were assumed to be 1,000 and 50,000 individuals, respectively. This was based upon an assumption that the size of core group is 2% of the non-core population. It is assumed that individuals in the core group are 10 times as sexually active as those in the non-core group. Individuals in the core group tend to have a new sexual partner every 5 days and every 50 days for individuals in the non-core group (Hethcote & Yorke (1984)). The information was based on the daily basis, therefore we will see much smaller parameter values in this case. Parameter estimates from the literature are summarised in Table 4.13. Parameters used in our model which have already been recalculated in order to fit with our model assumption are shown in Table 4.14. The results are illustrated in Table 4.15.

Table 4.13: Parameters taken from Over & Piot (1993)

parameter	estimated value
Transmission probability per sexual partner	
Male to female	0.6
Female to male	0.4
Duration of infectivity	
Male	45
Female	120
New sexual partners per day	
Core group	0.2
Non-core group	0.02

Table 4.14: Recalculated parameter values

		Sexual activity	
parameter	symbol	core	non-core
Infection rate from male to female	β_1	0.12	0.012
Infection rate from female to male	β_2	0.08	0.008
Recover rate for male	γ_1	0.02	0.02
Recover rate for female	γ_2	0.008	0.008
Relationship formation rate	δ	0.4	0.04
Relationship dissolution rate	α	1.6	0.16

Table 4.15: Result from Model 1 using parameters in Table 4.14

	Result	
Quantity of interest	core	non-core
R_0	1.1165	0.7248
Probability of extinction (π_1, π_2)	(0.3600 , 0.2712)	1
Endemic level	0.5612	0

The results show that the epidemic also persists in the core group in this case. This agrees with many studies the literature that a small core group can be very important in the spread of gonorrhoea causing gonorrhoea to remain endemic (Hethcote & Yorke (1984)).

Case study 3: In this study, we focus on the case where individuals in the population are unfaithful. Namely, individuals seek to engage with casual sex outside

relationships. Sexual contacts outside relationships are often termed as casual sex (Garcia et al. (2002)). There have been several studies concerning high-risk sexual behaviour including casual sex (Bersamin et al. (2012); Sonnenberg et al. (2015); Castor et al. (2002)). Here, we based our case study upon Kretzschmar et al. (1996) in which the authors focus attention on a highly sexually active core group where the unfaithfulness mostly occurs. Our objective is not to specifically compare our results with the primary literature, but to investigate whether or not the spread of gonorrhoea could similarly be described by our model based upon the empirical data and parameter estimates provided in the literature. The data was drawn from a national survey in the Netherlands of the sexual behaviour of Dutch adults (Zessen & Sandfort (1991)). The relationships were stratified into 2 types, steady and casual relationships. Individuals were either involved in a steady or a casual relationship or both. Table 4.16 shows the number of respondents (based on 926 respondents who reported only heterosexual relationships) that had no sex, only a steady relationship, only casual relationships, or both in the last year.

Table 4.16: Relational status in the past year for survey respondents

	Survey	
Relational status	Men	Women
No sex	129	11
Steady	636	737
Both	53	35
Casual	107	54

The author estimated their transmission and recovery rates for both steady and casual relationships based upon the transmission probability and the infectious period provided in the classical literature for gonorrhoea (Hethcote & Yorke (1984)). Table 4.17 summaries their parameter values in which they correspond to our model parameters as presented in the last column.

Table 4.17: Disease-specific parameter values (/day)

Parameter	value for gonorrhoea	model parameter
Transmission rate from male to female, steady relationships	0.15	β_1
Transmission rate from female to male, steady relationships	0.0625	β_2
Transmission rate from male to female, casual relationships	0.6	ω_1
Transmission rate from female to male, casual relationships	0.25	ω_2
Recovery rate for men	0.04	γ_1
Recovery rate for women	0.03	γ_2
Separation of a relationship	0.0004	δ
Formation of a partnership	0.006	α

Table 4.17 has given us most of the parameters needed in our model. Note that, α and δ given in the table were based on the length of steady relationships. In order to proceed further, we also need to specify the probability that somebody in a relationship agrees to have a one-night stand (r). According to Table 4.16, we can see that 53 of men and 35 of women are in both steady and casual relationships, respectively. This shows the proportion of individuals in relationships that would agree to casual contacts. Note that, the probability r is not differentiated according to gender. Therefore, we will take their average which is 44. According to 926 individuals, we have $r = 0.0457$. Hence, we choose $r = 0.0457$. The results are obtained as follows : $R_0 = 1.7341$ and the endemic level is 46 which is the number of infected cases in the population. The number of infected cases in Kretzschmar et al. (1996) based on gonorrhoea for the case with no intervention or prevention for 10,000 individuals is 74 people. We can see that the results obtained from our model have a similar number of infected cases to the survey.

We now turn our attention to the control measure. We are also interested in seeing how a control measure (v_1, v_2) reduces the prevalence of gonorrhoea. Note that, we will not assess the cost-effectiveness. Therefore, we will choose the most effective strategy in which $v_1 = 1$ and $v_2 = 1$ are chosen. We consider the above

3 case studies, the results are illustrated in Table 4.18, showing the proportion infected. Clearly the prevalence of gonorrhoea is reduced and it is most effective on Case study 1 such that the disease completely dies out.

Table 4.18: Intervention results

Case study	Endemic level (No control)	Endemic level (control)
1	0.5059	0
2	0.5612	0.4488
3	0.4641	0.3905

We have noticed that v_1 and v_2 have the most effect on Case study 1. This could be explained in terms of the parameter values. As we have studied in Section 4.4.1, the effectiveness of the control measure increases in α and β since the control measure helps recovery only within relationships. We can see in Case study 1 that $\alpha = 60.80$ and $\beta = 32.70$ relative to $\gamma = 3.33$ are large. Wherease, in Case study 2 and 3, β and α relative to γ are not that high. Therefore, this makes sense that the control measure has the most effect on Case study 1.

Moreover, since the control measure manages to completely control the disease for Case study 1, it is worthwhile to look at what value of v would be optimal. We have done this by increasing the value of v_1 and v_2 from 0 to 1, where $v_1 = v_2$. The results are plotted in Figure 4.9.

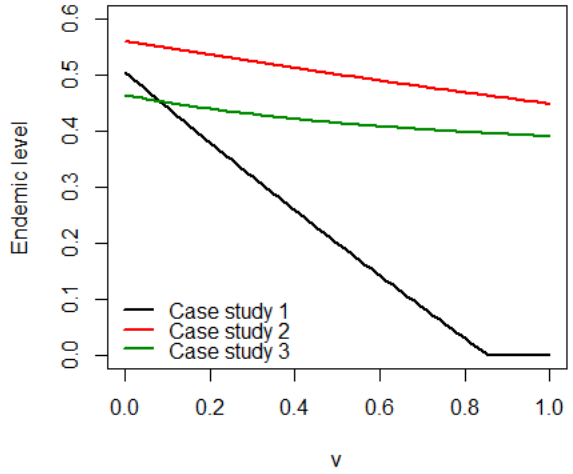


Figure 4.9: Plot of the endemic level against v for Case study 1, Case study 2, and Case study 3.

According to Figure 4.9, we can see that the endemic level goes to 0 at v around 0.85. Therefore, choosing $v > 0.85$ does not confer any additional benefits. For Case study 2 and Case study 3, the graphs are close to linear with a small concavity in Case study 3. In all cases the endemic level is monotonically decreasing with v. Therefore in Case studies 2 and 3 we should look to try and make v as large as possible but also look to control the transmission via one-night stands.

Chapter 5

Conclusions

5.1 Recap of Thesis

In this thesis, we propose two mathematical models describing dynamics of STDs, one of which incorporates one-night stands. We focus our studies on both early behaviour and long-term behaviour of the STD. A branching process approximation is employed to approximate the early stages of epidemics leading us to the results of the basic reproduction number (R_0) and the probability of extinction. The long-term behaviour concerning the endemic level, fluctuations about the endemic level and the mean time to extinction are also investigated. Those two models are amendable to control strategies. We suggest a medication use based control measure to prevent the epidemic from causing a major outbreak.

In Chapter 2, we study the model of sexually transmitted diseases without one-night stands. We begin by establishing a branching process approximation for the early stages of the epidemic which leads us to a surprising result such that R_0 has no effect by swapping parameter values between male and female. If the proportion of relationships in the population is fixed ($\frac{\bar{\alpha}}{\delta}$ is fixed), the result shows that the probability of extinction is decreasing as δ increases. In terms of the long-term behaviour of the disease, we establish a threshold result such that if $R_0 \leq 1$, the endemic level is zero, and if $R_0 > 1$ the endemic level displays a positive value. As the endemic level increases, we observe that the time to extinction is

generally increasing. We explore fluctuations of the epidemic about the endemic level for different parameter sets whilst having the endemic level fixed. The results show that there is not an obvious relationship between the variance and the mean time to extinction. When δ and α increase to infinity, we achieve shorter time to extinction as compared to other parameter sets, even though we consider the same endemic level. Additionally. the variance is smaller which tells us that there are smaller fluctuations about the endemic level for large δ and α.

In Chapter 3, we extend the sexual model from Chapter 2. New parameters regarding one-night stands are introduced. The model in Chapter 2 is a special case of this model when the model parameters representing one-night stands are 0. A 5-type branching process is established. In the simplest case where the rate parameters of male and female are identical, i.e. $\beta_1 = \beta_2$, $\gamma_1 = \gamma_2, \omega_1 = \omega_2$, the 5-type branching process can be reduced to 3 types. Quantities of interest are also explored through similar approaches in Chapter 2. In the case where we set $\omega_1 = \omega_2 = 0$, Model 2 becomes a model without one-night stands, linking back to Model 1. The basic reproduction number obtained from Model 1 (R_0^1) and the basic reproduction number obtained from Model 2 (R_0^2) will have different values but still play the same role as threshold parameters. In this chapter, we concentrate on numerical analysis due to the complexity of mathematical formulae. Therefore, most results concerning epidemic behaviours are obtained through numerical studies using Matlab and R programming language.

The proposed models give answer to the questions we are interested in. They are also amenable to mathematical and numerical analysis. In Chapter 4, a control measure is suggested for both models by introducing a new parameter governing the medication use (v). We found that R_0 decreases when v increases. In addition, we also illustrate some applications in this Chapter.

5.2 Suggested future work

Taking into account all the factors in the spread of STDs can make the model too difficult to analyse. Therefore, it remains extremely challenge to make the model more realistic while retaining the simplicity of the network model.

The proposed models showed reasonable results for general sexually transmitted diseases. However, more work can be done in order to obtain more realistic models. The following is our suggestions.

5.2.1 Extended compartment structure (SEIS and SEIRS)

The present model is an SIS epidemic model which can be extended to include compartment structures such as SEIS and SEIRS model. In the early stages of infection, infected individuals may not exhibit obvious sign of infection, and more importantly, the individuals are not yet able to transmit the disease as it develops within them. When an "exposed" stage is taken into account the model become SEIS model. This is interesting because whilst individuals are in an exposed stage, they have no ability to infect other individuals for some period of time until they become infectious. This might affect the incidence of the disease. Another interesting model is when temporary immunity is taken into account, and after the immunity wears off, the individuals become susceptible again (SEIRS).

5.2.2 Non-exponentially distribution for infectious period

In our model we assume that the infectious period follows an exponential distributed, which implies that the rate of recovery within a given time interval is constant. This is unrealistic in an epidemiological context. Other infectious period distributions are more realistic. Non-exponential distributed infection periods have already been studied in the literature for simple epidemic models (see, Keeling & Grenfell (1988) and Feng et al. (2007)). Particularly, Vergu et al. (2010)

explores the spread of epidemic in the context of metapopulation model when the infection period is chosen to be either constant or gamma distributed. It would be interesting to explore how this applies in sexually transmitted disease models, especially, how the gamma-distributed infectious periods make an impact on the epidemic dynamics. According to Krylova & Earn (2013), a gamma distribution with integer shape parameter is strongly preferred as it is equivalent to a sequence of independent and identically distributed exponential distributions. This can be done using the method of stages as follows. If the infectious period is $Gamma(k, l)$, then the infectious period is the sum of k exponential random variables with mean $1/l$. We then let an individual go through the stages of the infectious period with an exponential stay in each. This is useful because the model is still Markovian.

5.2.3 Age groups

Another interesting factor influencing the dynamics of sexual behaviour is the age ranges of individuals. We can classify individuals in terms of teenagers and adults, where teenagers have higher contact rates. To deal with this assumption, for our model where individuals are classified according to sex, relationship and disease status, we can include the status of individuals being teenagers or adults. Individuals are more likely to form relationships with individuals of a similar age. We can also divide the population into two groups: adults and teenagers. Individuals are allowed to make both local and global contacts, in other words, individuals can be mixed within groups (teenagers contact with teenagers and adults contact with adults), and among groups (teenagers contact with adults). The heterogeneity in transition rate could be applied in our case because teenagers tend to have higher sexual contacts with teenagers and less with adults, and vice versa.

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