MATHEMATICAL MODEL AND ANALYSIS OF CHLAMYDIA AND GONORRHEA CO-INFECTION

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CERTIFICATION

This is to certify that this work **"MATHEMATICAL MODEL AND ANALYSIS OF CHILAMYDIA TRACHOMATIS AND GONORRHEA CO-INFECTION"** was carried out by Chukukere Eziaku Chinomso (20174079158), in partial fulfillment of for the award of degree of **Master in Science (MSc) in Mathematic** in the department of mathematics of the federal university of technology Owerri.

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DEDICATION

This work is dedicated to God Almighty.

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Contents

ABSTRACT

A model for Chlamydia trachomatis (CT) and gonorrhea co-dynamics is studied and analysed to assess the impact of targeted treatment for each of the diseases on their co-infections population. The model exhibits the dynamical feature of backward bifurcation when associated reproduction number is less than unity. The global asymptotic stability of the disease-free equilibrium was also analyzed. The numerical simulations show that the strategy which implements female chlamydia trachomatis treatment and male gonorrhea treatment is the most effective in combating co-infections Chlamydia trachomatis and gonorrhea.

Keywords: Chlamydia, Tracchomatis, Gonorrhoea, Stability, Optimal Control.

Chapter 1

Introduction

1.1 Background information

Chlamydia trachomatis (CT) is one of the most common sexually transmitted diseases (STDs). Chlamydia trachomatis is transmitted through sexual contact with the penis, vagina, mouth or anus of an infected partner. According to Richmond *et al* (1972) some of the symptoms include burning sensation when urinating, abnormal vaginal discharge (for females) and pain and swelling in one or both testicles (for males). Just like Chlamydia trachomatis, gonorrhea is a very common STD, with symptoms similar to those of Chlamydia trachomatis, including white, yellow or green discharge from the penis (for males) and increased vaginal bleeding between menstrual periods.

The World Health Organization (WHO) reported that an estimated 127 million people and another 87 million people worldwide are infected with Chlamydia trachomatis and gonorrhea, respectively WHO (2019).Chlamydia trachomatis and gonorrhea infections can lead to complications, such as cervicitis and pelvic inflammatory disease (PID) WHO(2019). Clinical Reports also indicate that there is an increased progression to the development of PID by individuals co-infected with Chlamydia trachomatis and gonorrhea . According to Oriel *et al* (1978), double infections with *gonococci* and *chlamydiae* are very common, especially among women receiving treatment for venereal diseases. It has been pointed out that gonorrhea may reactivate latent chlamydial infections Richmond *et al* (1972). Results have also revealed that gonorrhea was transmitted more often than chlamydial infection, from infected women to male sexual partners Lycke *et al* (1980). According to a clinical report, re-infection is common after patients who have recovered from Chlamydia trachomatis and gonorrhea CDC (2016). In the study, Van Rooijen

et al (2015) reported that "However, over 50 percent of women with gonorrhea and 75 percent of women with Chlamydia trachomatis remain asymptomatic during the entire infection process" thereby increasing their chances of spreading the infectious to un-infected sexual partners. Tadele *et al* (2019) reported that Chlamydia and gonorrhea co-infection was more prevalent among female commercial sex workers, especially who do not use condoms than those who use condoms consistently. Recently, Dela *et al* (2019) in a cohort study in Ghana, pointed multiple sexual partners and alcohol consumption as part of the risk factors for Chlamydia trachomatis and gonorrhea co-infections. In a population based study for Chlamydia screening programme in the Netherlands, van Bergen *et al* (2006) reported that, to reduce the burden of the co-infections of Chlamydia trachomatis and gonorrhea, individuals should also be tested for gonorrhea even if only Chlamydia trachomatis infection is found. Rose *et al* (2017) pointed out in another research, that in order to reduce re-infection with Chlamydia and gonorrhea, efforts must be enhanced to increase repeated testing.

Mathematical models have become important tools in studying the behaviour of infectious diseases Nwankwo and Okuonghae (2019),Okuonghae (2019),Omame *et al* (2014) ,Omame *et al*(2020) ,Tanvi and Aggarwal (2020), Omame *et al* (2015), Omame *et al* (2020), Umana *et al* (2016), Uwakwe *et al* (2020). Mathematical models have also been developed to understand the dynamics of Chlamydia trachomatis, gonorrhea or their co-infections Sharomi and Gumel (2009), Sharomi and Gumel (2011), Mushayabasa (2012). For instance Mushayabasa (2012) considered a co-infection model for Chlamydia trachomatis and gonorrhea in a female-only population setting, to examine the impact on complications arising from their co-infections, such as pelvic inflammatory disease. he showed that whenever both diseases are endemic in a population, the cumulative pelvic inflammatory disease cases tend to increase. Sharomi and Gumel (2009) analyzed and designed a two-sex deterministic model for Chlamydia trachomatis. The model exhibited the phenomenon of backward bifurcation, caused by the re-infection of individuals who recovered from the disease.

Chlamydia trachomatis is caused by a bacteria called chlamydia trachomatis is a gram-negative bacteria, that can only replicate within the host cell. Chlamydia is known as a 'Silient infection' because most infected people are asymptomatic and lack abnormal physical examination findings (CDC 2016).According to Holmes (2008)The primary site of infection for bacteria is the columnar epithelium of the endocervix in women and urethra in men, both also infect the rectum, pharynx

and conjuctiva and can be transmitted from the infected mother during pregnancy or delivery also Zarker *et al* (2014) says untreated Chlamydia infection can result in pelvic inflammatory disease (PID) in women, which can lead to ectopic pregnancy and tubal factor infertility and chlamydia can cause epididymitis in men. Chlamydia trachomatis is transmitted through sexual contact with the penis, vagina, mouth or anus of an infected partner. Ejaculation does not have to occur for chlamydia to be transmitted or acquired. The infection can also be spread prenatally from untreated mother to her baby during vaginal child birth, resulting in conjunctivitis or pneumonia in some exposed infant. according to Bell *et al* (1992), rectal or genital chlamydia trachomatis infection has been shown to persist longer in infants infected at birth.

Eaton *et al* (2011) Sexually active young people are at risk of contacting chlamydia trachomatis infection for a combination of behavioural, biololgical and cultural reasons. some young people do not use condom frequently, Torrone *et al* (2012) estimated that 1 in 20 sexually active young women aged 14-24 years has chlamydia.However, chlamydia infection is asymptomatic in most people. But in symptomatic patient the infection usually appears between 1 and 3 weeks after having unprotected sex with an infected person. Wiesenfeld *et al* (2005) mentioned common symptoms in women which includes mucopurulent endocervical discharge, easily induced endocervical bleeding , pyuria, dysuria, urinary frequency, pelvic pain and conjunctivitis inflammation (redness) of the eyes this is caused by an infected semen or vaginal fluid getting into the eyes. Berger *et al* (1978) reported that in men the common symptoms includes urethritis, watery urethral discharge and dysuria. A minority of infected men develop epididymitis (with or without symptomatic urethritis), unilateral testicular pain, tenderness and swelling. Jones *et al* (1985) chlamydia trachomatis can also be found in the throats of women and men having sex with an infected partner.

Chlamydia trachomatis infection causes complications most commonly resulting from spread of the lower to upper genital tract. Upper genital tract infections occurs in both sexes but is more common and has more servere consequences in women, Stamm *et al* (2008) In women , chlamydia trachomatis ascends to the upper genital tract in approximately 10 percent of cases cause symptomatic pelvic inflammatory disease (PID),Oakeshott *et al* (2010),Herzog *et al* (2012). according to Paavonen *et al* (2008) Chlamydia trachomatis results to tubal damage which can cause ectopic pregnancy, tubal infertility and chronic pelvic pain.Oakeshott *et al* (1995) Salpingitis which means the inflammation of the fallopian tubes, fallopian tubes carry eggs from the

ovary to the uterus salpingits prevent an egg from travelling from the ovary to the womb hence this can lead to infertility. Bartholintis means swollen bartholin gland, bartholin gland produce a woman lubricating mucus. chlamydia can cause this gland to become blocked and infected leading to cyst that can become infected and develop into abscess and some of the complications in men includes epididymitis infection of the tubes that carries sperms to the testicles this can result in fever scrotal pain swelling and urethritis. Prostatitis means infection of the prostrate gland this can result in pain during or affter sex, fever and chills, painful urination and lower back pain. As many as half of all infants (Neonate 0-28 days) born to mothers with chlamydia trachomatis will be born with the disease, it can affect infants by causing pre-mature birth and conjunctivitis this may lead to blindness. Conjunctivitis due to chlamydia occurs one week after birth, the infant will have symptoms like rednedd of the eyes, eye discharge and fever. Anyone who has been recently diagnosed with chlamydia trachomatis should see a health care provider for evaluation because chlamydia trachomatis is usually asymptomatic. Centers for Disease controls, (CDC 2016) recommends yearly chlamydia screening for all sexually active women younger than 25 years and older women with risk factor such as new or multiple partners or sex partner who has sexually transmitted infection. There are number of diagnostic test for chlamydia trachomatis including first void urine microscopy culture and sensitivity , high vagina microscopy culture and sensitivity, nasopharyngear swap microscopy culture and sensitivity this is for infants with pneunomia, seminal fluid microscopy culture and sensitivity, vaginal swab for women and conjunctival swab. Manhart *et al* (2013) Chlamydia trachomatis can be treated with tetracyclines (usually doxycycline) or marcrolide (usually azithromycin) antibiotic with short-term microbiological cure rates 90 percent to 95 percent. Persons with chlamydia trachomatis should abstain from sexual activity for 7 days after single does antibiotics or untill completion of a 7 days course of antibiotics to prevent spreading the infections to partners and it is very important to take all medication prescribed to cure chlamydia trachomatis. Chlamydia trachomatis can be avoided if we abstain from vaginal, anal and oral sex or using dental dam during sex.According to Holmes *et al* (2008) use of latex male condoms consistently and correctly can reduce the risk of getting chlamydia trachomatis.

Gonorrhea is a sexually transmitted disease (STD) caused by the bacterium Neisseria gonorrhoeae that multiples the reproductive tract, which includes the cevix, uterus, fallopian tubes and urethra. it is sometimes referred to as "The clap" untreated gonorrhea can lead to severe

complications in both women and men. In women, Untreated gonorrhea can lead to pelvic inflammatory disease, which is the infection of the uterus, fallopian tubes and other reproductive organs. Pelvic inflammatory disease can cause infertility, ectopic pregnancy, abscess formation, and chronic pelvic pain. In menn, untreated gonorrhea can lead to epididymitis, a condition of the ducts attached to the testicles, which can cause infertility in both men and women, it is possible for untreated gonorrhea to spread to the blood or joints and/or increase the contraction of HIV (Human Immunodeficiency Virus). Gonorrhea can be contacted through unprotected sexual intercourse with someone who has gonorrhea, even if they do not have symptoms. gonorrhea can also be contacted by sharing sex toys with an infected person and genital contact with an infected person this means one can get gonorrhea by coming in contact with infected semen or vagina fluid even if there is no penetration, orgasm or ejaculation. A pregnant woman with gonorrhea can pass the infection unto her baby during child birth. Anyone who is sexually active can contact gonorrhea if you have multiple sexual partners who has a sexually transmitted disease (gonorrhea), inconsistent use of condom and previous history of gonorrhea infections. Symptoms usually occur within 2-14 days after exposure to gonorrhea infection however some people infected with gonorrhea never develop noticeable symptoms. A person is more likely to spread the infection to other partners when they do not have noticeable symptoms. Furthermore, symptoms in women includes vagina discharge which can be watery, creamy or slightly greeen, pain or burning sensation while urinating, pain during sexual intercourse, sharp lower abdominal pain, sore-throat, heavier periods or spotting and fever while symptoms in men includes burning or painful sensation during urination, urinary frequency or urgency, pus-like discharge from the penis which may be whitish, yellowish or greenish in colour, swelling or redness in the opening of the penis and persistent sore-throat. Untreated gonorrhea can cause serious and permanent health problems in both men and women. some of the complications in women includes spread into uterus, fallopian tube, hence causing pelvic inflammatory disease which may result in scarring the tubes. Greater risk of ectopic pregnancy and infertility. while men with untreated gonorrhea can experience epididymitis increased risk of HIV/AIDS. Babies who contact gonorrhea from their mothers during vagina child birth can develop conjunctivitis, redness and eye discharge if not properly treated can lead to blindness, Testing for gonorrhea is done by swabbing the infected site (rectum, throat, cervix) and identifying the bacteria the laboratory either through culturing the material from the swab.Gonorrhea can be cured with the right antibiotics such as Ceftriaxone, Azithromycin and

Doxycycline it is important you take all medications your dictor prescribes to cure your infection and medication should not be shared with anyone although medication will stop the infection, it will not undo any permanent damage caused by the disease. The safest way to prevent gonorrhea is through abstinence. Is important to abstain from sexual activity during period of medications and use condom during any type of contact including vaginal sex, anal sex or oral sex.

In this study, we shall be investigating the impact of female only treatment and male only treatment on the co-dynamics of Chlamydia trachomatis and gonorrhea.

1.2 Problem statement

Chlamydia and Gonorrhoea co-infection is among the common bacterial sexually transmitted infections worldwide. Currently,the high percentages of asymptomatically infected individuals cause a problem for public health. Estimates indicate that every year, millions of people are been infected with either chlamydia or gonorrhoea infection and in some cases both and if untreated can cause complications later such as epididymitis in men and pelvic inflammatory disease (PID),which may lead infertility and ectopic pregnancy in women.

1.3 Aim and Objectives of Study

The general aim of this study is to develop deterministic mathematical models for Chlmydia trachomatis and gonorrhoea co-infection that will provide solution to frequently aksed questions.

The Specific objectives of the study are:

i To formulate the basic properties of the developed models.

ii Determine the equilibrium points of the formulated models.

iii To solve numerical simulations & global sensitivity analysis of the models, using appropriate data.

iv Detemine the numerical solutions using appropriate data .

v Obtain Qualitative Analysis of the model and the positive of solution

1.4 Justification of Study

Millions of people worldwide are been diagnosed of pelvic inflammatory disease in women and epididymitis in men as result of untreated Chlamydia trachomatis or Gonorrhea infection in some cases both infections. However it is relevant to develop mathematical models that can help eliminate or reduce the spread of this sexually transmitted infection (STI) and also evaluate the results of chlamydia trachomatis symptoms,diagnosis,condom,treatment,partner notification and co-infection with gonorrhea. This will help to control and manage chlamydia trachomatis and gonorrhea co-infection.Our model is concentrated on reducing Chlamydia trachomatis and Gonorrhea in high risk population.

1.5 Scope of Study

mathematical model will be use to study the impact of treatment and partner notification on the transmission of chalmydia trachomatis or gonorrhea infection. we also consider a case where individuals with co-infection of chamydia trachomatis and gonorrhea in population where dually infected. That is individuals with chlamydia trachomatis infection can infect an individual with gonorrhea infection and vice-versa. while in the co-infection model we presume that transmission of chlamydia trachomatis or gonorrhea occurs between female and male individuals. We want to examine a case where chlamydia trachomatis and gonorrhea infection can be reduced and treatment is always readily available.

Chapter 2

Literature Review

2.1 Conceptual Literature

Chlamydia trachmatis and Nesisseria Gonorrhea co-infection is a common sexually transmitted infection. In most cases, the proportion of people with Neisseria gonorrhea who have Chlamydia treachomatis co-infection is higher than the proportion of people with Chlamydia Trachomatis who have Nesisseria gonorrhea Nsuami *et al.* (2004), most likely reflecting higher chlamydia trachomatis prevalence.

Factors associated with chlamydia trachomatis and Neisseria gonorrhea co-infection vary across studies or populations and include sex, age, sex work, drug and alcohol use Rose *et al.* (2017), Trecker *et al.* (2017). MSM (men sleeping with men) have been reported as more likely to harbor co-infections than heterosexual males, with the co-infection rate increasing with age in MSM (men sleeping with men), but decreasing with age in heterosexual men. Zhang and Van Der Veen (2019), while others have found similar co-infection rates for MSM and heterosexual men Khaw *et al.* (2012). However, men sleeping with men have higher overall infection rates than heterosexual men, which may impact such analyses. Co-infection in most cases, have been linked with increased risk of re-infections. Co-infection increases risk of (i) re-infection with chlamydia trachomatis or Neisseria gonorrhea when retested between 6 weeks and 6 months Rose *et al.* (2017), (ii) Neisseria gonorrhea re-infection in a high Neisseria gonorrhea prevalence population Trecker *et al.* (2017), and (iii) chlamydia trachomatis re-infection in women Hillis *et al.* (1994). However, a study that collected daily samples for 28 days after antibiotic treatment, evaluating 23 patients with Neisseria gonorrhea and chlamydia trachomatis co-infection for chlamydia trachomatis clearance,

noted 100 percent clearance with no re-infections Wind *et al.* (2016).

2.2 Related Literature

many mathematical models have been developed to understand the dynamics of co-infection of two or more diseases (Hussaini *et al.,* 2016; Sharomi *et al.,* 2008; Naresh and Tripathi, 2005; West and Thompson JR, 1996; Roeger *et al.,* 2009; Bhunu *et al.,* 2009; Bhunu and Mushayabasa 2013; Okuneye *et al.,* 2017; Mtisi *et al.* 2009; Mukandavire *et al.* 2009; Mushayabasa *et al.,* 2011). considered a mathematical model for the transmission dynamics of HIV/TB co-infection when there is treatment. They observed that the HIV-only treatment strategy could reduce mixed infection cases than the TB-only treatment strategy. Roeger *et al.* (2009) modelled TB and HIV co-infection. They observed that high progression rates from HIV to AIDS may increase the prevalence of HIV. Also, Naresh and Tripathi (2005) studied the dynamics of HIV and TB co-infection. They showed that if the HIV infection is minimized at an early stage through the use of drugs, the progression to AIDS stage could be significantly reduced. The dynamics of HIV/AIDS and TB in the presence of treatment was studied by Bhunu *et al.* (2009). They reported that AIDS-only treatment strategy could bring down the number of latent individuals moving to active TB stage, and that treatment of latent and active TB cases could equally reduce movement to AIDS stage by individuals infected with HIV. Mukandavire *et al.* (2009) formulated and analyzed a co-infection model for HIV and Malaria. They showed that the reduction in sexual activity of individuals with malaria symptoms decreases the number of new cases of HIV and the mixed HIV-malaria infection. Mtisi *et al.* (2009) rigorously analyzed a mathematical model for the co-dynamics of tuberculosis and malaria. They simulated the model to investigate the co-existence of the two diseases when the associated reproduction numbers of the two diseases are greater than unity and equally carried out sensitivity analysis on the main parameters that drive the dynamics of the diseases. Mushayabasa *et al* (2011) designed a mathematical model to explore the co-interaction of gonorrhea and HIV in the presence of antiretroviral therapy and gonorrhea treatment. They showed that gonorrhea only treatment strategy is highly significant in reducing the co-infection new cases. In another paper, Hussaini *et al* (2016) developed a mathematical model for the transmission dynamics of HIV and Anthroponetic Visceral Leishmaniasis (AVL) co-infection in a population. They showed that the two diseases co-exist, with AVL dominating

HIV whenever the reproduction number of each disease is greater than unity. Recently, Nwankwo and Okuonghae (2019) studied the transmission dynamics of HIV Syphilis co-infection when there is treatment for Syphilis. They showed that high treatment rates for primary syphilis (in both singly and dually infected individuals) will result in a significant decrease in the incidence of co-infection of the two diseases in the population.

2.3 Specific Literature

Anderson and May (1991), carried out mathematical model using various treatment and intervention strategies aimed at gonorrhea, chlamydia trachomatis or both, during the cause of the simulation.The act to reduce the duration of infection in those infected is to simulate an increased rate of diagnosis and treatment.The influence of patterns of sexual partner choice and sexual mixing on the impact of treatment was also assessed. Khaw *et al.*(2012) Modelling studies describe the transmission of chlamydia trachomatis using different structures and levels of complexity which have been applied to inform and guide public health decision about screening programmes and models have been used to investigate aspect of immunity.Omame *et al.* (2018) studied a two-sex deterministic model for Human Papillomavirus (HPV) that assesses the impact of treatment and vaccination on its transmission dynamics and the numerical simulations of the model showed that 75 percent efficacy for male population with about 40 percent condom compliance by females will result in a significant reduction in the disease burden in the population. Omame *et al.* (2018) also showed that 70 percent condom compliance by males and adminstering female vaccine (with 45 percent efficacy) is sufficient for effective control of the disease. Gottlie *et al.*(2013) considered infectious diseases like Chlamydia trachomatis were mathematical models are often used to examine transmission dynamics, synthesize knowledge about natural history and diseases outcomes to predict future events and quantify the potential impact of interventions.such analyses is to inform policy decisions, especially where there are limited empirical data available for comparative evaluation of different interventions. Wind *et al.,*(2016) considered mathematical models that dynamically incorporate the progression to infertility related complications of chlamydia trachomatis are needed to help understand the impact of screening programs on primary outcomes.Generally, the longer a person is infected, the more likely it is that they will be reached by a screening programe and will receive treatment. chlamydia trachomatis infection is

indeed characterised by a long asymptomatic period but the duration of this period is not known. In addition, it is unclear what fraction of infections will cause symptoms that prompt treatment seeking behaviour or whether natural clearance is followed by a period of temporary immunity, Brunham *et al* (2005). discussed the chlamydia trachomatis epidermic model with two treatment controls. An objective functional is considered which is based on a combination of minimizing the number of exposed and infective individuals and the cost of treatment.Then an optimal control pair is obtained which minimizes the objective functional. Numerical findings are illustrated through computer simulations using MATLAB,which show the reliability of the model from the pratical point of view.

The purpose of the current study is to extend some of the aforementioned studies

Chapter 3

Methodology

This chapter introduces some of the key mathematical theories and methodologies relevant to the dissertation.

Definition 3.1. *Consider the system:*

$$\begin{aligned} \dot{x} &= f(x), \quad x \in \mathbb{R}^n, \\ \dot{y} &= g(y), \quad y \in \mathbb{R}^n, \end{aligned} \tag{3.1}$$

where f and g are two $C^r(r \geq 1)$ ODEs defined on $\mathbb{R}^n$. The dynamics generated by the vector field f and g of (3.1) are said to be locally C^k conjugate $(k \leq r)$ if there exists a C^k diffeomorphism h which takes the orbits of the flow generated by f, $\phi(x,t)$, to the orbits of the flow generated by g, $\psi(t,y)$ preserving orientation and parametrization by time.

Theorem 3.1 (Hartman and Grobman (Wiggins, 1983))**.** *Consider a $C^r(r \geq 1)$ vector field f and the system*

$$\dot{x} = f(x), \quad x \in \mathbb{R}^n, \tag{3.2}$$

with domain of f an open subset of $\mathbb{R}^n$. Suppose also that (3.2) has equilibrium solutions which are hyperbolic. Consider the associated linear ODE system

$$\dot{\xi} = Df(\bar{x})\xi, \quad \xi \in \mathbb{R}^n. \tag{3.3}$$

Then the flow generated by (3.2) is C^0 conjugate to the flow generated by the linearized system (3.3) in a neighborhood of the equilibrium point.

The Hartman-Grobman guarantees that an orbit structure near a hyperbolic equilibrium solution is topologically-equivalent to the orbit structure given by the associated linearized dynamical system.

3.1 Derivation of basic reproduction number

The next generation operator method (van den Driessche and Watmough, 2002) is used to establish the local asymptotic stability (LAS) of the disease-free equilibrium (DFE) of a disease transmission model. Suppose the given disease transmission model, with non-negative initial conditions, can be written in terms of the following system:

$$\dot{x}_i = f(x) = \mathcal{F}_i(x) - \mathcal{V}_i(x), i = 1, ..., n, \tag{3.4}$$

where $\mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$ and the functions satisfy Axioms (B1)-(B5) below.

The function $\mathcal{F}_i(x)$ represents the rate of appearance of new infections in compartment i. The function $\mathcal{V}_i^+(x)$ represents the rate of transfer of individuals into compartment i, $\mathcal{V}_i^-(x)$ represents the rate of transfer of individuals out of compartment i. Furthermore, the number of individuals in each compartment is given by $x = (x_1, ..., x_n)^t, x_i \geq 0$, and $X_s = \{x \geq 0 | x_i = 0, i = 1, ..., m\}$ is defined as the disease-free states (non-infected variables of the model).

B1 If $x \geq 0$, then $\mathcal{F}_i, \mathcal{V}_i^-, \mathcal{F}_i^+ \geq 0$ for $i = 1, ..., m$;

B2 if $x_i = 0$, then $\mathcal{V}_i^- = 0$. In particular, if $x \in X_s$ then $\mathcal{V}_i^- = 0$ for $i = 1, ..., m$;

B3 $\mathcal{F}_i = 0$ if $i > m$;

B4 if $x \in X_s$ then $\mathcal{F}_i(x) = 0$ and $\mathcal{V}_i^+ = 0$ for $i = 1, ..., m$;

B5 if $F(x)$ is set to zero, then all eigenvalues of $Df(x_0)$ have negative real parts

Lemma 3.2. *(van den Driessche and Watmough, 2002). If $\bar{x}$ is a DFE of (3.4) and $\mathcal{F}_i(x)$ satisfy (B1)-(B5), then the derivatives $D\mathcal{F}(\bar{x})$ and $D\mathcal{V}(\bar{x})$ are partitioned as*

$$D\mathcal{F}(\bar{x}) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}, \quad D\mathcal{V}(\bar{x}) = \begin{bmatrix} V & 0 \\ J_3 & J_4 \end{bmatrix}$$

where F and V are the $m \times m$ matrices defined by

$$F = \frac{\partial \mathcal{F}_i}{\partial x_j}(\bar{x}), \quad V = \frac{\partial \mathcal{V}_i}{\partial x_j}(\bar{x}), \quad with \quad 1 \leq i, j \leq m.$$

Further, F is a non-negative matrix, V is a non-singular M-matrix and J_3, J_4 are matrices associated with the transition terms of the model, and all eigenvalues of J_4 have positive real parts.

Theorem 3.3. *(van den Driessche and Watmough, 2002). Consider the disease transmission model given by* (3.4) *with $f(x)$ satisfying Axioms (B1)-(B5). If $\bar{x}$ is a DFE of the model, then $\bar{x}$ is locally asymptotically stable (LAS) if $\mathcal{R}_0 = \rho(FV^{-1}) < 1$ (where ρ is spectral radius), but unstable if $\mathcal{R}_0 > 1$.*

The following theorem is used to establish the presence of the backward bifurcation phenomenon for the models considered in this dissertation.

Theorem 3.4 (Castillo-Chavez & Song (2004))**.** *Consider the following system of ordinary differential equations with a parameter ϕ*

$$\frac{dx}{dt} = f(x, \phi), \quad f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R} \text{ and } f \in C^2(\mathbb{R}^n \times \mathbb{R}), \tag{3.5}$$

where 0 is an equilibrium point of the system (that is, $f(0, \phi) \equiv 0$ for all ϕ) and assume

B1: *$B = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$ is the linearization matrix of the system* (3.5) *around the equilibrium 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of B and other eigenvalues of B have negative real parts;*

B2: *Matrix B has a right eigenvector w and a left eigenvector v (each corresponding to the zero eigenvalue).*

Let f_k be the kth component of f and

$$b = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$
$$c = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0).$$

The local dynamics of the system around 0 is totally determined by the sign of b and c.

i $b > 0$, $c > 0$. *When* $\phi < 0$ *with* $|\phi| \ll 1$, *0 is locally asymptotically stable and there exists a positive unstable equilibrium; when* $0 \leq \phi \ll 1$, *0 is unstable and there exists a negative, locally asymptotically stable equilibrium;*

ii $b < 0$, $c < 0$. *When* $\phi < 0$ *with* $|\phi| \ll 1$, *0 is unstable; when* $0 < \phi \ll 1$, *0 is locally asymptotically stable equilibrium, and there exists a positive unstable equilibrium;*

iii $b > 0$, $c < 0$. *When* $\phi < 0$ *with* $|\phi| \ll 1$, *0 is unstable and there exists a locally asymptotically stable negative equilibrium; when* $0 \leq \phi \ll 1$, *0 is stable and a positive unstable equilibrium appears;*

iv $b < 0$, $c > 0$. *When* ϕ *changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.*

Particularly, if $b > 0$ *and* $c > 0$, *then a backward bifurcation occurs at* $\phi = 0$.

Theorem 3.5 (Descartes Rule of Signs, Wang(2004))**.** *Let* $p(x) = a_0 x^{b_0} + a_1 x^{b_1} + \cdots + a_n x^{b_n}$ *denote a polynomial with nonzero real coefficients* a_i *, where the* b_i *are integers satisfying* $0 \leq b_0 < b_1 < b_2 < \cdots < b_n$. *Then the number of positive real zeros of* $p(x)$ *(counted with multiplicities) is either equal to the number of variations in sign in the sequence* $a_0, ..., a_n$ *of the coefficients or less than that by an even whole number. The number of negative zeros of* $p(x)$ *(counted with multiplicities) is either equal to the number of variations in sign in the sequence of the coefficients of* $p(-x)$ *or less than that by an even whole number.*

3.2 Lyapunov Function Theory

Establishing the global properties of a dynamical system is generally not trivial and the direct Lyapunov method (Dushoff et al., 1998) is one of the most powerful methods. The method requires the construction of an auxiliary function with certain properties, that is a Lyapunov function.

Definition 3.2. *Consider the following system*

$$\dot{x} = f(x), \quad x \in \mathbb{R}^n \tag{3.6}$$

Let, $\bar{x}$ be an equilibrium solution of (3.6) *and let $V : U \to \mathbb{R}$ be a c^1 function defined on some neighborhood U of $\bar{x}$ such that*

i) *V is positive-definite,*

ii) *$\dot{V}(x) \leq 0$ in $U \backslash \{\bar{x}\}$.*

Any function, V, that satisfies the Conditions (i) and (ii) above is called a *Lyapunov function.*

Theorem 3.6 (La Salle's Invariance Principle (La Salle and Lefschetz, 1976))**.** *Consider the following system* (3.6)*. Let,*

$$S = \{x \in \bar{U} : \dot{V} = 0\} \tag{3.7}$$

and M be the largest invariant set of (3.6) *in S. If V is a Lyapunov function on U and $\gamma^+(x_0)$ is a bounded orbit of* (3.6) *which lies in S, then the $\omega -$ limit of set $\gamma^+(x_0)$ belongs to M; that is, $x(t, x_0) \to M$ as $t \to \infty$*

Corollary 3.1. *If $V(x) \to \infty$ as $|x| \to \infty$ and $\dot{V} \leq 0$ on $\mathbb{R}^n$, then every solution of* (3.6) *is bounded and approaches the largest invariant set M of* (3.6) *in the set where $\dot{V} = 0$. In particular, if $M = \{0\}$, then the solution $x = 0$ is globally-asymptotically stable (GAS)*

Construction of Lyapunov functions to prove the GAS of the disease free equilibrium (DFE)

Suppose that there are $n > 0$ disease compartments and $m > 0$ non-disease compartments. Then a general compartmental disease transmission model can be written as

$$\dot{x} = \mathcal{F}(x, y) - \mathcal{V}(x, y), \quad \dot{y} = g(x, y) \tag{3.8}$$

with $g = (g_1, g_2, ..., g_m)^T \in \mathbb{R}^m$, $x = (x_1, x_2, ..., x_n)^T \in \mathbb{R}^n$ and $y = (y_1, y_2, ..., y_m)^T \in \mathbb{R}^m$ represents the populations in disease compartments and non-disease compartments respectively; $\mathcal{F} = (\mathcal{F}_1, \mathcal{F}_2, ..., \mathcal{F}_n)^T$ and $\mathcal{V} = (\mathcal{V}_1, \mathcal{V}_2, ..., \mathcal{V}_n)^T$, where $\mathcal{F}_i$ represents the rate of appearance of new infections in the *ith* disease compartment, $\mathcal{V}_i$ represents the transitions in and out of the *ith* disease compartment, for example, death, recovery, etc. Following van den Driessche and Watmough (2002), define two $n \times n$ matrices

$$F = \frac{\partial \mathcal{F}_i}{\partial x_j}(\bar{x}), \quad V = \frac{\partial \mathcal{V}_i}{\partial x_j}(\bar{x}), \quad with \quad 1 \leq i, j \leq m. \tag{3.9}$$

it is assumed that $F \geq 0$ and $V \geq 0$. Set

$$f(x, y) = (F - V)x - \mathcal{F}(x, y) + \mathcal{V}(x, y) \tag{3.10}$$

Then (3.8) for the disease compartments can be written as

$$\dot{x} = (F - V)(x) - f(x, y) \tag{3.11}$$

Let $\omega^T \geq 0$ be the left eigenvector of the non-negative matrix $V^{-1}F$ corresponding to the eigenvalue $\mathcal{R}_0$. The following result provides a general method to construct a Lyapunov function to prove the GAS of DFE of the system (3.8).

Theorem 3.7 (Shuai and van den Driessche , 2013)**.** *Let F, V and $f(x, y)$ be defined as in* (3.9) *and* (3.10)*, respectively. If $f(x, y) \geq 0$ in $\Gamma \subset \mathbb{R}^{n+m}_+, F \geq 0, V^{-1} \geq 0$ and $\mathcal{R}_0 \leq 1$, then the function $Q = \omega^T V^{-1} x$ is a Lyapunov function for model* (3.8) *on* Γ

Construction of Lyapunov functions to prove the GAS of the endemic equilibrium point (EEP)

The non-linear Lyapunov functions used for studying the global properties of the endemic equilibria are of the Goh-Volterra type (Goh, 1976):

$$W_1(x_1, x_2, ..., x_n) = \sum_{i=1}^{n} c_i(x_i - x_i^{**} - x_i^{**} ln \frac{x_i}{x_i^{**}}). \tag{3.12}$$

Other types of Lyapunov functions that can be used include the quadratic Lyapunov functions (Vargas-De-Leon, 2011):

$$W_2(x_1, x_2, ..., x_n) = \sum_{i=1}^{n} \frac{c_i}{2}(x_i - x_i^{**})^2, \tag{3.13}$$

and composite-Volterra type (Vargas-De-Leon, 2011):

$$W_3(x_1, x_2, ..., x_n) = c\left[\sum_{i=1}^{n}(x_i - x_i^{**}) - \sum_{i=1}^{n} ln \frac{\sum_{i=1}^{n} x_i}{\sum_{i-1}^{n} x_i^{**}}\right] \tag{3.14}$$

3.3 Chlamydia Trachomatis and Gonorrhoea co-infection model

3.3.1 Model Assumption

The model was developed under the following specific assumptions

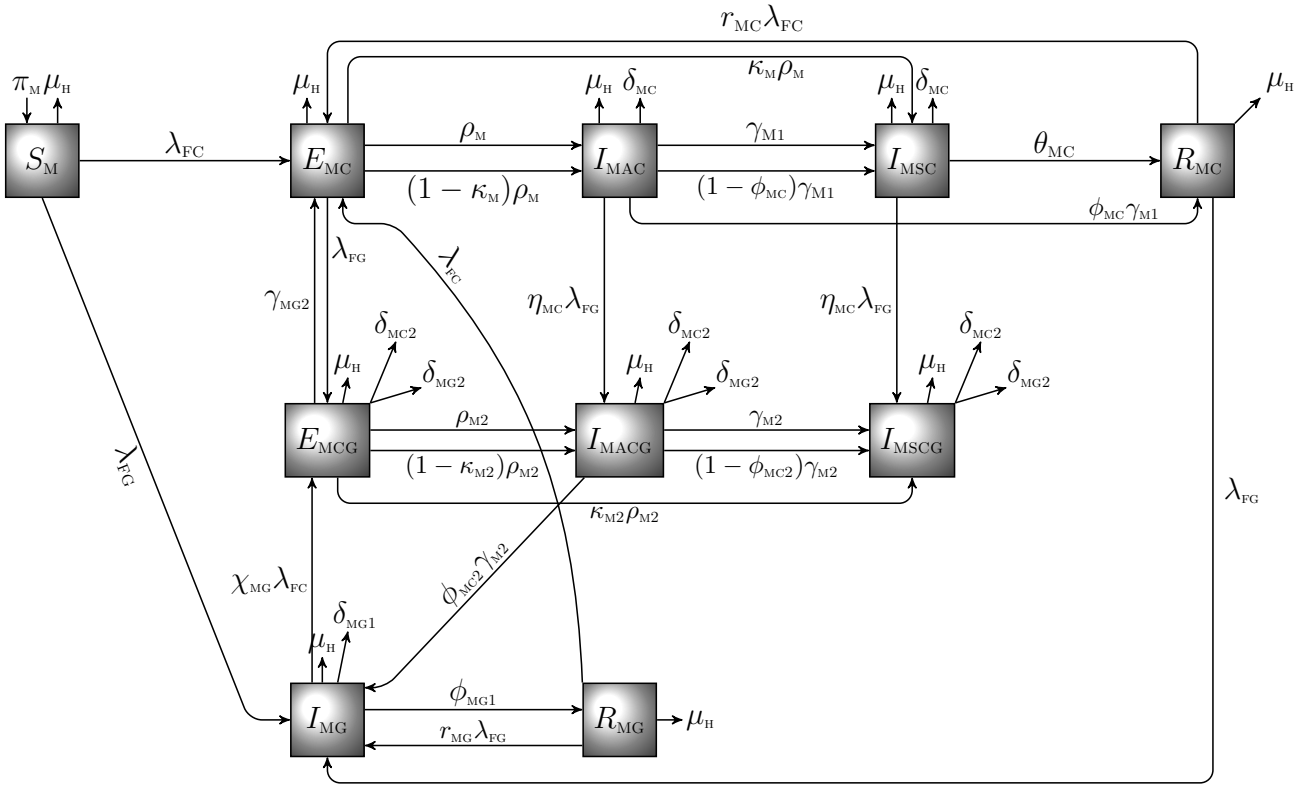
- The population is finite with a constant recruitment rate
- Members of the population interrelate freely not subject to any quarantine policy
- The population is recharged by birth only
- Recovered individuals becomes susceptible since recovery from chlamydia trachomatis or gonorrhoea co-infections does not confer parmanent immunity
- All members of the population suffer natural mortality at a uniform rate.
- individuals who are dually infected can transmit either Chlamydia trachomatis or Gonorrhea infection.
- modification parameter for increase in infectiousness of dually infected males and females.

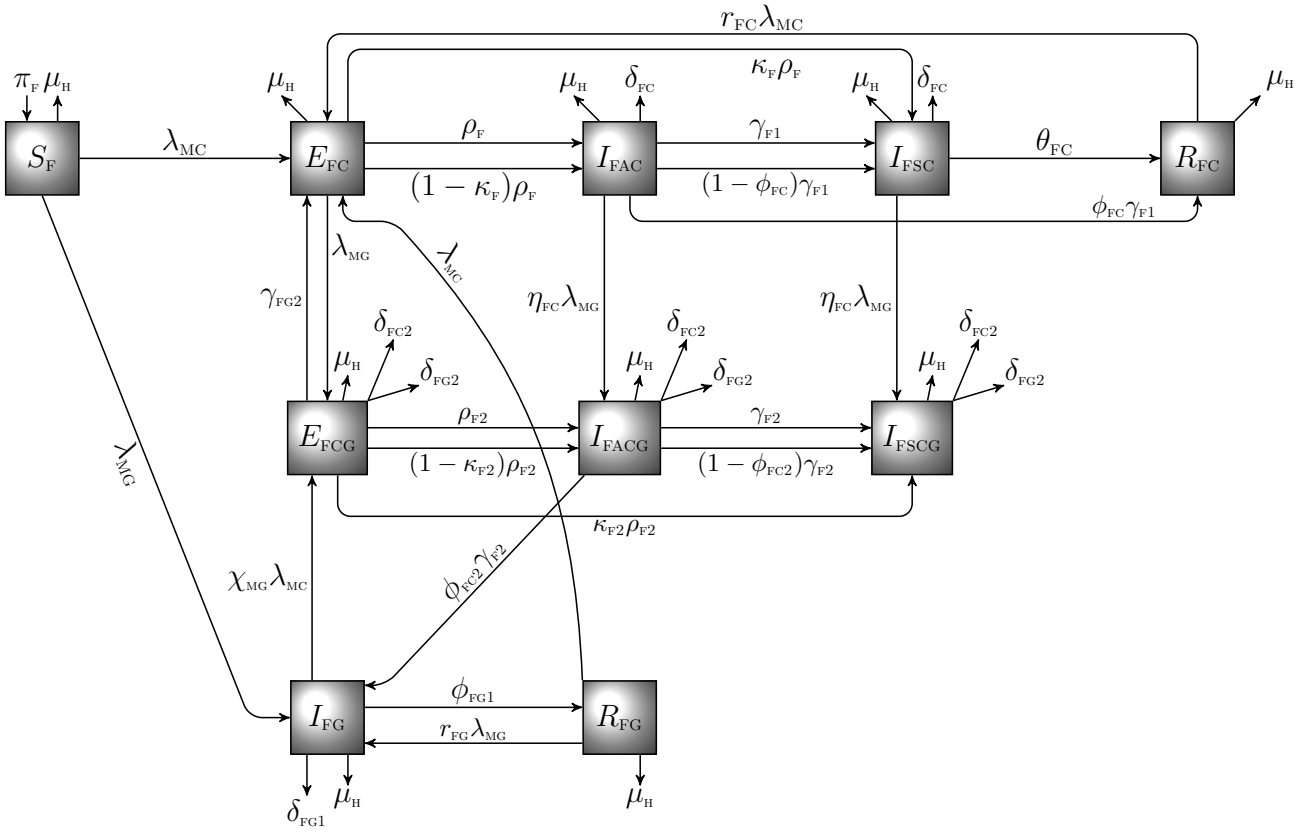
Table 3.1: Description of variables in the model (3.15).

Variable	**Interpretation**
$S_{\text{M}}(S_{\text{FC}})$	population of susceptible males (females)
$E_{\text{MC}}(E_{\text{FC}})$	Population of exposed males and females to chlamydia
$I_{\text{MAC}}(I_{\text{FAC}})$	Population of infectious males and females not showing symptoms of chlamydia
$I_{\text{MSC}}(I_{\text{FSC}})$	Population of males (females) showing symptoms of chlamydia
$R_{\text{MC}}(R_{\text{FC}})$	Population of recovered males and females
$I_{\text{MG}}(I_{\text{FG}})$	Population of of infectious males and females showing symptoms of gonorrhea
$R_{\text{MG}}(R_{\text{FG}})$	Population of recovered males and females from gonorrhea
$E_{\text{MCG}}(E_{\text{FCG}})$	Population of exposed males and females chlamydia and gonorrhea
$I_{\text{MACG}}(I_{\text{FACG}})$	Population of assymptomatics infected males and females with chlamydia and gonorrhea
$I_{\text{MSCG}}(I_{\text{FSCG}})$	Population of infectious males and females showing symptoms of chamydia and gonorrhea (males)
$N_{\text{HM}}(N_{\text{HF}})$	Total Population of males and females
N_{H}	Total Population

Table 3.2: Description of parameters in the model (3.15).

Parameter	**Interpretation**
$\pi_{\text{M}}(\pi_{\text{F}})$	Recruitment rates for males (females)
$\beta_{\text{M}}(\beta_{\text{F}})$	Probability of transmission for males and females
$\kappa_{\text{M}}(\kappa_{\text{F}})$	Fraction of infectious individuals that shows symptoms for males and females
$\rho_{\text{M}}(\rho_{\text{F}})$	Rate of symptoms development for exposed males and females
$\phi_{\text{M}}(\phi_{\text{F}})$	Natural recovery rates for males and females
$r_{\text{MC}}(r_{\text{FC}})$	Re-infection rates for males and females who have recovered from Chlamydia
$r_{\text{MG}}(r_{\text{FG}})$	Re-infection rates for males and females who have recovered from gonorrhoea
μ_{H}	natural death
$\gamma_{\text{M}}(\gamma_{\text{F}})$	Rate at which males and females leave the asymptomatic class
$\delta_{\text{M}}(\delta_{\text{F}})$	disease induced mortality rate for males and females
$\eta_{\text{MC}}(\eta_{\text{FC}})$	Modification for increased susceptibility to gonorrhoea due to chlamydia by males and females
χ_{MC}	Modification for increased susceptibility to chlamydia due to gonorrhoea by males
$\theta_{\text{M}}(\theta_{\text{F}})$	Natural clearance rates for males and females showing symptoms of chlamydia and gonorrhea
$\phi_{\text{M}}\gamma_{\text{M}}(\phi_{\text{F}}\gamma_{\text{F}})$	Natural clearance rate for males and females in the asymptomatic class


$r_{\mathrm{MC}}\lambda_{\mathrm{FC}}$
$\pi_{\mathrm{M}}\mu_{\mathrm{H}}$
μ_{H}
$\kappa_{\mathrm{M}}\rho_{\mathrm{M}}$
μ_{H} δ_{MC}
μ_{H} δ_{MC}
μ_{H}
S_{M}
λ_{FC}
E_{MC}
ρ_{M}
$(1-\kappa_{\mathrm{M}})\rho_{\mathrm{M}}$
I_{MAC}
γ_{M1}
$(1-\phi_{\mathrm{MC}})\gamma_{\mathrm{M1}}$
I_{MSC}
θ_{MC}
R_{MC}
$\phi_{\mathrm{MC}}\gamma_{\mathrm{M1}}$
λ_{FG}
γ_{MG2}
$\eta_{\mathrm{MC}}\lambda_{\mathrm{FG}}$
$\eta_{\mathrm{MC}}\lambda_{\mathrm{FG}}$
δ_{MC2} μ_{H} δ_{MG2}
E_{MCG}
ρ_{M2}
$(1-\kappa_{\mathrm{M2}})\rho_{\mathrm{M2}}$
I_{MACG}
γ_{M2}
$(1-\phi_{\mathrm{MC2}})\gamma_{\mathrm{M2}}$
I_{MSCG}
$\kappa_{\mathrm{M2}}\rho_{\mathrm{M2}}$
$\chi_{\mathrm{MG}}\lambda_{\mathrm{FC}}$
$\phi_{\mathrm{MC2}}\gamma_{\mathrm{M2}}$
δ_{MG1}
μ_{H}
I_{MG}
ϕ_{MG1}
$r_{\mathrm{MG}}\lambda_{\mathrm{FG}}$
R_{MG}
μ_{H}


$r_{\mathrm{FC}}\lambda_{\mathrm{MC}}$
$\pi_{\mathrm{F}}\mu_{\mathrm{H}}$
μ_{H}
$\kappa_{\mathrm{F}}\rho_{\mathrm{F}}$
μ_{H} δ_{FC}
μ_{H} δ_{FC}
μ_{H}
S_{F}
λ_{MC}
E_{FC}
ρ_{F}
$(1-\kappa_{\mathrm{F}})\rho_{\mathrm{F}}$
I_{FAC}
γ_{F1}
$(1-\phi_{\mathrm{FC}})\gamma_{\mathrm{F1}}$
I_{FSC}
θ_{FC}
R_{FC}
$\phi_{\mathrm{FC}}\gamma_{\mathrm{F1}}$
λ_{MG}
γ_{FG2}
$\eta_{\mathrm{FC}}\lambda_{\mathrm{MG}}$
$\eta_{\mathrm{FC}}\lambda_{\mathrm{MG}}$
δ_{FC2} μ_{H} δ_{FG2}
E_{FCG}
ρ_{F2}
$(1-\kappa_{\mathrm{F2}})\rho_{\mathrm{F2}}$
I_{FACG}
γ_{F2}
$(1-\phi_{\mathrm{FC2}})\gamma_{\mathrm{F2}}$
I_{FSCG}
$\kappa_{\mathrm{F2}}\rho_{\mathrm{F2}}$
$\chi_{\mathrm{MG}}\lambda_{\mathrm{MC}}$
$\phi_{\mathrm{FC2}}\gamma_{\mathrm{F2}}$
I_{FG}
ϕ_{FG1}
$r_{\mathrm{FG}}\lambda_{\mathrm{MG}}$
R_{FG}
δ_{FG1}
μ_{H}
μ_{H}

Chlamydia Trachomatis and Gonorrhea Co-infection Model equation

$$
\begin{aligned}
\frac{dS_{\text{M}}}{dt} &= \Pi_{\text{M}} - \lambda_{\text{FC}} S_{\text{M}} - \lambda_{\text{FG}} S_{\text{M}} - \mu_{\text{H}} S_{\text{M}} \\
\frac{dE_{\text{MC}}}{dt} &= \lambda_{\text{FC}} S_{\text{M}} + r_{\text{MC}} \lambda_{\text{FC}} R_{\text{MC}} - (\mu_{\text{H}} + \rho_{\text{M}}) E_{\text{MC}} - \lambda_{\text{FG}} E_{\text{MC}} + \gamma_{\text{MG2}} E_{\text{MCG}} + \lambda_{\text{FC}} R_{\text{MG}} - \lambda_{\text{MG}} E_{\text{MC}} \\
\frac{dI_{\text{MAC}}}{dt} &= (1-\kappa_{\text{M}}) \rho_{\text{M}} E_{\text{MC}} - (\mu_{\text{H}} + \delta_{\text{MC}} + \gamma_{\text{M1}}) I_{\text{MAC}} - \eta_{\text{MC}} \lambda_{\text{FG}}) I_{\text{MAC}} \\
\frac{dI_{\text{MSC}}}{dt} &= \kappa_{\text{M}} \rho_{\text{M}} E_{\text{MC}} + (1-\phi_{\text{MC}}) \gamma_{\text{M1}} I_{\text{MAC}} - (\mu_{\text{H}} + \delta_{\text{MC}} + \theta_{\text{MC}}) I_{\text{MSC}} - \eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MSC}} \\
\frac{dR_{\text{MC}}}{dt} &= \theta_{\text{MC}} I_{\text{MSC}} + \phi_{\text{MC}} \gamma_{\text{M1}} I_{\text{MAC}} - \mu_{\text{H}} R_{\text{MC}} - r_{\text{MC}} \lambda_{\text{FC}} R_{\text{MC}} - \lambda_{\text{FG}} R_{\text{MC}} \\
\frac{dS_{\text{F}}}{dt} &= \Pi_{\text{F}} - \lambda_{\text{MC}} S_{\text{F}} - \mu_{\text{H}} S_{\text{F}} - \lambda_{\text{MG}} S_{\text{F}} \\
\frac{dE_{\text{FC}}}{dt} &= \lambda_{\text{MC}} S_{\text{F}} + r_{\text{FC}} \lambda_{\text{MC}} R_{\text{FC}} - (\mu_{\text{H}} + \rho_{\text{F}}) E_{\text{FC}} - \lambda_{\text{MG}} E_{\text{FC}} + \gamma_{\text{FG2}} E_{\text{FCG}} + \lambda_{\text{MC}} R_{\text{FG}} \\
\frac{dI_{\text{FAC}}}{dt} &= (1-\kappa_{\text{F}}) \rho_{\text{F}} E_{\text{FC}} - (\mu_{\text{H}} + \delta_{\text{FC}} + \gamma_{\text{F1}}) I_{\text{FAC}} - \eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FAC}} \\
\frac{dI_{\text{FSC}}}{dt} &= \kappa_{\text{F}} \rho_{\text{F}} E_{\text{FC}} + (1-\phi_{\text{FC}}) \gamma_{\text{F1}} I_{\text{FAC}} - (\mu_{\text{H}} + \delta_{\text{FC}} + \theta_{\text{FC}}) I_{\text{FSC}} - \eta_{\text{FC}} \lambda_{\text{MG}}) I_{\text{FSC}} \\
\frac{dR_{\text{FC}}}{dt} &= \theta_{\text{FC}} I_{\text{FSC}} + \phi_{\text{FC}} \gamma_{\text{F1}} I_{\text{FAC}} - \mu_{\text{H}} R_{\text{FC}} - r_{\text{FC}} \lambda_{\text{MC}} R_{\text{FC}} \\
\frac{dI_{\text{MG}}}{dt} &= \lambda_{\text{FG}} S_{\text{M}} - (\mu_{\text{H}} + \phi_{\text{MG1}} + \delta_{\text{MG1}}) I_{\text{MG}} - \chi_{\text{MG}} \lambda_{\text{FC}} I_{\text{MG}} + \lambda_{\text{FG}} R_{\text{MC}} + r_{\text{MG}} \lambda_{\text{FG}} R_{\text{MG}} + \phi_{\text{MC2}} \gamma_{\text{M2}} I_{\text{MACG}} \\
\frac{dR_{\text{MG}}}{dt} &= \phi_{\text{MG1}} I_{\text{MG}} - \mu_{\text{H}} R_{\text{MG}} - \lambda_{\text{FC}} R_{\text{MG}} - r_{\text{MG}} \lambda_{\text{FG}} R_{\text{MG}} \\
\frac{dI_{\text{FG}}}{dt} &= \lambda_{\text{MG}} S_{\text{F}} - (\mu_{\text{H}} + \phi_{\text{FG1}}) I_{\text{FG}} - \chi_{\text{MG}} \lambda_{\text{MC}} - \delta_{\text{FG1}} I_{\text{FG}} + r_{\text{FG}} \lambda_{\text{MG}} R_{\text{FG}} + \phi_{\text{FC2}} \gamma_{\text{F2}} I_{\text{FACG}} \\
\frac{dR_{\text{FG}}}{dt} &= \phi_{\text{FG1}} I_{\text{FG}} - \mu_{\text{H}} R_{\text{FG}} - \lambda_{\text{MC}} R_{\text{FG}} - r_{\text{FG}} \lambda_{\text{MG}} R_{\text{FG}} \\
\frac{dE_{\text{MCG}}}{dt} &= \lambda_{\text{FG}} E_{\text{MC}} + \lambda_{\text{FG}} E_{\text{MC}} + \chi_{\text{MG}} \lambda_{\text{FC}} I_{\text{MG}} - (\mu_{\text{H}} + \delta_{\text{MG2}} + \gamma_{\text{MG2}} + \rho_{\text{M2}}) E_{\text{MCG}} \\
\frac{dI_{\text{MACG}}}{dt} &= \eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MAC}} + (1-\kappa_{\text{M2}}) \rho_{\text{M2}} E_{\text{MCG}} - (\mu_{\text{H}} + \delta_{\text{MC2}} + \delta_{\text{MG2}} + \gamma_{\text{M2}}) I_{\text{MACG}} \\
\frac{dI_{\text{MSCG}}}{dt} &= \eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MSC}} + \kappa_{\text{M2}} \rho_{\text{M2}} E_{\text{MCG}} - (\mu_{\text{H}} + \delta_{\text{MC2}} + \delta_{\text{MG2}}) I_{\text{MSCG}} + (1-\phi_{\text{MC2}}) \gamma_{\text{M2}} I_{\text{MACG}} \\
\frac{dE_{\text{FCG}}}{dt} &= \lambda_{\text{MG}} E_{\text{FC}} - (\mu_{\text{H}} + \delta_{\text{FG2}} + \gamma_{\text{FG2}} + \rho_{\text{F2}}) E_{\text{FCG}} + \chi_{\text{MG}} \lambda_{\text{MC}} I_{\text{FG}} + \lambda_{\text{MG}} E_{\text{FC}} \\
\frac{dI_{\text{FACG}}}{dt} &= \eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FAC}} + (1-\kappa_{\text{F2}}) \rho_{\text{F2}} E_{\text{FCG}} - (\mu_{\text{H}} + \delta_{\text{FC2}} + \delta_{\text{FG2}} + \gamma_{\text{F2}}) I_{\text{FACG}} \\
\frac{dI_{\text{FSCG}}}{dt} &= \eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FSC}} + \kappa_{\text{F2}} \rho_{\text{F2}} E_{\text{FCG}} - (\mu_{\text{H}} + \delta_{\text{FC2}} + \delta_{\text{FG2}}) I_{\text{FSCG}} + (1-\phi_{\text{FC2}}) \gamma_{\text{F2}} I_{\text{FACG}}
\end{aligned}
\tag{3.15}
$$

3.3.2 Description of model equations

The total sexually-active population at time t, denoted by ($N_{H}(t)$) is divided into 2 classes, namely the total male population ($N_{HM}(t)$) and the total female ($N_{HF}(t)$), respectively. The total male population is further sub-divided into 10 mutually exclusive compartments, susceptible males ($S_{M}(t)$), exposed males to chlamydia ($E_{MC}(t)$), infectious males showing no symptoms of chlamydia ($I_{MAC}(t)$), infectious males who have cleared (or recovered from) chlamydia infection ($R_{MC}(t)$), infectious males with gonorrhoea ($I_{MG}(t)$), infectious males who have cleared (or recovered from) gonorrhoea infection ($R_{MG}(t)$), exposed males to chlamydia and gonorrhoea infection ($E_{MCG}(t)$), infectious males showing no symptoms of chlamydia and gonorrhoea infections ($I_{MACG}(t)$), infectious males showing symptoms of chlamydia and gonorrhoea ($I_{MSCG}(t)$) similarly, The total female population is sub-divided into susceptible females ($S_{F}(t)$), exposed females to chlamydia ($E_{FC}(t)$), infectious females showing no symptoms of chlamydia ($I_{FAC}(t)$), infectious females who have cleared (or recovered from) chlamydia infection ($R_{FC}(t)$), infectious females with gonorrhoea ($I_{FG}(t)$), infectious females who have cleared (or recovered from) gonorrhoea infection ($R_{FG}(t)$), exposed females to chlamydia and gonorrhoea infection ($E_{FCG}(t)$), infectious females showing no symptoms of chlamydia and gonorrhoea infections ($I_{FACG}(t)$), infectious females showing symptoms of chlamydia and gonorrhoea ($I_{FSCG}(t)$).

$$N_{H}(t) = N_{HM}(t) + N_{HF}(t) \tag{3.16}$$

$$N_{HM}(t) = S_{M}(t)+E_{MC}(t)+I_{MAC}(t)+I_{MSC}(t)+R_{MC}(t)+I_{MG}(t)+R_{MG}(t)+E_{MCG}(t)+I_{MACG}(t)+I_{MSCG}(t) \tag{3.17}$$

$$N_{HF}(t) = S_{F}(t)+E_{FC}(t)+I_{FAC}(t)+I_{FSC}(t)+R_{FC}(t)+I_{FG}(t)+R_{FG}(t)+E_{FCG}(t)+I_{FACG}(t)+I_{FSCG}(t) \tag{3.18}$$

The susceptible populations (for both males and females) are increased by the recruitment of new sexually-active individuals. (assumed susceptible) into the population at rate Π_{M} and Π_{F} for male and female population, respectively. Susceptible males acquire chlamydia or gonorrhoea infection and become exposed, following effective contact with infected females (ie those in the E_{FC}, I_{FAC}, I_{FSC}, I_{FG}, E_{FCG}, I_{FACG}, I_{FSCG} classes at rate λ_{FC} and λ_{FG}) given by.

$$\lambda_{FC} = \frac{\beta_{FC}[I_{FAC} + \eta_{F1}I_{FSC} + \omega_{FG}(I_{FACG} + \eta_{F2}I_{FSCG})]}{N_{HF}} \tag{3.19}$$

$$\lambda_{FG} = \frac{\beta_{FG}[I_{FG} + E_{FCG} + \omega_{FC}(I_{FACG} + \eta_{F2} I_{FSCG})]}{N_{HF}} \tag{3.20}$$

Similarly, susceptible females acquire chlamydia infection following effective contact with males infected with chlamydia (ie those in the E_{MC}, I_{MAC}, I_{MSC}, I_{MG}, E_{MCG}, I_{MACG}, I_{MSCG} classes at rate λ_{FC} and λ_{FG}) given by.

$$\lambda_{MC} = \frac{\beta_{MC}[I_{MAC} + \eta_{M1} I_{MSC} + \omega_{MG}(I_{MACG} + \eta_{M2} I_{MSCG})]}{N_{HM}} \tag{3.21}$$

$$\lambda_{MG} = \frac{\beta_{MG}[I_{MG} + E_{MCG} + \omega_{MC}(I_{MACG} + \eta_{M2} I_{MSCG})]}{N_{HM}} \tag{3.22}$$

in (3.19),(3.20),(3.21),(3.22), β_{FC},β_{MC},β_{FG},β_{MG} are the probabilities of transmitting chlamydia or gonorrhoea from male to female and from female to male respectively.The terms ω_{FC},ω_{MC},ω_{FG},ω_{MG}. represents the rate at which males and females Further, the modification parameters η_{F1},η_{F2},η_{M1},η_{M2} account for the assumed increase in the relative infectiousness of individuals in I_{MAC} and I_{FAC} (infectious individuals showing no symptoms of chlamydia) I_{MACG} and I_{FACG} (infectious individuals showing no symptoms of chlamydia or gonorrhoea) I_{MSC} and I_{FSC} (infectious individuals showing symptoms of chlamydia) I_{MSCG} and I_{FSCG} (infectious individuals showing symptoms of chlamydia or gonorrhoea) classes in comparison to exposed males and females in E_{MC},E_{FC},E_{MCG},E_{FCG}, respectively. That it is assumed that infected females in the I_{FAC},I_{FSC},I_{FG},I_{FACG} and I_{FSCG} classes are more infectious than infected females in the E_{FC} and E_{FCG} class similarly, infected males in the I_{MAC},I_{MSC},I_{MG},I_{MACG} and I_{MSCG} are assumed to be more infectious than infected males in the E_{MC} and E_{MCG}. After 1 - 10 days of becoming exposed to chlamydia and gonorrhoea infection, such individuals will become at rate $\rho_{M}(\rho_{F})$,$\rho_{M2}(\rho_{F2})$for male(female), a fraction $\kappa_{M}(\kappa_{F})$,$\kappa_{M2}(\kappa_{F2})$ of these individuals will start to show symptoms of chlamydia or gonorrhoea infection and move to the class (I_{MSC},I_{MG},I_{MSCG})(I_{FSC},I_{FG},I_{FSCG}),while the remaining fraction $(1-\kappa_{M})$, $(1-\kappa_{F})$,$(1-\kappa_{M2})$, $(1-\kappa_{F2})$ will not (but still remain capable of infecting others). infectious individuals that do not initially show symptoms (ie those in the I_{MAC},I_{FAC},I_{MACG} and I_{FACG} classes) are expected to suffer severe complications, such as infertility, if untreated However, they eventually show symptoms at rate $(1-\phi_{MC})\gamma_{M1}$, $(1-\phi_{FC})\gamma_{F1}$,$(1-\phi_{MC2})\gamma_{M2}$,$(1-\phi_{FC"})\gamma_{F2}$ for the male(female) population, while the remaining fraction $\phi_{MC}\gamma_{M1}$, $\phi_{FC}\gamma_{F1}$,$\phi_{MC}\gamma_{M2}$ and $\phi_{FC2}\gamma_{F2}$ recovers naturally,infectious individuals showing symptoms of chlamydia and gonorrhoea infections at rate $\theta_{MC}(\theta_{FC})$ for male(female) population. Recovered individuals can be re-infected at rates $r_{MG}\lambda_{FG}$,$r_{FG}\lambda_{MG}$,$r_{FC}\lambda_{MC}$ and $r_{MC}\lambda_{FC}$ for males and females, respectively with $r_{MG} > 0$,$r_{FG} > 0$,$r_{FC} > 0$, and $r_{MC} > 0$ representing the re-infection parameters for males and fe-

males, $\eta_{MC}(\eta_{FC})$ this is the modification for increased susceptibility to gonorrhoea due to chlamydia by males and females.while $\chi_{MC}(\chi_{FC})$ is the modification increased susceptibility to chlamydia due to gonorrhoea by males and females. Further, natural mortality occurs in all classes at a rate μ_H while individuals in the I_{MAC},I_{MSC},I_{MG},I_{MACG}, I_{MSCG} and I_{FAC},I_{FSC},I_{FG},I_{FACG},I_{FSCG} classes suffer an additional disease-induced death at rates δ_{MC},δ_{FC},δ_{MG1},δ_{FG1},δ_{MG2},δ_{MC2},δ_{FG2} and δ_{FC2}.respectively combining all these definitions and assumptions it follows that the basic model for the transmission of chlamydia and gonorrhoea co-infection in a sexually-active population is given by the following system of differential equations. The population of Susceptible male S_M is increased by recruitment of susceptible males at the rate Π_M the population is decreased due to th infection with chlamydia trachomatis (CT) from infected females at rate λ_{FC} and also due to infection with gonorrhea from infected females at rate λ_{FG}. The population is further reduced due natural death at the rate μ_H Thus,

$$\frac{dS_M}{dt} = \Pi_M - \lambda_{FC}S_M - \lambda_{FG}S_M - \mu_H S_M$$

The population of males exposed to Chlamydia Trachomatis (CT) ($E_{MC}(t)$) is increased due to infection with chlamydia Trachomatis (CT) by susceptible males at the rate λ_{FC} the population is further increased due to the re-infection by males who have recovered from Chlamydia Trachomatis (CT) at the rate ($r_{MC}\lambda_{FC}$) Moreover, it is increased due to the infection with Chlamydia Trachomatis by males who have recovered from Gonorrhea at the rate λ_{FC}, and also increased when individuals co-infected with exposed chlamydia and Gonorrhea recover from Gonorrhea at the rate γ_{MG2}. The population is further decreased due to natural death rate μ_H, and due to transition out of (E_{MC}) compartment at the rate ρ_M, population is decreased due to infection with gonorrhea by males exposed to Chlamydia Trachomatis at rate λ_{FG}.

$$\frac{dE_{MC}}{dt} = \lambda_{FC}S_M + r_{MC}\lambda_{FC}R_{MC} - (\mu_H + \rho_M)E_{MC} - \lambda_{FG}E_{MC} + \gamma_{MG2}E_{MCG} + \lambda_{FC}R_{MG} - \lambda_{MG}E_{MC}$$

The population of males with asymptomatic Chlamydia Trachomatis infection ($I_{MAC}(t)$) is increased by fraction, $(1-\kappa_M)\rho_M$ of males who are exposed to Chlamydia Trachomatis (CT) infection. The population is further reduced due to increase in modification susceptibility by males with asymptomatic Chlamydia Trachomatis (CT) at the rate $\eta_{MC}\lambda_{FG}$ and the population is also reduced due to natural death rate,disease induced death and males infected with asymptomatic Chlamydia

Trachomatis (CT) at the rates μ_{H},δ_{MC},γ_{M1} respectively.

$$\frac{dI_{MAC}}{dt} = (1-\kappa_{M})\rho_{M}E_{MC} - (\mu_{H} + \delta_{MC} + \gamma_{M1})I_{MAC} - \eta_{MC}\lambda_{FG})I_{MAC}$$

The population of males with symptomatic Chlamydia Trachomatis ($I_{MSC}(t)$) is increased by a fraction, $\kappa_{M}\rho_{M}$ of males exposed to chlamydia infection. The population is further increased by a fraction $(1-\phi_{MC})\gamma_{M1}$ of males infected with asymptomatic Chlamydia Trachomatis (CT) infection. This population is reduced due to increased in modification susceptibility by males infected with Chlamydia Trachomatis (CT) at the rate $\eta_{MC}\lambda_{FG}$. The population is further reduced by natural death, disease induced death and natural clearance of males infected with Chlamydia Trachomatis (CT) at the rates μ_{H},δ_{MC},θ_{MC} respectively.

$$\frac{dI_{MSC}}{dt} = \kappa_{M}\rho_{M}E_{MC} + (1-\phi_{MC})\gamma_{M1}I_{MAC} - (\mu_{H} + \delta_{MC} + \theta_{MC})I_{MSC} - \eta_{MC}\lambda_{FG}I_{MSC}$$

The population of males who have recovered from Chlamydia Trachomatis ($R_{MC}(t)$) is increased due to natural clearance by males with Chlamydia Trachomatis (CT) at rate θ_{MC}, the population is further increased by a fraction natural clearance of males infected with Chlamydia Trachomatis (CT) that are asymptomatic at the rate $\phi_{MC}\gamma_{M1}$ and the population is decreased due to natural death at rate μ_{H}. However, The population is decreased due to re-infection by males who have recovered from Chlamydia Trachomatis (CT) at the rate $r_{MC}\lambda_{FC}$ and also the population is decreased due to males with gonorrhea infection who have recovered from chlamydia at the rate λ_{FG}.

$$\frac{dR_{MC}}{dt} = \theta_{MC}I_{MSC} + \phi_{MC}\gamma_{M1}I_{MAC} - \mu_{H}R_{MC} - r_{MC}\lambda_{FC}R_{MC} - \lambda_{FG}R_{MC}$$

The population of Susceptible female S_{F} is increased by recruitment of susceptible females at the rate Π_{F} the population is decreased due to th infection with chlamydia trachomatis (CT) from infected males at rate λ_{MC} and also due to infection with gonorrhea from infected males at rate λ_{MG}. The population is further reduced due natural death at the rate μ_{H}. Thus,

$$\frac{dS_{F}}{dt} = \Pi_{F} - \lambda_{MC}S_{F} - \mu_{H}\ S_{F} - \lambda_{MG}S_{F}$$

The population of females exposed to Chlamydia Trachomatis (CT) ($E_{FC}(t)$) is increased due to infection with chlamydia Trachomatis (CT) by susceptible females at the rate λ_{MC} the population is further increased due to the re-infection by females who have recovered from Chlamydia Trachomatis (CT) at the rate ($r_{FC}\lambda_{MC}$) Moreover, it is increased due to the infection with Chlamydia Trachomatis by females who have recovered from Gonorrhea at the rate λ_{MC}, and also increased when individuals co-infected with exposed chlamydia and Gonorrhea recover from Gonorrhea at the rate γ_{FG2}. The population is further decreased due to natural death rate μ_H, and due to transition out of (E_{FC}) compartment at the rate ρ_F, population is decreased due to infection with gonorrhea by females exposed to Chlamydia Trachomatis at rate λ_{MG}.

$$\frac{dE_{FC}}{dt} = \lambda_{MC}S_F + r_{FC}\lambda_{MC}R_{FC} - (\mu_H + \rho_F)E_{FC} - \lambda_{MG}E_{FC} + \gamma_{FG2}E_{FCG} + \lambda_{MC}R_{FG}$$

The population of females with asymptomatic Chlamydia Trachomatis infection ($I_{FAC}(t)$) is increased by fraction, $(1-\kappa_F)\rho_F$ of females who are exposed to Chlamydia Trachomatis (CT). The population is further reduced due to increase in modification susceptibility by females with asymptomatic Chlamydia Trachomatis (CT) infection at the rate $\eta_{FC}\lambda_{MG}$ and the population is also reduced due to natural death rate,disease induced death and males infected with asymptomatic Chlamydia Trachomatis (CT) at the rates μ_H,δ_{FC},γ_{F1} respectively.

$$\frac{dI_{FAC}}{dt} = (1-\kappa_F)\rho_F E_{FC} - (\mu_H + \delta_{FC} + \gamma_{F1})I_{FAC} - \eta_{FC}\lambda_{MG}I_{FAC}$$

The population of females with symptomatic Chlamydia Trachomatis ($I_{FSC}(t)$) infection is increased by a fraction, $\kappa_F\rho_F$ of females who are exposed to Chlamydia Trachomatis infection. The population is further increased by a fraction $(1-\phi_{FC})\gamma_{F1}$ of females with asymptomatic Chlamydia Trachomatis (CT). This population is reduced due to increased in modification susceptibility by females with symptomatic Chlamydia Trachomatis (CT) infection at the rate $\eta_{FC}\lambda_{MG}$. The population is further reduced by natural death, disease induced death and natural clearance of males with Chlamydia Trachomatis (CT) at the rates μ_H,δ_{FC},θ_{FC} respectively.

$$\frac{dI_{FSC}}{dt} = \kappa_F\rho_F E_{FC} + (1-\phi_{FC})\gamma_{F1}I_{FAC} - (\mu_H + \delta_{FC} + \theta_{FC})I_{FSC} - \eta_{FC}\lambda_{MG})I_{FSC}$$

The population of females who recovered from Chlamydia Trachomatis (CT) is increased due to natural clearance by females infected with Chlamydia Trachomatis (CT) infection at rate θ_{FC}, the population is further increased by a fraction natural clearance of females infected with Chlamydia Trachomatis (CT) that are asymptomatic at the rate $\phi_{FC}\gamma_{F1}$ and the population is decreased due to natural death at rate μ_{H}. However, The population is also decreased due to re-infection by females who have recovered from Chlamydia Trachomatis (CT) at the rate $r_{FC}\lambda_{MC}$.

$$\frac{dR_{FC}}{dt} = \theta_{FC}I_{FSC} + \phi_{FC}\gamma_{F1}I_{FAC} - \mu_{H}R_{FC} - r_{FC}\lambda_{MC}R_{FC}$$

The population of males with Gonorhea infection ($I_{MG}(t)$) is increased due to infection with Gonorrhea by susceptible males at the rate λ_{FG}. The population is further increased due to infection with Gonorrhea by males who have recovered from Chlamydia Trachomatis at the rate λ_{FG} and also increased due to re-infection by males who have recovered from Gonorrhea at the rate $r_{MG}\lambda_{FG}$ Moreover, the population is increased by a fraction $\phi_{MC2}\gamma_{M2}$ of males infected with Chlamydia Trachomatis and Gonorrhea infection that are asymptomatic.The population is decreased by increase in modification susceptibility by males infected with gonorhea infection at rate $\chi_{MG}\lambda_{FG}$. the population is further reduced following by natural death, disease induced death,naturalat recovery by males infected with Gonorrhea the rates μ_{H},δ_{MG1},ϕ_{MG1} repectively.

$$\frac{dI_{MG}}{dt} = \lambda_{FG}S_{M} - (\mu_{H} + \phi_{MG1} + \delta_{MG1})I_{MG} - \chi_{MG}\lambda_{FC}I_{MG} + \lambda_{FG}R_{MC} + r_{MG}\lambda_{FG}R_{MG} + \phi_{MC2}\gamma_{M2}I_{MACG}$$

The population of males who recover from gonorrhea infections ($R_{MG}(t)$) is increased due to natural recovery by males infected with Gonorrhea and population is further decreased by natural death at rate μ_{H} also the population is reduced due to infection with gonorrhea by males who recovered from gonorrhea at rate λ_{FC}. However, the population is reduced due to re-infection by males who have recovered from gonorrhea infection at the rate $r_{MG}\lambda_{FG}$.

$$\frac{dR_{MG}}{dt} = \phi_{MG1}I_{MG} - \mu_{H}\ R_{MG} - \lambda_{FC}R_{MG} - r_{MG}\lambda_{FG}R_{MG}$$

The population of females with Gonorhea infection ($I_{FG}(t)$) is increased due to infection with Gonorrhea by susceptible females at the rate λ_{MG}. The population is further increased due to re-infection by females who have recovered from Gonorrhea at the rate $r_{FG}\lambda_{MG}$ Moreover, the

population is increased by a fraction $\phi_{FC2}\gamma_{F2}$ of females co-infected with Chlamydia Trachomatis and Gonorrhea that are asymptomatic.The population is decreased by increase in modification susceptibility by females infected with gonorhea infection at rate $\chi_{MG}\lambda_{FG}$. the population is further reduced by natural death, disease induced death and natural recovery by females infected with gonorrhea infection at the rates μ_H,δ_{FG1},ϕ_{FG1} repectively.

$$\frac{dI_{FG}}{dt} = \lambda_{MG}S_F - (\mu_H + \delta_{FG1} + \phi_{FG1})I_{FG} - \chi_{MG}\lambda_{MC}I_{FG} + r_{FG}\lambda_{MG}R_{FG} + \phi_{FC2}\gamma_{F2}I_{FACG}$$

The population of females who recover from gonorrhea infections ($R_{FG}(t)$) is increased due to natural recovery by females infected with Gonorrhea at the rate ϕ_{FG1} and population is further decreased by natural death at rate μ_H also the population is reduced due to infection with gonorrhea by females who recovered from gonorrhea at rate λ_{MC}. Moreover, the population is reduced due to re-infection by females who have recovered from gonorrhea infection at the rate $r_{FG}\lambda_{MG}$.

$$\frac{dR_{FG}}{dt} = \phi_{FG1}I_{FG} - \mu_H\ R_{FG} - \lambda_{MC}R_{FG} - r_{FG}\lambda_{MG}R_{FG}$$

The population of males exposed to Chlamydia Trachomatis and Gonorrhea co-infection ($E_{MCG}(t)$) is increased due to the infection with gonorrhea by males exposed to Chlamydia Trachomatis (CT) at the rate λ_{FG} and the population is also increased in modification susceptibility by males infected with gonorrhea infection at the rate $\chi_{MG}\lambda_{FG}$ However the population is reduced by natural death,disease induced death,males with asymptomatic Gonorhea infection and due to the transition out of E_{MCG} compartment at the rates μ_H,δ_{MG2},γ_{MG2} and ρ_{M2}

$$\frac{dE_{MCG}}{dt} = \lambda_{FG}E_{MC} + \lambda_{FG}E_{MC} + \chi_{MG}\lambda_{FC}I_{MG} - (\mu_H + \delta_{MG2} + \gamma_{MG2} + \rho_{M2})E_{MCG}$$

The population of males with asymptomatic Chlamydia Trachomatis and Gonorrhea co-infection ($I_{MACG}(t)$) is increased due to modification susceptibility of males infected with Chlamydia Trachomatis (CT) that are asymptomatic at the rate $\eta_{MC}\lambda_{FG}$ and the population is further increased by fraction, $(1-\kappa_{M2})\rho_{M2}$ of males who are exposed to Chlamydia Trachomatis (CT) and Gonorrhea infections. The population is further reduced due to natural death rate,disease induced death from males infected with Chlamydia Trachomatis,disease induced death from males infected with Gonorrhea and males infected with Chlamydia Trachomatis (CT) and Gonorrhea that are

asymptomatic at the rates μ_{H},δ_{MC2},δ_{MG2},γ_{M2} respectively.

$$\frac{dI_{MACG}}{dt} = \eta_{MC}\lambda_{FG}I_{MAC} + (1-\kappa_{M2})\rho_{M2}E_{MCG} - (\mu_{H} + \delta_{MC2} + \delta_{MG2} + \gamma_{M2})I_{MACG}$$

The population of males with symptomatic Chlamydia Trachomatis and gonorrhea infection ($I_{MSCG}(t)$) is increased by the modification susceptibility by males infected with Chlamydia Trachomatis infection at the rate $\eta_{MC}\lambda_{FG}$.The population is increased by a fraction, $\kappa_{M2}\rho_{M2}$ of males exposed to Chlamydia Trachomatis and gonorrhea infection. The population is further increased by a fraction $(1-\phi_{MC2})\gamma_{M2}$ of males infected with asymptomatic Chlamydia Trachomatis (CT) and Gonorrhea.The population is further reduced by natural death, disease induced death of males infected with Chlamydia Trachomatis infection and disease induced death of males infected with Gonorrhea at the rates μ_{H},δ_{MC2},δ_{MG2} respectively.

$$\frac{dI_{MSCG}}{dt} = \eta_{MC}\lambda_{FG}I_{MSC} + \kappa_{M2}\rho_{M2}E_{MCG} - (\mu_{H} + \delta_{MC2} + \delta_{MG2})I_{MSCG} + (1-\phi_{MC2})\gamma_{M2}I_{MACG}$$

The population of females exposed to Chlamydia Trachomatis and Gonorrhea co-infection ($E_{FCG}(t)$) is increased due to the infection with gonorrhea by females exposed to Chlamydia Trachomatis (CT) at the rate λ_{MG} and the population is also increased in modification susceptibility by females infected with gonorrhea infection at the rate $\chi_{MG}\lambda_{MC}$ However the population is reduced by natural death,disease induced death,females with asymptomatic Gonorhea infection and due to the transition out of E_{FCG} compartment at the rates μ_{H},δ_{FG2},γ_{FG2} and ρ_{F2}.

$$\frac{dE_{FCG}}{dt} = \lambda_{MG}E_{FC} - (\mu_{H} + \delta_{FG2} + \gamma_{FG2} + \rho_{F2})E_{FCG} + \chi_{MG}\lambda_{MC}I_{FG} + \lambda_{MG}E_{FC}$$

The population of females infected with asymptomatic Chlamydia Trachomatis and Gonorrhea infection ($I_{FACG}(t)$) is increased due to modification susceptibility by females with asymptomatic Chlamydia Trachomatis infection at the rate $\eta_{FC}\lambda_{MG}$, the population is increased by a fraction, $(1-\kappa_{F2})\rho_{F2}$ of females who are exposed to Chlamydia Trachomatis (CT) and Gonorrhea infection. The population is also reduced due to natural death rate,disease induced death by females with Chlamydia Trachomatis infection, disease induced death by females with Gonorrhea and females

with asymptomatic Chlamydia Trachomatis (CT) at the rates μ_{H},δ_{FC2},δ_{FG2},γ_{F2} respectively.

$$\frac{dI_{FACG}}{dt} = \eta_{FC}\lambda_{MG}I_{FAC} + (1-\kappa_{F2})\rho_{F2}E_{FCG} - (\mu_{H} + \delta_{FC2} + \delta_{FG2} + \gamma_{F2})I_{FACG}$$

The population of females with symptomatic Chlamydia Trachomatis and gonorrhea infection ($I_{FSCG}(t)$) is increased due to modification susceptibility by females infected with Chlamydia Trachomatis infection at the rate $\eta_{FC}\lambda_{MG}$, and this population is increased by a fraction, $\kappa_{F2}\rho_{F2}$ of females who are exposed to Chlamydia Trachomatis and gonorrhea infection. The population is further increased by a fraction $(1-\phi_{FC2})\gamma_{F2}$ of females with asymptomatic Chlamydia Trachomatis (CT) and Gonorrhea infection. Moreover, The population is further reduced by natural death, disease induced death by females infected with Chlamydia Trachomatis (CT) and disease induced death by females infected with Gonorrhea infection at the rates μ_{H},δ_{FC2},δ_{FG2} respectively.

$$\frac{dI_{FSCG}}{dt} = \eta_{FC}\lambda_{MG}I_{FSC} + \kappa_{F2}\rho_{F2}E_{FCG} - (\mu_{H} + \delta_{FC2} + \delta_{FG2})I_{FSCG} + (1-\phi_{FC2})\gamma_{F2}I_{FACG}$$

Chapter 4

Results and Discussion

4.1 Mathematical analysis of the model (3.15)

4.1.1 Basic properties of the Chlamydia Trachomatis and Gonorrhea co-infection model (3.15)

Positivity and boundedness of solutions

For the model (3.15) to be epidemiologically meaningful, it is important to prove that all its state variables are non-negative for all time (t). In other words, solutions of the model system (3.15) with positive initial data will remain positive for all time $t > 0$.

Theorem 4.1. *Let the initial data* $S_{M} > 0, E_{MC} > 0, I_{MAC} > 0, I_{MSC} > 0, R_{MC} > 0, S_{F} > 0, E_{FC} > 0, I_{FAC} > 0, I_{FSC} > 0, R_{FC} > 0, I_{MG} > 0, R_{MG} > 0,\ I_{FG} > 0, R_{FG} > 0, E_{MCG} > 0, I_{MACG} > 0, I_{MSCG} > 0, E_{FCG} > 0, I_{FACG} > 0, I_{FSCG} > 0$

Then the solutions

$(S_{M}, E_{MC}, I_{MAC}, I_{MSC}, R_{MC}, S_{F}, E_{FC}, I_{FAC}, I_{FSC}, R_{FC}, I_{MG}, R_{MG}, I_{FG}, R_{FG}, E_{MCG}, I_{MACG}, I_{MSCG}, E_{FCG}, I_{FACG}, I_{FSCG}$

of the model (3.15) *are positive for all time* $t > 0$.

Proof. Let

$t_1 = \sup\{t > 0 : S_{M} > 0, E_{MC} > 0, I_{MAC} > 0, I_{MSC} > 0, R_{MC} > 0, S_{F} > 0, E_{FC} > 0, I_{FAC} > 0, I_{FSC} > 0, R_{FC} > 0, I_{MG} > 0, R_{MG} > 0, I_{FG} > 0, R_{FG} > 0, E_{MCG} > 0, I_{MACG} > 0, I_{MSCG} > 0, E_{FCG} > 0, I_{FACG} > 0, I_{FSCG} > 0 \in [0, t]\}$. Thus, $t_1 > 0$.

We have, from the first equation of the system (3.15) that

$$\frac{dS_{M}}{dt} = \Pi_{M} - \lambda_{FC}S_{M} - \lambda_{FG}S_{M} - \mu_{H}S_{M}$$

which can be re-written as

$$\frac{d}{dt}\left\{S_{M}(t)\exp\left[\int_{0}^{t}(\lambda_{FC}(u)+\lambda_{FG}(u))du+\mu_{H}(t)\right]\right\} = \Pi_{M}\exp\left[\int_{0}^{t}(\lambda_{FC}(u)+\lambda_{FG}(u))du+\mu_{H}(t)\right]$$

Hence:

$$\frac{d}{dt}\left\{S_{M}(t_1)\exp\left[\int_{0}^{t_1}(\lambda_{FC}(u)+\lambda_{FG}(u))du+\mu_{H}(t)_1\right]\right\} - S_{M}(0) = \Pi_{M}\int_{0}^{t_1}\exp\left[\int_{0}^{x}(\lambda_{FC}(u)+\lambda_{FG}(u))du+\mu_{H}(x)\right]d$$

so that

$$S_{M}(t_1) = S_{M}(0)\exp\left[-\int_{0}^{t_1}(\lambda_{FC}(u)+\lambda_{FG}(u))du-\mu_{H}t_1\right] + \exp\left[-\int_{0}^{t_1}(\lambda_{FC}(u)+\lambda_{FG}(u))du-\mu_{H}t_1\right]$$
$$\times\,\Pi_{M}\int_{0}^{t_1}\exp\left[\int_{0}^{x}(\lambda_{FC}(u)+\lambda_{FG}(u))du+\mu_{H}(x)\right]dx > 0$$

Similarly, it can be shown that:

$S_{M} > 0, E_{MC} > 0, I_{MAC} > 0, I_{MSC} > 0, R_{MC} > 0, S_{F} > 0, E_{FC} > 0, I_{FAC} > 0, I_{FSC} > 0, R_{FC} > 0, I_{MG} > 0, R_{MG} > 0, I_{FG} > 0, R_{FG} > 0, E_{MCG} > 0, I_{MACG} > 0, I_{MSCG} > 0, E_{FCG} > 0, I_{FACG} > 0, I_{FSCG} > 0.$

□

4.1.2 Invariant regions

The Co-infection model (3.15) will be analyzed in a biologically feasible region as follows. We first show that the system (3.15) is dissipative in a proper subset $\mathcal{D} \subset \mathfrak{R}^{20}_{+}$. The system (3.15) is split into two parts, namely the male population (N_{HM}) (with $N_{HM} = S_{M} + E_{MC} + I_{MAC} + I_{MSC} + R_{MC} + I_{MG} + R_{MG} + E_{MCG} + I_{MACG} + I_{MSCG}$) and the female population,
(N_{HF}) (with $N_{HF} = S_{F} + E_{MC} + I_{FAC} + I_{FSC} + R_{FC} + I_{FG} + R_{FG} + E_{FCG} + I_{FACG} + I_{FSCG}$).
Consider the feasible region $\mathcal{D} = \mathcal{D}_F \cup \mathcal{D}_M \subset \mathfrak{R}^{10}_{+} \times \mathfrak{R}^{10}_{+}$,
with:

$$\mathcal{D}_F = \Bigg\{(S_{\text{F}}, E_{\text{FC}}, I_{\text{FAC}}, I_{\text{FSC}}, R_{\text{FC}}, I_{\text{FG}}, R_{\text{FG}}, E_{\text{FCG}}, I_{\text{FACG}}, I_{\text{FSCG}}) \in \mathfrak{R}^{10}_{+} : S_{\text{F}} + E_{\text{FC}} + I_{\text{FAC}} + I_{\text{FSC}} + R_{\text{FC}} + I_{\text{FG}} + R_{\text{FG}} + E_{\text{FCG}} + I_{\text{FACG}} + I_{\text{FSCG}} \leq \frac{\Pi_{\text{F}}}{\mu_{\text{H}}}\Bigg\}$$

and

$$\mathcal{D}_M = \Bigg\{(S_{\text{M}}, E_{\text{MC}}, I_{\text{MAC}}, I_{\text{MSC}}, R_{\text{MC}}, I_{\text{MG}}, R_{\text{MG}}, E_{\text{MCG}}, I_{\text{MACG}}, I_{\text{MSCG}}) \in \mathfrak{R}^{10}_{+} :: S_{\text{M}} + E_{\text{MC}} + I_{\text{MAC}} + I_{\text{MSC}} + R_{\text{MC}} + I_{\text{MG}} + R_{\text{MG}} + E_{\text{MCG}} + I_{\text{MACG}} + I_{\text{MSCG}} \leq \frac{\Pi_{\text{F}}}{\mu_{\text{H}}}\Bigg\}$$

The following steps are followed to establish the positive invariance of $\mathcal{D}$ (i.e. solutions in $\mathcal{D}$ remain in $\mathcal{D}$ for all time $t > 0$).

Adding the first ten which is the total population of the female and the last ten equations which is the total population of the male in the differential system (3.15) gives

$$\begin{aligned}
\frac{dN_{\text{HF}}}{dt} &= \Pi_{\text{F}} - \mu_{\text{H}} N_{\text{HF}}(t) - [\delta_{\text{FC}} I_{\text{FAC}} + \delta_{\text{FC}} I_{\text{FSC}} + \delta_{\text{FG1}} I_{\text{FG}} + \delta_{\text{FG2}} E_{\text{FCG}} + (\delta_{\text{FC2}} + \delta_{\text{FG2}}) I_{\text{FACG}} \\
&\quad + (\delta_{\text{FC2}} + \delta_{\text{FG2}}) I_{\text{FSCG}} +] \\
\frac{dN_{\text{HM}}}{dt} &= \Pi_{\text{M}} - \mu_{\text{H}} N_{\text{HM}}(t) - [\delta_{\text{MC}} I_{\text{MAC}} + \delta_{\text{MC}} I_{\text{MSC}} + \delta_{\text{MG1}} I_{\text{MG}} + \delta_{\text{MG2}} E_{\text{MCG}} + (\delta_{\text{MC2}} + \delta_{\text{MG2}}) I_{\text{MACG}} \\
&\quad + (\delta_{\text{MC2}} + \delta_{\text{MG2}}) I_{\text{MSCG}} +]
\end{aligned} \tag{4.1}$$

From (4.1), we have that

$$\begin{aligned}
\Pi_{\text{F}} - (\mu_{\text{H}} + 8\delta_{\text{F}}) N_{\text{HF}} \leq \frac{dN_{\text{HF}}}{dt} < \Pi_{\text{F}} - \mu_{\text{H}} N_{\text{HF}} \\
\Pi_{\text{M}} - (\mu_{\text{H}} + 8\delta_{\text{M}}) N_{\text{HM}} \leq \frac{dN_{\text{HM}}}{dt} < \Pi_{\text{M}} - \mu_{\text{H}} N_{\text{HM}}
\end{aligned}$$

where $\delta_F = \min\{\delta_{\text{FC}}, \delta_{\text{FG1}}, \delta_{\text{FG2}}, \delta_{\text{FC2}}\}$ and $\delta_{\text{M}} = \min\{\delta_{\text{MC}}, \delta_{\text{MG1}}, \delta_{\text{MG2}}, \delta_{\text{MC2}}\}$.

Using the Comparison theorem (Lakshmikantham, et al., 1989), we have that $N_{\text{HF}}(t) \leq N_{\text{HF}}(0)\text{e}^{-\mu_{\text{H}} t} + \frac{\Pi_F}{\mu_{\text{H}}}(1 - \text{e}^{-\mu_{\text{H}} t})$ and $N_{\text{HM}}(t) \leq N_{\text{HM}}(0)\text{e}^{-\mu_{\text{H}} t} + \frac{\Pi_M}{\mu_{\text{H}}}(1 - \text{e}^{-\mu_{\text{H}} t})$.In particular, $N_{\text{HF}}(t) \leq \frac{\Pi_{\text{F}}}{\mu_{\text{H}}}$ and $N_{\text{HM}}(t) \leq \frac{\Pi_{\text{M}}}{\mu_{\text{H}}}$ if $N_{\text{HF}}(0) \leq \frac{\Pi_{\text{F}}}{\mu_{\text{H}}}$ and $N_{\text{HM}}(0) \leq \frac{\Pi_{\text{M}}}{\mu_{\text{H}}}$ respectively. Thus, the region $\mathcal{D}$ is positively invariant. Hence, it is sufficient to consider the dynamics of the flow generated by the system (3.15) in $\mathcal{D}$. In this region, the model can be considered as being epidemiologically and mathematically well-posed (Hethcote, 2000). Thus, every solution of the model (3.15) with initial conditions in $\mathcal{D}$ remains in $\mathcal{D}$ for all time $t \geq 0$. Therefore, the $\omega-$limit sets of the system (3.15) are contained in $\mathcal{D}$. Thus result is summarized thus.

Lemma 4.2. *The region* $\mathcal{D} = \mathcal{D}_f \cup \mathcal{D}_m \subset \mathfrak{R}^{10}_+ \times \mathfrak{R}^{10}_+$ *is positively-invariant for the model* (3.15) *with initial conditions in* $\mathfrak{R}^{20}_+$.

4.1.3 Local stability of Disease-Free Equilibrium (DFE) of the co-infection model

we would determine the local stability at disease free equilibruim.

Lemma 4.3. *The DFE* (ξ_0) *of the model* (3.15) *is locally asymptotically stable (LAS) if* $\mathcal{R}_0 < 1$, *and unstable if* $\mathcal{R}_0 > 1$.

The co-infection model (3.15) has a DFE, obtained by setting the right-hand sides of the equations in model (3.15) to zero, given by

$$\begin{aligned}\xi_0 &= (S^*_{\text{M}}, E^*_{\text{MC}}, I^*_{\text{MAC}}, I^*_{\text{MSC}}, R^*_{\text{MC}}, S^*_{\text{F}}, E^*_{\text{FC}}, I_{\text{FAC}}, I^*_{\text{FSC}}, R^*_{\text{FC}}, I^*_{\text{MG}}, R^*_{\text{MG}}, I^*_{\text{FG}}, R^*_{\text{FG}}, E^*_{\text{MCG}}, I^*_{\text{MACG}}, I^*_{\text{MSCG}}, E^*_{\text{FCG}}, I^*_{\text{FACG}}, I^*_{\text{FSCG}}) \\ &= (S^*_{\text{M}}, 0, 0, 0, 0, S^*_{\text{F}}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)\end{aligned} \tag{4.2}$$

with

$$S^*_{\text{M}} = \frac{\Pi_{\text{M}}}{\mu_{\text{H}}} \quad \text{and} \quad S^*_{\text{F}} = \frac{\Pi_{\text{F}}}{\mu_{\text{H}}}$$

The linear stability of the disease free equilibrium, (ξ_0) can be established using the next generation operator method on the system (3.15). Using the notation in van den Driessche and Watmough (2002), the matrix $\mathcal{F}_i$ (of new infections) and the matrix $\mathcal{V}_i$ (of the transfer of individuals between

compartments) are respectively, given by

$$\mathcal{F}_i = \begin{bmatrix} \lambda_{\text{FC}} S_{\text{M}} \\ 0 \\ 0 \\ \lambda_{\text{MC}} S_{\text{F}} \\ 0 \\ 0 \\ \lambda_{\text{FG}} S_{\text{M}} \\ \lambda_{\text{MG}} S_{\text{F}} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad \mathcal{V}_i = \begin{bmatrix} K_1 E_{\text{MC}} - (r_{\text{MC}} \lambda_{\text{FC}} R_{\text{MC}} + \gamma_{\text{MG2}} E_{\text{MCG}} + \lambda_{\text{FC}} R_{\text{MG}}) \\ K_2 I_{\text{MAC}} - (1 - \kappa_{\text{M}}) \rho_{\text{M}} E_{\text{MC}} \\ K_3 I_{\text{MSC}} - (\kappa_{\text{M}} \rho_{\text{M}} E_{\text{MC}} + (1 - \phi_{\text{M}}) \gamma_{\text{M}} I_{\text{MAC}} \\ K_4 E_{\text{FC}} - (r_{\text{FC}} \lambda_{\text{MC}} R_{\text{FC}} + \gamma_{\text{FG2}} E_{\text{FCG}} + \lambda_{\text{MC}} R_{\text{FG}}) \\ K_5 I_{\text{FAC}} - (1 - \kappa_{\text{F}}) \rho_{\text{F}} E_{\text{FC}} \\ K_6 I_{\text{FSC}} - (\kappa_{\text{F}} \rho_{\text{F}} E_{\text{FC}} + (1 - \phi_{\text{F}}) \gamma_{\text{F}} I_{\text{FAC}} \\ K_7 I_{\text{MG}} - (\lambda_{\text{FG}} R_{\text{MC}} + r_{\text{MG}} \lambda_{\text{FG}} R_{\text{MG}} + \phi_{\text{MC}} \gamma_{\text{M2}} I_{\text{MACG}}) \\ K_8 I_{\text{FG}} - (r_{\text{FG}} \lambda_{\text{MG}} R_{\text{FG}} + \phi_{\text{FC2}} \gamma_{\text{F2}} I_{\text{FACG}}) \\ K_9 E_{\text{MCG}} - (\lambda_{\text{FG}} E_{\text{MC}} + \chi_{\text{MG}} \lambda_{\text{FC}} I_{\text{MG}}) \\ K_{10} I_{\text{MACG}} - (\eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MAC}} + (1 - \kappa_{\text{M2}}) \rho_{\text{M2}} E_{\text{MCG}}) \\ K_{11} I_{\text{MSCG}} - (\eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MSC}} + \kappa_{\text{M2}} \rho_{\text{M2}} E_{\text{MCG}} + (1 - \phi_{\text{MC2}} \gamma_{\text{M2}} I_{\text{MACG}})) \\ K_{12} E_{\text{FCG}} - (\lambda_{\text{MG}} E_{\text{FC}} + \chi_{\text{MG}} \lambda_{\text{MC}} I_{\text{FG}}) \\ K_{13} I_{\text{FACG}} - (\eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FAC}} + (1 - \kappa_{\text{F2}}) \rho_{\text{F2}} E_{\text{FCG}}) \\ K_{14} I_{\text{FSCG}} - (\eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FSC}} + \kappa_{\text{F2}} \rho_{\text{F2}} E_{\text{FCG}} + (1 - \phi_{\text{FC2}} \gamma_{\text{F2}} I_{\text{FACG}})) \end{bmatrix}$$

The matrices F and V, for the new infection terms and the remaining transfer terms, evaluated at the disease free equilibrium (DFE) are, respectively, given by

$$F = \begin{bmatrix} F_{11} & F_{12} \\ F_{21} & F_{22} \end{bmatrix},$$

where

$$F_{11} = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\beta_{\text{FC}} S^*_{\text{M}}}{N^*_{\text{HF}}} & \frac{\beta_{\text{FC}} \eta_{\text{F1}} S^*_{\text{M}}}{N^*_{\text{HF}}} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{\text{MC}} S^*_{\text{F}}}{N^*_{\text{HM}}} & \frac{\beta_{\text{MC}} \eta_{\text{M1}} S^*_{\text{F}}}{N^*_{\text{HM}}} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$F_{12} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & \frac{\beta_{\mathrm{FC}}\omega_{\mathrm{FG}}S_{\mathrm{M}}^*}{N_{\mathrm{HF}}^*} & \frac{\beta_{\mathrm{FC}}\omega_{\mathrm{FG}}\eta_{\mathrm{F}2}S_{\mathrm{M}}^*}{N_{\mathrm{HF}}^*} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_{\mathrm{MC}}\omega_{\mathrm{MG}}S_{\mathrm{F}}^*}{N_{\mathrm{HM}}^*} & \frac{\beta_{\mathrm{MC}}\omega_{\mathrm{MG}}\eta_{\mathrm{M}2}S_{\mathrm{F}}^*}{N_{\mathrm{HM}}^*} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_{\mathrm{FG}}S_{\mathrm{M}}^*}{N_{\mathrm{HF}}^*} & 0 & 0 & 0 & \frac{\beta_{\mathrm{FG}}S_{\mathrm{M}}^*}{N_{\mathrm{HF}}^*} & \frac{\beta_{\mathrm{FG}}\omega_{\mathrm{FC}}S_{\mathrm{M}}^*}{N_{\mathrm{HF}}^*} & \frac{\beta_{\mathrm{FG}}\omega_{\mathrm{FC}}\eta_{\mathrm{F}2}S_{\mathrm{M}}^*}{N_{\mathrm{HF}}^*} \end{bmatrix}$$

$$F_{21} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{\mathrm{MG}}S_{\mathrm{F}}^*}{N_{\mathrm{HM}}^*} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$F_{22} = \begin{bmatrix} 0 & \frac{\beta_{\mathrm{MG}}S_{\mathrm{F}}^*}{N_{\mathrm{HM}}^*} & \frac{\beta_{\mathrm{MG}}\omega_{\mathrm{MC}}S_{\mathrm{F}}^*}{N_{\mathrm{HM}}^*} & \frac{\beta_{\mathrm{MG}}\omega_{\mathrm{MC}}\eta_{\mathrm{M}2}S_{\mathrm{F}}^*}{N_{\mathrm{HM}}^*} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix}
K_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -G_1 & 0 & 0 & 0 & 0 & 0 \\
-G_2 & K_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
-G_3 & -G_4 & K_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & K_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -G_5 & 0 & 0 \\
0 & 0 & 0 & -G_6 & K_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -G_7 & -G_8 & K_6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & K_7 & 0 & 0 & -G_9 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & K_8 & 0 & 0 & 0 & 0 & -G_{10} & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & K_9 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -G_{11} & K_{10} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -G_{12} & -G_{13} & K_{11} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & K_{12} & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -G_{14} & K_{13} & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -G_{15} & -G_{16} & K_{14}
\end{bmatrix}$$

where

$K_1 = \mu_{\text{H}} + \rho_{\text{M}}$, $K_2 = \mu_{\text{H}} + \delta_{\text{MC}} + \gamma_{\text{M1}}$,$K_3 = \mu_{\text{H}} + \delta_{\text{MC}} + \theta_{\text{MC}}$,$K_4 = \mu_{\text{H}} + \rho_{\text{F}}$,$K_5 = \mu_{\text{H}} + \delta_{\text{FC}} + \gamma_{\text{F1}}$, $K_6 = \mu_{\text{H}} + \delta_{\text{FC}} + \theta_{\text{FC}}$,$K_7 = \mu_{\text{H}} + \phi_{\text{MG1}} + \delta_{\text{MG1}}$,$K_8 = \mu_{\text{H}} + \phi_{\text{FG1}}$,$K_9 = \mu_{\text{H}} + \delta_{\text{MG2}} + \gamma_{\text{MG2}} + \rho_{\text{M2}}$,$K_{10} = \mu_{\text{H}} + \delta_{\text{MC2}} + \delta_{\text{MG2}} + \gamma_{\text{M2}}$,$K_{11} = \mu_{\text{H}} + \delta_{\text{MC2}} + \delta_{\text{MG2}}$,$K_{12} = \mu_{\text{H}} + \delta_{\text{FG2}} + \gamma_{\text{FG2}} + \rho_{\text{F2}}$,$K_{13} = \mu_{\text{H}} + \delta_{\text{FC2}} + \delta_{\text{FG2}} + \gamma_{\text{F2}}$,$K_{14} = \mu_{\text{H}} + \delta_{\text{FC2}} + \delta_{\text{FG2}}$, $G_1 = \gamma_{\text{MG2}}$, $G_2 = \rho_{\text{M}}(1 - \kappa_{\text{M}})$, $G_3 = \kappa_{\text{M}}\rho_{\text{M}}$, $G_4 = \gamma_{\text{M}}(1 - \phi_{\text{M}})$, $G_5 = \gamma_{\text{FG2}}$, $G_6 = \rho_{\text{F}}(1 - \kappa_{\text{F}})$, $G_7 = \kappa_{\text{F}}\rho_{\text{F}}$, $G_8 = \gamma_{\text{F}}(1 - \phi_{\text{F}})$, $G_9 = \phi_{\text{MC}}\gamma_{\text{M2}}$, $G_{10} = \phi_{\text{FC2}}\gamma_{\text{F2}}$, $G_{11} = \rho_{\text{M2}}(1 - \kappa_{\text{M2}})$, $G_{12} = \kappa_{\text{M2}}\rho_{\text{M2}}$, $G_{13} = \gamma_{\text{M2}}(1 - \phi_{\text{MC2}})$, $G_{14} = \rho_{\text{F2}}(1-\kappa_{\text{F2}})$, $G_{15} = \kappa_{\text{F2}}\rho_{\text{F2}}$, $G_{16} = \gamma_{\text{F2}}(1-\phi_{\text{FC2}})$, $M_1 = \mu_{\text{H}} + \phi_{MG1} + \delta_{MG1}$, $M_2 = \mu_{\text{H}} + \phi_{FG1} + \delta_{FG1}$

Hence, it follows from van den Driessche and Watmough (2002) that the basic reproduction number of the model (3.15), denoted by $\mathcal{R}_0$, is given by (where ρ is the spectral radius) $\mathcal{R}_0 = \rho(FV^{-1}) = \max\{\mathcal{R}_{0G}, \mathcal{R}_{0C}\}$ where $\mathcal{R}_{0G}$ and $\mathcal{R}_{0C}$ are the associated reproduction numbers respectively, given by

$$\mathcal{R}_{0\text{G}} = \sqrt{\frac{\beta_{\text{FG}}\beta_{\text{MG}}}{K_7K_8}},$$

$$\mathcal{R}_{0\text{C}} = \sqrt{\frac{\beta_{\text{FC}}\beta_{\text{MC}}\rho_{\text{F}}\rho_{\text{M}}[(Q_1 + \eta_{\text{F1}}(\kappa_{\text{F}}K_5 + \gamma_{\text{F}}(1 - \kappa_{\text{F}})(1 - \phi_{\text{F}})))(Q_2 + \eta_{\text{M1}}(\kappa_{\text{M}}K_2 + \gamma_{\text{M}}(1 - \kappa_{\text{M}})(1 - \phi_{\text{M}})))]}{K_1K_2K_3K_4K_5K_6}}$$

where

$Q_1 = K_6(1-\kappa_{\text{F}})$, $Q_2 = K_3(1-\kappa_{\text{M}})$,

The result follows from Theorem 2 in van den Driessche and Watmough (2002). The threshold quantity, $\mathcal{R}_0 = \max\{\mathcal{R}_{0G}, \mathcal{R}_{0C}\}$, is the basic reproduction number of the disease Anderson and May (1982), Anderson and May (1991) and Hethcote,(2000). It represents the average number of secondary cases generated by a typical infected individual in a completely susceptible population van den Driessche and Watmough, (2002). The epidemiological implication of Lemma 4.3 is that when $\mathcal{R}_0$ is less than unity, a small flux of infected individuals into the community will not generate large outbreak, and the disease will die out. In other words, the disease can be effectively controlled in the community if the initial sizes of the sub-populations of the model (3.15) are in the basin of attraction of the DFE (ξ_0).

4.1.4 Global Stability of Disease -free Equilibrium

in this section, to investigate the global asymptotic stability of the disease -free equlibrium we write system equation (3.15) as

$$\begin{aligned} \frac{dX}{dt} &= F(X, I) \\ \frac{dI}{dt} &= G(X, I), G(X, 0) = 0 \end{aligned} \tag{4.3}$$

where $X \in R^m$ denotes the number of non-infectious individuals and $I \in R^n$ denotes the number of infected individuals. Let $U_0 = (X^*, 0)$ denote the disease free equilibrium of the system Furthermore, suppose the following assumptions are true: The conditions $(H1)$ and $(H2)$ below must be met to guarantee local asymptotic stability:

$(H1)$: For $\frac{dX}{dt} = F(X, 0), X^*$is globally asymptotically stable (GAS),

$(H2)$: $G(X, I) = AI - \hat{G}(X, I), G(X, I) \geq 0$ for $(X, I) \in A$,

where $A = D_1 G(X^*, 0)$ is an M-matrix (the off-diagonal elements of A are nonnegative)

Theorem 4.4. *The fixed point $U_0 = (X^*, 0)$ is globally asymptotic stable equilibruim of the model equation* (3.15) *provided that $R_0 \leq 1$ and that assuptions (H_1) and (H_2) hold.*

Proof: To prove this we write (3.15) in the form of (4.3) that is

$$\begin{aligned} X &= (S_{\text{M}}, , R_{\text{MC}}, S_{\text{F}}, R_{\text{FC}}, R_{\text{MG}}, R_{\text{FG}}.) \\ I &= (E_{\text{MC}}, , I_{\text{MAC}}, I_{\text{MSC}}, I_{\text{FAC}}, I_{\text{FSC}}, I_{\text{MG}}, I_{\text{FG}}, E_{\text{MCG}}, I_{\text{MACG}}, I_{\text{MSCG}}, E_{\text{FCG}}, I_{\text{FACG}}, I_{\text{FSCG}}.) \end{aligned} \tag{4.4}$$

$$F(X,I) = \begin{bmatrix} \Pi_M - \lambda_{\text{FC}} S_{\text{M}} - \lambda_{\text{FG}} S_{\text{M}} - \mu_{\text{H}} S_{\text{M}} \\ \theta_{MC} I_{\text{MSC}} + \phi_{MC} \gamma_{M1} I_{\text{MAC}} - \mu_{\text{H}} R_{\text{MC}} - r_{\text{FC}} \lambda_{\text{FC}} R_{\text{MC}} \\ \Pi_F - \lambda_{\text{MC}} S_{\text{F}} - \lambda_{\text{MG}} S_{\text{F}} - \mu_{\text{H}} S_G F \\ \theta_{FC} I_{\text{FSC}} + \phi_{FC} \gamma_{F1} I_{\text{FAC}} - \mu_{\text{H}} R_{\text{FC}} - r_{\text{FC}} \lambda_{\text{MC}} R_{\text{FC}} \\ \phi_{MG1} I_{\text{MG}} - \mu_{\text{H}} R_{\text{MG}} - \lambda_{\text{FC}} R_{\text{MG}} - r_{\text{MG}} \lambda_{\text{FG}} R_{\text{MG}} \\ \phi_{FG1} I_{\text{FG}} - \mu_{\text{H}} R_{\text{FG}} - \lambda_{\text{MC}} R_{\text{MG}} - r_{\text{FG}} \lambda_{\text{MG}} R_{\text{FG}} \end{bmatrix}$$

$$F(X,0) = \begin{bmatrix} \Pi_M - \mu_{\text{H}} S_{\text{M}} \\ 0 \\ \Pi_F - \mu_{\text{H}} S_{\text{F}} \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

$$G(X,I)=\begin{bmatrix}\lambda_{\text{FC}}S_{\text{M}}+r_{\text{MC}}\lambda_{\text{FC}}R_{\text{MC}}-\lambda_{\text{FG}}E_{\text{MC}}-K_1E_{\text{MC}}+G_1E_{\text{MCG}}+\lambda_{\text{FC}}R_{\text{MG}}\\ G_2E_{\text{MC}}-K_2I_{\text{MAC}}-\eta_{\text{MC}}\lambda_{\text{FG}}I_{\text{MAC}}\\ G_3E_{\text{MC}}+G_4I_{\text{MAC}}-K_3I_{\text{MSC}}-\eta_{\text{MC}}\lambda_{\text{FG}}I_{\text{MSC}}\\ \lambda_{\text{MC}}S_{\text{F}}+r_{\text{FC}}\lambda_{\text{MC}}R_{\text{FC}}-K_4E_{\text{FC}}-\lambda_{\text{MG}}E_{\text{FC}}+G_5E_{\text{FCG}}+\lambda_{\text{MC}}R_{\text{FG}}\\ G_6E_{\text{FC}}-K_5I_{\text{FAC}}-\eta_{\text{FC}}\lambda_{\text{MG}}I_{\text{FAC}}\\ G_7E_{\text{FC}}+G_8I_{\text{FAC}}-K_6I_{\text{FSC}}-\eta_{\text{FC}}\lambda_{\text{MG}}I_{\text{FSC}}\\ \lambda_{\text{FG}}S_{\text{M}}-K_7I_{\text{MG}}+\lambda_{\text{FG}}R_{\text{MC}}-\chi_{\text{MG}}\lambda_{\text{FG}}I_{\text{MG}}+r_{\text{MG}}\lambda_{\text{FG}}R_{\text{MG}}+G_9I_{\text{MACG}}\\ \lambda_{\text{MG}}S_{\text{F}}-K_8I_{\text{FG}}+r_{\text{FG}}\lambda_{\text{MG}}R_{\text{FG}}+G_{10}I_{\text{FACG}}-\chi_{\text{MG}}\lambda_{\text{MC}}I_{\text{FG}}\\ \lambda_{\text{FG}}E_{\text{MC}}+\chi_{\text{MG}}\lambda_{\text{FC}}I_{\text{MG}}-K_9E_{\text{MCG}}\\ \eta_{\text{MC}}\lambda_{\text{FG}}I_{\text{MAC}}+G_{11}E_{\text{MCG}}-K_{10}I_{\text{MACG}}\\ \eta_{\text{MC}}\lambda_{\text{FG}}I_{\text{MSC}}+G_{12}E_{\text{MCG}}-K_{11}I_{\text{MSCG}}+G_{13}I_{\text{MACG}}\\ \lambda_{\text{MG}}E_{\text{FC}}+\chi_{\text{MG}}\lambda_{\text{MC}}I_{\text{FG}}-K_{12}E_{\text{FCG}}\\ \eta_{\text{FC}}\lambda_{\text{MG}}I_{\text{FAC}}+G_{14}E_{\text{FCG}}-K_{13}I_{\text{FACG}}\\ \eta_{\text{FC}}\lambda_{\text{MG}}I_{\text{FSC}}+G_{15}E_{\text{FCG}}+G_{16}I_{\text{FACG}}-K_{14}I_{\text{FSCG}}\end{bmatrix}$$

$$A=\begin{bmatrix}A_{11} & A_{12}\\ A_{21} & A_{22}\end{bmatrix},$$

where

$$A_{11}=\begin{bmatrix}-K_1 & 0 & 0 & 0 & \frac{\beta_{\text{FC}}S_M^*}{N_{\text{HF}}^*} & \frac{\beta_{\text{FC}}\eta_{F1}S_M^*}{N_{\text{HF}}^*} & 0\\ G_2 & -K_2 & 0 & 0 & 0 & 0 & 0\\ G_3 & G_4 & -K_3 & 0 & 0 & 0 & 0\\ 0 & \frac{\beta_{\text{MC}}S_F^*}{N_{\text{HM}}^*} & \frac{\beta_{\text{MC}}\eta_{M1}S_F^*}{N_{\text{HM}}^*} & -K_4 & 0 & 0 & 0\\ 0 & 0 & 0 & G_6 & -K_5 & 0 & 0\\ 0 & 0 & 0 & G_7 & G_8 & -K_6 & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & -K_7\end{bmatrix}$$

$$A_{12} = \begin{bmatrix} 0 & G_1 & 0 & 0 & 0 & \frac{H_4 S_M^*}{N_{\text{HF}}^*} & \frac{H_4 \eta_{F1} S_M^*}{N_{\text{HF}}^*} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{H_1 S_F^*}{N_{\text{HM}}^*} & \frac{H_1 \eta_{M2} S_F^*}{N_{\text{HM}}^*} & G_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_{\text{FG}} S_M^*}{N_{\text{HF}}^*} & 0 & G_9 & 0 & \frac{\beta_{\text{FG}} S_M^*}{N_{\text{HF}}^*} & \frac{H_3 S_M^*}{N_{\text{HF}}^*} & \frac{H_3 \eta_{F2} S_M^*}{N_{\text{HF}}^*} \end{bmatrix}$$

$$A_{21} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{\text{MG}} S_F^*}{N_{\text{HM}}^*} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$A_{22} = \begin{bmatrix} -K_8 & \frac{\beta_{\text{MG}} S_F^*}{N_{\text{HM}}^*} & \frac{H_2 S_F^*}{N_{\text{HM}}^*} & \frac{H_2 \eta_{M2} S_F^*}{N_{\text{HM}}^*} & 0 & G_{10} & 0 \\ 0 & -K_9 & 0 & 0 & 0 & 0 & 0 \\ 0 & G_{11} & -K_{10} & 0 & 0 & 0 & 0 \\ 0 & G_{12} & G_{13} & -K_{11} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -K_{12} & 0 & 0 \\ 0 & 0 & 0 & 0 & G_{14} & -K_{13} & 0 \\ 0 & 0 & 0 & 0 & G_{15} & G_{16} & -K_{14} \end{bmatrix}$$

where

$$AI = \begin{bmatrix} -K_1 E_{\text{MC}} + \frac{\beta_{\text{FC}} S_M^* I_{\text{FAC}}}{N_{\text{HF}}^*} + \frac{\beta_{\text{FC}} \eta_{F1} S_M^* I_{\text{FSC}}}{N_{\text{HF}}^*} + G_1 E_{\text{MCG}} + \frac{H_4 S_M^* I_{\text{FACG}}}{N_{\text{HF}}^*} + \frac{H_4 \eta_{F1} S_M^* I_{\text{FSCG}}}{N_{\text{HF}}^*} \\ G_2 E_{\text{MC}} - K_2 I_{\text{MAC}} \\ G_3 E_{\text{MC}} + G_4 I_{\text{MAC}} - K_3 I_{\text{MSC}} \\ \frac{\beta_{\text{MC}} S_F^* I_{\text{MAC}}}{N_{\text{HM}}^*} + \frac{\beta_{\text{MC}} \eta_{M1} S_F^* I_{\text{MSC}}}{N_{\text{HM}}^*} + -K_4 E_{\text{FC}} + \frac{H_1 S_F^* I_{\text{MACG}}}{N_{\text{HM}}^*} + \frac{H_1 \eta_{M2} S_F^* I_{\text{MSCG}}}{N_{\text{HM}}^*} + G_5 E_{\text{FCG}} \\ G_6 E_{\text{FC}} - K_5 I_{\text{FAC}} \\ G_7 E_{\text{FC}} + G_8 I_{\text{FAC}} - K_6 I_{\text{FSC}} \\ -K_7 I_{\text{MG}} + \frac{\beta_{\text{FG}} S_M^* I_{\text{FG}}}{N_{\text{HF}}^*} + G_9 I_{\text{MACG}} + \frac{\beta_{\text{FG}} S_M^* E_{\text{FCG}}}{N_{\text{HF}}^*} + \frac{H_3 S_M^* I_{\text{FACG}}}{N_{\text{HF}}^*} + \frac{H_3 \eta_{F2} S_M^* I_{\text{FSCG}}}{N_{\text{HF}}^*} \\ \frac{\beta_{\text{MG}} S_F^* I_{\text{MG}}}{N_{\text{HM}}^*} + -K_8 I_{\text{FG}} + \frac{\beta_{\text{MG}} S_F^* E_{\text{MCG}}}{N_{\text{HM}}^*} + \frac{H_2 S_F^* I_{\text{MACG}}}{N_{\text{HM}}^*} + \frac{H_2 \eta_{M2} S_F^* I_{\text{MSCG}}}{N_{\text{HM}}^*} + G_{10} I_{\text{FACG}} \\ -K_9 E_{\text{MCG}} \\ G_{11} E_{\text{MCG}} - K_{10} I_{\text{MACG}} \\ G_{12} E_{\text{MCG}} + G_{13} I_{\text{MACG}} - K_{11} I_{\text{MSCG}} \\ -K_{12} E_{\text{FCG}} \\ G_{14} E_{\text{FCG}} - K_{13} I_{\text{FACG}} \\ G_{15} E_{\text{FCG}} + G_{16} I_{\text{FACG}} - K_{14} I_{\text{FSCG}} \end{bmatrix}$$

and

$$\hat{G}(X, I) = AI - G(X, I) = \begin{bmatrix} -\lambda_{\text{FC}}(r_{\text{MC}} R_{\text{MC}} + R_{\text{MG}}) + \lambda_{\text{FG}} E_{\text{MC}} \\ \eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MAC}} \\ \eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MSC}} \\ -\lambda_{\text{MC}}(r_{\text{FC}} R_{\text{FC}} + R_{\text{FG}}) + \lambda_{\text{MG}} E_{\text{FC}} \\ \eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FAC}} \\ \eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FSC}} \\ -\lambda_{\text{FG}}(R_{\text{MC}} + \chi_{\text{MG}} I_{\text{MG}} - r_{\text{MG}} R_{\text{MG}}) \\ -\lambda_{\text{MG}} r_{\text{FG}} R_{\text{FG}} + \chi_{\text{MG}} \lambda_{\text{MC}} I_{\text{FG}} \\ -\lambda_{\text{FG}} E_{\text{MC}} - \chi_{\text{MG}} \lambda_{\text{FC}} I_{\text{MG}} \\ -\eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MAC}} \\ -\eta_{\text{MC}} \lambda_{\text{FG}} I_{\text{MSC}} \\ -\lambda_{\text{MG}} E_{\text{FC}} - \chi_{\text{MG}} \lambda_{\text{MC}} I_{\text{FG}} \\ -\eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FAC}} \\ -\eta_{\text{FC}} \lambda_{\text{MG}} I_{\text{FSC}} \end{bmatrix}$$

it can be clearly seen that $G(X, I) \geq 0$. This implies that, the disease-free equilibrium point is globally asymptotically stable for $R_{MC} \leq 1$, $R_{FC} \leq 1$, $R_{MG} \leq 1$, $R_{FG} \leq 1$. epidemiologically, the disease would eradicate from the population in the long run.

4.1.5 Backward bifurcation analysis

It is instructive to characterize the type of bifurcation the model (3.15) may undergo. We claim the following result:

Theorem 4.5. *he model* (3.15) *exhibits backward bifurcation at* $\mathcal{R}_0 = 1$ *whenever a bifurcation coefficient, denoted by a (given by* (4.6)*), is positive.*

Proof Suppose

$$\xi_e = (S_M^{**}, E_{MC}^{**}, I_{MAC}^{**}, I_{MSC}^{**}, R_{MC}^{**}, S_F^{c**}, E_{FC}^{**}, I_{FAC}^{**}, I_{FSC}^{**}, R_{FC}^{**}, I_{MG}^{**}, R_{MG}^{**}, I_{FG}^{**}, R_{FG}^{**}, E_{MCG}^{**}, I_{MACG}^{**}, I_{MSCG}^{**}, E_{FCG}^{**}, I_{FACG}^{**}, I_{FSCG}^{**},)$$

represents any arbitrary endemic equilibrium of the model (that is, an endemic equilibrium in which at least one of the infected components is non-zero). The existence (or otherwise) of backward bifurcation will be explored using the Centre Manifold Theory Castillo-Chavez and Song (2004). To apply this theory, it is necessary to carry out the following change of variables. Let

$$S_M = x_1, E_{MC} = x_2, I_{MAC} = x_3, I_{MSC} = x_4, R_{MC} = x_5, S_F = x_6, E_{FC} = x_7, I_{FAC} = x_8, I_{FSC} = x_9,$$
$$R_{FC} = x_{10}, I_{MG} = x_{11}, R_{MG} = x_{12}, I_{FG} = x_{13}, R_{FG} = x_{14}, E_{MCG} = x_{15}, I_{MACG} = x_{16}, I_{MSCG} = x_{17},$$
$$E_{FCG} = x_{18}, I_{FACG} = x_{19}, I_{FSCG} = x_{20}$$

so that

$$N = \sum_{i=1}^{20} x_i.$$

Further, using the vector notation

$$X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8, x_9, x_{10}, x_{11}, x_{12}, x_{13}, x_{14}, x_{15}, x_{16}, x_{17}, x_{18}, x_{19}, x_{20})^T$$

the model can be re-written in the form

$$\frac{dX}{dt} = f = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9, f_{10}, f_{11}, f_{12}, f_{13}, f_{14}, f_{15}, f_{16}, f_{17}, f_{18}, f_{19}, f_{20})^T$$

as follows:

$$\begin{aligned}
\frac{dx_1}{dt} &\equiv f_1 = \Pi_M - (\lambda_{FC} + \lambda_{FG} + \mu_{\text{н}})\, x_1 \\
\frac{dx_2}{dt} &\equiv f_2 = \lambda_{FC}x_1 + r_{\text{мс}}\lambda_{\text{фс}}x_5 + \gamma_{MG2}x_{15} + \lambda_{\text{фс}}x_{12} - (\lambda_{FG} + \mu_{\text{н}} + \rho_M)\, x_2 \\
\frac{dx_3}{dt} &\equiv f_3 = (1-\kappa_M)\rho_M x_2 - \eta_{\text{мс}}\lambda_{\text{фс}}x_3 - (\mu_{\text{н}} + \delta_{MC} + \gamma_{M1})\, x_3 \\
\frac{dx_4}{dt} &\equiv f_4 = \kappa_M\rho_M x_2 + (1-\phi_{MC})\gamma_{M1}x_3 - \eta_{\text{мс}}\lambda_{\text{фг}}x_4 - (\mu_{\text{н}} + \delta_{MC} + \theta_{MC})\, x_4 \\
\frac{dx_5}{dt} &\equiv f_5 = \theta_{MC}x_4 + \phi_{MC}\gamma_{M1}x_3 - \mu_{\text{н}}x_5 - r_{\text{мс}}\lambda_{\text{фс}}x_5 - \lambda_{\text{фг}}x_5 \\
\frac{dx_6}{dt} &\equiv f_6 = \Pi_F - \lambda_{\text{мс}}x_6 - \mu_{\text{н}}x_6 - \lambda_{\text{мг}}x_6 \\
\frac{dx_7}{dt} &\equiv f_7 = \lambda_{\text{мс}}x_6 + r_{\text{фс}}\lambda_{\text{мс}}x_{10} - (\mu_{\text{н}} + \rho_F)x_7 - \lambda_{\text{мг}}x_7 + \gamma_{FG2}x_{18} + \lambda_{\text{мс}}x_{14} \\
\frac{dx_8}{dt} &\equiv f_8 = (1-\kappa_F)\rho_F x_7 - (\mu_{\text{н}} + \delta_{FC} + \gamma_{F1})x_8 - \eta_{\text{фс}}\lambda_{\text{мг}}x_8 \\
\frac{dx_9}{dt} &\equiv f_9 = \kappa_F\rho_F x_7 + (1-\phi_{FC})\gamma_{F1}x_8 - (\mu_{\text{н}} + \delta_{FC} + \theta_{FC})x_9 - \eta_{\text{фс}}\lambda_{\text{мг}}x_9 \\
\frac{dx_{10}}{dt} &\equiv f_{10} = \theta_{FC}x_9 + \phi_{FC}\gamma_{F1}x_8 - (\mu_{\text{н}} + r_{\text{фс}}\lambda_{\text{мс}})x_{10} \\
\frac{dx_{11}}{dt} &\equiv f_{11} = \lambda_{\text{фг}}x_1 - (\mu_{\text{н}} + \phi_{MG1} + \delta_{MG1})x_{11} + \lambda_{\text{фг}}x_5 - \chi_{\text{мг}}\lambda_{\text{фс}}x_{11} + r_{\text{мг}}\lambda_{\text{фг}}x_{12} + \phi_{MC2}\gamma_{M2}x_{16} \\
\frac{dx_{12}}{dt} &\equiv f_{12} = \phi_{MG1}x_{11} - \mu_{\text{н}}x_{12} - \lambda_{\text{фс}}x_{12} - r_{\text{мг}}\lambda_{\text{фг}}x_{12} \\
\frac{dx_{13}}{dt} &\equiv f_{13} = \lambda_{\text{мг}}x_6 - (\mu_{\text{н}} + \phi_{FG1} + \delta_{FG1})x_{13} - \chi_{\text{мг}}\lambda_{\text{мс}}x_{13} + r_{\text{фг}}\lambda_{\text{мг}}x_{14} + \phi_{FC2}\gamma_{F2}x_{19} \\
\frac{dx_{14}}{dt} &\equiv f_{14} = \phi_{FG1}x_{13} - \mu_{\text{н}}x_{14} - \lambda_{\text{мс}}x_{14} - r_{\text{фс}}\lambda_{\text{мс}}x_{14} \\
\frac{dx_{15}}{dt} &\equiv f_{15} = \lambda_{\text{фг}}x_2 + \chi_{\text{мг}}\lambda_{\text{фс}}x_{11} - (\mu_{\text{н}} + \delta_{MG2} + \gamma_{MG2} + \rho_{M2})\, x_{15} \\
\frac{dx_{16}}{dt} &\equiv f_{16} = \eta_{\text{мс}}\lambda_{\text{фг}}x_3 + (1-\kappa_{M2})\rho_{M2}x_{15} - (\mu_{\text{н}} + \delta_{MC2} + \delta_{MG2} + \gamma_{M2})x_{16} \\
\frac{dx_{17}}{dt} &\equiv f_{17} = \eta_{\text{мс}}\lambda_{\text{фг}}x_4 + \kappa_{M2}\rho_{M2}x_{15} - (\mu_{\text{н}} + \delta_{MC2} + \delta_{MG2})x_{17} + (1-\phi_{MC2})\gamma_{M2}x_{16} \\
\frac{dx_{18}}{dt} &\equiv f_{18} = \lambda_{\text{мг}}x_7 + \chi_{\text{мг}}\lambda_{\text{мс}}x_{13} - (\mu_{\text{н}} + \delta_{FG2} + \gamma_{FG2} + \rho_{F2})\, x_{18} \\
\frac{dx_{19}}{dt} &\equiv f_{19} = \eta_{\text{фс}}\lambda_{\text{мг}}x_8 + (1-\kappa_{F2})\rho_{F2}x_{18} - (\mu_{\text{н}} + \delta_{FC2} + \delta_{FG2} + \gamma_{F2})\, x_{19} \\
\frac{dx_{20}}{dt} &\equiv f_{20} = \eta_{\text{фс}}\lambda_{\text{мг}}x_9 + \kappa_{F2}\rho_{F2}x_{18} + (1-\phi_{FC2})\gamma_{F2}x_{19} - (\mu_{\text{н}} + \delta_{FC2} + \delta_{FG2})\, x_{20}
\end{aligned} \tag{4.5}$$

where,

$$\lambda_{FC} = \frac{\beta_{\text{FC}}((x_8 + \eta_{F1}x_9 + \omega_{FG}(x_{19} + \eta_{F2}x_{20})}{\sum_{i=6}^{10} x_i + \sum_{i=13}^{14} x_i + \sum_{i=18}^{20} x_i}$$
$$\lambda_{MC} = \frac{\beta_{\text{MC}}((x_3 + \eta_{M1}x_4 + \omega_{MG}(x_{16} + \eta_{M2}x_{17})}{\sum_{i=1}^{5} x_i + \sum_{i=11}^{12} x_i + \sum_{i=15}^{17} x_i}$$
$$\lambda_{FG} = \frac{\beta_{\text{FG}}((x_{13} + x_{18} + \omega_{FC}(x_{19} + \eta_{F2}x_{20})}{\sum_{i=6}^{10} x_i + \sum_{i=13}^{14} x_i + \sum_{i=18}^{20} x_i}$$
$$\lambda_{MG} = \frac{\beta_{\text{MG}}((x_{11} + x_{15} + \omega_{MC}(x_{16} + \eta_{M2}x_{17})}{\sum_{i=1}^{5} x_i + \sum_{i=11}^{12} x_i + \sum_{i=15}^{17} x_i}$$

The Jacobian of the transformed system evaluated at the DFE (ξ_0) is given by

$$J(\xi_0) = \begin{bmatrix} J_{11} & J_{12} \\ 0_{10\times 10} & J_{22} \end{bmatrix}$$

where

$$J_{11} = \begin{bmatrix}
-\mu_{\text{H}} & 0 & 0 & 0 & 0 & 0 & 0 & \frac{-\beta_{\text{FC}}x_1^*}{N_{\text{HF}}^*} & \frac{-\beta_{\text{FC}}\eta_{F1}x_1^*}{N_{\text{HF}}^*} & 0 \\
0 & -K_1 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{\text{FC}}x_1^*}{N_{\text{HF}}^*} & \frac{\beta_{\text{FC}}\eta_{F1}x_1^*}{N_{\text{HF}}^*} & 0 \\
0 & G_2 & -K_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & G_3 & G_4 & -K_3 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \phi_{MC}\gamma_{M1} & \theta_{MC} & -\mu_{\text{H}} & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \frac{-\beta_{\text{MC}}x_6^*}{N_{\text{HM}}^*} & \frac{-\beta_{\text{MC}}\eta_{M1}x_6^*}{N_{\text{HM}}^*} & 0 & -\mu_{\text{H}} & 0 & 0 & 0 & 0 \\
0 & 0 & \frac{\beta_{\text{MC}}x_6^*}{N_{\text{HM}}^*} & \frac{\beta_{\text{MC}}\eta_{M1}x_6^*}{N_{\text{HM}}^*} & 0 & 0 & -K_4 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & G_6 & -K_5 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & G_7 & G_8 & -K_6 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \phi_{FC}\gamma_{F1} & \theta_{FC} & -\mu_{\text{H}}
\end{bmatrix}$$

$$J_{12} = \begin{bmatrix} 0 & 0 & \frac{-\beta_{\text{FG}} x_1^*}{N_{\text{HF}}^*} & 0 & 0 & 0 & 0 & \frac{-\beta_{\text{FG}} x_1^*}{N_{\text{HF}}^*} & \frac{-(H_4+H_3)x_1^*}{N_{\text{HF}}^*} & \frac{-(H_4+H_3)\eta_{F2} x_1^*}{N_{\text{HF}}^*} \\ 0 & 0 & 0 & 0 & G_1 & 0 & 0 & 0 & \frac{H_4 x_1^*}{N_{\text{HF}}^*} & \frac{H_4 \eta_{F2} x_1^*}{N_{\text{HF}}^*} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{-\beta_{\text{MG}} x_6^*}{N_{\text{HM}}^*} & 0 & 0 & 0 & \frac{-\beta_{\text{MG}} x_6^*}{N_{\text{HM}}^*} & \frac{-(H_1+H_2)x_6}{N_{\text{HM}}} & \frac{-(H_1+H_2)\eta_{M2} x_6^*}{N_{\text{HM}}^*} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{H_1 x_6^*}{N_{\text{HM}}^*} & \frac{H_1 \eta_{M2} x_6^*}{N_{\text{HM}}^*} & G_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$J_{22} = \begin{bmatrix} -M_1 & 0 & \frac{\beta_{\text{FG}} x_1^*}{N_{\text{HF}}^*} & 0 & 0 & G_9 & 0 & \frac{\beta_{\text{FG}} x_1^*}{N_{\text{HF}}^*} & \frac{H_3 x_1^*}{N_{\text{HF}}^*} & \frac{H_3 \eta_{F2} x_1^*}{N_{\text{HF}}^*} \\ \phi_{MG1} & -\mu_{\text{H}} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_{\text{MG}} x_6^*}{N_{\text{HM}}^*} & 0 & -M_2 & 0 & \frac{\beta_{\text{MG}} x_6^*}{N_{\text{HM}}^*} & \frac{H_2 x_6^*}{N_{\text{HM}}^*} & \frac{H_2 \eta_{M2} x_6^*}{N_{\text{HM}}^*} & 0 & 0 & 0 \\ 0 & 0 & \phi_{FG1} & -\mu_{\text{H}} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -K_9 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & G_{11} & -K_{10} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & G_{12} & G_{13} & -K_{11} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -K_{12} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & G_{14} & -K_{13} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & G_{15} & G_{16} & -K_{14} \end{bmatrix}$$

where, $N_{\text{HM}}^* = x_1^*$, $N_{\text{HF}}^* = x_6^*$

$H_1 = \beta_{MC}\omega_{MG}$, $H_2 = \beta_{MG}\omega_{MC}$, $\quad H_3 = \beta_{FG}\omega_{FC}$, $H_4 = \beta_{FC}\omega_{FG}$.

It can be shown that the Jacobian has a right eigenvector (associated with the zero eigenvalue) given by

$$w = \left[\omega_1, \omega_2, \omega_3, \omega_4, \omega_5, \omega_6, \omega_7, \omega_8, \omega_9, \omega_{10}, \omega_{11}, \omega_{12}, \omega_{13}, \omega_{14}, \omega_{15}, \omega_{16}, \omega_{17}, \omega_{18}, \omega_{19}, \omega_{20}\right]^T$$

with,

$$-\mu_{\text{H}}\omega_1 - \frac{\beta_{\text{FC}}x_1^*}{N_{\text{HF}}^*}\omega_8 - \frac{\beta_{\text{FC}}\eta_{F1}x_1^*}{N_{\text{HF}}^*}\omega_9 - \frac{\beta_{\text{FG}}x_1^*}{N_{\text{HF}}^*}\omega_{13} - \frac{\beta_{\text{FG}}x_1^*}{N_{\text{HF}}^*}\omega_{18} - \frac{(H_4+H_3)x_1^*}{N_{\text{HF}}^*}\omega_{19} - \frac{(H_4+H_3)\eta_{F2}x_1^*}{N_{\text{HF}}^*}\omega_{20} = 0$$

$$-K_1\omega_2 + \frac{\beta_{\text{FC}}x_1^*}{N_{\text{HF}}^*}\omega_8 + \frac{\beta_{\text{FC}}\eta_{F1}x_1^*}{N_{\text{HF}}^*}\omega_9 + G_1\omega_{15} + \frac{H_4x_1^*}{N_{\text{HF}}^*}\omega_{19} + \frac{H_4\eta_{F2}x_1^*}{N_{\text{HF}}^*}\omega_{20} = 0$$

$$G_2\omega_2 - K_2\omega_3 = 0$$

$$G_3\omega_2 + G_4\omega_3 - K_3\omega_4 = 0$$

$$\phi_{MC}\gamma_{M1}\omega_3 + \theta_{MC}\omega_4 - \mu_{\text{H}}\omega_5 = 0$$

$$-\frac{\beta_{\text{MC}}x_6^*}{N_{\text{HM}}^*}\omega_3 - \frac{\beta_{\text{MC}}\eta_{M1}x_6^*}{N_{\text{HM}}^*}\omega_4 - \mu_{\text{H}}\omega_6 - \frac{\beta_{\text{MG}}x_6^*}{N_{\text{HM}}^*}\omega_{11} - \frac{\beta_{\text{MG}}x_6^*}{N_{\text{HM}}^*}\omega_{15} - \frac{(H_1+H_2)x_6^*}{N_{\text{HM}}^*}\omega_{16} - \frac{(H_1+H_2)\eta_{M2}x_6^*}{N_{\text{HM}}^*}\omega_{17} = 0$$

$$\frac{\beta_{\text{MC}}x_6^*}{N_{\text{HM}}^*}\omega_3 + \frac{\beta_{\text{MC}}\eta_{M1}x_6^*}{N_{\text{HM}}^*}\omega_4 - K_4\omega_7 + \frac{H_1x_6^*}{N_{\text{HM}}^*}\omega_{16} + \frac{H_1\eta_{M2}x_6^*}{N_{\text{HM}}^*}\omega_{17} + G_5\omega_{18} = 0$$

$$G_6\omega_7 - K_5\omega_8 = 0$$

$$G_7\omega_7 + G_8\omega_8 - K_6\omega_9 = 0$$

$$\phi_{FC}\gamma_{F1}\omega_8 + \theta_{FC}\omega_9 - \mu_{\text{H}}\omega_{10} = 0$$

$$-M_1\omega_{11} + \frac{\beta_{\text{FG}}x_1^*}{N_{\text{HF}}^*}\omega_{13} - G_9\omega_{16} - \frac{\beta_{\text{FG}}x_1^*}{N_{\text{HF}}^*}\omega_{18} + \frac{H_3x_1^*}{N_{\text{HF}}^*}\omega_{19} + \frac{H_3\eta_{F2}x_1}{N_{\text{HF}}}\omega_{20} = 0$$

$$\phi_{MG1}\omega_{11} - \mu_{\text{H}}\omega_{12} = 0$$

$$\frac{\beta_{\text{MG}}x_6}{N_{\text{HM}}}\omega_{11} - M_2\omega_{13} + \frac{\beta_{\text{MG}}x_6}{N_{\text{HM}}}\omega_{15} + \frac{\beta_{\text{MG}}\omega_{MC}x_6}{N_{\text{HM}}}\omega_{16} + \frac{\beta_{\text{MG}}\omega_{MC}\eta_{M2}x_6}{N_{\text{HM}}}\omega_{17} = 0$$

$$\phi_{FG1}\omega_{13} - \mu_{\text{H}}\omega_{14} = 0$$

$$-K_9\omega_{15} = 0$$

$$G_{11}\omega_{15} - K_{10}\omega_{16} = 0$$

$$G_{12}\omega_{15} + G_{13}\omega_{16} - K_{11}\omega_{17} = 0$$

$$-K_{12}\omega_{18} = 0$$

$$G_{14}\omega_{18} - K_{13}\omega_{19} = 0$$

$$G_{15}\omega_{18} + G_{16}\omega_{19} - K_{14}\omega_{20} = 0$$

where,

$$\omega_1 = \frac{-\left\{\beta_{FC}x_1^*\omega_8 + \beta_{FC}\eta_{F1}x_1^*\omega_9 + \beta_{FG}x_1^*\omega_{13}\right\}}{\mu_{\text{н}}N_{\text{нF}}^*},$$

$$\omega_2 = \omega_2 > 0,$$

$$\omega_3 = \frac{G_2\omega_2}{K_2},$$

$$\omega_4 = \frac{(K_2G_3 + G_2G_4)\omega_2}{K_2K_3},$$

$$\omega_5 = \frac{\left\{K_3G_2\phi_{MC}\gamma_{M1} + \theta_{MC}(K_2G_3 + G_2G_4)\right\}\omega_2}{\mu_{\text{н}}K_2K_3},$$

$$\omega_6 = \frac{-\left\{\beta_{MC}x_6^*\omega_3 + \beta_{MC}\eta_{M1}x_6^*\omega_4 + \beta_{MG}x_6^*\omega_{11}\right\}}{\mu_{\text{н}}N_{\text{нМ}}^*},$$

$$\omega_7 = \frac{\left\{K_3G_2 + \eta_{M1}(K_2G_3 + G_2G_4)\right\}\beta_{MC}x_6^*\omega_2}{K_2K_3K_4N_{\text{нМ}}^*},$$

$$\omega_8 = \frac{\left\{K_3G_2 + \eta_{M1}(K_2G_3 + G_2G_4)\right\}G_6\beta_{MC}x_6^*\omega_2}{K_2K_3K_4K_5N_{\text{нМ}}^*},$$

$$\omega_9 = \frac{\left\{(K_5G_7 + G_6G_8)(K_3G_2 + \eta_{M1}(K_2G_3 + G_2G_4))\right\}\beta_{MC}x_6^*\omega_2}{K_2K_3K_4K_5K_6N_{\text{нМ}}^*},$$

$$\omega_{10} = \frac{\left\{(K_6G_6\phi_{FC}\gamma_{F1} + \theta_{FC}G_7K_5 + \theta_{FC}G_6G_8)(K_3G_2 + \eta_{M1}K_2G_3 + \eta_{M1}G_2G_4)\right\}\beta_{MC}x_6^*\omega_2}{\mu_{\text{н}}K_2K_3K_4K_5K_6N_{\text{нМ}}^*},$$

$$\omega_{11} = \frac{\beta_{FG}x_1^*}{M_1M_2N_{\text{нF}}^*}, \quad \omega_{12} = \frac{\phi_{MG1}\beta_{FG}x_1^*}{\mu_{\text{н}}M_1M_2N_{\text{нF}}^*},$$

$$\omega_{13} = \frac{M_1}{\beta_{FG}\beta_{MG}}, \quad \omega_{14} = \frac{\phi_{FG1}M_1}{\mu_{\text{н}}\beta_{FG}\beta_{MG}},$$

$$\omega_{15} = \omega_{16} = \omega_{17} = \omega_{18} = \omega_{19} = \omega_{19} = \omega_{20} = 0,$$

Furthermore, the corresponding left eigenvector (associated with the zero eigenvalue) is given by

$$\upsilon = [\nu_1, \nu_2, \nu_3, \nu_4, \nu_5, \nu_6, \nu_7, \nu_8, \nu_9, \nu_{10}, \nu_{11}, \nu_{12}, \nu_{13}, \nu_{14}, \nu_{15}, \nu_{16}, \nu_{17}, \nu_{18}, \nu_{19}, \nu_{20}]$$

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$$\upsilon = [\nu_1, \nu_2, \nu_3, \nu_4, \nu_5, \nu_6, \nu_7, \nu_8, \nu_9, \nu_{10}, \nu_{11}, \nu_{12}, \nu_{13}, \nu_{14}, \nu_{15}, \nu_{16}, \nu_{17}, \nu_{18}, \nu_{19}, \nu_{20}]$$

where,

$$-\mu_{\text{н}}\nu_1 = 0$$

$$-K_1\nu_2 + G_2\nu_3 + G_3\nu_4 = 0$$

$$-K_2\nu_3 + G_4\nu_4 + \phi_{MC}\gamma_{M1}\nu_5 - \frac{\beta_{\text{MC}}x_6^*}{N_{\text{HM}}^*}\nu_6 + \frac{\beta_{\text{MC}}x_6^*}{N_{\text{HM}}^*}\nu_7 = 0$$

$$-K_3\nu_4 + \theta_{MC}\nu_5 - \frac{\beta_{\text{MC}}\eta_{M1}x_6^*}{N_{\text{HM}}^*}\nu_6 + \frac{\beta_{\text{MC}}\eta_{M1}x_6^*}{N_{\text{HM}}^*}\nu_7 = 0$$

$$-\mu_{\text{H}}\nu_5 = 0$$

$$-\mu_{\text{H}}\nu_6 = 0$$

$$-K_4\nu_7 + G_6\nu_8 + G_7\nu_9 = 0$$

$$-\frac{\beta_{\text{FC}}x_1^*}{N_{\text{HF}}^*}\nu_1 + \frac{\beta_{\text{FC}}x_1^*}{N_{\text{HF}}^*}\nu_2 - K_5\nu_8 + G_8\nu_9 + \phi_{FC}\gamma_{F1}\nu_{10} = 0$$

$$-\frac{\beta_{\text{FC}}\eta_{F1}x_1^*}{N_{\text{HF}}^*}\nu_1 + \frac{\beta_{\text{FC}}\eta_{F1}x_1^*}{N_{\text{HF}}^*}\nu_2 - K_6\nu_9 + \theta_{FC}\nu_{10} = 0$$

$$-\mu_{\text{H}}\nu_{10} = 0$$

$$-\frac{\beta_{\text{MG}}x_6^*}{N_{\text{HM}}^*}\nu_6 - M_1\nu_{11} + \phi_{MG1}\nu_{12} + \frac{\beta_{\text{MG}}x_6^*}{N_{\text{HM}}^*}\nu_{13} = 0$$

$$-\mu_{\text{H}}\nu_{12} = 0$$

$$-\frac{\beta_{\text{FG}}x_1^*}{N_{\text{HF}}^*}\nu_1 + \frac{\beta_{\text{FG}}x_1^*}{N_{\text{HF}}^*}\nu_{11} - M_2\nu_{13} + \phi_{FG1}\nu_{14} = 0$$

$$-\mu_{\text{H}}\nu_{14} = 0$$

$$G_1\nu_2 - \frac{\beta_{\text{MG}}x_6^*}{N_{\text{HM}}^*}\nu_6 + \frac{\beta_{\text{MG}}x_6^*}{N_{\text{HM}}^*}\nu_{13} - K_9\nu_{15} + G_{11}\nu_{16} + G_{12}\nu_{17} = 0$$

$$\frac{-(H_1+H_2)x_6^*}{N_{\text{HM}}^*}\nu_6 + \frac{H_1x_6^*}{N_{\text{HM}}^*}\nu_7 + G_9\nu_{11} + \frac{H_2x_6^*}{N_{\text{HM}}^*}\nu_{13} - K_{10}\nu_{16} + G_{13}\nu_{17} = 0$$

$$\frac{-(H_1+H_2)\eta_{M2}x_6^*}{N_{\text{HM}}^*}\nu_6 + \frac{H_1\eta_{M2}x_6^*}{N_{\text{HM}}^*}\nu_7 + \frac{H_2\eta_{M2}x_6^*}{N_{\text{HM}}^*}\nu_{13} - K_{11}\nu_{17} = 0$$

$$-\frac{\beta_{\text{FG}}x_1^*}{N_{\text{HF}}^*}\nu_1 + G_5\nu_7 + \frac{\beta_{\text{FG}}x_1^*}{N_{\text{HF}}^*}\nu_{11} - K_{12}\nu_{18} + G_{14}\nu_{19} + G_{15}\nu_{20} = 0$$

$$\frac{-(H_4+H_3)x_1^*}{N_{\text{HF}}^*}\nu_1 + \frac{H_4x_1^*}{N_{\text{HF}}^*}\nu_2 + \frac{H_3x_1^*}{N_{\text{HF}}^*}\nu_{11} - K_{13}\nu_{19} + G_{16}\nu_{20} = 0$$

$$\frac{-(H_4+H_3)\eta_{F2}x_1^*}{N_{\text{HF}}^*}\nu_1 + \frac{H_4\eta_{F2}x_1^*}{N_{\text{HF}}^*}\nu_2 + \frac{H_3\eta_{F2}x_1^*}{N_{\text{HF}}^*}\nu_{11} - K_{14}\nu_{20} = 0$$

Solving, gives

$$
\begin{aligned}
\nu_1 &= \nu_5 = \nu_6 = \nu_{10} = \nu_{12} = \nu_{14} = 0 \\
\nu_2 &= \frac{(G_2G_4\eta_{M1} + G_2K_3 + K_2G_3\eta_{M1})\beta_{MC}x_6^*\nu_7}{K_1K_2K_3N_{\text{HM}}^*}, \\
\nu_3 &= \frac{(G_4\eta_{M1} + K_3)\beta_{MC}x_6^*\nu_7}{K_2K_3N_{\text{HM}}^*}, \\
\nu_4 &= \frac{\beta_{MC}\eta_{M1}x_6^*\nu_7}{K_3N_{\text{HM}}^*}, \\
\nu_7 &= \nu_7 > 0, \\
\nu_8 &= \frac{\left\{(K_6 + G_8\eta_{F1})(G_2G_4\eta_{M1} + G_2K_3 + K_2G_3\eta_{M1})\right\}\beta_{FC}\beta_{MC}\nu_7}{K_1K_2K_3K_5K_6}, \\
\nu_9 &= \frac{(G_2G_4\eta_{M1} + G_2K_3 + K_2G_3\eta_{M1})\beta_{FC}\beta_{MC}\eta_{F1}\nu_7}{K_1K_2K_3K_6}, \\
\nu_{11} &= \frac{\beta_{MG}x_6}{M_1M_2N_{\text{HM}}^*}, \\
\nu_{13} &= \frac{M_1}{\beta_{MG}\beta_{FG}}, \\
\nu_{15} &= \frac{(G_1\nu_2 + G_{11}\nu_{16} + G_{12}\nu_{17})N_{\text{HM}} + \beta_{MG}x_6^*\nu_{13}}{K_9N_{\text{HM}}^*}, \\
\nu_{16} &= \frac{(H_1\nu_7 + H_2\nu_{13})x_6^* + (G_9\nu_{11} + G_{13})\nu_{17})N_{\text{HM}}}{K_{10}N_{\text{HM}}^*}, \\
\nu_{17} &= \frac{(H_1\nu_7 + H_2\nu_{13})\eta_{M2}x_6^*}{K_{11}N_{\text{HM}}^*}, \\
\nu_{18} &= \frac{(G_5\nu_7 + G_{14}\nu_{19} + G_{15}\nu_{20})N_{\text{HM}} + \beta_{FG}x_1^*\nu_{11}}{K_{12}N_{\text{HM}}^*}, \\
\nu_{19} &= \frac{(H_4\nu_2 + H_3\nu_{11})x_1^* + N_{\text{HF}}G_{16}\nu_{20}}{K_{13}N_{\text{HF}}^*}, \\
\nu_{20} &= \frac{(H_2\nu_4 + H_3\nu_{11})\eta_{F2}x_1^*}{K_{14}N_{\text{HF}}^*},
\end{aligned}
$$

Based on Theorem 3.4 in Castillo-Chavez and Song (2004), and by computing the non-zero partial derivatives of $F(x)$(evaluated at the disease free equilibrium, DFE (ξ_0)), we have that the associated bifurcation coefficients defined by a and b, are given by

$$
a = \sum_{k,i,j=1}^{n} \nu_k\omega_i\omega_j\frac{\partial^2 f_k}{\partial x_i\partial x_j}(0,0) \quad \text{and} \quad b = \sum_{k,i=1}^{n} \nu_k\omega_i\frac{\partial^2 f_k}{\partial x_i\partial \beta_S^*}(0,0),
$$

are computed to be

$$
\begin{aligned}
a = & \frac{2\beta_{FC}\nu_2}{N^*_{\text{HF}}}\left\{\omega_1\omega_8 + \omega_1\omega_9\eta_{F1} + \omega_5\omega_8 r_{\text{MC}} + +\omega_5\omega_9 r_{\text{MC}}\eta_{F1} + \omega_8\omega_{12} + \omega_9\omega_{12}\eta_{F1}\right\} \\
& - \frac{2\beta_{FC}\nu_2 x_1\omega_8}{N^{*2}_{\text{HF}}}\left\{\omega_6 + \omega_7 + \omega_8 + \omega_9 + \omega_{10} + \omega_{13} + \omega_{14}\right\} \\
& - \frac{2\beta_{FC}\nu_2\eta_{F1}x_1\omega_9}{N^{*2}_{\text{HF}}}\left\{\omega_6 + \omega_7 + \omega_8 + \omega_9 + \omega_{10} + \omega_{13} + \omega_{14}\right\} \\
& - \frac{2\beta_{FC}\chi_{\text{MG}}\nu_{11}\omega_8\omega_{11}}{N^*_{\text{HF}}} + \frac{2\beta_{FC}\chi_{\text{MG}}\nu_{15}\omega_{11}}{N^*_{\text{HF}}}\left\{\omega_8 + \omega_9\eta_{F1}\right\} \\
& - \frac{2\beta_{MG}\omega_{11}}{N^*_{\text{HM}}}\left\{\nu_2\omega_2 + \nu_7\omega_7 + \nu_8\omega_8\eta_{\text{FC}} + \nu_9\omega_9\eta_{\text{FC}}\right\} - \frac{2\beta_{MG}\nu_{13}x_6\omega_{11}}{N^{*2}_{\text{HM}}}\left\{\omega_1 + \omega_2 + \omega_3 + \omega_4 + \omega_5 + \omega_{11} + \omega_{12}\right\} \\
& + \frac{2\beta_{MG}\nu_{13}\omega_{11}}{N^{*2}_{\text{HM}}}\left\{\omega_6 + \omega_{14}r_{\text{FG}}\right\} + \frac{2\beta_{MG}\omega_{11}}{N^*_{\text{HM}}}\left\{\nu_{18}\omega_7 + \nu_{19}\omega_8\eta_{\text{FC}} + \nu_{20}\omega_{19}\eta_{\text{FC}}\right\} \\
& - \frac{2\beta_{FG}\omega_{13}}{N^*_{\text{HF}}}\left\{\nu_2\omega_2 + \nu_3\omega_3\eta_{\text{MC}} + \nu_4\omega_4\eta_{\text{MC}}\right\} - \frac{2\beta_{FG}\nu_{11}x_1\omega_{13}}{N^{*2}_{\text{HF}}}\left\{\omega_6 + \omega_7 + \omega_8\right\} \\
& + \frac{2\beta_{FG}\omega_{13}}{N^*_{\text{HF}}}\left\{\nu_{11}\omega_1 + \omega_5 + \nu_{15}\omega_2 + \nu_{16}\omega_2\eta_{\text{MC}} + \nu_{17}\omega_4\eta_{\text{MC}}\right\} \\
& - \frac{2\beta_{MC}\nu_7 x_6\omega_1}{N^{*2}_{\text{HM}}}\left\{\omega_3 + \omega_4\eta_{M1}\right\} - \frac{2\beta_{MC}\nu_{13}\chi_{\text{MG}}\omega_{13}}{N^*_{\text{HM}}}\left\{\omega_3 + \omega_4\eta_{M1}\right\} + \frac{2\beta_{MC}\nu_{18}\chi_{\text{MG}}\eta_{M1}\omega_4\omega_{13}}{N^*_{\text{HM}}}
\end{aligned}
\tag{4.6}
$$

and

$$b = \frac{x_6}{N^*_{\text{HM}}}\nu_7\left(\omega_3 + \ \omega_4\eta_{M1}\right) > 0$$

Since the bifurcation coefficient b is positive, it follows from Theorem 4.1 in Castillo-Chavez and Song (2004) that the model (3.15), or the transformed model (4.5), will undergo a backward bifurcation if the backward bifurcation coefficient, a, given by (4.6) is positive.

4.1.6 Numerical simulations

We now simulate the model (3.15) numerically using the parameter estimates in Table 4.1 (unless otherwise stated), to assess the potential impact of various targeted control strategies on the transmission dynamics of Chlamydia trachomatis and gonorrhea co-infection in the population. Demographic parameters relevant to Ontario in canada were chosen. Specifically, since the total population of sexually active susceptible females and males (15-64 years) in Ontario, Canada are estimated to be 4,892,258 and 4,986,919 respectively, at disease free equilibrium, $\frac{\Pi_{\text{F}}}{\mu_{\text{H}}} = 4,892,258$ and $\frac{\Pi_{\text{M}}}{\mu_{\text{H}}} = 4,986,919$. In Ontario, the life expectancy is estimated at 82 years (Ontario Profile, 2020). Hence we have that $\mu_{\text{H}} = 0.0122$, so that $\Pi_{\text{F}} = 59,662$ and $\Pi_{\text{M}} = 60,816$ per year. In 2017,

the reported cases for females and males with Chlamydia trachomatis in Ontario, Canada, are 26,134 and 18,433, respctively whereas the cumulaative reported cases for females and males with gonorrhea in Ontario, Canada are 2,281 and 5,534, respectively (Sexually Transmitted Infections in Canada, 2017). Hence, we set the initial conditions as follows: $S_{M}(0) = 4,960,000, E_{MC}(0) = 3,352, I_{MAC}(0) = 5,000, I_{MSC}(0) = 5000, R_{MC}(0) = 0, S_{F}(0) = 4,860,000, E_{FC}(0) = 6,317, I_{FAC}(0) = 8,000, I_{FSC}(0) = 7,000, R_{FC}(0) = 0, I_{MG}(0) = 2,851, R_{MG}(0) = 0, I_{FG}(0) = 1,685, R_{FG}(0) = 0, E_{MCG}(0) = 2000, I_{MACG}(0) = 2000, I_{MSCG}(0) = 2000, E_{FCG}(0) = 3000, I_{FACG}(0) = 3000, I_{FSCG}(0) = 2000.$

Table 4.1: Baseline values and ranges of the parameters of the model (3.15).

Parameter	nominal values(*per* year)	Reference
μ_{H}	0.0122	Ontario Demo. Pro. (2020)
Π_{F}	59,662	Ontario Demo. Pro. (2020)
Π_{M}	60,816	Ontario Demo. Pro. (2020)
$\beta_{FC}(\beta_{MC})$	1.1 (1.1)	Sharomi and Gumel(2009)
$\beta_{FG}(\beta_{MG})$	0.40	Mushayabasa(2012)
$\eta_{FC}(\eta_{MC})$	[0.05,0.05]	Sharomi and Gumel (2009)
$\eta_{F1}(\eta_{M1})$	[0.01,0.01]	Assumed
$\eta_{F2}(\eta_{M2})$	[0.01,0.01]	Assumed
$\omega_{FC}(\omega_{MC})$	[0.01,0.01]	Sharomi and Gumel (2009)
$\omega_{FG}(\omega_{MG})$	[0.01,0.01]	Assumed
χ_{MG}	[0.05,0.05]	Assumed
$\theta_{FC}(\theta_{MC})$	[0.025-0.03]*365	Sharomi and Gumel (2009)
$r_{FC}(r_{MC})$	[0.35,0.11]	Sharomi and Gumel (2009)
$r_{FG}(r_{MG})$	[0.3,0.1]	Assumed
$\delta_{FC}(\delta_{MC})$	[0.001,0.001]	Sharomi and Gumel (2009)
$\delta_{FG1}(\delta_{MG1})$	[0.01-0.0998]	Mushasyabasa(2012)
$\delta_{FC2}(\delta_{MC2})$	[0.001,0.001]	Assumed
$\delta_{FG2}(\delta_{MG2})$	[0.05,0.05]	Assumed
$\phi_{FG1}(\phi_{MG1})$	[0.01-0.0172]	Mushayabasa(2012)
$\phi_{FC2}\gamma_{F2}(\phi_{MC2}\gamma_{M2})$	[0.0029-0.007]	Assumed
$\gamma_{FC1}(\gamma_{MC1})$	[1.971,3.65]	Sharomi and Gumel(2009)
$\gamma_{FC2}(\gamma_{MC2})$	[1.80,3.01]	Assumed
$\gamma_{FG2}(\gamma_{MG2})$	[3.50,1.99]	Assumed
$\rho_{F}(\rho_{M})$	$[\frac{2}{52}, \frac{2}{52}]$	Sharomi and Gumel(2009)
$\rho_{F2}(\rho_{M2})$	[0.04,0.04]	Assumed
$\kappa_{F}(\kappa_{M})$	[0.25,0.5]	Sharomi and Gumel(2009)
$\kappa_{F2}(\kappa_{M2})$	[0.5,0.5]	Assumed

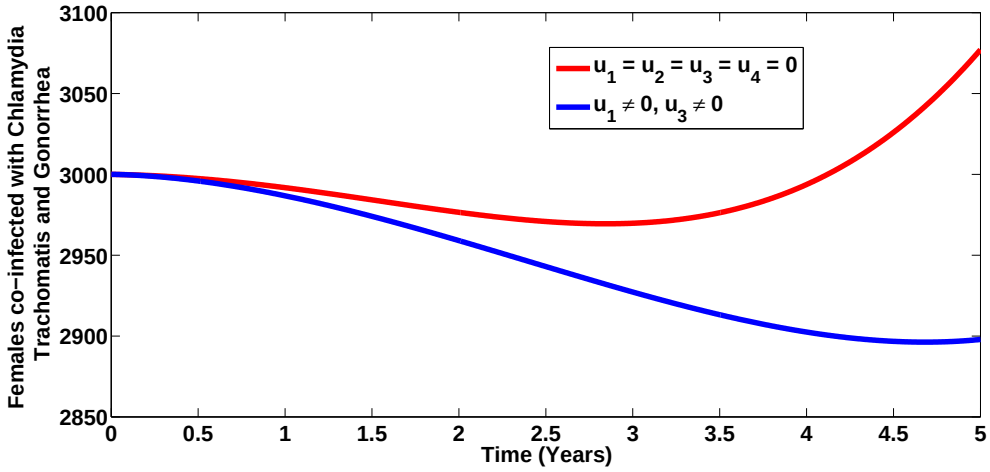

Figure 4.1: Plots of the total number of females co-infected with Chlamyidia trachomatis and gonorrhea when strategy A is implemented. Here, $\beta_{FC} = \beta_{MC} = 1.5$, $\beta_{FG} = \beta_{MG} = 1.2$. All other parameters as in Table 3.2

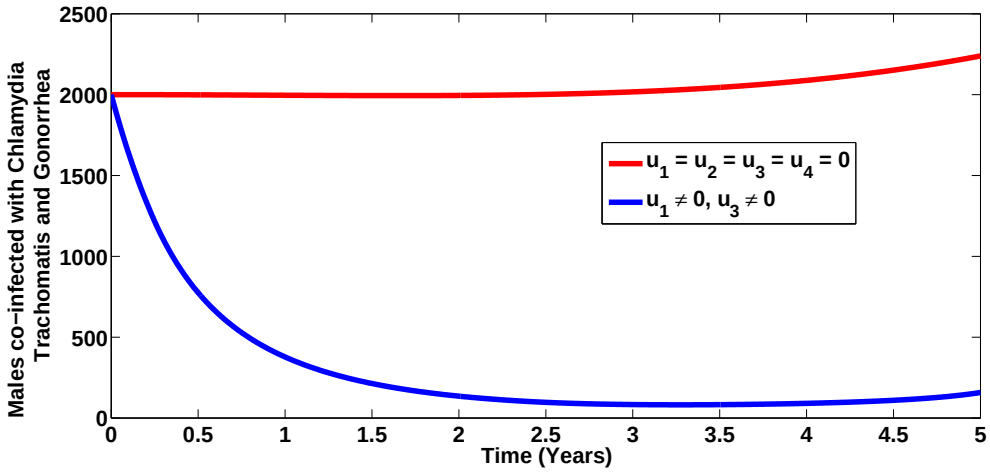

Figure 4.2: Plots of the total number of males co-infected with Chlamyidia trachomatis and gonorrhea when strategy A is implemented. Here, $\beta_{FC} = \beta_{MC} = 1.5$, $\beta_{FG} = \beta_{MG} = 1.2$. All other parameters as in Table 3.2

4.1.7 Strategy A: male Chlamydia trachomatis treatment and male gonorrhea treatment

Simulations of the model when strategy that implements male Chlamydia trachomatis treatment and male gonorrhea treatment is implemented, are presented in Figures 4.1 and 4.2. It is observed that when this intervention strategy is applied, there is a reduction in the total number of females and males co-infected with Chlmaydia trachomatis and gonorrhea. However, the co-infected cases averted for males (2,082) is much higher than the co-infected cases averted for females (179). In general, this strategy prevents a total of 2,261 co-infected cases (for both females and males).

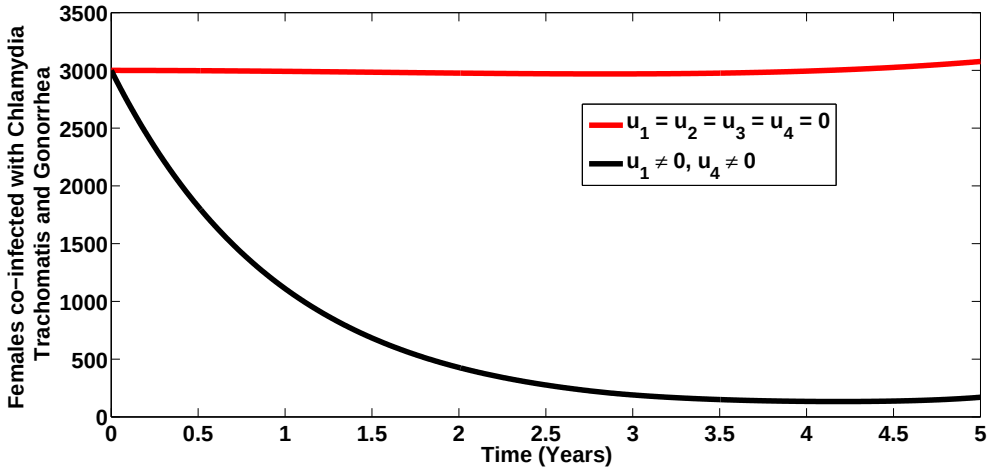

Figure 4.3: Plots of the total number of females co-infected with Chlamyidia trachomatis and gonorrhea when strategy B is implemented. Here, $\beta_{FC} = \beta_{MC} = 1.5$, $\beta_{FG} = \beta_{MG} = 1.2$. All other parameters as in Table 3.2

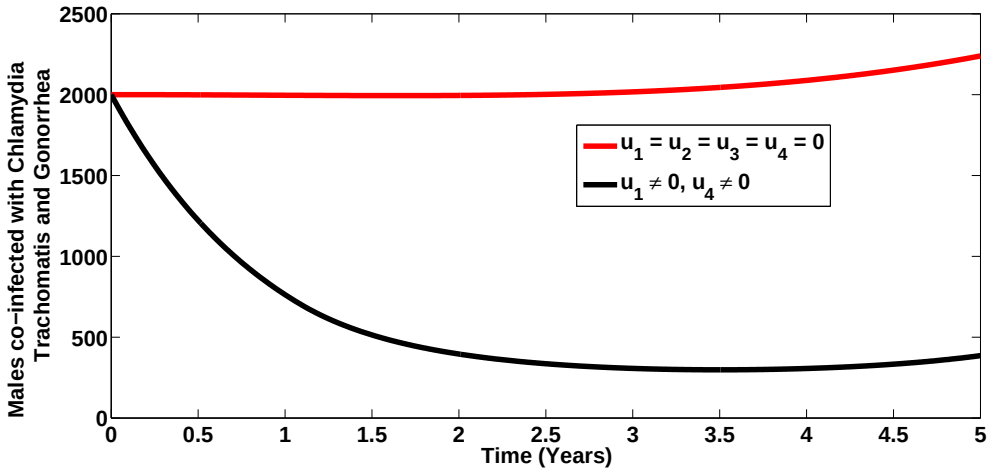

Figure 4.4: Plots of the total number of males co-infected with Chlamyidia trachomatis and gonorrhea when strategy B is implemented. Here, $\beta_{FC} = \beta_{MC} = 1.5$, $\beta_{FG} = \beta_{MG} = 1.2$. All other parameters as in Table 3.2

4.1.8 Strategy B: male Chlamydia trachomatis treatment and female Gonorrhea treatment

Simulations of the model when strategy that implements male Chlamydia trachomatis treatment and female gonorrhea treatment is implemented, are presented in Figures 4.3 and 4.4. It is seen that when this intervention strategy is implemented, there is a reduction in the total number of females and males co-infected with Chlmaydia trachomatis and gonorrhea. Particularly, the co-infected cases averted for males (1,853) is much higher than the co-infected cases averted for females (290). In general, this strategy prevents a total of 2,143 co-infected cases (for both females and males).

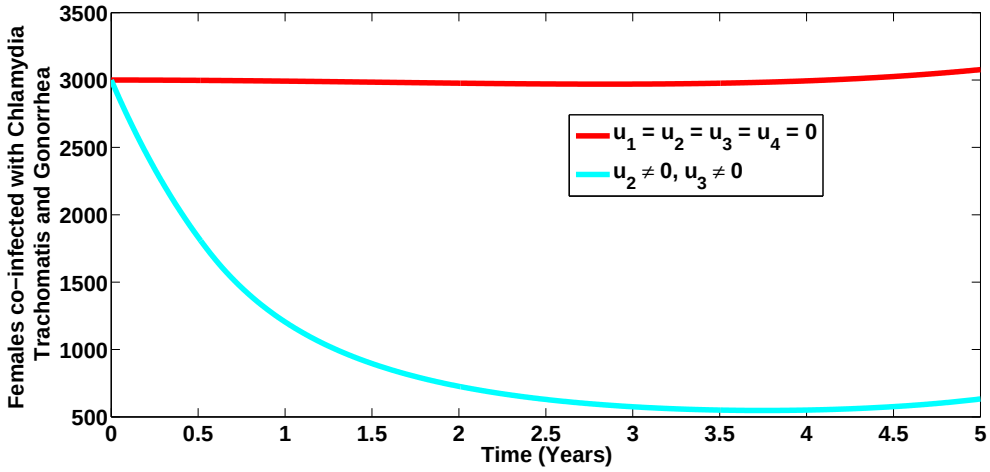

Figure 4.5: Plots of the total number of females co-infected with Chlamyidia trachomatis and gonorrhea when strategy C is implemented. Here, $\beta_{FC} = \beta_{MC} = 1.5$, $\beta_{FG} = \beta_{MG} = 1.2$. All other parameters as in Table 3.2

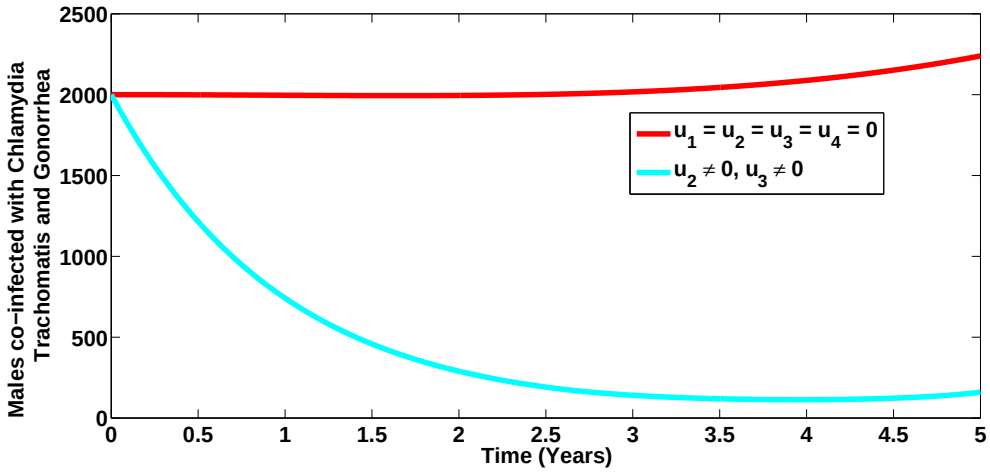

Figure 4.6: Plots of the total number of males co-infected with Chlamyidia trachomatis and gonorrhea when strategy C is implemented. Here, $\beta_{FC} = \beta_{MC} = 1.5$, $\beta_{FG} = \beta_{MG} = 1.2$. All other parameters as in Table 3.2

4.1.9 Strategy C: female Chlamydia trachomatis treatment and male gonorrhea treatment

Simulations of the model when strategy that implements female Chlamydia trachomatis treatment and male gonorrhea treatment is implemented, are depicted by Figures 4.5 and 4.6. It is observed that when this strategy is applied, there is a reduction in the total number of females and males co-infected with Chlamydia trachomatis and gonorrhea. In particular, the co-infected cases averted for males is 2,444 while the co-infected cases averted for females is 2,080. Generally, this strategy averts a total of 4,524 co-infected cases (for both females and males). This is actually the highest co-infected cases averted, when all the intervention strategies are compared.

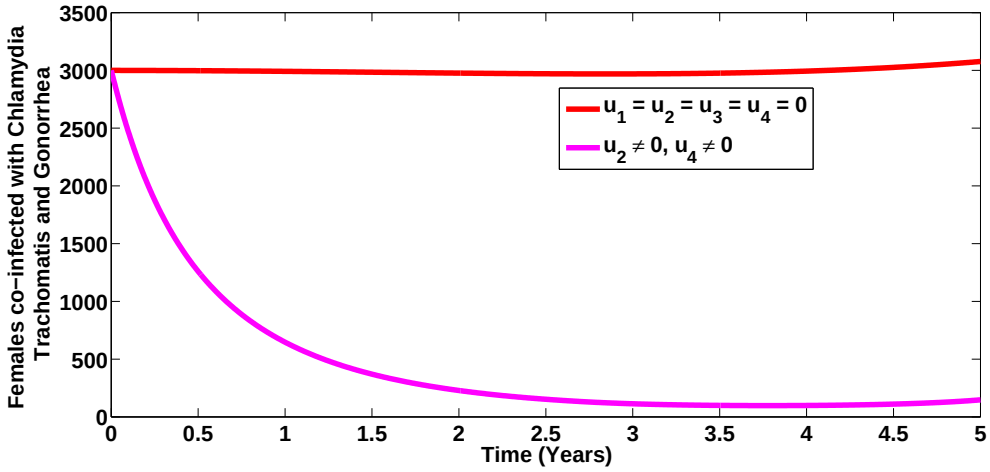

Figure 4.7: Plots of the total number of females co-infected with Chlamyidia trachomatis and gonorrhea when strategy D is implemented. Here, $\beta_{FC} = \beta_{MC} = 1.5$, $\beta_{FG} = \beta_{MG} = 1.2$. All other parameters as in Table 3.2

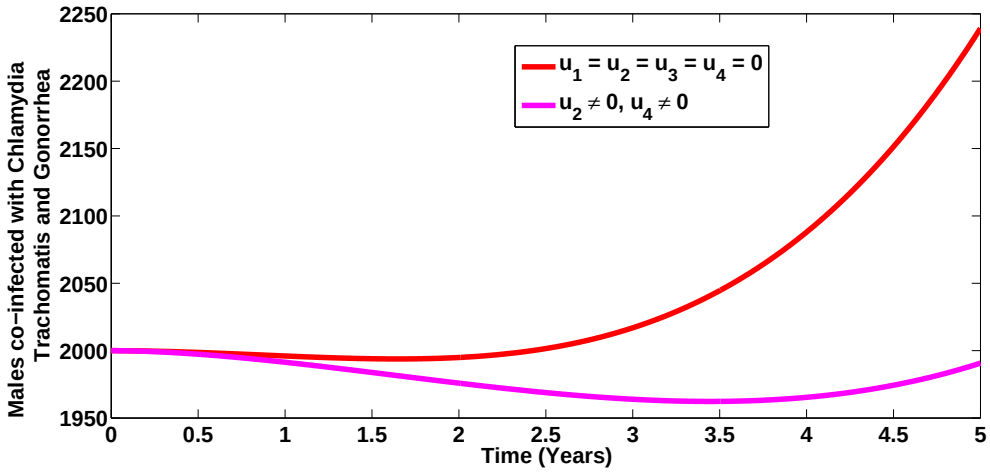

Figure 4.8: Plots of the total number of males co-infected with Chlamyidia trachomatis and gonorrhea when strategy D is implemented. Here, $\beta_{FC} = \beta_{MC} = 1.5$, $\beta_{FG} = \beta_{MG} = 1.2$. All other parameters as in Table 3.2

4.1.10 Strategy D: female Chlamydia trachomatis treatment and female gonorrhea treatment

Simulations of the model when strategy that implements female Chlamydia trachomatis treatment and female gonorrhea treatment is implemented, are shown in Figures 4.7 and 4.8. It is observed that when this strategy is applied, there is a reduction in the total number of females and males co-infected with Chlamydia trachomatis and gonorrhea. interestingly, the co-infected cases averted for males is 2,929 while the co-infected cases averted for females is 249. Generally, a total of 3,178 co-infected cases (for both females and males) is averted by this strategy.

Chapter 5

Conclusion and Recommendation

5.1 Recommendation

people who are sexually active are advised to go for medical check up regularly, this would help prevent or detect any sexually transmitted infection on time, and they will be treated before it is complicated. However, all sexually active women younger than 25 years should be tested for gonorrhea and chlamydia every year. women 25 years and older with risk factors such as new or multiple sex partners or a sex partner who has an sexually transmitted disease should also be tested for gonorrhea and chlamydia every year.These recommendations would help in the reduction of number of people who are been sexually infected with either chlamydia or gonorrhea

5.2 Contribution to Knowledge

1 In this work we have developed and analysed a new co-infection model of chlamydia trachomatis and gonorrhea have not been done by anyone else before to the best of my knowledge

2 we have been able to show that targeted treatment involving female with chlamydia trachomatis and male with gonorrhea can significantly reduce the number of co-infection cases.

3 we have also been able to show the existence of backward bifurcation for the model we have developed

5.3 Conclusion

In this work, a model for Chlamydia trachomatis (CT) and Gonorrhea codynamics was studied and analyzed to assess the impact of targetted treatment for each of the diseases on their co-infections in a population. The model exhibitted the dynamical feature of backward bifurcation when the associated reproduction number is less than unity. The global asymptotic stability of the disease-free equilibrium of the co-infection model was shown not to exist, when the associated reproduction number is below unity. Simulations of the model reveal that the intervention strategy which implements female Chlamydia trachomatis treatment and male gonorrhea treatment is the most in combating the co-infections of Chlamydia trachomatis and gonorrhea.

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