Mathematical Modeling in Epidemiology

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in

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by

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(Enrollment No.: 2K14/Ph.D/AM/01)

Under the Supervision
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DECLARATION

I declare that the research work reported in this thesis entitled **"Mathematical Modeling in Epidemiology"** for the award of the degree of *Doctor of Philosophy in Mathematics* has been carried out by me under the supervision of *Dr. Nilam*, Department of Applied Mathematics, Delhi Technological University, Delhi, India.

The research work in this thesis, except where otherwise indicated, is my original research. This thesis has not been submitted by me earlier in part or full to any other University or Institute for the award of any degree or diploma. This thesis does not contain other person's data, graph or the information unless specifically acknowledged.

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This is to certify that the above statement made by the candidate is correct to the best of my knowledge.

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CERTIFICATE

This is to certify that the research work embodied in the thesis entitled ***"Mathematical Modeling in Epidemiology"*** submitted by **Mr. Abhishek Kumar** with enrollment number **2K14/Ph.D/AM/01** is the result of his original research carried out in the Department of Applied Mathematics, Delhi Technological University, for the award of **Doctor of Philosophy** under the supervision of **Dr. Nilam.**

It is further certified that this work is original and has not been submitted in part or fully to any other University or Institute for the award of any degree or diploma.

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Dedicated to

My parents and teachers, *for making it possible to commence and complete this journey*

CONTENTS

ABSTRACT

In the present thesis, various aspects of the transmission dynamics of epidemics are discussed through the mathematical models. We have proposed and analyzed the various mathematical models to control the spread of emerging/ re-emerging epidemics. We have investigated the facts and reasons behind the spread and control of infectious diseases/ epidemics. After analyzing several systems, various results obtained by analysis of the problem are discussed. The mathematical models have been analyzed for positiveness, boundedness, and stability. Locals stability, global stability, Routh-Hurwitz stability criterion, Descartes' rule of signs, Lyapunov function, MATLAB 2012b (ODE 45, DDE 23), MATHEMATICA 11 are the main tools applied for analysis and simulations of mathematical models.

We have studied two types of mathematical models: ordinary differential equations (ODEs) model and delay differential equations (DDEs) model. The time delay exists almost in every biological phenomenon and is responsible for the severity of the disease and hence in its treatment. Therefore, the importance of the DDE model cannot be ignored in the control and transmission dynamics of the epidemic. The DDE models have been developed for a better understanding of the transmission dynamics of epidemics.

Keywords: Epidemic; Delay differential equations (DDE); Ordinary differential equations (ODE); Time Delay; Nonlinear incidence rates; Nonlinear treatment rates; Bifurcation; Stability.

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CHAPTER 1

INTRODUCTION

Infectious diseases/ epidemics pose a constant threat to human's life as they can affect any individual when contacting or living with the infected individual. The emergence and re-emergence of infectious diseases have turned into critical overall issues. This chapter is introductory in nature which gives a short review of the work done in the field of mathematical epidemiology till now for the transmission dynamics of epidemics. The purpose of the current chapter is to provide some rudimentary information about the infection mechanisms, the control mechanism of epidemics, the role of mathematical models in the field of epidemiology and the motivation behind the work carried out in this thesis. Also, a glimpse of the work carried out has been presented in the present chapter.

1.1 Epidemiology

The study of epidemics is the investigation of components influencing the health and illness of populations and serves as the foundation and logic of interventions made in the interest of public health and preventive drugs. It is viewed as a foundation philosophy of public health research and is much respected in evidence-based medicine for distinguishing hazard factors for disease and deciding ideal treatment ways to deal with clinical practice [Allen (1994); Padma (2008)]. The ultimate aim of any epidemiological study is to eliminate or reduce health problems thereby promoting the health and well-being of the society as a whole. Epidemiological studies are useful for the following reasons:

- It provides relevant information on the rise and fall of disease in a given population.
- Helps in the search for cause and risk factors for disease.
- Elucidate natural course and transmission of the disease.
- Promotes the planning and evaluation of health care facilities and programs.
- Risk assessment of the individual and society.
- Identification of new diseases and syndromes.

Transmission of the diseases decides the severity of the disease in society. Based on the type of transmission, diseases can be classified as:

- **Communicable diseases**

  "A communicable disease is an illness due to a specific infectious (biological) agent or its toxic products capable of being directly or indirectly transmitted from man to man, from animal to man, from animal to animal, or from the environment" [Barreto *et al.* (2006)]. Examples: H1N1, Ebola, Malaria, HIV/AIDS, Cholera, etc.

- **Non-communicable diseases**

  A non-communicable disease is a disease that is not transmissible directly from one person to another. Non-communicable diseases include autoimmune diseases, strokes, heart diseases, cancer, diabetes, chronic kidney disease, etc.

1.2 Modes of transmission

Infectious diseases/epidemics can spread in different ways and pathogens cause contamination by various methods of transmission. A few diseases may occur through immediate contact while others might be caused through backhanded contacts. Transmission can likewise be made through carriers or vectors. For examples, Malaria, Dengue, and Chikungunya spread through mosquitoes. However, two methods of transmission are especially fascinating: airborne infections and sexually transmitted illnesses, and they have been given careful consideration. The examples of airborne infections are flu, SARS, etc. The airborne infection spreads from an infected individual to an uninfected individual through sneeze, cough and even through a laugh. The organisms that are released from a contaminated individual may stay on the dust particles or some other medium. Contamination may happen when these organisms are breathed in or reach bodily fluid film of an uninfected individual through body contact [Rahman (2016)]. Hand-shaking likewise could be a potential path for the transmission of diseases.

On the other hand, a significant number of diseases are sexually transmitted and they are likewise transmitted through contaminated blood and semen, breastfeeding, or during childbirth. HIV stands out amongst the most causing demise due to sexually transmitted infections. Other sexually transmitted diseases including herpes, syphilis, gonorrhea, and chlamydia likewise cause huge contamination and mortality [Rahman (2016)]. These stances serious social and financial results because of longer infectious life, infected people with the sexually transmitted disease may contribute an expanded number of contaminations and subsequently remain a noteworthy issue in the counteraction of diseases. Another basic part of the sexually transmitted diseases is that it may not show any indications on the infectious individual for a longer period. As an outcome, a contaminated individual may transmit disease unknowingly.

The disease transmission specialist can draw its dynamics from various demonstrating models, including compartmental, environmental, atmospheric, and survival models. Models can be developed using deterministic or stochastic approaches; continuous, discrete in time or non-temporal; non-spatial or spatial; homogeneous or heterogeneous with the mixed population; and static or real-time. Significantly more modern epidemiological models are conceivable [Rahman (2016)].

In this thesis, we present only deterministic compartmental models.

1.3 Disease prevention and control

A powerful approach to control the epidemics is to reduce contacts. However, in modern life with increased interactions among people, this approach is not easy to achieve. In addition, to maintain social separation, alternative counteractive actions need to be adopted. Immunization and treatment are the generally utilized counteractive tools that can possibly lessen transmissions and control the diseases.

The spread of infection has been controlled during the last decade due to a combination of behavior change in the population, scaling-up of prevention services, and treatment of disease and vaccination. The main motivation for the prevention of infections among people is that it is feasible and effective if properly implemented. Implementation of effective measures can prevent disease and minimize the harm caused by infectious diseases. Effective measures to prevent infections exist but are either not offered or not accessible to a high proportion of those in need of them. The treatment represents an important component in a comprehensive response to prevent health-related harm. Treatment of infected individuals prevent further transmission, reduce the total healthcare and social costs, improve productivity (health and quality of life) and reduce mortality and morbidity among the target group (*i.e.* susceptible individuals). For the effective treatments, its services must be well-organized, and of high-quality, including the level of training of staff, to achieve the best results. Availability of effective treatment services presents an opportunity to reduce the spread of infection at a higher rate in society.

An immunization (vaccine) is utilized to help the immune system against some particular pathogen. The substance contained in immunization has comparative physical properties to those of a pathogen. Typically, an antibody can be thought of like a phony pathogen that has no capacity to replicate and cause disease. It very well may be made of a powerless or slaughtered pathogen. As immunizations are like pathogenic microorganism, they can stimulate the immune system of the host which develops antibodies against the pathogens to remember them as foreign organisms. In this way, at whatever point such a genuine microorganism is experienced inside a host, the immune system destroys it. This phenomenon is known as resistance or immunity. Therefore, as long as an antibody for a disease is accessible, it is a perfect method for shielding the population from the infection. After Edward Jenner's cowpox antibody, the first known immunization, various effective

campaigns have been propelled against numerous infectious diseases [Lakhani (1992)]. Actually, antibodies have saved millions of lives. Before presenting the primary measles immunization in 1963, around 400,000 measles incidences used to be reported in the United States each year [Rahman (2016)]. Polio, rubella, mumps and other childhood infections likewise used to cause huge mortality and morbidity. With the adequate execution of the immunizations, these diseases no longer remained epidemic.

Vaccines additionally have had a fruitful history against the transmission of flu, the most widely recognized infectious disease around the globe. Prior to the invention of influenza vaccines, controlling a flu pandemic was an incomprehensible assignment. It was evaluated that 20-50 million individuals overall died in the outbreak of Spanish influenza in 1918-19. After a century, the worldwide loss of life for the 2009-10 pandemic was just around 0.3 million [Bryan (2014)]. The vaccine has decreased the loss rate to a huge extent. Flu vaccination presently turns into a routine procedure. An individual is prescribed to get an updated influenza immunization (vaccine) as influenza season approaches with more up to date strains of influenza infections. In spite of the fact that vaccines are extremely powerful against transmission, commonly, there are constraints on the quantities, particularly in developing nations. In this way, how to circulate the limited vaccines becomes crucial for optimal advantages. Social, geographical, monetary and moral issues could be significant obstructions in the implementation of vaccines [Medlock and Galvani (2009)]. Moreover, certain groups of people may have a higher susceptibility to the infections than others. In flu, for instance, school-going youngsters can easily catch the infection and spread the disease more quickly than other people [Foy *et al.* (1976); Longini and Halloran (2005); Jordan *et al.* (2006); Loeb *et al.* (2010)].

1.4 Basic compartmental models

The general idea for most deterministic models is to look at compartmental models, in which the population is divided into compartments based on infection dynamics. Individuals already in one compartment may either transfer to another compartment (for example by recovering from the disease) or may leave compartment altogether (e.g. by disease-induced death). Individuals may enter into a compartment through processes such as immigration or birth. Some basic entrance, exit, and transfer mechanisms among

various compartments such as SI, SIR, and SEIR (where S, E, I and R are denoting the susceptible, exposed, infected and recovered individuals compartment according to the disease status.) are shown in Fig.1.1-1.3. Compartmental models may become very complex; they may use many compartments, or assume complicated disease distribution or incidence, or have parameters which are time or even state-dependent. These three concepts, however, of entering the population, transferring between compartments, and leaving the population, always underlie the assumptions.

1.5 Epidemic models

The earliest mathematical modeling can be traced back to the eighteenth century when Daniel Bernoulli figured a model for smallpox to evaluate the effectiveness of control measures on the infected population with smallpox [Benenson (1995); Hethcote (2000)]. However, mathematical models have been developing since the middle of the twentieth century after Kermack and McKendrick [1927] published their paper on epidemic models which contained threshold results that decide if an epidemic outbreak may occur or not [Hethcote (2000); Kermack and McKendrick (1927)]. In the course of the most recent two rapid increases in modeling, practices have been employed in the biological sciences [Anderson and May (1982); Hethcote (2000)]. These models have addressed numerous aspects of biological phenomena, for example, phases of infection, vertical transmission, disease vectors, age structure, social and sexual mixing groups, spatial spread, chemotherapy, immunization, isolate, passive immunity, steady loss of vaccine and disease-acquired immunity [Anderson and May (1982); Hethcote (2000); Grassly and Fraser (2008)]. A few models were focused on diseases like measles, rubella, chickenpox, diphtheria, cancer, smallpox, malaria, rabies, herpes, syphilis, and HIV/AIDS [Anderson and May (1982); Usher (1994); Hethcote (2000); Longini and Halloran (2005)].

The disease transmission models describe the transmission procedure and trace the infected population. Such models can recognize the number or extent of the population that is left uninfected towards the end of an epidemic. In epidemic models, the idea of population compartments is broadly utilized [Anderson and May (1982); Murray (1989); Diekmann and Heesterbeek (2000); Hethcote (2000)]. For mathematical convenience, these compartments are normally denoted by their first letter, for example, *S*, *E*, *I*, and *R*

denotes the number of susceptible, exposed, infected and recovered population respectively. People who are vulnerable against infection are known as susceptible and have been placed into the *S* (susceptible) compartment. A person, who is presently infected, yet does not indicate symptoms or can't contaminate others has been placed into the *E* (exposed) compartment. Once an infected individual begins contaminating others, he/she has been considered as infectious and is placed into *I* (infected) compartment. Finally, when an individual has been cured of the infection, he/she has been placed into the *R* (recovered) compartment. Depending upon the particular disease, a recovered individual either stays there if he/she gets permanent recovery or may become susceptible again and move once more into *S* compartment. Different models can be developed by considering these compartments in light of the idea of pathogens and infections, for example, SIS, SIR, SIRS and so on. If an infected individual becomes susceptible again after cure, a SEIS or SIS type model would be appropriate for the disease dynamics. Bacterial diseases could be considered an example of SIS models. Then again, if recovery is lasting and the recovered people are not any more vulnerable to that pathogen, as observed in viral infection, at that point a SIR-type model would be appropriate. In all cases, the population is thought to be homogeneously mixed and people catching infections or be cured at constant rates. Some representational diagrams are also shown in Figs. 1.1-1.3.

A basic SIR epidemic model is described by following ordinary differential equations [Kermack and McKendrick (1927)]:

$$\begin{aligned} \frac{dS(t)}{dt} &= -\beta S(t)I(t), \\ \frac{dI(t)}{dt} &= \beta S(t)I(t) - \gamma I(t), \\ \frac{dR(t)}{dt} &= \gamma I(t). \end{aligned} \tag{1.1}$$

where β is the transmission rate and γ is the recovery rate. Without considering the demography of the host population, this simple model describes how sub-populations of susceptible, infected and recovered classes evolve. Model (1.1) has been modified by incorporating various factors to capture the important aspects of the specific problems one is concerned with, but such modifications increase the complexity of the model and make the analysis challenging and sometimes even impossible (see, *e.g.* [Murray (1989); Diekmann and Heesterbeek (2000); Guo and Li (2006); Lewis (2009)]). Therefore,

balancing the rationality and mathematical tractability of a model always remains an important issue when using a mathematical modeling approach to study disease dynamics.

In model (1.1) the term $\beta S(t)I(t)$ is referred to as bilinear or mass action incidence rate which demonstrates that incidence increases with the numbers of susceptible and infected. This transmission rate is the product of the rate of contact among individuals and the probability that a susceptible individual coming in contact with an infectious individual will become infected (Park, 1997). Numerous other nonlinear saturated incidence rates are also commonly used by various researchers [Murray (1989); Diekmann and Heesterbeek (2000); Guo and Li (2006); Dubey *et al.* (2013) & (2016)]. We explore some of the nonlinear incidence rates here:

- **Holling functional type II**

  The expression $F(S,I) = \frac{\beta I}{1+\gamma I} S,\ \beta, \gamma > 0$, is known as Holling functional type II incidence rate. This incidence rate is also known as the saturated incidence rate and it was proposed by C. S. Holling [1959]. In Holling type II, "for any outbreak of the disease, its incidence is first very low and then grows slowly with increase in infection. Further, when the number of infected individuals is very large, the infection reaches its maximum due to the crowding effect" [Dubey (2016)].

- **Ratio-dependent functional type**

  The expression $f(S,I) = G\left(\frac{S}{\gamma I}\right) I = \frac{\beta S}{\alpha S+\gamma I} I,\ \beta,\ \alpha, \gamma > 0$ is known as ratio-dependent functional type incidence rate. This incidence rate is obtained by putting $\frac{S}{\gamma I}$ in Holling type II *i.e.* $\frac{\beta S}{1+\alpha S}$. This incidence rate is applicable for a low density of susceptible population.

- **Beddington-DeAngelis (B-D) functional type**

  "The expression $F(S,\ I) = \frac{\beta SI}{(1+\alpha S+\gamma I)},\ \beta, \alpha, \gamma > 0$ is known as Beddington-DeAngelis type incidence rate. Here β is the transmission rate, α is a measure of inhibition effect, such as preventive measure taken by susceptibles and γ is a measure of inhibition effect such as treatment with respect to infectives" [Dubey *et al.* (2015)]. This incidence rate was introduced by Beddington [1975] and

DeAngelis *et al.* [1975] independently. “This incidence rate considers the effect of inhibition among infectives in case of the low density of susceptible populations” [Dubey *et al.* (2015)].

In the model (1.1), an ODE framework has been used in which the time-dependence is given by the present time t. In contrast to Ordinary Differential Equations (ODEs), Delay Differential Equations (DDEs) take into account the inclusion of past activities into mathematical frameworks, thus making the model closer to the real-world phenomenon. In the study of epidemics, the time delay can represent the latent or incubation period, or the time in which, a host stays infected. However, delay equations can also be used to investigate the phenomenon of waning immunity. When the body gets contaminated by a virus, indeed, the immune system develops a certain resistance against it. In fact, disease-induced immunity tends to wane and, a long time after recovery, an individual might again become susceptible to the virus. Delay equations can have more rich dynamics than ODEs and can be a superior fit to complex real phenomenon *e. g.* spread of an epidemic. They can be far more complicated than ODEs because a delay differential equation is infinite-dimensional; thus chaos may happen even in low-order systems.

The disease transmission models with latent or incubation period have been studied by various authors, because numerous diseases, for example, flu, tuberculosis, H1N1, have a latent or incubation period, during which the individual is said to be infected but not infectious. Delay differential equations (DDEs) have been effectively used to model varying infectious periods in the scope of SIR, SIS, SEIR, and SIRS epidemic models [Mukherjee (1996); Naresh *et al.* (2009); Huang *et al.*(2011); Huang and Takeuchi (2011); Mishra *et al.*(2011); Paulhus and Wang (2015); Waezizadeh (2016)]. Many researchers [Xu and Ma (2009a); Hattaf *et al.* (2013); Li and Liu (2014)] have considered an epidemic model with constant time delay, which represents the duration of infectiousness. The epidemic model (1.1) is an example which involves the transmission of disease through one population (*i.e.* Humans). On the other hand, vector-borne diseases models can utilize mixed delays because of the interaction between two species population, for example, mosquitos spreading malaria in humans.

Meng *et al.* [2010] discussed a condition with the delay in the infected (I) of the incidence, however not in S; that is, the bilinear incidence rate is $\beta S(t)\, I(t-\tau)$ with an incubation period τ. The present rate of new infective individuals relies upon the present number of vulnerable individuals and upon the present number of infective mosquitoes.

It is established that proper and timely treatment methodology can substantially reduce the effect of disease on society. In classical epidemic models, the treatment rate of infected individuals is assumed to be either constant or proportional to the number of infected individuals. However, we know that there are limited treatment resources available in the community [Zhou and Fan (2012)] for new and mutated re-emerging infections. In the absence of effective therapeutic treatments and vaccines, the epidemic control strategies are based on the choice of appropriate preventive measures. Many researchers [Gumel *et al.* (2006); Hattaf and Yousfi (2009); Naresh *et al.* (2009)] incorporated treatment rate as constant or linear while some have been found that have nonlinear saturated treatments such as Holling type II & III [Zhang and Liu (2008); Zhou and Fan (2012); Dubey *et al.* (2013); Goel and Nilam (2019)] and give a better alternative due to its saturated behavior. Some infections cannot be controlled completely by treatment only due to the limited availability of medical resources. The dissemination of awareness about prevention, spread, and treatment modalities of infectious diseases through public and social media and health care workers is also an important tool to control and restrain further infection [Dubey (2016)].

1.6 Basic reproduction number

A typical parameter utilized in modeling diseases is the basic reproduction number R_0, describes various aspects, for example, contact rate, duration of contamination and infectiousness of the causative agent. Contact rate strongly affects the transmission and spread of infectious disease. Contact patterns are some of the more complicated aspects of predicting outbreaks, because the human behavior is quite complex and does not remain consistent among all individuals. The basic reproduction number R_0 is characterized as "the average number of secondary infections caused by one infected individual during his/her entire infectious period in a completely vulnerable population" [Driessche and Watmough (2002)]. Contact rate has a large effect on this parameter, as higher the

effective contacts lead to higher rates of new infection. Another determinant of R_0 is the duration of infectiousness; people with longer infectiousness periods will contact and thus possibly infect more people over the whole range of the infection. The basic reproduction number is a fundamental determinant of the dynamics of disease infection in the population level. An epidemic outbreak will occur if and only if R_0 is greater than one. This threshold property provides important information about the progression of disease spread and the impact of control mechanisms.

1.7 Stability analysis

Mathematical models are becoming increasingly complicated when a higher level of non-linearity is adopted to address real-world problems. Finding an explicit solution of these models is relatively impossible. Through numerical simulations, approximate solutions with fix parameters can be found, though still, the general solution may remain unknown. At the point when the general solution is difficult to accomplish, stability analysis can be used to get a sense of the solution's long-term behavior exceptionally well. In general, there are two kinds of model solution widely used in literature: local and global. Local stability is concerned with the behavior of the model solution around the equilibrium point, while global stability can describe solution behavior in the whole domain.

Delay differential equations are often of interest to determine whether or not the delay values affect the stability of a steady-state. Mainly, the delay is treated as a bifurcation parameter. To determine whether or not a stable steady-state can become unstable by changing the delay value, we look at the eigenvalues from roots of characteristic equations. If all the roots have a negative real part, the steady-state is stable. When we vary the delay, if one of the root changes from having a negative real part to having a positive real part, the steady-state will become unstable. This is also equivalent to having the root crossing the imaginary-axis (imagine the root as a graph with a real part on the x-axis and imaginary part on the y-axis). Therefore, if the root really changes to positive real part, there must be a root that is purely imaginary part (*i.e.* the intersection between the graph of the root and the imaginary-axis exist.).

In this thesis, the Routh-Hurwitz (R-H) criterion and Lyapunov direct method [Sastry (1999)] are mainly used for the stability of model equilibria. The Routh-Hurwitz (R-H) criterion is useful to check the local stability of an equilibrium point. The local stability describes the qualitative behavior of the solution in a certain neighborhood. It does not give any information about the behavior of the solution beyond that neighborhood. The Lyapunov direct method can be useful to study the stability behavior of nonlinear systems. The physical validity of this method is contained in the fact that the stability of the system depends on the energy of the system which is a function of system variables. The Lyapunov direct method consists in finding out such energy functions termed as Lyapunov function which need not be unique. The major role in this process is played by positive or negative definite functions which can be obtained in general by the trial of some particular functions of state variables, and in some case with a planned procedure [Dubey (2016)].

1.8 The organization of the thesis

The thesis entitled **"Mathematical Modeling in Epidemiology"** contains nine chapters followed by conclusion & future scope and bibliography. The thesis is organized as follows:

Chapter 1: Chapter 1 is introductory and gives a general background of epidemic modeling theory, basic terminology, important concepts, and types of models. The purpose of this chapter is to give the chronological development in epidemiology and motivation behind the work done in the thesis.

Chapter 2: In Chapter 2, the dynamic behavior of a susceptible-infected-recovered (SIR) epidemic model is presented and analyzed with the incidence rate of new infection as Monod-Haldane (M-H) functional type and treatment rate also as Monod- Haldane (M-H) functional type. M-H type incidence rate defines the inhibitory or psychological effect from the behavioral change of the susceptible individuals in case of a large number of infected individuals. It has great significance, as the number of effective contacts between infected and susceptible individuals decreases at high levels, because of either isolation of infected individuals or precautionary measures taken by susceptible individuals. In M-H

treatment rate, the removal/treatment at first increases with the increasing number of infectives and attains its peak, *i.e.*, the treatment is being given to the maximum number of infectives; after that decay in the slope begins and the incidence rate approaches zero. The limitation in the availability of treatment for a large number of infected people can be captured mathematically by M-H treatment rate. The numerical results of the model demonstrate the impact of M-H treatment and incidence rates on the infected population.

The work presented in this chapter has been communicated under the title **"A SIR Disease Transmission Model with Nonlinear Functional Response"**.

Chapter 3: This chapter demonstrates the control strategy for the epidemics in which infection grows at a very high rate and even at some stage; the infected population is more than the susceptible population. Such a situation can be modeled by considering the incidence rate of infection as a ratio-dependent functional. Because of the large number of infected individuals, the treatment facilities may not be made available for the entire infected population at the same time. This limitation of the treatment facilities can be represented mathematically by using Holling functional type II treatment rate. Therefore, the progression of the epidemic has been modeled by taking the combination of ratio-dependent incidence and Holling functional type II treatment rates. The impact of this combination on the transmission dynamics of the epidemic has been demonstrated with the help of numerical simulations.

The work presented in this chapter has been communicated under the title **"A Study on the Dynamics of An Epidemic Model with Ratio-Dependent Incidence and Holling Type II Treatment Rates."**

Chapter 4: For most communicable diseases there is a time interval between infection and occurrence of symptoms (the incubation period) in which the infectious agents are increasing with time. The incubation period is a deciding factor for the severity of the disease because the disease cannot be identified initially and hence can't be treated during the incubation period. This incubation period has been modeled as the delay time in Holling type II incidence rate with an inhibitory effect. Since the treatment facilities will be extremely constrained for new or evolved infection, therefore, Holling functional type

II will be used as a treatment rate. The model has been analyzed for local stability for model equilibria. The outcomes of numerical simulation recommend that the disease be precisely controlled when treatment is given to infectives under Holling functional type II treatment rate.

The results of this part of the chapter have been published entitled **"Stability of a Time Delayed SIR Epidemic Model Along with Nonlinear Incidence Rate and Holling Type – II Treatment Rate"** in *International Journal of Computational Methods, 2018 (World Scientific).*

An attempt has also been made to find the more suitable treatment method of a known disease which has re-emerged and has available treatment methods. Treatment rate of such diseases can be modeled by the Holling functional type III. Therefore, the treatment rate has been changed to Holling functional type III while the incidence rate will be kept as Holling functional type II with time delay only to find out the changes in the infected population theoretically. Hopf bifurcation analysis of endemic equilibrium and global stability analysis of model equilibria are also discussed.

The results of this part of the chapter have been communicated under the title "**A Deterministic Time-Delayed SIR Epidemic Model: Mathematical Modeling and Analysis".**

Chapter 5: Some communicable diseases can be transmitted in humans even without showing clinical symptoms. The time taken between infection and infectiousness is called the latency period. The duration of this period may be responsible for disseminating the disease. Therefore, to capture the role of latency period mathematically, it has been used as the time delay in both susceptible and infectives in Holling functional type II incidence rate. The treatment rate of infected is taken as Monod-Haldane (M-H) functional type. This treatment rate consists of the cure rate and limitation rate in treatment availability. In the M-H treatment rate, the removal/treatment rate grows initially with the development of infectives and diminishes after attaining its maxima. The results obtained from the numerical simulations have been discussed in detail in chapter 5.

The work reported in this chapter entitled **"Dynamical Model of Epidemic Along with Time Delay; Holling Type II Incidence Rate and Monod–Haldane Type Treatment Rate"** has been published in *Differential Equations and Dynamical Systems, 2018 (Springer)*.

Chapter 6: Monod-Haldane (M-H) type incidence rate is a possible mathematical representation to explain the psychological effects from the behavioral changes of the susceptible individuals when the number of infectives is relatively very high. As discussed earlier in chapter 4, incubation period is also a vital factor in disseminating the disease, therefore in the present chapter, the same delay term has been used in incidence rate to push the epidemic model into a more realistic state. Now, according to the newly modeled incidence rate, the infection force may decrease with increasing numbers of infectious people because the number of contacts in the population may tend to reduce per unit time in the presence of a large number of infectious individuals. Hence, the effects of the novel combination of M-H functional incidence rate with the inclusion of time delay and Holling type II functional treatment rate on susceptibles and the infected population has been studied with the help of the present model.

Then, there will be a situation when for some communicable diseases, effective pre- and post-vaccination/treatment can be made available. In such cases, the removal/treatment rate is relatively high initially in spite of increasing infectives, and afterward, it decreases gradually, finally reaching a saturated value. After this, any expansion in numbers of infectives won't affect the removal rate. Such a situation can be modeled with the Holling functional type III treatment rate. Therefore, an alternate approach by considering the combination of Holling type III treatment rate along with M-H functional type incidence rate has been used to study the epidemic in a more realistic way. The mathematical analysis of the model consists of the local stability as well as global stability analysis at model equilibria and results have been discussed in detail for the modified model.

Some results of the work presented in this chapter have been published entitled **"Mathematical Analysis of a Delayed Epidemic Model with Nonlinear Incidence and Treatment Rates"** in *Journal of Engineering Mathematics, 2019 (Springer)* and some

results are communicated under the title **"Analysis of a Disease Transmission Model with Time Delay, Nonlinear Functional Type Incidence Rate and Saturated Treatment Rate".**

Chapter 7: In this chapter, the incidence rate of a new infection is considered as Crowley-Martin (C-M) functional type because it considers the effect of inhibition among infectives even in case of the high density of susceptible population which is neglected by any other incidence rate. The latency time has been used as a delay in the incidence rate to understand the dynamics of the epidemic more pragmatically. Therefore, a combination of C-M incidence rate along with time delay and Holling functional type II treatment rate is studied. The local stability, as well as global stability analysis of the model equilibria, is discussed. The numerical outcomes demonstrate the impact of inhibitory effects, time delay and nonlinear treatment on the infectious population.

The work presented in this chapter has been communicated under the title **"Dynamic Behavior of An SIR Epidemic Model Along With Time Delay; Crowley-Martin Type Incidence Rate and Holling Type II Treatment Rate"**.

Chapter 8: It has been reported that awareness can play a vital role in the spread of an epidemic. Therefore, to quantify the impact of the effectiveness of being aware of an emerging/re-emerging epidemic a new compartment called alert (A) is introduced in the SIR epidemic model. This requires two incidence rates: one from the susceptible class to infectious class and another from alert class to infectious class which are taken as Holling functional type II. The treatment rate of infectious is taken as Holling functional type II on the grounds that for an outbreak of the disease its treatment effectiveness is low initially and improves gradually with the introduction of hospital facilities, availability of more effective medicines, etc. The result obtained from numerical simulations of the model having a combination of above explained incidence and treatment rates demonstrates the impact of alert class on the infectives.

In this model, it has been considered that health agencies are spreading awareness while the concept of immunization has not been covered. To save the lives of humans from the epidemics it is very important to provide immunization to the general public, but

immunization is not possible for all, and also it has some failure aspects. However, immunization minimizes the effect of the epidemic in societies. Therefore, two explicit treatment classes which are pre-treated individuals class and post-treated individual's class are introduced in the SIR model resulting in a five-compartment model. The pretreated class is introduced to minimize the infectives in the general public, and post-treated class is introduced for the recovery of the infectives. This motivates us to take Holling functional type I and III rates for pre-treatment and post-treatment of individuals, respectively, to control the illness. The incidence rate from susceptible to infectives has been considered as Holling function type II with incubation period as a time delay to study the dynamic of the epidemic more realistically. The numerical outcomes suggest that disease can be controlled in the general public if preventive measures and treatment of susceptible and infectious are managed by Holling type I and III treatment rates separately.

Some results of the work presented in this chapter have been published in *Computational and Applied Mathematics, 2019 (Springer)* under the title "**Stability of a Delayed SIR Epidemic Model by Introducing Two Explicit Treatment Classes Along With Nonlinear Incidence Rate and Holling Type Treatment"** and some results have been published in *SeMA Journal, 2019 (Springer)* under the title "**A Short Study of An SIR Model with Inclusion of An Alert Class, Two Explicit Nonlinear Incidence Rates And Saturated Treatment Rate".**

Chapter 9: This chapter contains the conclusion of the work done and future scope of the models discussed in the thesis.

Fig. 1.1: Progression of infection through basic susceptible-infected (SI) compartment model with transmission rate (β).

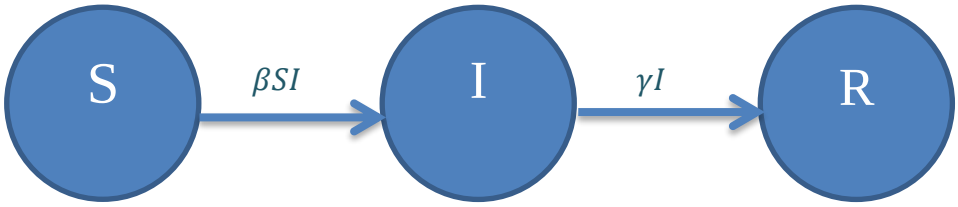


Fig. 1.2: Progression of infection through basic susceptible-infected-recovered (SIR) compartment model with transmission rate (β) and recovery rate (γ).

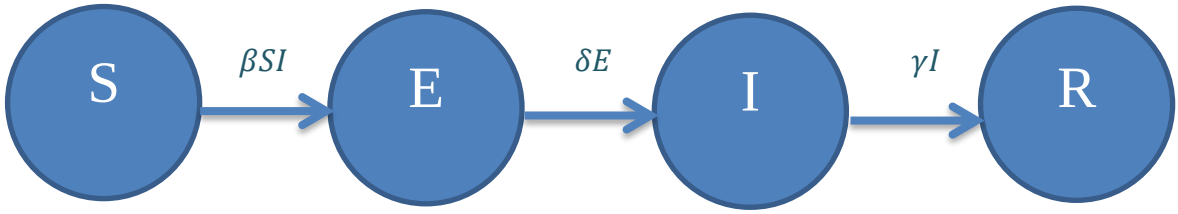


Fig. 1.3: Progression of infection through basic susceptible-exposed-infected-recovered (SEIR) compartment model with transmission rate (β), exposed rate (δ) and recovery rate (γ).

CHAPTER 2

A SIR EPIDEMIC MODEL WITH MONOD-HALDANE FUNCTIONAL TYPE INCIDENCE AND TREATMENT RATES

In this chapter, a SIR epidemic model has been presented and analyzed with a novel combination of incidence and treatment rates both of Monod-Haldane (M-H) functional type. Stability of the disease-free (DFE) and endemic equilibria (EE) has been discussed. The stability of DFE has been discussed in terms of the basic reproduction number(R_0) and it was shown that DFE is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. Stability of DFE at $R_0 = 1$ has been investigated using center manifold theory; it was found that DFE exhibits a forward bifurcation. Conditions have been obtained for the stability of EE. Furthermore, the global stability of DFE has also been discussed. Lastly, numerical simulations have been performed to illustrate the results predicted by the analysis.

2.1 Introduction

To control the spread of infectious diseases, mathematical modeling always plays a vital role. In the literature of mathematical epidemiology, numerous mathematical models have already been proposed for disease dynamics such as SIS [Li *et al.*(2006)], SIR [Shulgin *et al*. (1998); Kaddar (2009) & (2010); Li *et al.* (2009); Xu and Ma (2009b), McCluskey (2010); Pathak *et al.* (2010); Xu and Du (2011); Abta *et al*. (2012); Hattaf *et al.* (2013); Adebimpe *et al.* (2015); Dubey *et al.* (2015) & (2016); Chen *et al.* (2016)], SIRS [Xu and Ma (2009a); Sun and Yang (2010)], SEI [McCluskey (2012)], SEIR [Li and Muldowney (1995); Zhang and Ma (2003); Li and Jin (2005); Katim and Razali (2011); Abta *et al*. (2012); Dubey *et al.* (2013); Tipsri and Chinviriyasit (2014); Liu *et al.* (2015)], SEIS [Guo *et al.* (2010)], SVIR [Liu *et al.*(2008); Wang *et al.* (2016)], SVEIR [Gumel *et al.* (2006); Wei *et al.* (2009); Wang *et al.* (2015)](where, $S, V, E, I,$ and R denotes the susceptible, vaccinated, exposed, infected and recovered individuals respectively) and many others. The spread of the epidemic mainly depends on the incidence rate. The number of individuals who become infected per unit of time in epidemiology is known as the incidence rate [Dubey *et al.* (2015)]. Kermack and McKendrick [1927] proposed that the dynamics of an infectious disease could be described using a bilinear incidence rate βSI of infection. However, this bilinear type incidence rate depends on the law of mass action, which is unreasonable for a large infected population. Indeed, one can infer from the term βSI that, if the number of susceptible individuals increases, incidence rate also increases, which is unrealistic. Hence, there is a need to modify the classical linear incidence rate in order to study the dynamics of infection among a large population. Many researchers [Xu and Ma (2009a); Hattaf *et al.* (2013); Dubey *et al.* (2015) & (2016)] have proposed transmission laws that include nonlinearity, such as the Holling type II functional, Crowley–Martin functional, Beddington-DeAngelis functional, etc., for the dynamics of infectious diseases. The general incidence rate

$$g(I)S = \frac{kI^p S}{1 + \alpha I^q},$$

was suggested by Liu et al. [1987] and thereafter considered by numerous researchers in their models (see, for example, [Hethcote and Levin (1989); Hethcote and Driessche (1991); Derrick and Driessche (1993); Hethcote (2000); Alexander and Moghadas (2004)]). If the function $g(I)$ is non-monotonic, that is, $g(I)$ is increasing when I is

small but decreasing when I is large, it can be used to interpret the "psychological" effect, *i.e*. the infection force may get reduced as the number of infected increases for a large number of infected population, because in such situation the number of contacts per unit time may tend to reduce for $p < q$. For example, the epidemic outbreak of SARS showed such psychological effects on the general public; aggressive measures and policies, such as border screening, mask wearing, quarantine, isolation, etc., have been proven to be very effective [Xiao and Ruan (2007)] in reducing the infective rate at the later stage of the SARS outbreak, even when the number of infected individuals was increasing. The above-mentioned phenomenon can be modelled by using the nonlinear Monod–Haldane (M-H) incidence rate:

$$f(S,I) = g(I)S = \frac{kIS}{1+\alpha I^2}, \text{ where } k, \alpha > 0.$$

where kI measures the force of infection of the disease and $1/\,(1 + \alpha I^2)$ describes the psychological effect from the behavioral change of susceptibles when the number of infectives is very large.

Treatment rate always helps doctors and health agencies to control/ eradicate the infection from the population. It is known that there are limited treatment resources available in the community [Zhou and Fan (2012)] for large infected populations. In the absence of effective therapeutic treatments and vaccines, epidemic control strategies are based on taking appropriate preventive measures. Therefore, we incorporate the treatment rate as nonlinear Monod-Haldane (M-H) type in our epidemic model. In the M-H functional type treatment, "the removal/treatment rate firstly increases with the growth of infectives, reaches the maximum and then starts decaying. Such a situation may arise due to the limitation in the availability of treatment for a large number of infected populations. When supplies of treatment (medicine, immunization, etc.) are depleted, then in spite of the high number of infectives the available treatments become very scarce. This case may arise when there is the re-emergence and spread of disease in the presence of limited treatment facilities" [Dubey *et al.* (2013)].

This chapter presents the dynamics of a susceptible-infected-recovered (SIR) mathematical model with M-H functional type incidence and treatment rates to capture the impact of physiological effect and limitation of resources on infectives. The basic properties of the model have been discussed. The stability of the model has been

discussed for the two equilibria which are named as disease-free equilibrium (DFE) and endemic equilibrium (EE). We discuss the local and global stability of DFE through the basic reproduction number and Lyapunov function. Furthermore, the stability of EE has also been discussed by application of the Routh-Hurwitz criterion.

2.2 Mathematical model

In this section, a mathematical epidemic transmission model is being proposed. For this, it is considered that the total population at time t is $P(t)$, with the immigration of susceptible individuals at a constant rate A. Further, the total population $P(t)$ has been divided into three classes (or compartments), which are named as: susceptible class $S(t)$, infected class $I(t)$ and recovered class $R(t)$. It is assumed that the disease can spread due to the direct contact between susceptibles and infectives only. It is also assumed that recovered individuals are immune for their entire life and they will not re-infect and therefore no movement is possible from $R(t)$ to $S(t)$ compartment. Let μ be the natural death rate of the population, d the disease-induced mortality and δ the recovery rate of infected individuals.

By taking above assumptions into consideration, the dynamics of the model will be given by the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dS(t)}{dt} &= A - \mu S(t) - \frac{\beta S(t) I(t)}{1+\alpha I^2(t)}, \\
\frac{dI(t)}{dt} &= \frac{\beta S(t) I(t)}{1+\alpha I^2(t)} - (\mu + d + \delta) I(t) - \frac{aI(t)}{1+bI^2(t)}, \\
\frac{dR(t)}{dt} &= \frac{aI(t)}{1+bI^2(t)} + \delta I(t) - \mu R(t).
\end{aligned} \tag{2.1}$$

where $S(0) > 0, I(0) \geq 0$ and $R(0) \geq 0$.

In the model (2.1), the term $\frac{\beta S(t) I(t)}{1+\alpha I^2(t)}$ represents the M-H functional type incidence rate, here, β is the transmission rate of disease and α is the inhibitory effect. This nonlinear functional response was suggested by Sokol and Howell [1981]. It is understood that if we take $\alpha = 0$, the bilinear incidence rate [Gumel *et al.* (2006)] can also be derived from

this incidence rate. The term $\frac{aI(t)}{1+bI^2(t)}$, represent the M-H functional type treatment rate, where a and b are non-negative constants. The constants a and b are the cure rate of the infected people and limitation rate in the availability of treatment, respectively. Furthermore, it is assumed that all parameters of the model are positive, as required by the biological interpretation.

It should be mentioned that although the recovered population continues to make contact with other members of the population, it does not contribute to the transmission dynamics of the disease. Since the recovered population, $R(t)$, does not feature in the first two equations of the model. Therefore this equation can be omitted for the analysis without loss of generality. Thus, we consider the following reduced system for the mathematical analysis:

$$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta S(t)I(t)}{1+\alpha I^2(t)},$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t)}{1+\alpha I^2(t)} - (\mu + d + \delta)I(t) - \frac{aI(t)}{1+bI^2(t)}. \quad (2.2)$$

2.3 Basic properties of the model

For the system (2.2), we find that all solutions with nonnegative initial data will remain non-negative and bounded for all time. It can be shown as follows:
Let, the total population $N(t)$ is

$$N(t) = S(t) + I(t)$$

Then

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dI(t)}{dt} = A - \mu N(t) - (d + \delta)I(t) - \frac{aI(t)}{1 + bI^2(t)} \leq A - \mu N(t)$$

Then,

$$N(t) \leq N(0)e^{-\mu t} + \frac{A}{\mu}(1 - e^{-\mu t})$$

Thus,

$$\underset{t\to\infty}{Lim}\, sup\ N(t) \leq \frac{A}{\mu}.$$

Furthermore, $\frac{dN(t)}{dt} < 0$ if $N(t) > 0$. This shows that all solutions of the system (2.2) approaches towards the region D defined in Lemma 2.1 discussed below. Hence, the region D is positively invariant and solutions of the system (2.2) are bounded. Thus, we can establish the following lemma:

Lemma 2.1: The set $D = \{(S, I): 0 < S + I \leq \frac{A}{\mu}\}$ is a positively invariant region of the system (2.2).

Lemma 2.1 shows that all solutions of the system (2.2) are bounded and non-negative. Hence, the system (2.2) is well-posed mathematically and epidemiologically.

2.4 Equilibria and their stability analysis

The system (2.2) has two non-negative equilibria which are obtained by setting the right-hand sides of the system (2.2) equal to zero. They are as follows:

i. Disease-free equilibrium (DFE) $Q\left(\frac{A}{\mu}, 0\right)$.

ii. Endemic equilibrium (EE) $Q^*(S^*, I^*)$.

For the stability of equilibria, first, we find the basic reproduction number R_0 as given below:

2.4.1 Computation of the basic reproduction number (R_0)

The characteristic equation at $Q\left(\frac{A}{\mu}, 0\right)$ of the system (2.2) is given by

$$(\mu + \lambda)\left(\frac{\beta A}{\mu} - \mu - d - \delta - a - \lambda\right) = 0 \qquad (2.3)$$

One of the roots of Eq. (2.3) is given by $\lambda_1 = -\mu$ and the other root is given by $\lambda_2 = (\mu + d + \delta + a)(R_0 - 1)$.

where

$$R_0 = \frac{\beta A}{\mu(\mu + d + \delta + a)}.$$

This R_0 is known as the basic reproduction number.

Clearly, if $R_0 < 1$, then λ_2 is negative. Hence, we have the following theorem:

Theorem 2.1: DFE $Q\left(\frac{A}{\mu}, 0\right)$ is locally asymptotically stable when R_0 is less than unity and unstable when R_0 is greater than unity.

2.4.2 Analysis at $R_0 = 1$

Now, we check the behavior of the system (2.2) when $R_0 = 1$.

Let us redefine $S = x_1$ and $I = x_2$ then the system (2.2) can be rewritten as

$$\frac{dx_1(t)}{dt} = A - \mu x_1(t) - \frac{\beta x_1(t) x_2(t)}{1+\alpha {x_2}^2(t)} \equiv f_1 ,$$
$$\frac{dx_2(t)}{dt} = \frac{\beta x_1(t) x_2(t)}{1+\alpha {x_2}^2(t)} - (\mu + d + \delta) x_2(t) - \frac{a x_2(t)}{1+b {x_2}^2(t)} \equiv f_2. \quad (2.4)$$

The linearization matrix of the model the system (2.4) at $Q\left(\frac{A}{\mu}, 0\right)$ and on choosing the bifurcation parameter β given by $\beta^* = \frac{\mu(\mu+d+\delta+a)}{A}$ so $R_0 = 1$ when $\beta = \beta^*$ is given by

$$J = \begin{pmatrix} -\mu & -\frac{\beta^* A}{\mu} \\ 0 & \frac{\beta^* A}{\mu} - \mu - d - \delta - a \end{pmatrix} = \begin{pmatrix} -\mu & -\frac{\beta^* A}{\mu} \\ 0 & 0 \end{pmatrix}.$$

The matrix J admits a simple zero (null) eigenvalue at $R_0 = 1$ and other eigenvalue of $J(\lambda = -\mu)$ has a negative real part. Consequently, the linearization technique fails to determine the behavior of the system (2.4) [Dubey *et al.* (2016)]. Therefore, we use Theorem 4.1 of [Chavez and Song (2004)] which is based on center manifold theory. Then, the bifurcation constants a_1 and b_1 are given by

$$a_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q ,$$

and

$$b_1 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q$$

where $w = (w_1, w_2)^T$ and $u = (u_1, u_2)$ are the right and left eigenvectors of the matrix J associated with the null eigenvalue respectively. Thus, we get

$$u_1 = 0, u_2 = 1 \text{ and } w_1 = -\frac{\beta^* A}{\mu^2}, w_2 = 1$$

The partial derivatives related to the functions of the system (2.4) evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$$\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_Q = \beta^*, \left(\frac{\partial^2 f_2}{\partial x_2 \partial x_1}\right)_Q = \beta^*, \left(\frac{\partial^2 f_2}{\partial {x_1}^2}\right)_Q = 0, \left(\frac{\partial^2 f_2}{\partial {x_2}^2}\right)_Q = 0 \text{ and } \left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_Q = \frac{A}{\mu}$$

Therefore,

$$a_1 = u_2(2w_1 w_2\, \beta^* + {w_2}^2.0 + {w_1}^2.0)$$

$$= -2\frac{{\beta^*}^2 A}{\mu^2} < 0,$$

and

$$b_1 = u_2\left(w_2 \frac{A}{\mu}\right)$$

$$= \frac{A}{\mu} > 0.$$

It can be seen that a_1 is negative and b_1 is positive. Hence, bifurcation is forward. Therefore, we obtain the following theorem:

Theorem 2.2: The system (2.4) exhibits a forward bifurcation at DFE $Q\left(\frac{A}{\mu}, 0\right), R_0 = 1$ and bifurcation parameter $\beta = \beta^* = \frac{\mu(\mu+d+\delta+a)}{A}$.

2.4.3 Global stability of the disease-free equilibrium (DFE)

In this subsection, the global stability behavior of disease-free equilibrium $Q\left(\frac{A}{\mu}, 0\right)$ is discussed using a Lyapunov function.

Theorem 2.3: DFE $Q\left(\frac{A}{\mu}, 0\right)$ is globally asymptotically stable at $R_0 \leq 1$ when the following condition hold true simultaneously:

$$ab \leq \alpha(\mu + d + \delta).$$

Proof: Let L is the Lyapunov function defined as:

$$L = S - S_0 - S_0 \ln\frac{S}{S_0} + I \text{ , where } S_0 = \frac{A}{\mu}$$

Differentiating the L along the solutions of the system (2.2), then

$$\frac{dL}{dt} = \frac{\partial L}{\partial S}.\frac{dS}{dt} + \frac{\partial L}{\partial I}.\frac{dI}{dt}$$

$\Rightarrow$

$$\frac{dL}{dt} = -\frac{\mu(S - S_0)^2}{S} + \frac{(\mu + d + \delta + a)(R_0 - 1)I}{1 + \alpha I^2} + \left(\frac{ab - \alpha(\mu + d + \delta) - b\alpha(\mu + d + \delta)I^2)}{(1 + \alpha I^2)(1 + bI^2)}\right)I^3.$$

Since all parameters of the model are positive, it follows that $\frac{dL}{dt} < 0$ if $R_0 \leq 1$, $\frac{ab}{\alpha(\mu+d+\delta)} \leq 1$ simultaneously and $\frac{dL}{dt} = 0$ if $S = S_0 = \frac{A}{\mu}$ and $I = I_0 = 0$. Hence, L is a Lyapunov function on $D = \left\{(S, I): 0 < S + I \leq \frac{A}{\mu}\right\}$.

This implies that the largest compact invariant set in $\{(S, I) \in D: \frac{dL}{dt} = 0\}$ is the singleton set $\{Q\}$. From LaSalle's invariance principle [LaSalle (1976); Sastry (1999)] DFE is globally asymptotically stable.

2.4.4 Existence of endemic equilibrium (EE)

To find the conditions for the existence of endemic equilibrium $Q^*(S^*, I^*)$, the system (2.2) is rearranged to get S^* and I^* which gives:

$$S^* = \frac{\left((\mu + d + \delta)\left(1 + bI^{*2}\right) + a\right)\left(1 + \alpha I^{*2}\right)}{\beta\left(1 + bI^{*2}\right)},$$

and I^* is given by the equation

$$C_1 I^{*4} + C_2 I^{*3} + C_3 I^{*2} + C_4 I^* + C_5 = 0 \qquad (2.5)$$

where,

$C_1 = b\mu\alpha(\mu + d + \delta)$,

$C_2 = \beta b(\mu + d + \delta)$,

$C_3 = (\mu\alpha(\mu + d + \delta + a) + b\mu(\mu + d + \delta) - \beta Ab)$,

$C_4 = \beta(\mu + d + \delta + a)$,

$C_5 = (\mu(\mu + d + \delta + a) - \beta A) = \mu(\mu + d + \delta + a)(1 - R_0)$.

Using the Descartes' rule of signs [Wang (2004)], for $R_0 > 1$ there exists a unique positive real root of biquadratic equation (2.5) if the condition $C_1 > 0, C_2 > 0, C_3 > 0, C_4 > 0$ and $C_5 < 0$ is satisfied.

After getting the value of I^*, we can find the value of S^*. Thus, there exists a unique positive $Q^*(S^*, I^*)$ if the above condition holds true. Hence, we state the following theorem:

Theorem 2.4: If $R_0 > 1$, the system (2.2) admits a unique positive endemic equilibrium $Q^*(S^*, I^*)$.

Now, we show the uniform persistence of the system (2.2). Let D_1 denotes the interior of D and ∂D denotes the boundary of D. Epidemiologically, persistence implies that the subpopulation exists always and will not lead to elimination if initially, they exist. For this, we propose the following theorem:

Theorem 2.5: If $R_0 > 1$, the system (2.2) is uniformly persistent, this means that there exist a positive constant K such that every solution (S, I) of the system (2.2) with the initial data $(S(0), I(0)) \in D_1$ satisfies

$$\liminf_{t\to\infty} S(t) \geq K, \liminf_{t\to\infty} I(t) \geq K,$$

where K is independent of initial data in D_1

Proof: From theorem 2.4 for $R_0 > 1$, there exists a unique endemic equilibrium Q^*. From theorem 2.1 we know that $R_0 > 1$ implies that the DFE Q is unstable. By Theorem 4.3 in [Freedman *et al.* (1994)], the instability of Q, together with $Q \in \partial D$, imply the uniform persistence of the state variables of the system (2.2). Therefore, there exists a positive constant K such that every solution (S, I) of the system (2.2) with the initial data $(S(0), I(0)) \in D_1$ satisfies

$$\liminf_{t\to\infty} S(t) \geq K, \liminf_{t\to\infty} I(t) \geq K,$$

where K is independent of initial data in D_1 .

The uniform persistence, along with the boundedness of D, is equivalent to the existence of a compact set in the interior of D which is absorbing for the system (2.2) [Hutson and Schmit (1992)]. So, we have the following theorem:

Theorem 2.6: If $R_0 > 1$, then there exists a compact absorbing set $B \subset D_1$.

Next, the local stability of $Q^*(S^*, I^*)$ has been discussed.

2.4.5 Stability of endemic equilibrium

The local stability of $Q^*(S^*, I^*)$ is explored as follows: the variational matrix corresponding to $Q^*(S^*, I^*)$ of the system (2.2) is

$$J_{Q^*} = \begin{pmatrix} -\mu - \frac{\beta I^*}{1+\alpha I^{*2}} & -\frac{\beta S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2} \\ \frac{\beta I^*}{1+\alpha I^{*2}} & \frac{\beta S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2} - (\mu + d + \delta) - \frac{a(1-bI^{*2})}{(1+bI^{*2})^2} \end{pmatrix}.$$

The characteristic equation of the variational matrix J_{Q^*} is as follows:

$$\varepsilon^2 + p_1\varepsilon + p_2 = 0 \tag{2.6}$$

where,

$$p_1 = \left(2\mu + d + \delta + \frac{\beta I^*}{1+\alpha I^{*2}} + \frac{a}{(1+bI^{*2})^2} + \frac{\beta\alpha S^* I^{*2}}{(1+\alpha I^{*2})^2} - \left(\frac{abI^{*2}}{(1+bI^{*2})^2} + \frac{\beta S^*}{(1+\alpha I^{*2})^2}\right)\right),$$

$$p_2 = \left(\mu(\mu + d + \delta) + \frac{\beta(\mu+d+\delta)I^*}{1+\alpha I^{*2}} + \frac{a\beta I^*}{(1+\alpha I^{*2})(1+bI^{*2})^2} + \frac{\mu a}{(1+bI^{*2})^2} + \frac{\mu\beta\alpha S^* I^{*2}}{(1+\alpha I^{*2})^2} - \left(\frac{ab\beta I^{*3}}{(1+\alpha I^{*2})(1+bI^{*2})^2} + \frac{\mu ab}{(1+bI^{*2})^2} + \frac{\mu\beta S^*}{(1+\alpha I^{*2})^2}\right)\right).$$

The real part of the eigenvalues of the variational matrix J_{Q^*} is negative if and only if $p_1 > 0$ and $p_2 > 0$. Hence, the results are stated in the form of theorems given below:

Theorem 2.7: The endemic equilibrium $Q^*(S^*, I^*)$ is locally asymptotically stable if the following inequalities hold true simultaneously

$$\left(\frac{abI^{*2}}{(1+bI^{*2})^2} + \frac{\beta S^*}{(1+\alpha I^{*2})^2}\right) < M_1 \tag{2.7}$$

$$\left(\frac{ab\beta I^{*3}}{(1+\alpha I^{*2})(1+bI^{*2})^2} + \frac{\mu ab}{(1+bI^{*2})^2} + \frac{\mu\beta S^*}{(1+\alpha I^{*2})^2}\right) < M_2 \tag{2.8}$$

where

$$M_1 = \left(2\mu + d + \delta + \frac{\beta I^*}{1+\alpha I^{*2}} + \frac{a}{(1+bI^{*2})^2} + \frac{\beta\alpha S^* I^{*2}}{(1+\alpha I^{*2})^2}\right),$$

$$M_2 = \left(\mu(\mu + d + \delta) + \frac{\beta(\mu+d+\delta)I^*}{1+\alpha I^{*2}} + \frac{a\beta I^*}{(1+\alpha I^{*2})(1+bI^{*2})^2} + \frac{\mu a}{(1+bI^{*2})^2} + \frac{\mu\beta\alpha S^* I^{*2}}{(1+\alpha I^{*2})^2}\right).$$

Theorem 2.8: The endemic equilibrium $Q^*(S^*, I^*)$, whenever exists, is a saddle point if the inequality (2.7) and the following inequality holds true

$$\left(\frac{ab\beta I^{*3}}{(1+\alpha I^{*2})(1+bI^{*2})^2} + \frac{\mu ab}{(1+bI^{*2})^2} + \frac{\mu\beta S^*}{(1+\alpha I^{*2})^2}\right) > M_2 \tag{2.9}$$

Theorem 2.9: The endemic equilibrium $Q^*(S^*, I^*)$, whenever exists is unstable if the inequality (2.8) and the following inequality hold true

$$\left(\frac{abI^{*2}}{(1+bI^{*2})^2} + \frac{\beta S^*}{(1+\alpha I^{*2})^2}\right) > M_1 \tag{2.10}$$

Theorem 2.10: The system (2.2) exhibits the Hopf bifurcation near endemic equilibrium $Q^*(S^*, I^*)$ if the inequality (2.8) and following equality hold true

$$\left(\frac{abI^{*2}}{(1+bI^{*2})^2} + \frac{\beta S^*}{(1+\alpha I^{*2})^2}\right) = M_1 \tag{2.11}$$

Proof: Equality (2.11) implies that $p_1 = 0$ in Eq. (2.6) and inequality (2.8) implies that $p_2 > 0$. Thus, Eq. (2.6) has purely imaginary roots. From the theorem 2.7 and theorem 2.9, it follows that $Q^*(S^*, I^*)$ changes its behavior from stable to instability as the parameter β passes through the critical value $\beta = \beta^*$[Dubey *et. al.* (2015)], where

$$\beta^* = \frac{(1+\alpha I^{*2})^2}{S^*(1-\alpha I^{*2}) - I^*(1+\alpha I^{*2})}\left(2\mu + d + \delta + \frac{a(1-bI^{*2})}{(1+bI^{*2})^2}\right)$$

Again, we have

$$\frac{d}{d\beta}\left[tr\left(J_{Q^*}\right)\right]_{\beta=\beta^*} = \frac{S^*(1-\alpha I^{*2})-I^*(1+\alpha I^{*2})}{(1+\alpha I^{*2})^2} = \frac{1}{\beta^*}\left(\frac{a(1-bI^{*2})}{(1+bI^{*2})^2} + (2\mu + d + \delta)\right) \neq 0.$$

This condition is required for Hopf bifurcation to occur. Hence, the system (2.2) shows a Hopf bifurcation near the equilibrium point $Q^*(S^*, I^*)$ when $\beta = \beta^*$.

Now, we prove the non-existence of the periodic solution. For this, the following theorem has been proved:

Theorem 2.11: If $\alpha \geq b$, then the system (2.2) does not admit any periodic solution in the interior of the positive quadrant of the $S - I$ plane.

Proof: We take a real-valued function in the interior of the $S - I$ plane as given below:

$$H(S, I) = \frac{1 + \alpha I^2}{SI}$$

Let us consider

$$h_1(S, I) = A - \mu S - \frac{\beta SI}{1 + \alpha I^2},$$

$$h_2(S, I) = \frac{\beta SI}{1 + \alpha I^2} - (\mu + d + \delta)I - \frac{aI}{1 + bI^2}$$

Then we have

$$div\,(Hh_1, Hh_2) = \frac{\partial}{\partial S}(Hh_1) + \frac{\partial}{\partial I}(Hh_2)$$

$$= -\frac{A(1 + \alpha I^2)}{IS^2} - \frac{2\alpha(\mu + d + \delta)I}{S} - \frac{2a\,(\alpha - b)I}{S(1 + bI^2)^2}.$$

Clearly, it can be seen that the above expression can never be equal to zero when $\alpha > b$ and also, the sign of this expression will not be changed in the positive quadrant of the $S - I$ plane if the condition $\alpha \geq b$ holds true. Then, by Dulac's criterion [Sastry (1999)], it can be said that the system (2.2) does not have any periodic solution in the interior of the positive quadrant of the $S - I$ plane. Epidemiologically, this theorem implies that if the given condition holds true, then the disease will not reappear in the society.

Since the set D defined in the Lemma 2.1 is a positively invariant set, then the following theorem is a direct result of the Poincare-Bendixon theorem [Sastry (1999)] showing the existence of a limit cycle in the interior of the positive quadrant of the $S-I$ plane.

Theorem 2.12: If either inequality (2.8) and (2.10) or (2.9) are satisfied, then the system (2.2) has at least one limit cycle in the interior of the positive quadrant of the $S-I$ plane.

Epidemiologically, the above theorem implies that if the positive equilibrium $Q^*(S^*, I^*)$ is a saddle point or unstable then disease may reoccur in the society in the future.

2.5 Numerical simulation

In this section, the model is simulated numerically. The following set of tested parameters is used for the simulation [Dubey *et al.* (2015) & (2016)]:

$$A = 9\,, \beta = 0.005\,, \alpha = 0.05, \mu = 0.03\,, d = 0.005\,, \delta = 0.01\,, a = 0.02, b = 0.05.$$

At the above parameters values, the endemic equilibrium (Q^*) is calculated as $(264.0335, 23.6191)$ and the eigenvalues of the matrix J_{Q^*} are calculated as $(-0.0905, -0.0310)$. Hence, Q^* is stable.

The initial values are as follow:

$$S(0) = 280, I(0) = 8.$$

Fig. 2.1 shows the combined population of susceptible and infected individuals. It can be observed from the Fig.2.1 that the susceptible individuals are decreasing and infected individuals are increasing, and both populations approaching the endemic equilibrium.

Fig. 2.2 and Fig. 2.3 show the infected population at different values of the transmission rate (β) and inhibitory effect (α) respectively. It can be seen from the Fig. 2.2 that the number of infected individuals is increasing with the increase in transmission rate and in Fig. 2.3 the number of infected individuals are decreasing with increment in the inhibitory effect. In both the figures, the infected population is initially increasing and as time

passes, they approach to their steady state. This steady state may be achieved due to treatment or inhibitory effect.

Fig. 2.4 shows the behavior of the infected population at increased values of cure rate (a). It can be observed from the figure that the number of infected individuals is decreasing with the increment in the cure rate of infected.

Fig. 2.5 shows the total number of infected individuals at various values of $I(0)$ (*i.e.* initially infected population). It can be seen from this figure that the infected population approaches to the same endemic point for all values of $I(0)$(i. e. $I(0) = 8, 12, 15$).

Fig. 2.6 shows the difference between the infected individuals with M-H treatment rate and without M-H treatment rate. It can be observed from the figure that when M-H treatment is given to the infected population then the total number of infected individuals is less in comparison of the population without M-H treatment rate.

Fig. 2.7 shows the phase plane (S-I plot) and the occurrence of the limit cycle. For this, the following set of parameters values are taken:

$$A = 7\,, \beta = 0.021\,, \alpha = 0.0002, \mu = 0.002\,, d = 0.005\,, \delta = 0.01\,, a = 2, b = 0.0002.$$

2.6 Conclusions

In this chapter, we have proposed a new SIR epidemic model with the incidence rate of infection and treatment rate of infectives both are Monod-Haldane (M-H) functional type. We found that the model has two equilibria which are disease-free (Q) and endemic (Q^*). We found that the disease-free equilibrium (DFE) is locally asymptotically stable when the basic reproduction number (R_0) is less than unity and unstable when greater than unity which leads to the existence of the endemic equilibrium. Further, we also investigated that the model exhibits a forward bifurcation at $R_0 = 1$. The local and global stability of DFE (Q) and local stability of endemic equilibrium (Q^*) have been studied and it has been investigated that persistence or eradication of infection is uniformly persistent under the conditions stated in Theorem 2.5. Furthermore, we found that endemic equilibrium is locally asymptotically stable when the condition stated in theorem 2.7 hold true. The non-existence of periodic solutions under the condition stated in

theorem 2.11 shows that the infection will not reappear in the society in the future if these conditions are fulfilled. We also showed that the model exhibits a Hopf bifurcation at the endemic equilibrium. The numerical simulations of the model showed that the newly infectives can be controlled in the society due to the inhibitory effect and treatment rate of infectives according to Monod-Haldane functional.

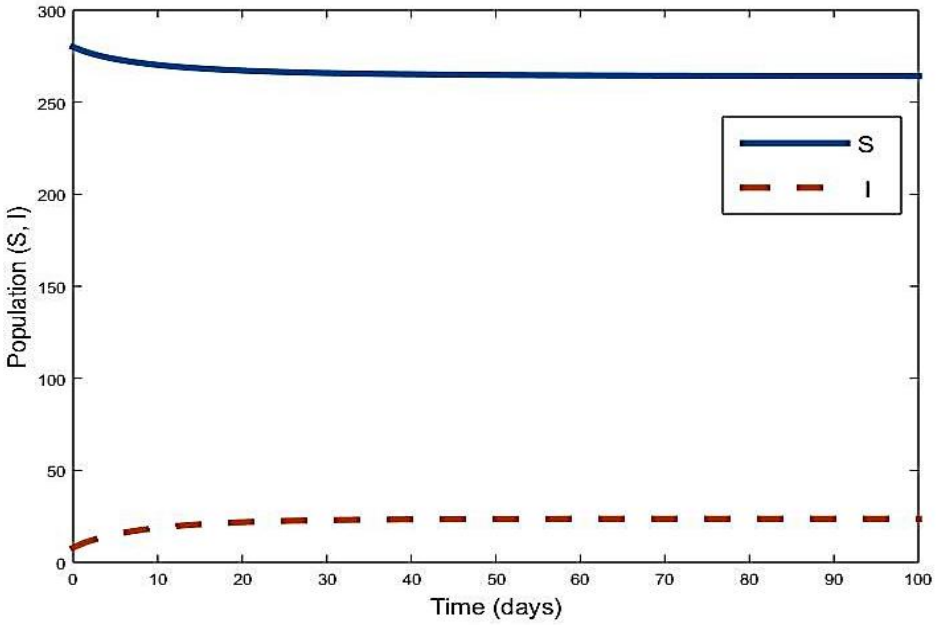


Fig 2.1: Susceptible (S) and infected (I) population.

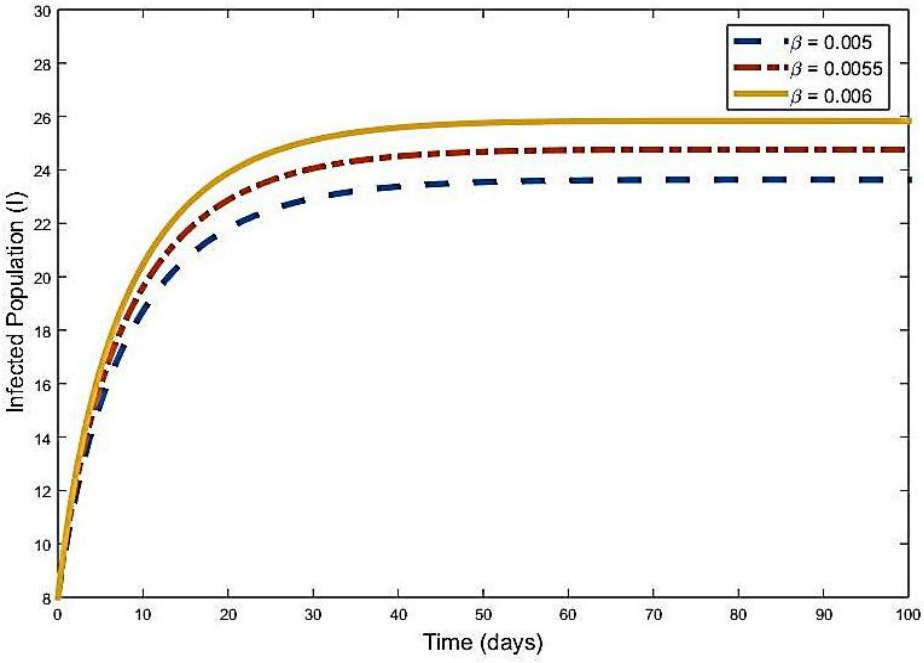


Fig 2.2: Infected population (I) at increased values of the transmission rate (β).

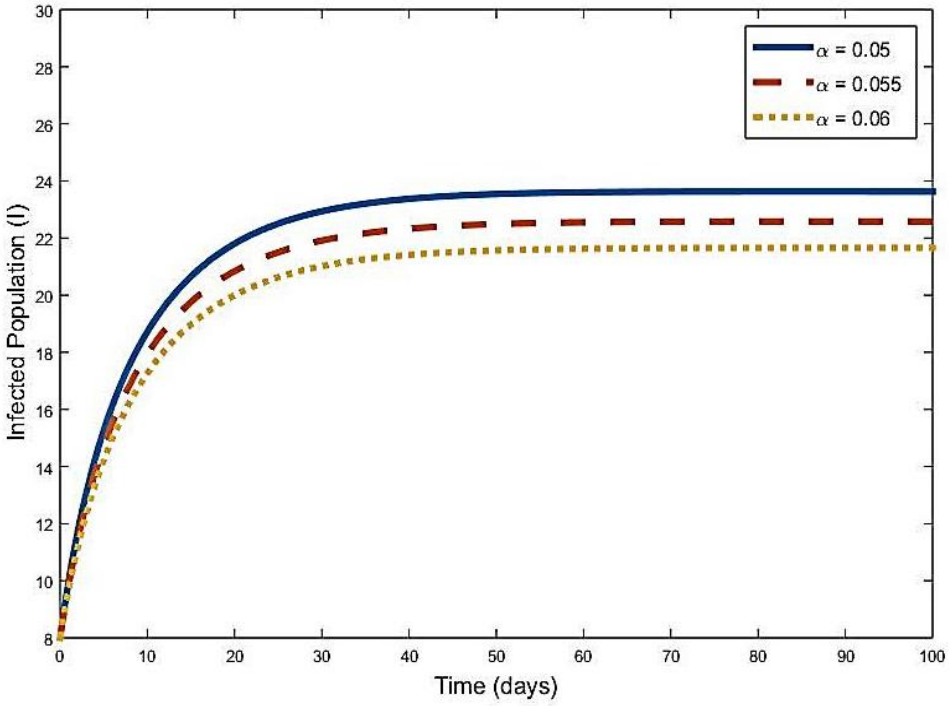


Fig 2.3: Infected population (I) at increased values of inhibitory effects (α).

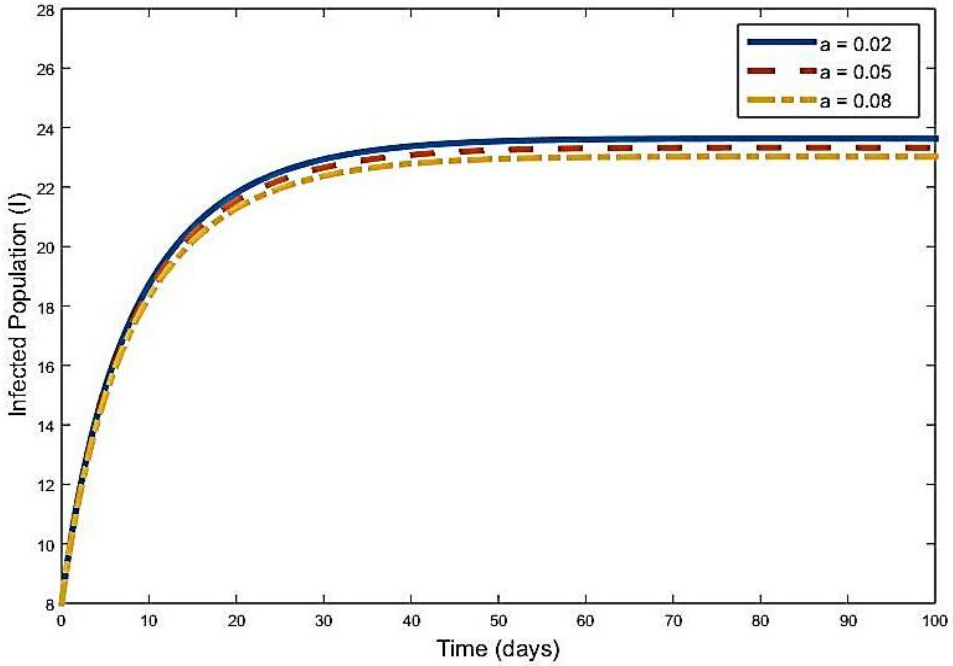


Fig 2.4: Infected population (I) at increased values of cure rate (a).

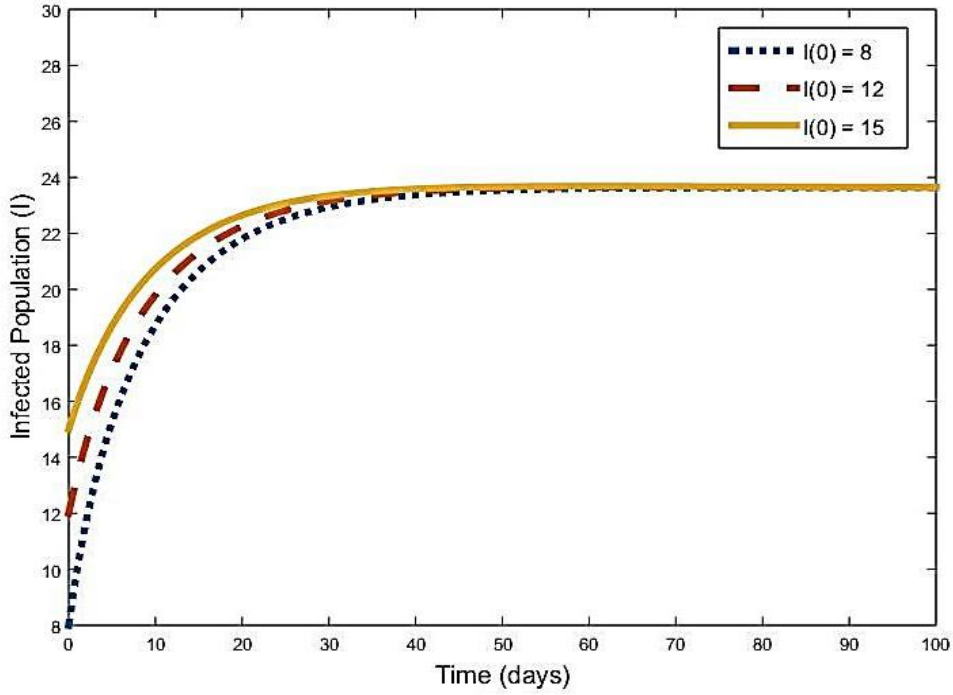


Fig 2.5: Infected population (I) at various values of initially infected population $I(0)$.

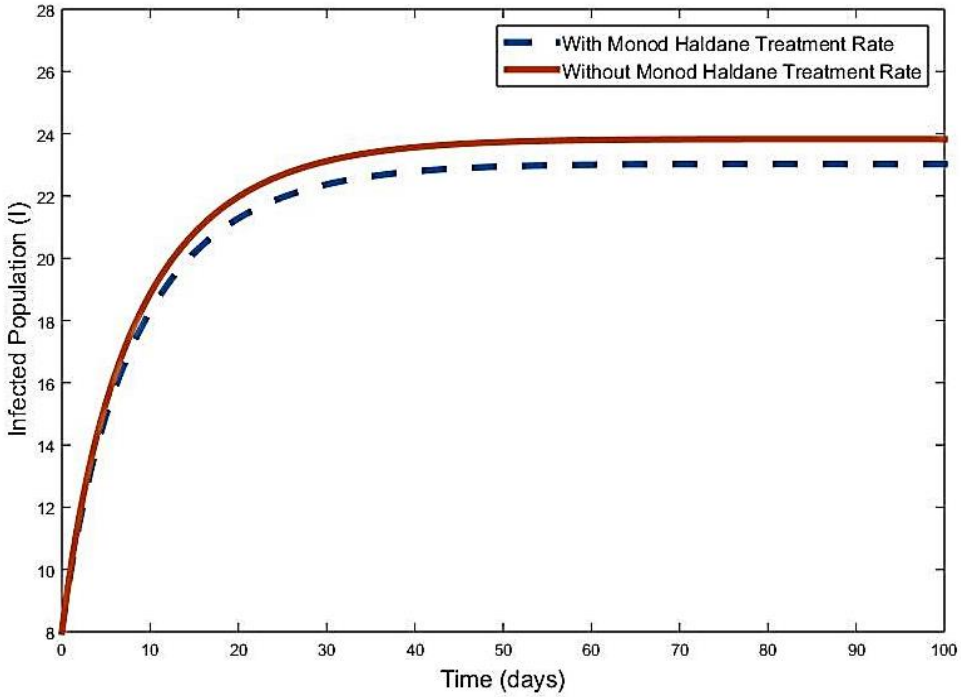


Fig 2.6: Infected population (I) with and without Monod-Haldane (M-H) treatment rate.

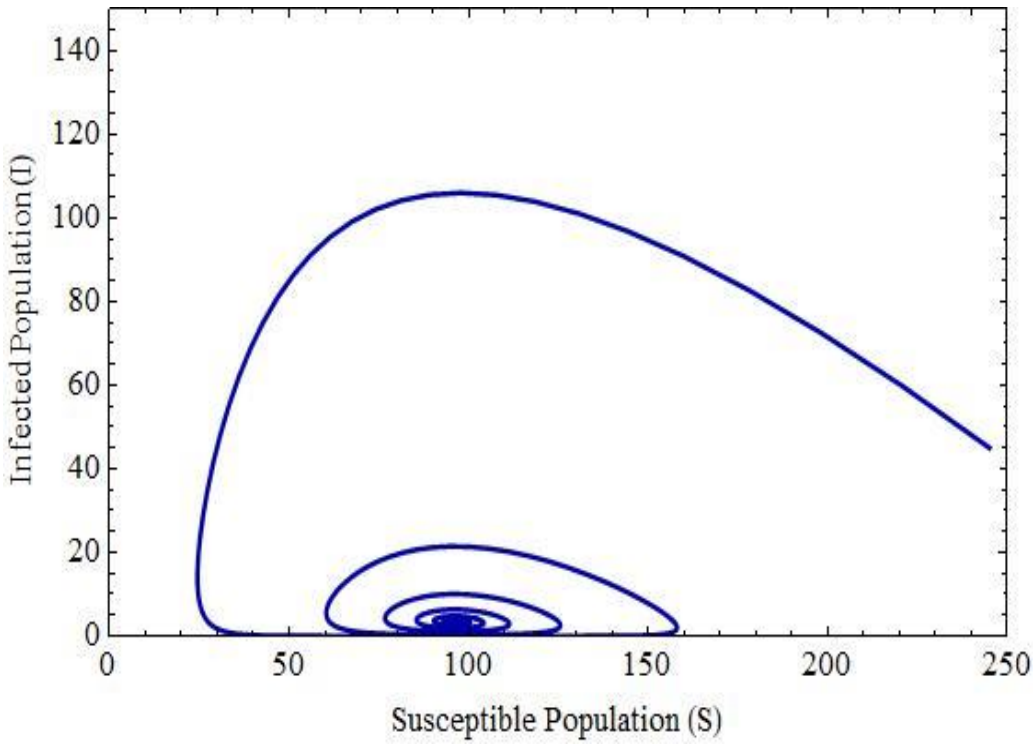


Fig. 2.7: S-I plot and occurrence of the limit cycle.

CHAPTER 3

A SIR EPIDEMIC MODEL WITH RATIO-DEPENDENT INCIDENCE AND HOLLING FUNCTIONAL TYPE II TREATMENT RATES

In this chapter, a nonlinear susceptible-infected-recovered (SIR) epidemic model has been proposed. The infection spreads very rapidly at the time of the outbreak of the disease (*e.g.* Cholera, Pneumonic plague, and Ebola), and at some stage, there are more infectives than susceptibles. This condition has been modeled by taking the incidence rate of infection as a ratio-dependent functional and the treatment rate as Holling type II functional. Two types of equilibrium points of the model have been obtained, which are named as disease-free equilibrium (DFE) and endemic equilibrium (EE) points. Stability of the model has been discussed for equilibrium points. The local stability of the model for DFE has been discussed by the basic reproduction number (R_0). The model for DFE is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. The stability of the model for DFE at $R_0 = 1$ has been examined using center manifold theory and showed that DFE exhibits a forward bifurcation. The local stability of the model at EE has also been discussed. Further, the model has been simulated numerically to explain the theoretical results.

3.1 Introduction

Proper and timely treatment methodology can substantially reduce the effect of the disease such as Cholera, Pneumonic plague, Ebola, etc. on society. In classical epidemic models, the treatment rate of infected individuals is assumed to be either constant or proportional to the number of infected individuals. However, we know that there are limited treatment resources available in the community in case of outbreak of an unknown epidemic. Therefore, this is very important to choose a suitable treatment rate of a disease. In the absence of effective therapeutic treatments and vaccines, the epidemical control strategies are based on taking appropriate preventive measures. Since every nation or city has its maximal limit with regards to the treatment of an infection, Wang and Ruan [2004] presented an arranged treatment function which describes that the treatment rate is relative to the number of infectives when the limit of treatment isn't reached, and otherwise, takes the maximal saturated level *i. e.*

$$H(I) = \begin{cases} b, & I > 0, \\ 0, & I = 0. \end{cases}$$

here b is a positive constant and I is the number of infected people. This appears to be more sensible than the usual linear function. Other than this, we realize that the effectiveness for treatment will be genuinely influenced if the infective people are postponed for treatment. To show the saturated phenomenon of the treatment discussed above, Zhou and Fan [2012] proposed a function $H(I)$ as given below:

$$H(I) = \frac{aI}{1 + bI}, I \geq 0, a > 0, b > 0.$$

where a is the cure rate. We can see that this function is more realistic than the previous ones. Firstly, for small I, $H(I) \sim aI$, whereas for large $I, H(I) \sim a/b$. This characterizes the saturated phenomenon of the treatment by a continuous and differentiable function. Further, $1/(1 + bI)$ describes the reverse effect of the infected being delayed for treatment. If $b = 0,$ saturated treatment function comes back to the direct one. The treatment rate function $H(I) = \frac{aI}{1+bI}$ is also known as Holling type II treatment rate. Dubey *et al.* [2015] have also utilized the Holling type II treatment rate to propose the dynamics of a SIR model.

In the dynamics of epidemics, it is well known that the form of incidence rate is an important factor. Arditi and Ginzburg [1989] proposed a ratio-dependent functional for a

low-density population of prey in prey-predator dynamics. Using the concept of this above important work, in the present study we introduce the incidence rate for the low density of susceptible individuals as a ratio-dependent functional type for disease dynamics. The per capita effect of infection on the susceptible population is modeled by a function G which is a function of the ratio $S/\gamma I$ of susceptible to infected. For the incidence rate of new infection, we incorporate the Arditi and Ginzburg type functional in Holling type II functional [Anderson and May (1982)] which is given below:

$$f(S,I) = G\left(\frac{S}{\gamma I}\right) I = \frac{\beta SI}{\alpha S + \gamma I}, \text{where } \beta, \alpha, \gamma > 0,$$

parameters β, α and γ represent the transmission rate of infection, the measure of inhibition due to susceptible and measure of inhibition due to infected, respectively.

In this chapter, a nonlinear SIR epidemic model is proposed with an incidence rate of the epidemic as a ratio-dependent functional type and treatment rate as Holling functional type II. Further, the basic properties of the model have been discussed. We also discuss the stability of the model at equilibrium points with the help of the basic reproduction number (R_0) and Routh-Hurwitz criterion.

3.2 Mathematical model

In this section, a nonlinear mathematical model for the epidemic has been proposed. For this, we consider the total population $P(t)$ at time t, with the immigration of susceptible individuals with a constant rate A. Further, the total population $P(t)$ has been divided into three classes (or compartments), which are named as: Susceptible class $S(t)$, infected class $I(t)$ and recovered class $R(t)$. It is assumed that the disease can spread due to the direct contact between susceptible and infective only. We also assume that the susceptible population is a low-density population. Let μ be the natural death rate of the population, d the death rate due to the disease and δ the recovery rate of infected individuals. The progression of an epidemic in different compartments is shown by the block diagram in Fig.3.1.

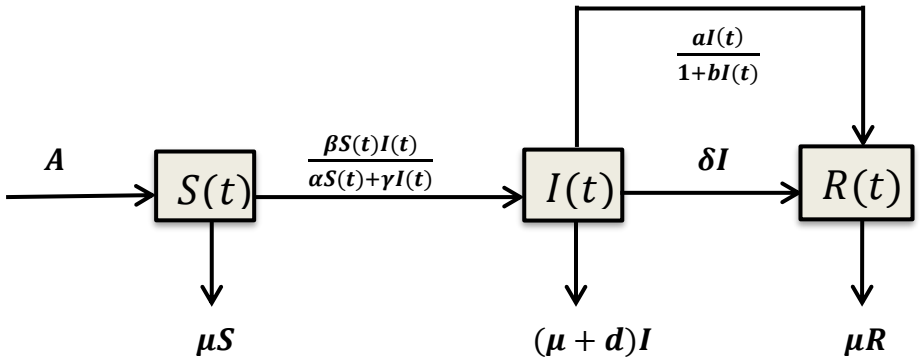


Fig. 3.1: Progression of the infection from susceptible (S) individuals through infected (I) and recovered (R) compartments for the model.

The rate of change in each compartment is given by the following system of nonlinear ordinary differential equations:

$$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta S(t)I(t)}{\alpha S(t)+\gamma I(t)},$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t)}{\alpha S(t)+\gamma I(t)} - (\mu + d + \delta)I(t) - \frac{aI(t)}{1+bI(t)}, \tag{3.1}$$

$$\frac{dR(t)}{dt} = \frac{aI(t)}{1+bI(t)} + \delta I(t) - \mu R(t).$$

where $S(0) > 0, I(0) > 0$ and $R(0) > 0$.

The term $\frac{\beta S(t)I(t)}{\alpha S(t)+\gamma I(t)}$ in the model (3.1) represents the ratio-dependent incidence rate where β is transmission rate of infection, and α is the effect of inhibition due to the susceptible individuals and γ is the effect of inhibition due to the infected individuals. The term $\frac{aI(t)}{1+bI(t)}$ in the model represent the Holling type II treatment rate, where a and b both are non-negative constants. The constants a and b both are named as cure rate and limitation rate in the treatment availability [Dubey *et al.* (2015)] respectively. Furthermore, it is assumed that all parameters of the model are positive.

From the above model (3.1), the first two equations do not depend on the third equation, and therefore this equation can be omitted for the analysis without loss of generality. Thus, it is enough to consider the following reduced system for mathematical analysis:

$$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta S(t)I(t)}{\alpha S(t)+\gamma I(t)},$$
$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t)}{\alpha S(t)+\gamma I(t)} - (\mu + d + \delta)I(t) - \frac{aI(t)}{1+bI(t)} \quad . \tag{3.2}$$

where $S(0) > 0$ and $I(0) > 0$.

3.3 Basic properties of the model

For the above system (3.2), we find a region of attraction which is given by Lemma 3.1.

Lemma 3.1: The set $D = \{(S, I): 0 < S + I \leq \frac{A}{\mu}\}$ is a positively invariant region for the disease transmission model given by the system (3.2).

Proof: Let, the total population $N(t)$ is

$$N(t) = S(t) + I(t)$$

Then

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dI(t)}{dt} = A - \mu N(t) - (d + \delta)I(t) - \frac{aI(t)}{1 + bI(t)} \leq A - \mu N(t)$$

Then,

$$N(t) \leq N(0)e^{-\mu t} + \frac{A}{\mu}(1 - e^{-\mu t})$$

Thus,

$$\underset{t\to\infty}{Lim\, sup}\ N(t) \leq \frac{A}{\mu}.$$

Furthermore, $\frac{dN(t)}{dt} < 0$ if $N(t) > 0$. This shows that all solutions of the system (3.2) approach the region D defined in Lemma 3.1 above. Hence, the region D is positively invariant and solutions of the system (3.1) are bounded.

The above Lemma 3.1 shows that all solutions of the model are non-negative and bounded. Thus, the system (3.2) is well-posed mathematically and epidemiologically.

3.4 Equilibria and their stability analysis

In this section, we analyze the system (3.2) by finding its equilibria and stability analysis. We observe that the model has two equilibrium points which are obtained by setting right-hand side of equations of the system (3.2) to zero, given as:

i. Disease-free equilibrium (DFE) point $Q(\frac{A}{\mu}, 0)$.

ii. Endemic equilibrium (EE) point $Q^*(S^*, I^*)$.

3.4.1 Computation of the basic reproduction number (R_0)

In this section, we compute the basic reproduction number R_0 for disease free equilibrium Q. The characteristic equation at $Q\left(\frac{A}{\mu}, 0\right)$ of the system (3.2) is given by

$$(\mu + \lambda)\left(\frac{\beta}{\alpha} - \mu - d - \delta - a - \lambda\right) = 0 \tag{3.3}$$

One of the roots of Eq. (3.3) is given by $\lambda_1 = -\mu$ and the other root is given by $\lambda_2 = (\mu + d + \delta + a)(R_0 - 1)$.

where

$$R_0 = \frac{\beta}{\alpha(\mu + d + \delta + a)}$$

Clearly, if $R_0 < 1$, then λ_2 is negative. Therefore, we can state the following theorem indicating the stability of disease-free equilibrium Q.

Theorem 3.1: The DFE $Q\left(\frac{A}{\mu}, 0\right)$ is locally asymptotically stable if basic reproduction number R_0 is less than one and unstable if R_0 is greater than one.

3.4.2 Analysis at $R_0 = 1$

In this section, we analyze the behavior of the system (3.2) when the basic reproduction number equals one.

Let us redefine $S = x_1$ and $I = x_2$ then the system (3.2) can be rewritten as

$$\frac{dx_1(t)}{dt} = A - \mu x_1(t) - \frac{\beta x_1(t)x_2(t)}{\alpha x_1(t)+\gamma x_2(t)} \equiv f_1 ,$$
$$\frac{dx_2(t)}{dt} = \frac{\beta x_1(t)x_2(t)}{\alpha x_1(t)+\gamma x_2(t)} - (\mu + d + \delta)x_2(t) - \frac{ax_2(t)}{1+bx_2(t)} \equiv f_2. \quad (3.4)$$

The linearization matrix of the system (3.2) at $Q\left(\frac{A}{\mu}, 0\right)$ and bifurcation parameter $\beta = \beta^* = \alpha(\mu + d + \delta + a)$ is given by

$$J = \begin{pmatrix} -\mu & -\frac{\beta^*}{\alpha} \\ 0 & \frac{\beta^*}{\alpha} - \mu - d - \delta - a \end{pmatrix}$$

The matrix J has a simple zero eigenvalue at $R_0 = 1$ and other eigenvalue of the matrix has a negative real part. At this stage, the linearization technique fails to capture the behavior of the system (3.4) [Dubey *et al.* (2016)]. Therefore, we applied the center manifold theory to study the behavior of the equilibrium. Then, from theorem 4.1 of [Chavez and Song (2004)], the bifurcation constants a_1 and b_1 are given by

$$a_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q ,$$

and

$$b_1 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q$$

where $w = (w_1, w_2)^T$ and $u = (u_1, u_2)$ are the right eigenvector and left eigenvector of the matrix J corresponding to the zero eigenvalue respectively. Then we have

$$u_1 = 0, u_2 = 1 \text{ and } w_1 = -\frac{\beta^*}{\alpha\mu}, w_2 = 1$$

The non-zero partial derivatives associated with the functions of the system (3.4) evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$$\left(\frac{\partial^2 f_2}{\partial {x_2}^2}\right)_Q = -\frac{2\mu\beta^*\gamma}{\alpha^2 A} \text{ and } \left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_Q = \frac{1}{\alpha}.$$

Therefore,

$$a_1 = u_2\left(2w_1w_2.0 + {w_2}^2.\left(-\frac{2\mu\beta^*\gamma}{\alpha^2 A}\right) + {w_1}^2.0\right)$$

$$= -\frac{2\mu\beta^*\gamma}{\alpha^2 A} < 0,$$

and

$$b_1 = u_2\left(w_2.\frac{1}{\alpha}\right)$$

$$= \frac{1}{\alpha} > 0.$$

It can be seen that a_1 is negative and b_1 is positive. Hence, by theorem 4.1 [Chavez and Song (2004)], we state the following theorem:

Theorem 3.2: DFE $Q\left(\frac{A}{\mu}, 0\right)$changes its stability from stable to unstable at $R_0 = 1$ and there exists a positive equilibrium as R_0 crosses one. Hence, the system (3.2) undergoes a forward bifurcation with bifurcation parameter $\beta = \beta^* = \alpha(\mu + d + \delta + a)$ at $R_0 = 1$.

3.4.3 Existence of endemic equilibrium (EE)

To find the conditions for the existence of equilibrium $Q^*(S^*, I^*)$ for which the disease is endemic in the population, the system (3.2) is rearranged to get S^* and I^* which gives:

$$S^* = \frac{\big((\mu + d + \delta + a) + b(\mu + d + \delta)I^*\big)\gamma I^*}{(R_0 - 1)(\alpha(\mu + d + \delta + a) + \alpha b I^*) + a\alpha b I^*}$$

and I^* is given by the equation

$$C_1 I^{*3} + C_2 I^{*2} + C_3 I^* + C_4 = 0 \qquad (3.5)$$

where,

$C_1 = b^2(\mu + d + \delta)(\alpha(\mu + d + \delta + a)(R_0 - 1) + (a\alpha + \gamma\mu)),$

$C_2 = -Ab^2\beta + ab(2a\alpha + \gamma\mu - \beta) + 2b(R_0 - 1)\big(a\alpha(\mu + d + \delta + a) + (\mu + d + \delta)\big) + Ab^2\alpha(\mu + d + \delta) + 2b(\mu + d + \delta)(a\alpha + \gamma\mu),$

$C_3 = -a^2\alpha + a(Ab\alpha - 2d\alpha + \beta - 2\alpha\delta - 2\alpha\mu + \gamma\mu) + \gamma\mu(d + \delta + \mu) + (d + \delta + \mu - 2Ab)\alpha(d + \delta + \mu + a)(R_0 - 1) + a\alpha(d + \delta + \mu - 2Ab),$

$C_4 = -A\alpha(a + d + \delta + \mu)(R_0 - 1).$

Using the Descartes' rule of signs [Wang (2004)], if any of the following conditions are satisfied for $R_0 > 1$, then Eq. (3.5) admits a unique positive real value of I^*:

i. $C_1 > 0, C_2 > 0, C_3 > 0,$ and $C_4 < 0$.

ii. $C_1 > 0, C_2 > 0, C_3 < 0, \text{and } C_4 < 0$.

iii. $C_1 > 0, C_2 < 0, C_3 < 0, \text{and } C_4 < 0$.

Remark: For $R_0 > 1, C_1 > 0, \text{and } C_4 < 0$, it is also possible to have $C_2 < 0$ and $C_3 > 0$. In this case, Eq. (3.5) admits three positive real value of I^*. In the above conditions, we have considered the case of unique endemic equilibrium only.

After getting I^*, we can obtain S^*. It implies the existence of a unique positive $Q^*(S^*, I^*)$ if one of the above conditions holds true. Hence, we state the following theorem:

Theorem 3.3: If $R_0 > 1$, the system (3.2) has a unique endemic equilibrium $Q^*(S^*, I^*)$.

Now, we show the uniform persistence of the system (3.2). Let D_1 be the interior of D and ∂D denotes the boundary of D. Epidemiologically, persistence implies that the subpopulation exists always and will not lead to elimination if initially, they exist. For this, we propose the following theorem:

Theorem 3.4: If $R_0 > 1$, the system (3.2) is uniformly persistent, which means that there exists a positive constant K such that every solution (S, I) of the system (3.2) with the initial data $(S(0), I(0)) \in D_1$ satisfies

$$\underset{t\to\infty}{Lim}\, inf\, S(t) \geq K, \underset{t\to\infty}{Lim}\, inf\, I(t) \geq K,$$

where K is independent of initial data in D_1.

Proof: The proof of this theorem is similar to the proof of theorem 2.5 as discussed in Chapter 2. Hence, we omitted.

The uniform persistence, along with the boundedness of D, is equivalent to the existence of a compact set in the interior of D which is absorbing for the system (3.2) [Hutson and Schmit (1992)]. So, the following theorem is stated:

Theorem 3.5: If $R_0 > 1$, then there exists a compact absorbing set $B \subset D_1$.

Next, the local stability of $Q^*(S^*, I^*)$ will be discussed.

3.4.4 Stability of endemic equilibrium

The local stability of $Q^*(S^*, I^*)$ is explored as follow:

The variational matrix corresponding to $Q^*(S^*, I^*)$ of the system (3.2) is given by

$$J_{Q^*} = \begin{pmatrix} -\mu - \frac{\beta\gamma I^{*2}}{(\alpha S^* + \gamma I^*)^2} & -\frac{\beta\alpha S^{*2}}{(\alpha S^* + \gamma I^*)^2} \\ \frac{\beta\gamma I^{*2}}{(\alpha S^* + \gamma I^*)^2} & \frac{\beta\alpha S^{*2}}{(\alpha S^* + \gamma I^*)^2} - (\mu + d + \delta) - \frac{a}{(1 + bI^*)^2} \end{pmatrix}$$

The characteristic equation of the variational matrix J_{Q^*} is given by the following equation

$$\tau^2 + \epsilon_1\tau + \epsilon_2 = 0 \tag{3.6}$$

where

$$\epsilon_1 = \left(-\frac{\beta\alpha S^{*2}}{(\alpha S^*+\gamma I^*)^2} + \frac{\beta\gamma I^{*2}}{(\alpha S^*+\gamma I^*)^2} + (2\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right),$$

$$\epsilon_2 = \left(\left(\mu + \frac{\beta\gamma I^{*2}}{(\alpha S^*+\gamma I^*)^2}\right)\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) - \frac{\mu\beta\alpha S^{*2}}{(\alpha S^*+\gamma I^*)^2}\right).$$

Clearly, the eigenvalues of the variational matrix J_{Q^*} have the negative real part if and only if $\epsilon_1 > 0$ and $\epsilon_2 > 0$. Thus, the results are stated in the form of theorems given below:

Theorem 3.6: The endemic equilibrium $Q^*(S^*, I^*)$ is locally asymptotically stable if the following inequalities hold true simultaneously

$$\frac{\beta\alpha S^{*2}}{(\alpha S^*+\gamma I^*)^2} < M_1 \tag{3.7}$$

$$\frac{\mu\beta\alpha S^{*2}}{(\alpha S^*+\gamma I^*)^2} < M_2 \tag{3.8}$$

where

$$M_1 = \left(\frac{\beta\gamma I^{*2}}{(\alpha S^* + \gamma I^*)^2} + (2\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right),$$

$$M_2 = \left(\left(\mu + \frac{\beta\gamma I^{*2}}{(\alpha S^* + \gamma I^*)^2}\right)\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right)\right).$$

Theorem 3.7: The endemic equilibrium $Q^*(S^*, I^*)$, whenever exists, is a saddle point if the inequality (3.7) and the following inequality hold true

$$\frac{\mu\beta\alpha S^{*2}}{(\alpha S^* + \gamma I^*)^2} > M_2 \tag{3.9}$$

Theorem 3.8: The endemic equilibrium $Q^*(S^*, I^*)$, whenever exists is unstable if the inequality (3.8) and the following inequality hold true

$$\frac{\beta\alpha S^{*2}}{(\alpha S^* + \gamma I^*)^2} > M_1 \tag{3.10}$$

Theorem 3.9: The system (3.2) exhibits the Hopf bifurcation near endemic equilibrium $Q^*(S^*, I^*)$ if the inequality (3.8) and following equality hold true

$$\frac{\beta\alpha S^{*2}}{(\alpha S^* + \gamma I^*)^2} = M_1 \tag{3.11}$$

Proof: Equality (3.11) implies that $\epsilon_1 = 0$ in Eq. (3.6) and inequality (3.8) implies that $\epsilon_2 > 0$. Thus, Eq. (3.6) has purely imaginary roots. From the theorem 3.6 and theorem 3.8, it follows that $Q^*(S^*, I^*)$ changes its behavior from stable to instability as β passes through the critical value $\beta = \beta^*$ [Dubey *et al.* (2016)], where

$$\beta^* = \left(2\mu + d + \delta + \frac{a}{(1+bI^*)^2}\right)\left(\frac{(\alpha S^* + \gamma I^*)^2}{\alpha S^{*2} - \gamma I^{*2}}\right)$$

Again, we have

$$\frac{d}{d\beta}\left[tr\left(J_{Q^*}\right)\right]_{\beta=\beta^*} = \frac{\alpha S^{*2} - \gamma I^{*2}}{(\alpha S^* + \gamma I^*)^2} = \frac{1}{\beta^*}\left(2\mu + d + \delta + \frac{a}{(1+bI^*)^2}\right) \neq 0.$$

Hence, the system (3.2) shows a Hopf bifurcation around (S^*, I^*) when $\beta = \beta^*$.

Theorem 3.10: If $S \leq \frac{\gamma}{\alpha b}$, then the system (3.2) does not admit any periodic solution in the interior of the positive quadrant of the S – I plane.

Proof: We define a real-valued function in the interior of the S-I plane as follows:

$$G(S,I) = \frac{\alpha S + \gamma I}{SI}$$

Let us consider

$$g_1(S,I) = A - \mu S - \frac{\beta SI}{\alpha S + \gamma I},$$

$$g_2(S,I) = \frac{\beta SI}{\alpha S + \gamma I} - (\mu + d + \delta)I - \frac{aI}{1 + bI}$$

Then we have

$$div\,(Gg_1, Gg_2) = \frac{\partial}{\partial S}(Gg_1) + \frac{\partial}{\partial I}(Gg_2)$$

$$= -\frac{A\gamma}{S^2} - \frac{\mu\alpha}{I} - \frac{(\mu + d + \delta)\gamma}{S} - \frac{a}{S}\left(\frac{\gamma - \alpha bS}{(1 + bI)^2}\right)$$

Clearly, it can be seen that the above expression can never be equal to zero and also, the sign of this expression will not be changed in the positive quadrant of the $S - I$ plane, if the condition $S \leq \frac{\gamma}{\alpha b}$ holds true. Then, by Dulac's criterion [Sastry (1999)], it can be said that the system (3.2) does not have any periodic solution in the interior of the positive quadrant of the S-I plane. Epidemiologically, this theorem implies that if the given condition holds true, then the disease will not reappear in the society.

Since the set D defined in the Lemma 3.1 is a positively invariant set, then the following theorem is a direct result of the Poincare-Bendixon theorem [Sastry (1999)] showing the existence of a limit cycle in the interior of the positive quadrant of the S – I plane.

Theorem 3.11: If either inequalities (3.8) and (3.10) or (3.9) are satisfied, then the system (3.2) has at least one limit cycle in the interior of the positive quadrant of the *S-I* plane.

Epidemiologically, the above theorem implies that if the positive equilibrium $Q^*(S^*, I^*)$ is a saddle point or unstable then disease may reoccur in the society in the future.

3.5 Numerical simulation

In this section, numerical simulation of the system (3.2) is performed. The following set of numerically experimental values of the parameters is taken for the simulation [Dubey *et al.* (2015) & (2016); Goel and Nilam (2019)]:

$$\mathrm{A} = 9, \alpha = 0.05, \beta = 0.005, \mu = 0.03, d = 0.01, \gamma = 0.05, a = 0.01, b = 0.005, \delta = 0.005.$$

At the above parameters values, the endemic equilibrium (Q^*) is calculated as $(114.6418, 107.9844)$ and the eigenvalues of the matrix J_{Q^*} are calculated as $(-0.0381147 \pm 0.0196544\, i)$. Hence, Q^* is stable.

Initial values are taken as

$$S(0) = 255, I(0) = 17$$

Fig. 3.2 shows the changes in susceptible and infected individuals with time. From Fig. 3.2 it can be seen that both susceptible and infected population are approaching to endemic equilibrium (Q^*) with the passage of time.

Fig. 3.3 shows the variation in the infected population at numerous values of initially infected $I(0)$. Clearly, it can be observed from the Fig. 3.3 that for all values of $I(0)$ infected population is approaching to same steady state.

Figs. 3.4, 3.5 and 3.6 demonstrate the infected population at numerous values of the transmission rate(β), measures of inhibition (α) taken by susceptibles, and measures of inhibition (γ) taken by infectives respectively. It is clear from the Figs 3.5 and 3.6 that the number of infected individuals is increasing when (α) and (γ) are decreasing and Fig. 3.4 demonstrates that the number of infected individuals is increasing when β is increasing.

Figs. 3.7 and 3.8 exhibit the changes in infected populations at various values of cure rate (a) and limitation rate (b) in treatment availability respectively. It can be seen form the

figures that the number of infected are decreasing with increment in cure rate while they are decreasing with the increment in limitation rate in treatment availability.

Fig. 3.9 indicates the difference in the total number of infected individuals between with and without Holling type II treatment rate. Clearly, it can be seen that when Holling type II treatment is given to the infected population then the number of newly infected become less while the number of newly infected individuals is higher when no Holling type II treatment given to infectives. Hence, it can be concluded that Holling type II treatment plays a vital role to control the infection.

Fig. 3.10 shows the phase plane (S-I plot) and the occurrence of the limit cycle. For this, the following set of tested parameters values are taken:

$A = 7\,, \beta = 0.064\,, \alpha = 0.0613, \gamma = 024,\ \mu = 0.002\,, d = 0.005\,, \delta = 0.01\,, a = 1, b = 0.0002.$

3.6 Conclusions

In this chapter, we contributed to the nonlinear dynamics of the epidemics by proposing a mathematical SIR epidemic model with a nonlinear incidence rate as the ratio-dependent functional type for the low density of susceptibles and treatment rate as Holling functional type II. Equilibria are obtained for the model, which are called disease-free and endemic. We investigated the stability of model equilibria and found that disease-free equilibrium (DFE) is locally asymptotically stable when the basic reproduction number is less than unity and unstable when greater than unity. We also investigated that model undergoes a transcritical forward bifurcation with bifurcation parameter β^* for DFE at $R_0 = 1$. Further, we investigated that endemic equilibrium (EE) is locally asymptotically stable when the conditions in theorem 3.6 hold true and unstable when the conditions stated in theorem 3.8 hold true. Furthermore, we showed that the model exhibits the Hopf bifurcation near to EE. Non-existence of periodic solutions under the condition stated in theorem 3.10 shows that the infection will not reappear in the society in the future if the mentioned conditions are fulfilled. Numerical simulations showed that

infection can be controlled in the society if the treatment is given to the infected accordingly managed by Holling type II treatment.

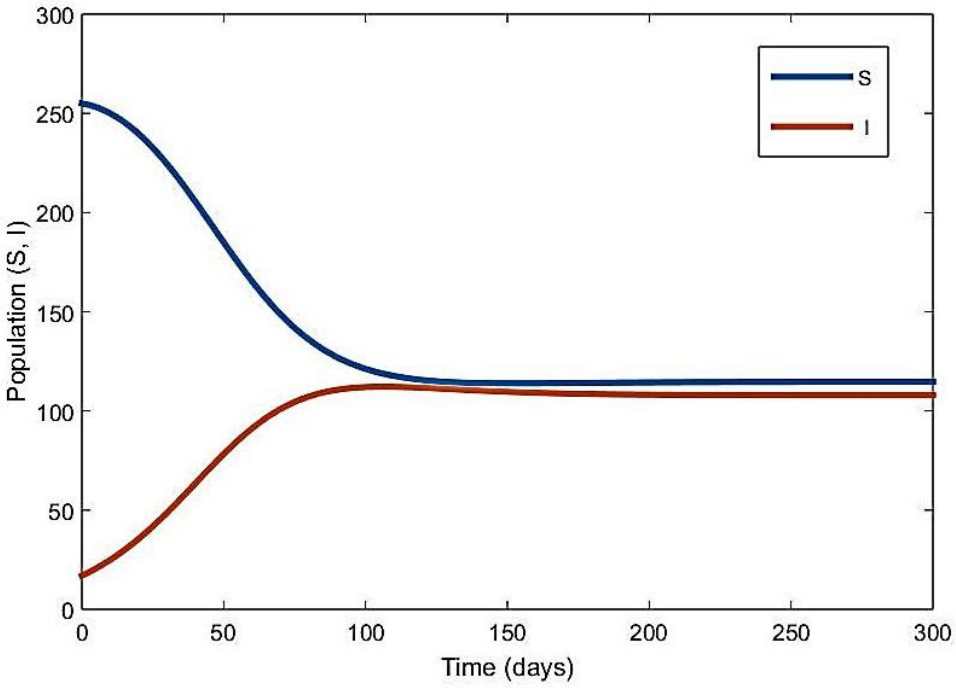


Fig. 3.2: Susceptible (S) and infected (I) population.

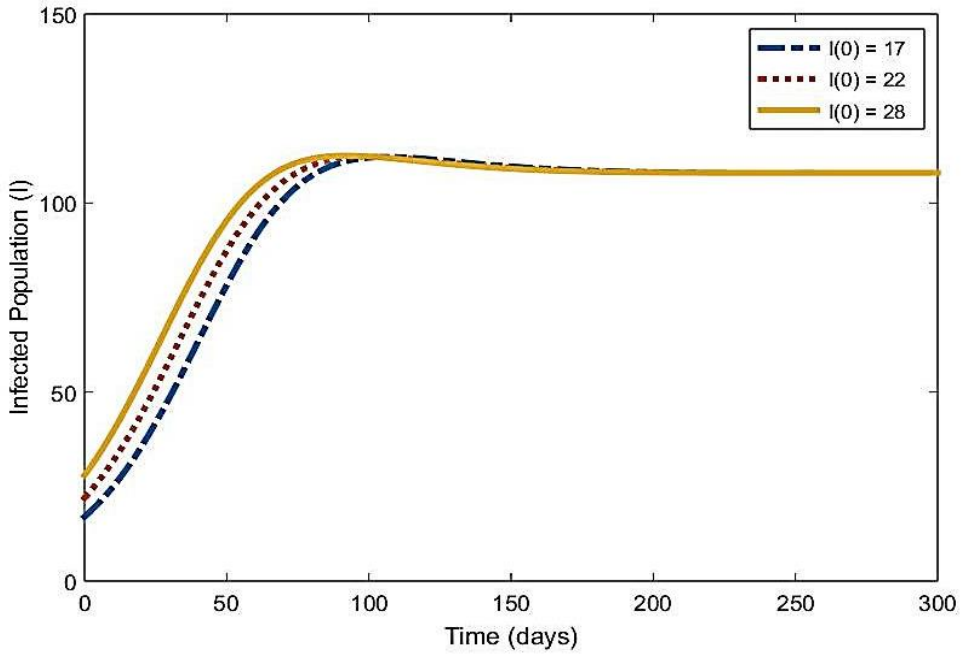


Fig 3.3: Infected population (I) at various values of initially infected population $I(0)$.

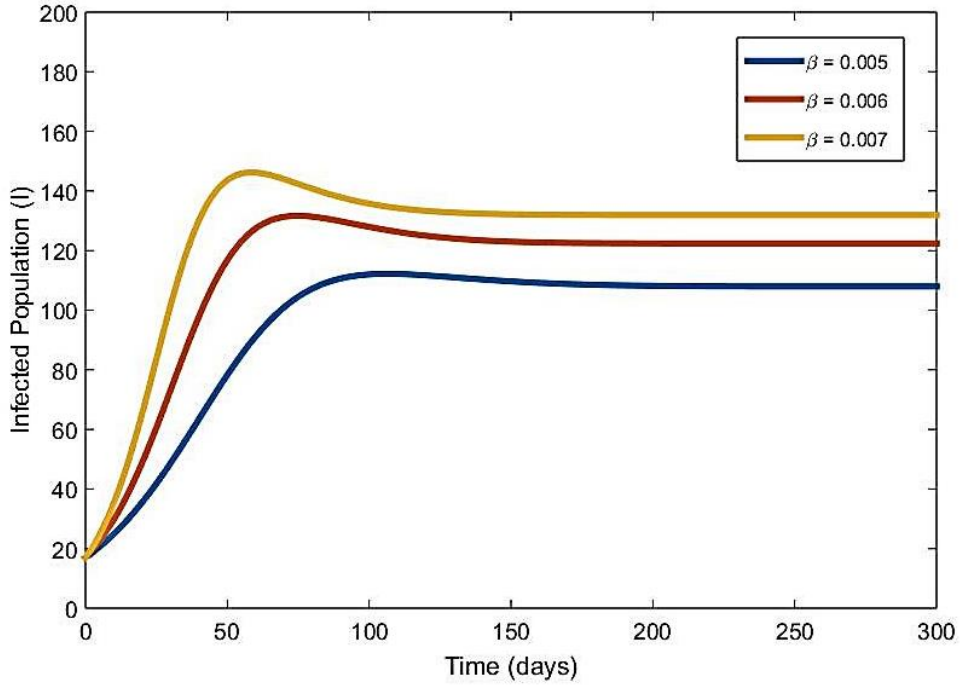


Fig. 3.4: Variation in the infected population (I) at various values of the transmission rate (β).

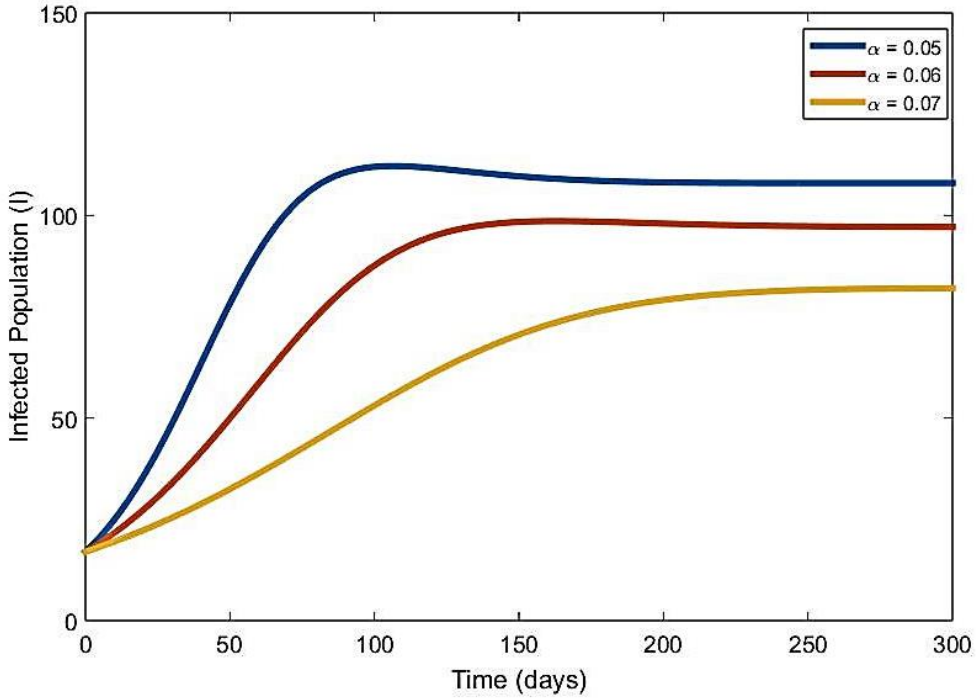


Fig. 3.5: Variation in the infected population (I) at various values of measure of inhibition (α) due to the susceptibles.

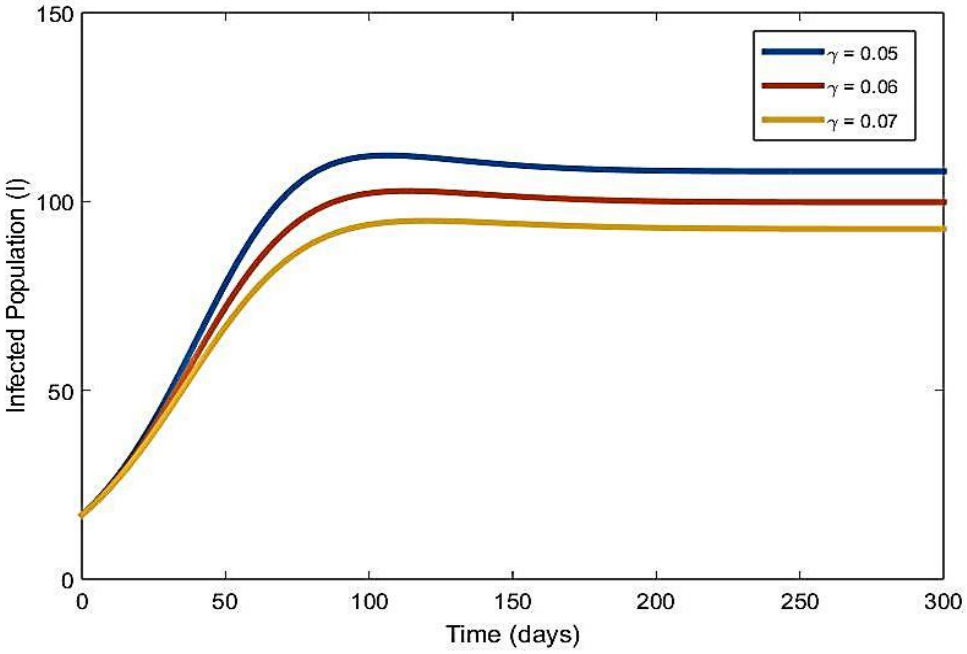


Fig. 3.6: Variation in the infected population (I) at various values of measure of inhibition (γ) due to the infectives.

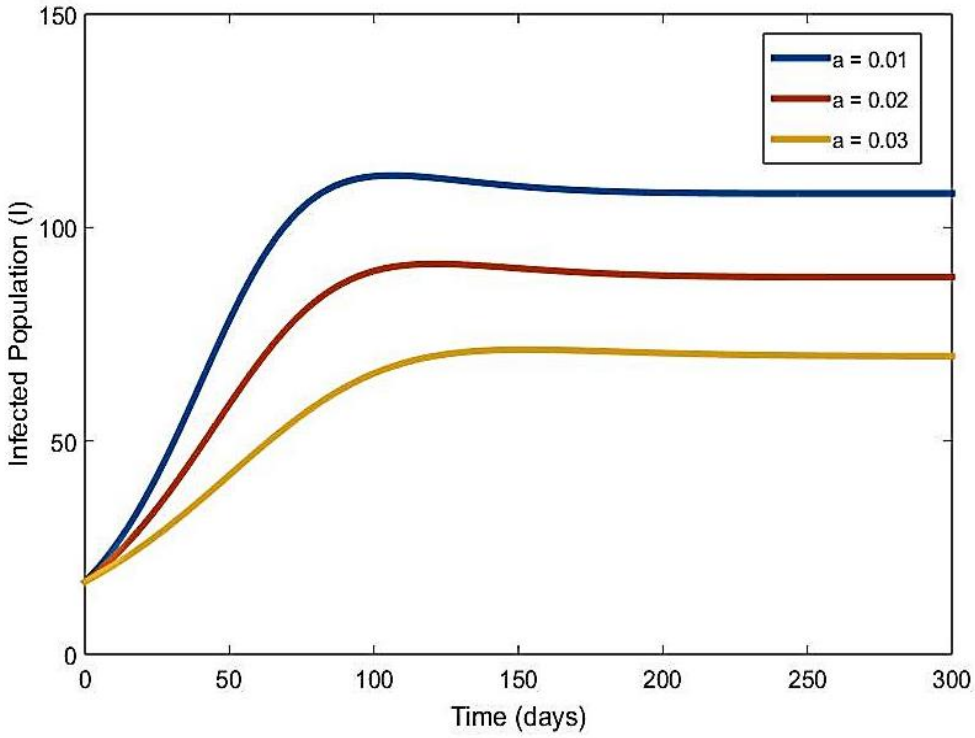


Fig. 3.7: Variation in the infected population (I) at various values of cure rate (a).

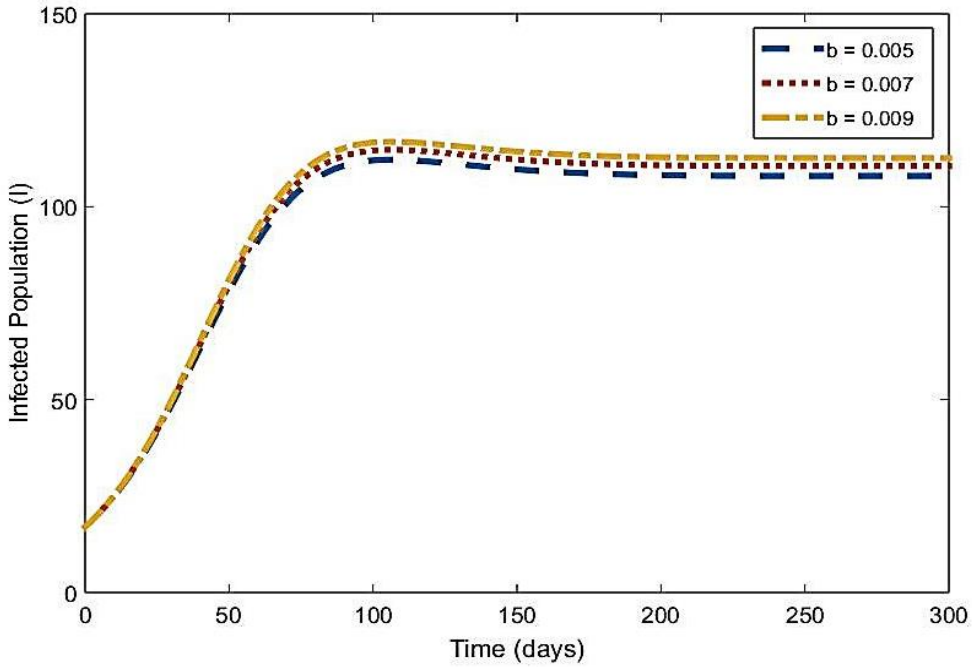


Fig. 3.8: Variation in the infected population (I) at various values of limitation rate (b) in treatment availability.

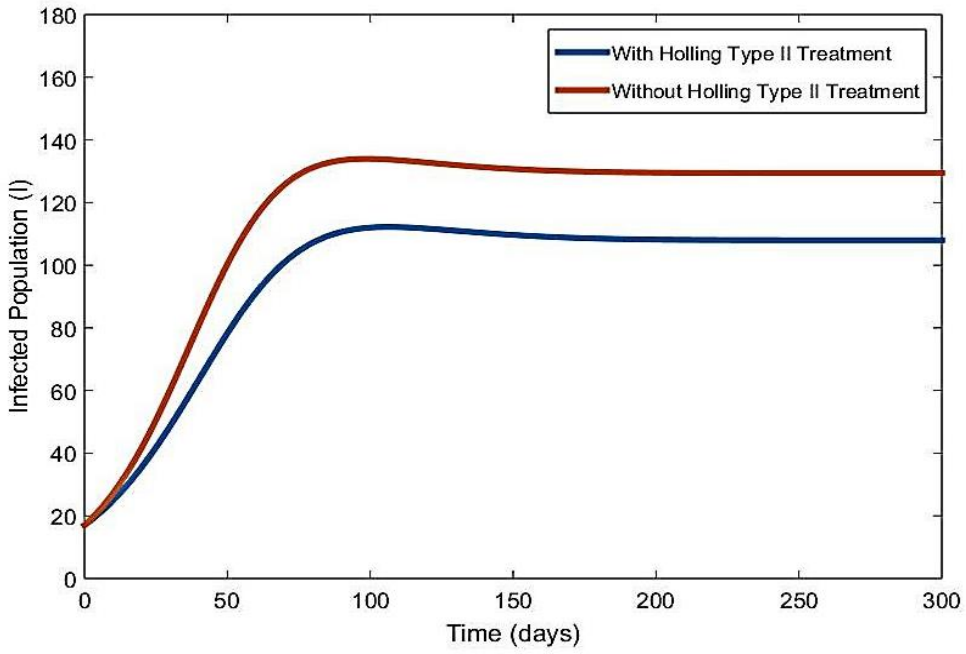


Fig. 3.9: Infected population (I) with and without Holling type II treatment rate.

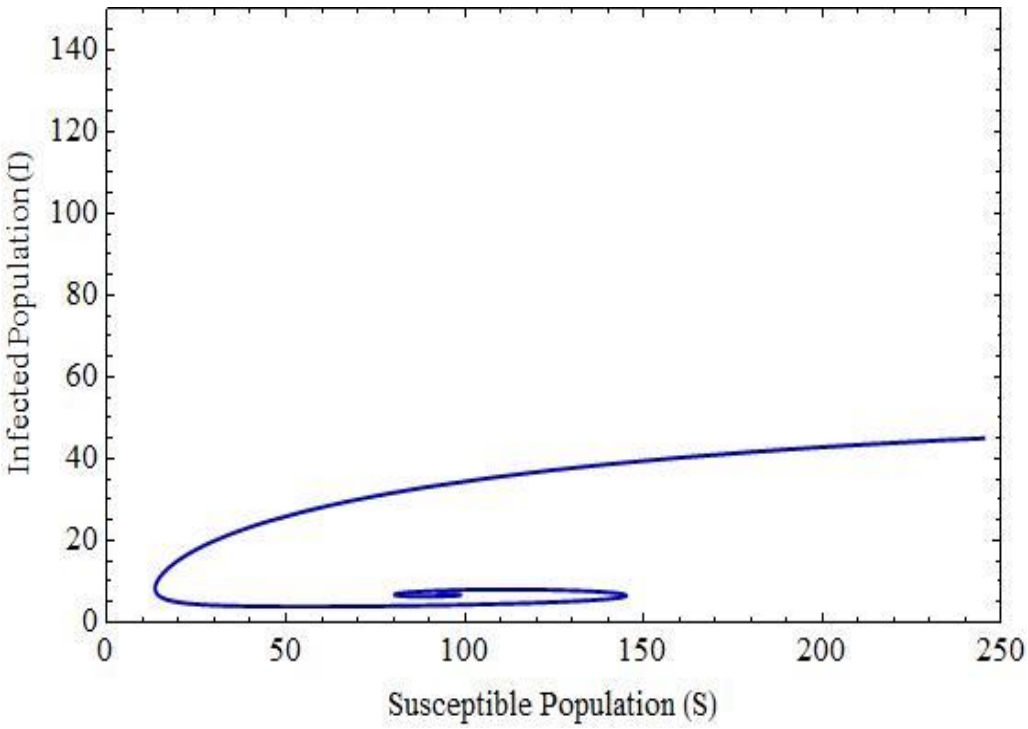


Fig. 3.10: S-I plot and occurrence of the limit cycle.

CHAPTER 4

TIME-DELAYED SIR EPIDEMIC MODEL WITH HOLLING FUNCTIONAL TYPE II INCIDENCE RATE AND DIFFERENT TREATMENT RATES

In this chapter, a SIR model has been studied for the transmission and control of an epidemic. The incidence rate of susceptible becoming infectious is extremely crucial in the spread of disease. The time delay in infectives in the incidence rate is also crucial. A susceptible-infected-recovered (SIR) mathematical model, with time delay as the incubation period of the disease, is proposed for the disease transmission dynamics. We have taken the incidence rate of new infection as Holling functional type II along with two different nonlinear treatment rates (Holling functional type II & III) for understanding the dynamics of the epidemic. The model stability has been analyzed in terms of the basic reproduction number. Mathematical analysis of the model demonstrates that disease-free equilibrium (DFE) is locally asymptotically stable when the basic reproduction number is less than unity. We have explored the stability of the model for disease-free equilibrium when the basic reproduction number equals to unity by center manifold theory. We likewise examined some stability conditions for endemic equilibrium (EE). Further, numerical simulations of the model have been carried out to support theoretical results.

4.1 Introduction

The structure of deterministic mathematical models for observing and controlling the spread of various human diseases is of public health interest in the light of the fact that the mathematics helps to formulate effective mechanisms for controlling their spread. After the first compartmental model given by Kermack and Mckendrick [1927], various mathematical models involving some complex assumptions [Michael *et al.* (1999); Wang (2002); Wang and Ruan (2004); Korobeinikov and Maini (2004); Gumel *et al.* (2006); Xiao and Ruan (2007); Naresh *et al.* (2009); Xu and Ma (2009b); Huang *et al.* (2010a); Zhang and Yaohong (2010); Buonomo and Lacitignola (2011); Hattaf *et al.* (2012); Zhou and Fan (2012); Hattaf and Yousfi (2013); Dubey *et al.* (2013), (2015) & (2016); Cui *et al.* (2017); Li and Zhang (2017); Goel and Nilam (2019)] have been proposed and considered, for instance, SIR, SIS, SEIR, and SEIRS models. In recent times, considerable attention has been paid to study the dynamics of epidemic models with epidemiologically meaningful time delays. In the context of disease transmission, delays can be caused by a variety of factors. The most well-known reasons for the delay are (i) the latency of the infection in a vector and (ii) the latency of the infection in an infected host [Huang *et al.* (2010a) & (2010b); Li and Liu (2014)]. In these cases, some time should elapse before the level of infection in the infected host or the vector is sufficiently high to transmit the infection further.

It is well known that disease transmission progress plays a vital role in epidemic dynamics; that is, different incidence rates can potentially change the behavior of the system. In many epidemic models, numerous incidence functions with or without delay are widely used in different epidemiological backgrounds [Li and Liu (2014)]. The incidence rate can also be modeled by many other kinds of more general functions. It is interesting whether the functional form of the incidence rate can change the epidemic dynamics or not. Liu *et al.* [1987] suggested a nonlinear saturated incidence function $\beta SI^l/(1+\alpha I^h)$ to model the impact of behavioral changes in particular communicable diseases, where βSI^l describes the infection force of the disease, $1/(1+\alpha I^h)$ measures the inhibition effect from the behavioral change of the susceptible people when the number of infectious people increases; l, h and β are all positive constants, and α is a nonnegative constant. The case $l = h = 1$, i.e. the incidence function becomes $\beta SI/(1+$

αI), was used by Capasso and Serio [1978] to represent a ‘‘protection measure’’ in modeling the cholera epidemics in Bari in 1973.

In classical epidemic models, the treatment rate of infected individuals is assumed to be either constant or proportional to the number of infected individuals. However, we know that there are limited treatment resources available in the community. Therefore, this is very important to choose a suitable treatment rate for a disease. Zhou and Fan [2012] proposed an epidemic model with following treatment rate (Holling type II):

$$H(I) = \frac{aI}{1+bI}\ , I \geq 0, a > 0, b > 0.$$

They have shown that with varying amount of medical resources and their supply efficiency, the target model admits various bifurcations. The detail explanations of the Holling type II treatment rate is already given in section 3.1. Further, to contribute more in the study of nonlinear treatment, we incorporate the Holling type III [Dubey *et al.* (2013) & (2016)] treatment rate, which characterizes the case in which removal rate is initially fast with increment in infectives and then it grows slowly and finally settles to a saturated value. Any subsequent expansion in infectives won’t influence the removal /recovery rate. This case relates to a known disease which has re-emergence and available treatment methods. The following functional form is known as Holling functional type III treatment rate:

$$H(I) = \frac{aI^2}{1+bI^2}, a, b, I > 0.$$

In this chapter, we propose and analyze a mathematical susceptible-infected-recovered model to gain a better understanding of transmission and subsequent control of the spread of infectious/communicable disease via a combination of nonlinear saturated incidence and different treatment rates. We incorporate time delay in incidence rate as the incubation period of the disease. We show the positivity and boundedness of the solution of the model. Further, we find the equilibrium points of the model and discuss the local and global stability of the equilibria. For the combination of Holling type II incidence and treatment rates, we explore only local stability of equilibria and for the combination of Holling type II incidence and Holling type III treatment rates, we explore the local as well as global stability of the equilibria. The stability of equilibria is discussed by using the basic reproduction number [Driessche and Watmogh (2002)], Routh-Hurwitz criterion,

and Lyapunov functional. Moreover, bifurcation analysis is also discussed. Our goal is to study the effect of nonlinear incidence along with time delay and Holling type II & III treatment rates, on the transmission dynamics of the infectious disease in the human population.

4.2 Mathematical model

In this section, an epidemic transmission model is being proposed. For this, it is assumed that the total population at time t is $N(t)$, which we divide into three compartments: susceptible individuals compartment $S(t)$, infected individuals compartment $I(t)$ and recovered individuals compartment $R(t)$. The definitions of susceptible, infected and recovered people have already been given in section 1.5. Further, it is assumed that infected people are being treated for the recovery with Holling type treatment rates. Furthermore, the susceptible population is recruited at a constant rate A. The natural death rate is supposed to be the same for all the individuals and is represented by μ. The contact capable of leading the infection in the human population is assumed as a rate β (transmission rate of infection). The protection measures (psychological or inhibitory effect) are considered at a rate α. The infected people also die due to disease related death at a rate d (disease-induced mortality). The rate of cure of infectious is a and limitation rate in the treatment of infected is b. The rate of recovery from the infection is γ. The progression of an epidemic in a different compartment is shown by the block diagram in Fig. 4.1 below:

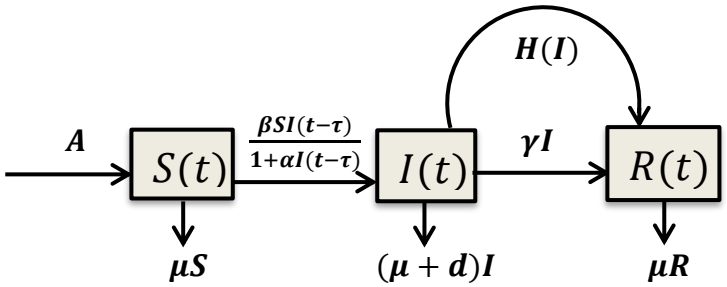


Fig. 4.1: Transfer diagram of the infection through various compartments.

These assumptions lead to the following nonlinear system of the delay differential equations to describe the changes in $S(t)$, $I(t)$ and $R(t)$ with respect to time t:

$$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta SI(t-\tau)}{1+\alpha I(t-\tau)},$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)} - (\mu + d + \gamma)I(t) - H(I(t)), \quad (4.1)$$

$$\frac{dR(t)}{dt} = H(I(t)) + \gamma I(t) - \mu R(t).$$

where $\tau > 0$ is a fixed time during which the infectious agents develop in the vector and it is only after that time that the infected vector can infect a susceptible person.
The initial conditions of (4.1) are given by
$S(\theta) = \varphi_1(\theta), I(\theta) = \varphi_2(\theta), R(\theta) = \varphi_3(\theta),\ \ \varphi_i(\theta) \geq 0, \theta \in [-\tau, 0], \varphi_i(0) > 0\ (i = 1,2,3)$ (4.1.1)

where $\big(\varphi_1(\theta), \varphi_2(\theta), \varphi_3(\theta)\big) \in C([-\tau, 0], \mathbb{R}^3_+)$. Here C denotes the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3_+$.

The term $H(I(t))$ is denoting the nonlinear saturated treatment rate. $H(I(t))$ is taken in following two form:

i. $H_1\big(I(t)\big) = \frac{a\, I(t)}{(1+b\, I(t))}$ (Holling type II treatment rate).

ii. $H_2\big(I(t)\big) = \frac{a\, I^2(t)}{(1+b\, I^2(t))}$ (Holling type III treatment rate).

The incidence rate $\frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)}$ represents the rate at the time $(t-\tau)$ at which susceptible individuals leave the susceptible class and enter in the infectious class at time *t*.

4.3 Basic properties of the model

For ecological regions, it is assumed that all parameters $A, \mu, \beta, d, \gamma, a, b$ are positive and state variables of the model (4.1) are nonnegative *i.e.* $(S, I, R) \in \mathbb{R}^3_+$. This can be seen as follows:

Theorem 4.1: The set $D = \{(S, I, R) \in \mathbb{R}^3_+ : 0 < S(t) + I(t) + R(t) \leq \frac{A}{\mu}\}$ is a positively invariant and attracting region for the model (4.1.)

Proof: We assume that the state variables and parameters of the model are non-negative. Since the right-hand side of the model (4.1) is continuous and differentiable, therefore the model is well-posed for $N(t) > 0$. The invariant region for the existence of the solutions is obtained as follows:

$$\frac{d[S(t)+I(t)+R(t)]}{dt} = \frac{dN(t)}{dt} \leq A - \mu N(t) \tag{4.2}$$

$$\Rightarrow \quad 0 < lim\ inf\ N(t) \leq lim\ sup\ N(t) \leq \frac{A}{\mu}\ (as\ t \to \infty). \tag{4.3}$$

Since $N(t) > 0$ on $[-\tau, 0]$, by assumption $N(t) > 0$ for all $t \geq 0$. Therefore, from Eq. (4.2) above, $N(t)$ can't approach to infinity in finite time. The model system is dissipative and therefore, the solution exists globally for all $t > 0$ in the invariant and compact set $D = \left\{(S, I, R) \in \mathbb{R}^3_+ : S(t) + I(t) + R(t) = N(t) \leq \frac{A}{\mu}\right\}$. As $N \to 0$, $S(t), I(t)$ and $R(t)$ also tend to zero. Hence, each of these terms tends to zero as $N(t)$ does. It is therefore natural to interpret these terms as zero when $N(t) = 0$.

Without loss of generality, for mathematical analysis of the above system (4.1) we consider the following reduced framework (system):

$$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta S I(t-\tau)}{1+\alpha I(t-\tau)},$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t) I(t-\tau)}{1+\alpha I(t-\tau)} - (\mu + d + \gamma) I(t) - H(I(t)), \tag{4.4}$$

with initial conditions

$$S(\theta) = \varphi_1(\theta), I(\theta) = \varphi_2(\theta), \varphi_i(\theta) \geq 0, \theta \in [-\tau, 0], \varphi_i(0) > 0\ (i = 1,2) \tag{4.4.1}$$

where $(\varphi_1(\theta), \varphi_2(\theta),) \in C([-\tau, 0], \mathbb{R}^2_+)$. Here C denotes the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^2_+$.

Theorem 4.2: All state variables $(S(t), I(t))$ of the system (4.4) with the initial condition (4.4.1) are nonnegative.

Proof: First we show that $S(t)$ is nonnegative for all $t \geq 0$. On the contrary, it is assumed that there exist $t_1 > 0$ be the first time such that $S(t_1) = 0$, then by the first equation of the system (4.4) we have $S'(t_1) = A > 0$, and hence $S(t) < 0$ for $t \in (t_1 -$

$\varepsilon, t_1)$, where $\varepsilon > 0$ is sufficiently small. This contradicts $S(t) > 0$ for $t \in [0, t_1)$. It follows that $S(t) > 0$ for $t > 0$. Now, we prove that positivity of solution $I(t)$. Integrating the second equation of the system (4.4) from 0 to t for $0 < t \leq \tau$, by applying the variation of constant formula and the step by step integration method, we obtain:

$$I(t) = I(0)e^{-(\mu+d+\gamma)t}.e^{\int_0^t F(S(\delta),I(\delta-\tau),I(\delta))\,d\delta}$$

here, $$F\big(S(\delta),\ I(\delta-\tau), I(\delta)\big) = \left(\frac{\beta S(\delta)I(\delta-\tau)}{(1+\alpha I(\delta-\tau))I(\delta)} - \frac{H\big(I(\delta)\big)}{I(\delta)}\right).$$

It is easy to see that $I(t) > 0$ for all $0 \leq t \leq \tau$. Integrating the second equation of the system (4.4) from τ to t for $\tau < t \leq 2\tau$ gives

$$I(t) = I(\tau)e^{-(\mu+d+\gamma)t}.e^{\int_\tau^t F(S(\delta),I(\delta-\tau),I(\delta))\,d\delta}$$

Note that $I(t) > 0$ for all $\tau \leq t \leq 2\tau$ and this procedure can easily carry on. It follows that for all $t > 0$, we have $I(t) > 0$. This completes the proof.

4.4 Equilibrium points

In this section, we obtain the equilibrium points of the system (4.4). The equilibrium solutions of a system with time delay are the same as those of the corresponding system with zero delays [Tipsri and Chinviriyasit (2014)]. The equilibria of the system (4.4) are calculated by putting the right-hand terms to zero which are as follows:

i. Disease-free equilibrium (DFE) $Q\left(\frac{A}{\mu}, 0\right)$,

ii. Endemic equilibrium (EE) $Q^*(S^*, I^*)$.

4.5 Stability analysis of the equilibria for the combination of Holling type II incidence and treatment rates

In this section, we discuss the local stability of model equilibria when the incidence and treatment rates are Holling functional type II. For the stability behavior, first, we compute the basic reproduction number (R_0).

4.5.1 Computation of basic reproduction number (R_0)

The characteristic equation of the system (4.4) evaluated at Q is given as:

$$(\mu + \lambda)\left(\frac{\beta A}{\mu}\, e^{-\lambda\tau} - \delta - a - \lambda\right) = 0, \text{ where } \delta = (\mu + d + \gamma) \tag{4.5}$$

Eq. (4.5) has a root $\lambda_1 = -\mu$ and other roots can be evaluated from

$$\frac{\beta A}{\mu}\, e^{-\lambda\tau} - \delta - a - \lambda = 0.$$

The term $\frac{\beta A}{\mu(\delta+a)} e^{-\lambda\tau}$ at $\tau = 0$, is known as the basic reproduction number, denoted by R_0. Therefore, we define the basic reproduction number R_0 of our model by $R_0 = \frac{\beta A}{\mu(\delta+a)}$.

4.5.2 Analysis for $R_0 \neq 1$

Clearly, Eq. (4.5) always has one negative root $\lambda_1 = -\mu$ and other roots are determined by the solution of the equation

$$\lambda + \delta + a - \frac{\beta A}{\mu}\, e^{-\lambda\tau} = 0 \tag{4.6}$$

Let

$$f(\lambda) = \lambda + \delta + a - \frac{\beta A}{\mu}\, e^{-\lambda\tau}$$

If $R_0 > 1$, for λ real,

$$f(0) = \delta + a - \frac{\beta A}{\mu} < 0, \lim_{\lambda\to\infty} f(\lambda) \to +\infty$$

Hence, there exists a positive real root of $f(\lambda) = 0$ if $R_0 > 1$.

If $R_0 < 1$, we assume that $Re\ \lambda \geq 0$.

We notice that

$$Re\ \lambda = \frac{\beta A}{\mu} e^{-Re\,\lambda\,\tau} \cos Im\,\lambda\,\tau - \delta - a \leq \frac{\beta A}{\mu} - \delta - a < 0.$$

a contradiction to our assumption. Hence, if $R_0 < 1$ then the root λ of Eq. (4.5) has a negative real part.

Thus, we state the following theorem:

Theorem 4.3: DFE (Q) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ for $\tau \geq 0$.

4.5.3 Analysis at $R_0 = 1$

i. For $\tau = 0$

We notice that the system (4.4) is being evaluated at $R_0 = 1$ and $\beta = \beta^* = \frac{\mu(\delta+a)}{A}$ has a zero eigenvalue and another eigenvalue is negative. The stability behavior of the equilibrium point at $R_0 = 1$ cannot be determined using linearization so we use center manifold theory [Sastry (1999)]. For this, we redefined $S = x_1$ and $I = x_2$, then the system (4.4) can be rewritten as

$$\frac{dx_1}{dt} = A - \mu x_1 - \frac{\beta x_1 x_2}{1+\alpha x_2} \equiv f_1,$$

$$\frac{dx_2}{dt} = \frac{\beta x_1 x_2}{1+\alpha x_2} - (\mu + d + \gamma)x_2 - \frac{a x_2}{1+b x_2} \equiv f_2. \qquad (4.7)$$

Let J^* be the Jacobian matrix at $R_0 = 1$ and bifurcation parameter $\beta = \beta^*$. Then

$$J^* = \begin{bmatrix} -\mu & -\frac{\beta^* A}{\mu} \\ 0 & 0 \end{bmatrix}$$

Let $u = [u_1, u_2]$ and $w = [w_1, w_2]^T$ be the left eigenvector and right eigenvector of J^*associated with the zero (null) eigenvalue. Then we have

$$u_1 = 0, u_2 = 1 \text{ and } w_1 = -\frac{\beta^* A}{\mu^2}, w_2 = 1.$$

The non-zero partial derivatives corresponding to the functions of the system (4.7) evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$$\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_Q = \beta^*, \left(\frac{\partial^2 f_2}{\partial x_2 \partial x_1}\right)_Q = \beta^*, \left(\frac{\partial^2 f_2}{\partial {x_2}^2}\right)_Q = \frac{-2\alpha\beta^* A}{\mu} + 2ab \text{ and } \left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_Q = \frac{A}{\mu}.$$

The bifurcation constants $a_1$and b_1 may be computed using the theorem 4.1 of [Chavez and Song (2004)] as follows:

$$a_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q$$

$$= u_2\left(2w_1 w_2 \beta^* + {w_2}^2\left(\frac{-2\alpha\beta^* A}{\mu} + 2ab\right)\right)$$

$$= 2\left(ab - \left(\frac{\beta^* A(\beta^* + \mu\alpha)}{\mu^2}\right)\right),$$

and

$$b_1 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q$$

$$= u_2\left(w_2 \frac{A}{\mu}\right)$$

$$= \frac{A}{\mu} > 0.$$

Thus, the following theorem may be stated using theorem 4.1(iv) of [Chavez and Song (2004)], as follows:

Theorem 4.4: For transcritical bifurcation, we have the following results:

- **i)** If $ab < \frac{\beta^* A(\beta^* + \mu\alpha)}{\mu^2}$, the behavior of DFE (Q) changes from stable to unstable at $R_0 = 1$ and there exists a positive equilibrium as R_0 crosses one. Thus, the system (4.4) undergoes a forward transcritical bifurcation at $R_0 = 1$.
- **ii)** If $ab > \frac{\beta^* A(\beta^* + \mu\alpha)}{\mu^2}$, there will be either a backward transcritical bifurcation or a saddle-node bifurcation.

ii. For $\tau > 0$

If $R_0 = 1$, then $\lambda = 0$ is a simple root of Eq. (4.5). Let $\lambda = x + iy$ any of the other solutions, then Eq. (4.6) change into:

$$x + iy + \delta + a - \frac{\beta A}{\mu} e^{-(x+iy)\tau} = 0 \qquad (4.8)$$

By using Euler's formula and by separating real and imaginary parts we can write

$$x + \delta + a = \frac{\beta A}{\mu}\cos y\tau \ e^{-x\tau},\ y = -\frac{\beta A}{\mu}\sin y\tau \ e^{-x\tau} \tag{4.9}$$

Observing that $R_0 = 1$ implies $\frac{\beta A}{\mu} = (\delta + a)$. Moreover, there exists a root satisfying both Eqs. (4.9), so they also satisfy the equation obtained by squaring and adding them member to member; we obtain

$$(x + \delta + a)^2 + y^2 = (\delta + a)^2 e^{-2x\tau}. \tag{4.10}$$

For Eq. (4.10) to be satisfied, we must have $x \leq 0$. Thus, we proposed the following theorem:

Theorem 4.5: DFE of the system (4.4) is linearly neutrally stable if $R_0 = 1$.

4.5.4 Existence and stability analysis of endemic equilibrium

To find the conditions for the existence of an equilibrium $Q^*(S^*, I^*)$ for which the disease is endemic in the population, the system (4.4) is rearranged to get $S^*$and I^* which gives

$$I^* = \frac{A - \mu S^*}{\beta S^* - (A - \mu S^*)\alpha},$$

where S^* is given by the following equation

$$C_1 {S^*}^2 + C_2 S^* + C_3 = 0 \tag{4.11}$$

here,

$C_1 = -\beta^2 + b\beta\mu - 2\alpha\beta\mu + b\alpha\mu^2 - \alpha^2\mu^2,$

$C_2 = a\beta - Ab\beta + 2A\alpha\beta + \beta\delta + a\alpha\mu - 2Ab\alpha\mu + 2A\alpha^2\mu - b\delta\mu + \alpha\delta\mu\ ,$

$C_3 = -aA\alpha + A^2 b\alpha - A^2\alpha^2 + Ab\delta - A\alpha\delta.$

Using Descartes' rule of signs [Wang (2004)], the existence of unique positive real I^* of Eq. (4.11) is required to satisfy any of the following conditions is satisfied:

i. $C_1 > 0, C_2 > 0$ and $C_3 < 0$.
ii. $C_1 > 0, C_2 < 0$ and $C_3 < 0$.
iii. $C_1 < 0, C_2 < 0$ and $C_3 > 0$.

After getting S^*, we can get I^*. Hence, a unique positive $Q^*(S^*, I^*)$ exists if one of the above conditions hold true.

The local stability of Q^* is explored as follows:

The characteristic equation of the system (4.4) at the endemic equilibrium point Q^* is given by the following second degree transcendental equation

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\tau} = 0,$$

where

$$p_0 = \left(\delta + \mu + \frac{a}{(1+bI^*)^2} + \frac{\beta I^*}{(1+\alpha I^*)}\right),$$

$$q_0 = \left(\delta\mu + \frac{\mu a}{(1+bI^*)^2} + \frac{\delta\beta I^*}{(1+\alpha I^*)} + \frac{a\beta I^*}{(1+\alpha I^*)(1+bI^*)^2}\right),$$

$$p_1 = \frac{-\beta S^*}{(1+\alpha I^*)^2},$$

$$q_1 = \frac{-\mu\beta S^*}{(1+\alpha I^*)^2}.$$

Theorem 4.6: For $\tau = 0$, Q^* is locally asymptotically stable if both $\frac{S^*}{I^*} \leq \frac{\delta}{\mu}$ and $\frac{S^*}{I^*} \leq 1$ hold true simultaneously.

Proof: At Q^*, the characteristic equation at $\tau = 0$ is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1) = 0, \tag{4.12}$$

It is easy to show that if $\frac{S^*}{I^*} \leq \frac{\delta}{\mu}$ and $\frac{S^*}{I^*} \leq 1$ are satisfied simultaneously then

$$p_0 + p_1 = \delta + \mu + \frac{a}{(1+bI^*)^2} + \frac{\beta I^*}{(1+\alpha I^*)} - \frac{\beta S^*}{(1+\alpha I^*)^2}$$

$$= \delta + \mu + \frac{a}{(1+bI^*)^2} + \frac{(\beta\alpha {I^*}^2 + (\beta I^* - \beta S^*))}{(1+\alpha I^*)^2} > 0 \text{ if } \frac{S^*}{I^*} \leq 1,$$

$$q_0 + q_1 = \delta\mu + \frac{\mu a}{(1+bI^*)^2} + \frac{\delta\beta I^*}{(1+\alpha I^*)} + \frac{a\beta I^*}{(1+\alpha I^*)(1+bI^*)^2} - \frac{\mu\beta S^*}{(1+\alpha I^*)^2}$$

$$= \delta\mu + \frac{\mu a}{(1+bI^*)^2} + \frac{a\beta I^*}{(1+\alpha I^*)(1+bI^*)^2} + \frac{(\beta\alpha\delta {I^*}^2 + (\delta\beta I^* - \mu\beta S^*))}{(1+\alpha I^*)^2} > 0 \text{ if } \frac{S^*}{I^*} \leq \frac{\delta}{\mu}.$$

Hence, by the definition of the Routh-Hurwitz criterion, Q^* is locally asymptotically stable when $\tau = 0$.

Theorem 4.7: For $\tau > 0$, Q^* is locally asymptotically stable if all three $\frac{1}{(1+bI^*)^2} \leq \frac{\mu}{\alpha} \leq 1$, $\frac{S^*}{I^*} \leq \frac{\delta}{\mu}$ and $\frac{S^*}{I^*} \leq 1$ are satisfied simultaneously.

Proof: At Q^*, the characteristic equation at $\tau > 0$ is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\tau} = 0, \tag{4.13}$$

For $\tau > 0$, corollary 2.4 of Ruan and Wei [2003] ensure that if the endemic equilibrium Q^* is unstable for particular values of delay then roots of the characteristic equation (4.13) must intersect the imaginary axis. Thus, to prove the stability of the system (4.4), we will use the contradictory assumption i.e. we assume that $\lambda = i\omega, \omega > 0$ is the root of the Eq. (4.13).

Substituting $\lambda = i\omega$ in equation (4.13), we get

$$-\omega^2 + q_0 + p_1\omega \sin\omega\tau + q_1 \cos\omega\tau + i\,(p_1\omega \cos\omega\tau - q_1 \sin\omega\tau + p_0\omega) = 0. \tag{4.14}$$

On separating real and imaginary part of Eq. (4.14), we get

$$p_1\omega \sin\omega\tau + q_1 \cos\omega\tau = \omega^2 - q_0 \tag{4.15}$$

$$p_1\omega \cos\omega\tau - q_1 \sin\omega\tau = -p_0\omega \tag{4.16}$$

On squaring and adding both sides of Eqs. (4.15) & (4.16) yield

$$\omega^4 + ({p_0}^2 - 2q_0 - {p_1}^2)\omega^2 + ({q_0}^2 - {q_1}^2) = 0 \tag{4.17}$$

Letting $\omega^2 = z_1$, Eq. (4.17) becomes

$${z_1}^2 + Pz_1 + T = 0 \tag{4.18}$$

Here, $P = ({p_0}^2 - 2q_0 - {p_1}^2)$ and $T = ({q_0}^2 - {q_1}^2)$

It is easy to show that if $\frac{1}{(1+bI^*)^2} \leq \frac{\mu}{\alpha} \leq 1$, $\frac{S^*}{I^*} \leq \frac{\delta}{\mu}$ and $\frac{S^*}{I^*} \leq 1$ are satisfied simultaneously then

$$P = ({p_0}^2 - 2q_0 - {p_1}^2)$$
$$= \left(\delta + \mu + \frac{a}{(1+bI^*)^2} + \frac{\beta I^*}{(1+\alpha I)}\right)^2 - 2\left(\delta\mu + \frac{\mu a}{(1+bI^*)^2} + \frac{\delta\beta I^*}{(1+\alpha I^*)} + \frac{a\beta I^*}{(1+\alpha I^*)(1+bI^*)^2}\right) - \left(\frac{-\beta S^*}{(1+\alpha I^*)^2}\right)^2$$

$$= \delta^2 + a^2 + \mu^2 + 2\delta a + 2a\mu\left(1 - \frac{1}{(1+bI^*)^2}\right) + \frac{2\beta aI^*}{(1+\alpha I^*)} + \frac{2\beta I^*}{\alpha(1+\alpha I^*)}\left(\frac{\mu}{\alpha} - \frac{1}{(1+bI^*)^2}\right) + \frac{\beta^2}{(1+\alpha I^*)^4}\left(I^{*2}(1+\alpha I^*)^2 - I^{*2} + (I^{*2} - S^{*2})\right) > 0,$$

$$T = ({q_0}^2 - {q_1}^2)$$

$$= \left(\delta\mu + \frac{\mu a}{(1+bI^*)^2} + \frac{\delta\beta I^*}{(1+\alpha I^*)} + \frac{a\beta I^*}{(1+\alpha I^*)(1+bI^*)^2}\right)^2 - \left(\frac{-\mu\beta S^*}{(1+\alpha I^*)^2}\right)^2$$

$$= \left(\delta\mu + \frac{\mu a}{(1+bI^*)^2} + \frac{\delta\beta I^*}{(1+\alpha I^*)} + \frac{a\beta I^*}{(1+\alpha I^*)(1+bI^*)^2}\right)^2 - \left(\frac{\delta\beta I^*}{(1+\alpha I^*)}\right)^2 + \frac{\beta^2}{(1+\alpha I^*)^4}\left(\delta^2 I^{*2}(1+\alpha I^*)^2 - \delta^2 I^{*2} + \left(\delta^2 I^{*2} - \mu^2 S^{*2}\right)\right) > 0.$$

Clearly, if $P > 0$ and $T > 0$ are satisfied simultaneously then by the definition of Routh-Hurwitz criterion Eq. (4.18) will always have roots with a negative real part. It contradicts to our assumption for instability that $\lambda = i\omega$ is the root of Eq. (4.13). Hence, the endemic equilibrium Q^* of the system (4.4) is locally asymptotically stable when $\tau > 0$.

4.6 Stability analysis of the equilibria for the combination of Holling type II incidence and Holling type III treatment rates

4.6.1 Computation of the basic reproduction number (R_0)

The characteristic equation of the system (4.4) at Q is given by the following equation:

$$(\mu + \lambda)\left(\lambda - \frac{\beta A}{\mu}\, e^{-\lambda\tau} + (\mu + d + \gamma)\right) = 0 \qquad (4.19)$$

The term $\frac{\beta A}{\mu(\mu+d+\gamma)}\, e^{-\lambda\tau}$ at $\tau = 0$ is known as basic reproduction number denoted as R_0. The threshold parameter R_0 is helpful in describing the spread of an infectious disease. Thus, R_0 for the system (4.4) is obtained as

$$R_0 = \frac{\beta A}{\mu(\mu + d + \gamma)}.$$

4.6.2 Analysis for $R_0 \neq 1$

One of the roots of Eq. (4.19) is given by $\lambda_1 = -\mu$ and the other roots can be obtained from

$$\frac{\beta A}{\mu} e^{-\lambda\tau} - (\mu + d + \gamma) - \lambda = 0 \tag{4.20}$$

Suppose that,

$$G(\lambda) = \lambda - \frac{\beta A}{\mu} e^{-\lambda\tau} + (\mu + d + \gamma) \tag{4.21}$$

If $R_0 > 1$, for λ real,

$G(0) = (\mu + d + \gamma) - \frac{\beta A}{\mu} = (\mu + d + \gamma)(1 - R_0)$, $\lim_{n\to\infty} G(\lambda) \to +\infty$.

Hence, $G(\lambda) = 0$ has a positive real root if $R_0 > 1$.

If $R_0 < 1$, we assume that $Re\ \lambda \geq 0$.

We see that

$$Re\ \lambda = \frac{\beta A}{\mu} e^{-Re\,\lambda\,\tau} \cos Im\ \lambda\ \tau - (\mu + d + \gamma) \leq (\mu + d + \gamma)(R_0 - 1) < 0.$$

a contradiction to our assumption. Hence, if $R_0 < 1$ then the characteristic root λ of Eq. (4.20) has a negative real part.

Thus, the following theorem is proposed:

Theorem 4.8: DFE Q is locally asymptotically stable (LAS) when $R_0 < 1$ and unstable when $R_0 > 1$.

4.6.3 Analysis at $R_0 = 1$

If $R_0 = 1$, then $\lambda = 0$ is a simple root of Eq. (4.19). Let $\lambda = x + iy$ be any of the other solutions, then Eq. (4.20) becomes:

$$x + iy + \mu + d + \gamma - \frac{\beta A}{\mu} e^{-(x+iy)\tau} = 0 \tag{4.22}$$

By using Euler's formula and by separating real and imaginary parts we can write

$$x + \mu + d + \gamma = \frac{\beta A}{\mu} \cos y\tau \; e^{-x\tau}, \; y = -\frac{\beta A}{\mu} \sin y\tau \; e^{-x\tau} \tag{4.23}$$

Observing that $R_0 = 1$ implies $\frac{\beta A}{\mu} = (\mu + d + \gamma)$. Moreover, if there exists a root satisfying both the equations of (4.23), then it also satisfies the equation obtained by squaring and adding them member to member,

$$(x + \mu + d + \gamma)^2 + y^2 = (\mu + d + \gamma)^2 e^{-2x\tau}. \tag{4.24}$$

For Eq. (4.24) to be verified, we must have $x \leq 0$. Thus, we propose the following theorem:

Theorem 4.9: DFE Q of the system (4.4) is linearly neutrally stable if $R_0 = 1$ for $\tau > 0$.

4.6.4 Existence and stability analysis of the endemic equilibrium

To establish the existence of an endemic equilibrium $Q^*(S^*, I^*)$, the right-hand side of the system (4.4) is equated to zero. Thus, the solution of the following set of algebraic equations gives the endemic equilibrium point $Q^*(S^*, I^*)$ for the proposed model system:

$$A - \mu S^* - \frac{\beta S^* I^*}{1+\alpha I^*} = 0, \; \frac{\beta S^* I^*}{1+\alpha I^*} - (\mu + d + \gamma) I^* - \frac{a I^{*2}}{1+b I^{*2}} = 0. \tag{4.25}$$

The solution of Eq. (4.25) gives

$$S^* = \frac{A(1 + \alpha I^*)}{\mu + (\mu\alpha + \beta) I^*},$$

and I^* is given by the following cubic equation

$$P(I^*) = K_0 + K_1 I^* + K_2 I^{*2} + K_3 I^{*3} = 0 \tag{4.26}$$

where

$K_0 = A\beta - \gamma\mu - \mu^2 - \mu d = \mu(\mu + d + \gamma)(R_0 - 1),$

$K_1 = -(\beta + \alpha\mu)(\gamma + \mu + d) - \mu a,$

$K_2 = \mu(\gamma + \mu + d) b(R_0 - 1) - (\beta + \alpha\mu) a,$

$K_3 = -(\beta + \alpha\mu)(\gamma + \mu + d)b.$

Next, we propose the following result for the existence of endemic equilibrium:

Theorem 4.10: If $R_0 > 1$, then there are either one or three positive endemic equilibria, if all equilibria are simple roots and if $R_0 \leq 1$ then no positive endemic equilibria exist.

Proof: It is evident from the expressions of K_0, K_1, K_2 and K_3 that K_1 and K_3 are always negative. Suppose $R_0 > 1$ $(K_0 > 0)$. The leading coefficient K_3 is negative. Hence, $\lim_{I^* \to \infty} P(I^*) = -\infty$. Also, note that $P(0) > 0$ when $R_0 > 1$. $P(I^*)$ is a continuous function of I^* and by applying fundamental theorem of algebra, it is evident that Eq. (4.26) can have at most three real roots. By a geometric argument, it is readily seen that there is either one or three positive endemic equilibria, if all equilibria are simple roots. Whereas, when $R_0 < 1$ then the coefficients K_0, K_1, K_2 and K_3 all are negative then by a fundamental theorem of algebra, we know that Eq. (4.26) can't have any positive real root and when $R_0 = 1$ then the coefficients K_0 is zero and other coefficients K_1, K_2 and K_3 all are negative then by a fundamental theorem of algebra, this polynomial cannot have any positive real root.

To discuss the local stability of the system (4.4) at Q^*, we linearize the system (4.4) at Q^* and obtained the characteristic equation which is as given below:

$$\lambda^2 + M_1\lambda + N_1 + (M_2\lambda + N_2)e^{-\lambda\tau} = 0 \tag{4.27}$$

$$M_1 = \frac{(1+I^*\alpha)\left(\gamma+2\mu+d+I^{*3}\left(\beta+\alpha(\gamma+2\mu+d)\right)b^2+I^*\left(\beta+\alpha(\gamma+2\mu+d)+2(\gamma b+2\mu b+db+a)\right)\right)}{(1+I^*\alpha)^2(1+I^*b)^2} +$$

$$\frac{(1+I^*\alpha)I^{*2}\left(2\beta b+(\gamma+2\mu+d)b^2+2\alpha(\gamma b+2\mu b+db+a)\right)}{(1+I^*\alpha)^2(1+I^*b)^2},$$

$$M_2 = \frac{-S^*\beta}{(1+I^*\alpha)^2},$$

$$N_1 = \frac{(1+I^*\alpha)(\mu+I^*(\beta+\alpha\mu))(\gamma+\mu+d)}{(1+I^*\alpha)^2},$$

$$N_2 = \frac{-S^*\beta\mu}{(1+I^*\alpha)^2}.$$

Theorem 4.11: For $\tau > 0$, the system (4.4) at Q^* is locally asymptotically stable if ${M_1}^2 - 2N_1 - {M_2}^2 > 0$ and ${N_1}^2 - {N_2}^2 > 0$ hold true simultaneously.

Proof: At endemic equilibrium Q^* the characteristic equation of the system for $\tau > 0$ is given by the Eq. (4.27)

$$\lambda^2 + M_1\lambda + N_1 + (M_2\lambda + N_2)e^{-\lambda\tau} = 0$$

For $\tau > 0$, by corollary 2.4 of Ruan and Wei [2003], a characteristic root of the Eq. (4.27) must cross the imaginary axis for instability, for a specific value of τ. Accordingly, let $\lambda = i\omega, \omega > 0$ is the root of the characteristic equation (4.27). Putting $\lambda = i\omega$ in the Eq. (4.27) gives:

$$-\omega^2 + N_1 + M_2\omega \sin\omega\tau + N_2 \cos\omega\tau + i\,(M_2\omega \cos\omega\tau - N_2 \sin\omega\tau + M_1\omega) = 0 \tag{4.28}$$

Using Euler's formula and separating the real and imaginary part of Eq. (4.28), we get

$$M_2\omega \sin\omega\tau + N_2 \cos\omega\tau = \omega^2 - N_1 \tag{4.29}$$

$$M_2\omega \cos\omega\tau - N_2 \sin\omega\tau = -M_1\omega \tag{4.30}$$

Squaring and adding both sides of Eqs. (4.29) & (4.30) yields

$$\omega^4 + \left({M_1}^2 - 2N_1 - {M_2}^2\right)\omega^2 + \left({N_1}^2 - {N_2}^2\right) = 0 \tag{4.31}$$

Setting $\omega^2 = Z_1$, Eq. (4.31) becomes

$${Z_1}^2 + MZ_1 + T = 0 \tag{4.32}$$

Here, $M = \left({M_1}^2 - 2N_1 - {M_2}^2\right)$ and $T = \left({N_1}^2 - {N_2}^2\right)$.

Clearly, if $M = \left({M_1}^2 - 2N_1 - {M_2}^2\right) > 0$ and $T = \left({N_1}^2 - {N_2}^2\right) > 0$ are satisfied simultaneously then by Routh-Hurwitz Criterion Eq. (4.32) will always have roots with the negative real part. It contradicts our assumption for instability that $\lambda = i\omega$ is a root of Eq. (4.27). Hence, Q^* is locally asymptotically stable for $\tau > 0$.

4.6.5 Hopf bifurcation analysis

In this section, we discuss the Hopf bifurcation of the system (4.4).

If $T = \left({N_1}^2 - {N_2}^2\right)$ in Eq. (4.32) is negative then there is unique positive ω_0 satisfying Eq. (4.32) i. e. there is a single pair of purely imaginary roots $\pm i\omega_0$ to Eq. (4.32).
From Eqs. (4.39) & (4.30) τ_n corresponding to ω_0 can be obtained as

$$\tau_n = \frac{1}{\omega_0}\, arccos\left(\frac{(N_2 - M_1M_2){\omega_0}^2 - N_1N_2}{{M_2}^2{\omega_0}^2 + {N_2}^2}\right) + \frac{2n\pi}{\omega_0},\ n = 0, 1, 2, ... \tag{4.33}$$

Endemic equilibrium Q^* is stable for $\tau < \tau_0$ if transversality condition holds true *i.e.* if $\frac{d}{d\tau}(Re\ (\lambda))\Big|_{\lambda = i\omega_0} > 0$.

Differentiating Eq. (4.27) with respect to τ, we get

$$\left(2\lambda + M_1 + M_2e^{-\lambda\tau} - (M_2\lambda + N_2)\tau e^{-\lambda\tau}\right)\frac{d\lambda}{d\tau} = \lambda(M_2\lambda + N_2)e^{-\lambda\tau} \tag{4.34}$$

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{\left(2\lambda + M_1 + M_2e^{-\lambda\tau} - (M_2\lambda + N_2)\tau e^{-\lambda\tau}\right)}{\lambda(M_2\lambda + N_2)e^{-\lambda\tau}} = \frac{(2\lambda + M_1)}{\lambda(M_2\lambda + N_2)e^{-\lambda\tau}} + \frac{M_2}{\lambda(M_2\lambda + N_2)} - \frac{\tau}{\lambda}$$

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{(2\lambda + M_1)}{-\lambda(\lambda^2 + M_1\lambda + N_1)} + \frac{M_2}{\lambda(M_2\lambda + N_2)} - \frac{\tau}{\lambda}$$

$$\frac{d}{d\tau}\left(Re\ (\lambda)\right)^{-1}\Big|_{\lambda = i\omega_0} = Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\Big|_{\lambda = i\omega_0}$$

$$= Re\left(\frac{(2i\omega_0 + M_1)}{-i\omega_0(-{\omega_0}^2 + iM_1\omega_0 + N_1)} + \frac{M_2}{i\omega_0(iM_2\omega_0 + N_2)} - \frac{\tau}{i\omega_0}\right)$$

$$= Re\left(\frac{1}{\omega_0}\left(\frac{(2i\omega_0 + M_1)}{({\omega_0}^2 - N_1)i + M_1\omega_0)} + \frac{M_2}{(-M_2\omega_0 + iN_2)} + i\tau\right)\right)$$

$$= \frac{1}{\omega_0}\left(\frac{2\omega_0({\omega_0}^2 - N_1) + {M_1}^2\omega_0}{({\omega_0}^2 - N_1)^2 + (M_1\omega_0)^2} - \frac{{M_2}^2\omega_0}{(M_2\omega_0)^2 + {N_2}^2}\right)$$

$$= \frac{2{\omega_0}^2 + ({M_1}^2 - 2N_1 - {M_2}^2)}{(M_2\omega_0)^2 + {N_2}^2}$$ (Since, from Eqs. (4.29) & (4.30), $({\omega_0}^2 - N_1)^2 + (M_1\omega_0)^2 = (M_2\omega_0)^2 + {N_2}^2$)

Under the condition ${M_1}^2 - 2N_1 - {M_2}^2 \neq 0$, we have $\frac{d}{d\tau}(Re\ (\lambda))\Big|_{\lambda = i\omega_0} > 0$.

Thus, the transversality condition holds and Hopf bifurcation occurs at $\omega = \omega_0, \tau = \tau_0$.

By summarizing the above analysis, we arrive at the following Theorem.

Theorem 4.12: The endemic equilibrium (EE) of the system (4.4) is locally asymptotically stable for $\tau \in [0, \tau_0)$ and it exhibits Hopf bifurcation at $\tau = \tau_0$.

4.6.6 Global stability analysis

We suppose that,

$$H(S(t)) = \beta S(t), F\big(I(t)\big) = \frac{I(t)}{1 + \alpha I(t)}.$$

To prove our results, we need the following assumptions:

A1. $H(0) = F(0) = 0$; $H'(S) > 0$, for all $S, I > 0$.

A2. $F'(I) > 0$; $\frac{\partial^2 F(I)}{\partial I^2} \leq 0$, for all $S, I > 0$.

A3. $\frac{F(I)}{F(I^*)} \leq 1$; $\frac{(\mu+d+\gamma)I+\frac{aI^2}{1+bI^2}}{H(S^*)F(I)} \geq 1$ or $\frac{F(I)}{F(I^*)} \geq 1$; $\frac{(\mu+d+\gamma)I+\frac{aI^2}{1+bI^2}}{H(S^*)F(I)} \leq 1$ for all $S, I > 0$.

Theorem 4.13: Suppose that assumptions (A1-A3) are satisfied.

i. If $R_0 > 1$, the endemic equilibrium $Q^*(S^*, I^*)$ is globally asymptotically stable for any $\tau \geq 0$.

ii. If $R_0 \leq 1$, the disease-free equilibrium $Q(S_0, 0) = Q\left(\frac{A}{\mu}, 0\right)$ is globally asymptotically stable for any $\tau \geq 0$.

Proof:

i) Let us consider the solution $(S(t), I(t))$ of the system (4.4) with the initial conditions. For any $\tau \geq 0$, we define the function $U_1(t)$ as follows:

$$U_1(t) = S(t) - \int_{S^*}^{S(t)} \frac{H(S^*)}{H(\eta)} d\eta + I(t) - \int_{I^*}^{I(t)} \frac{F(I^*)}{F(\eta)} d\eta.$$

Korobeinikov and Maini [2004] showed that Q^* is the only internal stationary point and the minimum point of $U_1(t) \to \infty$ at the boundary of the positive quadrant. Therefore, Q^* is the global minimum point, and the function is bounded from below.

Let

$$U_2 = \int_0^\tau \left[\frac{F(I(t-\xi))}{F(I^*)} - 1 - \ln\frac{F(I(t-\xi))}{F(I^*)}\right] d\xi,$$

It is easy to see that $U_2 > 0$ and $U_2 = 0$ if and only if $I(t-\xi) = I^*$ for all $\xi \in [0,\tau]$. For any positive $I(t-\xi)$ for ξ in $[0,\tau]$, U_2 will be finite and can be differentiated. Therefore, the derivative of U_2 is

$$\frac{dU_2}{dt} = \frac{d}{dt}\int_0^\tau \left[\frac{F(I(t-\xi))}{F(I^*)} - 1 - \ln\frac{F(I(t-\xi))}{F(I^*)}\right] d\xi$$

$$= \int_0^\tau \frac{d}{dt}\left[\frac{F(I(t-\xi))}{F(I^*)} - 1 - \ln\frac{F(I(t-\xi))}{F(I^*)}\right] d\xi$$

$$= -\int_0^\tau \frac{d}{d\xi}\left[\frac{F(I(t-\xi))}{F(I^*)} - 1 - \ln\frac{F(I(t-\xi))}{F(I^*)}\right] d\xi$$

$$= -\left[\frac{F(I(t-\xi))}{F(I^*)} - 1 - \ln\frac{F(I(t-\xi))}{F(I^*)}\right]_{\xi=0}^{\tau}$$

$$= -\frac{F(I(t-\tau))}{F(I^*)} + \frac{F(I(t))}{F(I^*)} + \ln\frac{F(I(t-\tau))}{F(I)}.$$

Now we study the behavior of Lyapunov functional

$$V_1 = U_1(t) + (\mu + d + \gamma + \tfrac{aI^*}{1+bI^{*2}})I^*U_2$$

The derivative of V_1 along the solution of (4.4) is given by

$$\frac{dV_1}{dt} = \left(1 - \frac{H(S^*)}{H(S)}\right)\dot{S}(t) + \left(1 - \frac{F(I^*)}{F(I)}\right)\dot{I}(t) + \left(\mu + d + \gamma + \frac{aI^*}{1+bI^{*2}}\right)I^*\frac{dU_2}{dt}$$

$$= \left(1 - \frac{H(S^*)}{H(S)}\right)(\mu S^* + H(S^*)F(I^*) - \mu S - H(S)F(I(t-\tau)))$$

$$+ \left(1 - \frac{F(I^*)}{F(I)}\right)\left(H(S)F\big(I(t-\tau)\big) - (\mu + d + \gamma)I - \frac{aI^2}{1+bI^2}\right)$$

$$- \left(\mu + d + \gamma + \frac{aI^*}{1+bI^{*2}}\right)I^*\left(\frac{F\big(I(t-\tau)\big)}{F(I^*)} - \frac{F(I)}{F(I^*)}\right.$$

$$\left. - \ln\frac{F\big(I(t-\tau)\big)}{F(I)}\right).$$

By noting that

$$\ln\frac{F(I(t-\tau))}{F(I^*)} = \ln\frac{H(S^*)}{H(S)} + \ln\frac{H(S)F(I(t-\tau))}{H(S^*)F(I)},$$

and

$$\left(\mu + d + \gamma + \frac{aI^*}{1+bI^{*2}}\right) I^* = H(S^*)F(I^*),$$

It is easy to see that

$$\frac{dV_1}{dt} = \frac{\mu}{H(S)}(H(S) - H(S^*))(S^* - S) + H(S^*)F(I^*)\left(1 - \frac{H(S^*)}{H(S)} + \ln\frac{H(S^*)}{H(S)}\right) +$$

$$H(S^*)F(I^*)\left(1 + ln\frac{H(S)F(I(t-\tau))}{H(S^*)F(I)} - \frac{H(S)F(I(t-\tau))}{H(S^*)F(I)}\right) + H(S^*)F(I^*)\left(\left(\frac{F(I)}{F(I^*)} -\right.\right.$$

$$\left.\left.1\right)\left(1 - \frac{(\mu+d+\gamma)I+\frac{aI^2}{1+bI^2}}{H(S^*)F(I)}\right)\right).$$

Here,

$1 - \frac{H(S^*)}{H(S)} + \ln\frac{H(S^*)}{H(S)} \leq 0$; for all $S > 0$, and $1 + \ln\frac{H(S)F(I(t-\tau))}{H(S^*)F(I)} - \frac{H(S)F(I(t-\tau))}{H(S^*)F(I)} \leq 0$; for all $I(t-\tau) > 0, S > 0$. (4.35)

For monotonically increasing function $H(S), H(S) \geq H(S^*)$ holds when $S \geq S^*$ and hence the following inequalities holds:

$$(S^* - S)(H(S) - H(S^*)) \leq 0. \quad (4.36)$$

Hence, by condition (A3) and inequalities (4.35)-(4.36), all the conditions of corollary 5.2 of [Kuang (1993)] hold true. Hence, Q^* is globally asymptotically stable for any $\tau \geq 0$ when $R_0 > 1$.

ii) We consider the Lyapunov functional

$$V_2 = S(t) - \int_{S_0}^{S(t)} \frac{H(S_0)}{H(\eta)} d\eta + I(t) + H(S_0)\int_0^\tau F\big(I(t-\xi)\big)d\xi. \quad (4.37)$$

Let

$$U_3 = \int_0^\tau F\big(I(t-\xi)\big)d\xi.$$

The derivative of U_3 is

$$\frac{dU_3}{dt} = \int_0^\tau \frac{d}{dt}F\big(I(t-\xi)\big)d\xi = -\int_0^\tau \frac{d}{d\xi}F\big(I(t-\xi)\big)d\xi = -F\big(I(t-\tau)\big) + F\big(I(t)\big).$$

Hence, we obtain

$$\frac{dV_2}{dt} = \left(1 - \frac{H(S_0)}{H(S)}\right)\dot{S}(t) + \dot{I}(t) + H(S_0)\frac{dU_3}{dt}$$

$$= \left(1 - \frac{H(S_0)}{H(S)}\right)(\mu S_0 - \mu S - H(S)F(I(t-\tau))) + \left(H(S)F\big(I(t-\tau)\big) - (\mu + d + \gamma)I - \frac{aI^2}{1+bI^2}\right) - H(S_0)F\big(I(t-\tau)\big) + H(S_0)F\big(I(t)\big)$$

$$= -\frac{\mu}{H(S)}(S - S_0)\big(H(S) - H(S_0)\big) - \frac{aI^2}{1+bI^2} + (\mu + d + \gamma)\left(\frac{H(S_0)F\big(I(t)\big)}{(\mu+d+\gamma)} - I\right).$$

Here,

$$(S - S_0)\big(H(S) - H(S_0)\big) \geq 0 \tag{4.38}$$

and the condition (A2) ensure that $F(I) \leq \frac{\partial F(0)}{\partial I} I$ for all $I > 0$. Hence,

$$\frac{H(S_0)F\big(I(t)\big)}{(\mu+d+\gamma)} - I \leq \left(\frac{H(S_0)\frac{\partial F(0)}{\partial I}}{(\mu+d+\gamma)} - 1\right) I = (R_0 - 1)I. \tag{4.39}$$

Therefore, $R_0 < 1$ ensures that $\frac{dV_2}{dt} \leq 0$ for all $S(t), I(t) \geq 0$. Hence, again from Corollary 5.2 of [Kuang (1993)], we have that Q is stable. Furthermore, for $R_0 = 1, \frac{dV_2}{dt} = 0$ implies that $S(t) = S_0$. Hence, it can be shown that $Q(S_0, 0)$ is the largest invariant set in $\left\{\big(S(t), I(t)\big) \,\middle|\, \dot{V}_2 = 0\right\}$. With the help of the classical Lyapunov-LaSalle invariance principle [Hale and Lunel (1993); Sastry (1999)], Q is globally stable.

This completes the proof of theorem 4.13.

4.6.7 Undelayed system

In this subsection, we consider the case of instantaneous transmission of primary infection. We perform a qualitative analysis of the system (4.4) without delay, *i.e.*, we set $\tau = 0$. This analysis has interest in itself and will also allow getting some information on the stability of coexistence equilibrium in the case with delay.

It is useful to investigate the stability properties of the system (4.4), without delay, near the criticality (that is at Q and $R_0 = 1$). To achieve this aim, we use the bifurcation

theory approach developed in [Buonomo and Cerasuolo (2015)], which is based on the center manifold theory [Sastry (1999)]. In particular, we are interested to assess that if there is a stable coexistence equilibrium bifurcation form Q, and Q changes from being stable to unstable. This behaviour is called forward bifurcation [Buonomo and Cerasuolo (2015)].

Now, for the undelayed system, we propose the following result:

Theorem 4.14: When $\tau = 0$, the system (4.4) exhibits a forward bifurcation at Q and $R_0 = 1$.

Proof: Clearly, from the expression of R_0 it can be seen that R_0 is directly related to β. Subsequently, we choose β as the bifurcation parameter. Moreover, $R_0 = 1$ implies that $\beta = \beta^* = \frac{\mu(\mu+d+\gamma)}{A}$. Since the linearization technique is not applicable to check the stability behavior at $R_0 = 1$, so we use center manifold theory [Sastry (1999)]. For this we redefine $S = x_1$ and $I = x_2$, then the system (4.4) takes the form

$$\frac{dx_1}{dt} = A - \mu x_1 - \frac{\beta x_1 x_2}{1+\alpha x_2} \equiv \mathrm{f}_1 ,$$
$$\frac{dx_2}{dt} = \frac{\beta x_1 x_2}{1+\alpha x_2} - (\mu + d + \gamma)x_2 - \frac{a{x_2}^2}{1+b{x_2}^2} \equiv \mathrm{f}_2. \quad (4.40)$$

The Jacobian matrix J' of the system (4.40) evaluated at $R_0 = 1$ and $\beta = \beta^*$ around the disease-free equilibrium is

$$J' = \begin{bmatrix} -\mu & -\frac{\beta^* A}{\mu} \\ 0 & 0 \end{bmatrix}$$

J' has a simple zero eigenvalue while the other eigenvalue is negative. The right eigenvector, $w = [w_1, w_2]^T$ of J' corresponding to zero eigenvalue can be obtained as under

$$w_1 = -\frac{\beta^* A}{\mu^2}, w_2 = 1$$

Similarly, the left eigenvector, $u = [u_1, u_2]$ of J' corresponding to zero eigenvalue is obtained as $[0, 1]$. The non-zero partial derivatives associated with the functions f_1 and f_2 evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$$\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_Q = \left(\frac{\partial^2 f_2}{\partial x_2 \partial x_1}\right)_Q = \beta^*, \left(\frac{\partial^2 f_2}{\partial {x_2}^2}\right)_Q = -\frac{2\alpha\beta^* A}{\mu} - 2a \text{ and } \left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_Q = \frac{A}{\mu}.$$

Using theorem 4.1 of [Chavez and Song (2004)], the coefficients $a_1$and b_1 can be computed as

$$a_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q$$

$$= u_2\left(2w_1 w_2 \beta^* - {w_2}^2\left(\frac{2\alpha\beta^* A}{\mu} + 2a\right)\right)$$

$$= -\left(2\frac{\beta^* A}{\mu^2}\beta^* + \frac{2\alpha\beta^* A}{\mu} + 2a\right) < 0$$

and

$$b_1 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q$$

$$= u_2\left(w_2\frac{A}{\mu}\right)$$

$$= \frac{A}{\mu} > 0.$$

From the expressions of a_1 and b_1, it is evident that $a_1 < 0$ and $b_1 > 0$. Therefore, from theorem 4.1 (iv) of [Chavez and Song (2004)] bifurcation is forward. This completes the proof.

Theorem 4.15: For $\tau = 0$, the system (4.4) at Q^* is locally asymptotically stable if $M_1 + M_2 > 0$ and $N_1 + N_2 > 0$ are satisfied simultaneously.

Proof: At endemic equilibrium Q^* the characteristic equation of the system (4.4) is obtaining by putting $\tau = 0$ in the Eq. (4.27) as given below:

$$\lambda^2 + M_1\lambda + N_1 + (M_2\lambda + N_2) = 0 \qquad (4.41)$$

Clearly, if $M_1 + M_2 > 0$ and $N_1 + N_2 > 0$ are satisfied simultaneously then by Routh-Hurwitz Criterion Eq. (4.41) will always have roots with the negative real part and hence, the system (4.4) at Q^* for $\tau = 0$ is locally asymptotically stable.

4.7 Numerical simulation

In this section, we will simulate the system (4.2) numerically. The set of values of parameters is given in Table 4.1.

Graphs have been plotted for S and I for various values of τ. The trajectory of S and I approach to steady state as shown in Fig. 4.2 and Fig. 4.3 for $\tau = 0$ and 1 respectively. In Figs. 4.2 & 4.3, the number of the infected individuals initially increases and as time passes, they approach to the steady state which may be due to the treatment. These individuals once recovered have become immunized to the infection and will not get re-infected in the future. Furthermore, the number of susceptible individuals decreases to attain a steady state.

Fig. 4.4 shows the variation in the infected population for the various values of τ. It can be seen that the infected population is less at $\tau = 0$ than the infected population at $\tau = 1,2$ and 3 respectively. It can be depicted that delay in showing the symptoms of the disease will cause the increment in the infected population.

Figs. 4.5 & 4.6 demonstrate the effect of treatment/ cure rate (a) and limitation rate (b) in treatment availability on the infected population with various values of a and b. Fig. 4.5 shows the decline in infected population as treatment rate (a) increases and it settles down at its steady state, but the disease is not getting totally eliminated rather it will persist at a much lower level. Fig. 4.6 shows the increment in the infected population as b increases, which is due to the limited availability of resources in the society.

Figs. 4.7 & 4.8 show the variation in the infected population with and without treatment rate Holling type II treatment rate at $\tau = 0$ and 1 respectively. It can be observed that the infected population will decrease drastically if Holling type II treatment is given at the appropriate time.

Figs. 4.9 & 4.10 show the infected population at various values of β and α respectively. Clearly, it can be seen that the infected population decreases in both situations, when the transmission rate (β) is decreasing and the measures of inhibition (α) is increasing respectively.

Fig. 4.11 shows the variation in the infected population when treatment to infectives is given according to Holling type II and Holling type III treatment rates respectively.

4.8 Conclusions

In this chapter, we proposed a time-delayed SIR model with Holling functional type II incidence rate and two different treatment rates (Holling functional type II & III). The model analysis showed that the model has two equilibria, namely; disease-free and endemic. The stability analysis of the model equilibria discussed separately for both combinations of incidence and treatment rates (*i.e.* incidence rate as Holling functional type II and treatment rate as Holling functional type II & III). The local stability of disease-free equilibrium (DFE) Q has been explained in terms of the basic reproduction number R_0 for both combinations separately. Further, we have shown that the DFE at $R_0 = 1$ is linearly neutrally stable for time delay $\tau > 0$ which reveals that disease may persist at a very low level in society; and exhibits either a forward bifurcation or backward or possibly saddle-node bifurcation for the time delay $\tau = 0$ under certain conditions. Furthermore, we showed that the endemic equilibrium (EE) Q^* of the system (4.2) is locally asymptotically stable for both the combinations at $\tau = 0$ if the conditions stated in theorems 4.6 and 4.15 are satisfied respectively. Furthermore, conditions for the existence of Hopf bifurcation were discussed. Moreover, for the combination of Holling type II incidence rate and Holling type II treatment rate, we showed that both DFE and EE are globally asymptotically stable when $R_0 \leq 1$ and $R_0 > 1$ for time lag $\tau \geq 0$ respectively. Numerical simulations demonstrate that there will be marginal decrement in the lessening in the infected population in the two circumstances; when the transmission rate (β) decreases and measures of inhibition (α) increases (Figs. 4.9 & 4.10). It very well may be reasoned that the infected population increases with the increment in a delay in the incidence rate (Fig. 4.4) and the infected population diminishes when treatment of infectives is given according to Holling type treatment rates at the proper time (Figs. 4.8 & 4.11).

Table 4.1: Description and numerical values of parameters for simulation

Parameter	**Value**
Recruitment rate $(\boldsymbol{A})$	6
Measures of inhibition (α)	0.05
Effective contact rate or Transmission rate (β)	0.007
Natural mortality rate (μ)	0.05
Disease induced mortality rate (d)	0.005
Recovery rate (γ)	0.003
Treatment or Cure rate (a)	0.02
Limitation rate in treatment availability (b)	0.02

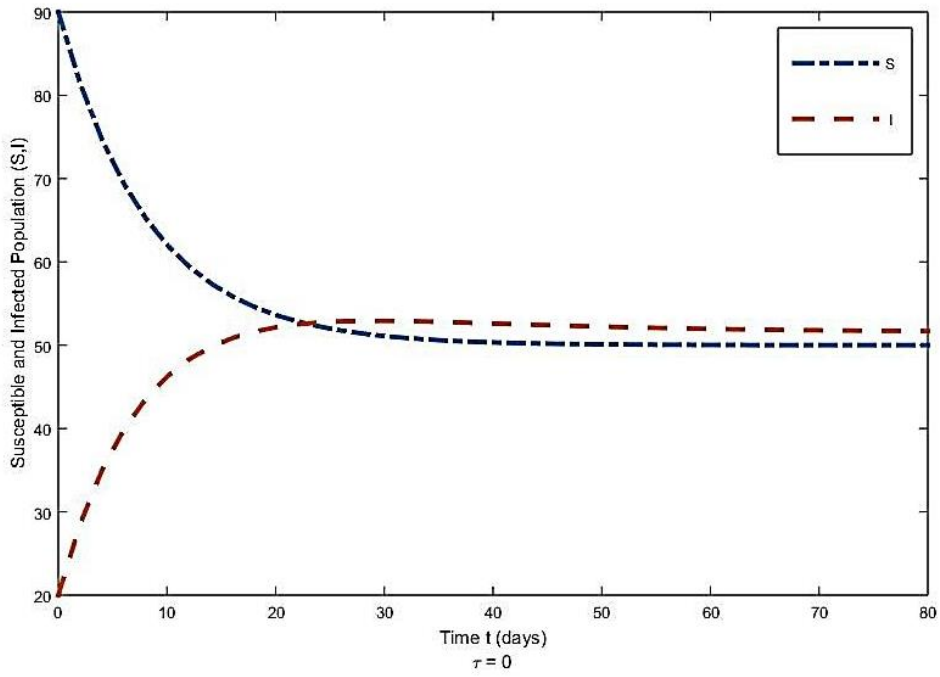


Fig. 4.2: Susceptible (S) and infected (I) population versus time at time lag $\tau = 0$.

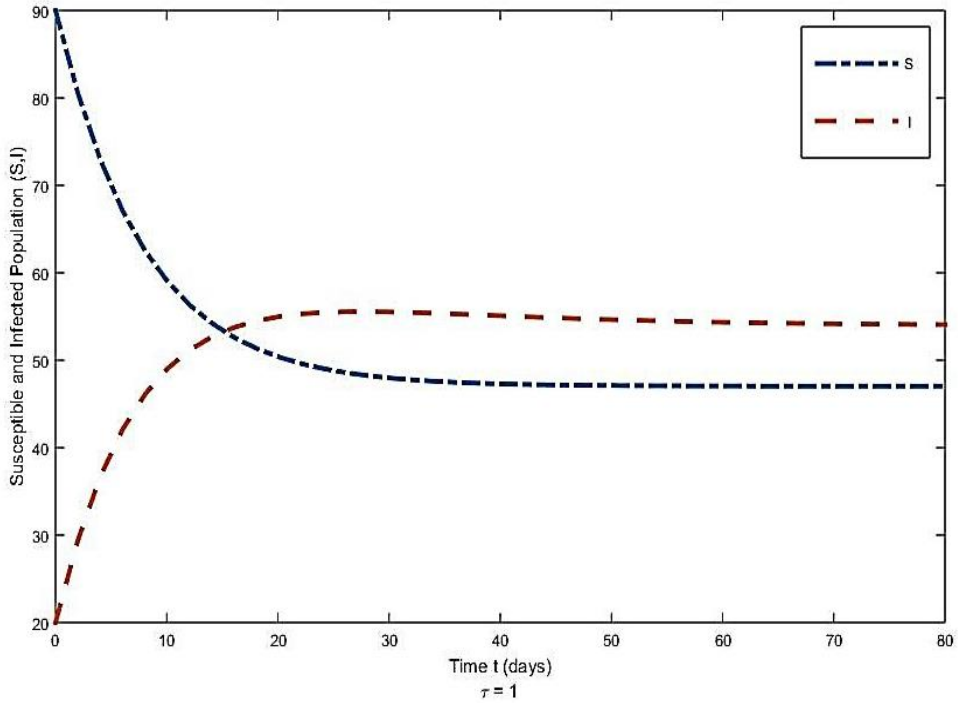


Fig. 4.3: Susceptible (S) and infected (I) population versus time at time lag $\tau = 1$.

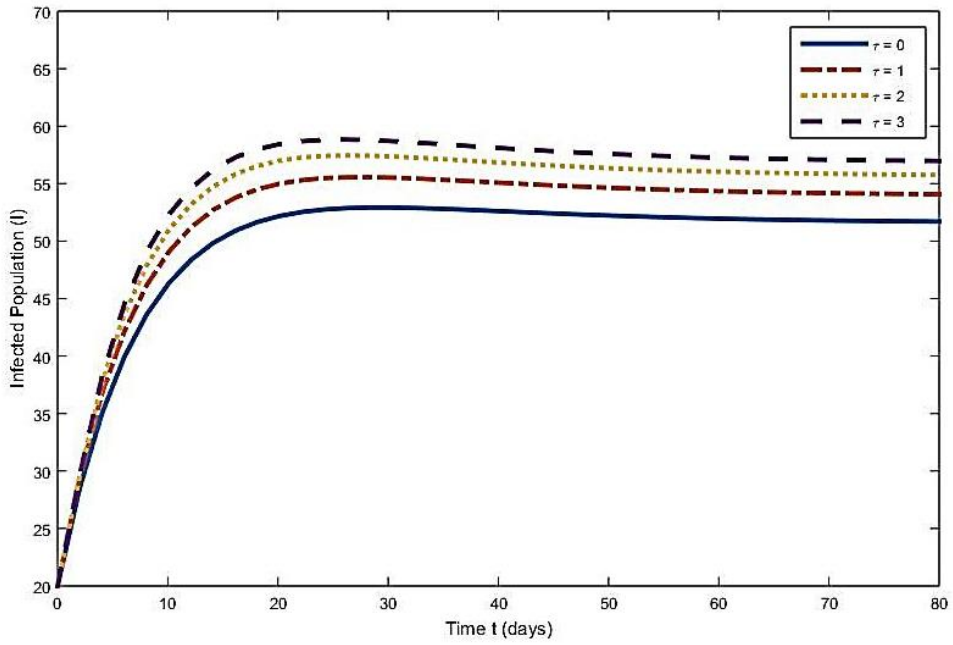


Fig. 4.4: Infected population (I) at various values of time lag τ.

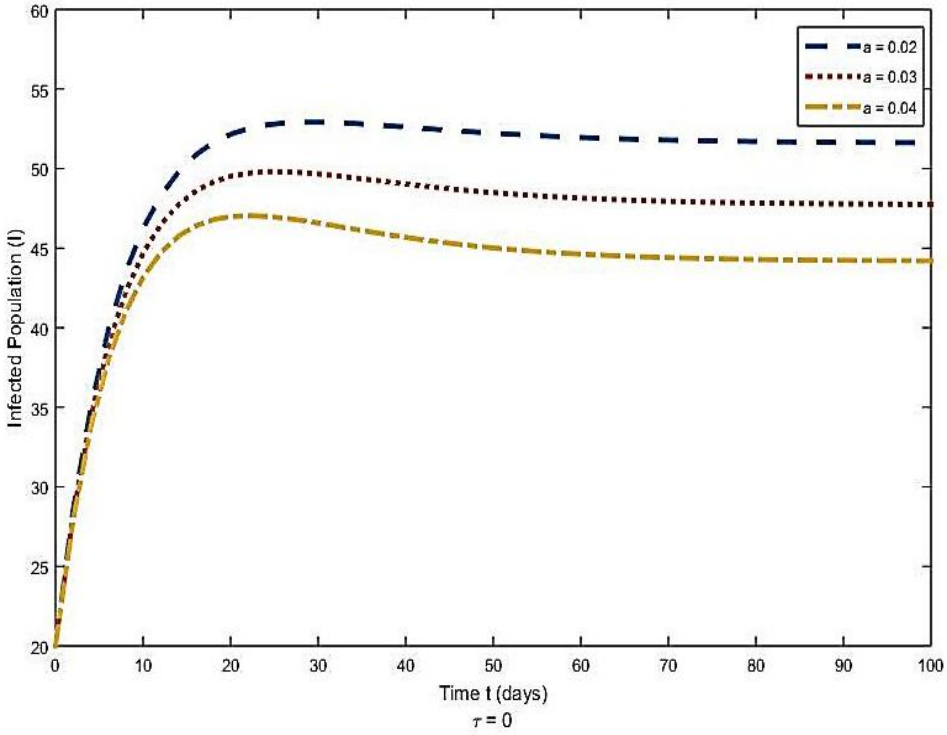


Fig. 4.5: Infected population (I) at various values of cure rate (a) for $\tau = 0$.

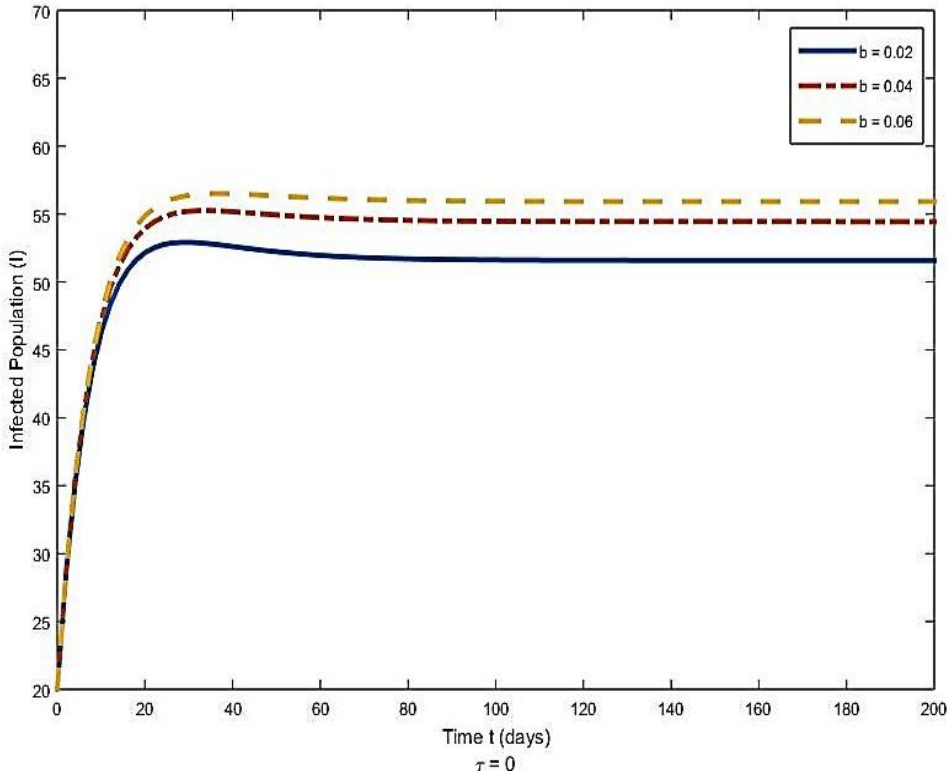


Fig. 4.6: Infected population (I) at various values of limitation rate (b) in treatment availability for $\tau = 0$.

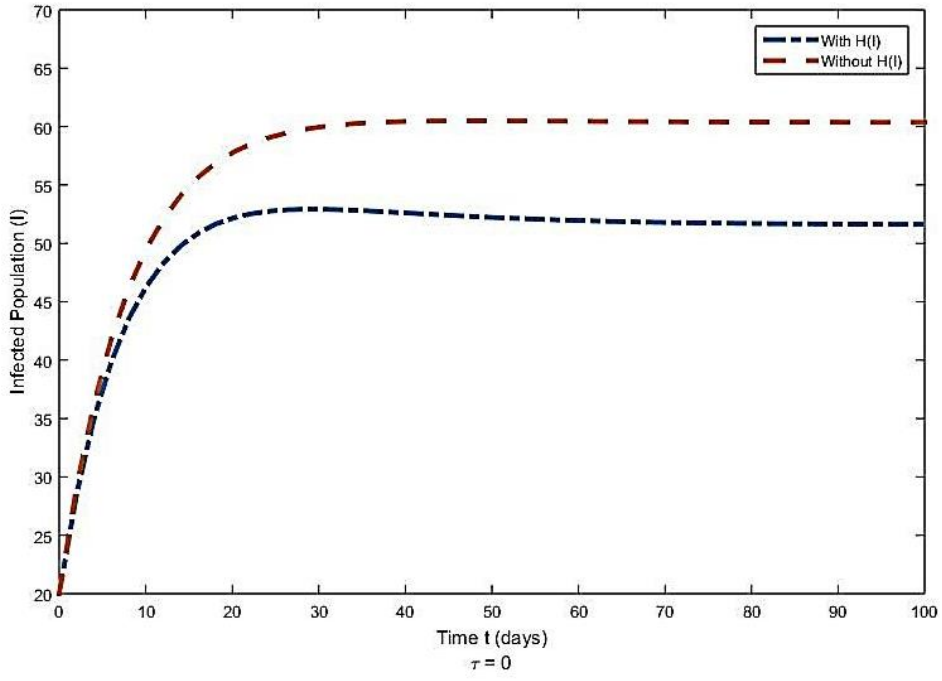


Fig. 4.7: Infected population (I) with and without Holling type II treatment rate at time lag$\tau = 0$.

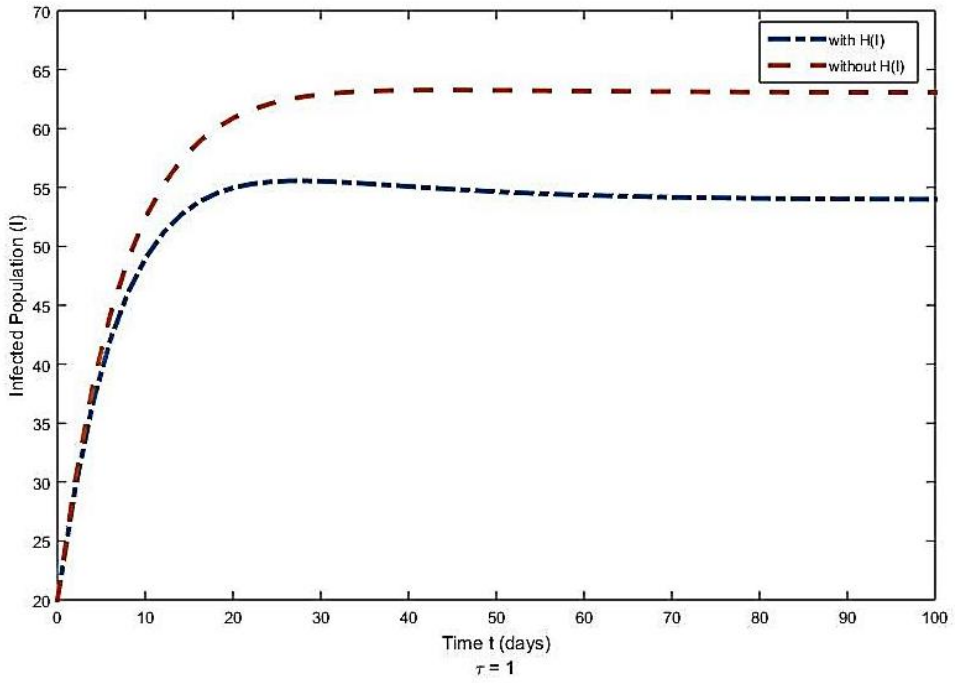


Fig.4.8: Infected population (I) with and without Holling type II treatment rate at time lag $\tau = 1$.

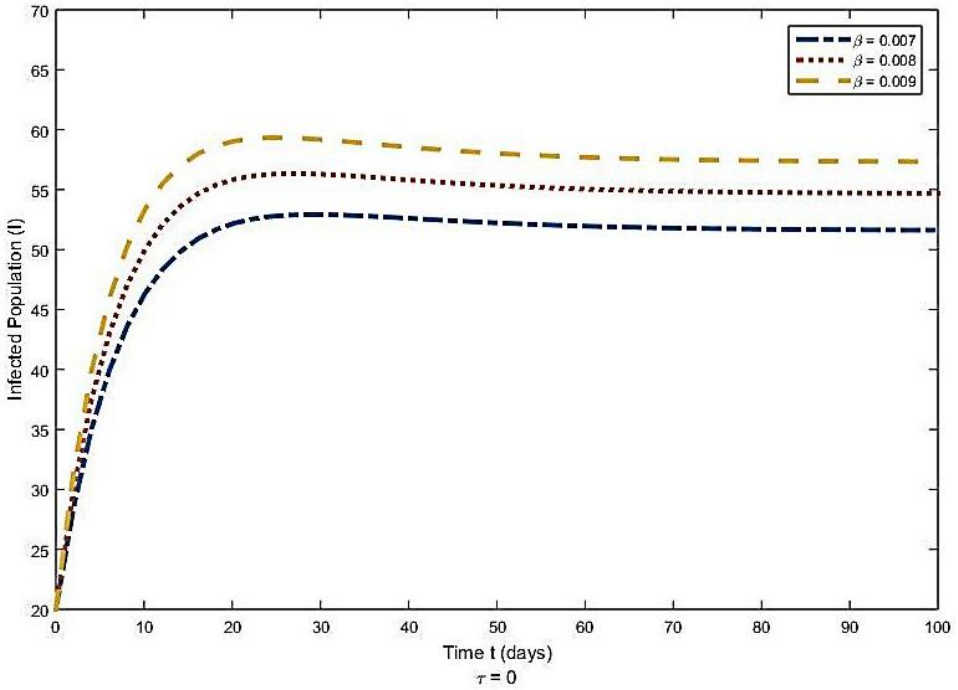


Fig. 4.9: Infected population (I) at various values of the transmission rate (β) at $\tau = 0$.

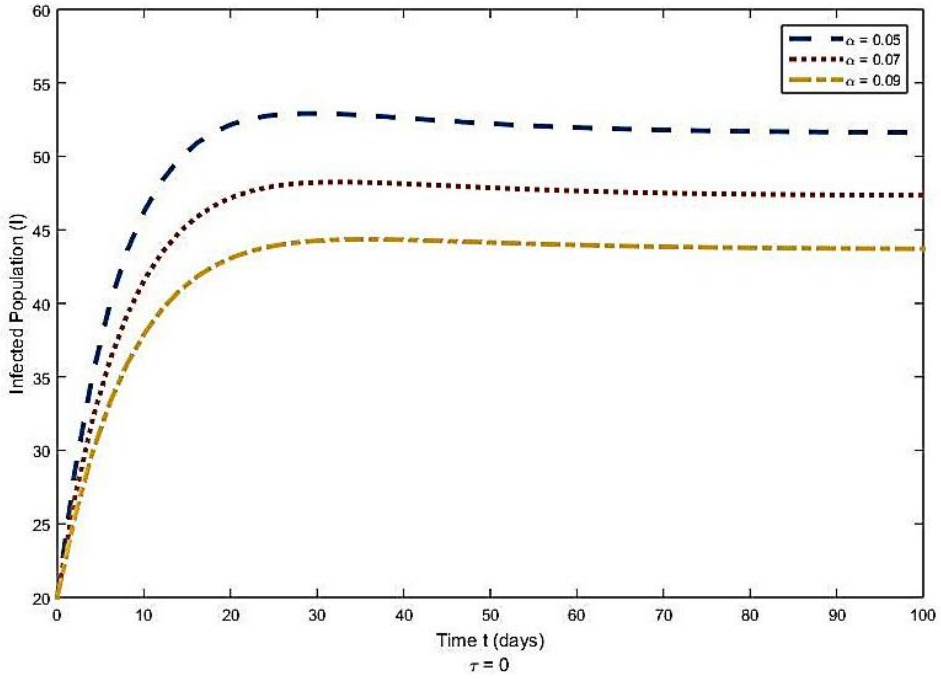


Fig. 4.10: Infected population (I) at various values of measures of inhibition (α) at $\tau = 0$.

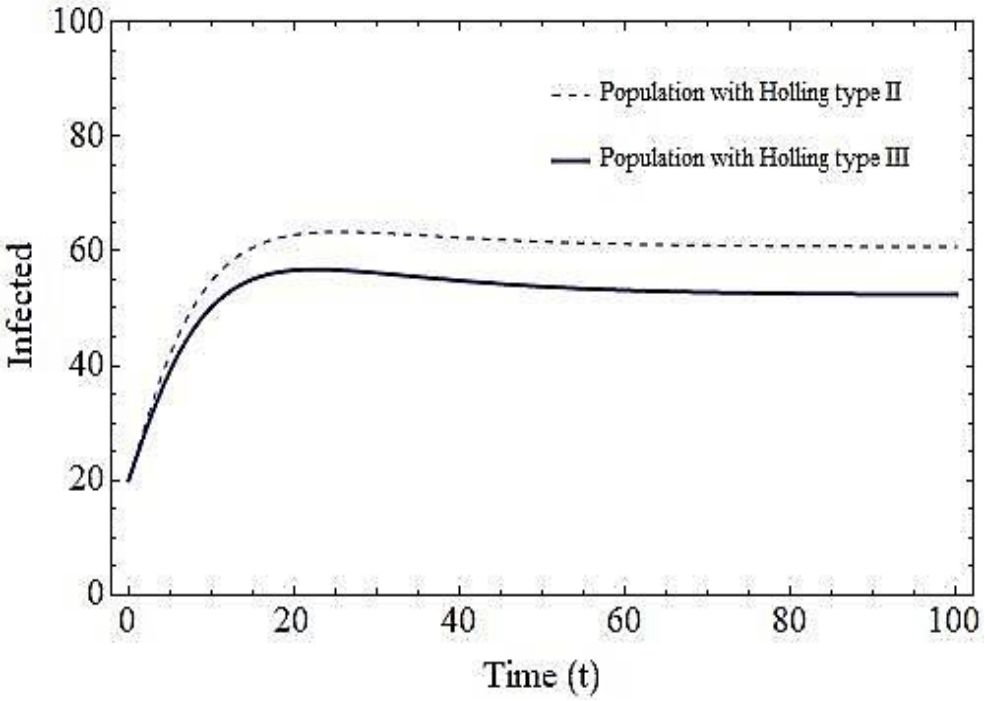


Fig. 4.11: Variation in the infected population (I) with Holling type II & III treatment rates.

CHAPTER 5

DYNAMICAL STUDY OF A SIR EPIDEMIC MODEL ALONG WITH TIME DELAY; HOLLING FUNCTIONAL TYPE II INCIDENCE RATE AND MONOD-HALDANE FUNCTIONAL TYPE TREATMENT RATE

In this chapter, a time-delayed susceptible-infected-recovered epidemic model is being proposed to capture the role of latency period mathematically along with Holling functional type incidence rate and Monod-Haldane (M-H) functional type treatment rate for the diseases like SARS, MERS, *etc*. The stability of model equilibria has been established in the three regions of the basic reproduction number R_0 *i. e.* R_0 equals to one, greater than one and less than one. The model is locally asymptotically stable for disease-free equilibrium when the basic reproduction number is less than one and unstable when the basic reproduction number is greater than one. We have investigated the stability of the disease-free equilibrium at R_0 equals to one using center manifold theory. We proved that at $R_0 = 1$, disease-free equilibrium changes its stability from stable to unstable. We also investigated the stability for endemic equilibrium. Further, numerical simulations have been carried out to strengthen the theoretical findings.

5.1 Introduction

In mathematical epidemiology literature, many authors [Gumel *et al.* (2006); Moghadas and Alexander (2006); Xu and Ma (2009a) & (2009b); Dubey *et al.* (2013); Hattaf *et al.* (2013); Sahani and Yashi (2016)] have suggested various mathematical models for the disease transmission such as susceptible-infected-recovered (SIR) model, susceptible-infected-recovered-susceptible (SIRS), susceptible-exposed-infected-recovered (SEIR) and many others. In this chapter, an attempt has been made to understand the disease transmission process by incorporating the incidence rate as Holling functional type II and treatment rate as Monod-Haldane (M-H) functional type for the diseases like SARS, MERS, *etc*. In the modulation of population dynamics, both transmission and treatment rates play an important role. The incidence of infection is the process in which susceptible becomes infected via infected population through various channels [Dubey *et al.* (2016)]. Several authors have suggested that the disease transmission process may have a nonlinear incidence rate [Moghadas and Alexander (2006); Xu and Ma (2009)]. The explanations of bilinear and Holling type II nonlinear incidence rates have already been given in chapter 2.

In the field of epidemiology, treatment, vaccination, and many more play an important role in controlling the disease spread. Recently, many researchers [Dubey *et al.* (2013), (2015) & (2016); Li and Liu (2014)] have focused on the nonlinear type treatment rates. Different type of treatment rates like Holling type II, Holling type III and many others have been implemented by the authors in their model to study the dynamics of infectious diseases. Andrews [1968] had suggested a functional having the form

$$G(I) = \frac{mI}{a + bI + I^2}$$

called the Monod-Haldane functional. Sokol and Howell [1981] had proposed a simplified Monod-Haldane (M-H) functional having the form

$$G(I) = \frac{mI}{a + I^2}$$

Baek *et al.* [2009] used this M-H functional to describe the dynamical relation between prey and predator. Considering these facts, we have incorporated treatment rate as

simplified M-H functional type in our delayed SIR model with Holling type II incidence rate.

In this chapter, we have analyzed the effect of time-delay on a SIR epidemic model along with Holling functional type II incidence rate and M-H functional type treatment rate for the better understanding of transmission dynamics of the diseases like SARS, MERS, *etc*. Furthermore, we evaluate the basic reproduction number R_0, analyzed the dynamical behavior of the model and also discussed the stability of the model equilibria. The stability analysis of equilibria has been done by Descartes's rule of signs [Wang (2004)] along with the Routh-Hurwitz criterion.

5.2 Mathematical model

We assume that the total population $N(t)$ is divided into three compartments: susceptible individuals compartment $S(t)$, infected individuals compartment $I(t)$ and recovered individuals compartment $R(t)$. We considered that the treatment of infectives is given according to the simplified Monod-Haldane type treatment rate $(h(I) = aI/(I^2 + b))$ for the recovery. The progression of an epidemic in different compartments has been shown by block diagram as given in Fig. 5.1.

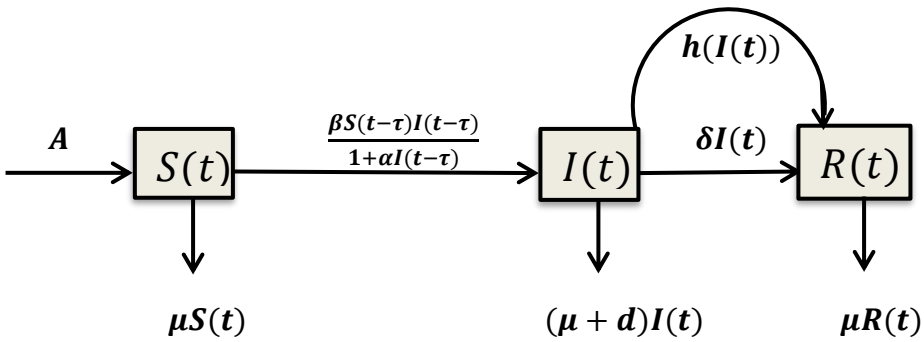


Fig. 5.1: Transfer diagram of the infection through various compartments.

The rate of change of the population in each compartment is given by the following nonlinear system of the delay differential equations:

$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)}$,

$$\frac{dI(t)}{dt} = \frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)} - (\mu + d + \delta)I(t) - \frac{aI(t)}{I^2(t)+b}, \quad (5.1)$$

$$\frac{dR(t)}{dt} = \frac{aI(t)}{I^2(t)+b} + \delta I(t) - \mu R(t).$$

where $\tau > 0$ is a fixed time during which the infectious agents develop in the vector and it is only after this time that the infected vector can infect a susceptible individual.

Let $C = C([-\tau, 0], \mathbb{R}^3)$ be the Banach space of continuous functions mapping the interval $[-\tau, 0]$ to $\mathbb{R}^3$ with the topology of uniform convergence. By the fundamental theory of functional differential equations [Hattaf *et al.* (2013)], it can be shown that there exists a unique solution $(S(t),\ I(t),\ R(t))$ of the model (5.1) with initial data $(S_0, I_0, R_0) \in C$. For ecological reasons, we assume that the initial conditions of the model (5.1) satisfy:

$$S_0(\varphi) \geq 0, I_0(\varphi) \geq 0, R_0(\varphi) \geq 0,\ \varphi \in [-\tau, 0]. \quad (5.2)$$

The term $h(I(t)) = \frac{aI(t)}{b + I^2(t)}$ in the model (5.1) represents the Monod-Haldane (M-H) type treatment rate, where a is the cure/treatment rate and b is the rate of limitation in treatment availability. The detailed explanation of the M-H treatment rate has already been given in section (2.1). The term $\frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)}$ in the model (5.1) represent the Holling functional type II incidence rate, and, here, τ is taken in both susceptible (S) and infected (I) populations to capture the role of latency period as time delay (see section (3.1) for detailed explanation).

5.3 Basic properties of the model

From the model (5.1) we can infer that S and I are free from the effect of R. Thus it is enough to consider the following reduced system for mathematical analysis:

$$\frac{dS}{dt} = A - \mu S - \frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)},$$

$$\frac{dI}{dt} = \frac{\beta S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)} - (\mu + d + \delta)I - \frac{aI}{I^2+b}. \quad (5.3)$$

with initial conditions

$$S_0(\varphi) \geq 0, I_0(\varphi) \geq 0,\ \varphi \in [-\tau, 0]. \quad (5.4)$$

The system (5.3) monitors the population. It is assumed that the parameters $A, \mu, \beta, d, \delta, a, \alpha, b > 0$. The description of parameters is given in Table 5.1. From Hattaf *et al.* [2013] it follows that all dependent variables of the system (5.3) are nonnegative i.e. $(S, I) \in \mathbb{R}^2_+$.

Theorem 5.1: The system (5.3) has a nonnegative solution with the initial value (5.4).

Proof: The proof of this theorem is similar to theorem (4.2) as in section (4.3). Hence, it is omitted here.

Theorem 5.2: All solutions of the system (5.3) starting in $\mathbb{R}^2_+$ are bounded and enter in the set $D = \left\{(S, I) \in S(t) + I(t) \leq \frac{A}{\mu}\right\}$.

Proof: The proof of this theorem is similar to theorem (4.1) as in section (4.3). Hence, it is omitted here.

5.4 Equilibria and their stability analysis

In this section, we find the equilibrium points and discuss their stability. Equilibria of the system (5.3) are obtained by setting the right-hand sides of the equations of the system to zero as given below:

i. Disease-free equilibrium $Q(\frac{A}{\mu}, 0)$ (DFE),

ii. Endemic equilibrium $Q^*(S^*, I^*)$ (EE).

5.4.1 Computation of the basic reproduction number (R_0)

The characteristic equation at DFE (Q) of the system (5.3) is given by

$$(\mu + \lambda)\left(\frac{\beta A}{\mu}\, e^{-\lambda\tau} - \mu - d - \delta - \frac{a}{b} - \lambda\right) = 0 \qquad (5.5)$$

One of the roots of Eq. (5.5) is given by $\lambda_1 = -\mu$ and other can be obtained from

$$\left(\frac{\beta A}{\mu}\, e^{-\lambda\tau} - \mu - d - \delta - \frac{a}{b} - \lambda\right) = 0$$

where,

The term $\frac{\beta A}{\mu\left(\mu+d+\delta+\frac{a}{b}\right)} e^{-\lambda\tau}$ at $\tau = 0$, is known as the basic reproduction number, denoted by R_0. Therefore, we define the basic reproduction number R_0 of our model by

$$R_0 = \frac{\beta A}{\mu\left(\mu + d + \delta + \frac{a}{b}\right)}.$$

5.4.1.1 Analysis for $R_0 \neq 1$

It can be observed that Eq. (5.5) always has one negative root $\lambda_1 = -\mu$ and other roots are the solutions of the equation

$$\lambda + \mu + d + \delta + \frac{a}{b} - \frac{\beta A}{\mu}\, e^{-\lambda\tau} = 0$$

Let

$$f(\lambda) = \lambda + \mu + d + \delta + \frac{a}{b} - \frac{\beta A}{\mu}\, e^{-\lambda\tau}$$

If $R_0 > 1$, for real λ ,

$$f(0) = \mu + d + \delta + \frac{a}{b} - \frac{\beta A}{\mu} = \left(\mu + d + \delta + \frac{a}{b}\right)(1 - R_0) < 0, \underset{\lambda \to +\infty}{Lim} f(\lambda) \to +\infty$$

Hence, there exists a positive real root of $f(\lambda) = 0$ if $R_0 > 1$.

If $R_0 < 1$, we assume that $Re\ \lambda \geq 0$.

We notice that

$$Re\ \lambda = \frac{\beta A}{\mu} e^{-Re\,\lambda\,\tau}\, cos\ Im\ \lambda\ \tau - \left(\mu + d + \delta + \frac{a}{b}\right) \leq \frac{\beta A}{\mu} - \left(\mu + d + \delta + \frac{a}{b}\right)$$
$$= \left(\mu + d + \delta + \frac{a}{b}\right)(\, R_0 - 1) < 0.$$

a contradiction to our assumption. Hence, if $R_0 < 1$ then Eq. (5.5) has a root λ with a negative real part.

Hence, we state the following theorem:

Theorem 5.3: DFE $Q(\frac{A}{\mu}, 0)$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

5.4.1.2 Analysis at $R_0 = 1$

We notice that the system (5.3), when evaluated at $R_0 = 1$, so that $\beta = \beta^* = \frac{\mu\left(\mu+d+\delta+\frac{a}{b}\right)}{A}$, has a zero eigenvalue and another eigenvalue that is negative. The stability behavior of the equilibrium point at $R_0 = 1$ cannot be determined using linearization so we use center manifold theory [Sastry (1999)]. For this, we redefined $S = x_1$ and $I = x_2$ then the system (5.3) can be rewritten as

$$\frac{dx_1}{dt} = A - \mu x_1 - \frac{\beta x_1(t-\tau)x_2(t-\tau)}{1+\alpha x_2(t-\tau)} \equiv f_1,$$

$$\frac{dx_2}{dt} = \frac{\beta x_1(t-\tau)x_2(t-\tau)}{1+\alpha x_2(t-\tau)} - (\mu + d + \delta)x_2 - \frac{a x_2}{b+{x_2}^2} \equiv f_2. \quad (5.6)$$

Let J^* be the Jacobian matrix at $R_0 = 1$ and bifurcation parameter $\beta = \beta^*$. Then

$$J^* = \begin{bmatrix} -\mu & -\frac{\beta^* A}{\mu} \\ 0 & 0 \end{bmatrix}$$

Let $u = [u_1, u_2\,]$ and $w = [w_1, w_2\,]^T$ denotes the left eigenvector and right eigenvector of J^* associated with null eigenvalue. Then, we get

$$u_1 = 0, u_2 = 1 \text{ and } w_1 = -\frac{\beta^* A}{\mu^2}, w_2 = 1.$$

The non-zero partial derivatives corresponding to the functions of the system (5.6) evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$$\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_Q = \beta^*, \left(\frac{\partial^2 f_2}{\partial x_2 \partial x_1}\right)_Q = \beta^*, \left(\frac{\partial^2 f_2}{\partial {x_2}^2}\right)_Q = \frac{-2\alpha\beta^* A}{\mu} \text{ and } \left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_Q = \frac{A}{\mu}$$

Then from [Chavez and Song (2004)], the bifurcation constants a_1 and b_1 are

$$a_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q$$

$$= u_2\left(2w_1 w_2 \beta^* + {w_2}^2\left(\frac{-2\alpha\beta^* A}{\mu}\right)\right)$$

$$= -\left(\frac{2\beta^* A(\beta^* + \mu\alpha)}{\mu^2}\right) < 0$$

and

$$b_1 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q = u_2\left(w_2 \frac{A}{\mu}\right)$$

$$= \frac{A}{\mu} > 0.$$

Thus, the following theorem is established:

Theorem 5.4: The behavior of DFE $Q\left(\frac{A}{\mu}, 0\right)$ changes from stable to unstable at $R_0 = 1$ and it implies the existence of a positive equilibrium as R_0 crosses one. Hence, the system (5.3) undergoes a forward transcritical bifurcation at $R_0 = 1$.

5.4.2 Existence and stability analysis of endemic equilibrium

To find the conditions for the existence of an equilibrium $Q^*(S^*, I^*)$ for which the disease is endemic in the population, the system (5.3) is rearranged to get S^*, and I^* which gives

$$S^* = \frac{(1 + \alpha I^*)\left((\mu + d + \delta)\left(b + I^{*2}\right) + a\right)}{\beta\left(b + I^{*2}\right)},$$

where I^* is given by the following equation

$$C_1 I^{*3} + C_2 I^{*2} + C_3 I^* + C_4 = 0 \tag{5.7}$$

where

$C_1 = (\mu + d + \delta)(\mu\alpha + \beta)$,

$C_2 = \mu(\mu + d + \delta) - A\beta$,

$C_3 = (b(\mu + d + \delta) + a)(\mu\alpha + \beta)$,

$C_4 = -\beta bA + \mu(a + \mu b + \delta b + db) = \mu b(\mu + d + \delta + \frac{a}{b})(1 - R_0)$.

Now applying Descartes's rule of signs [Wang (2004)], for $R_0 > 1$, the cubic equation admits a unique positive real root I^* if the condition $C_1 > 0, C_2 > 0, C_3 > 0$ and $C_4 < 0$ is satisfied.

After getting I^* we can obtain S^*. Thus, there exists an endemic equilibrium $Q^*(S^*, I^*)$ if above condition hold true.

The local stability of Q^* is explored as follows:

The characteristic equation of the system (5.3) obtained at $Q^*(S^*, I^*)$ is given as

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\tau} = 0,$$

where

$$p_0 = \left((2\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right), \; q_0 = \mu\left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right),$$

$$p_1 = \left(\frac{\beta I^*}{(1+\alpha I^*)} - \frac{\beta S^*}{(1+\alpha I^*)^2}\right), \; q_1 = \left(\beta\left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right)\frac{I^*}{(1+\alpha I^*)} - \frac{\mu\beta S^*}{(1+\alpha I^*)^2}\right).$$

Theorem 5.5: For $\tau = 0$, Q^* is locally asymptotically stable if both conditions $\frac{S^*}{(1+\alpha I^*)I^*} \leq 1$, and $\frac{\mu S^*}{(1+\alpha I^*)} \leq \left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right) I^*$ are satisfied simultaneously.

Proof: At Q^*, the characteristic equation at $\tau = 0$ is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1) = 0, \tag{5.8}$$

It is easy to show that if both conditions $\frac{S^*}{(1+\alpha I^*)I^*} \leq 1$, and $\frac{\mu S^*}{(1+\alpha I^*)} \leq \left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right) I^*$ are satisfied simultaneously, then

$$p_0 + p_1 = \left((2\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right) + \left(\frac{\beta I^*}{(1+\alpha I^*)} - \frac{\beta S^*}{(1+\alpha I^*)^2}\right)$$

$$= \left(\mu + (\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right) + \frac{\beta}{(1+\alpha I^*)}\left(I^* - \frac{S^*}{(1+\alpha I^*)}\right) > 0,$$

$$q_0 + q_1 = \mu\left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right) + \left(\beta\left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right)\frac{I^*}{(1+\alpha I^*)} - \frac{\mu\beta S^*}{(1+\alpha I^*)^2}\right)$$

$$= \mu\left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right) + \frac{\beta}{(1+\alpha I^*)}\left(\left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right) I^* - \frac{\mu S^*}{(1+\alpha I^*)}\right) > 0.$$

Hence, using the Routh-Hurwitz criterion, the endemic equilibrium Q^* of the system (5.3) is locally asymptotically stable at $\tau = 0$.

Theorem 5.6: For $\tau > 0$, Q^* is locally asymptotically stable if all the three conditions $\frac{\mu S^*}{(1+\alpha I^*)} \leq \left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right) I^*, \frac{\beta}{\mu} \leq \frac{(1+\alpha I^*)}{I^*}$ and $M_2 < M_1$ hold true simultaneously,

where

$$M_1 = \mu^2 + \left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b + I^{*2}\right)^2}\right)^2,$$

$$M_2 = \left(\frac{\beta I^*}{(1+\alpha I^*)} - \frac{\beta S^*}{(1+\alpha I^*)^2}\right)^2.$$

Proof: At Q^* the characteristic equation at $\tau > 0$ is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\tau} = 0 \quad (5.9)$$

For $\tau > 0$, by [Ruan and Wei (2003)], if instability occurs for a particular value of the delay τ, a characteristic root of Eq. (5.9) must intersect the imaginary axis. Therefore, we take $\lambda = i\omega, \omega > 0$ is the roots of Eq. (5.9.). Substituting $\lambda = i\omega$ in Eq. (5.9), we get

$$-\omega^2 + q_0 + p_1\omega \sin\omega\tau + q_1 \cos\omega\tau + i\,(p_1\omega \cos\omega\tau - q_1 \sin\omega\tau + p_0\omega) = 0 \quad (5.10)$$

On separating real and imaginary part of Eq. (5.10)

$$p_1\omega \sin\omega\tau + q_1 \cos\omega\tau = \omega^2 - q_0 \quad (5.11)$$

$$p_1\omega \cos\omega\tau - q_1 \sin\omega\tau = -p_0\omega \quad (5.12)$$

On squaring and adding both sides of Eqs. (5.11) and (5.12), we find

$$\omega^4 + ({p_0}^2 - 2q_0 - {p_1}^2)\omega^2 + ({q_0}^2 - {q_1}^2) = 0 \quad (5.13)$$

Let $\omega^2 = z_1$, Eq. (5.13) becomes

$$z_1{}^2 + Pz_1 + T = 0 \tag{5.14}$$

where $P = (p_0{}^2 - 2q_0 - p_1{}^2)$ and $T = (q_0{}^2 - q_1{}^2)$

It is easy to show that if all three conditions $\frac{\mu S^*}{(1+\alpha I^*)} \leq \left((\mu + d + \delta) + \frac{a\left(b - I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right) I^*, \frac{\beta}{\mu} \leq \frac{(1+\alpha I^*)}{I^*}$ and $M_2 < M_1$ are satisfied simultaneously, then

$$P = (p_0{}^2 - 2q_0 - p_1{}^2) = \left((2\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right)^2 - 2\mu\left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right) - \left(\frac{\beta I^*}{(1+\alpha I^*)} - \frac{\beta S^*}{(1+\alpha I^*)^2}\right)^2$$

$$= \mu^2 + \left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right)^2 - \left(\frac{\beta I^*}{(1+\alpha I^*)} - \frac{\beta S^*}{(1+\alpha I^*)^2}\right)^2$$

$$= M_1 - M_2 > 0.$$

$$T = (q_0{}^2 - q_1{}^2) = (q_0 - q_1)(q_0 + q_1)$$

$$= \left(\mu\left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right) - \left(\beta\left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right)\frac{I^*}{(1+\alpha I^*)} - \frac{\mu\beta S^*}{(1+\alpha I^*)^2}\right)\right)\left(\mu\left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right) + \left(\beta\left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right)\frac{I^*}{(1+\alpha I^*)} - \frac{\mu\beta S^*}{(1+\alpha I^*)^2}\right)\right)$$

$$= \left(\left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right)\left(\mu - \frac{\beta I^*}{(1+\alpha I^*)}\right) + \frac{\mu\beta S^*}{(1+\alpha I^*)^2}\right)\left(\mu\left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right) + \left(\beta\left((\mu + d + \delta) + \frac{a\left(b-I^{*2}\right)}{\left(b+I^{*2}\right)^2}\right)\frac{I^*}{(1+\alpha I^*)} - \frac{\mu\beta S^*}{(1+\alpha I^*)^2}\right)\right) > 0.$$

By Routh-Hurwitz criterion, Eq. (5.14) will have roots with negative real part, so this contradicts to our assumption for instability *i. e.* $\lambda = i\omega$. Hence, it is proved that Q^* of the model is locally asymptotically stable for $\tau > 0$.

5.5 Numerical simulation

In this section, we will discuss the results of numerical simulations of the system (5.3) numerically. We choose numerically experimental values of the parameters as given in Table 5.1.

Fig. 5.2 shows the behavior of susceptible and infected at different values of time lag $\tau = 0,1 \text{ and } 2$. It is evident that when the time lag is increasing, the susceptibles are decreasing and the number of infected is increasing. Furthermore, both populations are decreasing and increasing respectively to attain a steady state. Hence, it can be concluded that the time delay plays an important role to understand the infection progression in the human population.

Fig. 5.3 shows the difference in the infected population at various values of the transmission rate (β). It can be seen that as the values of β increases, the numbers of infected individuals also increase.

Fig. 5.4 demonstrates the changes in the infected population at various values of inhibition rate (α) according to which, the infected population is decreasing with the increase in the value of α .

Fig. 5.5 portrays the infected population at various values of cure rate/treatment rate (a). Clearly, as the value of treatment rate is increasing the infected population is decreasing.

Fig. 5.6 demonstrates the variation in the infected population with and without Monod-Haldane type treatment rate. According to the behavior of the graph, the infected population is less with M-H type treatment rate in comparison to the infected population without M-H type treatment rate. Hence, the M-H function type treatment rate plays a significant role in controlling the infection in the population.

5.6 Conclusions

In this chapter, we proposed a delayed SIR model along with Holling type II incidence and M-H functional type treatment rates. We showed that the model admits the disease-free equilibrium (DFE) and the endemic equilibrium (EE). We showed the stability of

DFE with the help of the basic reproduction number R_0. We proved that DFE is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$ for time lag $\tau \geq 0$. The stability of DFE at $R_0 = 1$ has been discussed by center manifold theory and it is investigated that the model undergoes a forward transcritical bifurcation. The stability of EE has been investigated by the Routh-Hurwitz criterion. We investigated that EE is locally asymptotically stable when the theorem 5.5 and theorem 5.6 hold true for time lag $\tau = 0$ and $\tau > 0$ respectively. The numerical simulations indicate that the infection will increase with the increased transmission rate, infection settles down even when there is no treatment, but at a higher value than with treatment. Moreover, the infection will decrease when there is an increase in the measures of inhibition adopted by infected. With the help of numerical simulations, we also observed that the infection may eradicate only when the treatment given to the infectives is appropriately managed according to the availability of the resources.

Table 5.1: Description and numerical values of parameters for simulation

Parameter	**Value**
Constant recruitment rate (A)	5
Inhibition rate due to infected (α)	0.002
Transmission rate (β)	0.004
Natural mortality rate (μ)	0.05
Disease induced mortality rate (d)	0.001
Recovery rate (δ)	0.002
Treatment rate or Cure rate (a)	0.2
Limitation rate (b) in treatment availability	0.004

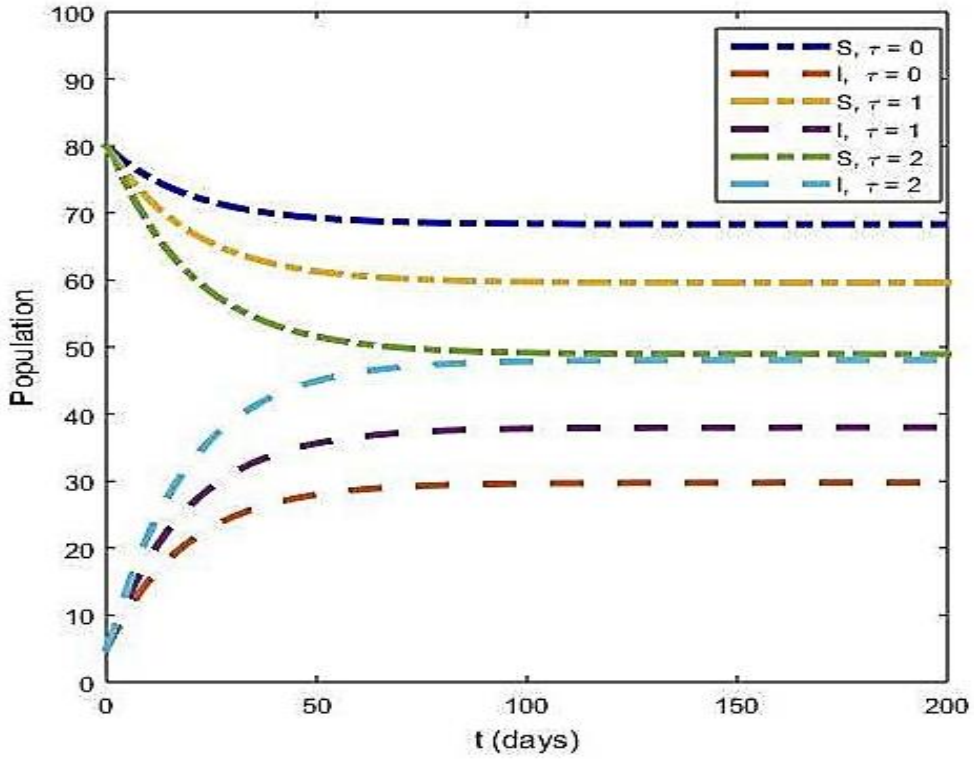


Fig. 5.2: Susceptible (S) and infected (I) population at various values of time lag τ.

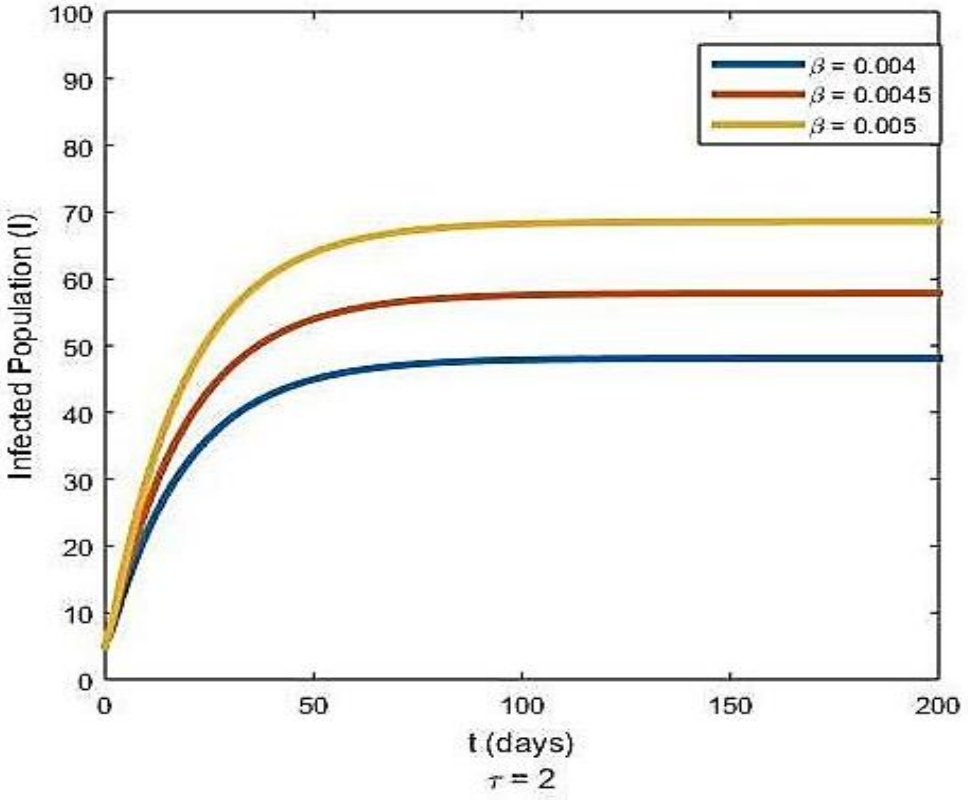


Fig. 5.3: Infected population (I) at various values of the transmission rate (β).

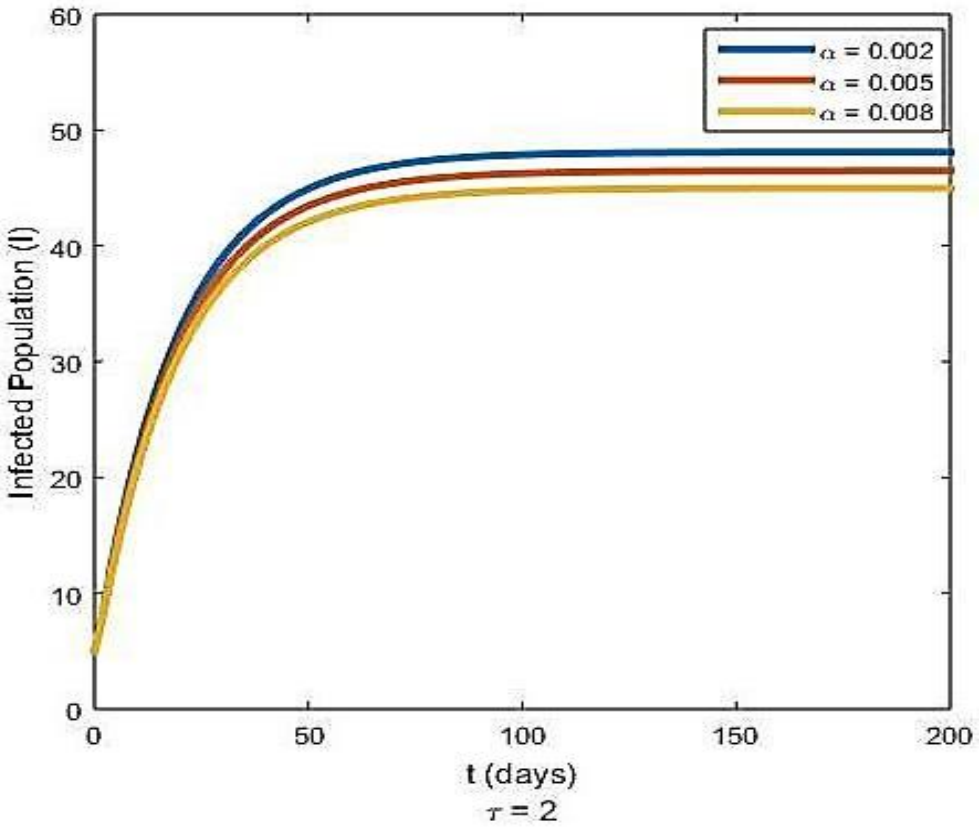


Fig. 5.4: Infected population (I) at various values of measures of inhibition(α).

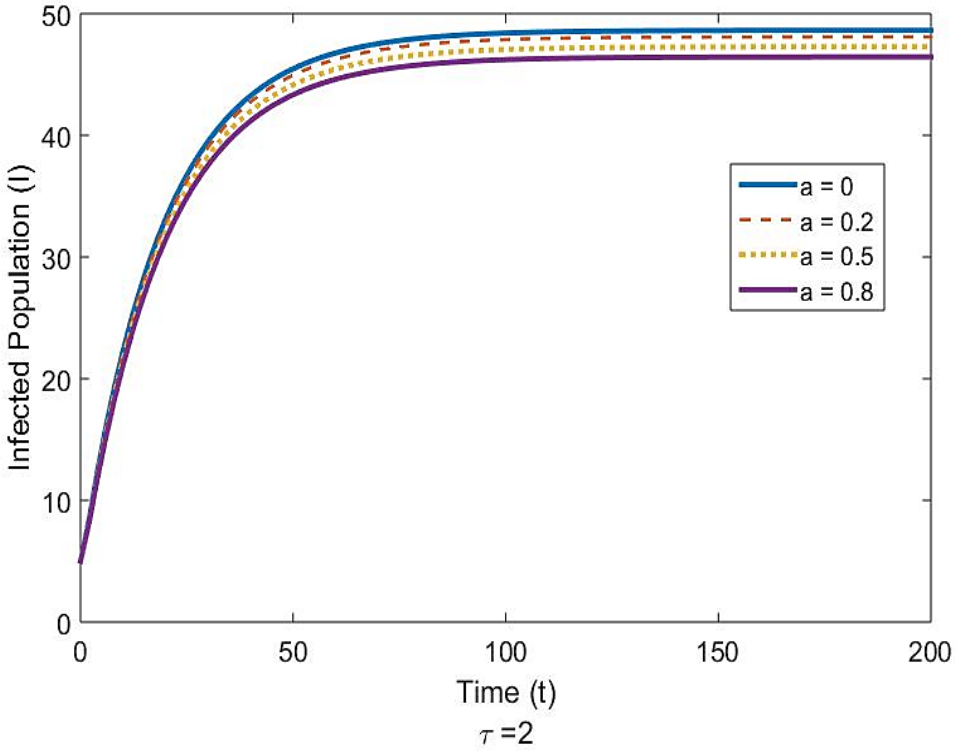


Fig. 5.5: Infected population (I) at various values of cure rate (a).

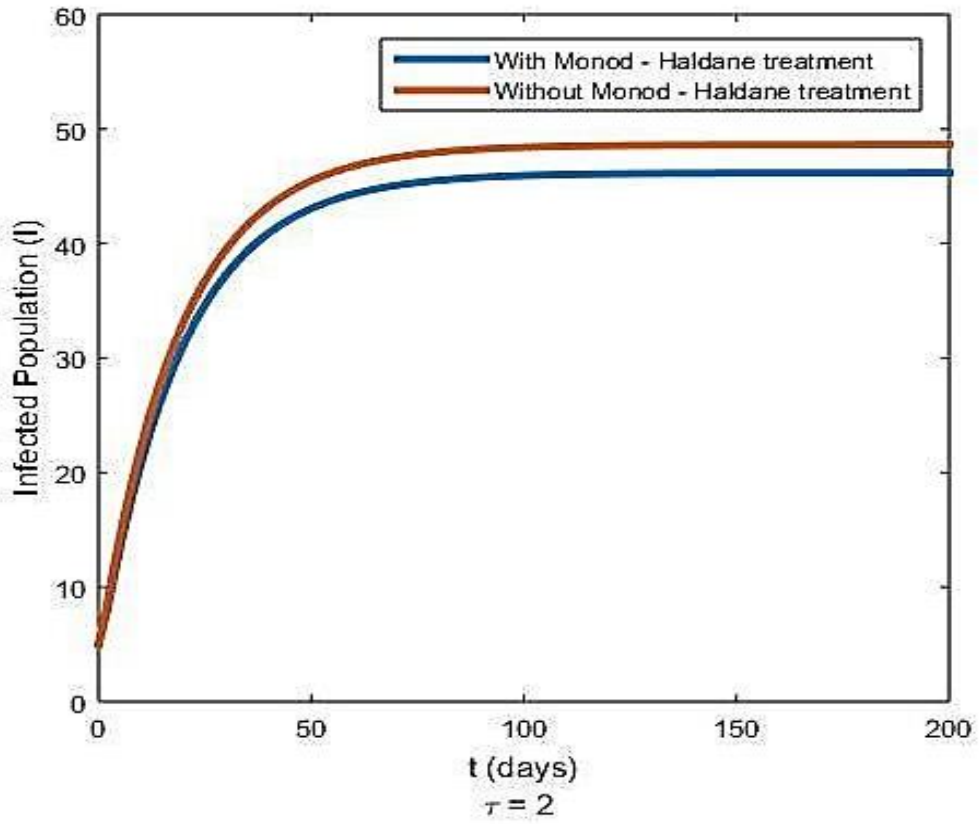


Fig. 5.6: Infected population (I) with and without Monod-Haldane (M-H) treatment rate with $a = 0.8$ and $b = 0.004$.

CHAPTER 6

TIME DELAYED SIR EPIDEMIC MODEL WITH MONOD-HALDANE INCIDENCE RATE AND DIFFERENT TREATMENT RATES

In case of an outbreak of an epidemic, psychological or inhibitory effects and various limitations on treatment methods play major roles in controlling the impact of an epidemic in society. The Monod-Haldane (M-H) functional type incidence rate is taken to annotate the psychological or inhibitory effect on the population with time delay representing the incubation period of the disease. The Holling type II & III treatment rates are considered to incorporate the limitation in treatment availability for infectives. Therefore, in the present paper, a novel combination of M-H incidence rate and two different treatment rates (Holling type II & III) is applied to the time-delayed SIR epidemic model to incorporate these important aspects. The mathematical analysis shows that the model has two equilibria, namely, disease-free equilibrium (DFE) and endemic equilibrium (EE). The detailed dynamical analysis of the model has been performed using the basic reproduction number R_0, center manifold theory, Routh-Hurwitz criterion and Lyapunov functional. It has been investigated that disease can be eradicated when R_0 is less than unity and disease will persist when R_0 is greater than unity. The Hopf bifurcation at endemic equilibrium has also been addressed. Further, global stability behavior of equilibria only for the second combination *i.e.* M-H incidence and Holling type III treatment rates has also been discussed. Finally, the numerical simulations have been performed to support our analytical findings.

6.1 Introduction

The widespread and frequent occurrences of many communicable diseases are a major problem for healthcare workers and policymakers all over the world. Controlling infectious diseases has been an increasingly complex issue in recent years. In order to control or to remove disease, a complete understanding of the dynamics of the disease progression is required. Based on the observed characteristics of infectious diseases, epidemiologists [Michael *et al.* (1999); Alexander *et al.* (2004); Gumel *et al.* (2006); Hattaf and Yousfi (2009); Xu and Ma (2009a); Zhang and Yaohong (2010); Hattaf *et al.* (2013); Zhou and Fan (2012); Dubey *et al.* (2013), (2015) & (2016)] have attempted to construct mathematical models that would make it possible to understand various aspects of many diseases and to suggest its control strategy. A pivotal issue in the study of the spread of an infectious disease is how it is transmitted. In epidemiology, the transmission of infectious disease is determined by the incidence rate which is defined as the average number of new cases infected by a disease per unit time. Therefore, the incidence rate plays a key role to study the qualitative description of transmission dynamics of the infectious disease.

In this chapter, to describe the psychological effect from the behavioral change of the susceptible individuals when the number of infective people is very high, we have considered Monod-Haldane (M-H) type incidence rate. This is a non-monotone type incidence rate, which interprets the "psychological" effects [Liu *et al.* (1987)]. The detailed explanation of the M-H functional type incidence rate has already been given in section 2.1. For most communicable diseases there is an interval between infection and visibility of symptoms (the incubation period) in which the infectious agent is multiplying or developing. To push the epidemic models into a more realistic state, we have considered M-H functional type incidence rate with the inclusion of time delay (representing the incubation period). To contribute to the nonlinear mechanism of the epidemic, we have incorporated nonlinear incidence rate as M-H functional with the inclusion of time delay and treatment rate in two different forms (Holling functional type II & III) in our model. The detailed explanation of Holling type II and Holling type III treatment rates have already been given in section 3.1 and section 4.1 respectively.

In this chapter, we have examined the impact of time lag on the SIR epidemic model with Monod-Haldane functional type incidence rate and Holling type II & III treatment rates, separately, for better understanding of the disease mechanism. Further, we have evaluated the basic reproduction number (R_0) [Driessche and Watmough (2002)] for both the combinations of incidence and treatment rates. Furthermore, for the model dynamics, stability analysis of the equilibria has been analyzed by the basic reproduction number, center manifold theory, Descartes's Rule [Wang (2004)], Routh-Hurwitz criterion and Lyapunov direct method. Only local stability of model equilibria is discussed for the combination of M-H functional type incidence and Holling type II treatment rates, whereas for the combination of M-H functional type treatment and Holling type III treatment rates local as well as global stability of equilibria are discussed.

6.2 Mathematical model

Mathematical models help to study the transmission dynamics and spread of infectious diseases, to recognize the factors governing the transmission process in order to improve effective control strategies and to evaluate the efficacy of surveillance strategies and possible interventions. Therefore, we propose a mathematical SIR model along with time delay, nonlinear M-H functional type incidence rate, and two different treatment rates. We assume that at time t the total population is $N(t)$, with the immigration of susceptible individuals at a constant rate A. Further, it is assumed that the total population $N(t)$ is divided into three disjoint subclasses of individuals, namely; susceptible $S(t)$, infectives $I(t)$, and recovered $R(t)$. It is assumed that the disease can be spread due to the direct contact between susceptible and infectives only. Let μ be the natural death rate of the population, d be the disease induced death rate and δ be the recovery rate of infected individuals. The progression dynamics of the infection is given by the block diagram in Fig. 6.1 below.

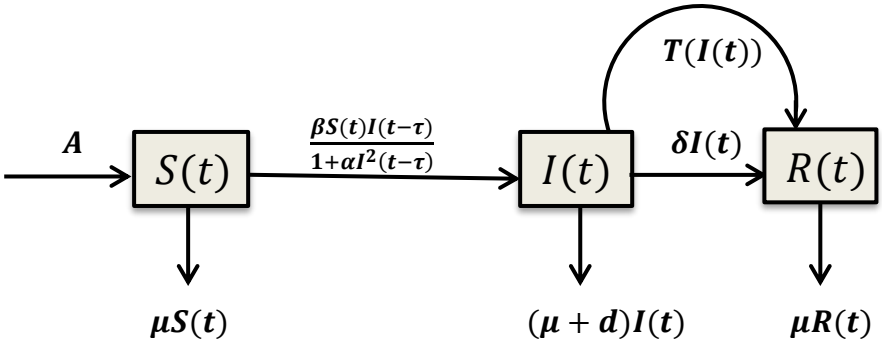


Fig. 6.1: Transfer diagram of the infection through various compartments.

The dynamics of the model is given by the following system of nonlinear delay differential equations:

$$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta S(t)I(t-\tau)}{1+\alpha I^2(t-\tau)},$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t-\tau)}{1+\alpha I^2(t-\tau)} - (\mu + d + \delta)I(t) - T(I(t)), \tag{6.1}$$

$$\frac{dR(t)}{dt} = T(I(t)) + \delta I(t) - \mu R(t).$$

where time lag $\tau > 0$ represents the incubation period of the disease.

The term $T(I(t))$ denotes the nonlinear saturated treatment rate. $T(I(t))$ Is taken in two following form:

i. $T_1(I(t)) = \frac{a\, I(t)}{(1+b\, I(t))}$ (Holling type II treatment rate).

ii. $T_2(I(t)) = \frac{a\, I^2(t)}{(1+b\, I^2(t))}$ (Holling type III treatment rate).

Let $C = C([-\tau, 0], \mathbb{R}^3)$ denotes the Banach space of continuous functions, mapping the interval $[-\tau, 0]$ to $\mathbb{R}^3$ with the topology of uniform convergence. It is well known by the fundamental theory of functional differential equations [Hattaf *et al.* (2013); Xu and Ma (2009a); Hale and Lunel (1993); Kuang (1993)] that the model (6.1) admits a unique solution $(S(t),\ I(t),\ R(t))$ with initial data $(S_0, I_0, R_0) \in C$. For biological reasons the initial conditions of the model (6.1) are non-negative continuous functions

$$S_0(\varphi) \geq 0, I_0(\varphi) \geq 0, R_0(\varphi) \geq 0,\ \varphi \in [-\tau, 0]. \tag{6.2}$$

The term $\frac{\beta S(t)I(t-\tau)}{1+\alpha I^2(t-\tau)}$ in the model represents the nonlinear M-H functional type incidence rate with time lag τ, here β is the transmission rate of infection and α measures the inhibitory or psychological effects due to the infected individuals. The term $\frac{aI(t)}{1+bI(t)}$ & $\frac{aI^2(t)}{1+bI^2(t)}$ in the model, represent Holling type II & III treatment rates, where a and b are both non-negative constants. The parameters a and b are the cure rate given to infectives and the rate of limitation in treatment availability, respectively. The movement of an epidemic in various classes is presented by the transfer diagram as given in Fig. 6.1.

6.3 Basic properties of the model

The first two equations of the model (6.2) do not depend on the third equation; therefore, without loss of generality, it is sufficient to consider the following reduced system for the analysis:

$$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta S(t)I(t-\tau)}{1+\alpha I^2(t-\tau)},$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t-\tau)}{1+\alpha I^2(t-\tau)} - (\mu + d + \delta)I(t) - T(I(t)) \ , \tag{6.3}$$

with initial conditions

$$S(\theta) = \varphi_1(\theta), I(\theta) = \varphi_2(\theta), \ \varphi_i(\theta) \geq 0, \theta \in [-\tau, 0], \varphi_i(0) > 0 \ (i = 1,2) \tag{6.4}$$

In the system (6.3), for ecological reasons, it is assumed that all parameters $A, \mu, \beta, d, \delta, a, \alpha$ and b are positive. Since the system (6.3) monitors the population, it is important to show that all state variables with non-negative initial data will remain non-negative and bounded for all time. Thus, we have the following theorem:

Theorem 6.1: All state variables of the system (6.3), subject to the condition (6.4), remain non-negative and bounded for all $t \geq 0$.

Proof: The proof of this theorem is similar to the proof of theorems 4.1 & 4.2 as given in section 4.1. Hence, it is omitted here.

6.4 Equilibrium points

In this section, we obtain the equilibrium points of the system (6.3). The equilibria of the system (6.3) are calculated by putting the right-hand terms to zero which are as follow:

i. Disease-free equilibrium (DFE) $Q(\frac{A}{\mu}, 0)$,

ii. Endemic equilibrium (EE) $Q^*(S^*, I^*)$.

6.5 Stability analysis of the equilibria for the combination of M-H type incidence and Holling type II treatment rates

In this section, we discuss the local stability of model equilibria when the incidence rate is M-H functional type and treatment rate is Holling functional type II. To study the stability behavior, first, we compute the basic reproduction number (R_0).

6.5.1 Computation of basic reproduction number (R_0)

The characteristic equation at $Q\left(\frac{A}{\mu}, 0\right)$ of the system (6.3) is given by

$$(\mu + \lambda)\left(\frac{\beta A}{\mu}\, e^{-\lambda\tau} - \mu - d - \delta - a - \lambda\right) = 0 \tag{6.5}$$

One root of Eq. (6.5) is given by $\lambda_1 = -\mu$ and other roots can be obtained from the following equation:

$$\left(\frac{\beta A}{\mu}\, e^{-\lambda\tau} - \mu - d - \delta - a - \lambda\right) = 0$$

The term $\frac{\beta A}{\mu(\mu+d+\delta+a)} e^{-\lambda\tau}$ at $\tau = 0$ is the basic reproduction number R_0 [Driessche and Watmough (2002)] of our model i.e.

$$R_0 = \frac{\beta A}{\mu(\mu + d + \delta + a)}.$$

6.5.2 Analysis for $R_0 \neq 1$

Clearly, Eq. (6.5) has a negative root $\lambda_1 = -\mu$ and other roots are the solution of the equation

$$\lambda + \mu + d + \delta + a - \frac{\beta A}{\mu} e^{-\lambda\tau} = 0 \tag{6.6}$$

Let

$$f(\lambda) = \lambda + \mu + d + \delta + a - \frac{\beta A}{\mu} e^{-\lambda\tau}$$

If $R_0 > 1$, then for real λ,

$$f(0) = \mu + d + \delta + a - \frac{\beta A}{\mu} < 0, \qquad \lim_{\lambda \to +\infty} f(\lambda) \to +\infty$$

Hence, if $R_0 > 1$ then $f(\lambda) = 0$ has a positive real root.

If $R_0 < 1$, we suppose that $Re\ \lambda \geq 0$.

We observe that

$$Re\ \lambda = \frac{\beta A}{\mu} e^{-Re\,\lambda\,\tau} \cos Im\ \lambda\ \tau - (\mu + d + \delta + a) \leq \frac{\beta A}{\mu} - (\mu + d + \delta + a) < 0$$

a contradiction to our assumption. Hence, if $R_0 < 1$ then λ is a root of Eq. (6.5) with the negative real part.

Thus, the following theorem is proposed:

Theorem 6.2: DFE is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ for $\tau \geq 0$.

6.5.3 Analysis at $R_0 = 1$

i. For $\tau > 0$

If $R_0 = 1$, then $\lambda = 0$ is a simple root of Eq. (6.5). Let $\lambda = x + iy$ be any of the other solutions, then Eq. (6.6) change into:

$$x + iy \ + \mu + d + \delta + a - \frac{\beta A}{\mu} \, e^{-(x+iy)\tau} = 0 \tag{6.7}$$

By using Euler's formula and by separating real and imaginary parts we can write

$$x + \mu + d + \delta + a = \frac{\beta A}{\mu} \, Cos \, y\tau \, e^{-x\tau}, \; y = -\frac{\beta A}{\mu} \, Sin \, y\tau \, e^{-x\tau} \tag{6.8}$$

$R_0 = 1$ implies $\frac{\beta A}{\mu} = (\mu + d + \delta + a)$. Moreover, there exists root that satisfies both the equations of (6.8), then they will also satisfy the equation obtained on squaring and adding them member to member, as follows

$$(x + \mu + d + \delta + a)^2 + y^2 = (\mu + d + \delta + a)^2 e^{-2x\tau}. \tag{6.9}$$

To verify Eq. (6.9), we must have $x \leq 0$. Thus, we proposed the following theorem:

Theorem 6.3: DFE of the system (6.3) is linearly neutrally stable if $R_0 = 1$.

ii. For $\tau = 0$

When we evaluate the system (6.3) at $R_0 = 1$ and bifurcation parameter $\beta = \beta^* = \frac{\mu(\mu+d+\delta+a)}{A}$, we obtain that system has a zero eigenvalue and another eigenvalue is negative. Therefore, the stability behavior of DFE at $R_0 = 1$ cannot be examined using linearization technique. So, to examine the behavior of the equilibrium point, we use center manifold theory [Sastry (1999)]. To apply the center manifold theory, we redefine $S(t) = x_1$ and $I(t) = x_2$ then the system (6.3) can be re-written as

$$\frac{dx_1}{dt} = A - \mu x_1 - \frac{\beta x_1 x_2}{1+\alpha {x_2}^2} \equiv f_1 \,,$$

$$\frac{dx_2}{dt} = \frac{\beta x_1 x_2}{1+\alpha {x_2}^2} - (\mu + d + \delta)x_2 - \frac{a x_2}{1+b x_2} \equiv f_2. \tag{6.10}$$

The Jacobian matrix, denoted by J^* of the system (6.10) evaluated at $R_0 = 1$ and $\beta = \beta^*$is given by

$$J^* = \begin{bmatrix} -\mu & -\dfrac{\beta^* A}{\mu} \\ 0 & 0 \end{bmatrix}$$

Let $u = [u_1, u_2\,]$ and $w = [w_1, w_2\,]^T$ be the left eigenvector and right eigenvector of J^*corresponding to the zero eigenvalue. Then we have

$$u_1 = 0, u_2 = 1 \text{ and } w_1 = -\frac{\beta^* A}{\mu^2}, w_2 = 1.$$

The non-zero partial derivatives associated with the functions of the system (6.10) evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$$\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_Q = \left(\frac{\partial^2 f_2}{\partial x_2 \partial x_1}\right)_Q = \beta^*, \text{ and } \left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_Q = \frac{A}{\mu}.$$

Then, from [Chavez and Song (2004)], the bifurcation constants $a_1$and b_1 are

$$a_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q$$

$$= u_2(2w_1 w_2\, \beta^* + {w_2}^2.0 + {w_1}^2.0)$$

$$= -2\frac{{\beta^*}^2 A}{\mu^2} < 0,$$

and

$$b_1 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q$$

$$= u_2\left(w_2 \frac{A}{\mu}\right)$$

$$= \frac{A}{\mu} > 0.$$

The bifurcation constants $a_1 < 0$ and $b_1 > 0$. Hence, from Theorem 4.1(iv) of [Chavez and Song (2004)], we propose the following theorem:

Theorem 6.4: DFE exhibits the forward bifurcation when the basic reproduction number is equal to unity.

The bifurcation is illustrated in Fig. 6.2.

6.5.4 Existence and stability analysis of endemic equilibrium

To investigate the conditions for the existence of the endemic equilibrium $Q^*(S^*, I^*)$, the system (6.3) is rearranged to get S^*, and I^* which gives

$$S^* = \frac{\big((\mu + d + \delta)(1 + bI^*) + a\big)(1 + \alpha I^{*2})}{\beta(1 + bI^*)},$$

and I^* is given by the equation

$$C_1 I^{*3} + C_2 I^{*2} + C_3 I^* + C_4 = 0 \qquad (6.11)$$

where

$C_1 = \mu\alpha b(\mu + d + \delta)$,

$C_2 = \mu\alpha(\mu + d + \delta + a) + \beta b(\mu + d + \delta)$,

$C_3 = (\mu b + \beta)(\mu + d + \delta) + \beta a - A\beta b$,

$C_4 = \mu(\mu + d + \delta + a) - A\beta = \mu(\mu + d + \delta + a)(1 - R_0)$.

Using Descartes' rule of the signs, for $R_0 > 1$, the existence of a unique positive real root I^* of Eq. (6.11) is required to satisfy any of the following conditions:

i. $C_1 > 0, C_2 > 0, C_3 < 0$ and $C_4 < 0$.

ii. $C_1 > 0, C_2 > 0, C_3 > 0$ and $C_4 < 0$.

After getting the value of I^*, we can obtain the value of S^*. Hence, a unique $Q^*(S^*, I^*)$ exists if one of the above conditions holds true.

Now, we explore the local stability of Q^* as follows:

The characteristic equation of the system (6.3) obtained at Q^* is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\tau} = 0 \qquad (6.12)$$

where

$p_0 = (2\mu + d + \delta) + \frac{a}{(1+bI^*)^2} + \frac{\beta I^*}{(1+\alpha I^{*2})}$,

$q_0 = \left(\mu + \frac{\beta I^*}{(1+\alpha I^{*2})}\right)\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right)$,

$p_1 = -\frac{\beta S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2}$,

$q_1 = -\frac{\beta\mu S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2}$.

Theorem 6.5: At $\tau = 0$, Q^* is locally asymptotically stable if $S^* \leq I^*\left(1 + \alpha I^{*2}\right)$ holds true.

Proof: At Q^*, the characteristic equation at $\tau = 0$ is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1) = 0 \tag{6.13}$$

It is easy to show that if $S^* \leq I^*\left(1 + \alpha I^{*2}\right)$ is satisfied then

$$p_0 + p_1 = (2\mu + d + \delta) + \frac{a}{(1+bI^*)^2} + \frac{\beta I^*}{\left(1+\alpha I^{*2}\right)} - \frac{\beta S^*\left(1-\alpha I^{*2}\right)}{\left(1+\alpha I^{*2}\right)^2}$$

$$= (2\mu + d + \delta) + \frac{a}{(1+bI^*)^2} + \frac{\alpha\beta S^* I^{*2}}{\left(1+\alpha I^{*2}\right)^2} + \frac{\beta}{\left(1+\alpha I^{*2}\right)}\left(I^* - \frac{S^*}{\left(1+\alpha I^{*2}\right)}\right) > 0.$$

$$q_0 + q_1 = \left(\mu + \frac{\beta I^*}{\left(1+\alpha I^{*2}\right)}\right)\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) - \frac{\beta\mu S^*\left(1-\alpha I^{*2}\right)}{\left(1+\alpha I^{*2}\right)^2}$$

$$= \mu\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) + \frac{\beta I^*}{\left(1+\alpha I^{*2}\right)}\left((d + \delta) + \frac{a}{(1+bI^*)^2}\right) + \frac{\beta\mu\alpha I^{*2} S^*}{\left(1+\alpha I^{*2}\right)^2} + \frac{\beta\mu}{\left(1+\alpha I^{*2}\right)}\left(I^* - \frac{S^*}{\left(1+\alpha I^{*2}\right)}\right) > 0.$$

Therefore, using the Routh-Hurwitz criterion, Q^* is locally asymptotically stable when $\tau = 0$.

Theorem 6.6: For $\tau > 0$, Q^* is locally asymptotically stable if $S^* \leq \frac{I^*\left(1+\alpha I^{*2}\right)}{1-\alpha I^{*2}}$ holds true.

Proof: At Q^* the characteristic equation for $\tau > 0$ is given by the Eq. (6.12)

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\tau} = 0$$

For $\tau > 0$, according to Ruan and Wei [2003], for the occurrence of the instability, a characteristic root of the Eq. (6.12) must cross the imaginary axis for a specific value of τ. In this manner, we assume that $\lambda = i\omega, \omega > 0$ is the root of the Eq. (6.12). Putting $\lambda = i\omega$ in Eq. (6.12) gives:

$$-\omega^2 + q_0 + p_1\omega \sin\omega\tau + q_1 \cos\omega\tau + i\,(p_1\omega \cos\omega\tau - q_1 \sin\omega\tau + p_0\omega) = 0 \tag{6.14}$$

By using Euler's formula and separating the real and imaginary part of Eq. (6.14), we get

$$p_1\omega \sin \omega\tau + q_1 \cos \omega\tau = \omega^2 - q_0 \tag{6.15}$$

$$p_1\omega \cos \omega\tau - q_1 \sin \omega\tau = -p_0\omega \tag{6.16}$$

Squaring and adding both sides of Eqs. (6.15) & (6.16) yields

$$\omega^4 + ({p_0}^2 - 2q_0 - {p_1}^2)\omega^2 + ({q_0}^2 - {q_1}^2) = 0 \tag{6.17}$$

Assuming $\omega^2 = z_1$, Eq. (6.17) becomes

$$ {z_1}^2 + Pz_1 + T = 0 \tag{6.18}$$

here, $P = ({p_0}^2 - 2q_0 - {p_1}^2)$ and $T = ({q_0}^2 - {q_1}^2)$

It is easy to show that if $S^* \leq \frac{I^*\left(1+\alpha {I^*}^2\right)}{1-\alpha {I^*}^2}$ is satisfied then

$$P = ({p_0}^2 - 2q_0 - {p_1}^2)$$

$$= \left((2\mu + d + \delta) + \frac{a}{(1+bI^*)^2} + \frac{\beta I^*}{\left(1+\alpha {I^*}^2\right)}\right)^2 - 2\left(\mu + \frac{\beta I^*}{\left(1+\alpha {I^*}^2\right)}\right)\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) - \left(\frac{\beta S^*\left(1-\alpha {I^*}^2\right)}{\left(1+\alpha {I^*}^2\right)^2}\right)^2 = \left(\mu + \frac{\beta I^*}{\left(1+\alpha {I^*}^2\right)}\right)^2 + \left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right)^2 - \left(\frac{\beta S^*\left(1-\alpha {I^*}^2\right)}{\left(1+\alpha {I^*}^2\right)^2}\right)^2$$

$$= \mu^2 + \frac{2\mu\beta I^*}{\left(1+\alpha {I^*}^2\right)} + \left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right)^2 + \frac{\beta^2}{\left(1+\alpha {I^*}^2\right)^2}\left({I^*}^2 - \frac{\left(S^*\left(1-\alpha {I^*}^2\right)\right)^2}{\left(1+\alpha {I^*}^2\right)^2}\right) > 0,$$

$$T = ({q_0}^2 - {q_1}^2)$$

$$= \left(\left(\mu + \frac{\beta I^*}{\left(1+\alpha {I^*}^2\right)}\right)\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right)\right)^2 - \left(\frac{\beta\mu S^*\left(1-\alpha {I^*}^2\right)}{\left(1+\alpha {I^*}^2\right)^2}\right)^2$$

$$= \left(\mu\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) + \frac{\beta I^*}{\left(1+\alpha {I^*}^2\right)}\left((d + \delta) + \frac{a}{(1+bI^*)^2}\right) + \frac{\beta\mu I^*}{\left(1+\alpha {I^*}^2\right)}\right)^2 - \left(\frac{\beta\mu S^*\left(1-\alpha {I^*}^2\right)}{\left(1+\alpha {I^*}^2\right)^2}\right)^2$$

$$= \left(\mu\left((\mu+d+\delta)+\frac{a}{(1+bI^*)^2}\right)+\frac{\beta I^*}{(1+\alpha I^{*2})}\left((d+\delta)+\frac{a}{(1+bI^*)^2}\right)\right)^2 + 2\left(\mu\left((\mu+d+\delta)+\frac{a}{(1+bI^*)^2}\right)+\frac{\beta I^*}{(1+\alpha I^{*2})}\left((d+\delta)+\frac{a}{(1+bI^*)^2}\right)\right)\left(\frac{\beta\mu I^*}{(1+\alpha I^{*2})}\right)+\frac{\beta^2\mu^2}{(1+\alpha I^{*2})^2}\left(I^{*2}-\frac{\left(S^*\left(1-\alpha I^{*2}\right)\right)^2}{(1+\alpha I^{*2})^2}\right) > 0.$$

Clearly, if $P = ({p_0}^2 - 2q_0 - {p_1}^2) > 0$ and $T = ({q_0}^2 - {q_1}^2) > 0$ are satisfied simultaneously then by Routh-Hurwitz Criterion, Eq. (6.18) will always have roots with the negative real part. It contradicts our assumption for instability that $\lambda = i\omega$ is a root of Eq. (6.12). Hence, the endemic equilibrium Q^* of the system (6.3) is locally asymptotically stable for $\tau > 0$.

6.5.5 Hopf bifurcation analysis

If $T < 0$ in Eq. (6.18) then there is unique positive ω_0 satisfying the Eq. (6.18) i. e. there is a single pair of purely imaginary roots $\pm i\omega_0$ to Eq. (6.18).

From Eq. (6.15) and Eq. (6.16), τ_n corresponding to ω_0 can be obtained as

$$\tau_n = \frac{1}{\omega_0}\arccos\left(\frac{(q_1-p_0p_1){\omega_0}^2-q_0q_1}{{p_1}^2{\omega_0}^2+{q_1}^2}\right)+\frac{2n\pi}{\omega_0},\ n = 0, 1, 2, ... \tag{6.19}$$

For $\tau = 0$, endemic equilibrium Q^* is stable, it remains stable for $\tau < \tau_0$ if $\frac{d}{dt}(Re\ (\lambda))\Big|_{\lambda=i\omega_0} > 0$.

Differentiating Eq. (6.12) with respect to τ, we get

$$\left(2\lambda + p_0 + p_1e^{-\lambda\tau} - (p_1\lambda+q_1)\tau e^{-\lambda\tau}\right)\frac{d\lambda}{d\tau} = \lambda(p_1\lambda+q_1)e^{-\lambda\tau} \tag{6.20}$$

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{\left(2\lambda+p_0+p_1e^{-\lambda\tau}-(p_1\lambda+q_1)\tau e^{-\lambda\tau}\right)}{\lambda(p_1\lambda+q_1)e^{-\lambda\tau}} = \frac{(2\lambda+p_0)}{\lambda(p_1\lambda+q_1)e^{-\lambda\tau}} + \frac{p_1}{\lambda(p_1\lambda+q_1)} - \frac{\tau}{\lambda}$$

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{(2\lambda+p_0)}{-\lambda(\lambda^2+p_0\lambda+q_0)} + \frac{p_1}{\lambda(p_1\lambda+q_1)} - \frac{\tau}{\lambda}$$

$$\frac{d}{d\tau}\left(Re\ (\lambda)\right)\Big|_{\lambda=i\omega_0} = Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\Big|_{\lambda=i\omega_0}$$

$$= Re\left(\frac{(2i\omega_0+p_0)}{-i\omega_0(-{\omega_0}^2+ip_0\omega_0+q_0)} + \frac{p_1}{i\omega_0(ip_1\omega_0+q_1)} - \frac{\tau}{i\omega_0}\right)$$

$$= Re\left(\frac{1}{\omega_0}\left(\frac{(2i\omega_0+p_0)}{({\omega_0}^2-q_0)i+p_0\omega_0)} + \frac{p_1}{(-p_1\omega_0+iq_1)} + i\tau\right)\right)$$

$$= \frac{1}{\omega_0}\left(\frac{2\omega_0({\omega_0}^2-q_0)+{p_0}^2\omega_0}{({\omega_0}^2-q_0)^2+(p_0\omega_0)^2} - \frac{{p_1}^2\omega_0}{(p_1\omega_0)^2+{q_1}^2}\right)$$

$$= \frac{2{\omega_0}^2+({p_0}^2-2q_0-{p_1}^2)}{(p_1\omega_0)^2+{q_1}^2}.$$ (Since, from Eqs. (6.15) & (6.16),$({\omega_0}^2 - q_0)^2 + (p_0\omega_0)^2 = (p_1\omega_0)^2 + {q_1}^2$)

Under the condition ${p_0}^2 - 2q_0 - {p_1}^2 > 0$, we have $\frac{d}{d\tau}\left(Re\ (\lambda)\right)\Big|_{\lambda=i\omega_0} > 0$.

Hence, the transversality condition holds and Hopf bifurcation occurs at $\omega = \omega_0, \tau = \tau_0$.

By summarizing the above analysis, we arrive at the following Theorem.

Theorem 6.7: The endemic equilibrium (Q^*) of the system (6.3) is asymptotically stable for $\tau \in [0, \tau_0)$ and it undergoes Hopf bifurcation at $\tau = \tau_0$.

6.6 Stability analysis of the equilibria for the combination of M-H type incidence and Holling type III treatment rates

In this section, we discuss the local and global stability of equilibria when the incidence rate is M-H functional type and treatment rate is Holling functional type III. For the stability of equilibria first, we determine the basic reproduction number R_0 as given below:

6.6.1 Computation of basic reproduction number(R_0)

The characteristic equation of the system (6.3) at Q is given by

$$(\mu+\lambda)\left(\frac{\beta A}{\mu}\,\mathrm{e}^{-\lambda\tau}-\mu-d-\delta-\lambda\right)=0. \tag{6.21}$$

The one root of Eq. (6.21) is given by $\lambda_1=-\mu$ and other roots are the solution to the following equation:

$$\left(\frac{\beta A}{\mu}\,\mathrm{e}^{-\lambda\tau}-\mu-d-\delta-\lambda\right)=0.$$

The term $\frac{\beta A}{\mu(\mu+d+\delta+a)}\mathrm{e}^{-\lambda\tau}$ at $\tau=0$, is defined as the basic reproduction number denoted by R_0 *i.e.* the basic reproduction number for our model is

$$R_0=\frac{\beta A}{\mu(\mu+d+\delta)}.$$

6.6.2 Analysis at $R_0\neq 1$

Clearly, Eq. (6.21) has a negative real root $\lambda_1=-\mu$ and other root can be obtained by solving the following equation:

$$\lambda+\mu+d+\delta-\frac{\beta A}{\mu}\,\mathrm{e}^{-\lambda\tau}=0.$$

Let

$$f(\lambda)=\lambda+\mu+d+\delta-\frac{\beta A}{\mu}\,\mathrm{e}^{-\lambda\tau}.$$

If $R_0>1$, it can be seen that for real λ,

$$f(0)=(\mu+d+\delta)\left(1-\frac{\beta A}{\mu(\mu+d+\delta)}\right)<0,\ \lim_{\lambda\to+\infty} f(\lambda)\to+\infty.$$

Hence, if $R_0>1$ then there exists at least one positive root of $f(\lambda)=0$.

If $R_0<1$, we assume that $Re\ (\lambda)\geq 0$.

We notice that

$$Re\ (\lambda)=\frac{\beta A}{\mu}\,\mathrm{e}^{-Re(\lambda\tau)}\cos\big(Im\ (\lambda\tau)\big)-(\mu+d+\delta)\leq\left(\frac{\beta A}{\mu(\mu+d+\delta)}-1\right)(\mu+d+\delta)<0.$$

It contradicts to our assumption that $Re\ (\lambda) \geq 0$. Thus, the root λ of Eq. (6.21) has a negative real part if $R_0 < 1$. Hence, we state the following theorem:

Theorem 6.8: DFE $Q\left(\frac{A}{\mu}, 0\right)$ of the system (6.3) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

6.6.3 Analysis at $R_0 = 1$

i. For $\tau > 0$

If $R_0 = 1$, then $\lambda = 0$ is a simple root of Eq. (6.21). Let $\lambda = x + iy$ be any other solution, then Eq. (6.21) changes into:

$$x + iy\ + \mu + d + \delta - \frac{\beta A}{\mu}\ e^{-(x+iy)\tau} = 0 \tag{6.22}$$

By using Euler's formula and on separating real and imaginary parts we can write

$$x + \mu + d + \delta = \frac{\beta A}{\mu}\cos y\tau\ e^{-x\tau},\ y = -\frac{\beta A}{\mu}\sin y\tau\ e^{-x\tau} \tag{6.23}$$

$R_0 = 1$ implies $\frac{\beta A}{\mu} = (\mu + d + \delta)$. Moreover, there exists root satisfying both the equations of Eq. (6.23), then they also satisfy the equation obtained by squaring and adding them member to member, as given below:

$$(x + \mu + d + \delta)^2 + y^2 = (\mu + d + \delta)^2 e^{-2x\tau}. \tag{6.24}$$

To verify Eq. (6.24), we must have $x \leq 0$. Thus, we propose the following theorem:

Theorem 6.9: DFE of the system (6.3) is linearly neutrally stable if $R_0 = 1$.

ii. For $\tau = 0$

We notice that the system (6.3) is being evaluated at $R_0 = 1$ and bifurcation parameter $\beta = \beta^* = \frac{\mu(\mu+d+\delta)}{A}$ has a zero eigenvalue and another eigenvalue is negative. Since it is

not possible to analyse the stability behaviour of DFE Q at $R_0 = 1$ using linearization, therefore we use center manifold theory [Sastry (1999)]. For this, we redefine $S = y_1$ and $I = y_2$ then the system (6.3) can be rewritten as:

$$\frac{dy_1}{dt} = A - \mu y_1 - \frac{\beta y_1 y_2}{1+\alpha {y_2}^2} \equiv G_1,$$

$$\frac{dy_2}{dt} = \frac{\beta y_1 y_2}{1+\alpha {y_2}^2} - (\mu + d + \delta) y_2 - \frac{a {y_2}^2}{1+b {y_2}^2} \equiv G_2. \quad (6.25)$$

Let J^* be the Jacobian matrix at $R_0 = 1$ and bifurcation parameter $\beta = \beta^*$. Then

$$J^* = \begin{bmatrix} -\mu & -\frac{\beta^* A}{\mu} \\ 0 & 0 \end{bmatrix}.$$

Let $u = [u_1, u_2]$ and $w = [w_1, w_2]^T$ denote the left eigenvector and right eigenvector of the Jacobian matrix J^*corresponding to the zero eigenvalue. Then we have

$$u_1 = 0, u_2 = 1 \text{ and } w_1 = -\frac{\beta^* A}{\mu^2}, w_2 = 1.$$

The non-zero partial derivatives associated with the functions G_1 and G_2 of the system (6.22) evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$$\left(\frac{\partial^2 G_2}{\partial y_1 \partial y_2}\right)_Q = \left(\frac{\partial^2 G_2}{\partial y_2 \partial y_1}\right)_Q = \beta^*, \frac{\partial^2 G_2}{\partial^2 y_2} = -2a \text{ and } \left(\frac{\partial^2 G_2}{\partial y_2 \partial \beta^*}\right)_Q = \frac{A}{\mu}.$$

Using theorem 4.1 of [Chavez and Song (2004)], we obtain the bifurcation constants B_1 and B_2 as

$$B_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 G_k}{\partial y_i \partial y_j}\right)_Q = u_2(2w_1 w_2 \beta^*) + u_2 w_2^2(-2a) = -2\left(\frac{{\beta^*}^2 A}{\mu^2} + a\right) < 0,$$

and

$$B_2 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 G_k}{\partial y_i \partial \beta^*}\right)_Q = u_2\left(w_2 \frac{A}{\mu}\right) = \frac{A}{\mu} > 0.$$

Thus, from theorem 4.1(iv) of [Chavez and Song (2004)], we state the following theorem:

Theorem 6.10: DFE $Q\left(\frac{A}{\mu}, 0\right)$ changes its behavior from stable to unstable at $R_0 = 1$ and there exists a positive equilibrium as R_0 crosses one. Hence, the system (6.25) undergoes a forward transcritical bifurcation at $R_0 = 1$.

6.6.4. Existence and stability analysis of endemic equilibrium

To find the condition for the existence of the endemic equilibrium $Q^*(S^*, I^*)$, the system (6.3) is rearranged to get S^*, and I^* which gives

$$S^* = \frac{\left((\mu + d + \delta)(1 + bI^*) + aI^{*2}\right)\left(1 + \alpha I^{*2}\right)}{\beta\left(1 + bI^{*2}\right)},$$

and I^* is given by the equation

$$K_1 I^{*4} + K_2 I^{*3} + K_3 I^{*2} + K_4 I^* + K_5 = 0, \tag{6.26}$$

where

$K_1 = \mu\alpha b(\mu + d + \delta)$,

$K_2 = b\beta(\mu + d + \delta) - a\mu\alpha$,

$K_3 = \mu(\alpha + b)(\mu + d + \delta) - (a + A)\beta$,

$K_4 = \beta(\mu + d + \delta) - a\mu$,

$K_5 = \mu(\mu + d + \delta) - \beta A = \mu(\mu + d + \delta)(1 - R_0)$.

Using Descartes' rule of signs, the existence of a unique positive real root I^* of the biquadratic Eq. (6.26) is required to satisfy any of the following conditions:

i. $K_1 > 0, K_2 > 0, K_3 > 0, K_4 > 0$, and $K_5 < 0$.

ii. $K_1 > 0, K_2 > 0, K_3 > 0, K_4 < 0$, and $K_5 < 0$.

iii. $K_1 > 0, K_2 > 0, K_3 < 0, K_4 < 0$, and $K_5 < 0$.

iv. $K_1 > 0, K_2 < 0, K_3 < 0, K_4 < 0$, and $K_5 < 0$.

Once we get the value of I^*, we can obtain the value of S^* as well. Thus, it implies that the system (6.3) admits a unique endemic equilibrium $Q^*(S^*, I^*)$ if one of the above conditions holds true.

To investigate the local stability of endemic equilibrium Q^*, we linearize this system (6.3) at Q^* and obtain the characteristic equation as given below:

$$\lambda^2 + M_0\lambda + N_0 + (M_1\lambda + N_1)\mathrm{e}^{-\lambda\tau} = 0, \tag{6.27}$$

where

$M_0 = (2\mu + d + \delta) + \frac{2a}{(1+bI^{*2})^2} + \frac{\beta I^*}{(1+\alpha I^{*2})}$,

$N_0 = \left(\mu + \frac{\beta I^*}{(1+\alpha I^{*2})}\right)\left((\mu + d + \delta) + \frac{2a}{\left(1+bI^{*2}\right)^2}\right)$,

$M_1 = -\frac{\beta S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2}$,

$N_1 = -\frac{\beta\mu S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2}$.

Theorem 6.11: For $\tau = 0$, endemic equilibrium Q^* of the system (6.3) is locally asymptotically stable if $M_0 + M_1 > 0$ and $N_0 + N_1 > 0$ hold true simultaneously.

Proof: At endemic equilibrium Q^*, the characteristic equation of the system for $\tau = 0$ is given by putting $\tau = 0$ in Eq. (6.27) as given below:

$$\lambda^2 + M_0\lambda + N_0 + (M_1\lambda + N_1) = 0.$$

$$\Rightarrow \quad \lambda^2 + (M_0 + M_1)\lambda + (N_0 + N_1) = 0. \tag{6.28}$$

Clearly, if $M_0 + M_1 > 0$ and $N_0 + N_1 > 0$ are satisfied simultaneously then by Routh – Hurwitz Criterion, Eq. (6.28) will always has roots with the negative real part and hence the system (6.3) at Q^* for $\tau = 0$ is locally asymptotically stable. This completes the proof.

Theorem 6.12: For $\tau > 0$, endemic equilibrium Q^* of the system (6.3) is locally asymptotically stable if ${M_0}^2 - 2N_0 - {M_1}^2 > 0$ and ${N_0}^2 - {N_1}^2 > 0$ are satisfied simultaneously.

Proof: At endemic equilibrium Q^*, the characteristic equation of the system for $\tau > 0$ is given by the Eq. (6.27)

$$\lambda^2 + M_0\lambda + N_0 + (M_1\lambda + N_1)\mathrm{e}^{-\lambda\tau} = 0.$$

For $\tau > 0$, corollary 2.4 of Ruan and Wei [2003] ensures that if the endemic equilibrium Q^* is unstable for a particular value of the delay parameter, then the roots of the characteristic equation (6.27) must intersect the imaginary axis. Thus, to prove the stability of the system (6.3), we will use the contradictory assumption i. e. we assume that $\lambda = \mathrm{i}\omega, \omega > 0$ is a root of the equation (6.27). On substituting $\lambda = \mathrm{i}\omega$ in Eq. (6.27):

$$-\omega^2 + N_0 + M_1\omega\ \sin(\omega\tau) + N_1\cos(\omega\tau) + \mathrm{i}\,(M_1\omega\ \cos(\omega\tau) - N_1\ \sin(\omega\tau) + M_0\omega) = 0. \tag{6.29}$$

By using Euler's formula and by separating the real and imaginary part of Eq. (6.29), we get

$$M_1\omega\ \sin(\omega\tau) + N_1\ \cos(\omega\tau) = \omega^2 - N_0, \tag{6.30}$$

$$M_1\omega\cos(\omega\tau) - N_1\ \sin(\omega\tau) = -M_0\omega. \tag{6.31}$$

On squaring and adding both the sides of the Eqs. (6.30) and (6.31) yield

$$\omega^4 + \left({M_0}^2 - 2N_0 - {M_1}^2\right)\omega^2 + \left({N_0}^2 - {N_1}^2\right) = 0. \tag{6.32}$$

Let $\omega^2 = Z_1$, Eq. (6.32) becomes

$$ {Z_1}^2 + MZ_1 + T = 0. \tag{6.33}$$

Here, $M = \left({M_0}^2 - 2N_0 - {M_1}^2\right)$ and $T = \left({N_0}^2 - {N_1}^2\right)$.

Clearly, if $M = \left({M_0}^2 - 2N_0 - {M_1}^2\right) > 0$ and $T = \left({N_0}^2 - {N_1}^2\right) > 0$ are satisfied simultaneously then by Routh-Hurwitz Criterion Eq. (6.33) will always has roots with the negative real part. It contradicts our assumption for instability that $\lambda = \mathrm{i}\omega$ is a root of Eq. (6.27). Hence, the endemic equilibrium Q^* of the system (6.3) is locally asymptotically stable for $\tau > 0$. It completes the proof.

6.6.5. Hopf bifurcation analysis

The Hopf bifurcation analysis is similar to section 6.5.5. Hence, we omitted the proof of the following result:

Theorem 6.13: The endemic equilibrium (EE) of the system (6.3) is asymptotically stable for $\tau \in [0, \tau_0)$ and it undergoes a Hopf bifurcation at $\tau = \tau_0$.

6.6.6. Global stability analysis

In this section, we discuss the global stability analysis of equilibria. For this, we state the results in the form of theorems and prove them.

We see that $Y\big(S(t), I(t)\big) = \frac{\beta S(t) I(t)}{1+\alpha I^2(t)}$ and $T_2\big(I(t)\big) = \frac{aI^2(t)}{1+bI^2(t)}$ are always positive, continuously differentiable and monotonically increasing for all $S > 0$ and $I > 0$. That is, they satisfy the following conditions:

A1. $Y\big(S(t),I(t)\big)>0, Y'_S(S,I)>0$, for $S>0$ and $I>0$ and $Y'_I(S,I)>0$ for $I<1/\sqrt{\alpha}$.

A2. $Y(S,0)=Y(0,I)=0, Y'_S(S,0)=0, Y'_I(S,0)>0$ for $S>0$ and $I>0$.

A3. $T_2(0)=0, T'_2(I)>0$ for $I>0$.

These properties will be used to prove the global stability of equilibria.

6.6.6.1. Global stability of disease-free equilibrium (DFE)

In this subsection, we show the global stability of the DFE of the system (6.3). For this, we suppose the following condition:

A4. $\varphi(S,I)=\frac{Y(S,I)}{I}$ is a bounded and monotonic decreasing function of $I>0$, for any fixed $S\geq 0$, and $K(S)=\lim_{I\to+0}\varphi(S,I)$ is continuous on $S\geq 0$ and a monotone increasing function of $S\geq 0$.

For the global stability of DFE, we prove the following theorem:

Theorem 6.14: DFE $Q\left(\frac{A}{\mu},0\right)$ of the system (6.3) is globally asymptotically stable if and only if $R_0\leq 1$.

Proof: To prove this theorem, we consider the following Lyapunov function:

$$W(t)=W_1(t)+W_2(t)+I(t),$$

where

$$W_1(t)=\int_{S_0=\frac{A}{\mu}}^{S(t)}\left(1-\frac{K(S_0)}{K(S)}\right)\mathrm{d}s \text{ and } W_2(t)=\int_{t-\tau}^{t}Y\big(s(u+\tau),I(u)\big)\frac{K(S_0)}{K(S(u+\tau))}\mathrm{d}u\ .$$

The derivative of $W_1(t)$ is

$$\frac{\mathrm{d}W_1(t)}{\mathrm{d}t}=\left(1-\frac{K(S_0)}{K(S(t))}\right)\left(A-\mu S(t)-Y(S(t),I(t-\tau))\right)$$

$$=-\left(1-\frac{K(S_0)}{K(S(t))}\right)Y\big(S(t),I(t-\tau)\big)-\mu(S(t)-S_0)\left(1-\frac{K(S_0)}{K(S(t))}\right).$$

The derivative of $W_2(t)$ is

$$\frac{\mathrm{d}W_2(t)}{\mathrm{d}t} = Y\big(S(t+\tau), I(t)\big)\left(\frac{K(S_0)}{K\big(S(t+\tau)\big)}\right) - Y\big(S(t), I(t-\tau)\big)\frac{K(S_0)}{K\big(S(t)\big)}.$$

Hence, we obtain

$$\begin{aligned}\frac{\mathrm{d}W(t)}{\mathrm{d}t} &= -\left(1-\frac{K(S_0)}{K\big(S(t)\big)}\right)Y\big(S(t), I(t-\tau)\big) - \mu(S(t)-S_0)\left(1-\frac{K(S_0)}{K\big(S(t)\big)}\right)\\ &\quad + Y\big(S(t+\tau), I(t)\big)\left(\frac{K(S_0)}{K\big(S(t+\tau)\big)}\right) - Y\big(S(t), I(t-\tau)\big)\frac{K(S_0)}{K\big(S(t)\big)}\\ &\quad + Y\big(S(t), I(t-\tau)\big) - (\mu+d+\delta)I(t) - \frac{aI^2(t)}{1+bI^2(t)}\\ &= -\mu(S(t)-S_0)\left(1-\frac{K(S_0)}{K\big(S(t)\big)}\right) + Y\big(S(t+\tau), I(t)\big)\left(\frac{K(S_0)}{K\big(S(t+\tau)\big)}\right) - \Big(\mu+d+\delta+\\ &\quad \frac{aI}{1+bI^2}\Big)I(t).\end{aligned}$$

Here, by the conditions (A1-A2), we obtain that

$$-\mu(S(t)-S_0)\left(1-\frac{K(S_0)}{K\big(S(t)\big)}\right) \le 0,$$

with equality if and only if $S(t) = S_0$. From the condition (A4), it follows that

$$\begin{aligned}&Y\big(S(t+\tau), I(t)\big)\left(\frac{K(S_0)}{K\big(S(t+\tau)\big)}\right) - \left(\mu+d+\delta+\frac{aI}{1+bI^2}\right)I(t)\\ &\le \left(\frac{Y\big(S(t+\tau),I(t)\big)}{(\mu+d+\delta)I(t)}\left(\frac{K(S_0)}{K\big(S(t+\tau)\big)}\right) - 1\right)(\mu+d+\delta)I(t)\\ &\le \left(\frac{K\big(S(t+\tau)\big)}{(\mu+d+\delta)} \times \frac{K(S_0)}{K\big(S(t+\tau)\big)} - 1\right)(\mu+d+\delta)I(t)\\ &= \left(\frac{K(S_0)}{\big(\mu+d+\delta+T_2'(0)\big)} - 1\right)(\mu+d+\delta)I(t)\\ &= (R_0-1)(\mu+d+\delta)I(t).\end{aligned}$$

Therefore, $R_0 \le 1$ ensures that $\frac{\mathrm{d}W(t)}{\mathrm{d}t} \le 0$ for all $t > 0$, where $\frac{\mathrm{d}W(t)}{\mathrm{d}t} = 0$ holds if $S(t) = S_0$. Hence, it immediately follows from the system (6.3) that DFE Q is the largest invariant set in $\{(S(t), I(t)) \in \mathbb{R}^2_{+0} \,\big|\, \frac{\mathrm{d}W(t)}{\mathrm{d}t} = 0\}$. From the Lyapunov-LaSalle asymptotic stability theorem [Hale and Lunel (1993)], we obtain that DFE Q is globally asymptotically stable. This completes the proof.

6.6.6.2. Global stability of endemic equilibrium (EE)

In this subsection, we discuss the global stability of endemic equilibrium $Q^*(S^*, I^*)$ of the system (6.3) using the Lyapunov direct method. For this, we propose the following hypotheses:

A5. $\frac{I(t)}{I^*} \leq \frac{Y(S(t),I(t-\tau))}{Y(S(t),I^*)}$ for $I \in (0, I^*)$, $\frac{Y(S(t),I(t-\tau))}{Y(S(t),I^*)} \leq \frac{I(t)}{I^*}$ for $I \geq I^*$.

A6. $\frac{T_2(I(t))}{T_2(I^*)} \leq \frac{I(t)}{I^*}$ for $I \in (0, I^*)$, $\frac{T_2(I(t))}{T_2(I^*)} \geq \frac{I(t)}{I^*}$ for $I \geq I^*$ and $I^* < \frac{1}{\sqrt{b}}$.

Theorem 6.15: Suppose that conditions (A1) - (A3) and (A5) - (A6) are satisfied. Then the endemic equilibrium $Q^*(S^*, I^*)$ of the system (6.3) is globally asymptotically stable if $R_0 > 1$.

Proof: We consider the following Lyapunov functional

$$X(t) = X_1(t) + X_2(t),$$

where

$$X_1(t) = S(t) - S^* - \int_{S^*}^{S(t)} \frac{Y(S^*,I^*)}{Y(u,I^*)} \mathrm{d}u + I(t) - I^* - I^* \log_{\mathrm{e}} \frac{I(t)}{I^*} - \int_{t-\tau}^{t} Y\big(S(u+\tau), I(u)\big) \mathrm{d}u,$$

$$X_2(t) = Y(S^*, I^*) \int_{t-\tau}^{t} \left(\frac{Y\big(S(u+\tau),I(u)\big)}{Y(S^*,I^*)} - 1 - \log_{\mathrm{e}} \frac{Y\big(S(u+\tau),I(u)\big)}{Y(S^*,I^*)} \right) \mathrm{d}u.$$

$X(t) = X_1(t) + X_2(t)$ is defined and continuously differentiable for all $S(t), I(t) > 0$. and $X(0) = 0$ at $Q^*(S^*, I^*)$. At $Q^*(S^*, I^*)$,

$A - \mu S^* = Y(S^*, I^*),\ Y(S^*, I^*) = (\mu + d + \delta)I^* + T_2(I^*)$.

The time derivative of $X_1(t)$ along the solution of system (6.3) is given by

$$\begin{aligned}
\frac{\mathrm{d}X_1(t)}{\mathrm{d}t} &= S'(t) - \frac{Y(S^*,I^*)}{Y(S(t),I^*)} S'(t) + I'(t) - \frac{I^*}{I(t)} I'(t) - Y(S(t+\tau), I(t)) + Y(S(t), I(t-\tau)) \\
&= \left(1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)}\right) (\mu S^* - \mu S(t) + Y(S^*, I^*) - Y((S(t), I(t-\tau))) \\
&\quad + \left(1 - \frac{I^*}{I(t)}\right) \Big(Y\big(S(t), I(t-\tau)\big) - Y(S^*, I^*) \frac{I(t)}{I^*} + T_2(I^*) \frac{I(t)}{I^*} - T_2\big(I(t)\big) \Big) \\
&\quad - Y(S(t+\tau), I(t)) + Y(S(t), I(t-\tau)) \\
&= \mu S^* \left(1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)}\right) \left(1 - \frac{S(t)}{S^*}\right) + Y(S^*, I^*) \left(1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)} + \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\right) \\
&\quad + Y(S^*, I^*) \left(1 - \frac{I(t)}{I^*} - \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)} \frac{I^*}{I(t)}\right) + T_2(I^*) \left(\frac{I(t)}{I^*} - 1 - \frac{T_2(I(t))}{T_2(I^*)} + \frac{T_2(I(t))}{T_2(I^*)} \frac{I^*}{I(t)}\right) \\
&\quad - Y(S(t+\tau), I(t)) + Y(S(t), I(t-\tau)).
\end{aligned}$$

Further,

$$\frac{\mathrm{d}X_2(t)}{\mathrm{d}t} = Y(S^*, I^*)\left(\frac{Y\big(S(t+\tau),I(t)\big)}{Y(S^*,I^*)} - 1 - \log_{\mathrm{e}}\frac{Y\big(S(t+\tau),I(t)\big)}{Y(S^*,I^*)} - \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S^*,I^*)} + 1 + \log_{\mathrm{e}}\frac{Y\big(S(t),I(t-\tau)\big)}{Y(S^*,I^*)}\right)$$

$$= Y\big(S(t+\tau),I(t)\big) - Y\big(S(t),I(t-\tau)\big) + Y(S^*,I^*)\log_{\mathrm{e}}\frac{Y\big(S(t),I(t-\tau)\big)}{Y\big(S(t+\tau),I(t)\big)}.$$

Then we have,

$$\frac{\mathrm{d}X(t)}{\mathrm{d}t} = \mu S^*\left(1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)}\right)\left(1 - \frac{S(t)}{S^*}\right) + Y(S^*,I^*)\left(1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)} + \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\right)$$
$$+ Y(S^*,I^*)\left(1 - \frac{I(t)}{I^*} - \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\frac{I^*}{I(t)}\right) + T_2(I^*)\left(\frac{I(t)}{I^*} - 1 - \frac{T_2\big(I(t)\big)}{T_2(I^*)} + \frac{T_2\big(I(t)\big)}{T_2(I^*)}\frac{I^*}{I(t)}\right)$$
$$- Y\big(S(t+\tau),I(t)\big) + Y\big(S(t),I(t-\tau)\big) + Y\big(S(t+\tau),I(t)\big)$$
$$- Y\big(S(t),I(t-\tau)\big) + Y(S^*,I^*)\log_{\mathrm{e}}\frac{Y\big(S(t),I(t-\tau)\big)}{Y\big(S(t+\tau),I(t)\big)}$$
$$= \mu S^*\left(1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)}\right)\left(1 - \frac{S(t)}{S^*}\right) + Y(S^*,I^*)\left(1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)} + \log_{\mathrm{e}}\frac{Y(S^*,I^*)}{Y(S(t),I^*)}\right)$$
$$+ Y(S^*,I^*)\left(1 - \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\frac{I^*}{I(t)} + \log_{\mathrm{e}}\frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\frac{I^*}{I(t)}\right)$$
$$+ Y(S^*,I^*)\left(1 - \frac{I(t)}{I^*}\frac{Y(S(t),I^*)}{Y\big(S(t),I(t-\tau)\big)} + \log_{\mathrm{e}}\frac{I(t)}{I^*}\frac{Y(S(t),I^*)}{Y\big(S(t),I(t-\tau)\big)}\right)$$
$$+ Y(S^*,I^*)\left(\frac{I(t)}{I^*} - \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\right)\left(\frac{Y(S(t),I^*)}{Y\big(S(t),I(t-\tau)\big)} - 1\right) + T_2(I^*)\left(\frac{\big(T_2(I(t)\big)}{T_2(I^*)} - \frac{I(t)}{I^*}\right)\left(\frac{I^*}{I(t)} - 1\right).$$

The function $Y(S,I)$ is monotonically increasing for any $S > 0$; hence the following inequality holds:

$$\left(1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)}\right)\left(1 - \frac{S(t)}{S^*}\right) \le 0. \tag{6.34}$$

and by the properties of the function $r(x) = 1 - x + \log_{\mathrm{e}} x$, $(x > 0)$, we note that $r(x)$ has its global maximum $r(1) = 0$. Hence $r(x) \le 0$ when $x > 0$ and the following inequalities hold true:

$$1 - \frac{Y(S^*,I^*)}{Y(S(t),I^*)} + \log_{\mathrm{e}}\frac{Y(S^*,I^*)}{Y(S(t),I^*)} \le 0,\ 1 - \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\frac{I^*}{I(t)} + \log_{\mathrm{e}}\frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\frac{I^*}{I(t)} \le 0, \text{ and}$$
$$1 - \frac{I(t)}{I^*}\frac{Y(S(t),I^*)}{Y\big(S(t),I(t-\tau)\big)} + \log_{\mathrm{e}}\frac{I(t)}{I^*}\frac{Y(S(t),I^*)}{Y\big(S(t),I(t-\tau)\big)} \le 0. \tag{6.35}$$

Further by conditions (A5) - (A6) the following inequalities hold:

$$\left(\frac{I(t)}{I^*} - \frac{Y\big(S(t),I(t-\tau)\big)}{Y(S(t),I^*)}\right)\left(\frac{Y(S(t),I^*)}{Y\big(S(t),I(t-\tau)\big)} - 1\right) \le 0, \left(\frac{T_2\big(I(t)\big)}{T_2(I^*)} - \frac{I(t)}{I^*}\right)\left(\frac{I^*}{I(t)} - 1\right) \le 0. \tag{6.36}$$

By inequalities (6.34) – (6.36), we see that $\frac{\mathrm{d}X(t)}{\mathrm{d}t} \leq 0$ for all $S(t) \geq 0, I(t) \geq 0$. It is easy to verify that the largest invariant set in $\{(S(t), I(t)) \left| \frac{dX(t)}{dt} = 0 \right.\}$ is the singleton $\{Q^*\}$. By the Lyapunov-LaSalle asymptotic stability theorem [Hale and Lunel (1993)], endemic equilibrium Q^* is globally asymptotically stable.

6.7. Numerical simulations

In this section, we elaborate the results obtained by the simulation of the model.

6.7.1 Numerical simulation of the model for the combination of M-H type incidence and Holling type II treatment rates

For numerical computation, we take the following numerically experimental values of the parameters:

$$A = 12, \alpha = 0.001, \beta = 0.005, \mu = 0.05, d = 0.01, \delta = 0.002, a = 0.02, b = 0.002\,.$$

Fig. 6.3 shows the variations in the susceptible and infected population at time lag $\tau = 0\,,1$. The figure depicts that as time passes both types of population approaches to steady state. It is also seen that as delay occurs, the infected population increases and susceptible population decreases.

Fig. 6.4 shows variation in the infected population at several values of time lag τ. It can be observed from the figure that the infected population is increasing as the value of time lag increases. Hence, whenever the delays occur in effective incidence between susceptible and infected individuals, the number of infected individuals always increases.

Fig. 6.5 depicts variation in the infected population at time lag $\tau = 1$ for numerous values of the transmission rate (β). It can be seen from the figure that the increase in transmission rate results in the increase of the infected population.

Fig. 6.6 demonstrates the variation in the infected population at the numerous values of the inhibitory effect (α). It is easily observed that as the value of α increases, the infected population decreases.

Fig. 6.7 shows variation in the infected population at time lag $\tau = 1$ for different values of cure rate (a). It is evident that, as the value of the cure rate (a) is increasing, the infected population is decreasing. Hence, the cure rate is playing a vital role in controlling the spread of the epidemic.

Fig. 6.8 illustrates the variations in the infected population at numerous values of limitation rate (b) in treatment availability. It can be viewed from the figure that the infected population is increasing as the value of b is also increasing.

Fig. 6.9 delineates the difference in the infected population with and without the Holling type II treatment rate. It can be interpreted that the infected population with Holling type II treatment is less in comparison to the infected population without Holling type II treatment. Hence, the Holling type II treatment rate may be proved as a better option in minimizing the loss of lives and wealth of society.

Fig. 6.10 shows the variation in the infected population for a different set of values of inhibitory effect and treatment rate. It can be interpreted from the figure that with our novel combination of Monod-Haldane incidence and Holling type II treatment rates (in the presence of inhibitory effect and H-II treatment rate); the infected individuals are less in comparison of rest.

Fig. 6.11 shows the oscillatory behavior of the infected population. For this, we take the following numerical experimental values of the parameters:

$$\mathrm{A} = \mathbf{5}, \alpha = 1.2, \beta = 0.06, \mu = 0.05, d = 0.001, \delta = 0.002, a = 1.2, b = 0.82.$$

At this set of parameters values, the model approaches to the endemic equilibrium $Q^*(76.36, 3.031)$.

6.7.2 Numerical simulation of the model for the combination of M-H type incidence and Holling type III treatment rates

In this section, we will display the results of numerical simulation. All computations have been carried out with the following data.

$$A = 3, \mu = 0.05, \beta = 0.004, \alpha = 0.08, d = 0.001, \delta = 0.002, a = 0.02, b = 0.0004$$

Variation in the susceptible and infected population with respect to time delay by taking various values of time lag $\tau = 0, 2$ and 4 at different initial values have been shown in Figs. 6.12(a) & 6.13(a) and Figs. 6.12(b) & 6.13(b) respectively. Figs. 6.12(a) & 6.13(a) depict the decrement in susceptible population and Figs. 6.12(b) & 6.13(b) show the increment in the infected population as time lag τ increases. Thus, the higher the delay, the higher will be the occurrence of infection in the society, which is biologically to be expected.

The influence of transmission rate (β) and inhibitory effect (α) on the infected population has been interpreted in Figs. 6.14 and 6.15 respectively. Fig. 6.14 shows that the infected population is increasing with the increase in the transmission rate (β), while decrement in the infected population is observed with increasing value of inhibitory effects (α) (Fig. 6.15). Based on these figures, it can be concluded that inhibitions must be exercised to control the disease from the society. Further, Figs. 6.14 and 6.15 observe resemblance in nature and hence ensure the validation of the mathematical structure of the model.

Fig. 6.16 shows the impact of the cure rate on the infected population at different values of cure rate $a = 0.02, 0.04$ and 0.006 respectively. The diminution of the infected population with the increment in cure rate a can be seen from the graph and then, the infected population settles down to its steady state. Also, it is readily seen from the graph that when there are low treatment facilities available then infection is occurring at a higher rate.

Fig. 6.17 represents the variation in the population of infected individuals in the presence of inhibitory effect and Holling type III treatment rate, in the absence of inhibitory effect and in presence of Holling type III treatment rate, in presence of inhibitory effect and in the absence of Holling type III treatment rate, and in absence of inhibitory effect and Holling type III treatment rate respectively. This figure shows how the combination of Monod-Haldane incidence (presence of inhibitory effects) and Holling type III treatment rates help to control the spread of infectious disease effectively. From the graph, it can be seen that when infected individuals have been treated using Holling type III rate then the number of the infected individuals sharply decreases initially, and thereafter it begins to decrease gradually and reaches its steady state.

Fig.6.18 shows the infected population at increased values of limitation rate in treatment availability. It can be observed from the figure that higher the limitation in treatment availability, the higher will be the infection.

To illustrate the Hopf bifurcation numerically, the oscillatory and periodic behavior of the infected population has been drawn in Figs. 6.19, 6.20, and 6.21. For this, we take the following data:

$$A = 5\,, \mu = 0.05\,, \beta = 0.54\,, \alpha = 1.2, d = 0.001\,, \delta = 0.002\,, a = 0.1\,, b = 0.0387\,.$$

Figs. 6.19 and 6.20 show damped oscillations for the time delay $\tau = 9$ and $\tau = 11$ respectively, which mean the inherent dynamics contain a strong oscillatory component, but the amplitude of these fluctuations declines over time as the system equilibrates. It shows how the fraction of infectives oscillates with decreasing amplitude as it settles towards the equilibrium whereas Fig. 6.21 shows the periodic solution of the infected population with respect to the time for the time delay $\tau = 13.5$, which confirms the occurrence of Hopf bifurcation.

6.8 Conclusions

In this chapter, we proposed a time-delayed SIR epidemic model with a novel combination of Monod-Haldane (M-H) Incidence rate and two different treatment rates

(Holling type II & III treatment rates). We analyzed the model at equilibrium points. It has been found that the model has two equilibria: disease-free and endemic equilibria. We investigated the local stability of DFE by basic reproduction number R_0 and it is concluded that DFE is locally asymptotical stable when $R_0 < 1$ for $\tau \geq 0$ for both combination of incidence and treatment rates separately. We have also shown that DFE at $R_0 = 1$: (i) DFE is linearly neutrally stable for $\tau > 0$, (ii) exhibits the forward bifurcation for $\tau = 0$. Further, we also discussed the stability of EE and investigated that EE is locally asymptotically stable for time lag $\tau \geq 0$ under the conditions stated in Theorem 6.5, 6.6, 6.11 & 6.12 respectively for both the combinations of incidence and treatment rates. We showed that the model exhibits a Hopf bifurcation at endemic equilibrium under the conditions stated in theorem 6.7 & 6.13. Further, for the combination of M-H incidence rate and Holling type III treatment rate, we investigated that DFE is globally asymptotically stable when $R_0 \leq 1$ and EE is globally asymptotically stable when $R_0 > 1$ under the conditions (A1-A3), (A4) and (A5-A6) respectively. We also simulated the model numerically in the support of our theoretical findings and have drawn the graphs for time delay, transmission rate, measures of inhibition and treatment rate. From the graphs, we observed that the higher the delay, the higher will be the infection and also observed that Holling type II & III treatment rates may play a crucial role in controlling the infection. The effect of inhibitory measures and transmission rate of disease on the infected population has been shown and it is evident from the figures that the infected population is increasing with the increased value of transmission rate, while it is decreasing with the increasing values of inhibitory measures. This implies that the higher the inhibitory effects, the lesser will be the infection. With the help of figures, we also showed the oscillatory and periodic behavior of infection in the population. It shows the occurrence of Hopf bifurcation and also confirms the appearance of the periodic solution.

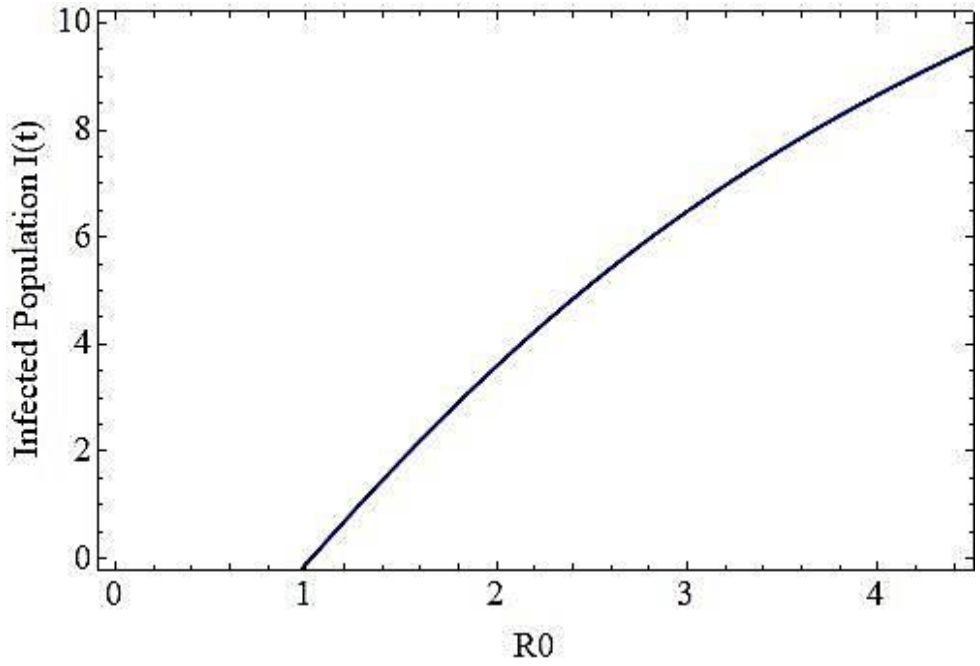


Fig 6.2: Forward bifurcation diagram for the data $A = 12, \alpha = 0.001, \beta = 0.0003, \mu = 0.05, d = 0.01, \delta = 0.002, a = 0.02, b = 0.002.$

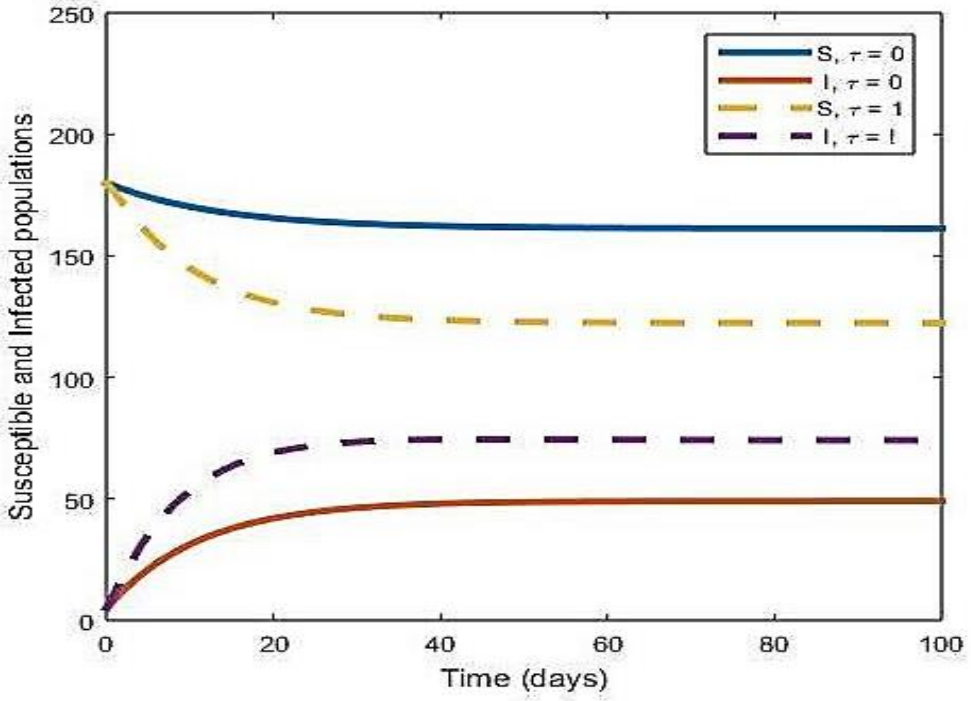


Fig. 6.3: Susceptible (S) and Infected (I) population at time lag $\tau = 0, 1$.

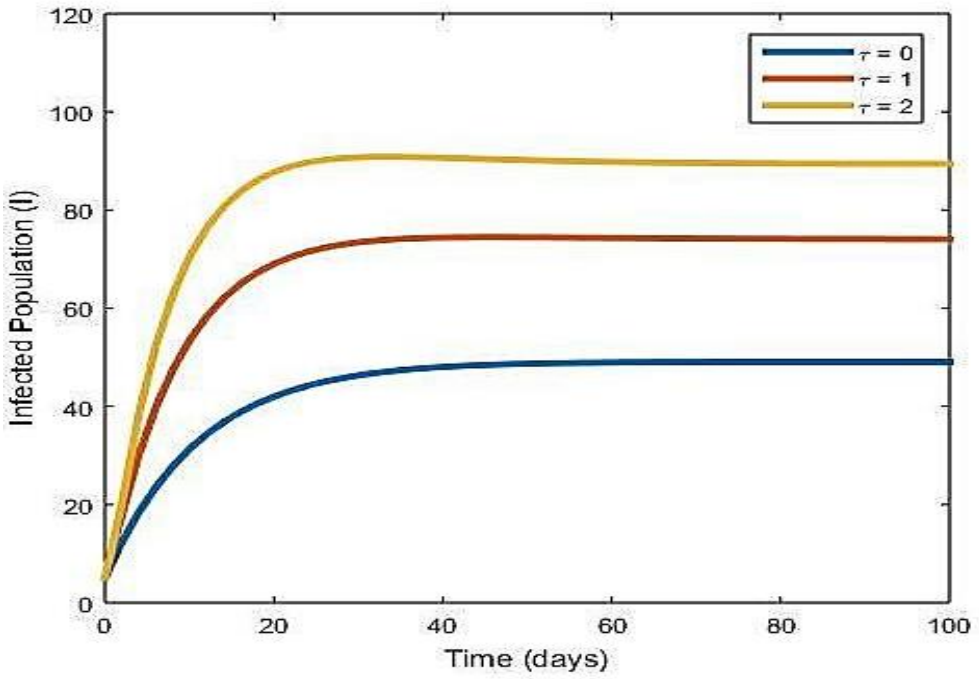


Fig. 6.4: Variation in the infected population (I) at various values of time lag τ.

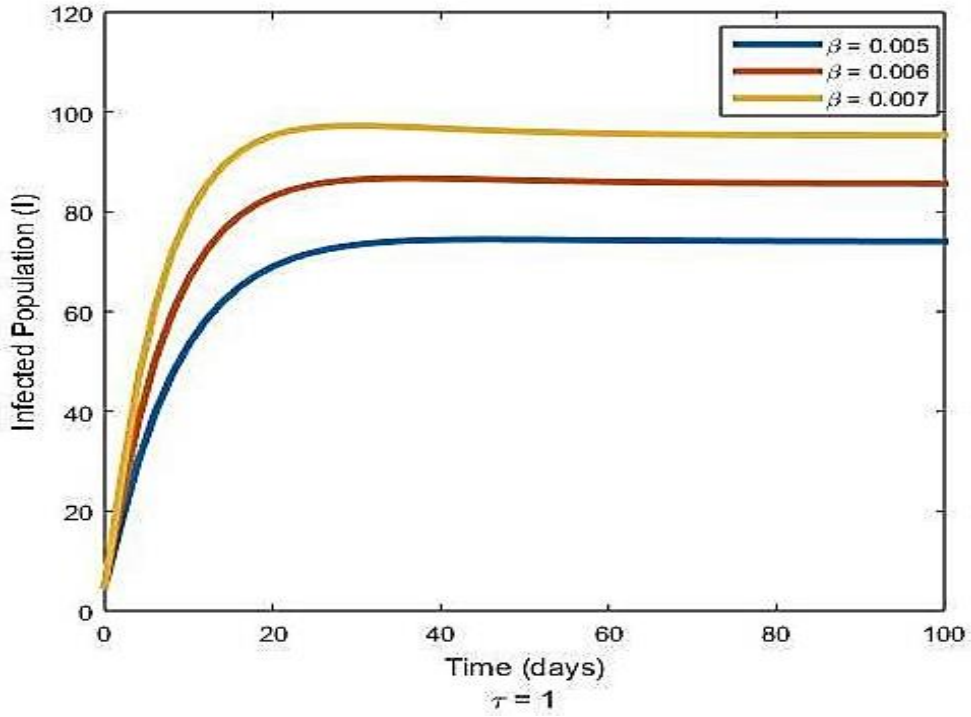


Fig. 6.5: Variation in the infected population (I) at various values of the transmission rate (β).

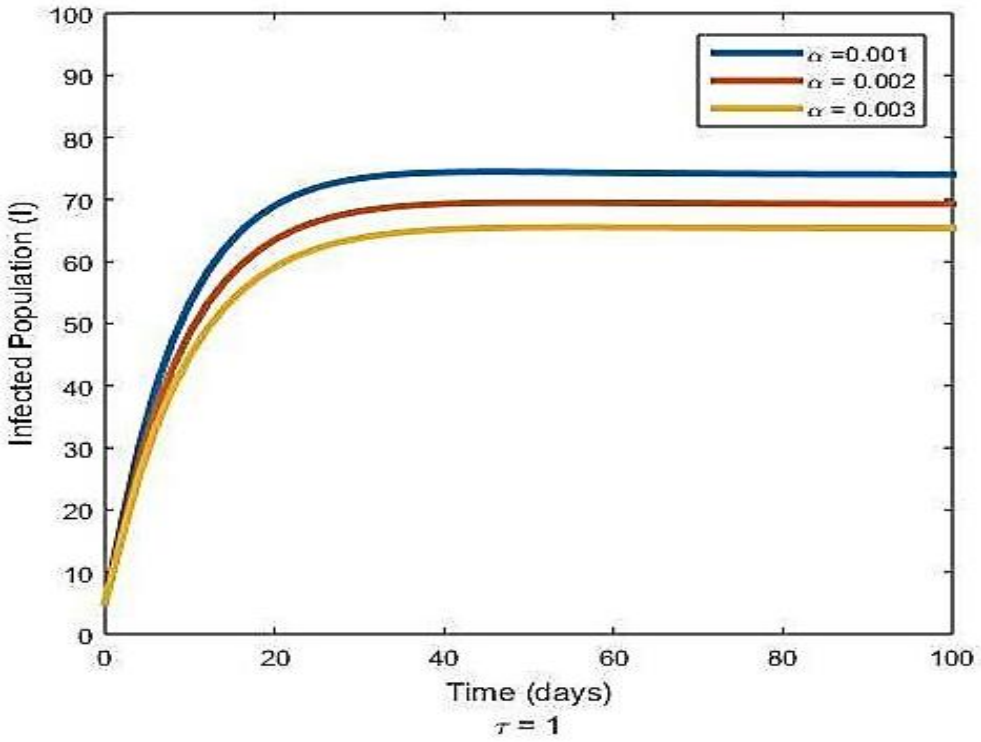


Fig. 6.6: Variation in the infected population (I) at various values of psychological effects/ inhibitory effects (α).

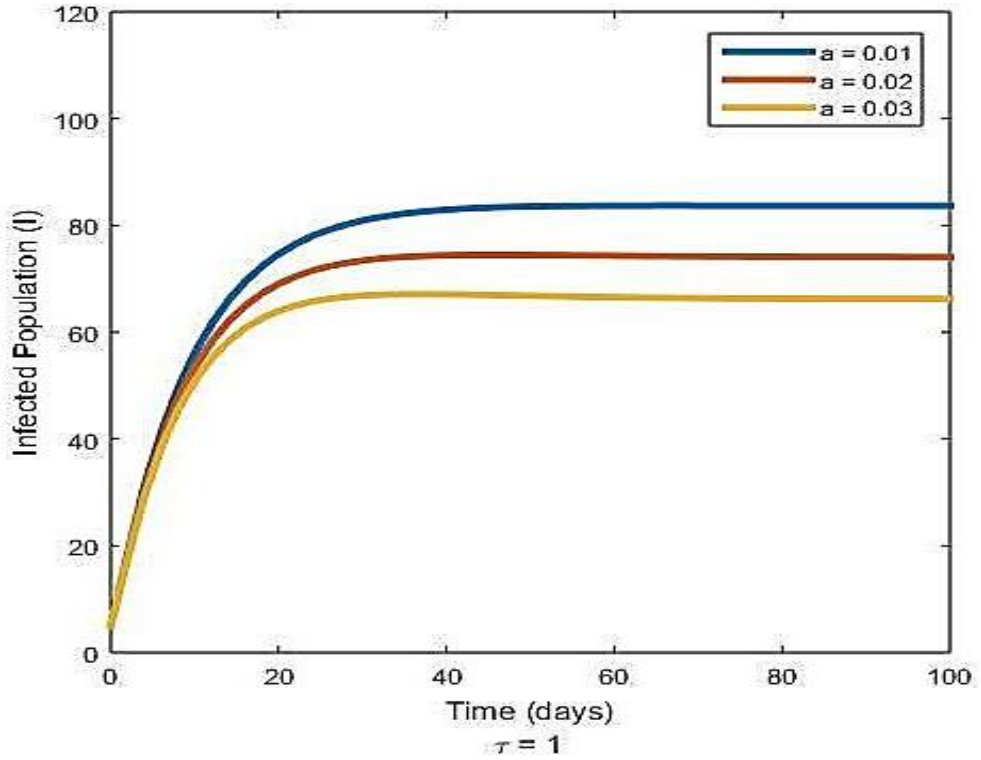


Fig. 6.7: Variation in the infected population (I) at various values of cure rate(a).

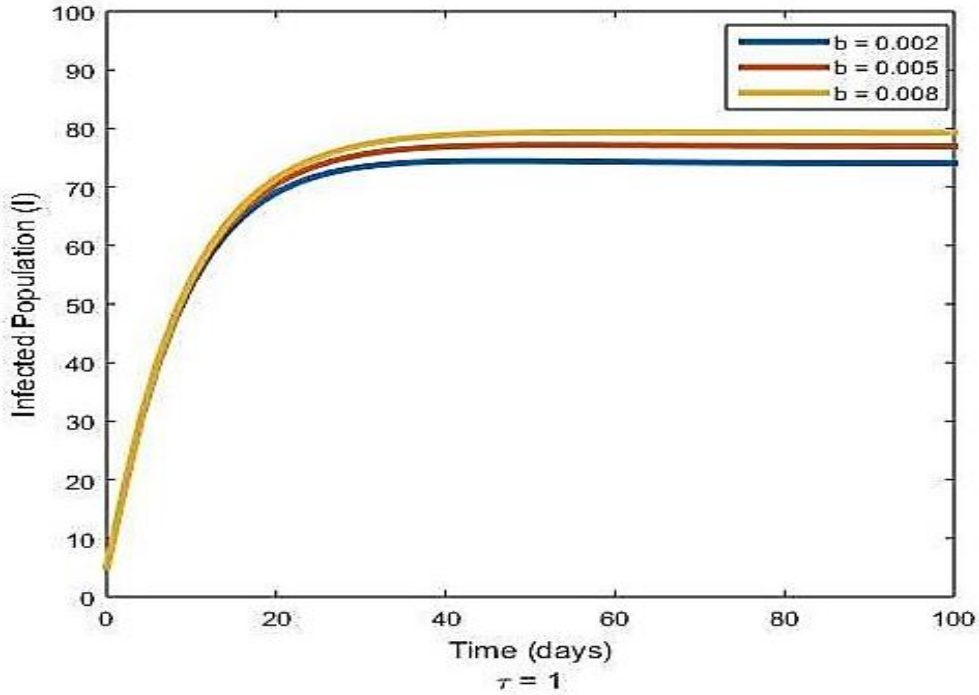


Fig. 6.8: Variation in the infected population (I) at various values of limitation rate (b) in treatment availability.

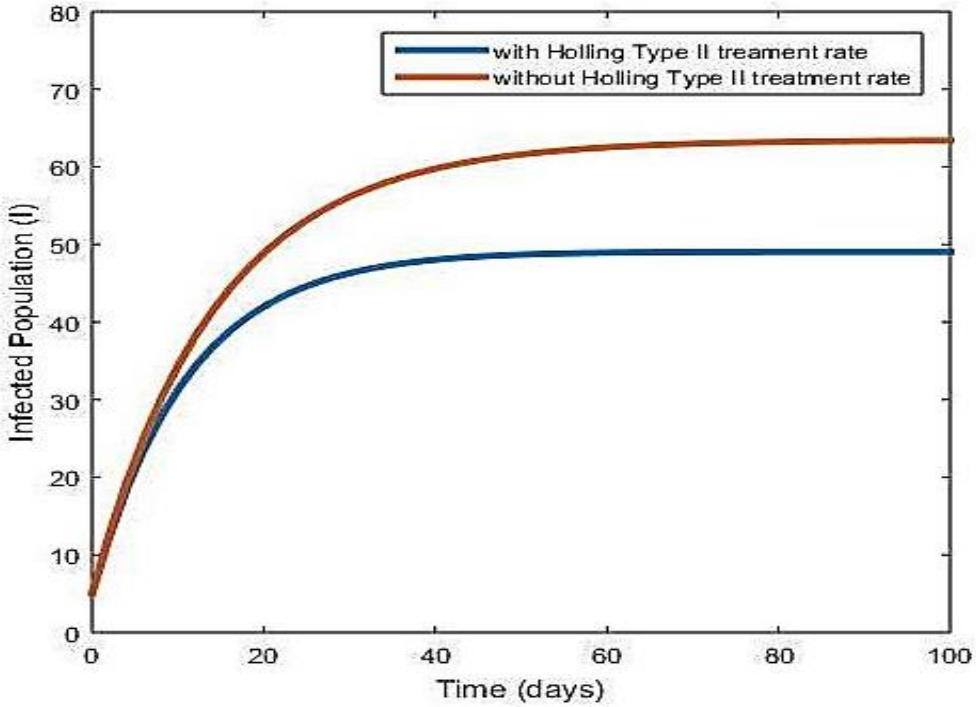


Fig. 6.9: Variation in the infected population (I) with and without Holling type II treatment rate with $a = 0.05$ and $b = 0.002$.

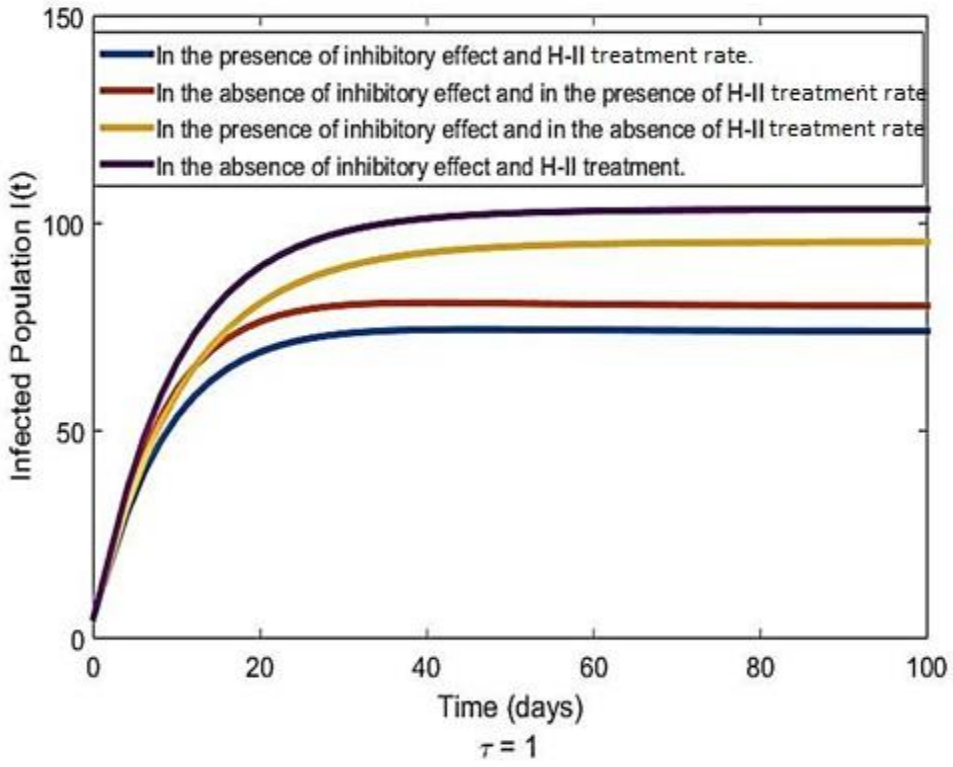


Fig. 6.10: Variation in the infected population (I) with the various combinations of inhibitory effect and treatment rate.

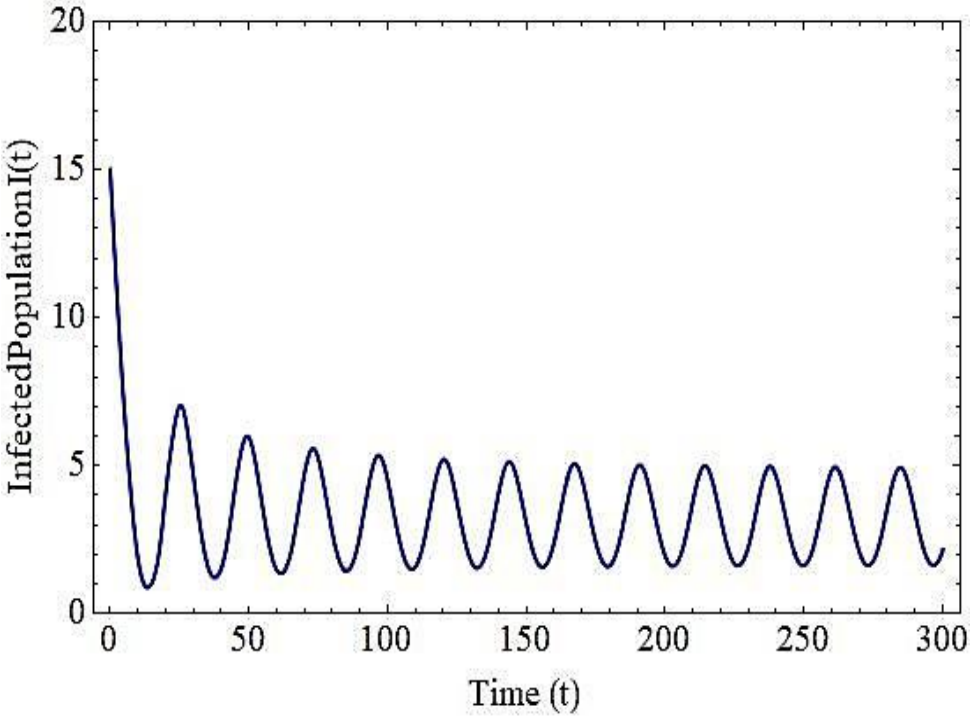


Fig. 6.11: Oscillatory behavior of the infected population (I) at time lag $\tau = 8$.

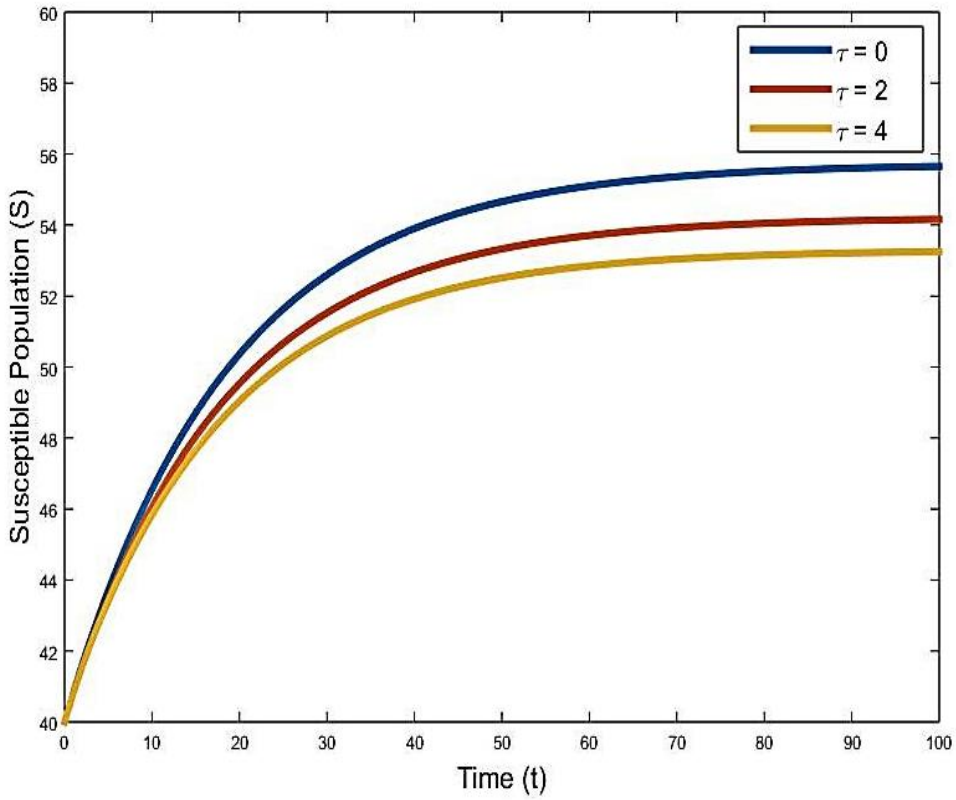


Fig. 6.12(a): Variation in the susceptible population (S) at various values of time lag τ.

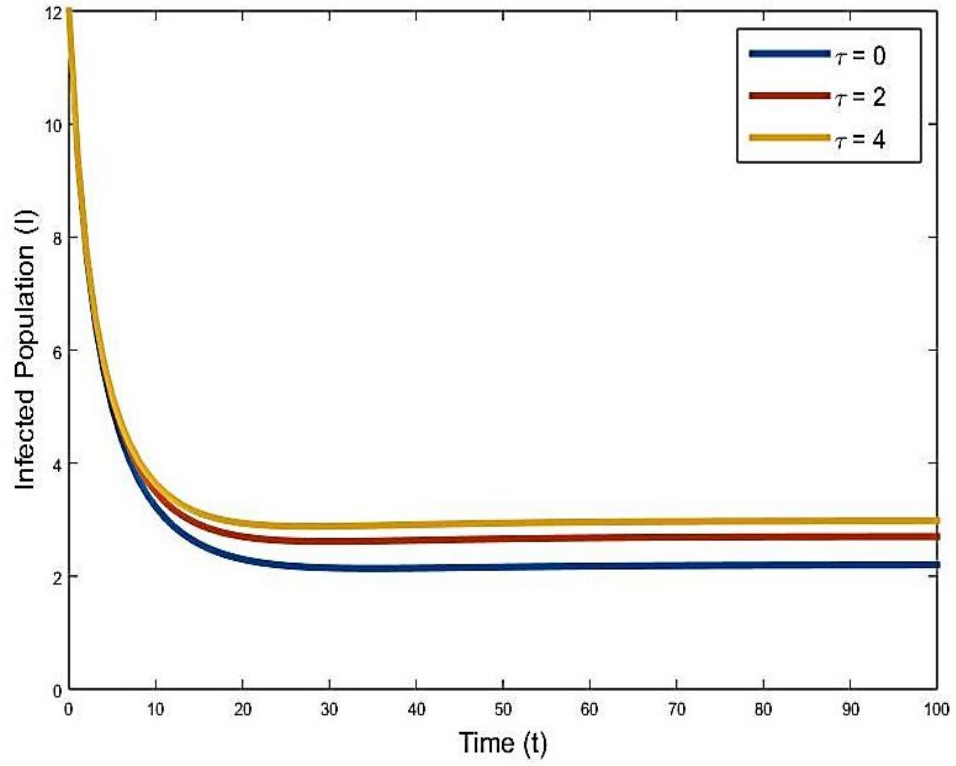


Fig. 6.12(b): Variation in the infected population (I) at various values of time lag τ.

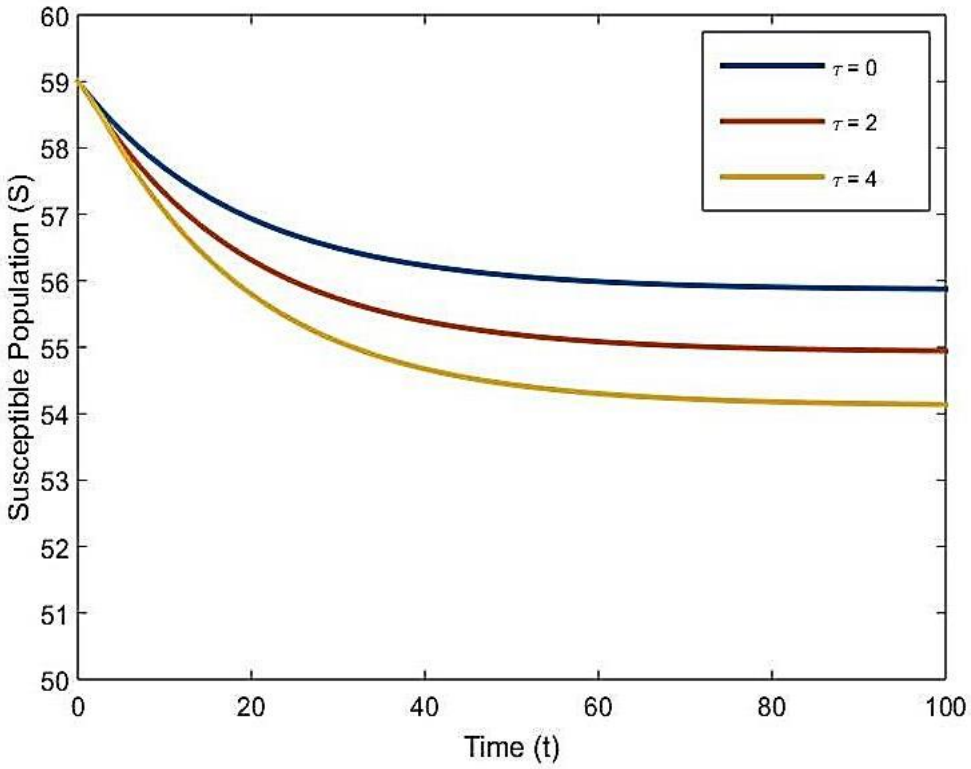


Fig. 6.13(a): Variation in the susceptible population (S) at various values of time lag τ.

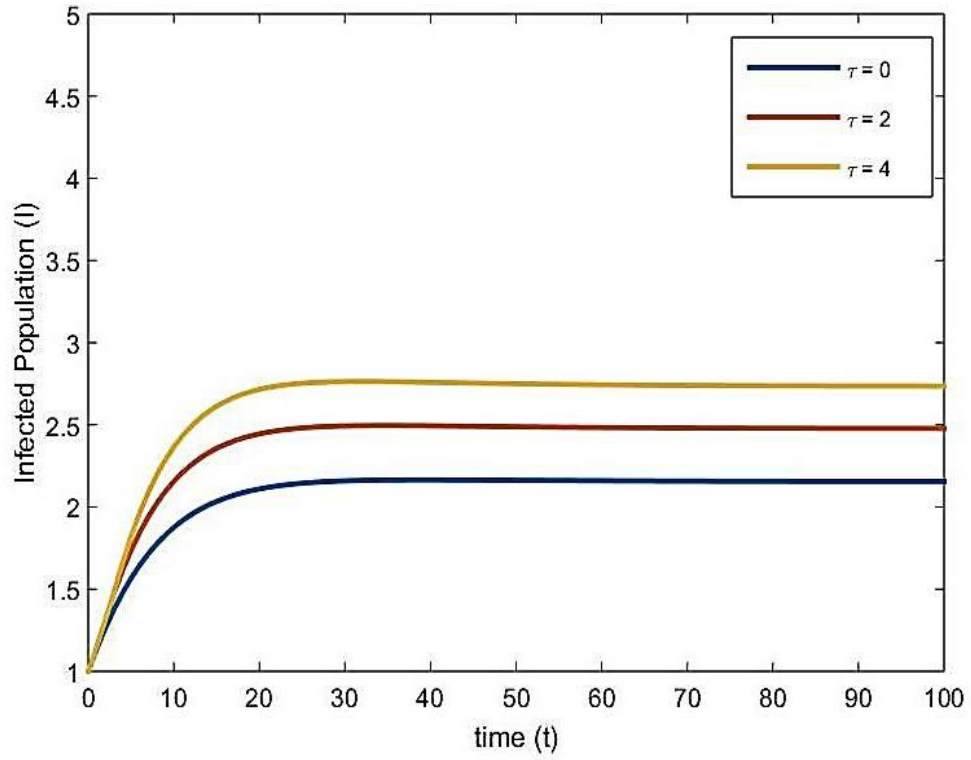


Fig. 6.13(b): Variation in the infected population (I) at various values of time lag τ.

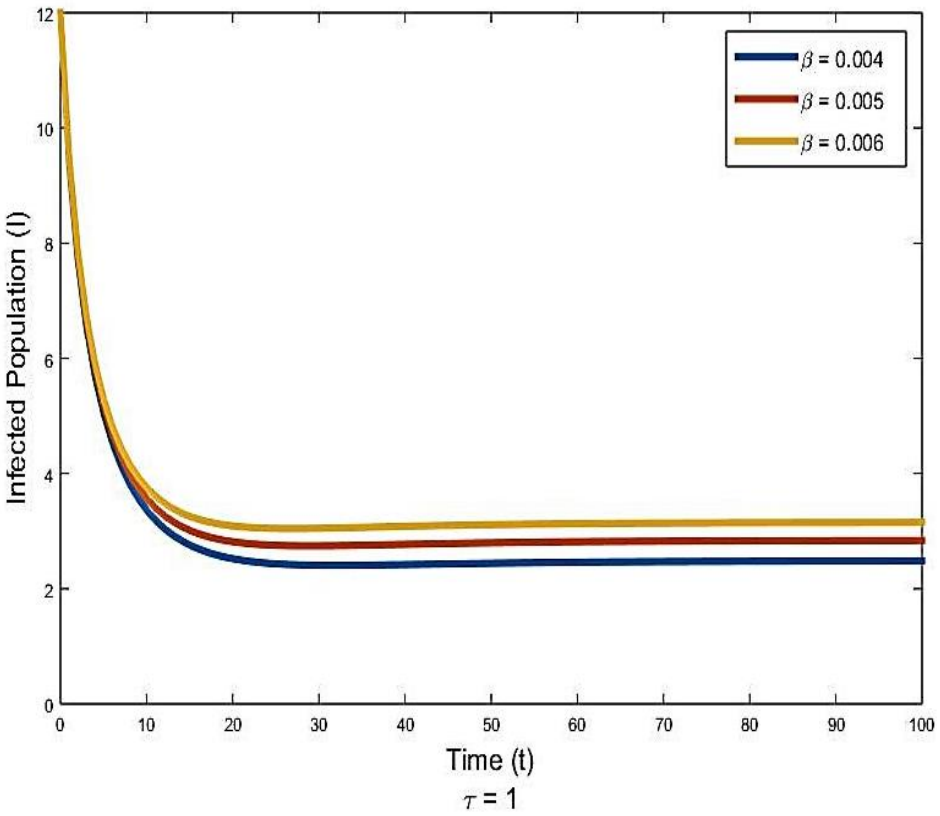


Fig. 6.14: Effect of the transmission rate (β) on the infected population (I).

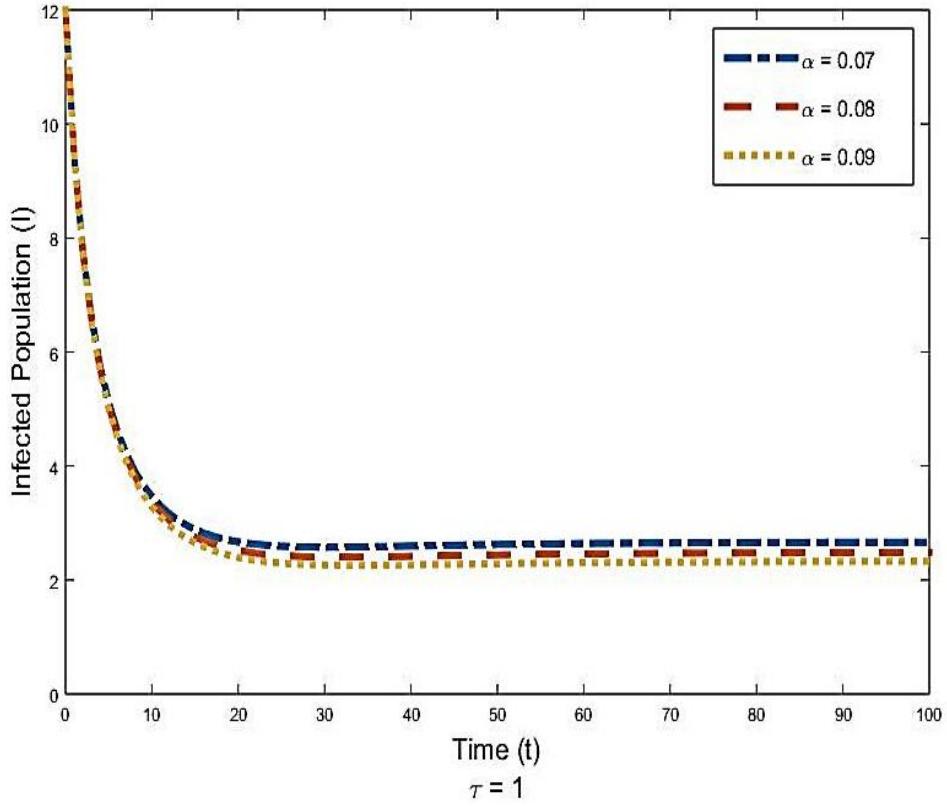


Fig. 6.15: Impact of inhibitory effect (α) on the infected population(I).

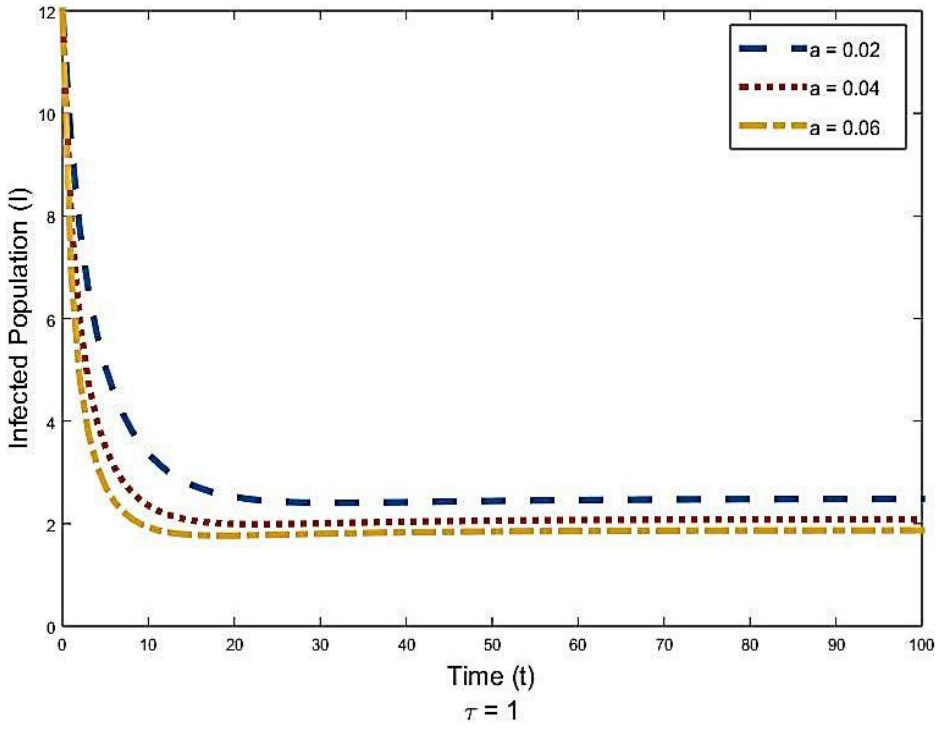


Fig. 6.16: Impact of cure rate (a) on the infected population (I).

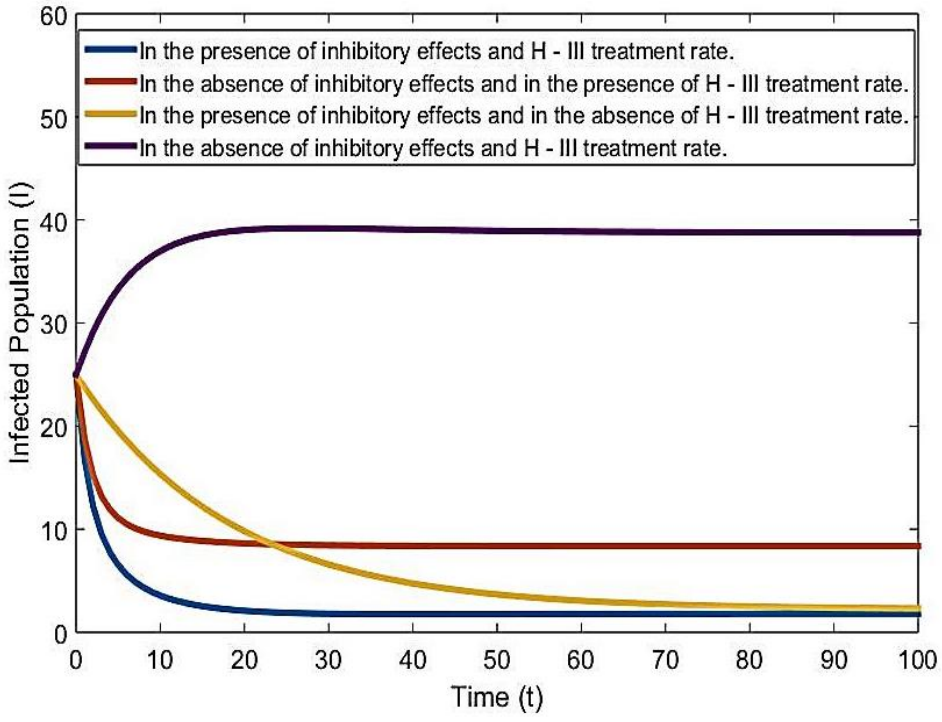


Fig. 6.17: Variation in the infected population (I) with various combinations of incidence and treatment rates at time lag $\tau = 1$.

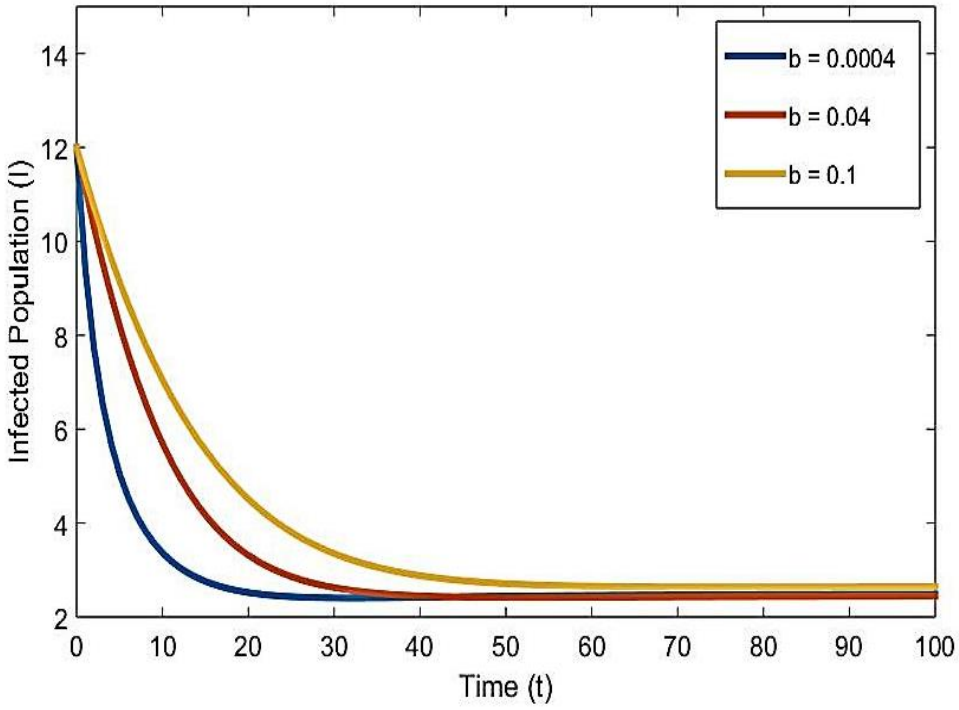
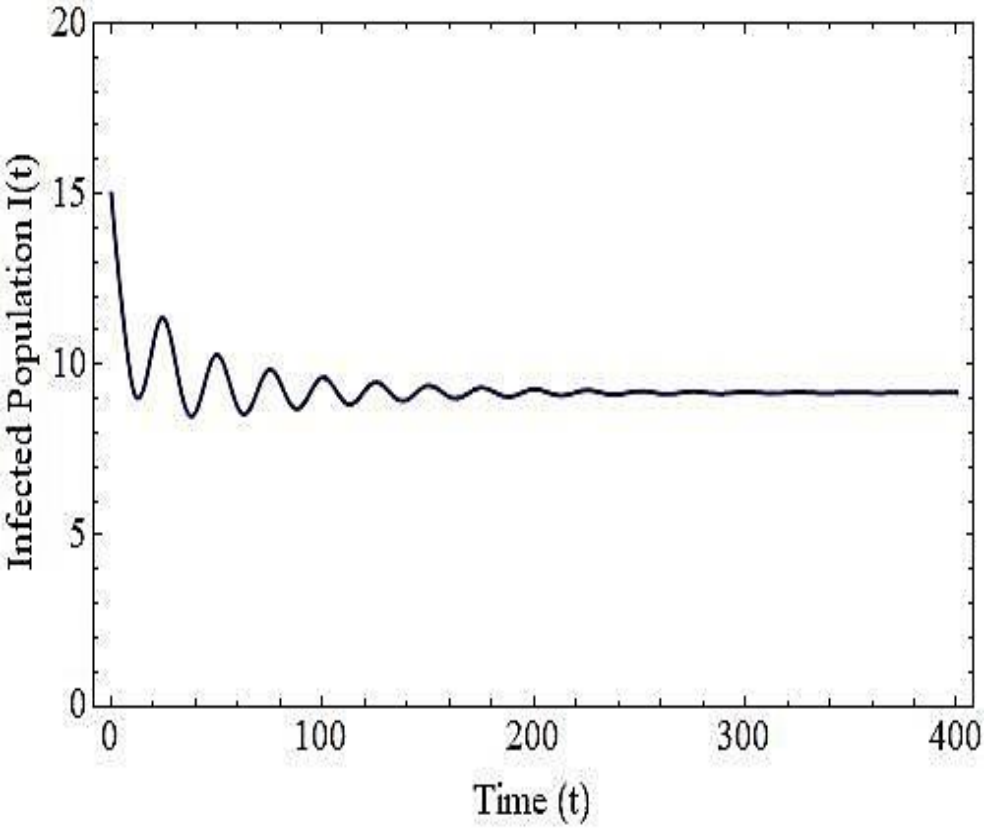
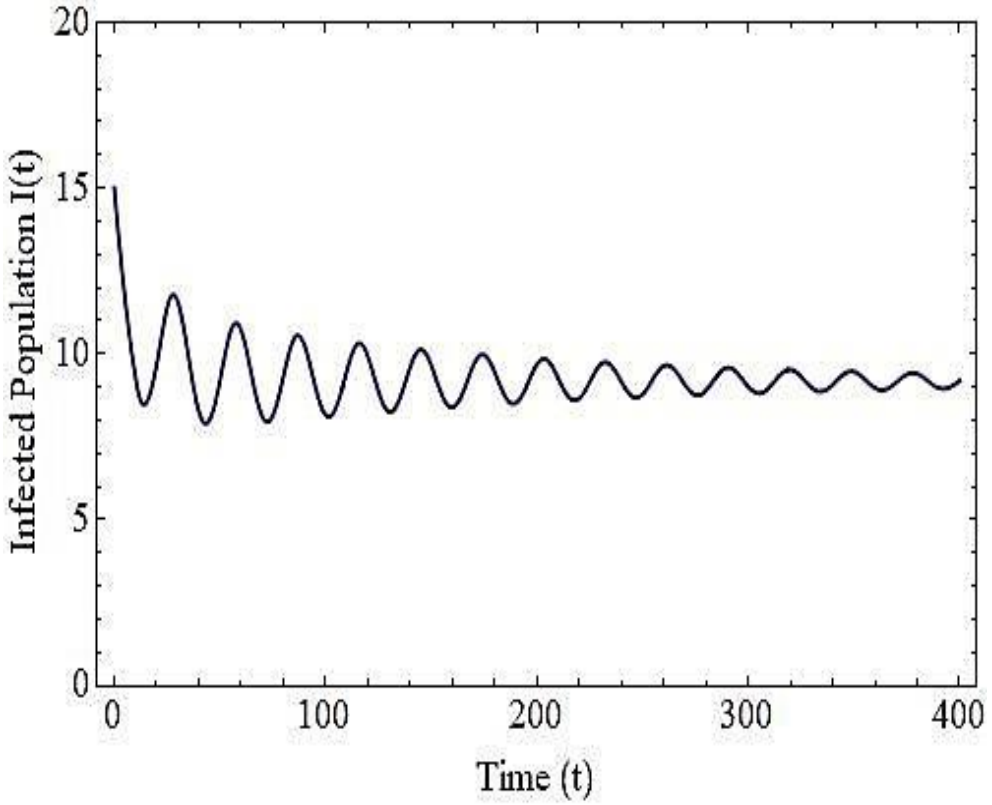


Fig. 6.18: Variation in the infected population (I) at various values of limitation rate (b) in treatment availability.



Figs. 6.19: Oscillatory behavior of the infected population (I) at time lag $\tau = 9$.



Figs. 6.20: Oscillatory behavior of the infected population (I) at time lag $\tau = 11$.

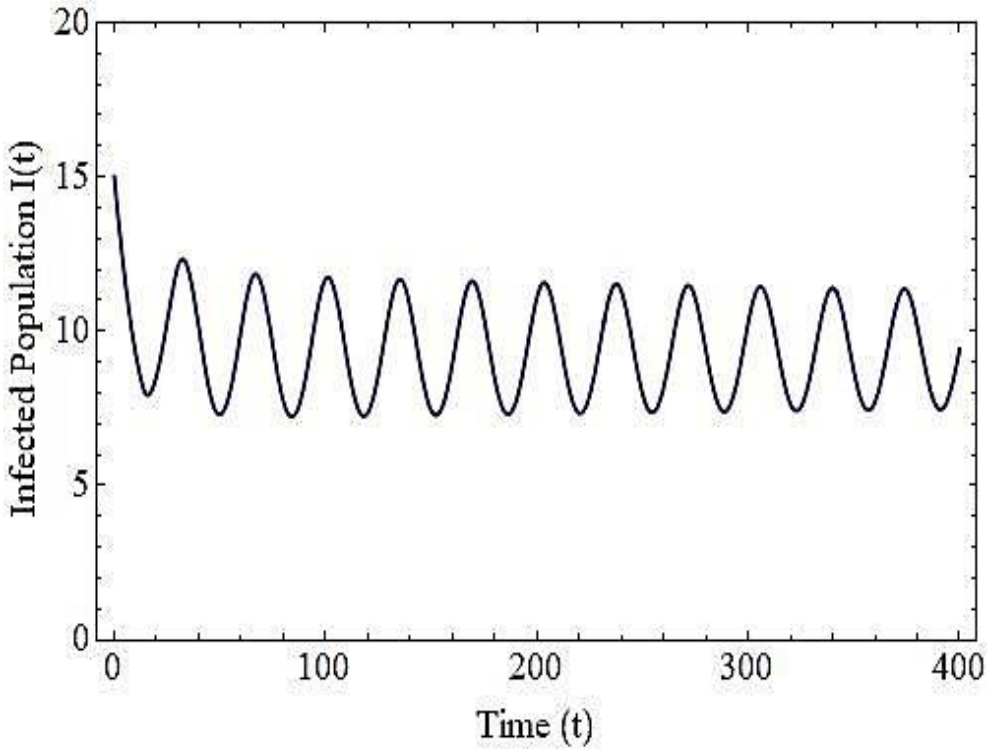


Fig. 6.21: Periodic behavior of the infected population (I) at time lag $\tau = 13.5$.

CHAPTER 7

ANALYSIS OF A TIME-DELAYED SIR EPIDEMIC MODEL WITH CROWLEY-MARTIN FUNCTIONAL TYPE INCIDENCE RATE AND HOLLING FUNCTIONAL TYPE II TREATMENT RATE

In this chapter, the incidence rate of a new infection is considered as Crowley-Martin (C-M) functional type because it considers the effect of inhibition among infectives even in case of the high density of susceptible population which is neglected by any other incidence rate. The latency time has been used as a delay in the incidence rate to understand the dynamics of the epidemic more pragmatically. Therefore, a combination of C-M incidence rate along with time delay and Holling functional type II treatment rate is studied. The local stability, as well as global stability analysis of the model equilibria, is being discussed. The numerical outcomes demonstrate the impact of measure of inhibition, time delay and nonlinear treatment on the infectious population.

7.1 Introduction

The Crowley-Martin (C-M) type of functional response was introduced by Crowley and Martin [1989] and is expressed as below:

$$g\big(S(t), I(t)\big) = \frac{\beta S(t)}{(1 + \alpha S(t))(1 + \gamma I(t))}$$

where α, β, γ are positive constants. From the expression, we observe that similar to the Beddington-DeAngelis type incidence rate (see section 1.5), one can easily derive other forms of incidence rates. The important difference between the Beddington-DeAngelis type and the Crowley-Martin type incidence rate is that the latter considers the effect of inhibition among infectives even in case of the high density of susceptible populations while the former neglects the aforesaid effect. This can be seen as follows:

For Beddington-DeAngelis type incidence rate

$$\lim_{S(t)\to\infty} g(S(t), I(t)) = \frac{\beta}{\alpha},$$

and for Crowley-Martin type incidence rate

$$\lim_{S(t)\to\infty} g(S(t), I(t)) = \frac{\beta}{\alpha(1 + \gamma I(t))}.$$

Models in which the rates of transfer depend on the sizes of compartments over the past as well as at the moment of transfer lead to more general types of realistic models. Time delay has a significant effect on the epidemic dynamics and it strongly influences the model output. Therefore, the present paper aims to study the impact of time delay on an epidemic model with Crowley-Martin incidence rate that determines the course of infection within the individuals and treatment rate as Holling type II (the detailed explanations of Holling functional type II treatment rate is already given in section (3.1)) that design the programs for the control of infection and disease within the different communities. Furthermore, dynamical behavior and stability of the model is governed by the value of the basic reproduction number R_0.

7.2 Mathematical model

In the study of disease transmission model, the total population is divided into three classes of individuals, labeled as susceptible class $S(t)$, infected class $I(t)$ and recovered class $R(t)$. $S(t)$ denotes the individuals who are susceptible to the disease (who are not yet infected at time t) but are capable of catching the disease and can become infected. $I(t)$ denotes the individuals who are infected and are capable of transmitting the disease to others. $R(t)$ denotes the individuals who have been infected and removed from the possibility of being re-infected and enter into the recovered compartment, because of the autoimmune response of the body and treatment. We assume that susceptible individuals are recruited at the rate A. The movements out of the susceptible compartment into the infective compartment is governed by Crowley-Martin incidence rate $\frac{\beta S(t-\tau)I(t-\tau)}{(1+\alpha S(t-\tau))(1+\gamma I(t-\tau))}$; where β denotes the transmission rate, α is the measure of inhibition adopted by susceptibles, γ is the measure of inhibition adopted by infectives, and τ denotes the time delay. The parameters $\mu, d,$ and δ are defined as natural death rate, disease induced death rate and recovery rate respectively. The term $h(I(t)) = \frac{aI(t)}{1+bI(t)}$ defines the Holling type II treatment rate where a is cure rate, b represents limitation rate in resources availability. The movement of the individuals in different compartments is shown by the block diagram in Fig 7.1 below.

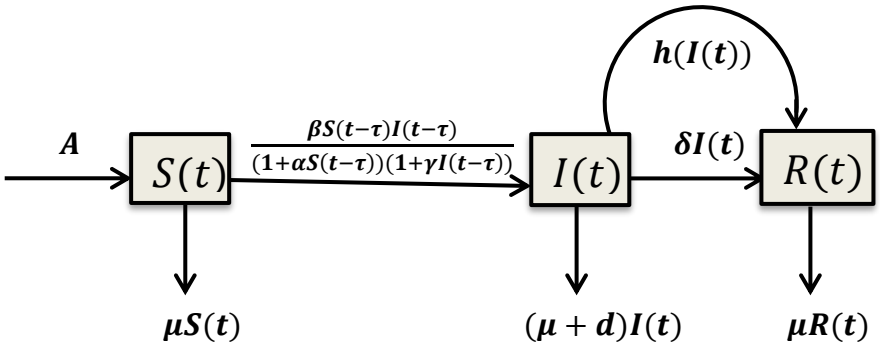


Fig. 7.1: Transfer diagram of the infection through various compartments.

These assumptions lead to the following nonlinear system of the delay differential equations to describe the changes in $S(t)$, $I(t)$ and $R(t)$ with respect to time t:

$\frac{dS(t)}{dt} = A - \mu S(t) - \frac{\beta S(t-\tau)I(t-\tau)}{(1+\alpha S(t-\tau))(1+\gamma I(t-\tau))}$,

$$\frac{dI(t)}{dt} = \frac{\beta S(t-\tau)I(t-\tau)}{(1+\alpha S(t-\tau))(1+\gamma I(t-\tau))} - (\mu + d + \delta)I(t) - \frac{aI(t)}{1+bI(t)} \ , \quad (7.1)$$

$$\frac{dR(t)}{dt} = \frac{aI(t)}{1+bI(t)} + \delta I(t) - \mu R(t) \ .$$

where $\tau > 0$ is a fixed time during which the infectious agents develop in the vector, and it is only after this time that the infected vector can infect a susceptible individual.

The initial conditions of the model (7.1) are given by

$S(\theta) = \varphi_1(\theta), I(\theta) = \varphi_2(\theta), R(\theta) = \varphi_3(\theta),\ \varphi_i(\theta) \geq 0, \theta \in [-\tau, 0], \varphi_i(0) > 0\ (i = 1,2,3)$ (7.2)

where $\big(\varphi_1(\theta), \varphi_2(\theta), \varphi_3(\theta)\big) \in C([-\tau, 0], \mathbb{R}^3_+)$. Here C denotes the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3_+$.

7.3 Basic properties of the model

Since the first two equations of the model (7.1) are free from the effect of R, we consider the following reduced system for analysis:

$$\frac{dS}{dt} = A - \mu S - \frac{\beta S(t-\tau)I(t-\tau)}{(1+\alpha S(t-\tau))(1+\gamma I(t-\tau))},$$

$$\frac{dI}{dt} = \frac{\beta S(t-\tau)I(t-\tau)}{(1+\alpha S(t-\tau))(1+\gamma I(t-\tau))} - (\mu + d + \delta)I - \frac{aI}{1+bI} \ . \quad (7.3)$$

with initial conditions

$$S(\theta) = \varphi_1(\theta), I(\theta) = \varphi_2(\theta),\ \ \varphi_i(\theta) \geq 0, \theta \in [-\tau, 0], \varphi_i(0) > 0\ (i = 1,2), \quad (7.4)$$

where $(\varphi_1(\theta), \varphi_2(\theta),) \in C([-\tau, 0], \mathbb{R}^2_+)$. Here C denotes the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^2_+$.

The equations of the system (7.3) monitor population. It is assumed that all state variables of the system (7.3) are nonnegative *i.e.* $(S, I) \in \mathbb{R}^2_+$ and all parameters $A, \mu, \beta, \alpha, d, \gamma, \delta, a, b > 0$.

We also assume that $F(S(t), I(t)) = \frac{\beta S(t)I(t)}{(1+\alpha S(t))(1+\gamma I(t))}$ and $h(I(t)) = \frac{aI(t)}{1+bI(t)}$ are always positive, continuously differentiable, and monotonically increasing for all $S(t) > 0$ and $I(t) > 0$. That is, they satisfy the following conditions:

A1. $F(S(t),I(t))>0, F'_S(S(t),I(t))>0, F'_I(S(t),I(t))>0$ for $S(t)>0$ and $I(t)>0$.

A2. $F(S(t),0)=F(0,I(t))=0, F'_S(S(t),0)=0, F'_I(S(t),0)>0$ for $S(t)>0$ and $I(t)>0$.

A3. $h(0)=0, h'(0)>0$ for $I(t)\geq 0$.

Theorem 7.1: The set $D=\{(S,I)\in\mathbb{R}^2_+: 0<S(t)+I(t)\leq\frac{A}{\mu}\}$ is a positively invariant and attracting region for the system (7.3).

Proof: The proof of this theorem is as given in section 4.1. Hence, it is omitted here.

7.4 Equilibria and their stability analysis

In this section, we will illustrate our results on the local stability of the equilibria of the system (7.3). Time delayed systems will have the same equilibrium solution as those of zero delayed systems [Tipsri and Chinviriyasit (2014)].

7.4.1 Disease-free equilibrium (DFE)

By setting the derivatives of the system (7.3) to zero, we obtain a unique DFE of the form $Q(\frac{A}{\mu},0)$.

The characteristic equation of the system (7.3) evaluated at disease-free equilibrium Q is obtained as:

$$(\mu+\lambda)\left(\frac{\beta A}{(\mu+\alpha A)}\,e^{-\lambda\tau}-\mu-d-\delta-a-\lambda\right)=0 \qquad (7.5)$$

One of the roots of Eq. (7.5) is given by $\lambda_1=-\mu$ and the other root can be obtained from the equation

$$\frac{\beta A}{(\mu+\alpha A)}\,e^{-\lambda\tau}-\mu-d-\delta-a-\lambda=0.$$

The term $\frac{\beta A}{(\mu+\alpha A)(\mu+d+\delta+a)}\,e^{-\lambda\tau}$ evaluated at $\tau=0$ is termed as the basic reproduction number, represented by R_0. Therefore, R_0 for the system (7.3) is

$$R_0 = \frac{\beta A}{(\mu+\alpha A)(\mu+d+\delta+a)}.$$

7.4.1.1 Analysis for $R_0 \neq 1$

Eq. (7.5) has a negative root $\lambda_1 = -\mu$ and another root can be obtained from equation

$$\lambda + \mu + d + \delta + a - \frac{\beta A}{(\mu+\alpha A)}\, e^{-\lambda\tau} = 0.$$

Let

$$f(\lambda) = \lambda + \mu + d + \delta + a - \frac{\beta A}{(\mu+\alpha A)}\, e^{-\lambda\tau}.$$

If $R_0 > 1$ then for real value of λ,

$$f(0) = \mu + d + \delta + a - \frac{\beta A}{(\mu+\alpha A)} < 0, \qquad \lim_{\lambda\to\infty} f(\lambda) \to +\infty.$$

Hence, $f(\lambda) = 0$ has a positive real root if $R_0 > 1$.

If $R_0 < 1$, we assume that $Re\ \lambda \geq 0$.

Also,

$$Re\ \lambda = \frac{\beta A}{(\mu+\alpha A)}\, e^{-Re\,\lambda\,\tau} \cos Im\ \lambda\ \tau - (\mu + d + \delta + a) \leq \frac{\beta A}{(\mu+\alpha A)} - (\mu + d + \delta + a) < 0,$$

a contradiction to our assumption. Hence, if $R_0 < 1$ then λ is a root of Eq. (7.5) whose real part is negative.

Hence, the following theorem can be stated:

Theorem 7.2: If $R_0 < 1$ then the disease-free equilibrium $Q(\frac{A}{\mu}, 0)$ is locally asymptotically stable and if $R_0 > 1$ then Q is unstable.

7.4.1.2 Analysis at $R_0 = 1$

We see that roots of the system (7.3) at $R_0 = 1$ and bifurcation parameter $\beta = \beta^* = \frac{(\mu+\alpha A)(\mu+d+\delta+a)}{A}$ has one zero eigenvalue and one negative eigenvalue respectively. To analyze the stability of the equilibrium point at $R_0 = 1$, linearization is not applicable to investigate the stability behavior of equilibrium Q, at $R_0 = 1$. We may use the center manifold theory [Sastry (1999)]. For this purpose, we use the notations $S = x_1$ and $I = x_2$. Thus, the system (7.3) can be rewritten as:

$$\frac{dx_1}{dt} = A - \mu x_1 - \frac{\beta x_1 x_2}{(1+\alpha x_1)(1+\gamma x_2)} \equiv f_1,$$

$$\frac{dx_2}{dt} = \frac{\beta x_1 x_2}{(1+\alpha x_1)(1+\gamma x_2)} - (\mu + d + \delta)x_2 - \frac{a x_2}{1+b x_2} \equiv f_2. \qquad (7.6)$$

Let J^* denotes the Jacobian matrix of the system (7.6) evaluated at bifurcation parameter $\beta = \beta^*$ and $R_0 = 1$. Then,

$$J^* = \begin{bmatrix} -\mu & -\frac{\beta^* A}{\mu + \alpha A} \\ 0 & 0 \end{bmatrix}$$

Let $u = [u_1, u_2\,]$ be the left eigenvector and $w = [w_1, w_2\,]^T$ be the right eigenvector of the Jacobian matrix J^*corresponding to the zero eigenvalue. Then we have

$$u_1 = 0, u_2 = 1 \text{ and } w_1 = -\frac{\beta^* A}{\mu(\mu + \alpha A)}, w_2 = 1.$$

The non-zero partial derivatives of the functions f_1 and f_2 of the system (7.6) at $R_0 = 1$ and $\beta = \beta^*$ are obtained as

$$\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_Q = \frac{\beta^* \mu^2}{(\mu+\alpha A)^2}, \left(\frac{\partial^2 f_2}{\partial x_2 \partial x_1}\right)_Q = \frac{\beta^* \mu^2}{(\mu+\alpha A)^2}, \left(\frac{\partial^2 f_2}{\partial {x_2}^2}\right)_Q = \frac{-2\gamma\beta^* A}{(\mu+\alpha A)} + 2ab,$$

$$\left(\frac{\partial^2 f_2}{\partial {x_1}^2}\right)_Q = -\frac{2\alpha\beta^* \mu^3}{\mu+\alpha A}, \left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_Q = \frac{A}{(\mu+\alpha A)}.$$

Then using theorem 4.1 of [Chavez and Song (2004)], the bifurcation constants a_1 and b_1 are

$$a_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q$$

$$= -2\left(\left(\frac{\beta^* A}{\mu+\alpha A}\right)^3 \frac{\alpha\mu}{A(\mu+\alpha A)} + \left(\frac{\beta^* A}{\mu+\alpha A}\right)^2 \frac{\mu}{A(\mu+\alpha A)} + \gamma\frac{\beta^* A}{\mu+\alpha A}\right) + 2ab$$

$$= 2\left(ab - \frac{\mu\beta^* A}{\mu+\alpha A}\left(\frac{{\beta^*}^2 A\alpha}{(\mu+\alpha A)^3} + \frac{\beta^*}{(\mu+\alpha A)^2} + \frac{\gamma}{\mu}\right)\right)$$

$= ab - c$, where $c = \frac{\mu\beta^* A}{\mu+\alpha A}\left(\frac{{\beta^*}^2 A\alpha}{(\mu+\alpha A)^3} + \frac{\beta^*}{(\mu+\alpha A)^2} + \frac{\gamma}{\mu}\right)$

and

$b_1 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q$

$= u_2\left(w_2 \frac{A}{(\mu+\alpha A)}\right)$

$= \frac{A}{(\mu+\alpha A)} > 0.$

Hence, according to the sign of a_1, the following theorem is being proposed:

Theorem 7.3: For the transcritical bifurcations, we have the following results:

- **i)** When $ab < c, a_1 < 0$, the system (7.3) exhibits a forward transcritical bifurcation at disease-free equilibrium (Q) and $R_0 = 1$.
- **ii)** When $ab > c, a_1 > 0$, the system (7.3) either exhibits a backward transcritical bifurcation or saddle-node bifurcation at disease-free equilibrium (Q) and $R_0 = 1$.

The forward bifurcation is illustrated in Fig. 7.2.

7.4.2 Existence and stability analysis of endemic equilibrium

For the existence of an endemic equilibrium $Q^*(S^*, I^*)$, the system (7.3) is rearranged to get S^* and I^* which gives

$$S^* = \frac{A + (Ab - \mu - d - \delta - a)I^* - b(\mu + d + \delta){I^*}^2}{\mu(1 + bI^*)},$$

and I^* is given by the following equation

$$C_1 {I^*}^4 + C_2 {I^*}^3 + C_3 {I^*}^2 + C_4 I^* + C_5 = 0 \quad (7.7)$$

where

$C_1 = \gamma b^2 \alpha(\mu + d + \delta)^2,$

$C_2 = \big(\gamma b\alpha(\mu + d + \delta)(\mu + d + \delta + a) + b(\mu + d + \delta)\big(-\alpha b(\mu + d + \delta) + \gamma\mu b +$

$\gamma\alpha(Ab - \mu - d - \delta - a)\big) + \beta b^2(\mu + d + \delta)\big),$

$C_3 = \Big((\mu + d + \delta + a)\big(-\alpha b(\mu + d + \delta) + \gamma\mu b + \gamma\alpha(Ab - \mu - d - \delta - a)\big) + b(\mu + d + \delta)(\mu b + \alpha(Ab - \mu - d - \delta - a) + \gamma\mu + \gamma\alpha A) - \beta b(Ab - 2\mu - 2d - 2\delta - a)\Big)$,

$C_4 = \big((\mu + d + \delta + a)(\mu b + \alpha(Ab - \mu - d - \delta - a) + \gamma\mu + \gamma\alpha A) + b(\mu + d + \delta)(\mu + \alpha A) - \beta(2Ab - \mu - d - \delta - a)\big)$,

$C_5 = \big((\mu + d + \delta + a)(\mu + \alpha A) - \beta A\big) = (\mu + d + \delta + a)(\mu + \alpha A)(1 - R_0)$.

Using Descartes' rule of signs, there exists a unique positive real root I^* of the biquadratic equation (7.7) if any of the following condition is satisfied:

i. $C_1 > 0, C_2 < 0, C_3 < 0, C_4 < 0$ and $C_5 < 0$.

ii. $C_1 > 0, C_2 > 0, C_3 < 0, C_4 < 0$ and $C_5 < 0$.

iii. $C_1 > 0, C_2 > 0, C_3 > 0, C_4 < 0$ and $C_5 < 0$.

iv. $C_1 > 0, C_2 > 0, C_3 > 0, C_4 > 0$ and $C_5 < 0$.

If any of the above conditions is satisfied then there is a unique $I^* > 0$, from which the value of S^* may be determined as well. This implies that there exists a unique endemic equilibrium $Q^*(S^*, I^*)$.

We now investigate the local stability of Q^*. The characteristic equation of the system (7.3) evaluated at Q^* is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\tau} = 0 \qquad (7.8)$$

where

$p_0 = (2\mu + d + \delta) + \frac{a}{(1+bI^*)^2}$,

$q_0 = \mu\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right)$,

$p_1 = \frac{\beta}{(1+\alpha S^*)(1+\gamma I^*)}\left(\frac{I^*}{(1+\alpha S^*)} - \frac{S^*}{(1+\gamma I^*)}\right)$,

$q_1 = \frac{\beta I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) - \frac{\mu\beta S^*}{(1+\gamma I^*)^2(1+\alpha S^*)}$.

Theorem 7.4: At $\tau = 0$, Q^* is locally asymptotically stable if $\frac{S^*}{I^*} \leq \frac{(1+\gamma I^*)}{(1+\alpha S^*)}$ is satisfied.

Proof: At Q^*, the characteristic equation at $\tau = 0$ is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1) = 0. \tag{7.9}$$

It is easy to show that if $\frac{S^*}{I^*} \leq \frac{(1+\gamma I^*)}{(1+\alpha S^*)}$ is satisfied then

$$p_0 + p_1 = (2\mu + d + \delta) + \frac{a}{(1+bI^*)^2} + \frac{\beta}{(1+\alpha S^*)(1+\gamma I^*)}\left(\frac{I^*}{(1+\alpha S^*)} - \frac{S^*}{(1+\gamma I^*)}\right) > 0,$$

$$q_0 + q_1 = \mu\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) + \frac{\beta I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) - \frac{\mu\beta S^*}{(1+\gamma I^*)^2(1+\alpha S^*)}$$

$$= \mu\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) + \frac{\beta I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}\left((\delta + d) + \frac{a}{(1+bI^*)^2}\right) + \frac{\mu\beta}{(1+\alpha S^*)(1+\gamma I^*)}\left(\frac{I^*}{(1+\alpha S^*)} - \frac{S^*}{(1+\gamma I^*)}\right) > 0.$$

Hence, by the definition of the Routh-Hurwitz criterion, the endemic equilibrium Q^* of the system (7.3) is locally asymptotically stable when $\tau = 0$.

Theorem 7.5: For $\tau > 0$, Q^* is locally asymptotically stable if $(1 + bI^*)^2 \geq \frac{2\mu a}{((\mu+d+\delta)^2+\mu^2)}$, $L_1 \geq L_2$, $L_3 \geq L_4$ and $(1 + \alpha S^*)^2 \geq \frac{\beta I^*}{\mu(1+\gamma I^*)}$ hold true simultaneously, where

$$L_1 = \left(\frac{a}{(1+bI^*)^2}\right)^2 + \frac{2\beta^2 S^* I^*}{(1+\alpha S^*)^3(1+\gamma I^*)^3},$$

$$L_2 = \frac{\beta^2}{(1+\alpha S^*)^2(1+\gamma I^*)^2}\left(\left(\frac{I^*}{(1+\alpha S^*)}\right)^2 + \left(\frac{S^*}{(1+\gamma I^*)}\right)^2\right),$$

$$L_3 = \frac{a}{(1+bI^*)^2}\left(\mu + \frac{\beta I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}\right),$$

$$L_4 = \frac{\mu\beta S^*}{(1+\gamma I^*)^2(1+\alpha S^*)}.$$

Proof: At Q^* the characteristic equation for $\tau > 0$ is given by Eq. (7.8)

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\tau} = 0,$$

For $\tau > 0$, corollary 2.4 of Ruan and Wei [2003] ensure that if the endemic equilibrium Q^* is unstable for the particular value of delay, then roots of the characteristic equation (7.8) must intersect the imaginary axis. Thus, to prove the stability of the system (7.3), we

will use the contradictory assumption *i.e.* we assume that $\lambda = i\omega, \omega > 0$ is the roots of Eq. (7.8). Let $\lambda = i\omega$. Then Eq. (7.8) becomes:

$$-\omega^2 + q_0 + p_1\omega \sin\omega\tau + q_1 \cos\omega\tau + i\,(p_1\omega \cos\omega\tau - q_1 \sin\omega\tau + p_0\omega) = 0 \qquad (7.10)$$

On separating real and imaginary parts of Eq. (7.10), we obtain

$$p_1\omega \sin\omega\tau + q_1 \cos\omega\tau = \omega^2 - q_0 \qquad (7.11)$$

$$p_1\omega \cos\omega\tau - q_1 \sin\omega\tau = -p_0\omega \qquad (7.12)$$

Squaring and adding both sides of Eqs. (7.11) and (7.12) gives

$$\omega^4 + ({p_0}^2 - 2q_0 - {p_1}^2)\omega^2 + ({q_0}^2 - {q_1}^2) = 0 \qquad (7.13)$$

Assuming $\omega^2 = z_1$, Equation (7.13) becomes

$$ {z_1}^2 + Pz_1 + T = 0 \qquad (7.14)$$

where $P = ({p_0}^2 - 2q_0 - {p_1}^2)$ and $T = ({q_0}^2 - {q_1}^2)$.

It is easy to show that if $(1 + bI^*)^2 \geq \frac{2\mu a}{((\mu+d+\delta)^2+\mu^2)}$, $L_1 \geq L_2$, $L_3 \geq L_4$ and $(1 + \alpha S^*)^2 \geq \frac{\beta I^*}{\mu(1+\gamma I^*)}$ are satisfied simultaneously then

$$P = ({p_0}^2 - 2q_0 - {p_1}^2)$$

$$= \left((2\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right)^2 - 2\mu\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right) - \left(\frac{\beta}{(1+\alpha S^*)(1+\gamma I^*)}\left(\frac{I^*}{(1+\alpha S^*)} - \frac{S^*}{(1+\gamma I^*)}\right)\right)^2$$

$$= \frac{2a(2\mu+d+\delta)}{(1+bI^*)^2} + \left((2\mu + d + \delta)^2 - 2\mu\left((\mu + d + \delta) + \frac{a}{(1+bI^*)^2}\right)\right) + \left(\frac{a}{(1+bI^*)^2}\right)^2 + \frac{2\beta^2 S^* I^*}{(1+\alpha S^*)^3(1+\gamma I^*)^3} - \frac{\beta^2}{(1+\alpha S^*)^2(1+\gamma I^*)^2}\left(\left(\frac{I^*}{(1+\alpha S^*)}\right)^2 + \left(\frac{S^*}{(1+\gamma I^*)}\right)^2\right)$$

$$= \frac{2a(2\mu+d+\delta)}{(1+bI^*)^2} + \left((\mu + d + \delta)^2 + \mu^2 - \frac{2\mu a}{(1+bI^*)^2}\right) + \left(\left(\frac{a}{(1+bI^*)^2}\right)^2 + \frac{2\beta^2 S^* I^*}{(1+\alpha S^*)^3(1+\gamma I^*)^3} - \frac{\beta^2}{(1+\alpha S^*)^2(1+\gamma I^*)^2}\left(\left(\frac{I^*}{(1+\alpha S^*)}\right)^2 + \left(\frac{S^*}{(1+\gamma I^*)}\right)^2\right)\right)$$

$$= \frac{2a(2\mu+d+\delta)}{(1+bI^*)^2} + \left((\mu + d + \delta)^2 + \mu^2 - \frac{2\mu a}{(1+bI^*)^2}\right) + (L_1 - L_2) > 0,$$

$$T = ({q_0}^2 - {q_1}^2) = (q_0 + q_1)(q_0 - q_1)$$

$$= \left(\mu\left((\mu+d+\delta)+\frac{a}{(1+bI^*)^2}\right)+\left(\frac{\beta I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}\left((\mu+d+\delta)+\frac{a}{(1+bI^*)^2}\right)-\right.\right.$$

$$\left.\left.\frac{\mu\beta S^*}{(1+\gamma I^*)^2(1+\alpha S^*)}\right)\right)\left(\mu\left((\mu+d+\delta)+\frac{a}{(1+bI^*)^2}\right)-\left(\frac{\beta I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}\left((\mu+d+\delta)+\right.\right.\right.$$

$$\left.\left.\left.\frac{a}{(1+bI^*)^2}\right)-\frac{\mu\beta S^*}{(1+\gamma I^*)^2(1+\alpha S^*)}\right)\right)$$

$$= \left(\mu(\mu+d+\delta)+\frac{\beta(\mu+d+\delta)I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}+\frac{a}{(1+bI^*)^2}\left(\mu+\frac{\beta I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}\right)-\frac{\mu\beta S^*}{(1+\gamma I^*)^2(1+\alpha S^*)}\right)\left(\left((\mu+\right.\right.$$

$$\left.\left.d+\delta)+\frac{a}{(1+bI^*)^2}\right)\left(\frac{\mu}{(1+\alpha S^*)^2}\right)\left((1+\alpha S^*)^2-\frac{\beta I^*}{\mu(1+\gamma I^*)}\right)+\frac{\mu\beta S^*}{(1+\gamma I^*)^2(1+\alpha S^*)}\right)$$

$$= \left(\mu(\mu+d+\delta)+\frac{\beta(\mu+d+\delta)I^*}{(1+\alpha S^*)^2(1+\gamma I^*)}+(L_3-L_4)\right)\left(\left((\mu+d+\delta)+\frac{a}{(1+bI^*)^2}\right)\left(\frac{\mu}{(1+\alpha S^*)^2}\right)\left((1+\right.\right.$$

$$\left.\left.\alpha S^*)^2-\frac{\beta I^*}{\mu(1+\gamma I^*)}\right)+\frac{\mu\beta S^*}{(1+\gamma I^*)^2(1+\alpha S^*)}\right)>0.$$

Evidently, if $P>0$ and $T>0$ are satisfied simultaneously, then by Routh – Hurwitz Criterion, Eq. (7.14) will always have roots with a negative real part. It contradicts our assumption for instability that $\lambda = i\omega$ is a root of Eq. (7.8). Hence, the endemic equilibrium Q^* of the system (7.3) is locally asymptotically stable for $\tau > 0$. Alternatively, by Descartes' rule of signs, Eq. (7.14) does not have any positive roots, implying ω is not real, which contradicts our assumption.

7.4.2.1 Hopf bifurcation analysis

If $T<0$ in Eq. (7.14), then there is a unique positive z_0 satisfying Eq. (7.14) *i.e.* there is a single pair of purely imaginary roots $\pm iz_0$ to Eq. (7.8).

From Eqs. (7.11) and (7.12) τ_n corresponding to z_0 can be obtained as

$$\tau_n = \frac{1}{z_0}\, arccos\left(\frac{(q_1-p_0p_1){z_0}^2-q_0q_1}{{p_1}^2{z_0}^2+{q_1}^2}\right)+\frac{2n\pi}{z_0},\ n=0,1,2,.... \qquad (7.15)$$

For $\tau = 0$, endemic equilibrium Q^* is stable; it remains stable for $\tau < \tau_0$ if $\frac{d}{d\tau}(Re\ (\lambda))\Big|_{\lambda=iz_0} > 0$.

Differentiating Eq. (7.8) with respect to τ, we get

$$\left(2\lambda + p_0 + p_1 e^{-\lambda\tau} - (p_1\lambda + q_1)\tau e^{-\lambda\tau}\right)\frac{d\lambda}{d\tau} = \lambda(p_1\lambda + q_1)e^{-\lambda\tau} \tag{7.16}$$

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{\left(2\lambda+p_0+p_1e^{-\lambda\tau}-(p_1\lambda+q_1)\tau e^{-\lambda\tau}\right)}{\lambda(p_1\lambda+q_1)e^{-\lambda\tau}} = \frac{(2\lambda+p_0)}{\lambda(p_1\lambda+q_1)e^{-\lambda\tau}} + \frac{p_1}{\lambda(p_1\lambda+q_1)} - \frac{\tau}{\lambda}$$

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{(2\lambda+p_0)}{-\lambda(\lambda^2+p_0\lambda+q_0)} + \frac{p_1}{\lambda(p_1\lambda+q_1)} - \frac{\tau}{\lambda}$$

$$\frac{d}{d\tau}\left(Re\,(\lambda)\right)\Big|_{\lambda=iz_0} = Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\Big|_{\lambda=iz_0}$$

$$= Re\left(\frac{(2iz_0+p_0)}{-iz_0(-{z_0}^2+ip_0z_0+q_0)} + \frac{p_1}{iz_0(ip_1z_0+q_1)} - \frac{\tau}{iz_0}\right)$$

$$= Re\left(\frac{1}{z_0}\left(\frac{(2iz_0+p_0)}{({z_0}^2-q_0)i+p_0z_0)} + \frac{p_1}{(-p_1z_0+iq_1)} + i\tau\right)\right)$$

$$= \frac{1}{z_0}\left(\frac{2z_0\left({z_0}^2-q_0\right)+{p_0}^2z_0}{({z_0}^2-q_0)^2+(p_0z_0)^2} - \frac{{p_1}^2z_0}{(p_1z_0)^2+{q_1}^2}\right)$$

$$= \frac{2{z_0}^2+({p_0}^2-2q_0-{p_1}^2)}{(p_1z_0)^2+{q_1}^2}$$ (Since, from Eqs. (7.11) & (7.12), $({z_0}^2 - q_0)^2 + (p_0z_0)^2 = (p_1z_0)^2 + {q_1}^2$).

Under the condition ${p_0}^2 - 2q_0 - {p_1}^2 > 0$, we have $\frac{d}{d\tau}\left(Re\,(\lambda)\right)\Big|_{\lambda=iz_0} > 0$.

Therefore, the transversality condition holds and Hopf bifurcation occurs at $\omega = z_0, \tau = \tau_0$.

By summarizing the above analysis, we arrive at the following theorem:

Theorem 7.6: The endemic equilibrium Q^* of the system (7.3) is asymptotically stable for $\tau \in [0, \tau_0)$ and it undergoes Hopf bifurcation at $\tau = \tau_0$.

7.4.3 Global stability analysis

In this section, we study the global stability of disease-free (Q) and endemic (Q^*) equilibria:

7.4.3.1 Global stability of disease-free equilibrium (DFE)

In this subsection, we show the global asymptotic stability for disease-free equilibrium $Q\left(\frac{A}{\mu}, 0\right)$ of the system (7.3). For this, we assume the following:

A4. $F_I'(S(t),0)$ is increasing with respect to $S(t) > 0$,

A5. $\frac{F_I'(S_0,0)}{F_I'(S(t),0)} < 1$ for $S(t) > S_0$; $\frac{F_I'(S_0,0)}{F_I'(S(t),0)} > 1$ for $S(t) \in (0, S_0)$.

A6. $F\big(S(t), I(t)\big) \leq I(t)\left(\frac{\partial F(S(t),I(t))}{\partial I}\Big|_{(S_0,0)} - \frac{\partial G(I)}{\partial I}\Big|_{I=0}\right) + G\big(I(t)\big), I(t) > 0.$

Under these assumptions, we have the following theorem:

Theorem 7.7: Suppose that (A1) – (A6) are satisfied, the disease-free equilibrium $\left(Q(S_0, 0),\ S_0 = \frac{A}{\mu}\right)$ of the system (7.3) is globally asymptotically stable for any $\tau \geq 0$ if $R_0 \leq 1$.

Proof: From the conditions (A1) and (A2), it follows that the disease-free equilibrium $Q(S_0, 0)$ is the only equilibrium of the system (7.3). We define the following Lyapunov functional:

$W_1(t) = Y_1(t) + Y_2(t)$

where

$$Y_1(t) = S(t) - S_0 - \int_{S_0}^{S(t)} \lim_{I \to 0^+} \frac{F\big(S_0, I(t)\big)}{F\big(\sigma, I(t)\big)}\, d\sigma + I(t),$$

$$Y_2(t) = \int_0^{\tau} F\big(S(t-\rho), I(t-\rho)\big)\, d\rho.$$

By (A1) – (A3), it can be seen that $W_1(t)$ is defined and continuously differentiable for all $S(t), I(t) > 0$, and $W_1(t) = 0$ at $Q(S_0, 0)$. We show that $\frac{dW_1}{dt} \leq 0$ for all $t \geq 0$. First, we calculate $\frac{dY_1}{dt}$.

$$\frac{dY_1}{dt} = \left(1 - \lim_{I \to 0^+} \frac{F\big(S_0, I(t)\big)}{F\big(S, I(t)\big)}\right) S'(t) + I'(t)$$

Now, $A - \mu S = -\mu\left(S - \frac{A}{\mu}\right) = -\mu(S - S_0)$

Therefore,

$$\frac{dY_1}{dt} = \left(1 - \lim_{I\to 0^+} \frac{F\left(S_0, I(t)\right)}{F\left(S, I(t)\right)}\right)\left(-\mu(S - S_0) - F\left(S(t-\tau), I(t-\tau)\right)\right) + F\left(S(t-\tau), I(t-\tau)\right) - G(I(t))$$

where,

$$G(I(t)) = (\mu + d + \delta)I(t) + h\left(I(t)\right).$$

Now, calculating

$$\frac{dY_2}{dt} = -F\left(S(t-\tau), I(t-\tau)\right) + F(S(t), I(t))$$

Therefore, it follows that

$$\frac{dW_1}{dt} = \frac{dY_1}{dt} + \frac{dY_2}{dt}.$$

$$\frac{dW_1}{dt} = \left(1 - \lim_{I\to 0^+} \frac{F\left(S_0, I(t)\right)}{F\left(S, I(t)\right)}\right)\left(-\mu(S - S_0) - F\left(S(t-\tau), I(t-\tau)\right)\right) + F\left(S(t-\tau), I(t-\tau)\right) - G(I) - F\left(S(t-\tau), I(t-\tau)\right) + F(S(t), I(t))$$

$$= \mu\left(1 - \lim_{I\to 0^+} \frac{F\left(S_0, I(t)\right)}{F\left(S, I(t)\right)}\right)(S_0 - S(t)) + F\left(S(t-\tau), I(t-\tau)\right)\left(\lim_{I\to 0^+} \frac{F\left(S_0, I(t)\right)}{F\left(S, I(t)\right)} - 1\right) + F\left(S(t), I(t)\right) - G(I).$$

Furthermore, (A4)-(A6) implies that

$$\frac{dW_1}{dt} \leq \mu\left(1 - \frac{F_I'(S_0, 0)}{F_I'(S(t),0)}\right)\left(S_0 - S(t)\right) + F\left(S(t-\tau), I(t-\tau)\right)\left(\frac{F_I'(S_0, 0)}{F_I'(S(t),0)} - 1\right) + I(t)\left(\left.\frac{\partial F\left(S(t), I(t)\right)}{\partial I}\right|_{(S_0,0)} - \left.\frac{\partial G(I)}{\partial I}\right|_{I=0}\right)$$

$$= \mu\left(1 - \frac{F_I'(S_0,0)}{F_I'(S(t),0)}\right)\left(S_0 - S(t)\right) + F\left(S(t-\tau), I(t-\tau)\right)\left(\frac{F_I'(S_0,0)}{F_I'(S(t),0)} - 1\right) + I(t).\left.\frac{\partial G(I)}{\partial I}\right|_{I=0}.(R_0 - 1).$$

Therefore, $R_0 \leq 1$ ensures that $\frac{dW_1}{dt} \leq 0$ for all $t \geq 0$, where $\frac{dW_1}{dt} = 0$ holds if $S(t) = S_0$. Hence it follows from the system (7.3) that largest invariant set in $\left\{\left(S(t), I(t)\right) \in \mathbb{R}_+^2 \left| \frac{dW_1}{dt} = 0\right.\right\}$ is the singleton set $Q(S_0, 0)$. From the Lyapunov-LaSalle asymptotic stability theorem [Huang *et al.* (2010a) & (2010b); Hale and Lunel (1993); Li and Liu (2014)], Q is the only equilibrium of the system (7.3) and globally asymptotically stable. This completes the proof.

7.4.3.2 Global stability of endemic equilibrium (EE)

In this subsection, we study the global stability of the endemic equilibrium $Q^*(S^*, I^*)$ of the system (7.3) using the Lyapunov direct method. We propose the following hypothesis:

A7. $\left(\frac{F(S^*,I^*)}{F(S(t),I^*)} - \frac{I^*}{I(t)}\right) \leq 0; \left(\frac{F(S(t),I(t))}{F(S^*,I^*)} - 1\right) \leq 0; \left(\frac{F(S(t),I^*)}{F(S(t),I(t))} - \frac{I(t)}{I^*}\right) \leq 0$ for $I \geq I^*$.

A8. $\left(\frac{h(I^*)}{h(I(t))} - \frac{I^*}{I(t)}\right)\left(1 - \frac{I(t)}{I^*}\right) \leq 0$ for $I \geq I^*$.

Based on these hypotheses, the following theorem may be stated:

Theorem 7.8: Assume that the conditions (A1) – (A3) and (A7) – (A8) are satisfied. Then the endemic equilibrium $Q^*(S^*, I^*)$ of the system (7.3) is globally asymptotically stable for any $\tau \geq 0$ if $R_0 > 1$.

Proof: We assume the following Lyapunov functional

$$W_2(t) = X_1(t) + X_2(t)$$

where

$$X_1(t) = S(t) - S^* - \int_{S^*}^{S(t)} \frac{F(S^*, I^*)}{F(\varphi, I^*)}\, d\varphi + I(t) - I^* - I^* \ln \frac{I(t)}{I^*},$$

$$X_2 = F(S^*, I^*) \int_0^\tau \left(\frac{F\big(S(t-\theta), I(t-\theta)\big)}{F(S^*, I^*)} - 1 - \ln \frac{F\big(S(t-\theta), I(t-\theta)\big)}{F(S^*, I^*)} \right) d\theta.$$

From (A1)–(A3), $W_2(t) = X_1(t) + X_2(t)$ is defined and continuously differentiable for all $S(t), I(t) > 0$ and $W_2(0) = 0$ at $Q^*(S^*, I^*)$. At $Q^*(S^*, I^*)$, the system (7.3) has

$$A - \mu S^* = F(S^*, I^*) = (\mu + d + \delta) I^* + h(I^*) \tag{7.17}$$

The time derivative of $X_1(t)$ along the solution of the system (7.3) is given by

$$\frac{dX_1}{dt} = \left(1 - \frac{F(S^*, I^*)}{F(S(t), I^*)}\right) S'(t) + \left(1 - \frac{I^*}{I(t)}\right) I'(t)$$

$$= \left(1 - \frac{F(S^*, I^*)}{F(S(t), I^*)}\right) \left(\mu S^* - \mu S(t) + F(S^*, I^*) - F\big(S(t-\tau), I(t-\tau)\big)\right)$$

$$+ \left(1 - \frac{I^*}{I(t)}\right) \left(F\big(S(t-\tau), I(t-\tau)\big) \right.$$

$$\left. - \frac{\big(F(S^*, I^*) - h(I^*)\big)}{I^*} I(t) - h\big(I(t)\big) \right)$$

$$= \mu\big(S^* - S(t)\big) \left(1 - \frac{F(S^*, I^*)}{F(S(t), I^*)}\right)$$

$$+ F(S^*, I^*) \left(1 - \frac{F(S^*, I^*)}{F(S(t), I^*)} + \frac{F\big(S(t-\tau), I(t-\tau)\big)}{F(S(t), I^*)}\right)$$

$$- \frac{F\big(S(t-\tau), I(t-\tau)\big)}{I(t)} I^* + F(S^*, I^*) \left(1 - \frac{I(t)}{I^*}\right)$$

$$+ \left(h(I^*) - \frac{h\big(I(t)\big)}{I(t)} I^*\right) \left(1 - \frac{I(t)}{I^*}\right).$$

Further, we have

$$\frac{dX_2(t)}{dt} = F(S^*, I^*). \frac{d}{dt} \int_0^\tau \left(\frac{F\big(S(t-\theta), I(t-\theta)\big)}{F(S^*, I^*)} - 1 - \ln \frac{F\big(S(t-\theta), I(t-\theta)\big)}{F(S^*, I^*)} \right) d\theta$$

$$= F\big(S(t), I(t)\big) - F\big(S(t-\tau), I(t-\tau)\big) + F(S^*, I^*) \ln \frac{F\big(S(t-\tau), I(t-\tau)\big)}{F\big(S(t), I(t)\big)}.$$

Then we have

$$\frac{dW_2(t)}{dt} = \frac{dX_1(t)}{dt} + \frac{dX_2(t)}{dt}$$

$$\begin{aligned}
\frac{dW_2(t)}{dt} &= \mu\big(S^* - S(t)\big)\left(1 - \frac{F(S^*, I^*)}{F(S(t), I^*)}\right) \\
&\quad + F(S^*, I^*)\left(1 - \frac{F(S^*, I^*)}{F(S(t), I^*)} + \frac{F\big(S(t-\tau), I(t-\tau)\big)}{F(S(t), I^*)}\right) \\
&\quad - \frac{F\big(S(t-\tau), I(t-\tau)\big)}{I(t)} I^* + F(S^*, I^*)\left(1 - \frac{I(t)}{I^*}\right) \\
&\quad + \left(h(I^*) - \frac{h\big(I(t)\big)}{I(t)} I^*\right)\left(\frac{I(t)}{I^*} - 1\right) + F\big(S(t), I(t)\big) \\
&\quad - F\big(S(t-\tau), I(t-\tau)\big) + F(S^*, I^*) \ln \frac{F\big(S(t-\tau), I(t-\tau)\big)}{F\big(S(t), I(t)\big)}
\end{aligned}$$

$$\begin{aligned}
&= \mu\big(S^* - S(t)\big)\left(1 - \frac{F(S^*, I^*)}{F(S(t), I^*)}\right) \\
&\quad + F(S^*, I^*)\left(1 - \frac{F(S^*, I^*)}{F(S(t), I^*)} + \ln \frac{F(S^*, I^*)}{F(S(t), I^*)}\right) \\
&\quad + F\big(S(t-\tau), I(t-\tau)\big)\left(\frac{F(S^*, I^*)}{F(S(t), I^*)} - \frac{I^*}{I(t)}\right) \\
&\quad + F(S^*, I^*)\left(1 - \frac{I(t)}{I^*} + \ln \frac{I(t)}{I^*}\right) \\
&\quad + \left(\frac{h(I^*)}{h\big(I(t)\big)} - \frac{I^*}{I(t)}\right)\left(\frac{I(t)}{I^*} - 1\right) h\big(I(t)\big) \\
&\quad + F(S^*, I^*)\left(1 - \frac{F\big(S(t-\tau), I(t-\tau)\big)}{F\big(S(t), I(t)\big)} \cdot \frac{F(S(t), I^*)}{F(S^*, I^*)} \cdot \frac{I^*}{I(t)}\right. \\
&\quad \left. + \ln\left(\frac{F\big(S(t-\tau), I(t-\tau)\big)}{F\big(S(t), I(t)\big)} \cdot \frac{F(S(t), I^*)}{F(S^*, I^*)} \cdot \frac{I^*}{I(t)}\right)\right) \\
&\quad + F(S^*, I^*)\left(\frac{F\big(S(t), I(t)\big)}{F(S^*, I^*)} - 1\right) \\
&\quad + F\big(S(t-\tau), I(t-\tau)\big) \cdot \frac{I^*}{I(t)}\left(\frac{F(S(t), I^*)}{F(S(t), I(t))} - \frac{I(t)}{I^*}\right).
\end{aligned}$$

The function $F(S(t), I(t))$ is monotonically increasing for any $S(t) > 0$; hence, the following inequality holds:

$$\big(S^* - S(t)\big)\left(1 - \tfrac{F(S^*, I^*)}{F(S(t), I^*)}\right) \leq 0. \tag{7.18}$$

By the properties of the function $K(x) = 1 - x + \ln x$, $(x > 0)$, we note that $K(x)$ has its global maximum $K(1) = 0$. Hence, $K(x) \leq 0$ when $x > 0$ and the following inequalities hold true:

$$\left(1-\frac{F(S^*,I^*)}{F(S(t),I^*)}+\ln\frac{F(S^*,I^*)}{F(S(t),I^*)}\right)\leq 0,\left(1-\frac{I(t)}{I^*}+\ln\frac{I(t)}{I^*}\right)\leq 0,$$

$$\left(1-\frac{F\left(S(t-\tau),I(t-\tau)\right)}{F\left(S(t),I(t)\right)}.\frac{F(S(t),I^*)}{F(S^*,I^*)}.\frac{I^*}{I(t)}+\ln\frac{F\left(S(t-\tau),I(t-\tau)\right)}{F\left(S(t),I(t)\right)}.\frac{F(S(t),I^*)}{F(S^*,I^*)}.\frac{I^*}{I(t)}\right)\leq 0. \quad (7.19)$$

Hence, by (A7) - (A8) and inequalities (7.18) - (7.19), we see that $\frac{dW_2}{dt}\leq 0$ for all $S(t)\geq 0, I(t)\geq 0$. It is easy to verify that the largest invariant in $\left\{\left(S(t),I(t)\right)\in\mathbb{R}_+^2:\frac{dW_2}{dt}=0\right\}$ is singleton $\{Q^*\}$. By the Lyapunov-LaSalle asymptotic stability theorem [Huang *et al.* (2010a) & (2010b); Hale and Lunel (1993); Li and Liu (2014)], Q^* is globally asymptotically stable.

7.5 Numerical simulation

In this section, we represent the results of numerical simulation. For simulations, we take the numerical experimental values of parameters as given in Table 7.1.

Graphs have been plotted for *S* and *I* for different values of time lag τ. The trajectory of *S* and *I* approach to the endemic equilibrium as shown in Fig. 7.3 and Fig. 7.4 for $\tau = 0$ and 2 respectively.

Fig. 7.3 and Fig. 7.4 depict the combined population of susceptible and infected individuals for time lag $\tau = 0$ and 2 respectively. According to the data given in Table 7.1, we found that the value of R_0 is 4.3. Thus, the disease will be endemic in the population, which can be observed from the figures that as time passes both the populations approaches to the endemic equilibrium.

Fig. 7.5 delineates the difference between the infected populations at the various values of time lag τ. It is observed that the infected population is less at $\tau = 0$ than the infected population at $\tau = 1, 2$ and 3 respectively. It can be deduced that delay in showing the symptoms of the disease will cause a slight increase in the infected population.

Fig. 7.6 shows the susceptible population at various values of the transmission rate (β) at $\tau = 1$. It is evident that the susceptible population increases when the transmission rate (β) decreases.

Fig. 7.7 demonstrates the variation in the infected population for different values of β at $\tau = 1$. It shows the dynamics of the model for larger values of β. This figure shows that for an increase in value of the probability of transmission per contact rate (or effective contact rate) β, the number of infectives increases, which is biologically true. It can be observed that the decline in transmission rate (β) contributed to a sharp drop in the spread of disease. As the value of β decreases, the number of infected individuals also decreases and settles down to a steady state which shows that disease will not eradicate completely, rather it will persist in the community.

Fig. 7.8 and Fig. 7.9 are depicting the infected population at different values of α and γ at $\tau = 1$. It is noticed that the infected population is decreasing when α and γ are increasing. It shows that higher values of α and γ, will restrict the possibility of spreading the disease. Thus, it is concluded that inhibitory effects among susceptibles and infectives will help in diminishing the spread of disease. Also, as inhibitory effects increase, the delay in peak infection increases.

Fig. 7.10 and Fig. 7.11 show the effect of cure rate (a) and limitation rate (b) in treatment availability on the infected population with various values of a and b at $\tau = 1$ day. Fig. 7.10 shows the decrease in infected population as cure rate (a) increases and it settles down at its steady state, but the disease is not getting totally eliminated as it will persist at a lower level. Fig. 7.11 shows an increase in infected population as b increases, which is due to the limited availability of treatment resources in the society.

Fig. 7.12 demonstrates the difference in the infected population with and without treatment rate $h\,(I)$ at $\tau = 1$ day. It can be clearly observed that the infected population will decrease drastically if Holling type II treatment is given to the infected population.

Fig. 7.13 presents the oscillatory behavior of the infected population with time. Fig. 7.14 shows the population in S-I & S-I-R plane respectively. According to Eq. (7.14) and theorem 7.6, the values have been computed as; $\tau_0 = 10.0726, {p_0}^2 - 2q_0 - {p_1}^2 = 0.00100295 > 0, \text{and } {q_0}^2 - {q_1}^2 = -0.0000373748 < 0$. From Fig. 7.13 it can be viewed that when $\tau = 9.5 < \tau_0 = 10.0726$ then the endemic equilibrium is asymptotically stable.

Figs. 7.15 & 7.16 show the variation in the infected population for increased cure rate (a) and measures of inhibition (γ) taken by infected respectively for the data given in Table 7.1. In both figures, the infectives are decreasing due to the increments in the cure rate and measure of inhibition taken by infected respectively.

7.6 Conclusions

In this chapter, we developed and analyzed a time delayed SIR epidemic model with Crowley-Martin (C-M) functional type incidence rate and Holling type II treatment rate. From the analysis of the model, we demonstrated that the model has two equilibria named as disease-free equilibrium (DFE) and endemic equilibrium (EE). The stability analysis of DFE is investigated by the basic reproduction number R_0 and it is concluded that DFE is locally and globally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$ for time lag $\tau \geq 0$. Clearly, it indicates that the infection will persist in the society when the basic reproduction number is greater than one and disease will die out when the basic reproduction number is less than one. We also showed that the system (7.3) undergoes either a forward bifurcation or backward bifurcation or saddle-node bifurcation under certain conditions at $R_0 = 1$. We have investigated that EE of the system (7.3) for time lag $\tau \geq 0$ is locally asymptotically stable if the inequalities as stated in theorem 7.4 and theorem 7.5 respectively hold true. Hopf bifurcation analysis of the system (7.3) at EE has also presented. Further, we analyzed the global stability of DFE & EE and obtained the conditions as stated in theorems 7.7 and 7.8 respectively. Simulation has been carried out to delineate the effects of time delay, cure rate, limitation rate in available treatment, and measures of inhibition accepted by susceptible and infected individuals. The numerical simulation of the model shows that the infection will see a rise with the increment in transmission rate and settles down at a lower level because of the availability of treatment. Further, decrement in infection is being observed with an increment in the measure of inhibition adopted by susceptible and infective. Furthermore, Hopf bifurcation at endemic equilibrium has also been discussed numerically.

Table 7.1: Description and numerical values of parameters for simulation

Parameter	**Value**
A (Recruitment rate)	11
α (Measure of inhibition taken by susceptible)	0.005
β (Transmission rate)	0.003
μ (Natural death rate)	0.03
d (Disease induced death rate)	0.04
γ (Measure of inhibition taken by infected)	0.005
δ (Recovery rate)	0.001
a (Cure rate)	0.02
b (Limitation rate in treatment availability)	0.02

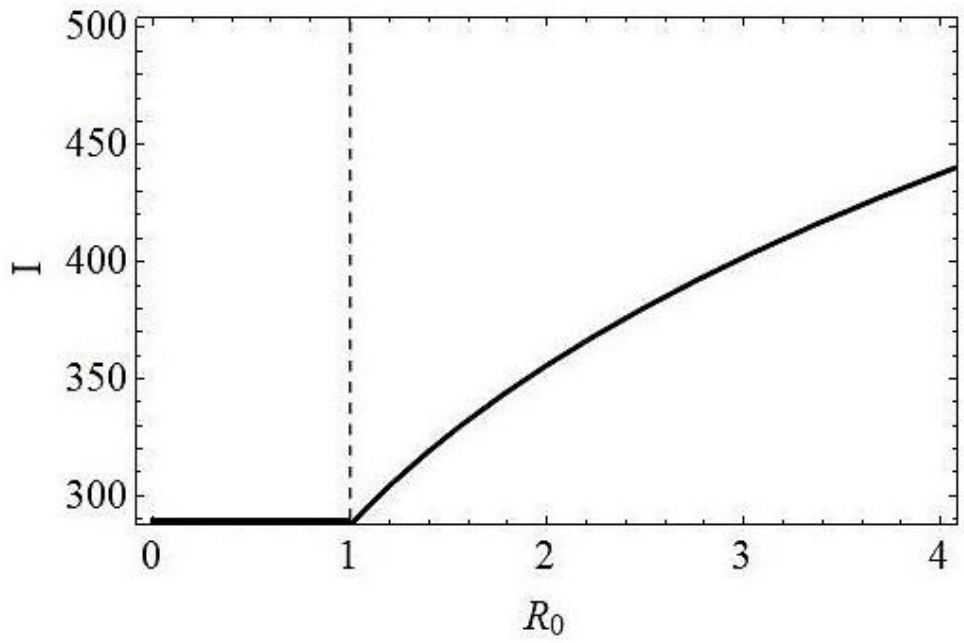


Fig. 7.2: Bifurcation diagram in (R_0, I) plane: forward bifurcation for the data set $A = 10, \alpha = 0.005, \beta = 0.00073, \gamma = 0.005, \mu = 0.03, \delta = 0.001, d = 0.04, a = 0.02$ and $b = 0.02$.

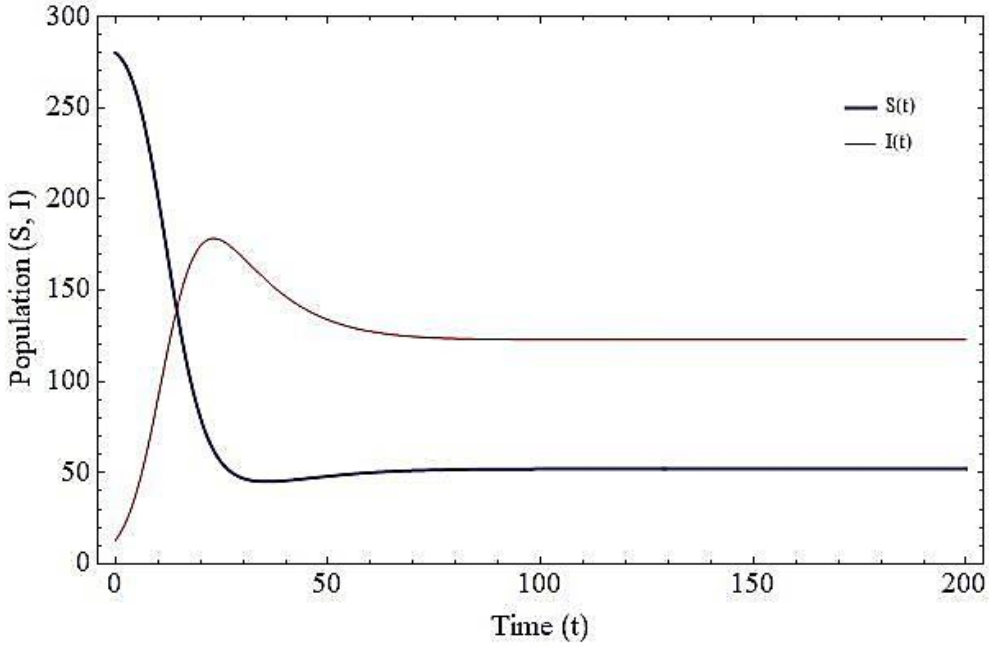


Fig. 7.3: Population (S, I) at time lag $\tau = 0$ and $R_0 = 4.3$.

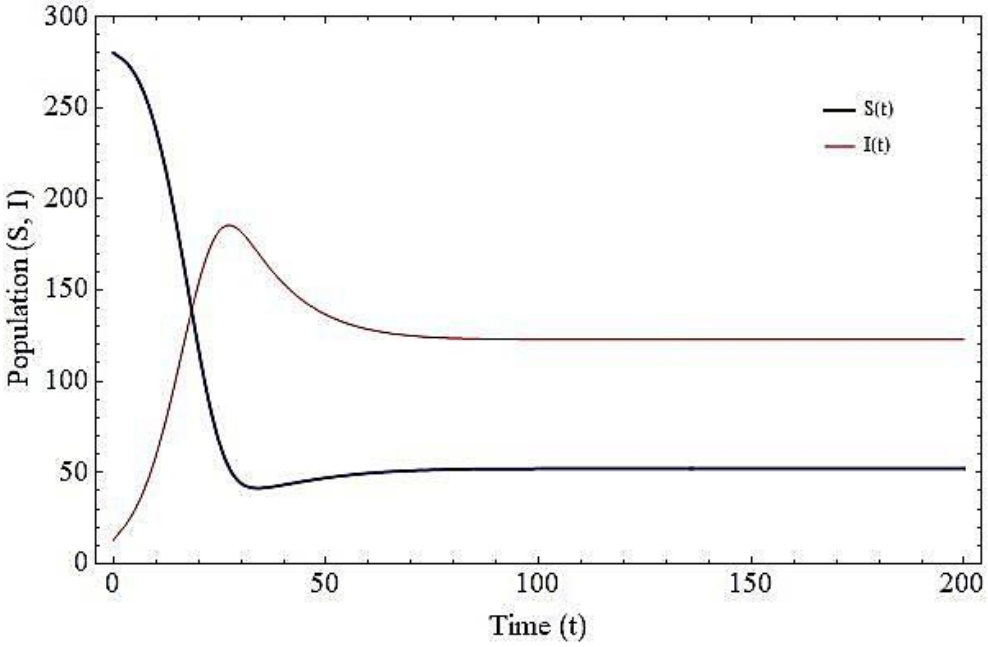


Fig. 7.4: Population (S, I) at time lag $\tau = 2$ and $R_0 = 4.3$.

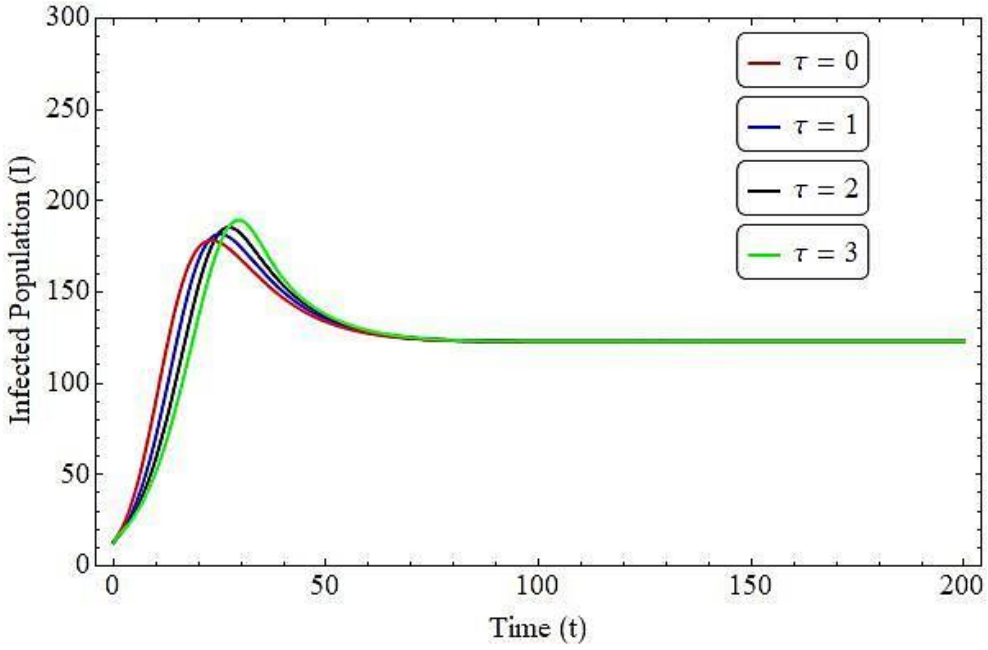


Fig. 7.5: Infected Population (I) at increased values of time lag τ.

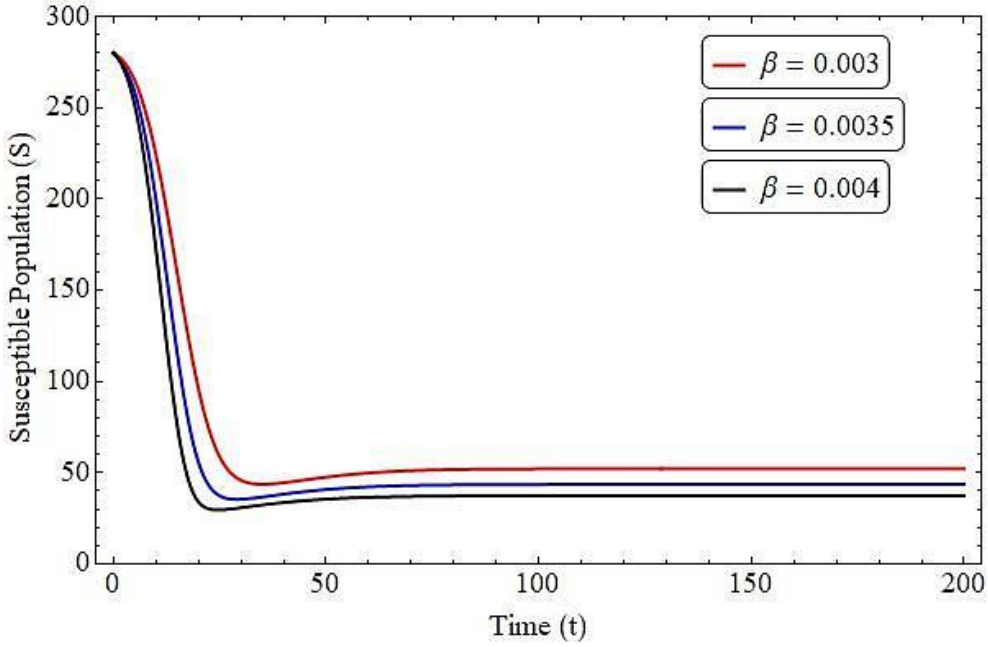


Fig. 7.6: Susceptible population (S) for increased values of transmission rate (β) at time lag $\tau = 1$.

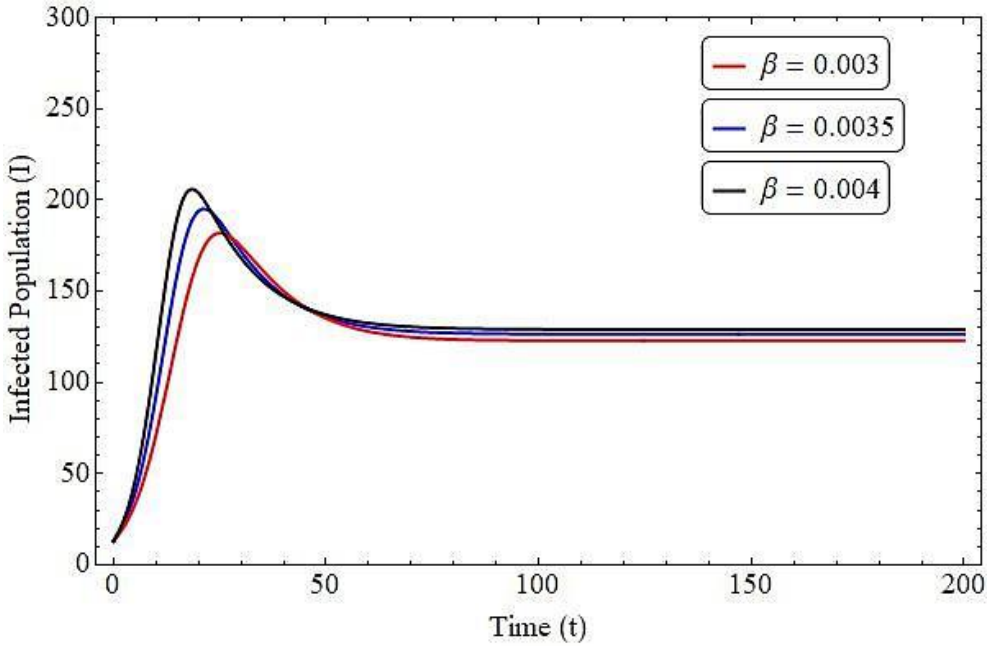


Fig. 7.7: Infected population (I) for increased values of the transmission rate (β) at time lag $\tau = 1$.

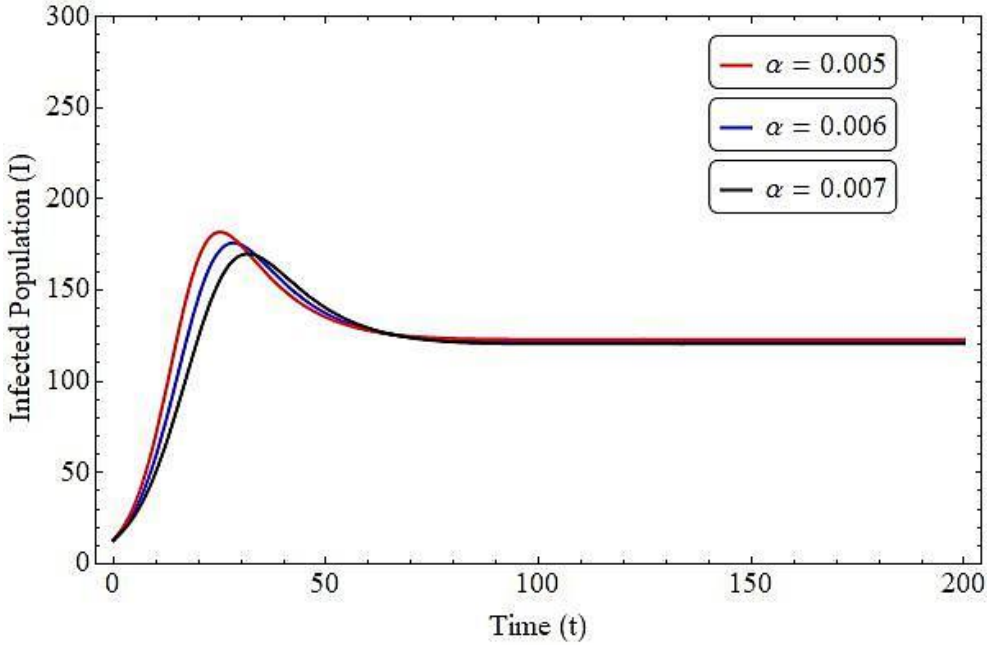


Fig. 7.8: Infected population (I) at increased values of measures of inhibition (α) due to the susceptibles at time lag$\tau = 1$.

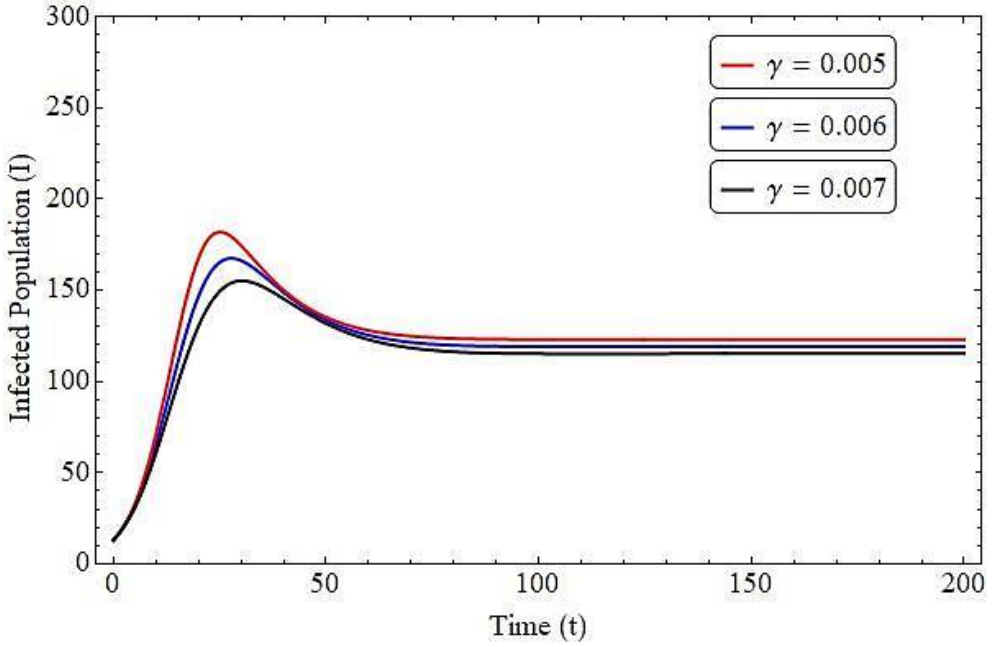


Fig. 7.9: Infected population (I) at increased values of measures of inhibition (γ) due to the infectives at time lag $\tau = 1$.

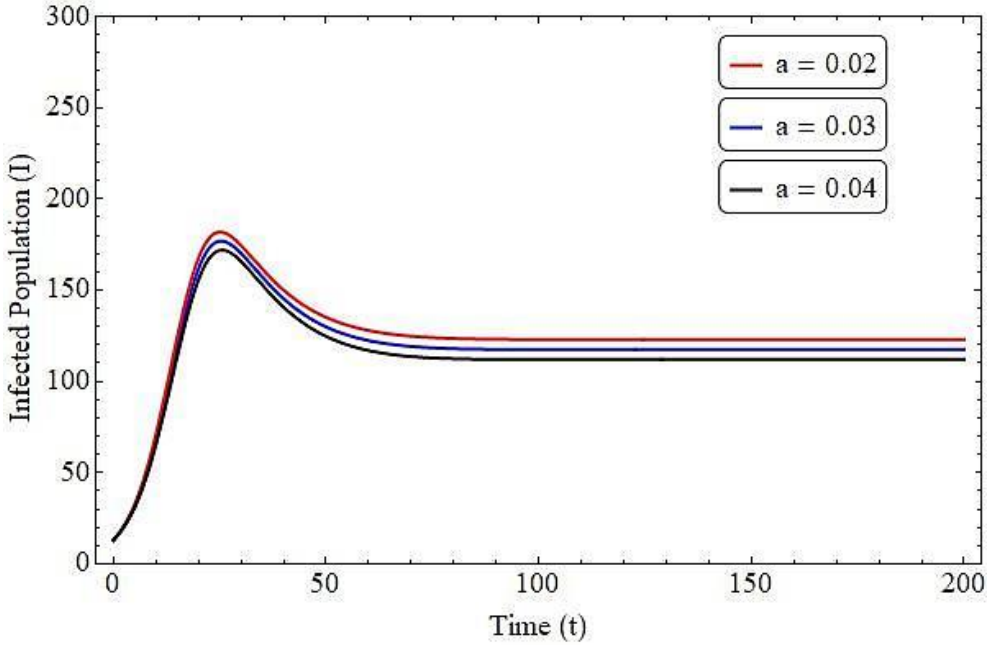


Fig. 7.10: Infected population (I) at increased values of cure rate (a) at time lag $\tau = 1$.

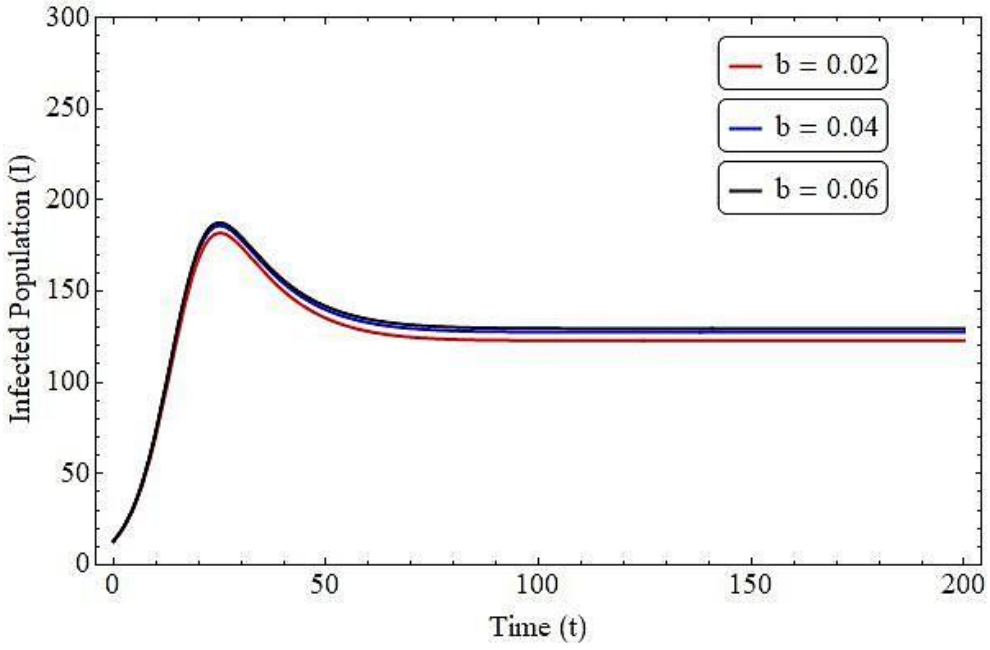


Fig. 7.11: Infected population (I) at increased values of limitation rate (b) in treatment availability at time lag $\tau = 1$.

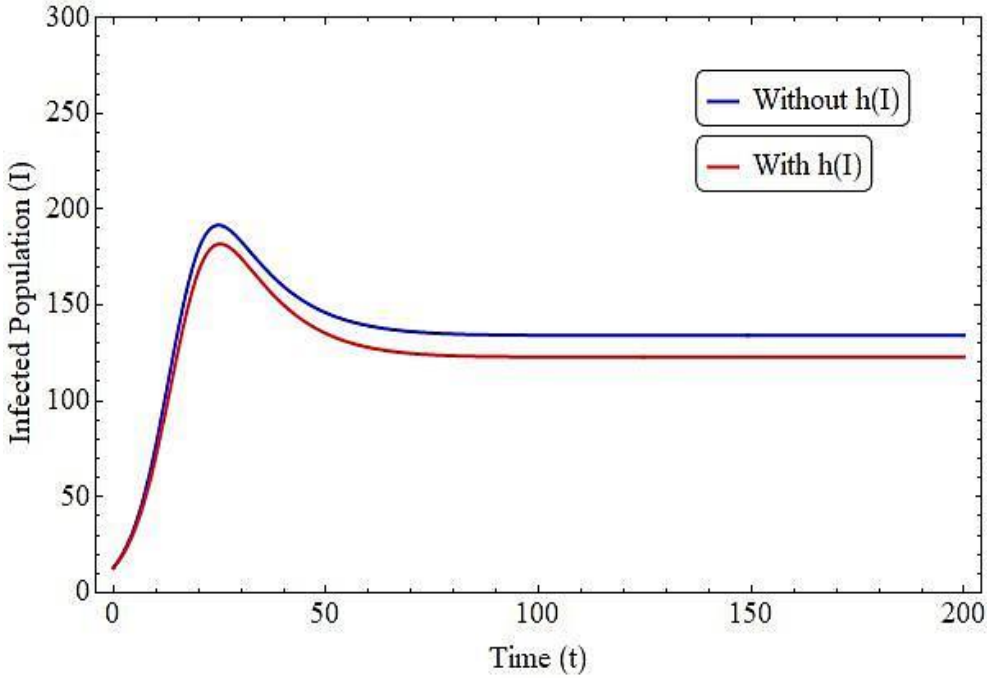


Fig. 7.12: Infected population (I) with and without Holling type II treatment rate.

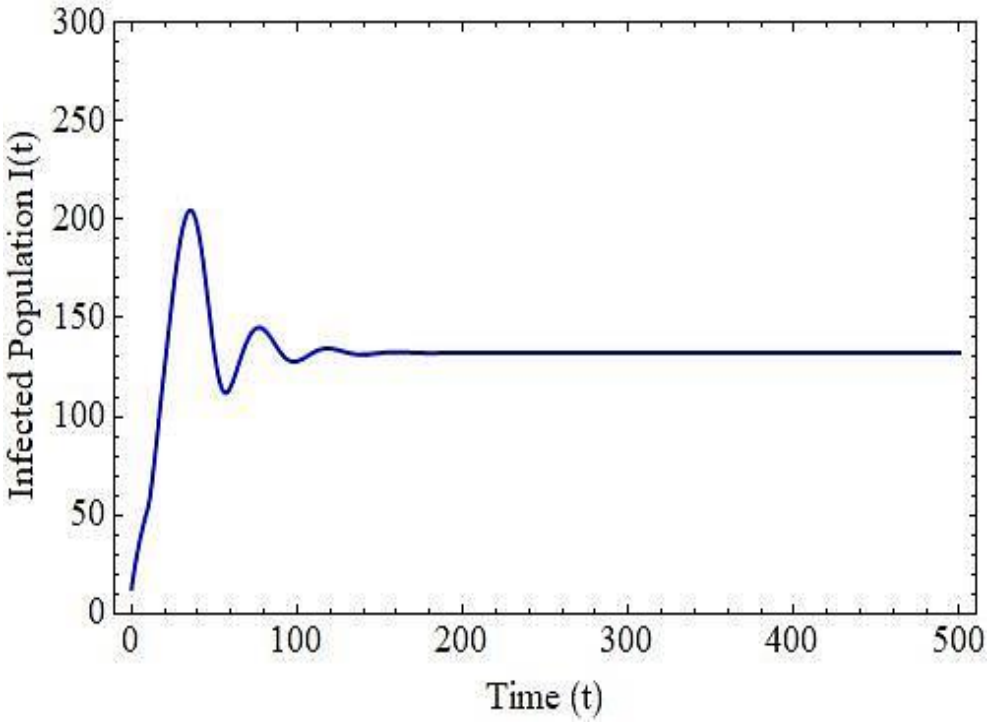


Fig. 7.13: Oscillatory graph of the infected population (I) at $A = 12, \beta = 0.00585, \gamma = 0.02, \tau = 9.5$.

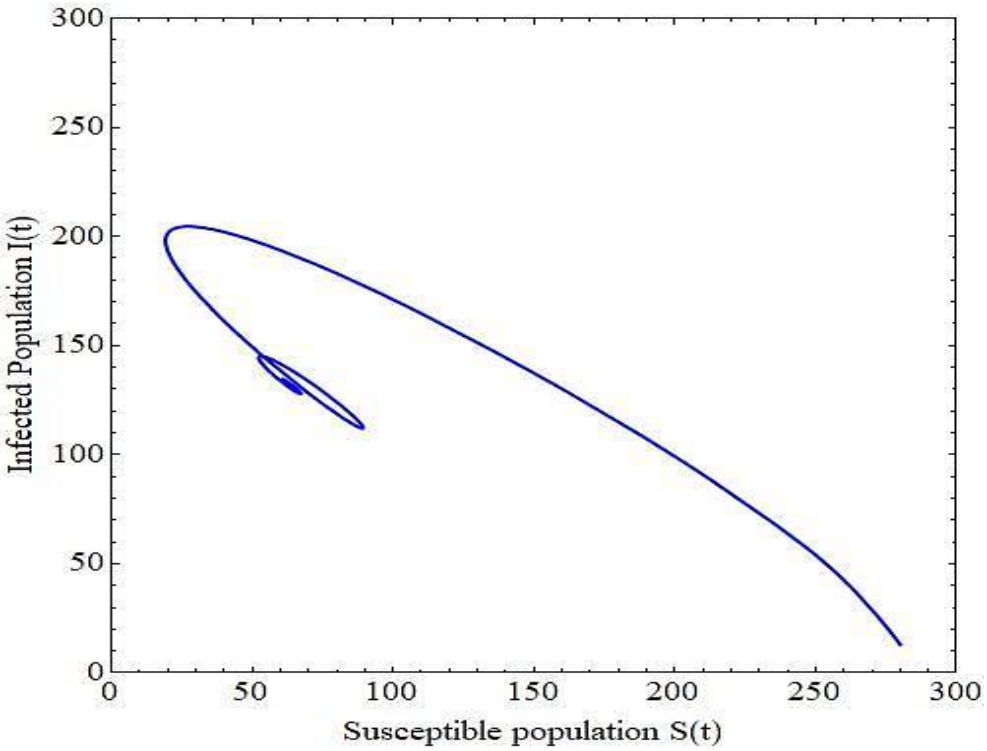


Fig. 7.14 (a): Population in $S-I$ plane at $A=12, \beta=0.00585, \gamma=0.02, \tau=9.5$.

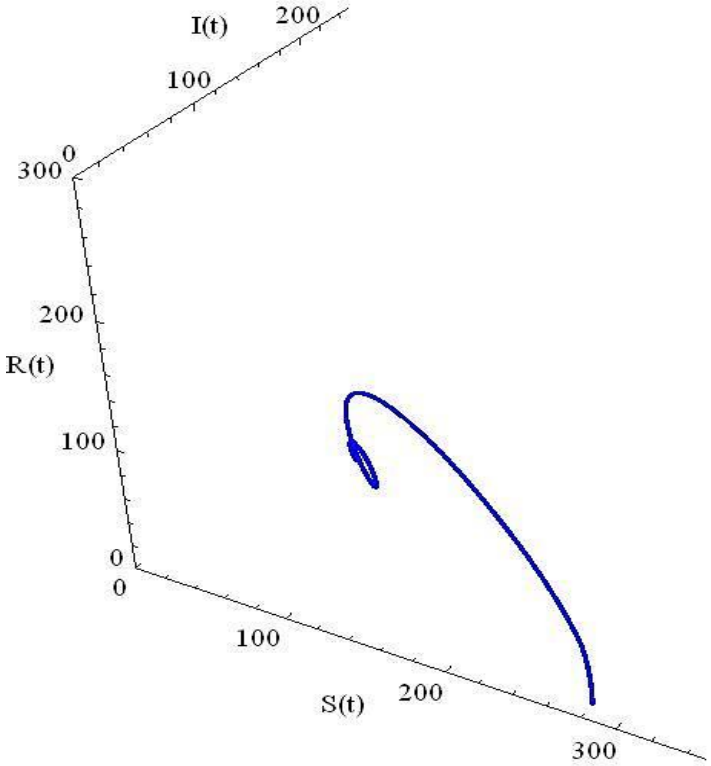


Fig. 7.14 (b): Population in $S-I-R$ plane at $A=12, \beta=0.00585, \gamma=0.02, \tau=9.5$.

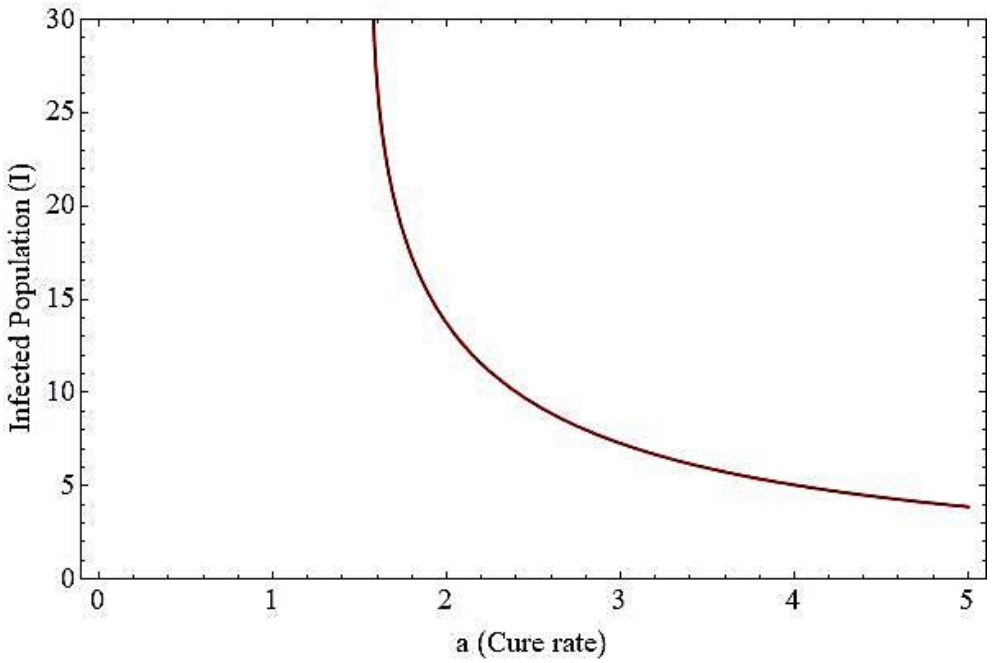


Fig. 7.15: Infected population (I) versus cure rate (a) graph.

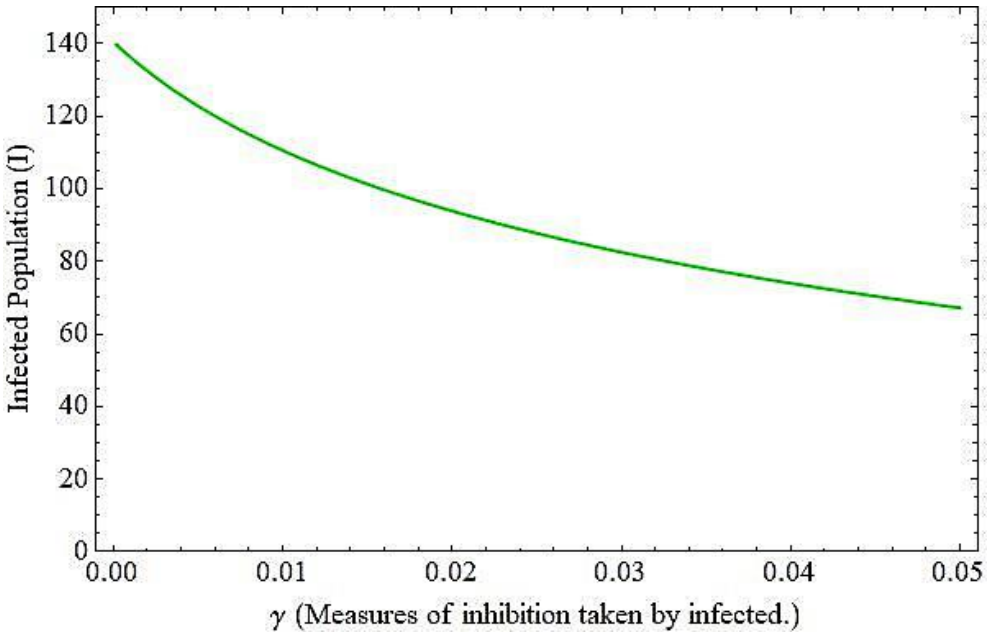


Fig. 7.16: Infected population (I) versus measures of inhibition (γ) due to the infectives**.**

CHAPTER 8

MATHEMATICAL AND NUMERICAL STUDY OF A SIR EPIDEMIC MODEL WITH THE INCLUSION OF ALERTNESS, INCUBATION PERIOD AND PRE & POST TREATMENT CLASSES

In this chapter, we present two different epidemic transmission models by incorporating different compartments according to disease status. Firstly, we present and analyze a susceptible-infected-recovered epidemic model by incorporating an alert individual's compartment along with the consideration of two explicit saturated incidence rates and Holling functional type II treatment rate. Awareness about the epidemic may play a significant role in the control of the spread of an epidemic. Hence, an alert compartment has been incorporated into the SIR model. It motivates us to take two incidence rates: one from the susceptible class to infected class and another from alert class to infected class. Holling functional type II treatment rate has been introduced to capture the effects of resource limitation in treating infectives. Secondly, we propose and analyze a time-delayed susceptible-infected-recovered epidemic model by introducing two explicit treatment classes (or compartments) along with nonlinear incidence rate. The treatment classes are named as a pre-treated class and post-treated class. The pre-treatment and post-treatment rates are being considered as Holling type I and Holling type III respectively. Long term qualitative analysis has been carried out after incorporating incubation time delay (τ) into the incidence rate. Further, we analyze both the models mathematically and obtain the model equilibria and discuss their local stability for both models separately. Finally, numerical simulations are presented to epitomize the analytical studies.

8.1 Introduction

In this chapter, we have proposed two different susceptible-infected-recovered (SIR) models with Holling type incidence rate and treatment rates. In the first model, we proposed a SIR model by incorporating an alert class along with Holling type II incidence rate and treatment rate. Alertness from the infection in the population always helps the health agencies to minimize the infection in society. When the population is alert about the infection, they always try to take precautionary measures which definitely reduces the spread of the disease. Therefore, an alert population compartment is incorporated into the SIR model. In this model two Holling functional type II incidence rates have been used: one from susceptible to infectives and second from alert individuals to infectives.

In the second model, a SIR model is proposed by incorporating two explicit treatment compartments along with Holling type incidence rates, time delay and Holling type III treatment rate. In this model, not all susceptible population who are in effective contact with infected individuals become infected. Some of them enter into the infected compartment but some of them are already vaccinated before symptoms of the disease are visible. On the other hand, individuals who have already entered into the infected compartment will be given treatment according to the severity of the disease. Therefore, to take into account these pre-treated and post-treated individuals, two compartments have been incorporated in the SIR model namely pre-treated compartment and post-treated compartment respectively. The incidence rate and treatment rates are considered as Holling functional type. The detailed explanation of Holling functional type incidence rate and Holling functional type II & III treatment rates have already been given in section 4.1 in chapter 4.

In this chapter, we propose and analyze both models separately. Further, we investigate the basic reproduction number R_0 for both models separately and analyze the dynamical behavior of the model's equilibria. The stability analysis of the equilibria for both models has been done using Descartes' rule of signs [Wang (2004)] with the Routh-Hurwitz criterion separately. Finally, models are simulated numerically to support our theoretical findings.

8.2 A susceptible-alert-infected-recovered (SAIR) model

To formulate the mathematical epidemic model, the total population $N(t)$ is divided into four compartments (or classes); susceptible individual class (S), alert individual class (A), infected individual class (I), and recovered individual class (R). Vulnerable (susceptible) people are those individuals who are healthy and can get the infection under appropriate conditions. Alert people are the individuals who know about the effect of disease, infection and symptoms and are taking necessary precautions to control the infection, and can get the infection only under adverse conditions. Infected people are the individuals who have caught the infection and can transmit it to susceptible and alert people via contacts. As time passes, via auto recuperation which could be due to the immune system reaction of the body or by treatment, the infected individuals lose infectivity and transfer to the recover class. We consider the Holling functional type II treatment rate $H(I)$ for the recovery of the infected people since it considers the cure rate along with the limitation rate in treatment availability which is more realistic. The transfer diagram of the epidemic for the various classes is shown by the block diagram in Fig. 8.1 below:

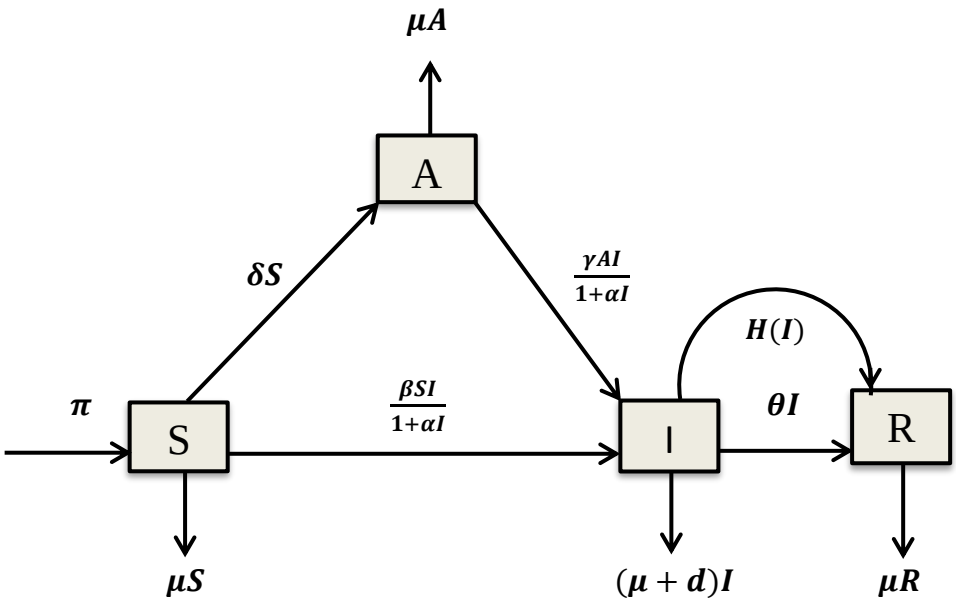


Fig. 8.1: Transfer diagram of the infection through various compartments for the model (8.1).

The proposed model is given by the following system of nonlinear ordinary differential equations:

$$\frac{dS}{dt} = \pi - \delta S - \mu S - \frac{\beta SI}{1+\alpha I},$$

$$\frac{dA}{dt} = \delta S - \mu A - \frac{\gamma AI}{1+\alpha I}, \tag{8.1}$$

$$\frac{dI}{dt} = \frac{\beta SI}{1+\alpha I} + \frac{\gamma AI}{1+\alpha I} - (\mu + d + \theta)I - \frac{aI}{1+bI},$$

$$\frac{dR}{dt} = \frac{aI}{1+bI} + \theta I - \mu R.$$

where, $S(0) > 0, A(0) \geq 0, I(0) \geq 0,$ and $R(0) \geq 0.$

The parameter π represents the constant recruitment rate of susceptible by birth or immigration. We consider that the susceptible individuals are moving into alert class at a rate δ, hence δ defines the rate of alertness of susceptible individuals. The incidence term $\frac{\beta SI}{1+\alpha I}$ represents the nonlinear saturation-limited rate (where β denotes the transmission rate of susceptible to infected individuals and α is the measure of inhibition taken by infectives), describing the rate of new infection when the susceptible individuals are becoming infected. μ is the natural mortality rate. The term $\frac{\gamma AI}{1+\alpha I}$ represents the incidence rate of alert population (where γ denotes the transmission rate of alert to infected individuals), describing the number of new infections when the alert individuals are becoming infected. The parameters d and θ represent the disease-induced death rate and recovery rate of the infected individuals, respectively. The term $H(I) = \frac{aI}{1+bI}$ represents the Holling functional type II treatment rate, where a is the cure rate and b is the rate, taking into account as resource limitations [Zhou and Fan (2012); Dubey *et al.* (2015)].

8.2.1 Basic properties of the model

Since the model (8.1) monitors the population, therefore for the biological reasons it is supposed that all the state variables are nonnegative and all the parameters of the model are positive.

For the model (8.1), we find that all the solutions with nonnegative initial data will remain non-negative and bounded for all time t. It can be observed as follows:

The total population $N(t)$ is

$$N(t) = S(t) + A(t) + I(t) + R(t).$$

The rate of change in the total population is

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dA(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt} = \pi - \mu N(t) - dI(t) \leq \pi - \mu N(t)$$

It implies that,

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t})$$

Thus, we get

$$\lim_{t\to\infty} N(t) \leq \frac{\pi}{\mu}.$$

Furthermore, $\frac{dN(t)}{dt} < 0$ if $N(t) > 0$. Thus, all the solutions of the model (8.1) tend to the positively invariant region $D = \left\{(S, A, I, R) \in \mathbb{R}_+^4 : 0 < S + A + I + R \leq \frac{\pi}{\mu}\right\}$. Hence, all the solutions of the model (8.1) are bounded and nonnegative. Thus, the model (8.1) is well-posed mathematically and epidemiologically.

Hence, we establish the following theorem:

Theorem 8.1: The set $D = \left\{(S, A, I, R) \in \mathbb{R}_+^4 : 0 < S + A + I + R \leq \frac{\pi}{\mu}\right\}$ is a positively invariant region of the Model (8.1).

Since the recovered population $R(t)$ does not feature in the first three equations of the model (8.1), without loss of generality, this equation can be omitted for theoretical analysis. Thus, we consider the following reduced system for the analysis:

$\frac{dS}{dt} = \pi - \delta S - \mu S - \frac{\beta SI}{1+\alpha I}$,

$$\frac{dA}{dt} = \delta S - \mu A - \frac{\gamma AI}{1+\alpha I}, \tag{8.2}$$

$$\frac{dI}{dt} = \frac{\beta SI}{1+\alpha I} + \frac{\gamma AI}{1+\alpha I} - (\mu + d + \theta)I - \frac{aI}{1+bI}.$$

In the next section, we obtain the equilibria of the system (8.2) and discuss their stability behavior.

8.2.2 Equilibria and their stability analysis

The system (8.2) has two equilibria which are obtained by equating the derivatives of the system (8.2) to zero. These are as follows:

i. Disease-free equilibrium (DFE) $Q\left(\frac{\pi}{\mu+\delta}, \frac{\delta\pi}{\mu(\mu+\delta)}, 0\right)$.

ii. Endemic (Positive) equilibrium (EE) $Q^*(S^*, A^*, I^*)$.

To investigate the stability of the system (8.2) at obtained equilibria, we first find the linearized matrix. For this, we assume that $F = (F_1, F_2, F_3)^T$, where F_1, F_2 and F_3 represent the right-hand sides of the system (8.2) respectively. Furthermore, let $X = (S, A, I)^T$. Then, the linearized matrix for the system (8.2) is obtained as follows:

$$J = \begin{pmatrix} \left(-\delta - \mu - \frac{\beta I}{1+\alpha I}\right) & 0 & -\frac{\beta S}{(1+\alpha I)^2} \\ \delta & \left(-\mu - \frac{\gamma I}{1+\alpha I}\right) & -\frac{\gamma A}{(1+\alpha I)^2} \\ \frac{\beta I}{1+\alpha I} & \frac{\gamma I}{1+\alpha I} & \left(\frac{\beta S}{(1+\alpha I)^2} + \frac{\gamma A}{(1+\alpha I)^2} - d - \mu - \theta - \frac{a}{(1+bI)^2}\right) \end{pmatrix}.$$

Now, we will obtain the threshold parameter R_0, known as the basic reproduction number, which determines the stability of the equilibria.

8.2.2.1 Computation of the basic reproduction number (R_0)

The characteristic equation of the matrix J in λ at DFE(Q) is given by

$$(\mu + \lambda)(\mu + \delta + \lambda)[\lambda - (d + \mu + \theta + a)(R_0 - 1)] = 0 \tag{8.3}$$

The characteristic equation (8.3) has three roots $\lambda_1 = -\mu, \lambda_2 = -(\mu + \delta)$, and $\lambda_3 = (d + \mu + \theta + a)(R_0 - 1)$.

where,

$$R_0 = \frac{\pi(\mu\beta + \gamma\delta)}{\mu(\mu + \delta)(d + \mu + \theta + a)}.$$

The term R_0 is known as the basic reproduction number for the model.

8.2.2.2 Stability analysis of disease-free equilibrium

In this subsection, we analyze the stability of the disease-free equilibrium (Q).

8.2.2.2.1 Analysis for $R_0 \neq 1$

From Eq. (8.3) we note that the matrix J evaluated at Q has two negative eigenvalues $\lambda_1 = -\mu$ and $\lambda_2 = -\mu - \delta$, and the sign of third eigenvalue λ_3 depends on the basic reproduction number R_0. λ_3 is negative if and only if $R_0 < 1$. Thus, with the help of the Routh-Hurwitz criterion, we propose the following theorem:

Theorem 8.2: The DFE $Q\left(\frac{\pi}{\mu+\delta}, \frac{\delta\pi}{\mu(\mu+\delta)}, 0\right)$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

8.2.2.2.2 Analysis at $R_0 = 1$

We notice that the matrix J of the system (8.2) is being evaluated at $R_0 = 1$ and bifurcation parameter $\beta = \beta^* = \left(\frac{(\delta+\mu)(d+\mu+\theta+a)}{\pi} - \frac{\gamma\delta}{\mu}\right)$ has a simple zero eigenvalue and the other eigenvalues are negative. Therefore, the disease-free equilibrium Q is a non-hyperbolic equilibrium when $R_0 = 1$. To analyze the behavior of the system (8.2) for the basic reproduction number R_0 equals to one, we use the bifurcation theory approach which is based on the center manifold theory [Sastry (1999); Dubey *et al.* (2015)]. Through this approach, we are interested to assess if there is a stable coexistence

equilibrium bifurcating from Q, and Q changes from being stable to unstable. This behavior is known as transcritical bifurcation [Chavez and Song (2004); Buonomo and Cerasuolo (2015)]. For the analysis, we assume that $S = x_1, A = x_2$ and $I = x_3$ then the system (8.2) can be rewritten as

$$\frac{dx_1}{dt} = \pi - \delta x_1 - \mu x_1 - \frac{\beta x_1 x_3}{1+\alpha x_3} \equiv f_1,$$
$$\frac{dx_2}{dt} = \delta x_1 - \mu x_2 - \frac{\gamma x_2 x_3}{1+\alpha x_3} \equiv f_2, \tag{8.4}$$
$$\frac{dx_3}{dt} = \frac{\beta x_1 x_3}{1+\alpha x_3} + \frac{\gamma x_2 x_3}{1+\alpha x_3} - (\mu + d + \theta)x_3 - \frac{a x_3}{1+b x_3} \equiv f_3.$$

The Jacobian matrix J^* evaluated at $R_0 = 1$ and $\beta = \beta^*$ is obtained as

$$J^* = \begin{pmatrix} (-\delta - \mu) & 0 & -\dfrac{\beta^*\pi}{(\mu+\delta)} \\ \delta & -\mu & -\dfrac{\gamma\delta\pi}{\mu(\mu+\delta)} \\ 0 & 0 & 0 \end{pmatrix}.$$

Let $u = [u_1, u_2, u_3\,]$ denotes the left eigenvector and $w = [w_1, w_2, w_3\,]^T$ denotes the right eigenvector of J^*corresponding to the zero eigenvalue. Then, we obtain that

$$u_1 = 0, u_2 = 0, u_3 = 1 \text{ and } w_1 = -\frac{\beta^*\pi}{(\delta+\mu)^2}, w_2 = -\frac{\delta\pi(\gamma\delta + \gamma\mu + \mu\beta^*)}{\mu^2(\delta+\mu)^2}, w_3 = 1.$$

The non-zero partial derivatives associated with the functions $f_1, f_2,$ and f_3 of the system (8.4) calculated at $R_0 = 1$ and $\beta = \beta^*$ are

$$\left(\frac{\partial^2 f_3}{\partial x_1 \partial x_3}\right)_Q = \beta^*, \left(\frac{\partial^2 f_3}{\partial x_2 \partial x_3}\right)_Q = \gamma\,, \left(\frac{\partial^2 f_3}{\partial x_3 \partial x_1}\right)_Q = \beta^*,$$
$$\left(\frac{\partial^2 f_3}{\partial x_3 \partial x_2}\right)_Q = \gamma\,, \left(\frac{\partial^2 f_3}{\partial {x_3}^2}\right)_Q = -\frac{2\alpha\pi}{\mu(\mu+\delta)}(\mu\beta^* + \gamma\delta) + 2ab, \text{ and } \left(\frac{\partial^2 f_3}{\partial x_3 \partial \beta^*}\right)_Q = \frac{\pi}{(\delta+\mu)}.$$

From [Chavez and Song (2004); Buonomo and Cerasuolo (2015)], we obtain the bifurcation constants $a_1$and b_1 as given below:

$$a_1 = \sum_{k,i,j=1}^{3} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q$$
$$= u_3\left(w_1 w_3 \beta^* + w_2 w_3 \gamma + w_3 w_1 \beta^* + w_3 w_2 \gamma + {w_3}^2\left(2ab - \frac{2\alpha\pi}{\mu(\mu+\delta)}(\mu\beta^* + \gamma\delta)\right)\right)$$

$$= -\left(2\frac{\beta^{*2}\pi}{(\delta+\mu)^2} + 2\frac{\delta\gamma\pi(\gamma\delta+\gamma\mu+\mu\beta^*)}{\mu^2(\delta+\mu)^2} + \frac{2\alpha\pi}{\mu(\delta+\mu)}(\mu\beta^* + \gamma\delta)\right) + 2ab$$

$$b_1 = \sum_{k,i=1}^{3} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q$$

$$= w_3\left(\frac{\pi}{(\delta+\mu)}\right)$$

$$= \frac{\pi}{(\delta+\mu)} > 0.$$

Thus, according to sign of a_1, we propose the following theorem:

Theorem 8.3: The DFE $Q\left(\frac{\pi}{\mu+\delta}, \frac{\delta\pi}{\mu(\mu+\delta)}, 0\right)$ either exhibits a forward or backward bifurcation at $R_0 = 1$.

8.2.2.3 Existence and stability analysis of endemic equilibrium

To obtain the conditions for the existence of the endemic equilibrium $Q^*(S^*,\ A^*, I^*)$, the system (8.2) is rearranged to get S^*, A^* and I^* which gives:

$S^* = \frac{\pi(1+\alpha I^*)}{(\delta+\mu)(1+\alpha I^*)+\beta I^*}$, $A^* = \frac{\delta S^*(1+\alpha I^*)}{\mu(1+\alpha I^*)+\gamma I^*}$,

and I^* is given by the cubic equation

$$C_4 I^{*3} + C_3 I^{*2} + C_2 I^* + C_1 = 0 \tag{8.5}$$

where,

$C_1 = \mu(\delta+\mu)(a+d+\theta+\mu) - \pi(\gamma\delta+\beta\mu) = \mu(\delta+\mu)(a+d+\theta+\mu)(1-R_0)$,

$C_2 = -\pi((b+\alpha)\gamma\delta + \beta(\gamma+(b+\alpha)\mu)) + a(\gamma(\delta+\mu) + \mu(\beta+2\alpha(\delta+\mu))) + (d+\theta+\mu)(\gamma(\delta+\mu)+\mu(\beta+(b+2\alpha)(\delta+\mu)))$,

$C_3 = a(\gamma+\alpha\mu)(\beta+\alpha(\delta+\mu)) - \pi b(\alpha\gamma\delta+\beta(\gamma+\alpha\mu)) + (d+\theta+\mu)(\beta(\gamma+(b+\alpha)\mu) + (\delta+\mu)(\alpha(\gamma+\alpha\mu)+b(\gamma+2\alpha\mu)))$,

$C_4 = b(d+\theta+\mu)(\gamma+\alpha\mu)\big(\beta+\alpha(\delta+\mu)\big)$.

After simplifying the coefficients C_2 and C_3, we get that

$C_2 = -\pi\big((b+\alpha)\gamma\delta + \beta(\gamma+(b+\alpha)\mu)\big) + a\big(\gamma n + \mu(\beta+2\alpha n)\big) + m\big(\gamma n + \mu(\beta+(b+2\alpha)n)\big)$,

$$C_3 = -\pi b\big(\alpha\gamma\delta + \beta(\gamma + \alpha\mu)\big) + a(\gamma + \alpha\mu)(\beta + \alpha n) + m\Big(\beta(\gamma + (b + \alpha)\mu) + n\big((b + \alpha)\gamma + \alpha(2b + \alpha)\mu\big)\Big),$$

where, $m = d + \theta + \mu$, and $n = \delta + \mu$.

Using Descartes' rule of the signs, for $R_0 > 1$, there exists a unique positive real root I^* of Eq. (8.5) if any of the following conditions holds true:

i. $C_1 < 0, C_2 > 0, C_3 > 0, C_4 > 0.$

ii. $C_1 < 0, C_2 < 0, C_3 > 0, C_4 > 0.$

iii. $C_1 < 0, C_2 < 0, C_3 < 0, C_4 > 0.$

Here, C_1 is negative for $R_0 > 1$, and C_4 is always positive whereas the coefficients C_2, and C_3 are positive or negative under the conditions given below:

$$C_2 \begin{cases} > 0 \text{ when } \pi\big((b + \alpha)\gamma\delta < \beta(\gamma + (b + \alpha)\mu)\big) + a\big(\gamma n + \mu(\beta + 2\alpha n)\big) + m\big(\gamma n + \mu(\beta + (b + 2\alpha)n)\big). \\ < 0 \text{ when } \pi\big((b + \alpha)\gamma\delta > \beta(\gamma + (b + \alpha)\mu)\big) + a\big(\gamma n + \mu(\beta + 2\alpha n)\big) + m\big(\gamma n + \mu(\beta + (b + 2\alpha)n)\big). \end{cases}$$

and

$$C_3 \begin{cases} > 0 \text{ when } \pi b\big(\alpha\gamma\delta + \beta(\gamma + \alpha\mu)\big) < a(\gamma + \alpha\mu)(\beta + \alpha n) + m\Big(\beta(\gamma + (b + \alpha)\mu) + n\big((b + \alpha)\gamma + \alpha(2b + \alpha)\mu\big)\Big). \\ < 0 \text{ when } \pi b\big(\alpha\gamma\delta + \beta(\gamma + \alpha\mu)\big) > a(\gamma + \alpha\mu)(\beta + \alpha n) + m\Big(\beta(\gamma + (b + \alpha)\mu) + n\big((b + \alpha)\gamma + \alpha(2b + \alpha)\mu\big)\Big). \end{cases}$$

After getting the value of I^* we can obtain the values of S^* and A^*. It indicates the existence of a unique positive equilibrium $Q^*(S^*, A^*, I^*)$ if any one of the above conditions is satisfied.

The local stability analysis of the endemic equilibrium Q^* is explored as follows: The characteristic equation of the system (8.2) at Q^* is a third-degree polynomial:

$$\lambda^3 + p_0\lambda^2 + p_1\lambda + p_2 = 0, \tag{8.6}$$

where,

$$p_0 = \delta + 2\mu + \frac{(\beta+\gamma)I^*}{1+\alpha I^*} + \left(\frac{a}{(1+bI^*)^2} + \mu + d + \theta - \frac{(\beta S^* + \gamma A^*)}{(1+\alpha I^*)^2}\right),$$

$$p_1 = \left(\delta + \mu + \frac{\beta I^*}{1+\alpha I^*}\right)\left(\mu + \frac{\gamma I^*}{1+\alpha I^*}\right) + \frac{(\beta^2 S^* + \gamma^2 A^*)I^*}{(1+\alpha I^*)^3} + \left(\delta + 2\mu + \frac{(\beta+\gamma)I^*}{1+\alpha I^*}\right)\left(\frac{a}{(1+bI^*)^2} + \mu + d + \theta - \frac{(\beta S^* + \gamma A^*)}{(1+\alpha I^*)^2}\right),$$

$$p_2 = \left(\delta + \mu + \frac{\beta I^*}{1+\alpha I^*}\right)\left(\mu + \frac{\gamma I^*}{1+\alpha I^*}\right)\left(\frac{a}{(1+bI^*)^2} + \mu + d + \theta - \frac{(\beta S^* + \gamma A^*)}{(1+\alpha I^*)^2}\right) + \frac{\gamma^2 A^* I^*}{(1+\alpha I^*)^3}\left(\delta + \mu + \frac{\beta I^*}{1+\alpha I^*}\right) + \frac{\beta\delta\gamma S^* I^*}{(1+\alpha I^*)^3} + \frac{\beta S^* I^*}{(1+\alpha I^*)^3}\left(\mu + \frac{\gamma I^*}{1+\alpha I^*}\right).$$

Theorem 8.4: The endemic equilibrium Q^* is locally asymptotically stable when $\frac{(\beta S^* + \gamma A^*)}{(1+\alpha I^*)^2} \leq \frac{a}{(1+bI^*)^2} + \mu + d + \theta$.

Proof: From Eq. (8.6), it is clear that the coefficients p_0, p_1 and p_2 of λ^2, λ and 1, respectively, are positive if $\frac{(\beta S^* + \gamma A^*)}{(1+\alpha I^*)^2} \leq \frac{a}{(1+bI^*)^2} + \mu + d + \theta$. Therefore, by the Descartes' rule of the signs [Wang 2004], it is clear that all the eigenvalues of Eq. (8.6) are negative. Hence, by the definition of the Routh-Hurwitz criterion Q^* is locally asymptotically stable when the condition $\frac{(\beta S^* + \gamma A^*)}{(1+\alpha I^*)^2} \leq \frac{a}{(1+bI^*)^2} + \mu + d + \theta$ is satisfied.

8.3 A susceptible−pre-treated−infected−post treated−recovered model

We propose a mathematical model for epidemic through compartments (or classes). For this, we consider the total population $N(t)$ at time t, with the immigration of susceptible individuals with a constant rate A. We divided the total population into five compartments: susceptible (S) compartment, pre-treated (T_1) compartment, infected (I) compartment, post-treated (T_2) compartment and recovered (R) compartment. Susceptible are those who can get a disease under appropriate conditions, pre-treated are those who are vaccinated before the infection and can get a disease under adverse conditions, infected are those who have contracted the disease and can spread the disease to susceptible and pre – treated individuals under suitable conditions, post-treated are those who are infected and taking the medical treatments and recovered are those who are immunized after the medical treatment and are free from the effect of disease. The pre-treatment and post – treatment of individuals are given by the Holling type I and Holling type III treatment rates respectively. It has been considered that treatment before getting infected will be given in a linear form; therefore, Holling type I functional response has been considered for the treatment rate in the pre-treated class. The treatment given to infected individuals will be more than a linearly increasing function because of the limitation to the treatment availability which is well explained by Holling type III

functional response and therefore Holling type III treatment rate will be considered for the treatment of infected individuals. Incubation time delay has been incorporated as a time delay in force of infection and inhibition effect which form nonlinear incidence rate. This time delay ($\tau > 0$) will be a fixed time during which the infectious agents develop in the vector and it is only after this time that the infected vector can infect a susceptible individual. Let μ be the natural death rate of the population, d the death rate due to the disease and θ the recovery rate of post - treated individuals. The progression of the epidemic in different compartments is shown by the block diagram as given in Fig 8.2 below.

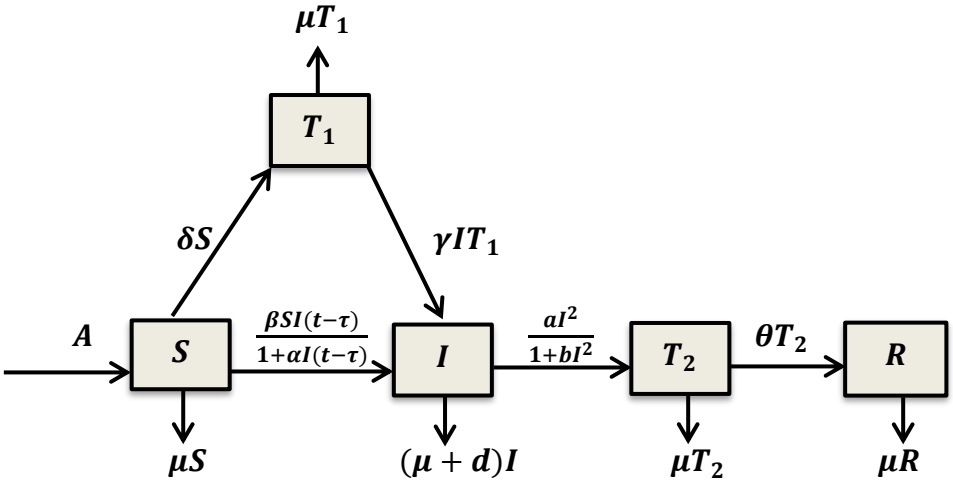


Fig 8.2: Progression of infection from susceptible (S) and pre-treated (T_1) compartments through infected (I), post treated (T_2) and recovered (R) compartments.

The rate of change in each compartment is given by the following system of nonlinear delay differential equations:

$$\frac{ds(t)}{dt} = A - (\mu + \delta)S(t) - \frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)},$$

$$\frac{dT_1(t)}{dt} = \delta S(t) - \mu T_1(t) - \gamma I(t)T_1(t),$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)} + \gamma I(t)T_1(t) - (\mu + d)I(t) - \frac{aI^2(t)}{1+bI^2(t)}, \quad (8.7)$$

$$\frac{dT_2(t)}{dt} = \frac{aI^2(t)}{1+bI^2(t)} - (\mu + \theta)T_2(t),$$

$$\frac{dR(t)}{dt} = \theta T_2(t) - \mu R(t).$$

The initial conditions of the model are given by

$$S(\theta) = \varphi_1(\theta),\ T_1(\theta) = \varphi_2(\theta), I(\theta) = \varphi_3(\theta), T_2(\theta) = \varphi_4(\theta), R(\theta) = \varphi_5(\theta),$$
$$\varphi_i(\theta) \geq 0, \theta \in [-\tau, 0], \varphi_i(0) > 0\ (i = 1,2,3,4,5) \quad (8.8)$$

where $\left(\varphi_1(\theta), \varphi_2(\theta), \varphi_3(\theta), \varphi_4(\theta), \varphi_5(\theta)\right) \in C([-\tau, 0], \mathbb{R}_+^5)$. Here C denotes the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}_+^5$.

The term δS represents the Holling type I treatment rate, where, δ is defined as pre-treatment rate and the term $\frac{aI^2}{1+bI^2}$ represents the Holling type III treatment rate, where, both a and b are positive constants defined as cure rate of the infected individuals and limitation rate in treatment availability [Dubey et al. 2016] respectively. The term $\frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)}$ represents the Holling functional type II incidence rate, where, β is the transmission rate of infection in susceptible individuals and α is the measure of inhibition effect due to the infected individuals. The term $\gamma I(t)T_1(t)$ in the model, represent the bilinear incidence rate of infection between pre-treated and infected individuals, where, γ is the transmission rate of infection in pre-treated individuals. The incidence rate $\frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)}$ represents the rate at the time $(t-\tau)$ at which susceptible individuals leave the susceptible class and enter into the infectious class at time *t*. Furthermore, it is assumed that all other parameters of the model are positive which is also biologically true.

It should be mentioned that although the recovered population continues to make contacts with other individuals of the population, it does not contribute to the transmission dynamics of the disease. Since, the recovered population R, does not feature in the first four equations of the model, therefore, without loss of generality we consider the following reduce system for theoretical analysis:

$$\frac{dS(t)}{dt} = A - (\mu + \delta)S(t) - \frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)},$$

$$\frac{dT_1(t)}{dt} = \delta S(t) - \mu T_1(t) - \gamma I(t)T_1(t),$$

$$\frac{dI(t)}{dt} = \frac{\beta S(t)I(t-\tau)}{1+\alpha I(t-\tau)} + \gamma I(t)T_1(t) - (\mu + d)I(t) - \frac{aI^2(t)}{1+bI^2(t)}, \quad (8.9)$$

$\frac{dT_2(t)}{dt} = \frac{aI^2(t)}{1+bI^2(t)} - (\mu + \theta)T_2(t).$

8.3.1 Basic properties of the model

It is assumed that all state variables of the model are positive i.e. $(S, T_1, I, T_2) \in \mathbb{R}^4_+$. It follows that nonnegative cone $\mathbb{R}^4_+$ is invariant, as the disease-free plane ($I = T_2 = 0$).
It is convenient to define $N_0 = \frac{A}{\mu}$, $S_0 = \frac{A}{(\mu+\delta)}$ and $T_{1_0} = \frac{\delta A}{\mu(\mu+\delta)}$.

The rate of change of the total population of the model (8.7) is given by

$$\frac{dN}{dt} = A - \mu N - dI \leq A - \mu N \tag{8.10}$$

with $N = S + T_1 + I + T_2 + R$.

Since as $t \to \infty$, the disease will disappear, therefore $\underset{t\to\infty}{\text{Lim}\,sup}\, I(t) = 0$

Eq. (8.10) $\Longrightarrow \frac{dN}{dt} = A - \mu N$

$\Longrightarrow N(t) = \frac{A}{\mu} + \left(N(0) - \frac{A}{\mu}\right) e^{-\mu t}$

$\Longrightarrow \lim_{t\to\infty} N(t) = \frac{A}{\mu} = N_0$

This follows that

$0 < \underset{t\to\infty}{\text{Lim}\,sup} N(t) \leq N_0$ if and only if $\underset{t\to\infty}{\text{Lim}\,sup}\, I(t) = 0$

From the first equation of the model, it follows that

$$0 < \underset{t\to\infty}{\text{Lim}\,sup} S(t) \leq S_0 \tag{8.11}$$

and then from the second equation of system (8.9),

$$0 < \underset{t\to\infty}{\text{Lim}\,sup} T_1(t) \leq T_{1_0}. \tag{8.12}$$

From the Eq. (8.10) it follows that if $N > N_0$ then $\frac{dN}{dt} < 0$. This establishes the following theorem:

Theorem 8.5: The set $D = \{(S, T_1, I, T_2) \in \mathbb{R}_+^4 : S + T_1 + I + T_2 \leq N_0, S \leq S_0, T_1 \leq T_{1_0}\}$ is a positively invariant and attracting region for the disease transmission system (8.9) with initial conditions in $\mathbb{R}_+^4$.

In the absence of disease (I = 0), the total population N approaches the carrying capacity N_0 asymptotically, and in the presence of disease, the total population is less than or equals to N_0.

Thus, every solution of the system (8.9) with initial conditions in $\mathbb{R}_+^4$ tends towards D as $t \to \infty$, and every solution with an initial condition in D remains there for $t > 0$. Furthermore, in D, the usual existence, uniqueness and continuation results hold for the system so that the system (8.9) is well posed mathematically and biologically.

8.3.2 Equilibria and their stability analysis

In this subsection, we obtain the model equilibria and discuss their stability behavior.

8.3.2.1 Disease-free equilibrium and its stability analysis

The system (8.9) has a unique disease-free equilibrium of the form $Q\left(\frac{A}{\mu+\delta}, \frac{\delta A}{\mu(\mu+\delta)}, 0, 0\right)$.

The characteristic equation at $Q\left(\frac{A}{\mu+\delta}, \frac{\delta A}{\mu(\mu+\delta)}, 0, 0\right)$ of the system (8.9) is given by

$$(\lambda + \mu)(\lambda + \mu + \theta)(\lambda + \mu + \delta)\left(\lambda + (\mu + d)\left(1 - \frac{A(\gamma\delta + \beta\mu e^{-\lambda\tau})}{\mu(\mu+\delta)(\mu+d)}\right)\right) = 0 \qquad (8.13)$$

At $\tau = 0$ the term $\frac{A(\gamma\delta+\beta\mu e^{-\lambda\tau})}{\mu(\mu+\delta)(\mu+d)}$ is known as a basic reproduction number R_0. Therefore, we define the basic reproduction number R_0 of our model by $\frac{A(\gamma\delta+\beta\mu)}{\mu(\mu+\delta)(\mu+d)}$.

8.3.2.1.1 Analysis for $R_0 \neq 1$

Eq. (8.13) always has three negative roots $\lambda_1 = -\mu,\ \lambda_2 = -(\mu+\theta),\ \lambda_3 = -(\mu+\delta)$ and one other root is determined by the solution of the equation

$$\lambda + \mu + d - \frac{A(\gamma\delta+\beta\mu e^{-\lambda\tau})}{\mu(\mu+\delta)} = 0.$$

Let

$$f(\lambda) = \lambda + \mu + d - \frac{A(\gamma\delta+\beta\mu e^{-\lambda\tau})}{\mu(\mu+\delta)}$$

If $R_0 > 1$, for real λ,

$$f(0) = \mu + d - \frac{A(\gamma\delta+\beta\mu)}{\mu(\mu+\delta)} < 0,\ \lim_{\lambda\to\infty} f(\lambda) \to +\infty.$$

Hence, $f(\lambda) = 0$ has a positive real root if $R_0 > 1$.

If $R_0 < 1$, we assume that $Re\ \lambda \geq 0$.

We notice that

$$Re\ \lambda = \frac{A(\gamma\delta+\beta\mu e^{-Re\,\lambda\,\tau}\cos Im\,\lambda\,\tau\ )}{\mu(\mu+\delta)} - (\mu+d) < \frac{A(\gamma\delta+\beta\mu)}{\mu(\mu+\delta)} - (\mu+d) < 0.$$

a contradiction to our assumption. Hence, if $R_0 < 1$ then the root λ of Eq. (8.13) has a negative real part.

Hence, we state the following theorem:

Theorem 8.6: DFE $Q\left(\frac{A}{\mu+\delta}, \frac{\delta A}{\mu(\mu+\delta)}, 0,0\right)$ of the system (8.9) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ for $\tau \geq 0$.

8.3.2.1.2 Analysis at $R_0 = 1$

This section is to analyze the behavior of the system (8.9) when $R_0 = 1$. The Jacobian matrix of the system (8.9) is being obtained at $R_0 = 1$ and bifurcation parameter $\beta = \beta^* = \left(\frac{\mu(\delta+\mu)(d+\mu)-\gamma\delta A}{A\mu}\right)$ has one of the eigenvalue as zero and the remaining eigenvalues are negative. The stability behavior of equilibrium points at $R_0 = 1$ cannot be determined

using linearization, so we apply center manifold theory [Sastry (1999)]. For this, we consider that $S = x_1, T_1 = x_2, I = x_3$ and $T_2 = x_4$ then the system (8.9) can be rewritten as

$$\frac{dx_1}{dt} = A - \delta x_1 - \mu x_1 - \frac{\beta x_1 x_3}{1+\alpha x_3} \equiv f_1,$$
$$\frac{dx_2}{dt} = \delta x_1 - \mu x_2 - \gamma x_2 x_3 \equiv f_2,$$
$$\frac{dx_3}{dt} = \frac{\beta x_1 x_3}{1+\alpha x_3} + \gamma x_2 x_3 - (\mu + d)x_3 - \frac{a{x_3}^2}{1+b{x_3}^2} \equiv f_3, \quad (8.14)$$
$$\frac{dx_4}{dt} = \frac{a{x_3}^2}{1+b{x_3}^2} - (\theta + \mu)x_4 \equiv f_4.$$

Let J^* be the Jacobian matrix evaluated at $R_0 = 1$ and $\beta = \beta^*$. Also, let $u = [u_1, u_2, u_3, u_4]$ and $w = [w_1, w_2, w_3, w_4]^T$ be the left eigenvector and right eigenvector of J^*associated with the zero eigenvalue.

$$J^* = \begin{pmatrix} -\mu - \delta & 0 & -\beta^* A/(\mu + \delta) & 0 \\ \delta & -\mu & -\gamma\delta A/\mu(\mu + \delta) & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mu - \theta \end{pmatrix}$$

Then, we get

$u_1 = 0, u_2 = 0, u_3 = 1, u_4 = 0$ and $w_1 = -\frac{\beta^* A}{(\delta+\mu)^2}, w_2 = -\frac{(\gamma\delta A\mu + \gamma A\delta^2 + \mu\delta A\beta^*)}{\mu^2(\delta+\mu)^2}, w_3 = 1, w_4 = 0.$

The non-zero partial derivatives corresponding to the functions of the system (8.14) evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$\left(\frac{\partial^2 f_3}{\partial x_1 \partial x_3}\right)_Q = \left(\frac{\partial^2 f_3}{\partial x_3 \partial x_1}\right)_Q = \beta^*$, $\left(\frac{\partial^2 f_3}{\partial x_2 \partial x_3}\right)_Q = \left(\frac{\partial^2 f_3}{\partial x_3 \partial x_2}\right)_Q = \gamma$, $\left(\frac{\partial^2 f_3}{\partial {x_3}^2}\right)_Q = -2\left(\frac{A\alpha\beta^*}{\mu+\delta} + a\right)$ and $\left(\frac{\partial^2 f_3}{\partial x_3 \partial \beta^*}\right)_Q = \frac{A}{(\delta+\mu)}$. .

Then, using [Chavez and Song (2004)], the bifurcation constants $a_1$and b_1 are obtained as follows:

$$a_1 = \sum_{k,i,j=1}^{4} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_Q$$
$$= u_3\left(w_1 w_3 \beta^* + w_2 w_3 \gamma + w_3 w_1 \beta^* + w_3 w_2 \gamma - {w_3}^2\left(\frac{A\alpha\beta^*}{\mu+\delta} + a\right)\right)$$
$$= -\frac{2}{\mu+\delta}\left(A{\beta^*}^2 + \frac{\gamma(\mu\delta\beta^* A + \gamma A\delta\mu + \gamma\delta^2 A)}{\mu(\mu+\delta)} + \alpha A\beta^* + a(\mu + \delta)\right) < 0,$$

$$b_1 = \sum_{k,i=1}^{4} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_Q$$

$$= w_3 \left(\frac{A}{(\delta+\mu)}\right)$$

$$= \frac{A}{(\delta+\mu)} > 0.$$

Thus, we state the following theorem:

Theorem 8.7: DFE $Q\left(\frac{A}{\mu+\delta}, \frac{\delta A}{\mu(\mu+\delta)}, 0,0\right)$ changes its behavior from stable to unstable at $R_0 = 1$ and there exists a positive equilibrium as R_0 crosses one. Hence, the system (8.14) exhibits transcritical forward bifurcation with a bifurcation parameter β^* at $R_0 = 1$.

8.3.2.2 Existence and stability analysis of the endemic equilibrium

To find conditions for the existence of an equilibrium $Q^*(S^*, T_1^*, I^*, T_2^*)$ for which the disease is endemic in the population, the system (8.9) are rearranged to get S^*, T_1^*, I^* and T_2^* which gives

$$S^* = \frac{A(1+\alpha I^*)}{(\delta+\mu)(1+\alpha I^*)+\beta I^*}, T_1^* = \frac{A\delta(1+\alpha I^*)}{(\mu+\gamma I^*)((\delta+\mu)(1+\alpha I^*)+\beta I^*)}, T_2^* = \frac{a I^{*2}}{(\mu+\theta)(1+b I^{*2})},$$

and I^* is given by the equation

$$C_1 I^{*4} + C_2 I^{*3} + C_3 I^{*2} + C_4 I^* + C_5 = 0 \qquad (8.15)$$

where

$$C_1 = \{b\gamma(\mu+d)\big(\beta+\alpha(\delta+\mu)\big)\},$$

$$C_2 = \{b(\mu+d)\Big(\gamma(\mu+\delta)+\mu\big(\beta+\alpha(\delta+\mu)\big)\Big)+a\gamma\beta+a\gamma\alpha(\delta+\mu)-Ab(\beta\gamma+\gamma\delta\alpha)\},$$

$$C_3 = \{\mu b(\mu+d)(\mu+\delta)+a\mu\big(\beta+\alpha(\delta+\mu)\big)+\gamma^2(\mu+d)\big(\beta+\alpha(\delta+\mu)\big)-Ab(\beta\mu+\gamma\delta)\},$$

$$C_4 = \{a\mu(\mu+\delta)+(\mu+d)\Big(\gamma(\delta+\mu)+\mu\big(\beta+\alpha(\delta+\mu)\big)\Big)-A(\beta\gamma+\gamma\delta\alpha)\},$$

$$C_5 = \{\mu(\mu+d)(\mu+\delta)-A(\beta\mu+\gamma\delta)\} = \mu(\mu+d)(\mu+\delta)(1-R_0).$$

Now using Descartes' rule of signs, there exists a unique positive real root I^* of the biquadratic equation (8.15) if any of the following conditions holds true:

I. $C_1 > 0, C_2 < 0, C_3 < 0, C_4 < 0$ and $C_5 < 0$.

II. $C_1 > 0, C_2 > 0, C_3 < 0, C_4 < 0$ and $C_5 < 0$.

III. $C_1 > 0, C_2 > 0, C_3 > 0, C_4 < 0$ and $C_5 < 0$.

IV. $C_1 > 0, C_2 > 0, C_3 > 0, C_4 > 0$ and $C_5 < 0$.

After finding I^*, we can find $S^*, {T_1}^*$ and ${T_2}^*$. Thus, a unique positive $Q^*(S^*, {T_1}^*, I^*, {T_2}^*)$ exists if one of the above conditions holds true.

The local stability of Q^* is explored as follows:

The characteristic equation of the system (8.9) evaluated at the endemic equilibrium point Q^* is given by

$$(\lambda + \mu + \theta)(\lambda^3 + p_0\lambda^2 + q_0\lambda + r_0 - (p_1\lambda^2 + q_1\lambda + r_1)e^{-\lambda\tau}) = 0 \qquad (8.16)$$

where

$$p_0 = \left(3\mu + \delta + \frac{\beta I^*}{1+\alpha I^*} + \gamma I^* + d + \frac{2aI^*}{(1+b{I^*}^2)^2}\right),$$

$$q_0 = \left(\left(2\mu + \gamma I^* - \gamma T_1^* + d + \frac{2aI^*}{(1+b{I^*}^2)^2}\right)\left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right) + (\mu + \gamma I^*)\left(\mu + d + \frac{2aI^*}{(1+b{I^*}^2)^2} - \gamma T_1^*\right) + \gamma^2 {T_1}^* I^*\right),$$

$$r_0 = \left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right)\left((\mu + \gamma I^*)\left(\mu + d - \gamma T_1^* + \frac{2aI^*}{(1+b{I^*}^2)^2}\right) + \gamma^2 {T_1}^* I^*\right),$$

$$p_1 = \frac{\beta S^*}{(1+\alpha I^*)^2},$$

$$q_1 = (2\mu + \delta + \gamma I^*)\frac{\beta S^*}{(1+\alpha I^*)^2},$$

$$r_1 = (\mu^2 + \mu\gamma I^* + \delta\mu)\frac{\beta S^*}{(1+\alpha I^*)^2}.$$

Clearly, $\lambda_1 = -(\mu + \theta)$ is a root of the characteristic equation (8.16) for $\tau \geq 0$, others roots λ_2, λ_3 and λ_4 can be obtained by the following equation:

$$\lambda^3 + p_0\lambda^2 + q_0\lambda + r_0 - (p_1\lambda^2 + q_1\lambda + r_1)e^{-\lambda\tau} = 0 \qquad (8.17)$$

Theorem 8.8: For $\tau = 0$, Q^* is locally asymptotically stable if all three $\frac{S^*}{I^*} \leq 1, \frac{S^*}{I^*} \leq \frac{(2\mu+d)}{(\delta+\gamma I^*)}$ and $\frac{S^*}{I^*} \leq \frac{(\mu+\gamma I^*)(\mu+d)}{(\mu^2+\mu\gamma I^*+\delta\mu)}$ are satisfied simultaneously.

Proof: At Q^*, the roots of the system (8.9) are $\lambda_1 = -(\mu + \theta)$, and other roots λ_2, λ_3 and λ_4 of the system are given by the equation $\lambda^3 + p_0\lambda^2 + q_0\lambda + r_0 - (p_1\lambda^2 + q_1\lambda + r_1) = 0$ for $\tau = 0$.

It is easy to show that if $\frac{S^*}{I^*} \leq 1, \frac{S^*}{I^*} \leq \frac{(2\mu+d)}{(\delta+\gamma I^*)}$ and $\frac{S^*}{I^*} \leq \frac{(\mu+\gamma I^*)(\mu+d)}{(\mu^2+\mu\gamma I^*+\delta\mu)}$ are satisfied simultaneously then

$$p_0 - p_1 = 3\mu + \delta + \frac{\beta I^*}{1+\alpha I^*} + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2} - \frac{\beta S^*}{(1+\alpha I^*)^2}$$

$$= 3\mu + \delta + \gamma I^* + d + \frac{\alpha\beta I^{*2}}{(1+\alpha I^*)^2} + \frac{2aI^*}{\left(1+bI^{*2}\right)^2} + \frac{\beta(I^*-S^*)}{(1+\alpha I^*)^2} > 0,$$

$$q_0 - q_1 = \left(2\mu + \gamma I^* + d - \gamma T_1^* + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right)\left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right) + (\mu + \gamma I^*)\left(\mu + d - \gamma T_1^* + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \gamma^2 {T_1}^* I^* - (2\mu + \delta + \gamma I^*)\frac{\beta S^*}{1+\alpha I^*} = (\mu + \gamma I^*)\left(\mu + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \gamma^2 {T_1}^* I^* + \left(2\mu + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right)(\mu + \delta) + \left(\frac{2aI^*}{\left(1+bI^{*2}\right)^2} + \gamma I^*\right)\left(\frac{\beta I^*}{1+\alpha I^*}\right) + \frac{\beta}{1+\alpha I^*}\left((2\mu + d)I^* - (\delta + \gamma I^*)S^*\right) > 0,$$

$$r_0 - r_1 = \left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right)\left((\mu + \gamma I^*)(\mu + d - \gamma T_1^*) + \frac{2aI^*(\mu+\gamma I^*)}{\left(1+bI^{*2}\right)^2} + \gamma^2 {T_1}^* I^*\right) - (\mu^2 + \mu\gamma I^* + \delta\mu)\frac{\beta S^*}{(1+\alpha I^*)^2}$$

$$= \left((\mu + \gamma I^*)(\mu + d) + \frac{2aI^*(\mu+\gamma I^*)}{\left(1+bI^{*2}\right)^2} + \gamma^2 {T_1}^* I^*\right)(\mu + \delta) + \left(\frac{2aI^*(\mu+\gamma I^*)}{\left(1+bI^{*2}\right)^2} + \gamma^2 {T_1}^* I^*\right)\frac{\beta I^*}{1+\alpha I^*} + \frac{\beta}{1+\alpha I^*}\left((\mu + \gamma I^*)(\mu + d)I^* - (\mu^2 + \mu\gamma I^* + \delta\mu)S^*\right) > 0.$$

Hence, by the Routh-Hurwitz criterion, the endemic equilibrium Q^* is locally asymptotically stable when $\tau = 0$.

Theorem 8.9: For $\tau > 0$, Q^* is locally asymptotically stable if all three $L_1 > L_2, L_3 > L_4$ and $\frac{S^*}{I^*} \leq \frac{(\mu+\gamma I^*)(\mu+d)}{(\mu^2+\mu\gamma I^*+\delta\mu)}$ are satisfied simultaneously,

where

$$L_1 = \left(3\mu + \delta + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2} + \frac{\beta(I^*+S^*)}{1+\alpha I^*}\right)\left(3\mu + \delta + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2} + \frac{\beta(I^*-S^*)}{1+\alpha I^*}\right),$$

$$L_2 = 2\left(\left(2\mu + \gamma I^* + d + \frac{2aI^*}{(1+bI^{*2})^2}\right)\left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right) + (\mu + \gamma I^*)\left(\mu + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \gamma^2 {T_1}^* I^*\right),$$

$$L_3 = \left(2\left((\mu^2 + \mu\gamma I^* + \delta\mu)\frac{\beta S^*}{1+\alpha I^*}\right)\left(\frac{\beta S^*}{1+\alpha I^*}\right) + \left(\left(2\mu + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right)\left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right) + (\mu + \gamma I^*)\left(\mu + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \gamma^2 {T_1}^* I^*\right)^2\right),$$

$$L_4 = \left(2\left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right)\left((\mu + \gamma I^*)\left(\mu + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \gamma^2 {T_1}^* I^*\right)\left(3\mu + \delta + \frac{\beta I^*}{1+\alpha I^*} + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \left((\delta + \gamma I^*)\frac{\beta S^*}{1+\alpha I^*}\right)^2\right).$$

Proof: At Q^*, the roots of Eq. (8.16) are $\lambda_1 = -(\mu + \theta)$, and other roots λ_2, λ_3 and λ_4 are given by the equation $\lambda^3 + p_0\lambda^2 + q_0\lambda + r_0 - (p_1\lambda^2 + q_1\lambda + r_1)e^{-\lambda\tau} = 0$ for $\tau > 0$. Assume that $\lambda = i\omega, \omega > 0$ is the root of Eq. (8.17).

Substituting $\lambda = i\omega$ in Eq. (8.17), we get

$$(r_0 - p_0\omega^2) - i\omega(\omega^2 - q_0) = (r_1 - p_1\omega^2)\cos\,\omega\tau + q_1\omega\,\sin\,\omega\tau - i((r_1 - p_1\omega^2)\sin\omega\tau - q_1\omega\cos\omega\tau) \quad (8.18)$$

On separating real and imaginary part of Eq. (8.18)

$$(r_0 - p_0\omega^2) = (r_1 - p_1\omega^2)\cos\,\omega\tau + q_1\omega\,\sin\,\omega\tau \quad (8.19)$$

$$\omega(\omega^2 - q_0) = (r_1 - p_1\omega^2)\sin\ \omega\tau - q_1\omega\cos\ \omega\tau \tag{8.20}$$

On squaring and adding both sides of Eqs. (8.19) & (8.20), we yield

$$(r_0 - p_0\omega^2)^2 + \omega^2(\omega^2 - q_0)^2 = (r_1 - p_1\omega^2)^2 + {q_1}^2\omega^2 \tag{8.21}$$

Letting $\omega^2 = z_1$, Eq. (8.21) becomes

$${z_1}^3 + ({p_0}^2 - 2q_0 - {p_1}^2){z_1}^2 + ({q_0}^2 + 2r_1p_1 - 2r_0p_0 - {q_1}^2)z_1 + ({r_0}^2 - {r_1}^2) = 0 \tag{8.22}$$

It is easy to show that if $L_1 > L_2, L_3 > L_4$ and $\frac{S^*}{I^*} \leq \frac{(\mu+\gamma I^*)(\mu+d)}{(\mu^2+\mu\gamma I^*+\delta\mu)}$ are satisfied simultaneously, then

$${p_0}^2 - 2q_0 - {p_1}^2 = (p_0 + p_1)(p_0 - p_1) - 2q_0$$

$$= \left(3\mu + \delta + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2} + \frac{\beta(I^*+S^*)}{1+\alpha I^*}\right)\left(3\mu + \delta + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2} + \frac{\beta(I^*-S^*)}{1+\alpha I^*}\right) - 2\left(\left(2\mu + \gamma I^* + d - \gamma T_1^* + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right)\left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right) + (\mu + \gamma I^*)\left(\mu + d - \gamma T_1^* + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \gamma^2 {T_1}^* I^*\right)$$

$$= L_1 - L_2 > 0,$$

$$2r_1p_1 + {q_0}^2 - 2r_0p_0 - {q_1}^2 = 2\left((\mu^2 + \mu\gamma I^* + \delta\mu)\frac{\beta S^*}{(1+\alpha I^*)^2}\right)\left(\frac{\beta S^*}{(1+\alpha I^*)^2}\right) + \left(\left(2\mu + \gamma I^* + d - \gamma T_1^* + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right)\left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right) + (\mu + \gamma I^*)\left(\mu + d - \gamma T_1^* + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \gamma^2 {T_1}^* I^*\right)^2 - \left(2\left(\mu + \delta + \frac{\beta I^*}{1+\alpha I^*}\right)\left((\mu + \gamma I^*)\left(\mu + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \gamma^2 {T_1}^* I^*\right)\left(3\mu + \delta + \frac{\beta I^*}{1+\alpha I^*} + \gamma I^* + d + \frac{2aI^*}{\left(1+bI^{*2}\right)^2}\right) + \left((2\mu + \delta + \gamma I^*)\frac{\beta S^*}{1+\alpha I^*}\right)^2\right) = L_3 - L_4 > 0,$$

$${r_0}^2 - {r_1}^2 = (r_0 + r_1)(r_0 - r_1)$$

$$= \left(\left((\mu + \gamma I^*)(\mu + d - \gamma T_1^*) + \frac{2aI^*(\mu+\gamma I^*)}{(1+bI^{*2})^2} + \gamma^2 {T_1}^* I^* \right)(\mu + \delta) + \left(\frac{2aI^*(\mu+\gamma I^*)}{(1+bI^{*2})^2} + \gamma^2 {T_1}^* I^* \right) \frac{\beta I^*}{1+\alpha I^*} + \frac{\beta}{1+\alpha I^*} \left((\mu + \gamma I^*)(\mu + d) I^* + (\mu^2 + \mu\gamma I^* + \delta\mu) S^* \right) \right) \left(\left((\mu + \gamma I^*)(\mu + d - \gamma T_1^*) + \frac{2aI^*(\mu+\gamma I^*)}{(1+bI^{*2})^2} + \gamma^2 {T_1}^* I^* \right)(\mu + \delta) + \left(\frac{2aI^*(\mu+\gamma I^*)}{(1+bI^{*2})^2} + \gamma^2 {T_1}^* I^* \right) \frac{\beta I^*}{1+\alpha I^*} + \frac{\beta}{1+\alpha I^*} \left((\mu + \gamma I^*)(\mu + d) I^* - (\mu^2 + \mu\gamma I^* + \delta\mu) S^* \right) \right) > 0.$$

Hence, by the Routh-Hurwitz criterion, the endemic equilibrium Q^* is locally asymptotically stable for $\tau > 0$.

8.4 Numerical simulations

This section is devoted to the demonstration of the results of the numerical simulation of the models.

8.4.1 Results of the system (8.1)

For the simulation of the system (8.1), we take the following numerically experimental values of parameters:

$$\pi = 2, \alpha = 0.5, \beta = 0.003, \mu = 0.007, d = 0.05, \gamma = 0.001, \delta = 0.002, a = 0.2, b = 0.2, \theta = 0.002.$$

With the above values of parameters, we calculate the coefficients of Eq. (8.5) as $C_1 = -0.0000044829$, $C_2 = 0.000004607, C_3 = 0.00000503207, C_4 = 3.0267 \times 10^{-7}$ with the basic reproduction number $R_0 = 1.274$ which satisfies the condition (i) i.e. $C_1 < 0, C_2 > 0, C_3 > 0, C_4 > 0$ for the existence of a unique positive I^*. The trajectories of $S, A, I,$

and R with initial conditions $S(0) = 200,\ A(0) = 15,\ I(0) = 2,\ R(0) = 1$ approaches to the endemic equilibrium $Q^*(\ 154.6402, 37.1406,\ \ 3.8084, 62.1365)$ as shown in Fig. 8.4.

In Fig. 8.4, we plot the graphs for S, A, I and R populations. We observe that the number of infected individuals goes up, and after some days it decreases and become constant due to Holling type II treatment, and these individuals once recovered, become immunized to the infection and will not get re-infected in future. Susceptible individuals decrease to attain a steady state. This decrease may be due to increase in the number of alert individuals. Further, the alert population increases as the alertness behavior prevents infection and finally, the population settles down to its steady state. This increase causes a decrease in the number of infected individuals because of alertness in society. Furthermore, the recovered individuals increase which may be due to the Holling type II treatment of infected individuals, and settles to the steady state.

Fig. 8.5 portrays the difference between the infectives with alert and without alert class. It can be seen that the number of infected individuals without the alert class is higher than the number of infected individuals with the alert class. Hence, the alert class plays a vital role in controlling the infection of an epidemic in society.

Fig. 8.6 exhibits the difference between the infected population with Holling type II treatment rate and without treatment. It is evident from this figure that the number of infected individuals without treatment rate is very high in comparison to the number of infected individuals with Holling type II treatment rate. Hence, the Holling type II treatment rate has an important role in suppressing the infection in society.

Figs. 8.7 and 8.8 show the effect of the cure rate (a) and limitation rate (b) in treatment availability on the infected population at numerous values of a and b. Fig. 8.7 demonstrates the decrease in the infected population as the cure rate (a) increases and it settles down at its steady state, but the disease is not eliminated entirely as it will persist at a much lower level. Fig. 8.8 expresses an increase in the infected population as b increases, which is due to the limited availability of resources in the society.

Figs. 8.9 & 8.10 depict the infected population at various values of transmission rates (β) and (γ) respectively. Clearly, in these figures, the infected population (I) increases with the increase in the values of β and γ.

Fig. 8.11 depicts the infected population at numerous values of α. It can be seen that the number of infected individuals decreases as the values of α increases.

Fig. 8.12 shows the combined population of susceptible (S) and alert (A) individuals with respect to the time.

8.4.2 Results of the system (8.9)

For the numerical simulation, we take the following numerically experimental values of parameters.

$$A = 9, \alpha = 0.05, \beta = 0.003, \mu = 0.02, d = 0.05, \gamma = 0.001, \delta = 0.01, a = 0.2, b = 0.02, \theta = 0.002.$$

Figs. 8.13 and 8.14 delineate the population in various compartments at two different values of incubation time delay $\tau = 1, 2$ approaches to endemic equilibrium. The number of infected individuals is initially increasing and as time passes, they are decreasing due to treatment and recovery from infection. The susceptible population decreases to attain its steady state. Also, pre-treated and post-treated populations increase to attain a steady state for both the time delays as shown in Figs. 8.13 and 8.14. The increment in the pre-treated population is because of the vaccination of susceptible population as a result of which susceptible population decreases. The post treated population increases because the treatment is given to the infected population and hence infected population decreases. We also observe that the final number of infected is lower for $\tau = 1$ than $\tau = 2$.

Fig. 8.15 exhibits the effect of delay on the infected individuals. It is evident that as the value of τ increases, the number of infected individuals also increases in comparison to decreasing values of time lag τ. Delay clearly indicates that as much time we take to initiate the preventive measures, the greater number of individuals will be infected.

Fig. 8.16 expresses the effect of delay on the pre-treated population. According to the figure, as the value of τ increases, the number of pre- treated individuals decreases. This decrease may be due to the time taken by health agencies to provide the precautionary treatment to the susceptible individuals. It can be understood that time will be consumed to provide vaccination or any other precautionary treatment to susceptibles; this will result in the increment of the infected population.

The effect of delay on the post-treated population is shown in Fig. 8.17. Clearly, as τ increases the number of post-treated individual's increases. Delay indicates that greater time we take to initiate the post treatment to infected, requires us to provide the treatment to more infected individuals.

Figs. 8.18 and 8.19 exhibit the variations in the infected population at various values of β and α respectively. The graphs show the decrement in the infected population with a decline in transmission rate (β) and an enhancement of inhibition rate (α).

Fig. 8.20 shows the pre-treated population at various values of the pre-treatment rate (δ). Clearly, as δ increases, the number of pre-treated individuals also increases.

Figs. 8.21and 8.22 differentiate in the infected population at various values of a and b respectively. Fig. 8.21 shows the increment in an infected population with a decrement in a and it settles down at its steady state, but the disease is not getting totally eradicated as it will persist at a much lower level. Fig. 8.22 shows the increment in an infected population with increment in b which is due to limited availability of resources in the society.

8.5 Conclusions

In this chapter, we have proposed and analyzed two different models: $(\boldsymbol{i})$ A susceptible-alert-infected-recovered (SAIR) model with two explicit saturated incidence rates along with Holling type II treatment rate $(\boldsymbol{ii})$ A time-delayed SIR model by introducing two explicit treatment classes along with the saturating incidence rate, Holling type I treatment to susceptible population and Holling type III treatment to infected population,

to study the transmission and control of the epidemic. The models analysis show that there exist only two types of equilibria: disease-free, *i.e.* when there is no infection in the society, and endemic, *i.e.* when the infection is present in the society. The disease-free equilibrium is locally asymptotically stable when the basic reproduction number is less than unity. We have also shown that both the systems (8.2) & (8.9) undergo a transcritical bifurcation at $R_0 = 1$. We also investigated the stability of the endemic equilibrium (EE) for both the models and showed that endemic equilibrium is locally asymptotically stable when the condition stated in theorem 8.4 holds true for the system (8.2) and the conditions stated in theorems 8.8 & 8.9 holds true for the system (8.9). Numerical simulations have been carried out to explore the effect of alertness, Holling type II treatment rate and the incubation time delay on various classes of the population. It is observed that the number of infected individuals decreases as the cure rate increases. However, the number of infected individuals increases as the limitation rate in treatment availability increases. It shows that for effective treatment, the resource limitation should be minimized. We also observed that the number of infected individuals increases as the transmission rate increases and it decreases as the rate of inhibition increases. Further, we observed that the delay can play a very crucial role to control the disease and in providing the preventive measures.

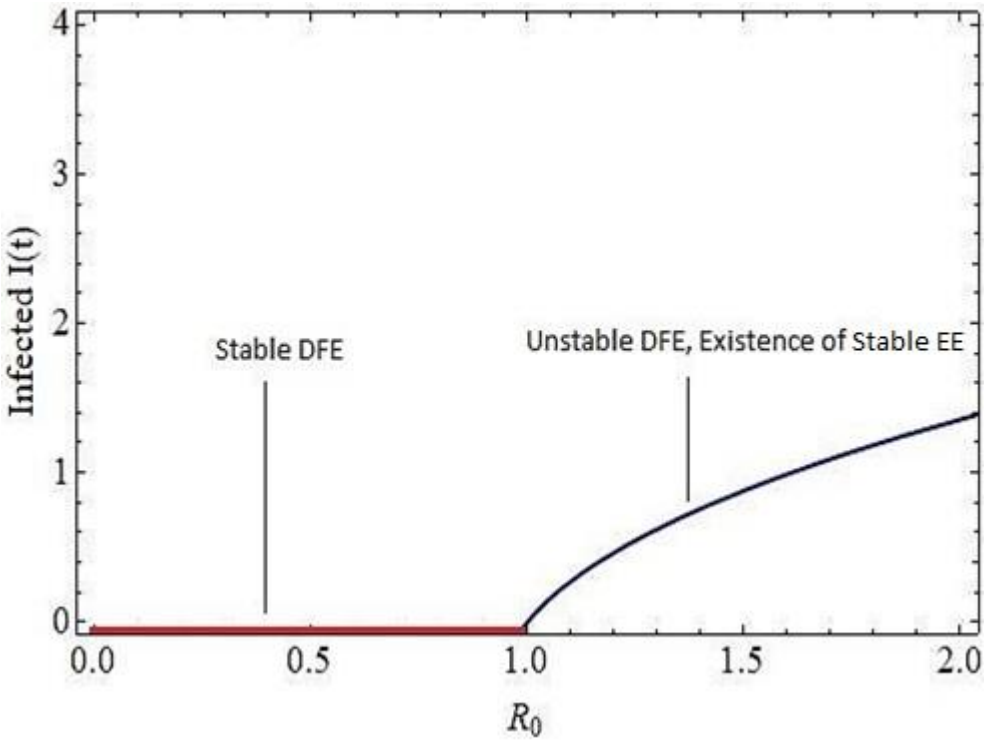


Fig. 8.3: Graph in support of theorem 8.2 & 8.3 with parameter values $\pi = 2, \alpha = 0.5, \gamma = 0.0009,\ \mu = 0.007,\ d = 0.05, \delta = 0.002, b = 0.2,\ a = 0.2, \theta = 0.002,\ \beta = 0.0012.$

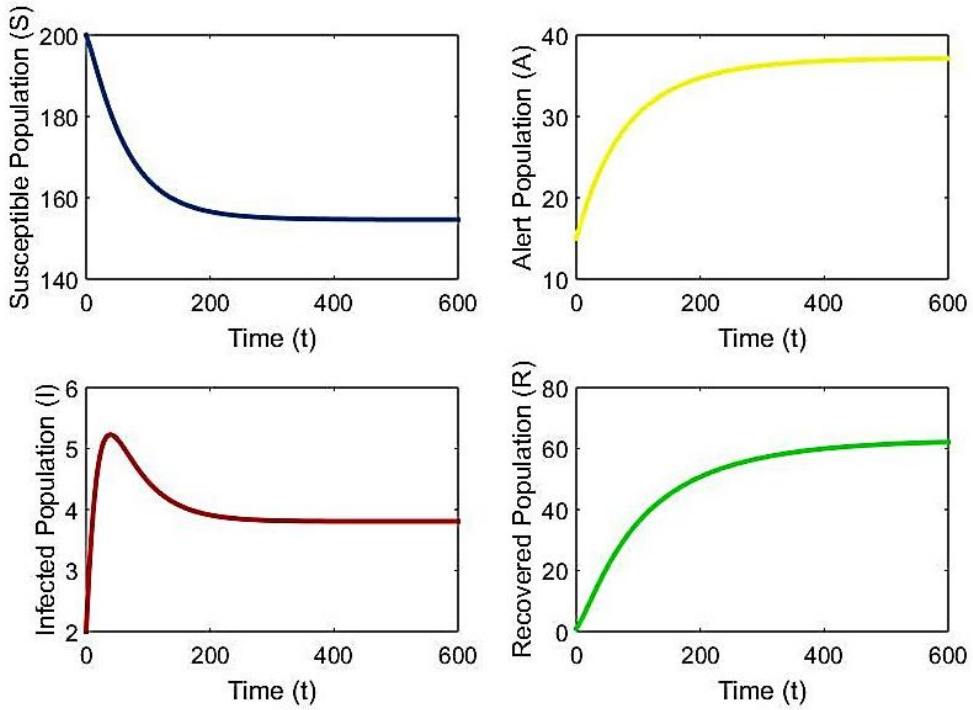


Fig. 8.4: Susceptible (S), alert (A), infected (I) and recovered (R) population.

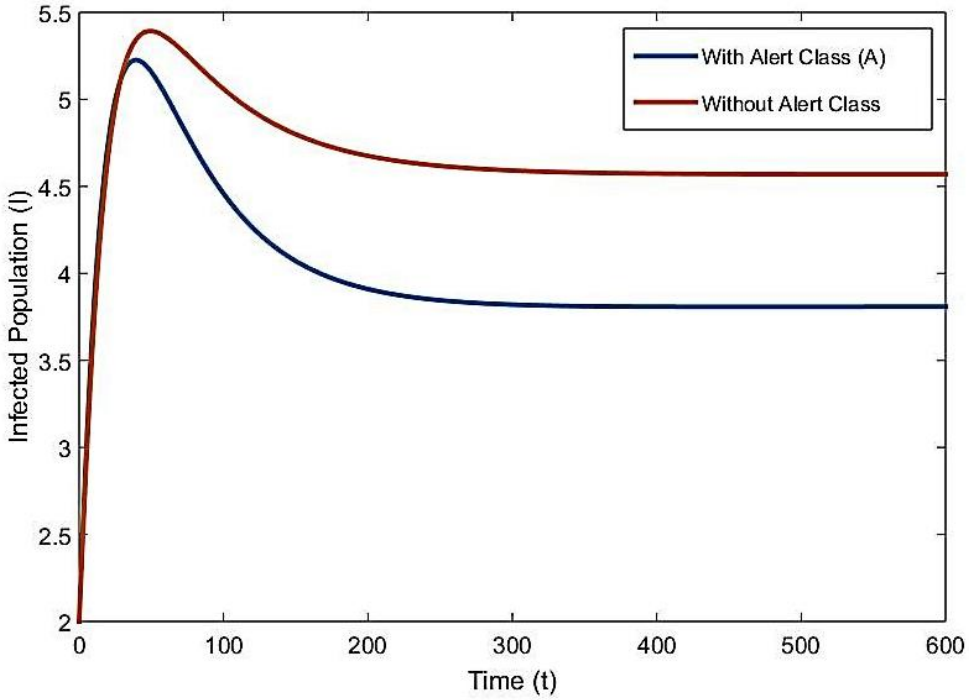


Fig. 8.5: Infected population (I) with and without alert class(A).

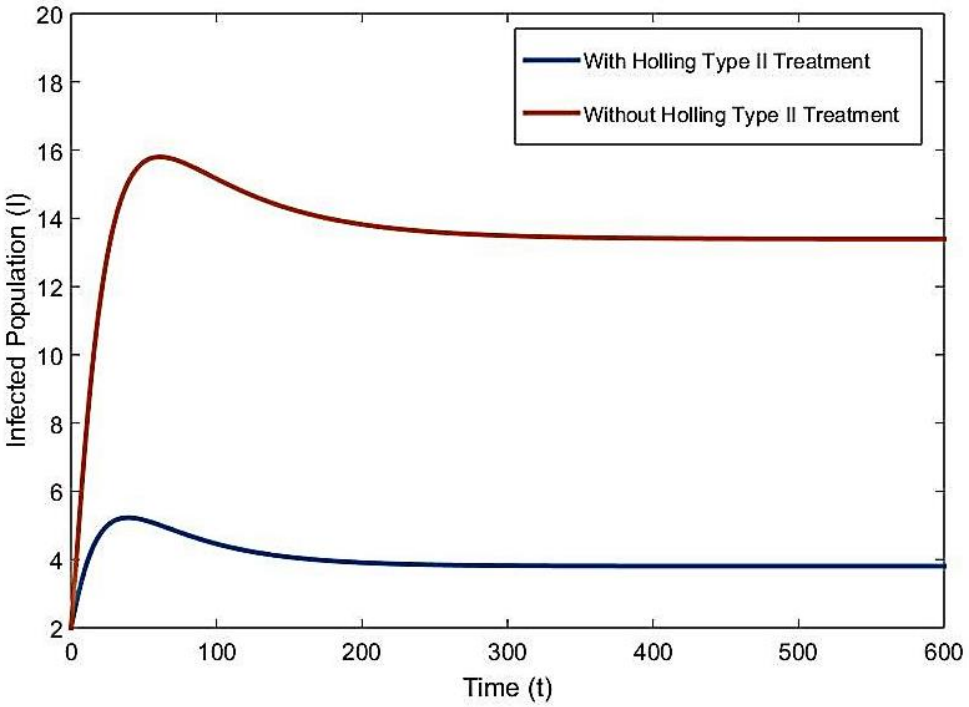


Fig. 8.6: Graph depicting the behavior of the infected population (I) with and without Holling Type II treatment rate.

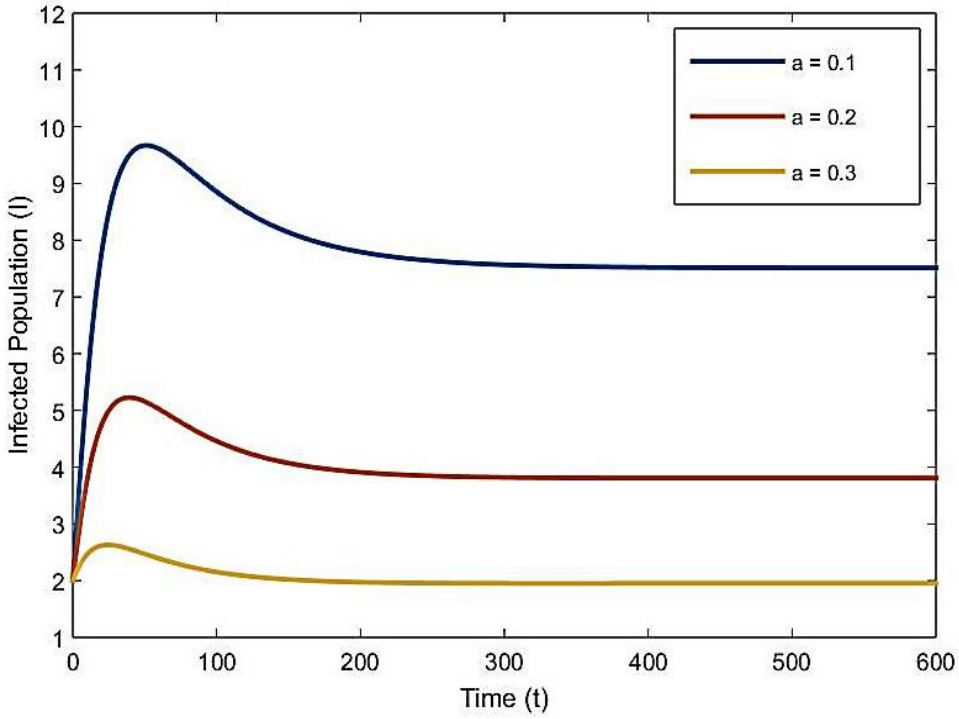


Fig. 8.7: Impact of the cure rate (a) on the infected population (I).

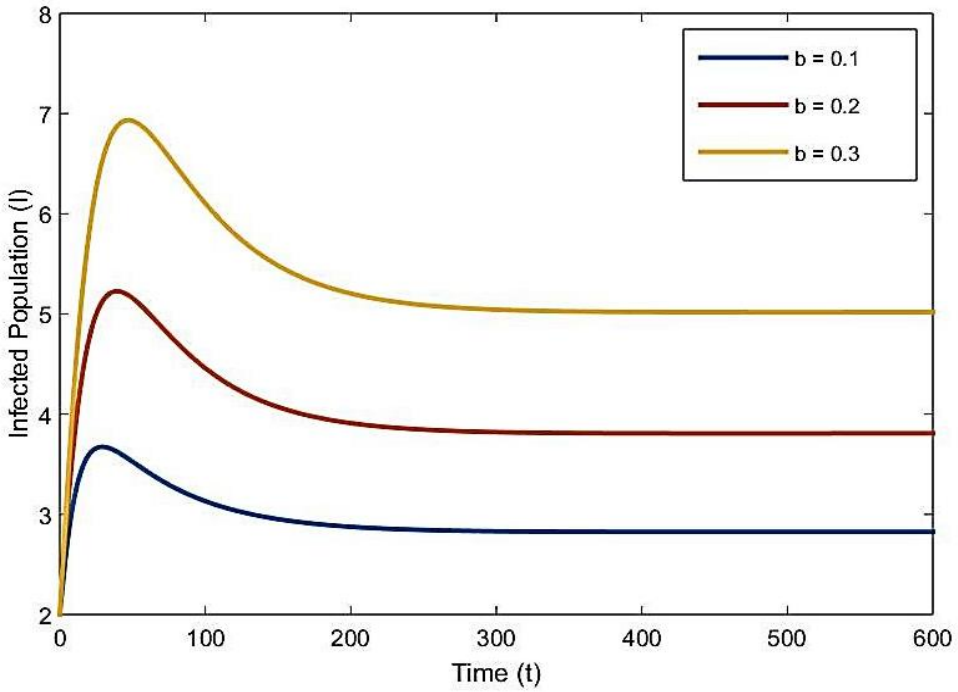


Fig. 8.8: Impact of limitation rate (b) in treatment availability on the infected population (I).

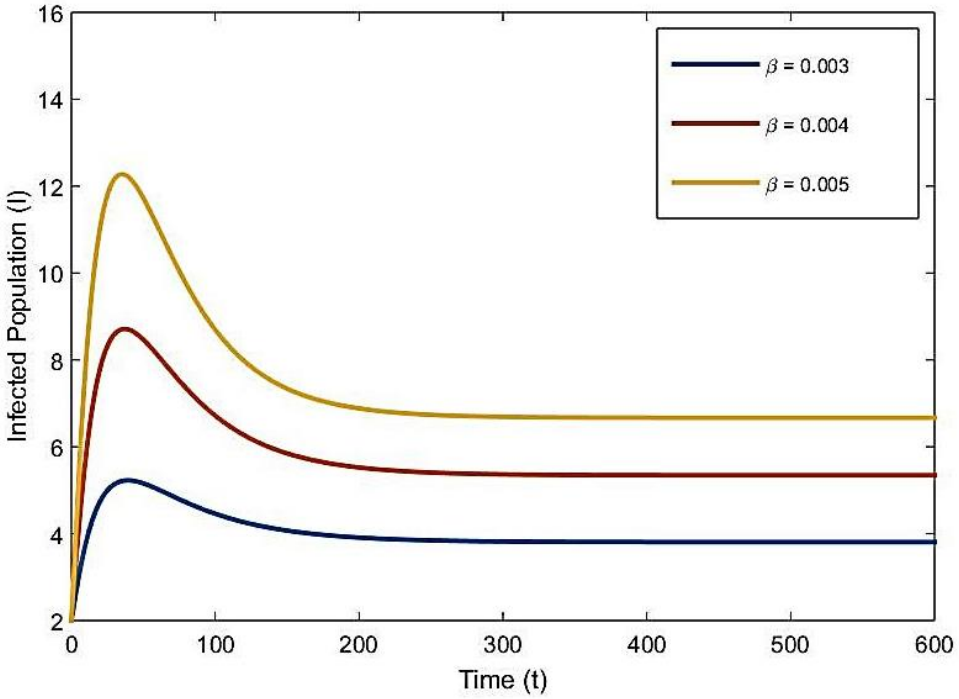


Fig. 8.9: Impact of the transmission rate (β) on the infected population(I).

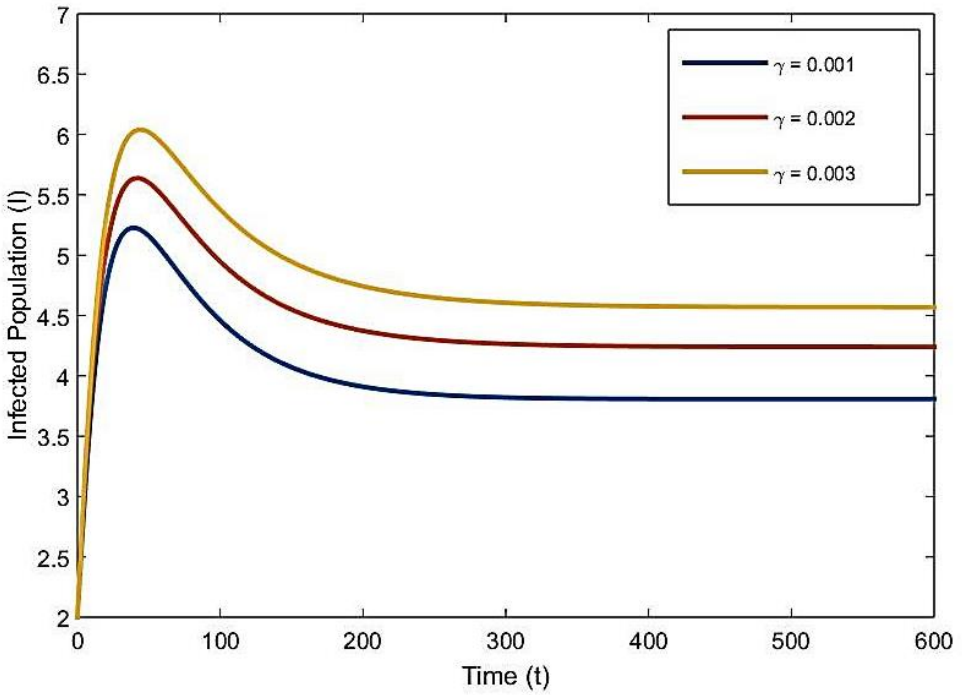


Fig. 8.10: Impact of the transmission rate (γ) on the infected population (I).

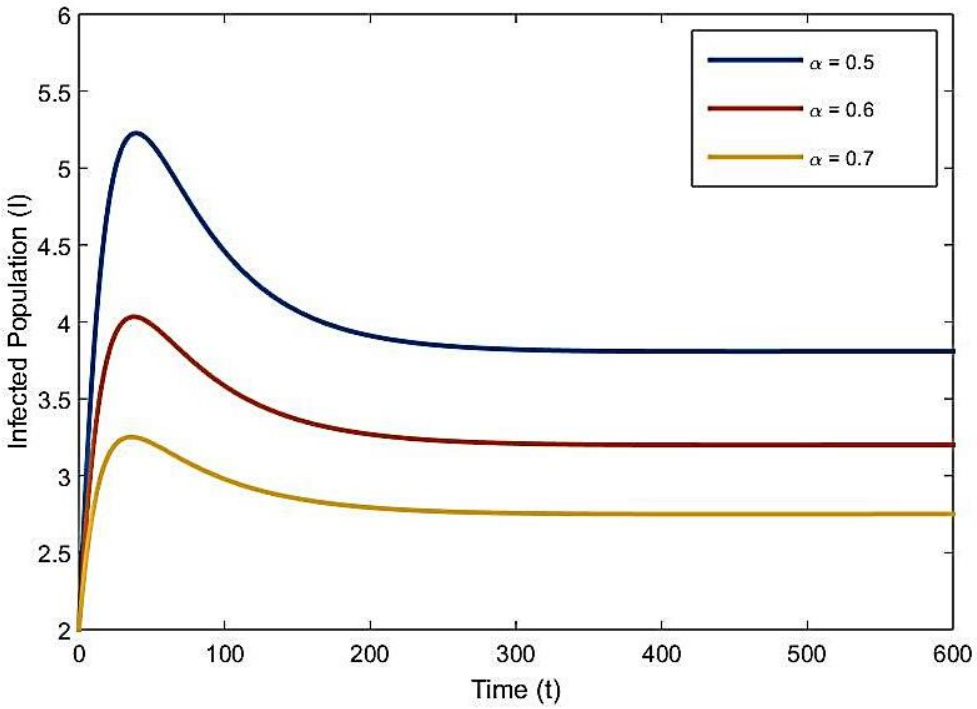


Fig. 8.11: Infected population (I) at various values of measures of inhibition (α).

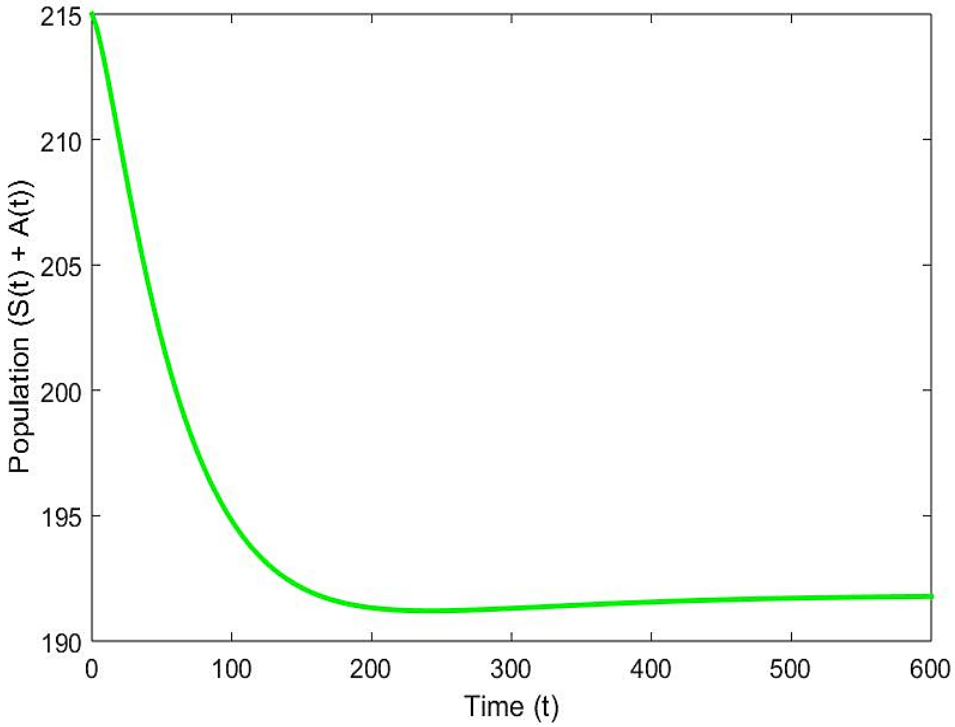


Fig.12: Combined population $(S(t) + A(t))$ with respect to time (t).

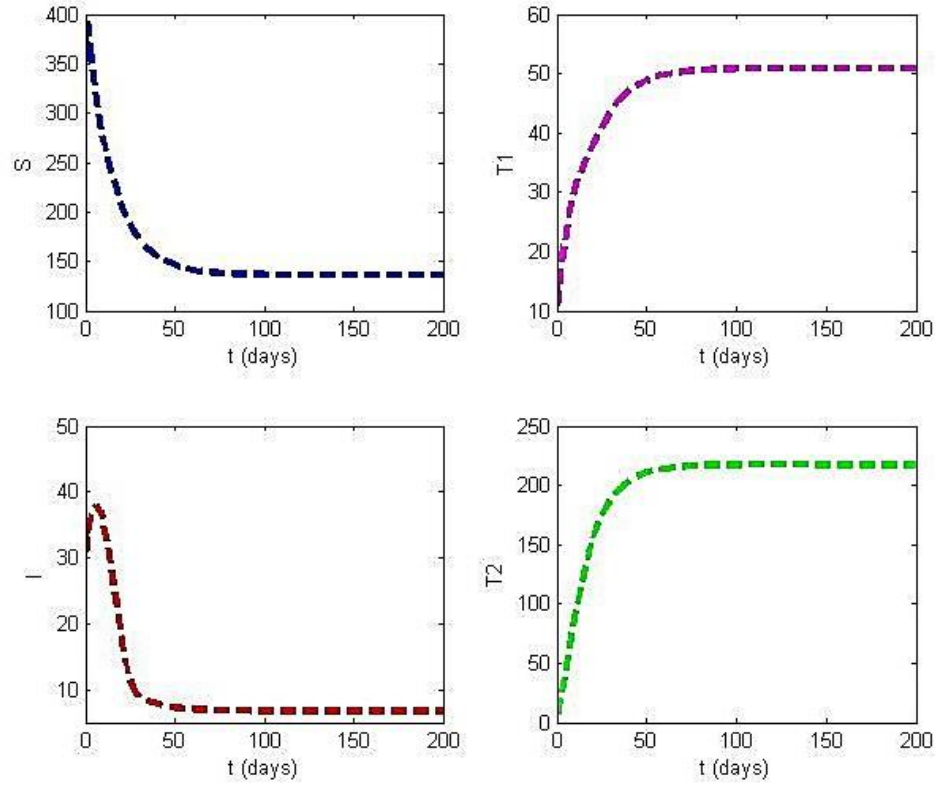


Fig. 8.13: Population (S, T_1, I, T_2) at time lag $\tau = 1$.

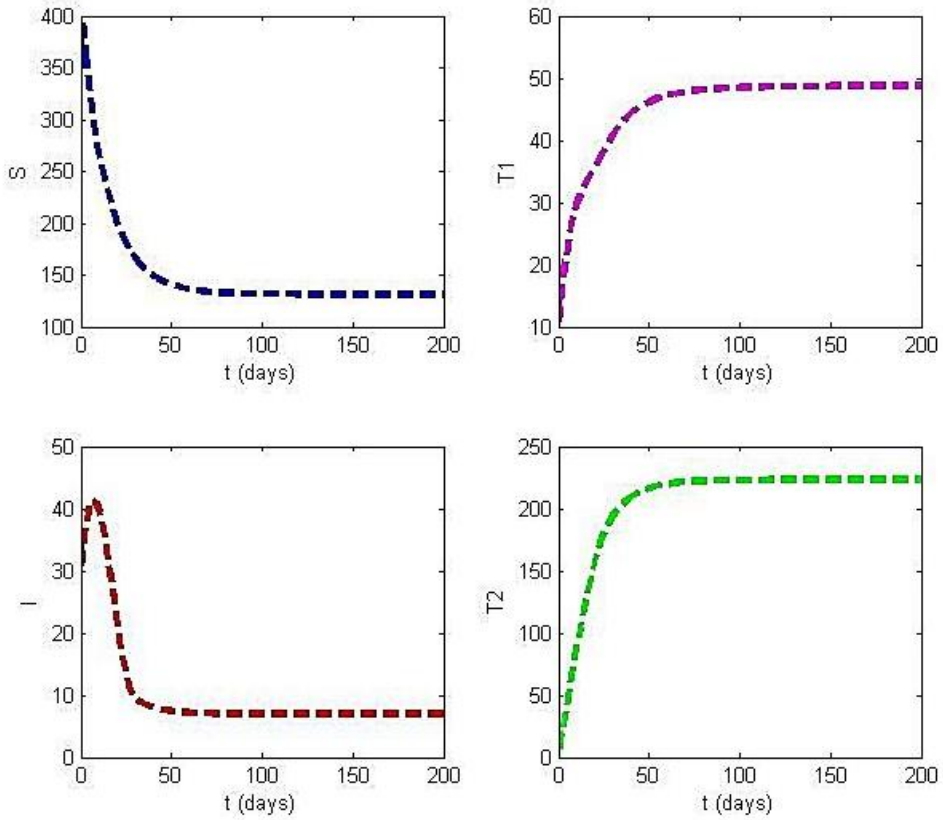


Fig. 8.14: Population (S, T_1, I, T_2) at time lag $\tau = 2$.

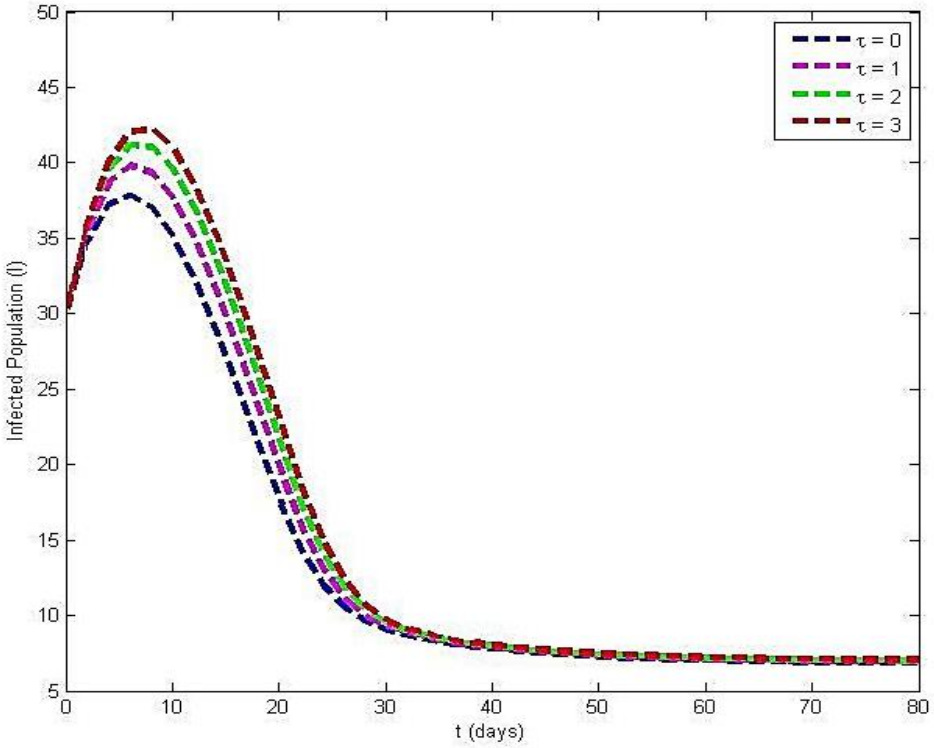


Fig. 8.15: Infected population (I) for various values of time lag τ.

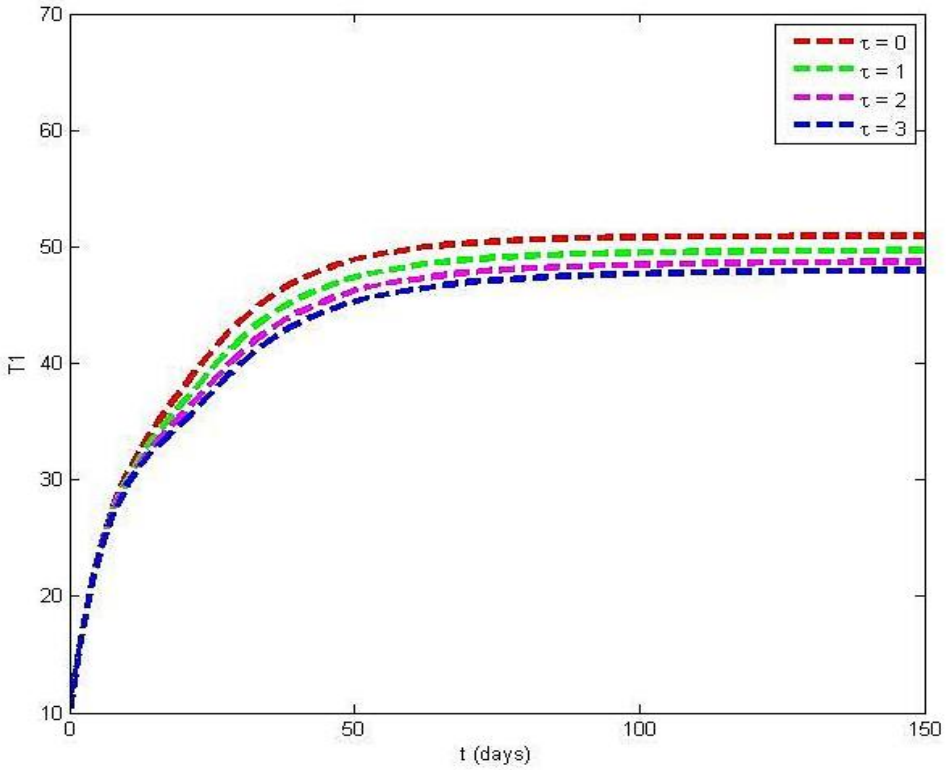


Fig. 8.16: Pre-treated population (T_1) at various values of time lag τ.

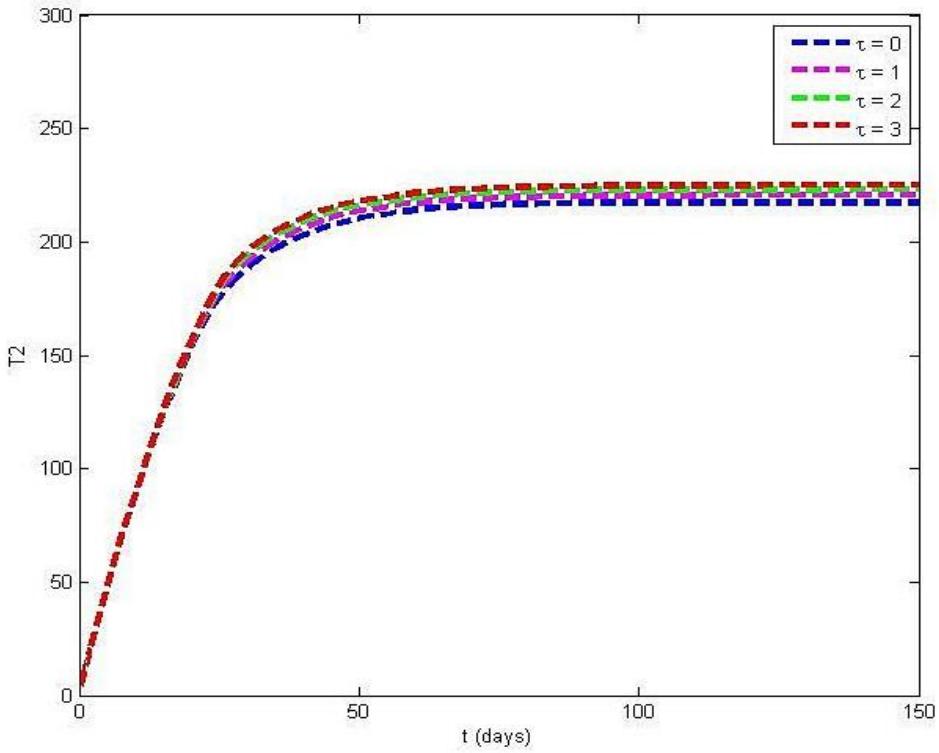


Fig. 8.17: Post-treated population (T_2) at various values of time lag τ.

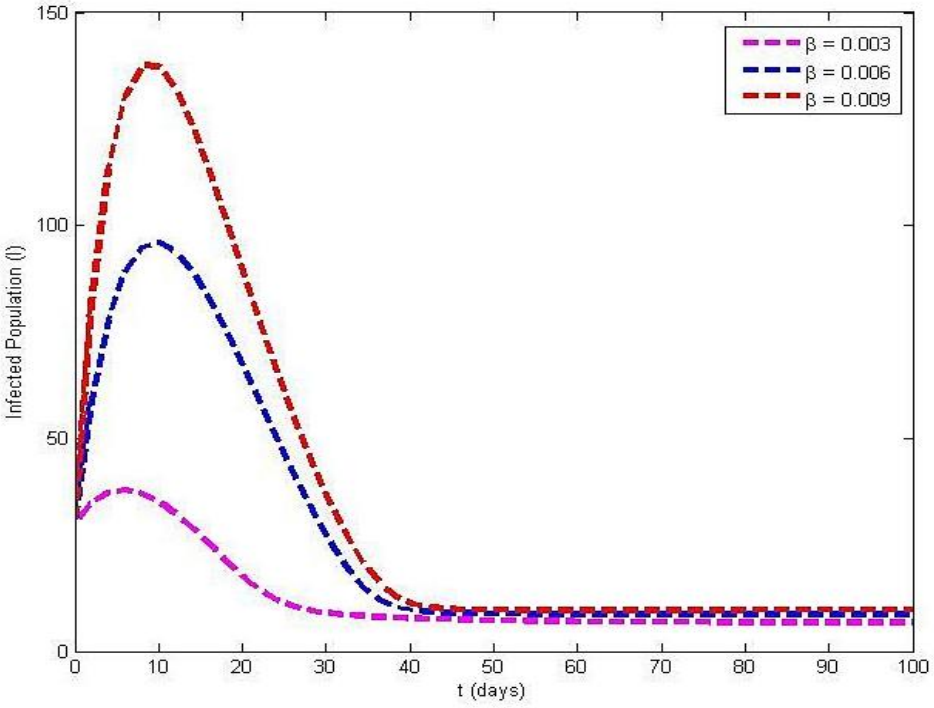


Fig. 8.18: Infected population (I) at various values of the transmission rate (β) at $\tau = 0$.

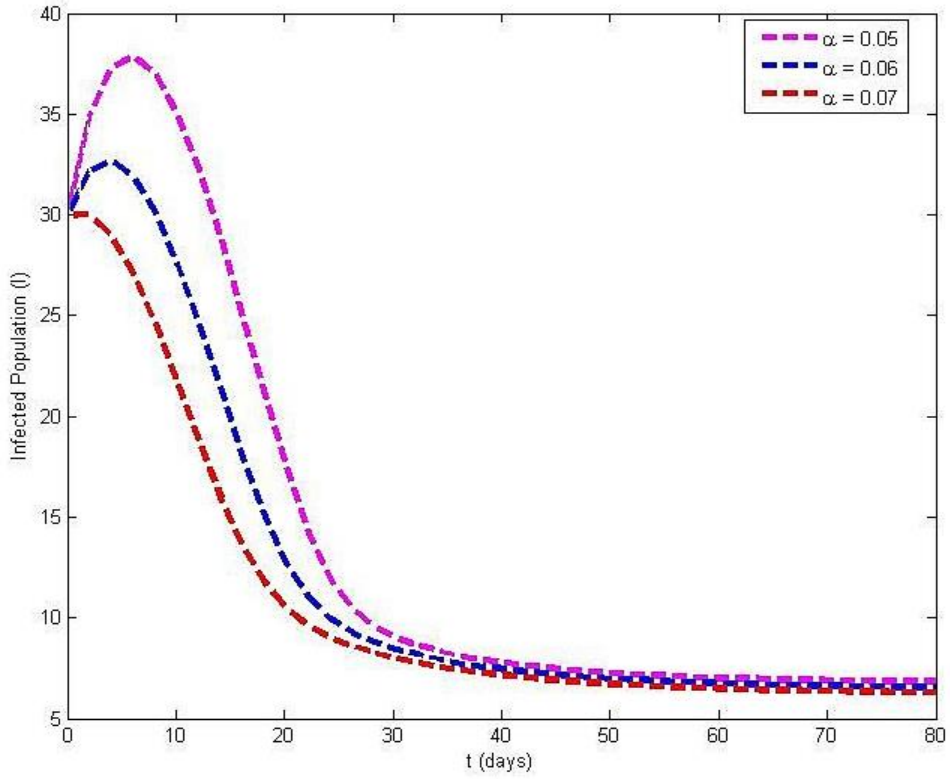


Fig. 8.19: Infected population (I) at various values of measures of inhibition (α) at $\tau = 0$.

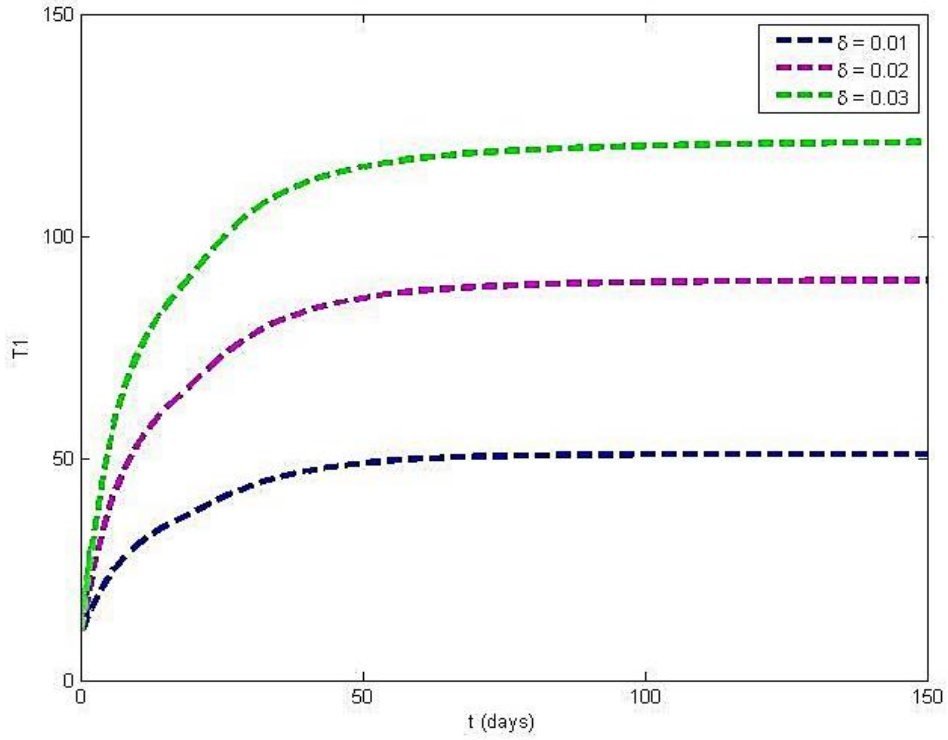


Fig. 8.20: Pre-treated population (T_1) at various values of pre-treatment rate (δ) at $\tau = 0$.

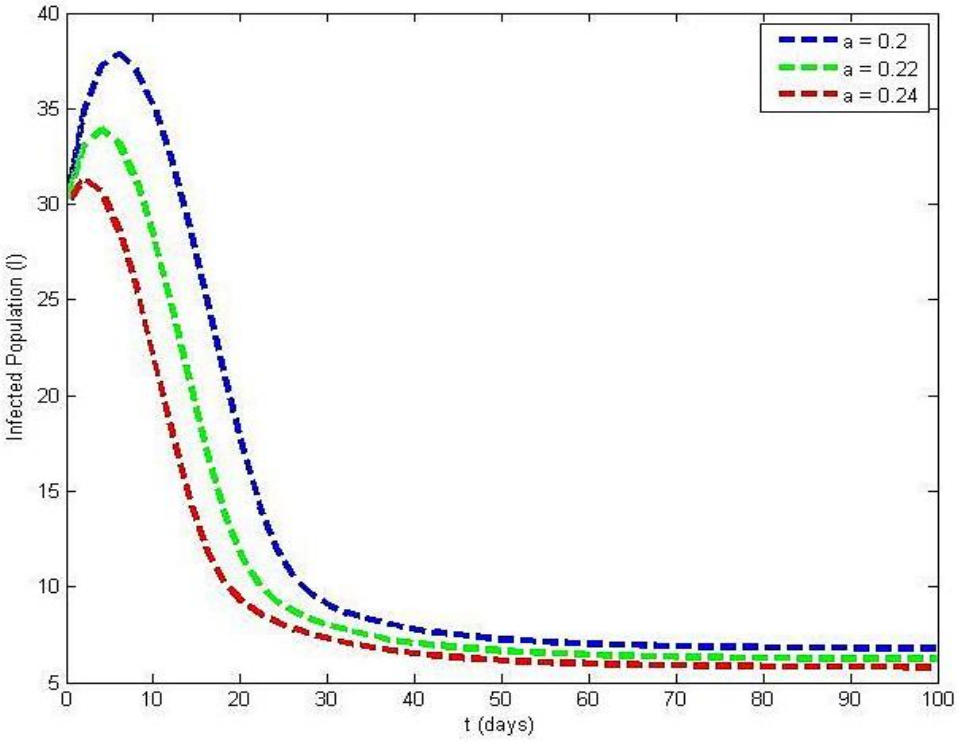


Fig. 8.21: Infected population (I) at various values of cure rate (a) at $\tau = 0$.

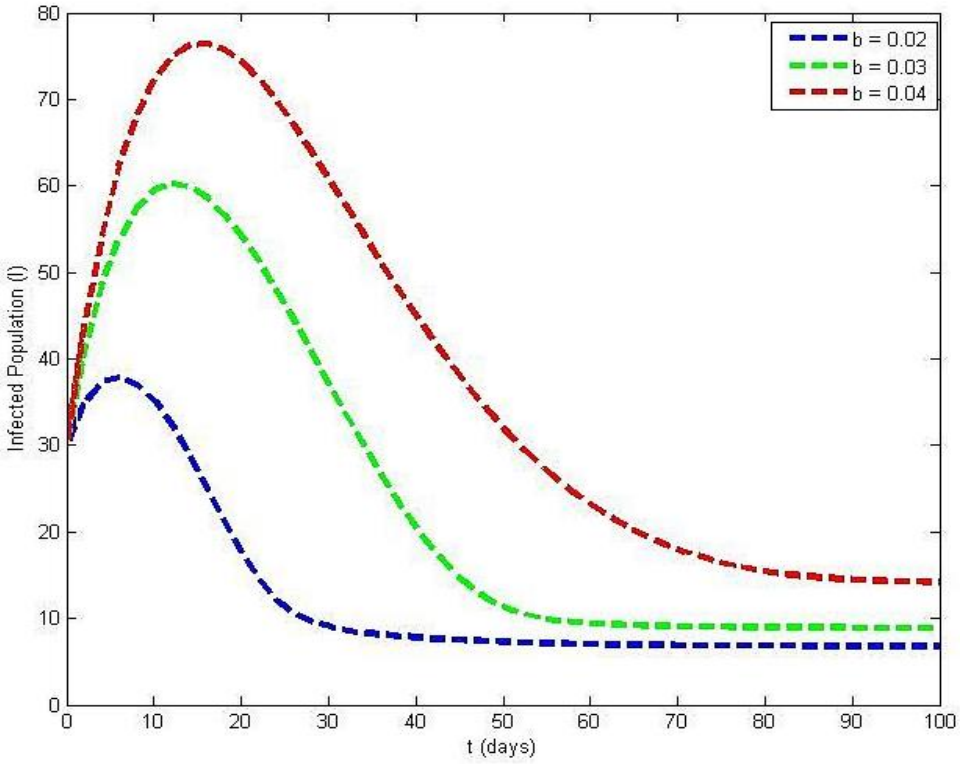


Fig. 8.22: Infected population (I) at various values of limitation rate (b) in treatment availability at $\tau = 0$.

CHAPTER 9

CONCLUSIONS AND FUTURE WORK

In this chapter, we summarize the main outcomes of the thesis and some future aspects have been reported which may be studied in the future course of time.

9.1. Conclusions

There is no doubt that mathematical modeling is essential in planning and formulation of policy on contagious diseases. In this thesis, we studied transmission and prevention mechanisms of the epidemics through mathematical models. We focused mainly on SIR models and also incorporated various compartments according to epidemiological states in SIR model. Delay differential equations are being used and they can, for instance, accommodate the phenomenon of an incubation period and latency period in SIR models. We studied their stability properties, paying particular attention to the basic reproductive number. We also addressed the global stability of some epidemic models.

The main focus of the thesis is to provide the epidemics transmission process and control strategies with nonlinear incidence and treatment rates to provide more realism for eradication/ minimization/possible control of the infection in society. Models have been classified according to the outbreak, transmission, and spread of the disease by incorporating various factors, e.g. psychological effects, low density of susceptibles, measures of inhibition, and limitations in treatment. Extra compartments in the SIR model have been introduced for various stages according to the requirements of the diseases, for example, pre-treated, post-treated, and alert compartments. The novel combination of different types of nonlinear incidence and treatment rates as per the need of the disease has also been introduced in the models. The incidence rates of infection are the nonlinear functional type which provides desired dynamics of transmission of infection in case of a large population. It is shown that proposed models are epidemiologically well-behaved. Equilibrium analysis of the models proves the existence and uniqueness of equilibria.

Local and global stability analysis of the equilibria have been investigated and further validated through numerical simulations. We found the threshold parametric value of infection, *i.e.* the basic reproduction number R_0 for each model, to determine the persistence of infection in the endemic zone. The explained models are able to capture successfully better incidence and treatment rates for the different type of diseases according to the dynamics. The proposed combinations of incidence and treatment rates may be adopted by the public health agencies to monitor and further control of the epidemic.

9.2. Future work

In this thesis, we have proposed only deterministic models for disease transmission. We have studied the stability analysis of these models and presented the numerical computations in the form of graphs in the supports of theoretical results. As further studies and future directions, we may explore the models for chaotic behaviour and stochasticity. We may also present disease transmission dynamics by fractional order derivatives for a better understanding of the disease dynamics.

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2. **Abhishek Kumar**, Nilam (2019) "Dynamical Model of Epidemic Along with Time Delay; Holling Type II Incidence Rate and Monod-Haldane Type Treatment Rate" in *Differential Equations and Dynamical Systems **27**(1-3): 299-312 (Springer).*
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7. **Abhishek Kumar**, Kanica Goel, Nilam "A Deterministic Time-Delayed SIR Epidemic Model: Mathematical Modeling and Analysis". (Under review)
8. **Abhishek Kumar**, Nilam "Analysis of a Disease Transmission Model with Time Delay, Nonlinear Functional Type Incidence Rate and Saturated Treatment Rate". (Under review)
9. **Abhishek Kumar**, Nilam "Dynamical Study of an Epidemic Model with Nonlinear Functional Type Incidence and Treatment". (Under review)

10. **Abhishek Kumar**, Manoj Kumar, Nilam "A Study on the Dynamics of an Epidemic Model with Ratio-Dependent Incidence and Holling type II Treatment Rates". (Under review)

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1. **Abhishek Kumar**, Nilam "A SAIR model with two explicit nonlinear incidence rate and saturated treatment rate" presented at International Conference on Recent Advances in Pure and Applied Mathematics held at DTU Delhi, India, October 23-25, 2018.
2. **Abhishek Kumar**, Nilam "Mathematical control strategy with time-dependent contact rate for contagious disease" presented at 5th International Conference on Advancements in Engineering and Technology held at BGIET Sangrur, Punjab, March 24-25, 2017.
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Stability of a Time Delayed SIR Epidemic Model Along with Nonlinear Incidence Rate and Holling Type-II Treatment Rate

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In this paper, we present a mathematical study of a deterministic model for the transmission and control of epidemics. The incidence rate of susceptible being infected is very crucial in the spread of disease. The delay in the incidence rate is proved fatal. In the present study, we propose an SIR mathematical model with the delay in the infected population. We are taking nonlinear incidence rate for epidemics along with Holling type II treatment rate for understanding the dynamics of the epidemics. Model stability has been done by the basic reproduction number $\boldsymbol{R_0}$. The model is locally asymptotically stable for disease-free equilibrium $\boldsymbol{Q}$ when the basic reproduction number $\boldsymbol{R_0}$ is less than one ($\boldsymbol{R_0 < 1}$). We investigated the stability of the model for disease-free equilibrium at $\boldsymbol{R_0}$ equals to one using center manifold theory. We also investigated the stability for endemic equilibrium $\boldsymbol{Q^*}$ at $\boldsymbol{\tau \geq 0}$. Further, numerical simulations are presented to exemplify the analytical studies.

Keywords: Epidemic; SIR model; delay differential equation; Holling type II treatment rate; stability; center manifold theory.

1. Introduction

Control of epidemics in human's society is a major task for government, doctors and health agencies. Various biological reasons lead to the introduction of time delays in a model of disease transmission. Delay model is used in an attempt to better understanding of more and more complicated phenomena for describing several aspects of infectious disease dynamics. In this paper, an attempt has been made to show that the control of epidemics is possible in a very effective manner using time

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ORIGINAL RESEARCH

Dynamical Model of Epidemic Along with Time Delay; Holling Type II Incidence Rate and Monod–Haldane Type Treatment Rate

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Published online: 10 May 2018

Abstract The present study aims to control the infectious diseases and epidemics in the human population. Therefore, in the present article, we have proposed a delayed SIR epidemic model along with Holling type II incidence rate and treatment rate as Monod–Haldane type. Model stability has been established in the three regions of the basic reproduction number R_0 i.e. R_0 equals to one, greater than one and less than one. The model is locally asymptotically stable for disease-free equilibrium Q when the basic reproduction number R_0 is less than one ($R_0 < 1$) and unstable when $R_0 > 1$ for time lag $\tau \geq 0$. We investigated the stability of the model for disease-free equilibrium at R_0 equals to one using central manifold theory. Using center manifold theory, we proved that at $R_0 = 1$, disease-free equilibrium changes its stability from stable to unstable. We also investigated the stability for endemic equilibrium Q^* for time lag $\tau \geq 0$. Further, numerical simulations are presented to exemplify the analytical studies.

Keywords Epidemic · SIR model · Delay differential equation · Monod–Haldane type treatment rate · Holling type II incidence rate · Stability · Center manifold theory

Mathematics Subject Classification 34D20 · 92B05 · 37M05

Introduction

In mathematical epidemiology literature many authors [1–3, 10, 13, 14] have suggested the various models for the disease transmission like susceptible–infected–recovered (SIR) model, susceptible–infected–recovered–susceptible (SIRS) and many more. In the population

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Mathematical analysis of a delayed epidemic model with nonlinear incidence and treatment rates

Abhishek Kumar · Nilam

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Abstract In the case of an outbreak of an epidemic, psychological or inhibitory effects and various limitations on treatment methods play a major role in controlling the impact of the epidemic on society. The Monod–Haldane functional-type incidence rate is taken to interpret the psychological or inhibitory effect on the population with time delay representing the incubation period of the disease. The Holling type III saturated treatment rate is considered to incorporate the limitation in treatment availability to infective individuals. This novel combination of the Monod–Haldane incidence rate and Holling type III treatment rate is applied herein to a time-delayed susceptible–infected–recovered epidemic model to incorporate these important aspects. The mathematical analysis shows that the model has two equilibrium points, namely disease-free and endemic. Detailed dynamical analysis of the model is performed using the basic reproduction number R_0, center manifold theory, and Routh–Hurwitz criterion. The results show that that the disease can be eradicated when the basic reproduction number is less than unity, while the disease will persist when the basic reproduction number is greater than unity. The Hopf bifurcation at endemic equilibrium is addressed. Furthermore, the global stability behavior of the equilibria is discussed. Finally, numerical simulations are performed to support the analytical findings.

Keywords Holling type III treatment rate, local and global stability · Hopf bifurcation · Monod–Haldane incidence rate · SIR model · Time delay

Mathematics Subject Classification 34D20 · 92B05 · 37M05

1 Introduction

The widespread and frequent occurrence of many communicable diseases represents a major problem for healthcare workers and policymakers worldwide. Controlling infectious diseases has become an increasingly complex issue in recent years. To control or remove a disease, complete understanding of the dynamics of its progression is required. Based on the observed characteristics of infectious diseases, epidemiologists [1–13] have attempted to construct

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A short study of an SIR model with inclusion of an alert class, two explicit nonlinear incidence rates and saturated treatment rate

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Abstract
In this paper, we present a susceptible–alert–infected–recovered (SAIR) epidemic model with the consideration of two explicit saturated incidence rates and Holling functional type II treatment rate. Awareness about the epidemic may play a vital role in the control of the spread of an epidemic. Hence, an alert compartment has been incorporated into the model. It strives us to take two incidence rates: one from the susceptible class to infected class and another from alert class to infected class. Holling functional type II treatment rate has been introduced to capture the effects of resource limitation in treating infectives. The model has a disease-free equilibrium (DFE), which is locally asymptotically stable when $R_0 < 1$. Using the center manifold theory, we show that DFE exhibits the forward bifurcation at $R_0 = 1$. Stability of the endemic equilibrium has also been analyzed and discussed. Numerical simulations have been done by MATLAB 2012b and the outcomes have been discussed with the help of graphs in the paper.

Keywords Epidemic · SAIR model · Saturated treatment rate · Basic reproduction number · Center manifold theory · Stability

Mathematics Subject Classification 34D20 · 92B05 · 37M05

1 Introduction

In mathematical epidemiology literature, numerous epidemic models [1, 2, 6–9, 11–16, 19–21] like SIR, SEIR, SVEIR, etc. have been proposed for infection transmission and

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Stability of a delayed SIR epidemic model by introducing two explicit treatment classes along with nonlinear incidence rate and Holling type treatment

Abhishek Kumar[1] · **Nilam**[1]

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Abstract

In this article, we analyze the stability of a time-delayed susceptible–infected–recovered (S–I–R) epidemic model by introducing two explicit treatment classes (or compartments) along with nonlinear incidence rate. The treatment classes are named as a pre-treated class (T_1) and post-treated class (T_2). The pre-treatment and post-treatment rates are being considered as Holling type I and Holling type III, respectively. Long-term qualitative analysis has been carried out after incorporating incubation time delay (τ) into the incidence rate. The model analysis shows that the model has two equilibrium points, named as disease-free equilibrium (DFE) and endemic equilibrium (EE). The disease-free equilibrium is locally asymptotically stable when the basic reproduction number (R_0) is less than one and unstable when R_0 is greater than one for time lag $\tau \geq 0$, and when $R_0 = 1$ by Castillo-Chavez and Song theorem, the disease-free equilibrium changes its stability from stable to unstable and the model exhibits transcritical bifurcation. Furthermore, some conditions for stability of the endemic equilibrium are obtained. Finally, numerical simulations are presented to exemplify the analytical studies.

Keywords Epidemic · Delayed SIR model · Holling type treatment rates · Stability · Center manifold theory · Transcritical bifurcation

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EDUCATION

Ph.D. (Mathematics): 2014-Present, Delhi Technological University
Dissertation: Mathematical Modeling in Epidemiology
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RESEARCH INTEREST

Mathematical biology, Mathematical epidemiology, Nonlinear analysis, and Stability theory of dynamical systems.

PUBLICATIONS

- **Abhishek Kumar**, Nilam "Dynamic Behavior of an SIR Epidemic Model Along with Time Delay; Crowley-Martin Type Incidence Rate and Holling Type II Treatment Rate" in International Journal of Nonlinear Sciences and Numerical Simulation, 2019 (Walter de Gruyter GmbH). *(Accepted)*
- **Abhishek Kumar**, Nilam **"**Stability of a delayed SIR epidemic model by introducing two explicit treatment classes along with nonlinear incidence rate and Holling type treatment**"** in Computational & Applied Mathematics, 2019. https://doi.org/10.1007/s40314-019-0866-9 (Springer)
- **Abhishek Kumar**, Nilam, Raj Kishor **"**A Short Study of an SIR Model with Inclusion of an Alert Class, Two explicit Nonlinear Incidence Rates and Saturated Treatment Rate**"** in SeMA Journal, 2019. https://doi.org/10.1007/s40324-019-00189-8 (Springer).
- **Abhishek Kumar**, Nilam "Mathematical Analysis of a Delayed Epidemic Model with Nonlinear Incidence and Treatment Rates" in Journal of Engineering Mathematics, 2019. https://doi.org/10.1007/s10665-019-09989-3 (Springer).
- **Abhishek Kumar**, Nilam "Dynamical Model of Epidemic Along with Time Delay; Holling Type II Incidence Rate and Monod–Haldane Type Treatment Rate" in Differential Equations and Dynamical Systems Vol. 27 (1-3), 2019. (Springer)
- **Abhishek Kumar**, Nilam "Stability of a Time Delayed SIR epidemic Model Along with Nonlinear Incidence Rate and Holling Type-II Treatment Rate" in International Journal of Computational Methods Vol. 15 (6), 2018. (World Scientific)

CONFERENCES/ WORKSHOPS ATTENDED/ PAPER PRESENTED

- Presented a paper entitled “A SAIR model with two explicit nonlinear incidence rate and saturated treatment rate” at International Conference on Recent Advances in Pure and Applied Mathematics held at DTU Delhi, India, October 23-25, 2018.
- Attended an Indo-German Workshop on “Optimal Control, Inverse Problem and Their Applications” at IIT Delhi, India, February 07-09, 2018.
- Attended an international conference on “Current Trends in Theoretical and Computational Differential Equations with Applications” at SAU, Delhi, India, December 01- 05, 2017.
- Attended a National Workshop on “Mathematical Modelling and Computational Techniques using MATHEMATICA” at University of Delhi, India, March 30-31, 2017.
- Presented a paper entitled “Mathematical control strategy with time-dependent contact rate for contagious disease” at 5th International Conference on “Advancements in Engineering and Technology” held at BGIET Sangrur, Punjab, India, March 24-25, 2017.
- Presented a paper entitled “A mathematical model with time-dependent contact rate to control the spread of H1N1 ” at International Conference on Innovations & Sustainable Development in Sciences, Management & Technology held at SSIPMT, Raipur, India, March 25-26, 2017
- Presented a paper entitled “A delayed SIR model with nonlinear transmission rate and Holling type-II recovery for infectious disease” in International Conference on “Innovative Approach in Applied Physical, Mathematical/ Statistical, Chemical Science and Emerging Technology for Sustainable Development” at JNU, Delhi, India, January 15, 2017.
- Attended an advanced workshop on “Finite Difference Methods For Differential Equations” at SAU, Delhi, India, March 13-17, 2015.