NONLINEAR DYNAMICS AND SIMULATION OF INFECTIOUS DISEASES IN HUMANS

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Delhi Technological University

for the Award of Degree of

Doctor of Philosophy

In

Mathematics

By

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(Enrollment No.: 2K16/Ph.D/AM/11)

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DECLARATION

I declare that the research work reported in this thesis entitled "**Nonlinear Dynamics and Simulation of Infectious Diseases in Humans"** 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 embodied 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, graphs or other information, unless specifically acknowledged.

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CERTIFICATE

This is to certify that the research work embodied in the thesis entitled **"Nonlinear Dynamics and Simulation of Infectious Diseases in Humans"** submitted by **Ms. Kanica Goel** with enrollment number **(2K16/Ph.D/AM/11)** is the result of her original research carried out in the Department of Applied Mathematics, Delhi Technological University, Delhi, 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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Date : July 2, 2021 **(Kanica Goel)**

Place : Delhi, India.

To my parents Mr. Anil Kumar Goel & Mrs. Pratibha Goel

and

My Teachers . . .

Contents

Abstract

Infectious diseases impose a critical challenge to humans and remain to be a matter of global concern. Sometimes contagious diseases that had become rare or had been only local suddenly start occurring worldwide, for instance, SARS, Ebola, and Zika fever. The last two decades have seen several large-scale epidemics outbreaks such as Ebola, SARS, Zika virus, and swine flu, which leads to low socioeconomic status and inadequate access to health care. The mathematical modeling of infectious diseases has become a vital tool to understand, predict and control the spread of contagious diseases.

The present thesis aims to discuss the various aspects of transmission dynamics of infectious diseases through time-delayed mathematical epidemic models. The inclusion of time delay in the study of epidemiology is an important aspect. Persons with asymptomatic infections play an essential role in spreading infectious diseases, especially as they are unaware of their illness and take no special hygiene precautions. Thus, the study of disease-transmission dynamics involves time delay, which needs to be considered for practical purposes. The time delay may arise due to delays caused by the latency in a vector and delay caused by a latent period in the host. Therefore, this thesis comprises the Delay-differential equations for formulating the epidemic models. We have proposed time-delayed epidemic models with different compartments and analyzed them mathematically for positiveness, boundedness, and stability to provide the control strategies of emerging or re-emerging infectious diseases. The mathematical analysis and simulations of the proposed models have been done using the Routh-Hurwitz stability criterion, Descartes' rule of signs, Lyapunov direct method, and Mathematica 11.

Keywords: Infectious diseases (Epidemic); Delay differential equations (DDEs); Latent period; Nonlinear incidence rates; Nonlinear treatment rates; Stability; Bifurcation.

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Chapter 1

Introduction

Infectious diseases have a substantial impact on community health worldwide and remain a significant cause of death and suffering in developing countries [35]. The human population's presence is large enough to sustain and amplify parasites, subsequently contributing to increased disease. As disease agents adapt, survive, and evolve, the emergence of new diseases and re-emergence of existing diseases have become a significant worldwide problem [29, 35]. Therefore, controlling infectious diseases has become an increasingly complex issue in recent years. This introductory chapter provides some elementary information about the infection mechanisms, the control mechanism of epidemics, the role of mathematical models in epidemiology, and the motivation behind the work carried out in this thesis.

1.1 Infectious diseases

"An infectious disease is a disease due to a specific infectious agent or its toxic products that arise through the transmission of that agent or its products from an infected person, animal, or reservoir to a susceptible host, either directly or indirectly through an intermediate plant or animal host, vector, or the inanimate environment" [60]. Various microbes or pathogens cause infectious diseases. Most of them are generally microorganisms, and few of them are observable by naked eyes. The most commonly known pathogens are different types of viruses and bacteria. Fungi and Protozoa are also known as pathogens that are responsible for many diseases. Diseases caused by these pathogens

are termed as "infectious" as these pathogens can be transmitted from one infected person to another non-infected person. The most common and well-known example of such diseases is influenza or flu caused by some kinds of viruses. Pathogens can be transmitted either directly or indirectly. Direct transmission involves the spread of pathogens by direct body-to-body contact, such as mother to child as exemplified with HIV, Zika, and syphilis, touching (MRSA), kissing (herpes simplex virus), and sexual contact (human papillomavirus or HPV). Indirect contact transmission occurs when pathogens are transferred between individuals via a contaminated intermediate person, object, or environmental surface [53]. Many infectious diseases (for example, HIV, mumps, measles, rubella, smallpox, malaria) are still prevalent at local or global scales and threaten public health. An outbreak of an infectious disease affecting a disproportionately large number of individuals in a population, community, or region within a short period is known as an epidemic.

1.2 Mode of transmission

Infectious diseases can spread in various ways, and pathogens cause infections by different modes of transmission. Contact is one of the most critical and frequent methods of transmission of infectious diseases. It can be either direct or indirect. Direct contact involves the physical transfer of microorganisms to a susceptible host from an infected or colonized person. Direct contact includes a large droplet spread of infectious agents. Other direct contact transmission examples are kissing, shaking hands or other skin contacts, sexual contact, and soil contact. Indirect contact occurs when organisms from an infected host or other reservoir are transmitted to a susceptible host via a contaminated intermediate object. Indirect contact transmission may be airborne, vector-borne, or vehicle-borne. Many diseases, e.g., influenza, SARS, are airborne and can be transmitted through the air. The airborne infection spreads from an infected person to an uninfected person through sneezing, cough, and even laughter. The microbes that are discharged from an infected person may remain on the dust particles or any other medium. Infection may occur when these microbes are inhaled or reach an uninfected person's mucus membrane through body contact. Hand-shaking could also be a potential way for transmission of infections.

A substantial number of diseases are sexually transmitted diseases that are trans-

mitted through contaminated blood and semen, childbirth, or breastfeeding. HIV and other sexually transmitted infections such as herpes, gonorrhea, trichomoniasis, syphilis, and chlamydia cause noteworthy infection and mortality. Many of these diseases, such as AIDS and Herpes, cannot be cured and last forever, which pose severe social and economic consequences and are most troublesome to public health. Due to longer infectious life, infected individuals with sexually transmitted diseases may contribute to increased infections and remain a significant problem in preventing diseases. Another life-threatening facet of these diseases is that the infected person may not produce any symptoms; consequently, the infected individual may transmit infection unknowingly.

1.3 Prevention and control of infectious diseases

Preventing infectious diseases is the central goal to minimize the outbreak impact and spread to a larger population. The two main strategies for controlling epidemics of contagious illness are reducing the number of cases through preventive activities such as raising awareness, early case identification, and reducing mortality due to disease through effective therapy. These actions should be instituted quickly and should not be delayed while waiting for laboratory confirmation of the disease in question. The prevention of the spread of infectious diseases can be achieved by reducing contact. However, in modern life, with increased interactions among individuals, this is not easy to accomplish. Therefore, awareness, vaccination, and drug therapy are the piers in preventing and controlling infectious diseases.

1.3.1 Awareness

The last two decades have seen several large-scale epidemics outbreaks such as Ebola, SARS, Zika virus, and swine flu, which lead to low socioeconomic status, and inadequate access to health care. People get information about these outbreaks quite quickly due to significant advances in social media, which can have an insightful effect on the actual epidemic dynamics [69, 121]. Therefore, at the beginning of an epidemic outbreak, the initial step is to make the individuals aware of the disease and its preventive methods. Awareness leads to sharing necessary information about the condition to the general population, getting thought, making the individuals familiar with the disorder, and providing the most substantial protection against infectious diseases. Thus, awareness about the spread of a disease is a valuable ally in affecting susceptibles' behavior, mitigating

further infection.

Awareness programs can alert the vulnerable population towards the contagious disease [99]. Suppose vulnerable individuals have adequate knowledge and accurate information about the infection. In that case, they take preventive measures such as regular hand sanitization, face masks, wear hand gloves, vaccination, and even quarantine, reducing the impact of illness. For instance, ranging from the plague outbreak in the English village of Eyam in 1665–1666 [34], where the town completely sealed itself off to prevent further transmission of plague, to more recent outbreaks of swine influenza [69] and Ebola [121]. During the 2003 SARS outbreak, the Chinese Southern Weekend newspaper spread the instant message, "There is a fatal flu in Guangzhou" 126 million times in Guangzhou alone, which affected people's behavior to take necessary preventive measures [49]. This figure remains a distinct difference to the nearly low number of 5,327 cases recorded in the entire China [36].

In rural health services, the challenge of the government health care scheme is that there are numerous gaps in primary health services, and the health care facilities are mainly urban-centric. The rural residents may have low health knowledge awareness, and their receiving way of health knowledge can be traditional and straightforward. Knowledge level increases with higher education level; thus, education level is one of the main factors in making people aware and further adopting preventive measures. Therefore, many people are not fully but partially aware of the spread and control of infectious diseases. Due to partial awareness, some individuals often medicate themselves adopting antibiotics, even when advised against doing so, which weakens their immune system and makes them at a high risk of catching the infection [72]. Therefore, complete awareness about the cycle of disease and the utilization of appropriate precautions and adequate decontamination procedures are vital. Full awareness of the disease in humans develops a habit of taking preventive measures against it. They follow the instructions given by health workers and the government and lower their risk of becoming infected.

The spread of the disease can also be controlled by vaccination. But, immunizations can never be completely safe, and there is always a risk of some side-effect. Also, it is difficult to vaccinate all individuals due to various limitations. Even some fatal diseases such as AIDS, Malaria, Chikungunya, Plague, and Dengue have no vaccination; only a

person's awareness can prevent the spread of these diseases efficiently and effectively. For instance, the habit of using mosquito nets and mosquito coils helps in preventing Dengue and Chikungunya [93, 129].

1.3.2 Drugs

The role of drugs in providing cures for infectious diseases can also significantly reduce disease transmission. It can be taken either as Pre-Exposure Prophylaxis (PrEP) or Post-Exposure Prophylaxis (PEP). Healthy people who are likely to expose to infection are endorsed to take PrEP, while the infected people are recommended to take PEP to halt or lessen transmission. For instance, in malaria disease, when people intend to travel to a malaria-infected area, they are recommended to take malaria medication, which could prevent voyagers from malaria infection during their travel whenever bitten by a contaminated mosquito [114]. Effective vaccines have not been developed for many diseases such as HIV. Typically, vaccination development is a long and complex process. Drug-oriented interventions can be an elective technique for lessening the contamination trouble when vaccines are not available. In HIV, notable progress has been accounted for PrEP and PEP utilization [67, 77, 79, 96]. Genuinely broad outcomes have been published, showing a huge decrease of viral heaps of HIV-tainted people related with antiretroviral [79,96]. The expulsion of viral burden is connected to a lower transmission likelihood. Hence a drug-oriented intervention could be a possible way to deal with relieving the burden of HIV disease.

1.3.3 Vaccination

One methodology to control the spread of infectious diseases is vaccination. Vaccination plays a vital role among the health interventions aimed at reducing the spread of contagious diseases thanks to its safety and cost-effectiveness. Indeed, high immunization take-up levels have brought about radical decreases in numerous vaccine-preventable infectious diseases or even their eradication, as in the very notable instance of smallpox [66]. The cowpox vaccine, introduced by Edward Jenner in 1796, is known as the first vaccine. After this, several effective campaigns have been conducted against many contagious diseases [74]. Vaccines save millions of human lives and serve to be a highly effective method to prevent infectious diseases. In the United States, before introducing the first measles vaccine in 1963, around 400,000 measles occurrences used to be reported

every year [33]. Child diseases such as Polio, mumps, and rubella also cause extensive mortality and morbidity. These diseases are no more prolonged epidemic after the implementation of the vaccines [33]. Vaccines against influenza transmission also have a successful history, which is the most common infectious disease worldwide. Before the development of flu vaccines, controlling an influenza pandemic was a terrible mission. It was assessed that around 20-50 million individuals worldwide died in the Spanish flu outbreak in 1918-19. A century later, the 2009-10 pandemic global death toll was only around 300,000 [101]. The development of the vaccine has reduced the casualty rate to such a level. Nevertheless, a critical aspect of vaccination is its level of safety as far as viability in preventing the illness and the duration of the induced immunity. Some vaccines may be highly effective, e.g., measles [127], while others may not, as is the case of varicella [100]. The effectiveness and levels of protection provided by a vaccine may naturally decrease over time because of several medical conditions (medications, aging, low immune system, etc.) and the alteration and evolution of infectious diseases; for example, the flu virus [128] can change very rapidly, meaning that last year's flu vaccine is unlikely to protect individuals from virus strains circulating this year. In contrast, the measles virus [127] prevented by the measles– mumps–rubella (MMR) vaccine hardly changes from year to year, indicating that it is as likely to protect individuals today as it was ten years ago. Some vaccines minimize the infection risk but do not entirely prevent a vaccinated individual from catching and transmitting the infection. These imperfect vaccines may not completely prevent infection but could decrease the likelihood of becoming infected or reduce its consequences, thereby lessening the infectious disease burden. We will address the problem of imperfect vaccination exhaustively in Chapter 7.

1.4 Mathematical epidemic model

Infectious diseases remain a significant challenge for human survival. Therefore, it is indispensable to analyze the dynamics of disease development to control or eliminate the disease. The dynamics of infectious diseases can be investigated using mathematical models. The mathematical modeling of infectious diseases is a valuable tool that can enhance understanding of the disease's spread mechanisms and help make health decisions more cost-effective and accurate than the experimental studies. By analyzing the model, we can foresee the future course of an outbreak up to a large extent to evaluate

control methodologies. Many researchers have developed mathematical models in epidemiology to facilitate the formation of public health policies [1,4,10,29,31,32,40]. The basic principle of these mathematical models was to investigate the underlying factors causing the disease, its development and hence foresee the future course of action. The mathematical models are categorized into two types: Deterministic and Stochastic. The deterministic model determines every set of variable states uniquely by parameters and the initial conditions and provides the same outcome for the same set of parameters with the same initial conditions. A stochastic model is a mode in which randomness is present, and variable states are not described by unique values but by probability distributions. Stochastic models show randomness and provide different outcomes for the same set of parameters with the same initial conditions. In this thesis, we study the various aspects of infectious diseases using deterministic modeling. The general idea for most deterministic models is to look at a so-called compartmental model, in which the population is divided into compartments based on disease status.

Epidemiological models are also known as compartmental models since they assume that the entire host population can be divided into compartments. In 1927, Kermack and McKendrick [1] gave the basic compartmental SIR epidemic model in which they describe the transmission mechanism of the infectious disease by dividing the total population into distinct subclasses according to their epidemiological status. Their model involves three compartments (or classes): Susceptible, Infected, and Recovered. The susceptible class, usually denoted by S, consists of those individuals who are healthy but can contract the disease. The infected class I is the class of those individuals who have contracted the disease and are now infectious and can transmit the disease to susceptibles through contact. As time progresses, infectious individuals lose the infectivity and move to either a removed compartment R (by death) or recovered compartment R (either by suitable treatment or auto-recovery by the immune system). Recovered individuals are resistant to infectious microbes and thus do not get the infection again in permanent immunity. Thus, the Kermack's and McKendrick's SIR epidemic model is given by the following

system of ordinary differential equations:

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

where, β and γ are the transmission and recovery rates, respectively. This model delineates how subpopulations of susceptible, infected, and recovered classes progress without considering the host population's demography. The researchers have employed the idea of population compartments broadly in epidemic models [16, 29, 34]. Different epidemic models, relying upon the invulnerability against the contamination, are developed. For example, in 1976, Hethcote [7] studied the dynamics of the SIS and SIRS epidemic models. The SIS model looks at the disease without immunity wherein the recovered individual will get the infection again and become susceptible. In contrast, the SIRS model is the case of a disease with temporary immunity.

In the study of the transmission of infectious diseases, the incidence rate has a vital role as it determines the number of infectives per unit of time. In model (1.1), the term $\beta S(t)I(t)$ represents the bilinear (or mass action) incidence rate which is assumed to be proportional to the product of the infected and susceptible members of the population. In the bilinear incidence rate, the number of infectives increases linearly, which might be real for a small population of infected individuals, but impractical for a large number of infectives. Therefore, several studies are devoted to considering nonlinear incidence rate for disease transmission dynamics [123, 134–137, 139, 141, 142]. Nonlinear incidence rate permits the incorporation of social/behavioral changes and prevents unbounded contact rates. Thus, changing the form of incidence rate can potentially fill the gap in studying the disease transmission behavior of the system. We explore some of the different nonlinear incidences.

(i) **Holling type II:** This incidence rate was proposed by C. S. Holling in 1959 [3], and it is also known as the saturated incidence rate. It is of the form $f(S,I) = \frac{\alpha SI}{1+\beta I}$, where $\alpha,\ \beta > 0$. "In Holling type II, for any disease outbreak, its incidence is first very low and then grows slowly with an increase in infection. Further, when number of infected individuals is very large, the infection reaches to its maximum due to crowding effect" [123].

(ii) **Beddington-DeAngelis type:** This incidence rate was introduced by Beddington et al. [5] and DeAngelis et al. [6] in 1975 independently. "It is of the form $f(S,I) = \frac{\beta SI}{(1+\alpha S+\gamma I)}$, where $\beta,\ \alpha,\ \gamma > 0$. 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 to infectives. This incidence rate considers the impact of inhibition among infectives in case of the low density of susceptible populations" [123]. Also, we can derive the other incidences from this incidence, such as bilinear incidence rate if $\alpha = \gamma = 0$; saturated incidence rate for the susceptible if $\gamma = 0$; and saturated incidence rate for the infectives if $\alpha = 0$.

(iii) **Crowley Martin Type:** In 1989, P. H. Crowley and E. K. Martin introduced the incidence rate of the form $f(S,I) = \frac{\alpha SI}{(1+\beta S)(1+\gamma I)}$, where $\alpha,\ \beta,\ \gamma > 0$. This incidence rate contains other forms of incidences analogous to the Beddington-DeAngelis incidence form. "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 effect as mentioned earlier" [123].

Treatment is vital to cure the infection and prevent the development of resistant bacteria. Therefore, consideration of the treatment rate in the epidemic model is of great importance. In 2004, Wang and Ruan [39] studied the SIR epidemic model with bilinear incidence rate and the constant treatment rate to know the impact of treatment capacity on disease transmission dynamics. Their model's critical observation is that the periodic oscillations in diseases can be seen with a constant treatment rate, whereas the model without the treatment is globally stable. This kind of treatment rate is appropriate when there are small numbers of infectives and treatment resources are sufficient and inappropriate when the number of infected individuals is large, and treatment resources are limited. Therefore, in 2012, Zhou and Fan [102] improved the treatment rate by considering a saturated treatment rate and explore the SIR epidemic model to understand the effect of the limited medical resources and their supply efficiency on the transmission of infectious diseases. To control the disease, most researchers focus on a nonlinear type treatment rate. Dubey et al. [106, 123, 125] introduced the nonlinear treatment rate as Holling Type II, Holling Type III, and Holling Type IV in their model and proposed nonlinear dynamics to control the epidemic. In this thesis, we explore the impact of various nonlinear treatment rates on disease transmission models.

Authors have stressed that apart from the nonlinearity in the incidence rate and infection treatment rate, public awareness is also an important tool to reduce the spread of disease. The impact of information and awareness on the spread of epidemics has been studied by many authors [54, 84, 86, 94, 111, 112, 115, 119, 122]. Another methodology of controlling infectious diseases is vaccination. But, sometimes, vaccination can be temporary. Therefore, many studies have dealt with epidemic models in mathematical epidemiology literature, including imperfect vaccination [41, 92, 97, 110, 126]. We will discuss the impact of awareness and vaccination in detail in Chapters 5, 6, and 7, respectively.

Delay differential equation (DDE) plays a significant role in estimating both past and ongoing epidemics and the structure of future-focused control interventions. It can be said with a high level of conviction that when a disease emerges, there will be an initial delay in recognizing it. Thus, the time delay is an influential parameter in the dynamical behavior of infectious diseases, and it can change the dynamical system's behavior. Delay differential equations (DDEs) can have more affluent dynamics than ordinary differential equations and better fit the real-world situation. Driver [8] gave a solid introduction to this. Time delays are generally used to model the condition in which an individual may not be infectious until some time after becoming infected. The latency period of contagious diseases, which is defined as the period between exposure and infection since the pathogen is present in a latent stage without clinical symptoms or signs of infection in the host, can be modeled by DDE. In the context of epidemiology, various factors cause the delay. The most prominent reasons for considering time delay are (i) the infection's latency in a vector and (ii) the infection's latency in an infected host. In these cases, some time should elapse before the infected host's infection level, or the vector will rise to an adequately significant level to further transmit the disease. Delay is challenging to deal with mathematically because straightforward incorporating it into a mathematical model generally leads to delay differential equations which are difficult to handle mathematically. A commonly used palliative comprises an extra compartment such as an exposed class (E) of the SEIR model held responsible for the delay. While this approach is mathematically suitable as it prompts a higher-order system of ordinary differential equations rather than an equation with a delay, it is also biologically questionable. It generally implies an assumption that the delay is exponentially distributed, whereas, in an epidemiological context, it often appears that an assumption of a constant delay is more reasonable. Kuang [22] discusses delay and distributed delay differential equations

in the context of population models, including predator-prey systems with logistic-type equations that can be applied to epidemic models. Brauer and Castillo-Chavez have an excellent introduction to epidemic models in [32] with extensions to delay systems. Ma and Song [43], McCluskey [80], and Rost and Wu [63] analyzed the SIR and SEIR models with discrete delays. Further, Meng et al. [78], Xu and Ma [75], Zhao et al. [58] studied the delay epidemic models with nonlinear incidence. Most of these studies proved the global stability of disease-free equilibrium. McCluskey [71, 80] gave excellent proofs for the endemic equilibrium's global stability for two classes of SIR and SEIR epidemic models with both finite time delay and infinite time delay. In literature, many authors study the impact of time delay on the epidemic model. Motivated by the work mentioned above, this thesis studies the impact of time delay as a latent period in the dynamical study of epidemic models.

1.5 Basic reproduction number

The basic reproduction number is a crucial concept in studying disease transmission dynamics and one of the most valuable ideas that mathematical thinking has brought to epidemic theory. It often serves as a threshold parameter that predicts whether an infection will spread or die out. The basic reproduction number R_0 is defined as "the average number of secondary infections caused by one infected individual during their entire infectious period in a completely vulnerable population" [33]. In this framework, it is clear that the critical parameter is the number of secondary cases generated by the initial case. If this number is greater than unity, then there is a positive chance of a large outbreak affecting nearly the total population. Thus, it is essential to know whether this number is much or a little above the threshold value of one for evaluating control programs. It will determine the necessary amount of control strategy needed to reduce the corresponding parameters to achieve a specified goal sufficiently. The parameter contact rate of susceptible and infective individuals has a significant effect on R_0, as higher the influential contacts, higher the possibility of a new infection. Another influential factor is the duration of infectiousness. People with extended infectiousness periods will come in contact with more peoples and therefore possibly infect more peoples.

1.6 Stability analysis

Mathematical models become increasingly intricate when a higher degree of nonlinearity is considered to mark real-world situations. It becomes almost impossible to find a definitive solution to these models. The reasonable approximate solution to these complicated models with fixed parameters can be obtained using numerical simulations, but the general solution remains unknown. In this situation, the stability analysis plays a crucial tool in getting a sense of the solution's behavior. Even the long-time behavior of the model solutions can be assessed by stability analysis. Generally, two types of stability analysis are extensively used in the literature: local and global. Local stability describes the behavior of the model's solution around an equilibrium point, whereas global stability describes the solution's behavior in the whole domain.

Delay differential equations are often of interest to determine whether or not the delay values affect a steady-state's stability. 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 the roots of characteristic equations. If all the roots have a negative real part, the steady-state is stable. When we vary the delay values, if one of the roots changes from having a negative real part to having a positive real part because of the delay, it implies that the steady-state will become unstable. This is equivalent to having the root crossing the imaginary axis (imagine the root as a graph with a real part on the x-axis and an imaginary part on the y-axis). Therefore, if the root turns positive, there must be a purely imaginary part (i.e., the intersection between the graph of the root and the imaginary axis exists).

In this thesis, the Routh-Hurwitz (R-H) criterion and Lyapunov direct method [28] are mainly used for the stability of model's equilibria. The Routh-Hurwitz (R-H) criterion is helpful 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 out of that neighborhood. The Lyapunov direct method can be helpful 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 of finding out such energy functions termed 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 cases, with a planned procedure [125].

1.7 Contents of thesis

The thesis entitled "Nonlinear Dynamics and Simulation of Infectious diseases in Humans" contains eight chapters followed by a conclusion & future scope, and a bibliography. The thesis is organized as follows:

Chapter 1 Chapter 1 is introductory, which gives a general background of epidemic modeling theory, basic terminology, essential concepts, and types of models. This chapter aims to provide the chronological development done in epidemiology and the motivation behind the thesis's work.

Chapter 2 In chapter 2, the dynamical behavior of a susceptible-infected-recovered (SIR) epidemic model is being proposed and analyzed by incorporating time delay as the latent period, the nonlinear incidence rate, and the nonlinear treatment rate. The nonlinear incidence rate is considered as the Beddington-DeAngelis type functional response. This incidence rate has a significant role in studying disease transmission dynamics as it includes inhibition measures taken by both susceptible and infected individuals. If individuals are familiar with the diseases and are acquainted with the transmission modality of infection, they can take necessary preventive measures to avoid infections. These measures are called the measure of inhibition. When a disease emerges, there will be an initial delay in recognizing it. Therefore, to study the more natural disease transmission phenomenon, we incorporate the time delay into the Beddington-DeAngelis functional type incidence rate, which is an essential parameter in the dynamical behavior of infectious diseases and can change the behavior of the dynamical system. The treatment rate plays a substantial role in preventing and controlling the spread of epidemics. Therefore, to provide a control strategy for infectious diseases, we consider a nonlinear saturated functional type treatment rate, which includes the fact of limited treatment availability. For the dynamics of the model, we discuss the existence and stability behavior of the model for equilibriums. The existence of a Hopf bifurcation is also discussed. The model's numerical results demonstrate the impact of time delay, incidence, and treat-

ment rates on the infected population.

The work reported in this chapter has been published entitled **"A mathematical and numerical study of a SIR epidemic model with time delay, nonlinear incidence and treatment rates"** in *Theory in Biosciences, 2019 (Springer).*

Chapter 3 This chapter proposes a mathematical SIR epidemic model with Beddington-DeAngelis type incidence rate with the inclusion of a latent period and a nonlinear treatment rate to study the dynamics of a SIR model. The treatment rate is considered as the Holling type II treatment rate, which refers to the condition that when there is a large number of infected people, then the treatment capacity reaches its maximum because of limited treatment facilities that mimic the more realistic phenomena. For an outbreak of epidemic disease, the treatment capacity of Holling type II is initially very slow, which develops gradually with improved treatment facilities. Stability analysis is resorted to getting a sense of the behavior of solutions. A precise indication of the bifurcation phenomenon has been given using center manifold theory, ensuring that either forward or backward bifurcation occurs. The backward bifurcation demonstrates that the disease-free equilibrium coexists with the endemic equilibrium when the basic reproduction number is less than one. It has important qualitative implications since reducing the basic reproduction number below one is not sufficient to eradicate the disease from society. Our theoretical results suggest that backward bifurcation depends on infected individuals' therapeutic treatment, which shows the importance of considering the Holling type II treatment rate. The nonlinearity in the epidemic model due to the latent period (time delay) can give rise to periodicity via a Hopf bifurcation. The numerical simulations validate the theoretical results.

The work reported in this chapter has been published entitled **"Stability behavior of a nonlinear mathematical epidemic transmission model with time delay"** in *Nonlinear Dynamics, 2019 (Springer).*

Chapter 4 In the beginning phase of an outbreak, disease follows either an exponential or generalized exponential growth. However, it slows down and reaches its maxima as the increasing number of cases reaches its inflection point, and the daily incidence curve reaches its extreme. Thus, the growth pattern departs from the (sub-) exponential path and follows a logistic growth rate. To capture this situation in the epidemic

model, in this chapter, we propose the susceptible-infected-recovered (SIR) epidemic model with the logistic growth of susceptible individuals. We incorporate Crowley Martin type incidence rate, Holling type III treatment rate, and two explicit time-delays: a time delay in the incidence rate, which represents the latent period; a time delay in the nonlinear treatment function, which measures the impact of delay in providing the appropriate therapy to infectives. The numerical simulations verify the analytical results and demonstrate the significant role of the following nonlinearities: capturing time lags between the exposure of disease, onset of its symptoms, and then providing treatment to infectives; susceptibles' and infectives' protection level against the infectious diseases; the limitation in the availability of the medical resources. The latent period and the delay in treating patients have a significant impact on the number of infected individuals. These delays result in spreading infections at a high rate, and so, the disease stays for a longer period. Considering the time delays as the bifurcation parameters can affect the obtained equilibrium's stability. When the delay is suitably small, the system reaches its steady-state, but as it crosses the critical value, it will produce a limit cycle and destabilize the endemic stability, making it difficult to control the spread of infection.

The work presented in this chapter is communicated for publication.

Chapter 5 In this chapter, we investigate the effect of awareness during an epidemic. We propose a time-delayed epidemic model by incorporating a class of aware susceptible individuals in the SIR compartmental model. We considered the Michaelis-Menten functional type nonlinear incidence rates for unaware and aware susceptibles with the latent period and a saturated treatment rate for infectives. Michaelis-Menten functional response type incidence rate is suitable when the number of adequate contacts per infective in unit time grows less rapidly as the total population increases. We perform mathematical analysis that allows long-term qualitative predictions of outbreaks and the persistence of the disease. The numerical experiments show the significance of the model's variables and parameters and suggest strategies that could prevent infection.

The work presented in this chapter is communicated for publication.

Chapter 6 This chapter is an extension of Chapter 5. Here, we introduce fully aware and partially aware susceptible compartments into the SIR epidemic model due to heterogeneous protection levels of individuals and extend the epidemic model to include the

behavioral change of susceptibles, which can change the transmission patterns and reduce the prevalence of disease to a more extent. We consider three specific nonlinear incidence rates of unaware susceptibles, fully aware susceptibles, and partially aware susceptible, respectively, with the inclusion of time delay as a latent phase. Also, with awareness, treatment of infectives is essential to mitigate the infection. Therefore, we consider the nonlinear saturated treatment rate, which includes limitations in the availability of resources. After formulating the nonlinear time-delayed mathematical epidemic model, we perform stability analysis to demonstrate the eradication or persistence of the disease and validate the theoretical results numerically. The results can help to understand the role of varying protection levels of susceptibles in the transmission pattern of infectious diseases, suggesting the control strategies to prevent the spread of infections at a massive scale.

The work reported in this chapter has been published entitled **"Nonlinear dynamics of a time-delayed epidemic model with two explicit aware classes, saturated incidences, and treatment"** in *Nonlinear Dynamics, 2020 (Springer)*.

Chapter 7 This chapter presents a time-delayed susceptible-vaccinated-infected-recovered-susceptible (SVIRS) compartmental epidemic model to study the impact of the imperfect vaccine. We incorporate the Holling type II incidence rate with a latent period and saturated treatment rate and perform qualitative analysis through the stability and bifurcation theory approach using center manifold theory. Our results suggest that the imperfect vaccine and a saturated treatment rate may lead to backward bifurcation, but at the same time, we emphasize that these facilities reduce the size of the infected population. High vaccine take-up levels brought about radical decreases in infectious diseases. If a vaccine can be made, which is completely effective, this plausibility does not emerge, and a program that reduces the contact rate can moreover control infection without inciting backward bifurcations.

The work reported in this chapter has been published entitled **"A deterministic time-delayed SVIRS epidemic model with incidences and saturated treatment"** in *Journal of Engineering Mathematics, 2020 (Springer)*.

Chapter 8 This chapter contains the conclusion of the work done and the future scope of the problems discussed in the thesis.

Chapter 2

Dynamics of a time delayed nonlinear SIR epidemic model with Beddington-DeAngelis incidence and saturated treatment rates

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In this chapter, a novel nonlinear time-delayed Susceptible - Infected - Recovered (SIR) epidemic model with Beddington-DeAngelis type incidence rate and saturated functional type treatment rate is proposed and analyzed mathematically and numerically to control the spread of the epidemic in the society. Analytical study of the model shows that it has two equilibrium points; disease-free equilibrium (DFE) and endemic equilibrium(EE). The stability of the model at DFE is discussed with the help of a basic reproduction number, denoted by R_0 and it is shown that if the basic reproduction number R_0 is less than one, the DFE is locally asymptotically stable and unstable if R_0 is greater than one. The stability of the model at DFE for $R_0 = 1$ is analyzed using center manifold theory, revealing a forward bifurcation. We also derived the conditions for the stability and occurrence of Hopf bifurcation of the model at endemic equilibrium. Further, to illustrate the analytical results, the model is simulated numerically.

2.1 Introduction

Mathematical analysis and modeling of infectious diseases play a crucial role in studying a wide range of infectious diseases to understand the transmission dynamics better and have the capacity to influence expectations; to decide and assess control strategies. Authors around the world have proposed different kinds of epidemic models such as SI (Susceptible - Infected) [26], SIS (Susceptible - Infected - Susceptible) [25], SIR (Susceptible - Infected - Recovered) [132], SIRS (Susceptible - infected - Recovered - Susceptible) [21], SEIR (Susceptible - Exposure - Infected - Recovered) [106, 109], SVEIR (Susceptible - Vaccinated - Exposure - Infected - Recovered) [51] and many more, to understand the dynamics of disease transmission. In the mathematical epidemiological literature, several authors have studied the epidemiological models with latent or incubation period because many diseases have a latent or incubation period, during which the susceptible individual becomes infected but is not yet infectious. Such latency in disease transmission can be modeled by a delay differential equation. Delay differential equations (DDE) have been a very successful tool to capture the effect of a varying infectious period in a range of SIR, SIS, SIRS, and other epidemic models. Hethcote and van den Driessche [25] studied an SIS epidemic model with constant time delay, which accounts for the duration of infectiousness. Song and Cheng [44] studied the impact of time delay on the stability of the positive equilibrium, as a resultant of which, conditions have been stated for the asymptotical stability of the endemic equilibriumn for all delays. Xu et al. [76], Khalid Hattaf et al. [107] and Kumar and Nilam [131, 132, 135, 141] considered the effect of time delay on SIRS and SIR models respectively and provided the conditions for the stability of their proposed models.

The incidence rate of a disease is the number of new cases per unit time and plays a crucial role in studying the transmission of disease dynamics. Several authors suggested different types of incidence rates. Firstly, the bilinear incidence rate βSI [1, 4, 20, 32, 82] is based on the law of mass action, describes the situation that if the number of susceptibles increases, the number of individuals infected per unit of time increases, which is not realistic. In reality, however, by the impact of media, open mindfulness, or individual experience, people apply careful steps that decrease the contact number or the transmission potential. Since nonlinearity in the incidence rates has been seen in disease transmission dynamics, it has been proposed that the standard bilinear incidence rate shall be modified

into a nonlinear incidence rate by numerous authors [12, 52, 135, 139, 141]. Several authors such as Anderson and May [10], Wei and Chen [61], Zhang et al. [62], Li et al. [73], Li and Muldowney [24], Korobeinikov and Maini [45], Xu and Ma [76], Capasso and Serio [12] suggested different types of nonlinear incidence rates, and they incorporated these incidence rates in their model and studied the disease dynamics. In 1975, Beddington [5] and DeAngelis [6] independently introduced nonlinear incidence rate known as Beddington-DeAngelis type incidence rate. Later, some authors [81, 105, 123] used this incidence rate in their epidemic models. In the present chapter, we introduce the incidence rate as Beddington-DeAngelis type to contribute to the nonlinear dynamics of infectious disease. Since the nonlinear type incidence rate alone can not wholly determine the transmission of the disease dynamics, the inclusion of time delay must be considered for a more realistic model. Therefore, we incorporate time lag into Beddington DeAngelis functional type incidence rate and investigate its impact on the disease dynamics.

It is well known that the treatment rate always plays a substantial role in preventing and controlling the spread of epidemics. In the classical epidemic model, the treatment rate was either constant [39] or proportional to the number of infected individuals [47]. This type of treatment rate is suitable in the case when the number of infectives is small and treatment resources are sufficient, and unsuitable when the number of the infectives is large, and treatment resources are limited. To control the disease, most researchers focus on a nonlinear type treatment rate. Dubey et al. [106, 123, 125] introduced the nonlinear treatment rate such as Holling Type II, Holling Type III, and Holling Type IV in their model and proposed nonlinear dynamics to control the epidemic. Holling type III treatment rate (also known as a saturated treatment rate) defines the condition in which removal rate initially becomes quick with increment in infectives, and then it develops gradually and settles down to maximum saturated value. After this, any increase in infectives won't influence the removal rate [106]. Motivated by the work of Dubey et al. [106], in the present chapter, we take the nonlinear saturated treatment rate of the form

$$g(I) = \frac{aI^2}{bI^2 + cI + 1} \quad (\text{where } I \geq 0,\ a, b > 0 \text{ and } c \geq 0)$$

The aim is to understand and predict the actual transmission of infectious diseases and investigate the effect of treatment rate to provide an effective control strategy. Therefore, motivated by Dubey et al. [106, 123, 125], we consider a SIR (susceptible-infected-

recovered) epidemic model setting with a nonlinear Beddington-DeAngelis functional type incidence rate with the inclusion of time lag (described as the latent period) and a nonlinear saturated treatment rate to provide a control strategy of the infectious diseases. It is shown that this simple-looking system can exhibit interesting dynamics if this behavioral response is delayed. For the dynamics of the model, we discuss the existence and stability behavior of the model for equilibriums. In addition, the existence of a Hopf bifurcation is also discussed.

2.2 Mathematical framework

This section presents the mathematical model described by the system of delay differential equations constructed from the interaction among the compartments: susceptible, infective, and recovered for the model equation.

2.2.1 The basic model equations

We consider that in the region under consideration, the total population is $N(t)$ at time t. The total population $N(t)$ is divided into three subclasses; the susceptible class $S(t)$, the infective $I(t)$, and the recovered individuals $R(t)$. We assume that the disease can spread due to the direct contact between susceptibles and infectives only. A block diagram given in Fig. 2.1 illustrates the conceptual description of the model.

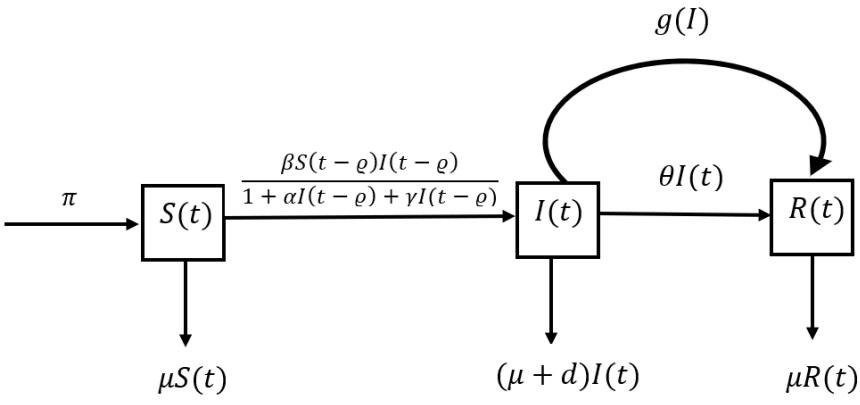


Figure 2.1: Transfer diagram for the delayed SIR model with the susceptible class $S(t)$, the infective class $I(t)$, and the recover class $R(t)$.

The model is presented by the following system of non-linear delay differential equa-

tions:

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

The initial conditions $\phi = (\phi_1, \phi_2, \phi_3)$ of (2.1) are defined in the Banach space

$$C_+ = \{\phi \in C([-\rho, 0], R^3_+) : \phi_1(\Omega) = S(\Omega), \phi_2(\Omega) = I(\Omega), \phi_3(\Omega) = R(\Omega)\},$$

where, $R^3_+ = \{(S,I,R) \in R^3 : S \geq 0, I \geq 0, R \geq 0\}$. Biologically, we assume that $\phi_i > 0 \quad (i = 1,2,3)$.

In model (2.1), we consider the total population $N(t)$ at time t, with the immigration of susceptible population with a constant rate π. The parameters μ, d, and θ represent the natural death rate, disease-induced death rate, and recovery rate, respectively. The term $f(S,I) = \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha S(t-\rho)+\gamma I(t-\rho)}$ represents the Beddington-DeAngelis type incidence rate with time delay ρ. Here, β is the effective contact rate (or transmission rate) of susceptibles with infectives, α is a measure of inhibition effect, such as preventive measures taken by susceptible individuals, γ is a measure of inhibition effects taken by infectives, and the time delay $\rho > 0$ represents the latent period of the disease. The incidence function $f(S,I)$ includes some special cases. For instance, if we set $\alpha = 0, \gamma = 0$ then the incidence rate is bilinear [51] and if $\alpha = 0$ the incidence rate, describing saturated effects of the prevalence of infectious diseases, is used in [75, 83]. The term $g(I) = \frac{aI^2}{bI^2+cI+1}$ in model (2.1) is the saturated treatment rate, where $a > 0$ represents the treatment rate of infected individuals (cure rate), $b > 0$ is the limitation rate in treatment availability and $c \geq 0$ is the saturation constant in absence of inhibitory effect.

2.2.2 Basic properties of the model

The equations of the model (2.1) monitor populations. From the Proposition 2.1 in Hattaf et al. [107] and Proposition 2.3. in Yang et al. [124], it can be shown that all state variables of the model (2.1) are nonnegative. That is, $(S,I,R) \in R^3_+$. Also, for ecological reasons, we suppossed that all parameters π, μ, β, α, γ, d, θ, a, b are positive and c is nonnegative.

Since $N(t) = S(t) + I(t) + R(t)$, the governing equations of model (2.1) can be rewritten

as

$$\frac{dN}{dt} = \pi - \mu N - dI \tag{2.2}$$

$$\leq \pi - \mu N \tag{2.3}$$

Lemma 2.2.1. *All solutions of the model (2.1) starting in R^3_+ are bounded and eventually enter a compact attracting set*

$$\Phi = \{(S,I,R) \in R^3_+ : S(t)+I(t)+R(t) = N(t) \leq \frac{\pi}{\mu}\}.$$

Proof. Continuity of the right-hand side of the model (2.1) and its derivative assure the well-posedness of the model for $N(t) > 0$. The invariant region for the existence of the solutions can be determined as given below:

$$0 < \liminf_{t\to\infty} N(t) \leq \limsup_{t\to\infty} N(t) \leq \frac{\pi}{\mu}.$$

Since $N(t) > 0$ on $[-\rho, 0]$ by assumption, $N(t) > 0$ for all $t \geq 0$. Therefore, with the help of Eq. (2.3), it can be seen that for any finite time t, $N(t)$ cannot blow up to infinity. The model system is dissipative (solutions are bounded), and consequently, the solution exists globally for all $t > 0$ in the invariant and compact set

$$\Phi = \{(S,I,R) \in R^3_+ : S(t)+I(t)+R(t) = N(t) \leq \frac{\pi}{\mu}\}.$$

As $N(t)$ approaches zero, $S(t)$, $I(t)$, and $R(t)$ also approach zero. Thus, each of these terms tend towards zero as $N(t)$ does. Thus, it is reasonable to interpret these terms as zero when $N(t) = 0$. ■

Remark 2.2.2. *In this region Φ, basic results such as usual local existence, uniqueness and continuation of solutions are valid for model (2.1). Hence, there exists a unique solution $(S(t),I(t),R(t))$ of model (2.1) starting in the interior of Φ that exists on a maximal interval $[0,\infty)$ if solutions remain bounded [22].*

2.3 Equilibrium and stability analysis

From the model (2.1), we infer that, since $R(t)$ does not appear in equations for $\frac{dS}{dt}$ and $\frac{dI}{dt}$, it is sufficient to analyze the behavior of solutions of (2.1) by the following system of

DDEs:

$$\begin{aligned}\frac{dS}{dt} &= \pi - \mu S - \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha S(t-\rho)+\gamma I(t-\rho)} \\ \frac{dI}{dt} &= \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha S(t-\rho)+\gamma I(t-\rho)} - (\mu+d+\theta)I - \frac{aI^2}{bI^2+cI+1}\end{aligned} \tag{2.4}$$

with initial conditions $\phi = (\phi_1, \phi_2)$ of (2.4) are defined in the Banach space

$$C_+ = \{\phi \in C([-\rho, 0], R_+^2) : \phi_1(\Omega) = S(\Omega), \phi_2(\Omega) = I(\Omega)\},$$

where, $R_+^2 = \{(S,I) \in R^2 : S \geq 0, I \geq 0\}$, $\phi_i > 0 \quad (i = 1,2)$.

Now, we obtain the equilibria of the system (2.4). There are only two types of physically, and in addition biologically, relevant equilibria, namely,

(i) $E_0(S_0, 0) = E_0\left(\frac{\pi}{\mu}, 0\right)$, disease-free equilibrium (DFE).

(ii) $E_e(S^*, I^*)$, positive or endemic equilibrium (EE), where S^* and I^* are given in Section 2.3.2.

2.3.1 Disease free equilibrium and its stability

System (2.4) has a disease free equilibria of the form $E_0\left(\frac{\pi}{\mu}, 0\right)$ (that is, there is no infection present in the community and all individuals are susceptible) which is obtained by setting right hand sides of the system (2.4) to zero. The characteristic equation of the linearization of model (2.4) near the disease-free equilibrium $E_0\left(\frac{\pi}{\mu}, 0\right)$ is given by

$$(\mu+\lambda)\left(\frac{\beta\pi e^{-\lambda\rho}}{\mu+\alpha\pi} - (\mu+d+\theta) - \lambda\right) = 0. \tag{2.5}$$

One of the roots of the Eq. (2.5) is given by $\lambda_1 = -\mu$ and other roots can be obtained from

$$\frac{\beta\pi e^{-\lambda\rho}}{\mu+\alpha\pi} - (\mu+d+\theta) - \lambda = 0.$$

The term $\frac{\beta\pi}{(\mu+\alpha\pi)(\mu+d+\theta)}e^{-\lambda\rho}$ at $\rho = 0$, is termed as the basic reproduction number R_0. Thus, the basic reproduction number for the system (2.4) is given by

$$R_0 = \frac{\beta\pi}{(\mu+\alpha\pi)(\mu+d+\theta)}.$$

Analysis for $\mathbf{R_0 \neq 1}$

Clearly, Eq. (2.5) has one negative root $\lambda_1 = -\mu$ and other roots can be obtained from the equation

$$\lambda + \mu + d + \theta - \frac{\beta\pi}{(\mu + \alpha\pi)} e^{-\lambda\rho} = 0.$$

Let

$$f(\lambda) = \lambda + \mu + d + \theta - \frac{\beta\pi}{(\mu + \alpha\pi)} e^{-\lambda\rho} = \lambda + (\mu + d + \theta)\left(1 - R_0 e^{-\lambda\rho}\right).$$

If $R_0 > 1$, it is readily seen that, for real λ

$$f(0) = \lambda + (\mu + d + \theta)(1 - R_0) < 0, \quad \lim_{\lambda \to \infty} f(\lambda) = +\infty$$

Hence, $f(\lambda) = 0$ and $f'(\lambda) > 0$. Therefore $f(\lambda) = 0$ has a unique positive real root if $R_0 > 1$.

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

We notice that

$$\mathrm{Re}\,\lambda = \frac{\beta\pi e^{-(\mathrm{Re}\,\lambda)\rho}\cos(Im\lambda)\rho}{(\mu + \alpha\pi)} - (\mu + d + \theta) < \frac{\beta\pi}{(\mu + \alpha\pi)} - (\mu + d + \theta) < 0,$$

which contradicts our assumption. Therefore if $R_0 < 1$ then λ will be a root of Eq. (2.5) with negative real part. The result can be written in form of the theorem, stated below.

Theorem 2.3.1. *The disease free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Analysis at $\mathbf{R_0 = 1}$ and $\rho = 0$

In this section, we analyze the behavior of system (2.4) when the basic reproduction number R_0 is equal to one and $\rho = 0$. We observe that the Jacobian matrix of system (2.4) evaluated at $R_0 = 1$ and bifurcation parameter $\beta = \beta^* = \frac{(\mu+\alpha\pi)(\mu+d+\theta)}{\pi}$ has a simple zero eigenvalue and another eigenvalue with negative real part. Since linearization is not suitable to analyze the stability behaviour of equilibrium points at $R_0 = 1$, therefore center manifold theory [28] is used. For simplicity, let $S = x_1$ and $I = x_2$, then the system (2.4) can be written as

$$\begin{aligned} \frac{dx_1}{dt} &= \pi - \mu x_1 - \frac{\beta x_1 x_2}{1 + \alpha x_1 + \gamma x_2} \equiv f_1 \\ \frac{dx_2}{dt} &= \frac{\beta x_1 x_2}{1 + \alpha x_1 + \gamma x_2} - (\mu + d + \theta) x_2 - \frac{a x_2^2}{{x_2}^2 + b x_2 + c} \equiv f_2 \end{aligned} \tag{2.6}$$

Let J denotes the Jacobian matrix evaluated at $R_0 = 1$, and $\beta = \beta^*$ then

$$J = \begin{bmatrix} -\mu & -\frac{\beta^*\pi}{(\mu+\alpha\pi)} \\ 0 & 0 \end{bmatrix}.$$

Let $w = [w_1,\ w_2]$ and $u = [u_1,\ u_2]^t$ be the left and right nullvectors of J corresponding to the zero eigenvalue. Then, we have

$$w_1 = 0, \quad w_2 = 1 \quad \text{and} \quad u_1 = -\frac{\beta^*\pi}{\mu(\mu+\alpha\pi)}, \quad u_2 = 1.$$

The nonzero partial derivatives associated with the functions f_1 and f_2 of the system (2.6) evaluated at $R_0 = 1$ and $\beta = \beta^*$ are

$\left(\frac{\partial^2 f_2}{\partial x_2^2}\right)_{E_0} = -2a - \frac{2\pi\mu\beta\gamma}{(\mu+\alpha\pi)^2}$, $\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_{E_0} = \left(\frac{\partial^2 f_2}{\partial x_2 \partial x_1}\right)_{E_0} = -\frac{\pi\alpha\beta\mu}{(\mu+\alpha\pi)^2} + \frac{\beta\mu}{\mu+\alpha\pi}$ and $\left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_{E_0} = \frac{\pi}{\mu+\alpha\pi}$.

From Theorem 4.1 of Castillo-Chavez and Song [40], the bifurcation coefficients a_1 and b_1 are calculated as

$$\begin{aligned} a_1 &= \sum_{k,i,j=1}^{2} w_k u_i u_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_{E_0} \\ &= -\frac{2\beta^*\pi}{(\mu+\alpha\pi)}\left[-\frac{\pi\alpha\beta^*}{(\mu+\alpha\pi)^2} + \frac{\beta^*}{\mu+\alpha\pi}\right] - 2a - \frac{2\mu\pi\beta^*\gamma}{(\mu+\alpha\mu)^2} \\ &= -\frac{2\beta^*\pi\mu}{\mu+\alpha\pi}\left(\frac{\beta^* + \pi(\mu+\alpha\pi)}{(\mu+\alpha\pi)^2}\right) - 2a < 0, \end{aligned}$$

and

$$b_1 = \sum_{k,i=1}^{2} w_k u_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_{E_0} = w_2 u_2 \left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_{E_0} = \frac{\pi}{\mu+\alpha\pi} > 0.$$

Sign of a_1 determines the nature of the bifurcation at $R_0 = 1$. The local analysis of the center manifold yields a parameter, a_1, whose sign indicates the existence and stability of a branch of endemic equilibria near the threshold $R_0 = 1$. If a_1 is negative, then a branch of super-threshold endemic equilibria exists, and the bifurcation is supercritical. This case is frequently alluded to as a forward bifurcation. Thus, with the help of Theorem 4.1(iv) of Castillo-Chavez and Song (2004), the following theorem is being concluded:

Theorem 2.3.2. *The disease-free equilibrium (DFE) is locally asymptotically stable if R_0 is slightly less than one and if R_0 is slightly greater than one, then the DFE is unstable, and there*

is a locally asymptotically stable positive equilibrium near the DFE. Hence the model system (2.4) exhibits forward bifurcation at $R_0 = 1$ *for* $\rho = 0$.

The bifurcation from the disease-free equilibrium at $R_0 = 1$ is forward, which can be observed from Fig. 2.2 for the parameter values listed in Table 2.1.

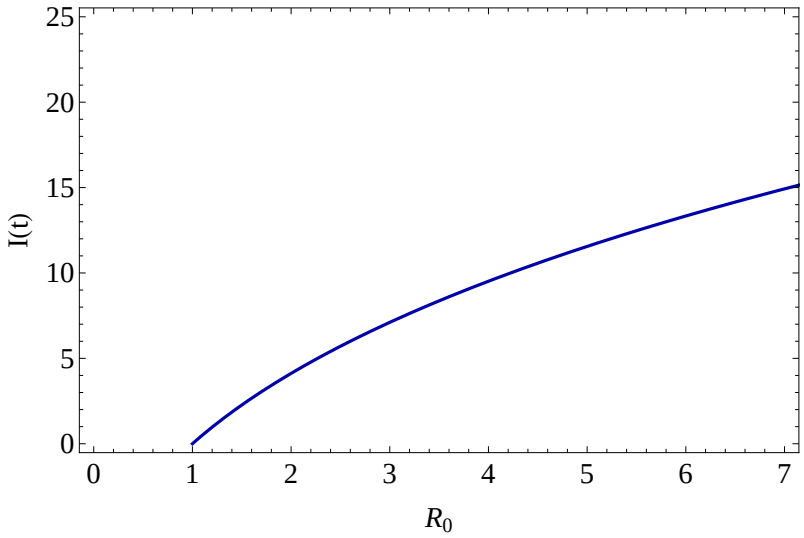


Figure 2.2: Plot of $I(t)$ versus R_0.

2.3.2 Endemic equilibrium and its stability

In this section, we investigate the stability of the system (2.4) at the endemic equilibrium $E_e(S^*, I^*)$.

Equating the second equation of the system (2.4) to zero, we get

$$\frac{\beta S^* I^*}{1 + \alpha S^* + \gamma I^*} - (\mu + d + \theta) I^* - \frac{a I^{*2}}{b I^{*2} + c I^* + 1} = 0. \tag{2.7}$$

After solving Eq. (2.7), we get S^* in terms of I^* as follows:

$$S^* = \frac{(1 + \gamma I^*)(p(b I^{*2} + c I^* + 1) + a I^*)}{(\beta - p\alpha)(b I^{*2} + 1) + I^* (c(\beta - p\alpha) - a\alpha)}, \tag{2.8}$$

where,

$$p = \mu + d + \theta.$$

$S^* > 0$ if

$$c(\beta - p\alpha) - \alpha a > 0. \tag{2.9}$$

This condition also implies that

$$\beta - p\alpha > 0. \tag{2.10}$$

Now, on adding the first and second equations of the system (2.4) and equate it to zero, we get

$$\pi - \mu S^* - (\mu + d + \theta) I^* - \frac{aI^{*2}}{bI^{*2} + cI* + 1} = 0. \tag{2.11}$$

Substituting the value of S^* from the Eq. (2.8) into the Eq. (2.11), we get the following equation in I^*:

$$A_0 + A_1 I^* + A_2 I^{*2} + A_3 I^{*3} + A_4 I^{*4} + A_5 I^{*5} = 0, \tag{2.12}$$

where,

$$\begin{aligned}
A_0 &= p(\mu + \pi\alpha)(1 - R_0), \\
A_1 &= a(\mu + \pi\alpha) + (\beta - p\alpha)(p - 2c\pi) + p\mu(2c + \gamma), \\
A_2 &= ac(\mu + \pi\alpha) + 2pc(\beta - p\alpha + \gamma\mu) + p\mu\left(2b + c^2\right) + a(\beta + \gamma\mu) \\
&\quad - \left(2\alpha pa + \pi\left(2b + c^2\right)(\beta - p\alpha)\right), \\
A_3 &= ab(\mu + \pi\alpha) + pc^2\gamma\mu + 2bp\mu(c + \gamma) + (\beta - p\alpha)(c(a + pc) + 2b(p - c\pi)) \\
&\quad - a^2\alpha - ac(p\alpha - \gamma\mu), \\
A_4 &= ab(\beta - 2p\alpha + \gamma\mu) + b(\beta - p\alpha)(2pc - b\pi) + bp\mu(b + 2c\gamma), \\
A_5 &= pb^2(\beta - \alpha p + \gamma\mu).
\end{aligned}$$

With the help of Descartes' rule of signs [42], the Eq. (2.12) has a unique positive real root I^* if any one of the following holds:

$$\begin{aligned}
&(i)\ A_5 > 0,\ A_4 < 0,\ A_3 < 0,\ A_2 < 0,\ A_1 < 0 \text{ and } A_0 < 0. \\
&(ii)\ A_5 > 0,\ A_4 > 0,\ A_3 < 0,\ A_2 < 0,\ A_1 < 0 \text{ and } A_0 < 0. \\
&(iii)\ A_5 > 0,\ A_4 > 0,\ A_3 > 0,\ A_2 < 0,\ A_1 < 0 \text{ and } A_0 < 0. \\
&(iv)\ A_5 > 0,\ A_4 > 0,\ A_3 > 0,\ A_2 > 0,\ A_1 < 0 \text{ and } A_0 < 0. \\
&(v)\ A_5 > 0,\ A_4 > 0,\ A_3 > 0,\ A_2 > 0,\ A_1 > 0 \text{ and } A_0 < 0.
\end{aligned} \tag{2.13}$$

After determining the value of I^*, we can determine the value of S^* from Eq. (2.8). Thus, there exists a unique positive endemic equilibrium $E_e(S^*, I^*)$ if one of the conditions (2.13) holds.

If conditions (2.13) are not satisfied, then we obtain the following result:

Proposition 2.3.3. *If $R_0 > 1$, then there is either a unique or three or five positive endemic equilibria if all equilibria are simple roots.*

Proof. Suppose $R_0 > 1$. From Eq. (2.12), we have fifth degree polynomial in I^*:

$$F(I^*) = A_0 + A_1 I^* + A_2 I^{*2} + A_3 I^{*3} + A_4 I^{*4} + A_5 I^{*5}.$$

Leading coefficient of I^* is $A_5 = pb^2(\beta - \alpha p + \gamma\mu) > 0$. Hence,

$$\lim_{I^* \to \infty} F(I^*) = +\infty.$$

Also, note that $F(0) = A_0$, and $A_0 < 0$ if $R_0 > 1$. $F(I^*)$ is a continuous function on I^*, and by the fundamental theorem of algebra, we know that this polynomial can have at most five real roots. ■

We now analyze the local stability of endemic equilibrium E_e as follows.

The characteristic equation of the system (2.4) evaluated at E_e is a second degree transcendental equation:

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\rho} = 0, \tag{2.14}$$

where,

$$\begin{aligned}
p_0 &= 2\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}, \\
q_0 &= \mu\left(\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}\right), \\
p_1 &= \frac{\beta}{(1 + \alpha S^* + \gamma I^*)^2}\left(I^*(1 + \gamma I^*) - S^*(1 + \alpha S^*)\right), \\
q_1 &= -\frac{\mu\beta S^*(1 + \alpha S^*)}{(1 + \alpha S^* + \gamma I^*)^2} + \frac{\beta I^*(1 + \gamma I^*)}{(1 + \alpha S^* + \gamma I^*)^2}\left(\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}\right).
\end{aligned}$$

Theorem 2.3.4. *At $\rho = 0$, E_e is locally asymptotically stable if $\frac{S^*}{I^*} \leq \frac{1+\gamma I^*}{1+\alpha S^*}$ is satisfied.*

Proof. The characteristic equation for the endemic equilibrium E_e at $\rho = 0$ is given by

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1) = 0.$$

It is easy to verify that if $\frac{ac - aI^{*2}}{\left(I^{*2} + bI^* + c\right)^2} \geq \frac{\beta S^*(1+\alpha S^*)}{(1+\alpha S^* + \gamma I^*)^2}$ is satisfied, then

$$p_0 + p_1 = 2\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{(bI^{*2} + cI^* + 1)^2} + \frac{\beta}{(1 + \alpha S^* + \gamma I^*)^2}\left(I^*(1 + \gamma I^*) - S^*(1 + \alpha S^*)\right) > 0,$$

$$q_0+q_1=\mu\left(\mu+d+\theta\right)+\frac{aI^*\left(2+cI^*\right)}{\left(bI^{*2}+cI^*+1\right)^2}\left(\mu+\frac{\beta I^*\left(1+\gamma I^*\right)}{\left(1+\alpha S^*+\gamma I^*\right)^2}\right)+\frac{\beta}{\left(1+\alpha S^*+\gamma I^*\right)^2}$$
$$\left(\left(d+\theta\right)I^*\left(1+\gamma I^*\right)+\mu\left(I^*\left(1+\gamma I^*\right)-S^*\left(1+\alpha S^*\right)\right)\right)>0.$$

Hence, by the Routh-Hurwitz criterion, it is concluded that the endemic equilibrium E_e of the system (2.4) is locally asymptotically stable when $\rho=0$. ■

Theorem 2.3.5. *For $\rho>0$, E_e is locally asymptotically stable if the conditions*

$$\mu\left(1+\alpha S^*+\gamma I^*\right)^2\geq\beta I^*\left(1+\gamma I^*\right)\quad \textit{and}\quad 2(\mu+d+\theta)I^*(1+\gamma I^*)\geq\mu S^*\left(1+\alpha S^*\right)$$

are satisfied simultaneously.

Proof. For $\rho>0$, the characteristic equation evaluated at E_e is given by the Eq. (2.14) which is

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

For $\rho>0$, by applying Theorem 2.4 in Ruan and Wei [37], it follows that the characteristic root of (2.14) must pass through the imaginary axis for the occurrence of instability for a fixed value of time delay ρ. Assume that $\lambda=i\eta,\eta>0$ is the root of the characteristic Eq. (2.14). Substituting $\lambda=i\eta$ in Eq. (2.14), we obtain

$$(-\eta^2+q_0+p_1\eta\sin\eta\rho+q_1\cos\eta\rho)+i(p_0\eta+p_1\eta\cos\eta\rho-q_1\sin\eta\rho)=0. \tag{2.15}$$

After separation of real and imaginary parts of Eq. (2.7), we get

$$p_1\eta\sin\eta\rho+q_1\cos\eta\rho=\eta^2-q_0, \tag{2.16}$$
$$p_1\eta\cos\eta\rho-q_1\sin\eta\rho=-\eta p_0. \tag{2.17}$$

On eliminating ρ by squaring and adding Eqs. (2.16) and (2.17), we obtain a biquadratic polynomial in η as

$$\eta^4+(p_0^2-2q_0-p_1^2)\eta^2+(q_0^2-q_1^2)=0. \tag{2.18}$$

Letting $\eta^2=x$. Then, Eq. (2.18) becomes

$$x^2+Ax+B=0, \tag{2.19}$$

where,

$$A = p_0^2 - 2q_0 - p_1^2, \quad B = q_0^2 - q_1^2.$$

$$\begin{aligned} A &= \left(2\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}\right)^2 - 2\mu\left(\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}\right) - \\ &\quad \frac{\beta^2}{(1 + \alpha S^* + \gamma I^*)^4}\left(I^*(1 + \gamma I^*) - S^*(1 + \alpha S^*)\right)^2 \\ &= (\mu + d + \theta)^2 + 2(\mu + d + \theta)\frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1} + \left(\frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}\right)^2 + \mu^2 - \\ &\quad \left(\frac{\beta}{(1 + \alpha S^* + \gamma I^*)^2}\left(I^*(1 + \gamma I^*) - S^*(1 + \alpha S^*)\right)\right)^2, \end{aligned}$$

$$\begin{aligned} B &= q_0^2 - q_1^2 \\ &= \left(\mu\left(\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}\right)\right)^2 - \\ &\quad \left(-\frac{\mu\beta S^*(1 + \alpha S^*)}{(1 + \alpha S^* + \gamma I^*)^2} + \frac{\beta I^*(1 + \gamma I^*)}{(1 + \alpha S^* + \gamma I^*)^2}\left(\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}\right)\right)^2 \end{aligned}$$

$$\begin{aligned} &= \left(\mu + d + \theta + \frac{acI^{*2} + 2aI^*}{bI^{*2} + cI^* + 1}\right)^2 \left(\mu^2 - \frac{\beta^2 I^{*2}(1 + \gamma I^*)^2}{(1 + \alpha S^* + \gamma I^*)^4}\right) + \\ &\quad \frac{2\mu\beta^2 S^*(1 + \alpha S^*) I^*(1 + \gamma I^*)(acI^{*2} + 2aI^*)}{(1 + \alpha S^* + \gamma I^*)^4 (bI^{*2} + cI^* + 1)} + \\ &\quad \frac{\mu\beta^2 S^*(1 + \alpha S^*)}{(1 + \alpha S^* + \gamma I^*)^4}\left(2(\mu + d + \theta) I^*(1 + \gamma I^*) - \mu S^*(1 + \alpha S^*)\right). \end{aligned}$$

$A > 0$ if and only if $\mu\left(1 + \alpha S^* + \gamma I^*\right)^2 \geq \beta\left(I^*\left(1 + \gamma I^*\right) - S^*\left(1 + \alpha S^*\right)\right)$, and $B > 0$ if and only if both the conditions $\mu\left(1 + \alpha S^* + \gamma I^*\right)^2 \geq \beta I^*\left(1 + \gamma I^*\right)$ and $2(\mu + d + \theta) I^*(1 + \gamma I^*) \geq \mu S^*\left(1 + \alpha S^*\right)$ are satisfied simultaneously.
Note that $\mu\left(1 + \alpha S^* + \gamma I^*\right)^2 \geq \beta I^*\left(1 + \gamma I^*\right)$ implies that

$$\mu\left(1 + \alpha S^* + \gamma I^*\right)^2 \geq \beta\left(I^*\left(1 + \gamma I^*\right) - S^*\left(1 + \alpha S^*\right)\right).$$

According to the Routh-Hurwitz criterion, a contradiction arises with the assumption of instability, i.e., $\lambda = i\eta$. Thus, it can be concluded that the endemic equilibrium E_e of the system (2.4) is locally asymptotically stable for $\rho > 0$. ■

Hopf bifurcation analysis

If $B = q_0^2 - q_1^2$ given in Eq. (2.19) is negative, then there is a unique positive η_0 satisfying Eq. (2.19), i.e., there is single pair of purely imaginary roots $\pm i\eta_0$ to Eq. (2.14).

From Eqs. (2.16) and (2.17), ρ_n corresponding to η_0 can be obtained as

$$\rho_n = \frac{1}{\eta_0}\arccos\left(\frac{\eta_0^2(q_1 - p_0p_1) - q_0q_1}{p_1^2\eta_0^2 + q_1^2}\right) + \frac{2n\pi}{\eta_0}, \quad n = 0, 1, 2\ldots \tag{2.20}$$

Endemic equilibrium E_e is stable for $\rho < \rho_0$ if transversality condition holds, i.e., $\frac{d}{dt}(\mathrm{Re}\,\lambda)\Big|_{\lambda=i\eta_0} \neq 0$.

On differentiating Eq. (2.14) with respect to ρ, we get

$$\left(2\lambda + p_0 + p_1e^{-\lambda\rho} - (p_1\lambda + q_1)\rho e^{-\lambda\rho}\right)\frac{\mathrm{d}\lambda}{\mathrm{d}\rho} = \lambda(p_1\lambda + q_1)e^{-\lambda\rho}. \tag{2.21}$$

$$\begin{aligned}\left[\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right]^{-1} &= \frac{2\lambda + p_0 + p_1e^{-\lambda\rho} - (p_1\lambda + q_1)\rho e^{-\lambda\rho}}{\lambda(p_1\lambda + q_1)e^{-\lambda\rho}} \\ &= \frac{2\lambda + p_0}{\lambda(p_1\lambda + q_1)e^{-\lambda\rho}} + \frac{p_1}{\lambda(p_1\lambda + q_1)} - \frac{\rho}{\lambda}\end{aligned}$$

$$= \frac{2\lambda + p_0}{-\lambda(\lambda^2 + p_0\lambda + q_0)} + \frac{p_1}{\lambda(p_1\lambda + q_1)} - \frac{\rho}{\lambda}.$$

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}\rho}(\mathrm{Re}\,\lambda)\Big|_{\lambda=i\eta_0} &= \mathrm{Re}\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right)^{-1}\Big|_{\lambda=i\eta_0} \\ &= \mathrm{Re}\left(\frac{2i\eta_0 + p_0}{-i\eta_0(-\eta_0^2 + ip_0\eta_0 + q_0)} + \frac{p_1}{-p_1\eta_0{}^2 + iq_1\eta_0} + \frac{i\rho}{\eta_0}\right) \\ &= \frac{1}{\eta_0}\left(\frac{2\eta_0(\eta_0^2 - q_0) + p_0^2\eta_0}{(p_0\eta_0)^2 + (\eta_0^2 - q_0)^2} - \frac{p_1^2\eta_0}{(p_1\eta_0)^2 + q_1^2}\right) \\ &= \frac{2(\eta_0^2 - q_0) + p_0^2}{(p_0\eta_0)^2 + (\eta_0^2 - q_0)^2} - \frac{p_1^2}{(p_1\eta_0)^2 + q_1^2}.\end{aligned} \tag{2.22}$$

Now, on squaring and adding Eqs. (2.16) and (2.17), we get

$$(p_1\eta_0)^2 + q_1^2 = (p_0\eta_0)^2 + (\eta_0^2 - q_0)^2$$

So that Eq. (2.22) can be written as

$$\frac{\mathrm{d}}{\mathrm{d}\rho}(\mathrm{Re}\,\lambda)\Big|_{\lambda=i\eta_0} = \frac{2\eta_0^2 + (p_0^2 - 2q_0 - p_1^2)}{p_1^2\eta_0^2 + q_1^2}. \tag{2.23}$$

Under the condition $A = p_0^2 - 2q_0 - p_1^2 > 0$, it can be seen that

$$\frac{\mathrm{d}}{\mathrm{d}\rho}(\mathrm{Re}\,\lambda)\Big|_{\lambda=i\eta_0} > 0.$$

Therefore, the transversality condition holds and Hopf bifurcation occurs at $\eta = \eta_0$, $\rho = \rho_0$.

Summarizing the above analysis, we arrive at the following theorem.

Theorem 2.3.6. *If condition $q_0^2 - q_1^2 < 0$ holds, the endemic equilibrium E_e of the system (2.4) is asymptotically stable for $\rho \in [0, \rho_0)$ and it undergoes Hopf bifurcation at $\rho = \rho_0$.*

2.4 Numerical simulation

Since it is essential to analyze the dynamical behavior of the model, therefore, in this section, the system (2.4) is integrated numerically using the set of tested parameters given in Table 2.1.

Table 2.1: List of parameters

Parameter	Interpretation	Value	Reference
π	Recruitment rate of susceptible	2	[125]
α	Measure of inhibition taken by susceptibles	0.004	[125]
β	Transmission rate	0.00924	Assumed
μ	Natural death rate	0.05	[125]
d	Disease-induced death rate	0.001	[125]
γ	Measure of inhibition taken by infectives	0.002	[125]
θ	Recovery rate	0.002	[125]
a	Treatment rate of infected individuals/Cure rate	0.002	Assumed
b	Limitation rate in treatment availability	0.0005	Assumed
c	Saturation constant	0.0002	Assumed

The computer simulations are performed for S and I for various values of ρ. The trajectory of S and I with initial conditions $S(0) = 33$, $I(0) = 5$, approach to the endemic equilibrium as shown in Fig. 2.3.

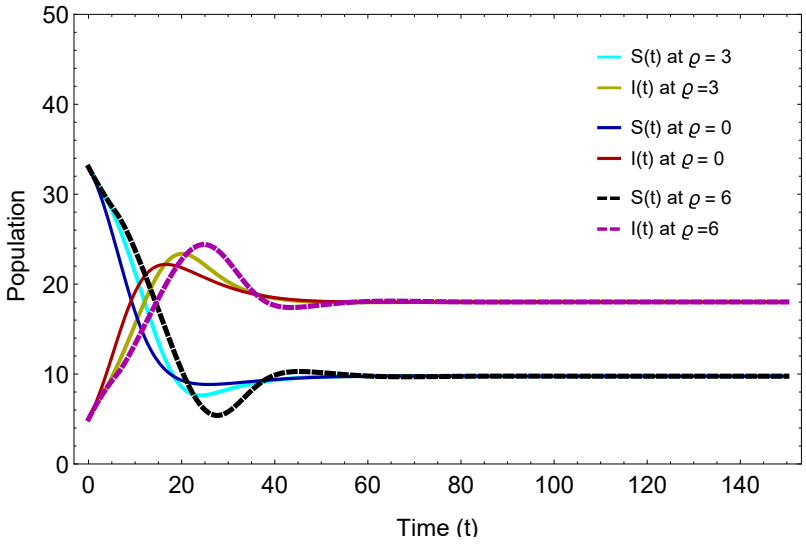


Figure 2.3: Susceptible and infected population for various values of ρ.

Fig. 2.3 shows the effect of time delay on susceptible population and infective population, respectively, for different values of time delay ρ. It is shown that as the time delay ρ increases, the number of susceptible starts decreasing, and the number of infectives starts growing. Due to the interplay between the number of infectives and susceptibles, infectives settle to their steady-state but never reach zero, which shows that the system approaches endemic equilibrium $E_e(9.8292, 18.4177)$.

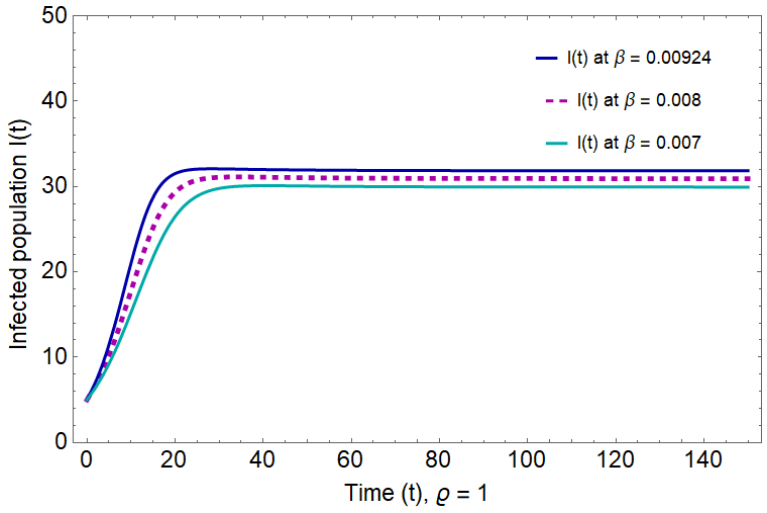


Figure 2.4: Behavior of infected population for various values of β at $\rho = 1$.

Fig. 2.4 shows the influence of transmission rate β on infected population for $\rho = 1$. The higher the effective contact rate, the higher will indeed be the possibility of spreading

the disease. In Fig. 2.4, we note that when the effective contact rate β is high, more people will be infected, and when the effective contact rate β is low, then fewer people are infected.

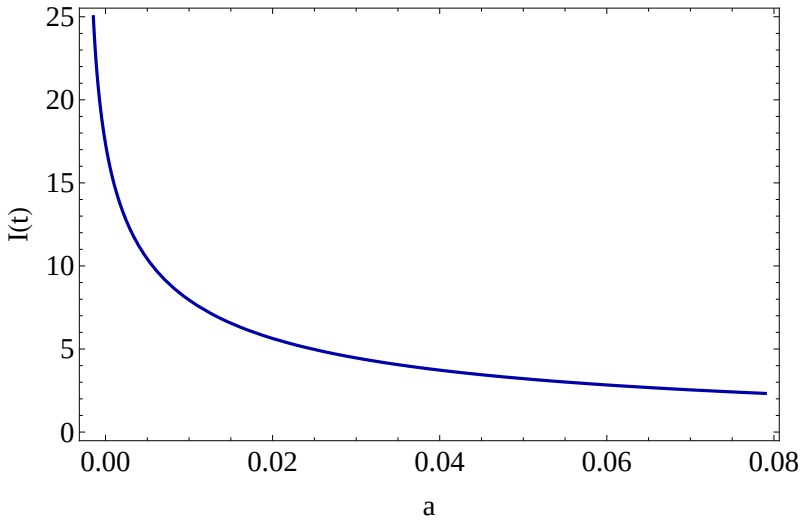
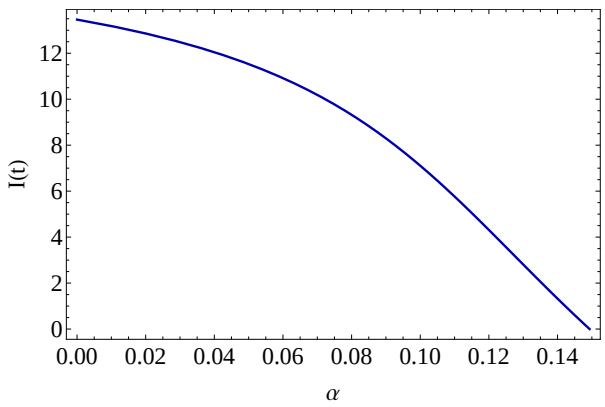
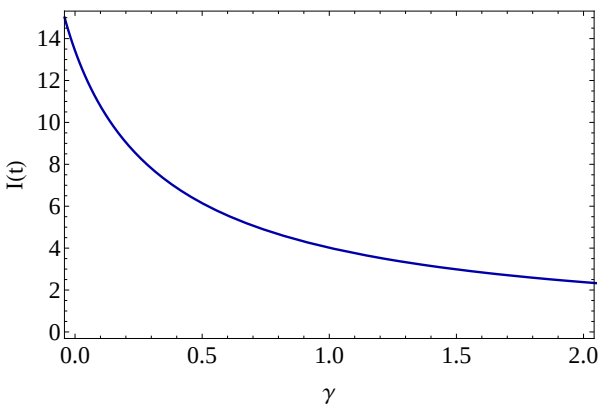


Figure 2.5: Infectives $I(t)$ versus treatment rate a.

Fig. 2.5 depicts the behavior of infected population $I(t)$ with respect to cure (treatment) rate a. From this figure, it can be seen that the infected population is decreasing with the increment in treatment rate a.



2.6.1: Infectives $I(t)$ versus inhibition effect α.



2.6.2: Infectives $I(t)$ versus inhibition effect γ.

Figure 2.6: Effects of measure of inhibitions.

Fig. 2.6 shows the effect of measure of inhibitions taken by susceptible and infectives, respectively. These figures show that when the inhibition is less, more people are getting infected, and when inhibition is more, fewer people are getting infected.

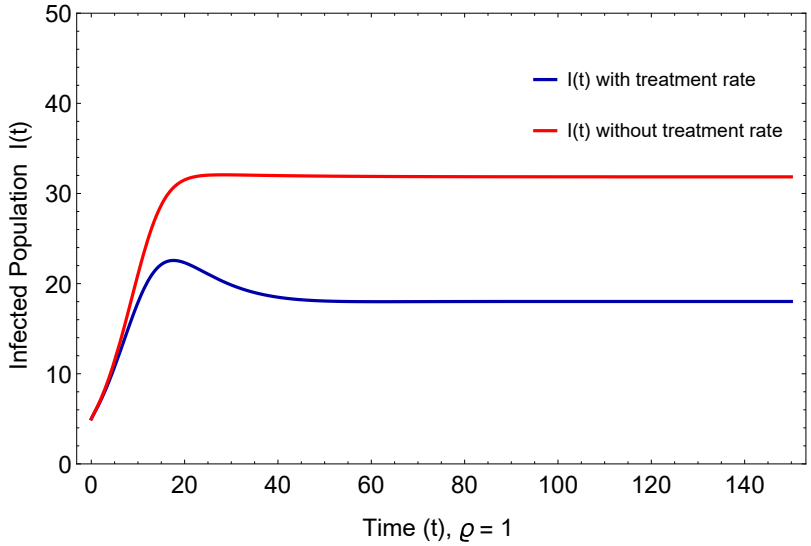
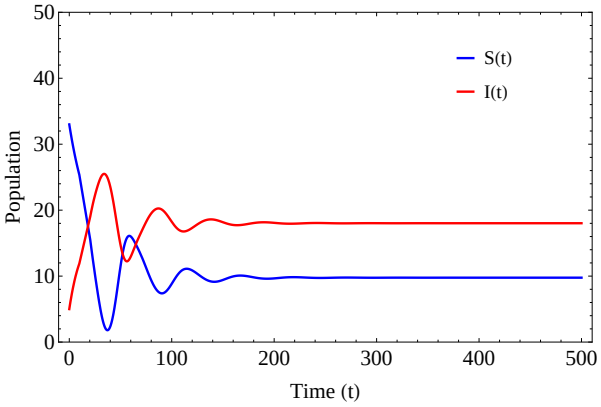
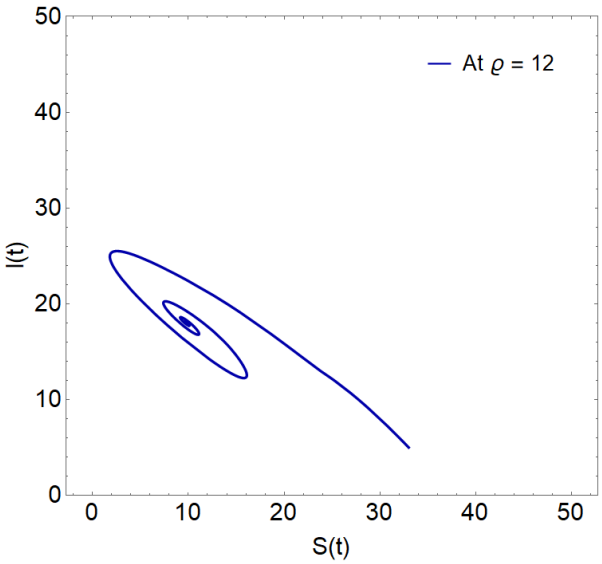


Figure 2.7: Infected population with and without saturated treatment rate at $\rho = 1$.

Fig. 2.7 shows the effect of saturated treatment rate on the infected individuals for the time lag $\rho = 1$. The treatment is an imperative strategy to diminish the spread of diseases. This figure shows that the saturated treatment rate is reducing the infection.



2.8.1: Plot of $S(t)$ and $I(t)$ with respect to time t.



2.8.2: $I(t)$ versus $S(t)$.

Figure 2.8: Behavior of susceptible and infected population for the time lag $\rho = 12$.

In Fig. 2.8.1, plot has been drawn for infected and susceptible population versus time t for time lag $\rho = 12$. Fig. 2.8.2 is the phase plot between susceptible $S(t)$ and infected $I(t)$ population which shows the limit cycle for time lag $\rho = 12$. It clear from Figs. 2.8.1 and 2.8.2 that $E_e = (9.8292, 18.4177)$ is the unique endemic equilibrium. According to the algorithm given in Eq. (2.20) and Theorem 2.3.6, we compute that $\rho_0 = 12.5801$, $p_0^2 - 2q_0 - p_1^2 = 0.0109139 > 0$ and $q_0^2 - q_1^2 = -0.000150942 < 0$. Clearly, from Fig. 2.8, it can

be seen that when $\rho = 12 < \rho_0 = 12.5801$ then the endemic equilibrium is asymptotically stable.

2.5 Discussion

This chapter formulates and analyzes a nonlinear time-delayed SIR mathematical model with Beddington- DeAngelis type incidence rate and a saturated type treatment rate. We assume that the time lag is present due to the latency period of pathogens. The mathematical analysis shows that the model exhibits two equilibria: the disease-free and endemic equilibrium. The local stability of the disease-free equilibrium is determined by the basic reproduction number R_0. The disease-free equilibrium has been shown to be stable for $R_0 < 1$, i.e., disease dies out for $R_0 < 1$ and for $R_0 > 1$, it becomes unstable and the endemic equilibrium exists. We also discuss the stability of disease-free equilibrium at $R_0 = 1$ using the center manifold theory. We observe that at $R_0 = 1$, the model exhibits forward bifurcation and changes its stability as R_0 crosses one. The stability analysis demonstrates that endemic equilibrium is locally asymptotically stable under certain conditions for the time lag $\rho \geq 0$ as stated in Theorem 2.3.4 and Theorem 2.3.5. Further, system (2.4) has been simulated numerically for the effect of time delay ρ and it is observed that as delay increases, the infected population also increases. Furthermore, we simulate the model numerically to see the effects of transmission rate, inhibitions, and treatment rate. The graphs show that the infection can be eradicated from society if the treatment given to the population is managed according to the saturated treatment rate. Also, analytical and numerical results show that oscillatory behavior of the infected population would also occur, indicating the existence of a Hopf bifurcation.

Chapter 3

Stability behavior of a time delayed nonlinear SIR epidemic transmission model with Beddington-DeAngelis incidence and Holling type II treatment rates

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In this chapter, we explore a time-delayed SIR mathematical model along with nonlinear incidence rate and Holling functional type II treatment rate for disease transmission. The mathematical study of the model demonstrates that the model exhibits two equilibria, to be specific, disease-free equilibrium (DFE) and endemic equilibrium (EE). We obtain the basic reproduction number R_0 and investigate that the model is locally asymptotically stable at DFE if $R_0 < 1$ and unstable if $R_0 > 1$ for the time lag $\rho > 0$. The stability of DFE at $R_0 = 1$ is also investigated for the time lag $\rho \geq 0$, and we show that for $\rho > 0$, the DFE is linearly neutrally stable whereas, for $\rho = 0$, the model exhibits backward bifurcation whereby the DFE will coexist with two endemic equilibria, when $R_0 < 1$. Also, we investigate the stability of the model at the endemic equilibrium and find that oscillatory solution may appear via Hopf bifurcation, taking the delay as a bifurcation parameter. Further, the global stability of the model equilibria has also been investigated. Finally, numerical simulations have been presented to illustrate the analytical studies.

3.1 Introduction

A major goal of the epidemiological study is to develop an understanding of the transmission of epidemics and interventions that can be taken to prevent and control the spread of infectious diseases. For the most part, an ideal condition for the elimination of infection is for the basic reproduction number (BRN) [33] to be less than one whereby the disease-free equilibrium (DFE) is stable; the unique endemic equilibrium (EE) will be asymptotically stable as far as when the BRN is greater than one, implies that the disease-free equilibrium will be unstable. However, many authors have exhibited that it is feasible for the disease-free equilibrium to exists together with two endemic equilibria even when the BRN is less than one, leading to the phenomenon of backward bifurcation [91]. More accurately, the phenomenon where the disease-free equilibrium loses its stability and a stable endemic equilibrium appears as R_0 increases through unity is the case of forward bifurcation, whereas the phenomenon where a stable endemic equilibrium coexists with a stable DFE when $R_0 < 1$ is the case of backward bifurcation. It demonstrates that for disease control and destruction, relying on the BRN to be less than one is only necessary but no longer sufficient. Therefore, the occurrence of backward bifurcation has important public health implications. Many authors have studied epidemic models characterized by backward bifurcation for both generic and specific diseases [30, 38, 40, 47].

Inhibitions are effective measures to control the spread of infectious diseases but to control the spread of further infection and eliminate the infection from society, there is a need for effective treatment. The treatment rate has a substantial role in reducing complications and preventing the transmission of infectious diseases to others. The appropriate and timely treatment strategy can significantly lessen the effect of disease on society. The basic idea behind any treatment is to cure a disease and to control its spread. In 2004, Wang and Ruan [39] considered a SIR epidemic model with a constant treatment rate (i.e., the recovery from infected subpopulation per unit time) as given below:

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

where b is a positive constant and I denotes the number of infected individuals. They investigated the stability of the model and proposed that their model could exhibit various bifurcations. This kind of treatment rate is appropriate for a small number of infectives and sufficient treatment resources but inappropriate when the number of infected individuals is large and treatment resources are limited. Therefore, Zhang and Liu [55] presented the improved continuous, differentiable nonlinear treatment rate function, which saturates at its maximum value. This saturated treatment rate is known as the Holling type II treatment rate [49]. For an outbreak of epidemic disease, the treatment capacity of Holling type II is initially very slow, which develops gradually with improved treatment facilities. Further, Holling type II treatment rate refers to the condition that when there is a large number of infected people, then the treatment capacity reaches its maximum because of limited treatment facilities, which mimic the more realistic phenomena.

Delay differential equation reveals more complex dynamical behavior than an ordinary differential equation. The time delay is an influential parameter in the dynamical behavior of infectious diseases, and it can change the behavior of the dynamical system. This chapter studies the effect of the latent period on the SIR model and analyzes the transmission of epidemics into the susceptible host with the impact of inhibitions taken by both susceptible and infected individuals. We aim to study the effect of treatment rate also. We have considered the limited availability of resources, which allows us to reach more realistic phenomena. For this, we have considered a SIR epidemic model along with Beddington DeAngelis functional type incidence rate by incorporating time delay as latent period and Holling functional type II treatment rate. We derive the basic reproduction number for the proposed model and study the dynamical behavior of the model through stability analysis. We give a precise indication of the bifurcation phenomenon using center manifold theory, ensuring that either forward or backward bifurcation occurs. We also discuss the Hopf bifurcation near the endemic equilibrium by considering time delay as a bifurcation parameter.

3.2 Mathematical model

The fundamental approach in studying epidemic models is to divide the total population into mutually exclusive compartments according to epidemic status. We assume that the total size of the population N is constant. Therefore, the total constant popu-

lation is divided into three disjoint compartments $S(t)$, $I(t)$, and $R(t)$, where $S(t)$, $I(t)$, and $R(t)$ denotes the size of compartment of susceptible, infected, and recovered at time t, respectively. We assume that each population of SIR is well mixed and interacts homogeneously with each other [137]. That is, $N \equiv S(t)+I(t)+R(t)$ which means that N is a fixed population and $S(t)$, $I(t)$ and $R(t)$ may vary at time t.

The model we present is under the framework of the following system of non-linear delay differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= A - \vartheta S - \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha S(t-\rho)+\gamma I(t-\rho)}, \\
\frac{dI}{dt} &= \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha S(t-\rho)+\gamma I(t-\rho)} - (\vartheta + d + \zeta)I - \frac{aI}{1+bI}, \\
\frac{dR}{dt} &= \frac{aI}{1+bI} + \zeta I - \vartheta R.
\end{aligned} \tag{3.1}$$

The model (3.1) is considered with the following initial conditions:

$$S(\theta) = \phi_1(\theta),\ I(\theta) = \phi_2(\theta),\ R(\theta) = \phi_3(\theta)\ \phi_i(\theta) \geq 0,\ \theta \in [-\rho, 0],\ \phi_i(\theta) > 0\ (i = 1,2,3) \tag{3.2}$$

where $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in C([-\rho,0], R^3_+)$. Here, C denotes the Banach space of continuous functions mapping the interval $[-\rho,0]$ into R^3_+, and $\rho > 0$ represents the latent period.

The description of the parameters used in the model (3.1) is as follows: A represents the recruitment rate of the susceptible population by birth or immigration. ϑ and d are the natural death rate and disease-induced death rate, respectively. ζ is the recovery rate of infectives. The term $\frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha S(t-\rho)+\gamma I(t-\rho)}$ describes the Beddington- DeAngelis type nonlinear incidence rate, where β is the force of infection, α is the measure of inhibitions taken by susceptibles, i.e., social awareness among the susceptibles, and γ is the measure of inhibition taken by infected individuals, i.e., the magnitude of interference among the infectives. This incidence rate represents the rate at time $(t-\rho)$ at which a susceptible individual leaves the susceptible compartment and enters the infectious compartment at time t. The latent period means that the force of infection at present is determined by the number of infectives in the past. The term $h(I) = \frac{aI}{(1+bI)}$ is the Holling functional type II treatment term, where a denotes the cure rate of infectives and b denotes the limitation in the availability of resources.

3.3 Basic properties

Under the assumption that the population size N is constant, the model (3.1) may be reduced to a two-dimensional system for analysis purposes only. The equation for $R(t)$ is traditionally omitted; it is easy to see that under the assumption of constant population size, this equation is decoupled from the first and second equations of the model (3.1). The condition $N = S+I+R = constant$ may be used to find R [88, 137]. In this manner, it is equivalent to study the following reduced system:

$$\frac{dS}{dt} = A - \vartheta S - \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha S(t-\rho)+\gamma I(t-\rho)}, \tag{3.3}$$

$$\frac{dI}{dt} = \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha S(t-\rho)+\gamma I(t-\rho)} - (\vartheta + d + \zeta)I - \frac{aI}{1+bI} \tag{3.4}$$

with initial conditions

$$S(\theta) = \phi_1(\theta),\ I(\theta) = \phi_2(\theta),\ \phi_i(\theta) \geq 0,\ \theta \in [-\rho, 0],\ \phi_i(\theta) > 0\ (i = 1,2) \tag{3.5}$$

where, $(\phi_1(\theta), \phi_2(\theta)) \in C([-\rho, 0], R^2_+)$. Here, C denotes the Banach space of continuous functions mapping the interval $[-\rho, 0]$ into R^2_+.

We assume that $\Phi(S(t), I(t)) = \frac{\beta S(t)I(t)}{1+\alpha S(t)+\gamma I(t)}$ and $h(I(t)) = \frac{aI(t)}{1+bI(t)}$ are always positive, continuously differentiable, and monotonically increasing for all $S,\ I > 0$, i.e., the following postulates are satisfied:

P1. $\Phi(S(t), I(t)) > 0$, $\Phi'_S(S(t), I(t)) > 0$, $\Phi'_I(S(t), I(t)) > 0$ for $S(t) > 0$ and $I(t) > 0$.

P2. $\Phi(S(t), 0) = \Phi(0, I(t)) = 0$, $\Phi'_S(S(t), 0) = 0$, $\Phi'_I(S(t), 0) > 0$ for $S(t) > 0$ and $I(t) > 0$.

P3. $h(0) = 0$, $h'(0) > 0$ for $I(t) \geq 0$.

For biological reasons, we consider that all the parameters of the system (3.3)–(3.4) are positive, i.e., $A,\ \vartheta,\ \beta,\ \alpha,\ d,\ \gamma,\ \zeta,\ a,\ b > 0$. The equations of the system (3.3)–(3.4) monitor populations, therefore, it is essential to prove the positivity of the state variables, i.e., $(S, I) \in R^2_+$.

Lemma 3.3.1. *Any solution of system (3.3)–(3.4) with $\phi_i(\theta) > 0$, $\theta \in [-\rho, 0]$ remains positive whenever it exists and bounded in the region*

$$D = \{(S, I) \in R^2_+ : S(t) + I(t) \leq \frac{A}{\vartheta}\}.$$

Proof. Note that

$$\frac{dS}{dt}+\frac{dI}{dt}=A-\vartheta S-(\vartheta+d+\zeta)I-\frac{aI}{1+bI}\leq A-\vartheta(S+I). \tag{3.6}$$

and so,

$$\lim_{t\to\infty}(S+I)\leq\frac{A}{\vartheta}. \tag{3.7}$$

It follows that the system is point dissipative. Without loss of generality, we assume that $S(t)+I(t)\leq\frac{2A}{\vartheta}$ for all $t\geq-\rho$. A consequence of this is that we may assume I is bounded above, which in turn implies $\frac{dS}{dt}$ is positive for small S, and so S is positive for $t>0$ [122].
We now prove the positivity of $I(t)$. Using the variation of constants formula and applying step by step integration method, for $0<t\leq\rho$, we integrate the equation (3.4) from 0 to t , we get

$$I(t)=I(0)\cdot e^{(\vartheta+d+\zeta)}\cdot e^{\int_0^t f(S(\omega-\rho),I(\omega-\rho),I(\omega))d\omega}. \tag{3.8}$$

We see that $I(t)>0$ for all $\rho\leq t\leq 2\rho$. Proceeding in the same way, this process will carry on. Hence, it is proved that $I(t)>0$ for all $t>0$. ■

From (3.7), it follows that (S,I) is bounded in the region

$$D=\{(S,I)\in R_+^2 : S(t)+I(t)\leq\frac{A}{\vartheta}\}.$$

Hence, we state the following lemma:

Lemma 3.3.2. *The solutions $(S(t),I(t))$ of the system (3.3)–(3.4) with initial conditions (3.5) uniquely exists in the region*

$$D=\{(S,I)\in R_+^2 : S(t)+I(t)\leq\frac{A}{\vartheta}\}.$$

Proof. We already have proved the positivity and the boundedness of solution $(S(t),I(t))$. Now, we will prove the existence and uniqueness of this solution. We notice that the right-hand of the model (3.3)–(3.4) is completely continuous and locally Lipschitzian on D. Then, it follows that the solution of model (3.3)–(3.4) exists and is unique. This completes the proof of Lemma 3.3.2. ■

3.4 Equilibria and stability analysis

In this section, we show the existence of the equilibria of the system (3.3)–(3.4) and investigate the local and global stability behavior of the obtained equilibria. We obtain the equilibrium points of the time-delayed system by setting the right-hand side terms of the system (3.3)–(3.4) to zero since equilibrium solutions of a system with time delays are equivalent to the corresponding system with zero delay [109].

3.4.1 The disease-free equilibrium (DFE) and its analysis

The equilibria are acquired by equating the rate of change of all the compartments to zero. Consequently, we see that the system (3.3)–(3.4) has a unique DFE of the form $E_0\left(\frac{A}{\vartheta},0\right)$.

The characteristic equation of the system (3.3)–(3.4), evaluated at E_0 is as follows:

$$(\vartheta+\lambda)\left(\frac{\beta A}{\vartheta+\alpha A}e^{-\lambda\rho}-\vartheta-d-\zeta-a-\lambda\right)=0. \tag{3.9}$$

Eq. (3.9) has one negative real root $\lambda_1=-\vartheta$ and other roots are the solutions of

$$\frac{\beta A}{\vartheta+\alpha A}e^{-\lambda\rho}-\vartheta-d-\zeta-a-\lambda=0. \tag{3.10}$$

The term $\frac{\beta A e^{-\lambda\rho}}{(\vartheta+\alpha A)(\vartheta+d+\zeta+a)}$ at $\rho=0$, is termed as the basic reproduction number. Hence, the basic reproduction number for system (3.3) is

$$R_0=\frac{\beta A}{(\vartheta+\alpha A)(\vartheta+d+\zeta+a)}.$$

Analysis for $\mathbf{R_0\neq 1}$

As mentioned above, Eq. (3.9) has one negative real root $\lambda_1=-\vartheta$ and other roots satisfy the equation

$$\zeta(\lambda):=\lambda+\vartheta+d+\zeta+a-\frac{\beta A}{\vartheta+\alpha A}e^{-\lambda\rho}=0. \tag{3.11}$$

I. Assuming that $R_0>1$, then

$$\zeta(0)=\vartheta+d+\zeta+a-\frac{\beta A}{\vartheta+\alpha A}<0.$$

Therefore, $\zeta(0) < 0$. Since $\lim_{\lambda\to\infty}\zeta(\lambda) = +\infty$, there exists at least one positive real root of (3.11) if $R_0 > 1$.

II. Let be $R_0 < 1$.

Our goal is to prove that the characteristic roots cannot reach the imaginary axis for any values of the parameters. It means that for any values of the parameters and all delays ρ, it happens that $\mathrm{Re}\,\lambda < 0$. On the contrary, suppose that $\mathrm{Re}\,\lambda \geq 0$. Note that

$$\mathrm{Re}\,\lambda = \frac{\beta A}{\vartheta+\alpha A}e^{-\mathrm{Re}\,\lambda\rho}\cos(Im\lambda)\rho - \vartheta - d - \zeta - a < \frac{\beta A}{\vartheta+\alpha A} - \vartheta - d - \zeta - a < 0.$$

a contradiction to our assumption. Hence, if $R_0 < 1$ then all the roots of Eq. (3.11) must have the negative real part. Using the Routh – Hurwitz criterion, we have the following result.

Theorem 3.4.1. *The DFE E_0 is locally asymptotically stable if $R_0 < 1$ and unstable at $R_0 > 1$ for time lag $\rho \geq 0$.*

***Analysis for* $\mathbf{R_0 = 1}$**

We analysis the system (3.3)–(3.4) at $R_0 = 1$ for $\rho > 0$ and $\rho = 0$ separately.

(i) $\boldsymbol{\rho > 0}$**:** If $R_0 = 1$, then $\lambda = 0$ is a simple characteristic root of Eq. (3.11). Let $\lambda = \alpha + \iota\omega$ any of the other solutions, then (3.11) turns into:

$$\alpha + \iota\omega + \vartheta + d + \zeta + a - \frac{\beta A}{\vartheta+\alpha A}e^{-(\alpha+\iota\omega)\rho} = 0. \tag{3.12}$$

Applying Euler's formula and parting real and imaginary parts, we can write

$$\alpha + \vartheta + d + \zeta + a = \frac{\beta A}{\vartheta+\alpha A}\cos(\omega\rho)e^{-\alpha\rho}, \tag{3.13}$$

$$\omega = -\frac{\beta A}{\vartheta+\alpha A}e^{-\alpha\rho}\sin(\omega\rho). \tag{3.14}$$

Observing that $R_0 = 1$ implies $\beta A = (\vartheta+\alpha A)(\vartheta+d+\zeta+a)$. Moreover, if there exist roots satisfying both Eqs. (3.13) and (3.14), then they also satisfy the equation obtained by squaring and adding them member to member, we obtain

$$(\alpha+\vartheta+d+\zeta+a)^2 + \omega^2 = (\vartheta+d+\zeta+a)^2 e^{-2\alpha\rho}. \tag{3.15}$$

For Eq. (3.15) to be verified we must have $\alpha \leq 0$. Therefore, DFE E_0 is linearly neutrally stable.

(ii) $\boldsymbol{\rho = 0}$**:** Note that the system (3.3)–(3.4) evaluated at $R_0 = 1$ and bifurcation parameter $\beta = \beta^* = \frac{(\vartheta+\alpha A)(\vartheta+d+\zeta+a)}{A}$ has a simple zero (null) eigenvalue and other eigenvalue is real and negative. Hence, when $R_0 = 1$, the DFE E_0 is a non-hyperbolic equilibrium. To investigate the stability of DFE at $R_0 = 1$, we use the bifurcation theory approach based on the center manifold theory [14]. For this, we redefine $S = x_1$ and $I = x_2$ then the system (3.3)–(3.4) can be rewritten as

$$\frac{dx_1}{dt} = A - \vartheta x_1 - \frac{\beta x_1 x_2}{1+\alpha x_1 + \gamma x_2}, \tag{3.16}$$

$$\frac{dx_2}{dt} = \frac{\beta x_1 x_2}{1+\alpha x_1 + \gamma x_2} - (\vartheta + d + \zeta)x_2 - \frac{a x_2}{1+b x_2} \tag{3.17}$$

Let $J(E_0, \beta^*)$ denotes the Jacobian matrix of the system (3.16)-(3.17) evaluated at $R_0 = 1$, and $\beta = \beta^*$. Then

$$J(E_0, \beta^*) = \begin{bmatrix} -\vartheta & -\frac{\beta^* A}{(\vartheta+\alpha A)} \\ 0 & 0 \end{bmatrix}.$$

The left eigenvector $u = [u_1, u_2]$ of the Jacobian matrix $J(E_0, \beta^*)$ is given by $u \cdot J(E_0, \beta^*)$. We obtain

$$u_1 = 0,\ u_2 = 1.$$

The right eigenvector $w = (w_1,\ w_2)^T$ of the Jacobian matrix $J(E_0, \beta^*)$ is given by is given by $J(E_0, \beta^*) \cdot w$. We obtain

$$w_1 = -\frac{\beta^* A}{\vartheta(\vartheta + \alpha A)},\ w_2 = 1.$$

Let f_i, $i = 1, 2$ denotes the right-hand side of the system (3.16)–(3.17). It can be checked that:
$\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_{E_0} = \frac{\beta^* \vartheta^2}{(A\alpha+\vartheta)^2}$, $\left(\frac{\partial^2 f_2}{\partial x_2 \partial x_1}\right)_{E_0} = \frac{\beta^* \vartheta^2}{(A\alpha+\vartheta)^2}$, $\left(\frac{\partial^2 f_2}{\partial x_2^2}\right)_{E_0} = 2ab - \frac{2A\beta^*\gamma\vartheta}{(A\alpha+\vartheta)^2}$, $\left(\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*}\right)_{E_0} = \frac{A}{A\alpha+\vartheta}$.

Then, by [40], we obtain the bifurcation constants a_1 and b_1 as

$$a_1 = \sum_{k,i,j=1}^{2} u_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_{E_0}$$

$$= \frac{2(ab(A\alpha+\vartheta)^3 - A\beta^*\vartheta(\beta^*+\gamma(A\alpha+\vartheta)))}{(A\ \alpha+\vartheta)^3},$$

$$b_1 = \sum_{k,i=1}^{2} u_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_{E_0}$$
$$= \frac{A}{A\alpha+\vartheta}.$$

It can be seen that b_1 is always positive. Therefore, by Theorem 4.1 of Castillo-Chavez and Song [40] the local dynamics of the system (3.16)–(3.17) depends on the sign of the bifurcation constant a_1. Evaluating a_1 at bifurcation constant β^*, we get

$$a_1 = -\frac{2(\vartheta a^2 + (-Ab(A\alpha+\vartheta)+\vartheta(2d+A\gamma+2(\zeta+\vartheta)))a+\vartheta(d+\zeta+\vartheta)(d+A\gamma+\zeta+\vartheta))}{A(A\alpha+\vartheta)},$$
$$= -\frac{g(a)}{A(A\alpha+\vartheta)},$$

where,

$$\begin{aligned} g(a) &= \vartheta a^2 + (\vartheta(2d+A\gamma+2(\zeta+\vartheta)) - Ab(A\alpha+\vartheta))a + \vartheta(d+\zeta+\vartheta)(d+A\gamma+\zeta+\vartheta) \\ &= A_1 a^2 + A_2 a + A_3, \end{aligned} \tag{3.18}$$

with

$$\begin{aligned} A_1 &= \vartheta, \\ A_2 &= \vartheta(2d+A\gamma+2(\zeta+\vartheta)) - Ab(A\alpha+\vartheta), \\ &= \vartheta(A(-b+\gamma)+2(d+\zeta+\vartheta)) - A^2 b\alpha, \\ A_3 &= \vartheta(d+\zeta+\vartheta)(d+A\gamma+\zeta+\vartheta). \end{aligned}$$

It can be seen that A_1 and A_3 are always positive. Let D denotes the discriminant of the quadratic $g(a)$ given in the Eq. (3.18). We obtain that

$$\begin{aligned} D &= A_2^2 - 4A_1A_3 \\ &= A^2\gamma^2\vartheta^2 + Ab(A\alpha+\vartheta)(A^2 b\alpha + \vartheta(A(b-2\gamma) - 4(d+\zeta+\vartheta))). \end{aligned} \tag{3.19}$$

Let a_1^* and a_2^* be two positive roots of the quadratic Eq. (3.18), given by

$$a_1^* = \frac{-A_2 - \sqrt{D}}{2A_1}, \quad \text{and} \quad a_2^* = \frac{-A_2 + \sqrt{D}}{2A_1}.$$

With the help of Theorem 4.1 [40], it can be concluded that if $g(a) > 0$, a forward bifurcation occurs, whereas if $g(a) < 0$, the system (3.16)–(3.17) exhibits a backward bifurcation.

We discuss these two cases separately.

1. **Backward bifurcation**

   The system (3.16)–(3.17) exhibits a backward bifurcation when $g(a) < 0$. The existence of a backward bifurcation implies a range that shows the region of coexistence of DFE and two endemic equilibrium: a smaller endemic equilibrium (i.e., there is less number of infectives) and a larger endemic equilibrium (i.e., there is a large number of infectives). In this case, the smaller endemic equilibrium is unstable, whereas the larger endemic equilibrium is stable. Thus, the occurrence of backward bifurcation shows that R_0 less than unity does not confirm the eradication of infection. Therefore, we now find the range of a for which backward bifurcation occurs.
   Now, $g(a) < 0$ if and only if the discriminant $D > 0$, $A_2 < 0$, and $a_1^* < a < a_2^*$. Thus the conditions, obtained for the occurrence of backward bifurcation are stated as below:

$$\begin{cases} A_2 < 0, \\ D > 0, \\ a_1^* < a < a_2^*. \end{cases} \tag{3.20}$$

2. **Forward bifurcation**

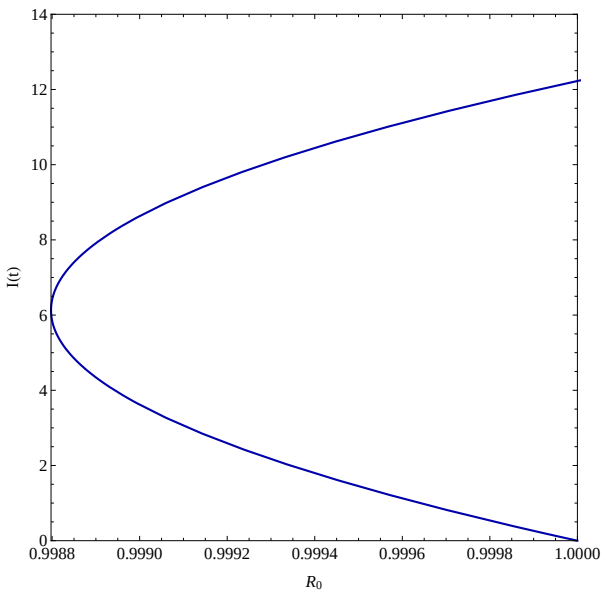
   The presence of forward bifurcation confirms the eradication of the disease when $R_0 < 1$. It shows that the DFE is stable when $R_0 < 1$ and if R_0 crosses unity, then the model admits a unique stable endemic equilibrium. From this analysis, we will be able to know that if there is a stable coexistence equilibrium bifurcating from E_0 and E_0 changes its stability from stable to unstable. This behavior is known as forward bifurcation. Therefore, it is very important to find the range for which forward bifurcation occurs. We see that the system (3.16)–(3.17) exhibits a forward bifurcation if $g(a) > 0$. $g(a) > 0$ if either $A_2 > 0$ or $D > 0$, $A_2 < 0$, and $a < a_1^*$ or $a > a_2^*$. These cases are described below:

$$\left\{ A_2 > 0 \text{ or, } b < \frac{(2d\vartheta + A\gamma\vartheta + 2\zeta\vartheta + 2\vartheta^2)}{A(A\alpha + \vartheta)}. \right. \tag{3.21}$$

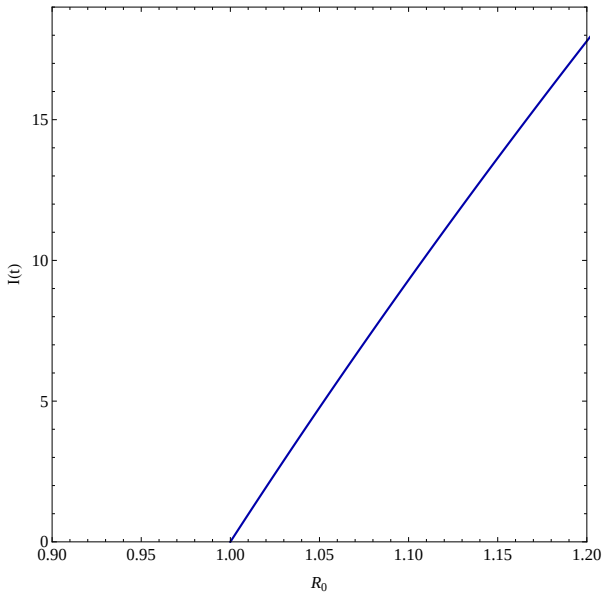
or

$$\begin{cases} A_2 < 0, \\ D > 0, \\ a < a_1^* \text{ or } a > a_2^*. \end{cases} \tag{3.22}$$

These bifurcations are illustrated in Figs. (3.1.1), and (3.1.2). We consider the parameter values $A = 5$, $\alpha = 1$, $\vartheta = 0.05$, $d = 0.001$, $\gamma = 0.01$, $\zeta = 0.002$, $b = 0.002$. Most of the parameter values are taken from [125]. At these values of parameters, we obtain that $D = 0.0420559$ and $A_2 = -0.0427$. According to (3.20), we have a backward bifurcation at $\beta = \beta^*$, for $a_1^* = 0.00644085 < a < 0.847559 = a_2^*$. Setting $a = 0.8$, the system (3.16)-(3.17) exhibits a backward bifurcation as shown in Fig. (3.1.1). A forward bifurcation occurs by choosing $a < a_1^*$ or $a > a_2^*$, as stated in (3.22). Setting $a = 0.005 < a_1^* = 0.00644085$, we see the occurrence of forward bifurcation as shown in Fig. (3.1.2).



3.1.1: Backward bifurcation.



3.1.2: Forward bifurcation.

Figure 3.1: Bifurcation diagrams in the plane (R_0, I).

Now, in order to satisfy inequality (3.21), we use the parameter $A = 5$, $\alpha = 0.004$, $\gamma = 0.01$, $\vartheta = 0.05$, $d = 0.001$, $\zeta = 0.002$, $b = 0.002$, $a = 0.005$. At these parameter values, we see that the inequality (3.21), $b = 0.002 < \frac{(2d\vartheta + A\gamma\vartheta + 2\zeta\vartheta + 2\vartheta^2)}{A(A\alpha + \vartheta)} = 0.0222857$ is satisfied and forward bifurcation occurs as shown in Fig. (3.2).

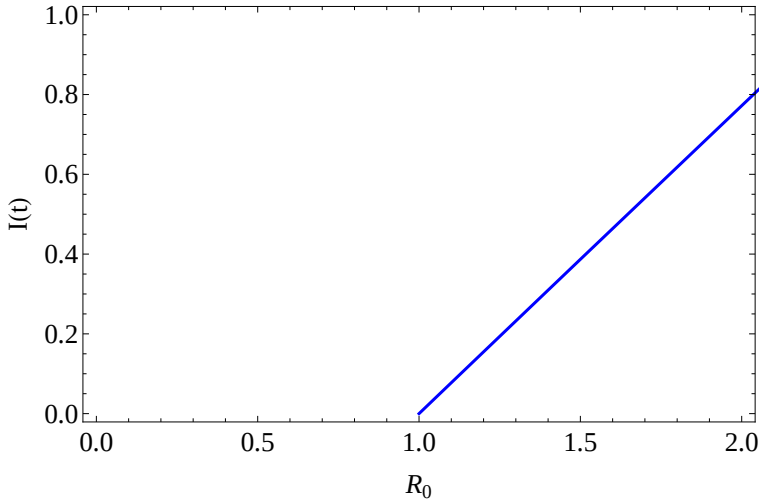


Figure 3.2: Forward bifurcation diagram in the plane (R_0, I), when the inequality (3.21) holds.

Theorem 3.4.2. *The disease-free equilibrium* $E_0 = \left(\frac{A}{\vartheta}, 0\right)$ *of the system (3.3)–(3.4) at* $R_0 = 1$ *is linearly neutrally stable for* $\rho > 0$.

Theorem 3.4.3. *When* $R_0 = 1$, *then the system (3.16)–(3.17) exhibits backward (forward) bifurcation at disease-free equilibrium* E_0 *if and only if* $g(a) < 0$ (> 0).

Remark 3.4.4. *The theorem above states that at* $\rho = 0$ *and for values of* R_0 *greater than* 1 *but close to* 1, *the system admits a unique infected equilibrium, which is locally asymptotically stable.*

3.4.2 Endemic equilibrium and its stability

To investigate the stability of the system (3.3)–(3.4) around the endemic equilibrium $E_e(S^*, I^*)$, we equate the second equation of the system (3.3)–(3.4) to zero:

$$\frac{\beta S^* I^*}{1 + \alpha S^* + \gamma I^*} - (\vartheta + d + \zeta) I^* - \frac{a I^*}{1 + b I^*} = 0. \tag{3.23}$$

After solving Eq. (3.23), we get S^* as follows

$$S^* = \frac{(1 + \gamma I^*)(a + (1 + b I^*) p)}{(\beta - \alpha a - \alpha p) + b I^* (\beta - \alpha p)}, \quad p = \vartheta + d + \zeta. \tag{3.24}$$

We see that $S^* > 0$ if and only if

$$\beta - \alpha a - \alpha p > 0, \quad \text{i.e.,} \quad \beta - \alpha p > \alpha a > 0. \tag{3.25}$$

Plug in the value of S^* from Eq. (3.24), into Eq. (3.3), we get the cubic equation in I^* as follows

$$H_0 + H_1 I^* + H_2 I^{*2} + H_3 I^{*3} = 0, \tag{3.26}$$

where,

$$\begin{aligned}
H_0 &= (\vartheta + \alpha A)(\vartheta + d + \zeta + a)(1 - R_0), \\
H_1 &= -a^2\alpha - (d + \zeta + \vartheta)(-\beta - 2b\vartheta - \gamma\vartheta + \alpha(d + \zeta + \vartheta)) + a(Ab\alpha + \beta + b\vartheta + \gamma\vartheta - \\
&\qquad 2\alpha(d + \zeta + \vartheta)) + 2Ab(-\beta + \alpha(d + \zeta + \vartheta)), \\
H_2 &= b(-(d + \zeta + \vartheta)(-2\beta - b\vartheta - 2\gamma\vartheta + 2\alpha(d + \zeta + \vartheta) + a(\beta + \gamma\vartheta - 2\alpha(d + \zeta + \vartheta))) + \\
&\qquad Ab(-\beta + \alpha(d + \zeta + \vartheta)), \\
H_3 &= b^2(d + \zeta + \vartheta)(\gamma\vartheta + \beta - \alpha(\vartheta + d + \zeta)).
\end{aligned} \tag{3.27}$$

Note that $H_3 > 0$ according to the condition given in Eq. (3.25).

Theorem 3.4.5. *If $R_0 > 1$, then there is either a unique or three positive endemic equilibria if all equilibria are simple roots.*

Proof. Suppose that $R_0 > 1$. From (3.27), it follows that H_0 is negative. From Eq. (3.26), we have a third-degree polynomial in I^*:

$$H_0 + H_1 I^* + H_2 I^{*2} + H_3 I^{*3} = 0.$$

Since H_3 is positive and H_0 is negative, we have the following possibilities of signs of H_1 and H_2:

$$\begin{aligned}
&(i)\ H_1 > 0, \text{ and } H_2 < 0, \\
&(ii)\ H_1 < 0, \text{ and } H_2 > 0, \\
&(iii)\ H_1 > 0, \text{ and } H_2 > 0, \\
&(iv)\ H_1 < 0, \text{ and } H_2 < 0.
\end{aligned} \tag{3.28}$$

Using Descartes' rule of signs [42], it follows that Eq. (3.26) has either three positive roots or a unique positive root. Hence the proof. ■

To analyze the local stability of endemic equilibrium E_e, we obtain the characteristic

equation of the system (3.3)–(3.4) at E_e as follows:

$$\lambda^2 + p_0\lambda + q_0 + (p_1\lambda + q_1)e^{-\lambda\rho} = 0, \tag{3.29}$$

where,

$$p_0 = 2\vartheta + d + \zeta + \frac{a}{(bI^*+1)^2},$$

$$q_0 = \vartheta(\vartheta + d + \zeta) + \frac{a\vartheta}{(bI^*+1)^2},$$

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

$$q_1 = \frac{\beta(aI^*(1+I^*\gamma) + (1+bI^*)^2(I^*(1+I^*\gamma)(d+\zeta) + (-S^*(1+S^*\alpha) + I^*(1+I^*\gamma))\vartheta))}{(bI^*+1)^2(1+S^*\alpha+y\gamma)^2}.$$

Theorem 3.4.6. *At $\rho = 0$, E_e is locally asymptotically stable if $p_0 + q_0 > 0$, and $p_1 + q_1 > 0$.*

Proof. The characteristic equation of the system (3.3)–(3.4) evaluated at E_e and $\rho = 0$ is

$$\lambda^2 + (p_0 + p_1)\lambda + (q_0 + q_1) = 0. \tag{3.30}$$

Using the Routh-Hurwitz criterion, it can be said that the endemic equilibrium E_e of the system (3.3)–(3.4) is locally asymptotically stable when $\rho = 0$ if $p_0 + p_1 > 0$ and $p_1 + q_1 > 0$ are satisfied simultaneuously. ■

Theorem 3.4.7. *For $\rho > 0$, E_e is locally asymptotically stable if both $p_0^2 - 2q_0 - p_1^2 > 0$ and $q_0^2 - q_1^2 > 0$ are satisfied simultaneuously.*

Proof. For $\rho > 0$, if instability occurs for a specific value of the time delay ρ, then a characteristic root of (3.29) must cross the imaginary axis [131].
Assume that $\lambda = \iota\xi$, $\xi > 0$ is a root of the characteristic Eq. (3.29). Substituting $\lambda = \iota\xi$ into equation (3.29), we get

$$(-\xi^2 + q_0 + p_1\xi\sin\xi\rho + q_1\cos\xi\rho) + \iota(p_0\xi + p_1\xi\cos\xi\rho - q_1\cos\xi\rho) = 0. \tag{3.31}$$

On separating real and imaginary part of the Eq. (3.31), we get:

$$p_1\xi\sin\xi\rho + q_1\cos\xi\rho = \xi^2 - q_0, \tag{3.32}$$

$$p_1\xi\cos\xi\rho - q_1\cos\xi\rho = -p_0\xi. \tag{3.33}$$

Eliminating ρ by squaring and adding Eqs. (3.32) and (3.33), we obtain a polynomial in ξ as follows

$$\xi^4 + (p_0^2 - 2q_0 - p_1^2)\xi^2 + (q_0^2 - q_1^2) = 0. \tag{3.34}$$

Letting $\xi^2 = x$, $p_0^2 - 2q_0 - p_1^2 = A$, and $q_0^2 - q_1^2 = B$, Eq. (3.34) becomes

$$x^2 + Ax + B = 0. \tag{3.35}$$

Using the Routh – Hurwitz Criterion, it can be observed that, the Eq. (3.35) has roots with negative real part, if $A > 0$ and $B > 0$ are satisfied simultaneously. It contradicts the assumption that Eq. (3.29) has a root $\lambda = \iota\xi$. Hence, the endemic equilibrium E_e of the system (3.3)–(3.4) is locally asymptotically stable for $\rho > 0$ if $p_0^2 - 2q_0 - p_1^2 > 0$ and $q_0^2 - q_1^2 > 0$ are satisfied simultaneuously. ■

Hopf-bifurcation

If $q_0^2 - q_1^2 < 0$, then there is a unique positive ω_0 satisfying the Eq. (3.35), i.e., there is a single pair of purely imaginary roots $\pm\iota\omega_0$ to Eq. (3.29).
Using Eqs. (3.32) and (3.33), we obtain ρ_k corresponding to ω_0

$$\rho_k = \frac{1}{\omega_0}\arccos\left(\frac{\omega_0^2(q_1 - p_0p_1) - q_0q_1}{p_1^2\omega_0^2 + q_1^2}\right) + \frac{2k\pi}{\omega_0}, \quad k = 0, 1, 2\ldots \tag{3.36}$$

For $\rho < \rho_0$, the endemic equilibrium E_0 is stable if transversality condition holds, That is,
$\frac{d}{dt}(\mathrm{Re}\,\lambda)\Big|_{\lambda=i\omega_0} \neq 0.$

Differentiating Eq. (3.29) with respect to ρ, we get

$$\left(2\lambda + p_0 + p_1e^{-\lambda\rho} - (p_1\lambda + q_1)\rho e^{-\lambda\rho}\right)\frac{d\lambda}{d\rho} = \lambda(p_1\lambda + q_1)e^{-\lambda\rho}. \tag{3.37}$$

$$\begin{aligned}\left[\frac{d\lambda}{d\rho}\right]^{-1} &= \frac{2\lambda + p_0 + p_1e^{-\lambda\rho} - (p_1\lambda + q_1)\rho e^{-\lambda\rho}}{\lambda(p_1\lambda + q_1)e^{-\lambda\rho}} \\ &= \frac{2\lambda + p_0}{\lambda(p_1\lambda + q_1)e^{-\lambda\rho}} + \frac{p_1}{\lambda(p_1\lambda + q_1)} - \frac{\rho}{\lambda} \\ &= \frac{2\lambda + p_0}{-\lambda(\lambda^2 + p_0\lambda + q_0)} + \frac{p_1}{\lambda(p_1\lambda + q_1)} - \frac{\rho}{\lambda}.\end{aligned}$$

$$\begin{aligned}
\frac{d}{d\rho}(\mathrm{Re}\,\lambda)\Big|_{\lambda=\iota\omega_0} &= \mathrm{Re}\left(\frac{d\lambda}{d\rho}\right)^{-1}\Big|_{\lambda=\iota\omega_0} \\
&= \mathrm{Re}\left(\frac{2\iota\omega_0+p_0}{-\iota\omega_0(-\omega_0^2+\iota p_0\omega_0+q_0)}+\frac{p_1}{-p_1{\omega_0}^2+\iota q_1\omega_0}+\frac{\iota\rho}{\omega_0}\right) \\
&= \frac{1}{\omega_0}\left(\frac{2\omega_0(\omega_0^2-q_0)+p_0^2\omega_0}{(p_0\omega_0)^2+(\omega_0^2-q_0)^2}-\frac{p_1^2\omega_0}{(p_1\omega_0)^2+q_1^2}\right) \\
&= \frac{2(\omega_0^2-q_0)+p_0^2}{(p_0\omega_0)^2+(\omega_0^2-q_0)^2}-\frac{p_1^2}{(p_1\omega_0)^2+q_1^2} \\
&= \frac{2\omega_0^2+(p_0^2-2q_0-p_1^2)}{p_1^2\omega_0^2+q_1^2} \quad \text{(Using Eqs. (3.32) and (3.33),} \\
&\qquad\qquad (p_1\omega_0)^2+q_1^2=(p_0\omega_0)^2+(\omega_0^2-q_0)^2).
\end{aligned}$$

Under the condition $p_0^2-2q_0-p_1^2>0$, we have $\frac{d}{d\rho}(\mathrm{Re}\,\lambda)\Big|_{\lambda=\iota\omega_0}>0$. Hence, the transversality condition holds, and Hopf bifurcation occurs at $\rho=\rho_0$ and ω_0. Thus, we state the following theorem:

Theorem 3.4.8. *If the condition $q_0^2-q_1^2<0$ is satisfied, then the endemic equilibrium E_e of the system (3.3)–(3.4) is asymptotically stable for $\rho\in[0,\rho_0)$ and it undergoes Hopf bifurcation at $\rho=\rho_0$.*

3.4.3 Global stability analysis

This subsection is devoted to analyzing the global stability behavior of the disease-free and endemic equilibria.

Global stability of disease-free equilibrium

For the global stability of the disease-free equilibrium $E_0\left(\frac{A}{\vartheta},0\right)$, we assume the following postulates:

P4. $\Phi_I'(S(t),0)$ is increasing with respect to $S(t)>0$.

P5. $\frac{\Phi_I'(S_0,0)}{\Phi_I'(S(t),0)}<1$ for $S(t)>S_0$, $\frac{\Phi_I'(S_0,0)}{\Phi_I'(S(t),0)}>1$ for $S(t)\in(0,S_0)$, where $S_0=\frac{A}{\vartheta}$.

P6. $\Phi(S(t),I(t))\le I(t)\left(\left(\frac{\partial\Phi(S(t),I(t))}{\partial I}\right)_{(S_0,0)}-\left(\frac{\partial H(I)}{\partial I}\right)_{I=0}\right)+H(I(t))$, $I(t)>0$,
where $H(I(t))=(\vartheta+d+\zeta)I(t)+h(I(t))$

Under these assumptions, we have the following theorem:

Theorem 3.4.9. *Assume that (P1.)–(P6.) are satisfied then, the disease-free equilibrium $E_0(S_0,0)$ of the system (3.3)–(3.4) is globally asymptotically stable for any $\rho\ge0$ if $R_0\le1$.*

Proof. From the conditions (P1.) and (P2.), it follows that the disease-free equilibrium $E_0(S_0,0)$ is the only equilibrium of the system (3.3)–(3.4). We define the following Lyapunov functional:

$$L(t) = L_1(t) + L_2(t)$$

where

$$L_1(t) = S(t) - S_0 - \int_{S_0}^{S(t)} \lim_{I\to 0^+} \frac{\Phi(S_0,I(t))}{\Phi(\sigma,I(t))} d\sigma + I(t),$$
$$L_2(t) = \int_0^{\rho} \Phi(S(t-\rho),I(t-\rho))d\rho.$$

The postulates (P1.)–(P3.) imply that $L_1(t)$ is defined and continuously differentiable for all $S(t) > 0$ and $I(t) > 0$, and $L(t) = 0$ at $E_0(S_0,0)$. We will show that $\frac{dL(t)}{dt} \leq 0$ for all $t \geq 0$. For this, first we calculate $\frac{dL_1(t)}{dt}$ as follows.

$$\frac{dL_1(t)}{dt} = \left(1 - \lim_{I\to 0^+} \frac{\Phi(S_0,I(t))}{\Phi(S,I(t))}\right) S'(t) + I'(t)$$

Since, $A - \vartheta S = -\vartheta\left(S - \frac{A}{\vartheta}\right) = -\vartheta(S - S_0)$, we get

$$\frac{dL_1(t)}{dt} = \left(1 - \lim_{I\to 0^+} \frac{\Phi(S_0,I(t))}{\Phi(S,I(t))}\right)(-\vartheta(S-S_0) - \Phi(S(t-\rho),I(t-\rho))) + \Phi(S(t-\rho),I(t-\rho)) - H(I(t)).$$

We now calculuate $\frac{dL_2(t)}{dt}$ as follows.

$$\frac{dL_2(t)}{dt} = -\Phi(S(t-\rho),I(t-\rho)) + \Phi(S(t),I(t))$$

Thus, $\frac{dL}{dt}$ is given by:

$$\begin{aligned}
\frac{dL(t)}{dt} &= \frac{dL_1(t)}{dt} + \frac{dL_2(t)}{dt} \\
&= \left(1 - \lim_{I\to 0^+} \frac{\Phi(S_0,I(t))}{\Phi(S,I(t))}\right)(-\vartheta(S-S_0) - \Phi(S(t-\rho),I(t-\rho))) + \Phi(S(t-\rho),I(t-\rho)) \\
&\quad - H(I) - \Phi(S(t-\rho),I(t-\rho)) + \Phi(S(t),I(t)) \\
&= \vartheta\left(1 - \lim_{I\to 0^+} \frac{\Phi(S_0,I(t))}{\Phi(S,I(t))}\right)(S_0 - S(t)) + \Phi(S(t-\rho),I(t-\rho))\left(\lim_{I\to 0^+} \frac{\Phi(S_0,I(t))}{\Phi(S,I(t))} - 1\right) \\
&\quad + \Phi(S(t),I(t)) - H(I).
\end{aligned}$$

Further, (P4.)–(P6.) implies that

$$\frac{dL(t)}{dt} \leq \vartheta\left(1-\frac{\Phi_I'(S_0,0)}{\Phi_I'(S(t),0)}\right)(S_0-S(t))+\Phi(S(t-\rho),I(t-\rho))\left(\frac{\Phi_I'(S_0,0)}{\Phi_I'(S(t),0)}-1\right)+$$
$$I(t)\left(\left(\frac{\partial\Phi(S(t),I(t))}{\partial I}\right)_{(S_0,0)}-\left(\frac{\partial H(I)}{\partial I}\right)_{I=0}\right)$$
$$=\vartheta\left(1-\frac{\Phi_I'(S_0,0)}{\Phi_I'(S(t),0)}\right)(S_0-S(t))+\Phi(S(t-\rho),I(t-\rho))\left(\frac{\Phi_I'(S_0,0)}{\Phi_I'(S(t),0)}-1\right)+$$
$$I(t)\left(\frac{\partial H(I)}{\partial I}\right)_{I=0}(R_0-1).$$

Thus, for $R_0 \leq 1$, $\frac{dL(t)}{dt} \leq 0$ for all $t \geq 0$. Also, $\frac{dL(t)}{dt} = 0$ if $S(t) = S_0$.
Hence, it follows from the system (3.3)–(3.4) that the largest invariant set $\left\{\left(S(t),I(t)\in R_+^2 \middle| \frac{dL(t)}{dt}=0\right)\right\}$ is singleton set $E_0(S_0,0)$. From the Lyapunov-LaSalle asymptotic stability theorem [23, 90, 113], E_0 is the only equilibrium of the system (3.3)–(3.4) and globally asymptotically stable. ■

Global stability of endemic equilibrium

We now study the global stability of the endemic equilibrium $E_e(S^*,I^*)$ of the system (3.3)–(3.4) by Lyapunov direct method. For this, the following hypothesis has been proposed:

P7. $\left(\frac{\Phi(S^*,I^*)}{\Phi(S(t),I^*)}-\frac{I^*}{I(t)}\right)\leq 0$; $\left(\frac{\Phi(S(t),I(t))}{\Phi(S^*,I^*)}-1\right)\leq 0$; $\left(\frac{\Phi(S(t),I^*)}{\Phi(S(t),I(t))}-\frac{I(t)}{I^*}\right)\leq 0$ for $I \geq I^*$.

P8. $\left(\frac{h(I^*)}{h(I(t))}-\frac{I^*}{I(t)}\right)\left(\frac{I(t)}{I^*}-1\right)\leq 0$ for $I \geq I^*$.

Under these conditions, we have the following theorem:

Theorem 3.4.10. *Suppose that the conditions (P1.)–(P3.) and (P7.)–(P8.) are satisfied then the endemic equilibrium $E_e(S^*,I^*)$ of the system (3.3)–(3.4) is globally asymptotically stable for any $\rho \geq 0$ if $R_0 > 1$.*

Proof. We assume the following Lyapunov functional

$$M(t) = M_1(t) + M_2(t),$$

where,

$$M_1(t) = S(t) - S^* - \int_{S^*}^{S(t)} \frac{\Phi(S^*,I^*)}{\Phi(\phi,I^*)}d\phi + I(t) - I^* - I^* \ln\frac{I(t)}{I^*},$$

$$M_2(t) = \Phi(S^*, I^*) \int_0^\rho \left(\frac{\Phi(S(t-\theta), I(t-\theta))}{\Phi(S^*, I^*)} - 1 - \ln \frac{\Phi(S(t-\theta), I(t-\theta))}{\Phi(S^*, I^*)} \right) d\theta. \quad (3.38)$$

By (P1.)–(P3.), $M(t) = M_1(t) + M_2(t)$ is defined and continuously differentiable for all $S(t)$, $I(t) > 0$ and $M(0) = 0$ at $E_e(S^*, I^*)$.

At $E_e(S^*.I^*)$, the system (3.3)–(3.4) has

$$A - \vartheta S^* = \Phi(S^*, I^*) = (\vartheta + d + \zeta) I^* + h(I^*). \quad (3.39)$$

The time derivative of $M_1(t)$ along the solution of system (3.3)–(3.4) is given by

$$\begin{aligned}
\frac{dM_1(t)}{dt} &= \left(1 - \frac{\Phi(S^*, I^*)}{\Phi(S(t), I^*)}\right) S'(t) + \left(1 - \frac{I^*}{I(t)}\right) I'(t) \\
&= \left(1 - \frac{\Phi(S^*, I^*)}{\Phi(S(t), I^*)}\right) (\vartheta S^* - \vartheta S(t) + \Phi(S^*, I^*) - \Phi(S(t-\rho), I(t-\rho))) + \\
&\quad \left(1 - \frac{I^*}{I(t)}\right) \left(\Phi(S(t-\rho), I(t-\rho)) - \frac{(\Phi(S^*, I^*) - h(I^*))}{I^*} I(t) - h(I(t))\right)
\end{aligned}$$

$$\begin{aligned}
&= \vartheta(S^* - S(t)) \left(1 - \frac{\Phi(S^*, I^*)}{\Phi(S(t), I^*)}\right) + \Phi(S^*, I^*) \left(1 - \frac{\Phi(S^*, I^*)}{\Phi(S(t), I^*)} + \frac{\Phi(S(t-\rho), I(t-\rho))}{\Phi(S(t), I^*)}\right) \\
&\quad - \frac{\Phi(S(t-\rho), I(t-\rho))}{I(t)} I^* + \Phi(S^*, I^*) \left(1 - \frac{I(t)}{I^*}\right) + \left(h(I^*) - \frac{h(I(t))}{I(t)} I^*\right) \left(\frac{I(t)}{I^*} - 1\right)
\end{aligned}$$

Further, the time derivative of $M_2(t)$ is given by

$$\begin{aligned}
\frac{dM_2(t)}{dt} &= \Phi(S^*, I^*) \frac{d}{dt} \int_0^\rho \left(\frac{\Phi(S(t-\theta), I(t-\theta))}{\Phi(S^*, I^*)} - 1 - \ln \frac{\Phi(S(t-\theta), I(t-\theta))}{\Phi(S^*, I^*)} \right) d\theta. \\
&= \Phi(S(t), I(t)) - \Phi(S(t-\rho), I(t-\rho)) + \Phi(S^*, I^*) \ln \frac{\Phi(S(t-\rho), I(t-\rho))}{\Phi(S(t), I(t))}.
\end{aligned}$$

Then, we have

$$\begin{aligned}
\frac{dM(t)}{dt} &= \frac{dM_1(t)}{dt} + \frac{dM_2(t)}{dt} \\
&= \vartheta(S^* - S(t)) \left(1 - \frac{\Phi(S^*, I^*)}{\Phi(S(t), I^*)}\right) + \Phi(S^*, I^*) \left(1 - \frac{\Phi(S^*, I^*)}{\Phi(S(t), I^*)} + \frac{\Phi(S(t-\rho), I(t-\rho))}{\Phi(S(t), I^*)}\right) \\
&\quad - \frac{\Phi(S(t-\rho), I(t-\rho))}{I(t)} I^* + \Phi(S^*, I^*) \left(1 - \frac{I(t)}{I^*}\right) + \left(h(I^*) - \frac{h(I(t))}{I(t)} I^*\right) \left(\frac{I(t)}{I^*} - 1\right) \\
&\quad + \Phi(S(t), I(t)) - \Phi(S(t-\rho), I(t-\rho)) + \Phi(S^*, I^*) \ln \frac{\Phi(S(t-\rho), I(t-\rho))}{\Phi(S(t), I(t))}
\end{aligned}$$

$$
\begin{aligned}
&= \vartheta(S^* - S(t))\left(1 - \frac{\Phi(S^*,I^*)}{\Phi(S(t),I^*)}\right) + \Phi(S^*,I^*)\left(1 - \frac{\Phi(S^*,I^*)}{\Phi(S(t),I^*)} + \ln\frac{\Phi(S^*,I^*)}{\Phi(S(t),I^*)}\right) \\
&+ \Phi(S(t-\rho),I(t-\rho))\left(\frac{\Phi(S^*,I^*)}{\Phi(S(t),I^*)} - \frac{I^*}{I(t)}\right) + \Phi(S^*,I^*)\left(1 - \frac{I(t)}{I^*} + \ln\frac{I(t)}{I^*}\right) \\
&+ \left(\frac{h(I^*)}{h(I(t))} - \frac{I^*}{I(t)}\right)\left(\frac{I(t)}{I^*} - 1\right)h(I(t)) + \Phi(S^*,I^*)\Bigg(1 - \frac{\Phi(S(t-\rho),I(t-\rho))}{\Phi(S(t),I(t))}\cdot\frac{\Phi(S(t),I^*)}{\Phi(S^*,I^*)}\cdot \\
&\frac{I^*}{I(t)} + \ln\frac{\Phi(S(t-\rho),I(t-\rho))}{\Phi(S(t),I(t))}\cdot\frac{\Phi(S(t),I^*)}{\Phi(S^*,I^*)}\cdot\frac{I^*}{I(t)}\Bigg) + \Phi(S^*,I^*)\left(\frac{\Phi(S(t),I(t))}{\Phi(S^*,I^*)} - 1\right) \\
&+ \Phi(S(t-\rho),I(t-\rho))\cdot\frac{I^*}{I(t)}\left(\frac{\Phi(S(t),I^*)}{\Phi(S(t),I(t))} - \frac{I(t)}{I^*}\right).
\end{aligned}
$$

The function $\Phi(S(t),I(t))$ is monotonically increasing for all $S(t) > 0$. Therefore,

$$
(S^* - S(t))\left(1 - \frac{\Phi(S^*,I^*)}{\Phi(S(t),I^*)}\right) \leq 0. \tag{3.40}
$$

The function $f(x) = 1 - x + \ln x$, $(x > 0)$ has its global maximum $f(1) = 0$. Hence, for $x > 0$, $f(x) \leq 0$ and we have the following inequalities:

$$
\begin{aligned}
\left(1 - \frac{\Phi(S^*,I^*)}{\Phi(S(t),I^*)} + \ln\frac{\Phi(S^*,I^*)}{\Phi(S(t),I^*)}\right) &\leq 0, \\
\left(1 - \frac{I(t)}{I^*} + \ln\frac{I(t)}{I^*}\right) &\leq 0, \\
\left(1 - \frac{\Phi(S(t-\rho),I(t-\rho))}{\Phi(S(t),I(t))}\cdot\frac{\Phi(S(t),I^*)}{\Phi(S^*,I^*)}\cdot\frac{I^*}{I(t)}\right) + \ln\frac{\Phi(S(t-\rho),I(t-\rho))}{\Phi(S(t),I(t))}\cdot\frac{\Phi(S(t),I^*)}{\Phi(S^*,I^*)}\cdot\frac{I^*}{I(t)} &\leq 0.
\end{aligned} \tag{3.41}
$$

Hence, by (P7.)–(P8.) and inequalities (3.40)–(3.41), it follows that $\frac{dM(t)}{dt} \leq 0$ for all $S(t) \geq 0$, $I(t) \geq 0$. It can be verified that the largest invariant in $\left\{(S(t),I(t)) \in R_+^2 : \frac{dM(t)}{dt} = 0\right\}$ is singleton $\{E_e\}$. By the Lyapunov-LaSalle asymptotic stability theorem [23, 90, 113], E_e is globally asymptotically stable.

■

3.5 Numerical simulation

In this section, we present numerical simulations of the proposed model. Parameters description and values are given in Table 3.1 and the total population size is considered as $N = 100$.

Table 3.1: List of parameters

Parameter	Description	Value	Reference
A	Recruitment rate	5	Assumed
α	Inhibitions taken by susceptibles	0.004	[125]
β	Transmission rate	0.0045	Assumed
ϑ	Natural death rate	0.05	[125]
d	Disease-induced death rate	0.001	[125]
γ	Inhibitions taken by infectives	0.002	[125]
ζ	Recovery rate	0.002	[125]
a	Cure rate	0.04	Assumed
b	Rate of limitation in treatment availability	0.002	Assumed

Initial conditions are given by

$$S(0) = 90, \quad I(0) = 8, \quad R(0) = 2.$$

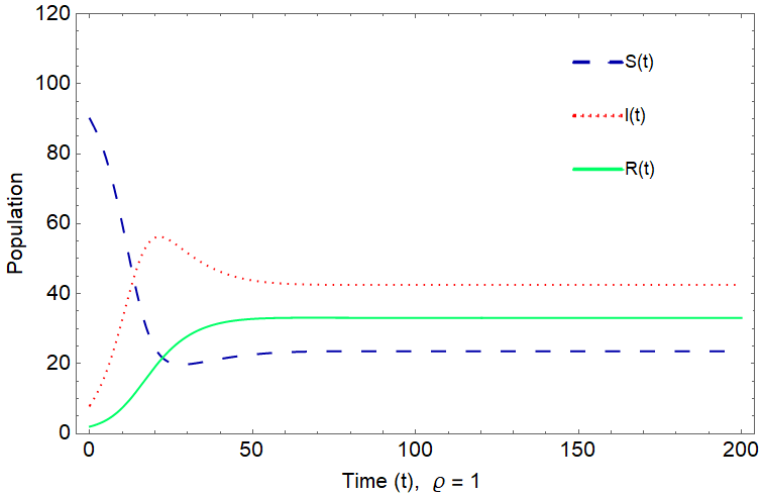


Figure 3.3: Behavior of susceptible-infected-recovered populations at time delay $\rho = 1$.

Fig. 3.3 shows the behavior of susceptible-infected-recovered populations at time delay $\rho = 1$. At the parameters values given in table 3.1, we obtained that $S^* = 23.5845$, $I^* = 42.5367$. From this S^*, and I^*, R^* can be evaluated as: $R^* = N - S^* - I^* = 100 - 23.5845 - 42.5367 = 33.8788$. This is also verified from the Fig. 3.3. This figure shows that as

time increases, all the subpopulations reach to their endemic equilibrium $(S^*, I^*, R^*) = (23.5845, 42.5367, 33.8788)$.

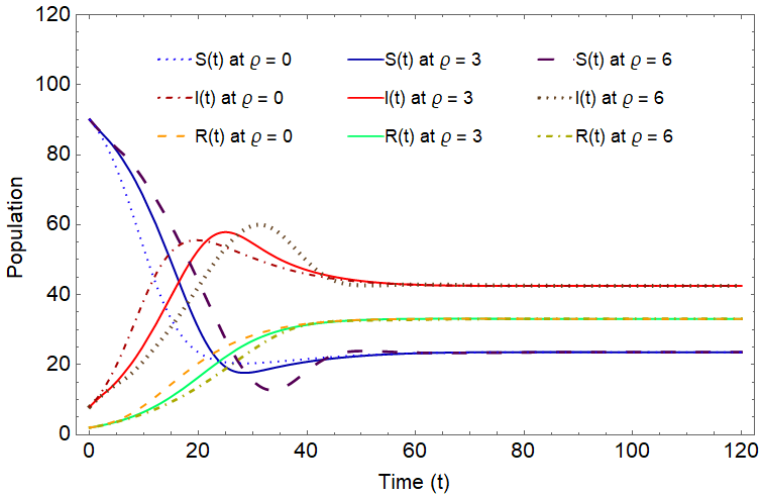


Figure 3.4: Impact of time delay ρ on susceptible, infected and recovered populations.

Fig. 3.4 shows the effect of time delay on the population with respect to time t. In this figure, susceptible, infected, and recovered populations are being drawn for the time delay $\rho = 0$, 3, and 6 respectively. It can be seen that with the increased values of time delay ρ, the infected individuals increases whereas susceptible and recovered individuals decrease and finally settles down to their endemic equilibrium $(23.5845, 42.5367, 33.8788)$. Thus, the time delay is an influential parameter in the transmission dynamics of infection.

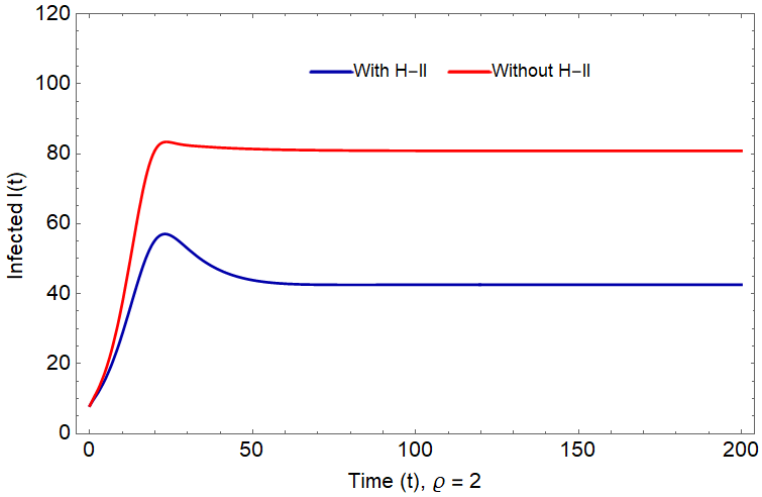
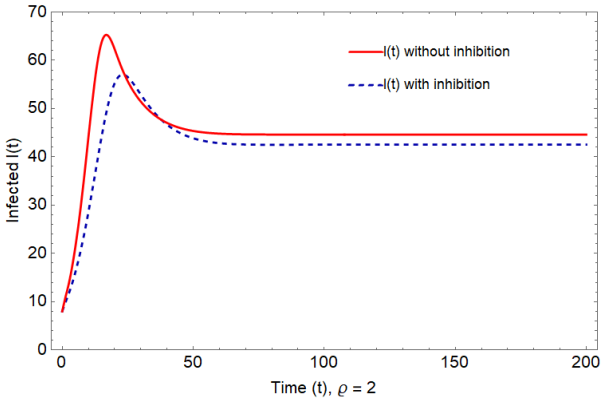


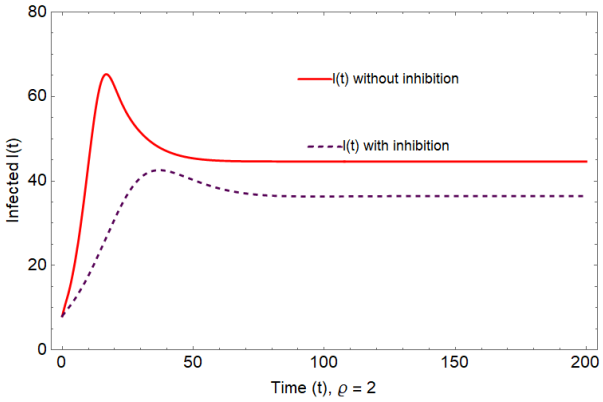
Figure 3.5: Infected population with and without Holling type II treatment rate.

Fig. 3.5 depicts the infected population with and without the effect of the Holling type II treatment rate. This figure shows the significant difference between the progression of treated infected individuals and non-treated infected individuals. When Holling type II

treatment rate is considered, then the infected individuals first increase and then stabilize to steady-state at a lower level, whereas in the absence of treatment rate, infections increase at a higher rate and then settle down to steady-state with a big difference.



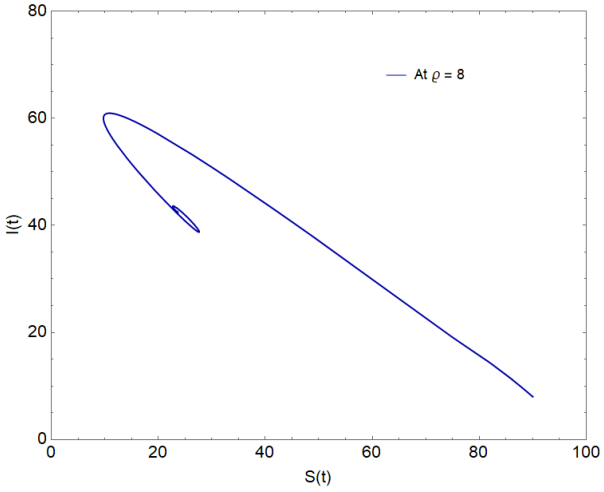
3.6.1: $\alpha = 0.004, \gamma = 0.002$.



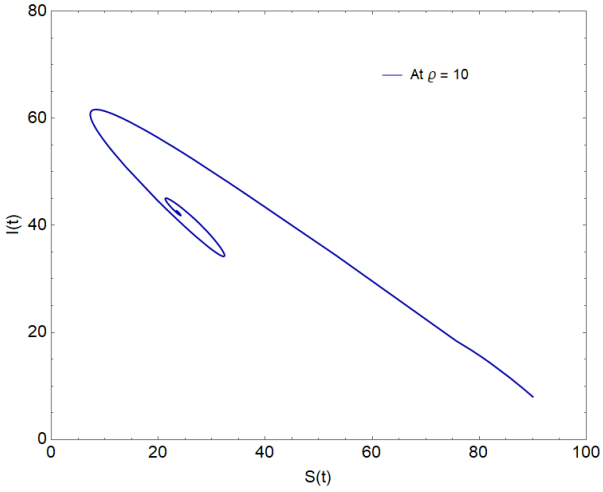
3.6.2: $\alpha = 0.01,\ \gamma = 0.01$.

Figure 3.6: Graphs depicting the effect of inhibitions taken by both susceptible and infected individuals.

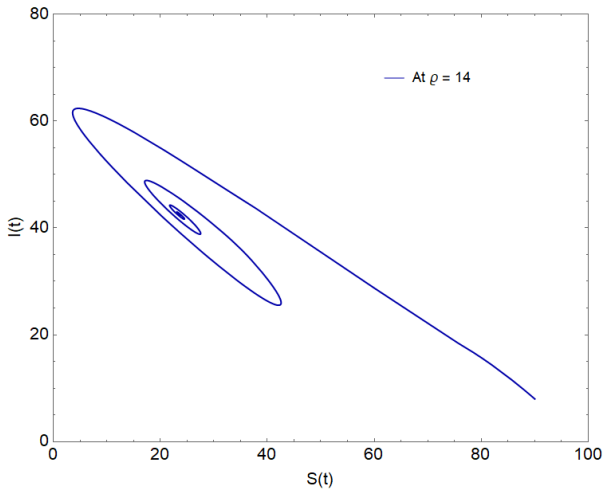
Fig. 3.6 shows the impact of the measure of inhibitions taken by susceptible and infected individuals. Figs. 3.6.1 and 3.6.2 show the difference between the infected population with and without inhibition effects. The solid red line shows the infected population without inhibition effects. When there is no inhibition taken by susceptible and infectives, then infectives increase at a higher rate. The dashed blue line shows the infected population when we consider $\alpha = 0.004$ and $\gamma = 0.002$ whereas dashed-purple line shows the infected population when $\alpha = 0.01$ and $\gamma = 0.01$. Thus, the higher the inhibitions rate, the higher the decline in the infected population.



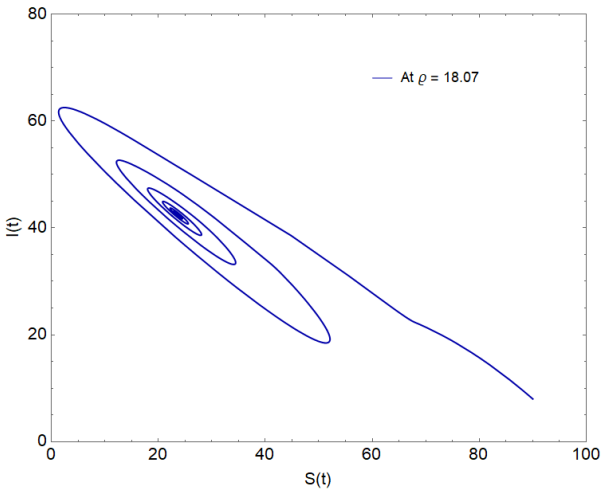
3.7.1: $S(t)$ vs. $I(t)$ for $\rho = 8$.



3.7.2: $S(t)$ vs. $I(t)$ for $\rho = 10$.



3.7.3: $S(t)$ vs. $I(t)$ for $\rho = 14$.



3.7.4: $S(t)$ vs. $I(t)$ for $\rho = 18.07$.

Figure 3.7: Hopf bifurcation in the plane (S,I) for various values of time delay ρ.

Figs. 3.7 shows the occurrence of Hopf bifurcation at different values of time delay ρ in the plane (S,I). We find that $q_0^2 - q_1^2 = -0.0000587362 < 0$, $p_0^2 - 2q_0 - p_1^2 = 0.00573205 > 0$, and there is a unique pair of purely imaginary roots $\pm\iota\omega$ of characterstic Eq. (3.29) with $\omega = \pm 0.105111$. Hence, the hypothesis of the occurrence of Hopf bifurcation is satisfied. According to the algorithm given in (3.36), we obtain the value of ρ_0 as $\rho_0 = 18.07$. Figs. 3.7.1, 3.7.2, and 3.7.3 show the oscillatory solutions for the time delay $\rho = 8,\ 10,\ 14 \in [0,\rho_0]$, respectively. Theorem 3.4.8 implies that E_e is asymptotically stable for $[0,\rho_0)$ which follows from the Figs. 3.7.1, 3.7.2, and 3.7.3, thus, periodic solution appears and endemic equilibrium is locally asympotitically stable which converges to $E_e = (23.5845, 42.5367)$. Also, Hopf bifurcation occurs at $\rho_0 = 18.07$ (Fig. 3.7.4) which confirms the Theorem 3.4.8.

3.6 Discussion

This chapter investigates the role of time delay and effects of inhibition measures and treatment on the SIR model with a nonlinear Beddington − DeAngelis type incidence rate and Holling type II treatment rate. We obtain that the model exhibits two equilibria; disease-free equilibrium E_0 and endemic equilibrium E_e. Stability analysis is resorted to getting a sense of the behavior of solutions. We investigate the local stability of the model near disease-free equilibrium point E_0 by deriving the basic reproduction number R_0. We observe that the disease-free equilibrium E_0 is locally asymptotically stable when the basic reproduction number R_0 is less than one, unstable when R_0 is greater than one, and linearly neutrally stable when R_0 equals to one for the time delay $\rho > 0$. By applying

center manifold theory, we investigate the stability behavior of the undelayed system at E_0 when R_0 is one, and show that the model exhibits a backward (forward) bifurcation under certain conditions. Backward bifurcation plays a relevant role in disease control and eradication. We find the bifurcation range for backward (forward) occurrence, which depends on the cure rate a. More accurately, if the cure rate a lies in a specific range, $a_1^* < a < a_2^*$, then there is an occurrence of backward bifurcation, and if a is sufficiently small or sufficiently large, i.e., if $a < a_1^*$ or $a > a_2^*$ then forward bifurcation occurs, as obtained in the inequalities (3.20) and (3.22). Also, forward bifurcation may occur if the limitation in the availability of resources is below a certain quantity, as obtained in the inequality (3.21). This shows that the resource limitation should be minimized for eradicating the epidemic when $R_0 < 1$. Thus, the Holling type II treatment rate has a significant role in eradicating disease. Further, we found the conditions for the existence of endemic equilibrium E_e and investigated the local stability behavior of the system near-endemic equilibrium E_e and obtained some interesting results. We also discussed the Hopf bifurcation near-endemic equilibrium by considering delay (ρ) as a bifurcation parameter, and we obtained that the endemic equilibrium is asymptotically stable for $\rho \in [0, \rho_o)$ and the model undergoes Hopf bifurcation at the critical value ρ_0. Moreover, the global stability of disease-free and endemic equilibria has been investigated and obtained that the disease-free equilibrium is globally asymptotically stable when $R_0 \leq 1$, as stated in Theorem 3.4.9, whereas the endemic equilibrium is globally asymptotically stable when $R_0 > 1$, as stated in Theorem 3.4.10, epidemiologically this means that the disease will die out or persist in a population concerning certain restrictions on the parameters.

In addition, we present the numerical simulations of the proposed model to illustrate the theoretical results. We observe that when the time delay is high, the infection occurs at a higher rate than the situation when there is no delay. Further, when the Holling type II treatment rate is given to the infected individuals at the appropriate time, there is a major difference in the occurrence of infection. Hence to eliminate the disease, there is a need to improve medical technology and invest more medicines, beds, etc., in giving the patients timely treatment. Also, it is depicted that if treatment is not available, then infection is high even if inhibitions are considered. Therefore, the Holling type II treatment rate leads to a diminished level of infection. The impact of inhibitions is also shown in the numerical simulation, which guarantees infection control in society if inhibitions are taken properly. The data shows that critical value ρ_0 is 18.07 and for the time lag $\rho \in [0, \rho_0)$, E_e is asymptotically stable as shown in Figs. 3.7.1, 3.7.2, 3.7.3

implies that the disease will persist in society, and at $\rho = \rho_0$, periodic solution appears, which confirms the occurrence of Hopf bifurcation at ρ_0.

The epidemic model without delay shows the backward bifurcation, which demonstrates that the disease-free equilibrium coexists with the endemic equilibrium when the basic reproduction number is less than one. It has important qualitative implications since reducing the basic reproduction number below one is not sufficient to eradicate the disease from society. Thus, it is vital to explain those components that can control or maintain a strategic distance from such backward situations. Our theoretical results suggest that the existence of backward bifurcation depends on the treatment of infected individuals. Under the point of view of the disease control campaigns, public policymakers might try to prevent the dangerous, backward scenario by keeping the treatments of infected individuals suitably low. In this case, the disease control may straightly follow the classic road of reducing R_0 below 1. If the backward scenario cannot be avoided, then decreasing contact rate with infected individuals through aimed public sensitization programs that affect individual behaviors can act appropriately to reduce the disease transmission. The nonlinearity in the epidemic model due to the latent period (time delay) can give rise to periodicity via a Hopf bifurcation, which shows the oscillatory behavior of the disease at the critical value of time delay.

The findings of the model consisting of Beddington-DeAngelis incidence rate with the latent period as time delay and Holling type II treatment rate are capable of demonstrating the substantial role of latent period, social awareness among susceptibles, the magnitude of interference among infectives, and limitation in available facilities. Furthermore, the results can understand the transmission dynamics of the outbreak of newly emerging diseases and hence further suggest the control strategies to prevent the spread of diseases at a large scale—for example, the largest ebola outbreak in 2014 in the parts of West Africa.

Chapter 4

Stability analysis of a logistic growth SIR epidemic model with two explicit time-delays, the nonlinear incidence and treatment rates

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In this chapter, a time-delayed SIR epidemic model with a logistic growth of susceptibles, Crowley-Martin type incidence, and Holling type III treatment rates is proposed and analyzed mathematically. We consider two explicit time-delays: one in the incidence rate of new infection to measuring the impact of the latent period, and another in the treatment rate of infectives to analyzing the effect of late treatment availability. The stability behavior of the model is analyzed for two equilibria: the disease-free equilibrium (DFE) and the endemic equilibrium (EE). We derive the threshold quantity, the basic reproduction number R_0, which determines the eradication or persistence of infectious diseases in the host population. Using the basic reproduction number, we show that the DFE is locally asymptotically stable when $R_0 < 1$, linearly neutrally stable when $R_0 = 1$, and unstable when $R_0 > 1$ for the time-delayed system. The system without a latent period reveals the forward bifurcation at $R_0 = 1$, which implies that keeping R_0 below unity can diminish the disease. Further, the stability behavior for the EE is investigated, demonstrating the occurrence of oscillatory and periodic solutions through Hopf bifurcation concerning every possible grouping of two time-delays as the bifurcation parameter. To conclude, the numerical simulations in support of the theoretical findings are carried

out.

4.1 Introduction

Infectious diseases are significant reasons for affliction and mortality in developing nations. Therefore, understanding the transmission attributes of irresistible ailments and the surveillance and continuation of control interventions are necessary to maintain infectious disease control achievements. In the beginning phase of an outbreak, it follows an exponential or generalized - exponential growth. However, it slows down and reaches its maximum, as the increasing number of cases reaches its inflection point, and the daily incidence curve reaches its most extreme. Thus, the growth pattern departs from the (sub-)exponential path and follows a logistic growth rate [140]. To capture this situation, some authors studied the effect of the logistic growth rate in their epidemic models [62, 85, 104, 130]. Wang et al. [85] assumed that the logistic growth governed the population growth in susceptible individuals and proposed the epidemic model with bilinear incidence rate, including the incubation period. They investigated the stability behavior and showed the existence of Hopf bifurcation in their model. Zhang et al. [62] also studied the logistic growth SIR epidemic model with the saturated incidence rate, including the incubation time and the inhibitory effects of infectives, and studied the dynamical behavior of their model. Xue et al. [104] extended the work of Zhang et al. [62] by incorporating the impact of the latent period in the saturated incidence rate and obtained sufficient conditions for the Hopf bifurcations. Further, Li et al. [130] considered the logistic growth SIR epidemic model with bilinear incidence and saturated treatment rates and studied the stability analysis and various bifurcations. They showed that to eradicate the disease, the government should raise the efficiency and enlarge treatment capacity.

An incidence rate is a valuable tool that determines the number of new infection cases per unit time. A significant assumption in disease transmission models is how individuals make contact with each other. In 1989, Crowley and Martin [19] introduced an incidence rate of the form $\frac{\beta SI}{(1+\alpha S)(1+\gamma I)}$, which includes the forms of other incidences also, for instance, bilinear incidence rate if $\alpha = 0, \gamma = 0$; saturated incidence rate with susceptibles if $\gamma = 0$; Holling-II incidence rate if $\alpha = 0$. This incidence rate has the critical property

of including preventive anti-epidemic measures adopted by susceptible and infected populations (such as disease surveillance and hygienic standards, social distancing, travel restrictions, quarantine, case isolation, etc.). It normalizes the influence of inhibition on infectives even in the high density of susceptible populations [125, 139]. Apart from the preventive measures, treatment of infectives is the most important medical intervention for reducing disease spread and deaths during an outbreak. The adequacy and effectiveness of the therapy may influence the recovery pace of infectives [108].

Although many authors have studied models with time delay or saturated treatment, to the best of our knowledge, there are not many models that incorporate time delays in both nonlinear incidence and saturated treatment functions. Therefore, in the present chapter, we study a susceptible-infected-recovered (SIR) epidemic model with two explicit time-delays, the logistic growth of susceptible individuals, Crowley-Martin incidence rate, and the Holling type III treatment rate. The logistic growth of susceptibles comprises the increasing growth of the epidemic initially, but a decreasing development at a later stage as the number of infections approaches its maximum. To measure the transmission of infection in susceptibles individuals with the condition of taking protective anti-epidemic measures by both susceptibles and infected individuals, and the time-lag between being exposed to a disease and having its symptoms, we have incorporated the Crowley-martin incidence rate with the latent period. To study the impact of treatment on infected individuals with limited medical resources and delay in providing the appropriate therapy to infectives, we have considered the Holling type III treatment rate with time delay. The two explicit time delays have a significant role in the present chapter. A delay in identifying the disease leads to a delay in applying the right protection measures, which allow the virus to spread unseen. If the infection remains undiagnosed after the recognition, this time lag enables the virus to reach capital cities, where the outbreak grows into large epidemics. Therefore, the latent period and the delay in providing medical services to infectives let the disease stay for an extended period and spread faster at a higher rate. By incorporating the above-enlightened facts, we formate and analyze a mathematical epidemic model. To explore the disease dynamics through equilibrium analysis, we investigate the stability behavior of the disease-free and endemic equilibria by deriving the basic reproduction number R_0. This threshold quantity determines the eradication or persistence of the disease infection in a host population. The results indicate that for $R_0 < 1$, there is a unique disease-free equilibrium, which is locally asymptotically stable; this equilibrium becomes unstable when $R_0 > 1$.

Meanwhile, if R_0 is greater than one, then there exists an endemic equilibrium. The stability switches may happen near this equilibrium for different ranges of explicit time delays, and the oscillatory and periodic solutions may appear via Hopf bifurcation. We discuss the significance of two explicit time delays and other parameters by simulating the proposed model numerically, revealing the disease's endemic behavior, and suggesting its control strategies.

4.2 Model description and basic properties

We assume that the total population $N(t)$ at time t is divided into three catogories: susceptible $S(t)$, infected $I(t)$ and $R(t)$, respectively. The description of the model's parameters is as follow: The logistic growth $rS(t)\left(1-\frac{S(t)}{K}\right)$ governs the susceptible population with a carrying capacity K and a specific growth rate r. The term $\frac{\beta S(t-\rho_1)I(t-\rho_1)}{(1+\alpha S(t-\rho_1))(1+\gamma I(t-\rho_1))}$ represents the Crowley-Martin incidence rate with latent period ρ_1, where β denotes the transmission rate of infection, and α and γ denote the saturating factors representing the inhibitory effects adopted by susceptibles and infectives, respectively. ϑ, d, and θ are the natural mortality, disease-induced mortality, and recovery rate of infectives, respectively. The term $\frac{\sigma I^2(t-\rho_2)}{1+\xi I^2(t-\rho_2)}$ represents the Holling type III treatment rate, where σ is the cure rate (or the maximum treatment rate), ξ is the saturating rate that measures the effect of limited availability of treatment resources, and ρ_2 measures the time delay in giving treatment to infectives. Due to epidemiological reasons, we assume that σ is non-negative, and all other parameters are positive.

Thus, the model under the assumptions and descriptions mentioned above follows the following system of delay differential equations:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= rS\left(1-\frac{S}{K}\right) - \frac{\beta S(t-\rho_1)I(t-\rho_1)}{(1+\alpha S(t-\rho_1))(1+\gamma I(t-\rho_1))},\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\beta S(t-\rho_1)I(t-\rho_1)}{(1+\alpha S(t-\rho_1))(1+\gamma I(t-\rho_1))} - (\vartheta+\theta+d)I - \frac{\sigma I^2(t-\rho_2)}{1+\xi I^2(t-\rho_2)},\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \theta I + \frac{\sigma I^2(t-\rho_2)}{1+\xi I^2(t-\rho_2)} - \vartheta R.
\end{aligned} \tag{4.1}$$

Let $h=\max\{\rho_1,\rho_2\}$. The initial conditions of the model (4.1) are given by

$$S(\Theta)=\Phi_1(\Theta),\ I(\Theta)=\Phi_2(\Theta),\ R(\Theta)=\Phi_3(\Theta),\ \text{for } \Theta\in[-h,0], \tag{4.2}$$

where, $\Phi_i\in C\left([-h,0],\mathbb{R}^3_+\right)$, $i=1,2,3$, the space of continuous functions from $[-h,0]$ to

$\mathbb{R}^3_+$, equipped with the sup-norm: $\|\Phi\| = \sup_{\Theta\in[-h,0]} \Phi(\Theta)$.

By the theory of functional differential equations [23], it follows that all the solutions of model (4.1) with initial condition (4.2) exist and are differentiable for all $t \geq 0$.

It is noticed that the variable $R(t)$ is not present in the first two equations of the model (4.1); therefore, without loss of generality, for the analysis purpose, we can omit the third equation and study the following reduced system:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= rS\left(1-\frac{S}{K}\right) - \frac{\beta S(t-\rho_1)I(t-\rho_1)}{(1+\alpha S(t-\rho_1))(1+\gamma I(t-\rho_1))}, \\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\beta S(t-\rho_1)I(t-\rho_1)}{(1+\alpha S(t-\rho_1))(1+\gamma I(t-\rho_1))} - (\vartheta+\theta+d)I - \frac{\sigma I^2(t-\rho_2)}{1+\xi I^2(t-\rho_2)}.
\end{aligned} \tag{4.3}$$

Lemma 4.2.1. *Let* $\nu_m = \min\{1, \vartheta+\theta+d\}$ *and* $\Omega = \max\{S(0), K\}$. *Then the compact set*

$$D = \left\{(S,I) \in \mathbb{R}^2_+ : S \leq \Omega,\ S+I \leq \frac{(r+1)\Omega}{\nu_m}\right\}$$

is a positively invariant set for the system (4.3).

Proof. From the first equation of the system (4.3), we have

$$\frac{\mathrm{d}S}{\mathrm{d}t} \leq rS(1-\frac{S}{K}).$$

Thus, a standard comparison argument gives that

$$\limsup_{t\to\infty} S(t) \leq \Omega.$$

On adding first and second equations of the system (4.1), we obtain

$$\begin{aligned}
\frac{\mathrm{d}}{\mathrm{d}t}(S+I) &= rS\left(1-\frac{S}{K}\right) - (\vartheta+\theta+d)I - \frac{\sigma I^2(t-\rho_2)}{1+\xi I^2(t-\rho_2)} \\
&\leq (r+1)S - S - (\vartheta+\theta+d)I \\
&\leq (r+1)\Omega - \nu_m(S+I).
\end{aligned}$$

Thus, we get

$$0 \leq (S+I) \leq \frac{(r+1)\Omega}{\nu_m} \text{ for sufficiently large } t.$$

Hence, all the solutions of the system (4.3) are closed and bounded, and enter the region

$$D = \left\{ (S,I) \in \mathbb{R}^2_+ : S \leq \Omega,\ S+I \leq \frac{(r+1)\Omega}{\nu_m} \right\}.$$

Hence, the system (4.3) is well-posed mathematically and epidemiologically. ■

4.3 Mathematical analysis of the model

In this section, the mathematical analysis of the model is established with the following steps: the existence of equilibria, the derivation of basic reproduction number, stability behavior of obtained equilibria, and the presence of Hopf bifurcation near-endemic equilibrium regarding the possible combinations of two time-delays.

4.3.1 Existence of model's equilibria

On substituting the right-hand side of the system (4.3) to zero, we get two equilibrium points, namely:
(i) The disease-free equilibrium (DFE) $E_0(S_0,I_0) = (K,0)$,
(ii) The endemic (positive) equilibrium (EE) $E_e(S^*,I^*)$, where S^* and I^* are evaluated in Subsection 4.3.3.

4.3.2 Disease-free equilibrium and its stability analysis

The disease-free equilibrium (DFE) is defined as the point at which no disease is present. The DFE of the system (4.3) lies at $E_0(S_0,I_0) = (K,0)$.

The Jacobian matrix J of the system (4.3) at DFE E_0 is given as

$$J(E_0) = \begin{bmatrix} -r & -\frac{\beta K e^{-\lambda\rho_1}}{1+\alpha K} \\ 0 & \frac{\beta K e^{-\lambda\rho_1}}{1+\alpha K} - (\vartheta + d + \theta) \end{bmatrix}. \tag{4.4}$$

The characteristic equation associated with $J(E_0)$ is

$$(\lambda + r)\left(\lambda - \frac{\beta K e^{-\lambda\rho_1}}{1+\alpha K} + \vartheta + d + \theta\right) = 0. \tag{4.5}$$

The roots of Eq. (4.5) are $\lambda_1 = -r$ and λ_2, where λ_2 is a solution of the following transcendental equation:

$$T(\lambda) := \lambda - \frac{\beta K e^{-\lambda \rho_1}}{1+\alpha K} + \vartheta + d + \theta = 0. \tag{4.6}$$

The term $\frac{\beta K e^{-\lambda \rho_1}}{(1+\alpha K)(\vartheta+d+\theta)}$ at $\rho_1 = 0$ is termed as the basic reproduction number (R_0) of the system (4.3). Thus, R_0 of the system (4.3) is

$$R_0 = \frac{\beta K}{(1+\alpha K)(\vartheta+d+\theta)}.$$

Now, we state and prove the following results using basic reproduction number R_0 for the local stability behavior of disease-free equilibrium $E_0(S_0, I_0)$.

Theorem 4.3.1. *The disease-free equilibrium $E_0(S_0, I_0)$ of the system (4.3) has the following properties:*

1. *When $R_0 > 1$, then $E_0(S_0, I_0)$ is unstable for $\rho_1 > 0$.*
2. *When $R_0 < 1$, then $E_0(S_0, I_0)$ is locally asymptotically stable for $\rho_1 > 0$.*
3. *When $R_0 = 1$, then $E_0(S_0, I_0)$ is linearly neutrally stable for $\rho_1 > 0$.*
4. *When $R_0 = 1$ and $\rho_1 = 0$, then the system (4.3) exhibits a forward bifurcation at $E_0(S_0, I_0)$.*

Proof. We have

$$T(\lambda) := \lambda - \frac{\beta K e^{-\lambda \rho_1}}{1+\alpha K} + \vartheta + d + \theta = 0.$$

The stability of the disease-free equilibrium E_0 is investigated for a different ranges of R_0 under the following cases:

1. Assume that $R_0 > 1$. Then, we have

$$\begin{aligned} T(0) &= -\frac{\beta K}{1+\alpha K} + \vartheta + d + \theta \\ &= (\vartheta + d + \theta)(1 - R_0) \\ &< 0. \end{aligned}$$

Also, $\lim_{\lambda \to \infty} T(\lambda) = +\infty$.

Note that $T(\lambda) = 0$, $T(0) < 0$, and $\lim_{\lambda \to \infty} T(\lambda) = +\infty$ imply that $T(\lambda)$ is a increasing

function for real λ. Therefore, $T(\lambda) = 0$ has a real positive root when $R_0 > 1$ and hence, E_0 is unstable.

2. Assume that $R_0 < 1$. On the contrary, let $T(\lambda)$ has a root $\lambda_* \in \mathrm{C}$ with $Re(\lambda_*) \geq 0$. Then, we have $\lambda_* = \frac{\beta K \mathrm{e}^{-\lambda_* \rho_1}}{1+\alpha K} - (\vartheta + d + \theta)$.
So, $Re(\lambda_*) \leq \frac{\beta K}{1+\alpha K} - (\vartheta + d + \theta) = (\vartheta + d + \theta)(R_0 - 1) < 0$, which is a contradiction to our assumption. Thus, all the roots of $T(\lambda)$ has a negative real part, which proves that E_0 is locally asymptotically stable.

3. Let $R_0 = 1$. When $R_0 = 1$, then we have $\beta K = (1 + \alpha K)(\vartheta + d + \theta)$ and one root of $T(\lambda) = 0$ vanishes.
Let $\lambda = m + \mathrm{i}n$ be any other solution of $T(\lambda) = 0$, Then, we have

$$m + \mathrm{i}n - \frac{\beta K \mathrm{e}^{-(m+\mathrm{i}n)\rho_1}}{1 + \alpha K} + \vartheta + d + \theta = 0. \tag{4.7}$$

Splitting real and imaginary parts of Eq. (4.7) and using $R_0 = 1$, we obtain:

$$(\vartheta + d + \theta)\mathrm{e}^{-m\rho_1} \cos n\rho_1 = m + \vartheta + d + \theta, \tag{4.8}$$

$$(\vartheta + d + \theta)\mathrm{e}^{-m\rho_1} \sin n\rho_1 = -n. \tag{4.9}$$

Squaring Eqs. (4.8) and (4.9) and then summing up the resultant yields:

$$(\vartheta + d + \theta)^2 \mathrm{e}^{-2m\rho_1} = (m + \vartheta + d + \theta)^2 + n^2. \tag{4.10}$$

If there exists a root satisfying the Eqs. (4.8) and (4.9), then that root will satisfy Eq. (4.10) too. For Eq. (4.10) to be verified, we must have $m \leq 0$. Therefore, E_0 is linearly neutrally stable.

4. In case when $R_0 = 1$ and $\rho_1 = 0$, then one of the eigenvalue of Jacobian matrix $J(E_0)$, given in Eq. (4.4), vanishes. So, the linearization method is not applicable, and we can resort to center manifold theory [17].
If we consider β as the bifurcation parameter, then the critical value β^* can be calculated as $\beta^* = \frac{(1+\alpha K)(\vartheta+d+\theta)}{K}$. We see that $J(E_0)$ has a simple zero eigenvalue $\lambda = 0$.

The components of right eigenvector $V = (V_1, V_2)^T$ and the left eigenvector of $U =$

(U_1, U_2) of $J(E_0)$ corresponding to $\lambda = 0$ are

$$V_1 = -\frac{\beta k}{r(\alpha k+1)},\ V_2 = 1;\ U_1 = 0,\ U_2 = 1.$$

Let $g = (g_1, g_2)$ represents the right hand side of the system (4.3). Using Theorem 4.1 of [40], we calculate the bifurcation constants a_1 and b_1 as follows:

$$a_1 = \sum_{k,i,j=1}^{2} U_k V_i V_j \left(\frac{\partial^2 g_k}{\partial x_i \partial x_j}\right)_{E_0},$$
$$b_1 = \sum_{k,i=1}^{2} U_k V_i \left(\frac{\partial^2 g_k}{\partial x_i \partial \beta^*}\right)_{E_0}$$

where, x_1 and x_2 represent the susceptible individuals S and the infected individuals I.

The non-zero partial derivatives associated with a_1 and b_1 are:

$$\frac{\partial^2 g_2(E_0,\beta^*)}{\partial S \partial I} = \frac{\partial^2 g_2(E_0,\beta^*)}{\partial I \partial S} = \frac{\beta^*}{(\alpha K+1)^2},\ \frac{\partial^2 g_2(E_0,\beta^*)}{\partial I^2} = -\frac{2\beta^*\gamma K}{\alpha K+1} - 2\sigma, \text{ and}$$

$$\frac{\partial^2 g_2(E_0,\beta^*)}{\partial I \partial \beta^*} = \frac{K}{\alpha K+1}.$$

So, a_1 and b_1 take the form

$$\begin{aligned} a_1 &= V_1 V_2 \left(\frac{\partial^2 g_2(E_0,\beta^*)}{\partial S \partial I}\right) + V_2 V_1 \left(\frac{\partial^2 g_2(E_0,\beta^*)}{\partial I \partial S}\right) + V_2^2 \left(\frac{\partial^2 g_2(E_0,\beta^*)}{\partial I^2}\right) \\ &= -\frac{2\beta^* k\left(\gamma r(\alpha k+1)^2 + \beta^*\right)}{r(\alpha k+1)^3} - 2\sigma, \\ b_1 &= U_2 V_2 \left(\frac{\partial^2 g_2(E_0,\beta^*)}{\partial I \partial \beta^*}\right), \\ &= \frac{K}{\alpha K+1}. \end{aligned}$$

It can be seen that $a_1 < 0$ and $b_1 > 0$. Thus, using Theorem 4.1(iv) of [40], there exists a forward bifurcation at $R_0 = 1$. Forward bifurcation means that if some infectives are introduced into the population then the system will return to the disease-free equilibrium $I = 0$. That is, disease will die out if $R_0 < 1$.

■

The graphical representation of the forward bifurcation is illustrated in the Example 4.1.

Example 4.1. Forward Bifurcation: We consider the following set of parameters: $r = 0.08$, $K = 100$, $\alpha = 0.002$, $\gamma = 0.001$, $\vartheta = 0.01$, $d = 0.008$, $\theta = 0.02$, $\sigma = 0.004$, $\xi = 0.009$. At these parameters' values, the bifurcation parameter is obtained as $\beta = \beta^* = 0.000456$. Fig. (4.1) shows that when $R_0 < 1$ then, there is a stable disease free equilbrium, and when $R_0 > 1$, then the disease-free equilibrium becomes unstable and there exists a stable endemic equilibrium. It guarantees the eradication of disease when $R_0 < 1$, whereas the disease will invade when $R_0 > 1$.

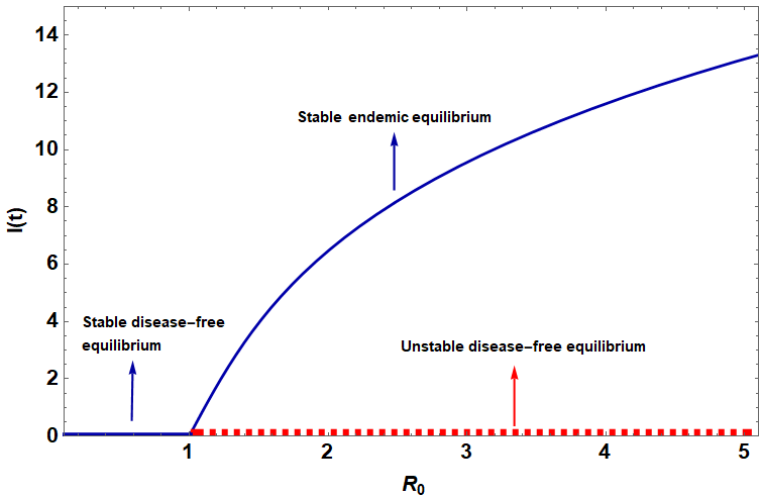


Figure 4.1: Forward bifurcation.

4.3.3 Endemic equilibrium and stability analysis

To establish the existence of endemic equilibrium $E_e(S^*.I^*)$, first, we put the right-hand side term of the second equation of the system (4.3) to zero. We obtain:

$$S^* = \frac{(\gamma I^* + 1)\left(\left(\xi I^{*2} + 1\right)(d + \theta + \vartheta) + \sigma I^*\right)}{\beta\left(\xi I^{*2} + 1\right) - (\gamma I^* + 1)\left(\alpha\left(\xi I^{*2} + 1\right)(d + \theta + \vartheta) + \alpha\sigma I^*\right)}. \tag{4.11}$$

$S^* > 0$ if

$$\beta\left(\xi I^{*2} + 1\right) > (\gamma I^* + 1)\left(\alpha\left(\xi I^{*2} + 1\right)(d + \theta + \vartheta) + \alpha\sigma I^*\right).$$

On setting the right-hand side term of the first equation of the system (4.3) to zero and putting the value of S^* using Eq. (4.11), we obtain that I^* is a solution of the following seventh-degree equation:

$$P(I^*) := A_0 + A_1 I^* + A_2 I^{*2} + A_3 I^{*3} + A_4 I^{*4} + A_5 I^{*5} + A_6 I^{*6} + A_7 I^{*7} = 0, \tag{4.12}$$

where, the coefficients $A_i's$, $i = 0,1,2,\ldots,7$ are given below:

$$
\begin{aligned}
A_0 &= r(\alpha k+1)(d+\theta+\vartheta)(1-R_0),\\
A_1 &= \alpha^2 d^2 k + 2d(\alpha k(\alpha(\theta+\vartheta)-\beta+\gamma r)+\gamma r)+k(\beta^2-\beta(2\alpha(\theta+\vartheta)+\gamma r)+\\
&\quad \alpha(\theta+\vartheta)(\alpha(\theta+\vartheta)+2\gamma r)+\alpha r\sigma)+r(2\gamma(\theta+\vartheta)+\sigma),
\end{aligned}
$$

$$
\begin{aligned}
A_3 &= \alpha^2 d^2 k\left(\gamma^2+2\xi\right)+2d\left(\alpha k\left(\alpha\left(\gamma^2+2\xi\right)(\theta+\vartheta)+2\alpha\gamma\sigma-2\beta\xi+2\gamma\xi r\right)+2\gamma\xi r\right)\\
&\quad +k(\alpha^2\left(\left(\gamma^2+2\xi\right)(\theta+\vartheta)^2+4\gamma\sigma(\theta+\vartheta)+\sigma^2\right)+\alpha\sigma\left(r\left(\gamma^2+\xi\right)-2\beta\gamma\right)\\
&\quad -4\alpha\xi(\theta+\vartheta)(\beta-\gamma r)+2\beta\xi(\beta-\gamma r))+r\sigma\left(\gamma^2+\xi\right)+4\gamma\xi r(\theta+\vartheta),\\
A_4 &= 4\alpha^2\gamma d^2 k\xi+2\alpha^2 dk\sigma\left(\gamma^2+\xi\right)+d\xi(2\gamma(\alpha k(4\alpha(\theta+\vartheta)-2\beta+\gamma r)+\gamma r)+\xi r(\alpha k+1))\\
&\quad +2\alpha^2\gamma k\sigma^2+2\sigma\left(\alpha k\left(\alpha\left(\gamma^2+\xi\right)(\theta+\vartheta)-\beta\xi+\gamma\xi r\right)+\gamma\xi r\right)+\xi(2\gamma(\theta+\vartheta)\\
&\quad \times(\alpha k(2\alpha(\theta+\vartheta)-2\beta+\gamma r)+\gamma r)+\xi r(\theta+k(\alpha(\theta+\vartheta)-\beta)+\vartheta)),\\
A_5 &= \alpha^2 d^2 k\xi\left(2\gamma^2+\xi\right)+2d\xi\left(\alpha k\left(\alpha\left(2\gamma^2+\xi\right)(\theta+\vartheta)+2\alpha\gamma\sigma-\beta\xi+\gamma\xi r\right)+\gamma\xi r\right)\\
&\quad +k(\alpha^2\left(\xi\left(2\gamma^2+\xi\right)(\theta+\vartheta)^2+\gamma^2\sigma^2+4\gamma\xi\sigma(\theta+\vartheta)\right)+\alpha\xi(\gamma\sigma(\gamma r-2\beta)\\
&\quad -2\xi(\theta+\vartheta)(\beta-\gamma r))+\beta\xi^2(\beta-\gamma r))+\gamma\xi r(\gamma\sigma+2\xi(\theta+\vartheta)),\\
A_6 &= \gamma\xi(d+\theta+\vartheta)\left(2\alpha^2 dk\xi+\alpha k(2\alpha(\gamma\sigma+\xi(\theta+\vartheta))-2\beta\xi+\gamma\xi r)+\gamma\xi r\right),\\
A_7 &= \alpha^2\gamma^2 k\xi^2(d+\theta+\vartheta)^2.
\end{aligned}
$$

On analyzing Eq. (4.12), we propose the following theorem:

Theorem 4.3.2. *When $R_0 > 1$, then there exists either a unique, three, five, or seven solutions of the polynomial $P(I^*)$.*

Proof. Let $R_0 > 1$. Note that $P(0) = A_0 < 0$ when $R_0 > 1$ and the coefficient $A_7 = \alpha^2\gamma^2 k\xi^2(d+\theta+\vartheta)^2$ is always positive.
Thus, we have

$$\lim_{I^*\to\infty} P(I^*) = +\infty.$$

The polynomial $P(I^*)$ is a seventh-degree polynomial in I^*, and it is a continuous function of I^*. Thus, by the fundamental theorem of algebra, $P(I^*)$ can have at most seven roots. ■

In the present study, we analyze the system for the existence of a unique endemic equilibrium only. Using Descartes' rules of signs [42], the polynomial $P(I^*)$ can have a

unique root when $R_0 > 1$, if any of the following conditions is satisfied:

$$\begin{aligned}
A_i > 0 \ (i = 1 \text{ to } 6),\\
A_1 < 0, \ \ A_i > 0 \ (i = 2 \text{ to } 6),\\
A_1 < 0, \ A_2 < 0, \ A_i > 0 \ (i = 3 \text{ to } 6),\\
A_i < 0 \ (i = 1 \text{ to } 3), \ A_j > 0 \ (j = 4 \text{ to } 6),\\
A_i < 0 \ (i = 1 \text{ to } 4), \ A_j > 0 \ (j = 5 \text{ to } 6),\\
A_i < 0 \ (i = 1 \text{ to } 5), \ A_6 > 0.
\end{aligned} \tag{4.13}$$

After obtaining the value of I^*, the value of S^* can be obtained from Eq. (4.11). Thus, there exists a unique endemic equilibrium $E_e(S^*, I^*)$ if one of the conditions (4.13) holds.

To determine the stability behavior of the endemic equilibrium $E_e(S^*, I^*)$, the characteristic equation of the system (4.3) at E_e is obtained as:

$$\lambda^2 + m_1\lambda + m_2 + (n_1\lambda + n_2)\, e^{-\lambda\rho_1} + (l_1\lambda + l_2)\, e^{-\lambda\rho_2} + o_1 e^{-\lambda(\rho_1+\rho_2)} = 0, \tag{4.14}$$

where,

$$\begin{aligned}
m_1 &= d + \theta + r\left(\frac{2S^*}{K} - 1\right) + \vartheta,\\
m_2 &= -\frac{r(K - 2S^*)(d + \theta + \vartheta)}{K},\\
n_1 &= \frac{\beta(I^*(\gamma I^* + 1) - S^*(\alpha S^* + 1))}{(\alpha S^* + 1)^2(\gamma I^* + 1)^2},\\
n_2 &= \frac{\beta\left(K(I^*(\gamma I^* + 1)(d + \theta + \vartheta) + rS^*(\alpha S + 1)) - 2rS^{*2}(\alpha S^* + 1)\right)}{K(\alpha S^* + 1)^2(\gamma I^* + 1)^2},\\
l_1 &= \frac{2\sigma I^*}{(\xi I^{*2} + 1)^2},\\
l_2 &= -\frac{2r\sigma I^*(K - 2S^*)}{K(\xi I^{*2} + 1)^2},\\
o_1 &= \frac{2\beta\sigma I^{*2}}{(\alpha S^* + 1)^2(\gamma I^* + 1)(\xi I^{*2} + 1)^2}.
\end{aligned} \tag{4.15}$$

Now, we investigate the stability of E_e for the following possible cases of ρ_1 and ρ_2:

Case (i) $\rho_1 = \rho_2 = 0$.

In this case, the characteristic equation (4.14) becomes:

$$\lambda^2 + m_{01}\lambda + m_{11} = 0, \tag{4.16}$$

where,

$$\begin{aligned} m_{01} &= (m_1 + n_1 + l_1), \\ m_{11} &= (m_2 + n_2 + l_2 + o_1). \end{aligned} \tag{4.17}$$

Using Routh-Hurwitz criteria, it follows that the roots of Eq. (4.16) must have negative real parts if the condition (H1:) $m_{01} > 0$ and $m_{11} > 0$. Thus, we state the following theorem:

Theorem 4.3.3. *The endemic equilibrium $E_e(S^*, I^*)$ is locally asymptotically stable for $\rho_1 = \rho_2 = 0$, if the condition (**H1**) holds.*

Case (ii) $\rho_1 > 0,\ \rho_2 = 0$.

The characteristic equation (4.14) at E_e when $\rho_1 > 0,\ \rho_2 = 0$ becomes

$$\lambda^2 + l_{01}\lambda + l_{11} + (n_1\lambda + n_2 + o_1)e^{-\lambda\rho_1} = 0, \tag{4.18}$$

where,

$$\begin{aligned} l_{01} &= (m_1 + l_1), \\ l_{11} &= (m_2 + l_2). \end{aligned} \tag{4.19}$$

where, $m_1, m_2, n_1, n_2, l_1, l_2$ and o_1 are given in Eq. (4.15).

From Theorem 4.3.3, we know that all the solutions of Eq. (4.14) have negative real parts when $\rho_1 = \rho_2 = 0$ if (H1) holds. If for a particular value of $\rho_1 > 0$ and fixed $\rho_2 = 0$, a characteristic root of Eq. (4.18) must pass through the imaginary axis if it lies on the right half-plane. Hence, on the contrary, suppose that $\lambda = i\kappa$ $(\kappa > 0)$ is a root of the Eq. (4.18).

Substituting $\lambda = i\kappa$ into Eq. (4.18) and separating the real and imaginary parts, we

obtain:

$$\kappa^2 - l_{11} = n_1 \kappa \sin \kappa\rho_1 + (n_2 + o_1)\cos \kappa\rho_1, \tag{4.20}$$

$$-l_{01}\kappa = n_1 \kappa \cos \kappa\rho_1 - (n_2 + o_1)\sin \kappa\rho_1. \tag{4.21}$$

On squaring and adding Eqs. (4.20) and (4.21), we get

$$\kappa^4 + A_{11}\kappa^2 + A_{12} = 0, \tag{4.22}$$

where,

$$A_{11} = -2l_{11} - n_1^2 + l_{01}^2,$$
$$A_{12} = l_{11}^2 - (n_2 + o_1)^2.$$

Let $\kappa^2 = \zeta$, then Eq. (4.22) becomes

$$H(\zeta) := \zeta^2 + A_{11}\zeta + A_{12} = 0. \tag{4.23}$$

Let $\mathbf{H2_a} : A_{11} > 0$, and $A_{12} > 0$. Using Routh Hurwitz criterion, we have that Eq. (4.23) cannot have a positive root, which is a contraction to the fact that $\zeta = \kappa^2$ is such a root. Thus, we state the following theorem:

Theorem 4.3.4. *The endemic equilibrium $E_e(S^*, I^*)$ is locally asymptotically stable for $\rho_1 > 0$ and $\rho_2 = 0$, if $\mathbf{H2_a}$ holds.*

From Theorem 4.3.4, it is inferred that there exists a purely imaginary root $\mathbf{i}\kappa$ of the characteristic equation at E_e if and only if Eq. (4.18) has a positive real root ζ. On analyzing the quadratic polynomial $H(\zeta)$ graphically, we see that Eq. (4.23) has at least one positive root if any of the following conditions hold:

$\mathbf{H2_b}$. $A_{12} = l_{11}^2 - (n_2 + o_1)^2 < 0$,

$\mathbf{H2_c}$. $A_{11} < 0$, and $A_{11}^2 - 4A_{12} > 0$.

Without loss of generality, we assume that Eq. (4.23) has two positive roots ζ_1 and ζ_2, and let $\kappa_i = \sqrt{\zeta_i}$, $i = 1, 2$.

From Eqs. (4.20) and (4.21), ρ_{1_j} corresponding to κ_i can be obtained as

$$\rho_{1_i}^{(j)} = \frac{1}{\kappa_i}\arccos\left(\frac{\kappa_i^2((n_2+o_1)-l_{01}n_1)-l_{11}(n_2+o_1)}{n_1^2\kappa_i^2+(n_2+o_1)^2}\right)+\frac{2j\pi}{\kappa_i}, \quad i=1,2, \quad j=0,1,2,\ldots. \tag{4.24}$$

Let $\rho_1^* = \rho_{1_{i_0}}^{(j_0)} = \min\left\{\rho_{1_i}^{(j)} : i=1,2, \quad j=0,1,2,\ldots.\right\}$.

Lemma 4.3.5. *Suppose that **H1** holds.*

1. *If either* $\mathbf{H2_b}$ *or* $\mathbf{H2_c}$ *satisfies, then Eq. (4.18) has all the roots with negative real parts for* $\rho_1 \in [0,\rho_1^*)$.
2. *If neither* $\mathbf{H2_b}$ *nor* $\mathbf{H2_c}$ *satisfies, then Eq. (4.18) has all the roots with negative real parts for* $\rho_1 \geq 0$.

let $\Gamma(\kappa) = \gamma(\rho_1)+\mathrm{i}\kappa(\rho_1)$ be the root of Eq. (4.18) such that $\gamma(\rho_1^*)=0$ and $\kappa(\rho_1^*) = \kappa_{i_0} := \kappa_0$.

To estabilish the Hopf bifurcation, we show the transversality condition $\left[\frac{\mathrm{d}(Re\lambda)}{\mathrm{d}\rho_1}\right]\Big|_{\lambda=\mathrm{i}\kappa_0} \neq 0$.

Lemma 4.3.6. *Suppose that* $\mathbf{H2_d} : H'(\kappa_0^2) \neq 0$,

then the transversality condition $\left[\frac{\mathrm{d}(Re\lambda)}{\mathrm{d}\rho_1}\right]\Big|_{\lambda=\mathrm{i}\kappa_0} \neq 0$ *holds.*

Proof. On differentiating Eq. (4.18) with respect to $\lambda(\rho_1)$ and then simplifying, we obtain

$$\left[\frac{\mathrm{d}\lambda}{\mathrm{d}\rho_1}\right]^{-1} = \frac{2\lambda+l_{01}}{-\lambda(\lambda^2+l_{01}\lambda+l_{11})}+\frac{n_1}{\lambda(n_1\lambda+(n_2+o_1))}-\frac{\rho_1}{\lambda}.$$

$$\begin{aligned}
\mathrm{Re}\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\rho_1}\right)^{-1}\Big|_{\lambda=\mathrm{i}\kappa_0} &= \mathrm{Re}\left(\frac{2\mathrm{i}\kappa_0+l_{01}}{-\mathrm{i}\kappa_0(-\kappa_0^2+\mathrm{i}l_{01}\kappa_0+l_{11})}+\frac{n_1}{-n_1{\kappa_0}^2+\mathrm{i}(n_2+o_1)\kappa_0}+\frac{\mathrm{i}\rho_1}{\kappa_0}\right)\\
&= \frac{1}{\kappa_0}\left(\frac{2\kappa_0(\kappa_0^2-l_{11})+l_{01}^2\kappa_0}{(l_{01}\kappa_0)^2+(\kappa_0^2-l_{11})^2}-\frac{n_1^2\kappa_0}{(n_1\kappa_0)^2+(n_2+o_1)^2}\right)\\
&= \frac{2(\kappa_0^2-l_{11})+l_{01}^2}{(l_{01}\kappa_0)^2+(\kappa_0^2-l_{11})^2}-\frac{n_1^2}{(n_1\kappa_0)^2+(n_2+o_1)^2}.
\end{aligned}$$

From Eqs. (4.20), and (4.21), we get

$$(n_1\kappa_0)^2+(n_2+o_1)^2 = (l_{01}\kappa_0)^2+(\kappa_0^2-l_{11})^2. \tag{4.25}$$

Thus, we obtain

$$\begin{aligned}
\operatorname{sign}\left\{\frac{\mathrm{d}}{\mathrm{d}\rho_1}(\mathrm{Re}\,\lambda)\Big|_{\lambda=\mathrm{i}\kappa_0}\right\} &= \operatorname{sign}\left\{\mathrm{Re}\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\rho_1}\right)^{-1}\Big|_{\lambda=\mathrm{i}\kappa_0}\right\} \\
&= \operatorname{sign}\left\{\frac{2\kappa_0^2+(l_{01}^2-2l_{11}-n_1^2)}{n_1^2\kappa_0^2+(n_2+o_1)^2}\right\} \\
&= \operatorname{sign}\left\{\frac{H'(\kappa_0^2)}{n_1^2\kappa_0^2+(n_2+o_1)^2}\right\} \\
&\neq 0.
\end{aligned} \tag{4.26}$$

Thus, it follows that $\left[\frac{\mathrm{d}(Re\lambda)}{\mathrm{d}\rho_1}\right]\Big|_{\lambda=\mathrm{i}\kappa_0} \neq 0$ and hence the proof. ■

From the above analysis, we have the following theorem:

Theorem 4.3.7. *Suppose that* **H1** *holds.*

1. *If neither* $\mathbf{H2_b}$ *nor* $\mathbf{H2_c}$ *holds, then* E_e *is locally asymptotically stable for all* $\rho_1 \geq 0$.
2. *If either* $\mathbf{H2_b}$ *or* $\mathbf{H2_c}$ *holds, then* E_e *is locally asymptotically stable for* $\rho_1 \in [0, \rho_1^*)$.
3. *If either* $\mathbf{H2_b}$ *or* $\mathbf{H2_c}$ *holds, and* $H'(\kappa_0^2) \neq 0$, *then the system (4.3) undergoes a Hopf bifurcation at* E_e *when* $\rho_1 = \rho_1^*$, *and a family of periodic solutions bifurcate from* E_e *when* ρ_1 *crosses* ρ_1^*.

Case (iii) $\rho_1 = 0,\ \rho_2 > 0$.

The characteristic equation (4.14) at E_e when $\rho_1 = 0,\ \rho_2 > 0$ becomes

$$\lambda^2 + m_{01}\lambda + m_{02} + (m_{11}\lambda + m_{12})\,e^{-\lambda\rho_2} = 0, \tag{4.27}$$

where,

$$\begin{aligned}
m_{01} &= m_1 + n_1, \\
m_{02} &= m_2 + n_2, \\
m_{11} &= l_1, \\
m_{12} &= l_2 + o_1.
\end{aligned} \tag{4.28}$$

Let $\lambda = \mathrm{i}\kappa$ be root of Eq. (4.27). On putting $\lambda = \mathrm{i}\kappa$ in Eq. (4.27) and simplifying, we

obtain

$$\kappa^2 - m_{02} = m_{12}\cos\kappa\rho_2 + m_{11}\kappa\sin\kappa\rho_2, \tag{4.29}$$

$$m_{01}\kappa = m_{12}\sin\kappa\rho_2 - m_{11}\kappa\cos\kappa\rho_2. \tag{4.30}$$

From Eqs. (4.29) and (4.30), we obtain

$$\kappa^4 + B_{11}\kappa^2 + B_{12} = 0, \tag{4.31}$$

where,

$$\begin{aligned} B_{11} &= -2m_{02} + m_{01}^2 - m_{11}^2, \\ B_{12} &= m_{02}^2 - m_{12}^2. \end{aligned} \tag{4.32}$$

Let $\kappa^2 = \eta$. Then, Eq. (4.31) becomes

$$G(\eta) := \eta^2 + B_{11}\eta + B_{12} = 0. \tag{4.33}$$

In the same way, as explained in Case (ii), Eq. (4.33) has atleast one postive root when either of the following conditions holds:

H3$_\mathbf{a}$. $B_{12} < 0$,

H3$_\mathbf{b}$. $B_{11} < 0$, and $B_{11}^2 - 4B_{12} > 0$.

Without loss of generality, we assume that Eq. (4.32) has two positive roots η_1 and η_2, and let $\kappa_i = \sqrt{\eta_i}$, $i = 1,2$.
From Eqs. (4.29) and (4.30), ρ_{2_j} corresponding to κ_i can be obtained as

$$\rho_{2_i}^{(j)} = \frac{1}{\kappa_i}\arccos\left(\frac{\kappa_i^2(m_{12} - m_{01}m_{11}) - m_{02}m_{12}}{m_{11}^2\kappa_i^2 + m_{12}^2}\right) + \frac{2j\pi}{\kappa_i}, \quad i = 1,2, \quad j = 0,1,2,\ldots. \tag{4.34}$$

Let $\rho_2^* = \rho_{2_{i_0}}^{(j_0)} = \min\left\{\rho_{2_i}^{(j)} : i = 1,2, \quad j = 0,1,2,\ldots.\right\}$.

As shown in Case(ii), **H3$_\mathbf{c}$** : $G'(\kappa_0^2) \neq 0$, then the transversality condition $\left[\frac{\mathrm{d}(Re\lambda)}{\mathrm{d}\rho_2}\right]\Big|_{\lambda=\mathrm{i}\kappa_0} \neq 0$ holds, where $\kappa_0 = \kappa(\rho_2^*)$. Thus, the following theorem is stated:

Theorem 4.3.8. *Suppose that* **H1** *holds.*

1. *If neither* **H3$_\mathbf{a}$** *nor* **H3$_\mathbf{b}$** *satisfies, then E_e is locally asymptotically stable for all $\rho_2 \geq 0$.*

2. *If either* **H3$_a$** *or* **H3$_b$** *satisfies, then E_e is locally asymptotically stable for $\rho_2 \in [0, \rho_2^*)$.*

3. *If either* **H3$_a$** *or* **H3$_b$** *satisfies, and $G'(\kappa_0^2) \neq 0$, then the system (4.3) undergoes a Hopf bifurcation at E_e when $\rho_2 = \rho_2^*$ and a family of periodic solutions bifurcate from E_e when ρ_2 crosses ρ_2^*.*

Case (iv) $\rho_1 = \rho_2 = \rho$.

In this case, the characteristic equation (4.14) becomes

$$\lambda^2 + m_1\lambda + m_2 + (q_1\lambda + q_2)\,\mathrm{e}^{-\lambda\rho} + o_1\mathrm{e}^{-2\lambda\rho} = 0, \tag{4.35}$$

where,

$$n_1 + l_1 = q_1, \quad \text{and} \quad n_2 + l_2 = q_2.$$

On multiplying Eq. (4.35) by $\mathrm{e}^{\lambda\rho}$, we get

$$\mathrm{e}^{\lambda\rho}\left(\lambda^2 + m_1\lambda + m_2\right) + (q_1\lambda + q_2) + o_1\mathrm{e}^{-\lambda\rho} = 0. \tag{4.36}$$

Let $\lambda = \mathrm{i}\kappa$ be root of Eq. (4.36). Then, Eq. (4.36) becomes:

$$\left(-\kappa^2 + m_2 + o_1\right)\cos\kappa\rho - m_1\kappa\sin\kappa\rho + q_2 + \mathrm{i}\left(\left(-\kappa^2 + m_2 - o_1\right)\sin\kappa\rho + m_1\kappa\cos\kappa\rho + q_1\kappa\right) = 0. \tag{4.37}$$

On separating real and imaginary parts of Eq. (4.37), we obtain

$$J_1(\kappa)\cos\kappa\rho - J_2(\kappa)\sin\kappa\rho = J_3(\kappa), \tag{4.38}$$

$$J_4(\kappa)\sin\kappa\rho + J_5(\kappa)\cos\kappa\rho = J_6(\kappa), \tag{4.39}$$

where,

$$J_1(\kappa) = -\kappa^2 + m_2 + o_1, \quad J_2(\kappa) = m_1\kappa, \quad J_3(\kappa) = -q_2,$$
$$J_4(\kappa) = -\kappa^2 + m_2 - o_1, \quad J_5(\kappa) = m_1\kappa, \quad J_6(\kappa) = -q_1\kappa.$$

From Eqs. (4.38), and (4.39), we obtain

$$\cos\kappa\rho = \frac{J_{01(\kappa)}}{J_{00}(\kappa)}, \tag{4.40}$$

$$\sin\kappa\rho = \frac{J_{02(\kappa)}}{J_{00}(\kappa)}, \tag{4.41}$$

where,

$$J_{01}(\kappa) = J_3(\kappa)J_4(\kappa) + J_2(\kappa)J_6(\kappa),$$
$$J_{02}(\kappa) = J_1(\kappa)J_6(\kappa) - J_3(\kappa)J_5(\kappa),$$
$$J_{00}(\kappa) = J_1(\kappa)J_4(\kappa) + J_2(\kappa)J_5(\kappa).$$

Eqs. (4.40) and (4.41) implies that

$$J_{01}^2 + J_{02}^2 = J_{00}^2. \tag{4.42}$$

Now, assume that, (**H4$_\mathbf{a}$**): Eq. (4.42) has at least one positive root κ_0. Then, Eq. (4.36) has a pair of purely imaginary roots $\pm i\kappa_0$. For κ_0, the corresponding critical value of the time delay ρ is obtained as

$$\rho_j = \frac{1}{\kappa_0}\arccos\frac{J_{01}(\kappa_0)}{J_{00}(\kappa_0)} + \frac{2\pi æ}{\kappa_0}, \quad j = 0,1,2,\ldots. \tag{4.43}$$

Let $\rho_0 = \min\{\rho_j, \quad j = 0,1,2,\ldots\}$.

To estabilish Hopf bifurcation, we show that

$$\left[\frac{d(Re\lambda)}{d\rho}\right]\Big|_{\lambda=i\kappa_0} \neq 0.$$

On differentiating Eq. (4.36) with respect to $\lambda(\rho)$, we obtain

$$\frac{d\lambda}{d\rho}\left((2\lambda+m_1)e^{\lambda\rho} + \rho(\lambda^2+m_1\lambda+m_2)e^{\lambda\rho} + q_1 - o_1\rho e^{-\lambda\rho}\right) + \lambda(\lambda^2+m_1\lambda+m_2)e^{\lambda\rho} - \lambda o_1 e^{-\lambda\rho} = 0. \tag{4.44}$$

$$\begin{aligned}\left[\frac{d\lambda}{d\rho}\right]^{-1} &= \frac{(2\lambda+m_1)e^{\lambda\rho}+q_1}{\lambda(o_1e^{-\lambda\rho} - (\lambda^2+m_1\lambda+m_2)e^{\lambda\rho})} - \frac{\rho}{\lambda} \\ &= \frac{(2\lambda+m_1)e^{\lambda\rho}+q_1}{\lambda(2o_1e^{-\lambda\rho}+q_1\lambda+q_2)} - \frac{\rho}{\lambda} \quad \text{(using Eq. (4.36))}.\end{aligned}$$

$$\left[\frac{d(Re\lambda)}{d\rho}\right]\Big|_{\lambda=i\kappa_0}^{-1} = \frac{L_1L_3+L_2L_4}{L_3^2+L_4^2},$$

where,

$$L_1 = m_1 \cos \kappa_0 \rho_0 - 2\kappa_0 \sin \kappa_0 \rho_0 + q_1,$$
$$L_2 = 2\kappa_0 \cos \kappa_0 \rho_0 + m_1 \sin \kappa_0 \rho_0,$$
$$L_3 = 2\kappa_0 o_1 \sin \kappa_0 \rho_0 - q_1 \kappa_0^2,$$
$$L_4 = 2\kappa_0 o_1 \cos \kappa_0 \rho_0 + \kappa_0 q_2.$$

If $\mathbf{H_{4_b}} : L_1 L_3 + L_2 L_4 \neq 0$, then $\left[\frac{\mathrm{d}(Re\lambda)}{\mathrm{d}\rho}\right]\Big|^{-1}_{\lambda=\mathrm{i}\kappa_0} \neq 0$. Thus, the following theorem is stated using [15]:

Theorem 4.3.9. *Suppose that $\mathbf{H_{4_a}} - \mathbf{H_{4_b}}$ hold. Then, the endemic equilibrium E_e is locally asmptotically stable when $\rho \in [0, \rho_0)$; the system (4.3) undergoes a Hopf bifurcation at E_e when $\rho = \rho_0$, and a family of periodic solutions bifurcate from E_e near $\rho = \rho_0$.*

Case (v) $\rho_1 > 0,\ \rho_2 > 0$ and $\rho_2 \in (0, \rho_2^*)$.

We consider Eq. (4.14) with ρ_2 lies in stable interval and ρ_1 is regarded as the parameter.

Let $\lambda = \mathrm{i}\kappa_*$, $(\kappa_* > 0)$ be root of Eq. (4.14). Then,

$$G_1 \sin \kappa_* \rho_1 + G_2 \cos \kappa_* \rho_1 = G_3, \tag{4.45}$$
$$G_1 \cos \kappa_* \rho_1 - G_2 \sin \kappa_* \rho_1 = G_4, \tag{4.46}$$

where,

$$G_1 = n_1 \kappa_* - o_1 \sin \kappa_* \rho_2,$$
$$G_2 = n_2 + o_1 \cos \kappa_* \rho_2,$$
$$G_3 = \kappa_*^2 - m_2 - l_1 \kappa_* \sin \kappa_* \rho_2 - l_2 \cos \kappa_* \rho_2,$$
$$G_4 = -m_1 \kappa_* - l_1 \kappa_* \cos \kappa_* \rho_2 + l_2 \sin \kappa_* \rho_2.$$

From Eqs. (4.45) and (4.46), we obtain

$$\kappa_*^4 - 2\kappa_*^3 l_1 \sin(\kappa_* \rho_2) + \kappa_*^2 \left(-2(l_2 - l_1 m_1)\cos(\kappa_* \rho_2) + l_1^2 + m_1^2 - 2m_2 - n_1^2\right) + 2\kappa_* \sin(\kappa_* \rho_2)$$
$$\times (-l_2 m_1 + l_1 m_2 + n_1 o_1) + 2\cos(\kappa_* \rho_2)(l_2 m_2 - n_1 o_1) + l_2^2 + m_2^2 - n_1^2 - o_1^2 = 0. \tag{4.47}$$

H5$_\mathbf{a}$: Let Eq. (4.47) has at least finite positive root. We denote the positive roots of Eq. (4.47) as κ_{1*}, κ_{2*}, $\kappa_{3*},\ldots,\kappa_{k*}$. For every κ_{i*}, $i=1,2,\ldots,k$, the corresponding critical value of time delay is

$$\rho_{1i*}^{(j)}=\frac{1}{\kappa_{i*}}\arccos\left\{\frac{G_1G_4+G_2G_3}{G_1^2+G_2^2}\right\}_{\kappa_*=\kappa_{i*}}+\frac{2\pi j}{\kappa_{i*}},\quad j=0,1,2,\ldots. \tag{4.48}$$

Let $\rho_1^*=\min\left\{\rho_{1i*}^{(0)}\right\},\kappa^*=\kappa_{i*}\Big|_{\rho_1=\rho_1^*},\ i=1,2,\ldots,k.$

Differentiating Eq. (4.14) with respect to ρ_1 yields:

$$\left[\frac{\mathrm{d}\lambda}{\mathrm{d}\rho_1}\right]^{-1}=\frac{2\lambda+m_1+n_1\mathrm{e}^{-\lambda\rho_1}+l_1\mathrm{e}^{-\lambda\rho_2}-\rho_2(l_1\lambda+l_2)\mathrm{e}^{\lambda\rho_2}-o_1\rho_2\mathrm{e}^{-\lambda(\rho_1+\rho_2)}}{\lambda\left((n_1\lambda+n_2)\mathrm{e}^{-\lambda\rho_1}+o_1\mathrm{e}^{-\lambda(\rho_1+\rho_2)}\right)}-\frac{\rho_1}{\lambda}.$$

$$\left[\frac{\mathrm{d}\left(Re\lambda\right)}{\mathrm{d}\rho_1}\right]\Big|_{\lambda=\mathrm{i}\kappa^*}^{-1}=\frac{D_1D_3+D_2D_4}{D_3^2+D_4^2},$$

where,

$$D_1=-\kappa^*l_1\rho_2\sin\left(\kappa^*\rho_2\right)+l_1\cos\left(\kappa^*\rho_2\right)-l_2\rho_2\cos\left(\kappa^*\rho_2\right)+m_1+n_1\cos\left(\kappa^*\rho_1\right)-o_1\rho_2\cos\left(\kappa^*\left(\rho_1^*+\rho_2\right)\right),$$
$$D_2=2\kappa^*-l_1\sin\left(\kappa^*\rho_2\right)+l_2\rho_2\sin\left(\kappa^*\rho_2\right)+\kappa^*l_1\rho_2\left(-\cos\left(\kappa^*\rho_2\right)\right)-n_1\sin\left(\kappa^*\rho_1^*\right)+o_1\rho_2\sin\left(\kappa^*\left(\rho_1^*+\rho_2\right)\right)$$
$$D_3=-n_1\kappa^{*2}\cos\kappa^*\rho_1^*+n_2\kappa^*\sin\kappa^*\rho_1^*+o_1\kappa^*\sin\kappa^*(\rho_1^*+\rho_2),$$
$$D_4=n_2\kappa^*\cos\kappa^*\rho_1^*+n_1\kappa^{*2}\sin\kappa^*\rho_1^*+o_1\kappa^*\cos\kappa^*(\rho_1^*+\rho_2).$$

H5$_\mathbf{b}$: $D_1D_3+D_2D_4\neq 0$.

If **H5$_\mathbf{b}$** holds, then $\left[\frac{\mathrm{d}\left(Re\lambda\right)}{\mathrm{d}\rho_1}\right]\Big|_{\lambda=\mathrm{i}\kappa^*}^{-1}\neq 0$. Thus, using the Hopf bifurcation theorem in [15], we state the following theorem:

Theorem 4.3.10. *If the conditions* **H5$_\mathbf{a}$** $-$ **H5$_\mathbf{b}$** *hold and* $\rho_2\in(0,\rho_2^*)$*, then the endemic equilibrium* E_e *of system (4.3) is locally asymptotically stable for* $\rho_1\in[0,\rho_1^*)$*, undergoes a Hopf bifurcation at* $\rho_1=\rho_1^*$ *and a branch of periodic solutions bifurcate from* E_e *near* $\rho_1=\rho_1^*$.

4.4 Numerical simulation

In this section, the analytical results are illustrated numerically using Mathematica 11.

Example 4.2. Let $r = 0.08,\ K = 100,\ \beta = 0.0012,\ \alpha = 0.002,\ \gamma = 0.001,\ \vartheta = 0.01,\ d = 0.008,\ \theta = 0.02.$

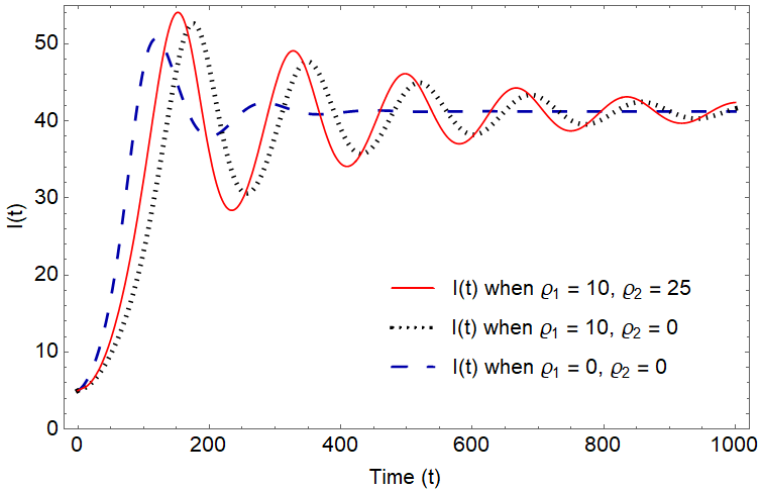
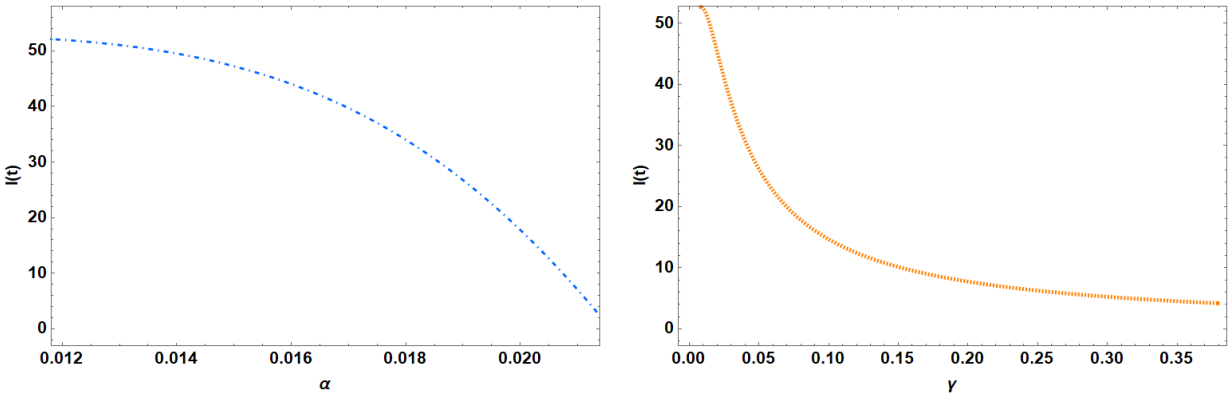


Figure 4.2: Delay effects on $I(t)$.

Fig. 4.2 is plotted for $\sigma = 0.004$ and $\xi = 0.009$, which shows the effect of two time delays ρ_1 and ρ_2 on the infected population $I(t)$. It depicts that when there is no delay, i.e., $\rho_1 = \rho_2 = 0$, the infected individuals will increase and settle down to a steady state after a duration. If there is a time delay in incidence, i.e., $\rho_1 \neq 0$, for instance, $\rho_1 = 10$, then the infective will appear with oscillation and persist at a high level and stabilize after a long period. Lastly, if there are time delays in both incidence and treatment rates, i.e., $\rho_1 > 0,$ and $\rho_2 > 0$, for instance, $\rho_1 = 10$ and $\rho_2 = 25$, then the disease will spread at a higher rate with higher oscillation. It shows the significance of considering both the time delays in studying the spread and control of an epidemic.



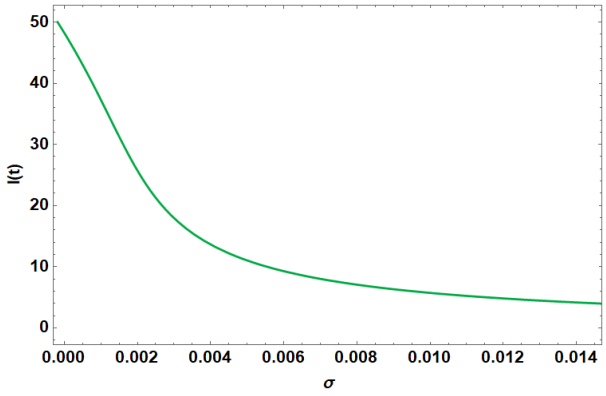
4.3.1: $I(t)$ versus α.

4.3.2: $I(t)$ versus γ.

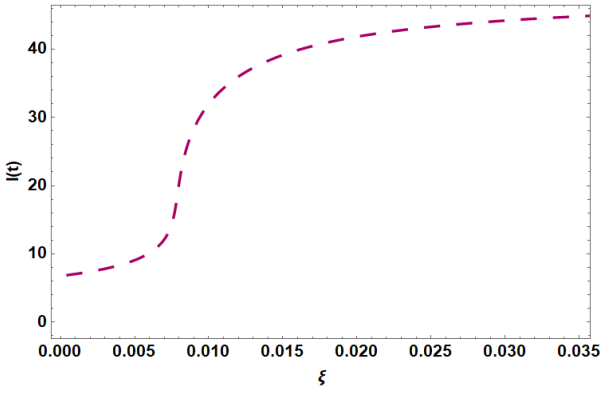
Figure 4.3: Effects of measure of inhibitions α and γ on infectives $I(t)$.

Fig. 4.3 shows the effects of anti-epidemic preventive measures taken by susceptibles and infectives (recalling that α represents the rate of preventive measures adopted by susceptibles

and γ is the rate of preventive measures adopted by infectives). The responsible actions taken by susceptibles and infectives will have different effects and impacts on how rapidly and extensively the infectious diseases will spread. It can be concluded that if both susceptibles and infectives follow effective protection measures, it can significantly increase the ability to control infectious diseases and prevent epidemics.



4.4.1: $I(t)$ versus σ for $\xi = 0.001$.

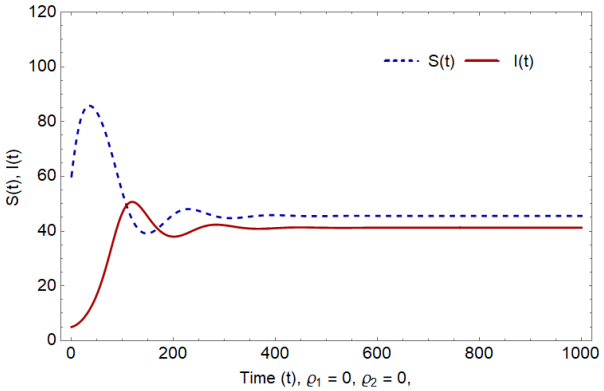


4.4.2: $I(t)$ versus ξ for $\sigma = 0.008$.

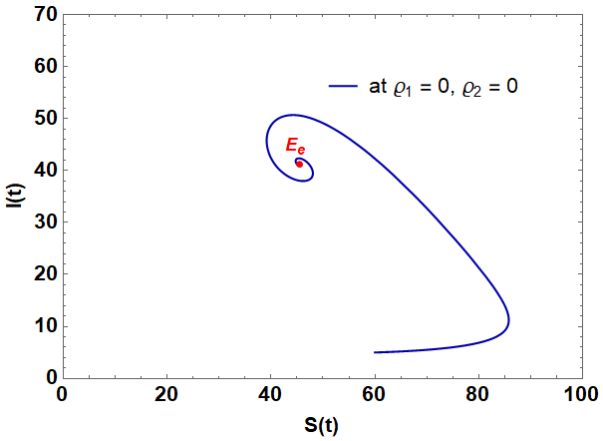
Figure 4.4: Effects of cure rate σ and resouces limitation rate ξ on the infected individuals $I(t)$.

Fig. 4.4.1 is drawn to see the effect of the cure rate σ on the infectives, whereas Fig. 4.4.2 reflects the impact of limited cure resources on the infected community. It shows that the availability of the treatment can substantially control the disease spread and the inadequate access to treatment creates significant obstacles for eliminating the infection.

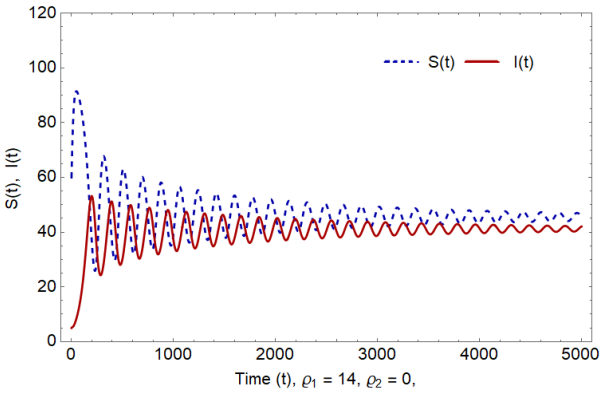
Example 4.3. Let $r = 0.08,\ K = 100,\ \beta = 0.0012,\ \alpha = 0.002,\ \gamma = 0.001,\ \vartheta = 0.01,\ d = 0.008,\ \theta = 0.02,\ \sigma = 0.004,\ \xi = 0.009$. Using these values of parameters, we obtain that $R_0 = 2.63158 > 1$ and the endemic equilibrium $E_e(S^*, I^*) = (45.554, 41.237)$.
We illustrate the Case (ii) of Section 4.3.3, i.e., $\rho_1 > 0$ and $\rho_2 = 0$. We evaluate that $A_{12} = l_{11}^2 - (n_2 + o_1)^2 = -3.51368 \times 10^{-6} < 0$ and $H'(\kappa_0^2) = 0.00405708 > 0$ which confirms the hypothesis $H2_b$ of Theorem 4.3.7. We calculate the critical value of ρ_1 as $\rho_1^* = 14.7333$. As exhibited in subfigures 4.5.1–4.5.4, the endemic equilibrium $E_e(45.554, 41.237)$ is locally asymptotically stable when $\rho_1 < \rho_1^* = 14.7333$, and when the value of ρ_1 crosses the critical value ρ_1^*, then the periodic solutions bifurcates from E_e, which confirms the results of Theorem 4.3.7.



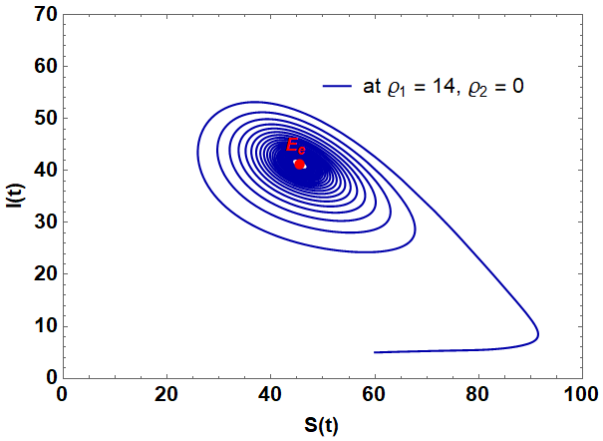
4.5.1: Time series solution of $S(t)$ and $I(t)$ for $\rho_1 = \rho_2 = 0$.



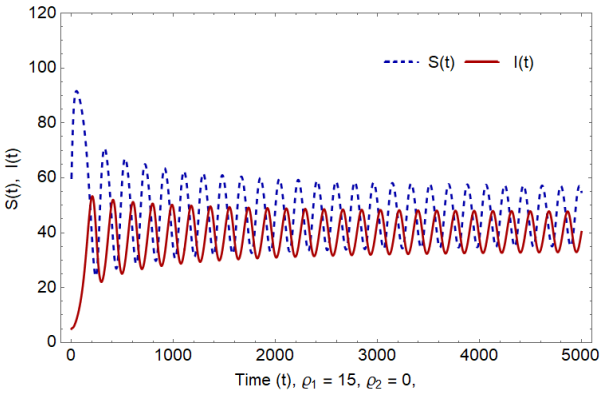
4.5.2: Phase plot of $S(t)$ and $I(t)$ for $\rho_1 = \rho_2 = 0$.



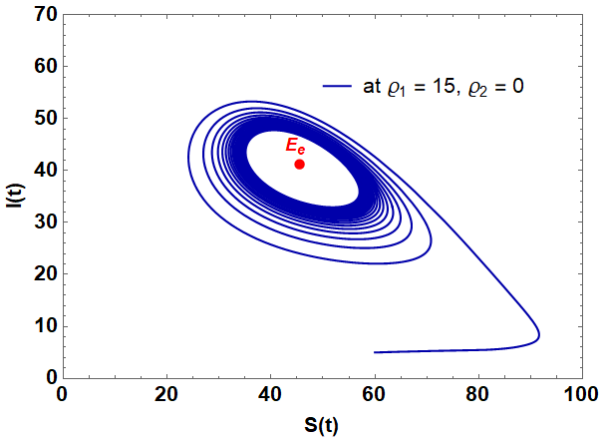
4.5.3: Time series solution of $S(t)$ and $I(t)$ for $\rho_1 = 14$ and $\rho_2 = 0$.



4.5.4: Phase plot of $S(t)$ and $I(t)$ for $\rho_1 = 14$ and $\rho_2 = 0$.



4.5.5: Time series solution of $S(t)$ and $I(t)$ for $\rho_1 = 15$ and $\rho_2 = 0$.

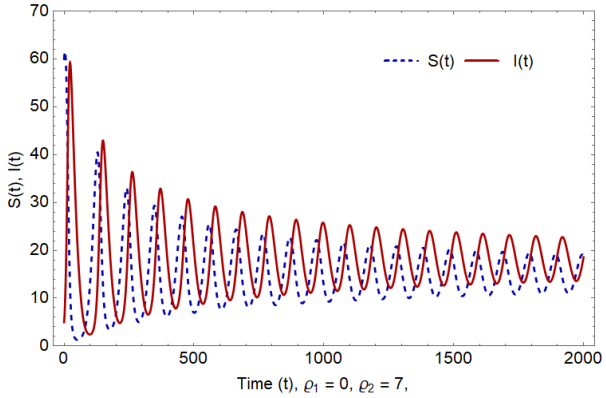


4.5.6: Phase plot of $S(t)$ and $I(t)$ for $\rho_1 = 15$ and $\rho_2 = 0$.

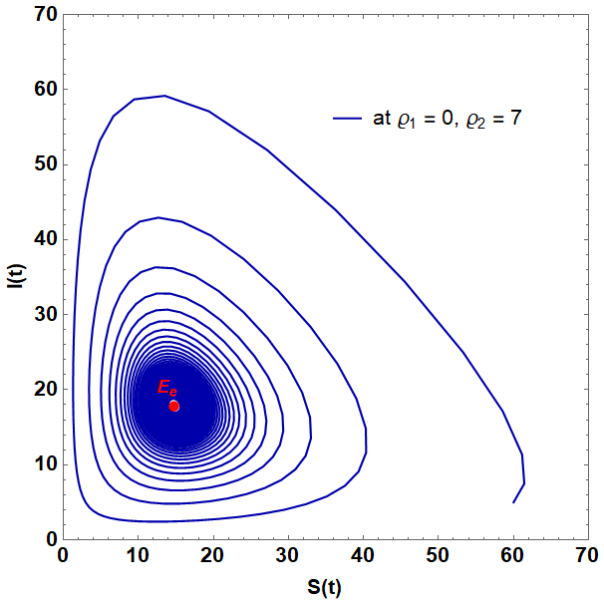
Figure 4.5: Plots of Hopf bifurcation, illustrating case (ii) of Section 4.3.3.

Example 4.4. To illustrate the Case (iii) of Section 4.3.3, i.e., $\rho_1 = 0$ and $\rho_2 > 0$, we consider the following parameters: $r = 0.08$, $K = 100$, $\beta = 0.004$, $\alpha = 0.002$, $\gamma = 0.001$, $\vartheta =$

0.01, $d = 0.008$, $\theta = 0.02$, $\sigma = 0.004$, $\xi = 0.009$. We evaluate that $R_0 = 8.77193 > 1$, $E_e(S^*, I^*) = (14.7911, 17.8592)$, $B_{12} = m_{02}^2 - m_{12}^2 = 0.0000122401 > 0$, $B_{11}^2 - 4B_{12} = 5.03027 * 10^{-7} > 0$, and $G'(\kappa_0^2) = 0.117404 > 0$. Thus, the hypothesis $H3_b$ of the Theorem 4.3.8 holds. The critical value of ρ_2 is calculated as $\rho_2^* = 8.17751$.

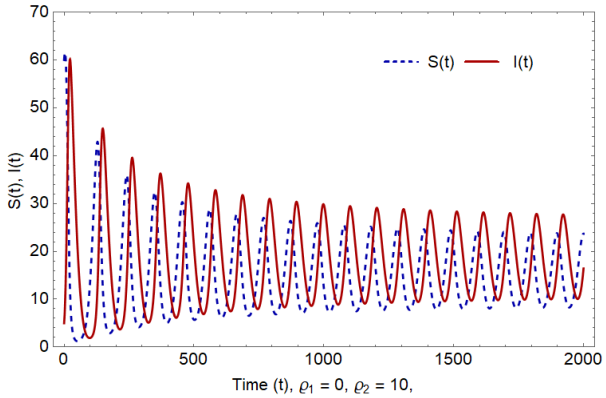


4.6.1: Time series solution of $S(t)$ and $I(t)$ for $\rho_1 = 0$ and $\rho_2 = 7$.

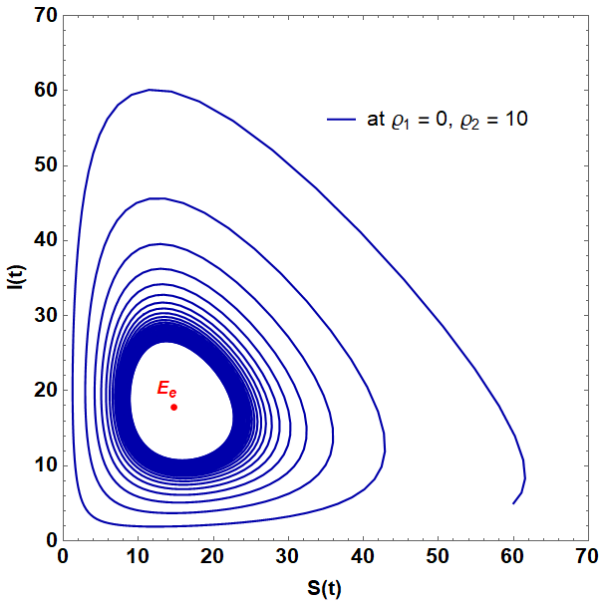


4.6.2: Phase plot of $S(t)$ and $I(t)$ for $\rho_1 = 0$ and $\rho_2 = 7$.

Figs. 4.6.1 and 4.6.2 show the presence of periodic solutions, where the trajectories of $S(t)$ and $I(t)$ reach to the steady-state $E_e(14.7911, 17.8592)$, when the delay value ρ_2 is less than its critical value ρ_2^*.



4.6.3: Time series solution of $S(t)$ and $I(t)$ for $\rho_1 = 0$ and $\rho_2 = 10$.

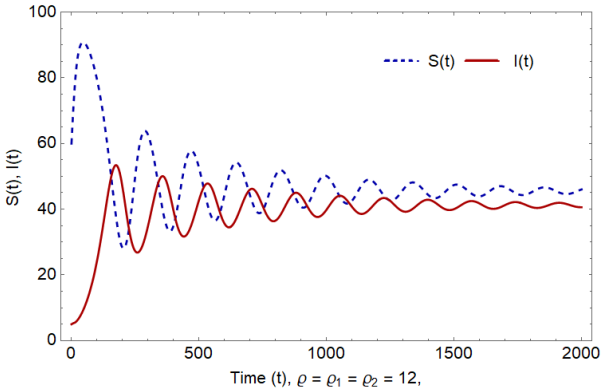


4.6.4: Phase plot of $S(t)$ and $I(t)$ for $\rho_1 = 0$ and $\rho_2 = 10$.

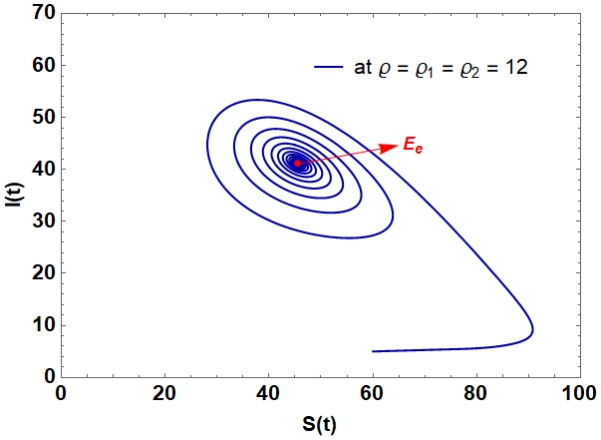
Figure 4.6: Presence of Hopf bifurcation, illustrating case (iii) of Section 4.3.3.

Figs. 4.6.3 and 4.6.4 are plotted for $\rho_2 > \rho_2^*$, which shows that the periodic solutions appear and the trajectories of $S(t)$ and $I(t)$ go away from E_e and destabilization of the system (4.3) at E_e.

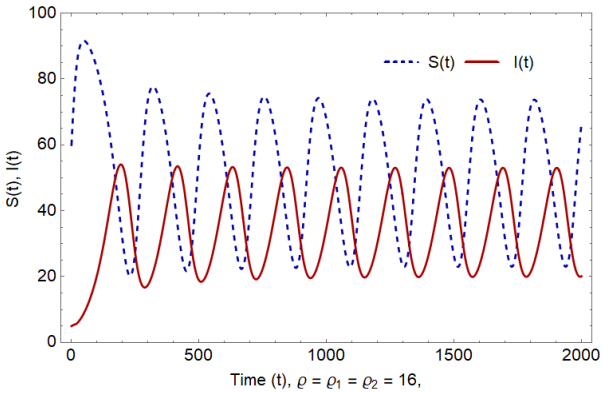
Example 4.5. We consider the case (iv) of Section 4.3.3 (i.e., $\rho_1 = \rho_2 = \rho$) with the same values of parameters as taken in Example (4.3). The critical value of delay ρ is obtained as $\rho_0 = 14.3942$. The time series solutions and the phase plane of $S(t)$ and $I(t)$ are plotted in the Figs. 4.7.1 and 4.7.2 for the delay $\rho = 12$, where the trajectories reaches to the stable endemic equilibrium E_e. On the other hand, Figs. 4.7.3 and 4.7.4 show the instability of E_e when the time delay is higher than its critical values.



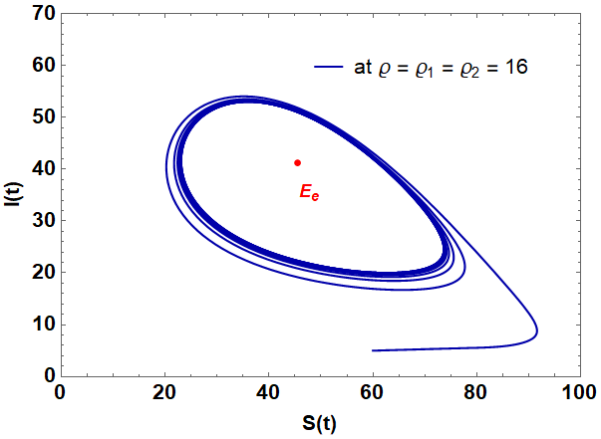
4.7.1: Time series solution of $S(t)$ and $I(t)$ for $\rho = \rho_1 = \rho_2 = 12$.



4.7.2: Phase plot of $S(t)$ and $I(t)$ for $\rho = \rho_1 = \rho_2 = 12$.



4.7.3: Time series solution of $S(t)$ and $I(t)$ for $\rho = \rho_1 = \rho_2 = 16$.

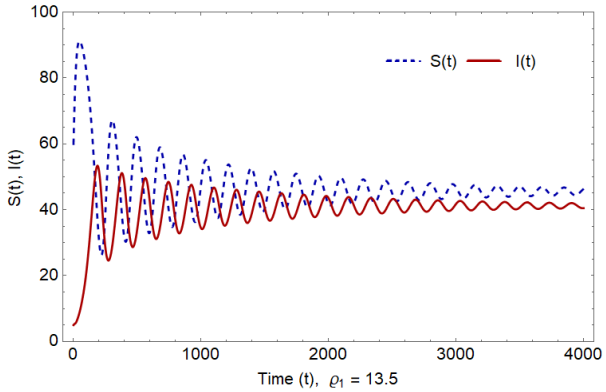


4.7.4: Phase plot of $S(t)$ and $I(t)$ for $\rho = \rho_1 = \rho_1 = 16$.

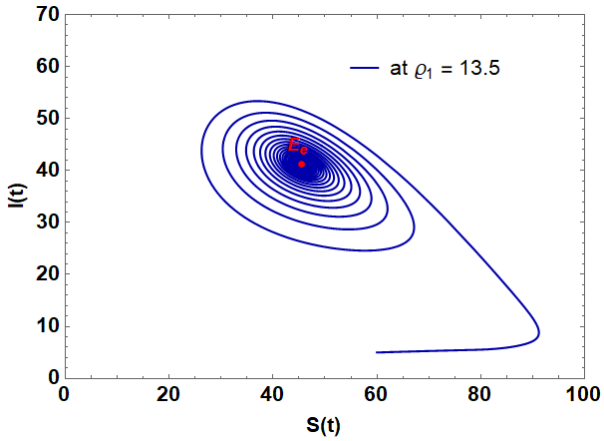
Figure 4.7: Plots of Hopf bifurcation, illustrating case (iv) of Section 4.3.3.

Example 4.6. In this example, Case (v) of Section 4.3.3 is illustrated with the same values of parameters as taken in Example (4.3). We fix $\rho_2 = 7$. The critical value of ρ_1 is obtained as $\rho_1^* = 14.2$. Cleary, the impact of both the delay parameters ρ_1 and ρ_2 can be seen. Figs.

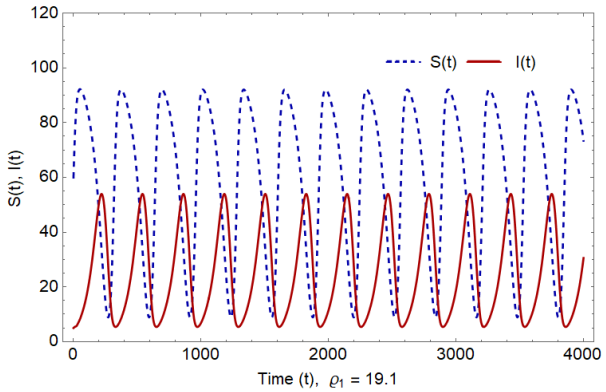
4.8.1 and 4.8.2 show the oscillatory trajectories of $S(t)$ and $I(t)$, which stabilized to endemic equilibrium E_e. Figs. 4.8.3 and 4.8.4 show the stable periodic solutions and a stable limit cycle, which destabilized the endemic equilibrium E_e. It shows that the infectious diseases continue to proliferate periodically as ρ_1 crosses ρ_1^*. Thus, the consideration of both the delays majorly affects the number of infectives and the spread of infection.



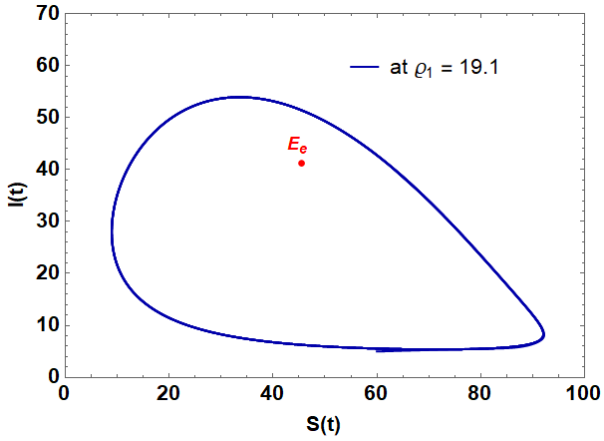
4.8.1: Time series solution of $S(t)$ and $I(t)$ for $\rho_1 = 13.5$ and $\rho_2 = 7$.



4.8.2: Phase plot of $S(t)$ and $I(t)$ for $\rho_1 = 13.5$ and $\rho_2 = 7$.



4.8.3: Time series solution of $S(t)$ and $I(t)$ for $\rho_1 = 19.1$ and $\rho_2 = 7$.



4.8.4: Phase plot of $S(t)$ and $I(t)$ for $\rho_1 = 19.1$ and $\rho_2 = 7$.

Figure 4.8: Plots of Hopf bifurcation, illustrating case (v) of Section 4.3.3.

4.5 Discussion

A time-delayed SIR epidemic model is proposed and analyzed mathematically with the logistic growth of susceptible individuals. The Crowley –Martin type nonlinear incidence rate is incorporated to examine the transmission of infectious diseases from the infected population to the susceptible population. The Holling type III treatment function is considered for the treatment of infectives, which incorporates the limited availability of

medical resources such as ICU (intensive care unit) beds, medications, ventilators, hospital conditions, etc. We include two explicit time-delays in the present model: a time delay in the incidence rate, which represents the latent period; a time delay in the nonlinear treatment function, which measures the impact of delay in providing the appropriate therapy to infectives. We obtain the model's feasible equilibria, namely, disease-free equilibrium (DFE) and the endemic equilibrium (EE). The basic reproduction number R_0 is derived. We show that the DFE of the delayed system is locally asymptotically stable when R_0 is less than unity, unstable when R_0 is greater than unity, and neutrally stable when R_0 is equal to unity. When there is no latent period, then the DFE for $R_0 = 1$ reveals the presence of forward bifurcation. The occurrence of forward bifurcation has an important implication in the epidemic models. It shows that the necessary and sufficient condition to eliminate the disease is to bring the basic reproduction number below unity. Further, the prerequisite for the existence of EE is obtained, and the stability of the EE is investigated. Hopf bifurcation near EE is shown for different possible cases of time delays, and the explicit formulas for the critical values of time-delays are derived.

Further, the numerical simulations verify the analytical results and show the importance of considering nonlinearity and time delays. The latent period and the delay in treating patients have a significant impact on the number of infected individuals (Fig. 4.2). These delays result in spreading infections at a high rate, and so, the infection stays for a longer period. Considering the time delays as the bifurcation parameters can affect the equilibrium's stability (Figs. 4.5–4.8). When the delay is suitably small, the system reaches its steady state, but stabilizing the system to its equilibrium will be out of control as it crosses the critical value. The course will produce a limit cycle and destabilize the endemic stability, making it difficult to control the spread of infection. The effect of preventive measures adopted by susceptibles and infectives has been shown numerically (Fig. 4.3). It shows that human behavior towards the disease exerts a powerful influence on pathogen invasion dynamics. A better understanding of risk factors for developing infectious disease in the general public is a requirement for disease prevention. Increasing knowledge, changing attitudes, and reducing risky behaviors towards the disease can reduce the death rate, disability due to illness, and epidemic growth. The effects of cure rate and limitation in medical resources are also shown numerically (Fig. 4.4). It is observed that increasing medical resource acquisition has effects on eliminating infection from society. A reasonable and timely treatment can speed up the recovery process and prevent disease completely, whereas the limited medical resources availability increases

the number of infected people. Thus, timely treatment and resource accessibility need to be readily available. The earlier treatment accessibility in the disease process can mitigate the disease spread and save human lives.

Chapter 5

A nonlinear time-delayed epidemic model with aware individuals class, Michaelis-Menten incidences, and nonlinear treatment

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Awareness plays a vital role in informing and educating the public about infection risk during an outbreak and further modifying their behavior, which influences the incidence pattern. Therefore, this chapter presents a time-delayed epidemic model incorporating a class of aware susceptible individuals in the SIR compartmental model, contributing to public policy development in controlling disease spread. We have considered the Michaelis-Menten functional type nonlinear incidence rates for unaware and aware susceptibles with the latent period and a saturated treatment rate for infectives. We analyze the model mathematically to describe disease transmission dynamics using the stability theory of delay differential equations for two obtained equilibria; disease-free (DFE) and endemic (EE). Moreover, numerical simulations validate the analytical findings.

5.1 Introduction

When a disease emerges, changes in people's behavior concerning that disease can change the development of the pathogen. Individuals who are aware of the illness's spread

take precautions to diminish their susceptibility, which can suppress disease transmission in society. Many authors have studied the effect of awareness on the epidemic models (for instance, [54, 56, 59]). Funk et al. [70] deliberate the influence and significance of awareness programs on the spread and control of the outbreak. Misra et al. and Dubey et al. [94,115] studied a nonlinear mathematical model to discuss the effects of awareness programs on the transmission of infectious diseases. Kumar et al. [136] incorporated the alert individuals class into the SIR epidemic model and studied the effect of alertness in infectious disease transmission dynamics.

A significant factor in the dynamical study of infectious disease is the incidence rate by which infection transmits to susceptible individuals. Therefore, numerous authors are keen to deliberate nonlinear incidence rates to study the transmission dynamics of infectious diseases (e.g., [10, 12, 61, 62, 73, 134, 142]). The Michaelis–Menten type functional response also has a nonlinear form. In this incidence rate, the number of adequate contacts per infective in unit time grows less rapidly as the total population increases. Michaelis-Menten contact rate is of the form $g(I) = \beta\sigma I/(1+I)$. It combines the bilinear and standard incidence rate approaches by assuming that if the number of infectives I is low, the number of actual per capita infectives $g(I)$ is proportional to I, whereas if the number of infected individuals I is large, there is a saturation effect which makes the number of actual infectives constant [32, 57].

Affordable and safe medical treatments are also necessary, which leads to a decline in the number of infected individuals. When a disease emerges, medical facilities and subsequent treatments may need some time to develop and implement; therefore, choosing a reasonable treatment rate is essential. Due to the limited medical resources, providing treatment to all the infectives puts a tremendous burden on public health associations. Thus, considering a reasonable treatment rate in the disease transmission and control epidemic models is essential. The saturating treatment rate makes the epidemic model more realistic, as, in this treatment rate, the treatment capacity tends to a finite limit as the number of infected individuals increases. [106, 116, 125].

Motivated by the work and facts as mentioned above, in this chapter, we present an infectious disease transmission compartmental model comprising four subpopulations: fully susceptible population, aware susceptible population, infected population, and recovered population and formulate a time-delayed epidemic mathematical model that incorporates two explicit nonlinear incidences with a latent period and a nonlinear treatment rate for the infected individuals. We have considered the Michaelis–Menten type

nonlinear incidence rate that prevents the unboundedness of the infected individuals and the saturating treatment rate for infected people, including the limited accessibility of treatment resources. At the beginning of the infectious disease, there is a time known as the latency period, before an infected person can transmit the infection to another person. Therefore, the inclusion of the latent period in the incidence pattern makes the present model more realistic. After formulating the model, we perform a mathematical analysis that allows long-term qualitative predictions of outbreaks and the persistence of the disease. We derive the basic reproduction number and estimate how an infection can spread in a population. Using R_0, the local and global stability behavior of two obtained equilibria, namely, disease-free and endemic, is investigated, revealing the persistence or eradication of infection. The global stability behavior of both the equilibria is established by constructing Lyapunov functionals and using the Lyapunov-LaSalle invariance approach. The Hopf bifurcation regarding the time-delay as a bifurcation parameter is established, which shows the oscillatory and periodic solutions near-endemic equilibrium. The numerical experiments show the significance of the model's variables and parameters and suggest strategies that could prevent infection.

5.2 Model derivation

Let P denotes the total population and the transmission of infectious diseases involves four types of subpopulations: Susceptible individuals $S_p(t)$, Aware individuals $A_p(t)$, Infected individuals $I_p(t)$, and Recovered individuals $R_p(t)$. That is, $P = S_p(t) + A_p(t) + I_p(t) + R_p(t)$, which means that the individuals categorized in $S_p(t)$, $A_p(t)$, $I_p(t)$ and $R_p(t)$ may vary with time t and P is a fixed population. It is assumed that each subpopulation of the SAIR model is well mixed and interact homogeneously with each other.

Let κ denotes the constant recruitment rate of susceptibles. ξ is the awareness rate in susceptible individuals, and thus the term ξS_p enters the class $A_p(t)$. We consider Michaelis–Menten type two explicit nonlinear incidence rates with the following interpretation: the term $\Psi(S_p(t-\rho), I_p(t-\rho)) = \frac{\beta\sigma S_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)}$ represents the incidence rate when susceptible individuals catch the infection from infected individuals, and the term $\Lambda(A_p(t-\rho), I_p(t-\rho)) = \frac{\gamma\sigma A_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)}$ represents the incidence rate when aware individuals catch the infection from infectives. Here, β and γ denote the force of infection among susceptible and aware individuals, respectively, and σ denotes the average num-

ber of contact partners. We assume that $\gamma < \beta$, as the aware individuals are at a lower risk of getting infected than fully susceptible individuals. The parameter $\rho > 0$ denotes the time delay representing the latent phase having a fixed duration. During the latent period, a host may or may not show symptoms, but the host cannot infect other hosts in both cases. Thus, the latent period significantly influences the spreading dynamics of an infectious disease or epidemic. Since the aware individuals can also become infected, perhaps at a lower rate than fully susceptibles, they also have some behavioral responses and have a latency phase due to immunological reasons. Thus, the time delay ρ is the constant latency time and represents the time taken by the fully susceptible and aware individuals, that, infected at a time t can infect other susceptible and aware individuals at time $t+\rho$ only. The parameters ϑ represents the natural death rate, d represents the disease-induced death rate, and θ represents the recovery rate of infected individuals. The nonlinear term $h(I_p(t)) = \frac{aI_p^2}{bI_p^2+cI_p+1}$ represents the saturated treatment rate of infectives, where a denotes the maximum treatment (cure) rate, b denotes the rate of limitations in treatment availability, and c denotes the saturation constant. The transition diagram of the model (5.1) is shown in Fig. 5.1 and the description of the parameters is given in Table 5.1.

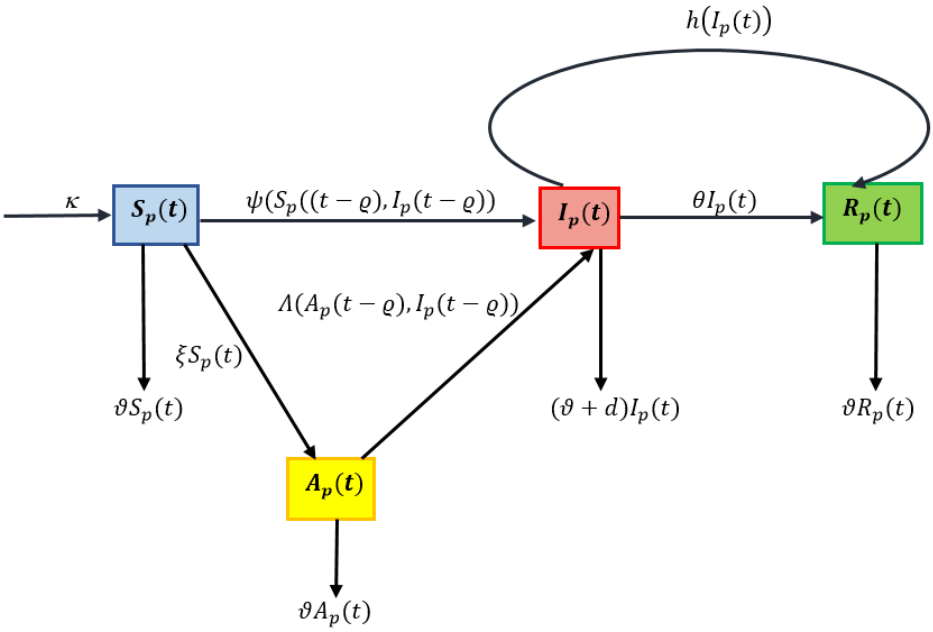


Figure 5.1: Block diagram of the model (5.1).

Table 5.1: Symbolizations of variables and parameters.

Notation	Description
P	Total constant population
$S_p(t)$	Susceptible subpopulation (full vulnerable)
$A_p(t)$	Aware subpopulation
$I_p(t)$	Infected subpopulation
$R_p(t)$	Recovered subpopulation
ρ	Time delay
κ	Susceptibles' recruitment rate
β	Rate of transmission of susceptible class to infected class
γ	Rate of transmission of aware class to infected class
σ	Average contact partners
ξ	Awareness rate in susceptibles
ϑ	Natural mortality rate
d	Death rate due to disease
θ	Recovery rate
a	Cure rate
b	Limitations rate in treatment availability
c	Saturation constant
$\Psi(S_p(t), I_p(t))$	Michaelis–Menten incidence rate among susceptibles
$\Lambda(S_p(t), I_p(t))$	Michaelis–Menten incidence rate among aware individuals
$h(I_p(t))$	Saturated treatment rate of infected individuals

The novel mathematical disease transmission and control model based on the above assumptions is presented under the following system of delay differential equations:

$$\begin{aligned}
\frac{\mathrm{d}S_p}{\mathrm{d}t} &= \kappa - \vartheta S_p - \frac{\beta\sigma S_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)} - \xi S_p,\\
\frac{\mathrm{d}A_p}{\mathrm{d}t} &= \xi S_p - \vartheta A_p - \frac{\gamma\sigma A_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)},\\
\frac{\mathrm{d}I_p}{\mathrm{d}t} &= \frac{\beta\sigma S_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)} + \frac{\gamma\sigma A_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)} - (\vartheta+d+\theta)I_p - \frac{aI_p^2}{bI_p^2+cI_p+1},\\
\frac{\mathrm{d}R_p}{\mathrm{d}t} &= \theta I_p - \vartheta R_p + \frac{aI_p^2}{bI_p^2+cI_p+1}.
\end{aligned} \tag{5.1}$$

subject to the initial conditions $\phi = (\phi_1, \phi_2, \phi_3, \phi_4)$ defined in the Banach space

$$C_+ = \{\phi \in C([-\rho, 0], R_+^4) : \phi_1(\Omega) = S_p(\Omega), \phi_2(\Omega) = A_p(\Omega), \phi_3(\Omega) = I_p(\Omega), \phi_4(\Omega) = R_p(\Omega)\}, \tag{5.2}$$

where $R_+^4 = \{(S_p, A_p, I_p, R_p) \in R^4 : S_p \geq 0, A_p \geq 0, I_p \geq 0, R_p \geq 0\}$. Biologically, it is assumed that $\phi_i > 0 \quad (i = 1,2,3,4)$.

We observe that the incidence functions $\Psi(S_p(t-\rho), I_p(t-\rho))$, $\Lambda(A_p(t-\rho), I_p(t-\rho))$ and the treatment rate function $h(I_p(t))$ are continuously differentiable, positive, and monotonically increasing for all $S_p(t)$, $A_p(t)$, $I_p(t) > 0$. That is, the following postulates hold:

P1. $\Psi(S_p(t), I_p(t)) > 0$, $\Psi'_{S_p}(S_p(t), I_p(t)) > 0$, $\Psi'_{I_p}(S_p(t), I_p(t)) > 0$ for $S_p(t) > 0$, $\Lambda(A_p(t), I_p(t)) > 0$, $\Lambda'_{A_p}(A_p(t), I_p(t)) > 0$, $\Lambda'_{I_p}(A_p(t), I_p(t)) > 0$ for $A_p(t) > 0$ and $I_p(t) > 0$.

P2. $\Psi(S_p(t), 0) = \Psi(0, I_p(t)) = 0$, $\Psi'_{S_p}(S_p(t), 0) = 0$, $\Psi'_{I_p}(S_p(t), 0) > 0$ for $S_p(t) > 0$, $I_p(t) > 0$ and,
$\Lambda(A_p(t), 0) = \Lambda(0, I_p(t)) = 0$, $\Lambda'_{A_p}(A_p(t), 0) = 0$, $\Lambda'_{I_p}(A_p(t), 0) > 0$ for $A_p(t) > 0$, $I_p(t) > 0$.

P3. $h(0) = 0$, $h'(0) > 0$ for $I_p(t) \geq 0$.

5.3 Basic properties

For biological reasons, we assume that all the parameters of the model (5.1) are positive. That is, $\kappa,\ \vartheta,\ \beta,\ \sigma,\ \xi,\ \gamma,\ d,\ \theta,\ a,\ b,\ c\ >\ 0$.

Positivity: We see that the positivity of the above initial conditions for S_p, A_p, I_p and R_p in $[-\rho,0]$ imply positivity for all solutions $(S_p(t),A_p(t),I_p(t),R_p(t))$, $t>0$ of model (5.1). We further note that $S_p(t)$ can never vanish since at each time $t>0$ where $S_p(t)$ vanishes, it is $\frac{\mathrm{d}S_p}{\mathrm{d}t}=\kappa>0$.

We prove the following lemma.

Lemma 5.3.1. *The compact set*

$$D=\{(S_p,A_p,I_p,R_p)\in R_+^4 : S_p(t)+A_p(t)+I_p(t)+R_p(t)\leq \frac{\kappa}{\vartheta}\}$$

is globally attractive and invariant for the solutions of (5.1).

Proof. Since the right-hand side of the model (5.1), and its derivatives are continuous, therefore, it assures the wellpossness of the model (5.1). Summing up all the equations of the model (5.1) results to

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}t}(S_p(t)+A_p(t)+I_p(t)+R_p(t)) &= \kappa-\vartheta(S_p+A_p+I_p+R_p)-dI\\ &\leq \kappa-\vartheta(S_p+A_p+I_p+R_p).\end{aligned} \tag{5.3}$$

Thus, we obatin

$$0<S_p(t)+A_p(t)+I_p(t)+R_p(t)\leq \frac{\kappa}{\vartheta}-\left(S_p(0)+A_p(0)+I_p(0)+R_p(0)-\frac{\kappa}{\vartheta}\right)e^{-\vartheta t} \tag{5.4}$$

Thus, the invariant region for the existence of the solutions is given as

$$0<\lim_{t\to\infty}(S_p(t)+A_p(t)+I_p(t)+R_p(t))\leq \frac{\kappa}{\vartheta}. \tag{5.5}$$

Hence, the model (5.1) has closed and bounded solutions. ■

5.4 Mathematical analysis

This section obtains the disease-free equilibrium (DFE), then the basic reproduction number R_0, and investigates the system's stability at DFE. We show the existence of

the endemic equilibrium (EE) and examine its stability and show the presence of Hopf bifurcation around it. Also, the global stability behavior of the DFE and EE is investigated with the help of R_0.

We observe that the first three equations of the model (5.1) are free from $R(t)$; therefore, without loss of generality, the following reduced system of the delay differential equations is sufficient to study for the analysis purpose:

$$\begin{aligned}
\frac{\mathrm{d}S_p}{\mathrm{d}t} &= \kappa - \vartheta S_p - \frac{\beta\sigma S_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)} - \xi S_p,\\
\frac{\mathrm{d}A_p}{\mathrm{d}t} &= \xi S_p - \vartheta A_p - \frac{\gamma\sigma A_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)},\\
\frac{\mathrm{d}I_p}{\mathrm{d}t} &= \frac{\beta\sigma S_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)} + \frac{\gamma\sigma A_p(t-\rho)I_p(t-\rho)}{1+I_p(t-\rho)} - (\vartheta+d+\theta)I_p - \frac{aI_p^2}{bI_p^2+cI_p+1}.
\end{aligned} \tag{5.6}$$

Taking the system (5.6) into rest, we find that the system (5.6) exhibits two equilibria:

(i) The disease-free equilibrium (DFE): $E_0(S_0,A_0,I_0) = E_0\left(\frac{\kappa}{\vartheta+\xi}, \frac{\kappa\xi}{\vartheta(\vartheta+\xi)}, 0\right)$.
(ii) The positive or endemic equilibrium (EE): $E_e(S_p^*,A_p^*,I_p^*)$, where S_p^*, A_p^* and I_p^* are obtained in Subsection 5.4.2.

5.4.1 Disease-free equilibrium and stability

The system (5.6) has a disease-free equilibrium (DFE) of the form $E_0(S_0,A_0,I_0) = E_0\left(\frac{\kappa}{\vartheta+\xi}, \frac{\kappa\xi}{\vartheta(\vartheta+\xi)}, 0\right)$, and at E_0, the characteristic equation is obtained as

$$(-\vartheta-\xi-\lambda)(-\vartheta-\lambda)\left(\frac{\kappa\sigma \mathrm{e}^{-\lambda\rho}}{\vartheta(\vartheta+\xi)}(\beta\vartheta+\xi\gamma) - (\vartheta+d+\theta) - \lambda\right) = 0. \tag{5.7}$$

The roots of the Eq. (5.7) are $\lambda_1 = -\vartheta-\xi$, $\lambda_2 = -\vartheta$, and the remaining roots are the solutions of the transcendental equation

$$\frac{\kappa\sigma}{\vartheta(\vartheta+\xi)}(\beta\vartheta+\xi\gamma)\mathrm{e}^{-\lambda\rho} - (\vartheta+d+\theta) - \lambda = 0. \tag{5.8}$$

Assume that

$$g(\lambda) := \lambda + \vartheta + d + \theta - \frac{\kappa\sigma(\beta\vartheta+\xi\gamma)\mathrm{e}^{-\lambda\rho}}{\vartheta(\vartheta+\xi)} = 0. \tag{5.9}$$

We define the term $\frac{\kappa\sigma(\beta\vartheta+\xi\gamma)e^{-\lambda\rho}}{\vartheta(\vartheta+\xi)(\vartheta+d+\theta)}$ at $\rho=0$ as the basic reproduction number R_0 of the system (5.6). Thus, the system (5.6) has R_0 of the form

$$R_0=\frac{\kappa\sigma(\beta\vartheta+\xi\gamma)}{\vartheta(\vartheta+\xi)(\vartheta+d+\theta)}.$$

Analysis for $R_0\neq 1$

The roots λ_1 and λ_2 of Eq. (5.7) preserve negative signs. Therefore, the analysis is now based on the Eq. (5.9).
Note that

$$g(0)=\vartheta+d+\theta-\frac{\kappa\sigma(\beta\vartheta+\xi\gamma)}{\vartheta(\vartheta+\xi)}=(\vartheta+d+\theta)(1-R_0). \tag{5.10}$$

If $R_0>1$, then $g(0)<0$. Also,

$$g'(\lambda)=1+\rho\frac{\kappa\sigma(\beta\vartheta+\xi\gamma)e^{-\lambda\rho}}{\vartheta(\vartheta+\xi)}>0. \tag{5.11}$$

Hence, $g(0)<0$ and $g'(\lambda)>0$ imply that $g(\lambda)=0$ has a unique and positive real root when $R_0>1$.

Now, when $R_0<1$, then

$$\mathrm{Re}\,\lambda=\frac{\kappa\sigma(\beta\vartheta+\xi\gamma)\cos(Im\lambda)\rho}{\vartheta(\vartheta+\xi)}e^{-(\mathrm{Re}\,\lambda)\rho}-(\vartheta+d+\theta)<\frac{\kappa\sigma(\beta\vartheta+\xi\gamma)}{\vartheta(\vartheta+\xi)}-(\vartheta+d+\theta)<0. \tag{5.12}$$

Therefore, $R_0<1$ implies that λ is a root of equation (5.7) with negative real part. Thus, the following theorem is stated:

Theorem 5.4.1. *The disease-free equilibrium (DFE) E_0 is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$ for $\rho\geq 0$.*

Analysis at $R_0=1$

Now we analyze the system (5.6) at E_0 and $R_0=1$ for $\rho>0$ and $\rho=0$, seperately.

Case (i) $\rho>0$

When $R_0=1$, then Eq. (5.9) has a simple characteristic root $\lambda=0$. It is also noticed that $R_0=1$ gives $\kappa\sigma(\beta\vartheta+\xi\gamma)=\vartheta(\vartheta+\xi)(\vartheta+d+\theta)$.

Let $\lambda = p + \mathrm{i}q$ be the other solution of Eq. (5.9), then we get:

$$p + \mathrm{i}q + d + \vartheta + \theta - (\cos q\rho - \mathrm{i}\sin q\rho)(d + \vartheta + \theta)\mathrm{e}^{-p\rho} = 0. \tag{5.13}$$

Using Euler's formula and on splitting real and imaginary parts of Eq. (5.13), we get

$$\begin{aligned} p + d + \vartheta + \theta &= \mathrm{e}^{-p\rho}\ (d + \vartheta + \theta)\ \cos q\rho, \\ q &= -(d + \vartheta + \theta)\ \mathrm{e}^{-p\rho}\ \sin q\rho. \end{aligned} \tag{5.14}$$

A root satisfying both the equations of (5.14) must be a solution to the equation attained by squaring and adding these two equations. Hence, we get

$$(p + d + \vartheta + \theta)^2 + q^2 = (d + \vartheta + \theta)^2\ \mathrm{e}^{-2p\rho}. \tag{5.15}$$

For Eq. (5.15) to hold, we must have $p \leq 0$. Thus, E_0 is linearly neutrally stable.

Case (ii) $\rho = 0$

In this case, we study the qualitative behavior of the undelayed system (5.6) (i.e., $\rho = 0$) through the stability analysis near critical points, i.e., at E_0 and $R_0 = 1$, using the bifurcation theory approach [40], depending upon the center manifold theory [17].

For simplicity, let $S_p = x_1$, $A_p = x_2$, and $I_p = x_3$. So, the system (5.6) reduces to

$$\begin{aligned} \frac{\mathrm{d}x_1}{\mathrm{d}t} &= \kappa - \vartheta x_1 - \frac{\beta\sigma x_1(t)x_3(t)}{1 + x_3(t)} - \xi x_1 \equiv f_1, \\ \frac{\mathrm{d}x_2}{\mathrm{d}t} &= \xi x_1 - \vartheta x_2 - \frac{\gamma\sigma x_2(t)x_3(t)}{1 + x_3(t)} \equiv f_2, \\ \frac{\mathrm{d}x_3}{\mathrm{d}t} &= \frac{\beta\sigma x_1(t)x_3(t)}{1 + x_3(t)} + \frac{\gamma\sigma x_2(t)x_3(t)}{1 + x_3(t)} - (\vartheta + d + \theta)x_3 - \frac{ax_3^2}{bx_3^2 + cx_3 + 1} \equiv f_3. \end{aligned} \tag{5.16}$$

Observe that $R_0 = 1 \iff$ the bifurcation parameter $\sigma = \sigma^* = \frac{\vartheta(\vartheta+\xi)(\vartheta+d+\theta)}{\kappa(\beta\vartheta+\xi\gamma)}$.

The Jacobian matrix $J(E_0,\ \sigma^*)$ of (5.16) obtained at E_0 and σ^* is

$$J(E_0, \sigma^*) = \begin{bmatrix} -\vartheta - \xi & 0 & -\frac{\beta\sigma^*\kappa}{\vartheta+\xi} \\ \xi & -\vartheta & -\frac{\gamma\sigma^*\kappa\xi}{\vartheta(\vartheta+\xi)} \\ 0 & 0 & 0 \end{bmatrix}.$$

The eigenvalues of $J(E_0,\ \sigma^*)$ are $\lambda_1 = -\vartheta - \xi$, $\lambda_2 = -\vartheta$, and $\lambda_3 = 0$. Clearly, λ_1 and λ_2 are negative eigenvalues and λ_3 is a simple zero eigenvalue. Hence, when $R_0 = 1$, the

DFE E_0 is a non-hyperbolic equilibrium.

The right eigenvector $v = (v_1, v_2, v_3)$ corresponding to $\lambda_3 = 0$ of the Jacobian matrix $J(E_0,\ \sigma^*)$ is obtained as

$$\begin{aligned} v_1 &= -\frac{\beta\kappa\sigma^*}{(\vartheta+\xi)^2}, \\ v_2 &= -\frac{\kappa\vartheta\xi\sigma^*(\beta+\gamma)+\gamma\kappa\xi^2\sigma^*}{\vartheta^2(\vartheta+\xi)^2}, \\ v_3 &= 1. \end{aligned}$$

The left eigenvector $w = (w_1, w_2, w_3)$ of the Jacobian matrix $J(E_0,\ \sigma^*)$ corresponding to $\lambda_3 = 0$ is obtained as

$$\begin{aligned} w_1 &= 0, \\ w_2 &= 0, \\ w_3 &= 1. \end{aligned}$$

Let f_k's represent the right-hand side of the system (5.16). The bifurcation cofficients a_1 and b_1 defined in Theorem 4.1 of Castillo –Chavez and Song [40] are given by:

$$\begin{aligned} a_1 &= \sum_{k,i,j=1}^{3} w_k v_i v_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_{E_0}, \\ b_1 &= \sum_{k,i=1}^{3} w_k v_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \sigma^*}\right)_{E_0}. \end{aligned}$$

The non-zero partial derivative associated with the functions f_k's calculated at E_0 are evaluated as

$\left(\frac{\partial^2 f_3}{\partial x_3 \partial x_1}\right)_{E_0} = \beta\sigma^*,\ \left(\frac{\partial^2 f_3}{\partial x_3 \partial x_2}\right)_{E_0} = \gamma\sigma^*,\ \left(\frac{\partial^2 f_3}{\partial x_1 \partial x_3}\right)_{E_0} = \beta\sigma^*,\ \left(\frac{\partial^2 f_3}{\partial x_2 \partial x_3}\right)_{E_0} = \gamma\sigma^*,\ \left(\frac{\partial^2 f_3}{\partial x_3^2}\right)_{E_0} = -2a - 2\beta\sigma^*\left(\frac{\kappa}{\vartheta+\xi}\right) - 2\gamma\sigma^*\left(\frac{\kappa\xi}{\vartheta(\vartheta+\xi)}\right)$, and $\left(\frac{\partial^2 f_3}{\partial x_3 \partial \sigma^*}\right)_{E_0} = \frac{\beta\kappa\vartheta+\gamma\kappa\xi}{\vartheta(\vartheta+\xi)}$.

Thus, the coefficients a_1 and b_1 are computed as

$$\begin{aligned} a_1 &= -\frac{2(a\vartheta^2(\vartheta+\xi)^2+\kappa\sigma^*(\beta^2\ \vartheta^2\sigma^*+\gamma\xi(\vartheta+\xi)(\vartheta+\gamma\ \sigma^*)+\beta\vartheta(\vartheta^2+\vartheta\xi+\gamma\xi\ \sigma^*)))}{\vartheta^2(\vartheta+\xi)^2}, \\ b_1 &= \frac{\beta\kappa\vartheta+\gamma\kappa\xi}{\vartheta(\vartheta+\xi)}. \end{aligned}$$

It can be seen that the bifurcation coefficients $a_1 < 0$, and $b_1 > 0$. Therefore, the bifur-

cation is forward.

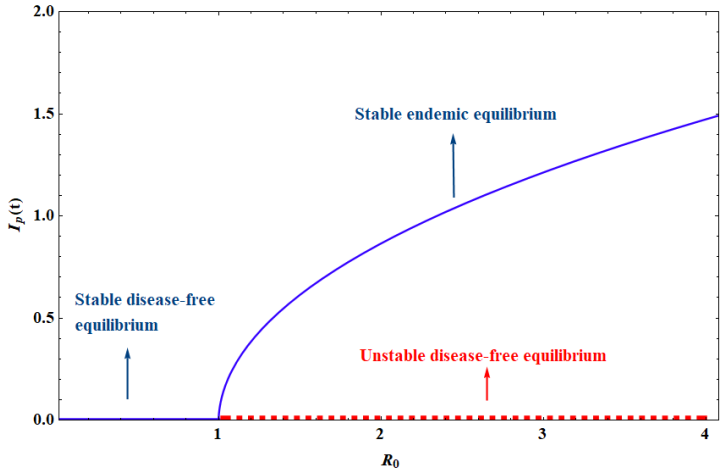


Figure 5.2: $I_p(t)$ vs. R_0, revealing forward bifurcation.

Fig. 5.2 illustrates the forward bifurcation with the following set of parameters: $\kappa = 2$, $\beta = 0.09$, $\xi = 0.12$, $\sigma = 0.08$, $\vartheta = 0.01$, $\gamma = 0.009$, $d = 0.08$, $\theta = 0.03$, $a = 0.005$, $b = 0.01$, $c = 0.03$.. This figure provides the qualitative description of infectives when the basic reproduction number R_0 varies from unity. It shows that when R_0 crosses unity from below, a small positive asymptotically stable endemic equilibrium exists, and E_0 changes its stability from stable to unstable.

In concluding, we state the following theorem.

Theorem 5.4.2. *The disease-free equilibrium E_0 at $R_0 = 1$ of the system (5.6) is linearly nuetrally stable for $\rho > 0$, and when $\rho = 0$, then the undelayed system (5.6) exhibits a forward bifurcation at E_0 and $R_0 = 1$.*

5.4.2 Endemic equilibrium and stability

Now, we determine the conditions for endemic equilibrium existence. For that, assuming that S_p, A_p, $I_p \neq 0$ and setting the system (5.6) to zero, we get:

$$\kappa - \vartheta S_p - \frac{\beta\sigma S_p I_p}{1+I_p} - \xi S_p = 0, \tag{5.17}$$

$$\xi S_p - \vartheta A_p - \frac{\gamma\sigma A_p I_p}{1+I_p} = 0, \tag{5.18}$$

$$\frac{\beta\sigma S_p I_p}{1+I_p} + \frac{\gamma\sigma A_p I_p}{1+I_p} - (\vartheta + d + \theta) I_p - \frac{a I_p^2}{b I_p^2 + c I_p + 1} = 0. \tag{5.19}$$

On obtaining S_p from Eq. (5.17) and A_p from Eq. (5.18), and then substituting it in (5.19), the following biquadratic equation in I_p is obtained:

$$F(I_p) := K_0 + K_1 I_p + K_2 I_p^2 + K_3 I_p^3 + K_4 I_p^4 = 0, \tag{5.20}$$

where,

$$\begin{aligned}
K_0 &= \vartheta(\vartheta+\xi)(d+\vartheta+\theta)(1-R_0),\\
K_1 &= \vartheta(a+(2+c)(d+\theta+\vartheta))(\vartheta+\xi)+(\vartheta(d(\beta+\gamma)+\gamma(\theta+\vartheta)+\beta(\theta-(1+c)\kappa+\vartheta))+\\
&\quad \gamma(d+\theta-(1+c)\kappa+\vartheta)\xi)\sigma-\beta\gamma\kappa\sigma^2,\\
K_2 &= \vartheta(2a+(1+b+2c)(d+\theta+\vartheta))(\vartheta+\xi)+(\vartheta(a(\beta+\gamma)+(1+c)d(\beta+\gamma)+\beta\theta+c\beta\theta+\gamma\theta+\\
&\quad c\gamma\theta-b\beta\kappa-c\beta\kappa+(1+c)(\beta+\gamma)\vartheta)+\gamma(a+(1+c)d+\theta-b\kappa+\vartheta+c(\theta-\kappa+\vartheta))\xi)\sigma+\\
&\quad \beta\gamma(d+\theta-c\kappa+\vartheta)\sigma^2,\\
K_3 &= a(\vartheta+\xi+\beta\sigma)(\vartheta+\gamma\sigma)+c(d+\theta+\vartheta)(\vartheta+\xi+\beta\sigma)(\vartheta+\gamma\sigma)+b(2\vartheta(d+\theta+\vartheta)(\vartheta+\xi)+\\
&\quad (\vartheta(d(\beta+\gamma)+\gamma(\vartheta+\theta)+\beta(\theta+\vartheta-\kappa))+\gamma(\theta+d-\kappa+\vartheta)\xi)\sigma-\beta\gamma\kappa\sigma^2),\\
K_4 &= b(\sigma\gamma+\vartheta)(d+\theta+\vartheta)(\sigma\beta+\vartheta+\xi).
\end{aligned} \tag{5.21}$$

For the positive root I_p^* of polynomial $F(I_p)$, we can make

$$S_p^* = \frac{(1+I_p^*)\kappa}{\sigma I_p^*\beta+(1+I_p^*)(\vartheta+\xi)} > 0. \tag{5.22}$$

and

$$A_p^* = \frac{(1+I_p^*)^2\kappa\xi}{(\sigma I_p^*\gamma+\vartheta+I_p^*\vartheta)(\sigma I_p^*\beta+(1+I_p^*)(\vartheta+\xi))} > 0. \tag{5.23}$$

So, $E_e(S_p^*, A_p^*, I_p^*)$ is an endemic equilibrium of the system (5.6).

Theorem 5.4.3. *When $R_0 > 1$, there is either a unique or three positive endemic equilibria if all equilibria are simple roots.*

Proof. Let $R_0 > 1$. We see that the coefficient K_4 is always positive. On the other hand, $K_0 < 0$ when $R_0 > 1$. From Eq. (5.20), we have a fourth-degree polynomial in I_p, given below:

$$F(I_p) := K_0 + K_1 I_p + K_2 I_p^2 + K_3 I_p^3 + K_4 I_p^4 = 0.$$

The following possibilities for the signs of K_1, K_2, and K_3 exist:

$$\mathbf{U}_1 : K_1 > 0,\ K_2 > 0,\ \text{and } K_3 > 0,$$
$$\mathbf{U}_2 : K_1 < 0,\ K_2 < 0,\ \text{and } K_3 > 0,$$
$$\mathbf{U}_3 : K_1 < 0,\ K_2 > 0,\ \text{and } K_3 > 0,$$
$$\mathbf{U}_4 : K_1 < 0,\ K_2 < 0,\ \text{and } K_3 < 0,$$
$$\mathbf{U}_5 : K_1 > 0,\ K_2 > 0,\ \text{and } K_3 < 0,$$
$$\mathbf{U}_6 : K_1 > 0,\ K_2 < 0,\ \text{and } K_3 > 0,$$
$$\mathbf{U}_7 : K_1 > 0,\ K_2 < 0,\ \text{and } K_3 < 0,$$
$$\mathbf{U}_8 : K_1 < 0,\ K_2 > 0,\ \text{and } K_3 < 0.$$

Using Descartes' rule of signs [42], $F(I_p)$ can have either a unique or three positive roots. If any of the conditions $\mathbf{U}_1$–$\mathbf{U}_4$ holds, then there is unique endemic equilibrium, whereas for the existence of three endemic equilibria, any one of the conditions $\mathbf{U}_5$–$\mathbf{U}_8$ must satisfy. ■

Theorem 5.4.4. *Assume that any of the conditions **H1:** ($\mathbf{U}_1$–$\mathbf{U}_4$, and $R_0 > 1$) hold, then the system (5.6) has a unique endemic equilibrium.*

For the present study, we consider the case of unique endemic equilibrium only. Therefore, we investigate the stability behavior of the unique endemic equilibrium.

At E_e, the characteristic equation of the system (5.6) is obtained as

$$\lambda^3 + L_2\lambda^2 + L_1\lambda + L_0 + \left(M_2\lambda^2 + M_1\lambda + M_0\right)\mathrm{e}^{-\lambda\rho} + (N_1\lambda + N_0)\,\mathrm{e}^{-2\lambda\rho} = 0, \tag{5.24}$$

where,

$$L_2 = d + \frac{aI_p^*(2+cI_p^*)}{(1+I_p^*(c+bI_p^*))^2} + \theta + 3\vartheta + \xi,$$
$$L_1 = 2\theta\vartheta + 3\vartheta^2 + \theta\xi + 2\vartheta\xi + d(2\vartheta+\xi) + \frac{a(2\vartheta+\xi)I_p^*(2+cI_p^*)}{(1+I_p^*(c+bI_p^*))^2},$$
$$L_0 = \vartheta\left(d + \frac{aI_p^*(2+cI_p^*)}{(1+I_p^*(c+bI_p^*))^2} + \theta + \vartheta\right)(\vartheta + \xi),$$
$$M_2 = \frac{(-S_p^*\beta - A_p^*\gamma + I_p^*(1+I_p^*)(\beta+\gamma))\sigma}{(1+I_p^*)^2},$$
$$M_1 = \frac{aI_p^{*2}(2+cI_p^*)(\beta+\gamma)\sigma}{(1+I_p^*)(1+I_p^*(c+bI_p^*))^2} + \frac{I_p^*((\beta+\gamma)(d+\theta+2\vartheta)+\gamma\xi)\sigma}{1+I_p^*} - \frac{(S_p^*\beta + A_p^*\gamma)(2\vartheta+\xi)\sigma}{(1+I_p^*)^2},$$

$$M_0 = \frac{I_p^*(d+\theta+\vartheta)(\beta\vartheta+\gamma(\ \vartheta+\xi))\sigma}{1+I_p^*} + \frac{aI_p^{*2}(2+cI_p^*)(\beta\vartheta+\gamma(\vartheta+\xi))\ \sigma}{(1+I_p^*)(1+I_p^*(c+bI_p^*))^2} - \frac{(S_p^*\beta+A_p^*\gamma)\ \vartheta\ (\vartheta+\xi)\ \sigma}{(1+I_p^*)^2},$$

$$N_1 = \frac{I_p^*(-A_p^*-S_p^*+I_p^*+I_p^{*2})\beta\gamma\sigma^2}{(1+I_p^*)^3},$$

$$N_0 = \frac{I_p^*\beta\gamma(-(A_p^*+S_p^*)\vartheta+I_p^*(1+I_p^*)(d+\theta+\vartheta))\sigma^2}{(1+I_p^*)^3} + \frac{aI_p^{*3}(2+cI_p^*)\beta\gamma\sigma^2}{(1+I_p^*)^2(1+I_p^*(c+bI_p^*))^2}.$$

Multiplying Eq. (5.24) by $\mathrm{e}^{\lambda\rho}$, we get

$$M_2\lambda^2+M_1\lambda+M_0+\left(\lambda^3+L_2\lambda^2+L_1\lambda+L_0\right)\mathrm{e}^{\lambda\rho}+(N_1\lambda+N_0)\,\mathrm{e}^{-\lambda\rho}=0. \tag{5.25}$$

When $\rho=0$, Eq. (5.25) becomes

$$\lambda^3+L_{00}\lambda^2+L_{01}\lambda+L_{02}=0, \tag{5.26}$$

where,

$$L_{00}=M_2+L_2,$$

$$L_{01}=M_1+L_1+N_1,$$

$$L_{02}=M_0+L_0+N_0.$$

Using the Routh-Hurwitz criterion, Eq. (5.26) has all the roots with negative real parts under the following inequalities:

$$\text{H2:}\quad L_{00}>0,\quad L_{02}>0,\quad \text{and}\quad L_{00}L_{01}>L_{02}. \tag{5.27}$$

Hence, we state the following Theorem:

Theorem 5.4.5. *Assume that **H2** holds. Then, the endemic equilibrium E_e is locally asmptotically stable at $\rho=0$.*

Now, for $\rho>0$, let $\mathrm{i}\omega$ $(\omega>0)$ be root of Eq. (5.25). Then, replacing λ with $\mathrm{i}\omega$ $(\omega>0)$ in Eq. (5.25) and splitting real part and imaginary part, we obtain

$$B_1(\omega)\cos\omega\rho-B_2(\omega)\sin\omega\rho=B_3(\omega), \tag{5.28}$$

$$B_4(\omega)\sin\omega\rho+B_5(\omega)\cos\omega\rho=B_6(\omega). \tag{5.29}$$

where,

$$\begin{aligned} B_1(\omega) &= -L_2\omega^2 + L_0 + N_0, \\ B_2(\omega) &= \omega(L_1 - N_1) - \omega^3, \\ B_3(\omega) &= M_2\omega^2 - M_0, \\ B_4(\omega) &= -L_2\omega^2 + L_0 - N_0, \\ B_5(\omega) &= (L_1 + N_1)\omega - \omega^3, \\ B_6(\omega) &= -M_1\omega. \end{aligned} \tag{5.30}$$

From Eqs. (6.48) and (5.29), we obtain

$$\sin\omega\rho = \frac{B_{01}(\omega)}{B_{00}(\omega)}, \tag{5.31}$$

$$\cos\omega\rho = \frac{B_{02}(\omega)}{B_{00}(\omega)}, \tag{5.32}$$

where,

$$\begin{aligned} B_{00} &= L_0^2 - N_0^2 + (L_1^2 - 2L_0L_2 - N_1^2)\omega^2 + (-2L_1 + L_2^2)\omega^4 + \omega^6, \\ B_{01} &= (L_1M_0 - L_0M_1 - M_1N_0 + M_0N_1)\omega + (-M_0 + L_2M_1 - L_1M_2 - M_2N_1)\omega^3 + M_2\omega^5, \\ B_{02} &= -L_0M_0 + M_0N_0 + (L_2M_0 - L_1M_1 + L_0M_2 - M_2N_0 + M_1N_1)\omega^2 + (M_1 - L_2M_2)\omega^4. \end{aligned} \tag{5.33}$$

On squaring Eqs. (5.31) and (5.32), and then adding, we obtain

$$B_{01}^2(\omega) + B_{02}^2(\omega) - B_{00}^2(\omega) = 0. \tag{5.34}$$

Suppose that (H3:) Eq. (5.34) at least one positive root ω_0, i.e., Eq. (5.25) has a pair of purely imaginary roots $\pm i\omega_0$.

For ω_0, we obtain the corresponding critical value of the time delay ρ_k as follows:

$$\rho_k = \frac{1}{\omega_0}\arccos\left(\frac{B_{02}(\omega_0)}{B_{00}(\omega_0)}\right) + \frac{2k\pi}{\omega_0}, \qquad k = 0,1,2,3,\ldots. \tag{5.35}$$

To estabish Hopf bifurcation, we must have $Re\left[\frac{d\lambda}{d\rho}\right]^{-1}\Big|_{\lambda=i\omega_0} \neq 0$.

On differentiating Eq. (5.25) with respect to ρ, we get

$$\left[\frac{d\lambda}{d\rho}\right]^{-1} = \frac{2\lambda M_2 + M_1 + N_1 e^{-\lambda\rho} + e^{\lambda\rho}(3\lambda^2 + 2\lambda L_2 + L_1)}{\lambda\left((N_1\lambda + N_0)e^{-\lambda\rho} - (\lambda^3 + L_2\lambda^2 + L_1\lambda + L_0)\right)e^{\lambda\rho}} - \frac{\rho}{\lambda}.$$

From Eq. (5.25), we have

$$-(\lambda^3 + L_2\lambda^2 + L_1\lambda + L_0)e^{\lambda\rho} = M_2\lambda^2 + M_1\lambda + M_0 + (N_1\lambda + N_0)\,e^{-\lambda\rho}.$$

Thus, we obtain

$$\left[\frac{d\lambda}{d\rho}\right]^{-1} = \frac{2\lambda M_2 + M_1 + N_1 e^{-\lambda\rho} + e^{\lambda\rho}(3\lambda^2 + 2\lambda L_2 + L_1)}{\lambda\left(2(N_1\lambda + N_0)e^{-\lambda\rho} + (M_2\lambda^2 + M_1\lambda + M_0)\right)} - \frac{\rho}{\lambda}.$$

So,

$$Re\left[\frac{d\lambda}{d\rho}\right]^{-1}\Big|_{\lambda=i\omega_0} = \frac{Y}{P^2+Q^2},$$

where,

$$\begin{aligned} Y =& \omega_0(-M_1^2\omega_0 + 2M_0M_2\omega_0 - 2N_1^2\omega_0 - 2M_2^2\omega_0^3 + \omega_0(-L_1M_1 + 4M_2N_0 - 3M_1N_1 + 3M_1\omega_0^2 + \\ & 2L_2(M_0 - M_2\omega_0^2))\cos\rho\omega + 2\omega(2L_2N_0 - L_1N_1 + 3N_1\omega_0^2)\cos 2\rho\omega + (-M_0N_1 - 3M_0\omega_0^2 + \\ & 5M_2N_1\omega_0^2 + 3M_2\omega_0^4 + 2M_1(N_0 + L_2\omega_0^2) + L_1(M_0 - M_2\omega_0^2))\sin\rho\omega + 2(L_1N_0 + \\ & (-3N_0 + 2L_2N_1)\omega_0^2)\sin 2\rho\omega), \end{aligned}$$

$$P = -M_1\omega_0^2 - 2N_1\omega_0^2\cos\rho\omega_0 + 2N_0\omega_0\sin\rho\omega_0,$$

$$Q = M_0\omega_0 - M_2\omega_0^3 + 2N_0\omega_0\cos\rho\omega_0 + 2N_1\omega_0^2\sin\rho\omega_0.$$

Obviously, if H4: $Y \neq 0$, then $Re\left[\frac{d\lambda}{d\rho}\right]^{-1}\Big|_{\lambda=i\omega_0} \neq 0$.

Thus, the following theorem is stated:

Theorem 5.4.6. *Suppose **(H1-H4)** holds. Then, the endemic equilibrium $E_e(S_p^*, A_p^*, I_p^*)$ of the system (5.6) is*

1. *locally asmptotically stable when $\rho \in [0, \rho_0)$,*
2. *undergoes a Hopf bifurcation when $\rho = \rho_0$, and a family of periodic solutions bifurcate from $E_e(S_p^*, A_p^*, I_p^*)$.*

5.4.3 Global stability

In this subsection, we establish the global stability of the DFE and EE.

Global stability of disease-free equilibrium

We now investigate the global stability behavior of DFE $E_0(S_0,A_0,I_0) = E_0\left(\frac{\kappa}{\vartheta+\xi}, \frac{\kappa\xi}{\vartheta(\vartheta+\xi)}, 0\right)$ for $R_0 \leq 1$ by constructing a suitable Lyapunov function. For this, the following postulates are proposed:

P4. $\Psi'_{I_p}(S_p(t),0)$ is increasing for $S_p(t) > 0$ and $\Lambda'_{I_p}(A_p(t),0)$ is increasing for $A_p(t) > 0$.

P5. $\frac{\Psi'_{I_p}(S_0,0)}{\Psi'_{I_p}(S_p(t),0)} < 1$ for $S_p(t) > S_0$, $\frac{\Psi'_{I_p}(S_0,0)}{\Psi'_{I_p}(S_p(t),0)} > 1$ for $S_p(t) \in (0,S_0)$,
$\frac{\Lambda'_{I_p}(A_0,0)}{\Lambda'_{I_p}(A_p(t),0)} < 1$ for $A_p(t) > A_0$, $\frac{\Lambda'_{I_p}(S_0,0)}{\Lambda'_{I_p}(A_p(t),0)} > 1$ for $A_p(t) \in (0,A_0)$.

P6. $\Phi(S_p(t),I_p(t)) + \Lambda(A_p(t),I_p(t)) \leq I_p(t)\left(\left(\frac{\partial\Phi(S_p(t),I_p(t))}{\partial I_p}\right)_{(S_0,0)} + \left(\frac{\partial\Lambda(A_p(t),I_p(t))}{\partial I_p}\right)\right)_{(A_0,0)} - \left(\frac{\partial H(I_p)}{\partial I_p}\right)_{I_p=0} + H(I_p(t))$, where $H(I_p(t)) = (\vartheta + d + \zeta)I_p(t) + h(I_p(t))$ and $I_p(t) > 0$.

P7. $\frac{S_p}{S_0} > \frac{A_p}{A_0}$ if $\frac{S_p}{S_0} > 1$ and $\frac{S_p}{S_0} < \frac{A_p}{A_0}$ if $\frac{S_p}{S_0} < 1$.

Under these postulates, the following theorem is proposed:

Theorem 5.4.7. *Suppose that (P1.)–(P7.) and $R_0 \leq 1$ hold. Then, the disease-free equilibrium $E_0(S_0,A_0,0)$ of the system (5.6) is globally asymptotically stable for $\rho \geq 0$.*

Proof. (P1.) and (P2.) establish that $E_0(S_0,A_0,0)$ is the only equilibrium of the system (5.6). We define the following Lyapunov functional

$$V(t) = V_1(t) + V_2(t),$$

where,

$$V_1(t) = S_p(t) + A_p(t) - S_0 - A_0 - \int_{S_0}^{S_p(t)} \lim_{I_p\to 0^+} \frac{\Psi(S_0,I_p(t))}{\Psi(\varepsilon,I_p(t))}\,d\varepsilon - \int_{A_0}^{A_p(t)} \lim_{I_p\to 0^+} \frac{\Lambda(A_0,I_p(t))}{\Lambda(\varepsilon,I_p(t))}\,d\varepsilon + I_p(t),$$

$$V_2(t) = \int_0^{\rho} (\Psi(S_p(t-\rho),I_p(t-\rho)) + \Lambda(A_p(t-\rho),I_p(t-\rho)))\,d\rho.$$

Using the postulates (P1.)–(P3.), it follows that $V_1(t)$ is well-defined and continuously differentiable function for all $S_p(t) > 0$, $A_p(t) > 0$, $I_p(t) > 0$, and $V(t) = 0$ at $E_0(S_0,A_0,0)$. Now,

we show that $\frac{\mathrm{d}V(t)}{\mathrm{d}t} \leq 0$ for all $t \geq 0$. First, we compute $\frac{\mathrm{d}V_1(t)}{\mathrm{d}t}$ as follows:

$$\begin{aligned}
\frac{\mathrm{d}V_1(t)}{\mathrm{d}t} &= \left(1 - \lim_{I\to 0^+} \frac{\Psi(S_0, I(t))}{\Psi(S_p, I_p(t))}\right) S_p'(t) + \left(1 - \lim_{I_p\to 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(A_p, I_p(t))}\right) A_p'(t) + I_p'(t) \\
&= \left(1 - \lim_{I_p\to 0^+} \frac{\Psi(S_0, I_p(t))}{\Psi(S_p, I_p(t))}\right) \left(\kappa - \vartheta S_p - \Psi(S_p(t-\rho), I_p(t-\rho)) - \xi S_p\right) \\
&\quad + \left(1 - \lim_{I_p\to 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(A_p, I_p(t))}\right) \left(\xi S_p - \vartheta A_p - \Lambda(A_p(t-\rho) I_p(t-\rho))\right) + \Psi(S_p(t-\rho), I_p(t-\rho)) \\
&\quad + \Lambda(A_p(t-\rho) I_p(t-\rho)) - (\vartheta + d + \theta) I_p - \frac{a I_p^2}{b I_p^2 + c I_p + 1}.
\end{aligned}$$

Since $\kappa - (\vartheta + \xi) S_p = (\vartheta + \xi)(S_0 - S_p)$, thus, we obtain:

$$\begin{aligned}
\frac{\mathrm{d}V_1(t)}{\mathrm{d}t} &= \left(1 - \lim_{I\to 0^+} \frac{\Psi(S_0, I_p(t))}{\Psi(S_p, I_p(t))}\right) \left((\vartheta + \xi)(S_0 - S_p) - \Psi(S_p(t-\rho), I_p(t-\rho))\right) \\
&\quad + \left(1 - \lim_{I_p\to 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(A_p, I_p(t))}\right) \left((\xi S_p - \vartheta A_p) - \Lambda(A_p(t-\rho) I_p(t-\rho))\right) + \Psi(S_p(t-\rho), I_p(t-\rho)) \\
&\quad + \Lambda(A_p(t-\rho) I_p(t-\rho)) - H(I_p(t)).
\end{aligned}$$

We now obtain the derivative of $V_2(t)$ as below:

$$\frac{\mathrm{d}V_2(t)}{\mathrm{d}t} = -\Psi(S_p(t-\rho), I_p(t-\rho)) + \Psi(S_p(t), I_p(t)) - \Lambda(A_p(t-\rho), I_p(t-\rho)) + \Lambda(A_p(t), I_p(t)).$$

Thus, the derivative of $V(t)$ is obtained as:

$$\begin{aligned}
\frac{\mathrm{d}V(t)}{\mathrm{d}t} &= \frac{\mathrm{d}V_1(t)}{\mathrm{d}t} + \frac{\mathrm{d}V_2(t)}{\mathrm{d}t} \\
&= \left(1 - \lim_{I_p\to 0^+} \frac{\Psi(S_0, I_p(t))}{\Psi(S_p, I_p(t))}\right) \left((\vartheta + \xi)(S_0 - S_p) - \Psi(S_p(t-\rho), I_p(t-\rho))\right) \\
&\quad + \left(1 - \lim_{I_p\to 0^+} \frac{\Lambda(A_0, I_p(t))}{\Lambda(A_p, I_p(t))}\right) \left((\xi S_p - \vartheta A_p) - \Lambda(A_p(t-\rho) I_p(t-\rho))\right) + \Psi(S_p(t-\rho), I_p(t-\rho)) \\
&\quad + \Lambda(A_p(t-\rho) I_p(t-\rho)) - H(I_p(t)) - \Psi(S_p(t-\rho), I_p(t-\rho)) + \Psi(S_p(t), I_p(t)) \\
&\quad - \Lambda(A_p(t-\rho), I_p(t-\rho)) + \Lambda(A_p(t), I_p(t)).
\end{aligned}$$

The postulates P4–P6 imply that

$$\begin{aligned}
\frac{\mathrm{d}V(t)}{\mathrm{d}t} &\leq (\vartheta+\xi)\left(1-\frac{\Psi'_{I_p}(S_0,0)}{\Psi'_{I_p}(S_p(t),0)}\right)(S_0-S_p)+\Psi(S_p(t-\rho),I_p(t-\rho))\left(\frac{\Psi'_{I_p}(S_0,0)}{\Psi'_{I_p}(S_p(t),0)}-1\right)\\
&\quad+\left(1-\frac{\Lambda'_{I_p}(A_0,0)}{\Lambda'_{I_p}(A_p(t),0)}\right)(\xi S_p-\vartheta A_p)+\Lambda(A_p(t-\rho),I_p(t-\rho))\left(\frac{\Lambda'_{I_p}(A_0,0)}{\Lambda'_{I_p}(A_p(t),0)}-1\right)\\
&\quad+\frac{I_p(t)}{\vartheta+d+\theta}(R_0-1)\\
&=\vartheta\left(1-\frac{\Psi'_{I_p}(S_0,0)}{\Psi'_{I_p}(S_p(t),0)}\right)(S_0-S_p)+\xi S_0\left(1-\frac{S_p}{S_0}\right)\frac{A_0}{A_p}\frac{S_0}{S_p}\left(\frac{S_p}{S_0}-\frac{A_p}{A_0}\right)\\
&\quad+\Psi(S_p(t-\rho),I_p(t-\rho))\left(\frac{\Psi'_{I_p}(S_0,0)}{\Psi'_{I_p}(S_p(t),0)}-1\right)+\vartheta(A_0-A_p)\left(1-\frac{\Lambda'_{I_p}(A_0,0)}{\Lambda'_{I_p}(A_p(t),0)}\right)\\
&\quad+\Lambda(A_p(t-\rho),I_p(t-\rho))\left(\frac{\Lambda'_{I_p}(A_0,0)}{\Lambda'_{I_p}(A_p(t),0)}-1\right)+\frac{I_p(t)}{\vartheta+d+\theta}(R_0-1).
\end{aligned}$$

Thus, $R_0\leq 1$ implies that $\frac{\mathrm{d}V(t)}{\mathrm{d}t}\leq 0$ for all $t\geq 0$. Also, $\frac{\mathrm{d}V(t)}{\mathrm{d}t}=0$ if $S_p(t)=S_0,\ A_p(t)=A_0,$ and $I_p(t)=0$.

Hence, from the system (5.6), it follows that the largest invariant set $\left\{\left(S_p(t),A_p(t),I_p(t)\in R_+^3\middle|\frac{\mathrm{d}V(t)}{\mathrm{d}t}=0\right)\right\}$ is singleton set $\{E_0\}$. Using the Lyapunov-LaSalle asymptotic stability theorem [23, 90, 113], E_0 is the only equilibrium of the system (5.6) and it is globally asymptotically stable. ■

Global stability of endemic equilibrium

Now, we investigate the global stability of $E_e(S^*,A^*,I^*)$ of the system (5.6) by constructing a Lyapunov functional and employing the Lyapunov direct method. To proceed, we propose the following postulates:

P7. $\left(\frac{\Phi(S_p^*,I_p^*)}{\Phi(S_p(t),I_p^*)}-\frac{I_p^*}{I_p(t)}\right)\leq 0;\ \left(\frac{\Phi(S_p(t),I_p(t))}{\Phi(S_p^*,I_p^*)}-1\right)\leq 0;\ \left(\frac{\Phi(S_p(t),I_p^*)}{\Phi(S_p(t),I_p(t))}-\frac{I_p(t)}{I_p^*}\right)\leq 0;$
$\left(\frac{\Lambda(A_p^*,I_p^*)}{\Lambda(A_p(t),I_p^*)}-\frac{I_p^*}{I_p(t)}\right)\leq 0;\ \left(\frac{\Lambda(A_p(t),I_p(t))}{\Lambda(A_p^*,I_p^*)}-1\right)\leq 0;\ \left(\frac{\Lambda(A_p(t),I_p^*)}{\Lambda(A_p(t),I_p(t))}-\frac{I_p(t)}{I_p^*}\right)\leq 0$ for $I_p\geq I_p^*$.

P8. $\left(\frac{h(I_p^*)}{h(I_p(t))}-\frac{I_p^*}{I_p(t)}\right)\left(\frac{I_p(t)}{I^*}-1\right)\leq 0$ for $I_p\geq I_p^*$.

P9. $\frac{S_p}{S_p^*}-\frac{A_p}{A_p^*}>0$ for $\frac{S_p}{S_p^*}>1$ and $\frac{S_p}{S_p^*}-\frac{A_p}{A_p^*}<0$ for $\frac{S_p}{S_p^*}<1$.

The following theorem is proposed under these postulates:

Theorem 5.4.8. *Suppose that (P1.)–(P3.), (P7.)–(P9.), and $R_0>1$ hold. Then, the endemic equilibrium $E_e(S_p^*,A_p^*,I_p^*)$ of the system (5.6) is globally asymptotically stable for $\rho\geq 0$.*

Proof. We define the following Lyapunov functional

$$W(t) = W_1(t) + W_2(t),$$

where,

$$W_1(t) = S_p(t) - S_p^* - \int_{S_p^*}^{S_p(t)} \frac{\Psi(S_p^*, I_p^*)}{\Psi(\phi, I_p^*)}\,\mathrm{d}\phi + A_p(t) - A_p^* - \int_{A_p^*}^{A_p(t)} \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(\Lambda, I_p^*)}\,\mathrm{d}\phi + I_p(t) - I_p^* - I_p^* \ln \frac{I_p(t)}{I_p^*},$$

$$W_2(t) = \Phi(S_p^*, I_p^*) \int_0^\rho \left(\frac{\Phi(S_p(t-\theta), I_p(t-\theta))}{\Phi(S_p^*, I_p^*)} - 1 - \ln \frac{\Phi(S_p(t-\theta), I_p(t-\theta))}{\Phi(S_p^*, I^*)} \right) \mathrm{d}\theta$$
$$+ \Lambda(A_p^*, I_p^*) \int_0^\rho \left(\frac{\Lambda(A_p(t-\theta), I_p(t-\theta))}{\Lambda(A_p^*, I_p^*)} - 1 - \ln \frac{\Lambda(A_p(t-\theta), I_p(t-\theta))}{\Lambda(A_p^*, I_p^*)} \right) \mathrm{d}\theta.$$

(P1.)–(P3.) imply that, $W(t) = W_1(t) + W_2(t)$ is defined and continuously differentiable for all $S_p(t),\ A_p(t),\ I_p(t) > 0$ and $W(0) = 0$ at $E_e(S_p^*, A_p^*, I_p^*)$.

The derivative of $W_1(t)$ with respect to time t is computed as

$$\frac{\mathrm{d}W_1(t)}{dt} = \left(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}\right) S_p'(t) + \left(1 - \frac{\Lambda(A_p^*, I^*)}{\Lambda(A_p(t), I_p^*)}\right) A_p'(t) + \left(1 - \frac{I_p^*}{I_p(t)}\right) I_p'(t)$$

$$= \left(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}\right) \left((\vartheta + \xi)(S_p^* - S_p(t)) + \Psi(S_p^*, I_p^*) - \Psi(S_p(t-\rho), I_p(t-\rho))\right)$$
$$+ \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) (\xi S_p - \vartheta A_p - \Lambda(A_p(t-\rho), I_p(t-\rho))) + \left(1 - \frac{I_p^*}{I_p(t)}\right)$$
$$\times \Big(\Psi(S_p(t-\rho), I_p(t-\rho)) + \Lambda(A_p(t-\rho), I_p(t-\rho)) - \frac{\left(\Psi(S_p^*, I_p^*) + \Lambda(A_p^*, I_p^*) - h(I_p^*)\right)}{I_p^*} I_p(t)$$
$$- h(I_p(t))\Big)$$

$$= (\vartheta + \xi) \left(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}\right) (S_p^* - S_p(t)) + \Psi(S_p^*, I_p^*) \Big(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}$$
$$+ \frac{\Psi(S_p(t-\rho), I_p(t-\rho))}{\Psi(S_p(t), I_p^*)}\Big) + (\xi S_p - \vartheta A_p) \left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) - \Lambda(A_p(t-\rho), I_p(t-\rho)) \frac{I_p^*}{I_p(t)}$$
$$+ \frac{\Lambda(A_p(t-\rho), I_p(t-\rho))}{\Lambda(A_p(t), I_p^*)} \Lambda(A_p^*, I_p^*) - \frac{I_p^*}{I_p(t)} \Psi(S_p(t-\rho), I_p(t-\rho)) + \Psi(S_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*}\right)$$
$$+ \Lambda(A_p^*, I_p^*) \left(1 - \frac{I_p}{I_p^*}\right) + \left(h(I_p^*) - \frac{h(I_p(t) I_p^*)}{I_p(t)}\right) \left(\frac{I_p(t)}{I_p^*} - 1\right).$$

Since $\xi S_p^* - \vartheta A_p^* - \Lambda(A_p^*, I_p^*) = 0$, or, $-\xi S_p^* + \vartheta A_p^* + \Lambda(A_p^*, I_p^*) = 0$, thus we have

$$\frac{\mathrm{d}W_1(t)}{dt} = (\vartheta + \xi)\left(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}\right)(S_p^* - S_p(t)) + \Psi(S_p^*, I_p^*)\big(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)} + \frac{\Psi(S_p(t\rho), I_p(t-\rho))}{\Psi(S_p(t), I_p^*)}\big) - \frac{I_p^*}{I_p(t)}\Psi(S_p(t-\rho), I_p(t-\rho)) + \Psi(S_p^*, I_p^*)\left(1 - \frac{I_p}{I_p^*}\right) + \xi(S_p - S_p^*)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) - \Lambda(A_p(t-\rho), I_p(t-\rho))\frac{I_p^*}{I_p(t)} + \Lambda(A_p^*, I_p^*)\left(1 - \frac{I_p}{I_p^*}\right) + \vartheta\left(A_p^* - A_p\right)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) + \Lambda(A_p^*, I_p^*)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} + \frac{\Lambda(A_p(t-\rho), I_p(t-\rho))}{\Lambda(A_p(t), I_p^*)}\right).$$

Now, we compute the derivative of $W_2(t)$ as follows:

$$\frac{\mathrm{d}W_2}{\mathrm{d}t} = \Psi(S_p(t), I_p(t)) - \Psi(S_p(t-\rho), I_p(t-\rho)) + \Psi(S_p^*, I_p^*)\ln\frac{\Psi(S_p(t-\rho), I_p(t-\rho))}{\Psi(S_p(t), I_p(t))} + \Lambda(A_p(t), I_p(t)) - \Lambda(A_p(t-\rho), I_p(t-\rho)) + \Lambda(A_p^*, I_p^*)\ln\frac{\Lambda(A_p(t-\rho), I_p(t-\rho))}{\Lambda(S_p(t), I_p(t))}.$$

Thus, the time derivative of $W(t)$ is obtained as follows:

$$\begin{aligned}\frac{\mathrm{d}W}{\mathrm{d}t} &= \frac{\mathrm{d}W_1}{\mathrm{d}t} + \frac{\mathrm{d}W_2}{\mathrm{d}t} \\ &= (\vartheta + \xi)\left(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}\right)(S_p^* - S_p(t)) + \Psi(S_p^*, I_p^*)\Big(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)} + \frac{\Psi(S_p(t-\rho), I_p(t-\rho))}{\Psi(S_p(t), I_p^*)}\Big) - \frac{I_p^*}{I_p(t)}\Psi(S_p(t-\rho), I_p(t-\rho)) + \Psi(S_p^*, I_p^*)\left(1 - \frac{I_p}{I_p^*}\right) + \\ &\quad \xi(S_p - S_p^*)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) - \Lambda(A_p(t-\rho), I_p(t-\rho))\frac{I_p^*}{I_p(t)} + \Lambda(A_p^*, I_p^*)\left(1 - \frac{I_p}{I_p^*}\right) + \\ &\quad \vartheta\left(A_p^* - A_p\right)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) + \Lambda(A_p^*, I_p^*)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} + \frac{\Lambda(A_p(t-\rho), I_p(t-\rho))}{\Lambda(A_p(t), I_p^*)}\right) + \end{aligned}$$

$$\Psi(S_p(t), I_p(t)) - \Psi(S_p(t-\rho), I_p(t-\rho)) + \Psi(S_p^*, I_p^*)\ln\frac{\Psi(S_p(t-\rho), I_p(t-\rho))}{\Psi(S_p(t), I_p(t))} + \Lambda(A_p(t), I_p(t)) - \Lambda(A_p(t-\rho), I_p(t-\rho)) + \Lambda(A_p^*, I_p^*)\ln\frac{\Lambda(A_p(t-\rho), I_p(t-\rho))}{\Lambda(S_p(t), I_p(t))}$$

$$
\begin{aligned}
&= \vartheta(S_p^* - S_p)\left(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}\right) + \vartheta(A_p^* - A_p)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) \\
&+ \Psi(S_p^*, I_p^*)\left(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)} + \ln\frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}\right) + \Psi(S_p^*, I_p^*)\left(1 - \frac{I_p}{I_p^*} + \ln\frac{I_p}{I_p^*}\right) \\
&+ \Lambda(A_p^*, I_p^*)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} + \ln\frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) + \Lambda(A_p^*, I_p^*)\left(1 - \frac{I_p}{I_p^*} + \ln\frac{I_p}{I_p^*}\right) \\
&+ \Psi(S_p^*, I_p^*)\Big(1 - \frac{\Psi(S_p(t-\rho), I_p(t-\rho))}{\Psi(S_p(t), I_p(t))}\frac{\Psi(S_p(t), I_p^*)}{\Psi(S_p^*, I_p^*)}\frac{I_p^*}{I_p(t)} \\
&\quad + \ln\frac{\Psi(S_p(t-\rho), I_p(t-\rho))}{\Psi(S_p(t), I_p(t))}\frac{\Psi(S_p(t), I_p^*)}{\Psi(S_p^*, I_p^*)}\frac{I_p^*}{I_p(t)}\Big) \\
&\quad + \Lambda(A_p^*, I_p^*)\Big(1 - \frac{\Lambda(A_p(t-\rho), I_p(t-\rho))}{\Lambda(A_p(t), I_p(t))}\frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p^*, I_p^*)}\frac{I_p^*}{I_p(t)} \\
&\quad + \ln\frac{\Lambda(A_p(t-\rho), I_p(t-\rho))}{\Lambda(A_p(t), I_p(t))}\frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p^*, I_p^*)}\frac{I_p^*}{I_p(t)}\Big) \\
&+ \Psi(S_p(t-\rho), I_p(t-\rho))\left(\frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)} - \frac{I_p^*}{I_p(t)}\right) + \Psi(S_p^*, I_p^*)\left(\frac{\Psi(S_p(t), I_p(t))}{\Psi(S_p^*, I_p^*)} - 1\right) \\
&+ \Lambda(A_p^*, I_p^*)\left(\frac{\Lambda(A_p(t), I_p(t))}{\Lambda(A_p^*, I_p^*)} - 1\right) + \Psi(S_p(t-\rho), I_p(t-\rho))\frac{I_p^*}{I_p(t)}\left(\frac{\Psi(S_p(t), I_p^*)}{\Psi(S_p(t), I_p(t))} - \frac{I_p(t)}{I_p^*}\right) \\
&+ \Lambda(A_p(t-\rho), I_p(t-\rho))\frac{I_p^*}{I_p(t)}\left(\frac{\Lambda(A_p(t), I_p^*)}{\Lambda(A_p(t), I_p(t))} - \frac{I_p(t)}{I_p^*}\right) + \Lambda(A_p(t-\rho), I_p(t-\rho))\Big(\frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)} \\
&\quad - \frac{I_p^*}{I_p(t)}\Big) + \xi S_p^*\left(1 - \frac{S_p}{S_p^*}\right)\left(\frac{S_p}{S_p^*} - \frac{A_p}{A_p^*}\right)\frac{A_p^*}{A_p}\frac{S_p^*}{S_p} + \left(\frac{h(I_p^*)}{h(I_p(t))} - \frac{I_p^*}{I_p(t)}\right)\left(\frac{I_p(t)}{I_p^*} - 1\right)h(I_p(t)).
\end{aligned}
$$

The functions $\Psi(S_p(t), I_p(t))$ and $\Lambda(A_p(t), I_p(t))$ are monotonically increasing for all $S_p(t) > 0$, and $A_p(t) > 0$. Therefore,

$$
\begin{aligned}
\left(S_p^* - S_p\right)\left(1 - \frac{\Psi(S_p^*, I_p^*)}{\Psi(S_p(t), I_p^*)}\right) &\leq 0, \\
\left(A_p^* - A_p\right)\left(1 - \frac{\Lambda(A_p^*, I_p^*)}{\Lambda(A_p(t), I_p^*)}\right) &\leq 0.
\end{aligned} \tag{5.36}
$$

The function $g(y) = 1 - y + \ln y$, $(y > 0)$ has global maximum at $y = 1$. Henceforth, for $y > 0$, $g(y) \leq 0$ and the resulting inequalities are as follows:

$$\left(1-\frac{\Psi(S_p^*,I_p^*)}{\Psi(S_p(t),I_p^*)}+\ln\frac{\Psi(S_p^*,I_p^*)}{\Psi(S_p(t),I_p^*)}\right)\leq 0,$$

$$\left(1-\frac{I_p}{I_p^*}+\ln\frac{I_p}{I_p^*}\right)\leq 0,$$

$$\left(1-\frac{\Lambda(A_p^*,I_p^*)}{\Lambda(A_p(t),I_p^*)}+\ln\frac{\Lambda(A_p^*,I_p^*)}{\Lambda(A_p(t),I_p^*)}\right)\leq 0,$$

$$\left(1-\frac{\Psi(S_p(t-\rho),I_p(t-\rho))}{\Psi(S_p(t),I_p(t))}\frac{\Psi(S_p(t),I_p^*)}{\Psi(S_p^*,I_p^*)}\frac{I_p^*}{I_p(t)}+\ln\frac{\Psi(S_p(t-\rho),I_p(t-\rho))}{\Psi(S_p(t),I_p(t))}\frac{\Psi(S_p(t),I_p^*)}{\Psi(S_p^*,I_p^*)}\frac{I_p^*}{I_p(t)}\right)\leq 0,$$

$$\left(1-\frac{\Lambda(A_p(t-\rho),I_p(t-\rho))}{\Lambda(A_p(t),I_p(t))}\frac{\Lambda(A_p(t),I_p^*)}{\Lambda(A_p^*,I_p^*)}\frac{I_p^*}{I_p(t)}+\ln\frac{\Lambda(A_p(t-\rho),I_p(t-\rho))}{\Lambda(A_p(t),I_p(t))}\frac{\Lambda(A_p(t),I_p^*)}{\Lambda(A_p^*,I_p^*)}\frac{I_p^*}{I_p(t)}\right)\leq 0. \tag{5.37}$$

Thus, using (P7.)–(P9.) and the inequalities (5.36)–(5.37), it follows that $\frac{dW(t)}{dt}\leq 0$ for all $S_p(t)\geq 0$, $A_p(t)\geq 0$, $I_p(t)\geq 0$. Thus, it is easy to verify that the largest invariant set in $\left\{(S_p(t),A_p(t),I_p(t))\in R_+^3:\frac{dW(t)}{dt}=0\right\}$ is singleton $\{E_e\}$. Hence, by the Lyapunov-LaSalle asymptotic stability theorem [23, 90, 113], the endemic equilibrium E_e is globally asymptotically stable. ■

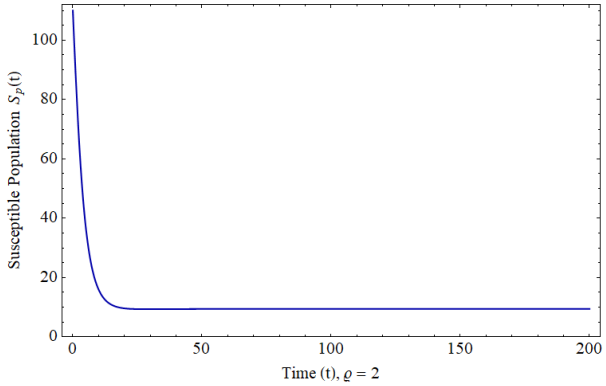
5.5 Numerical simulation

This section presents the numerical results to show the significance of the analytical findings and varying parameters.

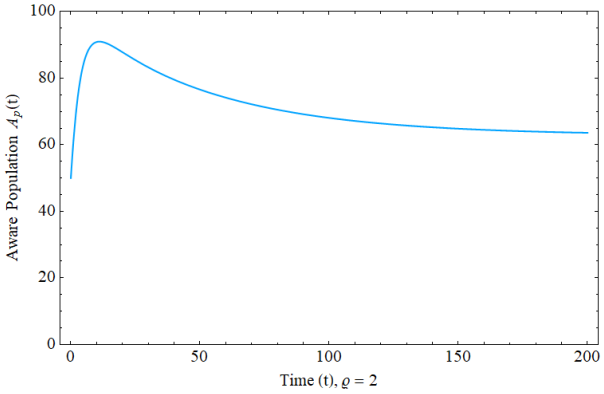
Example 5.1. We use the following set of experimental data: $\kappa=2$, $\beta=0.09$, $\xi=0.12$, $\sigma=1$, $\vartheta=0.01$, $\gamma=0.009$, $d=0.08$, $\theta=0.03$, $a=0.005$, $b=0.01$, $c=0.03$. At these values of parameters, the endemic equilibrium of the system (5.6) is $E_e(9.47806,\ 62.8333,\ 9.01515)$ and the basic reproduction number is $R_0=25.3846>1$.

Fig. 5.3 shows the qualitative behavior of all the subpopulations for a time delay $\rho=1$. Fig. 5.3.1 shows that the susceptible population decreases over time and settles down to their steady-state after a fixed time. Fig. 5.3.2 shows that initially, a large population gets aware of the disease, and as time passes, they become less serious and finally settle down to a steady state. Fig. 5.3.3 elucidates that initially, infectives increase at a high rate and then start decreasing and eventually reach their steady-state. Finally, Fig. 5.3.4 shows the behavior of recovered individuals. Infected individuals recover at high speed and stabilize to their steady

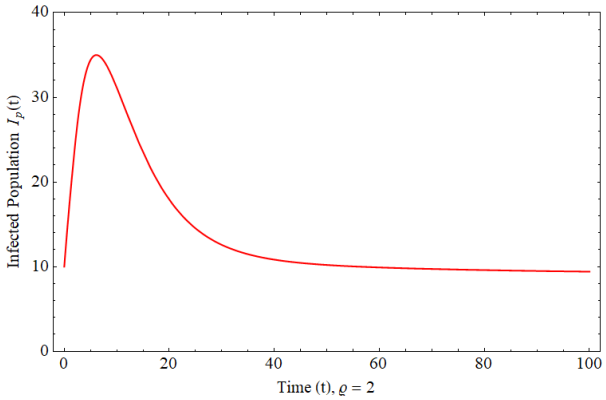
state over time.



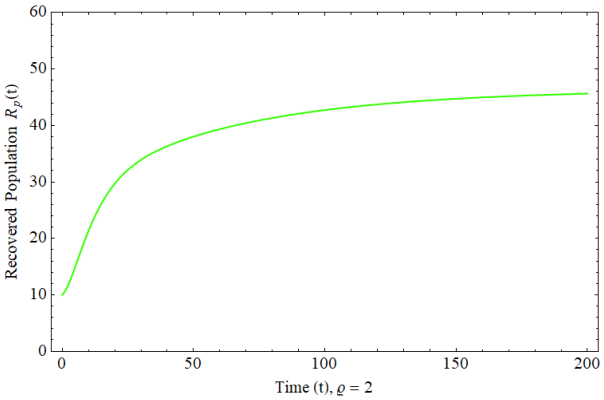
5.3.1: Susceptible population $S_p(t)$.



5.3.2: Aware population $A_p(t)$.



5.3.3: Infected population $I_p(t)$.



5.3.4: Recovered population $R_p(t)$.

Figure 5.3: The temporal behavior of subpopulations at time-delay $\rho = 1$.

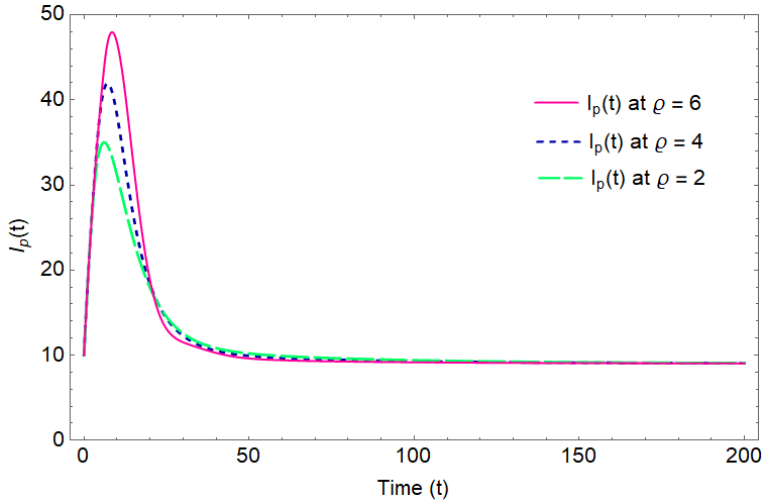
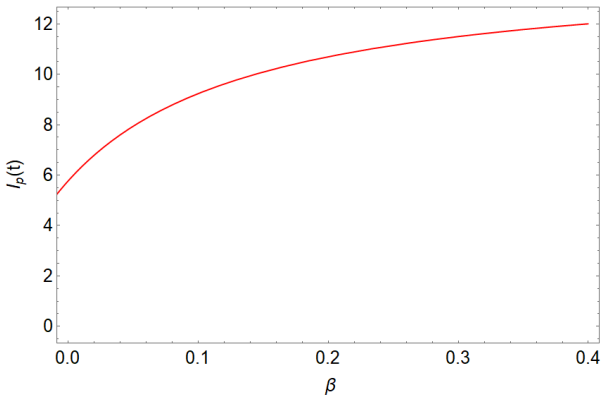
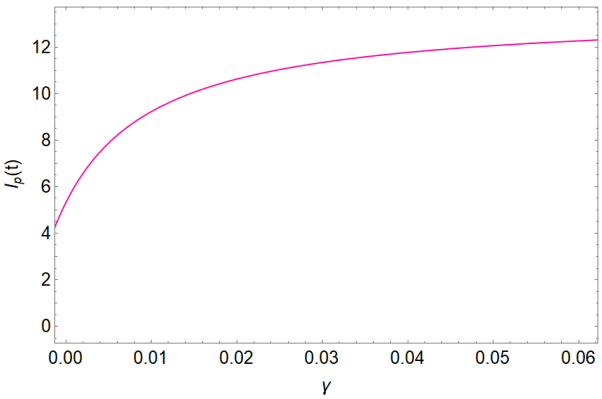


Figure 5.4: $I_p(t)$ for various values of time-delay ρ.

Fig. 5.4 indicates the impact of latent period ρ on infected population $I_p(t)$. We can see the variation in the number of infectives for higher values of time delay. This figure confirms that the longer the time delay, the higher the spread of infection, which shows the importance of considering time delay in studying infectious disease's dynamical behavior.



5.5.1: $I_p(t)$ vs. β



5.5.2: $I_p(t)$ vs. γ

Figure 5.5: Influence of transmission rates β and γ, on infecetd population $I_p(t)$ for $\rho = 2$.

Fig. 5.5 shows the influence of transmission rates β of susceptibles and γ of aware individuals on infectives. The higher the transmission rates, the higher the possibility of spreading infection. Therefore, it is imperative to minimize the contact rate of susceptible and aware individuals with infected individuals.

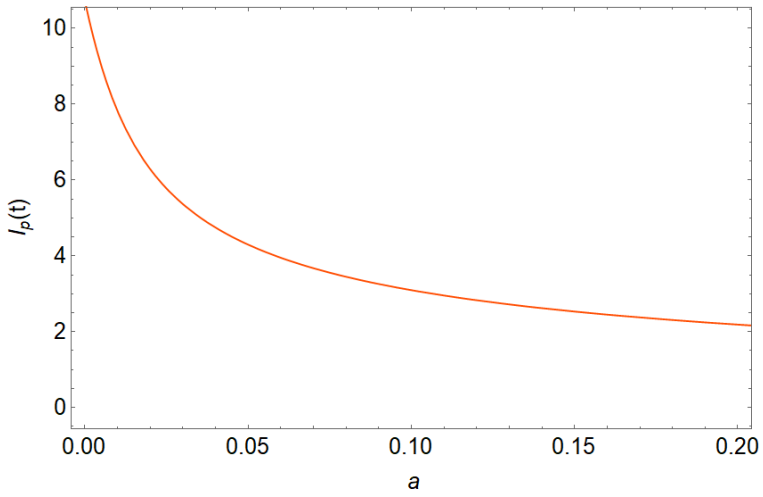


Figure 5.6: Impact of cure rate (a) on infected individuals ($I_p(t)$).

Fig. 5.6 shows the effect of the cure rate in decreasing the infected population. An increased

cure rate can reduce the infection level efficiently. Thus, the accessibility of treatment resources and adequate treatment is significant in reducing the number of infected individuals.

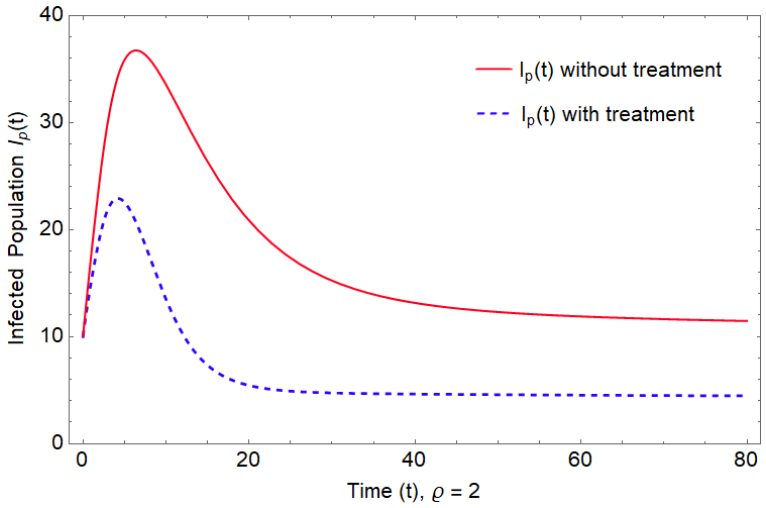


Figure 5.7: Effect of saturating treatment rate $H(I_p(t))$ on infected population $I_p(t)$.

Fig. 5.7 demonstrates the impact of nonlinear saturating treatment rate on infected individuals. The infected population is being drawn for the cure rate $a = 0.05$, revealing the significance of the availability of treatment to infected people. If the health system has sufficient treatment facilities, then the spread of infection can be controlled on a large scale.

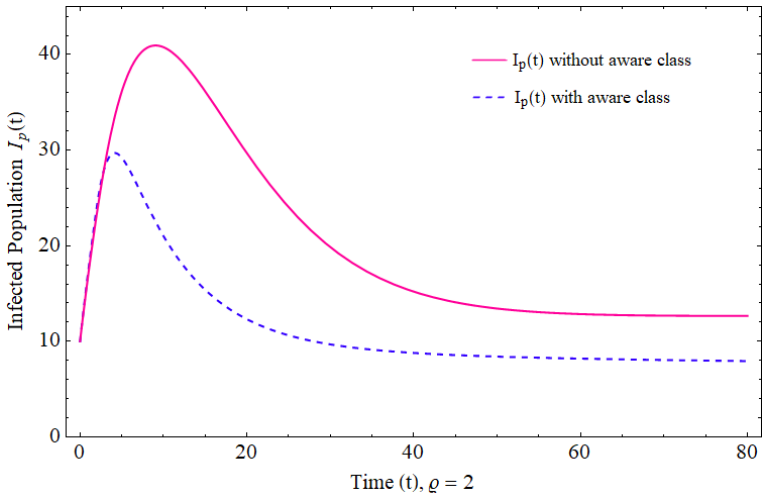


Figure 5.8: Awareness effects on $I_p(t)$ for $\rho = 2$, .

Fig. 5.8 depicts awareness effects on the SIR epidemic model. It shows the difference between the number of infected individuals with and without aware individuals' classes, deliberating that unaware individuals are becoming infected faster than those familiar with the disease spread. It shows the relationship between human awareness and the spread of infection. The graph of infected individuals with awareness class is drawn for the awareness rate $\xi = 0.6$,

revealing that a considerable value of awareness rate causes more individuals to be safe from illnesses. Awareness makes them alert about the spread of disease and helps them take necessary protection measures, reducing infection. It is worth saying that public awareness plays a significant role in making people alert of the disease's risk and reducing its spread.

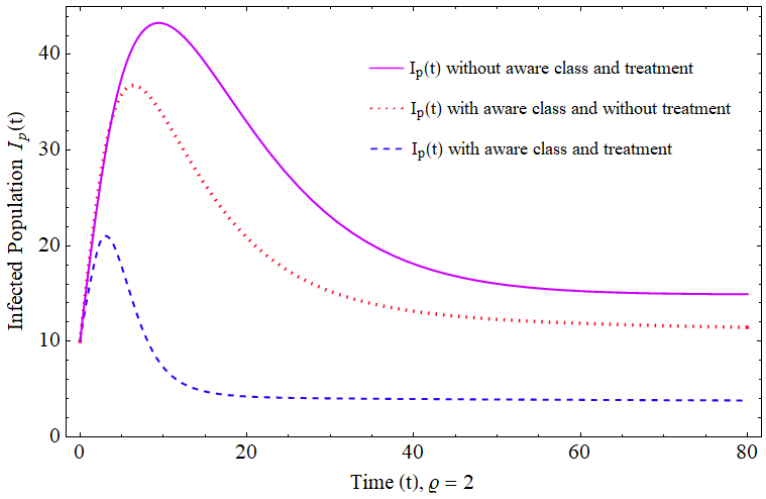
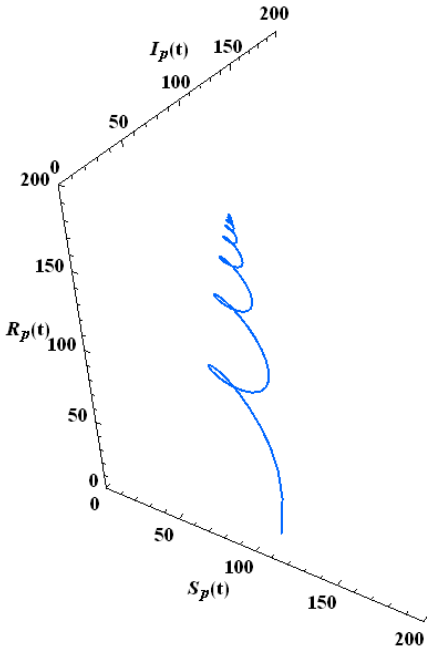


Figure 5.9: Effect of Awareness class $A_p(t)$ and saturated treatment rate on $I_p(t)$ for $\rho = 2$.

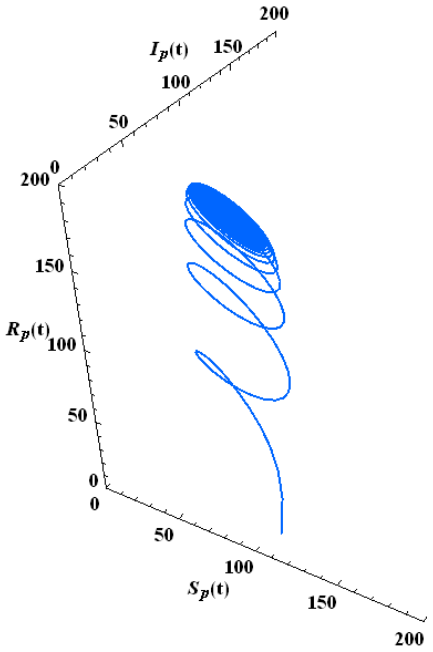
Fig. 5.9 depicts the number of the infected population for the cases: neither awareness nor treatment is available (shown by the solid purple line); people are aware of the disease, but treatment to infectives is not available (indicated by the dotted red line); and when both treatment and awareness are present (indicated by the dashed blue line). This figure captures the impact of both awareness and treatment on infectives. When awareness and treatment are absent, the infected population stabilizes at a high level. If the treatment is not available, then the awareness among people reduces the spread of infection with a big difference. For eradicating disease or lowering it at the lowest level, the presence of both awareness among susceptibles and sufficient treatment availability has a vital role.

Example 5.2. The following numerical experimental data is considered to show the occurrence of Hopf bifurcation:

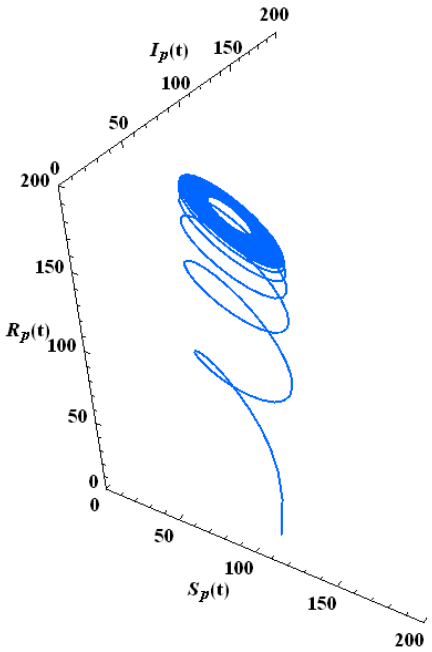
$\kappa = 6.5,\ \xi = 0.001,\ \vartheta = 0.01,\ \beta = 0.009,\ \gamma = 0.0009,\ \sigma = 10.\ d = 0.08,\ \theta = 0.03,\ a = 0.005,\ b = 0.01,\ c = 0.03.$



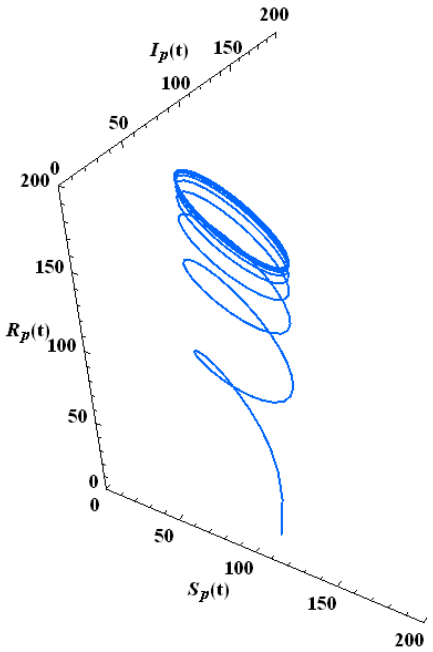
5.10.1: $S_p - I_p - R_p$ at $\rho = 16$.



5.10.2: $S_p - I_p - R_p$ at $\rho = 19.2$.

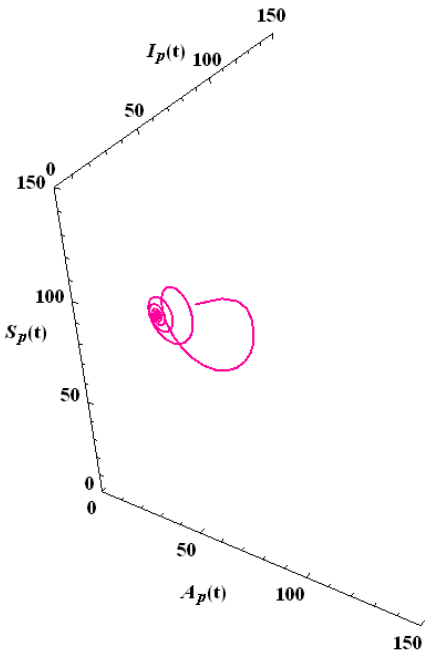


5.10.3: $S_p - I_p - R_p$ at $\rho = 19.5$.

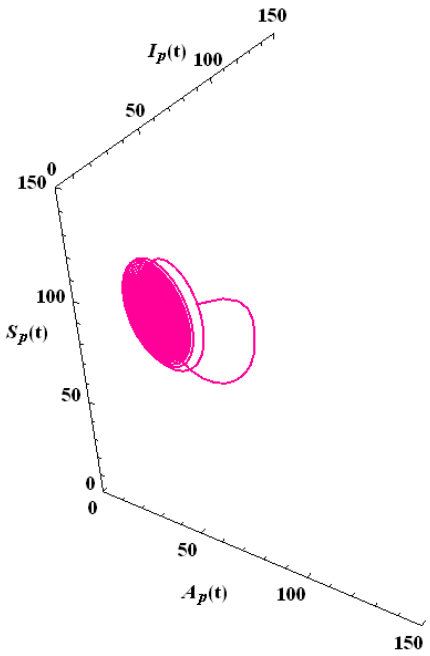


5.10.4: $S_p - I_p - R_p$ at $\rho = 19.6$.

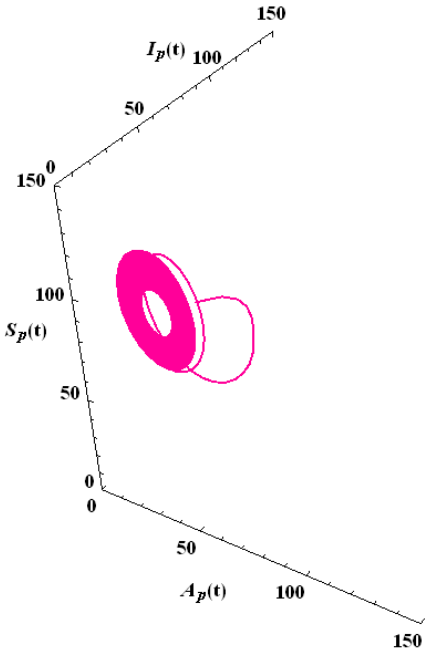
Figure 5.10: The phase portraits of susceptible, infected and recovered individuals.



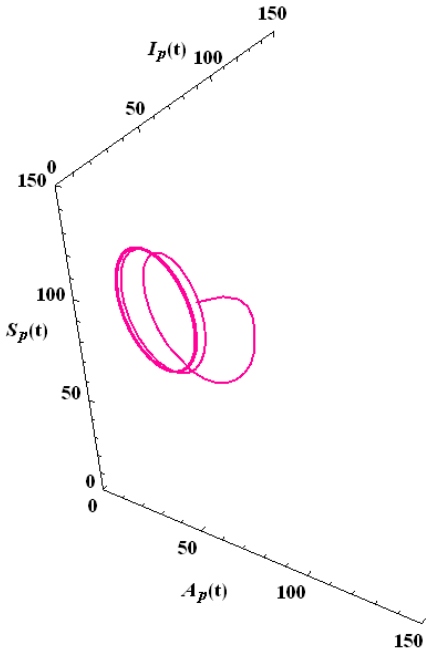
5.11.1: $A_p - S_p - I_p$ at $\rho = 16$.



5.11.2: $A_p - S_p - I_p$ at $\rho = 19.2$.



5.11.3: $A_p - S_p - I_p$ at $\rho = 19.5$.



5.11.4: $A_p - S_p - I_p$ at $\rho = 19.6$.

Figure 5.11: The phase portraits of susceptible-aware-infected individuals.

Figs. 5.10 and 5.11 ensure the presence of Hopf bifurcation. Fig. 5.10 is plotted for susceptible, infected, and recovered classes, whereas Fig. 5.11 is plotted for susceptible, infected, and aware individuals for the time delay $\rho = 16,\ 19.2,\ 19.5$, and 19.6, respectively. These figures show that oscillatory and periodic solutions may appear via Hopf bifurcation, considering time delay as the bifurcation parameter. Figs. 5.10.1, 5.10.2, 5.11.1, and 5.11.2 are plotted for the time delay $\rho = 16$ and 19.2, respectively, revealing that the endemic equilibrium is stable

and the orbit spirals to it. Figs. 5.10.3 and 5.11.3 are plotted for $\rho = 19.5$, whereas 5.10.4 and 5.11.4 are plotted for $\rho = 19.6$. These figures reveal that the orbits spiral go away from endemic equilibrium as time delay ρ crosses its critical value, and the endemic equilibrium becomes unstable. Thus, the time delay can increase the periodicity of the prevalence of the infection, and it may be a foremost aspect in maintaining the oscillation.

5.6 Discussion

During an outbreak, awareness about the transmission routes and interventions of a disease can alert individuals regarding the infection risk, resulting in a change in human behavior and disease transmission patterns. Therefore, the present chapter studies a mathematical epidemic model with awareness effects to study disease transmission and control dynamics. We comprised four dynamical variables in our model: susceptible, aware, infected, and recovered individual; and proposed a nonlinear time-delayed SAIR epidemic model by incorporating Michaelis-Menten type incidences with latent period and a saturating treatment rate. We assume that aware individuals can also become infected, probably at a lower rate than fully vulnerable individuals. We analyze the model mathematically, revealing two equilibria: the disease-free equilibrium (complete eradication of infection) and the endemic equilibrium (persistence of disease). We obtain the model's threshold quantity, the basic reproduction number R_0, and perform the stability analysis to determine whether the disease eliminates or persists. The basic reproduction number determines the potential for an infectious agent to start an outbreak, the degree of transmission without control measures, and the capacity of control measures to diminish spread. The delayed system analysis reveals that the disease-free equilibrium is locally asymptotically stable when R_0 is less than unity, unstable when R_0 is greater than unity, and neutrally linearly stable when R_0 is equal to unity. However, the undelayed system exhibits a forward bifurcation at $R_0 = 1$ using the center manifold theory approach, indicating that reducing the basic reproduction number below unity is sufficient to eradicate society's infection. Further, we investigate the endemic equilibrium's local stability, revealing the existence of oscillatory and periodic solutions near-endemic equilibrium via Hopf bifurcation, regarding time-delay as the bifurcation parameter. Furthermore, the global stability behavior of the disease-free and endemic equilibria is performed using the Lyapunov functionals by employing the Lyapunov direct method. It is shown that the disease-free equilibrium is globally asymptotically

stable when $R_0 < 1$, and the endemic equilibrium is globally asymptotically stable when $R_0 > 1$.

The numerical simulations validate the effectiveness of theoretical findings and show the impact of the model's parameters. It is seen that the higher the time delay, the higher the number of individuals who catch the infection. The oscillatory solutions for various values of time delay establish Hopf bifurcations near-endemic equilibrium. Moreover, if the time delay crosses its critical value, then the trajectories of the solutions bifurcate from endemic equilibrium and destabilize the system at endemic equilibrium. We show that the number of infected individuals is much higher in the SIR model (i.e., without awareness) than the number of infected in the SAIR model (i.e., with awareness). If susceptible individuals are aware of infection risk, they will be on high alert and choose not to go to crowded areas, avoid unnecessary contact with infected individuals, and implement other anti-epidemic inhibition measures which reduce the infection spread effectively. The numerical result shows the impact of saturating treatment rate, which reveals that adequate treatment availability is crucial in controlling infection spread. If the treatment facilities are not enough, individuals' awareness and willingness to adopt anti-epidemic measures are the only way to reduce infection. Together with sufficient treatment facilities for infectives, individuals' awareness can reduce even eradicate the infectious disease from society.

The present study consisting of nonlinear incidences of unaware and aware susceptibles with the latent period, and saturated treatment rate, signifies the substantial role of the latent period in the disease transmission process, susceptibles' behavior in preventing disease spread, and limitation in treatment facilities in curing infectives. The results indicate that awareness about the spread of infection in susceptible individuals is vital in preventing disease transmission and is a potential strategy for controlling the disease spread in the absence of treatment availabilities. The public health authorities and the government have a significant contribution to raising awareness among people and encouraging them to adopt anti-epidemic measures. For example, the government enforced different non-pharmaceutical interventions to obstruct COVID-19 transmission due to the absence of proper therapeutics or vaccines. In addition, several countries focus on raising awareness through media advertising campaigns to encourage people to maintain social distancing, wear a face mask, adopt healthy sanitation practices, frequent hand washing, etc. These behaviors urge people to reduce contact with others and adopt all

possible preventive measures, consequently suppressing disease burden. Thus, to eradicate infectious diseases, programs related to knowledge about the infection spread and its harm to the public and health care workers can improve their general attitude toward it. Awareness about the disease with the encouragement of adopting preventive measures and appropriate treatment facilities to infectives can efficiently reduce disease spread.

Chapter 6

Nonlinear dynamics of a time-delayed epidemic model with two explicit aware classes, saturated incidences, and treatment

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This chapter presents and analyzes a time-delayed epidemic model in which the class of susceptible individuals is divided into three subclasses: unaware susceptibles, fully aware susceptibles, and partially aware susceptibles to the disease, respectively, which emphasizes to consider three explicit incidences. The saturated type of incidence rates and treatment rate of infectives are deliberated herein. The mathematical analysis shows that the model has two equilibria: disease-free and endemic. We derive the basic reproduction number R_0 of the model and study the stability behavior of the model at both disease-free and endemic equilibria. Through analysis, it is demonstrated that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, unstable when $R_0 > 1$, and linearly neutrally stable when $R_0 = 1$ for the time delay $\rho > 0$. Further, an undelayed epidemic model is studied when $R_0 = 1$, which reveals that the model exhibits forward and backward bifurcations under specific conditions, which also has important implications in the study of disease transmission dynamics. Moreover, we investigate the stability behavior of the endemic equilibrium and show that Hopf bifurcation occurs near-endemic equilibrium when we choose time-delay as a bifurcation parameter. Lastly, numerical simulations are performed in support of the analytical results.

6.1 Introduction

The last two decades have seen several large-scale epidemics outbreaks. People get information about these outbreaks quite quickly due to significant advances in social media, which can have an insightful effect on the actual epidemic dynamics [69, 121]. The impact of information and awareness on the spread of epidemics has been studied by many authors [54, 84, 86, 94, 111, 112, 115, 119, 122]. Funk et al. [84] considered the aware susceptible and aware infected populations in their epidemic model, on the assumption that the aware susceptibles will be at a lower rate of catching the infection than the unaware susceptibles, and studied that disease dynamics in a well-mixed population. Kiss et al. [86] studied the effect of information transmission on the dynamics of sexually transmitted diseases. They assumed that the entire population is aware of the risk of infection; however, only a specific portion decides to react by constraining their contact with contaminated people. Misra et al. and Dubey et al. [94, 115] investigated the impact of awareness programs on the transmission dynamics of infectious diseases in their nonlinear epidemic models. Some researchers studied the impact of awareness on the disease transmission dynamics along with the influence of time delay. Zuo et al. [119] introduced a time delay in the media variable to emphasize the delay in reporting cases of infections. Zhao et al. [111] studied the SIRS epidemic model by incorporating time delay in media coverage. Zuo and Liu [112] studied the effect of awareness programs driven by media and the delay on the prevalence of infectious disease. In all these models, the study reveals that the disease-free equilibrium (DFE) is stable when a basic reproduction number R_0 is less than one, unstable when $R_0 > 1$, irrespective of the value of the time delay and has a stable endemic equilibrium for time delay equals to zero. In the study of Zuo et al. [119], Zhao et al. [111], and Misra et al. [94], the occurrence of Hopf bifurcation is shown for the particular value of the time delay. Greenhalgh et al. [122] included two delays, one in reporting of infected cases and another delay represents the loss of disease awareness after a fixed period. Their study reveals the reduction in infected individuals with an increment in the duration of awareness. It is also shown that both the time delays can produce oscillations and destabilize the endemic equilibrium.

The public is a coalition of many subgroups of individuals with vastly different social, educational, and economic backgrounds. During an outbreak, people adopt full or partial awareness, depending on how they perceive risks and communicate about the effectiveness of protective measures. Therefore, instead of going directly from susceptible to infected class, we introduce fully aware and partial aware susceptible compartments into the SIR epidemic model due to heterogeneous protection level and extend the epidemic model to include the behavioral change of susceptibles, which can change the transmission patterns and reduce the prevalence of disease to the more extent. The precaution level of susceptible individuals is heterogeneous as they may take different levels of precautions to protect themselves from being infected based on the severity of epidemics or their characteristics. We consider three specific nonlinear incidence rates of unaware susceptibles, fully aware susceptibles, and partially aware susceptible, respectively, with the inclusion of time delay as a latent phase having a fixed duration. In the considered incidence rates, the interaction term is of the form $Sg(I)$, where, $g(I) = \beta I/(1+\varepsilon I)$, βI measures the force of infection, and $1/(1+\varepsilon I)$ measures the inhibition effect of the behavioral change of the susceptibles when their number increases and the crowding effect of the infective individuals [12, 98]. In this incidence rate, the number of infectives depends on the nonlinear bounded map $g(I)$, which tends to saturation level β/ε when I gets large. It prevents the unboundedness of the contact rate as it includes the psychological effects and, thus, more realistic than the bilinear incidence rate. Also, with awareness, treatment of infectives is essential to mitigate the infection. Therefore, we deliberate the Holling type II treatment rate (also called saturated treatment rate) and study the effect of treatment rate with limited medical resources in the current epidemic model. A detailed explanation of Holling type II treatment rate is given in Chapter two. We formulate the nonlinear time-delayed mathematical epidemic model and perform the stability analysis to demonstrate the eradication or persistence of the disease with the help of the basic reproduction number R_0 for the time delay $\rho \geq 0$. The bifurcation theory approach using the center manifold theory is performed, revealing the existence of backward and forward bifurcations. Further, choosing time delay as a bifurcation parameter, the periodic and oscillatory solutions appear via Hopf bifurcation. Moreover, the numerical experiments show the importance of considering full and aware susceptible individuals and nonlinear terms such as time delay, incidences, and treatment.

6.2 Mathematical model and basic properties

We assume that N denotes the total constant population, and it is divided into five compartments according to the disease status: unaware susceptible class $S(t)$, fully aware class $A_F(t)$, partially aware class $A_P(t)$, infected individuals class $I(t)$, and removed individuals class $R(t)$. The unaware susceptible class consists of those individuals who are vulnerable to the disease and taking no precautions against it. A fully aware susceptibles class involves those individuals who have adequate knowledge and proper information about the spread of the disease and taking precautionary measures against it. Partially aware susceptibles consist of those individuals who have an incomplete understanding of the spread and prevention of the disease and have low resources available to escape from the infection. Including a behavioral response among unaware susceptibles would unavoidably call for response among fully aware susceptibles and partially aware susceptibles. Therefore, we have considered three explicit same functional forms of saturated incidence rates describe below:
(i) $F_1(S(t-\rho),I(t-\rho)) = (\beta_1 S(t-\rho)I(t-\rho))/(1+\varepsilon I(t-\rho))$, representing the saturated incidence rate among unaware susceptibles,
(ii)$F_2(A_F(t-\rho),I(t-\rho)) = (\beta_2 A_F(t-\rho)I(t-\rho))/(1+\varepsilon I(t-\rho))$, representing the saturated incidence rate among fully aware susceptibles,
(iii) $F_3(A_P(t-\rho),I(t-\rho)) = (\beta_2 A_F(t-\rho)I(t-\rho))/(1+\varepsilon I(t-\rho))$, representing the saturated incidence rate among partially aware susceptibles.

In these incidences, ρ denotes the time delay, representing the latent period. The latent period is defined as the period between exposure and infection, since the pathogen is present in a latent stage, without clinical symptoms or signs of infection in the host. The delay term in $S(t)$, $A_F(t)$, and $A_P(t)$ is introduced because people, who are unaware, fully aware, or partially aware of the disease, may consider themself in their respective classes after becoming infected. We assume that such individuals are in the latent period.

Let κ denotes the constant rate of inflow of new unaware susceptibles due to the recruitment of new members by the current members or immigration. Let δ_1 denotes the rate at which unaware susceptibles $S(t)$ become fully aware. δ_2 denotes the rate at which $S(t)$ becomes partially aware of the disease. The parameters β_1, β_2, and β_3 are the transmission rates of infection of unaware, fully aware, and partially aware susceptible populations, respectively. We suppose that all the susceptible classes can become infected by contact with infected individuals, but the fully aware class has less chance

to be infected as compared to the unaware susceptible, and partially aware susceptible individuals [122]. Therefore, it is assumed that β_1 and β_3 are greater β_2. The parameter ε represents the behavioral changes or measures of inhibition adopted by infectives. ϑ denotes the natural mortality rate of all individuals, whereas d and θ denote the disease-induced mortality rate and recovery rate of the infectives, respectively. The function $h(I(t)) = aI/(1+bI)$ represents the treatment rate of infectives, where a and b denote the cure rate and limitation rate in the availability of resources, respectively. The symbols and description of the parameters and state variables are given in Table 6.1 briefly.

Table 6.1: Notations of model's variables and parameters.

Symbol	**Description**
N	Total constant population
$S(t)$	Unaware susceptibles
$A_F(t)$	Fully aware susceptibles
$A_P(t)$	Partially Aware susceptibles
$I(t)$	Infected population
$R(t)$	Removed population
ρ	Latent period (time delay)
κ	Constant recruitment rate of unaware susceptibles
β_1	Transmission rate of susceptibles to infected individuals
β_2	Transmission rate of fully aware to infected individuals
β_3	Transmission rate of partially aware to infected individuals
ε	Inhibition measures by infectives
δ_1	Rate of full awareness in unaware susceptibles
δ_2	Rate of partial awareness in unaware susceptibles
ϑ	Natural death rate
d	Disease-induced death rate
θ	Recovery rate
a	Cure rate
b	Rate of limitations in treatment availability

The flow diagram of the proposed epidemic model is given in Fig. 6.1 and the model

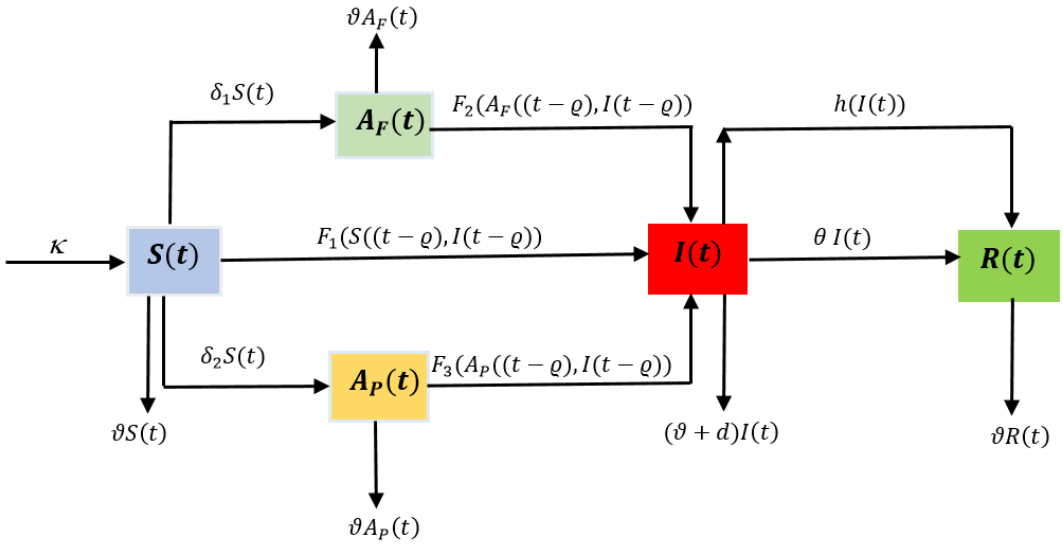


Figure 6.1: Flow diagram of the model (6.1).

is represented mathematically under the following system of delay differential equations:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= \kappa - \delta_1 S - \delta_2 S - \frac{\beta_1 S(t-\rho)I(t-\rho)}{1+\varepsilon I(t-\rho)} - \vartheta S,\\
\frac{\mathrm{d}A_F}{\mathrm{d}t} &= \delta_1 S - \frac{\beta_2 I(t-\rho)A_F(t-\rho)}{1+\varepsilon I(t-\rho)} - \vartheta A_F,\\
\frac{\mathrm{d}A_P}{\mathrm{d}t} &= \delta_2 S - \frac{\beta_3 I(t-\rho)A_P(t-\rho)}{1+\varepsilon I(t-\rho)} - \vartheta A_P,\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\beta_1 S(t-\rho)I(t-\rho)}{1+\varepsilon I(t-\rho)} + \frac{\beta_2 I(t-\rho)A_F(t-\rho)}{1+\varepsilon I(t-\rho)} + \frac{\beta_3 I A_P(t-\rho)}{1+\varepsilon I(t-\rho)} - (d+\theta+\vartheta)I - \frac{aI}{1+bI},\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \theta I + \frac{aI}{1+bI} - \vartheta R.
\end{aligned} \tag{6.1}$$

Suppose that the initial conditions of the model (6.1) takes the form

$$\begin{aligned}
&S(\nu) = \chi_1(\nu),\ A_F(\nu) = \chi_2(\nu),\ A_P(\nu) = \chi_3(\nu),\ I(\nu) = \chi_4(\nu),\ R(\nu) = \chi_5(\nu),\\
&\chi_i(\nu).\ \nu \in [-\rho,\ 0],\ \chi_i(0) > 0\ (i = 1,2,3,4,5),
\end{aligned} \tag{6.2}$$

where, $(\chi_1(\nu),\chi_2(\nu),\chi_3(\nu),\chi_4(\nu),\chi_5(\nu)) \in C\left([-\rho,\ 0],\ \mathbb{R}^5_+\right)$, which is the Banach space of continuous functions mapping the interval $[-\rho,\ 0]$ into $\mathbb{R}^5_+$, where,

$$\mathbb{R}^5_+ = \{(S,A_F,A_P,I,R)\,/S \geq 0, A_F \geq 0, A_P \geq 0, I \geq 0, R \geq 0\}.$$

Using Proposition 2.3. in [124] and proposition 2.1 in [107], it follows that all state

variables of the model (6.1) are nonnegative, i.e., $(S,A_F,A_P,I,R) \in R^5_+$. For biological reasons, we assume that the model's parameters κ, δ_1, δ_2 β_1, β_2, β_3, ε, ϑ, θ, d, a, b are positive. By the fundamental theory of functional differential equations [23], the model (6.1) has a unique solution $(S(t)A_F(t),A_P(t),I(t),R(t))$ satisfying the initial conditions (6.2).

Lemma 6.2.1. *The compact set*

$$\Omega = \left\{(S(t),A_F(t),A_P(t),I(t),R(t)) \in \mathbb{R}^5_+ : S(t)+A_F(t)+A_P(t)+I(t)+R(t) \leq \frac{\kappa}{\vartheta}\right\}$$

is invariant for the solutions of the model (6.1).

Proof. Since the right-hand side of the model (6.1), and its derivatives are continuous, therefore, it assures the wellpossness of the model (6.1).
On adding the equations of the model (6.1), we get

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}t}(S(t)+A_F(t)+A_P(t)+I(t)+R(t)) &= \kappa-\vartheta(S(t)+A_F(t)+A_P(t)+I(t)+R(t))-dI \\ &\leq \kappa-\vartheta(S(t)+A_F(t)+A_P(t)+I(t)+R(t)).\end{aligned} \tag{6.3}$$

Thus, the invariant region for the existence of the solutions is given as

$$0 < \lim_{t\to\infty}(S(t)+A_F(t)+A_P(t)+I(t)+R(t)) \leq \frac{\kappa}{\vartheta}. \tag{6.4}$$

Hence, the solution of the model (6.1) are compact. ■

In the next section, we obtain the model's equilibria and perform stability analysis to investigate the behavior of the equilibrium points.

6.3 Mathematical analysis

Since $R(t)$ does not appear in the first four equations of the model (6.1), therefore, without loss of generality, it is sufficient to consider first four equations of the model for

the analysis purpose:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= \kappa - \delta_1 S - \delta_2 S - \frac{\beta_1 S(t-\rho)I(t-\rho)}{1+\varepsilon I(t-\rho)} - \vartheta S,\\
\frac{\mathrm{d}A_F}{\mathrm{d}t} &= \delta_1 S - \frac{\beta_2 I(t-\rho)A_F(t-\rho)}{1+\varepsilon I(t-\rho)} - \vartheta A_F,\\
\frac{\mathrm{d}A_P}{\mathrm{d}t} &= \delta_2 S - \frac{\beta_3 I(t-\rho)A_P(t-\rho)}{1+\varepsilon I(t-\rho)} - \vartheta A_P,\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\beta_1 S(t-\rho)I(t-\rho)}{1+\varepsilon I(t-\rho)} + \frac{\beta_2 I(t-\rho)A_F(t-\rho)}{1+\varepsilon I(t-\rho)} + \frac{\beta_3 I A_P(t-\rho)}{1+\varepsilon I(t-\rho)} - (d+\theta+\vartheta)I - \frac{aI}{1+bI}.
\end{aligned} \tag{6.5}$$

For the existence of model's equilibrium, we set the right hand side terms of the system (6.5) to zero and obtain that the model (6.5) has two equilibria given below:

1. Disease-free equilibrium $E_0(S_0, A_{F_0}, A_{P_0}, 0)$ (represents the eradication of infected individuals, i.e. $I \equiv 0$ for all $t > 0$), discussed in subsection 6.3.1.

2. Endemic equilibrium $E_e(S^*, A_F^*, A_P^*, I^*)$ (represents the persistence of infected individuals above a certain positive level, i.e., $I > 0$ for all $t > 0$), discussed in subsection 6.3.2.

6.3.1 Disease-free equilibrium

The system (6.5) has a disease-free equilibrium of the form $E_0(S_0, A_{F_0}, A_{P_0}, 0)$, where,

$$S_0 = \frac{\kappa}{\delta_1+\delta_2+\vartheta},\ A_{F_0} = \frac{\delta_1\kappa}{\vartheta(\delta_1+\delta_2+\vartheta)},\ A_{P_0} = \frac{\delta_2\kappa}{\vartheta(\delta_1+\delta_2+\vartheta)}.$$

The characteristic equation of the system (6.5) at disease-free equilibrium is obtained as

$$(-\vartheta-\lambda)^2(-\delta_1-\delta_2-\vartheta-\lambda)\left(\lambda+(\vartheta+d+\theta+a)-(\beta_1 S_0+\beta_2 A_{F_0}+\beta_3 A_{P_0})\mathrm{e}^{-\lambda\rho}\right)=0. \tag{6.6}$$

Eq. (6.6) is fourth degree transcendental equation in λ with roots $\lambda_1 = -\vartheta$, $\lambda_2 = -\vartheta$, $\lambda_3 = -(\delta_1+\delta_2+\vartheta)$ and the other roots are the solution of

$$\lambda+(\vartheta+d+\theta+a)-(\beta_1 S_0+\beta_2 A_{F_0}+\beta_3 A_{P_0})\mathrm{e}^{-\lambda\rho}=0. \tag{6.7}$$

On simplification, Eq. (6.7) can be written as

$$X(\lambda) := \lambda+(\vartheta+d+\theta+a)-\frac{\kappa(\beta_1\vartheta+\beta_2\delta_1+\beta_3\delta_2)}{\vartheta(\delta_1+\delta_2+\vartheta)}\mathrm{e}^{-\lambda\rho}=0. \tag{6.8}$$

The term $\frac{\kappa(\beta_1\vartheta+\beta_2\delta_1+\beta_3\delta_2)}{\vartheta(\vartheta+d+\theta+a)(\delta_1+\delta_2+\vartheta)}e^{-\lambda\rho}$ at $\rho=0$ is defined as the basic reproduction number of the system (6.5). Thus, the basic reproduction number of the system (6.5) is

$$R_0=\frac{\kappa(\beta_1\vartheta+\beta_2\delta_1+\beta_3\delta_2)}{\vartheta(\vartheta+d+\theta+a)(\delta_1+\delta_2+\vartheta)}.$$

Analysis for $R_0\neq 1$

As mentioned above, Eq. (6.6) has three negative real roots $\lambda_1=-\vartheta$, $\lambda_2=-\vartheta$, $\lambda_3=-(\delta_1+\delta_2+\vartheta)$, and other roots are the solutions of the Eq. (6.8).

Note that

$$X(0)=(\vartheta+d+\theta+a)(1-R_0). \tag{6.9}$$

If $R_0>1$, then $X(0)<0$. Also,

$$X'(\lambda)=1+\frac{\kappa(\beta_1\vartheta+\beta_2\delta_1+\beta_3\delta_2)}{\vartheta(\delta_1+\delta_2+\vartheta)}e^{-\lambda\rho}\rho>0. \tag{6.10}$$

Thus, $\lim_{\lambda\to\infty}X(\lambda)=+\infty$.

Hence, $X(\lambda)=0$, $X(0)<0$, $X'(\lambda)>0$, and $\lim_{\lambda\to\infty}X(\lambda)=+\infty$ indicate that there always exists a unique and positive real root of $X(\lambda)=0$ if $R_0>1$.

Now, if $R_0<1$, then

$$\begin{aligned}\mathrm{Re}\,\lambda&=\frac{\kappa(\beta_1\vartheta+\beta_2\delta_1+\beta_3\delta_2)e^{-(\mathrm{Re}\,\lambda)\rho}\cos(Im\lambda)\rho}{\vartheta(\delta_1+\delta_2+\vartheta)}-(\vartheta+d+\theta+a)\\&<\frac{\kappa(\beta_1\vartheta+\beta_2\delta_1+\beta_3\delta_2)}{\vartheta(\delta_1+\delta_2+\vartheta)}-(\vartheta+d+\theta+a)\\&=(\vartheta+d+\theta+a)(R_0-1)\\&<0.\end{aligned} \tag{6.11}$$

Therefore, $R_0<1$ implies that λ is a root of Eq. (6.6) with negative real part.

Analysis at $R_0=1$

In this subsection, the system (6.5) is analyzed at $E_0(S_0,A_{F_0},A_{P_0},0)$ and $R_0=1$ for the time-delay $\rho\geq 0$.

Case (i) $\rho>0$

If $R_0=1$, then $\lambda=0$ is a simple characteristic root of Eq. (6.8). Note that $R_0=1$

implies that $\kappa(\beta_1\vartheta+\beta_2\delta_1+\beta_3\delta_2)=\vartheta(\vartheta+d+\theta+a)(\delta_1+\delta_2+\vartheta)$.

Let $\lambda=r+\mathrm{i}s$ be any of the other solutions, then (6.8) becomes:

$$r+\mathrm{i}s+(\vartheta+d+\theta+a)-(\vartheta+d+\theta+a)\mathrm{e}^{-r\rho}(\cos s\rho-\mathrm{i}\sin s\rho)=0. \tag{6.12}$$

By applying Euler's formula and splitting real and imaginary parts, Eq. (6.12) can be written as

$$\begin{aligned} r+\vartheta+d+\theta+a&=(\vartheta+d+\theta+a)\cos s\rho\ \mathrm{e}^{-r\rho},\\ s&=-(\vartheta+d+\theta+a)\sin s\rho\ \mathrm{e}^{-r\rho}. \end{aligned} \tag{6.13}$$

On squaring and adding both the equations of Eq. (6.13), we obtain

$$(r+\vartheta+d+\theta+a)^2+s^2=(r+\vartheta+d+\theta+a)^2\mathrm{e}^{-2r\rho}. \tag{6.14}$$

If there exists a root satisfying both the equations of (6.13), then this root also satisfies the Eq. (6.14) obtained by squaring and adding these equations. For Eq. (6.14) to be verified, we must have $r\leq 0$. Therefore, E_0 is linearly neutrally stable.

Case (ii) $\rho=0$

We now analyze the qualitative behavior of the system (6.5) without time delay, i.e., we take $\rho=0$. This analysis has an interest in itself and will also allow getting some information on the stability of the coexistence equilibrium in the case with delay [120]. The bifurcation theory approach obtained in [40], which is based on the center manifold theory [17], has been used to decide the local stability of a nonhyperbolic equilibrium (i.e., linearization matrix has at least one eigenvalue with zero real part) near the criticality E_0 and $R_0=1$. It allows us to clarify the direction of the bifurcation and describes the local behavior of disease-free equilibrium near $R_0=1$. For simplicity, we redefine the state variables as $S=x_1$, $A_F=x_2$, $A_P=x_3$, and $I=x_4$. So, the system (6.5) takes the

form:

$$\begin{aligned}
\frac{dx_1}{dt} &= \kappa - \delta_1 x_1 - \delta_2 x_1 - \frac{\beta_1 x_1 x_4}{\varepsilon x_4 + 1} - x_1 \vartheta \equiv g_1, \\
\frac{dx_2}{dt} &= -\frac{\beta_2 x_2 x_4}{\varepsilon x_4 + 1} - \vartheta x_2 + \delta_1 x_1 \equiv g_2, \\
\frac{dx_3}{dt} &= -\frac{\beta_3 x_3 x_4}{\varepsilon x_4 + 1} - \vartheta x_3 + \delta_2 x_1 \equiv g_3, \\
\frac{dx_4}{dt} &= \frac{\beta_1 x_1 x_4}{\varepsilon x_4 + 1} + \frac{\beta_2 x_2 x_4}{\varepsilon x_4 + 1} + \frac{\beta_3 x_3 x_4}{\varepsilon x_4 + 1} - (d + \theta + \vartheta) x_4 - \frac{a x_4}{b x_4 + 1} \equiv g_4.
\end{aligned} \tag{6.15}$$

We observe that when $R_0 = 1$, the chosen bifurcation parameter β_2 takes the form

$$\beta_2 = \beta_2^* = \frac{\delta_2 \left(\vartheta(a + d + \theta + \vartheta) - \beta_3 \kappa\right) + \vartheta \left(\delta_1 + \vartheta\right)(a + d + \theta + \vartheta) + \beta_1 \kappa(-\vartheta)}{\delta_1 \kappa}.$$

The Jacobian matrix $J(E_0,\ \beta_2^*)$ of the system (6.15) obtained at criticality (that is, at E_0 and β_2^*) is

$$J(E_0, \beta_2^*) = \begin{pmatrix}
-\vartheta - \delta_1 - \delta_2 & 0 & 0 & -\frac{\kappa \beta_1}{\vartheta + \delta_1 + \delta_2} \\
\delta_1 & -\vartheta & 0 & -\frac{\kappa \beta_2^* \delta_1}{\vartheta(\vartheta + \delta_1 + \delta_2)} \\
\delta_2 & 0 & -\vartheta & -\frac{\kappa \beta_3 \delta_2}{\vartheta(\vartheta + \delta_1 + \delta_2)} \\
0 & 0 & 0 & 0
\end{pmatrix}.$$

The eigenvalues of the Jacobian matrix $J(E_0, \beta_2^*)$ are $\lambda_1 = -\vartheta,\ \lambda_2 = -\vartheta,\ \lambda_3 = -\vartheta - \delta_1 - \delta_2,\ \lambda_4 = 0$. We see that λ_1, λ_2, and λ_3 are real and negative eigenvalues and $\lambda_4 = 0$ is a simple zero eigenvalue (since, the algebraic multiplicity of $\lambda_4 = 0$ is 1) of $J(E_0, \beta_2^*)$. Thus, when $R_0 = 1$, the DFE E_0 is a non-hyperbolic equilibrium.

The right eigenvector $v = (v_1, v_2, v_3, v_4)$ of $J(E_0, \beta_2^*)$ corresponding to $\lambda_4 = 0$ is obtained as

$$\begin{aligned}
v_1 &= -\frac{\beta_1 \kappa}{(\delta_1 + \delta_2 + \vartheta)^2}, \\
v_2 &= -\frac{\delta_1 \kappa (\beta_2 (\delta_1 + \delta_2 + \vartheta) + \beta_1 \vartheta)}{\vartheta^2 (\delta_1 + \delta_2 + \vartheta)^2}, \\
v_3 &= -\frac{\delta_2 \kappa (\beta_3 (\delta_1 + \delta_2 + \vartheta) + \beta_1 \vartheta)}{\vartheta^2 (\delta_1 + \delta_2 + \vartheta)^2}, \\
v_4 &= 1.
\end{aligned}$$

The left eigenvector $w = (w_1, w_2, w_3, w_4)$ of $J(E_0,\ \beta_2^*)$ corresponding to $\lambda_4 = 0$ is obtained

as

$$w_1 = 0,$$
$$w_2 = 0,$$
$$w_3 = 0,$$
$$w_4 = 1.$$

Let g_k's, $k = 1,2,3,4$, denote the right-hand side of the system (6.15). The bifurcation coefficients a_1 and b_1 defined in Theorem 4.1 of [40] are given by:

$$a_1 = \sum_{k,i,j=1}^{4} w_k v_i v_j \left(\frac{\partial^2 g_k}{\partial x_i \partial x_j}\right)_{E_0},$$
$$b_1 = \sum_{k,i=1}^{4} w_k v_i \left(\frac{\partial^2 g_k}{\partial x_i \partial \beta_2^*}\right)_{E_0}.$$

The non-zero partial derivatives of the functions g_k's at E_0 are evaluated as

$\left(\frac{\partial^2 g_4}{\partial x_4 \partial x_1}\right)_{E_0} = \beta_1,\ \left(\frac{\partial^2 g_4}{\partial x_4 \partial x_2}\right)_{E_0} = \beta_2^*,\ \left(\frac{\partial^2 g_4}{\partial x_4 \partial x_3}\right)_{E_0} = \beta_3,\ \left(\frac{\partial^2 g_4}{\partial x_1 \partial x_4}\right)_{E_0} = \beta_1,\ \left(\frac{\partial^2 g_4}{\partial x_2 \partial x_4}\right)_{E_0} = \beta_2^*,$
$\left(\frac{\partial^2 g_4}{\partial x_3 \partial x_4}\right)_{E_0} = \beta_3,\ \left(\frac{\partial^2 g_4}{\partial x_4^2}\right)_{E_0} = -\frac{2\varepsilon\beta_1\kappa}{\delta_1+\delta_2+\vartheta} - \frac{2\varepsilon\beta_2^*\delta_1\kappa}{\vartheta(\delta_1+\delta_2+\vartheta)} - \frac{2\varepsilon\beta_3\delta_2\kappa}{\vartheta(\delta_1+\delta_2+\vartheta)} + 2ab$, and
$\left(\frac{\partial^2 g_4}{\partial x_4 \partial \beta_2^*}\right)_{E_0} = \frac{\delta_1\kappa}{\vartheta(\delta_1+\delta_2+\vartheta)}$.

The bifurcation coefficients a_1 and b_1 are calculated at the bifurcation parameter β_2^* as follows:

$$\begin{aligned}
a_1 = &-\frac{2}{\delta_1\kappa\vartheta^2(\delta_1+\delta_2+\vartheta)}\big(\delta_1(\delta_2(\vartheta^2\left(\kappa(a(\varepsilon-b)+\varepsilon(d+\theta+\vartheta))+2(a+d+\theta+\vartheta)^2\right)\\
&+\beta_3\kappa(\beta_3\kappa-2\vartheta(a+d+\theta+\vartheta)))+\vartheta^3\left(\kappa(a(\varepsilon-b)+\varepsilon(d+\theta+\vartheta))+2(a+d+\theta+\vartheta)^2\right))\\
&+\delta_1^2\vartheta^2\left(\kappa(a(\varepsilon-b)+\varepsilon(d+\theta+\vartheta))+(a+d+\theta+\vartheta)^2\right)+(\delta_2(\vartheta(a+d+\theta+\vartheta)-\beta_3\kappa)\\
&+\vartheta^2(a+d+\theta+\vartheta))^2-\beta_1\kappa\vartheta(2\delta_2(\vartheta(a+d+\theta+\vartheta)-\beta_3\kappa)+\vartheta(\delta_1+2\vartheta)(a+d+\theta+\vartheta))\\
&+\beta_1^2\kappa^2\vartheta^2\big)\\
= &-\frac{2}{\delta_1\kappa\vartheta^2(\delta_1+\delta_2+\vartheta)}\times G(\beta_3),\\
b_1 = &\frac{\kappa}{\delta_1+\delta_2+\vartheta}.
\end{aligned}$$

where,

$$G(\beta_3) = \left(\zeta_2\ \beta_3^2 + \zeta_1\ \beta_3 + \zeta_0\right), \tag{6.16}$$

where, the coefficients ζ_0, ζ_1 and ζ_2 are

$$\begin{aligned}
\zeta_2 &= \delta_2\,(\delta_1+\delta_2)\,\kappa^2,\\
\zeta_1 &= 2\delta_2\kappa\vartheta\,(\beta_1\kappa-(\delta_1+\delta_2+\vartheta)\,(a+d+\theta+\vartheta)),\\
\zeta_0 &= \vartheta^2\big(\big(\delta_1+\delta_2+\vartheta\big)\big(\delta_1\big(\kappa(a(\varepsilon-b)+\varepsilon(d+\theta+\vartheta))+(a+d+\theta+\vartheta)^2\big)+\\
&\quad \big(\delta_2+\vartheta\big)(a+d+\theta+\vartheta)^2\big)+\beta_1\delta_1\kappa(a+d+\theta+\vartheta)+\beta_1\kappa\big(\beta_1\kappa-\\
&\quad 2\big(\delta_1+\delta_2+\vartheta\big)(a+d+\theta+\vartheta)\big)\big).
\end{aligned} \tag{6.17}$$

It can be seen that the bifucation coefficient b_1 is always positive and the sign of a_1 depends the sign of $G(\beta_3)$, given in Eq. (6.16). If $G(\beta_3) < 0$, then $a_1 > 0$, whereas, if $G(\beta_3) > 0$ then $a_1 < 0$.

The discriminant of quadratic polynomial $G(\beta_3)$ is obtained as

$$\begin{aligned}
D = a\big(\delta_1+\delta_2\big)\kappa(b-\varepsilon)\big(\delta_1+\delta_2+\vartheta\big)+\big(-\delta_1-\delta_2-\vartheta\big)\big(\big(\delta_1+\delta_2+\vartheta\big)(a+d+\theta+\vartheta)^2\\
+\varepsilon\big(\delta_1+\delta_2\big)\kappa(d+\theta+\vartheta)\big)+\beta_1\kappa\big(\delta_1+\delta_2+2\vartheta\big)(a+d+\theta+\vartheta)+\beta_1^2\big(-\kappa^2\big).
\end{aligned} \tag{6.18}$$

Let β_3^* and β_3^{**} be two roots of the Eq. (6.16), then we get

$$\beta_3^* = \frac{-\zeta_1+D}{2\zeta_2},\ \text{and}\ \beta_3^{**} = \frac{-\zeta_1-D}{2\zeta_2}. \tag{6.19}$$

Using Theorem 4.1(iv) in [40], the type of bifucation is govenered by the sign of a_1 and hence by the sign of $G(\beta_3)$. If $G(\beta_3)$ is of positive sign then forward bifurcation occurs, whereas, if the sign of $G(\beta_3)$ is negative then the system (6.15) reveals a backward bifurcation. These behavior differences are essential in planning how to control a disease; a backward bifurcation at $R_0 = 1$ makes control more difficult. These two cases are discussed below separately.

(I) Forward bifurcation: When there is a forward bifurcation at $R_0 = 1$, it is not possible for a disease to invade a population if $R_0 < 1$ because the system will return to the disease-free equilibrium $I = 0$ if some infectives are introduced into the population. For values of R_0 slightly greater than 1, E_0 changes its stability from stable to unstable

and the model admits a unique endemic equilibrium, which is locally asymptotically stable [40]. Therefore, it is imperative to find the range for which forward bifurcation occurs. The range of forward bifurcation is governed by the positivity of $G(\beta_3)$. Thus, there are two cases in which $G(\beta_3)$ is found to be positive. These are given as follows:

$$\begin{cases} \zeta_1 > 0, \\ \zeta_0 > 0. \end{cases} \tag{6.20}$$

or

$$\begin{cases} D > 0, \\ \text{either of } (\zeta_1 > 0,\ \zeta_0 < 0),\ (\zeta_1 < 0.\ \zeta_0 > 0),\ \text{or } (\zeta_1 < 0,\ \zeta_0 < 0) \text{ holds}, \\ \beta_3 < \beta_3^* \text{ or } \beta_3 > \beta_3^{**}. \end{cases} \tag{6.21}$$

(II) Backward bifurcation: The backward bifurcation is characterized as when $R_0 < 1$; a small unstable endemic equilibrium appears while the disease-free equilibrium and a larger endemic equilibrium are locally asymptotically stable. When $R_0 > 1$, then an unstable disease-free equilibrium and a stable endemic equilibrium exist [40]. It is illustrated in Fig. (6.4). The range of existence of backward bifurcation (i.e., when $G(\beta_3) < 0$) as follows:

$$\begin{cases} D > 0, \\ \text{either of } (\zeta_1 > 0,\ \zeta_0 < 0),\ (\zeta_1 < 0.\ \zeta_0 > 0),\ \text{or } (\zeta_1 < 0,\ \zeta_0 < 0) \text{ holds}, \\ \beta_3^{**} < \beta_3 < \beta_3^*. \end{cases} \tag{6.22}$$

Based on the analysis above, we state the following theorems:

Theorem 6.3.1. *The disease-free equilibrium $E_0(S_0, A_{F_0}, A_{P_0}, 0)$ of the delayed system (6.5) is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ for $\rho > 0$.*

Theorem 6.3.2. *The disease-free equilibrium $E_0(S_0, A_{F_0}, A_{P_0}, 0)$ of the delayed system (6.5) at $R_0 = 1$ is linearly neutrally stable for $\rho > 0$.*

Theorem 6.3.3. *When $R_0 = 1$, then the undelayed system (6.15) reveals a backward (forward) bifurcation at disease-free equilibrium $E_0(S_0, A_{F_0}, A_{P_0}, 0)$ if and only if $G(\beta_3) < 0$ (> 0).*

The graphical presentations of the forward and backward bifurcations are shown in Figs. 6.2, 6.3, and 6.4 for the experimental data metioned below:

$\kappa = 2,\ \varepsilon = 0.1,\ \vartheta = 0.01,\ d = 0.001,\ b = 0.8,\ \theta = 0.01,\ a = 0.9,\ \beta_1 = 0.004,\ \delta_1 = 0.01,\ \delta_2 = 0.009$. At these values of parameters, we evaluate that the range of β_3 is $\beta_3^{**} = 0.00098164 \leq \beta_3 \leq 0.0088652 = \beta_3^*$. The cases of occurrence of forward and backward bifurcations, given in the inequalities (6.20), (6.21), and (6.22), are illustrated from (1.)–(3.) as below:

(1.) If we take $\beta_3 = 0.00008$, then we obtain that $\zeta_2 = 0.000522$, $\zeta_1 = 1.242 \times 10^{-6} > 0$, and $\zeta_0 = 7.75344 \times 10^{-9} > 0$. This case illustrates the inequality (6.20) and the graph is shown in Fig. 6.2.

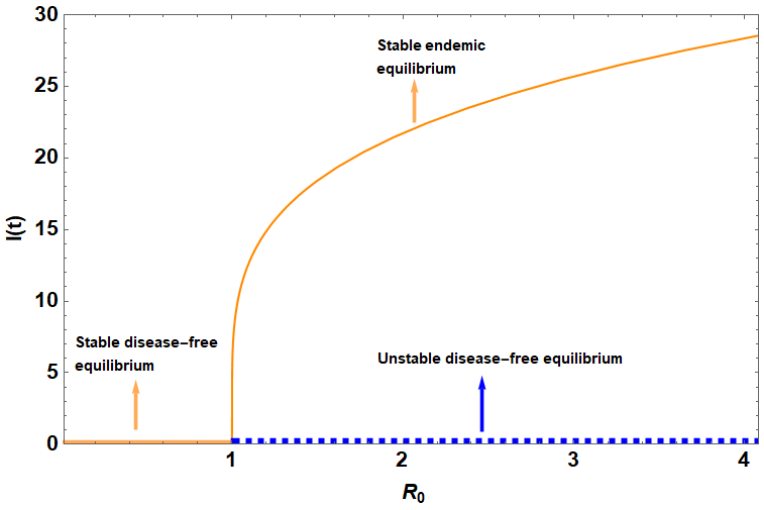


Figure 6.2: Plot of R_0 versus $I(t)$, showing forward bifurcation.

(2.) On considering $\beta_3 = 0.01$, we obtain that $\zeta_2 = 0.000522$, $\zeta_1 = 1.242 \times 10^-6 > 0$, $\zeta_0 = -5.44699 \times 10^{-8} < 0$, and the discriminant $D = 1.15276 \times 10^{-10} > 0$. It illustrates the inequality (6.21) and the graph is shown in Fig. 6.3.

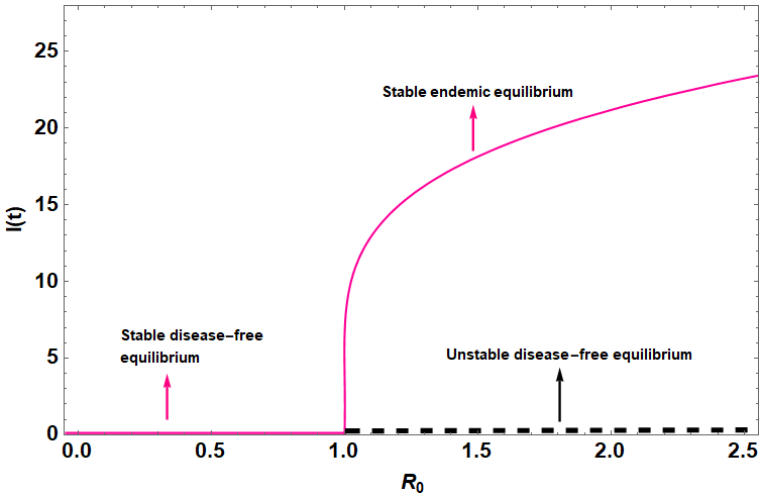


Figure 6.3: Plot of R_0 versus $I(t)$, showing forward bifurcation.

(3.) If we take $\beta_3 = 0.006$, which lies between β_3^{**} and β_3^*, then we obtain that the coefficients $\zeta_2 = 0.000522$, $\zeta_1 = 1.242 \times 10^{-6} > 0$ and $\zeta_0 = -4.05047 \times 10^{-8} < 0$, and the discriminant $D = 8.61164 \times 10^{-11} > 0$. This case illustrates the inequality (6.22) and the graph is shown in Fig. 6.4.

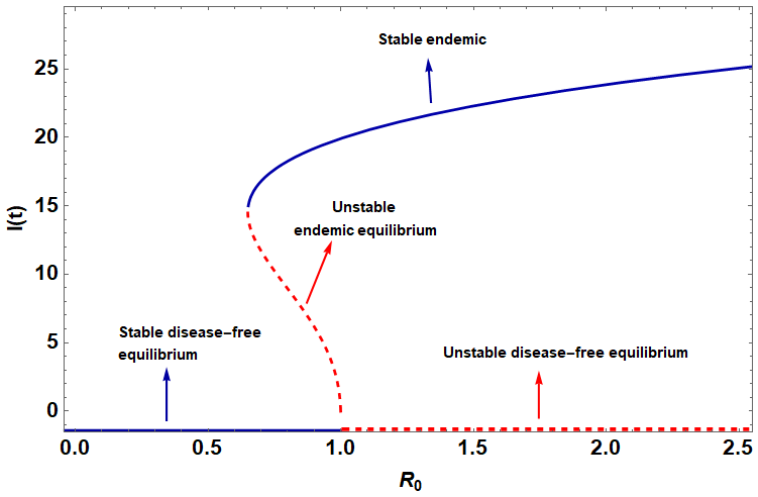


Figure 6.4: Plot of R_0 versus $I(t)$, showing backward bifurcation.

In Figs. 6.2, 6.3, and 6.4, the solid lines show stability and the dashed lines show instability.

6.3.2 Endemic equilibrium and stability

Assuming that S, A_F, A_P, $I \neq 0$. To determine the conditions for the existence of endemic (positive) equilibrium $E_e(S^*, A_F^*, A_P^*, I^*)$, we put the right-hand side of the system (6.5) to zero. we get:

$$\kappa - \delta_1 S - \delta_2 S - \frac{\beta_1 SI}{1+\varepsilon I} - S\vartheta = 0, \tag{6.23}$$

$$-\frac{\beta_2 A_F I}{1+\varepsilon I} - \vartheta A_F + \delta_1 S = 0, \tag{6.24}$$

$$-\frac{\beta_3 A_P I}{1+\varepsilon I} - \vartheta A_P + \delta_2 S = 0, \tag{6.25}$$

$$\frac{\beta_1 SI}{1+\varepsilon I} + \frac{\beta_2 A_F I}{1+\varepsilon I} + \frac{\beta_3 A_P I}{1+\varepsilon I} - (d+\theta+\vartheta)I - \frac{aI}{1+bI} = 0. \tag{6.26}$$

Solving for S, A_F and A_P in terms of I, from the Eqs. (6.23), (6.24) and (6.25), respectively, and substituting the resulting expression in Eq. (6.26), after simplification, we obtain the following equation in I:

$$P(I) := A_0 + A_1 I + A_2 I^2 + A_3 I^3 + A_4 I^4 = 0, \tag{6.27}$$

where,

$$
\begin{aligned}
A_0 &= \vartheta\,(\delta_1+\delta_2+\vartheta)\,(a+d+\theta+\vartheta)(1-R_0),\\
A_1 &= \vartheta\,((\delta_1+\delta_2+\vartheta)\,(3\varepsilon\vartheta+\beta_2+\beta_3)+\beta_1\vartheta)\,(a+d+\theta+\vartheta)-\beta_2\delta_1\kappa\vartheta(2\varepsilon+b)-\beta_3\delta_2\kappa\vartheta\\
&\quad\times(2\varepsilon+b)-\beta_1\kappa\vartheta^2(2\varepsilon+b)+b\vartheta^2\,(\delta_1+\delta_2+\vartheta)\,(d+\theta+\vartheta)-\beta_2\beta_3\delta_1\kappa\\
&\quad-\beta_2\beta_3\delta_2\kappa-\beta_1\,(\beta_2+\beta_3)\,\kappa\vartheta,\\
A_2 &= \vartheta\Big(\beta_3\,(\delta_2(2a\varepsilon-\varepsilon\kappa(\varepsilon+2b)+(2\varepsilon+b)(d+\theta+\vartheta))+(\delta_1+\vartheta)\,(2a\varepsilon+(2\varepsilon+b)(d+\theta+\vartheta)))\\
&\quad+3\varepsilon\vartheta\,(\delta_1+\delta_2+\vartheta)\,(a\varepsilon+(\varepsilon+b)(d+\theta+\vartheta))\Big)+\beta_2\Big(\beta_3\Big(\,(\delta_1+\delta_2)\,(a-\kappa(\varepsilon+b)\\
&\quad+d+\theta+\vartheta)+\vartheta(a+d+\theta+\vartheta)\Big)+\vartheta\big(\delta_1(2a\varepsilon-\varepsilon\kappa(\varepsilon+2b)+(2\varepsilon+b)(d+\theta+\vartheta))\\
&\quad+(\delta_2+\vartheta)\,(2a\varepsilon+(2\varepsilon+b)(d+\theta+\vartheta))\big)\Big)-\beta_1\Big(\beta_2\,(-\vartheta(a+d+\theta+\vartheta)+\kappa\vartheta(\varepsilon+b)+\beta_3\kappa)\\
&\quad+\vartheta\,(\vartheta(-2a\varepsilon+\varepsilon\kappa(\varepsilon+2b)-(2\varepsilon+b)(d+\theta+\vartheta))-\beta_3(a-\kappa(\varepsilon+b)+d+\theta+\vartheta))\Big),\\
A_3 &= -\,(\varepsilon\vartheta+\beta_3)\,((\varepsilon\vartheta+\beta_2)\,(-a\varepsilon\vartheta-\beta_1(a-b\kappa))-\varepsilon\delta_1\,(a\varepsilon\vartheta+\beta_2(a-b\kappa)))+\varepsilon\delta_2\,(\varepsilon\vartheta+\beta_2)\\
&\quad\times\big(a\varepsilon\vartheta+\beta_3(a-b\kappa)\big)-(d+\theta+\vartheta)\big(\beta_1\,(\vartheta\,(-\beta_3(\varepsilon+b)-\varepsilon\vartheta(\varepsilon+2b))-\beta_2\,(\vartheta(\varepsilon+b)+\beta_3))\\
&\quad-(\delta_1+\delta_2+\vartheta)\,(\beta_2\,(\beta_3(\varepsilon+b)+\varepsilon\vartheta(\varepsilon+2b))+\varepsilon\vartheta\,(\beta_3(\varepsilon+2b)+\varepsilon\vartheta(\varepsilon+3b)))\big),\\
A_4 &= b\,(\varepsilon\vartheta+\beta_2)\,(\varepsilon\vartheta+\beta_3)\,(d+\theta+\vartheta)\,(\varepsilon\,(\delta_1+\delta_2+\vartheta)+\beta_1)\,.
\end{aligned}
\tag{6.28}
$$

For a positive root of I^* of the polynomial $P(I)$, we have

$$S^* = \frac{\kappa+\varepsilon\kappa I^*}{(\delta_1+\delta_2+\vartheta)\,(\varepsilon I^*+1)+\beta_1 I^*}, \tag{6.29}$$

$$A_F^* = \frac{\delta_1 S^*(\varepsilon I^*+1)}{\varepsilon I^*\vartheta+\beta_2 I^*+\vartheta}, \tag{6.30}$$

$$A_P^* = \frac{\delta_2 S^*(\varepsilon I^*+1)}{\varepsilon I^*\vartheta+\beta_3 I^*+\vartheta}. \tag{6.31}$$

So, $E_e(S^*,A_F^*,A_P^*,I^*)$ is a positive equilibrium of the system (6.5).

Theorem 6.3.4. *When $R_0>1$, then there is either a unique or three positive endemic equilibria if all equilibria are simple roots.*

Proof. Let $R_0>1$. We see that the coefficient A_4 is always positive. On the other hand, $A_0<0$ when $R_0>1$. From Eq. (6.27), we have a fourth degree polynomial in I, given below:

$$P(I) := A_0+A_1I+A_2I^2+A_3I^3+A_4I^4=0.$$

The following possibilities for the signs of A_1, A_2, and A_3 exist:

$$\begin{aligned}
&\mathbf{V}_1 : A_1 > 0,\ A_2 > 0,\ \text{and}\ A_3 > 0,\\
&\mathbf{V}_2 : A_1 < 0,\ A_2 < 0,\ \text{and}\ A_3 > 0,\\
&\mathbf{V}_3 : A_1 < 0,\ A_2 > 0,\ \text{and}\ A_3 > 0,\\
&\mathbf{V}_4 : A_1 < 0,\ A_2 < 0,\ \text{and}\ A_3 < 0,\\
&\mathbf{V}_5 : A_1 > 0,\ A_2 > 0,\ \text{and}\ A_3 < 0,\\
&\mathbf{V}_6 : A_1 > 0,\ A_2 < 0,\ \text{and}\ A_3 > 0,\\
&\mathbf{V}_7 : A_1 > 0,\ A_2 < 0,\ \text{and}\ A_3 < 0,\\
&\mathbf{V}_8 : A_1 < 0,\ A_2 > 0,\ \text{and}\ A_3 < 0.
\end{aligned}$$

Using Descartes' rule of signs [42], $P(I)$ can have either a unique or three positive roots. If any of the conditions $\mathbf{V}_1$–$\mathbf{V}_4$ holds, then there is unique endemic equilibrium, whereas for the existence of three endemic equilibria, any one of the conditions $\mathbf{V}_5$–$\mathbf{V}_8$ must satisfy. ■

For the present study, we consider the case of unique endemic equilibrium only. H1: Suppose that any of the conditions (V_1–V_4, and $R_0 > 1$) holds, then the system (6.5) admits a unique endemic equilibrium.

Now, we study the local stability behavior of endemic equilibrium $E_e(S^*,A_F^*,A_P^*,I^*)$. For this, we obtain the characteristic equation of the system (6.5) at $E_e(S^*,A_F^*,A_P^*,I^*)$ as given below:

$$P_0(\lambda) + \mathrm{e}^{-\lambda\rho}P_1(\lambda) + \mathrm{e}^{-2\lambda\rho}P_2(\lambda) + \mathrm{e}^{-3\lambda\rho}P_3(\lambda) = 0, \tag{6.32}$$

where,

$$\begin{aligned}
P_0(\lambda) &= \lambda^4 + K_1\lambda^3 + K_2\lambda^2 + K_3\lambda + K_4,\\
P_1(\lambda) &= K_5\lambda^3 + K_6\lambda^2 + K_7\lambda + K_8,\\
P_2(\lambda) &= K_9\lambda^2 + K_{10}\lambda + K_{11},\\
P_3(\lambda) &= K_{12}\lambda + K_{13}.
\end{aligned} \tag{6.33}$$

where, the coefficients K_i, $i = 1$ to 13 are given in Appendix. For $\rho = 0$, the characteristic equation becomes:

$$\lambda^4 + Q_3\lambda^3 + Q_2\lambda^2 + Q_1\lambda + Q_0 = 0, \tag{6.34}$$

where, $Q_i's$, $i = 0,1,2,3$, are given in Appendix.

Based on Routh-Hurwitz criterion, it can be concluded that all the roots of Eq. (6.34) have negative real parts if the following inequalities hold:

$$\text{H2: } Q_i > 0 \ (i = 0,1,2,3),\ Q_3Q_2 - Q_1 > 0, \text{ and } Q_3Q_2Q_1 - Q_1^2 - Q_3^2Q_0 > 0. \tag{6.35}$$

Thus, we state the following Theorem:

Theorem 6.3.5. *At $\rho = 0$, the endemic equilibrium $E_2(S^*, A_F^*, A_P^*, I^*)$ is locally asmptotically stable if **H2** holds.*

Now multiply by $e^{-\lambda\rho}$ on both sides of the characteristic equation (6.32), we get:

$$P_0(\lambda)\mathrm{e}^{\lambda\rho} + P_1(\lambda) + \mathrm{e}^{-\lambda\rho}P_2(\lambda) + \mathrm{e}^{-2\lambda\rho}P_3(\lambda) = 0. \tag{6.36}$$

Change of stability and hence the existence of oscillatory solution may appear if the roots of the characteristic equation are purely imaginary. Therefore, to study the stability of E_e, assuming that $\lambda = \mathrm{i}\omega$, $(\omega > 0)$ is a root of Eq. (6.36). Then, Eq. (6.36) becomes:

$$P_0(\mathrm{i}\omega)\mathrm{e}^{\mathrm{i}\omega\rho} + P_1(\mathrm{i}\omega) + \mathrm{e}^{-\mathrm{i}\omega\rho}P_2(\mathrm{i}\omega) + \mathrm{e}^{-2\mathrm{i}\omega\rho}P_3(\mathrm{i}\omega) = 0. \tag{6.37}$$

Eq. (6.37) can be rewritten as

$$\begin{aligned}(M_0 + \mathrm{i}N_0)(\cos\omega\rho + \mathrm{i}\sin\omega\rho) + (M_1 + \mathrm{i}N_1) + (M_2 + \mathrm{i}N_2)(\cos\omega\rho - \mathrm{i}\sin\omega\rho)\\ + (M_3 + \mathrm{i}N_3)(\cos 2\omega\rho - \mathrm{i}\sin 2\omega\rho) = 0,\end{aligned} \tag{6.38}$$

where, M_0, M_1, M_2, M_3, N_0, N_1, N_2 and N_3 denote the real and imaginary parts of $P_0(\mathrm{i}\omega)$, $P_1(\mathrm{i}\omega)$, $P_2(\mathrm{i}\omega)$ and $P_3(\mathrm{i}\omega)$, respectively, given as:

$$\begin{aligned}
&M_0 = \omega^4 - K_2\omega^2 + K_4,\ N_0 = -K_1\omega^3 + K_3\omega,\\
&M_1 = -K_6\omega^2 + K_8,\ N_1 = -K_5\omega^3 + K_7\omega,\\
&M_2 = -K_9\omega^2 + K_{11},\ N_2 = K_10\omega,\\
&M_3 = K_{13},\ N_3 = K_{12}\omega.
\end{aligned}$$

When Eq. (6.38) splits into its real and imaginary parts, we get

$$M_1 + M_4 \cos\omega\rho + V_4 \sin\omega\rho = -N_3 \sin 2\omega\rho - M_3 \cos 2\omega\rho, \tag{6.39}$$

$$N_1 + N_5 \cos\omega\rho + M_5 \sin\omega\rho = -N_3 \cos 2\omega\rho + M_3 \sin 2\omega\rho, \tag{6.40}$$

where, $M_4 = M_0 + M_2,\ N_4 = N_2 - N_0,\ M_5 = M_0 - M_2$ and $N_5 = N_2 + N_0$.

On squaring Eqs. (6.39) and (6.40), and then adding, we obtain

$$(M_1 + M_4 \cos\omega\rho + N_4 \sin\omega\rho)^2 + (N_1 + N_5 \cos\omega\rho) + M_5(\sin\omega\rho)^2 - N_3^2 - M_3^2 = 0. \tag{6.41}$$

On substituting $\sin\omega\rho = \sqrt{1-\cos^2\omega\rho}$ in Eq. (6.41), we obtain

$$L_1 \cos^4\omega\rho + L_2 \cos^3\omega\rho + L_3 \cos^2\omega\rho + L_4 \cos\omega\rho + L_5 = 0, \tag{6.42}$$

where,

$$\begin{aligned}
&L_1 = p_1^2 + 4p_5^2,\ L_2 = 4p_1p_2 + 8p_4p_5,\\
&L_3 = 4p_2^2 + 2p_1p_3 + 4p_4^2 - 4p_5^2,\ L_4 = 4p_2p_3 - 8p_4p_5,\\
&L_5 = p_3^2 - 4p_4^2,\\
&p_1 = M_4^2 + N_5^2 - N_4^2 - M_5^2,\ p_2 = M_1M_4 + N_1N_4,\\
&p_3 = M_1^2 + N_4^2 + N_1^2 + M_5^2 - N_3^2 - M_3^2,\ p_4 = M_1N_4 + N_1M_5,\\
&p_5 = M_4N_4 + M_5N_5.
\end{aligned}$$

Let $\cos\omega\rho = x$, then Eq. (6.42) can be written as

$$f(x) := L_1x^4 + L_2x^3 + L_3x^2 + L_4x + L_5 = 0. \tag{6.43}$$

From Eq. (6.43), we obtain

$$f'(x) = 4L_1x^3 + 3L_2x^2 + 2L_3x + L_4 = 0. \tag{6.44}$$

For convenience, we assume that $x = y - L_2/4L_1$. Then, Eq. (6.44) becomes:

$$y^3 + Ay + B = 0, \tag{6.45}$$

where,

$$A = \frac{L_3}{2L_1} - \frac{3}{16}\left(\frac{T_2}{T_1}\right)^2,$$
$$B = \frac{1}{32}\left(\frac{L_2}{L_1}\right)^3 - \frac{L_2 L_3}{8L_1^2} + \frac{L_4}{4L_1}.$$

Now, roots of Eq. (6.45) are given as

$$y_1 = \sqrt[3]{-\frac{B}{2} + \sqrt{O}} + \sqrt[3]{-\frac{B}{2} - \sqrt{O}},$$
$$y_2 = \eta_1 \sqrt[3]{-\frac{B}{2} + \sqrt{O}} + \eta_2 \sqrt[3]{-\frac{B}{2} - \sqrt{O}},$$
$$y_3 = \eta_2 \sqrt[3]{-\frac{B}{2} + \sqrt{O}} + \eta_1 \sqrt[3]{-\frac{B}{2} - \sqrt{O}},$$

where, $\eta_1 = \frac{-1+\sqrt{3}\mathrm{i}}{2}$, $\eta_2 = \frac{-1-\sqrt{3}\mathrm{i}}{2}$ and $O = \frac{B^2}{4} + \frac{A_3}{27}$.

It follows from $\cos\omega\rho = x$ and $x = y - L_2/4L_1 = F_1(\omega)$ that

$$\cos\omega\rho = F_1(\omega). \tag{6.46}$$

On substituting Eq. (6.46) in Eq. (6.41) and solving for $\sin\omega\rho$, we obtain

$$\sin\omega\rho = F_2(\omega). \tag{6.47}$$

From Eqs. (6.46) and (6.47), we get

$$F_1^2(\omega) + F_2^2(\omega) = 1. \tag{6.48}$$

Assume that H3: Eq. (6.48) has atleast one positive root ω_0, such that the characteristic equation (6.32) has a pair of purely imaginary roots $\mathrm{i}\omega_0$.

For ω_0, the corresponding critical value of time delay ρ_k can be obtained as:

$$\rho_k = \frac{1}{\omega_0}\left(\arccos F_1(\omega_0) + 2k\pi\right),\ k = 0,1,2,\ldots. \tag{6.49}$$

Assume that ρ is a bifurcation parameter and ρ_0=$\min\rho_k$, $k = 0,1,2,\ldots$ is the criticle

value.

To estabilish the Hopf bifurcation, we show that $Re\left[\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right]^{-1}\Big|_{\lambda=\mathrm{i}\omega_0} \neq 0$.

Differentiating Eq. (6.32) with respect to ρ, we obtain:

$$\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\Big(P_0'(\lambda)+\mathrm{e}^{-\lambda\rho}P_1'(\lambda)-\rho\mathrm{e}^{-\lambda\rho}P_1(\lambda)+\mathrm{e}^{-2\lambda\rho}P_2'(\lambda)-2\rho P_2(\lambda)\mathrm{e}^{-2\lambda\rho}+\mathrm{e}^{-3\lambda\rho}P_3'(\lambda)- 3\rho P_3(\lambda)\mathrm{e}^{-3\lambda\rho}\Big)-\lambda\mathrm{e}^{-\lambda\rho}P1(\lambda)-2\lambda P_2(\lambda)\mathrm{e}^{-2\lambda\rho}-3\lambda P_3(\lambda)\mathrm{e}^{-3\lambda\rho}=0.$$

$$\Longrightarrow \frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\Big(P_0'(\lambda)+\mathrm{e}^{-\lambda\rho}P_1'(\lambda)-\rho\mathrm{e}^{-\lambda\rho}P_1(\lambda)+\mathrm{e}^{-2\lambda\rho}P_2'(\lambda)-2\rho P_2(\lambda)\mathrm{e}^{-2\lambda\rho}+\mathrm{e}^{-3\lambda\rho}P_3'(\lambda)- 3\rho P_3(\lambda)\mathrm{e}^{-3\lambda\rho}\Big)=\lambda\mathrm{e}^{-\lambda\rho}P1(\lambda)+2\lambda P_2(\lambda)\mathrm{e}^{-2\lambda\rho}+3\lambda P_3(\lambda)\mathrm{e}^{-3\lambda\rho}.$$

$$\Longrightarrow \frac{\mathrm{d}\lambda}{\mathrm{d}\rho}=\frac{\lambda\left(P_1(\lambda)+2P_2(\lambda)\mathrm{e}^{-\lambda\rho}+3P_3(\lambda)\mathrm{e}^{-2\lambda\rho}\right)}{\left(P_0'(\lambda)\mathrm{e}^{\lambda\rho}+P_1'(\lambda)-\rho P_1(\lambda)+\mathrm{e}^{-\lambda\rho}\left(P_2'(\lambda)-2\rho P_2(\lambda)\right)+\mathrm{e}^{-2\lambda\rho}\left(P_3'(\lambda)-3\rho P_3(\lambda)\right)\right)}.$$

Thus, we get

$$\left[\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right]^{-1}=\frac{\left(P_0'(\lambda)\mathrm{e}^{\lambda\rho}+P_1'(\lambda)+\mathrm{e}^{-\lambda\rho}P_2'(\lambda)+\mathrm{e}^{-2\lambda\rho}P_3'(\lambda)\right)}{\lambda\left(P_1(\lambda)+2P_2(\lambda)\mathrm{e}^{-\lambda\rho}+3P_3(\lambda)\mathrm{e}^{-2\lambda\rho}\right)}- \frac{\left(\rho P_1(\lambda)+2\rho P_2(\lambda)\mathrm{e}^{-\lambda\rho}+3\rho P_3(\lambda)\mathrm{e}^{-2\lambda\rho}\right)}{\lambda\left(P_1(\lambda)+2P_2(\lambda)\mathrm{e}^{-\lambda\rho}+3P_3(\lambda)\mathrm{e}^{-2\lambda\rho}\right)}.$$

$$\text{i.e., } \left[\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right]^{-1}=\frac{\left(P_0'(\lambda)\mathrm{e}^{\lambda\rho}+P_1'(\lambda)+\mathrm{e}^{-\lambda\rho}P_2'(\lambda)+\mathrm{e}^{-2\lambda\rho}P_3'(\lambda)\right)}{\lambda\left(P_1(\lambda)+2P_2(\lambda)\mathrm{e}^{-\lambda\rho}+3P_3(\lambda)\mathrm{e}^{-2\lambda\rho}\right)}-\frac{\rho}{\lambda}.$$

$$\mathrm{Re}\left[\frac{d\lambda}{d\rho}\right]^{-1}\Big|_{\lambda=\mathrm{i}\omega_0}=\frac{X_1X_2+Y_1Y_2}{X_2^2+Y_2^2},$$

where,

$$\begin{aligned}
X_1 &= -\sin\omega_0\rho_0\left(-4\omega_0^3+2K_2\omega_0\right)+\cos\omega_0\rho_0\left(-3K_1\omega_0^2+K_3\right)+\left(-3K_5\omega_0^2+K_7\right)\\
&\quad+\left(K_{10}\cos\omega_0\rho_0+2\omega_0K_9\sin\omega_0\rho_0\right)+K_{16}\cos 2\omega_0\rho_0,\\
X_2 &= K_5\omega_0^4-K_7\omega_0^2-2K_{10}\omega_0^2\cos\omega_0\rho_0+2\left(-K_9\omega_0^2+\omega_0K_{11}\right)\sin\omega_0\rho_0-3\omega_0^2K_{12}\cos 2\omega_0\rho_0\\
&\quad+3\omega_0K_{13}\sin 2\omega_0\rho_0,
\end{aligned}$$

$$Y_1 = \cos\omega_0\rho_0\left(-4\omega_0^3+2K_2\omega_0\right)+\sin\omega_0\rho_0\left(-3K_1\omega_0^2+K_3\right)+2\omega_0K_6+2\omega_0K_9\cos\omega_0\rho_0-K_{10}\sin\omega_0\rho_0$$
$$-K_{16}\sin 2\omega_0\rho_0,$$
$$Y_2 = -K_6\omega_0^3+\omega_0K_8+2\left(-K_9\omega_0^3+\omega_0K_{11}\right)\cos\omega_0\rho_0+3\left(\omega_0^2K_{12}\sin 2\omega_0\rho_0+\omega_0K_{13}\cos 2\omega_0\rho_0\right)$$
$$+2\omega_0^2K_{10}\sin\omega_0\rho_0.$$

Obviously, if H4: $X_1X_2+Y_1Y_2\neq 0$, then $Re\left[\frac{d\lambda}{d\rho}\right]^{-1}\Big|_{\lambda=i\omega_0}\neq 0$.

Thus, we state the following theorem:

Theorem 6.3.6. *For the system (6.5), if the conditions **(H1-H4)** hold, then the endemic equilibrium $E_e=(S^*,A_F^*,A_P^*,I^*)$ is locally asmptotically stable when $\rho\in[0,\rho_0)$; the system (6.5) undergoes a Hopf bifurcation at $E_e=(S^*,A_F^*,A_P^*,I^*)$ when $\rho=\rho_0$, and a family of periodic solutions bifurcate from $E_e=(S^*,A_F^*,A_P^*,I^*)$.*

6.4 Numerical simulation

In this section, numerical experiments are presented to show the analytical results using Mathematica 11.

We have considered the following set of experimental data: $\kappa=2$, $\vartheta=0.01$, $\beta_1=0.02$, $\beta_2=0.006$, $\beta_3=0.008$, $\delta_1=0.1$, $\delta_2=0.02$, $a=0.04$, $d=0.009$, $\theta=0.004$, $\varepsilon=0.5$, $b=0.09$.

At these parameters values, the endemic equilibrium is

$E_e(S^*,A_F^*,A_P^*,I^*)=(11.903,55.602,9.443,38.504)$ with $R_0>1$.

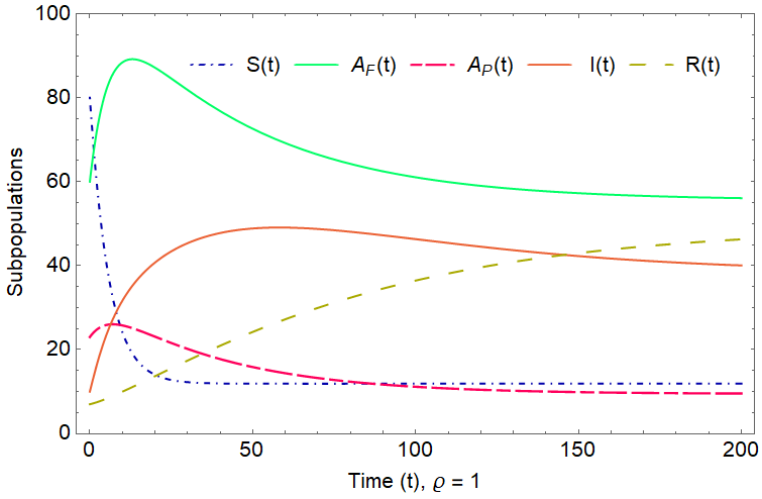


Figure 6.5: Subpopulations $S-A_P-A_F-I-R$ at $\rho=1$.

Fig. 6.5 shows the behavior of different subpopulations for the time delay $\rho=1$.

Evidently, as time increases, unaware susceptibles decrease, and the fully aware and partially aware, infected, and removed individuals population increase and then start decaying and settle down to steady-state $E_e(11.903, 55.602, 9.443, 38.504)$.

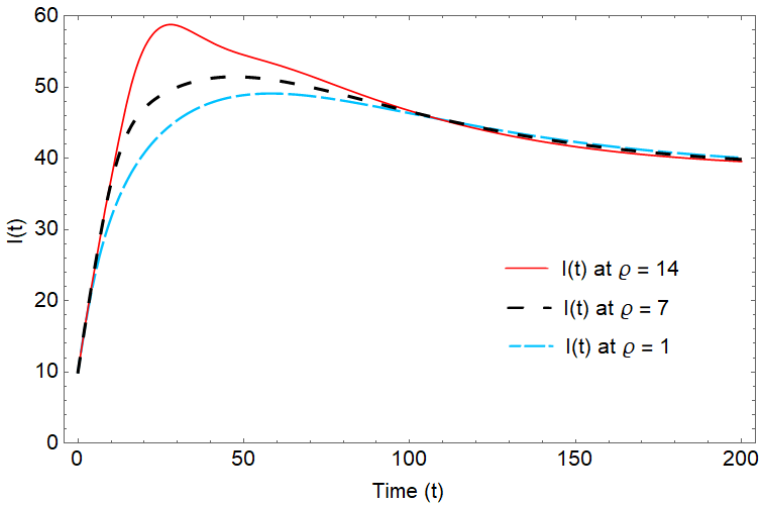
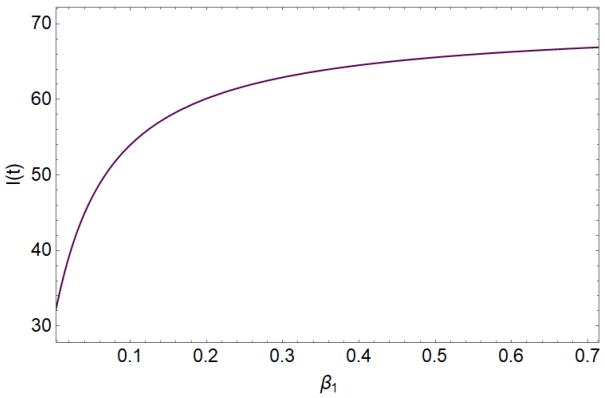
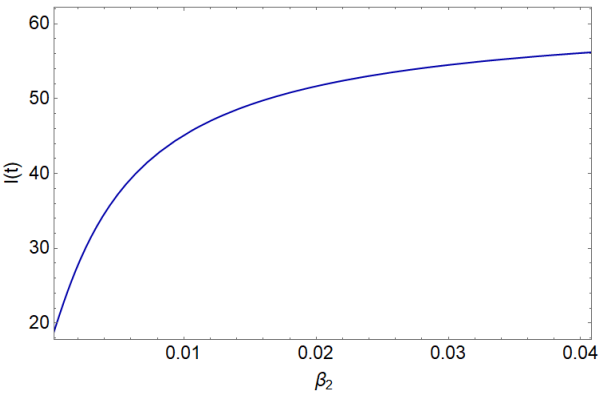


Figure 6.6: Infected population $I(t)$ at different values of time delay ρ.

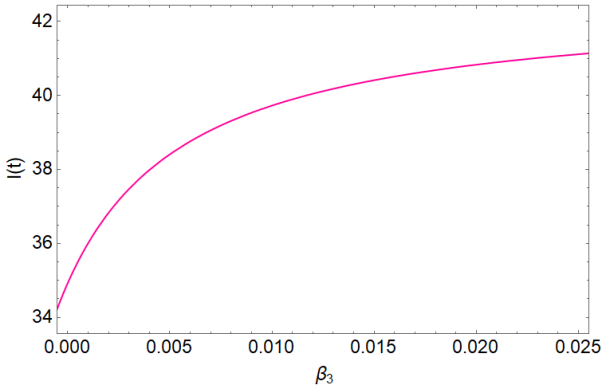
Fig. 6.6 depicts the effect of time delay on the infected population. We plot the infected population for different values of time delay $\rho = 1, 7$, and 14, respectively. It reveals that the large value of time delay causes an increment in the infected population.



6.7.1: Infected population $I(t)$ when β_1 varies.



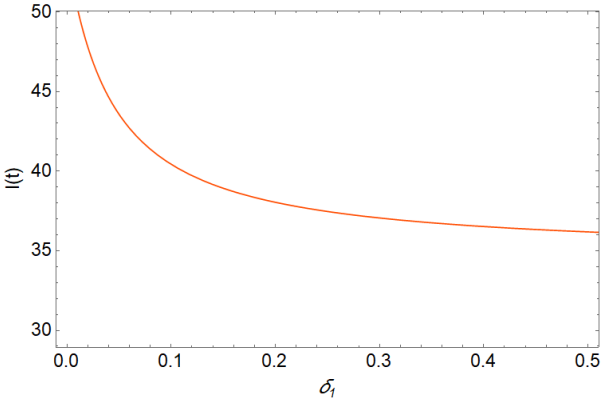
6.7.2: Infected population $I(t)$ when β_2 varies.



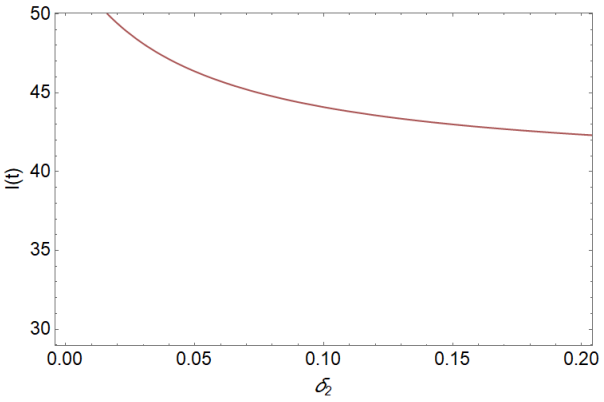
6.7.3: Infected population $I(t)$ when β_3 varies.

Figure 6.7: Infected population for the transmission rates of unaware, fully aware, and partially aware susceptibles for the time-delay $\rho = 1$.

Figs. 6.7.1, 6.7.2, and 6.7.3 show the influence of different transmission rates on infected population $I(t)$. It validates the increment in the number of infected population as the transmission rates increase, which is biologically true.



6.8.1: Infected population $I(t)$ for δ_1.



6.8.2: Infected population $I(t)$ for δ_2.

Figure 6.8: Impact of full and partial awareness rates on infected population for the time delay $\rho = 1$.

Figs. 6.8.1 and 6.8.2 show the impact of full and partial awareness rates (δ_1 and δ_2) on the infected population for the time delay $\rho = 1$. It is evident that if the full awareness rate is high, then infection diminishes at a high level. Partially awareness in humans also helps them to escape from the infection, as depicted by Fig. 6.8.2. Thus, more efforts should be put to spread full awareness among people.

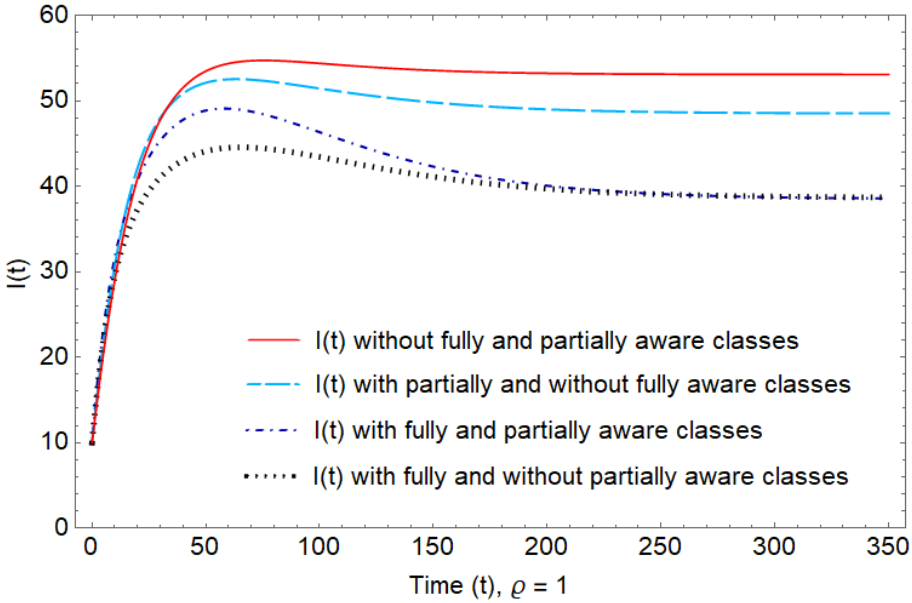
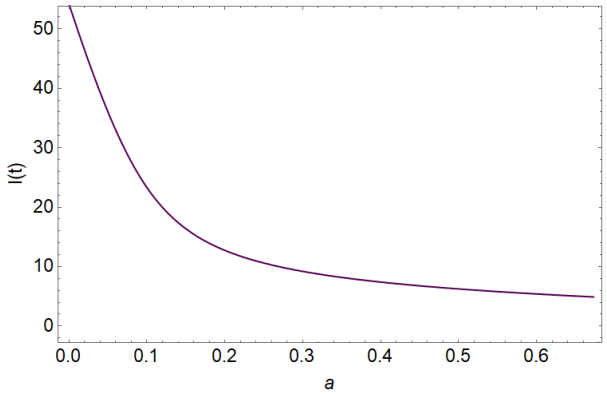
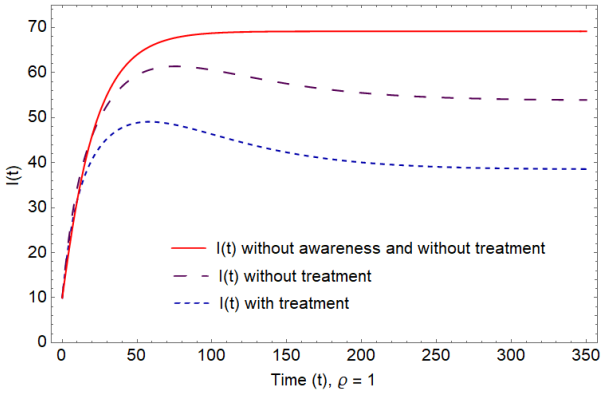


Figure 6.9: Dynamics of infectious diseases showing the impact of aware classes on infected individuals $I(t)$ for the time delay $\rho = 1$.

Fig. 6.9 examines the potential of fully and partially aware classes in minimizing the impact of an epidemic. From the graph, it is evident that when people are not aware of the spread of disease (shown by a solid red line), the infection occurs at a higher rate. Awareness in humans motivates them to escape from the infection. By the complete and correct information about a disease, individuals change their attitudes and actions to reduce their chances of becoming infected, spreading the disease further, or experiencing prolonged periods of medical treatment. Attempts to raise awareness of an infectious disease may also build a sense of threat in inadequately informed individuals. When there is only partial information available, infection reduces, but a low level (as shown by the dashed green line). Because of weak, inaccessible, and absence of education, uneducated individuals are rarely formally trained in handling diseases, its prognosis, diagnosis, preventions, and cures. They take preventive measures by word of mouth or by social media, which leads to a reduction in infection at a low level. Also, if people are partially mindful, then there are chances that they can take unnecessary medications due to fear of catching the infection, which can weaken their immune system. So those people are at more risk of getting infected (shown by the dot-dashed blue line). Therefore, the absence of partial awareness and the presence of full awareness about the spread and prevention of disease leads to a reduction in the range of illness at a high rate (shown by the dotted black line).



6.10.1: Effect of cure rate a on infected population $I(t)$ for the time delay $\rho = 1$.



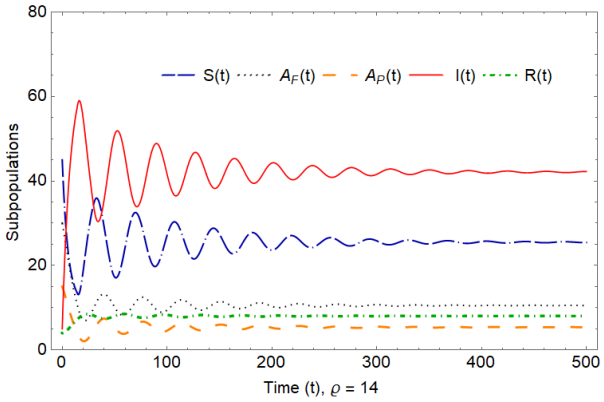
6.10.2: Infected individuals $I(t)$ with and without saturated treatment rate and aware classes for the time delay $\rho = 1$.

Figure 6.10: Impact of cure rate, awareness, and saturated treatment on the infected population for $\rho = 1$.

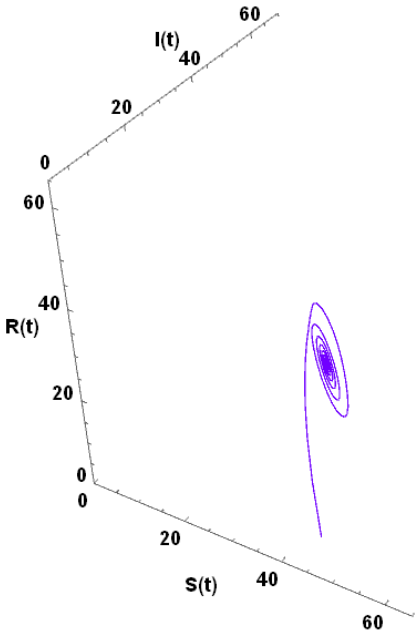
Fig. 6.10.1 shows that the increase in the cure rate can increase the reduction of infected individuals. Fig. 6.10.2 shows the influence of saturated treatment rate on infected population $I(t)$. We have plotted the infected population when there is neither treatment nor awareness available. Clearly, in this case, infection is spreading at a very high rate. Purple dashed line shows the infected population when people are aware, but treatment is not available, and the solid red line shows the infected population in the absence of awareness and treatment. The major difference can be seen between these two lines. Blue dashed line shows the infected population when both awareness and treatment are present, which is stabilizing at the lowest level among three lines. Thus, the treatment rate with awareness help in reducing infection at a faster rate.

To show the presence of Hopf bifurcation, we take the following experimental data: $\kappa = 10,\ \varepsilon = 0.92,\ \beta_1 = 0.2,\ \beta_2 = 0.02,\ \beta_3 = 0.04,\ \delta_1 = 0.05,\ \delta_2 = 0.03,\ \vartheta = 0.1,\ a = 0.2,\ b = 0.5,\ \theta = 0.01,\ d = 0.01.$

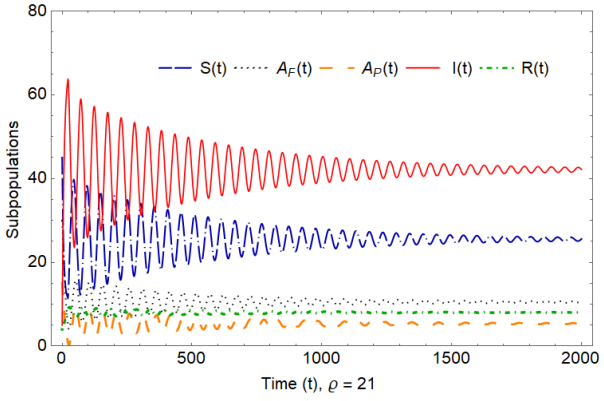
We obtain that $E_e(S^*, A_F^*, A_P^*, I^*) = (25.5152, 10.5267, 5.37598, 42.1257)$, at these values of parameters.



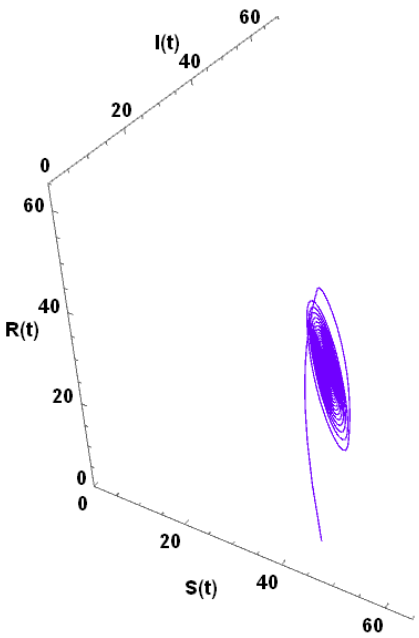
6.11.1: Time series solution of the model (6.1) for $\rho = 14$.



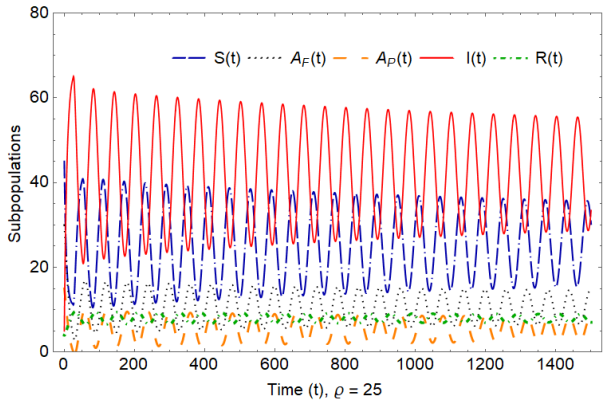
6.11.2: Phase plot of susceptible, infected and removed population when $\rho = 14$.



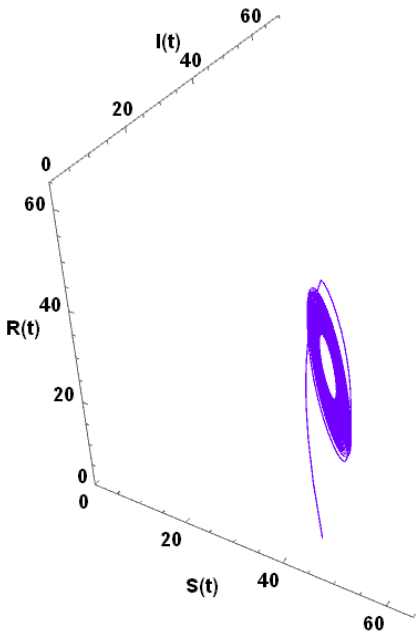
6.11.3: Time series solution of the model (6.1) for $\rho = 21$.



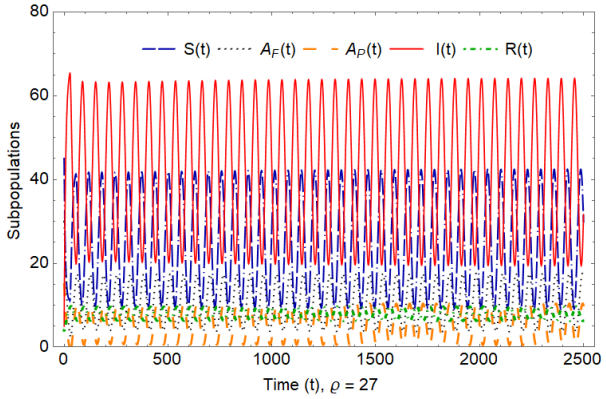
6.11.4: Phase plot of susceptible, infected and removed population when $\rho = 21$.



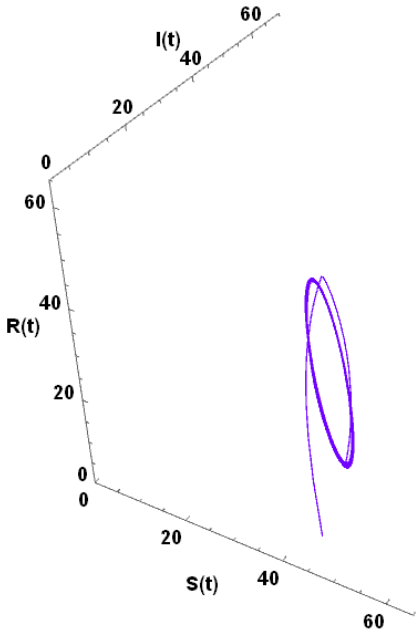
6.11.5: Time series solution of the model (6.1) for $\rho = 25$.



6.11.6: Phase plot of susceptible, infected and removed population when $\rho = 25$.



6.11.7: Time series solution of the model (6.1) for $\rho = 27$.



6.11.8: Phase plot of susceptible, infected and removed population when $\rho = 27$.

Figure 6.11: Graphs depicting the presence of Hopf bifurcation for different values of time-delay ρ.

Figs. 6.11 depicts the time series solutions of the model (6.1) with their respective SIR phase plot for distinct values of time delay ρ. It is observed that the spread of infectious disease can be controlled for $\rho = 14$, and $\rho = 21$. That is, Figs. 6.11.1, 6.11.2, 6.11.3, and 6.11.4 show that initially periodic solutions appear but after a time, the endemic

equilibrium E_e reaches to its steady state. Figs. 6.11.5, 6.11.6, 6.11.7, and 6.11.8 show the unstable limit cycle around the endemic equilibrium equilibrium when time delay $\rho > \rho_0 = 24$.

6.5 Discussion

In the present chapter, we divide the total population into five compartments: unaware susceptibles, fully aware susceptibles, partially aware susceptibles, infected, and removed individuals; and study a time-delayed epidemic model with saturated incidences and treatment rates. We analyze the model mathematically and study the dynamic behaviors of the epidemic model with and without time delay ρ. The mathematical analysis of the model shows that it exhibits two equilibria: disease-free and endemic. By deriving the basic reproduction number R_0, we prove that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, unstable when $R_0 > 1$, and linearly neutrally stable when $R_0 = 1$ for the time delay $\rho > 0$. When we don't consider the time delay, then using the center manifold theory, it is obtained that the forward or backward bifurcation occurs when $R_0 = 1$. We obtain the bifurcation range of forward and backward bifurcations, given in the inequalities (6.20), (6.21), and (6.22), respectively. In the presence of forward bifurcation, as R_0 increases through unity, then the disease-free equilibrium loses its stability, and a stable endemic equilibrium appears. In contrast, the presence of backward bifurcation shows that a stable endemic equilibrium coexists with a stable DFE when $R_0 < 1$. It has an important implication as it fails the ideal condition of reducing R_0 below unity to eradicate the diseases from society. Thus, the control programs must reduce R_0 further than below unity to eliminate the disease. Schematic diagrams of forward and backward bifurcation are depicted in Figs. (6.2), (6.3), and (6.4). Further, the local stability of the endemic equilibrium has been studied, which demonstrates that by choosing time delay as a bifurcation parameter, the Hopf bifurcation occurs near the endemic equilibrium, revealing the presence of oscillatory and periodic solutions.

The numerical simulations show the graphical representation of the effectiveness of theoretical results. We see that the consideration of time delay has a significant role as it impacts the number of infectives. It is seen that when the time delay is high, then the infection spreads at a higher rate. The periodic and oscillatory solutions have been plotted near-endemic equilibrium, showing Hopf bifurcation at different values of time delay ρ. As ρ passes through the critical value ρ_0, then the endemic equilibrium loses

its stability, and an unstable limit cycle appears. When there is no treatment available, then knowledge about the spread of disease is the main focus. The role of full and partial awareness in susceptibles has been shown numerically with and without saturated treatment. When treatment is not given to infected individuals, only susceptibles' full or partial awareness shows a significant difference in the number of infectives (Figs. 9 and 10(b)). Whereas, if we consider saturated treatment rate along with awareness in the susceptibles, then the transmission pattern of infectious diseases changes more effectively, and the reduction in the prevalence of the disease can be seen to a more extent.

The findings of the model, consisting of explicit saturated incidences with latent period and saturated treatment rate, are capable of demonstrating the significant role of the latent period, the behavior of susceptibles through different subclasses, and limitation in available facilities of treatment. The results can understand the role of varying protection levels of susceptibles in the transmission pattern of infectious diseases, suggesting the control strategies to prevent the spread of infections at a massive scale. A full awareness about the spread of infectious disease increases public perception to avert infection and willingness to adopt prevention methods, which mitigates disease transmission. Due to the different social, educational, and limited information resources, some people may have incomplete information. Therefore, these partially aware individuals adopt insufficient preventive methods, through which infection reduces but at a low level. Public health initiatives can put extra effort into making individuals fully aware by enhancing health literacy and providing sufficient information resources. It can contribute to early case detection and help in reducing the transmission of infection. Thus, for eradicating the disease, programs related to regular knowledge, full information, education, and communication, concerning the spread of infectious diseases and their importance to the public and health care workers will help them improve their general attitude toward it. Timely disease pieces of information updates are highly needed. Together with awareness, appropriate treatment to infectives, and the availability of health resources will help in diminishing the infection from society effectively.

Appendix

Using Mathematica 11, we obtain the coefficients of the characteristic equation (6.32) as follows:

$$K_1 = \frac{a+(1+bI^*)^2(d+\theta+4\vartheta)+\delta_1(1+bI^*)^2+\delta_2(1+bI^*)^2}{(1+bI^*)^2},$$

$$K_2 = \frac{(\delta_1+\delta_2)\left(a+(1+bI^*)^2(\theta+d+3\vartheta)\right)+3\vartheta\left(a+(d+\theta+2\vartheta)(bI^*+1)^2\right)}{(1+bI^*)^2},$$

$$K_3 = \vartheta\left((\delta_1+\delta_2)\left(2a+(1+bI^*)^2(2(d+\theta)+3\vartheta)\right)+\vartheta\left(3a+(bI^*+1)^2(3(d+\theta)+4\vartheta)\right)\right),$$

$$K_4 = \frac{\vartheta^2(\delta_1+\delta_2+\vartheta)\left(a+(d+\theta+\vartheta)(1+bI^*)^2\right)}{(1+bI^*)^2},$$

$$K_5 = \frac{\beta_3\left(-A_P^*+\varepsilon I^{*2}+I^*\right)+\beta_2\left(-A_F^*+\varepsilon I^{*2}+I^*\right)+\beta_1\left(-S^*+\varepsilon I^{*2}+I\right)}{(\varepsilon I^*+1)^2},$$

$$\begin{aligned}K_6 = \frac{1}{(1+bI^*)^2(1+\varepsilon I^*)^2}&(\beta_3(I^*(\varepsilon I^*+1)\left(a+(d+\theta+3\vartheta)(1+bI^*)^2\right)+(1+bI^*)^2((\delta_1+\delta_2)I^*(1+\varepsilon I^*)\\&-A_P^*(\delta_1+\delta_2+3\vartheta)))+\beta_2(I^*(\varepsilon I^*+1)\left(a+(1+bI^*)^2(\theta+d+3\vartheta)\right)+(1+bI^*)^2((\delta_1+\delta_2)\\&\times I^*(\varepsilon I^*+1)-A_F^*(\delta_1+\delta_2+3\vartheta)))+\beta_1(aI^*(\varepsilon I^*+1)+(1+bI^*)^2(I^*(1+\varepsilon I^*)(\theta+d+3\vartheta)\\&-3S^*\vartheta)-(\delta_1+\delta_2)S^*(bI^*+1)^2)),\end{aligned}$$

$$\begin{aligned}K_7 = \frac{1}{(1+bI^*)^2(1+\varepsilon I^*)^2}&(\beta_3(I^*(1+\varepsilon I^*)((\delta_1+\delta_2)\left(a+(1+bI^*)^2(\theta+d+2\vartheta)\right)+\vartheta(2a+(1+bI^*)^2\\&\times(2(d+\theta)+3\vartheta)))-\vartheta A_P^*(1+bI^*)^2(2\delta_1+2\delta_2+3\vartheta))+\beta_2(I^*(\varepsilon I^*+1)((\delta_1+\delta_2)\\&\times\left(a+(1+bI^*)^2(\theta+d+2\vartheta)\right)+\vartheta\left(2a+(1+bI^*)^2(2(d+\theta)+3\vartheta)\right))-\vartheta A_F^*(bI^*+1)^2\\&(2\delta_1+2\delta_2+3\vartheta))+\beta_1\vartheta(2aI^*(\varepsilon I^*+1)+(bI^*+1)^2(I^*(\varepsilon I^*+1)(2(d+\theta)+3\vartheta)-3S^*\vartheta)\\&-2(\delta_1+\delta_2)S^*(bI^*+1)^2)),\end{aligned}$$

$$\begin{aligned}K_8 = \frac{\vartheta}{(bI^*+1)^2(\varepsilon I^*+1)^2}&((\delta_1+\vartheta+\delta_2)(\beta_3(I^*(\varepsilon I^*+1)\left(a+(1+bI^*)^2(\theta+d+\vartheta)\right)-\vartheta A_P^*(1+bI^*)^2)\\&+\beta_2\left(I^*\left(a+(bI^*+1)^2(\theta+d+\vartheta)\right)(1+\varepsilon I^*)-\vartheta A_F^*(1+bI^*)^2\right))+\beta_1\vartheta(aI^*(1+\varepsilon I^*)\\&+(1+bI^*)^2(I^*(\theta+d+\vartheta)(\varepsilon I^*+1)-S^*\vartheta)-(\delta_1+\delta_2)S^*(1+bI^*)^2)),\end{aligned}$$

$$\begin{aligned}K_9 = \frac{1}{(1+\varepsilon I^*)^3}&(I^*(\beta_1\left(\beta_3\left(-A_P^*-S^*+\varepsilon I^{*2}+I^*\right)+\beta_2\left(-A_F^*-S^*+\varepsilon I^{*2}+I^*\right)\right)\\&+\beta_2\beta_3\left(-A_P^*-A_F^*+\varepsilon I^{*2}+I^*\right))^3),\end{aligned}$$

$$K_{10} = \frac{I^*}{(1+\varepsilon I^*)^3(1+bI^*)^2}(\beta_1(\beta_3((1+\varepsilon I^*)aI^* - 2\vartheta A_P^*(1+bI^*)^2 + (1+bI^*)^2(I^*(\theta+d+2\vartheta)\times$$
$$(1+\varepsilon I^*) - 2S^*\vartheta) - \delta_1 S^*(1+bI^*)^2) + \beta_2((1+\varepsilon I^*)aI^* - 2\vartheta A_F^*(1+bI^*)^2 +$$
$$(1+bI^*)^2(I^*(1+\varepsilon I^*)(\theta+d+2\vartheta) - 2S^*\vartheta) - \delta_2 S^*(1+bI^*)^2)) +$$
$$\beta_2\beta_3((1+\varepsilon I^*)I^*\left(a+(1+bI^*)^2(\theta+d+2\vartheta)\right) + (1+bI^*)^2((\delta_1+\delta_2)\times I^*(\varepsilon I^*+1) -$$
$$(\delta_1+\delta_2+2\vartheta)(A_P^*+A_F^*)))),$$
$$K_{11} = \frac{I^*}{(1+\varepsilon I^*)^3(1+bI^*)^2}(\beta_1\vartheta(\beta_3((1+\varepsilon I^*)aI^* - \vartheta A_P^*(1+bI^*)^2 + (1+bI^*)^2(I^*(1+\varepsilon I^*)\times$$
$$(d+\theta+\vartheta) - S^*\vartheta) - \delta_1 S^*(bI^*+1)^2) + \beta_2(aI^*(\varepsilon I^*+1) - \vartheta A_F^*(1+bI^*)^2 +$$
$$(1+bI^*)^2(I^*(1+\varepsilon I^*)(\theta+d+\vartheta) - S^*\vartheta) - \delta_2 S^*(bI^*+1)^2)) +$$
$$\beta_2\beta_3(\delta_1+\delta_2+\vartheta)(I^*(1+\varepsilon I^*)\left(a+(1+bI^*)^2(\theta+d+\vartheta)\right) - \vartheta(1+bI^*)^2(A_P^*+A_F^*))),$$
$$K_{12} = -\frac{\beta_1\beta_2\beta_3 S^* I^{*2}(A_P^*+A_F^*+S^*-I^*(1+\varepsilon I^*))}{(1+\varepsilon I^*)^4},$$
$$K_{13} = \frac{\beta_1\beta_2\beta_3 S^* I^{*2}\left(\begin{smallmatrix}(1+\varepsilon I^*)aI^* - \vartheta(1+bI^*)^2(A_P^*+A_F^*) + \\ (1+bI^*)^2(I^*(\theta+d+\vartheta)(1+\varepsilon I^*) - S^*\vartheta)\end{smallmatrix}\right)}{(bI^*+1)^2(\varepsilon I^*+1)^4}.$$

The coefficients of the characteristic equation (6.36) are obtained as follows:

$$Q_0 = \vartheta^2(\delta_1+\delta_2+\vartheta)(d+\theta+\vartheta) + \frac{I^*\vartheta((\beta_2+\beta_3)(\delta_1+\delta_2+\vartheta)+\beta_1\vartheta)(d+\theta+\vartheta)}{\varepsilon I^*+1}\times$$
$$\frac{1}{(\varepsilon I^*+1)^3}\big(I^*(\beta_2\beta_3(-\vartheta)(\delta_1+\delta_2+\vartheta)(A_P^*+A_F^*) - \beta_1(\beta_2\Big(\vartheta^2 A_F^* + S^*\big(\vartheta(\delta_2+\vartheta) -$$
$$\beta_3 I^{*2}(d+\theta+\vartheta)\big)\Big) + \beta_3\vartheta(\vartheta A_P^* + S^*(\delta_1+\vartheta)))))\frac{1}{(\varepsilon I^*+1)^2}\big((\delta_1+\delta_2+\vartheta)(\beta_2\vartheta^2(-A_F^*) -$$
$$\beta_3\left(\vartheta^2 A_P^* - \beta_2 I^{*2}(d+\theta+\vartheta)\right)) + \beta_1\vartheta\Big(\beta_2 I^{*2}(d+\theta+\vartheta) + \beta_3 I^{*2}(d+\theta+\vartheta) -$$
$$S^*\vartheta(\delta_1+\delta_2+\vartheta)\Big)) - \frac{\beta_1\beta_2\beta_3 S^* I^{*2}\vartheta(A_P^*+A_F^*+S^*)}{(\varepsilon I^*+1)^4} + \frac{1}{(bI^*+1)^2(\varepsilon I^*+1)^3}\times$$
$$\big(a(\beta_1 I^*(\beta_2 I^*(\beta_3 S^* I^* + \varepsilon I^*\vartheta + \vartheta) + \vartheta(\varepsilon I^*+1)(\varepsilon I^*\vartheta + \beta_3 I^* + \vartheta)) +$$
$$(\delta_1+\delta_2+\vartheta)(\varepsilon I^*+1)(I^*\vartheta\varepsilon + \vartheta + \beta_2 I^*)(\varepsilon\vartheta I^* + \vartheta + \beta_3 I^*))\big),$$

$$
\begin{aligned}
Q_1 = & \frac{1}{(bI^*+1)^2(1+\varepsilon I^*)^4}\big(\beta_1(\beta_2 I^*((1+\varepsilon I^*)(aI^*(1+\varepsilon I^*)+(1+bI^*)^2(I^*(1+\varepsilon I^*)(d+\theta+2\vartheta)-2\vartheta S^*)) \\
& -(bI^*+1)^2(\beta_3 S^* I^*(A_P^*+S^*-I^*(\varepsilon I^*+1))+A_F^*(\beta_3 S^* I^*+2(\varepsilon I^*\vartheta+\vartheta))+\delta_2 S^*(\varepsilon I^*+1))) \\
& +(\varepsilon I^*+1)(\beta_3 I^*((1+\varepsilon I^*)aI^*-2\vartheta A_P^*(1+bI^*)^2+(1+bI^*)^2(I^*(1+\varepsilon I^*)(\theta+d+2\vartheta)-2S^*\vartheta) \\
& -(1+bI^*)^2\delta_1 S^*)+\vartheta(1+\varepsilon I^*)(2aI^*(1+\varepsilon I^*)+(1+bI^*)^2(I^*(1+\varepsilon I^*)(2(d+\theta)+3\vartheta)-3\vartheta S^*) \\
& -2(\delta_1+\delta_2)S^*(1+bI^*)^2)))+(1+\varepsilon I^*)(\beta_2(I^*(\beta_3(I^*(\varepsilon I^*+1)\left(a+(1+bI^*)^2(\theta+d+2\vartheta)\right) \\
& +(1+bI^*)^2(1+\varepsilon I^*)((\delta_1+\delta_2)I^*-A_P^*(\delta_1+\delta_2+2\vartheta)))+(\varepsilon I^*+1)^2((\delta_1+\delta_2) \\
& \times\left(a+(1+bI^*)^2(\theta+d+2\vartheta)\right)+\vartheta(2a+(bI^*+1)^2(2(d+\theta)+3\vartheta)))) \\
& -A_F^*(bI^*+1)^2(\vartheta(2\delta_1+2\delta_2+3\vartheta)(\varepsilon I^*+1)+\beta_3 I^*(\delta_1+\delta_2+2\vartheta)))+(\varepsilon I^*+1)(\beta_3(I^*(\varepsilon I^*+1) \\
& \times((\delta_1+\delta_2)(a+(1+bI^*)^2(d+\theta+2\vartheta))+\vartheta\left(2a+(1+bI^*)^2(2(d+\theta)+3\vartheta)\right))-\vartheta A_P^*(bI^*+1)^2 \\
& \times(2\delta_1+2\delta_2+3\vartheta))+\vartheta(\varepsilon I^*+1)^2\times((\delta_1+\delta_2)\left(2a+(1+bI^*)^2(2(d+\theta)+3\vartheta)\right) \\
& +\vartheta\left(3a+(1+bI^*)^2(4\vartheta+3(\theta+d))\right)))))\big),
\end{aligned}
$$

$$
\begin{aligned}
Q_2 = & \frac{1}{(1+\varepsilon I^*)^3(1+bI^*)^2}\big(\beta_1((1+\varepsilon I^*)\left((1+\varepsilon I^*)aI^*+(1+bI^*)^2(I^*(1+\varepsilon I^*)(\theta+d+3\vartheta)-3S^*\vartheta)\right) \\
& -(bI^*+1)^2(\beta_3 I^*(A_P^*+S^*-I^*(\varepsilon I^*+1))+\beta_2 I^*(A_F^*+S^*-I^*(\varepsilon I^*+1))+(\delta_1+\delta_2)S^*(\varepsilon I^*+1))) \\
& +\beta_2(I^*(1+\varepsilon I^*)^2\left(a+(1+bI^*)^2(d+\theta+3\vartheta)\right)+(1+bI^*)^2(I^*(\beta_3\left(-A_P^*+\varepsilon I^{*2}+I^*\right) \\
& +(\delta_1+\delta_2)(\varepsilon I^*+1)^2)-A_F^*((\delta_1+\delta_2+3\vartheta)(\varepsilon I^*+1)+\beta_3 I^*)))+(\varepsilon I^*+1) \\
& \times(\beta_3(I^*(1+\varepsilon I^*)\left(a+(1+bI^*)^2(\theta+3\vartheta+d)\right)+(1+bI^*)^2((\delta_1+\delta_2)I^*(\varepsilon I^*+1) \\
& -A_P^*(\delta_1+\delta_2+3\vartheta)))+(\delta_1+\delta_2)(\varepsilon I^*+1)^2\left(a+(1+bI^*)^2(\theta+d+3\vartheta)\right)))\big) \\
& +3\vartheta\left(\frac{a}{(1+bI^*)^2}+d+\theta+2\vartheta\right), \\
Q_3 = & \frac{a}{(bI^*+1)^2}+\frac{\beta_3\left(-A_P^*+\varepsilon I^{*2}+I^*\right)+\beta_2\left(-A_F^*+\varepsilon I^{*2}+I^*\right)+\beta_1\left(-S+\varepsilon I^{*2}+I^*\right)}{(\varepsilon I^*+1)^2} \\
& +d+\delta_1+\delta_2+\theta+4\vartheta.
\end{aligned}
$$

Chapter 7

A deterministic time-delayed SVIRS epidemic model with incidences and saturated treatment

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A novel nonlinear delayed susceptible–vaccinated–infected–recovered–susceptible (SVIRS) epidemic model with a Holling type II incidence rate for fully susceptible and vaccinated classes, a saturated treatment rate, and an imperfect vaccine given to susceptibles is proposed herein. Analysis of the model shows that it exhibits two equilibria, namely disease-free and endemic. The basic reproduction number R_0 is derived, and it is demonstrated that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ and linearly neutrally stable when $R_0 = 1$. Furthermore, bifurcation analysis is performed for the undelayed model, revealing backward and forward bifurcation when the basic reproduction number varies from unity. The stability behavior of the endemic equilibrium is also discussed, showing that oscillatory and periodic solutions may appear via Hopf bifurcation when regarding delay as the bifurcation parameter. Moreover, numerical simulations are carried out to illustrate the theoretical findings.

7.1 Introduction

Vaccination is one of the most cost-effective means to prevent and control infectious diseases. It remains a considerable challenge to achieve desirable vaccination coverage

for herd immunity to be in effect. The vaccine may be imperfect as there can exist unwanted, adverse side effects of various degrees, or the vaccination can only confer partial protection against the disease. In mathematical epidemiology literature, many studies have dealt with epidemic models, including imperfect vaccination (see, just to specify a couple of studies, [92, 97, 110, 126]). With imperfect vaccination, the outcome of an epidemic model may lead to backward bifurcation under particular conditions because vaccinated individuals may return to the susceptible pool or become directly infected by transmission. Backward bifurcation thus plays a relevant role in disease control and eradication. Indeed, it is well known that, in classical disease transmission models, a necessary condition for disease eradication is that the basic reproductive number R_0 [33] be less than unity. This type of bifurcation is known as forward bifurcation, where for $R_0 < 1$ the disease-free equilibrium (DFE) is the only equilibrium and is asymptotically stable, while for $R_0 > 1$ the DFE is unstable and only one asymptotically stable, endemic equilibrium exists. However, via the occurrence of backward bifurcation, an endemic equilibrium may also exist even when the basic reproduction number R_0 is less than unity. From the public viewpoint, the occurrence of backward bifurcation may have significant health implications regarding disease elimination. In literature, many epidemic models, including backward bifurcation, have been studied for both generic and specific diseases [40, 47].

To determine the dynamics of epidemic models, the incidence rate (the rate of new infections) plays a major role in modeling infectious diseases. Capasso and Serio [12] introduced the nonlinear incidence rate in the form $g(I)S$ with $g'(I) < 0$, which allows the introduction of some "psychological" effects. Capasso and Serio motivated their formulation with behavioral changes: in epochs of high prevalence, the perceived risk of infection might become very large, yielding dramatic changes in individuals' behavior and reducing the actual risk of getting the disease (as widely discussed in [68]). Numerous authors have focused on the significance of considering nonlinear incidence rates in the study of the transmission dynamics of infectious diseases (see, for example, [9–12, 61, 62, 73, 131, 132, 135]). Li et al. [73] proposed a SIR model with a nonlinear incidence rate given by

$$f(S,I) = \frac{\beta SI}{1+\gamma I}.$$

In this incidence rate, the number of effective contacts between infective and susceptible individuals may saturate at high infective levels due to overcrowding of infective indi-

viduals. The delay differential equation plays a significant role in estimating past and ongoing epidemics and the structure of future-focused control interventions. In mathematical epidemiology literature, many studies have dealt with the time delay (called the latent or incubation period) (see, e.g., [25, 44, 76, 131, 132, 135]) and studied its impact on their models. Motivated by the work of Capasso and Serio [12], d'Onofrio and Manfredi [68], and Li et al. [73], a saturated nonlinear incidence rate, reflecting the psychological or inhibition effect, with the inclusion of a time delay as the latent period, is considered herein.

The loss of quality of life and economic productivity due to severe illness further increases the societal cost. Therefore, it is very important to prevent and reduce the spread of infectious diseases among people. Treatment is the key to fight many infectious diseases. Therefore, Holling type II treatment rate is considered herein, and its effect on the present epidemic model is studied (the detailed explanation is given in Chapter two of this thesis).

The purpose of this chapter is to study the effect of saturated incidence, an imperfect vaccine, and saturated treatment to achieve substantial progress in implementing measures to prevent and control infectious diseases among people. For this, a compartmental susceptible–vaccinated–infected–recovered–susceptible (SVIRS) epidemic model with a saturated incidence rate and including a time delay (representing the latent period) and saturated treatment rate is considered. Qualitative analysis is performed through the stability and bifurcation theory approach using center manifold theory, revealing the existence of backward, forward, and Hopf bifurcations under certain conditions, which enrich the dynamics of infectious diseases among humans.

7.2 The model and its basic properties

Assume that the epidemiological status of the total population $N(t)$ of individuals can be identified by dividing them into susceptibles $S(t)$, vaccinated $V(t)$, infectives $I(t)$, and recovered $R(t)$ classes. Individuals move from one state to another as their status concerning the disease evolves. A is the recruitment rate of susceptibles and hence entering the susceptible state. Susceptible individuals are vaccinated at a rate of δ and enter the state $V(t)$. The term $f(S(t-\rho),I(t-\rho)) = (\beta S(t-\rho)I(t-\rho))/(1+\alpha I(t-\rho))$

is the Holling type II functional response representing the incidence of infection among susceptibles, where β is the force of infection, α describes the inhibition measures taken by the infected, and the time delay parameter ρ represents the latent period. The protection provided by an imperfect vaccine is only partial, so some individuals can catch the disease when they come into contact with infected individuals. Therefore, it is assumed that γ is the rate at which vaccinated individuals become infected when coming into contact with infected individuals. This occurs due to the imperfect nature of the vaccine, which leaves a percentage of the susceptibles unprotected even if vaccinated. Assume that $\beta > \gamma$, as it is expected that the vaccine will be at least partly effective in preventing infection, yielding a reduction in the force of infection. The term $g(V(t-\rho),I(t-\rho)) = (\gamma\, V(t-\rho)I(t-\rho))/(1+\alpha I(t-\rho))$ represents the incidence of infection among vaccinated individuals who move from state $V(t)$ to state $I(t)$. The term $(aI)/(1+bI)$, where a is the treatment (cure) rate, and b is a rate of limitation in medical resources, describes the treated individuals who recover and thus move from state $I(t)$ to $R(t)$. Also, it is assumed that recovered individuals become susceptible again, thus the term θR describes recovered individuals who re-enter the class of susceptible individuals. The parameters μ and d are the natural and disease-induced mortality rates, respectively. The parameter ς denotes the recovery rate, hence ςI individuals move from the infected to the recovered class.

Thus, the proposed SVIRS epidemic model consists of the following system of delay differential equations:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= A - \delta S - \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha I(t-\rho)} - \mu S + \theta R,\\
\frac{\mathrm{d}V}{\mathrm{d}t} &= \delta S - \frac{\gamma V(t-\rho)I(t-\rho)}{1+\alpha I(t-\rho)} - \mu V,\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\beta S(t-\rho)I(t-\rho)}{1+\alpha I(t-\rho)} + \frac{\gamma V(t-\rho)I(t-\rho)}{1+\alpha I(t-\rho)} - (\mu+d+\varsigma)I - \frac{aI}{1+bI},\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \varsigma I + \frac{aI}{1+bI} - \theta R - \mu R.
\end{aligned} \tag{7.1}$$

For biological reasons, the initial conditions are nonnegative continuous functions

$$S(\Theta) = \phi_1(\Theta), V(\Theta) = \phi_2(\Theta), I(\Theta) = \phi_3(\Theta), R(\Theta) = \phi_4(\Theta),$$

where $\phi(\Theta) = (\phi_1,\phi_2,\phi_3,\phi_4)^T$ are functions such that $\phi_i(\Theta) \geq 0, (-\rho \leq \Theta \leq 0, i = 1,2,3,4)$. C denotes the Banach space $C\left([-\rho,0],\mathbb{R}^4_+\right)$ of continuous functions mapping the interval

$[-\rho,0]$ into $\mathbb{R}^4_+$ with supremum norm

$$\|\phi\| = \sup_{\Theta\in[-\rho,0]} |\phi(\Theta)|,$$

where $|.|$ is any norm in $\mathbb{R}^4_+$.

The transition diagram of the model (7.1) is shown in Fig. 7.1.

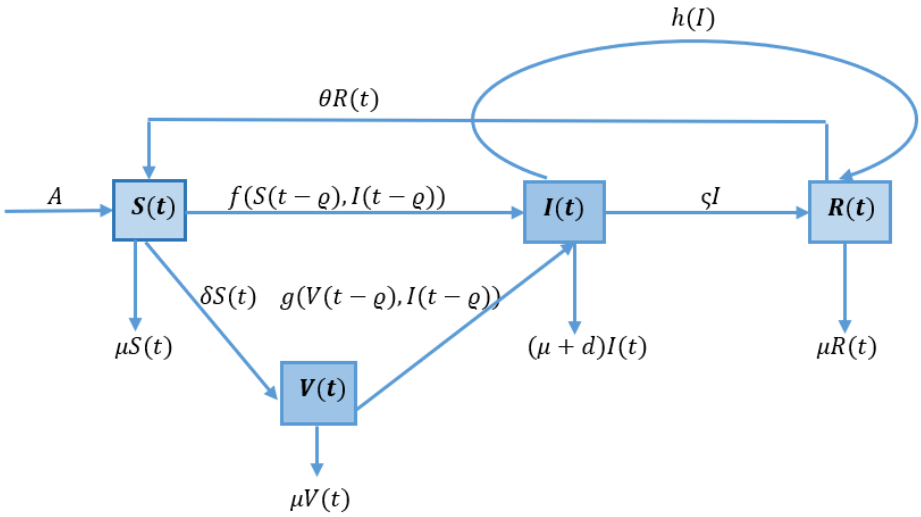


Figure 7.1: Transition diagram of the model (7.1).

The model (7.1) monitors populations. Using Proposition 2.3. in Yang et al. [124] and Proposition 2.1 given in Hattaf et al. [107], it can be checked that all state variables of the model (7.1) are nonnegative; i.e., $(S,V,I,R) \in R^4_+$. For ecological reasons, it is assumed that all the parameters are positive, i.e., A, δ, β, α, μ, θ, γ, d, ς, a, and b are positive.

Lemma 7.2.1. *The compact set*

$$\Omega = \left\{(S(t),V(t),I(t),R(t)) \in \mathbb{R}^4_+ : N(t) = S(t)+V(t)+I(t)+R(t) \leq \frac{A}{\mu}\right\}$$

is invariant for the solutions of the model (7.1).

Proof. The well-posedness of the model is ensured by the continuity of the terms on the right-hand side of the model (7.1) and its derivatives.

Addition of the equations of the model (7.1), yields

$$\frac{\mathrm{d}N}{\mathrm{d}t} = A - \mu N - dI \leq A - \mu N. \tag{7.2}$$

Thus, the invariant region for the existence of the solutions is given as

$$0 < \liminf_{t\to\infty} N(t) \leq \limsup_{t\to\infty} N(t) \leq \frac{A}{\mu}. \tag{7.3}$$

Hence, the solutions of the model (7.1) are closed and bounded. ■

7.3 Equilibria and stability analysis

In this section, the existence of equilibria of the model (7.1) is confirmed. The model has two equilibria, namely,

1. The disease-free equilibrium E_0, discussed in subsection 7.3.1.
2. Endemic equilibrium E_e, discussed in subsection 7.3.2.

7.3.1 The disease-free equilibrium and its stability

Here, it is established that the model (7.1) has a disease-free equilibrium of the form $E_0 = \left(\frac{A}{\mu+\delta}, \frac{\delta A}{\mu(\mu+\delta)}, 0, 0\right)$.

At E_0, the characteristic equation of the linearized model (7.1) is obtained as follows:

$$(\lambda+\mu)(\lambda+\mu+\delta)(\lambda+\mu+\theta)\left(\lambda + (d+\mu+\varsigma+a) - \frac{A(\beta\mu+\gamma\delta)\mathrm{e}^{-\lambda\rho}}{\mu(\delta+\mu)}\right) = 0. \tag{7.4}$$

Eq. (7.4) has real negative roots $\lambda_1 = -\mu$, $\lambda_2 = -\mu-\delta$, $\lambda_3 = -\mu-\theta$, and other roots are the solution of

$$\lambda + (d+\mu+\varsigma+a) - \frac{A(\beta\mu+\gamma\delta)\mathrm{e}^{-\lambda\rho}}{\mu(\delta+\mu)} = 0. \tag{7.5}$$

The term $(A(\mu\beta+\gamma\delta)\mathrm{e}^{-\lambda\rho})/(\mu(\mu+\delta)(d+\mu+\varsigma+a))$ at $\rho = 0$ is defined as the basic reproduction number R_0 of the model (7.1). Therefore, R_0 for the model (7.1) is given as

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

The stability of E_0 is shown as follows.

Theorem 7.3.1. *The disease-free equilibrium* $E_0 = \left(\frac{A}{\mu+\delta}, \frac{\delta A}{\mu(\mu+\delta)}, 0, 0\right)$ *of the model (7.1) is*

1. *Unstable if* $R_0 > 1$
2. *Linearly nuetrally stable if* $R_0 = 1$
3. *Asymptotically stable if* $R_0 < 1$.

Proof. As mentioned above, Eq. (7.4) has real negative roots $\lambda_1 = -\mu,\ \lambda_2 = -\mu - \delta,\ \lambda_3 = -\mu - \theta$, and other roots are the solution of

$$f(\lambda) := \lambda + (d + \mu + \varsigma + a) - \frac{A(\beta\mu + \gamma\delta)\mathrm{e}^{-\lambda\rho}}{\mu(\delta + \mu)} = 0. \tag{7.6}$$

1. Assuming that $R_0 > 1$, then

$$\begin{aligned} f(0) &= d + \mu + \varsigma + a - \frac{A(\beta\mu + \gamma\delta)}{\mu(\delta + \mu)} \\ &= (d + \mu + \varsigma + a)\left(1 - \frac{A(\beta\mu + \gamma\delta)}{\mu(\mu + \delta)(d + \mu + \varsigma + a)}\right) \\ &= (d + \mu + \varsigma + a)(1 - R_0) \\ &< 0. \end{aligned}$$

   i.e., when $R_0 > 1$ then $f(0) < 0$. Also, $f'(\lambda) = 1 + \frac{\rho A(\beta\mu+\gamma\delta)}{\mu(\delta+\mu)}\mathrm{e}^{-\lambda\rho} > 0$ so, $\lim_{\lambda\to\infty} f(\lambda) = +\infty$.

   Hence $f(\lambda) = 0$ and $f'(\lambda) > 0$ imply that there exists a unique positive root of Eq. (7.6) when $R_0 > 1$.

2. If $R_0 = 1$, then $\lambda = 0$ is a simple characteristic root of Eq. (7.6). Let $\lambda = \alpha + \mathrm{i}\omega$ be any of the other solutions of Eq. (7.6), then Eq. (7.6) turns into

$$\alpha + \mathrm{i}\omega + (d + \mu + \varsigma + a) - \frac{A(\beta\mu + \gamma\delta)}{\mu(\delta + \mu)}\mathrm{e}^{-\alpha\rho}(\cos\omega\rho - \mathrm{i}\sin\omega\rho) = 0. \tag{7.7}$$

   Using Euler's formula and separating real and imaginary parts, yields

$$\alpha + d + \mu + \varsigma + a = \frac{A(\beta\mu + \gamma\delta)}{\mu(\delta + \mu)}\mathrm{e}^{-\alpha\rho}\cos\omega\rho, \tag{7.8}$$

$$\omega = -\frac{A(\beta\mu + \gamma\delta)}{\mu(\delta + \mu)}\mathrm{e}^{-\alpha\rho}\sin\omega\rho. \tag{7.9}$$

Note that $R_0 = 1$ implies that $(A(\beta\mu + \gamma\delta))/(\mu(\mu+\delta)) = (d+\mu+\varsigma+a)$. In addition, if there exists a root satisfying both Eqs. (7.8) and (7.9), then this root also satisfies the equation obtained by squaring and adding Eqs. (7.8) and (7.9), thus

$$(\alpha + d + \mu + \varsigma + a)^2 + \omega^2 = (d+\mu+\varsigma+a)^2 \mathrm{e}^{-2\alpha\rho}. \tag{7.10}$$

For Eq. (7.10) to be verified, $\alpha \leq 0$ must apply. Therefore, E_0 is linearly neutrally stable.

3. Let $R_0 < 1$. The goal is to prove that, for any values of the parameters, the roots of the characteristic equation cannot reach the imaginary axis, which means that, for any values of the parameters and all delays ρ, then $\mathrm{Re}(\lambda) < 0$.
   Note that

   $$\mathrm{Re}(\lambda) = \frac{A(\beta\mu+\gamma\delta)\mathrm{e}^{-\mathrm{Re}(\lambda)\rho}\cos(Im\lambda)\rho}{\mu(\delta+\mu)} - (d+\mu+\varsigma+a) < \frac{A(\beta\mu+\gamma\delta)}{\mu(\delta+\mu)} - (d+\mu+\varsigma+a) < 0.$$

   Therefore, all the roots of Eq. (7.6) must have a negative real part. Thus, E_0 is asymptotically stable.

■

Bifurcation analysis

In this section, a qualitative analysis of model (7.1) is performed without delay, i.e., with $\rho = 0$. The stability properties of the model (7.1) without delay are investigated near criticality (i.e., at E_0 and $R_0 = 1$). To this aim, the bifurcation theory approach developed in [40], which is based on the center manifold theory [17], is applied. For this, redefine $S = x_1$, $V = x_2$, $I = x_3$ and $R = x_4$, so that the model (7.1) reduces to

$$\begin{aligned}
\frac{\mathrm{d}x_1}{\mathrm{d}t} &= A - \delta x_1 - \frac{\beta x_1 x_3}{1+\alpha x_3} - \mu x_1 + \theta x_4,\\
\frac{\mathrm{d}x_2}{\mathrm{d}t} &= \delta x_1 - \frac{\gamma x_2 x_3}{1+\alpha x_3} - \mu x_2,\\
\frac{\mathrm{d}x_3}{\mathrm{d}t} &= \frac{\beta x_1 x_3}{1+\alpha x_3} + \frac{\gamma x_2 x_3}{1+\alpha x_3} - (\mu + d + \varsigma)x_3 - \frac{a x_3}{1+b x_3},\\
\frac{\mathrm{d}x_4}{\mathrm{d}t} &= \varsigma x_3 + \frac{a x_3}{1+b x_3} - \theta x_4 - \mu x_4.
\end{aligned} \tag{7.11}$$

observing that $R_0 = 1 \iff \gamma = \gamma^* = \frac{\mu(\mu+\delta)(\mu+d+\varsigma+a) - A\mu\beta}{\delta A}$.

The Jacobian matrix $J(E_0, \gamma^*)$ of the system (7.11) at the disease-free equilibrium E_0

is given by

$$J(E_0,\gamma^*)=\begin{pmatrix} -\delta-\mu & 0 & -\frac{A\beta}{\delta+\mu} & \theta \\ \delta & -\mu & -\frac{A\delta\gamma^*}{\mu(\delta+\mu)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & a+\varsigma & -\theta-\mu \end{pmatrix} \tag{7.12}$$

The eigenvalues of the Jacobian matrix $J(E_0,\gamma^*)$ are given by $\lambda_1=0,\lambda_2=-\mu,\lambda_3=-\delta-\mu$ and $\lambda_4=-\theta-\mu$.

Thus, $\lambda_1=0$ is a simple zero eigenvalue, and other eigenvalues are real and negative. Hence, when $\gamma=\gamma^*$ (or equivalently when $R_0=1$), the disease-free equilibrium E_0 is a nonhyperbolic equilibrium.

The right eigenvector $\boldsymbol{u}=(u_1,u_2,u_3,u_4)^T$ of (7.12) associated with $\lambda_1=0$ is given by $J(E_0,\gamma^*)\cdot\boldsymbol{u}=0$. Thus,

$$\begin{aligned} u_1&=\frac{\theta(a+\varsigma)(\delta+\mu)-A\beta(\theta+\mu)}{(a+\varsigma)(\delta+\mu)^2},\\ u_2&=\frac{\delta\theta}{\mu(\delta+\mu)}-\frac{(\theta+\mu)\left((\delta+\mu)^2(a+d+\mu+\varsigma)-A\beta\mu\right)}{\mu(a+\varsigma)(\delta+\mu)^2},\\ u_3&=\frac{\theta+\mu}{a+\varsigma},\\ u_4&=1. \end{aligned}$$

The left eigenvector $\boldsymbol{w}=(w_1,w_2,w_3,w_4)$ of (7.12) associated with $\lambda_1=0$ is given by $\boldsymbol{w}\cdot J(E_0,\gamma^*)=0$. We obtain

$$\boldsymbol{w}=(0,0,1,0)\,.$$

Let f_k's denote the right-hand side of the model system (7.11). The coefficients a_1 and b_1 defined in Theorem 4.1 of Castillo-Chavez and Song [40] are given by:

$$\begin{aligned} a_1&=\sum_{k,i,j=1}^{4} w_k u_i u_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_{E_0},\\ b_1&=\sum_{k,i=1}^{4} w_k u_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \beta^*}\right)_{E_0}. \end{aligned}$$

Consideration of only nonzero partial derivative associated with functions f_k's evaluated at E_0 gives

$\left(\frac{\partial^2 f_3}{\partial x_1\partial x_3}\right)_{E_0}=\beta$, $\left(\frac{\partial^2 f_3}{\partial x_2\partial x_3}\right)_{E_0}=\gamma^*$, $\left(\frac{\partial^2 f_3}{\partial x_3\partial x_1}\right)_{E_0}=\beta$, $\left(\frac{\partial^2 f_3}{\partial x_3\partial x_2}\right)_{E_0}=\gamma^*$, $\left(\frac{\partial^2 f_3}{\partial x_3^2}\right)=2ab-\frac{2\alpha\beta A}{\mu+\delta}$.

Thus, the bifurcation coefficients a_1 and b_1 can be computed as

$$\begin{aligned}
a_1 &= -\frac{2(\theta+\mu)}{A\delta(a+\varsigma)^2(\delta+\mu)}(-A(\theta+\mu)(a(\delta(b-\alpha)(\delta+\mu)+\beta(\delta+2\mu))+(d+\mu+\varsigma)(\beta(\delta+2\mu)\\
&\quad -\alpha\delta(\delta+\mu)))+(\delta+\mu)(a+d+\mu+\varsigma)(\mu(a(\delta+\theta+\mu)+\delta(\theta+\mu+\varsigma)+(\theta+\mu)(\mu+\varsigma))\\
&\quad +d(\delta+\mu)(\theta+\mu))+A^2\beta^2(\theta+\mu)),\\
&= -\frac{2(\theta+\mu)}{A\delta(a+\varsigma)^2(\delta+\mu)}\eta(\beta),\\
b_1 &= \frac{A(\mu+\theta)}{(a+\varsigma)(\mu+\delta)}.
\end{aligned}$$

where,

$$\begin{aligned}
\eta(\beta) &= A^2\beta^2(\theta+\mu)+\beta(-aA(\delta+2\mu)(\theta+\mu)-A(\delta+2\mu)(\theta+\mu)(d+\mu+\varsigma))\\
&\quad -aA\delta(b-\alpha)(\delta+\mu)(\theta+\mu)+(\delta+\mu)(a+d+\mu+\varsigma)(\mu(a(\delta+\theta+\mu)+\delta(\theta+\mu+\varsigma)\\
&\quad +(\theta+\mu)(\mu+\varsigma))+d(\delta+\mu)(\theta+\mu))+\alpha A\delta(\delta+\mu)(\theta+\mu)(d+\mu+\varsigma).
\end{aligned}$$

$\eta(\beta)$ can be written as

$$\eta(\beta) = A_1\beta^2+A_2\beta+A_3, \tag{7.13}$$

where,

$$\begin{aligned}
A_1 &= A^2(\theta+\mu),\\
A_2 &= -A(\delta+2\mu)(\theta+\mu)(a+d+\mu+\varsigma),\\
A_3 &= (\delta+\mu)(aA\delta(\alpha-b)(\theta+\mu)+(a+d+\mu+\varsigma)(\mu(a(\delta+\theta+\mu)+\varsigma(\delta+\theta+\mu)+(\delta+\mu)(\theta+\mu))\\
&\quad +d(\delta+\mu)(\theta+\mu))+\alpha A\delta(\theta+\mu)(d+\mu+\varsigma)).
\end{aligned}$$

Note that b_1 is always positive. Thus, according to Theorem 4.1 of Castillo-Chavez and Song [40], the sign of a_1, and hence the sign of $\eta(\beta)$, determines the local dynamics around the disease-free equilibrium. It is noticed that, $A_1 > 0$ and $A_2 < 0$. The discriminant D of (7.13) is obtained as

$$\begin{aligned}
D &= A_2^2-4A_1A_3\\
&= A^2\delta(\theta+\mu)\Big(a^2\left(\delta(\theta-3\mu)-4\mu^2\right)+2a\big(2A(b-\alpha)(\delta+\mu)(\theta+\mu)-(d+\mu)(\delta(\theta+3\mu)+\\
&\quad 2\mu(\theta+2\mu))+\varsigma\left(\delta(\theta-3\mu)-4\mu^2\right)\big)-(d+\mu+\varsigma)\big((\theta+\mu)(4\alpha A(\delta+\mu)+d(3\delta+4\mu)+\\
&\quad \mu(3\delta+4\mu))+\varsigma\left(-\delta\theta+3\delta\mu+4\mu^2\right)\big)\Big).
\end{aligned} \tag{7.14}$$

Let β_1 and β_2 are the two real positive roots of the equation $A_1\beta^2+A_2\beta+A_3=0$, given by

$$\beta_1=\frac{-A_2-\sqrt{D}}{2A_1},\quad \text{and}\quad \beta_2=\frac{-A_2+\sqrt{D}}{2A_1}.$$

By applying Theorem 4.1 [40], the occurrence of forward and backward bifurcations is discussed separately below:

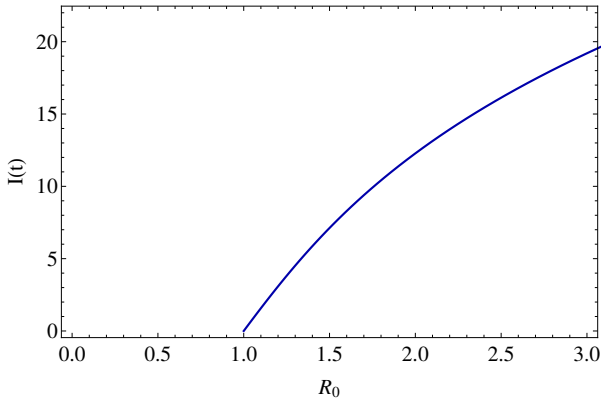
Backward bifurcation The model (7.11) exhibits a backward bifurcation if $\eta(\beta)<0$. Hence, the following conditions allow the existence of backward bifurcation around E_0:

$$\begin{cases} D>0, \\ \beta_1<\beta<\beta_2. \end{cases} \tag{7.15}$$

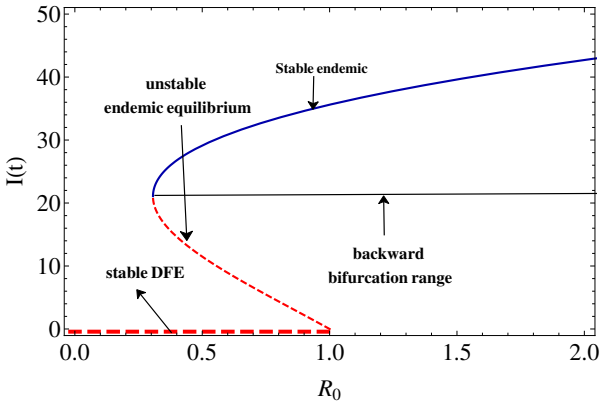
Forward bifurcation: The forward bifurcation occurs if $\eta(\beta)>0$. Thus, the conditions for the existence of forward bifurcation are as follows:

$$\begin{cases} D>0, \\ \beta<\beta_1 \quad \text{or} \quad \beta>\beta_2. \end{cases} \tag{7.16}$$

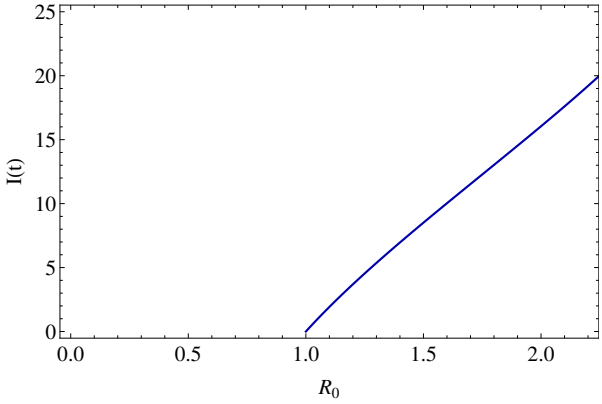
The forward and backward bifurcations are illustrated numerically in Fig. (7.2) for the set of parameters values: $A=5,\ \alpha=0.01,\ \mu=0.01,\ \theta=0.04,\ d=0.01,\ \varsigma=0.01,\ a=2,\ b=.1,\ \delta=0.01$, the discriminant D, obtained in Eq. (7.14), is evaluated as $D=0.0000278476>0$. According to the theoretical results, stated in inequalities (7.15), the backward bifurcation occurs for $D>0$, and $\beta_1=0.00397917<\beta<\beta_2=0.00820083$. Setting $\beta=0.007$, the model (7.11) exhibits a backward bifurcation, as shown in Fig. (7.2.2). This figure shows that the reduction of the value of R_0 below unity does not guarantee the elimination of the infection. This implies a range that exhibits a region of coexistence of the disease-free equilibrium and two endemic equilibria: a smaller endemic equilibrium, i.e., with a small number of infected individuals, which is unstable, and a larger one, i.e., with a larger number of infected individuals, which is stable. According to the inequalities given in (7.16), the forward bifurcation occurs when $D>0$ and if β is sufficiently small or sufficiently large, i.e., if either $\beta<\beta_1$ or $\beta>\beta_2$ holds. Fig. (7.2.1) is plotted for $\beta=0.003<\beta_1=0.00397917$, and Fig. (7.2.3) for $\beta=0.009>\beta_2$, revealing that, when $R_0<1$, the disease-free equilibrium is stable, while if R_0 crosses unity, the model admits a stable unique endemic equilibrium.



7.2.1: Forward bifurcation ($\beta = 0.003$).



7.2.2: Backward bifurcation ($\beta = 0.007$).



7.2.3: Forward bifurcation ($\beta = 0.009$).

Figure 7.2: Graphs depicting the forward and backward bifurcation.

7.3.2 Endemic equilibrium

Stability analysis of the endemic equilibrium E_e for the model (7.1) is now carried out. First, equate the right-hand terms of the model (7.1) to zero to establish the existence of endemic equilibrium, as given below:

$$A - \delta S^* - \frac{\beta S^* I^*}{1+\alpha I^*} - \mu S^* + \theta R^* = 0, \tag{7.17}$$

$$\delta S^* - \frac{\gamma V^* I^*}{1+\alpha I^*} - \mu V^* = 0, \tag{7.18}$$

$$\frac{\beta S^* I^*}{1+\alpha I^*} + \frac{\gamma V^* I^*}{1+\alpha I^*} - (\mu + d + \varsigma) I^* - \frac{a I^*}{1+b I^*} = 0, \tag{7.19}$$

$$\varsigma I^* + \frac{a I^*}{1+b I^*} - \theta R^* - \mu R^* = 0. \tag{7.20}$$

The solution of these algebraic equations yields the endemic equilibrium $E_e = (S^*, V^*, I^*, R^*)$ as

$$
\begin{aligned}
S^* &= \frac{(\alpha I^*+1)(\theta I^*(a+bI^*\varsigma+\varsigma)+A(bI^*+1)(\theta+\mu))}{(bI^*+1)(\theta+\mu)(\delta+\mu+I^*(\alpha(\delta+\mu)+\beta))},\\
V^* &= \frac{\delta(\alpha I^*+1)^2(\theta I^*(a+bI^*\varsigma+\varsigma)+A(bI^*+1)(\theta+\mu))}{(bI^*+1)(\theta+\mu)(\mu+I^*(\alpha\mu+\gamma))(\delta+\mu+I^*(\alpha(\delta+\mu)+\beta))},\\
R^* &= \frac{I^*(a+bI^*\varsigma+\varsigma)}{(bI^*+1)(\theta+\mu)},
\end{aligned}
$$

where, I^* is given by the real positive solutions of the equation

$$A_3 I^{*3} + A_2 I^{*2} + A_1 I^* + A_0 = 0, \tag{7.21}$$

with the coefficients A_3, A_2, A_1 and A_0 given as

$$
\begin{aligned}
A_3 &= -b(\mu(\beta(\alpha\mu+\gamma)(\theta+\mu+\varsigma)+\alpha(\alpha(\delta+\mu)(\theta+\mu)(\mu+\varsigma)+\gamma\varsigma(\delta+\theta+\mu)\\
&\quad +\gamma(\delta+\mu)(\theta+\mu)))+d(\theta+\mu)(\alpha\mu+\gamma)(\alpha(\delta+\mu)+\beta)),\\
A_2 &= -\mu(a(\beta(\alpha\mu+\gamma)+\alpha(\alpha(\delta+\mu)(\theta+\mu)+\gamma(\delta+\theta+\mu)))+\varsigma(\alpha(\alpha(\delta+\mu)(\theta+\mu)\\
&\quad +\gamma(\delta+\theta+\mu))+\beta(\mu(\alpha+b)+\gamma)+b(2\alpha(\delta+\mu)(\theta+\mu)+\gamma(\delta+\theta+\mu)))\\
&\quad +(\theta+\mu)(\beta(\mu(\alpha+b)+\gamma)+(\delta+\mu)(\gamma(\alpha+b)+\alpha\mu(\alpha+2b))))+Ab(\theta+\mu)(\beta(\alpha\mu+\gamma)\\
&\quad +\alpha\gamma\delta)-d(\theta+\mu)(\beta(\mu(\alpha+b)+\gamma)+(\delta+\mu)(\gamma(\alpha+b)+\alpha\mu(\alpha+2b))),\\
A_1 &= -\mu(a(2\alpha(\delta+\mu)(\theta+\mu)+\beta\mu+\gamma(\delta+\theta+\mu))+\varsigma((2\alpha+b)(\delta+\mu)(\theta+\mu)+\beta\mu\\
&\quad +\gamma(\delta+\theta+\mu))+(\theta+\mu)((\delta+\mu)(\mu(2\alpha+b)+\gamma)+\beta\mu))+A(\theta+\mu)(\beta(\mu(\alpha+b)+\gamma)\\
&\quad +\gamma\delta(\alpha+b))-d(\theta+\mu)((\delta+\mu)(\mu(2\alpha+b)+\gamma)+\beta\mu),\\
A_0 &= \mu(\delta+\mu)(\theta+\mu)(a+d+\mu+\varsigma)(R_0-1).
\end{aligned}
$$

Theorem 7.3.2. *If $R_0 > 1$, then there is either one unique or three positive endemic equilibria, if all equilibria are simple roots.*

Proof. Suppose $R_0 > 1$. Eq. (7.21) gives a third-degree polynomial in I^*:

$$F(I^*) = A_3 I^{*3} + A_2 I^{*2} + A_1 I^* + A_0.$$

The leading coefficient of I^* is A_3, which is negative. Hence

$$\lim_{I^*\to\infty} F(I^*) = -\infty.$$

Also, $F(0) = A_0$ and $A_0 > 0$ if $R_0 > 1$. $F(I^*)$ is a continuous function of I^*, and using the fundamental theorem of algebra, this polynomial can have at most three real roots. ■

The case of a unique endemic equilibrium only is considered herein. (H1): Suppose that $R_0 > 1$. It is noted that A_3 is negative and A_0 is positive. For the existence of a unique endemic equilibrium, the following possibilities for the signs of A_1 and A_2 exist:

$$\begin{aligned} &(i)\ A_1 > 0, \text{ and } A_2 > 0, \\ &(ii)\ A_1 > 0, \text{ and } A_2 < 0, \\ &(iii)\ A_1 < 0, \text{ and } A_2 < 0. \end{aligned} \tag{7.22}$$

Now, the local stability of the endemic equilibrium of the model (7.1) is discussed.

The characteristic equation of the model (7.1) at E_e is a fourth-degree transcendental equation:

$$\lambda^4 + (p_0\lambda^3 + q_0\lambda^2 + r_0\lambda + s_0) + (p_1\lambda^3 + q_1\lambda^2 + r_1\lambda + s_1)\mathrm{e}^{-\lambda\rho} + (q_2\lambda^2 + r_2\lambda + s_2)\mathrm{e}^{-2\lambda\rho} = 0, \tag{7.23}$$

where,

$$s_0 = \frac{\mu(\delta+\mu)(\theta+\mu)\left(a+(bI^*+1)^2(d+\mu+\varsigma)\right)}{(bI^*+1)^2},$$

$$\begin{aligned} s_1 = {} & \frac{1}{(bI^*+1)^2(\alpha I^*+1)^2}(aI^*(\alpha I^*+1)(\beta\mu(2\theta+\mu)+\gamma(\delta(2\theta+\mu)+\mu(\theta+\mu))) \\ & +(bI^*+1)^2(dI^*(\theta+\mu)(\alpha I^*+1)(\beta\mu+\gamma(\delta+\mu))+\beta\mu(-S^*)(\delta+\mu)(\theta+\mu)- \\ & \mu(\theta+\mu)(\gamma V^*(\delta+\mu)-I^*(\alpha I^*+1)(\mu(\beta+\gamma)+\gamma\delta))+I^*\varsigma(\alpha I^*+1)(\beta\mu(2\theta+\mu)+ \\ & \gamma(\mu(\delta+\theta)+2\delta\theta+\mu^2)))), \end{aligned}$$

$$\begin{aligned} s_2 = {} & \frac{1}{(bI^*+1)^2(\alpha I^*+1)^3}((\beta\gamma I^*(aI^*(2\theta+\mu)(\alpha I^*+1)+(bI^*+1)^2((\theta+\mu)(I^*(d+\mu)(\alpha I^*+1) \\ & +\mu(-S^*-V^*))+I^*\varsigma(2\theta+\mu)(\alpha I^*+1))))), \end{aligned}$$

$$\begin{aligned} r_1 = {} & \frac{1}{(bI^*+1)^2(\alpha I^*+1)^2}(aI^*(\alpha I^*+1)(2\beta(\theta+\mu)+\gamma(\delta+\theta+2\mu))+(bI^*+1)^2(dI^*(\alpha I^*+1)\times \\ & (\beta(\theta+2\mu)+\gamma(\delta+\theta+2\mu))-\beta S^*(2\mu(\delta+\theta)+\delta\theta+3\mu^2)-\gamma\delta\theta V^*-2\gamma\delta\mu V^*-2\gamma\theta\mu V^*- \\ & 3\gamma\mu^2V^*+2\alpha\beta\theta\mu I^{*2}+3\alpha\beta\mu^2I^{*2}+\alpha\gamma\delta\theta I^{*2}+2\alpha\gamma\delta\mu I^{*2}+2\alpha\gamma\theta\mu I^{*2}+3\alpha\gamma\mu^2I^{*2}+ \\ & I^*\varsigma(\alpha I^*+1)(2\beta(\theta+\mu)+\gamma(\delta+\theta+2\mu))+2\beta\theta\mu I^*+3\beta\mu^2I^*+\gamma\delta\theta I^*+2\gamma\delta\mu I^*+ \\ & 2\gamma\theta\mu I^*+3\gamma\mu^2I^*)), \end{aligned}$$

$$r_2 = \frac{\beta\gamma I^*\left(aI^*(\alpha Y+1)+(bI^*+1)^2(I^*(\alpha I^*+1)(d+\theta+2\mu+\varsigma)+(\theta+2\mu)(-S^*-V^*))\right)}{(bI^*+1)^2(\alpha I^*+1)^3},$$

$$r_0 = \frac{1}{(bI^*+1)^2}(a(2\mu(\delta+\theta)+\delta\theta+3\mu^2)+(bI^*+1)^2(d(2\mu(\delta+\theta)+\delta\theta+3\mu^2)+\varsigma(2\mu(\delta+\theta)$$
$$+\delta\theta+3\mu^2)+\mu(3\mu(\delta+\theta)+2\delta\theta+4\mu^2))),$$

$$q_0 = \frac{a(\delta+\theta+3\mu)+(bI^*+1)^2(d(\delta+\theta+3\mu)+\delta(\theta+3\mu+\varsigma)+\varsigma(\theta+3\mu)+3\mu(\theta+2\mu))}{(bI^*+1)^2},$$

$$q_1 = \frac{1}{(bI^*+1)^2(\alpha I^*+1)^2}(aI^*(\beta+\gamma)(\alpha I^*+1)+(bI^*+1)^2(dI^*(\beta+\gamma)(\alpha I^*+1)-\beta S^*(\delta+\theta+3\mu)$$
$$-\gamma\delta V^*-\gamma\theta V^*-3\gamma\mu V^*+\alpha\beta\theta I^{*2}+3\alpha\beta\mu I^{*2}+\alpha\gamma\delta I^{*2}+\alpha\gamma\theta I^{*2}+3\alpha\gamma\mu I^{*2}+$$
$$I^*\varsigma(\beta+\gamma)(\alpha I^*+1)+\beta\theta I^*+3\beta\mu I^*+\gamma\delta I^*+\gamma\theta I^*+3\gamma\mu I^*)),$$

$$q_2 = \frac{\beta\gamma I^*\left(-S^*-V^*+\alpha I^{*2}+I^*\right)}{(\alpha I^*+1)^3},$$

$$p_0 = \frac{a+(bI^*+1)^2(d+\delta+\theta+4\mu+\varsigma)}{(bI^*+1)^2},$$

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

Theorem 7.3.3. *At $\rho = 0$, the endemic equilibrium E_e is locally asymptotically stable if the real parts of all the roots of (7.23) are negative.*

Proof. Eq. (7.23) reveals that the characteristic equation at $\rho = 0$ near E_e is given by

$$\lambda^4+(p_0+p_1)\lambda^3+(q_0+q_1+q_2)\lambda^2+(r_0+r_1+r_2)\lambda+(s_0+s_1+s_2)=0. \tag{7.24}$$

The proof of this theorem is based on the conditions proposed by the Routh-Hurwitz criterion. Using this criterion, all roots of Eq. (7.24) have negative real parts if and only if

(H2): $p_0+p_1>0$, $r_0+r_1+r_2>0$, $s_0+s_1+s_2>0$ and,
$(p_0+p_1)(q_0+q_1+q_2)(r_0+r_1+r_2)>(p_0+p_1)^2(s_0+s_1+s_2)+(r_0+r_1+r_2)^2$. ■

Eq. (7.23) yields

$$p_1\lambda^3+q_1\lambda^2+r_1\lambda+s_1+(\lambda^4+p_0\lambda^3+q_0\lambda^2+r_0\lambda+s_0)e^{\lambda\rho}+(q_2\lambda^2+r_2\lambda+s_2)e^{-\lambda\rho}=0. \tag{7.25}$$

Let $\mathrm{i}\omega$ ($\omega > 0$) be a root of Eq. (7.25), then

$$-\mathrm{i}p_1\omega^3 - q_1\omega^2 + \mathrm{i}r_1\omega + s_1 + \left(\omega^4 - \mathrm{i}p_0\omega^3 - q_0\omega^2 + \mathrm{i}r_0\omega + s_0\right)\mathrm{e}^{\mathrm{i}\omega\rho} + \left(-q_0\omega^2 + \mathrm{i}r_2\omega + s_2\right)\mathrm{e}^{-\mathrm{i}\omega\rho} = 0. \tag{7.26}$$

Eq. (7.26) implies that

$$\begin{aligned}&\left(\left(p_0\omega^3 - r_0\omega + r_2\omega\right)\sin(\rho\omega) + \left(-q_0\omega^2 - q_2\omega^2 + s_0 + s_2 + \omega^4\right)\cos(\rho\omega) - q_1\omega^2 + s_1\right) + \\ &\quad \mathrm{i}\left(\left(-p_0\omega^3 + r_0\omega + r_2\omega\right)\cos(\rho\omega) - p_1\omega^3 + \left(-q_0\omega^2 + q_2\omega^2 + s_0 - s_2 + \omega^4\right)\sin(\rho\omega) + r_1\omega\right) = 0.\end{aligned} \tag{7.27}$$

Separation of real and imaginary parts gives

$$q_1\omega^2 - s_1 = \left(p_0\omega^3 - r_0\omega + r_2\omega\right)\sin(\rho\omega) + \left(-q_0\omega^2 - q_2\omega^2 + s_0 + s_2 + \omega^4\right)\cos(\rho\omega), \tag{7.28}$$

$$p_1\omega^3 - r_1\omega = \left(-p_0\omega^3 + r_0\omega + r_2\omega\right)\cos(\rho\omega) + \left(-q_0\omega^2 + q_2\omega^2 + s_0 - s_2 + \omega^4\right)\sin(\rho\omega). \tag{7.29}$$

That is,

$$h_1(\omega)\cos\omega\rho - h_2(\omega)\sin\omega\rho = h_3(\omega), \tag{7.30}$$

$$h_4(\omega)\sin\omega\rho + h_5(\omega)\cos\omega\rho = h_6(\omega), \tag{7.31}$$

where,

$$\begin{aligned}h_1(\omega) &= \omega^4 - (q_0 + q_2)\,\omega^2 + s_0 + s_2,\\ h_2(\omega) &= (r_0 - r_2)\,\omega - p_0\omega^3,\\ h_3(\omega) &= q_1\omega^2 - s_1,\\ h_4(\omega) &= \omega^4 - (q_0 - q_2)\,\omega^2 + s_0 - s_2,\\ h_5(\omega) &= (r_0 + r_2)\,\omega - p_0\omega^3,\\ h_6(\omega) &= p_1\omega^3 - r_1\omega.\end{aligned}$$

Thus,

$$\begin{aligned}\cos\omega\rho &= \frac{P_{01}(\omega)}{P_{00}(\omega)},\\ \sin\omega\rho &= \frac{P_{02}(\omega)}{P_{00}(\omega)}.\end{aligned} \tag{7.32}$$

with

$$
\begin{aligned}
P_{00}(\omega) &= \omega^4\left(-2p_0r_0+q_0^2-q_2^2+2s_0\right)+\omega^6\left(p_0^2-2q_0\right)+\omega^2\left(-2q_0s_0+2q_2s_2+r_0^2-r_2^2\right)\\
&\quad +s_0^2-s_2^2+\omega^8,\\
P_{01}(\omega) &= \omega^4\left(p_0r_1+p_1\left(r_0-r_2\right)+q_1\left(q_2-q_0\right)-s_1\right)+\omega^6\left(q_1-p_0p_1\right)\\
&\quad +\omega^2\left(\left(q_0-q_2\right)s_1+q_1\left(s_0-s_2\right)+r_1\left(r_2-r_0\right)\right)-s_0s_1+s_1s_2,\\
P_{02}(\omega) &= p_1\omega^7+\omega^5\left(p_0q_1-p_1\left(q_0+q_2\right)-r_1\right)+\omega^3\left(-p_0s_1+p_1\left(s_0+s_2\right)+\left(q_0+q_2\right)r_1-q_1\left(r_0+r_2\right)\right)\\
&\quad +\omega\left(\left(r_0+r_2\right)s_1-r_1\left(s_0+s_2\right)\right).
\end{aligned}
\tag{7.33}
$$

Eq. (7.32) gives

$$
P_{01}^2(\omega)+P_{02}^2(\omega)=P_{00}^2(\omega). \tag{7.34}
$$

Now, assume that, (H3): Eq. (7.34) has at least one positive root ω_0.
Then, Eq. (7.25) has a pair of purely imaginary roots $\pm i\omega_0$. For ω_0, the corresponding critical value of the time delay is obtained as

$$
\rho_0=\frac{1}{\omega_0}\arccos\frac{P_{01}(\omega_0)}{P_{00}(\omega_0)}+\frac{2\pi j}{\omega_0},\quad j=0,1,2,\ldots \tag{7.35}
$$

To establish the Hopf bifurcation at $\rho=\rho_0$, it must be shown that

$$
\Re\left(\frac{d\lambda}{d\rho}\right)\neq 0.
$$

Differentiating Eq. (7.23) with respect to ρ gives

$$
\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}=\frac{-\lambda\mathrm{e}^{\lambda\rho}\left(\lambda^4+P_0\lambda^3+q_0\lambda^2+r_0\lambda+s_0\right)+\lambda\left(q_2\lambda^2+r_2\lambda+s_2\right)\mathrm{e}^{-\lambda\rho}}{L}, \tag{7.36}
$$

where,

$$
\begin{aligned}
L &= 3P_1\lambda^2+2q_1\lambda+r_1+\left(4\lambda^3+3P_0\lambda^2+2q_0\lambda+r_0\right)\mathrm{e}^{\lambda\rho}+\rho\left(\lambda^4+P_0\lambda^3+q_0\lambda^2+r_0\lambda+s_0\right)\mathrm{e}^{\lambda\rho}\\
&\quad +\left(2q_2\lambda+r_2\right)\mathrm{e}^{-\lambda\rho}-\rho\mathrm{e}^{-\lambda\rho}\left(q_2\lambda^2+r_2\lambda+s_2\right).
\end{aligned}
$$

It follows that

$$
\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right)^{-1}=-\frac{Y_1(\lambda)}{Y_2(\lambda)}-\frac{\rho}{\lambda}, \tag{7.37}
$$

where,

$$\begin{aligned} Y_1(\lambda) &= \left(3P_1\lambda^2 + 2q_1\lambda + r_1\right) + \left(4\lambda^3 + 3P_0\lambda^2 + 2q_0\lambda + r_0\right)\mathrm{e}^{\lambda\rho} + \left(2q_2\lambda + r_2\right)\mathrm{e}^{-\lambda\rho}, \\ Y_2(\lambda) &= \lambda\mathrm{e}^{\lambda\rho}\left(\lambda^4 + P_0\lambda^3 + q_0\lambda^2 + r_0\lambda + s_0\right) - \left(q_2\lambda^2 + r_2\lambda + s_2\right)\mathrm{e}^{-\lambda\rho}. \end{aligned} \tag{7.38}$$

Application of $\lambda = \mathrm{i}w_0$ yields

$$\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right)^{-1}\Big|_{\lambda=\mathrm{i}\omega_0} = \frac{-3P_1\omega_0^2 + 2\mathrm{i}q_1\omega_0 + r_1 + \mathrm{e}^{\mathrm{i}\rho\omega}\left(-4\mathrm{i}\omega_0^3 - 3P_0\omega_0^2 + 2\mathrm{i}q_0\omega_0 + r_0\right) + \mathrm{e}^{-\mathrm{i}\rho\omega}\left(2\mathrm{i}q_2\omega_0 + r_2\right)}{\mathrm{i}\omega_0\ \mathrm{e}^{\mathrm{i}\rho\omega_0}\left(\omega_0^4 - \mathrm{i}P_0\omega_0^3 + \mathrm{i}r_0\omega_0 - q_0\omega_0^2 + s_0\right) - \mathrm{e}^{-\mathrm{i}\rho\omega_0}\left(\mathrm{i}r_2\omega_0 - q_2\omega_0^2 + s_2\right) + \mathrm{i}\dfrac{\rho}{\omega_0}}.$$

$$\Re\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right)^{-1}\Big|_{\lambda=\mathrm{i}\omega_0} = \frac{V_1V_2 + V_3V_4}{V_2^2 + V_4^2}.$$

where,

$$\begin{aligned} V_1 &= 4\omega_0^3\sin(\rho\omega_0) - 3P_0\omega_0^2\cos(\rho\omega_0) - 3P_1\omega_0^2 - 2q_0\omega_0\sin(\rho\omega_0) + 2q_2\omega_0\sin(\rho\omega_0) + \\ &\quad r_0\cos(\rho\omega_0) + r_2\cos(\rho\omega_0) + r_1, \\ V_2 &= P_0\omega_0^4\sin(\rho\omega_0) + q_0\omega_0^3\sin(\rho\omega_0) + q_2\omega_0^2\cos(\rho\omega_0) - r_0\omega_0^2\cos(\rho\omega_0) - r_2\omega_0\sin(\rho\omega_0) - \\ &\quad s_0\omega_0\sin(\rho\omega_0) - s_2\cos(\rho\omega_0) + \omega_0^5(-\sin(\rho\omega_0)), \\ V_3 &= 3P_0\omega_0^2\sin(\rho\omega_0) - 2q_0\omega_0\cos(\rho\omega_0) - 2q_2\omega_0\cos(\rho\omega_0) - 2q_1\omega_0 - r_0\sin(\rho\omega_0) + \\ &\quad r_2\sin(\rho\omega_0) + 4\omega_0^3\cos(\rho\omega_0), \\ V_4 &= -P_0\omega_0^4\cos(\rho\omega_0) - q_0\omega_0^3\cos(\rho\omega_0) - q_2\omega_0^2\sin(\rho\omega_0) - r_0\omega_0^2\sin(\rho\omega_0) - r_2\omega_0\cos(\rho\omega_0) + \\ &\quad s_2\sin(\rho\omega_0) + s_0\omega_0\cos(\rho\omega_0) + \omega_0^5\cos(\rho\omega_0). \end{aligned}$$

If (H4): $V_1V_2 + V_3V_4 \neq 0$ holds, then $\Re\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\rho}\right)^{-1}\Big|_{\lambda=\mathrm{i}\omega_0} \neq 0$.

Thus, the following theorem can be stated:

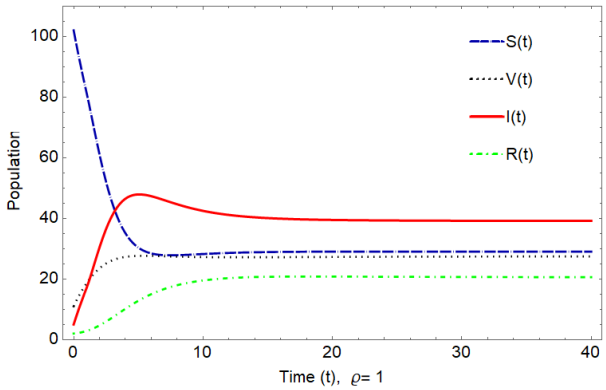
Theorem 7.3.4. *For the model (7.1), if conditions (H1-H4) hold, then the endemic equilibrium $E_e = (S^*, V^*, I^*, R^*)$ is locally asymptotically stable when $\rho \in [0, \rho_0)$; the model (7.1) undergoes a Hopf bifurcation at $E_e = (S^*, V^*, I^*, R^*)$ when $\rho = \rho_0$, and a family of periodic solutions bifurcate from $E_e = (S^*, V^*, I^*, R^*)$.*

7.4 Numerical simulation

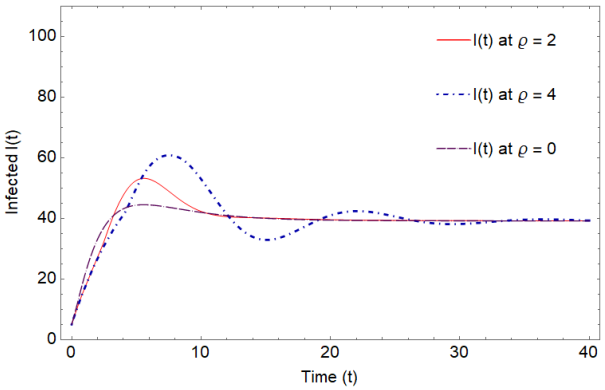
In this section, numerical simulations are carried out to illustrate the effectiveness of the obtained results.

The case of endemic equilibrium is illustrated for the following numerical data: $A = 12$, $\beta = 0.05$, $\alpha = 0.15$, $\mu = 0.1$, $d = 0.01$, $\theta = 0.1$, $\delta = 0.1$, $a = 2$, $b = 10$, $\varsigma = 0.1$, and $\gamma = 0.001$. It is estimated that, with these parameter values, the basic reproduction number of the model (7.1) is $R_0 = 1.38462$ and the endemic equilibrium is $E_e(S,V,I,R) = (28.9926, 27.4302, 39.1123, 20.5536)$.

Fig. 7.3.1 shows the behavior of the susceptible, vaccinated, infected, and recovered populations at $\rho = 1$. It can be seen that, as time increases, the susceptible population decreases, while the vaccinated, infected, and recovered population increase, and finally, all the subpopulations settle down to endemic equilibrium E_e. Fig. 7.3.2 shows the impact of time delay on the infected population. This figure clearly reveals that infection increases among society with an increase in the time delay ρ.



7.3.1: Suceptible-vaccinated-infected-recovered population for $\rho = 1$.



7.3.2: Infected population for various values of time delay ρ.

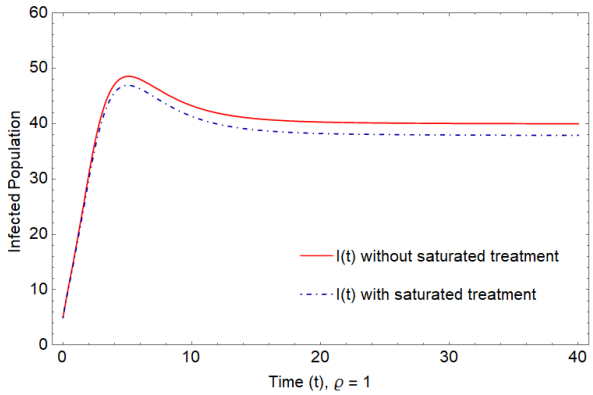
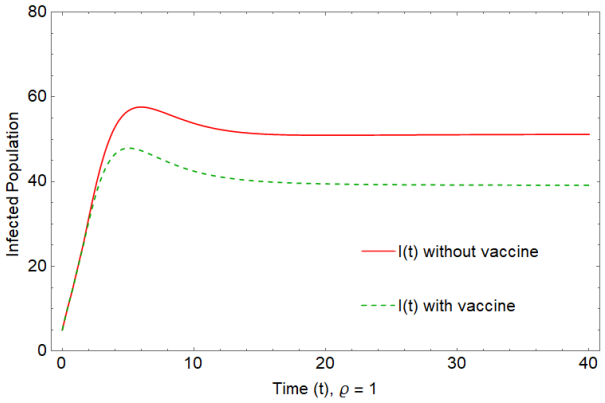
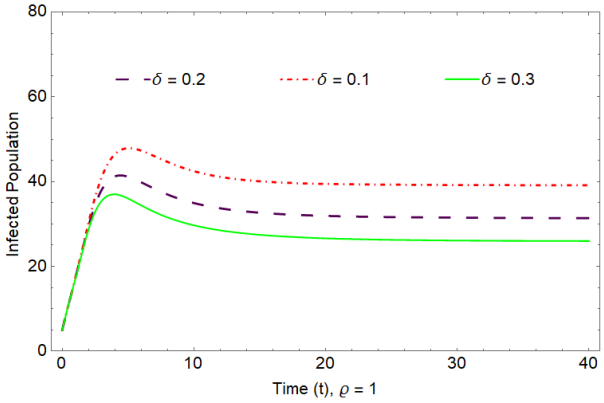


Figure 7.4: Infected population with and without saturated treatment rate for $\rho = 1$.

Fig. 7.4 reveals the impact of saturated treatment on the infected population for a time-delay $\rho = 1$. When treatment is given to infected individuals, the infection spreads at a lower level than the case without treatment, revealing that medical resources and their supply efficiency greatly influence the spread and control of an epidemic. Thus, it can be seen that the saturated treatment rate helps to lessen the transmission and to control the spread of the infection. Therefore, it is very important to make treatment facilities available quickly to infectives to diminish the infection among society.



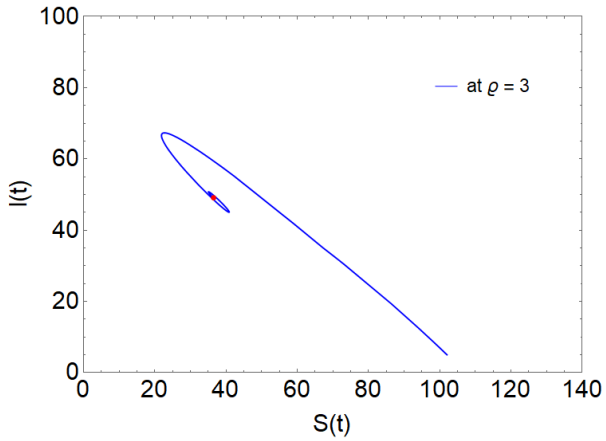
7.5.1: Infected population with and without vaccine.



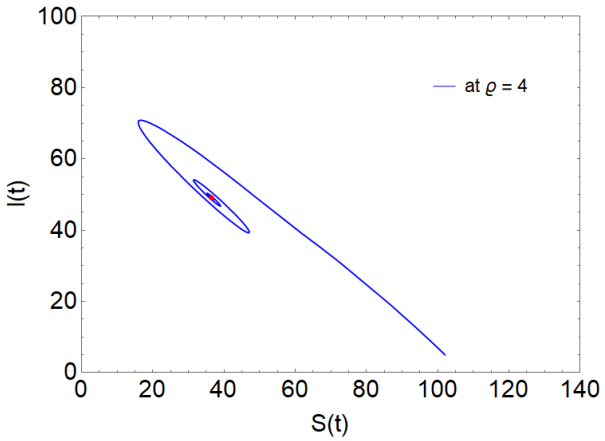
7.5.2: Infected Population for different values of vaccination rate.

Figure 7.5: Impact of vaccination on infected population.

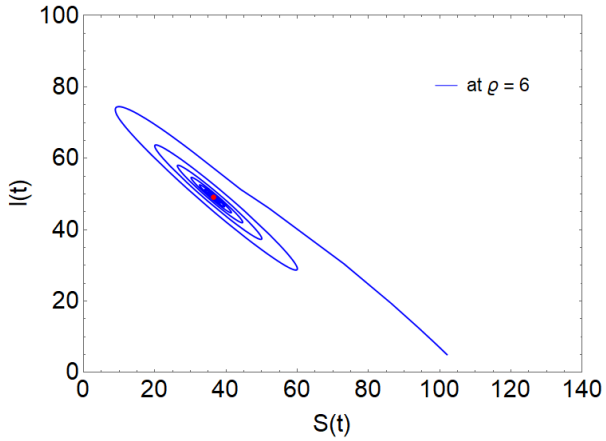
Figs. 7.5 shows the impact of vaccination on infected individuals. Fig. 7.5.1 shows the infected population with and without vaccination, revealing that, even though the vaccine is imperfect, it helps to reduce the spread of the infection. Meanwhile, Fig. 7.5.2 shows the variation in the infected population for different values of the vaccination rate, where the infected population is plotted for $\delta = 0.1,\ 0.2$, and 0.3 respectively. It can be seen that, when the vaccination rate is high, then the infected population diminishes at a higher rate. Thus, immunization by vaccination is a counteractive tool that can lessen the transmission of an infection and control its spread. Therefore, public health agencies need to ensure effective vaccination by increasing the time until loss of immunity and immunizing the maximum number of individuals.



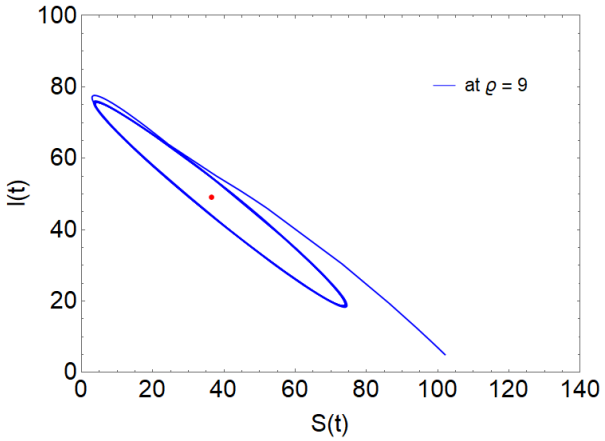
7.6.1: Susceptible versus infected population at $\rho = 3$.



7.6.2: Susceptible versus infected population at $\rho = 4$.



7.6.3: Susceptible versus infected population at $\rho = 6$.



7.6.4: Susceptible versus infected population at $\rho = 9$.

Figure 7.6: Hopf bifurcation for various values of time delay ρ.

Figs. 7.6 is plotted for the parameter values $A = 12$, $\beta = 0.05$, $\alpha = 0.15$, $\delta = 0.01$, $\mu = 0.1$, $\theta = 0.1$, $\gamma = 0.001$, $d = 0.01$, $\varsigma = 0.1$, $a = 5$, $b = 10$ and shows the relation between the susceptible and infected populations for different values of time delay, confirming the occurrence of Hopf bifurcation. The red dot on the curves indicates the endemic equilibrium E_e whose components are $(S,I) = (36.4105, 48.5345)$ and at this value the results give $(S,V,I,R) = (36.4105, 3.43944, 48.5345, 26.7621)$. These figures show how the fraction of infectives oscillates for higher values of the time delay, and finally, approaching the endemic equilibrium E_e. Figs. 7.6.3 and 7.6.4 indicate that increasing the time delay ρ results in a longer period of periodic oscillations around the endemic equilibrium E_e.

7.5 Discussion

An SVIRS epidemic model with a Holling type II functional incidence rate, time-delay, imperfect vaccine, and saturated treatment rate is studied herein. The analysis of the model shows that it exhibits two equilibria: disease-free equilibrium (DFE) and endemic equilibrium (EE). The basic reproduction number R_0 is obtained, and the dynamics of the model for disease transmission is characterized by R_0, both with time delay and without time delay. For $\rho > 0$, it is shown that the DFE is locally asymptotically stable when R_0 is less than unity, unstable when R_0 is greater than unity, and linearly neutrally stable when R_0 is equal to unity. However, the use of center manifold theory reveals that the model undergoes backward or forward bifurcation at $R_0 = 1$ when there is no time delay. This analysis is of interest in itself as it provides some information on the stability of the DFE and EE. The model exhibits forward or backward bifurcation under particular conditions obtained by inequalities 7.15, and 7.16. Schematic diagrams of forward and backward bifurcation are depicted in Fig. 7.2. Furthermore, stability analysis of the endemic equilibrium is performed, and the local stability of the endemic equilibrium is shown in Theorems 7.3.3 and 7.3.4. Regarding time delay as a bifurcation parameter and analyzing the corresponding characteristic equation, the occurrence of Hopf bifurcation near the endemic equilibrium is shown, illustrating the presence of oscillatory and periodic solutions. Numerical simulations are performed to demonstrate the effectiveness of the theoretical findings. The graphical representation elucidates the impact of time delay on infected individuals, revealing that when the time delay is high, then there is a large number of infected individuals. The effect of saturated treatment rate has been seen graphically, and it can be said that the considered treatment is imperative to control the spread of the infection as it lessens transmission and reduces the number of infectives. The occurrence of the oscillatory and periodic solution is also illustrated, confirming the existence of Hopf bifurcation. The present study demonstrates that an imperfect vaccine and saturated treatment rate may lead to backward bifurcation, but at the same time, it should be emphasized that these measures reduce the size of the infected population. High vaccine take-up levels result in radical decreases of infectious disease, as shown in Fig. 7.5. If a completely effective vaccine can be made, this plausibility does not emerge, while a program that reduces the contact rate can further control infection without inciting backward bifurcation.

Chapter 8

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.

8.1 Conclusion

There is no doubt that mathematical epidemic models help to understand the transmission and spread of infectious diseases, recognize the components administering the transmission procedure to create successful control techniques, and evaluate the effectiveness of surveillance strategies and intervention measures. This thesis studies the transmission and prevention mechanisms of epidemics through mathematical compartmental models with nonlinear incidences, latent period, and treatment rates using the system of delay differential equations (DDEs). New compartments have been introduced into the SIR model for various stages according to the dynamics of the disease. For example, compartments of partially aware, fully aware, and vaccinated individuals. Novel combinations of different nonlinear incidence and treatment rates are considered. The incidence rates of infection are considered nonlinear functional types that provide rich and more realistic transmission dynamics in a large population. It is shown that the proposed models are epidemiologically well-behaved. Equilibrium analysis of the models proves and emanates the existence and uniqueness of equilibria. The local and global stability behavior of the equilibria have been analyzed and further validated through numerical simulations. We found the threshold value, the basic reproduction number R_0 for each model to determine disease persistence in the endemic zone. The impact of the

latent period has been seen for all presented models. It is concluded that the latency phase can increase the ability of the disease to stay for an extended period and give rise to Hopf bifurcation. In summarizing, in Chapter two, we explore the time-delayed SIR model with Beddington-DeAngelis type incidence rate and a saturated treatment rate. The local stability has been investigated using R_0 for the equilibria: disease-free and endemic. The results suggest that as delay increases, the infected population increases at a higher rate, and the oscillatory behavior of the infected population may occur, which shows the presence of Hopf bifurcation. Moreover, the disease can eradicate from society if the treatment given to infectives is managed according to the saturated treatment rate. Chapter three is an extension of Chapter two, where we consider Beddington-DeAngelis type incidence rate with Holling type II treatment rate and study the local and global stability behaviors of the model's equilibria. We show that the model exhibits various bifurcations such as forward, backward and Hopf bifurcations. This model is interesting in itself because the forward and backward bifurcation scenarios occur and depend on the parameter values of Holling type II treatment rate. Chapter four studies a time-delayed SIR epidemic model with a logistic growth of susceptibles, Crowley-Martin type incidence, and Holling type III treatment rates. The analytical results and numerical simulation of this chapter are capable of demonstrating the significant role of the following nonlinearities: capturing lags between the exposure of disease, onset of its symptoms, and then providing treatment to infectives; susceptibles' and infectives' protection level against the infectious diseases; the limitation in the availability of the medical resources. The results further suggest control strategies to prevent the spread of diseases. Chapter five incorporates the compartment of aware individuals in the SIR epidemic model with Michaelis-Menten type incidence rates. The long-term qualitative behavior of the model is investigated. The relationship between human awareness and the spread of infection is seen by observing the difference between the number of infected individuals with and without aware individuals compartment, deliberating that unaware individuals are becoming infected faster than those familiar with the disease spread. Also, the results suggest that for eradicating disease, there is a need for awareness among people and sufficient treatment availability. Chapter six is an extension of Chapter five. The class of susceptible individuals is divided into three subclasses due to people's different social, educational, and economic backgrounds: unaware susceptibles, fully aware susceptibles, and partially aware susceptibles to the disease, respectively. The model is formulated by incorporating three explicit Holling type II incidences with latent period and Holling type II treatment rate. The local stability behavior of equilibria is investigated. The

results show the existence of transcritical bifurcation (such as forward and backward) and Hopf bifurcation near-endemic equilibrium. Moreover, the significant role of the latent period, the behavior of susceptibles through different subclasses, and limitation in available treatment facilities is observed. The results demonstrate the role of varying protection levels of susceptibles in the transmission pattern of infectious diseases. Chapter seven introduces the compartment of imperfect vaccinated individuals in the SIRS epidemic model and proposes a susceptible–vaccinated–infected–recovered–susceptible (SVIRS) epidemic compartmental model along with a Holling type II explicit incidences and a saturated treatment rate. It is concluded that an imperfect vaccine and saturated treatment rate may lead to backward bifurcation, but at the same time, it reduces the number of the infected population, and high vaccine take-up levels result in a significant decline of infectious cases.

8.2 Future scope

In this thesis, we proposed deterministic models for disease transmission. We have studied the stability analysis of these models and presented the numerical computations in graphs to support analytical findings. As further studies and future directions, we may explore the models for chaotic behavior and stochasticity. Also, fractional-order derivatives can be applied in the presented epidemic models for the memory effect.

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List of Publications

1. **K. Goel**, Nilam. A mathematical and numerical study of a SIR epidemic model with time delay, nonlinear incidence and treatment rates, *Theory in Biosciences*, vol. **138**, pp. 203–213 (2019) Springer.

2. **K. Goel**, Nilam. Stability behavior of a nonlinear mathematical epidemic transmission model with time delay, Nonlinear Dynamics, vol. **98**, pp. 1501–1518 (2019), Springer.

3. **K. Goel**, A. Kumar, Nilam. Stability analysis of a logistic growth epidemic model with two explicit timedelays, (communicated).

4. **K. Goel**, Nilam. A time-delayed nonlinear epidemic model with aware individuals class: modeling and analysis, (communicated).

5. **K. Goel**, A. Kumar, Nilam. Nonlinear dynamics of a time-delayed epidemic model with two explicit aware classes, saturated incidences, and treatment, Nonlinear Dynamics, vol. **101**, pp. 1693–1715 (2020), Springer.

6. **K. Goel**, A. Kumar, Nilam. A deterministic time-delayed SVIRS epidemic model with incidences and saturated treatment, vol. **121**, pp. 19–38 (2020), Springer.

List of Conferences

1. **K. Goel**, Nilam. A delayed SIR epidemic model along with Beddington- DeAngelis type incidence rate and Holling type II treatment rate, presented at 2nd International Conference on Modern Mathematical Methods and High Performance Computing in Science and Technology (M3HPCST-2018) organized by Inderprastha Engineering College, Ghaziabad, India, January 04-06, 2018.

2. **K. Goel**, A. Kumar, Nilam. A delayed SIR epidemic model along with Beddington-DeAngelis type incidence rate and Holling type III treatment rate, presented at 1st International Conference on New Frontiers in Engineering, Science, and Technology (NFEST-2018), held in New Delhi, India, January 08-12, 2018.

3. **K. Goel**, Nilam. A mathematical model to control the spread of Ebola in human population, presented at International Conference on Recent Advances in Pure and Applied Mathematics held at DTU Delhi, India, October 23-25, 2018.

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ORIGINAL ARTICLE

A mathematical and numerical study of a SIR epidemic model with time delay, nonlinear incidence and treatment rates

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Abstract
A novel nonlinear time-delayed susceptible–infected–recovered epidemic model with Beddington–DeAngelis-type incidence rate and saturated functional-type treatment rate is proposed and analyzed mathematically and numerically to control the spread of epidemic in the society. Analytical study of the model shows that it has two equilibrium points: disease-free equilibrium (DFE) and endemic equilibrium (EE). The stability of the model at DFE is discussed with the help of basic reproduction number, denoted by R_0, and it is shown that if the basic reproduction number R_0 is less than one, the DFE is locally asymptotically stable and unstable if R_0 is greater than one. The stability of the model at DFE for $R_0 = 1$ is analyzed using center manifold theory and Castillo-Chavez and Song theorem which reveals a forward bifurcation. We also derived the conditions for the stability and occurrence of Hopf bifurcation of the model at endemic equilibrium. Further, to illustrate the analytical results, the model is simulated numerically.

Keywords Epidemic model · Beddington–DeAngelis-type incidence rate · Saturated treatment rate · Stability · Bifurcation · Center manifold theory

Mathematics Subject Classification 34D20 · 92B05 · 37M05

Introduction

Mathematical analysis and modeling of infectious diseases play a crucial role in studying a wide range of infectious diseases to better understand the transmission dynamics and have the capacity to influence expectations and to decide and assess control strategies. Elementary descriptions of infectious diseases have been considered mainly by three epidemiological classes which measure the susceptible portion of population, the infected and the removed (recovered) ones. Authors around the world have proposed different kinds of epidemic models such as susceptible–infected (SI) (Mukherjee 1996), susceptible–infected–susceptible (SIS) (Hethcote and van den Driessche 1995), susceptible–infected–recovered (SIR) (Kumar and Nilam 2018a), susceptible–infected–recovered–susceptible (SIRS) (Mena-Lorca and Hethcote 1992), susceptible–exposure–infected–recovered (SEIR) (Dubey et al. 2013; Tipsri and Chinviriyasit 2014), susceptible–Vaccinated–exposure–infected–recovered (SVEIR) (Gumel et al. 2007) and many more, to understand the dynamics of disease transmission. In the mathematical epidemiological literature, several authors have studied the epidemiological models with latent or incubation period, because many diseases have a latent or incubation period, during which the susceptible individual becomes infected but is not yet infectious. Such latency in disease transmission can be modeled by a delay differential equation. Delay differential equations (DDE) have been a very successful tool to capture the effect of varying infectious period in a range of SIR, SIS, SIRS and other epidemic models. Hethcote and van den Driessche (1995) have studied an SIS epidemic model with constant time delay, which accounts for duration of infectiousness. Also, Song and Cheng (2005) have studied the impact of time delay on the stability of the positive equilibrium, as a resultant of which conditions have been stated for the asymptotical stability of the endemic equilibrium for all delays. Xu and Ma

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ORIGINAL PAPER

Stability behavior of a nonlinear mathematical epidemic transmission model with time delay

Kanica Goel · Nilam

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Abstract In this article, we study a time-delayed susceptible–infected–recovered mathematical model along with nonlinear incidence rate and Holling functional type II treatment rate for epidemic transmission. The mathematical study of the model demonstrates that the model exhibits two equilibria, to be specific, disease-free equilibrium (DFE) and endemic equilibrium (EE). We obtain the basic reproduction number R_0 and investigate that the model is locally asymptotically stable at DFE if $R_0 < 1$ and unstable if $R_0 > 1$ for the time lag $\nu > 0$. The stability of DFE at $R_0 = 1$ is also investigated for the time lag $\nu \geq 0$, and we show that for $\nu > 0$, the DFE is linearly neutrally stable, whereas for $\nu = 0$, the model exhibits backward bifurcation whereby the DFE will coexists with two endemic equilibria, when $R_0 < 1$. We also investigate the stability of the model at the EE and find that oscillatory solution may appear via Hopf bifurcation, taking the delay as a bifurcation parameter. Further, global stability of the model equilibria has also been analyzed. Finally, numerical simulations have been presented to illustrate the analytical studies.

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Keywords Time delay · Nonlinear incidence · Holling functional type II · Basic reproduction number (BRN) · Stability · Bifurcations

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1 Introduction

Mathematical modeling is progressively used in various fields such as biology, engineering, and economics [1–5]. In epidemiology, mathematical modeling is an effective method to study a diverse range of infectious diseases, for a better understanding of transmission dynamics. Many authors have proposed numerous epidemic models, such as susceptible–infected–recovered (SIR) [2,6–8], SEIR [9], SVEIR [10], etc., and studied the spread of infectious diseases mathematically and quantitatively. A major goal of the epidemiological study is to develop an understanding of the transmission of epidemics and interventions that can be taken to prevent and control the spread of infectious diseases. For the most part, an exemplary condition for the elimination of an infection is for the basic reproduction number (BRN) [11] to be less than one whereby the disease-free equilibrium is stable; the unique endemic equilibrium will be asymptotically stable as far as when the BRN is greater than one, implies that the disease-free equilibrium will be unstable. However, many authors have exhibited that it is feasible for the disease-free

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Nonlinear dynamics of a time-delayed epidemic model with two explicit aware classes, saturated incidences, and treatment

Kanica Goel · Abhishek Kumar · Nilam

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Abstract Whenever a disease emerges, awareness in susceptibles prompts them to take preventive measures, which influence individuals' behaviors. Therefore, we present and analyze a time-delayed epidemic model in which class of susceptible individuals is divided into three subclasses: unaware susceptibles, fully aware susceptibles, and partially aware susceptibles to the disease, respectively, which emphasizes to consider three explicit incidences. The saturated type of incidence rates and treatment rate of infectives are deliberated herein. The mathematical analysis shows that the model has two equilibria: disease-free and endemic. We derive the basic reproduction number R_0 of the model and study the stability behavior of the model at both disease-free and endemic equilibria. Through analysis, it is demonstrated that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, unstable when $R_0 > 1$, and linearly neutrally stable when $R_0 = 1$ for the time delay $\varrho > 0$. Further, an undelayed epidemic model is studied when $R_0 = 1$, which reveals that the model exhibits forward and backward bifurcations under specific conditions, which also has important implications in the study of disease transmission dynamics. Moreover, we investigate the stability behavior of the endemic equilibrium and show that Hopf bifurcation occurs near endemic equilibrium when we choose time delay as a bifurcation parameter. Lastly, numerical simulations are performed in support of our analytical results.

Keywords Full and partial awareness · Time delay · Nonlinear incidences and treatment rates · Bifurcations · Stability · Numerical simulations

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1 Introduction

The last two decades have seen several large-scale epidemics outbreaks such as Ebola, SARS, Zika virus, and swine flu, which leads to low socioeconomic status and inadequate access to health care. People get information about these outbreaks quite quickly due to significant advances in social media, which can have an insightful effect on the actual epidemic dynamics [17,31]. Therefore, at the beginning of an epidemic outbreak, the initial step is to make the individuals aware of the disease and its preventive methods. Awareness programs can alert the susceptible population toward the

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A deterministic time-delayed SVIRS epidemic model with incidences and saturated treatment

Kanica Goel · Abhishek Kumar · Nilam

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Abstract A novel nonlinear delayed susceptible–vaccinated–infected–recovered–susceptible (SVIRS) epidemic model with a Holling type II incidence rate for fully susceptible and vaccinated classes, a saturated treatment rate, and an imperfect vaccine given to susceptibles is proposed herein. Analysis of the model shows that it exhibits two equilibria, namely disease-free and endemic. The basic reproduction number R_0 is derived, and it is demonstrated that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ and linearly neutrally stable when $R_0 = 1$. Furthermore, bifurcation analysis is performed for the undelayed model, revealing that it exhibits backward and forward bifurcation when the basic reproduction number varies from unity. The stability behavior of the endemic equilibrium is also discussed, revealing that oscillatory and periodic solutions may appear via Hopf bifurcation when regarding delay as the bifurcation parameter. Moreover, numerical simulations are carried out to illustrate the theoretical findings.

Keywords Bifurcation · Holling type II functional response · Saturated treatment rate · SVIRS epidemic model · Time delay

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1 Introduction

Epidemiology is often called the core science of public health, considering the appropriation and determinants of infection risk in human populations. Mathematical epidemic models help to understand the transmission and spread of infectious diseases, recognize the characteristics controlling the transmission process to identify successful control techniques, and evaluate the effectiveness of surveillance strategies and intervention measures. Deterministic models for communicable diseases have been introduced systematically by Kermack and McKendrick [1,2]. In

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