MATHEMATICAL MODELLING OF INFECTIOUS DISEASES USING DELAY DIFFERENTIAL EQUATIONS

Thesis Submitted For the award of

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in

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DECLARATION

I hereby declare that the thesis entitled "**Mathematical Modelling of Infectious Diseases using Delay Differential Equations**" submitted for award of degree of **Doctor of Philosophy (Ph.D)** in Mathematics, Lovely Professional University, Phagwara, is my own work conducted under the supervision of Dr. Pankaj Kumar (Supervisor), Associate Professor in Department of Mathematics at School of Chemical Engineering and Physical Sciences, Lovely Professional University, Phagwara, Punjab. I further declare that to the best of my knowledge the thesis does not contain any part of any work which has been submitted for the award of any degree in this University or in any other University/Deemed University without proper citation.

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CERTIFICATE

This is to certify that the work entitled "***Mathematical Modelling of Infectious Diseases using Delay Differential Equations***" is a piece of research work done by Mr. Raminder Pal Singh under my guidance and supervision for the degree of Doctor of Philosophy of Mathematics, Lovely Professional University, Phagwara (Punjab) India. To the best of my knowledge and belief the thesis:

1. Embodies the work of the candidate himself.

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4. Is up to the standard both in respect of contents and language for being referred to the examiner.

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Dedicated

To My

Mentor, God,

Friends

and

My Family

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ABSTARCT

In the proposed work, an incubation time delay in the detection of the susceptible, infected and recovered population is explored and assessed using an SIR epidemic model. Individuals who are susceptible, infected, and recovered hosts are taken into account as state variables. It is assumed that the inner equilibrium is not zero. The local and global stability of the endemic equilibrium is investigated by using the time delay as a bifurcation parameter, and establish whether the condition is absolutely stable or conditionally stable. A Hopf bifurcation also happens under certain circumstances.

Mathematically, the positivity and boundedness of all analytical solutions is established using comparison theorem. Local stability analysis of interior equilibrium is established. Stability analysis of interior equilibrium considering delay parameter resulted into Hopf-bifurcation showing the complex dynamical behaviour. Nature of the roots has been studied in detail using Rouche's theorem. Sensitivity analysis of state variables with respect to model parameters is done for almost all the models using 'Direct Method'. Numerical simulation is done using MATLAB where all the model parameters have been assigned different numerical values. This helped to find the critical value of the delay parameter below which the system exhibited stability and above this critical value, the system lost stability and Hopf-bifurcation occurred.

In chaper-2, A traditional SIR model is analyzed by means of delay differential equations in this work. With the assumption that the susceptible population grows logistically and that the incidence rate follows the bilinear rule of mass action. The delay parameter represents the incubation time that must pass before infected susceptible become infectious. Using the transcendental characteristics equation, the non-zero epidemic equilibrium point is identified and also stability study is performed on that point. When the delay parameter value is less than the critical value, the system is destabilized and shows asymptotic stability. Hopf bifurcation occurs as the delay reaches the critical

point. Directional analysis depicting the direction and periodicity of bifurcating solutions is performed using the center manifold theory. The sensitivity analysis is conducted on the parameters of the model and the basic reproductive number R_0 which indicate the relative significance of these for fluctuations in the different values of state variables. MATLAB is used to validate the numerical results.

In chapter-3, An SIR epidemic model is investigated and evaluated by including a lag in the incubation time into the method of determining who is infected with the disease. The state variables that are taken into account are the persons who are susceptible to infection, who have contracted infection, and who have recovered from infection. The computation of the inner equilibrium that does not equal zero is carried out. By using the time delay as a bifurcation parameter, the local stability of the endemic equilibrium is examined, and a condition that is either absolutely stable or conditionally stable is identified. In addition, there are some conditions that must be met before a Hopf bifurcation may take place. Using MATLAB, numerical simulations have been run in order to better show the primary findings that have been drawn.

In chapter-4, Mathematical examination of the SIR model with mutation, including the inclusion of an incubation time lag as well as a general non-linear incidence rate based on the law of mass action. When the virus undergoes a mutation, the restored population loses its immunity and becomes susceptible once again at a pace specified by the letter 'c', which stands for the mutation rate. Before taking effect, this mutation waits for the period of grace time specified by τ. The three-state variables susceptible population, infected population, and recovered population are each denoted by the symbols S(t), I(t), and R(t), respectively. The basic reproduction number, denoted by the notation 'R_0', is determined, and a graphic representation of its significance to infected and recovered populations is provided. For the purposes of studying stability and bifurcation, the non-zero equilibrium is taken into consideration. When the delay parameter reaches its critical point value, the

Hopf-bifurcation condition will take place. Both sensitivity analysis and directional analysis are done out using the direct method. In order to corroborate analytical results with numerical simulations, MATLAB is utilized to conduct the simulations.

In chapter-5, A discrete delayed SIR epidemic model with logistic growth is analysed. A threshold value is obtained which depict a dynamics and outcomes of the disease. A basic reproductive number $\Re_0$is defined, with this a disease free and non-zero endemic equilibrium is discussed. A disease-free equilibrium is shown to be asymptotically stable for a given value of the delay parameter τ. The disease disappears when the basic fundamental reproductive number $\Re_0 < 1$. For a certain value of the delay parameter τ, it is observed that the equilibrium E^* is asymptotically stable and devoid of diseases. When $\Re_0 > 1$, disease persist. Stability switches are seen for $\tau \neq 0$ and Hopf-Bifurcation occurred when τ going beyond some critical point value. The delayed system for DFE (disease-free equilibrium) at $\Re_0 = 1$, has also been shown to be linearly neutrally stable. We also analyze the sensitivity of the state variable with respect to the fundamental reproductive number $\Re_0$. We conclude with some MATLAB-based numerical simulations to demonstrate the analytical findings.

LIST OF FIGURES

LIST OF TABLES

Contents

Chapter 1

1.1 General Introduction

Diseases that are caused by organisms, such as bacteria, viruses, fungus, or parasites, are referred to as infectious diseases. Our bodies are home to a wide variety of species, both inside and out. They seldom cause damage and can even help the organism. However, if the right circumstances are present, some microbes may cause sickness. It is possible for some infectious diseases to be spread from one person to another. There are other diseases that may be passed on by insects and other animals. In addition to that, you might get further diseases if you consume tainted food or water or if you come into contact with organisms that are found in the natural world. Fever and exhaustion are common indications of a disease, but the signs and symptoms might vary based on the organism that is causing it. Infections that are not life-threatening may respond to rest and home treatments, whereas infections that are life-threatening may need hospitalization. Vaccines are an effective means of protecting against a wide variety of contagious diseases, including measles and chickenpox. Washing your hands often and thoroughly may help prevent you from the vast majority of infectious infections. The following are examples of different phases of infectious diseases:

- Endemic Phase: The prefix "en" means "in" and the suffix noun "demos" means "people," therefore the term "endemic" literally translates to "in-people". It is a term that is used to describe a disease that is prevalent at a level that is generally consistent across a whole community or nation. It is possible for any nation to be home to a disease that is distinct from all others. For instance, malaria is an infectious disease that is endemic to Africa.
- Epidemic Phase: An epidemic is a scenario in which a disease spreads quickly to a large number of individuals in a specific community within a short period of time. The term "epidemic" originates the Greek prefix "epi," means upon or above, suffix "demos," is people.

The word "epidemic" is used to more than merely outbreaks of infectious diseases. It is also applied with any circumstance that leads to a damaging growth of health hazards inside a society, for instance the rise in obesity internationally which is commonly called as "obesity epidemic".

- Pandemic Phase: The Greek prefix “pan”, meaning "all," and the noun demos, meaning "people," are the origins of the English term pandemic. It is a phrase that is used to describe the fast spread of an infectious or communicable disease spanning numerous continents or globally. The term "pandemic" is given to an epidemic when it has reached a worldwide scale and is affecting a significant proportion of the world's population, like in the case of the covid-19 virus.
- Syndemic Phase: A syndemic is the aggregation of two or more contemporaneous or sequential epidemics or disease clusters in a community with biological interactions that make the prognosis and burden of disease worse. A hookworm, malaria, and HIV/AIDS pandemic is an example of a syndemic, also known as a synergistic epidemic.

The following infectious disease affected the history of mankind:

- 430 BC - Around 100,000 people perished as a result of the Plague of Athens, which is considered to be the world's first pandemic to spread across many regions. Nobody knows for sure what sickness was responsible for this epidemic.
- 541 BC - The pathogen Yersinia pestis was to blame for the Justinian Plague, which was responsible for the deaths of 30 million to 50 million people.
- In the 1340s, the bubonic plague produced by Yersinia pestis, sometimes known as the "Black Death," claimed the lives of around 50 million people.
- In the year 1494, an epidemic of syphilis, which originated in the Americas and was transported to Europe by its carrier, the bacterium

Treponema pallidum, claimed the lives of more than 50,000 individuals in Europe.

- Tuberculosis is an old disease that first became widespread during the Middle Ages and was responsible for the deaths of millions of people.
- In the year 1520, over 3.5 million people perished as a result of smallpox, which was caused by the bacterium Variola major.
- Between the years 1793 and 1798, yellow fever, sometimes known as "The American Plague," claimed the lives of around 25,000 persons in colonial America.
- The cholera epidemic of 1832 claimed the lives of around 18,402 individuals and spread over Asia, Europe, and the America.
- In 1918, the Spanish flu caused the deaths of over 50 million people and was responsible for subsequent pandemics in 1957, 1968, and 2009.
- Since its discovery in 1976, the Ebola virus has been responsible for 29 outbreaks and the deaths of 15,258 persons as of the year 2020.
- Acute haemorrhagic conjunctivitis, a kind of pink eye that was originally identified in 1969 and went on to trigger a pandemic in 1981, is a derivation of pink eye.
- Since it was discovered in 1981, HIV and AIDS have been responsible for the deaths of around 37 million people. It is now being referred to as a pandemic.
- 2002 was the year that saw the most fatalities from SARS, with 813 individuals losing their lives to the "near-pandemic" that year.
- In 2009, the H1N1 "swine flu" pandemic claimed the lives of 284,000 individuals, making it the sixth flu pandemic of this century.
- Chikungunya, a virus that is spread by mosquitoes and was discovered for the first time in 1952, did not become a pandemic until 2014. Rarely do people pass away as a result of this infection.
- 2015: The Zika virus belongs to the family of flaviviruses and has been around for many years. The mosquito-borne virus did not begin to

spread at an alarming rate until 2015, most likely as a result of a mutation in the virus.

- In 2019 the infectious disease known as COVID-19 is caused by a newly discovered strain of the coronavirus. The letters 'CO' and 'VI' represent for corona, while the letter 'D' stands for disease.

As a result of the fact that humans originated on a planet that was mostly inhabited by microorganisms, which are astounding in both their quantity and their variety, humans have been closely intertwined with them from the very beginning. The term "commensals," which literally means "those that eat at the same table," is given to microorganisms that live in close relationship with their hosts because these bacteria often benefit or gain advantages from this interaction. The bacteria are referred to be "mutualists" when they are both beneficial to others and to themselves. In a practical sense, it is difficult to determine if a particular microbe is a commensal, a mutualist, or none of these things. This is because a microbe's participation in the ecosystem may be indirect and its influence may be subtle due to the interactions it has with other members of the community. Microorganisms almost always reside in the environment in the form of intricate communities. The number of microbial cells that are linked with the human body is comparable to the total number of human cells, and the number of unique genes and gene functions these are connected with the human microbiome surpasses the number of unique human genes by a factor that is at least one hundred times greater.

The scope of the scientific discipline of infectious diseases has broadened to include not just the study of injuries and violence, but well as the exploration of any exposure that impacts health outcomes, including molecular and genetic variables. Historically, infectious diseases focused on the learning of outbreaks of infectious illness. The field of infectious diseases has a wide range of applications, including the assessment of a population's overall health, the facilitation of individual decision-making, and the identification of the factors that contribute to an event's cause. Infectious diseases are able to provide answers to difficult issues by using rigorous statistical approaches.

The data related to infectious diseases may also be used to advise policies or recommendations that impact groups or people, such as selecting which populations to vaccinate, establishing speed limits on a highway, or determining how many times per week a person should exercise.

It is not always necessary for a microorganism to cause disease in order for it to be considered a pathogen. Many infectious diseases that are common and serious in immunocompetent hosts are caused by organisms that are typically found within the human microbiota. These organisms compete with other indigenous microbes and, for the most part, adopt a commensal lifestyle. Illness, however, that is produced by these so-called commensal pathogens is probably definitely an accident due to the fact that illness is not necessary for the evolutionary survival of these pathogens.

The term "mathematical model" refers to a description of a system that is expressed in mathematical language and makes use of mathematical concepts. The act of creating a mathematical model is referred to as "mathematical modelling," and the phrase describes the process itself. Not only are models based on mathematics utilised in the chemical sciences, physical science, biological sciences, earth science, and computer engineering stream and other engineering, but they are also utilised in non-physical sciences as economics, psychology, sociology, and political science. The explanation of a system, the investigation of the impacts of various components, and the formulation of hypotheses on behaviour may all be aided by the use of models. Mathematical modelling is the skill of transforming issues from an application field into workable mathematical formulations whose theoretical and numerical analysis gives insight, solutions, and direction that might be valuable for the application from where the problems originated.

Mathematical models might be used in order to establish a connection between biological mechanism of communication and the outgoing dynamics of infection in populations. This would be done in order to better understand how infectious diseases spread across populations. At its most fundamental level, an epidemic may be explained by listing who infected whom and when

this took place. Mathematical models may be used to estimate how infectious diseases evolve, which can reveal the expected result of an epidemic and assist influence activities that are taken to improve public health. Models discover parameters for various infectious diseases by using fundamental assumptions or obtained data in conjunction with mathematics. Models then utilise these parameters to quantify the impacts of various treatments, such as mass vaccination programmes. The models may assist in determining which intervention should be avoided and which should be tested, as well as anticipate future development patterns, among other applications.

Since the beginning of infectious disease epidemiology as a field of study more than a century ago, representation the disease system mathematically and also a complete analysis like sensitivity, other behaviour of infectious diseases has been two of the most important aspects of the field. The recent advancements in technology of data management, the capacity to exchange and deposit data over the internet, as well as rapid diagnostic tests and genetic sequence analysis, the comprehensive electronic monitoring of infectious disease has become increasingly commonplace in present years. This is due to the fact that there have been numerous recent improvements in all of these areas. This is due to the fact that these developments have made it possible to monitor infectious diseases in greater detail than ever before. Because of these continuous breakthroughs, the use of mathematical models to generate and evaluate fundamental scientific theories as well as to build actionable methods for disease prevention has expanded significantly. Mathematical analysis and models have successfully shed light on previously baffling facts and have been of critical importance in the creation of public health programmes in a significant number of countries throughout the world.

In the discipline of mathematics, delay differential equations, which are sometimes referred to as DDEs for short, are a specialized form of differential equation in which the derivative of an unidentified function at a specific time is provided in terms of the values of the function at earlier periods. This type of differential equation is commonly referred to as a DDE. Delay Differential-

difference equations (DDEs) are sometimes referred to as time-delay systems, hereditary systems, systems with after effect or dead-time, differential-difference equations, and equations with diverging argument.

In infectious diseases, the effect is not felt immediately, but takes some time to mature. This delay may be due to gestation period, incubation, mating or identification of infected ones. This gives a good scope for incorporation and application of delay differential equations in the modelling of infectious disease. The proposed research work will be oriented and bounded towards modelling of infectious diseases, performing the stability analysis and observing bifurcations due to critical delay parameters.

1.2 Literature Review

Infectious diseases are a leading cause of mortality across the globe. In the past, infectious diseases have been responsible for the deaths of many more people than any and all conflicts combined (for example, the Spanish flu). Bernoulli Daniel is credited as being the first person to use mathematics to the modelling of infectious diseases (Bernoulli Daniel, 1760). Kermack and McKendrick's research, which was first made public in 1927, was a significant factor in shaping the modelling framework. Their SIR model continues to be used in the modelling of epidemics of infectious illnesses. This fundamental model, in addition to a few of its adaptations (Bacaër, 2011; Bacaër & Bacaër, 2011a, 2011b) will be the focus of our attention.

A deterministic SIR model is presented, in which the daily contact rate is assumed to remain unchanged, the rate in which the compartment of infected person recovers from the illness is assumed to rely solely on the amount of time that it has been infected, and the recovered people are assumed to be permanently immune from further assault (Wang, 1979). A comprehensive examination of a broad category of SIR epidemic models is presented, after which the adequate criteria that ensure the long-term stability of the endemic equilibrium solution are obtained (Stech & Williams, 1981) . It has been shown that a variety of epidemiological processes are responsible for periodic resolutions. The technique used is called extrinsic forcing, and it uses a

parameter like the contact rate as the driving force that results in periodicity the most directly, nevertheless, periodicity may also emerge of its own accord. This is a less common occurrence. According to (Hethcote & Levin, 1989) , cyclic models of the SIR or SEIR type are capable of having periodic solutions if there is a significant amount of temporal delay in the eliminated class. (Mena-Lorcat & Hethcote, 1992) analyses five SIRS epidemic models for populations of varied sizes in which the rate of infection is supplied by law of mass action. These terms pressing the count of infectives person and the number of susceptible or the proportion of the population that are susceptible. These incidences are given for populations in which the size of the susceptible population ranges from zero to one hundred percent.

To investigate the variation behaviour of a SIR epidemic model with a incubation period in the removed compartment and using a nonlinear incidence rate, a mathematical model is presented (Lin, 1992) . This model will be used to analyse the asymptotic behaviour. A disease transmission model of the SEIR type with an exponential demographic structure is developed in this article. According to Cooke and Van Den Driessche's (Cooke & Van Den Driessche, 1996) research, it is believed that all babies are susceptible to the disease, there is a constant for the normal mortality rate, and there is a constant for the extra death rate for infective people. In many infectious diseases, one of the most often seen phenomena is the process by which susceptible individuals might eventually build up immunity to the disease by exposure to a persistently low level of infection over time. This significant aspect has been included in a SIRS epidemiology model together with the rates of incidence and the rise in immunity (Ghosh et al., 1996). The equations for epidemics such as malaria that are transmitted by vectors such as mosquitoes and need an incubation period before becoming contagious are provided. A set of distributed delay differential equations will be generated as a result of the model's application. A model was presented for the propagation of an infectious disease that was transmitted by a vector after an incubation period had elapsed. (Takeuchi et al., 2000) model may still be seen as an

extension of the same thing, and the current model can be seen as an extension of that. An SIRS epidemic model with immunity provided by subclinical infection on a population has been taken into consideration. This has allowed for the examination of the geographical spread of a disease. Both the rate of infection and the rate of immunisation may be described as being of a nonlinear kind. (Ghosh et al., 2000) conducted research to examine the dynamics of the infectious disease as well as its endemicity on both a regional and international scale. According to (Evans et al., 2002), a generic SIR epidemic model is created with the force of infection susceptible to seasonal change, and a percentage of the number of infectives measured is unidentifiable. In this model, the fraction of unidentified infectives is not specified.

An epidemic of SARS, which was acquired by a new coronavirus, has spread from China to the rest of the world in a way that is confounding in terms of its behaviour as a contagious agent. The outbreak began in the province of Guangdong and has since spread to the rest of China and the rest of the world, (Ng et al., 2003) states that determining the factors that led to this behaviour is necessary for accurately anticipating what the future holds for the current epidemic as well as for developing and executing effective preventative measures. In the suggested stochastic model for the development of a susceptible-infective-removed (SIR) pandemic within a closed and limited population, there are two levels of severity of infectious individuals: mild and severe. (Ball & Britton, 2005) within the context of the SIRS model, the propagation of two distinct diseases in a small world network is examined, with the constraint that each individual may only be afflicted with one disease at any one moment. According to Kosinski and Grabowski's (Kosiński & Grabowski, 2006), there is a correlation between the existence of a second disease and a considerable reduction in the number of persons who were discovered to have the first disease. This correlation was established within a certain control parameter range. An investigation is conducted into the global dynamics of a SIRS model with a nonlinear incidence rate. (Jin et al., 2007)

determine a cutoff point for a illness to be considered extinct, investigate the presence and asymptotic behaviour and also the stability of equilibrium point, and prove the existence of two types of stable states, which may either be a disease-free stable equilibrium state, a stable endemic state, or a stable limit cycle state. (Yoshida & Hara, 2007) a SIR epidemic model that takes into account density-dependent birth and death rates has been developed. It is assumed in this model that the total number of people in the population is controlled by an equation called the logistic equation. According to Yoshida and Hara's research, it is presumed that the disease is spread in the usual manner. (Z. Song et al., 2009) , a model that investigates the presence of disease-free equilibrium as well as endemic equilibria which are stable for the SIRS epidemic model with the saturation incidence rate has been developed. This model takes into consideration the factors of population dynamics related to disease like mortality, natural mortality, and constant birth of population. The results of this study show that disease-free equilibrium and endemic equilibria do exist and are stable. (Dybiec, 2009), An expanded and modified SIR model of epidemic transmission is given, according to which susceptible agents are exposed to the illness and, as a result, may become infectious. This model is based on the idea that susceptible agents interact with infected neighbours.

(Lu, 2009) an epidemiological model that is a SIRS model with or without distributed time delay that is impacted by random perturbations is presented as a proposition in this article. In this paper, the disease-free equilibria of the related stochastic SIRS model is analysed, and the stability criteria of this equilibrium are presented. (R. Xu et al., 2010) An investigation is being conducted using a delayed SIRS epidemic model with saturation incidence and transitory immunity. According to research, the immunity that is developed by having a disease is only transitory, and sick persons who get infected will revert back to the vulnerable class after a certain amount of time. (Jiang et al., 2011) investigate a stochastic SIR model and demonstrate that this model can only generate one positive solution at the global level. In

addition to that, the asymptotic behaviour of this solution has also been shown. In this study, (Abta et al., 2012) a time lag delayed 'SIR' model and its matching SEIR model are examined side-by-side and compared with regard to global stability. According to this model, when a saturated incidence rate is taken into consideration, It is possible to determine, with the help of Lyapunov functionals, the conditions under which the endemic equilibrium and the disease-free equilibrium are both globally asymptotically stable. An SIR model that takes into consideration the influence of available resources in the public health system, particularly the number of hospital beds, has been developed by (Shan & Zhu, 2014). (Harko et al., 2014) proposed a model, that includes a standard incidence rate as well as a nonlinear recovery rate. There is the possibility of arriving at a parametric variation of the exact analytical solution to an SIR epidemic model. In order to investigate certain explicit models that match to predetermined values of the parameters, precise solutions are used, and the findings indicate that the numerical solution reproduces the analytical solution in an exact manner. (L. Liu, 2015) proposed an SIR epidemic model, examined and analysed based on the incorporation of an incubation time delay as well as a general nonlinear incidence rate, in which the increase of susceptible people is guided by the logistic equation. It is common practise to employ discrete epidemic models for the purpose of diagnosing the disease's aetiology, tracking its progression, and preventing its transmission. According to research carried out by (Hu et al., 2016), the three-dimensional discrete SIRS epidemic models are superior than their two-dimensional discrete counterparts when it comes to accurately describing the characteristics of the illnesses' transmission.

An SIR model of the transmission of the Zika virus (ZIKV) is being developed, and it will incorporate Zika virus infections in newborns. The model has one disease-free equilibrium point in addition to one endemic equilibrium point. According to (Kibona & Yang, 2017), the free one is stable under certain situations but unstable under other ones. Dengue fever and its potentially fatal consequences, such as dengue hemorrhagic fever, have

emerged as one of the most common disease that are spread by mosquitoes in recent decades. An SIR epidemiological model that describes the phases of the disease's transmission from vectors to hosts and from hosts to vectors has been presented by (Páez Chávez et al., 2017). An age-structured susceptible-infective-recovered epidemic model is examined in order to determine the asymptotic stability of the nontrivial endemic equilibrium of the model. The model is reduced into a system of four ordinary differential equations (ODEs) and the process is carried out (Kuniya, 2018). This reduction is conducted for a particular version of the disease transmission function.

An investigation into the usefulness of modelling techniques is carried out about the pandemic that was brought about by the spread of the novel COVID-19 illness, and a SIR model is developed. This model offers a theoretical framework to study the spread of the illness within a population (Cooper et al., 2020). There is now being carried out some mathematical and numerical analysis for COVID-19. A time-dependent SIR model is presented, which records the transmission and recovery rate at time 't' (Chen et al., 2020). This model is proposed in order to anticipate the trend of COVID-19. The COVID-19 pandemic serves as an example of how important it is for people to make treatment-related decisions. An example is given in which neither the rate of disease spread nor the effectiveness of therapies can be determined with absolute confidence (Gatto & Schellhorn, 2021). Since the first case of the COVID-19 coronavirus disease was discovered in China, there have been a number of efforts made to forecast the spread of the pandemic around the globe. These attempts have various degrees of accuracy and dependability. A short-term prediction of new cases is performed in order to anticipate the large number of cases in India and selected high-incidence states, and assess the efficacy of a three-week lock down period using a range of different models. This is done in order to prevent the spread of the disease across the country. In addition, a short-term forecast of new cases is carried out in order to determine the maximum number of active cases for certain states with a high frequency of the disease. This is done in order to lower the

risk of missing the greatest number of active cases that might possibly be present in India and in a few other states with a higher incidence rate. In order to examine the impact of lockdown as well as the impacts of other interventions, the Time Interrupted Regression model is used (Malavika et al., 2021), In order to make short-term predictions, the logistic growth curve model is used. When attempting to predict the peak period and maximum number of active cases, SIR models are used. The Time Interrupted Regression model is used in order to determine the effect that lockdown and various other interventions have had.

1.3 Proposed objectives of the study

On the basis of literature review and research gaps, the following objectives have been proposed in this present study:

1. To Analyze the classic SIR Model for infectious diseases using the delay differential equations.
2. To perform the stability analysis and Hopf-bifurcation for the virus mutation in SIR Model for infectious diseases using delay differential equations.
3. To perform the sensitivity analysis and directional analysis of the proposed model.

1.4 Basic Concepts of General SIR Model Used in the thesis

1.4.1 Basic Reproduction Number

The term "basic reproduction number" is used in the scientific discipline of epidemiology to refer to a method of determining how infectious a disease is and how easily it may be passed on to others. This is the number that is represented by the acronym R_0, which may be pronounced "R naught." It is an accurate representation of the normal number of new infections that can be traced back to a single infected individual in a population that is completely susceptible to the disease.

To be more explicit, the basic reproduction number is an estimate of the number of secondary infections that would be generated by an infected person in a community where everyone is susceptible to the disease, supposing that there are neither preventative measures in place nor any immunity to the disease. It takes into consideration things like how contagious the disease is, how long it remains contagious, and how often people interact with one another. If the basic reproduction number is less than one ($R_0 < 1$), it indicates that each infected individual will, on average, infect fewer than one other person. Under these circumstances, there is a good chance that the disease will start to recede and then disappear altogether from the population. On the other hand, if the basic reproduction number is more than one ($R_0 > 1$), this suggests that one infected individual will, on average, spread the infection to more than one other person. This provides evidence that the disease may have the capability of spreading across the population.

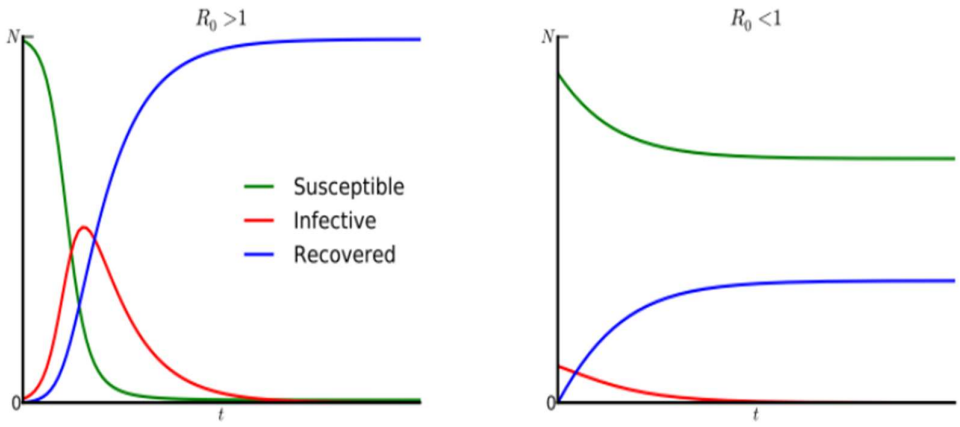

Figure 1.1 : The progression of time for all compartments displaying epidemic curves in the SIR model.

1.4.2 Law of Mass Action

The SIR model operates on the presumption that the people who make up the population behave in accordance with the law of mass action, which states that they interact with one another in a haphazard manner. The constant parameter "c" is used to quantify the possibility that an infected person may come into

touch with any other members of the population. The value of the parameter "a" gives an indication of how likely it is that the disease will in fact be passed on. These two constants, 'a' and 'c', may be collapsed into a single constant disease transmission rate, denoted by the letter 'r', if they are multiplied together. However, when it comes to disease propagation in a more general sense, it is essential to take into account the various aspects of 'r' separately. The assumption behind the rule of mass action is that the rate of disease transmission is proportional to the size of the population. While this is fairly accurate for many diseases like the flu.

1.4.3 The Basic SIR model without demography

The Susceptible, Infectious, and Removed/Recovered compartments bring the number of compartments in this basic compartmental model. Individuals who are not already infected but have the potential to become so are included in the susceptible compartment (S). This indicates that those who are in the susceptible compartment have the potential to get infected and migrate into the infectious compartment, or they may maintain their immunity and stay in the susceptible compartment without being infected. The infectious compartment (I) is made up of diseased people who are capable of passing the disease on to the susceptible people in the other compartment (S). This indicates that people who are contained inside the infected compartment either continue to be infectious or are transferred to the removed compartment if they recover or pass away. The persons that make up the removed compartment (R) are those who have either made a full recovery and are now immune or have passed away. All of the above indicates that the people are able to travel between the compartments in the following order $S \rightarrow I \rightarrow R$ and the rate of change are given by the following system of equations.

$$\frac{dS}{dt} = -\frac{\beta SI}{N}$$
$$\frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

The ODE that was used in the SIR model may be seen in the equations that were just presented. This population is closed, which means there are no new births and no deaths. *N* is the total number of people present throughout all of the different compartments, and this value will stay the same from one time step to the next. The infection rate, denoted by the Greek letter beta β, is calculated by multiplying the likelihood of being infected after coming into touch with an infected person by the typical number of people with whom one comes into daily contact. The recovered/removal rate, also known as gamma γ, is the rate at which a person is transported from the infected compartment to the recovered compartment. This determines how quickly an individual is removed from the infectious compartment. β and γ have the values that are predetermined from the start, whereas *S, I,* and *R* begin with the values *S* (t = 0), *I* (t = 0), and *R* (t = 0), respectively. By combining the three previous equations, we arrive to the conclusion that $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$, which tells us that *S(t) + I(t) + R(t)* = Constant = *N*. As a result, *S, I*, and *R* are constrained by *N*.

1.4.4 The SIR model with demography

Assuming there is a natural host lifetime of $\frac{1}{\alpha}$ day/years is the quickest, most straightforward, and most often used approach to incorporating demography into the SIR model. The rate at which individuals inside every epidemiological compartment succumb to natural mortality may thus be expressed as the mathematical constant α. It is essential to emphasise that this component is independent of the disease and is not meant to represent the pathogenicity of the infectious agent. This is something that has to be emphasised since it is crucial. It has been assumed throughout history that μ also indicates the population's crude birth rate. This ensures that the entire population number does not vary over the course of time, or, to put it another way, $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$**.** The SIR model, which takes into account both births and deaths, may thus be characterised as:

$$\frac{dS}{dt} = \mu - \beta SI - \alpha S$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \alpha I$$

$$\frac{dR}{dt} = \gamma I - \alpha R$$

Assuming beginning circumstances of S(t=0) greater than zero, I(t=0) greater than zero, and R(t=0) greater than zero. For the purpose of this model, it is necessary to initiate the formula for the reproduction number R_0. The parameter stands for the transmission rate per infectious, and the fact that the equation contains negative components tells us that each person spends an average of $\frac{1}{(\alpha+\gamma)}$ time units in this class. Therefore, if we assume that everyone in the community is at risk for contracting the disease, we can calculate the average number of new infections that occur per infected person using the formula $R_0 = \frac{\beta}{(\alpha+\gamma)}$. The consideration of demographic dynamics may make it possible for a disease to be eradicated from a population or to remain there over the long run. Because of this, it is very essential to investigate what takes on while the system is in a state of equilibrium.

1.4.5 Delay Differential Equation

A delay differential equation (DDE) is a type of differential equation that incorporates time delays. Unlike ordinary differential equations (ODEs), where the derivative of a function depends only on the current time, DDEs involve derivatives that depend on the function's past values. The general form of a delay differential equation can be represented as follows:

$$\frac{dy(t)}{dx} = f(y(t), y(t-\tau_1), y(t-\tau_2), y(t-\tau_3) \dots \dots \dots)$$

In this equation, *y(t)* represents the unknown function, τ_1, τ_2, ... are positive time delays, and '*f* 'represents a given function that describes the dynamics of the system. The function *f* can involve the present state of the system, as well as the state values at previous times determined by the delays.

Analytically solving DDEs can be challenging due to their inherent mathematical complexity. Unlike ODEs, DDEs require initial conditions over a range of past time values to determine the solution accurately. Therefore, numerical methods are often employed to approximate solutions to DDEs.

1.5 Mathematical Preliminaries

1.5.1 Existence of Unique, Bounded and Positive Solution of Delay differential equation

A differential equation is said to be in the form of a delay differential equation if the derivative of the current time relies on the solution as well as derivatives of preceding times. Here an initial history function, rather than an initial condition, needs to be defined. A delayed state variable can be used to demonstrate the past dependence of a differential equation. The derivative of state variable is not required in this case. The corresponding delay differential equation with a single delay $\tau > 0$ is given by (Smith, 2010)

$$\dot{x}(t) = f(x, x(t), x(t-\tau)) \tag{1.1}$$

Suppose that $f(t, x, y)$ and $f_x(t, x, y)$ are continuous on R^3. Let $s \in R$ and $\emptyset: [s-\tau, s] \to R$ be continuous. We seek a solution $x(t)$ of equation (1.1) satisfying

$$x(t) = \emptyset(t), t \in [s-\tau, s], x(0) = x_0 \tag{1.2}$$

And satisfying equation (1.1) on $t \in [s,\ s+\sigma]$ for some $\sigma > 0$.

Theorem 1.5.1 (Existence of unique solution). Let $f(t, x, y)$ and $f_x(t, x, y)$ are continuous on R^3. Let $s \in R$ and $\emptyset: [s-\tau, s] \to R$ be continuous. Then there exists $\sigma > s$ and a unique solution of the initial-value problem (1.1)-(1.2) on $[s-\tau, \sigma]$.

$$\dot{x}(t) = f(x, x(t), x(t-\tau)) \tag{1.3}$$

Theorem 1.5.2 (Boundedness of solution). Let f satisfy the hypothesis of theorem1.5.1 and let $x: [s-\tau, \sigma) \to R$ be the noncontinuable solution of the initial value problem (1.1)- (1.2). If $\sigma < \infty$ then $\lim_{t \to \sigma-} |x(t)| = \infty$

Remark 1.5.3 Theorems 1.5.1 and 1.5.2 extend immediately to the case that $x \in R^n$ and $f: R \times R^n \times R^n \to R^n$, it also extends to multiple discrete delays $\tau_0 < \tau_1 < \cdots < \tau_m$ where $f = f(t, x(t), x(t-\tau_0), x(t-\tau_1), \ldots, x(t-\tau_m))$.

Theorem 1.5.4 (Positivity of solution). Suppose that $f: R \times R_+^n \times R_+^n \to R^n$satisfies the hypothesis of theorem 1.5.1 and remark 1.5.3 and for all i, t and for all $x, y \in R_+^n$: $x_i = 0 \Rightarrow f_i(t, x, y) \geq 0$,If the initial data $\emptyset$ in equation (1.2) satisfy $\emptyset \geq 0$, then the corresponding solution $x(t)$ of equation (1.1) satisfy $x(t) \geq 0$ for all $t \geq s$ where it is defined.

1.5.2 Stability by Variational matrix method

Let an autonomous system of equations be

$$\frac{dy}{dt} = f(y) \tag{1.4}$$

Where y is an n-tuple vector i.e. $y = (y_1, y_2, - - - y_n)$. Let $\phi(t)$ be the solution of system (1.3). The linear part of the expansion of the system (1.3) about $\phi(t)$ is given by the variational equation of the system (1.3) with respect to $\phi(t)$, written as

$$\frac{dx}{dt} = f_y(\phi(t))x \tag{1.5}$$

Where $f_y(\phi(t)) = \frac{df_i}{(dy_j)_{n \times n}}$ at $\phi(t)$. Since the stability of the variational system depicts the stability of any solution of a non- linear system governed by it, so stability of $x = 0$ of (1.4) determines the stability of $y = \phi(t)$ of (1.3). Particularly, when $\phi(t) = \phi_0$, a constant, the system (1.3) becomes

$$\frac{dx}{dt} = A \tag{1.6}$$

Where $A = f_y(\phi_0)$. Since a small perturbation of the system (1.3) is represented by system (1.4), so the stability of $y = \phi_0$ of (1.5) actually gives the stability of the solution of $x = 0$ of (1.4). The description of stability of every solution of $x' = Ax$ is given by following theorems.(Ahmad & Rao, 2014)

Theorem 1.5.5 If all the latent roots of A have negative real parts, then every solution of the system $x' = Ax$,where $A = (a_{ij})$ is a constant matrix, is asymptotically stable.

Theorem 1.5.6 If all the latent values of matrix A with multiplicity grater than one has negative real parts and all its roots with multiplicity one has non-positive real parts, then all the solutions of the system $x' = Ax$ are bounded and hence stable. Following theorem (Ahmad & Rao, 2014)to determine the sign of real parts of the roots of characteristic equation.

Theorem 1.5.7 Hurwitz's Theorem. A necessary and sufficient condition for the negativity of the real parts of all the roots of the polynomial

$L(\lambda) = \lambda^n + a_1\lambda^{n-1} + a_2\lambda^{n-2} + \ - - - - + a_n$ with real coefficients is the positivity of all the principal diagonals of the minors of the Hurwitz matrix

$$H_n = \begin{bmatrix} a_1 & 1 & 0 & 0 & 0 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & 1 & 0 & 0 & \cdots & 0 \\ a_5 & a_4 & a_3 & a_2 & a_1 & 1 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & . & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \cdots & 0 \end{bmatrix}$$

Theorem 1.5.8. Let $\varsigma_1, \varsigma_2 \ldots . \varsigma_m$ are all non-negative and $\zeta_i^{\,j} (j = 0,1,2, \ldots m: i = 1,2, \ldots n)$ are constants. As $(\varsigma_1, \varsigma_2, \ldots, \varsigma_m)$ vary, the sum of the orders of the zeros of exponential polynomial $P(\chi, e^{-\chi\varsigma_1}, \ldots ., e^{-\chi\varsigma_m})$ on the open half plane can change only if a zero appears on or crosses the imaginary axis, where;

$$P(\chi, e^{-\chi\varsigma_1}, \ldots ., e^{-\chi\varsigma_m}) = \chi^n + {\zeta_1}^0\chi^{n-1} + \cdots + {\zeta_{n-1}}^0\chi^n + {\zeta_n}^0 + \left[{\zeta_1}^1\chi^{n-1} + \cdots + {\zeta_{n-1}}^1\chi^n + {\zeta_n}^1\right]e^{-\chi\varsigma_1} + \cdots + [{\zeta_1}^m\chi^{n-1} + \cdots + {\zeta_{n-1}}^m\chi^n + {\zeta_n}^m]e^{-\chi\varsigma_m}$$

Ruan and Wei (Ruan & Wei, 2001), (Ruan & Wei, 2003a)proved this theorem using Rouches theorem (Dieudonne, 1960).

1.6 Hopf-Bifurcation

The expansion of the problem from two dimensions to higher dimensions was the most important contribution that Hopf made. The term "Poincaré-Andronov-Hopf bifurcation" is frequently used interchangeably with the term "Hopf bifurcation." (Marsden et al., 1978) The Hopf-bifurcation theorem provides a description of the manner in which a topological property of a flow changes as a result of the variation of one or more parameters. The most important thing to realise about flows is that their local behaviour will be entirely dictated by the linearized flow if the critical point gives the hyperbolic. In light of this, it can be deduced that the eigenvalues of the linearized flow at the stationary point will all have real parts that are not equal to zero. As a result, bifurcations of stationary points are only able to take place at parameter values for which a stationary point does not have a hyperbolic slope. A bifurcation value of a parameter is a value at which there is a shift in the qualitative character of the flow. This is a more exact definition. Because it requires a non-hyperbolic stationary point with linearized eigenvalues $\mp i\omega$, and therefore a two-dimensional centre manifold, and bifurcating solutions are periodic rather than stationary, the Hopf bifurcation is many orders of magnitude more difficult to investigate.

Theorem 1.6.1. Hopf-Bifurcation Theorem.

Let us consider one parameter family of delay equations

$$x'(t) = (x_t, \mu) \tag{1.7}$$

Where $F: C \times R \to R^n$ is a twice continuously differentiable in its arguments and $x = 0$ is a steady state for all values of μ: $F(0, \mu) \equiv 0$.

We may linearize F about $\emptyset = 0$ as follows

$$F(\emptyset, \mu) = L(\mu)\emptyset + f(\emptyset, \mu)$$

Where $L(\mu): C \to R^n$ is a bounded linear operator and f is higher order

$$\lim_{\emptyset \to 0} \frac{|f(\emptyset, \mu)|}{\|\emptyset\|} = 0$$

Following is the characteristic equation about L:

$$|\lambda I - A(\mu, \lambda)| = 0, A_{ij}(\mu) = L(\mu)_i\left(e_\lambda e_j\right)$$

The roots of this equation constitute the main assumption. (H) The latent equation w having a pair of simple roots $\mp i\omega$ with $\omega_0 \neq 0$ and no other root that is an integer multiple of $i\omega_0$ for $\mu = 0$

Here a root of order one means (Pandey et al., 2016) a simple root. If the characteristic equation is written as $h(\mu, \lambda) = 0$, then (H) implies $h_\lambda(0, i\omega_0) \neq 0$. So, by the implicit function theorem, there exists a continuously differentiable family of roots $\lambda = \lambda(\mu) = \alpha(\mu) + i\omega(\mu)$ for small μ satisfying $\lambda(0) = i\omega_0$. In particular, $\alpha(0) = 0$ and $\omega(0) = \omega_0$. Next assumption is that as μ increases through zero, the line of imaginary axis is crossed transversally by these roots. Actually, the assumption is:

$$\alpha'(0) > 0 \qquad (1.8)$$

In case $\alpha'(0) < 0$, we always ensure that equation (1.18) holds by changing the sign of the parameter i.e. we take parameter $v = -\mu$. Thus, the positive sign is basically a normalization which ensures that if $\mu < 0$, then the pair of roots has a negative real part and if $\mu > 0$, then it has positive real part.

Theorem 1.6.2. Let **(**H**)** and equation (1.8) hold. Then there exist $\varepsilon_0 > 0$, real valued even function $\mu(\varepsilon)$ and $T(\varepsilon) > 0$ satisfying $\mu(0) = 0$ and $T(\varepsilon) = {}^{2\pi}/_{\omega_0}$, and a non-constant $T(\varepsilon)$- periodic function $p(t, \varepsilon)$ with all functions being continuously differentiable in ε for $|\varepsilon| < \varepsilon_0$, such that $p(t, \varepsilon)$ is a solution of equation (1.7) and $p(t, \varepsilon) = \varepsilon\, q(t, \varepsilon)$ where $q(t, 0)$ is a ${}^{2\pi}/_{\omega_0}$-periodic solution of $q' = L(0)q$.

$$\mu(\varepsilon) = \mu_1\varepsilon^2 + O(\varepsilon^4) \qquad (1.9)$$

$$T(\varepsilon) = \frac{2\pi}{\omega_0}[1 + \tau_1\varepsilon^2 + O(\varepsilon^4)] \qquad (1.10)$$

Moreover, there exist $\mu_0, \beta_0, \delta > 0$ such that if equation (1.7) has a non-constant periodic solution $x(t)$ of period P for some μ satisfying $|\mu| < \mu_0$ with $max_t|x_t| < \beta_0$ and $\left|P - {}^{2\pi}/_{\omega_0}\right| < \delta$, then $\mu = \mu(\varepsilon)$ and $x(t) = p(t + \theta, \varepsilon)$ for some $|\varepsilon| < \varepsilon_0$ and some θ. If F is five times continuously differentiable then, all other latent roots for $\mu = 0$ have strictly negative real parts except for $\mp i\omega$ then $p(t, \varepsilon)$ is asymptotically stable if $\mu_1 > 0$ and unstable if $\mu_1 < 0$.

1.7 Sensitivity Analysis of State Variables with respect to Model Parameters

Systematic evaluation of the effects of model parameters on system solutions is called sensitivity analysis. There are number of methods to do sensitivity analysis of systems without delay, but there are only a few methods for sensitivity analysis of systems involving delays. The knowledge of how a small change in model parameter can bring change in the state variable, can be a great help in modelling process. It helps in elimination of ineffective and irrelevant parameters. It gives a complete insight into the overall behaviour of the proposed model.

If all the parameters in the given system (1.1) - (1.2) are considered to be constants, then sensitivity analysis includes just the calculation of partial derivatives of solution with respect to each parameter (Rihan, 2003a). The matrix of sensitivity functions is of the form:

$$S(t) \equiv S(t, \alpha) = \left[\frac{\partial}{\partial \alpha}\right]^T x(t, \alpha) \qquad (1.11)$$

Its jth column is: $S_j(t, \alpha) = \left[\frac{\partial x_j(t,\alpha)}{\partial \alpha_1}, \frac{\partial x_j(t,\alpha)}{\partial \alpha_2}, \dots, \frac{\partial x_j(t,\alpha)}{\partial \alpha_n}\right]^T$

This column vector gives sensitivity of the solution $x_j(t, \alpha)$ for small change in parameter $\alpha_i, i = 1,2,3, \dots, n$.

Theorem 1.7.1 $S(t)$ satisfies the delay differential equation:

$$S'(t) = J(t)S(t) + J_\tau(t)S(t-\tau) + B(t), t \geq 0 \qquad (1.12)$$

Where $J(t) = \frac{\partial}{\partial x} f(t, x, x_\tau)$, $J_\tau(t) = \frac{\partial}{\partial x_\tau} f(t, x, x_\tau)$, $B(t) = \frac{\partial}{\partial \alpha} f(t, x, x_\tau)$

1.8 Summary

This thesis consists of five chapters whose detail is as follows:

In chapter-1, A general information is given about the infectious diseases, its various stages and also some infectious diseases which was spread in the history. Infectious diseases take time to develop, therefore the impact is not seen right away. This delay might be brought on by the gestation period, incubation, mating, or the detection of sick individuals. Incorporating and using delay differential equations in the modelling of infectious diseases is made possible by this. The proposed study will be focus on simulating infectious illnesses, conducting stability analyses, and monitoring bifurcations caused by crucial delay factors. The basic terms and the theorems are explained in this chapter.

In chaper-2, The study of stability of SIR model is examined with a non-linear incidence function. Delay breaks the system, causing limit cycles and sporadic solutions. Equilibrium E* stabilising after early fluctuations without a delay. An infected person can be adequately treated and the system will stabilize if a critical delay τ is shorter than that value. As the delay τ constant varies below 2.241, the equilibrium E* loses stability and becomes asymptotical. The equilibrium E* undergoes a Hopf-bifurcation after reaching to the value 2.241.

The system involving all three states will always repeat the epidemic if the delay crosses the critical threshold. The direction analysis of the model has been performed which reveals the direction, amplitude, and periodicity of the bifurcating solutions as per the criteria given by Hassard (Schneider, 1982). Not only delay but other model parameters also affect the system. Thus, the sensitivity with model parameters and basic reproduction number R_0 are tested. Disease transmission rate α_2 affects the system greatly. As

transmission rate decreases, the system changes from Hopf bifurcation to asymptotic stability to absolute stability. The model's dynamics and basic reproductive number R_0 are investigated. We studied the threshold parameter R_0 factors that ensured the endemic and infection-free equilibria's asymptotic stability, $R_0 > 1$ makes the disease endemic and spreads in the neighbourhood, while $R_0 < 1$ eliminates it. The simple DDE model has chaotic and quasiperiodic dynamics and is suitable for high population numbers.

In chapter-3, With the assistance of the suggested model, an analysis of the traditional SIR model may be carried out using DDE. The delay causes the system to become unstable and sets off a complicated pattern of behaviour that includes limit cycles and stable periodic solutions through hopf-bifurcation. An investigation of the equilibrium's degree of stability, E*, is carried out. After the first few variations, the equilibrium E* tends to remain stable when there is no delay in the process. It indicates that if the delay in the identification of contaminated section is below a certain value, the suspected and infected may be recovered well, and the system will become stable. The equilibrium E* begins to lose its stability and eventually leads to asymptotic stability when the value of the delay parameter τ falls below the critical point, which is denoted by the number $\tau < 8.5$. When the delay parameter reaches the critical value, which is denoted by the number $\tau \geq 8.5$, the equilibrium E* displays the complicated dynamics in the form of a Hopf bifurcation. This indicates that if the delay exceeds the critical value in the identification of infected individuals, the system that involves all three states and is involved in the epidemic will constantly remain in the vicious loop of repeating the epidemic after each cycle of delay length. The periodic solutions that are stable and have a big amplitude as well as a limit cycle trajectory.

In chapter-4, An SIR model with virus mutation is analysed using delay differential equations. When the virus mutates, the recovered population losses its immunity and becomes susceptible again. The incubation period is denoted by τ. For the study of stability, feasible non-zero equilibrium point E^* has been considered. The delay factor has been incorporated in the term

defining recovered hosts. The nature of the roots of exponential characteristic equation formed by the system of equation has been studied in detail using Routh-Hurwitz criteria and results of theory of equations with the help of lemmas. It is found that the system shows asymptotic satiability as long as the value of the delay parameter τ is less than the critical point value that is $\tau < 7.15$. But, as soon as the value of time lag parameter τ crosses the critical point value that is $\tau \geq 7.15$, the system losses stability and the limit cycles are seen via Hopf- bifurcation. The period, direction and stability of these bifurcating periodic solutions have also been determined. The conditions for supercritical and subcritical bifurcating solutions have been laid down using an algorithm given by (Schneider, 1982). Sensitivity analysis has been carried out, telling us about the model parameters that are responsible for dynamic behaviour of the equilibrium point, apart from the delay parameter τ. We used the direct method of sensitivity given by (Rihan, 2003b). The system is very sensitive to the model parameter 'c' as well as the parameter 'a' which represents the rate of mutation and growth rate respectively. As the value of 'c' and 'a' increase, the state variables S(t), I(t) and R(t) tends to move from limit cycles to more of asymptotic stability and finally to absolute stability.

The effect of reproduction number R_0 on the shapes of infected and recovered population is studied. The figures 12 shows that the peak of infected population is reached on 11^{th} day when the value of reproduction number $R_0 = 0.5$. But the infection spreads comparatively fast if the value of reproduction number increases to $R_0 = 1.33$ and the peak of infected population is reached on 3^{rd} day. The infection spreads so rapidly that the steepest of these peaks of infected population is reached on 2^{nd} day only, if the value of reproduction number $R_0 = 10$, the recovery rises and the system approaches stability with increasing values of R_0.

In chapter-5, The global dynamic behavior of a SIR model with incubation period are investigated, and the susceptible population is shown to expand in a logistic fashion. There is a critical value of $\Re_0$ in the system that controls

how the disease behaves. The impact of incubation period on the dynamic behaviours of systems are the focus of this research. This paper's findings demonstrate that the proximity to an endemic value and the stability of solutions are dependent on two variables: the incubation period duration and the threshold value $\Re_0$. System has global asymptotic stability in the disease-free equilibrium E^* if $\Re_0 = 1$. In the case where $\Re_0 > 1$, E^* is no longer stable, and the endemic equilibrium E^* is indeed irreversible. The requisites for achieving local stability and Hopf bifurcation at E^* were derived by treating the delay time as a parameter. If $1 < \Re_0 \leq 2$, then E^* is absolutely stable; that is, E^* is always locally stable when τ is non zero. When $\Re_0 > 2$, E^* is conditionally stable in the range $\tau \in (0, \tau_0)$, as soon as the value of τ surpasses the critical value τ_0 i.e., $\tau > \tau_0$, E^*become unstable and shows Hopf-bifurcations. This chapter shows that for short time delay in has no effect on the positive invariance. Even the boundedness of solutions, the global stability of DFE, or the permanence of the endemic equilibrium remains intact. However, for long delays, the delay time can destabilize the unique endemic equilibrium, and a variety of stable periodic solutions through Hopf bifurcation. Our theoretical findings are reflected in the results of the numerical simulations that were performed.

Chapter 2

Analysis of Classic SIR Model using Delay Differential Equations

2.1 Introduction

For both humans and animals, illnesses and infections have long posed a serious hazard. The phrase "communicable illnesses" refers to conditions that transfer from person to person by indirect or direct physical contact. Diseases and illnesses may be transmitted by contact with a virus-carrying person or by breathing the virus. They have the potential to spread and become endemic, resulting in both physical and financial suffering. Millions of people die each year from these diseases, most of them in developing countries. Mathematical modelling makes it feasible to investigate these disorders from a variety of perspectives, comprehend transmission rates, estimate future losses, and devise preventative strategies. For the investigation of endemic disease transmission rates, several models have been developed (Anderson & May, 1979a; Diekmann & Heesterbeek, 2000; Hethcote & Tudor, 1980a; Huo & Ma, 2010; Koff, 1992; McCluskey, 2010a; Singh, 2022; Xiao & Ruan, 2007a).

In these models of infection propagation, three fundamental epidemiological concepts are usually used: the susceptible chunk of the peoples S(t), the infected people I(t), and the recovered one R(t). To estimate theoretically the number of persons affected by an infectious illness, a straight forward SIR model was devised ("Contributions to the Mathematical Theory of Epidemics. II. The Problem of Endemicity," 1932). The dynamic transmission process between infected and susceptible is modelled in simple SIR models, and this modelling determines how simple SIR models behave. SIR models do an adequate job at representing viral agent illnesses including measles, mumps, and smallpox. Numerous researchers have modelled and examined such infectious illnesses (A. Rihan et al., 2012; Berezovsky et al., 2005a; Cai et al., 2009a; d'Onofrio et al., 2007; D'Onofrio et al., 2007; Esteva & Matias, 2001; Hsu & Zee, 2004). Some illnesses, like the flu and TB, have an incubation or

latent period. It is claimed that they are infected yet not contagious throughout the incubation phase, and can be precisely modelled by the delay parameter in SIR models using DDE. The worldwide stability of the SIR epidemic model with distributed delays has been examined (Beretta et al., 2001). Additionally, the prerequisites for the endemic equilibrium's asymptotic stability for all potential delays have been established (X. Song & Cheng, 2005).

Global asymptotic stability of endemic and disease-free equilibria is studied by the vast bulk of mathematical models. Many of these models, however, are strict, necessitating precise numerical analysis. Changes to state variables and settings can have far-reaching effects on the system's behavior. In a comprehensive qualitative analysis, a saturated incidence rate is used to highlight the features of the delayed SIR model that contribute to the asymptotically stable nature of the associated steady states. Stability of the DDE model is discussed in terms of time lag, under the circumstances under which it holds. Hopf bifurcation analysis is also covered in detail (Rihan & Anwar, 2012)**.** Absolute consistency, conditional consistency, and bifurcation consistency were all studied in depth for a predator-prey system with discontinuous delays (Ruan, 2001). Deep-rooted analysis was done on the exponential characteristic equation's zero (Ruan & Wei, 2003b).

The influence of reproduction number on the forms of the infected and recovered population is investigated, and two alternative population layouts are reported (Park & Bolker, 2020). (Rihan, 2003b) Introduced adjoint and direct sensitivity analysis techniques for delay differential equation-based numerical modelling. A nonlinear system of delay differential equations is used to determine the stability of equilibria points. It has been shown that the SIR model's solutions are constrained, and stability with a limited incubation time has been explored (Takeuchi et al., 2000) .SIR epidemic models having nonlinear incidence rates and scattered delays were examined for their stability (Elazzouzi et al., 2019), and two control measures taken into account in this model were vaccination terms and general treatment functions. (C. Yan & Jia, 2014) For a delayed SIR model with logistic growth, the stability and

Hopf bifurcation has been examined by using the reproduction number. Song Y, (Y. Song & Peng, 2006) Examines the use of a discrete and distributed delay logistic model. The existence of local Hopf bifurcations and the stability of the positive equilibrium have also been debated. (Abta et al., 2020) The influence of spatial diffusion of solution and delay on the dynamical behavior of the SIR epidemic model is investigated.

In light of the foregoing, the following mathematical model is used in this work to examine the function of delay in the traditional SIR model. A directional analysis and a stability study of the non-zero endemic equilibrium point is also carried out.

2.2 Mathematical Model

We'll use the symbol $\tau > 0$ to represent the incubation duration. The infected become a vector throughout the incubation phase, and it is not until after this time that they develop the infectious disease. let $S(t)$, $I(t)$, and $R(t)$, be the three state variables; susceptible, infected, and recovered populations respectively. The resulting design is as follows:

$$\frac{dS}{dt} = \alpha_1 S\left(1 - \frac{S}{k}\right) - \alpha_2 SI(t-\tau) \qquad (2.1)$$

$$\frac{dI}{dt} = \alpha_2 SI(t-\tau) - (\alpha_3 + \delta_1)I \qquad (2.2)$$

$$\frac{dR}{dt} = \alpha_3 I - \delta_2 R \qquad (2.3)$$

Initially, $S(t), I(t), R(t)$ are positive for all the time t, and $I(t-\tau)$ is constant for $t \in [0, \tau]$.

Table 2.1 : The parameter analyzed by the model (2.1) – (2.3)

Parameter	Description
k	Carrying capacity
α_1	Natural birth rate

α_2	Rate of infection per encounter with infected hosts on susceptible hosts
α_3	The rate of recovery
δ_1	Infected hosts' mortality rate
δ_2	The mortality rate after recovery.
τ	Delay parameter or time lag

It is acceptable to assume that each parameter has constant positive values.

2.3 Non-Zero Epidemic Equilibrium

At the equilibrium point the underlying assumption is $I(t-\tau) \cong I$. $E^*(S^*, I^*, R^*)$ be the non-zero epidemic equilibrium determined as:

$\frac{dI^*}{dt} = 0 \Rightarrow S^* = \frac{(\alpha_3+\delta_1)}{\alpha_2}$

$\frac{dS^*}{dt} = 0 \Rightarrow I^* = \frac{\alpha_1}{{\alpha_2}^2 k}(k-1)(\alpha_3+\delta_1)$

$\frac{dR^*}{dt} = 0 \Rightarrow R^* = \frac{\alpha_1 \gamma}{{\alpha_2}^2 \delta_2 k}(k-1)(\alpha_3+\delta_1)$

So, we have $E^*(S^*, I^*, R^*) = E^*\left(\frac{(\alpha_3+\delta_1)}{\alpha_2}, \frac{\alpha_1}{\alpha_2 k}(k-1)(\alpha_3+\delta_1), \frac{\alpha_1\alpha_3}{{\alpha_2}^2\delta_2 k}(k-1)(\alpha_3+\delta_1)\right)$

2.4 Stability And Bifurcation Analysis

The set of equations (2.1) – (2.3) is transformed into the following equations at the equilibrium $E^*(S^*, I^*, R^*)$

$$\frac{dS^*}{dt} = \alpha_1 S^*\left(1 - \frac{S^*}{k}\right) - \alpha_2 S^* I^*(t-\tau) \quad (2.4)$$

$$\frac{dI^*}{dt} = \alpha_2 S^* I^*(t-\tau) - (\alpha_3+\delta_1) I^* \quad (2.5)$$

$$\frac{dR^*}{dt} = \alpha_3 I^* - \delta_2 R^* \quad (2.6)$$

Y. Takeuchi et.al (Takeuchi et al., 2000) have shown the boundedness of all possible solutions for systems (2.1) – (2.3) and that of systems (2.4)-(2.6). The set of equations (2.4) and (2.5), serve as the primary determinants of the system's dynamics. The transcendental equation about the equilibrium $E^*$is given by:

$$\begin{vmatrix} \lambda - d_1 & -d_2 \\ -d_3 & \lambda - d_4 \end{vmatrix} = 0 \qquad (2.7)$$

Where d_1, d_2 are the partial derivative of equations (2.4), (2.5) w.r.t S^*And d_3, d_4 are that of w.r.t to I^*. $d_1 = \alpha_1 - \frac{2\alpha_1}{k}S^*$, $d_2 = 0$, $d_3 = -\alpha_2 S^* e^{-\lambda\tau}$, $d_4 = \alpha_2 S^* e^{-\lambda\tau} - (\alpha_3 + \delta_1)$.

By expanding the determinant (2.7) we have:

$$\lambda^2 - (d_1 + d_4)\lambda + d_1.d_4 - d_3.d_2 = 0$$

By substituting the values of $d_1, d_2\, d_3,\ and\ d_4$ we have the following equation $\lambda^2 + a_1\lambda + a_2 + (a_3\,\lambda + a_4)e^{-\lambda\tau} = 0$ (2.8)

Where $a_1 = \left(\frac{2\alpha_1}{k}S^* + \alpha_3 + \delta_1 - \alpha_1\right), a_2 = \left(\frac{2\alpha_1}{k}S^* - \alpha\right)(\alpha_3 + \delta_1), a_3 = -\alpha_2 S^*$ $a_4 = \alpha_2 S^*\left(\alpha_1 - \frac{2\alpha_1}{k}S^*\right)$

When $\tau = 0$, the equation (2.8) implies:

$$\lambda^2 + (a_1 + a_3)\lambda + (a_2 + a_4) = 0 \qquad (2.9)$$

The roots of (2.9) contain a -ve real component according to Routh- Hurwitz's, suggesting that the system is stable if $(\mathbb{H}_1)$: $(a_1 + a_3) > 0$, $(\mathbb{H}_2)$: $(a_2 + a_4) > 0$

We now want to investigate how changing the value of τ affects the roots' negative real to the positive real part.

If $\lambda = i\omega$ is a root of (2.8) , then we have the following;

$$(i\omega)^2 + a_1(i\omega) + a_2 + (a_3(i\omega) + a_4)e^{-(i\omega)\tau} = 0$$

$$\Rightarrow -\omega^2 + a_1(i\omega) + a_2 + (a_3(i\omega) + a_4)(\cos(\omega\tau) - isin\,(\omega\tau)) = 0$$

Sorting out the real and imaginary, it follows that ω satisfies:

$$\omega^4 - ({a_3}^2 - {a_1}^2 + 2a_2)\omega^2 + ({a_2}^2 - {a_4}^2) = 0 \qquad (2.10)$$

The two roots of equation (2.10) are:

$$\omega_{1,2}^2 = \frac{({a_3}^2 - {a_1}^2 + 2a_2) \pm \sqrt{({a_3}^2 - {a_1}^2 + 2a_2)^2 - 4({a_2}^2 - {a_4}^2)}}{2} \qquad (2.11)$$

Both the two roots $\omega_{1,2}^2$ are non-positive if:

$(\mathbb{H}_3)$: $({a_3}^2 - {a_1}^2 + 2a_2) < 0 \; and \; ({a_2}^2 - {a_4}^2) > 0 \; or \; ({a_3}^2 - {a_1}^2 + 2a_2)^2 < 4({a_2}^2 - {a_4}^2)$

Therefore, if condition $(\mathbb{H}_3)$ is true, equation (2.10) has non-positive roots. The following Conjecture is made by Ruan. S.(Ruan, 2001)

Conjecture 1. Zeros of (2.8) have negative real part for all $\tau \geq 0$ If $(\mathbb{H}_1) - (\mathbb{H}_2)$ are true, However, if;

$(\mathbb{H}_4)$: $({a_2}^2 - {a_4}^2) < 0 \; or \; ({a_3}^2 - {a_1}^2 + 2a_2) > 0 \; and \; ({a_3}^2 - {a_1}^2 + 2a_2)^2 = 4({a_2}^2 - {a_4}^2)$, So, the equation (2.8) has +ve root ω_1^2. Using the same logic, if,

$(\mathbb{H}_5)$: $({a_2}^2 - {a_4}^2) > 0 \; or \; ({a_3}^2 - {a_1}^2 + 2a_2) > 0 \; and \; ({a_3}^2 - {a_1}^2 + 2a_2)^2 > 4({a_2}^2 - {a_4}^2)$, Then, the equation (2.8) has two +ve $\omega_{1,2}^2$ roots.

When delay τ takes values, the real part of the roots of equation (2.8) is zero. The latent values $\tau_c^{\pm}$ of τ can be determined as;

$$\tau_c^{\pm} = \frac{1}{\omega_{1,2}} \cos^{-1}\left[\frac{a_4(\omega_{1,2}^2 - a_2) - a_1 a_3 \omega_{1,2}^2}{{a_3}^2 \omega_{1,2}^2 + {a_4}^2}\right] + \frac{2j\pi}{\omega_{1,2}}, c = 0,1,2, \ldots \qquad (2.12)$$

Ruan. S.'s. (Ruan, 2001) The following supposition summarizes the previous findings.

Conjecture 2. (I). The roots of equation (2.8) are wholly imaginary i.e $\pm i\omega_1$ if $(\mathbb{H}_1) - (\mathbb{H}_2) \; and \; (\mathbb{H}_4)$ hold and $\tau = \tau_c^+$.

(II) The roots of equation (2.8) are wholly imaginary $\pm i\omega_1 (\pm i\omega_2$ respectively) if $(\mathbb{H}_1) - (\mathbb{H}_2)\ and\ (\mathbb{H}_5)$ are valid and that $\tau = \tau_c^+ (\tau = \tau_c^-)$ respectively.

We anticipate that at $\tau > \tau_c^+$ and $\tau < \tau_c^-$ some roots of equation (2.8) will shift from having a negative real part to having a positive real part. Let's notice the following to investigate its potential. Let $\tau_c^{\pm} = \mu_c^{\pm}(\tau) + i\omega_c^{\pm}(\tau); c = 0,1,2,3, \ldots$

The equation (2.8) satisfies: $\mu_c^{\pm}(\tau_c^{\pm}) = 0, \omega_c^{\pm}(\tau_l^{\pm})$ =$\omega_{1,2}$

As indicated below, we can verify that the transversality criteria are accurate; $\frac{d}{d\tau}\left(\text{Re}\ \lambda_c^+(\tau_c^+)\right) > 0\ and\ \frac{d}{d\tau}\left(\text{Re}\ \lambda_c^-(\tau_c^-)\right) < 0$

In other words, $\tau_c^{\pm}$ is a bifurcation value. The zeros of equation (2.8) are scattered according to the following hypothesis.

Hypothesis: if $\tau_c^+ (c = 0,1,2,3, \ldots)$ be defined by equation (2.12)

(I) we have the negative real part for all the roots of (2.8), if $(\mathbb{H}_1), (\mathbb{H}_2)$ is true, for all $\tau \geq 0$.

(II) we have the negative real part for all the roots of (2.8), if $(\mathbb{H}_1), (\mathbb{H}_2)\ , and\ (\mathbb{H}_4)$ hold and when $\tau \in [0, \tau_0^+)$. further, if $\tau = \tau_0^+$, equation has a pair of +ve complex roots $\pm i\omega_1$, also at minimum one root with +ve real part if $\tau > \tau_0^+$.

(III) If $(\mathbb{H}_1), (\mathbb{H}_2)\ , and\ (\mathbb{H}_5)$ are true, then there is a +ve integer k such that we have k shift from stability to instability defined as $0 < \tau_0^+ < \tau_0^- < \tau_1^+ < \tau_1^- - - - < \tau_{k-1}^- < \tau_k^+$. This indicates that when $\tau \in [0, \tau_0^+), (\tau_0^-, \tau_1^+), - - -, (\tau_{k-1}^-, \tau_k^+)$, all the roots of (7) have negative real components. further if $\tau \in (\tau_0^+, \tau_0^-), (\tau_1^+, \tau_1^-), - - -, (\tau_{k-1}^+, \tau_{k-1}^-)$ and $\tau > \tau_k^+$, (2.8) has minimum one root with +ve real components.

2.5 Directional analysis of Hopf-bifurcating solution:

We find a sequence of repeated solutions that bifurcate from the positive steady state E* at the critical point. It is also crucial to think about the solutions' period, stability, and the way in which they bifurcate. In this part, we'll use normal form theory and manifold reduction by K. R. Schneider, Hassard, and B. D. (Schneider, 1982) to get an explicit formula for determining the Hopf-properties bifurcations at the critical Point.

Let $U_1 = S(t\tau) - S^*(t), U_2 = I(t\tau) - I^*(t), U_3 = R(t\tau) - R^*(t)$ and the delay τ can be normalizing by time scaling $t \to \frac{t}{\tau}$, the system of equations (2.1) - (2.2) are transformed into

$$\left.\begin{aligned} \frac{dU_1}{dt} &= \left(\alpha_1 - \frac{2\alpha_1}{k}S^*\right)U_1 - \frac{\alpha_1}{k}{U_1}^2 - \alpha_2 U_1.U_2(t-1) - \alpha_2 S^*.U_2(t-1) \\ \frac{dU_2}{dt} &= \alpha_2 S^*.U_2(t-1) + \alpha_2 U_1.U_2(t-1) - (\alpha_3 + \delta_1).U_2 \end{aligned}\right\} \quad (2.13)$$

Thus, in phase $\mathfrak{D} = \mathfrak{D}\left([-1,0], {\mathcal{R}_+}^2\right)$ work is possible. We indicate the critical value τ_j by τ_0 without loss of the generality. Let $\tau = \tau_0 + \mu$, then the system of equations (2.13)
has $\mu = 0$ a Hopf-bifurcation value. Rewrite this system as follows for ease of notation:

$$v'(t) = L_\mu(U_t) + F(\mu, v_t) \quad (2.14)$$

Where $v(t) = (v_1(t), v_2(t))^T \in \mathcal{R}^2, v_t(\theta) \in \mathfrak{D}$ is defined by $v_t(\theta) = v_t(t+\theta)$, and

$L_\mu: \mathfrak{D} \to \mathcal{R}$, $F: \mathcal{R} \times \mathfrak{D} \to \mathcal{R}$ are given, respectively by

$$L_\mu \emptyset = (\tau_0 + \mu)\begin{bmatrix} \left(\alpha_1 - \frac{2\alpha_1}{k}S^*\right) & 0 \\ 0 & -(\alpha_3 + \delta_1) \end{bmatrix}\begin{bmatrix} \emptyset_1(0) \\ \emptyset_2(0) \end{bmatrix} + (\tau_0 + \mu)\begin{bmatrix} 0 & -\alpha_2 S^* \\ 0 & \alpha_2 S^* \end{bmatrix}\begin{bmatrix} \emptyset_1(-1) \\ \emptyset_2(-1) \end{bmatrix}$$

And $F(\mu,\emptyset) = (\tau_0+\mu)\begin{bmatrix}F_1\\F_2\end{bmatrix}$ respectively where $F_1 = -\frac{\alpha_1}{k}\,\emptyset^2{}_1(0) - \alpha_2\emptyset_1(0)\emptyset_2(-1), F_2 = \alpha_2\emptyset_1(0)\emptyset_2(-1),$ $\emptyset(\theta) = \big(\emptyset_1(\theta),\emptyset_2(\theta)\big)^T \in \mathfrak{D}\big((-1{,}0),\ \mathcal{R}\big)$.

By the Representation theorem given by F. Riesz (Fuglede, 1955), there exists function $\eta(\theta,\mu)$ of bounded variation for $\theta \in [-1\,,0], \ni, L_\mu\emptyset = \int_{-1}^{0} d\,\eta(\theta,0)\emptyset(\theta)$ for $\emptyset \in \mathfrak{D}$

Here $L_\mu\emptyset = (\tau_0+\mu)\begin{bmatrix}\left(\alpha_1 - \frac{2\alpha_1}{k}S^*\right) & 0\\ 0 & -(\alpha_3+\delta_1)\end{bmatrix}\delta(\theta) + (\tau_0+\mu)\begin{bmatrix}0 & -\alpha_2 S^*\\ 0 & \alpha_2 S^*\end{bmatrix}\delta(\theta+1)$

δ represent the Dirac delta function.

For $\emptyset \in \mathfrak{D}\big([-1{,}0],\mathcal{R}_+{}^2\big)$, Then the system is equivalent to

$$v'(t) = \mathcal{A}(\mu)v_t + \mathcal{F}(\mu)v_t \tag{2.15}$$

For $\psi \in \mathfrak{D}^1\big([-1\,,0],\mathcal{R}_+{}^2\big)$, define

$$\mathcal{A}^*\psi(s) = \begin{cases} -\frac{d\psi(s)}{ds}, & s \in [-1\,,0)\\ \int_{-1}^{0} d\,\eta^T(-t\,,0)\,\psi(-t), & s = 0. \end{cases} \tag{2.16}$$

And bilinear inner product

$$< \psi(s), \emptyset(\theta) > = \overline{\psi(0)}\emptyset(0) - \int_{-1}^{0}\int_{\xi=\theta}^{\theta}\overline{\psi}(\xi-\theta)d\eta(\theta)\phi(\xi)\,d\xi \tag{2.17}$$

Since $i\omega_0$ are eigenvalues of $\mathcal{A}(0)\ and\ \mathcal{A}^*, \mathcal{A} = \mathcal{A}(0)$ are adjoint operators. Thus they become the latent values of A*. Suppose that $q(\theta) = q(0)e^{i\omega_0\theta}$ is latent vector of $\mathcal{A}(0)$ corresponding to the latent value $i\omega_0$. Then $\mathcal{A}(0) = i\omega_0\, q(\theta)$.

When $\theta = 0$, $\left[i\omega_0 I - \int_{-1}^{0} d\eta(\theta)e^{i\omega_0\theta}\right]q(0) = 0$, this gives $q(0) = (1, U_1)^T$ where $U_1 = \frac{i\omega_0 - \left(\alpha_1 - \frac{2\alpha_1}{k}S^*\right)}{(i\omega_0 + (\alpha_3+\delta_1))}$

By the same way it can be proved that $q^*(s) = D(1, {U_1}^*)e^{i\omega_0\tau_0 s}$ represent the eigen value of $\mathcal{A}^*$ corresponding to eigen vector $-i\omega_0$, where ${U_1}^* = \frac{-i\omega_0-\left(\alpha_1-\frac{2\alpha_1}{k}S^*\right)}{(-i\omega_0+(\alpha_3+\delta_1))}$ The value of D is required to assure that $< q^*(s), q(\theta) > = 1$, From equation (2.17),

$$< q^*(s), q(\theta)$$
$$> \overline{D}\left(1, \overline{{U_1}^*}\right)(1, U_1)^T$$
$$-\int_{-1}^{0}\int_{\xi=\theta}^{\theta} \overline{D}\left(1, \overline{{U_1}^*}\right)e^{-i\omega_0\tau_0(\xi-\theta)}d\eta(\theta)(1, U)^T e^{i\omega_0\tau_0}\, d\xi$$

$$= \overline{D}\left\{1 + U_1\overline{U^*} - \int_{-1}^{0}\left(1, \overline{{U_1}^*}\right)\theta e^{i\omega_0\tau_0\theta}(1, U_1)^T\right\}$$

$$= \overline{D}\left\{1 + U_1\overline{{U_1}^*} + \tau_0\overline{U^*}W^*(\alpha_2 U - \alpha_1 U)e^{i\omega_0\tau_0}\right\}$$

Hence, choose $\overline{D} = \frac{1}{\left\{1+U_1\overline{{U_1}^*}+\tau_0\overline{U^*}W^*(\alpha_2 U-\alpha_1 U)e^{i\omega_0\tau_0}\right\}}$

such that $< q^*(s), q(\theta) > = 1, < q^*(s), \overline{q(\theta)} > = 0$.

By following K. R. Schneider, Hassard, B. D (Schneider, 1982) approach we compute the coordinates describing the center manifold $\mathfrak{D}_0$ at $\mu = 0$ by using the same notations. manifold $\mathfrak{D}_0$ at $\mu = 0$. Let equation (2.13) has solution v_t with $\mu = 0$. We Define

$$z(t) =< q^*(s), v_t(\theta) >,\ \mathbb{W}(t, \theta) = v_t(\theta) - 2Re(z(t)q(\theta)) \quad (2.18)$$

On the center manifold $\mathcal{C}_0$, $\mathbb{W}(t, \theta) = \mathbb{W}\left(z(t), \overline{z(t)}, \theta\right)$

Where $\mathbb{W}(z, \overline{z}, \theta) = \mathbb{W}_{20}(\theta)\frac{z^2}{2} + \mathbb{W}_{11}(\theta)z\overline{z} + \mathbb{W}_{02}(\theta)\frac{\overline{z}^2}{2} + \cdots$,

In the direction of q^* and $\overline{q^*}$ the local coordinates for center manifold $\mathcal{C}_0$ are z and $\overline{z}$. Note that v_t is real is the necessary condition for $\mathbb{W}$ to be real. For solution $v_t \in \mathfrak{D}_0$ of equation (2.16), since $\mu = 0$ $z'(t) = i\omega_0\tau_0 z + < \overline{q^*}(\theta), F\left(0, \mathbb{W}(z, \overline{z}, \theta) + 2Re\left(z(t)q(\theta)\right)\right) >$

$= i\omega_0\tau_0 z + \overline{q^*}(0)\, F\big(0, \mathbb{W}(z,\overline{z},0) + 2Re\big(z(t)q(\theta)\big)\big) \equiv i\omega_0\tau_0 z + \overline{q^*}(0)F_0(z,\overline{z})$

Rewrite this equation as: $z'(t) = i\omega_0\tau_0 z(t) + g(z,\overline{z})$ (2.19)

Where $g(z,\overline{z}) = \overline{q^*}(0)F_0(z,\overline{z}) = g_{20}(\theta)\frac{z^2}{2} + g_{11}(\theta)z\overline{z} + g_{02}(\theta)\frac{\overline{z}^2}{2} + g_{21}(\theta)\frac{z^2\overline{z}}{2} + \cdots$ (2.20)

As $v_t(\theta) = (v_{1t}, v_{2t}\ ) = \mathbb{W}(t,\theta) + z\, q(\theta) + \overline{z}\overline{q(\theta)}$ and $q(0) = (1, u_1)^T \mathrm{e}^{\mathrm{i}\omega_0\tau_0\theta}$, so

As a result, we get: $g_{20} = \overline{D}(1, U_1)f_{z^2}$, $g_{02} = \overline{D}\big(1,\overline{U_1}\big)f_{\overline{z}^2}$, $g_{11} = \overline{D}\big(1,\overline{U_1}\big)f_{z\overline{z}}, g_{21} = \overline{D}\big(1,\overline{U_1}\big)f_{z^2\overline{z}}$

To find the value of g_{21}, the computation of $\mathbb{W}_{20}(\theta)$ and $\mathbb{W}_{11}(\theta)$ should be prioritized. From the equations (2.15) and (2.18) we have;

$$W' = v_t{}' - z'q - \overline{z}'q = \begin{cases} \mathcal{A}\mathbb{W} - 2Re\big[\overline{q^*}(0)F_0 q(\theta)\big], & \theta \in [-1,0) \\ \mathcal{A}\mathbb{W} - 2Re\big[\overline{q^*}(0)F_0 q(0)\big] + F_0, & \theta = 0 \end{cases}$$

Let $\mathrm{W}' = \mathcal{A}\mathbb{W} + H(z,\overline{z},\theta)$, (2.21)

Where $H(z,\overline{z},\theta) = H_{20}(\theta)\ \frac{z^2}{2} + H_{11}(\theta)z\overline{z} + H_{02}(\theta)\frac{\overline{z}^2}{2} + H_{21}(\theta)\frac{z^2\overline{z}}{2} + \cdots$, (2.22)

However, on $\mathfrak{D}_0$ near the origin we have $\mathbb{W}' = \mathbb{W}_z z' + \mathbb{W}_z\overline{z}'$

Simplifying the equating we get,

$\det(\mathcal{A} - 2i\omega_0 I)\,.\,\mathbb{W}_{20}(\theta) = -H_{20}(\theta),\ \mathcal{A}\mathbb{W}_{11}(\theta) = -H_{11}(\theta)$ (2.23)

And for $\theta \in [-1\,, 0)$, $H(z,\overline{z},\theta) = -\overline{q^*}(0)\overline{F_0}q(\theta) - \overline{q^*}(0)\overline{F_0}\,\overline{q}(\theta) = -gq(\theta) - \overline{g}\ \overline{q}(\theta)$

Comparing the coefficients with (2.23) for $\theta \in [-1,0]$ that

$H_{20}(\theta) = -g_{20}q(\theta) - \overline{g_{02}}\,\overline{q}(\theta),\ H_{11}(\theta) = -g_{11}q(\theta) - \overline{g_{11}}\,\overline{q}(\theta).$

From equations (2.19), (2.22) and the definition of A we obtain

$\mathbb{W}_{20}(\theta) = 2i\omega_0\tau_0\mathbb{W}_{20}(\theta) + g_{20}q(\theta) + \overline{g_{02}}\,\overline{q}(\theta)$

Solving for $\mathbb{W}_{20}(\theta)$: $\mathbb{W}_{20}(\theta) = \frac{ig_{20}}{\omega_0\tau_0}q(0)e^{i\omega_0\tau_0\theta} + \frac{i\overline{g_{02}}}{3\omega_0\tau_0}\overline{q}(0)e^{-i\omega_0\tau_0\theta} + \mathbb{E}_1 e^{2i\omega_0\tau_0\theta}$,

And similarly, $\mathbb{W}_{11}(\theta) = \frac{-ig_{11}}{\omega_0\tau_0}q(0)e^{i\omega_0\tau_0\theta} + \frac{i\overline{g_{11}}}{\omega_0\tau_0}\overline{q}(0)e^{-i\omega_0\tau_0\theta} + \mathbb{E}_2$

we can find a 3D vectors $\mathbb{E}_1$ and $\mathbb{E}_2$, by setting $\theta = 0$ in H. In fact since $H(z,\overline{z},\theta) = -2Re\left[\overline{q^*}(0)F_0q(0)\right] + F_0$, So

$H_{20}(\theta) = -g_{20}q(\theta) - \overline{g_{02}}\,\overline{q}(\theta) + F_{z^2}$, $H_{11}(\theta) = -g_{11}q(\theta) - \overline{g_{11}}\,\overline{q}(\theta) + F_{z\overline{z}}$

Where $F_0 = F_{z^2}\frac{z^2}{2} + F_{z\overline{z}}z\overline{z} + F_{\overline{z}^2}\frac{\overline{z}^2}{2} + \cdots$

Notice that $\left[i\omega_0\tau_0 I - \int_{-1}^{0} e^{i\omega_0\tau_0\theta}d\eta(\theta)\right]q(0) = 0$ and $\left[-i\omega_0\tau_0 I - \int_{-1}^{0} e^{-i\omega_0\tau_0\theta}d\eta(\theta)\right]\overline{q}(0) = 0$, Which Implies $\left[2i\omega_0\tau_0 I - \int_{-1}^{0} e^{2i\omega_0\tau_0\theta}d\eta(\theta)\right]\mathbb{E}_1 = F_{z^2}$ and $-\left[\int_{-1}^{0} d\eta(\theta)\right]\mathbb{E}_2 = F_{z\overline{z}}$

The arguments can thus be used to express g_{21} the parameters. Based on the above study, every g_{ij} can be find out by the parameters. As a result, the following quantities can be calculated:

$C_1(0) = \frac{i}{2\omega_0\tau_0}\left(g_{11}g_{20} - 2|g_{11}|^2 - \frac{|g_{02}|^2}{3}\right) + \frac{g_{21}}{2}$, $\mu_2 = -\frac{Re\{C_1(0)\}}{Re\{\lambda'(\tau_0)\}}$, $\beta_2 = 2Re\{C_0(0)\}$,

$T_2 = -\frac{Im\{C_1(0)\} + \mu_2 Im\{\lambda'(\tau_0)\}}{\omega_0\tau_0}$ (2.24)

Theorem 2.1: Hopf-bifurcation's direction is defined by μ_2 , if $\mu_2 > 0$ ($\mu_2 < 0$), the periodic bifurcating solutions exist for $\tau > \tau_0$ ($\tau < \tau_0$) Hopf bifurcation is supercritical (subcritical), and. β_2 is used to determine the bifurcating stability of solutions, are orbitally asymptotically stable (unstable)

if $\beta_2 < 0$ ($\beta_2 > 0$). T_2 defines the period of bifurcating solutions if T_2 is greater than zero (less than zero), the period increases (decreases).

2.6 Numerical Simulation

In this section numerical simulations of model with different values of parameters is carried out Initially S = 10, I = 5, R = 1 ,and $k = 100, \alpha_1 = 0.1, \delta_1 = 0.1, \delta_2 = 0.051, \alpha_3 = 0.1, \alpha_2 = 0.01$ and the equilibrium $S^* = 20.0953, I^* = 8.0289$, $R^* = 15.9686$ and also with reproductive number R_0 . For different values of delay parameter τ, the system of moves from stable to complex dynamics about equilibrium E*, shown in the graphic below.

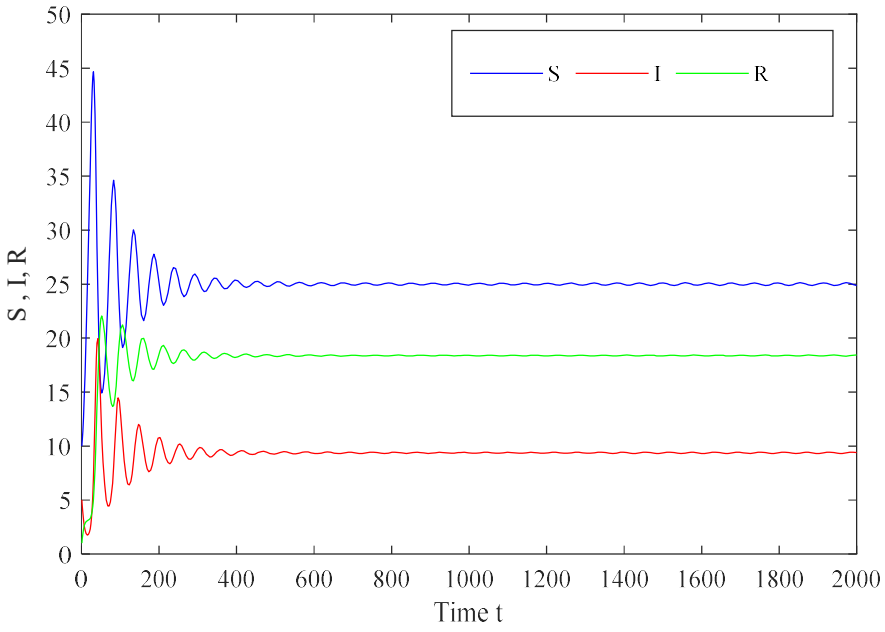

Figure 2.1 : System's time series graph without delay, E* (S*, I*, R*) moves to stabilize after early oscillations.

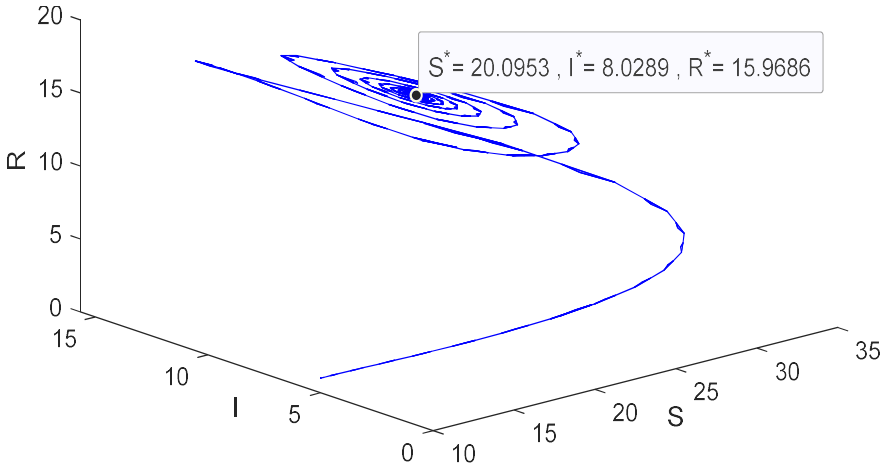

Figure 2.2 : Phase space view of E* (S*, I*, R*) without delay

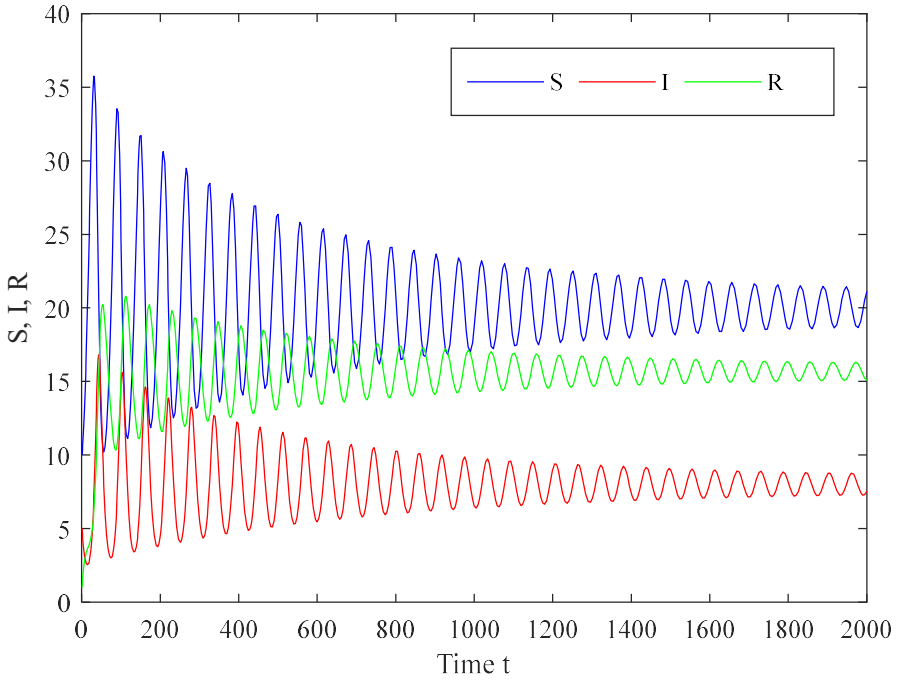

Figure 2.3 : Time series graph of the system when $\tau < 2.241$, the critical value. The equilibrium E* (S*, I*, R*) is asymptotically stable.

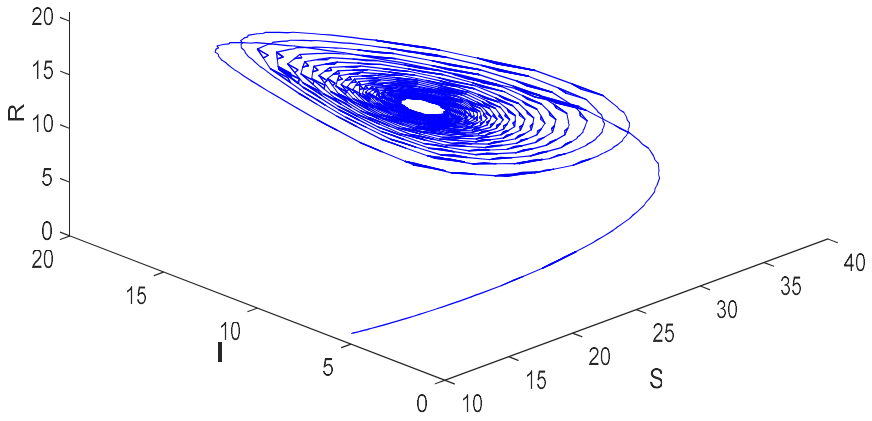

Figure 2.4 : Phase space view of E* (S*, I*, R*) for $\tau < 2.241$

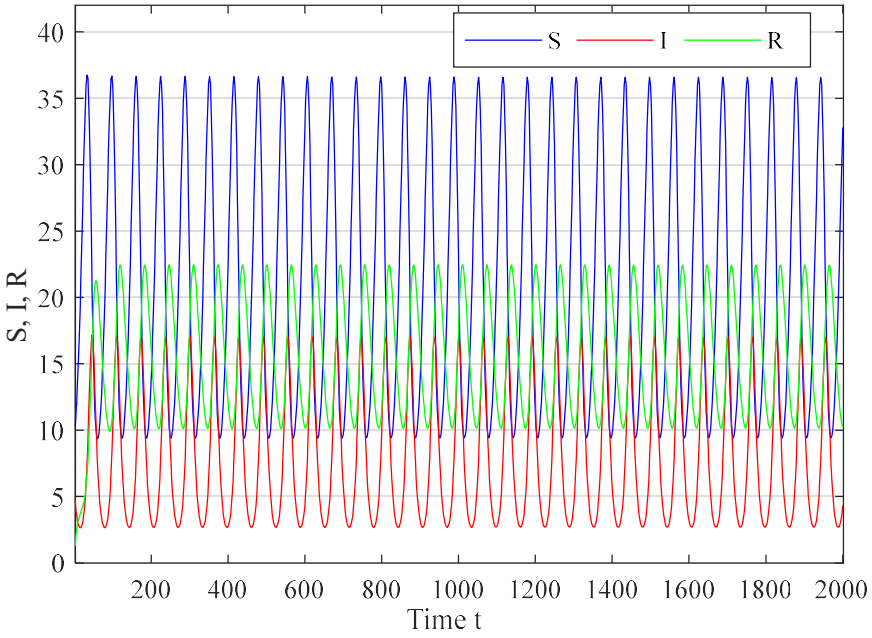

Figure 2.5 : Time series graph shows that equilibrium E* (S*, I*, R*) shows Hopf-bifurcation when $\tau \geq 2.241$

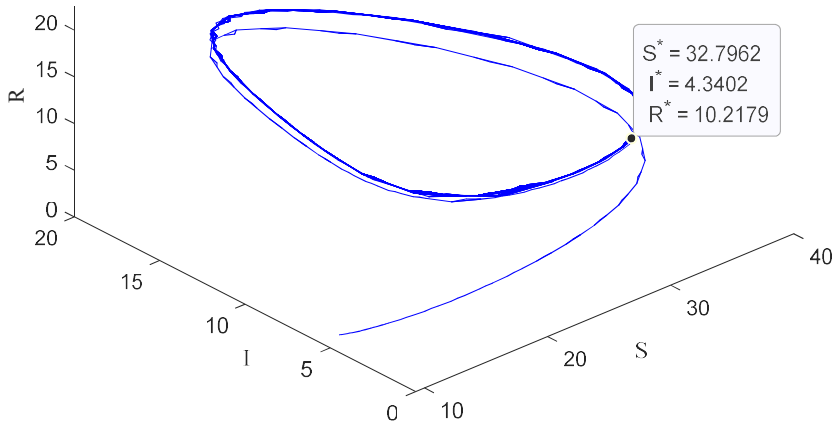

Figure 2.6 : Phase space view of E* (S*, I*, R*) when $\tau \geq 2.241$

2.7 Sensitivity Analysis

2.7.1 With respect to model parameters

In this study, to estimate the general sensitivity “Direct Method" is used. In the direct approach, all parameters are taken into account as constants, and sensitivity coefficients are calculated by resolving sensitivity equations alongside the original system. In this scenario, all of the parameters $(\alpha_1, \alpha_2, \alpha_3)$ appearing in the system model (2.1)-(2.3) are assumed to be constants. If parameter α_2 is taken into consideration, partial derivatives of the solution (S, I, R) with respect to α_2 result in the following set of sensitivity equations:

$$\frac{dx_1}{dt} = (\alpha_1 - \frac{2\alpha_1}{k}S - \beta I(t-\tau))x_1 - \alpha_2 S x_2(t-\tau) \qquad (2.25)$$

$$\frac{dx_2}{dt} = \alpha_2 S x_2(t-\tau) + \alpha_2 I(t-\tau)x_1 - (\alpha_3 + \delta_1)x_2 \qquad (2.26)$$

$$\frac{dx_3}{dt} = \alpha_3 x_2 - \delta_2 x_3 \qquad (2.27)$$

Where $x_1 = \frac{\partial S}{\partial \alpha_2}, x_2 = \frac{\partial I}{\partial \alpha_2}, x_3 = \frac{\partial R}{\partial \alpha_2}$

The system of sensitivity equations (2.25) – (2.27) and equations (2.1) – (2.3) are used to predict the sensitivity of the state variables (S, I, R) for the model parameter 'α_2'. Similar steps and justifications are used to estimate the state variables' sensitivity to the parameters α_1 & α_3 .

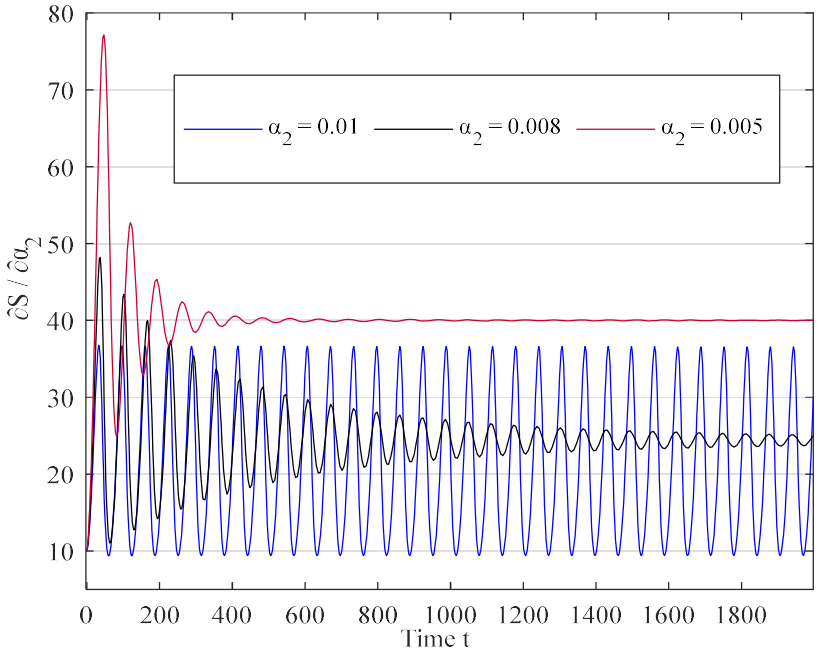

Figure 2.7 : Sensitivity of the susceptible population w.r.t transmission rate α_2 of the disease.

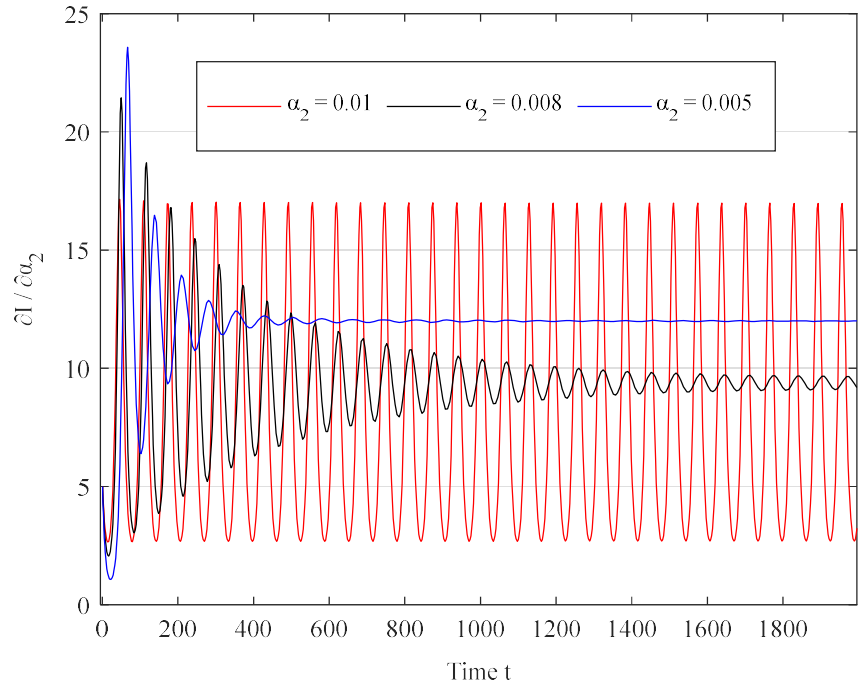

Figure 2.8 : Sensitivity of the infected population w.r.t transmission rate α_2 of the disease

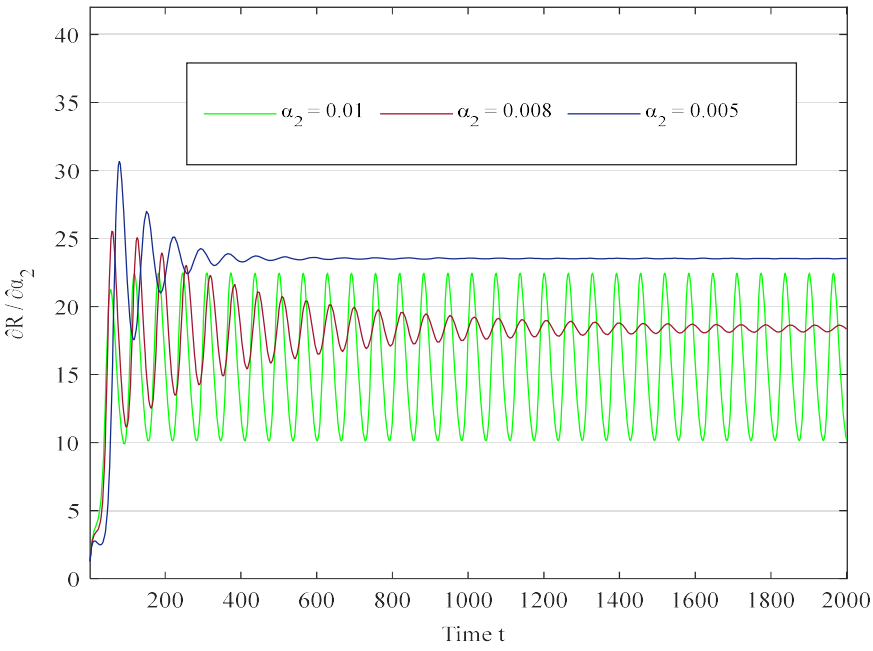

Figure 2.9 : Sensitivity of the recovered population w.r.t transmission rate α_2 of the disease

As the transmission rate α_2 of the disease decreases the system should tend to become stable. The same situation is depicted in figure 2.7, figure 2.8, and figure 2.9. As the value of 'α_2' decreases from 0.01 to 0.008, the system shift from a limit cycle to asymptotic stability, and when it further decreases from 0.008 to 0.005 the system tends to become absolutely stable.

2.7.2 With respect to reproductive number R_0:

The basic reproduction number R_0 is defined as "the expected number of secondary cases directly generated by typical infection in a population where all individuals are susceptible to infection". For the model (2.1) – (2.3) the basic reproductive number is given by $R_0 = \frac{k\alpha_2}{(\alpha_3+\delta_1)}$.

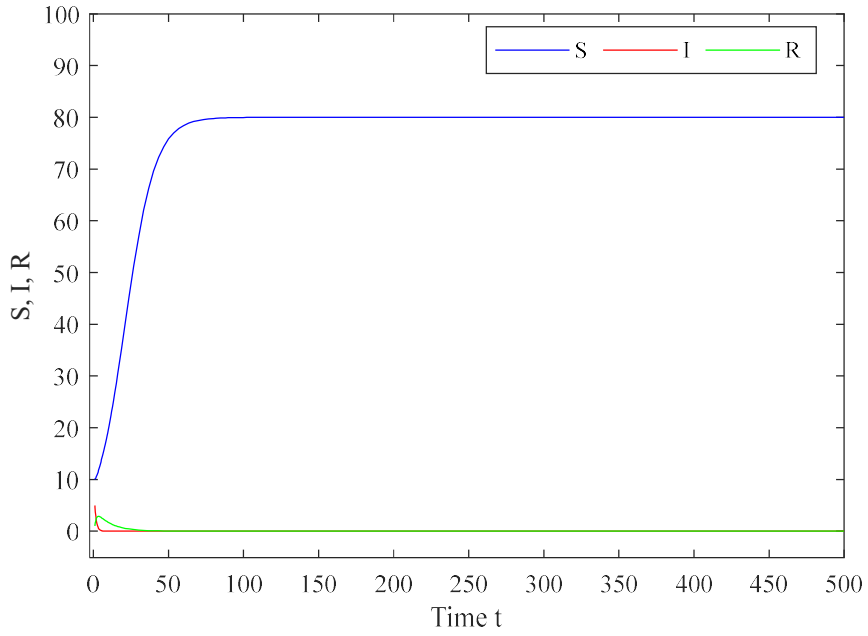

Figure 2.10 : Solution of delayed SIR model (2.1) - (2.3). In the absence of delay system is stable have infection-free equilibrium when reproductive number R_0 <1

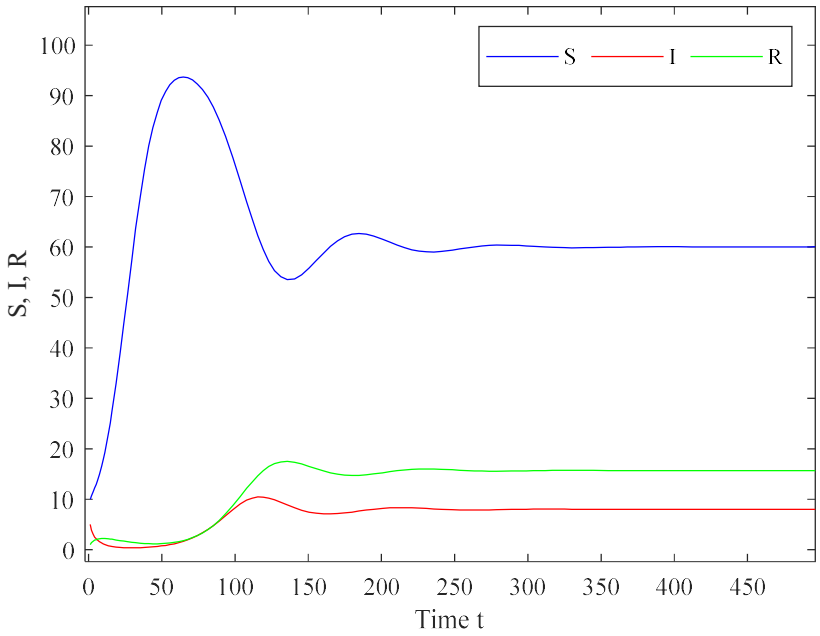

Figure 2.11 : Solution of delayed SIR model (2.1) - (2.3). In the absence of delay, the system has a small portion of endemic equilibrium when reproductive number $R_0 > 1$

2.8 Conclusion

This study examines SIR model stability with a non-linear incidence function. Delay breaks the system, causing limit cycles and sporadic solutions. Figure 2.1 & Figure 2.2 show equilibrium E* stabilising after early fluctuations without a delay. An infected person can be adequately treated and the system will stabilize if a critical delay τ is shorter than that value. Conjecture 1, $(\mathbb{H}_1) - (\mathbb{H}_2)$ shows the same truth. Figure 2.3 & Figure 2.4 show that as the delay τ constant varies below 2.241, the equilibrium E* loses stability and becomes asymptotical. The equilibrium E* undergoes a Hopf-bifurcation after reaching to the value 2.241.

The system involving all three states will always repeat the epidemic if the delay crosses the critical threshold. Figure 2.5 & Figure 2.6 show stable periodic solutions with big amplitudes and limit cycle trajectories. The system's (2.1) – (2.3) complex behaviour matches conjecture-2 $(\mathbb{H}_4) - (\mathbb{H}_5)$ The direction analysis of the model has been performed which reveals the

direction, amplitude, and periodicity of the bifurcating solutions as per the criteria given by Hassard (Schneider, 1982).

Not only delay but other model parameters also affect the system. Thus, the sensitivity with model parameters and basic reproduction number R_0 are tested. Disease transmission rate α_2 affects the system greatly. As transmission rate decreases, the system changes from Hopf bifurcation to asymptotic stability to absolute stability, as shown in Figure 2.7, Figure 2.8 & Figure 2.9 . The model's dynamics and basic reproductive number R_0 are investigated. We studied the threshold parameter R_0 factors that ensured the endemic and infection-free equilibria's asymptotic stability, $R_0 > 1$ makes the disease endemic and spreads in the neighbourhood, while $R_0 < 1$ eliminates it. Figure 2.10 & Figure 2.11 depict the same scene. The simple DDE model has chaotic and quasiperiodic dynamics and is suitable for high population numbers.

Chapter 3

Bifurcation and Stability Analysis of Delayed SIR Model

3.1 Introduction

Infections and diseases have always been a great threat to human kind and animals. The spread of diseases from one person to another via blood or physical contact can be termed as communicable diseases. Infections and diseases can also spread by breathing in an airborne virus or bitten by a virus carrier. These infections and diseases have a great potential to become endemic and cause suffering and social economic loss. Millions of people lose their lives every year due to these diseases, especially in developing countries. Mathematical modelling a great tool to study these kinds of diseases from various perspective, transmission rates, estimate the potential losses and formulate the strategies to overcome these. Many models have been formulated for study of transmission rates of different kinds of endemics (Anderson & May, 1979b; Beretta & Capasso, 1987; Diekmann & Heesterbeek, 2000; Hethcote & Tudor, 1980b; Huo & Ma, 2010; Koff, 1992; May & Anderson, 1979; McCluskey, 2010b; Xiao & Ruan, 2007b) . Three basic epidemiological terms are always used in these infections spread models: the susceptible population $S(t)$, the infected portion $I(t)$ and the removed or recovered one $R(t)$. A simple SIR model was proposed to calculate theoretically, the number of infected people with a contagious sickness ("A Contribution to the Mathematical Theory of Epidemics," 1927) . The modelling of dynamic transmission process between infected and susceptible determines the behaviour of simple SIR models. SIR models accurately portray infectious diseases caused by viral agents such as measles, mumps, and smallpox. A significant number of researchers have simulated and investigated infectious diseases like these (A. Rihan et al., 2012; Berezovsky et al., 2005b; Cai et al., 2009b; d'Onofrio et al., 2007; D'Onofrio et al., 2007; Esteva & Matias, 2001; Hsu & Zee, 2004). There are a number of diseases, like influenza and TB, that have a period of time during which

they remain dormant or incubating. During the time that they are in the incubation stage, it is claimed that they are infected but that they are not infectious. This incubation time may be accurately represented by a delay parameter in SIR models by making use of delay differential equations, (Beretta & Kuang, 2001) conducted research on the topic of the global stability of the SIR epidemic model with distributed delays. In addition, the requirements for asymptotic stability of endemic equilibrium have been established (X. Song & Cheng, 2005). These constraints apply to all feasible delays.

In most of the mathematical models, disease free and endemic equilibrium are analysed for global asymptotic stability. But many of these models are stiff and numerical analysis needs to be performed carefully. Small perturbations in state variables and parameters can result in changes in overall behaviour of the system. A saturated incidence rate has been applied in detailed qualitative analysis of delayed SIR model (Rihan & Anwar, 2012). In the setting of a predator-prey system with discrete delays, a precise analysis was presented for a number of distinct kinds of stability, including absolute, conditional and bifurcation. This study was done for a number of distinct types of stabilities (Ruan, 2001). (Ruan & Wei, 2003a) performed an in-depth analysis about the nature of the roots that make up the exponential latent equation. It is carried out an investigation of the stability of steady state points utilising a non-linear DDE (Ruan, 2009). It has been shown that the solutions of the SIR model are bounded, and an analysis of the asymptotic stability of the model with a limited incubation time has also been conducted (Huang et al., 2010; Takeuchi et al., 2000). (Elazzouzi et al., 2019) conducted research to investigate the stability analysis of a generalised SIR epidemic model that included a nonlinear incidence rate and dispersed delays. The two control measures that were taken into consideration in this model were the vaccination term and the general treatment functions.

Due to the information presented above, the following mathematical model is used in this article to investigate the effect that delay in the traditional SIR

model. In addition to this, stability analysis is carried out about the non-zero endemic steady state point.

3.2 Mathematical Model

Let the incubation period be denoted by $\tau > 0$. During incubation period, the infected turn into a vector and only after this period, the infected becomes infectious. Let three state variables be: the susceptible population $S(t)$, the infected portion $I(t)$ and the removed or recovered one $R(t)$. The model formulated is:

$$\frac{dS}{dt} = \alpha S - \beta SI(t-\tau) \tag{3.1}$$

$$\frac{dI}{dt} = \beta SI(t-\tau) - \gamma I \tag{3.2}$$

$$\frac{dR}{dt} = \gamma I - \delta_2 R \tag{3.3}$$

Such that $S + I + R = N$ (constant)

Where: $S(0) > 0, I(0) > 0, R(0) > 0$ for all t and $I(t-\tau) =$ constant for $t \in [0, \tau]$. The parameters considered in this model are;

Table 3.1 : Description of the parameters of the system (3.1)-(3.3)

Parameter	Description
α	Intrinsic birth rate
β	Infection rate per contact between susceptible and infected hosts
γ	Recovery rate of infected hosts
τ	Delay parameter or Incubation period
δ_2	Death rate of recovered hosts

It is justified to assume all the parameters as positive constants.

The dynamics of the system is mainly determined by the set of following two equations (3.1) and (3.2). So, the focus will be on first two equations (3.1) and (3.2) and equation (3.3)

can be ignored for analysis of the model.

3.3 Non-Zero Epidemic Equilibrium

It is assumed that at equilibrium point $I(t-\tau) \cong I$. The non-zero epidemic equilibrium $E^*(S^*, I^*, R^*)$ is calculated as:

$$\frac{dI^*}{dt} = 0 \Rightarrow S^* = \frac{\gamma}{\beta}$$

$$\frac{dS^*}{dt} = 0 \Rightarrow I^* = \frac{\alpha}{\beta}$$

$$\frac{dR^*}{dt} = 0 \Rightarrow R^* = N - \frac{\gamma}{\beta} - \frac{\alpha}{\beta}$$

Thus, we have non-zero equilibrium:

$$E^*(S^*, I^*, R^*) = E^*\left(\frac{\gamma}{\beta}, \frac{\alpha}{\beta}, N - \frac{\gamma}{\beta} - \frac{\alpha}{\beta}\right)$$

3.4 Stability Analysis about Equilibrium E^* and Hopf- Bifurcation

At the equilibrium $E^*(S^*, I^*, R^*)$, the system of equations (3.1)-(3.3)

becomes:

$$\frac{dS^*}{dt} = \alpha S^* - \beta S^* I^*(t-\tau) \quad (3.4)$$

$$\frac{dI^*}{dt} = \beta S^* I^*(t-\tau) - \gamma I^* \quad (3.5)$$

$$\frac{dR^*}{dt} = \gamma I^* - \delta_2 R^* \quad (3.6)$$

The boundedness of all feasible solutions of system (3.1)-(3.3)

and that of system (3.4)-(3.5) has been proved by (Takeuchi et al., 2000). The fluctuation of the system is mainly determined by the set of two equations (3.4) and (3.5). The characteristic equation of equations (3.4 and (3.5) about the equilibrium $E^*$is given by:

$$\lambda^2 + a\lambda + \mathscr{b} + (c\lambda + d)e^{-\lambda\tau} = 0 \quad (3.7)$$

Where $a = (\gamma - \alpha), \mathscr{b} = -\alpha\gamma, c = -\beta S^*, d = \alpha\beta$

When $\tau = 0$, the equation (3.7) becomes:

$$\lambda^2 + (a + c)\lambda + (\mathscr{b} + d) = 0 \quad (3.8)$$

By Routh-Hurwitz's criteria, roots of equation (3.8) will have non positive non zero real part i.e., the stability of the system arises if ;

$(\mathbb{H}_1)$: $(a + c) > 0$;

$(\mathbb{H}_2)$: $(\mathscr{b} + d) > 0$

which obviously is true. Now that this is done, we will check to see if the negative real sections of the roots have been replaced by positive real ones with the change in the value of τ.

Let $\lambda = i\omega$ be a solution of characteristics of equation (3.7), so we have;

$$-\omega^2 + a(i\omega) + \mathscr{b} + (c(i\omega) + d)(\cos\omega\tau - isin\,\omega\tau) = 0$$

Separating real and imaginary parts:

$$-\omega^2 + \mathscr{b} = -d\cos\omega\tau - c\omega sin\,\omega\tau \quad (3.9)$$

$$a\omega = -c\cos\omega\tau + dsin\,\omega\tau \quad (3.10)$$

Squaring and adding:

$$\omega^4 - (c^2 - a^2 + 2\mathscr{b})\omega^2 + (\mathscr{b}^2 - d^2) = 0 \quad (3.11)$$

The two roots of equation (3.11) are:

$$\omega_{1,2}^2 = \frac{(2\mathscr{b} - a^2 + c^2) \pm \sqrt{(a^2 - c^2 + 2\mathscr{b})^2 - 4(\mathscr{b}^2 - d^2)}}{2} \quad (3.12)$$

None of the two roots $\omega_{1,2}^2$ is positive if:

$(\mathbb{H}_3)$: $(2\mathscr{b} - a^2 - c^2) < 0\ and\ (\mathscr{b}^2 - d^2) > 0\ or\ (a^2 - c^2 + 2\mathscr{b}) < 4(\mathscr{b}^2 - d^2)$

This indicate that equation (3.12) does not have a solution which is positive if condition $(\mathbb{H}_3)$ holds. We have the following lemma (Ruan, 2001).

Lemma 3.1. If $(\mathbb{H}_1) - (\mathbb{H}_2)$ hold, then all the roots of equation (3.7) have negative real parts for all $\tau \geq 0$.

On the other hand, if:

$(\mathbb{H}_4)$: $(\mathscr{b}^2 - d^2) < 0 \; or \; (2\mathscr{b} - a^2 - c^2) > 0 \; and \; (a^2 - c^2 + 2\mathscr{b})^2 = 4(\mathscr{b}^2 - d^2)$

Then, +ve root of equation (3.7) is ω_1^2.

On the same basis, if:

$(\mathbb{H}_5)$: $(\mathscr{b}^2 - d^2) > 0 \; or \; (2\mathscr{b} - a^2 - c^2) > 0 \; and \; (a^2 - c^2 + 2\mathscr{b})^2 > 4(\mathscr{b}^2 - d^2)$

Then, two +ve roots of equation (3.12) are $\omega_{1,2}^2$.

For given particular values, the equation (3.7) has entirely complex roots when τ takes certain values expressed in both $(\mathbb{H}_4)$ and $(\mathbb{H}_5)$. The critical values $\tau_j^{\pm}$ of τ comes from the setup of the equations (3.9)-(3.10) , given by;

$$\tau_j^{\pm} = \frac{1}{\omega_{1,2}} cos^{-1}\left[\frac{d(\omega_{1,2}^2 - \mathscr{b}) - ac\omega_{1,2}^2}{c^2\omega_{1,2}^2 + d^2}\right] + \frac{2j\pi}{\omega_{1,2}}, j = 0,1,2, \ldots \qquad (3.13)$$

The preceding discussion may be summarised in the lemma (Ruan, 2001) that is about to be presented.

Lemma 3.2. (I) If $(\mathbb{H}_1) - (\mathbb{H}_2) \; and \; (\mathbb{H}_4)$ hold and $\tau = \tau_j^+$, then equation (3.7) has a pair of purely imaginary roots $\pm i\omega_1$.

(II) If $(\mathbb{H}_1) - (\mathbb{H}_2) \; and \; (\mathbb{H}_5)$ hold and $\tau = \tau_j^+ \big(\tau = \tau_j^- \text{ respectively}\big)$, then equation (3.7) has a pair of complex roots with real part zero $\pm i\omega_1 (\pm i\omega_2 \; respectively)$.

Our expectation is the that the formerly negative real parts of certain roots of the equation (3.7) will change sign and become positive real. when $\tau > \tau_j^+$ and $\tau < \tau_j^-$. In order to investigate this option, let us label it as,

$\tau_j^{\pm} = \mu_j^{\pm}(\tau) + i\omega_j^{\pm}(\tau); j = 0,1,2,3, \ldots$

The roots of equation (3.7) satisfy: $\mu_j^{\pm}(\tau_j^{\pm}) = 0, \omega_j^{\pm}(\tau_j^{\pm}) = \omega_{1,2}$

We can verify that the following transversality condition holds:

$\frac{d}{d\tau}\left(Re\ \lambda_j^+(\tau_j^+)\right) > 0\ and\ \frac{d}{d\tau}\left(Re\ \lambda_j^-(\tau_j^-)\right) < 0$

It concludes that $\tau_j^{\pm}$ are bifurcating values.

3.5 Numerical Simulation

In order to visually portray the dynamics that are shown by the system of equations, the following set of parametric values has been adopted (3.1)−(3.3)

$\alpha = 0.1, \beta = 0.01, \gamma = 0.1$, As the value of the delay parameter τ is varied, The fluctuating patterns of behaviour seen by the equation system (3.1)- (3.3)

from being stable to exhibiting complicated dynamics revolving around the equilibrium $E^*(S^*, I^*, R^*)$, is shown below:

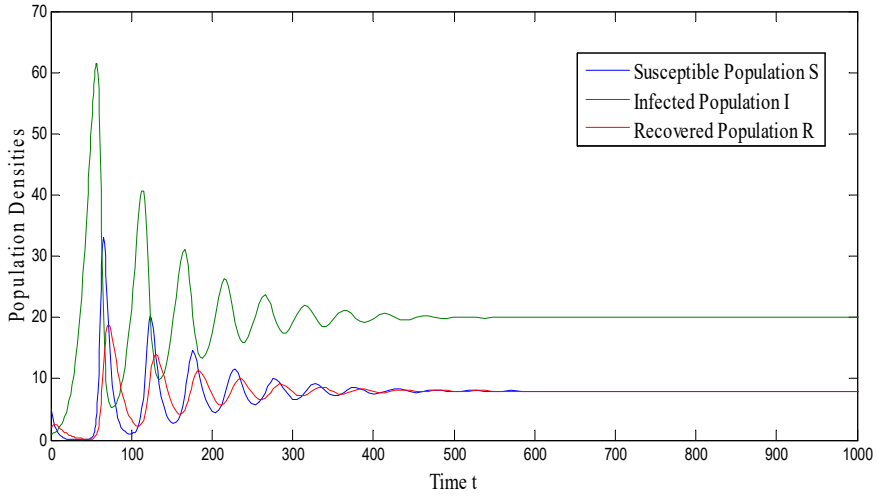

Figure 3.1 : The Equilibrium $E^*(S^*, I^*, R^*)$ tends to be stable after initial fluctuations in the presence of negligible delay i.e., when $\tau \approx 0$

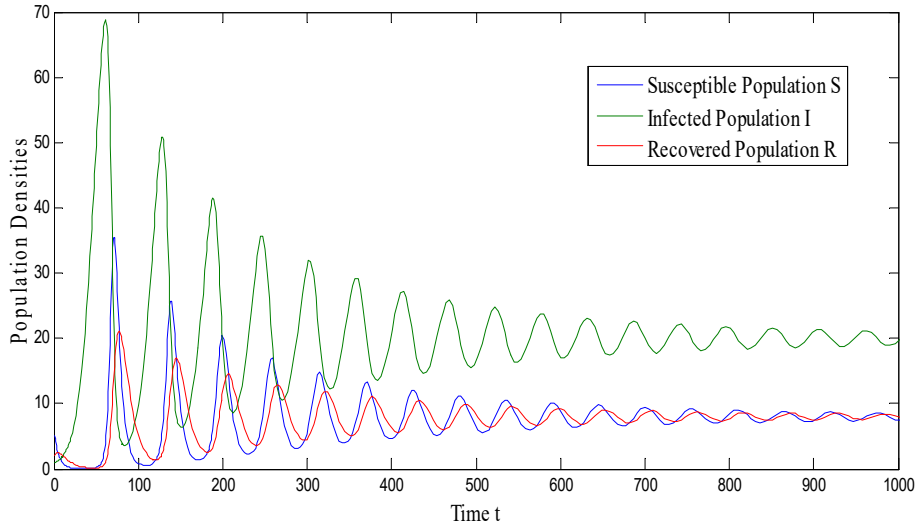

Figure 3.2 : The Equilibrium $E^*(S^*, I^*, R^*)$ is asymptotically stable when delay is less than critical vale i.e., when $\tau < 8.5$

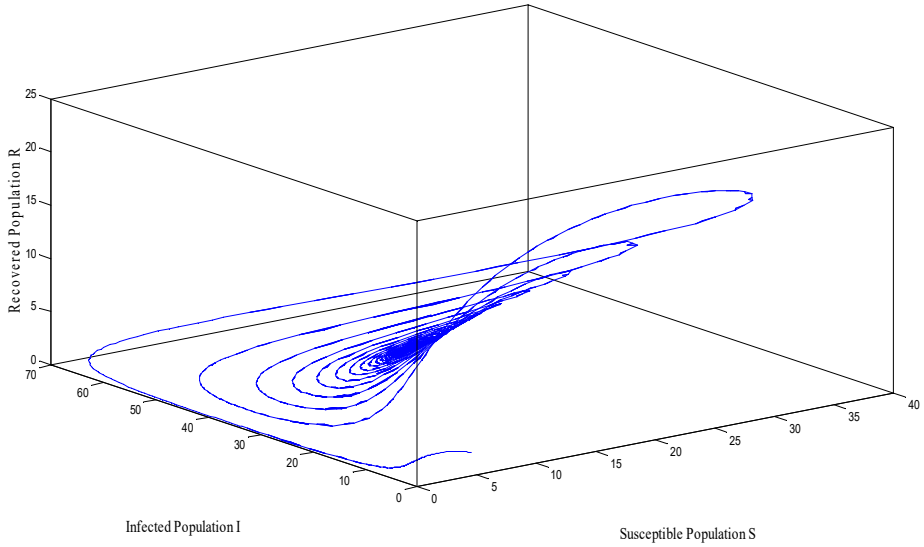

Figure 3.3 : Phase space of equilibrium $E^*(S^*, I^*, R^*)$ when the delay is less than critical value i.e., $\tau < 8.5$

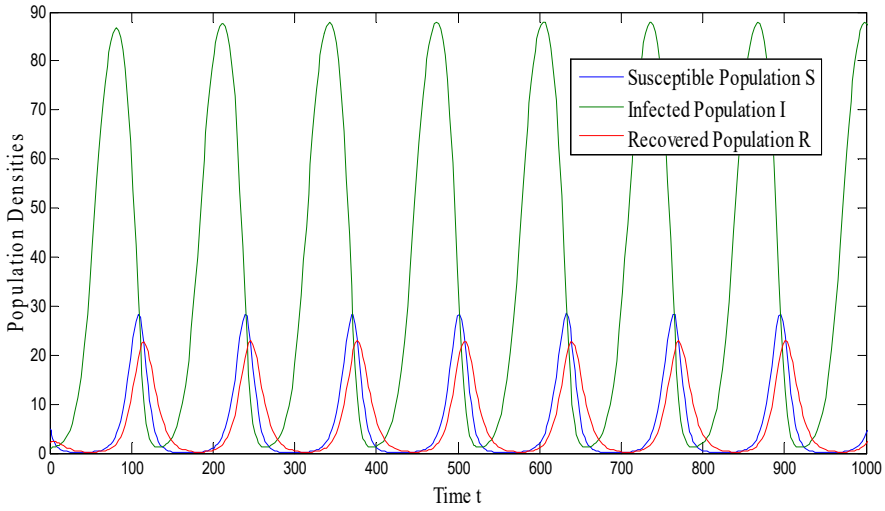

Figure 3.4 : The Equilibrium $E^*(S^*, I^*, R^*)$ moves from stability to Hopf-bifurcation when delay is crosses the critical vale i.e., when $\tau \geq 8.5$

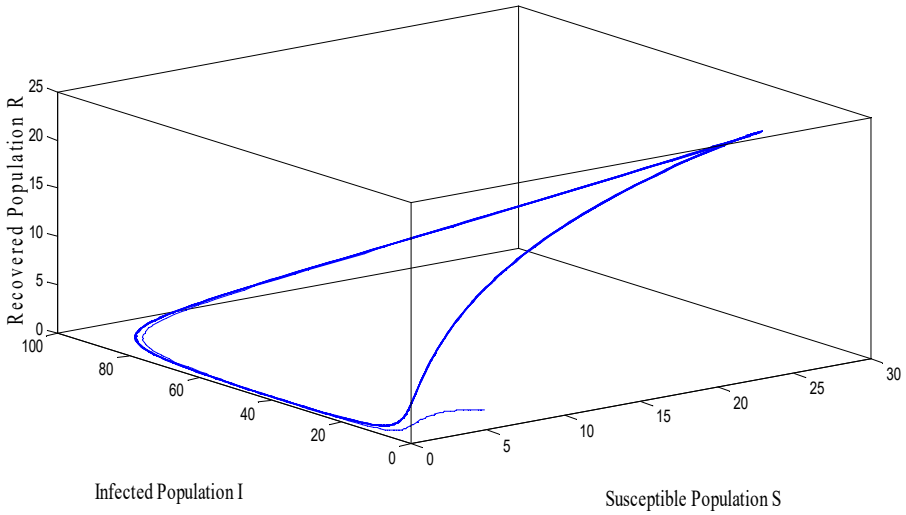

Figure 3.5 : Phase space of equilibrium $E^*(S^*, I^*, R^*)$ when the delay crosses the critical value i.e., $\tau \geq 8.5$

3.6 Conclusion

With the assistance of the suggested model, an analysis of the traditional SIR model may be carried out using DDE. The delay causes the system to become unstable and sets off a complicated pattern of behaviour that contain limit cycles as well as periodic solution that are stable over Hopf-bifurcation. An investigation of the equilibrium's degree of stability, E*, s carried out. As can be seen in Figure 3.1, if there is no delay involved, the equilibrium E* will typically return to its original state after experiencing some early oscillations. It indicates that if the delay in the identification of contaminated section is below a certain value, the suspected and infected may be recovered well, and the system will become stable. In addition, the fact that $(\mathbb{H}_1) - (\mathbb{H}_2)$ exists, as in lemma 3.1, may be used to augment the same fact. As illustrated in Figure 3.2 and Figure 3.3, the asymptotic stability occurs when the delay τ falls below the crucial point, which in this case is $\tau < 8.5$. This causes the equilibrium E* to begin to lose its stability and ultimately results in the asymptotic stability. When the delay parameter τ reaches the critical value,

which is denoted by the number $\tau \geq 8.5$, the equilibrium E* displays the complicated dynamics in the form of a Hopf bifurcation. This indicates that if the delay exceeds the critical value in the identification of infected individuals, the system that involves all three states and is involved in the epidemic will constantly remain in the vicious loop of repeating the epidemic after each cycle of delay length. Figure 3.4 and Figure 3.5 provide examples of stable periodic solutions with high amplitudes and limit cycle trajectories, respectively. This observation of the system's complicated behaviour, as indicated by (3.1)-(3.3)

Chapter 4

Mathematical Analysis of SIR Model with Virus Mutation Using Delay Differential Equation

4.1 Introduction

In today's world, diseases are the most serious menace. Globalization, urbanization, and a slew of other variables are making life easier for us, but they are also key causes of sickness. These infections then spread like wildfire, resulting in widespread death. As a result, infectious disease modelling is critical in order to control outbreaks and prevent Endemics. It is a tool that is used to investigate the mechanisms by which solutions to manage the Endemic might be devised. An Endemic model is a simple way of describing how infectious diseases spread through a population. Mutation is a change in an organism's genes that results in differences that are handed down through generations through reproduction. The spread of the Endemic is heavily influenced by mutation. The immune system has a very specialized way of recognizing foreign substances. Immune cells or antibodies that detect one pathogen strain's proteins may not recognize proteins from another pathogen strain. As a result, virus mutations may enhance the virus's infection rate, rendering the vaccine ineffective. As a result, studying the transmission properties of altered viruses via mutation in the population is a worthwhile endeavour.

Mathematical modelling of infectious diseases was initiated by Bernoulli Daniel (Bernoulli Daniel, 1760),(Gabriel & de la Harpe, 2010) . Since the work of Kermack and McKendrick, (Hernandez-Ceron et al., 2013),(Bacaër & Bacaër, 2011b),(Bacaër & Bacaër, 2011a) pioneered, compartment mathematical modelling has been a powerful tool for assessing infectious disease transmission and management. Just now, there has been a lot of focus on constructing realistic mathematical models for infectious disease transmission dynamics. Their SIR model is still used to model Endemics of infectious diseases. A deterministic SIR model is proposed, in which an

infected person's recovery is only dependent on how long they have been unwell, and recovered people are permanently immune to infection (Wang, 1979). In a complete investigation of a generic class of SIRS Endemic models (Stech & Williams, 1981), the appropriate conditions that ensure that the endemic equilibrium solution are determined are globally stabile. Delays have a crucial role in population dynamics. The current dynamics of state variables, particularly in medical phenomena, are reliant not only on the current phase of the proceedings, but upon the past experience of the phenomenon also, i.e., on the previous state variables' values. The temporal time lag may have an impact on the dynamics of infectious diseases. Many diseases, such as immunity period time lag (Q. Liu & Jiang, 2016),(Hao et al., 2013), infection period time lag (P. Yan & Feng, 2010), and incubation period time lag (Ashyani et al., 2016) ,(Safi & Gumel, 2011), (Naresh et al., 2011) ,(J. Liu et al., 2016) , have distinct types of delays when they spread. Considerable study has been conducted on the dynamical behaviours of the system with incubation time. Especially interesting are the features of recurring solutions originating from the Hopf-bifurcation. A variety of time-delayed Endemic models has been constructed for the study to obtain observation into the influence of time lag on the model's dynamic behaviour. The properties of solutions that are found repeatedly because of Hopf-bifurcation are particularly intriguing. Diverse time-delayed Endemic models have been built to examine the effect of time lag on the model's dynamic behaviour. For age-structured SEIR endemic models (Li et al., 2001) investigate whether or not there exists a positive solution and whether or not the steady state is stable. (Röst & Wu, 2008) explored the global stability, how infection age impacts infectivity in an SEIR model with a time lag. The study and control of infectious disease propagation and transmission was given by (Gao et al., 2008) with two-time lags. (Kalra & Kumar, 2020) stability and sensitivity of model is performed using delay parameter. In a nonlinear SEIR model, (Tipsri & Chinviriyasit, 2015) looked at the influence of time lag on the stability and the direction of Hopf-bifurcation of recurring solutions

A mutation-based SIR Endemic model can capture these complex interactions between the virus and the host, allowing us to use analysis to explain more complex events. The evolutionary processes of mutations are taken into consideration in an Endemic model (Gubar et al., 2018). The amount of viral transformation to a susceptible chamber and the number of beneficial mutations by time t were both determined. To do this, a simple compartmental SIR model was created with a single parameter reflecting vaccination-induced transmission decrease. The model was then changed to include vaccination rate and short duration of immunity. It has been proven that a mutating disease can cause fluctuations in the number of affected people in a community (Girvan et al., 2002). A stochastic SIR model is intended to better depict the characteristics of a disease in practise, where minor influences can induce unexpected and sudden changes in transmission behaviour (Hale & Lunel, 1993). (Schneider, 1982) When the model's parameters aren't considered as constants, adjoint equations and direct method are used to estimate the sensitivity functions. Two different layouts of infected and recovered population are reported by(Park & Bolker, 2020) and the effect of reproduction number on the shapes of infected and recovered population is studied. (Kreck & Scholz, 2022) use the basic SIR model given by Kermack-Mc Kendrick in 1927 for the analysis of Covid-19 and seems to be a black box for the improvement. (Rihan, 2003b) described adjoint and direct sensitivity analysis methods for numerical modelling employing delay differential equations.

Keeping the above scenario in consideration, a mathematical model is proposed in this chapter, which studies the stability and existence of bifurcation in SIR model under the mutation factor, using delay differential equations.

4.2 Mathematical model

An infected person does not turn into infectious immediately but need a maturation time to become an infection vector. That maturation time is the incubation period and denoted by $\tau > 0$. We suppose $S(t)$, $I(t)$, $R(t)$, are

respectively the susceptible population, infected population, and the removed population. The model depicting these dynamics is given as follows:

$$\frac{dS}{dt} = aS(t) - bS(t)I(t) + cR(t-\tau) \quad (4.1)$$

$$\frac{dI}{dt} = bS(t)I(t) - dI(t) \quad (4.2)$$

$$\frac{dR}{dt} = dI(t) - (\mathfrak{D} + c)R(t-\tau) \quad (4.3)$$

Here: $S(t)$, $I(t)$, $R(t)$ are all positive for all t and $R(t-\tau) = k$ (constant) for $t \in [0, \tau]$.

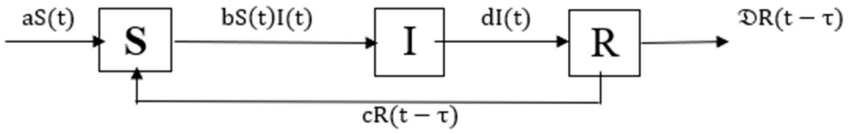

Figure 4.1: Model's flowchart

Table 4.1 : Description of the parameters of the system (4.1) - (4.3)

Parameter	Description	Units
a	Susceptible Individuals' growth rate	$(day)^{-1}$
b	Rate of transmission	$(day)^{-1}$
c	Rate at which the recovered ones become susceptible (mutation rate)	$(day)^{-1}$
d	Recovery rate	$(day)^{-1}$
$\mathfrak{D}$	Rate of permanent immunity	$(day)^{-1}$
$(\mathfrak{D} + c)$	Rate of total recovery	$(day)^{-1}$
τ	Time lag	$(day)^{-1}$

All of the parameters can be assumed to be positive constants.

4.3 Endemic equilibrium $\mathbf{E^*(S^* \neq 0, I^* \neq 0, R^* \neq 0)}$

At equilibrium point $R(t-\tau) \cong R$. Endemic equilibrium $E^*(S^*, I^*, R^*)$ is calculated as:

$\frac{dI^*}{dt} = 0 \Rightarrow S^*(t) = \frac{d}{b}$

$\frac{dR^*}{dt} = 0 \Rightarrow I^*(t) = \frac{a(\mathfrak{D}+c)}{\mathfrak{D}b}$

$\frac{dS^*}{dt} = 0 \Rightarrow R^*(t) = \frac{ad}{\mathfrak{D}b}$

Thus, $E^*(S^*, I^*, R^*) = E^*\left(\frac{d}{b}, \frac{a(\mathfrak{D}+c)}{\mathfrak{D}b}, \frac{ad}{\mathfrak{D}b}\right)$

4.4 Boundedness of solutions

Following lemma determines model solution's boundedness.

Lemma 4.1. The model's entire solution is contained within the region $D = \left\{(S(t), I(t), R(t)) \in {R_+}^3 : \frac{(\mathfrak{D}+c+d)}{\varphi_m} \leq S(t) + I(t) + R(t) \leq \frac{(a+1+d)}{\varphi}\right\}, as\ t \to \infty$, for all positive initial values $\{S(0), I(0), R(0), N(t-\tau) = K(\text{Constant})$ for all $t \in [0, \tau]\} \in D \subset {R_+}^3$, where $\varphi = \min(1, d, \mathfrak{D})$, and $\varphi_m = \max\{a, d, c\}$.

Proof: Consider the following function: $W(t) = S(t) + I(t) + R(t)$,

$\frac{dw(t)}{dt} = \frac{d}{dt}(S(t) + I(t) + R(t))$ Using Equations (1) - (3) and $\varphi = \min(1, d, \mathfrak{D})$. and assuming that $R(t-\tau) \approx R(t)$ as $t \to \infty$, $\frac{dW(t)}{dt} \leq \big((a+1+d)\big) - \varphi W(t)$. Applying the comparison theorem, as $t \to \infty$: $W(t) \leq \frac{(a+1+d)}{\varphi}$. Also again $\frac{dW(t)}{dt} \leq \varphi_m W(t) - (\mathfrak{D} + c + d)$, where $\varphi_m = \max\{a, d, c\}$ by Applying the comparison theorem, as $t \to \infty$: $W(t) \geq \frac{(\mathfrak{D}+c+d)}{\varphi_m}$.

Therefore, we have $\frac{(\mathfrak{D}+c+d)}{\varphi_m} \leq S(t) + I(t) + R(t) \leq \frac{(1+a+d)}{\varphi}$

S(t), I(t), and R(t) are thus uniformly confined at the end. The proof is finished.

4.5 Positivity of solutions

The term "positivity" refers to the stability of a system. For positive solutions, one must show that Equations (1) – (3) provide all of the system's solutions, where initial conditions are $S(0) > 0, I(0) > 0, R(0) > 0, \forall t$ and $R(t-\tau) = k$, for $t \in [0, \tau]$, Models solutions $(S(t), I(t), R(t))$ remain +ve for all time $t > 0$.

By equation (4.2): $\frac{dI(t)}{dt} = b.S(t).I(t) - d.I(t)$ i.e $\frac{dI(t)}{dt} \geq -d\, I(t)$ i. e $\frac{dI(t)}{I} \geq -d\ dt$, $I(t) \geq k_0 e^{-dt}$, here k_0 is the integration constant. As a result, $I(t) > 0$ for all the time t. S and R have a similar argument.

4.6 Hopf-bifurcation and Stability Analysis

In the presence of endemic equilibrium $E^*(S^*, I^*, R^*)$, the equation (4.1)–(4.3) implies,

$$\frac{dS^*}{dt} = aS^*(t) - bS^*(t)I^*(t) + cR^*(t-\tau) \qquad (4.4)$$

$$\frac{dI^*}{dt} = bS^*(t)I^*(t) - dI^*(t) \qquad (4.5)$$

$$\frac{dR^*}{dt} = dI^*(t) - (\mathfrak{D} + c)R^*(t-\tau) \qquad (4.6)$$

The characteristic equation of system (4.4)-(4.6) about the endemic equilibrium $E^*$is given by:

$$\begin{vmatrix} \lambda - m_1 & -m_2 & -m_3 \\ -m_4 & \lambda - m_5 & -m_6 \\ -m_7 & -m_8 & \lambda - m_9 \end{vmatrix} = 0 \qquad (4.7)$$

Where $m_1 = a - bI^*$, $m_2 = bI^*$, $m_3 = 0$, $m_4 = bS^*$, $m_5 = bS^* - d$, $m_6 = d$, $m_7 = ce^{-\lambda\tau}$, $m_8 = 0$, $m_9 = -(\mathfrak{D} + c)e^{-\lambda\tau}$

The equation (4.7) after expanding become:

$$\lambda^3 + P_1\lambda^2 + P_2\lambda + (P_3\lambda^2 + P_4\lambda + P_5)e^{-\lambda\tau} = 0 \qquad (4.8)$$

Where $P_1 = d + b(I^* - S^*) - a$, $P_2 = a(bS^* - d) + bdI^*$, $P_3 = (\mathfrak{D} + c)$, $P_4 = (\mathfrak{D} + c)(d + b(I^* - S^*) - a)$, $P_5 = (\mathfrak{D} + c)(bdI^* + a(bS^* - d) - bcdI^*)$, Where P_1, P_2, P_3, P_4, P_5 are all positive .

When $\tau = 0$, the equation (4.8) becomes:

$$\lambda^3 + P_1\lambda^2 + P_2\lambda + (P_3\lambda^2 + P_4\lambda + P_5) = 0$$

$$\lambda^3 + (P_1 + P_3)\lambda^2 + (P_2 + P_4)\lambda + P_5 = 0 \qquad (4.9)$$

By Routh-Hurwitz's criteria implies that when $\tau = 0$, $E^*(S^*, I^*, R^*)$, the endemic equilibrium is locally asymptotically stable equilibrium, if

$(\mathcal{H}_1)$: $P_5 > 0$, $(P_1 + P_3) > 0, (P_2 + P_4) > 0$, $(P_1 + P_3)(P_2 + P_4) > P_5$

Hold, the incubation period parameter can influence the stability of $E^*(S^*, I^*, R^*)$, and Hopf-bifurcation may appear whenever the time lag parameter going beyond a critical point value, as demonstrated in the following.

Lemma - 4.2 Supposed that the condition $\mathcal{H}_1$are satisfied, the equation (4.8) with $\tau = \tau_j (j = 0,1\ldots)$ has a simple pair of conjugate purely complex roots $\pm i\omega_0$, were

$$\tau_j = \frac{1}{\omega_0}\left\{\arccos\frac{\omega_0[P_1\omega_0(P_5 - P_3{\omega_0}^2) + P_4({\omega_0}^3 - P_2\omega_0]}{(P_5 - P_3{\omega_0}^2)^2 + {P_4}^2{\omega_0}^2} + 2j\pi\right\} \quad j = 0,1,2,3,4 \ldots\ldots\ldots\ldots$$

Further we have the following

1. If $\tau \in [0, \tau_0)$, all zeros of equation (4.8) (4.9) have -ve real parts.

2. If $\tau = \tau_0$, equation (4.8) has a set of conjugate purely complex zeros $\pm i\omega_0$, all the other zeros have -ve real parts.

Proof. The equation (4.8) has a solution $\lambda = 0$, iff $P_5 = 0$. This directly oppose to the third condition in $(\mathcal{H}_1)$. Therefore, the equation (4.8) has not a solution

$\lambda = 0$. Let for some value of $\tau \geq 0$, The equation (4.8) has a solution $\lambda = i\omega$,where $\omega > 0$.

$$(i\omega)^3 + P_1(i\omega)^2 + P_2(i\omega) + (P_3(i\omega)^2 + P_4(i\omega) + P_5)e^{-i\omega\tau} = 0$$

$$[-i\omega^3 - P_1\omega^2 + P_2 i\omega] + [P_3\omega^2 - P_4 i\omega - P_5](i\sin\omega\tau - \cos\omega\tau) = 0$$

This implies: $-P_1\omega^2 + (P_5 - P_3{\omega_0}^2)\cos(\omega\tau) + P_4\omega\sin(\omega\tau) = 0$

$P_2\omega - \omega^3 - (P_5 - P_3{\omega_0}^2)\sin(\omega\tau) + P_4\omega\cos(\omega\tau) = 0$

$$(P_5 - P_3\omega^2)\cos(\omega\tau) + P_4\omega\sin(\omega\tau) = P_1\omega^2 \qquad (4.10)$$

$$P_4 w\cos(\omega\tau) - (P_5 - P_3\omega^2)\sin(\omega\tau) = \omega^3 - P_2\omega \qquad (4.11)$$

Squaring and adding equations (4.10) and (4.11) we get

$$\omega^6 + \left({P_1}^2 - {P_3}^2 - 2P_2\right)\omega^4 + \left(2P_3P_5 - P_4 + {P_2}^2\right)\omega^2 + \left(-{P_5}^2\right) = 0$$

(4.12)

Let $\alpha = \left({P_1}^2 - {P_3}^2 - 2P_2\right)$, $\beta = \left(2P_3P_5 - P_4 + {P_2}^2\right)$, $\gamma = \left(-{P_5}^2\right)$, and $\omega^2 = y$

Equation (4.12) Implies: $y^3 + \alpha y^2 + \beta y + \gamma = 0$ (4.13)

Let y_1, y_2, y_3 denotes the three roots of the equation (4.12) so they have the following relation,

$$y_1 + y_2 + y_3 = -\alpha,\ y_1.y_2.y_3 = -\gamma \qquad (4.14)$$

It is clear from the second equation of (4.14) that reliant on the value of the determinant D_1 there exist one or all positive real roots of equation (4.13), where $D_1 = \left(\frac{P}{3}\right)^2 + \left(\frac{Q}{3}\right)^2$ and $P = \beta - \frac{1}{3}\alpha^2, Q = \frac{2}{27}\alpha^3 - \frac{1}{3}\alpha\beta + \gamma$. here are three cases for the solution of equation (4.13)

Case-I: If $D_1 > 0$, There is a real root and two conjugate imaginary roots in equation (4.13). The positive real root of equation (4.13) is given by $y_1 = 2.\sqrt[3]{-\frac{Q}{2} + \sqrt{D_1}} - \frac{1}{3}\alpha$

Case-II: If $D_1 = 0$, There are three real roots in equation (4.13), two of which are same. Especially if $\alpha > 0$, only one +ve solution exists, $y_1 = 2.\sqrt[3]{-\frac{Q}{2}} - \frac{1}{3}\alpha$. if $\alpha < 0$ there exist either a +ve real root $y_1 = 2.\sqrt[3]{-\frac{Q}{2}} - \frac{1}{3}\alpha$ for $\sqrt[3]{-\frac{Q}{2}} > -\frac{1}{3}\alpha$ and three positive real roots for $\frac{\alpha}{6} < \sqrt[3]{-\frac{Q}{2}} < -\frac{1}{3}\alpha$; $y_1 = 2.\sqrt[3]{-\frac{Q}{2}} - \frac{1}{3}\alpha$, $y_2 = y_3 = -\sqrt[3]{-\frac{Q}{2}} - \frac{1}{3}\alpha$.

Case-III: If $D_1 < 0$ there exist three distinct real root of the equation (4.13) as $y_1 = 2.\sqrt{\frac{|P|}{3}}\cos(\frac{k}{3}) - \frac{1}{3}\alpha$ $y_2 = 2.\sqrt{\frac{|P|}{3}}\cos(\frac{k}{3} + \frac{2\pi}{3}) - \frac{1}{3}\alpha$, $y_2 = 2.\sqrt{\frac{|P|}{3}}\cos(\frac{k}{3} + \frac{4\pi}{3}) - \frac{1}{3}\alpha$, where $\cos k = \left(-\frac{Q}{2\sqrt{\left(\frac{|P|}{3}\right)^3}}\right); 0 < k < \pi$

Further if $\alpha > 0$ there exist only one positive real root of the equation (4.13) otherwise if $\alpha < 0$ one or all +ve real roots may exist. If there is one +ve real root, will be equivalent to $\max\{y_1, y_2, y_3\}$.

Clearly the positive real solutions of the equation (4.13) depend upon sign of α. When $\alpha \geq 0$, the equation (4.13) has one +ve real root otherwise there may exist three real roots. It is easy to prove that $\alpha = \left(\mathrm{P_1}^2 - \mathrm{P_3}^2 - 2\mathrm{P_2}\right) > 0$ Hence equation (4.13) has only one +ve real root.

Let we denote the root by y_0. Thus, only one positive real equation (4.12) has, given by $\omega_0 = \sqrt{y_0}$, we have from the (4.10) and (4.11).

$$\cos(\omega_0\tau)\left[(\mathrm{P_5} - \mathrm{P_3}\omega_0{}^2)^2 + \mathrm{P_4}{}^2\omega_0{}^2\right] = \omega_0[\mathrm{P_1}\omega_0(\mathrm{P_5} - \mathrm{P_3}\omega_0{}^2) + \mathrm{P_4}(\omega_0{}^3 - \mathrm{P_2}\omega_0)]$$

$$\cos(\omega_0\tau) = \omega_0\frac{P_1\omega_0(P_5 - P_3\omega_0{}^2) + P_4(\omega_0{}^3 - P_2\omega_0)}{(P_5 - P_3\omega_0{}^2)^2 + P_4{}^2\omega_0{}^2}$$

We denote $\tau_j = \frac{1}{\omega_0}\left\{\arccos\frac{\omega_0[\mathrm{P_1}\omega_0(\mathrm{P_5} - \mathrm{P_3}\omega_0{}^2) + \mathrm{P_4}(\omega_0{}^3 - \mathrm{P_2}\omega_0)]}{(\mathrm{P_5} - \mathrm{P_3}\omega_0{}^2)^2 + \mathrm{P_4}{}^2\omega_0{}^2} + 2\mathrm{j}\pi\right\}$ $j = 0,1,2,3, \ldots$ (4.15)

Then $\pm i\omega_0$, purely complex solution of the equation (4.9) with $\tau = \tau_j$ (j = 0,1…).we also know that if the $\mathcal{H}_1$ condition are met, every root of equation (4.9) with $\tau = 0$ have negative real components. We get result of Lemma - 4.1 by summarizing the above discussion.

Theorem-4.1: Suppose the conditions of $\mathcal{H}_1$are satisfied. If $\tau \in [0, \tau_0)$, all solutions of equation (4.8) have -ve real component, this implies asymptotical stability of the solutions of the equations (4.4)-(4.6).

From prior description, we have the following utilising the classic Hopf-bifurcation theorem for delayed functional differential equations (Hale & Lunel, 1993).

Lemma-4.3: Assume $h(y_0) = (3{y_0}^2 + 2\alpha y_0 + \beta) \neq 0$ and the conditions in ($\mathcal{H}_1$)are satisfied. For (*j=0,1…*), denote $\lambda(\tau) = \varphi(\tau) + i\omega(\tau)$ be the solution of equation (4.8) satisfying $\varphi(\tau_j) = 0;\ \omega(\tau_j) = \omega_0$, where $\tau_j = \frac{1}{\omega_0}\left\{\arccos\frac{\omega_0[P_1\omega_0(P_5 - P_3{\omega_0}^2) + P_4({\omega_0}^3 - P_2\omega_0)]}{(P_5 - P_3{\omega_0}^2)^2 + {P_4}^2{\omega_0}^2} + 2j\pi\right\}$ j = 0,1,2,3,4 ….., When $\pm i\omega_0$ are simple purely imaginary roots of (4.8). If the transversality condition $\varphi'(\tau_j) = \left(\frac{dRe\lambda(\tau)}{d\tau}\right)_{\lambda = i\omega 0} \neq 0$ holds, for system of equations (4.4) - (4.6) a Hopf-bifurcation occurs at $\tau = \tau_j$.

Proof. Let the root of equation (4.8) be $\lambda = \lambda(\tau)$. By placing the value of $\lambda(\tau)$ in (4.8) and differentiating both side of equation (4.8) w.r.t. τ, we have;

$$\left[(3\lambda^2 + 2P_1\lambda + P_2) + (P_3\lambda^2 + P_4\lambda + P_5)(-\tau)e^{-\lambda\tau} + (2P_3\lambda + P_4)e^{-\lambda\tau}\right]\frac{d\lambda}{d\tau} = \lambda(P_3\lambda^2 + P_4\lambda + P_5)e^{-\lambda\tau}$$

Thus $\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{(3\lambda^2 + 2P_1\lambda + P_2)e^{\lambda\tau}}{\lambda(P_3\lambda^2 + P_4\lambda + P_5)} + \frac{(2P_3\lambda + P_4)}{\lambda(P_3\lambda^2 + P_4\lambda + P_5)} - \frac{\tau}{\lambda}$

From equation (4.10)-(4.13) we have

$$\varphi'(\tau_j) = \mathrm{Re}\left[\frac{(3\lambda^2 + 2P_1\lambda + P_2)e^{\lambda\tau}}{\lambda(P_3\lambda^2 + P_4\lambda + P_5)}\right] + \mathrm{Re}\left[\frac{(2P_3\lambda + P_4)}{\lambda(P_3\lambda^2 + P_4\lambda + P_5)}\right]$$

$$\varphi'(\tau_0) = \frac{1}{(P_5 - P_3\omega_0{}^2)^2 + P_4{}^2\omega_0{}^2}\left[3\omega_0{}^6 + 2\left(P_1{}^2 - P_3{}^2 - 2P_2\right)\omega_0{}^4 + (2P_3P_5 - P_4)\omega_0{}^2\right]$$

$$\varphi'(\tau_0) = \frac{1}{\Delta}[3\omega_0{}^6 + 2\alpha\omega_0{}^4 + \beta\omega_0{}^2] = \frac{\omega_0{}^2}{\Delta}[3\omega_0{}^4 + 2\alpha\omega_0{}^2 + \beta] = \frac{\omega_0{}^2}{\Delta}\, h(y_0)$$

Where $\Delta = (P_5 - P_3\omega_0{}^2)^2 + P_4{}^2\omega_0{}^2$ Notice that $\Delta > 0$ *and* $\omega_0 > 0$, we conclude that $Sign\left[\varphi'(\tau_0)\right] = sign[h(y_0)]$ This proves the Lemma-4.2.

4.7 Directional analysis of Hopf-bifurcating solution

In the last section, at the critical point values, a set of recurrent solutions obtained which bifurcates from the positive steady state E*. Using the normal form theory and manifold reduction proposed by (Schneider, 1982), an explicit formula for finding the characteristics of the Hopf-bifurcation at the critical value $\tau_j$will be derived in this section.

Let $u_1 = S - S^*, u_2 = I - I^*, u_3 = R - R^*$ and the delay τ can be normalizing by time scaling $t \to \frac{t}{\tau}$, equations (4.1)-(4.3) are transformed into

$$\frac{du_1}{dt} = (a - bI^*)u_1 - bS^*u_2 + cu_3(t-1) - bu_1u_2 + cR^*(t-1) \qquad (4.16)$$

$$\frac{du_2}{dt} = bI^*u_1 + (bS^* - d)u_2 + bu_1u_2 \qquad (4.17)$$

$$\frac{du_3}{dt} = du_2 - (\mathfrak{D} + c)u_3(t-1) - (\mathfrak{D} + c)R^*(t-1) \qquad (4.18)$$

Thus, in the phase $\mathcal{P} = \mathcal{P}\left([-1,0], \mathcal{R}_+{}^3\right)$ work can be done. Without losing of the generality, we denote the critical value τ_j by τ_0. Let $\tau = \tau_0 + \mu$, then the system of equations (4.16) - (4.18) has $\mu = 0$ a Hopf-bifurcation value. Rewrite this system as follows for ease of notation:

$$u'(t) = L_\mu(u_t) + F(\mu, u_t) \qquad (4.19)$$

Where $u(t) = (u_1(t), u_2(t), u_3(t))^T \in \mathcal{R}^3, u_t(\theta) \in \mathcal{P}$ is defined by $u_t(\theta) = u_t(\mathrm{t} + \theta)$, and

$L_\mu: \mathcal{P} \to \mathcal{R}$, $F: \mathcal{R} \times \mathcal{P} \to \mathcal{R}$ are given, respectively by

$$L_\mu \emptyset = (\tau_0 + \mu) \begin{bmatrix} \mathrm{a} - \mathrm{bI}^* & -\mathrm{bS}^* & 0 \\ \mathrm{bI}^* & (\mathrm{bS}^* - \mathrm{d}) & 0 \\ 0 & \mathrm{d} & 0 \end{bmatrix} \begin{bmatrix} \emptyset_1(0) \\ \emptyset_2(0) \\ \emptyset_3(0) \end{bmatrix} + (\tau_0 + \mu) \begin{bmatrix} 0 & 0 & c \\ 0 & 0 & 0 \\ 0 & 0 & -(\mathfrak{D} + \mathrm{c}) \end{bmatrix} \begin{bmatrix} \emptyset_1(-1) \\ \emptyset_2(-1) \\ \emptyset_3(-1) \end{bmatrix}$$

And $F(\mu, \emptyset) = (\tau_0 + \mu) \begin{bmatrix} F_1 \\ F_2 \\ F_3 \end{bmatrix}$ respectively where $F_1 = -\mathrm{b}\emptyset_1(0)\emptyset_2(0), F_2 = \mathrm{b}\emptyset_1(0)\emptyset_2(0), F_3 = 0, \emptyset(\theta) = \big(\emptyset_1(\theta), \emptyset_2(\theta), \emptyset_3(\theta)\big)^T \in \mathcal{P}\big([-1,0], {\mathcal{R}_+}^3\big)$.

By representation theorem given by F. Riesz (Fuglede, 1955), there occur a function $\eta(\theta, \mu)$ of bounded variation for $\theta \in [-1,0]$, so that $L_\mu \emptyset = \int_{-1}^{0} d\,\eta(\theta, 0)\emptyset(\theta)$ for $\emptyset \in \mathcal{P}$. In fact, choose

$$L_\mu \emptyset = (\tau_0 + \mu) \begin{bmatrix} \mathrm{a} - \mathrm{bI}^* & -\mathrm{bS}^* & 0 \\ \mathrm{bI}^* & (\mathrm{bS}^* - \mathrm{d}) & 0 \\ 0 & \mathrm{d} & 0 \end{bmatrix} \begin{bmatrix} \emptyset_1(0) \\ \emptyset_2(0) \\ \emptyset_3(0) \end{bmatrix} + (\tau_0 + \mu) \begin{bmatrix} 0 & 0 & c \\ 0 & 0 & 0 \\ 0 & 0 & -(\mathfrak{D} + \mathrm{c}) \end{bmatrix} \begin{bmatrix} \emptyset_1(-1) \\ \emptyset_2(-1) \\ \emptyset_3(-1) \end{bmatrix}$$

Here δ is Dirac delta function. For $\emptyset \in \mathcal{P}\big([-1,0], {\mathcal{R}_+}^3\big)$, the system (4.19) is equivalent to

$$u'(t) = \mathcal{A}(\mu)u_t + \mathcal{F}(\mu)u_t \tag{4.20}$$

For $\psi \in \mathcal{P}^1\big([-1,0], {\mathcal{R}_+}^3\big)$, define

$$\mathcal{A}^*\psi(s) = \begin{cases} -\frac{d\psi(s)}{ds}, & s \in [-1,0) \\ \int_{-1}^{0} d\,\eta^T(-t, 0)\psi(-t), & s = 0. \end{cases}$$ And bilinear inner product

$$< \psi(s), \emptyset(\theta) > = \overline{\psi(0)}\emptyset(0) — 10 \int_{\xi=\theta}^{\theta} \overline{\psi}(\xi - \theta) d\eta(\theta)\phi(\xi)\, d\xi$$

(4.21)

Since $i\omega_0$ are eigen values of $\mathcal{A}(0)\ and\ \mathcal{A}^*$,$\mathcal{A} = \mathcal{A}(0)$ are adjoint operators. Thus they become the eigen values of$\mathcal{A}^*$.Suppose that $q(\theta) = q(0)e^{i\omega_0\theta}$ is a latent vector of $\mathcal{A}(0)$ corresponding to the proper value $i\omega_0$. Then $\mathcal{A}(0) = i\omega_0\, q(\theta)$. When $\theta = 0$, $\left[i\omega_0 I - \int_{-1}^{0} d\eta(\theta)e^{i\omega_0\theta}\right] q(0) = 0$, this gives $q(0) = (1, v_1, v_2)^T$ where

$v_1 = \frac{(\mathrm{a} - \mathrm{bI}^*) - i\omega_0}{\mathrm{bS}^*}\ \ and\ v_2 = \frac{\mathrm{d}\,(i\omega_0 - (\mathrm{a} - \mathrm{bI}^*))}{i\omega_0 \mathrm{bS}^*}$

By the same way it can be proved that $q^*(s) = D(1, {v_1}^*, {v_2}^*)e^{i\omega_0\tau_0 s}$ represent the latent value of $\mathcal{A}^*$ corresponds to latent vector $-i\omega_0$, where ${v_1}^* = \frac{(\mathrm{a} - \mathrm{bI}^*) + i\omega_0}{\mathrm{bS}^*}\ \ and\ {v_2}^* = \frac{\mathrm{d}\,(i\omega_0 + (\mathrm{a} - \mathrm{bI}^*))}{i\omega_0 \mathrm{bS}^*}$. the value of D is required to assure that $< q^*(s), q(\theta) > = 1$, From equation (4.20), $< q^*(s), q(\theta) > = \overline{D}\left(1, \overline{{v_1}^*}, \overline{{v_2}^*}\right)(1, v_1, v_2)^T -$

$\int_{-1}^{0} \int_{\xi=\theta}^{\theta} \overline{D}\left(1, \overline{{v_1}^*}, \overline{{v_2}^*}\right) e^{-i\omega_0\tau_0(\xi-\theta)} d\eta(\theta)(1, v_1, v_2)^T e^{i\omega_0\tau_0}\, d\xi$

$= \overline{D}\left\{1 + v_1\overline{{v_1}^*} + v_2\overline{{v_2}^*} - \int_{-1}^{0}\left(1, \overline{{v_1}^*}, \overline{{v_2}^*}\right)\theta e^{i\omega_0\tau_0\theta}(1, v_1, v_2)^T\right\}$

$= \overline{D}\left\{1 + v_1\overline{{v_1}^*} + v_2\overline{{v_2}^*} + \tau_0\overline{{v_1}^*}W^*(\beta v_2 - \alpha v_1)e^{i\omega_0\tau_0}\right\}$

Hence, choose $\overline{D} = \frac{1}{\left(1 + v_1\overline{{v_1}^*} + v_2\overline{{v_2}^*} + \tau_0\overline{{v_1}^*}W^*(\beta v_2 - \alpha v_1)e^{i\omega_0\tau_0}\right)}$

such that $< q^*(s), q(\theta) > = 1, < q^*(s), \overline{q(\theta)} > = 0$.

By following K. R. Schneider, Hassard, B. D. (Li et al., 2001) approach we compute the coordinates describing the 'centre manifold' $\mathcal{P}_0$ at $\mu = 0$ by using the same notations. manifold $\mathcal{P}_0$ at $\mu = 0$. Let equation (4.19) has solution u_t with $\mu = 0$. We Define

$z(t) =< q^*(s), u_t(\theta) >,$

$\mathrm{W}(t, \theta) = u_t(\theta) - 2Re(z(t)q(\theta))$ (4.22)

On the centre manifold $\mathcal{P}_0$, $\mathrm{W}(t,\theta) = \mathrm{W}\left(z(t), \overline{z(t)}, \theta\right)$

In the direction of q^* and $\overline{q^*}$ the local coordinates for centre manifold $\mathcal{P}_0$ are z and $\overline{z}$. Note that u_t is real is the necessary condition for W is real. For solution $u_t \in \mathcal{P}_0$ of equation (4.20), since $\mu = 0$, $z'(t) = i\omega_0\tau_0 z + <$ $\overline{q^*}(\theta), F\big(0, W(z,\overline{z},\theta) + 2Re\big(z(t)q(\theta)\big)\big) >$

$= i\omega_0\tau_0 z + \overline{q^*}(0)\, F\big(0, \mathrm{W}(z,\overline{z},0) + 2Re\big(z(t)q(\theta)\big)\big) \equiv i\omega_0\tau_0 z +$ $\overline{q^*}(0)F_0(z,\overline{z})$

Rewrite this equation as: $z'(t) = i\omega_0\tau_0 z(t) + g(z,\overline{z})$ (4.23)

Where $g(z,\overline{z}) = \overline{q^*}(0)F_0(z,\overline{z}) = g_{20}(\theta)\frac{z^2}{2} + g_{11}(\theta)z\overline{z} + g_{02}(\theta)\frac{\overline{z}^2}{2} +$ $g_{21}(\theta)\frac{z^2\overline{z}}{2} + \cdots$ (4.24)

As $u_t(\theta) = (u_{1t}, u_{2t}, u_{3t}) = \mathrm{W}(t,\theta) + z\,q(\theta) + \overline{z}\overline{q(\theta)}$ and $q(0) = (1, v_1, v_2)^T \mathrm{e}^{\mathrm{i}\omega_0\tau_0\theta}$, so $u_{1t}(0) = z + \overline{z} + \mathrm{W}_{20}{}^{(1)}(0)\,\frac{z^2}{2} + \mathrm{W}_{11}{}^{(1)}(0)\; z\overline{z} +$ $\mathrm{W}_{02}{}^{(1)}(0)\,\frac{\overline{z}^2}{2} + \cdots,$

$u_{2t}(0) = v_1 + \overline{v_1}\,\overline{z} + \mathrm{W}_{20}{}^{(2)}(0)\frac{z^2}{2} + \mathrm{W}_{11}{}^{(2)}(0)\; z\overline{z} + \mathrm{W}_{02}{}^{(2)}(0)\frac{\overline{z}^2}{2} + \cdots,$

$u_{3t}(0) = v_{2_1} z + \overline{v_{2_1}}\,\overline{z} + \mathrm{W}_{20}{}^{(3)}(0)\frac{z^2}{2} + \mathrm{W}_{11}{}^{(3)}(0)\; z\overline{z} +$ $\mathrm{W}_{02}{}^{(3)}(0)\frac{\overline{z}^2}{2} + \cdots,$

$u_{1t}(-1) = ze^{-i\omega_0\tau_0} + \overline{z}e^{i\omega_0\tau_0} + \mathrm{W}_{20}{}^{(1)}(-1)\frac{z^2}{2} + \mathrm{W}_{11}{}^{(1)}(-1)\; z\overline{z} +$ $\mathrm{W}_{02}{}^{(1)}(-1)\frac{\overline{z}^2}{2} + \cdots,$

$u_{2t}(-1) = v_1 e^{-i\omega_0\tau_0} z + \overline{v_1}\, e^{i\omega_0\tau_0}\,\overline{z} + \mathrm{W}_{20}{}^{(2)}(-1)\frac{z^2}{2} + \mathrm{W}_{11}{}^{(2)}(-1)\; z\overline{z} +$ $\mathrm{W}_{02}{}^{(2)}(-1)\frac{\overline{z}^2}{2} + \cdots,$

As a result of comparing coefficients with equation (4.24) we get: $g_{20} = \overline{D}(1, v_1, v_2)f_{z^2}$, $g_{02} = \overline{D}\left(1, \overline{v_1}, \overline{v_2^*}\right)f_{\overline{z}^2}$, $g_{11} = \overline{D}\left(1, \overline{v_1}, \overline{v_2^*}\right)f_{z\overline{z}}, g_{21} = \overline{D}\left(1, \overline{v_1}, \overline{v_2^*}\right)f_{z^2\overline{z}}$

In order to find the value of g_{21}, the computation of $\mathrm{W}_{20}(\theta)$ and $\mathrm{W}_{11}(\theta)$ should be prioritised. From the equations (4.20) and (4.22) ;

Let $\mathrm{W}' = \mathcal{A}\mathrm{W} + H(z, \overline{z}, \theta)$, (4.25)

Where $H(z, \overline{z}, \theta) = H_{20}(\theta)\frac{z^2}{2} + H_{11}(\theta)z\overline{z} + H_{02}(\theta)\frac{\overline{z}^2}{2} + H_{21}(\theta)\frac{z^2\overline{z}}{2} + \cdots$, (4.26)

However, in lieu, on $\mathcal{P}_0$ close to the origin $\mathrm{W}' = \mathrm{W}_z z' + \mathrm{W}_z\overline{z}'$

Simplifying and equating the coefficients, we get

$[\mathcal{A} - 2i\omega_0 I]\mathrm{W}_{20}(\theta) = -H_{20}(\theta)$, $\mathcal{A}\mathrm{W}_{11}(\theta) = -H_{11}(\theta)$ (4.27)

By equation (4.20), for $\theta \in [-1,0)$,

$H(z, \overline{z}, \theta) = -\overline{q^*}(0)\overline{F_0}q(\theta) - \overline{q^*}(0)\overline{F_0}\,\overline{q}(\theta) = -gq(\theta) - \overline{g}\,\overline{q}(\theta)$

Comparing the coefficients with (4.23) for $\theta \in [-1,0]$ that

$H_{20}(\theta) = -g_{20}q(\theta) - \overline{g_{02}}\,\overline{q}(\theta)$, $H_{11}(\theta) = -g_{11}q(\theta) - \overline{g_{11}}\,\overline{q}(\theta)$.

From equations (4.23) & (4.26) and the definition of A we obtain

$\mathrm{W}_{20}(\theta) = 2i\omega_0\tau_0\mathrm{W}_{20}(\theta) + g_{20}q(\theta) + \overline{g_{02}}\,\overline{q}(\theta)$

Solving for $\mathrm{W}_{20}(\theta)$: $\mathrm{W}_{20}(\theta) = \frac{ig_{20}}{\omega_0\tau_0}q(0)e^{i\omega_0\tau_0\theta} + \frac{i\overline{g_{02}}}{3\omega_0\tau_0}\overline{q}(0)e^{-i\omega_0\tau_0\theta} + E_1e^{2i\omega_0\tau_0\theta}$,

And similarly, $\mathrm{W}_{11}(\theta) = \frac{-ig_{11}}{\omega_0\tau_0}q(0)e^{i\omega_0\tau_0\theta} + \frac{i\overline{g_{11}}}{\omega_0\tau_0}\overline{q}(0)e^{-i\omega_0\tau_0\theta} + E_2$

The three-dimensional vectors E_1 and E_2,can be determined by setting $\theta = 0$ in H. In fact since $H(z, \overline{z}, \theta) = -2Re\left[\overline{q^*}(0)F_0q(0)\right] + F_0$, So

$H_{20}(\theta) = -g_{20}q(\theta) - \overline{g_{02}}\ \overline{q}(\theta) + F_{z^2}, \qquad H_{11}(\theta) = -g_{11}q(\theta) - \overline{g_{11}}\ \overline{q}(\theta) + F_{z\bar{z}}$

Where $F_0 = F_{z^2}\frac{z^2}{2} + F_{z\bar{z}}z\bar{z} + F_{\bar{z}^2}\frac{\bar{z}^2}{2} + \cdots$ Hence combining the definition of $\mathcal{A}$,

$\int_{-1}^{0} d\,\eta(\theta)\mathrm{W}_{20}(\theta) = 2i\omega_0\tau_0\mathrm{W}_{20}(0) + g_{20}q(0) + \overline{g_{02}}\ \overline{q}(0) - F_{z^2}$ and $\int_{-1}^{0} d\,\eta(\theta)\mathrm{W}_{11}(\theta) = g_{11}q(0) - \overline{g_{11}}\,\overline{q}(0) - F_{z\bar{z}}$.

Notice that $\left[i\omega_0\tau_0 I - \int_{-1}^{0} e^{i\omega_0\tau_0\theta}d\eta(\theta)\right]q(0) = 0$ and $\left[-i\omega_0\tau_0 I - \int_{-1}^{0} e^{-i\omega_0\tau_0\theta}d\eta(\theta)\right]\overline{q}(0) = 0,$ Which Implies $\left[2i\omega_0\tau_0 I - \int_{-1}^{0} e^{2i\omega_0\tau_0\theta}d\eta(\theta)\right]E_1 = F_{z^2}$ and $-\left[\int_{-1}^{0} d\eta(\theta)\right]E_2 = F_{z\bar{z}}$

Hence,

$$\begin{bmatrix} (2i\omega_0 - (\mathrm{a} - \mathrm{bI}^*)) & \mathrm{bS}^* & -ce^{-2i\omega_0\tau_0} \\ -\mathrm{bS}^* & (2i\omega_0 - (\mathrm{bS}^* - \mathrm{d})) & 0 \\ 0 & -\mathrm{d} & \left(2i\omega_0 + (\mathfrak{D} + \mathrm{c})e^{-2i\omega_0\tau_0}\right) \end{bmatrix} E_1 = -2\begin{bmatrix} \mathrm{b}v_1 e^{-i\omega_0\tau_0\theta} \\ -\mathrm{b}v_1 e^{-i\omega_0\tau_0\theta} \\ 0 \end{bmatrix}$$

and

$$\begin{bmatrix} (-(\mathrm{a} - \mathrm{bI}^*) & \mathrm{bS}^* & -c \\ -\mathrm{bS}^* & (-(\mathrm{bS}^* - \mathrm{d})) & 0 \\ 0 & -\mathrm{d} & (\mathfrak{D} + \mathrm{c}) \end{bmatrix} E_2 = -2\begin{bmatrix} \mathrm{b}Re\{v_1\}e^{i\omega_0\tau_0\theta} \\ -\mathrm{b}Re\{v_1\}e^{i\omega_0\tau_0\theta} \\ 0 \end{bmatrix}$$

The arguments can thus be used to express g_{21} the parameters. Based on the above study, every g_{ij} can be find out. As a result, the successive quantities can be find out:

$\mathcal{P}_1(0) = \frac{i}{2\omega_0\tau_0}\left(g_{11}g_{20} - 2|g_{11}|^2 - \frac{|g_{02}|^2}{3}\right) + \frac{g_{21}}{2}$, $\mu_1 = -\frac{Re\{C_1(0)\}}{Re\{\lambda'(\tau_0)\}}$, $\beta_1 = 2Re\{P_0(0)\}$,

$T_1 = -\frac{Im\{C_1(0)\} + \mu_2 Im\{\lambda'(\tau_0)\}}{\omega_0\tau_0}$ (4.28)

Theorem 4.7.1: μ_1 gives the direction of Hopf-bifurcation. If $\mu_1 > 0(\mu_1 < 0)$, then the Hopf bifurcation is supercritical (subcritical) and bifurcating periodic solutions exist for $\tau > \tau_0$ $(\tau < \tau_0)$. β_1 gives the stability of bifurcating solutions and are orbitally asymptotically stable (unstable) if $\beta_1 < 0$ $(\beta_1 > 0)$. The value of T_1 defines the period if $T_1 > 0$ $(T_1 < 0)$ the bifurcating periodic solution's period increases (decreases) .

4.8 Numerical Example

Numerical simulation is carried by using the following values:

$a = 0.147, b = 0.1, c = 0.0001, d = 0.8, \mathfrak{D} = 0.0749,$

$E^*(S^* = 2.78007, I^* = 0.0932, R^* = 25.8356)$ with starting values: $S(0) = 10, I(0) = 10, R(0) = 10.$

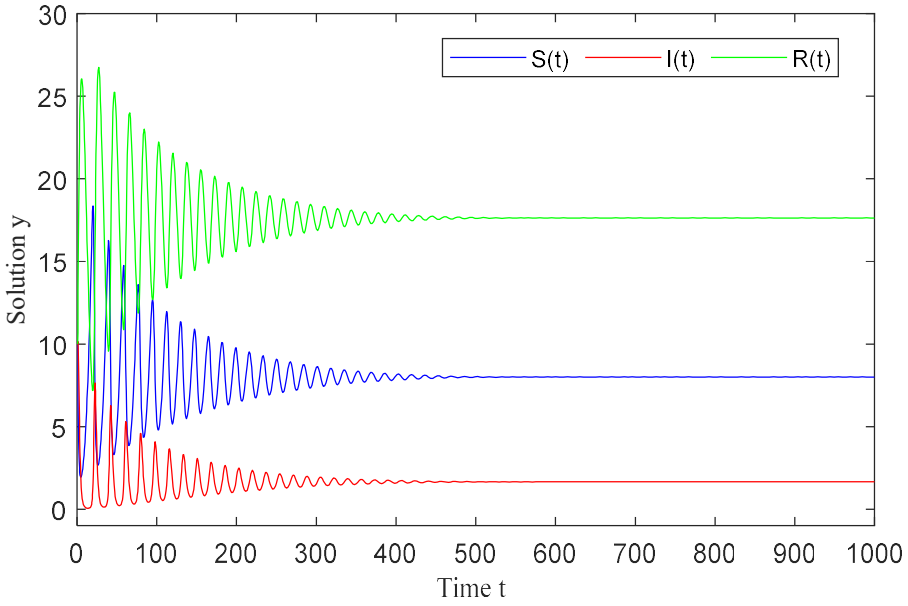

Figure 4.2 : When the time delay parameter τ has value less than 7.15, i.e., τ < 7.15, the Equilibrium $E^*(S^*, I^*, R^*)$ demonstrates asymptotic stability

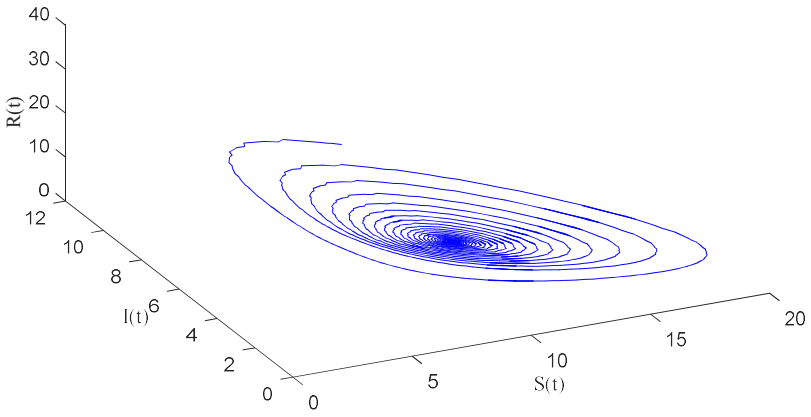

Figure 4.3 : Phase space view when the time delay parameter τ has value less than 7.15, i.e., τ < 7.15, the Equilibrium E*(S*, I*, R*) demonstrates asymptotic stability.

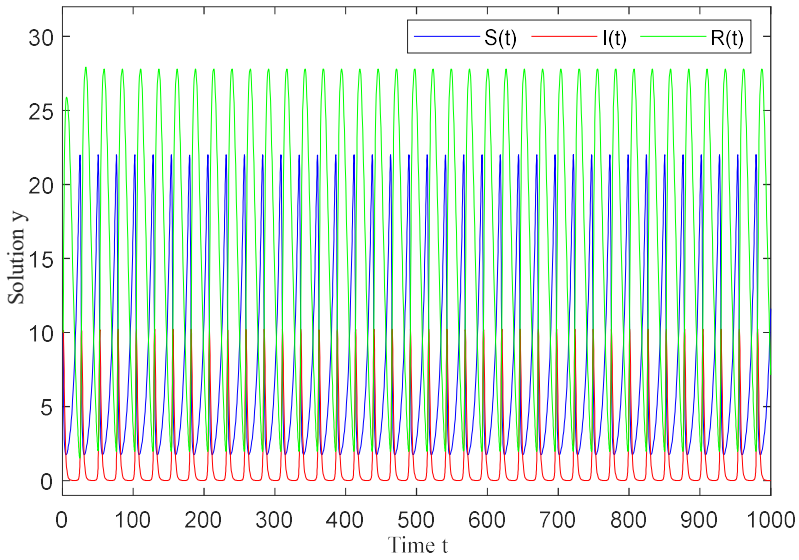

Figure 4.4 : The Equilibrium E*(S*, I*, R*) shows Hopf-bifurcation when the time delay parameter τ crosses the critical point value i.e., $\tau \geq 7.15$

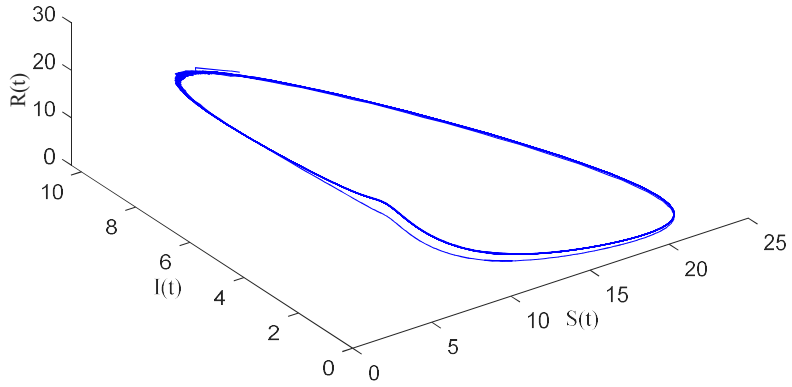

Figure 4.5 : Phase space view of the Equilibrium E*(S*, I*, R*) shows Hopf-bifurcation when the time delay parameter τ crosses the critical point value i.e., $\tau \geq 7.15$

4.9 State variable sensitivity analysis with respect to model parameters

The model in this study includes constant parameters. To calculate the general sensitivity coefficients, the 'Direct Method' is used. In this method we assume that all parameters are constants, and then estimates the sensitivity coefficients by solving sensitivity equations alongside the original system. If all of the parameters (a, b, c, d) in the system model (4.1) – (4.3) are presumed to be constants, all that required to conduct sensitivity analysis is to calculate the partial derivatives of the solution for each parameter. Consider model parameter 'c' as an example: partial derivatives of the solution (S, I, R) with respect to parameter 'c' give rise to the set of sensitivity equations:

$$\frac{dS_1}{dt} = \big(a - bI(t)\big)\, S_1 - bS(t)\, S_2 + c\, S_3(t-\tau) \qquad (4.29)$$

$$\frac{dS_2}{dt} = bI(t)\, S_1 + bS(t)\, S_2 - dS_2 \qquad (4.30)$$

$$\frac{dS_3}{dt} = dS_2 - (\mathfrak{D} + c)S_3(t-\tau) \qquad (4.31)$$

Where $S_1 = \frac{\partial S}{\partial c}, S_2 = \frac{\partial I}{\partial c}, S_3 = \frac{\partial R}{\partial c}$

The sensitivity of the state variables (S, I, R) with respect to the model parameter ‘c’ is then estimated using this system of sensitivity equations (4.29) – (4.31) and the original system of equations (4.1) – (4.3). Estimating the sensitivity of the state variables with regard to the parameters a, b and d follows a similar process and arguments.

The parameter ‘c’ represents the mutation rate. As the mutation rate increases the system should tend to become stable. The same situation is depicted by the Figure 4.6, Figure 4.7 and Figure 4.8. As the value of ‘c’ is increases from 0.0001 to 0.0021, the system shift from limit cycle to asymptotic stability, and when it further increases from 0.0021 to 0.01 the system tends to become absolutely stable.

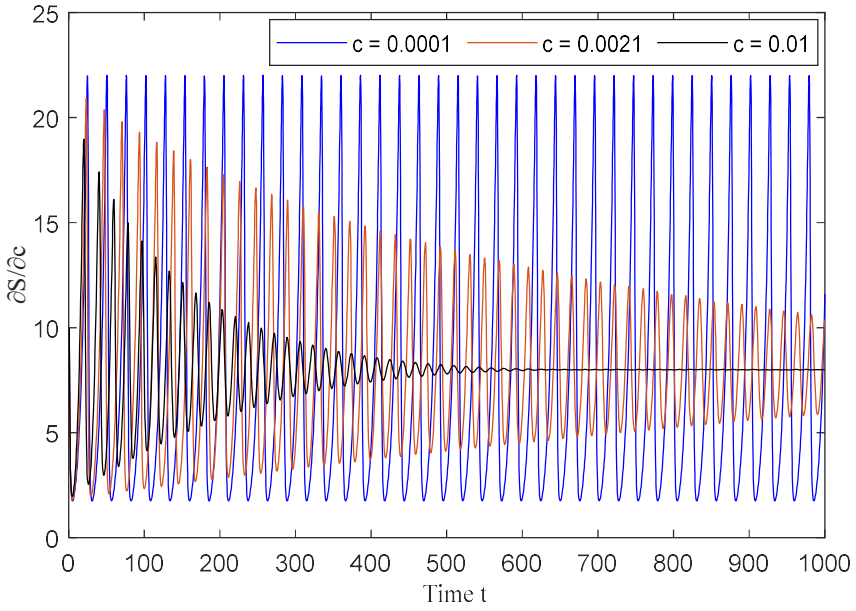

Figure 4.6 : Time series graph depicting partial changes in the susceptible population S(t) for various values of the mutation rate ‘c’.

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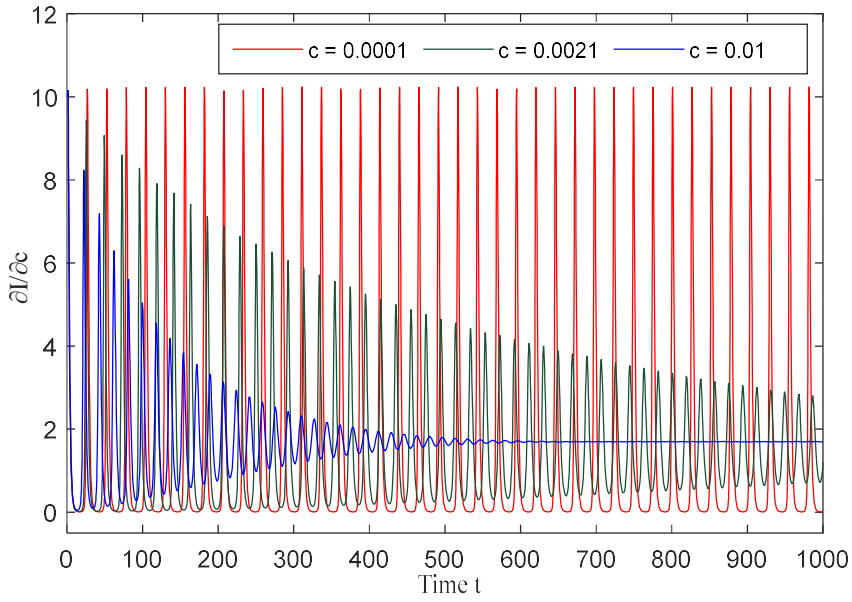

Figure 4.7 : Time series graph depicting partial changes in the infected population I(t) for various values of mutation rate ‘c’.

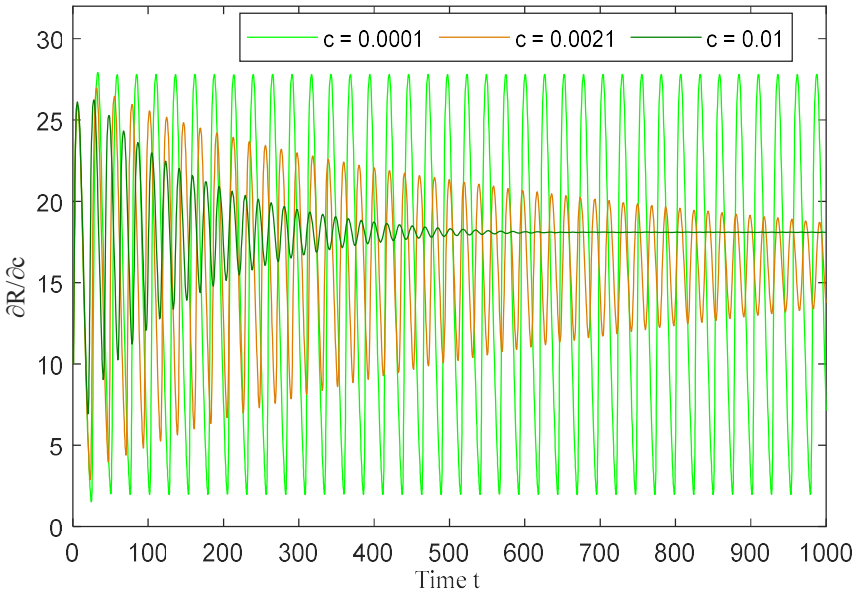

Figure 4.8 : Time series graph depicting partial changes in the recovered population R(t) for various values of mutation rate ‘c’.

The parameter ‘a’ represents the growth rate of susceptible population. As the growth rate increases the system should tend to become stable. The same situation is depicted by the Figure 4.9, Figure 4.10 and Figure 4.11. As the value of ‘a’ is increases from 0.141 to 0.447, the system shift from limit cycle to asymptotic stability, and when it further increases from 0.447 to 0.647 the system tends to become absolutely stable.

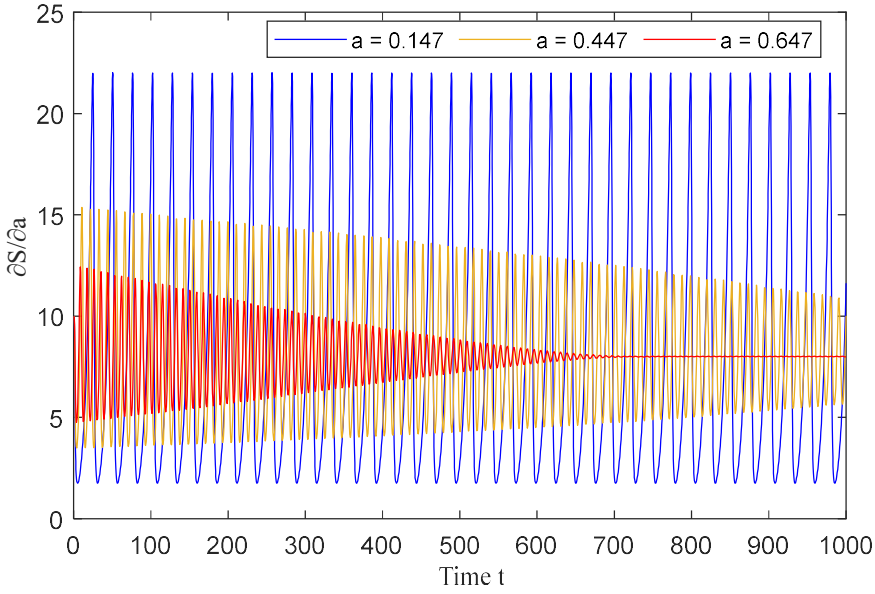

Figure 4.9 : Time series graph depicting partial changes in the susceptible population S(t) for various values of the growth rate ‘a’.

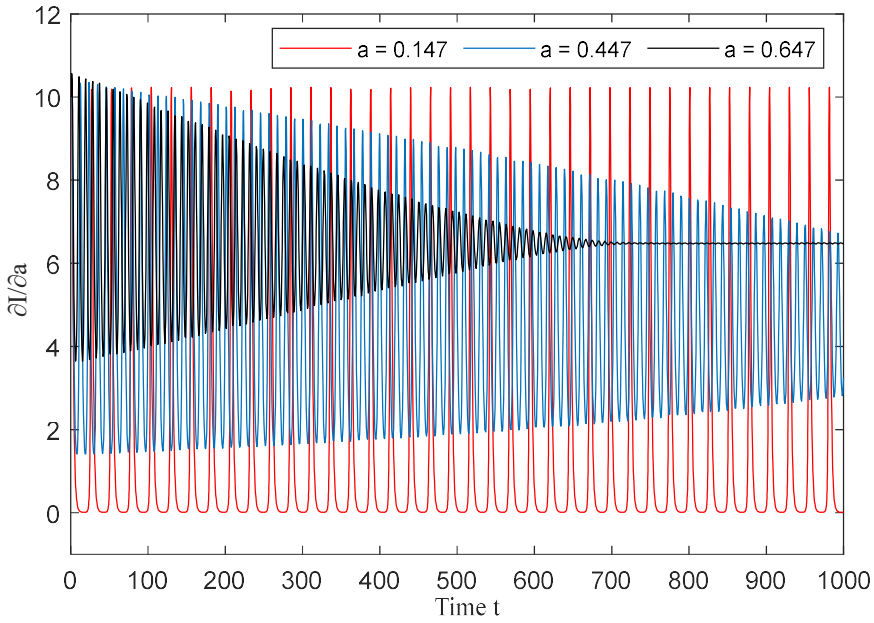

Figure 4.10 : Time series graph depicting partial changes in the infected population I(t) for various values of the growth rate ‘a’.

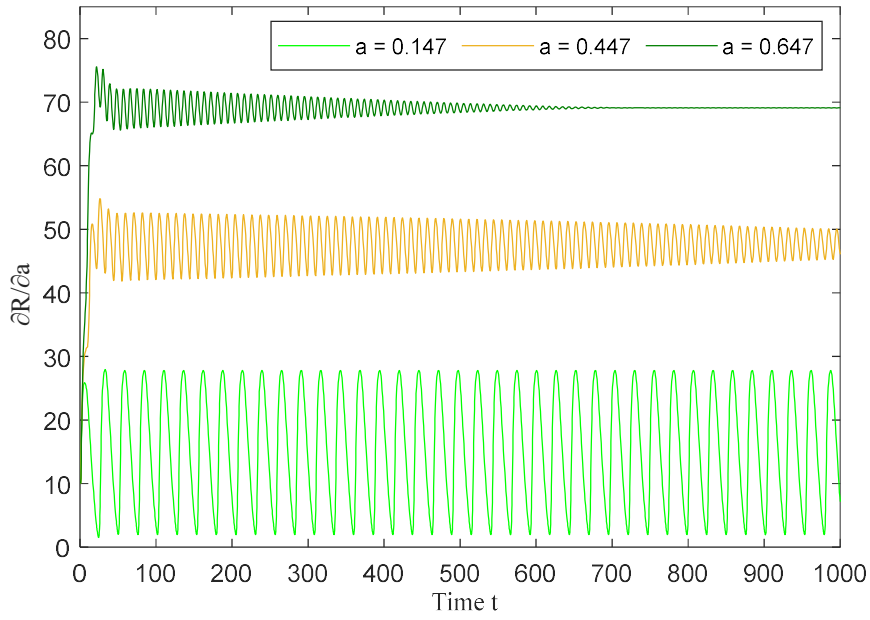

Figure 4.11 : Time series graph depicting partial changes in the recovered population R(t) for various values of the growth rate ‘a’.

4.10 Discussion about basic reproduction number (R_0)

The basic reproduction number R_0 of an infection is defined as "the expected number of cases directly generated by one case in a population where all individuals are susceptible to infection" The value of R_0 is calculate here by next generation matrix approach.

Let H be the matrix of infection rates and K be the matrix of transmission rates.

$$H = \begin{bmatrix} 0 & \mathrm{b} \\ 0 & 0 \end{bmatrix}, K = \begin{bmatrix} \mathrm{d} & 0 \\ -\mathrm{d} & (\mathfrak{D} + \mathrm{c}) \end{bmatrix} \text{ such that } |K| = \mathrm{d}(\mathfrak{D} + \mathrm{c}) \neq 0$$

$$K^{-1} = \begin{bmatrix} 1/\mathrm{d} & 0 \\ 1/(\mathfrak{D} + \mathrm{c}) & 1/(\mathfrak{D} + \mathrm{c}) \end{bmatrix}$$

$$HK^{-1} = \begin{bmatrix} \mathrm{b}/(\mathfrak{D} + \mathrm{c}) & \mathrm{b}/(\mathfrak{D} + \mathrm{c}) \\ 0 & 0 \end{bmatrix}$$

The basic reproduction number R_0 is given by spectral radius of HK^{-1} (Britton & Ouédraogo, 2018). Hence, $R_0 = \rho(HK^{-1}) = \mathrm{b}/(\mathfrak{D} + \mathrm{c})$

The comparison of three curves in the Figure 4.12 tells that the increase and decrease of the value of reproduction number R_0 has significant impact on the Infected population I. The reproduction number is directly proportion to transmission rate b and inversely proportional to total recovery rate $(\mathfrak{D} + c)$. If $(\mathfrak{D} + c) = 0.075$ is kept same for three curves, but b is increased gradually that is $b = 0.0375 \rightarrow 0.1 \rightarrow 0.75$, we get three increasing values of R_0that is $R_0 = 0.5 \rightarrow 1.33 \rightarrow 10$. It is observed that the peak of infected population is reached on 11$^{\text{th}}$ day when the value of reproduction number $R_0 = 0.5$. But the infection spreads comparatively fast if the value of reproduction number increases to $R_0 = 1.33$ and the peak of infected population is reached on 3$^{\text{rd}}$ day. The infection spreads so rapidly that the steepest of these peaks of infected population is reached on 2$^{\text{nd}}$ day only, if the value of reproduction number $R_0 = 10$.

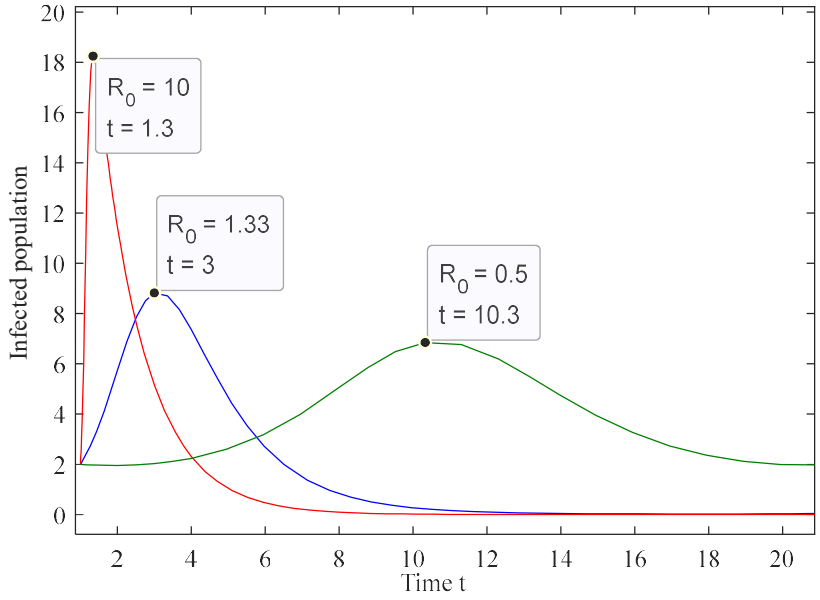

Figure 4.12 : The peaks of infected population for different values of reproduction number.

Figure 13 shows the significance of reproduction number R_0 on recovered population R. As reproduction number R_0 is inversely proportional to total recovery rate $(\mathfrak{D} + \mathrm{c})$. So, here the values of $(\mathfrak{D} + \mathrm{c})$ are decreased, there is an increase in the values of reproduction number R_0. As the value of $(\mathfrak{D} + \mathrm{c})$ is gradually decreased that is $(\mathfrak{D} + \mathrm{c}) = 0.75 \rightarrow 0.2 \rightarrow 0.1$, the corresponding increased values of R_0 are $R_0 = 0.13 \rightarrow 0.5 \rightarrow 10$. The Figure 4.13 clearly tells that how the recovery rises and the system approaches stability with increasing values of R_0.

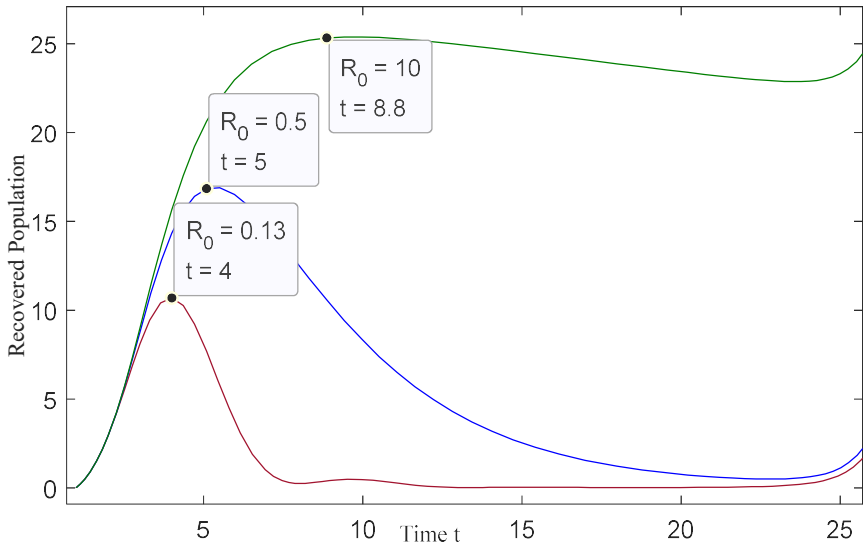

Figure 4.13 : The recovered population approaches stability with increasing value of reproduction number.

4.11 Conclusion

In this chapter an SIR model with virus mutation is analysed using delay differential equations. When the virus mutates, the recovered population losses its immunity and becomes susceptible again. The incubation period is denoted by τ. For the study of stability, feasible non-zero equilibrium point E^* has been considered. The delay factor has been incorporated in the term defining recovered hosts. The nature of the roots of exponential characteristic equation formed by the system of equation (4.1) - (4.3) has been studied in detail using Routh-Hurwitz criteria and results of theory of equations with the help of lemmas (4.1) - (4.3). It is found that the system shows asymptotic satiability as long as the value of the time lag parameter τ is less than the critical point value that is $\tau < 7.15$. The same fact is also shown by the Figure 4.2 and the Figure 4.3 shows the phase view of asymptotic satiability. But, as soon as the value of time lag parameter τ crosses the critical point value that is $\tau \geq 7.15$, the system losses stability and the limit cycles are seen via Hopf-bifurcation. The same is also shown by Figure 4.4 and the Figure 4.5 represents the phase space diagram of Hopf- bifurcation. The period, direction and stability of these bifurcating periodic solutions have also been

determined. The conditions for supercritical and subcritical bifurcating solutions have been laid down using an algorithm (Schneider, 1982). Sensitivity analysis has been carried out, telling us about the model parameters that are responsible for dynamic behaviour of the equilibrium point, apart from the delay parameter τ. We used the direct method of sensitivity (Rihan, 2003b). The system is very sensitive to the model parameter 'c' as well as the parameter 'a' which represents the rate of mutation and growth rate respectively. As the value of 'c' and 'a' increase, the state variables S(t), I(t) and R(t) tends to move from limit cycles to more of asymptotic stability and finally to absolute stability as shown by Figure 4.6 to Figure 4.11.

The effect of reproduction number R_0 on the shapes of infected and recovered population is studied. The Figure 4.12 shows that the peak of infected population is reached on 11^{th} day when the value of reproduction number $R_0 = 0.5$. But the infection spreads comparatively fast if the value of reproduction number increases to $R_0 = 1.33$ and the peak of infected population is reached on 3^{rd} day. The infection spreads so rapidly that the steepest of these peaks of infected population is reached on 2^{nd} day only, if the value of reproduction number $R_0 = 10$. The Figure 4.13 clearly tells that how the recovery rises and the system approaches stability with increasing values of R_0.

Chapter 5

Modelling the Effect of Discrete Delay in SIR Epidemic Model with Logistic Growth

5.1 Introduction

To trace the mathematical modelling of epidemics back through history, we need to start with Daniel Bernoulli (Bacaër & Bacaër, 2011a; Bernoulli, 1766; Bernoulli Daniel, 1760; Gabriel & de la Harpe, 2010). By separating the population into three groups susceptible (S), infected (I), and recovered (R), Kermack and Mckendric ("A Contribution to the Mathematical Theory of Epidemics," 1927) propose a conventional SIR epidemic model. This SIR model is helpful for analysing illnesses nowadays. A large body of work has been devoted to the study of nonlinear incidence epidemic models, and the SIR disease transmission model has a similar amount of background research (Cooke, 1979; X. A. Zhang & Chen, 1999). Numerous models illustrate the dynamics of disease using simple differential equations with no time lag. To depict the genuine dynamical behaviour of models that rely on system history, time delays are suitable. In reality, delays in epidemic models make them more realistic by describing disease latency or immunity. Delay affects population dynamics. In many real-world processes, especially biological ones, state variable dynamics depend on past values. Delay may affect infectious dynamics. Many sicknesses spread slowly due to immunity period delays (Q. Liu et al., 2016; C. Xu & Li, 2018; R. Xu et al., 2010), infection period delays (Singh, 2022; P. Yan & Feng, 2010), and incubation period delays (Ashyani et al., 2016; S. Liu et al., 2015; Naresh et al., 2011). stability, attractiveness, tenacity, cyclic oscillation, chaotic behaviour, and bifurcation are studied extensively in population models with time delay. Hopf bifurcation periodic solutions are of special importance (Cao et al., 2021; Kaddar & Talibi Alaoui, 2009; Sun, Cao, et al., 2007; Sun, Han, et al., 2007; Wu et al., 2012; C. Yan & Jia, 2014). Multiple researchers have outlined the overall features of a delayed SIR model that includes both transient immunity and a nonlinear incidence rate (Kyrychko & Blyuss, 2005; Takeuchi et al.,

2000; Wanduku & Ladde, 2012) , Considered the delay as a transient immunity interval and found the endemic equilibrium to be globally stable. This indicated that the delay did not have any impact on the system, which had a nonlinear incidence rate. (Muroya et al., 2011) proved that a nonmonotonic incidence rate delayed SIRS pandemic model is robust on a global scale, this showed that delay couldn't alter nonmonotonic system dynamics, also use Lyapunov function technique for the global stability analysis (Enatsu et al., 2012). Using a Lyapunov function, (McCluskey, 2010b) SIR model was investigated with incubation period and nonlinear incidence rate for the global stability. Delays affect system dynamics, causing Hopf bifurcation. (Ashyani et al., 2016; J. Liu, 2016; Schneider, 1982; Wu et al., 2012; C. Yan & Jia, 2014; J. Zhang et al., 2009; T. Zhang et al., 2010; Y. Zhang et al., 2021) addressed a generic nonlinear differential equation problem with a delay by discussing its Hopf bifurcation analysis. The directional analysis for the solution of delayed differentia equation using the normal form theory and manifold reduction proposed by K. R. Schneider, Hassard, B. D. (Schneider, 1982).

Basic reproductive number $\mathfrak{R}_0$ or some time called reproduction number is one of the key parameters to calculate the stability of SIR model. Mathematical analysis is employed to establish a threshold value $\mathfrak{R}_0$, that indicates when the disease is nearing an endemic level and when the solutions begin to vary. The values of $\mathfrak{R}_0$ and incubation period duration totally control the global dynamics of system. Disease always dies out and the DFE is globally asymptotically stable if $\mathfrak{R}_0 < 1$, but if $\mathfrak{R}_0 > 1$ the disease continues. (Avram et al., 2022; Sirijampa et al., 2018; Y. Song & Peng, 2006; Sun, Han, et al., 2007; Xia et al., 2018; Xue & Li, 2013) (Kumar et al., 2020; L. Liu, 2015; L. Liu & Wang, 2017; Naresh et al., 2009; Xue & Li, 2013; Y. Zhang et al., 2021) in each of these research papers stability, bifurcation and sensitivity analysis has been performed with special reference to the reproduction numbers. By analyse the incubation parameter for bifurcation, this research investigates the endemic equilibrium's local stability. It identifies

the circumstances under which the system must be both absolutely and conditionally stable. In numerical simulations, it's proven that a system with a time delay has a rich, complicated dynamic, and that as the delay rises, a series of periodic solutions develops, showing complex periodic patterns.

This chapter looks like, Section (5.2) offers the delayed SIR epidemic model and reproduction number. Sections (5.3) – (5.6) offer findings on nonzero endemic equilibrium, positivity, boundedness of solutions, and system permanence. Section (5.8) includes system (5.2)'s equilibria and the crucial value for endemic equilibrium. Section (5.7) addresses the global stability of the DEF and the lifetime of the endemic equilibrium. Section (5.9) presents the direction and stability of Hopf-bifurcation to the system of equation (5.2) by applying manifold reduction and normal form theory. Sections (5.10) and (5.11) of this research reports include numerical analysis and easy explanation.

5.2 Basic properties and Model formulation

The following are the presumptions upon which the delayed model is based. The three-dimensional model tracks the progression of the susceptible people *S(t)*, the infected people *I(t)*, and the recovered ones *R(t)* at a given time t. Thus, the overall population at time t remains the same, with carrying capacity $\mathcal{K}$. The susceptible individuals become infected when they come in contact with infected individuals at the rate of α_2. We hypothesized that a susceptible person comes into touch with an infected one at time $t-\tau$ and becomes infectious at time τ later. Under these conditions, the SIR epidemic model with a bilinear incidence rate is expressed as:

$$\left.\begin{aligned} \frac{dS}{dt} &= \alpha_1 S(t)\left(1-\frac{S(t)}{\mathcal{K}}\right) - \alpha_2 S(\mathrm{t}-\tau) I(\mathrm{t}-\tau) \\ \frac{dI}{dt} &= \alpha_2 S(\mathrm{t}-\tau) I(\mathrm{t}-\tau) - (\alpha_3+\alpha_4+\alpha_5) I(t) \\ \frac{dR}{dt} &= \alpha_3 I(t) - \alpha_5 R(t) \end{aligned}\right\} \qquad (5.1)$$

Where: $S(t) > 0,\ I(t) > 0,\ R(t) > 0$ for all t and $S(t-\tau) = k_1, I(\mathrm{t}-\tau) = k_2$, k_1, k_2 are constant for $t \in [0, \tau]$

Where α_1 is the intrinsic birth rate, α_2 is the rate at the susceptible individuals become infected when they come in contact with infected individuals, α_3 is the recovery rate of infected individuals, α_4 disease mortality rate, α_5 is the natural death rate, and $\mathcal{K}$ is the parameter that measure the carrying capacity of population.

It is expected in model (5.1) that those affected who are given effective treatment would recover, joining the recovery class after they develop temporary immunity to the disease. Note that since this model is used to track human populations, it is supposed that none of the parameters and variables are negative constants.

For the simplicity we non-dimension Alize the system of equations (5.1) as

$\acute{S} = \frac{S}{\mathcal{K}},\ \acute{I} = \frac{I}{\mathcal{K}},\ \ \acute{R} = \frac{R}{\mathcal{K}},\ \ \acute{t} = \alpha_2 \mathcal{K} t,\ \ \acute{\alpha_1} = \frac{\alpha_1}{\alpha_2 \mathcal{K}},\ \acute{\alpha_3} = \frac{\alpha_3}{\alpha_2 \mathcal{K}},\ \acute{\alpha_4} = \frac{\alpha_4}{\alpha_2 \mathcal{K}},$
$, \acute{\alpha_5} = \frac{\alpha_5}{\alpha_2 \mathcal{K}}$

Dropping the sign " ` " the system of equation (5.1) become as:

$$\left.\begin{aligned} \frac{dS}{dt} &= \alpha_1 S(t)\big(1 - S(t)\big) - S(\mathrm{t}-\tau) I(\mathrm{t}-\tau) \\ \frac{dI}{dt} &= S(\mathrm{t}-\tau) I(\mathrm{t}-\tau) - (\alpha_3 + \alpha_4 + \alpha_5) I(t) \\ \frac{dR}{dt} &= \alpha_3 I(t) - \alpha_5 R(t) \end{aligned}\right\} \qquad (5.2)$$

With initial conditions $\xi = (\xi_1, \xi_2, \xi_3)$ of system (5.2) are defined in the Banach space of continuous mapping.

$\mathbb{C}_+ = \{\xi \in \mathbb{C}([-\tau, 0], \mathbb{R}^3{}_+) : \xi_1(\vartheta) = S(\vartheta), \xi_2(\vartheta) = I(\vartheta), \xi_3(\vartheta) = R(\vartheta)\}, \vartheta \in [-\tau, 0]$ (A)

where $\xi_i(0) > 0, i = 1,2,3$, the feasible area of the system (5.2) is defined as

$\Lambda = \{ (S, I, R) \in \mathbb{R}^3 : S(0) > 0,\ I(0) > 0,\ R(0) > 0\}$

It is easy to show that the feasible region Λ is positively not changeable w.r.t system (5.1). System (5.2) has DFE $\mathbb{E}_0(1,0,0)$ .The basic reproductive number is defined as $\mathfrak{R}_0 = \frac{1}{r}$ where $r = (\alpha_3 + \alpha_4 + \alpha_5)$. (5.3)

5.3 Non-Zero Endemic Equilibrium

It is to be supposed that at equilibrium point $S(t-\tau) \cong S,\ I(t-\tau) \cong I$. The non-zero endemic equilibrium $E^*(S^*, I^*, R^*)$ is calculated as:

$$\frac{dI^*}{dt} = 0 \Rightarrow S^* = (\alpha_3 + \alpha_4 + \alpha_5) = r$$

$$\frac{dS^*}{dt} = 0 \Rightarrow I^* = \alpha_1(1-r) = \alpha_1 r(\mathfrak{R}_0 - 1)$$

$$\frac{dR^*}{dt} = 0 \Rightarrow R^* = \frac{\alpha_3 I^*}{\alpha_5} = \frac{\alpha_3\alpha_1(1-r)}{\alpha_5} = \frac{\alpha_3\alpha_1 r(\mathfrak{R}_0-1)}{\alpha_5}$$

Thus, for $\mathfrak{R}_0 > 1$, we have a unique non-zero endemic equilibrium:

$$E^*(S^*, I^*, R^*) = E^*\left(r, \alpha_1(1-r), \frac{\alpha_3\alpha_1(1-r)}{\alpha_5}\right) = E^*\left(r, \alpha_1 r(\mathfrak{R}_0 - 1), \frac{\alpha_3\alpha_1 r(\mathfrak{R}_0-1)}{\alpha_5}\right)$$

5.4 Positivity of solutions

The term "positivity" refers to the stability of a system. For positive solutions, one must show that the system of Equations (5.2) provides all of the system's solutions, where initial conditions are $S(t) > 0,\ I(t) > 0,\ R(t) > 0$ for all t and $S(t-\tau) = k_1, I(\mathrm{t}-\tau) = k_2$, k_1, k_2 are constant for $t \in [0, \tau]$, the model solutions $(\mathrm{S(t)}, \mathrm{I(t)}, \mathrm{R(t)})$ remain +ve for all time $\mathrm{t} > 0$.

Now we have $\frac{dR}{dt} = \alpha_3 I(t) - \alpha_5 R(t)$, i.e $\frac{dR}{dt} \geq -\alpha_5 R(t)$ i.e $\frac{dR}{R(t)} \geq -\alpha_5 dt$ i.e $R(t) = c_0 e^{-\alpha_5 t}$. here c_0 is the integration constant. As a result, $\mathrm{R(t)} > 0$ for all the time t. so, *S* and *I* have a similar argument.

5.5 Boundedness of solutions

Lemma:(Tchuenche et al., 2007) All feasible solutions of the system (5.2) are bounded and enter the region

$\Lambda = \{ (S, I, R) \in \mathbb{R}^3 : 0 \leq S + I + R \leq (\alpha_1 + 1)/\mu_m \}, as \text{ t} \to \infty,$
where $\mu_m = \min\{1, \alpha_4, \alpha_5\}$ for all positive initial values $\{S(0) > 0,$ $I(0) > 0,\ R(0) > 0$ for all t and $S(t-\tau) = k_1, I(\text{t}-\tau) = k_2\ ,\ k_1, k_2$ are constant for $t \in [0, \tau]\}$

With $\mathfrak{R}_0 = \frac{1}{r}$ where $r = (\alpha_3 + \alpha_4 + \alpha_5)$, for the system (5.2) there always exist the equilibrium $E_0(1,0,0)$ and if $\mathfrak{R}_0 > 1$, we have a unique non-zero endemic equilibrium: $E^*(S^*, I^*, R^*) = E^*\left(r, \alpha_1(1-r), \frac{\alpha_3\alpha_1(1-r)}{\alpha_5}\right) = E^*\left(r, \alpha_1 r(\mathfrak{R}_0 - 1), \frac{\alpha_3\alpha_1 r(\mathfrak{R}_0-1)}{\alpha_5}\right)$, where $r = (\alpha_3 + \alpha_4 + \alpha_5)$.

5.6 Permanence of the system

The set of equations(5.2) simplifies to: when E* (S*,I*,R*) is in equilibrium,

$$\left.\begin{aligned} \frac{dS^*}{dt} &= \alpha_1 S^*(t)\big(1 - S^*(t)\big) - S^*(\text{t}-\tau)I^*(\text{t}-\tau) \\ \frac{dI^*}{dt} &= S^*(\text{t}-\tau)I^*(\text{t}-\tau) - (\alpha_3 + \alpha_4 + \alpha_5)I^*(t) \\ \frac{dR^*}{dt} &= \alpha_3 I^*(t) - \alpha_5 R^*(t) \end{aligned}\right\} \quad (5.4)$$

All possible solutions to systems (5.2) and (5.4) have been shown to be constrained by (Takeuchi et al., 2000). The transcendent equation about the equilibrium $E^*$is given by:

$$\begin{vmatrix} \lambda - m_1 & -m_2 & -m_3 \\ -m_4 & \lambda - m_5 & -m_6 \\ -m_7 & -m_8 & \lambda - m_9 \end{vmatrix} = 0 \quad (5.5)$$

Where $m_1 = \alpha_1 - 2\alpha_1 S^* - I^* e^{-\lambda\tau}\ ,\ m_2 = I^* e^{-\lambda\tau}\ ,\ m_3 = 0\ ,\ m_4 = -e^{-\lambda\tau}S^*,\ m_5 = S^* e^{-\lambda\tau} - r,\ m_6 = \alpha_3,\ m_7 = 0,\ m_8 = 0,\ m_9 = -\alpha_5$

The equation (5.5) after expanding become:

$$(\lambda + \alpha_5)\left[\lambda^2 - (m_1 + m_5)\lambda + m_1 m_5 + S^* I^* e^{-2\lambda\tau}\right] = 0 \quad (5.6)$$

$\lambda^3 + (r - \alpha_1 + 2\alpha_1 S^* + \alpha_5)\lambda^2 + (2r\alpha_1 S^* - r\alpha_1 + \alpha_5(r - \alpha_1 +$

$2\alpha_1 S^*)) + \alpha_5\,(2r\alpha_1 S^* - r\alpha_1) + \Big[(I^* - S^*)\lambda^2 + \Big(\alpha_1 S^* - 2\alpha_1 S^{*2} + r I^* + \alpha_5((I^* - S^*)\Big)\lambda + \alpha_5\big(\alpha_1 S^* - 2\alpha_1 S^{*2} + r I^*\big)\Big] e^{-\lambda\tau} = 0$

$$\lambda^3 + \mathfrak{m}_1\lambda^2 + \mathfrak{m}_2\lambda + \mathfrak{m}_3 + [\mathfrak{m}_4\lambda^2 + \mathfrak{m}_5\lambda + \mathfrak{m}_6]e^{-\lambda\tau} = 0 \qquad (5.7)$$

Where $\mathfrak{m}_1 = (r - \alpha_1 + 2\alpha_1 S^* + \alpha_5)$, $\mathfrak{m}_2 = (2r\alpha_1 S^* - r\alpha_1 + \alpha_5(r - \alpha_1 + 2\alpha_1 S^*))$, $\mathfrak{m}_3 = \alpha_5\,(2r\alpha_1 S^* - r\alpha_1)$, $\mathfrak{m}_4 = (I^* - S^*)$, $\mathfrak{m}_5 = \alpha_1 S^* - 2\alpha_1 S^{*2} + r I^* + \alpha_5(I^* - S^*)$, $\mathfrak{m}_6 = \alpha_5\big(\alpha_1 S^* - 2\alpha_1 S^{*2} + r I^*\big)$, all $\mathfrak{m}_1, \mathfrak{m}_2, \mathfrak{m}_3, \mathfrak{m}_4, \mathfrak{m}_5, \mathfrak{m}_6 > 0$.

5.7 Local and global stability of disease-free equilibrium

Now in this section we will prove that DFE is asymptotically stable locally. when $\tau \in [0, \tau_0)$, endemic equilibrium is locally asymptotically stable locally and is unstable when $\tau > \tau_0$, at the same time system (5.2)(5.1) moves toward Hopf-Bifurcation at $E^*(S^*, I^*, R^*)$ when $\tau = \tau_i$.

Theorem 5.1: The disease-free equilibrium (DFE) $E_0(1,0,0)$ of the system (5.2) is,

(a) Absolutely stable if $\Re_0 < 1$,

(b) linearly neutrally stable if $\Re_0 = 1$,

(c) unstable if $\Re_0 > 1$.

Proof: When $\tau \neq 0$, the transcendental equation at the DFE, $E_0(1,0,0)$ of (5.2) will be of the form as;

$$(\lambda + \alpha_1)(\lambda + \alpha_5)\big(\lambda + r - e^{-\lambda\tau}\big) = 0 \qquad (5.8)$$

$$\Rightarrow \lambda = -\alpha_1 \;,\quad \lambda = -\alpha_5 \;,\qquad \lambda = -r + e^{-\lambda\tau} \qquad (5.9)$$

The equation (5.8) has two negative real roots given by $\lambda_1 = -\alpha_1$, $\lambda_2 = -\alpha_5$ and the third root is coming from the equation $\lambda = -r + e^{-\lambda\tau}$

(a) When $\Re_0 < 1$, from the third equation of (5.8) $\lambda = -r + e^{-\lambda\tau}$

$R_e(\lambda) = e^{-R_e(\lambda)}Cos(pq\tau\lambda) - r$, let $R_e(\lambda) \geq 0$, that implies that

$$R_e(\lambda) \leq 1 - \gamma \Rightarrow R_e(\lambda) \leq \gamma(\Re_0 - 1), \quad (5.10)$$

as $\Re_0 < 1$, we have $R_e(\lambda) < 0$ (5.11)

Hence the DFE of the system of equations (5.2) is locally asymptotically stable.

(b) If $\Re_0 = 1$, from (5.8) we have $R_e(\lambda) = 0$, This shows that the root has a -ve real part except $\lambda = 0$, which gives the fact that E_0 is linear neutral stable state when $\Re_0 = 1$.

(c) If $\Re_0 > 1$, let $\lambda + \gamma - e^{-\lambda\tau} = \digamma(\lambda)$, because $\digamma(0) = \gamma - 1 = -\gamma(\Re_0 - 1) < 0$, $\digamma(\infty) > 0$, there exist a positive value of λ . which clearly shows that the DFE of the system of equations (5.2) is asymptotically unstable. In addition to the above when $\tau = 0$, and $\Re_0 < 1$, the third root of characteristics equation of system (5.2) will be $\lambda = -\gamma + 1 = \gamma(\Re_0 - 1) < 0$, hence the system become locally asymptotically stable. Hence the theorem.

Theorem 5.2: If the basic reproductive number $\Re_0 < 1$ then the DFE $E_0(1,0,0)$ is globally stable for any value of time delay parameter τ.

Proof: for t > 1, let l_i be the translation of roots of the system if equations (5.2) with initial conditions $\{l_1(\vartheta) = S(\vartheta), l_2(\vartheta) = I(\vartheta), l_3(\vartheta) = R(\vartheta)\}$, $\vartheta \in [-\tau, 0]$. We can define a Lyapunov function as:

$$L(t) = I(t) + \int_{t-\tau}^{t} S(\vartheta)\, I(\vartheta)\, d\vartheta \quad (5.12)$$

Further $L(t) > 0$ along with the solution of the system of equations (2.8), also $L(t) = 0$, iff both $I(t)$ and $R(t)$ are zero. The derivative of (5.12) is as;

$$\frac{dL(t)}{dt} = \frac{dI(t)}{dt} + \big(S(t)I(t) - S(\mathrm{t}-\tau)I(\mathrm{t}-\tau)\big)$$

$$= S(\mathrm{t}-\tau)I(\mathrm{t}-\tau) - \gamma I(t) + \big(S(t)I(t) - S(\mathrm{t}-\tau)I(\mathrm{t}-\tau)\big)$$

$$= S(t)I(t) - \gamma I(t)$$

As $S(t) \leq 1, so$ the above equation implies $\frac{dL(t)}{dt} = S(t)I(t) - \gamma I(t) \leq \gamma I(t)(\Re_0 - 1) \leq 0$, as $\Re_0 < 1$

So we have $\frac{dL(t)}{dt} \leq 0,$ as $\Re_0 < 1$, and $\frac{dL(t)}{dt} = 0,$ if $I(t) = 0, R(t) = 0$

The maximum invariance set $\left\{\left(S(t), I(t), R(t)\right): \frac{dL(t)}{dt} = 0\right\},$ is a singleton set $\{E_0(1,0,0)\}$, when $\Re_0 < 1$. Therefore by (Hale, 2007), the DEF $E_0(1,0,0)$ is globally asymptotically stable.

Hence the theorem.

5.8 Bifurcation Analysis

In this section, we will find the conditions for Hopf-Bifurcations to exist using τ as parameter for the result. In this part we assume that the basic reproduction number that $\Re_0 > 1$, so the endemic equilibrium $E^*(S^*, I^*, R^*) = E^*\left(r, \alpha_1 r(\Re_0 - 1), \frac{\alpha_3 \alpha_1 r(\Re_0 - 1)}{\alpha_5}\right)$ exist.

Case-1: When $\tau = 0$, the characteristics equation (5.6) takes the form;

$$(\lambda + \alpha_5)[\lambda^2 - (m_1 + m_5)\lambda + m_1 m_5 + S^* I^*] = 0 \qquad (5.13)$$

Where $m_1 = \alpha_1 - 2\alpha_1 S^* - I^*, m_5 = S^* - r,$

Now $m_1 + m_5 = \alpha_1 - 2\alpha_1 S^* - I^* + S^* - r = \alpha_1 - 2\alpha_1 r - \alpha_1(1 - r) + r - r = -\alpha_1 r$

$\Rightarrow -(m_1 + m_5) = \alpha_1 r > 0$

$$m_1 m_5 + S^* I^* = (\alpha_1 - 2\alpha_1 S^* - I^*)(S^* - r) + S^* I^* = r\alpha_1(1 - r)$$
$$= \alpha_1 r^2(\Re_0 - 1) > 0$$

Therefore, all the roots of the equation (5.13) are negative, By Routh-Hurwitz's criteria $E^*(S^*, I^*, R^*)$is locally asymptotically stable equilibrium,

Now we will find the positive solution of (5.7) for $\tau > 0$ let $i\omega$ be the roots of the transcendental equation (5.7).

So, we have $(i\omega)^3 + \mathfrak{m}_1(i\omega)^2 + \mathfrak{m}_2 i\omega + \mathfrak{m}_3 + [\mathfrak{m}_4(i\omega)^2 + \mathfrak{m}_5 i\omega + \mathfrak{m}_6]e^{-\lambda i\omega} = 0$

$$-i(\omega)^3 - \mathfrak{m}_1(\omega)^2 + \mathfrak{m}_2 i\omega + \mathfrak{m}_3 + [-\mathfrak{m}_4(\omega)^2 + \mathfrak{m}_5 i\omega + \mathfrak{m}_6](cos\ (\omega\tau) - isin(\omega\tau)\ ) = 0$$

We have $(\mathfrak{m}_6 - \mathfrak{m}_4(\omega)^2)\ cos\ (\omega\tau) + \mathfrak{m}_5\omega sin(\omega\tau) = (\mathfrak{m}_1(\omega)^2 - \mathfrak{m}_3)$ (5.14)

$$\mathfrak{m}_5\omega\ cos\ (\omega\tau) - (\mathfrak{m}_6 - \mathfrak{m}_4(\omega)^2)sin(\omega\tau) = ((\omega)^3 - \mathfrak{m}_2\omega) \qquad (5.15)$$

By solving

(5.14) and (5.15) we get;

$$\Rightarrow \omega^6 + ({\mathfrak{m}_1}^2 - {\mathfrak{m}_4}^2 - 2\mathfrak{m}_2)\omega^4 + ({\mathfrak{m}_2}^2 - 2\mathfrak{m}_1\mathfrak{m}_3 + 2\mathfrak{m}_4\mathfrak{m}_6 - {\mathfrak{m}_5}^2)\omega^2 + ({\mathfrak{m}_3}^2 - {\mathfrak{m}_6}^2) = 0$$

$$\Rightarrow x^3 + \beta_1 x^2 + \beta_2 x + \beta_3 = 0 \qquad (5.16)$$

Were, $\beta_1 = {\mathfrak{m}_1}^2 - {\mathfrak{m}_4}^2 - 2\mathfrak{m}_2, \beta_2 = {\mathfrak{m}_2}^2 - 2\mathfrak{m}_1\mathfrak{m}_3 + 2\mathfrak{m}_4\mathfrak{m}_6 - {\mathfrak{m}_5}^2, \beta_3 = {\mathfrak{m}_3}^2 - {\mathfrak{m}_6}^2, x = \omega^2$

Now $\beta_3 = {\mathfrak{m}_3}^2 - {\mathfrak{m}_6}^2 = \left(\alpha_5\ (2r\alpha_1 S^* - r\alpha_1)\right)^2 - \left(\alpha_5(\alpha_1 S^* - 2\alpha_1 {S^*}^2 + rI^*)\right)^2$

$\beta_3 = -{\alpha_5}^2{\alpha_1}^2 r^2(r-1)(5r-3)$ as $\Re_0 > 1 \Rightarrow r < 1$, when $r < \frac{3}{5}$, we have $\beta_3 < 0$

Therefore, there exist only one pair of purely complex roots of the equation (5.16) say $x = \mp i\omega_0$, At the same time, by substituting $\omega = \omega_0$ in

(5.14) & (5.15) and solving it for the delay parameter τ, we have;

$$\tau_p = \frac{1}{\omega_0}\left\{arcsin\frac{\mathfrak{m}_5\omega_0(\mathfrak{m}_1(\omega_0)^2 - \mathfrak{m}_3) - ((\omega_0)^3 - \mathfrak{m}_2\omega_0)(\mathfrak{m}_6 - \mathfrak{m}_4(\omega_0)^2)}{(\mathfrak{m}_5\omega_0)^2 + (\mathfrak{m}_6 - \mathfrak{m}_4(\omega_0)^2)^2} + 2l\pi\right\},$$

$l = 0,1,2,3, \ldots\ldots$ (5.17)

Hence by the lemma in (Tipsri & Chinviriyasit, 2015), all the zeros of (5.7) has -ve real parts for $\tau \in [0, \tau_0)$, so we have, for the bifurcation analysis, we will show that there exist at least one eigen value who has positive real part for $\tau > \tau_0$, and $\left(\frac{dRe\lambda(\tau)}{d\tau}\right)_{\tau=\tau_0} \neq 0$

By differentiating equation (5.7) w.r.t $'\tau'$ and after rearranging the terms we have

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{(3\lambda^2+2m_1\lambda+m_2)e^{\lambda\tau}}{\lambda(m_4\lambda^2+m_5\lambda+m_6)} + \frac{(2m_4\lambda+m_5)}{\lambda(m_4\lambda^2+m_5\lambda+m_6)} - \frac{\tau}{\lambda}$$

$$\text{sign}\left\{\left(\frac{dRe\lambda(\tau)}{d\tau}\right)_{\tau=\tau_0}\right\} = \text{sign}\left\{Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\Big|_{\lambda=i\omega_0}\right\} =$$

$$\text{sign}\left\{Re\left(\frac{(3\lambda^2+2m_1\lambda+m_2)e^{\lambda\tau}}{\lambda(m_4\lambda^2+m_5\lambda+m_6)}\right) + Re\left(\frac{(2m_4\lambda+m_5)}{\lambda(m_4\lambda^2+m_5\lambda+m_6)}\right)\right\}$$

$$= \text{sign}\left\{\frac{3\omega_0{}^6+(m_1{}^2-m_4{}^2-2m_2)\omega_0{}^4+(2m_2{}^2-2m_1m_3+2m_4m_6-m_5{}^2)\omega_0{}^2}{(m_5\omega_0)^2+(m_6-m_4(\omega_0)^2)^2}\right\}$$

$$= \text{sign}\left\{\frac{3\omega_0{}^6+(\beta_1)\omega_0{}^4+(\beta_2)\omega_0{}^2}{(m_5\omega_0)^2+(m_6-m_4(\omega_0)^2)^2}\right\} = \text{sign}\left\{\frac{\omega_0{}^2(3\omega_0{}^4+(\beta_1)\omega_0{}^2+\beta_2)}{(m_5\omega_0)^2+(m_6-m_4(\omega_0)^2)^2}\right\}$$

Because $r > 0$, and when $r < \frac{3}{5}$, $\beta_2 > 0$, and also if $r < \frac{1}{2}$, that is if $\Re_0$>2, both $\beta_1 > 0, \beta_2 > 0$, so $\frac{\omega_0{}^2(3\omega_0{}^4+(\beta_1)\omega_0{}^2+\beta_2)}{(m_5\omega_0)^2+(m_6-m_4(\omega_0)^2)^2} > 0$, $\Rightarrow \left(\frac{dRe\lambda(\tau)}{d\tau}\right)_{\tau=\tau_0} > 0$, which ensure that the conditions for transversality holds and Hopf-Bifurcation occurs at $E^*\left(r, \alpha_1 r(\Re_0 - 1), \frac{\alpha_3\alpha_1 r(\Re_0-1)}{\alpha_5}\right)$, from the above, we draw the following conclusions;

Theorem 5.3: For the non-zero endemic positive equilibrium

$E^*\left(r, \alpha_1 r(\Re_0 - 1), \frac{\alpha_3\alpha_1 r(\Re_0-1)}{\alpha_5}\right)$, The following is what we've found

(a) If $1 < \Re_0 \leq 2$, holds, then the equilibrium $E^*\left(r, \alpha_1 r(\Re_0 - 1), \frac{\alpha_3\alpha_1 r(\Re_0-1)}{\alpha_5}\right)$ is asymptotical stable.

(b) If $\Re_0 > 2$, then the equilibrium $E^*\left(\mathcal{r}, \alpha_1\mathcal{r}(\Re_0 - 1), \frac{\alpha_3\alpha_1 r(\Re_0 - 1)}{\alpha_5}\right)$ is stable but conditionally, i.e when we have a delay critical parameter value τ_0, the equilibrium $E^*\left(\mathcal{r}, \alpha_1\mathcal{r}(\Re_0 - 1), \frac{\alpha_3\alpha_1 r(\Re_0 - 1)}{\alpha_5}\right)$ is asymptotical stable if $\tau \in [0, \tau_0)$, and when $\tau > \tau_0$ and system gives Hopf-Bifurcation at $E^*\left(\mathcal{r}, \alpha_1\mathcal{r}(\Re_0 - 1), \frac{\alpha_3\alpha_1 r(\Re_0 - 1)}{\alpha_5}\right)$, further more at $\tau = \tau_p, p = 1,2,3 \ldots\ldots$

5.9 Direction and stability analysis of Hopf-Bifurcation

In the last section, at the critical point values, a set of recurrent solutions obtained which bifurcates from the positive steady state E*. It's also worth looking at the direction, stability, and period of these bifurcating periodic solutions. Using the normal form theory and manifold reduction proposed by K. R. Schneider, Hassard, B. D. (Schneider, 1982), an explicit formula for finding the characteristics of the Hopf-bifurcation at the critical value $\tau_j$will be derived in this section.

Let $v_1 = S(t\tau) - S^*(t), v_2 = I(t\tau) - I^*(t), v_3 = R(t\tau) - R^*(t)$ and the delay τ can be normalizing by time scaling $t \to \frac{t}{\tau}$, the system of equations (2.8) are transformed into

$$\begin{aligned}
\frac{dv_1}{dt} &= (\alpha_1 - 2\alpha_1 S^*)v_1 - \alpha_1 {v_1}^2 + (\alpha_1 S^* - \alpha_1 {S^*}^2) - (I^* v_1 + S^* v_2 + v_1 v_2 + I^* S^*)(t-1) \\
\frac{dv_2}{dt} &= (I^* v_1 + S^* v_2 + v_1 v_2 + I^* S^*)(t-1) - \mathcal{r}(v_2 + I^*) \\
\frac{dv_3}{dt} &= \alpha_3(v_2 + I^*) - \alpha_5(v_3 + R^*)
\end{aligned} \tag{5.18}$$

Thus, in the phase $\mathfrak{D} = \mathfrak{D}\left([-1,0], {\mathcal{R}_+}^3\right)$ work can be done. Without losing of the generality, we denote the critical value τ_j by τ_0. Let $\tau = \tau_0 + \mu$, then the system of equations (5.18) has $\mu = 0$ a Hopf-bifurcation value. Rewrite this system as follows for ease of notation:

$$v'(t) = L_\mu(v_t) + F(\mu, v_t) \tag{5.19}$$

Where $v(t) = (v_1(t), v_2(t), v_3(t))^T \in \mathcal{R}^3, v_t(\theta) \in \mathfrak{D}$ is defined by $v_t(\theta) = v_t(\mathrm{t} + \theta)$, and

$L_\mu: \mathfrak{D} \to \mathcal{R}$, $F: \mathcal{R} \times \mathfrak{D} \to \mathcal{R}$ are given, respectively by

$$L_\mu \emptyset = (\tau_0 + \mu) \begin{bmatrix} (\alpha_1 - 2\alpha_1 S^*) & 0 & 0 \\ 0 & -\mathcal{r} & 0 \\ 0 & \alpha_3 & -\alpha_5 \end{bmatrix} \begin{bmatrix} \emptyset_1(0) \\ \emptyset_2(0) \\ \emptyset_3(0) \end{bmatrix} + (\tau_0 + \mu) \begin{bmatrix} -I^* & -S^* & c \\ I^* & S^* & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \emptyset_1(-1) \\ \emptyset_2(-1) \\ \emptyset_3(-1) \end{bmatrix}$$

And $F(\mu, \emptyset) = (\tau_0 + \mu) \begin{bmatrix} F_1 \\ F_2 \\ F_3 \end{bmatrix}$ respectively where $F_1 = -\alpha_1 \emptyset^2{}_1(0) - \emptyset_1(-1)\emptyset_2(-1), F_2 = -\emptyset_1(-1)\emptyset_2(-1), F_3 = 0$, $\emptyset(\theta) = \big(\emptyset_1(\theta), \emptyset_2(\theta), \emptyset_3(\theta)\big)^T \in \mathfrak{D}\big((-1,0), \mathcal{R}\big)$.

By representation theorem given by F. Riesz (Fuglede, 1955), there exist a function $\eta(\theta, \mu)$ of bounded variation for $\theta \in [-1,0]$, such that $L_\mu \emptyset = \int_{-1}^{0} d\,\eta(\theta, 0)\emptyset(\theta)$ for $\emptyset \in \mathfrak{D}$. In fact, choose

$$L_\mu \emptyset = (\tau_0 + \mu) \begin{bmatrix} (\alpha_1 - 2\alpha_1 S^*) & 0 & 0 \\ 0 & -\mathcal{r} & 0 \\ 0 & \alpha_3 & -\alpha_5 \end{bmatrix} \delta(\theta) + (\tau_0 + \mu) \begin{bmatrix} -I^* & -S^* & c \\ I^* & S^* & 0 \\ 0 & 0 & 0 \end{bmatrix} \delta(\theta + 1)$$

Here δ represent the Dirac delta function. For $\emptyset \in \mathfrak{D}\big([-1,0], \mathcal{R}_+{}^3\big)$, The system (5.19) is equivalent to

$$v'(t) = \mathcal{A}(\mu) v_t + \mathcal{F}(\mu) v_t \qquad (5.20)$$

For $\psi \in \mathfrak{D}^1\big([-1,0], \mathcal{R}_+{}^3\big)$, define

$$\mathcal{A}^*\psi(s) = \begin{cases} -\frac{d\psi(s)}{ds}, & s \in [-1,0) \\ \int_{-1}^{0} d\,\eta^T(-t,0)\psi(-t), & s = 0. \end{cases}$$ And bilinear inner product $< \psi(s), \emptyset(\theta) > = \overline{\psi(0)}\emptyset(0) - \int_{-1}^{0}\int_{\xi=\theta}^{\theta} \overline{\psi}(\xi-\theta)d\eta(\theta)\phi(\xi)\,d\xi$ (5.21)

Since $i\omega_0$ are eigen values of $\mathcal{A}(0)\ and\ \mathcal{A}^*$,$\mathcal{A} = \mathcal{A}(0)$ are adjoint operators. Thus they become the eigen values of$\mathcal{A}^*$.Suppose that $q(\theta) = q(0)e^{i\omega_0\theta}$ is an eigen vector of $\mathcal{A}(0)$ corresponding to the eigen value $i\omega_0$. Then $\mathcal{A}(0) = i\omega_0\, q(\theta)$.

When $\theta = 0$, $\left[i\omega_0 I - \int_{-1}^{0} d\eta(\theta)e^{i\omega_0\theta}\right]q(0) = 0$, this gives $q(0) = (1, u_1, u_2)^T$ where

$u_1 = \frac{i\omega_0-(\alpha_1-2\alpha_1 S^*)}{(i\omega_0+r)}$ $and\ u_2 = \frac{\alpha_3(i\omega_0-(\alpha_1-2\alpha_1 S^*))}{(i\omega_0+r)(i\omega_0+\alpha_5)}$

By the same way it can be proved that $q^*(s) = D(1, {u_1}^*, {u_2}^*)e^{i\omega_0\tau_0 s}$ represent the eigen value of $\mathcal{A}^*$ corresponding to eigen vector $-i\omega_0$, where ${u_1}^* = \frac{-i\omega_0-(\alpha_1-2\alpha_1 S^*)}{(-i\omega_0+r)}$ $and\ {u_2}^* = \frac{\alpha_3(-i\omega_0-(\alpha_1-2\alpha_1 S^*))}{(-i\omega_0+r)(i-\omega_0+\alpha_5)}$ The value of D is required to assure that $< q^*(s), q(\theta) > = 1$,

From equation (5.21) we have,

$< q^*(s), q(\theta) > = \overline{D}\left(1, \overline{{u_1}^*}, \overline{{u_2}^*}\right)(1, u_1, u_2)^T -$

$\int_{-1}^{0}\int_{\xi=\theta}^{\theta} \overline{D}\left(1, \overline{{u_1}^*}, \overline{{u_2}^*}\right)e^{-i\omega_0\tau_0(\xi-\theta)}d\eta(\theta)(1, u, u)^T e^{i\omega_0\tau_0}\,d\xi$

$= \overline{D}\left\{1 + u_1\overline{u_1}^* + u_2\overline{{u_2}^*} - \int_{-1}^{0}\left(1, \overline{{u_1}^*}, \overline{{u_2}^*}\right)\theta e^{i\omega_0\tau_0\theta}(1, u, u)^T\right\}$

$= \overline{D}\left\{1 + u_1\overline{u_1}^* + u\overline{{u_2}^*} + \tau_0\overline{u^*}W^*(u-\alpha_1 u)e^{i\omega_0\tau_0}\right\}$

Hence, choose $\overline{D} = \frac{1}{\left\{1+u_1\overline{u_1}^*+u\overline{{u_2}^*}+\tau_0\overline{u^*}W^*(u-\alpha_1 u)e^{i\omega_0\tau_0}\right\}}$

such that $< q^*(s), q(\theta) > = 1, < q^*(s), \overline{q(\theta)} > = 0$.

By following (Schneider, 1982) approach we compute the coordinates describing the center manifold $\mathfrak{D}_0$ at $\mu = 0$ by using the same notations. manifold $\mathfrak{D}_0$ at $\mu = 0$. Let equation (5.19) has solution u_t with $\mu = 0$. We Define $z(t) =< q^*(s), v_t(\theta) >$,

$$\mathrm{W}(t,\theta) = v_t(\theta) - 2Re(z(t)q(\theta)) \qquad (5.22)$$

On the center manifold $\mathbb{C}_0$, $\mathrm{W}(t,\theta) = \mathrm{W}\left(z(t), \overline{z(t)}, \theta\right)$ In the direction of q^* and $\overline{q^*}$ the local coordinates for center manifold $\mathbb{C}_0$ are z and $\overline{z}$. Note that u_t is real is the necessary condition for W is real. For solution $u_t \in \mathfrak{D}_0$ of equation (5.20), since $\mu = 0$, $z'(t) = i\omega_0\tau_0 z + < \overline{q^*}(\theta), F\big(0, W(z,\overline{z},\theta) + 2Re\big(z(t)q(\theta)\big)\big) > \; = i\omega_0\tau_0 z + \overline{q^*}(0)\, F\big(0, \mathrm{W}(z,\overline{z},0) + 2Re\big(z(t)q(\theta)\big)\big)$ $\equiv i\omega_0\tau_0 z + \overline{q^*}(0)F_0(z,\overline{z})$

Rewrite this equation as: $z'(t) = i\omega_0\tau_0 z(t) + g(z,\overline{z})$ (5.23)

Where $g(z,\overline{z}) = \overline{q^*}(0)F_0(z,\overline{z}) = g_{20}(\theta)\frac{z^2}{2} + g_{11}(\theta)z\overline{z} + g_{02}(\theta)\frac{\overline{z}^2}{2} + g_{21}(\theta)\frac{z^2\overline{z}}{2} + \cdots$ (5.24)

As $v_t(\theta) = (v_{1t}, v_{2t}, v_{3t}) = \mathrm{W}(t,\theta) + z\,q(\theta) + \overline{z}\overline{q(\theta)}$ and $q(0) = (1, u_1, u_2)^T \mathrm{e}^{\mathrm{i}\omega_0\tau_0\theta}$, so

$v_{1t}(0) = z + \overline{z} + \mathrm{W}_{20}{}^{(1)}(0)\frac{z^2}{2} + \mathrm{W}_{11}{}^{(1)}(0)\, z\overline{z} + \mathrm{W}_{02}{}^{(1)}(0)\frac{\overline{z}^2}{2} + \cdots,$

$v_{2t}(0) = u_1 z + \overline{u_1}\,\overline{z} + \mathrm{W}_{20}{}^{(2)}(0)\frac{z^2}{2} + \mathrm{W}_{11}{}^{(2)}(0)\, z\overline{z} + \mathrm{W}_{02}{}^{(2)}(0)\frac{\overline{z}^2}{2} + \cdots,$

$v_{3t}(0) = u_{2_1} z + \overline{u_{21}}\,\overline{z} + \mathrm{W}_{20}{}^{(3)}(0)\frac{z^2}{2} + \mathrm{W}_{11}{}^{(3)}(0)\, z\overline{z} + \mathrm{W}_{02}{}^{(3)}(0)\frac{\overline{z}^2}{2} + \cdots,$

$v_{1t}(-1) = ze^{-i\omega_0\tau_0} + \overline{z}e^{i\omega_0\tau_0} + \mathrm{W}_{20}{}^{(1)}(-1)\frac{z^2}{2} + \mathrm{W}_{11}{}^{(1)}(-1)\, z\overline{z} + \mathrm{W}_{02}{}^{(1)}(-1)\frac{\overline{z}^2}{2} + \cdots,$

$v_{2t}(-1) = ue^{-i\omega_0\tau_0}z + \overline{u_1}\, e^{i\omega_0\tau_0}\,\overline{z} + \mathrm{W}_{20}{}^{(2)}(-1)\frac{z^2}{2} +$

$\mathrm{W}_{11}{}^{(2)}(-1)\, z\overline{z} + \mathrm{W}_{02}{}^{(2)}(-1)\frac{\overline{z}^2}{2} + \cdots,$

As a result of comparing coefficients with equation (5.24) we get: $g_{20} = \overline{D}(1, u_1, u_2)f_{z^2},\ g_{02} = \overline{D}\big(1, \overline{u_1}, \overline{u_2{}^*}\big)f_{\overline{z}^2},\ g_{11} = \overline{D}\big(1, \overline{u_1}, \overline{u_2{}^*}\big)f_{z\overline{z}},\ g_{21} = \overline{D}\big(1, \overline{u_1}, \overline{u_2{}^*}\big)f_{z^2\overline{z}}$

In order to find the value of g_{21}, the computation of $\mathrm{W}_{20}(\theta)$ and $\mathrm{W}_{11}(\theta)$ should be prioritized. From the equations (5.20) and (5.22) we have;

$W' = v_t{}' - z'q - \overline{z}'q =$

$$\begin{cases} \mathcal{A}\mathrm{W} - 2Re\big[\overline{q^*}(0)F_0q(\theta)\big], & \theta \in [-1,0) \\ \mathcal{A}\mathrm{W} - 2Re\big[\overline{q^*}(0)F_0q(0)\big] + F_0, & \theta = 0 \end{cases}$$

Let $\mathrm{W}' = \mathcal{A}\mathrm{W} + H(z, \overline{z}, \theta)$ (5.25)

Where$H(z, \overline{z}, \theta) = H_{20}(\theta)\frac{z^2}{2} + H_{11}(\theta)z\overline{z} + H_{02}(\theta)\frac{\overline{z}^2}{2} + H_{21}(\theta)\frac{z^2\overline{z}}{2} +$ …. (5.26)

However, on the other hand, on $\mathfrak{D}_0$ near the origin $\mathrm{W}' = \mathrm{W}_z z' + \mathrm{W}_z\overline{z}'$

Simplifying the above series and equating the coefficients, we get

$[\mathcal{A} - 2i\omega_0 I]\mathrm{W}_{20}(\theta) = -H_{20}(\theta),\ \mathcal{A}\mathrm{W}_{11}(\theta) = -H_{11}(\theta)$ (5.27)

By equation (2.1), for $\theta \in [-1,0)$,

$H(z, \overline{z}, \theta) = -\overline{q^*}(0)\overline{F_0}q(\theta) - \overline{q^*}(0)\overline{F_0}\,\overline{q}(\theta) = -gq(\theta) - \overline{g}\,\overline{q}(\theta)$

Comparing the coefficients with (2.3) for $\theta \in [-1,0]$ that

$H_{20}(\theta) = -g_{20}q(\theta) - \overline{g_{02}}\,\overline{q}(\theta),\ H_{11}(\theta) = -g_{11}q(\theta) - \overline{g_{11}}\,\overline{q}(\theta).$

From equations (5.23), (5.26) and the definition of A we obtain

$\mathrm{W}_{20}(\theta) = 2i\omega_0\tau_0\mathrm{W}_{20}(\theta) + g_{20}q(\theta) + \overline{g_{02}}\,\overline{q}(\theta)$

Solving for $W_{20}(\theta)$: $W_{20}(\theta) = \frac{ig_{20}}{\omega_0\tau_0} q(0)e^{i\omega_0\tau_0\theta} + \frac{i\overline{g_{02}}}{3\omega_0\tau_0}\overline{q}(0)e^{-i\omega_0\tau_0\theta} + E_1 e^{2i\omega_0\tau_0\theta}$,

And similarly, $W_{11}(\theta) = \frac{-ig_{11}}{\omega_0\tau_0} q(0)e^{i\omega_0\tau_0\theta} + \frac{i\overline{g_{11}}}{\omega_0\tau_0}\overline{q}(0)e^{-i\omega_0\tau_0\theta} + E_2$

The three-dimensional vectors E_1 and E_2, can be determined by setting $\theta = 0$ in H. In fact since $H(z,\overline{z},\theta) = -2Re\left[\overline{q^*}(0)F_0 q(0)\right] + F_0$, So

$H_{20}(\theta) = -g_{20}q(\theta) - \overline{g_{02}}\,\overline{q}(\theta) + F_{z^2}$, $H_{11}(\theta) = -g_{11}q(\theta) - \overline{g_{11}}\,\overline{q}(\theta) + F_{z\overline{z}}$

Where $F_0 = F_{z^2}\frac{z^2}{2} + F_{z\overline{z}}z\overline{z} + F_{\overline{z}^2}\frac{\overline{z}^2}{2} + \cdots$ Hence combining the definition of $\mathcal{A}$,

$\int_{-1}^{0} d\,\eta(\theta)W_{20}(\theta) = 2i\omega_0\tau_0 W_{20}(0) + g_{20}q(0) + \overline{g_{02}}\,\overline{q}(0) - F_{z^2}$ and $\int_{-1}^{0} d\,\eta(\theta)W_{11}(\theta) = g_{11}q(0) - \overline{g_{11}}\,\overline{q}(0) - F_{z\overline{z}}$.

Notice that $\left[i\omega_0\tau_0 I - \int_{-1}^{0} e^{i\omega_0\tau_0\theta}d\eta(\theta)\right]q(0) = 0$ and $\left[-i\omega_0\tau_0 I - \int_{-1}^{0} e^{-i\omega_0\tau_0\theta}d\eta(\theta)\right]\overline{q}(0) = 0$, Which Implies $\left[2i\omega_0\tau_0 I - \int_{-1}^{0} e^{2i\omega_0\tau_0\theta}d\eta(\theta)\right]E_1 = F_{z^2}$ and $-\left[\int_{-1}^{0} d\eta(\theta)\right]E_2 = F_{z\overline{z}}$

Hence,

$$\begin{bmatrix} \left(2i\omega_0 - (\alpha_1 - 2\alpha_1 S^*) + I^* e^{-2i\omega_0\tau_0}\right) & S^* e^{-2i\omega_0\tau_0} & -ce^{-2i\omega_0\tau_0} \\ -I^* e^{-2i\omega_0\tau_0} & \left(2i\omega_0 + \varkappa - S^* e^{-2i\omega_0\tau_0}\right) & 0 \\ 0 & -\alpha_3 & (2i\omega_0 + \alpha_5) \end{bmatrix} E_1 = -2\begin{bmatrix} u_1 e^{-i\omega_0\tau_0\theta} \\ -u_1 e^{-i\omega_0\tau_0\theta} \\ 0 \end{bmatrix}$$

and

$$\begin{bmatrix} \left(-(\alpha_1 - 2\alpha_1 S^*) + I^* e^{-2i\omega_0\tau_0}\right) & S^* e^{-2i\omega_0\tau_0} & -ce^{-2i\omega_0\tau_0} \\ -I^* e^{-2i\omega_0\tau_0} & \left(\curlyvee - S^* e^{-2i\omega_0\tau_0}\right) & 0 \\ 0 & -\alpha_3 & (\alpha_5) \end{bmatrix} E_2 =$$

$$-2\begin{bmatrix} Re\{u_1\}e^{i\omega_0\tau_0\theta} \\ -Re\{u_1\}e^{i\omega_0\tau_0\theta} \\ 0 \end{bmatrix}$$

The arguments can thus be used to express g_{21} the parameters. Based on the above study, every g_{ij} can be find out by the parameters. As a result, the following quantities can be calculated:

$$\mathbb{C}_1(0) = \frac{i}{2\omega_0\tau_0}\left(g_{11}g_{20} - 2|g_{11}|^2 - \frac{|g_{02}|^2}{3}\right) + \frac{g_{21}}{2} \quad , \quad \mu_2 = -\frac{Re\{\mathbb{C}_1(0)\}}{Re\{\lambda'(\tau_0)\}}, \quad \beta_2 = 2Re\{\mathbb{C}_0(0)\}, \; T_2 = -\frac{Im\{\mathbb{C}_1(0)\} + \mu_2 Im\{\lambda'(\tau_0)\}}{\omega_0\tau_0} \qquad (5.28)$$

Theorem 5.4: The direction of the Hopf-bifurcation is calculated by the value of μ_2: if $\mu_2 > 0 (\mu_2 < 0)$, then the Hopf bifurcation is supercritical (subcritical) and the bifurcating periodic solutions exist for $\tau > \tau_0$ $(\tau < \tau_0)$. The stability of bifurcating solutions is calculated by the value of β_2, the bifurcating recurring solutions are orbitally asymptotically stable (unstable) if $\beta_2 < 0$ $(\beta_2 > 0)$. The value of T_2 defines the period of the bifurcating periodic solutions, the period increases (decreases) if $T_2 > 0$ $(T_2 < 0)$.

5.10 Numerical Simulation and Discussion

The purpose of this section is to provide some numerical simulations in order to show the theoretical findings that were gained from this work. By using MATLAB, we will also demonstrate the analytical findings by displaying various bifurcation diagrams, and we will investigate a novel dynamical behavior that occurs when the parameters are altered. For the goal of the aforementioned endeavour, we take into consideration the hypothetical and establish the values of the parameters in such a manner that they fulfil the requirements derived by analytical means in earlier parts.

With the set of the parameters $\mathcal{K} = 90,\ \alpha_1 = 0.145, \alpha_2 = 0.0045, \alpha_3 = 0.185, \alpha_4 = 0.25, \alpha_5 = 0.0347\ and\ \tau = 1$, we get $\Re_0 = 0.5179 < 1$.we have DFE which is absolute globally stable and the dynamics of the system (5.1) has been plotted in Figure 5.1 .

When $1 < \Re_0 = 1.597 \leq 2$, then the system (5.1) holds the asymptotic stability. the dynamics of the system are given by $\mathcal{K} = 150, \alpha_1 = 0.156, \alpha_2 = 0.051, \alpha_3 = 0.180, \alpha_4 = 0.2551, \alpha_5 = 0.0346$, Figure 5.2 When $\Re_0 > 2$ the dynamics of the system (2) is given by the following simulations,

For $\mathcal{K} = 20, \alpha_1 = 0.112, \alpha_2 = 0.0521, \alpha_3 = 0.0751, \alpha_4 = 0.0477, \alpha_5 = 0.0374$, by direct computation we get the basic reproduction number $\Re_0 = 6.616 > 1$

(a) When $\tau = 0$ the endemic equilibrium $E^*$is locally asymptotically stable and the equilibrium point is $E^*(3.1329,1.7642,3.5459)$ see the Figure 5.3 to Figure 5.6.

(b) When $\tau = 0.85 < \tau_c = 1.0125$ the endemic equilibrium is also asymptotically stable see Figure 5.7 & Figure 5.8.

(c) When $\tau = \tau_c = 1.0125$ the dynamics of the system shows hopf bifurcation see Figure 5.9 to Figure 5.12

(d) When $\tau = 1.27 > \tau_c = 1.0125$, then the system going to be super critical. See Figure 5.13 to Figure 5.16.

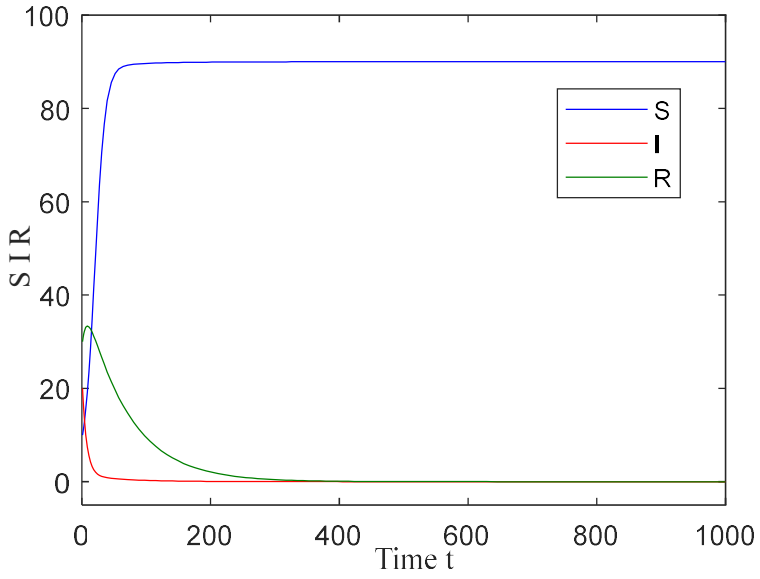

Figure 5.1 : When $1 < \mathfrak{R}_0 = 1.597 \leq 2$, then the system holds the asymptotic stability

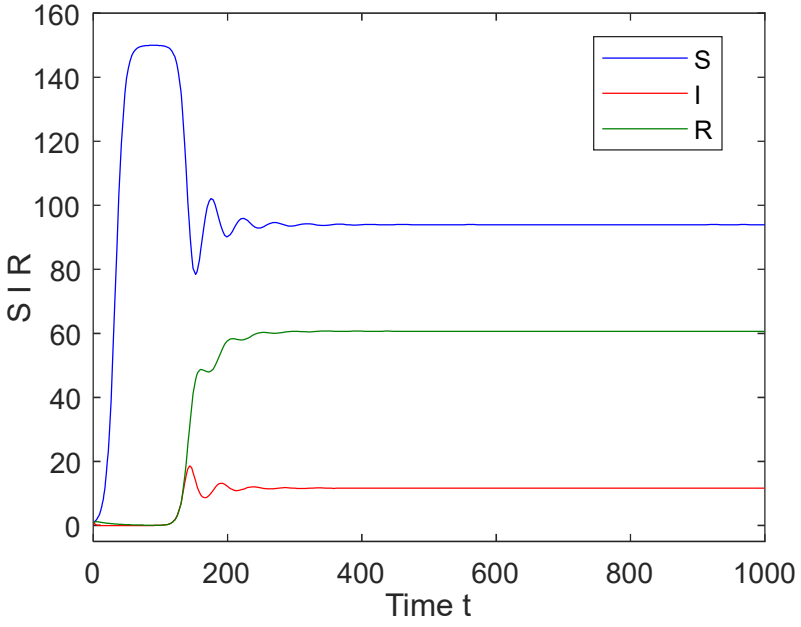

Figure 5.2 : When $\mathfrak{R}_0 > 2$ system has some limit cycle and then stable.

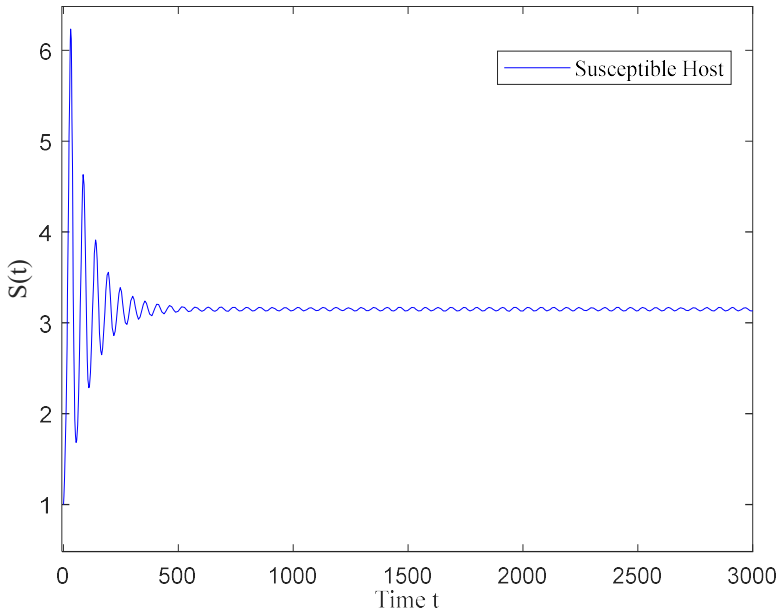

Figure 5.3 : When $\tau = 0$ the endemic equilibrium $E^*$is locally asymptotically stable for Susceptible host

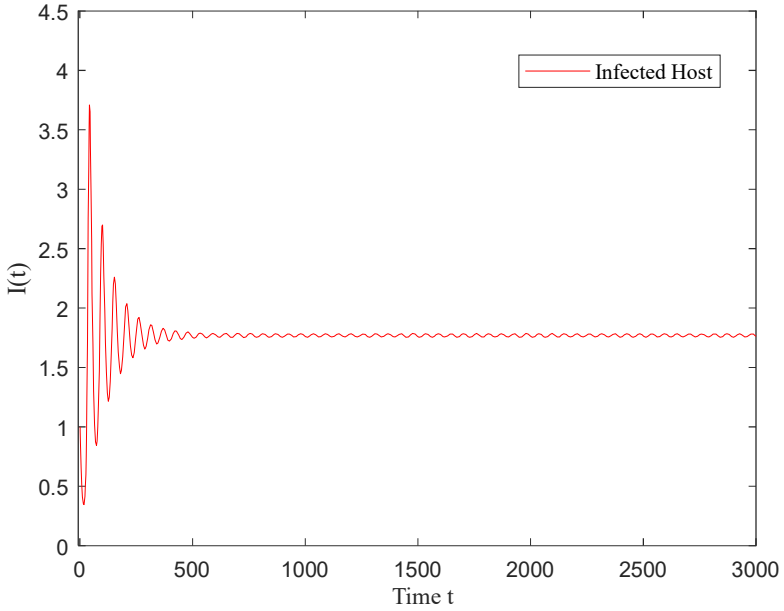

Figure 5.4 : When $\tau = 0$ the endemic equilibrium $E^*$is locally asymptotically stable for Infected host.

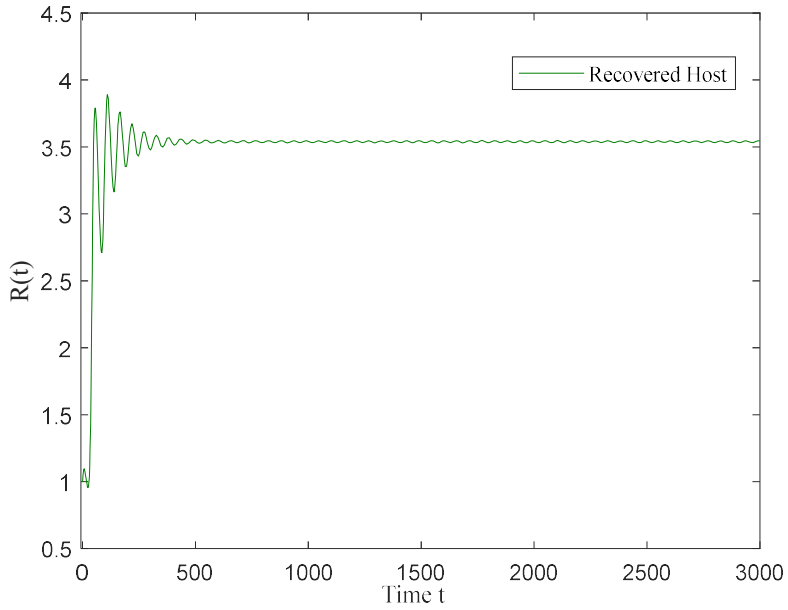

Figure 5.5 : When $\tau = 0$ the endemic equilibrium $E^*$is locally asymptotically stable for Recovered host.

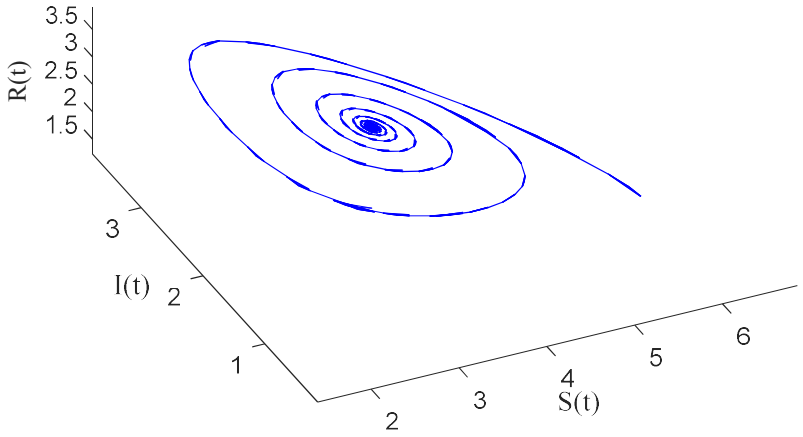

Figure 5.6 : Phase space view when $\tau = 0$ the endemic equilibrium $E^*$is locally asymptotically stable.

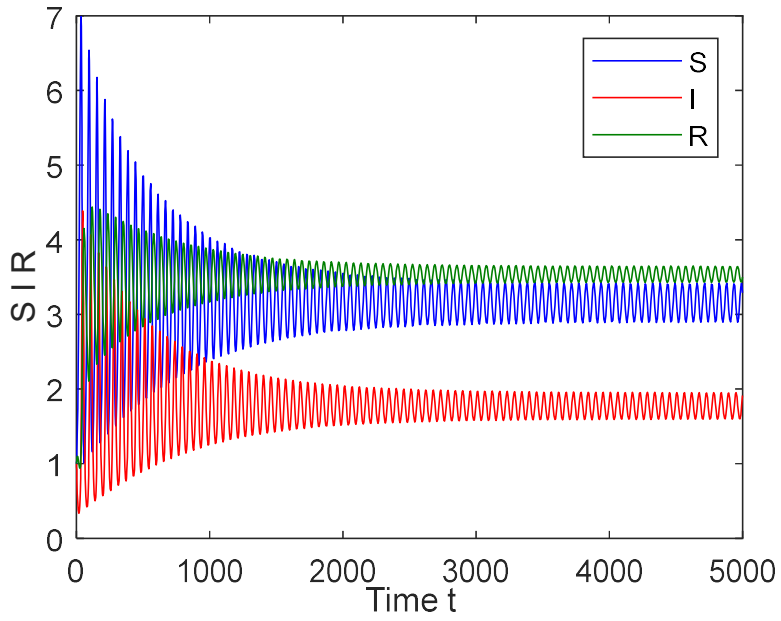

Figure 5.7 : When $\tau = 0.85 < \tau_c = 1.0125$ the endemic equilibrium is also asymptotically stable

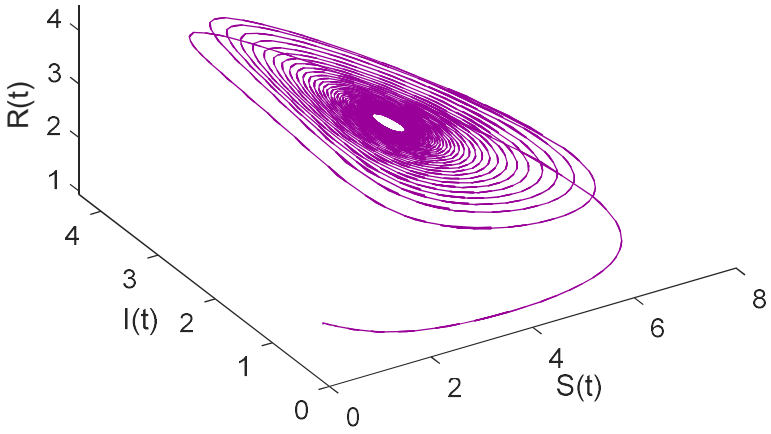

Figure 5.8 : Phase space view when $\tau = 0.85 < \tau_c = 1.0125$ the endemic equilibrium is also asymptotically stable

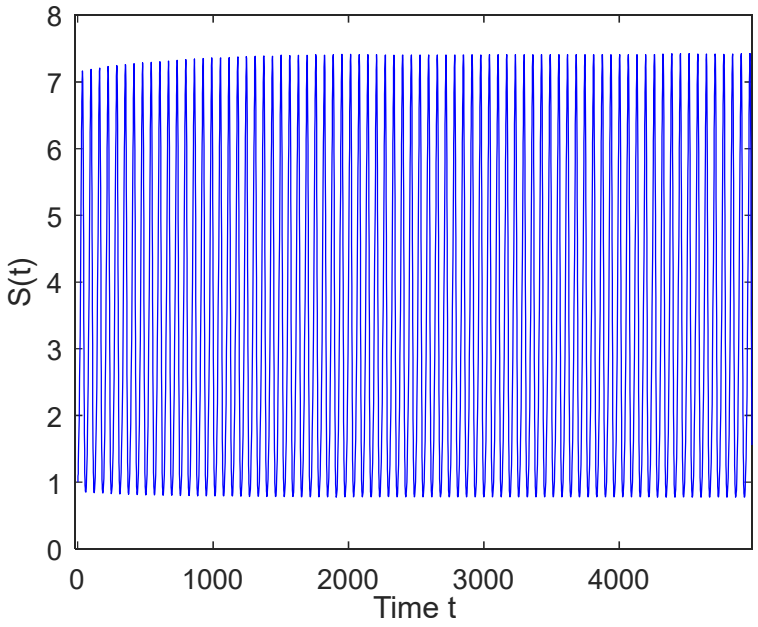

Figure 5.9 : When $\tau = \tau_c = 1.0125$ the dynamics of the system shows hopf bifurcation

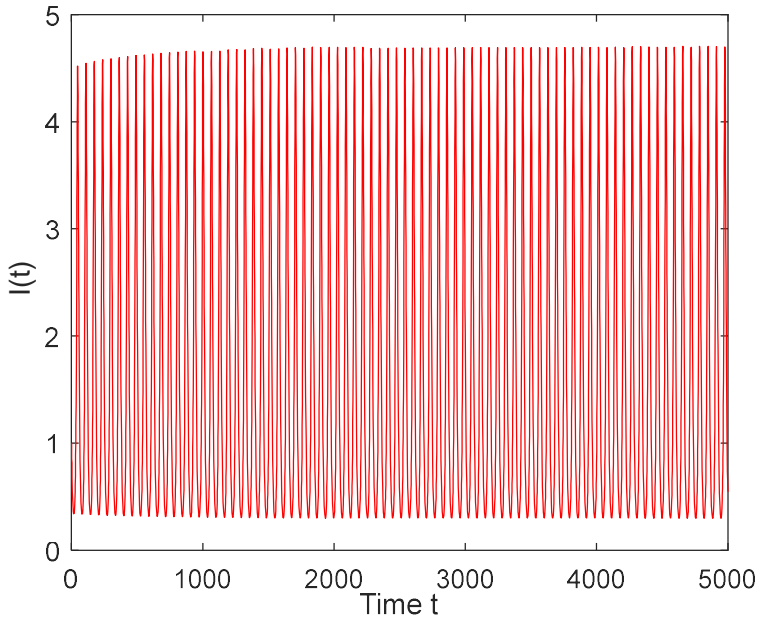

Figure 5.10 : When $\tau = \tau_c = 1.0125$ the dynamics of the system shows hopf bifurcation

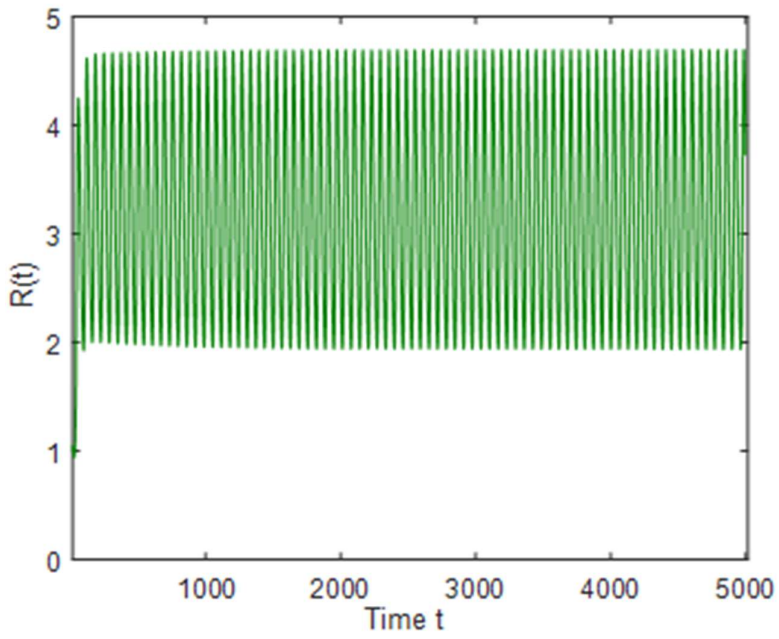

Figure 5.11 : When $\tau = \tau_c = 1.0125$ the dynamics of the system shows hopf bifurcation

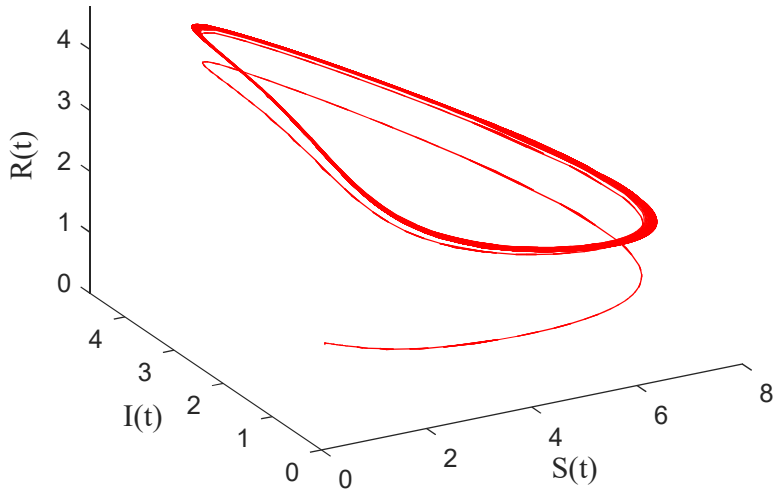

Figure 5.12 : Phase space view when $\tau = \tau_c = 1.0125$ the dynamics of the system shows hopf bifurcation

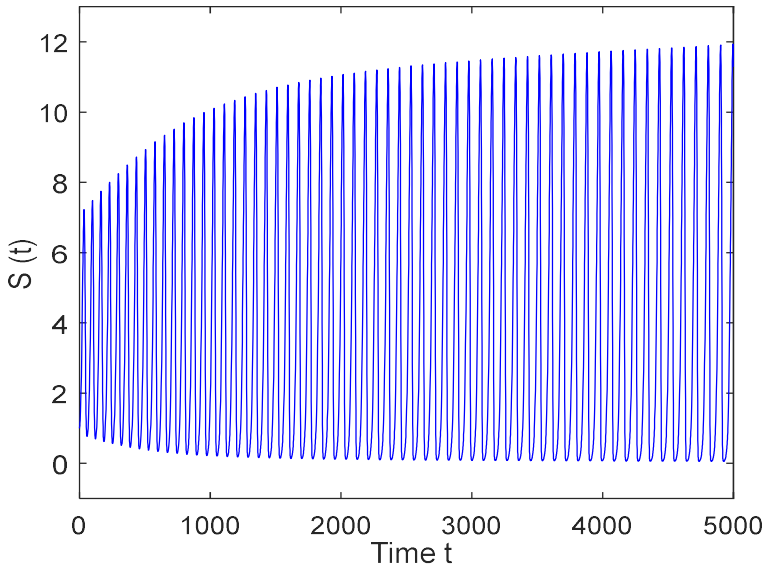

Figure 5.13 : When $\tau = 1.27 > \tau_c = 1.0125$, the system going to be super critical

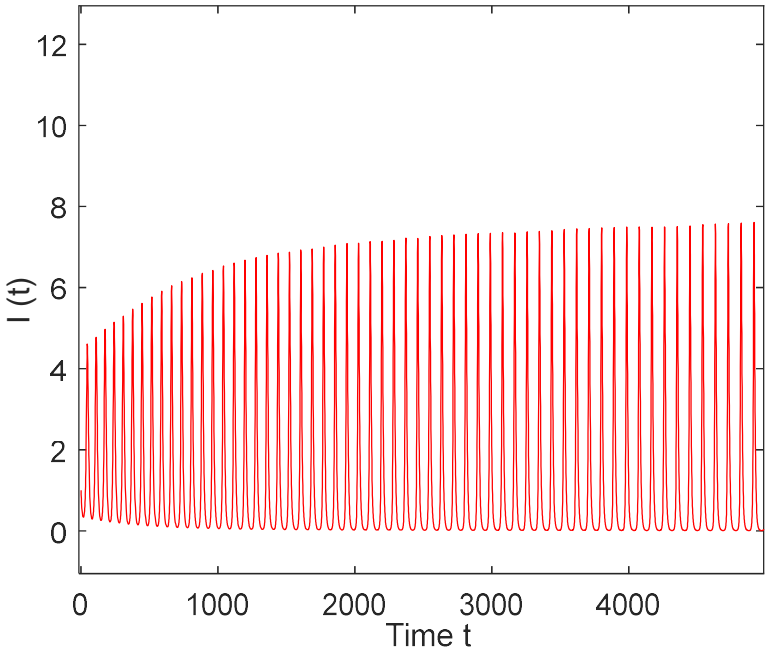

Figure 5.14 : When $\tau = 1.27 > \tau_c = 1.0125$, the system going to be super critical

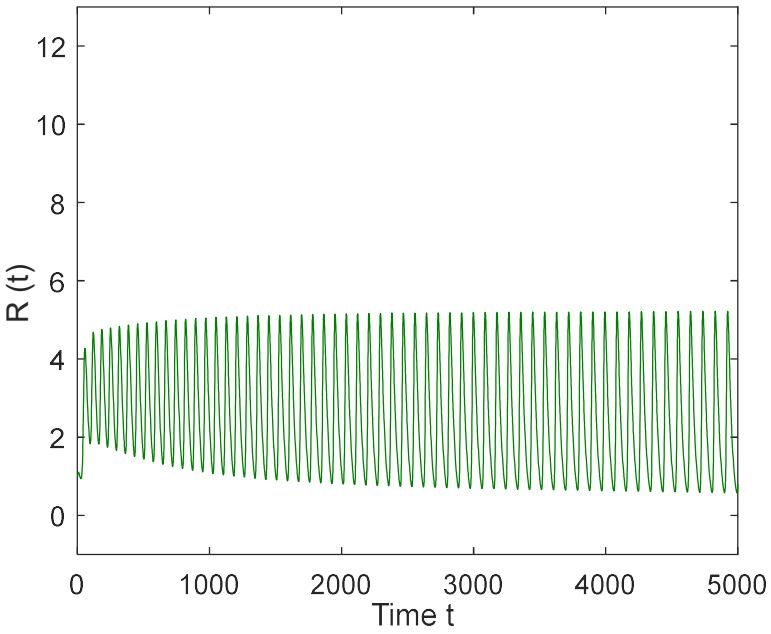

Figure 5.15 : When $\tau = 1.27 > \tau_c = 1.0125$, the system going to be super critical

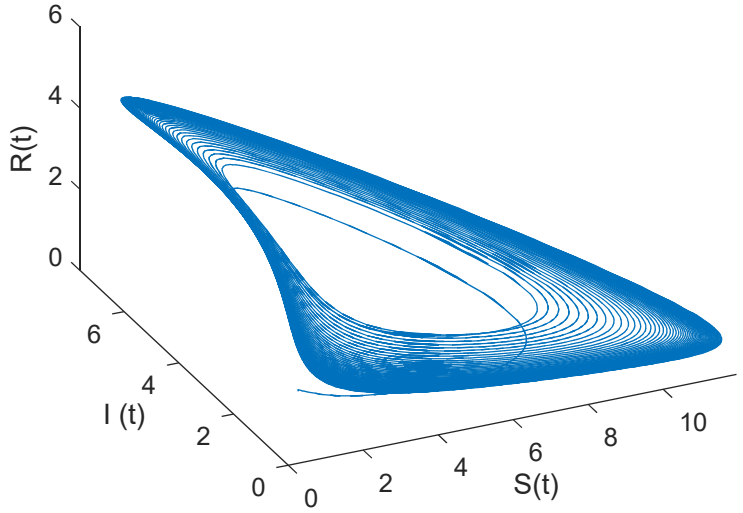

Figure 5.16 : Phase Space view when $\tau = 1.27 > \tau_c = 1.0125$, the system going to be super critical

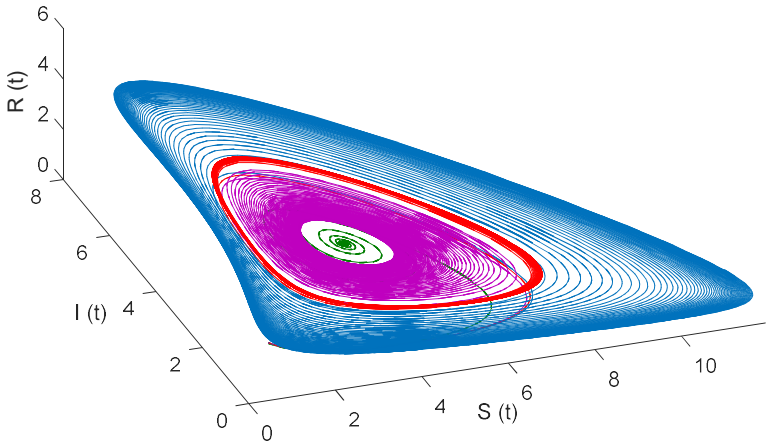

Figure 5.17: Phase Space view showing the dynamics of all the states of stability i.e., globally (Green), Asymptotic (purple), Hopf bifurcation (Red) and Super critical (Blue)

5.11 Conclusion

The global dynamic behaviors of a SIR model with incubation period are investigated in this work, and the susceptible population is shown to expand in a logistic fashion. There is a critical value of $\mathfrak{R}_0$ in the system (5.2) that controls how the disease behaves. The impact of incubation period on the dynamic behaviours of systems are the focus of this research. This chapter findings demonstrate that the proximity to an endemic value and the stability of solutions are dependent on two variables: the incubation period duration and the threshold value $\mathfrak{R}_0$. System (5.2) has global asymptotic stability in the disease-free equilibrium E^* if $\mathfrak{R}_0 = 1$. In the case where $\mathfrak{R}_0 > 1$, E^* is no longer stable, and the endemic equilibrium E^* is indeed irreversible. The requisites for achieving local stability and Hopf bifurcation at E^* were derived by treating the delay time as a parameter. If $1 < \mathfrak{R}_0 \leq 2$, then E^* is absolutely stable; that is, E^* is always locally stable when τ is non zero. When $\mathfrak{R}_0 > 2$, E^* is conditionally stable in the range $\tau \in (0, \tau_0)$, as soon as the value of τ surpasses the critical value τ_0 i.e., $\tau > \tau_0$, E^* become unstable and shows Hopf-bifurcations. This study shows that for short time delay in (5.2) has no effect on the positive invariance. Even the boundedness of solutions, the

global stability of DFE, or the permanence of the endemic equilibrium remains intact. However, for long delays, the time delay can destabilize the unique endemic equilibrium, and a variety of stable periodic solutions through Hopf bifurcation. Our theoretical findings are reflected in the results of the numerical simulations that were performed.

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List of Papers Published and Communicated from the Thesis

1. Singh, R. P. (2022). Bifurcation and Stability Analysis of Delayed SIR Model. *Journal of Physics: Conference Series*, *2267*(1), 012011. https://doi.org/10.1088/1742-6596/2267/1/012011. (Scopus Indexed with SJR-0.183)

2. Pankaj Kumar, Raminder Pal Singh. *Mathematical analysis of SIR Model with Virus Mutation using Delay Differential Equation,* Latin American Journal of Pharmacy,42(2): 294-308 (2023). **(**Scopus Indexed, Q4 with SJR – 0.126)

3. Pankaj Kumar, Raminder Pal Singh. *Analysis of Classic SIR Model using Delay Differential Equations,* [Under review]. Journal of mathematical and fundamental Science. (Scopus Indexed, Q3 with SJR – 0.18)

4. Pankaj Kumar, Raminder Pal Singh & Dipesh. *Modelling the Effect of Discrete Delay in SIR Epidemic Model with Logistic Growth,* [Under review] Advance in mathematical and Computational Sciences. (Scopus Indexed)

List of Conferences

1. Presented the paper titled **"**Bifurcation and Stability Analysis of Delayed SIR Model**"** in Recent Advances in Fundamental and Applied Sciences (RAFAS) June,2021 Organized by School of Chemical Engineering & Physical Sciences Lovely Professional University Punjab, India.

2. Presented the paper titled "Analysis of Classic SIR Model Using Delay Differential Equations" in Recent Advances In Fundamental And Applied Sciences (RAFAS) March,2023 Organized By School Of Chemical Engineering & Physical Sciences Lovely Professional University Punjab, India.