

**Analysis of Mathematical Model on the Transmission
Dynamics of COVID-19 with Protected and
Hospitalized individuals.**



MSc. Thesis

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Declaration

I hereby declare that the thesis entitled “**Analysis of Mathematical Model on the Transmission Dynamics of COVID-19 with Protected and Hospitalized individuals**” is my own original work and that all the source materials I have used have been properly indicated and acknowledged by complete references. I earnestly, declare that all or parts of this thesis have not been submitted to any other universities for the award of any academic degree, diploma or certificate.

Name of students

Signature

Date

Place: Hawassa University, Ethiopia

Date: November, 2024

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This is to certify that the thesis titled “**Analysis of Mathematical Model on the Transmission Dynamics of COVID-19 with Protected and Hospitalized individuals.**” submitted in partial fulfillment of the requirement for the degree of Master of Science in Mathematics to the Department of Mathematics, Hawassa University. The thesis is a record of original work carried out by Teshale Deboch Mathk/073/09 under my supervision and no part of the thesis has been submitted for another degree or diploma. The assistance and the help received during the course of this investigation have been duly acknowledged. Therefore, I recommended that it may be accepted as fulfilling the thesis requirement.

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We, the undersigned, members of the Board of examiners of the final open defense made by Teshale Deboch have read and evaluated his thesis entitled **“Analysis of Mathematical Model on the Transmission Dynamics of COVID-19 with Protected and Hospitalized individuals.”** and examined the candidate. This is therefore to certify that the thesis has been accepted in partial fulfillment of the requirement of the Degree of Master of Science in Mathematics.

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Final approval and acceptance of the thesis is contingent upon the submission of the final copy of the thesis to the school of graduate studies (SGS) through the department/School graduate committee (DGC/SGC) of the candidates department.

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List of Abbreviations

Abbreviations

Meaning

COVID -19

Corona Virus Disease 2019

DFE

Disease Free Equilibrium

EEP

Endemic Equilibrium Point

ODE

Ordinary Differential Equation

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Abstract

Mathematical modelling is important for better understanding of disease dynamics and developing strategies to manage rapidly spreading infectious diseases. In this thesis, we propose a mathematical model to investigate coronavirus diseases (COVID-19) transmission in the presence of protected and hospitalized classes. Analytical and numerical approach is employed to investigate the results. In the analytical study of the model, we have shown the local and global stability of disease-free equilibrium, existence of the endemic equilibrium and its local stability, positivity of the solution, invariant region of the solution and sensitivity analysis of the model is conducted. From these analyses, we found that the disease-free equilibrium is globally asymptotically stable for $R_0 < 1$ and is unstable for $R_0 > 1$. A locally stable endemic equilibrium exists for $R_0 > 1$, which shows the persistence of the disease if the reproduction number is greater than unity. Using sensitivity analysis we establish that R_0 is most sensitive to the rate of Protection of Susceptible individuals θ and that a high level of protection needs to be maintained as well as hospitalization to control the disease. Finally, we performed numerical simulations using MATLAB software Ode 45 codes to supplement the effectiveness of the analytical findings.

Keywords: *COVID-19, Protected, Hospitalized, Stability analysis and Sensitivity analysis.*

Chapter 1

Introduction

1.1 Backgrounds of the Study

Coronavirus disease 2019 (COVID-19) is an infectious disease caused by a newly discovered coronavirus [2]. The new virus was first appeared late December 2019 in the Chinese city of Wuhan and eventually invaded the world due to fast modern air transportation. The novel corona virus now referred to as COVID-19 is caused by severe acute respiratory syndrome (SARS-CoV-2) and consists of single-stranded ribonucleic acid (RNA) structure [3].

Symptoms of the disease may appear 2–14 days after exposure and may include fever, cough shortness of breath, chills, muscle pain and loss of taste or smell [7]. Many of these are common with influenza, however high persistent fever, dry cough and difficulty in breathing seem to characterize COVID-19 [8]. The symptoms may range from very mild (in 80% of the cases) to severe (in 15% of the cases) to critical (in 5% of the cases) [7]. Those at higher risk for severe illness include the elderly (people ages 65 and above) and people with underlying medical conditions which may include chronic lung diseases, diabetes, kidney diseases, serious heart conditions and immune compromised individuals [9].

Coronavirus (COVID-19) is an infectious disease caused by a novel coronavirus, which is a respiratory illness that can spread in a population in several different ways. A person can be infected when droplets containing the virus are inhaled or come directly into contact with the eyes, nose, or mouth. The novel coronavirus has been spreading worldwide starting from the first identification in December 2019. The World Health Organization (WHO) declared COVID-19 as pandemic on March 12, 2020. From the first day of the outbreak to February 21, 2023, more than 757.2 million confirmed cases and more than 6.8 million confirmed deaths are registered worldwide [30]. The same report shows 499833

confirmed cases and 7,572 confirmed deaths in the same period of time in Ethiopia.

Studies involving mathematical models of infectious disease are helping the public health authorities by giving them an in-depth information through analysis of dynamics of the disease to make an informed decisions and policy making. Oftentimes, deterministic models based on classical derivatives are used to study the disease transmission dynamics. These studies are also powerful tools for predicting the future aspects of a disease. As far as COVID-19 is concerned, currently there are several such researches which have been conducted and are helping the struggle towards containing the spread.

The outbreak of several pandemics such as COVID-19 requires the development of mathematical models in order to exhibit key epidemiological features, investigate transmission dynamics, and develop adequate control policies. Mathematical modeling when dealing with infectious diseases allows revealing inherent patterns and underlying structures that govern outbreaks. Simple models that contain the essential components and interactions are powerful tools to test different hypotheses and understand disease control for both short and long time. The stability analysis near the free disease equilibrium will show if the apparition of new infection cases will yield to disease outbreak. Some countries such Tunisia and Jordan registered zero cases for days in Summer 2020 but the introduction of new cases resulted in critical endemic situation by Autumn.

Coronavirus is a single-stranded RNA virus and many RNA viruses have already been adopted by our body like HIV. Therefore, sometimes drugs used for the diseases with RNA virus are also being used in case of COVID-19. However, the treatment strategies like, antibiotics and other medicines are just supporting systems not any specific treatment for COVID19. The good point is that throughout the world, different research and development section's researchers are working and performing different kinds of experiments to investigate specific

treatment particularly targeted to COVID-19. However, the development of medicine must take sufficient time due to different steps involved in the drug development procedure. Therefore, at present, the key question is how to control the spread of COVID-19 until there is no treatment available.

The novel coronavirus is mainly spread from person to person, through respiratory droplets, the spread is more likely when people are within 6 feet of each other [6]. There is no known curing medicine to combat the COVID-19 pandemic. Standard recommendations by the WHO to prevent the spread of COVID-19 include frequent cleaning of hands using soap or alcohol-based sanitizer, covering the nose and mouth with a flexed elbow or disposable tissue when coughing and sneezing and avoiding close contact with anyone that has a fever and cough [6].

Infectious diseases are known as transmissible diseases or communicable diseases. The illness of infectious diseases is caused by the infection, presence, and growth of pathogenic biological agents (known as pathogens) in an individual host organism. Pathogen is the microorganism (or microbe) that causes illness. Infectious pathogens include viruses, bacteria, fungi, protozoa, multicellular parasites, and aberrant proteins known as prions.

Mathematical modeling in epidemiology provides understanding of the underlying mechanisms that influence the spread of disease. The model formulation process clarifies assumptions, variables, and parameters. Moreover, models provide conceptual results such as thresholds, basic reproduction numbers, contact numbers, and replacement numbers. Mathematical models and computer simulations are useful experimental tools for building and testing theories, assessing quantitative conjectures, answering specific questions, determining sensitivities to changes in parameter values, and estimating the key parameters from data [1].

The complex spreading patterns of COVID-19 and the various spread speed of its variants make its containing and mitigating real challenges. The existing models vary in form and complexity, but the common objective is to provide important information for global health decision makers about the disease dynamics. The first control measure was lockdown and then health authorities imposed mask wearing and social distancing. Khajanchi et al. (2021) showed, by extending the classical SEIR model, that social distance is an important factor to reduce the reproduction number and this to reduce the virus spread. Despite the herd immunity acquired via vaccine or infection, the social distancing is still recommended as a public health measure. Driven by the observed characteristics of COVID-19, we propose a mathematical model with two infectious states. It was reported by World Health Organization that one in three people who get COVID-19 do not show any symptoms. This is a challenging problem for health authorities as the asymptomatic individuals carry the virus and may infect other people without knowing it. Moreover, consequent efforts were made worldwide since the authorization of new vaccines by the end of 2020. By the end of November 2021, more than 50% of the world population has first dose administered and only 40% has second dose administered.

This paper gives theoretical and numerical analysis associated with COVID-19 epidemic dynamics in order to answer these critical questions. The main contributions of this research are given as follows:

- ❖ Developing a novel mathematical model to predict the spread of COVID-19, with the presence of protected and hospitalization compartments
- ❖ Analyzing the existence of endemic equilibrium point and the stability of disease-free equilibrium.
- ❖ Investigating a real case study in Ethiopia, discussing the impact of protection and hospitalization on disease dynamics.

1.2 Statement of the problem

COVID-19 is highly contagious, it has spread across the globe within months. The death toll is rising and the infectious population is increasing at an alarming rate. There is still no known curing medicine to fight the COVID-19 pandemic. Hence, it seems to be quite important to undertake more researches on the spread dynamics of the virus in such areas and a serious control strategy must be established. In 2020, Abeer D. Algarni and Monia Hamdi studied mathematical modeling and optimal control analysis of COVID-19. In the model the impacts of hospitalized and protected individuals who uses hand sanitizer properly and wearing face masks in public places were not considered. Thus, the present work is a modification of Abeer D. Algarni and etl model by considering protected and hospitalized individuals. This study is expected to address the following basic questions:

- i. How to modify an existing mathematical model for the transmission dynamics of COVID-19 by incorporating protected and hospitalized individuals?
- ii. How to analyze the modified mathematical model of COVID-19?
- iii. To which parameters is the dynamics of the disease more sensitive?
- iv. How to simulate a non-linear mathematical model using MATLAB software?
- v. What is the impact of protection and hospitalization on the dynamics of COVID-19 transmission?

1.3 Objective the study

The study has general and specific objectives.

1.3.1 General objective

The general objective of this study is to formulate and analyze a modified mathematical model on the transmission dynamics of COVID-19 with protected and hospitalized individuals.

1.3.2 Specific Objectives

The specific objectives of this study are:

- To formulate a modified model for transmission dynamics of COVID-19.
- To establish the conditions for the existence of the disease-free and endemic equilibrium points of the model.
- To compute the basic reproduction number of the model.
- To perform the stability analysis of the equilibrium states of the model.
- To identify the sensitive parameter.
- To evaluate the impact of protection and hospitalization in the disease progression.

1.4 Significance of the Study

This study is motivated on the serious problem of the spread of COVID-19 in the world. For such serious problem, simple models can provide decision support for planning restrictions to be imposed on the society and control practices. This study is significant in the following ways:

- To identify the best strategy to eradicate the spread of COVID-19 in the population.
- The analysis of COVID-19 transmission could be used to predict COVID-19 outbreak.
- It can be useful for policy makers and medical administrators to create further awareness on usage of hand sanitizer and wearing of face masks in a population.
- It can be used as an input for further investigation on COVID-19 intervention strategies.

1.5 Delimitation of the study

This thesis mainly focuses on the study of the transmission dynamics of the disease Coronavirus disease 2019 (COVID-19). Due to the limited time to

conduct this study, we did not collect real data. We rather used secondary data from the literature that are related to our context for simulation purposes.

1.6 Organization of the thesis

The thesis is organized as follows. Chapter 2 presents review of related literature. Chapter 3 presents some epidemiological preliminaries and the methodology. The mathematical model is formulated and described in Chapter 4. The qualitative analysis of the modified model by examining the equilibrium points and its stability analysis is also studied in chapter 5. In chapter 6, we present numerical simulations for the model. Conclusions and recommendations of the study are given in Chapter 7.

Chapter 2

Literature Review

In 1927, Kermack and Mckendrick [11] introduced a prominent compartmental model to analyze the plague disease in Mumbai and succeeded in revealing its epidemiology. After that mathematical modeling have been playing a significant role in analyzing the spread and control of different infectious diseases [13, 14].

A number of compartmental models have been proposed and analyzed for the COVID-19 outbreak in different countries. In particular, Yang et al. [5] proposed a mathematical model for COVID-19 incorporating multiple transmission pathways, including both human-to-human and environment-to-human transmission routes. The authors employed a bilinear incidence rate based on the law of mass action and fitted the model with the data of Wuhan city of China and estimated the reproduction number. They also found that the contribution of the environmental reservoir I significant in shaping the overall disease risk. Their results also indicate that the COVID-19 infection remain endemic, which necessitates intervention programs and long-term disease prevention policies.

Ngonghala et al. in 2020 [12] developed a mathematical model of COVID-19 pandemic in US (particularly, in New York) for assessing the population-level impact of the mitigation strategies. The authors performed the rigorous analysis of the model and the impacts of non-pharmaceutical intervention strategies, social distancing, quarantine, contact-tracing, isolation, face mask, etc. Authors in [11] proposed a conceptual mathematical framework to explore the role of government and individual behavioral reaction on the outbreak of COVID-19 using South Africa reported cases. In addition, Nabi [10] proposed and calibrated a deterministic compartmental model for describing the transmission dynamics of the novel coronavirus disease based on the publicly available epidemiological data for Brazil, Russia, India and Bangladesh.

In 2020, Haileyesus and Getachew [31] proposed a conceptual SEIR model to study the pandemic COVID-19 transmission in Ethiopia. Global stability was analyzed using Lyapunov function. Furthermore, they introduced time dependent controls to the basic model and extended to an optimal control model of the disease.

Shaobo and etl. [32] proposed the generalized SEIR epidemic model to investigate the dynamics of COVID-19. In their study, infectious individuals are investigated by dividing into two classes; infectious with intervention and infectious without intervention, besides the proposed model has considered quarantine and treatment. The parameters are estimated using Particle Swarm Optimization (PSO) algorithm. Their study also explained that the parameters are changing since the control from the government is changing with time.

Alberto Godio in [33] proposed model based study on the dynamics of COVID-19 prediction and control. In the study the researches formulated a mathematical model introducing a quarantine class and government intervention measures to mitigate disease dynamics. In addition to this, authors analyzed the how these measures and control strategies affect the disease dynamics.

Authors in [31] developed a deterministic mathematical model of COVID-19 transmission in Ethiopia. This study proposed SEIR model with ordinary differential equation. Stability analysis and equilibrium point and sensitive analysis were carefully analyzed. However, the basic prevention and control strategies (quarantine, isolation and self-protection) are not analyzed and also the impacts of asymptomatic individuals are not considered separately.

Chapter 3

Mathematical Methodologies

In this chapter, we will present the methodological approaches that we will use in the next chapters to analyze our COVID-19 model. A deterministic compartmental model will be formulated by using a system of non-linear ordinary differential equation which is well known to describe the epidemiology of infectious diseases. We will analyze the local stability behavior of equilibria by using Routh-Hurwitz stability criteria. We will analyze the global stability of equilibria by using Castillo-Chavez theorem. Mathematical software such as MATLAB is used to compute and simulate the model numerically. Basic concepts of dynamical systems and epidemiology are also important tools to model the problem and analyze solutions. We introduce the following definitions and theorems that are necessary for the discussion to come.

3.1 System of Differential Equations

Definition 3.1.1: (Autonomous System of ordinary differential equations)
An autonomous n -dimensional system of ordinary differential equation has the form:

$$\begin{aligned}\dot{x}(t) &= f(x(t)), \\ x(t_0) &= x_0\end{aligned}\tag{3.1}$$

Where, $x_0, x \in D \subset \mathbb{R}^n$ and $f : \mathbb{R}^n \rightarrow \mathbb{R}^n$; with f is continuous at $x \in D \subset \mathbb{R}^n$.

Definition 3.1.2: [15] (Well-posedness)

An initial value problem, (IVP) given in (3.1) is mathematically said to be well-posed if the followings conditions hold:

- i.** Its solution exists,
- ii.** Its solution is unique,
- iii.** Its solution continuously depends on the initial conditions.
- iv.** The solution should be non-negative over time and should be bounded.

Theorem 3.1.1 [16] (Picard's theorem)

Consider the initial value problem given in (3.1.1), if the function f is continuous and that all its partial derivatives $\frac{\partial f_i}{\partial x_j}$, for $i, j = 1, 2, 3, \dots, n$ are continuous for x in some open connected set $D \subset \mathbb{R}^n$, then for $x_0 \in D$ the problem (3.1.1) has a solution $x(t)$ on some time interval $(-\tau, \tau)$, $\tau > 0$ about $t = 0$, and the solution is unique.

Definition 3.1.3 [17] (Positivity of solutions)

The solution of a given autonomous system, (3.1) is said to be positive if all trajectories $x(t)$ is positive $\forall t \geq 0$.

Definition 3.1.4: [18] (Boundedness of solutions)

The positive solution of an autonomous system, (3.1) is said to be bounded if any solution, $x(t, t_0, x_0)$ of (3.1) satisfies;

$$\|x(t, t_0, x_0)\| \leq C(\|x_0\|, t_0)$$

for all $t \geq t_0$ where, $C: \mathbb{R}^+ \rightarrow \mathbb{R}^+$ is a constant that depends on t_0 and x_0 .

Theorem 3.1.2: [19] Integrating factor method

Let a linear ordinary differential equation is given by:

$$\dot{y}(x) + p(x)y = q(x) \tag{3.2}$$

Then the integrating factor and its general solution respectively are given by:

$$I(x) = e^{\int p(x)dx} \tag{3.3}$$

$$y(x) = \frac{1}{I(x)} \left[\int I(x)q(x)dx + C \right] \tag{3.4}$$

Where, C is any arbitrary constant of integration.

3.2 Stability analysis of equilibrium points

The equilibrium points to a system of first order differential equations are the points at which each differential equation is equal to zero.

Definition 3.2.1: [20]. Given the autonomous system (3.1), a state x^* is said to be an equilibrium point of the system if $f(x^*) = 0$.

Definition 3.2.2: [21]. The solution x^* is said to be stable if for every $\epsilon > 0$, there exists a $\delta = \delta(\epsilon) > 0$ such that $|x^* - x_0| < \delta \Rightarrow |x^* - x(t)| < \epsilon, t > t_0 \in \mathbb{R}$ for every solution $x(t)$ of (3.1) with $x(t_0) = x_0$.

Definition 3.2.3: [21]. An equilibrium point x^* is said to be globally asymptotically stable if it is asymptotically stable \forall initial condition $x_0 \in \mathbb{R}^n$.

Definition 3.2.4: [21]. An equilibrium point of the model (3.1) is said to be locally asymptotically stable if it is locally stable and every trajectory that starts sufficiently close to tend towards as A steady state which is not stable is said to be unstable.

Definition 3.2.5: [21]. An equilibrium point of a given dynamical system is stable means all solution curves of the equation attracts towards the equilibrium point, while an equilibrium point is unstable means all solution curves of the dynamic system go away from the equilibrium point.

Definition 3.2.6: [21]. An equilibrium point x^* is globally stable if all trajectories converge to x^* , that is $\lim_{t \rightarrow \infty} x(t) = x^*$

3.2.1 Local stability by linearization

Mathematically, the stability of equilibrium point can be analyzed using the linearized system at the equilibrium point.

Definition 3.2.7: [23]. The Jacobian matrix associated to the system (3.1) at the equilibrium point x^* , which is denoted by $Df(x^*)$, is given by the matrix

$$Df(x^*) = \left[\frac{\partial f_i(x^*)}{\partial x_j} \right] = \left[\begin{array}{ccc} \frac{\partial f_1(x)}{\partial x_1} & \frac{\partial f_1(x)}{\partial x_2} & \dots & \frac{\partial f_1(x)}{\partial x_n} \\ \vdots & \ddots & & \vdots \\ \frac{\partial f_n(x)}{\partial x_1} & \dots & & \frac{\partial f_n(x)}{\partial x_n} \end{array} \right]_{x=x^*}$$

where $i, j = 1, 2, \dots, n$.

Proposition 3.2.1: (24). An equilibrium point x^* of the dynamical system (3.1) is locally asymptotically stable if all eigenvalues of the Jacobian $Df(x^*) = \left[\frac{\partial f_i(x^*)}{\partial x_j} \right]$ evaluated at x^* are negative. The equilibrium x^* is unstable if at least one of the eigenvalues of $Df(x^*)$ is positive.

3.2.2 Routh-Hurwitz stability criterion

Routh Hurwitz criterion is an important criteria that gives necessary and sufficient condition of for all of the roots of the characteristics polynomial (with real coefficients). Routh-Hurwitz Criteria is used to determine asymptotic stability of an equilibrium point for non-linear system of differential equations. Consider the characteristic equation of degree n given by

$$P(\lambda) = \lambda^n + a_1\lambda^{n-1} + a_2\lambda^{n-2} + \dots + a_n = 0 \quad (3.5)$$

where all the polynomial coefficients a_i , for $i = 1, 2, \dots, n$ are real constant. Define the $n \times n$ Hurwitz matrix using the coefficients a_i of the characteristic polynomial.

$$H_1 = (a_1), H_2 = \begin{pmatrix} a_1 & 1 \\ a_3 & a_2 \end{pmatrix}, H_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{pmatrix}, \dots, H_n = \begin{pmatrix} a_1 & 1 & 0 & 0 & \dots & 0 \\ a_3 & a_2 & a_1 & 1 & \dots & 0 \\ a_5 & a_4 & a_3 & a_2 & \dots & 0 \\ \cdot & \cdot & \cdot & \cdot & \dots & 0 \\ \cdot & \cdot & \cdot & \cdot & \dots & 0 \\ 0 & 0 & 0 & 0 & \dots & a_n \end{pmatrix},$$

where $a_j = 0$ if $j > n$. All of the roots of the polynomial $P(\lambda)$ are negative or negative real part if and only if the determinants of all Routh-Hurwitz matrices are positive, i.e.

$$\det(H_j) > 0, \quad j = 1, 2, 3, \dots, n$$

For polynomials of degree $n = 2, 3, 4, 5$ the Routh-Hurwitz criteria are summarized as:

$$n = 2: a_1 > 0 \text{ and } a_2 > 0$$

$$n = 3: a_1 > 0, a_3 > 0 \text{ and } a_1 a_2 > a_3$$

$$n = 4: a_1 > 0, a_3 > 0, a_4 > 0 \text{ and } a_1 a_2 a_3 > a_3^2 + a_1^2 a_4$$

$$n = 5: a_i > 0, i = 1, 2, 3, 4, 5, a_1 a_2 a_3 > a_3^2 + a_1^2 a_4 \text{ and } (a_1 a_4 - a_5)(a_1 a_2 a_3 - a_3^2 - a_1^2 a_4) > a_5(a_1 a_2 - a_3)^2 + a_1 a_5^2$$

3.2.3 Next Generation Matrix

In Eco-epidemiology, the next-generation matrix is a method used to derive the basic reproduction number, for a compartmental model of the spread of infectious diseases. This method is given by Diekmann and van den Driessche and Watmough [28]. To calculate the basic reproduction number by using a next-generation matrix, the whole population is divided into n compartments in which there are $m < n$ infected compartments. Let $x_i, i = 1, 2, 3, \dots, m$ be the numbers of infected individuals in the i^{th} infected compartment at time t [27].

3.2.4 Sensitivity Analysis

Sensitivity analysis is usually performed as a series of test in which one can use different sets of input parameters to observe how a change in the predictor parameter values affects the dynamical behavior of the system. It is a useful tool to identify how closely input parameters are related to a predictor parameter. It helps to determine the level of change necessary for an input parameter to reach the desired value of a predictor parameter. Changing the values of the most sensitivity parameters will be the most effective strategy in changing the results of the model.

Definition 3.2.3.1. The absolute sensitivity coefficient of a quantity Q with respect to a parameter p is the rate of change of Q with respect to p . It is denoted by $\frac{\partial Q}{\partial p}$.

This sensitivity coefficient informs whether a quantity increases or decreases as a parameter varies. However, it does not tell how fast the parameter influences the quantity Q .

Definition 3.2.3.2. The relative sensitivity coefficient (or normalized forward sensitivity index) of a quantity Q with respect to a parameter p is defined as:

$$Q_p = \frac{\partial Q}{\partial p} \times \frac{p}{Q}$$

3.3 Basic concepts in epidemiological modeling

Definition 3.4.1: [25]. Epidemiology is the study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to the prevention and control of health problems.

Mathematical model of infectious disease is finding and understanding of biological Phenomena, within appropriate assumptions according to biological landscapes to change mathematical language. The major merit of mathematics in epidemiology is to enhancement awareness on epidemics, in addition to this we identified how the dynamics of an infectious disease depends on essential parameters. By analyzing some details about the sensitive parametric value, we understand and determine new infection incidence as well as spreading and controlling effects of it [26].

3.3.1 Disease free equilibrium point (DFE)

When a population is affected by an epidemic disease, the epidemic can disappear completely from the population. In this case we say that the population is disease free. In epidemiological modeling, a disease free equilibrium (DFE) is a steady state in which the coordinates in the disease compartments are zero. If the disease free equilibrium is stable, then it is expected that the population will be disease free over time.

3.3.2 Endemic equilibrium point (EE)

Unlike the disease free equilibrium, an endemic equilibrium point (EE) is a steady state in which at least one of its coordinates in the infected compartment is non-zero. It is a steady state solution where the disease persists in the population.

3.3.3 Basic Reproduction Number

The concept of the basic reproduction number is one of the central topics in mathematical modeling of infectious diseases due to its meaning and extreme importance. It is very important in disease modeling because it gives an indication regarding the future state of the infection. It tells us whether or not the disease will persist or will be eradicated [28].

Definition 3.4.2: The basic reproduction number, denoted by R_0 , is defined as the average number of secondary infections that are produced when a single infected individual is introduced into a population of purely susceptible individuals.

If $R_0 > 1$, then each single infected individual can infect more than one person (in average). Hence the total number of infected will continuously increase and the disease will become endemic (it will remain in the population forever). However, if $R_0 < 1$, then each single infected individual can infect zero or at most one person (in average). Over time the number of newly infected will decrease and the population can become disease free. Therefore it is always expected that the disease free equilibrium is stable when $R_0 < 1$ and unstable when $R_0 > 1$ [27]. The basic reproduction number R_0 is computed using the method of next-generation matrix. Assume there are n infected compartment and m non-infected compartment. Let $x \in \mathbb{R}^n$ and $y \in \mathbb{R}^m$ be sub populations in each compartment. Further denote by \mathcal{F}_i the rate of secondary infection increase in the i^{th} compartment and \mathcal{V}_i the disease progression rate, death and recovery decrease the i^{th} compartment. The compartment model can be written as:

$$\frac{dx_i}{dt} = \mathcal{F}_i - \mathcal{V}_i(x, y), i = 1, 2, 3, \dots, n$$

$$\frac{dy_j}{dt} = G_j(x, y), j = 1, 2, 3, \dots, m$$

Denoting F and V the matrices,

$$F = \left(\frac{\partial \mathcal{F}_i}{\partial x_j}(0, y^*) \right), 1 \leq i, j \leq n$$

$$V = \left(\frac{\partial \mathcal{V}_i}{\partial x_j}(0, y^*) \right), 1 \leq i, j \leq n$$

then, FV^{-1} is the next generation matrix [27]. Therefore, the basic reproduction number is the spectral radius (the dominant eigenvalue) of the matrix FV^{-1} is basic reproduction number $R_0 = \rho(FV^{-1})$.

Definition 3.4.3: [22]. An individual not yet infected with the disease or those susceptible for the disease, that can be caught by the disease is known as Susceptible.

Definition 3.4.4: [22]. An individual who have been infected by the disease and are capable of transmitting the disease to those in the susceptible is known as Infected.

Definition 3.4.5: [22]. An individual who is immune to a disease is known as Recovered.

Chapter 4

Mathematical Model Formulation

4.1 The Existing Mathematical Model

The mathematical model used to study the transmission of COVID-19 that was proposed by Abeer D. Algarni and Monia Hamdi. The dynamics of the model system consists of five nonlinearly interacting dependent variables, namely; Susceptible individuals (denoted by S) are those who are not infected by the disease pathogen but there is a possibility to be infected. Vaccinated individuals (denoted by V) are individuals who are vaccinated and not infected by the disease. Infected individuals (denoted by I) are individuals who developed the symptom of the disease. Asymptomatic individuals (denoted by A) either recovered without undergo treatment or moved to infected class after some duration and Recovered individuals (denoted by R) are those individuals who recovered from the disease.

All newborns are assumed to be susceptible. The natural recruitment and the natural death are denoted by Λ and μ respectively. The disease-induced death rate is ignored. Susceptible individuals are vaccinated at rate constant ψ . The parameters α and β are the infecting rates of asymptomatic and infectious individuals, respectively. γ_1 and γ_2 are the rates that the infectious and asymptomatic individuals become recovered and acquire temporary immunity, respectively. The vaccinated individuals need a period of time to develop their immunity against the virus, represented by $\frac{1}{\eta}$. The virus may infect vaccinated individuals but at a lower rate than susceptible individuals who are not unvaccinated. Thus in this case, the transmission rates β and α are multiplied by a scaling factor ε_1 and ε_2 . The governing equations of the existing mathematical model was given following the flow chart.

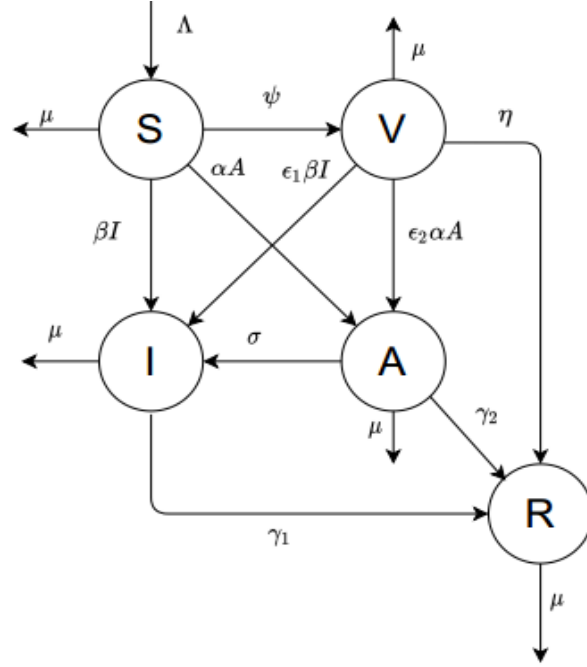


Figure 4.1. The Flow diagram of the existing mathematical model

$$\frac{dS}{dt} = \Lambda - \mu S - \psi S - \beta IS - \alpha AS \quad (4.1)$$

$$\frac{dV}{dt} = \mu V + \psi S - \eta V - \epsilon_1 \beta IV - \epsilon_2 \alpha AV \quad (4.2)$$

$$\frac{dI}{dt} = -\mu I + \beta IS + \epsilon_1 \beta IV + \sigma A - \gamma_1 I \quad (4.3)$$

$$\frac{dA}{dt} = -\mu A + \alpha AS - \sigma A + \epsilon_2 \alpha AV - \gamma_2 A \quad (4.4)$$

$$\frac{dR}{dt} = -\mu R + \eta V + \gamma_1 I + \gamma_2 A \quad (4.5)$$

with the initial conditions $S(0) = S_0 > 0, V(0) = V_0 > 0, I(0) = I_0 > 0, A(0) = A_0 > 0$ and $R(0) = R_0 > 0$

4.2 The Modified Mathematical Model

In this section, we modify the existing model to study the transmission dynamics of COVID-19 infection in a population. Our model is an extension of the existing SVIAR model for COVID-19 outbreak. In the present model we extended SVIAR model by including protected and hospitalized classes.

4.2.1 Assumption made for the model

The model divides the total population into seven sub-classes according to their disease status. Susceptible (S), Protected (P), Vaccinated (V), Infected (I), Hospitalized (H) Asymptomatic (A) and recovered (R).

The following **Assumptions** have been used in the formulation of the model:

- A₁: The population under study is heterogeneous and varying with time.
- A₂: All recruited human population is susceptible.
- A₃: Susceptible individuals who keeping social distancing, using an alcohol-based hand sanitizer and wearing face masks progress into protected class.
- A₄: We assume that individuals have no permanent immunity after recovery from the disease that is the recovered individuals have a chance to be susceptible again.
- A₅: Age, sex, social status, and race do not affect the probability of being infected.
- A₆: The natural mortality rates are assumed to be the same for all the compartments.
- A₇: The hospitalized individuals have potential of spreading the disease (i.e., hospital-acquired infections) at a lower rate than the infected individuals.
- A₈: The Vaccinated individuals have potential of not spreading the disease.

4.2.2 Variables and Parameters Used in the Model

Variables	Description of the state variables
S	Susceptible individuals
P	Protected individuals, who keeping social distancing, using an alcohol-based hand sanitizer and wearing face masks properly to protect themselves from the virus
V	Vaccinated Individuals
I	Infected individuals, who have active COVID-19 virus disease and can infect other people
A	Asymptomatic individuals either recovered without undergo treatment or moved to infected class after some duration.
H	Hospitalized individuals, who are admitted to health care facility or isolated in their home due to virus infection active cases.
R	Recovered individuals

Table 4.1: The state variables and their descriptions

Parameters

Biological meaning

Λ	Recruitment rate of Susceptible individuals
θ	Protection rate of Susceptible individuals
β	Effective contact rate
τ	Waning rate of Protected individuals to susceptible class
δ	The Asymptomatic progression rate
μ	Natural death rate of all state variables
ξ	Proportion of Asymptomatic individuals who join infected class
$(1 - \xi)$	The progression from asymptomatic class to recovered class
ϕ	Waning immunity rate of recovered class
α	Vaccination coverage rate of Protected class
ψ	Vaccination coverage rate of Susceptible Class
ω	Recovery rate of hospitalized patients
γ	Hospitalization rate of infected individuals
δ_1	Disease-induced death rate of infected individuals
δ_2	Disease-induced death rate of hospitalized individuals

Table 4.2 Parameters of the modified model and their descriptions.

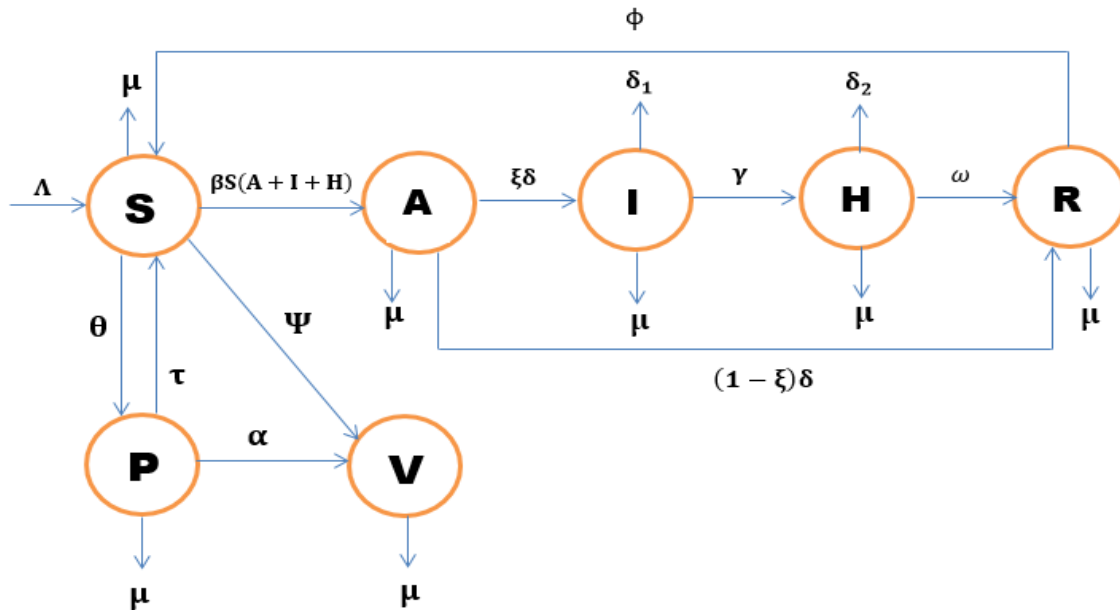


Figure 4.2: The flow chart for the modified model

Based on our assumptions and the flow chart, the modified model for the transmission dynamics of COVID-19 is given by the following deterministic system of non-linear differential equations:

$$\begin{cases} \frac{dS}{dt} = \Lambda + \phi R + \tau P - (\theta + \mu + \psi)S - (A + I + H)\beta S \\ \frac{dP}{dt} = \theta S - (\tau + \mu + \alpha)P \\ \frac{dV}{dt} = \psi S + \alpha P - \mu V \\ \frac{dA}{dt} = (A + I + H)\beta S - (\mu + \delta)A \\ \frac{dI}{dt} = \xi \delta A - (\mu + \omega + \delta_1)I \\ \frac{dH}{dt} = \omega I - (\mu + \delta_2 + \gamma)H \\ \frac{dR}{dt} = \gamma H + (1 - \xi)\delta A - (\mu + \phi)R \end{cases} \quad (4.2)$$

with the initial conditions $S(0) = S_0 > 0, P(0) = P_0 > 0, V(0) = V_0 > 0, I(0) = I_0 > 0, A(0) = A_0 > 0, H(0) = H_0 > 0,$ and $R(0) = R_0 > 0.$

4.3 Qualitative analysis of the modified model

In this section, we present some basic qualitative properties of the modified model. These analysis seek to show that the modified model is epidemiologically appropriate in the sense that the model and its predictions make sense. These analysis include finding the set inside which the model can be sufficiently studied (i.e., the invariant region); local and global stability of equilibrium points of the model.

4.3.1 Well-posedness.

Since all the functions on the right hand side of the system (4.6) are continuously differentiable. Thus, the existence and uniqueness of the solutions is established by the Picard's theorem (3.1). Now, we show the positivity and boundedness of solutions.

Theorem 4.3.1 (Positivity)

If $S(0) > 0, P(0) \geq 0, V(0) \geq 0, I(0) \geq 0, A(0) \geq 0, H(0) \geq 0$ and $R(0) \geq 0$, then the solution $(S(t), P(t), V(t), I(t), H(t), A(t), R(t))$ of the dynamical system (4.2) is non-negative for all time $t \geq 0$.

Proof. To show the positivity of the solution of the dynamical system (4.2), we will perform the proof by using contradiction. We assume that $S(t) \leq 0$ for some $t \geq 0$, that is there exists small $t_0 > 0$ such that $S(t_0) = 0, S'(t_0) \leq 0$ and $S(t) > 0$ for $t \in [0, t_0)$. Then $P(t) \geq 0, V(t) \geq 0, A(t) \geq 0$ and $I(t) \geq 0$ for $t \in [0, t_0]$. If this be not the case, there exists

Option I: $t_1 \in [0, t_0]$ such that $P(t_1) = 0, P'(t_1) < 0$ and $P(t) > 0$ for $t \in [0, t_1)$. Then $A(t) \geq 0, V(t) \geq 0$ and $I(t) \geq 0$ for $t \in [0, t_1]$.

Option II: $t_2 \in [0, t_1]$ such that $A(t_2) = 0, A'(t_2) < 0$ and $A(t) > 0$ for $t \in [0, t_2)$. Then $P(t) \geq 0, V(t) \geq 0$ and $I(t) \geq 0$ for $t \in [0, t_2]$.

Option III: $t_3 \in [0, t_2]$ such that $I(t_3) = 0, I'(t_3) < 0$ and $I(t) > 0$ for $t \in [0, t_3)$. Then $P(t) \geq 0, V(t) \geq 0$ and $A(t) \geq 0$ for $t \in [0, t_3]$.

Option IV: $t_4 \in [0, t_3]$ such that $V(t_4) = 0, V'(t_4) < 0$ and $V(t) > 0$ for $t \in [0, t_4)$. Then $P(t) \geq 0$ and $A(t) \geq 0$ for $t \in [0, t_4]$.

From equation $\frac{dI}{dt} = \xi\delta A - (\mu + \omega + \delta_1)I$ we have

$$I'(t_4) = \xi\delta A(t_4) - (\mu + \omega + \delta_1)I(t_4)$$

This implies that $I'(t_4) = \xi\delta A(t_4) \geq 0$. This is a contradiction. Integration of equation $\frac{dH}{dt} = \omega I - (\mu + \delta_2 + \gamma)H$ leads to

$$H(t) = e^{-(\mu + \delta_2 + \gamma)t} \left(H(0) + \omega \int_0^t I(s) e^{(\mu + \delta_2 + \gamma)s} ds \right) \geq 0 \text{ for } t \in [0, t_3].$$

Then $A'(t_3) = (I(t_3) + H(t_3))\beta S(t_3) \geq 0$. This is contradiction. Hence $H(t) \geq 0$ for every $t \in [0, t_2]$. Integration of $\frac{dR}{dt} = \gamma H + (1 - \xi)\delta A - (\mu + \phi)R$ lead us to

$$R(t) = e^{-(\mu+\phi)t} \left(R(0) + \int_0^t (1-\xi)\delta A(s) + \gamma H(s) e^{(\mu+\phi)s} ds \right) \geq 0 \text{ for } t \in [0, t_2].$$

This implies $V'(t_2) = (\psi S(t_2) + \alpha P(t_2)) \geq 0$. This is contradiction. Hence $S(t) \geq 0$ for every $t \in [0, t_1]$. Integration of $\frac{dP}{dt} = \theta S - (\tau + \mu + \alpha)P$ lead us to

$$P(t) = e^{-(\tau+\mu+\alpha)t} \left(P(0) + \int_0^t \theta S(s) e^{(\tau+\mu+\alpha)s} ds \right) \geq 0 \text{ for } t \in [0, t_1].$$

Then, $P'(t_1) = (I(t_3) + H(t_3))\beta S(t_3) \geq 0$. This is a contradiction. Hence $V(t) \geq 0$ for every $t \in [0, t_0]$. Thus $S'(t_0) = \Lambda + \phi R(t_0) + \tau P(t_0) > 0$, but this leads to a contradiction to the above assumption that $S'(t_0) \leq 0$. Therefore, the solutions $S(t), P(t), V(t), A(t), I(t), H(t), R(t)$ in the model system (4.2) remains positive for all $t > 0$. This completes the proof.

Theorem 4.3.2 (Boundedness)

There exists a positively invariant region Ω in which the solution $(S(t), P(t), V(t), A(t), I(t), H(t), R(t))$ of the model system (4.2) is bounded.

Proof. The positivity has already been established by Theorem (4.3.1). For this model the total population is $N(t) = S(t) + P(t) + V(t) + A(t) + I(t) + H(t) + R(t)$. Then, we obtain:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dP}{dt} + \frac{dV}{dt} + \frac{dA}{dt} + \frac{dI}{dt} + \frac{dH}{dt} + \frac{dR}{dt}$$

$$\begin{aligned} \frac{dN}{dt} = & \Lambda + \phi R + \tau P - \theta S - \mu S - \psi S - (A + I + H)\beta S + \theta S - \tau P - \mu P - \alpha P + \psi S + \alpha P - \mu V + (A + I + \\ & H)\beta S - \xi \delta A - \mu A - (1 - \xi)\delta A + \xi \delta A - \mu I - \omega I - \delta_1 I + \omega I - \mu H - \delta_2 H - \gamma H + \gamma H + (1 - \xi)\delta A - \mu R - \\ & \phi R \end{aligned}$$

$$\frac{dN}{dt} = \Lambda - \mu(S + P + V + I + H + A + R) - (\delta_1 I + \delta_2 H)$$

$$\frac{dN}{dt} = \Lambda - \mu N - (\delta_1 I + \delta_2 H)$$

This implies that

$$\frac{dN}{dt} \leq \Lambda - \mu N,$$

$$N' + \mu N \leq \Lambda,$$

$$e^{\mu t} N' + \mu N e^{\mu t} \leq \Lambda e^{\mu t},$$

$$\frac{d}{dt} (N e^{\mu t}) \leq \Lambda e^{\mu t}$$

Integrating $\frac{d}{dt} (N e^{\mu t}) \leq \Lambda e^{\mu t}$ we get

$$e^{\mu t} N \leq \frac{\Lambda}{\mu} e^{\mu t} + c$$

$N \leq \frac{\Lambda}{\mu} e^{\mu t} e^{-\mu t} + c e^{-\mu t}$, this simplifies to

$$N(t) \leq \frac{\Lambda}{\mu} + c e^{-\mu t}$$

Since the solution $H(t)$ and $I(t)$ are positive. Solving the differential inequality we get the relation,

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t},$$

If $N(0) \leq \frac{\Lambda}{\mu}$, then we obtain $0 \leq N(t) \leq \frac{\Lambda}{\mu}$, for all $t \geq 0$. If $N(0) \geq \frac{\Lambda}{\mu}$, then we have $0 \leq N(t) \leq N(0)$, for all $t \geq 0$. Thus, the feasible solution set of the system (4.2) remains in the region

$$\Omega = \left\{ (S, P, V, A, I, H, R) \in \mathbb{R}_+^7 : 0 \leq N(t) \leq \max \left(N(0), \frac{\Lambda}{\mu} \right) \right\}.$$

If we start with initial data $N(0) \in \Omega$, then the solution $N(t) \in \Omega$, for every $t > 0$. This shows the positively invariance of Ω . Thus, the solution of the dynamical system (4.2) is bounded. This completes the proof

Chapter 5

Equilibrium Points and Stability

The dynamical behavior of the equilibrium can be studied by computing the Jacobian matrix corresponding to each equilibrium point. Numerical methods can also be used to observe features and trends using Matlab ode45. Let us write the model (4.2) in the form:

$$\begin{cases} \frac{dS}{dt} = \Lambda + \phi R + \tau P - (\theta + \mu + \psi)S - (A + I + H)\beta S = f_1(S, P, V, A, I, H, R) \\ \frac{dP}{dt} = \theta S - (\tau + \mu + \alpha)P = f_2(S, P, V, A, I, H, R) \\ \frac{dV}{dt} = \psi S + \alpha P - \mu V = f_3(S, P, V, A, I, H, R) \\ \frac{dA}{dt} = (A + I + H)\beta S - (\mu + \delta)A = f_4(S, P, V, A, I, H, R) \\ \frac{dI}{dt} = \xi \delta A - (\mu + \omega + \delta_1)I = f_5(S, P, V, A, I, H, R) \\ \frac{dH}{dt} = \omega I - (\mu + \delta_2 + \gamma)H = f_6(S, P, V, A, I, H, R) \\ \frac{dR}{dt} = \gamma H + (1 - \xi)\delta A - (\mu + \phi)R = f_7(S, P, V, A, I, H, R) \end{cases} \quad (5.1)$$

with the initial conditions $S(0) = S_0 > 0, P(0) = P_0 > 0, V(0) = V_0 > 0, I(0) = I_0 > 0, A(0) = A_0 > 0, H(0) = H_0 > 0$, and $R(0) = R_0 > 0$

5.1 Equilibrium Points

In this section, we present the model equilibrium points, which are obtained by setting the right-hand sides of the system (5.1) to be zero. The model exhibits two equilibrium points namely, disease free equilibrium point (DFE) and endemic equilibrium point (EEP).

5.2 Steady state

The steady states of the system (4.2) are solutions of the following equations:

$$\Lambda + \phi R + \tau P - \theta S - \mu S - \psi S - (A + I + H)\beta S = 0$$

$$\theta S - \tau P - \mu P - \alpha P = 0$$

$$\psi S + \alpha P - \mu V = 0$$

$$(A + I + H)\beta S - \mu A - \delta A = 0$$

$$\xi\delta A - \mu I - \omega I - \delta_1 I = 0$$

$$\omega I - \mu H - \delta_2 H - \gamma H = 0$$

$$\gamma H + \delta A - \xi\delta A - \mu R - \phi R = 0$$

There are two steady states for the model system (4.2): the disease free equilibrium e_0 and endemic equilibrium e_1 which we proof later.

5.2.1 Disease Free equilibrium

The disease-free equilibrium point of our model is obtained by setting the disease state variables $A = 0$, $I = 0$ and $H = 0$.

$$\Lambda + \tau P - \theta S - \mu S - \psi S = 0 \quad (5.2)$$

$$\theta S - \tau P - \mu P - \alpha P = 0 \quad (5.3)$$

$$\psi S + \alpha P - \mu V = 0 \quad (5.4)$$

From (5.3) we have,

$$P = \frac{\theta S}{\tau + \mu + \alpha}$$

From (5.2) we have,

$$S = \frac{\Lambda(\tau + \mu + \alpha)}{(\tau + \mu + \alpha)(\theta + \mu + \psi) - \tau\theta} \quad (5.5)$$

$$P = \frac{\Lambda\theta}{(\tau + \mu + \alpha)(\theta + \mu + \psi) - \tau\theta} \quad (5.6)$$

From (5.4), (5.5) and (5.6) we have

$$V = \frac{\Lambda}{\mu} \left(\frac{A + \alpha\theta}{(\tau + \mu + \alpha)(\theta + \mu + \psi) - \tau\theta} \right) \quad (5.7)$$

Thus, the disease free equilibrium point is given by $e_0 = (S^0, P^0, V^0, A^0, I^0, H^0, R^0)$

$$= \left(\frac{\Lambda(\tau + \mu + \alpha)}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)}, \frac{\Lambda\theta}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)}, \frac{\Lambda}{\mu} \left(\frac{\psi(\tau + \mu + \alpha) + \alpha\theta}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)} \right), 0, 0, 0, 0 \right)$$

The existence of the endemic equilibrium point depends on the basic reproduction number R_0 and will be presented later.

Basic Reproduction number

The basic reproduction number, which is denoted by R_0 , and defined as the average number of secondary infections produced by a single infected individual in a completely susceptible population. Using the next generation matrix method [28], the basic reproduction number R_0 can be calculated from the relation $R_0 = \rho(FV^{-1})$. In the model presented in (4.2), the reproduction number could be obtained by the next generation matrix technique that is explained in the methodology section of the thesis. Re-arranging the differential equations so that we start with infective classes, we obtain:

$$\frac{dA}{dt} = (A + I + H)\beta S - \xi\delta A - \mu A - (1 - \xi)\delta A = 0$$

$$\frac{dI}{dt} = \xi\delta A - \mu I - \omega I - \delta_1 I = 0$$

$$\frac{dH}{dt} = \omega I - \mu H - \delta_2 H - \gamma H = 0$$

From the given system, new infections are generated in A, I, and H through contact with H, A, and I.

For A and I:

$$F_1 = (A + I + H)\beta S$$

$$F_2 = \xi\delta A$$

For H, new bacteria are generated from A and I:

$$F_3 = \omega I$$

For A:

$$V_1 = (\delta + \mu)A$$

For I:

$$V_2 = (\mu + \omega + \delta_1)I$$

For H:

$$V_3 = (\mu + \delta_2 + \gamma)H$$

We now construct the matrices F and V and then compute the next-generation matrix $K = FV^{-1}$. The new infections matrix F captures the rates at which new infections are introduced into the system from each of the infected compartments.

$$F = \begin{bmatrix} F_1 \\ F_2 \\ F_3 \end{bmatrix} = \begin{bmatrix} A \\ I \\ H \end{bmatrix} = \begin{bmatrix} \frac{\partial F_1}{\partial A} & \frac{\partial F_1}{\partial I} & \frac{\partial F_1}{\partial H} \\ \frac{\partial F_2}{\partial A} & \frac{\partial F_2}{\partial I} & \frac{\partial F_2}{\partial H} \\ \frac{\partial F_3}{\partial A} & \frac{\partial F_3}{\partial I} & \frac{\partial F_3}{\partial H} \end{bmatrix} = \begin{bmatrix} \frac{\partial A}{\partial A} & \frac{\partial A}{\partial I} & \frac{\partial A}{\partial H} \\ \frac{\partial I}{\partial A} & \frac{\partial I}{\partial I} & \frac{\partial I}{\partial H} \\ \frac{\partial H}{\partial A} & \frac{\partial H}{\partial I} & \frac{\partial H}{\partial H} \end{bmatrix} = \begin{bmatrix} \beta S^* & \beta S^* & \beta S^* \\ \xi \delta & 0 & 0 \\ 0 & \omega & 0 \end{bmatrix}$$

The transition matrix V describe show individuals or bacteria transition out of the infected compartments.

$$V = \begin{bmatrix} v_1 \\ v_2 \\ v_3 \end{bmatrix} = \begin{bmatrix} \frac{\partial V_1}{\partial A} & \frac{\partial V_1}{\partial I} & \frac{\partial V_1}{\partial H} \\ \frac{\partial V_2}{\partial A} & \frac{\partial V_2}{\partial I} & \frac{\partial V_2}{\partial H} \\ \frac{\partial V_3}{\partial A} & \frac{\partial V_3}{\partial I} & \frac{\partial V_3}{\partial H} \end{bmatrix} = \begin{bmatrix} \delta + \mu & 0 & 0 \\ 0 & \mu + \omega + \delta_1 & 0 \\ 0 & 0 & \mu + \delta_2 + \gamma \end{bmatrix}$$

The inverse of the transition matrix is:

$$V^{-1} = \begin{bmatrix} \frac{1}{\delta + \mu} & 0 & 0 \\ 0 & \frac{1}{\mu + \omega + \delta_1} & 0 \\ 0 & 0 & \frac{1}{\mu + \delta_2 + \gamma} \end{bmatrix}$$

Now, we compute the next-generation matrix $K = FV^{-1}$:

$$K = \begin{bmatrix} \beta S^* & \beta S^* & \beta S^* \\ \xi \delta & 0 & 0 \\ 0 & \omega & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\delta + \mu} & 0 & 0 \\ 0 & \frac{1}{\mu + \omega + \delta_1} & 0 \\ 0 & 0 & \frac{1}{\mu + \delta_2 + \gamma} \end{bmatrix}$$

Multiplying these matrices gives:

$$K = \begin{bmatrix} \frac{\beta S^*}{\delta + \mu} & \frac{\beta S^*}{\mu + \omega + \delta_1} & \frac{\beta S^*}{\mu + \delta_2 + \gamma} \\ \frac{\xi \delta}{\delta + \mu} & 0 & 0 \\ 0 & \frac{\omega}{\mu + \omega + \delta_1} & 0 \end{bmatrix}$$

Let F be the vector for the newly infected and V be the vector for the transfer of individuals into and out of the infected compartments. Let $x = (A, I, H)$, then we obtain:

$$F(x) = \begin{bmatrix} (A + I + H)\beta S \\ 0 \\ 0 \end{bmatrix} \text{ and } V(x) = \begin{bmatrix} (\mu + \delta)A \\ (\mu + \omega + \delta_1)I - \xi \delta A \\ (\mu + \delta_2 + \gamma)H - \omega I \end{bmatrix}$$

The Jacobian matrix to F and V are

$$F = \left[\frac{\partial F_i(e_0)}{\partial x_j} \right] = \begin{bmatrix} \beta S^0 & \beta S^0 & \beta S^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$V = \left[\frac{\partial V_i(e_0)}{\partial x_j} \right] = \begin{bmatrix} \mu + \delta & 0 & 0 \\ -\xi \delta & t_1 & 0 \\ 0 & -\omega & t_2 \end{bmatrix},$$

where $t_1 = \mu + \omega + \delta_1$ and $t_2 = \mu + \delta_2 + \gamma$. After some algebraic computations, the inverse of the matrix V is given by

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu + \delta} & 0 & 0 \\ \frac{\xi \delta}{t_1(\mu + \delta)} & \frac{1}{t_1} & 0 \\ \frac{\xi \delta \omega}{t_1 t_2(\mu + \delta)} & \frac{\omega}{t_1 t_2} & \frac{1}{t_2} \end{bmatrix},$$

The next-generation matrix FV^{-1} is given by

$$FV^{-1} = \begin{bmatrix} \beta S^0 & \beta S^0 & \beta S^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\mu + \delta} & 0 & 0 \\ \frac{\xi \delta}{t_1(\mu + \delta)} & \frac{1}{t_1} & 0 \\ \frac{\xi \delta \omega}{t_1 t_2(\mu + \delta)} & \frac{\omega}{t_1 t_2} & \frac{1}{t_2} \end{bmatrix},$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta S^0}{\mu + \delta} + \frac{\xi \delta \beta S^0}{t_1(\mu + \delta)} + \frac{\xi \delta \omega \beta S^0}{t_1 t_2(\mu + \delta)} & \frac{(t_2 + \omega) \beta S^0}{t_1 t_2} & \frac{\beta S^0}{t_2} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} K_1 + K_2 + K_3 & B & C \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

where, $K_1 = \frac{\beta S^0}{\mu + \delta}$, $K_2 = \frac{\xi \delta \beta S^0}{t_1(\mu + \delta)}$, $K_3 = \frac{\xi \delta \omega \beta S^0}{t_1 t_2(\mu + \delta)}$, $B = \frac{(t_2 + \omega) \beta S^0}{t_1 t_2}$ and $C = \frac{\beta S^0}{t_2}$

We find the eigenvalues of FV^{-1} by solving the characteristic equation

$$|FV^{-1} - \lambda I| = 0 \text{ as } \lambda_1 = K_1 + K_2 + K_3, \lambda_2 = \lambda_3 = 0.$$

The basic reproduction number R_0 is the spectral radius (the largest eigenvalues in modulus) of FV^{-1} which is given by:

$$R_0 = \rho(FV^{-1}) = K_1 + K_2 + K_3$$

The parts K_1, K_2 and K_3 represent the contributions from the human-to-human transmission routes (Asymptomatic-to-Susceptible, Infected-to-Susceptible and Hospitalized-to-Susceptible individuals, respectively). We can rewrite the basic reproduction number as follows:

$$R_0 = \left(\frac{\Lambda \beta (\tau + \mu + \alpha)}{(\mu + \delta)((\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi))} \right) \left(1 + \frac{\xi \delta}{\mu + \omega + \delta_1} + \frac{\xi \delta \omega}{(\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma)} \right)$$

5.2.2 Endemic equilibrium point

Endemic equilibrium point is a steady state solution where the disease persists in the population. In the presence of disease in the population, there exist an equilibrium point called endemic equilibrium point denoted by

$$e_1 = (S^*, P^*, V^*, A^*, I^*, H^*, R^*).$$

It can be obtained by setting each equation of the system (4.2) equal to zero.

$$\Lambda + \phi R + \tau P - (\theta + \mu + \psi)S - (A + I + H)\beta S = 0 \quad (5.8)$$

$$\theta S - (\tau + \mu + \alpha)P = 0 \quad (5.9)$$

$$\psi S + \alpha P - \mu V = 0 \quad (5.10)$$

$$(A + I + H)\beta S - (\mu + \delta)A = 0 \quad (5.11)$$

$$\xi \delta A - (\mu + \omega + \delta_1)I = 0 \quad (5.12)$$

$$\omega I - (\mu + \delta_2 + \gamma)H = 0 \quad (5.13)$$

$$\gamma H + (1 - \xi)\delta A - (\mu + \phi)R = 0 \quad (5.14)$$

From (5.12) we get,

$$A^* = \frac{q_1 I^*}{\xi \delta} \quad (5.15)$$

From (5.13) we get,

$$H^* = \frac{\omega I^*}{q_2} \quad (5.16)$$

By substituting equations (5.15) and (5.16) into equation (5.14), we get

$$R^* = \frac{I^*}{(\mu + \phi)} \left(\frac{\omega \gamma}{q_2} + \frac{(1 - \xi)q_1}{\xi} \right) \quad (5.17)$$

We substitute equations (5.15) and (5.16) into equation (5.11), it gives us

$$S^* = \frac{q_1 q_2 (\mu + \delta)}{\beta (q_1 q_2 + \xi \delta q_2 + \xi \delta \omega)} \quad (5.18)$$

Equation (5.18) can be written in terms of the basic reproduction number R_0 , and we obtain

$$S^* = \frac{S^0}{R_0} \quad (5.19)$$

By substituting equations (5.17) and (5.19) into equation (5.9), we have

$$P^* = \frac{\theta S^0}{R_0 (\tau + \mu + \alpha)} \quad (5.20)$$

By substituting equations (5.19) and (5.20) into equation (5.10), we have

$$\psi \frac{S^0}{R_0} + \alpha \frac{\theta S^0}{R_0 (\tau + \mu + \alpha)} - \mu V^* = 0 \quad (5.21)$$

Equation (5.21) can be simplified as

$$V^* = \frac{S^0}{\mu R_0} \left(\frac{\psi(\tau+\mu+\alpha)+\alpha\theta}{(\tau+\mu+\alpha)} \right)$$

By substituting equations (5.15), (5.16), (5.17), (5.19) and (5.20) into equation (5.8), we obtain

$$I^* = \frac{\left(S^0(\theta + \mu + \psi)(\tau + \mu + \alpha) - (\tau\theta S^0 + \Lambda R_0(\tau + \mu + \alpha)) \right) (\xi\delta q_2(\mu + \phi))}{(\tau + \mu + \alpha) \left(\phi\omega\delta^2\xi q_2 R_0 + (1 - \xi)q_1 q_2^2 \phi R_0 + (\mu + \phi)\beta S^0(q_1 q_2 + \xi q_2 + \omega\xi\delta) \right)}$$

Then the components $S^*, P^*, V^*, A^*, I^*, H^*$ and R^* are given by

$$S^* = \frac{S^0}{R_0}$$

$$P^* = \frac{\theta S^0}{R_0(\tau + \mu + \alpha)}$$

$$V^* = \frac{S^0}{\mu R_0} \left(\varphi + \frac{\alpha\theta}{(\tau + \mu + \alpha)} \right)$$

$$A^* = \frac{\left(S^0(\theta + \mu + \psi)(\tau + \mu + \alpha) - (\tau\theta S^0 + \Lambda R_0(\tau + \mu + \alpha)) \right) (\xi\delta q_1 q_2(\mu + \phi))}{(\tau + \mu + \alpha) \left(\phi\omega\delta^2\xi q_2 R_0 + (1 - \xi)q_1 q_2^2 \phi R_0 + (\mu + \phi)\xi\delta\beta S^0(q_1 q_2 + \xi q_2 + \omega\xi\delta) \right)}$$

$$I^* = \frac{\left(S^0(\theta + \mu + \psi)(\tau + \mu + \alpha) - (\tau\theta S^0 + \Lambda R_0(\tau + \mu + \alpha)) \right) (\xi\delta q_2(\mu + \phi))}{(\tau + \mu + \alpha) \left(\phi\omega\delta^2\xi q_2 R_0 + (1 - \xi)q_1 q_2^2 \phi R_0 + (\mu + \phi)\beta S^0(q_1 q_2 + \xi q_2 + \omega\xi\delta) \right)}$$

$$H^* = \frac{\left(S^0(\theta + \mu + \psi)(\tau + \mu + \alpha) - (\tau\theta S^0 + \Lambda R_0(\tau + \mu + \alpha)) \right) (\xi\delta\omega(\mu + \phi))}{(\tau + \mu + \alpha) \left(\phi\omega\delta^2\xi q_2 R_0 + (1 - \xi)q_1 q_2^2 \phi R_0 + (\mu + \phi)\xi\delta\beta S^0(q_1 q_2 + \xi q_2 + \omega\xi\delta) \right)}$$

$$I^* = \frac{\left(S^0(\theta + \mu + \psi)(\tau + \mu + \alpha) - (\tau\theta S^0 + \Lambda R_0(\tau + \mu + \alpha)) \right) (\xi\delta q_2(\mu + \phi))}{(\tau + \mu + \alpha) \left(\phi\omega\delta^2\xi q_2 R_0 + (1 - \xi)q_1 q_2^2 \phi R_0 + (\mu + \phi)\beta S^0(q_1 q_2 + \xi q_2 + \omega\xi\delta) \right)}$$

$$R^* = \frac{\left(\left(S^0(\theta + \mu + \psi)(\tau + \mu + \alpha) - (\tau\theta S^0 + \Lambda R_0(\tau + \mu + \alpha)) \right) (\xi\delta q_2(\mu + \phi)) \right) (\xi\omega\gamma + (1 - \xi)q_1 q_2)}{Q(\mu + \phi)q_2\xi}$$

Where,

$$Q = (\tau + \mu + \alpha)(\phi\omega\delta^2\xi q_2 R_0 + (1 - \xi)q_1 q_2^2 \phi R_0 + (\mu + \phi)\beta S^0(q_1 q_2 + \xi q_2 + \omega\xi\delta)),$$

Provided that $R_0 > 1$. From this we see that for the endemic equilibrium to exist $R_0 > 1$.

5.3 Stability analysis of equilibrium points

5.3.1 Local stability of DFE, E_0

Once the diseases free equilibrium point of our model is identified, we need to show its local stability, which is concerned with the behavior of the model solution near the DFE. Local stability of DFE can be determined from the sign of the Eigen values of the Jacobian matrix; that is, if all the eigenvalues are negative or have negative real parts, then the diseases free equilibrium point of the system is locally asymptotically stable and unstable if at least one of the eigenvalues has positive real part. The Jacobian matrix of the system (5.1) is given by:

$$J(e_0) = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial P} & \frac{\partial f_1}{\partial V} & \frac{\partial f_1}{\partial A} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial H} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial P} & \frac{\partial f_2}{\partial V} & \frac{\partial f_2}{\partial A} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial H} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial P} & \frac{\partial f_3}{\partial V} & \frac{\partial f_3}{\partial A} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial H} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial P} & \frac{\partial f_4}{\partial V} & \frac{\partial f_4}{\partial A} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial H} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial P} & \frac{\partial f_5}{\partial V} & \frac{\partial f_5}{\partial A} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial H} & \frac{\partial f_5}{\partial R} \\ \frac{\partial f_6}{\partial S} & \frac{\partial f_6}{\partial P} & \frac{\partial f_6}{\partial V} & \frac{\partial f_6}{\partial A} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial H} & \frac{\partial f_6}{\partial R} \\ \frac{\partial f_7}{\partial S} & \frac{\partial f_7}{\partial P} & \frac{\partial f_7}{\partial V} & \frac{\partial f_7}{\partial A} & \frac{\partial f_7}{\partial I} & \frac{\partial f_7}{\partial H} & \frac{\partial f_7}{\partial R} \end{pmatrix} \quad (5.22)$$

$$= \begin{pmatrix} -(\theta + \mu + \psi) & \tau & 0 & -\beta S^0 & -\beta S^0 & -\beta S^0 & \phi \\ \theta & -(\tau + \mu + \alpha) & 0 & 0 & 0 & 0 & 0 \\ \psi & \alpha & -\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta S^0 - (\mu + \delta) & \beta S & \beta S & 0 \\ 0 & 0 & 0 & \xi\delta & -(\mu + \omega + \delta_1) & 0 & 0 \\ 0 & 0 & 0 & 0 & \omega & -(\mu + \delta_2 + \gamma) & 0 \\ 0 & 0 & 0 & (1 - \xi)\delta & 0 & \gamma & -(\mu + \phi) \end{pmatrix}$$

Theorem 5.3.1:- The disease free equilibrium point of model (4.2) is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

Proof:-The Jacobian matrix associated with the system of equation (4.2) is: at the disease-free equilibrium e_0 is given by:

$$J(e_0) = \begin{pmatrix} -(\theta + \mu + \psi) & \tau & 0 & -\beta S^0 & -\beta S^0 & -\beta S^0 & \phi \\ \theta & -\tau - \mu - \alpha & 0 & 0 & 0 & 0 & 0 \\ \psi & \alpha & -\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta S^0 - \mu - \delta & \beta S^0 & \beta S^0 & 0 \\ 0 & 0 & 0 & \xi \delta & -\mu - \omega - \delta_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & \omega & -\mu - \delta_2 - \gamma & 0 \\ 0 & 0 & 0 & (1 - \xi)\delta & 0 & \gamma & -\mu - \phi \end{pmatrix}$$

Where, $S^0 = \frac{\Lambda(\tau + \mu + \alpha)}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)}$.

The matrix $J(e_0)$ is an upper triangular block matrix. Its eigenvalues are $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$ and λ_7 . Where λ_1, λ_2 and λ_3 are eigenvalues of the first block matrix of $J(e_0)$ and $\lambda_4, \lambda_5, \lambda_6$ and λ_7 are eigenvalues of the fourth block matrix of $J(e_0)$. The first block matrix of $J(e_0)$ given by:

$$J_1(e_0) = \begin{pmatrix} -(\theta + \mu + \psi) & \tau & 0 \\ \theta & -(\tau + \mu + \alpha) & 0 \\ \psi & \alpha & -\mu \end{pmatrix} = \begin{pmatrix} -A & \tau & 0 \\ \theta & -B & 0 \\ \psi & \alpha & -\mu \end{pmatrix}$$

Where

$$A = \theta + \mu + \psi \text{ and } B = \tau + \mu + \alpha$$

We find the eigenvalues of $J_1(e_0)$ by solving the characteristic equation

$$\det(J_1(e_0) - \lambda I_3) = 0$$

The characteristic equation of $J_1(e_0)$ is given by:

$$\begin{vmatrix} -A - \lambda & \tau & 0 \\ \theta & -B - \lambda & 0 \\ \psi & \alpha & -\mu - \lambda \end{vmatrix} = 0$$

$$(-\mu - \lambda)((-A - \lambda)(-B - \lambda) - \theta\tau) = 0$$

$$(-\mu - \lambda)((A + \lambda)(B + \lambda) - \theta\tau) = 0$$

$$(-\mu - \lambda)(\lambda^2 + (A + B)\lambda + AB - \theta\tau) = 0$$

as $\lambda_1 = -\mu, \lambda_2$ and λ_3 are the solutions of the quadratic equation $\lambda^2 + C\lambda + D = 0$.

Where $C = A + B$ and $D = AB - \tau\theta = (\mu + \psi)(\tau + \mu + \alpha) + \theta(\mu + \alpha)$.

Based on the Routh-Hurwitz criteria, the roots of the equation will be negative or the real parts will be negative if and only if $C > 0$ and $D > 0$.

Hence,

$$2\mu + \theta + \psi + \tau + \alpha > 0,$$

$$(\mu + \psi)(\tau + \mu + \alpha) + \theta(\mu + \alpha) > 0$$

Therefore, the equilibrium point E_0 , is locally asymptotically stable if the above conditions hold otherwise, it will be unstable.

The fourth block matrix of $J(e_0)$ is given by

$$J_4(e_0) = \begin{pmatrix} \Delta_3 & \beta S^0 & \beta S^0 & 0 \\ \xi \delta & -\Delta_1 & 0 & 0 \\ 0 & \omega & -\Delta_2 & 0 \\ (1 - \xi)\delta & 0 & \gamma & -(\mu + \phi) \end{pmatrix}$$

Where,

$$\Delta_1 = \mu + \omega + \delta_1, \quad \Delta_2 = \mu + \delta_2 + \gamma \text{ and } \Delta_3 = \beta S^0 - (\mu + \delta)$$

Thus the eigenvalues $\lambda_4, \lambda_5, \lambda_6$ and λ_7 are obtained from the characteristic equation of $J_4(e_0)$ is given by:

$$\begin{vmatrix} \Delta_3 - \lambda & \beta S^0 & \beta S^0 & 0 \\ \xi \delta & -\Delta_1 - \lambda & 0 & 0 \\ 0 & \omega & -\Delta_2 - \lambda & 0 \\ (1 - \xi)\delta & 0 & \gamma & -(\mu + \phi) - \lambda \end{vmatrix} = 0$$

Therefore, after some steps of computation, the characteristic equation becomes:

$$(-(\mu + \phi) - \lambda)[\lambda^3 + (\Delta_1 + \Delta_2 - \Delta_3)\lambda^2 + (\Delta_1\Delta_2 - \Delta_1\Delta_3 - \Delta_2\Delta_3 - \xi\delta\beta S^0)\lambda + (\Delta_1\Delta_2\Delta_3 + \xi\delta\beta S^0(\omega + \Delta_2))] = 0$$

From this equation, we obtain the values for λ to be $\lambda_4 = -\mu - \phi$, and the eigenvalues λ_5, λ_6 and λ_7 are the roots of the cubic polynomial:

$$P(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3$$

Where

$$a_1 = \Delta_1 + \Delta_2 - \Delta_3 > 0,$$

$$a_2 = \Delta_1\Delta_2 - (\Delta_1 + \Delta_2)\Delta_3 - \xi\delta\beta S^0 > 0,$$

$$a_3 = (\Delta_1\Delta_2\Delta_3 + \xi\delta\beta S^0(\omega + \Delta_2))$$

All the roots of the polynomial $P(\lambda)$ are negative or have negative real part if and only if the determinants of all Hurwitz matrices are positive. i.e. $\det(H_j) > 0$ for all $j = 1, 2, 3$. For the above characteristic polynomial, the Hurwitz Matrices can be constructed using the coefficients a_i and its determinant is computed as follows:

$$\begin{aligned}
H_1 &= [a_1], & |H_1| &= a_1 > 0. \\
H_2 &= \begin{bmatrix} a_1 & 1 \\ 0 & a_2 \end{bmatrix}, & |H_2| &= \begin{vmatrix} a_1 & 1 \\ 0 & a_2 \end{vmatrix} = a_1 a_2 > 0 \text{ if } a_1 > 0 \text{ and } a_2 > 0. \\
H_3 &= \begin{bmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{bmatrix}, & |H_3| &= \begin{vmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{vmatrix} = (a_1 a_2 - a_3) > 0 \\
&&& \text{if } a_i > 0, i = 1.2.3 \text{ and } a_1 a_2 > a_3
\end{aligned}$$

Here,

- $a_0 = 1 > 0$
- $a_1 = \Delta_1 + \Delta_2 - \Delta_3 = 3\mu + \omega + \delta_1 + \delta_2 + \gamma + \delta - \beta S^0 = 3\mu + \omega + \delta_1 + \delta_2 + \gamma + \delta - \frac{\beta\Lambda(\tau+\mu+\alpha)}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)}$
 $\Rightarrow a_1 > 0$
- $a_2 = \Delta_1 \Delta_2 - (\Delta_1 + \Delta_2) \Delta_3 - \xi \delta \beta S^0$
 $= (\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma) - (\mu + \omega + \delta_1 + \mu + \delta_2 + \gamma) \left(\frac{\beta\Lambda(\tau+\mu+\alpha) - (\mu+\delta)((\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi))}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right) - \left(\frac{\Lambda\xi\delta\beta(\tau+\mu+\alpha)}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right) > 0$
- $a_3 = -(\Delta_1 \Delta_2 \Delta_3 + \xi \delta \beta S^0 (\omega + \Delta_2))$
 $= (\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma) (\beta S^0 - (\mu + \delta)) + \xi \delta \beta S^0 (\omega + \Delta_2)$
 $= (\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma) (\beta S^0 - (\mu + \delta)) + \xi \delta \beta S^0 (\omega + \Delta_2) > 0$

Thus, $a_1 > 0, a_2 > 0$ and $a_3 > 0$.

$$\begin{aligned}
a_1 a_2 - a_3 &= \left(3\mu + \omega + \delta_1 + \delta_2 + \gamma + \delta - \frac{\beta\Lambda(\tau+\mu+\alpha)}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right) \left((\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma) - \right. \\
&\left. (\mu + \omega + \delta_1 + \mu + \delta_2 + \gamma) \left(\frac{\beta\Lambda(\tau+\mu+\alpha) - (\mu+\delta)((\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi))}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right) - \left(\frac{\Lambda\xi\delta\beta(\tau+\mu+\alpha)}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right) \right) > \\
&0 (\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma) (\beta S^0 - (\mu + \delta)) + \xi \delta \beta S^0 (\omega + \Delta_2) \\
a_1 a_2 - a_3 &= \left(\frac{(3\mu+\omega+\delta_1+\delta_2+\gamma+\delta)((\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)) - \beta\Lambda(\tau+\mu+\alpha)}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right) \left((\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma) - (\mu + \right. \\
&\left. \omega + \delta_1 + \mu + \delta_2 + \gamma) \left(\frac{\beta\Lambda(\tau+\mu+\alpha) - (\mu+\delta)((\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi))}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right) - \left(\frac{\Lambda\xi\delta\beta(\tau+\mu+\alpha)}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right) \right) + (\mu + \omega + \\
&\delta_1)(\mu + \delta_2 + \gamma) (\beta S^0 - (\mu + \delta)) + \xi \delta \beta S^0 (\omega + \Delta_2) > 0
\end{aligned}$$

Hence, by Routh-Hurwitz criteria, all roots are real distinct and negative. Therefore, the diseases free equilibrium point, of model system (4.2) is locally asymptotically stable if $R_0 < 1$, otherwise unstable.

5.3.2 Global stability of disease free equilibrium

In this subsection, we will look at the global stability of the disease free equilibrium. We will use theorem [3] to prove the global stability of the disease free equilibrium.

Theorem 5.3.2:

For $R_0 < 1$, the disease free equilibrium e_0 of the system (4.2) is globally asymptotically stable if $S^0 \geq S$.

Proof:

The proof is based on comparison theorem. Let us rewrite the model system (4.6) as:

$$\frac{dX}{dt} = F(X, I) \quad (5.23)$$

$$\frac{dI}{dt} = G(X, I), G(X, 0) = 0 \quad (5.24)$$

Where, $X = (S, P, V, R) \in \mathbb{R}_+^4$ represents the class of non-infected individuals and $I = (A, I, H) \in \mathbb{R}_+^3$ represents the class of infected individuals. The disease free equilibrium point of the model is denoted by $X^0 = (X^*, 0)$ where, $X^* = \left(\frac{\Lambda(\tau+\mu+\alpha)}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)}, \frac{\Lambda\theta}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)}, \frac{\Lambda}{\mu} \left(\frac{\psi(\tau+\mu+\alpha)+\alpha\theta}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} \right), 0 \right)$.

Since the diseases free equilibrium point is locally asymptotically stable, then to prove global stability, we will apply the Castillo-Chavez theorem. From system (4.2), we have:

$$\frac{dX}{dt} = F(X, I) = \begin{pmatrix} \Lambda + \phi R + \tau P - (\theta + \mu + \psi)S - (A + I + H)\beta S \\ \theta S - (\tau + \mu + \alpha)P \\ \psi S + \alpha P - \mu V \\ \gamma H + (1 - \xi)\delta A - (\mu + \phi)R \end{pmatrix} \quad (5.25)$$

$$\frac{dI}{dt} = G(X, I) = \begin{pmatrix} (A + I + H)\beta S - (\mu + \delta)A \\ \xi\delta A - (\mu + \omega + \delta_1)I \\ \omega I - (\mu + \delta_2 + \gamma)H \end{pmatrix} \quad (5.26)$$

1. To show X^* is globally asymptotically stable for the system $\frac{dX}{dt} = F(X, 0)$. let us consider the reduced system:

$$\frac{dX}{dt} = F(X, 0) = \begin{pmatrix} \Lambda + \phi R + \tau P - (\theta + \mu + \psi)S \\ \theta S - (\tau + \mu + \alpha)P \\ \psi S + \alpha P - \mu V \\ -(\mu + \phi)R \end{pmatrix}$$

We can rewrite the system (5.6) as

$$\begin{cases} \frac{dS}{dt} = \Lambda + \phi R + \tau P - (\theta + \mu + \psi)S \\ \frac{dP}{dt} = \theta S - (\tau + \mu + \alpha)P \\ \frac{dV}{dt} = \psi S + \alpha P - \mu V \\ \frac{dR}{dt} = -(\mu + \phi)R \end{cases} \quad (5.27)$$

The system (5.27) is non-homogeneous linear system of ordinary differential equations. By applying integration for the system (4.2), we obtain solutions:

From $\frac{dR}{dt} = -(\mu + \phi)R$, by separation of variable method we have

$$\begin{aligned} \frac{dR}{R} &= -(\mu + \phi)dt \\ \int \frac{dR}{R} &= -(\mu + \phi) \int dt \\ \ln R &= -(\mu + \phi)t + c \end{aligned}$$

where c is constant of integration. Applying the initial condition $R(0) = R_0$, it gives $c = \ln R_0$,

$$R(t) = \ln R e^{-(\mu+\phi)t}$$

As $t \rightarrow \infty$, $R(t) \rightarrow 0$.

From $\frac{dS}{dt} = \Lambda + \phi R + \tau P - (\theta + \mu + \psi)S$, we have

$$\frac{dS}{dt} + (\theta + \mu + \psi)S = \Lambda + \phi R + \tau P$$

Using integration factor method,

$$S(t) = e^{-(\theta+\mu+\psi)t} \left[\int e^{(\theta+\mu+\psi)t} (\Lambda + \phi R + \tau P) dt + C \right]$$

After simplification, we get

$$S(t) = \frac{\Lambda(\tau+\mu+\alpha)}{(\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi)} + \frac{1}{(\theta+\tau)} \left[\left(\tau(S(0) + R(0) + P(0)) - \frac{\Lambda\tau}{\phi} \right) e^{-(\theta+\mu)t} + \left((\theta + \mu)S(0) - \tau P(0) + \left(\frac{\tau-\phi(\theta+\tau)}{\theta+\tau} \right) R(0) - \frac{\Lambda\theta}{\theta+\mu+\psi} \right) e^{-(\theta+\mu+\psi)t} \right] - \left(\frac{\tau-\phi}{\theta+\tau} \right) R(0) e^{-\mu t}$$

$$P(t) = \frac{\Lambda\theta}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)} + \frac{1}{(\theta + \tau)} \left[\left((\theta(S(0) + P(0)) - \frac{\Lambda\tau}{\phi}) e^{-(\theta+\mu)t} + \left(\theta S(0) - \tau P(0) - \frac{\Lambda\theta}{\theta + \mu + \psi} \right) e^{-(\theta+\mu+\psi)t} \right] \right]$$

$$V(t) = \frac{\Lambda}{\mu} \left(\frac{\psi(\tau + \mu + \alpha) + \alpha\theta}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)} \right) + \frac{1}{(\mu + \psi)} \left[\left((\alpha(S(0) + V(0) + P(0)) - \frac{\tau}{\alpha}) e^{-(\theta+\mu)t} + \left(\mu V(0) - \alpha P(0) + \psi S(0) - \frac{\mu}{\mu + \psi} \right) e^{-(\theta+\mu+\psi)t} \right] - \left(\frac{\alpha}{\alpha + \mu} \right) P(0) e^{-\mu t}$$

Taking the limit as t goes to ∞ , we obtain

$$(S(t), P(t), V(t), R(t)) \rightarrow \left(\frac{\Lambda(\tau + \mu + \alpha)}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)}, \frac{\Lambda\theta}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)}, \frac{\Lambda}{\mu} \left(\frac{\psi(\tau + \mu + \alpha) + \alpha\theta}{(\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi)} \right), 0 \right) = X^*$$

Therefore, X^* is globally asymptotically stable for the system $\frac{dX}{dt} = F(X, 0)$

2. We need to show that $G(X, I) = AI - \widehat{G}(X, I)$, $\widehat{G}(X, I) \geq 0$ for $(X, I) \in R$ where

$A = \frac{\partial G}{\partial I}(X^*, 0)$ is a Metzler matrix (the off diagonal elements of A are non-negative) and R is the region where the model makes biological sense.

Consider a matrix:

$$A = \frac{\partial G}{\partial I}(X^*, 0) = \begin{pmatrix} (A + I + H)\beta S - (\mu + \delta)A \\ \xi\delta A - (\mu + \omega + \delta_1)I \\ \omega I - (\mu + \delta_2 + \gamma)H \end{pmatrix}$$

$$A = \frac{\partial G}{\partial I}(X^*, 0) = \begin{pmatrix} (\beta - (\mu + \delta))S^0 & \beta S^0 & \beta S^0 \\ \xi\delta & -(\mu + \omega + \delta_1) & 0 \\ 0 & \omega & -(\mu + \delta_2 + \gamma) \end{pmatrix}$$

Hence, the off diagonal elements of A are non-negatives, means A is a Metzler matrix.

$$\widehat{G}(X, I) = AI - G(X, I)$$

$$\widehat{G}(X, I) = \begin{pmatrix} (\beta - (\mu + \delta))S^0 & \beta S^0 & \beta S^0 \\ \xi\delta & -(\mu + \omega + \delta_1) & 0 \\ 0 & \omega & -(\mu + \delta_2 + \gamma) \end{pmatrix} \begin{pmatrix} A \\ I \\ H \end{pmatrix} - \begin{pmatrix} (A + I + H)\beta S - (\mu + \delta)A \\ \xi\delta A - (\mu + \omega + \delta_1)I \\ \omega I - (\mu + \delta_2 + \gamma)H \end{pmatrix}$$

$$\widehat{G}(X, I) = \begin{pmatrix} (\beta - (\mu + \delta))A^0 S^0 + \beta I^0 S^0 + \beta H^0 S^0 - (A^0 + I^0 + H^0)\beta S^0 + (\mu + \delta)A^0 \\ \xi\delta A^0 - (\mu + \omega + \delta_1)I^0 - \xi\delta A + (\mu + \omega + \delta_1)I \\ \omega I^0 - (\mu + \delta_2 + \gamma)H^0 - \omega I^0 + (\mu + \delta_2 + \gamma)H^0 \end{pmatrix}$$

After some simplification, we get

$$\widehat{G}(X, I) = \begin{pmatrix} \beta A(S^0 - S) + \beta I(S^0 - S) + \beta H(S^0 - S) \\ 0 \\ 0 \end{pmatrix}$$

$$\widehat{G}(X, I) = (S^0 - S) \begin{pmatrix} \beta(A + I + H) \\ 0 \\ 0 \end{pmatrix} \geq 0$$

Therefore by Castillo-Chavez theorem, the disease free equilibrium point e_0 of the system (4.2) is globally asymptotically stable for $R_0 < 1$.

5.3.3 Local Stability of EEP, \mathbf{E}^*

We analyze the stability of the endemic equilibrium point by linearizing the system of differential equations to get the Jacobian matrix. The Jacobian matrix is computed by differentiating each equation of system 5.1 with respect to the state variables at \mathbf{E}^* . The local stability of \mathbf{E}^* is determined by considering the sign of the eigenvalues of the Jacobian matrix of system 5.1 and finally, we will use Routh Hurwitz stability Criteria, which is used to determine asymptotic stability of equilibrium for a non-linear system of ordinary differential equations.

Theorem 5.2.1. A unique endemic equilibrium point $e_1 = (S^*, P^*, v^*, A^*, I^*, H^*, R^*)$ exists for the model (4.2) and is positive if $R_0 > 1$.

Proof: Stability of \mathbf{E}^* of the model system is determined based on the signs of the eigenvalues of the Jacobian matrix. The Jacobean matrix of system (4.2) around the endemic equilibrium, is given by:

$$J(e_1) = \begin{pmatrix} a & \tau & 0 & -\beta S^0 & -\beta S^0 & -\beta S^0 & \phi \\ \theta & b & 0 & 0 & 0 & 0 & 0 \\ \psi & \alpha & -\mu & 0 & 0 & 0 & 0 \\ g & 0 & 0 & c & \beta S & \beta S & 0 \\ 0 & 0 & 0 & \xi \delta & d & 0 & 0 \\ 0 & 0 & 0 & 0 & \omega & e & 0 \\ 0 & 0 & 0 & (1 - \xi)\delta & 0 & \gamma & f \end{pmatrix}$$

Where,

$$a = -(\theta + \mu + \psi + (A^* + I^* + H^*)\beta)$$

$$b = -(\tau + \mu + \alpha)$$

$$c = \beta S^* - \mu - \delta$$

$$d = -(\omega + \mu + \delta_1)$$

$$e = -(\gamma + \mu + \delta_2)$$

$$f = -(\mu + \phi)$$

$$g = (A^* + I^* + H^*)\beta$$

The characteristic polynomial of the Jacobian matrix at e_1 is given by; $\det(J_1(e_0) - \lambda I_7) = 0$ where, I_7 is the identity matrix of order 7 and λ is the eigenvalue of the Jacobian matrix

$$\begin{aligned}
|J(e_1) - \lambda I_7| = 0 &\Rightarrow \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* & -\beta S^* & -\beta S^* & \phi \\ \theta & b-\lambda & 0 & 0 & 0 & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 & 0 & 0 & 0 \\ g & 0 & 0 & c-\lambda & \beta S^* & \beta S^* & 0 \\ 0 & 0 & 0 & \xi\delta & d-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & \omega & e-\lambda & 0 \\ 0 & 0 & 0 & (1-\xi)\delta & 0 & \gamma & f-\lambda \end{vmatrix} = 0 \\
- \omega \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* & -\beta S^* & \phi \\ \theta & b-\lambda & 0 & 0 & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 & 0 & 0 \\ g & 0 & 0 & c-\lambda & \beta S^* & 0 \\ 0 & 0 & 0 & \xi\delta & d-\lambda & 0 \\ 0 & 0 & 0 & (1-\xi)\delta & \gamma & f-\lambda \end{vmatrix} + (e-\lambda) \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* & -\beta S^* & \phi \\ \theta & b-\lambda & 0 & 0 & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 & 0 & 0 \\ g & 0 & 0 & c-\lambda & \beta S^* & 0 \\ 0 & 0 & 0 & \xi\delta & d-\lambda & 0 \\ 0 & 0 & 0 & (1-\xi)\delta & 0 & f-\lambda \end{vmatrix} = 0 \\
\omega \xi \delta \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* & \phi \\ \theta & b-\lambda & 0 & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 & 0 \\ g & 0 & 0 & \beta S^* & 0 \\ 0 & 0 & 0 & \gamma & f-\lambda \end{vmatrix} - (e-\lambda)\xi\delta \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* & \phi \\ \theta & b-\lambda & 0 & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 & 0 \\ g & 0 & 0 & \beta S^* & 0 \\ 0 & 0 & 0 & 0 & f-\lambda \end{vmatrix} + \\
(e-\lambda)(d-\lambda) \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* & \phi \\ \theta & b-\lambda & 0 & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 & 0 \\ g & 0 & 0 & c-\lambda & 0 \\ 0 & 0 & 0 & (1-\xi)\delta & f-\lambda \end{vmatrix} = 0 \\
- \omega \xi \delta \gamma \begin{vmatrix} a-\lambda & \tau & 0 & \phi \\ \theta & b-\lambda & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 \\ g & 0 & 0 & 0 \end{vmatrix} + \omega \xi \delta (f-\lambda) \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* \\ \theta & b-\lambda & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 \\ g & 0 & 0 & -\beta S^* \end{vmatrix} \\
- (e-\lambda)(f-\lambda)\xi\delta \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* \\ \theta & b-\lambda & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 \\ g & 0 & 0 & \beta S^* \end{vmatrix} - (e-\lambda)(d-\lambda)(1-\xi)\delta \begin{vmatrix} a-\lambda & \tau & 0 & \phi \\ \theta & b-\lambda & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 \\ g & 0 & 0 & 0 \end{vmatrix} + \\
(e-\lambda)(d-\lambda)(f-\lambda) \begin{vmatrix} a-\lambda & \tau & 0 & -\beta S^* \\ \theta & b-\lambda & 0 & 0 \\ \psi & \alpha & -\mu-\lambda & 0 \\ g & 0 & 0 & c-\lambda \end{vmatrix} = 0 \\
\Rightarrow \omega \xi \delta \gamma \begin{vmatrix} \tau & 0 & \phi \\ b-\lambda & 0 & 0 \\ \alpha & -\mu-\lambda & 0 \end{vmatrix} - g \omega \xi \delta (f-\lambda) \begin{vmatrix} \tau & 0 & -\beta S^* \\ b-\lambda & 0 & 0 \\ \alpha & -\mu-\lambda & 0 \end{vmatrix} - \omega \xi \delta \beta (f-\lambda) S^* \begin{vmatrix} a-\lambda & \tau & 0 \\ \theta & b-\lambda & 0 \\ \psi & \alpha & -\mu-\lambda \end{vmatrix} - \\
g(e-\lambda)(f-\lambda)\xi\delta \begin{vmatrix} \tau & 0 & -\beta S^* \\ b-\lambda & 0 & 0 \\ \alpha & -\mu-\lambda & 0 \end{vmatrix} - (e-\lambda)(f-\lambda)\xi\delta\beta S^* \begin{vmatrix} a-\lambda & \tau & 0 \\ \theta & b-\lambda & 0 \\ \psi & \alpha & -\mu-\lambda \end{vmatrix} + \\
g(e-\lambda)(d-\lambda)(1-\xi)\delta \begin{vmatrix} \tau & 0 & \phi \\ b-\lambda & 0 & 0 \\ \alpha & -\mu-\lambda & 0 \end{vmatrix} - g(e-\lambda)(d-\lambda)(f-\lambda) \begin{vmatrix} \tau & 0 & -\beta S^* \\ b-\lambda & 0 & 0 \\ \alpha & -\mu-\lambda & 0 \end{vmatrix} + \\
(c-\lambda)(e-\lambda)(d-\lambda)(f-\lambda) \begin{vmatrix} a-\lambda & \tau & 0 \\ \theta & b-\lambda & 0 \\ \psi & \alpha & -\mu-\lambda \end{vmatrix} = 0 \\
\Rightarrow \alpha \phi \omega \xi \delta \gamma g (b-\lambda) + g \omega \xi \delta \alpha \beta (b-\lambda) S^* - \omega \xi \delta \beta (f-\lambda) S^* (-\mu-\lambda) ((b-\lambda)(a-\lambda) - \theta \tau) - g \beta (e-\lambda)(f-\lambda) (b-\lambda) \xi \delta S^* (-\mu-\lambda) - (e-\lambda)(f-\lambda) \xi \delta \beta S^* (-\mu-\lambda) ((a-\lambda)(b-\lambda) - \theta \tau) - g \phi (e-\lambda)(d-\lambda)(1-
\end{aligned}$$

$$\xi)\delta(b-\lambda)(-\mu-\lambda) - g\beta S^*(e-\lambda)(d-\lambda)(f-\lambda)(b-\lambda)(-\mu-\lambda) + (c-\lambda)(e-\lambda)(d-\lambda)(f-\lambda)(-\mu-\lambda)((a-\lambda)(b-\lambda) - \theta\tau) = 0$$

$$\Rightarrow (\alpha\phi\omega\xi\delta\gamma g + g\omega\xi\delta\alpha\beta S^*)(b-\lambda) - \omega\xi\delta\beta S^*(f-\lambda)(-\mu-\lambda)((b-\lambda)(a-\lambda) - \theta\tau) - g\beta(e-\lambda)(f-\lambda)(b-\lambda)\xi\delta S^*(-\mu-\lambda) - (e-\lambda)(f-\lambda)\xi\delta\beta S^*(-\mu-\lambda)((a-\lambda)(b-\lambda) - \theta\tau) - g\phi(e-\lambda)(d-\lambda)(1-\xi)\delta(b-\lambda)(-\mu-\lambda) - g\beta S^*(e-\lambda)(d-\lambda)(f-\lambda)(b-\lambda)(-\mu-\lambda) + (c-\lambda)(e-\lambda)(d-\lambda)(f-\lambda)(-\mu-\lambda)((a-\lambda)(b-\lambda) - \theta\tau) = 0$$

$$\Rightarrow k(b-\lambda) - r(f-\lambda)(-\mu-\lambda)((b-\lambda)(a-\lambda) - \theta\tau) - l(e-\lambda)(f-\lambda)(b-\lambda)(-\mu-\lambda) - m(e-\lambda)(f-\lambda)(-\mu-\lambda)((a-\lambda)(b-\lambda) - \theta\tau) - n(e-\lambda)(d-\lambda)(b-\lambda)(-\mu-\lambda) - o(f-\lambda)(e-\lambda)(d-\lambda)(b-\lambda)(-\mu-\lambda) + (c-\lambda)(e-\lambda)(d-\lambda)(f-\lambda)(-\mu-\lambda)((a-\lambda)(b-\lambda) - \theta\tau) = 0$$

Where,

$$k = \alpha\phi\omega\xi\delta\gamma g + g\omega\xi\delta\alpha\beta S^*, l = g\beta\xi\delta S^*, m = \xi\delta\beta S^*, n = g\phi\delta(1-\xi), r = \omega\xi\delta\beta S^* \text{ and } o = g\beta S^*$$

$$\begin{aligned} &\Rightarrow -\lambda^7 + (a+b+c+d+e+f-\mu)\lambda^6 \\ &- (df - \mu(d+f) + (a+b+c+e)(\mu - (d+f)) + ce + (a+b)(c+e) + (ab - \theta\tau) - o - m)\lambda^5 \\ &+ (ce(a+b) + (c+e)(ab - \theta\tau) + (ce + (a+b)(c+e) + ab - \theta\tau)(\mu - (d+f)) - df\mu + (a+b+c+e)(df - (d+f)) + o(\mu - (b+d+e+f)) - n + m(\mu - (a+b)) - r - l)\lambda^4 \\ &- (r(f - \mu + a + b) - l(\mu - (b + e + f)) + m(\mu(a + b + e + f)) + e + f + \theta\tau - ab - ef + n(\mu - (b + d + e)) + o(\mu(b + d + e + f) - (bd + ef) - (b + d)(e + f)) - df\mu(a + b + c + e) + ce(ab - \theta\tau) + (df - \mu(d + f))(ce + (a + b)(c + e) + ab - \theta\tau) - (ce(a + b) + (c + e)(ab - \theta\tau)(\mu - (d + f))))\lambda^3 \\ &+ (l((e + f)(\mu - b) + b\mu - ef) - r(\theta\tau + f\mu - ab - (a + b)(f - \mu)) + m(ef\mu + \mu(ab - \theta\tau) - (a + b)(ef - (e + f)(\mu + 1))) + n(b\mu + (d + e)(\mu - b) - de) + o(bd\mu + \mu(b + d)(e + f) + ef\mu - bd(e + f) - ef(b + d)) - ce(ab - \theta\tau)(\mu - (d + f)) + (ce(a + b) + (c + d)(ab - \theta\tau)(df - \mu(d + f))) - df\mu(ab + ce - \theta\tau + (a + b)(c + e))\lambda^2 \\ &- (r(ab - \theta\tau)(f - \mu) + l(ef(\mu - b) + b\mu(e + f) + m(ef\mu(a + b) + e + f + (\mu(e + f) - ef)(ab - \theta\tau))) + b\mu(d + e) + de(\mu - b) + ef\mu(b + d) + bd\mu(e + f) - bdef + ce(ab - \theta\tau)(df - \mu(d + f)) - df\mu(ce(a + b) + (c + e)(ab - \theta\tau)))\lambda \\ &+ (\mu rf + ef\mu - cdef\mu)(ab - \theta\tau) + \mu lbef + nbde\mu - obdef\mu = 0 \end{aligned}$$

$$-\lambda^7 + a_1\lambda^6 + a_2\lambda^5 + a_3\lambda^4 + a_4\lambda^3 + a_5\lambda^2 + a_6\lambda + a_7 = 0$$

Where,

$$a_1 = (a + b + c + d + e + f - \mu)$$

$$a_2 = -(df - \mu(d + f) + (a + b + c + e)(\mu - (d + f)) + ce + (a + b)(c + e) + (ab - \theta\tau) - o - m) > 0$$

$$a_3 = (ce(a + b) + (c + e)(ab - \theta\tau) + (ce + (a + b)(c + e) + ab - \theta\tau)(\mu - (d + f)) - df\mu + (a + b + c + e)(df - (d + f)) + o(\mu - (b + d + e + f)) - n + m(\mu - (a + b)) - r - l) > 0$$

$$a_4 = -\left(r(f - \mu + a + b) - l(\mu - (b + e + f)) + m(\mu(a + b + e + f)) + e + f + \theta\tau - ab - ef + n(\mu - (b + d + e)) + o(\mu(b + d + e + f) - (bd + ef) - (b + d)(e + f)) - df\mu(a + b + c + e) + ce(ab - \theta\tau) + (df - \mu(d + f))(ce + (a + b)(c + e) + ab - \theta\tau) - (ce(a + b) + (c + e)(ab - \theta\tau)(\mu - (d + f)))\right) > 0$$

$$a_5 = \left(l((e + f)(\mu - b) + b\mu - ef) - r(\theta\tau + f\mu - ab - (a + b)(f - \mu)) + m(ef\mu + \mu(ab - \theta\tau) - (a + b)(ef - (e + f)(\mu + 1))) + n(b\mu + (d + e)(\mu - b) - de) + o(bd\mu + \mu(b + d)(e + f) + ef\mu - bd(e + f) - ef(b + d)) - ce(ab - \theta\tau)(\mu - (d + f)) + (ce(a + b) + (c + d)(ab - \theta\tau)(df - \mu(d + f))) - df\mu(ab + ce - \theta\tau + (a + b)(c + e))\right) > 0$$

$$a_6 = -\left(r(ab - \theta\tau)(f - \mu) + l(ef(\mu - b) + b\mu(e + f) + m(ef\mu(a + b) + e + f + (\mu(e + f) - ef)(ab - \theta\tau))) + b\mu(d + e) + de(\mu - b) + ef\mu(b + d) + bd\mu(e + f) - bdef + ce(ab - \theta\tau)(df - \mu(d + f)) - df\mu(ce(a + b) + (c + e)(ab - \theta\tau))\right) > 0$$

$$a_7 = (\mu rf + efm\mu - cdef\mu)(ab - \theta\tau) + \mu l bef + nbde\mu - obdef\mu > 0$$

Hence, we can determine the signs of the eigenvalues of the characteristic polynomial by using Descartes's rule of signs which is:

Let $P(\lambda) = a_n\lambda^n + a_{n-1}\lambda^{n-1} + a_{n-2}\lambda^{n-2} + \dots + a_2\lambda^2 + a_1\lambda + a_0 = 0$ be a polynomial with real coefficients.

1. The number of positive zeros of P is either equal to the number of variations in sign of $P(\lambda)$ or less than by an even number

2. The number of negative zeros of P is either equal to the number of variations in signs of $P(-\lambda)$ or less than this by an even number.

By considering the characteristics polynomial of

$$\text{Let } P(\lambda) = \lambda^7 + a_1\lambda^6 + a_2\lambda^5 + a_3\lambda^4 + a_4\lambda^3 + a_5\lambda^2 + a_6\lambda + a_7 = 0$$

$$P(-\lambda) = -\lambda^7 + a_1\lambda^6 - a_2\lambda^5 + a_3\lambda^4 - a_4\lambda^3 + a_5\lambda^2 - a_6\lambda + a_7 = 0$$

The signs of the coefficients of $P(\lambda)$ in the descending powers are + + + + + + +. Since there is **no sign change** in the coefficients, by **Descartes' Rule of Signs**, the number of **positive real roots** is zero, meaning there are **no positive eigenvalues**.

By Descartes's Rules of signs $P(\lambda)$ will have non-positive eigenvalues. Also, the signs of the coefficients of $P(-\lambda)$ in the descending power of are - + - + - + - +. Clearly, there are 7 changes in signs which means there could be **7 negative real roots (eigenvalues)** or fewer, reduced by an even number from $P(-\lambda)$.

Since all the coefficients of the polynomial and all parameters are positive, by Descartes's rule of signs, all the roots are negative. Hence, the unique endemic equilibrium e_1 is locally asymptotically stable for $R_0 > 1$.

5.4 Sensitivity analysis

Sensitivity analysis is a useful tool in model building as well as in model evaluation by showing how the model behavior responds to changes in parameter values. It is used to discover parameters that have a high impact on the threshold R_0 and should be targeted by intervention strategies. In examining sensitivity analysis, it is not biologically reasonable to suggest that to decrease the secondary infection increasing the natural death rate. The threshold parameter R_0 which determines stability is a function of the parameters $\Lambda, \beta, \tau, \mu, \alpha, \xi, \delta, \omega, \delta_1, \delta_2, \gamma, \theta, \psi$. In order to study the effect of this parameters on R_0 we

performed a sensitivity analysis on R_0 with respect to this parameters. We recall that the basic reproduction number R_0 is given by

$$R_0 = \left(\frac{\Lambda\beta(\tau + \mu + \alpha)}{(\mu + \delta)((\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi))} \right) \left(1 + \frac{+\xi\delta}{\mu + \omega + \delta_1} + \frac{\xi\delta\omega}{(\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma)} \right)$$

$$R_0 = \frac{\Lambda\beta(\tau+\mu+\alpha)((\mu+\omega+\delta_1)(\mu+\gamma+\delta_2+\xi\delta)+\xi\delta\omega)}{(\mu+\delta)((\mu+\alpha)(\theta+\mu+\psi)+\tau(\mu+\psi))(\mu+\omega+\delta_1)(\mu+\gamma+\delta_2)}$$

$$R_0 = \left(\frac{A}{B} \right) (1 + \text{term 1} + \text{term 2})\Lambda$$

$$\text{term 1} = \frac{\xi\delta}{\mu+\omega+\delta_1}$$

$$\text{term 2} = \frac{\xi\delta\omega}{(\mu+\omega+\delta_1)(\mu+\delta_2+\gamma)}$$

$$A = \beta(\tau + \mu + \alpha)$$

$$B = (\mu + \delta)((\mu + \alpha)(\theta + \mu + \psi) + \tau(\mu + \psi))$$

Thus, in order to identify the most sensitive parameters for model (4.2), we compute the relative sensitivity of R_0 with respect to the above parameters. We display the sensitivity indices of R_0 with respect to the parameters.

$$\Delta_{\Lambda}^{R_0} = \frac{\partial R_0}{\partial \Lambda} \cdot \frac{\Lambda}{R_0} = 1 > 0$$

$$\Delta_{\beta}^{R_0} = \frac{\partial R_0}{\partial \beta} \cdot \frac{\beta}{R_0} = 1 > 0$$

$$\Delta_{\tau}^{R_0} = \frac{\partial R_0}{\partial \tau} \cdot \frac{\tau}{R_0} = \frac{\tau}{R_0} \left(\frac{\Lambda\beta}{B} \right) > 0$$

$$\Delta_{\alpha}^{R_0} = \frac{\partial R_0}{\partial \alpha} \cdot \frac{\alpha}{R_0} = \frac{\alpha}{R_0} \left(\frac{\Lambda\beta}{B} \right) > 0$$

$$\Delta_{\xi}^{R_0} = \frac{\partial R_0}{\partial \xi} \cdot \frac{\xi}{R_0} = \frac{\xi}{R_0} \left(\frac{\delta}{\mu + \omega + \delta_1} + \frac{\omega}{(\mu + \omega + \delta_1)(\mu + \delta_2 + \gamma)} \right) > 0$$

$$\Delta_{\omega}^{R_0} = \frac{\partial R_0}{\partial \omega} \cdot \frac{\omega}{R_0} = \frac{\omega}{R_0} \left(\frac{\xi\delta}{(\mu+\omega+\delta_1)^2} \right) > 0$$

$$\Delta_{\mu}^{R_0} = \frac{\partial R_0}{\partial \mu} \cdot \frac{\mu}{R_0} = -\frac{\mu}{R_0} \left(\frac{\Lambda\beta(\tau+\mu+\alpha)}{(1+\text{term 1}+\text{term 2})} \right) < 0$$

$$\Delta_{\delta}^{R_0} = \frac{\partial R_0}{\partial \delta} \cdot \frac{\delta}{R_0} = -\frac{\delta}{R_0} \left(\frac{\Lambda\beta(\tau+\mu+\alpha)}{B(\mu+\delta)} \right) < 0$$

$$\Delta_{\theta}^{R_0} = \frac{\partial R_0}{\partial \theta} \cdot \frac{\theta}{R_0} = -\frac{\theta}{R_0} \left(\frac{\Lambda\beta}{B(\theta+\mu+\psi)} \right) < 0$$

$$\Delta_{\psi}^{R_0} = \frac{\partial R_0}{\partial \psi} \cdot \frac{\psi}{R_0} = -\frac{\psi}{R_0} \left(\frac{\Lambda \beta}{B(\theta + \mu + \psi)} \right) < 0$$

$$\Delta_{\delta_1}^{R_0} = \frac{\partial R_0}{\partial \delta_1} \cdot \frac{\delta_1}{R_0} = -\frac{\delta_1}{R_0} \left(\frac{\xi \delta \omega}{(\mu + \omega + \delta_1)^2} \right) < 0$$

$$\Delta_{\delta_2}^{R_0} = \frac{\partial R_0}{\partial \delta_2} \cdot \frac{\delta_2}{R_0} = -\frac{\delta_2}{R_0} \left(\frac{\xi \omega}{(\mu + \delta_2 + \gamma)^2} \right) < 0$$

$$\Delta_{\gamma}^{R_0} = \frac{\partial R_0}{\partial \gamma} \cdot \frac{\gamma}{R_0} = -\frac{\gamma}{R_0} \left(\frac{\xi \omega}{(\mu + \delta_2 + \gamma)^2} \right) < 0$$

Note that the sensitivity index may depend on several parameters of the system, but also can be constant, independent of any parameter. For example, $\Delta_{\Lambda}^{R_0} = 1$ means that increasing (decreasing) Λ by a given percentage increases (decreases) always R_0 by that same percentage.

Sensitivity indices of R_0 with respect to Parameters

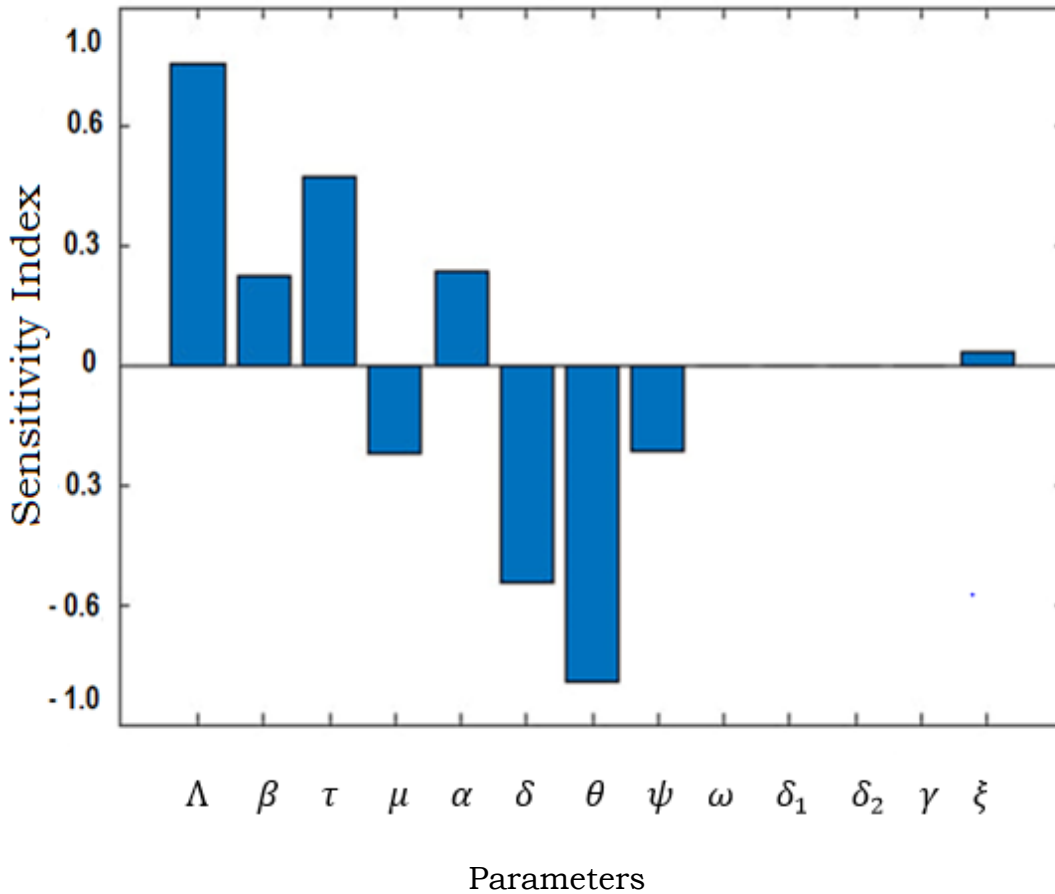


Figure 5.1: Sensitivity indices of R_0 with respect to parameters

Figure 5.1 shows, that the recruitment rate (Λ), Effective contact rate (β), Waning rate of Protected individuals to susceptible class (τ), Vaccination coverage rate of Protected class (α) and Proportion of Asymptomatic individuals who join infected class (ξ) are the most sensitive parameters for R_0 . That is they have positive impact on the expansion of COVID-19, while increasing the value of the Asymptomatic progression rate (δ), Protection rate of Susceptible individuals (θ) and Vaccination coverage rate of Susceptible Class (ψ) have negative impact on the expansion of COVID-19.

We summarize the sensitivity analysis indices of the reproduction number with respect to some parameters in Table 5.1.

Parameter Symbol	Sensitivity Indices
Λ	+ve
β	+ve
τ	+ve
ω	+ve
α	+ve
ξ	+ve
θ	-ve
μ	-ve
ψ	-ve
δ_1	-ve
δ_2	-ve
γ	-ve
δ	-ve

From Table 5.1: The sensitivity indices with negative signs such as θ , μ , ψ , δ_1 , δ_2 , γ , δ indicate that the value of R_0 decreases when the parameter values are increased and the value of R_0 increases when the parameter values are decreased. That is have an effect of minimizing the burden of the disease in the community as their values increase. While sensitivity indices with positive signs

such as $\Lambda, \beta, \tau, \omega, \alpha$ and ξ indicate that the value of R_0 increases when the parameter values are increased and the value of R_0 decreases when the parameter values are decreased. That is they have great impact on expanding the disease in the community if their values are increasing.

Chapter 6

Numerical Simulations and Discussions

In this section, we provide some numerical solutions to support our analytical findings and stability results obtained in the previous sections. Numerical methods are carried out to monitor the model system (4.6) and used to approximate solutions of equations when exact solutions cannot be determined via algebraic methods. It constructs successive approximations that converge to the exact solution of an equation or system of equations [29]. Numerical solution of model equations generally mimics the processes described in the model and it is carried out by help of numerical software called MATLAB ode 45. The attention is to assess the effect of different parameters on the spread of COVID -19 and these simulations can also show the behavior of the populations in time and the stabilities of both disease free equilibrium point and endemic equilibrium points.

To show the analytical results are in agreement with numerical solutions, we used parametric values given in table (6.1) below and the following hypothetical initial population of 10,000 such that; $S(0) = 5,000$, $P(0) = 3,000$, $V(0) = 1,320$, $A(0) = 1112$, $I(0) = 2450$, $H(0) = 2100$ and $R(0) = 2600$ for the period of days. And the results are given in the next pages of figures (6.1 – 6.3).

Table 6.1: Different parametric values used in our model

Parameter	Values	Sources	Units
Λ	500	[3]	Per day
θ	0.2	Assumed	Per day
β	0.0000009	[3]	Per day
τ	0.00000031	Assumed	Per day
δ	0.00586	[3]	Per day
μ	0.1	[3]	Per day
ξ	0.0029	[3]	Per day
$(1 - \xi)$	0.9971	[3]	Per day

ϕ	0.002	[3]	Per day
α	0.202	Assumed	Per day
ψ	0.0001	[11]	Per day
ω	0.0874	Assumed	Per day
γ	0.9	[11]	Per day
δ_1	0.896	Assumed	Per day
δ_2	0.032	Assumed	Per day

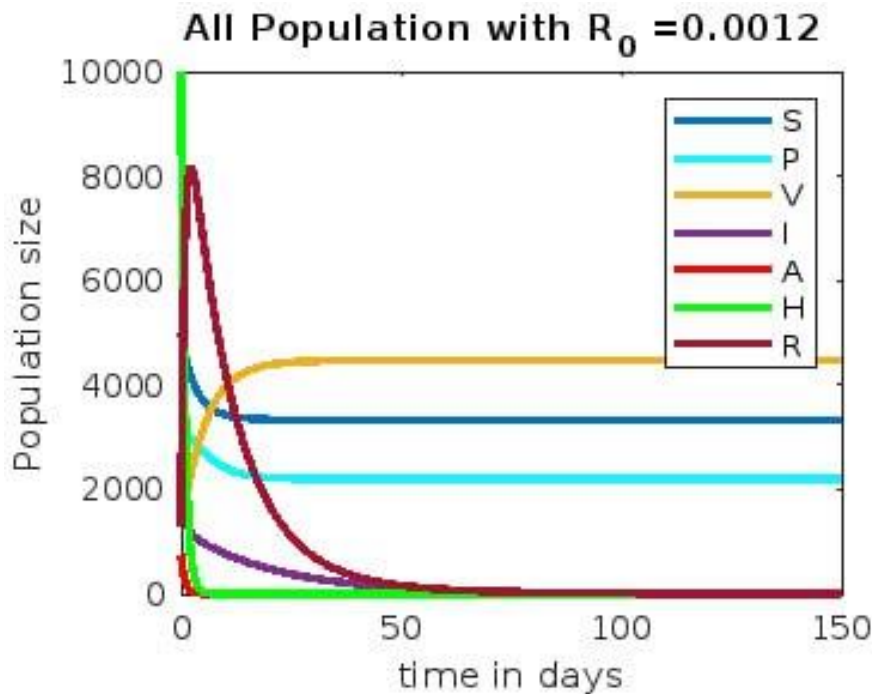


Figure 6.1: Trajectories of state variables for $R_0 = 0.0012$

In figure 6.1 with $R_0 = 0.0012$ we observed that for the basic reproduction number $R_0 < 1$ all solution curves goes to the disease free equilibrium point. As a result, the disease dies out. In Figure 6.1 with $R_0 < 1$ and different initial conditions for the state variables, all trajectories of state variables goes to their components of the disease free equilibrium point. This indicate that the disease-free equilibrium point is locally and globally asymptotically stable for the values of $R_0 < 1$.

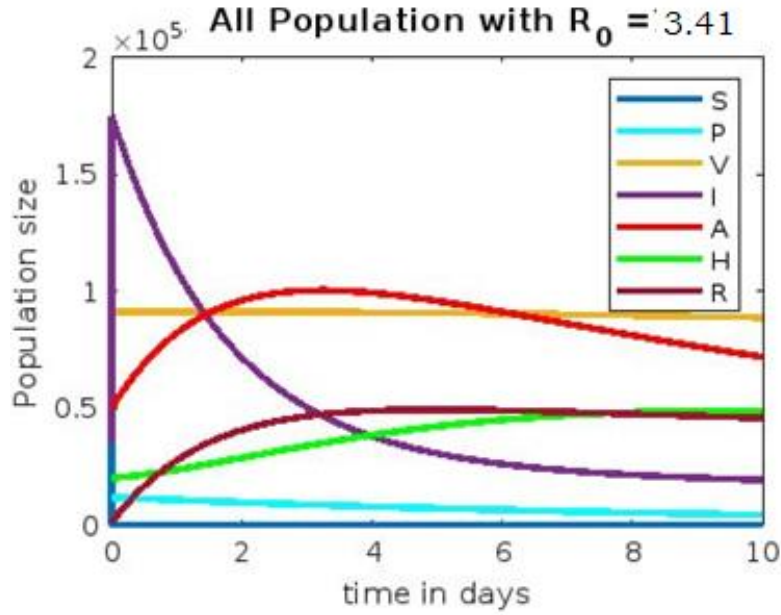


Figure 6.2 Trajectories of state variables for $R_0 = 3.41$.

In Figure 6.2 we tried to show the possible dynamics of disease in population and we can see from the graph that at $R_0 > 1$, the infected classes of the populations are increasing. This means that there is a need for reduction of the value of the basic reproduction number to a value less than unity.

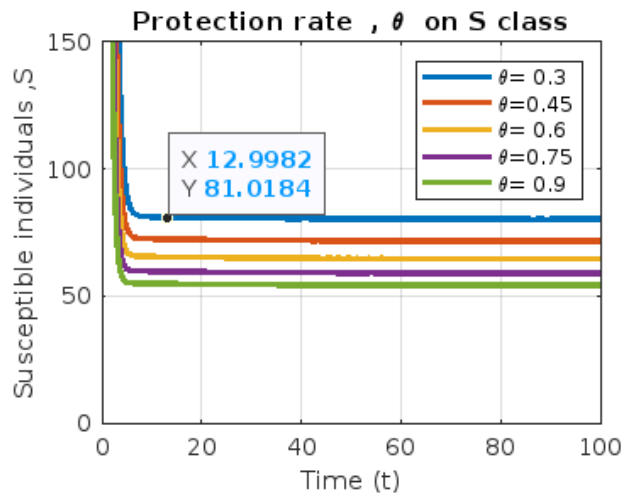


Figure 6.3: Graphical illustration of the Protection rate θ of susceptible individuals

From figure 6.3, we can observe that when there is high Protection rate θ , it reduces susceptible individuals, which means Protection rate θ is an effective way to reduce Susceptible population from COVID19 .

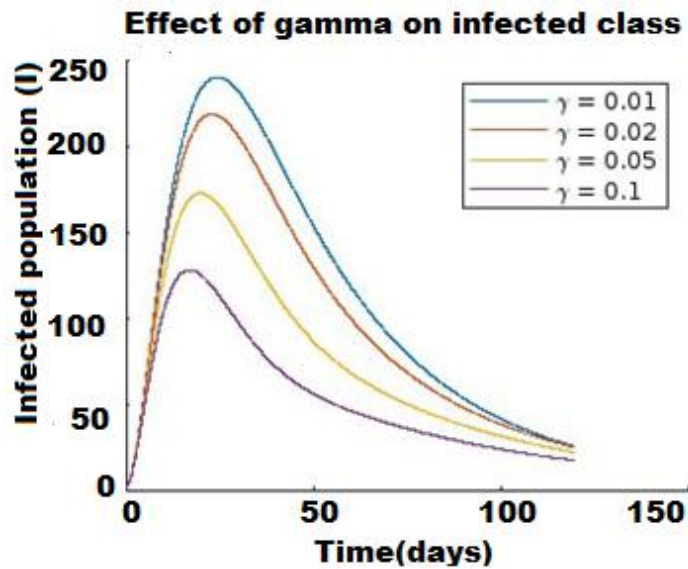
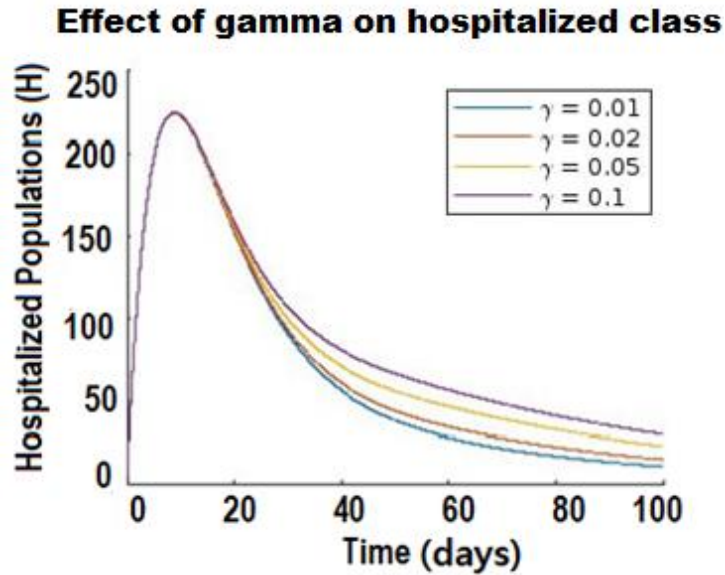


Figure 6.4: The effect of varying the hospitalization rate γ on infected and hospitalized population.

From the above figures 6.4, we observe that as the hospitalization rate increases, more individuals join the hospitalized compartment from infected class. The infected population will significantly decrease over time. The hospitalized population will increase.

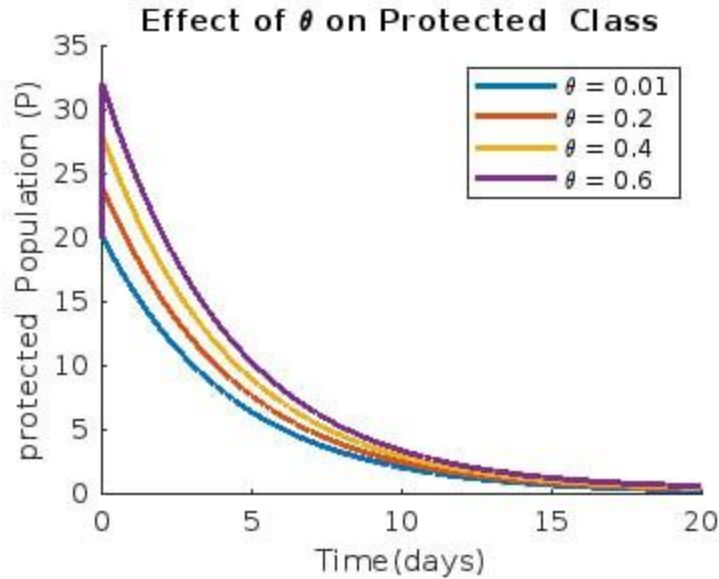


Figure 6.5: The effect of varying the protection rate θ on protected population.

Figure (6.5) shows that the effect of the protection rate θ on protected population. We observe that as the rate of protection increases, the protected individuals also increase because of susceptible individuals joined the protected class due to proper use of an alcohol-based hand sanitizer and wearing face masks in public places. This will result in decreasing on the transmission of COVID 19 disease.

Chapter 7

Conclusion and Recommendation

7.1 Conclusion

In this section, we have proposed a deterministic compartmental model to study the transmission dynamics of COVID-19. The modified model was an extension of the existing SVAIR model by including protected and hospitalized individuals. We have established the well-posedness of the modified model by proving the existence, positivity, and boundedness of the solutions.

We computed the steady states and the basic reproduction number R_0 . Based on the reproduction number R_0 , it is shown that whenever $R_0 < 1$, the system has only disease free equilibrium e_0 which is locally as well as globally asymptotically stable. When $R_0 > 1$, the system has a unique endemic equilibrium e_1 which is locally stable and the disease free equilibrium e_0 becomes unstable. We have observed that the outbreak of the disease dies out if $R_0 < 1$, and the disease is endemic if $R_0 > 1$.

Furthermore from sensitivity analysis of R_0 , we have observed that the recruitment rate and contact rate are most sensitive parameters to our model. Numerical results support the fact that decrease in the contact rate causes the decrease in the value of R_0 and after a certain level of contact rate, R_0 become less than one. If the protection rate increases, then all infected classes are decrease. It is predicted that the population will be disease-free.

7.2 Recommendation

Based on the results of this thesis, we strongly recommend the following points:

1. The stockholders should work on decreasing the positive indices and increasing negative indices parameters.

2. Possible extensions can be done in our model by incorporating co-infections.
3. The general public should be continually updated on how to protect the individuals from infection as well as how to take care of infected individuals so that he/she might not infect others.
4. Incorporate another parameters and assumptions to investigate the effect of contaminated materials for the transmission dynamics of COVID-19 pandemic.
5. Consider sub-populations related to age, gender, etc.

Reference

- [1] Allen, L., Brauer, F., van den Driessche, P., and Wu, J. (2008). *Mathematical epidemiology*, volume 1945 of *lecture notes in mathematics*. Springer, Berlin, 13:14.
- [2] Brauer, F., Castillo-Chavez, C., and Feng, Z. (2019). *Mathematical models in epidemiology*, volume 32. Springer.
- [3] Gurmu, E. D., Batu, G. B., and Wameko, M. S. (2020). Mathematical model of novel covid-19 and its transmission dynamics. *International Journal of Mathematical Modelling & Computations*, 10(2 (SPRING)):141–159.
- [4] Sohrabi, C., Alsafi, Z., O'Neill, N., Khan, M., Kerwan, A., Al-Jabir, A., Iosifidis, C., and Agha, R. (2020). World health organization declares global emergency: A review of the 2019 novel coronavirus (covid-19). *International journal of surgery*, 76:71–76.
- [5] Yang, C. and Wang, J. (2020). A mathematical model for the novel coronavirus epidemic in wuhan, china. *Mathematical biosciences and engineering: MBE*, 17(3):2708.
- [6] oquero, C. M. (2020). Challenges and opportunities for higher education amid the covid-19 pandemic: The philippine context. *Pedagogical Research*, 5(4).
- [7] Organization, W. H. et al. (2020). Infection prevention and control during health care when novel coronavirus (ncov) infection is suspected: interim guidance, 25 january 2020.
- [8] Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., Hu, Y., Zhang, L., Fan, G., Xu, J., Gu, X., et al. (2020). Clinical features of patients infected with 2019 novel coronavirus in wuhan, china. *The lancet*, 395(10223):497–506.
- [9]. Yang, J., Zheng, Y., Gou, X., Pu, K., Chen, Z., Guo, Q., Ji, R., Wang, H., Wang, Y., and Zhou, Y. (2020). Prevalence of comorbidities and its effects in patients infected with sars-cov-2: a systematic review and meta-analysis. *International Journal of Infectious Diseases*, 94:91–95.
- [10] Kermack, W. O. and McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772):700–721.

- [11] Mushayabasa, S., Ngarakana-Gwasira, E. T., and Mushanyu, J. (2020). On the role of governmental action and individual reaction on covid-19 dynamics in south africa: A mathematical modeling study. *Informatics in Medicine Unlocked*, 20:100387.
- [12] Ngonghala, C. N., Iboi, E., Eikenberry, S., Scotch, M., MacIntyre, C. R., Bonds, M. H., and Gumel, A. B. (2020). Mathematical assessment of the impact of non-pharmaceutical interventions on curtailing the 2019 novel coronavirus. *Mathematical biosciences*, 325:108364.
- [13] Anderson, R. M. and May, R. M. (1992). *Infectious diseases of humans: dynamics and control*. Oxford university press.
- [14] Castillo-Chavez, C. and Song, B. (2004). Dynamical models of tuberculosis and their applications. *Mathematical Biosciences & Engineering*, 1(2):361.
- [15] Zachmanoglou, E. C. and Thoe, D. W. (1986). *Introduction to partial differential equations with applications*. Courier Corporation.
- [16] Miranker, W. (1962). Existence, uniqueness and stability of solutions of systems of nonlinear difference-differential equations. *Journal of Mathematics and Mechanics*, 11(1):101–107.
- [17] Valcher, M. E. (2002). Positive systems in the behavioral approach: main issues and recent results. *Electronic proceedings of MTNS, 2002*.
- [18] Raffoul, Y. N. (2003). Boundedness in nonlinear differential equations. *Nonlinear Studies*, 10(4):343–350.
- [19] Korn, G. A. and Korn, T. M. (2000). *Mathematical handbook for scientists and engineers: definitions, theorems, and formulas for reference and review*. Courier Corporation.
- [20] Perko, L. (2013). *Differential equations and dynamical systems, volume 7*. Springer Science & Business Media.
- [21] Melesse, D. Y. (2010). Mathematical analysis of an seirs model with multiple latent and infectious stages in periodic and non-periodic environments.

- [22] Allotey, C. (2017). A comparison of existing measles models.
- [23] Tilahun, G. T. (2018). Mathematical Model for Co-Infection of Pneumonia and Typhoid Fever Disease with Optimal Control. PhD thesis, JKUAT.
- [24] Perko, L. (2013). Differential equations and dynamical systems, volume 7. Springer Science & Business Media.
- [25] Nelson, K. and Williams, C. (2013). Infectious disease epidemiology: theory and practice. third. nelson ke, williamns cm, editors.
- [26] Heffernan, J. M., Smith, R. J., and Wahl, L. M. (2005). Perspectives on the basic reproductive ratio. *Journal of the Royal Society Interface*, 2(4):281–293.
- [27] P. van den Driessche and James Watmough. Reproduction numbers and sub threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences* 180(2002)29 – 48.
- [28] Diekmann, O., Heesterbeek, J., and Roberts, M. G. (2010). The construction of next-generation matrices for compartmental epidemic models. *Journal of the Royal Society Interface*, 7(47):873– 885.
- [29] Nur Adila Faruk Senan (NY). ‘_A brief introduction to using ode 45 in MATLAB’. Department of Mechanical Engineering University of California at Berkeley.
- [30] WHO, Who coronavirus (covid-19) dashboard, 2022, <https://covid19.who.int/>.
- [31] Haileyesus Tessema Alemneh and Getachew Teshome Tilahun. Mathematical Modeling and Optimal Control Analysis of COVID-19 in Ethiopia. <https://doi.org/10.1101/2020.07.23.20160473>.
- [32] Shaobo He, Yuexi Peng and Kehui Sun. SEIR Modeling of the COVID-19 and its Dynamics. *Nonlinear Dyn*, Springer Nature B.V. 2020. <https://doi.org/10.1007/s11071-020-05743-y>
- [33] Alberto Godio , Francesca Pace and Andrea Vergnano. SEIR Modeling of the Italian Epidemic of SARS-CoV-2 Using Computational Swarm Intelligence. *International journal of environmental research and public health*. 2020, 17, 3535; <https://doi:10.3390/ijerph17103535>.

Appendices

MATLAB codes for stability analysis at different points:

```
function f =fikir16(~,y,Lambda,gamma,mu,phi,beta,tau,...
    omega,delta,theta,alpha,psi,xi,delta2,delta1)
S = y(1);
P = y(2);
V = y(3);
A = y(4);
I = y(5);
H = y(6);
R = y(7);
dS = Lambda + phi*R+tau*P-(theta+mu+psi)*S-(A+I+H)*beta*S;
dP= theta*S-(tau+mu+alpha)*P;
dV = psi*S+alpha*P-mu*V;
dA = (A+I+H)*beta*S-xi*delta*A-mu*H-(1-xi)*delta*A;
dI = xi*delta*A-(mu+omega+delta1)*I;
dH = omega*I-(delta2+mu+gamma)*H;
dR = gamma*H+(1-xi)*delta*A-(mu+phi)*R;
f = [dS;dP;dV;dA;dI;dH;dR];
end

%%%% Simulation of Disease free equilibrium point figure 6.1
Lambda=500; beta=0.0000009;delta=0.00586;mu=0.1;tau=0.00000031;
xi=0.00297;alpha=0.202;delta2=0.032;gamma=0.9;theta=0.2;
delta1=0.896;psi=0.0001;omega=0.0874;phi=0.002;
tsp=[0,150];
```

```

Y_0=[5000 3000 1320 1112 2450 2100 2600];
fprintf('Value of parameter R0 is %.5f,((Lambda*beta*(tau+mu+alpha))/(mu+delta)*...

((mu+alpha)*(theta+mu+psi)+tau*(mu+psi))*(1+(xi*delta)/(mu+omega+delta2)+(xi*delta
*omega)...

/((mu+omega+delta1)*(mu+delta2+gamma)))

[T,Y]=ode45(@(t,y)fikir16(t,y,Lambda,gamma,mu,phi,beta,tau,omega,delta,theta,...
alpha,psi,xi,delta2,delta1),tsp,Y_0);

plot(T,Y(:,1) ,T,Y(:,2),'c',T,Y(:,3),T,Y(:,4),T,Y(:,5),'r',T,Y(:,6),'g',T,Y(:,7),'LineWidth',2.5)
% plot(T,Y(:,1),T,Y(:,2),'c',T,Y(:,3),T,Y(:,4),'LineWidth',2.5)

legend('Susceptible','Protected individuals','Vaccinated Individuals',...
'Infected individuals','Asymptomatic individuals','Hospitalized individuals','Recovered
individuals')

xlabel('time in days')

ylabel('Population size')

title ('All Population with R_0 =0.0012 ');

%title ('All Population');

%saveas(h,'f1','pdf')

%set(h,'Units','Inches');

%pos=get(h,'Position');

%set(h,'PaperPositionMode','Auto','PaperUnits','Inches','PaperSize',[pos(3),pos(4)])%prin
t(h,'FDEP2','-dpdf'-r0'


% Simulation of Endemic equilibrium point figure 6.2

Lambda=500; beta=0.02;delta=0.586;mu=0.1;tau=0.00000031;

xi=0.00297;alpha=0.202;delta2=0.32;gamma=0.9;theta=0.000042;

delta1=0.896;psi=0.81;omega=0.9874;phi=0.0032;

tsp=[0,50];


```

```

Y_0=[5000 3000 1320 1112 1450 1100 1600];
fprintf('Value of parameter R0 is %.5f,((Lambda*beta*(tau+mu+alpha))/(mu+delta)*...

((mu+alpha)*(theta+mu+psi)+tau*(mu+psi))*(1+(xi*delta)/(mu+omega+delta2)+(xi*delta
*omega)...

/((mu+omega+delta1)*(mu+delta2+gamma)))

[T,Y]=ode45(@(t,y)fikir16(t,y,Lambda,gamma,mu,phi,beta,tau,omega,delta,theta,...
alpha,psi,xi,delta2,delta1),tsp,Y_0);

plot(T,Y(:,1) ,T,Y(:,2),'c',T,Y(:,3),T,Y(:,4),T,Y(:,5),'r',T,Y(:,6),'g',T,Y(:,7),'LineWidth',2.5)
% plot(T,Y(:,1),T,Y(:,2),'c',T,Y(:,3),T,Y(:,4),'LineWidth',2.5)

legend('Susceptible','Protected individuals','Vaccinated Individuals',...
'Infected individuals','Asymptomatic individuals','Hospitalized individuals','Recovered
individuals')

xlabel('time in days')

ylabel('Population size')

title ('All Population with R_0 = 1.21 ');

%title ('All Population');

%saveas(h,'f1','pdf')

%set(h,'Units','Inches');

%pos=get(h,'Position');

%set(h,'PaperPositionMode','Auto','PaperUnits','Inches','PaperSize',[pos(3),pos(4)])%prin
t(h,'FDEP2','-dpdf'-r0'

%% figure 6.3 parameters on compartments%% sigma=linspace(0.01,0.9,5)

Lambda=100; beta=0.000007;delta=0.056;mu=0.00086;tau=0.031;

xi=0.00297;alpha=linspace(0.31,0.9,5);%0.202;

delta2=0.032;gamma=0.531;theta=0.06;%linspace(0.3,0.9,5);%0.0601;

delta1=0.000896;psi=0.861;omega=0.00000874;phi=0.002;

```

```

tspan=[0,400];
y_0=[10000 10000 120 1112 1050 800 600];
for i=1:length(alpha)
[T,Y]=ode45(@(t,y)fikir16(t,y,Lambda,gamma,mu,phi,beta,tau,omega,delta,theta,alpha(i)
),psi,xi,delta2,delta1),...
tspan,y_0);
plot(T,Y(:,2),'Linewidth',2.5)
legend(strcat('\alpha=',num2str(alpha)))
xlabel('Time (t)')
ylabel('Protected individuals ,P')
title(' \alpha on P class')
axis([0 120 0 150]);
hold on
grid on
end
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

Figure 6.4 (a)

```

function effect_of_gamma_on_infected

% Define parameter values

% Define parameters

Lambda = 0.1; % Population influx rate

Phi = 0.05; % Recovery from hospitalized rate

tau = 0.02; % Transfer rate from population to infected class

theta = 0.05; % Birth rate to susceptible class

mu = 0.01; % Natural death rate

psi = 0.01; % Vaccination rate

```

```

beta = 0.2; % Infection transmission rate
alpha = 0.03; % Vaccination effectiveness
delta = 0.1; % Disease progression rate
xi = 0.7; % Fraction progressing to infectious
omega = 0.1; % Progression rate from infected to hospitalized
delta1 = 0.02; % Death rate for infected
delta2 = 0.03; % Death rate for hospitalized
% Time span for the simulation
tspan = [0 100];
% Initial conditions for [S, P, V, A, I, H, R]
init_conditions = [1000, 50, 20, 10, 5, 2, 0];
% Hospitalization rate values to test
%gamma_values = [0.05, 0.1, 0.15, 0.2];
    % Values of beta_1 to test
gamma_values = [0.01, 0.02, 0.05, 0.1]; % Different beta_1 values to analyze
% Set up plot
figure;
hold on;
    % Loop over different beta_1 values
for i = 1:length(gamma_values)
    gamma = gamma_values(i); % Current value of beta_1
        % Solve the system using ode45
    params = [Lambda, phi, tau, theta, mu, psi, beta, alpha, delta, xi, omega, delta1, delta2, gamma];
    % Solve ODE
[t, y] = ode45(@(t, y) system_eqns(t, y, params), tspan, init_conditions);

```

```

% Plot susceptible class over time

plot(t, y(:,5), 'DisplayName', ['\gamma = ', num2str(gamma)]);

end

% Customize plot

xlabel('Time');

ylabel('Infected Population (I)');

title('Effect of \gamma on Infected Class');

legend show;

hold off;

end

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

Figure 6.4 (b)

```

function effect_of_gamma_on_hospitalized

% Define parameter values

% Define parameters

Lambda = 0.1; % Population influx rate

phi = 0.05; % Recovery from hospitalized rate

tau = 0.02; % Transfer rate from population to infected class

theta = 0.05; % Birth rate to susceptible class

mu = 0.01; % Natural death rate

psi = 0.01; % Vaccination rate

beta = 0.2; % Infection transmission rate

alpha = 0.03; % Vaccination effectiveness

delta = 0.1; % Disease progression rate

xi = 0.7; % Fraction progressing to infectious

```

```

omega = 0.1; % Progression rate from infected to hospitalized
delta1 = 0.02; % Death rate for infected
delta2 = 0.03; % Death rate for hospitalized
% Time span for the simulation
tspan = [0 120];
% Initial conditions for [S, P, V, A, I, H, R]
Init_conditions = [1000, 50, 20, 10, 5, 2, 0];
% Hospitalization rate values to test
%gamma_values = [0.05, 0.1, 0.15, 0.2];
    % Values of beta_1 to test
gamma_values = [0.01, 0.02, 0.05, 0.1]; % Different beta_1 values to analyze
% Set up plot
figure;
hold on;
    % Loop over different beta_1 values
for i = 1:length(gamma_values)
    gamma = gamma_values(i); % Current value of beta_1
        % Solve the system using ode45
    params = [Lambda, phi, tau, theta, mu, psi, beta, alpha, delta, xi, omega, delta1, delta2, gamma];
    % Solve ODE
[t, y] = ode45(@(t, y) system_eqns(t, y, params), tspan, init_conditions);
    % Plot susceptible class over time
    plot(t, y(:,6), 'DisplayName', ['\gamma = ', num2str(gamma)]);
end
% Customize plot

```

```

xlabel('Time(days)');
ylabel('hospitalized Population (H)');
title('Effect of \gamma on hospitalized Class');
legend show;
hold off;
end
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

```

Figure 6.5

```

function effect_of_theta_on_protected

% Define parameter values

% Define parameters

Lambda = 0.1; % Population influx rate

phi = 0.05; % Recovery from hospital rate

tau = 0.2; % Transfer rate from population to infected class

mu = 0.01; % Natural death rate

psi = 0.01; % Vaccination rate

beta = 0.2; % Infection transmission rate

alpha = 0.02; % Vaccination effectiveness

delta = 0.1; % Disease progression rate

xi = 0.7; % Fraction progressing to infectious

omega = 0.1; % Progression rate from infected to hospitalized

delta1 = 0.02; % Death rate for infected

delta2 = 0.03; % Death rate for hospitalized

gamma = 0.05; % Recovery rate from hospital

% Time span for the simulation

```

```

tspan = [0 20];

% Initial conditions for [S, P, V, A, I, H, R]
init_conditions = [1000, 20, 20, 10, 5, 2, 0];

% Hospitalization rate values to test
%gamma_values = [0.05, 0.1, 0.15, 0.2];

    % Values of beta_1 to test

theta_values = [0.01, 0.2, 0.4, 0.6]; % Different beta_1 values to analyze

% Set up plot

figure;

hold on;

    % Loop over different beta_1 values
for i = 1:length(theta_values)

    theta = theta_values(i); % Current value of beta_1

        % Solve the system using ode45

    params = [Lambda, phi, tau, theta, mu, psi, beta, alpha, delta, xi, omega, delta1, delta2, gamma];

    % Solve ODE

[t, y] = ode45(@(t, y) system_eqns(t, y, params), tspan, init_conditions);

    % Plot susceptible class over time

    plot(t, y(:,2), 'DisplayName', ['\theta = ', num2str(theta)], 'LineWidth', 2.5);

end

    % Customize plot

xlabel('Time(days)');

ylabel('protected Population (P)');

title('Effect of \theta on Protected Class');

legend show;

hold off;

end

```