

**Mathematical Modeling and Analysis on the Dynamics of  
Tobacco Smoking by Considering Occasional and Habitual  
Smokers**



**Genet Endale**

**MSc Thesis Submitted to  
the Department of Mathematics  
College of Natural and Computational Sciences  
Hawassa University**

**June, 2024**

**Hawassa, Ethiopia**

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**Advisor: Tadele Tesfa (PhD)**

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**In Partial Fulfillment of the Requirement for the Masters of  
Sciences in Mathematical and Statistical Modeling  
(MASTMO)**

**June, 2024**

**Hawassa, Ethiopia**

# Declaration

I declare that this Thesis entitled "**Mathematical Modeling and Analysis on the Dynamics of Tobacco Smoking by Considering Occasional and Habitual Smokers**" is my own work and has not been submitted to any university for similar purpose. The references used in this thesis are duly recognized by proper citations.

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Name of student

Signature

Date

# Approval Sheet - I

This is to officially state that the study entitled “ **Mathematical Modeling and Analysis on the Dynamics of Tobacco Smoking by Considering Occasional and Habitual Smokers** ” is an original work carried out by Genet Endale, ID No. GPMASTR/0003/15 under my guidance and supervision. This is a genuine work that has been done by Genet Endale for the partial fulfillment of the award of the Degree of Master of Science in Mathematical and Statistical Modeling from Hawassa University. Daily acknowledgments are done during his course of investigation. Therefore, I recommend that it would be accepted as fulfilling the thesis requirements.

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Advisor

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# Approval Sheet - II

We, the undersigned, members of the Board of Examiners of the final open defense by Genet Endale have read and evaluated his thesis entitled “**Mathematical Modeling and Analysis on the Dynamics of Tobacco Smoking by Considering Occasional and Habitual Smokers**” 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 Mathematical and Statistical Modeling.

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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 candidate’s department.

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# List of Abbreviations

IVP	Initial-Value Problem
ODE	Ordinary Differential Equation
SFEP	Smoking Free Equilibrium Point
SHS	Second Hand Smoker
SPEP	Smoking Present Equilibrium Point
WHO	World Health Organization

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# Abstract

This study investigate smoking tobacco considering occasional and habitual smokers using mathematical model. The study aims to understand how to decrease smoking habit. we classified the total population in to four compartments namely; susceptible, secondhand smokers, occasional smokers, habitual smokers and Recovered. Positivity of the solutions and boundedness of the model are discovered. Using Routh-Hurwitz criteria ,center manifold theory and Castillo Chavez Theorem, we assed equilibrium point of the model and investigate their stability. The basic reproduction number ( $R_0$ ) is computed using next generation matrix method. We have proved that the smoking free equilibrium is locally and globally asymptotically stable when  $R_0 < 1$  the smoking habit removed & unstable. Also smoking present equilibrium point is locally asymptotically stable,when  $R_0 > 1$  smoking habit persist. Additionally, our model indicates that a forward bifurcation and sensitive analysis is performed. Using sensitivity analysis the rate of interaction of smokers with susceptible and rate of recruitment has positive impact on the spread of smoking habit and rate of death due to smoking and treatment rate has negative impact on the spread of smoking habit. Finally, we perform numerical simulations using MATLAB software Ode 45 codes to support the analytical results are in agreement with numerical solutions.

**Keyword :** smoking, secondhand, occasional, habitual, smoking generation number, Stability analysis, Forward bifurcation, Sensitivity analysis,

# Chapter 1

## Introduction

This chapter describes the general introduction of the study. It mainly focuses on the background of the study, the statement of the problem, objectives and significance of the study.

### 1.1 Background of the study

Tobacco is a plant that belongs to the *Nicotiana* genus, and its leaves are commonly used for smoking, chewing, or snuffing. The addictive properties of tobacco can be attributed to the presence of nicotine, a highly addictive substance found in tobacco leaves. When tobacco is consumed, nicotine enters the bloodstream and binds to nicotine receptors in the brain. This binding of nicotine to receptors in the brain triggers the release of dopamine, a neurotransmitter associated with pleasure and reward [31]. The Addictive Nature of Tobacco addiction occurs when an individual becomes dependent on nicotine, the addictive substance in tobacco. Nicotine addiction is a gripping and multifaceted disease that is increasing in our global population.

Nicotine addiction is characterized by compulsive and repetitive use of tobacco products, despite the negative health consequences. Nicotine addiction is a complex disease that is characterized by compulsive and repetitive use of tobacco products [4]. Tobacco contains various chemicals in addition to nicotine. These chemicals include tar, carbon monoxide, formaldehyde, hydrogen cyanide, and benzene, among others. These chemicals are harmful to human health and are known to cause various diseases, including cancer, cardiovascular diseases, respiratory disorders, and reproductive problems. The World Health Organization estimates that about 6million people die each year as a result of tobacco, yet 5million are active consumers and more than 600,000 are passive smokers. This makes tobacco one of the biggest threats to public health [32].

Tobacco smoking is known to have numerous adverse health effects, both on the individuals who smoke and on those who are exposed to secondhand smoke. These effects include an increased risk of developing various types of cancer, such as lung, throat, and mouth can-

cer. Furthermore, smoking is a major cause of cardiovascular diseases, including heart attacks and strokes. In addition to these serious health risks, smoking also contributes to respiratory diseases [7]. Secondhand smoke is defined as “the combination of smoke emitted from the burning end of a cigarette or other tobacco products and smoke exhaled by the smoker” (WHO,2007).Secondhand tobacco smoke exposure (SHS) remains a world issue, affecting 40% of children and 34% of non-smoking adults. This exposure leads to approximately 603,000 deaths attributable to conditions such as ischemic heart disease, respiratory infections, asthma, and lung cancer [24].

In 1986, when the US Surgeon General determined that Environmental Tobacco Smoke (ETS) was responsible for causing lung cancer in non-smokers without preexisting health conditions, it was based on 10 epidemiological studies conducted across different locations. These studies revealed an approximate 30% elevation in the risk of death due to ischemic heart disease or myocardial infarction among non-smokers who lived with smokers. Moreover, the larger studies also revealed a notable dose-response relationship, indicating that higher exposure to Environmental Tobacco Smoke was linked to a greater risk of death from heart disease. These findings were accompanied by various physiological and biochemical evidence illustrating the adverse impact of Environmental Tobacco Smoke on platelet function and its ability to harm arterial endothelium, there by increasing the risk of heart disease [15].

Out of the 1.1 billion global smokers, approximately 17% (equivalent to 189 million individuals) are occasional smokers, meaning they smoke on certain days of the month. This subset of non-daily smokers is becoming more prevalent among current smokers in the United States. While the count of daily cigarette smokers reduced from 37 million to 28 million between 2005 and 2015, the population of non-daily smokers who indulge in smoking on specific days of the month experienced a slight increase. Consequently, in 2015, an estimated 8.9 million people in the United States belonged to the category of non-daily cigarette smokers.The limited existing literature suggests that regular non daily cigarette smokers may have higher mortality rates than never smokers, even if they have never smoked every day [18].

## 1.2 Statement of the problem

Tobacco is one of the biggest public health threats the world has ever faced, killing over 8 million people a year around the world. More than 7 million of those deaths are the result of direct tobacco use while around 1.3 million are the result of non-smokers being exposed to second-hand smoke [17]. In 2000, around 32.7% of the global population (both sexes combined) and aged 15 years and older were current users of some form of tobacco. In 2020, maximum total tobacco use prevalence rate 18.7% for the total population aged 15 years and older, 29.6% for males and 7.8% for females [33].

Secondhand smoke is also one of the cause of respiratory disease. Secondhand smoker is a big problem in the world as the result of leading peoples to diseases and death. It come from the community surrounding by addicted smoker. Thirty minute of exposure to secondhand smoke can cause heart damage similar to that of habitual smokers. Worldwide, 40% of children, 33% of male nonsmokers and 35% of female nonsmokers were exposed to secondhand smoke. It is common in developing country also Ethiopia is one of the developing country, still many people are suffering with SHS here. The prevalence of SHS exposure among adolescents in Ethiopia is highest. Moreover, exposure to SHS at public places is much higher than at home. [1]

Secondhand smoke is the initial stage. Where a person is exposed to tobacco smoke indirectly by being around people who smoke. After exposure to SHS, a person may become occasional smoker. During this stage ,the person smoke occasionally and begins to associate smoking with certain activities,people or emotions. Then smoking becomes more frequent and regular. The individual smoke more consistently. In 2020, Fekede and Mebrate [14] studied Sensitivity and mathematical model analysis on secondhand smoking tobacco. In the model the impacts of occasional smoker is not consider. Thus, the present work is a modification of [14] by considering occasional smoker and the process of smoking addiction by adding new parameter. Due to this we applied  $SEI_1I_2R$  mathematical model to analyse the dynamics of tobacco smoking and in this research we will try to answer the following questions:

- How can a mathematical model for the analysis of smoking tobacco by considering occasional and habitual smokers be formulated?
- How to find equilibrium points and analyze their stability?

- How to determine the most sensitive parameter?
- How to estimate the parameters of the model?
- How can we interpret the model's solution for the analysis of smoking tobacco?

## **1.3 Objective of the study**

### **1.3.1 General objective**

The general objective of this study is to formulate and analyse a Mathematical Model on the dynamics of tobacco smoking by considering Occasional smokers and Habitual smokers.

### **1.3.2 Specific objective**

The specific objectives of the study are:

- To formulate a modified  $SEI_1I_2R$  Model of Smoking Tobacco by Considering Occasional Smokers and Habitual Smokers.
- To determine the smoking generation number for the model using the next generation matrix.
- To show local and global stability of smoking free and smoking present equilibrium point.
- Determine the sensitive parameter which has a great impact on the reproduction number.
- To simulate numerical solution using MATLAB software & ode45 and predict future dynamics of the model.

## **1.4 Significance of the Study**

By assessing the smoking generation number and other relevant parameters, this model can determine whether the smoking habit will expand throughout the population or fade away. Additionally, the findings of this study have the following significance:

- To identify the impact of treatment on smoking cessation.
- It used as an input for further investigation on smoking intervention strategies.
- To analysis spread of smoking tobacco could be used to predict smoking habit out break.
- To be used as a reference of related researches in the field of epidemiology.

## **1.5 Organization of the Thesis**

The thesis is organized as follows. Chapter (1) is the introduction which comprises the background of the study, statement of the problem, objectives and significance of the study. Chapter (2) presents review of the 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 this chapter. In this chapter, we also present numerical simulations by using MATHLAB software and ode 45 codes. Conclusions and recommendations of the study are given in Chapter (5).

# Chapter 2

## Literature Review

Many mathematical models have been proposed for tobacco smoking with assisting in the formulation of smoking control strategies. Here we have seen some of the literature on smoking models that are done before this study, and these are the guide to do our thesis.

[Maria Pulecio Montoya](#) (2019) around the world, one of the public health problems that has been recognized in recent years is smoking addict, which has developed into an epidemic causing many deaths [3]. Considering that People can be in one of two main states : Exposed and Infected. The first state is divided into two sub-states: passive smokers or those at risk of smoking,  $E_1$ ; and people who have stopped smoking but are at risk of relapsing,  $E_2$ . people who have stopped smoking have a chance to become addicted and they assume that the community of passive smoker move to addicted .The authors use SIS deterministic mathematical model and the qualitative analysis like smoking tobacco free equilibrium point ( $E_0$ ), endemic equilibrium point ( $E^*$ ), basic reproduction number  $R_0$ , were computed. From the stability analysis, they found that the smoking tobacco free equilibrium point was locally asymptotically stable if  $R_0 < 1$ . [26]

[Fekede and Mebrate](#) (2020) in Worldwide, 40% of children, 33% of male nonsmokers and 35% of female nonsmokers were exposed to secondhand smoke. The highest proportions exposed were estimated in Europe, the Western Pacific, and Southeast Asia, with more than 50% of population exposed. Proportion of people exposed was lowest in Africa [7]. In this article, the authors tried to show the effect of secondhand smoke in the society and the preparation of a mathematical model and interpret the model graphically People can be in one of the three groups: smoker,second hand smoker and who have stopped smoking. Developed a deterministic mathematical model of second hand smoke. Major qualitative analysis like the smoking tobacco free equilibrium point ( $E_0$ ), endemic equilibrium point ( $E^*$ ), basic reproduction number  $R_0$ , were computed. From the stability analysis, they found that the smoking tobacco free equilibrium point was locally asymptotically stable if  $R_0 < 1$ . The global asymptotic stability was established using Castillo-Chavez theorem. If  $R_0 > 1$  the unique endemic equilibrium was locally asymptotically stable.[14]

Z Alkudhari, S Al-Sheikh, S Al-Tuwairqi (2014) like many infectious disease, mathematical model can be used to understand the spread of smoking and to predict the impact of smoker on the community in order to help reducing the number of smokers. The authors introduced the new model depend on the model of [5] by dividing the smokers in to two sub classes : those are occasional smokers and heavy smokers and the impact of these two sub classes on the existence and stability of equilibrium point and analyze the model by using stability theory. The population divide in to four groups potential smoker, occasional(lights) smokers, smokers who temporary quit smoking. In this model they tried to treat occasional smokers and heavy smokers and ignore the permanent quitter. Proved the smoking free and endemic equilibrium point , local and global stability and numerical simulation.[2]

Castillo-Garsow et al. the first time in 2000 suggested a straight forward mathematical model for giving up smoking. They address a scheme with a total unchanging community which is split up into three classes: promise smokers, that is, persons who are not smoking but might become smokers in the future ( $S$ ), smokers ( $I_1$ ), and persons (former smokers) who have stop smoking lastingly ( $I_2$ ). The basic reproduction number and equilibria of the model were computed and the local stability of different equilibria is discussed in detail.[9]

Alkudhari et al. (2014) a mathematical model to analyze the behavior of smoking dynamics in a population with peer pressure effect on temporary quitters  $Q_t(t)$ . The total population could be split into four groups: those who don't smoke (nonsmokers) denoted by  $P(t)$ , smokers denoted by  $S(t)$ , smokers who have quit temporarily denoted by  $Q_t(t)$ , and smokers who have quit for good denoted by  $Q_p(t)$ . In simple words, the total population  $N(t)$  is the sum of nonsmokers, smokers, temporary quitters, and permanent quitters. To describe how these groups change over time regarding smoking habits, they concluded that the smoking-free equilibrium state is locally stable and the smoking-present equilibrium state is locally asymptotically stable.[3]

Though different researchers have done mathematical modeling on the impact of passive smoker, the other have done by considering occasional smokers and heavy smokers but they didn't consider passive smoker, occasional smokers and heavy smokers. In our case we have tried to consider the impact of second hand smoker, occasional and habitual smokers on the dynamics of tobacco smoking. This is the reason why we will develop a mathematical model on the impact of treatment on the dynamics of tobacco smoking by applying different way to minimizing tobacco smoking habit.

# Chapter 3

## Methodology of the Study

The mathematical model have be formulated using differential equations. The methods to be used in this study are to divide the population into five compartments( $S, E, I_o, I_h, R$ ), consisting of Susceptible, Second hand smoker, Occasional smokers, Habitual smokers and Recoverd groups and then to identify system model by differential equations, determination of equilibrium points and their stability is also performed as well as basic reproductive number is found. We analyze the local stability behavior of equilibria by using Routh-Hurwitz stability criteria and center manifold theory. We analyze the global stability of equilibria by using Castillo-Chavez theorem. Simulate numerical solution using MATLAB software & ode45 and predict future dynamics of the model.

### 3.1 System of Ordinary Differential Equations

**Definition 3.1.1 :** (Autonomous System of Ordinary Differential Equations)

An autonomous n-dimensional system of ordinary differential equation has the form :

$$\begin{aligned}\dot{x}(t) &= f(x(t)) \\ x(t_o) &= x_o\end{aligned}\tag{3.1.1}$$

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

**Definition 3.1.2 :** [11] (Well-posedness)

An initial value problem, (IVP) given in (3.1.1) is mathematically said to be well-posed if the followings conditions hold:

1. Its solution exists,
2. Its solution is unique,
3. Its solution continuously depends on the initial conditions.

The following additional condition is also important as we are dealing with populations:

4. The solution should be non-negative over time,
5. The solution should be non-negative over time and should be bounded.

**Definition 3.1.3:** [35] (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 3.1.4:** (Positivity of Solutions)

The solution of a given autonomous system, (3.1.1) is said to be positive, if all trajectories  $x(t)$  is positive for any  $t \geq 0$ .

**Definition 3.1.5:** [27] (Boundedness of Solutions)

The positive solution of an autonomous system, (3.1.1) is said to be bounded if any solution.  $x(t, t_0, x_0)$  of (3.1.1) satisfies;

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

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

**Theorem 3.1.1:[20] Linear Equations: Integrating factor method**

We consider the equation

$$\frac{dy}{dt} = f(t, y)$$

The function  $f(t, y)$  is a linear function in  $y$ , that is, we can write

$$f(t, y) = -p(t)y + q(t) :$$

So we will study the equation

$$\begin{aligned} \frac{dy}{dt} + p(t)y &= q(t) \\ \frac{dy}{dt} &= f(t, y) \end{aligned} \tag{3.1.2}$$

Is said to be well-posed if its solution exists, unique and continuously depends on its initial

values. We introduce the method of integrating factors; we multiply the equation  $\frac{dy}{dt} + p(t)y = q(t)$  by a function  $I(t)$  on both sides

$$I(t)\frac{dy}{dt} + I(t)p(t)y = I(t)q(t)$$

The function  $I$  is chosen such that the equation is integrable, meaning the LHS (Left Hand Side) is the derivative of something. In particular, we require:

$$\frac{dy}{dt} + p(t)y = \frac{d}{dt}(I(t)y) \Rightarrow I(t)\frac{dy}{dt} + I(t)p(t)y = I(t)\frac{dy}{dt} + \frac{I(t)}{dt}y$$

This requires

$$\frac{dI}{dt} = I(t)p(t) \Rightarrow \frac{dI}{I} = p(t)dt$$

Integrating both sides, we get

$$\int \frac{dI}{I} = \int p(t)dt \Rightarrow \ln I(t) = \int p(t)dt \text{ which gives a formula to compute}$$

$$I(t) = e^{\int p(t)dt}$$

Therefore, this  $I$  is called the integrating factor. Putting back into equation

$$\frac{dy}{dt} + p(t)y = q(t), \text{ we have got}$$

$$\frac{d}{dt}(I(t)y) = I(t)p(t)$$

$$\Rightarrow I(t)y = \int I(t)q(t)dt + c$$

Which gives the formula for the solution?

$$y(t) = \frac{1}{I(t)} \left\{ \int I(t)q(t)dt + c \right\} \text{ where } I(t) = e^{\int p(t)dt}$$

$$y(t) = e^{-\int p(t)dt} \left\{ \int e^{\int p(t)dt} q(t)dt + c \right\}$$

The first order, linear homogeneous differential equation has the form:

$$\frac{dy}{dt} + p(t)y = 0, \text{ then the solution has}$$

$$Y(t) = ce^{-\int p(t)dt}, \text{ where 'c' is an arbitrary constant of integration.}$$

## 3.2 Stability Analysis of Equilibrium Points

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

**Definition 3.2.1:** [4] Given the autonomous system (3.1.1), a state  $x^*$  is said to be an equilibrium point of the system if  $f(x^*) = 0$ .

**Definition 3.2.2:** [25] The solution  $x^*$  is said to be stable if for every  $\epsilon > 0$ , there exists a  $\delta = \delta(\epsilon) > 0$  such that  $|x^* - x_o| < \delta \Rightarrow |x^* - x(t)| < \epsilon, t > t_o \in \mathbb{R}$ , for every solution  $x(t)$  of (3.1) with  $x(t_o) = x_o$ .

**Definition 3.2.3:** [22] An equilibrium point (3.1.1) is said to be  $x^*$  locally asymptotically stable if it is locally stable and every trajectory that starts sufficiently close to towards  $x^*$  as  $t \rightarrow \infty$ , a steady state  $x^*$  which is not stable is said to be unstable.

**Definition 3.2.4:** [22] An equilibrium point  $x^*$  is said to be global asymptotically stable if it is asymptotically stable for all initial condition  $x^* \in \mathbb{R}^n$ .

**Definition 3.2.5:** [22] An equilibrium point of a give 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:** [25] An equilibrium point  $x^*$  is said to be global stable if all trajectories converge to  $x^*$ , i.e  $\lim_{t \rightarrow \infty} x(t) = x^*$ .

### 3.2.1 Local Stability by Linearization

**Definition 3.2.7:** [28] The Jacobian matrix associated to the system (3.1.1) at the equilibrium point  $x^*$ , which is denoted by  $D f(x^*)$ , is given by the matrix;

$$Df(x^*) = \left[ \frac{\partial f_i(x^*)}{\partial x_j} \right] = \begin{bmatrix} \frac{\partial f_1(x^*)}{\partial x_1} & \frac{\partial f_1(x^*)}{\partial x_2} & \dots & \frac{\partial f_1(x^*)}{\partial x_n} \\ \frac{\partial f_2(x^*)}{\partial x_1} & \frac{\partial f_2(x^*)}{\partial x_2} & \dots & \frac{\partial f_2(x^*)}{\partial x_n} \\ \vdots & \vdots & \dots & \vdots \\ \frac{\partial f_n(x^*)}{\partial x_1} & \frac{\partial f_n(x^*)}{\partial x_2} & \dots & \frac{\partial f_n(x^*)}{\partial x_n} \end{bmatrix} \text{ Where, } i, j = 1, 2, 3, 4, \dots, n.$$

**Proposition 3.2.8:** (21) An equilibrium point  $x^*$  of the dynamical system (3.1) is locally asymptotically stable if all the 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 Global Stability

Here we will present the most two famous results used in epidemiological modeling to establish the global stability.

#### **Theorem 3.2.1: [8] (Castillo Chavez Theorem)**

Assume that the system (3.1) can be rewritten in the form:

$$\frac{dx}{dt} = F(x, I) \tag{3.2.1}$$

$$\frac{dI}{dt} = G(x, I), G(x, 0) = 0 \tag{3.2.2}$$

Where  $X \in \mathbb{R}^m$  (represents the classes of non-smoker population individuals) and  $I \in \mathbb{R}^n$  (represents the classes of smoker population individuals). Assume that  $G(x, 0) = 0$  and  $E_0 = (x^*, 0)$  let be a steady state (the smoking free equilibrium point) of (3.1).

If the following conditions are satisfied:

- For the system  $\frac{dx}{dt} = F(x, 0)$ , the steady state  $x^*$  is globally asymptotically stable.
- $G(x, I) = AI - \tilde{G}(x, I)$ ,  $\tilde{G}(x, I) \geq 0$  for  $(x, I) \in G$ , where A is a Metzler matrix (the diagonal elements of A are non-negative) and G is the region where the model makes biological sense. Then the steady state  $E_0 = (x^*, 0)$  is globally asymptotically stable for the system (3.2 - 3.3) provided that the smoking generation number of the model is less than one.

### 3.2.3 Routh-Hurwitz stability criterion

The Routh-Hurwitz Criteria is used to determine local stability of smoking present equilibrium point for a non-linear system of differential equation. We used Routh-Hurwitz stability criterion method for determining the stability of linear continuous data systems without involving root solving. It gives the necessary and sufficient condition for all roots of the characteristic polynomial to have negative real parts, thus implying asymptotic stability.

Consider the characteristic equation of degree n given by [19]

$$p(\lambda) = \lambda^n + a_1\lambda^{n-1} + a_2\lambda^{n-2} + \dots + a_{n-1}\lambda + a_n = 0$$

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

$$H_1 = [a_1], H_2 = \begin{bmatrix} a_1 & 1 \\ 0 & a_2 \end{bmatrix}, H_3 = \begin{bmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{bmatrix}, \dots, H_n = \begin{bmatrix} a_1 & 1 & 0 & 0 & \dots & 0 \\ a_3 & a_2 & a_1 & 1 & \dots & 0 \\ a_5 & a_4 & a_3 & a_2 & \dots & 0 \\ \vdots & \vdots & \vdots & \vdots & \dots & \vdots \\ 0 & 0 & 0 & 0 & \dots & a_n \end{bmatrix}$$

Where if  $a_j = 0$  if  $j > n$ . 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.

That is  $\det H_j > 0$  for all  $j = 1, 2, 3, \dots, n$ .

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

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

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

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

$$n = 5 : a_j > 0, i = 1, 2, 3, 4, 5; (a_1 a_2 a_3 > a_3^2 + a_1^2 a_4) ((a_1 a_4 - a_5)(a_1 a_2 a_3 - a_3^2 - a_1^2 a_4) > a_5 (a_1 a_2 - a_3)^2 + a_5^2 a_1)$$

## 3.3 Basic concepts in epidemiological modeling

**Definition 3.3.1:** Epidemiology is a study of disease, the cause of their occurrence and spread in space and time. Epidemiological is the study of the distribution and determinants of health related states or events in a specified population of this study to the prevention and control of health populations.

### 3.3.1 Smoking Free Equilibrium Point (SFEP)

We know that the population is smoking free if a tobacco smoking habit can disappear completely from the population. In epidemiological modeling, a smoking free equilibrium (SFE) is a steady state in which the coordinates in the smoker compartments are zero. If the smoking free equilibrium is stable, then it is expected that the population will be smoking free over time.

### 3.3.2 Smoking Present Equilibrium Point (SPEP)

A smoking present equilibrium point (SPEP) is a steady state in which at least one of its coordinates in the smoker compartment is non-zero. It is a steady state solution where the smoking habit persists in the population.

### 3.3.3 Smoking Reproduction Number

**Definition 3.3.2 :** The basic reproduction number, denoted by  $R_0$ , is defined as the average number of secondary smokers that are produced when a single smoker individual is introduced into a population of purely susceptible individuals.  $R_0$  is a very important parameter that helps us to determine whether tobacco smoking habit spreads in the population or ends up. Based on the value of  $R_0$  we have the following conclusions;

- If  $R_0 < 1$ , then on average, a smoker individual produces less than one new smoker over the course of the smoking period and the habit of smoking cannot grow.
- If  $R_0 > 1$ , then each smoker individual produces, on average, more than one new smoker and the smoking habit can spread in to the population.

- But if  $R_0 = 1$ , then the smoking habit remains at a consistent rate.

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  $x \in R^n$  and  $y \in R^m$  be sub populations in each compartment. Further denote by  $F_i$  the rate of secondary infection increase in the  $i^{th}$  compartment. and  $V_i$  the disease progression rate ,death and recovery decrease the  $i^{th}$  compartment. The compartment model can be written as:

$$\begin{aligned}\frac{dx_i}{dt} &= f_i - v_i(x, y), i = 1, 2, 3, \dots, n \\ \frac{dy_j}{dt} &= G_j(x, y), j = 1, 2, \dots, m\end{aligned}$$

Denoting F and V the matrices,

$$\begin{aligned}F &= \left( \frac{\partial f_i}{\partial x_j(0, y^*)} \right) 1 \leq i, j \leq n \\ V &= \left( \frac{\partial v_i}{\partial x_j}(0, y^*) \right) 1 \leq i, j \leq n\end{aligned}$$

then  $FV^{-1}$  is called the next generation matrix. The basic reproduction number is the spectral radius (dominant eigenvalue) of the matrix  $FV^{-1}$  that is  $R_0 = \rho(FV^{-1})$ .

**Definition 3.3.3:** Compartment is a group of population with similar status.

**Definition 3.3.4:** Deterministic model is a mathematical model in which individuals in the population are assigned to different subgroups or compartments.

**Definition 3.3.6:** Treatment is a means of helping smokers stop or quit smoking tobacco by applying some behavioral and pharmacological therapies.[16]

**Definition 3.3.7:** Susceptible are individuals who are not smoking tobacco for the time being but they will smoke it in the future.

**Definition 3.3.8:** smoke generated by the burning tip of a cigarette or other smoked tobacco product [24]. And mainstream smoke exhaled by a smoker,a mixture now referred to as "secondhand smoke" or"environmental tobacco smoke"[29]

**Definition 3.3.9:** Occasional smoker, who has smoked at least 100 cigarettes in his or her lifetime, who smokes now, but does not smoke every day.

**Definition 3.3.10:** Smoker is a person who smokers tobacco regularly.

**Definition 3.3.11:** Recovered is the number of people in the removed compartment where the

people are considered to be recovered from the endemic or died.

### 3.4 Invariant Region

A set  $G$  is an invariant set with respect to a system of ordinary differential equations  $\dot{x} = f(x)$  if  $x(0) \in G \Rightarrow x(t) \in G$ , for all  $t \in R$ .

A set  $M$  is a positively invariant set with respect to  $\dot{x} = f(x)$  if  $x(0) \in G \Rightarrow x(t) \in G$ , for all  $t \geq 0$ .

### 3.5 Bifurcation in disease transmission models

When the value of  $R_0$  increases from  $R_0 < 1$  to  $R_0 > 1$ , behaviors of solutions to system of equation in the feasible region undergo qualitative changes in both the number or type of equilibria and their stability. We say that system undergoes a bifurcation and  $R_0 = 1$  is the bifurcation value. Bifurcation occurs when a parameter changes causes the stability of an equilibrium point or the number or type of equilibria:

#### Forward bifurcation

Typically, when  $R_0 < 1$  in this case, the corresponding (DFE) is asymptotically-stable. On the other hand, the disease will persist if  $R_0 > 1$ , where a stable endemic equilibrium exists. This phenomenon, where the DFE loses its stability and a stable EE appears as increases through one, is known as forward bifurcation. In the case of forward bifurcation there is no positive (endemic) equilibria near the DFE when  $R_0 < 1$  ( in this setting, the DFE is often the only equilibrium when  $R_0 < 1$  ); If there is a forward bifurcation at  $R_0 = 1$ , it is not possible for a disease to invade a population if  $R_0 < 1$ , If there is a forward bifurcation at  $R_0 = 1$  then there will be a low level of endemicity when  $R_0$  is slightly above unity.[23]

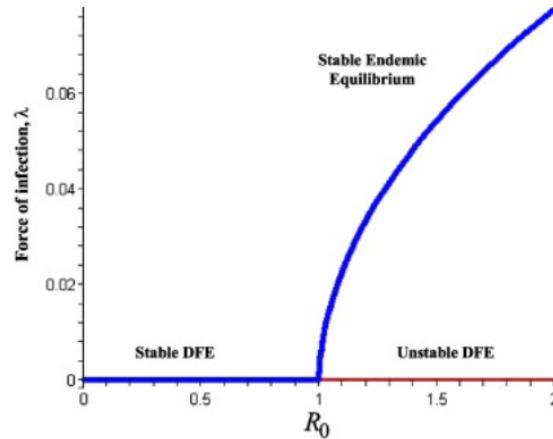


Figure 3.1: Forward bifurcation.

### Backward bifurcation

Another type of bifurcation, known as backward bifurcation, where a stable endemic equilibrium co-exists with a stable DFE when  $R_0 < 1$ . If there is a backward bifurcation at  $R_0 = 1$ , then the disease may spread even though  $R_0 < 1$ . In a backward bifurcation setting, once  $R_0$  crosses unity, the disease can invade to a relatively high endemic level. If there is a unique EE point when  $R_0 > 1$ , hence there is no possibility of backward bifurcation at  $R_0 = 1$  i.e there can not be an endemic equilibrium when  $R_0 < 1$ . [25]

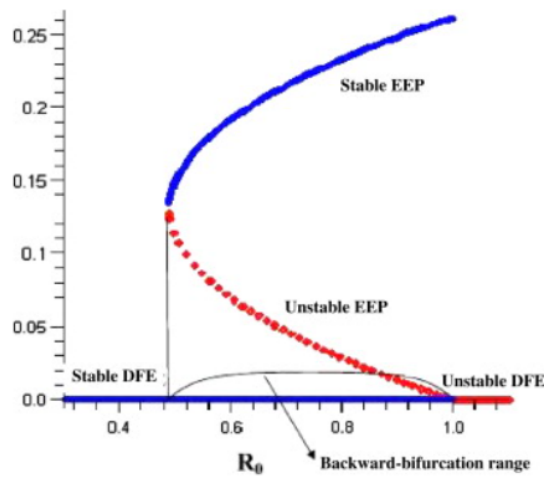


Figure 3.2: Backward bifurcation.

### 3.5.1 Center manifold theory

Center manifold theory has been used to decide the local stability of a non-hyperbolic equilibrium (linearization matrix has at least one eigenvalue with zero real part)[10]. We shall de-

scribe a theory that not only can determine the local stability of the non-hyperbolic equilibrium but also  $t$  settles the question of the existence of another equilibrium (bifurcated from the non-hyperbolic equilibrium). This theory is based on the general center manifold theory. To describe it, consider a general system of ODEs with a parameter  $\phi$ :

$$\frac{dx}{dt} = f(x, \phi), f : \mathbb{R}^n \times \mathbb{R} \rightarrow \mathbb{R}^n \text{ \& } f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R}). \quad (3.5.1)$$

Without loss of generality, it is assume that 0 is an equilibrium for system (3.2.3) for all values of the parameter  $\phi$ , that is

$$f(0, \phi) = 0 \text{ for all } \phi. \quad (3.5.2)$$

**Theorem 3.2.2 :** [11] Assume

- $A_1 : D_x f(0, 0) = (\frac{\partial f_i}{\partial x_j}(0, 0))$  is the linearization matrix of system (3.2.3) around the equilibrium 0 with  $\phi$  evaluated at 0. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;
- $A_2$ : Matrix A has a non-negative right eigenvector  $w$  and  $a$  left eigenvector  $v$  corresponding to the zero eigenvalue.

Let  $f_k$  be the  $k^{th}$  component of  $f$  and

$$a = \sum_{k,i,j=1}^n v_k \omega_i \omega_j \frac{\partial f_k}{\partial y_i \partial y_j}(0, 0) \quad (3.5.3)$$

$$b = \sum_{k,i=1}^n v_k \omega_i \frac{\partial f_k}{\partial y_i \partial \beta_1}(0, 0) \quad (3.5.4)$$

The local dynamics of system (3.2.3) around 0 is totally determined by the signs of  $a$  and  $b$ :

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

- $a > 0, b < 0$ . When  $\phi < 0$  with  $|\phi| \ll 1$ , 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when  $0 < \phi \ll 1$ , 0 is stable, and a positive unstable equilibrium appears;
- $a < 0, b > 0$ . When  $\phi$  changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if  $a < 0, b > 0$  then a forward bifurcation occurs at  $\phi = 0$ ; if  $a > 0, b > 0$  then a backward bifurcation occurs at  $\phi = 0$ .

### 3.6 Sensitivity Analysis

Sensitivity analysis is used to determine the relative importance of model parameters to disease transmission. We perform the analysis by calculating the sensitivity indices of the basic reproduction number,  $R_0$ , because it determines whether or not the infectious disease will spread in the population. Sensitivity analysis is commonly used to determine the robustness of model predictions to parameter values, since there usually errors in data collection and pre-assumed values. It also allows for the measurement of relevant changes in a state variable when a parameter changes. In performing the sensitivity analysis, we apply the method called normalized forward sensitivity index of a variable that has been used quite commonly, and it is defined as the ratio of relative change in the variable to the relative change in the parameter. The sensitivity may also be defined using partial derivatives when the variable is a differentiable function of the parameter.[12]

**Definition 3.7:** : [12] The normalized forward sensitivity index of an invariant, ‘k’ that depends on parameter ‘b’ is defined as:

$$r_b^k = \frac{\partial k}{\partial b} X \frac{b}{k}$$

# Chapter 4

## Mathematical Model Formulation and Analysis

### 4.1 Existing Model

Fekede and Mebrate [14] have developed a deterministic mathematical model of second hand smoking. They considered three (3) compartmental models in the second hand tobacco smoker in a community. The model comprises of addicted individuals  $S(t)$ , second hand smoker individuals  $P(t)$  and quitter individuals  $Q(t)$  so that

$$N(t) = P(t) + S(t) + Q(t)$$

The population second hand smoker individual increase by the new exposure immigrants that joins the compartment at rate of  $\alpha$ . Reduced by the natural death rate ( $\mu P$ ) and decreased by exist of second hand smoker to the healthy people (the rate of  $\sigma$ ) and death of second hand smoker because of being second hand smoker (rate of  $\nu$ ). Finally decreased by a person move to active addicted group (rate of  $\lambda$ ).

$$\frac{dP}{dt} = \alpha - (\nu + \mu + \sigma + \lambda S)P$$

The Addicted individual increased by the number of addicted community by second hand smoker (rate of  $\lambda$ ) and the person move quitter to addicted (rate of  $\delta Q$ ). The population reduced by who has stopped smoking (rate of  $\zeta$ ), the natural death rate of  $\mu S$  and death for reason of smoking habit rate of the parameter  $\kappa$ .

$$\frac{dS}{dt} = \lambda P + \delta Q - (\mu + \kappa + \zeta)S$$

The quitter individuals increased by the person who has stopped smoking group from addicted at rate of  $\zeta$ . The population decreased by the person who has stopped smoking to the healthy population. The population quitter individual further reduced by natural death at rate of  $\mu$  and death by their smoking habit before at the rate of  $\rho$ .

$$\frac{dQ}{dt} = \zeta S - (\delta S + \eta + \mu + \xi)Q$$

The mathematical model constructed the form of

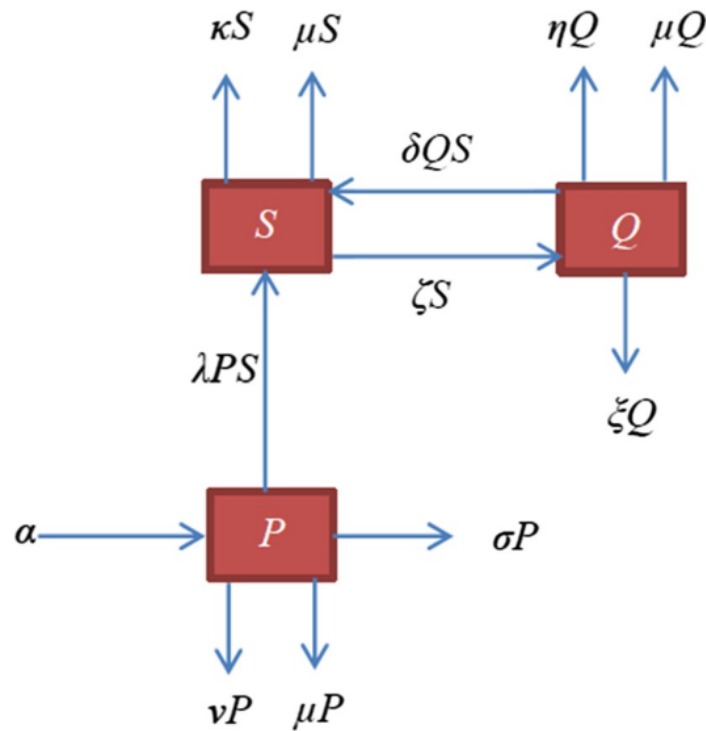


Figure 4.1: Existing model Flow chart.

$$\frac{dP}{dt} = \alpha - (v + \mu + \sigma + \lambda S)P \quad (4.1.1)$$

$$\frac{dS}{dt} = \lambda P + \delta Q - (\mu + k + \zeta)S \quad (4.1.2)$$

$$\frac{dQ}{dt} = \zeta S - (\delta S + \eta + \mu + \xi)Q \quad (4.1.3)$$

## 4.2 Modified Mathematical Model

We formulate a modified  $SEI_1I_2R$  Model of Smoking Tobacco by Considering Occasional Smokers and Habitual Smokers. We extend the existing model to include occasional Smokers and habitual Smokers.

### 4.2.1 Model Assumption

We consider the general model have developed based on the following assumptions

- All parameters in the model assumed to be non-negative.
- The natural death rates are assumed to be the same for all the compartments  $\mu > 0$ .
- There is death as a result of occasional smoking ,habitual smoking and second hand smoking.
- The second hand smoker individual can not influence the susceptible individual, but they are indirect smoker.
- susceptible group being smokers if and only if who have contact with Occasional and Habitual smokers.
- Treatment is considered for those most addicted groups.
- Smoking death rate for treatment group will be less.
- Smokers who stop permanently smoking will not return to susceptible.
- Age, sex, social status and race do not affect/considered/.

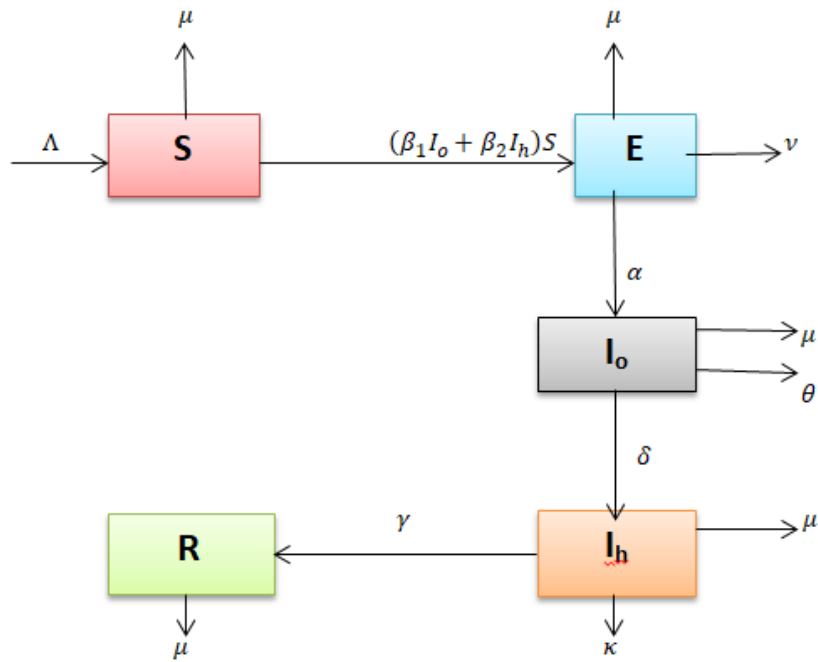


Figure 4.2: Modified model Flow chart.

#### 4.2.2 Model Formulation

In this model, we attempt to divide the population into five classes : susceptible ( $S$ ) which refers to the number of non-smokers individuals at time t, second hand smokers ( $E$ ) which refers to the number non-smoker but those at risk of others smoking, occasional smokers ( $I_o$ ) An adult who has smoked at least 100 cigarettes in his or her lifetime, who smokes now, but does not smoke every day , Habitual smokers ( $I_h$ ) those individual who are active smokers and who now smokes every day. Previously called a “regular smoker” , recovered group( $R$ ) constitutive which have recovery from the smoking habit. Based on the above assumption and flow chart in figure

4.2, we formulate the following system of ordinary differential equations(ODEs).

$$\frac{dS}{dt} = \Lambda - (\beta_1 I_o + \beta_2 I_h)S - \mu S \quad (4.2.1)$$

$$\frac{dE}{dt} = (\beta_1 I_o + \beta_2 I_h)S - (\alpha + \nu + \mu)E \quad (4.2.2)$$

$$\frac{dI_o}{dt} = \alpha E - (\delta + \theta + \mu)I_o \quad (4.2.3)$$

$$\frac{dI_h}{dt} = \delta I_o - (\gamma + \kappa + \mu)I_h \quad (4.2.4)$$

$$\frac{dR}{dt} = \gamma I_h - \mu R \quad (4.2.5)$$

$$S > 0, E \geq 0, I_o \geq 0, I_h \geq 0, R \geq 0$$

Description the variables and parameters of the model in the following tables, respectively..

Variable	Description
$S(t)$	Number of susceptible, they are non-smokers but they may become smoker future.
$E(t)$	Number of Second hand smoker(Environmental smoker)
$I_o(t)$	Number of Occasional(light) smoker,they smoke occasionally.
$I_h(t)$	Number of Habitual smoker, who are active smokers.
$R(t)$	Number of individuals recovered from smoking habit.

Table 4.1: Description of state variables.

Parameter	Description
$\Lambda$	Recruitment rate of humans (new birth rate)
$\beta_1$ & $\beta_2$	Contact rate
$\alpha$	Rate at which second hand smoker individuals become occasional smokers
$\gamma$	Treatment rate
$\delta$	Rate at which occasional smokers becomes habitual smoker
$\mu$	Rate of natural death for all compartments
$\kappa$	Death rate of habitual smokers as a consequence of smoking tobacco.
$\nu$	Death rate due to secondhand smoking tobacco
$\theta$	Death rate of occasional smokers as a consequence of smoking tobacco.

Table 4.2: Description of Parameters.

## 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 epidemiological appropriate in the sense 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 (4.2.1 - 4.2.5) are continuously differentiable. Thus, the existence and uniqueness of the solutions is established by the Picard's theorem (3.1.3). Now, we show the positivity and boundedness of solutions.

**Theorem 4.3.1:** If  $S(0) > 0$ ,  $E(0) \geq 0$ ,  $I_o(0) \geq 0$ ,  $I_h(0) \geq 0$  and  $R(0) \geq 0$  then the solution  $(S(t), E(t), I_o(t), I_h(t), R(t))$  of the dynamic system (4.2.1 - 4.2.5) is non-negative for all time  $t \geq 0$ .

**proof :** -In order to show that the positivity of each of the state variables for all  $t \geq 0$ . Let us use proof by contradiction for equations (4.2.1 - 4.2.5). To begin with the proof we take the negation of theorem (4.3.1) as an assumption. So let us assume that theorem (4.3.1) is not true. This implies, there are at least one or two state variables that violate theorem (4.3.1) at a given time  $t$ .

Let ,without loss of generality it is the state variable  $S(t)$  that violate theorem (4.3.1). First say at  $t$ . Now, if we analyses each of this state variables on the interval  $(0, t_0]$  we have the following results:

- $S(t_0) = 0$
- $S'(t_0) \leq 0$  and
- $S(t) > 0$  for  $t \in [0, t_0]$ , Then  $I_o(t) \geq 0$  and  $I_h(t) \geq 0$  for  $t \in [0, t_0]$ .

If this is not the case, then there exists:

- $t_1 \in [0, t_0]$  such that  $I_o(t_1) = 0$ ,  $I_o'(t_1) < 0$  and  $I_o(t) > 0$  for  $t \in [0, t_1]$ , Then  $I_h(t) \geq 0$  for  $t \in [0, t_0]$ .

- $t_2 \in [0, t_0]$  such that  $I_h(t_2) = 0$ ,  $I_h'(t_2) < 0$  and  $I_h(t) > 0$  for  $t \in [0, t_2]$ , Then  $I_o(t) \geq 0$  for  $t \in [0, t_2]$ .

First taking the left & right hand side of the differential equations (4.2.1) and evaluating at time  $t = t_0$  we have:

$$\left(\frac{dS}{dt}\right)|_{t=t_0} = S'(t_0) < 0 \quad (4.3.1)$$

$$= [\Lambda - (\beta_1 I_o + \beta_2 I_h + \mu)S]|_{t=t_0}$$

$$\frac{dS}{dt} = \Lambda - (\beta_1 I_o + \beta_2 I_h + \mu)S$$

$$\begin{aligned} S'(t_0) &= \Lambda - (\beta_1 I_o(t_0) + \beta_2 I_h(t_0) + \mu)S(t_0) \\ &= \Lambda > 0. \end{aligned} \quad (4.3.2)$$

since  $\Lambda > 0$  and  $(\beta_1 I_o(t_0) + \beta_2 I_h(t_0) + \mu)S(t_0) = 0$

From 4.3.1 & 4.3.2 we can see that

$$\left(\frac{dS}{dt}\right)|_{t=t_0} \neq [\Lambda - (\beta_1 I_o + \beta_2 I_h + \mu)S]|_{t=t_0}$$

This contradicts the fact that the differential equation (4.2.1) is satisfied for all  $t$  including  $t_0$ .

This contradiction results from our initial assumption, indicating that the assumption is false.

Consequently, Theorem 4.3.1 is true, and the state variable  $S(t)$  is positive for all  $t \geq 0$ .

Consider the differential equation (4.2.2):

$$\frac{dE}{dt} = (\beta_1 I_o + \beta_2 I_h)S - (\alpha + \nu + \mu)E$$

$$\frac{dE}{dt} \geq -(\alpha + \nu + \mu)E$$

$$\frac{dE}{E} \geq -(\alpha + \nu + \mu)dt$$

since  $S(t) = 0$

Now, integrating both sides of the inequality gives:

$$\int \frac{dE}{E} \geq - \int (\alpha + \nu + \mu)E$$

$$\ln E \geq -(\alpha + \nu + \mu)t + c$$

$$E(t) \geq e^{-(\alpha + \nu + \mu)t + c}, E(0) \geq e^c > 0.$$

$$E(t) \geq E(0)e^{-(\alpha + \nu + \mu)t} > 0.$$

Consider the differential equation (4.2.3):

$$\begin{aligned}\frac{dI_o}{dt} &= \alpha E - (\delta + \theta + \mu)I_o \\ \frac{dI_o}{dt} &\geq -(\delta + \theta + \mu)I_o, \quad E(0) > 0 \\ \frac{dI_o}{I_o} &\geq -(\delta + \theta + \mu)I_o\end{aligned}$$

Now, integrating both sides of the inequality gives:

$$\begin{aligned}\int \frac{dI_o}{I_o} &\geq \int -(\delta + \theta + \mu)dt \\ \ln I_o &\geq -(\delta + \theta + \mu)t + c \\ I_o(t) &\geq e^{-(\delta + \theta + \mu)t + c}, \quad I_o(0) \geq e^c > 0. \\ I_o(t) &\geq I_o(0)e^{-(\delta + \theta + \mu)t} > 0.\end{aligned}$$

Consider the differential equation (4.2.4):

$$\begin{aligned}\frac{dI_h}{dt} &= \delta I_o - (\gamma + \kappa + \mu)I_h \\ \frac{dI_h}{dt} &\geq -(\gamma + \kappa + \mu)I_h\end{aligned}$$

Now, integrating both sides of the inequality gives:

$$\begin{aligned}\int \frac{dI_h}{I_h} &\geq \int -(\gamma + \kappa + \mu)dt \\ \Rightarrow \ln I_h &\geq -(\gamma + \kappa + \mu)t + c \\ \Rightarrow I_h(t) &\geq e^{-(\gamma + \kappa + \mu)t + c}, \quad I_h(0) \geq e^c > 0. \\ \Rightarrow I_h(t) &\geq I_h(0)e^{-(\gamma + \kappa + \mu)t} > 0.\end{aligned}$$

Consider the differential equation (4.2.5):

$$\begin{aligned}\frac{dR}{dt} &= \gamma I_h - \mu R. \\ \frac{dR}{dt} &\geq -\mu R\end{aligned}$$

Now, integrating both sides of the inequality gives:

$$\begin{aligned} \int \frac{dR}{R} &\geq \int -\mu dt \\ \Rightarrow \ln R &\geq -\mu t + c \\ \Rightarrow R(t) &\geq e^{-\mu t}, R(0) \geq e^c > 0 \\ \Rightarrow R(t) &\geq R(0)e^{-\mu t} > 0. \end{aligned}$$

Thus  $R(t) > 0$  for all  $t > 0$ .

**Theorem 4.3.2:** There exists a unique and bounded solution of the system of equations (4.4-4.8) in a positively invariant set  $G = \{S(t), E(t), I_o(t), I_h(t), R(t) \in \mathbb{R}_+^5\}$  that remains for all finite time  $t \geq 0$ .

**proof :** In order to show that the population sizes of each compartment is bounded, we prefer to show that the total population size of the whole system  $N(t)$  is bounded. The total population size  $N$  is given by :

$$\begin{aligned} N(t) &= S(t) + E(t) + I_o(t) + I_h(t) + R(t) \\ \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_o}{dt} + \frac{dI_h}{dt} + \frac{dR}{dt} \\ \frac{dN}{dt} &= (\Lambda - (\beta_1 I_o + \beta_2 I_h)S - \mu S) + ((\beta_1 I_o + \beta_2 I_h)S - (\alpha + \nu + \mu)E) + \\ &\quad (\alpha E - (\delta + \theta + \mu)I_o) + (\delta I_o - (\gamma + \kappa + \mu)I_h) + (\gamma I_h - \mu R) \end{aligned}$$

After some steps of computations, we get:

$$\begin{aligned} &= \Lambda - \mu(S + E + I_o + I_h + R) - \nu E - \theta I_o - \kappa I_h \\ &= \Lambda - \mu N - \nu E - \theta I_o - \kappa I_h \\ &\leq \Lambda - \mu N \end{aligned}$$

since  $E, I_o$  and  $I_h$  are positive.

$$\begin{aligned} \frac{dN}{dt} + \mu N &\leq \Lambda \\ e^{\mu t} \frac{dN}{dt} + e^{\mu t} \mu N &\leq e^{\mu t} \Lambda \end{aligned}$$

$$\begin{aligned} \frac{d}{dt}(Ne^{\mu t}) &\leq e^{\mu t} \Lambda \\ \int_0^t \frac{d}{dt}(Ne^{\mu t}) &\leq \int_0^t e^{\mu t} \Lambda \\ Ne^{\mu t} \Big|_0^t &\leq \frac{\Lambda}{\mu} e^{\mu t} \Big|_0^t \\ e^{\mu t} N(t) - N(0) &\leq \frac{\Lambda}{\mu} (e^{\mu t} - e^0) \\ e^{\mu t} N(t) &\leq N(0) - \frac{\Lambda}{\mu} + \frac{\Lambda}{\mu} e^{\mu t} \\ N(t) &\leq \left( N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t} + \frac{\Lambda}{\mu} \\ \lim_{t \rightarrow \infty} N(t) &\leq \lim_{t \rightarrow \infty} \left[ \left( N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t} + \frac{\Lambda}{\mu} \right] \\ \text{When } t \rightarrow \infty, N(t) &\rightarrow \frac{\Lambda}{\mu} \end{aligned}$$

$$0 < N(t) \leq \frac{\Lambda}{\mu}$$

Thus, the total population  $N(t)$  and each population classes are remain bounded for all time  $t \geq 0$ .

Therefore, the model (4.2.1 - 4.2.5) is well posed epidemiologically and mathematically in a positively invariant set  $G = \{(S(t), E(t), I_o(t), I_h(t), R(t)) \in \mathbb{R}_+^5 : 0 < N \leq \frac{\Lambda}{\mu}\}$ .

### 4.3.2 Smoking Free Equilibrium Point (SFEP)

Smoking free equilibrium point (SFEP) is denoted by  $E_0$ , is a steady state solution of the model. Indicating that there are no smokers. It is obtained by making the right-hand sides of the

system of equations of model (4.2.1 - 4.2.5) equal to zero. That is:

$$\frac{dS}{dt} = \Lambda - (\beta_1 I_o + \beta_2 I_h)S - \mu S = 0 \quad (4.3.3)$$

$$\frac{dE}{dt} = (\beta_1 I_o + \beta_2 I_h)S - (\alpha + \nu + \mu)E = 0 \quad (4.3.4)$$

$$\frac{dI_o}{dt} = \alpha E - (\delta + \theta + \mu)I_o = 0 \quad (4.3.5)$$

$$\frac{dI_h}{dt} = \delta I_o - (\gamma + \kappa + \mu)I_h = 0 \quad (4.3.6)$$

$$\frac{dR}{dt} = \gamma I_h - \mu R = 0 \quad (4.3.7)$$

The human population of smokers is located in compartments of smokers  $E$ ,  $I_o$  and  $I_h$ . Then, the smoking free equilibrium points  $E_0$ , of the model is given by the following theorem.

**Theorem 5.1** :The model given by a system of equations (4.2.1 - 4.2.5) has a unique feasible smoking free equilibrium point which is given by:

$$E_0 = (S^0, E^0, I_o^0, I_h^0, R^0) = \left( \frac{\Lambda}{\mu}, 0, 0, 0, 0 \right)$$

**proof:-** If there are no smokers, then we have :

$$E = I_o = I_h = 0 \quad (4.3.8)$$

consider differential equation (4.3.7) and substitute (4.3.8) we have

$$\gamma I_h - \mu R = 0$$

$$R = 0$$

consider differential equation (4.3.3)

$$\Lambda - (\beta_1 I_o + \beta_2 I_h)S - \mu S = 0$$

substituting (4.3.8) in to equation (4.3.3)

$$\Lambda - (\beta_1(0) + \beta_2(0))S - \mu S = 0$$

$$\Lambda - \mu S = 0$$

$$\Lambda = \mu S$$

$$S = \frac{\Lambda}{\mu}$$

By computing with some steps. We have

$$E^0 = I_o^0 = I_h^0 = 0$$

Hence the smoking free equilibrium point of the model exists, and is given by:

$$E_0 = (S^0, E^0, I_o^0, I_h^0, R^0) = \left( \frac{\Lambda}{\mu}, 0, 0, 0, 0 \right)$$

### 4.3.3 Smoking Reproduction Number (Ro)

#### Computation of Ro using Next Generation Matrix Method

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 [13], the basic reproduction number  $R_0$  can be calculated from the relation  $R_0 = \rho(FV^{-1})$ . 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  $x = (E, I, H)$ , then we obtain:

$$F(x) = \begin{bmatrix} (\beta_1 I_o + \beta_2 I_h) S \\ 0 \\ 0 \end{bmatrix}, V(x) = \begin{bmatrix} (\alpha + \nu + \mu) E \\ (\delta + \theta + \mu) I_o - \alpha E \\ (\gamma + \kappa + \mu) I_h - \delta I_o \end{bmatrix}$$

The Jacobian matrix to  $F$  and  $V$  are

$$F = \left[ \frac{\partial F_i(e_o)}{\partial x_j} \right] = \begin{bmatrix} \frac{\partial F_E}{\partial E} & \frac{\partial F_E}{\partial I_o} & \frac{\partial F_E}{\partial I_h} \\ \frac{\partial F_{I_o}}{\partial E} & \frac{\partial F_{I_o}}{\partial I_o} & \frac{\partial F_{I_o}}{\partial I_h} \\ \frac{\partial F_{I_h}}{\partial E} & \frac{\partial F_{I_h}}{\partial I_o} & \frac{\partial F_{I_h}}{\partial I_h} \end{bmatrix} = \begin{bmatrix} 0 & \beta_1 S^0 & \beta_2 S^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$V = \left[ \frac{\partial V_i(e_0)}{\partial x_j} \right] = \begin{bmatrix} \frac{\partial V_E}{\partial E} & \frac{\partial V_E}{\partial I_o} & \frac{\partial V_E}{\partial I_h} \\ \frac{\partial V_{I_o}}{\partial E} & \frac{\partial V_{I_o}}{\partial I_o} & \frac{\partial V_{I_o}}{\partial I_h} \\ \frac{\partial V_{I_h}}{\partial E} & \frac{\partial V_{I_h}}{\partial I_o} & \frac{\partial V_{I_h}}{\partial I_h} \end{bmatrix} = \begin{bmatrix} (\alpha + \nu + \mu) & 0 & 0 \\ -\alpha & (\delta + \theta + \mu) & 0 \\ 0 & -\delta & (\gamma + \kappa + \mu) \end{bmatrix}$$

Where  $k_1 = (\alpha + \nu + \mu)$ ,  $k_2 = (\delta + \theta + \mu)$  and  $k_3 = (\gamma + \kappa + \mu)$ . After some algebraic computations, the inverse of the matrix  $V$  is given by

$$V = \begin{bmatrix} k_1 & 0 & 0 \\ -\alpha & k_2 & 0 \\ 0 & -\delta & k_3 \end{bmatrix}$$

$$V^{-1} = \frac{Adj(V)}{det(V)}, det(V) \neq 0$$

$$Adj(V) = \begin{bmatrix} (\delta + \theta + \mu)(\gamma + \kappa + \mu) & 0 & 0 \\ \alpha(\gamma + \kappa + \mu) & (\alpha + \nu + \mu)(\gamma + \kappa + \mu) & 0 \\ \alpha\delta & \delta(\alpha + \nu + \mu) & (\alpha + \nu + \mu)(\delta + \theta + \mu) \end{bmatrix}$$

$$det(V) = (\alpha + \nu + \mu) (\delta + \theta + \mu) (\gamma + \kappa + \mu) = (k_1) (k_2) (k_3)$$

$$V^{-1} = \frac{1}{(k_1) (k_2) (k_3)} \begin{bmatrix} (\delta + \theta + \mu)(\gamma + \kappa + \mu) & 0 & 0 \\ \alpha(\gamma + \kappa + \mu) & (\alpha + \nu + \mu)(\gamma + \kappa + \mu) & 0 \\ \alpha\delta & \delta(\alpha + \nu + \mu) & (\alpha + \nu + \mu)(\delta + \theta + \mu) \end{bmatrix}$$

The next generation matrix is defined as:

$$FV^{-1} = \frac{1}{k_1 k_2 k_3} \begin{bmatrix} 0 & \beta_1 S^0 & \beta_2 S^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} k_2 k_3 & 0 & 0 \\ \alpha(k_3) & (k_1)(k_3) & 0 \\ \alpha\delta & \delta(k_1) & (k_1)(k_3) \end{bmatrix}$$

$$FV^{-1} = \frac{1}{k_1 k_2 k_3} \begin{bmatrix} \beta_1 S^0(\alpha k_3) + \beta_2 S^0(\alpha\delta) & \beta_1 S^0(k_1 k_3) + \beta_2 S^0(\delta k_1) & \beta_2 S^0(k_1 k_2) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$FV^{-1} = \frac{1}{k_1 k_2 k_3} \begin{bmatrix} \frac{\alpha \Lambda}{\mu} (\beta_1 k_3 + \beta_2 \delta) & \frac{\Lambda}{\mu} k_1 (\beta_1 k_3 + \beta_2 \delta) & \frac{\Lambda}{\mu} \beta_2 (k_1 k_2) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$FV^{-1} = \frac{1}{k_1 k_2 k_3} \begin{bmatrix} \frac{\alpha \Lambda}{\mu} (\beta_1 k_3 + \beta_2 \delta) - \lambda & \frac{\Lambda}{\mu} k_1 (\beta_1 k_3 + \beta_2 \delta) & \frac{\Lambda}{\mu} \beta_2 (k_1 k_2) \\ 0 & 0 - \lambda & 0 \\ 0 & 0 & 0 - \lambda \end{bmatrix} = 0$$

We find the eigenvalues of  $FV^{-1}$  by solving the characteristic equation  $|FV^{-1} - \lambda I| = 0$  as  $\lambda_1 = \frac{\alpha \Lambda (\beta_1 k_3 + \beta_2 \delta)}{\mu (k_1 k_2 k_3)}$ ,  $\lambda_2 = 0$  and  $\lambda_3 = 0$ .

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

$$R_0 = \rho(FV^{-1}) = \frac{\alpha \Lambda (\beta_1 (\gamma + \kappa + \mu) + \beta_2 \delta)}{\mu ((\alpha + \nu + \mu) (\delta + \theta + \mu) (\gamma + \kappa + \mu))}$$

#### 4.3.4 Smoking Present Equilibrium Point (SPEP)

Endemic equilibrium point is a steady state solution where the smoking persists in the population. In the presence of smoking in the population, there exist an equilibrium point called endemic equilibrium point denoted by  $e_1 = (S^*, E^*, I_o^*, I_h^*, R^*)$ . It can be obtained by setting each equation of the system (4.2.1 - 4.2.5) equal to zero:

**Theorem 4.3.5 :** A unique endemic equilibrium point  $e_1 = (S^*, E^*, I_o^*, I_h^*, R^*)$  exists and is positive if  $R_0 > 1$ .

**proof :**

$$\Lambda - (\beta_1 I_o^* + \beta_2 I_h^*) S^* - \mu S^* = 0 \quad (4.3.9)$$

$$(\beta_1 I_o^* + \beta_2 I_h^*) S^* - (\alpha + \nu + \mu) E^* = 0 \quad (4.3.10)$$

$$\alpha E^* - (\delta + \theta + \mu) I_o^* = 0 \quad (4.3.11)$$

$$\delta I_o^* - (\gamma + \kappa + \mu) I_h^* = 0 \quad (4.3.12)$$

$$\gamma I_h^* - \mu R^* = 0 \quad (4.3.13)$$

From equation (4.3.13) we have :

$$\begin{aligned}
 \gamma I_h^* - \mu R^* &= 0 \\
 \gamma I_h^* &= \mu R^* \\
 R^* &= \frac{\gamma}{\mu} I_h^*
 \end{aligned} \tag{4.3.14}$$

From equation (4.3.12) we have :

$$\begin{aligned}
 \delta I_o^* - (\gamma + \kappa + \mu) I_h^* &= 0 \\
 \delta I_o^* &= (\gamma + \kappa + \mu) I_h^* \\
 I_h^* &= \frac{\delta}{(\gamma + \kappa + \mu)} I_o^*
 \end{aligned} \tag{4.3.15}$$

From equation (4.3.11) we have :

$$\begin{aligned}
 \alpha E^* - (\delta + \theta + \mu) I_o^* &= 0 \\
 \alpha E^* &= (\delta + \theta + \mu) I_o^* \\
 E^* &= \frac{(\delta + \theta + \mu)}{\alpha} I_o^*
 \end{aligned} \tag{4.3.16}$$

From equation (4.3.10) we have :

$$\begin{aligned}
 (\beta_1 I_o^* + \beta_2 I_h^*) S^* - (\alpha + \nu + \mu) E^* &= 0 \\
 (\beta_1 I_o^* + \beta_2 I_h^*) S^* &= (\alpha + \nu + \mu) E^*
 \end{aligned}$$

Substituting (4.3.15 ) and (4.3.16 ) at (4.3.10 ) , then we have get :

$$\begin{aligned}
(\beta_1 I_o^* + \beta_2 \frac{\delta}{(\gamma + \kappa + \mu)} I_o^*) S^* &= (\alpha + \nu + \mu) \left( \frac{\delta + \theta + \mu}{\alpha} \right) I_o^* \\
(\beta_1 + \beta_2 \frac{\delta}{(\gamma + \kappa + \mu)}) I_o^* S^* &= (\alpha + \nu + \mu) \left( \frac{\delta + \theta + \mu}{\alpha} \right) I_o^* \\
(\beta_1 + \beta_2 \frac{\delta}{(\gamma + \kappa + \mu)}) S^* &= (\alpha + \nu + \mu) \left( \frac{\delta + \theta + \mu}{\alpha} \right) \\
\left( \frac{\beta_1(\gamma + \kappa + \mu) + \beta_2 \delta}{\gamma + \kappa + \mu} \right) S^* &= (\alpha + \nu + \mu) \left( \frac{\delta + \theta + \mu}{\alpha} \right) \\
S^* &= \frac{(\alpha + \nu + \mu) (\delta + \theta + \mu) (\gamma + \kappa + \mu)}{\alpha (\beta_1(\gamma + \kappa + \mu) + \beta_2 \delta)} \\
S^* &= \frac{S^0}{R_0}
\end{aligned} \tag{4.3.17}$$

From equation (4.3.9) we have :

$$\begin{aligned}
\Lambda - (\beta_1 I_o^* + \beta_2 I_h^*) S^* - \mu S^* &= 0 \\
\Lambda - (\beta_1 I_o^* + \beta_2 I_h^* + \mu) S^* &= 0 \\
\Lambda &= (\beta_1 I_o^* + \beta_2 I_h^* + \mu) S^* \\
S^* &= \frac{\Lambda}{(\beta_1 I_o^* + \beta_2 I_h^* + \mu)} \\
S^* &= \frac{\Lambda}{(\beta_1 I_o^* + \beta_2 \frac{\delta}{\gamma + \kappa + \mu} I_o^* + \mu)}
\end{aligned}$$

By substituting equations (4.3.17 ) in the above equation and some simplification we have :

$$\begin{aligned}
I_o^* &= \frac{\alpha \Lambda (\beta_1(\gamma + \kappa + \mu) + \beta_2 \delta) - \mu (\gamma + \kappa + \mu) (\alpha + \nu + \mu) (\delta + \theta + \mu)}{(\alpha + \nu + \mu) (\delta + \theta + \mu) (\beta_1(\gamma + \kappa + \mu) + \beta_2 \delta)} \\
I_o^* &= \frac{(R_0 - 1) (\mu (\gamma + \kappa + \mu))}{\beta_1(\gamma + \kappa + \mu) + \beta_2 \delta}
\end{aligned} \tag{4.3.18}$$

From equation (4.3.15 ) we have :

$$I_h^* = \frac{\delta}{(\gamma + \kappa + \mu)} I_o^*$$

By substituting equations (4.3.18) into equation (4.3.15 ) , we have

$$I_h^* = \frac{\delta}{(\gamma + \kappa + \mu)} \left( \frac{(R_0 - 1) (\mu (\gamma + \kappa + \mu))}{\beta_1(\gamma + \kappa + \mu) + \beta_2 \delta} \right) \tag{4.3.19}$$

From equation (4.3.16 ) we have :

$$E^* = \frac{(\delta + \theta + \mu)}{\alpha} I_o^*$$

By substituting equations (4.3.18 ) into equation (4.3.16 ), we have

$$E^* = \frac{(\delta + \theta + \mu)}{\alpha} \left( \frac{(R_0 - 1)(\mu(\gamma + \kappa + \mu))}{\beta_1(\gamma + \kappa + \mu) + \beta_2\delta} \right) \quad (4.3.20)$$

From equation (4.3.14 ) we have :

$$R^* = \frac{\gamma}{\mu} I_h^*$$

By substituting equations (4.3.18 ) into equation ( 4.3.14 ), we have

$$R^* = \frac{\gamma\delta}{\mu(\gamma + \kappa + \mu)} \left( \frac{(R_0 - 1)(\mu(\gamma + \kappa + \mu))}{\beta_1(\gamma + \kappa + \mu) + \beta_2\delta} \right) \quad (4.3.21)$$

Then the components  $S^*$ ,  $E^*$ ,  $I_o^*$ ,  $I_h^*$  and  $R^*$  are given by

$$\begin{aligned} S^* &= \frac{S^0}{R_0} \\ E^* &= \frac{(\delta + \theta + \mu)}{\alpha} \left( \frac{(R_0 - 1)(\mu(\gamma + \kappa + \mu))}{\beta_1(\gamma + \kappa + \mu) + \beta_2\delta} \right) \\ I_o^* &= \frac{(R_0 - 1)(\mu(\gamma + \kappa + \mu))}{\beta_1(\gamma + \kappa + \mu) + \beta_2\delta} \\ I_h^* &= \frac{\delta}{(\gamma + \kappa + \mu)} \left( \frac{(R_0 - 1)(\mu(\gamma + \kappa + \mu))}{\beta_1(\gamma + \kappa + \mu) + \beta_2\delta} \right) \\ R^* &= \frac{\gamma\delta}{\mu(\gamma + \kappa + \mu)} \left( \frac{(R_0 - 1)(\mu(\gamma + \kappa + \mu))}{\beta_1(\gamma + \kappa + \mu) + \beta_2\delta} \right) \end{aligned}$$

## 4.4 Stability Analysis of Equilibrium Points

### 4.4.1 Local Stability of Smoking free Equilibrium point

Mathematically the stability of equilibrium point can be analyzed using the linearized system at the equilibrium or in other words the Jacobian matrix of first derivatives of the right hand side of the differential equation. The eigen values of this matrix given us the information about

the stability of equilibrium and the behavior of solutions near the equilibrium point.[5]

Hence, the smoking free equilibrium point of our model is identified ,we need to show that its local stability, which is concerned with the behavior of the model solution near to smoking free equilibrium point by due to the presence of awareness creation. Local stability of smoking free the equilibrium point can be determined from the sign of the eigen values are negative or have negative real parts the smoking free equilibrium point of the system is locally asymptotically stable and unstable if at least one of the eigen values has positive real part.

**Theorem 4.4.2 :** The smoking free equilibrium point of model (4.2.1 - 4.2.5) is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .

**Proof :** The Jacobin matrix associated with the system of equations of model is:

$$J = \begin{pmatrix} -(\beta_1 I_o + \beta_2 I_h) - \mu & 0 & -\beta_1 S & -\beta_2 S & 0 \\ \beta_1 I_o + \beta_2 I_h & -(\alpha + \nu + \mu) & \beta_1 S & \beta_2 S & 0 \\ 0 & \alpha & -(\delta + \theta + \mu) & 0 & 0 \\ 0 & 0 & \delta & -(\gamma + \kappa + \mu) & 0 \\ 0 & 0 & 0 & \gamma & -\mu \end{pmatrix}$$

$$J(e_0) = \begin{pmatrix} -\mu & 0 & -\beta_1 S^0 & -\beta_2 S^0 & 0 \\ 0 & -(\alpha + \nu + \mu) & \beta_1 S^0 & \beta_2 S^0 & 0 \\ 0 & \alpha & -(\delta + \theta + \mu) & 0 & 0 \\ 0 & 0 & \delta & -(\gamma + \kappa + \mu) & 0 \\ 0 & 0 & 0 & \gamma & -\mu \end{pmatrix}$$

Now, we need to find all the Eigen values of  $J(e_0)$  by solving  $\det(J(e_0) - \lambda I) = 0$  for  $\lambda$  ,where  $\lambda$  is an Eigen value and  $I$  is a 5x5 identity matrix.

$$\begin{pmatrix} -\mu - \lambda & 0 & -\beta_1 S^0 & -\beta_2 S^0 & 0 \\ 0 & -(\alpha + \nu + \mu) - \lambda & \beta_1 S^0 & \beta_2 S^0 & 0 \\ 0 & \alpha & -(\delta + \theta + \mu) - \lambda & 0 & 0 \\ 0 & 0 & \delta & -(\gamma + \kappa + \mu) - \lambda & 0 \\ 0 & 0 & 0 & \gamma & -\mu - \lambda \end{pmatrix} = 0$$

$$-\mu - \lambda \begin{pmatrix} -(\alpha + \nu + \mu) - \lambda & \beta_1 S^0 & \beta_2 S^0 & 0 \\ \alpha & -(\delta + \theta + \mu) - \lambda & 0 & 0 \\ 0 & \delta & -(\gamma + \kappa + \mu) - \lambda & 0 \\ 0 & 0 & \gamma & -\mu - \lambda \end{pmatrix} + 0 + 0 + 0 + 0$$

After some computations the characteristic equation becomes:

$$[-\mu - \lambda][(-(\alpha + \nu + \mu) - \lambda)(-(\delta + \theta + \mu) - \lambda)(-(\gamma + \kappa + \mu) - \lambda)(-\mu - \lambda) - \beta_1 S^0 \alpha (-(\gamma + \kappa + \mu) - \lambda)$$

$$(-\mu - \lambda) + \beta_2 S^0 \alpha \delta (-\mu - \lambda)] = 0$$

$$\Rightarrow \lambda_1 = -\mu < 0$$

$$((-\alpha + \nu + \mu) - \lambda)(-(\delta + \theta + \mu) - \lambda)(-(\gamma + \kappa + \mu) - \lambda)(-\mu - \lambda) - \beta_1 S^0 \alpha (-(\gamma + \kappa + \mu) - \lambda)$$

$$(-\mu - \lambda) + \beta_2 S^0 \alpha \delta (-\mu - \lambda)] = 0$$

$$\Rightarrow \lambda_2 = -\mu < 0$$

$$(-(\alpha + \nu + \mu) - \lambda)(-(\delta + \theta + \mu) - \lambda)(-(\gamma + \kappa + \mu) - \lambda) - \beta_1 S^0 \alpha (-(\gamma + \kappa + \mu) - \lambda) + \beta_2 S^0 \alpha \delta$$

Let,  $k_1 = (\alpha + \nu + \mu)$

$$k_2 = (\delta + \theta + \mu)$$

$$k_3 = (\gamma + \kappa + \mu)$$

Finally, after some computations the characteristic polynomial becomes:

$$P(\lambda) = \lambda^3 + (k_1 + k_2 + k_3)\lambda^2 + (k_1 k_2 + k_1 k_3 + k_2 k_3 - \beta_1 S^0 \alpha)\lambda + k_1 k_2 k_3 - k_3 \beta_1 S^0 \alpha - \beta_2 S^0 \alpha \delta = 0.$$

$$P(\lambda) = a_0 \lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3$$

Now let  $a_0 = 1$

$$a_1 = (k_1 + k_2 + k_3)$$

$$a_2 = (k_1 k_2 + k_1 k_3 + k_2 k_3 - \beta_1 S^0 \alpha)$$

$$a_3 = (k_1 k_2 k_3 - k_3 \beta_1 S^0 \alpha - \beta_2 S^0 \alpha \delta)$$

Then ,

$$P(\lambda) = a_0 \lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0. \quad (4.4.1)$$

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 positives.

That is  $\det(H_j) > 0$  for all  $j = 1, 2, 3, 4$

For the above degree four (4) characteristics polynomial Routh-Hurwitz matrix can be constructed and its determinant is computed as follows:

$$H_1 = \begin{bmatrix} a_1 \end{bmatrix} \quad \det(H_1) = a_1 > 0$$

$$H_2 = \begin{bmatrix} a_1 & 1 \\ 0 & a_2 \end{bmatrix} \quad \det(H_2) = \begin{bmatrix} a_1 & 1 \\ 0 & a_2 \end{bmatrix} = a_1 a_2 > 0$$

if  $a_1 > 0$  and  $a_2 > 0$

$$H_3 = \begin{bmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{bmatrix} \quad \det(H_3) = \begin{bmatrix} a_1 & 1 & 0 \\ 0 & a_2 & a_1 \\ 0 & 0 & a_3 \end{bmatrix} = (a_1 a_2 a_3 - a_3^2) > 0$$

if  $a_i > 0, i = 1, 2, 3$  and  $a_1 a_2 > a_3$

Here

- $a_1 = (k_1 + k_2 + k_3) > 0$

$$a_1 = [(\alpha + \nu + \mu) + (\delta + \theta + \mu) + (\gamma + \kappa + \mu)] > 0$$

$$a_1 > 0$$

- $a_2 = (k_1 k_2 + k_1 k_3 + k_2 k_3 - \beta_1 S^0 \alpha) > 0$

$$a_2 = [(\alpha + \nu + \mu)(\delta + \theta + \mu) + (\alpha + \nu + \mu)(\gamma + \kappa + \mu) + (\delta + \theta + \mu)(\gamma + \kappa + \mu) - \frac{\beta_1 \alpha \Lambda}{\mu}]$$

$$= \frac{\mu(\alpha + \nu + \mu)(\delta + \theta + \mu) + \mu(\alpha + \nu + \mu)(\gamma + \kappa + \mu) + \mu(\delta + \theta + \mu)(\gamma + \kappa + \mu) - \beta_1 \Lambda \alpha}{\mu}$$

$$= \mu(\alpha + \nu + \mu)(\delta + \theta + \mu) - \beta_1 \Lambda \alpha - \frac{\alpha \Lambda \beta_2 \delta}{(\gamma + \kappa + \mu)} + \frac{\alpha \Lambda \beta_2 \delta}{(\gamma + \kappa + \mu)}$$

$$\text{and } (\alpha + \nu + \mu)(\delta + \theta + \mu) + (\alpha + \nu + \mu)(\gamma + \kappa + \mu) > 0$$

after some computations we have:

$$a_2 = \frac{(\gamma + \kappa + \mu)\mu((\alpha + \nu + \mu)(\delta + \theta + \mu))(1 - R_0) + \alpha \Lambda \beta_2 \delta}{(\gamma + \kappa + \mu)} > 0$$

- $a_3 = (k_1 k_2 k_3 - k_3 \beta_1 S^0 \alpha - \beta_2 S^0 \alpha \delta) > 0$

$$a_3 = [(\alpha + \nu + \mu)(\delta + \theta + \mu)(\gamma + \kappa + \mu) - ((\gamma + \kappa + \mu)\beta_1 S^0 \alpha) - \beta_2 S^0 \alpha \delta] > 0$$

$$a_3 = \frac{\mu(\alpha + \nu + \mu)(\delta + \theta + \mu)(\gamma + \kappa + \mu) - \alpha\Lambda(\beta_1(\gamma + \kappa + \mu) + \beta_2\delta)}{\mu}$$

$$a_3 = (1 - R_0)(\alpha + \nu + \mu)(\delta + \theta + \mu)(\gamma + \kappa + \mu) > 0$$

If  $R_0 < 1$  the coefficients  $a_1$ ,  $a_2$  and  $a_3$  are positive and  $a_1a_2 > a_3$ . Thus, all the eigenvalues of  $J(e_0)$  are negative. It follows by Routh-Hurwitz criteria that the smoking free equilibrium  $e_0$  is locally asymptotically stable for  $R_0 < 1$ .

#### 4.4.2 Global Stability of Smoking free Equilibrium point

**Theorem 4.4.3 :** For  $R_0 < 1$ , the smoking free equilibrium  $e_0$  of the system (4.2.1 - 4.2.5) is globally asymptotically stable if  $S^0 \geq S$ .

**proof :** Let us rewrite our model system (4.2.1 - 4.2.5) and apply the Castillo-Chavez theorem (3.2.1 ). From system , we have

$$\begin{aligned} \frac{dZ_1}{dt} &= F(Z_1, Z_2) \\ \frac{dZ_2}{dt} &= G(Z_1, Z_2), G(Z_1, 0) = 0. \end{aligned}$$

$$X = (S, R) = Z_1$$

$$I = (E, I_o, I_h) = Z_2$$

$$\frac{dZ_1}{dt} = F(Z_1, Z_2) = \begin{pmatrix} \Lambda - (\beta_1 I_o + \beta_2 I_h)S - \mu S \\ \gamma I_h - \mu R \end{pmatrix}$$

$$\frac{dZ_2}{dt} = G(Z_1, Z_2) = \begin{pmatrix} (\beta_1 I_o + \beta_2 I_h)S - (\alpha + \nu + \mu)E \\ \alpha E - (\delta + \theta + \mu)I_o \\ \delta I_o - (\gamma + \kappa + \mu)I_h \end{pmatrix}$$

**(I)** To show  $Z_1^*$  is globally asymptotically stable for the system  $\frac{dZ_1}{dt} = F(Z_1, 0)$ , let us consider the reduced system

$$\frac{dZ_1}{dt} = F(Z_1, 0) = \begin{pmatrix} \Lambda - \mu S \\ -\mu R \end{pmatrix} \quad (4.4.1)$$

We can rewrite the system (4.4.1) as:

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \mu S \\ \frac{dR}{dt} &= -\mu R\end{aligned}\tag{4.4.2}$$

The system (4.4.2) is non-homogeneous linear system of ordinary differential equations. By applying Theorem (3.2.1) for the system (4.4.2), we obtain solutions

$$\begin{aligned}S(t) &= \left( (S(0) - \frac{\Lambda}{\mu})e^{-\mu t} + \frac{\Lambda}{\mu} \right) \\ R(t) &= e^{-\mu t}\end{aligned}$$

Taking the limit as  $t$  goes to  $\infty$ , we obtain

$$(S(t), R(t)) \rightarrow \left( \frac{\Lambda}{\mu}, 0 \right) = Z_1^*$$

Therefore,  $Z_1^*$  is globally asymptotically stable for the system  $\frac{dZ_1}{dt} = F(Z_1, 0)$ .

**(II)** We will show that  $\hat{G}(Z_1, Z_2) = AZ_2 - G(Z_1, Z_2)$ ,  $\hat{G}(Z_1, Z_2) \geq 0$  for  $(Z_1, Z_2) \in \Omega$  where  $A = \frac{\partial G}{\partial Z_2}(Z_1^*, 0)$  is a Metzler matrix (the off diagonal elements of  $A$  are non-negative) and  $\Omega$  is the region where the model makes biological sense. Consider a matrix

$$A = \frac{\partial G}{\partial Z_2}(Z_1^*, 0) = \begin{pmatrix} -(\alpha + \nu + \mu) & \beta_1 S^0 & \beta_2 S^0 \\ \alpha & -(\delta + \theta + \mu) & 0 \\ 0 & \delta & -(\gamma + \kappa + \mu) \end{pmatrix}$$

Hence,  $A$  is a Metzler matrix (off diagonal elements are non-negative). Here,

$$\hat{G}(Z_1, Z_2) = AZ_2 - G(Z_1, Z_2)$$

$$\hat{G}(Z_1, Z_2) = \begin{pmatrix} -(\alpha + \nu + \mu)E + (\beta_1 I_o S^0 + \beta_2 I_h S^0) \\ \alpha E - (\delta + \theta + \mu)I_o \\ \delta I_o - (\gamma + \kappa + \mu)I_h \end{pmatrix} - \begin{pmatrix} (\beta_1 I_o + \beta_2 I_h)S - (\alpha + \nu + \mu)E \\ \alpha E - (\delta + \theta + \mu)I_o \\ \delta I_o - (\gamma + \kappa + \mu)I_h \end{pmatrix}$$

After some simplification, we obtain

$$\hat{G}(Z_1, Z_2) = \begin{pmatrix} (\beta_1 I_o + \beta_2 I_h) S^0 - (\beta_1 I_o + \beta_2 I_h) S \\ 0 \\ 0 \end{pmatrix}$$

$$\hat{G}(Z_1, Z_2) = (S^0 - S) \begin{pmatrix} (\beta_1 I_o + \beta_2 I_h) \\ 0 \\ 0 \end{pmatrix} \geq 0$$

Therefore by Castillo-Chavez theorem (3.2.1), the smoking free equilibrium point  $e_0$  of the system (4.2.1 - 4.2.5) is globally asymptotically stable for  $R_0 < 1$ .

We consider the force of infection  $\lambda$  which is given by

$$\lambda = (\beta_1 I_o + \beta_2 I_h) S$$

$\beta_1$  and  $\beta_2$  are contact rate they contact with occasional and habitual smoker transfer smoking habit. The force of infection can be updated as

$$\lambda^* = (\beta_1 I_o^* + \beta_2 I_h^*) S^* \quad (4.4.3)$$

when we substitute the expression for  $I_o^*$ ,  $I_h^*$  and  $S^*$  in to the force of infection  $\lambda^*$ , we obtain

$$\lambda^* = \frac{(\mu(\alpha + \nu + \mu)(\delta + \theta + \mu)(\gamma + \kappa + \mu))(R_0 - 1)}{\alpha(\beta_1(\gamma + \kappa + \mu) + \beta_2\delta)} \quad (4.4.4)$$

### 4.4.3 Local Stability of Smoking Present Equilibrium point

**Theorem 4.4.1:** The smoking present equilibrium point  $e_1$  of the system (4.2.1 - 4.2.5) is locally asymptotically stable if  $R_0 > 1$ .

**proof:** To determine the local stability of endemic equilibrium, we used the center manifold theory(3.2.1), by taking  $\beta_1$  as a bifurcation parameter. we make the following change of variables on the system (4.2.1 - 4.2.5). Let  $S = y_1, E = y_2, I_o = y_3, I_h = y_4$  and  $R = y_5$ . Moreover by using vector notation  $y = (y_1, y_2, y_3, y_4, y_5)^T$ , the system(4.2.2 - 4.2.5) can be written in the

form  $\frac{dy}{dt} = F(y)$  with  $F = (f_1, f_2, f_3, f_4, f_5)^T$ , as shown below:

$$\begin{aligned}
\frac{dy_1}{dt} &= \Lambda - (\beta_1 y_3 + \beta_2 y_4 + \mu) y_1 \\
\frac{dy_2}{dt} &= (\beta_1 y_3 + \beta_2 y_4) y_1 - (\alpha + \nu + \mu) y_2 \\
\frac{dy_3}{dt} &= \alpha y_2 - (\delta + \theta + \mu) y_3 \\
\frac{dy_4}{dt} &= \delta y_3 - (\gamma + \kappa + \mu) y_4 \\
\frac{dy_5}{dt} &= \gamma y_4 - \mu y_5
\end{aligned} \tag{4.4.5}$$

We choose  $\beta_1 = \beta^*$  as a bifurcation parameter. Solving for  $\beta^*$  from  $R_0 = 1$ , we obtain

$$\beta^* = \frac{(\mu(\alpha + \nu + \mu)(\delta + \theta + \mu)(\gamma + \kappa + \mu) - \alpha\Lambda\delta\beta_2)}{\alpha\Lambda(\gamma + \kappa + \mu)}$$

The Jacobian matrix of the system (4.30) evaluated at the disease free equilibrium  $e_0$  with  $\beta_1 = \beta^*$  is given by

$$J^* = \begin{pmatrix} -\mu & 0 & -\beta^* S^0 & -\beta_2 S^0 & 0 \\ 0 & -(\alpha + \nu + \mu) & \beta^* S^0 & \beta_2 S^0 & 0 \\ 0 & \alpha & -(\delta + \theta + \mu) & 0 & 0 \\ 0 & 0 & \delta & -(\gamma + \kappa + \mu) & 0 \\ 0 & 0 & 0 & \gamma & -\mu \end{pmatrix}$$

The Jacobian matrix  $J^*$  of the linearized system has a simple zero eigenvalue with all other eigenvalues having negative real part, hence the center manifold theory will be used to analyse the dynamics of the system near  $\beta_1 = \beta^*$ . Thus,  $e_0$  is a non-hyperbolic equilibrium, when  $\beta_1 = \beta^*$ . Now, we calculate a right eigenvector  $w = (\omega_1, \omega_2, \omega_3, \omega_4, \omega_5, \omega_6)^T$  of  $J^*$  associated with the zero eigenvalue.

Where  $k = (\alpha + \nu + \mu)$ ,  $h = (\delta + \theta + \mu)$  and  $p = (\gamma + \kappa + \mu)$

$$\begin{pmatrix} -\mu & 0 & -\beta^* S^0 & -\beta_2 S^0 & 0 \\ 0 & -k & \beta^* S^0 & \beta_2 S^0 & 0 \\ 0 & \alpha & -h & 0 & 0 \\ 0 & 0 & \delta & -p & 0 \\ 0 & 0 & 0 & \gamma & -\mu \end{pmatrix} \begin{pmatrix} \omega_1 \\ \omega_2 \\ \omega_3 \\ \omega_4 \\ \omega_5 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

This implies that,

$$\begin{aligned}
-\omega_1\mu - \omega_3\beta^*S^0 - \omega_4\beta_2S^0 &= 0 \\
-\omega_2\mu + \omega_3\beta^*S^0 + \omega_4\beta_2S^0 &= 0 \\
\omega_2\alpha - \omega_3h &= 0 \\
\omega_3\delta - \omega_4p &= 0 \\
\omega_4\gamma - \omega_5\mu &= 0
\end{aligned} \tag{4.4.6}$$

Solving system of equation (4.4.6), we obtain

$$\begin{aligned}
\omega_1 &= -\left(\frac{\beta^*pS^0 + \beta_2\delta S^0}{\mu p}\right)\omega_3 \\
\omega_2 &= \frac{h}{\alpha}\omega_3, \omega_3 = \omega_3 > 0 \\
\omega_4 &= \frac{\delta}{p}\omega_3 \\
\omega_5 &= \frac{\gamma\delta}{\mu p}\omega_3
\end{aligned}$$

$$\begin{pmatrix} -\mu & 0 & -\beta^*S^0 & -\beta_2S^0 & 0 \\ 0 & -k & \beta^*S^0 & \beta_2S^0 & 0 \\ 0 & \alpha & -h & 0 & 0 \\ 0 & 0 & \delta & -p & 0 \\ 0 & 0 & 0 & \gamma & -\mu \end{pmatrix}^T = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 \\ 0 & -k & \alpha & 0 & 0 \\ -\beta^*S^0 & \beta^*S^0 & -h & \delta & 0 \\ -\beta_2S^0 & \beta_2S^0 & 0 & -p & \gamma \\ 0 & 0 & 0 & 0 & -\mu \end{pmatrix}$$

The left eigenvectors of  $J^*$  associated with the zero eigenvalue is given by  $v = (v_1, v_2, v_3, v_4, v_5)^T$ , is calculated as:

$$\begin{pmatrix} -\mu & 0 & 0 & 0 & 0 \\ 0 & -k & \alpha & 0 & 0 \\ -\beta^*S^0 & \beta^*S^0 & -h & \delta & 0 \\ -\beta_2S^0 & \beta_2S^0 & 0 & -p & \gamma \\ 0 & 0 & 0 & 0 & -\mu \end{pmatrix} \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

This implies that,

$$\begin{aligned}
-v_1\mu &= 0 \\
-v_2k + v_3\alpha &= 0 \\
-v_1\beta^*S^0 + v_2\beta^*S^0 - v_3h + v_4\delta &= 0 \\
-v_1\beta_2S^0 + v_2\beta_2S^0 - v_4p + v_5\gamma &= 0 \\
-v_5\mu &= 0
\end{aligned} \tag{4.4.7}$$

Solving system of equation (4.4.7), we obtain

$$\begin{aligned}
v_1 = v_5 = 0, v_2 &= 1 \\
v_3 &= \frac{k}{\alpha}v_2, v_4 = \frac{\beta_2S^0}{p}v_2
\end{aligned}$$

Since the first and fifth component of  $v$  are zero, we don't need the partial derivatives of  $f_1$  and  $f_5$ . From the partial derivatives of  $f_2, f_3$  and  $f_4$  at the disease free equilibrium point, the only ones that are nonzero are the following:

$$\begin{aligned}
\frac{\partial^2 f_2}{\partial y_1 \partial y_3} = \frac{\partial^2 f_2}{\partial y_3 \partial y_1} &= \beta^*, \quad \frac{\partial^2 f_2}{\partial y_1 \partial y_4} = \frac{\partial^2 f_2}{\partial y_4 \partial y_1} = \beta_2 \\
\frac{\partial^2 f_2}{\partial y_1 \partial \beta_1} = \frac{\partial^2 f_2}{\partial \beta_1 \partial y_1} &= I_o, \quad \frac{\partial^2 f_2}{\partial y_3 \partial \beta_1} = \frac{\partial^2 f_2}{\partial \beta_1 \partial y_3} = S^0
\end{aligned}$$

Find the coefficients of  $a$  and  $b$

$$\begin{aligned}
a &= v_k \sum_{k,i,j=1}^n \omega_i \omega_j \frac{\partial f_k}{\partial y_i \partial y_j} (S^0, 0, 0, 0, 0) \\
&= 2v_2 (\omega_1 \omega_3 \frac{\partial f_2}{\partial y_1 \partial y_3} + \omega_1 \omega_4 \frac{\partial f_2}{\partial y_1 \partial y_4}) \\
&= 2v_2 (\omega_1 \omega_3 \beta_1 + \omega_1 \omega_4 \beta_2) \\
&= 2v_2 \omega_1 (\beta_1 + \frac{\delta}{p} \beta_2) \omega_3 \\
&= -2 \left( \frac{\beta_1 S^0}{\mu} + \frac{\beta_2 \delta S^0}{\mu p} \right) \left( \beta_1 + \frac{\beta_2 \delta}{p} \right) v_2 \omega_3^2 < 0. \\
b &= v_k \sum_{k,i=1}^n \omega_i \frac{\partial f_k}{\partial y_i \partial \beta_1} (S^0, 0, 0, 0, 0) \\
&= 2v_2 (\omega_1 \frac{\partial f_2}{\partial y_1 \partial \beta_1} + \omega_3 \frac{\partial f_2}{\partial y_3 \partial \beta_1}) \\
&= 2v_2 (\omega_1 I_o + \omega_3 S^0) \\
&= 2v_2 \omega_3 S^0 > 0.
\end{aligned}$$

Since  $a < 0$  and  $b > 0$  at  $\beta_1 = \beta^*$  Based on Theorem (3.2.1), the system (4.2.1 - 4.2.5) undergoes a forward bifurcation at  $R_0 = 1$  and the unique endemic equilibrium  $e_1$  is locally asymptotically stable for  $R_0 > 1$ .

There are two important quantities: the coefficients  $a$  and  $b$ , of the normal form representing the dynamics of the system on the central manifold. These coefficients decide the bifurcation. In particular, if  $a < 0$  and  $b > 0$ , then the bifurcation is forward. we have already justified the system (4.2.1 - 4.2.5) undergoes a forward bifurcation at  $R_0 = 1$ . The forward bifurcation diagram can be seen in Figure (4.3). If  $R_0 < 1$  the system has no endemic equilibrium point and the smoking free equilibrium is stable. If  $R_0 > 1$ , smoking present equilibrium point stable and smoking free equilibrium point unstable. Stability of the equilibrium's (forward bifurcation) occurs at the bifurcation point  $R_0^* = 1$ .

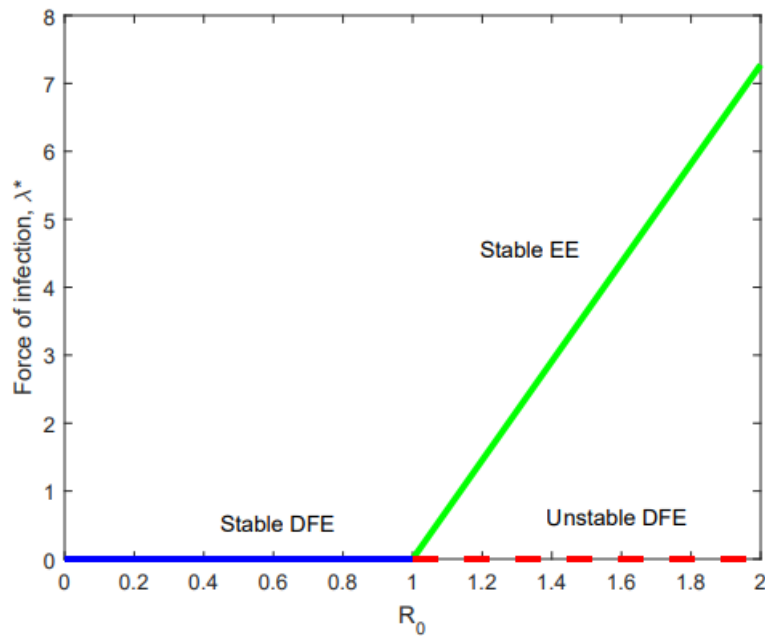


Figure 4.3: Forward bifurcation diagram for the Smoking tobacco model (4.2.1-4.2.1).

## 4.5 Sensitivity Analysis of $R_0$

In this section, 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 [21]. The basic reproduction number is pivotal in determining the stability of equilibrium points is a function of some parameters. It is important for us to understand the behavior of  $R_0$  with respect to the different parameters it depends on. which includes  $\beta_1, \beta_2, \alpha, \delta, \gamma, \nu, \theta, \kappa, \Lambda, \mu$  and the sensitivity of these parameters show us how they are important to the spread of tobacco smoking habit. Sensitivity indicates allow us to measure the relative change to a parameter change in when a parameter changes.

The normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter, which is given by the following

form.

$$\begin{aligned}
r_{\beta_1}^{R_0} &= \frac{\partial R_0}{\partial \beta_1} X \frac{\beta_1}{R_0} = \frac{\beta_1(\gamma + \kappa + \mu)}{\beta_1(\gamma + \kappa + \mu) + \beta_2\delta} \\
r_{\beta_2}^{R_0} &= \frac{\partial R_0}{\partial \beta_2} X \frac{\beta_2}{R_0} = \frac{\beta_2\delta}{\beta_1(\gamma + \kappa + \mu) + \beta_2\delta} \\
r_{\alpha}^{R_0} &= \frac{\partial R_0}{\partial \alpha} X \frac{\alpha}{R_0} = \frac{\nu + \mu}{(\alpha + \nu + \mu)} \\
r_{\delta}^{R_0} &= \frac{\partial R_0}{\partial \delta} X \frac{\delta}{R_0} = \frac{\delta(\beta_2(\delta + \theta + \mu) - \beta_1(\gamma + \kappa + \mu) + \beta_2\delta)}{(\delta + \theta + \mu)(\beta_1(\gamma + \kappa + \mu) + \beta_2)} \\
r_{\gamma}^{R_0} &= \frac{\partial R_0}{\partial \gamma} X \frac{\gamma}{R_0} = -\frac{\gamma\beta_2\delta}{(\gamma + \kappa + \mu)(\beta_1(\gamma + \kappa + \mu) + \beta_2\delta)} \\
r_{\nu}^{R_0} &= \frac{\partial R_0}{\partial \nu} X \frac{\nu}{R_0} = -\frac{\nu}{\alpha + \nu + \mu} \\
r_{\theta}^{R_0} &= \frac{\partial R_0}{\partial \theta} X \frac{\theta}{R_0} = -\frac{\theta}{\delta + \theta + \mu} \\
r_{\kappa}^{R_0} &= \frac{\partial R_0}{\partial \kappa} X \frac{\kappa}{R_0} = -\frac{\kappa\beta_2\delta}{(\gamma + \kappa + \mu)(\beta_1(\gamma + \kappa + \mu) + \beta_2\delta)} \\
r_{\Lambda}^{R_0} &= \frac{\partial R_0}{\partial \Lambda} X \frac{\Lambda}{R_0} = 1 \\
r_{\mu}^{R_0} &= \frac{\partial R_0}{\partial \mu} X \frac{\mu}{R_0} = \frac{1}{(\alpha + \nu + \mu)(\delta + \theta + \mu)(\gamma + \kappa + \mu)(\beta_1(\gamma + \kappa + \mu) + \beta_2\delta)} [A - D]
\end{aligned}$$

$$A = \beta_1(\mu(\alpha + \nu + \mu)(\delta + \theta + \mu)(\gamma + \kappa + \mu))$$

$$D = (\beta_1(\gamma + \kappa + \mu) + \beta_2\delta)((\alpha + \nu + \mu)(\delta + \theta + \mu)(\gamma + \kappa + \mu) + \mu(\delta + \theta + \mu)(\gamma + \kappa + \mu) + (\mu(\alpha + \nu + \mu)(\gamma + \kappa + \mu) + (\mu(\delta + \theta + \mu)(\alpha + \nu + \mu))))$$

Since  $\beta_1, \beta_2, \alpha, \delta$  and  $\Lambda$  are directive proportional to  $R_0$ . We can conclude that increasing the values of  $\beta_1, \beta_2, \alpha, \delta$  and  $\Lambda$  has positive impact on the spread of tobacco smoking while,  $\nu, \kappa, \gamma, \theta$  &  $\mu$  are inversely proportional to  $R_0$  and increasing these values has negative impact on the spread of tobacco smoking. In general, targeting the most positively and negatively sensitive parameters on  $R_0$  will be most effective in reducing the spread of smoking habit.

According to  $R_0$ , the sensitivity index for the parameters is provided in the following table.

Parameters of the model	Sensitivity indexes
$\mu$	-1.792
$\Lambda$	1
$\beta_2$	0.9176
$\delta$	0.6023
$\theta$	-0.5405
$\gamma$	-0.3797
$\alpha$	0.2566
$\nu$	-0.115
$\beta_1$	0.0824
$\kappa$	-0.0316

Table 4.3: Sensitivity indices of  $R_0$ .

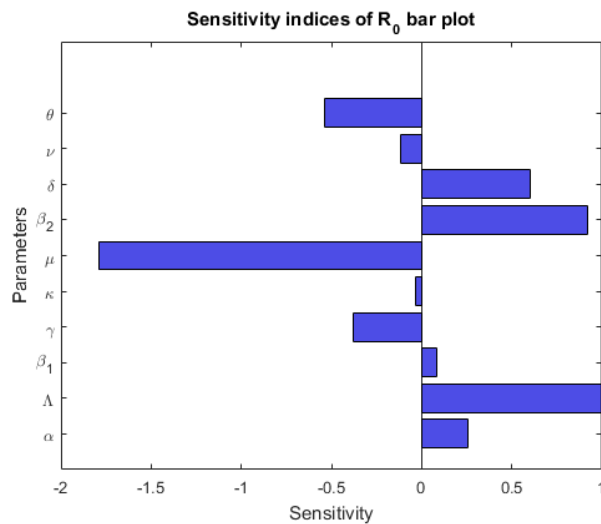


Figure 4.4: Sensitivity Analysis of the Parameters.

## 4.6 Numerical Simulations and Discussion

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 (4.2.1 - 4.2.5) 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 [23]. 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 focus is to assess the effect of different parameters on the spread of the habit of smoking tobacco and these simulations can also show the behavior of the populations in time and the stabilities of both smoking free and smoking

present equilibrium points.

To show that the analytical results are in agreement with numerical solutions, we used parametric values given in table (4.4) below and the following hypothetical initial population of 2100 such that;  $S(0) = 2000$ ,  $E(0) = 500$ ,  $I_o = 300$ ,  $I_h = 100$ , and  $R(0) = 50$  for the period of days. And the results are given in the next pages of Figures (3-15).

Parameter	value	source	unit
$\Lambda$	100	Assumption	per week
$\beta_1$	$1.3 \times 10^{-5}$	Assumption	per week
$\beta_2$	$1.2 \times 10^{-4}$	Assumption	per week
$\mu$	0.016	[14]	per week
$\gamma$	0.012	Assumption	per week
$\delta$	0.035	Assumption	per week
$\kappa$	0.001	[14]	per week
$\nu$	0.013	[14]	per week
$\alpha$	0.084	Assumption	per week
$\theta$	0.06	[26]	per week

Table 4.4: Different parameter values used in our model.

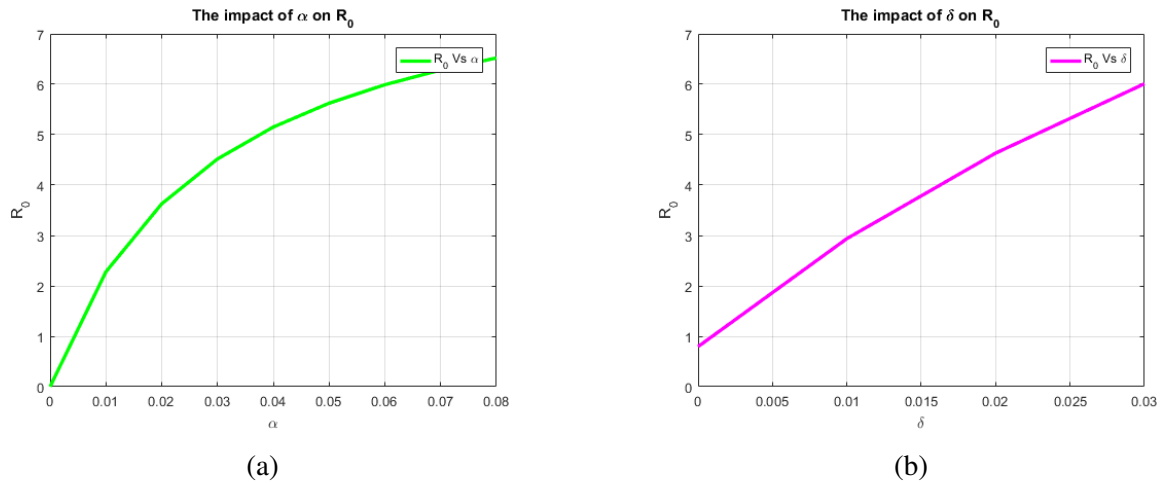


Figure 4.5: Parameters having positive impact on the expansion of smoking habit.

Figure(4.5) indicates that the value of smoking generation number  $R_0$  increase as both the value of rate at which second hand smoke individuals become occasional smokers  $\alpha$  and rate at which occasional smokers becomes habitual smokers  $\delta$  are increase. However, there is a difference in  $\alpha$  and  $\delta$  which means  $\delta$  is more sensitive parameter than  $\alpha$  to increases the and decrease  $R_0$ .

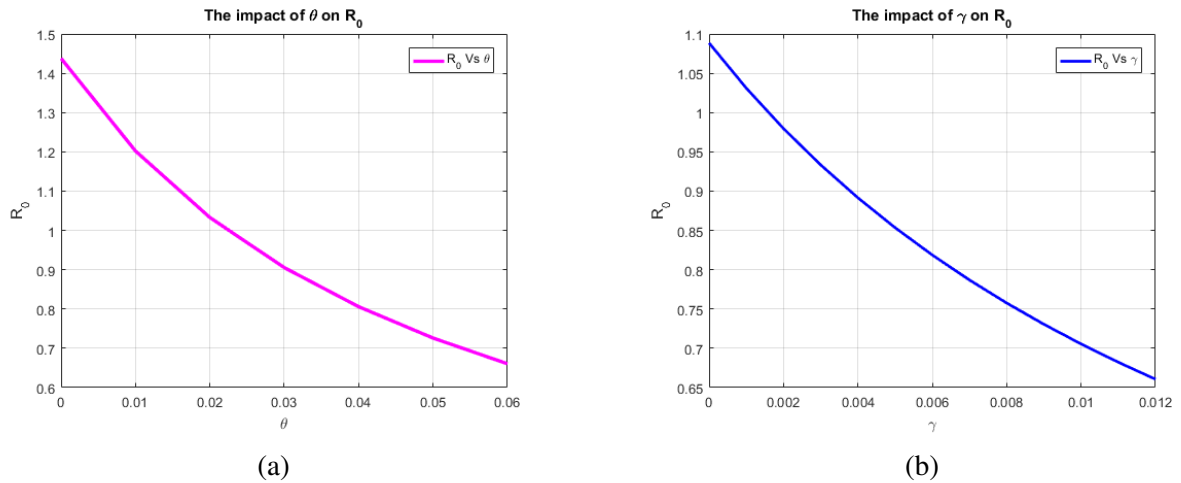


Figure 4.6: Parameters having negative impact on the expansion of smoking habit

Figure(4.6) indicates that the value of smoking generation number  $R_0$  decreases as both the value of  $\theta$  and  $\gamma$  increased and decreased those two parameters and the smoking generation number increases. From figure(4.7) we observed that the value of smoking generation number

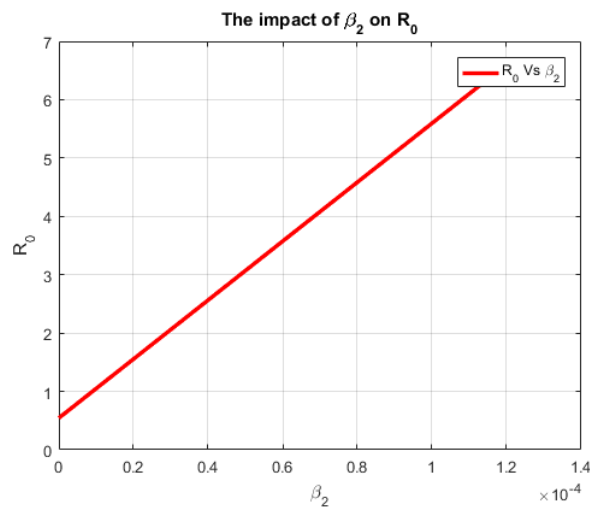
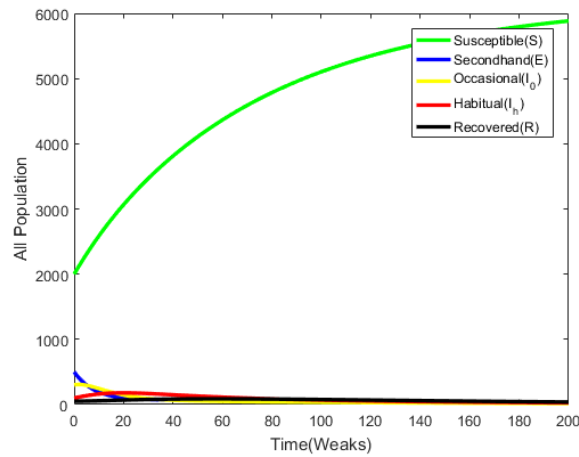
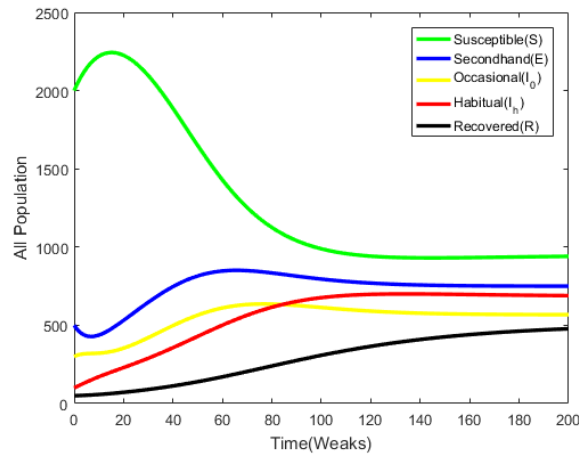


Figure 4.7: Parameters having positive impact on the expansion of smoking habit.

$R_0$  increase as the value of contact rate  $\beta_2$  increases. This relation tells us the contact rate  $\beta_2$  is most sensitive parameter of the model.



(a)



(b)

Figure 4.8: The graph dynamics of the state variables of modified tobacco smoking model with  $R_0 = 0.6606$  and  $R_0 = 6.6060$ .

In figure (4.8a)  $R_0 = 0.6606$ , we observe that for the basic reproductive number  $R_0 < 1$ , all solutions curves goes to the smoking free equilibrium point. As a result the habit of smoking is eradicate in the long run from the population. These indicate that the smoking free equilibrium point is locally and globally asymptotically stable for the values of  $R_0 < 1$ .

In figure (4.8b)  $R_0 = 6.6606$ , we observe that for the basic reproductive number  $R_0 > 1$ , all solutions curves goes away from smoking free equilibrium point. These indicate that the disease-free equilibrium point is unstable for the values of  $R_0 > 1$ , and the solutions will go to the endemic equilibrium point. These indicate that the endemic equilibrium point is locally asymptotically stable for the values of  $R_0 > 1$ .

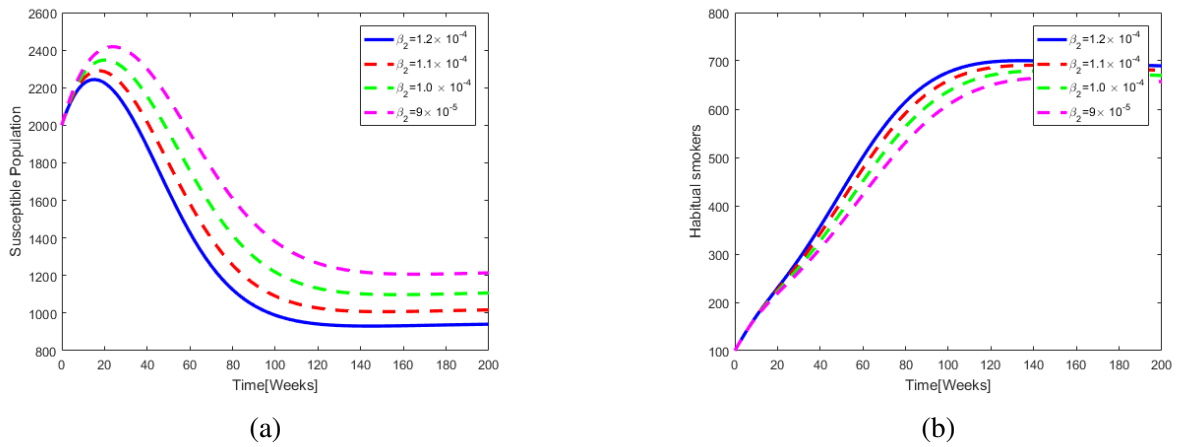


Figure 4.9: Effect of contact rate ( $\beta_2$ ) on susceptible individuals  $S$  and habitual smokers  $I_h$ .

Figure(4.9) indicates when habitual smokers interact with the susceptible population then the habitual smoker population rapidly grows by the contact rate  $\beta_2$ , this indicates that the habit of smoking increases through out the population. In this case the value of  $\beta_2$  decrease susceptible individual increase and also habitual smokers decrease.

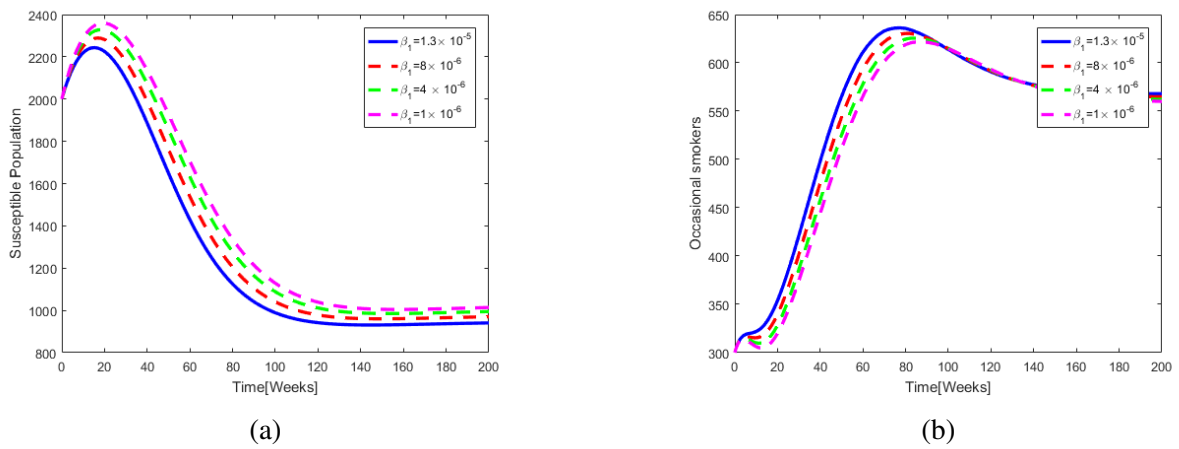
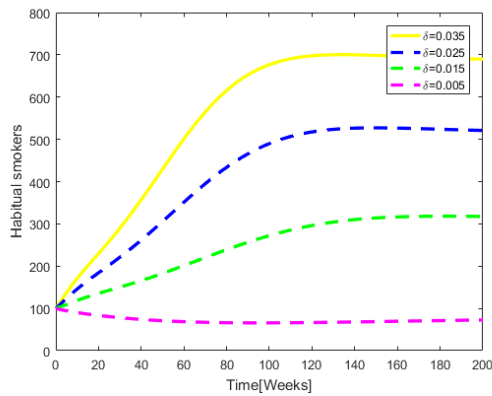
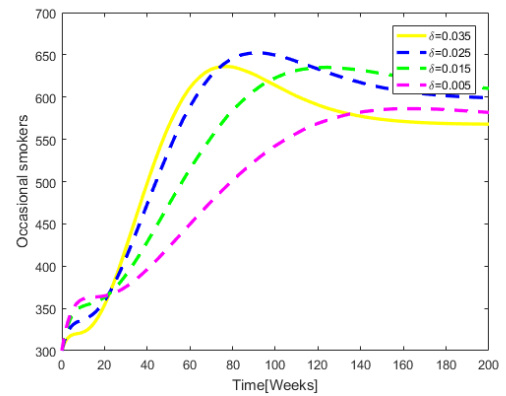


Figure 4.10: Effect of contact rate ( $\beta_1$ ) on susceptible individuals  $S$  and occasional smokers  $I_o$ .

Figure(4.10) indicates when smokers interact with the susceptible population the occasional smokers rapidly grows by the contact rate  $\beta_1$ , this indicates that the habit of smoking increases through out the population. we observe the figure (4.10) the contact rate  $\beta_1$  increases as the susceptible individuals decrease and the group of occasional smokers increase.



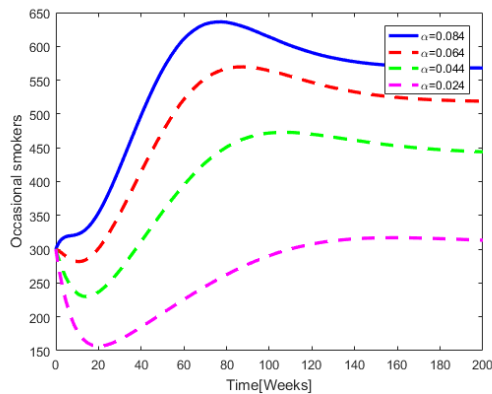
(a)



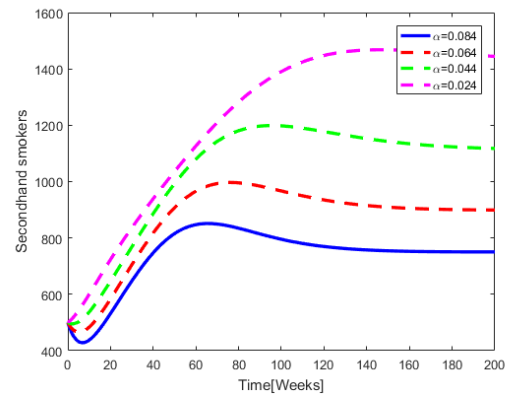
(b)

Figure 4.11: Effect of parameter ( $\delta$ ) on occasional smokers  $I_o$  and habitual smokers  $I_h$ .

In Figure (4.11) the parameter  $\delta$  increases the number of occasional smoker decrease and the habitual smoker rapidly increase. This is due to the case that more individuals join the habitual smoker from occasional smokers.



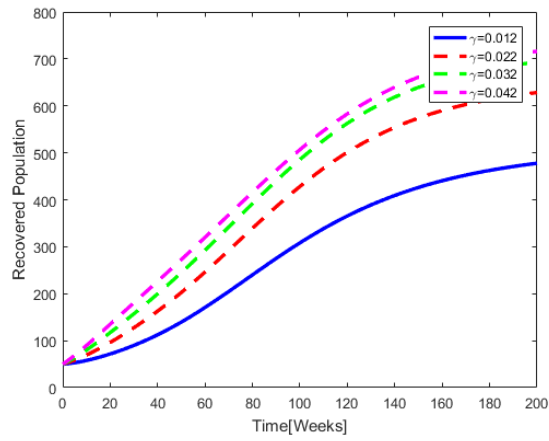
(a)



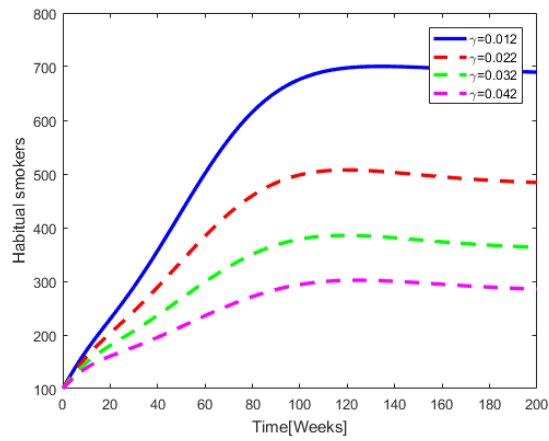
(b)

Figure 4.12: Effect of parameter ( $\alpha$ ) on secondhand smokers  $E$  and occasional smokers  $I_o$ .

In Figure (4.12) parameter  $\alpha$  increase the number of secondhand smoker  $E$  decrease and the number of occasional smokers  $I_o$  is increase. This is due to the case that more individuals join the secondhand smoker group from occasional smoker classes.



(a)



(b)

Figure 4.13: Effect of treatment rate ( $\gamma$ ) on habitual smokers  $I_h$  and recovered group  $R$ .

In Figure (4.13) the treatment rate ( $\gamma$ ) increase the number of active(habitual) smoker  $I_h$  decrease and Recovered population  $R$  is increase. This is due to the case that more individuals join the Recovered group from active(habitual) smoker classes.

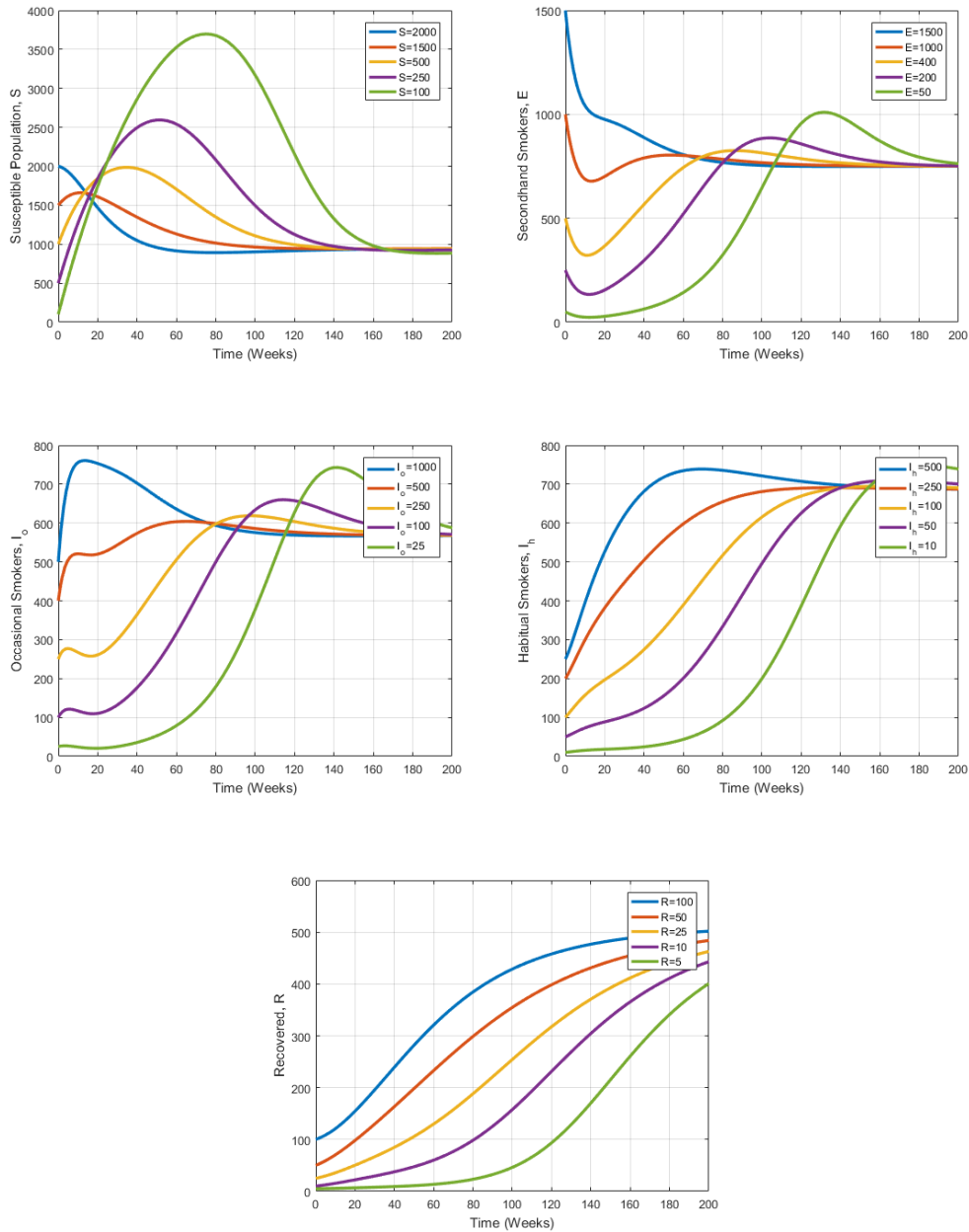


Figure 4.14: Trajectories of state variables for  $R_0 = 6.60603$  with different initial value populations.

Figure 4.14 show the effect of initial population on the dynamics of susceptible, Second hand smokers, Occasional smokers, Habitual smokers and Recovered classes. From all the simulations we see that the number of initial population greatly affect the dynamics and later on go back to the endemic equilibrium points. Finally the populations in each compartment tends to be a positive constant, which means the smoking present equilibrium point is asymptotically stable if  $R_0 > 1$ .

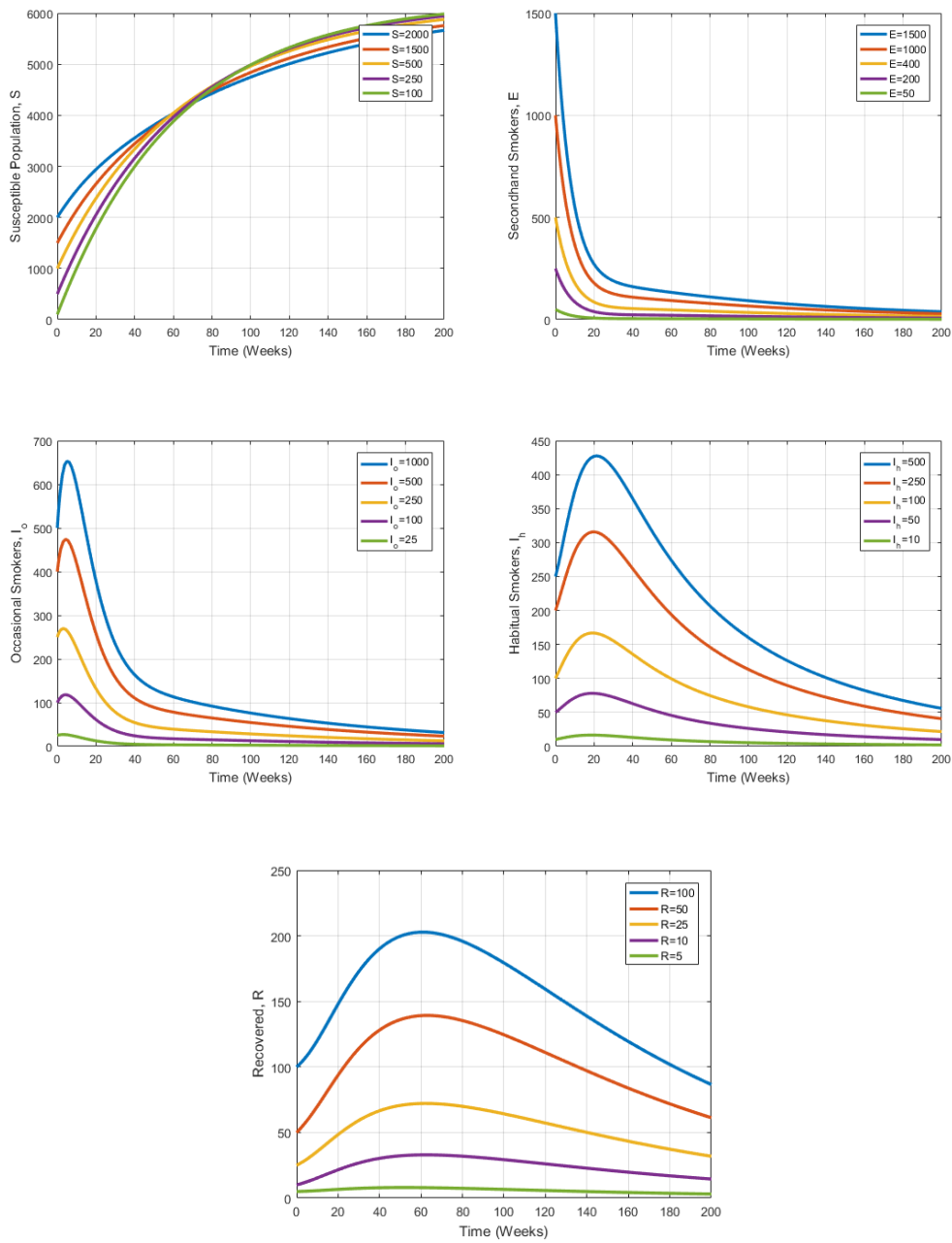


Figure 4.15: Trajectories of state variables for  $R_0 = 0.6606$  with different initial value populations.

For the given initial populations, all trajectories of the state variables converge to their respective components of the smoking-free equilibrium point. This indicates that the smoking-free equilibrium point is both locally and globally asymptotically stable for the specified parameter values.

# Chapter 5

## Conclusion and Recommendation

Here under, we gave the general conclusion of the thesis and some recommendations to be given to the concerned bodies.

### 5.1 Conclusion

In this thesis, a mathematical model is proposed to study the analysis of tobacco smoking by considering occasional and habitual smokers. We have established and proved positivity and boundedness of the solution. Two equilibrium points exist namely Smoking Free Equilibrium point ( $E_0$ ) and Endemic Equilibrium point ( $E^*$ ). We computed the steady states and new smoking generation number  $R_0$ .

Based on  $R_0$  it is revealed that whenever  $R_0 < 1$ , the system has only smoking free equilibrium point ( $E_0$ ), which is locally & globally asymptotically stable. We have also observed that the individual smoking habit losing on occurrence on average less than one new smoking habit. This means smoking habit does not transmit the other population. When  $R_0 > 1$ , smoking present equilibrium point of the model is locally stable, which means the habit of smoking can spread out and smoking free equilibrium point ( $E_0$ ) become unstable. We observed that smoking habit individual transmits on average more than one new smokers and the smoker habit can transmit to the other population.

Using center manifold theory, the bifurcation analysis of the modified model was established, demonstrating that the model exhibits a forward bifurcation at  $R_0 = 1$ . From sensitivity analysis of  $R_0$ , we observed that contact rates  $\beta_1$  &  $\beta_2$ ,  $\alpha$ ,  $\delta$  and recruitment rate  $\Lambda$  have positive impact on the spread of tobacco smoking habit. And the remaining parameters have negative impact on the spread of tobacco smoking habit. Numerical results support the fact that decrease in contact rate decrease the value of  $R_0$ .

From numerical simulation, We have observed that increasing the treatment rate ( $\gamma$ ) leads to decrease in the number of habitual smokers ( $I_h$ ). When susceptible individuals come into contact

with occasional smokers ( $I_o$ ) and habitual smokers ( $I_h$ ) at a rate of  $\beta$ , the numbers of occasional and habitual smokers increase, while the number of susceptible individuals decreases.

## 5.2 Recommendation

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

1. The stakeholders should work on decreasing the positive indices  $\alpha$ ,  $\delta$ ,  $\beta_2$ ,  $\beta_1$  and increasing negative indices parameters  $\gamma$ .
2. Efforts should be made to minimize the interaction of susceptible and smokers; this is because the spread of smoking habit largely depends on the interaction (contact) rate  $\beta$ .
3. Based on the results of our study it is cordially recommended that there should be a treatment for the smoker one of the mechanism to control the transmission of smoking habit and individuals have to reduce the number of smoker friends.
4. General public should be regularly informed about how to protect individuals from becoming addicted, as well as how to treat those who are already addicted, to prevent the spread of smoking habits.

## 5.3 Limitation of the Study

Since this thesis will be conducted with out a real data. We rather used real data from the literature that are very closed to our context for simulation purposes. Sex and age structures are not considered in the study.

## 5.4 Future work

The research can be improved and built upon in many ways, such as:

- Including the incorporation of additional parameters and assumptions regarding the population with additional habits.

- Considering age and employment factors among adults to capture a more comprehensive view of the population dynamics.
- Exploring the optimal control to develop more effective strategies based on the model's insights.

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# Appendix

```
1      function f = Genet(t,y,Lambda,alpha,beta1,beta2,gamma,delta,mu
      ,kappa,nu,theta)
2      S = y(1);
3      E = y(2);
4      I0 = y(3);
5      Iu = y(4);
6      R = y(5);
7      dS = Lambda-(beta1*I0+beta2*Iu+mu)*S;
8      dE = (beta1*I0+ beta2*Iu)*S-(alpha+nu+mu)*E;
9      dI0 = alpha*E-(delta+theta+mu)*I0;
10     dIu = delta*I0-(gamma+kappa+mu)*Iu;
11     dR = gamma*Iu-mu*R;
12     f = [dS;dE;dI0;dIu;dR];
```

## MATLAB Code for figure 4.8

```
1      C = ['g ','b ','y ','r ','k '];
2      Lambda = 100;
3      alpha =0.084;
4      beta1 = 1.3*10^(-5);
5      beta2 = 1.2*10^(-4);
6      gamma = 0.012;
7      delta = 0.035;
8      mu = 0.016;
9      kappa = 0.001;
10     nu = 0.013;
11     theta = 0.06;
12     R0= (alpha*Lambda*(beta1*(gamma+kappa+mu)+beta2*delta))/(mu*(
      alpha+nu+mu)*(delta+theta+mu)*(gamma+kappa+mu))
13     tspan = [0 200];
14     y0 = [2000 500 300 100 50];
15     [t, y] = ode45(@Genet,tspan,y0,[],Lambda,alpha,beta1,beta2,
      gamma,delta,mu,kappa,nu,theta);
```

```

16     for i = 1:5
17         plot(t,y(:,i),C(i,:), 'LineWidth',2.5)
18         legend('Susceptible(S)', 'Secondhand(E)', 'Occasional(I_0)', '
           Habitual(I_h)', 'Recovered(R)')
19         xlabel('Time(Weeks)')
20         ylabel('All Population')
21         hold on
22         end

```

### MATLAB Code for figure 4.14 and 4.15

```

1     % Define global variables
2     global Lambda alpha beta1 beta2 gamma delta mu kappa nu theta
3
4     % Initialize variables
5     Lambda = 100;
6     alpha = 0.084;
7     beta1 = 1.3 * 10^(-6);
8     beta2 = 1.2 * 10^(-5);
9     gamma = 0.012;
10    delta = 0.035;
11    mu = 0.016;
12    kappa = 0.001;
13    nu = 0.013;
14    theta = 0.06;
15
16    % Calculate the parameter R0
17    R0 = (alpha * Lambda * (beta1 * (gamma + kappa + mu) + beta2 *
           delta)) / ...
18    (mu * (alpha + nu + mu) * (delta + theta + mu) * (gamma +
           kappa + mu));
19    fprintf('Value of parameter R0 is %.5f\n', R0);
20
21    % Time span for the ODE solver
22    tsp = [0, 200];

```

```

23
24 % Initial conditions for different scenarios
25 y_0 = [
26 2000 1500 500 250 100;
27 1500 1000 400 200 50;
28 1000 500 250 100 25;
29 500 250 100 50 10;
30 100 50 25 10 5
31 ];
32
33 % Loop through each set of initial conditions
34 for i = 1:5
35 [T, Y] = ode45(@(t, y) Genet(t, y, Lambda, alpha, beta1, beta2
36 , gamma, delta, mu, kappa, nu, theta), tsp, y_0(i, :));
37 % plot(T, Y(:, 1), 'linewidth', 2.5)
38 % plot(T, Y(:, 2), 'linewidth', 2.5)
39 % plot(T, Y(:, 3), 'linewidth', 2.5)
40 % plot(T, Y(:, 4), 'linewidth', 2.5)
41 plot(T, Y(:, 5), 'linewidth', 2.5)
42 hold on
43 end
44
45 % Customize the plot
46 xlabel('Time (Weeks)')
47 % ylabel('Susceptible Population, S')
48 % ylabel('Secondhand Smokers, E')
49 % ylabel('Occasional Smokers, I_{o}')
50 % ylabel('Habitual Smokers, I_{h}')
51 ylabel('Recovered, R')
52 % legend('S=2000', 'S=1500', 'S=500', 'S=250', 'S=100')
53 % legend('E=1500', 'E=1000', 'E=400', 'E=200', 'E=50')
54 % legend('I_o=1000', 'I_o=500', 'I_o=250', 'I_o=100', 'I_o
=25')
55 % legend('I_h=500', 'I_h=250', 'I_h=100', 'I_h=50', 'I_h

```

```

    =10')
55 legend('R=100', 'R=50', 'R=25', 'R=10', 'R=5')
56 grid on

```

### MATLAB Code for figure 4.9-4.13

```

1      C = ['g ' ; 'b ' ; 'y ' ; 'r ' ; 'm--'];
2      Lambda = 100;
3      alpha = 0.084;
4      beta1 = 1.3*10^(-5);
5      beta2 = 1.2*10^(-4);
6      %      gamma = 0.012;
7      %      gamma = 0.022;
8      %      gamma = 0.032;
9      gamma = 0.042;
10     delta = 0.035;
11     mu = 0.016;
12     kappa = 0.001;
13     nu = 0.013;
14     theta = 0.06;
15     R0= (alpha*Lambda*(beta1*(gamma+kappa+mu)+beta2*delta))/(mu*(
        alpha+nu+mu)*(delta+theta+mu)*(gamma+kappa+mu))
16     tspan = [0 200];
17     y0 = [2000 500 300 100 50];
18     [t, y] = ode45(@Genet,tspan,y0,[],Lambda,alpha,beta1,beta2,
        gamma,delta,mu,kappa,nu,theta);
19     for i = 5
20     plot(t,y(:,i),C(i,:), 'LineWidth', 2.5)
21     legend('\gamma=0.012', '\gamma=0.022', '\gamma=0.032', '\gamma
        =0.042')
22     xlabel('Time[Weeks]')
23     ylabel('Recovered Population')
24     hold on
25     end

```

#### MATLAB Code for figure 4.4

```
1      % Define parameter values
2      a_val = 0.084;
3      b_val = 100;
4      c_val = 1.3*10^-5;
5      d_val = 0.012;
6      e_val = 0.001;
7      f_val = 0.016;
8      g_val = 1.2*10^-4;
9      h_val = 0.035;
10     k_val = 0.013;
11     m_val = 0.06;
12
13     % Define symbolic variables
14     syms a b c d e f g h k m
15
16     % Define the expression for R0
17     R0 = (a * b * (c * (d + e + f) + g * h)) / (f * (a + k + f) *
18           (h + m + f) * (d + e + f));
19
20     % Compute partial derivatives of R0 with respect to each
21     parameter
22     sa = diff(R0, a) * a / R0;
23     sb = diff(R0, b) * b / R0;
24     sc = diff(R0, c) * c / R0;
25     sd = diff(R0, d) * d / R0;
26     se = diff(R0, e) * e / R0;
27     sf = diff(R0, f) * f / R0;
28     sg = diff(R0, g) * g / R0;
29     sh = diff(R0, h) * h / R0;
30     sk = diff(R0, k) * k / R0;
31     sm = diff(R0, m) * m / R0;
32
33     % Create a vector of the parameters
```

```

32 param_values = [a_val, b_val, c_val, d_val, e_val, f_val,
33                g_val, h_val, k_val, m_val];
34
35 % Evaluate the sensitivity expressions at the given parameter
36 % values
37 sa_val = double(subs(sa, {a, b, c, d, e, f, g, h, k, m},
38                       param_values));
39 sb_val = double(subs(sb, {a, b, c, d, e, f, g, h, k, m},
40                       param_values));
41 sc_val = double(subs(sc, {a, b, c, d, e, f, g, h, k, m},
42                       param_values));
43 sd_val = double(subs(sd, {a, b, c, d, e, f, g, h, k, m},
44                       param_values));
45 se_val = double(subs(se, {a, b, c, d, e, f, g, h, k, m},
46                       param_values));
47 sf_val = double(subs(sf, {a, b, c, d, e, f, g, h, k, m},
48                       param_values));
49 sg_val = double(subs(sg, {a, b, c, d, e, f, g, h, k, m},
50                       param_values));
51 sh_val = double(subs(sh, {a, b, c, d, e, f, g, h, k, m},
52                       param_values));
53 sk_val = double(subs(sk, {a, b, c, d, e, f, g, h, k, m},
54                       param_values));
55 sm_val = double(subs(sm, {a, b, c, d, e, f, g, h, k, m},
56                       param_values));
57
58 % Display the sensitivities
59 sensitivities = table(sa_val, sb_val, sc_val, sd_val, se_val,
60                      sf_val, sg_val, sh_val, sk_val, sm_val, ...
61                      'VariableNames', {'sa', 'sb', 'sc', 'sd', 'se', 'sf', 'sg', 'sh', 'sk', 'sm'});
62
63 disp('Sensitivities:');
64 disp(sensitivities);

```

```

52
53 % Plot the sensitivities as a horizontal bar chart with the
      same color
54 sensitivity_values = [sa_val, sb_val, sc_val, sd_val, se_val,
      sf_val, sg_val, sh_val, sk_val, sm_val];
55 param_names = {'\alpha', '\Lambda', '\beta_1', '\gamma', '\
      kappa', '\mu', '\beta_2', '\delta', '\nu', '\theta'};
56
57 figure;
58 b = barh(sensitivity_values, 'FaceColor', [0.3, 0.3, 0.9]); %
      Set to desired color
59 %barh(sensitivity_values);
60 set(gca, 'YTickLabel', param_names, 'YTick', 1:numel(
      param_names));
61 xlabel('Sensitivity');
62 ylabel('Parameters');
63 title('Sensitivity indices of R_0 bar plot');
64 %grid on;

```

### MATLAB Code for figure 4.3

```

1 %MATLAB codes for bifurcation analysis:
2 L=10;b=1.3*10^-5;d=1.2*10^-4;a=0.084;r=0.012;s=0.035;m=0.016;k
      =0.001;v=0.013;o=0.06;
3 %alpha=0.20; A=0.4;b1=0.45;beta2=0.057;beta3=0.05;mu1=0.05;mu2
      =0.0303;beta1=0.0390;
4 %mu3=0.0303;delta=0.0819;phi=0.0707;Q=0.02;K=10000;
5 %D=0.05;r=0.005;Lambda=0.45;C=0.6;
6 R=1:0.1:2;
7 R1=0:0.1:1;
8 %fprintf('Value of parameter R0 is %.5f', (sqrt((alpha*beta1*K)
      /((A+K)*(alpha+mu1)*(delta+phi+mu1))*((b1*beta2*Q)...
9 % /((C*mu2*mu2)+(b1*delta*beta3*Q)/(mu2*mu2*mu3*D))))))
10 fprintf('Value of parameter R0 is %.5f', (a*L*(b*(r+k+m)+d*s))
      / (m*(m+a+v)*(m+s+o)*(r+k+m)));

```

```

11     Lambda = (m*(a+v+m)*(s+o+m)*(r+k+m)*(R-1))/(a*(b*(r+k+m)+ d*s)
12         );
13     Lambd1 =zeros(length(R),1);
14     Lambd2 =zeros(length(R),1);
15     h=figure;
16     plot(R,Lambda,'g','Linewidth',3)
17     hold on
18     plot(R,Lambd1,'r--','Linewidth',3)
19     plot(R1,Lambd2,'b','Linewidth',3)
20     xlabel('R_0');
21     ylabel('Force of infection,\lambda*');
22     hold on
23     set(h,'Units','Inches');
24     pos=get(h,'Position');
25     set(h,'PaperPositionMode','Auto','PaperUnits','Inches','
        PaperSize',[pos(3),pos(4)])
        print(h,'Forwardbifurcation','-dpdf','-r0')

```