MODELLING DIABETES AND ITS COMPLICATION IN A RESOURCE CONSTRAINED SETTING

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DECLARATION

This thesis is my original work and has not been presented for a degree or other award in any other university.

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DEDICATION

This thesis is dedicated to my late brother, Charles Moindi Andima for his trust in me and unending encouragement that I attain a Ph.D. To my brothers Dr. Andima and Gilbert for their support, prayers and encouragement throughout my study.

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NOMENCLATURE

Abbreviations			
GoK	Government of Kenya	LMIC	Low and Middle Income
GDP	Gross Domestic Product	MDG4	Millennium Development Goals Number 4
MoH	Ministry of Health	NCDs	Non Cmmuncable diseases
WHO	World Health Organisation		

ABSTRACT

Diabetes mellitus is a chronic non-communicable disease resulting from the body's inability to metabolise excess glucose due to impaired insulin secretion or function. It has become a major global burden, with the World Health Organization (WHO) reporting that non-communicable diseases account for 71% of all annual deaths, 85% of which occur in low- and middle-income countries. In this study, the dynamics of diabetes are examined under the influence of strained healthcare resources and patient response behaviour. Two compartmental mathematical models are formulated to describe the transitions between susceptible, diabetic, and hospitalized populations. The first model assumes a constant hospitalization rate, while the second introduces a variable hospitalisation rate that depends on the system's carrying capacity and per capita hospitalization rate. The basic reproduction number and equilibrium states are derived and analysed to assess disease persistence conditions. Numerical simulations using the explicit Runge-Kutta (4,5) method in MATLAB illustrate the system's behaviour under varying parameters. The results show that enhancing lifestyle quality among susceptible increases their stability while reducing diabetes prevalence; a higher treatment rate among diabetics raises the hospitalized proportion, whereas increasing the carrying capacity diminishes hospitalization levels.

CHAPTER ONE

INTRODUCTION

1.1 Background Information

Chronic illnesses, sometimes referred to as non-communicable diseases (NCDs), are conditions that develop over an extended period of time as a result of a combination of genetic, physiological, environmental, and behavioral variables. The four primary categories of NCDs are diabetes, cancer, chronic respiratory illnesses, and cardiovascular disorders. Non-communicable illnesses, which account for 71 percent of all yearly fatalities, are by far the biggest cause of mortality worldwide, according to the WHO. More than 41 million people die from NCDs each year, and low- and middle-income countries account for 85% of these fatalities (Mohamed *et al.*, 2018). When insulin-glucose-glucagon regulation system is undermined, diabetes develops. As a result, the pancreas either generates insufficient amounts of insulin or the body is unable to utilize the insulin that is produced. A hormone called insulin controls blood sugar.

Uncontrolled diabetes frequently results in hyperglycemia, or elevated blood sugar, which over time causes substantial harm to many different bodily systems, including the neurons and blood vessels. Diabetes is a significant contributor to renal disease, heart attacks, strokes, blindness, and lower limb amputation. Increased appetite and thirst, weight loss, excessive urination, hazy vision, intense exhaustion, and sores that don't heal are some of the common symptoms of diabetes. Men with diabetes may also have diminished sex desire, erectile dysfunction (ED), and weak muscles in addition to these fundamental symptoms. On the other side, women with diabetes might also have symptoms including yeast infections, dry, itchy skin, and urinary tract infections (Papatheodorou *et al.*, 2018; Afaya *et al.*, 2020).

Diabetes comes in two varieties: Type 1 diabetes, also referred to as

insulin-dependent, juvenile, or childhood-onset diabetes, is characterized by inadequate insulin production. It requires daily insulin administration and presents with symptoms such as excessive urination (polyuria), excessive thirst, constant hunger, loss of weight, changes in vision, and fatigue. Additionally, we have Type 2 diabetes, commonly known as non-insulin-dependent or adult-onset diabetes, which is brought on by the body's inefficient utilization of insulin. The majority of persons with diabetes worldwide have type 2 diabetes, which is mostly brought on by being overweight and being inactive. Although frequently less severe, its symptoms might be comparable to type 1 diabetes (Kreider, 2019). As a consequence, the condition may not be discovered until after it has developed problems. This kind of diabetes was previously exclusively observed in adults, but it is now increasingly common in youngsters as well (Kharroubi, 2015).

Diabetes now affects 422 million people worldwide, up from 180 million in 1980. Age-specific prevalence is on the rise (28%) and population growth and aging (40%) are the main causes of this increase (32 percent). Trends in age-standardized prevalence are displayed in In adults aged 18 and older, age-standardized prevalence increased from 4.7 percent in 1980 to 8.5 percent in 2014. In 1980, high-income nations had the highest incidence (5.2%), while low-income countries had the lowest prevalence (3.3 percent). By 2014, low-income nations had a prevalence of 7.4%, which was greater than high-income countries' prevalence of 7.0%. Diabetes prevalence has increased more quickly in low- and middle-income nations, and it is presently greatest in higher middle-income nations (9.3 percent) (Khan *et al.*, 2019).

Like other developing nations, Kenya is dealing with the growing diabetes epidemic. Diabetes prevalence in the nation is thought to be around 3.3%. Unless this trend is reversed, this number is predicted to increase to 4.5 percent by 2025. In 2015, Kenya reported over 8,700 diabetes-related fatalities, virtually all of whom were under 60 (Guariguata *et al.*, 2014). The incidence of diabetes is greater in urban areas and among males, but the majority of national figures are based on adult populations, and

it is mostly unknown how common Type 1 diabetes is in kids and teenagers. Although the prevalence of diabetes is under-reported in this environment, it is believed that up to 60% of diabetics in Kenya go untreated. Although a household study in Nairobi's informal settlement of Kibera found that 22.6 percent of people with a medical diagnosis of diabetes took insulin, it is unknown how many adults in Kenya with Type 2 diabetes are administered insulin (Ngwiri *et al.*, 2015).

Since the 2010 Kenyan Constitution's decentralization of authority, each of the 47 Counties now oversees its own health systems, including those that provide care for NCDs. Six stages of progressively more extensive county health services are set up. Community-based care is offered through Level 1 units in each community. Facilities that fall under Level 2 offer basic prenatal care, immunizations, and other fundamental healthcare services. A level 3 facility is a medical centre that is more expansive than a level 1 or level 2 facility and offers a wider range of services, including the possibility of dispensing certain drugs and providing some basic inpatient treatment. Level 4 services correspond to subcounty hospitals, whereas Level 5 services stand in for the county referral hospital. The national referral hospitals in Nairobi are classified as Level 6 services.

Despite the Kenyan Constitution's declaration that everyone has the right to health care, the country has made very little progress towards universal health coverage (UHC) (Ngugi *et al.*, 2017). Additionally, Otieno *et al.* (2021) noted that 68 percent of Kenyans' fundamental health requirements are not being satisfied. Diabetes and other NCDs are becoming more widely recognized as health issues. Diabetes is one of the four most prevalent chronic diseases in Kenya, according to the 2015-2020 National Strategy for the Prevention and Control of NCDs, which also lays out a plan for health system improvement, risk factor reduction, and promotion of good health. A particular chronic illness package of care was also recently introduced by the National Hospital Insurance Fund (NHIF). Despite these national attempts, sub-county and county-level diabetes care systems continue to be unreliable, and

there are very little diabetes data available.

In addition to creating added burden to an already overwhelmed healthcare system, the emergence of NCDs like diabetes makes it difficult for the system to meet the different demands of both acute and chronic diseases. When diabetes is discovered early and treated well, life expectancy and quality of life for those who have it can both improve (World Health Organization, 2016). The loss of eyesight, cardiovascular disease, end-stage renal disease, and lower extremity amputation are just a few of the serious and irreversible problems that diabetes may cause if it is not properly diagnosed or controlled. In order to address this, a fundamental package for diabetes care should include a balanced diet, regular exercise, blood pressure management, foot and eye care, and a steady supply of insulin or anti-hyperglycemic medicine, as necessary. Many patients in LMICs, such as Kenya, struggle to get this fundamental diabetic treatment. They encounter a number of obstacles to care, such as the distance to the medical facility, lack of knowledge, the cost of drugs, the unavailability of diagnostic and monitoring tests, and the inadequate capacity of the local health system.

1.2 Statement of the Problem

Obesity and other health issues are enhanced by changes in typical lifestyle and eating habits which emanates from the predominance of sedentary occupations, and rapidly increasing number of fast food, particularly in industrialized nations. Additionally, as a result of this, there are now more individuals with diabetes. Gestational diabetes, in which pregnant women have diabetic symptoms after giving birth, has become more common. There is frequently a probability that the youngster also has diabetes. So, in addition to developing diabetes as a result of lifestyle changes, hereditary diabetes is also increasing the already enormous number of people with diabetes. Diabetes is an epidemic that has to be addressed urgently due to both its medical effects and the financial burden it places on the world economy.

Diabetes must be acknowledged as being devastating for both individual health and national economy, particularly in third-world countries where the majority of the population does not engage in regular physical activity. While some initiatives have offered short-term assistance for diabetes patient's urgent requirements, their long-term needs might be better handled by integrating into the larger healthcare system. As was previously stated, many individuals in need of care cannot afford the price of diabetic care. There has been a lot of study on modelling diabetes, but much of it has been done without taking hospital capacity into account. Therefore, the goal of this study is to formulate and analyse a mathematical model for managing diabetes and associated consequences in a context with limited resources.

1.3 Objectives

1.3.1 General objective

To formulate a deterministic mathematical model of the diabetes behaviour and its complications under resource constraints condition.

1.3.2 Specific objectives

The specific goals of this research are to;

i. Formulate a model for diabetes dynamics and its complications under different hospitalisation cases.

ii. Investigate the impact of increasing recovery rates among the hospitalised individuals impact the trends in diabetes.

iii. Examine the influence of quality of lifestyle on the management of diabetes.

iv. Investigate how the trends of diabetes are impacted by limited resources.

1.4 Justification of the Study

It is impossible to overemphasize the toll that diabetes has on people, society, and economies, particularly in underdeveloped and emerging nations. Diabetes treatment is expensive, putting a strain on families' finances. Life-threatening problems necessitate expensive, sophisticated care, which drives many people to sell their limited possessions to pay for treatment, impoverishing the person, the family, and the society. The main agenda of avoiding or reducing the progression of diabetic and its associated complications in developing nations, improving the quality of life and reducing premature mortality in persons with diabetes, calls for the rapid creation of a framework and implementation instructions.

1.5 Significance of the Study

A given response variable's influence and connections with other variables or treatments are finally determined using mathematical models to produce forecasts, prognoses, or other analyses. The decision and the researcher's ability to adhere to the assumptions regulating the usage of the selected model determine the effectiveness of the models and their resilience in major part. By evaluating population risk, forecasting the outcomes of treatments, and contributing to the evaluation of existing programs, our model will inform the accuracy of the predictions made and the implementation of the study findings to public health practice. This model will offer more insight into the benefits of healthy lifestyle intervention techniques, such as exercise and nutritious eating, on diabetes management. This will be used to account for preventative care, as well as to enable policymakers in the MoH understand which policies can be put into place to help remove the threat, give insight into diabetes's future trends, and define the crucial parameter on the disease's treatment and diagnosis. This adds to MoH policy that emphasizes preventative care rather than curative care, which will help the GoK,

MoH, and other stakeholders plan.

CHAPTER TWO

LITERATURE REVIEW

2.1 Literature Review

Mathematical modelling has become an indispensable tool in understanding the mechanisms, dynamics, and control of chronic diseases such as tuberculosis (Basti *et al.*, 2024), COVID-19 (Sabherwal *et al.*, 2024), HBV (Teklu and Workie, 2024), and diabetes mellitus (Singh *et al.*, 2024). Diabetes is a complex metabolic disorder characterised by high blood glucose levels resulting from defects in insulin secretion, insulin action, or both (Yameny, 2025). Beyond its medical and biological significance, diabetes has emerged as a public health and socio-economic concern, particularly in low- and middle-income countries where access to quality healthcare remains limited. The World Health Organization (WHO) reports that non-communicable diseases (NCDs), including diabetes, account for approximately 71% of global deaths annually, with 85% of these fatalities occurring in resource-limited settings. This increasing burden underscores the necessity of quantitative frameworks capable of explaining disease trends, assessing the effects of treatment and hospitalization, and evaluating the impact of limited healthcare resources. Mathematical models provide such frameworks by translating biological and epidemiological assumptions into differential equations that describe how different population classes evolve over time.

The earliest mathematical studies on diabetes focused primarily on physiological modelling of glucose-insulin dynamics (Ahmed *et al.*, 2025; Rihan and Udhayakumar, 2025). These models captured the intricate feedback between insulin secretion, glucose uptake, and β-cell function. Notable works include the Bergman's minimal model (Boscolo *et al.*, 2025) and its subsequent modifications (Dagher and Haggege, 2025), which form the basis for understanding insulin resistance and

glucose tolerance. Over time, the focus of modelling extended beyond the individual physiological scale to population-level representations of diabetes dynamics. In these epidemiological models, the human population is divided into compartments such as susceptible, diabetic, and treated or hospitalized individuals. Transition rates between these compartments depend on parameters such as the incidence rate of diabetes, the rate of progression to complications, and the rate of recovery or treatment. Such models are analogous to those used in infectious disease epidemiology but adapted to capture the non-communicable nature and chronic progression of diabetes. They are particularly useful in evaluating the long-term effects of interventions such as lifestyle modification, treatment coverage, and healthcare access.

Food consumed by a healthy human is converted to glucose, which percolates into the bloodstream. Increase in the level of glucose in the bloodstream sends a signal to the pancrease. The pancrease produces insulin transport the glucose to the body cells where the glucose is converted to energy. In a situation where insulin is not produced by the pancrease or when insulin is produced but not properly used, the glucose level in the bloodstream continues to rise everytime the individual consumes another food. This condition where the glucose content of the bloodstream is high is called Diabetes (also called High Blood Sugar level) (Boutayeb *et al.*, 2004; Karachaliou *et al.*, 2020). The incident and prevalence of diabetes is increasing at an alarming rate all across the world (World Health Organization, 2016). Despite the concerted efforts of scientists across the world to combat the disease, no cure has been found for diabetes yet. Considering the rising prevalence rate of diabetes globally, it becomes very imperative to unravel the dynamics of diabetes in our society. Simulation of real-life situations using mathematical modelling have proven very useful in many life endeavours. Ajmera *et al.* (2013) compiled contributions of mathematical modelling over the past 50 years. The study pointed out that the major challenge facing the mathematical modelling of the dynamics of diabetes is the significance of "prolonged hyperglycemia acting differently in different individuals".

Scholars have proposed varieties of mathematical modelling devoted to study the glucose-insulin dynamics on diabetes, management and preventions of complications in the dynamics of diabetes, cost and cost-effectiveness of strategies for treating diabetes and the epidemiology of diabetes. Boutayeb and Derouich (2002) modelled the use of physical activity to enhance the sensitivity of insulin and thereby regulate the blood glucose concentration in both non-insulin-dependent diabetes (NIDD) and insulin-dependent-diabetes (IDD) individuals. They illustrated the need and the effect of involvement in physical exercises on the trend of glucose-insulin in diabetes patients and they inferred from the study that physical activity can be used as a prevention for people at risk. Physical exercises improve insulin sensitivity in NIDD patients and also compensate for the insulin inadequacies. The IDD cases are also improved when a good combination is found between the doses of insulin, intake of carbohydrates and involvement in physical activities.

Boutayeb *et al.* (2004) considered the case of complicated and non-complicated diabetes in diabetic patients and proposed a mathematical model to study the dynamics of diabetes. The model is a system of coupled ordinary differential equations and a numerical approximation is used to find the trend of the diabetic population with complication and the diabetic population without complication. A modification to the system of equations that model the dynamics of diabetes was introduced by Li *et al.* (2006). The modification is based on the application of conservation law and the inclusion of explicit time-delay in modelling glucose-insulin regulatory system. The study identified that the periodical fluctuation in ultradian insulin secretion can be simulated as time-delay equations for insulin secretion due to the risen glucose concentration in the bloodstream. The glucose-insulin regulatory system was modelled mathematically using the delay differential equations with two discrete time delays. The findings agreed with the current existing physiological observation and reveal more insightful information.

Grant (2013) identified the rapid increase in the incidence rate of diabetes mellitus in

sub-Saharan Africa and explicated some of the good practices that have been introduced to curb the expansion in the number of diabetes patients. Zhang *et al.* (2014) used the SEIR mathematical model to investigate the effects of the rate of diabetes incidence and the saturated treatment on the trend of diabetic population. It was inferred that increasing the treatment rate can control the expansion in the population of the diabetes. Karachaliou *et al.* (2020) highlighted the challenges facing the prevention of non-communicable diseases in low-income settings. They proposed a model for the prevention of diabetes and reiterated that effective prevention of diabetes and provision of effective care for diabetes patients can help reduce the burden of such diseases in low-income communities.

López-Palau and Olais-Govea (2020) constructed a mathematical model by starting with the dynamics of concentration of glucose in the bloodstream of healthy individuals. The model is proposed to be useful for studying the regulation of glucose concentration in the bloodstream. Regassa and Tola (2021) assessed the effects of the rate of admission and readmission of diabetic patients into hospitals in Ethiopia. The parametric survival analysis was used to predict the hospital admission rate as 9.85 per 1000 persons per year and readmission rate. Banzi *et al.* (2021) modified the existing mathematical model and proposed a mathematical model for investigating the dynamics of glucose-insulin interaction in a diabetic patients and the model parameters were fitted using the non-linear optimisation. Anusha and Athithan (2021) developed a deterministic mathematical model of 4 compartments that consists of the susceptible compartment, Diabetic compartment, Treatment compartment and the Restrain compartment. Ali *et al.* (2022) attempted to predict the concentration of glucose in the bloodstream using two mathematical models where one consider consider the presence of β-cells while the other is without β-cells. Bayesian framework was implemented with the Markov chain Monte Carlo to obtain the "best model structure" for Kigali, Rwanda. The results from the study showed that model without β-cells is more suitable to study the dynamics of type I diabetes.

Meanwhile, Nasir and Daud (2021) harmonised several differential equations to explain the dynamics of diabetes. The study reviewed several mathematical models including the models without complications, models with constant rate of complication, models with rate-dependent complications, models with prediabetic class, models with complication control, models for diabetic with co-infection with other diseases and several other models. The study did a thorough qualitative analysis of the models and established the existence and uniqueness of the solution, the nonnegativity and boundedness of the solution. Areas for further research were suggested in the concluding remark and one of the suggested areas is to consider how available resources may limit the total number of treated diabetics with complications. With this motivation, this thesis is aimed at unravelling the effects of constant and varying resource allocations on the diabetic population.

CHAPTER THREE

MODELLING THE DYNAMICS OF DIABETES UNDER RESOURCE CONSTRAIN

3.1 Introduction

Two mathematical models that considers the significance of intervention and the limited hospital bed space on the dynamics of diabetes are formulated in sections of this chapter. The population is classified into four compartments. The first compartment is the Susceptible $S(t)$ compartment consisting of the population of individuals who do not have diabetes but are capable of having the disease. The second compartment is the Diabetic $D(t)$ compartment which consists of individuals who already have the disease but who have not developed complications. The third compartment is the Complicated $C(t)$ compartment which represents diabetic people who have developed complications as a result of disease. Finally, the fourth compartment is the Hospital $H(t)$ compartment which consists of the the complicated population that has been hospitalised. The reproduction number is calculated for the two models, non-negativity conditions are established for the two models and the stability of the equilibrium points are also obtained.

The models are formulated based on the following assumptions;

i. Healthy individuals give birth to healthy children.

ii. Diabetic adults give birth to children with disorders but still have possibility of giving birth to healthy children.

iii. Complications associated with the diabetes are curable but the individuals still remain diabetic.

iv. All diabetic population suffers related complications.

3.2 Under Constant Resource Allocation Rate

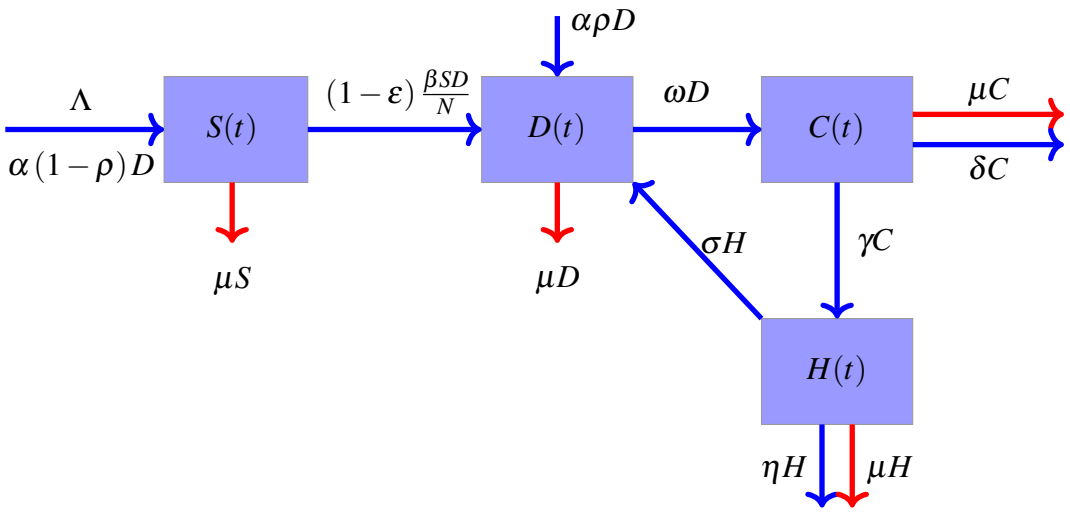

Figure 3.1: Flowchart for the model considering the available number of bed spaces

The dynamics of diabetes is modelled mathematically in this section. The four compartments are the Susceptible class $S(t)$ which represents the population of non-diabetic individuals who has the tendency to become diabetic, the Diabetic class $D(t)$ which represents the population of diabetic individuals, the Complicated class $C(t)$ which represents the class of those who have developed complications as a result of their diabetic conditions, and the Hospitalised class $H(t)$ which represents the class of the complicated diabetic patients who have been placed under close hospital monitoring and treatments. Figure (3.1) shows the flowchart of the dynamics of diabetes with a constant hospitalisation rate. The rate at which the lifestyle incidence occurs is ε $(0 < \varepsilon < 1.)$ The case of no impacted lifestyle is represented as $\varepsilon = 0$ while the case of the highest lifestyle standards $\varepsilon = 1$. If the rate of interaction between the susceptible and the diabetic compartments leading to an incidence is β, then the number of incident that occur due to lifestyle factors is

$$\frac{(1-\varepsilon)\beta SD}{N}.$$

Also, a number of diabetics are also caused by genetic factors of the parent who have a lineage of diabetes. The siblings of diabetics have the possibility of having a genetic disorder that can cause malfunction of the pancreas. We make an assumption that healthy individuals have heathy children and diabetics have children with disorders, but with a possibility of having heathy children. Suppose the birth rate is α and the proportion of genetic disorder's births is taken as ρ. More so, the number of births to the susceptible class is given by $\alpha S + \alpha(1-\rho)D$ while the children born with genetic disorders into the diabetic compartment is $\alpha\rho D$. It the rate at which diabetic patients develop complication is ω, then ωD will migrate from D compartment into the C compartment. The proportion of patients who die from complication is taken to be δ and therefore δC is removed from the C compartment. Setting γ as the constant hospitalization rate, then γC represents the population that migrates from the complicated class to the hospitalised class at any time t. Knowing that the complications arising from the diabetic conditions can be cured but there is no cure for diabetes, the patients whose complications have been cured are returned into the diabetic compartment at the rate σ, while the rate at which patients under treatment die at the rate η. Also, it is assumed that the natural mortality rate is μ. The governing differential equations is therefore given as

$$\frac{dS}{dt} = \Lambda + \alpha(1-\rho)D - (1-\varepsilon)\beta\frac{SD}{N} - \mu S, \tag{3.2.1}$$

$$\frac{dD}{dt} = \alpha\rho D + (1-\varepsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D, \tag{3.2.2}$$

$$\frac{dC}{dt} = \omega D - \gamma C - \delta C - \mu C, \tag{3.2.3}$$

$$\frac{dH}{dt} = \gamma C - \sigma H - \eta H - \mu H. \tag{3.2.4}$$

3.2.1 Equilibrium points

To obtain the equilibrium points, we shall set the right hand side of each of the equations (3.2.1 - 3.2.4) to zero as follows

$$0 = \Lambda + \alpha(1-\rho)D - (1-\varepsilon)\beta\frac{SD}{N} - \mu S, \tag{3.2.5}$$

$$0 = \alpha\rho D + (1-\varepsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D, \tag{3.2.6}$$

$$0 = \omega D - \gamma C - \delta C - \mu C, \tag{3.2.7}$$

$$0 = \gamma C - \sigma H - \eta H - \mu H. \tag{3.2.8}$$

The DFE is the equilibrium obtained in the absence of diabetes in the population, i.e.

$$D = C = H = 0. \tag{3.2.9}$$

and this gives the DFE E_0 as

$$E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right).$$

The endemic equilibrium point (EEP) represents the points at which the disease persists in the population. To obtain the EEP, the system of equations (3.2.5 - 3.2.8) is solved without any pre-determined value for the state variables. Considering the last two equations (3.2.7) and (3.2.8), we have

$$D = \frac{1}{\omega}(\gamma + \delta + \mu)C, \text{ and } H = \frac{\gamma C}{\sigma + \eta + \mu}.$$

Adding the equations (3.2.5) and (3.2.6), we have

$$\begin{aligned}
&\Lambda+(\alpha-\omega-\mu)D-\mu S+\sigma H=0\\
\Rightarrow &S=\frac{1}{\mu}\left(\Lambda+(\alpha-\omega-\mu)D+\sigma H\right)\\
\Rightarrow &S=\frac{\Lambda}{\mu}+\left(\frac{(\alpha-\omega-\mu)(\gamma+\delta+\mu)}{\omega}+\frac{\sigma\gamma}{\sigma+\eta+\mu}\right)\frac{C}{\mu}
\end{aligned}$$

Hence, letting $C=C^*$ the EEP E_1 is

$$D^*=\frac{C^*}{\omega}(\gamma+\delta+\mu),\ H^*=\frac{\gamma C^*}{\sigma+\eta+\mu},\ S=\frac{\Lambda}{\mu}+\left(\frac{(\alpha-\omega-\mu)(\gamma+\delta+\mu)}{\omega}+\frac{\sigma\gamma}{\sigma+\eta+\mu}\right)\frac{C^*}{\mu},$$

and the EEP is

$$E_1=(C^*,D^*,H^*,S^*).$$

3.2.2 Reproduction Number

The reproduction number R_0 is a measure of the number of secondary infections that will result from introducing a diabetic individual into the population. The next generation matrix method is used to determine R_0. Let the matrices F and V represent the new infections and negated outward transitions from these compartments respectively. The infected compartments are the D and C compartments and therefore we have

$$F=\begin{pmatrix}(1-\varepsilon)\beta\frac{SD}{N}\\0\end{pmatrix},\ V=\begin{pmatrix}-\sigma H+(\omega+\mu-\alpha\rho)D\\-\omega D+(\gamma+\delta+\mu)C\end{pmatrix}$$

and thus

$$\nabla F = \begin{pmatrix} (1-\varepsilon)\beta\frac{S}{N} & 0 \\ 0 & 0 \end{pmatrix}, \Rightarrow (\nabla F)_{E_0} = \begin{pmatrix} (1-\varepsilon)\frac{\beta\Lambda}{N\mu} & 0 \\ 0 & 0 \end{pmatrix}$$

$$\nabla V = (\nabla V)_{E_0} = \begin{pmatrix} \omega+\mu-\alpha\rho & 0 \\ -\omega & \gamma+\delta+\mu \end{pmatrix}$$

and

$$(\nabla V)^{-1}_{E_0} = \begin{pmatrix} \frac{1}{(\omega+\mu-\alpha\rho)} & 0 \\ \frac{\omega}{(\omega+\mu-\alpha\rho)(\gamma+\delta+\mu)} & \frac{1}{(\gamma+\delta+\mu)} \end{pmatrix}.$$

From which

$$(\nabla F)_{E_0}(\nabla V)^{-1}_{E_0} = \begin{pmatrix} \frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)} & 0 \\ 0 & 0 \end{pmatrix}.$$

The characteristic equation is obtained thus

$$\begin{vmatrix} \frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)} - \lambda & 0 \\ 0 & -\lambda \end{vmatrix} = 0 \Rightarrow \lambda\left(\frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)} - \lambda\right) = 0,$$

and the eigenvalues are

$$\lambda_1 = 0,\ \lambda_2 = \frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)},\ \text{with } \omega+\mu-\alpha\rho > 0$$

Finally, the basic reproduction number is

$$R_0 = \max\{\lambda_1, \lambda_2\} = \frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)},$$

3.2.3 Local Stability of the equilibrium points

Having obtained the equilibrium points and the basic reproduction number, the next step is to establish that the conditions for stability of the equilibrium points. Now, the

Jacobian matrix for the system (3.2.1 - 3.2.4) is

$$J = \begin{pmatrix} -\frac{(1-\varepsilon)\beta D}{N} - \mu & \alpha(1-\rho) - \frac{(1-\varepsilon)\beta S}{N} & 0 & 0 \\ \frac{(1-\varepsilon)\beta D}{N} & \alpha\rho + \frac{(1-\varepsilon)\beta S}{N} - \omega - \mu & 0 & \sigma \\ 0 & \omega & -\gamma - \delta - \mu & 0 \\ 0 & 0 & \gamma & -\sigma - \eta - \mu \end{pmatrix}.$$

The Jacobian at the DFE E_0 is

$$J_0 = \begin{pmatrix} -\mu & \alpha(1-\rho) - \frac{(1-\varepsilon)\beta\Lambda}{N\mu} & 0 & 0 \\ 0 & \alpha\rho + \frac{(1-\varepsilon)\beta\Lambda}{N\mu} - \mu - \omega & 0 & \sigma \\ 0 & \omega & -\gamma - \delta - \mu & 0 \\ 0 & 0 & \gamma & -\sigma - \eta - \mu \end{pmatrix}$$

and the Jacobian at the EEP E_1 is

$$J_1 = \begin{pmatrix} -\frac{(1-\varepsilon)\beta D^*}{N} - \mu & \alpha(1-\rho) - \frac{(1-\varepsilon)\beta S^*}{N} & 0 & 0 \\ \frac{(1-\varepsilon)\beta D^*}{N} & \alpha\rho + \frac{(1-\varepsilon)\beta S^*}{N} - \mu - \omega & 0 & \sigma \\ 0 & \omega & -\gamma - \delta - \mu & 0 \\ 0 & 0 & \gamma & -\sigma - \eta - \mu \end{pmatrix}.$$

The following theorems verify the local asymptotic stability conditions for the equilibrium points.

Theorem 3.2.1. *The DFE of system (3.2.1 - 3.2.4) is locally asymptotically stable if* $R_0 < 1$ *but unstable if* $R_0 > 1$.

Proof. The characteristic equation of the Jacobian at the DFE is given as

$$|J_{E_0} - \lambda I| = 0$$

$$\begin{vmatrix} -\mu-\lambda & \alpha(1-\rho) - \frac{(1-\varepsilon)\beta\Lambda}{N\mu} & 0 & 0 \\ 0 & \alpha\rho + \frac{(1-\varepsilon)\beta\Lambda}{N\mu} - \mu - \omega - \lambda & 0 & \sigma \\ 0 & \omega & -\gamma-\delta-\mu-\lambda & 0 \\ 0 & 0 & \gamma & -\sigma-\eta-\mu-\lambda \end{vmatrix} = 0,$$

$$(-\mu-\lambda)\begin{vmatrix} \alpha\rho + \frac{(1-\varepsilon)\beta\Lambda}{N\mu} - \mu - \omega - \lambda & 0 & \sigma \\ \omega & -\gamma-\delta-\mu-\lambda & 0 \\ 0 & \gamma & -\sigma-\eta-\mu-\lambda \end{vmatrix} = 0,$$

$$(-\mu-\lambda)\left(\left(\alpha\rho + \frac{(1-\varepsilon)\beta\Lambda}{N\mu} - \mu - \omega - \lambda\right)(-\gamma-\delta-\mu-\lambda)(-\sigma-\eta-\mu-\lambda) + \omega\sigma\gamma\right) = 0,$$

$$(\mu+\lambda)\left(\left(\alpha\rho + \frac{(1-\varepsilon)\beta\Lambda}{N\mu} - \mu - \omega - \lambda\right)(\gamma+\delta+\mu+\lambda)(\sigma+\eta+\mu+\lambda) + \omega\sigma\gamma\right) = 0,$$

$$(\mu+\lambda)\left((A_1-\lambda)(A_2+\lambda)(A_3+\lambda) + \omega\sigma\gamma\right) = 0,$$

where

$$A_1 = \frac{(1-\varepsilon)\beta\Lambda}{N\mu} - (\omega+\mu-\alpha\rho),\ A_2 = \gamma+\delta+\mu,\ A_3 = \sigma+\eta+\mu.$$

The first eigenvalue is $\lambda_1 = -\mu$ and the remaining three eigenvalues are obtained from

$$\lambda^3 + \xi_2\lambda^2 + \xi_1\lambda + \xi_0 = 0,$$

with

$$\xi_2 = A_2 + A_3 - A_1,\ \xi_1 = A_2A_3 - A_1A_2 - A_1A_3,\ \xi_0 = -(A_1A_2A_3 + \sigma\omega\gamma). \quad (3.2.10)$$

Using Routh-Hurwitz criteria, the three eigenvalues have negative real parts if

$$\xi_2 > 0, \quad \xi_2\xi_1 - \xi_0 > 0, \quad \xi_0 > 0. \tag{3.2.11}$$

By substituting (3.2.10) into (3.2.11), we have

condition 1: $A_2 + A_3 - A_1 > 0$,

condition 2: $(A_2 + A_3 - A_1)(A_2A_3 - A_1A_2 - A_1A_3) + (A_1A_2A_3 + \sigma\omega\gamma) > 0$

condition 3: $-(A_1A_2A_3 + \sigma\omega\gamma) > 0$.

From condition (1),

$$A_2 + A_3 > A_1$$

Assuming condition (1) and (3) hold, then condition (3) reduces to

$$A_2A_3 - A_1A_2 - A_1A_3 > 0 \Rightarrow A_2A_3 > A_1(A_2 + A_3) > A_1^2 > 0.$$

Now, back to condition (3),

$$A_1A_2A_3 + \sigma\omega\gamma < 0 \Rightarrow A_1A_2A_3 < -\sigma\omega\gamma < 0 \Rightarrow A_1 < 0 \text{ (since } A_2A_3 > 0)$$

therefore,

$$\frac{(1-\varepsilon)\beta\Lambda}{N\mu} - (\omega + \mu - \alpha\rho) < 0 \Rightarrow \frac{(1-\varepsilon)\frac{\beta\Lambda}{N\mu}}{(\omega + \mu - \alpha\rho)} - 1 < 0 \Rightarrow R_0 < 1.$$

Therefore, the DFE is asymptotically stable if $R_0 < 1$ but unstable if $R_0 > 1$. □

Theorem 3.2.2. *The EEP of system (3.2.1 - 3.2.4) is locally asymptotically stable if* $R_0 < 1$.

Proof. Letting

$$B_1 = \frac{(1-\varepsilon)\beta}{N},$$

the Jacobian at the EEP E_1 is

$$J_1 = \begin{pmatrix} -B_1D^* - \mu & \alpha(1-\rho) - B_1S^* & 0 & 0 \\ B_1D^* & \alpha\rho + B_1S^* - \mu - \omega & 0 & \sigma \\ 0 & \omega & -\gamma - \delta - \mu & 0 \\ 0 & 0 & \gamma & -\sigma - \eta - \mu \end{pmatrix}.$$

The characteristic equation of J_1 is

$$\begin{vmatrix} -B_1D^* - \mu - \lambda & \alpha(1-\rho) - B_1S^* & 0 & 0 \\ B_1D^* & \alpha\rho + B_1S^* - \mu - \omega - \lambda & 0 & \sigma \\ 0 & \omega & -\gamma - \delta - \mu - \lambda & 0 \\ 0 & 0 & \gamma & -\sigma - \eta - \mu - \lambda \end{vmatrix} = 0,$$

$$(-B_1D^* - \mu - \lambda) \begin{vmatrix} \alpha\rho + B_1S^* - \mu - \omega - \lambda & 0 & \sigma \\ \omega & -\gamma - \delta - \mu - \lambda & 0 \\ 0 & \gamma & -\sigma - \eta - \mu - \lambda \end{vmatrix}$$

$$- B_1D^* \begin{vmatrix} \alpha(1-\rho) - B_1S^* & 0 & 0 \\ \omega & -\gamma - \delta - \mu - \lambda & 0 \\ 0 & \gamma & -\sigma - \eta - \mu - \lambda \end{vmatrix} = 0,$$

and thus,

$$
\begin{aligned}
&(-B_1D^*-\mu-\lambda)\{(\alpha\rho+B_1S^*-\mu-\omega-\lambda)(-\gamma-\delta-\mu-\lambda)(-\sigma-\eta-\mu-\lambda)+\sigma\omega\gamma\}\\
&\qquad -B_1D^*(\alpha(1-\rho)-B_1S^*)(-\gamma-\delta-\mu-\lambda)(-\sigma-\eta-\mu-\lambda)=0,\\
&[B_1D^*(\alpha(1-\rho)-B_1S^*)+(\lambda+B_1D^*+\mu)(\alpha\rho+B_1S^*-\mu-\omega-\lambda)]\times\\
&\qquad (\lambda+B_2+\delta+\mu)(\lambda+\sigma+\eta+\mu)+\sigma\omega\gamma(\lambda+B_1D^*+\mu)=0,\\
&\left[\lambda^2+(B_1D^*+\mu-\alpha\rho-B_1S^*+\mu+\omega)\lambda+(B_1D^*+\mu)(\mu+\omega)-\mu(\alpha\rho+B_1S^*)-\alpha B_1D^*\right]\times\\
&\left(\lambda^2+(B_2+\delta+\mu+\sigma+\eta+\mu)\lambda+(\gamma+\delta+\mu)(\sigma+\eta+\mu)\right)-\sigma\omega\gamma\lambda-\sigma\omega\gamma(B_1D^*+\mu)=0.
\end{aligned}
$$

Setting

$$
\begin{aligned}
A_1 =& B_1D^*+\mu-\alpha\rho-B_1S^*+\mu+\omega,\\
A_2 =& (B_1D^*+\mu)(\mu+\omega)-\mu(\alpha\rho+B_1S^*)-\alpha B_1D^*,\\
A_3 =& \gamma+\delta+\mu+\sigma+\eta+\mu,\\
A_4 =& (\gamma+\delta+\mu)(\sigma+\eta+\mu),
\end{aligned}
$$

and the characteristic equation becomes

$$\lambda^4+\xi_3\lambda^3+\xi_2\lambda^2+\xi_1\lambda+\xi_0=0,$$

where

$$
\begin{aligned}
\xi_0 =& A_2A_4-\sigma\omega\gamma(B_1D^*+\mu),\\
\xi_1 =& A_1A_4+A_2A_3-\sigma\omega\gamma,\\
\xi_2 =& A_1A_3+A_2+A_4,\ \xi_3=A_1+A_3,
\end{aligned}
\tag{3.2.12}
$$

Using Routh-Hurwitz criteria, the four eigenvalues have negative real parts if

$$\xi_3 > 0, \quad \frac{\xi_3\xi_2 - \xi_1}{\xi_3} > 0, \quad \frac{(\xi_3\xi_2 - \xi_1)\,\xi_1}{\xi_3} - \xi_3\xi_0 > 0, \quad \xi_0 > 0. \tag{3.2.13}$$

By substituting (3.2.12) into (3.2.13), we have

$$\begin{aligned}
&\text{condition 1: } A_1 + A_3 > 0,\\
&\text{condition 2: } \frac{(A_1+A_3)(A_1A_3+A_2+A_4) - (A_1A_4+A_2A_3-\sigma\omega B_2)}{A_1+A_3} > 0\\
&\text{condition 3: } \left(\frac{(A_1+A_3)(A_1A_3+A_2+A_4) - (A_1A_4+A_2A_3-\sigma\omega B_2)}{A_1+A_3}\right)\\
&\qquad\qquad - (A_1+A_3)(A_2A_4 - \sigma\omega B_2(B_1D^* + \mu)) > 0\\
&\text{condition 4: } A_2A_4 - \sigma\omega B_2(B_1D^* + \mu) > 0
\end{aligned}$$

Using condition (1) in (2) gives

$$(A_1+A_3)(A_1A_3+A_2+A_4) - (A_1A_4+A_2A_3-\sigma\omega B_2) > 0. \tag{3.2.14}$$

Rewriting condition (3) gives

$$\frac{(A_1+A_3)(A_1A_3+A_2+A_4) - (A_1A_4+A_2A_3-\sigma\omega B_2)}{(A_2A_4 - \sigma\omega B_2(B_1D^* + \mu))} > (A_1+A_3)^2 > 0,$$

provided

$$A_2A_4 - \sigma\omega B_2(B_1D^* + \mu) > 0.$$

Hence, the four conditions are satisfied as long as condition (4) is satisfied, i.e.

$$A_2A_4 - \sigma\omega B_2(B_1D^* + \mu) > 0 \tag{3.2.15}$$

which implies that

$$
\begin{aligned}
&A_2A_4 > \sigma\omega B_2\left(B_1D^* + \mu\right) > 0 \;\Rightarrow\; A_2 > 0 \;\text{ since } A_4 > 0\\
&-\left(B_1D^* + \mu\right)(\mu + \omega) + \mu\left(\alpha\rho + B_1S^*\right) + \alpha B_1D^* < 0\\
&-\left(\omega + \mu - \alpha\rho - B_1S^*\right)\mu - B_1D^*\left(\mu + \omega - \alpha\right) < 0\\
&-\left(1 - \frac{B_1S^*}{\omega + \mu - \alpha\rho}\right)\mu - B_1D^*\frac{(\mu + \omega - \alpha)}{\omega + \mu - \alpha\rho} < 0\\
&\left(\frac{B_1S^*}{\omega + \mu - \alpha\rho} - 1\right)\mu < B_1D^*\frac{(\mu + \omega - \alpha)}{\omega + \mu - \alpha\rho} < 0\\
&(R_0 - 1)\,\mu < 0 \;\Rightarrow\; R_0 < 1
\end{aligned}
$$

Therefore, the EEP is asymptotically stable if $R_0 < 1$ but unstable if $R_0 > 1$. □

3.2.4 Global Stability

Theorem 3.2.3. *The solution remains positive and bounded for positive initial conditions* $S(t_0), D(t_0), C(t_0)$ *and* $H(t_0)$.

Proof. Setting

$$N = S + D + C + H$$

and summing up the system (3.2.1 - 3.2.4), then it gives

$$\frac{dN}{dt} = \Lambda - \delta C - \eta H - \mu N \leq \Lambda - \mu N$$

which on solving gives

$$N \leq \frac{\Lambda}{\mu} - \left(\frac{\Lambda}{\mu} - N(t_0)\right)\exp\left(\mu t_0 - \mu t\right).$$

As $t \to \infty$,

$$N \leq \frac{\Lambda}{\mu}.$$

Hence, the solution space $\mathscr{R}$ is bounded, so that

$$\mathscr{R} = \left\{ (S,D,C,H) \ni N = S+D+C+H \leq \frac{\Lambda}{\mu} \right\}.$$

Now, from equation (3.2.1 - 3.2.4),

$$\begin{aligned}
&\frac{dS}{dt} = \Lambda + \alpha(1-\rho)D - (1-\varepsilon)\beta\frac{SD}{N} - \mu S \leq -\mu S \Rightarrow S \leq S(t_0)\exp(\mu t_0 - \mu t),\\
&\frac{dD}{dt} = \alpha\rho D + (1-\varepsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D \leq -(\mu+\omega)D \Rightarrow D \leq D(t_0)\exp((\mu+\omega)(t_0-t)),\\
&\frac{dC}{dt} = \omega D - \gamma C - \delta C - \mu C \leq -(\gamma+\delta+\mu)C \Rightarrow C \leq C(t_0)\exp((\gamma+\delta+\mu)(t_0-t)),\\
&\frac{dH}{dt} = \gamma C - \sigma H - \eta H - \mu H \leq -(\sigma+\delta+\mu)C \Rightarrow H \leq H(t_0)\exp((\sigma+\delta+\mu)(t_0-t)).
\end{aligned}$$

Thus, as long as the initial conditions $S(t_0), D(t_0), C(t_0)$ and $H(t_0)$ are positive, then the solutions S,D,C,H remain positive in the region $\mathscr{R}$. □

3.3 Mathematical Model of the Significance of Strained Resources on Diabetes Dynamics

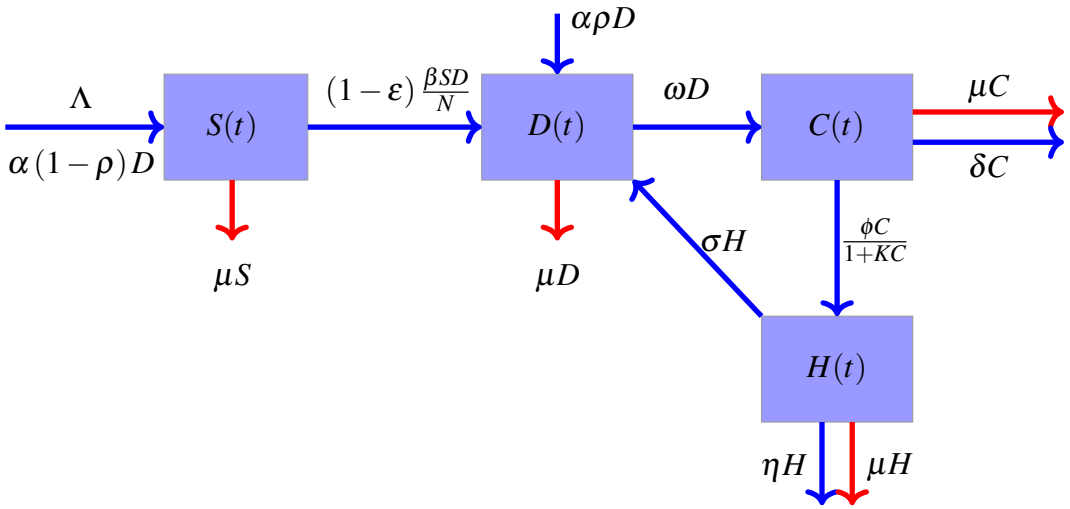

Figure 3.2: Flowchart for the model considering intervention

Figure (3.2) shows the flowchart of the dynamics of diabetes with the treatment as an intervention. The increase in the number of diabetics is dependent on the number of incidents that occur. One of causes of the incidences is unhealthy lifestyle comprising of irregular poor eating patterns and low level of physical exercise. According to Hill (2013) interaction between diabetics who have unhealthy lifestyle and healthy individuals can lead to lifestyle transmissions. Lifestyle transmission results in incident and it increases the prevalence of diabetes. Let ε be the rate at which the lifestyle incidence occurs with the condition that $0 < \varepsilon < 1$, such that $\varepsilon = 0$ implies that there is no impacted lifestyle and $\varepsilon = 1$ implies the highest standards of lifestyle. If the rate of interaction between the susceptible and the diabetic compartments leading to an incidence is β, then the number of incident that occur due to lifestyle factors is

$$\frac{(1-\varepsilon)\beta SD}{N}.$$

Also, a number of diabetics are also caused by genetic factors of the parent who have a lineage of diabetes. The siblings of diabetics have the possibility of having a genetic disorder that can cause malfunction of the pancreas. We make an assumption that healthy individuals have heathy children and diabetics have children with disorders, but with a possibility of having heathy children. Suppose the birth rate is α and the proportion of genetic disorder's births is taken as ρ. More so, the number of births to the susceptible class is given by $\Lambda + \alpha(1-\rho)D$ while the children born with genetic disorders into the diabetic compartment is $\alpha\rho D$. It the rate at which diabetic patients develop complication is ω, then ωD will migrate from D compartment into the C compartment. The proportion of patients who die from complication is taken to be δ and therefore δC is removed from the C compartment. Therefore a saturation treatment function proposed by (Zhang *et al.*, 2014; Abdelrazec *et al.*, 2016) is of the form

$$f(C) = \frac{\phi C}{1+KC}$$

where ϕ is the per capita hospitalisation rate at any time and K is the saturation parameter that influences delay before treatment due to insufficient resources). For a few diabetic cases, $f(C) \approx \phi C$, and for a large number of diabetic cases, $f(C) \to \phi/K$. This is shown in Figure (3.3).

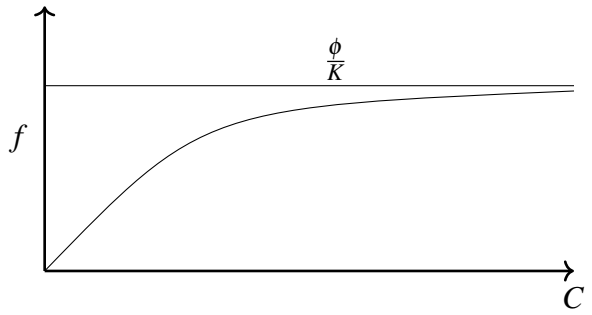

Figure 3.3: Graph of saturation treatment f against the complicated cases C.

Knowing that the complications arising from the diabetic conditions can be cured but there is no cure for diabetes, the patients whose complications have been cured are returned into the diabetic compartment at the rate σ, while the rate at which patients under treatment die at the rate η. Also, it is assumed that the natural death rate is μ. The governing differential equations is therefore given as

$$\frac{dS}{dt} = \Lambda + \alpha(1-\rho)D - (1-\varepsilon)\beta\frac{SD}{N} - \mu S, \tag{3.3.1}$$

$$\frac{dD}{dt} = \alpha\rho D + (1-\varepsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D, \tag{3.3.2}$$

$$\frac{dC}{dt} = \omega D - \frac{\phi C}{1+KC} - \delta C - \mu C, \tag{3.3.3}$$

$$\frac{dH}{dt} = \frac{\phi C}{1+KC} - \sigma H - \eta H - \mu H. \tag{3.3.4}$$

3.3.1 Equilibrium points

To obtain the equilibrium points, we shall set the right hand side of each of the equations (3.3.1 - 3.3.4) to zero as follows

$$0 = \Lambda + \alpha(1-\rho)D - (1-\varepsilon)\beta\frac{SD}{N} - \mu S, \tag{3.3.5}$$

$$0 = \alpha\rho D + (1-\varepsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D, \tag{3.3.6}$$

$$0 = \omega D - \frac{\phi C}{1+KC} - \delta C - \mu C, \tag{3.3.7}$$

$$0 = \frac{\phi C}{1+KC} - \sigma H - \eta H - \mu H. \tag{3.3.8}$$

Two types of equilibrium points are obtained from here, namely; the diabetes-free equilibrium (DFE) and the endemic equilibrium point (EEP). The DFE is the equilibrium obtained in the absence of diabetes in the population i.e.

$$D = C = H = 0. \tag{3.3.9}$$

The only solution allowed for equations (3.3.5 - 3.3.8) under the conditions (3.3.9) is the diabetes-free equilibrium E_0 obtained as

$$E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right).$$

The endemic equilibrium point (EEP) represents the points at which the disease persists in the population. To obtain the EEP, the system of equations (3.3.5 - 3.3.8) is solved. From equations (3.3.7) and (3.3.8), we have

$$D = \frac{1}{\omega}\left(\frac{\phi}{1+KC} + \delta + \mu\right)C, \text{ and } H = \frac{\phi C}{(\sigma+\eta+\mu)(1+KC)}.$$

Adding the equations (3.3.5) and (3.3.6), we have

$$\Lambda + (\alpha - \omega - \mu)D - \mu S + \sigma H = 0 \Rightarrow S = \frac{1}{\mu}(\Lambda + (\alpha - \omega - \mu)D + \sigma H).$$

Hence, letting $C = C^*$ the EEP is

$$D^* = \frac{C^*}{\omega}\left(\frac{\phi}{1+KC^*} + \delta + \mu\right),\ H^* = \frac{\phi C^*}{(\sigma+\eta+\mu)(1+KC^*)},$$
$$S^* = \frac{\Lambda + (\alpha - \omega - \mu)D^* + \sigma H^*}{\mu},$$

and the EEP is

$$E_1 = (C^*, D^*, H^*, S^*).$$

3.3.2 Reproduction Number

The reproduction number R_0 is a measure of the number of secondary infections that will result from introducing a diabetic individual into the population. The next generation matrix method is used to determine R_0. Let the matrices F and V represent the new infections and negated outward transitions from these compartments respectively. The infected compartments are the D and C compartments and therefore we have

$$F = \begin{pmatrix} (1-\varepsilon)\beta\frac{SD}{N} \\ 0 \end{pmatrix},\ V = \begin{pmatrix} -\alpha\rho D - \sigma H + \omega D + \mu D \\ -\omega D + \frac{\phi C}{1+KC} + \delta C + \mu C \end{pmatrix}$$

and thus

$$\nabla F = \begin{pmatrix} (1-\varepsilon)\beta\frac{S}{N} & 0 \\ 0 & 0 \end{pmatrix},\ \Rightarrow\ (\nabla F)_{E_0} = \begin{pmatrix} (1-\varepsilon)\frac{\beta\Lambda}{N\mu} & 0 \\ 0 & 0 \end{pmatrix}$$
$$\nabla V = \begin{pmatrix} -\alpha\rho + \omega + \mu & 0 \\ -\omega & \frac{\phi}{(1+KC)^2} + \delta + \mu \end{pmatrix}$$
$$(\nabla V)_{E_0} = \begin{pmatrix} -\alpha\rho + \omega + \mu & 0 \\ -\omega & \phi + \delta + \mu \end{pmatrix}$$

The determinant of ∇V is obtained as

$$\left|(\nabla V)_{E_0}\right| = (\omega+\mu-\alpha\rho)(\phi+\delta+\mu)$$

and the inverse of ∇V is

$$(\nabla V)^{-1}_{E_0} = \begin{pmatrix} \frac{1}{(\omega+\mu-\alpha\rho)} & 0 \\ \frac{\omega}{(\omega+\mu-\alpha\rho)(\phi+\delta+\mu)} & \frac{1}{(\phi+\delta+\mu)} \end{pmatrix}$$

and

$$(\nabla F)_{E_0}(\nabla V)^{-1}_{E_0} = \begin{pmatrix} \frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)} & 0 \\ 0 & 0 \end{pmatrix}.$$

The characteristic equation is obtained thus

$$\begin{vmatrix} \frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)} - \lambda & 0 \\ 0 & -\lambda \end{vmatrix} = 0 \Rightarrow \lambda\left(\frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)} - \lambda\right) = 0,$$

and the eigenvalues are

$$\lambda_1 = 0,\ \lambda_2 = \frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)},$$

Finally, the reproduction number is

$$R_0 = \max\{\lambda_1,\lambda_2\} = \frac{(1-\varepsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)},\ \text{with } \omega+\mu-\alpha\rho > 0 \tag{3.3.10}$$

3.3.3 Local Stability of the equilibrium points

The Jacobian matrix for the system (3.3.1 - 3.3.4) is

$$J = \begin{pmatrix} -\frac{(1-\varepsilon)\beta D}{N} - \mu & \alpha(1-\rho) - \frac{(1-\varepsilon)\beta S}{N} & 0 & 0 \\ \frac{(1-\varepsilon)\beta D}{N} & \alpha\rho + \frac{(1-\varepsilon)\beta S}{N} - \omega - \mu & 0 & \sigma \\ 0 & \omega & -\frac{\phi}{(1+KC)^2} - \delta - \mu & 0 \\ 0 & 0 & \frac{\phi}{(1+KC)^2} & -\sigma - \eta - \mu \end{pmatrix}.$$

The Jacobian at the DFE E_0 is obtained as

$$J_0 = \begin{pmatrix} -\mu & \alpha(1-\rho) - \frac{(1-\varepsilon)\beta\Lambda}{N\mu} & 0 & 0 \\ 0 & \alpha\rho + \frac{(1-\varepsilon)\beta\Lambda}{N\mu} - \mu - \omega & 0 & \sigma \\ 0 & \omega & -\phi - \delta - \mu & 0 \\ 0 & 0 & \phi & -\sigma - \eta - \mu \end{pmatrix}$$

and the Jacobian at the EEP E_1 is obtained as

$$J_1 = \begin{pmatrix} -\frac{(1-\varepsilon)\beta D^*}{N} - \mu & \alpha(1-\rho) - \frac{(1-\varepsilon)\beta S^*}{N} & 0 & 0 \\ \frac{(1-\varepsilon)\beta D^*}{N} & \alpha\rho + \frac{(1-\varepsilon)\beta S^*}{N} - \mu - \omega & 0 & \sigma \\ 0 & \omega & -\frac{\phi}{(1+KC^*)^2} - \delta - \mu & 0 \\ 0 & 0 & \frac{\phi}{(1+KC^*)^2} & -\sigma - \eta - \mu \end{pmatrix}.$$

The following theorems verify the local asymptotic stability of the equilibrium points.

Theorem 3.3.1. *The DFE of system (3.3.1 - 3.3.4) is locally asymptotically stable if* $R_0 < 1$ *but unstable if* $R_0 > 1$.

Proof. The characteristic equation of the Jacobian at the DFE is given as $|J_{E_0} - \lambda I| =$

0 and thus

$$\begin{vmatrix} -\mu-\lambda & \alpha(1-\rho)-\frac{(1-\varepsilon)\beta\Lambda}{N\mu} & 0 & 0 \\ 0 & \alpha\rho+\frac{(1-\varepsilon)\beta\Lambda}{N\mu}-\mu-\omega-\lambda & 0 & \sigma \\ 0 & \omega & -\phi-\delta-\mu-\lambda & 0 \\ 0 & 0 & \phi & -\sigma-\eta-\mu-\lambda \end{vmatrix} = 0,$$

Evaluating along the first column gives

$$(-\mu-\lambda)\begin{vmatrix} \alpha\rho+\frac{(1-\varepsilon)\beta\Lambda}{N\mu}-\mu-\omega-\lambda & 0 & \sigma \\ \omega & -\phi-\delta-\mu-\lambda & 0 \\ 0 & \phi & -\sigma-\eta-\mu-\lambda \end{vmatrix} = 0.$$

Evaluating along the first row further gives

$$(-\mu-\lambda)\left(\alpha\rho+\frac{(1-\varepsilon)\beta\Lambda}{N\mu}-\mu-\omega-\lambda\right)(-\phi-\delta-\mu-\lambda)(-\sigma-\eta-\mu-\lambda) - (\mu+\lambda)\omega\sigma\phi = 0$$

Rearranging gives

$$(\mu+\lambda)\left(\alpha\rho+\frac{(1-\varepsilon)\beta\Lambda}{N\mu}-\mu-\omega-\lambda\right)(\phi+\delta+\mu+\lambda)(\sigma+\eta+\mu+\lambda) + (\mu+\lambda)\omega\sigma\phi = 0,$$

and thus

$$(\mu+\lambda)((A_1-\lambda)(A_2+\lambda)(A_3+\lambda)+\omega\sigma\phi) = 0,$$

where

$$A_1 = \frac{(1-\varepsilon)\beta\Lambda}{N\mu}-(\omega+\mu-\alpha\rho),\ A_2 = \phi+\delta+\mu,\ A_3 = \sigma+\eta+\mu.$$

The first eigenvalue is $\lambda_1 = -\mu$ and the remaining three eigenvalues are obtained from

$$\lambda^3 + \xi_2\lambda^2 + \xi_1\lambda + \xi_0 = 0,$$

with

$$\xi_2 = A_2 + A_3 - A_1,\ \xi_1 = A_2A_3 - A_1A_2 - A_1A_3,\ \xi_0 = -\left(A_1A_2A_3 + \sigma\omega\phi\right). \quad (3.3.11)$$

Using Routh Hurwitz criteria, the three eigenvalues have negative real parts if

$$\xi_2 > 0, \quad \xi_1 > 0, \quad \xi_0 > 0. \quad (3.3.12)$$

By substituting (3.3.11) into (3.3.12), we have

$$\text{condition 1: } A_2 + A_3 - A_1 > 0,$$

$$\text{condition 2: } A_2A_3 - A_1A_2 - A_1A_3 > 0$$

$$\text{condition 3: } -\left(A_1A_2A_3 + \sigma\omega\phi\right) > 0.$$

From condition (1), $A_2 + A_3 > A_1$ and from condition (2),

$$A_2A_3 > A_1\left(A_2 + A_3\right) > A_1^2 > 0.$$

From condition (3),

$$A_1A_2A_3 + \sigma\omega\phi < 0 \ \Rightarrow\ A_1A_2A_3 < -\sigma\omega\phi < 0 \ \Rightarrow\ A_1 < 0 \ (\text{since } A_2A_3 > 0)$$

therefore,

$$\frac{(1-\varepsilon)\beta\Lambda}{N\mu} - (\omega + \mu - \alpha\rho) < 0 \ \Rightarrow\ \frac{(1-\varepsilon)\frac{\beta\Lambda}{N\mu}}{(\omega + \mu - \alpha\rho)} - 1 < 0 \ \Rightarrow\ R_0 < 1.$$

Therefore, the DFE is asymptotically stable if $R_0 < 1$ but unstable if $R_0 > 1$. □

Theorem 3.3.2. *The EEP of system (3.3.1 - 3.3.4) is locally asymptotically stable if* $R_0 > 1$.

Proof. The Jacobian at the EEP E_1 is

$$J_1 = \begin{pmatrix} -\frac{(1-\varepsilon)\beta D^*}{N} - \mu & \alpha(1-\rho) - \frac{(1-\varepsilon)\beta S^*}{N} & 0 & 0 \\ \frac{(1-\varepsilon)\beta D^*}{N} & \alpha\rho + \frac{(1-\varepsilon)\beta S^*}{N} - \mu - \omega & 0 & \sigma \\ 0 & \omega & -\frac{\phi}{(1+KC^*)^2} - \delta - \mu & 0 \\ 0 & 0 & \frac{\phi}{(1+KC^*)^2} & -\sigma - \eta - \mu \end{pmatrix}$$

and letting

$$B_1 = \frac{(1-\varepsilon)\beta}{N},\ B_2 = \frac{\phi}{(1+KC^*)^2}$$

then

$$J_1 = \begin{pmatrix} -B_1 D^* - \mu & \alpha(1-\rho) - B_1 S^* & 0 & 0 \\ B_1 D^* & \alpha\rho + B_1 S^* - \mu - \omega & 0 & \sigma \\ 0 & \omega & -B_2 - \delta - \mu & 0 \\ 0 & 0 & B_2 & -\sigma - \eta - \mu \end{pmatrix}$$

The characteristic equation of J_1 is

$$\begin{vmatrix} -B_1 D^* - \mu - \lambda & \alpha(1-\rho) - B_1 S^* & 0 & 0 \\ B_1 D^* & \alpha\rho + B_1 S^* - \mu - \omega - \lambda & 0 & \sigma \\ 0 & \omega & -B_2 - \delta - \mu - \lambda & 0 \\ 0 & 0 & B_2 & -\sigma - \eta - \mu - \lambda \end{vmatrix} = 0,$$

$$(-B_1D^*-\mu-\lambda)\begin{vmatrix} \alpha\rho+B_1S^*-\mu-\omega-\lambda & 0 & \sigma \\ \omega & -B_2-\delta-\mu-\lambda & 0 \\ 0 & B_2 & -\sigma-\eta-\mu-\lambda \end{vmatrix}$$
$$-B_1D^*\begin{vmatrix} \alpha(1-\rho)-B_1S^* & 0 & 0 \\ \omega & -B_2-\delta-\mu-\lambda & 0 \\ 0 & B_2 & -\sigma-\eta-\mu-\lambda \end{vmatrix}=0,$$

and thus,

$$\begin{aligned}(-B_1D^*-\mu-\lambda)(\alpha\rho+B_1S^*-\mu-\omega-\lambda)(-B_2-\delta-\mu-\lambda)(-\sigma-\eta-\mu-\lambda)\\ -B_1D^*(\alpha(1-\rho)-B_1S^*)(-B_2-\delta-\mu-\lambda)(-\sigma-\eta-\mu-\lambda)\\ -(B_1D^*+\mu+\lambda)\sigma\omega B_2=0,\\ [B_1D^*(\alpha(1-\rho)-B_1S^*)+(\lambda+B_1D^*+\mu)(\alpha\rho+B_1S^*-\mu-\omega-\lambda)]\times\\ (\lambda+B_2+\delta+\mu)(\lambda+\sigma+\eta+\mu)+\sigma\omega B_2(\lambda+B_1D^*+\mu)=0,\end{aligned}$$

$$\begin{aligned}[-(B_1D^*+\mu)(\mu+\omega)+\mu(\alpha\rho+B_1S^*)+\alpha B_1D^*]\left(\lambda^2+(B_2+\delta+\mu+\sigma+\eta+\mu)\lambda\right)\\ +[-(B_1D^*+\mu)(\mu+\omega)+\mu(\alpha\rho+B_1S^*)+\alpha B_1D^*](B_2+\delta+\mu)(\sigma+\eta+\mu)\\ +\left[\lambda^2+(B_1D^*+\mu-\alpha\rho-B_1S^*+\mu+\omega)\lambda\right]\left(\lambda^2+(B_2+\delta+\mu+\sigma+\eta+\mu)\lambda\right)\\ +\left[\lambda^2+(B_1D^*+\mu-\alpha\rho-B_1S^*+\mu+\omega)\lambda\right](B_2+\delta+\mu)(\sigma+\eta+\mu)\\ -\sigma\omega B_2\lambda-\sigma\omega B_2(B_1D^*+\mu)=0.\end{aligned}$$

Setting

$$\begin{aligned}A_1&=B_1D^*+\mu-\alpha\rho-B_1S^*+\mu+\omega,\\ A_2&=-(B_1D^*+\mu)(\mu+\omega)+\mu(\alpha\rho+B_1S^*)+\alpha B_1D^*,\\ A_3&=B_2+\delta+\mu+\sigma+\eta+\mu,\ A_4=(B_2+\delta+\mu)(\sigma+\eta+\mu),\end{aligned}$$

and the characteristic equation becomes

$$\lambda^4 + \xi_3\lambda^3 + \xi_2\lambda^2 + \xi_1\lambda + \xi_0 = 0,$$

where

$$\begin{aligned}
\xi_0 &= A_2A_4 - \sigma\omega B_2\left(B_1D^* + \mu\right), \\
\xi_1 &= A_1A_4 + A_2A_3 - \sigma\omega B_2, \\
\xi_2 &= A_1A_3 + A_2 + A_4,\ \xi_3 = A_1 + A_3,
\end{aligned} \tag{3.3.13}$$

Using Routh-Hurwitz criteria, the four eigenvalues have negative real parts if

$$\xi_3 > 0, \quad \frac{\xi_3\xi_2 - \xi_1}{\xi_3} > 0, \quad \frac{\left(\xi_3\xi_2 - \xi_1\right)\xi_1}{\xi_3} - \xi_3\xi_0 > 0, \quad \xi_0 > 0. \tag{3.3.14}$$

By substituting (3.3.13) into (3.3.14), we have

$$\begin{aligned}
&\text{condition 1: } A_1 + A_3 > 0, \\
&\text{condition 2: } \frac{\left(A_1 + A_3\right)\left(A_1A_3 + A_2 + A_4\right) - \left(A_1A_4 + A_2A_3 - \sigma\omega B_2\right)}{A_1 + A_3} > 0 \\
&\text{condition 3: } \left(\frac{\left(A_1 + A_3\right)\left(A_1A_3 + A_2 + A_4\right) - \left(A_1A_4 + A_2A_3 - \sigma\omega B_2\right)}{A_1 + A_3}\right) \\
&\qquad\qquad - \left(A_1 + A_3\right)\left(A_2A_4 - \sigma\omega B_2\left(B_1D^* + \mu\right)\right) > 0 \\
&\text{condition 4: } A_2A_4 - \sigma\omega B_2\left(B_1D^* + \mu\right) > 0
\end{aligned}$$

Using condition (1) in (2) gives

$$\left(A_1 + A_3\right)\left(A_1A_3 + A_2 + A_4\right) - \left(A_1A_4 + A_2A_3 - \sigma\omega B_2\right) > 0. \tag{3.3.15}$$

Rewriting condition (3) gives

$$\frac{\left(A_1 + A_3\right)\left(A_1A_3 + A_2 + A_4\right) - \left(A_1A_4 + A_2A_3 - \sigma\omega B_2\right)}{\left(A_2A_4 - \sigma\omega B_2\left(B_1D^* + \mu\right)\right)} > \left(A_1 + A_3\right)^2 > 0,$$

provided

$$A_2A_4 - \sigma\omega B_2 (B_1D^* + \mu) > 0.$$

Hence, the four conditions are satisfied as long as condition (4) is satisfied, i.e.

$$A_2A_4 - \sigma\omega B_2 (B_1D^* + \mu) > 0 \tag{3.3.16}$$

which implies that

$$\begin{aligned}
&A_2A_4 > \sigma\omega B_2 (B_1D^* + \mu) > 0 \Rightarrow A_2 > 0 \text{ since } A_4 > 0 \\
&-(B_1D^* + \mu)(\mu + \omega) + \mu(\alpha\rho + B_1S^*) + \alpha B_1D^* > 0 \\
&-(\omega + \mu - \alpha\rho - B_1S^*)\mu - B_1D^*(\mu + \omega - \alpha) > 0 \\
&-\left(1 - \frac{B_1S^*}{\omega + \mu - \alpha\rho}\right)\mu - B_1D^*\frac{(\mu + \omega - \alpha)}{\omega + \mu - \alpha\rho} > 0 \\
&-\left(1 - \frac{B_1S^*}{\omega + \mu - \alpha\rho}\right)\mu > B_1D^*\frac{(\mu + \omega - \alpha)}{\omega + \mu - \alpha\rho} > 0 \\
&-(1 - R_0)\mu > 0 \Rightarrow 1 - R_0 < 0 \Rightarrow \quad R_0 > 1
\end{aligned}$$

Therefore, the EEP is asymptotically stable if $R_0 > 1$ but unstable if $R_0 < 1$. □

3.3.4 Global Stability

Theorem 3.3.3. *The solution remains positive and bounded for positive initial conditions* $S(t_0), D(t_0), C(t_0)$ *and* $H(t_0)$.

Proof. Setting $N = S + D + C + H$ and summing up the system (3.3.1 - 3.3.4), then it gives

$$\frac{dN}{dt} = \Lambda - \delta C - \eta H - \mu N \leq \Lambda - \mu N$$

which on solving gives

$$N \leq \frac{\Lambda}{\mu} - \left(\frac{\Lambda}{\mu} - N(t_0)\right)\exp(\mu t_0 - \mu t).$$

As $t \to \infty$, $N \to \frac{\Lambda}{\mu}$. Hence, the solution space $\mathscr{R}$ is bounded, so that

$$\mathscr{R} = \left\{ (S,D,C,H) \ni N = S+D+C+H \leq \frac{\Lambda}{\mu} \right\}.$$

Now, from equation (3.2.1 - 3.2.4),

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda + \alpha(1-\rho)D - (1-\varepsilon)\beta\frac{SD}{N} - \mu S \geq -\mu S \\
&\Rightarrow S \geq S(t_0)\exp(\mu t_0 - \mu t), \\
\frac{dS}{dt} &= \Lambda + \alpha(1-\rho)D - (1-\varepsilon)\beta\frac{SD}{N} - \mu S \geq -\mu S \\
&\Rightarrow S \geq S(t_0)\exp(\mu t_0 - \mu t), \\
\frac{dD}{dt} &= \alpha\rho D + (1-\varepsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D \geq -(\mu+\omega)D \\
&\Rightarrow D \geq D(t_0)\exp((\mu+\omega)(t_0-t)), \\
\frac{dC}{dt} &= \omega D - \frac{\phi C}{1+KC} - \delta C - \mu C \geq -(\phi+\delta+\mu)C \\
&\Rightarrow C \geq C(t_0)\exp((\phi+\delta+\mu)(t_0-t)), \\
\frac{dH}{dt} &= \frac{\phi C}{1+KC} - \sigma H - \eta H - \mu H \geq -(\sigma+\delta+\mu)C \\
&\Rightarrow H \geq H(t_0)\exp((\sigma+\delta+\mu)(t_0-t)).
\end{aligned}$$

Thus, as long as the initial conditions $S(t_0), D(t_0), C(t_0)$ and $H(t_0)$ are positive, then the solutions S,D,C,H remain positive in the region $\mathscr{R}$. □

Theorem 3.3.4. *There is a Lyapunov function candidate*

Proof. Consider the linear Lyapunov function

$$\mathscr{L} = D + aC + bH,\ a > 0, b > 0.$$

Now, the time-derivative is

$$\dot{\mathscr{L}} = \dot{D} + a\dot{C} + b\dot{H}$$

and

$$\begin{aligned}\dot{\mathscr{L}} =&\alpha\rho D+(1-\varepsilon)\beta\frac{SD}{N}+\sigma H-\omega D-\mu D\\ &+a\left(\omega D-\frac{\phi C}{1+KC}-\delta C-\mu C\right)+b\left(\frac{\phi C}{1+KC}-\sigma H-\eta H-\mu H\right)\\ \dot{\mathscr{L}} =&\left(\alpha\rho-\omega-\mu+(1-\varepsilon)\beta\frac{S}{N}+a\omega\right)D+(\sigma-b\sigma-b\eta-b\mu)H\\ &+\left(\frac{b\phi}{1+KC}-\frac{a\phi}{1+KC}-a\delta-a\mu\right)C\end{aligned}$$

Choose a,b so that

$$\sigma-b\sigma-b\eta-b\mu=0\ \Rightarrow\ b=\frac{\sigma}{\sigma+\eta+\mu},$$

and setting $a=b=\frac{\sigma}{\sigma+\eta+\mu}$, then

$$\dot{\mathscr{L}}=\left(\alpha\rho-\omega-\mu+(1-\varepsilon)\beta\frac{S}{N}+\frac{\sigma\omega}{\sigma+\eta+\mu}\right)D-(a\delta+a\mu)C$$

Since $-(a\delta+a\mu)<0$, then the sign of $\dot{\mathscr{L}}$ depends on the first term. Considering that $D>0$, then the terms in the bracket needs to satisfy the condition

$$(1-\varepsilon)\beta\frac{S}{N}+\frac{\sigma\omega}{\sigma+\eta+\mu}-(\omega+\mu-\alpha\rho)\leq 0$$

for the derivative $\dot{\mathscr{L}}\leq 0$. Rewrite $c=\omega+\mu-\alpha\rho$, $d=\sigma+\eta+\mu$, and recall that $S=\frac{\Lambda}{\mu}$ at the DFE, then the condition reduces to

$$(1-\varepsilon)\frac{\beta\Lambda}{\mu N}\leq\omega+\mu-\alpha\rho-\frac{\sigma\omega}{\sigma+\eta+\mu}\leq\omega+\mu-\alpha\rho$$

and consequently,

$$\frac{(1-\varepsilon)\frac{\beta\Lambda}{\mu N}}{\omega+\mu-\alpha\rho}\leq 1\ \Rightarrow\ R_0\leq 1.$$

Finally, taking the Lyapunov function as

$$\mathscr{L} = D + \frac{\sigma}{\sigma+\eta+\mu}(C+H),$$

then the derivative

$$\dot{\mathscr{L}} \leq 0, \text{ if } R_0 \leq 1.$$

However the $\dot{\mathscr{L}} = 0$ only if $D = C = H = 0$. □

Theorem 3.3.5. *The DFE is globally asymptotically stable for*

$$(1-\varepsilon)\beta\frac{S}{N} + \frac{\sigma\omega}{\sigma+\eta+\mu} \leq \omega+\mu-\alpha\rho.$$

Proof. Consider the set

$$\mathscr{W} = \left\{(S,D,C,H) \geq 0 : \dot{\mathscr{L}} = 0\right\}$$

characterized by $C = 0$. Meanwhile $C = 0$ implies that

$$\begin{aligned}\dot{H} &= \frac{\phi C}{1+KC} - \sigma H - \eta H - \mu H = -\sigma H - \eta H - \mu H \text{ since } C = 0\\ &= -(\sigma+\eta+\mu)H\end{aligned}$$

with the solution

$$H = \exp(-(\sigma+\eta+\mu)) \to 0.$$

This shows that $H = 0$ on any invariant set where $\mathscr{L} = 0$. With $C = H = 0$, then

$$\begin{aligned}\dot{D} &= \alpha\rho D + (1-\varepsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D = (\alpha\rho-\omega-\mu)D + (1-\varepsilon)\beta\frac{SD}{N}\\ &= \left(-(\omega+\mu-\alpha\rho) + (1-\varepsilon)\frac{\beta\Lambda}{\mu N}\right)D = \left(-1 + \frac{(1-\varepsilon)\frac{\beta\Lambda}{\mu N}}{\omega+\mu-\alpha\rho}\right)(\omega+\mu-\alpha\rho)D\\ &= (-1+R_0)(\omega+\mu-\alpha\rho)D.\end{aligned}$$

Having that $R_0 \leq 1 \Rightarrow -1 + R_0 \leq 0$ and $\omega + \mu - \alpha\rho \geq 0$, then $\dot{D} \leq 0$ if $D > 0$, leading to a contradiction and the only invariant solution inside $\mathscr{W}$ is $C = H = D = 0$. By LaSalle's invariance principle, every solution starting in the feasible region approaches the largest invariant set in $\mathscr{W}$ and so the DFE is globally asymptotically stable for $R_0 < 1$. □

CHAPTER FOUR

DISCUSSION

4.1 Introduction

Having established the positivity and stability of the solutions of the governing model, the equations are solved using the Runge-Kutta scheme of the fourth order. The choice of the fourth order Runge-Kutta Scheme is due to its stability and large region of convergence. Absolute error tolerance is set to 10^{-8} and the numerical solutions obtained are plotted on graphs to evaluate the trends as the parameter values are varied. Default values chosen for the parameters are

$$\Lambda = 3.3; \alpha = 0.1; \rho = 0.2; \varepsilon = 0.41; \mu = 1/65; \beta = 0.2; \sigma = 0.1;$$
$$\omega = 0.1; \gamma = 0.1; \delta = 0.3; \eta = 0.08; \phi = 0.15; K = 100;$$

4.2 Model I

People with diabetes and those who are prone to their effects may transfer their lifestyles to the other as a result of their relationship. The parameter β reflects the portion of such interaction that affects lifestyle and, as a result, results in diabetes infection. The results of an increasing percentage of lifestyle-influence on the trend of diabetes in the population are shown in the figures (4.3 - 4.4). Rapid migration from the susceptible class to the diabetic class is enhanced by an increase in the proportion of diabetic/susceptible interactions that resulted in infection. As a result, the diabetes class briefly experiences a surge before the class begins to decline once more. Figure (4.1) demonstrates that the diabetic class has a surge for the first year before the class begins to decline again after that year. The quick migration into the diabetic class has caused a drop in the susceptible class, resulting in a decline in the

susceptible class. Thus, a smaller percentage will migrate into the diabetes population as a result of the less vulnerable susceptible population. The Complicated and Hospitalized classes exhibit the similar trend, as seen in figures (4.2) and (4.3). Since there is no treatment for diabetes, those who leave the susceptible class cannot return. Due to the growing productive lifestyle-impact between the diabetic and susceptible classes, the susceptible class continues to decline. The susceptible class decreases with time as β grows, as seen in Figure (4.4).

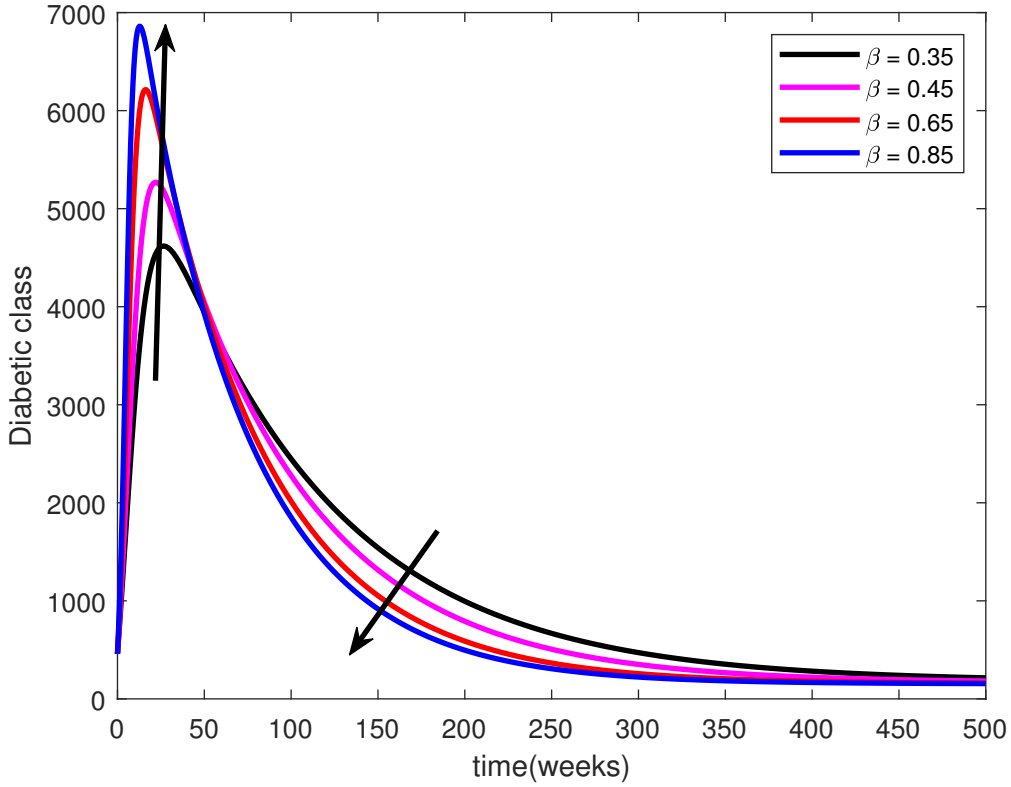

Figure 4.1: variation of the Diabetic cases with Diabetic-Susceptible interactions

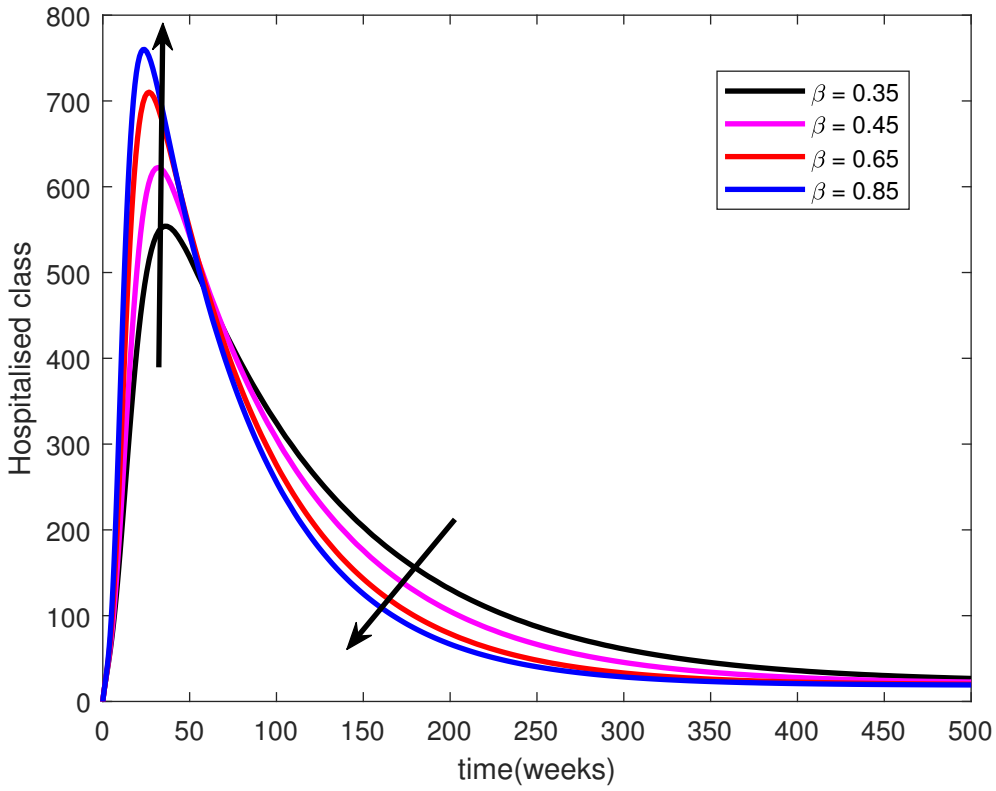

Figure 4.2: variation of the Hospitalised cases with Diabetic-Susceptible interactions

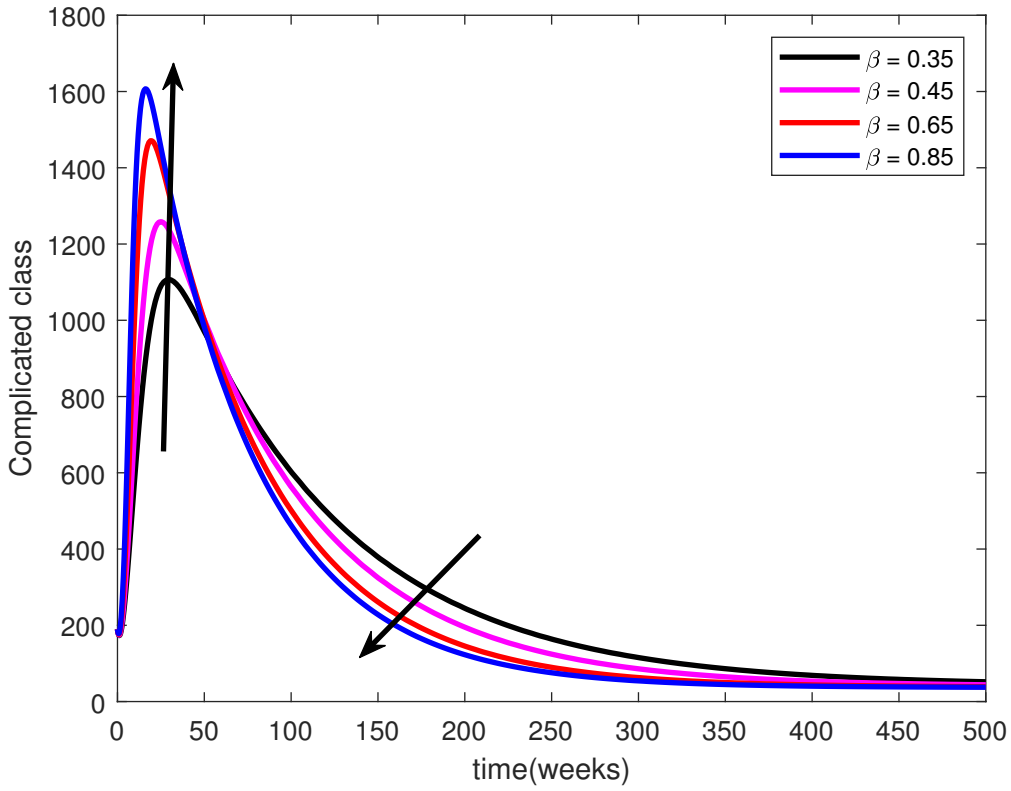

Figure 4.3: variation of the Complicated cases with Diabetic-Susceptible interactions

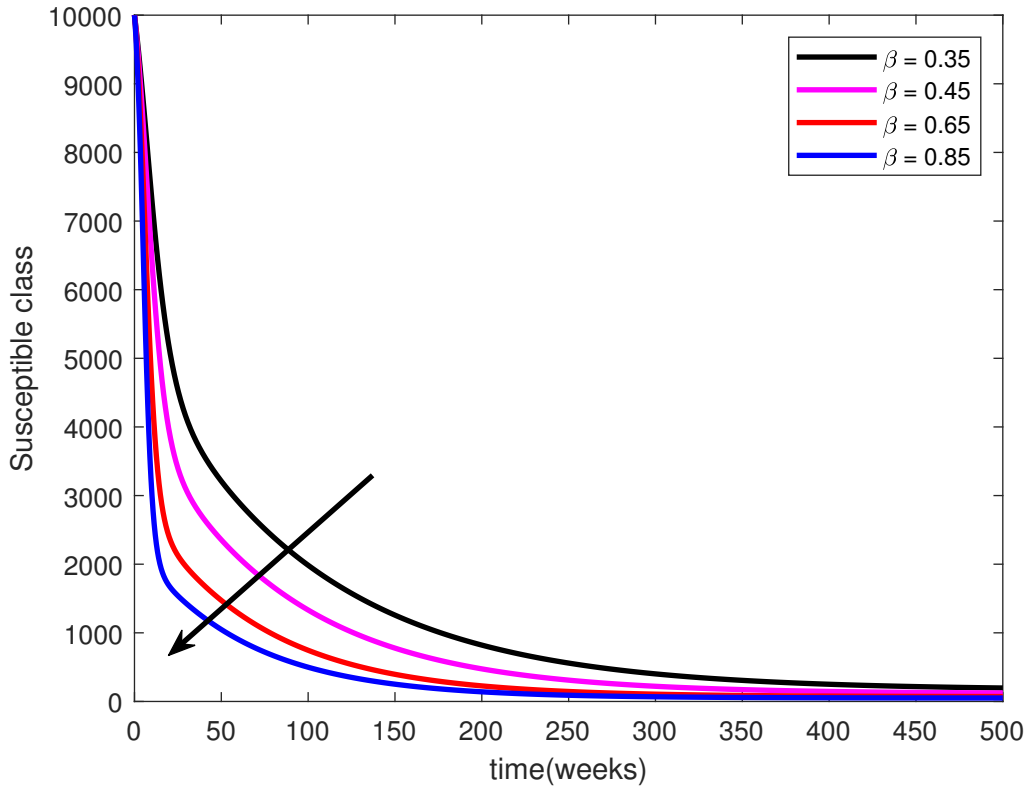

Figure 4.4: variation of the Susceptible class with Diabetic-Susceptible interactions

The lifestyle incidence rate parameter, ε, assumes values between 0 and 1, where 0 denotes the lowest lifestyle standard and 1 denotes the greatest lifestyle standard. The quality of living is raised by increasing ε. Practically speaking, raising lifestyle standards include reducing excess weight, becoming physically active, eating healthy nutritious foods, and drinking the recommended amount of water. In this study, a person approaches the maximum living standard as the value of ε goes from 0 to 1. As the overall quality of life rises, the susceptible class also rises (see figure (4.5)). The migration into the diabetic class decreases when the majority of people in the susceptible class raise their standard of living. Figure (4.6) illustrates how the diabetic class gradually decreases until a point at which the susceptible class reaches saturation and people begin to migrate into the diabetic class. Similar to the diabetic class, the hospitalized and complicated classes likewise follow this pattern.

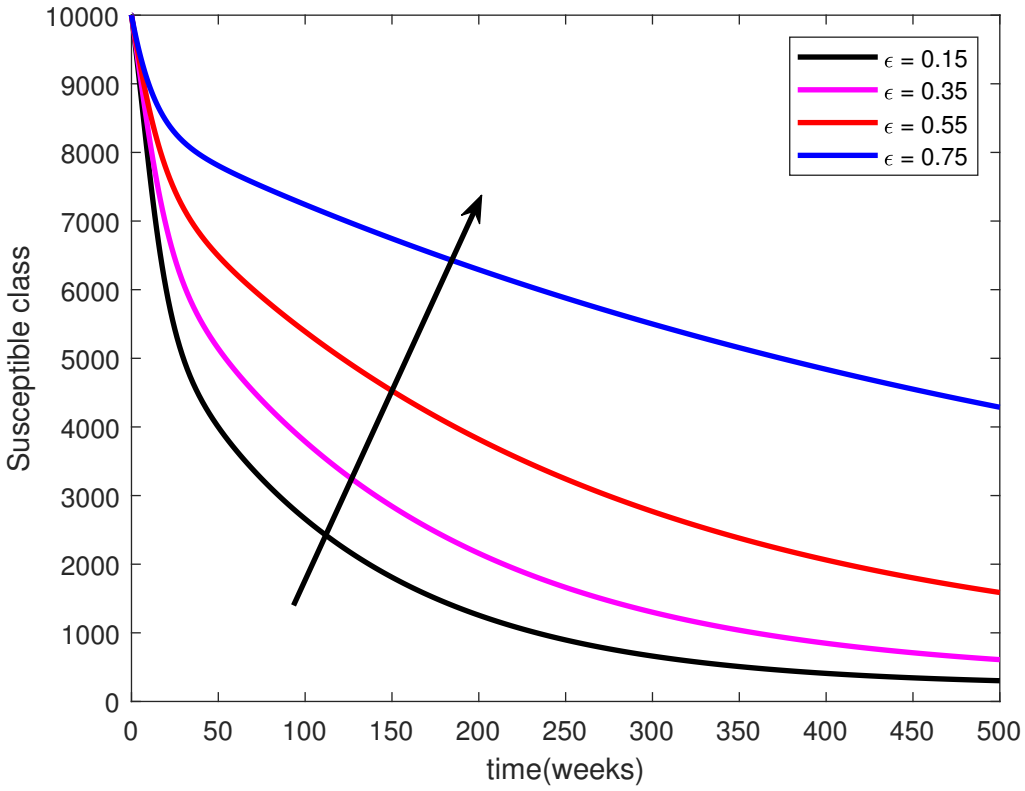

Figure 4.5: variation of the Susceptible class with lifestyle impact

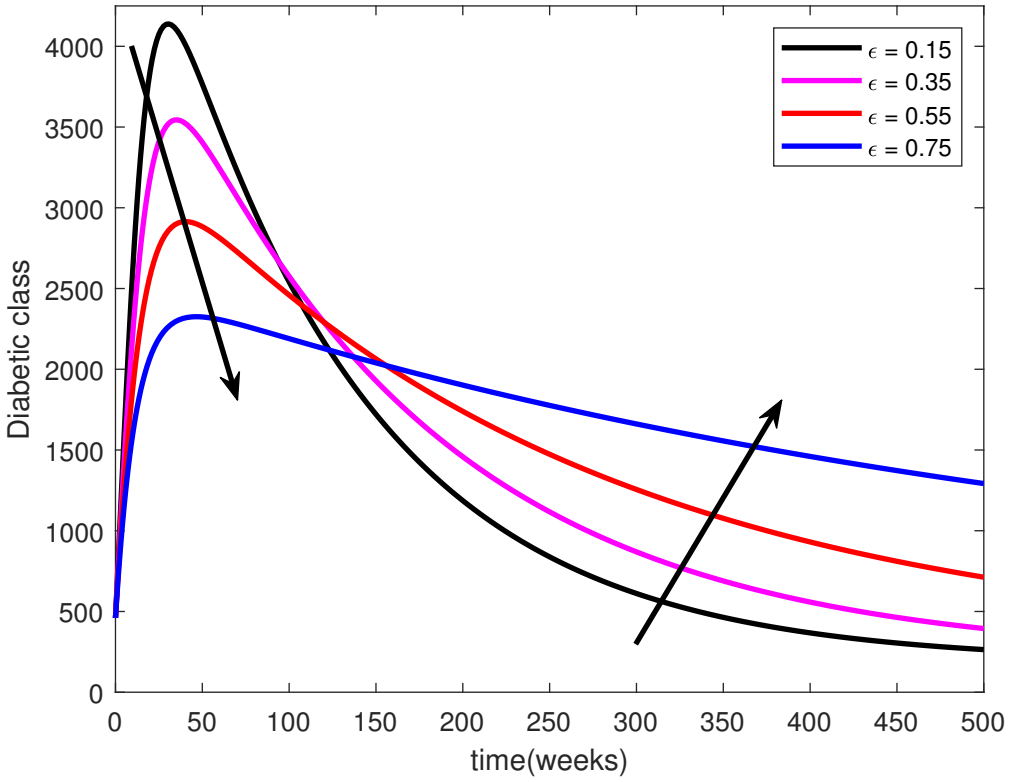

Figure 4.6: variation of the Diabetic class with lifestyle impact

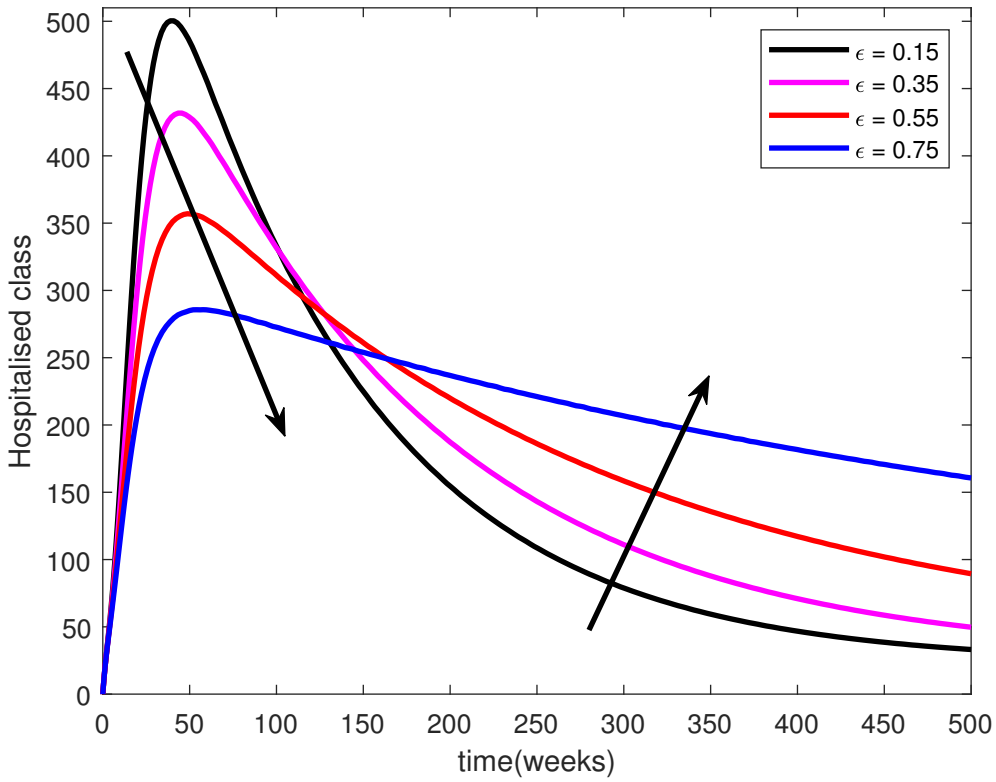

Figure 4.7: variation of the Hospitalised class with lifestyle impact

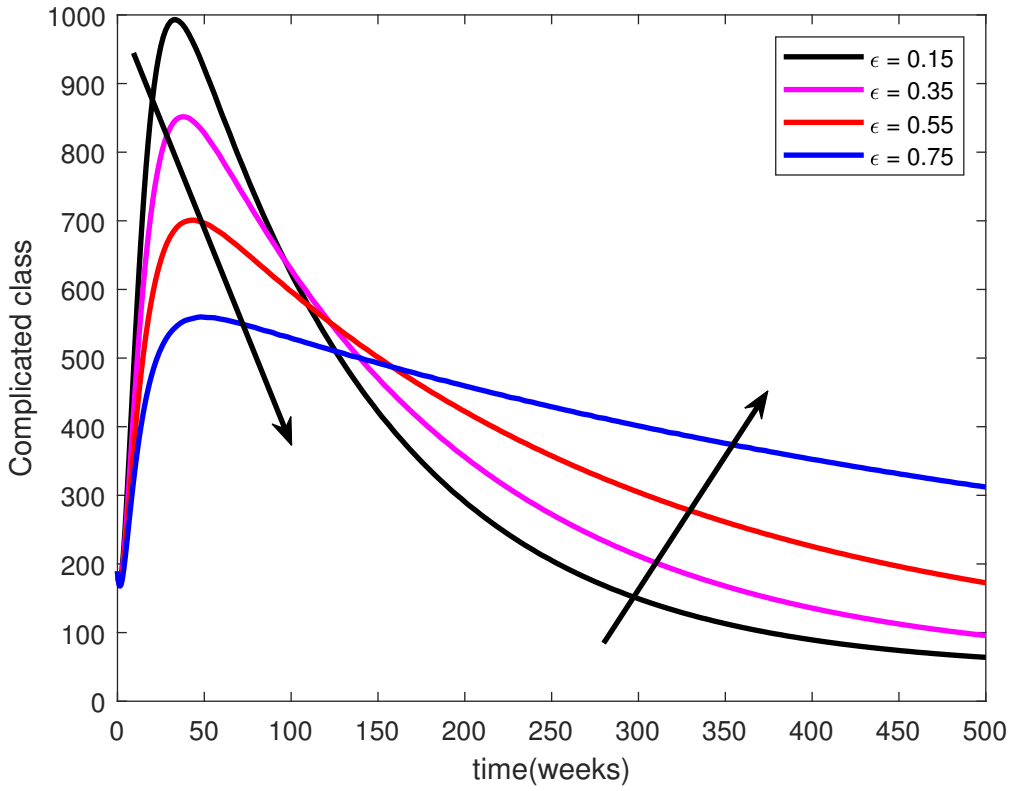

Figure 4.8: variation of the Complicated cases with lifestyle impact

There is currently no known treatment for diabetes. However, diabetes can be controlled, and those who have it can also have normal lives. Additionally, diabetic people with complications can go back to the diabetic class and have normal lives. The parameter σ is used to illustrate the rate at which diabetic people who have complications recover and return to the diabetic class. Owing to migration from the complicated class, as seen in figure (4.9), the diabetic class rises, while hospitalized cases fall (see figure (4.10).

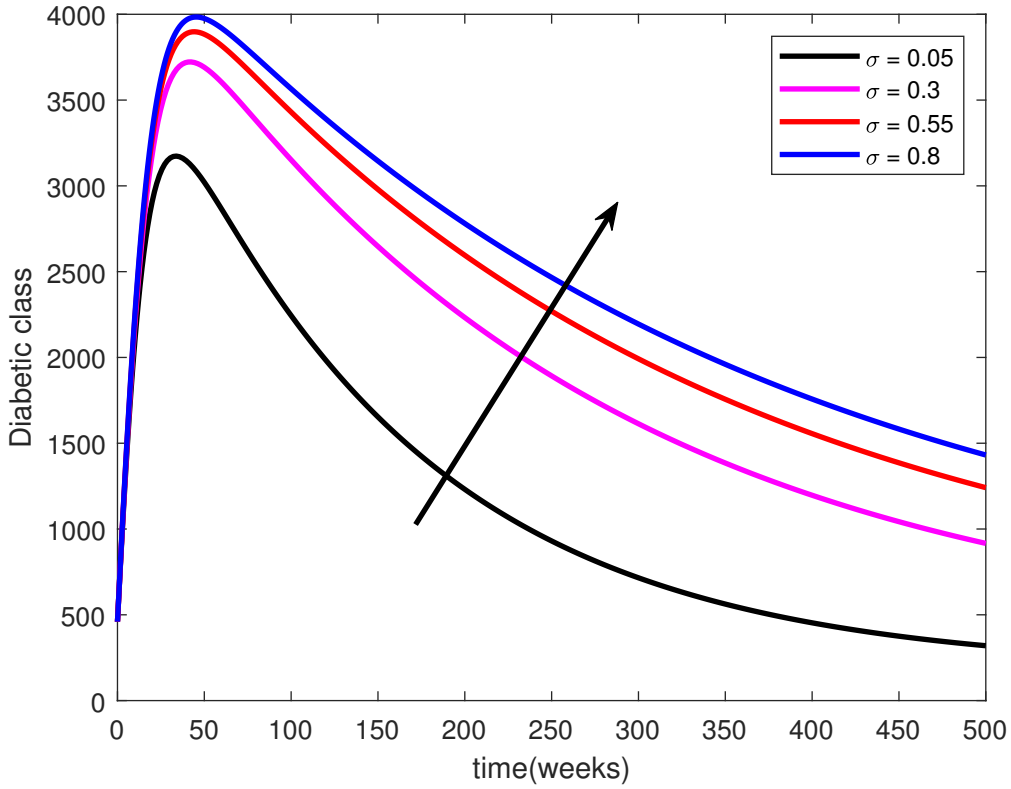

Figure 4.9: variation of the Diabetic class with recovery rate

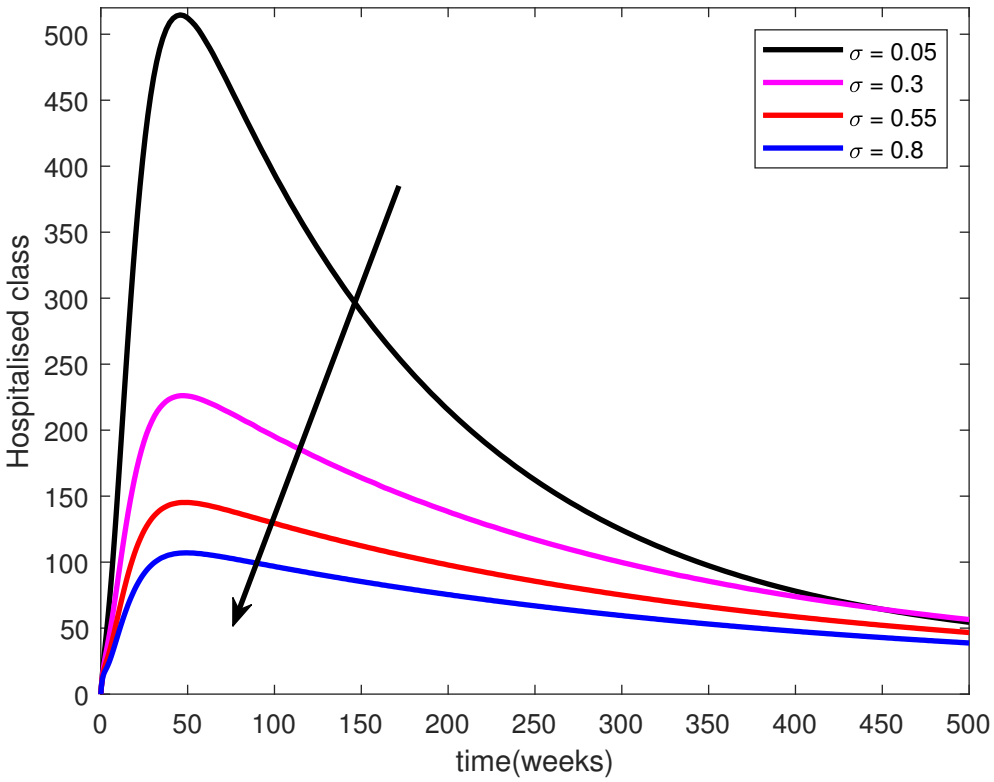

Figure 4.10: variation of the Hospitalised class with recovery rate

The hospitalisation rate γ indicates the rate at which individuals migrate from the Complicated class to the Hospitalised compartment. This model assumes a constant hospitalisation rate. Constant hospitalisation rate indicates that the same proportion of the complicated diabetic cases receive treatment at any point in time. At first, the as the constant hospitalisation increases, the Complicated population goes down. As time progresses, the diabetic class continues to rise (as shown in figure (4.12)) which eventually leads to change in trend for the Complicated class. The complicated class starts rising after a certain time due to constant hospitalisation rate (figure (4.11)). Meanwhile, figure (4.13) illustrates the rise in the hospitalised class as the constant hospitalisation rate increases. If the constant hospitalisation rate continue to increase, then the hospitals will become congested and overloaded. Hence, constant hospitalisation rate is not a good way to control the complications in the diabetic patients.

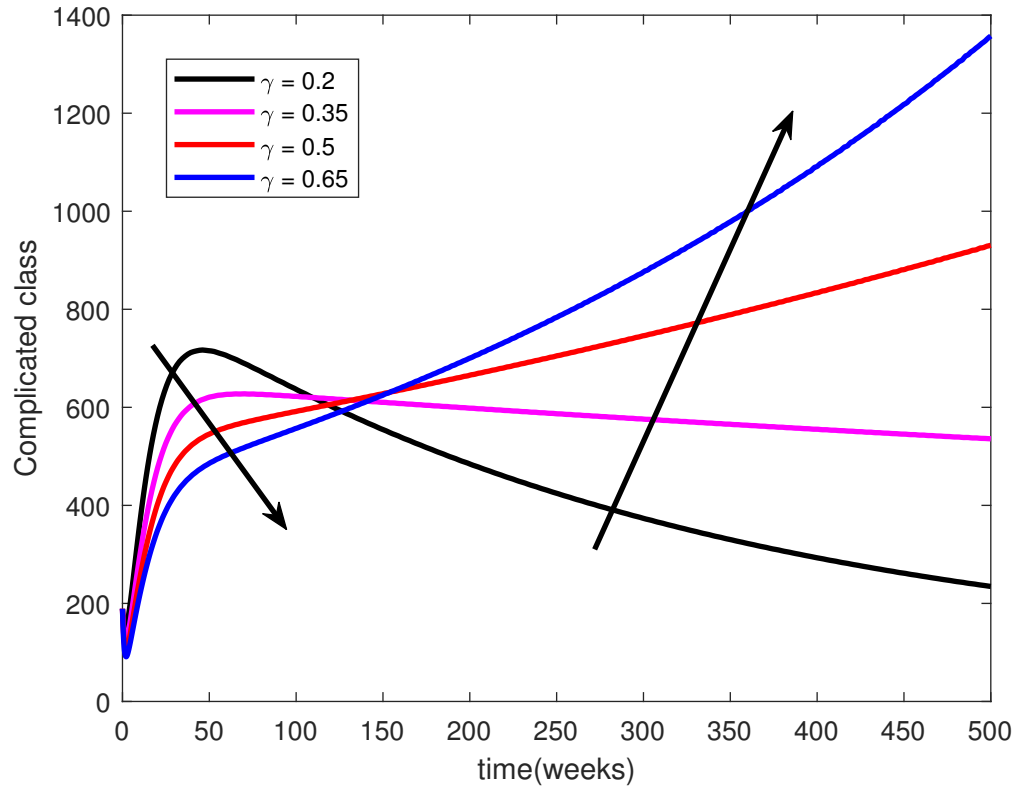

Figure 4.11: variation of the Complicated cases with Hospitalisation rate

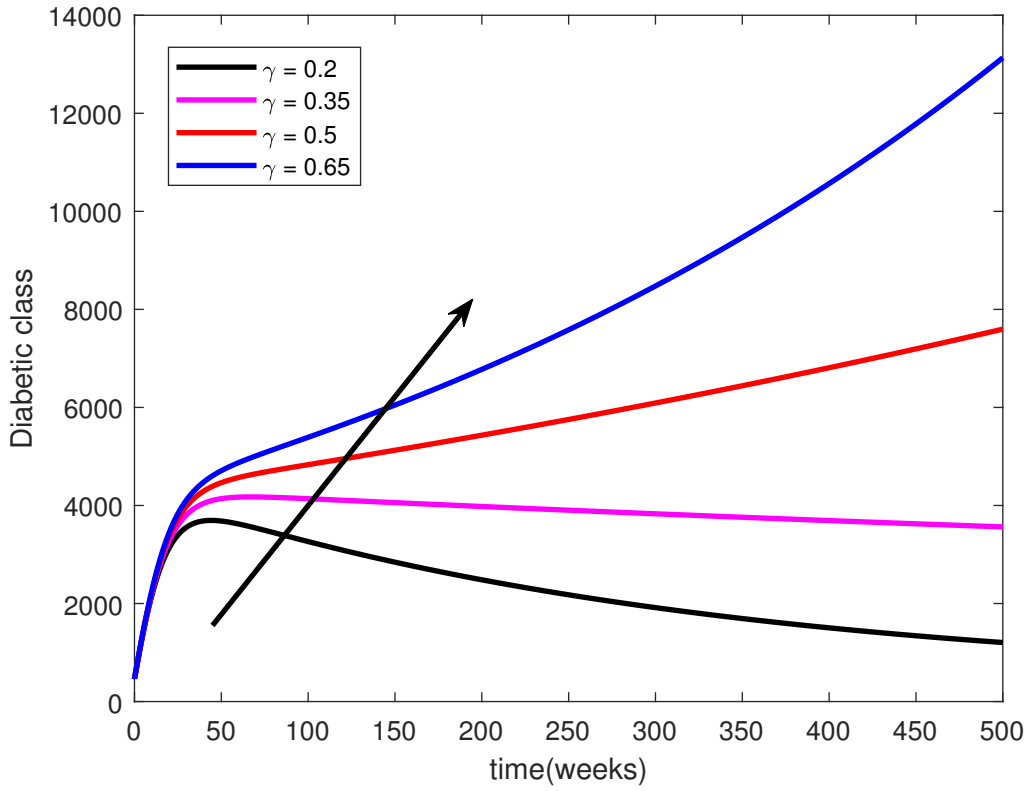

Figure 4.12: variation of the Diabetic class with Hospitalisation rate

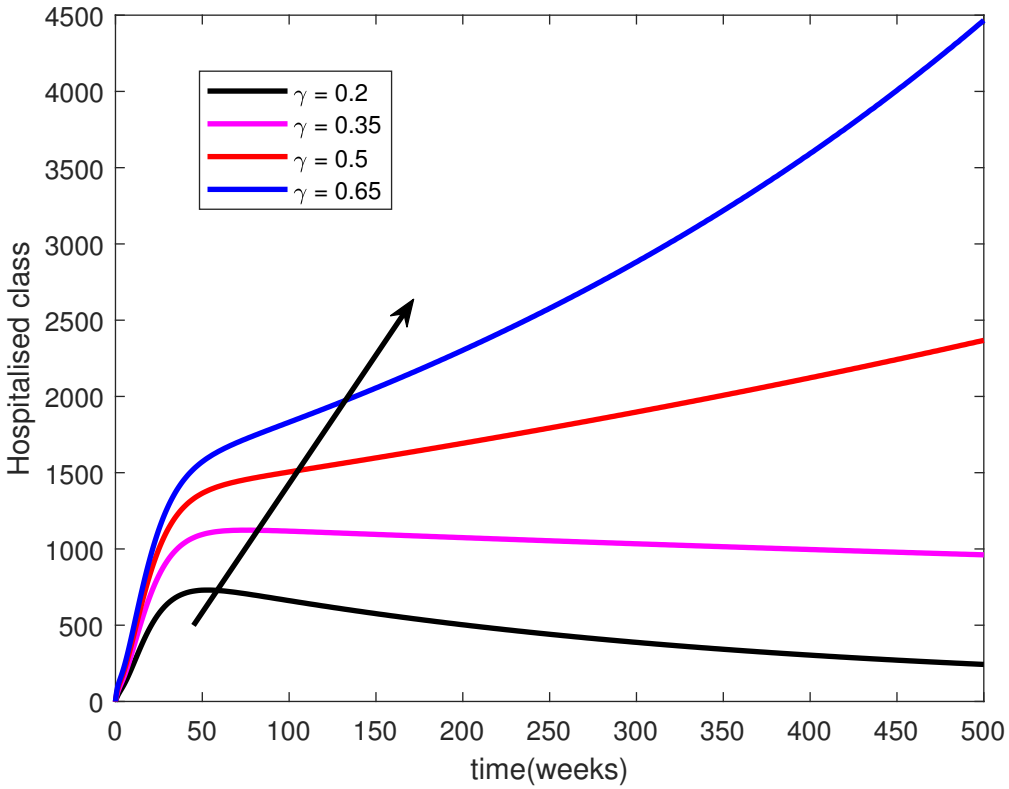

Figure 4.13: variation of the Hospitalised class with Hospitalisation rate

4.3 Model II

Based on the results highlighted above, the use of constant hospitalisation may seem very effective at the beginning but it will eventually lead to overburdening of the health facilities. It is important to put the carrying capacity of the hospitals into consideration. The second model proposed uses the saturation treatment function

$$f(C) = \frac{\phi C}{1 + KC}.$$

By setting the carrying capacity K to zero, the treatment function becomes a constant rate ϕC. In this case, model II coincides with model I. Without loss of generalisation, model II is a generalisation of model I. Equations (3.3.1 - 3.3.4) are solved numerically

using the default values

$$\Lambda = 3.3; \alpha = 0.1; \rho = 0.2; \varepsilon = 0.41; \mu = 1/65; \beta = 0.2;$$
$$\sigma = 0.1; \omega = 0.1; \delta = 0.3; \eta = 0.08; \phi = 0.15; K = 100.$$

The outcomes of the numerical simulation are discussed below.

The quality of lifestyle lived by the individuals in the susceptible population is labelled as ε; which takes on values between 0 (the lowest quality of lifestyles) and 1 (the highest quality of lifestyle). Hence, by increasing the value of ε, we are increasing the quality of lifestyle of the susceptible class. Increasing the quality of lifestyle implies that the susceptible population are able to maintain quality healthy lifestyle which are void of influence by the diabetic patients. The susceptible class therefore continue to rise as the quality of lifestyle increases. Figure (4.14) shows the rise in the susceptible class as the quality of lifestyle among the susceptible class increases. Figure (4.15) shows that the diabetic class rises initially but later decreases with time. However, increasing the quality of lifestyle in the susceptible class controls the initial surge in the diabetic class. This shows that increasing lifestyle among those who are not diabetic can control the population of individuals who become diabetic yearly. Similar results is experienced in the complicated class, figure (4.16) shows that the increased lifestyle controls the initial surge in the diabetic population.

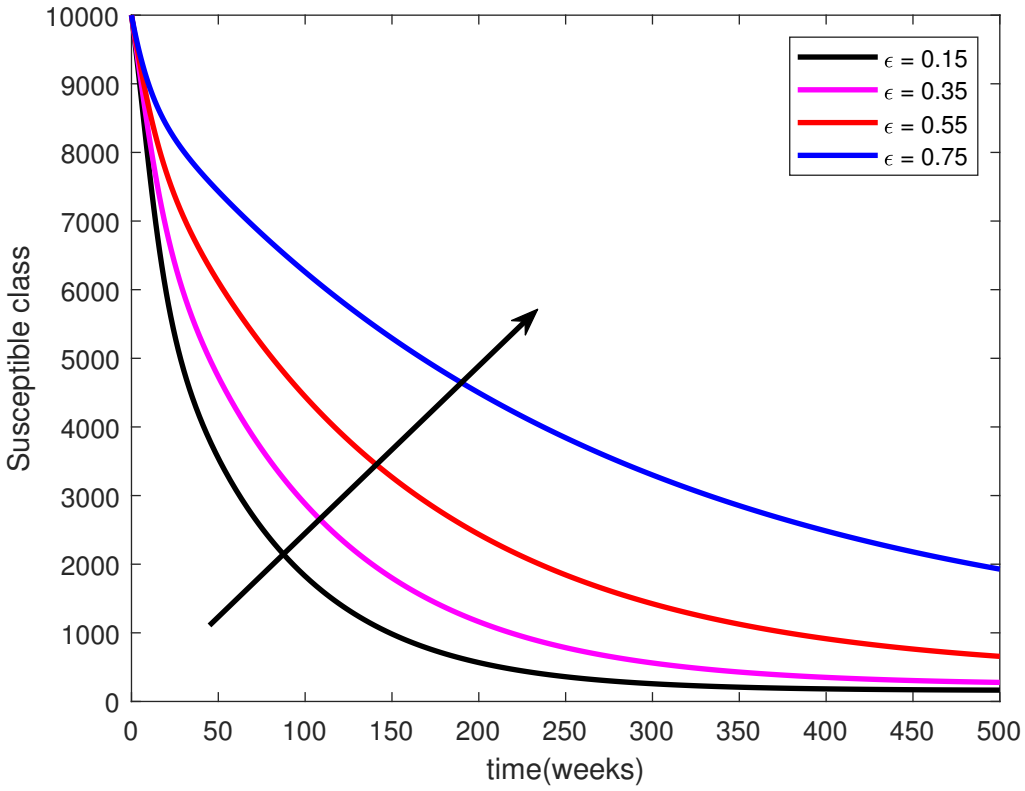

Figure 4.14: variation of the Susceptible class with lifestyle impact

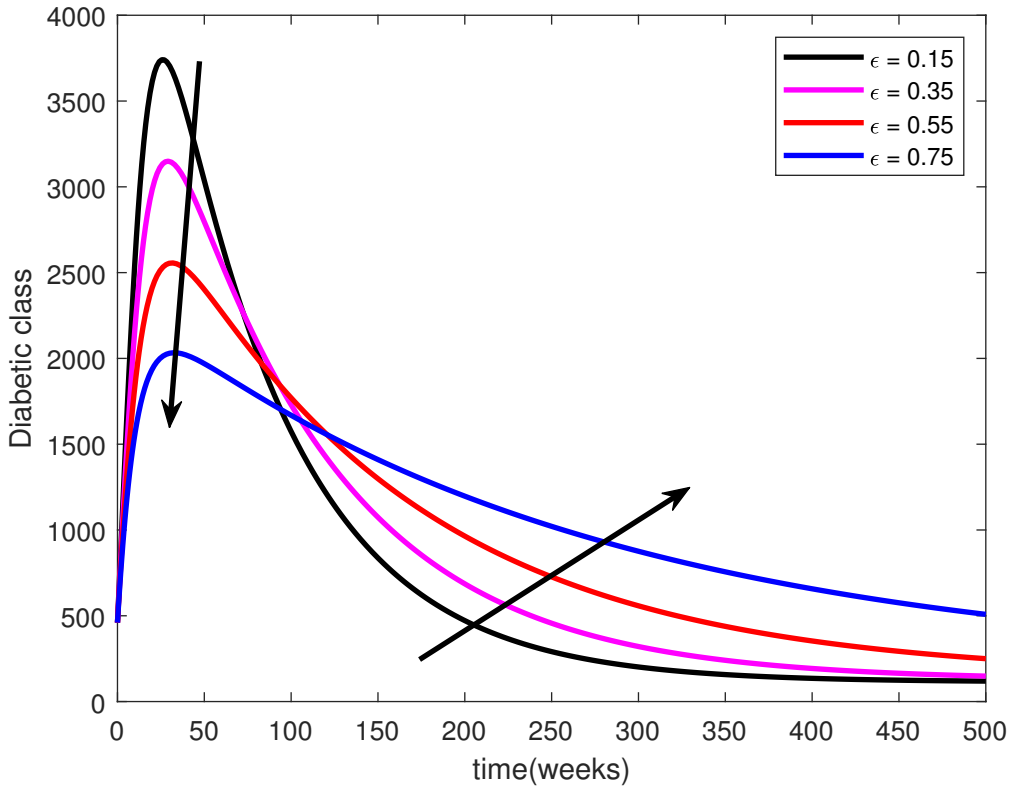

Figure 4.15: variation of the Diabetic class with lifestyle impact

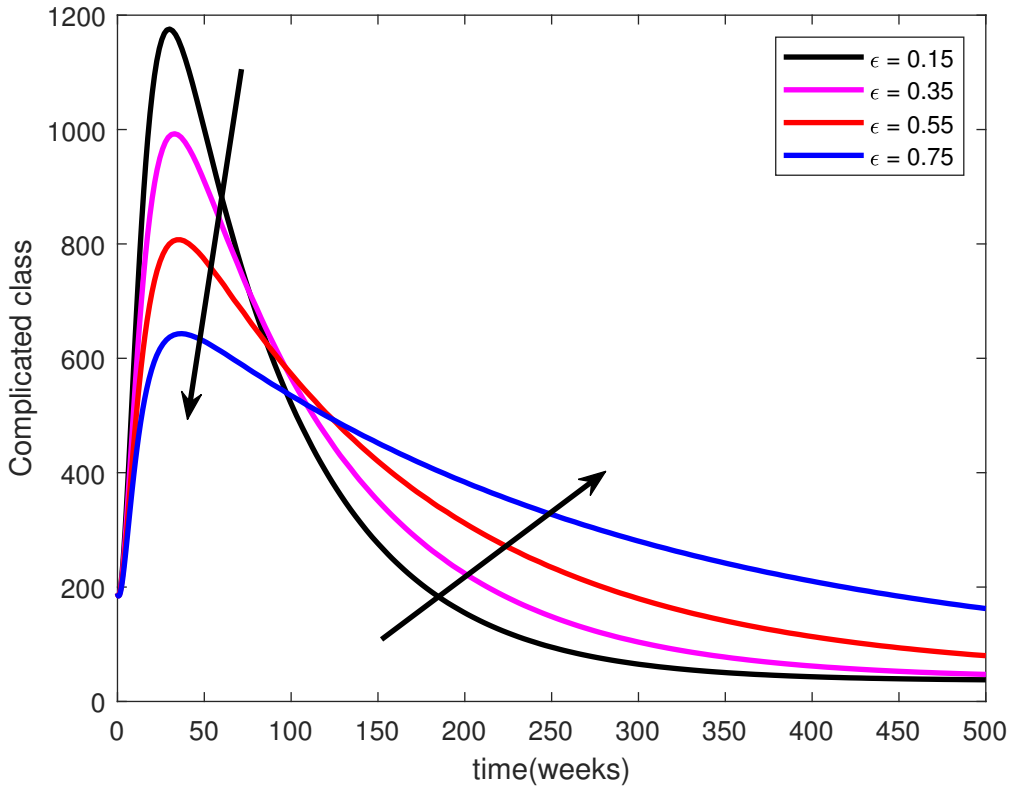

Figure 4.16: variation of the Complicated cases with lifestyle impact

Although scientists are still unable to find a cure for diabetes based on the current research, an individual who has become diabetic can still live normal life if proper medications administered and a healthy lifestyle is ensured. The parameter σ is set to mean the rate at which complicated individual are cured of their complications. Increasing σ is tantamount to increasing the the rate of cure for complications. Figures (4.17 - 4.19) reveal that such cures have no significant effects on the complicated, susceptible and diabetic classes. Increasing the rate of recovery from complications has a mild decreasing effects on the complicated class and a mild increasing effects on susceptible and diabetic classes. The effect of increasing recovery rate is only very significant in the hospitalised class where the population of hospitalised individuals reduce as recovery rate increases (figure (4.20)). It is therefore worth concluding that increasing the rate of recovery can reduce the congestion in the health facilities.

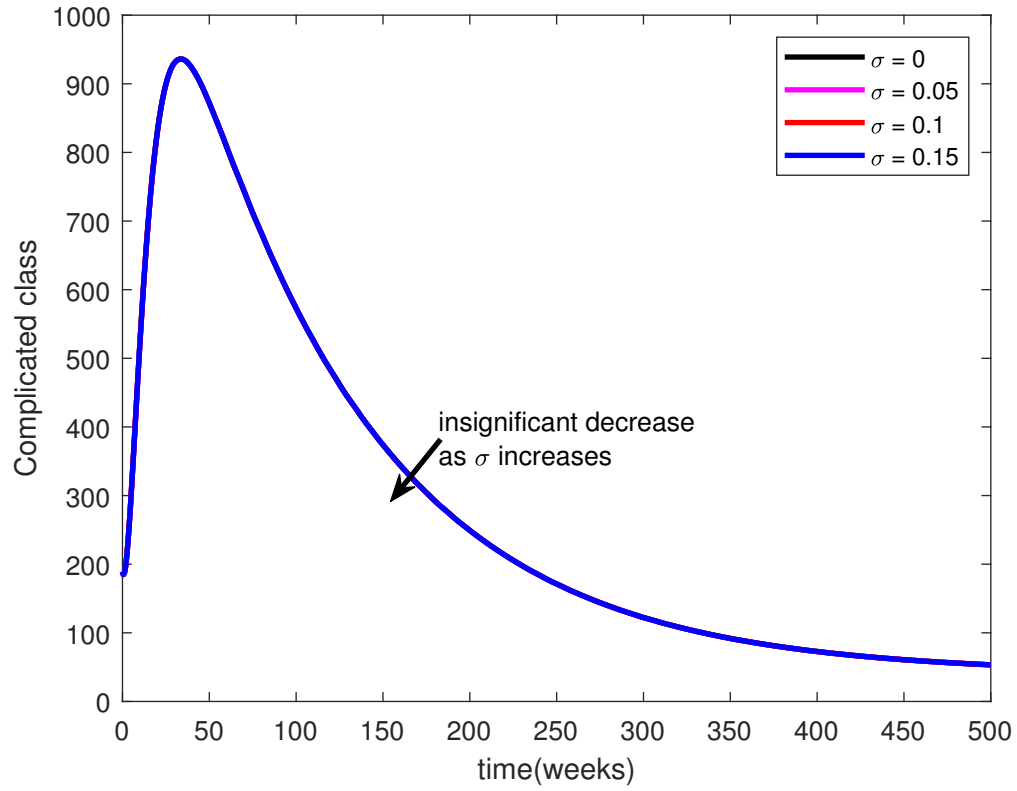

Figure 4.17: variation of the Complicated class with recovery rate

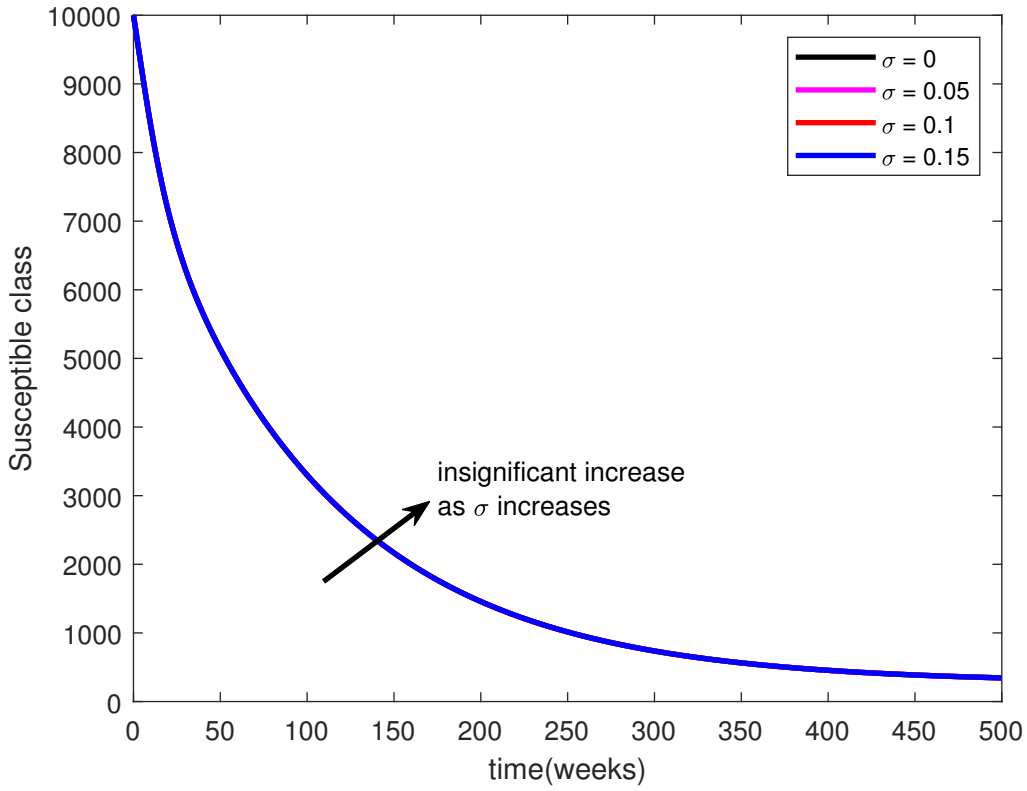

Figure 4.18: variation of the Susceptible class with recovery rate

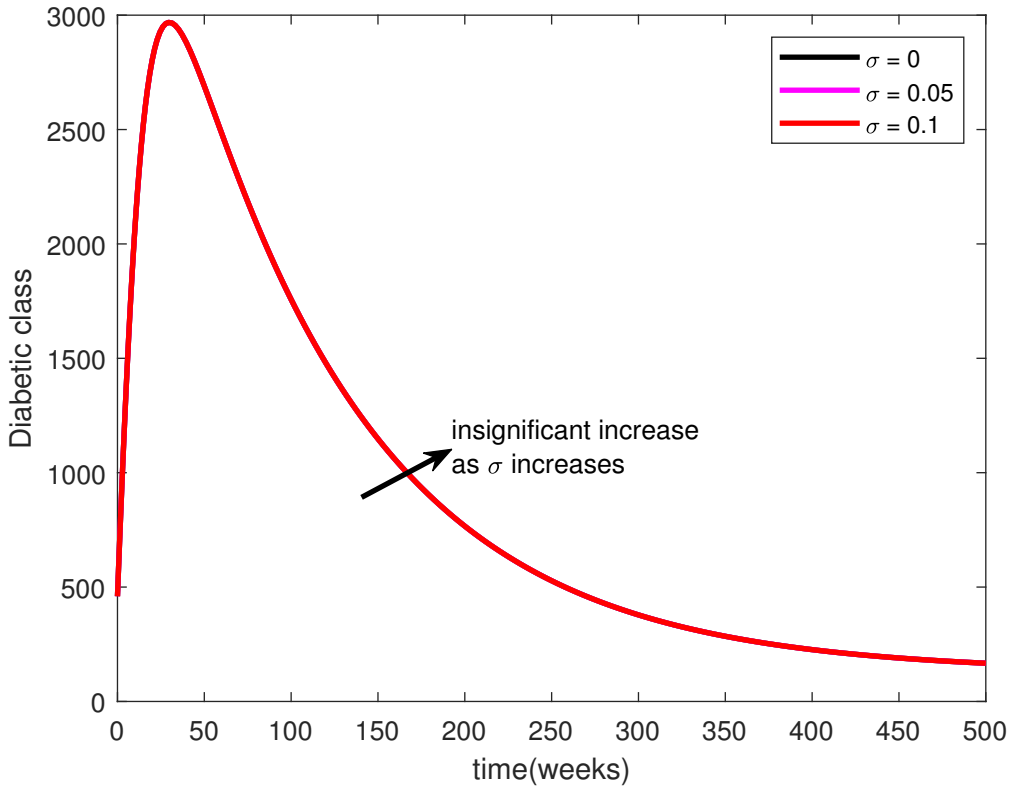

Figure 4.19: variation of the Diabetic class with recovery rate

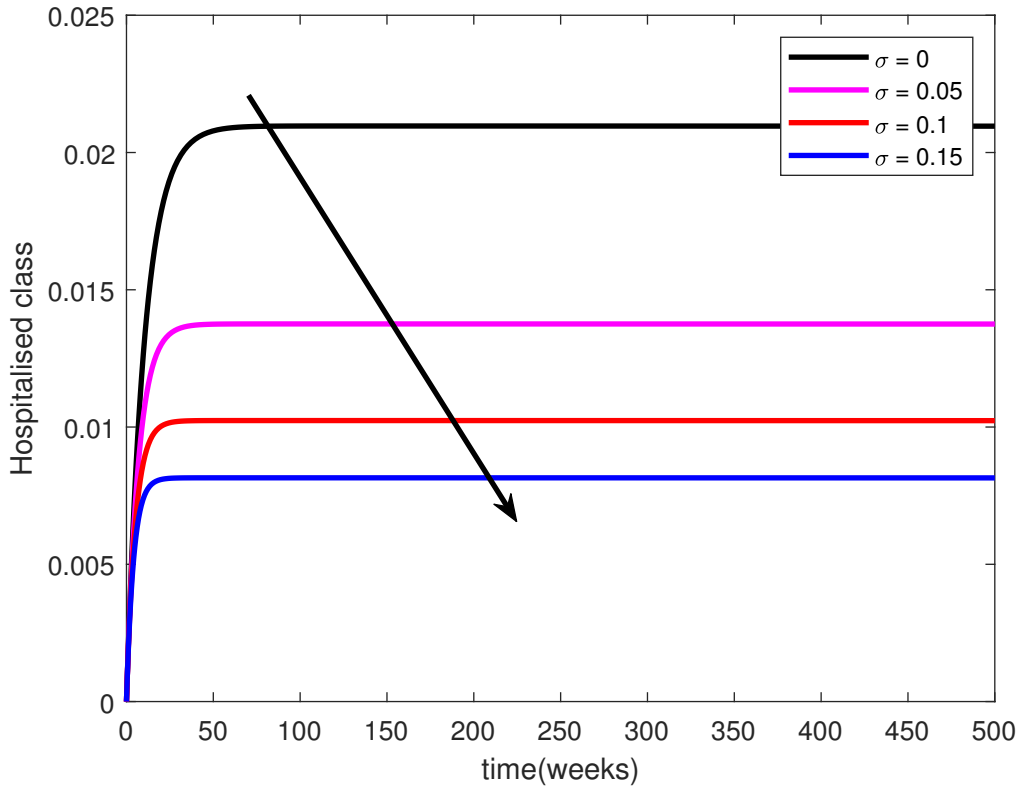

Figure 4.20: variation of the Hospitalised class with recovery rate

The per capita hospitalisation rate ϕ represents the proportion of the diabetic class that go for treatment per month. Increasing the per capita hospitalisation rate ϕ translates to an increase in the number of individuals who get into the Hospitalised class. Hence, figure (4.21) shows that the population in the hospitalised class grows as the per capita hospitalisation rate increases. The carrying capacity K represents the maximum capacity of the hospitals. It could represent the maximum number of bed space or the maximum number of diabetic patients that can access the medical practitioners for care and treatment. More population from the complicated class gets access to care and treatment as the carrying capacity increases. This enables the population of the hospitalised individuals to reduce as most of the complicated cases receive care and treatment and migrate back into the diabetic class and thereby reducing the congestion at the hospitals. Figure (4.22) shows that increasing carrying capacity leads to a reduction in the hospitalised class. Hence, an increase in the carrying capacity of the hospitals is able to control the number of hospitalised individuals.

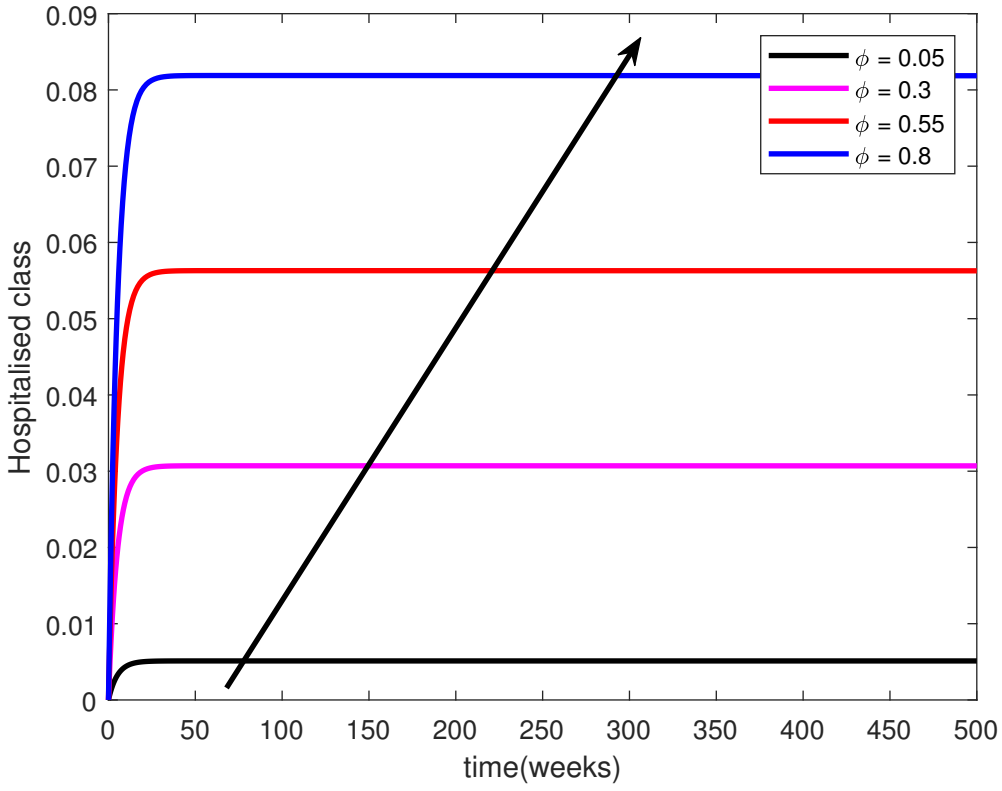

Figure 4.21: variation of the Hospitalised class with per capita hospitalisation rate

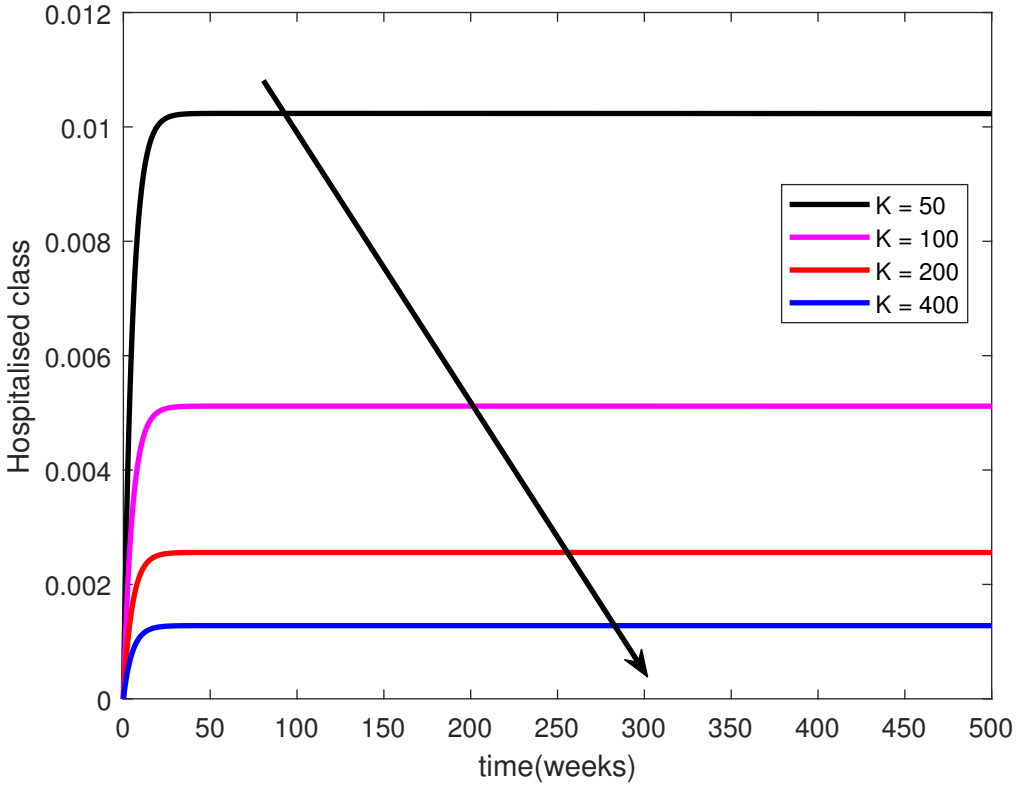

Figure 4.22: variation of the Hospitalised cases with hospital carrying capacity

CHAPTER FIVE

CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

The response of diabetic population is studied in this research, putting the complicated diabetic patients and available resources into consideration. Two mathematical models are developed to study the trend. The first considers a constant hospitalisation rate while the second considers a hospitalisation rate that depends carrying capacity and the per capita hospitalisation rate. The models are solved numerically in MATLAB using the explicit Runge-Kutta (4,5) method and the outcomes reported graphically.

In the case of constant hospitalisation rate, the following outcomes were obtained;

1. Increasing the proportion of susceptible class that are prone to lifestyle infection will lead to

    - a surge in the Diabetic class, Complicated class and Hospitalized class for the first year before they begin to decline again.

    - a decrease in the susceptible class.

2. Increasing the the quality of lifestyle in the susceptible class will lead to an increase in the susceptible class and a decrease in the diabetic cases.

3. Increasing the rate at which an hospitalised individual returns to the the diabetic class will lead to a rise in the diabetic class rises and a decline in the hospitalized class.

4. Increasing the constant hospitalisation rate will lead to

    - the Complicated population initially reduces but begins to rise with time.

- a rise in the hospitalised class.

In the second, the saturation treatment function

$$f(C) = \frac{\phi C}{1+KC}.$$

is used. The model coincides with the model for constant hospitalisation rate when $K = 0$ and thus, the second model generalises model I. The outcomes of the numerical simulation are summarised below;

1. When the quality of lifestyle is increased, the following are observed

   - The susceptible class increases.
   - The diabetic and the complicated classes rise initially but later decrease with time.

2. Increasing the rate of recovery from complication leads to the following outcomes

   (a) there are no significant effects on the complicated, susceptible and diabetic classes.

   (b) the population of the hospitalised individuals reduces.

3. Increasing the proportion of the diabetic class that go for treatment per month leads to an increase in the number of individuals who get into the Hospitalised class.

4. Increasing the carrying capacity leads to a reduction in the hospitalised class.

5.2 Recommendations

Based on the outcomes of this research, the following recommendations are provided;

1. Interaction between the diabetic patients and the healthy individuals should be reduced to reduce the population of the diabetic patients.

2. Enhancing and increasing the quality of lives of the the vulnerable groups can reduce the population of individuals who get into the diabetic class.

3. Retaining constant hospitalisation rate is not a good way to control the complications in the diabetic patients.

4. Increasing the rate of recovery can reduce the congestion in the health facilities.

5. An increase in the carrying capacity of the hospitals is able to control the number of hospitalised individuals.

6. future research should consider including machine learning for decision-making.

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APPENDICES

Appendix I: MATLAB codes for Model I

```
function dx=First_Equations(t,x)
global LA alpha rho eps mu beta sigma omega gamma delta eta
        N = sum(x);
        dx=[LA+alpha*(1-rho)*x(2)-(1-eps)...
                *beta*x(1)*x(2)/N-mu*x(1);
                alpha*rho*x(1)+(1-eps)*beta*x(1)*x(2)/N+...
                sigma*x(4)-(omega+mu)*x(2);
                omega*x(2)-gamma*x(3)-(delta+mu)*x(3);
                gamma*x(3) - (sigma + eta + mu)*x(4)];
        end

clc
clear all
global LA alpha rho eps mu beta sigma omega gamma delta eta
LA=3.3; alpha=0.1; rho=0.2; eps=0.41; mu=1/65; beta=0.2;
sigma=0.1; omega=0.1; gamma=0.1; delta=0.3; eta=0.08;
tspan = [0 10]; i = 1; xzero = [10000 460 189 0];
Legend_text={'\gamma = 0.25','\gamma = 0.40','\gamma = 0.55',...
        '\gamma = 0.70'}
for gamma = [0.2, 0.35,0.50, 0.65]
        txt_Name = 'case1_gamma';
        if i==1
                txt='k-';
        elseif i==2
                txt='m-';
        elseif i==3
                txt='r-';
        elseif i==4
```

```
        txt='b-';
elseif i==5
        txt='g-';
end
[t,x] = ode45(@First_Equations,tspan,xzero);
if i~=4
        figure(1), plot(t,x(:,1), txt, 'LineWidth',3)
        hold on
        figure(2), plot(t,x(:,2), txt, 'LineWidth',3)
        hold on
        figure(3), plot(t,x(:,3), txt, 'LineWidth',3)
        hold on
        figure(4), plot(t,x(:,4), txt, 'LineWidth',3)
        hold on
        i=i+1;
else
        figure(1), plot(t,x(:,1), txt, 'LineWidth',3)
        xlabel('time(days)'),ylabel('Susceptible class')
        annotation('arrow',[0.2,0.4], [0.2, 0.4],'LineWidth',3);
        legend(Legend_text)
        txt_Save = strcat(txt_Name,'_Susceptible');
        saveas(gcf,txt_Save)
        figure(2), plot(t,x(:,2), txt, 'LineWidth',3)
        xlabel('time(days)'),ylabel('Diabetic class')
        annotation('arrow',[0.2,0.4], [0.2, 0.4],...
                                'LineWidth',3);
        legend(Legend_text)
        txt_Save = strcat(txt_Name,'_Diabetic');
        saveas(gcf,txt_Save)
        figure(3), plot(t,x(:,3), txt, 'LineWidth',3)
```

```
                xlabel('time(days)'),ylabel('Complicated class')
                annotation('arrow',[0.2,0.4], [0.2, 0.4],...
                                        'LineWidth',3);
                legend(Legend_text)
                txt_Save=strcat(txt_Name,'_Complicated');
                saveas(gcf,txt_Save)
                figure(4), plot(t,x(:,4), txt, 'LineWidth',3)
                xlabel('time(days)'),ylabel('Hospitalised class')
                annotation('arrow',[0.2,0.4], [0.2, 0.4],...
                        'LineWidth',3);
                legend(Legend_text)
                txt_Save = strcat(txt_Name,'_Hospitalised');
                saveas(gcf,txt_Save)
                i=i+1;
        end
end
```