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Impact of Fixed Allocation of Health Resources on Diabetes in Kenya: Mathematical Modelling Approach

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Abstract: Diabetes is a human disease that can lead to blindness, strokes, and amputations of people's limbs. The effects of diabetes are not limited to the sickness it causes in the human body, it also has a major influence on the worldwide economy, as evidenced by the fact that over 500 billion USD is spent each year on the diagnosis, care, and treatment of diabetes. Diabetes is gradually becoming a menace in Kenya, considering that the number of deaths from diabetes and diabetes-related illnesses have increased in the recent time. With the rapid increase in the reported diabetic cases, it is only a matter of time before the Healthcare facilities and resources become overburdened. This study investigates the effect of a fixed number of available health resources on the progression of diabetes. To represent the dynamics of diabetes with a constant hospitalization rate, a system of ordinary differential equations is formulated. The model is established to be well-posed, positive, and bounded, and the local stability of the equilibrium points is established. The reproduction number is calculated using the next generation matrix. The model is numerically solved and the results are graphed using the explicit Runge-Kutta (4,5)-th order. Improvements in the susceptible class's lifestyle quality diminish migration from the susceptible subpopulation to the diabetic population.

Keywords: Diabetes, Hospitalisation, Mathematical Modelling, Per Capita Hospitalisation Rate

1. Introduction

Chronic illnesses emerge gradually from a combination of genetic, environmental, and behavioural variables. Diabetes, cancer, chronic respiratory disorders, and cardiovascular difficulties are the four groups of noncommunicable diseases (NCDs). More than 41 million people die from NCDs each year, with low- and middle-income countries recording 85 percent of these deaths [1]. Food consumed by humans is turned into glucose, which is then delivered into the bloodstream. When the circulation glucose level rises, the pancreas receives a signal to secrete insulin. Insulin turns glucose into useable energy that other cells can use. Diabetes develops when the insulin-glucose-glycogen regulation system fails. The pancreas either generates too little insulin, which is inadequate to transform the body's sugar, or produces too much unwanted insulin [2, 3].

Diabetes is the leading cause of kidney illness, cardiovascular disease, blindness, and lower limb amputation [4, 5]. Diabetes presently affects 422 million people globally, up from 180 million in 1980.

In 2014, low-income countries had a prevalence of 7.4%, which was higher than the prevalence of 7.0% in high-income countries. Diabetes prevalence has risen faster in low- and middle-income countries, and it is now highest in upper middle-income countries [6]. 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. According to the 2015-2020 survey by the National Strategy for the Prevention and Control of NCDs, diabetes is one of the four most common chronic illnesses in Kenya, along with a strategy for health system reform, risk factor reduction, and promotion of good health. The National Hospital Insurance Fund (NHIF) has also lately created a specific chronic sickness care package. Despite these national efforts, sub-county and county-level diabetes care infrastructures remain unreliable, and there is a scarcity of diabetes data [7, 8]. Although the prevalence of diabetes in Kenya is under-reported, it is estimated that up to 60% of diabetics in the country go untreated [9]. Despite the devolution in the Kenyan system of government in 2010 where counties are given the exclusive rights to oversee their health system, only very miniature progress has been made in the country towards universal health coverage (UHC) [10]. According to Otieno et al. [11], 68 percent of Kenyans' basic health needs are not being met.

Ajmera et al. [12] showed that mathematical analysis of diabetes has been effective. Li et al. [13] modified the mathematical model for the dynamics of diabetes by introducing the law of conservation in time-delay equations. The findings, however, are in agreement with the physiological observations but with more insightful information. Zhang et al. [14] considered the effects of saturated treatment on the trend of the diabetic population and the results show that treatment rate can control the diabetic population. Karachaliou et al. [3] offered a diabetes prevention model and highlighted that good diabetes prevention and diabetic patient care can help lower the burden of such illnesses in low-income countries. The rates of admission of diabetic patients into Ethiopian hospitals were mathematically modelled by Regassa and Tola [15] and using the parametric survival analysis, the admission rate for Ethiopia was estimated as 9.85 per 1000 persons per year. [16] proposed a mathematical model for the dynamics of diabetes and solved model using the homotopy analysis method. Nasir and Daud [17] compiled several differential equations on the dynamics of diabetes and suggested an area for further is to consider how available resources may limit the total number of treated diabetics with complications. With this motivation, this study explores the effects of fixed unchanging health resources on the diabetic population.

A mathematical model for treating diabetes and its repercussions in an environment with constant resources is developed and tested in this work. This study explores the impact of increasing recovery rates, healthy lifestyle, constant hospitalisation rate on the population.

2. Methodology

2.1. Formulation of the Mathematical Model

Figure 1 shows the flowchart of the model under consideration. The population is compartmentalised into four classes; the Susceptible class $S(t)$, Diabetic class $D(t)$, Complicated class $C(t)$, and Hospitalised class $H(t)$. The susceptible class are the non-diabetics who are can become diabetic, the diabetic class are those who are already diabetic, the complicated class are diabetic individuals who have developed complications, and the hospitalised class are complicated class that are in the hospital. Suppose that (i) healthy people have healthy children, (ii) diabetic adults have either healthy or infected children, and (iii) complications arising from diabetes can be treated, but not diabetes.

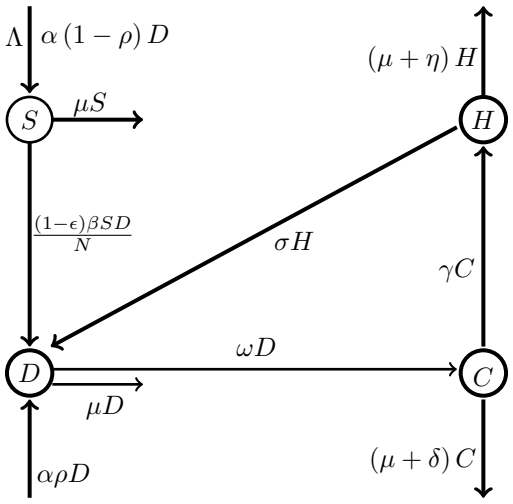

Figure 1. *Flowchart for the model.*

α is the birth rate, ρ is the proportion of diabetic births, ω is the rate of developing complications due to diabetes, δ is the proportion of death from complication, γ is the per capita hospitalization rate, σ is the recovery rate from complications, η is mortality rate among the hospitalised individuals, μ is taken as the natural mortality rate, and ϵ is the lifestyle incidence rate ($\epsilon = 0$ represents the case of no impacted lifestyle and $\epsilon = 1$ is the highest lifestyle standards). β represents the proportion of interactions leading to incidence, and hence, the total incidence due to lifestyle is

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

The equations governing the dynamics shown in figure 1 is

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

$$\frac{dD}{dt} = \alpha\rho D + (1-\epsilon)\,\beta\frac{SD}{N} + \sigma H - \omega D - \mu D, \qquad (2)$$

$$\frac{dC}{dt} = \omega D - \gamma C - \delta C - \mu C, \tag{3}$$

$$\frac{dH}{dt} = \gamma C - \sigma H - \eta H - \mu H. \tag{4}$$

2.2. Qualitative Analysis

2.2.1. Reproduction Number

The disease free equilirbium point is

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

and the endemic equilibrium

$$E_1 = (C_1, D_1, H_1, S_1).$$

where

$$D_1 = \frac{C_1}{\omega}(\gamma + \delta + \mu),\ H_1 = \frac{\gamma C_1}{\sigma+\eta+\mu}, \tag{5}$$

$$S = \frac{\Lambda}{\mu} + \left(\frac{(\alpha-\omega-\mu)(\gamma+\delta+\mu)}{\omega} + \frac{\sigma\gamma}{\sigma+\eta+\mu}\right)\frac{C_1}{\mu}, \tag{6}$$

The reproduction number R_0 is determined using the next generation matrix method as follows (see [18, 19, 20, 21]). Let the matrices F and V represent the new infections and negated outward transitions from these compartments respectively, then

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

from which

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

and the eigenvalues are

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

Finally, the basic reproduction number is

$$R_0 = \frac{(1-\epsilon)\frac{\Lambda\beta}{N\mu}}{(\omega+\mu-\alpha\rho)}.$$

2.2.2. Local Stability of the Equilibrium Points

According to the formulations of Oke and Bada [22], the Jacobian matrix for the system (1 - 4) is

$$J = \begin{pmatrix} -\frac{(1-\epsilon)\beta D}{N} - \mu & \alpha(1-\rho) - \frac{(1-\epsilon)\beta S}{N} & 0 & 0 \\ \frac{(1-\epsilon)\beta D}{N} & \alpha\rho + \frac{(1-\epsilon)\beta S}{N} - \omega - \mu & 0 & \sigma \\ 0 & \omega & -\gamma - \delta - \mu & 0 \\ 0 & 0 & \gamma & -\sigma - \eta - \mu \end{pmatrix}. \tag{7}$$

Evaluating J at E_0, we have

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

and evaluating at E_1, we have

$$J = \begin{pmatrix} -\frac{(1-\epsilon)\beta D_1}{N} - \mu & \alpha(1-\rho) - \frac{(1-\epsilon)\beta S_1}{N} & 0 & 0 \\ \frac{(1-\epsilon)\beta D_1}{N} & \alpha\rho + \frac{(1-\epsilon)\beta S_1}{N} - \omega - \mu & 0 & \sigma \\ 0 & \omega & -\gamma - \delta - \mu & 0 \\ 0 & 0 & \gamma & -\sigma - \eta - \mu \end{pmatrix}. \tag{8}$$

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

Theorem 2.1. The DFE of the system (1 - 4) is locally asymptotically stable if $R_0 < 1$.

Proof The characteristic equation of the Jacobian at the DFE is $|J_{E_0} - \lambda I| = 0$, so that

$$\begin{vmatrix} -\mu-\lambda & \alpha(1-\rho) - \frac{(1-\epsilon)\beta\Lambda}{N\mu} & 0 & 0 \\ 0 & \alpha\rho + \frac{(1-\epsilon)\beta\Lambda}{N\mu} - \mu - \omega - \lambda & 0 & \sigma \\ 0 & \omega & -\gamma-\delta-\mu-\lambda & 0 \\ 0 & 0 & \gamma & -\sigma-\eta-\mu-\lambda \end{vmatrix} = 0. \tag{9}$$

Letting

$$\begin{aligned} \xi_2 &= (\gamma+\delta+\mu+\sigma+\eta+\mu) - \frac{(1-\epsilon)\beta\Lambda}{N\mu} + (\omega+\mu-\alpha\rho), \\ \xi_1 &= (\gamma+\delta+\mu)(\sigma+\eta+\mu) + \left(\frac{(1-\epsilon)\beta\Lambda}{N\mu} - (\omega+\mu-\alpha\rho)\right) \times (\gamma+\delta+\mu+\sigma+\eta+\mu), \\ \xi_0 &= -\sigma\omega\gamma - \left(\frac{(1-\epsilon)\beta\Lambda}{N\mu} - (\omega+\mu-\alpha\rho)\right)(\gamma+\delta+\mu)(\sigma+\eta+\mu). \end{aligned}$$

Then the characteristic equation becomes

$$(\mu+\lambda)\left(\lambda^3 + \xi_2\lambda^2 + \xi_1\lambda + \xi_0\right) = 0.$$

The first eigenvalue is $\lambda_1 = -\mu$ is negative. Using Routh-Hurwitz criteria, the other three eigenvalues are of negative real parts if (i) $\xi_2 > 0$, (ii) $\xi_2\xi_1 > \xi_0$, and (iii) $\xi_0 > 0$. Hence, from (i) it is required that

$$(\gamma+\delta+\mu+\sigma+\eta+\mu) - (\omega+\mu-\alpha\rho)(R_0-1) > 0, \tag{10}$$

$$\Rightarrow R_0 - 1 < 0 \;\Rightarrow\; R_0 < 1. \tag{11}$$

From (iii),

$$-\sigma\omega\gamma - \left(\frac{(1-\epsilon)\beta\Lambda}{N\mu} - (\omega+\mu-\alpha\rho)\right)(\gamma+\delta+\mu)(\sigma+\eta+\mu) > 0, \tag{12}$$

and this implies $\Rightarrow\; R_0 < 1$. Now, from (ii), $\xi_2\xi_1 > \xi_0 \;\Rightarrow\; \xi_1 > 0$ and hence,

$$\begin{aligned} -1 &> (1-R_0)(\omega+\mu-\alpha\rho)\left(\frac{1}{\gamma+\delta+\mu} + \frac{1}{\sigma+\eta+\mu}\right) \\ &> (1-R_0)(\omega+\mu-\alpha\rho), \\ \Rightarrow R_0 - 1 &< -\frac{1}{\omega+\mu-\alpha\rho} < 0 \;\Rightarrow\; R_0 < 1 \end{aligned}$$

Therefore, the DFE is asymptotically stable if $R_0 < 1$.

Theorem 2.2. The EEP of the system (1 - 4) is locally asymptotically stable if $R_0 < 1$.

Proof Letting $B_1 = \frac{(1-\epsilon)\beta}{N}$, then the Jacobian at the EEP E_1 is

$$J_1 \;=\; \begin{pmatrix} -B_1D_1 - \mu & \alpha(1-\rho) - B_1S_1 & 0 & 0 \\ B_1D_1 & \alpha\rho + B_1S_1 - \mu - \omega & 0 & \sigma \\ 0 & \omega & -\gamma-\delta-\mu & 0 \\ 0 & 0 & \gamma & -\sigma-\eta-\mu \end{pmatrix}. \tag{13}$$

The characteristic equation of J_1 is

$$\begin{vmatrix} -B_1D_1 - \mu - \lambda & \alpha(1-\rho) - B_1S_10 & 0 & 0 \\ B_1D_1 & \alpha\rho + B_1S_1 - \mu - \omega - \lambda & 0 & \sigma \\ 0 & \omega & -\gamma-\delta-\mu-\lambda & 0 \\ 0 & 0 & \gamma & -\sigma-\eta-\mu-\lambda \end{vmatrix} = 0. \tag{14}$$

and thus on setting,

$$\begin{aligned} A_1 &= B_1D_1 + \mu - \alpha\rho - B_1S_1 + \mu + \omega, \\ A_2 &= (B_1D_1+\mu)(\mu+\omega) - \mu(\alpha\rho + B_1S_1) - \alpha B_1D_1, \\ A_3 &= \gamma+\delta+\mu+\sigma+\eta+\mu, \\ A_4 &= (\gamma+\delta+\mu)(\sigma+\eta+\mu), \end{aligned}$$

then the characteristic equation becomes

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

where

$$\begin{aligned} \xi_0 &= A_2A_4 - \sigma\omega\gamma\left(B_1D_1 + \mu\right), \\ \xi_1 &= A_1A_4 + A_2A_3 - \sigma\omega\gamma, \\ \xi_2 &= A_1A_3 + A_2 + A_4,\ \xi_3 = A_1 + A_3. \end{aligned} \tag{15}$$

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

$$\xi_3 > 0, \quad \frac{\xi_3\xi_2 - \xi_1}{\xi_3} > 0, \quad \frac{\left(\xi_3\xi_2 - \xi_1\right)\xi_1}{\xi_3} - \xi_3\xi_0 > 0, \quad \xi_0 > 0. \tag{16}$$

By substituting (15) into (16), we have;

$$A_1 + A_3 > 0; \tag{17}$$

$$\frac{\left(A_1 + A_3\right)\left(A_1A_3 + A_2 + A_4\right) - \left(A_1A_4 + A_2A_3 - \sigma\omega B_2\right)}{A_1 + A_3} > 0; \tag{18}$$

$$A_2A_4 - \sigma\omega B_2\left(B_1D_1 + \mu\right) > 0; \tag{19}$$

$$\frac{\left(A_1 + A_3\right)\left(A_1A_3 + A_2 + A_4\right) - \left(A_1A_4 + A_2A_3 - \sigma\omega B_2\right)}{A_1 + A_3} - \left(A_1 + A_3\right)\left(A_2A_4 - \sigma\omega B_2\left(B_1D_1 + \mu\right)\right) > 0. \tag{20}$$

Equation (17) is always true. Rewriting equation (18), then we have

$$\left(A_1 + A_3\right)\left(A_1A_3 + A_2 + A_4\right) - \left(A_1A_4 + A_2A_3 - \sigma\omega B_2\right) > 0. \tag{21}$$

Rewriting equation (19) gives

$$\frac{\left(A_1 + A_3\right)\left(A_1A_3 + A_2 + A_4\right) - \left(A_1A_4 + A_2A_3 - \sigma\omega B_2\right)}{\left(A_1 + A_3\right)^2\left(A_2A_4 - \sigma\omega B_2\left(B_1D_1 + \mu\right)\right)} > 1, \tag{22}$$

Observe that the inequality (22) if the inequalities (21) and (19) hold. The four conditions therefore are reduced to

$$A_2A_4 - \sigma\omega B_2\left(B_1D_1 + \mu\right) > 0 \tag{23}$$

which implies that

$$A_2A_4 > \sigma\omega B_2\left(B_1D_1 + \mu\right) > 0 \ \Rightarrow\ A_2 > 0 \ \text{ since } A_4 > 0$$

$$\Rightarrow -\left(B_1D_1 + \mu\right)\left(\mu + \omega\right) + \mu\left(\alpha\rho + B_1S_1\right) + \alpha B_1D_1 < 0$$

$$\Rightarrow -\left(1 - \frac{B_1S_1}{\omega + \mu - \alpha\rho}\right)\mu - B_1D_1\frac{\left(\mu + \omega - \alpha\right)}{\omega + \mu - \alpha\rho} < 0$$

$$\Rightarrow \left(R_0 - 1\right)\mu < 0 \ \Rightarrow\ R_0 < 1$$

Therefore, the EEP is asymptotically stable if $R_0 < 1$.

2.2.3. Positivity and Boundedness of Solution

In what follows, we shall show that the solution space for the model equations (1 - 4) is bounded and positive provided $S_0 > 0, D_0 > 0, C_0 > 0$ and $H_0 > 0$. Setting $N = S + D + C + H$ and summing up the system (1 - 4), then

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

which on solving gives

$$N \leq \frac{\Lambda}{\mu} - \left(\frac{\Lambda}{\mu} - N_0\right)\exp\left(-\mu t\right) + \alpha\exp\left(-\mu t\right)\int_0^t D\exp\left(\mu\tau\right)d\tau$$

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

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

Also, from equation (1 - 4),

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda + \alpha (1-\rho) D - (1-\epsilon) \beta \frac{SD}{N} - \mu S \geq -\mu S \Rightarrow S \geq S_0 \exp(-\mu t), \\
\frac{dD}{dt} &= \alpha \rho D + (1-\epsilon) \beta \frac{SD}{N} + \sigma H - \omega D - \mu D \geq -(\mu + \omega) D \Rightarrow D \geq D_0 \exp(-(\mu + \omega) t), \\
\frac{dC}{dt} &= \omega D - \gamma C - \delta C - \mu C \geq -(\gamma + \delta + \mu) C \Rightarrow C \geq C_0 \exp(-(\gamma + \delta + \mu) t), \\
\frac{dH}{dt} &= \gamma C - \sigma H - \eta H - \mu H \geq -(\sigma + \delta + \mu) H \Rightarrow H \geq H_0 \exp(-(\sigma + \delta + \mu) t).
\end{aligned}$$

Thus, solutions S, D, C, H remain positive in the region $\mathcal{R}$.

2.3. Numerical Procedure

Equations (1 - 4) are solved using the Runge-Kutta scheme of the fourth order. The fourth order Runge-Kutta scheme for the autonomous differential equations

$$\dot{X} = F(X), \; X(0) = X_0$$

where

$$\begin{cases} X = (x_1, x_2, \cdots x_n)^T, \\ \dot{X} = (\dot{x}_1, \dot{x}_2, \cdots \dot{x}_n)^T, \\ F(X) = (f_1, f_2, \cdots, f_n)^T. \end{cases}$$

is given as

$$K_4 = hF\left(X_n + \frac{1}{2}\left(hF\left(X_n + \frac{1}{2}\left(hF\left(X_n + \frac{1}{2}\left(hF\left(X_n\right)\right)\right)\right)\right)\right)\right), \tag{24}$$

and

$$X_{n+1} = X_n + \frac{1}{6}(K_1 + 2K_2 + 2K_3 + K_4).$$

The choice of the fourth order Runge-Kutta Scheme is due to its stability and large region of convergence (see [23] for other methods). Absolute error tolerance is set to 10^{-8} and the numerical solutions obtained are plotted as graphs to evaluate the trends as the parameter values are varied. The parameter values are chosen according to the studied of [24] on the Kenyan population as follows;

$$\Lambda = 3.3; \alpha = 0.1; \rho = 0.2; \epsilon = 0.41; \mu = 1/65; \beta = 0.2;$$
$$\sigma = 0.1; \omega = 0.1; \gamma = 0.1; \delta = 0.3; \eta = 0.08.$$

The results are validated with the study of [24] and there is a great agreement.

3. Analysis and Discussion of Results

Lifestyle can be transmitted from a diabetic patient to susceptible individual due to interaction between diabetic patients and the susceptible class. It is also important to note that not all interactions lead to lifestyle transfer, hence β represents the proportion of such interactions that lead to a transfer of lifestyle. Figures 2-5 show the effect of β on the population. As β increases, most susceptible individuals will acquire an unhealthy lifestyle from the diabetic subpopulation due to interactions. For this reason, the susceptible class declines as β increases (as seen in Figure 2). Hence, this will cause a sudden rise in the diabetic class which will decline due to a reduction in the susceptible (see Figure 3). The same pattern obtained in the diabetic class is also obtained in the hospitalised and the complicated classes (Figures 4 and 5).

Healthy lifestyles can also be transmitted among the individuals within the susceptible class. The parameter ϵ represents the rate at which people within the susceptible class embrace healthy lifestyle. As ϵ changes from 0 to 1, the quality of the healthy lifestyle increases from the least to the best quality of lifestyle. By increasing ϵ, the quality of lifestyle gets better among the individuals in the susceptible subpopulation. With this in place, the susceptible class continues to rise (see Figure 6). Consequently, the number of people who migrate into the diabetic class reduces, the complicated class reduces and the hospitalised class reduces (see Figures 7-9).

As no cure has been found for diabetes yet, diabetes can only be managed. Complications arising as a result of diabetes can be cured but not the underlying diabetes. The parameter σ represents the rate at which complicated cases are cured of their complications so that they can return to the diabetic class. Hence, increasing σ means increasing Progression from Hospitalised class back into the diabetic class. This leads to a reduction in the hospitalised class (see Figure 11) but an increase in the diabetic class (see Figure 10).

The per capita hospitalisation rate γ represents a constant hospitalisation rate from the complicated class. By raising the values of γ, the Complicated class begins to reduce (see figure 12) while both the diabetic and hospitalised classes increase. If the constant hospitalisation rate continues to increase, then the hospitals will become congested and overloaded. Hence, a constant hospitalisation rate is not a good way to control the complications in the diabetic patients.

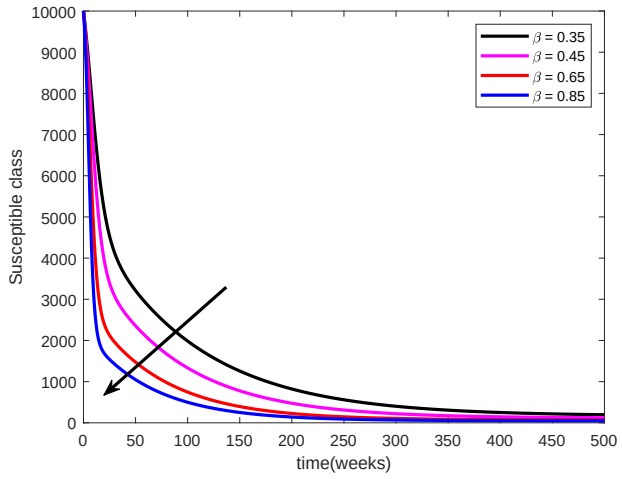

Figure 2. *Variation of the Susceptible class with Diabetic-Susceptible interactions.*

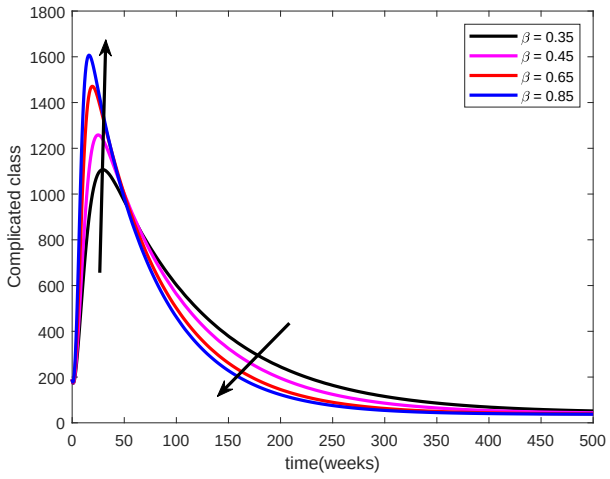

Figure 5. *Variation of the Complicated cases with Diabetic-Susceptible interactions.*

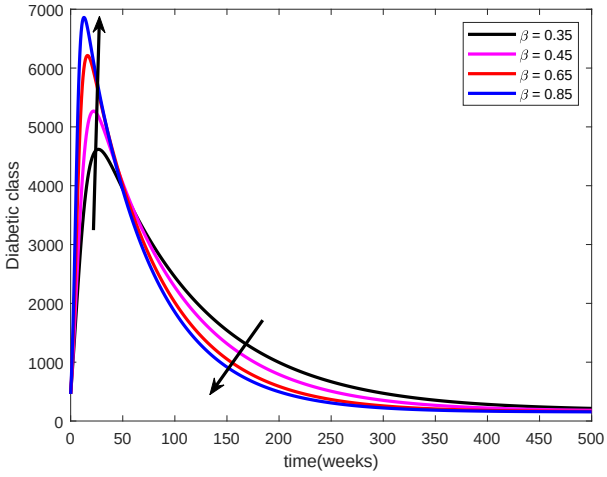

Figure 3. *Variation of the Diabetic cases with Diabetic-Susceptible interactions.*

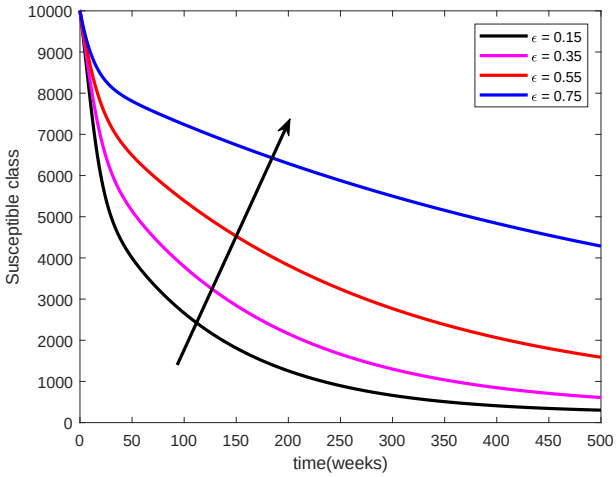

Figure 6. *Variation of the Susceptible class with lifestyle impact.*

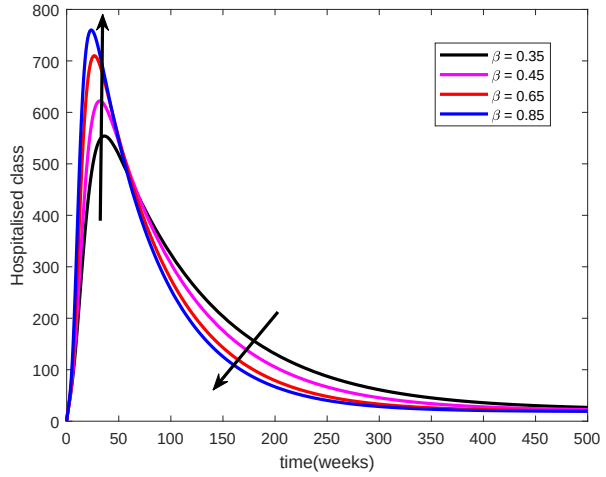

Figure 4. *Variation of the Hospitalised cases with Diabetic-Susceptible interactions.*

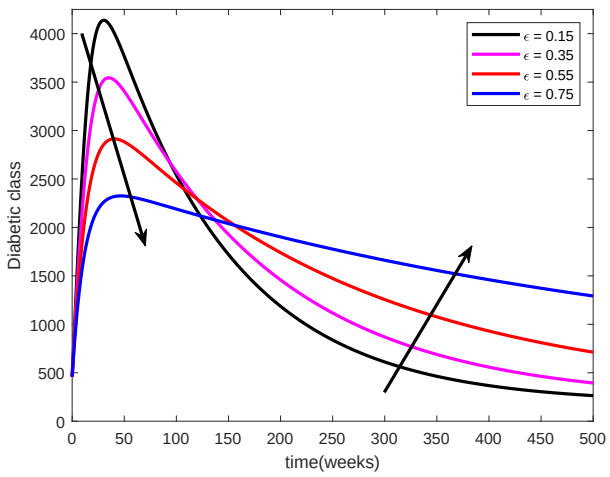

Figure 7. *Variation of the Diabetic class with lifestyle impact.*

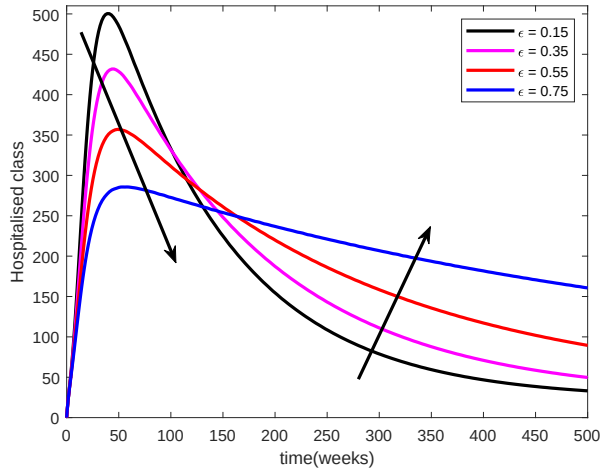

Figure 8. *Variation of the Hospitalised class with lifestyle impact.*

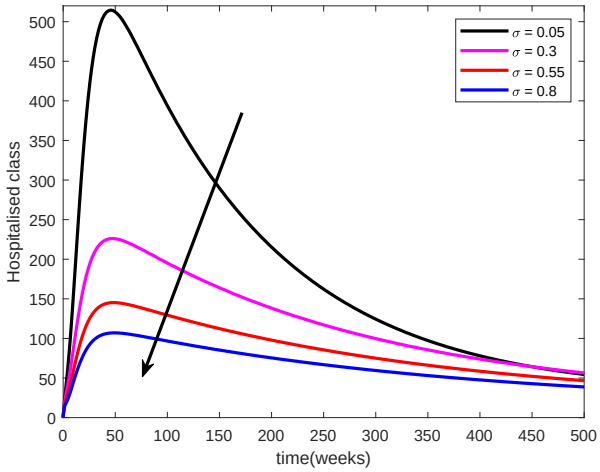

Figure 11. *Variation of the Hospitalised class with recovery rate.*

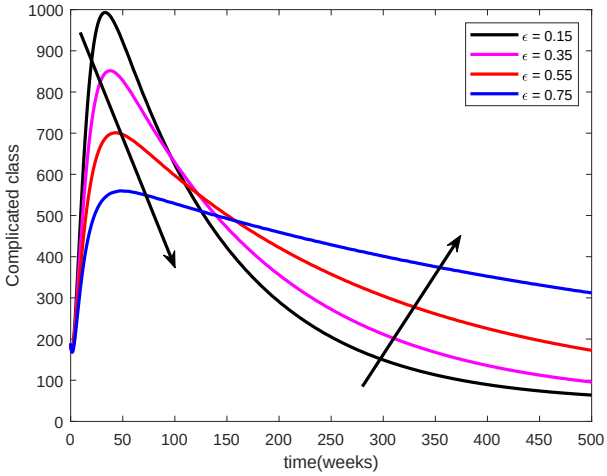

Figure 9. *Variation of the Complicated cases with lifestyle impact.*

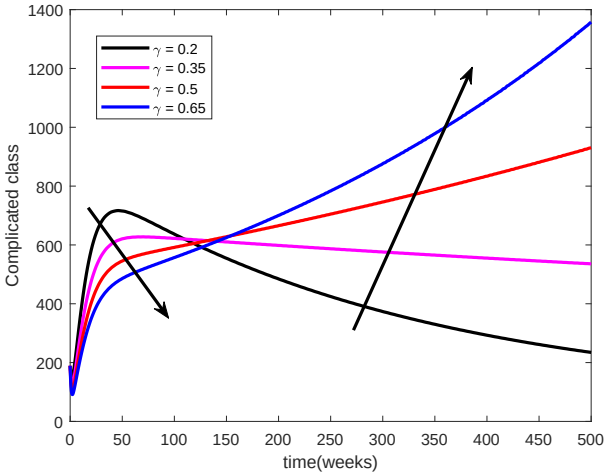

Figure 12. *Variation of the Complicated cases with Hospitalisation rate.*

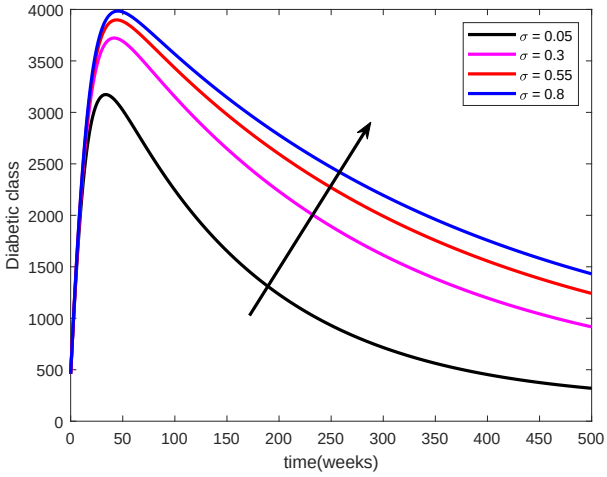

Figure 10. *Variation of the Diabetic class with recovery rate.*

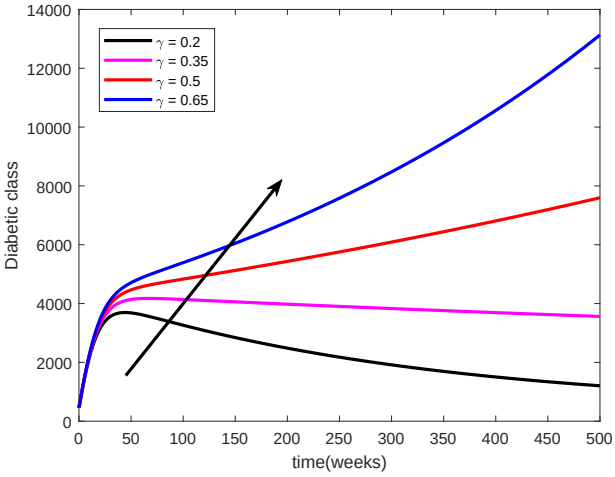

Figure 13. *Variation of the Diabetic class with Hospitalisation rate.*

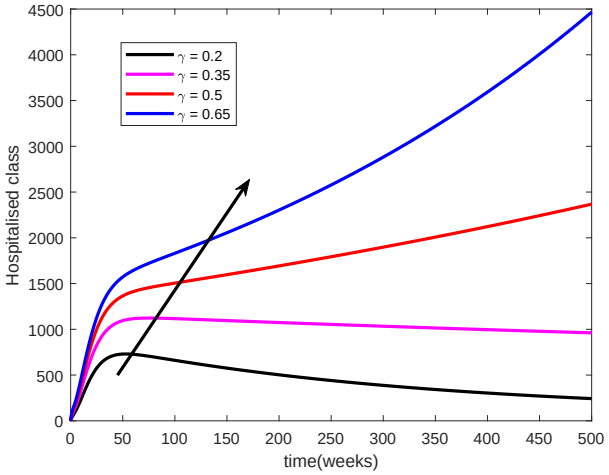

Figure 14. *Variation of the Hospitalised class with Hospitalisation rate.*

4. Conclusion

This study investigates the behaviour of the diabetic population, taking into account the complicated diabetic cases with a constant per capita hospitalization rate. A mathematical model is developed to investigate the impact of the parameters on the diabetic populations. The equilibrium points and the reproduction numbers are obtained. The equilibrium points are shown to be locally stable if $R_0 < 1$. The model is shown to be well-posed, positive and bounded. The models are numerically solved using the Runge-Kutta 4 technique, and the results are graphed. The following results were obtained:

1. An increase in the unhealthy lifestyle leads to
    a. a decrease in the susceptible class.
    b. a sudden rise in the diabetic, the hospitalised and the complicated classes and a decline over time.
2. An increase in the healthy lifestyle leads to an increase in the susceptible class and a reduction in the diabetic, the complicated and the hospitalised classes.
3. A constant per capita hospitalisation rate will lead to overburdening of the health facility. Hence, resources at the health facilities should not be left fixed for too long.

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