Mathematical Modelling of Diabetes with Constant Hospitalisation Rate in Kenya

R. N. Andima[1], W. N. Mutuku[1], A. S. Oke[1,*], N. Farai[2], K. Awuor[1], B. A. Juma[1]

[1]Department of Mathematics and Actuarial Science, Kenyatta University, Kenya
[2]Department of Mathematics and Applied Mathematics, University of Johannesburg

Abstract Blindness, stroke, and amputations are some of the consequences of diabetes on humans. The fact that over 500 billion USD is spent on diabetes diagnosis, care, and treatment each year demonstrates its massive impact on the global economy. In Kenya, the incidence rate of diabetes and the number of death from diabetes and diabetes-related infections keep rising. At this rate, healthcare facilities and resources would undoubtedly become overloaded. The impact of a constant hospitalization rate on the trajectory of diabetes is explored in this study. A system of ordinary differential equations is developed to describe the dynamics of diabetes with a constant hospitalization rate. The model is shown to be well-posed, positive, and bounded, and the requirements for the local stability of the equilibrium points are established. The next generation matrix is used to calculate the reproduction number. The model is numerically solved using the explicit Runge-Kutta 4th order and the results are graphed. Improvement in lifestyle quality among the susceptible class reduces the migration from the susceptible subpopulation to the diabetic population.

Keywords Diabetes, Hospitalisation, Mathematical modelling, Per capita hospitalisation rate

1. Background Information

Chronic diseases develop over time as a result of a mix of genetic, environmental, and behavioural factors. Noncommunicable diseases are classified into four categories: diabetes, cancer, chronic respiratory ailments, and cardiovascular problems. Each year, more than 41 million people die from NCDs, with low- and middle-income countries accounting for 85% of these fatalities [13]. Food taken into the human body is converted into glucose which is transported into the bloodstream. A signal is received by the pancreas to secret insulin when there is an increase in the bloodstream glucose level. The insulin converts the glucose into usable energy for the other cells to use. Diabetes is a disease that results when the insulin-glucose-glycogen control mechanism fails. In this condition, the pancreas produces either too little insulin, which is insufficient to convert the body's sugar, or too much unhelpful insulin [7,9]. Diabetes is the leading cause of kidney illness, cardiovascular disease, blindness, and lower limb amputation [22,1]. 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 [10]. 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 [8,24]. Although the prevalence of diabetes in Kenya is under-reported, it is estimated that up to 60% of diabetics in the country go untreated [16]. 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) [15]. According to Otieno et al. [21], 68 percent of Kenyans' basic health needs are not being met.

According to Ajmera et al. [2], analysis of the dynamics of diabetes using mathematical models has shown to be quite effective. Several mathematical models have been presented

by researchers to investigate glucose-insulin behaviour in diabetes, diabetes prevention and management, the cost and cost-effectiveness of diabetes treatment alternatives, and diabetes epidemiology. Boutayeb and Derouich [6] modelled the use of physical activities to increase insulin sensitivity and consequently regulate blood glucose levels. The importance and impact of physical activity in diabetes patients were demonstrated. Physical activity has been shown to enhance insulin sensitivity and compensate for insulin deficiencies in patients. Boutayeb et al. [7] considered the case of complicated and non-complicated diabetes in diabetic patients and proposed a mathematical model to study the dynamics of diabetes. Li et al. [12] 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. [26] 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. [9] 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 [23] and using the parametric survival analysis, the admission rate for Ethiopia was estimated as 9.85 per 1000 persons per year. [4] proposed a mathematical model for the dynamics of diabetes and solved model using the homotopy analysis method. Ali et al. [3] proposed a model for measuring the glucose concentration in the bloodstream. The Bayesian framework with Markov chain Monte Carlo was used to study diabetes in Kigali, Rwanda.

Nasir and Daud [14] compiled several differential equations on the dynamics of diabetes. Complications were considered as non-existent, constant, rate-dependent, and with control. A qualitative analysis of the models were carried out and areas for further research were suggested in the concluding remark. One suggested area for further study

is to consider how available resources may limit the total number of treated diabetics with complications. With this motivation, this study is aimed at unravelling the effects of constant resource allocations on the diabetic population.

In this study, a mathematical model for managing diabetes and associated consequences in a context with constant resources is formulated and analysed. The questions answered in this study include;

i. What is the impact of increasing recovery rates among the hospitalised individuals on the trends in diabetes?
ii. What is the impact effect of increasing healthy lifestyle on the susceptible and diabetic populations?
iii. What is the impact effect of increasing unhealthy lifestyle on the susceptible and diabetic populations?
iv. What is the impact of a constant per capita hospitalisation rate on the entire population?

2. Methodology

2.1. Formulation of the Mathematical Model

The flowchart for the mathematical model that considers the significance of intervention and the limited healthcare resources on the dynamics of diabetes is shown in Figure (1). The entire population is divided into four compartments; $S(t)$ is the first compartment called Susceptible compartment, $D(t)$ is the Diabetic compartment, $C(t)$ is the Complicated compartment, and $H(t)$ is the Hospital compartment. The susceptible compartment includes non-diabetic individuals who are at risk of becoming diabetic, the diabetic compartment includes individuals who are already diabetic, the complicated compartment includes diabetic individuals who have developed complications as a result of their diabetes, and the hospital compartment includes complicated cases that have been hospitalized. In this study, it is assumed that healthy people can only have healthy children, diabetic adults can have either diabetic or healthy children, and complicated cases can be treated to cure the complications but not the diabetes.
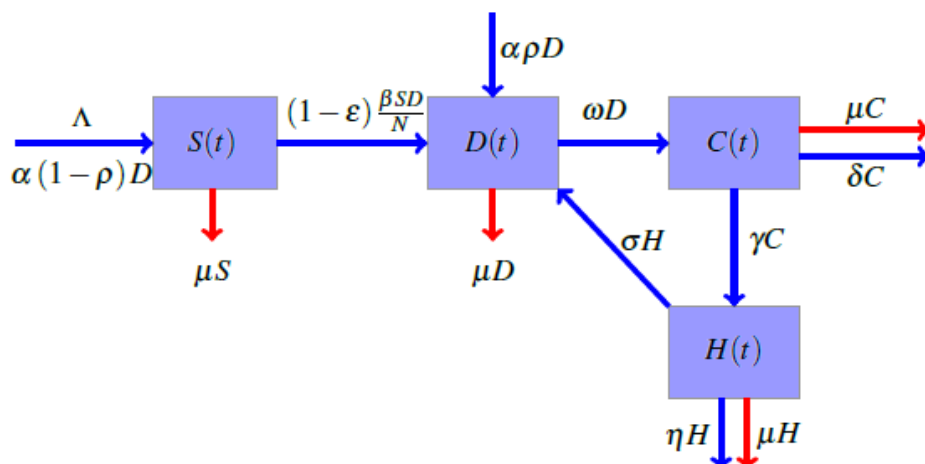

Figure 1. Flowchart for the model

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

$$\frac{dS}{dt} = \Lambda + \alpha(1-\rho)D - (1-\epsilon)\beta\frac{SD}{N} - \mu S, \qquad (2.1)$$

$$\frac{dD}{dt} = \alpha\rho D + (1-\epsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D, \qquad (2.2)$$

$$\frac{dC}{dt} = \omega D - \gamma C - \delta C - \mu C, \qquad (2.3)$$

$$\frac{dH}{dt} = \gamma C - \sigma H - \eta H - \mu H. \qquad (2.4)$$

2.2. Qualitative Analysis

2.2.1. Reproduction Number

Following the studies of Kimulu et al. [11] and Oke et al. [19], the Diabetic free equilibrium (DFE) E_0 is

$E_0 = \left(\dfrac{\Lambda}{\mu}, 0, 0, 0\right)$ and the endemic equilibrium point (EEP) E_1 is $E_1 = \left(C^*, D^*, H^*, S^*\right)$, where

$$D^* = \frac{C^*}{\omega}(\gamma + \delta + \mu), \quad H^* = \frac{\gamma C^*}{\sigma + \eta + \mu},$$

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

The reproduction number R_0 is determined using the next generation matrix method as follows (see [25,5]). Let the matrices F and V represent the new infections and negated outward transitions from these compartments respectively, then

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

from which

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

and the eigenvalues are

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

Finally, the basic reproduction number is

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

2.2.2. Local Stability of the Equilibrium Points

According to the formulations of Oke and Bada [18], the Jacobian matrix for the system (2.1 – 2.4) is

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

Evaluating J at E_0, we have

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

and evaluating at E_1, we have

$$J_1 = \begin{pmatrix} -\dfrac{(1-\epsilon)\beta D^*}{N} - \mu & \alpha(1-\rho) - \dfrac{(1-\epsilon)\beta S^*}{N} & 0 & 0 \\[3mm] \dfrac{(1-\epsilon)\beta D^*}{N} & \alpha\rho + \dfrac{(1-\epsilon)\beta S^*}{N} - \mu - \omega & 0 & \sigma \\[3mm] 0 & \omega & -\gamma - \delta - \mu & 0 \\[2mm] 0 & 0 & \gamma & -\sigma - \eta - \mu \end{pmatrix}.$$

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

Theorem 2.1. *The DFE of the system (2.1-2.4) is locally asymptotically stable if $R_0 < 1$.*

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

$$\begin{vmatrix} -\mu - \lambda & \alpha(1-\rho) - \dfrac{(1-\epsilon)\beta\Lambda}{N\mu} & 0 & 0 \\[3mm] 0 & \alpha\rho + \dfrac{(1-\epsilon)\beta\Lambda}{N\mu} - \mu - \omega - \lambda & 0 & \sigma \\[3mm] 0 & \omega & -\gamma - \delta - \mu - \lambda & 0 \\[2mm] 0 & 0 & \gamma & -\sigma - \eta - \mu - \lambda \end{vmatrix} = 0.$$

Letting

$$\xi_2 = (\gamma + \delta + \mu + \sigma + \eta + \mu) - \dfrac{(1-\epsilon)\beta\Lambda}{N\mu} + (\omega + \mu - \alpha\rho).$$

$$\xi_1 = (\gamma + \delta + \mu)(\sigma + \eta + \mu) + \left(\dfrac{(1-\epsilon)\beta\Lambda}{N\mu} - (\omega + \mu - \alpha\rho) \right)(\gamma + \delta + \mu + \sigma + \eta + \mu)$$

$$\xi_0 = -\left(\dfrac{(1-\epsilon)\beta\Lambda}{N\mu} - (\omega + \mu - \alpha\rho) \right)(\gamma + \delta + \mu)(\sigma + \eta + \mu) - \sigma\omega\gamma.$$

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 the following three conditions are satisfied;

$$\begin{cases} \xi_2 > 0, \\ \xi_2\xi_1 > \xi_0, \\ \xi_0 > 0 \end{cases} \tag{2.5}$$

Condition 1 implies that

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

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

$$\Rightarrow (\gamma + \delta + \mu + \sigma + \eta + \mu) - (\omega + \mu - \alpha\rho)(R_0 - 1) > 0.$$

which finally means

$$R_0 - 1 < 0 \Rightarrow R_0 < 1.$$

Condition 3 implies that

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

$$\Rightarrow(R_0-1)(\omega+\mu-\alpha\rho)(\gamma+\delta+\mu)(\sigma+\eta+\mu)<-\sigma\omega\gamma<0,$$

$$\Rightarrow R_0-1<0\Rightarrow R_0<1.$$

Condition 2 implies that

$$\xi_2\xi_1>\xi_0\Rightarrow\xi_1>\frac{\xi_0}{\xi_2}\Rightarrow\xi_1>0$$

and hence,

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

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

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

$$-1>(1-R_0)(\omega+\mu-\alpha\rho)\left(\frac{1}{\gamma+\delta+\mu}+\frac{1}{\sigma+\eta+\mu}\right)>(1-R_0)(\omega+\mu-\alpha\rho),$$

$$R_0-1<-\frac{1}{\omega+\mu-\alpha\rho}<0\Rightarrow R_0<1$$

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

Theorem 2.2. *The EEP of the system (2.1-2.4) is locally asymptotically stable if* $R_0<1$.

Proof. Letting $B_1=\dfrac{(1-\epsilon)\beta}{N}$, then 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

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

and thus on setting,

$$A_1=B_1D^*+\mu-\alpha\rho-B_1S^*+\mu+\omega,$$

$$A_2=\left(B_1D^*+\mu\right)(\mu+\omega)-\mu\left(\alpha\rho+B_1S^*\right)-\alpha B_1D^*,$$

$$A_3=\gamma+\delta+\mu+\sigma+\eta+\mu,$$

$$A_4=(\gamma+\delta+\mu)(\sigma+\eta+\mu),$$

then the characteristic equation becomes

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

where

$$\xi_0 = A_2 A_4 - \sigma\omega\gamma\left(B_1 D^* + \mu\right), \ \xi_1 = A_1 A_4 + A_2 A_3 - \sigma\omega\gamma, \ \xi_2 = A_1 A_3 + A_2 + A_4, \ \xi_3 = A_1 + A_3. \tag{2.6}$$

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

$$\xi_3 > 0, \quad \frac{\xi_3\xi_2 - \xi_1}{\xi_3} > 0, \quad \frac{(\xi_3\xi_2 - \xi_1)\xi_1}{\xi_3} - \xi_3\xi_0 > 0, \quad \xi_0 > 0. \tag{2.7}$$

By substituting (2.6) into (2.7), we have;

$$A_1 + A_3 > 0; \tag{2.8}$$

$$\frac{(A_1 + A_3)(A_1 A_3 + A_2 + A_4) - (A_1 A_4 + A_2 A_3 - \sigma\omega B_2)}{A_1 + A_3} > 0; \tag{2.9}$$

$$A_2 A_4 - \sigma\omega B_2 \left(B_1 D^* + \mu\right) > 0; \tag{2.10}$$

$$\frac{(A_1 + A_3)(A_1 A_3 + A_2 + A_4) - (A_1 A_4 + A_2 A_3 - \sigma\omega B_2)}{A_1 + A_3} - (A_1 + A_3)\left(A_2 A_4 - \sigma\omega B_2 \left(B_1 D^* + \mu\right)\right) > 0. \tag{2.11}$$

Equation (2.8) is always true. Rewriting equation (2.9), then we have

$$(A_1 + A_3)(A_1 A_3 + A_2 + A_4) - (A_1 A_4 + A_2 A_3 - \sigma\omega B_2) > 0. \tag{2.12}$$

Rewriting equation (2.10) gives

$$\frac{(A_1 + A_3)(A_1 A_3 + A_2 + A_4) - (A_1 A_4 + A_2 A_3 - \sigma\omega B_2)}{(A_1 + A_3)^2 \left(A_2 A_4 - \sigma\omega B_2 \left(B_1 D^* + \mu\right)\right)} > 1 > 0, \tag{2.13}$$

Observe that the inequality (2.13) if the inequalities (2.12) and (2.10) hold. The four conditions therefore are reduced to

$$A_2 A_4 - \sigma\omega B_2 \left(B_1 D^* + \mu\right) > 0 \tag{2.14}$$

which implies that

$$A_2 A_4 > \sigma\omega B_2 \left(B_1 D^* + \mu\right) > 0 \Rightarrow A_2 > 0 \ \text{ since } A_4 > 0$$

$$\Rightarrow -\left(B_1 D^* + \mu\right)(\mu + \omega) + \mu\left(\alpha\rho + B_1 S^*\right) + \alpha B_1 D^* < 0$$

$$-\left(1 - \frac{B_1 S^*}{\omega + \mu - \alpha\rho}\right)\mu - B_1 D^* \frac{(\mu + \omega - \alpha)}{\omega + \mu - \alpha\rho} < 0$$

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

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 (2.1-2.4) 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 (2.1 - 2.4), then

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

which on solving gives

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

As $t \to \infty$, then $N \leq \dfrac{\Lambda}{\mu}$. 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 (2.1 – 2.4),

$$\frac{dS}{dt} = \Lambda + \alpha(1-\rho)D - (1-\epsilon)\beta\frac{SD}{N} - \mu S \geq -\mu S \Rightarrow S \geq S_0 \exp(-\mu t),$$

$$\frac{dD}{dt} = \alpha\rho D + (1-\epsilon)\beta\frac{SD}{N} + \sigma H - \omega D - \mu D \geq -(\mu + \omega)D \Rightarrow D \geq D_0 \exp(-(\mu + \omega)t),$$

$$\frac{dC}{dt} = \omega D - \gamma C - \delta C - \mu C \geq -(\gamma + \delta + \mu)C \Rightarrow C \geq C_0 \exp(-(\gamma + \delta + \mu)t),$$

$$\frac{dH}{dt} = \gamma C - \sigma H - \eta H - \mu H \geq -(\sigma + \delta + \mu)H \Rightarrow H \geq H_0 \exp(-(\sigma + \delta + \mu)t).$$

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

2.3. Numerical Procedure

Equations (2.1 – 2.4) 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

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

is given as

$$K_1 = hF(X_n), K_2 = hF\left(X_n + \frac{1}{2}K_1\right), K_3 = hF\left(X_n + \frac{1}{2}K_2\right), K_4 = hF\left(X_n + \frac{1}{2}K_3\right),$$

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 [17,20] 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. Default values chosen for the parameters are Kenyan

$$\Lambda = 3.3; \alpha = 0.1; \rho = 0.2; \epsilon = 0.41; \mu = 1/65; \beta = 0.2;$$

$$\sigma = 0.1; \omega = 0.1; \gamma = 0.1; \delta = 0.3; \eta = 0.08;$$

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. Figure (2) shows the effect of β on the diabetic 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. Hence, this will cause a sudden rise in the diabetic class which will decline due to a reduction in the susceptible (see Figure 2). The same pattern obtained in the diabetic class is also obtained in the hospitalised and the complicated classes.

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. Consequently, the number of people who migrate into the diabetic class reduces, the complicated class reduces and the hospitalised class reduces (see Figure (3)).

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 but an increase in the diabetic class (see Figure (4)).

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 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.
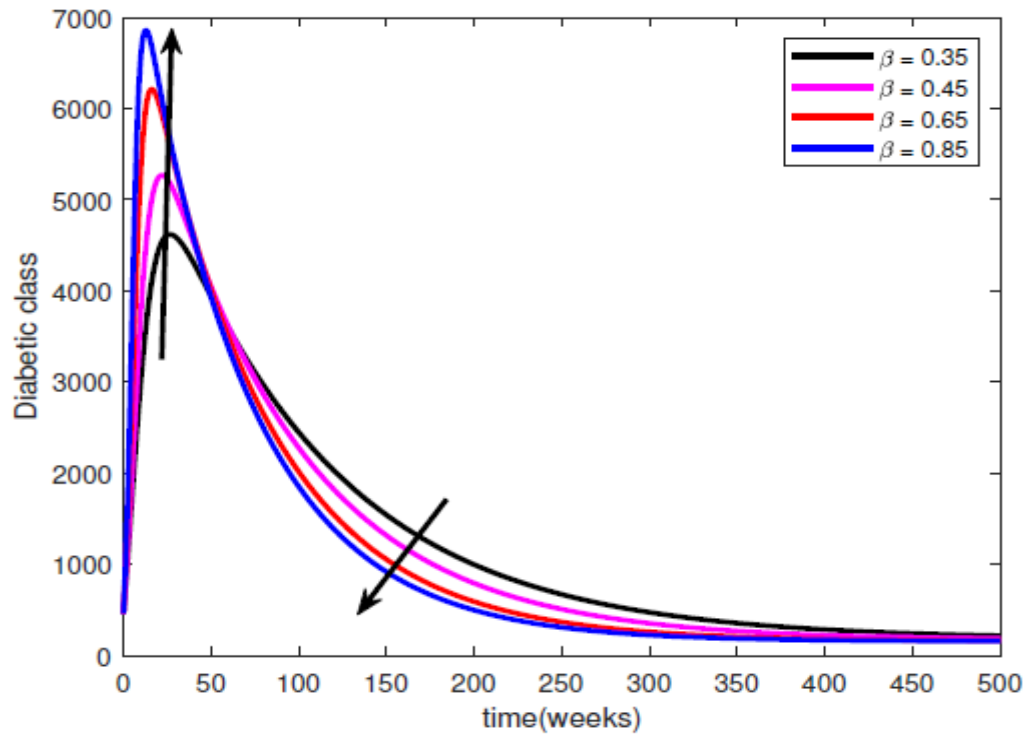

Figure 2.   Variation of the Diabetic cases with Diabetic-Susceptible interactions
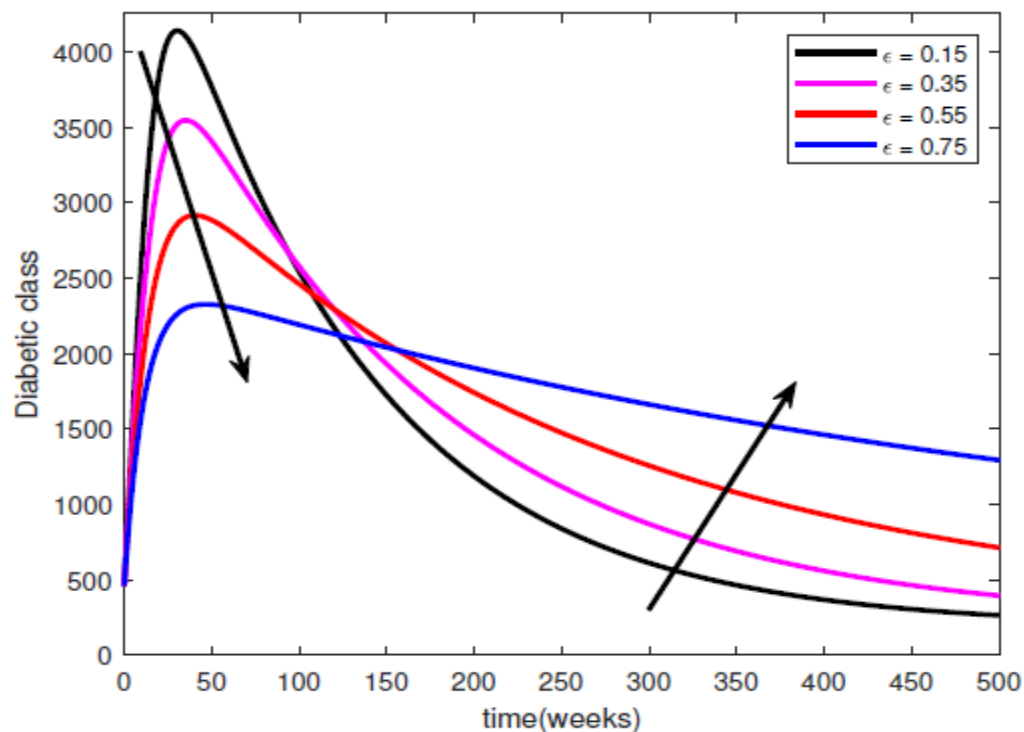

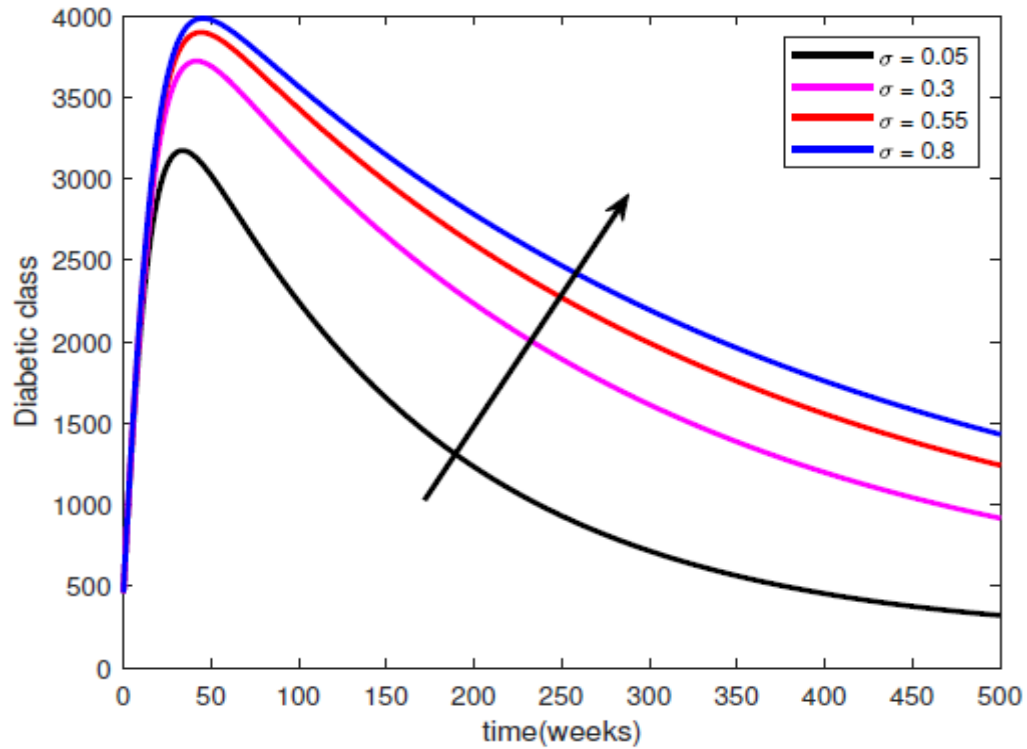**Figure 3.**   Variation of the Diabetic class with lifestyle impact

Figure 4.   Variation of the Diabetic class with recovery rate
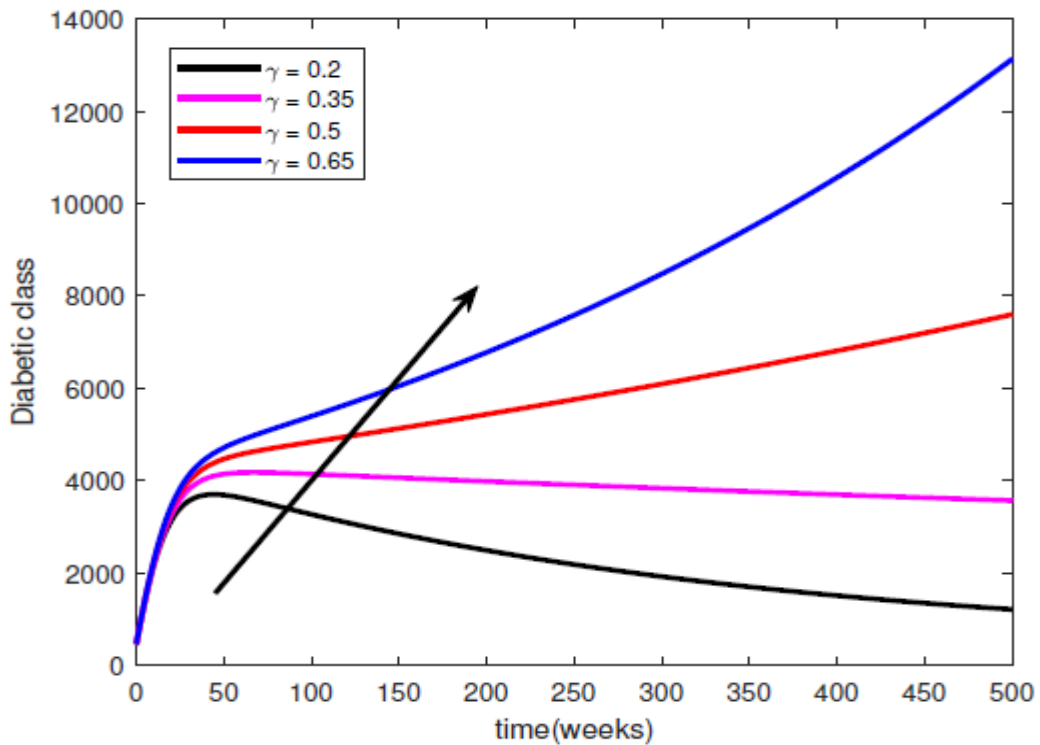

Figure 5.   Variation of the Diabetic class with Hospitalisation rate

4. Conclusions

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-possed,

positive and bounded. The models are numerically solved using the Runge-Kutta 4 technique, and the results are graphed. The following results were obtained:

i. An increase in the unhealthy lifestyle leads to a sudden rise in the diabetic which declines over time.

ii. An increase in the healthy lifestyle leads to a reduction in the diabetic class.

iii. 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.

REFERENCES

[1] Afaya, R. A., Bam, V., Azongo, T. B., and Afaya, A. (2020). Knowledge of chronic complications of diabetes among persons living with type 2 diabetes mellitus in northern Ghana. *PLOS ONE,* 15(10): e0241424.

[2] Ajmera, I., Swat, M., Laibe, C., Novere, N. L., and Chelliah, V. (2013). The impact of mathematical modeling on the understanding of diabetes and related complications. *CPT: Pharmacometrics & Systems Pharmacology,* 2(7): 54.

[3] Ali, H. A., Daneshkhah, A., Boutayeb, A., and Mukandavire, Z. (2022). Examining Type 1 Diabetes Mathematical Models Using Experimental Data. *International Journal of Environmental Research and Public Health,* 19(2): 737.

[4] Aye, P. O., Adeyemo, K. A., Oke, A. S., and Omotoye, A. E. (2021). Analysis of Mathematical Model for the Dynamics of Diabetes Mellitus and Its Complications. *Applied Mathematics and Computational Intelligence,* 10(1): 57−77.

[5] Bada, O. I., Oke, A. S., Mutuku, W. N., and Aye, P. O. (2020). Analysis of the Dynamics of SI-SI-SEIR Avian Influenza A (H7N9) Epidemic Model with Re-infection. *Earthline Journal of Mathematical Sciences,* pages 43-73.

[6] Boutayeb, A. and Derouich, M. (2002). Age structured models for diabetes in East Morocco. *Mathematics and Computers in Simulation,* 58(3): 215-229.

[7] Boutayeb, A., Twizell, E. H., Achouayb, K., and Chetouani, A. (2004). A mathematical model for the burden of diabetes and its complications. *BioMedical Engineering OnLine,* 3(1): 20.

[8] Guariguata, L., Whiting, D. R., Hambleton, I., Beagley, J., Linnenkamp, U., and Shaw, J. E. (2014). Global estimates of diabetes prevalence for 2013 and projections for 2035. *Diabetes Research and Clinical Practice,* 103(2): 137-149.

[9] Karachaliou, F., Simatos, G., and Simatou, A. (2020). The Challenges in the Development of Diabetes Prevention and Care Models in Low-Income Settings. *Frontiers in Endocrinology,* 11.

[10] Khan, M. A. B., Hashim, M. J., King, J. K., Govender, R. D., Mustafa, H., and Kaabi, J. A. (2019). Epidemiology of Type 2 Diabetes – Global Burden of Disease and Forecasted Trends. *Journal of Epidemiology and Global Health,* 10(1): 107.

[11] Kimulu, A. M., Mutuku, W. N., Mwalili, S. M., Malonza, D.,

and Oke, A. S. (2022). Male Circumcision: A Means to Reduce HIV Transmission between Truckers and Female Sex Workers in Kenya. *Journal of Mathematical Analysis and Modeling,* 3(1): 50-59.

[12] Li, J., Kuang, Y., and Mason, C. C. (2006). Modeling the glucose-insulin regulatory system and ultradian insulin secretory oscillations with two explicit time delays. *Journal of Theoretical Biology,* 242(3): 722-735.

[13] Mohamed, S. F., Mwangi, M., Mutua, M. K., Kibachio, J., Hussein, A., Ndegwa, Z., Owondo, S., Asiki, G., and Kyobutungi, C. (2018). Prevalence and factors associated with pre-diabetes and diabetes mellitus in Kenya: results from a national survey. *BMC Public Health,* 18(3):1215.

[14] Nasir, H. and Daud, A. A. M. (2021). Population models of diabetes mellitus by ordinary differential equations: a review. *Mathematical Population Studies,* pages 1−33.

[15] Ngugi, H. N., Mutuku, F. M., Ndenga, B. A., Musunzaji, P. S., Mbakaya, J. O., Aswani, P., Irungu, L. W., Mukoko, D., Vulule, J., Kitron, U., and LaBeaud, A. D. (2017). Characterization and productivity profiles of Aedes aegypti (L.) breeding habitats across rural and urban landscapes in western and coastal Kenya. *Parasites & Vectors,* 10(1): 331.

[16] Ngwiri, T., Were, F., Predieri, B., Ngugi, P., and Iughetti, L. (2015). Glycemic Control in Kenyan Children and Adolescents with Type 1 Diabetes Mellitus. *International Journal of Endocrinology,* 2015: 1-7.

[17] Oke, A. (2017). Convergence of Differential Transform Method for Ordinary Differential Equations. *Journal of Advances in Mathematics and Computer Science,* 24(6): 1-17.

[18] Oke, A. S. and Bada, O. I. (2019). Analysis of the dynamics of avian influenza A (H7N9) epidemic model with re-infection. *Open Journal of Mathematical Sciences,* 3(1): 417−432.

[19] Oke, A. S., Bada, O. I., Rasaq, G., and Adodo, V. (2021). Mathematical analysis of the dynamics of COVID-19 in Africa under the influence of asymptomatic cases and re-infection. *Mathematical Methods in the Applied Sciences,* 45(1): 137-149.

[20] Oke, A. S., Fatunmbi, E. O., Animasaun, I. L., and Juma, B. A. (2022). Exploration of ternaryhybrid nanofluid experiencing Coriolis and Lorentz forces: case of three-dimensional flow of water conveying carbon nanotubes, graphene, and alumina nanoparticles. *Waves in Random and Complex Media,* pages 1−20.

[21] Otieno, P. O., Kiroro, F., Runyenje, C., and Kamau, P. (2021). Unmet need for primary healthcare and associated individual and household-level factors in Kenya: results from a national survey. *BMJ Open,* 11(5).

[22] Papatheodorou, K., Banach, M., Bekiari, E., Rizzo, M., and Edmonds, M. (2018). Complications of Diabetes 2017. *Journal of Diabetes Research,* 2018: 1-4.

[23] Regassa, L. D. and Tola, A. (2021). Magnitude and predictors of hospital admission, readmission, and length of stay among patients with type 2 diabetes at public hospitals of Eastern Ethiopia: a retrospective cohort study. *BMC Endocrine Disorders,* 21(1).

[24] World Health Organization (2016). *Global Report on*

Diabetes. WORLD HEALTH ORGN.

[25] Zafar, Z. U. A., Hussain, M. T., Inc, M., Baleanu, D., Almohsen, B., Oke, A. S., and Javeed, S. (2022). Fractional-order dynamics of human papillomavirus. *Results in Physics,* 34:105281.

[26] Zhang, J., Jia, J., and Song, X. (2014). Analysis of an SEIR Epidemic Model with Saturated Incidence and Saturated Treatment Function. *The Scientific World Journal,* 2014: 1-11.