

Mathematical Modelling of Diabetes under a Constrained Hospitalisation Resources

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Abstract

Diabetes is a chronic disease in which the body is unable to convert the excess sugar into a useable form. It is chronic disease that is fast becoming a menace in the Kenyan communities. In this study, the response of the complicated diabetic cases is examined under a constrained hospitalisation setting. The mathematical model is formulated by incorporating the carrying capacity and the per capita hospitalization rate. The models are numerically solved in MATLAB using the explicit Runge-Kutta (4, 5) technique, and the results are shown as graphs. The findings suggest that improving the quality of life in the susceptible class will result in an increase in the susceptible class and a decrease in diabetes cases. Increasing the proportion of diabetics who seek treatment each month leads to an increase in the number of people admitted to the hospital. Increasing carrying capacity reduces the number of hospitalized people.

Subject Areas

Dynamical System, Mathematics, Ordinary Differential Equation

Keywords

Diabetes, Hospitalisation, Mathematical Modelling, Constrained Hospitalisation Resources

1. Background Information

In 2014, a record of 422 million people was found to be diabetic globally. This equates to a startling 6% of the global population. However, the low-income countries had a prevalence of 7.4%, which was higher than the prevalence of

7.0% in high-income countries [1] [2]. Diabetes prevalence has risen faster in low- and middle-income countries, and it is now highest in middle-income countries (9.3 percent) [3]. Kenya, like other developing countries, is contending with an increasing diabetes epidemic. The country's diabetes prevalence is estimated to be approximately 3.3%. Unless this trend changes, this figure is expected to rise to 4.5 percent by 2025. Kenya recorded nearly 8700 diabetes-related mortality in 2015, almost all of whom were under the age of 60 [4]. Since the devolution of powers in the Kenyan 2010 Constitution, each of the 47 counties currently supervises its own health systems, including those that offer care for non-communicable diseases (NCDs). There are six levels of health services. Level 1 units provide community-based care. Level 2 facilities provide basic prenatal care, vaccines, and other essential healthcare services, Level 3 institution is a larger medical center that provides a broader variety of services, including the ability to dispense some medications and provide some basic inpatient treatment. Level 4 services are equivalent to subcounty hospitals, whilst Level 5 services are equivalent to the county referral hospital. The national referral hospitals in Nairobi are 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) [5]. Furthermore, Otieno et al. [6] reported that 68 percent of Kenyans' basic health needs are not met. Diabetes and other non-communicable diseases (NCDs) are becoming more commonly recognized as health hazards. According to the 2015-2020 National Strategy for the Prevention and Control of NCDs, diabetes is one of the four most common chronic illnesses in Kenya. The National Hospital Insurance Fund (NHIF) has also recently 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.

Boutayeb et al. [7] studied the dynamics of diabetes in both the diabetic individuals with complications and those without complicated diabetic cases and developed a mathematical model. Li et al. [8] presented a modification to the system of equations by including time-delay to model the glucose-insulin regulating mechanism. The findings corroborated previous physiological observations. Zhang et al. [9] investigated the impact of diabetes incidence rate and saturation treatment on the diabetic population trend using the SEIR mathematical model. It was concluded that raising the treatment rate can control the diabetic population's growth. Karachaliou *et al.* [10] emphasized the difficulties in preventing non-communicable illnesses in low-income settings. They developed a mathematical model for diabetes prevention and highlighted that good diabetes prevention and treatment for diabetes patients can help lessen the burden of such diseases in low-income regions. Regassa and Tola [11] investigated the impact of the frequency of admittance and readmission of diabetic patients into Ethiopian hospitals. The parametric survival analysis was utilized to forecast the hospital admission rate, which was 9.85 per 1000 people per year, as well as the readmission rate. Aye et al. [12] proposed a mathematical model for the dynamics of diabetes and solved model using the homotopy analysis method. Ali *et al.* [13] 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 David [14] harmonised mathematical models for the study of diabetes. The models include those without complications and those with different rate of complications. With the harmonisation of the models, a comprehensive qualitative analysis was carried out for the different models. Areas of further research proposed in the study include the study on the effects of constrained resources on the diabetic populations.

In this study, a mathematical model is proposed for the study of the dynamics of diabetic population in Kenya. The model considers the effect of congestion on the available health resources (such as available bed spaces, health workers, etc.) on the growth of the diabetic population in Kenya. The specific goals of this study are to:

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

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

- 3) Ascertain how the quality of lifestyle affects the management of diabetes.
- 4) Investigate how the trends of diabetes are impacted by limited resources.

2. Methodology

2.1. Formulation of the Mathematical Model

The flowchart for the mathematical model that considers the significance of constrained 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 diabetic individuals who are already diabetic, the complicated compartment includes diabetic individuals who have developed complications as a result of their diabetes, and

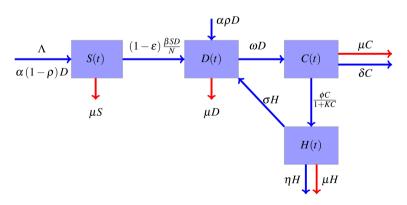


Figure 1. Flowchart for the model considering intervention.

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.

Hill [15] proposed that the number of incident that occur due to lifestyle factors is

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

where ε ($0 < \varepsilon < 1$) is the rate of lifestyle incidence ($\varepsilon = 0,1$ represents the lowest and the highest lifestyle standards respectively), β is proportion of interaction leading to incidence, 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, σ is the recovery rate from complications, η is mortality rate among the hospitalised individuals, and μ is taken as the natural mortality rate. In a constrained setting, the saturation treatment function proposed by [9] [16] 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). The governing differential equations is therefore given as

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda + \alpha \left(1 - \rho\right) D - \left(1 - \varepsilon\right) \beta \frac{SD}{N} - \mu S,\tag{1}$$

$$\frac{\mathrm{d}D}{\mathrm{d}t} = \alpha \rho D + (1 - \varepsilon) \beta \frac{SD}{N} + \sigma H - \omega D - \mu D, \qquad (2)$$

$$\frac{\mathrm{d}C}{\mathrm{d}t} = \omega D - \frac{\phi C}{1 + KC} - \delta C - \mu C,\tag{3}$$

$$\frac{\mathrm{d}H}{\mathrm{d}t} = \frac{\phi C}{1+KC} - \sigma H - \eta H - \mu H. \tag{4}$$

2.2. Qualitative Analysis

2.2.1. Equilibrium Points and Reproduction Number

The diabetes-free equilibrium (DFE) E_0 is

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

and the endemic equilibrium point (EEP) E_1 is

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

where

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

$$S^* = \frac{\Lambda + (\alpha - \omega - \mu)D^* + \sigma H^*}{\mu}.$$

The reproduction number R_0 is calculated using the next generation matrix method [17] [18] [19]. Given that *F* and *V* are the new infections and negated outward transitions from these compartments respectively, then

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

and thus

$$\left(
abla F
ight)_{E_0} \left(
abla V
ight)_{E_0}^{-1} = \left(egin{matrix} (1 - arepsilon) rac{\Lambda eta}{N \mu} & 0 \ arphi + \mu - lpha
ho & 0 \ 0 & 0 \end{array}
ight).$$

The characteristic equation is obtained thus

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

and the eigenvalues are

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

Finally, the reproduction number is

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

Theorem 2.1 (Routh Hurwitz Criterion) A polynomial

$$a_0\lambda^n + a_1\lambda^{n-1} + a_2\lambda^{n-2} + \dots + a_{n-2}\lambda^2 + a_{n-1}\lambda + a_n = 0$$

of positive coefficients $a_i > 0$ if the minors of the principal diagonal of the matrix

(a_1)	a_0	0	0	0	0		0)
a_3	a_{2}	a_1	a_0	0	0	•••	0
a_5	a_4	a_3	a_2	a_1	a_0		$\begin{bmatrix} 0\\ \vdots\\ a_n \end{bmatrix}$
:	÷	÷	÷	÷	÷	·.	:
0)	0	0	0	0	0		a_n

are all positive.

2.2.2. Local Stability of the Equilibrium Points

According to the formulations of Oke and Bada [20], the Jacobian matrix for the system (1 - 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 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 $\ 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 2.2. The DFE of system (1 - 4) is locally asymptotically stable 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

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

which evaluates to

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

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 eigenvalue $\lambda_1 = -\mu$ is negative and the other three eigenvalues can be found by solving the cubic polynomial

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

with

$$\xi_2 = A_2 + A_3 - A_1, \ \xi_1 = A_2 A_3 - A_1 A_2 - A_1 A_3, \ \xi_0 = -(A_1 A_2 A_3 + \sigma \omega \phi).$$
(5)

Routh Hurwitz criteria states that the three eigenvalues are negative if $\xi_2>0,\xi_1>0,\xi_0>0$. Hence, the conditions are:

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

condition 2: $A_2A_3 - A_1A_2 - A_1A_3 > 0$,

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

It is easy to see from the first condition that $A_2 + A_3 > A_1$ and from the second condition,

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

The last condition simply becomes,

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

therefore,

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

The DFE is asymptotically stable if $R_0 < 1$.

Theorem 2.3. The EEP of system (1 - 4) is locally asymptotically stable if $R_0 < 1$.

Proof. The Jacobian at the EEP E_1 is

$$J_{1} = \begin{pmatrix} -B_{1}D^{*} - \mu & \alpha(1-\rho) - B_{1}S^{*} & 0 & 0 \\ B_{1}D^{*} & \alpha\rho + B_{1}S^{*} - \mu - \omega & 0 & \sigma \\ 0 & \omega & -B_{2} - \delta - \mu & 0 \\ 0 & 0 & B_{2} & -\sigma - \eta - \mu \end{pmatrix}$$

where

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

The characteristic equation of J_1 is

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

which becomes

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

where

$$\begin{aligned} \xi_0 &= A_2 A_4 - \sigma \omega B_2 \left(B_1 D^* + \mu \right), \\ \xi_1 &= A_1 A_4 + A_2 A_3 - \sigma \omega B_2, \\ \xi_2 &= A_1 A_3 + A_2 + A_4, \\ \xi_3 &= A_1 + A_3, \end{aligned}$$
(6)

and

$$\begin{split} A_1 &= B_1 D^* + \mu - \alpha \rho - B_1 S^* + \mu + \omega, \\ A_2 &= - \Big(B_1 D^* + \mu \Big) \big(\mu + \omega \big) + \mu \Big(\alpha \rho + B_1 S^* \Big) + \alpha B_1 D^*, \\ A_3 &= B_2 + \delta + \mu + \sigma + \eta + \mu, \\ A_4 &= \Big(B_2 + \delta + \mu \Big) \big(\sigma + \eta + \mu \big). \end{split}$$

By Routh-Hurwitz criteria, all eigenvalues are negative if

$$\xi_{3} > 0, \quad \frac{\xi_{3}\xi_{2} - \xi_{1}}{\xi_{3}} > 0, \quad \frac{\left(\xi_{3}\xi_{2} - \xi_{1}\right)\xi_{1}}{\xi_{3}} - \xi_{3}\xi_{0} > 0, \quad \xi_{0} > 0.$$
(7)

By substituting (6) into (7), we have the four conditions as condition 1: $A_1 + A_3 > 0$

condition 2:
$$\frac{(A_1 + A_3)(A_1A_3 + A_2 + A_4) - (A_1A_4 + A_2A_3 - \sigma\omega B_2)}{A_1 + A_3} > 0$$

condition 3:
$$\frac{(A_1 + A_3)(A_1A_3 + A_2 + A_4) - (A_1A_4 + A_2A_3 - \sigma\omega B_2)}{A_1 + A_3}$$

condition 3:

$$-(A_{1}+A_{3})(A_{2}A_{4}-\sigma\omega B_{2}(B_{1}D^{*}+\mu)) > 0$$

condition 4: $A_2 A_4 - \sigma \omega B_2 (B_1 D^* + \mu) > 0$

For condition (1) to hold, then

$$B_1 D^* + \mu - \alpha \rho - B_1 S^* + \mu + \omega + B_2 + \delta + \mu + \sigma + \eta + \mu > 0$$
$$\Rightarrow -\alpha \rho - B_1 S^* > 0$$

Suppose condition (1) holds, then condition (2) can be rearranged as follows:

$$(A_{1} + A_{3})(A_{1}A_{3} + A_{2} + A_{4}) - (A_{1}A_{4} + A_{2}A_{3} - \sigma\omega B_{2}) > 0.$$
(8)

Also, rearranging condition (3) gives

$$\frac{(A_{1}+A_{3})(A_{1}A_{3}+A_{2}+A_{4})-(A_{1}A_{4}+A_{2}A_{3}-\sigma\omega B_{2})}{A_{2}A_{4}-\sigma\omega B_{2}(B_{1}D^{*}+\mu)}>(A_{1}+A_{3})^{2}>0,$$

provided

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

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

$$A_2 A_4 - \sigma \omega B_2 \left(B_1 D^* + \mu \right) > 0 \tag{9}$$

which implies that

$$A_{2}A_{4} > \sigma\omega B_{2}(B_{1}D^{*} + \mu) > 0 \Longrightarrow A_{2} > 0 \text{ since } A_{4} > 0$$
$$-(B_{1}D^{*} + \mu)(\mu + \omega) + \mu(\alpha\rho + B_{1}S^{*}) + \alpha B_{1}D^{*} > 0$$
$$\Longrightarrow \omega + \mu - \alpha\rho - B_{1}S^{*} < 0 \Longrightarrow 1 - \frac{B_{1}S^{*}}{\omega + \mu - \alpha\rho} < 0 \Longrightarrow R_{0} < 1$$

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

2.3. Positivity and Boundedness of Solution

Putting N = S + D + C + H, then

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \Lambda - \delta C - \eta H - \mu N + \alpha D \le \Lambda + \alpha D - \mu N$$

which on solving gives

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

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

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

Now, from Equations (1)-(4),

$$\begin{split} \frac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda + \alpha \left(1 - \rho\right) D - \left(1 - \varepsilon\right) \beta \frac{SD}{N} - \mu S \ge -\mu S \Longrightarrow S \ge S_0 \exp\left(-\mu t\right), \\ &\frac{\mathrm{d}D}{\mathrm{d}t} = \alpha \rho D + \left(1 - \varepsilon\right) \beta \frac{SD}{N} + \sigma H - \omega D - \mu D \ge -\left(\mu + \omega\right) D \\ &\Rightarrow D \ge D_0 \exp\left(-\left(\mu + \omega\right) t\right), \\ &\frac{\mathrm{d}C}{\mathrm{d}t} = \omega D - \frac{\phi C}{1 + KC} - \delta C - \mu C \ge -\left(\phi + \delta + \mu\right) C \\ &\Rightarrow C \ge C_0 \exp\left(-\left(\phi + \delta + \mu\right) t\right), \\ &\frac{\mathrm{d}H}{\mathrm{d}t} = \frac{\phi C}{1 + KC} - \sigma H - \eta H - \mu H \ge -\left(\sigma + \delta + \mu\right) H \\ &\Rightarrow H \ge H_0 \exp\left(-\left(\sigma + \delta + \mu\right) t\right). \end{split}$$

Thus, as long as the initial conditions are positive, then the S, D, C, H are positive in \mathcal{R} .

2.4. 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

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

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

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

The choice of the fourth order Runge-Kutta Scheme is due to its stability and large region of convergence (see [21] [22] 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 obtained from [13] [23] are chosen for the parameters as

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

3. Analysis and Discussion of Results

Figures 2(a)-(c) shows the effect of lifestyle quality on the susceptible class, diabetic class and complicated class. By increasing the lifestyle quality among the susceptible class, the number of individuals who get out of the susceptible class reduce significantly. Hence, the susceptible class increases as shown in **Figure 2(a)**, and both the diabetic class and complicated class reduce (**Figure 2(b)** and **Figure 2(c)**).

The per capita hospitalisation rate ϕ denotes the fraction of diabetics who seek treatment. Increasing ϕ results in a rise in the number of people classified as Hospitalised. 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. Figure 3(a) demonstrates that as ϕ rises, so does the population in the hospitalised class. The carrying capacity K denotes the maximum available resources in the health facility. It might reflect the maximum number of available bed spaces or the maximum number of diabetes patients who can seek care and treatment from medical practitioners. As carrying capacity grows, more people from the complicated class have access to care and treatment. This allows the number of hospitalized persons to decrease as most complicated cases receive care and treatment and return to the diabetic class, minimizing hospital congestion. Figure 3(b) illustrates that as carrying capacity increases, the hospitalised class decreases. As a result, an increase in hospital carrying capacity can control the number of hospitalized persons.

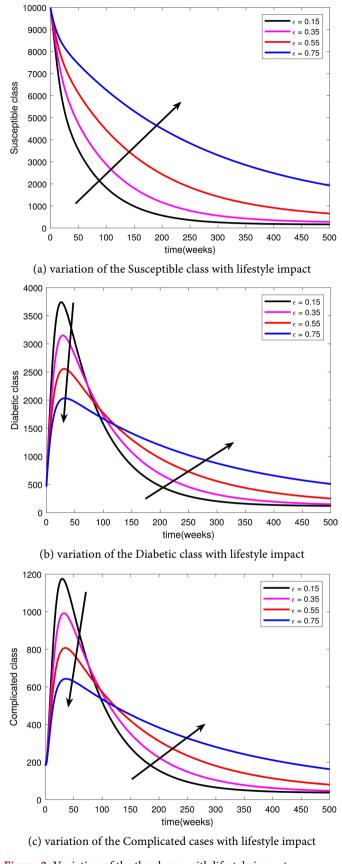
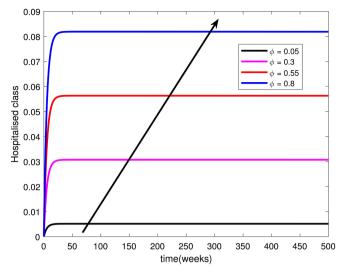
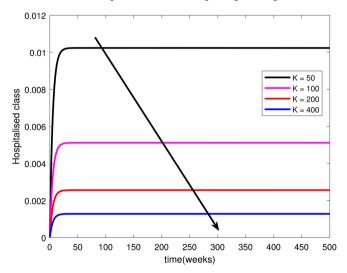


Figure 2. Variation of the the classes with lifestyle impact.



(a) variation of the Hospitalised class with per capita hospitalisation rate



(b) variation of the Hospitalised cases with hospital carrying capacity

Figure 3. Variation of hospitalised class with the per capita hospitalisation and carrying capacity.

4. Conclusions

The response of diabetic population under constrained health resources is modelled. 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:

- Increasing the lifestyle quality leads to an increase in the susceptible class but a decrease in the diabetic and complicated classes.
- Increasing the per capita hospitalisation rate leads to a rise in the Hospitalised class.
- As carrying capacity increases, the hospitalized class decreases.

From this outcome, it becomes clear that increasing the carrying capacity of the health facilities can control the number of diabetic individuals.

Conflicts of Interest

The authors declare no conflicts of interest.

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