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EPIDEMIOLOGICAL DYNAMICS OF DIABETES: MODELING APPROACHES AND STABILITY EVALUATION

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Abstract:

Diabetes is one of the most vital global health issues. It spreads rapidly all over the world's population, with the World Health Organization estimating that 415 million people worldwide have a medical condition; by 2040, that number is expected to rise to 642 million. Diabetes is known to be a condition caused by both genetic and lifestyle factors. A bad lifestyle makes a person more at risk of acquiring diabetes, and risky social interactions play a major role in poor habits. On the other hand, a genetic component is the primary cause of the genetic condition associated with diabetes. The two most common types of diabetes are Type 1 and Type 2, which are caused by decreased insulin production and decreased body responsiveness to insulin, respectively. Initial indicators of diabetes, such as increased pee, impaired vision, unexplained weight loss, and abnormal energy metabolism, are mostly caused by hyperglycemia. For the majority of diabetics, maintaining an optimal blood glucose level requires continuous treatment. Effective control is therefore essential to improving the control of diabetes. An ordinary differential equation system forms the foundation of the model. We analyze the model's endemic equilibrium, disease-free equilibrium, and reproduction number. A stability study indicates that when the equilibrium of disease-free is locally asymptotically stable [LAS], alternatively, unstable when $R_0 > 1$ .We also develop MATLAB to assist with the equations of the model. Eventually, this study will provide a comprehensive account of how diabetes complications develop following a diagnosis. The outcomes can be utilized to learn how to improve a nation's general public health, since governments should create smart and successful initiatives for diabetes screening and treatment.

Keywords: Diabetes, mathematical modeling, stability, equilibrium points, reproduction number.

1. Introduction

Diabetes is a persistent illness that mostly impacts type 1 and type 2 blood sugar levels. Type 1 diabetes, which often appears in young people, is caused by an autoimmune reaction that destroys the insulin-producing cells in the pancreas, making the use of insulin essential for the duration of one's life. On the other hand, type 2 diabetes is more common in adults but is also affecting younger populations as a result of obesity trends. It is caused by either insufficient insulin production or cellular resistance to the effects of insulin. Both forms are frequently accompanied by symptoms that indicate the need for immediate medical intervention, such as increased thirst, frequent urination, and fatigue. Diabetes can have serious side effects if it is not controlled, such as nerve damage, kidney failure, and heart disease. The goal of treatment is to stabilize blood sugar levels using prescription medication in addition to dietary and activity adjustments. Type 2 diabetes can be greatly reduced by adopting lifestyle modifications like maintaining a healthy weight and engaging in physical activity, but type 1 diabetes cannot be prevented. This

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highlights the significance of proactive health management and preventive measures in the fight against this widespread condition.

Our research aims to examined the spread of diabetes infected people and therefore we determined a mathematical model of diabetes disease$(\mathrm{D_t}, \mathrm{D_1}, \mathrm{D_2}, \mathrm{C_S})$. The four compartments are ; Diabetes people $(\mathrm{D_t})$, Type-1 $(\mathrm{D_1})$, Type-2 $(\mathrm{D_2})$,Cure or Stable $(\mathrm{C_S})$. we used matrix to prove all the Eigen values are negative, we examined a certain endemic equilibrium is asymptotically stable, and we solved the concept of locally asymptotically stable condition , This work is identified to control the infection of diabetes people, on which one can act to control the spread of the infection. The numerical reproductions are done and they explore our vague analysis. We used matrix to calculate the reproduction number $\mathrm{R_0}$, solving various parameter numerical values of the model of the provided mathematical statement using MATLAB. Following analysis, the findings indicate a definite increase in the number of indictments, an increase in the illness measure of the effect of diabetes, and a decrease in the number of indictments related to the disease measure of diabetes for mortality populations.

2. Characterization of the exemplary confines:

D_t : diabetes people

D_1 : Type 1

D_2: Type 2

C_S : Cure or Stable

ψ : The rate of diabetes infected Type 1 people

ϕ : The rate of diabetes infected Type 2 people

γ: The rate of diabetes infected Type 1 stable people

δ: The rate of diabetes infected people Type 2 cure / stable stage

μ : The rate of natural death rate

3. Model diagram

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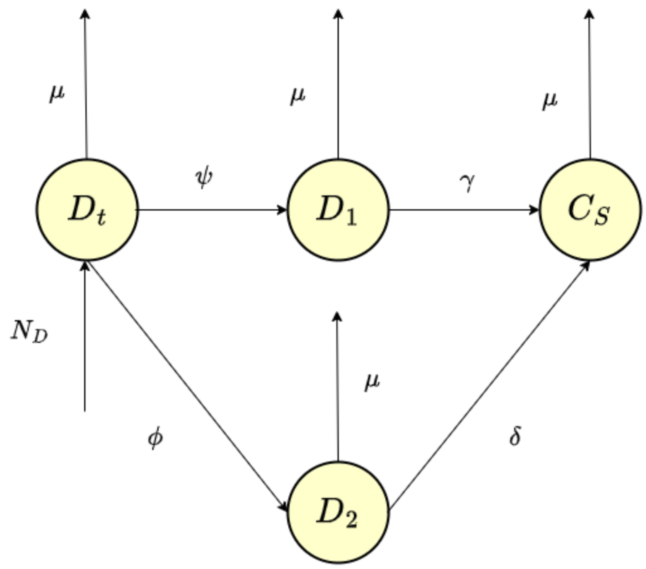

Fig.1 compartmental diagram for human population affected by diabetes

$$\frac{dD_t}{dt} = N_D - \psi D_t - \phi D_t - \mu D_t \quad (1)$$

$$\frac{dD_1}{dt} = \psi D_t - \mu D_1 - \gamma D_1 \quad (2)$$

$$\frac{dD_2}{dt} = \phi D_t - \mu D_2 - \delta D_2 \quad (3)$$

$$\frac{dC_S}{dt} = \delta D_2 - \mu C_S + \gamma D_1 \quad (4)$$

Subject to initial conditions below

$D_t(0) \geq 0, D_1(0) \geq 0, D_2(0) \geq 0, C_S(0) \geq 0$

4. Disease free equilibrium

We assume that the lack of the substance does not harm every mortal person; hence, the population is immune to the infection.

$$\frac{dD_t}{dt} = N_D - \psi D_t - \phi D_t - \mu D_t = 0$$

$$D_t = \frac{N_D}{(\psi + \phi + \mu)}$$

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$$\left(\frac{N_D}{(\psi+\phi+\mu)},0,0,0\right)$$

Endemic equilibrium point:

$$D_t(\psi+\phi+\mu)-N_D=0$$

$$D_t^*=0$$

The complimentary equilibrium defect is expressed as follows:

$$D_1=\frac{\psi D_t}{(\mu+\gamma)}$$

In equation D_1 Replace D_t,

$$D_1^*=\frac{\psi\left(\frac{N_D}{\psi+\phi+\mu}\right)}{(\mu+\gamma)}$$

$$D_2=\frac{\phi D_t}{(\mu+\delta)}$$

In equation D_2 Replace D_t,

$$D_2^*=\frac{\phi\left(\frac{N_D}{\psi+\phi+\mu}\right)}{(\mu+\delta)}$$

$$C_S=\frac{\delta D_2+\gamma D_1}{\mu}$$

In equation C_S Replace D_2 and D_1,

$$C_S^*=\frac{\delta\left(\frac{\phi D_t}{\mu+\delta}\right)+\gamma\left(\frac{\psi D_t}{\mu+\gamma}\right)}{\mu}$$

Hence the endemic equilibrium points are$(D_t^*, D_1^*, D_2^*, C_S^*)$

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$$D_t^* = 0, D_1^* = \frac{\psi\left(\frac{N_D}{\psi+\phi+\mu}\right)}{(\mu+\gamma)}, D_2^* = \frac{\phi\left(\frac{N_D}{\psi+\phi+\mu}\right)}{(\mu+\delta)}, C_S^* = \frac{\delta\left(\frac{\phi D_t}{\mu+\delta}\right)+\gamma\left(\frac{\psi D_t}{\mu+\gamma}\right)}{\mu}$$

D_t are affected cases, we find out reproduction number R_0,let $\mathrm{x} = (\mathrm{D_t}, \mathrm{D_1}, \mathrm{D_2}, \mathrm{C_S})$, F_R be the indication of a modern disease entering the system and $\mathrm{V_R}$the indication of a sickness leaving the structure, the following will serve as an instance:

The above cases infectious classes is $\mathrm{D_1}$

$$F_R = \begin{bmatrix} \psi \mathrm{D_t} \\ 0 \\ 0 \\ 0 \end{bmatrix} \text{and} \quad V_R = \begin{bmatrix} (\mu+\gamma)D_1 \\ (\psi+\phi+\mu) \\ \mu+\delta \\ \mu \end{bmatrix} \frac{dD_1}{dt} = \psi \mathrm{D_t} - \mu \mathrm{D_1} - \gamma \mathrm{D_1}$$

The derivatives of $F_R$and $V_R$are given by $F_R = \psi$, and $V_R = (\mu+\gamma)$Respectively

The inverse of $V_R$is given by $V_R^{-1} = \frac{1}{(\mu+\gamma)}$

So, a values of $F_R V_R^{-1}$gives the well-known basic reproduction number:

$$R_0(D_1) = \frac{\psi}{(\mu+\gamma)}$$

5. Stability of the system

5.1 Local stability of disease free equilibrium

If $R_0 < 1$, The equilibrium free from disease is either locally asymptotically stable or unstable, if $R_0 > 1$.

The model's Jacobian matrix can be found using;

$$\mathrm{J} = \begin{bmatrix} -(\psi+\phi+\mu) & 0 & 0 & 0 \\ \psi & -(\gamma+\mu) & 0 & 0 \\ \phi & 0 & -(\delta+\mu) & 0 \\ 0 & 0 & \delta & -\mu \end{bmatrix}$$

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$$D_t = D_1 = D_2 = C_S = 0$$

Computing the Jacobian matrix yields the determinant of disease-free equilibrium;

$|\mathrm{J} - \lambda I| = 0$, where λ is the Eigen values.

$$\mathrm{J} = \begin{bmatrix} -(\psi + \phi + \mu) - \lambda & 0 & 0 & 0 \\ \psi & -(\gamma + \mu) - \lambda & 0 & 0 \\ \phi & 0 & -(\delta + \mu) - \lambda & 0 \\ 0 & 0 & \delta & -\mu - \lambda \end{bmatrix}$$

Four negative Eigen values are obtained here, and the DFE is asymptotically stable locally.

5.2 Global Stability of Endemic Equilibrium

If , $R_0 > 1$After that, the equilibrium of the disease endemic will be asymptotically stable.
We shall demonstrate the following using the Lyapunov function:

$DL = (D_t^*, D_1^*, D_2^*, C_S^*)$

$$\left(D_t - D_t^* - D_t In\frac{D_t^*}{D_t^*}\right) + \left(D_1 - D_1^* - D_1 In\frac{D_1^*}{D_1^*}\right) + \left(D_2 - D_2^* - D_2 In\frac{D_2^*}{D_2^*}\right) + \left(C_S - C_S^* - C_S In\frac{C_S^*}{C_S^*}\right)$$

Computing the derivative of DL , we get

$$\frac{DdL}{dt} = \left(\left(\frac{D_t - D_t^*}{D_t}\right)\frac{dD_t}{dt} + \left(\frac{D_1 - D_1^*}{D_1}\right)\frac{dD_1}{dt} + \left(\frac{D_2 - D_2^*}{D_2}\right)\frac{dD_2}{dt} + \left(\frac{C_S - C_S^*}{C_S}\right)\frac{dC_S}{dt}\right)$$

Substituting our model equation in $\frac{DdL}{dt}$above we get

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$$\frac{DdL}{dt} = \left(\left(\frac{D_t - D_t^*}{D_t}\right)(\mathrm{N_D} - \psi \mathrm{D_t} - \phi \mathrm{D_t} - \mu \mathrm{D_t}) + \left(\frac{D_1 - D_1^*}{D_1}\right)(\psi \mathrm{D_t} - \mu \mathrm{D_1} - \gamma \mathrm{D_1}) + \left(\frac{D_2 - D_2^*}{D_2}\right)(\phi D_t - \mu D_2 - \delta D_2) + \left(\frac{C_S - C_S^*}{C_S}\right)(\delta D_2 - \mu C_S + \gamma D_2)\right)$$

Here consider A is negative and B non negative values. Then $\frac{DdL}{dt}$ =A-B

A=$(-\psi - \phi - \mu)D_t^* + (-\mu - \gamma)D_1^* + (-\mu - \delta)D_2^* + (-\mu)C_S^*$

A=$-(\psi + \phi + \mu)D_t^* - (\mu + \gamma)D_1^* - (\mu + \delta)D_2^* - (\mu)C_S^*$

B=$N_D\left(\frac{D_t^*}{D_t}\right) + \psi D_t\left(\frac{D_1^*}{D_1}\right) + \phi D_t\left(\frac{D_2^*}{D_2}\right) + (\delta + \gamma)D_2\left(\frac{C_S^*}{C_S}\right)$

If A<B then $\frac{DdL}{dt} \leq 0$, $\frac{DdL}{dt} = 0$ if and only if

$$D_t = D_t^* = D_1 = D_1^* = D_2 = D_2^* = C_S = C_S^*$$

$(D_t, D_1, D_2, C_S) \in \alpha \frac{DdL}{dt} = 0$

We prove the asymptotic stability of the endemic equilibrium result.

6. Numerical reproduction

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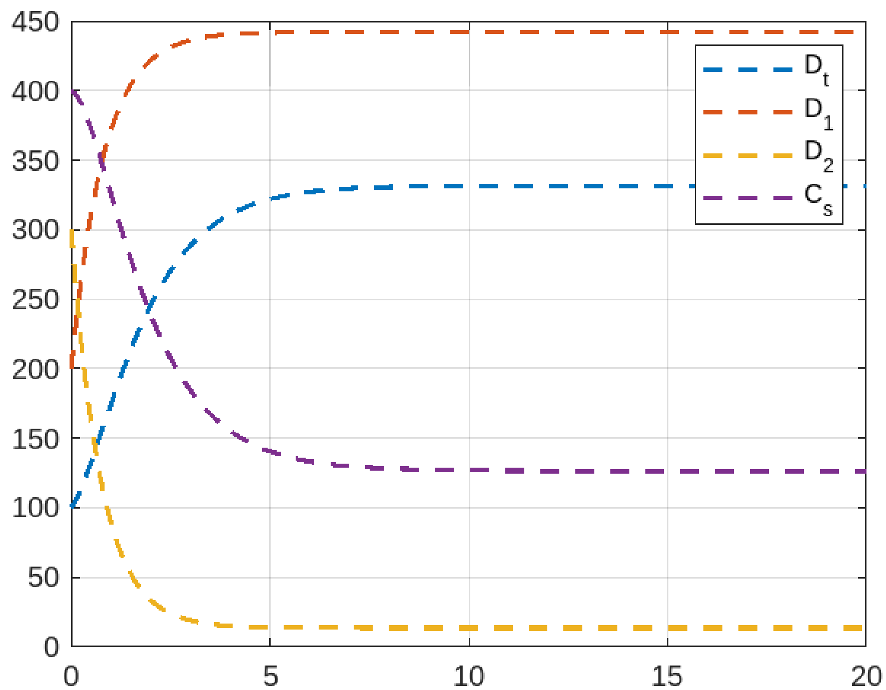

Fig.2 stability analysis of diabetes population when $N_D = 10$

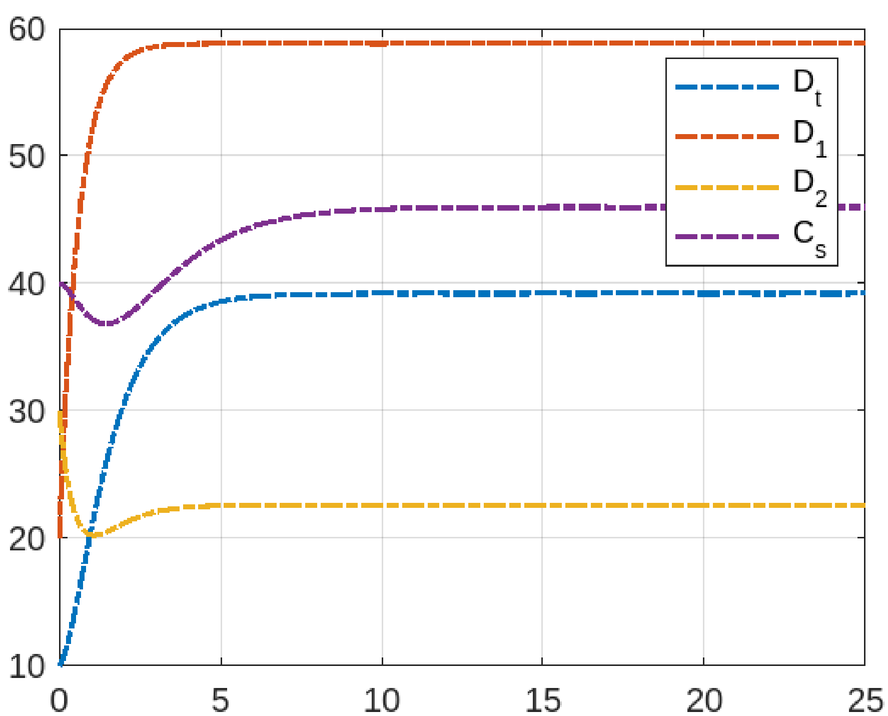

Fig.3 stability analysis of diabetes population when $N_D = 100$

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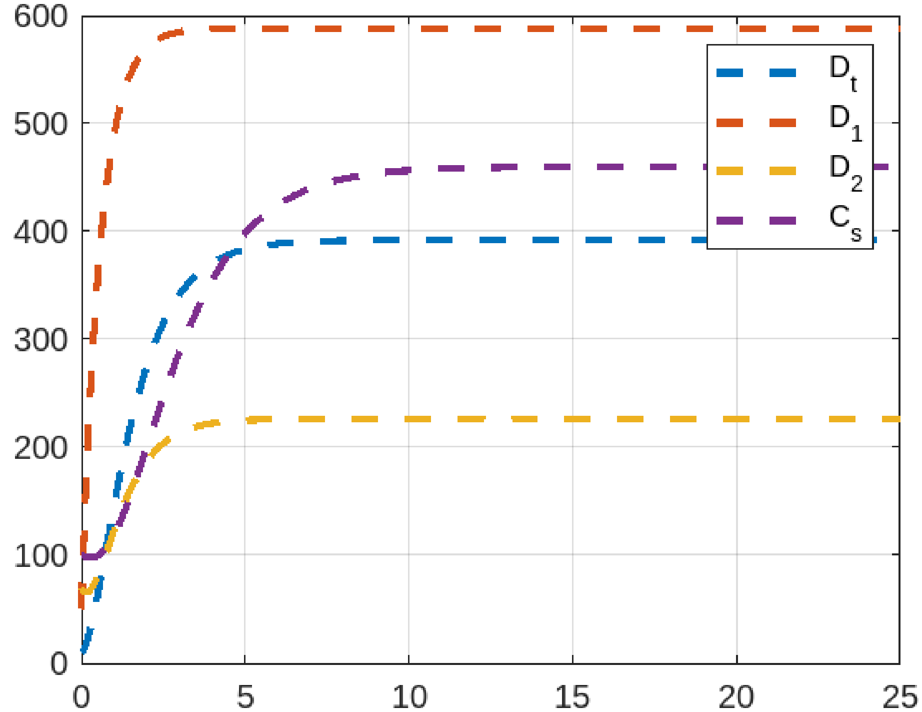

Fig.4 stability analysis of diabetes people when $N_D = 1000$

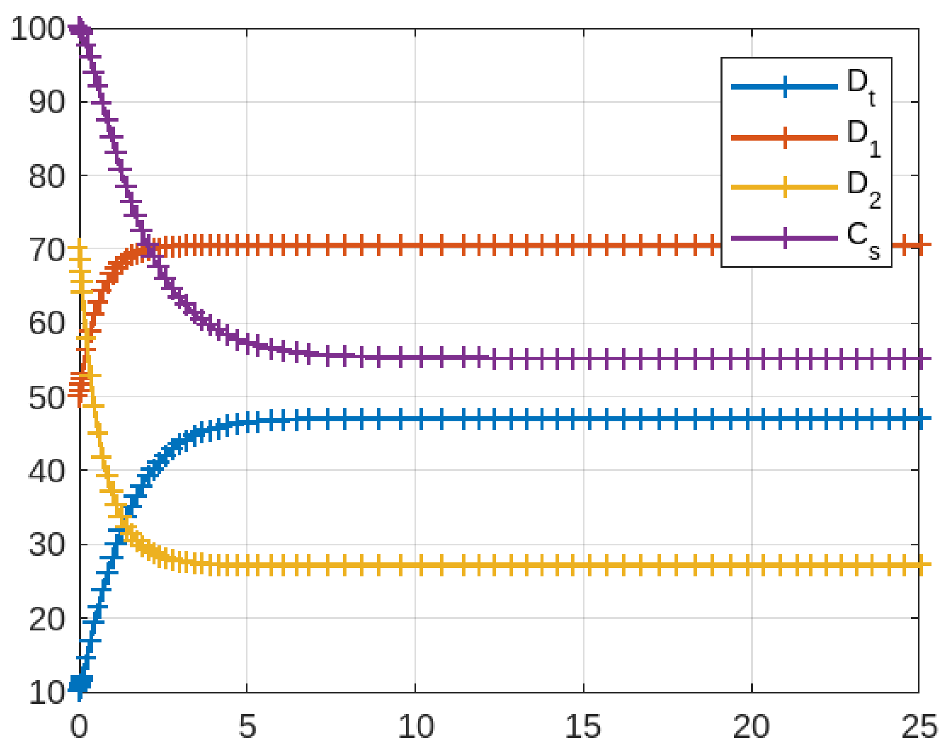

Fig.5 stability analysis of diabetes people when $N_D = 10,000$

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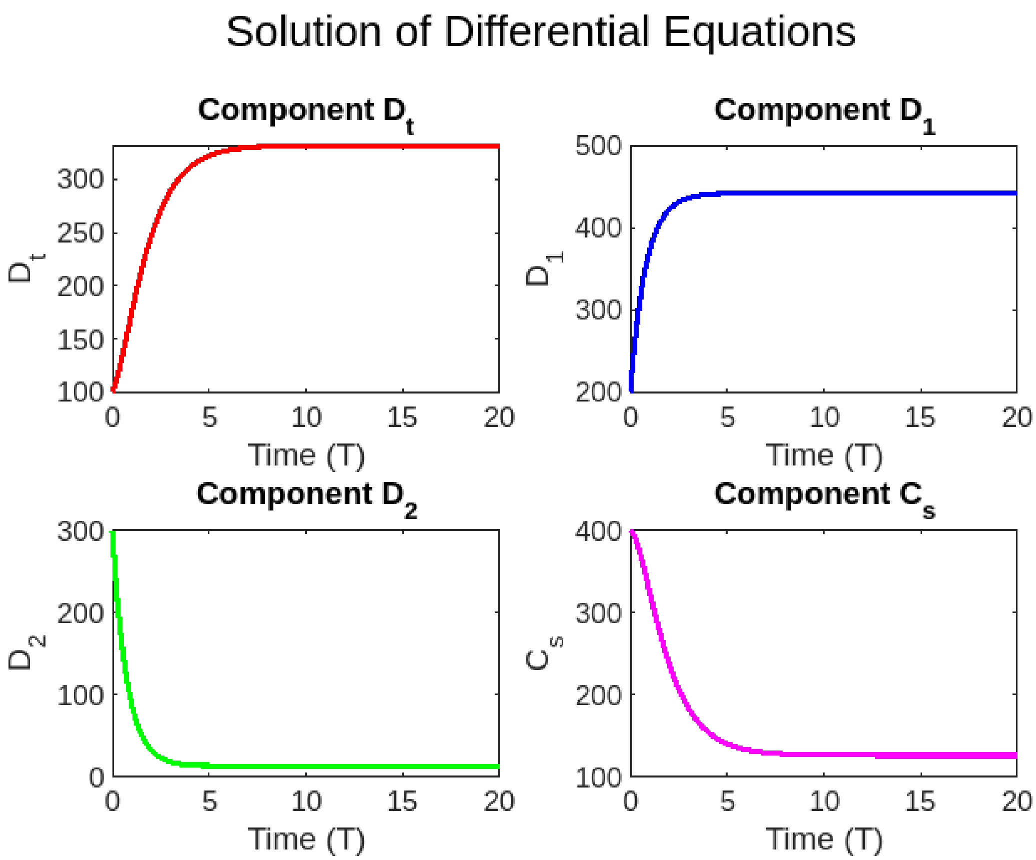

Fig.6 Comprehensive analysis of differential equations

In this paper, we utilize distinct numerical values for Fig. 2 to Fig. 5 to ensure the stabilization of all parameters. Fig. 6 illustrates the solution of the differential equation. The results of a stability analysis of the mathematical model for diabetes within the framework of dynamic systems theory are presented in this research. We find that the DFE is locally asymptotically stable, the endemic equilibrium is asymptotically stable, and the reproduction number R_0 represents the disease equilibrium. We also show that all of the eigenvalues in the matrix are negative. The disease-endemic equilibrium that is asymptotically stable will be held to if $R_0 < 1$. We showed that the Lyapunov function is used to obtain the parameters of the human populations affected by diabetes at random values.

7. Conclusions

This study delves into the dynamic modeling and stability analysis of diabetes epidemiology using mathematical frameworks. Our methodology entails delineating the Disease-Free Equilibrium (DFE) to characterize uninfected individuals, alongside deriving the endemic equilibrium point. Leveraging the Jacobian matrix, we elucidate the asymptotic stability of diabetic populations, supplemented by the establishment of global stability conditions through Lyapunov functions. MATLAB simulations, based on random data inputs, further illustrate the dynamics of diabetes infection. Diagrammatic representations in Figures 2-6 offer visual clarity on the model equations. Ultimately, this research contributes valuable insights into comprehending and addressing the complexities of diabetes epidemiology dynamics.

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