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Mathematical Study of Diabetes and its Complication Using the Homotopy Perturbation Method

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Abstract

In this work, we carried out a mathematical study of diabetes and its complications. A deterministic mathematical model of the Diabetes Mellitus disease was presented. The model equations were solved using the Homotopy Perturbation Method. Graphs were generated from the results obtained using Maple software. It was observed that the parameters involved play a crucial role in the size of population of diabetics and the number of diabetics with complications at time t.

Key words and phrases: Diabetes, Mathematical Model, Homotopy Perturbation.
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1 Introduction

Diabetes Mellitus is simply caused by the failure of the body to produce the right amount of insulin to stabilize the amount of sugar in the body [1].

Most patients who suffer this type of body failure are recommended to take insulin injection. This is called diabetes type I. Diabetes type II is the patient's body rejection to insulin. This type of patient is recommended to undergo a certain health meal program as well as performing exercises to lose weight in addition of oral medication. However, heart diseases are likely to strike these patients in the long run [2].

Gestational Diabetes can occur temporarily during Pregnancy which is due to the hormonal changes and usually begins in the fifth or sixth month of pregnancy (between the 24th and 28th weeks). Gestational Diabetes usually resolves once the baby is born. However, 25-50 % of women with gestational diabetes will eventually develop diabetes later in their life, especially those who require insulin during pregnancy and those who are overweight after their delivery [3].

Sharief and Sheta [3] enhanced the detection of diabetic by using a set of attributes collected from the patients to develop a mathematical model using Multigene Symbolic Regression Genetic Programming technique. Genetic Programming (GP) showed significant advantages on evolving nonlinear model which can be used for prediction. The developed GP model was evaluated using Pima Indian data set and showed higher capability and accuracy in detection and diagnosis of Diabetes.

Rosado [4] presented a mathematical model that determines diabetes in patients based on the results of the 5-hour glucose intolerance test. Their model extended the one proposed by Ackerman [5] to include three instead of two hormone concentrations. In particular, they included concentrations for glucose, glucagon and a global variable that includes other hormones such as insulin. The model was based on a 3x3 system of non-homogenous ordinary differential equations. A nonlinear least square method was used to determine the coefficient parameters of the system based on actual data from the Glucose Tolerance Test. The simulations also provide an indicator similar to the one proposed by Ackerman [5], to diagnose a diabetic condition. Additionally, they developed a graphical user interface to facilitate keying the

patient's data and the visualization of the results.

De Gaetano et al [6] formulated a model of the pancreatic islet compensation, its physiological assumptions were presented, some fundamental qualitative characteristics of its solutions were established, the numerical values assigned to its parameters were extensively discussed (also with reference to available cross-sectional epidemiologic data), and its performance over the span of a lifetime was simulated under various conditions, including worsening insulin resistance and primary replication defects. The differences with respect to two previously proposed models of diabetes progression were highlighted, and therefore, the model was proposed as a realistic, robust description of the evolution of the compensation of the glucose-insulin system in healthy and diabetic individuals.

Boutayeb et al [7] used ordinary differential equations and numerical approximations to monitor the size of populations of diabetes with and without complications. They discussed different scenarios according to a set of parameters and the dynamical evolution of the population from the stage of diabetes to the stage of diabetes with complications was clearly illustrated. Their model shows how efficient and cost-effective strategies can be obtained by acting on diabetes incidence and/or controlling the evolution to the stage of complications.

Fundamentals of Homotopy Perturbation Method (HPM), first proposed by Ji Huan [8,9], has successfully been applied to solve many types of linear and nonlinear functional equations. This method, which is a combination of homotopy in topology and classic perturbation techniques, provides a convenient way to obtain analytic or approximate solutions for a wide variety of problems arising in different fields.

The HPM used by He to solve the Lighthill equation [8], the Duffing equation [10] and the Blasius equation [11], found its way into sciences and has been used to solve nonlinear wave equations [12], boundary value problems [13, 14], quadratic Riccati differential equations [15], integral equations [16, 17, 18], Klein–Gordon and sine–Gordon equations [19, 20], initial value problems [21, 22], Schrödinger equation [23], Emden–Fowler type equations [24], nonlinear revolution equations [25], differential-difference equations [26], modified KdV equations [27] and many other problems. This wide variety of applications

shows the power of HPM in solving functional equations.
In this study we extended the work of Boutayeb [7] by carrying out a mathematical study of diabetes and its complication using the Homotopy perturbation method.

2 The Mathematical Model

Following Boutayeb et al [7], the mathematical equation describing the dynamics of diabetes and its complications is given by the ordinary differential equations (ODEs).

$$\left.\begin{array}{l} \frac{dD}{dt} = I - (\lambda + \mu) D + \gamma C \\ \frac{dC}{dt} = I + \lambda D - (\gamma + \mu + \nu + \delta) C \end{array}\right\} \quad (2.1)$$

$D = D(t)$ represents the number of diabetics without complications.
$C = C(t)$ represents the number of diabetics with complications.
$I = I(t)$ denotes the incidence of Diabetes Mellitus.
μ represents natural mortality rate.
λ represents the probability of a diabetic person developing a complication.
γ represents the rate at which complications are cured.
ν represents the rate at which diabetic patients with complication become severely disabled.
δ represents the mortality rate due to complications.
$N = N(t) = C(t) + D(t)$ denotes the size of population of diabetics at time t.
With $N = D + C$, we obtained

$$\left.\begin{array}{l} \frac{dC}{dt} = -(\lambda + \theta) C + \lambda N, t > 0 \\ \frac{dN}{dt} = I - (\nu + \delta) C - \mu N \end{array}\right\} \quad (2.2)$$

where $\theta = \gamma + \mu + \nu + \delta$
with initial conditions

$$C(0) = C_0, \; N(0) = N_0 \quad (2.3)$$

3 Solution by the Homotopy Perturbation Method

To illustrate the basic ideas of the method, He [9] considered the following nonlinear differential equation:

$$A(u) - f(r) = 0, r \in \Omega \qquad (3.1)$$

subject to the boundary condition:

$$B(u, \frac{du}{dn}) = 0, r \in \Gamma \qquad (3.2)$$

where $\boldsymbol{A}$ is a general differential operator, B is a boundary operator, $f(r)$ is a known analytical function and Γ is the boundary of the domain Ω. The operator $\boldsymbol{A}$ can be divided into two parts $\boldsymbol{L}$ and $\boldsymbol{N}$, where $\boldsymbol{L}$ is the linear part, and $\boldsymbol{N}$ is the nonlinear component. Equation (3.1) may therefore be rewritten as:

$$L(u) + N(u) - f(r) = 0, r \in \Omega \qquad (3.3)$$

The homotopy perturbation structure is shown as follows

$$H(v, p) = (1 - p)[L(v) - L\, u_0] + p[A(v) - f(r)] = 0 \qquad (3.4)$$

where

$$v(r, p) : \Omega \to R \qquad (3.5)$$

In equation (3.4), $p \in [0, 1]$ is an embedding parameter and u_0 is the first approximation that satisfies the boundary condition. It can be assumed that the solution of equation (3.4) can be written as power series as follows:

$$v = v_0 + pv_1 + pv_2 + ... \qquad (3.6)$$

The best approximation for the solution is:

$$u = \lim_{p \to 1} v = v_0 + v_1 + v_2 + ... \qquad (3.7)$$

The series (3.6) is convergent for most cases; however, the convergence rate depends on the nonlinear operator $A(v)$ [9].

From (2.2)

$$\frac{dC}{dt} = -(\lambda + \theta) C + \lambda N \qquad (3.8)$$

$$\frac{dN}{dt} = I - (\nu + \delta) C - \mu N \qquad (3.9)$$

Applying homotopy perturbation to equation (3.8) and (3.9) with

$$q = \lambda + \theta$$

m $= \nu + \delta$
we obtained,

$$(1-p)\frac{dC}{dt} + P\left(\frac{dC}{dt} + qC - \lambda N\right) = 0 \quad (3.10)$$

$$(1-p)\frac{dN}{dt} + P\left(\frac{dN}{dt} - I + mC + \mu N\right) = 0 \quad (3.11)$$

i.e.,

$$\frac{dC}{dt} + P(qC - \lambda N) = 0 \qquad (3.12)$$

$$\frac{dN}{dt} + p(-I + mC + \mu N) = 0 \qquad (3.13)$$

Let

$$\left.\begin{array}{l} C = C_0 + pC_1 + P^2C_2 + \ldots \\ N = N_0 + pN_1 + P^2N_2 + \ldots \end{array}\right\} \qquad (3.14)$$

$$\left.\begin{array}{l} \frac{dN}{dt} = \frac{dC_0}{dt} + \frac{dC_1}{dt} + P^2\frac{dC_2}{dt} + \ldots \\ \frac{dN}{dt} = \frac{dN_0}{dt} + \frac{dN_1}{dt} + P^2\frac{dN_2}{dt} + \ldots \end{array}\right\} \quad (3.15)$$

Substituting (3.14) and (3.15 into equations (3.12) and (3.13), we have

$$\frac{dC_0}{dt} + p\frac{dC_1}{dt} + p^2\frac{dC_2}{dt}$$

$$+p\left[\begin{array}{c} q(C_0 + pC_1 + P^2C_2) \\ +\lambda(N_0 + pN_1 + p^2N_2) \end{array}\right] = 0 \quad (3.16)$$

$$\frac{dN_0}{dt} + p\frac{dN_1}{dt} + p^2\frac{dN_2}{dt}$$

$$+p\left[\begin{array}{c} -I + m\left(C_0 + pC_1 + p^2C_2\right) \\ +\mu\left(N_0 + pN_1 + p^2N_2\right) \end{array}\right] = 0 \qquad (3.17)$$

$$P^0 : \frac{dC_0}{dt} = 0, C_0\left(0\right) = C \qquad (3.18)$$

$$p^1 : \frac{dC_1}{dt} + qC_0 - \lambda N_0 = 0,\ C_1\left(0\right) = 0 \qquad (3.19)$$

$$P^2 : \frac{dC_2}{dt} + qC_1 - \lambda N_1 = 0, C_2\left(0\right) = 0 \qquad (3.20)$$

$$P^0 : \frac{dN_0}{dt} = 0, N_0\left(0\right) = N_0 \qquad (3.21)$$

$$P^1 : \frac{dN_1}{dt} - I + mC_0 + \mu N_0 = 0, N_1\left(0\right) = 0 \qquad (3.22)$$

$$p^2 : \frac{dN_2}{dt} + mC_1 + \mu N_1 = 0, N_2\left(0\right) = 0 \qquad (3.23)$$

From equation (3.18) we get

$$C_0\left(t\right) = C_0.$$

Similarly from equation (3.21) we get

$$N_0\left(t\right) = N_0.$$

From equation (3.19)

$$\frac{dC_1}{dt} = -qC_0 + \lambda N_0.$$

Integrating with respect to t, we have

$$C_1\left(t\right) = \left(-qC_0 + \lambda N_0\right)t + K_1$$

$$C_1\left(0\right) = 0 + K_1 = 0.$$

This implies

$$K_1 = 0.$$

Therefore

$$C_1(t) = (-qC_0 + \lambda N_0)\, t;$$

i.e.,

$$C_1(t) = \alpha t,$$

where

$$\alpha = -qC_0 + \lambda N_0.$$

From equation (3.22)

$$\frac{dN_1}{dt} = I - mC_0 - \mu N_0.$$

Integrating with respect to t, we have

$$N_1(t) = (I - mC_0 - \mu N_0)\, t + K_2\ .$$

At $t = 0$

$$N_1(0) = 0 + K_2 = 0.$$

This implies

$$K_2 = 0.$$

Therefore

$$N_1(t) = (I - mC_0 - \mu N_0)\, t;$$

i.e.,

$$N_1(t) = \beta t,$$

where

$$\beta = I - mC_0 - \mu N_0.$$

From equation (3.20)

$$\frac{dC_2}{dt} + qC_1 - \lambda N_1 = 0$$

$$\frac{dC_2}{dt} = -q\alpha t + \lambda \beta t.$$

Integrating with respect to t, we have

$$C_2(t) = -\frac{1}{2}q\alpha t^2 + \frac{1}{2}\lambda\beta t^2 + K_3.$$

At $t = 0$

$$C_2(0) = 0 + K_3 = 0.$$

This implies

$$K_3 = 0$$

Therefore,

$$C_2(t) = \frac{1}{2}(\lambda\beta - q\alpha)\, t^2.$$

From equation (3.23)

$$\frac{dN_2}{dt} + mC_1 + \mu N_1 = 0$$

$$\frac{dN_2}{dt} = -mC_1 - \mu N_1$$

$$\frac{dN_2}{dt} = -m\alpha t - \mu\beta t.$$

Integrating with respect to t, we have

$$N_2(t) = \frac{1}{2}(-m\alpha - \mu\beta)\, t^2 + K_4.$$

At $t = 0$

$$N_2(0) = 0 + K_4 = 0$$

This implies

$$K_4 = 0.$$

Therefore

$$N_2(t) = \frac{1}{2}(-m\alpha - \mu\beta)\, t^2.$$

According to Homotopy Perturbation

$$C(t) = \lim_{p\to 1} C_0\ (t) + P^1 C_1(t) + P^2 C_2(t)$$

$$C(t) = C_0\ (t) + C_1(t) + C_2(t)$$

i.e.,

$$C(t) = C_0\ + \alpha t + \frac{1}{2}(\lambda\beta - q\alpha)\, t^2$$

$$N(t) = \lim_{p\to 1} N_0\ (t) + P^1 N_1(t) + P^2 N_2(t)$$

$N(t) = N_0\ (t) + N_1(t) + N_2(t)$ i.e.,

$$N(t) = N_0\ + \beta t + \frac{1}{2}(-m\alpha - \mu\beta)\, t^2.$$

Hence the solution of equation (2.2) is given by

$$C(t) = C_0\ + \alpha t + \frac{1}{2}(\lambda\beta - q\alpha)\, t^2$$

and

$$N(t) = N_0\ + \beta t + \frac{1}{2}(-m\alpha - \mu\beta)\, t^2,$$

where

$$\alpha = (-qC_0 + \lambda N_0),$$

$$\beta = (I - mC_0 - \mu N_0),$$

$$\theta = \gamma + \mu + \nu + \delta,$$

$q = \lambda + \theta$ and
$m = \nu + \delta$.

4 Results

In this section, we used a mathematical software (Maple 16) to carry out the numerical simulations of $\boldsymbol{N(t)}$ and $\boldsymbol{C(t)}$ and the results are presented below in graphical form.

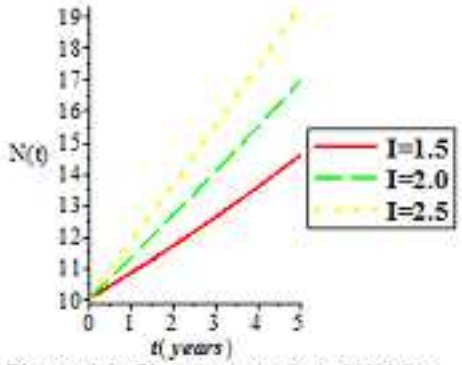

Figure 4.1: Showing graphs of $N(t)$ for different values of I when $\nu=0.05$, $\delta=0.05, \mu=0.02, \gamma=0.08, \lambda=0.02,$

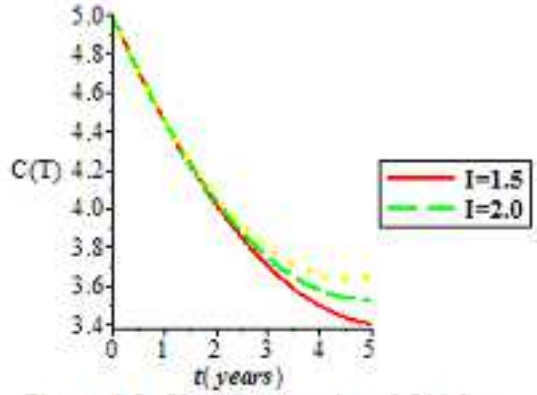

Figure 4.2: Showing graphs of C(t) for different values of I when $\nu=0.05$, $\delta=0.05, \mu=0.02, \gamma=0.08, \lambda=0.02,$

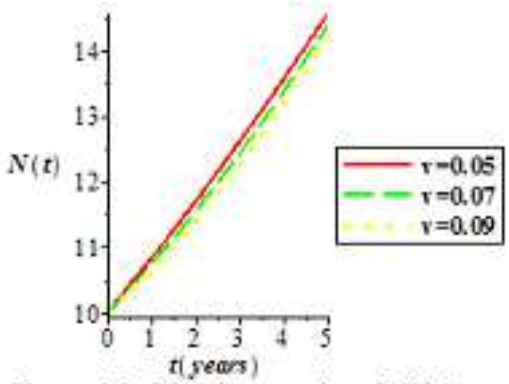

Figure 4.3: Showing graphs of *N(t)* for different values of *v* when $I = 1.5$, $\delta = 0.05$, $\mu = 0.02$, $\gamma = 0.08$, $\lambda = 0.02$,

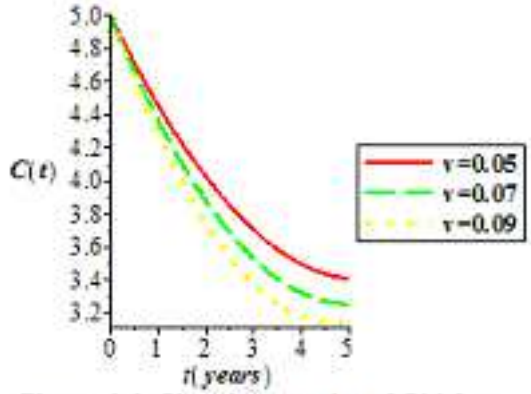

Figure 4.4: Showing graphs of C(t) for different values of *v* when $I = 1.5$, $\delta = 0.05$, $\mu = 0.02$, $\gamma = 0.08$, $\lambda = 0.02$,

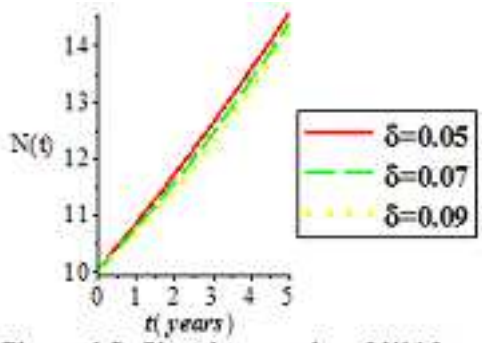

Figure 4.5: Showing graphs of *N(t)* for different values of δ when $I = 1.5$, $\nu = 0.05$, $\mu = 0.02$, $\gamma = 0.08$, $\lambda = 0.02$

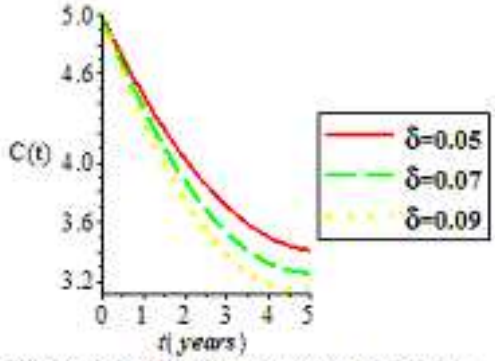

Figure 4.6: Showing graphs of *C(t)* for different values of δ when $I = 1.5$, $\nu = 0.05$, $\mu = 0.02$, $\gamma = 0.08$, $\lambda = 0.02$

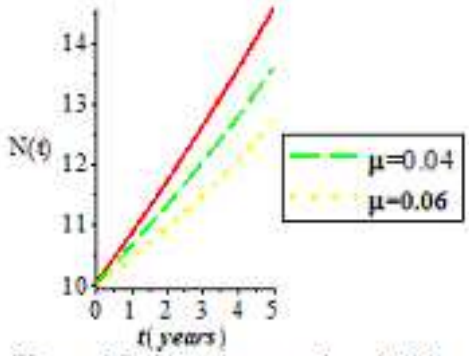

Figure 4.7: Showing graphs of *N(t)* for different values of μ when $I = 1.5$, $\nu = 0.05$, $\delta = 0.05$, $\gamma = 0.08$, $\lambda = 0.02$

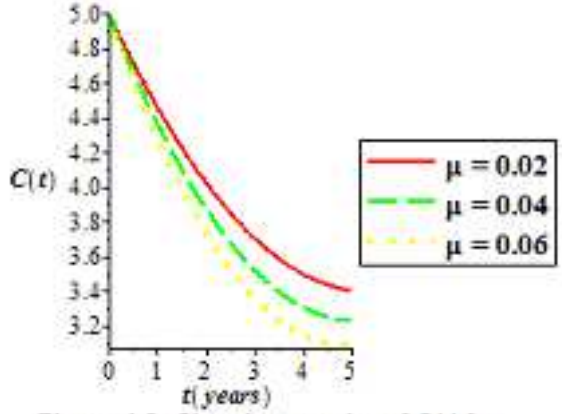

Figure 4.8: Showing graphs of *C(t)* for different values of μ when $I = 1.5$, $\nu = 0.05$, $\delta = 0.05$, $\gamma = 0.08$, $\lambda = 0.02$

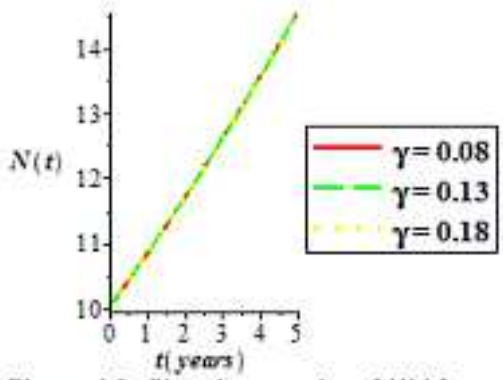

Figure 4.9: Showing graphs of *N(t)* for different values of γ when *I* = 1.5, *v* = 0.05, δ = 0.05, μ = 0.02, λ = 0.02

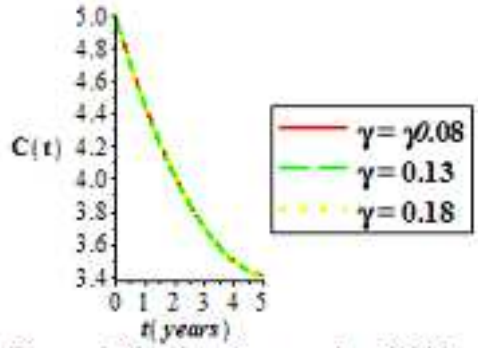

Figure 4.10: Showing graphs of *C(t)* for different values of γ when *I* = 1.5, *v* = 0.05, δ = 0.05, μ = 0.02, λ = 0.02

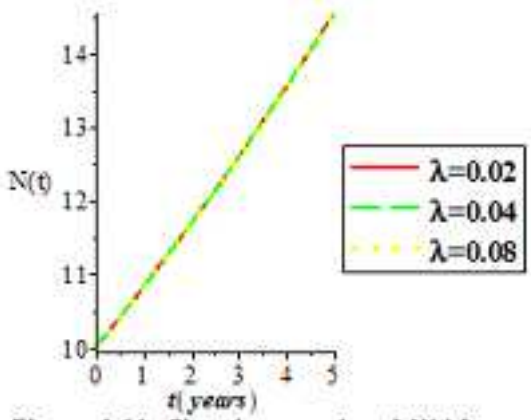

Figure 4.11: Showing graphs of *N(t)* for different values of λ when $I = 1.5$, $\nu = 0.05$, $\delta = 0.05$, $\mu = 0.02$, $\gamma = 0.08$

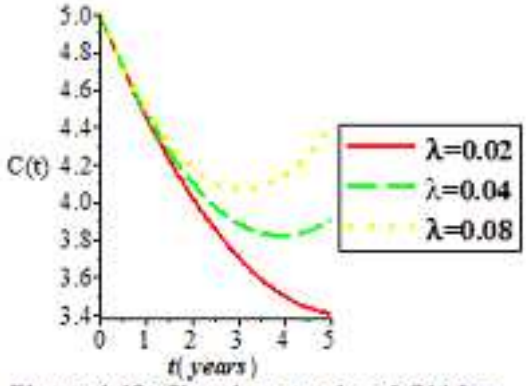

Figure 4.12: Showing graphs of *C(t)* for different values of λ when $I = 1.5$, $\nu = 0.05$, $\delta = 0.05$, $\mu = 0.02$, $\gamma = 0.08$

5 Discussion of Results, Conclusion, and Recommendations

5.1 Discussion of Results

Figure 4.1 and 4.2 show the graphs of ***N(t)*** and ***C(t)*** against t respectively for different values of I. It was observed that the number of diabetics increases faster as the incidence of Diabetes Mellitus increases while diabetics with complications decreases faster as the incidence of Diabetes Mellitus decreases.

Figure 4.3 and 4.4 display the graphs of ***N(t)*** and ***C(t)*** against t respectively for different values of υ. It was observed that the number of diabetics increases slightly faster as the rate at which diabetic patients with complications become severely disabled increases. while diabetics with complications decreases faster as the incidence of Diabetes Mellitus increases.

Figure 4.5 and 4.6 display the graph of ***N(t)*** and ***C(t)*** against t respectively for different values of δ. It was observed that the number of diabetics increases slightly faster as the mortality rate due to complications decreases. while diabetics with complications decreases faster as the mortality rate due to complications increases

Figure 4.7 and 4.8 display the graph of ***N(t)*** and ***C(t)*** respectively against t for different values of μ. It was observed that ***N(t)***increases faster as the natural mortality rate decreases while ***C(t)*** decreases faster as the natural mortality rate increases.

Figure 4.9 and 4.10 display the graph of ***N(t)*** and ***C(t)*** respectively against t for different values of γ. It was observed that the rate at which complications are cured had no effect on both ***N(t)*** and ***C(t)***.

Figure 4.11 displays the graph of ***N(t)*** against t for different values of λ. It was observed that the rate at which complications are cured had no effect on ***N(t).***

Figure 4.12 displays the graph of $\boldsymbol{C(t)}$ against t for different values of λ. It was observed that the number of diabetics with complication increases faster as the probability of diabetic persons developing a complication increases.

5.2 Conclusion

We studied a deterministic mathematical model of diabetes using the Homotopy Perturbation method and the result obtained shows that the parameters involved played a crucial role in the size of population of diabetes at time t and the number of diabetics with complication.

5.3 Recommendations

Like every other work, this work is not without limitation and can be improved. Diabetes is a deadly disease if not detected and taken care off early; hence it is recommended that further research should be carried out such as considering the variable incidence of Diabetes Mellitus and so on.

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