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Analysis of Mathematical Model for the Dynamics of Diabetes Mellitus and Its Complications

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ABSTRACT

Diabetes mellitus is a metabolic disorder associated with impaired glucose, lipid and protein metabolism as a consequence of impaired production, secretion or action of insulin in human body system. In this work, we developed a mathematical model for the dynamics of diabetes mellitus and its complications and carried out analysis of the model. The analytical solution of the model equations is obtained using Homotopy Perturbation Method. Numerical simulation of the model solution was done using Mapple 18 Mathematical software. The parameters are varied and their effects on the model dynamics are presented graphically. The results showed that the dearths due to diabetes complications can be reduced drastically if the rate at which complications are treated is high and the rate of developing a complication is slow.

Keywords: Diabetes mellitus, complications, homotopy perturbation method, simulation and mathematical model.

1 INTRODUCTION

Diabetes is a metabolic disorder characterized by the inability of the body system to regulate the amount of glucose (special sugar in the body) in the body. This occurs when the insulin secreted by the organ (pancreas) in the beta cell is not sufficient or the body does not utilise the insulin produced effectively. The resulting effect is that the body system cannot function effectively and glucose level in the blood goes beyond normal (IDF, 2004; WHO and IDF, 2004; WHO, 2014). Globally, people living with diabetes constitutes over 3 percent of the world population, out of which 75 - 90 percent are non - insulin dependent (NIDDM) and 10 - 25 percent are insulin dependent (IDDM). The NIDDM are normally diagnosed at the age of 40 years and IDDM diagnosed before 40 years. The non - insulin dependent diabetes after diagnosis require treatment with tablets and diet or diet alone while insulin dependent diabetes necessitate treatment with diet and insulin after diagnosis.

The prevalence of diabetes cases, the burden associated with diabetes and its attendant complications have become a source of concern to the growing population of the world. The number of people diagnosed of diabetes yearly was stated to rise from 366 million in 2011 to 552 million by 2030 (Wild *et al.,* 2000). The number of deaths attributable to diabetes yearly worldwide is alarming. Recently, there have been an increase in life expectancy around the world. Death rate caused by other leading non-communicable diseases such as Cancer, Cardiovascular disease and Stroke have been decreasing but that of diabetes is rising (Ali *et al.,* 2015).

Mathematical modelling have been used effectively to provide insight about incidence and prevalence of diabetes and help understand factors affecting disease development risk. Boutayeb *et al*. (2004) formulated a Mathematical model to study the dynamics of diabetes mellitus and its complications in a population. Their model assumption is the constant rate of diabetes person developing complications. Diabetics population were splitted into two groups: diabetics with complications and diabetics without complications. The model is a system of ordinary differential equations and the solution of the model was obtained using numerical method. The results shows that the incidence of diabetes and occurence of complications can be controlled with efficient and effective control strategies. Akinsola and Oluyo (2014), proposed a mathematical model on the dynamics and control of diabetes mellitus and its complications. Their model is an improvement on the work of Boutayeb *et al* (2006) and it was based on the size of diabetics without complications and diabetics with complications. Their study revealed that diabetes persists but its complications can be controlled. They investigated the sensitivity of each parameters to the model and the results obtained shows that the size of diabetics with complications can be reduced with adequate control measures. Enagi *et al* (2017), proposed the method of Homotopy Perturbation to solve a system of equations of model of diabetes mellitus disease. Analytical solution was obtained and graphical profile of the solution was shown using Mapple software. The results showed that model parameters play a very important role in determining the size of diabetics population and the number of diabetics with and without complications at time t.

In this paper, we improved on the existing model by incorporating three classes namely: healthy class, susceptible class and diabetics with complications undergoing treatment class. The study also considered genetic factors and lifestyle as two causes of diabetes in the population. In section two, we described the proposed model and its parameters and obtained the analytical solution of the model equations. In section three, we carried out numerical simulation of the results obtained in section two and presents the graphical profile of the system responses. In section four, we discussed our results.

1.1 Homotopy Pertubation Method (HPM)

The method of Homotopy perturbation for solving problems of nonlinear equation was first introduced by Ji Huan (He, 1999; He, 2000) and subsequently, it was improved by He, (2000), He, (2000a), and He, (2006). The method was successfully applied to solve problems made up of nonlinear and linear fucntional equations. In recent years, scientists, technologists and engineers have applied homotopy perturbation method to reduce problem of nonlinear and linear differential equations that seem to be difficult into simpler one that is easier to solve. The method is a combination of homotopy method in topology and traditional perturbation method (He, 2000b). The method of homotopy perturbation have provided a very convenient approach for obtaining analytical or approximate solution of series of problems in various field. Many researchers have successfully applied the method to physical problems such as non-liear wave equations, bifurcation, asymptotology, SIR infection disease model (Abubakar *et al.,* 2013), equation of schrodinger (Biazar & Ghazvini, 2007), diabetes model (Enagi *et al.,* 2017), heat radiation equation and reaction- duffision equation (Ganji and Rajabi, 2006; Ganji and Sadighi, 2006), oscillators with discontinuities (He, 2004ab, 2005a,b), Sine-Gordon and Klein-Gordon equations (Odibat & Momani, 2007; Chowdhury & Hashim, 2007), MHD Jeffery–Hamel problem (Moghimi *et al.,* 2011).

1.2 Fundamental of Homotopy Pertubation Method

The basic illustration of the method according to He (2000), using non-linear differential equation is stated as follows:

$$D(u)-k(x)=0, \qquad \chi \in \Lambda \tag{1}$$

With boundary condition of:

$$E\left(u,\frac{\partial u}{\partial n}\right)=0 \quad , \qquad \chi \in \prod \tag{2}$$

D denotes general differential operator, the boundary operator is *E*, the known analytical function is *k* (*x*) and $\prod$ denotes domain boundary of Λ. The operator *D* can be divided into two parts of *G* and *H*, the linear part is *G*, while *H* is a non-linear one. Equation (1) can be rewritten as follows:

$$G(u)+H(u)-k(x)=0, \quad \chi \in \Lambda$$

The stated HPM equation is as follows:

$$Q(w,r)=(1-r)[G(w)-G(u_0)]+r[G(w)+H(w)-k(x)]=0 \tag{3}$$

The embedding parameter is $r\in[0,1]$ in equation (3) and the first assumption that meets the boundary condition is μ_0. The assumed solution of equation (3) can be expressed in power series as follows:

$$w=w_0+r\,w_1+r^2\,w_2+\cdots \tag{4}$$

The correct solution of (1) is obtained by setting $r=1$

$$u=\lim_{p\to 1} w=w_0+w_1+w_2+\cdots \tag{5}$$

In most cases, the series (4) converges. The convergent rate depends on the nonlinear operator $D(w)$ (He, 2000).

2 MATERIALS AND METHODS

2.1 Model Formulation

We considered the model proposed by Enagi *et al. (2017),* with some modifications and incorporate three classes. Based on their health status, the model population are classified into five classes. They are healthy class *H(t)*, susceptible class *S(t),* diabetic without complications class *D(t),* diabetic with complications class*C(t)* and diabetic with complications that undergo treatment class $T(t)$. In this model, we assume that the healthy individual will give birth to a healthy children that will be born into healthy compartment while parent who is diabetic or have history of diabetes will give birth to children with genetic factors that will be born into susceptible compartment. The proportion of children born into healthy compartment is denoted by θ while proportion of children that are born into susceptible compartment is denoted by $1-\theta$.

Table 1 Definition of variables of the model

S/N	Variables	Description
1	$H(t)$	Healthy class
2	$S(t)$	Susceptible class
3	$D(t)$	Diabetics without complications class
4	$C(t)$	Diabetics with complications class
5	$T(t)$	Diabetics with complications undergoing treatment class
6	$N(t)$	Total population

Table 2 Definition of parameters of the model

S/N	Parameters	Description
1	α	Probability rate of incidence of diabetes
2	β	Birth rate
3	μ	Natural mortality rate
4	τ	Rate at which healthy individual become susceptible
5	σ	Rate at which susceptible individual become healthy
6	λ	Rate at which $D(t)$ develop a complications
7	γ	Rate at which $C(t)$ are treated
8	ω	Rate at which $C(t)$ after treatment return to $D(t)$
9	δ	Mortality rate due to complications

10	θ	Proportion of children born into the healthy class
11	$1-\theta$	Proportion of children born into the susceptible class

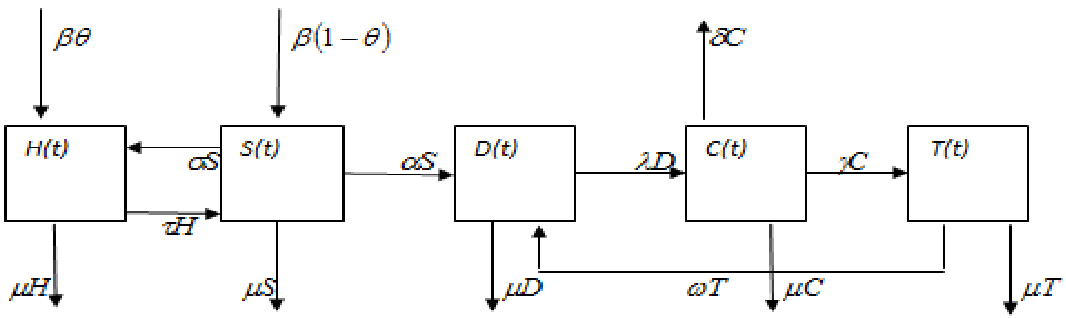

Figure 1: Schematic diagram of the model.

2.2 The Model Equations

The model equations are stated as follows:

$$\frac{dH(t)}{dt} = \beta\theta - \tau H(t) + \sigma S(t) - \mu H(t) \tag{6}$$

$$\frac{dS(t)}{dt} = \beta(1-\theta) - \mu S(t) - \alpha S(t) - \sigma S(t) + \tau H(t) \tag{7}$$

$$\frac{dD(t)}{dt} = \alpha S(t) - \mu D(t) - \lambda D(t) + \omega T(t) \tag{8}$$

$$\frac{dC(t)}{dt} = \lambda D(t) - \gamma C(t) - \delta C(t) - \mu C(t) \tag{9}$$

$$\frac{dT(t)}{dt} = \gamma C(t) - \omega T(t) - \mu T(t) \tag{10}$$

$$N(t) = H(t) + S(t) + D(t) + C(t) + T(t)$$

The initial values conditions are $H(o) = H_o, S(o) = S_o, D(o) = D_o, C(o) = C_o$ and $T(o) = T_o$.

2.3 Solution of Model Equations by Homotoppy Perturbation Method

Let equations (6) to (10) be written as

$$\frac{dH}{dt} + k_1 H - \sigma S - \beta\theta = 0 \tag{11}$$

$$\frac{dS}{dt} - \tau H + k_2 S - \beta(1-\theta) = 0 \tag{12}$$

$$\frac{dD}{dt} - \alpha S + k_3 D - \omega T = 0 \tag{13}$$

$$\frac{dC}{dt}-\lambda D+k_4C=0 \tag{14}$$

$$\frac{dT}{dt}-\gamma C+k_5T=0 \tag{15}$$

where $k_1=\mu+\tau$, $k_2=\mu+\alpha+\sigma$, $k_3=\mu+\lambda$, $k_4=\mu+\delta+\gamma$, $k_5=\mu+\omega$

With initial conditions $H(0)=H_0$, $S(0)=S_0$, $D(0)=D_0$, $C(0)=C_0$ and $T(0)=T_0$

Let

$$H(t)=m_0+pm_1+p^2m_2+\cdots \tag{16}$$

$$S(t)=n_0+pn_1+p^2n_2+\cdots \tag{17}$$

$$D(t)=x_0+px_1+p^2x_2+\cdots \tag{18}$$

$$C(t)=y_0+py_1+p^2y_2+\cdots \tag{19}$$

$$T(t)=z_0+pz_1+p^2z_2+\cdots \tag{20}$$

Applying HPM to (6) gives

$$(1-p)\frac{dH}{dt}+p\left[\frac{dH}{dt}+k_1H-\sigma S-\beta\theta\right]=0 \tag{21}$$

Substituting (16) to (20) into (21) and simplify gives

$$(1-p)\left[m'_0+pm'_1+p^2m'_2+\cdots\right]+p\begin{bmatrix}m'_0+pm'_1+p^2m'_2+\cdots\\+k_1\left(m_0+pm_1+p^2m_2+\cdots\right)\\-\sigma\left(n_0+pn_1+p^2n_2+\cdots\right)-\beta\theta\end{bmatrix}=0$$

$$\begin{aligned}&m'_0+pm'_1+p^2m'_2+\cdots+pk_1\left(m_0+pm_1+p^2m_2+\cdots\right)\\&-p\sigma\left(n_0+pn_1+p^2n_2+\cdots\right)-p\beta\theta=0\end{aligned} \tag{22}$$

Collecting the coefficients of power of P gives

$$\left.\begin{aligned}&p^0: m'_0=0\\&p^1: m'_1+k_1m_0-\sigma n_0-\beta\theta=0\\&p^2: m'_2+k_1m_1-\sigma n_1=0\end{aligned}\right\} \tag{23}$$

Applying HPM to (7) gives

$$(1-p)\frac{dS}{dt}+p\left[\frac{dS}{dt}-\tau H+k_2S+\beta\theta-\beta\right]=0 \tag{24}$$

Substituting (16) to (20) into (24) and simplify gives

$$(1-p)\left[n'_0 + pn'_1 + p^2 n'_2 + \cdots\right] + p\begin{bmatrix} n'_o + pn'_1 + p^2 n'_2 + \cdots \\ -\tau\left(m_0 + pm_1 + p^2 m_2 + \cdots\right) \\ +k_2\left(n_0 + pn_1 + p^2 n_2 + \cdots\right) \\ +\beta\theta - \beta \end{bmatrix} = 0$$

$$\begin{aligned} &n'_0 + pn'_1 + p^2 n'_2 + \cdots - p\tau\left(m_0 + pm_1 + p^2 m_2 + \cdots\right) \\ &+ pk_2\left(n_0 + pn_1 + p^2 n_2 + \cdots + \beta\theta - \beta\right) = 0 \end{aligned} \tag{25}$$

Collecting the coefficients of powers of P gives

$$\left.\begin{aligned} &p^0 : n'_0 = 0 \\ &p^1 : n'_1 - \tau m_0 + k_2 n_0 + \beta\theta - \beta = 0 \\ &p^2 : n'_2 - \tau m_1 + k_2 n_1 = 0 \end{aligned}\right\} \tag{26}$$

Applying HPM to (8) gives

$$(1-p)\frac{dD}{dt} + p\left[\frac{dD}{dt} - \alpha S + k_3 D - \omega T\right] = 0 \tag{27}$$

Substituting (16) to (20) into (27) and simplify gives

$$(1-p)\left[x'_0 + px'_1 + p^2 x'_2 + \cdots\right] + p\begin{bmatrix} x'_0 + px'_1 + p^2 x'_2 + \cdots \\ -\alpha\left(n_0 + pn_1 + p^2 n_2 + \cdots\right) \\ +k_3\left(x_0 + px_1 + p^2 x_2 + \cdots\right) \\ -\omega\left(z_0 + pz_1 + p^2 z_2 + \cdots\right) \end{bmatrix} = 0$$

$$\begin{aligned} &x'_0 + px'_1 + p^2 x'_2 + \cdots - p\alpha\left(n_0 + pn_1 + p^2 n_2 + \cdots\right) \\ &+ pk_3\left(x_0 + px_1 + p^2 x_2 + \cdots\right) - p\omega\left(z_0 + pz_1 + p^2 z_2 + \cdots\right) = 0 \end{aligned} \tag{28}$$

Collecting the coefficients of the powers of P gives

$$\left.\begin{aligned} &p^0 : x'_0 = 0 \\ &p^1 : x'_1 - \alpha n_0 + k_3 x_0 - \omega z_0 = 0 \\ &p^2 : x'_2 - \alpha n_1 + k_3 x_1 - \omega z_1 = 0 \end{aligned}\right\} \tag{29}$$

Applying HPM to (9) gives

$$(1-p)\frac{dC}{dt}+p\left[\frac{dC}{dt}-\lambda D+k_4 C\right]=0 \tag{30}$$

Substituting (16) to (20) into (30) and simplify gives

$$(1-p)\left[y'_0+py'_1+p^2y'_2+\cdots\right]+p\begin{bmatrix} y'_0+py'_1+p^2y'_2+\cdots \\ -\lambda\left(x_0+px_1+p^2x_2+\cdots\right) \\ +k_4\left(y_0+py_1+p^2y_2+\cdots\right)\end{bmatrix}=0$$

$$\begin{aligned} &y'_0+py'_1+p^2y'_2+\cdots-p\lambda\left(x_0+px_1+p^2x_2+\cdots\right) \\ &+pk_4\left(y_0+py_1+p^2y_2+\cdots\right)=0 \end{aligned} \tag{31}$$

Collecting the coefficients of the powers of P gives

$$\left.\begin{aligned} &p^0: y'_0=0 \\ &p^1: y'_1-\lambda x_0+k_4y_0=0 \\ &p^2: y'_2-\lambda x_1+k_4y_1=0 \end{aligned}\right\} \tag{32}$$

Applying HPM to (10) gives

$$(1-p)\frac{dT}{dt}+p\left[\frac{dT}{dt}-\gamma C+k_5 T\right]=0 \tag{33}$$

Substituting (16) to (20) into (33) and simplify gives

$$(1-p)\left[z'_0+pz'_1+p^2z'_2+\cdots\right]+p\begin{bmatrix} z'_0+pz'_1+p^2z'_2+\cdots \\ -\gamma\left(y_0+py_1+p^2y_2+\cdots\right) \\ +k_5\left(z_0+pz_1+p^2z_2+\cdots\right)\end{bmatrix}=0$$

$$\begin{aligned} &z'_0+pz'_1+p^2z'_2+\cdots-p\gamma\left(y_0+py_1+p^2y_2+\cdots\right) \\ &+pk_5\left(z_0+pz_1+p^2z_2+\cdots\right)=0 \end{aligned} \tag{34}$$

Collecting the coefficients of powers of P gives

$$\left.\begin{aligned} p^0 &: z'_0 = 0 \\ p^1 &: z'_1 - \gamma y_0 + k_5 z_0 = 0 \\ p^2 &: z'_2 - \gamma y_1 + k_5 z_1 = 0 \end{aligned}\right\} \tag{35}$$

Solving the first equations of (23), (26), (29), (32) and (35) gives

$$\left.\begin{aligned} m_0 &= H_0 \\ n_0 &= S_0 \\ x_0 &= D_0 \\ y_0 &= C_0 \\ z_0 &= T_0 \end{aligned}\right\} \tag{36}$$

Substituting (36) into second equations of (23), (26), (29), (32) and (35) gives

$$\left.\begin{aligned} & m'_1 + k_1 H_0 - \sigma S_0 - \beta\theta = 0 \\ & n'_1 - \tau H_0 + k_2 S_0 + \beta\theta - \beta = 0 \\ & x'_1 - \alpha S_0 + k_3 D_0 - \omega T_0 = 0 \\ & y'_1 - \lambda D_0 + k_4 C_0 = 0 \\ & z'_1 - \gamma C_0 + k_5 T_0 = 0 \end{aligned}\right\} \tag{37}$$

Solving (37) gives

$$\left.\begin{aligned} m_1 &= (\beta\theta - k_1 H_0 + \sigma S_0)t \\ n_1 &= (\tau H_0 - k_2 S_0 - \beta\theta + \beta)t \\ x_1 &= (\alpha S_0 - k_3 D_0 + \omega T_0)t \\ y_1 &= (\lambda D_0 - k_4 C_0)t \\ z_1 &= (\gamma C_0 - k_5 T_0)t \end{aligned}\right\} \tag{38}$$

Substituting (38) into the third equations of (23), (26), (29), (32) and (35) gives

$$m'_2 = -k_1(\beta\theta - k_1 H_0 + \sigma S_0)t + \sigma(\tau H_0 - k_2 S_0 - \beta\theta + \beta)t = 0$$

Integrating with respect to t, we have

$$m_2 = [\sigma(\tau H_0 - k_2 S_0 - \beta\theta + \beta) - k_1(\beta\theta - k_1 H_0 + \sigma S_0)]\frac{t^2}{2} + \cdots \tag{39}$$

$$n'_2 = \tau(\beta\theta - k_1 H_0 + \sigma S_0)t - k_2(\tau H_0 - k_2 S_0 - \beta\theta + \beta)t$$

Integrating with respect to t, we have

$$n_2 = [\tau(\beta\theta - k_1 H_0 + \sigma S_0) - k_2(\tau H_0 - k_2 S_0 - \beta\theta + \beta)]\frac{t^2}{2} + \cdots \tag{40}$$

$$x'_2 = \alpha(\tau H_0 - k_2 S_0 - \beta\theta + \beta)t - k_3(\alpha S_0 - k_3 D_0 + \omega T_0)t + \omega(\gamma C_0 - k_5 T_0)t$$

Integrating with respect to t, we have

$$x_2 = [\alpha(\tau H_0 - k_2 S_0 - \beta\theta + \beta) - k_3(\alpha S_0 - k_3 D_0 + \omega T_0) + \omega(\gamma C_0 - k_5 T_0)]\frac{t^2}{2} + \tag{41}$$

$$y'_2 = \lambda(\alpha S_0 - k_3 D_0 + \omega T_0)t - k_4(\lambda D_0 - \lambda\phi D_0 - k_4 C_0)t$$

Integrating with respect to t, we have

$$y_2 = [\lambda(\alpha S_0 - k_3 D_0 + \omega T_0) - k_4(\lambda D_0 - \lambda\phi D_0 - k_4 C_0)]\frac{t^2}{2} + \cdots \tag{42}$$

$$z'_2 = \gamma(\lambda D_0 - \lambda\phi D_0 - k_4 C_0)t - k_5(\gamma C_0 - k_5 T_0)t$$

Integrating with respect to t, we have

$$z_2 = [\gamma(\lambda D_0 - \lambda\phi D_0 - k_4 C_0) - k_5(\gamma C_0 - k_5 T_0)]\frac{t^2}{2} + \cdots \tag{43}$$

$$\left.\begin{aligned}
m_2 &= [\sigma(\tau H_0 - k_2 S_0 - \beta\theta + \beta) - k_1(\beta\theta - k_1 H_0 + \sigma S_0)]\frac{t^2}{2} + \cdots \\
n_2 &= [\tau(\beta\theta - k_1 H_0 + \sigma S_0) - k_2(\tau H_0 - k_2 S_0 - \beta\theta + \beta)]\frac{t^2}{2} + \cdots \\
x_2 &= \begin{bmatrix} \alpha(\tau H_0 - k_2 S_0 - \beta\theta + \beta) - k_3(\alpha S_0 - k_3 D_0 + \omega T_0) \\ + \omega(\gamma C_0 - k_5 T_0) \end{bmatrix}\frac{t^2}{2} + \cdots \\
y_2 &= [\lambda(\alpha S_0 - k_3 D_0 + \omega T_0) - k_4(\lambda D_0 - \lambda\phi D_0 - k_4 C_0)]\frac{t^2}{2} + \cdots \\
z_2 &= [\gamma(\lambda D_0 - k_4 C_0) - k_5(\gamma C_0 - k_5 T_0)]\frac{t^2}{2} + \cdots
\end{aligned}\right\} \tag{44}$$

Substituting (36), (38) and (44) into (16) gives

$$H(t) = H_0 + p[(\beta\theta - k_1 H_0 + \sigma S_0)t] + p^2 \begin{bmatrix} \sigma(\tau H_0 - k_2 S_0 - \beta\theta + \beta) \\ - k_1(\beta\theta - k_1 H_0 + \sigma S_0) \end{bmatrix}\frac{t^2}{2} + \cdots \tag{45}$$

Setting $p = 1$ of (45) becomes

$$H(t) = \lim_{p \to 1} m = m_0 + m_1 + m_2 \tag{46}$$

Hence,

$$H(t)=H_0+(\beta\theta-k_1H_0+\sigma S_0)t-\begin{bmatrix}\sigma(\tau H_0-k_2S_0-\beta\theta+\beta)\\-k_1(\beta\theta-k_1H_0+\sigma S_0)\end{bmatrix}\frac{t^2}{2}+\cdots \tag{47}$$

Substituting (36), (38) and (44) into (17) gives

$$S(t)=S_0+p[(\tau H_0-K_2S_0-\beta\theta+\beta)t]+p^2\begin{bmatrix}\tau(\beta\theta-k_1H_0+\sigma S_0)\\-k_2(\tau H_0-k_2S_0-\beta\theta+\beta)\end{bmatrix}\frac{t^2}{2}+. \tag{48}$$

Setting $p=1$ of (48) becomes

$$S(t)=\lim_{p\to1}n=n_0+n_1+n_2 \tag{49}$$

Hence,

$$S(t)=S_0+(\tau H_0-k_2S_0-\beta\theta+\beta)t+\begin{bmatrix}\tau(\beta\theta-k_1H_0+\sigma S_0)\\-k_2(\tau H_0-k_2S_0-\beta\theta+\beta)\end{bmatrix}\frac{t^2}{2}+\cdots \tag{50}$$

Substituting (36), (38) and (44) into (18) gives

$$D(t)=D_0+p[(\alpha S_0-k_3D_0+\omega T_0)t]+p^2\begin{bmatrix}\alpha(\tau H_0-k_2S_0-\beta\theta+\beta)\\-k_3(\alpha S_0-k_3D_0+\omega T_0)\\+\omega(\gamma C_0-k_5T_0)\end{bmatrix}\frac{t^2}{2}+\cdots \tag{51}$$

Setting $p=1$ of (51) becomes

$$D(t)=\lim_{p\to1}x=x_0+x_1+x_2 \tag{52}$$

Hence,

$$D(t)=D_0+(\alpha S_0-k_3D_0+\omega T_0)t+\begin{bmatrix}\alpha(\tau H_0-k_2S_0-\beta\theta+\beta)\\-k_3(\alpha S_0-k_3D_0+\omega T_0)\\+\omega(\gamma C_0-k_5T_0)\end{bmatrix}\frac{t^2}{2}+\cdots \tag{53}$$

Substituting (36), (38) and (44) into (19) gives

$$C(t)=C_0+p[(\lambda D_0-k_4C_0)t]+p^2\begin{bmatrix}\lambda(\alpha S_0-k_3D_0+\omega T_0)\\-k_4(\lambda D_0-k_4C_0)\end{bmatrix}\frac{t^2}{2}+\cdots \tag{54}$$

Setting $p=1$ of (54) becomes

$$C(t)=\lim_{p\to1}y=y_0+y_1+y_2 \tag{55}$$

Hence,

$$C(t)=C_0+(\lambda D_0-k_4C_0)t+\begin{bmatrix}\lambda(\alpha S_0-k_3D_0+\omega T_0)\\-k_4(\lambda D_0-k_4C_0)\end{bmatrix}\frac{t^2}{2}+\cdots \tag{56}$$

Substituting (36), (38) and (44) into (20) gives

$$T(t) = T_0 + p[(\gamma C_0 - k_5 T_0)t] + p^2[\gamma(\lambda D_0 - k_4 C_0) - k_5(\gamma C_0 - k_5 T_0)]\frac{t^2}{2} + \cdot \tag{57}$$

Setting $p = 1$ of (57) becomes

$$T(t) = \lim_{p \to 1} z = z_0 + z_1 + z_2 \tag{58}$$

Hence,

$$T(t) = T_0 + (\gamma C_0 - k_5 T_0)t + [\gamma(\lambda D_0 - k_4 C_0) - k_5(\gamma C_0 - k_5 T_0)]\frac{t^2}{2} + \cdots \tag{59}$$

Equations (47), (50), (53), (56) and (59) are the solutions of the system of equations (6) to (10)

$$\left.\begin{aligned}
H(t) &= H_0 + (\beta\theta - k_1 H_0 + \sigma S_0)t + \begin{bmatrix} \sigma(\tau H_0 - k_2 S_0 - \beta\theta + \beta) \\ -k_1(\beta\theta - k_1 H_0 + \sigma S_0) \end{bmatrix}\frac{t^2}{2} + \cdots \\
S(t) &= S_0 + (\tau H_0 - k_2 S_0 - \beta\theta + \beta)t + \begin{bmatrix} \tau(\beta\theta - k_1 H_0 + \sigma S_0) \\ -k_2(\tau H_0 - k_2 S_0 - \beta\theta + \beta) \end{bmatrix}\frac{t^2}{2} + \cdots \\
D(t) &= D_0 + (\alpha S_0 - k_3 D_0 + \omega T_0)t + \begin{bmatrix} \alpha(\tau H_0 - k_2 S_0 - \beta\theta + \beta) \\ -k_3(\alpha S_0 - k_3 D_0 + \omega T_0) \\ +\omega(\gamma C_0 - k_5 T_0) \end{bmatrix}\frac{t^2}{2} + \cdots \\
C(t) &= C_0 + (\lambda D_0 - k_4 C_0)t + \begin{bmatrix} \lambda(\alpha S_0 - k_3 D_0 + \omega T_0) \\ -k_4(\lambda D_0 - k_4 C_0) \end{bmatrix}\frac{t^2}{2} + \cdots \\
T(t) &= T_0 + (\gamma C_0 - k_5 T_0)t + \begin{bmatrix} \gamma(\lambda D_0 - k_4 C_0) \\ -k_5(\gamma C_0 - k_5 T_0) \end{bmatrix}\frac{t^2}{2} + \cdots
\end{aligned}\right\} \tag{60}$$

2.4 Variables and Estimation of Parameter Values

Variables and parameters values were estimated based on the available data from United Nations (2019), International Diabetes Federation (IDF, 2014; IDF, 2018), World Health Organization (WHO, 2016;WHO, 2017), Fasanmade and Dagogo-Jack (2018), Africa Check (2018) and Adeloye *et al.*(2017).

Table 3: Values of Variables used for Numerical Simulation

Variables	**Values**
$H(0)$	198,195,839
$S(0)$	101,535,728
$D(0)$	940,000
$C(0)$	3,760,000
$T(0)$	1,193,250
N(t)	202,895,839

Table 4: Values of Parameters used for Numerical Simulation

Parameters	**Values**	**Source**
α	0.02	IDF (2018)
β	0.038	UN (2019)
γ	0.08	Derouich *et al* (2014)
λ	0.05	Permatasari *et al* (2018)
μ	0.118	UN (2019)
σ	0.08	Boutayeb *et al* (2004)
θ	0.923	Purnami *et al* (2018)
ω	0.08	Permatasari *et al* (2018)
δ	0.02	Boutateb *et al* (2004)
τ	0.04	Permatasari *et al* (2018)
$1-\theta$	0.077	Purnami *et al* (2018)

3 NUMERICAL SIMULATION

Numerical simulation of the results obtained in section 2.3 was carried out using mathematical software (MAPLE 18) and the graphical profiles of the system responses are presented below.

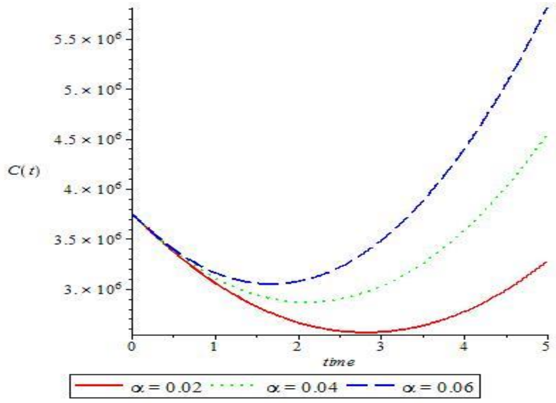

Figure 2 : Effect of probability rate of incidence of diabetes on Diabetics with complications class

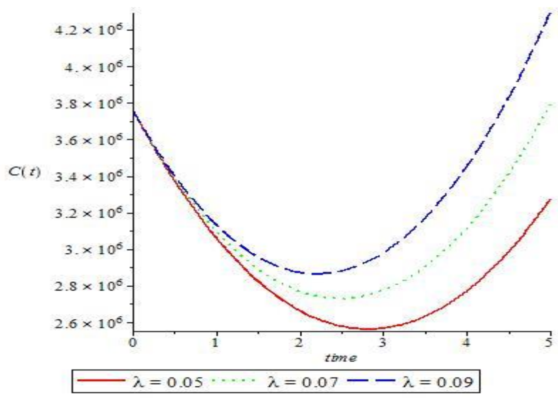

Figure 3: Effect of Probability rate of Diabetes persons developing Complications on Diabetics with Complications Class.

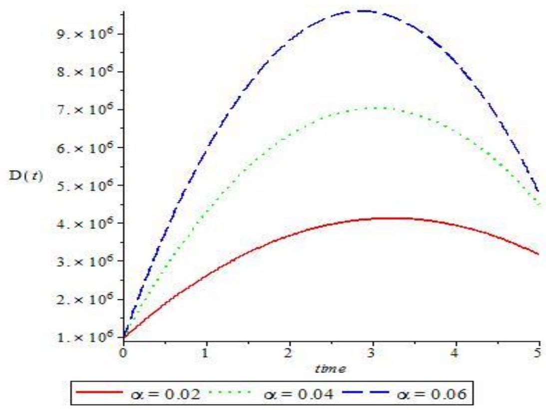

Figure 4: Effect of Probability rate of Incidence of Diabetes on Diabetics without Complications Class.

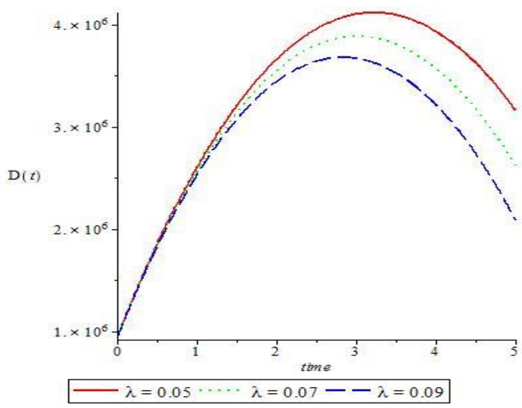

Figure 5: Effect of Probability rate of Diabetes persons developing Complications on Diabetics without Complications Class.

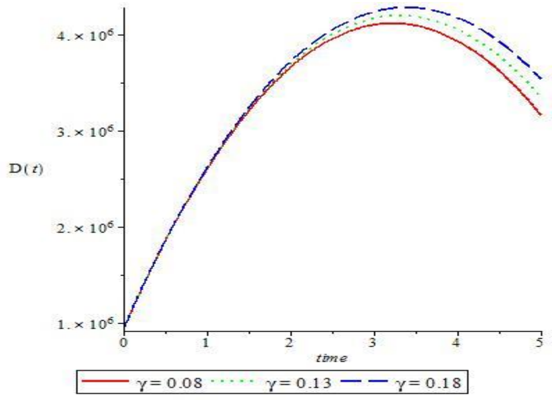

Figure 6. Effect of Treatment rate on Diabetics without Complications Class

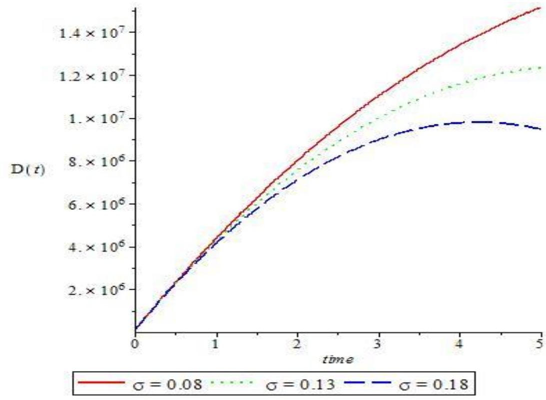

Figure 7. Effect of rate at which Susceptible Individual become Healthy on Diabetics without complications Class

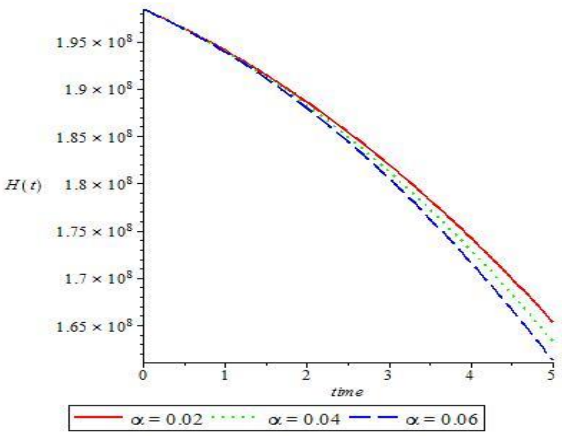

Figure 8. Effect of Probability rate of Incidence of Diabetes on Healthy Class

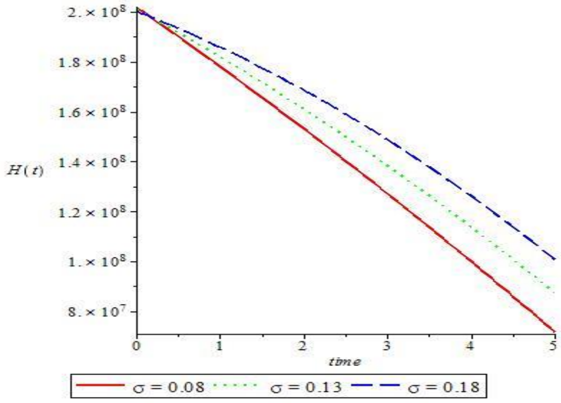

Figure 9. Effect of rate at which Susceptible Individual become Healthy on Healthy Class

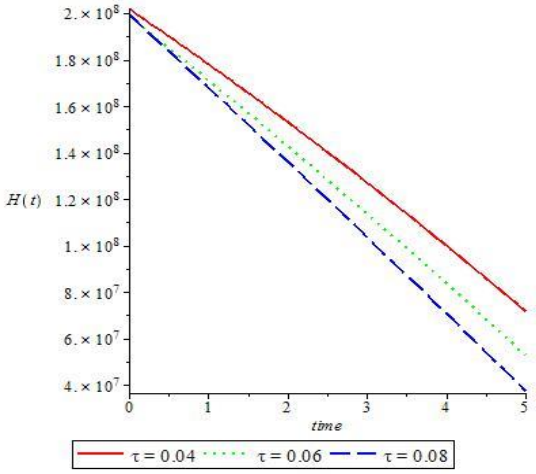

Figure 10: Effect of Susceptibility rate on Healthy Class

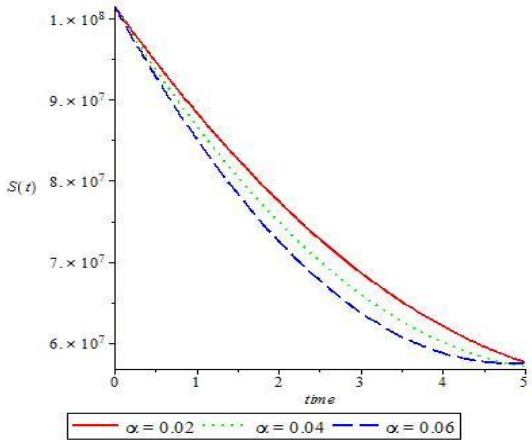

Figure 11: Effect of Probability rate of Incidence of Diabetes on Susceptible Class

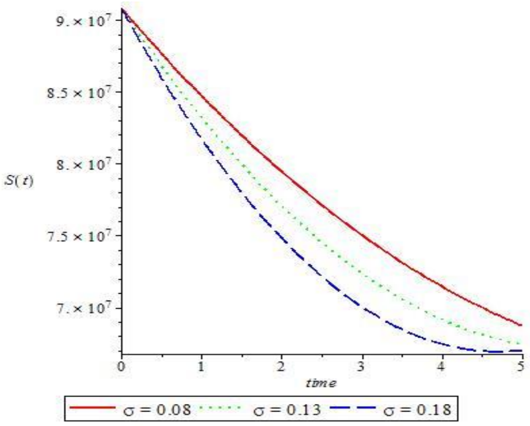

Figure 12. Effect of rate at which Susceptible Individual become Healthy on Susceptible Class

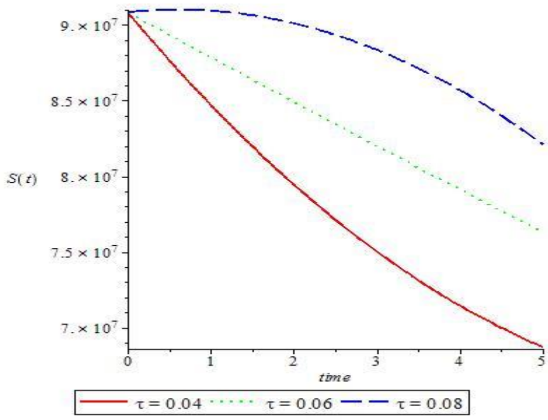

Figure 13. Effect of Susceptibility rate on susceptible Class

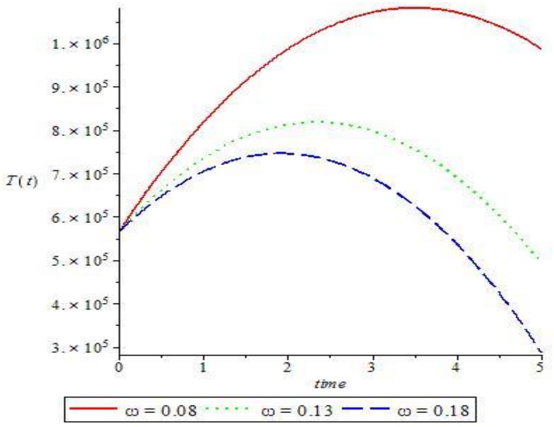

Figure 14. Effect of Complications Recovery rate after Treatment on Diabetics with Complications undergoing Treatment Class

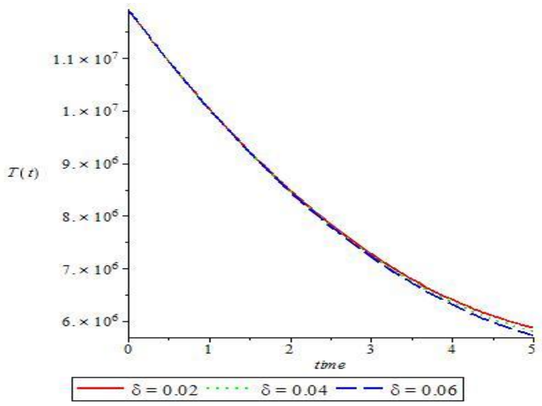

Figure 15. Effect of Death rate due to Complications on Diabetics with Complications undergoing Treatment Class.

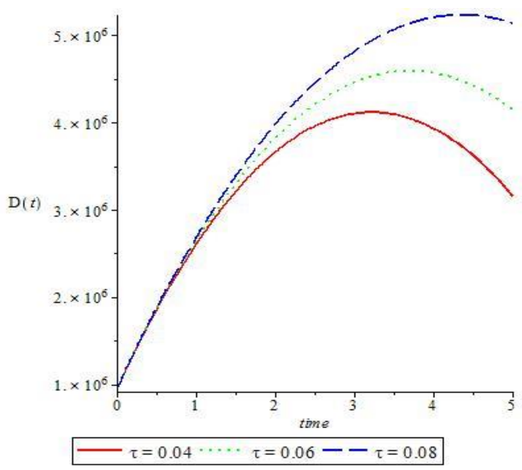

Figure 16. Effect of Susceptibility rate on Diabetics without Complications

4 ANALYSIS OF RESULTS

Figure 2 shows that, as the probability rate of incidence of diabetes increases, diabetics with complications increases. This shows that the more incidence of diabetes occur due to genetic factors and lifestyle factors such as smoking, alcoholism, glutonning and lack of physical excercises, the more cases of diabetes complications while Figure 3 shows that, as the probability rate of diabetes persons developing complications increases, diabetics with complications increases. This translate to the need to control the transition from diabetics without complications to diabetics with complications by effective management of diabetes.

Figure 4 shows that, as the probability rate of incidence of diabetes increases, diabetics without complications class increases. This shows that the more incidence of diabetes occur, the more cases of diabetes while Figure 5 shows that, as the probability rate of diabetics developing complications increases, diabetics with complications increases and diabetics without complications decreases faster.

Figure 6 is the effect of treatment rate on diabetics without complications class. This shows that high treatment rate increased diabetics with complications undergoing treatment. It is shown that effective treatment helps to controlled the menance of diabetes thereby reducing the number of death due to diabetes while Figure 7 shows that, as the rate at which susceptible individual become healthy increases, the diabetics without complications class decreases. This may be attributable to high positive lifestyle such as good dieting and physical excercises. This shows that the more people adopt positive lifestyle, the less they are susceptible to diabetes.

Figure 8 shows that, as the probability rate of incidence of diabetes increases, the healthy class decreases. This is attributable to the high rate of unhealthy lifestyle such as alcoholism, smoking, glutoning (over eating) and lack of physical excercises among the people while Figure 9 shows that, as the rate at which susceptible individual become healthy increases, the healthy class increases.This may be attributable to high positive lifestyle such as good dieting and physical excercises. This shows that the more people adopt positive lifestyle, the less they are susceptible to diabetes.

Figure 10 shows that, as rate of susceptability increases, the healthy class decreases. This shows that the more people live unhealthy lifestyle, the more people are susceptible to diabetes. This may be attributable to low level of awareness among the populace while Figure 11 shows that, as the probability rate of incidence of diabetes increases, the susceptible class decreases. This is attributable to the high rate of unhealthy lifestyle such as alcoholism, smoking, glutoning (over eating) and lack of physical excercises among the people.

Figure 12 shows that, as the rate at which susceptible individual become healthy increases, the susceptible class decreases slowly. This may be attributable to high positive lifestyle such as good dieting and physical excercises. This shows that the more people adopt positive lifestyle, the less they are susceptible to diabetes while Figure 13 shows that, as susceptibility rate increases, the susceptable class increases. This shows that the more people are living unhealthy lifestyle such as smoking, alcoholism, glutoning and lack of physical excercises, the more people are susceptible to diabetes.

Figure 14 shows that, as the rate of recovery from complications increases, diabetics with complications undergoing treatment decreases. This shows that the more diabetics with complications are treated, the more they recovered and move to diabetics without complications class thereby reducing the number of death due to complications while Figure 15 shows that, as the rate of death due to complications increases, diabetics with complications undergoing treatment decreases. This shows that with high rate of death due to complications, diabetics with complications undergoing treatment will reduces to zero.

Figure 16 shows that, as the rate of susceptability increases, diabetics without complications class increases. This shows that the more people are susceptible to diabetes, the more incidence of diabetes occur.

5 CONCLUSIONS

The model described well the dynamics of diabetes mellitus and its complications and the results obtained showed that the parameters involved has an effect in determining the size of diabetics population in the country. However, an effective control measure is proposed to curtails the menace of diabetes thereby reducing the number dearths attributable to complications of diabetes.

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