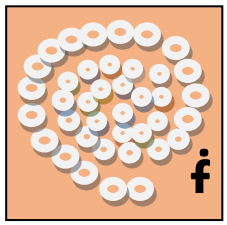

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Controlling the dynamics of Diabetes Mellitus with mathematical model

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Abstract

A suitably and mathematically well posed model of the dynamics of diabetes mellitus; incorporating three control parameters, was formulated. The obtained equilibrium point shows that there is no disease free equilibrium (DFE) but endemic equilibrium point (EEP) of the model, The Analysis of the model shows that the endemic equilibrium point of the model is both locally asymptotically stable (LAS) and globally asymptotically stable (GAS). Homotopy perturbation method was used to obtain the model solution. Nigeria diabetes values and parameters are used to obtain the sensitivity analysis of the sensitive parameters, and graphical simulations of the numerical solution of the state variables. The study provides a comprehensive guidance for the prevention and control of Type 1, Type 2 and gestational diabetes.

***Keywords and phrases*:** model; diabetes mellitus; equilibrium point; model solution; graphical simulation.

1 Introduction

Diabetes mellitus is an endocrine-metabolic disorder characterised by persistent hyperglycaemia (high blood sugar) due to insulin shortage, inadequate insulin production or ineffective use of the insulin produced [1]. This condition lowers life expectancy, quality of life and increases the risk of micro

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vascular damage (retinopathy, nephropathy, and neuropathy) as well as macro vascular damage (ischemic heart disease, stroke, and peripheral vascular disease) [2-11].

It results from either ineffectiveness of insulin or a hereditary or acquired deficit non pancreatic insulin production [12]. According to the World Health Organization, the number of persons with diabetes increased from an estimated 30 million in 1985 to over 171 million in 2000. By 2030, it is predicted that there would be over 366 million, with emerging nations experiencing a notable surge in the number, especially among individuals between the ages of 45 and 64 [13-14]. The population of diabetics is currently rising significantly in several countries [15]. Neonatal diabetes mellitus (NDM) is a severe and monogenic form of diabetes mellitus (DM) which is characterized by onset of insulin requiring hyperglycaemia within the first month of life [16]; type 1 diabetes can occur before six months of life [17] or in the first six months [15]. NDM can be either transient or permanent. Nearly half of the individuals with NDM are permanent [15]. Type 1 diabetes, or insulin-dependent diabetes, and type 2 diabetes, or non-insulin-dependent diabetes, are the two major forms of diabetes mellitus. Five to ten percent of cases of diabetes are type 1 diabetes mellitus, generally referred to as insulin-dependent diabetes mellitus (IDDM). IDDM, which is most common in adolescents and young adults due to pancreatic insufficiency, is a condition in which the body fails to manufacture insulin [18]. Type 1 diabetes has an unknown aetiology and, at this point in time, is not preventive [19]. Nonetheless, there have been theories regarding autoimmune cell destruction. Sudden symptoms include excessive excretion of urine (polyuria), thirst (polydipsia), constant hunger, weight loss, vision changes, and fatigue [19,20]. Type 2 diabetes mellitus is also known as non-insulin-dependent diabetes mellitus (NIDDM), in which the body does not produce enough insulin or makes improper use of secreted insulin [21]. It is the most common form of diabetes, accounting for 90–95% of diabetes. Type 2 diabetes is nearing epidemic proportions due to an increased number of elderly people and a greater prevalence of obesity and sedentary lifestyles [21]. Symptoms may be like those of type 1 diabetes but are often less marked. Thus, the disease may be diagnosed several years after onset, once complications have already arisen [19]. Gestational diabetes is a disorder that occurs when a woman without diabetes develops hyperglycaemia during pregnancy [19, 21]. Gestational diabetes can cause health problems for both the mother and the baby. Gestational diabetes increases the risk of pre-eclampsia, depression, and requiring a Caesarean section, and babies born to mothers with poorly treated gestational diabetes are at increased risk of being too large, having low blood sugar after birth, and having jaundice [19]. If untreated, it can result in a stillbirth [13, 16, 19]. In 90% of cases, gestational diabetes will resolve after the baby is born [19]. Women, however, are at an increasing risk of developing type 2 diabetes [16].

This study improved on the existing studies by incorporating all types of diabetes, stages before, when, and after, diabetes is discovered, in determining the compartments and the control parameters in the formulated mathematical model. One of the three control parameters, is assumed to be moderately consumption of medicinal plants (strictly organic plants) in human daily diets. Properties of some of the many medicinal plants were studied in the work of [18]. The formulated mathematically well posed model is qualitatively and quantitatively analysed in order to be suitable for individual, health personnel, government and nongovernmental organisations, whenever solution to problem of diabetes is required.

1.1 Motivational Problem

Awareness to prevent, control and manage the problematic rising trend of diabetes mellitus and its complications on human population, requires mathematical model for both qualitative and quantitative analysis towards an improved quality of human lives on any part of the earth surface.

2 Materials and Methods

2.1 Model formulation

The total population at time t, denoted by N(t), and is sub divided into mutually exclusive compartments of healthy individuals H(t); susceptible individuals to diabetes, S(t); individuals with diabetes, D(t); and individuals with diabetes who are either receiving treatment or managed, using medicine or herbs, disciplined eating habit, exercises and other chiropractic approaches, M(t).

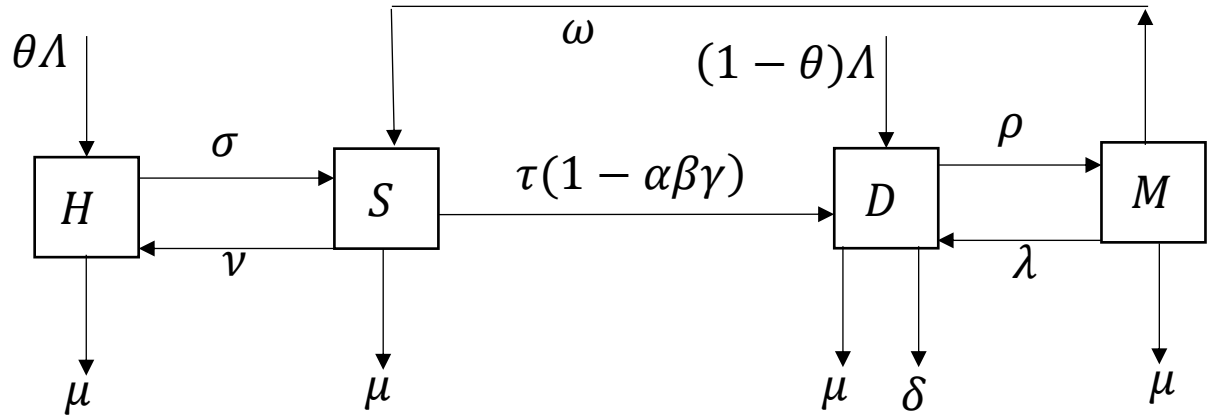

Figure I: Flow Diagram of the Model

2.2 The Mathematical Model

$$\left(\begin{array}{c} \dot{H} = \theta\Lambda - (\mu+\sigma)H + \nu S \\ \dot{S} = \sigma H - [\tau(1-\alpha\beta\gamma) + \nu + \mu)]\mathrm{S} + \omega M \\ \dot{D} = (1-\theta)\Lambda + \tau(1-\alpha\beta\gamma)S - (\lambda+\mu+\delta)D + \rho M \\ \dot{M} = \lambda D - (\omega+\rho+\mu)M \end{array}\right] \quad (1)$$

Table 1: Parameters and their meanings

Parameters	Description
Λ	Birth rate of human being
θ	Proportion of birth into the healthy compartment
$1-\theta$	Proportion of birth into the diabetes compartment
σ	Rate at which healthy individuals become susceptible to diabetes
τ	Rate of incidence of diabetes from susceptible to diabetes compartment
α	Public campaign that enhanced diabetes awareness
β	Control parameter due to regular exercise and other chiropractic activities
γ	Control parameter due to consistent and disciplined eating habit, i.e. herbal medicinal food (Organic)
λ	Rate at which neonatal and gestational diabetes are being moved for treatment
ν	Rate at which susceptible individuals move to the healthy compartment
μ	Natural mortality rate of human beings
δ	Mortality rate due to diabetes
ω	Rate at which transient and gestational diabetes move to susceptible compartment
ρ	Rate at which managed diabetes move back to diabetes compartment

Table 2: State Variables and their meanings

Variables	**Description**
H	Healthy compartment
S	Susceptible compartment
D	Diabetes compartment
M	Treatment/Managed Diabetes compartment
N	Total human population

Theorem1: Consider the feasible region, $\Sigma = \left\{ H, S, D, M \in R_+^4 : 0 \le N \le \frac{\Lambda}{\mu} \right\}$, Σ is a positively invariant set and a global attractor of the model

Let $H_0 \ge 0, S_0 \ge 0, D_0 \ge 0, M_0 \ge 0.$ and $H_0 + S_0 + D_0 + M_0 = N_0,$ then there exist solutions, H(t), S(t), D(t) and M(t) for the dynamical system (1) with the initial data H_0, S_0, D_0 and M_0 at time t = 0, that are defined for all $t \ge 0$. Adding the four equations of the model (1) and applying the standard comparison theorem [22] we have,

$$\frac{dN}{dt} \le \Lambda - \mu N \tag{2}$$

Applying integrating factor to equation (2) gives

$$N(t) \le \frac{\Lambda}{\mu} + [N(0) - \frac{\Lambda}{\mu}]e^{-\mu t} \tag{3}$$

In particular, $N(0) \le \frac{\Lambda}{\mu}$ implies $N(t) \le \frac{\Lambda}{\mu}$. Hence Σ is positively invariant and an attractor, so that no solution path leaves through any boundary of Σ.

Lemma 1: The region Σ is positively invariant for the system (1) and if $D_0 > 0,$ then $H_0 > 0, S > 0, M > 0$ for all time t, greater than zero and M is bounded by $\dot{M} = \max\left\{ M_0, \frac{\lambda}{\omega + \rho + \mu} \right\}$

The feasible region, Σ is positively invariant and the state variables are nonnegative, Solution of the model exists and unique.

Proof:

The right hand side of the system (1) is continuously differentiable and hence it is locally Lipschitz and therefore, there exist a unique solution H(t), S(t), D(t), M(t) to the system (1) with the initial data H_0, S_0, D_0, M_0 that is defined on a maximum forward interval of existence, [23]. Suppose that the initial condition is positive, $H_0 > 0$, $S_0 > 0$, $D_0 > 0$, $M_0 > 0$. We assumed by contradiction as it was in [24], that the state variables of the system (1) are positive if we suppose that there exists first time, t_1: $H(t_1) = 0$ and $H(t) > 0$, $S(t) > 0$, $D(t) > 0$, $M(t) > 0$, $0 < t < t_1$ or there exists t_2: $S(t_2) = 0$ and $H(t) > 0$, $S(t) > 0$, $D(t) > 0$, $M(t) > 0$, $0 < t < t_2$ or t_3: $D(t_3) = 0$ and $H(t) > 0$, $S(t) > 0$, $D(t) > 0$, $M(t) > 0$, $0 < t < t_3$ or t_4 : $M(t) = 0$ and $H(t) > 0$, $S(t) > 0$, $D(t) > 0$, $M(t) > 0$, $0 < t < t_4$. If $H(t_1) = 0$, $\Rightarrow \frac{dH(t_1)}{dt} = \lim_{t \to t_1} \frac{H(t_1) - H(t)}{t_1 - t} < 0$. Similarly $\frac{dS(t_1)}{dt} < 0, \frac{dD(t_1)}{dt} < 0, \frac{dM(t_1)}{dt} < 0$. Putting $H(t_1) = 0$ in the first equation of the system (1) implies $\frac{dH(t_1)}{dt} = \theta\Lambda + \nu S > 0$ which contradict our assumption. This implies that $H(t_1) \neq 0$, and H(t) will remain positive for all t, Similarly, S(t), D(t), M(t) are shown to be positive for all t.

To show that M is bounded by $\dot{M} = \max\{M_0, \frac{\lambda}{\omega + \rho + \mu}\}$ we need to show that $M \leq \xi$ for $t \geq 0$ and that $\xi \geq \frac{\lambda}{k_4}$ if $M_0 \leq \xi$. Suppose that the above inequalities do not hold, then there exist a time t_1 with $M''(t_1) > 0$ and $M'(t) > \xi$. Using the fourth equation for M in the system (1) gives $\frac{dM(t_1)}{dt} = \lambda D(t_1) - k_4 M(t_1) \leq \lambda(D(t_1) - 1) < 0.$ Since $(D(t_1) - 1) < 0$ and $\lambda > 0$ $\Rightarrow$ contradiction, therefore $\Rightarrow M(t) < \xi$ for all $t \geq 0.$ Suppose that $M(0) > \xi \geq \frac{\lambda}{k_4}.$ To show that $M(t) \leq M(0)$ for all $t \geq 0.$ We assume if there exists a time $t_2 > 0$ then $M(t_2) \geq M(0)$ and $M'(t_2) > 0$. However, since $M(t_2) > \frac{\lambda}{k_4}$ $\frac{dM(t_1)}{dt} = \lambda D(t_1) - k_4 M(t_1) \leq \lambda(D(t_1) - 1) \leq 0. \Rightarrow$ contradiction, because $M'(t_2) > 0$.

Therefore, M(t) is bounded from above by $\dot{M}$ that is $\dot{M} = \max\{M_0, \frac{\lambda}{k_4}\}$. Therefore, the model is epidemiologically meaningful and mathematically well posed, that is the model is suitable for analysis [25] ■

2.3 Equilibrium point of the model

Model (1) implies

$$\begin{bmatrix} \dot{H} = \theta\Lambda - k_1 H + \nu S \\ \dot{S} = \sigma H - k_2 \mathrm{S} + \omega M \\ \dot{D} = (1-\theta)\Lambda + k_0 S - k_3 D + \rho M \\ \dot{M} = \lambda D - k_4 M \end{bmatrix} \tag{4}$$

At the equilibrium point,

$$\dot{H} = \dot{S} = \dot{D} = \dot{M} = 0 \tag{5}$$

Equation (5) implies the endemic equilibrium point of the system (4)

$$\varepsilon_E = \begin{bmatrix} H^{\square} = \dfrac{\begin{pmatrix} k_4\theta\Lambda(k_1k_2-\nu\sigma)[(k_1k_2-\nu\sigma)(k_4-\lambda\rho)-k_1\lambda\tau(1-\alpha\beta\gamma)+ \\ \nu\{k_4\theta\sigma\Lambda[(k_1k_2-\nu\sigma)(k_4-\lambda\rho)-k_1\lambda\tau(1-\alpha\beta\gamma)]+ \\ k_1k_4\lambda[\theta\Lambda\sigma\tau(1-\alpha\beta\gamma)+(k_1k_2-\nu\sigma)(1-\theta)\Lambda]\} \end{pmatrix}}{k_1k_4(k_1k_2-\nu\sigma)[(k_1k_2-\nu\sigma)(k_4-\lambda\rho)-k_1\lambda\tau(1-\alpha\beta\gamma)]} \\ S^{\square} = \dfrac{\begin{matrix} k_4\theta\sigma\Lambda[(k_1k_2-\nu\sigma)(k_4-\lambda\rho)-k_1\lambda\tau(1-\alpha\beta\gamma)]+ \\ k_1k_4\lambda[\theta\Lambda\sigma\tau(1-\alpha\beta\gamma)+(k_1k_2-\nu\sigma)(1-\theta)\Lambda] \end{matrix}}{k_4(k_1k_2-\nu\sigma)[(k_1k_2-\nu\sigma)(k_4-\lambda\rho)-k_1\lambda\tau(1-\alpha\beta\gamma)]} \\ D^{\square} = \dfrac{k_4[\theta\sigma\Lambda\tau(1-\alpha\beta\gamma)+(k_1k_2-\nu\sigma)(1-\theta)\Lambda]}{(k_1k_2-\nu\sigma)(k_4-\lambda\rho)-k_1\lambda\tau(1-\alpha\beta\gamma)} \\ M^{\square} = \dfrac{\lambda[\theta\sigma\Lambda\tau(1-\alpha\beta\gamma)+(k_1k_2-\nu\sigma)(1-\theta)\Lambda]}{(k_1k_2-\nu\sigma)(k_4-\lambda\rho)-k_1\lambda\tau(1-\alpha\beta\gamma)} \end{bmatrix} \tag{6}$$

2.4 Model Solution, Using Homotopy Perturbation Method (HPM)

To use HPM, as it was in [26-28] the model (4) implies

$$\begin{pmatrix} \dot{H} + k_1 H - \nu S - \theta\Lambda &= 0 \\ \dot{S} - \sigma H + k_2 S + \omega M &= 0 \\ \dot{D} - \tau(1-\alpha\beta\gamma)S + k_3 D - (1-\theta)\Lambda + \rho M &= 0 \\ \dot{M} - \lambda D + k_4 M &= 0 \end{pmatrix} \tag{7}$$

With the initial conditions: $H(0) = H_0, S(0) = S_0, D(0) = D_0, M(0) = M_0$

Let

$$\begin{pmatrix} H(t) &= n_0 + pn_1 + p^2 n_2 + ... \\ S(t) &= x_0 + px_1 + p^2 x_2 + ... \\ D(t) &= y_0 + py_1 + p^2 y_2 + ... \\ M(t) &= z_0 + pz_1 + p^2 z_2 + ... \end{pmatrix} \quad (8)$$

Applying HPM to the first equation of the system (7) gives $(1-p)\dot{H} + p(\dot{H} + k_1 H - \nu S - \theta\Lambda) = 0$

$$\Rightarrow \dot{H} + pk_1 H - p\nu S - p\theta\Lambda = 0 \quad (9)$$

Substitute the first two equations in the system (8) into the equation (9), simplifying and then collecting the coefficients of power of p, gives

$$\begin{bmatrix} p^0 &\Rightarrow & n_0' &= 0 \\ p^1 &\Rightarrow & n_1' + k_1 n_0 - \nu x_0 - \theta\Lambda &= 0 \\ p^2 &\Rightarrow & n_2' + k_1 n_1 - \nu x_1 &= 0 \end{bmatrix} \quad (10)$$

Similarly applying HPM to second, third and the fourth equations of the system (7), give the system (11), (12) and (13)

$$\begin{bmatrix} p^0 &\Rightarrow & x_0' &= 0 \\ p^1 &\Rightarrow & x_1' - \sigma n_0 + k_2 x_0 + \omega z_0 &= 0 \\ p^2 &\Rightarrow & x_2' - \sigma n_1 + k_2 x_1 + \omega z_1 &= 0 \end{bmatrix} \quad (11)$$

$$\begin{bmatrix} p^0 &\Rightarrow & y_0' &= 0 \\ p^1 &\Rightarrow & y_1' - \tau(1-\alpha\beta\gamma)x_0 + k_3 y_0 + \rho z_0 - (1-\theta)\Lambda &= 0 \\ p^2 &\Rightarrow & y_2' - \tau(1-\alpha\beta\gamma)x_1 + k_3 y_1 + \rho z_1 &= 0 \end{bmatrix} \quad (12)$$

$$\begin{bmatrix} p^0 &\Rightarrow & z_0' &= 0 \\ p^1 &\Rightarrow & z_1' - \lambda y_0 + k_4 z_0 &= 0 \\ p^2 &\Rightarrow & z_2' - \lambda y_1 + k_4 z_1 &= 0 \end{bmatrix} \quad (13)$$

Solving the first equations of the system (11) – (13) $\Rightarrow$

$$n_0 = H_0, \quad x_0 = S_0, \quad y_0 = D_0, \quad z_0 = M_0 \quad (14)$$

Substitute (14) into the second equations of the system (10) – (13) then integrate $\Rightarrow$

$$\begin{pmatrix} n_1 &= & (\nu S_0 + \theta\Lambda - k_1 H_0)t \\ x_1 &= & (\sigma H_0 - k_2 S_2 + \omega M_0)t \\ y_1 &= & (\tau(1-\alpha\beta\gamma)S_0 - k_3 D_0 - \rho M_0 + (1-\theta)\Lambda)t \\ z_1 &= & (\lambda D_0 - k_4 M_0)t \end{pmatrix} \quad (15)$$

Substitute (15) into the third equations of the system (10) – (13) then integrate $\Rightarrow$

$$\left(\begin{array}{rcl} n_2 &=& [\nu(\sigma H_0 - k_2 S_0 + \omega M_0) - k_1(\nu S_0 + \theta\Lambda - k_1 H_0)]\frac{t^2}{2} + \ldots \\ x_2 &=& [\sigma(\nu S_0 + \theta\Lambda - k_1 H_0) - k_2(\sigma H_0 - k_2 S_0 + \omega M_0) - \omega(\lambda D_0 - k_4 M_0)]\frac{t^2}{2} + \ldots \\ y_2 &=& \{\tau(1-\alpha\beta\gamma)(\sigma H_0 - k_2 S_2 + \omega M_0) - k_3[\tau(1-\alpha\beta\gamma)S_0 - k_3 D_0 - \rho M_0 + (1-\theta)\Lambda)] - \rho(\lambda D_0 - k_4 M_0)\}\frac{t^2}{2} + \ldots \\ z_2 &=& \lambda[\tau(1-\alpha\beta\gamma)S_0 - k_3 D_0 - \rho M_0 + (1-\theta)\Lambda] - k_4(\lambda D_0 - k_4 M_0)\}\frac{t^2}{2} + \ldots \end{array}\right) \quad (16)$$

Substitute for $n_0, n_1, n_2, x_0, x_1, x_2, y_0, y_1, y_2, z_0, z_1, z_2$ in the system (8) and the then let $p \to 1$ we therefore obtain the model solution as given in the system (17)

$$\left(\begin{array}{rl} H(t) = & H_0 + (\nu S_0 + \theta\Lambda - k_1 H_0)t + [\nu(\sigma H_0 - k_2 S_0 + \omega M_0) - \\ & k_1(\nu S_0 + \theta\Lambda - k_1 H_0]\frac{t^2}{2} + \ldots \\ S(t) = & S_0 + (\sigma H_0 - k_2 S_0 + \omega M_0)t + [\sigma(\nu S_0 + \theta\Lambda - k_1 H_0) - \\ & k_2(\sigma H_0 - k_2 S_0 + \omega M_0) - \omega(\lambda D_0 - k_1 M_0)]\frac{t^2}{2} + \ldots \\ D(t) = & D_0 + \big(\tau(1-\alpha\beta\gamma)\big)S_0 - k_3 D_0 - \rho M_0 + (1-\theta)\Lambda)t + \\ & \{\big(\tau(1-\alpha\beta\gamma)\big)(\sigma H_0 - k_2 S_0 + \omega M_0 - k_3[\big(\tau(1-\alpha\beta\gamma)\big)S_0 - \\ & k_3 D_0 - \rho M_0 + (1-\theta)\Lambda - \rho(\lambda D_0 - k_4 M_0\}\frac{t^2}{2} + \ldots \\ M(t) = & M_0 + (\lambda D_0 - k_4 M_0)t + \{\lambda[\tau(1-\alpha\beta\gamma)S_0 - k_3 D_0 - \\ & \rho M_0 + (1-\theta)\Lambda] - k_4(\lambda D_0 - k_4 M_0)\}\frac{t^2}{2} + \ldots \end{array}\right) \quad (17)$$

3 Local stability of the endemic equilibrium point

Lemma 2: The model (4) has a unique endemic equilibrium whenever $\tau > 0$

Theorem 2: Consider the model (4). The associated unique endemic equilibrium of the model (4) denoted by ε_E is Locally Asymptotically Stable (LAS) if all the eigenvalues of the characteristic equation associated with the linearized System (4) have negative real parts.

Proof: Model (1) $\Rightarrow \frac{dN}{dt} = \Lambda - (\mu + \delta)N$, so that $N \to \frac{\Lambda}{\mu + \delta} = N^{\square}$ as $t \to \infty$, hence, using substitution

$H = N^{\square} - S - D - M$, model (1) can be re-written as

$$\left(\begin{array}{c} \dot{S} = \sigma\left(N^{\square} - S - D - M\right) - [\tau(1-\alpha\beta\gamma) + \nu + \mu)]\mathrm{S} + \omega M \\ \dot{D} = (1-\theta)\Lambda + \tau(1-\alpha\beta\gamma)S - (\lambda + \mu + \delta)D + \rho M \\ \dot{M} = \lambda D - (\omega + \rho + \mu)M \end{array}\right) \quad (18)$$

Linearizing system (8) gives

$$\left(\begin{array}{c} \dot{S} = \{\sigma x - [\tau(1-\alpha\beta\gamma) + \nu + \mu)]\}\mathrm{S} + xD + (x+\omega)M \\ \dot{D} = (1-\theta)\Lambda + \tau(1-\alpha\beta\gamma)S - (\lambda + \mu + \delta)D + \rho M \\ \dot{M} = \lambda D - (\omega + \rho + \mu)M \end{array}\right) \quad (19)$$

The Jacobian of the system (19) evaluated at ε_E is given as

$$\left(\begin{array}{ccc} \{\sigma x - [\tau(1-\alpha\beta\gamma) + \nu + \mu)]\} & x & (x+\omega) \\ \tau(1-\alpha\beta\gamma) & -(\lambda + \mu + \delta) & \rho \\ 0 & \lambda & -(\omega + \rho + \mu) \end{array}\right) \quad (20)$$

The proof of Theorem 2 is based on using a Krasnoselskii sublinearity trick (see, for instance, [29-31]. It entails showing that the system (19), $z'(t) = Mf(\bar{x})z$ has solution of the form

$$z(t) = z_0 e^{\omega_1 t} \quad (21)$$

with $z_0 \in C \setminus \{0\}, z_0 = (z_1, z_2, z_3), z_i \in C, \omega_1 \in C,$ and $\mathrm{Re}(\omega_1) < 0$ where C denotes complex numbers. Substituting a solution of the form (21) into the system (19) gives

$$\left(\begin{array}{c} \omega_1 z_1 = \{\sigma x - [\tau(1-\alpha\beta\gamma) + \nu + \mu)]\}z_1 + xz_2 + (x+\omega)z_3 \\ \omega_1 z_2 = (1-\theta)\Lambda + \tau(1-\alpha\beta\gamma)z_1 - (\lambda + \mu + \delta)z_2 + \rho z_3 \\ z_3 = \lambda z_2 - (\omega + \rho + \mu)z_3 \end{array}\right) \quad (22)$$

System (22) becomes

$$\left(\begin{array}{c} (1+\frac{\omega_1}{k_2})z_1 = \frac{\sigma H^{\square}}{k_2} z_1 + \frac{H^{\square}}{k_2} z_2 + \frac{(H^{\square} + \omega)}{k_2} z_3 \\ (1+\frac{\omega_1}{k_3})z_2 = \frac{(1-\theta)\Lambda}{k_3} + \frac{k_0}{k_3} z_1 + \frac{\rho}{k_3} z_3 \\ (1+\frac{\omega_1}{k_4})z_3 = \frac{\lambda}{k_4} z_2 \end{array}\right) \quad (23)$$

The simplification of results in

$$\begin{pmatrix} [1+F_1(\omega_1)] & = & (Mz)_1 \\ [1+F_2(\omega_2)] & = & (Mz)_2 \\ [1+F_2(\omega_3)] & = & (Mz)_3 \end{pmatrix} \tag{24}$$

with

$$M = \begin{pmatrix} \frac{\sigma H^{\square}}{k_2} & \frac{k_0 k_3 (k_4+\omega_1)}{k_4(1+\omega_1)(k_4+\omega_1)-\lambda\rho} & \frac{k_0 k_3}{k_4(1+\omega_1)(k_4+\omega_1)-\lambda\rho} \\ \frac{k_0}{k_3} & 0 & 0 \\ 0 & \frac{\lambda}{k_4} & 0 \end{pmatrix} \tag{25}$$

The equilibrium $\varepsilon_E = \{S^{\square}, D^{\square}, M^{\square}\} \Rightarrow \varepsilon_E = M\varepsilon_E$, where the matrix M has non-negative entries. It should be mentioned that, $(Mz)_i$, with i = 1, 2, 3 denotes the i^{th} coordinates of the vector Mz. If z is a solution of (21), such that [29- 31] and which is in the form $[1+F_i(\omega)] = (Mz)_i$ for $i = 1,2,3$ then it is possible to find a minimum positive real number, s, such that

$$\|z\| \le s\varepsilon_E \tag{26}$$

where, $\|z\| = (\|z_1\|, \|z_2\|, \|z_3\|)$ $\|.\|$ is a norm in C. Our mission is to show that $\mathrm{Re}(\omega) < 0$. Suppose by contradiction that, $\mathrm{Re}(\omega) \ge 0$, then there are $\omega = 0$ and $\omega \ne 0$ for consideration.

Case 1, $\omega = 0$ is not applicable to the model (4) because there is no disease free. The determinant of this system is given by

$$\Delta = -K_2 \cdot -K_3 \cdot -K_4 < 0 \tag{27}$$

Since determinant Δ is negative, it implies that the system (4) has a unique solution.

Case 2, $\omega \neq 0$

By assumption if $\mathrm{Re}(\omega) > 0$ then $[1+F_i(\omega)] > 1$ for $i = 1,2,3$ If $F(\omega) = \min_i[1+F_i(\omega)]$ then

$F(\omega) > 1 \Rightarrow \frac{s}{F(\omega)} < s.$ Since, s is the minimum positive real number such that $\|z\| \leq s\varepsilon_E$, then

$$\|z\| > \frac{s}{F(\omega)}\varepsilon_E \qquad (28)$$

Taking the norm of both sides of the second equation in (24) implies

$$F(\omega)\|z_2\| \leq \|1+F_2(\omega)\|\|z_2\| = \|(Mz)_2\| \leq M\|z_2\| \leq sM(\varepsilon_E)_2 = s(\varepsilon_E)_2 = s(D^{\square}) \qquad (29)$$

(29) implies, $\|z_2\| \leq \frac{s}{F(\omega)}(D^{\square})$ which contradicts (28). Hence, $\mathrm{Re}(\omega_1) < 0$ Thus, all eigenvalues of the characteristic equations associated with the linearized system (19) have negative real parts so that the unique endemic equilibrium (ε_E) point is locally asymptomatically stable whenever $\tau > 0$

3.1 Global Stability Analysis (GAS) of the Endemic Equilibrium Point

To determine GAS, as it was in [32] and applied quantitatively in [26] we obtain the Lyapunov Equation (quadratic Lyapunov function) of the system (4) as $A^TP + PA = -Q \Rightarrow A^TP + PA = -I$ if Q = I from the model (7), where

$$P = \begin{pmatrix} P_{11} & P_{12} & P_{13} & P_{14} \\ P_{21} & P_{22} & P_{23} & P_{24} \\ P_{31} & P_{32} & P_{33} & P_{34} \\ P_{41} & P_{42} & P_{43} & P_{44} \end{pmatrix}, \text{ and } Q = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}$$

Then the Jacobian matrix of the system (4) is

$$A=\begin{pmatrix} -k_1 & \nu & 0 & 0 \\ \sigma & -k_2 & 0 & \omega \\ 0 & k_0 & -k_3 & \rho \\ 0 & 0 & \lambda & -k_4 \end{pmatrix} \Rightarrow A^T=\begin{pmatrix} -k_1 & \sigma & 0 & 0 \\ \nu & -k_2 & k_0 & 0 \\ 0 & 0 & -k_3 & \lambda \\ 0 & \omega & \rho & -k_4 \end{pmatrix}$$

To determine the variables in the matrix P, we assumed, $\begin{pmatrix} a=k_1+k_2 & b=k_1+k_3 & c=k_1+k_4 \\ d=k_2+k_3 & e=k_2+k_4 & f=k_3+k_4 \end{pmatrix}$.
From the Lyapunov equation we derived the system (30) in order obtain the entries of the matrix P.

$$\begin{pmatrix} 2k_1P_{11}+\sigma P_{12}+\sigma P_{21} &=& -1 \\ \nu P_{11}-aP_{12}-k_0P_{13}+\sigma P_{22} &=& 0 \\ -bP_{13}+\lambda P_{14}+\sigma P_{23} &=& 0 \\ -cP_{14}+\omega P_{12}+\rho P_{13}+\sigma P_{24} &=& 0 \\ \nu P_{11}-aP_{21}+\sigma P_{22}+k_0P_{31} &=& 0 \\ \nu P_{12}-2k_2P_{22}-k_0P_{23}+k_0P_{32}+\nu P_{21} &=& -1 \\ \nu P_{13}-dP_{23}+\lambda P_{24}+k_0P_{33} &=& 0 \\ \nu P_{14}+\omega P_{22}+\rho P_{23}-eP_{24}+k_0P_{34} &=& 0 \\ -bP_{31}+\sigma P_{32}+\lambda P_{41} &=& 0 \\ \nu P_{31}-dP_{32}-k_0P_{33}+\lambda P_{22} &=& 0 \\ 2k_3P_{33}+\lambda P_{34}+\lambda P_{43} &=& -1 \\ \omega P_{32}+\rho P_{33}-fP_{34}+\lambda P_{44} &=& 0 \\ \omega P_{21}+\rho P_{31}-cP_{41}+\sigma P_{42} &=& 0 \\ \omega P_{22}+\rho P_{32}+\nu P_{41}-eP_{42}-k_0P_{43} &=& 0 \\ \omega P_{23}+\rho P_{33}-fP_{43}+\lambda P_{44} &=& 0 \\ \omega P_{24}+\rho P_{34}+\omega P_{42}+\rho P_{43}-2k_4P_{44} &=& -1 \end{pmatrix} \quad (30)$$

Solving the System (30), we obtained a diagonal matrix in which the diagonal entries are positive and all other entries are zeros. $P_{11}=\frac{1}{2k_1}>0, P_{22}=\frac{X}{Y}>0, P_{33}=\frac{1}{2k_3}>0, P_{44}=\frac{q}{(L+M+H)}>0$

$$X=k_1(2ak_1-\sigma\nu)[k_1(\sigma\nu-2ak_1)^2-2(a\nu\sigma)^2][(\sigma\nu-2ak_1)\nu+2ak_0][k_1(\sigma\nu-2ak_1)-a\nu\sigma]$$

$$Y=4a\sigma[k_1(\sigma\nu-2ak_1)-a\nu\sigma][k_2(\sigma\nu-2ak_1)+a\sigma\nu][(\sigma\nu-2ak_1)\nu+2ak_0][k_1(\sigma\nu-2ak_1)-a\nu\sigma]$$

$$\text{Matrix } P=\begin{pmatrix} P_{11} & P_{12} & P_{13} & P_{14} \\ P_{21} & P_{22} & P_{23} & P_{24} \\ P_{31} & P_{32} & P_{33} & P_{34} \\ P_{41} & P_{42} & P_{43} & P_{44} \end{pmatrix} \text{ becomes } P=\begin{pmatrix} P_{11} & 0 & 0 & 0 \\ 0 & P_{22} & 0 & 0 \\ 0 & 0 & P_{33} & 0 \\ 0 & 0 & 0 & P_{44} \end{pmatrix}$$

$$\Delta_1 = P_{11} > 0, \Delta_2 = \begin{vmatrix} P_{11} & 0 \\ 0 & P_{22} \end{vmatrix} > 0, \ \Delta_3 = \begin{vmatrix} P_{11} & 0 & 0 \\ 0 & P_{22} & 0 \\ 0 & 0 & P_{33} \end{vmatrix} > 0 \text{ and } \Delta_4 = \begin{vmatrix} P_{11} & 0 & 0 & 0 \\ 0 & P_{22} & 0 & 0 \\ 0 & 0 & P_{33} & 0 \\ 0 & 0 & 0 & P_{44} \end{vmatrix} > 0$$

P is positive definite matrix which therefore implies that EEP is Globally Asymptotically Stable

3.2 Variables and estimation of parameter values

Variables and Parameters values are obtained based on Nigeria Population, with their respective sources, stated in the Table 3 and 4 respectively.

Table 3: Values of Variables and the Sources

S N	Variables	Value	Source
1	H(0)	41,634,136	Estimated based on number [5, 6, 13]
2	S(0)	31,688,476	Estimated based on [5, 6, 13]
3	D(0)	96,812,400	[5, 6, 13]
4	M(0)	48,406,200	50% of D (0), based on [5, 6, 13]
5	N(0)	218,541,212	[5, 6, 13]

Table 4: Values of Parameters and the Sources

S N	Variables	Value	Source
1	τ	0.043	[6]
2	Λ	0.036	[17]
3	θ	0.999	[14]
4	1-θ	0.001	[14]

5	μ	0.009	[17]
6	ν	0.55	[33]
7	σ	0.101	[34]
8	α	0.95	Suggestion
9	β	0.95	Suggestion
10	γ	0.95	Suggestion
11	ω	0.8	[9]
12	λ	0.338	[35]
13	δ	0.316	Estimated based on type 1, 2 & gest.
14	ρ	0.20	[9]

3.3 Numerical Solution of the Model

Considering the four level of controls in the model solution (17), the numerical solution of the model solution is obtained as shown in (31) – (18)

$$\begin{pmatrix} \alpha=\beta=\gamma=0.00 \Rightarrow & H(t)=41,634,136+12,837,906.9t+2,486,737.6t^2+\ldots \\ \alpha=\beta=\gamma=0.25 \Rightarrow & H(t)=41,634,136+12,848,906.9t+3,952,592.4t^2+\ldots \\ \alpha=\beta=\gamma=0.95 \Rightarrow & H(t)=41,634,136+12,848,906.9t+5,693,301.4t^2+\ldots \\ \alpha=\beta=\gamma=1.00 \Rightarrow & H(t)=41,634,136+12,848,906.9t+6,225,669.6t^2+\ldots \end{pmatrix} \quad (31)$$

$$\begin{pmatrix} \alpha=\beta=\gamma=0.00 \Rightarrow & S(t)=31,688,476+11,609,885.0t-16,390,245.4t^2+\ldots \\ \alpha=\beta=\gamma=0.25 \Rightarrow & S(t)=31,688,476+11,803,051.5t-13,915,475t^2+\ldots \\ \alpha=\beta=\gamma=0.95 \Rightarrow & S(t)=31,688,476+23,272,695.4t-17,527,478.4t^2+\ldots \\ \alpha=\beta=\gamma=1.00 \Rightarrow & S(t)=31,688,476+25,216,1t-15,007,565.2t^2+\ldots \end{pmatrix} \quad (32)$$

$$\begin{pmatrix} \alpha=\beta=\gamma=0.00 \Rightarrow & D=96,812,400-60,241,816.5t+43,610,202.0t^2+\ldots \\ \alpha=\beta=\gamma=0.25 \Rightarrow & D=96,812,400-42,740,905.0t+21,969,634.9t^2+\ldots \\ \alpha=\beta=\gamma=0.95 \Rightarrow & D=96,812,400-71,924,407.0t+26,303,901.6t^2+\ldots \\ \alpha=\beta=\gamma=1.00 \Rightarrow & D=96,812,400-73,867,861.0t+26,099,122.5t^2+\ldots \end{pmatrix} \quad (33)$$

$$\begin{pmatrix} \alpha=\beta=\gamma=0.00 \Rightarrow & M(t)=48,406,200-16,119,264.6t+9,160,893.9t^2+\ldots \\ \alpha=\beta=\gamma=0.25 \Rightarrow & M(t)=48,406,200-16,119,264.6t-2,084,398.1t^2+\ldots \\ \alpha=\beta=\gamma=0.95 \Rightarrow & M(t)=48,406,200-16,119,264.6t-4,022,789.6t^2+\ldots \\ \alpha=\beta=\gamma=1.00 \Rightarrow & M(t)=48,406,200-16,119,264.6t-4,359,446.4t^2+\ldots \end{pmatrix} \quad (34)$$

$$\begin{pmatrix} D(1)=49,043,660.4+33,791,821.1\tau-15,844,238\tau^2 \\ D(2)=49,043,225.7+41,106,829.5\tau-15,353,129.7\tau^2 \\ D(3)=48,455,574.+4,810,325.9\tau-322,271.8\tau^2 \\ D(4)=24,470,108.8 \end{pmatrix} \quad (35)$$

$$\begin{pmatrix} \varsigma=\alpha\beta\gamma \Rightarrow & H(t)=58,630,731.1+3,748,746.7\varsigma \\ \varsigma=\alpha\beta\gamma \Rightarrow & S(t)=61,612,246.7+50,859,283.5\varsigma-8,950,306.4\varsigma^2 \\ \varsigma=\alpha\beta\gamma \Rightarrow & D(t)=63,971,808.9-8,671,610.1\varsigma-2,931,184.0\varsigma^2 \\ \varsigma=\alpha\beta\gamma \Rightarrow & M(t)=32,881,948.7-5,859,199.2\varsigma \end{pmatrix} \quad (36)$$

3.4 Graphical Simulation of the Model Solution with four level of Controls

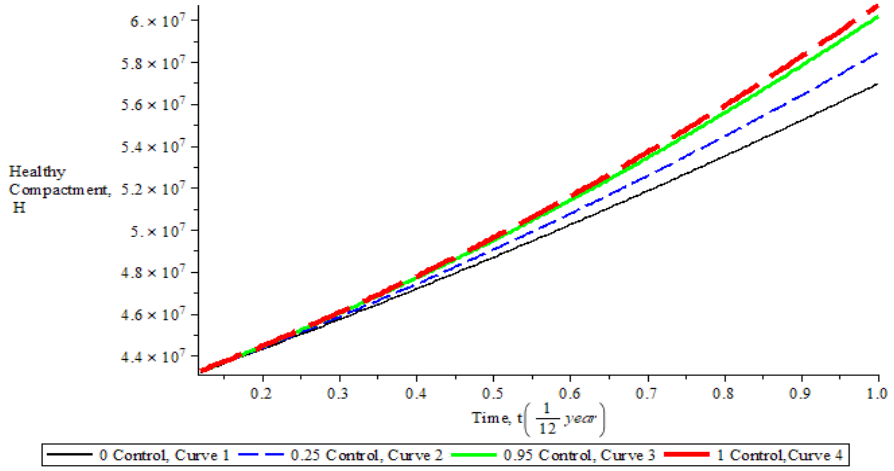

Figure 1: Effects of various controls on the Simulation of healthy Compartment

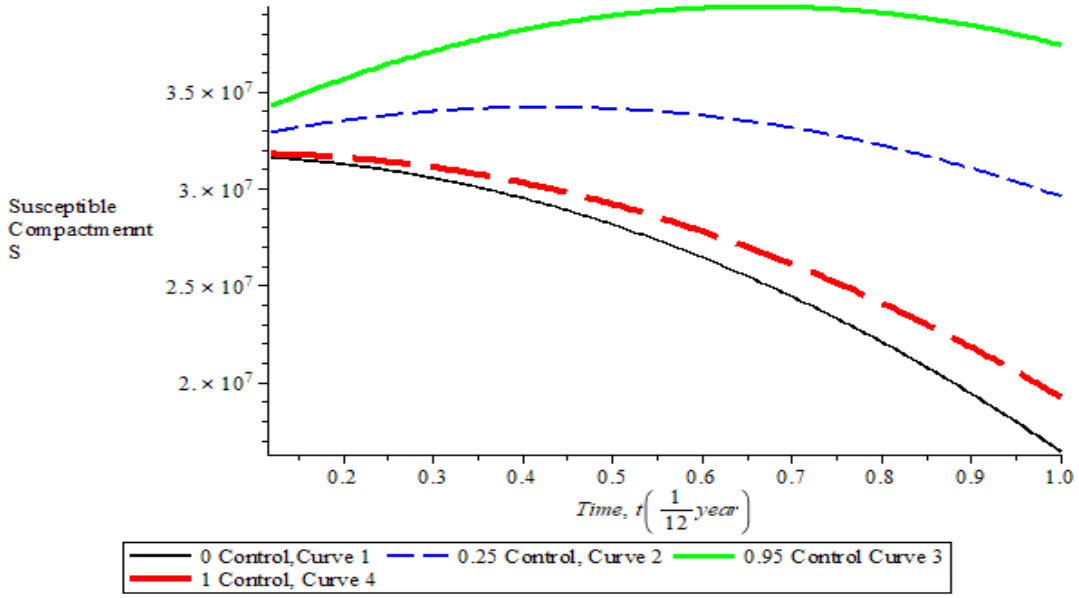

Figure 2: Effects of various controls on the Simulation of Susceptible Compartment

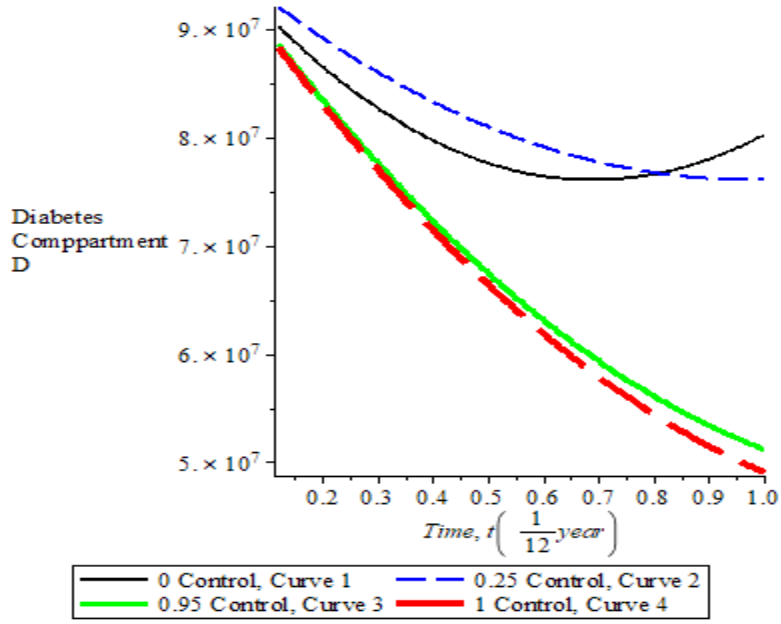

Figure 3: Effects of various controls on the Simulation of Diabetes Compartment

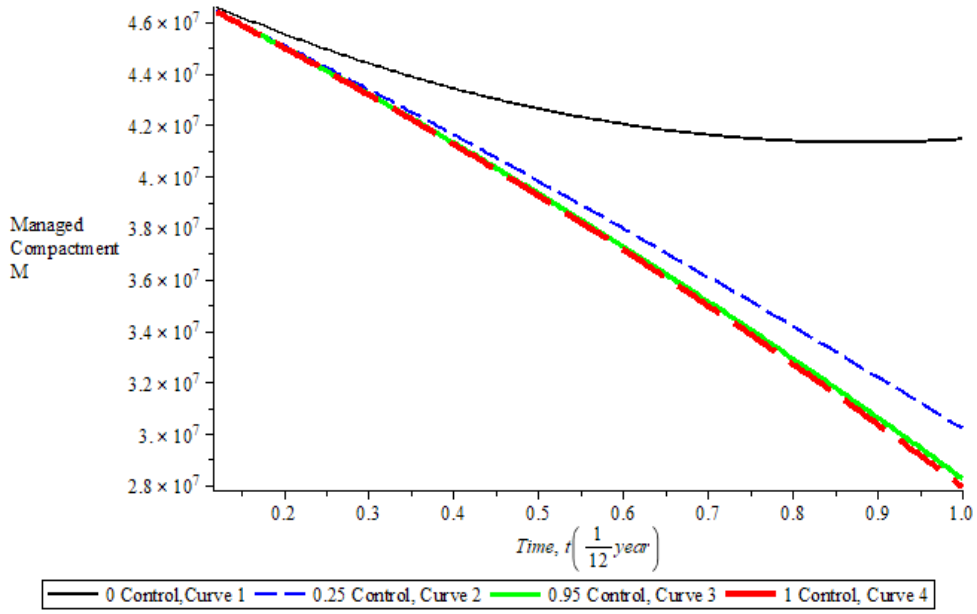

Figure 4: Effects of various controls on the Simulation of Managed Compartment

3.5 Sensitivity Analysis of the sensitive parameters

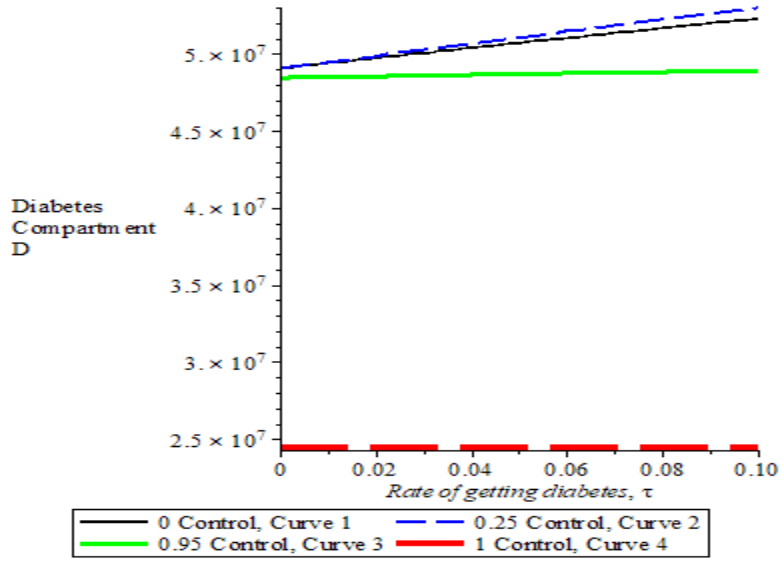

Figure 5: Effect of incidence of diabetes on the Diabetes Compartment

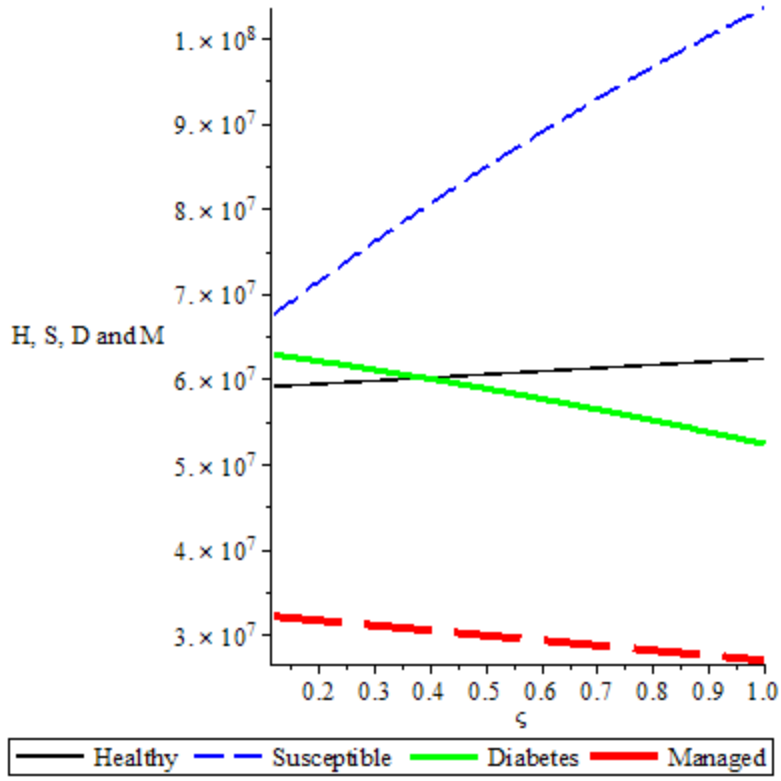

Figure 5: Effect of Control on the State Variables

4 Results and discussion

Equilibrium point of the formulated, mathematically, well posed model, indicates that Diabetes Mellitus has no disease free equilibrium (DFE) point, but endemic equilibrium point (EEP) and it is proved that the EEP is locally and globally asymptotically stable, that is (LAS and GAS). Graphical Simulation of the Numerical Solution of the healthy Compartment of the total population shows that the incorporated control parameters impacted positively on the healthy Compartment. The Numerical Simulation shows is directly impacted based on the sequence of the controls, that is, the positive effects are in accordance with the respective sizes of the controls. Level 0, no measure reflected the least population, while 0.25, 0.95 and 1.00 reflected positive effects proportional to their sizes; Therefore, the higher the control, the higher the size of the healthy compartment.

In the Simulation of the susceptible population, the effect of zero, 0, no measure, rapidly depleted the population, because of unhindered movement of susceptible individuals to the diabetes compartment. Impact of one, 1 control measure is faster than 0.25 and 0.95 control measures because of effect of public enlightenment campaign on the people. The higher the healthy population, the lower the susceptible population, as indicated in the figure 1 and 2. Based on the level of control and the initial value of the curve.

The Diabetes Mellitus population is positively impacted by 0.95 and 1.00 control measures, than 0.25 measure and no measure, by reducing the population of the compartment of diabetes, in proportion to the level of control.

The effects of control of measures on the managed compartment are positively impacted as 0.25, 0.95 and 1.00 reduce the respective population while the effect of no measure is negative, as the population is on the increase.

On the sensitivity analysis of the incidence of diabetes mellitus, no control measure, 0 and 0.25 show increasing trend on the population of the diabetes mellitus compartment.
The population of diabetes is kept constant with the control measure of 0.95 and 1 on the simulation of the diabetes population. The constant value in 0.95 is higher than the constant value in 1.00; this shows a perfect control will keep diabetes mellitus at bay.

5 Conclusion

Prevention, controlling and managing the rising trend of the associated complications of diabetes mellitus on human population; will be effectively checked if either control measure 0.95, $\alpha = \beta = \gamma = 0.95,$ or 1.00, $\alpha = \beta = \gamma = 1.00,$ is, adequately adopted; as incorporated in the Mathematical Model of the study; by Diabetes Patient or Health Personnel or Governmental Agencies or Nongovernmental Agencies; for type 1 diabetes or type 2 diabetes or gestational diabetes, if the advice from the study is strictly adhered.

5.1 Recommendation

Each of the three types of diabetes: type 1, type 2 and gestational diabetes can be uniquely studied mathematically.

6.0 Declarations:

6.1 Authors Contribution Statement

K. A. Adeyemo, T.A. Lawal, E. Philemon, I. A. Azeez, K.I. Falade, F. C Nwachukwu, A. Omame, N.O. Lasisi and P.O. Aye were all involved in the rigorous work of this study from conceiving the idea of the study, writing the abstract; introduction to the study; formulation of the model, solving the model equations, designing the analysis, collection of data, the results and discussion; and drawing the conclusion from the study.

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6.3 Data availability statement

Data included in article/supplied materials are referenced in the article.

6.4 Declaration of interest statement

The authors declare no conflict of interest.

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References

[1] W.H.O. (2021) Definition of Diabetes
[2] Nansseru J. R. N., Ngo –Um S. S and Balti E. V. (2016) Incidence, prevalence and genetic determinants of neonatal diabetes mellitus: a systemic review and meta –analysis protocol, Systemic Review: Volume 5(188), 0369-0373, Doi: 10:1186/s13643.0369-3
[3] Shankar PR, Partha P, Shenoy N, Subish P, Vijay B, Dubey AK, Nepal. (2007) Knowledge and perceptions about diabetes in the community: a survey among individuals attending a diabetes-screening programme in pokhara city, western Nepal Pharm cology online, Manipal College of Medical Sciences Pokhara Volume 1: 148-161
[4] W. H. O. (2018) Diabetes, httpps://www.who.int/news-room/fact-sheet/detail/diabetes.
[5] W.H.O. (2014) Global Health Estimates: Death by cause, Age, Sex and Country, Geneva 2002-2012, www.who.org or www.idf.who.org
[6] https://www.wordometers.info>world-population>nigeria population
[7] Neelesh J., Sanjay and Sapna M. (2010) Anti diabetic potential of medicinal plants, Acta poloniae pharmaceutical-Drug Research, Volume 10(1): 113-118.
[8] Patel K. P., Joshi H. M., Majmuar F. D and Patel V. J. (2013) Newer Approaches in the treatment of diabetes mellitus. NHL Journal of medicinal Sciences, Volume 2(1): 6-11
[9] Wellness, Be well Boston, 2024
[10] Kumar V. K., Sharathnath P., Yogeswaran P., Harani A.,Sudhakar K., Sudha P., David B. (2010) Medicinal potency of Mamordica charantia, International of pharmaceutical Sciences, Review and Research. Volume 1(2):95-106
[11] Ekam V. S., Ebong P. E., Johnson J. T and Dasofunjo K. (2013) Effect of activity directed fractions of Vernonia amygdalna on total body weight and blood glucose levels of diabetic wistar albino rats. International Journal of Science and Technology, Volume 2(1): 153 -157
[12] Mahora R., Stoicescu S –M., (2016) Congenital Diabetes Mellitus, Medica –a Journal of Clinical Medicine Volume 11(2): 154 -157
[13] International Diabetes Federation (I.D.F) (2021), Diabetes in Nigeria
[14] Beltran J., Busiah K., Vaivre—Douret L., Fauret., A. L., Berdugo M., Cave H and Polak M. (2020) Neonatal Diabetes Mellitus. Front. Pediatr 8: 540718 doi: 10 3389/fped.540718
[15] Chinenye S. and Young L. (2011) State of Diabetes in Nigeria, The Nigerian Health Journal , Volume 11(4): 101-106
[16] Alhassan A. J., Sule M. S., El-ta'alu A. B., and Lawal A. T. (2017) In vitro Inhibitory Activities of Persea Americana Seed Extracts on α -Amylase and α -Glucosidase. Bayero Journal of Pure and Applied Sciences, Volume 10(1), 546-552
[17] Wikipedia https://en.wikipedia.org>>wiki>Demographics of Nigeria
[18]Nagmoti D. M and Archana R. J. (2013) In vitro inhibitory effects of pithecellobiumdulce(Roxb) Benth seeds on intestinal α -glucosidase and pancreatic α -amylase, J Biochem Tech, Volume 4(3): 616-621.

[19] C.D.C (2024) Prevention of Gestational Diabetes, https://www.cdc.gov
[20] Donovan P. J., Mcintyre. H. D. (2010) Drugs for gestational diabetesAustralian presciber, 10. 18773austprescr.2010.066 33(5) https://www.nih.gov
[21] Implications of gestational diabetes (ADA, 2019).
[22] Lakshmiantham S., Leela S and Martynyuk A. A. (1989) Stability Analysis of nonlinear system Marcel Deker, Inc., New York and Basel
[23]Perko L. (2000) Differential equations and dynamical systems. Springer ; New York
[24] Huo, H. F., Chang, S. J.,&Li, Y. N. (2010).Stability of two strain tuberculosis model with general contact rate.Abstract and Applied Analysis. ID 293747, 31 pages. Doi: 10.1155/2010/293747.
[25] Hethccote H. W. (2000).The mathematics of infectious diseases, SIAM Rev, 42(4):599-653
[26] 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, University Malaysia Perlis, U – Map, Volume 10(1), 57-77
[27]He J. H. (2000) A coupling homotopy perturbation technique and perturbation technique for nonlinear problems, International Journal of Non-Linear Mechanics. Volume 35: 37-43
[28]He J. H. (2000). New perturbation technique which is also valid for large parameters, Journal Sound and Vibration. Volume 229: 1257-1263
[29]Dessalegn, Y. M annd Gumel A. B. (2010) Global Asymptotic Properties of an SEIRS Model with Multiple Infectious Stages. J. Math. Anal. Appl.366: 202 -217. www.elsvier.com/locate/jmaa
[30] Adeyemo, K. A., Akinwande N I., and Lasisi N. O. (2020) Local Stability Analysis of the Unique Endemic Equilibrium Point of a Mathematical Model for the Transmission and Control of Zika Virus disease Dynamics. Caliphate journal of science and technology (CaJoST), 2, 175 -181.
[31] Garba, S. M., &Gumel, A. B., (2010).Mathematical recipe for HIV elimination in Nigeria.Journal of the Nigerian Mathematical Society. 29, 51-112
[32]Hedrick J.K and Girard A. (2005) Control of nonlinear dynamical system, theory and application. Class note: 62-83
[33] Estimated based on ADA (2020)
[34] Bashir M.A., Yahaya M.M., Yusuf A.H., Mukhtar. (2021) Prediabetes Burden in Nigeria, A Systemic Review and Meter-Analysis, Front Public Health Sec. Clinical Diabetes, Volume 9, https//doi.org/10.3389/fpbh.2021.762429
[35]Bao S.Y., Han S.Y., Wang H., Bao W., Ao W. (2016b). Improvement Effects of Agiophyllum Oligosaccharides on General Characterization and Glucose and Lipid Metabolism of Diabete GK Rats. J. Jilin Univ. (Medicine Edition) 42, 1059–1065. [Google Scholar]