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A theoretical analysis of a model for diabesity dynamics

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Abstract. In this paper, we make use of the theory of dynamical system in order to investigate the well-posedness of a model governing the spread of diabesity with the effect of treatment. The model consists of a system of nonlinear ordinary differential equations with a nonlinear incidence response. Positivity, boundedness, global existence and uniqueness of the solutions are established. In particular, Lyapunov stability theory and spectral methods are employed to investigate the stability of the disease free and endemic equilibria.

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1. Introduction

Lifestyle diseases are associated with the way of people living. They include obesity, diabetes, atherosclerosis, heart disease, stroke and diseases associated with smoking and alcohol and drug abuse to name, but a few. Obesity has always been major health hazard and plays a central role in the pathogenesis of Type 2 diabetes. This type of diabetes accounts for at least 90 percent of all cases of diabetes and occurs when the body either stops producing enough insulin for its needs or becomes resistant to the effect of insulin produced [1, 2, 4, 5, 6].

In this article, we develop and analyze a model that investigates the dynamics of Type 2 diabetes induced by the prevalence of obesity within

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a population. A system of nonlinear ordinary differential equations is derived in order to predict the evolution of both obesity and diabetes in the population subdivided into five compartments.

The model is presented in Section 2 with a description of all the parameters and a representation of the flow between the various compartments. In Section 3, mathematical well-posedness of the model is explored.

In particular, positivity and boundedness of the solution will be established. Existence of equilibria and the computation of the basic reproduction ratio are established in section 4. Thereafter stability of both disease free and endemic equilibria are investigated in section 5, followed by concluding remarks and future direction.

2. The model

In this section, a five compartmental model is formulated to capture the dynamics of diabetes. It consists of a following system of nonlinear differential equations:

$$\begin{cases} \dfrac{dS}{dt} &= \Lambda - Sg(E) - (\mu+\alpha)S, \\ \dfrac{dE}{dt} &= Sg(E) - (\mu+\beta)E, \\ \dfrac{dI}{dt} &= \beta E - (\delta+\gamma+\mu)I, \\ \dfrac{dT}{dt} &= \gamma I - \mu T, \\ \dfrac{dR}{dt} &= \alpha S - \mu R, \end{cases} \tag{1}$$

endowed with initial conditions

$$
\begin{aligned}
S(0) &\equiv S_0 > 0, \\
E(0) &\equiv E_0 > 0, \\
I(0) &\equiv I_0 \geq 0, \\
T(0) &\equiv T_0 \geq 0, \text{ and} \\
R(0) &\equiv R_0 \geq 0,
\end{aligned}
$$

where $S(t)$, $E(t)$, $I(t)$, $T(t)$, and $R(t)$ denote susceptible individuals, obese individuals, people afflicted with diabetes and not receiving treatment, people affected with diabetes and undertaking medication, people on healthy diet, respectively, at time t. The parameters in the evolution system (1) are described as follows:

Table 1: **Biological meaning of parameters**

Parameters	**Description**
Λ	recruitment rate into the susceptible population
μ	natural death rate
α	the rate at which individuals embrace healthy diets
β	the rate at which obese individuals develop diabetes
γ	the rate at which people affected with diabetes get treated
δ	diabetes induced death rate

Following [3], it is assumed that obesity incidence is a nonlinear response to the size of obese population, taking the form $Sg(E)$, where the function $g(E)$ is positive. It is further assumed that

$$
\begin{aligned}
(\mathrm{H}_1) \quad &: \quad g(0) = 0,\ g'(0) > 0,\ g''(E) \leq 0 \ \text{ for } E \geq 0, \\
(\mathrm{H}_2) \quad &: \quad \lim_{E \to 0^+} \frac{g(E)}{E} = k,\ 0 < k < \infty.
\end{aligned}
$$

The flow chart of the above model is given by:

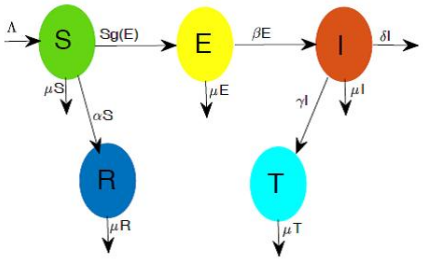

Figure 1: Flow Diagram

Note that the total population $N(t)$ is given by $S(t)+E(t)+I(t)+T(t)+R(t)$. The rate of change of the total population by adding all the equations considered in (1) is:

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I \leq \Lambda - \mu N. \tag{2}$$

Clearly, whenever $N(t) > \dfrac{\Lambda}{\mu}$, we have that $\dfrac{dN}{dt} < 0$ implying that we have a decrease in the size of the total population. Next we present a systematic analysis of our evolution equation.

3. Mathematical analysis

We start by ensuring that the model (1) is mathematically well-posed. Given the fact that the variables represent biologically densities, it is important to show that all the variables remain positive at all time.

Lemma 1. *For any non-negative initial conditions* $(S_0, E_0, I_0, T_0, R_0)$*, system (1) has a local solution which is unique.*

Proof. Let $x = (S,\ E,\ I,\ T,\ R)$, system (1) can be rewritten as $x'(t) = f(x(t))$, where $f : \mathbb{R}^5 \to \mathbb{R}^5$ is a C^1 vector field. By the classical differential equation theory, we can confirm that system (1) has a unique local solution defined in a maximum interval $[0, t_m)$. □

Lemma 2. *For any non-negative initial conditions* $(S_0, E_0, I_0, T_0, R_0)$*, the solution of (1) is non-negative and bounded for all* $t \in [0, t_m)$*.*

Proof. We start by showing positivity of the local solution for any non-negative initial conditions. It is easy to see that $S(t) \geq 0$ for all $t \in [0, t_m)$. Indeed, assume the contrary and let $t_1 > 0$ be the first time such that $S(t_1) = 0$ and $S'(t_1) \leq 0$.

From the first equation of the system (1), we have $S'(t_1) = \Lambda > 0$, which presents a contradiction. Therefore $s(t) \geq 0$ for all $t \in [0, t_m)$. Using the same argument, positivity $E(t)$, $I(t)$, $T(t)$ and $R(t)$ in the interval $[0, t_m)$ are established.

Furthermore, from (2), we have that

$$0 < N(t) \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu t} < \frac{\Lambda}{\mu} + N(0).$$

Therefore the solution $N(t)$ is bounded in the interval $[0, t_m)$. □

Theorem 1. *For any non-negative initial conditions* $(S_0, E_0, I_0, T_0, R_0)$*, system (1) has a unique global solution. Moreover, this solution is non-negative and bounded for all* $t \geq 0$*.*

Proof. The solution does not blow up in a finite time as it is bounded, it is therefore defined at all time $t \geq 0$. Other properties of the solution follow from Lemma (1) and Lemma (2). □

4. Equilibria and basic reproduction number

4.1. The basic reproduction number

The basic reproduction number denoted as R_0, is a threshold parameter describing the asymptotical behavior of the disease in the population. In this section, we will derive the basic reproduction number of the evolution equation by making use of the next generation matrix method formulated

by Van den Driessche and Watmough [7].

Let x be the transpose of (E, I, T, S, R). We rewrite system (1) in the matrix form

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x),$$

where

$$\mathcal{F}(x) = \begin{bmatrix} \mathcal{F}_1 \\ \mathcal{F}_2 \\ \mathcal{F}_3 \\ \mathcal{F}_4 \\ \mathcal{F}_5 \end{bmatrix} = \begin{bmatrix} Sg(E) \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \text{ and}$$

$$\mathcal{V}(x) = \begin{bmatrix} \mathcal{V}_1 \\ \mathcal{V}_2 \\ \mathcal{V}_3 \\ \mathcal{V}_4 \\ \mathcal{V}_5 \end{bmatrix}$$

$$= \begin{bmatrix} (\mu+\beta)E \\ (\delta+\gamma+\mu)I - \beta E \\ \mu T - \gamma I \\ Sg(E) + (\mu+\alpha)S - \Lambda \\ \mu R - \alpha S \end{bmatrix}.$$

It is obvious that the disease free equilibrium is

$$(E^0, I^0, T^0, S^0, R^0) \equiv \left(0, 0, 0, \frac{\Lambda}{\mu+\alpha}, \frac{\alpha\Lambda}{\mu(\mu+\alpha)}\right). \tag{3}$$

Following [7], we have that

$$\mathcal{R}_0 = \rho(FV^{-1}),$$

where

$$F = \left[\begin{array}{ccc} \dfrac{\partial \mathcal{F}_1}{\partial E} & \dfrac{\partial \mathcal{F}_1}{\partial I} & \dfrac{\partial \mathcal{F}_1}{\partial T} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{array} \right] \Bigg|_{(E^0, I^0, T^0, S^0, R^0)}$$

$$= \left[\begin{array}{ccc} \dfrac{k\Lambda}{\mu + \alpha} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{array} \right],$$

$$V = \left[\begin{array}{ccc} \dfrac{\partial \mathcal{V}_1}{\partial E} & \dfrac{\partial \mathcal{V}_1}{\partial I} & \dfrac{\partial \mathcal{V}_1}{\partial T} \\ \dfrac{\partial \mathcal{V}_2}{\partial E} & \dfrac{\partial \mathcal{V}_2}{\partial I} & \dfrac{\partial \mathcal{V}_2}{\partial T} \\ \dfrac{\partial \mathcal{V}_3}{\partial E} & \dfrac{\partial \mathcal{V}_3}{\partial I} & \dfrac{\partial \mathcal{V}_3}{\partial T} \end{array} \right] \Bigg|_{(E^0, I^0, T^0, S^0, R^0)}$$

$$= \left[\begin{array}{ccc} \mu + \beta & 0 & 0 \\ -\beta & \delta + \gamma + \mu & 0 \\ 0 & -\gamma & \mu \end{array} \right]$$

and ρ is the spectral radius of the matrix FV^{-1}.

It follows that

$$\mathcal{R}_0 = \frac{k\Lambda}{(\mu + \alpha)(\mu + \beta)}. \tag{4}$$

4.2. Existence of an endemic equilibrium

In this subsection, we explore the existence of an endemic equilibrium.

Proposition 1. *Assume $\mathcal{R}_0 > 1$, then system (1) has a unique endemic equilibrium $E^* = (S^*, E^*, I^*, T^*, R^*)$.*

Proof. The endemic equilibrium is obtained by solving the algebraic equa-

tion

$$\begin{cases} 0 &= \Lambda - Sg(E) - (\mu+\alpha)S, \\ 0 &= Sg(E) - (\mu+\beta)E, \\ 0 &= \beta E - (\delta+\gamma+\mu)I, \\ 0 &= \gamma I - \mu T, \\ 0 &= \alpha S - \mu R. \end{cases} \tag{5}$$

From the last four equations of (5), we have that

$$\begin{aligned} S &= \frac{(\mu+\beta)E}{g(E)}, \\ I &= \frac{\beta E}{\delta+\gamma+\mu}, \\ T &= \frac{\gamma\beta E}{\mu(\delta+\gamma+\mu)} \text{ and} \\ R &= \frac{\alpha(\mu+\beta)E}{\mu g(E)}. \end{aligned} \tag{6}$$

Substituting into the first equation of (5), we have that

$$g(E) = \frac{(\mu+\alpha)(\mu+\beta)E}{\Lambda-(\mu+\beta)E} := h(E). \tag{7}$$

Note that $E = 0$ yields the disease free equilibrium.

It is clear that $E = \dfrac{\Lambda}{\mu+\beta}$ is a vertical asymptote for the function $h(E)$. Also for $E > \dfrac{\Lambda}{\mu+\beta}$, we have that $g(E) > 0$ and $h(E) < 0$, so there is no solution for (7). Assuming that $0 < E < \dfrac{\Lambda}{\mu+\beta}$, we have

$$\begin{aligned} h'(E) &= \frac{\Lambda(\mu+\alpha)(\mu+\beta)}{[\Lambda-(\mu+\beta)E]^2} > 0 \text{ and} \\ h'(E) &= \frac{2\Lambda(\mu+\alpha)(\mu+\beta)^2}{[\Lambda-(\mu+\beta)E]^3} > 0. \end{aligned}$$

It follows that $h(E)$ is concave upward on the interval $0 < E < \dfrac{\Lambda}{\mu+\beta}$ and $h(E) \to \infty$ as $E \to \dfrac{\Lambda}{\mu+\beta}$.

From the assumption (H_1) on the function g, $g(0) = h(0) = 0$ and $g(E)$ is concave downward. Therefore the two functions $h(E)$ and $g(E)$ intersect at a unique point E^* within the interval $\left(0, \frac{\Lambda}{\mu+\beta}\right)$. □

5. Stability analysis

Next we investigate stability of both the disease free equilibrium and the endemic equilibrium.

5.1. Stability of the disease free equilibrium

Theorem 2. *The disease-free equilibrium is globally asymptotically stable if* $0 < \mathcal{R}_0 < 1$*, and unstable if* $\mathcal{R}_0 > 1$*.*

Proof. The jacobian matrix of system (1) at the disease free equilibrium is given as

$$\begin{pmatrix} -(\mu+\alpha) & -\frac{k\Lambda}{\mu+\alpha} & 0 & 0 & 0 \\ 0 & \frac{k\Lambda}{\mu+\alpha} - (\mu+\beta) & 0 & 0 & 0 \\ 0 & \beta & -(\delta+\mu+\gamma) & 0 & 0 \\ 0 & 0 & \gamma & -\mu & 0 \\ \alpha & 0 & 0 & 0 & -\mu \end{pmatrix}.$$

Solving the characteristic equations, we obtain the eigenvalues $-\mu$, $-(\mu+\alpha)$ and $-(\delta+\gamma+\mu)$ that are all strictly negative.

Note that the eigenvalue $-\mu$ has a geometric multiplicity of order 2. In addition, we obtain the eigenvalue $\frac{k\Lambda}{\mu+\alpha} - (\mu+\beta)$ which is strictly negative if $0 < \mathcal{R}_0 < 1$ and strictly positive if $\mathcal{R}_0 > 1$.

It follows that the disease free equilibrium is unstable if $\mathcal{R}_0 > 1$ and locally asymptotically stable if $0 < \mathcal{R}_0 < 1$. The global asymptotical

stability of the disease free equilibrium stems from the fact that there is no endemic equilibrium in the system when $0 < \mathcal{R}_0 < 1$. □

5.2. Stability of the endemic equilibrium

Lemma 3. *Let $g(E)$ be a positive smooth function defined on the interval $[0, \infty)$. Suppose that assumptions H_1 and H_2 hold, then following inequality is satisfied*

$$1 - \frac{Eg'(E)}{g(E)} \geq 0 \text{ for any } E > 0. \tag{8}$$

Proof. We have that

$$\frac{d[g(E) - Eg'(E)]}{dE} = -Eg''(E) \geq 0$$

as $g''(E) \leq 0$. This implies that the function $g(E) - Eg'(E)$ is increasing on the interval $[0, \infty)$.

Given the fact that $g(0) - 0g'(0) = 0$, it follows that

$$g(E) - Eg'(E) \geq 0.$$ □

Theorem 3. *If $\mathcal{R}_0 > 1$, then the endemic equilibrium $E^* = (S^*, E^*, I^*, T^*, R^*)$ is locally asymptotically stable.*

Proof. For the endemic equilibrium $E^* = (S^*, E^*, I^*, T^*, R^*)$, the Jacobian matrix is

$$\begin{pmatrix} -g(E^*) - (\mu + \alpha) & -S^*g'(E^*) & 0 & 0 & 0 \\ g(E^*) & S^*g'(E^*) - (\mu + \beta) & 0 & 0 & 0 \\ 0 & \beta & -(\delta + \mu + \gamma) & 0 & 0 \\ 0 & 0 & \gamma & -\mu & 0 \\ \alpha & 0 & 0 & 0 & -\mu \end{pmatrix}.$$

The characteristic equation of this matrix is given by

$$(\lambda+\mu)^2(\lambda+\delta+\mu+\gamma)[(\lambda+\mu+\beta-S^*g'(E^*))(\lambda+\mu+\alpha+g(E^*))$$
$$+S^*g'(E^*)g(E^*)]=0.$$

This implies that

$$(\lambda+\mu)^2(\lambda+\delta+\mu+\gamma)(\lambda^2+a_1\lambda+a_2)=0, \tag{9}$$

where

$$a_1=2\mu+\alpha+\beta+g(E^*)-S^*g'(E^*)$$

and

$$a_2=(\mu+\beta)(\mu+\alpha)+(\mu+\beta)g(E^*)-(\mu+\alpha)S^*g'(E^*).$$

It is obvious that equation (9) has real roots $\lambda_1=-\mu<0$, $\lambda_2=-(\delta+\mu+\gamma)<0$, with other roots being the solutions of the equation

$$\lambda^2+a_1\lambda+a_2=0.$$

Making use of Lemma 3 and the fact that

$$S^*=\frac{(\mu+\beta)E^*}{g(E^*)},$$

we have that

$$a_1=\mu+\alpha+g(E^*)+(\mu+\beta)\left[1-\frac{E^*}{g(E^*)}g'(E^*)\right]>0$$

and

$$a_2=(\mu+\beta)g(E^*)+(\mu+\alpha)(\mu+\beta)\left[1-\frac{E^*}{g(E^*)}g'(E^*)\right]>0. \qquad \square$$

6. Concluding remarks and future work

A system of nonlinear ordinary differential equations with a nonlinear incidence response was derived in order to predict the evolution of obesity

and diabetes in a population. Existence, uniqueness, boundedness and positivity of the solution of the system were discussed.

In particular, existence of equilibria and the computation of the basic reproduction ratio were established. Furthermore, stability of both disease free and endemic equilibria were investigated thanks to spectral methods and Lyapunov stability theory.

A future extension of this work will be to perform numerical simulations on the system (1) for different sets of parameters and different set of initial points in order to order to validate the analytical results obtained.

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