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Some issues on the mathematical modeling of population dynamics using differential equations

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Abstract

Mathematical modeling is a useful and widely used method in epidemiology. The paper highlights a number of mistakes or issues in the formulation of the previous population models using differential equations. The issues are the lack of mathematical models of Non-Communicable Diseases (NCDs), the wrongly formulated mathematical terms, the absence of time delay and the ill-posedness of governing equations. Finally, some brief results of a preliminary study which addresses the issues, is provided.

1 Introduction

A mathematical model is a representation or approximation in mathematical terms of the behavior of the real system under study. The motivations behind mathematical modeling are to understand and provide predictions of the behavior of the system. Population dynamics is the study of how and why populations, as dynamical systems, change with respect to time. The system

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will be described by a number of variables and a set of differential equations that establish the relationships between the variables. The purpose of this paper is to highlight several issues in the formulation of the previous population models of non-communicable diseases (NCDs). It is important to note that the scope of this paper is the mathematical models using differential equations.

2 The Issues of Previous Mathematical Models of NCDs

The first issue is the lack of mathematical models of NCDs. NCDs are diseases which are not passed from person to person. They are currently leading causes of morbidity and mortality worldwide and major Public Health challenges which increase the burden of infective diseases, especially for developing or low-and-middle income countries. One potential approach to address the issue is by developing mathematical models using differential equations, to understand the population dynamics of NCDs and assess the efficacy and effectiveness of different preventive measures and control programmes. Unlike infectious diseases, mathematical modelling using differential equations is seldom applied to NCDs. To date, there are only ten models of NCDs population in the literature, and all models focus on diabetes mellitus only. The governing equations of the models are as follows.

Model 1:

An age structured model for complications of diabetes mellitus [1]

$$\frac{\partial r}{\partial t} + \frac{\partial r}{\partial a} = \delta r^2 - \varepsilon r + \omega, \tag{2.1}$$

where $\delta = \delta(a,t)$,$\varepsilon = \varepsilon(a,t) = p(a,t)s(a,t) + e(a,t)q(a,t) + \delta(a,t) + \alpha(a,t)$, and $r(a,t)$ is the rate of developing complications, $d(a,t)$ is the death rate of diabetic individuals without complications, $d^{'}(a,t)$ is the death rate of diabetic individuals with complications ($d^{'} = d + \delta$), $s(a,t)$ is the survival rate of diabetic individuals without complication ($s = 1 - d$), $e(a,t)$ is the survival rate of diabetic individuals with complications ($e = 1 - d^{'}$), $p(a,t)$ is the rate at which complications are developed, $q(a,t)$ is the rate at which complications are cured, $\alpha_1(a,t)$ is the rate of incidence of diabetes without

complication, $\alpha_2(a,t)$ is the rate of incidence of diabetes with complication, $\alpha(a,t)$ is the rate of global incidence of diabetes ($\alpha = \alpha_1 + \alpha_2$), and $\delta(a,t)$ is the death rate due to complications.

Model 2:

A model for the burden of diabetes and its complications [2]

$$\frac{dN(t)}{dt} = I - (\nu + \delta)C(t) - \mu N(t), \tag{2.2a}$$

$$\frac{dC(t)}{dt} = -(\lambda + \theta)C(t) + \lambda N(t). \tag{2.2b}$$

where I is the incidence rate of diabetes mellitus, $N(t)$ is the total number of diabetic individuals ($N(t) = D(t) + C(t)$), $D(t)$ and $C(t)$ are the numbers of diabetic individuals without complications and with complications at time t, respectively, μ is the death rate due to non-diabetic related causes, λ is the rate of diabetic individuals who develop complications, γ is the rate of diabetic individuals whose complications are cured, ν is the rate of diabetic individuals who become severely disabled, δ is the rate of diabetic individuals who die from their complications and $\theta = \gamma + \mu + \nu + \delta$.

The dual optimal controls of this model are carried out in [3] and [4]. In [3], the transition of diabetic individuals to develop complications and vice-versa is controlled by optimal control u_1 and u_2, respectively. The objective function for dual optimal controls is expressed as:

$$F(u_1, u_2) = \int_0^T C + Au_1^2 + Bu_2^2 \quad dt, \tag{2.3}$$

where A and B are positive weights that balance the size of u_1 and u_2. Optimal control u_1 intended to reduce the transition of D into C, while u_2 intended to enhance the curable rate of complication. Meanwhile, in [4], the dual objective functional is defined as:

$$F(u_1, u_2) = \int_0^T k_1 C + \frac{1}{2}k_2 u_1^2 + \frac{1}{2}k_3 u_2^2 \quad dt, \tag{2.4}$$

where k_1, k_2, and k_3 are the relative weights to balance each terms in the integrand and avoid domination of any terms. The optimal control u_1 prevents some of the diabetic individuals from developing complications by the effective medical management, while u_2 controls diabetic individuals with complication and improve the curable rate using effective medical treatment.

Model 3:

A non-linear model of diabetes mellitus [5]

Substituting $\lambda = \beta\frac{C(t)}{N(t)}$ into Model (2.2):

$$\frac{dN(t)}{dt} = I - (\nu + \delta)C(t) - \mu N(t), \tag{2.5a}$$

$$\frac{dC(t)}{dt} = (\beta - \theta)C(t) - \beta\frac{C^2}{N}. \tag{2.5b}$$

where $\beta > 0$ is a real constant and the variables and parameters are as defined in **Model 2**).

Model 4:

A model of diabetes and pre-diabetes

Without optimal control

[6]

$$\frac{dE(t)}{dt} = I - (\lambda_1 + \mu + \lambda_3)E(t), \tag{2.6a}$$

$$\frac{dD(t)}{dt} = \lambda_1 E(t) - (\lambda_2 + \mu)D(t) + \gamma C(t), \tag{2.6b}$$

$$\frac{dC(t)}{dt} = \lambda_2 D(t) + \lambda_3 E(t) - (\gamma + \mu + \nu + \delta)C(t). \tag{2.6c}$$

where I is the incidence rate of pre-diabetics, $E(t)$, $C(t)$ and $D(t)$ are the number of pre-diabetic, diabetic individuals with complications and without complications at time t, respectively, μ is the death rate due to non-diabetic related causes, λ_1 is the rate of developing diabetic without complications, λ_2 is the rate of developing complication from the diabetic stage, λ_3 is the rate of developing diabetes at the stage of complications, γ is the rate at which complications are cured, ν is the rate at which patients with complications become severely disabled and δ is the mortality rate due to complications.

With optimal control

[7]

$$\frac{dE(t)}{dt} = I - (\mu + (\beta_1 + \beta_3)(1 - u(t)))E(t), \tag{2.7a}$$

$$\frac{dD(t)}{dt} = \beta_1(1 - u(t))E(t) - (\mu + \beta_2(1 - u(t)))D(t) + \gamma C(t), \tag{2.7b}$$

$$\frac{dC(t)}{dt} = \beta_3(1 - u(t))E(t) + \beta_2(1 - u(t))D(t) - (\mu + \gamma + \nu + \delta)D(t). \tag{2.7c}$$

where control $u(t)$ is defined as:

$$F(u) = \int_0^T D(t) + C(t) + Au^2(t) \quad dt. \tag{2.8}$$

and β_1 is the rate of developing diabetic without complications, β_2 is the rate of developing complications from the diabetic stage and β_3 is the rate of developing diabetes at the stage of complications and other parameters are as defined in [6].

Model 5:

The dynamics of a population of healthy people, pre-diabetics and diabetics with and without complications with optimal control [8]

Without optimal control

$$\frac{dP}{dt} = n - (I_1 + I_2 + I_3 + \mu)P + \gamma_1 E, \tag{2.9a}$$

$$\frac{dE}{dt} = I_1 P - (\gamma_1 + \beta_1 + \beta_3 + \mu)E, \tag{2.9b}$$

$$\frac{dD}{dt} = I_2 P + \beta_1 E + \gamma_2 C - (\beta_2 + \mu)D, \tag{2.9c}$$

$$\frac{dC}{dt} = I_3 P + \beta_2 D + \beta_3 E - (\gamma_2 + \mu + \delta)C. \tag{2.9d}$$

where n denotes the incidence of adult population, $P(t)$, $E(t)$, $D(t)$ and $C(t)$ are the numbers of healthy, pre-diabetic, diabetic individuals without, and with complications at time t, respectively, I_1 is the rate of healthy individuals to become pre-diabetic, I_2 is the rate of healthy individuals to become

diabetic, I_3 is the rate of healthy individuals developing complications, μ is the natural mortality rate, β_1 is the rate of pre-diabetic individual to become diabetic, β_2 is the rate of diabetic individual developing complications, β_3 is the probability of a pre-diabetic individual developing complications, γ_1 is the rate at which a pre-diabetic individual become healthy, γ_2 is the rate of curable complications and δ is the mortality rate due to complications.

With optimal control

$$\frac{dP}{dt} = n - I_1(1-u)P - (I_2 + I_3 + \mu)P + \gamma_1 E, \tag{2.10a}$$

$$\frac{dE}{dt} = I_1(1-u)P - (\gamma_1 + \beta_1 + \beta_3 + \mu)E, \tag{2.10b}$$

$$\frac{dD}{dt} = I_2 P + \beta_1 E + \gamma_2 C - (\beta_2 + \mu)D, \tag{2.10c}$$

$$\frac{dC}{dt} = I_3 P + \beta_2 D + \beta_3 E - (\gamma_2 + \mu + \delta)C. \tag{2.10d}$$

with the objective functional defined as:

$$F(u) = \int_0^T E(t) + Au^2(t) \quad dt. \tag{2.11}$$

where A is a positive weight that balances the size of the terms.

Model 6:

A model for determining age-specific diabetes incidence and prevalence using body mass index[9].

$$\frac{dQ_{H(1)}}{dt} = F^{birth} + F^{migr}_{H(1)} - F_{H(1)\_D(1)} - F^{death}_{H(1)} - F^{aging}_{H(1)}, \tag{2.12}$$

$$\frac{dQ_{D(1)}}{dt} = F_{H(1)\_D(1)} + F^{migr}_{D(1)} - F^{death}_{D(1)} - F^{aging}_{D(1)}, \tag{2.13}$$

$$\frac{dQ_{H(2)}}{dt} = F^{aging}_{H(1)} + F^{migr}_{H(2)} - F_{H(2)\_D(2)} - F^{death}_{H(2)}, \tag{2.14}$$

$$\frac{dQ_{D(2)}}{dt} = F_{H(2)\_D(2)} + F^{aging}_{D(1)} + F^{migr}_{D(2)} - F^{aging}_{D(2)} - F^{death}_{D(2)}, \tag{2.15}$$

$$\frac{dQ_{H(3)}}{dt} = F^{aging}_{H(2)} + F^{aging}_{H(3)} + F_{H(3)\_D(3)} - F^{death}_{H(3)} - F^{aging}_{H(3)}, \tag{2.16}$$

$$\frac{dQ_{D(3)}}{dt} = F_{H(3)\_D(3)} + F^{aging}_{D(2)} + F^{migr}_{D(3)} - F^{aging}_{D(3)} - F^{death}_{D(3)}, \tag{2.17}$$

$$\frac{dQ_{H(4)}}{dt} = F^{aging}_{H(3)} + F^{migr}_{H(4)} - F_{H(4)\_D(4)} - F^{death}_{H(4)}, \tag{2.18}$$

$$\frac{dQ_{D(4)}}{dt} = F_{H(4)\_D(4)} + F^{aging}_{D(3)} + F^{migr}_{D(4)} - F^{death}_{D(4)}. \tag{2.19}$$

where $H(i)$ is the number of nondiabetic individuals, and $D(i)$ is the number of diabetic individuals for $i = 1, 2, 3, 4$. F is the flow of individuals in (positive sign), and out (negative sign) of a compartment. For more details, see [9].

Model 7:

A model on the impact of media coverage in controlling diabetes [10].

$$\begin{aligned}
\frac{dP}{dt} &= A - (\lambda_1 + \lambda_3)P - \lambda PM + \lambda_0 P_M - \mu P,\\
\frac{dD}{dt} &= \lambda_1 P - \lambda_2 D + \gamma C - dD,\\
\frac{dC}{dt} &= \lambda_2 D + \lambda_3 P - (\gamma + d + \nu + \delta)C,\\
\frac{dP_M}{dt} &= \lambda PM - dP_M - \lambda_0 P_M,\\
\frac{dM}{dt} &= \mu(D + C) - \mu_0 M,
\end{aligned} \tag{2.20}$$

where A is the incidence of pre-diabetic class, the number of individuals of pre-diabetic, diabetic individuals without, and with complications at time t are denoted as $P(t)$, $D(t)$ and $C(t)$, respectively, $M(t)$ is the cumulative density of awareness programs driven by the media in that region, $P_M(t)$ is the number of aware prediabetic individuals at time t, λ_1 is the probability of developing diabetes, d the natural mortality rate, λ_2 is the probability of a diabetic class developing a complication, λ_3 is the probability of a pre-diabetic class developing a complication, γ is the rate at which complications are cured, ν is the rate at which diseased patients with complications become severely disabled, δ is the mortality rate due to complications, λ is the dissemination rate of awareness among susceptible class in a population due to which they form a different class, μ is the rate of awareness program being implemented, μ_0 is the depletion rate of awareness programs due to ineffectiveness, social problems in the population and λ_0 is the rate of transfer of aware class to susceptible class.

Model 8:

Existence and characterization of optimal control [11].

$$\begin{aligned}
\frac{dP}{dt} &= \rho - (1-u)\sigma_1 P - (\sigma_2 + \sigma_3 + \mu)P + \gamma_1 E, \\
\frac{dE}{dt} &= (1-u)\sigma_1 P - (\gamma_1 + \beta_1 + \beta_3 + \mu)E + \gamma_2 D, \\
\frac{dD}{dt} &= \sigma_2 P + \beta_1 E + \gamma_3 C - (\beta_2 + \gamma_2 + \nu_2 + \mu)D, \\
\frac{dC}{dt} &= \sigma_3 P + \beta_2 D + \beta_3 E - (\delta + \gamma_3 + \mu_1 + \mu)C, \\
\frac{dB}{dt} &= \mu_2 D + \mu_1 C - (\tau + \mu)Q,
\end{aligned} \tag{2.21}$$

where ρ is the incidence of healthy adult population, $P(t), E(t), D(t), C(t)$ and $B(t)$ respectively are the numbers of healthy people, pre-diabetics and diabetics without complications, diabetics with complications and diabetics become disabled at time t. The rate of healthy persons to become diabetic without complication and diabetic with complication, is σ_2 and σ_3, respectively. μ is the natural mortality rate. γ_1 and σ_1 are the rates of a pre-diabetic person becomes healthy and vice versa. γ_2 is the rate of a diabetic person to become pre-diabetic, and γ_3 is the rate of a diabetic with complications become diabetic without complications. μ_1 is the rate of a diabetic person become disabled and μ_2 is the rate of a diabetic with complication person become disabled. β_1 is the probability of a pre-diabetic person to become diabetic. β_2 is the probability of a diabetic person developing a complications. β_3 is the probability of a pre-diabetic person developing a complication. τ and δ are the mortality rate due to disabled and complications, respectively.

Model 9:

A model with lifestyle and genetic factors [12]

$$\frac{dS}{dt} = \alpha S + \alpha(1-\rho)(D+C) - \beta S\frac{D}{N} - \mu S, \tag{2.22a}$$

$$\frac{dD}{dt} = \alpha\rho(D+C) + \beta S\frac{D}{N} - (\lambda + \mu)D + \gamma C, \tag{2.22b}$$

$$\frac{dC}{dt} = \lambda D - (\gamma + \delta + \mu)C. \tag{2.22c}$$

where $S(t)$, $D(t)$ and $C(t)$ are the numbers of susceptible individuals of lifestyle "transmission", diabetic individuals without complication and with

complications at time t, respectively, α is the birth rate, ρ is the proportion of genetic disorder's birth related to diabetes, β is the rate of interaction causing diabetes incidence, λ is the rate of developing complications, γ is the rate of complications which are cured, δ is the rate of diabetes-induced death mortality and μ is the natural mortality rate.

Model 10:

A model to assess the diabetes screening and reporting programs and project the burden of undiagnosed diabetes [13].

The population is divided according to gender and age with 101 annual interval from 0 to 100 years old. Let C_{ga} be the number of people with gender g at age a and time t. C_{ga} is divided into three health status: healthy (C_{ga}^{H}), undiagnosed diabetes ($C_{ga}^{DM_{un}}$), and diagnosed diabetes (C_{ga}^{DM}). The rates of change in C_{ga}^{H}, $C_{ga}^{DM_{un}}$, and C_{ga}^{DM} are represented by ordinary differential equations (see [13] for further details).

The second issue is the aforementioned models did not take the time delay into consideration. The models utilise ordinary differential equations (ODEs) [2, 5, 7, 8, 12] and partial differential equations (PDE) [1]. Delay differential equations (DDEs) are never employed despite its advantages in modelling time-dependent biological processes. It is imperative to explicitly incorporate these delay times into mathematical models of NCDs due to the following facts. Firstly, NCDs are of long duration and generally slow progression. They are mostly chronic diseases which takes years to develop; hence, the complication of unhealthy lifestyles, for example, will not manifest in the short term. Secondly, NCDs may also have different incidence and mortality rates for different age groups [14]. Individuals with different ages may have different maturation time and take different time to develop complications, which are crucial in control and prevention of NCDs. Thirdly, the population dynamics of NCDs also involve threshold phenomena, that is, phenomena which express the transition of an individual through different stages [15].

The third issue is the mismatch between the physical meaning of formulated mathematical terms and the actual population dynamics. In the population models of infectious diseases, the presence of nonlinear terms is due to the interaction between state variables (which shows the infectious nature of the disease). However, this is not the case for non-communicable pathology, as the disease may not be transferred from one person to another on the basis of being in close contact with the patients. Example of such

mistakes can be found in the following term in [5]

$$\lambda = \lambda(t) = \beta \frac{C(t)}{N(t)} \tag{2.23}$$

where the variables and parameters are as defined in **Model 2** and **Model 3**. This term implies the number of diabetic patients who develop complication is jointly dependent to the number of diabetic patients without complication $D(t)$ and to the number of diabetic patients with complications $C(t)$. That is, the model wrongly assumes that diabetic complications is 'infectious' due to the close contact with patients with complications. Furthermore, the health educations and campaigns and the apparent complications of the behaviors (health or social problems) may discourage an individual to adopt the unhealthy lifestyles to avoid similar life complications.

The fourth issue is the mathematical and epidemiological well-posedness of the population models (i.e positivity and boundedness of solutions). Since the dependent variables in the population models denote physical quantities, we require that solutions that start from positive (nonnegative) initial conditions remain positive (nonnegative) for all time. Furthermore, a population should not be able to continue growing indefinitely and not exceed total population (i.e bounded). Differential equation models must be well posed to be mathematically relevant and biologically significant. A differential equation model is said to be well-posed if, through every point (initial condition), there exists a unique solution. There are population models using ODEs in the literature which are not proven to be well-posed, that is, the proof of mathematical and epidemiological well-posedness are not provided (see for examples [1, 2, 5, 16]). Even worse, under further investigation, the model in [16] is found to be ill-posed, because there are positive initial data that may provide negative solutions (e.g: initial values from $[0, 1]X[0, 1]$ may go beyond this region). The governing equations of the model are as follows

$$\frac{dX}{dt} = rx(t) + abm(1 - x(t - \tau_1))y(t - \tau_1)e^{-r\tau_1}, \tag{2.24a}$$

$$\frac{dY}{dt} = \mu y(t) + acx(t - \tau_2)(1 - y(t - \tau_2))e^{-\mu\tau_2}. \tag{2.24b}$$

where $X(t)$ and $Y(t)$ are the proportion of infected humans and mosquitoes at time t, respectively, m is the number of mosquitoes per human host, a is the biting rate on humans by a single mosquito (number of bites per unit time), b is the proportion of infected bites on humans that produce an infection, τ_1 is the incubation period in a human; r is the per capita rate of recovery in

humans, μ is the per capita rate of mortality in vectors, τ_2 is the incubation interval in the mosquito, and c is the transmission efficiency from human to mosquito,

3 The Preliminary Study

Recently, the author has been awarded a research grant by Ministry of Education, Malaysia on the mathematical modelling and stability analysis of several common NCDs using DDEs. New mathematical models of two NCDs using ODEs have been proposed and analyzed, and the findings have been published recently [17, 18]. It is found that both models have a stable equilibrium. Therefore, we believe that two of the aforementioned issues (i.e: the lack of differential equation models of NCDs and the linearity of the models) are being addressed. The governing equations of the models can easily be extended to account for the delay by introducing a constant or state-dependent delay in the known models. However, the models require different proof of mathematical well-posedness. Furthermore, the stability of the corresponding DDE models need to be studied further because, in general, DDEs exhibit much more complicated dynamics than ODEs, since time delay could cause a stable equilibrium to become unstable.

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