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# DYNAMICS OF A THREE-STATE DIABETIC POPULATION MODEL WITH A TIME DELAY AND LIMITED MEDICAL RESOURCES

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**Abstract** Diabetes is a chronic disease that can cause various long-term complications. This study proposes a diabetic model with a time delay and a saturating recovery rate of complications. The model considered two crucial factors of diabetic dynamics: the delay in the first incidence of complications and the limited medical resources. By analyzing the distribution of roots of the characteristic equation with respect to the delay model, the conditions for local stability and the properties of Hopf bifurcation were obtained. When the critical delay $\tau_0$ exists, the equilibrium point is locally asymptotically stable for $\tau < \tau_0$, while at $\tau = \tau_0$, a periodic orbit arises due to Hopf bifurcation. Furthermore, for the non-delay case, the global stability of the equilibrium point was proved by constructing a suitable Lyapunov function. Some numerical simulations were provided to validate the analytical results. Sensitivity analysis shows that the most sensitive model input is the incidence rate of diabetes. The model can provide information concerning the total number of people with diabetes and its complications and predict the outcomes of various model inputs. The results suggest that decreasing the incidence rates of diabetes and its complications and the inhibition effect due to limited medical resources are valid approaches to controlling the prevalence of diabetes and its complications.

## 1 Introduction

Diabetes mellitus, in short diabetes, is a life-long disease caused by hyperglycemia (high blood glucose levels) due to defects in either lack of insulin or cells resisting the insulin or both. In 2021, the International Diabetes Federation [1] estimated that 6.7 million adults died due to diabetes and its complications. Diabetes and its consequences cost the world economy USD966 billion. In Malaysia, the estimated percentage of diabetics among adults aged 18 and above has increased from 11.6% in 2006 to 18.3% in 2019 [2]. As a result, about 3.9 million Malaysian adults were living with diabetes in 2019. In 2021, Malaysia's total diabetes-related health expenditure was estimated at USD4833.5 million, with USD1090.7 per individual with diabetes [3]. If the number of people with diabetes is not carefully handled, many problems will arise, including an increased number of patients requiring complication treatments such as dialysis, kidney or heart transplant, and rehabilitation.

Regarding the scope and methodology used in the population models, Mat Daud highlighted various problems in the formulation of population models using ordinary differential equations [4, 5, 6]. Several models of non-communicable diseases (NCDs) have been studied including diabetes [7, 8], hypertension [9], thyroid disorders [10], and anaemia [11]. Nasir and Mat Daud [8] also emphasized the importance of considering time delay in the progression of diabetes.

**Table 1.** Definition of the symbols in Figure 2.

| Symbol | Definition | Dimension |
|---|---|---|
| $D(t)$ | number of diabetics who never had any complications at time $t$ | individuals |
| $D_c(t)$ | number of diabetics with complications at time $t$ | individuals |
| $D_p(t)$ | number of diabetics with recovered complications at time $t$ | individuals |
| $I$ | the incidence rate of diabetes | individuals time$^{-1}$ |
| $\gamma$ | rate of the first incidence of complications | time$^{-1}$ |
| $\tau$ | time delay in the first incidence of complications | time |
| $\kappa$ | the recovery rate of complications | time$^{-1}$ |
| $\beta$ | non-negative parameter measuring the limited medical resources | individuals$^{-1}$ |
| $\sigma$ | the recurrence rate of complications | time$^{-1}$ |
| $\mu_1$ | the diabetes-related mortality rate among diabetics without complications | time$^{-1}$ |
| $\mu_2$ | the diabetes-related mortality rate among diabetics with complications | time$^{-1}$ |
| $\mu_3$ | the diabetes-related mortality rate among diabetics with recovered complications | time$^{-1}$ |
| $\mu$ | the mortality rate due to causes other than diabetes | time$^{-1}$ |

As in [12], the author considered a delay parameter representing the delay in the first incidence of complications. Introducing this delay parameter is significant because, for example, approximately 8.9% of Malaysian adults in 2019 did not know they had diabetes [2]. If these individuals do not screen their glucose levels, they might remain unknown diabetics for several years until the first complication. In the present research, we will extend the model studied by Nasir [12] by considering the limited medical resources at the stage of treatment of complications.

The organization of the paper is as follows. Section 2 focuses on a brief discussion of the delay model proposed by Nasir [12], followed by the formulation of a delay model with a saturating recovery rate of complications. In Section 3, we focus on the qualitative analysis of the corresponding non-delay model. In Section 4, the qualitative analysis of the delay model is studied. In Section 5, we conduct sensitivity analysis and present the sensitivity indices at the equilibrium solutions. Numerical simulations are provided in Section 6 to validate the analytical results.

## 2 The mathematical model

### 2.1 A diabetic model with a time delay

Nasir [12] developed a diabetes population model with a time delay in the first incidence of complications. The governing equations of the Nasir [12] model are

$$\frac{dD(t)}{dt} = I - \gamma D(t-\tau) - (\mu + \mu_1)D(t), \tag{2.1a}$$

$$\frac{dD_c(t)}{dt} = \gamma D(t-\tau) - (\kappa + \mu + \mu_2)D_c(t) + \sigma D_p(t), \tag{2.1b}$$

$$\frac{dD_p(t)}{dt} = \kappa D_c(t) - (\sigma + \mu + \mu_3)D_p(t), \tag{2.1c}$$

where all variables and parameters are defined in Table 1.

### 2.2 Model formulation with a saturating recovery rate of complications

We modify model (2.1) by considering the effect of limited medical resources on treating diabetes-related complications. In model (2.1), Nasir [12] assumed the recovery rate of complications of

the form $h(D_c(t)) = \kappa D_c(t)$. It means the resources are sufficient for any complications. The recovery rate of diabetics with complications will be unaffected if the number of diabetics with complications increases. This assumption is unsatisfactory because not every country has enough resources for the treatment of complications of diabetes [13]. Therefore, we can improve this assumption by considering a more realistic assumption of limited medical resources.

We propose a saturating recovery rate of complications of the form $h(D_c(t)) = \frac{\kappa D_c(t)}{1+\beta D_c(t)}$, where $\kappa$ is the recovery rate of complications and $\beta$ is a non-negative parameter measuring the limited medical resources [14]. One example of medical resources is kidney transplantation for diabetics with end-stage kidney disease. The unavailability of this treatment may cause the patients to undergo an alternative treatment, such as hemodialysis, three times a week [15]. This term $h(D_c(t)) = \frac{\kappa D_c(t)}{1+\beta D_c(t)}$ will be replacing the term $h(D_c(t)) = \kappa D_c(t)$ in model (2.1). If $\beta = 0$, the saturating recovery rate reverts to the linear one: $h(D_c(t)) = \kappa D_c(t)$, representing the unlimited medical resources. Figure 1 shows the difference between a linear recovery rate and a saturating recovery rate using parameter values $\kappa = 0.95$ time$^{-1}$ and $\beta = 0.0002$ individuals$^{-1}$ (these values are chosen as an example). Using the saturating recovery rate seems more reasonable because it grows slower than the linear recovery rate due to limited medical resources.

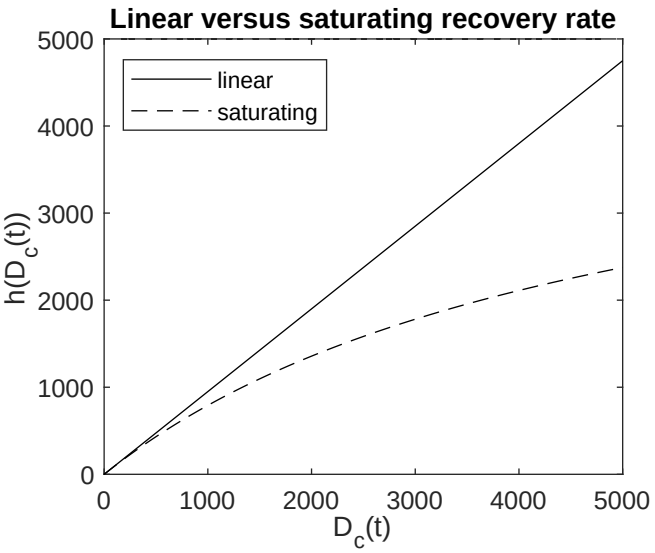



**Figure 1.** The graph of $h(D_c(t)) = \kappa D_c(t)$ versus $h(D_c(t)) = \frac{\kappa D_c(t)}{1+\beta D_c(t)}$, where $\kappa = 0.95$ time$^{-1}$ and $\beta = 0.0002$ individuals$^{-1}$.

Taking the above assumption into consideration, we develop a flow diagram of diabetic population dynamics in Figure 2.

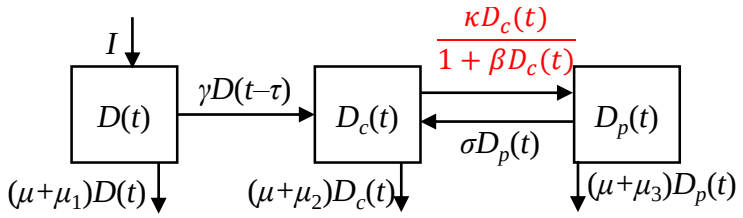



**Figure 2.** Three-state diabetic population. The red color represents the new contribution and the difference from model (2.1).

We assume all parameters are positive because they represent population dynamics. The

dynamics in Figure 2 are governed as follows:

$$\frac{dD(t)}{dt} = I - \gamma D(t-\tau) - (\mu+\mu_1)D(t), \tag{2.2a}$$

$$\frac{dD_c(t)}{dt} = \gamma D(t-\tau) - \frac{\kappa D_c(t)}{1+\beta D_c(t)} + \sigma D_p(t) - (\mu+\mu_2)D_c(t), \tag{2.2b}$$

$$\frac{dD_p(t)}{dt} = \frac{\kappa D_c(t)}{1+\beta D_c(t)} - (\sigma+\mu+\mu_3)D_p(t). \tag{2.2c}$$

The initial conditions of model (2.2) are

$$D(\varrho) = \phi(\varrho) > 0,\ \varrho \in [-\tau, 0],\ D_c(0) = D_{c0} > 0,\ D_p(0) = D_{p0} > 0,$$

where $\phi(\varrho)$ is a continuous function on $\varrho \in [-\tau, 0]$. The total population size with respect to model (2.2) is denoted as $N(t) = D(t) + D_c(t) + D_p(t)$. For simplifying the notations, the variables $D$, $D_c$, and $D_p$ are evaluated at time $t$, unless the argument is other than $t$ (for example, $D_c(0)$ and $D(t-\tau)$).

## 3 Qualitative analysis of the non-delay model

Model (2.2), under the assumption of instantaneous dynamics (that is, $\tau = 0$), is

$$\frac{dD}{dt} = I - (\gamma+\mu+\mu_1)D, \tag{3.1a}$$

$$\frac{dD_c}{dt} = \gamma D - \frac{\kappa D_c}{1+\beta D_c} + \sigma D_p - (\mu+\mu_2)D_c(t), \tag{3.1b}$$

$$\frac{dD_p}{dt} = \frac{\kappa D_c}{1+\beta D_c} - (\sigma+\mu+\mu_3)D_p, \tag{3.1c}$$

with the initial conditions:

$$D(0) = D_0 > 0,\ D_c(0) = D_{c0} > 0,\ D_p(0) = D_{p0} > 0.$$

### 3.1 Basic properties of the non-delay model (3.1)

#### Non-negativity and boundedness of the solutions

**Proposition 3.1.** *The solutions $D$, $D_c$, and $D_p$ of the non-delay model (3.1) remain non-negative and bounded for all $t > 0$. Furthermore, the region:*

$$\Omega = \left\{ \left(D, D_c, D_p\right) \in \mathbb{R}^3_+ \middle| D + D_c + D_p \le \frac{I}{\mu} \right\},$$

*is a positively-invariant region with respect to the non-delay model (3.1).*

*Proof.* For the non-negativity of the solution $D$ of the non-delay model (3.1), from Equation (3.1a), we obtain:

$$\frac{dD}{dt} + (\gamma+\mu+\mu_1)D = I. \tag{3.2}$$

Since $I \ge 0$, Equation (3.2) becomes:

$$\frac{dD}{dt} + (\gamma+\mu+\mu_1)D \ge 0. \tag{3.3}$$

The integrating factor is given as $e^{(\gamma+\mu+\mu_1)t}$. By multiplying inequality (3.3) with the integrating

factor and changing the variable $t$ to variable $\varepsilon$, we obtain:

$$\frac{d}{d\varepsilon}\left[D(\varepsilon)e^{(\gamma+\mu+\mu_1)\varepsilon}\right] \geq 0. \tag{3.4}$$

Integrating both sides of inequality (3.4) from $\varepsilon = 0$ to $\varepsilon = t$ gives:

$$D(t) \geq D(0)e^{-(\gamma+\mu+\mu_1)t}.$$

Therefore, the solution $D$ remains non-negative for all $t > 0$.

Since the solution is assumed to be a continuous and differentiable function, the functions $D_c(t), D_p(t)$ cannot become negative without crossing the axes $D_c = 0, D_p = 0$.

Let $t^* = \min\{t_c, t_p\}$ where $D_c(t_c) = 0, D_p(t_p) = 0$ and $t_c, t_p \geq 0$. If $t^* = t_c$ then $D_c(t_c) = 0$ and the functions $D_p(t_c) > 0$ at $t = t_c$, yields

$$\frac{dD_c}{dt}(t^* = t_c) = \gamma D(t) + \sigma D_p(t) \geq 0$$

Therefore, when the function $D_c(t)$ touches the axis $D_c = 0$, $\frac{dD_c}{dt}$ is always non-negative, and the function $D_c(t)$ will not decrease and will never cross to negative part. Using similar argument, it can be shown that

$$\frac{dD_p}{dt}(t^* = t_p) = \frac{\kappa D_c(t)}{1 + \beta D_c(t)} \geq 0$$

Thus, all solutions $D_c(t), D_p(t)$ are always non-negative for all $t > 0$.

For the boundedness of the solutions $D$, $D_c$, and $D_p$ of the non-delay model (3.1), adding all equations in the non-delay model (3.1) yields:

$$\frac{dN}{dt} = I - \mu N - \mu_1 D - \mu_2 D_c - \mu_3 D_p. \tag{3.5}$$

Since the terms $\mu_1 D \geq 0$, $\mu_2 D_c \geq 0$, and $\mu_3 D_p \geq 0$, Equation (3.5) becomes:

$$\frac{dN}{dt} + \mu N \leq I. \tag{3.6}$$

The integrating factor is given as $e^{\mu t}$. Multiplying inequality (3.6) with the integrating factor and changing the variable $t$ to variable $\varepsilon$, we obtain:

$$\frac{d}{d\varepsilon}\left[N(\varepsilon)e^{\mu\varepsilon}\right] \leq Ie^{\mu\varepsilon}. \tag{3.7}$$

By integrating both sides of inequality (3.7) from $\varepsilon = 0$ to $\varepsilon = t$, we obtain:

$$N(t) \leq \frac{I}{\mu} + \left(N(0) - \frac{I}{\mu}\right)e^{-\mu t}.$$

If $N(0) \leq \frac{I}{\mu}$, then we have $N(t) \leq \frac{I}{\mu}$. This implies that the upper bound of the total population size is $\frac{I}{\mu}$. If $N(0) > \frac{I}{\mu}$, then $N(t)$ will decrease to $\frac{I}{\mu}$ because $\lim_{t\to\infty} N(t) = \frac{I}{\mu}$.

The region $\Omega$ is a positively-invariant region with respect to the non-delay model (3.1). □

### 3.2 Stability analysis

#### Equilibrium point

Let $E^* = (D^*, D_c^*, D_p^*)$ be the equilibrium point of the non-delay model (3.1). By setting every equation of the non-delay model (3.1) equal to zero we have

$$I - (\gamma + \mu + \mu_1)D^* = 0, \tag{3.8a}$$

$$\gamma D^* - \frac{\kappa D_c^*}{1+\beta D_c^*} + \sigma D_p^* - (\mu+\mu_2)D_c^* = 0, \tag{3.8b}$$

$$\frac{\kappa D_c^*}{1+\beta D_c^*} - (\sigma+\mu+\mu_3)D_p^* = 0. \tag{3.8c}$$

From Equation (3.8c), we have $D_p^* = \dfrac{\kappa D_c^*}{(1+\beta D_c^*)(\sigma+\mu+\mu_3)}$. From Equation (3.8a), we have $D^* = \dfrac{I}{\gamma+\mu+\mu_1}$. From Equation (3.8b), we have:

$$z_1 D_c^{*2} + z_2 D_c^* + z_3 = 0, \tag{3.9}$$

where

$$z_1 = \beta(\mu+\mu_2)(\sigma+\mu+\mu_3), \tag{3.10a}$$

$$z_2 = (\mu+\mu_2)(\mu+\sigma+\mu_3) + \kappa(\mu+\mu_3) - \beta\gamma(\sigma+\mu+\mu_3)D^*, \tag{3.10b}$$

$$z_3 = -\gamma(\sigma+\mu+\mu_3)D^*. \tag{3.10c}$$

The roots of Equation (3.9) are:

$$\frac{-z_2 + \sqrt{z_2^2 - 4z_1z_3}}{2z_1}, \tag{3.11}$$

and

$$\frac{-z_2 - \sqrt{z_2^2 - 4z_1z_3}}{2z_1}. \tag{3.12}$$

Notice that $z_1$ and $z_3$ have opposite signs, we have $\sqrt{z_2^2 - 4z_1z_3} > \sqrt{z_2^2} = |z_2|$. Regardless of the sign of $z_2$ ($z_2$ can be either positive or negative), the root in (3.11) is always positive, while the root in (3.12) is always negative.

Since the state variables represent individuals in the population, the only biologically meaningful equilibrium point is

$$E^* = \left(\frac{I}{\gamma+\mu+\mu_1}, \frac{-z_2 + \sqrt{z_2^2 - 4z_1z_3}}{2z_1}, \frac{\kappa D_c^*}{(1+\beta D_c^*)(\sigma+\mu+\mu_3)}\right), \tag{3.13}$$

where $z_1$, $z_2$, and $z_3$ are given in Equations (3.10).

**Local stability**

**Theorem 3.2.** *$E^*$ is locally asymptotically stable.*

*Proof.* The characteristic equation with respect to the equilibrium point $E^*$ of the non-delay model (3.1) is obtained by computing:

$$\det[\lambda_1 \mathbf{I}_3 - \mathbf{Y}_1] = 0, \tag{3.14}$$

where $\lambda_1$ represents the eigenvalues, $\mathbf{I}_3$ is an identity matrix of dimension 3,

$$\mathbf{Y}_1 = \begin{bmatrix} -(\gamma+y_1) & 0 & 0 \\ \gamma & -y_2 & \sigma \\ 0 & y_3 & -y_4 \end{bmatrix},$$

and

$$y_1 = \mu + \mu_1,\ y_2 = y_3 + \mu + \mu_2,\ y_3 = \frac{\kappa}{(1+\beta D_c^*)^2},\ y_4 = \sigma + \mu + \mu_3. \tag{3.15}$$

After expanding Equation (3.14), we obtain

$$\xi_1(\lambda_1)(\lambda_1 + \gamma + y_1) = 0, \tag{3.16}$$

where

$$\xi_1(\lambda_1) = \lambda_1^2 + (y_2 + y_4)\lambda_1 + y_3(\mu + \mu_3) + y_4(\mu + \mu_2).$$

One of the roots of Equation (3.16) is $-(\gamma + y_1)$, which is negative because $\gamma + y_1 > 0$. The other two roots of Equation (3.16) are determined by $\xi_1(\lambda_1) = 0$ or:

$$\lambda_1^2 + (y_2 + y_4)\lambda_1 + y_3(\mu + \mu_3) + y_4(\mu + \mu_2) = 0. \tag{3.17}$$

By the Routh-Hurwitz criteria for a polynomial of degree two [16], all roots of Equation (3.17) are negative or have negative real parts because $y_2 + y_4 > 0$ and $y_3(\mu + \mu_3) + y_4(\mu + \mu_2) > 0$. As a result, $E^*$ is locally asymptotically stable. □

Theorem 3.2 indicates that the small displacement from $E^*$ will decrease to zero without restriction on any positive parameter values. No periodic orbit can appear for the non-delay model (3.1).

**Global stability**

Constructing an appropriate Lyapunov function to investigate global stability is known to be a difficult problem in general. In the following, we discuss two remarks, where Remark 3.3 is an example of an attempt to find a Lyapunov function, and Remark 3.4 is the numerical simulation to indicate the global stability of $E^*$.

**Remark 3.3.** A function is suggested as follows:

$$\begin{aligned}\zeta(D, D_c, D_p) = {} & \frac{(D - D^*)^2}{\gamma + \mu + \mu_1} + (2\mu + \mu_2 + \mu_3)(D_c - D_c^*)^2 \\ & + \sigma[(D_c - D_c^*) + (D_p - D_p^*)]^2,\end{aligned} \tag{3.18}$$

where $\zeta(D, D_c, D_p) \geq 0$ for all $(D, D_c, D_p) \geq (0, 0, 0)$ with equality if and only if $(D, D_c, D_p) = (D^*, D_c^*, D_p^*)$. The time derivative of $\zeta$ computed along the solutions of the non-delay model (3.1) is

$$\begin{aligned}\frac{d\zeta}{dt} = {} & \frac{2(D - D^*)}{\gamma + \mu + \mu_1}\Big(I - (\gamma + \mu + \mu_1)D\Big) \\ & + \Big[2(2\mu + \mu_2 + \mu_3)(D_c - D_c^*) + 2\sigma(D_c - D_c^*) + 2\sigma(D_p - D_p^*)\Big] \\ & \times \Big(\gamma D - \frac{\kappa D_c}{1 + \beta D_c} + \sigma D_p - (\mu + \mu_2)D_c\Big) \\ & + \Big[2\sigma(D_c - D_c^*) + 2\sigma(D_p - D_p^*)\Big]\Big(\frac{\kappa D_c}{1 + \beta D_c} - (\sigma + \mu + \mu_3)D_p\Big).\end{aligned} \tag{3.19}$$

Note that we have:

$$\begin{aligned} I &= (\gamma + \mu + \mu_1)D^*, \\ 0 &= -\gamma D^* + \frac{\kappa D_c^*}{1 + \beta D_c^*} - \sigma D_p^* + (\mu + \mu_2)D_c^*, \\ 0 &= -\frac{\kappa D_c^*}{1 + \beta D_c^*} + (\sigma + \mu + \mu_3)D_p^*. \end{aligned}$$

Therefore, Equation (3.19) becomes:

$$\begin{aligned}\frac{d\zeta}{dt} &= \frac{2(D-D^*)}{\gamma+\mu+\mu_1}\Big((\gamma+\mu+\mu_1)D^* - (\gamma+\mu+\mu_1)D\Big)\\ &\quad + \Big[2(2\mu+\mu_2+\mu_3+\sigma)(D_c-D_c^*) + 2\sigma(D_c-D_c^*)\Big]\Big(\gamma D - \frac{\kappa D_c}{1+\beta D_c}\\ &\quad + \sigma D_p - (\mu+\mu_2)D_c - \gamma D^* + \frac{\kappa D_c^*}{1+\beta D_c^*} - \sigma D_p^* + (\mu+\mu_2)D_c^*\Big)\\ &\quad + \Big[2\sigma(D_c-D_c^*) + 2\sigma(D_p-D_p^*)\Big]\Big(\frac{\kappa D_c}{1+\beta D_c} - (\sigma+\mu+\mu_3)D_p\\ &\quad - \frac{\kappa D_c^*}{1+\beta D_c^*} + (\sigma+\mu+\mu_3)D_p^*\Big)\\ &= -2(D-D^*)^2 + 2(2\mu+\mu_2+\mu_3+\sigma)\gamma(D-D^*)(D_c-D_c^*)\\ &\quad - \frac{2(2\mu+\mu_2+\mu_3)\kappa(D_c-D_c^*)^2}{(1+\beta D_c)(1+\beta D_c^*)} - 2(2\mu+\mu_2+\mu_3+\sigma)(\mu+\mu_2)(D_c-D_c^*)^2\\ &\quad + 2\sigma\gamma(D-D^*)(D_p-D_p^*) - 2\sigma(\mu+\mu_3)(D_p-D_p^*)^2.\end{aligned}$$

The term $\frac{d\zeta}{dt} \leq 0$ for all $(D, D_c, D_p) \geq (0,0,0)$ with equality if and only if $(D, D_c, D_p) = (D^*, D_c^*, D_p^*)$ is not satisfied because the terms $2(2\mu+\mu_2+\mu_3+\sigma)\gamma(D-D^*)(D_c-D_c^*)$ and $2\sigma\gamma(D-D^*)(D_p-D_p^*)$ are not less than or equal to zero for all $(D, D_c, D_p) \geq (0,0,0)$. Therefore, the function given in Equation (3.18) in this Remark 3.3 does not serve as a suitable Lyapunov function for establishing the global stability of the equilibrium point $(D, D_c, D_p) = (D^*, D_c^*, D_p^*)$.

**Remark 3.4.** As shown in Remark 3.3, it is not easy to show the global stability of $E^*$ of the non-delay model (3.1) by the Lyapunov direct method. We will use numerical simulations to indicate the global stability of $E^*$ of the non-delay model (3.1). Consider the following particular case of the non-delay model (3.1):

$$\frac{dD}{dt} = 50 - 0.07D, \tag{3.20a}$$

$$\frac{dD_c}{dt} = 0.01D - \frac{0.8D_c}{1+0.0005D_c} + 0.4D_p - 0.16D_c, \tag{3.20b}$$

$$\frac{dD_p}{dt} = \frac{0.8D_c}{1+0.0005D_c} - 0.51D_p. \tag{3.20c}$$

System (3.20) has the equilibrium point $E^* = (714.2857, 21.5988, 33.5186)$. Figure 3 shows the numerical simulations of model (3.20) with four sets of initial conditions $(D_0, D_{c0}, D_{p0})$: $(800, 100, 100)$, $(600, 150, 50)$, $(1000, 300, 110)$, and $(750, 50, 300)$. Figure 3 shows the global stability of $E^*$, where the solutions $D$, $D_c$, and $D_p$ of model (3.20) converge to $E^*$ as time $t$ increases regardless of any positive initial conditions. The numerical simulations indicate that the equilibrium point $E^*$ of the non-delay model (3.1) may be globally asymptotically stable.

## 4 Qualitative analysis of the delay model

### 4.1 Basic properties of the delay model (2.2)

**Non-negativity and boundedness of the solutions**

**Proposition 4.1.** *If condition:*

(H1) $I > \gamma D(t_1 - \tau)$ *at the boundary* $D(t_1) = 0$ *for any time* $t_1$,

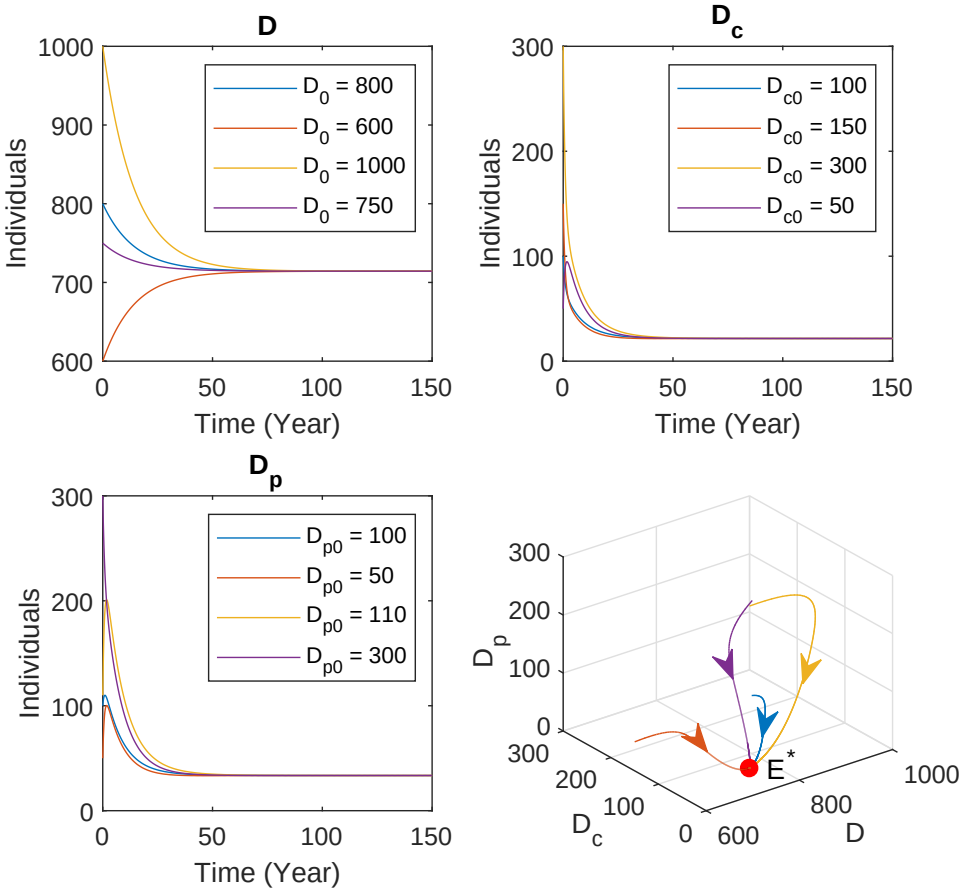



**Figure 3.** Numerical simulations of model (3.20).

*is satisfied, then the solutions $D$, $D_c$, and $D_p$ of the delay model (2.2) remain non-negative and bounded for all $t > 0$. Furthermore, the region:*

$$\Omega = \left\{ \left( D, D_c, D_p \right) \in \mathbb{R}^3_+ \middle| D + D_c + D_p \leq \frac{I}{\mu} \right\},$$

*is a positively-invariant region with respect to the delay model (2.2).*

Condition (H1) means that in any event that the number of diabetics who never had any complications drops to zero, the number of diabetics who will develop the first complication should be less than the number of newly developed diabetics.

*Proof.* We prove the non-negativity of variable $D$ of the delay model (2.2) as follows. $D(t) > 0$ for all $t > 0$. If this be not the case, suppose that there exists $t_1 > 0$ such that $D(t) > 0$ for $t \in [0, t_1)$, $D(t_1) = 0$, and $\dfrac{dD(t_1)}{dt} \leq 0$. From Equation (2.2a), we obtain:

$$\frac{dD(t_1)}{dt} = I - \gamma D(t_1 - \tau).$$

We have $\dfrac{dD(t_1)}{dt} \leq 0$ for $I \leq \gamma D(t_1 - \tau)$, which agrees to the supposition that $\dfrac{dD(t_1)}{dt} \leq 0$. Therefore, if $I \leq \gamma D(t_1 - \tau)$ at the boundary $D(t_1) = 0$ for any time $t_1$, the solution $D$ may enter the negative region. Condition (H1) indicates that the solution $D$ remains non-negative for all $t > 0$.

In the following, condition (H1) is satisfied.

Since variables $D_c$ and $D_p$ of the delay model (2.2) depend on each other, we prove the non-negativity as follows. $D_c(t) > 0$ for all $t > 0$. If this be not the case, suppose that there exists $t_2 > 0$ such that $D_c(t) > 0$ for $t \in [0, t_2)$, $D_c(t_2) = 0$, and $\frac{dD_c(t_2)}{dt} \leq 0$. We first find the integration of Equation (2.2c). From Equation (2.2c), we obtain:

$$\frac{dD_p}{dt} + (\sigma + \mu + \mu_3)D_p = \frac{\kappa D_c}{1 + \beta D_c}. \tag{4.1}$$

The integrating factor is given as $e^{(\sigma+\mu+\mu_3)t}$. By multiplying Equation (4.1) with the integrating factor and changing the variable $t$ to variable $\varepsilon$, we obtain:

$$\frac{d}{d\varepsilon}\left[D_p(\varepsilon)e^{(\sigma+\mu+\mu_3)\varepsilon}\right] = \frac{\kappa D_c(\varepsilon)}{1 + \beta D_c(\varepsilon)}e^{(\sigma+\mu+\mu_3)\varepsilon}. \tag{4.2}$$

Integrating both sides of Equation (4.2) from $\varepsilon = 0$ to $\varepsilon = t$ gives:

$$D_p(t) = D_p(0)e^{-(\sigma+\mu+\mu_3)t} + e^{-(\sigma+\mu+\mu_3)t}\int_0^t \frac{\kappa D_c(\varepsilon)}{1 + \beta D_c(\varepsilon)}e^{(\sigma+\mu+\mu_3)\varepsilon}\, d\varepsilon. \tag{4.3}$$

From Equation (4.3), we have $D_p(t) > 0$ for $t \in [0, t_2]$. Then, from Equation (2.2b), we have:

$$\frac{dD_c(t_2)}{dt} = \gamma D(t_2 - \tau) + \sigma D_p(t_2) > 0,$$

but this leads to a contradiction to the supposition that $\frac{dD_c(t_2)}{dt} \leq 0$. We can conclude that the solution $D_c$ remains non-negative for all $t > 0$. Consequently, from Equation (4.3), the solution $D_p$ also remains non-negative for all $t > 0$.

For the boundedness of the solutions $D$, $D_c$, and $D_p$ of the delay model (2.2), adding all equations in the delay model (2.2) yields:

$$\frac{dN}{dt} = I - \mu N - \mu_1 D - \mu_2 D_c - \mu_3 D_p. \tag{4.4}$$

Since the terms $\mu_1 D \geq 0$, $\mu_2 D_c \geq 0$, and $\mu_3 D_p \geq 0$, Equation (4.4) becomes:

$$\frac{dN}{dt} + \mu N \leq I. \tag{4.5}$$

The integrating factor is given as $e^{\mu t}$. By multiplying inequality (4.5) with the integrating factor and changing the variable $t$ to variable $\varepsilon$, we obtain:

$$\frac{d}{d\varepsilon}\left[N(\varepsilon)e^{\mu\varepsilon}\right] \leq Ie^{\mu\varepsilon}. \tag{4.6}$$

By integrating both sides of inequality (4.6) from $\varepsilon = 0$ to $\varepsilon = t$, we obtain:

$$N(t) \leq \frac{I}{\mu} + \left(N(0) - \frac{I}{\mu}\right)e^{-\mu t}.$$

If $N(0) \leq \frac{I}{\mu}$, then we have $N(t) \leq \frac{I}{\mu}$. This implies that the upper bound of the total population size is $\frac{I}{\mu}$. If $N(0) > \frac{I}{\mu}$, then $N(t)$ will decrease to $\frac{I}{\mu}$ because $\lim_{t\to\infty} N(t) = \frac{I}{\mu}$.

As a result, condition (H1) is required to be true so that the region $\Omega$ becomes a positively-invariant region with respect to the delay model (2.2). The proposition is proposed. □

### 4.2 Stability analysis

#### Equilibrium point

The equilibrium point $E^* = (D^*, D_c^*, D_p^*)$ given in Equation (3.13) is also the equilibrium point of the delay model (2.2) because when the solutions $D$, $D_c$, and $D_p$ of the delay model (2.2) reach equilibrium, we have:

$$D(t) = D(t-\tau) = D^*,\ D_c(t) = D_c^*,\ D_p(t) = D_p^*. \tag{4.7}$$

#### Local stability and the occurrence of Hopf bifurcation

**Theorem 4.2.** *For the delay model (2.2):*

*(i) If condition:*

(H2) $y_1 \geq \gamma$,

*holds, then $E^*$ is locally asymptotically stable for $\tau \geq 0$.*

*(ii) if condition:*

(H3) $y_1 < \gamma$,

*holds, then there exists a critical value:*

$$\tau_0 = \frac{1}{\omega_0}\cos^{-1}\left\{-\frac{y_1}{\gamma}\right\}, \tag{4.8}$$

*where*

$$\omega_0 = \sqrt{-(y_1+\gamma)(y_1-\gamma)}, \tag{4.9}$$

*such that $E^*$ is locally asymptotically stable for $\tau \in [0, \tau_0)$ and becomes unstable for $\tau > \tau_0$. Furthermore, a Hopf bifurcation occurs at $\tau = \tau_0$, and a family of periodic orbits arises from $E^*$.*

Condition (H2) means that the rate of the first incidence of complications is no more than the total death rate of diabetics who never had any complications. Meanwhile, condition (H3) means that the rate of the first incidence of complications is greater than the total death rate of diabetics who never had any complications.

*Proof.* The characteristic equation with respect to the equilibrium point $E^*$ of the delay model (2.2) is obtained by computing:

$$\det[\lambda_2 \mathbf{I}_3 - \mathbf{Y}_2 - e^{-\lambda_2\tau}\mathbf{Y}_3] = 0, \tag{4.10}$$

where $\lambda_2$ represents the eigenvalues, $\mathbf{I}_3$ denotes an identity matrix of dimension 3,

$$\mathbf{Y}_2 = \begin{bmatrix} -y_1 & 0 & 0 \\ 0 & -y_2 & \sigma \\ 0 & y_3 & -y_4 \end{bmatrix},\ \mathbf{Y}_3 = \begin{bmatrix} -\gamma & 0 & 0 \\ \gamma & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \tag{4.11}$$

and $y_1$, $y_2$, $y_3$, and $y_4$ are the same as in Equations (3.15). After expanding Equation (4.10), we obtain:

$$\xi_1(\lambda_2)\xi_2(\lambda_2) = 0, \tag{4.12}$$

where

$$\xi_1(\lambda_2) = \lambda_2^2 + (y_2+y_4)\lambda_2 + y_3(\mu+\mu_3) + y_4(\mu+\mu_2),$$
$$\xi_2(\lambda_2) = \lambda_2 + y_1 + e^{-\lambda_2\tau}\gamma.$$

Two of the roots of Equation (4.12) are determined by $\xi_1(\lambda_2) = 0$. According to the Routh-Hurwitz criteria, they are negative or have negative real parts because $y_2 + y_4 > 0$ and $y_3(\mu + \mu_3) + y_4(\mu + \mu_2) > 0$. The other roots of Equation (4.12) are determined by $\xi_2(\lambda_2) = 0$ or:

$$\lambda_2 + y_1 + e^{-\lambda_2 \tau}\gamma = 0. \tag{4.13}$$

Assume that for some $\tau > 0$, $\lambda_2 = i\omega$ ($i = \sqrt{-1}$ and $\omega > 0$) is one of the roots of Equation (4.13). Substituting $\lambda = i\omega$ into Equation (4.13) and separating the real and imaginary parts yields:

$$y_1 = -\gamma \cos \omega\tau, \tag{4.14a}$$

$$\omega = \gamma \sin \omega\tau. \tag{4.14b}$$

By squaring and adding both sides of Equations (4.14), we obtain:

$$\omega^2 + (y_1 + \gamma)(y_1 - \gamma) = 0. \tag{4.15}$$

Condition (H2) indicates that $\omega^2 \leq 0$, which is a contradiction because we initially assumed $\omega > 0$. Consequently, Equation (4.13) cannot have $\lambda_2 = i\omega$ as one of the roots. Hence, $E^*$ is locally asymptotically stable for $\tau \geq 0$. This proves Theorem 4.2(i).

For the second proof, suppose that condition (H3) holds. Condition (H3) implies that equation (4.15) has one positive root, that is, Equation (4.9). Then, the corresponding critical delays are

$$\tau^{(j)} = \frac{1}{\omega_0} \cos^{-1}\left\{ -\frac{y_1}{\gamma} \right\} + \frac{2j\pi}{\omega_0}, \quad j = 0, 1, 2, \ldots.$$

Let $\tau_0 = \tau^{(0)}$ be the first critical value at which Equation (4.13) has roots on the imaginary axis, and $\lambda_2 = \pm i\omega_0$ are the corresponding roots. Then, $E^*$ is locally asymptotically stable for $\tau \in [0, \tau_0)$. Moreover, to establish the occurrence of Hopf bifurcation at $\tau = \tau_0$ [17], the following transversality condition must be satisfied:

$$\operatorname{sign}\left\{ \Re\left[ \frac{d\lambda(\tau)}{d\tau} \right]\Big|_{\lambda(\tau_0)=i\omega_0} \right\} > 0.$$

Taking the derivative of Equation (4.13) with respect to $\tau$ yields:

$$\left[ \frac{d\lambda}{d\tau} \right]^{-1} = \frac{e^{\lambda\tau}}{\gamma\lambda} - \frac{\tau}{\lambda}. \tag{4.16}$$

Evaluating Equation (4.16) at $\lambda(\tau_0) = i\omega_0$ and taking its real part yields:

$$\Re\left[ \frac{d\lambda}{d\tau} \right]^{-1}\Big|_{\lambda(\tau_0)=i\omega_0} = \frac{\sin \omega_0\tau_0}{\gamma\omega_0}. \tag{4.17}$$

Using Equation (4.14b), we have $\sin \omega_0\tau_0 = \dfrac{\omega_0}{\gamma}$. Then, Equation (4.17) becomes:

$$\Re\left[ \frac{d\lambda}{d\tau} \right]^{-1}\Big|_{\lambda(\tau_0)=i\omega_0} = \frac{1}{\gamma^2}.$$

Hence, we obtain:

$$\operatorname{sign}\left\{ \Re\left[ \frac{d\lambda}{d\tau} \right]^{-1}\Big|_{\lambda(\tau_0)=i\omega_0} \right\} = \operatorname{sign}\left\{ \Re\left[ \frac{d\lambda}{d\tau} \right]\Big|_{\lambda(\tau_0)=i\omega_0} \right\} > 0.$$

□

**Remark 4.3.** We discussed the criteria implying that a periodic orbit arises for the delay model (2.2). Such results are interesting in situations where the equilibrium point $E^*$ of the delay model (2.2) can become unstable. For some parameter values, the solutions $D$, $D_c$, and $D_p$ of the delay model (2.2) do not approach $E^*$. Therefore, $E^*$ of the delay model (2.2) is not globally asymptotically stable, and the Lyapunov function method would not work.

**Properties of Hopf bifurcation with respect to the delay model (2.2)**

We obtained the conditions which guarantee that model (2.2) undergoes a Hopf bifurcation at the equilibrium point $E^*$ when $\tau = \tau_0$. We determine the direction, stability, and period of Hopf bifurcation of model (2.2) with respect to the critical value $\tau_0$ by using the normal form method and center manifold theory [18]. In the following, we consider that model (2.2) undergoes a Hopf bifurcation; condition (H3) holds.

**Theorem 4.4.** *For the delay model (2.2), assume that (H3) holds. We have:*

*(i) The sign of $\chi_2$ determines the direction of the Hopf bifurcation: if $\chi_2 > 0$ ($\chi_2 < 0$), then the Hopf bifurcation is supercritical (subcritical) and the bifurcating periodic solutions exist for $\tau > \tau_0$ ($\tau < \tau_0$).*

*(ii) The sign of $\beta_2$ determines the stability of the bifurcating periodic solutions: if $\beta_2 < 0$ ($\beta_2 > 0$), then the bifurcating periodic solutions are stable (unstable).*

*(iii) The sign of $T_2$ determines the period of the bifurcating periodic solutions: if $T_2 > 0$ ($T_2 < 0$), then the period increases (decreases).*

*Where the terms $\chi_2$, $\beta_2$, and $T_2$ are given in Equations (4.23), (4.24), and (4.25), respectively.*

*Proof.* Let $\tau = \tau_0 + \chi$, where $\chi \in \mathbb{R}$. Then, $\chi = 0$ is the Hopf bifurcation value of model (2.2). We rescale time $t \to (t/\tau)$ obtaining $\tau = 1$ in the new time variable. Model (2.2) can be transformed into a functional differential equation in $C = C([-1, 0], \mathbb{R}^3)$ as follows:

$$\frac{d\mathbf{x}(t)}{dt} = \mathbf{L}_\chi(\mathbf{x}_t) + \mathbf{F}(\chi, \mathbf{x}_t), \tag{4.18}$$

where $\mathbf{x}_t(\Theta) = [x_{1t}(\Theta), x_{2t}(\Theta), x_{3t}(\Theta)]^\top$, $\Theta \in [-1, 0]$, and $x_{it}(\Theta) = x_i(t + \Theta)$, $\forall i$. The terms $\mathbf{L}_\chi : C \to \mathbb{R}^3$ and $\mathbf{F} : \mathbb{R} \times C \to \mathbb{R}^3$ are

$$\mathbf{L}_\chi(\boldsymbol{\rho}) = (\tau_0 + \chi)\mathbf{Y}_2\boldsymbol{\rho}(0) + (\tau_0 + \chi)\mathbf{Y}_3\boldsymbol{\rho}(-1), \tag{4.19}$$

$$\mathbf{F}(\chi, \boldsymbol{\rho}) = (\tau_0 + \chi)(0, \hat{F}_1, \hat{F}_2)^\top, \tag{4.20}$$

where $\boldsymbol{\rho} = [\rho_1, \rho_2, \rho_3]^\top \in C$, and $\mathbf{Y}_2$ and $\mathbf{Y}_3$ are given in Equations (4.11). The terms $\hat{F}_1$ and $\hat{F}_2$ in Equation (4.20) are

$$\hat{F}_1 = b_5\rho_2^2(0) - b_6\rho_2^3(0) + \ldots,$$
$$\hat{F}_2 = -b_5\rho_2^2(0) + b_6\rho_2^3(0) + \ldots,$$

where

$$b_5 = \frac{\beta\kappa}{(1 + \beta D_c^*)^3},\ b_6 = \frac{\beta^2\kappa}{(1 + \beta D_c^*)^4}.$$

Accordingly, for $\Theta \in [-1, 0]$, there exists a function $\boldsymbol{\eta}(\Theta, \chi)$ such that:

$$\mathbf{L}_\chi(\boldsymbol{\rho}) = \int_{\Theta=-1}^{0} d\boldsymbol{\eta}(\Theta, \chi)\boldsymbol{\rho}(\Theta).$$

In view of Equation (4.19), we can choose:

$$\boldsymbol{\eta}(\Theta, \chi) = (\tau_0 + \chi)\mathbf{J}_0\delta(\Theta) - (\tau_0 + \chi)\mathbf{J}_\tau\delta(\Theta + 1),$$

where $\delta(\Theta)$ is the Dirac delta function:

$$\delta(\Theta) = \begin{cases} 0, & \Theta \neq 0, \\ 1, & \Theta = 0. \end{cases}$$

For $\boldsymbol{\varphi} = [\varphi_1, \varphi_2, \varphi_3] \in C^1([-1, 0], \mathbb{C}^3)$, we define:

$$A(\chi)\boldsymbol{\varphi}(\Theta) = \begin{cases} \dfrac{d\boldsymbol{\varphi}(\Theta)}{d\Theta}, & \Theta \in [-1, 0), \\ \int_{\varsigma=-1}^{0} d\boldsymbol{\eta}(\varsigma, \chi)\boldsymbol{\varphi}(\varsigma), & \Theta = 0, \end{cases}$$

and

$$R(\chi)\boldsymbol{\varphi}(\Theta) = \begin{cases} 0, & \Theta \in [-1, 0), \\ \mathbf{F}(\chi, \boldsymbol{\varphi}), & \Theta = 0. \end{cases}$$

Then, for $\Theta \in [-1, 0]$, we can transform Equation (4.18) into

$$\frac{d\mathbf{x}_t(\Theta)}{dt} = A(\chi)\mathbf{x}_t(\Theta) + R(\chi)\mathbf{x}_t(\Theta). \tag{4.21}$$

For $\boldsymbol{\psi} = [\psi_1, \psi_2, \psi_3]^\top \in C^1([0, 1], \mathbb{C}^3)$, we define the adjoint operator $A^*(0)$ of $A(0)$ as follows:

$$A^*(0)\boldsymbol{\psi}(s) = \begin{cases} -\dfrac{d\boldsymbol{\psi}(s)}{ds}, & s \in (0, 1], \\ \int_{\varsigma=-1}^{0} d\boldsymbol{\eta}^\top(\varsigma, 0)\boldsymbol{\psi}(-\varsigma), & s = 0, \end{cases}$$

and a bilinear form as follows:

$$\langle \boldsymbol{\psi}(s), \boldsymbol{\varphi}(\Theta) \rangle = \overline{\boldsymbol{\psi}}^\top(0)\boldsymbol{\varphi}(0) - \int_{\Theta=-1}^{0} \int_{s=0}^{\Theta} \overline{\boldsymbol{\psi}}^\top(s - \Theta)\, d\boldsymbol{\eta}(\Theta, 0)\, \boldsymbol{\varphi}(s)\, ds. \tag{4.22}$$

From Section 4.2, we know that $\pm i\omega_0\tau_0$ are eigenvalues of $A(0)$ and $A^*(0)$.

Let $\mathbf{q}(\Theta) = [1, a_1, a_2]^\top e^{i\omega_0\tau_0\Theta}$ be the eigenvector of $A(0)$ corresponding to $i\omega_0\tau_0$, and $\mathbf{q}^*(s) = \frac{1}{\overline{G}}[1, a_1^*, a_2^*]^\top e^{i\omega_0\tau_0 s}$ be the eigenvector of $A^*(0)$ corresponding to $-i\omega_0\tau_0$. By direct computation, we obtain:

$$\begin{aligned} a_1 &= \frac{(b_1 + i\omega_0)(b_4 + i\omega_0)}{\sigma b_3 - (b_2 + i\omega_0)(b_4 + i\omega_0)}, \\ a_2 &= \frac{b_3(b_1 + i\omega_0)}{\sigma b_3 - (b_2 + i\omega_0)(b_4 + i\omega_0)}, \\ a_1^* &= \frac{\gamma + (b_1 + i\omega_0)e^{-i\omega_0\tau_0}}{\gamma}, \\ a_2^* &= \frac{\sigma\gamma + \sigma(b_1 - i\omega_0)e^{-i\omega_0\tau_0}}{\gamma b_4 - i\gamma\omega_0}. \end{aligned}$$

From Equation (4.22), we obtain:

$$\overline{G} = 1 + a_1\overline{a_1^*} + a_2\overline{a_2^*} + \gamma\tau_0 e^{-i\omega_0\tau_0}(\overline{a_1^*} - 1),$$

such that $\langle \mathbf{q}^*(s), \mathbf{q}(\Theta) \rangle = 1$ and $\langle \mathbf{q}^*(s), \overline{\mathbf{q}}(\Theta) \rangle = 0$.

Following the algorithm given in [18] and using the similar computation process in [19], we obtain:

$$g_{20} = \frac{2\tau_0 b_5 a_1^2}{\overline{G}}\left(\overline{a_1^*} - \overline{a_2^*}\right),$$

$$g_{11} = \frac{2\tau_0 b_5 a_1 \overline{a_1}}{\overline{G}}\left(\overline{a_1^*} - \overline{a_2^*}\right),$$
$$g_{02} = \frac{2\tau_0 b_5 \overline{a_1}^2}{\overline{G}}\left(\overline{a_1^*} - \overline{a_2^*}\right),$$
$$g_{21} = \frac{2\tau_0(\overline{a_1^*} - \overline{a_2^*})}{\overline{G}}\left(b_5\overline{a_1}W_{20}^{(2)}(0) + 2b_5 a_1 W_{11}^{(2)}(0) - 3b_6 a_1^2\overline{a_1}\right),$$

with

$$\mathbf{W}_{20}(\Theta) = \frac{ig_{20}}{\omega_0\tau_0}\mathbf{q}(\Theta) + \frac{i\overline{g}_{02}}{3\omega_0\tau_0}\overline{\mathbf{q}}(\Theta) + \mathbf{F}_1 e^{2i\omega_0\tau_0\Theta},$$
$$\mathbf{W}_{11}(\Theta) = -\frac{ig_{11}}{\omega_0\tau_0}\mathbf{q}(\Theta) + \frac{i\overline{g}_{11}}{\omega_0\tau_0}\overline{\mathbf{q}}(\Theta) + \mathbf{F}_2,$$

where

$$\mathbf{W}_{20}(\Theta) = [W_{20}^{(1)}(\Theta), W_{20}^{(2)}(\Theta), W_{20}^{(3)}(\Theta)]^\top,$$
$$\mathbf{W}_{11}(\Theta) = [W_{11}^{(1)}(\Theta), W_1^{(2)}(\Theta), W_{11}^{(3)}(\Theta)]^\top,$$
$$\mathbf{F}_1 = [F_1^{(1)}, F_1^{(2)}, F_1^{(3)}]^\top,$$
$$\mathbf{F}_2 = [F_2^{(1)}, F_2^{(2)}, F_2^{(3)}]^\top.$$

The terms $\mathbf{F}_1$ and $\mathbf{F}_2$ satisfy:

$$\begin{bmatrix} 2i\omega_0 + b_1 + \gamma e^{-2i\omega_0\tau_0} & 0 & 0 \\ -\gamma e^{-2i\omega_0\tau_0} & 2i\omega_0 + b_2 & -\sigma \\ 0 & -b_3 & 2i\omega_0 + b_4 \end{bmatrix} \begin{bmatrix} F_1^{(1)} \\ F_1^{(2)} \\ F_1^{(3)} \end{bmatrix} = \begin{bmatrix} 0 \\ 2b_5 a_1^2 \\ -2b_5 a_1^2 \end{bmatrix},$$

$$\begin{bmatrix} b_1 + \gamma & 0 & 0 \\ -\gamma & b_2 & -\sigma \\ 0 & -b_3 & b_4 \end{bmatrix} \begin{bmatrix} F_2^{(1)} \\ F_2^{(2)} \\ F_2^{(3)} \end{bmatrix} = \begin{bmatrix} 0 \\ 2b_5 a_1\overline{a_1} \\ -2b_5 a_1\overline{a_1} \end{bmatrix}.$$

Then, the following equations can be computed to obtain the properties of Hopf bifurcation:

$$c_1(0) = \frac{i}{2\omega_0\tau_0}\left(g_{11}g_{20} - 2|g_{11}|^2 - \frac{|g_{02}|^2}{3}\right) + \frac{g_{21}}{2},$$
$$\chi_2 = -\frac{\Re\{c_1(0)\}}{\Re\{\lambda'(\tau_0)\}}, \tag{4.23}$$
$$\beta_2 = 2\Re\{c_1(0)\}, \tag{4.24}$$
$$T_2 = -\frac{\Im\{c_1(0)\} + \chi_2\Im\{\lambda'(\tau_0)\}}{\omega_0\tau_0}. \tag{4.25}$$

□

## 5 Sensitivity analysis

Model (2.2) has three model outputs (three state variables) and thirteen model inputs (ten parameters and three initial conditions). The model outputs are denoted as $\mathbf{x} = \{D, D_c, D_p\}$, while the model inputs are denoted as $\varsigma = \{I, \gamma, \kappa, \beta, \sigma, \mu, \mu_1, \mu_2, \mu_3, \tau, \phi(\varrho), D_{c0}, D_{p0}\}$. Using the direct differential method [22], the sensitivity index of an arbitrary model output $x_i$ with respect to an input $\varsigma_j$ is

$$S_{\varsigma_j}^{x_i}(t) = \frac{\partial x_i(t)}{\partial \varsigma_j}, \quad i = 1, 2, 3, \quad j = 1, 2, \ldots, 13.$$

For simplifying the notations, the variable $S_{\varsigma_j}^{x_i}(t)$ is denoted as $S_{\varsigma_j}^{x_i}$, unless the argument is other than $t$. According to [22], the system of differential equations for the sensitivity indices of the model outputs $D$, $D_c$, and $D_p$ with respect to an arbitrary model input $\varsigma_j$ is

$$\frac{dS_{\varsigma_j}^{D}}{dt} = -(\mu+\mu_1)S_{\varsigma_j}^{D} - \gamma S_{\varsigma_j}^{D}(t-\tau) + \frac{\partial}{\partial \varsigma_j}\left(\frac{dD}{dt}\right), \tag{5.1a}$$

$$\frac{dS_{\varsigma_j}^{D_c}}{dt} = -\frac{\kappa S_{\varsigma_j}^{D_c}}{(1+\beta D_c)^2} - (\mu+\mu_2)S_{\varsigma_j}^{D_c} + \sigma S_{\varsigma_j}^{D_p} + \gamma S_{\varsigma_j}^{D}(t-\tau) + \frac{\partial}{\partial \varsigma_j}\left(\frac{dD_c}{dt}\right), \tag{5.1b}$$

$$\frac{dS_{\varsigma_j}^{D_p}}{dt} = \frac{\kappa S_{\varsigma_j}^{D_c}}{(1+\beta D_c)^2} - (\sigma+\mu+\mu_3)S_{\varsigma_j}^{D_p} + \frac{\partial}{\partial \varsigma_j}\left(\frac{dD_p}{dt}\right), \tag{5.1c}$$

with the initial conditions:

$$S_{\varsigma_j}^{D}(\varrho) = \frac{\partial \phi(\varrho)}{\partial \varsigma_j},\ \varrho \in [-\tau, 0],\ S_{\varsigma_j}^{D_c}(0) = \frac{\partial D_{c0}}{\partial \varsigma_j},\ S_{\varsigma_j}^{D_p}(0) = \frac{\partial D_{p0}}{\partial \varsigma_j}.$$

In particular, the system of differential equations for the sensitivity indices of the model outputs $D$, $D_c$, and $D_p$ with respect to the diabetes incidence rate $(I)$ is

$$\frac{dS_I^{D}}{dt} = -(\mu+\mu_1)S_I^{D} - \gamma S_I^{D}(t-\tau) + 1, \tag{5.2a}$$

$$\frac{dS_I^{D_c}}{dt} = -\frac{\kappa S_I^{D_c}}{\left(1+\beta D_c\right)^2} - (\mu+\mu_2)S_I^{D_c} + \sigma S_I^{D_p} + \gamma S_I^{D}(t-\tau), \tag{5.2b}$$

$$\frac{dS_I^{D_p}}{dt} = \frac{\kappa S_I^{D_c}}{\left(1+\beta D_c\right)^2} - (\sigma+\mu+\mu_3)S_I^{D_p}, \tag{5.2c}$$

with the initial conditions: $S_I^{D}(\varrho) = 0$, $\varrho \in [-\tau, 0]$, $S_I^{D_c}(0) = 0$, and $S_I^{D_p}(0) = 0$. The equilibrium point of model (5.2) is

$$S_I^{D^*} = \frac{1}{\gamma+\mu+\mu_1},$$

$$S_I^{D_c^*} = \frac{\gamma(\sigma+\mu+\mu_3)(1+\beta D_c^*)^2 S_I^{D^*}}{\kappa(\mu+\mu_3)+(\mu+\mu_2)(\sigma+\mu+\mu_3)(1+\beta D_c^*)^2},$$

$$S_I^{D_p^*} = \frac{\kappa S_I^{D_c^*}}{(\sigma+\mu+\mu_3)(1+\beta D_c^*)^2}.$$

Next, the system of differential equations for the sensitivity indices of the model outputs $D$, $D_c$, and $D_p$ with respect to the time delay in developing the first complication $(\tau)$ is

$$\frac{dS_\tau^{D}}{dt} = -(\mu+\mu_1)S_\tau^{D} - \gamma S_\tau^{D}(t-\tau) + \gamma \frac{dD(t-\tau)}{dt}, \tag{5.3a}$$

$$\frac{dS_\tau^{D_c}}{dt} = -\frac{\kappa S_\tau^{D_c}}{\left(1+\beta D_c\right)^2} - (\mu+\mu_2)S_\tau^{D_c}(t) + \sigma S_\tau^{D_p} + \gamma S_\tau^{D}(t-\tau) - \gamma \frac{dD(t-\tau)}{dt}, \tag{5.3b}$$

$$\frac{dS_\tau^{D_p}}{dt} = \frac{\kappa S_\tau^{D_c}}{\left(1+\beta D_c\right)^2} - (\sigma+\mu+\mu_3)S_\tau^{D_p}, \tag{5.3c}$$

with the initial conditions: $S_\tau^{D}(\varrho) = 0$, $\varrho \in [-\tau, 0]$, $S_\tau^{D_c}(0) = 0$, and $S_\tau^{D_p}(0) = 0$. The equilibrium of model (5.3) is $S_\tau^{D^*} = 0$, $S_\tau^{D_c^*} = 0$, and $S_\tau^{D_p^*} = 0$.

Then, we compute the normalized forward sensitivity indices as follows:

$$\Upsilon_{\varsigma_j}^{x_i} = S_{\varsigma_j}^{x_i}\frac{\varsigma_j}{x_i}, \quad i = 1, 2, 3, \quad j = 1, 2, \ldots, 13. \tag{5.4}$$

The term $\Upsilon_{\varsigma_j}^{x_i}$ can be interpreted as the approximate percentage change in the model output $x_i$ when we increase the model input $\varsigma_j$ by 1%.

### 5.1 Sensitivity index of $E^*$

Using the values in Table 2 (see Appendix A for the details), the normalized forward sensitivity indices of $E^* = (D^*, D_c^*, D_p^*)$ with respect to every model input are computed and presented in Table 3.

**Table 2.** List of values for the model inputs of model (2.2).

| Model input | Value | Dimension | Source |
|---|---|---|---|
| $\phi(\varrho)$ | 2558998 | individuals | Assumed after [2] |
| $D_{c0}$ | 666483 | individuals | Assumed after [2] |
| $D_{p0}$ | 666483 | individuals | Assumed after [2] |
| $I$ | 90540.25 | individuals year$^{-1}$ | Estimated after [2] |
| $\gamma$ | 0.1 | year$^{-1}$ | Assumed |
| $\sigma$ | 0.15 | year$^{-1}$ | Assumed |
| $\mu$ | 0.008678 | year$^{-1}$ | [20, 21] |
| $\mu_1$ | $5.84\times 10^{-4}$ | year$^{-1}$ | Assumed after [20, 21] |
| $\mu_2$ | 0.002336 | year$^{-1}$ | Assumed after [20, 21] |
| $\mu_3$ | 0.001752 | year$^{-1}$ | Assumed after [20, 21] |
| $\kappa$ | 0.988986 | year$^{-1}$ | Assumed after [20, 21] |
| $\beta$ | $5\times 10^{-6}$ | individuals$^{-1}$ | Assumed |
| $\tau$ | 5 | years | [23] |

**Table 3.** Normalized forward sensitivity indices of $E^*$ using the values of model inputs in Table 2.

| $\varsigma_j$ | $\Upsilon_{\varsigma_j}^{D^*}$ | $\Upsilon_{\varsigma_j}^{D_c^*}$ | $\Upsilon_{\varsigma_j}^{D_p^*}$ |
|---|---|---|---|
| $I$ | 1 | 1.1708 | 0.0355 |
| $\gamma$ | –0.9152 | 0.0993 | 0.0030 |
| $\kappa$ | 0 | –0.1762 | 0.9947 |
| $\beta$ | 0 | 0.1708 | –0.9645 |
| $\sigma$ | 0 | 0.1647 | –0.9300 |
| $\mu$ | –0.0794 | –1.0137 | –0.0849 |
| $\mu_1$ | –0.0053 | –0.0063 | $-1.899\times 10^{-4}$ |
| $\mu_2$ | 0 | –0.2110 | –0.0064 |
| $\mu_3$ | 0 | –0.0277 | –0.0118 |
| $\tau$ | 0 | 0 | 0 |
| $\phi(\varrho)$ | 0 | 0 | 0 |
| $D_{c0}$ | 0 | 0 | 0 |
| $D_{p0}$ | 0 | 0 | 0 |

From Table 3, the model inputs $\tau$, $\phi(\varrho)$, $D_{c0}$, and $D_{p0}$ do not affect $E^*$. While increasing $\mu$ will reduce the size of all subpopulations, it is not proposed as a practical or ethical intervention, but is included to illustrate the sensitivity of the model dynamics. The same argument applies to the other death-related model inputs ($\mu_1$, $\mu_2$, and $\mu_3$). Apart from the model inputs $\tau$, $\phi(\varrho)$, $D_{c0}$, $D_{p0}$, $\mu$, $\mu_1$, $\mu_2$, and $\mu_3$, we rank the normalized forward sensitivity indices of $D^*$, $D_c^*$, and $D_p^*$ with respect to the other model inputs from the most sensitive to the least (see Table 4).

**Table 4.** Magnitude and sign of the normalized forward sensitivity indices of $D^*$, $D_c^*$, and $D_p^*$ with respect to the model inputs $I$, $\gamma$, $\kappa$, $\beta$, and $\sigma$.

| (A) indices of $D^*$ | | |
|---|---|---|
| $\varsigma_j$ | $\lvert\Upsilon_{\varsigma_j}^{D^*}\rvert$ | sign |
| $I$ | 1.0000 | + |
| $\gamma$ | 0.9152 | − |
| $\kappa$ | 0 | n/a |
| $\beta$ | | |
| $\sigma$ | | |

| (B) indices of $D_c^*$ | | |
|---|---|---|
| $\varsigma_j$ | $\lvert\Upsilon_{\varsigma_j}^{D_c^*}\rvert$ | sign |
| $I$ | 1.1708 | + |
| $\kappa$ | 0.1762 | − |
| $\beta$ | 0.1708 | + |
| $\sigma$ | 0.1647 | |
| $\gamma$ | 0.0993 | |

| (C) indices of $D_p^*$ | | |
|---|---|---|
| $\varsigma_j$ | $\lvert\Upsilon_{\varsigma_j}^{D_p^*}\rvert$ | sign |
| $\kappa$ | 0.9947 | + |
| $\beta$ | 0.9645 | − |
| $\sigma$ | 0.9300 | |
| $I$ | 0.0355 | + |
| $\gamma$ | 0.0030 | |

From Table 4, a 1% increase $(I)$ causes a significant increase in $(D^*)$ and $(D_c^*)$ with a magnitude greater than or equal to one. Meanwhile, $(D_p^*)$ increases by approximately 0.0355%. Therefore, decreasing the incidence rate of diabetes would be essential to reducing diabetes cases.

Furthermore $D^*$ decreases by approximately 0.9152% if $(\gamma)$ increases by 1%. However, a 1% increase in $\gamma$ causes $D_c^*$ and $D_p^*$ to increase by approximately 0.0993% and 0.0030%, respectively. Even though $D^*$ decreases, the interpretation of increasing $\gamma$ is unsatisfactory because we aim to reduce diabetes complications. We can reduce $D_c^*$ by decreasing the recurrence rate of complications $(\sigma)$ because a 1% increase in $\sigma$ causes $D_c^*$ to increase by approximately 0.1646%. Hence, the optimal way to reduce diabetes complications is to control the development of diabetes-related complications among all people with diabetes by decreasing both rates of first and recurring incidence of complications.

In Table 4(B), $(\kappa)$ is the second choice after $(I)$ to reduce $(D_c^*)$. However, $(\kappa)$ is at its maximal value because if $\beta = 0$ individuals$^{-1}$, $\kappa + \mu + \mu_2 = 1$ year$^{-1}$. Due to the saturating recovery rate of complications in model (2.2), $\kappa$ decreases by the inhibition effect term $\dfrac{1}{1+\beta D_c}$. In reality, $\dfrac{1}{1+\beta D_c}$ can be described as the reverse effect of diabetics with complications being postponed for treatment [14]. Reducing $\beta$ will maintain $(\kappa)$ at its maximal value. From Table 4(B), a 1% increase in $\beta$ causes an approximately 0.1708% increase in $(D_c^*)$. Thus, the decrease in $\beta$ is crucial to increase the number of diabetics who receive treatment for complications and, as a result, reduce $(D_c^*)$.

## 6 Numerical simulation

### 6.1 Stability of $E^*$

We use MATLAB to solve the equations numerically. Three examples are presented, and Table 5 shows the differences between them.

**Table 5.** Numerical examples with respect to model (2.2).

| Example | Theorem 3.2 | Theorem 4.2 | |
|---|---|---|---|
| | | (H2) | (H3) |
| 1 | • | | |
| 2 | | • | |
| 3 | | | • |

**Example 1**

Consider the model inputs: $I = 50$, $\gamma = 0.01$, $\kappa = 0.8$, $\beta = 0.0005$, $\sigma = 0.4$, $\mu = 0.01$, $\mu_1 = 0.05$, $\mu_2 = 0.15$, $\mu_3 = 0.1$, $\tau = 0$, $D_0 = 800$, $D_{c0} = 100$, and $D_{p0} = 100$, we have

$$\frac{dD}{dt} = 50 - 0.01D - 0.06D, \tag{6.1a}$$

$$\frac{dD_c}{dt} = 0.01D - \frac{0.8D_c}{1 + 0.0005D_c} + 0.4D_p - 0.16D_c, \tag{6.1b}$$

$$\frac{dD_p}{dt} = \frac{0.8D_c}{1 + 0.0005D_c} - 0.51D_p, \tag{6.1c}$$

with the initial conditions:

$$D(0) = 800,\ D_c(0) = 100,\ D_p(0) = 100.$$

Therefore $E^* = (714.2857, 21.5988, 33.5186)$. By Theorem 3.2, $E^*$ is locally asymptotically stable (see Figure 4).

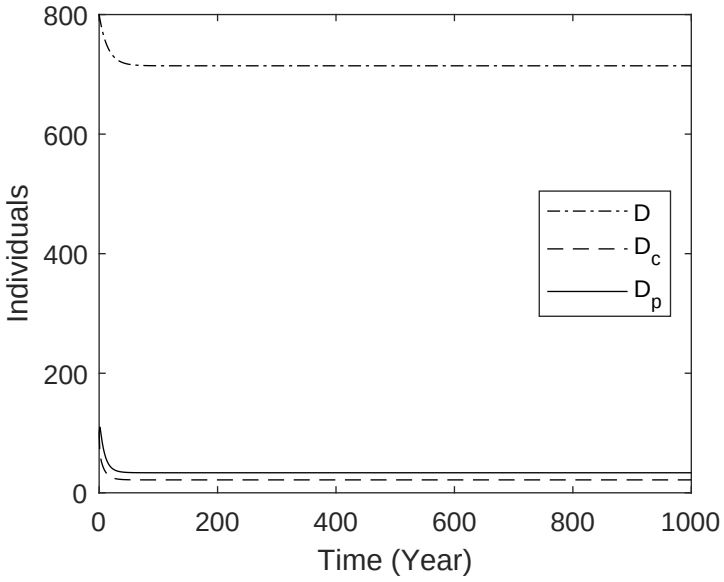



**Figure 4.** The dynamics of model (6.1).

**Example 2**

Consider the model inputs: $I = 50$, $\gamma = 0.01$, $\kappa = 0.8$, $\beta = 0.0005$, $\sigma = 0.4$, $\mu = 0.01$, $\mu_1 = 0.05$, $\mu_2 = 0.15$, $\mu_3 = 0.1$, $\phi(\varrho) = 800$, $D_{c0} = 100$, and $D_{p0} = 100$, we have

$$\frac{dD}{dt} = 50 - 0.01D(t - \tau) - 0.06D, \tag{6.2a}$$

$$\frac{dD_c}{dt} = 0.01D(t - \tau) - \frac{0.8D_c}{1 + 0.0005D_c} + 0.4D_p - 0.16D_c, \tag{6.2b}$$

$$\frac{dD_p}{dt} = \frac{0.8D_c}{1 + 0.0005D_c} - 0.51D_p, \tag{6.2c}$$

with the initial conditions:

$$D(\varrho) = 800,\ \varrho \in [-\tau, 0],\ D_c(0) = 100,\ D_p(0) = 100.$$

Therefore $E^* = (714.2857, 21.5988, 33.5186)$.

Following Theorem 4.2, we have $y_1 - \gamma = 0.05 > 0$. This shows that condition (H2) holds. From Theorem 4.2(i), $E^*$ is locally asymptotically stable for $\tau \geq 0$. Figure 5 displays the dynamics of model (6.2) with two different $\tau$ values: $\tau = 5$ and $\tau = 50$. The solutions $D$, $D_c$, and $D_p$ of model (6.2) converge to $E^*$ regardless of the value of $\tau$.

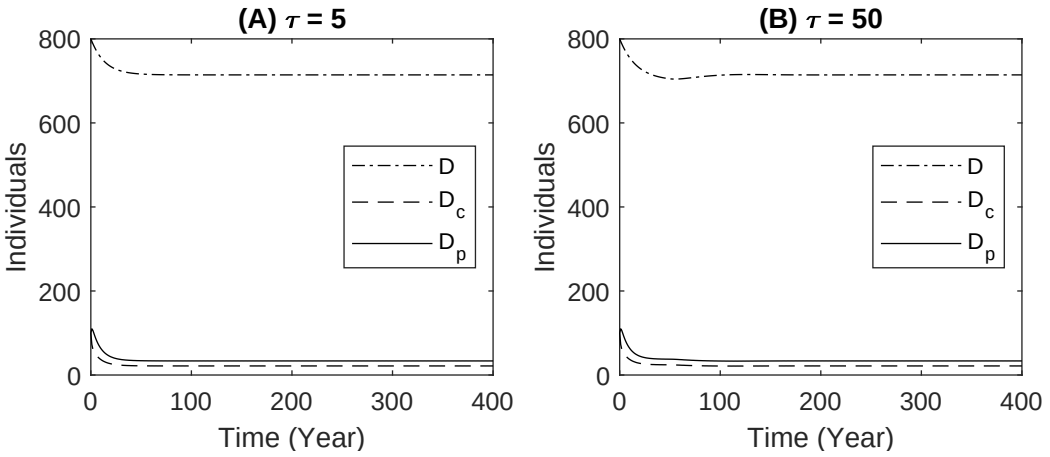



**Figure 5.** (A) The dynamics of model (3.20) with $\tau = 5$. (B) The dynamics of model (3.20) with $\tau = 50$.

**Example 3**

Consider the model inputs: $I = 50$, $\gamma = 0.15$, $\kappa = 0.8$, $\beta = 0.0005$, $\sigma = 0.4$, $\mu = 0.01$, $\mu_1 = 0.05$, $\mu_2 = 0.15$, $\mu_3 = 0.1$, $\phi(\varrho) = 100$, $D_{c0} = 30$, and $D_{p0} = 10$, we have

$$\frac{dD}{dt} = 50 - 0.15D(t-\tau) - 0.06D, \tag{6.3a}$$

$$\frac{dD_c}{dt} = 0.15D(t-\tau) - \frac{0.8D_c}{1+0.0005D_c} + 0.4D_p - 0.16D_c \tag{6.3b}$$

$$\frac{dD_p}{dt} = \frac{0.8D_c}{1+0.0005D_c} - 0.51D_p, \tag{6.3c}$$

with the initial conditions:

$$D(\varrho) = 100,\ \varrho \in [-\tau, 0],\ D_c(0) = 30,\ D_p(0) = 10.$$

Model (6.3) has a positive equilibrium point $E^* = (238.0952, 110.3917, 164.1055)$.

Following Theorem 4.2, we have $y_1 - \gamma = -0.09 < 0$, and condition (H3) is satisfied. Hence, we have the critical value $\tau_0 = 14.4192$. From Theorem 4.2(ii), $E^*$ is locally asymptotically stable when $0 \leq \tau < 14.4192$, where the solutions $D$, $D_c$, and $D_p$ of model (6.3) converge to $E^*$ (see Figure 6(A)). $E^*$ becomes unstable when $\tau > 14.4192$, where the solutions $D$, $D_c$, and $D_p$ of model (6.3) get larger as time $t$ increases (see Figure 6(B)). System (6.3) undergoes a Hopf bifurcation when $\tau = 14.4192$; a periodic orbit arises (see Figure 6(C)).

In addition, we can get $\chi_2 = -6.1961 < 0$, $\beta_2 = 3.1615 > 0$, and $T_2 = -8.3377 < 0$. From Theorem 4.4, the Hopf bifurcation is subcritical, the periodic solutions which exist for $\tau < 14.4192$ are unstable, and the period of bifurcating periodic solutions decreases.

### 6.2 Simulation with respect to the model inputs in Table 2

**Stability of $E^*$**

Using the values in Table 2, $E^* = (8.2865 \times 10^5, 6.3915 \times 10^6, 1.1955 \times 10^6)$. Condition (H3) for the existence of a pair of purely imaginary roots of characteristic Equation (4.13) is satisfied.

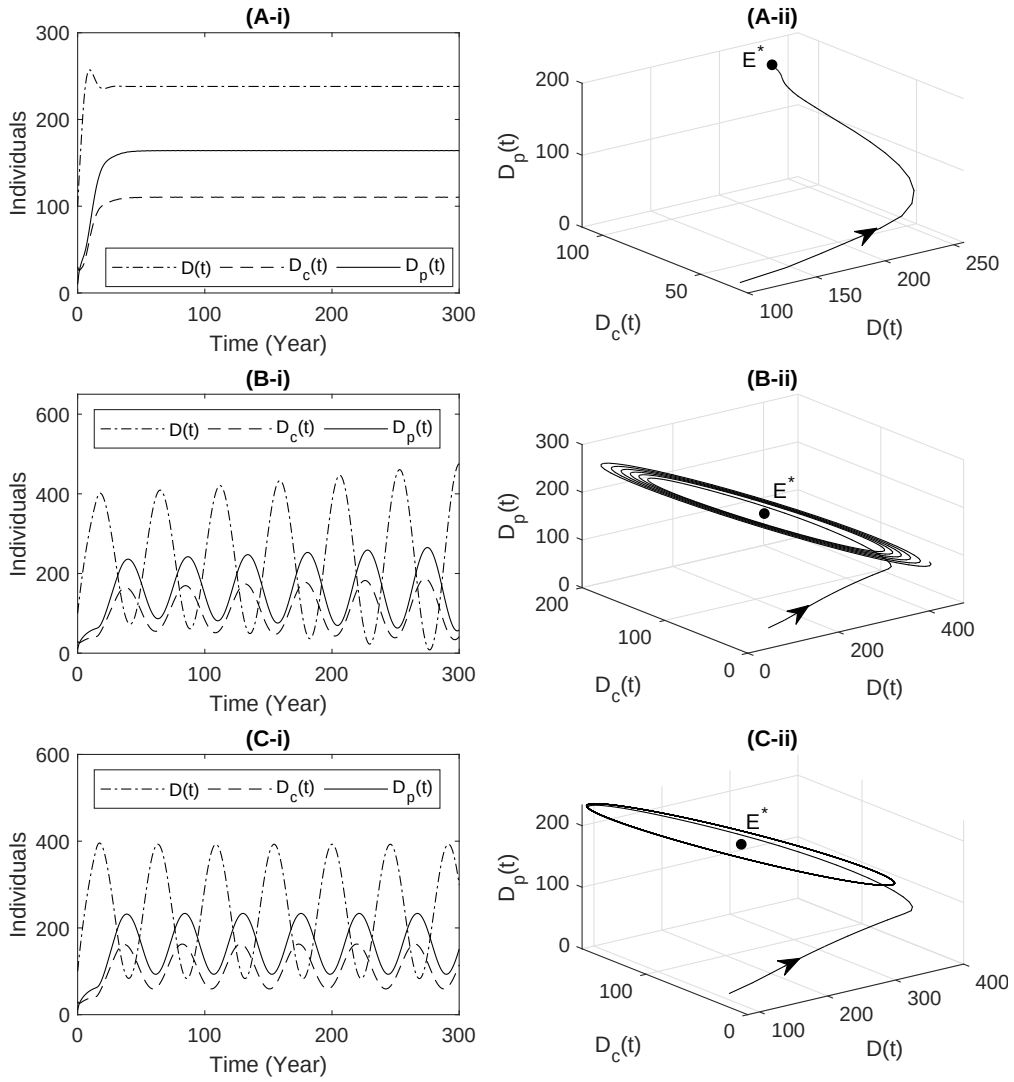



**Figure 6.** (A) $E^*$ is locally asymptotically stable for $\tau = 4 < \tau_0$. (B) $E^*$ is unstable for $\tau = 15.5 > \tau_0$. (C) System (6.3) undergoes a Hopf bifurcation when $\tau = \tau_0$.

We obtain the numerical value of $\tau_0$, which is 16.7073 years. From Theorem 4.2(ii), $E^*$ of model (2.2) is locally asymptotically stable for $\tau = 5$ years (see Figure 7). From Figure 7, it may be noted that the solutions $D$, $D_c$, and $D_p$ of model (2.2) approach $E^*$ as time $t$ increases, showing the stability of $E^*$ for $\tau = 5 < \tau_0$. At the current rates, if the trend continues and is left untreated, $(D_c)$ will approach its equilibrium value in the long-term simulation.

From the sensitivity analysis results (Section 5.1), reducing $(I)$ will reduce diabetes cases. Figure 8 shows that all diabetic subpopulations decrease and persist at lower levels by decreasing $I$. This can be achieved through education and awareness programs about the dangers of diabetes.

Second, we suggest decreasing $(\gamma)$ and $(\sigma)$ to control $(D_c)$. Figure 9 shows that by decreasing $\gamma$ and $\sigma$ simultaneously, $(D_c)$ decreases while $(D)$ and $(D_p)$ increases. To achieve a lower number of diabetics with complications, early detection of diabetes should continue, as well as improving the understanding of diabetes complications and self-care management among diabetics with recovered complications through education and family support.

Lastly, we suggest decreasing $(\beta)$ to decrease $(D_c)$. Figure 10 shows that by decreasing $\beta$, $(D_c)$ decreases while $(D_p)$ increases. Note that no changes can be observed for Figure 10(A) because the dynamics of $(D)$ are unaffected by $\beta$. Thus, we immediately recommend decreasing $\beta$, that is, to try to provide adequate medical resources for the treatment of complications.

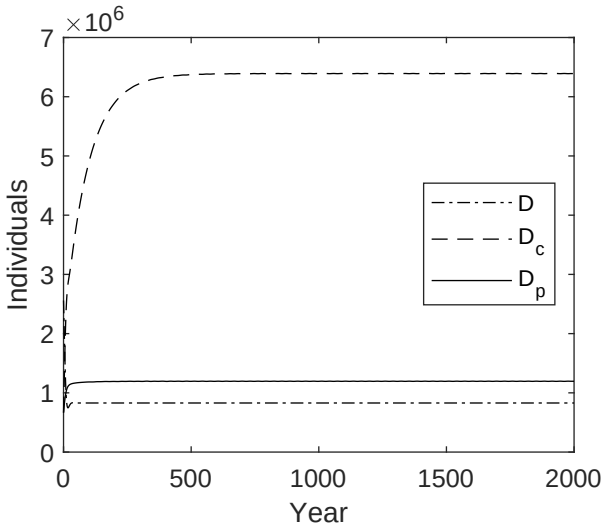



**Figure 7.** Dynamics of model (2.2) with the model inputs in Table 2.

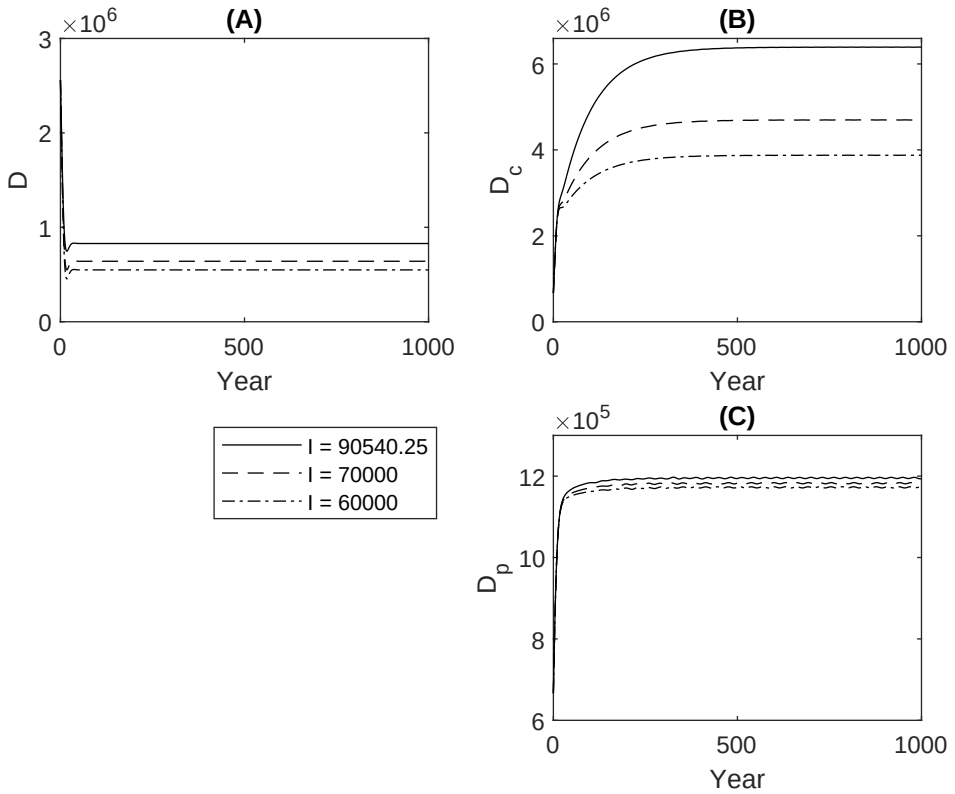



**Figure 8.** Simulation with decreasing value of $I$ ($I = 90540.25$, $I = 70000$, and $I = 60000$) with the other model inputs in Table 2.

### Influence of the first incidence of complications ($\gamma$) on Hopf bifurcation corresponds to the delay parameter $\tau$

From Theorem 4.2, the indicator for the occurrence of Hopf bifurcation is $\gamma > y_1$. Hence, we investigate the variation of the critical value of time delay $\tau_0$ (Equation (4.8)) with respect to the various rates of the first incidence of complications ($\gamma$). For the model inputs given in Table 2 and choosing $\gamma$ in the range of $0.01 \leq \gamma \leq 0.99$, we plot the stable and unstable regions in

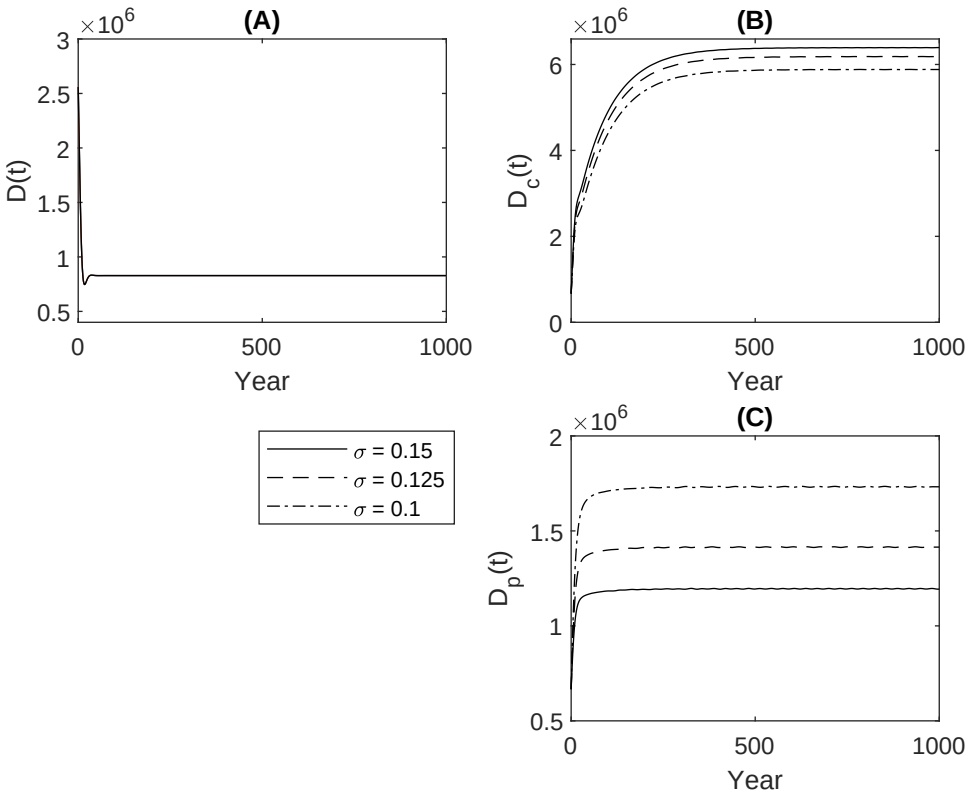



**Figure 9.** Simulation with decreasing value of $\gamma$ and $\sigma$ ($(\gamma, \sigma) = (0.1, 0.15)$, $(\gamma, \sigma) = (0.09, 0.14)$, and $(\gamma, \sigma) = (0.04, 0.10)$) with the other model inputs in Table 2.

Figure 11. This figure gives the minimum value of $\gamma$ so that $E^*$ remains locally asymptotically stable. This variation shows that the higher rate of the first incidence of complications $(\gamma)$ results in introducing a lower critical value $\tau_0$. The biological meaning is that if the rate $\gamma > 0.32$ year$^{-1}$, then the solutions $D$, $D_c$, and $D_p$ of model (2.2) will show unstable behaviors because $\tau = 5 > \tau_0$ (see Figure 11(B)). Sometimes the number of diabetics will be high and sometimes low. In this case, it will be difficult to predict the number of diabetic cases and assess the severity of diabetes. If we want to predict the size of all diabetic subpopulations, $(\gamma)$ should be no more than 0.32 year$^{-1}$ for the other model inputs in Table 2.

## 7 Conclusion

In this paper, we extended the work of [12] to incorporate a nonlinear term representing the limited resources for the recovery rate of diabetes complications. After the model formulation, we investigated the non-negativity and boundedness of the solutions of both delay and non-delay models. However, the non-negativity of the solutions $D$, $D_c$, and $D_p$ of the delay model (2.2) depends on condition (H1). In any event that the number of diabetics who never had any complications drops to zero, the number of diabetics who will develop the first complication should be less than the number of newly developed diabetics, that is if condition (H1) is met. Despite the possibility that the solutions may become negative, there exists a set of model inputs (parameters and initial conditions) guaranteeing the non-negativity.

In the stability analysis, we first investigated the local and global stability of $E^*$ of the corresponding non-delay model (3.1) in Section 3.2. It is found that $E^*$ is locally asymptotically stable without restriction on any positive parameter values. Subsequently, we attempted to construct a suitable Lyapunov function for $E^*$.

For the delay model (2.2), considering the time delay $\tau$ as the bifurcation parameter, we investigated the local stability and the existence of Hopf bifurcation of $E^*$. In Section 4.2, we

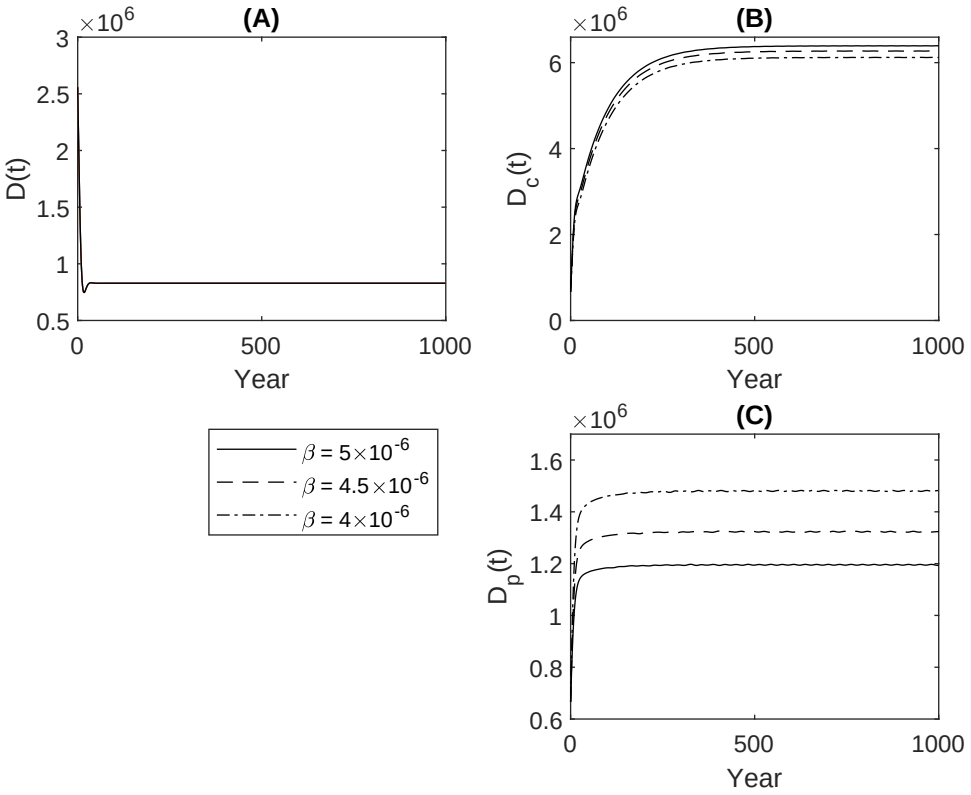



**Figure 10.** Simulation with decreasing value of $\beta$ ($\beta = 5 \times 10^{-6}$, $\beta = 4.5 \times 10^{-6}$, and $\beta = 4 \times 10^{-6}$) with the other model inputs in Table 2.

proved that if the rate of the first incidence of complications is no more than the total death rate of diabetics who never had any complications, that is, condition (H2) is met, then $E^*$ of model (2.2) is locally asymptotically stable for all $\tau \geq 0$. On the other hand, if the rate of the first incidence of complications is greater than the total death rate of diabetics who never had any complications, condition (H3) is met. Consequently, a Hopf bifurcation occurs at the critical value $\tau = \tau_0$, and the delay value affects the stability of $E^*$. If $\tau < \tau_0$, $E^*$ is locally asymptotically stable and $E^*$ becomes unstable for $\tau > \tau_0$. We validated these analytical results through numerical simulations.

In Section 5, we computed the normalized forward sensitivity indices of the model outputs with respect to the model inputs. Three main findings from sensitivity analysis are as follows:

(i) The decrease in $(I)$ led to a decrease in the diabetic equilibrium point $E^*$. We recommend that the government provide appropriate awareness programs about the dangers of diabetes at all levels of the population, starting from school.

(ii) The decrease $(\gamma)$ and $(\sigma)$ resulted in a decrease in $(D_c^*)$. We recommend that the government continue screening for diabetes and diabetes-related complications. Early detection and treatment of diabetes and diabetes-related complications can improve the outcomes and reduce the progression to more severe conditions.

(iii) The decrease in $(\beta)$ improved the recovery rate of diabetes complications. We recommend that the government provide adequate medical resources to treat complications, especially those involving organ transplantation. Awareness of the benefits of organ donation after death may help the organ shortage problem.

For the recommendation of future work, we would like to acknowledge that model (2.2) has several limitations that can be addressed. First, in the proposed model, the parameters are assumed to be constants. Second, the age structure within the diabetes population is also neglected in the proposed model. Furthermore, it is important to note that the present study is intended

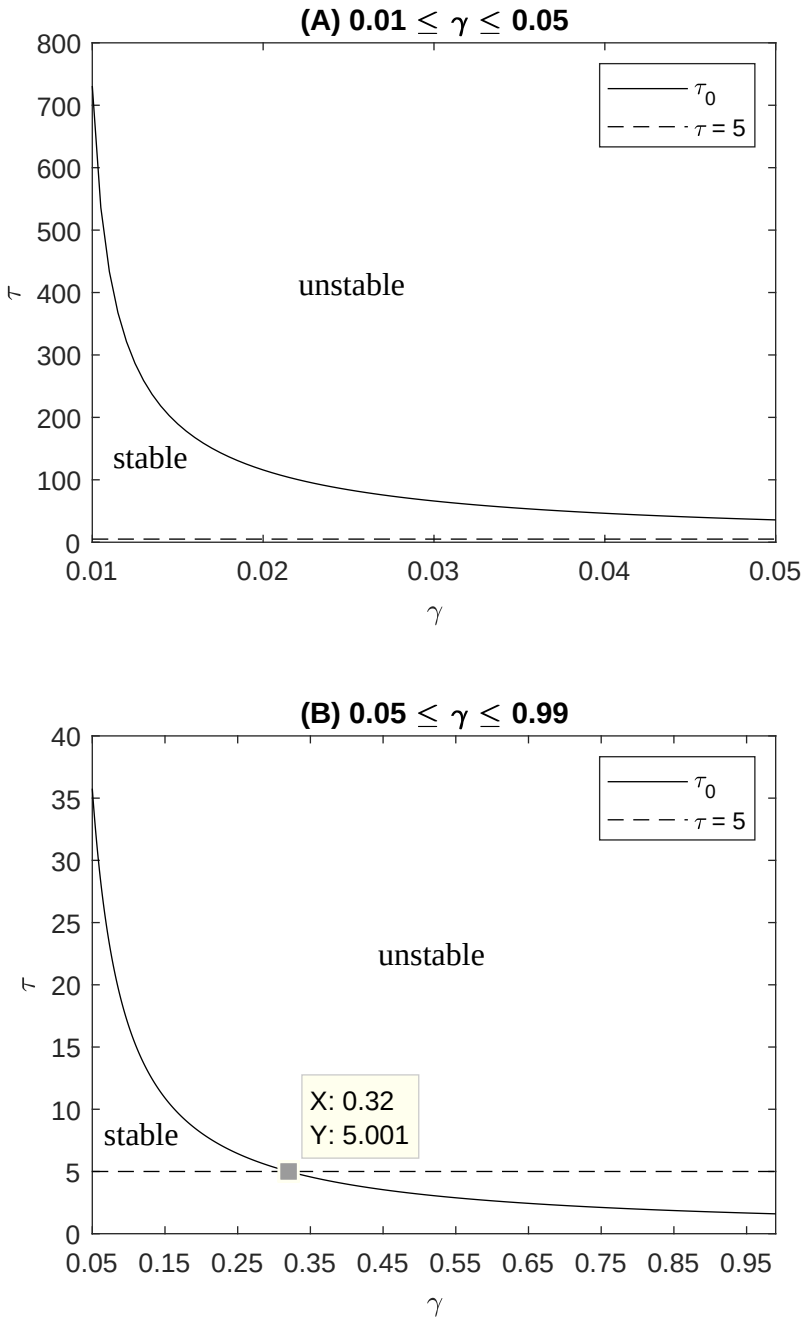



**Figure 11.** Graph of $\tau$ versus $\gamma$ showing the stable and unstable regions of model (2.2) with the other model inputs in Table 2 (the dashed line represents the line $\tau = 5$).

to focus on providing a theoretical and qualitative analysis for understanding diabetic population dynamics, rather than offering precise quantitative predictions. Consequently, the findings should be interpreted qualitatively, as they primarily capture essential behaviors of the system. A direct comparison with real epidemiological data (to validate the model predictions) was not feasible due to the limited availability of comprehensive and detailed time-series data on diabetes progression, complication onset, and recovery within the Malaysian population. However, we have parameterized the model using estimates derived from Malaysian national health surveys and demographic reports, and the values assigned to parameters such as incidence rate and mortality rates are grounded in published sources and reasonable assumptions, as detailed in Appendix A. Therefore, it is hoped that the analytical and numerical results can provide valuable

insights into the role of key parameters governing the system dynamics. Incorporating real epidemiological data for calibration and validation remains an important direction for future work.

## A Appendix A

First, we determine the values of the initial conditions of model (2.2), that is, the initial number of diabetics who never had any complications ($\phi(\varrho)$), the initial number of diabetics with complications ($D_{c0}$), and the initial number of diabetics with recovered complications ($D_{p0}$). Based on the 2019 Malaysian National Health and Morbidity Survey (NHMS) report [2], the estimated number of diabetics in 2019 was $3,891,965$. Among them, $1,892,515$ individuals did not know they had diabetes. We assume $1,892,515$ individuals in the category of diabetics who never had any complications because they did not know they had diabetes. Then, the estimated number of known diabetics was $3,891,965 - 1,892,515 = 1,999,450$ individuals. We assume that some portions of these known diabetics are in the category of diabetics who never had any complications, diabetics with complications, and diabetics with recovered complications. We assume each category represents one-third, that is, $666,483$ individuals of diabetics who never had any complications, $666,483$ individuals of diabetics with complications, and $666,483$ individuals of diabetics with recovered complications. We set the initial condition $\phi(\varrho)$ by combining $1,999,450$ and $666,483$. Therefore, we have $\phi(\varrho) = 2,558,998$ individuals, $D_{c0} = 666,483$ individuals, and $D_{p0} = 666,483$ individuals.

Next, we determine the value for the incidence rate of diabetes ($I$), the rate of the first incidence of complications ($\gamma$), and the recurrence rate of complications ($\sigma$). In the 2015 NHMS report, the estimated number of diabetics was $3,529,804$ individuals. In the interval of four years, the number increased by $362,161$ to a total of $3,891,965$ diabetics in 2019. By dividing $362,161$ by four, the yearly incidence rate of diabetes is given by $90,540.25$. We assume $I = 90,540.25$ individuals year$^{-1}$. For the rate of the first incidence of complications ($\gamma$) and the recurrence rate of complications ($\sigma$), due to unavailable information, we assume $\gamma = 0.1$ year$^{-1}$ and $\sigma = 0.15$ year$^{-1}$.

Next, we determine the value for the mortality-related parameters, that is, the death rate due to causes other than diabetes ($\mu$), diabetes-related death rate among diabetics who never had any complications ($\mu_1$), diabetes-related death rate among diabetics with complications ($\mu_2$), and diabetes-related death rate among diabetics with recovered complications ($\mu_3$). The life expectancy of Malaysian is about 74.9 years [20]. The average lifespan of 74.9 years corresponds to $\mu + \mu_1 + \mu_2 + \mu_3 = 1/74.9 \approx 0.013351$ year$^{-1}$. Note that diabetes accounted for 35% of total Malaysian mortality [21]. Therefore, we estimate the death rate due to causes other than diabetes as follows: $\mu = 0.65 \times 0.013351 \approx 0.008678$ year$^{-1}$, and the total diabetes-related death rate as follows: $\mu_1 + \mu_2 + \mu_3 = 0.35 \times 0.013351 \approx 0.004673$ year$^{-1}$. The specific mortality rate among the subpopulation of diabetics is unknown. Intuitively, we assume the diabetics who never had any complications ($D$) are still at the early stage of diabetes, while the diabetics with complications ($D_c$) and the diabetics with recovered complications ($D_p$) are not. We assume the values for the diabetes-related death rate as follows: $\mu_1 = 5.84 \times 10^{-4}$ year$^{-1}$, $\mu_2 = 0.002336$ year$^{-1}$, and $\mu_3 = 0.001752$ year$^{-1}$.

In model (2.2), we use the recovery rate of complications in the form of a saturating function $h(D_c) = \dfrac{\kappa D_c}{1 + \beta D_c}$. Note that $\dfrac{\kappa D_c}{1 + \beta D_c} \leq \kappa D_c$ with equality if and only if $\beta = 0$. The total rate of individuals leaving one particular compartment is at most 1 year$^{-1}$. Then, if the resources are unlimited (that is, $\beta = 0$), we need to satisfy $\kappa + \mu + \mu_2 = 1$ year$^{-1}$ for compartment $D_c$, which yields: $\kappa = 0.988986$ year$^{-1}$. For the non-negative parameter $\beta$ measuring the limited medical resources, due to unavailable information, we assume: $\beta = 5 \times 10^{-6}$ individuals$^{-1}$. The time delay $\tau$ for the first incidence of complications may vary for different types of complications. For the complications related to the (very) small blood vessels, the delay ranges from 6 to 13 years. In the case of retinopathy, it might appear five years after the onset of diabetes [23]. We assume $\tau = 5$ years.

The model inputs for model (2.2) are summarized in Table 2.

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