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Mathematical analysis and optimal control of a Caputo fractional diabetes system with parameter identification ☆

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ABSTRACT

In this study, mathematical modeling of diabetes disease is handled with the Caputo fractional derivative, and the resulting fractional differential equation system is examined in detail. The existence of the solution of this system was mathematically proven under appropriate conditions. Then, for the management and treatment of diabetes, three control parameters are added, and the number of people with diabetes is tried to be kept in the desired range with the optimal control theory. In addition to this, our model gains from parameter estimation and fitting with more accurately generated parameters. The numerical results of the fractional model and the effect of the control parameters are determined with the optimal control approach on glucose levels, examined with various simulations, and presented graphically.

1. Introduction

Diabetes mellitus, also known as diabetes, is a chronic disease in which the pancreas is unable to produce enough insulin or the body is unable to use insulin properly, resulting in high levels of sugar in the bloodstream. This affects the way the body converts food into energy, resulting in high levels of sugar in the blood.

The energy the body needs is provided by the basic nutrients protein, fats and carbohydrates. The most important part of these nutrients is glucose, a simple sugar. The blood carries glucose to all the cells of the body to use it for energy. Glucose is important because it is the body's most important source of energy, especially for the brain. Cells use the glucose they need with the help of the hormone insulin secreted by the pancreas. If the hormone insulin cannot be made in the body, glucose cannot be used as energy, and blood sugar will rise.

Diabetes is a disease that can be controlled with careful management and awareness, but can lead to serious health problems if left untreated. The biggest danger of diabetes is that it can cause long-term complications such as eye diseases, kidney diseases, heart diseases, nerve damage (neuropathy) and foot problems. Early detection, regular monitoring, healthy lifestyle habits and adherence to treatment are key to preventing complications of diabetes. This results in fewer hospitalizations and the need for emergency treatments, and prevention of complications also significantly reduces healthcare costs. Therefore, being aware of diabetes and consulting a healthcare professional if necessary is of great importance for both individual and societal health.

Among the many strategies and resources available to fight diseases, mathematical modeling is particularly noteworthy. Monitoring and combating diseases can be accomplished using mathematical methods, which is a powerful tool for predictive and interventional purposes. Mathematical models using data on disease incidence, environmental factors, and human health provide insights into the dynamics of disease spread and severity. This enables early detection and warning systems, allowing health

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authorities to adjust individuals' lifestyles, adopt the right treatment methods, and develop healthy eating and exercise habits. In the case of diabetes, these models are able to identify blood sugar anomalies early on and determine a person's risk of developing diabetes. Aye et al. [1] develop a mathematical model of diabetes mellitus and its consequences that includes treatment and a healthy lifestyle as a control. This study indicates that diabetes mellitus prevalence and incidence can be controlled with appropriate control measures, such as regular exercise, healthy eating, treatment, and a reduction in unhealthy lifestyle choices (such as smoking, drinking, and gluttony). This will lessen the danger of diabetes and prevent death coming from complications of diabetes. A model addressing diabetes is generated by Logaprakash and Monica [2] utilizing the effect of endocrine-disrupting chemicals (EDC). This model consider the daily consumption of food subjected to some detrimental chemicals causing health problems. Bassey [3] develops a series of linear mathematical type-II diabetes dynamic models using ordinary differential equations. The model is then converted into an optimal control problem, and an analysis was conducted using the traditional Pontryagin's maximum approach.

In mathematics, with the emergence of fractional calculus (FC), the concept of derivatives was extended beyond integer orders. This made it possible to simulate and understand complex events for which traditional integer-order derivatives could not produce meaningful solutions. The most important factor enabling this is the ability of fractional derivatives to capture the memory effects of the system, which determine its future behavior based on its past and current state [4–12]. It is observed that approaches based on the concept of fractional derivatives are increasingly being used in the modeling of diabetes, and they have been widely discussed in the literature. With regard to glucose–insulin regulation, mathematical models were developed using Caputo–Fabrizio approaches in [13] and fractal–fractional derivative approaches in [14]. Additionally, the role of beta cells in glucose–insulin interaction was examined using a fractal–fractional derivative model in study [15]. The co-occurrence of diabetes and tuberculosis was modeled using fractional-order derivatives in study [16], and the interaction between these two diseases was comprehensively addressed.

Optimal control theory provides an effective method for mathematically analyzing the behavior of infectious and chronic diseases and determining intervention strategies [17–20]. This technique aims to plan health policies and use limited resources in the most efficient way possible. Depending on the structure of the problem being addressed, optimal control problems have been designed in different structures, and solution methods have been developed accordingly. For this purpose, solution strategies for control problems with constraints have been developed and applied [21,22]. Additionally, there are approaches developed for solving optimal control problems when terminal cost terms are included in the objective function [23,24].

In this study, a mathematical model of diabetes was developed using Caputo fractional derivatives, and the resulting fractional differential equation system was analyzed analytically under appropriate conditions. Subsequently, three control parameters defined for the control and treatment of diabetes were integrated into the system with the aim of maintaining individuals' disease incidence levels within desired limits through optimal control theory. Additionally, parameter estimation was performed to make the model more consistent with real data.

The remainder of this paper is structured as follows. Section 1 provides an overview of the background and motivation. In Section 2, we introduce the necessary preliminaries on Caputo fractional derivatives. Section 3 formulates the fractional-order diabetes model, while Section 4 presents parameter estimation based on the number of diabetic patients in TÜrkiye between 2010 and 2022. Section 5 establishes the existence and uniqueness of its solutions, and Section 6 identifies the model's equilibrium points. In Section 7, we pose the fractional optimal control problem with three clinical interventions. Section 8 offers numerical results and a detailed discussion of glucose trajectories under optimal control, and finally, Section 9 concludes with key findings and avenues for future work.

2. Preliminaries

First, we provide a synopsis of FC and concentrate on the definition of the Caputo fractional derivative in this paper.

Definition 1 ([25,26])**.** The left and right Caputo fractional derivative with order $\kappa > 0$ are presented by

$$ {}_{a}^{C}D_{t}^{\kappa} f(t) = \frac{1}{\Gamma(m-\kappa)} \int_{a}^{t} \frac{f^{(m)}(\zeta)}{(t-\zeta)^{\kappa-m+1}} d\zeta, \ t > a $$

$$ {}_{t}^{C}D_{b}^{\kappa} f(t) = \frac{(-1)^{m}}{\Gamma(m-\kappa)} \int_{t}^{b} \frac{f^{(m)}(\zeta)}{(\zeta-t)^{\kappa-m+1}} d\zeta, \ t < b $$

where $m = [\kappa] + 1$ and f is an integrable function.

The corresponding left Riemann–Liouville fractional integral of order κ with $\mathrm{Re}(\kappa) > 0$ is presented by

$$ {}_{a}I_{t}^{\kappa} f(t) = \frac{1}{\Gamma(\kappa)} \int_{a}^{t} (t-\zeta)^{\kappa-1} f(\zeta)\, d\zeta, \ t > a. $$

3. Model formulation

The diabetes mellitus model [27] considers the disease and its complications in a population. The diabetes mellitus model [27] introduces a new mathematical system of diabetes and its complications, including healthy and treatment classes, building on the work based on [28–30]. The following structure defines what the integer order model takes into account:

$$\begin{aligned}
\frac{dH}{dt} &= \sigma u_1 S - (\mu + \tau) H, \\
\frac{dS}{dt} &= \Lambda + \tau H - \left(\mu + \alpha + \sigma u_1\right) S, \\
\frac{dD}{dt} &= \alpha S - \mu D - \lambda D + \omega T - u_2 D, \\
\frac{dC}{dt} &= \lambda D - \left(\mu + \delta + \gamma u_3\right) C, \\
\frac{dT}{dt} &= \gamma u_3 C - (\mu + \omega) T + u_2 D.
\end{aligned} \tag{1}$$

Below are the parameters and components of the explanations model:

$H(t)$: Healthy compartment,
$S(t)$: Susceptible compartment,
$D(t)$: Diabetic without complications compartment,
$C(t)$: Diabetic with complications compartment,
$T(t)$: Diabetic with complications that undergo treatment compartment,
Λ : Birth rate,
μ : Death by natural mortality,
τ : The rate at which healthy people are susceptible,
σ : The rate at which a susceptible person become healthy,
α : Diabetes incidence probability rate,
λ : The rate at which diabetic people develops complications,
γ : The rate of treatment for diabetics with complications,
ω : The rate at which a diabetic with complications returns to a diabetic without complications following treatment,
δ : Rate of death from complications,
$u_1(t)$: Represents public health campaigns and awareness training aimed at reducing the susceptibility of individuals to diabetes (e.g., education, lifestyle changes).
$u_2(t)$: Denotes prevention efforts implemented to avert the onset of diabetes-related complications in diagnosed individuals (e.g., regular check-ups, medication).
$u_3(t)$: Corresponds to treatment efforts directed at individuals already experiencing complications due to diabetes (e.g., increasing access to healthcare).

Fractional derivatives are essential mathematical tools for improving our comprehension of complicated systems where past states influence future dynamics, according to the information provided in the introduction. The Caputo fractional derivative stands out in this framework because it may be used to solve problems with easily applicable conventional initial conditions. So, the Caputo fractional derivative and control functions have been used to first rethink and the model in (1) as follows:

$$\begin{aligned}
{}_0^C D_t^\kappa H(t) &= \sigma u_1 S - (\mu + \tau) H, \\
{}_0^C D_t^\kappa S(t) &= \Lambda + \tau H - \left(\mu + \alpha + \sigma u_1\right) S, \\
{}_0^C D_t^\kappa D(t) &= \alpha S - \mu D - \lambda D + \omega T - u_2 D, \\
{}_0^C D_t^\kappa C(t) &= \lambda D - \left(\mu + \delta + \gamma u_3\right) C, \\
{}_0^C D_t^\kappa T(t) &= \gamma u_3 C - (\mu + \omega) T + u_2 D.
\end{aligned} \tag{2}$$

with the initial conditions $H(0) = H_0$, $S(0) = S_0$, $D(0) = D_0$, $C(0) = C_0$, $T(0) = T_0$.

4. Parameter estimation

Parameter estimation is one of the most important elements in assessing the accuracy of an epidemiological model. Parameter estimation allows for a more profound understanding of the transmission dynamics of the disease model under consideration and thus more realistic predictions for the future. The following describes the basic idea of this approach.

In this problem, we aim to determine the optimal b parameters for given $bdata$ inputs and observed $ydata$ outputs. This becomes an optimization problem to ensure that our model function $F(b, bdata)$ is as close as possible to the values of $ydata$. Mathematically, this optimization problem is formulated as follows,

$$\min_b \|F(b, bdata) - ydata\|^2$$

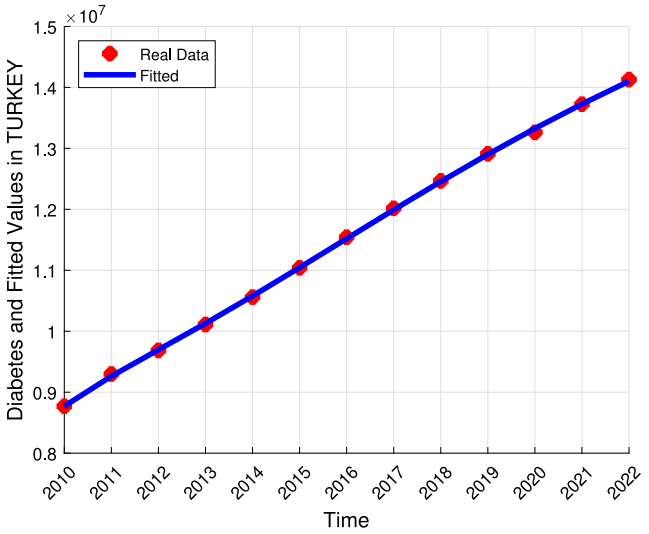

Fig. 1. Fitted model trajectory of individuals with diabetes complications $C(t)$ (blue solid line) and actual diabetes data (red dots) for the years 2010–2022.

Here $bdata$ and $ydata$ are vector quantities. $F(b, bdata)$ is a vector-valued function of the same dimension of the inputs $bdata$.

Parameter estimation for such problems is usually performed under constraints such as upper and lower bounds on the b parameters. To solve this optimization problem, numerical optimization algorithms and tools such as `fmincon` in MATLAB can be used.

In this study, the number of individuals living with diabetes in Türkiye between the years 2010 and 2022 was estimated by integrating data from two primary sources. The age-standardized prevalence rates of diabetes among adults (aged 18 years and above) were obtained from the NCD Risk Factor Collaboration [31], while population data were gathered from the Türkiye Statistical Institute (TÜİK) [32]. Using these datasets, the number of diabetic men and women in Türkiye was calculated separately for each year.

To account for demographic dynamics in the mathematical model, we considered both birth and natural death rates. According to life expectancy data provided by TÜİK, the average life span in Türkiye is approximately 78.6 years [33]. Based on this, the natural death rate μ was computed as the reciprocal of the average lifespan in days:

$$\mu = \frac{1}{365 \times 78.6} \approx 3.49 \times 10^{-5}.$$

Furthermore, the birth rate Λ was estimated by multiplying the natural death rate by the total population of Türkiye, which was approximately 84,664,944 in recent years:

$$\Lambda = \mu \times 84,664,944 \approx 2956.$$

These values provide a realistic demographic framework for use in mathematical modeling of diabetes dynamics in Türkiye. By incorporating reliable and country-specific data, the model aims to better capture the public health implications and future projections of diabetes prevalence.

The number of individuals diagnosed with diabetes in Türkiye between 2010 and 2022 is represented by red dots in Fig. 1. The solid blue curve in the figure shows the simulated trajectory of the compartment representing individuals with diabetes-related complications $C(t)$, obtained by fitting the model parameters to the actual prevalence data. This comparison allows for a visual evaluation of the model's ability to capture the observed dynamics of the disease.

The differences between the actual data and the fitted model values (residuals) are shown in Fig. 2. If the model fits well, the residuals should be randomly distributed. As seen in the figure, the residuals appear to be randomly scattered, indicating that the model provides a reasonably good fit.

The parameter values obtained through the parameter estimation process are presented in Table 1.

5. Existence and uniqueness

Here, we show that the system (2) has a unique solution. Performing the integral to the model (2), we find

$$H(t) - H(0) = \frac{1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left[\sigma u_1 S(\tau) - (\mu + \tau) H(\tau)\right] d\tau,$$

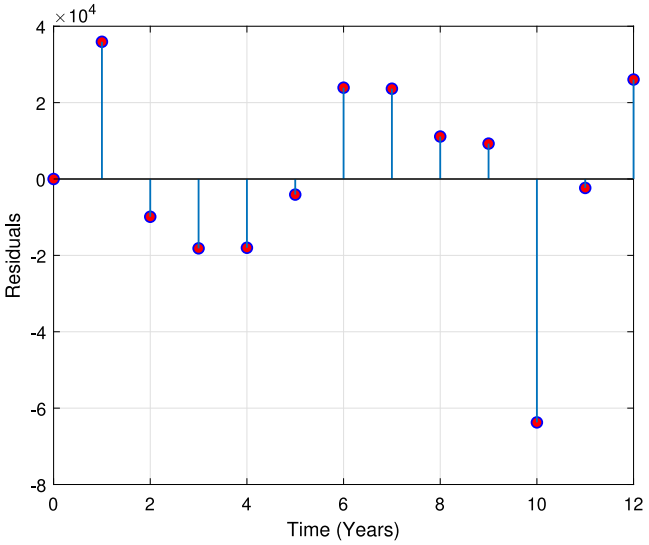

Fig. 2. Distributions of residuals.

Table 1
Model parameters, estimated and fitted values.

Parameter	Value	Source
τ	0.0499	Fitted
σ	0.0367	Fitted
α	0.0823	Fitted
λ	0.1628	Fitted
ω	0.1999	Fitted
δ	0.0129	Fitted
γ	0.0057	Fitted
Λ	2956	Estimated
μ	3.49×10^{-5}	Estimated

$$
\begin{aligned}
S(t) - S(0) &= \frac{1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left[\Lambda + \tau H(\tau) - \left(\mu + \alpha + \sigma u_1\right) S(\tau)\right] d\tau, \\
D(t) - D(0) &= \frac{1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left[\alpha S(\tau) - \mu D(\tau) - \lambda D(\tau) + \omega T(\tau) - u_2(\tau) D(\tau)\right] d\tau, \\
C(t) - C(0) &= \frac{1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left[\lambda D(\tau) - \left(\mu + \delta + \gamma u_3\right) C(\tau) + u_2 \gamma C(\tau)\right] d\tau, \\
T(t) - T(0) &= \frac{1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left[\gamma u_3 C(\tau) - (\mu + \omega) T(\tau) + u_2 D(\tau)\right] d\tau,
\end{aligned} \tag{3}
$$

Now, we can define the following kernels:

$$
\begin{aligned}
G_1(t, H) &= \sigma u_1 S(t) - (\mu + \tau) H(t), \\
G_2(t, S) &= \Lambda + \tau H(t) - \left(\mu + \alpha + \sigma u_1\right) S(t), \\
G_3(t, D) &= \alpha S(t) - \mu D(t) - \lambda D(t) + \omega T(t) - u_2 D(t), \\
G_3(t, C) &= \lambda D(t) - \left(\mu + \delta + \gamma u_3\right) C(t) + u_2 \gamma C(t), \\
G_3(t, T) &= \gamma u_3 C(t) - (\mu + \omega) T(t) + u_2 D(t).
\end{aligned} \tag{4}
$$

Theorem 1. *The kernels G_1, G_2, G_3, G_4 and G_5 provide Lipschitz condition and contraction if the following inequalities holds:*

$$0 < \mu + \tau \leq 1.$$

Proof. Let S and S_1 be two functions. We have

$$\begin{aligned}\left\|G_1(t,H)-G_1\left(t,H_1\right)\right\| &= \left\|-(\mu+\tau)\left(H(t)-H_1(t)\right)\right\| \\ &\leq (\mu+\tau)\left\|H(t)-H_1(t)\right\|.\end{aligned}$$

Let $q_1=\mu+\tau$, we find

$$\left\|G_1(t,H)-G_1\left(t,H_1\right)\right\| \leq q_1\left\|H(t)-H_1(t)\right\|.$$

Thus, we find the Lipschitz condition is satisfied by G_1 and because $0<\mu+\tau\leq 1$, G_1 is also a contraction.

In a similar way, the kernels G_2, G_3, G_4 and G_5 satisfy The Lipschitz condition as shown in the following:

$$\begin{aligned}\left\|G_2(t,S)-G_2\left(t,S_1\right)\right\| &\leq q_2\left\|S(t)-S_1(t)\right\|,\\ \left\|G_3(t,D)-G_3\left(t,D_1\right)\right\| &\leq q_3\left\|D(t)-D_1(t)\right\|,\\ \left\|G_4(t,C)-G_4\left(t,C_1\right)\right\| &\leq q_4\left\|C(t)-C_1(t)\right\|,\\ \left\|G_5(t,T)-G_5\left(t,T_1\right)\right\| &\leq q_5\left\|T(t)-T_1(t)\right\|.\end{aligned}$$

Taking into consideration the kernels G_1, G_2, G_3, G_4, G_5 we can reorganize the model (3) as below:

$$\begin{aligned}H(t) &= H(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_1(\tau,H)\,d\tau,\\ S(t) &= S(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_2(\tau,S)\,d\tau,\\ D(t) &= D(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_3(\tau,D)\,d\tau,\\ C(t) &= C(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_4(\tau,C)\,d\tau,\\ T(t) &= T(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_5(\tau,T)\,d\tau.\end{aligned} \tag{5}$$

We can use the recursive formula that follows:

$$\begin{aligned}H_n(t) &= H(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_1\left(\tau,H_{n-1}\right)d\tau,\\ S_n(t) &= S(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_2\left(\tau,S_{n-1}\right)d\tau,\\ D_n(t) &= D(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_3\left(\tau,D_{n-1}\right)d\tau,\\ C_n(t) &= C(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_4\left(\tau,CI_{n-1}\right)d\tau,\\ T_n(t) &= T(0)+\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}G_5\left(\tau,T_{n-1}\right)d\tau.\end{aligned} \tag{6}$$

where $H_0(t)=H(0)$, $S_0(t)=S(0)$, $D_0(t)=D(0)$, $C_0(t)=C(0)$, $T_0(t)=T(0)$. Then we can get

$$\begin{aligned}\Phi_{1n}(t) &= H_n(t)-H_{n-1}(t)=\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_1\left(\tau,H_{n-1}\right)-G_1\left(\tau,H_{n-2}\right)\right]d\tau,\\ \Phi_{2n}(t) &= S_n(t)-S_{n-1}(t)=\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_2\left(\tau,S_{n-1}\right)-G_2\left(\tau,S_{n-2}\right)\right]d\tau,\end{aligned}$$

$$\Phi_{3n}(t) = D_n(t) - D_{n-1}(t) = \frac{1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left[G_3\left(\tau, D_{n-1}\right) - G_3\left(\tau, D_{n-2}\right) \right] d\tau,$$

$$\Phi_{4n}(t) = C_n(t) - C_{n-1}(t) = \frac{1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left[G_4\left(\tau, C_{n-1}\right) - G_4\left(\tau, C_{n-2}\right) \right] d\tau,$$

$$\Phi_{5n}(t) = T_n(t) - T_{n-1}(t) = \frac{1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left[G_5\left(\tau, T_{n-1}\right) - G_5\left(\tau, T_{n-2}\right) \right] d\tau. \tag{7}$$

where $H_n(t) = \sum_{j=1}^{n} \Phi_{1j}(t)$, $S_n(t) = \sum_{j=0}^{n} \Phi_{2j}(t)$, $D_n(t) = \sum_{j=0}^{n} \Phi_{3j}(t)$, $C_n(t) = \sum_{j=0}^{n} \Phi_{4j}(t)$, $T_n(t) = \sum_{j=0}^{n} \Phi_{5j}(t)$. The norm of both sides of Eq. (7) is obtained as

$$\begin{aligned}
\|\Phi_{1n}(t)\| &= \|H_n(t) - H_{n-1}(t)\| \\
&\leq \frac{1}{\Gamma(\kappa)} \left\| \int_0^t (t-\tau)^{\kappa-1} \left[G_1\left(\tau, H_{n-1}\right) - G_1\left(\tau, H_{n-2}\right) \right] d\tau \right\|, \\
\|\Phi_{2n}(t)\| &= \|S_n(t) - S_{n-1}(t)\| \\
&\leq \frac{1}{\Gamma(\kappa)} \left\| \int_0^t (t-\tau)^{\kappa-1} \left[G_2\left(\tau, S_{n-1}\right) - G_2\left(\tau, S_{n-2}\right) \right] d\tau \right\|, \\
\|\Phi_{3n}(t)\| &= \|D_n(t) - D_{n-1}(t)\| \\
&\leq \frac{1}{\Gamma(\kappa)} \left\| \int_0^t (t-\tau)^{\kappa-1} \left[G_3\left(\tau, D_{n-1}\right) - G_3\left(\tau, D_{n-2}\right) \right] d\tau \right\|, \\
\|\Phi_{4n}(t)\| &= \|C_n(t) - C_{n-1}(t)\| \\
&\leq \frac{1}{\Gamma(\kappa)} \left\| \int_0^t (t-\tau)^{\kappa-1} \left[G_4\left(\tau, C_{n-1}\right) - G_4\left(\tau, C_{n-2}\right) \right] d\tau \right\|, \\
\|\Phi_{5n}(t)\| &= \|T_n(t) - T_{n-1}(t)\| \\
&\leq \frac{1}{\Gamma(\kappa)} \left\| \int_0^t (t-\tau)^{\kappa-1} \left[G_5\left(\tau, T_{n-1}\right) - G_5\left(\tau, T_{n-2}\right) \right] d\tau \right\|.
\end{aligned} \tag{8}$$

Since the kernels satisfy Lipschitz condition, we find

$$\begin{aligned}
\|H_n(t) - H_{n-1}(t)\| &\leq \frac{q_1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \|H_{n-1} - H_{n-2}\| d\tau, \\
\|S_n(t) - S_{n-1}(t)\| &\leq \frac{q_2}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \|S_{n-1} - S_{n-2}\| d\tau, \\
\|D_n(t) - D_{n-1}(t)\| &\leq \frac{q_3}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \|D_{n-1} - D_{n-2}\| d\tau \\
\|C_n(t) - C_{n-1}(t)\| &\leq \frac{q_4}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \|C_{n-1} - C_{n-2}\| d\tau, \\
\|T_n(t) - T_{n-1}(t)\| &\leq \frac{q_5}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \|T_{n-1} - T_{n-2}\| d\tau.
\end{aligned} \tag{9}$$

From the last inequality

$$\begin{aligned}
\|\Phi_{1n}(t)\| &\leq \frac{q_1}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left\| \Phi_{1(n-1)}(\tau) \right\| d\tau, \\
\|\Phi_{2n}(t)\| &\leq \frac{q_2}{\Gamma(\kappa)} \int_0^t (t-\tau)^{\kappa-1} \left\| \Phi_{2(n-1)}(\tau) \right\| d\tau,
\end{aligned}$$

$$\begin{aligned}
\left\|\Phi_{3n}(t)\right\| &\leq \frac{q_3}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|\Phi_{3(n-1)}(\tau)\right\| d\tau,\\
\left\|\Phi_{4n}(t)\right\| &\leq \frac{q_4}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|\Phi_{4(n-1)}(\tau)\right\| d\tau,\\
\left\|\Phi_{5n}(t)\right\| &\leq \frac{q_5}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|\Phi_{5(n-1)}(\tau)\right\| d\tau.
\end{aligned} \tag{10}$$

Using the aforementioned findings, we arrive at the following theorem:

Theorem 2. *The fractional model (2) has a solution under the condition which we are able to find t_0 satisfying*

$$\frac{q_i t_0^{\kappa}}{\Gamma(\kappa+1)} < 1,\ i = 1,2,3,4,5. \tag{11}$$

Proof. Considering the functions $H(t)$, $S(t)$, $D(t)$, $C(t)$, $T(t)$ are bounded and their kernels G_1, G_2, G_3, G_4 and G_5 hold the Lipschitz condition, using Eq. (10):

$$\begin{aligned}
\left\|\Phi_{1n}(t)\right\| &\leq \left\|H_0(t)\right\|\left[\frac{q_1 t_0^{\kappa}}{\Gamma(\kappa+1)}\right]^n,\\
\left\|\Phi_{2n}(t)\right\| &\leq \left\|S_0(t)\right\|\left[\frac{q_2 t_0^{\kappa}}{\Gamma(\kappa+1)}\right]^n,\\
\left\|\Phi_{3n}(t)\right\| &\leq \left\|D_0(t)\right\|\left[\frac{q_3 t_0^{\kappa}}{\Gamma(\kappa+1)}\right]^n,\\
\left\|\Phi_{4n}(t)\right\| &\leq \left\|C_0(t)\right\|\left[\frac{q_4 t_0^{\kappa}}{\Gamma(\kappa+1)}\right]^n\\
\left\|\Phi_{5n}(t)\right\| &\leq \left\|T_0(t)\right\|\left[\frac{q_5 t_0^{\kappa}}{\Gamma(\kappa+1)}\right]^n.
\end{aligned} \tag{12}$$

Here, we show the functions in Eq. (12) are the solutions of fractional model. Suppose that

$$\begin{aligned}
H(t) - H(0) &= H_n(t) - g_{1n}(t),\\
S(t) - S(0) &= S_n(t) - g_{2n}(t),\\
D(t) - D(0) &= D_n(t) - g_{3n}(t),\\
C(t) - C(0) &= C_n(t) - g_{4n}(t),\\
T(t) - T(0) &= T_n(t) - g_{5n}(t).
\end{aligned} \tag{13}$$

We have

$$\begin{aligned}
\left\|g_{1n}(t)\right\| &= \left\|\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_1(\tau,H) - G_1\left(\tau,H_{n-1}\right)\right] d\tau\right\|\\
&\leq \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_1(\tau,H) - G_1\left(\tau,H_{n-1}\right)\right\| d\tau\\
&\leq \frac{t^{\kappa} q_1}{\Gamma(\kappa+1)}\left\|H - H_{n-1}\right\|.
\end{aligned} \tag{14}$$

$$\begin{aligned}
\left\|g_{2n}(t)\right\| &= \left\|\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_2(\tau,S) - G_2\left(\tau,S_{n-1}\right)\right] d\tau\right\|\\
&\leq \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_2(\tau,S) - G_2\left(\tau,S_{n-1}\right)\right\| d\tau\\
&\leq \frac{t^{\kappa} q_2}{\Gamma(\kappa+1)}\left\|S - S_{n-1}\right\|.
\end{aligned} \tag{15}$$

$$\left\|g_{3n}(t)\right\| = \left\|\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_3(\tau,D) - G_3\left(\tau,D_{n-1}\right)\right] d\tau\right\|$$

$$\leq \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_3(\tau,D)-G_3\left(\tau,D_{n-1}\right)\right\|d\tau$$
$$\leq \frac{t^{\kappa}q_3}{\Gamma(\kappa+1)}\left\|D-D_{n-1}\right\|. \tag{16}$$

$$\left\|g_{4n}(t)\right\| = \left\|\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_4(\tau,C)-G_4\left(\tau,C_{n-1}\right)\right]d\tau\right\|$$
$$\leq \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_4(\tau,C)-G_4\left(\tau,C_{n-1}\right)\right\|d\tau$$
$$\leq \frac{t^{\kappa}q_4}{\Gamma(\kappa+1)}\left\|C-C_{n-1}\right\|. \tag{17}$$

and

$$\left\|g_{5n}(t)\right\| = \left\|\frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_5(\tau,T)-G_5\left(\tau,T_{n-1}\right)\right]d\tau\right\|$$
$$\leq \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_5(\tau,T)-G_5\left(\tau,T_{n-1}\right)\right\|d\tau$$
$$\leq \frac{t^{\kappa}q_5}{\Gamma(\kappa+1)}\left\|T-T_{n-1}\right\|, \tag{18}$$

repeating these steps

$$\left\|g_{1n}(t)\right\| \leq \left(\frac{t^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_1^n\gamma,$$

$$\left\|g_{2n}(t)\right\| \leq \left(\frac{t^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_2^n\gamma,$$

$$\left\|g_{3n}(t)\right\| \leq \left(\frac{t^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_3^n\gamma,$$

$$\left\|g_{4n}(t)\right\| \leq \left(\frac{t^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_4^n\gamma,$$

and

$$\left\|g_{5n}(t)\right\| \leq \left(\frac{t^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_5^n\gamma.$$

Considering above equations at the point t_0, we obtain

$$\left\|g_{1n}(t)\right\| \leq \left(\frac{t_0^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_1^n\gamma,$$
$$\left\|g_{2n}(t)\right\| \leq \left(\frac{t_0^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_2^n\gamma,$$
$$\left\|g_{3n}(t)\right\| \leq \left(\frac{t_0^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_3^n\gamma,$$
$$\left\|g_{4n}(t)\right\| \leq \left(\frac{t_0^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_4^n\gamma,$$
$$\left\|g_{5n}(t)\right\| \leq \left(\frac{t_0^{\kappa}}{\Gamma(\kappa+1)}\right)^{n+1} q_5^n\gamma.$$

Applying the limit to both sides of the last inequalities as $n \to \infty$ and taking into account Theorem 1, we obtain $\left\|g_{1n}(t)\right\| \to 0$, $\left\|g_{2n}(t)\right\| \to 0$, $\left\|g_{3n}(t)\right\| \to 0$, $\left\|g_{4n}(t)\right\| \to 0$, and $\left\|g_{5n}(t)\right\| \to 0$.

Theorem 3. *The fractional model (2) has a unique solution.*

Proof. Suppose that there exists another solution of the system, in other words $H_1(t)$, $S_1(t)$, $D_1(t)$, $C_1(t)$, $T_1(t)$. Then we write

$$\begin{aligned}
\|H(t)-H_1(t)\| &= \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_1(\tau,H)-G_1\left(\tau,H_1\right)\right]d\tau,\\
\|S(t)-S_1(t)\| &= \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_1(\tau,S)-G_1\left(\tau,S_1\right)\right]d\tau,\\
\|D(t)-D_1(t)\| &= \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_1(\tau,D)-G_1\left(\tau,D_1\right)\right]d\tau,\\
\|C(t)-C_1(t)\| &= \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_2(\tau,C)-G_2\left(\tau,C_1\right)\right]d\tau,\\
\|T(t)-T_1(t)\| &= \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left[G_3(\tau,T)-G_3\left(\tau,T_1\right)\right]d\tau.
\end{aligned} \tag{19}$$

Applying the norm to both side of Eq. (19), we gain

$$\begin{aligned}
\|H(t)-H_1(t)\| &\le \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_1(\tau,H)-G_1\left(\tau,H_1\right)\right\|d\tau,\\
\|S(t)-S_1(t)\| &\le \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_2(\tau,S)-G_2\left(\tau,S_1\right)\right\|d\tau,\\
\|D(t)-D_1(t)\| &\le \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_3(\tau,D)-G_3\left(\tau,D_1\right)\right\|d\tau,\\
\|C(t)-C_1(t)\| &\le \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_4(\tau,C)-G_4\left(\tau,C_1\right)\right\|d\tau,\\
\|T(t)-T_1(t)\| &\le \frac{1}{\Gamma(\kappa)}\int_0^t (t-\tau)^{\kappa-1}\left\|G_5(\tau,T)-G_5\left(\tau,T_1\right)\right\|d\tau.
\end{aligned} \tag{20}$$

The kernels G_1, G_2, G_3, G_4, G_5 verify the Lipschitz condition, we find

$$\begin{aligned}
\|H(t)-H_1(t)\| &\le \frac{q_1 t^{\kappa}}{\Gamma(\kappa+1)}\|H(t)-H_1(t)\|,\\
\|S(t)-S_1(t)\| &\le \frac{q_2 t^{\kappa}}{\Gamma(\kappa+1)}\|S(t)-S_1(t)\|,\\
\|D(t)-D_1(t)\| &\le \frac{q_3 t^{\kappa}}{\Gamma(\kappa+1)}\|D(t)-D_1(t)\|,\\
\|C(t)-C_1(t)\| &\le \frac{q_4 t^{\kappa}}{\Gamma(\kappa+1)}\|C(t)-C_1(t)\|,\\
\|T(t)-T_1(t)\| &\le \frac{q_5 t^{\kappa}}{\Gamma(\kappa+1)}\|T(t)-T_1(t)\|,
\end{aligned} \tag{21}$$

which yields

$$\begin{aligned}
\|H(t)-H_1(t)\|\left(1-\frac{q_1 t^{\kappa}}{\Gamma(\kappa+1)}\right) &\le 0,\\
\|S(t)-S_1(t)\|\left(1-\frac{q_2 t^{\kappa}}{\Gamma(\kappa+1)}\right) &\le 0,\\
\|D(t)-D_1(t)\|\left(1-\frac{q_3 t^{\kappa}}{\Gamma(\kappa+1)}\right) &\le 0\\
\|C(t)-C_1(t)\|\left(1-\frac{q_4 t^{\kappa}}{\Gamma(\kappa+1)}\right) &\le 0,\\
\|T(t)-T_1(t)\|\left(1-\frac{q_5 t^{\kappa}}{\Gamma(\kappa+1)}\right) &\le 0.
\end{aligned} \tag{22}$$

Thus, $\|H(t) - H_1(t)\| = 0$, $\|S(t) - S_1(t)\| = 0$, $\|D(t) - D_1(t)\| = 0$, $\|C(t) - C_1(t)\| = 0$, $\|T(t) - T_1(t)\| = 0$ which present $H(t) = H_1(t)$, $S(t) = S_1(t)$, $D(t) = D_1(t)$, $C(t) = C_1(t)$, $T(t) = T_1(t)$. In conclusion, the model has a unique solution.

6. Equilibrium points

In order to better understand the behavior of the system over time, we investigate its equilibrium points, focusing on both the disease-free and the endemic cases.

To determine the disease-free equilibrium point, we assume that there is no diabetes present in the population. This corresponds to setting the diabetes-related compartments to zero, that is,

$$H(t) = D(t) = C(t) = T(t) = 0.$$

Under this assumption and neglecting the control parameters (u_i, $i = 1, 2, 3$) of the model, we set the right-hand side of system (2) equal to zero and solve for the susceptible population $S(t)$. As a result, we obtain the disease-free equilibrium point as

$$E_0 = (0, \frac{\Lambda}{\mu + \alpha + \sigma}, 0, 0, 0).$$

Next, we consider the endemic equilibrium point, which represents a situation where diabetes remains in the population at a constant level over time. To find this equilibrium, we set the right-hand side of system (2) equal to zero and solving the resulting equations for all variables.

As a result, we obtain the endemic equilibrium point as

$$E_1 = (H^*, S^*, D^*, C^*, T^*),$$

where

$$\begin{aligned}
H^* &= \frac{\sigma\Lambda}{k_1k_2 - \tau\sigma}, \\
S^* &= \frac{k_1\Lambda}{k_1k_2 - \tau\sigma}, \\
D^* &= \frac{k_1k_4k_5\alpha\Lambda}{(k_3k_4k_5 - \gamma\lambda\omega)(k_1k_2 - \tau\sigma)}, \\
C^* &= \frac{k_1k_5\alpha\lambda\Lambda}{(k_3k_4k_5 - \gamma\lambda\omega)(k1k2 - \tau\sigma)}, \\
T^* &= \frac{k_1\alpha\lambda\gamma\Lambda}{(k_3k_4k_5 - \gamma\lambda\omega)(k_1k_2 - \tau\sigma)}.
\end{aligned}$$

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

This point reflects a long-term condition in which diabetes persists in the population.

To analyze the local asymptotic stability of the equilibrium points of system (2), we begin by computing the Jacobian matrix of the system.

After calculating the partial derivatives of the right-hand sides of system (2) with respect to each compartment, the Jacobian matrix is obtained as follows:

$$J = \begin{pmatrix} -\mu - \tau & \sigma & 0 & 0 & 0 \\ \tau & -\mu - \alpha - \sigma & 0 & 0 & 0 \\ 0 & \alpha & -\mu - \lambda & 0 & w \\ 0 & 0 & \lambda & -\mu - \delta - \gamma & 0 \\ 0 & 0 & 0 & \gamma & -\mu - \omega \end{pmatrix}. \tag{23}$$

As can be seen, the Jacobian matrix is independent of the model compartments. Therefore, showing that all the eigenvalues of the Jacobian matrix lie in the left half-plane is sufficient to prove the local asymptotic stability of both the disease-free and endemic equilibrium points.

To achieve this, let us consider the Jacobian matrix (23) as a block matrix, given by the following:

$$J = \begin{pmatrix} A & B \\ C & D \end{pmatrix}, \tag{24}$$

where

$$A = \begin{pmatrix} -\mu - \tau & \sigma & 0 \\ \tau & -\mu - \alpha - \sigma & 0 \\ 0 & \alpha & -\mu - \lambda \end{pmatrix}, \quad B = \begin{pmatrix} 0 & 0 \\ 0 & 0 \\ 0 & w \end{pmatrix}, \quad C = \begin{pmatrix} 0 & 0 & \lambda \\ 0 & 0 & 0 \end{pmatrix},$$

$$D = \begin{pmatrix} -\mu - \delta - \gamma & 0 \\ \gamma & -\mu - w \end{pmatrix}.$$

The characteristic equation of the Jacobian (24), expressed in block-matrix form, can then be stated as follows:

$$det(A - \rho I).det((D - \rho I) - C(A - \rho I)^{-1}B) = 0. \tag{25}$$

By computing the determinant of the block A matrix through a Laplace expansion along its third column, the first eigenvalue is obtained as $-\mu - \lambda$ The remaining 2×2 submatrix has a negative trace and a positive determinant, ensuring that the other two eigenvalues lie in the left half-plane.

Focusing now on the second factor, the characteristic equation

$$\det\left((D-\rho I)-C(A-\rho I)^{-1}B\right)=0$$

can be expressed as

$$\rho^3 + a_1\rho^2 + a_2\rho + a_3 = 0,$$

where

$$\begin{aligned}
a_1 &= \gamma + \delta + \lambda + 3\mu + \omega,\\
a_2 &= \lambda\delta + 2\mu\delta + \omega\delta + \gamma\lambda + 2\gamma\mu + \omega\gamma + 2\lambda\mu + \lambda\omega + 3\mu^2 + 2\mu\omega,\\
a_3 &= \delta\lambda\mu + \delta\lambda\omega + \delta\mu^2 + \delta\mu\omega + \gamma\lambda\mu + \gamma\mu^2 + \gamma\mu\omega + \lambda\mu^2 + \lambda\mu\omega + \mu^3 + \mu^2\omega.
\end{aligned}$$

Since $a_1 > 0$, $a_2 > 0$, $a_3 > 0$, and $a_1a_2 > a_3$ are satisfied, application of the Routh–Hurwitz criterion confirms that both the disease-free and endemic equilibrium points are locally asymptotically stable.

7. Fractional optimal control problem

In this section, we develop a fractional optimal control problem for the diabetes model using the Caputo fractional derivative of order $0 < \kappa < 1$. The model incorporates three time-dependent control variables corresponding to distinct intervention strategies: u_1 represents public health campaigns and awareness training aimed at reducing susceptibility; u_2 models prevention efforts to avert the onset of diabetes-related complications; and u_3 captures treatment efforts directed at individuals already experiencing complications due to diabetes. The mathematical model for diabetes, which includes control parameters, is formulated and presented in Eq. (2).

To analyze the optimal control problem, the objective functional, which quantifies the desired outcomes and associated costs, is introduced in the following subsection.

7.1. Objective functional

The objective of the optimal control problem is to minimize both the number of individuals with diabetes (D) and those with diabetes-related complications (C), as well as the economic or implementation costs associated with applying the control strategies. To achieve this, the performance index (also known as the objective function) is defined as follows:

$$\min_{u_1,u_2,u_3} \mathcal{J}(u_1,u_2,u_3) = \int_0^T \left(\widetilde{\kappa_1}D(t) + \widetilde{\kappa_2}C(t) + \widetilde{\kappa_3}u_1(t)^2 + \widetilde{\kappa_4}u_2(t)^2 + \widetilde{\kappa_5}u_3(t)^2\right)dt, \tag{26}$$

subject to the state system:

$$\begin{cases}
{}_0^C D_t^\kappa H(t) = -(\mu+\tau)H + \sigma u_1 S,\\
{}_0^C D_t^\kappa S(t) = \Lambda + \tau H - (\mu + \alpha + \sigma u_1)S,\\
{}_0^C D_t^\kappa D(t) = \alpha S - (\mu + \lambda + u_2)D + \omega T,\\
{}_0^C D_t^\kappa C(t) = \lambda D - (\mu + \delta + \gamma u_3)C,\\
{}_0^C D_t^\kappa T(t) = \gamma u_3 C - (\mu + \omega)T + u_2 D,
\end{cases} \tag{27}$$

with initial conditions given by:

$$(H(0), S(0), D(0), C(0), T(0)) = (H_0, S_0, D_0, C_0, T_0) \geq 0. \tag{28}$$

Parameters $\widetilde{\kappa_i} > 0$, $i = 1, \ldots, 5$ are positive weighting constants that reflect the relative importance of reducing the disease burden versus the cost of control implementation. The control functions $u_1(t), u_2(t), u_3(t)$ are chosen from the set of admissible control set $\mathcal{U}$, which is defined as:

$$\mathcal{U} = \left\{(u_1,u_2,u_3) \in [L^\infty(0,T)] \;\middle|\; 0 \leq u_i(t) \leq u_{i,\max},\ \forall t \in [0,T],\ i = 1,2,3\right\}, \tag{29}$$

where $u_{i,\max}$ denotes the upper bound for the ith control, which is taken as 1 in our simulations. The set $\mathcal{U}$ ensures that each control is bounded, measurable, and takes values within a realistic and implementable range throughout the time interval $[0, T]$.

To determine the optimal control strategy for the diabetes model involving three control variables, it is essential to derive the necessary conditions for optimality. In the following subsection, these conditions are established by applying Pontryagin's maximum principle for fractional optimal control [26,34,35], and the corresponding Hamiltonian function associated with the system is constructed accordingly.

7.2. Hamiltonian and necessary conditions

To determine the optimal controls $u_1(t)$, $u_2(t)$, and $u_3(t)$, we apply Pontryagin's Maximum Principle to our fractional optimal control problem. For this purpose, we define the Hamiltonian function $\mathcal{H}$, which incorporates the dynamics of the state variables, the control variables, and the associated adjoint variables $p_i(t)$, $i = 1, \dots, 5$. The Hamiltonian $\mathcal{H}$ takes the following form:

$$\begin{aligned}\mathcal{H} &= \widetilde{\kappa_1} D + \widetilde{\kappa_2} C + \widetilde{\kappa_3} u_1^2 + \widetilde{\kappa_4} u_2^2 + \widetilde{\kappa_5} u_3^2 \\ &+ p_1 \left(-(\mu+\tau)H + \sigma u_1 S\right) + p_2 \left(\Lambda + \tau H - (\mu+\alpha+\sigma u_1)S\right) \\ &+ p_3 \left(\alpha S - (\mu+\lambda+u_2)D + \omega T\right) + p_4 \left(\lambda D - (\mu+\delta+\gamma u_3)C\right) \\ &+ p_5 \left(\gamma u_3 C - (\mu+\omega)T + u_2 D\right). \end{aligned} \tag{30}$$

Here, the variables $p_i(t)$ represent the adjoint (or co-state) variables corresponding to each of the five state variables $H(t), S(t), D(t), C(t)$, and $T(t)$, respectively.

The evolution of the adjoint variables is governed by the system of differential equations given by:

$${}_tD_T^{\kappa} p_i(t) = \frac{\partial \mathcal{H}}{\partial x_i}, \quad \text{for } i = 1, 2, 3, 4, 5, \tag{31}$$

where $x_1 = H(t), x_2 = S(t), x_3 = D(t), x_4 = C(t), x_5 = T(t)$, and ${}_tD_T^{\kappa}$ denotes the right-sided Riemann–Liouville fractional derivative of order κ.

Together with the system dynamics, the Hamiltonian, and the transversality conditions $p_i(T) = 0$, the above adjoint equations constitute the necessary conditions for optimality. The optimal control functions $u_1(t), u_2(t), u_3(t)$ are then determined by minimizing the Hamiltonian pointwise with respect to the controls over the admissible set.

Theorem 4. *Consider the fractional optimal control problem defined by Eqs. (26)–(28). Let $(H^*, S^*, D^*, C^*, T^*)$ be the optimal state trajectory corresponding to the optimal control functions (u_1^*, u_2^*, u_3^*). Then, there exist costate functions $p_i(t)$, for $i = 1, \dots, 5$, that satisfy the following differential equations in the sense of right-sided Riemann–Liouville fractional derivative:*

$$\begin{aligned}
{}_tD_T^{\kappa} p_1(t) &= p_2\,\tau - p_1\,(\mu+\tau), \\
{}_tD_T^{\kappa} p_2(t) &= \alpha\,p_3 - p_2\,(\alpha+\mu+\sigma\,u_1) + p_1\,\sigma\,u_1, \\
{}_tD_T^{\kappa} p_3(t) &= \kappa_1 + \lambda\,p_4 + p_5\,u_2 - p_3\,(\lambda+\mu+u_2), \\
{}_tD_T^{\kappa} p_4(t) &= \kappa_2 - p_4\,(\delta+\mu+\upsilon+\gamma\,u_3) + \gamma\,p_5\,u_3, \\
{}_tD_T^{\kappa} p_5(t) &= \omega\,p_3 - p_5\,(\mu+\omega),
\end{aligned}$$

With transversality conditions:

$$p_i(T) = 0, \quad \textit{for } i = 1, \dots, 5.$$

Moreover, the optimal control functions (u_1^*, u_2^*, u_3^*) are characterized by:

$$\begin{aligned}
u_1^*(t) &= \min\left\{u_{1,\max}, \max\left\{0, -\frac{\sigma S(p_1 - p_2)}{2\widetilde{\kappa_3}}\right\}\right\}, \\
u_2^*(t) &= \min\left\{u_{2,\max}, \max\left\{0, \frac{D(p_3 - p_5)}{2\widetilde{\kappa_4}}\right\}\right\}, \\
u_3^*(t) &= \min\left\{u_{3,\max}, \max\left\{0, \frac{\gamma C(p_4 - p_5)}{2\widetilde{\kappa_5}}\right\}\right\}.
\end{aligned}$$

Proof. To derive the necessary conditions for optimality, we apply Pontryagin's Maximum Principle for fractional optimal control problems. The state dynamics are described by a system of Caputo fractional differential equations of order $\kappa \in (0, 1]$, and the adjoint system is governed by the right-sided Caputo fractional derivative.

Let the Hamiltonian $\mathcal{H}$ be defined as in Eq. (30). According to the maximum principle, the costate variables $p_i(t)$, for $i = 1, \dots, 5$, must satisfy the adjoint system:

$${}_tD_T^{\kappa} p_i(t) = \frac{\partial \mathcal{H}}{\partial x_i},$$

where x_i represents the corresponding state variable among H, S, D, C, and T.

Differentiating $\mathcal{H}$ with respect to each state variable yields:

$$\begin{aligned}
{}_tD_T^{\kappa} p_1 &= p_2\tau - p_1(\mu+\tau), \\
{}_tD_T^{\kappa} p_2 &= \alpha p_3 - p_2(\alpha+\mu+\sigma u_1) + p_1\sigma u_1, \\
{}_tD_T^{\kappa} p_3 &= \widetilde{\kappa_1} + \lambda p_4 + p_5 u_2 - p_3(\lambda+\mu+u_2), \\
{}_tD_T^{\kappa} p_4 &= \widetilde{\kappa_2} - p_4(\delta+\mu+\upsilon+\gamma u_3) + \gamma p_5 u_3.
\end{aligned}$$

Table 2
Reduction percentages and detailed values of the cost functional (26).

Strategy	Value of the cost functional	Reduction (%)
$u_1, u_2, u_3 = 0$	17633714541.8285	–
$u_1, u_2, u_3 \neq 0$	10683809170.123	39.42

The transversality conditions for the adjoint variables are:

$$p_i(T) = 0, \quad \text{for } i = 1, \dots, 5.$$

To determine the expressions for the optimal controls, we use the condition

$$\frac{\partial \mathcal{H}}{\partial u_i} = 0, \quad i = 1, 2, 3.$$

Solving these conditions yields the following characterizations of the optimal controls:

$$u_1^* = -\frac{\sigma S(p_1 - p_2)}{2\widetilde{\kappa_3}},$$
$$u_2^* = \frac{D(p_3 - p_5)}{2\widetilde{\kappa_4}},$$
$$u_3^* = \frac{\gamma C(p_4 - p_5)}{2\widetilde{\kappa_5}}.$$

This completes the proof.

8. Numerical results and discussion

To solve the proposed fractional optimal control problem, we employed a numerical scheme that combines the Predictor–Evaluator–Corrector–Evaluator (PECE) method with the Forward–Backward Sweep Method (FBSM). The PECE algorithm is a well-established approach for approximating solutions of fractional differential equations, particularly effective for handling Caputo derivatives due to its accuracy and stability properties [36]. In this study, the PECE method is used to numerically integrate the state and adjoint systems over the time interval of interest. The optimal controls are updated iteratively using the FBSM, which alternates between solving the state equations forward in time and the adjoint equations backward in time, refining the control variables at each iteration [37]. The implementation of the algorithm was carried out in MATLAB, inspired by the structure and methodology presented in [38], which offers a practical framework for implementing fractional optimal control strategies in a biological context.

In our numerical experiments, we use the parameter values listed in Table 1, which were obtained based on the prevalence of diabetic patients in Türkiye between 2010 and 2022. The fractional derivative order is set to $\kappa = 0.9$, and the initial conditions for each compartment are determined from epidemiological data. Specifically, the initial number of healthy individuals is $H(0) = 2.9 \times 10^7$, susceptible individuals $S(0) = 3.1 \times 10^7$, diabetic individuals without complications $D(0) = 1.41 \times 10^7$, diabetic individuals with complications $C(0) = 1.0 \times 10^7$, and individuals under treatment $T(0) = 1.0 \times 10^7$. Moreover, the weighting constants in (26) are taken as $\widetilde{\kappa_1} = 0.9$, $\widetilde{\kappa_2} = 0.9$, $\widetilde{\kappa_3} = 0.01$, $\widetilde{\kappa_4} = 0.01$, and $\widetilde{\kappa_5} = 0.01$.

The effect of the applied optimal control strategy on the system is clearly seen when evaluated through performance index (26) values. While the performance index value obtained in the uncontrolled scenario is approximately 1.76×10^{10}, this value drops to 1.07×10^{10} when the three control inputs are applied together. This decrease indicates that the control inputs have a significant effect on the model and that the system has been optimized in the desired direction. The relevant values are shown in Table 2. The approximate %39.4 decrease achieved by applying the controls is noteworthy, especially in terms of demonstrating the effectiveness of health policies in the long term.

Fig. 3 illustrates the temporal evolution of all compartments under both controlled and uncontrolled scenarios. Examining Fig. 3 in detail, it is observed that the implementation of the three control variables significantly influences the system dynamics. The control u_1, representing public health campaigns and awareness training, effectively reduces the number of susceptible individuals $S(t)$. The control u_2, which focuses on preventing complications among diabetics, leads to a dramatic decrease in the diabetic without complications class $D(t)$. Likewise, the control u_3, targeting treatment efforts for diabetics with complications, results in a notable reduction in the $C(t)$ class. Additionally, due to the combined effect of these controls, an increase is observed in the healthy $H(t)$ and treatment $T(t)$ compartments, highlighting the overall positive impact of the proposed optimal control strategy.

As can be seen in Fig. 4, the control parameters remain close to their upper limit value over time. This indicates that each control input is actively applied throughout the simulation period and has a continuous effect on the model. In particular, the simultaneous maintenance of the control variables $u_1(t)$, $u_2(t)$, and $u_3(t)$ plays a crucial role in reducing the spread rate of diabetes and preventing the development of complications. The exclusion of any of the control variables or the interruption of their implementation will slow down the decline in the number of individuals with diabetes and reduce the beneficial effects predicted by the model.

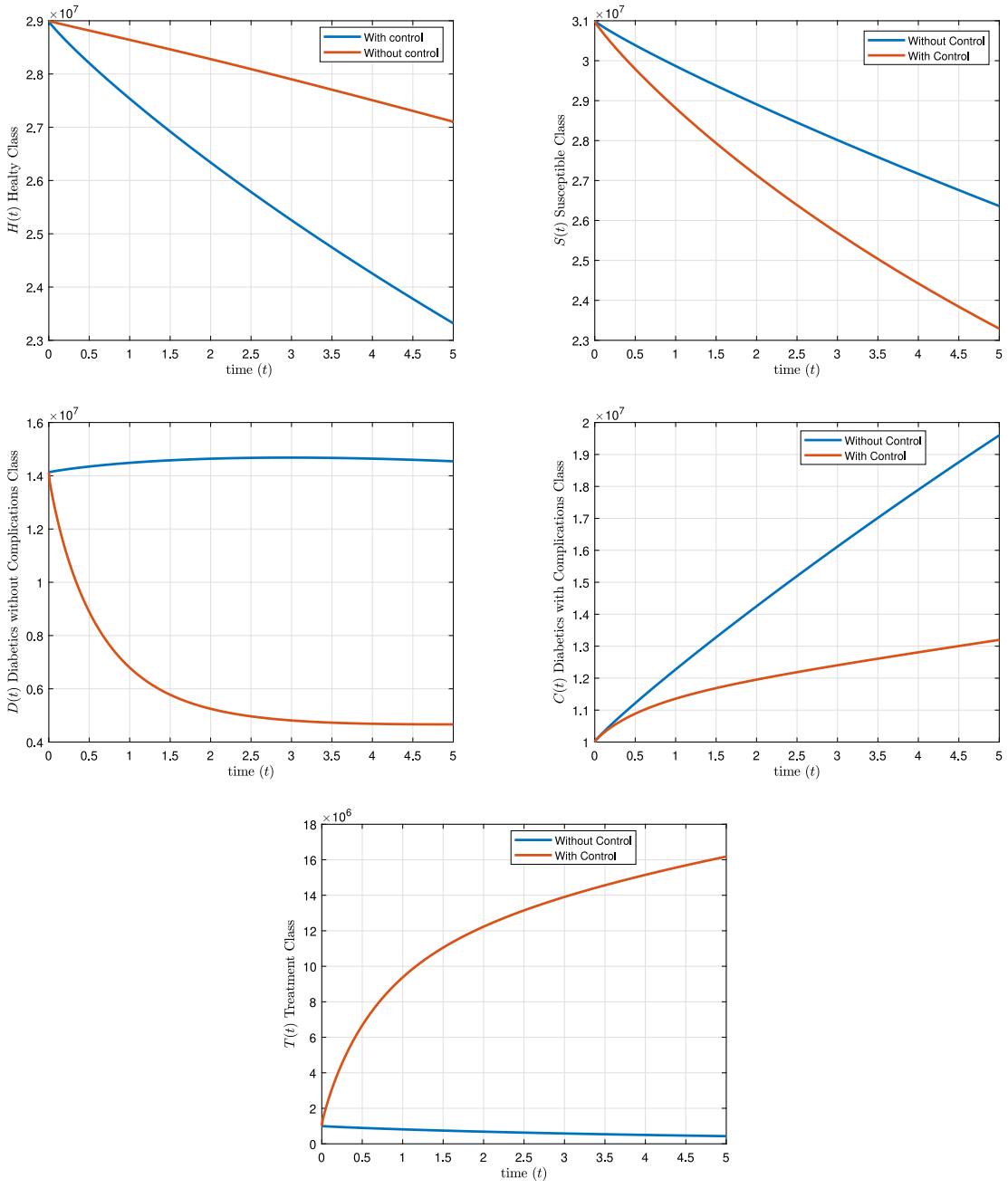

Fig. 3. Diabetes disease dynamics for $\alpha = 0.90$ with and without control.

In recent years, rapid technological advances, individuals' transition to more sedentary lifestyles, and the widespread adoption of fast food culture have contributed to the widespread prevalence of diabetes among the population. Our numerical experiments demonstrate that the integrated application of three control strategies is of significant importance in directly achieving epidemiological targets. Additionally, it is evident that this approach will also yield important socio-economic benefits indirectly. Reducing the number of individuals with diabetes over time could lower long-term healthcare expenditures, increase labor productivity, and ultimately contribute to national economic growth. Therefore, it is imperative that health authorities and relevant institutions take the necessary measures within this control framework to alleviate the burden of diabetes and strengthen the resilience of our healthcare systems.

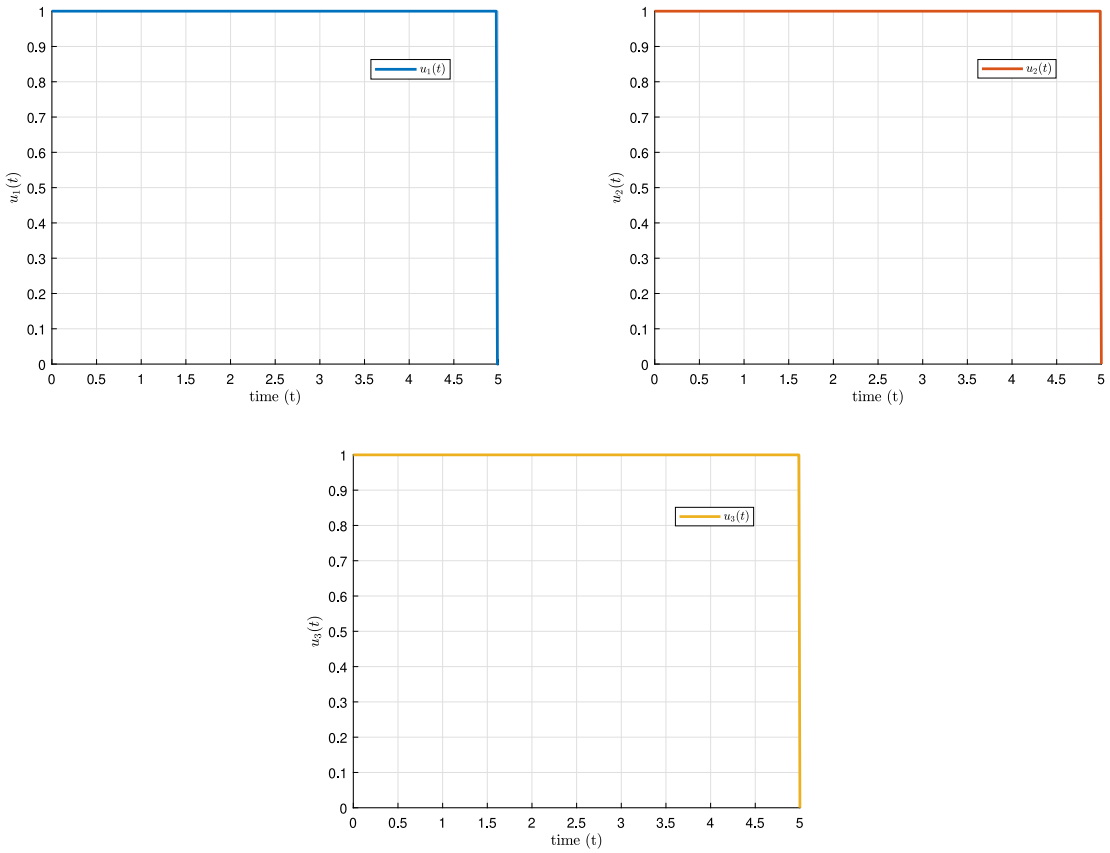

Fig. 4. The temporal progression of the control variables $u_1(t)$, $u_2(t)$, and $u_3(t)$.

9. Concluding remarks

In the model considered in this study, the control parameter needed to create awareness about diabetes is added, and appropriate differential equations are used to represent the development process of the disease. The parameter estimation process increased the accuracy of the model by ensuring that the model is compatible with real-life data. The analyses made within the framework of control theory offer significant potential in the development of strategies for the treatment of diabetes. Interventions such as awareness training is simulated through the model, and effective control strategies are determined. Finally, the obtained graphics provided a better understanding of creating awareness about diabetes by visualizing the dynamics of the model. These graphics provide useful information for clinical applications by visually displaying the effects of changes in different parameters on the course of the disease. Further data integration and model development with further research will be beneficial in terms of better understanding the disease and optimizing treatment strategies.

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Data availability

Data will be made available on request.

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