fractal and fractional

MDPI

Article

Mathematical and Numerical Analysis of a Fractional Diabetes Model with Singular Operator

Pratibha Verma * and Wojciech Sumelka

Institute of Structural Analysis, Poznan University of Technology, Piotrowo 5 Street, 60-965 Poznan, Poland; wojciech.sumelka@put.poznan.pl

* Correspondence: pratibha.verma@put.poznan.pl

Abstract

Diabetes mellitus is a chronic disease with complex progression dynamics. This study introduces a fractional order compartmental model based on the Caputo derivative, a singular-kernel derivative, to describe disease progression across four compartments: susceptible, insulin-resistant, diabetic without complications, and diabetic with complications. The model is novel for integrating memory effects into disease-stage transitions while maintaining dimensional consistency. Key mathematical properties, including existence, uniqueness, positivity, boundedness, equilibrium analysis, and both local and global stability, are established. Ulam–Hyers stability is also examined to evaluate the robustness of the model solutions. Numerical approximations are obtained using the Adomian Decomposition Method and its Laplace variant. Simulations indicate that lower fractional orders enhance memory effects, slow disease progression, and influence long-term dynamics. These results demonstrate that the proposed approach provides a flexible and robust framework for studying chronic disease progression and makes a meaningful contribution to the literature on fractional diabetes models.

Keywords: fractional order model; diabetes mellitus; singular kernel derivative; stability analysis; existence and uniqueness; numerical simulation; convergence analysis

1. Background and Literature Review

Type 2 diabetes is a long-term metabolic disease that occurs when the body cannot use insulin properly, leading to high glucose levels in the blood. It has become one of the most common chronic diseases worldwide and is considered a serious public health issue. The number of people affected by diabetes has increased rapidly in recent decades, especially in developing countries, creating a growing burden on healthcare systems. Many international efforts have been made to raise awareness about diabetes and its consequences. These initiatives encourage early diagnosis, healthier lifestyles, and better disease management. However, despite these efforts, the number of cases continues to increase, and a large proportion of people living with diabetes are not yet diagnosed. Current trends suggest that the global prevalence of diabetes will continue to increase in the coming years. The development of type 2 diabetes is related to both genetic and lifestyle factors. Although genetic predisposition cannot be controlled, lifestyle factors such as poor diet, lack of physical activity, and unhealthy habits play a major role in the onset of the disease. In addition, social and economic conditions can influence individual behavior, increasing the risk of developing diabetes. If not properly managed, the disease can lead to serious complications, including heart disease, kidney problems, and vision loss [1,2].

Check for updates

Academic Editor: Carlo Cattani

Received: 6 April 2026
Revised: 29 April 2026
Accepted: 6 May 2026
Published: 9 May 2026

Copyright: © 2026 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license.

Recent events, particularly the COVID-19 pandemic, have shown how lifestyle changes can affect health. Restrictions on movement and the closure of sports and recreational facilities reduced physical activity and increased sedentary behavior. As a result, many people experienced weight gain, which is a known risk factor for type 2 diabetes. Several studies have attempted to understand the progression of diabetes using mathematical approaches. These studies have provided useful information on how the disease develops and how various factors influence its progression. However, many of these works focus mainly on biological aspects and pay less attention to social and environmental influences. In reality, human behavior is strongly affected by social interactions. People often adopt habits similar to those around them, especially within the same age group. These interactions can either increase or reduce the risk of developing diabetes, depending on the group's lifestyle patterns. Unlike genetic factors, these influences can be modified, making them an important area of intervention [3,4].

In Algeria, type 2 diabetes has become increasingly common in recent years. Studies conducted in different regions have reported high prevalence rates and a significant number of undiagnosed cases. The disease is often associated with other conditions such as obesity and hypertension, which further complicates its management. Based on these observations, this work focuses on the role of biological and socio-environmental factors in the progression of type 2 diabetes. The aim is to provide a better understanding of how these factors interact and contribute to the spread of the disease, to support more effective prevention and management strategies [1].

Fractional calculus has become an important tool for describing complex real-world phenomena, particularly when classical integer-order models are insufficient. Unlike standard derivatives, fractional order operators allow non-integer orders, making them more flexible and better suited to capturing the dynamics of many natural processes. Over time, this field has attracted increasing attention, particularly for its ability to provide more accurate and realistic descriptions of models that involve memory and hereditary effects [5,6].

In recent years, a variety of fractional derivative operators have been introduced, each with different mathematical properties and applications. Classical definitions include the Caputo and Riemann–Liouville derivatives, both of which are based on singular kernels. More recent developments have led to operators without singular kernels, such as the Caputo–Fabrizio and Atangana–Baleanu fractional derivatives, which incorporate exponential and Mittag–Leffler (M-L)-type kernels, respectively (see, e.g., [7–9] and references therein). These newer formulations have been widely used for their improved analytical properties and their ability to better represent real-world dynamics [10,11].

The main advantage of fractional order models lies in their non-local nature. Unlike classical differential equations, fractional derivatives account for the entire history of the model rather than relying solely on its current state. This feature is particularly relevant for chronic diseases such as type 2 diabetes, where disease progression depends not only on current conditions, but also on past behaviors, long-term lifestyle patterns, and accumulated physiological changes.

Researchers have developed several mathematical models to describe the progression of diabetes. Traditional models often use ordinary differential equations (ODEs) to represent metabolic interactions and disease progression in different parts of the body. However, these models usually do not include the memory and hereditary factors that are important in chronic diseases such as diabetes.

To address these gaps, researchers have begun using fractional order models. These models use fractional derivatives, such as the Caputo derivative, to introduce memory effects. This approach provides a more realistic view of how the disease changes over time,

which is important in diabetes because past health conditions and accumulated effects shape its progression.

Fractional calculus has already been successfully applied in many fields, including engineering, physics, biology, and medicine. In epidemiology and health modeling, fractional order models have been shown to provide a more accurate description of disease dynamics than classical approaches. This is mainly because they can incorporate memory effects, which play a crucial role in long-term diseases and conditions characterized by gradual change [12,13].

In the context of diabetes, the use of fractional derivatives is particularly relevant. The development and progression of type 2 diabetes are strongly influenced by long-term factors such as diet, physical activity, and metabolic history. These factors evolve and cannot be fully captured using standard integer-order models. Fractional order models, on the other hand, provide a more realistic representation of these cumulative effects, making them well-suited for studying disease dynamics.

Among the available operators, the Caputo derivative is often preferred in applied modeling because it allows the use of classical initial conditions with clear physical interpretation, making it easier to relate the mathematical model to real-world data. In addition, fractional order models can account for the uncertainties and variability commonly observed in biological systems, which classical models may be limited in capturing [14]. In the present study, the Caputo derivative is adopted because it provides a suitable framework for capturing long-memory effects while supporting established analytical tools, including stability analysis. Although alternative operators, such as the Caputo–Fabrizio and Atangana–Baleanu derivatives, have also been proposed, concerns have been raised regarding the theoretical consistency and inverse properties of certain non-singular kernel operators, particularly in the context of initial value problems [15]. These considerations further motivate the use of the classical Caputo derivative in the present work.

Motivated by these considerations, the present study adopts a fractional order modeling approach to investigate the progression of type 2 diabetes. The objective is to develop a mathematical model that captures both the biological and behavioral aspects of the disease while accounting for its long-term cumulative nature. This approach can provide deeper insight into the disease dynamics and may support more effective prevention and management strategies.

Beyond its clinical impact, diabetes mellitus is a major challenge for long-term public health planning. Its complications are progressive and often irreversible, and the effects of interventions can be delayed. The disease is also strongly influenced by lifestyle, prevention efforts, and healthcare policy. International reports indicate that diabetes rates have increased steadily in the past few decades, particularly in developing countries, placing significant pressure on healthcare systems and national economies [1,16].

In Operations Research, diabetes progression is seen as a dynamic decision-making problem. Health authorities must allocate limited resources to prevention and treatment over the long term, accounting for biological limits, social and economic factors, and the delayed impact of interventions. Mathematical models support these decisions by enabling the evaluation of long-term outcomes and identifying points at which the disease burden may become difficult to control [1,17,18].

Managing diabetes involves both non-drug and drug treatments at different stages of the disease. Lifestyle changes, such as diet and exercise, are important in improving insulin sensitivity. Medications such as metformin and insulin are needed later. Because these treatments differ in timing, cost, and effectiveness, there is a need for mathematical models that can capture long-term changes and delayed effects.

To address these issues, we introduce a fractional order compartmental model to describe the progression of diabetes across different metabolic stages. The model has four groups: susceptible people, those who are insulin-resistant, those with diabetes but without complications, and those with complications. Using a fractional approach, the model includes memory effects, making it more flexible and realistic.

Nonlinear time-fractional partial differential equations (NTFPDEs) play a significant role in modeling complex phenomena within science and engineering. Due to their nonlinear and fractional characteristics, these equations are challenging to solve analytically. To overcome these difficulties, the Hybrid Laplace Adomian Variational Iteration Method (HLAVIM) has been introduced by integrating the Laplace Adomian Decomposition Method (LADM) and the Laplace Variational Iteration Method (LVIM). The HLAVIM has been evaluated using several test cases, demonstrating strong agreement with exact solutions and greater precision than the existing methods. In addition, the method exhibits rapid convergence, typically requiring only a few iterations. These attributes establish HLAVIM as an effective and efficient approach to solving NTFPDEs [19].

A recent study presents a crossover optimal control system for a time-delayed fractional order diabetes model. The proposed approach integrates variable order and fractional order operators, employing numerical schemes based on nonstandard finite differences and the Grünwald–Letnikov method. The findings indicate that this system effectively regulates blood glucose levels and outperforms existing methodologies [17]. Sadri et al. [20] introduced a matrix-based collocation method employing sixth-kind Chebyshev polynomials (SKCPs) to analyze a fractional order glaucoma model. The findings demonstrate that this approach yields efficient numerical approximations and provides theoretical assurances regarding the existence, uniqueness, and Ulam–Hyers (U-H) stability. Additionally, sensitivity analysis, feedback control, and error estimates further substantiate the accuracy and convergence of the method.

Unlike the classical integer-order model described in [1], this study adopts a fractional order model. This method accounts for memory effects, which are essential for modeling the progression of chronic diseases such as diabetes. Since diabetes develops through continuous physiological and metabolic changes, fractional order models more accurately capture both memory and hereditary characteristics in model behavior.

This research makes several key contributions. First, we construct a fractional order dynamical model and establish its main mathematical properties, including the existence, uniqueness, stability, positivity, and boundedness of solutions. Next, we study the model's equilibrium points and examine both local and global stability, including healthy and chronic states. We also provide analytical and numerical solutions using the ADM and its LADM, and examine how the fractional order parameter affects the system. Finally, our simulations show that memory effects can slow disease progression and influence long-term trends.

Recent research has used fractional order modeling to study diabetes and related biomedical systems in several ways. Some studies have examined how glucose and insulin interact, focusing on delayed responses, oscillations, bifurcations, chaotic dynamics, feedback control, and stabilization methods using fixed, variable, and fractional-fractal order models [13,20–22]. Other work has focused on advanced numerical methods for fractional compartmental disease models, such as collocation-based techniques, and has demonstrated properties such as existence, uniqueness, Ulam–Hyers stability, and convergence [23]. More recently, researchers have introduced piecewise stochastic–deterministic and variable order fractional models to capture random changes, diverse dynamics, and personalized treatment responses in the progression of diabetes [12]. While

these studies have made important contributions, most focus on metabolic regulation, control analysis, numerical methods, or stochastic treatment approaches.

In contrast, this study introduces a dimensionally consistent Caputo fractional-derivative model for diabetes progression that employs effective fractional transition rates. It includes diabetic complications through a four-compartment structure, examines threshold-driven progression using local and global stability analysis, and explores memory effects with ADM and LADM. Instead of focusing on chaotic regulation or on treatment-stabilized glucose and insulin control, the present work examines long-term disease progression and complication dynamics, offering a perspective distinct from previous fractional diabetes modeling studies.

The remainder of this paper is organized as follows. Section 2 introduces the necessary preliminaries. Section 3 presents the model formulation and its qualitative analysis, including equilibrium and stability results. Section 4 describes the numerical methods, while Section 5 discusses existence, uniqueness, and convergence. The Ulam–Hyers stability is studied in Section 6. The numerical results are given in Section 7. Finally, the conclusions and future work are presented in Sections 8 and 9, respectively.

2. Definitions and Preliminaries

This section provides essential definitions and concepts of fractional calculus needed to formulate and analyze the proposed model [16,24–26].

Definition 1 ([24,25]). *The fractional integral of order $\mu > 0$ for a function $\mathbf{X}(t) \in L^1([0,T],\mathbb{R})$ is defined as*

$$ {}^{RL}\mathcal{I}_t^{\mu}\mathbf{X}(t) = \frac{1}{\Gamma(\mu)}\int_0^t (t-s)^{\mu-1}\mathbf{X}(s)\,ds, \tag{1}$$

provided that the integral on the right-hand side converges.

Definition 2 ([24,25]). *The Caputo derivative of order $\mu > 0$ for a function $\mathbf{X}(t) \in C^n([a,b])$, where $n = \lfloor \mu \rfloor + 1$, is defined as*

$$ {}^{C}D_t^{\mu}\mathbf{X}(t) = \frac{1}{\Gamma(n-\mu)}\int_0^t (t-s)^{n-\mu-1}\mathbf{X}^{(n)}(s)\,ds, \tag{2}$$

where $n-1 < \mu \leq n$, and $\lfloor \mu \rfloor$ denotes the integer part of μ.

Lemma 1 ([24,25]). *Let $\mathbf{F} \in C([0,T],\mathbb{R})$ and let $n-1 < \mu \leq n$, where $n \in \mathbb{N}$. Consider the Caputo fractional differential equation (FDE)*

$$ {}^{C}D_t^{\mu}\mathbf{X}(t) = \mathbf{F}(t), \qquad t \in [0,T]. \tag{3}$$

Then the general solution of FDE (3) is given by

$$ \mathbf{X}(t) = \sum_{k=0}^{n-1} c_k t^k + \frac{1}{\Gamma(\mu)}\int_0^t (t-s)^{\mu-1}\mathbf{F}(s)ds, \tag{4}$$

where $c_k \in \mathbb{R}$, for $k = 0, 1, \ldots, n-1$, are constants determined by the initial conditions.

In this work, the term singular-kernel operator refers to the Caputo derivative, whose kernel $(t-s)^{-\mu}$ is singular at $t = s$. Thus, the model is developed using the Caputo derivative, interpreted as a singular-kernel fractional operator.

Definition 3 ([24,25]). *The Adomian Decomposition Method (ADM) is useful for solving complex non-linear ODEs/PDEs of integer order.*

In ADM, nonlinear terms are handled with Adomian polynomials (AP), denoted by $A_0, A_1, \ldots, A_m$. *The solution* $\mathbf{X}(t)$ *is expressed as an infinite series of the form.*

$$\mathbf{X}(t) = \sum_{m=0}^{\infty} \mathbf{X}_m. \tag{5}$$

Each component y_m *can be determined recursively and is associated with the corresponding AP* A_m. *APs are defined by*

$$A_m = \frac{1}{m!}\frac{d^m}{d\lambda^m}\left[G(t)\sum_{j=0}^{\infty}\mathbf{X}_j\lambda^j\right]_{\lambda=0}. \tag{6}$$

Lemma 2 ([24,25]). *The following property is valid for fractional differential operators:*

$${}^{RL}\mathcal{I}_t^{\mu}\left[{}^{C}D_t^{\mu}\mathbf{X}(t)\right] = \mathbf{X}(t) - \sum_{j=0}^{n-1}\frac{\mathbf{X}^{(j)}(0)}{j!}t^j, \quad \mu > 0, \tag{7}$$

where $n = \lfloor \mu \rfloor + 1$, *and* $\lfloor \mu \rfloor$ *denotes the integer part of* μ.

Definition 4 ([26]). *The Laplace transform of the Caputo derivative is defined as follows:*

$$\mathcal{L}\left\{{}^{C}D_t^{\mu}\mathbf{X}(t)\right\} = s^{\mu}\mathbf{X}(s) - \sum_{k=0}^{n-1}s^{\mu-k-1}\mathbf{X}^{(k)}(0), \tag{8}$$

where $n-1 < \mu \leq n$.

Remark 1. *To incorporate memory effects associated with long-term metabolic deterioration and cumulative exposure to risk factors, we extend the classical model by replacing the first-order time derivative with the Caputo derivative of order* μ, *with* $0 < \mu < 1$.

Theorem 1 ([27]). *Let* $(\mathcal{B}, \|\cdot\|)$ *be a Banach space, and let* $\mathcal{T} : \mathcal{B} \to \mathcal{B}$ *be a contraction. Then* $\mathcal{T}$ *has a unique fixed point, meaning there exists a single* $\mathbf{X} \in \mathcal{B}$ *such that*

$$\mathcal{T}(\mathbf{X}) = \mathbf{X}.$$

Now, we introduce the concept of Ulam–Hyers (U-H) stability for fractional differential equations (FDEs). Consider the general FDE

$$\mathcal{Q}\left(q(t), \mathbf{X}(t), {}^{C}D_t^{\mu_1}\mathbf{X}(t), \ldots, {}^{C}D_t^{\mu_n}\mathbf{X}(t)\right) = 0, \quad \forall t \in [a,b], \quad -\infty < a < b < +\infty, \tag{9}$$

where $\mathbf{X} : [a,b] \to \mathcal{B}$, with $(\mathcal{B}, |\cdot|_{\mathcal{B}})$ being a normed linear space, ${}^{C}D_t^{\mu_i}$, $i = 1, \ldots, n$, denote fractional order differential operators satisfying $0 < \mu_1 \leq \cdots \leq \mu_n$, and $q : [a,b] \to \mathcal{B}$ is a given function. Given $\varepsilon > 0$ and $\varphi : [a,b] \to \mathbb{R}_+$, suppose that $\mathbf{Y} : [a,b] \to \mathcal{B}$ satisfies one of the following inequalities:

$$\left|\mathcal{Q}\left(q(t), \mathbf{Y}(t), {}^{C}D_t^{\mu_1}\mathbf{Y}(t), \ldots, {}^{C}D_t^{\mu_n}\mathbf{Y}(t)\right)\right| \leq \varepsilon, \quad \forall t \in [a,b]. \tag{10}$$

$$\left|\mathcal{Q}\left(q(t), \mathbf{Y}(t), {}^{C}D_t^{\mu_1}\mathbf{Y}(t), \ldots, {}^{C}D_t^{\mu_n}\mathbf{Y}(t)\right)\right| \leq \varphi(t), \quad \forall t \in [a,b]. \tag{11}$$

The following concepts are introduced to evaluate the model's robustness to perturbations and ensure numerical solution consistency.

Definition 5 ([27]). *System* (9) *is said to be U-H stable if there exists a constant* $\sigma > 0$ *such that for every* $\varepsilon > 0$*, and for every solution* $\mathbf{Y}$ *satisfying* (10)*, there exists an exact solution* $\mathbf{X}$ *of* (9) *such that*

$$|\mathbf{Y}(t) - \mathbf{X}(t)| \leq \sigma\varepsilon, \quad \forall t \in [a, b]. \tag{12}$$

The constant σ *is called the U-H constant.*

Definition 6 ([27]). *System* (9) *is said to be Ulam–Hyers–Rassias (U-H-R) stable if there exists a constant* $\sigma > 0$ *such that for every solution* $\mathbf{Y}$ *satisfying* (11)*, there exists an exact solution* $\mathbf{X}$ *of* (9) *such that*

$$|\mathbf{Y}(t) - \mathbf{X}(t)| \leq \sigma\varphi(t), \quad \forall t \in [a, b]. \tag{13}$$

$$\left|\mathcal{Q}\left(q(t), \mathbf{Y}(t), D^{\mu_1}\mathbf{Y}(t), \ldots, D^{\mu_n}\mathbf{Y}(t)\right)\right| \leq \chi(t), \quad \forall t \in [a, b]. \tag{14}$$

Equation (10) corresponds to the U-H perturbation condition, while (14) represents the generalized U-H-R perturbation condition.

3. Singular Kernel Modeling of Type 2 Diabetes

This section formulates a fractional order model using a singular-kernel compartmental model for diabetes progression, introduces a singular-kernel operator, and provides a detailed description of its structure.

Using a Caputo derivative of order $0 < \mu \leq 1$, the model [1] can be written as a fractional order model

$$\begin{cases} {}^{C}D_t^{\mu} S(t) = bS(t)\left(1 - \dfrac{S(t)}{K}\right) - (\alpha + r)S(t), \\ {}^{C}D_t^{\mu} I(t) = \alpha S(t) - (\beta + r)I(t), \\ {}^{C}D_t^{\mu} D(t) = \beta I(t) - (\gamma + r)D(t), \\ {}^{C}D_t^{\mu} C(t) = \gamma D(t) - (r + r_1)C(t). \end{cases} \tag{15}$$

with initial conditions (ICs)

$$S(0) = S_0, \qquad I(0) = I_0, \qquad D(0) = D_0, \qquad C(0) = C_0. \tag{16}$$

In the classical integer-order system, the right-hand sides of the equations have dimension $(\text{time})^{-1}$, since all the transition and mortality parameters represent the rates per unit of time. However, when the first order derivative is replaced by the Caputo derivative of order $0 < \mu < 1$, the dimension of the left-hand side becomes $(\text{time})^{-\mu}$. Therefore, to preserve the homogeneity of dimensions, all rate parameters must be modified so that their dimensions change from $(\text{time})^{-1}$ to $(\text{time})^{-\mu}$ [11,28,29]. Following the approach commonly used in the fractional modeling literature, the parameters are redefined as effective fractional rates incorporating memory effects. Consequently, the dimensionally consistent fractional metabolic model is written as

$$\begin{cases} {}^{C}D_t^{\mu} S(t) = b^{\mu} S(t)\left(1 - \dfrac{S(t)}{K}\right) - (\alpha^{\mu} + r^{\mu})S(t), \\ {}^{C}D_t^{\mu} I(t) = \alpha^{\mu} S(t) - (\beta^{\mu} + r^{\mu})I(t), \\ {}^{C}D_t^{\mu} D(t) = \beta^{\mu} I(t) - (\gamma^{\mu} + r^{\mu})D(t), \\ {}^{C}D_t^{\mu} C(t) = \gamma^{\mu} D(t) - (r^{\mu} + r_1^{\mu})C(t), \end{cases} \tag{17}$$

subject to the ICs

$$S(0) = S_0, \qquad I(0) = I_0, \qquad D(0) = D_0, \qquad C(0) = C_0.$$

Here, the parameters b^μ, α^μ, β^μ, γ^μ, r^μ, and r_1^μ represent effective fractional transition and mortality rates that capture long-term memory effects in metabolic progression dynamics.

Dimensional consistency in fractional differential models can be achieved through various approaches in the literature. In the present work, we adopt the common approach of redefining the model parameters as effective fractional rates, with units adjusted from $(\text{time})^{-1}$ to $(\text{time})^{-\mu}$, which leads to parameters such as $b^\mu, \alpha^\mu, \beta^\mu$, and γ^μ. Another approach introduces a characteristic time-scale factor $\tau^{\mu-1}$ that multiplies the fractional derivative to balance dimensions after replacing the classical derivative with a fractional operator [30,31]. Both approaches preserve dimensional homogeneity. In this study, however, the effective fractional rate formulation is preferred because it also provides a meaningful interpretation of memory effects in disease progression. Specifically, the parameters $b^\mu, \alpha^\mu, \beta^\mu$, and γ^μ can be viewed as transition rates modified by accumulated metabolic history, reflecting that progression between compartments depends not only on the current state but also on hereditary and memory-dependent effects.

The diagram Figure 1 shows the transitions between compartments and their corresponding rates [1] .

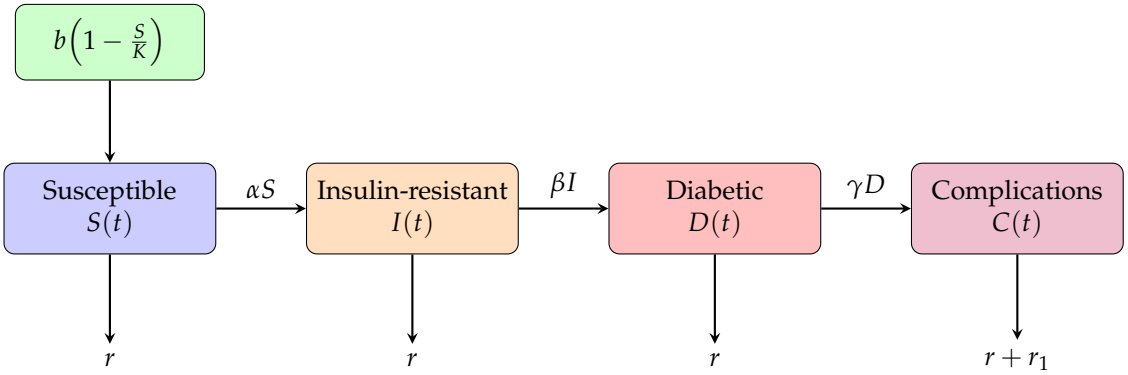


Figure 1. Schematic diagram of the proposed fractional order diabetes progression model.

3.1. Description of Variables and Parameters

For clarity, the state variables and model parameters of model (15) are summarized in Tables 1 and 2.

Table 1. State variables of model (15).

Variable	Description
$S(t)$	Population at metabolic risk
$I(t)$	Individuals with insulin resistance
$D(t)$	Diabetic individuals without complications
$C(t)$	Diabetic individuals with complications

Table 2. Model parameters of model (15).

Parameter	Description
b	Recruitment rate into the at-risk population
K	Carrying capacity
α	Transition rate from S to I
β	Transition rate from I to D
γ	Transition rate from D to C
r	Natural mortality rate
r_1	Excess mortality in C

Theorem 2. *Let* $0 < \mu \leq 1$ *and suppose that the initial data satisfy*

$$S_0 \geq 0, \qquad I_0 \geq 0, \qquad D_0 \geq 0, \qquad C_0 \geq 0.$$

Then the set

$$\Omega^0 = \left\{ (S, I, D, C) \in \mathbb{R}^4_+ : \ S \geq 0, \ I \geq 0, \ D \geq 0, \ C \geq 0 \right\}$$

is positively invariant for the system (17)*. In particular, the solution of the system* (17) *remains nonnegative for all* $t \geq 0$*.*

Proof. Consider the system (17) on the boundary of Ω^0. If $S = 0$, then

$${}^{C}D_t^{\mu} S(t) = 0.$$

If $I = 0$, then

$${}^{C}D_t^{\mu} I(t) = \alpha^{\mu} S(t) \geq 0.$$

If $D = 0$, then

$${}^{C}D_t^{\mu} D(t) = \beta^{\mu} I(t) \geq 0.$$

If $C = 0$, then

$${}^{C}D_t^{\mu} C(t) = \gamma^{\mu} D(t) \geq 0.$$

Therefore, each component of the fractional vector field is nonnegative on the corresponding boundary of Ω^0. It follows from the comparison principle for Caputo fractional order systems (17) that solutions that start in Ω^0 cannot leave Ω^0. Thus, Ω^0 is positively invariant, which completes the proof. For related details on the comparison principle, see [32]. □

3.2. Equilibrium Points and Asymptotic Stability

This subsection determines equilibrium points and analyzes their asymptotic stability.

To determine the equilibrium points of the Caputo fractional order metabolic progression system (17), we set the right-hand sides of the equations to zero, that is

$${}^{C}D_t^{\mu} S(t) = {}^{C}D_t^{\mu} I(t) = {}^{C}D_t^{\mu} D(t) = {}^{C}D_t^{\mu} C(t) = 0.$$

By solving the resulting algebraic system, we obtain the trivial metabolic equilibrium

$$E^0 = (0, 0, 0, 0),$$

which corresponds to the absence of individuals at all stages of metabolic progression. In addition, the system admits a biologically meaningful metabolic progression equilibrium $E^* = (S^*, I^*, D^*, C^*)$, where

$$\begin{aligned} S^* &= K\left(1 - \frac{\alpha^{\mu} + r^{\mu}}{b^{\mu}}\right), \\ I^* &= \frac{\alpha^{\mu}}{\beta^{\mu} + r^{\mu}} S^*, \\ D^* &= \frac{\beta^{\mu}}{\gamma^{\mu} + r^{\mu}} I^*, \\ C^* &= \frac{\gamma^{\mu}}{r^{\mu} + r_1^{\mu}} D^*. \end{aligned} \tag{18}$$

This equilibrium exists provided that

$$b^{\mu} > \alpha^{\mu} + r^{\mu}.$$

To investigate the local stability of the equilibrium points, we consider the Caputo fractional order linear system (17)

$$^{C}D_{t}^{\mu}\mathbf{X}(t) = J\mathbf{X}(t), \tag{19}$$

where J denotes the Jacobian matrix evaluated at equilibrium. According to fractional stability theory, an equilibrium point is locally asymptotically stable if all eigenvalues λ_i of the Jacobian matrix satisfy

$$|\arg(\lambda_i)| > \frac{\mu\pi}{2}.$$

3.3. Global Stability Analysis

In this subsection, the model's global stability properties are investigated to understand its long-term behavior. We investigate the global asymptotic dynamics of the model (17). The model describes a sequential metabolic progression associated with type 2 diabetes; therefore, the trivial equilibrium corresponds to the absence of sustained metabolic activation, rather than to demographic extinction.

The feasible region is

$$\Omega = \{(S, I, D, C) \in \mathbb{R}_{+}^{4}\},$$

with interior

$$\Omega^{\circ} = \{(S, I, D, C) \in \mathbb{R}_{+}^{4} : \ S, I, D, C > 0\}.$$

Proposition 1. *The set Ω is positively invariant for system* (17).

Proof. Evaluating the vector field of the system (17) on the boundary hyperplanes of Ω gives

$$^{C}D_{t}^{\mu}S\big|_{S=0} = 0, \qquad ^{C}D_{t}^{\mu}I\big|_{I=0} = \alpha^{\mu}S \geq 0,$$
$$^{C}D_{t}^{\mu}D\big|_{D=0} = \beta^{\mu}I \geq 0, \qquad ^{C}D_{t}^{\mu}C\big|_{C=0} = \gamma^{\mu}D \geq 0.$$

Hence, trajectories cannot leave Ω through any coordinate hyperplane. Therefore, Ω is positively invariant. □

3.3.1. Global Stability of the Trivial Equilibrium

We establish the global stability of the trivial equilibrium, treated here as a boundary equilibrium, under specified conditions [32].

Model (17) admits a trivial equilibrium

$$E_0 = (0, 0, 0, 0)$$

which is considered a boundary equilibrium in the model. It does not represent a biologically realistic healthy population state; rather, it reflects the absence of sustained metabolic progression. This equilibrium is included mainly for mathematical completeness and to help identify the threshold between non-persistent and persistent disease progression.

The first equation can be written as

$$^{C}D_{t}^{\mu}S = S\left[(b^{\mu} - \alpha^{\mu} - r^{\mu}) - \frac{b^{\mu}}{K}S\right]. \tag{20}$$

If

$$b^{\mu} < \alpha^{\mu} + r^{\mu}, \tag{21}$$

then

$$^{C}D_{t}^{\mu}S \leq -(\alpha^{\mu} + r^{\mu} - b^{\mu})S. \tag{22}$$

By the fractional comparison principle,

$$0 \leq S(t) \leq S(0)E_{\mu}(-(\alpha^{\mu} + r^{\mu} - b^{\mu})t^{\mu}), \tag{23}$$

and therefore

$$S(t) \to 0.$$

Due to the triangular structure of the system, substituting $S(t) \to 0$ into the remaining equations yields linear Caputo fractional order systems with asymptotically vanishing forcing terms.

$$^{C}D_{t}^{\mu} I = \alpha^{\mu} S - (\beta^{\mu} + r^{\mu})I,$$
$$^{C}D_{t}^{\mu} D = \beta^{\mu} I - (\gamma^{\mu} + r^{\mu})D,$$
$$^{C}D_{t}^{\mu} C = \gamma^{\mu} D - (r^{\mu} + r_{1}^{\mu})C.$$

M-L asymptotic stability of the homogeneous equations, together with the variation-of-constants formula, implies

$$I(t) \to 0, \qquad D(t) \to 0, \qquad C(t) \to 0.$$

Theorem 3. *If*

$$b^{\mu} < \alpha^{\mu} + r^{\mu},$$

then the equilibrium E_0 is globally asymptotically stable in Ω.

3.3.2. Global Stability of the Chronic Progression Equilibrium

This subsection examines the global stability of the chronic progression equilibrium and discusses its impact on disease dynamics.

If

$$b^{\mu} > \alpha^{\mu} + r^{\mu}, \tag{24}$$

system (17) admits the positive equilibrium

$$E^{*} = (S^{*}, I^{*}, D^{*}, C^{*}), \tag{25}$$

where

$$S^{*} = \frac{K(b^{\mu} - \alpha^{\mu} - r^{\mu})}{b^{\mu}}, \quad I^{*} = \frac{\alpha^{\mu} S^{*}}{\beta^{\mu} + r^{\mu}}, \quad D^{*} = \frac{\beta^{\mu} I^{*}}{\gamma^{\mu} + r^{\mu}}, \quad C^{*} = \frac{\gamma^{\mu} D^{*}}{r^{\mu} + r_{1}^{\mu}}. \tag{26}$$

The scalar equation for S is a fractional logistic equation with a unique positive equilibrium S^{*}. Fractional comparison arguments, together with the boundedness of solutions and scalar Caputo logistic stability results, imply

$$S(t) \to S^{*} \qquad \text{for all } S(0) > 0.$$

Define

$$e_{I} = I - I^{*}, \qquad e_{D} = D - D^{*}, \qquad e_{C} = C - C^{*}.$$

Using equilibrium identities, we obtain the triangular linear system.

$$^{C}D_{t}^{\mu} e_{I} = \alpha^{\mu}(S - S^{*}) - (\beta^{\mu} + r^{\mu})e_{I},$$
$$^{C}D_{t}^{\mu} e_{D} = \beta^{\mu} e_{I} - (\gamma^{\mu} + r^{\mu})e_{D},$$
$$^{C}D_{t}^{\mu} e_{C} = \gamma^{\mu} e_{D} - (r^{\mu} + r_{1}^{\mu})e_{C}.$$

Since $S(t) \to S^*$, all forcing terms vanish asymptotically. The M-L asymptotic stability of the homogeneous equations, together with the variation-of-constants formula, yields

$$e_I(t) \to 0, \qquad e_D(t) \to 0, \qquad e_C(t) \to 0.$$

Theorem 4. *If*

$$b^\mu > \alpha^\mu + r^\mu, \tag{27}$$

then the equilibrium E^* is globally asymptotically stable in Ω°.

3.3.3. Threshold Dynamics and Metabolic Interpretation

We analyze the system's overall metabolic dynamics to identify transitions between disease stages. System (17) exhibits a threshold-driven transition between two dynamical regimes:

- If $b^\mu < \alpha^\mu + r^\mu$, metabolic activation cannot sustain progression, and the population converges to a metabolically healthy equilibrium.
- If $b^\mu > \alpha^\mu + r^\mu$, metabolic progression persists and stabilizes in a chronic steady state.

Hence,

$$b^\mu - (\alpha^\mu + r^\mu)$$

acts as a metabolic persistence threshold that governs long-term disease dynamics.

The threshold quantity $b^\mu - (\alpha^\mu + r^\mu)$ arises from the balance between effective growth and losses due to disease progression and removal. It is consistent with the threshold interpretation [33] in the underlying model [1]. In the fractional order, it acts as a persistence condition: when $b^\mu > \alpha^\mu + r^\mu$, progression can be sustained, whereas the opposite condition drives the system toward the boundary equilibrium. Although it is not presented as a classical reproduction number, it provides a biologically meaningful threshold for persistence in the proposed fractional order model (17).

3.4. Local Stability Analysis

In this subsection, we study the local asymptotic stability of the equilibrium points of the dimensionally consistent fractional model (17). For a commensurate fractional order system with a singular kernel

$${}^{C}D_t^\mu \mathbf{X}(t) = \mathbf{F}(\mathbf{X}(t)), \qquad 0 < \mu \leq 1,$$

the local asymptotic stability of an equilibrium point E^* is determined by the well-known fractional stability criterion: E^* is locally asymptotically stable if all the eigenvalues λ_i of the Jacobian matrix $J(E^*)$ satisfy

$$|\arg(\lambda_i)| > \frac{\mu\pi}{2}, \qquad i = 1, 2, \ldots, n. \tag{28}$$

For model (17), the vector field is given by

$$\mathbf{F}(S, I, D, C) = \begin{pmatrix} b^\mu S\left(1 - \dfrac{S}{K}\right) - (\alpha^\mu + r^\mu)S \\ \alpha^\mu S - (\beta^\mu + r^\mu)I \\ \beta^\mu I - (\gamma^\mu + r^\mu)D \\ \gamma^\mu D - (r^\mu + r_1^\mu)C \end{pmatrix}. \tag{29}$$

Hence, the Jacobian matrix is

$$J(S,I,D,C) = \begin{pmatrix} b^{\mu} - \dfrac{2b^{\mu}S}{K} - (\alpha^{\mu} + r^{\mu}) & 0 & 0 & 0 \\ \alpha^{\mu} & -(\beta^{\mu} + r^{\mu}) & 0 & 0 \\ 0 & \beta^{\mu} & -(\gamma^{\mu} + r^{\mu}) & 0 \\ 0 & 0 & \gamma^{\mu} & -(r^{\mu} + r_1^{\mu}) \end{pmatrix}. \quad (30)$$

3.4.1. Trivial and Positive Equilibria

The equilibrium points of the model are determined by solving model (17) for steady-state solutions, where model (17) remains unchanged over time.

$$\mathbf{F}(S,I,D,C) = 0. \quad (31)$$

From the first equation,

$$b^{\mu}S\left(1 - \frac{S}{K}\right) - (\alpha^{\mu} + r^{\mu})S = 0,$$

which gives

$$S\left[(b^{\mu} - \alpha^{\mu} - r^{\mu}) - \frac{b^{\mu}}{K}S\right] = 0.$$

Therefore,

$$S = 0 \quad \text{or} \quad S^* = \frac{K(b^{\mu} - \alpha^{\mu} - r^{\mu})}{b^{\mu}}.$$

(i) Trivial Equilibrium

The trivial equilibrium is

$$E_0 = (0,0,0,0). \quad (32)$$

(ii) Positive Equilibrium

If $b^{\mu} > \alpha^{\mu} + r^{\mu}$, then the positive equilibrium exists and is given by

$$E^* = (S^*, I^*, D^*, C^*), \quad (33)$$

where

$$S^* = \frac{K(b^{\mu} - \alpha^{\mu} - r^{\mu})}{b^{\mu}}, \qquad I^* = \frac{\alpha^{\mu}S^*}{\beta^{\mu} + r^{\mu}}, \qquad D^* = \frac{\beta^{\mu}I^*}{\gamma^{\mu} + r^{\mu}}, \qquad C^* = \frac{\gamma^{\mu}D^*}{r^{\mu} + r_1^{\mu}}. \quad (34)$$

3.4.2. Stability of the Trivial Equilibrium

The stability of the trivial equilibrium is analyzed by evaluating the Jacobian matrix and its eigenvalues for model (17).

Evaluating the Jacobian at E_0, we obtain

$$J(E_0) = \begin{pmatrix} b^{\mu} - \alpha^{\mu} - r^{\mu} & 0 & 0 & 0 \\ \alpha^{\mu} & -(\beta^{\mu} + r^{\mu}) & 0 & 0 \\ 0 & \beta^{\mu} & -(\gamma^{\mu} + r^{\mu}) & 0 \\ 0 & 0 & \gamma^{\mu} & -(r^{\mu} + r_1^{\mu}) \end{pmatrix}. \quad (35)$$

Since this matrix is lower triangular, its eigenvalues are

$$\lambda_1 = b^{\mu} - \alpha^{\mu} - r^{\mu}, \qquad \lambda_2 = -(\beta^{\mu} + r^{\mu}), \qquad \lambda_3 = -(\gamma^{\mu} + r^{\mu}), \qquad \lambda_4 = -(r^{\mu} + r_1^{\mu}). \quad (36)$$

Clearly, $\lambda_2, \lambda_3, \lambda_4 < 0$. Therefore, by (28), the equilibrium E_0 is locally asymptotically stable if and only if

$$b^\mu - \alpha^\mu - r^\mu < 0,$$

that is,

$$b^\mu < \alpha^\mu + r^\mu. \tag{37}$$

Thus, the trivial equilibrium E_0 is locally asymptotically stable whenever $b^\mu < \alpha^\mu + r^\mu$.

3.4.3. Stability of the Positive Equilibrium

This subsection investigates the stability of the positive equilibrium under biologically relevant conditions for model (17).

Now, evaluating the Jacobian at E^* gives

$$J(E^*) = \begin{pmatrix} b^\mu - \dfrac{2b^\mu S^*}{K} - (\alpha^\mu + r^\mu) & 0 & 0 & 0 \\ \alpha^\mu & -(\beta^\mu + r^\mu) & 0 & 0 \\ 0 & \beta^\mu & -(\gamma^\mu + r^\mu) & 0 \\ 0 & 0 & \gamma^\mu & -(r^\mu + r_1^\mu) \end{pmatrix}. \tag{38}$$

Again, since the matrix is lower triangular, its eigenvalues are

$$\lambda_1 = b^\mu - \frac{2b^\mu S^*}{K} - (\alpha^\mu + r^\mu), \qquad \lambda_2 = -(\beta^\mu + r^\mu), \qquad \lambda_3 = -(\gamma^\mu + r^\mu), \qquad \lambda_4 = -(r^\mu + r_1^\mu).$$

Substituting

$$S^* = \frac{K(b^\mu - \alpha^\mu - r^\mu)}{b^\mu},$$

we obtain

$$\lambda_1 = b^\mu - 2(b^\mu - \alpha^\mu - r^\mu) - (\alpha^\mu + r^\mu) = -(b^\mu - \alpha^\mu - r^\mu).$$

Hence, for $b^\mu > \alpha^\mu + r^\mu$, one has

$$\lambda_1 < 0, \qquad \lambda_2 < 0, \qquad \lambda_3 < 0, \qquad \lambda_4 < 0.$$

Therefore, all eigenvalues satisfy

$$|\arg(\lambda_i)| = \pi > \frac{\mu\pi}{2}, \qquad 0 < \mu \leq 1.$$

By the fractional stability criterion, the positive equilibrium E^* is locally asymptotically stable whenever it exists, that is, for

$$b^\mu > \alpha^\mu + r^\mu. \tag{39}$$

Theorem 5. *For model (17), the trivial equilibrium (32), we have*

$$E_0 = (0, 0, 0, 0)$$

is locally asymptotically stable if

$$b^\mu < \alpha^\mu + r^\mu.$$

Moreover, if

$$b^\mu > \alpha^\mu + r^\mu,$$

then the positive equilibrium

$$E^* = \left(\frac{K(b^\mu - \alpha^\mu - r^\mu)}{b^\mu}, \frac{\alpha^\mu S^*}{\beta^\mu + r^\mu}, \frac{\beta^\mu I^*}{\gamma^\mu + r^\mu}, \frac{\gamma^\mu D^*}{r^\mu + r_1^\mu} \right) \tag{40}$$

exists and is locally asymptotically stable.

Proof. The proof follows from the Jacobian matrix evaluated at the corresponding equilibrium point and the fractional stability condition

$$|\arg(\lambda_i)| > \frac{\mu\pi}{2}, \qquad 0 < \mu \leq 1. \tag{41}$$

Since the Jacobian matrix is lower triangular, its eigenvalues are its diagonal entries. At E_0, the eigenvalues are

$$\lambda_1 = b^\mu - \alpha^\mu - r^\mu, \quad \lambda_2 = -(\beta^\mu + r^\mu), \quad \lambda_3 = -(\gamma^\mu + r^\mu), \quad \lambda_4 = -(r^\mu + r_1^\mu).$$

Hence, E_0 is locally asymptotically stable if and only if $b^\mu < \alpha^\mu + r^\mu$. At the positive equilibrium E^*, the eigenvalues are

$$\lambda_1 = -(b^\mu - \alpha^\mu - r^\mu), \quad \lambda_2 = -(\beta^\mu + r^\mu), \quad \lambda_3 = -(\gamma^\mu + r^\mu), \quad \lambda_4 = -(r^\mu + r_1^\mu).$$

Thus, whenever $b^\mu > \alpha^\mu + r^\mu$, all eigenvalues are negative real numbers and satisfy

$$|\arg(\lambda_i)| = \pi > \frac{\mu\pi}{2}. \tag{42}$$

Consequently, the equilibrium E^* exhibits local asymptotic stability for model (17). □

4. Numerical Methods

This section outlines numerical methods for approximating solutions to the proposed singular kernel model (17).

4.1. Adomian Decomposition Method (ADM)

In this section, we construct a semi-analytical solution of model (17) using the ADM. Let

$$\mathcal{J} = {}^{C}D_t^\mu, \qquad 0 < \mu \leq 1$$

denote the Caputo fractional differential operator. Then model (15) can be written in a compact vector form as

$$\mathcal{J}\mathbf{X}(t) = \mathbf{F}(\mathbf{X}(t)), \tag{43}$$

where

$$\mathbf{X}(t) = (S(t), I(t), D(t), C(t))^T.$$

Applying the fractional integral operator

$$\mathcal{J}^{-1} = {}^{RL}\mathcal{I}_t^\mu$$

yields the equivalent nonlinear Volterra model (17)

$$\begin{aligned}
S(t) &= S_0 + {}^{RL}\mathcal{I}_t^\mu \left[b^\mu S - \frac{b^\mu}{K} S^2 - (\alpha^\mu + r^\mu) S \right], \\
I(t) &= I_0 + {}^{RL}\mathcal{I}_t^\mu \left[\alpha^\mu S - (\beta^\mu + r^\mu) I \right], \\
D(t) &= D_0 + {}^{RL}\mathcal{I}_t^\mu \left[\beta^\mu I - (\gamma^\mu + r^\mu) D \right], \\
C(t) &= C_0 + {}^{RL}\mathcal{I}_t^\mu \left[\gamma^\mu D - (r^\mu + r_1^\mu) C \right].
\end{aligned} \tag{44}$$

According to ADM, the solution components are expressed as an infinite series

$$\begin{aligned} S(t) &= \sum_{n=0}^{\infty} S_m(t), \\ I(t) &= \sum_{n=0}^{\infty} I_m(t), \\ D(t) &= \sum_{n=0}^{\infty} D_m(t), \\ C(t) &= \sum_{n=0}^{\infty} C_m(t). \end{aligned} \tag{45}$$

The nonlinear term is decomposed using the Adomian polynomials introduced in Definition 3.

$$S^2(t) = \sum_{m=0}^{\infty} A_m, \tag{46}$$

where

$$A_m = \sum_{k=0}^{m} S_k S_{m-k}. \tag{47}$$

Substituting the series expansions into the integral system (44) gives the initial terms

$$S_0(t) = S_0, \qquad I_0(t) = I_0, \qquad D_0(t) = D_0, \qquad C_0(t) = C_0, \tag{48}$$

and the recursive scheme for $n \geq 0$

$$\begin{aligned} S_{m+1}(t) &= {}^{RL}\mathcal{I}_t^{\mu}\left[b^{\mu} S_m - \frac{b^{\mu}}{K} A_n - (\alpha^{\mu} + r^{\mu}) S_m\right], \\ I_{m+1}(t) &= {}^{RL}\mathcal{I}_t^{\mu}\left[\alpha^{\mu} S_m - (\beta^{\mu} + r^{\mu}) I_m\right], \\ D_{m+1}(t) &= {}^{RL}\mathcal{I}_t^{\mu}\left[\beta^{\mu} I_m - (\gamma^{\mu} + r^{\mu}) D_m\right], \\ C_{m+1}(t) &= {}^{RL}\mathcal{I}_t^{\mu}\left[\gamma^{\mu} D_m - (r^{\mu} + r_1^{\mu}) C_m\right]. \end{aligned} \tag{49}$$

4.2. Laplace Adomian Decomposition Method (LADM)

In this section, we derive a semi-analytical solution for model (17) by means of LADM. Let $\mathcal{L}\{\cdot\}$ denote the Laplace transform operator. Taking the Laplace transform of model (17) and using Definition 2, we have

$$\mathcal{L}\left\{{}^{C}D_t^{\mu} f(t)\right\} = s^{\mu}\mathcal{L}\{f(t)\} - s^{\mu-1} f(0), \qquad 0 < \mu \leq 1. \tag{50}$$

Applying this to system (17), we obtain

$$\begin{aligned} s^{\mu}\mathcal{L}\{S(t)\} - s^{\mu-1} S_0 &= \mathcal{L}\left\{b^{\mu} S(t)\left(1 - \frac{S(t)}{K}\right) - (\alpha^{\mu} + r^{\mu}) S(t)\right\}, \\ s^{\mu}\mathcal{L}\{I(t)\} - s^{\mu-1} I_0 &= \mathcal{L}\{\alpha^{\mu} S(t) - (\beta^{\mu} + r^{\mu}) I(t)\}, \\ s^{\mu}\mathcal{L}\{D(t)\} - s^{\mu-1} D_0 &= \mathcal{L}\{\beta^{\mu} I(t) - (\gamma^{\mu} + r^{\mu}) D(t)\}, \\ s^{\mu}\mathcal{L}\{C(t)\} - s^{\mu-1} C_0 &= \mathcal{L}\left\{\gamma^{\mu} D(t) - (r^{\mu} + r_1^{\mu}) C(t)\right\}. \end{aligned} \tag{51}$$

Hence,

$$\begin{aligned}
\mathcal{L}\{S(t)\} &= \frac{S_0}{s} + \frac{1}{s^\mu}\mathcal{L}\left\{b^\mu S(t) - \frac{b^\mu}{K}S^2(t) - (\alpha^\mu + r^\mu)S(t)\right\},\\
\mathcal{L}\{I(t)\} &= \frac{I_0}{s} + \frac{1}{s^\mu}\mathcal{L}\{\alpha^\mu S(t) - (\beta^\mu + r^\mu)I(t)\},\\
\mathcal{L}\{D(t)\} &= \frac{D_0}{s} + \frac{1}{s^\mu}\mathcal{L}\{\beta^\mu I(t) - (\gamma^\mu + r^\mu)D(t)\},\\
\mathcal{L}\{C(t)\} &= \frac{C_0}{s} + \frac{1}{s^\mu}\mathcal{L}\left\{\gamma^\mu D(t) - (r^\mu + r_1^\mu)C(t)\right\}.
\end{aligned} \tag{52}$$

Applying the inverse Laplace transform to system (52), we obtain

$$\begin{aligned}
S(t) &= S_0 + \mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\left\{b^\mu S(t) - \frac{b^\mu}{K}S^2(t) - (\alpha^\mu + r^\mu)S(t)\right\}\right],\\
I(t) &= I_0 + \mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\{\alpha^\mu S(t) - (\beta^\mu + r^\mu)I(t)\}\right],\\
D(t) &= D_0 + \mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\{\beta^\mu I(t) - (\gamma^\mu + r^\mu)D(t)\}\right],\\
C(t) &= C_0 + \mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\left\{\gamma^\mu D(t) - (r^\mu + r_1^\mu)C(t)\right\}\right].
\end{aligned} \tag{53}$$

We now assume the series representations

$$S(t) = \sum_{n=0}^{\infty} S_m(t), \qquad I(t) = \sum_{n=0}^{\infty} I_m(t), \qquad D(t) = \sum_{n=0}^{\infty} D_m(t), \qquad C(t) = \sum_{n=0}^{\infty} C_m(t). \tag{54}$$

The nonlinear term is decomposed by means of the Adomian polynomials defined in Definition 3:

$$S^2(t) = \sum_{n=0}^{\infty} A_m, \tag{55}$$

where

$$A_m = \sum_{k=0}^{m} S_k S_{m-k}, \qquad n \geq 0. \tag{56}$$

Substituting these expansions into the transformed system (53) and equating the same terms yields the following

$$S_0(t) = S_0, \qquad I_0(t) = I_0, \qquad D_0(t) = D_0, \qquad C_0(t) = C_0, \tag{57}$$

and for $n \geq 0$,

$$\begin{aligned}
S_{m+1}(t) &= \mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\left\{b^\mu S_m(t) - \frac{b^\mu}{K}A_m - (\alpha^\mu + r^\mu)S_m(t)\right\}\right],\\
I_{m+1}(t) &= \mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\{\alpha^\mu S_m(t) - (\beta^\mu + r^\mu)I_m(t)\}\right],\\
D_{m+1}(t) &= \mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\{\beta^\mu I_m(t) - (\gamma^\mu + r^\mu)D_m(t)\}\right],\\
C_{m+1}(t) &= \mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\left\{\gamma^\mu D_m(t) - (r^\mu + r_1^\mu)C_m(t)\right\}\right].
\end{aligned} \tag{58}$$

The first Adomian polynomial is

$$A_0 = S_0^2. \tag{59}$$

Therefore, the first-order components computed from the recursion formula (58) are

$$S_1(t) = \left[b^\mu S_0 - \frac{b^\mu}{K} S_0^2 - (\alpha^\mu + r^\mu) S_0\right] \frac{t^\mu}{\Gamma(\mu+1)}, \tag{60}$$

$$I_1(t) = [\alpha^\mu S_0 - (\beta^\mu + r^\mu) I_0] \frac{t^\mu}{\Gamma(\mu+1)}, \tag{61}$$

$$D_1(t) = [\beta^\mu I_0 - (\gamma^\mu + r^\mu) D_0] \frac{t^\mu}{\Gamma(\mu+1)}, \tag{62}$$

$$C_1(t) = \left[\gamma^\mu D_0 - (r^\mu + r_1^\mu) C_0\right] \frac{t^\mu}{\Gamma(\mu+1)}. \tag{63}$$

The second Adomian polynomial is

$$A_1 = 2 S_0 S_1. \tag{64}$$

Higher-order terms are then obtained recursively using the above iteration scheme (58).

Consequently, the N-term LADM approximation of model (15) is given by

$$S(t) \approx \sum_{m=0}^{N} S_m(t), \qquad I(t) \approx \sum_{m=0}^{N} I_m(t), \qquad D(t) \approx \sum_{m=0}^{N} D_m(t), \qquad C(t) \approx \sum_{m=0}^{N} C_m(t). \tag{65}$$

To establish the existence and uniqueness of solutions, convergence analysis, and Ulam–Hyers stability, we first rewrite system (17) in compact vector form. This formulation facilitates the subsequent theoretical analysis.

First, model (17) is rewritten in compact vector form. Let

$$\mathbf{X}(t) = \begin{pmatrix} S(t) \\ I(t) \\ D(t) \\ C(t) \end{pmatrix}, \quad \mathbf{X}_0 = \begin{pmatrix} S_0 \\ I_0 \\ D_0 \\ C_0 \end{pmatrix}. \tag{66}$$

Define

$$\begin{cases} \mathbf{X}_1(t,S,I,D,C) = b^\mu S\left(1 - \dfrac{S}{K}\right) - (\alpha^\mu + r^\mu) S, \\ \mathbf{X}_2(t,S,I,D,C) = \alpha^\mu S - (\beta^\mu + r^\mu) I, \\ \mathbf{X}_3(t,S,I,D,C) = \beta^\mu I - (\gamma^\mu + r^\mu) D, \\ \mathbf{X}_4(t,S,I,D,C) = \gamma^\mu D - (r^\mu + r_1^\mu) C. \end{cases} \tag{67}$$

Then model (17) can be written as

$$\begin{cases} {}^C D_t^\mu \mathbf{X}(t) = \mathcal{Q}(t, \mathbf{X}(t)), \quad t \geq 0,\ 0 < \mu \leq 1, \\ \mathbf{X}(0) = \mathbf{X}_0, \end{cases} \tag{68}$$

where

$$\mathcal{Q}(t, \mathbf{X}(t)) = \begin{pmatrix} \mathbf{X}_1(t,S,I,D,C) \\ \mathbf{X}_2(t,S,I,D,C) \\ \mathbf{X}_3(t,S,I,D,C) \\ \mathbf{X}_4(t,S,I,D,C) \end{pmatrix}. \tag{69}$$

According to the Lemma 1, model (68) is equivalent to the following Volterra integral equation:

$$\mathbf{X}(t) = \mathbf{X}_0 + \frac{1}{\Gamma(\mu)} \int_0^t (t-s)^{\mu-1} \mathcal{Q}(s, \mathbf{X}(s))\, ds. \tag{70}$$

5. Existence, Uniqueness and Convergence of the Iterative Schemes

This section demonstrates the existence, uniqueness, and convergence of the proposed iterative schemes. We establish the well-posedness of model (17) and justify the convergence of both the ADM and the LADM.

Let

$$\mathbf{X}(t) = (S(t), I(t), D(t), C(t))^T,$$

and consider the nonlinear Volterra operator

$$\mathcal{T}(\mathbf{X})(t) = \begin{pmatrix} S_0 + {}^{RL}\mathcal{I}_t^{\mu}\left[b^{\mu}S - \dfrac{b^{\mu}}{K}S^2 - (\alpha^{\mu} + r^{\mu})S\right] \\ I_0 + {}^{RL}\mathcal{I}_t^{\mu}[\alpha^{\mu}S - (\beta^{\mu} + r^{\mu})I] \\ D_0 + {}^{RL}\mathcal{I}_t^{\mu}[\beta^{\mu}I - (\gamma^{\mu} + r^{\mu})D] \\ C_0 + {}^{RL}\mathcal{I}_t^{\mu}\left[\gamma^{\mu}D - (r^{\mu} + r_1^{\mu})C\right] \end{pmatrix}.$$

Then the Caputo fractional order model (17) is equivalent to the fixed-point equation

$$\mathbf{X} = \mathcal{T}(\mathbf{X}). \tag{71}$$

Let

$$\mathcal{B} = C([0, T], \mathbb{R}^4), \qquad \|\mathbf{X}\| = \max_{t\in[0,T]} (|S(t)| + |I(t)| + |D(t)| + |C(t)|).$$

Theorem 6. *Assume that there exists $M > 0$ such that*

$$\max\{|S(t)| + |I(t)| + |D(t)| + |C(t)|\} \le M \qquad \textit{for all } t \in [0, T].$$

Then operator $\mathcal{T}$ is locally Lipschitz on the closed ball

$$B_M = \{\mathbf{X} \in \mathcal{B} : \ \|\mathbf{X}\| \le M\}.$$

Moreover, if

$$\left[\frac{T^{\mu}}{\Gamma(\mu + 1)}\right] L < 1, \tag{72}$$

then

1. *Model (17) admits a unique solution in B_M;*
2. *The ADM series*

$$\mathbf{X}(t) = \sum_{n=0}^{\infty} \mathbf{X}_n(t) \tag{73}$$

   converges uniformly to that solution on $[0, T]$;
3. *The LADM series generates the same fixed-point iteration and therefore converges to the same unique solution.*

Proof. Let $\mathbf{X} = (S, I, D, C)$ and $\mathbf{Y} = (\widetilde{S}, \widetilde{I}, \widetilde{D}, \widetilde{C})$ be two elements of B_M. Using the Definition of $\mathcal{T}$, we have

$$\|{}^{RL}\mathcal{I}_t^{\mu} f\|_{\infty} \le \frac{T^{\mu}}{\Gamma(\mu + 1)} \|f\|_{\infty}.$$

Let

$$\mathbf{X}(t) = (S, I, D, C)^T, \qquad \mathbf{Y}(t) = (\widetilde{S}, \widetilde{I}, \widetilde{D}, \widetilde{C})^T \in B_M.$$

Then

$$\mathcal{T}(\mathbf{X})(t) - \mathcal{T}(\mathbf{Y})(t) = \begin{pmatrix} \Delta_1(t) \\ \Delta_2(t) \\ \Delta_3(t) \\ \Delta_4(t) \end{pmatrix}, \tag{74}$$

where

$$\begin{aligned} \Delta_1(t) &= {}^{RL}\mathcal{I}_t^{\mu}\Big[b^{\mu}(S-\widetilde{S}) - \frac{b^{\mu}}{K}(S^2-\widetilde{S}^2) - (\alpha^{\mu}+r^{\mu})(S-\widetilde{S})\Big], \\ \Delta_2(t) &= {}^{RL}\mathcal{I}_t^{\mu}\Big[\alpha^{\mu}(S-\widetilde{S}) - (\beta^{\mu}+r^{\mu})(I-\widetilde{I})\Big], \\ \Delta_3(t) &= {}^{RL}\mathcal{I}_t^{\mu}\Big[\beta^{\mu}(I-\widetilde{I}) - (\gamma^{\mu}+r^{\mu})(D-\widetilde{D})\Big], \\ \Delta_4(t) &= {}^{RL}\mathcal{I}_t^{\mu}\Big[\gamma^{\mu}(D-\widetilde{D}) - (r^{\mu}+r_1^{\mu})(C-\widetilde{C})\Big]. \end{aligned} \tag{75}$$

For the first component Δ_1 in (75), we obtain

$$S^2 - \widetilde{S}^2 = (S+\widetilde{S})(S-\widetilde{S}),$$

hence

$$\begin{aligned} &\left|b^{\mu}(S-\widetilde{S}) - \frac{b^{\mu}}{K}(S^2-\widetilde{S}^2) - (\alpha^{\mu}+r^{\mu})(S-\widetilde{S})\right| \\ &= \left|\left(b^{\mu} - (\alpha^{\mu}+r^{\mu}) - \frac{b^{\mu}}{K}(S+\widetilde{S})\right)(S-\widetilde{S})\right| \\ &\le \left(|b^{\mu} - (\alpha^{\mu}+r^{\mu})| + \frac{b^{\mu}}{K}|S+\widetilde{S}|\right)|S-\widetilde{S}|. \end{aligned}$$

Since $\mathbf{X}, \mathbf{Y} \in B_M$, we have $|S(t)| \le M$ and $|\widetilde{S}(t)| \le M$, so

$$|S+\widetilde{S}| \le 2M.$$

Therefore,

$$\left|b^{\mu}(S-\widetilde{S}) - \frac{b^{\mu}}{K}(S^2-\widetilde{S}^2) - (\alpha^{\mu}+r^{\mu})(S-\widetilde{S})\right| \le \left(|b^{\mu} - (\alpha^{\mu}+r^{\mu})| + \frac{2b^{\mu}M}{K}\right)|S-\widetilde{S}|.$$

we obtain

$$\|\Delta_1\|_{\infty} \le \frac{T^{\mu}}{\Gamma(\mu+1)}\left(|b^{\mu} - (\alpha^{\mu}+r^{\mu})| + \frac{2b^{\mu}M}{K}\right)\|S-\widetilde{S}\|_{\infty}.$$

Similarly, for the second component Δ_2 in (75), we have

$$|\alpha^{\mu}(S-\widetilde{S}) - (\beta^{\mu}+r^{\mu})(I-\widetilde{I})| \le \alpha^{\mu}|S-\widetilde{S}| + (\beta^{\mu}+r^{\mu})|I-\widetilde{I}|,$$

and hence

$$\|\Delta_2\|_{\infty} \le \frac{T^{\mu}}{\Gamma(\mu+1)}\Big[\alpha^{\mu}\|S-\widetilde{S}\|_{\infty} + (\beta^{\mu}+r^{\mu})\|I-\widetilde{I}\|_{\infty}\Big].$$

For the third component Δ_3 in (75), we get

$$|\beta^{\mu}(I-\widetilde{I}) - (\gamma^{\mu}+r^{\mu})(D-\widetilde{D})| \le \beta^{\mu}|I-\widetilde{I}| + (\gamma^{\mu}+r^{\mu})|D-\widetilde{D}|,$$

thus

$$\|\Delta_3\|_{\infty} \le \frac{T^{\mu}}{\Gamma(\mu+1)}\Big[\beta^{\mu}\|I-\widetilde{I}\|_{\infty} + (\gamma^{\mu}+r^{\mu})\|D-\widetilde{D}\|_{\infty}\Big].$$

For the fourth component Δ_4 in (75), we obtain

$$|\gamma^\mu(D-\widetilde{D}) - (r^\mu + r_1^\mu)(C-\widetilde{C})| \le \gamma^\mu |D-\widetilde{D}| + (r^\mu + r_1^\mu)|C-\widetilde{C}|,$$

and therefore

$$\|\Delta_4\|_\infty \le \frac{T^\mu}{\Gamma(\mu+1)}\Big[\gamma^\mu \|D-\widetilde{D}\|_\infty + (r^\mu + r_1^\mu)\|C-\widetilde{C}\|_\infty\Big].$$

Now, we obtain

$$\|\mathbf{X}-\mathbf{Y}\| = \max_{t\in[0,T]}\big(|S-\widetilde{S}| + |I-\widetilde{I}| + |D-\widetilde{D}| + |C-\widetilde{C}|\big),$$

Clearly, we can say

$$\|S-\widetilde{S}\|_\infty,\ \|I-\widetilde{I}\|_\infty,\ \|D-\widetilde{D}\|_\infty,\ \|C-\widetilde{C}\|_\infty \le \|\mathbf{X}-\mathbf{Y}\|.$$

Consequently,

$$\|\Delta_1\|_\infty \le \frac{T^\mu}{\Gamma(\mu+1)}\left(|b^\mu - (\alpha^\mu + r^\mu)| + \frac{2b^\mu M}{K}\right)\|\mathbf{X}-\mathbf{Y}\|,$$

$$\|\Delta_2\|_\infty \le \frac{T^\mu}{\Gamma(\mu+1)}\left(\alpha^\mu + \beta^\mu + r^\mu\right)\|\mathbf{X}-\mathbf{Y}\|,$$

$$\|\Delta_3\|_\infty \le \frac{T^\mu}{\Gamma(\mu+1)}\left(\beta^\mu + \gamma^\mu + r^\mu\right)\|\mathbf{X}-\mathbf{Y}\|,$$

$$\|\Delta_4\|_\infty \le \frac{T^\mu}{\Gamma(\mu+1)}\left(\gamma^\mu + r^\mu + r_1^\mu\right)\|\mathbf{X}-\mathbf{Y}\|.$$

Therefore, from operator (74), we finally obtain the following contraction inequality:

$$\|\mathcal{T}(\mathbf{X}) - \mathcal{T}(\mathbf{Y})\| \le \frac{T^\mu}{\Gamma(\mu+1)} L\,\|\mathbf{X}-\mathbf{Y}\|, \tag{76}$$

where

$$L = \max\left\{|b^\mu - (\alpha^\mu + r^\mu)| + \frac{2b^\mu M}{K},\ \alpha^\mu + \beta^\mu + r^\mu,\ \beta^\mu + \gamma^\mu + r^\mu,\ \gamma^\mu + r^\mu + r_1^\mu\right\}.$$

Hence $\mathcal{T}$ is a contraction whenever

$$\frac{T^\mu}{\Gamma(\mu+1)}\,L < 1.$$

The Banach fixed point theorem [24,25] then implies the existence and uniqueness of the solution.

Since the ADM construction generates successive approximations associated with the fixed-point iteration, the partial sums converge uniformly to the unique solution.

Furthermore, noting that

$$\mathcal{L}^{-1}\left[\frac{1}{s^\mu}\mathcal{L}\{f(t)\}\right] = {}^{RL}\mathcal{I}_t^\mu f(t),$$

The LADM iteration is equivalent to the ADM iteration; therefore, it converges to the same fixed point. □

Remark 2. *The above result establishes the well-posedness of the Caputo fractional order metabolic progression model (17). It guarantees the validity of both ADM and LADM approximations on any finite time interval satisfying the contraction condition. Standard extension arguments on invariant bounded sets follow a global continuation.*

Convergence Analysis

The solution obtained by the proposed approach is expressed as a rapidly converging series that consistently approaches the exact solution. Standard analytical tools are used to verify the convergence behavior of the series (73), following the methodology outlined in [16].

Theorem 7. *Let $\mathcal{B}$ be a Banach space, and let $\mathcal{T} : \mathcal{B} \to \mathcal{B}$ be a nonlinear contraction mapping satisfying, from (76),*

$$|\mathcal{T}(\mathbf{X}) - \mathcal{T}(\mathbf{Y})| \leq k|\mathbf{X} - \mathbf{Y}|, \quad 0 < k < 1, \quad \forall, \mathbf{X}, \mathbf{Y} \in \mathcal{B}, \tag{77}$$

where $k = \frac{T^{\mu}}{\Gamma(\mu+1)} L$. Then $\mathcal{T}$ admits a unique fixed point $\mathbf{X} \in \mathcal{B}$ such that

$$\mathcal{T}(\mathbf{X}) = \mathbf{X}.$$

The series defined in (44) can be represented using the ADM as

$$\mathbf{X}_m = \mathcal{T}(\mathbf{X}_{m-1}), \quad m = 1, 2, 3, \ldots$$

Assume that the initial term $\mathbf{X}_0 \in \mathcal{B}_M(\mathbf{X})$, where

$$\mathcal{B}_M(\mathbf{X}) = \{\mathbf{Z} \in \mathcal{B} : \|\mathbf{Z} - \mathbf{X}\| < M\}.$$

Proof. (i) For $m = 1$, we obtain

$$\|\mathbf{X}_1 - \mathbf{X}\| = \|\mathcal{T}(\mathbf{X}_0) - \mathcal{T}(\mathbf{X})\| \leq k\|\mathbf{X}_0 - \mathbf{X}\|.$$

Assume that the result holds for $m = n$, i.e.,

$$\|\mathbf{X}_n - \mathbf{X}| \leq k^n \|\mathbf{X}_0 - \mathbf{X}\|.$$

Then, for $m = n + 1$, we have

$$\|\mathbf{X}_{n+1} - \mathbf{X}\| = \|\mathcal{T}(\mathbf{X}_n) - \mathcal{T}(\mathbf{X})\| \leq k\|\mathbf{X}_n - \mathbf{X}\| \leq k^{n+1}\|\mathbf{X}_0 - \mathbf{X}\|.$$

Thus,

$$\|\mathbf{X}_n - \mathbf{X}\| \leq k^n \|\mathbf{X}_0 - \mathbf{X}\| < M, \tag{78}$$

which shows that $\mathbf{X}_n \in \mathcal{B}_M(\mathbf{X})$.

(ii) Since $0 < k < 1$, it follows that $k^n \to 0$ as $n \to \infty$. Hence, from inequality (78),

$$\lim_{n\to\infty} \|\mathbf{X}_n - \mathbf{X}\| = 0,$$

which implies

$$\lim_{n\to\infty} \mathbf{X}_n = \mathbf{X}.$$

□

6. Ulam–Hyers Stability

This section examines Ulam–Hyers (U-H) stability to assess the robustness of the model solutions. Ulam first introduced this concept in 1940, and Hyers later developed it further. It has since been extended to Ulam–Hyers–Rassias (U-H-R) stability. In recent years, researchers in differential equations have paid close attention to this topic. To support the following analysis, the key definitions and main results of [12,13,34,35] are reviewed.

Definition 7. *The model* (17) *is U-H stable for* $\epsilon > 0$ *if for any* $W \in X$ *satisfying*

$$\|\mathbf{X} - \mathcal{T}\mathbf{X}\| \leq \epsilon, \quad t \in [0, T], \tag{79}$$

there exists a unique solution $\mathbf{Y}$ *of* (71) *such that there exists* $C_q > 0$ *satisfying*

$$\|\mathbf{Y} - \mathbf{X}\| \leq C_q \epsilon, \quad t \in [0, T]. \tag{80}$$

Definition 8. *For* $\Upsilon \in C(\mathbb{R}, \mathbb{R})$ *with* $\Upsilon(0) = 0$, *model* (71) *is said to be generalized U-H stable if for any solution W of* (79), *there exists a unique solution* $\mathbf{Y}$ *of* (71) *such that*

$$\|\mathbf{Y} - \mathbf{X}\| \leq \Upsilon(\epsilon). \tag{81}$$

Remark 3. *For* $x(t) \in C([0, T]; \mathbb{R})$, $\mathbf{Y} \in \mathcal{B}$ *satisfies* (79) *if*

(i) $|x(t)| \leq \epsilon, \forall t \in [0, T]$;
(ii) $\mathcal{T}\mathbf{X}(t) = \mathbf{Y} + x(t), \forall t \in [0, T]$.

The following relation is needed in future work. The perturbed problem corresponding to equation (68) is given by

$$\begin{cases} {}^{C}D_t^{\mu}\mathbf{X}(t) = \mathcal{Q}(t, \mathbf{X}(t)) + x(t), \\ \mathbf{X}(0) = \mathbf{X}_0. \end{cases} \tag{82}$$

Lemma 3. *The perturbed problem* (17) *satisfies the relation*

$$|\mathbf{X}(t) - \mathcal{T}\mathbf{X}(t)| \leq a\epsilon, \quad \text{where } a = \frac{T^{\mu}}{\Gamma(\mu + 1)}. \tag{83}$$

Proof. By Remark 3 and Lemma 1, we obtain the required result. □

Theorem 8. *The solution to model* (17) *is U-H stable and generalized U-H stable if* $aL < 1$.

Proof. For any solution $W \in \mathcal{B}$ of problem (82) and $\mathbf{Y} \in \mathcal{B}$ representing the unique solution to problem (68), we have

$$\begin{aligned} |\mathbf{X}(t) - \mathbf{Y}(t)| &= |\mathbf{X}(t) - \mathcal{T}\mathbf{X}(t)| \\ &\leq |\mathbf{X}(t) - \mathcal{T}\mathbf{X}(t)| + |\mathcal{T}\mathbf{X}(t) - \mathcal{T}\mathbf{Y}(t)| \\ &\leq a\epsilon + \frac{T^{\mu}L}{\Gamma(\mu + 1)}|\mathbf{X}(t) - \mathbf{Y}(t)|, \\ &\leq \frac{a\epsilon}{1 - aL}. \end{aligned} \tag{84}$$

Thus, problem (17) is U-H and generalized U-H stable. □

Definition 9. *Model (71) is U-H-R stable with $\chi \in C([0,T],\mathbb{R})$ if for $\epsilon > 0$ and any solution $W \in \mathcal{B}$ satisfying*

$$\|\mathbf{X} - \mathcal{T}\mathbf{X}\| \leq \chi(t)\epsilon, \quad t \in [0,T], \tag{85}$$

there exists a unique solution $\mathbf{Y}$ *of (71) such that*

$$\|\mathbf{Y} - \mathbf{X}\| \leq C_q\chi(t)\epsilon, \quad t \in [0,T]. \tag{86}$$

Definition 10. *For $\chi \in C([0,T],\mathbb{R})$, if there exist constants $C_{q,\chi}$ such that for any solution W of (85) and a unique solution $\mathbf{Y}$ of (71),*

$$\|\mathbf{Y} - \mathbf{X}\| \leq C_{q,\chi}\chi(t), \quad t \in [0,T], \tag{87}$$

then model (71) is generalized U-H-R stable.

Remark 4. *If there exists $x(t) \in C([0,T];\mathbb{R})$, then $\mathbf{Y} \in \mathcal{B}$ satisfies (17) if*

(i) $|x(t)| \leq \epsilon\chi(t), \forall t \in [0,T]$;
(ii) $\mathcal{T}\mathbf{X}(t) = \mathbf{Y} + x(t), \forall t \in [0,T]$.

Lemma 4. *For the perturbed problem (82), the following holds:*

$$|\mathbf{X}(t) - \mathcal{T}\mathbf{X}(t)| \leq a\chi(t)\epsilon. \tag{88}$$

Proof. Using Remark 3 and Lemma 1, one obtains the result. □

Theorem 9. *The solution to model (17) is U-H-R and generalized U-H-R stable if $aL < 1$.*

Proof. Let $\mathbf{X} \in \mathcal{B}$ be any solution and $\mathbf{Y} \in \mathcal{B}$ the unique solution to the problem (17). Then

$$\begin{aligned} |\mathbf{X}(t) - \mathbf{Y}(t)| &\leq |\mathbf{X}(t) - \mathcal{T}\mathbf{X}(t)| + |\mathcal{T}\mathbf{X}(t) - \mathcal{T}\mathbf{Y}(t)| \\ &\leq a\chi(t)\epsilon + \frac{T^{\mu}L}{\Gamma(\alpha+1)}|\mathbf{X}(t) - \mathbf{Y}(t)| \\ &\leq \frac{a\chi(t)\epsilon}{1-aL}. \end{aligned} \tag{89}$$

Thus, Caputo's fractional order model (68) is U-H-R and generalized U-H-R stable. □

7. Numerical Results and Discussion

This section presents numerical simulations that demonstrate the dynamic behavior of the model and examine the influence of the fractional order parameter.

In this study, numerical simulations of the proposed fractional order diabetes progression model with a singular kernel are performed using the Caputo derivative. Model (15) is analyzed using the ADM and the LADM. The initial conditions are taken as $S(0) = S_0$, $I(0) = I_0$, $D(0) = D_0$, and $C(0) = C_0$, with parameters chosen according to realistic population data.

The fractional order formulation introduces a memory effect into the singular-kernel model (15), allowing a more accurate representation of the progression of chronic diseases. The effectiveness of the ADM and LADM approaches is demonstrated by the convergence of the series solutions and the consistency of the results obtained from both methods.

The fractional order parameter μ has a significant impact on model (15) dynamics. Lower values of μ slow the progression between compartments, indicating that memory

effects delay the development of the disease. In contrast, the classical case $\mu = 1$ leads to faster transitions and higher peaks in the intermediate compartments.

Thus, the approximate solution obtained using the ADM is expressed as a truncated series:

$$\begin{aligned} S(t) &= S_0 + S_1(t) + S_2(t) + S_3(t) + \cdots, \\ I(t) &= I_0 + I_1(t) + I_2(t) + I_3(t) + \cdots, \\ D(t) &= D_0 + D_1(t) + D_2(t) + D_3(t) + \cdots, \\ C(t) &= C_0 + C_1(t) + C_2(t) + C_3(t) + \cdots. \end{aligned} \tag{90}$$

$$\begin{aligned} S_1(t) &= \frac{[(b-\alpha-r)S_0 - \frac{b}{K}S_0^2]}{\Gamma(\mu+1)}t^{\mu}, \\ I_1(t) &= \frac{[\alpha S_0 - (\beta+r)I_0]}{\Gamma(\mu+1)}t^{\mu}, \\ D_1(t) &= \frac{[\beta I_0 - (\gamma+r)D_0]}{\Gamma(\mu+1)}t^{\mu}, \\ C_1(t) &= \frac{[\gamma D_0 - (r+r_1)C_0]}{\Gamma(\mu+1)}t^{\mu}. \end{aligned} \tag{91}$$

$$\begin{aligned} S_2(t) &= \frac{[(b-\alpha-r)S_1 - \frac{b}{K}(2S_0S_1)]}{\Gamma(2\mu+1)}t^{2\mu}, \\ I_2(t) &= \frac{[\alpha S_1 - (\beta+r)I_1]}{\Gamma(2\mu+1)}t^{2\mu}, \\ D_2(t) &= \frac{[\beta I_1 - (\gamma+r)D_1]}{\Gamma(2\mu+1)}t^{2\mu}, \\ C_2(t) &= \frac{[\gamma D_1 - (r+r_1)C_1]}{\Gamma(2\mu+1)}t^{2\mu}. \end{aligned} \tag{92}$$

$$\begin{aligned} S_3(t) &= \frac{[(b-\alpha-r)S_2 - \frac{b}{K}(S_1^2+2S_0S_2)]}{\Gamma(3\mu+1)}t^{3\mu}, \\ I_3(t) &= \frac{[\alpha S_2 - (\beta+r)I_2]}{\Gamma(3\mu+1)}t^{3\mu}, \\ D_3(t) &= \frac{[\beta I_2 - (\gamma+r)D_2]}{\Gamma(3\mu+1)}t^{3\mu}, \\ C_3(t) &= \frac{[\gamma D_2 - (r+r_1)C_2]}{\Gamma(3\mu+1)}t^{3\mu}. \end{aligned} \tag{93}$$

Hence, the truncated approximate solutions are given by:

$$\begin{aligned} S(t) &\approx S_0 + S_1(t) + S_2(t) + S_3(t), \\ I(t) &\approx I_0 + I_1(t) + I_2(t) + I_3(t), \\ D(t) &\approx D_0 + D_1(t) + D_2(t) + D_3(t), \\ C(t) &\approx C_0 + C_1(t) + C_2(t) + C_3(t). \end{aligned} \tag{94}$$

To perform numerical simulations of the proposed fractional order diabetes model (17), appropriate initial conditions and parameter values are selected based on realistic population data.

The parameter values in Table 3 are adopted from [1] and retain the same units used in the source reference (annual rates, year^{-1}). In the fractional order model (17), the

corresponding effective rates are interpreted as having units of year$^{-\mu}$ to remain consistent with the model's dimensional structure.

The initial population distribution among the compartments is presented in Table 4, while the model parameters used in the analysis are listed in Table 3.

A three-term truncation is employed as a practical approximation, while higher-order terms are regarded as increasingly minor correction terms within the ADM/LADM expansions. Thus, substituting the parameter values and initial conditions into the ADM formulation and taking $\mu = 0.9$, the approximate solutions up to three terms are obtained as:

$$\begin{aligned} S(t) &= 39029000 - 1230834.6\, t^{0.9} - 114556.5\, t^{1.8} - 18703.6\, t^{2.7}, \\ I(t) &= 3000000 + 1048403.6\, t^{0.9} - 56366.2\, t^{1.8} - 10068.4\, t^{2.7}, \\ D(t) &= 3269000 + 73806.7\, t^{0.9} + 24460.8\, t^{1.8} + 5994.2\, t^{2.7}, \\ C(t) &= 1401000 + 46555.3\, t^{0.9} + 12532.6\, t^{1.8} + 2876.6\, t^{2.7}. \end{aligned} \tag{95}$$

Table 3. Model (17) parameters used in the simulations.

Parameter	Value	Source
b	0.0176	[1]
K	5.0×10^7	Assumed [1]
α	0.03	[1]
β	0.05	[1]
γ	0.02	[1]
r	0.0042	[1]
r_1	0.0105	[1]

Table 4. Initial conditions used in the numerical simulations.

Variable	Initial Value	Source
$S(0)$	3.9029×10^7	[1]
$I(0)$	3.0×10^6	[1]
$D(0)$	3.269×10^6	[1]
$C(0)$	1.401×10^6	[1]

Figure 2 illustrates how variations in the fractional order parameter μ influence the dynamics of the susceptible population $S(t)$. The results show that $S(t)$ decreases monotonically for all values of μ, reflecting the ongoing transition of individuals into the insulin-resistant class. A clear dependence on fractional order is observed: lower values of μ correspond to a slower decline in $S(t)$, suggesting that memory effects delay disease progression. In contrast, when $\mu = 1$, the susceptible population decreases more rapidly; these findings indicate that the fractional dynamics exert a moderating influence on model (17), thus maintaining a larger susceptible population over time.

Figure 3 illustrates how variations in the fractional order parameter μ influence the dynamics of the insulin-resistant population $I(t)$. For all values of μ, $I(t)$ increases, peaks, and then gradually decreases, reflecting the transition from susceptibility to insulin resistance and then to more advanced stages. The results indicate a strong dependence on fractional order: lower μ values delay and reduce the peak of $I(t)$, suggesting that memory effects slow disease progression. In contrast, higher μ values result in an earlier and higher peak. These findings demonstrate that fractional dynamics significantly influence both the timing and magnitude of the insulin-resistant population.

Figure 4 illustrates how variations in the fractional order parameter μ influence the dynamics of the diabetic population without complications, $D(t)$. $D(t)$ increases steadily for all μ values as individuals transition from the insulin-resistant group. Higher μ values

accelerate this increase, while lower values slow it down. This shows that memory effects, as reflected in fractional dynamics, can delay disease progression and reduce the rate at which individuals develop diabetes without complications.

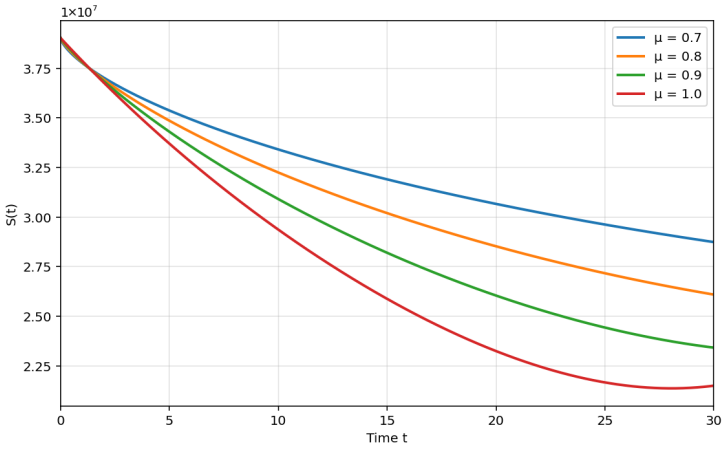


Figure 2. Comparison of ADM solutions for $S(t)$ at different fractional orders μ.

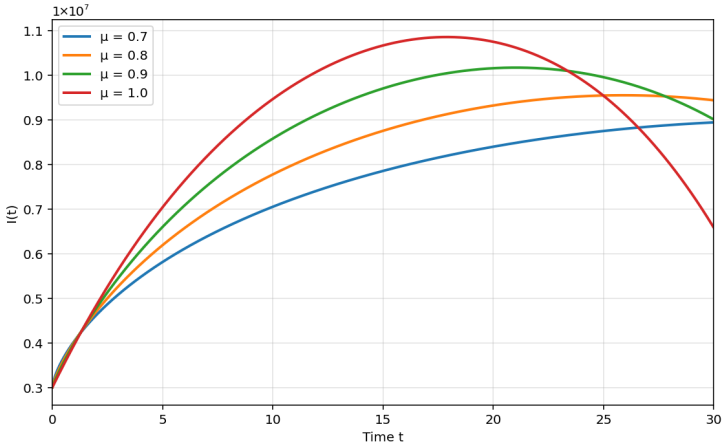


Figure 3. Comparison of ADM solutions for $I(t)$ at different fractional orders μ.

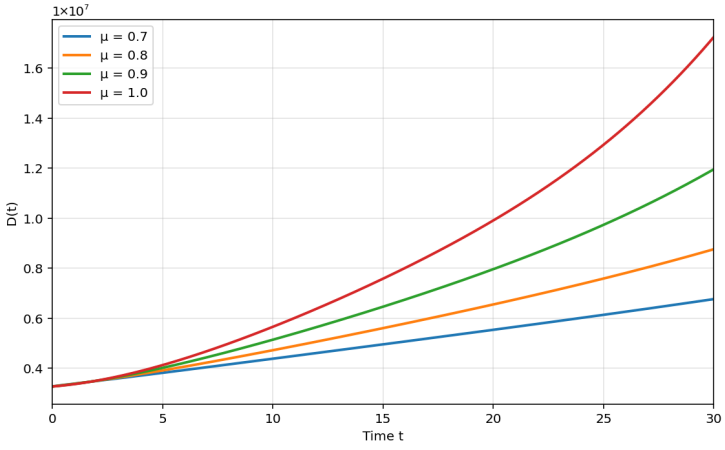


Figure 4. Comparison of ADM solutions for $D(t)$ at different fractional orders μ.

Figure 5 illustrates how variations in the fractional order parameter μ influence the dynamics of diabetic patients with complications, represented by $C(t)$. The results show that $C(t)$ increases steadily in all values of μ, highlighting how complications accumulate as the disease progresses. The fractional order affects how quickly this happens: higher

μ values make $C(t)$ grow faster, while lower values slow it down. This suggests that the memory effects of fractional dynamics can delay the start and worsening of severe disease outcomes.

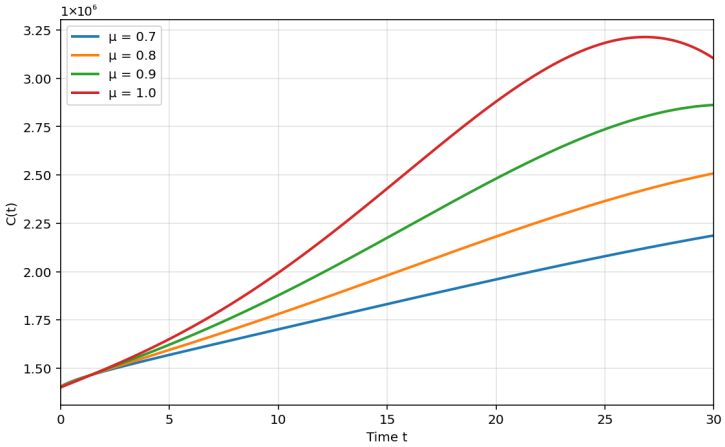


Figure 5. Comparison of ADM solutions for $C(t)$ at different fractional orders μ.

7.1. ADM vs. LADM Comparison at $\mu = 0.9$

The temporal dynamics of the model, as illustrated in Figures 2–5, show that the susceptible population $S(t)$ decreases monotonically over time. The insulin-resistant population $I(t)$ initially increases, reaches a peak, and then declines. Both the diabetic population without complications $D(t)$ and the complication class $C(t)$ increase over time, reflecting the progressive nature of the disease.

A comparison of ADM and LADM solutions confirms that both methods yield identical numerical results for the present model (17), thus validating the accuracy and robustness of the analytical approach.

Figures 6–9 compare ADM and LADM solutions for $\mathbf{X}(t) = (S(t), I(t), D(t), C(t))^T$ in $\mu = 0.9$, where $\mathbf{X}(t)$ denotes compartment variables. The plots overlap completely, demonstrating that both methods produce identical numerical results for this model. This confirms the accuracy and consistency of ADM and LADM in solving the fractional model (17).

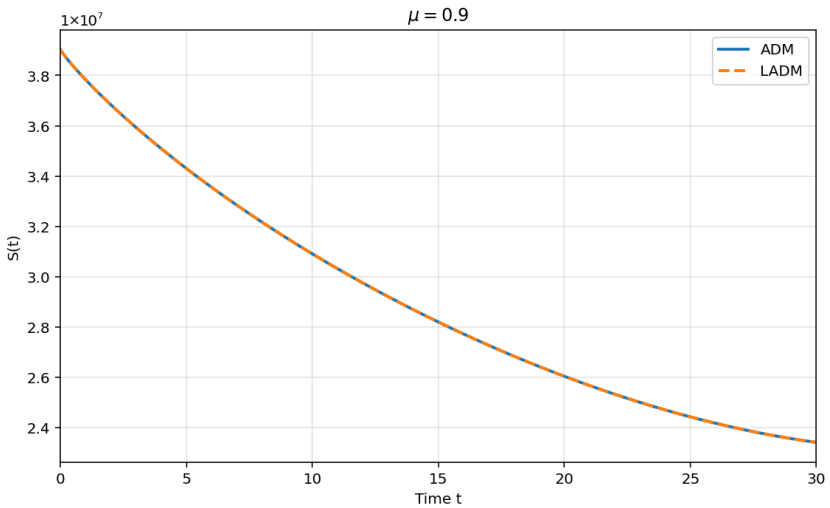


Figure 6. Comparison of ADM vs. LADM solutions for $S(t)$ at $\mu = 0.9$.

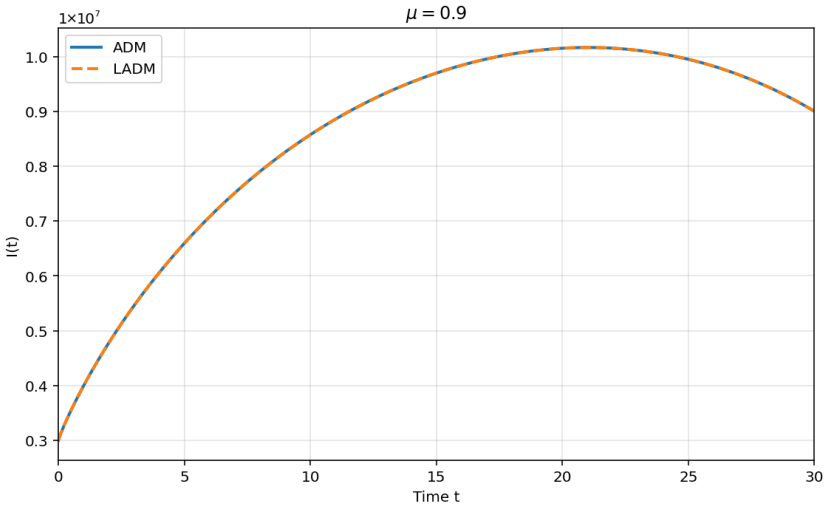


Figure 7. Comparison of ADM vs. LADM solutions for $I(t)$ at $\mu = 0.9$.

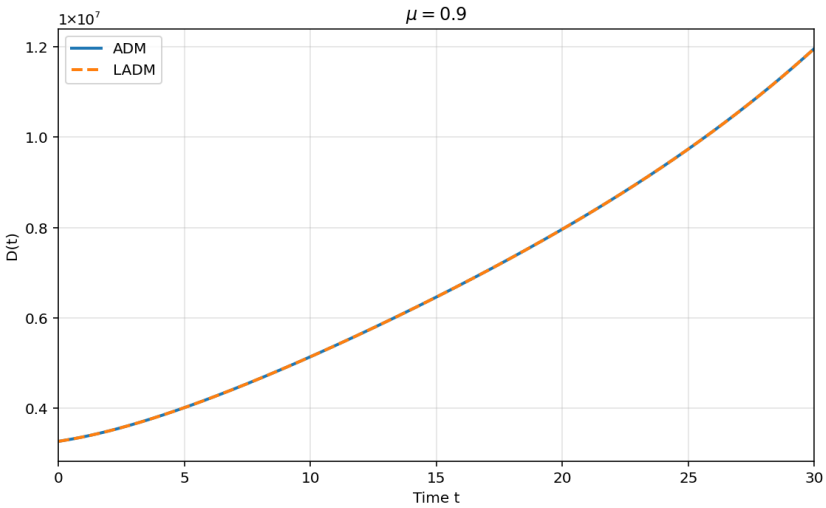


Figure 8. Comparison of ADM vs. LADM solutions for $D(t)$ at $\mu = 0.9$.

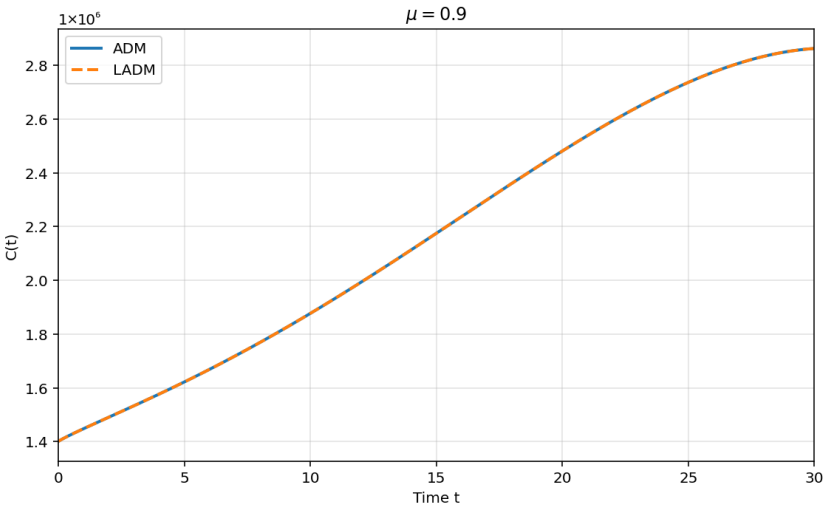


Figure 9. Comparison of ADM vs. LADM solutions for $C(t)$ at $\mu = 0.9$.

Figure 10 illustrates the dynamics of the state vector $\mathbf{X}(t) = (S(t), I(t), D(t), C(t))^T$ for the fractional order $\mu = 0.9$. The susceptible population decreases over time, whereas the insulin-resistant and diabetic populations initially increase as the disease progresses. The complication class $C(t)$ increases continuously, representing the long-term accumulation of severe cases. This behavior clearly reflects the sequential transition mechanism $S \to I \to D \to C$, which confirms the consistency of the dynamics of the singular kernel model (17).

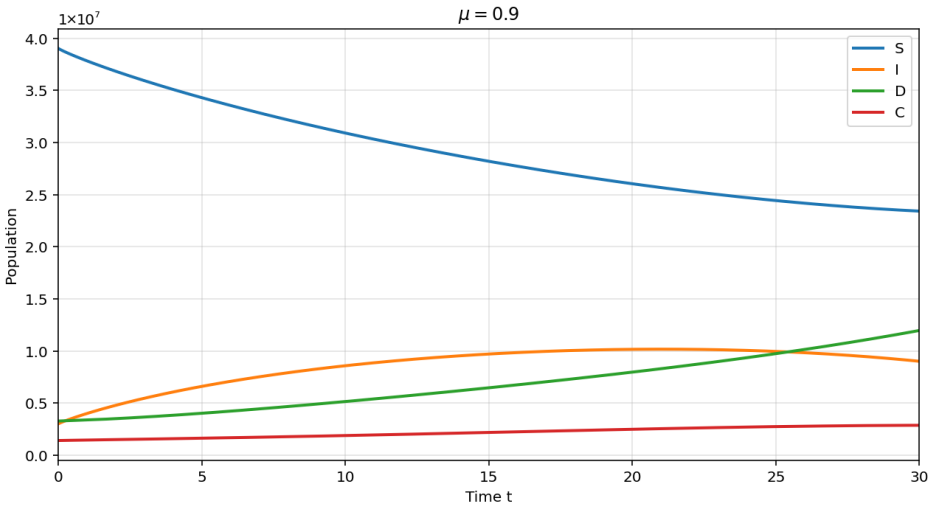


Figure 10. Temporal evolution of the state vector $\mathbf{X}(t) = (S(t), I(t), D(t), C(t))^T$ at $\mu = 0.9$.

7.2. Verification of the Contraction Condition

The parameter values from Table 3 are used to verify that the contraction condition from Theorem 6 is as follows:

$$M = S_0 + I_0 + D_0 + C_0 = 4.6699 \times 10^7$$

and using the representative fractional order $\mu = 0.9$, we obtain

$$b^\mu = 0.02636, \quad \alpha^\mu = 0.04260, \quad \beta^\mu = 0.06746, \quad \gamma^\mu = 0.02958,$$

$$r^\mu = 0.00726, \quad r_1^\mu = 0.01656.$$

Thus,

$$L = \max\left\{ |b^\mu - (\alpha^\mu + r^\mu)| + \frac{2b^\mu M}{K},\ \alpha^\mu + \beta^\mu + r^\mu,\ \beta^\mu + \gamma^\mu + r^\mu,\ \gamma^\mu + r^\mu + r_1^\mu \right\}.$$

Substituting the above values gives

$$L = \max\{0.07274, 0.11732, 0.10430, 0.05340\} = 0.11732.$$

Since $\Gamma(1.9) = 0.96177$, applying Theorem 6, we get

$$\frac{T^\mu}{\Gamma(\mu + 1)} L < 1$$

is satisfied with

$$T < \left(\frac{\Gamma(1.9)}{0.11732} \right)^{1/0.9} \approx 10.36.$$

Hence, the contraction condition is satisfied for $T < 10.36$ years, and therefore the existence and uniqueness result holds on the interval $0 \leq t < 10.36$.

Moreover, the numerical parameter set satisfies the condition $aL < 1$, which guarantees the validity of the Ulam–Hyers and generalized Ulam–Hyers stability results. This also supports consistency between the stability analysis and the numerical solutions presented for ADM and LADM.

8. Conclusions

The present study introduces a fractional order compartmental model for diabetes progression based on the Caputo derivative with a singular kernel. The model describes transitions among susceptible, insulin-resistant, diabetic without complications, and diabetic with complications groups. A key originality of the study lies in incorporating memory effects into disease-stage transitions within a dimensionally consistent fractional order model for chronic disease progression. The main mathematical properties of the model, including existence, uniqueness, positivity, and boundedness of solutions, were rigorously established. Equilibrium points were identified, and both local and global stability analyses showed that healthy and chronic progression states remain stable under specific conditions. Ulam–Hyers stability further confirmed the model's robustness. The ADM and LADM were both applied to obtain approximate solutions. The results show that the fractional order parameter significantly influences the dynamics of model (15); lower fractional orders introduce stronger memory effects that slow disease progression, whereas the classical case leads to faster transitions between groups. Overall, the fractional order model provides a flexible scheme for understanding the temporal progression of diabetes. These findings highlight the importance of incorporating memory effects in chronic disease models and support future research on control strategies and data-driven validation. Additionally, the agreement between ADM and LADM is used here as a consistency check for the numerical results. Because classical Runge–Kutta methods are not directly suitable for the Caputo fractional order system, a comparison with an independent fractional solver is left for future work.

Overall, the work novelty is shown in the following points to clarify the main contribution:

I. The underlying compartmental structure considered in this study is adapted from [1]; however, the contributions of the present work extend beyond this baseline model.
II. First, model [1] is reformulated in a dimensionally consistent fractional order to incorporate memory effects in disease progression.
III. Second, new analytical results in the fractional setting are established, including existence, positivity, boundedness, local and global stability, and Ulam–Hyers stability.
IV. Third, the influence of the fractional order parameter on long-term disease dynamics is investigated using numerical methods, including ADM and LADM simulations.

These contributions provide a substantive extension of the original model, beyond merely replacing the classical derivative with a Caputo derivative (a singular-based kernel).

9. Future Work

This study presents a fractional order framework for modeling the progression of diabetes with variable memory effects. There are several ways to make the model more useful and realistic. First, the model could be improved by using fractional derivatives of variable or complex orders [34–36], whose orders vary over time or with the system's state. This would allow for a more flexible way to show how memory effects change as the disease progresses. Second, future research could integrate optimal control strategies to design effective intervention policies. Incorporating control variables, such as lifestyle

modifications, treatment rates, and allocation of healthcare resources, would facilitate the identification of strategies that minimize disease burden and associated costs over time [18]. Third, the model could be improved by using real epidemiological data to estimate and check its parameters. Calibrating the model with real data would improve its accuracy and enable its application to real healthcare planning. In addition, adding random effects to the model could better capture uncertainties in disease progression and external influences [37]. This would help to analyze how model (15) changes and how robust it is to random events. Another promising direction is to expand the model by adding more compartments, such as pre-diabetic stages, treatment adherence, and other health conditions. This would give a more detailed view of the metabolic changes. The same approach could also be applied to other chronic diseases with similar patterns, demonstrating that fractional order modeling can be useful for studying long-term disease progression. These improvements would help us better understand how diabetes works and support the creation of more effective ways to manage and control the disease [38,39].

Author Contributions: P.V. and W.S.; Methodology, P.V.; Software, P.V.; Validation, P.V. and W.S.; formal analysis, P.V.; Investigation, W.S.; Resources, P.V.; Writing—original draft, W.S.; Writing—review and editing, W.S.; Supervision, W.S. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded in whole by the National Science Center, Poland, grant number 2023/51/B/ST8/01062. For the purpose of Open Access, the authors have applied a CC-BY public copyright license to any Author Accepted Manuscript (AAM) version arising from this submission.

Data Availability Statement: No new data were created or analyzed in this study.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Belkhodja, K.; Rahmoun, A. Dynamic Modeling and Optimal Control of Type 2 Diabetes: An Application to Algerian Data. *Oper. Res. Forum* **2026**, *7*, 34. [CrossRef]
2. Ahmed, K.I.; Safa, Z.M.; Mirgani, A.; Seadawy, A.; Saber, S. A comprehensive investigation of fractional glucose–insulin dynamics: Existence, stability, and numerical comparisons using residual power series and generalized Runge–Kutta methods. *J. Taibah Univ. Sci.* **2025**, *19*, 2460280. [CrossRef]
3. Akinsola, V.O.; Oluyo, T.O. Mathematical analysis with numerical solutions of the mathematical model for the complications and control of diabetes mellitus. *J. Stat. Manag. Syst.* **2019**, *22*, 845–869. [CrossRef]
4. Shurbaij, M.A.; Kader, L.A.; Hannan, H.; Mortula, M.; Husseini, G.A. Comprehensive Study of a Diabetes Mellitus Mathematical Model Using Numerical Methods with Stability and Parametric Analysis. *Int. J. Environ. Res. Public Health* **2023**, *20*, 939. [CrossRef]
5. Karaagac, B.; Owolabi, K.M.; Pindza, E. A computational technique for the Caputo fractal-fractional diabetes mellitus model without genetic factors. *Int. J. Dyn. Control* **2023**, *11*, 2161–2178. [CrossRef]
6. Sheikh, N.A.; Jamil, M.; Ching, D.L.C.; Khan, I.; Usman, M.; Nisar, K.S. A generalized model for quantitative analysis of sediments loss: A Caputo time fractional model. *J. King Saud Univ.-Sci.* **2021**, *33*, 101179. [CrossRef]
7. Eddine, N.C.; Ragusa, M.A.; Repovš, D.D. On the concentration-compactness principle for anisotropic variable exponent Sobolev spaces and its applications. *Fract. Calc. Appl. Anal.* **2024**, *27*, 725–756. [CrossRef]
8. Ma, D.; Wu, F. Fractional Navier-Stokes equations regularity criteria in terms of deformation tensor. *Filomat* **2025**, *39*, 239–247. [CrossRef]
9. Rezapour, S.; Azzaoui, B.; Tellab, B.; Etemad, S.; Masiha, H.P. An Analysis on the Positive Solutions for a Fractional Configuration of the Caputo Multiterm Semilinear Differential Equation. *J. Funct. Spaces* **2025**, *2025*, 9839230. [CrossRef]
10. Dubey, R.S.; Goswami, P. Mathematical model of diabetes and its complication involving fractional operator without singular kernal. *Discret. Contin. Dyn. Syst.-Ser. S* **2021**, *14*, 2151–2161. [CrossRef]
11. Sumelka, W.; Voyiadjis, G.Z. A hyperelastic fractional damage material model with memory. *Int. J. Solids Struct.* **2017**, *124*, 151–160. [CrossRef]

12. Saber, S.; Alahmari, A.; Shokeralla, A.A.; Guma, F.E. Numerical Techniques for Solving Fractional Glucose–Insulin Regulatory Systems. *Lobachevskii J. Math.* **2025**, *46*, 5471–5483. [CrossRef]
13. Saber, S.; Solouma, E.; Alharb, R.A.; Alalyani, A. Chaos in fractional-order glucose–insulin models with variable derivatives: Insights from the Laplace–Adomian decomposition method and generalized Euler techniques. *Fractal Fract.* **2025**, *9*, 149. [CrossRef]
14. Farman, M.; Akgul, A.; Sultan, M.; Riaz, S.; Asif, H.; Agarwal, P.; Hassani, M.K. Numerical study and dynamics analysis of diabetes mellitus with co-infection of COVID-19 virus by using fractal fractional operator. *Sci. Rep.* **2024**, *14*, 16489. [CrossRef]
15. Diethelm, K.; Garrappa, R.; Giusti, A.; Stynes, M. Why Fractional Derivatives with Nonsingular Kernels Should Not Be Used. *Fract. Calc. Appl. Anal.* **2020**, *23*, 610–634. [CrossRef]
16. Yunus, A.O.; Olayiwola, M.O.; Omoloye, M.A.; Oladapo, A.O. A fractional order model of Lassa disease using the Laplace–Adomian Decomposition Method. *Healthc. Anal.* **2023**, *3*, 100167. [CrossRef]
17. Al-Mekhlafi, S.M.; Hasan, M.M.A.; Al-Ali, H.A.; Mukandavire, Z. A crossover optimal control framework for a time-delayed fractional-order diabetes model: Stability, bifurcation, and numerical analysis. *Math. Methods Appl. Sci.* **2025**, *48*, 15723–15741. [CrossRef]
18. Das, P.; Das, S.; Das, P.; Rihan, F.A.; Uzuntarla, M.; Ghosh, D. Optimal control strategy for cancer remission using combinatorial therapy: A mathematical model-based approach. *Chaos Solitons Fractals* **2021**, *145*, 110789. [CrossRef]
19. Hossain, M.; Ullah, M.A. A new hybrid Laplace Adomian variational iteration method for solving nonlinear time-fractional partial differential equations. *Ann. Univ. Ferrara* **2026**, *72*, 37. [CrossRef]
20. Sadri, K.; Amilo, D.; Hincal, E. Mathematical analysis of fractional-order glaucoma disease with feedback control using Chebyshev polynomials of sixth kind. *J. Appl. Math. Comput.* **2026**, *72*, 92. [CrossRef]
21. Nisar, K.S.; Farman, M. Investigation of fractional order model for glucose-insulin monitoring with PID and controllability. *Sci. Rep.* **2025**, *15*, 8128. [CrossRef] [PubMed]
22. Nawaz, R.; Kainat, S.; Long, N.M.A.B.N.; Shah, K.; Abdeljawad, T. Fractal–fractional order mathematical modelling of co–infection dynamics of tuberculosis and diabetes mellitus with vaccination. *J. Appl. Math. Comput.* **2026**, *72*, 60. [CrossRef]
23. Hasan, M.M.A.; Alghanmi, A.M.; AL-Mekhlafi, S.M.; Ali, H.A.; Mukandavire, Z. A Novel Crossover Dynamics of Variable-Order Fractal-Fractional Stochastic Diabetes Model: Numerical Simulations. *J. Math.* **2025**, *2025*, 2986543. [CrossRef]
24. Verma, P.; Kumar, M. Analytical solution with existence and uniqueness conditions of non-linear initial value multi-order fractional differential equations using Caputo derivative. *Eng. Comput.* **2022**, *38*, 661–678. [CrossRef]
25. Verma, P.; Kumar, M. An analytical solution with existence and uniqueness conditions for fractional integro-differential equations. *Int. J. Model. Simul. Sci. Comput.* **2020**, *11*, 2050045. [CrossRef]
26. Adedokun, K.A.; Olayiwola, M.O.; Alaje, I.A.; Yunus, A.O.; Oladapo, A.O.; Kareem, K.O. A caputo fractional-order model of tuberculosis incorporating enlightenment and therapy using the Laplace–Adomian decomposition method. *Int. J. Model. Simul.* **2025**, *45*, 2097–2111. [CrossRef]
27. Shankar, M.; Metzler, R.; Li, C. A survey on the Ulam–Hyers stability of fractional-order differential equations. *J. Phys. A Math. Theor.* **2025**, *58*, 453001. [CrossRef]
28. Sumelka, W.; Łuczak, B.; Gajewski, T.; Voyiadjis, G. Modelling of AAA in the framework of time-fractional damage hyperelasticity. *Int. J. Solids Struct.* **2020**, *206*, 30–42. [CrossRef]
29. Voyiadjis, G.Z.; Sumelka, W. Brain modelling in the framework of anisotropic hyperelasticity with time fractional damage evolution governed by the Caputo-Almeida fractional derivative. *J. Mech. Behav. Biomed. Mater.* **2019**, *89*, 209–216. [CrossRef]
30. Chen, Y.; Liu, F.; Yu, Q.; Li, T. Review of fractional epidemic models. *Appl. Math. Model.* **2021**, *97*, 281–307. [CrossRef] [PubMed]
31. Almeida, R.; da Cruz, A.M.C.B.; Martins, N.; Monteiro, M.T.T. An epidemiological MSEIR model described by the Caputo fractional derivative. *Int. J. Dyn. Control* **2019**, *7*, 776–784. [CrossRef]
32. Fang, K.; Zhu, Z.; Chen, F.; Li, Z. Qualitative and Bifurcation Analysis in a Leslie–Gower Model with Allee Effect. *Qual. Theory Dyn. Syst.* **2022**, *21*, 86. [CrossRef]
33. Khan, N.A.; Razzaq, O.A.; Mondal, S.P.; Rubbab, Q. Fractional order ecological system for complexities of interacting species with harvesting threshold in imprecise environment. *Adv. Differ. Equ.* **2019**, *2019*, 405. [CrossRef]
34. Verma, P.; Sumelka, W. A qualitative analysis of nonlinear ψ-Hilfer equations with complex fractional order. *R. Soc. Open Sci.* **2026**, *13*, 251818. [CrossRef]
35. Verma, P.; Tiwari, S. Solutions of variable order mobile–immobile advection–dispersion models, their existence and uniqueness. *J. Nonlinear Convex Anal.* **2026**, *27* , 65–90. [CrossRef]
36. Hasan, M.M.A. Variable order fractional diabetes models: Numerical treatment. *Int. J. Model. Simul.* **2024**, 1–15. [CrossRef]
37. Das, P.; Das, P.; Mukherjee, S. Stochastic dynamics of Michaelis–Menten kinetics based tumor-immune interactions. *Phys. A Stat. Mech. Its Appl.* **2020**, *541*, 123603. [CrossRef]

38. Zhou, Y.; Tian, X.; Li, F. On coupling contact analysis of thermoelectric materials. *Appl. Math. Model.* **2021**, *89*, 1459–1474. [CrossRef]
39. Balakrishnan, G.P.; Chinnathambi, R.; Rihan, F.A. A fractional-order control model for diabetes with restraining and time-delay. *J. Appl. Math. Comput.* **2023**, *69*, 3403–3420. [CrossRef]

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.