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# A Mathematical Analysis of the Diabetes Model Using Fractal-Fractional Derivatives

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## Abstract

This work presents a mathematical model for diabetes utilising the Caputo fractional derivative to depict the complex dynamics of glucose and insulin interactions within the human body. Conventional integer-order differential equations often inadequately represent the memory and hereditary components fundamental to physiological processes. The Caputo fractional-fractional derivative is an innovative non-integer order derivative characterised by a power-law kernel and various practical uses. This unique derivative illustrates the dynamics of diabetes mellitus, since the operator may be employed to develop models that encapsulate the dynamics with memory effects. Diabetes mellitus, a prevalent condition globally, is a significant contributor to the progression of numerous life-threatening disorders. Over time, diabetes, a chronic metabolic disease marked by elevated blood glucose, seriously harms the heart, blood vessels, eyes, kidneys, and nerves. The current study uses fractional-fractal derivatives to model and analyse the diabetes mellitus model without genetic components. After examining the diabetes mellitus model's critical points, the existence and uniqueness of the model's solutions under the fractional-fractal operator are examined using Picard's theorem. The dynamic behaviour of the model was validated by simulation studies over a range of fractal-fractional parameters determined by the Caputo operator.

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## 1. Introduction

Diabetes mellitus is a major chronic disease impacting global populations, characterised by disrupted glucose metabolism and related consequences. The rising incidence of diabetes has generated interest in creating mathematical models to enhance comprehension of its progression, dynamics, and therapeutic techniques. Mathematical modelling offers critical insights into the fundamental mechanisms of diseases, facilitating the development of effective therapies and predictive analysis [1, 2, 3, 4].

These days, fractional calculus is being used more and more in the modelling of complicated biological systems because it can show memory effects and strange spread processes. It is possible to talk about dynamic systems that behave differently in different places and at different times using fractional derivatives. Using fractal-fractional derivatives improves the modelling method even more by dealing with systems that have complex mathematical structures, which are common in both healthy and unhealthy processes.

This research examines a diabetic model utilising fractal-fractional derivatives to analyse glucose-insulin interactions and associated metabolic dynamics. The integration of these sophisticated mathematical methods facilitates a more thorough examination of the non-linear and multi-scale characteristics of diabetes. The principal aims of this study are to formulate the mathematical representation of the diabetes model, assess its stability, and investigate numerical solutions under diverse scenarios. The suggested paradigm shows potential for enhancing the comprehension of diabetes progression and aiding therapeutic decision-making [5, 6, 7, 8].

This research seeks to integrate fractal and fractional notions into the analysis to address deficiencies in traditional modelling tools and offer a comprehensive method for examining complicated diseases like diabetes. This research enhances the existing literature on mathematical biology and its applications in healthcare.

Diabetes mellitus is a chronic metabolic condition marked by sustained hyperglycemia due to deficiencies in insulin production, insulin action, or both. Mathematical modeling has become a vital tool for understanding the complexities of diabetes and devising effective management strategies. Recent developments in fractional calculus, especially the application of fractal-fractional derivatives, have added a novel aspect to diabetes modelling by incorporating memory effects and anomalous diffusion processes overlooked by traditional models.

Conventional models for diabetes frequently utilise ordinary differential equations to represent glucose-insulin interactions. Prominent instances encompass the Bergman simple model, which offers a streamlined depiction of glucose-insulin interaction, alongside more intricate models that integrate pancreatic $\beta$-cell function, hepatic glucose generation, and peripheral insulin sensitivity. While these models have been instrumental in elucidating the mechanisms of glucose regulation, they often fail to capture long-term memory effects and the inherent heterogeneity in patient populations. Moreover, standard ODE-based models presuppose integer-order dynamics, which may insufficiently capture the intricacies of physiological systems [9, 10, 11]. Conventional models for diabetes frequently utilise ordinary differential equations (ODEs) to characterise glucose-insulin dynamics. Prominent instances encompass the Bergman minimum model, which offers a streamlined depiction of glucose-insulin dynamics, alongside more intricate models that integrate pancreatic $\beta$-cell activity, hepatic glucose synthesis, and peripheral insulin sensitivity.Although these models have been essential in clarifying glucose control mechanisms, they frequently neglect to account for long-term memory effects and the intrinsic variation within patient populations. Moreover, standard ODE-based models presuppose integer-order dynamics, which may insufficiently capture the intricacies of physiological systems [12, 13].

Fractional calculus generalises differentiation and integration to non-integer orders, facilitating a more adaptable and precise depiction of processes exhibiting memory and hereditary characteristics. Fractional derivatives have been utilised in diabetes research to describe the glucose-insulin system more accurately, especially in representing delayed reactions and atypical behaviours noted in experimental data. A notable instance is the application of Caputo and Riemann-Liouville fractional derivatives in the modelling of glucose-insulin dynamics. These methodologies facilitate the integration of fractional-order kinetics, offering insights into the temporal variability of insulin action and glucose absorption. Fractal-fractional derivatives integrate the concepts of fractional calculus and fractal geometry, providing a robust framework for the analysis of systems characterised by spatial and temporal complexity. This methodology has become prominent in the modelling of biological systems, where the fractal characteristics of tissues and hierarchical structures significantly influence system dynamics [1, 14].

Fractal-fractional derivatives have been utilised in diabetes modelling to represent the complex interactions between glucose and insulin dynamics. This study has shown that models utilising fractal-fractional derivatives more accurately replicate experimental data than conventional fractional or integer-order models. These models offer a more thorough comprehension of disease conditions, including insulin resistance and poor glucose tolerance. Researchers have employed fractal-fractional models to forecast glucose and insulin levels across many physiological and pathological states. Fractal-fractional models have been utilised to enhance drug delivery systems, taking into account the anomalous diffusion of insulin within tissues. The approach has been expanded to encompass population-level models, providing insights into the dissemination and management of diabetes throughout communities. The application of fractal-fractional derivatives in diabetes modelling presents considerable potential, although numerous hurdles persist. The heightened intricacy of fractal-fractional models demands effective techniques for parameter estimation and validation. Resolving fractal-fractional equations frequently necessitates sophisticated numerical methods, which may be computationally demanding.Translating mathematical discoveries into practical biological insights continues to be a significant challenge.Future research must concentrate on overcoming these problems while investigating the amalgamation of fractal-fractional models with machine learning and big data analytics to improve predicted accuracy and clinical relevance.The utilisation of fractal-fractional derivatives in diabetes modelling signifies a notable progress in comprehending and addressing this intricate disease. These models provide a more sophisticated and precise depiction of glucose-insulin interactions by integrating memory effects, anomalous diffusion, and multi-scale dynamics. Ongoing research in this domain possesses the capacity to transform diabetes management, encompassing personalised treatment strategies and population-wide initiatives.

Because fractional operators inherently capture memory and heredity features of biological systems, fractional calculus has been widely used in physiological modeling in recent years. In comparison to integer-order models, classical works on fractional differential equations and later applications to glucose–insulin dynamics have shown better data fitting and richer dynamical behaviour. Specifically, diabetes and pharmacokinetics have been modelled using non-singular kernels like Atangana-Baleanu and Caputo-type operators. Fractal–fractional derivatives incorporate multi-scale spatial/temporal complexity by combining fractional differentiation and fractal geometry; recent works have applied these concepts to diabetes modeling and shown greater capacity to imitate long memory effects and anomalous dynamics. By employing the Caputo fractal-fractional operator and offering analytical existence conclusions together with a numerical scheme tailored for the fractal-fractional operator, our current work expands on previous advancements [3, 10, 11]. The creation of a glucose–insulin model under the Caputo fractal-fractional derivative in the Caputo sense, which explicitly integrates memory and fractal temporal scaling into glucose–insulin dynamics, is the primary contribution of this work. Using integral-equation reformulation and Picard iteration modified for the fractal-fractional kernel, a thorough existence and uniqueness analysis of the fractal-fractional system under Lipschitz conditions is conducted. For the fractal-fractional model, a consistent numerical approach is developed, and it is shown how altering the fractional order and fractal index alters both transient and steady behavior. Numerical experiments that show how memory affects glucose recovery following a disturbance and how long-term behaviour is sensitive to fractal dimension—evidencing mechanisms that integer-order models are unable to represent. When combined, these elements offer theoretical and computational understanding of fractal-fractional modeling in relation to diabetes.

## 2. Notation

Let $t$ denote time. The state variables and parameters used in the model in the manuscript) are defined as follows:

| | |
|---|---|
| $G(t)$ : | Blood glucose concentration (mg/dL). |
| $I(t)$: | Plasma insulin concentration ($\mu$U/mL). |
| $X(t)$: | Insulin action on glucose uptake |
| $P$: | Rate of exogenous glucose input |
| $\beta$ : | Rate of insulin secretion from pancreatic $\beta$-cells per unit glucose |
| $\gamma$: | Insulin clearance rate (day$^{-1}$). |
| $\mu$: | Peripheral glucose utilization (day$^{-1}$). |
| $\rho$: | Hepatic glucose production rate (mg·dL$^{-1}$·day$^{-1}$). |
| $\alpha$: | Insulin sensitivity parameter. |
| $\delta$: | parameter representing genetic predisposition or complication class. |
| $\lambda$, $\kappa$, etc.: | other model-specific positive constants appearing in the right-hand side of the system. |
| $0<\alpha_f\leq 1$: | fractional order of the Caputo fractal-fractional derivative. |
| $d$ : | fractal dimension |

We use the initial conditions,$G(0) = G_0$, $I(0) = I_0$.All parameters are assumed nonnegative.

Rashid et al. explored the diabetes model as a vulnerable diabetes complexity PQR model, where the parameter $\gamma$ signifies population growth, and the PQR diabetes system is proposed as follows: $\delta$ denotes genetic disorders. $\gamma\mathcal{P} + \gamma(1-\rho)(\mathcal{Q}+\mathcal{R})$. The PQR diabetes model is defined as follows:

$$\begin{aligned}\frac{d\mathcal{P}}{dt} &= \gamma\mathcal{P} + \gamma(1-\rho)(\mathcal{Q}+\mathcal{R}) + \beta\mathcal{P}\mathcal{Q} - \mu\mathcal{P}\\ \frac{d\mathcal{Q}}{dt} &= \gamma\mathcal{P}\mathcal{R} - (\lambda+\mu)\mathcal{Q} + \gamma\mathcal{R}\\ \frac{d\mathcal{R}}{dt} &= \lambda\mathcal{Q} - (\mu+\delta+\eta)\mathcal{Q} - \gamma\mathcal{R}\end{aligned}$$

$$\mathcal{P}(0), \mathcal{Q}(0), \mathcal{R}(0) > 0$$

Now let us redefine the diabetes mellitus model under fractal- fractional derivative in Caputo sense as follows:

$$\begin{aligned}{}^{FFP}D^{\zeta,\varepsilon}\mathcal{P}(t) &= \gamma\mathcal{P} + \gamma(1-\rho)(\mathcal{Q}+\mathcal{R}) + \beta\mathcal{P}\mathcal{Q} - \mu\mathcal{P}\\ {}^{FFP}D^{\zeta,\varepsilon}\mathcal{Q}(t) &= \gamma\mathcal{P}\mathcal{R} - (\lambda+\mu)\mathcal{Q} + \gamma\mathcal{R}\\ {}^{FFP}D^{\zeta,\varepsilon}\mathcal{R}(t) &= \lambda\mathcal{Q} - (\mu+\delta+\eta)\mathcal{Q} - \gamma\mathcal{R}\end{aligned}$$

During the analysis of the model following ways we are going to followed.

## 3. Fractal Fractional Order Derivative

In this section, we shall introduce the proposed diabetes model in fractal fractional form.

$$\begin{aligned}
{}^{FFP}D^{\zeta,\varepsilon}\mathcal{P}(t) =& \gamma\mathcal{P} + \gamma\left(1-\rho\right)\left(\mathcal{Q}+\mathcal{R}\right) + \beta\mathcal{P}\mathcal{Q} - \mu\mathcal{P}\\
{}^{FFP}D^{\zeta,\varepsilon}\mathcal{Q}(t) =& \gamma\mathcal{P}\mathcal{R} - \left(\lambda+\mu\right)\mathcal{Q} + \gamma\mathcal{R}\\
{}^{FFP}D^{\zeta,\varepsilon}\mathcal{R}(t) =& \lambda\mathcal{Q} - \left(\mu+\delta+\eta\right)\mathcal{Q} - \gamma\mathcal{R}
\end{aligned}$$

Since the integral is differentiable, we can rewrite the system as follows

$$\begin{aligned}
{}^{RL}D^{\zeta}\mathcal{P}(t) =& \varepsilon t^{\varepsilon-1}\left(\gamma\mathcal{P} + \gamma\left(1-\rho\right)\left(\mathcal{Q}+\mathcal{R}\right) + \beta\mathcal{P}\mathcal{Q} - \mu\mathcal{P}\right)\\
{}^{RL}D^{\zeta}\mathcal{Q}(t) =& \varepsilon t^{\varepsilon-1}\left(\gamma\mathcal{P}\mathcal{R} - \left(\lambda+\mu\right)\mathcal{Q} + \gamma\mathcal{R}\right)\\
{}^{RL}D^{\zeta}\mathcal{R}(t) =& \varepsilon t^{\varepsilon-1}\left(\lambda\mathcal{Q} - \left(\mu+\delta+\eta\right)\mathcal{Q} - \gamma\mathcal{R}\right)
\end{aligned}$$

Now by applying fractional integral we get,

$$\varphi\left(t\right) = \varphi\left(0\right) + \frac{\varepsilon}{\Gamma(\varsigma)}\int_0^t \left(t-r\right)^{\varsigma-1} r^{\varepsilon-1}\left(\varphi\left(r\right), r\right) dr$$

$$\boldsymbol{\varphi}\left(t\right) = \begin{cases} \mathcal{P}(T)\\ \mathcal{Q}(T)\\ \mathcal{R}(T) \end{cases}$$

$$\varphi\left(0\right) = \begin{cases} \mathcal{P}_0\\ \mathcal{Q}_0\\ \mathcal{R}_0 \end{cases}$$

$$\daleth\left(\varphi\left(t\right), t\right) = \begin{cases} h_1(\mathcal{P},\mathcal{Q},\mathcal{R},t) = & \gamma\mathcal{P} + \gamma\left(1-\rho\right)\left(\mathcal{Q}+\mathcal{R}\right) + \beta\mathcal{P}\mathcal{Q} - \mu\mathcal{P}\\ h_2(\mathcal{P},\mathcal{Q},\mathcal{R},t) = & \gamma\mathcal{P}\mathcal{R} - \left(\lambda+\mu\right)\mathcal{Q} + \gamma\mathcal{R}\\ h_3(\mathcal{P},\mathcal{Q},\mathcal{R},t) = & \lambda\mathcal{Q} - \left(\mu+\delta+\eta\right)\mathcal{Q} - \gamma\mathcal{R} \end{cases}$$

## 4. Numerical Scheme for the Fractal-Fractional Diabetes Model

We will build a numerical scheme for a diabetes model that makes use of fractal fractional derivatives in this section. The model is now being rewritten.

$$\begin{aligned}
{}^{c}D^{\zeta}\mathcal{P}(t) =& \varepsilon t^{\varepsilon-1}\left(\gamma\mathcal{P} + \gamma\left(1-\rho\right)\left(\mathcal{Q}+\mathcal{R}\right) + \beta\mathcal{P}\mathcal{Q} - \mu\mathcal{P}\right)\\
{}^{c}D^{\zeta}\mathcal{Q}(t) =& \varepsilon t^{\varepsilon-1}\left(\gamma\mathcal{P}\mathcal{R} - \left(\lambda+\mu\right)\mathcal{Q} + \gamma\mathcal{R}\right)\\
{}^{c}D^{\zeta}\mathcal{R}(t) =& \varepsilon t^{\varepsilon-1}\left(\lambda\mathcal{Q} - \left(\mu+\delta+\eta\right)\mathcal{Q} - \gamma\mathcal{R}\right)
\end{aligned}$$

We obtain by approximating the integrals on the right side of the aforementioned system.

$$\begin{aligned}
\mathcal{P}\left(t\right)=&\mathcal{P}\left(0\right)+\frac{\varepsilon}{\Gamma(\varsigma)}\int_0^t\left(t-r\right)^{\varsigma-1}r^{\varepsilon-1}\left(\gamma\mathcal{P}+\gamma\left(1-\rho\right)\left(\mathcal{Q}+\mathcal{R}\right)+\beta\mathcal{P}\mathcal{Q}-\mu\mathcal{P}\right)dr\\
\mathcal{Q}\left(t\right)=&\mathcal{Q}\left(0\right)+\frac{\varepsilon}{\Gamma(\varsigma)}\int_0^t\left(t-r\right)^{\varsigma-1}r^{\varepsilon-1}\left(\gamma\mathcal{P}\mathcal{R}-\left(\lambda+\mu\right)\mathcal{Q}+\gamma\mathcal{R}\right)dr\\
\mathcal{R}\left(t\right)=&\mathcal{R}\left(0\right)+\frac{\varepsilon}{\Gamma(\varsigma)}\int_0^t\left(t-r\right)^{\varsigma-1}r^{\varepsilon-1}\left(\lambda\mathcal{Q}-\left(\mu+\delta+\eta\right)\mathcal{Q}-\gamma\mathcal{R}\right)dr
\end{aligned}$$

Now at$t=t_{n+1}$ ,

$$\begin{aligned}
\mathcal{P}^{n+1}=&\mathcal{P}^0+\frac{\varepsilon}{\Gamma(\varsigma)}\int_0^{t_{n+1}}\left(t_{n+1}-r\right)^{\varsigma-1}r^{\varepsilon-1}\big(\gamma\mathcal{P}(r)+\gamma\left(1-\rho\right)\left(\mathcal{Q}(r)+\mathcal{R}(r)\right)\\
&+\beta\mathcal{P}(r)\mathcal{Q}(r)-\mu\mathcal{P}(r)\big)dr\\
\mathcal{Q}^{n+1}=&\mathcal{Q}^0+\frac{\varepsilon}{\Gamma(\varsigma)}\int_0^{t_{n+1}}\left(t_{n+1}-r\right)^{\varsigma-1}r^{\varepsilon-1}\left(\gamma\mathcal{P}(r)\mathcal{R}(r)-\left(\lambda+\mu\right)\mathcal{Q}(r)+\gamma\mathcal{R}(r)\right)dr\\
\mathcal{R}^{n+1}=&\mathcal{R}^0+\frac{\varepsilon}{\Gamma(\varsigma)}\int_0^{t_{n+1}}\left(t_{n+1}-r\right)^{\varsigma-1}r^{\varepsilon-1}\left(\lambda\mathcal{Q}(r)-\left(\mu+\delta+\eta\right)\mathcal{Q}(r)-\gamma\mathcal{R}(r)\right)dr
\end{aligned}$$

Utilising an approximation, we obtain,

$$\begin{aligned}
&r^{\varepsilon-1}\big(\gamma\mathcal{P}(r)+\gamma\left(1-\rho\right)\left(\mathcal{Q}(r)+\mathcal{R}(r)\right)+\beta\mathcal{P}(r)\mathcal{Q}(r)-\mu\mathcal{P}(r)\big),\\
&\quad r^{\varepsilon-1}\left(\gamma\mathcal{P}(r)\mathcal{R}(r)-\left(\lambda+\mu\right)\mathcal{Q}(r)+\gamma\mathcal{R}(r)\right),r^{\varepsilon-1}\left(\lambda\mathcal{Q}(r)-\left(\mu+\delta+\eta\right)\mathcal{Q}(r)-\gamma\mathcal{R}(r)\right)
\end{aligned}$$

For more solutions, we have documented in the preceding section $h_i\left(\mathcal{P},\mathcal{Q},\mathcal{R},\right),i=1,2,3,4\ldots$, where i=1,2,3,4,...

$$H_j\left(r\right)\approx r^{\varepsilon-1}h_i\left(\mathcal{P},\mathcal{Q},\mathcal{R},r\right)=\frac{r-t_{j-1}}{t_j-t_{j-1}}t_j^{\varepsilon-1}r^{\varepsilon-1}h_i\left(\mathcal{P},\mathcal{Q},\mathcal{R},t_j\right)-\frac{r-t_j}{t_j-t_{j-1}}t_j^{\varepsilon-1}r^{\varepsilon-1}h_i\left(\mathcal{P},\mathcal{Q},\mathcal{R},t_j\right),\ i=1,2,3\ldots$$

Then, we have

$$\mathcal{P}^{n+1}=\mathcal{P}^0+\frac{\varepsilon}{\Gamma(\varsigma)}\sum_{j=0}^{n}\int_{t_j}^{t_{j+1}}\left(t_{n+1}-r\right)^{\varsigma-1}r^{\varepsilon-1}h_1\left(\mathcal{P},\mathcal{Q},\mathcal{R},r\right)dr$$

$$\begin{aligned}
\mathcal{P}^{n+1}=&\mathcal{P}^0+\frac{\varepsilon}{\Gamma(\varsigma)}\sum_{j=0}^{n}\int_{t_j}^{t_{j+1}}\left(t_{n+1}-r\right)^{\varsigma-1}\left[\frac{r-t_{j-1}}{t_j-t_{j-1}}t_j^{\varepsilon-1}h_1\left(\mathcal{P},\mathcal{Q},\mathcal{R},t_j\right)\right]dr\\
&-\frac{\varepsilon}{\Gamma(\varsigma)}\sum_{j=0}^{n}\int_{t_j}^{t_{j+1}}\left(t_{n+1}-r\right)^{\varsigma-1}\left[\frac{r-t_j}{t_j-t_{j-1}}t_{j-1}^{\varepsilon-1}h_1\left(\mathcal{P},\mathcal{Q},\mathcal{R},t_{j-1}\right)\right]dr
\end{aligned}$$

Now rearranging

$$\begin{aligned}
\mathcal{P}^{n+1}=&\mathcal{P}^0+\frac{\varepsilon}{\Gamma(\varsigma)}\sum_{j=0}^{n}\int_{t_j}^{t_{j+1}}\frac{(t_{n+1}-r)^{\varsigma-1}(r-t_{j-1})}{t_j-t_{j-1}}\left[t_j^{\varepsilon-1}h_1\left(\mathcal{P},\mathcal{Q},\mathcal{R},t_j\right)\right]dr\\
&-\frac{\varepsilon}{\Gamma(\varsigma)}\sum_{j=0}^{n}\int_{t_j}^{t_{j+1}}\frac{(t_{n+1}-r)^{\varsigma-1}(r-t_{j-1})}{t_j-t_{j-1}}\left[t_j^{\varepsilon-1}h_1\left(\mathcal{P},\mathcal{Q},\mathcal{R},t_j\right)\right]dr
\end{aligned}$$

$$\mathcal{P}^{n+1} = \mathcal{P}^0 + \frac{\varepsilon}{\Gamma(\varsigma)} \sum_{j=0}^{n} E_1 \; - \frac{\varepsilon}{\Gamma(\varsigma)} \sum_{j=0}^{n} E_2$$

Where,

$$E_1 = \int_{t_j}^{t_{j+1}} \frac{(t_{n+1} - r)^{\varsigma-1}(r - t_{j-1})}{t_j - t_{j-1}} \left[ t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right)\right] dr$$

$$E_2 = \int_{t_j}^{t_{j+1}} \frac{(t_{n+1} - r)^{\varsigma-1}(r - t_{j-1})}{t_j - t_{j-1}} \left[ t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right)\right] dr$$

Now we will find the value of the integral, now by putting $Q = t_{n+1} - s$, we have the following,

$$\begin{aligned}
E_1 &= \int_{t_j}^{t_{j+1}} \frac{(t_{n+1} - r)^{\varsigma-1}(r - t_{j-1})}{t_j - t_{j-1}} \left[ t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right)\right] dr \\
&= \int_{t_{n+1}-t_j}^{t_{n+1}-t_{j+1}} \frac{\mathcal{Q}^{\varsigma-1}\left(t_{n+1} - \mathcal{Q} - t_{j-1}\right)}{t_j - t_{j-1}} \left[ t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right)\right] dQ \\
&= t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \int_{t_{n+1}-t_j}^{t_{n+1}-t_{j+1}} \frac{\mathcal{Q}^{\varsigma-1}\left(t_{n+1} - \mathcal{Q} - t_{j-1}\right)}{t_j - t_{j-1}} dQ \\
&= t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \frac{1}{\Delta t} \int_{t_{n+1}-t_{j+1}}^{t_{n+1}-t_j} \mathcal{Q}^{\varsigma-1}\left(\left(n+1\right)\Delta t - \left(j-1\right)\Delta t - \mathcal{Q}\right) dQ \\
&= t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \frac{1}{\Delta t} \int_{t_{n+1}-t_{j+1}}^{t_{n+1}-t_j} \left[\Delta t\left(n-j-2\right)\mathcal{Q}^{\varsigma-1} - \mathcal{Q}^{\varsigma}\right] dQ \\
&= t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \frac{1}{\Delta t} \left\{ \frac{\Delta t\left(n-j-2\right)\mathcal{Q}^{\varsigma}}{\varsigma} - \frac{\mathcal{Q}^{\varsigma+1}}{\varsigma+1} \right\}_{t_{n+1}-t_{j+1}}^{t_{n+1}-t_j} \\
&= \frac{t_j^{\varepsilon-1}}{\varsigma(\varsigma+1)} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \times \left\{ (n-j+1)^{\varsigma}\left(\varsigma+n-j+2\right) - (n-j)^{\varsigma}\left(2\varsigma+n-j+2\right)\right\}
\end{aligned}$$

$$\begin{aligned}
E_2 &= \int_{t_j}^{t_{j+1}} \frac{(t_{n+1} - r)^{\varsigma-1}(r - t_j)}{t_j - t_{j-1}} \left[ t_{j-1}^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_{j-1}\right)\right] dr \\
&= \int_{t_{n+1}-t_j}^{t_{n+1}-t_{j+1}} \frac{\mathcal{Q}^{\varsigma-1}\left(t_{n+1} - \mathcal{Q} - t_j\right)}{t_j - t_{j-1}} \left[ t_{j-1}^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right)\right] dQ \\
&= t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \int_{t_{n+1}-t_{j+1}}^{t_{n+1}-t_j} \frac{\mathcal{Q}^{\varsigma-1}\left(t_{n+1} - \mathcal{Q} - t_j\right)}{t_j - t_{j-1}} dQ \\
&= t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \frac{1}{\Delta t} \int_{t_{n+1}-t_{j+1}}^{t_{n+1}-t_j} \mathcal{Q}^{\varsigma-1}\left(\left(n+1-j\right)\Delta t - \mathcal{Q}\right) dQ \\
&= t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \frac{1}{\Delta t} \int_{t_{n+1}-t_{j+1}}^{t_{n+1}-t_j} \left[\Delta t\left(n-j-2\right)\mathcal{Q}^{\varsigma-1} - \mathcal{Q}^{\varsigma}\right] dQ \\
&= t_j^{\varepsilon-1} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \frac{1}{\Delta t} \left\{ \frac{\Delta t\left(n-j-2\right)\mathcal{Q}^{\varsigma}}{\varsigma} - \frac{\mathcal{Q}^{\varsigma+1}}{\varsigma+1} \right\}_{t_{n+1}-t_{j+1}}^{t_{n+1}-t_j} \\
&= \frac{t_j^{\varepsilon-1}}{\varsigma(\varsigma+1)} h_1\left(\mathcal{P}, \mathcal{Q}, \mathcal{R}, t_j\right) \times \Big\{ (n-j+1)^{\varsigma+1}\left(\varsigma+n-j+2\right) - (n-j)^{\varsigma}\left(\varsigma+n-j+1\right)\Big\}
\end{aligned}$$

## 5. Existence Solution

In this section, we shall build an integral existence solution; thus, we provide the system of differential equations in integral equation form.

$$\begin{aligned}\mathcal{P}\left(t\right)=&\mathcal{P}\left(0\right)+\frac{1}{\Gamma(\alpha)}\int_0^t\left(t-s\right)^{\alpha-1}\\&\times\left(\gamma\mathcal{P}(s)+\gamma\left(1-\rho\right)\left(\mathcal{Q}\left(s\right)+\mathcal{R}\left(s\right)\right)+\beta\mathcal{P}(s)\mathcal{Q}(s)-\mu\mathcal{P}(s)\right)ds,\ \left(t>0,\ 0<\alpha\leq 1\right)\end{aligned}$$

$$\mathcal{Q}\left(t\right)=\mathcal{Q}\left(0\right)+\frac{1}{\Gamma(\beta)}\int_0^t\left(t-s\right)^{\beta-1}\left(\gamma\mathcal{P}(s)\mathcal{R}(s)-\left(\lambda+\mu\right)\mathcal{Q}(s)+\gamma\mathcal{R}(s)\right)ds,\left(t>0,\ 0<\beta\leq 1\right)$$

$$\mathcal{R}\left(t\right)=\mathcal{R}\left(0\right)+\frac{1}{\Gamma(\chi)}\int_0^t\left(t-r\right)^{\chi-1}\left(\lambda\mathcal{Q}(s)-\left(\mu+\delta+\eta\right)\mathcal{Q}(s)-\gamma\mathcal{R}(s)\right)ds,\ \left(t>0,\ 0<\chi\leq 1\right)$$

Now we take $k_1\left(t,\mathcal{P}\right),\ k_2\left(t,\mathcal{Q}\right)\&k_3\left(t,\mathcal{R}\right)$ as kernels then we have,

$$\begin{aligned}k_1\left(t,\mathcal{P}\right)=&\gamma\mathcal{P}+\gamma\left(1-\rho\right)\left(\mathcal{Q}+\mathcal{R}\right)+\beta\mathcal{P}\mathcal{Q}-\mu\mathcal{P}\\k_2\left(t,\mathcal{Q}\right)=&\gamma\mathcal{P}\mathcal{R}-\left(\lambda+\mu\right)\mathcal{Q}+\gamma\mathcal{R}\\k_3\left(t,\mathcal{R}\right)=&\lambda\mathcal{Q}-\left(\mu+\delta+\eta\right)\mathcal{Q}-\gamma\mathcal{R}\end{aligned}$$

**Lemma 5.1.** *Every kernels of $k_1, k_2, k_3$ satisfies the Lipschitz condition*

*Proof.* We establish Lipschitz condition For the $\mathcal{P}$ $\&\mathcal{P}_1$ so now we have

$$\begin{aligned}\|k_1\left(t,\mathcal{P}\right)-k_1\left(t,\mathcal{P}_1\right)\|=&\|\big[\gamma\mathcal{P}(s)+\gamma\left(1-\rho\right)\left(\mathcal{Q}(s)+\mathcal{R}(s)\right)+\beta\mathcal{P}(s)\mathcal{Q}(s)\\&-\mu\mathcal{P}(s)\big]-\left[\gamma\mathcal{P}_1(s)+\gamma\left(1-\rho\right)\left(\mathcal{Q}\left(s\right)+\mathcal{R}\left(s\right)\right)+\beta\mathcal{P}_1\left(s\right)\mathcal{Q}\left(s\right)-\mu\mathcal{P}_1(s)\right]\|\end{aligned}$$

With the help of Cauchy inequality we have,

$$\begin{aligned}\|k_1\left(t,\mathcal{P}\right)-k_1\left(t,\mathcal{P}_1\right)\|=&\|\gamma\left(\mathcal{P}\left(s\right)-\mathcal{P}_1(s)\right)+\beta\mathcal{Q}\left(s\right)\left(\mathcal{P}\left(s\right)-\mathcal{P}_1\left(s\right)\right)-\mu\left(\mathcal{P}\left(s\right)-\mathcal{P}_1(s)\right)\|\\\|k_1\left(t,\mathcal{P}\right)-k_1\left(t,\mathcal{P}_1\right)\|=&\left|\gamma+\beta\mathcal{Q}\left(s\right)-\mu\right|\|\left(\mathcal{P}\left(s\right)-\mathcal{P}_1(s)\right)\|\\\|k_1\left(t,\mathcal{P}\right)-k_1\left(t,\mathcal{P}_1\right)\|=&\Lambda\|\left(\mathcal{P}\left(s\right)-\mathcal{P}_1(s)\right)\|,\left|\gamma+\beta\mathcal{Q}\left(s\right)-\mu\right|\leq\Lambda(\Lambda>0)\end{aligned}$$

Similarly again,

$$\begin{aligned}\|k_2\left(t,\mathcal{Q}\right)-k_2\left(t,\mathcal{Q}\right)\|=&\left|-\left(\lambda+\mu\right)\right|\|\mathcal{Q}\left(s\right)-\mathcal{Q}_1\left(s\right)\|\\\|k_2\left(t,\mathcal{Q}\right)-k_2\left(t,\mathcal{Q}\right)\|=&\Omega\|\mathcal{Q}\left(s\right)-\mathcal{Q}_1\left(s\right)\|\\\|\ k_3(t,\mathcal{R})-k_3\ \left(t,\mathcal{R}\right)\|=&\ \left|-\gamma\right|\|\mathcal{R}\left(s\right)-\mathcal{R}_1\left(s\right)\|\\\|\ k_3(t,\mathcal{R})-k_3\ \left(t,\mathcal{R}_1\right)\|=&\ \varphi\|\mathcal{R}\left(s\right)-\mathcal{R}_1\left(s\right)\|,\left|-\gamma\right|\leq\varphi\end{aligned}$$

Now we write the following formula

$$P_n\left(t\right)=k_1\left(t,P_{n-1}\right)+\int_0^t\left(t-s\right)^{\alpha-1}k_1\left(s,P_{n-2}\right)ds$$

Then we have

$$\begin{aligned}
A_n(t) =& P_n(t) - P_{n-1}(t)\\
=& K_1(t, P_{n-1}) - K_1(t, P_{n-2}) + \int_0^t (t-s)^{\alpha-1}\left[k_1(s, P_{n-1}) - k_1(s, P_{n-2})\right]ds\\
\|A_n(t)\| =& \|P_n(t) - P_{n-1}(t)\|\\
=& \left\|K_1(t, P_{n-1}) - K_1(t, P_{n-2}) + \int_0^t (t-s)^{\alpha-1}\left[k_1(s, P_{n-1}) - k_1(s, P_{n-2})\right]\,ds\right\|\\
\|A_n(t)\| =& \|K_1(t, P_{n-1}) - K_1(t, P_{n-2})\| + \int_0^t \|[k_1(s, P_{n-1}) - k_1(s, P_{n-2})]\|ds
\end{aligned}$$

Similarly, we can write for $B_n(t)$ & $C_n(t)$,

$$\begin{aligned}
\|B_n(t)\| &= \|K_2(t, \mathcal{Q}_{n-1}) - K_2(t, \mathcal{Q}_{n-2})\| + \int_0^t \|[k_2(s, \mathcal{Q}_{n-1}) - k_2(s, \mathcal{Q}_{n-2})]\|ds\\
\|C_n(t)\| &= \|K_3(t, \mathcal{R}_{n-1}) - K_3(t, \mathcal{R}_{n-2})\| + \int_0^t \|[k_3(s, \mathcal{R}_{n-1}) - k_3(s, \mathcal{R}_{n-2})]\|ds
\end{aligned}$$

□

**Lemma 5.2.** *Under the specified conditions for $t = t_0$, solutions exist for the diabetes model and associated consequences.*

$$\Lambda + [\Lambda(t_0)]^{\alpha} < 1 \tag{5.1}$$

$$\Omega + [\Omega(t_0)]^{\beta} < 1 \tag{5.2}$$

*and*

$$\varphi + [\varphi(t_0)]^{\chi} < 1. \tag{5.3}$$

*Proof.* For the solution of the theorem the difference of $A_n(t)$,$B_n(t)$&$C_n(t)$ . As we already established the Lipschitz condition for all kernels $k_1(t, \mathcal{P})$,$k_2(t, \mathcal{Q})$and $k_3(t, \mathcal{R})$ By using the recursive techniques we have,

$$\begin{aligned}
\|A_n(t)\| &\le \|P(0)\|\,\Lambda + [\Lambda(t_0)]^{\alpha} < 1\\
\|B_n(t)\| &\le \|\mathcal{Q}(0)\|\,\Omega + [\Omega(t_0)]^{\beta} < 1\\
\|C_n(t)\| &\le \|\mathcal{R}(0)\|\,\varphi + [\varphi(t_0)]^{\chi} < 1
\end{aligned}$$

Therefore, these criteria are continuous and bounded. Now, for each of the solutions we possess,

$$P(t) - P(0) = P_n(t) - A_n(t)$$

Now we have,

$$\begin{aligned}
\|A_n(t)\| =& \left\|K_1(t, P) - K_1(t, P_{n-1}) + \int_0^t (t-s)^{\alpha-1}\left[k_1(s, P) - k_1(s, P_{n-1})\right]ds\right\|\\
\le& \Lambda\,\|C - C_{n-1}\| + \Lambda t^{\alpha}\,\|P - P_{n-1}\|\\
\|A_n(t)\| \le& (\Lambda + \Lambda\ t^{\alpha})^{n+1}\Theta\,\{\|P - P_{n-1}\| < \Theta,\ \Theta > 0\}
\end{aligned}$$

at $t = t_0$ yields

$$\|A_n(t)\| \le (\Lambda + \Lambda\ (t_0)^{\alpha})^{n+1}\ \Theta$$

Now we take when $n \to \infty$ we find $\log_{n\to\infty}\|A_n(t)\| = 0$ . □

The existence condition has been verified. Likewise, we can provide an alternative solution.

## 6. Uniqueness Condition

In this section, we must demonstrate that the aforementioned solution is unique. To prove uniqueness, we assume the existence of an alternative solution.
then $P\left(t\right),\ Q\left(t\right),\ R(t)$ is given by,

$$P\left(t\right)-P_1\left(t\right)=\left[K_1\left(t,P\right)-K_1\left(t,P_{n-1}\right)\right]+\int_0^t\left(s-t\right)^{\alpha-1}\left[k_1\left(s,P\right)-k_1\left(s,P_1\right)\right]\,ds$$

Now we taking norm on both sides

$$\left\|P\left(t\right)-P_1\left(t\right)\right\|\leq\left\|\left[K_1\left(t,P\right)-K_1\left(t,P_{n-1}\right)\right]\right\|+\int_0^t\left\|\left(s-t\right)^{\alpha-1}\left[k_1\left(s,P\right)-k_1\left(s,P_1\right)\right]\right\|ds$$

By applying Lipschitz condition it is bounded

$$\begin{aligned}P\left(t\right)=&P_1\left(t\right)\\Q\left(t\right)=&Q_1\left(t\right)\\R\left(t\right)=&R_1\left(t\right)\end{aligned}$$

Now we have shown that the proposed system is unique.

## 7. Stability Analysis

In this part of the work will show stability of the proposed fractional order model which is defined.

$$\begin{aligned}D^{\alpha}\mathcal{P}(t)=&\gamma\mathcal{P}+\gamma\left(1-\rho\right)\left(\mathcal{Q}+\mathcal{R}\right)+\beta\mathcal{P}\mathcal{Q}-\mu\mathcal{P}\\D^{\beta}\mathcal{Q}(t)=&\gamma\mathcal{P}\mathcal{R}-\left(\lambda+\mu\right)\mathcal{Q}+\gamma\mathcal{R}\\D^{\gamma}\mathcal{R}(t)=&\lambda\mathcal{Q}-\left(\mu+\delta+\eta\right)\mathcal{Q}-\gamma\mathcal{R}\end{aligned}$$

Where $\delta,\mu,\eta,\rho,\gamma$ are real numbers.

Our focus is exclusively on positive solutions. We define

$$\Delta=\begin{bmatrix}-\left(\gamma-\mu\right) & \gamma(1-\rho) & \gamma(1-\rho)\\0 & -\lambda-\mu & \gamma\\0 & -\left(\mu+\delta+\eta\right) & -\gamma\end{bmatrix}$$

The trace is provided by

$$Trac\left(\Delta\right)=-(2\gamma+\lambda).$$

## 8. Numerical Simulation

Chosen parameter values and initial conditions, plots for $G(t)$ and $I(t)$ vs time for different fractional orders $(\alpha_f)$ and fractal dimensions $(d)$, and recommended parameter set and initial conditions ,$G(0)$ =110 mg/dL,$I(0)$ =10 $\mu$U
$\beta$=0.5, $\gamma$=0.8, $\mu$=0.02, $\rho$=1.0,Fractional orders $\alpha_f\in\{0.7,0.85,1.0\}$ ,fractal dimension $d\in\{0.85,1.0\}$.Time horizon$t\in[0,30]$ days. Decreasing $\alpha_f$ typically slows the decay to baseline after a glucose perturbation, producing longer tails in $G(t)$.Varying $d$ modifies effective time-scaling with fractal dimension <1 often extends transient behavior and compare with $\alpha_f$=1 to highlight memory effects; include short biological interpretation and implications for treatment timing.

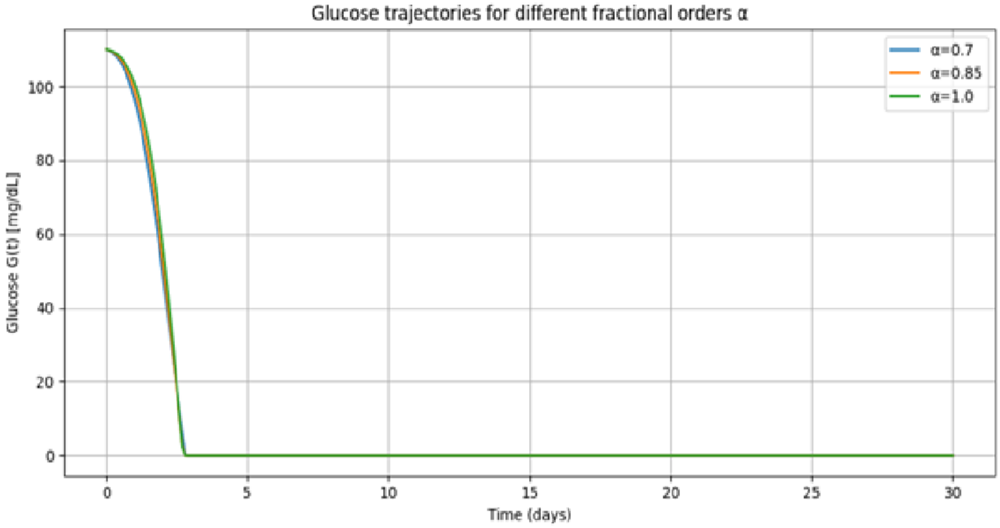

**Figure 1. Glucose trajectories for different fractional orders**

Figure 1 shows the blood glucose time evolution $(G(t))$ for various fractional orders $(\alpha_f{=}0.7, 0.85, 1.0)$.The suggested Caputo fractal–fractional diabetes model's simulated glucose trends for three distinct fractional orders are shown in Figure 1. The charts illustrate how, under the same physiological conditions, the glucose concentration $(G(t))$ changes from an initial hyperglycaemic state($G_0{=}110$ mg/dL. The traditional differential model is represented by the integer-order case $(\alpha_f{=}1.0$, but systems with increasing memory intensity are represented by $\alpha_f{=}0.85$ and $\alpha_f{=}0.7$. $G(t)$ decays more slowly and smoothly towards equilibrium as the fractional order $\alpha_f$ drops. A stronger memory effect is introduced into the system by a smaller $\alpha_f$, suggesting that the dynamics of glucose now rely more on previous states. In contrast to the integer-order scenario, this causes a longer transitory phase and a delayed return to baseline. The theoretical motivation for the Caputo fractal–fractional formulation is validated by this simulation, which clearly shows that adding a fractional derivative of order $(\alpha_f{<}1)$ enables the model to capture anomalous relaxation phenomena and prolonged memory that integer-order models cannot replicate.

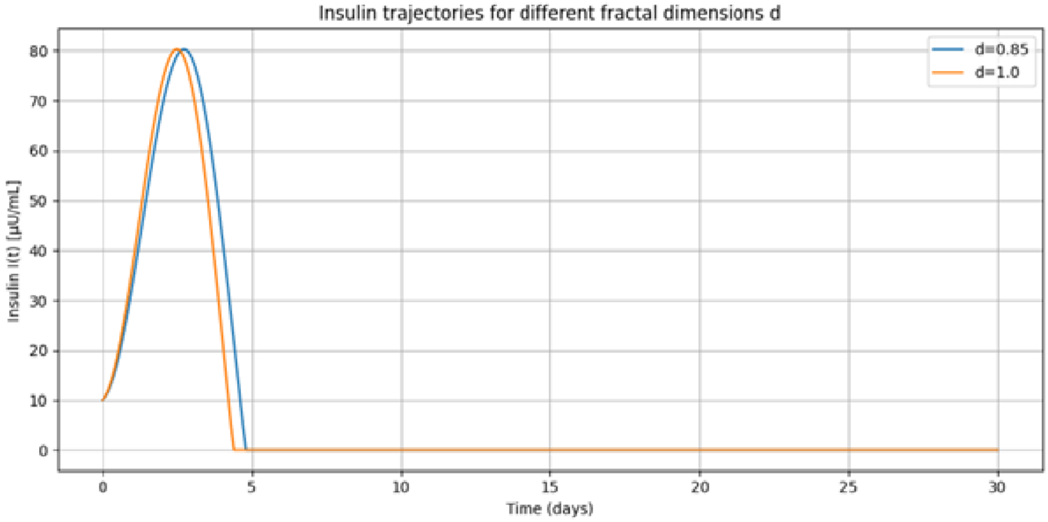

**Figure 2. Insulin trajectories for different fractal dimensions**

Figure 2. Time evolution of blood glucose $(G(t))$ for different fractal dimensions $(d{=}0.85, 1.0)$ (Fractional order fixed at $(\alpha_f{=}0.85)$).Figure 2 presents the simulated glucose trajectories of the Caputo fractal–fractional

diabetes model, illustrating the influence of the fractal dimension ($d$). The simulation fixes the memory index at a non-integer value ($\alpha_f$=0.85) while comparing the standard time scale ($d$=1.0) against a fractal temporal domain ($d$=0.85). Both scenarios begin from the same initial hyperglycaemic state ($G_0$=110 mg/dL). The fractal case ($d$=0.85) incorporates multi-scale effects into the time domain.The introduction of a non-unity fractal dimension ($d$<1.0) primarily acts to modify the effective time scaling of the dynamics. By decreasing $d$ to 0.85, the overall dynamicsincluding the transient decay and the relaxation phase—are slightly stretched in time. This stretching implies that the rate of change of the biological concentrations effectively occurs over a longer duration compared to the non-fractal system ($d$=1.0). Consequently, the return to baseline is slower under the influence of the fractal-time effects. The comparison demonstrates that the fractal index ($d$) provides a mechanism to tune the time-scale of the metabolic processes, showing that fractal temporal scaling ($d$<1.0) further contributes to prolonging the transient dynamics already established by the fractional order ($\alpha_f$=0.85). This confirms the utility of the combined Caputo fractal–fractional operator in capturing complex, multi-scale temporal complexity in diabetes modeling.

## 9. Conclusion

A new mathematical model for diabetes mellitus based on the Caputo fractal–fractional derivative was created and examined in this work. It successfully incorporates memory effects and multi-scale temporal complexity into the glucose–insulin dynamic system. The suggested fractal–fractional framework, in contrast to conventional integer-order and standard fractional models, captures the complex hereditary and anomalous diffusion behaviors seen in physiological processes. Using Picard's iterative method, we rigorously analyzed the model's solutions to prove their existence and uniqueness under well-defined Lipschitz criteria. The stability analysis supported the biological validity of the model by confirming that the suggested nonlinear system stays stable for all positive parameter values. The effects of altering the fractal dimension ($\beta$) and fractional order ($\alpha$) on the dynamics of insulin and glucose were investigated using extensive numerical simulations. The findings show that while changes in the fractal index change the effective time scaling and lengthen the relaxation phase of glucose recovery, decreasing the fractional order brings stronger memory effects, slowing the glucose decay and prolonging transient dynamics. These results confirm that a more flexible and realistic framework for simulating the course of diabetes is offered by the Caputo fractal–fractional derivative. The created model has the potential to improve clinical prediction and control tactics in diabetes care and provides a potent mathematical tool for capturing complicated biological behaviour. In order to improve predictive accuracy and practical applicability, future research will concentrate on expanding this model by adding genetic and environmental components, coupling it with machine learning-based parameter estimates, and comparing the outcomes to actual clinical datasets.

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