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Fractional Order Glucose Insulin Model with Generalized Mittag-Leffler Kernel

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Abstract: TIn this paper, We formulate a fractional-order mathematical model for the populations of diabetic patients consist threecompartment G, X, and I. Diabetes Model is investigated with fractal-fractional operator for normal and type-1 diabetes. Also, the deterministic mathematical model for diabetes mellitus is investigated with the effect of the fractional parameters. Solutions are derived to investigate the influence of fractional operator which shows the impact of the disease for type-1 diabetes. The existence and uniqueness results of the fractional-order model are derived using fixed point theory. Simulation has been made for developed solutions of fractional order diabetes model to check the actual behavior of a normal person as well as a type-1 diabetes patient.

Keywords: Diabetes model; Ulam-Hyres stability; Fractal-Fractional; Uniqueness; Glucose metabolism.

1 Introduction

Biomathematics is major subject with a large, rapidly growing literature that spans multiple disciplines [1,2]. To gain visibility into complicated biological, ecological circumstances, a vast number of mathematical models have been devised. To describe these concepts, a variety of mathematical methodologies are used [3,4]. Techniques for solving differential equations as well as linear, non-linear, dynamic, and stochastic programming etc are all included. We are not bound to employing only present mathematical models in this sector, but we may also switch to new mathematical methods to deal with the current complex issues in life sciences. Because the situation in life sciences is really complex, we need to acquire a better understanding of the situation before implementing a new mathematical model [5,6]. The model and its implications can be reduced using mathematical procedures in mathematical modelling, and the output can be assessed using observations. Diabetes is rapidly developing as a result of people's imbalanced lifestyles nowadays [7].

One of the greatest worldwide health challenges is the growing epidemic of diabetic mellitus (DM) [8]. Diabetes is a non-communicable disease with various "spread" characteristics, one of which being the influence of social contacts on lifestyle modifications. Diabetes is a long-term illness characterized by elevated blood sugar levels. A person is diagnosed with diabetes if their fasting blood sugar level is greater than 126 mg/dL or their blood sugar level is greater than 200 mg/dL two hours after eating. The pancreatic organs that produce the hormone insulin are unable to function properly. Because body cells cannot receive and digest glucose into energy without insulin, blood sugar levels rise (Diabetes Mellitus type I). Diabetes Mellitus type II is characterized by the body's inability to use the insulin that is present. According to the International Diabetes Federation in 2015 [9]. Diabetes mellitus affects millions of individuals worldwide, and if projections are correct, a $\frac{1}{4}$ billion individuals will be diabetic by 2025. Diabetes mellitus was expected to affect 2.8% of the global population in 2000 and 4.4% in 2030, according to data collected for all age categories. Diabetics are anticipated to increase in

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number from 171 million in 2000 to 366 million by 2030 [10]. In the year 2012, diabetes claimed the lives of 1.5 million people [11]. As per the World Health Organization, 12.9 million people in Pakistan (10 % of the total population) have diabetic, 9.4 million of whom have been recognized and 3.5 million of whom have not diagnosed. Prediabetes affects 38 million been individuals, with 20.5 % of women and 15.9 % of males having the condition. According to another study, Pakistan is ranked seventh among the top 10 countries with type 2 diabetes, and would be fourth by 2030. Consequently, type 2 diabetes and its complications are estimated to kill 120,000 people in Pakistan each year. Each of these numbers point to a concerning situation for Pakistanis [12]. In the year 2000, India had the largest percentage of people with insulin resistance in the world (31.7 million), followed by China (20.8 million) and the United States (17.7 million) [13]. Furthermore, diabetes is frequently linked to genetic factors passed down from parents with a history of the disease [14].

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First, the mechanism of glucose regulation in healthy person is summarized then difference between healthy person and diabetic person explained in order to indicate impermanence of required control scheme. Blood sugar level is balanced by insulin and glucagon in the human body. Insulin and glucagon is known as pancreatic endocrine hormones because they are secreted by pancreas. The relationship of insulin and glucagon is shown in the below Fig. 1. Diabetes, hypoglycemia and other sugar problems are due to imbalanced pancreatic hormones.

Fractional calculus was developed in 1695, only a few years after classical calculus. Liouville, Riemann, Leibniz, and others were credited with the first systematic studies. Fractional calculus was developed in 1695, only a few years after classical calculus. Liouville, Riemann, Leibniz, and others were credited with the first systematic studies [15, 16, 17, 18, 19]

For the years 2000 and 2030, Sarah Wild and her colleagues in (2004) investigated how to calculate diabetes incidence and the number of diabetics of all ages. Diabetes mellitus was estimated to affect 2.8 percent of the global population in 2000 and 4.4 percent in 2030, according to data collected for all age groups. Diabetes is more common in men than in women, but Diabetic women outnumber diabetic males, according toward statistics. These conclusions suggest that even if obesity levels remain constant, the diabetes occurrence will continue [19].

Boutayeb et al. (2006) investigated the diabetes population number. The number of patients who developed problems is also revealed in the report. The mathematical model can be stated as a by using the proper definition of a parameter. It might be linear or non-linear. The non-linear scenario is used for the stability condition. is discussed, and the population's crucial values are investigated [20].



Fig. 1: Physiology of Diabetes Mellitus

Athena Makroglou and her colleagues in (2006) provided a summary of many mathematical models that indicate the relationship between the glucose-insulin regulation system and diabetes, which was bolstered by a survey of existing software. The mathematical model is made up of partial differential, ordinary differential, integro-differential equations, and delay differential [21].

Emma Geraghty in (2008) discusses delay differential equations and their many possibilities, primarily in the biomedical fields, in the project. It uses the solution of differential equations to expand on the concept of insulin therapy [22].

Sh. Yasini et.al. (2009) created a novel model that depicts the regulation of a diabetic patient's blood glucose level during an intensive period. Treatment with insulin. In an unembellished initial state, reinforcement learning theory was used in conjunction with closed-loop control system expert knowledge to maintain a hypoglycemic average of 80 mg/dl and a normal state for total plasma insulin concentration. An offline insulin distribution rate was produced using the Q-learning algorithm without the need for a model of the atmosphere dynamic forces. Toward assess the efficacy of the planned model and compare it toward other current algorithms in terms of regulating high blood glucose level. Simulations on computers were used in [23].

Umer Saleem (2020) used the Caputo-Fabrizio derivative to construct a model for diabetes research and to solve the model's uniqueness alternatives. In Sect, the fixed-point theorem can be used to assess the uniqueness

and presence of system alternatives, and an iterative system stability assessment is used in the Picard Lindelof methodology [24].

We investigate the fractional modelling of diabetes mellitus utilizing fractional derivative definitions for a comprehensive method form a novel operator. To develop and test a fractional order derivatives system for a broad glucose insulin regulatory scheme for diabetes control using a glucose insulin pump. The fixed-point theorem and numerical approach determine existence and uniqueness.

2 Materials and Method

Definition 2.1 A sumulu transform for the function $\psi(z)$

$$S = \psi(z) : \exists \hbar, \chi_1, \chi_2 > 0, \quad \psi(z) < \hbar \exp(\frac{|\chi|}{\chi_1}),$$

if $z \in (-1)^j \times [0, \infty]$

can be expressed as

$$F(v) = ST[\psi(z)] = \int_0^\infty exp(-\chi)\psi(v\chi)d\chi, v \in (-\chi_1, \chi_2)(1)$$

Definition 2.2

For $z \in H^1(x, y)$ and $\kappa \in (0, 1)$. The Caputo-Fabrizio which is define in [25]

$${}^{CF}D_t^{\kappa}\Big(z(t)\Big) = \frac{M(\kappa)}{1-\kappa} \int_x^t z'(\rho) exp\Big[-\kappa \frac{t-\rho}{1-\kappa}\Big]$$
(2)

where $M(\kappa)$ is a normalization function.

Definition 2.3 Antagana-Baleanu in Caputo sense (ABC) can be defined [26] as

$${}^{ABC}_{\alpha}D^{\alpha}_{t}(\psi(t)) = \frac{AB(\alpha)}{n-\alpha} \int_{\alpha}^{\chi} \frac{d^{n}}{dw^{n}} f(w)E_{\alpha} -\alpha \frac{(\chi-w)^{\alpha}}{n-\alpha} dw, \ n-1 < \alpha < n,$$
(3)

where E_{α} is the Mittag-Leffler function and $AB(\alpha)$ is a normalization function. For eq (3), a laplace transformation is:

$$L^{[ABC}_{\alpha}D^{\alpha}_{t}(\psi(t))](S) = \frac{AB(\alpha)}{1-\alpha}\frac{S^{\alpha}L[\psi(\tau)](S) - S^{\alpha-1}\psi(0)}{S^{\alpha} + \frac{\alpha}{1-\alpha}} (4)$$

For using ST for (3), we obtain

$$ST[_{0}^{ABC}D_{t}^{\alpha}(\psi(t))](S) = \frac{B(\alpha)}{1 - \alpha + \alpha S^{\alpha}} \times [ST\psi(t) - \psi(0)](S)$$

Definition 2.4 Atangana-Baleanu fractional integral of order μ of a function $\psi(t)$ can be expressed as [27]

$$ABC_{\mu}^{ABC}I_{\chi}^{\mu}(\psi(\chi)) = \frac{1-\mu}{B-\mu}\psi(\chi) + \frac{\mu}{B(\mu)\Gamma(\mu)}\int_{\alpha}^{\chi}\psi(S)(\chi-S)^{\mu-1}ds.$$
 (6)

3 Fractional Order Mathematical Model

we consider a G-X-I epidemic model by dividing the overall population into three time-dependent classes, such as plasma glucose level in the blood G(t); Glucose absorption X(t); Insulin level in the blood I(t). Many parameters have been collected, and a mathematical model has been created based on the values of these constraints. This model also contains the Basel values G_b and I_b . Thus, the following three differential equations represent the mathematical model.

$$\frac{dG}{dt} = -m_1G + m_2I + m_1G_b \tag{7}$$

$$\frac{dX}{dt} = -m_2 X + m_3 I - m_3 I_b + m_6 I_b \tag{8}$$

$$\frac{dI}{dt} = -m_3 I + m_4 G + m_4 m_5 - m_6 I + m_6 I_b \tag{9}$$

with initial conditions $G(0) = G_0, X(0) = X_0, I(0) = I_0$, To formulate the model, G(t) represent the concentration of glucose in the blood at time t (mg/dl), X(t) represent the generalised insulin variable for the remote compartment, I(t) represent the insulin concentration in the blood at time t. G_b represent the basal pre-injection value of plasma glucose. I_b represent the basal pre-injection value of plasma insulin. m_1 represent insulin independent rate. m_2 represent the rate at which tissue glucose uptake capacity decrease. m_3 represent the insulin-independent increase in glucose absorption ability in I_b . m_4 represent after the glucose injection, the rate at which the pancreatic $\beta - cell$ releases insulin. m_5 represent the threshold glucose value. m_6 represent the Insulin's first-order decay rate in β – *cell* release insulin.

4 Existence and stability theory

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We'll focus on the model's existence and uniqueness results, as well as its HU-stability results. In the Atangana-Baleanu notion, under fractal-fractional operators. Consider

$$FFM D_{0,t}^{\eta,\nu} G(t) = -m_1 G + m_2 I + m_1 G_b$$

$$FFM D_{0,t}^{\eta,\nu} X(t) = -m_2 X + m_3 I - m_3 I_b + m_6 I_b$$

$$FFM D_{0,t}^{\eta,\nu} I(t) = -m_3 I + m_4 G + m_4 m_5 - m_6 I + m_6 I_b$$

with initial conditions $G(0) = G_0, X(0) = X_0, I(0) = I_0$

Utilizing fixed point results, we demonstrate that the model under investigation has at least one and unique solution. Because the integral is differentiable, the proposed model may be written as

$${}^{ABR}D_0^{\eta,\nu}G(t) = \nu t^{\nu-1}M(t,G,X,I)$$
(10)

$${}^{ABR}D_0^{\eta,\nu}X(t) = \nu t^{\nu-1}N(t,G,X,I)$$
(11)

$$^{ABR}D_{0}^{\eta,\nu}I(t) = \nu t^{\nu-1}O(t,G,X,I)$$
(12)

where

$$M(t, G, X, I) = -m_1G + m_2I + m_1G_b$$
$$N(t, G, X, I) = -m_2X + m_3I - m_3I_b + m_6I_b$$
$$O(t, G, X, I) = -m_3I + m_4G + m_4m_5 - m_6I + m_6I_b$$

we can write Equations (10 - 12) as:

$$\begin{aligned} & {}^{ABR}_{0} D^{\eta}_{t} = \mathbf{v} t^{\mathbf{v}-1} \Lambda(t, \Pi(t)) \\ & \Pi(0) = \Pi_{0} \end{aligned}$$

By replacing ${}^{ABR}_0 D^{\eta,\nu}$ by ${}^{ABC}_0 D^{\eta,\nu}$ by applying fractional integral, we get

$$\Pi(t) = \Pi(0) + \frac{\nu t^{\nu-1}}{AB(\eta)} \Lambda(t, \Pi(t))$$
$$\frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{\nu-1} (t-\vartheta)^{\nu-1} \Lambda(t, \Pi(t)) d\vartheta$$
Where $\Pi(t) = (G(t), X(t), I(t))$
$$\Pi(0) = (G(0), X(0), I(0))$$

$$\Lambda(t,\Pi(t)) = \{M(t,G,X,I), N(t,G,X,I), O(t,G,X,I)\}$$

Define a Banach space for the existence theory $S = B \times B \times B$, Where B = [0, T] under the norm

$$||\Pi| = \max_{t \in 0, T} |G(t) + X(t) + I(t)|$$

Define an operator $\Re: S \to S$ as

$$\Re(\Pi)(t) = \Pi(0) + \frac{\nu t^{\nu-1}}{AB(\eta)}\Lambda(t,\Pi(t) +$$

$$\frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{\nu-1} (t-\vartheta)^{\nu-1} \Lambda(t, \Pi(t)) d\vartheta$$
(13)

Now, we suppose that $\Lambda(t, \Pi(t))$ is the non-linear function with growth and Lipschitz conditions.

* For each $\Pi \varepsilon S$, \exists constants $H_{\Lambda} > 0$ and W_{Λ} , such that

$$|\Lambda(t,\Pi(t))| \le H_{\Lambda}|\Pi(t)| + W_{\Lambda} \tag{14}$$

* For each $\Pi, \overline{\Pi} \varepsilon S, \exists$ a constant $J_{\Lambda} > 0$ such that

$$|\Lambda(t,\Pi(t)) - \Lambda(t,\overline{\Pi(t)})| \le J_{\Lambda}|\Pi(t) - \overline{\Pi(t)}$$
(15)

Theorem 1:

For the set of continuous function $\Lambda : [0,T] \times S \rightarrow R$ there exists at least single outcome for system (10-12) holds

Proof: To begin, we must demonstrate that the operator Re specified in (10 - 12) is completed

continuous. Since Λ is continuous. Thus, \Re is also continuous.

Assume that $E = \{\Pi \varepsilon S : \|\Pi\| \le R, R > 0\}$. Then, for any $\Pi \varepsilon S$, we have

$$\begin{split} |\Re(\Pi)| &= \max_{t \in [0,T]} \left| \Pi(0) + \frac{v t^{\nu-1} (1-\eta)}{AB(\eta)} \Lambda(t,\Pi(t)) + \right. \\ &\left. \frac{\eta, \nu}{AB(\eta) \Gamma(\eta)} \int_0^t \vartheta^{\nu-1} (t-\vartheta)^{\nu-1} \Lambda(t,\Pi(t)) d\vartheta \right| \\ &\leq \Pi(0) + \frac{\nu t^{\nu-1} (1-\eta)}{AB(\eta)} (H_A ||\Pi| + W_A) + \end{split}$$

$$\max_{t\in[0,T]}\frac{\eta,\nu}{AB(\eta)\Gamma(\eta)}\int_0^t\vartheta^{\nu-1}(t-\vartheta)^{\nu-1}|\Lambda(\vartheta,\Pi(\vartheta))|d\vartheta$$

$$\leq \Pi(0) + \frac{\nu t^{\nu-1}(1-\eta)}{AB(\eta)} (H_{\Lambda} ||\Pi| + W_{\Lambda}) + \frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} (H_{\Lambda} ||\Pi| + W_{\Lambda}) T^{\eta+\nu-1} E(\eta, \nu) \leq R$$

As a result, the operator \Re has a uniform bound, where $E(\eta, \nu)$ denote the function. For equicontinuity of \Re , let us take $t_1 < t_2 \leq T$. Then consider

$$\begin{split} \Re(\Pi)(t_2) - \Re(\Pi)(t_2)| &== \left| \frac{v t_2^{\nu-1}(1-\eta)}{AB(\eta)} \Lambda(t_2,\Pi(t_2)) + \right. \\ &\left. \frac{\eta,\nu}{AB(\eta)\Gamma(\eta)} \int_0^{t_2} \vartheta^{\nu-1}(t_2-\vartheta)^{\nu-1} \Lambda(\vartheta,\Pi(\vartheta)) d\vartheta \right. \\ &\left. - \frac{v t_1^{\nu-1}(1-\eta)}{AB(\eta)} \Lambda(t_1,\Pi(t_1) + \right. \\ &\left. \frac{\eta,\nu}{AB(\eta)\Gamma(\eta)} \int_0^{t_1} \vartheta^{\nu-1}(t_1-\vartheta)^{\nu-1} \Lambda(\vartheta,\Pi(\vartheta)) d\vartheta \right| \\ &\leq \frac{v t_2^{\nu-1}(1-\eta)}{AB(\eta)} (H_\Lambda |\Pi| + W_\Lambda) + \right. \\ &\left. \frac{\eta,\nu}{AB(\eta)\Gamma(\eta)} (H_\Lambda |\Pi| + W_\Lambda) t_2^{\eta+\nu-1} E(\eta,\nu) \right. \\ &\left. - \frac{v t_1^{\nu-1}(1-\eta)}{AB(\eta)} (H_\Lambda |\Pi| + W_\Lambda) + \right. \\ &\left. \frac{\eta,\nu}{AB(\eta)\Gamma(\eta)} (H_\Lambda |\Pi| + W_\Lambda) t_1^{\eta+\nu-1} E(\eta,\nu) \right] \end{split}$$

Where $t_1 \to t_2$ then $|\Re(\Pi)(t_2) - \Re(\Pi)(t_2)| \to 0$. Consequently, we say that $||\Re(\Pi)(t_2) - \Re(\Pi)(t_2)| \to 0$, as $t_1 \to t_2$

 \Re is equicontinuous in this case. As a result, the Arzela-Ascoli theorem states that it is totally continuous. Thus, the presented model has at least one solution, according to Schauder's fixed point result.

4.1 Ulam-Hyres stability

The presented model is Ulam-Hyres stable if $\exists \Re_{\eta,v} \ge 0$ such that for any $\varepsilon > 0$ and for every $\Pi \varepsilon (L[0,T],R)$ fulfils:

$$|_{0}^{FFM} D_{t}^{\eta, \nu} \Pi(t) - \Lambda(t, \Pi(t))| \leq r, t \varepsilon[0, T]$$

and there exists a unique solution $\Psi \varepsilon(L[0,T],R)$ such that

$$|\Pi(t) - \Psi(t)| \le \Re_{\eta, \nu} \varepsilon, t \varepsilon[0, T]$$

We take into consideration a small perturbation $\Theta \varepsilon L[0,T]$ such that

$$\begin{aligned} Theta(0) &= 0 \\ *| \\ Theta(t)| &\leq \varepsilon \text{ for } \varepsilon > 0 \\ *|_{0}^{FFM} D_{t}^{\eta, \nu} \Pi(t) - \Lambda(t, \Pi(t))| + \Theta(t) \end{aligned}$$

$$\Pi(t) - \Psi(t)| = \left| \Pi(t) - \left(\Psi(0) + \frac{vt^{\nu-1}(1-\eta)}{AB(\eta)} \Lambda(t, \Psi(t)) + \frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{\nu-1}(t-\vartheta)^{\nu-1} \Lambda(\vartheta, \Psi(\vartheta)) d\vartheta \right) \right|$$

$$\leq \left| |\Pi(t) - \left(\Pi(0) + \frac{vt^{\nu-1}(1-\eta)}{AB(\eta)} \Lambda(t,\Pi(t)) + \frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{\nu-1}(t-\vartheta)^{\nu-1} \Lambda(\vartheta,\Pi(\vartheta)) d\vartheta \right) \right|$$

$$+ \left| \Pi(0) + \frac{1}{AB(\eta)} \Lambda(t,\Pi(t)) + \frac{\eta, v}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{v-1} (t-\vartheta)^{v-1} \Lambda(\vartheta,\Pi(\vartheta)) d\vartheta \right|$$

$$\left|\Psi(0) + \frac{vt^{\nu-1}(1-\eta)}{AB(\eta)}\Lambda(t,\Psi(t)) + \frac{\eta}{2}\right|_{t=0}^{t}$$

$$\frac{\eta, \mathsf{v}}{AB(\eta)\Gamma(\eta)} \int_0 \vartheta^{\mathsf{v}-1} (t-\vartheta)^{\mathsf{v}-1} \Lambda(\vartheta, \Psi(\vartheta)) d\vartheta|$$

$$\leq \eta_{\eta,\nu}\varepsilon + \left(\frac{\nu t^{\nu-1}(1-\eta)}{AB(\eta)} + \frac{\eta,\nu}{AB(\eta)\Gamma(\eta)}T^{\eta+\nu-1}E(\eta,\nu)J_A|\Pi(t) - \Psi(t)| \right) \\ \leq \eta_{\eta,\nu}\varepsilon + \alpha|\Pi(t) - \Psi(t)|$$

Lemma 1:

The perturbed model's solution

$$\begin{aligned} & \mathop{}_{0}^{FFM} D_{t}^{\eta, \nu} \Pi(t) = \Lambda(t, \Pi(t)) + \Theta(t) | \\ & \Pi(0) + \Pi_{0} \end{aligned}$$

fulfils the following relation

$$\begin{aligned} \left| \Re(t) - \left(\Pi(0) + \frac{\nu t^{\nu-1}(1-\eta)}{AB(\eta)} \Lambda(t,\Pi(t)) + \frac{\eta,\nu}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{\nu-1}(t-\vartheta)^{\nu-1} \Lambda(\vartheta,\Pi(\vartheta)) d\vartheta \right) \right| &\leq \eta_{\eta,\nu} \notin 16) \end{aligned}$$

$$\eta_{\eta,\nu} = \frac{\nu t^{\nu-1}(1-\eta)}{AB(\eta)} + \frac{\eta,\nu}{AB(\eta)\Gamma(\eta)}T^{\eta+\nu-1}I(\eta,\nu)$$

Proof: It's simple to demonstrate. It can be demonstrated by looking at system (13). The desired results would be achieved after some manipulations on the left-hand side of Ulam-Hyres stable.

Lemma no 2:

The suggested model's solution is Ulam-Hyres stable if meets its requirements and Lemma (1). if $\alpha < 1$

Proof: Assume that $\Psi \varepsilon S$ is a unique solution and $\Pi \varepsilon S$ be any solution of the presented model. Thus, we will reach:

$$\begin{split} |\Pi(t) - \Psi(t)| &= \left| \Pi(t) - \left(\Psi(0) + \frac{vt^{v-1}(1-\eta)}{AB(\eta)} \Lambda(t, \Psi(t)) + \frac{\eta, v}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{v-1}(t-\vartheta)^{v-1} \Lambda(\vartheta, \Psi(\vartheta)) d\vartheta \right) \right| \\ &\leq \left| |\Pi(t) - \left(\Pi(0) + \frac{vt^{v-1}(1-\eta)}{AB(\eta)} \Lambda(t, \Pi(t)) + \frac{\eta, v}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{v-1}(t-\vartheta)^{v-1} \Lambda(\vartheta, \Pi(\vartheta)) d\vartheta \right) \right| \\ &+ \left| \Pi(0) + \frac{vt^{v-1}(1-\eta)}{AB(\eta)} \Lambda(t, \Pi(t)) + \frac{\eta, v}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{v-1}(t-\vartheta)^{v-1} \Lambda(\vartheta, \Pi(\vartheta)) d\vartheta \right| \\ &\left| \Psi(0) + \frac{vt^{v-1}(1-\eta)}{AB(\eta)} \Lambda(t, \Psi(t)) + \frac{\eta, v}{AB(\eta)\Gamma(\eta)} \int_0^t \vartheta^{v-1}(t-\vartheta)^{v-1} \Lambda(\vartheta, \Psi(\vartheta)) d\vartheta \right| \\ &\leq \eta_{\eta, v} \varepsilon + \left(\frac{vt^{v-1}(1-\eta)}{AB(\eta)} + \frac{\eta, v}{AB(\eta)\Gamma(\eta)} T^{\eta+v-1} I(\eta, v)) J_\Lambda |\Pi(t) - \Psi(t)| \\ &\leq \eta_{\eta, v} \varepsilon + \alpha |\Pi(t) - \Psi(t)| \end{split}$$

As a result, one may write

$$\|\Pi - \Psi\| \leq \eta_{n,\nu}^* \varepsilon + \alpha \|\Pi - \Psi\|$$



The above relation may be written as

$$|\Pi - \Psi| \leq \eta^*_{\eta, v} arepsilon$$

Where

$$\Re_{\eta,\nu} = \frac{\eta_{\eta,\nu}}{1-\alpha}$$

. As a result, the proposed problem's solution is Ulam-Hyres stable.

5 Fractional Fractional Operator for Algorithm

We can extend the model (10 - 12) in Caputo sense by using the Atangana-Baleanu integral.

$$G(t) = G(0) + \frac{\nu t^{\nu - 1}(1 - \eta)}{AB(\eta)} E_1(t, G, X, I) +$$

$$\frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \Upsilon^{\nu-1}(t-\Upsilon) E_1(\Upsilon, G, X, I) d\Upsilon$$
(17)
$$X(t) = X(0) + \frac{\nu t^{\nu-1}(1-\eta)}{AB(\eta)} E_2(t, G, X, I) +$$

$$\frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \Upsilon^{\nu-1}(t-\Upsilon) E_2(\Upsilon, G, X, I) d\Upsilon$$
(18)

$$I(t) = I(0) + \frac{vt^{\nu-1}(1-\eta)}{AB(\eta)}E_3(t, G, X, I) +$$

 $\frac{\eta, \mathbf{v}}{AB(\eta)\Gamma(\eta)} \int_0^{\cdot} \Upsilon^{\mathbf{v}-1}(t-\Upsilon) E_3(\Upsilon, G, X, I) d\Upsilon$ (19)

We descend the numerical scheme at $t = t_{b+1}$, we have

$$G^{b+1} = G^0 + \frac{\nu t^{\nu-1}(1-\eta)}{AB(\eta)}E_1(t_b, G^b, X^b, I^b) +$$

$$\frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \Upsilon^{\nu-1}(t-\Upsilon) E_1(\Upsilon, G, X, I) d\Upsilon$$
(20)

$$X^{b+1} = X^0 + \frac{\nu t^{\nu-1}(1-\eta)}{AB(\eta)} E_2(t_b, G^b, X^b, I^b) +$$

$$\frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \Upsilon^{\nu-1}(t-\Upsilon) E_2(\Upsilon, G, X, I) d\Upsilon$$
(21)

$$I^{b+1} = I^0 + \frac{\nu t^{\nu-1}(1-\eta)}{AB(\eta)} E_3(t_b, G^b, X^b, I^b) + \frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \int_0^t \Upsilon^{\nu-1}(t-\Upsilon) E_3(\Upsilon, G, X, I) d\Upsilon$$
(22)

We get the following result

$$G^{b+1} = G^{0} + \frac{vt_{b}^{\nu-1}(1-\eta)}{AB(\eta)}E_{1}(t_{b}, G^{b}, X^{b}, I^{b}) + \frac{\eta, \nu}{AB(\eta)\Gamma(\eta)}\sum_{g=0}^{b}\int_{t_{g}}^{t_{g+1}}\Upsilon^{\nu-1}(t_{b+1}-\Upsilon)E_{1}(\Upsilon, G, X, I)d\Upsilon^{23}$$

$$X^{b+1} = X^0 + \frac{\forall t_b^{v-1}(1-\eta)}{AB(\eta)} E_2(t_b, G^b, X^b, I^b) +$$

 $\frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \sum_{g=0}^{b} \int_{t_g}^{t_{g+1}} \Upsilon^{\nu-1}(t_{b+1} - \Upsilon) E_2(\Upsilon, G, X, I) d\Upsilon 24)$

$$I^{b+1} = I^0 + \frac{vt_b^{\nu-1}(1-\eta)}{AB(\eta)}E_3(t_b, G^b, X^b, I^b) +$$

$$\frac{\eta, \nu}{AB(\eta)\Gamma(\eta)} \sum_{g=0}^{b} \int_{t_g}^{t_{g+1}} \Upsilon^{\nu-1}(t_{b+1} - \Upsilon) E_3(\Upsilon, G, X, I) d\Upsilon 25)$$

By utilizing Lagrangian piece-wise interpolation in $[t_g, t_{g+1}]$, we get

$$T_{s}(\Upsilon) = \frac{\Upsilon - t_{g-1}}{t_{g} - t_{g-1}} t_{g}^{\nu-1} E_{1}(t^{g}, G^{g}, X^{g}, I^{g}) - \frac{\Upsilon - t_{g-1}}{t_{g} - t_{g-1}} t_{g}^{\nu-1} E_{1}(t^{g-1}, G^{g-1}, X^{g-1}, I^{g-1})$$

$$T_{s}(\Upsilon) = \frac{1 - t_{g-1}}{t_{g} - t_{g-1}} t_{g}^{\nu-1} E_{1}(t^{g}, G^{g}, X^{g}, I^{g}) - \frac{\Upsilon - t_{g-1}}{t_{g} - t_{g-1}} t_{g}^{\nu-1} E_{1}(t^{g-1}, G^{g-1}, X^{g-1}, I^{g-1})$$

$$U_{s}(\Upsilon) = \frac{\Upsilon - t_{g-1}}{t_{g} - t_{g-1}} t_{g}^{\nu-1} E_{2}(t^{g}, G^{g}, X^{g}, I^{g})$$
$$-\frac{\Upsilon - t_{g-1}}{t_{g} - t_{g-1}} t_{g}^{\nu-1} E_{2}(t^{g-1}, G^{g-1}, X^{g-1}, I^{g-1})$$

$$V_{s}(\Upsilon) = \frac{\Upsilon - t_{g-1}}{t_{g} - t_{g-1}} t_{g}^{\nu-1} E_{2}(t^{g}, G^{g}, X^{g}, I^{g}) - \frac{\Upsilon - t_{g-1}}{t_{g} - t_{g-1}} t_{g}^{\nu-1} E_{3}(t^{g-1}, G^{g-1}, X^{g-1}, I^{g-1})$$

$$G^{b+1} = G^{0} + \frac{vt_{b}^{\nu-1}(1-\eta)}{AB(\eta)}E_{1}(t_{b}, G^{b}, X^{b}, I^{b}) + \frac{\eta(\Delta t)^{\eta}}{AB(\eta)\Gamma(\eta+2)} \sum_{g=0}^{b} \left[t_{g}^{\nu-1}E_{1}(t_{g}, G^{g}, X^{g}, I^{g}) \times ((b+1-g)^{\eta}(b-g+\eta+2) - (b-g)^{\eta}(2+2\eta+b-g))\right]$$

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$$\begin{split} &-t_{g-1}^{\nu-1}E_1(t_{g-1},G^{g-1},X^{g-1},I^{g-1}) \\ \times ((1+b-g)^{\eta+1}-(b-g)^{\eta}(1+\eta+b-g))] \end{split}$$

$$\begin{aligned} X^{b+1} &= X^0 + \frac{\nu t_b^{\nu-1}(1-\eta)}{AB(\eta)} E_2(t_b, G^b, X^b, I^b) \\ &+ \frac{\eta(\Delta t)^\eta}{AB(\eta)\Gamma(\eta+2)} \end{aligned}$$

$$\begin{split} &\sum_{g=0}^{b} \left[t_{g}^{\nu-1} E_{2}(t_{g}, G^{g}, X^{g}, I^{g}) \right. \\ &\times ((b+1-g)^{\eta} (b-g+\eta+2) - (b-g)^{\eta} (2+2\eta+b-g)) \end{split}$$

$$\begin{split} &-t_{g-1}^{\nu-1}E_2(t_{g-1},G^{g-1},X^{g-1},I^{g-1}) \\ &\times ((1+b-g)^{\eta+1}-(b-g)^{\eta}(1+\eta+b-g))) \end{split}$$

$$I^{b+1} = I^{0} + \frac{\nu t_{b}^{\nu-1}(1-\eta)}{AB(\eta)} E_{3}(t_{b}, G^{b}, X^{b}, I^{b}) + \frac{\eta(\Delta t)^{\eta}}{AB(\eta)\Gamma(\eta+2)}$$

$$\begin{split} &\sum_{g=0}^{b} \left[t_{g}^{\nu-1} E_{3}(t_{g}, G^{g}, X^{g}, I^{g}) \right. \\ &\times ((b+1-g)^{\eta} (b-g+\eta+2) - (b-g)^{\eta} (2+2\eta+b-g)) \end{split}$$

$$\begin{split} &-t_{g-1}^{\nu-1}E_3(t_{g-1},G^{g-1},X^{g-1},I^{g-1}) \\ &\times ((1+b-g)^{\eta+1}-(b-g)^\eta(1+\eta+b-g))] \end{split}$$

The general numerical findings of the investigated model are represented by system (26 - 28).

6 Numerical Results and Discussion

The mathematical analysis has been presented for the fractional-order GXI model having a nonlinear system of fractional differential equations. Simulation represents actual behavior and control situation for continued monitoring of glucose inulin system and insulin concentration in plasma which effect of glucose level. To observe the effects of the parameter on the dynamics of the fractional-order model for Case I (type 1 diabetes) and Case II (normal person), we conclude several numerical simulations varying the value of parameter with time 0 to 300 minutes. In Figs. 2(a,b) and 5(a,b) gives the rise of glucose level in human body for type 1 and normal person

at different values of fractional parameters . In Figs. 3(a,b) and 6(a,b) X and I bounded to the steady state point. Figures 4(a,b) represent the insulin concentration in plasma with the impact of glucose level. Figure 7 (a) represents no insulin production in the human body caused by type 1 diabetes with the passage of time. It is easily observed that we get better results by decreasing the fractional values for a normal person and type-1 diabetes patient as Plasma glucose approaches its actual level by decreasing fractional values. The rising of insulin will help to maintain the plasma glucose level for type-1 diabetic patients which is important to become a healthy person.



Fig. 2: G(t) with fractal fractional derivative at dimension $\alpha = 0.7$.



Fig. 3: X(t) with fractal fractional derivative at dimension $\alpha = 0.7$.



Fig. 4: I(t) with fractal fractional derivative at dimension $\alpha = 0.7$.



Fig. 5: G(t) with fractal fractional derivative at dimension $\alpha = 0.9$.

7 Conclusion

In this paper, we will design a model that will employ the fewest number of parameters possible and will be able to regulate the growing number of diabetics. This can be accomplished by utilizing a variety of diabetes-related factors such as exercise, diet, and medications, among others. As a set of non-linear coupled with ordinary differential equations, the model captures population dynamics during the disease. The efficiency of the proposed control measures was demonstrated by numerical simulation of the obtained findings. The solution is obtained for the fractional-order model using



Fig. 6: X(t) with fractal fractional derivative at dimension $\alpha = 0.9$.



Fig. 7: I(t) with fractal fractional derivative at dimension $\alpha = 0.9$.

the fractal fractional operator which is bounded for healthy persons and type-1 diabetic patients. The result shows significant changes using different fractional values for a normal person as well as a type-1 diabetes patient. Unique and bounded solutions are proved using the fixed point theory. Numerical simulations are carried out to check the actual behavior for a normal person as well as type-1 diabetes patients which will be helpful in the future analysis of diabetic patients and control strategies. The convergence analysis is provided to demonstrate the efficiency of the method. It is possible to do research in this area in order to control the growing diabetes population, which will be extremely beneficial to medical science.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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