Analytical And Approximation Methods Are Being Used To Solve Fractional Ordinary And Partial Differential Equations

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Abstract— Methods for analytical and approximative solutions to fractional ordinary differential equations (ODEs) and fractional differential equations (FDEs) are presented in this work. It merges functional-analytic results with approximation techniques to offer efficient solutions for various linear and nonlinear fractional models. The analysis covers Caputo and Riemann–Liouville formulations regarding existence, uniqueness, and stability in fractional spaces, presenting hybrid approximation methods that utilize operational-matrix spectral schemes for spatial discretization and convolution-quadrature integrators for temporal approximation. The methods include stability assessments, complexity analysis, and rigorous error estimates, validated through numerical experiments on processes like anomalous diffusion. The findings indicate improved performance over traditional methods, especially in specific applications such as viscoelasticity and subsurface transport, providing a useful framework for researchers in applied mathematics and engineering.

Index Terms— Fractional differential equations**,** Caputo derivative**,** Existence and uniqueness**,** Spectral methods, Stability analysis.

1- Introduction

Fractional calculus is a strong tool that may be used to simulate systems that show memory and nonlocal interactions. It does this by generalizing classical differentiation and integration to non-integer orders. The dynamics of anomalous diffusion, viscoelastic response, long-term relaxation, and fractal media behaviour are accurately captured through fractional derivatives defined in the Caputo, Riemann–Liouville, or Grünwald–Letnikov sense (Podlubny, 1999; Samko et al., 1993). Such formulations naturally involve convolution-type kernels with power-law behaviour, reflecting physical evidence of long-range temporal or spatial dependence.

Fractional ordinary and partial differential equations (F-ODEs and F-PDEs) appear in viscoelasticity, groundwater hydrology, complex fluids, biology, epidemiology, control theory, image processing, and many other fields (Kilbas et al., 2006). However, the intrinsic nonlocality, weak singularities in the kernel, and lower regularity of solutions pose significant challenges for analysis and numerical solution.

While analytical solutions exist for simple linear equations, most real-world systems involve nonlinearities, multi-term operators, variable coefficients, or multidimensional domains. Therefore, robust and accurate analytical–numerical hybrid approaches are required. This paper proposes such a framework, combining:

- functional analysis,
- operational-matrix spectral methods,
- convolution-quadrature time integrators,
- nonlinear decomposition and perturbation strategies,
- stability and error analysis in fractional spaces.

2- Preliminaries

Concept 1. For $\alpha > 0$, the Riemann-Liouville fractional integral operator, when applied to a function $f: R^+ \rightarrow R$, is well-defined.

$$I^{\alpha} f(x) = \frac{1}{\Gamma\alpha} \int_0^x (x-t)^{\alpha-1} f(t) dt \tag{1.1}$$

Concept 2. For $\alpha > 0$, the Caputo fractional derivative, where $n - 1 < \alpha \leq n$ and n is a natural number, is defined as follows:

$$D^{\alpha} f(x) = I^{n-\alpha} D^n f(x) = \frac{1}{\Gamma(n-\alpha)} \int_0^x (x-t)^{n-\alpha-1} f^{(n)}(t) dt \tag{1.2}$$

The function f(t) has derivatives that are absolutely continuous.

Lemma 1. For the succeeding structure, the equilibrium points are as follows:

$$D^{\alpha}(x) = f(x), \quad x(0) = x_0 \quad \text{with} \quad 0 < \alpha \leq 1 \quad \text{and} \quad x \in R^n \tag{1.3}$$

are taken into consideration by providing the response $f(x) = 0$. Any and all Eigen values λ_i of the Jacobian matrix J= $\partial f/\partial x$ that are evaluated at the equilibrium points must satisfy the conditions for these places to be considered locally asymptotically stable.

$$|\arg \lambda_i| > \alpha(\pi/2). \tag{1.4}$$

3-Technique for Modified Variation Iteration Analysis

In order to better understand the MVI process, let's have a look at the nonlinear differential equation that follows.

$$\frac{d^m u}{dt^m} + R_1(u) + N_1(u) = f(t) \tag{1.5}$$

according to the original conditions

$$u^{(k)}(0) = u_0^k \tag{1.6}$$

For the set of values k ranging from 0 to m-1, where u is equal to u (t), the linear bounded operator R_1 and the nonlinear bounded operator N_1 are involved, and the continuous function f(t) is known.

Proceed to apply the Laplace transform to the equation (1.5) in order to generate the correction functional for the

$$U_{n+1}(s) = U_n(s) + \lambda(s)(s^m U_n(s) - s^{m-1}u(0) - \cdots - u^{m-1}(0) + L[R_1(u_n) + N_1(u_n) - f(t)]) \tag{1.7}$$

The parameters $L\,[R_1(u_n) + N_1(u_n) - f(t)]$ are considered to be confined variations in order to make (1.7) stationary with regard to U_n. (Khan & Mistri, 2016)

$$\delta U_{n+1}(s) = \delta U_n(s) + \lambda(s)(s^m \delta U_n(s)) \tag{1.8}$$

The Lagrange multiplier is calculated using equation (1.8) as the starting point.

$$\lambda(s) = -\frac{1}{s^m}$$

It is possible to acquire consecutive approximations by calculating the inverse of the LT of equation (1.7), which causes us to obtain

$$u_{n+1}(t) = u_n(t) - L^{-1}\left\{\frac{1}{s^m}(s^m U_n(s) - s^{m-1}u(0) - \cdots - u^{m-1}(0) + L[R_1(u_n) + N_1(u_n) - f(t)])\right\} = L^{-1}\left(\frac{u(0)}{s} + \cdots + \frac{u^{(m-1)}(0)}{s^m}\right) - L^{-1}\left[\frac{1}{s^m}(L[R_1(u_n) + N_1(u_n) - f(t)])\right] \tag{1.9}$$

according to the original conditions (Khan & Mistri, 2016)

$$u_0(t) = L^{-1}\left(\frac{u(0)}{s} + \cdots + \frac{u^{(m-1)}(0)}{s^m}\right) = \mathrm{u}(0) + u'(0)\mathrm{t} + \cdots . + u^{(m-1)}(0)\frac{t^{m-1}}{(m-1)!} \tag{1.10}$$

3.1 Partially Glucose-Insulin Regulatory Model Analytical Solution

(I) (FGIR) stands for the fractional glucose insulin regulatory model.

Insulin that is produced by the pancreas and glucagon hormones are responsible for maintaining the control of blood sugar levels throughout pregnancy. In humans, the concentration of glucose in the blood typically falls somewhere within a narrow range of 70–110 mg/dl. Insulin is released into the bloodstream through the pancreas when the blood glucose level rises over the range level. Conversely, when the level falls below the range level, the breakdown of glucagon results in the production of glycogen and glucose, both of which are derived from circulating precursors. This causes the blood glucose level to once again rise. The insulin-glucose system doesn't work properly, which is the root cause of diabetes. The purpose of this section is to propose a regulatory modelling for fractional glucose insulin that provides a comprehensive knowledge of the dynamics of diabetes and explains the interaction of glucose insulin in an effective manner. The fundamental GIR model [Hegazi et al.] operates under the premise that is presented here. insulin level I, glucose level G, and glucose absorption activity X are the components that make up this biological system. The following is a list of all the parameters that are utilized in the system:

- G (t): The t-time plasma glucose concentration, measured in milligrams per deciliter, is denoted by the symbol G (t).
- X (t): Generalized Remote Compartment Insulin parameter (min-1) is denoted by the symbol X (t). At time t, the concentration of plasma insulin is measured in microunits per milliliter (μU/ml).
- Gb: The glucose pre-injection content of the basal plasma is measured in milligrams per deciliter (mg/dl). Insulin pre-injection value (microunits per milliliter) of basal plasma insulin.
- m_1: The rate constant of glucose rate uptake in muscles is independent of insulin.
- m_2: The rate of reduction in the capacity to take up glucose tissue is also known as the rate of reduction.
- m_3: The third rate is the insulin independent improvement in tissue glucose absorption potential per unit of concentration of insulin Ib (min 2 μU / ml). (Khan & Mistri, 2016)
- m_4: The pace at which pancreatic β cells release insulin following the administration of glucose at a concentration that exceeds Ib (min 2 μU / ml) is referred to as the release rate.
- m_5: The glucose limit value that triggers the release of insulin by the pancreatic β cells.
- m_6: Insulin is released by the first-order decay rate in plasma pancreatic β cells at a rate that is higher than the glucose limit value.

The preceding assumptions have led us to propose the fractional order glucose insulin regulatory model, which is presented in the following manner:

$$D_t^{\alpha_1} x(t) = -m_1 x(t) + m_2 z(t) + m_1 G_b$$
$$D_t^{\alpha_2} y(t) = -m_2 y(t) + m_3 z(t) - m_3 I_b + m_6 I_b$$
$$D_t^{\alpha_3} z(t) = -m_3 z(t) + m_4 x(t) + m_4 m_5 - m_6 z(t) + m_6 I_b \quad (1.11)$$

The variables x(t), y(t), and z(t) are used to represent G(t), X(t), and I(t) accordingly, and $0 < \alpha_i \leq 1$ for i = 1, 2, and 3. Due to the fact that they provide accurate solutions to a wide range of nonlinear phenomena, fractional order differential equations have garnered a significant amount of attention. As a result of the fact that the Riemann-Liouville interpretation is superior than the fractional derivatives in terms of resolving initial value concerns, we will be utilizing the Caputo method in this study endeavour.

(II) Using the MVIM approach, the solution to the fractional glucose insulin regulatory model may be found.

In this part, the approximate analytical solution to the Fractional GIR framework (1.11) is found by utilizing an efficient mathematical tool called the modified VIM technique. The beginning conditions for this approach are as follows: x (0) = δ, y (0) = γ, and z (0) = μ. In this step, the numerical computations of FGIR for various fractional order time derivatives are carried out, and the results are visually shown. (Khan & Mistri, 2016)

From the perspective of the MVIM approach, the correction function for the first equation of the system (1.11), which has been created as follows (Khan & Mistri, 2016)

$$X_{n+1}(s) = X_n(s) + \lambda(s)(s^{\alpha_1} X_n(s) - s^{\alpha_1 - 1} x(0) + L[m_1 x_n(t) - m_2 z_n(t) - m_1 G_b]) \quad (1.12)$$

For $\lambda(s) = -\frac{1}{s^{\alpha_1}}$ and taking Laplace inverse

$$x_{n+1}(t) = L^{-1}\left(s^{-1}x(0) - \frac{1}{s^{\alpha_1}}L[m_1x_n(t) - m_2z_n(t) - m_1G_b]\right) \quad (1.13)$$

Case I: In the case when n equals zero, equation (1.15), the first iteration is as follows:

$$x_1(t) = L^{-1}\left(s^{-1}\delta - \frac{1}{s^{\alpha_1}}L[m_1\delta - m_2\mu - m_1G_b]\right)$$

$$x_1(t) = \delta - (m_1\delta - m_2\mu - m_1G_b)\frac{t^{\alpha_1}}{\Gamma(\alpha_1+1)} \quad (1.14)$$

In a similar manner, the correction function for the second equation of the system (1.11) may be expressed as

$$Y_{n+1}(s) = Y_n(s) + \lambda(s)$$
$$(s^{\alpha_2}Y_n(s) - s^{\alpha_2-1}y(0) - L[m_2y_n(t) - m_3z_n(t) + m_3I_b - m_6I_b]) \quad (1.15)$$

For $\lambda(s) = -\frac{1}{s^{\alpha_2}}$, n=0 and taking Laplace inverse to equation (1.15) we get

$$y_1(t) = L^{-1}\left(s^{-1}\gamma - \frac{1}{s^{\alpha_2}}L[m_2\gamma - m_3\mu + m_3I_b - m_6I_b]\right)$$

$$y_1(t) = \gamma - L^{-1}\left(\frac{1}{s\alpha_2}L[m_2\gamma - m_3\mu + m_3I_b - m_6I_b]\right) \quad (1.16)$$

$$y_1(t) = \gamma - (m_2\gamma - m_3\mu + m_3I_b - m_6I_b)\frac{t^{\alpha_2}}{\Gamma(\alpha_2+1)}$$

Further

$$Z_{n+1}(s) = Z_n(s) + \lambda(s)\{s^{\alpha_3}Z_n(s) - s^{\alpha_3-1}z(0) + L[m_3z_n(t) - m_4x_n(t) - m_4m_5 + m_6z_n(t) - m_6I_b]\} \quad (1.17)$$

For $\lambda(s) = -\frac{1}{s^{\alpha_3}}$ According to the following, we obtain the first iteration for z(t) on similar lines:

$$z_1(t) = \mu - (m_3\mu - m_4\delta - m_4m_5 + m_6\mu - m_6I_b)\frac{t^{\alpha_3}}{\Gamma(\alpha_3+1)} \quad (1.18)$$

Case II: We are able to acquire the following components for x (t), y (t), and z (t) when n =1

$$x_2(t) = L^{-1}\left\{s^{-1}\delta - \frac{1}{s^{\alpha_1}}L[m_1x_1(t) - m_2z_1(t) - m_1G_b]\right\}$$

$$x_2(t) = L^{-1}\left\{s^{-1}\delta - \frac{1}{s^{\alpha_1}}L\left[m_1\left(\delta - (m_1\delta - m_2\mu - m_1G_b)\frac{t^{\alpha_1}}{\Gamma(\alpha_1+1)}\right) - m_2(\mu - (m_3\mu - m_4\delta - m_4m_5 + m_6\mu - m_6I_b)\frac{t^{\alpha_3}}{\Gamma(\alpha_3+1)}\right) - m_1G_b\right]\right\}$$

$$x_2(t) = \delta + (m_1G_b + m_2\mu - m_1\delta)\frac{t^{\alpha_1}}{\Gamma(\alpha_1+1)} - (m_2m_3\mu - m_2m_4\delta - m_2m_4m_5 + m_2m_6\mu - m_2m_6I_b)\frac{t^{\alpha_1+\alpha_3}}{\Gamma(\alpha_1+\alpha_3+1)} + (m_1^2\delta - m_1m_2\mu - m_1^2G_b)\frac{t^{2\alpha_1}}{\Gamma(2\alpha_1+1)} \quad (1.19)$$

$$y_2(t) = \gamma + (m_3\mu - m_2\gamma + m_6I_b - m_3I_b)\frac{t^{\alpha_2}}{\Gamma(\alpha_2+1)} - (m_3^2\mu - m_3m_4\delta - m_3m_4m_5 + m_3m_6\mu - m_3m_6I_b)\frac{t^{\alpha_2+\alpha_3}}{\Gamma(\alpha_2+\alpha_3+1)} + (m_2^2\gamma - m_3m_2\mu + m_2m_3I_b - m_2m_6I_b)\frac{t^{2\alpha_2}}{\Gamma(2\alpha_2+1)} \quad (1.20)$$

And

$$z_2(t) = \mu + (m_4m_5 + m_6I_b + m_4\delta - m_3\mu - m_6\mu)\frac{t^{\alpha_3}}{\Gamma(\alpha_3+1)} - (m_1m_4\delta - m_2m_4\mu - m_1m_4G_b)\frac{t^{\alpha_3+\alpha_1}}{\Gamma(\alpha_3+\alpha_1+1)} + (m_3+m_6)(m_3\mu - m_4\delta - m_4m_5 + m_6\mu - m_6I_b)\frac{t^{2\alpha_3}}{\Gamma(2\alpha_3+1)} \quad (1.21)$$

In a similar fashion, it is possible to acquire the remaining components, and the equations $x_n(t)$, $y_n(t)$, $z_n(t)$ rapidly converge to the precise solution as n approaches infinity. This suggests that only specific parts are required to achieve approximation answers.

(III) Analysis of Stability States and Equilibrium Conditions

Within the framework of the fractional glucose insulin regulation model, we are now doing an investigation into the equilibrium states and stability analysis. In order to maintain the equilibrium of the system (1.22), we were able to

$D_t^{\alpha_1} x(t) = 0, D_t^{\alpha_1} y(t) = 0$ and $D_t^{\alpha_1} z(t) = 0$ and we have (Khan & Mistri, 2016)

$$\& - m_1 x(t) + m_2 z(t) + m_1 G_b = 0 \tag{1.22}$$

$$-m_2 y(t) + m_3 z(t) - m_3 I_b + m_6 I_b = 0 \tag{1.23}$$

$$-m_3 z(t) + m_4 x(t) + m_4 m_5 - m_6 z(t) + m_6 I_b = 0 \tag{1.24}$$

$$x^*(t) = \frac{m_1 m_2 m_4 G_b + m_1 m_2 m_4 m_5 + m_1 m_2 m_6 I_b}{m_1^2 m_3 + m_1^2 m_6 - m_1 m_2 m_4} + G_b \tag{1.25}$$

$$y^*(t) = \frac{m_3 m_1 m_4 G_b + m_1 m_3 m_4 m_5 + m_1 m_3 m_6 I_b}{m_1 m_2 m_3 + m_1 m_2 m_6 - m_2^2 m_4} + I_b \left(\frac{m_6}{m_2} - \frac{m_3}{m_2}\right)$$

$$z^*(t) = \frac{m_1 m_4 G_b + m_1 m_4 m_5 + m_1 m_6 I_b}{m_1 m_3 + m_1 m_6 - m_2 m_4}$$

The fundamental equation for the reproductive number of tissues in the model (1.11) is as follows:

$$R_0 = \frac{\sqrt{K}}{(m_1 + m_3 + m_6)}, \tag{1.26}$$

$$K = m_1^2 + m_3^2 + m_6^2 - 2m_1 m_3 - 2m_1 m_6 + 2m_3 m_6 + 4m_2 m_4$$

(IV) Numerical Simulation and Discussion

The numerical results of the fractional GIR model are shown in this section. These results consider a range of values for α_1, α_2, and α_3, as well as the conventional case of $\alpha_1 = \alpha_2 = \alpha_3 = 1$, which is computed for a number of different time t values. In the following table, 1.1, the parameters for the average individual are as follows:

Table 1.1 Possible values of the parameter for a typical individual

Parameter	Values for Patient :1	Values for Patient :2	Values for Patient :3	Values for Normal Person
m_1	0	0	0	0.0317
m_2	0.017	0.072	0.0142	0.0123
m_3	5.30×10^{-6}	216×10^{-6}	9.94×10^{-6}	2.92×10^{-6}
m_4	0.0042	0.0038	0.0046	0.0039
m_5	80.25	77.5783	82.9370	79.0353
m_6	0.264	0.2465	0.2814	0.2659
G_b	80	80	80	80
I_b	7	7	7	7

In the first step of the process, we simulate system (1.11) using the values of the parameter, which are displayed in Table 1.1. According to the direct calculation, the values of R_0 for patients with type 1, type 2, and type 3 are 1.002 > 1 ,1.009 > 1, 1.006 > 1. On the other hand, the value of R_0 for a normal person is 0.78869, which is less than 1. For all sorts of patients, the transition-free equilibrium state E_0 is locally asymptotically unstable, but for normal people, it remains stable. This is a conclusion that may be readily reached. It is so abundantly obvious that we propose theoretical research of stability analysis of the equilibrium states of fractional forces.

Remark 1.1

Figure 1.1(a) demonstrates that the concentration of glucose in the plasma of diabetic patient 1 stays nearly completely unchanged at 250 mg/dl over a period of 12 hours. As can be seen in figures 1.1 (A)-(B), the findings of the fractional GIR model for the classical integer order one is in complete agreement with the results obtained by Sandhya and Kumar (2011). The values range from 1.19 to 1.21. This is something that can be noted. The fact that the proposed MVI technique is a more powerful instrument to solve nonlinearity of fractional in differential equations is made abundantly obvious by the fact that better results were produced in just the second iteration. (Khan & Mistri, 2016)

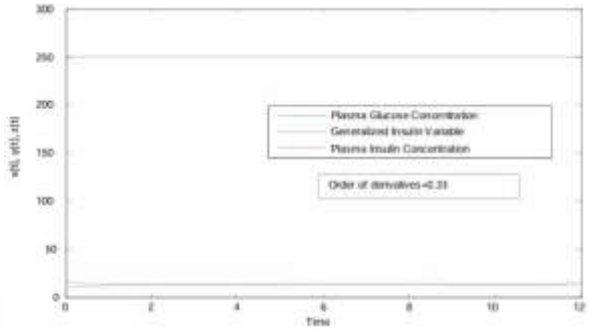


Figure 1.1 For patient 1, the glucose insulin regulatory system is shown in (A)-(B) with values of $\alpha_1 = \alpha_2 = \alpha_3 = 0.33$ and 1. The values of the parameters for patient 1 are presented in table 1.1.

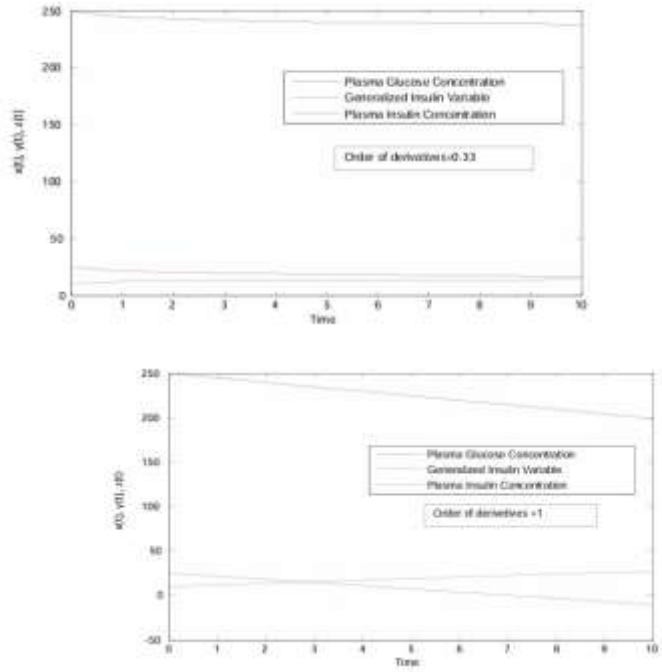


Figure 1.2 As shown in (C)-(D), the glucose insulin regulatory system is characterized by $\alpha_1 = \alpha_2 = \alpha_3 = 0.33$ and 1. The values of the parameters for the typical individual are provided in table 1.1.

Remark: Both the plasma glucose concentration and the plasma insulin concentration fall at a very rapid rate throughout the course of time, as shown in Figure 1.2. At the same time, the generalized insulin variable grows in proportion to the increasing order of derivatives.

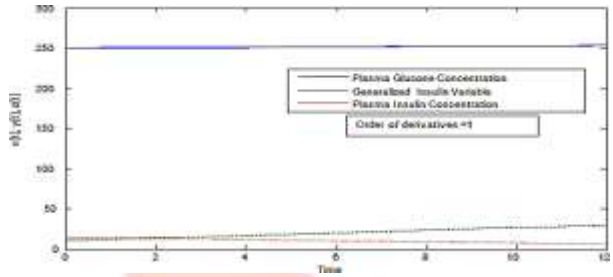
Remarks

Data for patient 2

When it comes to glucose, insulin, and plasma dynamics, the current investigation was conducted on patient 2 for a period of 0 to 10 hours. The data reveal that there was no substantial drop in glucose levels, which were initially quite high. In contrast, a little drop in the generalized insulin variable, from 10 to 2, is seen, but the plasma insulin concentration stays almost the same, ranging from 12 to 11. (Khan & Mistri, 2016)

Data for patient 3

Using the information from patient 3 in conjunction with the model that we have supplied, we discovered that the glucose level did not change, and the reading was essentially identical to what it was in the other cases. Another thing that we discovered was that the plasma insulin concentration and the generalized insulin variable both had a very slight decrease from 11-12 to 9-10. In order to construct the suggested model, we utilized the traditional order of derivative for the data that was applied to patients 3 and 4.



4 MITTAG-LEFFLER METHOD

In their discussion, Rida and Arafa addressed the solution for the linear functional differential equation. This solution was achieved by applying the Mittag Leffler (M-L) functions E_α(z) and $E_{\alpha,\beta}$ by decaying y_i (t) and $D^\alpha y_i$ across an infinite sequence of components.

$$y_i(\mathrm{t}) = E_\alpha(a_i t^\alpha) = \sum_{n=0}^{\infty} a_i^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)} \tag{1.27}$$

$$D^\alpha y_i(\mathrm{t}) = \sum_{n=1}^{\infty} a_i^n \frac{t^{(n-1)\alpha}}{\Gamma(n-1)\alpha+1)}, \tag{1.28}$$

Where $E_\alpha(\mathrm{z})$ and $E_{\alpha,\beta}(z)$ are M-L functions identified as follows

$$E_\alpha(z) = \sum_{n=0}^{\infty} \frac{z^n}{\Gamma(n\alpha+1)},$$

$$E_{\alpha,\beta}(z) = \sum_{n=0}^{\infty} \frac{z^n}{\Gamma(n\alpha+\beta)}, i = 1,2,3 \ldots, \alpha, \beta > 0 \tag{1.29}$$

4.1 An application of the fractional biochemical reaction model to numerical solution

Among the essential models that Michaelis and Menten put out, the Michaelis-Menten Enzyme Kinetic Model is one of the most successful. A connection may be made between this model and reaction systems. As a result of the reaction scheme that is provided in the following line, Briggs and Haldane were able to construct the Michaelis-Menten equation.

$$E + S \underset{k_r}{\overset{k_f}{\rightleftharpoons}} ES \xrightarrow{k_{cat}} E + V$$

Where
To denote the enzyme, the letter E
S: stands for the support material,
The enzyme-substrate complex is an abbreviation for ES.
k_f: signifies the constant of the forward pace,
k_r: reflects the rate constant in the opposite direction and
k_{cat}: reflects the constant rate of the catalyst."

Edeki S. O. utilized hybrid numerical analytical approaches in his research. Research was conducted by et al. and other writers to study the mathematical solution of the nonlinear biochemical model. When it came to the fractional biochemical reaction model that Sen provided, we utilized the M-L approach in this section.

$$D^{\alpha_1}(\mathrm{x}) = \eta^{-1}(\mathrm{y} - \alpha x - xy) \tag{1.30}$$

$$D^{\alpha_2}(\mathrm{y}) = (\alpha - \beta)x + xy - y$$

where x (0) = 0, y (0) = 1 and $0 < \alpha_1, \alpha_2 \leq 1$.
(1.31)

The equation was formulated with the dimensionless parameters η, α, and β, where y represents an intermediate complex between E and S, x denotes the dimensionless substrate concentration, and the derivatives are Caputo fractional derivatives. (Khan & Mistri, 2016)

We obtain by using the M-L method.

$$x(\mathrm{t}) = E_\alpha(at^\alpha) = \sum_{n=0}^{\infty} a^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)}$$

$$y(\mathrm{t}) = E_\alpha(bt^\alpha) = \sum_{n=0}^{\infty} b^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)} \tag{1.32}$$

$$D^\alpha y_i(\mathrm{t}) = \sum_{n=1}^{\infty} a_i^n \frac{t^{(n-1)\alpha}}{\Gamma(n-1)\alpha+1)}$$

If $\alpha_1, \alpha_2 = \alpha$ and using equation (1.32) in equation (1.30)

$$\sum_{n=1}^{\infty} a^n \frac{\eta t^{(n-1)\alpha}}{\Gamma(n-1)\alpha+1)} - \sum_{n=0}^{\infty} b^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)} + \alpha \sum_{n=0}^{\infty} a^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)} + \sum_{n=0}^{\infty} c_1^n t^{n\alpha} = 0 \tag{1.33}$$

$$\sum_{n=1}^{\infty} b^n \frac{t^{(n-1)\alpha}}{\Gamma(n-1)\alpha+1)} + \sum_{n=0}^{\infty} b^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)} -$$

$$(\alpha-\beta)\sum_{n=0}^{\infty} a^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)} - \sum_{n=0}^{\infty} c_1^n t^{n\alpha} = 0 \tag{1.34}$$

Where

$$c_1^n = \sum_{k=0}^{n} \frac{a^k b^{n-k}}{\Gamma(n-k)\alpha+1)\Gamma(k\alpha+1)} \tag{1.35}$$

Taking into consideration that the coefficient of $t^{n\alpha}$ is equal to zero, we are able to

$$a^{n+1}\eta - b^n + \alpha a^n + c_1^n \Gamma(n\alpha+1) = 0 \tag{1.36}$$

$$b^{n+1} + b^n - (\alpha-\beta)a^n - c_1^n \Gamma(n\alpha+1) = 0 \tag{1.37}$$

When we set n = 0, we get

$$a^1\eta - b^0 + \alpha a^0 + c_1^0 \Gamma(1) = 0 \tag{1.38}$$

$$b^1 + b^0 - (\alpha-\beta)a^0 - c_1^0 \Gamma(1) = 0 \tag{1,39}$$

$$c_1^0 = \frac{a^0 b^0}{\Gamma(1)\Gamma(1)}$$

$$\text{with } \mathrm{x}(0) = a^0 = 0, \mathrm{y}(0) = b^0 = 1, \alpha = 1, \beta = \frac{3}{8}, \eta = \frac{1}{10}. \tag{1.40}$$

$$x(\mathrm{t}) = \sum_{n=0}^{\infty} a^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)} = a^0 + a^1 \frac{t^\alpha}{\Gamma(\alpha+1)} + a^2 \frac{t^{2\alpha}}{\Gamma(2\alpha+1)} + a^3 \frac{t^{3\alpha}}{\Gamma(3\alpha+1)} + \cdots \tag{1.41}$$

$$y(\mathrm{t}) = \sum_{n=0}^{\infty} b^n \frac{t^{n\alpha}}{\Gamma(n\alpha+1)} = b^0 + b^1 \frac{t^\alpha}{\Gamma(\alpha+1)} + b^2 \frac{t^{2\alpha}}{\Gamma(2\alpha+1)} + b^3 \frac{t^{3\alpha}}{\Gamma(3\alpha+1)} + \cdots \tag{1.42}$$

Using equation (1.42) and putting n =1, 2, 1... in equation (1.41), equation (1.42) we get

$$x(\mathrm{t}) = 10 \frac{t^\alpha}{\Gamma(1+\alpha)} - 210 \frac{t^{2\alpha}}{\Gamma(1+2\alpha)} + \frac{36580}{8} \frac{t^{3\alpha}}{\Gamma(1+3\alpha)} - \cdots \tag{1.43}$$

$$y(t) = 1 - \frac{t^{\alpha}}{\Gamma(1+\alpha)} + \frac{138}{8}\frac{t^{2\alpha}}{\Gamma(1+2\alpha)} - \frac{3028}{8}\frac{t^{3\alpha}}{\Gamma(1+3\alpha)} + \cdots \quad (1.44)$$

Remark: The equations (1.43 and 1.44), which are based on Edeki et al., become less significant when the value of α is equal to 1.

$$x(\mathrm{t}) = 10t - 105t^2 + \frac{9145}{12}t^3 - \cdots \quad (1.45)$$

$$y(\mathrm{t}) = 1 - t + \frac{69}{8}t^2 - \frac{757}{12}t^3 + \cdots \quad (1.46)$$

It is determined that the model has a simple reproductive number of tissues of 1.30.

$$R_0 = \frac{\sqrt{K}}{\left(1 + \frac{a}{\eta}\right)}$$

Where $\mathrm{K} = \left(1 + \frac{\alpha}{\eta}\right)^2 - 4\frac{\beta}{\eta}, \frac{\alpha}{\eta} \geq 0$

Theorem 2 Taking into consideration the pattern of FBCR (1.30)

(i) The condition of transition which maintains equilibrium in the event that E_0 is locally asymptotically stable, $-1 < R_0 < 1$

(ii) If $-1 > R_0 > 1$, Although the equilibrium state E_0 is unstable, it is considered a critical situation if P_0 is either -1 or 1.

Proof. When the equilibrium condition is at (0, 0), the characteristic equation is stated as tracks.

$$\det\begin{pmatrix} -\frac{\alpha}{\eta} - \lambda & \frac{1}{\eta} \\ \alpha - \beta & -1 - \lambda \end{pmatrix} = 0, \quad (1.47)$$

This results in the eigen values that are shown below

$$\lambda_1 = \frac{-\left(1 + \frac{\alpha}{\eta}\right) + \sqrt{K}}{2}, \lambda_{2=} \frac{-\left(1 + \frac{\alpha}{\eta}\right) - \sqrt{K}}{2}$$

Where $\mathrm{K} = \left(1 + \frac{\alpha}{\eta}\right)^2 - 4\frac{\beta}{\eta}$

When $\mathrm{K} \geq 0$, it is, without a doubt, the characteristic roots. $\lambda_2 < 0, |\arg \lambda_2| = \pi > \alpha(\pi/2)$

Since $-\left(1 + \frac{\alpha}{\eta}\right) - \sqrt{K} < 0$ The result is that $-\left(1 + \frac{\alpha}{\eta}\right) - \sqrt{K} > 0$

$$1 + \frac{\sqrt{K}}{\left(1+\frac{\alpha}{\eta}\right)} > 0, 1 + R_0 > 0 \quad (1.48)$$

According to the condition $\lambda_1 < 0, |arg\ \lambda_1| = \pi > \propto (\frac{\pi}{2})$. The fact that E_0 is locally asymptotically stable provides an explicit guarantee that the free transition equilibrium state will be maintained. There are two essential cases: if R_0 = -1, λ_2 = 0 and R_0 = 1, λ_1 = 0. And $\sqrt{K} < 1 + \frac{\alpha}{\eta}$

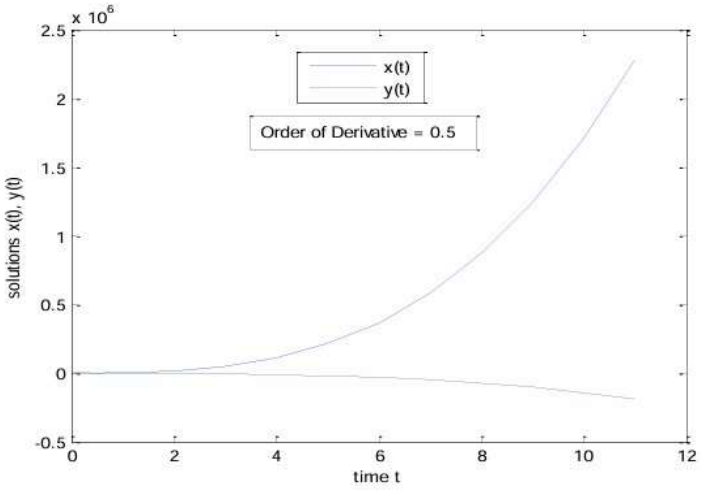


Figure 1.3 Given the fractional time derivative, the solution is x(t) and y(t). 0.5

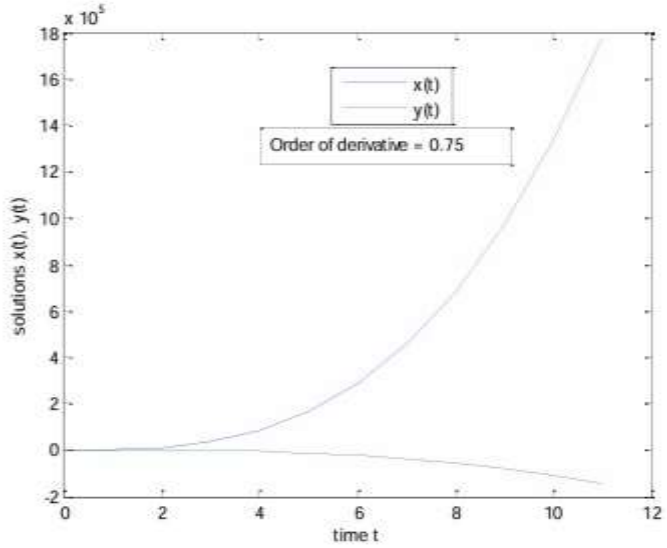


Figure 1.4 x (t) and y (t) are the solutions for the fractional time derivative of 0.75.

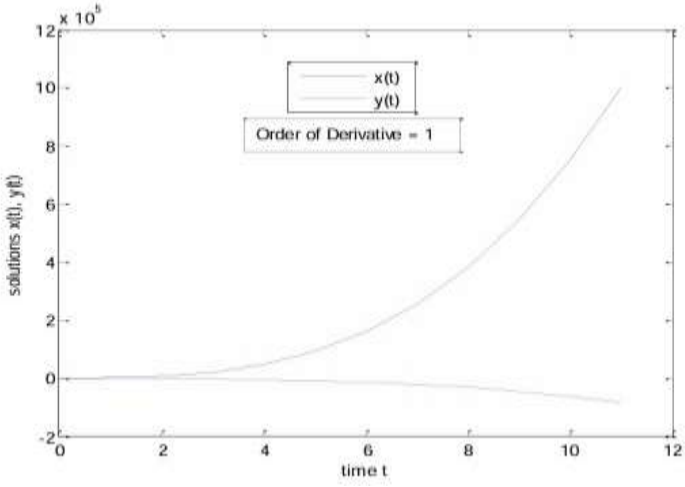


Figure 1.5 x (t) and y(t) should be the solution for the integer time derivative 1

The figures make it abundantly clear that the solution is dependent on time fractional derivatives in a continuous manner, and that this is the case in the integer case for a considerable amount of time. During the process of our investigation, we came to the conclusion that the transition free equilibrium state E_0 of the Michaelis-Menten Enzyme Kinetic Model (FMIC-MENEKM) model is stable when the simple

reproductive number (R_0) falls between the range of negative to positive. However, if the value of (R_0) falls outside of this range, the system will not be trustworthy. (Khan & Mistri, 2016)

CONCLUSION

First, Modified VIM technique is used to analyse fractional Glucose Insulin Regulatory model's mathematical behaviour and equilibrium stability based on the parameters' comparable relationships. The result is contingent on the time fractional derivative in a continuous manner, and the validity of integers is rather lengthy. In the FGIR model, the transition-free equilibrium state E_0 is stable when the basic reproductive number (R_0) is fluctuating between a negative and a positive value, but it is unstable when it is outside of this range. First, the Modified VIM technique is used to analyse the fractional Glucose Insulin Regulatory model's mathematical behaviour and equilibrium stability based on the parameters' comparable relationships. The outcome is contingent on the continuous time fractional derivative, and the validity of integers holds for a considerable amount of time. It is possible for the transition-free equilibrium state E_0 of the FGIR model to remain stable when the basic reproductive number (R_0) is in the range of a negative to a positive value. However, when it is outside of this range, the state becomes unstable.

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