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Development and simulation of a mathematical model representing the dynamics of type 1 diabetes mellitus with treatment

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ABSTRACT

The research aims to understand and study type 1 diabetes and its response to treatment using a mathematical model. We employ a novel method that combines the Shehu transformation with the Akbari–Ganji approach and the Padé approximation to derive approximate solutions for this model. The research findings convincingly show the effectiveness of the method used. The results show a positive impact of the investigated treatment on individuals with type 1 diabetes. Strong agreement is observed between the results obtained from this model's solutions and those of previous studies, confirming the accuracy and reliability of the simulation method employed. This method is considered a successful simulation technique for future studies, enhancing our understanding of the effects of treatments on individuals with type 1 diabetes. From a practical standpoint, the study's results can offer valuable insights to healthcare professionals, enabling them to make more informed decisions regarding treatment strategies. These insights have the potential to optimize treatment plans, potentially leading to improved health outcomes for patients. Furthermore, this research paves the way for further advanced studies in the field of medical modeling and simulation.

1. Introduction

Diabetes is one of the most common diseases worldwide, affecting millions of people of all ages. Sugar is one of the main sources of energy in the body, and the blood sugar level is regulated by insulin, which plays a critical role in metabolizing sugar and converting it into usable energy. However, this process is disrupted in the case of diabetes. Diabetes can be divided into two main types. Type 1 diabetes occurs when a person's body stops producing insulin completely or almost. Type 1 diabetes is less common, often appears at an early age, and requires regular insulin treatment.[1] Type 2 diabetes occurs when a person's body is unable to use insulin effectively or does not produce enough insulin. Type 2 diabetes is more common in people who are older and obese.[2] Understanding the causes and mechanisms of diabetes mellitus and its effects on health and the body is a very important topic, as it requires early and effective diagnosis and management to reduce potential complications and improve the quality of life for affected individuals.

Research and studies in this field are essential for the development of prevention and treatment methods and the improvement of health care provided to patients. Many researchers have attempted to investigate the interactions between glucose and insulin using mathematical models.[3,4] Shabestari et al.[5] presented a new mathematical model to describe the interactions between Beta cells, insulin, and glucose. The results showed that the new system can explain these interactions in various disorders such as diabetes or hypoglycemia. Ali and Tahir[6] proposed a mathematical model for the glucose and insulin regulation system in type 1 diabetes. The results of this study showed that the system has many dynamics in different conditions. Vorobyeva proposed a mathematical model to diagnose the course of retinopathy in diabetic patients, and its findings showed that it is consistent with the results of the clinical diagnosis of diabetes.[7] Nemati et al.[8] developed a new treatment for people with type 1 diabetes, and the treatment has proven successful after clinical trials, as the new treatment is based on the use of stem cells that produce insulin to treat people with type 1 diabetes. Karampelias et al.[9] identified a molecule that helps stimulate the growth of new insulin-producing cells. The molecule, CID661578 has been identified. Researchers have examined the molecular interactions in Beta cells and discovered that they bind to a protein called MNK2. It was also shown that when two other proteins are allowed to interact at higher levels, it ultimately leads to an increase in Beta cell regeneration and thus an increase in the level of insulin in the blood, which leads to a decrease in the amount of glucose in diabetic patients. The results of the study point to a new potential target for the treatment of diabetes. Nasif

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Nomenclature

SAGPM	Shehu–Akbari–Ganji–Padé method
AGM	Akbari–Ganji's method
ADM	Adomian decomposition method
CPU	Central processing unit
S	the Shehu operator
S^{-1}	the Shehu inverse operator
h	mesh size
N	natural numbers

and Al-Nassir presented a mathematical model for optimal control of diabetic patients and studied the dynamic behavior of the disease.[10] Furthermore, numerous studies have addressed the examination and analysis of various dynamic systems.[11–15]

Due to scientific and research developments and the discovery of new treatments for type 1 diabetes, we set out to develop a mathematical model that represents type 1 diabetes and study the effect of a new treatment (CID661578). We will create an accurate mathematical model that reflects the characteristics of the disease and the body's response to the treatment. This model will help in analyzing and evaluating the effects and effectiveness of treatment in improving patients' conditions. A mathematical model of type 1 diabetes will take into account key factors such as blood sugar levels, insulin levels, and Beta cell response. The effect of the new treatment on these factors will be studied, and data analysis will be performed to assess its effectiveness. To achieve this, we will use a combination of the Shehu transformation, Akbari–Ganji's method, and Padé approximation. Algorithms of a new method that we will denote by abbreviated SAGPM. The Akbari–Ganji method, which was pioneered by Ganji and Akbari; is one of several analytical approaches and can be used to solve a range of ordinary and partial nonlinear differential equations.[16] Moreover, Pade's approximation is a numerical technique used to approximate functions by representing them as partial fractions. It provides higher accuracy and improved computational performance compared to other approximation methods, this was created in 1890 by Henri Padé.[17] The Shehu transformation, established by Maitama and Zhao,[18] is an integral transformation used to solve ordinary and partial differential equations and has many applications in various fields. The application of the proposed methodology has yielded promising results, highlighting its effectiveness and efficiency compared to other methods. In addition, the study also focused on the topic of stability analysis, which has been extensively explored in previous research.[19–23] The strategy exhibits high precision and excellent convergence, making it a superior choice for addressing the problem at hand. The solutions of the proposed model have reasonable stability, indicating consistent blood sugar levels over time. This signifies relative stability and effective control of blood sugar levels, demonstrating the success of the implemented SAGPM procedure. Moreover, the results obtained provided strong evidence of the effectiveness of the treatment and its positive effect on managing diabetes by regulating insulin and glucose levels in the body. These results are consistent with the results of clinical studies and laboratory experiments.[9]

This work is organized as follows. Section 2 will elucidate the structure of our proposed methodology while also delving into the foundational principles behind it. This will be achieved by shedding light on the fundamental concept underpinning the Akbari–Ganji method, the Shehu transformation, and the Padé approximation. In Section 3, we will introduce a novel mathematical model for understanding type 1 diabetes and its response to treatment. Section 4 will involve the application of this newly devised algorithm to the proposed mathematical model. The subsequent section, Section 5, will be dedicated to presenting and discussing the results obtained from this application. Sections 6 and 7 will delve into the analysis of convergence and stability for the proposed model. The final section will encapsulate the primary findings of this study. When applying the suggested methodology, the results have demonstrated the effectiveness and efficiency of the proposed approach, characterized by high precision, good convergence, and stability.

2. The new SAGPM algorithm

The fundamental notion of the SAGPM is based on Shehu's transformation method and Akbari–Ganji's method with Padé's approximation algorithms, which will be summarized below:

2.1. Shehu transformation method

The Shehu transformation is a generalized form of the Laplace and Sumudu transforms. It was introduced by Maitama and Zhao in 2019. The authors have used it to solve many differential equations.[18]

The Shehu transformation is obtained over the set A:

$$A=\left\{f(t):\ \exists\, N,n_1,n_2>0,|f(t)|<N\exp\left(\frac{|t|}{n_j}\right),\ if\ t\in\ (-1)^j\times[0,\infty)\right\},$$

Which is defined by:

$$S[f(t)]=F(v,u)=\int_0^{\infty}\exp\left(\frac{-vt}{u}\right)f(t)\,dt=\lim_{a\to\infty}\int_0^{a}exp\left(\frac{-vt}{u}\right)f(t)\,dt,\ v>0,u>0. \tag{2.1}$$

The inverse Shehu transformation is given by

$$S^{-1}[\,F(v,u)]=f(t),\ for\ t\ \geq 0.$$

or equivalently

$$f(t)=\ S^{-1}[\,F(v,u)]=\frac{1}{2\pi i}\int_{a-i\infty}^{a+i\infty}\frac{1}{u}exp\left(\frac{vt}{u}\right)F(v,u)\,dv, \tag{2.2}$$

where, v and u are the variables of the Shehu transformation, α is the real constant and the integral in Eq. (2.2) is taken along $v=a$ in the complex plane $v=x+iy$.

2.1.1. Shehu transformation of derivatives and some functions

If $S\{x(t)\}=X(v,u)$ then

1) $S\left\{\frac{dx}{dt}\right\}=\frac{v}{u}X(v,u)-x(0)$,
2) $S\{x^{(n)}(t)\}=\frac{v^n}{u^n}X(v,u)-\sum_{k=0}^{n-1}\left(\frac{v}{u}\right)^{n-(k+1)}x^{(k)}$,
3) $S\{t\}=\frac{v^2}{u^2}$,
4) $S\{\exp(at)\}=\frac{v}{u-av}$,
5) $S\{\cos(t)\}=\frac{vu}{u^2+v^2}$,
6) $S\{\sin(t)\}=\frac{vu}{u^2-v^2}$,
7) $S\left\{\frac{t^n}{n!}\right\}=\left(\frac{v}{u}\right)^{n+1},\ n=0,1,2,\ldots$

2.2. Akbari–Ganji's method (AGM)

The method, which was developed by Akbari and Ganji, is an excellent computation methodology that may be utilized to resolve various nonlinear differential equations. In this approach, the solution is taken to be a finite series; hence, the result is obtained by resolving a sequence of algebraic problems.

To use AGM, the differential equation for a function $x(t)$ and its derivatives can be written as follows[16,24]:

The nonlinear differential equation of mth order derivatives can be written as

$$h_k = f\left(x, x^{'}, x^{''}, ..., x^{(m)}\right) = 0,\ x = x\ (t) \tag{2.3}$$

with boundary conditions:

$$x(t) = x_0,\ x^{'}(t) = x_1, ...,\ x^{(m-1)}(t) = x_{m-1},\ at\ t = 0 \tag{2.4a}$$

$$x(t) = x_{L_0}, x^{'}(t) = x_{L_1}, ...,\ x^{(m-1)}\ (t) = x_{L_{m-1}},\ at\ t = L \tag{2.4b}$$

Let us assume the solution for the differential Eq. (2.3) as follows:

$$x(t) = \sum_{i=0}^{n} a_i\ t^i. \tag{2.5}$$

Eq. (2.5) can be solved with high accuracy by selecting more terms. If the series (2.5) has (n) degrees, then there are ($n+1$) unknown coefficients that must be determined to find the solution to the differential Eq. (2.3). For Eq. (2.5), the boundary conditions (2.4a) and (2.4b) are applied as follows:

We have the following at $t = 0$.

$$\begin{cases} x(0) = a_0 = x_0 \\ x^{'}(0) = a_1 = x_1 \\ x^{''}(0) = a_2 = x_2 \\ \vdots \end{cases} \tag{2.6}$$

when $t = L$

$$\begin{cases} x(L) = a_0 + a_1 L + ... + a_n L^n = x_{L_0} \\ x^{'}(L) = a_1 + 2a_2 L + ... + n\, a_n L^{n-1} = x_{L_1} \\ x^{''}(L) = 2a_2 + 6a_3 L ... + n\,(n-1)\, a_n L^{n-2} = x_{L_2} \\ \vdots \end{cases} \tag{2.7}$$

After substituting Eqs. (2.6) and (2.7) into Eq. (2.3), and applying the boundary conditions on Eq. (2.3), we obtain:

$$\begin{cases} h_0 = f\left(x(0), x^{'}(0), x^{''}(0), ..., x^{(m)}(0)\right) \\ h_1 = f\left(x(L), x^{'}(L), x^{''}(L), ..., x^{(m)}(L)\right) \\ \vdots \end{cases} \tag{2.8}$$

Application of the boundary conditions on the derivatives of the differential Eq. (2.8) are:

$$h_k^{'} : \begin{cases} f\left(x^{'}(0), x^{''}(0), x^{'''}(0), ..., x^{(m+1)}(0)\right) \\ f\left(x^{'}(L), x^{''}(L), x^{'''}(L), ..., x^{(m+1)}(L)\right) \end{cases} \tag{2.9}$$

$$h_k^{''} : \begin{cases} f\left(x^{''}(0), x^{'''}(0), x^{''''}(0), ..., x^{(m+2)}(0)\right) \\ f\left(x^{''}(L), x^{'''}(L), x^{''''}(L), ..., x^{(m+2)}(L)\right) \end{cases} \tag{2.10}$$

Finally, the ($n+1$) equations are obtained, and by calculating the system of equations, the unknown coefficients a_0 , a_1 , a_2 ,..., and a_n are calculated. Finally, the solution of the differential Eq. (2.2) is obtained.

2.3. Padé approximation

Henri Padé is credited with the development of the Padé approximation, a traditional rational approximation proposed by George Frobenius. It is based on the properties of the rational power approximation and the quotient of two polynomials of different degrees. It offers a better approximation of the function than the Taylor series. The approximation has been widely used in computer science.[25–28]

Padé approximation is a ratio of two polynomials that come from the Taylor series expansion of a function $x(t)$ and is defined[17]

$$P_m^l = \frac{\sum_{n=0}^{l} a_n t^n}{\sum_{n=0}^{m} b_n t^n}, \tag{2.11}$$

where $b_0 = 1$.

The function $x(t)$can by written as

$$x(t) = \sum_{n=0}^{\infty} c_n t^n. \tag{2.12}$$

Also, we have that

$$x(t) - P_m^l = o\left(t^{l+m+1}\right)$$

Thus, we find that

$$\sum_{n=0}^{\infty} c_n t^n = \frac{\sum_{n=0}^{l} a_n t^n}{\sum_{n=0}^{m} b_n t^n}, \tag{2.13}$$

from Eq. (2.13), we obtain the following system equations

$$\begin{aligned} a_0 &= c_0 \\ a_1 &= c_1 + c_0 b_1 \\ a_2 &= c_2 + c_1 b_1 + c_0 b_2 \\ &\vdots \end{aligned}$$

where, c_n is given.

To solve the above system for a_n and b_n, we take the numerator degree to be l and the denominator degree to be m. Additionally, we take the Taylor series expansion of $x(t)$ up to t^{l+m}. Consequently, the following stages summarize the basic idea of the new method of SAGPM.

We consider the following system

$$\left.\begin{aligned} \frac{dx_1}{dt} &= f(x_1(t), x_2(t), a_1(t), ..., a_n(t)) \\ \frac{dx_2}{dt} &= g(x_1(t), x_2(t), b_1(t), ...b_n(t)) \end{aligned}\right\}, \tag{2.14}$$

with initial conditions:

$$x_1(0) = \alpha,\ x_2(0) = \beta.$$

Step 1: Take the Shehu transformation on both sides of (2.14), to get:

$$\left.\begin{aligned} S\left(\frac{dx_1}{dt}\right) &= S(f(x_1(t), x_2(t), a_1(t), ...a_n(t))) \\ S\left(\frac{dx_2}{dt}\right) &= S(g(x_1(t), x_2(t), b_1(t), ...b_n(t))) \end{aligned}\right\}. \tag{2.15}$$

Using the differentiation property of the Shehu transformation and the above initial conditions, we have that:

$$\left.\begin{aligned} X_1(u,v) &= \frac{\alpha\, u}{v} + \frac{u}{v}\, S(f(x_1(t), x_2(t), a_1(t), ...a_n(t))) \\ X_2(u,v) &= \frac{\beta\, u}{v} + \frac{u}{v}\, S(g(x_1(t), x_2(t), b_1(t), ...b_n(t))) \end{aligned}\right\}. \tag{2.16}$$

Step 2: Applying the inverse Shehu transformation on both sides of (2.16), to find:

$$\left.\begin{aligned} x_1(t) &= \alpha + S^{-1}\left(\frac{u}{v} S(f(x_1(t), x_2(t), a_1(t), ...a_n(t)))\right) \\ x_2(t) &= \beta + S^{-1}\left(\frac{u}{v} S(g(x_1(t), x_2(t), b_1(t), ...b_n(t)))\right) \end{aligned}\right\}. \tag{2.17}$$

Step 3: Consider the AGM polynomial series with constant coefficients as follows:

$$x_1(t) = \sum_{i=0}^{n} a_i t^i,\ x_2(t) = \sum_{i=0}^{n} b_i t^i. \tag{2.18}$$

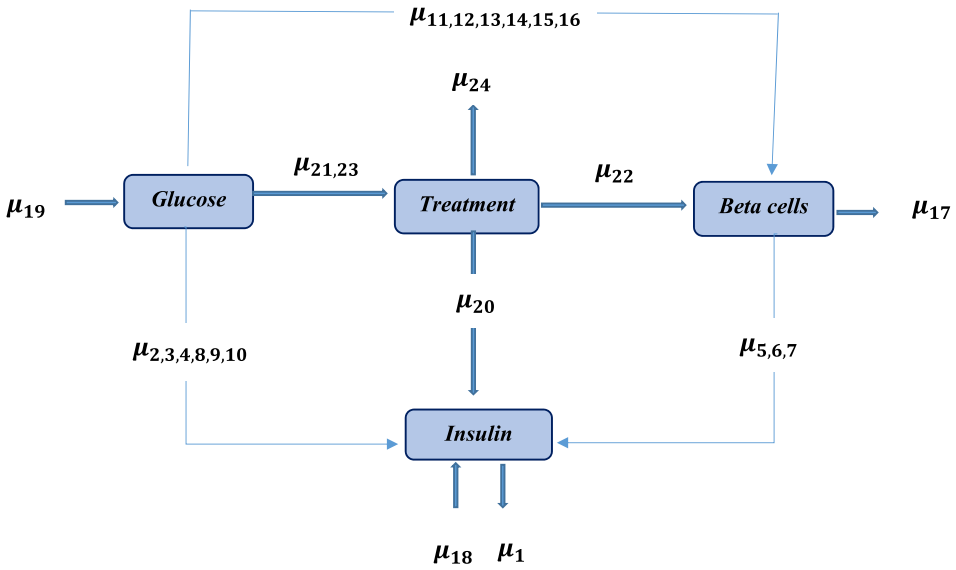

Fig. 1. A scheme of the dynamics of glucose, insulin, Beta cells and treatment.

Table 1
Parameter values used in system (24).

Symbol	Value	Source	Symbol	Value	Source
μ_1	0.30	[6]	μ_{13}	−0.02	[6]
μ_2	0.69	[6]	μ_{14}	0.83	[6]
μ_3	0.27	[6]	μ_{15}	1.01	[6]
μ_4	0.92	[6]	μ_{16}	1.25	[6]
μ_5	0.98	[6]	μ_{17}	1.43	[6]
μ_6	−0.62	[6]	μ_{18}	−0.83	[6]
μ_7	−0.10	[6]	μ_{19}	1.93	[6]
μ_8	1.24	[6]	μ_{20}	0.66	Estimated
μ_9	−1.06	[6]	μ_{21}	0.88	Estimated
μ_{10}	−0.29	[6]	μ_{22}	0.40	Estimated
μ_{11}	1.95	[6]	μ_{23}	0.20	Estimated
μ_{12}	0.75	[6]	μ_{24}	0.40	Estimated

Table 2
Comparison of the error and CPU time between ADM, AGM and SAGPM, when $h = 0.05,\ at\ t \in [0,1]$, with the parameters given in Table 1 except $\mu_{20} = \mu_{21} = \mu_{22} = \mu_{23} = \mu_{24} = 0$.

Functions	Errors	ADM *	AGM	SAGPM
$\varphi(t)$	L_2	0.3271165279	0.3271165279	0.1516653767
	L_∞	0.7954682477	0.7954682480	0.2270051841
	CPU(s)	0.016	0.016	0.015
$\varnothing(t)$	L_2	0.8485549788	0.8485549782	0.002731588407
	L_∞	2.063480395	2.063480394	0.0044218458
	CPU(s)	0.031	0.016	0.015
$\rho(t)$	L_2	0.2487277900	0.2487277900	0.2284040929
	L_∞	0.604845804	0.6048458040	0.3392946128
	CPU(s)	0.016	0.016	0.015
$\tau(t)$	———	———	———	———

After substituting (2.18) into (2.17), Eq. (2.17) becomes as follows:

$$\left.\begin{aligned}\sum_{i=0}^{n} a_i\, t^i &= \alpha + S^{-1}\left(\frac{u}{v} S\left(f\left(\sum_{i=0}^{n} a_i\, t^i, \sum_{i=0}^{n} b_i\, t^i, a_1(t), \ldots, a_n(t)\right)\right)\right)\\ \sum_{i=0}^{n} b_i\, t^i &= \beta + \left(\frac{u}{v} S\left(g\left(\sum_{i=0}^{n} a_i\, t^i, \sum_{i=0}^{n} b_i\, t^i, b_1(t), \ldots b_n(t)\right)\right)\right)\end{aligned}\right\}. \tag{2.19}$$

We apply the initial conditions to obtain some values of the coefficients. By deriving of Eq. (2.19) and substituting the initial conditions, we obtain the rest of the values a_i *and* b_i .

Step 4: Applying the Padé approximation of an order [l / m] to a power series solution obtained by using (SAGPM). The values l and m are arbitrarily selected. In this stage, we obtain the final solution.

Table 3
Comparison of the error and CPU time between ADM, AGM and SAGPM, when $h = 0.05,\ at\ t \in [0,1]$, with the parameters given in Table 1.

Functions	Errors	ADM *	AGM	SAGPM
$\varphi(t)$	L_2	0.09305357075	0.09305357097	0.08227117594
	L_∞	0.2262837692	0.2262837700	0.1514208974
	CPU(s)	0.031	0.031	0.016
$\varnothing(t)$	L_2	1.887985777	1.887985776	0.03370406584
	L_∞	4.591124600	4.591124597	0.0432695140
	CPU(s)	0.032	0.031	0.016
$\rho(t)$	L_2	0.8983607285	0.8983607291	0.3760973105
	L_∞	2.184595928	2.184595929	0.4883590074
	CPU(s)	0.032	0.032	0.016
$\tau(t)$	L_2	0.01716191994	0.01716191990	0.007426620375
	L_∞	0.0417336368	0.0417336368	0.0207398424
	CPU(s)	0.031	0.031	0.016

3. The new mathematical model of type 1 diabetes

Type 1 diabetes results from a disruption in regulating blood glucose levels due to an insufficient production of insulin by Beta cells in the pancreas. While treatment through daily insulin injections, lifestyle changes, and various medications can assist in managing the condition, there is currently no definitive cure available for this disease. Hence, stimulating the self-renewal of pancreatic Beta cells is considered a promising therapeutic option for treating diabetes. In this section, we will create a mathematical model that reflects the characteristics of diabetes and the body's response to treatment based on previous mathematical models in this field.

3.1. Prey-predator model

The Lotka–Volterra system is a mathematical model used to describe dynamic interactions in biological, chemical, and physical systems. This model was developed by Lotka and Volterra.[29] It is represented as

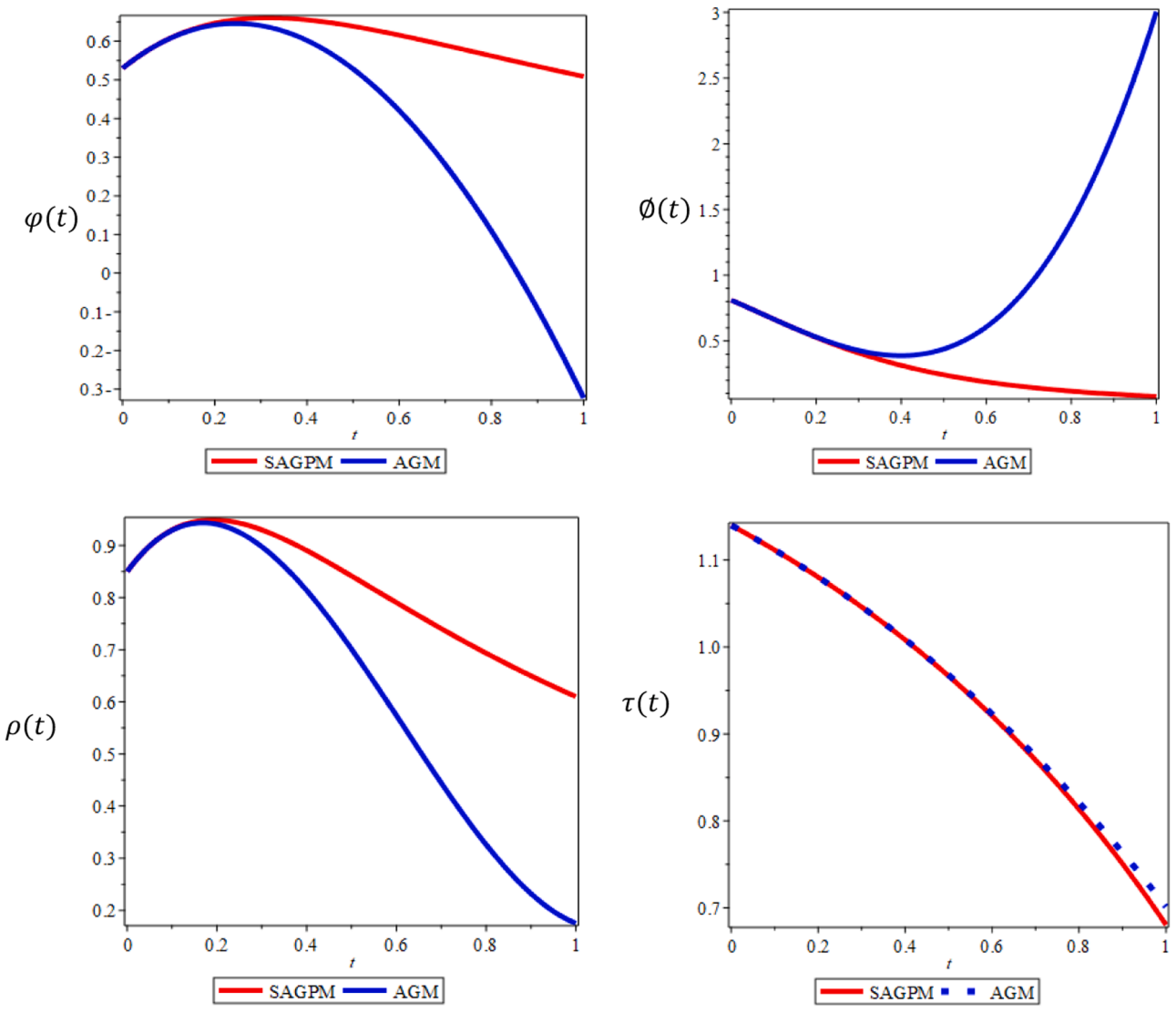

Fig. 2. Comparison of approximate solutions obtained by AGM and SAGPM for system (3.5).

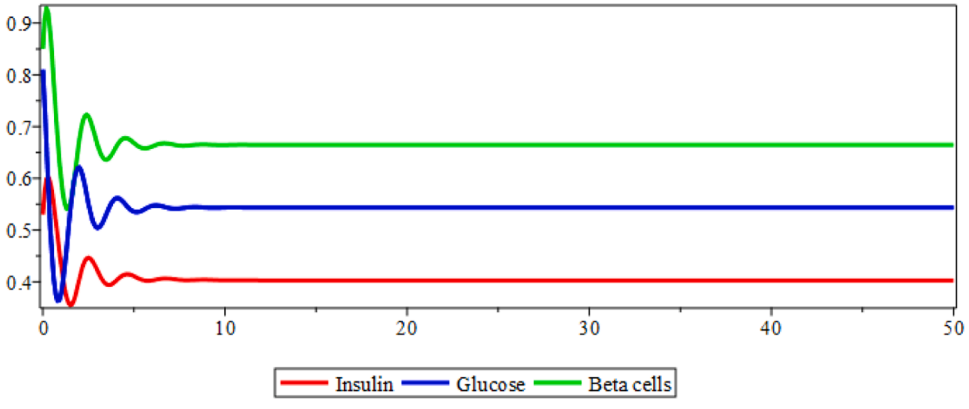

Fig. 3. Time series of the solution of system (3.5) for the data given in Table 1 except $\mu_{20} = \mu_{21} = \mu_{22} = \mu_{23} = \mu_{24} = 0$.

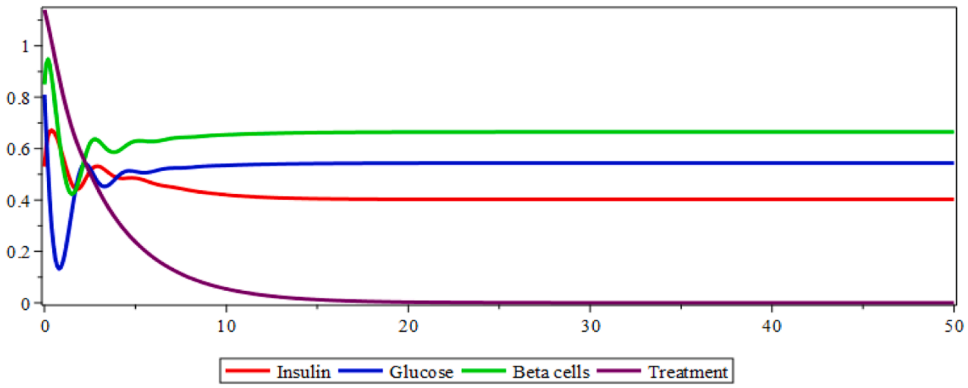

Fig. 4. Time series of the solution of system (3.5) for the data given in Table 1.

Table 4
The values of $\sigma^{j}_{1,2,3,4}$ using the convergence condition at $0 \leq t \leq 1$, for $\varphi(t)$, $\varnothing(t)$, $\rho(t)$ and $\tau(t)$ in system (3.5).

Functions	$\sigma^{0}_{1,2,3,4}$	$\sigma^{1}_{1,2,3,4}$	$\sigma^{2}_{1,2,3,4}$
$\varphi(t)$	0.1780653569	0.1498212851	0.008486957251
$\varnothing(t)$	0.1209900007	0.07840028743	0.004176006708
$\rho(t)$	0.8860648686	0.05138865251	0.03749947078
$\tau(t)$	0.6580441221	0.06402821514	0.003550141987

Table 5
Equilibria and eigenvalues of the system (3.5).

Equilibrium	Eigenvalues
(0.402, 0.543, 0.664, 0)	$\lambda_1 = -0.3527$ $\lambda_2 = -0.6886 + 2.9608i$ $\lambda_3 = -0.6886 - 2.9608i$ $\lambda_4 = -0.2914$

follows

$$\left.\begin{aligned} \frac{d\varphi}{dt} &= a\varphi(1-\varphi) - b\varphi\varnothing \\ \frac{d\varnothing}{dt} &= -c\varnothing + \varphi\varnothing \end{aligned}\right\}, \tag{3.1}$$

where, $\varphi(t)$ and $\varnothing(t)$ are the population densities of prey and predator, respectively, a, b, and c are parameters.

3.2. Previous models of insulin-glucose interaction systems

In 1964 Ackerman et al.[30] proposed a mathematical model representing the relationship between insulin and glucose in the body

$$\left.\begin{aligned} \frac{d\varphi}{dt} &= a_1\varnothing(t) - a_2\varphi(t) + c_1 \\ \frac{d\varnothing}{dt} &= -a_3\varnothing(t) - a_4\varphi(t) + c_2 + D(t) \end{aligned}\right\}, \tag{3.2}$$

where, insulin and glucose concentrations represent the prey and the predator, respectively.$D(t)$ represents the rate of increase in blood sugar.

In 1987 Bajaj et al.[31] proposed a mathematical model for the regulation of insulin, glucose, and Beta cells.

$$\left.\begin{aligned} \frac{d\varphi}{dt} &= b_1\varnothing(t) - b_2\varphi(t) + c_1 \\ \frac{d\varnothing}{dt} &= \frac{b_3 n}{\rho} - b_4\varphi(t) + c_2 \\ \frac{d\rho}{dt} &= b_5\varnothing(T-\rho) + b_6\rho(T-\rho) - b_7\rho \end{aligned}\right\}, \tag{3.3}$$

where, $\rho(t)$ represents the Beta cell population density. T represents the total density of Beta cells. c_1 is the rate of increase of φ in the absence of ρ and $\varnothing$. c_2 is the increase rate of $\varnothing$ in the absence of φ and ρ. b_1 is the rate of increase in insulin in response to an increase in glucose. b_2 is the rate of insulin reduction. b_3 and n are coefficients that control the effect of the hormone insulin and the concentration of disease-related components. b_4 is the rate of glucose decrease as a result of insulin secretion. b_5 is the rate of increase in Beta cell division as a result of interactions between glucose and nondividing Beta cells. b_6 is the rate of increase in Beta cell division as a result of interactions between dividing and nondividing Beta cells. b_7 is the rate of Beta cell decline.

In 2019, Ali and Tahir proposed a new model representing the interaction of insulin, glucose and Beta cells in type 1 diabetes mellitus[6]

$$\left.\begin{aligned} \frac{d\varphi}{dt} &= -\mu_1\varphi + \mu_2\varnothing + \mu_3\varnothing^2 + \mu_4\varnothing^3 + \mu_5\rho + \mu_6\rho^2 + \mu_7\rho^3 + \mu_{18} \\ \frac{d\varnothing}{dt} &= -\mu_8\varphi - \mu_9\varphi^2 - \mu_{10}\varphi^3 - \mu_{11}\rho - \mu_{12}\rho^2 - \mu_{13}\rho^3 + \mu_{19} \\ \frac{d\rho}{dt} &= \mu_{14}\varnothing + \mu_{15}\varnothing^2 + \mu_{16}\varnothing^3 - \mu_{17}\rho \end{aligned}\right\}, \tag{3.4}$$

where, μ_1 is the reduction rate of the insulin concentration, which is based on its current level. μ_2, μ_3 and μ_4 are the increased rates of insulin when the glucose concentration increases. μ_5, μ_6 and μ_7 show the increased rate of insulin concentration when the Beta cells' level increases. μ_8, μ_9 and μ_{10} are the rates of glucose reduction in response to increasing insulin levels. μ_{11}, μ_{12} and μ_{13} are the reduction rates of glucose concentration because of Beta cells' activity. μ_{14}, μ_{15} and μ_{16} the rate of increase in Beta cells due to the increase in glucose concentration. μ_{17} the rate of decrease in Beta cells due to its current level. μ_{18} the decrease rate of Beta cells and μ_{19} the increase rate of glucose in the absence of insulin and Beta cells.

3.3. Constructing a new model representing the dynamics of type 1 diabetes mellitus with treatment

In this section, we will review our development of the mathematical model (3.4) by adding to it a new treatment, CID661578, to study its effect on the process of regulating insulin, glucose, and Beta cells in type 1 diabetes, based on the following assumptions:

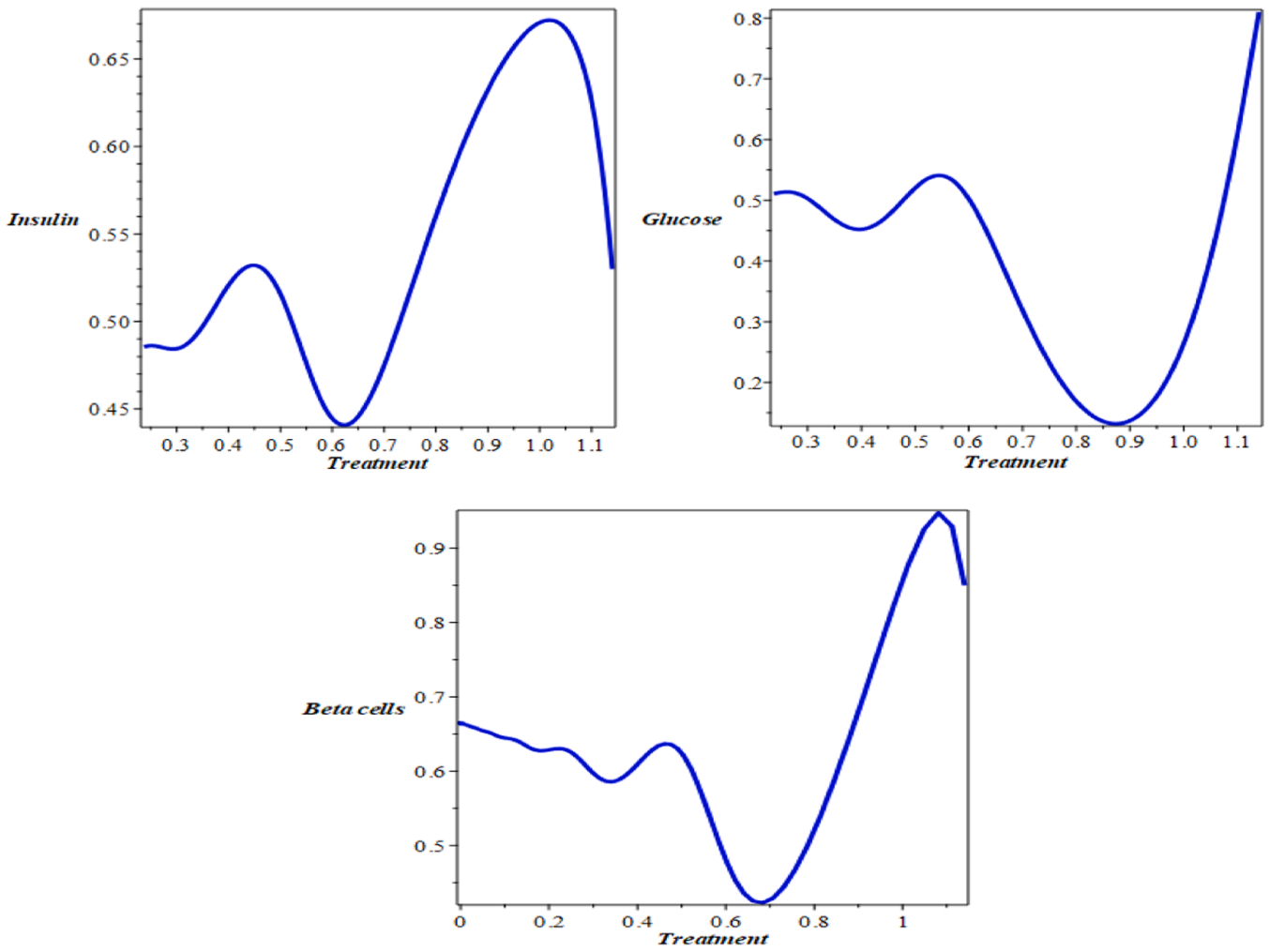

Fig. 5. Dynamics of treatment with insulin, glucose, and Beta cells for system (3.5), for the data given in Table 1.

A) Based on the prey and predator model, we assume that the treatment is the predator and the glucose is the prey, and on this basis, we will add another equation that represents the change in the concentration of treatment over time.
B) Assuming that the treatment positively affects Beta cells and contributes to their activation, which increases insulin secretion and thus reduces glucose concentration, we can add the following parameters to the form:
   - Coefficient governing the effect of treatment on Beta cell activation.
   - Coefficient representing treatment and its positive effect on insulin secretion from Beta cells.
   - Coefficient representing treatment and its effect in reducing glucose.

Once these coefficients are added, we can provide a better representation of the effect of treatment on Beta cells and the effect on insulin secretion and glucose concentration in the model.

Depending on the above hypotheses, the new mathematical model can be represented as follows

$$\left.\begin{aligned}
\frac{d\varphi}{dt} &= -\mu_1\varphi + \mu_2\varnothing + \mu_3\varnothing^2 + \mu_4\varnothing^3 + \mu_5\rho + \mu_6\rho^2 + \mu_7\rho^3 + \mu_{18} + \mu_{20}\varphi\tau \\
\frac{d\varnothing}{dt} &= -\mu_8\varphi - \mu_9\varphi^2 - \mu_{10}\varphi^3 - \mu_{11}\rho - \mu_{12}\rho^2 - \mu_{13}\rho^3 + \mu_{19} - \mu_{21}\varnothing\tau \\
\frac{d\rho}{dt} &= \mu_{14}\varnothing + \mu_{15}\varnothing^2 + \mu_{16}\varnothing^3 - \mu_{17}\rho + \mu_{22}\rho\tau \\
\frac{d\tau}{dt} &= \mu_{23}\varnothing\tau - \mu_{24}\tau
\end{aligned}\right\}, \tag{3.5}$$

where, $\tau(t)$ represents the concentration of treatment. μ_{20} the rate of increase in insulin secretion under the influence of treatment. μ_{21} the rate of decrease in glucose concentration under the influence of treatment. μ_{22} increased Beta cell count in response to treatment. μ_{23} the prevalence of treatment in the case of increased glucose concentration. μ_{24} the rate of glucose decrease in the presence of treatment. Thus, the dynamics of diabetes and its treatment can be described in system (3.5) in Fig. 1.

4. Application of SAGPM to the mathematical model

Assume that we have a system (3.5), with initial conditions $\varphi_0 = 0.53$, $\varnothing_0 = 0.81$, $\rho_0 = 0.85$, $\tau_0 = 1.14$, and the parameter values in Table 1.

By taking the Shehu transformation on both sides of (3.5), we obtain

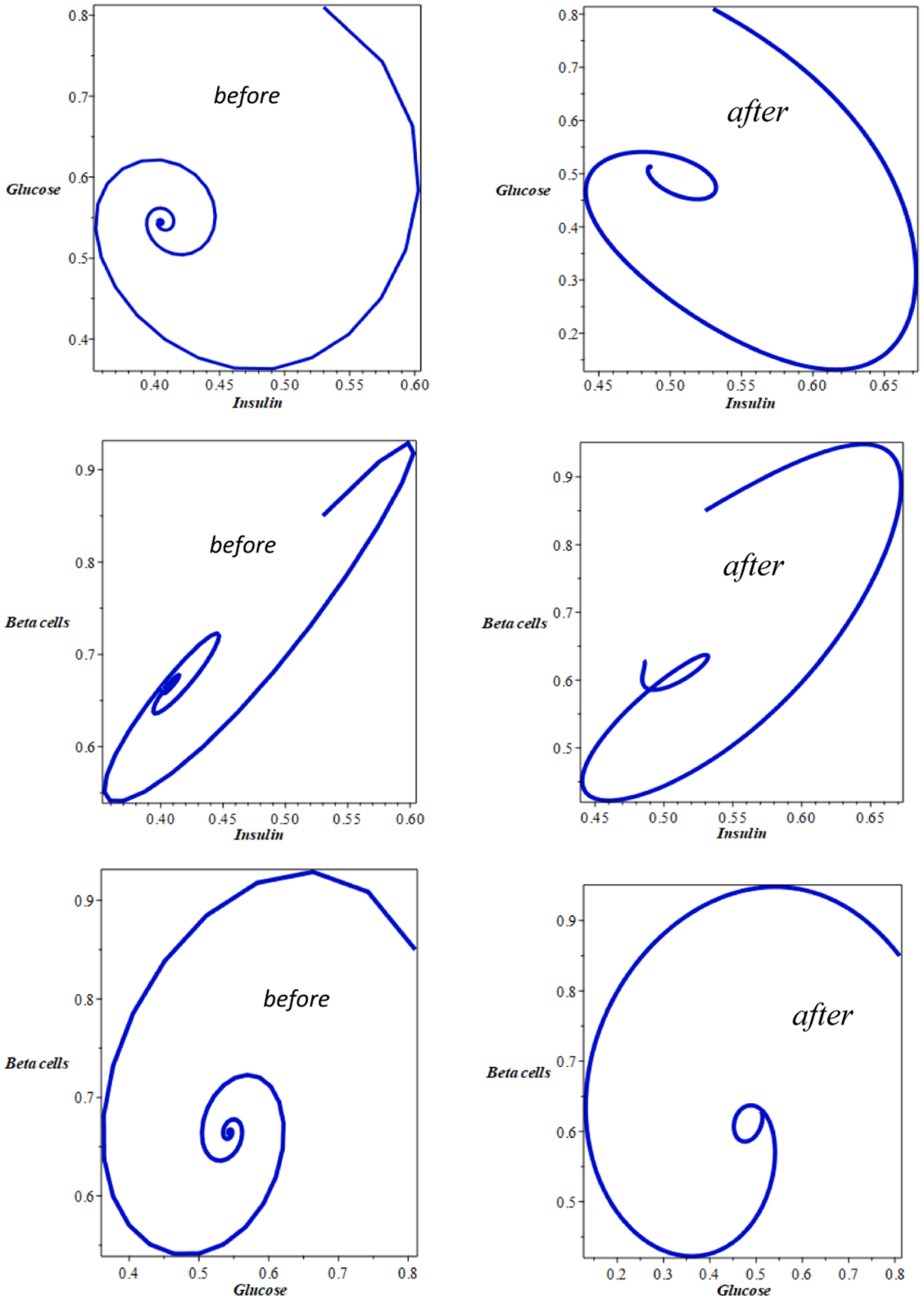

Fig. 6. Phase plane before and after treatment, for the data given in Table 1.

$$\left.\begin{aligned}
\varphi^*(u,s) &= \frac{0.53\ u}{s} + \frac{u}{s}S\begin{pmatrix} -\mu_1\varphi + \mu_2\varnothing + \mu_3\varnothing^2 + \mu_4\varnothing^3 + \\ \mu_5\rho + \mu_6\rho^2 + \mu_7\rho^3 + \mu_{18} + \mu_{20}\varphi\tau \end{pmatrix} \\
\varnothing^*(u,s) &= \frac{0.81\ u}{s} + \frac{u}{s}S\begin{pmatrix} -\mu_8\varphi - \mu_9\varphi^2 - \mu_{10}\varphi^3 - \mu_{11}\rho - \\ \mu_{12}\rho^2 - \mu_{13}\rho^3 + \mu_{19} - \mu_{21}\varnothing\tau \end{pmatrix} \\
\rho^*(u,s) &= \frac{0.85\ u}{s} + \frac{u}{s}S\begin{pmatrix} \mu_{14}\varnothing + \mu_{15}\varnothing^2 + \mu_{16}\varnothing^3 \\ -\mu_{17}\rho + \mu_{22}\rho\tau \end{pmatrix} \\
\tau^*(u,s) &= \frac{1.14\ u}{s} + \frac{u}{s}S(\mu_{23}\varnothing\tau - \mu_{24}\tau)
\end{aligned}\right\}, \tag{4.1}$$

where $\varphi^*(u,s) = S\{\varphi(t)\}$, $\varnothing^*(u,s) = S\{\varnothing(t)\}$, $\rho^*(u,s) = S\{\rho(t)\}$, and $\tau^*(u,s) = S\{\tau(t)\}$

Taking the inverse Shehu transformation on both sides of (4.1), we get,

$$\left.\begin{aligned}
\varphi(\mathrm{t}) &= 0.53 + \mathrm{S}^{-1}\left(\frac{\mathrm{u}}{\mathrm{s}}\,\mathrm{S}\begin{pmatrix} -\mu_1\varphi + \mu_2\varnothing + \mu_3\varnothing^2 + \mu_4\varnothing^3 + \\ \mu_5\rho + \mu_6\rho^2 + \mu_7\rho^3 + \mu_{18} + \mu_{20}\varphi\tau \end{pmatrix}\right) \\
\varnothing(\mathrm{t}) &= 0.81 + \mathrm{S}^{-1}\left(\frac{\mathrm{u}}{\mathrm{s}}\,\mathrm{S}\begin{pmatrix} -\mu_1\varphi + \mu_2\varnothing + \mu_3\varnothing^2 + \mu_4\varnothing^3 + \\ \mu_5\rho + \mu_6\rho^2 + \mu_7\rho^3 + \mu_{18} + \mu_{20}\varphi\tau \end{pmatrix}\right) \\
\rho(\mathrm{t}) &= 0.85 + \mathrm{S}^{-1}\left(\frac{\mathrm{u}}{\mathrm{s}}\,\mathrm{S}\left(\mu_{14}\varnothing + \mu_{15}\varnothing^2 + \mu_{16}\varnothing^3 - \mu_{17}\rho + \mu_{22}\rho\tau\right)\right) \\
\tau(\mathrm{t}) &= 1.14 + \mathrm{S}^{-1}\left(\frac{\mathrm{u}}{\mathrm{s}}\,\mathrm{S}(\mu_{23}\varnothing\tau - \mu_{24}\tau)\right)
\end{aligned}\right\} \tag{4.2}$$

By AGM, we substitute (2.18) into (4.2), and we obtain

$$\left.\begin{aligned}
\sum_{i=0}^{n} a_i\,t^i &= 0.53 + \mathrm{S}^{-1}\left(\frac{\mathrm{u}}{\mathrm{s}}\,\mathrm{S}\begin{pmatrix} -\mu_1\sum_{i=0}^{n} a_i\,t^i + \mu_2\sum_{i=0}^{n} b_i\,t^i + \mu_3\left(\sum_{i=0}^{n} b_i\,t^i\right)^2 + \\ \mu_4\left(\sum_{i=0}^{n} b_i\,t^i\right)^3 + \mu_5\sum_{i=0}^{n} c_i\,t^i + \mu_6\left(\sum_{i=0}^{n} c_i\,t^i\right)^2 + \\ \mu_7\left(\sum_{i=0}^{n} c_i\,t^i\right)^3 + \mu_{18} + \mu_{20}\sum_{i=0}^{n} a_i\,t^i * \sum_{i=0}^{n} d_i\,t^i \end{pmatrix}\right) \\
\sum_{i=0}^{n} b_i\,t^i &= 0.81 + \mathrm{S}^{-1}\left(\frac{\mathrm{u}}{\mathrm{s}}\,\mathrm{S}\begin{pmatrix} -\mu_1\sum_{i=0}^{n} a_i\,t^i + \mu_2\sum_{i=0}^{n} b_i\,t^i + \mu_3\left(\sum_{i=0}^{n} b_i\,t^i\right)^2 + \\ \mu_4\left(\sum_{i=0}^{n} b_i\,t^i\right)^3 + \mu_5\sum_{i=0}^{n} c_i\,t^i + \mu_6\left(\sum_{i=0}^{n} c_i\,t^i\right)^2 + \\ \mu_7\left(\sum_{i=0}^{n} c_i\,t^i\right)^3 + \mu_{18} + \mu_{20}\sum_{i=0}^{n} a_i\,t^i * \sum_{i=0}^{n} d_i\,t^i \end{pmatrix}\right) \\
\sum_{i=0}^{n} c_i\,t^i &= 0.85 + \mathrm{S}^{-1}\left(\frac{\mathrm{u}}{\mathrm{s}}\,\mathrm{S}\begin{pmatrix} \mu_{14}\sum_{i=0}^{n} b_i\,t^i + \mu_{15}\left(\sum_{i=0}^{n} b_i\,t^i\right)^2 + \mu_{16}\left(\sum_{i=0}^{n} b_i\,t^i\right)^3 \\ -\mu_{17}\sum_{i=0}^{n} c_i\,t^i + \mu_{22}\sum_{i=0}^{n} c_i\,t^i * \sum_{i=0}^{n} d_i\,t^i \end{pmatrix}\right) \\
\sum_{i=0}^{n} d_i\,t^i &= 1.14 + \mathrm{S}^{-1}\left(\frac{\mathrm{u}}{\mathrm{s}}\,\mathrm{S}\left(\mu_{23}\sum_{i=0}^{n} b_i\,t^i * \sum_{i=0}^{n} d_i\,t^i - \mu_{24}\sum_{i=0}^{n} d_i\,t^i\right)\right)
\end{aligned}\right\} \tag{4.3}$$

When $n = 3$, after simplifying and substituting the values of the parameters in Table 1, Eq. (4.3) becomes:

$$\begin{aligned} f(\varphi(\mathrm{t})) &= 0.83 + 0.30\ x - 0.69\ y - 0.27\ y^2 - 0.92y^3 - 0.98z + 0.62z^2 \\ &\quad + 0.10z^3 - 0.66rx \\ &= 0, \end{aligned} \tag{4.4}$$

$$\begin{aligned} g(\varnothing(\mathrm{t})) &= -1.93 + 1.24x - 1.06x^2 - 0.29x^3 + 1.95z + 0.75z^2 - 0.02z^3 \\ &\quad + 0.88ry \\ &= 0 \end{aligned} \tag{4.5}$$

$$h(\rho(\mathrm{t})) = -0.83y - 1.01y^2 - 1.25y^3 + 1.43z - 0.4rz = 0, \tag{4.6}$$

$$j(\tau(\mathrm{t})) = -0.2ry + 0.4r = 0, \tag{4.7}$$

where, $x = a_0 + a_1t + a_2t^2 + a_3t^3$, $y = b_0 + b_1t + b_2t^2 + b_3t^3$, $z = c_0 + c_1t + c_2t^2 + c_3t^3$, and $r = d_0 + d_1t + d_2t^2 + d_3t^3$.

The constant coefficients of Eqs. (4.4)–(4.7) $\{a_0, \ldots, a_3, b_0, \ldots b_3, c_0, \ldots, c_3$ *and* $d_0, \ldots, d_3\}$ can be computed by applying initial conditions:

$$\begin{aligned} \varphi(t=0) = 0.53 \rightarrow a_0 &= 0.53, \varnothing(t=0) = 0.81 \rightarrow b_0 = 0.81, \rho(t=0) \\ = 0.85 \rightarrow c_0 &= 0.85, \ \textit{and}\ \tau(t=0) = 1.14 \rightarrow d_0 = 1.14 \end{aligned}$$

On both Eqs. (4.4)–(4.7) and its derivatives, the initial conditions are applied as follows:

$$f(\varphi(t=0)): \ 0.83 + a_1 + 0.30a_0 - 0.69b_0 - 0.27b_0^2 - 0.92b_0^3 - 0.98c_0 + 0.62c_0^2 + 0.10c_0^3 - 0.66d_0a_0 = 0,$$

$$g(\varnothing(t=0)): -1.93 + b_1 + 1.24a_0 - 1.06a_0^2 - 0.29a_0^3 + 1.95c_0 + 0.75c_0^2 - 0.02c_0^3 + 0.88d_0b_0 = 0,$$

$$h(\rho(t=0)): c_1 - 0.83b_0 - 1.01b_0^2 - 1.25b_0^3 + 1.43c_0 - 0.4d_0c_0 = 0,$$

$$j(\tau(t=0)): d_1 - 0.2d_0b_0 + 0.4d_0 = 0,$$

$$f'(\varphi(t=0)): \ 2a_2 + 0.30a_1 - 0.69b_1 - 0.54b_0b_1 - 2.76b_0^2b_1 - 0.98c_1 + 1.24c_0c_1 + 0.30c_0^2c_1 - 0.66(d_1a_0 + d_0a_1) = 0,$$

$$g'(\varnothing(t=0)): \ 2b_2 + 1.24a_1 - 2.12a_0a_1 - 0.87a_0^2a_1 + 1.95c_1 + 1.50c_0c_1 - 0.06c_0^2c_1 + 0.88(d_1b_0 + d_0b_1) = 0,$$

$$h'(\rho(t=0)): \ 2c_2 - 0.83b_1 - 2.02b_0b_1 - 3.75b_0^2b_1 + 1.43c_1 - 0.4(d_1c_0 + d_0c_1) = 0,$$

$$j'(\tau(t=0)): \ 2d_2 - 0.2d_1b_0 - 0.2d_0b_1 + 0.4d_1 = 0,$$

$$f''(\varphi(t=0)): \ 6a_3 + 0.60a_2 - 1.38b_2 - 0.54b_1^2 - 1.08b_0b_2 - 5.52b_0b_1^2 - 5.52b_0^2b_2 - 1.96c_2 + 1.24c_1^2 + 2.48c_0c_2 + 0.60\left(c_0c_1^2 + c_0^2c_2\right) - 1.32(d_2a_0 + d_1a_1 + d_0a_2) = 0,$$

$$g''(\varnothing(t=0)): \ 6b_3 + 2.48a_2 - 2.12a_1^2 - 4.24a_0a_2 - 1.74a_0a_1^2 - 1.74a_0^2a_2 + 3.90c_2 + 1.50c_1^2 + 3.00c_0c_2 - 0.12\left(c_0c_1^2 + c_0^2c_2\right) + 1.76(d_2b_0 + d_1b_1 + d_0b_2) = 0,$$

$$h''(\rho(t=0)): \ 6c_3 - 1.66b_2 - 2.02b_1^2 - 4.04b_0b_2 - 7.50\left(b_0b_1^2 + b_0^2b_2\right) + 2.86c_2 - 0.8(d_2c_0 + d_1c_1 + d_0c_2) = 0,$$

$$j''(\tau(t=0)): \ 6d_3 - 0.4(d_2b_0 + d_1b_1 + d_0b_2) + 0.8d_2 = 0.$$

With the help of Maple software, unknown constant coefficients a_i and b_i can now be found by solving the above equations.

Then the solutions are

$$\varphi(t) = 0.53 - 0.95838222t - 2.037087753t^2 + 0.2262837699t^3,$$

$$\varnothing(t) = 0.81 - 1.38595617t - 1.010209973t^2 + 4.591124597t^3,$$

$$\rho(t) = 0.85 + 1.171362250t - 4.030586325t^2 + 2.184595929t^3,$$

$$\tau(t) = 1.14 - 0.27132t - 0.1257119234t^2 + 0.0417336368t^3.$$

Now, taking the Padé approximation, with $l=1$, $m=2$ for $\varphi(t)$, $l=0$, $m=3$ for $\varnothing(t)$, $l=1$, $m=2$ for $\rho(t)$ and $l=2$, $m=1$ for $\tau(t)$, we obtain the solutions of the system as follows:

$$\varphi_{[1,2]} = \frac{0.5299999 + 1.508033182t}{1.0 + 1.037077287t + 1.968247775t^2},$$

$$\varnothing_{[0,3]} = \frac{0.81}{0.999999 + 1.711057t + 4.174888864t^2 + 3.609401517t^3},$$

$$\rho_{[1,2]} = \frac{0.85 + 2.336713083t}{1.0 + 1.371000979t + 2.852526509t^2},$$

$$\tau_{[2,1]} = \frac{1.140 - 0.6497753180t - 0.03563955764t^2}{1.0 - 0.3319783491t}.$$

5. Results and discussion

In this section, we discuss the numerical computations, which have been obtained by the application of SAGPM for solving the new mathematical model. All calculations are run by Maple 2016 software. Tables 2 and 3 show the comparison between the new method SAGPM, AGM and Adomian decomposition method (ADM) by measurements of error and CPU time. We note from the error tables that the new method is more accurate and efficient than the AGM and ADM with a lower CPU time. Fig. 2 shows the comparison between the solution offered by the new method SAGPM and AGM for system (3.5), with the parameters given in Table 1. where; the measurement errors are defined as:

$$\| E\|_{L_2} = \sqrt{h\sum_{i=0}^{n}|x_{i+1} - x_i|^2}, \ \| E\|_{L_\infty} = max_{i=0..n}(|x_{i+1} - x_i|)$$

* For a more detailed understanding of this method and its algorithm, it is recommended to refer to.[32]

Fig. 3 shows the insulin and glucose concentrations before appropriate diabetes treatment. There may be an insufficient role for pancreatic Beta cells in the secretion of insulin. Consequently, the concentration of insulin in the blood decreases and the concentration of glucose increases, which leads to an increase in the level of sugar in the blood. Fig. 4 shows the effect of treatment on insulin and glucose concentrations. Through the graph, we can see that when the level of glucose in the body rises, insulin secretion increases at the same time. In addition, we observed a significant correlation between the curve representing glucose levels and the curve representing insulin levels in the body, which indicates that insulin begins to be secreted quickly and in greater quantities once blood glucose rises. This enables the body to efficiently manage the high sugar level and maintain it within the normal range. This rapid and effective insulin response acts as a positive indicator of the health and functioning of the pancreas and blood sugar control system. In general, the closer the correlation between the two curves is, the more effectively the body can handle fluctuations in glucose levels and improve blood sugar regulation. This is attributed to the role of Beta cells and their enhanced activity under the influence of additive treatment. Over time, we observed that the curves representing glucose, insulin, Beta cells, and treatment appeared to be linear. This indicates that the body maintains a stable pattern in regulating blood sugar levels.

6. Convergence analysis of SAGPM

This part illustrates how the approximate analytical solutions obtained from using SAGPM for system (3.5) converge.

Consider the system of equations in the following form:

$$\left.\begin{aligned} \varphi &= A(\varphi, \varnothing, \rho, \tau) \\ \varnothing &= B(\varphi, \varnothing, \rho, \tau) \\ \rho &= C(\varphi, \varnothing, \rho, \tau) \\ \tau &= D(\varphi, \varnothing, \rho, \tau) \end{aligned}\right\}, \tag{6.1}$$

where, A, B, C and D are nonlinear operators. The solution by using the present approach is equivalent to the following sequence:

$$\left.\begin{aligned} S_n &= \sum_{j=0}^{n} \varphi_j = \sum_{j=0}^{n} a_j \frac{t^j}{(j)!} \\ K_n &= \sum_{j=0}^{n} \varnothing_j = \sum_{j=0}^{n} b_j \frac{t^j}{(j)!} \\ R_n &= \sum_{j=0}^{n} \rho_j = \sum_{j=0}^{n} c_j \frac{t^j}{(j)!} \\ M_n &= \sum_{j=0}^{n} \tau_j = \sum_{j=0}^{n} d_j \frac{t^j}{(j)!} \end{aligned}\right\}. \tag{6.2}$$

Theorem (6.1). (Convergence of System).[33] Let H be a Hilbert space, let A, B, C and D be operators from H into H, and let $\varphi, \varnothing, \rho$ and τ be the exact solution of system (6.1).

$$J = \begin{pmatrix} -\mu_1 + \mu_{20}\tau & \mu_2 + 2\mu_3\varnothing + 3\mu_4\varnothing^2 & \mu_5 + 2\mu_6\rho + 3\mu_7\rho^2 & \mu_{20}\varphi \\ -\mu_8 - 2\mu_9\varphi - 3\mu_{10}\varphi^2 & -\mu_{21}\tau & -\mu_{11} - 2\mu_{12}\rho - 3\mu_{13}\rho^2 & -\mu_{21}\varnothing \\ 0 & \mu_{14} + 2\mu_{15}\varnothing + 3\mu_{16}\varnothing^2 & -\mu_{17} + \mu_{22}\tau & \mu_{22}\rho \\ -\mu_{24}\tau & \mu_{23}\tau & 0 \; \mu_{23}\varnothing - \mu_{24} & \end{pmatrix}.$$

The approximate solutions

$$\left.\begin{aligned} \sum_{j=0}^{\infty} \varphi_j &= \sum_{j=0}^{\infty} a_j \frac{t^j}{(j)!} \\ \sum_{j=0}^{\infty} \varnothing_j &= \sum_{j=0}^{\infty} b_j \frac{t^j}{(j)!} \\ \sum_{j=0}^{\infty} \rho_j &= \sum_{j=0}^{\infty} c_j \frac{t^j}{(j)!} \\ \sum_{j=0}^{\infty} \tau_j &= \sum_{j=0}^{\infty} d_j \frac{t^j}{(j)!} \end{aligned}\right\}. \tag{6.3}$$

are convergence to exact solutions $\varphi, \varnothing, \rho$ and τ respectively, for every $j \in N \cup \{0\}$, $\exists\ 0 \le \sigma_{1,2,3,4} < 1$, $\| \varphi_{j+1} \| \le \sigma_1 \| \varphi_j \|$, $\| \varnothing_{j+1} \| \le \sigma_2 \| \varnothing_j \|$, $\| \rho_{j+1} \| \le \sigma_3 \| \rho_j \|$, and $\| \tau_{j+1} \| \le \sigma_4 \| \tau_j \|$.

Definition (6.2). For every $j \in N \cup \{0\}$, we define

$$\sigma_1^j = \begin{cases} \dfrac{\| \varphi_{j+1} \|}{\| \varphi_j \|}, & \| \varphi_j \| \neq 0 \\ 0, \ otherwise \end{cases}, \quad \sigma_2^j = \begin{cases} \dfrac{\| \varnothing_{j+1} \|}{\| \varnothing_j \|}, & \| \varnothing_j \| \neq 0 \\ 0, \ otherwise \end{cases}, \quad \sigma_3^j = \begin{cases} \dfrac{\| \rho_{j+1} \|}{\| \rho_j \|}, & \| \rho_j \| \neq 0 \\ 0, \ otherwise \end{cases}$$

$$\sigma_4^j = \begin{cases} \dfrac{\| \tau_{j+1} \|}{\| \tau_j \|}, & \| \tau_j \| \neq 0 \\ 0, \ otherwise \end{cases}$$

where,

$$\| \cdot \| = \sqrt{\sum_{i=0}^{n} |()_i|^2}.$$

Corollary (6.3). From Theorem (6.1), system (6.3) converges to exact solutions when

$$0 \le \sigma_{1,2,3,4}^j < 1.$$

From Table 4, we note that the convergence of the approximate solutions of the proposed system is achieved, and with increasing iterations, the convergence becomes faster.

7. Stability analysis

The stable mathematical model allows us to assess treatment efficacy and interpret how it affects symptoms associated with diabetes, such as the regulation of blood glucose and insulin concentration. The analysis can guide us in making the right decisions regarding the use of treatment. The stability analysis of system (3.5) is discussed as follows:

The dynamic behavior of system solutions can be determined by finding the eigenvalues of the corresponding Jacobian matrix at each equilibrium point, and they are given in the following form

When we talk about the stability of a system, we refer to its ability to regain equilibrium after small disturbances. Generally, positive fixed points are of interest in stability analysis because they represent desirable equilibrium states. System (3.5) has one positive fixed point, which is given in Table 5.

The relationship between treatment and the levels of insulin, glucose, and Beta cells in the mathematical model studied to represent diabetes represents an important source for understanding the effect of treatment on the body, and on the other hand, helps us determine the optimal concentrations of treatment. Noticeable changes in the body's response to treatment can be seen in Fig. 5. It is worth noting that this response is not constant over time or for all treatment values, and depends on factors such as dose, health condition, and time. Therefore, this change in the curve demonstrates the importance of careful monitoring and adjusting the use of treatment according to the needs of each patient. The results indicate that the equilibrium point in our mathematical model, was relatively unstable before the application of treatment. This instability is evident from the frequent oscillations around the equilibrium point. Conversely, when treatment was applied to the model, a significant reduction in the frequency of these oscillations was observed. This reduction suggests that the treatment contributed to improving the system's stability. This improvement can be explained by the treatment's role in reducing fluctuations in insulin and glucose levels, making the system more sustainable and less susceptible to undesirable changes, which was particularly evident in Fig. 6.

8. Conclusion

Based on our study, we have drawn several significant conclusions as follows:

1. We have successfully developed a comprehensive mathematical model representing type 1 diabetes, incorporating essential factors such as blood glucose levels, insulin levels, Beta cell response and treatment.
2. Our mathematical model demonstrates that the stimulation of Beta cells in the pancreas results in a substantial increase in insulin

secretion. This heightened insulin secretion effectively regulates and reduces glucose levels in the body. These findings align seamlessly with clinical studies and laboratory experiments, validating the efficacy of this treatment approach.
3. Through stability analysis, we confirmed the robustness of our proposed model, implying consistent and stable blood glucose levels over time.
4. Our results unequivocally establish the effectiveness and efficiency of our novel method in solving the proposed model, showcasing its potential for practical applications.
5. The error tables provide compelling evidence that our new method surpasses AGM and ADM, exhibiting fewer errors and reduced CPU time. It can be regarded as an advancement over the AGM method.
6. Furthermore, we achieved excellent convergence of the approximate analytical solutions generated by applying our new algorithm.

This study holds practical promise, as it signifies a significant leap forward in the treatment of type 1 diabetes, potentially enhancing the quality of care and disease management. As we look ahead, our research paves the way for the exploration of real-world applications and the development of tangible solutions for future work.

Moreover, the importance of utilizing this method lies in its potential to revolutionize diabetes treatment. Refining our understanding of disease dynamics and treatment effects opens doors to more effective and innovative therapies. We anticipate that this novel approach will not only benefit individuals with type 1 diabetes but also serve as a stepping stone for further advancements in the field, offering hope and improved outcomes for patients in the future.

Declaration of Competing Interest

I hereby to certify that there is no conflict of interest regarding our paper "Development and simulation of a mathematical model representing the dynamics of Type 1 diabetes mellitus with treatment". Also this manuscript has not been published before. I interest the copy right for your journal.

Data availability

The authors do not have permission to share data.

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