



Available online at www.qu.edu.iq/journalcm

JOURNAL OF AL-QADISIYAH FOR COMPUTER SCIENCE AND MATHEMATICS

ISSN:2521-3504(online) ISSN:2074-0204(print)



A Two-Dimensional Reaction-Diffusion Model for Tumor-Drug Interaction: Analytical Solution via Triple Laplace Transform and Accelerated Adomian Method

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ARTICLE INFO

Article history:

Received: 09 /05/2026

Revised form: 03 /06/2026

Accepted : 07 /06/2026

Available online: 30 /09/2026

Keywords:

Mathematical modeling; reaction-diffusion model; drug diffusion; tumor growth; nonlinear partial differential equations; tumor-drug interaction

ABSTRACT

Mathematical modeling emerged as an important tool for understanding the dynamics of tumors and improving strategies for drug delivery. In this study, we constructed a two-dimensional reaction-diffusion model of a chemotherapeutic agent and a proliferating tumor which describes their spatiotemporal interaction. The model captures nonlinear decay of the tumor due to drug effect, logistic growth of the tumor up to a carrying capacity, and biological diffusion for both species. The solution is found using a hybrid approach which consists of the triple Laplace transform in space and time along with a modified Accelerated Adomian Decomposition Method (AADM). We start our analysis with bio plausible initial conditions which arise from a parabolic profile to simulate localized drug infusion and study the dynamics of drug concentration as well as the tumor concentration for a variety of parameters. Our findings illustrate the existence of critical drug and tumor suppression levels, and reinforce the nonlinearity of treatment response with diffusion, reaction parameters, and their efficacy. The analysis in this study is a step towards formulating clinically relevant solutions, optimizing treatment designs, and serving as a starting point for more bio realistic model additions.

MSC..

<https://doi.org/10.29304/jqcm.2026.18.32873>

1. Introduction

Over the last few decades, there have been advancements in the application of mathematical modeling in intricate areas of biology, specifically in analyzing the growth of tumors and the effectiveness of chemotherapy treatments [1,2]. The interactions of the drug and the tumor are now described using partial differential equations (PDEs), which makes the mathematical relationships more manageable to analyze and simulate. These models make

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it possible to predict the future behavior of the tumor in response to therapy and let clinicians evaluate the effect of different physiological and pharmacological variables on the therapeutic outcome [3]. Among these models, reaction–diffusion equations have emerged as a powerful tool, integrating temporal changes in concentrations with spatial dispersion across tissue domains [4,5]. They accurately represent the temporal evolution of the tumor response, the propagation of the drug, including the cytotoxic effects, and the tumor growth that is limited logistically. Here, we present a two-dimensional mathematical model and a two-dimensional model describing the nonlinear interaction of a diffusing drug and a proliferating tumor, including the processes of diffusion, nonlinear biochemical interaction, and logistic tumor growth. To compute analytical solutions, we introduce a hybrid approach based on the triple Laplace transform (in time and space) combined with the Accelerated Adomian Decomposition Method (AADM). This approach is particularly advantageous compared to numerical schemes which can be expensive or sensitive to the grid [6]. At the focus of this study is the precise monitoring of the interactions, continuous and dynamic, of the biologically motivated parabolic initial conditions, reflecting the centroid of a drug injection and the tumor. In addition, we focus on two distinct parameter values of the same biological significance that allow a relative treatment and mathematical analysis of the efficiency of the treatment and the evaluation of the analytical framework of the strategic therapy [7,8]. Our modeling framework incorporates tumor growth and drug diffusion literature, including the application of classic reaction–diffusion systems to glioma modeling, drug transport with tumor kill kinetics, and more recently, the application of Laplace–Adomian techniques to nonlinear PDEs [9]. This integrated approach improves interpretability and strengthens the framework, which allows for modeling on a single patient level for potential future applications.

2. Mathematical Model and Initial Conditions

This research presents a two-dimensional mathematical framework of the nonlinear interaction-diffusion type that captures the time-varying interaction between a therapeutic chemical agent and tumor cell proliferation within a limited biological domain. The dynamics are modeled by a coupled system of partial differential equations that integrates diffusion, interaction, and cell growth mechanisms. This construction allows for a quantitative description of spatial and temporal variations in concentrations, enabling the evaluation of the effectiveness of therapeutic interventions:

$$\begin{aligned}\frac{\partial u}{\partial t} &= D_u \nabla^2 u - \beta uv^2 \\ \frac{\partial v}{\partial t} &= D_v \nabla^2 v + rv\left(1 - \frac{v}{k}\right) - \alpha uv^2\end{aligned}\quad (1)$$

$u(x, y, t)$: concentration of the chemotherapeutic drug,
 $v(x, y, t)$: density of tumor cells,
 D_u, D_v : diffusion coefficients for the drug and tumor respectively,
 β : drug consumption rate by tumor interaction,
 α : nonlinear cytotoxic effect of the drug,
 r : intrinsic tumor growth rate,
 k : environmental carrying capacity for the tumor.

The model captures the way a drug gradually spreads through tissue and interacts with tumor cells in a way that becomes progressively less efficient. At the same time, the tumor expands according to a logistic curve, yet its growth is restrained by the toxic effects of the drug. To completely define the model, we impose the following initial conditions inspired by biological observations at $t=0$:

$$\begin{aligned}u(x, y, 0) &= 1 - (x - 0.5)^2 - (y - 0.5)^2 \\ v(x, y, 0) &= 1 - (x - 0.5)^2 - (y - 0.5)^2\end{aligned}\quad (2)$$

We fix the distribution at the point (0.5,0.5) to form a symmetrical shape with a peak and decreasing edges. Biologically, this represents the concentration of the drug in a tumorous region.

We choose $(x,y) \in [0,1] \times [0,1]$. The point we choose biologically represents a tissue segment. By choosing this tissue range, we can track how the concentrations change, and this choice ensures that the values remain positive without the appearance of negative values.

3. Accelerated Adomian decomposition method (AADM):

The Adomian method is an important method for solving nonlinear differential equations, and the essence of the method is to make the solution and the nonlinear terms converge in a series. We write the solution [10]

$$u = \sum_{n=0}^{\infty} u_n \quad (3)$$

and the nonlinearity

$$N(u) = \sum_{n=0}^{\infty} A_n \quad (4)$$

where A_n The Adomian polynomial represents a polynomial and has generative formulas through which it is created. [11]:

$$A_n = \frac{1}{n!} \frac{d^n}{d\lambda^n} N\left(\sum_{k=0}^{\infty} \lambda^k u_k\right) \Big|_{\lambda=0} \quad (5)$$

By reformulating the linear operator equation

$$Lu = N(u) - Ru - g \quad (6)$$

To improve convergence, Kalle proposed the formula [12,13]:

$$s_n = \sum_{i=0}^n u_i \quad (7)$$

$$A_n = N(s_n) - \sum_{i=0}^{n-1} A_i \quad (8)$$

This formula contributed to accelerating convergence and reducing computational steps. While the Adomian method alone and the Laplace method alone differ in their efficiency, combining them enhances efficiency and numerical stability, thus yielding solutions closer to accuracy. This combination makes it suitable for solving complex nonlinear differential equations.

4. Triple Laplace transform

The Laplace transform is applied to three-dimensional partial differential equations (one time dimension and two spatial dimensions), where the transformation scope is extended to three variables: s , p , and q . It is defined for a function $f(x, y, t)$ by the formula [14]:

$$F(p, q, s) = \iiint_0^{\infty} e^{-px} e^{-qy} e^{-st} f(x, y, t) dx dy dt \quad (9)$$

By shifting this transformation from differential representation to algebraic preference, finding solutions becomes easier. Therefore, the triplet transformation of the APLAS is a powerful and efficient tool for solving complex differential equations and initial and boundary value problems, and combining it with the ADOMEAN transformation for handling nonlinear boundary conditions accelerates the search for solutions.

Derivatives for the Triple Laplace Transform Starting with the one-dimensional Laplace transform in time only, treating x, y as constant during integration:

$$\mathcal{L}_t\{u_t(x, y, t)\} = \int_0^{\infty} e^{-st} \frac{\partial u}{\partial t}(x, y, t) dt \quad (10)$$

Use integration by parts for equation (10):

$$\mathcal{L}_t\{u_t\} = s \mathcal{L}_t\{u\} - u(x, y, 0) \quad (11)$$

Now apply Laplace transform in x, y to both sides (linearity):

$$\mathcal{L}_{x,y,t}\{u_t\} = s U(p, q, s) - \mathcal{L}_{x,y}\{u(x, y, 0)\} \quad (12)$$

Let:

$$U_0(p, q) := \mathcal{L}_{x,y}\{u(x, y, 0)\} \quad (13)$$

So, the final rule is:

$$\mathcal{L}_{x,y,t}\{u_t\} = s U(p, q, s) - U_0(p, q) \quad (14)$$

Repeat the same idea, or use the known rule:

$$\mathcal{L}_t\{u_{tt}\} = s^2 \mathcal{L}_t\{u\} - s u(x, y, 0) - u_t(x, y, 0) \quad (15)$$

Then apply Laplace transform in x, y :

$$\mathcal{L}_{x,y,t}\{u_{tt}\} = s^2 U(p, q, s) - s U_0(p, q) - U_1(p, q) \quad (16)$$

were

$$U_1(p, q) := \mathcal{L}_{x,y}\{u_t(x, y, 0)\} \quad (17)$$

The general rule (fundamental in all Laplace applications) is:

$$\mathcal{L}_t\left\{\frac{\partial^n u}{\partial t^n}\right\} = s^n \mathcal{L}_t\{u\} - \sum_{k=0}^{n-1} s^{n-1-k} \frac{\partial^k u}{\partial t^k}(x, y, 0) \quad (18)$$

Including Laplace transform in x, y :

$$\mathcal{L}_{x,y,t}\left\{\frac{\partial^n u}{\partial t^n}\right\} = s^n U(p, q, s) - \sum_{k=0}^{n-1} s^{n-1-k} U_k(p, q) \quad (19)$$

where

$$U_k(p, q) := \mathcal{L}_{x,y}\left\{\frac{\partial^k u}{\partial t^k}(x, y, 0)\right\} \quad (20)$$

5. Analytical Methodology: Triple Laplace Transform Combined with Accelerated Adomian Decomposition Method TLT-AAD

In this section, we present an innovative synthesis combining the Triple Laplace Transform (TLT) and the Accelerated Adomian Decomposition Method (AADM) as an analytical framework for solving the proposed interaction and diffusion model without resorting to traditional numerical methods. The Triple Laplace Transform provides a systematic reduction of partial differential equations to simpler algebraic formulas, facilitating mathematical processing and reducing computational overload [14,15]. Meanwhile, the AADM method enables the manipulation of nonlinearity without requiring decomposition of the network or the nonlinearity itself, with significant improvements in convergence and a reduction in the number of approximation limits required. By applying the proposed TLT-AADM formula for tumor-drug interaction.

Taking triple laplace to both sides of equations (1)

$$\begin{aligned}
sU(p, q, s) - U(p, q, 0) &= D_u L_{x,y,t} (u_{xx} + u_{yy}) - \beta L_{x,y,t} (uv^2) \\
sV(p, q, s) - V(p, q, 0) &= D_v L_{x,y,t} (v_{xx} + v_{yy}) + r L_{x,y,t} (v) - \frac{r}{k} L_{x,y,t} (v^2) - \alpha L_{x,y,t} (uv^2)
\end{aligned}
\quad (21)$$

By Accelerated Adomian Decomposition Method (7-8):

$$\begin{aligned}
U(p, q, s) &= \frac{1}{s} U(p, q, 0) + \frac{D_u}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} (u_{n_{xx}} + u_{n_{yy}}) \right) - \frac{\beta}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} A_n \right) \\
V(p, q, s) &= \frac{1}{s} V(p, q, 0) + \frac{D_v}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} (v_{n_{xx}} + v_{n_{yy}}) \right) + \frac{r}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} v_n \right) - \frac{r}{ks} L_{x,y,t} \left(\sum_{n=0}^{\infty} B_n \right) - \frac{\alpha}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} A_n \right)
\end{aligned}$$

Taking triple laplace inverse we get:

$$U_0 = L_{x,y,t}^{-1} \left(\frac{1}{s} U(p, q, 0) \right), \quad V_0 = L_{x,y,t}^{-1} \left(\frac{1}{s} V(p, q, 0) \right) \quad (22)$$

and

$$\begin{aligned}
U_{n+1} &= L_{x,y,t}^{-1} \left(\frac{D_u}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} (u_{n_{xx}} + u_{n_{yy}}) \right) - \frac{\beta}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} A_n \right) \right) \\
V_{n+1} &= L_{x,y,t}^{-1} \left(\frac{D_v}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} (v_{n_{xx}} + v_{n_{yy}}) \right) + \frac{r}{s} L_{x,y,t} \left(\sum_{n=0}^{\infty} v_n \right) - \frac{r}{ks} L_{x,y,t} \left(\sum_{n=0}^{\infty} B_n \right) \right)
\end{aligned}$$

Where:

$$A_n = N_1(s_n) - \sum_{i=0}^{n-1} A_i \quad s_n = \sum_{i=0}^n u_i \quad N_1 = uv^2$$

$$B_n = N_2(s_n) - \sum_{i=0}^{n-1} B_i \quad s_n = \sum_{i=0}^n v_i \quad N_2 = v^2$$

$$A_0 = u_0 v_0^2$$

$$A_1 = 2u_0 v_0 v_1 + u_0 v_1^2 + u_1 v_0^2 + 2u_1 v_0 v_1 + u_1 v_1^2$$

$$B_0 = v_0^2$$

$$B_1 = 2v_0 v_1 + v_1^2$$

for n=3 :

$$u_0 = 1 - (x - 0.5)^2 - (y - 0.5)^2$$

$$v_0 = 1 - (x - 0.5)^2 - (y - 0.5)^2$$

$$\begin{aligned}
u_1 &= -4 D_u t + 0.125 t \alpha (8x^6 + 24x^4 y^2 + 24x^2 y^4 + 8y^6 - 24x^5 - 24x^4 y - 48x^3 y^2 - 48x^2 y^3 - 24xy^4 - \\
&\quad 24y^5 + 12x^4 + 48x^3 y + 24x^2 y^2 + 48xy^3 + 12y^4 + 16x^3 + 16y^3 - 6x^2 - 24xy - 6y^2 - 6x - 6y - 1.) + \\
&\quad 0.5 (2x^2 + 2y^2 - 2x - 2y - 1.)
\end{aligned}$$

$$v_1 =$$

$$\begin{aligned}
&-4 D_v t - 0.5 r (2x^2 + 2y^2 - 2x - 2y - 1.) - 0.25 t r \frac{4x^4 + 8x^2 y^2 + 4y^4 - 8x^3 - 8x^2 y - 8xy^2 - 8y^3 + 8xy + 4x + 4y + 1.}{k} + \\
&0.125 t \alpha (8x^6 + 24x^4 y^2 + 24x^2 y^4 + 8y^6 - 24x^5 - 24x^4 y - 48x^3 y^2 - 48x^2 y^3 - 24xy^4 - 24y^5 + \\
&12x^4 + 48x^3 y + 24x^2 y^2 + 48xy^3 + 12y^4 + 16x^3 + 16y^3 - 6x^2 - 24xy - 6y^2 - 6x - 6y - 1) + \\
&0.5 \beta (2x^2 + 2y^2 - 2x - 2y - 1)
\end{aligned}$$

$$\begin{aligned}
u_2 &= 0.25 D_u (16 t + 3 \alpha t^2 (20x^4 + 24x^2 y^2 + 4y^4 - 40x^3 - 24x^2 y - 24xy^2 - 8y^3 + \\
&12x^2 + 24xy + 4y^2 + 8x - 1) + 3 \alpha t^2 (4x^4 + 24x^2 y^2 + 20y^4 - 8x^3 - 24x^2 y - 24xy^2 - \\
&4y^3 + 4x^2 + 24xy + 12y^2 + 8y - 1))
\end{aligned}$$

$$v_2 = 8 \alpha^2 r t^3 \frac{x^{10}}{k} - 15 r D_u \alpha^2 t^4 \frac{x^8}{k} - 15 \alpha^2 r D_v t^4 \frac{x^8}{k} - 4 \alpha^2 r t^3 \frac{x^{11}}{k} - 4 \alpha^2 \beta r t^3 \frac{x^{11}}{k} - 2 b r t^2 x^8 \frac{\alpha}{k} + 0.125 \alpha^2 r \frac{t^3 x}{k} - 3 \alpha^2 \beta r \frac{t^3 x^5}{k} + 2 r D_u D_v t^4 \frac{\alpha}{k}$$

Then

$$u = \sum_{i=0}^n u_i$$

$$u = 0.5 - x^2 + x - y^2 + y - 4 D_u t + 0.125 t \alpha (8 x^6 + 24 x^4 y^2 + 24 x^2 y^4 + 8 y^6 - 24 x^5 - 24 x^4 y - 48 x^3 y^2 - 48 x^2 y^3 - 24 x y^4 - 24 y^5 + 12 x^4 + 48 x^3 y + 24 x^2 y^2 + 48 x y^3 + 12 y^4 + 16 x^3 + 16 y^3 - 6 x^2 - 24 x y - 6 y^2 - 6 x - 6 y - 1.) + 0.5 (2 x^2 + 2 y^2 - 2 x - 2 y - 1) + 0.25 D_u (16 t + 3 \alpha t^2 (20 x^4 + 24 x^2 y^2 + 4 y^4 - 40 x^3 - 24 x^2 y - 24 x y^2 - 8 y^3 + 12 x^2 + 24 x y + 4 y^2 + 8 x - 1.) + 3 \alpha t^2 (4 x^4 + 24 x^2 y^2 + 20 y^4 - 8 x^3 - 24 x^2 y - 24 x y^2 - 40 y^3 + 4 x^2 + 24 x y + 12 y^2 + 8 y - 1))....$$

and

$$v = \frac{8 \alpha^2 r t^3 x^{10}}{k} - \frac{15 r D_u \alpha^2 t^4 x^8}{k} - \frac{15 \alpha^2 r D_v t^4 x^8}{k} - \frac{4 \alpha^2 r t^3 x^{11}}{k} - \frac{4 \alpha^2 \beta r t^3 x^{11}}{k} ...$$

6. Numerical Results and Analysis

This section presents the results of the analytical solution in two distinct biological scenarios, with the aim of evaluating the dynamic behavior of the model .We begin with the first scenario by presenting time series of drug and tumor concentrations at selected spatial points within the domain, allowing their evolution over time to be tracked .These results are followed by a thorough biological interpretation that explains the observed patterns and links them to relevant physiological implication

7. Temporal Evolution of Concentrations at Different Spatial Locations

We present the numerical values for the drug concentration $u(x, y, t)$ and tumor concentration $v(x, y, t)$ at four spatial points representing the domain, as reference samples for the time evolution trajectories. In two different scenarios

- **Peripheral region:** $x=y=0.1$
- **Center of the domain:** $x=y=0.5$
- **Near the edge:** $x=y=0.9$
- **Domain boundary:** $x=y=1.0$

7.1. Scenario 1: Baseline Biological Parameters

In this scenario, the following set of parameters is used to tune the model (1):

$$\alpha = 0.8, r = 0.9, k = 0.5, \beta = 0.9, D_v = 0.001, D_u = 0.01$$

Table 1: The approximation solutions for $u(x, y, t)$ and $v(x, y, t)$ via L-AADM when $x=y=0.1$ and $x=y=0.5$.

t	$u(x, y, t)$	$v(x, y, t)$	$u(x, y, t)$	$v(x, y, t)$
0	0.6120000000	0.6800000000	0.9000000000	1.0000000000
0.1	0.5904595137	0.5902213144	0.8457287299	0.8099522406

0.2	0.5758064163	0.5294373362	0.8223059806	0.7185260635
0.3	0.5664401479	0.4917870777	0.8170688625	0.6886476436
0.4	0.5609257634	0.4715751716	0.8200885118	0.6859771776
0.5	0.5579939325	0.4632718571	0.8241700878	0.6789088866
0.6	0.5565409382	0.4615129979	0.8248527786	0.6385710234
0.7	0.5556286802	0.4611000619	0.8204097903	0.5388258552
0.8	0.5544846697	0.4570001410	0.8118483599	0.3562696847
0.9	0.5525020345	0.4443459299	0.8029097532	0.3562696847
1	0.5492395158	0.4184357534	0.8029097532	0.6146820282

Table 2: The approximation solutions for $u(x, t)$ and $v(x, t)$ via L-AADM when $x=y=0.9$ and $x=y=1$.

t	$u(x, y, t)$	$v(x, y, t)$	$u(x, y, t)$	$v(x, y, t)$
0	0.6120000000	0.6800000000	0.4500000000	0.5000000000
0.1	0.5904595141	0.5902213144	0.4389305463	0.4514703969
0.2	0.5758064151	0.5294373355	0.4297024514	0.4137252519
0.3	0.5664401493	0.4917870743	0.4220015741	0.3853455283
0.4	0.5609257558	0.4715751665	0.4155344150	0.3649328140
0.5	0.5579939069	0.4632718663	0.4100280019	0.3511092930
0.6	0.5565409472	0.4615129609	0.4052303294	0.3425178754
0.7	0.5556287131	0.4611000533	0.4009095675	0.3378219420
0.8	0.5544848925	0.4570000844	0.3968553142	0.3357055738
0.9	0.5525020718	0.4443459781	0.3928748731	0.3348736165
1	0.5492398599	0.4421568701	0.3888017057	0.3340513426

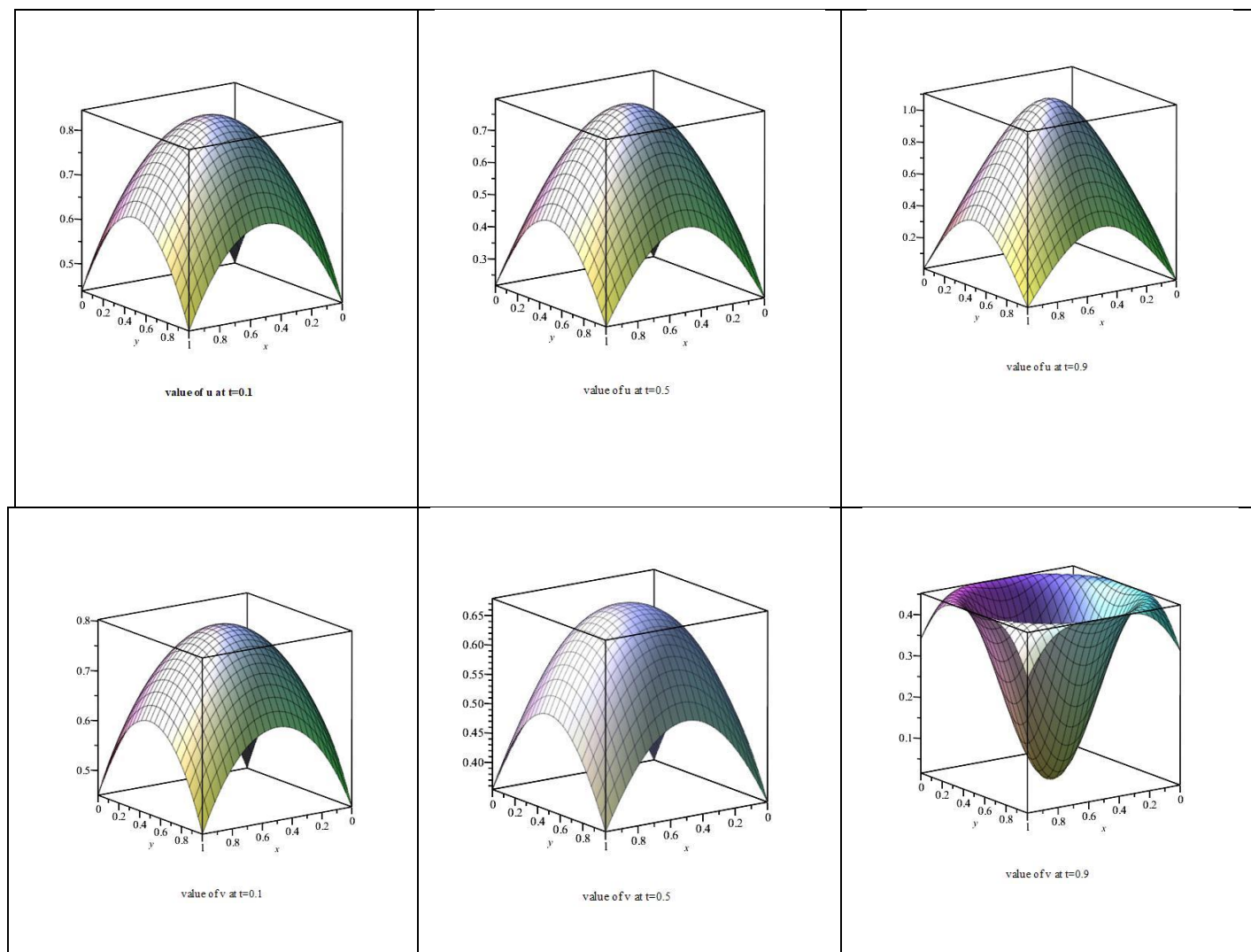
The model was calibrated under the parameters

$\alpha = 0.8, r = 0.9, k = 0.5, \beta = 0.9, D_v = 0.001, D_u = 0.01$, with biologically inspired initial conditions that reflect the spatial localization of tumor and drug concentrations. The spatiotemporal evolution was documented at four symmetrical points in the domain where $x = y \in \{0.1, 0.5, 0.9, 1.0\}$ and over the period $t \in [0, 1]$. At the center point $(x, y) = (0.5, 0.5)$ Both the drug and the tumor started at their maximum values $u(0) = 0.9, v(0) = 1.0$. The tumor showed a continuous decline, reflecting the effectiveness of the cytotoxic effect, while the dynamics of the drug were characterized by irregularity: it decreased to about 0.8029 at $t = 0.9$ and then rebounded slightly to return to a value close to $t = 1$. This behavior likely reflects a diffusive rebound following temporary depletion due to rapid consumption, followed by redistribution as the tumor mass declined and its resistance to diffusion decreased. On

the contrary, extreme points such as (1.0,1.0) showed a uniform decrease for both variables. When $x = y = 0.1$ the drug concentration decreased from 0.549, and the tumor regressed from 0.680 to 0.418. This pattern in the results shows that drug distribution is greater in less densely populated areas, resulting in a greater response to treatment, but in denser areas, a complex interaction process occurs between the tumor and the drug.

7.2. Visual Analysis of Drug and Tumor Distributions in Scenario 1

The three-dimensional diagrams show us the concentration of both the drug u and the tumor v at specific times $t=0.1, 0.5, 0.9$ in the interval $(x,y) \in [0,1] \times [0,1]$ where we observe that at the first time, both surfaces are convex, which shows us the symmetrical initial conditions. As we move to $t = 0.5$, the peaks decrease and the curves flatten, indicating effective diffusion and the beginning of tumor suppression due to the toxic effect of the drug. By $t = 0.9$, a striking geometric transformation in the distribution of the tumor from a “dome” to a “bowl” (from convex to concave upward) is evident, as clearly shown in the third diagram of tumor concentration. This reversal in curvature biologically indicates a significant local depletion of tumor cells near the center of drug injection—where cellular toxicity is at its peak—accompanied by a relative increase at the edges and the formation of a central “depression.” In contrast, the drug maintains a relatively smooth convex distribution across the field, indicating continuous and homogeneous diffusion, as opposed to a more spatially selective tumor regression.



Figures 1: illustrates the path of approximate solutions $u(x, y, t)$ and $v(x, y, t)$ for different time values.

7.3. Scenario 2

In this scenario, the following set of parameters is used to tune the model:

$$\alpha = 1, r = 0.9, k = 0.5, \beta = 0.9, D_v = 0.001, D_u = 0.1$$

Table 3: The approximation solutions for $u(x, y, t)$ and $v(x, y, t)$ via L-AADM when $x=y=0.1$ and $x=y=0.5$.

t	$u(x, y, t)$	$v(x, y, t)$	$u(x, y, t)$	$v(x, y, t)$
0	0.6120000000	0.6800000000	0.9000000000	1.0000000000
0.1	0.5547230804	0.5872088828	0.8092353930	0.8030219434
0.2	0.5078425284	0.5272315849	0.7703406211	0.7174170245
0.3	0.4683527468	0.4922952823	0.7633675790	0.6949324406
0.4	0.4338157819	0.4751947904	0.7749725943	0.6939198189
0.5	0.4023613237	0.4692925730	0.7984164162	0.6793352170
0.6	0.3726867045	0.4685187324	0.8335642357	0.6227391193
0.7	0.3440569018	0.4673710164	0.8868856524	0.5022964431
0.8	0.3163045353	0.4609148204	0.9714547221	0.3027765308
0.9	0.2898298681	0.4447831732	1.106949913	0.3027765308
1	0.2656008076	0.4151767559	1.319654132	0.6146820282

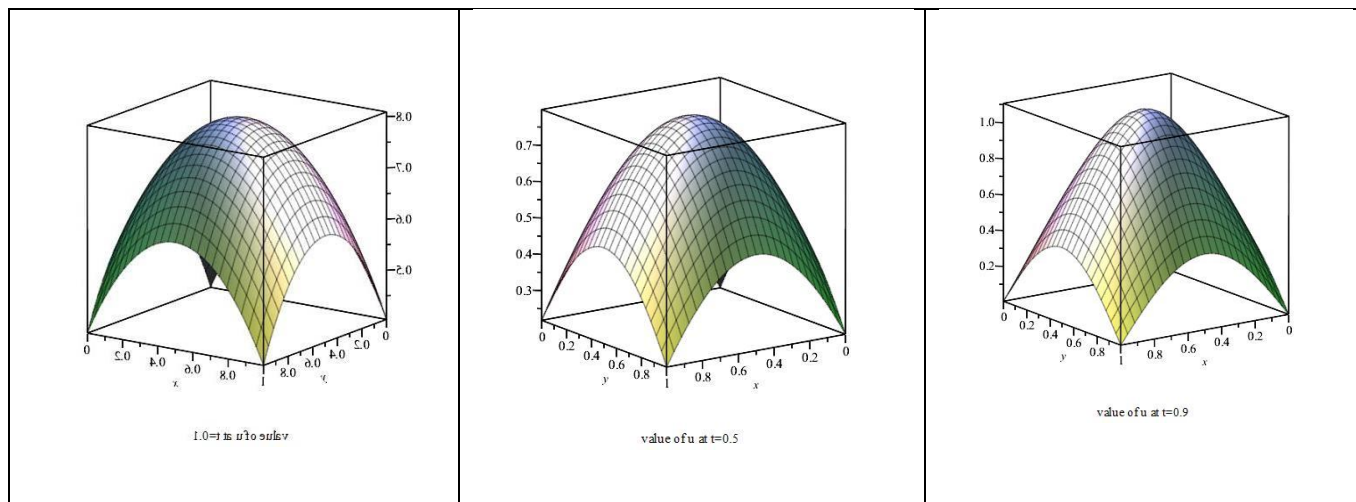
Table 4: The approximation solutions for $u(x, t)$ and $v(x, t)$ via L-AADM when $x=y=0.9$ and $x=y=1$.

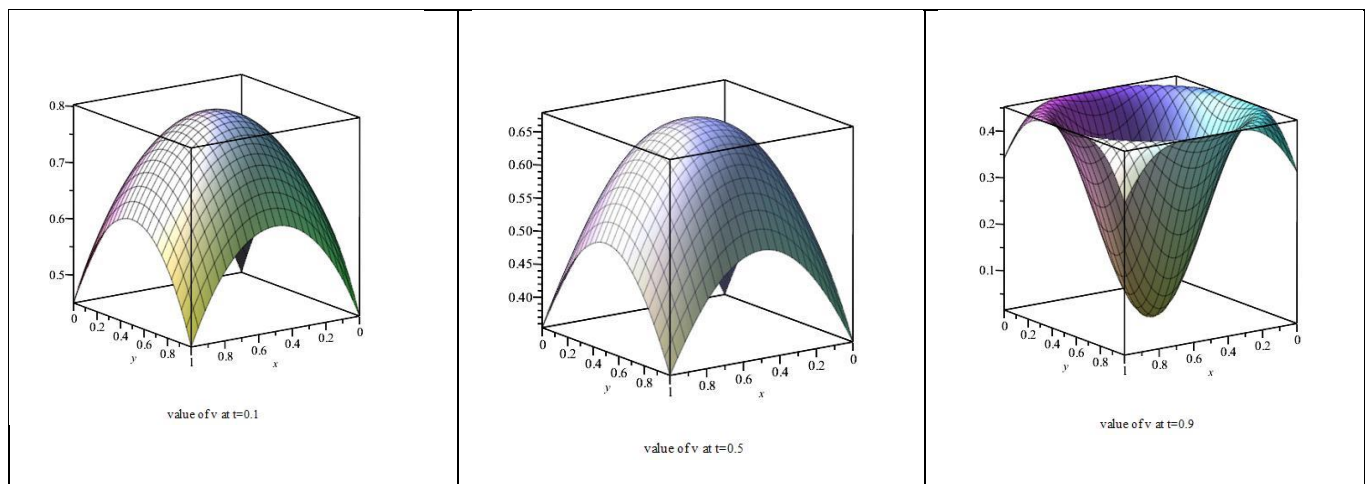
t	$u(x, y, t)$	$v(x, y, t)$	$u(x, y, t)$	$v(x, y, t)$
0	0.6120000000	0.6800000000	0.4500000000	0.5000000000
0.1	0.5547230804	0.5872088826	0.4038242886	0.4501566442
0.2	0.5078425292	0.5272315841	0.3582435080	0.4125683528
0.3	0.4683527493	0.4922952799	0.3125064166	0.3852769906
0.4	0.4338157787	0.4751947821	0.2659673396	0.3664300304
0.5	0.4023613317	0.4692925596	0.2180859498	0.3542804726
0.6	0.3726866963	0.4685186586	0.1684286289	0.3471871433
0.7	0.3440570361	0.4673710468	0.1166657170	0.3436141589
0.8	0.3163051328	0.4609147433	0.0625766689	0.3421314126
0.9	0.2898301073	0.4447831191	0.0060376505	0.3414153076
1	0.2656010403	0.4421568701	0.3888017057	0.3340513426

Second scenario. The interaction-diffusion system was evaluated under modified parameter settings that included an increase in the drug decay rate ($a = 1$) and an enhancement of its diffusion coefficient ($D_u = 0.1$) compared to the previous case. Initial spatially localized configurations were re-adopted for both the tumor and the drug, and observations were recorded over the period $t \in [0,1]$ at symmetrical points where $x = y \in \{0.1, 0.5, 0.9, 1.0\}$. At the center point $(x, y) = (0.5, 0.5)$ —where the maximum initial values $u(0) = 0.9, v(0) = 1.0$ —a non-simple time pattern emerges: the tumor concentration declines sharply and uniformly throughout the period until $s(1) \approx 0.615$, confirming its toxic efficacy. In contrast, the drug concentration follows a non-uniform trajectory, decreasing from 0.9 to a local minimum at $t \approx 0.4 - 0.5$, then rises abruptly to $u(1) = 1.319$. This reversal in values illustrates the nature of the drug-tumor interaction and redistribution to surrounding areas. This is due to a diffusion coefficient of 0.1 at the points $(0.9, 0.9)(0.1, 0.1)$. Initially, the density values are moderate, $u(0) = 0.612$ and $v(0) = 0.680$, then they decrease to $u(1) \approx 0.265$ and $v(1) \approx 0.415$. This indicates drug degradation and tumor inhibition. At the periphery, in the region $x=y=1.0$, we observe the same behavior in the results, indicating a weakening of the tumor-drug interaction in the periphery.

7.4. Visual Analysis of Drug and Tumor Distributions in Scenario 2

These three-dimensional graphs show the distribution of drug and tumor concentrations at successive time intervals of $t=0.1, 0.5, 0.9$ in the interval $(x,y) \in [0,1] \times [0,1]$ under modified biological conditions. At the initial time, the two shapes form a convex surface with a concentration at the point $(0.5, 0.5)$. At the next time, the surface remains convex, with an observed expansion of the spread, indicating tumor shrinkage under the influence of the drug. At the final time, the curve inverts from convex to concave. This is because drug exposure leads to tumor shrinkage, while the drug maintaining its convex shape indicates continued spread. Overall, the shift from convex to concave demonstrates the drug's effectiveness at the injection site.





Figures 2: illustrates the path of approximate solutions $u(x, y, t)$ and $v(x, y, t)$ for different time values.

8. Comparative Analysis of the Two Parameter Scenarios

For treatment evaluation, we compared two different biologic cases in terms of reaction coefficient and diffusion coefficient. In the first case, the values $\alpha=0.8$ and $D_u=0.01$ indicated slow but concentrated drug diffusion, leading to a gradual reduction in tumor size, with the tumor transitioning from a convex to a concave state at the end time. In the second case, the coefficients were $\alpha=1.0$ and $D_u=0.1$. In this case, diffusion was faster and more effective, resulting in a significant reduction in tumor size and a greater concavity than in the first case. Therefore, in general, the results show that increasing the reaction and diffusion coefficients enhances drug efficacy.

9. Conclusion and Future Perspectives

Our results demonstrate a strong relationship between drug diffusion and cytotoxicity. The concavity of the final drug concentration curve clearly indicates that areas with high tumor density consume the drug at high intensity. This supports the biological fact that drug consumption is highest at the tumor center and decreases towards the periphery. From a mathematical perspective, the shift from convexity to concavity indicates a non-linear relationship between diffusion and interaction. Furthermore, the hybrid method we used proved its ability to detect these geometric shifts, which are difficult for numerical methods to easily identify. Clinically, these results highlight the importance of spatial targeting and dose control. Areas with high tumor density may require continuous drug administration, while a single dose may suffice for areas with lower tumor density. Ultimately, these findings can be used to design a more precise and intelligent system for characterizing biological processes.

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