Applying Adomian Decomposition Method to Solve Burgess Equation with a Non-linear Source

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Abstract
In the present work we consider the mathematical model that describes brain tumour growth (glioblastomas) under medical treatment. Based on the medical study presented by R. Stupp et al. (New Engl Journal of Med 352: 987-996, 2005) which evidence that, combined therapies such as, radiotherapy and chemotherapy, produces negative tumour-growth, and using the mathematical model of P. K. Burgess et al. (J Neuropath and Exp Neur 56: 704-713, 1997) as an starting point, we present a model for tumour growth under medical treatment represented by a non-linear partial differential equation that is solved using the Adomian Decomposition Method (ADM). It is also shown that the non-linear term efficiently models the effects of the combined therapies. By means of a proper use of parameters, this model could be used for calculating doses in radiotherapy and chemotherapy.

Keywords: Burgess equation, Adomian polynomials, Glioblastoma, Temozolomide.

Introduction
The glioblastoma, also known as glioblastoma multiforme (GBM), is a highly invasive glioma in the brain [23]. It is the most common and most aggressive brain
tumour in humans. From a medical point of view, GBM is a fast growing tumour made up of an heterogeneous mixture of poorly differentiated astrocytes, with pleomorphism, necrosis, vascular proliferation and high rate mitosis. This glioma can appear at any age but is more frequent among adults older than 45 years. Usually, they appear in the cerebral hemispheres but they could also appear in the cerebellum. From a mathematical point of view, they can be considered to have a spherical geometry as it is illustrated in figure [1] see [14]. In 1997 P. K. Burgess et. al. proposed a 3-dimensional mathematical model that describes the growth of a glioblastoma free of any medical treatment that could grow with no restrictions. This model provides information about the density change of the tumour in any spatiotemporal point but does not give any information about the case in which some annihilation of tumour cells could appear due, possibly, to the administration of cancericidal substances and hence does not study the dynamics of proliferation-annihilation of gliomas. It is worthy to say that some bi-dimensional mathematical models preceded the Burgess model as the ones formulated in [25] y [28].

In the present work, and taking the Burgess model as an starting point, we will formulate a mathematical model that takes into account the action of some cancericidal substances (as temozolomide and chemotherapy) and hence the possibility to annihilate or diminish the growth of the gliomas. Our resulting model, in agreement with clinical data [24], is expressed in terms of a partial non-linear differential equation that is solved using the Adomian Decomposition Method [5], [4]. The model proposed also allows to compare the profile of a tumour growing without any treatment with the profile of a tumour subject to treatment, i.e., our model includes a term that gives the difference between the growth and annihilation of the glioma. Calibration of doses using this model as a basis could result in the lengthening of life for glioma patients [24].

**Analysis of the Method**

Adomian Decomposition Method (ADM) is a technique to solve ordinary and partial nonlinear differential equations. Using this method, it is possible to express analytic solutions in terms of a rapidly converging series [5]. In a nutshell, the method identifies and separates the linear and nonlinear parts of a differential equation. By inverting and applying the highest order differential operator that is contained in the linear part of the equation, it is possible to express the solution in terms of the the rest of the equation affected by this inverse operator. At this point, we propose to express this solution by means of a decomposition series.
with terms that will be well determined by recursion and that gives rise to the solution components. The nonlinear part is expressed in terms of the Adomian polynomials. The initial or the boundary condition and the terms that contain the independent variables will be considered as the initial approximation. In this way and by means of a recurrence relations, it is possible to calculate the terms of the series by recursion that gives the approximate solution of the differential equation.

Given a partial (or ordinary) differential equation

$$F u(x, t) = g(x, t)$$  \hspace{1cm} (1)

with the initial condition

$$u(x, 0) = f(x),$$  \hspace{1cm} (2)

where $F$ is differential operator that could itself, in general, be nonlinear and therefore includes linear and nonlinear terms.

In general, equation (1) is be written as

$$L_t u(x, t) + R u(x, t) + N u(x, t) = g(x, t)$$  \hspace{1cm} (3)

where $L_t = \frac{\partial}{\partial t}$, $R$ is the linear remainder operator that could include partial derivatives with respect to $x$, $N$ is a nonlinear operator which is presumed to be analytic.
and $g$ is a non-homogeneous term that is independent of the solution $u$.

Solving for $L_t u(x,t)$, we have

$$L_t u(x,t) = g(x,t) - Ru(x,t) - Nu(x,t). \quad (4)$$

As $L$ is presumed to be invertible, we can apply $L_t^{-1}(\cdot) = \int_0^t (\cdot) dt$ to both sides of equation (4) obtaining

$$L_t^{-1} L_t u(x,t) = L_t^{-1} g(x,t) - L_t^{-1} Ru(x,t) - L_t^{-1} Nu(x,t). \quad (5)$$

An equivalent expression to (5) is

$$u(x,t) = f(x) + L_t^{-1} g(x,t) - L_t^{-1} Ru(x,t) - L_t^{-1} Nu(x,t), \quad (6)$$

where $f(x)$ is the constant of integration with respect to $t$ that satisfies $L_t f = 0$. In equations where the initial value $t = t_0$, we can conveniently define $L_t^{-1}$.

The ADM proposes a decomposition series solution $u(x,t)$ given as

$$u(x,t) = \sum_{n=0}^{\infty} u_n(x,t). \quad (7)$$

The nonlinear term $Nu(x,t)$ is given as

$$Nu(x,t) = \sum_{n=0}^{\infty} A_n(u_0, u_1, \ldots, u_n) \quad (8)$$

where $\{A_n\}_{n=0}^{\infty}$ is the Adomian polynomials sequence given by (see deduction in appendix at the end of this paper)

$$A_n = \frac{1}{n!} \frac{d^n}{d\lambda^n} [N(\sum_{k=0}^{n} \lambda^k u_k)]|_{\lambda=0}. \quad (9)$$

Substituting (7), (8) y (9) into equation (6), we obtain

$$\sum_{n=0}^{\infty} u_n(x,t) = f(x) + L_t^{-1} g(x,t) - L_t^{-1} R \sum_{n=0}^{\infty} u_n(x,t) - L_t^{-1} \sum_{n=0}^{\infty} A_n(u_0, u_1, \ldots, u_n),$$

with $u_0$ identified as $f(x) + L_t^{-1} g(x,t)$, and therefore, we can write

$$u_0(x,t) = f(x) + L_t^{-1} g(x,t),$$

$$u_1(x,t) = -L_t^{-1} Ru_0(x,t) - L_t^{-1} A_0(u_0),$$

$$\vdots$$

$$u_{n+1}(x,t) = -L_t^{-1} Ru_n(x,t) - L_t^{-1} A_n(u_0, \ldots, u_n).$$

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From which we can establish the following recurrence relation, that is obtained in an explicit way for instance in reference [27],

\[
\begin{align*}
  u_0(x,t) &= f(x) + L_t^{-1}g(x,t), \\
  u_{n+1}(x,t) &= -L_t^{-1}Ru_n(x,t) - L_t^{-1}A_n(u_0, u_1, \ldots, u_n), \quad n = 0, 1, 2, \ldots.
\end{align*}
\]  

(11)

Using (11), we can obtain an approximate solution of (1), (2) as

\[
u(x,t) \approx \sum_{n=0}^{k} u_n(x,t), \quad \text{where } \lim_{k \to \infty} \sum_{n=0}^{k} u_n(x,t) = u(x,t).
\] 

(12)

This method has been successfully applied to a large class of both linear and non-linear problems [13]. The Adomian decomposition method requires far less work in comparison with traditional methods. This method considerably decreases the volume of calculations. The decomposition procedure of Adomian easily obtains the solution without linearising the problem by implementing the decomposition method rather than the standard methods. In this approach, the solution is found in the form of a convergent series with easily computed components; in many cases, the convergence of this series is extremely fast and consequently only a few terms are needed in order to have an idea of how the solutions behave. Convergence conditions of this series have been investigated by several authors, e.g., [1, 2, 8, 9].

The Mathematical Model of the Burgess Equation

Mathematical modelling of the spread of aggressive brain cancers such as glioblastoma multiforme has been discussed by several authors [7], [26], [16]. It is noteworthy to say that some authors like [26] have included a killing term. In any case, they describe tumour-growth by using spatiotemporal models that can be read as

\[
\text{Rate of change of tumour cell density} = (\text{Diffusion of tumour cells}) + (\text{Growth of tumour cells}) - (\text{Killing rate of the same cells})
\]

in mathematical terms,

\[
\frac{\partial \eta(r,t)}{\partial t} = D \frac{1}{r^2} \frac{\partial}{\partial r} \left(r^2 \frac{\partial \eta(r,t)}{\partial r}\right) + p(t)\eta(r,t) - k(t)\eta(r,t).
\] 

(13)
In this equation, $\eta(r,t)$ is the concentration of tumour cells at location $r$ at time $t$, $D$ is the diffusion coefficient, i.e., a measure of the areal speed of the invading glioblastoma cells, $p$ is the rate of reproduction of the glioblastoma cells, and $k$ the killing rate of the same cells. The last term has been used by some authors to investigate the effects of chemotherapy [25], [28]. In this model, the tumour is assumed to have spherical symmetry and the medium through which it is expanding, to be isotropic and uniform. We can assume that at the beginning of time (diagnostic time $t_0$), the density of cancer cells is $N_0$, i.e., $\eta(r_0,t_0) = N_0$ and so the equation (13) is

$$\left\{ \begin{array}{l}
\frac{\partial \eta(r,t)}{\partial t} = D(r,t)\left(\frac{\partial^2 \eta(r,t)}{\partial r^2} + \frac{2}{r} \frac{\partial \eta(r,t)}{\partial r}\right) + \left[p(t) - k(t)\right]\eta(r,t), \\
\eta(r_0,t_0) = N_0.
\end{array} \right.$$  

The solution of (14) is given, without many details, in [7] and in [15]. They solve this equation for the -non-very realistic- case in which $k(t) \equiv 0$ and $p(t)$ and $D(r,t)$ are constants. The solution for this case is given by

$$\eta(r,t) = \frac{N_0 e^{\left[p t - \frac{r^2}{4Dt}\right]}}{8\pi D t^{\frac{3}{2}}}.$$  

(15)

Using this equation (15), the mentioned authors calculate the expected survival time (in months) for a person that has a brain tumour modelled using equation (14).

Following [6], we propose the change of variables $\tau = 2Dt$, $u(r,\tau) = r\eta(r,t)$ and $\omega(r,\tau) = \frac{p - k}{2D}$. Using this change of variables and keeping $D$ constant, equation (14) is given by

$$\frac{\partial u(r,\tau)}{\partial \tau} = \frac{1}{2} \frac{\partial^2 u(r,\tau)}{\partial r^2} + \frac{\omega(r,\tau)}{2D}.$$  

(16)

In [24], a medical study is presented that stresses the advantages of using combined therapies such as chemotherapy and radiotherapy in the treatment of brain cancer. Concretely, they present the results of using temozolomide in combination with radiotherapy. The results show a lengthening in the patient life as a consequence of the tumour size decrease. Mathematically this is traduced as a negative growth of the tumour, in other words, the term $p(r,t) - k(r,t)$ (growth of the cancer cells minus eliminations of cancer cells) is negative. In present work, we will study the case presented by Roger Stupp et. al. in [24]. Our model will make use of equation (16) taking $D$ constant and $(r,\tau) \in (0,1] \times [0,1]$ that gives a renormalised time and space intervals. In order to take into account the combined
effects of radiotherapy and chemotherapy, we will introduce a term that models
the decay (negative growth) of the glioma, $\omega(r, \tau) = e^{-u} + \frac{1}{2}e^{-2u}$, with $u = u(r, \tau)$. 
The decrease of the tumour depending on the position $r$ and time $\tau$ as it shown in
figure [2]

![Figure 2: $\omega(r, \tau) = e^{-u} + \frac{1}{2}e^{-2u}$ with $(r, \tau) \in (0,1] \times [0,1]$](image)

The ADM has been used by several authors to solve linear and non-linear diffusion equations as well as fractional diffusion equations, some important references can be found in [10, 11, 12, 17, 18, 19, 20, 21, 22]. In the present work we are interested in the solution of the diffusion equation (16) in which a non-linear source $\omega(r, \tau)$ is modelling the effects of the combined use of radiotherapy and chemotherapy treatment with Temozolomide as is reported in [24].

**Solution of a nonlinear model**

Considering the equation (16), with $\omega(r, \tau) = e^{-u} + \frac{1}{2}e^{-2u}$ and $D = \frac{1}{2}$ our model will be given by the following non-linear partial differential equation
\[
\begin{aligned}
\frac{\partial u}{\partial \tau} &= \frac{1}{2} \frac{\partial^2 u}{\partial r^2} + e^{-u} + \frac{1}{2} e^{-2u}, \\
u(r,0) &= \ln(r+2).
\end{aligned}
\]

(17)

In equation (17) we have made the a priori assumption that the initial condition is \( u(r,0) = \ln(r+2) \). This assumption considers that the initial tumour growth profile is given by \( u(r,0) \) in the time we start the annihilation or attenuation of the gliomas by means of some treatment (as chemotherapy). The initial growth profile is illustrated in figure 3.

Figura 3: Initial growth-profile \( u(r,0) = \ln(r+2) \)

Using

\[
A_n(u_0, u_1, \ldots, u_n) = \frac{1}{n!} \frac{d^n}{d\alpha^n} [N(\sum_{k=0}^{n} \alpha^k u_k)]|_{\alpha=0} \quad n \geq 0
\]

to calculate the Adomian polynomials, we have:

\[
A_0(u_0) = N(u_0) = e^{-u_0} + \frac{1}{2} e^{-2u_0}
\]

\[
A_1(u_0, u_1) = N'(u_0)u_1 = -u_1 e^{-u_0} - u_1 e^{-2u_0}
\]

\[
A_2(u_0, u_1, u_2) = \frac{N''(u_0) u_2^2}{2} + N'(u_0)u_2
\]
\[
= \frac{u_2^2}{2} (e^{-u_0} + 2e^{-2u_0}) + u_2(-e^{-u_0} - e^{-2u_0})
\]
\[ A_3(u_0, u_1, u_2, u_3) = N'''(u_0) \frac{u_1^3}{3!} + N''(u_0)u_1u_2 + N'(u_0)u_3 \]

\[ = \frac{u_1^3}{6}(-e^{-u_0} - 4e^{-2u_0}) + u_1u_2(e^{-u_0} + 2e^{-2u_0}) - u_3(e^{-u_0} + e^{-2u_0}) \]

\[ \vdots \]

Using the sequence for \(\{A_n\}^\infty_{n=0}\) and the recurrence relation given in (11) we can calculate \(\{u_n\}\), in this way:

\[ u_0 = \ln(r + 2) \]

\[ u_1 = \int_0^t \left[-\frac{1}{2(r+2)^2} + e^{-u_0} + \frac{1}{2}e^{-2u_0}\right]dt = \frac{t}{r+2} \]

\[ \vdots \]

The partial sums of the Adomian series are

\[ S_0 = u_0 = \ln(r + 2) \]

\[ S_1 = u_0 + u_1 = \ln(r + 2) + \frac{\tau}{r+2} \]

\[ S_2 = u_0 + u_1 + u_2 = \ln(r + 2) + \frac{\tau}{r+2} - \frac{\tau^2}{2(r+2)^2} \]

\[ S_3 = u_0 + u_1 + u_2 + u_3 = \ln(r + 2) + \frac{\tau}{r+2} - \frac{\tau^2}{2(r+2)^2} + \frac{\tau^3}{3(r+2)^3} \]

\[ \vdots \]

\[ S_m = u_0 + u_1 + \ldots + u_m = \ln(r + 2) + \frac{\tau}{r+2} - \frac{\tau^2}{2(r+2)^2} + \ldots + \frac{(-1)^{m+1} \tau^m}{m(r+2)^m} \]

and taking into account the equation (12), we have

\[ u(r, \tau) = \ln(r + 2) + \frac{\tau}{r+2} - \frac{\tau^2}{2(r+2)^2} + \ldots + \frac{(-1)^{m+1} \tau^m}{m(r+2)^m} + \ldots \quad (18) \]

taking the sum of the first terms, we can see that the above series converges to \(\ln(\frac{r+\tau}{r+2})\). Then, using (18) we have

\[ u(r, \tau) = \ln(r + 2) + \ln(\frac{\tau + r + 2}{r+2}) = \ln(r + \tau + 2). \quad (19) \]
It is easy to verify that \( u(r, \tau) \) given by (19) is a solution of the initial value problem (17). With this solution we can given the density of cancer cells in every point of \((r, \tau) \in (0,1] \times [0,1]\). In figure 4 we can observe the approximate-linear tumour growth-profile after the patient is under chemotherapy treatment with Temozolomide in contrast with the fast exponential growth given by (15) and corresponding to free-growth tumour. The free-growth tumour profile was shown in [7], in which the value of \( \eta(r,t) \) is given for different values of the parameters \( D, p \) and \( k(t) = 0 \).

In order to see the effect of medical treatment, we can compare the radius of the tumour under medical treatment versus the radius of the untreated tumour. Using the solution (15) of the Burgess linear partial equation and solving for \( r \) (also see [15]) that accounts for free growth of an untreated tumour, we obtain

\[
r_{lin} = 2tD\sqrt{p} \sqrt{1 - \frac{1}{p} \ln \left[ \frac{C_0}{N_0} (4\pi D t)^{\frac{3}{2}} \right].}
\]

(20)

Solving for \( r \) in the solution of the non-linear equation (19) that accounts for case in which we have proliferation and annihilation of tumour cells due to medical treatment, we have

\[
r_{nonlin} = e^{N_0} - \tau - 2
\]

(21)

If we take \( D = 0.5, p = 1.5 \) and \( \frac{C_0}{N_0} = 4000 \) (see [15]) and recalling that \( \tau = 2Dt \),
then \( t = \tau \). Using this values in equation (20) and (21) we obtain the following table

| \( t \) (years) | \( r_{lin} \) (cm) | \( r_{nonlin} \) (cm) |
|----------------|-----------------|-----------------|
| 0.1            | 1.22            | 1.75            |
| 0.2            | 1.82            | 1.65            |
| 0.3            | 2.29            | 1.55            |
| 0.4            | 2.69            | 1.45            |
| 0.5            | 3.04            | 1.35            |
| 0.6            | 3.35            | 1.25            |
| 0.7            | 3.64            | 1.15            |
| 0.8            | 3.90            | 1.05            |
| 0.9            | 4.14            | 0.95            |
| 1.0            | 4.37            | 0.85            |

Table 1: Radius growth in untreated tumour versus radius growth in treated tumour.

Using the data of table 1 we obtain figures 5 and 6. From the figure 5 we observe that the radius of the untreated tumour grows as predicted in (15) meanwhile we observe, in figure 6, that the treated tumour’s radius decreases with time.

Figure 5: Tumour’s radius \( r_{lin} \) versus time \( t \in (0, 1] \) for equation (20)
Summary

In this work we have proposed a model for cerebral tumour (glioblastoma) growth under medical treatment. Taking the Burgess equation as departing point, we considered additional non-linear terms that represent the dynamics of proliferation and annihilation of gliomas resulting from medical care as suggested in the clinical study done by R. Stupp [24]. The effect of the medical treatment on the tumour is represented by a non-linear term. The final model that describes the proliferation and annihilation of tumour cells is represented by a partial non-linear differential equation that is solved using the Adomian Decomposition Method (ADM). Finally, as is observed in table 1 and figures 5 and 6 our model and the solution given using the ADM appropriately models the effects of the combined therapies. By means of a proper use of parameters, this model could be used for calculating doses in radiotherapy and chemotherapy.

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Appendix: Adomian polynomials

In this appendix we will deduce equation (9) that accounts for every term in the succession of the Adomian Polynomials assuming the following hypotheses stated in [9]:

(i) the series solution, \( u = \sum_{n=0}^{\infty} u_n \), of the problem given in equation (1) is absolutely convergent,

(ii) The non-linear function \( N(u) \) can be expressed by means of a power series whose radio of convergence is infinite, that is

\[
N(u) = \sum_{n=0}^{\infty} N^{(n)}(0) \frac{u^n}{n!}, \quad |u| < \infty.
\]  

(22)

Assuming the above hypotheses, the series whose terms are the Adomian Polynomials \( \{A_n\}_{n=0}^{\infty} \) results to be a generalisation of the Taylor’s series

\[
N(u) = \sum_{n=0}^{\infty} A_n(u_0, u_1, \ldots, u_n) = \sum_{n=0}^{\infty} N^{(n)}(u_0) \frac{(u - u_0)^n}{n!}.
\]  

(23)

Is worthy to note that (23) is a rearranged expression of the series (22), and note that, due to hypothesis, this series is convergent. Consider now, the parametrisation proposed by G. Adomian in [3] given by

\[
u_\lambda(x,t) = \sum_{n=0}^{\infty} u_n(x,t) f^n(\lambda),
\]  

(24)

where \( \lambda \) is a parameter in \( \mathbb{R} \) and \( f \) is a complex-valued function such that \( |f| < 1 \). With this choosing of \( f \) and using the hypotheses above stated, the series (24) is absolutely convergent.

Substituting (24) in (23), we obtain

\[
N(u_\lambda) = \sum_{n=0}^{\infty} N^{(n)}(u_0) \left( \sum_{j=1}^{\infty} u_j(x,t) f^j(\lambda) \right)^n.
\]  

(25)

Due to the absolute convergence of

\[
\sum_{j=1}^{\infty} u_j(x,t) f^j(\lambda),
\]  

(26)
we can rearrange $N(u_\lambda)$ in order to obtain the series of the form $\sum_{n=0}^{\infty} A_n f^n(\lambda)$. Using (25) we can obtain the coefficients $A_k$ de $f^k(\lambda)$, and finally we deduce the Adomian’s polynomials. That is,

\[
N(u_\lambda) = N(u_0) + N^{(1)}(u_0)u_1 f(\lambda) + N^{(2)}(u_0)\left(\frac{u_1^2}{2!}\right) f^2(\lambda) + N^{(3)}(u_0)\left(\frac{u_2^2}{3!}\right) f^3(\lambda) + \ldots
\]

Using (27) making $f(\lambda) = \lambda$ and taking derivative at both sides of the equation, we can make the following identification

\[
A_0(u_0) = N(u_0)
\]

\[
A_1(u_0, u_1) = N'(u_0)u_1
\]

\[
A_2(u_0, u_1, u_2) = N'(u_0)u_2 + \frac{u_1^2}{2!} N''(u_0)
\]

\[
A_3(u_0, u_1, u_2, u_3) = N'(u_0)u_3 + N''(u_0)u_1 u_2 + \frac{u_2^3}{3!} N'''(u_0)
\]

\[
A_4(u_0, \ldots, u_4) = u_4 N'(u_0) + (\frac{1}{2!} u_1^2 + u_1 u_3) N''(u_0) + \frac{u_2^3}{3!} N'''(u_0) + \frac{u_4^4}{4!} N''(u_0)
\]

\[
\vdots
\]

Hence we have obtain equation (9):

\[
A_n(u_0, u_1, \ldots, u_n) = \frac{1}{n!} \frac{d^n}{d\lambda^n} [N(\sum_{k=0}^{n} \lambda^k u_k)]|_{\lambda=0}.
\]

(28)

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