Reaction-Diffusion Model of Tumor Growth with Chemotherapy

Spencer Smith, Sai Kundan Vena, Parth Shah

Abstract

In this paper we present a mathematical model of single tumor growth in relation to healthy tissue and excess H^+ concentration. The work of prior literature is synthesized to incorporate basic effects of chemotherapy treatment in the model. This allows for a study comparison of neoplastic tissue density between identical systems with and without the chemotherapy. A numerical solution was developed for the reaction diffusion model using a Forward-Time Central-Space scheme, with radial symmetry assumed for simpler calculations and adoption of additional boundary conditions. A Gompertz function was used as an analytical solution to model the behavior of the neoplastic tissue in the presence of excess of H^+ concentration for validation of the numerical scheme.

1 Introduction

This paper is an extension of the work done by Gatenby and Gawlinski concerning a reaction-diffusion model of cancer tissue [1]. We seek to solve very similar PDEs (healthy tissue density, neoplastic tissue density, excess hydrogen ion concentration), but introduce terms to take into account the effects of chemotherapy of tissue densities. The likeness of our model derivation to theirs is intended to maximize understanding of the mathematical and physical process underlying the process and nothing else.

Results of this project are relevant namely due to the prevalence of cancer. With a precise numerical model of disease behavior, strides can be taken to understand the effects of different factors (*e.g.* biological parameters, dosing period, pre vs. post-metastasis) and optimize the scientific approach taken toward cancer research and treatment alike.

2 Model Development

This project deals with three coupled PDEs in cylindrical coordinates: the density of healthy tissue (N_1), the density of neoplastic tumor tissue (N_2), and the excess hydrogen ion concentration. The mass balance for the density of the healthy tissue, N_1, is as follows:

$$\begin{aligned}\frac{\partial N_1}{\partial t} &= r_1 N_1(1 - \frac{N_1}{K_1} - \alpha_{12}\frac{N_2}{K_2}) \\ &\quad - d_1 L N_1 + \nabla \cdot (D_{N_1}[N_2]\nabla N_1)\end{aligned} \tag{1}$$

The rate of change of the healthy tissue density change includes a number of terms. The natural growth term uses growth rate r_1 and carrying capacity K_1, which is the maximum value of N_1. A completely healthy system is represented by $N_1 = K_1$. The competition term, based on the Lotka-Volterra competition strength parameter α_{12}, typifies the spatial competition between N_1 and N_2. The next term is the death rate of N_1 in response to lower pH resulting from excess hydrogen ion concentration, with proportional constant L. The final term accounts for cellular diffusion of N_1, with a N_2-dependent diffusion coefficient.

The mass balance for the density of the cancer tissue, N_2, is as follows:

$$\begin{aligned}\frac{\partial N_2}{\partial t} &= r_2 N_2(1 - \frac{N_2}{K_2} - \alpha_{21}\frac{N_1}{K_1}) \\ &\quad + \nabla \cdot (D_{N_2}[N_1]\nabla N_2)\end{aligned} \tag{2}$$

The rate of change of the neoplastic tissue takes on an analogous form to that of the healthy tissue, except without the inclusion of a death rate for the tumor. This is because tumor cells are known to thrive in the acidic environments that harm healthy tissue, so no pH-dependent rate decrease is needed. α_{21} is the equivalent strength parameter for N_2 and K_2 is the carrying capacity for the cancer tissue.

The final mass balance for the concentration of excess hydrogen ion concentration, L, is as follows:

$$\frac{\partial L}{\partial t} = r_3 N_2 - d_3 L + D_3 \nabla^2 L \tag{3}$$

The rate of change of the ion concentration has a production term proportional to the tumor tissue density, a reabsorption term that accounts for changing local pH, and a chemical diffusion term characterizing the movement of the ions.

Each of these PDEs incurred a number of simplifications in the literature to bring them to leaner forms that are less numerically intensive. Both population interaction terms, α_{12} and α_{21}, were assumed to be zero. This is not the case for all systems, but this model considers only areas where the two types of tissues are not able to coexist, which justifies the assumption.

The diffusion terms were also simplified. D_{N_1} was assumed to be zero as tissue actively partaking in biological functions diffuse very little. The coefficient for the neoplastic tissue is defined to be

$$D_{N_2} = D_2 \times (1 - N_1/K_1)$$

where D_2 is the constant diffusion constant of neoplastic tissue in the absence of healthy tissue. This is a logical representation of diffusive behavior because for neoplastic tissue to spread, healthy tissue must be below its carrying capacity. Thus, the diffusion of this tissue is controlled by the "free space" available upon death of the healthy tissue by the excess ion concentration.

With simplifications for the numerical model out of the way, the system was rendered dimensionless using the following transformations from the literature:

$$\eta_1 = \frac{N_1}{K_1} \quad \eta_2 = \frac{N_2}{K_2} \quad \Lambda = \frac{L}{L_0}$$

$$\tau = r_1 t \quad \xi = \sqrt{\frac{r_1}{D_3}}x$$

where temporal and spatial dimensions are now represented by dimensionless τ and ξ, respectively; and L_0 with the rest of the constants are defined as:

$$L_0 = r3K_2/d_3 \quad \rho_2 = r_2/r_1$$
$$\delta_1 = (d_1/d_3) \times (r_3/r_1) \times K_2$$
$$\Delta_2 = D_2/D_3 \quad \delta_3 = d_3/r_1$$

Moving along, the boundary conditions deduced from the paper are of some concern as they are as $\xi \to \infty$, and are not enforceable in a non-infinite, numerical environment. Because of this, a change in variable was made, converting dimensionless ξ into dimensionless r. The change made was

$$r = \frac{1}{1+\xi} \tag{4}$$

which changes our domain of consideration from $\xi \in (0, \infty)$ to r $\in (1, 0)$, which is possible to enforce numerically. We then solve for $\frac{\partial r}{\partial \xi}$ to complete the change of variable using

$$\frac{\partial}{\partial \xi} = \frac{\partial}{\partial r}\frac{\partial r}{\partial \xi} \tag{5}$$

Solving the definition of r for ξ, we can find $\frac{\partial r}{\partial \xi}$ by differentiating:

$$\begin{aligned}\frac{\partial r}{\partial \xi} &= \frac{\partial}{\partial \xi}(\frac{1}{1+\xi}) \\ &= -\frac{1}{(1+\xi)^2} \\ &= -r^2\end{aligned} \tag{6}$$

$$\frac{\partial}{\partial \xi} = -r^2 \frac{\partial}{\partial r} \tag{7}$$

which can be used for each of our system variables. Using substitution of these equations and the chain rule, we can also compute $\frac{\partial^2}{\partial \xi^2}$.

$$\begin{aligned}\frac{\partial^2}{\partial \xi^2} &= \frac{\partial}{\partial \xi}(\frac{\partial}{\partial \xi}) \\ &= -r^2 \frac{\partial}{\partial r}(-r^2 \frac{\partial}{\partial r}) \\ &= r^4 \frac{\partial^2}{\partial r^2} + 3r^2 \frac{\partial}{\partial r}\end{aligned} \tag{8}$$

Combining these transformations and assumptions, the dimensionless PDEs can now be written as

$$\frac{\partial \eta_1}{\partial \tau} = \eta_1(1-\eta_1) - \delta_1 \Lambda \eta_1 \tag{9}$$

$$\begin{aligned}\frac{\partial \eta_2}{\partial \tau} &= \rho_2 \eta_2 (1-\eta_2) \\ &+ \Delta_2[(1-\eta_1)(r^4 \frac{\partial^2 \eta_2}{\partial r^2} - \frac{\partial \eta_2}{\partial r}(1-r)) \\ &- \frac{\partial \eta_1}{\partial r}\frac{\partial \eta_2}{\partial r} r^4\end{aligned} \tag{10}$$

$$\frac{\partial \Lambda}{\partial \tau} = \delta_3(\eta_2 - \Lambda) + \nabla_\xi^2 \Lambda \tag{11}$$

Finally, chemotherapy treatment was added to the model. Introducing additional terms into the equations themselves doesn't make sense, as the dose only affects the system for a small amount of time system. For this reason, a model approach similar to Panetta was used [2].

$$\eta_1(\tau^+) = \eta_1(\tau^-) \times F(D) \tag{12}$$

$$\eta_2(\tau^+) = \eta_2(\tau^-) \times F(D) \tag{13}$$

Here, F and $\bar{F}$ are the survival fractions of the healthy and neoplastic tissues, respectively; and τ^+ and τ^- represent the time just after and just before dosing, respectively. At the time of dose, the density ODEs are multiplied by a dosing function that cuts down the tissue density without bias. Thus, healthy and cancerous tissue are equally affected by the term.

Various parametrized functions are presented by Panetta in his work. Each function is not dependent on time and space, only a number of parameters not considered elsewhere in this model. For our purposes, this function was based on the form

$$F(D) = e^{-\alpha D} \tag{14}$$

where the value for α was manually tuned for the system. A number of different values of τ were chosen to invoke the chemotherapy term. This dosing function was used with the same parameters for both $F(D)$ and $\bar{F}(D)$.

3 Results

The model was numerically carried out using a FTCS (Forward-Time Central-Space) finite difference scheme.

$$\frac{\partial f}{\partial r} = \frac{f_{i+1} - f_{i-1}}{2\Delta r} \tag{15}$$

$$\frac{\partial^2 f}{\partial r^2} = \frac{f_{i+1} + f_{i-1} - 2f_i}{\Delta r^2} \tag{16}$$

$$\frac{\partial f}{\partial \tau} = \frac{f_{j+1} - f_j}{\Delta \tau} \tag{17}$$

where i denotes steps in space and j denotes steps in time.

The boundary conditions for the model came from two assumptions made about the system's behavior. The first arose from assuming radial symmetry, valid due to the equivalence of behavior r distance away or $-r$ distance away from the origin (center of the tumor). Because of this symmetry, it is known that $\frac{\partial f}{\partial r}|_{r=0} = 0$ for each of the system variables. This can be applied to the finite difference scheme in Equations 12-14,

$$\frac{\partial f}{\partial r} = \frac{f_{i+1} - f_{i-1}}{2\Delta r} = 0$$

yielding

$$f_{i+1} = f_{i-1} \tag{18}$$

an equality usable for the second derivatives for the initial and final boundary conditions. For the calculation of the first, the equality can substitute the value of f_1 for the value of f_{-1}, giving

$$\frac{\partial^2 f}{\partial r^2}|_{i=1} = \frac{2(f_2 - f_1)}{\Delta r^2} \tag{19}$$

and similarly for the final boundary condition, substituting f_{N-1} for the value of f_{N+1},

$$\frac{\partial^2 f}{\partial r^2}|_{i=N} = \frac{2(f_{N-1} - f_N)}{\Delta r^2} \tag{20}$$

This is the case for all three of the PDEs, giving six boundary conditions.

The second set of boundary conditions is for the system's behavior at steady state. The system reaches critical conditions $\xi \to \infty$, or as the spatial coordinate departs further and further from the tumor center. These conditions are that there is no more change in any of the system variables over time. Mathematically, this can be tabulated as

$$\frac{\partial \eta_1}{\partial \tau}|_{r=0} = \frac{\partial \eta_2}{\partial \tau}|_{r=0} = \frac{\partial \Lambda}{\partial \tau}|_{r=0} = 0 \tag{21}$$

which is the final set of boundary conditions for the system.

In order to compute the analytical solution from the model, a few analytical approximations for the interfacial profile structures and their dependence on the wave front propagation velocity had to be made. Wave propagation theory states that every point on a wave front is a source of wavelets. The wavefront velocity defined as

the high frequency limit of the phase velocity is assumed to be very small and that the tumor edge is sharp. Because the neoplastic population growth looks like logistic growth and the neoplastic growth in the spatial domain is much greater than its diffusive flux, it is assumed that the tumor edge is sharp.

From the literature, it also assumed that the Equations 9,10 and 11 have a solution of the form $f(\xi,\tau) = f(\xi - c\tau)$ where f is either η_1, η_2 *or* Λ and $\zeta = \xi - c\tau$. Each of this field is treated as a traveling wave and that each field has a wave front profile, f propagating in the $+\xi$ direction at c. When these forms are substituted into Equations 9, 10 and 11, the partial derivatives are converted to ordinary derivatives as follows:

$$-c\eta_1' = \eta_1(1-\eta_1) - \delta_1\Lambda\eta_1 \tag{22}$$

$$-c\eta_2' = \rho_2\eta_2(1-\eta_2) + \Delta_2(1-\eta_1)\eta_2'' - \eta_1'\eta_2' \tag{23}$$

$$-c\Lambda' = \delta_3(\eta_2 - \Lambda) + \Lambda'' \tag{24}$$

The above ODEs are a set of coupled ODEs that cannot be solved analytically without some major assumptions. The next logical assumptions would be to eliminate these coupled ODEs. This is done with the assumptions $\Delta_2 << \rho_2$, sharp tumor edge and small propagation velocity. These equations are solved analytically in the literature and the solutions are used in this paper to check the quality of the numerical model. The profiles for $\Lambda(\zeta)$, $\eta_2(\zeta)$ and $\eta_1(\zeta)$ are as follows:

$$\Lambda(\zeta) = 1 - \frac{1}{2}exp(\sqrt{\delta_3}\zeta) \tag{25}$$

for $\zeta < 0$, and

$$\Lambda(\zeta) = \frac{1}{2}e^{-\sqrt{\delta_3}\zeta} \tag{26}$$

for $\zeta \geq 0$.

$$\eta_2(\zeta) = \frac{1}{1+e^{\rho_2\zeta/c}} \tag{27}$$

$$\eta_1(\zeta) = \frac{ce^{-pe^{q\zeta}+s\zeta}p^{s/q}q}{\gamma(r/q, pe^{-qe})} \tag{28}$$

for $\zeta < 0$, and

$$\eta_1 = \frac{ce^{-pe^{-q\zeta}-r\zeta}p^{r/q}q}{\gamma(r/q,p)p^{(s-r)/q} + \gamma(s/q,p) - \gamma(s/q,pe^{q\zeta})} \tag{29}$$

for $\zeta \geq 0$, where

$$p = \delta_1/(2c\sqrt{\delta_3}) \quad q = \sqrt{\delta_3}$$

$$r = 1/c \quad s = (\delta_1)/c\gamma(a,x) = \int_0^x e^{-t}t^{a-1}$$

Getting onto the numerical results of the work, data presented logical results that showed a decreasing density of cancerous tissue and increasing of healthy tissue density as distance from the tumor center increased. As time (τ) continued, the excess hydrogen ion concentration eliminated some of the healthy tissue, allowing for neoplastic tissue to branch out from the tumor center as time continued.

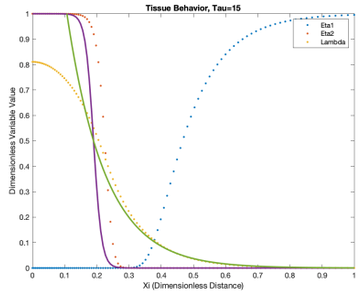

Figure 1: τ=15, no chemotherapy model. Analytical solution plotted for η_2 and Λ.

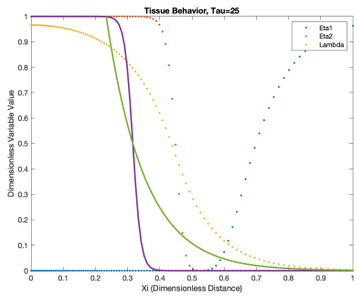

Figure 2: τ=25, no chemotherapy model. Analytical solution plotted for η_2 and Λ.

For the first two timesteps shared, seen in Figures 1 and 2, the anticipatory behavior of the ion concentration, as well as the evolving spatial behavior, is evident.

Commenting on the quality of the analytical fit, which was based used for the system without chemotherapy, the models capture the shapes of the plots decently. However, due to the high number of assumptions made to solve the analytical solution for this system, it is our belief that the numerical solution is the more accurate of the two. This is exemplified by the η_1 plot's exclusion due to its chaotic, noisy behavior not representative of the more realistic system decpited by the numerical solution.

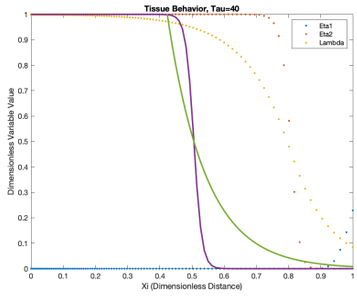

Figure 3: τ=40, no chemotherapy model. Analytical solution plotted for η_2 and Λ.

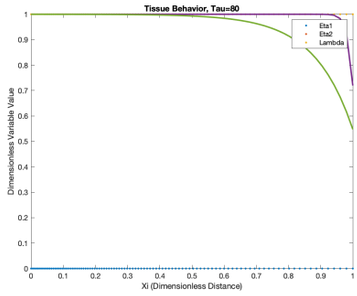

Figure 4: τ=80, no chemotherapy model. Analytical solution plotted for η_2 and Λ.

For the final two timesteps of τ=50 and τ=80 presented in Figures 3 and 4, the terminal behavior of the model is realized. Given sufficient time, the tumor density overwhelms the healthy tissue.

For the system considering chemotherapy treatment, behavior was clearly identical before a treatment dose was delivered.

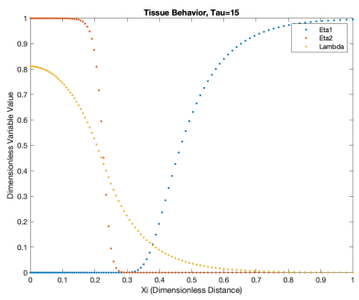

Figure 5: τ=15, chemotherapy model.

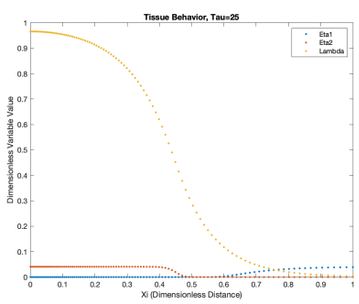

Figure 6: τ=25, no chemotherapy model.

The behavior of the dosing function, enacted at τ=14, is on full display when comparing Figures 5 and 6. Instead of the spreading behavior seen in the system without treatment, a sharp decrease in all tissue population occurs, with no spatial or typified (neoplastic vs. native) bias.

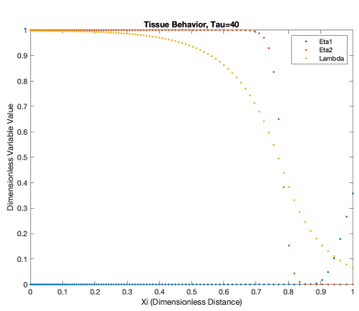

Figure 7: τ=40, chemotherapy model.

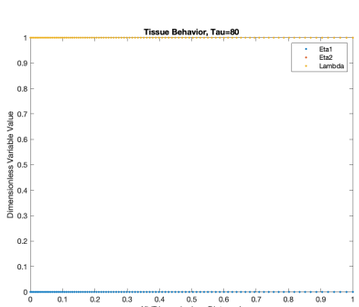

Figure 8: τ=80, no chemotherapy model.

Interesting to note here is how quickly the system returns to typical behavior after the dose. Though typical chemotherapy treatments would be applied for longer amounts of time and for repetitive doses, it is revealing of the persistence of neoplastic tissue diffusion (in reality and in this model). The validity of both schemes of numerical solutions, along with the analytical solution, are to be discussed in the Discussion section, below.

4 Discussion

Both numerical models accurately broadcast the expected end behavior of the system in time and distance, a sign of success at least in the slightest.

With that being said, the chemotherapy model suffers from a few glaring issues: minuscule treatment time (due to concerns with the numerical scheme), no space or time dependence, and lack of effect on the excess hydrogen ion concentration (leading to rapid regeneration of neoplastic tissue and return to an un-dosed system). This was a great introduction into numerical simulations, but the exact response to the treatment is too nuanced to be reliably modeled by a simple linear ODE approach.

The analytical model incurred many assumptions that hampered its accuracy, which made it a less adequate metric for validating the accuracy of the numerical methods presented. The trends of the plots match, however a delay/offset is evident for most values of τ.

One of the most interesting extensions of this problem realized from this paper is continued work with the dosing function. Due to its constant nature in this work (since the α and D parameters were unchagned), and only being implemented for a single value of τ, the chemotherapy results were interesting but limited in scope. The system returned to undisturbed behavior after a few τ steps, similar to enacting a noticeable delay on the system. This is a physically accurate model of tumor behavior, though the therapy has minimal effect on the long-term behavior of the tumor.

This single τ-point treatment was used because of the numerical scheme implemented. When a constant dose was attempted for multiple τ steps, issues with values of zero for the derivative arose and halted the progress of the simulation. Having a more complex model of the survival fraction would require restructuring the code, but would likely prove useful. Complexity of this project could be increased by: dosing with space or time dependence, dosing based off more biologically relevant parameters, or forcing for additional units of τ.

5 References

[1] Gatenby, Robert A., and Edward T. Gawlinski. "A reaction-diffusion model of cancer invasion." Cancer research 56.24 (1996): 5745-5753.

[2] Panetta, John Carl. "A mathematical model of periodically pulsed chemotherapy: tumor recurrence and metastasis in a competitive envi-

ronment." Bulletin of mathematical Biology 58.3 (1996): 425-447.

6 Contributions

Spencer Smith (50%): Numerical model and solution, Results, Model Development, Introduction, Numerical and analytical Discussion
Sai Kundan Vena (25%): Analytical discussion, Analytical solution, Abstract
Parth Shah (25%): Analytical solution, References

Special thanks to Kieran Fitzmaurice for helpful conversations & morale-boosting.

7 Appendix

7.1 MATLAB code

```
%spatial step
    Nr = 200; dr = 1/Nr; r = linspace(0,1,Nr+1).';

    %time step
    dt = 1E-5; Nt = 100;
    final_tau = 175; inner_time_steps = ceil(final˙tau / (Nt*dt));

    %create vectors for each variable
    eta1 = ones(Nr+1, 1); eta2 = zeros(Nr+1,1); lambda = zeros(Nr+1, 1);

    %dimensionless parameters via literature
    delta1 = 12.5; rho2 = 1; delta2 = 4E-5; delta3 = 70;

    eta1(end) = 0.01; eta2(end) = 1; lambda(end) = 1; %center of tumor
    eta1(1) = 1; eta2(1) = 0; lambda(1) = 0; %furthest from tumor

    %create vectors for each time derivative

    eta1_dt = zeros(Nr+1,1); eta2_dt = zeros(Nr+1,1); lambda_dt = zeros(Nr+1,1);

    %create vectors for each gradient and laplacian
    eta1_dr = zeros(Nr-1,1); eta2_dr = zeros(Nr-1,1); lambda_dr = zeros(Nr-1,1);
    eta2_dr2 = zeros(Nr-1,1); lambda_dr2 = zeros(Nr-1,1);

    %tau matrix
    tau = zeros(Nr+1,1);

    %create matrix to save each value
    eta1_matrix = zeros(Nt+1,Nr+1); eta2_matrix = zeros(Nt+1,Nr+1);
    lambda_matrix = zeros(Nt+1,Nr+1);

    %documented time loop
    for j = 1:Nt+1

        %inner time loop
        for k = 1:inner_time_steps

            %compute gradients for each variable
            eta1_dr = compute_gradient(eta1,dr);
            eta2_dr = compute_gradient(eta2,dr);
            lambda_dr = compute_gradient(lambda,dr);

            %compute laplacian for eta2 and lambda
            eta2_dr2 = compute_laplacian(eta2,dr);
            lambda_dr2 = compute_laplacian(lambda,dr);

            % calculate time derivative for regular points
            eta1_dt(2:end-1) = eta1(2:end-1).*(1 - eta1(2:end-1)) -
            delta1*lambda(2:end-1).*eta1(2:end-1);

            eta2_dt(2:end-1) = rho2 * eta2(2:end-1).*(1 - eta2(2:end-1)) +
            delta2.*((1-eta1(2:end-1)).*((r(2:end-1).ˆ4).*eta2_dr2(1:end) -eta2_dr(1:end).*
            (1-r(2:end-1)))-eta1_dr(1:end).*eta2_dr(1:end).*(r(2:end-1).ˆ4));

            lambda_dt(2:end-1) = delta3*(eta2(2:end-1) - lambda(2:end-1)) +
            r(2:end-1).ˆ3 .* (r(2:end-1).*lambda˙dr2(1:end) + (2 - 1./(1-r(2:end-1))).* lambda˙dr(1:end));

            %calculate time derivative for first point (r = 0)
            eta1_dt(end) = eta1(end) * (1 - eta1(end)) - delta1 * lambda(end) * eta1(end);

            eta2_dt(end) = rho2 * eta2(end) * (1 - eta2(end)) + ... delta2 * (1 - eta1(end)) *
        r(end)ˆ4 * (2 * eta2(end-1) - 2 * eta2(end)) / (drˆ2);
```

```
        lambda_dt(end) = delta3 * (eta2(end) - lambda(end)) ... + r(end)^4 * (2 * lambda(end-
    1) - 2 * lambda(end)) / (dr^2);

        %calculate time derivative for last point (r=1)
        eta1_dt(1) = 0; eta2_dt(1) = 0; lambda_dt(1) = 0;

        %increment time
        eta1 = eta1 + eta1_dt*dt; eta2 = eta2 + eta2_dt*dt;
        lambda = lambda + lambda_dt*dt;
    end

    %dose at tau = 25
    if (j == 25)
        D = 4; alpha = .8;
        eta1 = (eta1 + eta1_dt*dt).*exp(-alpha*D);
        eta2 = (eta2 + eta2˙dt*dt).* exp(-alpha*D);
    end

    eta1_matrix(j,:) = eta1; eta2_matrix(j,:) = eta2;
    lambda_matrix(j,:) = lambda; tau(j) = dt*j*inner˙time˙steps;
end

save('output_chemo', 'eta1_matrix', 'eta2_matrix', 'lambda_matrix', 'tau','Nt', 'Nr', 'r');
```

Python code for analytical solution

```
Import stuff to use
import numpy as np
from scipy.optimize import curve˙fit
import matplotlib.pyplot as plt

delta1 = 12.5
Delta2 = 4e-5
delta3 = 70
rho2 = 1
lim = 100
xi = np.linspace(-1,1,lim)
c = 2*np.sqrt(rho2*Delta2)
p = delta1/(2*c*np.sqrt(delta3))
q = np.sqrt(delta3)
r = 1/c
s = (delta1 - 1)/c
def funcgamma(a,x):
gamma = (np.exp(-x)*t^(x-1))/a - t^(a-1)/a
return gamma
Lambda =[]
for i in range(lim):
if xi[i] ¡ 0:
Lambda.append(1-(1/2)*np.exp(np.sqrt(delta3)*xi[i]))
else:
Lambda.append((1/2)*np.exp(-np.sqrt(delta3)*xi[i]))

    eta1 =[]
for i in range(lim):
if xi[i] ¡ 0:
eta1.append((c*np.exp(-p*np.exp(q*xi[i])+s*xi[i])*(p^(s/q))*q)/(gamma(r/q,p*np.exp(-q*xi[i]))))
else:
eta1.append((c*np.exp(-p*np.exp(-q*xi[i])-r*xi[i])*(p^(r/q))*q)/(gamma(r/q,p)p^((s-r)/q)+gamma(s/q,p)-
gamma(s/q,p*np.exp(q*xi[i]))))

    eta2 = 1/(1+np.exp(rho2*xi/c))
Lambda = np.array(Lambda)
plt.plot(xi,Lambda,'b-')
plt.plot(xi,eta1)
plt.plot(xi,eta2,'r-')
plt.xlabel('xi')
plt.ylabel('Dimensionless value eta1, eta2 and Lambda')
plt.savefig('output.jpg')
```