Cancer Model Simulation in Simulink Environment for Educational Purposes of Cancer Drug Dose Control

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***Abstract*— Cancer is one of the leading causes of death worldwide and the third cause of death in Iran after cardiovascular diseases and driving incidents. Therefore, having models that can describe and explain the process of cancer treatment is vital. One novel method for cancer treatment is to combine chemotherapy and immunotherapy. The purpose of immunotherapy is to enhance the body's immune system and reduce chemotherapy's side effects. Different models have been presented. The significance of the models is to understand the effect of each element on the treatment process. This article concerns the implementation of chemotherapy-immunotherapy cancer treatment in the Simulink toolbox of Matlab. Following the implementation of the model, the impact of chemotherapy, immunotherapy, and the combined technique on cancer cell growth is assessed. Simulating with Simulink allows for a graphical explanation as well as the ability to explain the hierarchy and effect of each piece. As a result, it may be advantageous for educational purposes. Simulink Toolboxes such as optimization, signal processing, and system identification allow us to analyze and control the cancer model.**

***Index Terms*—** Tumor cell model, chemotherapy, immunotherapy, Simulink, Simulation

I. INTRODUCTION

It is common to categorize cancers based on the cell tissue from which they have been generated. Generally, cancers are categorized into five categories. These classes are carcinoma, sarcoma, lymphoma, leukemia, and blastoma. Cancers occur due to the uncontrolled proliferation of cells and could exist in all parts of the body, from hard tissue such as bones to nervous tissue.

Most cancers do not have a particular symptom and most symptoms, do not appear in the same way from patient to patient. Many factors, such as substances containing arsenic, air pollutants, and some viruses, are involved in the existence of this disease. Cancer is a disease that disrupts the cellular disposition and results in interference with key and vital genes. This molecular disorder has a detrimental effect on cellular division and results in a lack of cellular differentiation [1, 2, 3]. There are various mathematical models regarding cancer therapy processes in the literature. Pillis and his colleagues [4] have suggested a model that involves Cancer cells, three types of defensive cells and two drug concentrations. In a continuation on his efforts Pillis has utilized empirical data to enhance the values of the parameters in his model [5]. Kim and colleagues proposed a model with cancer cells, regulatory cells, natural killer cells, and two types of immune cells (CD4T cells & CD8T cells). There are two types of cancer genes worth mentioning. The first are oncogene genes, which have a positive effect on tumor formation. The second type of gene is the tumor suppression gene, which has a negative effect on tumor formation. Cancer can be cured in a variety of ways. Radiotherapy is one of the most common ways that cancer cells are targeted by X-ray. Surgery is another common treatment option for immature tumors (in clinical terms, have not yet metastasized). Chemotherapy is a method of cancer treatment that uses various chemical substances in order to eliminate cancer cells. Although it may have unfavorable side effects, it has proven to be very effective in the majority of cases. Using chemotherapy may have multiple purposes, such as cancer treatment without the presence of other treatment methods or utilizing chemotherapy after specific therapies, such as surgery, to assist in the treatment process. Other purposes may include reducing pain and preparing the patient for further stages of the overall therapy. Different chemotherapy drugs have different side effects, but the key thing to note is that the injected drugs also have a negative effect on the immune system in addition to their positive effect on eliminating cancer cells. In order to neutralize this negative effect, immunotherapy is used. Immunotherapy utilizes the immune system of the individual to recover from cancer. The goal of immunotherapy is to boost the immune system of the patient against cancer cells by increasing its efficiency. Immunotherapy has three subcategories. Immune response modifiers, monoclonal antibodies, and vaccines. Immune response modifiers target the interleukins, which also include IL-2. Monoclonal antibodies target certain cancerous antigens. Monoclonal antibodies can differentiate between healthy and cancerous cells. Vaccines, which are produced by cancerous cells, assist the immune system in the identification of cancer cells [7]. Modeling is the correct portrayal of a system's behavior. We can examine and simulate the system under investigation using a model. Because the chosen model

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is heavily dependent on our demands, the system model is chosen depending on our control objectives. One of the most well-versed and well-rounded models is that of Pinho, which was published in 2013[8]. The model presented in [9] is based on the model of [7] which has more detail for cellular interactions and chemo-immunotherapy representation. Therefore, the assumptions of both papers are similar. In [10], a more improved and enhanced model is presented that utilizes interleukins to model cancer growth and its treatment. Naderi [11] has designed a controller to regulate the injected drug dosages based on the model presented in [6]. Sotolongo-Costa [12] proposed a model to represent the interaction dynamics of lymphocytes and tumor cells. They use cytokines alone as their periodical immunotherapy treatment and explain the dynamics by examining the interaction between the immune system and hostile cells. Robertson-Tessi [13] has also proposed a model for the interaction of tumor cells and the immune system. In order to govern the equations and parameters, experimental and clinical outcomes are incorporated. This model includes three types of T cells: dendritic cells, tumor cells, and plasma cells. The main purpose of this model is to find optimal antigenicity for maximum immune system response. It is stated that as antigenicity increases, immune cell access to tumor cells will be limited due to an increase in immunosuppression. Roberto A. Ku-Carrillo [14] has evaluated the effect of obesity on cancer growth and the immune system response. He does so by consolidating four differential equations containing the density of Immune cells, density of Cancer cells and the density of normal cells and the density of Fat. Denise Kirschner [15] utilizes a four-dimensional model that illustrates the dynamics between tumor cells, effector cells, and IL-2. Svetlana Bunimovich-Mendrazitsky [16] presents a system of nonlinear ODE's to Bladder Cancer and utilizes mouse and human in vitro data to verify the model. In regards to experimental and clinical studies, Alexandre Boissonnas [17] has evaluated the effect of cytotoxic CD8+ T-cells on the elimination of tumor cells through in vivo imaging. It is shown that the CD8+ T- cells migrate, and when they come into contact with tumor cells, their associated antigen is dispersed. It is seen that CD8+ T-cells continue their movement if they come across dead tumor cells. By including multiple state variables and evaluating the dynamical uncertainty of the system, Kim presents a complex model for cancer. This model represents the cancer treatment process by having four classifications, with three of those classes being related to immune system cells and one relating to cancer cells. The amount of drug concentration in the bloodstream is also considered. Model [6] includes a system of differential equations containing eleven ordinary differential equations (ODEs). We have used model [6] as the basis of our study. The purpose of this paper is to implement Kim's comprehensive model into the Simulink toolbox of MATLAB, thereby simulating and evaluating the model in different circumstances and in a more thorough manner. Because of the high level of abstraction, Simulink simulation allows for a graphical description as well as the ability to illustrate the hierarchy and effect of each element. As a result, it can be beneficial for educational purposes.

II. RESEARCH METHODOLOGY

The model used in this article is a hybrid of chemotherapy and immunotherapy. This model, which is the one presented in [6], is comprehensive and has great potential for simulating different treatment methods. This model has eleven ODEs. The state variables are introduced in Table I. In addition, the differential equations are presented.

TABLE I
State Variables and Their Definition

variable	type	Definition	Unit
T	Cancer cell	cancer cell	number
N	Immune system	natural immune response (Natural Killer cell-NK)	number
E_T		Adaptive Immune response(CD8+T)	number
D_U		Unlicensed dendritic cells	number
D_L		Licensed dendritic cells	number
H_T		CD4+T cells	number
I_2		IL-2	IU/L
G_T	Immune Suppressive	Regulatory cells	number
S		TGF-β	IU/L
I_10		IL-10	IU/L
M	Chemotherapy	Chemotherapy drug concentration	IU/L

$$\frac{dT}{dt} = aT(1-bT) - c1TN - \frac{dTE_T}{E_T + eT}\frac{1}{\left(1+\left(\frac{G_T}{E_T}\right)\right)\left(1+\left(\frac{S}{s_1}\right)\right)} - k_T(1-e^{-M})T \quad (1)$$

$$\frac{dN}{dt} = b1 - d_N N - c_2 TN + \frac{P_N N I_2}{q_N + I_2} - k_N(1-e^{-M})N \quad (2)$$

$$\frac{dE_T}{dt} = \frac{\alpha_1 I_2 D_L m_E}{\left(1+\left(\frac{S}{s_1}\right)\right)(i_1+I_2)(d_1+D_L)} - c_3 E_T T - K_{ET}(1-e^{-M})E_T + w_1 u_E(t) \quad (3)$$

$$\frac{dD_U}{dt} = \frac{pT}{\left(1+\left(\frac{I_{10}}{i_2}\right)\right)\left(\left(1+\left(\frac{G_T}{g_2}\right)\right)\right)} - \frac{\gamma_1 D_U}{1+\left(\frac{D_U}{m_H}\right)} - d_{D_U} D_U - K_{D_U}(1-e^{-M})D_U \quad (4)$$

$$\frac{dD_L}{dt} = \frac{\gamma_1 D_U}{1+\left(\frac{D_U}{m_H}\right)} - d_{D_L} D_L \quad (5)$$

$$\frac{dH_T}{dt} = \frac{\alpha_2 I_2 (D_L + D_U) m_H}{\left(1+\left(\frac{S}{s_2}\right)\right)(i_1+I_2)(d_1+D_L+D_U)} - \frac{\gamma_2 H_T S}{S+s_3} - d_{H_T} H_T - k_{H_T}(1-e^{-M})H_T \quad (6)$$

$$\frac{dG_T}{dt} = \frac{\gamma_2 H_T S}{S+s_3} + \frac{\alpha_3 I_2 D_L m_G}{(i_1+I_2)(d_1+D_L)} - d_{G_T} G_T - K_{GT}(1-e^{-M})G_T \quad (7)$$

$$\frac{dS}{dt} = p_1 G_T + p_2 T - d_s S \quad (8)$$

$$\frac{dI_2}{dt} = \frac{\alpha_4 H_T}{\left(1+\left(\frac{I_{10}}{i_3}\right)\right)\left(\left(1+\left(\frac{S}{s_4}\right)\right)\right)} - d_{I_2} I_2 + w_2 u_{I_2}(t) \quad (9)$$

$$\frac{dI_{10}}{dt} = p_3 G_T + p_4 T - d_{I_{10}} I_{10} \quad (10)$$

$$\frac{dM}{dt} = -d_M M + w_3 u_M(t) \quad (11)$$

The treatment process and eliminating cancer cells is as follow: Natural killer cells naturally attempt to oppose cancer cells. To enhance the treatment process to eliminate cancer cells, chemotherapy and immunotherapy are utilized. CD4 T cells and CD8 T cells are produced during chemotherapy and immunotherapy drug injection. CD4T & CD8T cells secrete IL-2, which reinforces one another. In addition, dendritic cells (DC) assist CD4T, CD8T, and NK cells in identifying cancer cells. DC cells are divided into two categories: licensed and unlicensed. The crucial element to remember is that unlicensed dendritic cells are incapable of detecting cancer cells. Another important point to consider is the unfavorable consequences of chemotherapy, which result in the creation of regulatory cells. Regulatory cells reduce the effectiveness of the immune system. As illustrated in Fig. 1, these cells release IL-10 and TGF-B, resulting in a decrease in the efficacy of the immune system. Table II displays the model parameters and the amount of each parameter. Table III also shows the model's initial settings.

Fig. 1 shows the implementation of the model in the Simulink environment. It can be seen that the model has three control inputs for cancer control. The goal in cancer control is to eliminate as many cancer cells as possible by utilizing the least amount of Chemo and immune therapies. In Fig. 2, the next implemented level in Simulink is presented, which shows eleven state variables in the frame of eleven distinct blocks. The relationships between the blocks are also visible. Fig. 3-Fig. 13 shows the details of the differential equations for each state variable at the next level.

TABLE II
Parameters of the System Model

variable	amount	variable	amount	variable	amount
a	$0.431Day^{-1}$	i_1	4909IU/L	k_{H_T}	0.6
b	1.02×10^{-9}	d_1	579579	α_3	$3.6Day^{-1}$
c_1	3.177×10^{-13} $Cells^{-1}Day^{-1}$	c_3	3.42×10^{-10}	m_G	175900
d	$0.9Day^{-1}$	K_{ET}	$0.6Day^{-1}$	d_{G_T}	$0.1Day^{-1}$
e	1.2	P	$0.1Day^{-1}$	K_{GT}	0.6
g_1	1.2	i_2	1200IU/L	p_1	3.6×10^{-4} IU/L $Cells^{-1}Day^{-1}$
s_1	5×10^4	g_2	2×10^{-9}	p_2	2.2×10^{-3} IU/L $Cells^{-1}Day^{-1}$
k_T	$0.9Day^{-1}$	γ_2	$0.5Day^{-1}$	d_s	$14.3Day^{\backslash -}$
b_1	$3121875Day^{-1}$	m_H	1053600	i_3	2250IU/L
d_N	$0.0125Day^{-1}$	d_{D_U}	$0.14Day^{-1}$	α_4	$0.278IU/LCells^{-1}Day^{-1}$
c_2	3.177×10^{-13}	K_{D_U}	$0.05Day^{-1}$	s_4	18000 IU/L
p_N	$0.0668Day^{-1}$	u_1	6053600	d_{I_2}	$12.5Day^{-1}$
q_N	250360 IU/L	d_{D_L}	$0.5Day^{-1}$	p_3	4.2×10^{-5}IU/L $Cells^{-1}Day^{-1}$
k_N	$0.6Day^{-1}$	α_2	$1.9Day^{-1}$	p_4	3.9×10^{-4}IU/L $Cells^{-1}Day^{-1}$
a_1	$16Day^{-1}$	γ_2	0.022	$d_{I_{10}}$	$20Day^{-1}$
m_E	526800	s_3	34000	d_M	$0.9Day^{-1}$
s_2	580000IU/L	d_{H_T}	$0.1Day^{-1}$		

TABLE III
Initial conditions of the state variable

state variable	initial condition	state variable	initial condition
T	1×10^8	G_T	1.795×10^5
N	2.5×10^8	S	0
E_T	5.268×10^5	I_2	1173
D_U	4.725×10^7	I_{10}	0
D_L	10	M	0
H_T	1.0536×10^6		

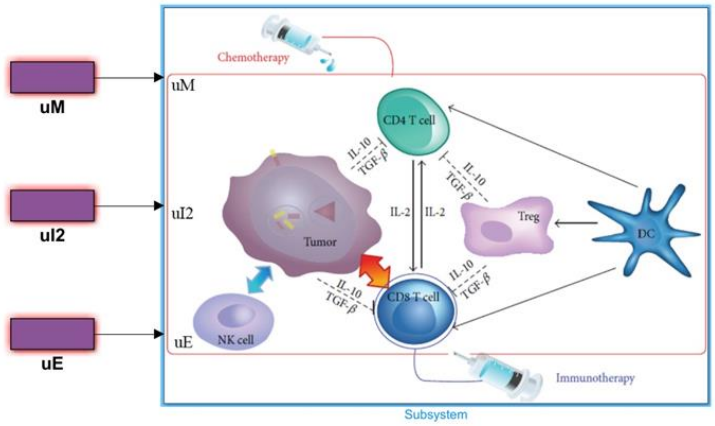

Fig. 1. Generlized Schematic of the implemented Cancer model in the Simulink environment .inputs of the model represent the drugs used for cancer treatment. The number of cancer cells in addition to other state variables can be obtained as the outputs.

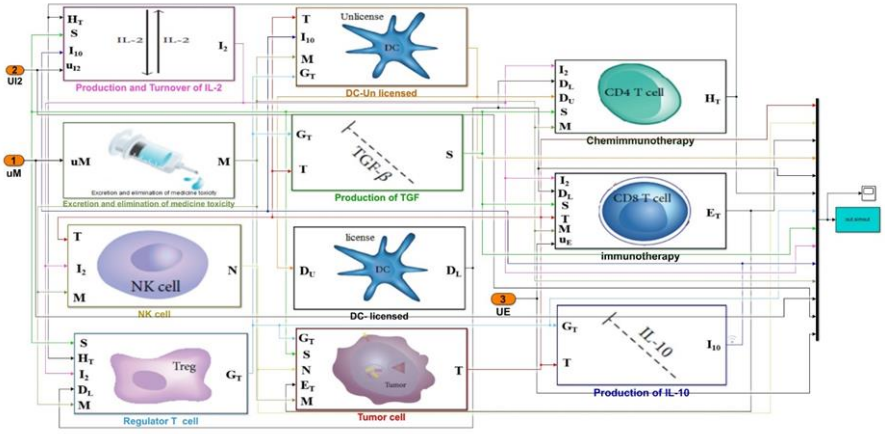

Fig. 2. Schematic of the block diagram of the implemented cancer model in the Simulink environment (this is the lower level to Figure 1) each block represents a state variable of each model. Number of cancer cells (T), number of Natural Killer cells (N), number of CD8T cells (E), number of CD4T cells (H), the amount of IL-2 discharge (I2),number of regulatory cells T (G), the amount of B-TGF discharge (S), the amount of IL-10 discharge (I10), number of licensed dendritic cells (DL), number of unsilenced dendritic cells (DU) and the amount of drug concentration in the blood stream (M) are represented.

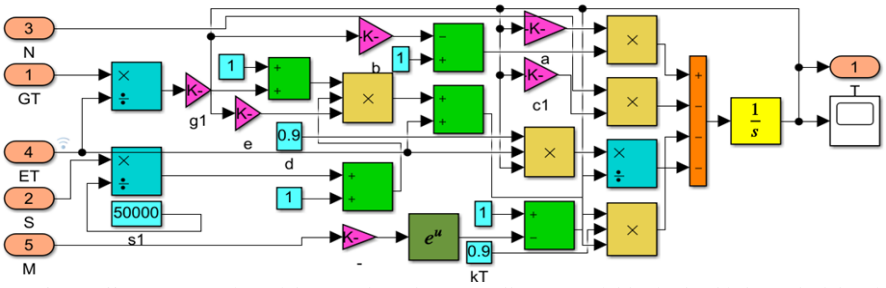

Fig. 3. Implementation of the differential equation of the "number of cancer cell" state variables in the third Level of the Simulink environment.

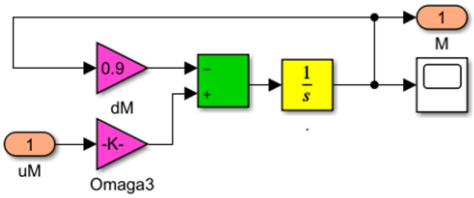

Fig. 4. Implementation of the differential equation of the "Chemotherapy drug concentration" state variable in the Simulink environment.

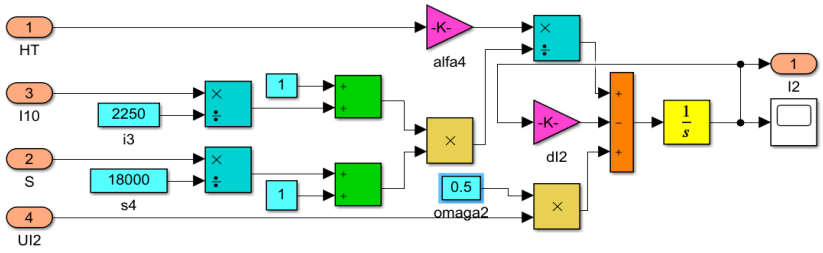

Fig. 5. Implementation of the differential equation of the amount of discharge of IL-2 in the Simulink environment.

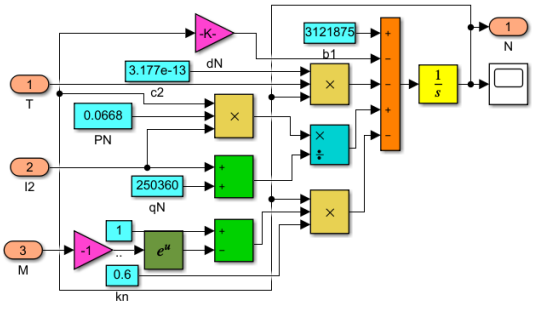

Fig. 6. Implementation of the differential equation of "number of Natural Killer cells" in the Simulink environment.

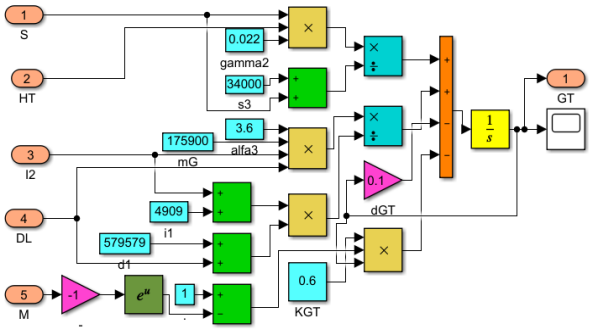

Fig. 7. Implementation of the differential equation of "number of Regulatory T cells" in the Simulink environment

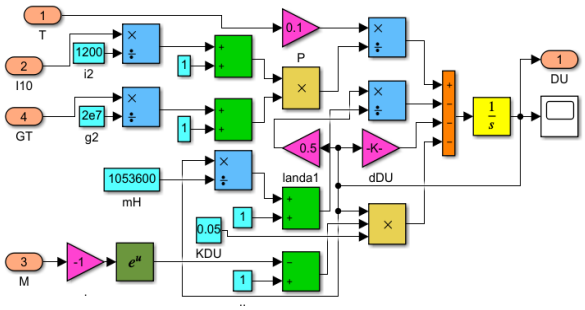

Fig. 8. Implementation of the differential equation of "Unlicensed Dendritic cells" in the Simulink environment.

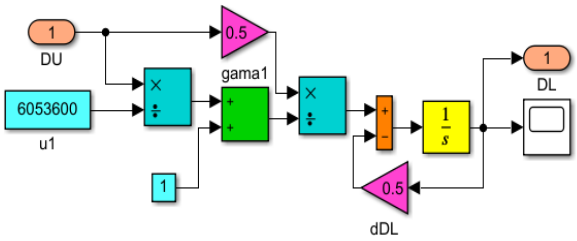

Fig. 9. Implementation of the differential equation of "licensed Dendritic cells" in the Simulink environment.

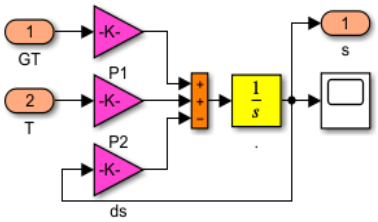

Fig. 10. Implementation of the differential equation of "TGF-β" discharge in the Simulink environment.

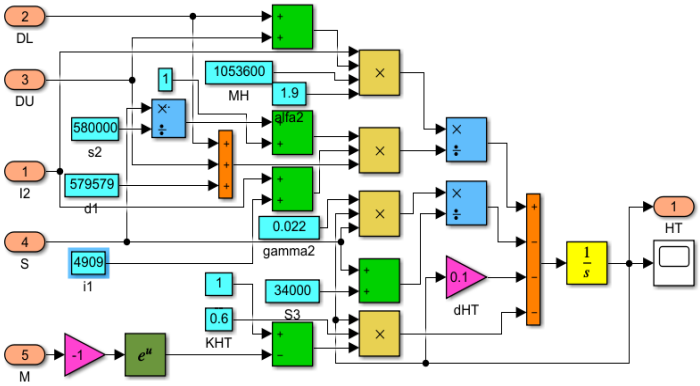

Fig. 11. Implementation of the differential equation of "number of CD4T cells" in the Simulink environment.

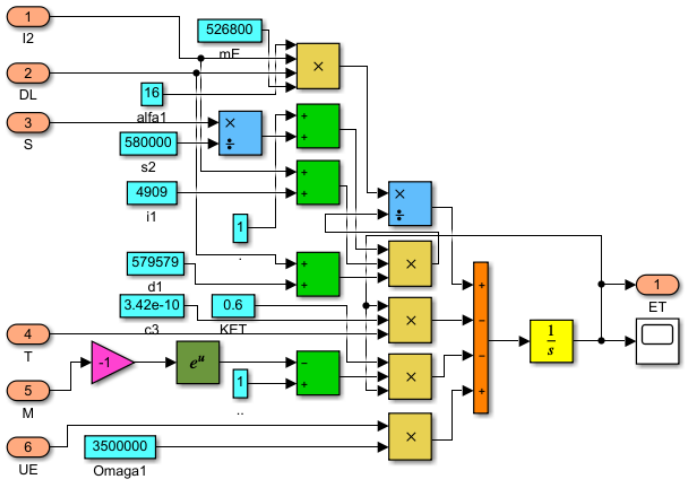

Fig. 12. Implementation of the differential equation of "number of CD8T cells" in the Simulink environment.

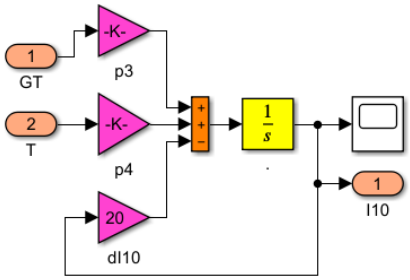

Fig.13. Implementation of the differential equation of the amount of discharge of IL-10 in the Simulink environment.

III. SIMULATION RESULTS

By simulating the implemented model, the dynamical changes of the eleven state variables of the model are evaluated. Figure 14 depicts the simulation results for the events of no therapeutic intervention and only chemotherapy over the course of fifty days. The results show that the number of cancer cells in the absence of medicine becomes ten times larger than its original size in the span of ten days. Only chemotherapy with the recommended drug dosage reduces the number of cancer cells in that time frame. Figure 15 shows the effect of the changes in drug dosage (u_M) for chemotherapy on eliminating cancer cells. With the increase in chemotherapy drug dosage, the rate of cancer cell elimination increases. This effect is such that in the absence of u_M the number of cancer cells increases and, after a while, remains constant, but with the increase in drug dosage, the uphill trend of the number of cancer cells is reduced. The reduction of the number of cancer cells is reduced in such a way that at u_M=1e-7 the ultimate number of cancer cells decreases and by further increasing the amount of dosage. This downhill trend continues until cancer cells do not have the opportunity to increase from the very beginning. This is evident at u_M=50e-7 Figure 16 shows the effect of Immunotherapy drug dosage on the number of cancer cells. With the increase of the drug dosage of immunotherapy, the rate at which the number of cancer cells increases also experiences a decline such that before uE=10 the effect of the Immunotherapy drug dosage is not apparent, and the number of cancer cells reaches their maximum after a few days. Even so, after uE=10 as we increase the amount of drug dosage not only does the rate of cancer cell growth decline but also the Maximum number of cancer cells also decreases. As shown in Figure 16 as uE reaches the amount of 10.9 cancer cell growth is limited and eventually reaches zero. By increasing the amount of drug dosage further and reaching uE =50, one can see the decrease in cancer cells from the very beginning of the treatment. In Figure 17, three different therapeutic methods with determined amounts of drug dosages are compared to each other and the state of no medicine in relation to the efficacy of cancer cell elimination. Immunotherapy has a significant effect on cancer cell elimination in such a way that in only fifteen days it stops Figure 16 shows the effect of Immunotherapy drug dosage on the number of cancer cells. With the increase of the drug dosage of immunotherapy, the rate at which the number of cancer cells increases also experiences a decline such that before uE=10 the effect of the Immunotherapy drug dosage is not apparent, and the number of cancer cells reaches their maximum after a few days. Even so, after uE=10 as we increase the amount of drug dosage not only does the rate of cancer cell growth decline but also the Maximum number of cancer cells also decreases. As shown in Figure 16 as uE reaches the amount of 10.9 cancer cell growth is limited and eventually reaches zero. By increasing the amount of drug dosage further and reaching uE =50, one can

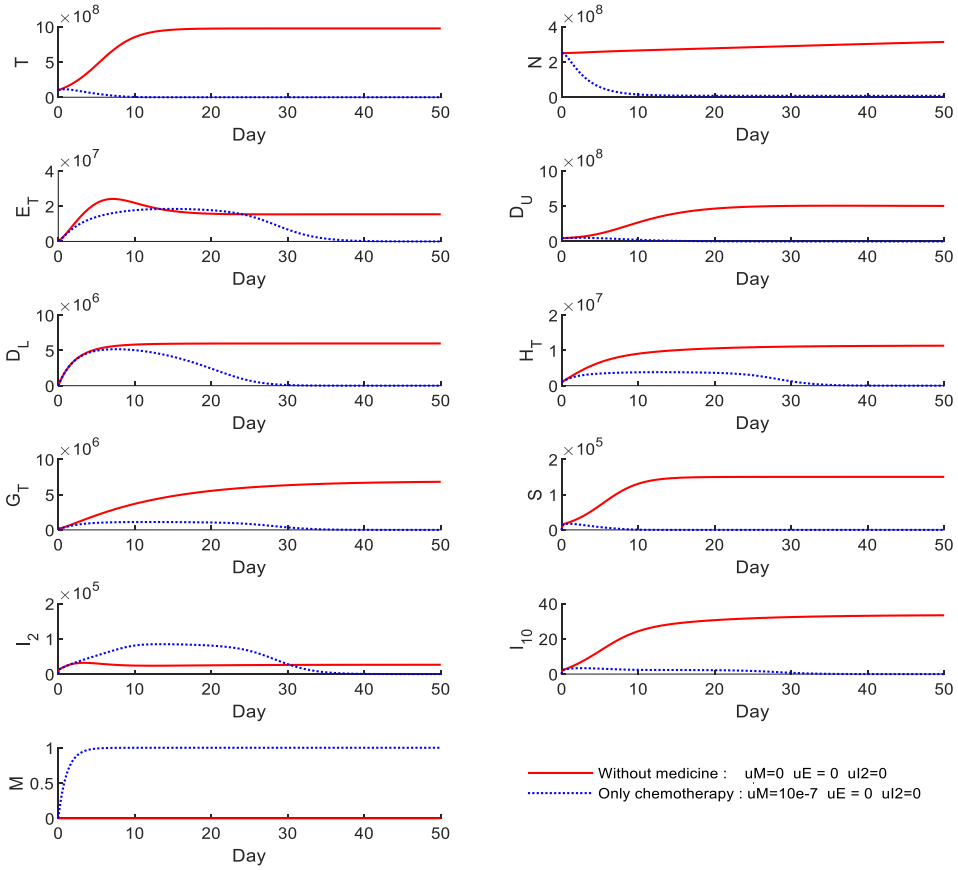

Fig. 14. State variable changes in the case of no medicine and only chemotherapy

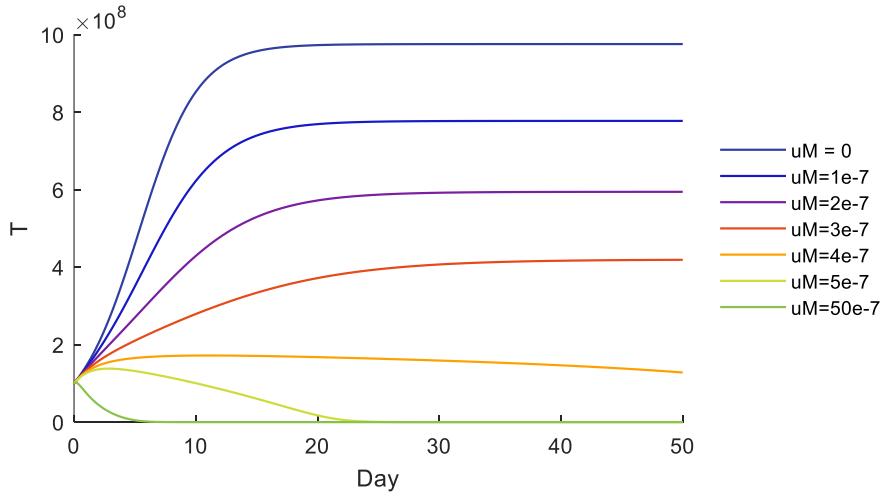

Fig. 15. The effect of Chemotherapy drug dosage (uM) on cancer cells elimination

see the decrease in cancer cells from the very beginning of the treatment. In Figure 17, three different therapeutic methods with determined amounts of drug dosages are compared to each other and the state of no medicine in relation to the efficacy of cancer cell elimination. Immunotherapy has a significant effect on cancer cell elimination in such a way that in only fifteen days it stops cancer cell growth and compared to no medicine the maximum amount of cancer cells is much lower. In immunotherapy, the number of cancer cells converges to zero only after 35 days. chemotherapy is even much more effective, that is it stops cancer cell growth very quickly and in a short span of time and causes a decrease in the number of cancer cells. The time needed for chemotherapy to eliminate cancer cells is quite long, such that after fifty days of treatment, there are still a significant number of cancer cells. Combining chemotherapy and immunotherapy with the same amount of dosage will give better results in the elimination of cancer cells. In the combined chemo-immunotherapy treatment, cancer cell growth is prevented from the very beginning and quickly results in cancer cell elimination, such that after ten days the number

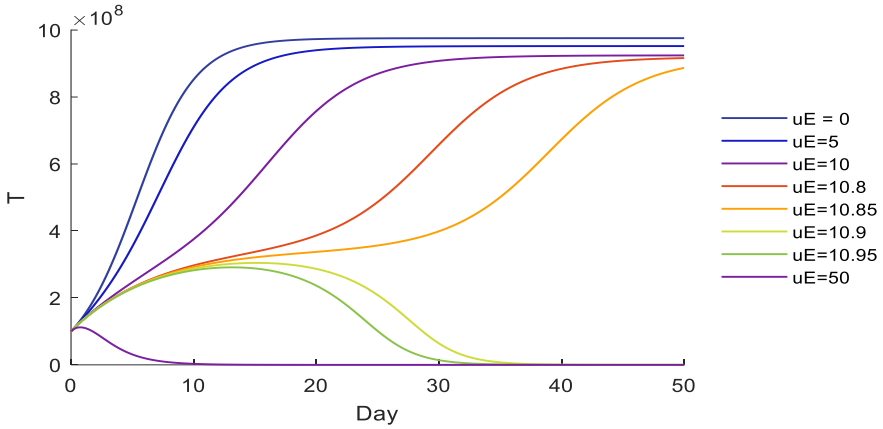

Fig. 16. The effect of changes in Immunotherapy drug dosage (uE) on cancer cell elimination

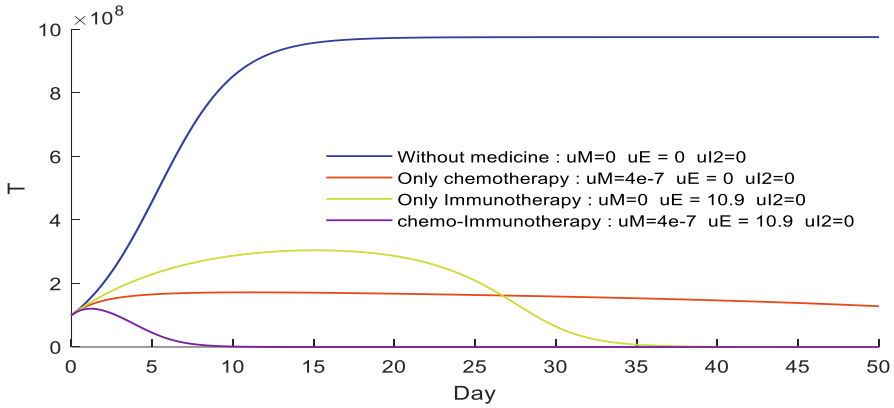

Fig. 17. Comparing the efficacy of three different treatment methods (only Chemotherapy, Only Immunotherapy, Chemo-Immunotherapy) in contrast to without medicine on cancer cell elimination

of cancer cells has converged to zero. The key point to note here is that these results are based on a particular amount of drug dosage. Therefore, utilizing different drug dosages may have different results.

The drug dosage change of Chemotherapy and Immunotherapy on different state variables is evaluated. Figure 18 shows the change in "the number of regulatory T cells" based on the increase of Immunotherapy drug dosage, against the amount of time (days) spent from the beginning of the treatment. In the absence of the Immunotherapy drug, "the number of regulatory T cells" reaches their maximum number of 10×10^8 in ten days. With the increase in the immunotherapy drug dosage, the maximum number of regulatory T cells decreases slightly, simultaneously the number of days it takes to reach this maximum increases. This trend can be seen up to uE=10.85 where it takes fifty days for the regulatory T cells to reach their maximum number. From this dosage on and as we increase the dosage, a strong downhill trend is apparent in which the number of regulatory T cells will eventually converge to zero. The amount of dosage at uE=50 has a more desirable effect compared to other dosages, such that from the start, the increase in the number of regulatory T cells is prevented and converges to zero after 10 days. Figure 19 illustrates the change in the number of regulatory T cells based on the increase in chemotherapy drug dosage and the amount of time (days) spent since the beginning of the treatment. In the absence of the immunotherapy drug, "the number of regulatory T cells" increases. With the increase in drug dosage, the rate of increase in the number of regulatory T cells declines, such that by injecting a small amount of chemotherapy uM=1e-7, the number of regulatory T cells decreases from the 7×10^6 reach in the absence of chemotherapy to 4×10^6. By continuing to increase the chemotherapy drug dosage, the increase in the number of regulatory T cells also continues to decline. In the maximum amount of drug dosage, uM=50e-7, the increase of the number of regulatory T cells is very limited and converges to a small value after thirty days.

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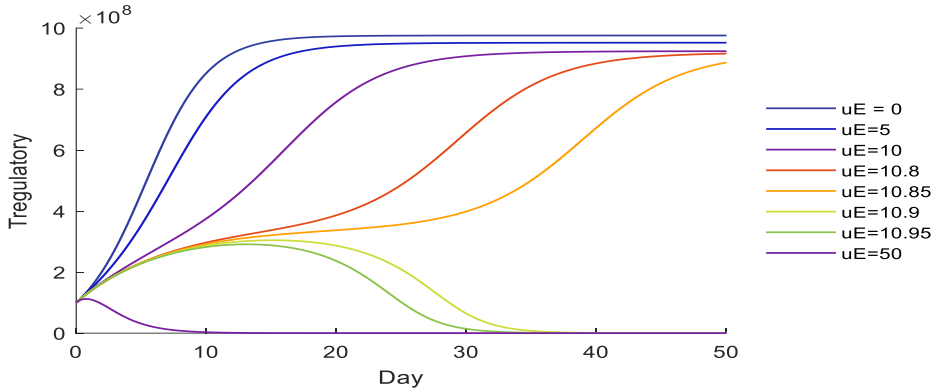

Fig .18. The change of the number of regulatory T cells based on the increase of the Immunotherapy drug dosage against time.

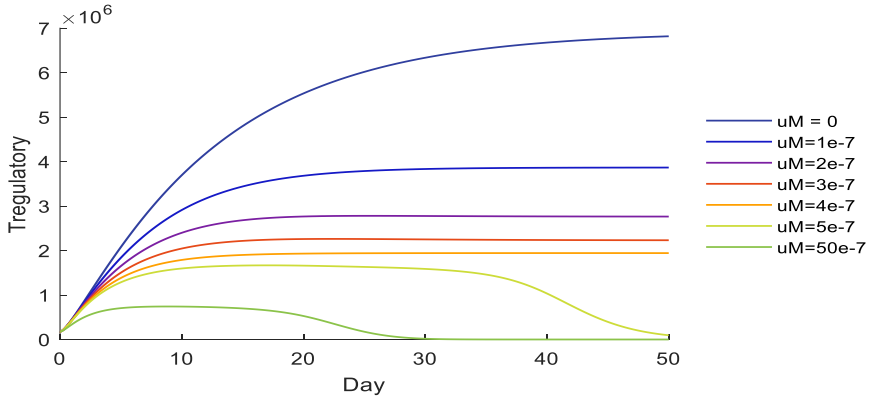

Fig .19. The change of the number of regulatory T cells based on the increase of the Chemotherapy drug dosage against time.

Figure 20 illustrates the change in CD4T cells which decreases with the increase in the chemotherapy drug dosage as the uphill trend of the number of CD4T cells decreases. In the absence of any drug dosage and after fifty days, the number of CD4T cells will reach approximately to12×10^6. By injecting a dosage of uM=1e-7 of the chemotherapy drug the number of CD4T cells will reach than 8×10^6 after fifty days. In addition, by increasing of the drug dosage to uM=50e-7 the maximum of number of CD4T cells will only be 2×10^6 and will converge to zero after thirty days. Figure 21 shows the change in CD8T cells. The number of CD8T cells increases with the increase in the chemotherapy drug dosage, such that in the absence of the drug dosage, after some fluctuation, the number of CD8T cells converges to 1.5×10^7 and by increasing the drug dosage, this convergence will be at a higher number. At uM=4e-7 the number of CD8T cells will not even have a fluctuation phase and will continuously increase. However, by increasing the Chemotherapy drug dosage further, the uphill trend suddenly stops and the number of CD8T cells will start decreasing such that at uM=5e-7 after thirty days, the number of CD8T cells will reach its maximum value, but will immediately have a strong downhill trend and after fifty days will have an approximate 0.5×10^7 cells. Eventually, at M=50e-7 the upward trend only takes ten days, and in only thirty days, the CD8T cells will converge to zero. This abrupt alternation in the number of CD8T cells with the increase of chemotherapy might be interpreted as one of the side effects of chemotherapy meaning, by further increasing the amount of chemotherapy from a certain dosage, a decrease and eventual elimination of other influential cells is expected.

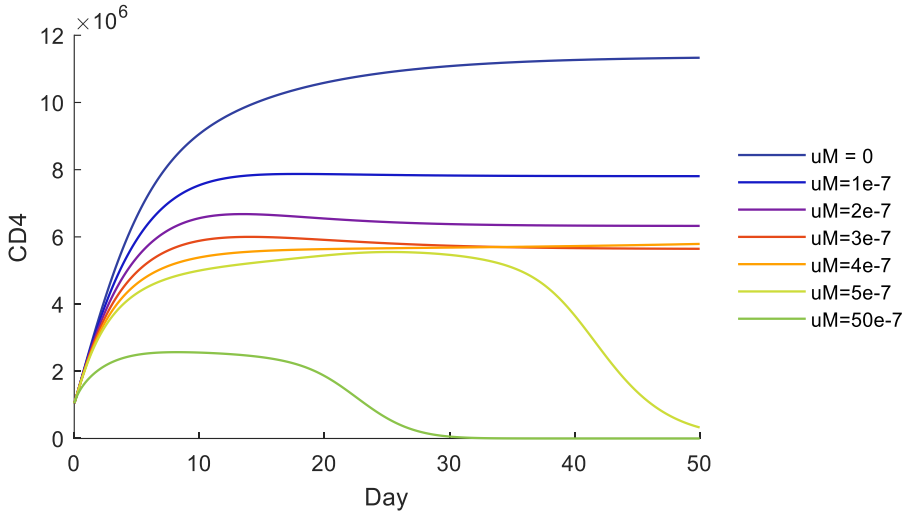

Fig. 20. The change of the number of CD4T cells based on the increase of the Chemotherapy drug dosage against time

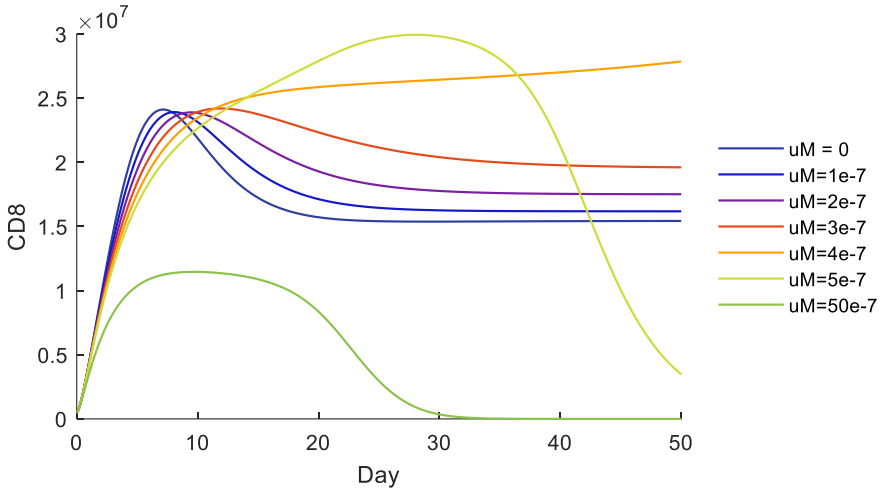

Fig. 21. Change in the CD8T cells, based on the increase of the Chemotherapy drug dosage against time

IV. DISCUSSION

A model of chemo-immunotherapy was simulated. Simulating this model in the graphical environment of Simulink provides us with valuable information regarding the evaluation of cancer growth and the treatment process. Due to the graphical description of the model in Simulink, understanding each element and their relations to one another is much simpler, and one can easily observe the effects of changes in each element on the model as a whole. The description of the model is done in a hierarchical manner, which makes it possible for individuals with different levels of knowledge and experience to connect with the model.

By simulating the cancer model in different circumstances, including solely chemotherapy, solely immunotherapy, and the combination method, it is seen that the combination method is much more effective. In future works, the efficacy of different controllers with the purpose of reducing cancer cells in the least amount of time and with the least amount of Chemo-Immunotherapy will be studied. From an educational standpoint, the knowledge acquired from the simulation can be utilized for other cancer models and other dynamical models in the fields of engineering and medical sciences.

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