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Interaction and Development of Breast Cancer Cells with Immune Response Using First Order ODE

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Abstract. Breast cancer arises when cells develop uncontrollably in the breast to form tumour cells. The risk of having breast cancer rises as a woman continues to age. Thus, details about the early stages of cancer progression can help a woman make early treatment choices, preventing them from being diagnosed with these harmful cancers. It is believed that cytotoxic T lymphocytes (CTLs) act as effector cells to eradicate cancer cells. CTLs and tumor cells were discovered to be around a "predator-prey" relationship, with CTL acting as the predator along with tumor cells acting as the prey. In this paper, we examined steady-state solutions for two numerical differentiation using the Jacobian matrix. We will also examine the stability region of breast cancer cells in two different phases to describe its progress in different types of the human body at various phases. Also, compare tumour cells population development in the duration of interphase and metaphase in the presence and the absence of immune response, which is dependent on the CTL population, will be observed by applying Fourth Order Runge Kutta (RK4) method. We can see that the Runge-Kutta method is an important method for approximate solutions to ordinary systems with known initial conditions. We achieved populations value of tumour cells throughout interphase and mitosis as well as the population of the immune system using the method. Based on our observations, we draw the conclusion that persons with a higher immune system are more likely to be able to fight cancer for for a period of time than cancer sufferers with a poor immune system. We may observe that the cancer cells were reduced in a shorter period of time with a high immune response population compared to other lower CTL values.

INTRODUCTION

The topic of tumour growth modelling is an extensive analysis by researchers, concentrating on various aspects of cancer progression. This highlights the role of immune systems in tumour control and applying an ordinary differential equation (ODE) model of cytotoxic T lymphocyte (CTL) reaction in conjunction with tumour cell populations. CTL can kill tumour cells, as demonstrated by latest significant advancements in cancer immunotherapy. It is believed that CTL act as effector cells to eradicate cancer cells [1]. Past researchers discovered that tumour cells and CTL were competing as 'predator-prey', with CTL as the predator and tumour cells as the prey [2]. Cancer has become one of the world's leading causes of mortality today. Per year, over 8.2 million individuals globally succumb to cancer [3]. In fact, more than 13 million people are expected to be infected by 2030 [4]. According to the World Health Organization, most cancer-related deaths occur in economically developing countries. Even though there have been numerous advancements in treating cancer, such as surgical procedures, chemotherapy, immunotherapy, and radiation therapy, much remains a mystery about the mechanisms of how cancerous cells are formed, transmitted, and killed. This scenario pushes scientists worldwide to build theories and practical methods to combat cancer [5]. Because the connections among tumour cells and some further forms of cells are complicated, most researchers concentrated on

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specific issues. Link to this issue, researchers found that the immune system performs a crucial function in the human body to combat tumours. However, due to unpredictable tumour behaviour, the immune system's capacity is limited [6].

Cancer is not just a leading source of fatality globally, but it is also a dynamic structure in its reasons and impacts on the human body. Breast cancer is a very diverse disease that affects individuals very differently and is triggered by a wide range of unique genetic changes in breast epithelial cells [7]. Cancer cells split uncontrollably, infiltrate healthy human tissue, and spread to remote locations in different tissues, where they develop additional metastases [8]. This significantly obscures the affecting and complete destruction of malignant cells. Chemotherapy, combined with radiation therapy and surgical procedures, is among the most often used anti-tumour remedial techniques today. Numerous lives have been saved with chemotherapy, and it has the lowest mortality rates of multiple cancer forms. Despite this, in specific, chemotherapy induces severe side effects, both physical and psychological destruction, resulting from the anti-cancer agents' lack of specificity [9]. The challenge is that the medications do not only target diseased tissue; they also attack healthy organs. Chemotherapy is an effective method for killing cancer cells in medical care, but it also kills healthy cells. Chemotherapy is typically the earliest medical therapy for any type of cancer, including breast cancer. Almost all of the drugs used are particular drugs in the cycle phase, like vincristine and paclitaxel, that disrupt certain cell cycle phases. It could prevent the cell from progressing its cycle, leading to the cessation of proliferation. Then, through its natural mechanism, the immune system attacks and kills the cancerous cell. By using this opportunity, the decline of normal cells can be reduced.

Chemotherapy treatment has shown to be effective in treating disseminated metastatic cancer and is widely used. Unfortunately, medications used in cancer chemotherapy suppress both harmless and cancerous cells. It is preferable to eliminate as many cancerous cells as possible while preserving the maximum number of normal cells available. One approach to achieving this aim is to benefit from the point that numerous chemotherapeutic drugs are period-dependent, meaning that they destroy cells only at specific points of the cell's life cycle. The cancerous cells are initially matched by one drug in the cell synchronisation process. When almost all cancerous cells have progressed to the desired phase, they are treated with a second, cycle-specific compound. This destroys most cancer cells while saving a significant amount of normal cells. Since the cell is prevented from progressing throughout the cell cycle, the drugs interrupt cell proliferation and enable the immune system to target and destroy cancerous cells naturally [10].

The issue of modelling tumour growth is being studied extensively by scholars, concentrating on a unique aspect of cancer progression [11]. This encompasses the significance of immune systems in tumour combat, as reviewed by Adam & Bellomo [12]. Using ordinary differential equations, Kuznetsov et al., (1994) suggested a cytotoxic T lymphocyte (CTL) model in a tumour cell population. They found that CTL and tumour cells engaged in a "predator-prey" relationship, in conjunction with CTL playing the predator role and tumour cells playing the prey role. Adam & Bellomo (1997) created a solid tumour cells population model, along with a reactive lymphocyte, which enabled them to discover the correlation between a triggered immune response and tumour survival rate. Recently, tumour growth models, which deviate from multi-state models and represent a promising solution, have been suggested. Researchers suggested using a hybrid pattern used in tumour development and symptomatic tumour detection capability in the non-existence of examination results [13]–[15]. Plevritis et al. (2007) expanded this to encompass tumour amount and lymph node and distant metastases. A continuous tumour development model was suggested in conjunction with an uninterrupted checking sensitivity corresponding to a tumour's size [17]. Recent researchers perform a continual tumour progression model for testing information that explains three ongoing procedures: tumour development, period to symptomatic diagnosis, and diagnosis sensitivity [18]. Based on their model, we identify methods for measuring lead time, contributing new ideas into duration preconception and analysing impacts on survival. In addition to these breakthroughs, several advancements in scientific and technological approaches have occurred to reflect the aspects of cancer. Another encouraging technique entails mathematical modelling [19], [20], which entails distinguishing the cells that contribute to cancer propagation, describing the relationships among these bodies, estimating parameters, doing stability analyses, and predicting tumour dynamics. Researchers and engineers have tried to grasp better how the tumour spreads because it proliferates. Typically, research into such treatments on tumour development models yields one or more ODEs, which provide insight into the relationship among such calculations and the development of cancer cells. Researchers developed a basic model of tumour progression and therapy focusing upon a specific nonlinear ODE to the exponential expansion formula to provide computational equations in tumour growth [21]. Although numerous immune models have been established with ordinary differential equations (ODEs), robust immune modelling for cancer that includes both immune cells and cytokines has yet to be formed from a biology technological standpoint [22].

This paper will be focussing on three different aspects. First, we will examine a steady-state solution to predict the oscillatory behaviour of the tumour system during interphase and metaphase in the human body. In order to obtain the