Journal of Advances in Science and Technology
Vol. 22, Issue No. 2, April-2025, ISSN 2230-9659

DOI: https://doi.org/10.29070/t02fsb58

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An Analysis On Cancer Data Using Mathematical Modelling

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Abstract: There has been an ongoing push to employ data-driven methods to better understand, diagnose, and treat cancer as it is still one of the top killers globally. In order to improve forecast accuracy and find meaningful patterns in cancer data, this research offers a mathematical modelling approach. A thorough dataset was created for analysis by collecting clinical data from hospital visits and supplementing it with secondary sources. Following data preprocessing, a model reflecting the illness's course and features was developed using relevant mathematical methods. Subsequently, the model's prediction power and applicability to actual cancer dynamics were evaluated by validation. The results show that mathematical modelling may be a useful tool for cancer research, providing information that can help with policymaking and clinical decision-making.

Keywords: Cancer Data, Mathematical Modeling, Predictive Analysis, Biomedical Data, Clinical Modelling, Disease Progression, Quantitative Analysis, Healthcare Research

INTRODUCTION

Cancer was the second-leading cause of death globally in 2018, with an estimated 9.6 million casualties [1]. Furthermore, there is evidence that the decline of the global biosphere is associated with a rise in cancer rates. More than twice as many people will be diagnosed with cancer by 2030, reaching 23.6 million [2]. Follicle carcinoma is one example of a complicated abnormality. Multiple bodily tissues are impacted by the illness in the vast majority of cancer cases. Before transforming into cancerous cells, the normal cells go through a series of transformations. Some examples of these alterations include epigenetic modifications brought about by physical and chemical carcinogens as well as biological illnesses, and genetic variances.

The human genome consists of around three billion base pairs. Only about 20% of human DNA really codes for proteins; the other 80% codes for pseudogenes, transposons, and retrotransposons [3, 4]. Many distinct kinds of microRNAs are also encoded by DNA. Accelerating the complete knowledge of gene expression patterns, researchers started monitoring transcriptomes on a genome-wide scale using microarray and RNA-sequencing data. Processing and analysis may now make use of massive amounts of high-throughput data. One of the most significant and difficult problems that doctors face is making accurate predictions and classifications [5]. Meanwhile, researchers have a huge ally in the rapidly improving state of computer technology, which helps them tackle the challenge of handling enormous data sets. Thanks to this, researchers are able to evaluate massive datasets with the help of automated computer models. To aid in the acquisition of further cancer-related knowledge, computational methods have been devised to manage such complicated mathematical issues. Researchers can't function without data mining

and ML techniques [6, 7].

Applying these methods to complicated datasets allows for the prediction of future cancer survival, recurrence, and susceptibility, as well as the diagnosis and prognosis of various cancers [8, 9, 10].

There is ongoing debate about how to most effectively use these assets and data to identify the true gene regulatory network. This is due to the fact that several models exhibit unique traits and excel when presented with diverse forms of data. Many popular machine learning algorithms and techniques were introduced in this introduction, including ANNs, DTs, SVMs, and naive Bayes. Additionally, we presented a number of well-liked approaches to constructing networks of gene regulation. Some examples of these models include regression and correlation as well as native and dynamic Bayesian methods. As a further step, we provide a number of cases for you to evaluate the analysis and precision. In order to build more accurate gene regulatory networks, various strategies have made use of previously collected data.

One way to quantitatively describe a system's non-linear dynamical development is using ordinary differential equations (ODEs). Differential equations that include additional noise components to represent random fluctuations are called stochastic differential equations (SDEs). Specific differential equations (SDEs), often called "Langevin" equations [11,12], are used to characterise dynamical systems. One way of looking about SDEs is as a way to apply dynamic system theory to situations where there is noise or fluctuations. This is a significant generalisation since actual systems are never totally isolated from their surroundings and are subject to constant external influences; hence, ODEs and SDEs find extensive use in molecular dynamics. In addition, this study demonstrates the course of cancer by providing analytical tools based on gene regulatory networks (ODE and SDE). To hone down on the dynamics of the gene regulatory network, we will specifically apply a landscape and flux theory to the cancer field. Data mining and mathematical models may help in cancer prediction and diagnosis, which might lead to better treatment options.

METHODOLOGY

This study follows a systematic approach to develop a mathematical model based on biological data related to a specific illness. The methodology encompasses data collection, preparation, modeling, and validation.

The process began with visits to hospitals to gather relevant clinical data and obtain a conceptual understanding of the targeted illness. This involved direct interactions with medical professionals and a comprehensive review of existing literature, including current treatment methods and recent advancements related to the illness.

Following data collection, the raw information was organized and formatted to suit the requirements of mathematical analysis. This included data cleaning, classification, and identifying key parameters and variables necessary for modeling.

Subsequently, appropriate mathematical techniques were applied to analyze the data and construct a model representing the biological system under study. The mathematical tools and methods were selected based on the nature of the data and the objectives of the study, ensuring robust and meaningful results.

Finally, the formulated model was verified for accuracy and reliability. This involved testing the model

against known datasets and comparing its performance with alternative models to evaluate its suitability and potential for broader application in similar biological contexts.

RESULT AND DISCUSSION

All female cancer patients reported to Saudi Arabia between 2004 and 2016 are included in this study. In data fitting, the year is the unit of time that is considered. Specifically, we examined the data fitting using the least-square curve-fitting method. X(0)=30,000, B(0)=12,300, and $a(0)$=783 are the model variables. The values of C(0)=783, $R(0)$=334R(0)=334 and $E(0)$=10E(0)=10 are considered. Using these values, we run the model simulation and get the required numerical results (Figures 1 and 2). These simulations were fitted to the cancer data, and the parameter values are shown in Table 2. The data fitting in contrast to the model simulations for patients with stage 4 cancer is shown in Figure 1. In order to fit the model correctly and predict future occurrences in the country, the data may be used. A long-term prediction of the cases vs. model is shown in Figure 2. Long-term behaviour fits the data better and shows that the disease will be around for a long time unless the government does anything to lower the cancer rate.

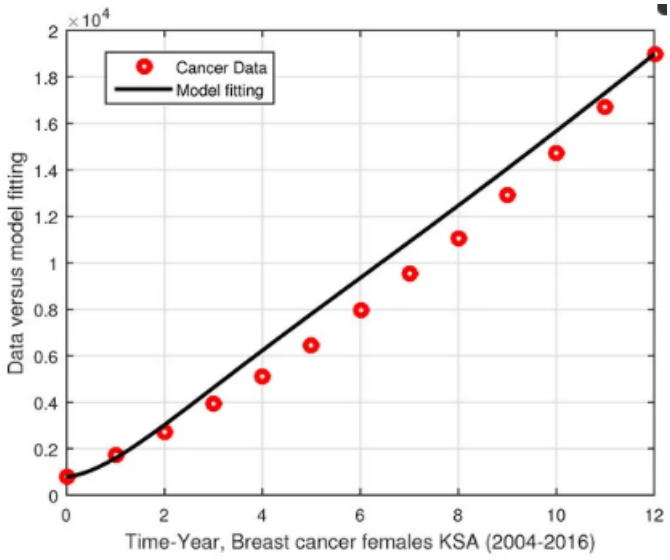

Figure 1: Saudi Arabian female cancer patient data from 2004 to 2016 in comparison to model fitting.

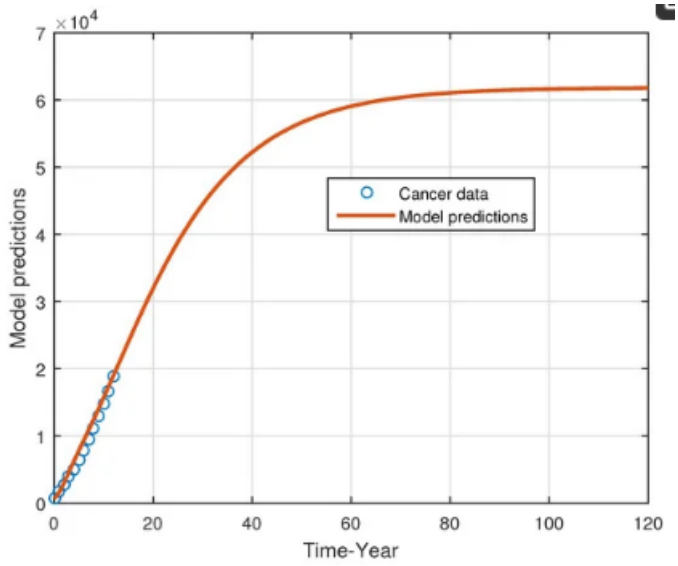

Figure 2: Saudi Arabian female cancer patient data from 2004 to 2016 in comparison to model predictions. 120 units are regarded as the time level.

Table 1: Parameters Details

Symbol	Value	Ref.	Symbol	Value	Ref.
Λ1Λ1	14,000	Estimated	Λ2Λ2	80	Estimated
Λ3Λ3	90	Fitted	β1β1	0.01	Fitted
β2β2	0.034	Fitted	ψ1ψ1	0.03	Fitted
ψ2ψ2	0.3	Fitted	τ1τ1	0.03	Fitted
τ2τ2	0.1	Fitted	τ3τ3	0.2	Fitted
μ1μ1	0.0256	[**26**]	μ2μ2	0.0256	[**26**]
μ3μ3	0.0256	[**26**]	ϕ1ϕ1	0.03	Fitted
ϕ2ϕ2	0.4	Fitted	ϕ3ϕ3	0.01	Fitted

Their graphical result is derived while considering the model compartments' simulation. As seen in Figure 3, the model compartments' dynamics are shown over a long time period (t=120). It is evident that the number of people with breast cancer would rise if there is currently no way to prevent or treat cancer patients, which would be a concerning condition for the nation.

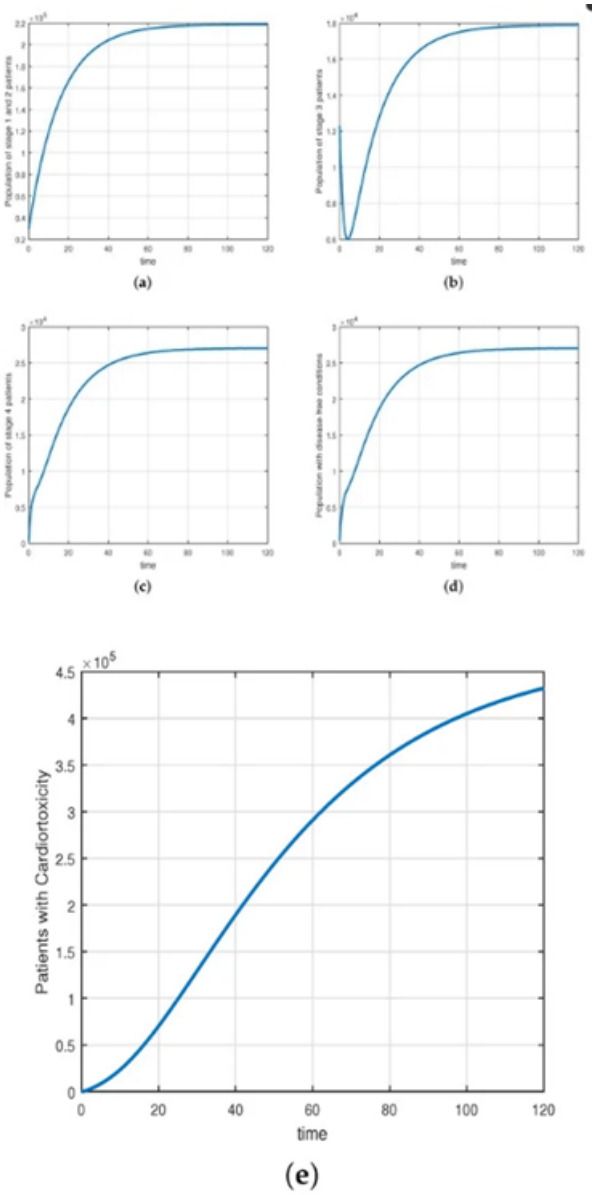

Figure 3: The model compartment simulations are described in the figure. The population of stage 1 and 2, stage 3, stage 4, disease-free, and cardiotoxic is shown in Figure (a–e).

Class E will see a little increase in patients, whereas classes B, C, and R would see a decrease in patient numbers as time goes on, as seen in Figure 4. As seen in Figure 4, vigorous chemotherapy benefits populations in stages 3 and 4 and those without illness, whereas it harms people with cardiotoxicity. This is shown by the parameter $\tau 11$. Recent years have seen remarkable progress in cancer therapy, which has been shown to enhance the chances of curing breast cancer and preventing its recurrence. Cardiotoxicity greatly raises mortality and morbidity rates[28,29], making it one of the most significant side effects of chemotherapy. Figure 4d indicates a little increase because of this.

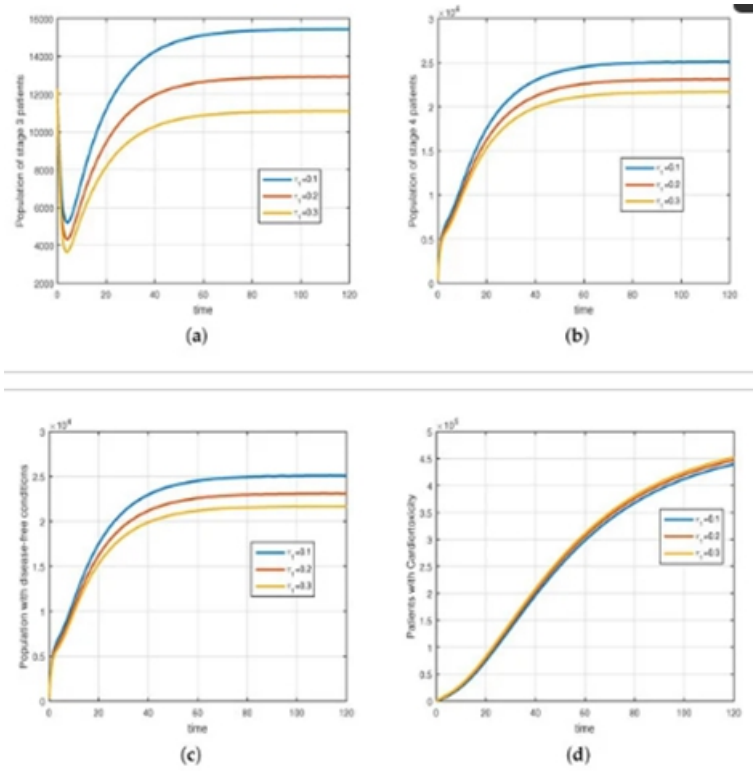

Figure 4: The model compartment simulations for τ1=0.1,0.2,0.3τ1=0.1,0.2,0.3 are shown in the figure. Stage 3, stage 4, disease-free, and cardiotoxic individuals are described in Figure (a–d).

The effects of intense chemotherapy on patients at stage 4, which might raise morbidity and death rates among those with cardiotoxicity, are shown in Figure 5. By increasing the chemotherapy treatment for patients in stages 3 and 4, those without disease, and those with cardiotoxicity, we can see that patients in stage 3 show little improvement (see Figure 5a), while patients in stage 4 and those without disease show good improvement in disease reduction (see Figure 5b,c). However, in the case of cardiotoxicity, there is no discernible improvement. This is because chemotherapy negatively affects patients and raises the risk of heart-related problems.

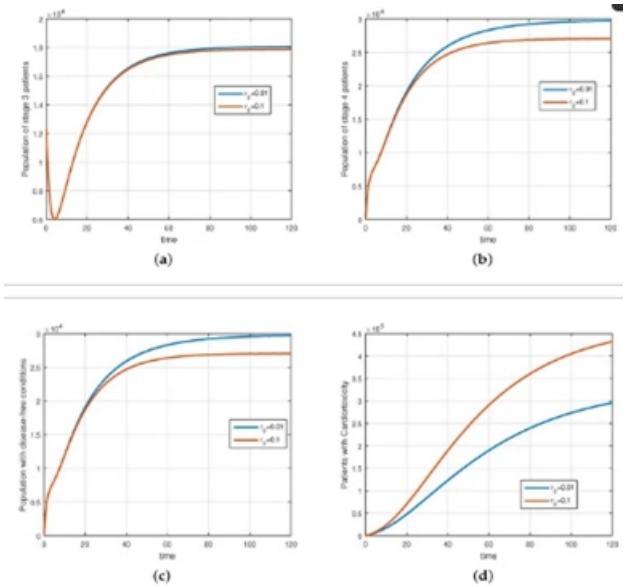

Figure 5: The model compartment simulations for $\tau 2$=0.01,0.1$\tau 2$=0.01,0.1 are shown in the figure. Stage 3, stage 4, disease-free, and cardiotoxic individuals are described in Figure (a–d).

The model compartment simulation during aggressive chemotherapy in the disease-free scenario is shown in Figure 6. The findings show that the population of cardiotoxicity has somewhat increased, but the populations of B, C, and R have somewhat decreased. For instance, the results in Figure 6 indicate that the number of cases in stage 3 patients has not improved much; in contrast, Figure 6b shows a good drop in instances in stage 4 patients. Figure 6c shows a decent decline in the number of patients in the disease-free population, however Figure 6d shows a little rise in the number of cardiotoxicity cases.

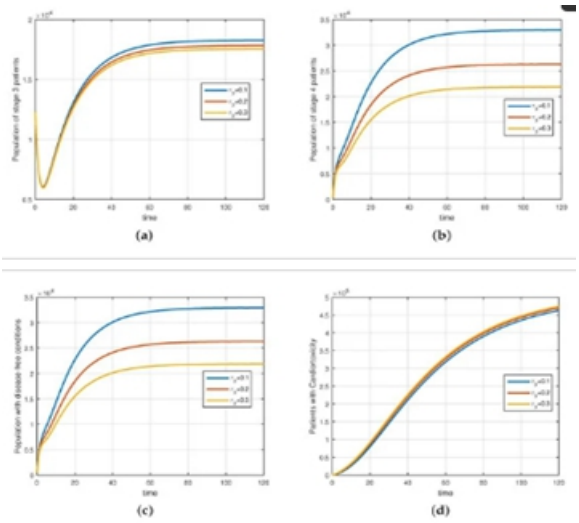

Figure 6: The model compartment simulations for $\tau 3$=0.1,0.2,0.3$\tau 3$=0.1,0.2,0.3 are shown in the figure. Stage 3, stage 4, disease-free, and cardiotoxic individuals are described in Figure (a–d).

The modeling of cardiotoxicity in persons with different treatment settings for stages 3 and 4 is shown in Figure 7. Figure 7 shows that when the amount of chemotherapy is reduced, fewer people are experiencing cardiotoxicity.

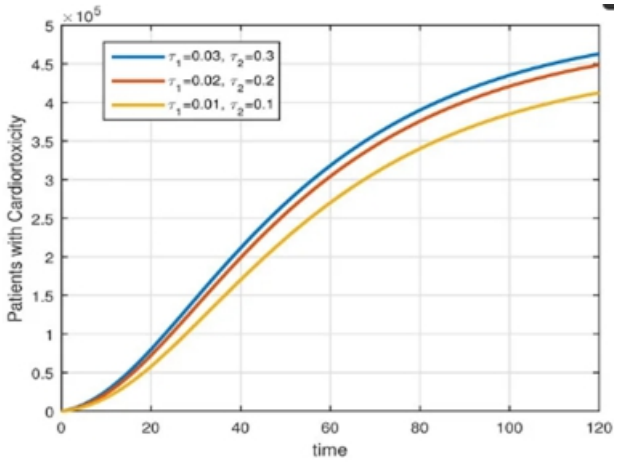

Figure 7: The plot describes the simulations of the cardiotoxicity with the impact of parameters $\tau 1$ = 0.03, 0.02, 0.01 $\tau 1$ = 0.03, 0.02, 0.01, and $\tau 2$ = 0.3, 0.2, 0.1 $\tau 2$ = 0.3, 0.2, 0.1.

Chemotherapy often raises the risk of cardiotoxicity; nevertheless, certain investigations have shown that even in people without a prior history of cardiovascular disease, the overall quantity given may cause heart failure symptoms. Because of this discovery, fewer people are undergoing chemotherapy, which has reduced its efficacy. In order to determine the optimal course of action, cardiologists and oncologists should evaluate the patient's cardiovascular risk prior to beginning cancer treatment. As a result, cardiotoxicity may be lessened.

CONCLUSION

Mathematical modeling is not just a tool but a transformative force in the battle against cancer. With continued research, development, and application, we can achieve earlier detection, more accurate diagnoses, and personalized treatments, ultimately saving lives and improving outcomes for patients worldwide.

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