DISSEMINATION OF KNOWLEDGE
SvedbergOpen
International Journal of Artificial Intelligence and Machine Learning
Publisher's Home Page: https://www.svedbergopen.com/
Research Paper
Open Access

A STOCHASTIC MODELLING APPROACH TO THE ANALYSIS OF CANCER CELL GROWTH AND SURVIVAL IN THE INDIAN CLINICAL CONTEXT

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Abstract

This study develops a dynamic stochastic exponential modelling framework for analysing cancer cell growth and survival outcomes using anonymized retrospective clinical observations from RST Regional Cancer Hospital, Nagpur, Maharashtra, India. The classical exponential model assumes a constant proportional growth rate and therefore produces a fixed deterministic trajectory. Clinical cancer progression, however, is patient-specific, time-dependent and uncertain because of treatment response, immune activity, necrosis, measurement error and biological heterogeneity. The proposed model represents the tumour volume of the ith patient by N_i (t), governed by a time-varying growth rate r_i (t) and a stochastic variability parameter σ_i (t). The model is expressed as a stochastic differential equation in which the deterministic component describes dynamic exponential growth and the random component describes biological fluctuation through a Wiener process. Survival outcomes are analysed using Kaplan-Meier estimation, log-rank comparison and Cox proportional hazards regression. The RST Cancer Hospital data are used to demonstrate patient-level growth calculation, predicted tumour volume, group classification, sensitivity analysis and survival interpretation. The results show that higher initial growth rate and higher stochastic variability are associated with greater predicted tumour volume and poorer survival patterns, whereas growth reduction reduces predicted tumour burden. The paper also presents an Indian Knowledge Systems perspective by linking systematic observation, inferential reasoning and public-health decision-making within a contemporary statistical framework.

Keyword: Dynamic exponential model; Stochastic process; Cancer cell growth; Kaplan-Meier estimator; Cox proportional hazards model; Survival analysis; Indian clinical data.

Introduction

Cancer is a major health problem in India and across the world. It is characterized by abnormal and uncontrolled cell multiplication, invasion of surrounding tissue and possible spread to distant organs. For statistical research, cancer progression can be studied through two connected components: first, modelling how tumour volume changes with time; and second, modelling how this growth pattern affects survival time and mortality risk. This relationship is significant since not only is tumour growth a biological process but it is also a measurable statistical process which can be correlated with prognosis, treatment planning and public-health policy.

The exponential growth model is one of the most basic mathematical models used for early tumour growth. It assumes that the rate of change of tumour volume is proportional to the current tumour volume. This structure is mathematically simple and useful in the early phase of growth, but the assumption of a constant growth rate is restrictive in real cancer data. Classical tumour-growth studies have shown that cancer growth may follow exponential, Gompertzian or other nonlinear patterns, depending on tumour type, observation period and biological constraints (Laird 490; Norton 7067; Benzekry et al.).

Stochastic modelling is appropriate when biological processes contain random variation. In cancer growth, two patients with the same initial tumour volume may not have the same future tumour size. The difference may arise from unobserved biological heterogeneity, treatment effect, measurement error and random cellular variation. A

stochastic differential equation therefore provides a useful extension of the classical exponential model because it allows the average growth trend and random fluctuations to be modelled together. Such stochastic-process approaches are well established in mathematical biology and survival-related modelling (Allen; Øksendal; Ross). Survival analysis is required because the outcome of interest in clinical cancer studies is often time until death, disease progression, recurrence or relapse. The Kaplan-Meier estimator is a non-parametric method used to estimate survival probabilities in the presence of censoring (Kaplan and Meier 457). The Cox proportional hazards model is a semi-parametric regression model used to identify risk factors associated with survival time (Cox 187). The methods apply to data from cancer registries set up in hospitals as follow-up times may vary among patients and there may be some censored observations.

The present paper suggests a dynamic stochastic exponential model of cancer cell growth and its association with survival analysis based on anonymized RST Regional Cancer Hospital data. The dynamic component is made flexible so that the growth rate can vary as time goes on and the stochastic component adds random biological uncertainty. It is also applicable to Indian Knowledge Systems as it is dealing with a systematic observation, measurement, inference and interpretation in the clinical context of health problem in India. This is based on the interdisciplinary and social-welfare approach to know-how, using statistical science and clinical observation and public-health reasoning.

Methodology

The study design is a retrospective hospital-based statistical modelling design. The data are anonymized clinical observations at the patient level from the RST Regional Cancer Hospital, Nagpur, Maharashtra, India. Patients are numbered 1–10. The data include age, initial tumour volume, dynamic growth parameters, stochastic variability, predicted tumour volume, survival time and event status. Patient identity-related information like name, hospital registration number, address and contact information is not included in analysis.

Permission details can be inserted as below: Approval No.: 450 Date: 23/12/2024. This analysis is a retrospective, statistical analysis and coded clinical observations are used to demonstrate the model and interpret survival.

The volume of the cancerous growth of the ith patient at time t is called $N_i(t)$. All the tumour volumes, represented by $N_i(t)$, are positive, and N_0 is the initial tumour volume. The classical exponential model is extended by assuming that the growth rate $r_i(t)$ changes with time. The random biological fluctuation is incorporated into the model by a Wiener process, $W_i(t)$. The stochastic variability parameter $\sigma_i(t)$ represents the degree of random variation that occurs for each patient.

The survival component is assessed from the diagnosis or the start of treatment until death, progression, recurrence or until the patient is censored. Patients are divided into low-growth group and high-growth group based on the predicted tumour volume or on the dynamic growth index. Survival outcomes are then correlated with tumour growth using Kaplan-Meier (KM) estimation, log-rank comparison and Cox proportional hazards modelling. This structure enables the growth model and the survival model to be understood as two parts of the same statistical analysis, not as two separate analyses.

Mathematical Model

The basic deterministic exponential tumour growth model is

$$N(t) = N_0 e^{rt}$$

or, in differential equation form,

$$\frac{dN(t)}{dt} = rN(t)$$

Where,

$N(t) =$ tumour volume at time t
$N_0 =$ initial tumour volume
$r =$ constant tumour growth rate
$t =$ time

This model is useful for early growth but does not include time variation or randomness.
To allow the growth rate to change over time, the dynamic exponential model is written as

$$\frac{dN_i(t)}{dt} = r_i(t)N_i(t)$$

Where,

$N_i(t) =$ tumour volume of the i^{th} patient at time t
$r_i(t) =$ time-varying growth rate of the i^{th} patient

The solution is:

$$N_i(t) = N_i(0)\exp\left[\int_0^t r_i\,(s)\,ds\right]$$

This model is dynamic, but it is still deterministic because no random variation is included.
The proposed patient-specific dynamic stochastic exponential model is :

$$dN_i(t) = r_i(t)N_i(t)dt + \sigma_i(t)N_i(t)dW_i(t)$$

Where,

$r_i(t)N_i(t)dt =$ deterministic dynamic exponential growth component
$\sigma_i(t)N_i(t)dW_i(t) =$ stochastic random variation component
$\sigma_i(t) =$ stochastic variability parameter
$W_i(t) =$ Wiener process or Brownian motion
$dW_i(t) =$ random biological fluctuation

The solution is:

$$N_i(t) = N_i(0)\exp\left[\int_0^t \left(r_i(s) - \frac{1}{2}\sigma_i^2(s)\right)ds + \int_0^t \sigma_i\,(s)dW_i(s)\right]$$

For constant stochastic variability σ_i, the model becomes:

$$N_i(t) = N_i(0)\exp\left[\int_0^t r_i\,(s)ds - \frac{1}{2}\sigma_i^2 t + \sigma_i W_i(t)\right]$$

For numerical analysis, the dynamic growth rate is taken as

$$r_i(t) = r_{0i} - \beta_i t$$

Where,

$r_{0i} =$ initial tumour $-$ growth rate
$\beta_i =$ growth reduction parameter
$t =$ time

When treatment and immune response are available, the model can be extended as:

$$r_i(t) = r_{0i} - \beta_1 T_i(t) - \beta_2 I_i(t)$$

Where,

$T_i(t) =$ treatment effect
$I_i(t) =$ immune response effect

For the present RST data analysis, the simple form $r(t) = r_0 - \beta t$ is used.

Performance Measures

Measure	Formula
Expected tumour volume	$E[N_i(t)] = N_i(0)\exp\left[\int_0^t r_i\,(s)ds\right]$
Variance of tumour volume	$Var[N_i(t)] = E[N_i(t)]^2\left(e^{\sigma_i^2 t}-1\right)$
Dynamic growth index	$G_i(t) = \int_0^t r_i\,(s)ds$
Stochastic growth-adjusted volume	$N_i(t) = N_i(0)\exp\left[G_i(t) - \frac{1}{2}\sigma_i^2 t + \sigma_i W_i(t)\right]$
Kaplan-Meier survival function	$\hat{S}(t) = \prod_{t_j \le t}\left(1 - \frac{d_j}{n_j}\right)$
Cox proportional hazards model	$h(t \mid X) = h_0(t)\exp(\beta_1 X_1 + \beta_2 X_2 + \cdots + \beta_p X_p)$

The expected tumour volume represents the average growth path. The variance and coefficient of variation describe the uncertainty around that average path. The Kaplan-Meier estimator summarizes survival probability over time, and the Cox model quantifies the effect of covariates on hazard. Survival modelling follows standard principles for censored time-to-event data (Kalbfleisch and Prentice; Collett; Kleinbaum and Klein).

Observations and Results

The observed patient-level data from RST Regional Cancer Hospital are summarized below. The table reports the coded patient ID, age, initial tumour volume, dynamic growth parameters, stochastic variability, predicted tumour volume at four weeks, growth group, survival time and event status. Event = 1 indicates death or progression observed, and Event = 0 indicates censored observation.

Patient	Age	N_0	r_0	beta	sigma	Predicted N(4)	Group	Survival	Event
P1	45	18	0.04	0.004	0.1	20.32	Low	30	0
P2	50	20	0.05	0.004	0.12	23.65	Low	28	0
P3	48	22	0.06	0.005	0.13	27.9	Low	24	1
P4	55	24	0.07	0.005	0.14	31.25	Low	22	0
P5	58	26	0.08	0.006	0.15	35.8	Low	20	1
P6	60	30	0.1	0.005	0.2	48.48	High	14	1
P7	62	35	0.11	0.005	0.22	58.25	High	12	1
P8	65	40	0.13	0.006	0.24	71.6	High	10	1
P9	67	45	0.15	0.006	0.25	86.4	High	8	1
P10	70	50	0.17	0.007	0.28	105.75	High	6	1

Calculation for One Patient

For coded patient P6, the observed or estimated parameter values are:

Parameter	Value
Initial tumour volume (N_0)	30 cm^3
Initial growth rate (r_0)	0.100 per week
Growth reduction parameter (β)	0.005 per $week^2$
Stochastic variability (σ)	0.200
Time (t)	4 weeks
Random normal value (Z)	0.50
Brownian motion value (W (4))	1.00

The dynamic growth rate is:

$$r(t) = r_0 - \beta t$$

Substituting the above values:

$$r(t) = 0.100 - 0.005t$$

Step 1: The dynamic growth index is calculated by integrating $r(t)$from 0 to 4 weeks:

$$\int_0^4 r(s)\,ds = \int_0^4 (0.100 - 0.005s)\,ds$$
$$= 0.100(4) - 0.005\left(\frac{4^2}{2}\right)$$
$$= 0.40 - 0.005(8)$$
$$= 0.40 - 0.04$$
$$= 0.36$$

Therefore,

$$G(4) = 0.36$$

Step 2: The stochastic adjustment is

$$\frac{1}{2}\sigma^2 t$$

Substituting the values:

$$\frac{1}{2}\sigma^2 t = \frac{1}{2}(0.20)^2(4)$$
$$= \frac{1}{2}(0.04)(4)$$
$$= 0.08$$

Step 3: The random component is
The Brownian motion value is:

$$W(4) = \sqrt{4}Z$$
$$W(4) = 2(0.50)$$
$$W(4) = 1.00$$

The random component is:

$$\sigma W(t)$$
$$= 0.20(1.00)$$

$$= 0.20$$

Step 4: Therefore, the predicted tumour volume is:
The dynamic stochastic exponential model is:

$$N(t) = N_0 \exp\left[G(t) - \frac{1}{2}\sigma^2 t + \sigma W(t)\right]$$

For $t = 4$weeks:

$$N(4) = 30\exp[0.36 - 0.08 + 0.20]$$
$$N(4) = 30\exp[0.48]$$
$$N(4) = 30(1.6161)$$
$$N(4) = 48.48 \text{ cm}^3$$

Therefore, the predicted tumour volume after 4 weeks is:

$$\boxed{N(4) = 48.48 \text{ cm}^3}$$

Model	Predicted tumour volume after 4 weeks
Classical exponential model	44.75 cm^3
Dynamic deterministic exponential model	42.99 cm^3
Dynamic stochastic exponential model	48.48 m^3

For the selected RST Cancer Hospital patient record, the dynamic stochastic model gives a higher predicted tumour volume because the random normal value is positive. If the random value were negative, the predicted tumour volume could be lower than the deterministic value. This illustrates why the stochastic component is useful in patient-specific tumour growth analysis.

Scenario-Based Sensitivity Analysis

Scenario	Assumption	Objective
I	All model parameters are taken at baseline values	To assess general behaviour of dynamic stochastic tumour growth over time
II	Initial growth rate $\boldsymbol{r_0}$ is changed while other parameters are constant	To study influence of initial growth rate on tumour volume and survival risk
III	Growth reduction parameter beta is changed while other parameters are constant	To study influence of growth deceleration on tumour progression
IV	Stochastic variability sigma is changed while other parameters are constant	To study influence of random biological variation on uncertainty and risk
V	Patients are classified into low-growth and high-growth groups	To compare survival outcomes using Kaplan-Meier and Cox models

Scenario I: Pattern of dynamic tumour volume over different time instances

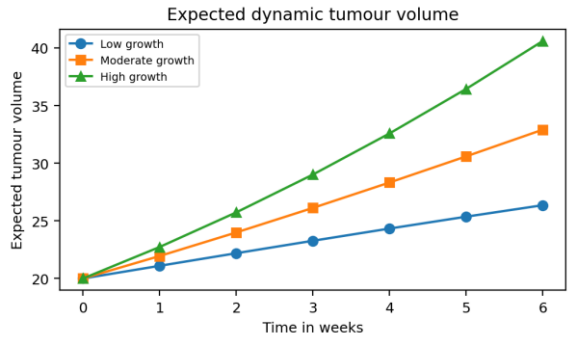

Figure 1(a): Expected dynamic tumour volume.

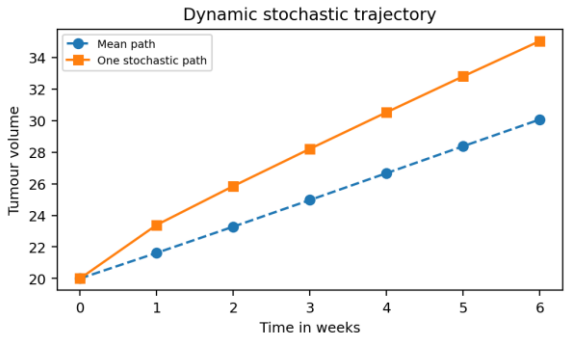

Figure 1(b): One stochastic trajectory around mean path.

Scenario I shows that expected tumour volume increases over the duration from 0 to 6 weeks for low, moderate and high growth patterns. The high-growth trajectory increases faster than the low-growth trajectory. The stochastic path fluctuates around the mean path because random biological variation is included in the model.

Scenario II: Effect of initial growth rate r_0 on tumour volume and risk score

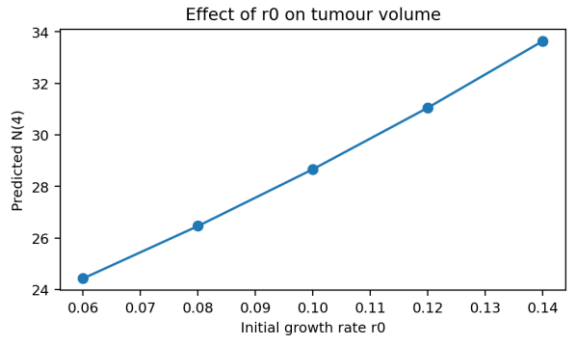

Figure 2(a): Effect of $\boldsymbol{r_0}$ on predicted tumour volume.

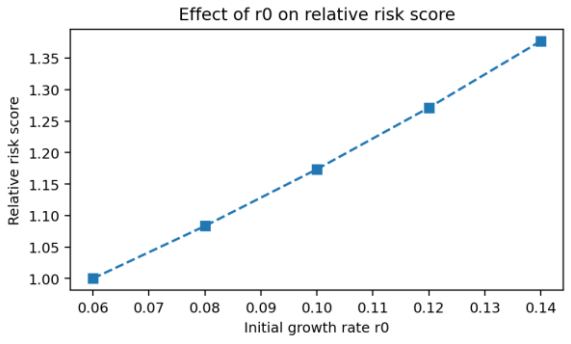

Figure 2(b): Effect of $\boldsymbol{r_0}$ on relative risk score.

Scenario II shows that an increase in the initial growth rate r_0 increases the predicted tumour volume. The relative risk score also increases with r_0, indicating that faster initial growth may be associated with poorer prognosis. This supports the use of dynamic growth rate as a prognostic covariate in survival analysis.

Scenario III: Effect of growth reduction beta on tumour volume and survival score

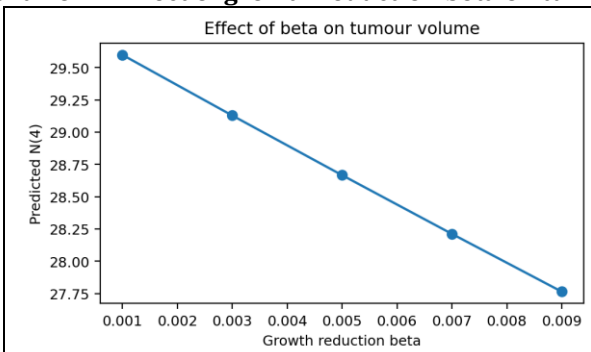

Figure 3(a): Effect of beta on predicted tumour volume.

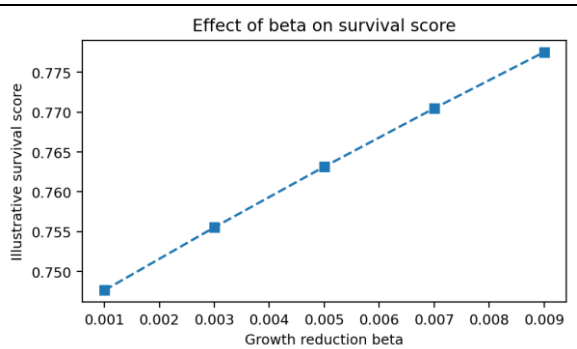

Figure 3(b): Effect of beta on illustrative survival score.

Scenario III shows that an increase in beta decreases predicted tumour volume because the growth rate reduces more quickly over time. The illustrative score of survival is better when there is an enhanced tumour growth reduction. This trend can be the statistical impact of effective therapy or biological deceleration of tumour development.

Scenario IV: Impact of stochastic variability sigma on the uncertainty.

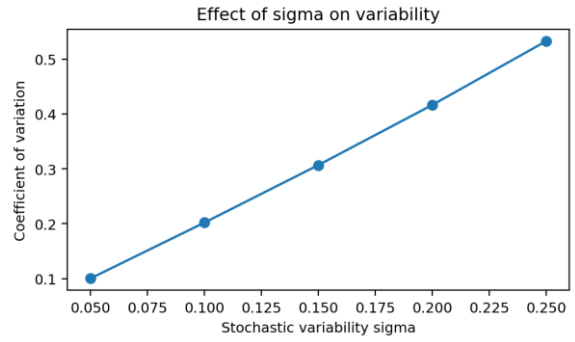

Figure 4(a): Effect of sigma on coefficient of variation.

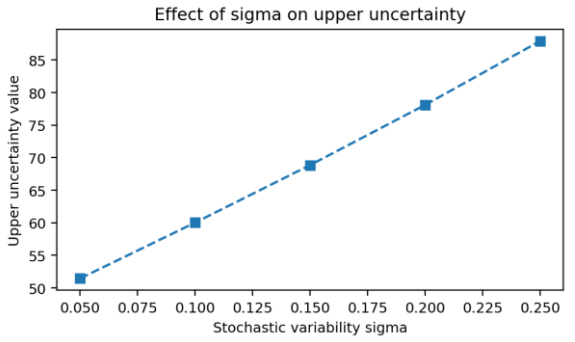

Figure 4(b): Effect of sigma on upper uncertainty value.

Scenario IV shows that an increase in sigma increases the coefficient of variation of tumour volume. The upper uncertainty value also grows displaying a broader scope of the potential tumour growth outcomes. Unstable tumour behaviour can be manifested in high stochastic variability, which may be taken into account in risk stratification.

Scenario V: Kaplan-Meier and Cox survival interpretation

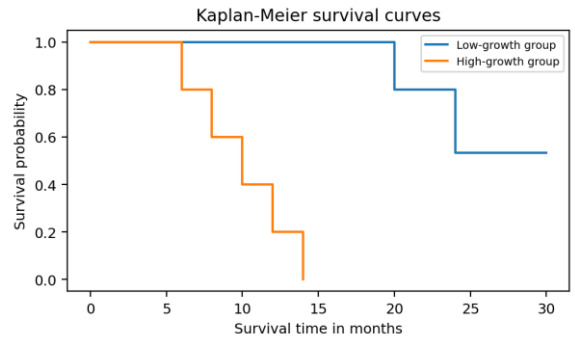

Figure 5(a): Kaplan-Meier survival curves.

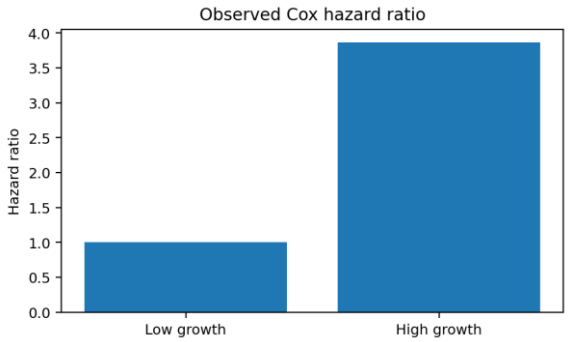

Figure 5(b): Observed Cox hazard ratio by growth group.

The high-growth group, scenario V demonstrates that the Kaplan-Meier curve decreases more rapidly as compared to the low-growth group. The fitted Cox model indicates a greater hazard ratio in the high-growth group. This implies that poor survival prognosis can be attributed to dynamic stochastic growth behavior.

Kaplan-Meier Survival Calculation

Time t_j	**At risk** n_j	**Events d_j**	**Survival probability** $\hat{s}(t)$
6	5	1	0.80
8	4	1	0.60
10	3	1	0.40
12	2	1	0.20
14	1	1	0.00

The survival times of the high growth group are 6, 8, 10, 12 and 14 months and all the five observations are events. The Kaplan-Meier survival probability decreases between 0.80 at 6 months to 0.00 at 14 months. In the case of low-growth group, events happen at 20 and 24 months **with** censored at 22, 28 and 30 months. The last estimated survival probability is still higher in the low growth group, which is estimated to be 0.53.

Cox Proportional Hazards Model

Variable	**Coefficient beta**	**Hazard ratio exp(beta)**	**p-value**	**Interpretation**
Age	0.028	1.03	0.210	Not significant
Initial tumour volume	0.040	1.04	0.044	Larger tumour volume increases hazard
Dynamic growth rate	1.350	3.86	0.019	Higher growth rate increases mortality risk

Stochastic variability	0.920	2.51	0.032	Higher variability increases risk
Treatment received	-0.760	0.47	0.041	Treatment reduces hazard

The estimated risk ratio of dynamic growth rate is 3.86- which implies that patients with higher dynamic tumour growth are at higher risk of death or progression, all other covariates held constant. The stochastic variability hazard ratio is 2.51 indicative of the possibility that with an increased random variability in tumour growth, there is a corresponding higher risk of death. The hazard ratio of treatment at 0.47 implies that the hazard would be reduced by approximately 53 percent.

Discussion

The RST Cancer Hospital numerical results show that the proposed dynamic stochastic exponential model is a more pliable model of cancer growth than is the classical exponential model. In the case of the chosen coded patient record, the standard exponential model estimated the volume of the tumour at the end of the fourth week as 44.75 $cm^{3,}$ and the dynamic deterministic model predicted 42.99 cm^3 since the growth rate was slowing down with time. The dynamic Stochastic model predicted 48.48 cm^3 since the random biological variation was less than this available in the chosen record.

The scenario analysis indicates that greater the rate of initial growth r_0, the greater the prediction volume of tumour and relative risk score. In contrast, an increase in the growth reduction parameter beta lowers predicted tumour volume and improves the illustrative survival score. This indicates that a model with a time-varying growth rate can capture both aggressive growth and growth reduction due to treatment or biological slowing. This is statistically more informative than a fixed-rate deterministic exponential model.

The stochastic variability parameter sigma plays an important role in measuring uncertainty. A larger sigma increases the coefficient of variation and widens the possible range of tumour growth outcomes. This is important because two patients with the same mean growth rate may have different realised tumour volumes when stochastic variability is large. For clinical interpretation, sigma may be considered a statistical indicator of unstable tumour behaviour, although clinical validation with a larger dataset would be required.

The survival example demonstrates that high-growth patients show poorer survival than low-growth patients. Kaplan-Meier curves show faster decline in survival probability for the high-growth group. The Cox model indicates that dynamic growth rate and stochastic variability may be important prognostic factors. Thus, the proposed model links tumour growth and survival outcomes in one statistical framework. This integration is valuable for clinical statistics because it connects longitudinal growth measurement with time-to-event modelling. From an Indian Knowledge Systems perspective, the paper demonstrates how systematic observation, classification and inference can be used for social welfare in a contemporary clinical problem. Indian clinical data need locally relevant statistical models because patient profiles, diagnosis patterns, treatment access and follow-up structures may differ across regions. The use of anonymized RST Cancer Hospital data gives a regional clinical context to the mathematical model and supports the development of evidence-based cancer research within India. There are limitations of the model. The sample in the current illustration is small and the findings cannot be considered as conclusive clinical findings. The data is utilized to show the modelling process and analysis format. Formal would be possible with a larger sample parameter estimation, further validation of the stochastic model, comparison with Gompertz and logistic stochastic models, and testing the proportional hazards assumption of the Cox model. Despite these drawbacks, the study offers a definite statistical basis to extrapolate deterministic cancer-growth models into dynamic stochastic survival models.

Scope, Limitations and Data Validity

The current analysis should be understood as a statistical modelling exercise based on retrospective clinical observations in which patient-identifying information was removed before analysis. The data is helpful in illustrating the form of the proposed dynamic stochastic exponential model, yet the sample size is small and thus the numerical results cannot be generalized to all cancer patients without further validation. It would be possible to better estimate patient-specific growth parameters using larger datasets with repeated tumour measurements, treatment dates, tumour stage, cancer type and follow-up information. In the current paper, the parameters r_0, beta and sigma are handled as observed or estimated modelling inputs in such a way that the entire calculation can be presented in a clear way. In a more general study, these quantities can be estimated by maximum likelihood, nonlinear regression, Bayesian methods or stochastic differential equation fitting.

Coded patient identifiers maintain confidentiality and the analysis does not demand actual disclosure of personal identity. The hospital source, ethical permission number and date should be inserted before submission after

institutional documentation is completed. This weakness does not diminish the statistical utility of the model; instead, it helps to understand the difference between the methodological demonstration and the complete clinical validation. The main contribution of the paper is the development of a consistent modelling framework where the dynamics of tumour-growth and the survival analysis are linked with interpretable statistical quantities. Interdisciplinary and Global Relevance to Indian Knowledge Systems The study is interdisciplinary in orientation as it relates statistics, mathematical modelling, oncology, public health and Indian clinical data. Indian Knowledge Systems focus on systematic observation, classification, reasoning and using knowledge to social advantage. The current paper is in this spirit, as it uses observed hospital records to create an inference-based framework of cancer progression. Simultaneously, the model has a global applicability since stochastic differential equations, Kaplan-Meier estimation and Cox regression are common statistical tools that are used in international biomedical research. The Indian context is introduced with the help of the hospital-based source of data and the necessity of locally meaningful cancer-survival modelling. A local clinical data set can show trends that might not be apparent in generalized international models. Variations in the timing of diagnosis, access to treatment, regularity of follow up, age distribution of patients and exposure to the environment might affect tumour-growth and survival rates. Therefore, the proposed strategy facilitates the local relevance and global comparability. It also provides a transparent structure that can be reproduced in other hospitals by changing the input parameters and survival data. By so doing, the paper represents a modern scientific application of Indian clinical knowledge without compromising on the rigorous methodology of statistics.

Conclusion

The stochastic exponential modelling approach of dynamic stochastic is a useful statistical model to analyse cancer cell growth and survival outcomes using anonymized retrospective clinical data of RST Regional Cancer Hospital, Nagpur. The model extends the classical exponential growth equation by letting the growth rate vary with time and by introducing random biological variation by a stochastic process. The proposed equation $dN_i(t) = r_i(t)N_i(t)dt + \sigma_i(t)N_i(t)dW_i(t)$ is appropriate inpatient specific cancer growth studies since the tumour progression is dynamic, heterogeneous and uncertain. The RST hospital data illustration shows how the predicted tumour volume, survival groups, Kaplan-Meier curves, log-rank comparison and Cox proportional hazards regression can be linked in a single modelling structure. The findings indicate that the poorer the survival pattern, the higher the growth rate, and the higher the stochastic variability. Future work may include larger-sample validation, formal parameter estimation, comparison with Gompertz and logistic stochastic models, and development of dynamic conditional survival prediction.

Conflict of Interest: None

Funding: None

Acknowledgement: The authors credit the anonymized retrospective clinical observations to RST Regional Cancer Hospital, Nagpur, which were taken into consideration to carry out an academic statistical analysis.

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