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Deterministic and Stochastic Gompertz Prey-Predator Model for the Effect of Thymoquinone on Oral Squamous Carcinoma Cell Lines (HSC-4)

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Abstract: Oral squamous cell carcinoma (OSCC) is one of the most common malignancies affecting the head and neck region. Thymoquinone (TQ), the primary bioactive compound of black seed (*Nigella sativa*), has shown antineoplastic properties in various tumor cell types. However, research on TQ effects in tumor cell models, particularly OSCC, remains at an early stage. TQ, a phytochemical with confirmed anticancer potential, has been shown to inhibit OSCC progression mainly through induction of apoptosis. In this study, a prey–predator stochastic Gompertz model is used to characterize the dynamics of the HSC-4 OSCC cell line in response to TQ treatment. The model is first formulated as an ordinary differential equation (ODE), then extended to a stochastic differential equation (SDE) to capture biological noise arising from intrinsic and extrinsic sources within the cellular environment. The Markov Chain Monte Carlo (MCMC) method is utilized to estimate the model's kinetic parameters, while fourth-order stochastic Runge-Kutta (SRK4) algorithms are applied for numerical simulation. Model outputs are validated against in vitro experimental data from TQ-treated HSC-4 cells. The deterministic framework is analyzed in terms of the positivity of solutions and equilibrium states, including the disease-free equilibrium (DFE), the treatment-failure equilibrium (TFE), and the successful treatment equilibrium (SFE). The root mean-square error (RMSE) is computed to assess the consistency between simulated outcomes and experimental observations. The findings demonstrate the model's capability in capturing and explaining the dynamic behavior of HSC-4 cells under TQ treatment.

Keywords: Deterministic model, stochastic model, thymoquinone, oral cancer.

1. Introduction

The five-year survival rate for oral cancer patients was 69%. The incidence rate of new oral cavity and pharynx cancer was 11.5 per 100,000 men and women annually. The mortality rate stood at 2.6 per 100,000 men and women per year. These figures are age-adjusted, derived from cases reported between 2017 and 2021 and deaths recorded from 2018 to 2022 (Global Cancer Observatory, 2025). The sources and methods used in compiling the Global Cancer Observatory: Cancer Today GLOBOCAN estimates have been documented and described online at the Global Cancer Observatory. According to the National Institutes of Health (NIH), the estimated new cases of oral cavity cancer were 59,660 from 2021 to April 2025, and the estimated deaths were 12,770 for the same period (National Cancer Institute, 2025). Cancer spreads by avoiding immune cell destruction, evading growth suppressors, and dysregulating proliferative signaling. Adjuvant chemotherapy and radiation therapy are administered as needed after surgical removal, which is the cornerstone of the current treatment strategy. Many treatment plans have also been developed using targeted approaches to specific molecular markers, such as breast cancers linked to breast cancer tissues. According to Jallah et al. (2023), treatments that target multiple cancer progression pathways may be a useful adjunct to cancer care. Cancer presents itself in a range of ways, including through irregular proliferative signaling, evasion of growth suppressors, and avoidance of immune cell destruction. Surgical excision, adjuvant chemotherapy, and radiation therapy, where necessary, are the mainstays of the current therapeutic approach. Targeted methods to specific molecular targets, like breast malignancies linked to the Breast Cancer gene (BRCA), have also demonstrated considerable therapeutic success. Multiple oncogenic pathway-targeting treatments could be a valuable addition to cancer therapy. The International Classification of Diseases for Oncology, Third Edition, was used to classify all cancer cases; however, the International Classification of Childhood Cancer was used for carcinomas that occurred in children and young adults (Steliarova-Foucher et al., 2005; Steliarova-Foucher et al., 2017).

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Tumors of the lip, oral cavity, and throat are all included in the phrase "oral cancer." The survival rate for oral cancer is about 80% if the disease is detected early (Steliarova-Foucher et al., 2005). Treatment options for oral cancer include surgery, chemoradiotherapy, and targeted therapy. However, combined therapy strategies have unsatisfactory outcomes in advanced illness because chemoradiotherapy can cause harmful effects on healthy cells, and cancer can recur. Recently, cancer treatment has evolved into targeted therapies that inhibit the apoptotic activity of cancer cells. The use of chemotherapy may be encouraged by an alternative strategy for reducing the growth of cancer. Using plant products, commonly referred to as nutritional supplements, is one strategy for advanced oral cancer care. These naturally occurring compounds are physiologically accessible, have several molecular targets for cancer chemoprevention, are cost-effective, and produce non-toxic effects (Steliarova-Foucher et al., 2005; Steliarova-Foucher et al., 2017). In the context of head and neck cancer, oral squamous cell carcinoma remains a highly challenging disease due to its high recurrence rate and relatively simple metastasis (González-Moles et al., 2022; Cadoni et al., 2017; Kasteb et al., 1990). Determining an effective tumor diagnostic and molecular therapeutic target is therefore essential. Through the suppression of serine/threonine kinase 1 (human) AKT and extracellular signal-regulated kinase signaling pathways, TQ reduces tumor angiogenesis and proliferation (Gali-Muhtasib et al., 2006).

2. Thymoquinone and cancer

For a while, TQ has been verified scientifically through the degradation of thymol with hydrogen peroxide. According to recent studies, black seed (Nigella sativa), also known as "Habbtul-barakah" in Arabic, is the most beneficial plant in Mediterranean and West Asian traditional medicine, and it can be used to target the progression of cancer. According to the Prophet's (PBUH) historical account, this plant has therapeutic potential (Gali-Muhtasib et al., 2006). The primary active compound, TQ, which is a strong anti-carcinogenic antioxidant and anti-mutagenic agent, is associated with the biological activities of Nigella sativa seeds (Tabassum et al., 2022). Due to its invasive properties, the oral cancer cell line Human Tongue Squamous Carcinoma Cell Line HSC-4 has been widely used to study metastatic squamous cell carcinoma (Steliarova-Foucher et al., 2005). For example, using oral cancer HSC-4 cell lines for personalized medical care, a recent study by Tabassum et al. (2024) examined the cytotoxic effects of TQ combined with cisplatin (CDDP). Even at lower doses (IC16.8 μM), they found that enhancing TQ with CDDP improves its carcinogenic cytotoxicity against HSC cell lines while reducing its adverse effects on healthy cells (Tabassum et al., 2024). Additionally, significant side effects of current treatment methods, such as surgery and chemoradiotherapy, remain major causes of treatment dissatisfaction. Local relapse and regional or systemic lesions are often the cause of death due to oral cancer (Alaufi et al., 2017).

The flowering plant black cumin (Nigella sativa) contains a phytochemical called TQ. Its anti-inflammatory, anti-tumor, anti-cancer, anti-neoplastic, anti-bacterial, anti-diabetic, antihypertensive, anti-parasitic, and antioxidant properties have been demonstrated in both in vitro and in vivo studies (Chen et al., 2011). Research on TQ's toxicity to normal cells or animal models remains limited. Despite the scarcity of studies on TQ's use in oral cancer, it has shown promising anticancer effects (Hannan et al., 2021). Using a mathematical model to depict the underlying mechanism can improve both quantitative and qualitative understanding of how HSC-4 responds to TQ. Both quantitative and qualitative knowledge can be applied in biomedical research. Ordinary differential equations are often used to develop mathematical models describing the proliferation of cancer cells under treatment (AbuKhader, 2013). However, cancer cell behavior is influenced by many uncontrollable factors, including stress, patient lifestyle, environmental exposures (such as pollution and radiation), genetic predisposition, immune system variability, and pre-existing chronic conditions. Deterministic models based on ordinary differential equations (ODEs) cannot capture the inherent variability and randomness related to these factors.

To address this, ODEs can be extended to stochastic differential equations (SDEs), which include random fluctuations and noise, providing a more realistic framework for modeling the complex and uncertain dynamics of oral cancer development and progression. To illustrate the behavior of oral cancer cell lines in the presence of TQ, we present a stochastic model in this study. We employ a predator-prey paradigm to illustrate the interaction between HSC-4 cell lines and TQ. Assuming that the HSC-4 growth rate follows the Gompertz law, the predator-prey model is modified by incorporating the Gompertz rule. This results in the development of the deterministic Gompertz predator-prey model. The SDE model is created by perturbing the deterministic model with uncontrollable factors, which are modeled by the Wiener process. Mathematical models of the HSC-4 cancer cell lines take the form of ODEs and SDEs in response to TQ. We then present the dynamics of the system regarding positive solutions and the existence of an equilibrium. The four-stage Stochastic Runge-Kutta method (SRK4) is applied for simulation, and parameter estimation is performed using the Markov Chain Monte Carlo (MCMC) method.

3. Mathematical model

Figure 1 depicts the schematic representation of the relationship between the cancer cell and TQ. Based on Figure 1, when TQ reacts with cancer cells, some cells are destroyed, while others continue to proliferate and form multiple cells. However,

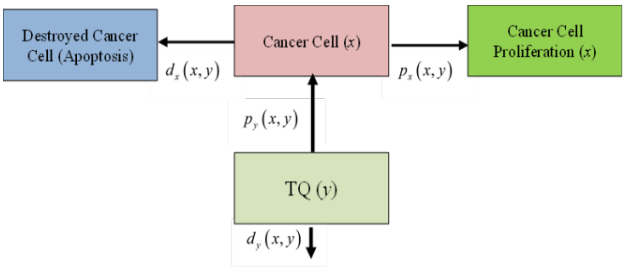


Figure 1. Schematic representation of TQ's relationship with cancerous cells.

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the presence of TQ can slow down the proliferation of cancer cells by interfering with the growth factors.

The cancer cell regression and proliferation in the presence of TQ, shown in Figure 1, can be translated into the predator-prey type model of

$$\frac{dx}{dt} = p_x(x,y) - d_x(x,y)$$
$$\frac{dy}{dt} = p_y(x,y) - d_y(x,y) \quad (1)$$

where $p_x(x,y)$ is the cancer cell growth term, and $d_x(x,y)$ is referred to as the destruction of cancer cells. $p_y(x,y)$ is referred to as the proliferation of immune cells induced by TQ and $d_y(x,y)$ represents the inhibition of the TQ. Both $p_x(x,y)$ and $p_y(x,y)$ depend on the interaction between the cancer cell and TQ. In numerous problems of non-exponential growth of cancer cells, the Gompertz model is described as the kinetics cell proliferation (Emanuel & Emmanuel, 2000; Mazlan et al., 2017). For the specific functions $d_x(x,y)$, $p_y(x,y)$ and $d_y(x,y)$, the generic model (1) reduces to specific models derived by Kuznetsov et al. (1994) as follows

$$p_x(x,y) = ax - bx\ln x$$
$$d_x(x,y) = \alpha xy$$
$$p_y(x,y) = \frac{\rho xy}{g+x} \quad (1)$$
$$d_y(x,y) = mxy.$$

Substituting Eq. (2) in Eq. (1) leads to a system of ODEs

$$\frac{dx}{dt} = (ax - bx\ln x) - \alpha xy$$
$$\frac{dy}{dt} = \frac{\rho xy}{g+x} - mxy \quad (3)$$

In this model, parameter a is the intrinsic growth rate of the cancer cell and b reflects the rate of antiangiogenetic response. The attraction of TQ to cancer cells is captured through a Michaelis-Menten term, $\frac{\rho xy}{g+x}$, which introduces a saturation effect observed in biological uptake processes. Additionally, the term involving ρ represents the proliferation rate of TQ and g is a positive constant. The competitive interaction between cancer cells and TQ is expressed through mortality and inhibition terms, represented mathematically by the terms $-\alpha xy$ and $-mxy$, respectively. Numerous mathematical equations have been developed to predict the dynamics of cancer cell growth, with the assumption that proliferation constants remain time-invariant and are not significantly influenced by temporal changes (Norton, 1988; Hu et al., 2024).

The Gompertz model with stochastic parameters has been extensively researched because it can accurately represent the cell decelerations that occur during the growth of tumor cells (AbuKhader, 2013). A clinical data-stimulated stochastic model of Breast cancer, using the Gompertz equation, is employed for tumor development in Kazerouni et al. (2020), where tumor growth initially shows a similar pattern. The Gompertzian properties are modified before applying a random time-dependent process that causes it to become kinetically variable (Rana, 2020). Since the outcomes of the noisy phenomena affect biological structures, such as varying brain control over personal traits or muscular activity, like anxiety, which causes irregular variations, equation (1) should be extended to the stochastic form of SDEs (Rana, 2020; Ly et al., 2022). Consequently, the Wiener process must disturb the intrinsic growth rate parameters. Assuming that the intrinsic growth rate parameter varies according to noisy behavior, and randomness is incorporated into this, for example

$$a \to a + \sigma\frac{dW}{dt}$$

$$\rho \to \rho + \sigma\frac{dW}{dt}$$

here $\sigma > 0$ represents the rate of absorption for $t > 0$ is the Gaussian procedure with an increase in time h with mean zero and W_t is the is the Wiener process. The stochastic model of Gompertz is formulated as

$$dx = (ax - bx\ln x - \alpha xy)\,dt + \sigma x dW_t$$
$$dy = \left(\frac{\rho xy}{g+x} - mxy\right)dt + \sigma\left(\frac{xy}{g+x}\right)dW_t \quad (4)$$

4. Experimentation

HSC-4 cell lines were cultured in Dulbecco's Modified Eagle's Medium (DMEM) supplemented with 1% Penicillin-Streptomycin and 10% fetal bovine serum (Gibco, US). Cell proliferation was monitored over five days in serum-containing medium. Cells were seeded in a labeled 6-well plate at a density of 4 × 10^4 cells/mL.

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Cells in plate one were collected after 24 hours on day 1. Before counting, cells were rinsed with PBS, trypsinized, and resuspended in an equal volume of medium. This procedure was repeated daily for continued analysis. To prepare stock solutions, TQ was dissolved in molecular-grade DMSO (100 mg/mL, Gibco, US). HSC-4 cells were then treated with various TQ concentrations for 0, 12, 24, 36, and 48 hours to assess its in vitro toxicity on oral cancer cells. All experiments were conducted in triplicate for reliability. Results demonstrated that TQ significantly reduced the viability of oral cancer cells while having a minimal effect on normal cells. TQ exhibited toxicity in a cell-type-dependent manner, with its effects on HSC-4 cells increasing with concentration and exposure time. The IC50 values at 0, 12, 24, 36, and 48 hours remained constant at 0.0012 mg/mL. Figure 2 illustrates HSC-4 cells under control conditions, before treatment initiation. In Figure 3, the effects of TQ treatment on the cancer cell line are clearly apparent. After 48 hours of TQ exposure, a significant reduction in cancer cell count was observed, confirming the anticancer potential of TQ.

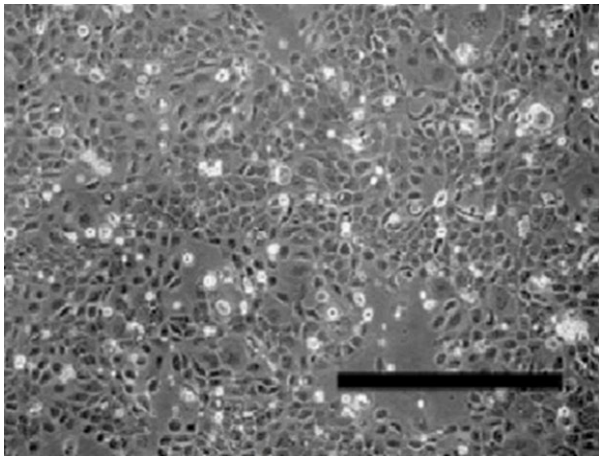


Figure 1. HSC-4 cell line (control).

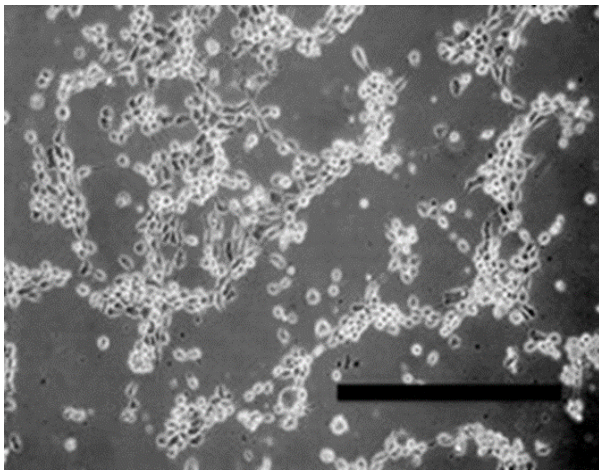


Figure 2. HSC-4 cell line after 24 hours of treatment.

5. Model analysis

We have established a mathematical model and in vitro experimentation that capture the TQ interaction with cancer cells. In this section, the characteristics of the deterministic model (3) in terms of equilibrium stability and positive solutions are investigated. We show that all solutions of system (3) with the initial conditions remain positive.

Theorem 1: Let $x(t)$ and $y(t)$ be the solutions to the system (3) with initial conditions $x(0)>0,\ y(0)>0.$ Then for all $t>0,$ the solutions of (3) remain positive, i.e., $x(t)>0,\ y(t)>0.$

Proof: System of ODEs (3) can be rewritten in a separable form of

$$\begin{aligned}\frac{dx}{x}&=(a-b\ln x-\alpha y)\,dt\\ \frac{dy}{y}&=\left(\frac{\rho x}{g+x}-mx\right)dy\end{aligned}\qquad(5)$$

By integrating both sides of system (5) from 0 to *t*, yield

$$x(t)=x(0)\exp\left[\int_0^t\left(a-b\ln x(s)-\alpha y(s)\right)ds\right]$$

$$y(t)=y(0)\exp\left[\int_0^t\left(\frac{\rho x(s)}{g+x(s)}-mx(s)\right)ds\right]$$

Given that $x(0)>0,\ y(0)>0$ and the exponential function is always positive, then it follows $x(t)>0,\ y(t)>0,$ for all $t>0.$ Thus, both $x(t)$ and $y(t)$ remain positive for all $t>0,$ which completes the proof.

Next, we identify the disease-free equilibrium (DFE), treatment failure equilibrium (TFE), and successful treatment equilibrium (SFE) points. After a period of treatment, it is assumed that the cancer cells may not increase but remain stable at the equilibrium points. To determine the equilibrium points, the right-hand side of the system (3) is set to zero such that

$$\begin{cases}(ax-bx\ln x)-\alpha xy=0\\ \dfrac{\rho xy}{g+x}-mxy-ey=0\end{cases}\qquad(2)$$

For disease-free equilibrium points, i.e., in the absence of tumor, it is clear that the cancer cells and the TQ, $y=0.$ Hence, the trivial is $E_0^*=(0,0).$ All biologically feasible equilibria admitted by system (3) and study the dynamics of the system

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around the treatment failure equilibrium (TFE) and successful treatment equilibrium (SFE) points are investigated. The treatment failure equilibrium occurs when the treatment level $y=0,$ implying the treatment does not affect the dynamics of the system. Using equation (6) yields either $x=0$ or $\ln(x)=\frac{a}{b},$ which implies $x=e^{\frac{a}{b}}$. Therefore, the treatment failure equilibrium is $E_1^*=\left(e^{\frac{a}{b}},0\right)$. The equilibrium for success treatment occurs when the treatment level of TQ, $y>0.$ From equation (6), we have $y=\frac{a-b\ln(x)}{\alpha}.$ Substituting y into the second equilibrium in equation (6) yields $y\left(\frac{\rho x}{g+x}-mx\right)=0.$ Since $y\neq 0,$ the term $\frac{\rho x}{g+x}-mx=0,$ i.e., $x=\frac{\rho}{m}-g>0,$ i.e. $\rho>gm.$ Hence, the success treatment equilibrium is

$$E_2^*=\left(\frac{\rho}{m}-g,\frac{a-b\ln\left(\frac{\rho}{m}-g\right)}{\alpha}\right),\quad \rho>gm.$$

Let $x(t)$ and $y(t)$ are solutions for system (3), and the system admits:

(i) Disease-free equilibrium, $E_0^*=(0,0).$

(ii) Treatment-failure equilibrium, $E_1^*=\left(e^{\frac{a}{b}},0\right).$

(iii) Successful treatment equilibrium,

$$E_2^*=\left(\frac{\rho}{m}-g,\frac{a-b\ln\left(\frac{\rho}{m}-g\right)}{\alpha}\right),\quad \frac{\rho}{m}-g>0.$$

6. Results and discussion

Figures 4 (a) – (c) show the dynamics in time and phase space plot of the Gompertz prey-predator model (3) for cancer cells (x) in the presence of TQ (y). The cancer cells tend to approach the equilibrium point of 0.19, as indicated by the red line graph in Figure 4(a). Meanwhile, the concentration of TQ tends toward 0.040, as indicated by the blue line in Figures 4(a) and 4(c).

Table 1 presents the estimated kinetic parameter values for the ODE (3) and SDE (4) models, obtained using the MCMC approach.

Table 1. The Kinetic Parameters of ODE and SDE

ODE (1)	SDE (2)
$g=0.1$	$g=0.2$
$b=0.95$	$b=0.95$
$m=0.71$	$m=0.71$
$\rho=1.1$	$\rho=1.1$
$\alpha=-0.003$	$\alpha=-0.003$
$a=0.6$	$a=0.4$
	$\sigma=0.01$

Figure 5 (a) and (b) present the experimental data and the simulated results for ODE (3) and SDE (4), respectively. In Figure 5(a), 500 simulations were conducted for SDE (4). Then, the average of the simulated results is computed. Both ODE and SDE demonstrate a decreasing trend of HSC-4 cell lines in the presence of TQ as an anticancer agent. The simulated results align with the experimental data of HSC-4 in the presence of TQ.

The root mean square error (RMSE) values are presented in Table 2, providing a quantitative measure of the models' adequacy in fitting the experimental data. Low values of RMSE signify a good fit, indicating that the model predictions closely correspond with the observed data. Since ODE and SDE Gompertz models yield RMSE values close to zero, they demonstrate sufficient ability to capture the underlying patterns in the experimental data. This result suggests that both models effectively represent the system's behavior.

Table 1. RMSE for HSC-4 Gompertz Stochastic and Deterministic Models

Model	RMSE
Deterministic (ODE)	0.015052227
Stochastic (SDE)	0.015043384

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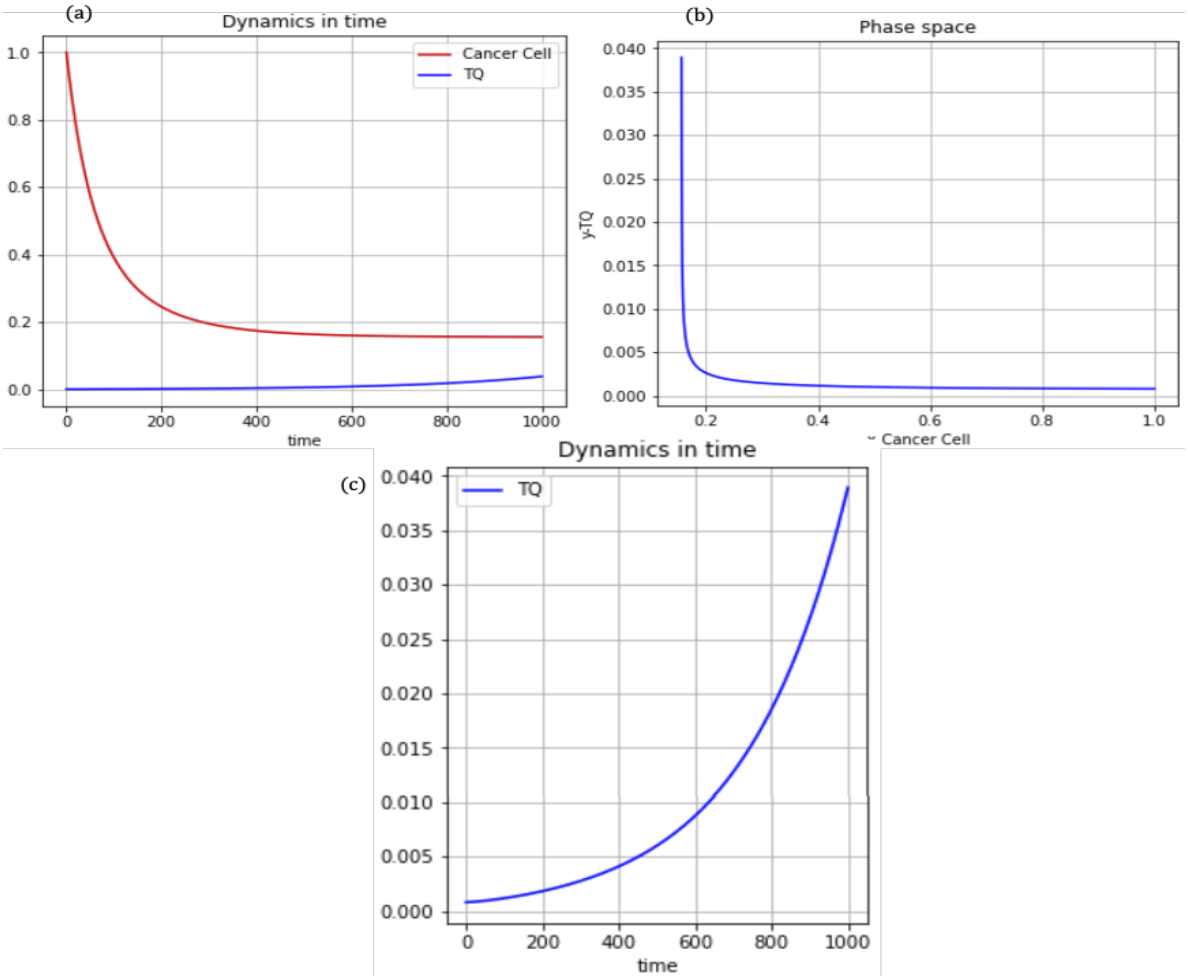


Figure 4. The phase plane plot of the Gompertz model (3) for $a = -0.5,\ b = 0.4,\ \alpha = 4.0,\ \rho = 1.1,\ g = 0.2$ and $m = 2.82.$

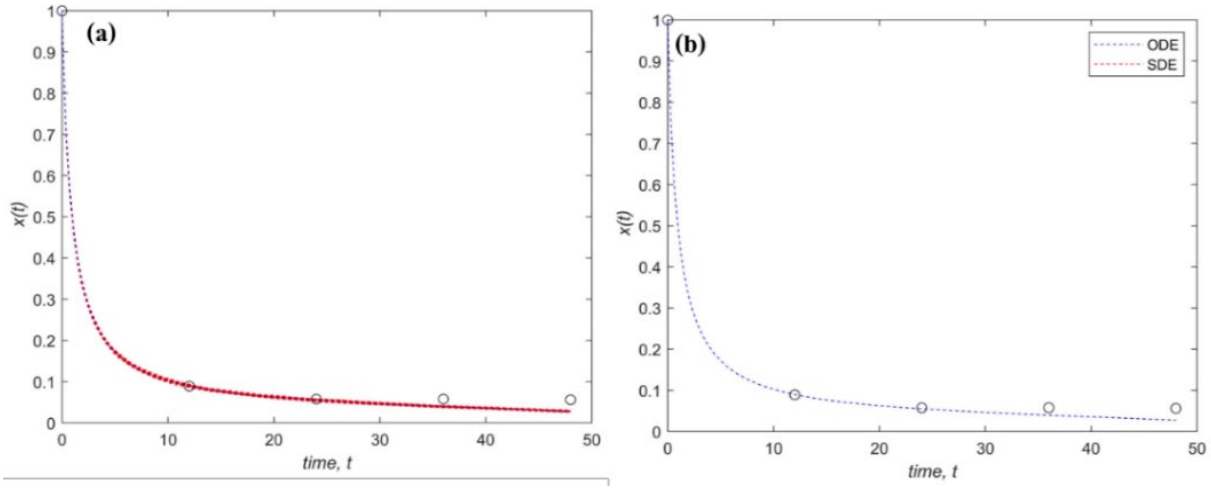


Figure 5. The Numerical Simulation for 500 simulations of the Gompertz model for the ODE (3), SDE (4), and the Experimental Data of HSC-4 Cell Lines.

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7. Conclusion

This study presents both deterministic and stochastic formulations of the Gompertz model to characterize the growth behavior of HSC-4 oral cancer cells in response to thymoquinone (TQ), a promising anticancer agent. The deterministic model assumes a predictable and smooth tumor growth trajectory, governed by fixed parameters and initial conditions. It is effective for representing the average behavior of the system. However, it fails to capture random biological fluctuations and uncertainties that arise from patient-specific variability, environmental factors, or measurement noise. To address this limitation, a stochastic version of the Gompertz model is introduced by incorporating noise terms into the differential equations. This extension permits modeling the probabilistic dynamics of tumor growth, offering a more realistic representation of the variability in cell response to TQ treatment.

The model demonstrates that TQ exhibited cytotoxicity in a cell-type-dependent manner, with its effects on HSC-4 cells increasing with concentration and exposure time. The observed decline in HSC-4 cell populations over time indicates a positive therapeutic response, supporting TQ's potential as a targeted oral cancer therapy. Furthermore, the study identifies equilibrium points, including the disease-free equilibrium (DFE), tumor-free equilibrium (TFE), and steady-state free equilibrium (SFE). These equilibrium states require further examination through both local and global stability analyses to understand the system's long-term behavior under various treatment conditions. Sensitivity analysis of the model parameters is also essential to assessing how changes in biological or pharmacological inputs affect model outcomes. This will enhance the reliability and predictive capability of the model, enabling more accurate and personalized treatment strategies. These aspects are intended to be explored in future research.

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