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Sensitivity analysis and simulation: Cells growth rate with mathematical modeling

Ghulam Murtaza[1], Riadh Marzouki[2], Muzamal Hussain*[1], Elimam Ali[3], Abdur Rauf[4] and Lubna Rasool[4]

[1]*Department of Mathematics, University of Sahiwal, Sahiwal, 57000, Pakistan*
[2]*Department of Chemistry, College of Science, King Khalid University, P.O. Box 9004, 61413 Abha, Saudi Arabia*
[3]*Department of Civil Engineering, College of Engineering in Al-Kharj, Prince Sattam Bin Abdulaziz University, Al-Kharj, 11942, Saudi Arabia*
[4]*Department of Chemistry, University of Sahiwal, Sahiwal, 57000, Pakistan*

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Abstract. Using the *NETCR* (normal-exposed-tumor-cancer-recovered) model, the present work is done to create a dynamic cancer model that shows how cancer progresses from normal cells to cancerous cells. By analyzing the dynamics of the disease and utilizing Python software to determine numerical simulation of cancer cells. The growth rates of normal, exposed, tumor and cancer cells are equivalent to the natural death rate of cells against concentrations of cells are depicted in various graphs. The rates at which cells divide are represented by the growth rates of normal, exposed, tumor, and cancer cells. Furthermore, sensitivity study is performed for various parameters and identified the optimal control strategies by utilizing nanotechnology approaches.
Keywords: cancer transmission; cells transmission; dynamical stability; nanotechnology; optimal control; sensitivity analysis

1. Introduction

One of the main causes of death in the modern world is cancer. More than 13 million people are predicted to be impacted by the cancer by 2030. Researchers began creating interaction models pertaining to tumor-immune cells in 1994 by utilizing the capabilities (Kuznetsov *et al*. 1994). The way in which infections interact with the immune system can be explained dynamically (Rajalakshmi and Ghosh 2018). Numerous models involving the use of various therapeutic modalities, including immunotherapy and chemotherapy, were presented by researchers based on these models (Feizabadi *et al*. 2009, Feizabadi and Witten 2010, Sharma and Samanta 2016). In order to explain clinically observed phenomena like tumor dormancy. The development of a unique tumor therapy strategy is described in the research conducted by (Kirschner and Tsygvintsev 1998). Cell is considered as a basic unit for living tissue. Cells are specified in structurally and physically for performing multiple tasks (Bosch *et al*. 2003, Zhu *et al*. 2024, Wang *et al*. 2024).

Nerve cells and muscle cells are excitable (Nicholls and Purves 1970). Currently, the most common cancer treatments are immunotherapy (Curti *et al*. 1996). Tumor-immune interaction modeling has garnered a lot of interest. This interaction is extremely complex, and De Boer *et al*. (1985) developed mathematical models in the form of ordinary differential equations. Many studies have employed various criteria, such as medication concentrations, to characterize the interaction between tumors and effector cells (Arafa *et al*. 2013). The dynamics of cancerous parts and cells have been explained by kinetic theory, agent-based models (ABM), hematopoietic stem cells and hematopoietic stem cells (Bianca 2017, Vizzari *et al*. 2020, Li *et al*. 2024, Yin *et al*. 2025). Researchers developed a variety of models based on these models that apply including immunotherapy, chemotherapy and cell therapy (Sun *et al*. 2025, Feizabadi 2010, Sharma 2016). Mathematical and numerical models have been used to investigate the effects of drug resistance levels on tumour cells, germ cell development and Photo-therapy of Tuberculosis, which exhibit drug resistance (Lavi 2012, Zhu *et al*. 2024, Wang *et al*. 2024). The bulk of malignancies manifest as a result of several anomalies accumulating over a long period of time (10–15 years). Some people having not shown symptoms before heart attack but some people show symptoms for a few days or hours (Messori *et al*. 2016). Lin *et al*. (2016) and Tong *et al*. (2022) investigated the sensitivity of Nasopharyngeal cells and effects of GLP-1 receptor agonists.

Lyons and Duggan (2015) presented some heart abnormal conditions as Heart rhythm variability: Heart rhythm does not work properly due to electric short circuits SA-nodes and AV- nodes. Heart failure: Heart failure occurs, when heart tissues are not pumping blood from heart. Heart arrest without warning: Sudden heart arrest condition is very dangerous. This condition occurs, when SA-nodes and AV-nodes electric circuits are breakdown and heart does not receive properly message. Hu *et al*. (2024) and Lin *et al*. (2016) studied expression of promotion of cells and resistance to nonsmall cell lung cancer cells.

Numerous important cancer treatment methods, including immunotherapy, chemotherapy, targeted drug therapy, virotherapy, and many more, were developed in 2019 by

*Corresponding author, Ph.D.,
E-mail: muzamal45@gmail.com;
muzamalhussain@uosahiwal.edu.pk

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Unni and Padmanabhan Seshaiyer. It develops a novel mathematical model that integrates medication delivery to immune system cells, including natural killer cells, with important interactions between tumor cells and these cells. Furthermore, some improvements in computational and analytical models have been made to help shed light on clinical observations. A system of numerically solved ordinary differential equations describes these interactions. Cancer is the undefined and uncontrolled proliferation of abnormal cells in the body. It ranks as the world's second most common cause of death. The body's cells divide and die in accordance with the needs of the body during a typical cycle of multiplication. The body's cells divide rapidly, and their proliferation becomes unmanageable. It could also happen because the cells do not die and forget their lifespan. Different types of cancer exist. Cancer can develop in any organ or tissue, including the brain, liver, ovary, breast, lungs, kidneys, bones, colon, and blood. Zhang *et al*. (2018) and Zeng *et al*. (2024) investigated how oridonin affects the expression of cytochrome P450 and controls the characteristics of bone marrow mesenchymal stem cells.

Hanif (2018) utilized the glucose model just as other comparative ODE frameworks as investigatory demonstrating devices measure insulin discharge and insulin affectability in singular patients. A variety of nonlinear modeling techniques have been employed recently by various researchers (Safaei *et al*. 2019, Benmansour *et al*. 2019, Akbaş 2020, Forsat *et al*. 2021, Luo *et al*. 2022, Moradi *et al*. 2023, Mishra *et al*. 2025, Meher *et al*. 2024a, b, Patel *et al*. 2023, He *et al*. 2024).

Wu *et al*. (2025) and Chen *et al*. (2024) conducted the research in myeloma cells through NRF2 activation and chemodynamics for reprogramming the Tumor immune system. It is crucial to incorporate the effects of pharmaceuticals into the models in order to comprehend the effects of the treatments that are administered to the location of the tumor cells. In order to accomplish this, we create a mathematical model that combines drug delivery models to several cell sites with crucial interactions between developing tumor cells and innate and specialized immune system cells. Our objective is to stop the spread of cancers, and these models were created to evaluate the effectiveness of anticancer medications.

In 1982, Witten put out the first mathematical model that illustrated the interplay between healthy and cancerous cells (Lyu *et al*. 2024, Li *et al*. 2018). Waldman and colleagues (Waldman *et al*. 2020) provided a thorough historical and biological perspective on the creation and application of cancer immunotherapy in a related study. They emphasized the critical role of T cell modulation and looked at several clinical trials that showed the effectiveness of several medication classes. They talked about the negative consequences as well. In order to analyze and simulate the dynamics of the normal-exposed-tumor-cancer-recovered ($NETCR$) model, which takes into account the primary cell populations that influence the appearance of tumor cells in tissue (normal cells, exposed cells, tumor cells, cancer cells and recovered cells), it is assumed that the immune system has been weakened due to a prolonged period of poor diet. The impacts of aberrant cells changing into tumors and subsequently cancer cells on the normal cell cycle are dynamically represented by this model. A cancer emergency is declared when abnormal cells appear and tissue grows more quickly than usual.

Investigated is the relationship between the growth rates of normal, exposed, tumor, and cancer cells and the corresponding natural cell death rate. Additionally, there is an impact on the rates of normal cell division, exposed cell division, tumor cell division, and cancer cell division, which are equivalent to the rates of normal cell division, exposed cell division, tumor cell division, and cancer cell division, respectively.

2. Formulation of the mathematical model

In order to learn more about the mechanisms controlling cancer transmission, researchers have thoroughly investigated cancer dynamics using mathematical models. The following is the formulation of the normal-exposed-tumor-cancer-recovered ($NETCR$) model, which describes the transmission of cancer from normal cells to recovered cells:

$$\begin{aligned} M(t) &= N(t) + E(t) + T(t) + C(t) + R(t) \\ \frac{dN}{dt} &= a_1\mathrm{N}(1 - b_1 N) - \alpha NE - \gamma_1 NT - \gamma_2 NC - \mu N \\ \frac{dE}{dt} &= a_2\mathrm{E}(1 - b_2 E) + \alpha NE - \rho_1 E - \mu E \\ \frac{dT}{dt} &= a_3\mathrm{T}(1 - b_3 T) + \gamma_1 NT + \rho_1 E - \beta_1 T - \beta_2 T - \mu T \\ \frac{dC}{dt} &= a_4\mathrm{C}(1 - b_4 C) + \gamma_2 NC - \rho_2 C + \beta_1 T - \mu C \\ \frac{dR}{dt} &= \beta_2 T - \mu R \end{aligned} \quad (1)$$

where $M(t)$ is the compartment for all population cells, $N(t)$ is the compartment for normal cells, $N(t)$ is the compartment for exposed cells, $T(t)$ is the compartment for tumor cells, $C(t)$ is the compartment for cancer cells, and $R(t)$ is the compartment for recovered cells, in that order.

2.1 Invariant Region

For the system (1) invariant region is stated as, $\Omega \in R_+^5$, where $\Omega = \left\{ \begin{array}{c} (N, E, T, C, R) \in R_+^5 : \\ \mathrm{M} = \mathrm{N} + \mathrm{E} + \mathrm{T} + \mathrm{C} + \mathrm{R} \\ = \frac{(a_1 + a_2 + a_3 + a_4)}{\mu} \end{array} \right\}$. The region Ω is verified to be positively invariant by the set theorem (Pradeep and Shrivastava 1990, Halanay and Rasvan 2012) of local invariant. It demonstrates that in this area, the definitions of the mathematical and epidemiological model (1) are valid.

3. Numerical simulations and discussion

Through the use of numerical simulations, which are displayed in Figs. 2-9, we have described the parameters in relation to sensitivity analysis and simulation. Parameters values based on the model (1).

Table1 Sensitivity indices of R_0 parameters for normal to cancer cells transmission

Parameters	Description	Values	Sign
a_1	Normal cells Growth rate	0.30	+ve
a_3	Tumor cells Growth rate	0.31	+ve
b_1	Division rate of Normal Cells	-1	-ve
γ_1	Inhibit rate of Tumor on normal cells	$+1$	+ve
β_1	Transmission rate of Tumor cells to Cancer cells	-0.66	-ve
β_2	Transmission rate of Tumor cells to Recovered	-0.66	-ve
μ	Natural death rate of cells	-0.30	-ve

Table 2 Parameters values based on the model (1) (Alharbi and Rambely 2020)

Parameters	Description	Values	Source
a_1	Normal cells Growth rate	0.4312	(Alharbi and Rambely 2020).
a_2	Exposed cells Growth rate	0.4426	(Alharbi and Rambely 2020).
a_3	Tumor cells Growth rate	0.4426	fitted
a_4	Cancer cells Growth rate	0.4426	fitted
b_1	Division rate of Normal Cells	2.99×10^{-6}	(Alharbi and Rambely 2020).
b_2	Division rate of Exposed Cells	0.4	(Alharbi and Rambely 2020).
b_3	Division rate of Tumor Cells	0.4	fitted
b_4	Division rate of Cancer Cells	0.4	fitted
α	Inhibit rate of Exposed on normal cells	0.1379	(Alharbi and Rambely 2020).
γ_1	Inhibit rate of Tumor on normal cells	0.9314	(Alharbi and Rambely 2020).
γ_2	Inhibit rate of Cancer on normal cells	0.9314	fitted
ρ_1	Transmission rate of Exposed Cells to Tumor cells	0.1379	fitted
ρ_2	Eliminated Cancer Cells	0.1379	fitte
β_1	Transmission rate of Tumor cells to Cancer cells	0.9314	fitted
β_2	Transmission rate of Tumor cells to Recovered cells	0.9314	fitted
μ	Natural death rate of cells	0.1	estimated

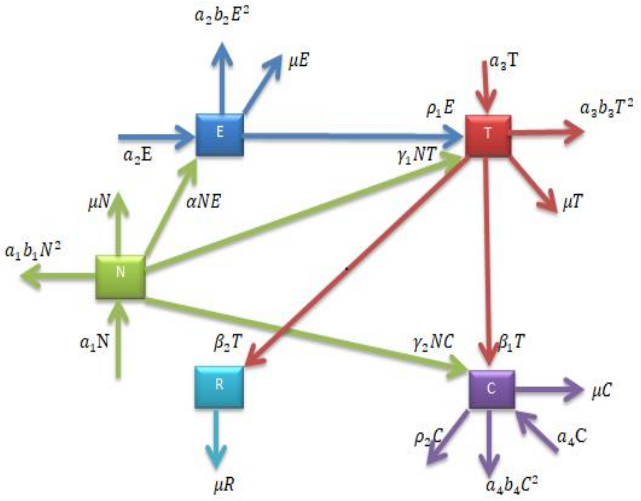

Fig. 1 Dynamical schematic diagram of the system (1)

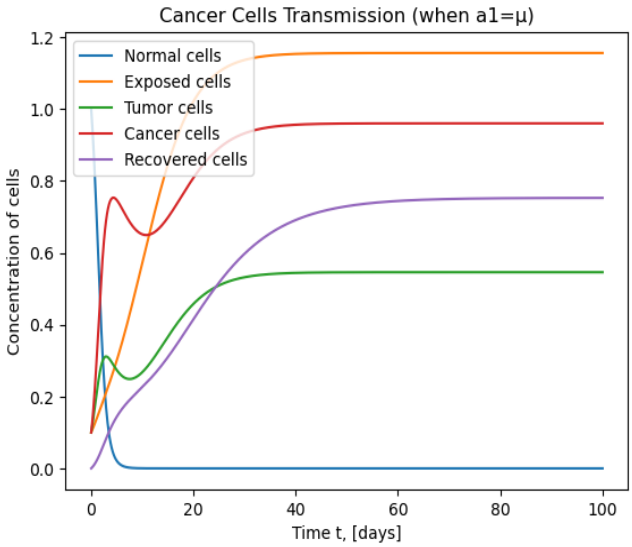

Fig. 2 Cancer cell transmission with a_1 = μ = 0.4312

Table 1 reveal the values discovered in the sensitivity indices, It is important to remember that any positive index that the sensitivity analysis produced that raised the threshold number of disease cells would also raise it, and vice versa. Examining Table 1 reveals a clear correlation between the upward trends in parameters, a_1, a_3 and γ_1, and the spread of cell transmission. Furthermore, it is clear that the bad values indicated by parameters, μ, b_1, β_1 and

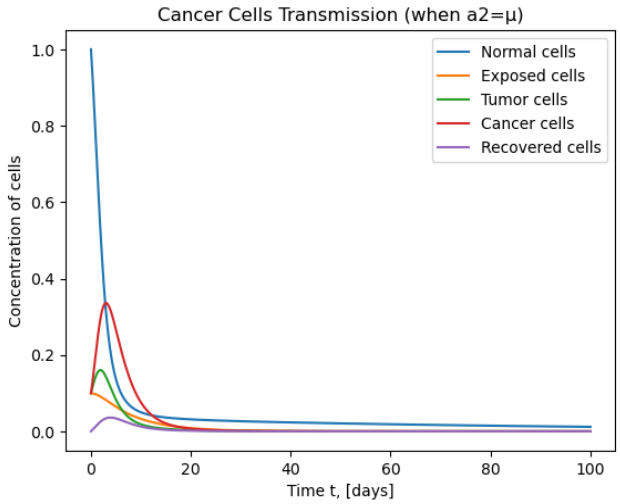

Fig. 3 Cancer cell transmission with $a_2 = \mu = 0.4426$

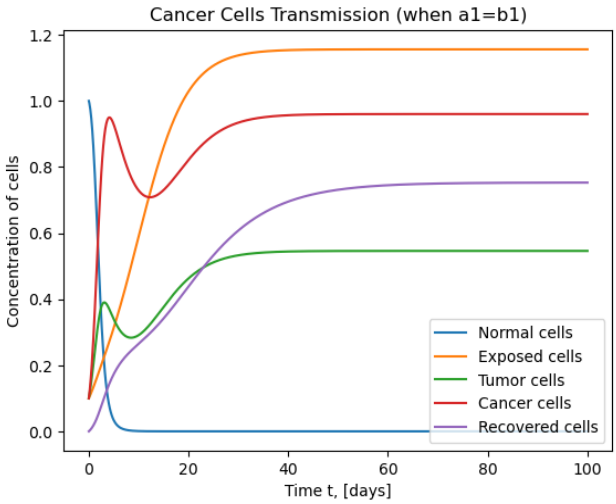

Fig. 6 Cancer cell transmission with $a_1 = b_1 = 2.99 \times 10^{-6}$

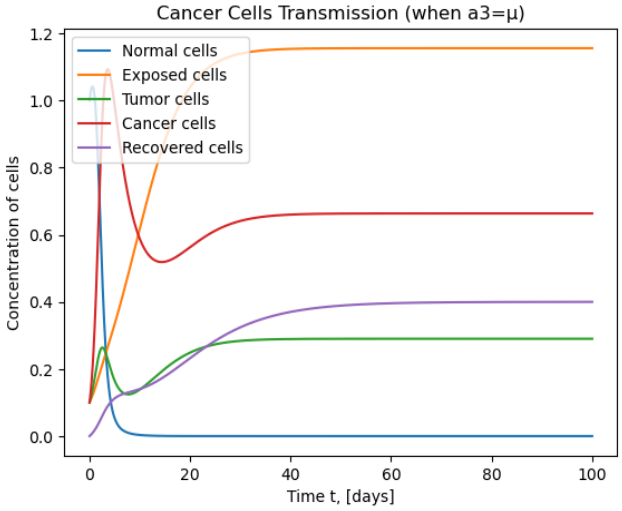

Fig. 4 Cancer cell transmission with $a_3 = \mu = 0.4426$

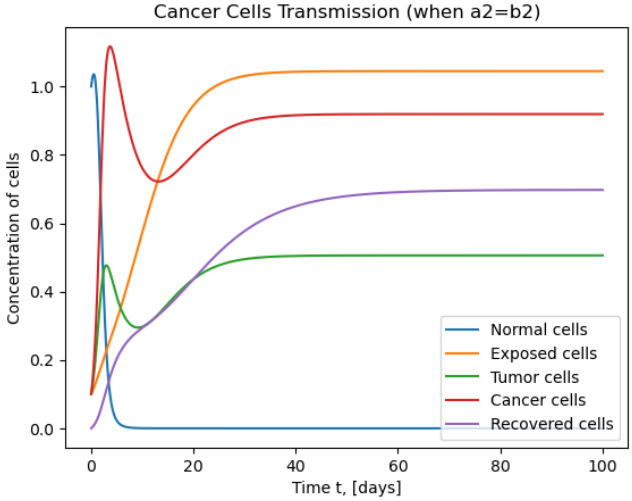

Fig. 7 Cancer cell transmission with $a_2 = b_2 = 0.4426$

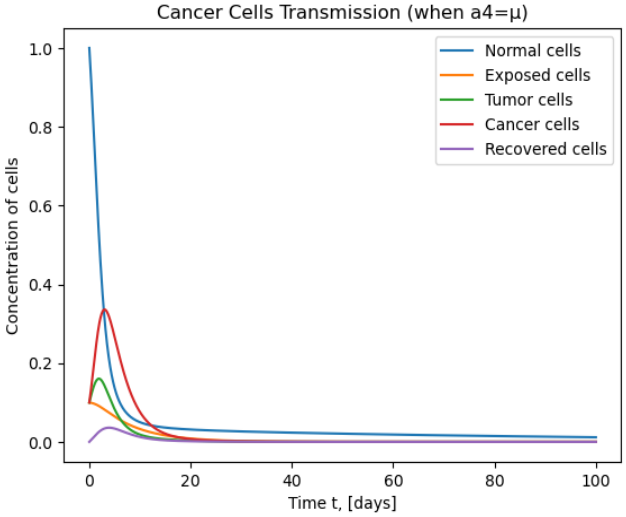

Fig. 5 Cancer cell transmission with $a_4 = \mu = 0.4426$

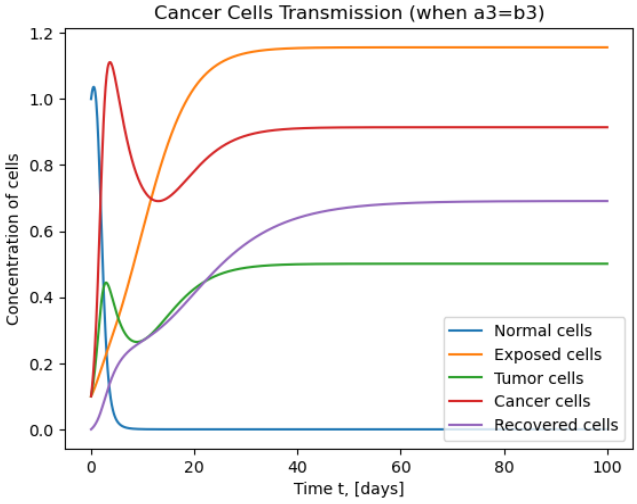

Fig. 8 Cancer cell transmission with $a_3 = b_3 = 0.4426$

β_2 are negatively correlated with cell transmission. The sensitivity indices indicate that the most sensitive parameter is γ_1, which represents the speed of transmission from normal to tumor cells. Parameters values are shown in Table

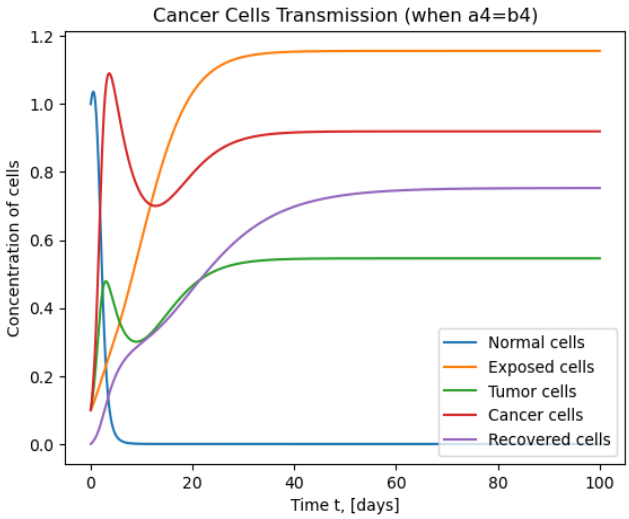

Fig. 9 Cancer cell transmission with $a_4 = b_4 = 0.4426$

2. Dynamical schematic diagram of the system is shown in Fig. 1.

Figs. 2-5 display the transmission rate of cells in the ($NETCR$) model with comparative sensitivity analysis and simulation, with the parameters $a_1 = \mu = 0.4312$, $a_2 = \mu = 0.4426$, $a_3 = \mu = 0.4426$ and $a_4 = \mu = 0.4426$ respectively. The simulation is shown in graphs (Figs. 2-5) where the growth rate of exposed, normal, tumor and cancer cells is equal to the cell death rate. Figs. 6-9 shows the cancer cell transmission with parameters, $a_1 = b_1 = 2.99 \times 10^{-6}$, $a_2 = b_2 = 0.4426$, $a_3 = b_3 = 0.4426$ and $a_4 = b_4 = 0.4426$ respectively. The transmission rate of cells in the ($NETCR$) model with comparative sensitivity analysis and simulation is shown in graphs Figs. 6-9. The growth rates of normal, exposed, tumor, and cancer cells in the simulation are equal to the rates at which cells divide.

4. Conclusions

This work examines the (NETCR) model for the transfer of healthy cells to malignant cells. Figures show the equilibrium points of the disease. Investigated is the relationship between the growth rates of normal, exposed, tumor, and cancer cells and the corresponding natural cell death rate. Additionally, there is an impact on the rates of normal cell division, exposed cell division, tumor cell division, and cancer cell division, which are equivalent to the rates of normal cell division, exposed cell division, tumor cell division, and cancer cell division, respectively. It is noticed that the variables that tie this system of equations together are the rates at which normal cells spread to cancer cells, exposed cells spread, tumor cells spread, and cancer cells spread.

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