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Control of Dissipative Dynamics in a Novel Biological Chaotic System

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ABSTRACT: In this study, we have demonstrated a novel procedure to model and control dissipative behaviour in biological systems by setting up synchronization and anti-synchronization between two non-identical nonlinear dynamical systems: one exhibiting tumour cell proliferation and the other capturing activity among environmental bacteria, viruses, and temperature. By designing the nonlinear controller through active control strategies, we have managed the chaotic nature of the trajectories of these systems. This work offers insights into temperature-sensitive therapeutic strategies and microbial applications in biomedicine.

Key Words: Bacteria, cancer, chaos synchronization, tumour growth, virus.

Contents

1. Introduction

In the natural world, Nonlinearity is omnipresent. Therefore, inspection of the messy nature of the inconsistent systems, which develop with time, is an intricate work, but it helps to understand the nature of the environmental behaviour. Dissipative systems are extremely conscious about their initial states, and this type of receptive behaviour is commonly familiar with the influence of butterflies described by [1]. Sensitive dependence, determinism and nonlinearity are the major components of these systems. In the past, primarily, the systems that could be identified were either straight or followed specific guidelines and configurations, as detailed in Blekman (1988) [2]. However, with the advent of modern computers, the analysis of disordered systems could be observed by mathematicians. Due to the widespread applications of dissipative systems, the study of chaos has great importance in the field of research.

A wide variety of synchronization states have been examined over the past thirty years. In these years, various researchers worked on complete or identical synchronization (Fujisaka and Yamada; Pecora and Carol; Ovich et al. (1986)) [3,4,5] phase synchronization (Rosa et. al (1998); Rosenblum et al. (1996) [6,7], Lag synchronization (Rosenblum et al. (1997)) [8], generalized synchronization (Kokarev and Parlitz (2004) [9]; Rulkov et al. (1995)) [10], intermittent lag synchronization (Boccaletti and Valladares (2000)) [11], imperfect phase synchronization (Zark et al. (1999)) [12] and almost synchronization (Femat and Perales (1999)) [13].

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In this research work, we employ complete synchronisation, which appears to be the most fundamental type of synchronisation in chaotic systems, as detailed in Fujisaka and Yamada (1983). In 1990, Pecora and Carroll introduced a different process to achieve synchronization of chaotic systems, popularly known as the PC method. The investigation done by Pecora and Carroll is highly important as chaos theory predicts that nearby trajectories diverge rapidly as time passes. Hence, achieving synchronization of chaotic systems appears to be unattainable due to the unavoidable slight variations in system characteristics and the presence of noise. In the majority of cases, synchronisation refers to the property of chaotic systems whereby they exhibit comparable behaviour at the same time or share a common moment.

The logical progression of these groundbreaking studies has pushed researchers to examine the synchronisation phenomena in extended or infinite-dimensional systems. The synchronization has been tested in experiments or real-world systems to understand the underlying mechanism of synchronization process. Itik and Banks (2010) [14] used a more rigorous mathematical approach and found chaos in the De Pillis and Randunskaya (2003) model [15]. They discovered the Lyapunov exponents for a particular set of parameters and proved that chaos existed in the basic model. Nikolov and Wolkenhauer (2010) [16] explored tumours as chaotic attractors and noted the complex behaviour of the anomalous attractors.

Cavicchioli et al. (2019) [17] recently searched for the effects of climate change caused by microorganisms. They hypothesised during their inquiry that the diversity of the microbial population plays a vital role in the change of environmental temperature.

In this paper, we introduce a novel chaotic dynamical system which characterises the changes in the population of tumour cells. This model evokes population dynamics caused by interactions and competition of tumour cells with other cells of the body. The other dynamical system, involving microorganisms, is constructed from the Chen and Lu system (2002) [18]. We have attained the synchronization and anti-synchronization between these two chaotic non-identical systems via active control technique proposed by Bai and Lonngren (1997) [19].

2. Lyapunov Exponents

Lyapunov exponents quantify the chaotic behaviour of dynamical systems. In a state-space with more than one dimension, we can associate Lyapunov exponents with the rate of expansion and contraction of trajectories; therefore, a measure of sensitivity to tiny changes in initial circumstances can be used to define chaotic behaviour in a dynamical system. A Lyapunov exponent can be determined using the time step (t) and the initial separation (r0) as follows:

$$\mu = \lim_{n\to\infty} \frac{1}{n\Delta t} \sum_{i=1}^{n} \ln\left(\frac{r_i}{r_0}\right) \tag{2.1}$$

At step i, r_i is the distance between neighbouring points given in Otto and Day (2007) [20]. A Lyapunov exponent exists for each dimension of a dynamical system, which can be arranged in descending order ($\mu_1 \geq \mu_2 \geq \mu_3 \dots$) to give μ_1, the maximal Lyapunov exponent (MLE). Only MLE will be explored in this paper because chaos can be recognized by the criteria "MLE" > 0, which implies chaotic behavior". Abernethy and Gooding (2018) observed that MLE dominates the divergence between neighbouring orbits for big enough n [21], and hence, Equation (1) can be understood as an expression for the MLE.

3. Mathematical Model

In this section, we design a mathematical model that depends on the interaction between microorganisms in the appropriate environment. This model is derived from Sparrow's model (1982) [22]. In particular, we consider the interaction between the population of viruses and bacteria in the environment with the following postulates: P1: We particularly include the population of arthrobacter and consider that these will increase with time. P2: Initially, the temperature is high. With these postulates, we consider the variables l1(t), the temperature; m1(t), the bacteria population and n1(t), the virus population. The model is designed as a system of ordinary differential equations, described as follows

$$\frac{dl_1}{dt} = -al_1 + \alpha m_1$$
$$\frac{dm_1}{dt} = -l_1 + cm_1 - l_1 n_1$$
$$\frac{dn_1}{dt} = l_1 m_1 - bn_1 \tag{3.1}$$

with initial conditions $l_1(0) = l_{1_0}, m_1(0) = m_{1_0}, n_1(0) = n_{1_0}$. Further, we construct a model which embodies the changes in the population of tumour cells and is structured by categorising the cell population into three classes. Corresponding to each class, we define a variable which represents the cell population, namely: $l_2(t)$, the tumour cells; $m_2(t)$, the healthy host cells near the tumour and $n_2(t)$, the effective immune cells; with the following assumptions:

A_1. Consider only non-metastatic malignancies in the interactions of the cell population.

A_2: The growth of the cancer cells is non-linear.

A_3: Interactions of immune cells with cancer cells affect the activity of host cells.

A_4: Interactions between host and immune cells slow down the activity of tumour cells.

With these assumptions, the model consists of a system of three ordinary differential equations categorising the changes in the cell population given as follows:

$$\frac{dl_2}{dt} = -ul_2 n_2 - m_2 + l_2^2$$
$$\frac{dm_2}{dt} = -l_2 n_2 + v$$
$$\frac{dn_2}{dt} = l_2 m_2 - wn_2 \tag{3.2}$$

4. Existence of Chaos

For System (2), we have determined that with parameters $a = 36, c = 16, b = 10$ and $\alpha = 2$, the Lyapunov exponents are $(1.4284, 0.0649, -34.5675)$, which will be observed in Figure 1. Therefore, the System (2) exhibits chaotic behaviour and the existence of an attractor, as shown in Figure 2.

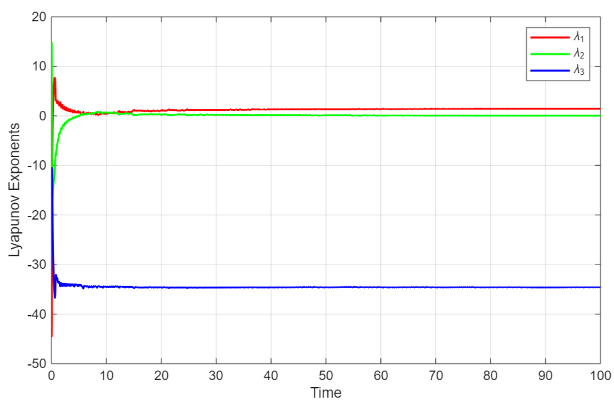

Figure 1: Convergence of Lyapunov Exponents.

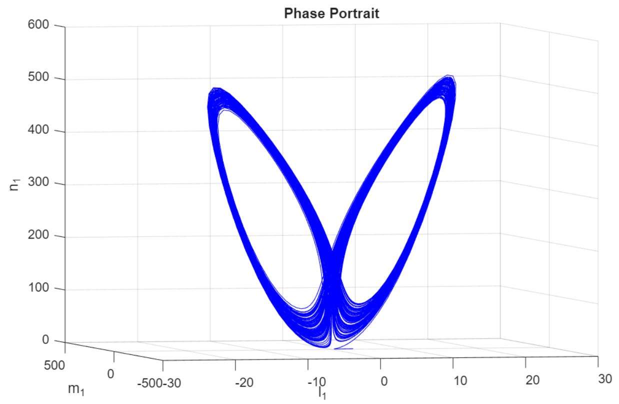

Figure 2: Onset of chaos in microbial model.

For System (3), we have observed that with the parameters $u = 5$, $v = 7$, and $w = 2$, the Lyapunov exponents are $(2.0319, -2.9460, -2.9460, -28.8831)$, which have observed in Figure 3. Therefore, System (3) exhibits chaotic behaviour, as shown in Figure 4.

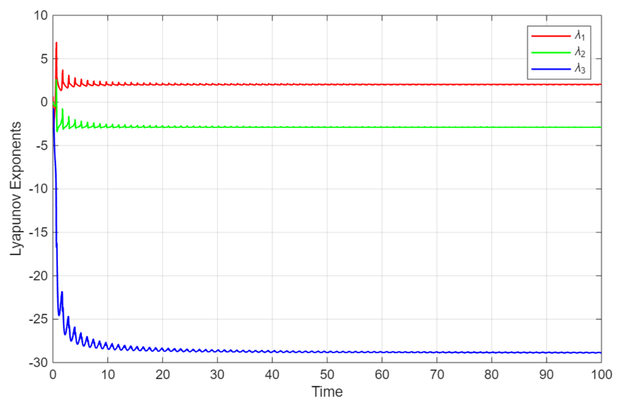

Figure 3: Lyapunov spectrum Exponents System (3).

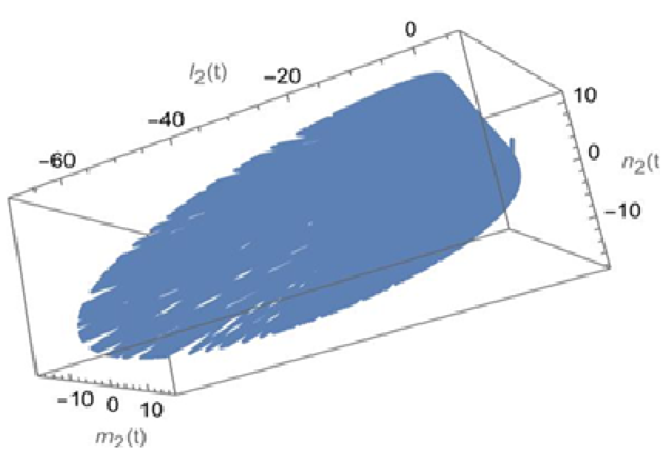

Figure 4: Onset of chaos in Cancer cells model.

5. Chaos Synchronization

If System 2 is considered as the drive system and System 3 as the response system, then the drive system and the response system are described as follows:

$$\begin{aligned} \frac{dl_1}{dt} &= -al_1 + \alpha m_1 \\ \frac{dm_1}{dt} &= -l_1 + cm_1 - l_1 n_1 \\ \frac{dn_1}{dt} &= l_1 m_1 - bn_1 \end{aligned} \tag{5.1}$$

$$\begin{aligned} \frac{dl_2}{dt} &= -ul_2 n_2 - m_2 + l_2^2 + h_1(t) \\ \frac{dm_2}{dt} &= -l_2 n_2 + v + h_2(t) \\ \frac{dn_2}{dt} &= l_2 m_2 - wn_2 + h_3(t) \end{aligned} \tag{5.2}$$

The nonlinear controllers $h_1(t), h_2(t) and h_3(t)$ are introduced in System (5). To determine the control functions $h_1(t), h_2(t)$ and $h_3(t)$, the state error variables between Systems (4) and (5) are described as

$$\begin{aligned} E_1 &= l_2 - l_1 \\ E_2 &= m_2 - m_1 \\ E_3 &= n_2 - n_1. \end{aligned} \tag{5.3}$$

The error dynamical System corresponds to Systems (4) and (5):

$$\begin{aligned} \dot{E}_1 &= -ul_2 n_2 + l_2^2 - m_2 + al_1 - \alpha m_1 + h_1(t) \\ \dot{E}_2 &= -l_2 n_2 + v - cm_1 + l_1 n_1 + l_1 + h_2(t) \\ \dot{E}_3 &= l_2 m_2 - wn_2 + bn_1 - l_1 m_1 + h_3(t) \end{aligned} \tag{5.4}$$

We define active control functions $h_1(t), h_2(t)$ and h_3(t) as follows

$$\begin{aligned} h_1(t) &= k_1(t) + ul_2 n_2 - l_2^2 + (1+\alpha)m_1 - al_1 \\ h_2(t) &= k_2(t) + l_2 n_2 - v - l_1 - l_1 n_1 + (c+1)m_1 - m_2 \\ h_3(t) &= k_3(t) - l_2 m_2 - (b-w)n_1 + l_1 m_1 \end{aligned} \tag{5.5}$$

where $k_1(t), k_2(t)$ and k$_3$(t) are control inputs defined as a function of error state variables E_l, E_2 and E_3. Using System 8, System 7 reduces to the following form

$$\begin{aligned} \dot{E}_1 &= -E_2 + k_1(t) \\ \dot{E}_2 &= -E_2 + k_2(t) \\ \dot{E}_3 &= -wE_3 + k_3(t). \end{aligned} \tag{5.6}$$

The error System (9) must be controlled as a linear system. When the System (9) is stabilized by control inputs $k_1(t), k_2(t)$ and $k_3(t)$, then E_l, E_2 and E_3 will converge to zero as time $t \to \infty$, which implies that Systems (2) and (3) are synchronized. To achieve this goal, consider the Lyapunov function defined by

$$V = \frac{1}{2}\left(E_1^2 + E_2^2 + E_3^2\right) \tag{5.7}$$

Differentiating V along the trajectories of the closed-loop error dynamics, we get

$$\begin{aligned} \dot{V} &= E_1\dot{E}_1 + E_2\dot{E}_2 + E_3\dot{E}_3 \\ &= E_1\left(-E_2 + k_1(t)\right) + E_2\left(-E_2 + k_2(t)\right) + E_3\left(-wE_3 + k_3(t)\right) \\ &= -E_1E_2 - E_2^2 - wE_3^2 + E_1k_1(t) + E_2k_2(t) + E_3k_3(t) \end{aligned} \tag{5.8}$$

where control inputs $k_1(t), k_2(t)$ and $k_3(t)$ are functions of E_l, E_2 and E_3 chosen as

$$\begin{bmatrix} k_1(t) \\ k_2(t) \\ k_3(t) \end{bmatrix} = B \begin{bmatrix} E_1 \\ E_2 \\ E_3 \end{bmatrix} \tag{5.9}$$

Where B is 3×3 constant matrix to be determined, with all the eigenvalues are negative, and $\dot{V}$ is negative semi-definite. We have determined the matrix B in the following form:

$$B = \begin{bmatrix} -1 & 1 & 0 \\ 0 & -1 & 0 \\ 0 & 0 & -1 \end{bmatrix} \tag{5.10}$$

Using 10 and 11, we have

$$\begin{aligned} \dot{V} &= -E_1E_2 - E_2^2 - E_3^2 - E_1^2 + E_1E_2 - E_2^2 - E_3^2 \\ &= -E_1^2 - 2E_2^2 - (w+1)E_3^2, \text{w} > 0. \end{aligned} \tag{5.11}$$

Using Lyapunov stability theory, this will lead to $\lim_{n\to\infty} \|e(t)\| = 0$, which implies that the synchronization between Systems 2 and 3 is achieved.

5.1. Numerical Simulation of Synchronization

The analysis of the numerical study is carried out using Matlab. We have considered the parameters of the drive System (4) and response System (5) as $a = 36, b = 10, c = 16,\ \alpha = 2$ and $u = 5, v = 27, w = 2$. Further, we take the initial consitions for Systems (4) and (5) as $l_1(0) = 50, m_1(0) = 10, n_1(0) = 10$ and $l_2(0) = 3, m_2(0) = 2, n_2(0) = 1$. As a result, the initial states of the error system (9) are $E_1(0) = -47, E_2\,(0 = -8$, and $E_3(0 = -9$. Figures 5,6, and 7 depict the dynamics of synchronization for the state variables for the Systems (4) and (5) under control Law 8. In Figure 8, we have noticed that the errors between Systems (2) and (3) converge rapidly to zero as time tends to infinity.

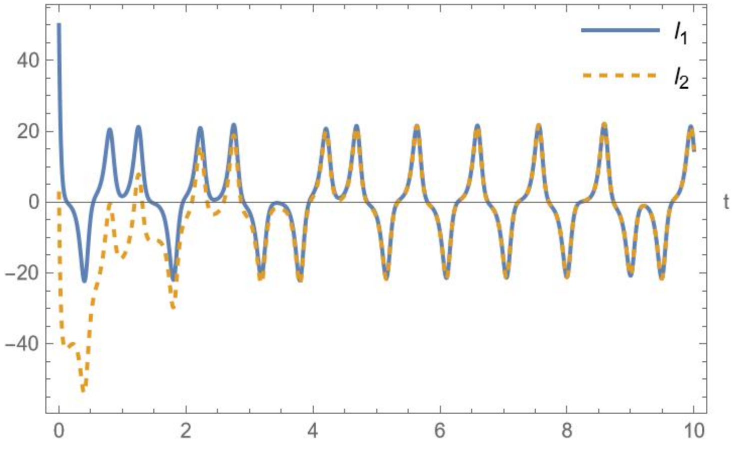

Figure 5: Controller impact on time series of l_1 and l_2.

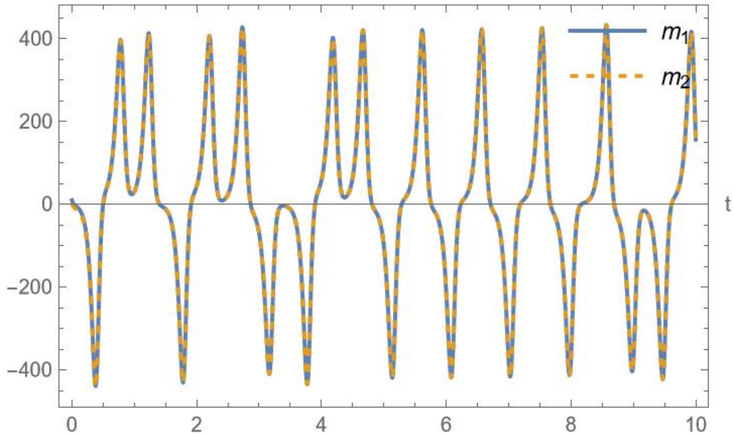

Figure 6: Controller impact on time series of m_1 and m_2.

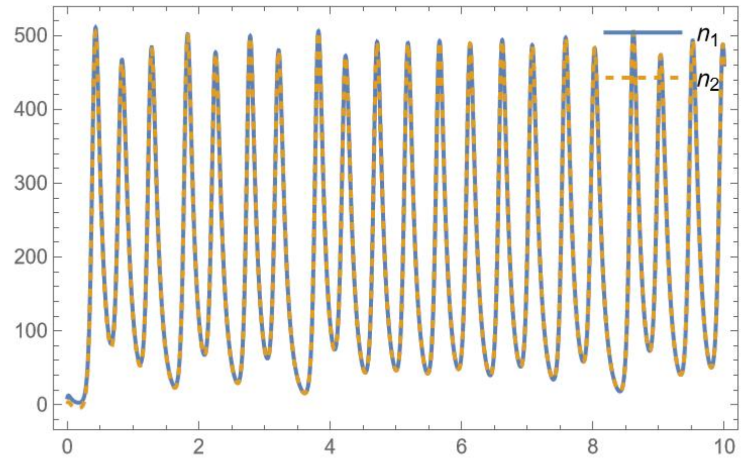

Figure 7: Controller impact on time series of m_1 and m_2.

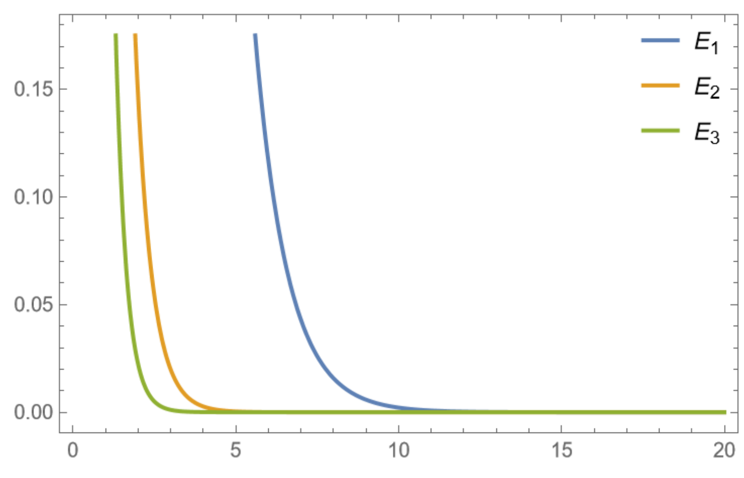

Figure 8: Controller impact on time series of m_1 and m_2.

6. Anti-synchronization

If System (2) is considered as the drive system and System (3) as the response system, then the drive system and the response system are described as follows:

$$\begin{aligned}\frac{dl_1}{dt} &= -al_1 + \alpha m_1\\ \frac{dm_1}{dt} &= -l_1 + cm_1 - l_1 n_1\\ \frac{dn_1}{dt} &= l_1 m_1 - bn_1\end{aligned} \tag{6.1}$$

$$\begin{aligned}\frac{dl_2}{dt} &= -ul_2 n_2 - m_2 + l_2^2 + r_1(t)\\ \frac{dm_2}{dt} &= -l_2 n_2 + v + r_2(t),\\ \frac{dn_2}{dt} &= l_2 m_2 - wn_2 + r_3(t),\end{aligned} \tag{6.2}$$

The nonlinear controllers $r_1(t), r_2(t)$ and $r_3(t)$ are introduced in System 12. To determine the control function $r_1(t), r_2(t)$ and $r_3(t)$, we defined state error variables between Systems 13 and 14 as

$$\begin{aligned}e_1 &= l_2 + l_1\\ e_2 &= m_2 + m_1\\ e_3 &= n_2 + n_1.\end{aligned} \tag{6.3}$$

The error dynamical System corresponds to Systems 13 and 14:

$$\begin{aligned}\dot{e}_1 &= -ul_2 n_2 + l_2^2 - m_2 - al_1 + am_1 + r_1(t)\\ \dot{e}_2 &= -l_2 n_2 + v + cm_1 - l_1 n_1 - l_1 + r_2(t)\\ \dot{e}_3 &= l_2 m_2 - wn_2 - bn_1 + l_1 m_1 + r_3(t).\end{aligned} \tag{6.4}$$

We define active control functions $r_1(t), r_2(t)$ and $r_3(t)$ as follows

$$\begin{aligned}r_1(t) &= s_1(t) + ul_2 n_2 - l_2^2 - (1+\alpha)m_1 + al_1\\ r_2(t) &= s_2(t) + l_2 n_2 - v + l_1 + l_1 n_1 - (c+1)m_1 - m_2\\ r_3(t) &= s_3(t) - l_2 m_2 + wn_2 + bn_1 - l_1 m_1 - n_1 - n_2.\end{aligned} \tag{6.5}$$

Where, $s_1(t), s_2(t)$ and $s_3(t)$ are control inputs defined as a function of error state variables e_1, e_2 and e_3. Using System 15, System 16 reduces to the following form:

$$\begin{aligned}\dot{e}_1 &= -e_2 + s_1(t)\\ \dot{e}_2 &= -e_2 + s_2(t)\\ \dot{e}_3 &= -e_3 + s_3(t).\end{aligned} \tag{6.6}$$

The error System 18 to be controlled is a linear system with control inputs $r_1(t), r_2(t)$ and $r_3(t)$. When the system 16 is stabilised by control inputs $s_1(t)$, $s_2(t)$ and $s_3(t)$, then e_1, e_2, and e_3 will converge to zero as time $t \to \infty$, which implies that Systems 2 and 3 are Anti synchronized. To achieve this goal, consider the Lyapunov function defined by

$$W = \frac{1}{2}\left(e_1^2 + e_2^2 + e_3^2\right) \tag{6.7}$$

Differentiating V along the trajectories of the closed-loop error dynamics, we get

$$\begin{aligned}\dot{W} &= e_1\dot{e_1} + e_2\dot{e_2} + e_3\dot{e_3} \\ &= e_1\left(-e_2 + s_1(t)\right) + e_2\left(-e_2 + s_2(t)\right) + e_3\left(-e_3 + s_3(t)\right) \\ &= -e_1e_2 - {e_2}^2 - {e_3}^2 + e_1s_1(t) + e_2s_2(t) + e_3s_3(t)\end{aligned} \tag{6.8}$$

Where control inputs $s_1(t), s_2(t)$ and $s_3(t)$ are functions of e_1, e_2 and e_3, chosen as

$$\begin{bmatrix} s_1(t) \\ s_2(t) \\ s_3(t) \end{bmatrix} = C \begin{bmatrix} e_1 \\ e_2 \\ e_3 \end{bmatrix} \tag{6.9}$$

Where C is 3×3 constant matrix to be determined, with all of its eigenvalues is negative, and $\dot{W}$ is negative semi-definite. We have determined the matrix C in the following form:

$$C = \begin{bmatrix} -1 & 1 & 0 \\ 0 & -1 & 0 \\ 0 & 0 & -1 \end{bmatrix} \tag{6.10}$$

Using (21) and (24), we have

$$\begin{aligned}\dot{W} &= -e_1e_2 - e_2^2 - e_3^2 - e_1^2 + e_1e_2 - e_2^2 - e_3^2 \\ &= -e_1^2 - 2e_2^2 - 2e_3^2\end{aligned} \tag{6.11}$$

This will lead to $\lim_{n\to\infty} \|e(t)\| = 0$, using Lyapunov stability theory. Hence, the antisynchronization between Systems 2 and 3 is achieved.

6.1. Numerical simulation of anti-synchronization

The analysis of the numerical study is carried out using Mathematica. We have considered the parameters of the drive System (13) and response System (14) as $a = 36, b = 10, c = 16, \alpha = 2$ and $u = 5, v = 27, w = 2$. Furthermore, we set the initial conditions of Systems (4) and (5) as $l_l(0) = 50, m_l(0) = 10, n_l(0) = 10$ and $l_2(0) = 3, m_2(0) = 2, n_2(0) = 1$. As a result, the initial states of the error system (13) are $e_2(0) = -47, e_2(0) = -8$, and $e_3(0) = -9$. Figures 7, 8 and 9 depict the dynamics of anti-synchronization for the state variables for Systems (13) and (14) under Control Law 17. In Figure 10, it is observed that the anti-synchronization errors converge rapidly to zero as time approaches infinity.

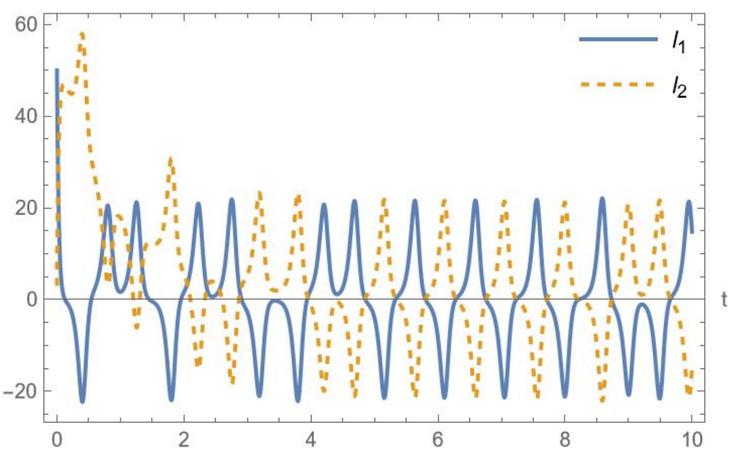

Figure 9: Controller impact on time series of l_1 and l_2.

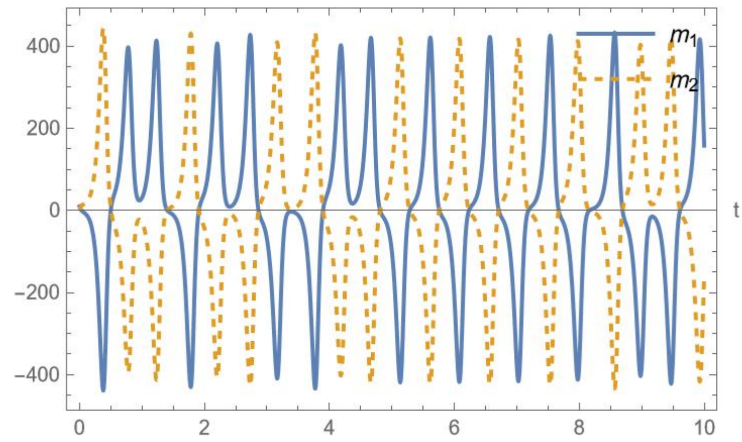

Figure 10: Controller impact on time series of m_1 and m_2.

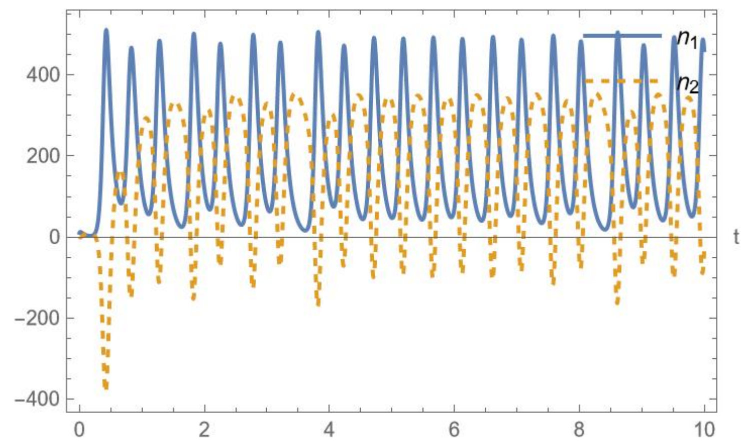

Figure 11: Controller impact on time series of m_1 and m_2.

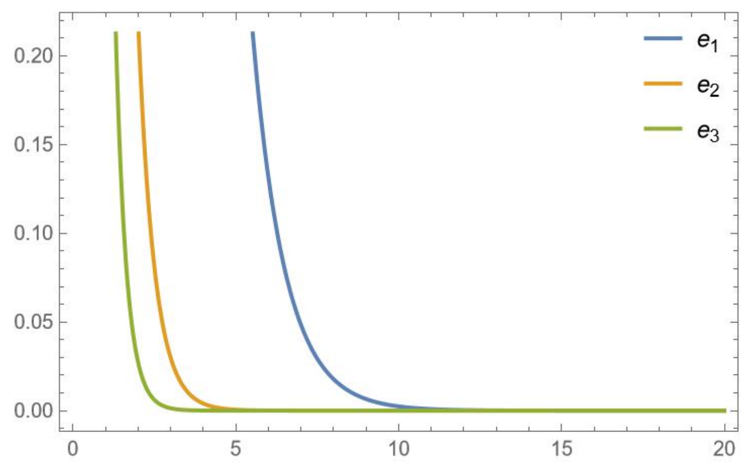

Figure 12: Convergence of error for anti-synchronization.

7. Conclusion

In this paper we established that temperature fluctuations, when strategically harnessed through active control techniques such as synchronization and anti-synchronization, can effectively regulate the proliferation of cancer cells. The implementation of nonlinear controllers provides a powerful framework for influencing tumour dynamics, offering a novel perspective for oncological interventions. Furthermore, this work breaks new ground by proposing the role of microbial populations as mediators of environmental temperature variations, thereby opening avenues for integrating ecological and biomedical systems. The findings not only enhance our understanding of tumour-environment interactions but also lay the foundation for innovative therapeutic strategies and healthcare policies grounded in climate-responsive biological control.

- **Author Contributions**: All authors contributed equally.

- **Conflict of Interest**: The authors declare no conflicts of interest.

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