

Mathematical analysis of complex biological systems using dynamical models

S. Pugazhendhi [§]

Department of Pharmacology

Meenakshi College of Pharmacy

Meenakshi Academy of Higher Education & Research

Chennai 600087

Tamil Nadu

India

Neha Rana [†]

School of Pharmacy

Noida International University

Greater Noida 201312

Uttar Pradesh

India

Chidambar Jalkute ^{*}

Department of Microbiology

Krishna Institute of Science and Technology

Krishna Vishwa Vidyapeeth (Deemed to be University)

Karad 415539

Maharashtra

India

Vikas Dilliwar [‡]

Department of Computer Science and Engineering

Rungta College of Engineering and Technology

Rungta International Skills University

Bhilai 490024

Chhattisgarh

India

[§] E-mail: pugazh@maher.ac.in

[†] E-mail: neha.rana@niu.edu.in

^{*} E-mail: chidujalkute@gmail.com (Corresponding Author)

[‡] E-mail: vikas.dilliwar@rungta.org

Kapil Mundada [@]

*Department of Instrumentation Engineering
Vishwakarma Institute of Technology
Pune 411037
Maharashtra
India*

Ayush Gandhi [#]

*Centre for Research Impact and Outcome (CRIO)
Chitkara University
Rajpura 140401
Punjab
India*

M. Gayathri ^{\$}

*Department of Management Studies
Meenakshi College of Arts and Science
Meenakshi Academy of Higher Education and Research
Chennai 600087
Tamil Nadu
India*

Abstract

The mathematical modelling of biological systems with multiple components provides a systematic solution to the study of the nonlinear interactions, regulatory mechanisms and dynamic behaviours of those multiple components. The aim in this study is to build the approach of dynamical modeling using ordinary differential equations to simulate the fact of time-varying biological populations, molecular concentrations and regulatory processes. The biological assumptions and initial conditions are specified to set realistic constraints on a system, while the nonlinear interaction functions represent feedback, competition, cooperation, saturation and regulatory effects. The obtained differential equation system is analyzed by considering the equilibrium, stability analysis and numerical simulation methods. A proper numerical solution method is used to determine system trajectories for various parameter and initial conditions. The capability of the proposed model to describe steady states, transient responses, oscillatory behaviour and sensitivity to the biological parameters is also illustrated by the simulation results.

Subject Classification: 34C28.

Keywords: *Dynamical models, Biological systems, Ordinary differential equations, Nonlinear interactions, Stability analysis, Numerical simulation.*

[@] E-mail: kapil.mundada@vit.edu

[#] E-mail: ayush.gandhi.orp@chitkara.edu.in

^{\$} E-mail: gayathrimba@maher.ac.in

1. Introduction

The behaviour of complex biological systems is the result of the interactions of their individual parts, which are nonlinear, nonlinear feedback, the environment and time-dependent regulatory processes. These systems are crucial to the understanding of many biological phenomena, such as population growth, disease dynamics, ecological competition, cellular regulation, biochemical reactions, and physiological dynamics [1, 2]. Experimental investigations offer important observations, but can be costly, time consuming, and limited in the amount of information they can provide in order to predict system behavior when conditions change. Mathematical modelling is therefore a useful tool for modelling biological processes mathematically, and understanding their dynamic characteristics [3, 4]. A dynamical model is a model that describes the changes in biological variables over time, especially those that are formulated as ordinary differential equations. These models allow one to simulate a combination of growth, decay, interaction, competition, regulation and saturation in a coherent mathematical framework [5, 6]. Dynamical models can also be used to uncover key relationships that are not directly seen in the experiments through analysis of equilibrium, stability analysis, parameter sensitivity and numerical simulation [7]. Non-linear formulations are particularly important as biological interactions are not necessarily proportional and are often threshold dependent, oscillating, with multiple equilibria, and with complex transitions [8, 9, 10]. This study introduces a mathematical model to analyze complex biological systems using dynamical models. An ordinary differential equation (ODE) formulation is developed, systematically including biological assumptions, initial conditions, interaction mechanisms and feedback mechanisms that are nonlinear [11, 12, 13].

2. Biological Assumptions and Initial Conditions

The biological system is taken to be a collection of interacting populations that is bounded, has continuous time evolution, positive initial states, bounded growth, environmental stability and biologically meaningful parameter values [14].

The Figure 1 shows biological assumptions, which are connected to the population, the resources, the environment, the interactions, the growth, and the limitations among each other and are linked to parameter sets in the initial condition and proposed for systematic modeling of a complex dynamical system.

$$X(t) = [X^1(t), X^2(t), \dots, X_n(t)]^T, X_i(t) \geq 0 \quad (1)$$

The biological system is described as a multi-component state vector, as in equation (1), and each component is a population, a concentration, a resource or a regulatory component that varies over time.

$$X(0) = X^0 = (X^{10}, X^{20}, \dots, X_n^0)^T, X_i^0 > 0 \quad (2)$$

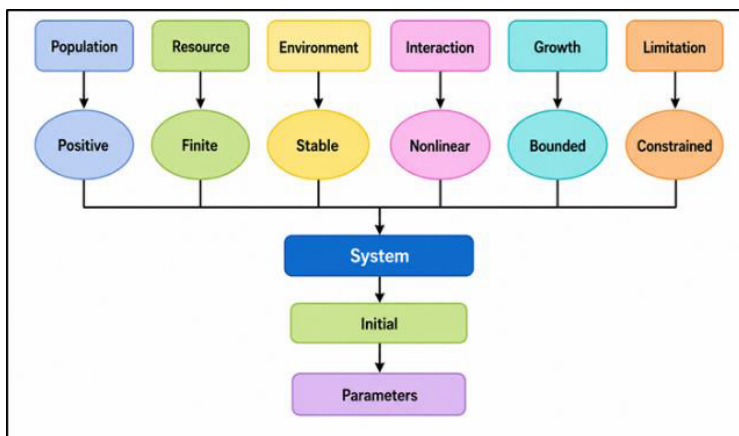


Figure 1

Biological Assumptions and Initial Conditions Framework for Complex Dynamical System Modeling

The initial conditions for all biological parameters are positive in Equation (2), which implies that the initial populations and molecular concentration are physically meaningful and will not give negative populations or molecular concentrations in the subsequent simulations.

$$K_i(t) = K_i^0 \left[1 + \frac{\alpha_i R(t)^{m_i}}{\theta_i^{m_i} + R(t)^{m_i}} \right] e^{-\delta_i t} \quad (3)$$

Dynamic carrying capacity (3) is a resource-dependent quantity that is affected by saturation and temporal decay and thus can be used as an environmental resource availability to control sustainable biological population levels.

$$r_i(t) = r_i^0 \exp \left[-\frac{(E(t) - E_i^*)^2}{2\sigma_i^2} \right] [1 + \eta_i \sin(\omega_i t)] \quad (4)$$

Equation (4) accounts for environmental deviations from the intrinsic biological growth and periodic fluctuations to the system, which capture the dynamic interaction between environmental changes and growth rates and temporal system behaviour.

$$G_i(X, t) = r_i(t) \left[1 - \frac{X_i}{K_i(t)} \right] - \sum_{j=1, j \neq i \text{ to } n} \left[\frac{\beta_{ij} X_j^{p_{ij}}}{\kappa_{ij}^{p_{ij}} + X_j^{p_{ij}}} \right] \quad (5)$$

The coupled expression for the competition/reaction limited intrinsic growth rate and nonlinear intercomponent interaction in equation (5) is a representation of the way competing or regulatory biological components influence effective growth as a result of concentration dependent saturation mechanisms with time.

$$\Omega = \{X \in R^{+n} : 0 \leq X_i(t) \leq K_i(t), \sum_{i=1 \text{ to } n} w_i X_i(t) \leq Rmax\} \quad (6)$$

3. Mathematical Formulation of System Interactions

The interactions among systems are mathematically modeled using coupled variables that describe growth, inhibition, competition, cooperation and regulatory relationships that describe temporal dynamics of interacting biological components.

$$\frac{dX_i}{dt} = F_i(X, t; \theta), i = 1, 2, \dots, n \quad (7)$$

Each biological component evolves in time as described by equation (7) which is a coupled nonlinear function of system states, time and parameters representing the biological interactions and dynamics.

$$F_i^g = r_i X_i \left[1 - \left(\frac{X_i}{K_i} \right)^{q_i} \right] \quad (8)$$

Equation (8) is an intrinsic nonlinear growth model, with the exponent measuring the strength of nonlinearity and the rate of its reduction at the approach of biological saturation.

$$F_i^c = -X_i \sum_{j=1, j \neq i}^n \left[\frac{\alpha_{ij} X_j^{m_{ij}}}{K_{ij}^{m_{ij}} + X_j^{m_{ij}}} \right] \quad (9)$$

In the case of competition this is described by the nonlinear saturation of equation (9), which initially allows the abundance of the competitor to decrease the growth of the biological state variable, but as the competitor continues to increase in abundance the growth of the biological state variable will progressively decrease over time.

$$F_i^m = \sum_{j=1, j \neq i}^n \left[\frac{\gamma_{ij} X_i^{a_{ij}} X_j^{b_{ij}}}{1 + \lambda_{ij} X_i^{a_{ij}} + \mu_{ij} X_j^{b_{ij}}} \right] \quad (10)$$

The nonlinear coupling of the denominator terms in equation (10) can be considered as a model of cooperative interactions between the biological components, where excessive interactions are limited by the denominator terms, and where the interaction strengths remain biologically realistic.

$$F_i^N = \sum_{j=1}^n A_{ij} \left[\frac{\rho_{ij} X_j^{h_{ij}}}{\theta_{ij}^{h_{ij}} + X_j^{h_{ij}}} - \xi_{ij} X_i X_j \right] \quad (11)$$

Network connectivity is added to the biological interactions in Equation (11) where complex communication among coupled biological components of the system is captured by a combination of saturating positive regulation and direct negative coupling.

$$\frac{dX_i}{dt} = F_i^g + F_i^c + F_i^m + F_i^N - \delta_i X_i \quad (12)$$

Combined (12) all of the above effects into a single dynamic equation, and gave a complete picture of how temporal evolution and regulation occur within a biological system.

4. Nonlinear Interaction and Regulatory Functions

These saturation, feedback, threshold response, competitive and regulatory mechanisms can be incorporated into nonlinear functions, and a

realistic representation of the complex biological behaviour under varying dynamic conditions can be achieved.

$$H_i^+(X_j) = \frac{X_j^{n_{ij}}}{K_{ij}^{n_{ij}} + X_j^{n_{ij}}} \quad (13)$$

The biological activation is nonlinear (13) with progressive increase of system response to the increase of the regulator concentration, up to the limits of saturation, set by two parameters: the threshold and the Hill coefficient in biological processes.

$$H_i^-(X_j) = \frac{K_{ij}^{m_{ij}}}{K_{ij}^{m_{ij}} + X_j^{m_{ij}}} \quad (14)$$

The parameters of threshold and Hill coefficient control the sensitivity, steepness and saturation behaviour of the inhibition, and equation (14) describes nonlinear inhibitory regulation, which is the gradual decrease of biological activity with increasing concentration of the regulator.

$$S_{ij}(X_j) = \frac{1}{\{1 + \exp[-\lambda_{ij}(X_j - \theta_{ij})]\}} \quad (15)$$

The sigmoidal regulatory response in equation (15) switches between an active state and an inactive state, and has threshold and sensitivity parameters which govern the switching behaviour in response to changes in biological conditions.

$$D_i(t) = \left[\frac{\alpha_i X_i^{p_i(t-\tau_i)}}{\beta_i p_i + X_i^{p_i(t-\tau_i)}} \right] e^{-\mu_i \tau_i} \quad (16)$$

Equation (16) is an explicit model of biological regulation that includes time-delayed feedback, such that previous states of the system affect the response, and includes explicit consideration of nonlinear saturation, delays, and degradation.

$$R_i(X, t) = \frac{\alpha_i R_i(X)}{[1 + \beta_i R_i(X)]} - \gamma_i D_i(t) + \sum_{j=1}^n \omega_{ij} S_{ij}(X_j) \quad (17)$$

It should be noted that equation (17) incorporates both activation and inhibition mechanisms, delayed feedback, and the nonlinear regulatory influences, so that these overlapping control mechanisms can be represented in a single function and used to comprehensively describe the dynamics of interacting control mechanisms in a complex biological system.

5. Numerical Solution Strategy

The coupled mathematical equations are solved numerically in a stable manner by the time-stepping methods and numerical values of initial conditions, parameters and convergence criteria, which provide accurate dynamic simulations systematically.

$$t_k = t^0 + k\Delta t, X_k \approx X(t_k), k = 0, 1, \dots, N \quad (18)$$

The time domain of the continuous simulation interval is broken down into a finite number of time steps in equation (18) so that the biological

state variables can be numerically approximated at the successive time points during the analysis of the simulation.

$$K^1 = \Delta t F(t_k, X_k; \theta) \quad (19)$$

The first Runge-Kutta slope is calculated in equation (19) and gives an initial estimate of the variation of the system over the chosen numerical integration step which is reasonably correct given the current biological state.

$$K^2 = \Delta t F\left(t_k + \frac{\Delta t}{2}, X_k + \frac{K^1}{2}; \theta\right) \quad (20)$$

The second slope is computed at the temporal midpoint using the first slope; this will improve the approximation accuracy, as more changes in the dynamics of the complex biological system are included.

$$K^3 = \Delta t F\left(t_k + \frac{\Delta t}{2}, X_k + \frac{K^2}{2}; \theta\right) \quad (21)$$

The second estimate is used to calculate another midpoint slope in equation (21) to obtain numerical representation of the nonlinear biological variations even more accurately, and the integration error in the temporal state prediction decreases significantly.

$$K_4 = \Delta t F(t_k + \Delta t, X_k + K_3; \theta) \quad (22)$$

The fourth slope at the end point is given in equation (22) based on the third slope, thus providing a description of the behaviour of the system over the entire range of integration before final state computation is performed accurately.

$$X_{k+1} = X_k + \left(\frac{1}{6}\right)(K^1 + 2K^2 + 2K^3 + K^4) \quad (23)$$

The four calculated slopes in the equation (23) are averaged using weighing factors to update the biological state in order to obtain high numerical accuracy, stability and convergence to solve complex nonlinear dynamical equations efficiently.

6. Ordinary Differential Equation-Based Model

The proposed model is a set of coupled ordinary differential equations to quantify the temporal variation, interaction rates, regulatory influences, and state transitions of the biological system dynamically.

$$\frac{dX}{dt} = r_x X \left(1 - \frac{X}{K_x}\right) - \frac{\alpha XY^m}{\kappa_x^m + Y^m} + \frac{\rho X Z^p}{\theta Z^p + Z^p} \quad (24)$$

The logistic growth, inhibitory interaction, and molecular stimulation terms in equation (24) models how a population of organisms evolves in time as a result of the effects of resource limitation and regulation.

$$\frac{dY}{dt} = r_y Y \left[1 - \frac{Y + \eta X}{K_y}\right] + \frac{\beta X^n Y}{\kappa_y^n + X^n} - \delta_y Y \quad (25)$$

Equation (25) models regulatory populations as density dependent growth, interacting with other biological populations, undergoing nonlinear

activation, and natural degradation, which are all dynamically captured by the equation.

$$\frac{dZ}{dt} = \frac{\sigma X^q}{KZ^q + X^q} - \frac{\mu Y^{sZ}}{\theta_Y^s + Y^s} - \delta Z \quad (26)$$

Equation (26) is a model of molecular regulator dynamics with nonlinear production, interaction-dependent consumption and degradation that accounts for the change in biochemical concentrations over time in response to the biological and regulatory population states.

$$\frac{dR}{dt} = \Lambda - \omega R - \frac{\gamma_x RX}{KRx + R} - \frac{\gamma_y RY}{KRy + R} \quad (27)$$

Relationship (27) describes the continuous availability of the resources in relation to their natural depletion and the consuming activity of interacting populations in a nonlinear way, which allows for realistic estimation of resource limitation impacting biological system development and stability.

$$\frac{dE}{dt} = \frac{\alpha E(X^u + Y^v)}{KE^u + X^u + Y^v} - \beta EE + \frac{\chi ZR}{1 + \lambda ZR} \quad (28)$$

The biological populations give feedback to the environment, as do the natural environmental processes, and also there are nonlinear interactions between the resources/regulators and the environment, which are modeled by equation (28), thus capturing the reciprocity between the environmental conditions and the biological processes.

7. Result and Discussion

Equilibrium behaviour, transient responses, nonlinear variations and parameter dependent behaviours are shown in simulation results, reflecting the ability of the model to capture the complex behaviour of the biological interaction in an effective way.

Table 1
Equilibrium and Stability Analysis Under Different Parameter Conditions

Condition	Growth Rate (r_X)	Interaction (α)	Regulation (β)	Equilibrium X^*	Equilibrium Y^*
C1	0.20	0.10	0.15	61.42	32.18
C2	0.25	0.15	0.20	68.37	35.74
C3	0.30	0.20	0.25	72.91	38.62
C4	0.35	0.25	0.30	76.84	40.73
C5	0.40	0.30	0.35	79.26	42.51
C6	0.45	0.35	0.40	81.73	44.28

Table 1 shows how the growth, interaction and regulation parameters affect the biological equilibrium states. A higher growth rate will lead to

higher values of X^* as the growth rate is increased from 0.20 to 0.45. Comparative Analysis of Growth Rate, Interaction, and Regulation Parameters Under Different Model Conditions shown in Figure 2 under different model conditions.

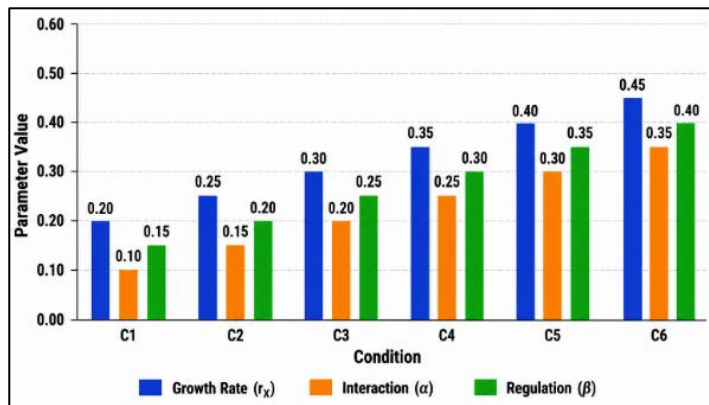


Figure 2
Comparative Analysis of Growth Rate, Interaction, and Regulation Parameters Under Different Model Conditions

Likewise, the value of equilibrium Y^* increases from 32.18 to 44.28. Increasing the biological state levels through stronger growth and interaction mechanisms and maintaining controlled and gradual equilibrium development under various parameter conditions with increasing regulatory strength is suggested by these trends.

8. Conclusion

In this study, a mathematical approach to analyzing complex biological systems with dynamical models constructed of ordinary differential equations (ODE) was presented. The proposed formulation was designed with biologically meaningful assumptions, initial conditions, interaction mechanisms and nonlinear regulatory functions that were used to simulate the temporal changes in interconnected biological processes. Mathematical relationships, that captured important behaviours, involved growth, competition, cooperation, inhibition, saturation, and feedback regulation. Systematic study of the system trajectories under various parameter values and initial conditions was possible with the help of numerical solution strategies. The analysis showed the capability of the dynamical model to determine equilibrium states, transient response, oscillatory behaviour, and sensitivity of biological behaviour to the parameters in the model. Experimental observation alone may not be enough to look at such mechanisms, and mathematical representations help to quantify them.

References

- [1] P. Kraikivski, "Mathematical modeling in systems biology," *Entropy*, vol. 25, no. 10, Art. no. 1380 (2023), doi: 10.3390/e25101380.
- [2] R. López-Ruiz, "Mathematical biology: Modeling, analysis, and simulations," *Mathematics*, vol. 10, no. 20, Art. no. 3892 (2022), doi: 10.3390/math10203892.
- [3] M. Pruthi, A. K. Yadav, and P. Soni, "Quantum synchronizable codes through generalized cyclotomy of order $2r$ over $\mathbb{Z}_{2n}$," *Journal of Interdisciplinary Mathematics*, vol. 29, no. 7, pp. 2205–2216 (2026), doi: 10.47974/JIM-2382.
- [4] A. Farea, O. Yli-Harja, and F. Emmert-Streib, "Using physics-informed neural networks for modeling biological and epidemiological dynamical systems," *Mathematics*, vol. 13, no. 10, Art. no. 1664 (2025), doi: 10.3390/math13101664.
- [5] J. E. Mahbuba and X.-S. Wang, "Stability analysis of biological systems under threshold conditions," *Symmetry*, vol. 17, no. 8, Art. no. 1193 (2025), doi: 10.3390/sym17081193.
- [6] Y. Chen and M. Frenkel-Morgenstern, "Artificial intelligence research for complex biological systems: Integrating data, models, and biological knowledge," *Biology*, vol. 15, no. 14, Art. no. 1137 (2026), doi: 10.3390/biology15141137.
- [7] P. Khobragade, G. Gandal, A. Somatkar, H. M. Panchabudhe, P. P. Ashok, and B. Madaminov, "Heat transfer modeling using fractional differential equations," *Journal of Interdisciplinary Mathematics*, vol. 29, no. 3, pp. 749–756 (2026).
- [8] J. Liu and L. Zhang, "A system dynamics model for simulating the development of postgraduate innovation capacity in smart learning environments," *Mathematics*, vol. 14, no. 9, Art. no. 1460 (2026), doi: 10.3390/math14091460.
- [9] K. G. Tharunraj and P. Ragukumar, "Log-linear algorithm to generate prime trees," *Journal of Information and Optimization Sciences*, vol. 47, no. 7, pp. 2635–2649 (2026), doi: 10.47974/JIOS-2176.
- [10] G. Ben-Mizrahi, "AI-driven cyber threat intelligence frameworks for next-generation network security," *Multidisciplinary Journal of Research in Engineering and Technology*, vol. 13, no. 2, pp. 55–60 (2026).
- [11] A. Lucido, O. Basallo, A. Marin-Sanguino, A. Eleiwa, E. S. Martinez, E. Vilapriño, A. Sorribas, and R. Alves, "Multiscale mathematical modeling in

systems biology: A framework to boost plant synthetic biology,” *Plants*, vol. 14, no. 3, Art. no. 470 (2025), doi: 10.3390/plants14030470.

- [12] M. Shams, N. Kausar, and I. A. Şomîtcă, “Efficient multiplicative calculus-based iterative scheme for nonlinear engineering applications,” *Mathematics*, vol. 12, no. 22, Art. no. 3517 (2024), doi: 10.3390/math12223517.
- [13] M. Nurmhammed, O. Akdağ, and T. Karadağ, “Anatolian caracal optimization algorithm: A three-stage hybrid bio- and physics-inspired framework for optimization problems,” *Mathematics*, vol. 14, no. 16, Art. no. 3007 (2026), doi: 10.3390/math14163007.
- [14] R. C. Poonia, R. Almakki, A. K. J. Saudagar, A. Altameem, and M. Albalthan, “An optimization approach for real-time object detection in IoT devices through edge computing and deep learning,” *Journal of Information and Optimization Sciences*, vol. 45, no. 5, pp. 1465–1475 (2024), doi: 10.47974/JIOS-1768.

Received April, 2026