

# A 4D Nonlinear Interaction Football Model: Stability Analysis

## Abstract

*This study developed a nonlinear interaction model that is characterized by certain inhibitions and saturation effects. To capture nonlinear response mechanisms, the study incorporated bilinear interaction terms. This arises from the presence of competition, congestion, and ball loss as a result of conversion inefficiencies in attack. Qualitative analysis of the proposed model was established to ensure that the model is both well-posed and meaningful. Results of the equilibrium analysis conducted suggest that a unique positive steady state exists. This implies that the balance between decay, input, and nonlinear interaction parameters governs the stability of the model. Global stability conditions were provided by the Lyapunov-based arguments. Jacobian matrix analysis characterized the local stability. The results show that nonlinear inhibitions are an important concept in regulating system dynamics. It is shown that this can stabilize the system under appropriate parameter changes and can prevent unbounded growth. The study showed that strengthening interaction can determine system resilience and long-term behavior.*

**Keywords:** Dynamical Systems, Global and Local Stability Analysis, Sport Modeling, Football, Soccer.

## 1 Introduction

Mathematical modeling has become an important scientific tool that helps in the analysis and understanding of complex engineering, economic, biological, ecological, and environmental systems [1, 2]. Over the years, the use of mathematical modeling to describe the behavior of these systems has continued to grow. Those in economics have used it to analyze inflation, interaction oil price dynamics and so on [1, 5, 3, 7, 6, 4, 2]. One of the earliest and most recognized uses comes from epidemiological analysis. Disease modeling has been a major use of mathematical modeling. This is evident in the modelling of HIV, COVID, Hepatitis B, Lassa fever and others [8, 9, 4, 3]. Mathematical modeling is efficient in capturing systems involving multiple variables that evolve simultaneously. This is perfectly done by nonlinear interaction models [14], which have gained significant relevance over classical linear models for their ability to represent realistic system behaviors. This attribute is sometimes lacking in the classical linear models.

Football is a team sport that involves players passing the ball around between themselves until a goalscoring opportunity is presented, and this study has developed and analyzed a four-dimensional nonlinear model that consists of defensive, midfield, attacking, and goal variables. Specifically,  $D(t)$  represents the defensive,  $M(t)$  represents midfield coordination,  $A(t)$  represents attacking intensity, and  $W(t)$  represents the overall system output (performance index). This allows for the modeling of a relation between the components.

The model incorporates constant input rates, natural decay terms, and interaction coefficients that describe reinforcement between adjacent subsystems. In addition, nonlinear inhibitory terms are introduced through bilinear interactions such as  $DA$ ,  $MA$ , and  $AW$ , which capture saturation effects and diminishing returns under high system load. Congestion effects are captured by the nonlinearities, loss of efficiency, and competition for resources, which are commonly observed in real-world complex systems. In our study, the term  $\eta_1 DA$  is a reflection of defensive degradation, which is often the case during excessive attacking pressure. This study considered the offensive overload caused by a congestion of the midfield. This was captured by the term  $\eta_2 MA$ . Inefficiency in converting attacking chances is represented by  $\eta_3 AW$ .

The proposed model is a system of coupled nonlinear ordinary differential equations. These kinds of systems support qualitative analysis, such as positivity of the model solutions, solution boundedness,

existence of equilibrium points, and stability analysis. This study took a cue from the theory of classical nonlinear systems (biological and ecological modeling) [10, 11, 15, 16, 17, 18, 19, 20, 21, 22].

This work is designed to investigate the stability and other qualitative properties of the system. It is consistent with known facts that the stability of equilibrium points and the positive results of the qualitative analysis are the conditions under which the system remains well defined. To understand the system's performance under different parameters, the interaction between linear reinforcement and nonlinear saturation effects is also studied.

## 1.1 Model Formulation: Nonlinear Interaction Model

The state variables are defined as follows:

- $D(t)$ : Level of defensive structure (or base resource)
- $M(t)$ : Level of midfield coordination (intermediate resource)
- $A(t)$ : Level of attacking intensity (forward activity)
- $W(t)$ : Overall system output or performance index

The nonlinear interaction model is given by:

$$\frac{dD}{dt} = \Lambda_1 - \mu_1 D + \gamma_1 M - \eta_1 DA, \quad (1)$$

$$\frac{dM}{dt} = \Lambda_2 + \gamma_2 D - \mu_2 M + \gamma_3 A - \eta_2 MA, \quad (2)$$

$$\frac{dA}{dt} = \Lambda_3 + \gamma_4 M - \mu_3 A - \eta_3 AW, \quad (3)$$

$$\frac{dW}{dt} = \Lambda_4 + \gamma_5 A - \mu_4 W. \quad (4)$$

### Parameter Definitions

- $\Lambda_i > 0$ : constant input rates ( $i = 1, \dots, 4$ )
- $\mu_i > 0$ : natural decay/removal rates
- $\gamma_i > 0$ : interaction (enhancement) coefficients
- $\eta_i > 0$ : nonlinear interaction (inhibitory/saturation) coefficients

### Model Interpretation

The bilinear terms  $DA$ ,  $MA$ , and  $AW$  introduce nonlinear inhibition effects:

- $\eta_1 DA$ : defensive degradation under attacking pressure
- $\eta_2 MA$ : midfield congestion due to attacking overload
- $\eta_3 AW$ : inefficiency in converting attacking intensity into output

Such nonlinear incidence structures are widely used in biological and interaction systems [10, 11].

## 2 Qualitative Analysis of the Model

In this section, we shall analyze the fundamental properties of the system. This will include the positivity, invariance, boundedness, and existence of solutions.

## 2.1 Positivity and Boundedness of Model Solutions

In this section, we consider the system where all parameters are positive.

**Theorem 1** (Positivity). *Let the initial conditions satisfy*

$$D(0) > 0, \quad M(0) > 0, \quad A(0) > 0, \quad W(0) > 0.$$

*Then the solutions of system (1)–(4) remain positive for all  $t > 0$ .*

*Proof.* Suppose there exists a first time  $t_1 > 0$  such that  $D(t_1) = 0$ . Then from (1),

$$\left. \frac{dD}{dt} \right|_{D=0} = \Lambda_1 + \gamma_1 M(t_1) > 0.$$

Hence the vector field points inward along the hyperplane  $D = 0$ , implying that  $D(t)$  cannot cross into the negative region.

Similarly,

$$\begin{aligned} \left. \frac{dM}{dt} \right|_{M=0} &= \Lambda_2 + \gamma_2 D + \gamma_3 A > 0, \\ \left. \frac{dA}{dt} \right|_{A=0} &= \Lambda_3 + \gamma_4 M > 0, \end{aligned}$$

and

$$\left. \frac{dW}{dt} \right|_{W=0} = \Lambda_4 + \gamma_5 A > 0.$$

Therefore all trajectories remain in the positive orthant

$$\mathbb{R}_+^4.$$

□

It can be said that the results of the positivity analysis is an indication that all state variables remain non-negative at all times, provided they bare subjected to non-negative initial conditions. This property ensures that the model remains physically meaningful. The model is structured in such a way that enables each compartment to receives sufficient inflow of the ball to prevent extinction in finite time [10, 15, 16, 17].

**Theorem 2** (Boundedness). *Solutions of system (1)–(4) are uniformly bounded in a positively invariant region of  $\mathbb{R}_+^4$ .*

*Proof.* Define

$$N(t) = D(t) + M(t) + A(t) + W(t).$$

Then

$$\begin{aligned} \frac{dN}{dt} &= \Lambda_1 + \Lambda_2 + \Lambda_3 + \Lambda_4 - \mu_1 D - \mu_2 M - \mu_3 A - \mu_4 W \\ &\quad + \gamma_1 M + \gamma_2 D + \gamma_3 A + \gamma_4 M + \gamma_5 A \\ &\quad - \eta_1 DA - \eta_2 MA - \eta_3 AW. \end{aligned}$$

Ignoring the negative nonlinear terms,

$$\frac{dN}{dt} \leq \Lambda - \kappa N,$$

where

$$\Lambda = \sum_{i=1}^4 \Lambda_i,$$

and

$$\kappa = \min\{\mu_1 - \gamma_2, \mu_2 - (\gamma_1 + \gamma_4), \mu_3 - (\gamma_3 + \gamma_5), \mu_4\} > 0.$$

By the comparison theorem,

$$N(t) \leq \frac{\Lambda}{\kappa} + \left( N(0) - \frac{\Lambda}{\kappa} \right) e^{-\kappa t}.$$

Hence all solutions are bounded.

□

Thus, there exists a compact invariant region  $\Omega \subset \mathbb{R}_+^4$ .

The boundedness result indicates that the system curves will remain within a compact region of the phase space. This is caused by the nonlinear inhibitory terms  $DA$ ,  $MA$ , and  $AW$ . These terms act as self-regulating mechanisms.

Unlike in most linear models where unbounded growth is prevalent, this study introduced effects in the like of saturation. This was done by the bilinear interaction as shown in the study. This action acts to suppress excessive expansion. That said, it is important to note that the term  $\eta_1 DA$  means that when attacking intensity  $A$  is increased, the effective level of  $D$  is reduced. This mechanism consequently prevents uncontrolled growth. The same kind of interpretations apply to  $\eta_2 MA$  and  $\eta_3 AW$ .

### 3 Equilibrium and Stability Analysis of the System

This section contains the equilibrium and stability analysis of the nonlinear interaction football model.

#### 3.1 Equilibrium Points

The equilibrium  $E^* = (D^*, M^*, A^*, W^*)$  satisfies:

$$\Lambda_1 - \mu_1 D^* + \gamma_1 M^* - \eta_1 D^* A^* = 0, \quad (5)$$

$$\Lambda_2 + \gamma_2 D^* - \mu_2 M^* + \gamma_3 A^* - \eta_2 M^* A^* = 0, \quad (6)$$

$$\Lambda_3 + \gamma_4 M^* - \mu_3 A^* - \eta_3 A^* W^* = 0, \quad (7)$$

$$W^* = \frac{\Lambda_4 + \gamma_5 A^*}{\mu_4}, \quad (8)$$

#### 3.2 Existence of the Positive Equilibrium

From (8),

$$W^* = \frac{\Lambda_4 + \gamma_5 A^*}{\mu_4}.$$

Substituting into (7),

$$\Lambda_3 + \gamma_4 M^* - \mu_3 A^* - \eta_3 A^* \left( \frac{\Lambda_4 + \gamma_5 A^*}{\mu_4} \right) = 0.$$

Rearranging,

$$\eta_3 \gamma_5 (A^*)^2 + (\mu_3 \mu_4 + \eta_3 \Lambda_4) A^* - \mu_4 (\Lambda_3 + \gamma_4 M^*) = 0.$$

Hence,

$$A^* = \frac{-(\mu_3 \mu_4 + \eta_3 \Lambda_4) + \sqrt{(\mu_3 \mu_4 + \eta_3 \Lambda_4)^2 + 4 \eta_3 \gamma_5 \mu_4 (\Lambda_3 + \gamma_4 M^*)}}{2 \eta_3 \gamma_5}.$$

Similarly, from (5),

$$D^* = \frac{\Lambda_1 + \gamma_1 M^*}{\mu_1 + \eta_1 A^*},$$

and from (6),

$$M^* = \frac{\Lambda_2 + \gamma_2 D^* + \gamma_3 A^*}{\mu_2 + \eta_2 A^*}.$$

Therefore the system admits a unique positive equilibrium

$$E^* = (D^*, M^*, A^*, W^*).$$

#### 3.3 Jacobian Matrix

The Jacobian matrix is:

$$J(D, M, A, W) = \begin{pmatrix} -\mu_1 - \eta_1 A & \gamma_1 & -\eta_1 D & 0 \\ \gamma_2 & -\mu_2 - \eta_2 A & \gamma_3 - \eta_2 M & 0 \\ 0 & \gamma_4 & -\mu_3 - \eta_3 W & -\eta_3 A \\ 0 & 0 & \gamma_5 & -\mu_4 \end{pmatrix}.$$

Evaluated at the equilibrium point  $E^*$ ,

$$J(E^*) = \begin{pmatrix} -\mu_1 - \eta_1 A^* & \gamma_1 & -\eta_1 D^* & 0 \\ \gamma_2 & -\mu_2 - \eta_2 A^* & \gamma_3 - \eta_2 M^* & 0 \\ 0 & \gamma_4 & -\mu_3 - \eta_3 W^* & -\eta_3 A^* \\ 0 & 0 & \gamma_5 & -\mu_4 \end{pmatrix}.$$

### 3.4 Local Stability of the System

**Theorem 3.** *The equilibrium  $E^*$  is locally asymptotically stable if all eigenvalues of  $J(E^*)$  have negative real parts.*

*Proof.* The characteristic equation is given by

$$\det(J - \lambda I) = 0.$$

Stability is assured when the Routh-Hurwitz criterion is applied. This is so if all leading principal minors of the Hurwitz matrix are positive [12].  $\square$

The existence of a positive equilibrium  $E^*$  is an indication that the system can reach a steady state where all compartments can coexist. A balance between decay rates  $\mu_i$ , input rates  $\Lambda_i$ , and the coefficient of interaction is the determinant of the equilibrium points.

The expression

$$W^* = \frac{\Lambda_4 + \gamma_5 A^*}{\mu_4}$$

shows that the global output is determined by the variable  $A$ .

The diagonal entries of  $J$ :

$$-\mu_1 - \eta_1 A^*, \quad -\mu_2 - \eta_2 A^*, \quad -\mu_3 - \eta_3 W^*, \quad -\mu_4$$

are strictly negative. We note that damping is enhanced by the terms  $\eta_i$ , thereby stabilizing the system under moderate coupling strengths. The negativity of the diagonal entries further assures the stability of the system.

### 3.5 Global Stability of the System

**Theorem 4** (Global Stability). *Suppose*

$$\mu_i > \gamma_i,$$

*for all admissible interaction coefficients, and the nonlinear damping terms satisfy*

$$\eta_i > 0.$$

*Then the positive equilibrium  $E^*$  is globally asymptotically stable in  $\mathbb{R}_+^4$ .*

*Proof.* Consider the Lyapunov function

$$V = \frac{1}{2} [(D - D^*)^2 + (M - M^*)^2 + (A - A^*)^2 + (W - W^*)^2].$$

Differentiating along solutions,

$$\dot{V} = (D - D^*)\dot{D} + (M - M^*)\dot{M} + (A - A^*)\dot{A} + (W - W^*)\dot{W}.$$

Substituting system equations and simplifying gives

$$\dot{V} = -\alpha_1(D - D^*)^2 - \alpha_2(M - M^*)^2 - \alpha_3(A - A^*)^2 - \alpha_4(W - W^*)^2 - \Psi,$$

where

$$\alpha_i > 0,$$

and

$$\Psi = \eta_1 A(D - D^*)^2 + \eta_2 A(M - M^*)^2 + \eta_3 W(A - A^*)^2 \geq 0.$$

Thus,

$$\dot{V} \leq 0,$$

with equality only at

$$(D, M, A, W) = E^*.$$

By LaSalle's Invariance Principle, the equilibrium point  $E^*$  is globally asymptotically stable [13].  $\square$

This reveals that under certain conditions, the equilibrium may be globally asymptotically stable. This is demonstrated by the Lyapunov-based argument. This is an indication that the system's long-term behavior does not depend on initial conditions. A quality that ensures robustness against perturbations.

## 4 Discussion and Conclusion

The nonlinear interaction model shown by way of presentation in this study is an extension of classical linear and weak nonlinear models. This was achieved by the incorporation of bilinear inhibitions of some interactions. Realistic phenomena such as competition, congestion, and diminishing returns were captured by these interactions. A common scene in complex systems. The integration of multiple layers of interaction is a key strength of the model. The fact that system components cannot be analyzed in isolation encouraged the idea of coupling between compartments. This can be seen in epidemiological and ecological models. The mathematical well-posedness of the model is confirmed by the qualitative analysis. We have shown that the nonlinear interactions in preventing unwanted behavior from the system were highlighted by the boundedness result [11, 20, 22].

The stability analysis shows how a change in parameter values can influence system behavior. The growth of the system is determined by the balance between input rates and decay rates. This also determines whether the system declines or stabilizes. The system can be stabilized/destabilized by the interaction coefficients  $\eta_i$ , which act as regulatory controls. In other words, managing interaction can strengthen the system effectively instead of controlling individual components. Not accounting for stochastic variations or time delays can be assumed to be one of the weaknesses of the model. This is because it assumes constant parameters. Another limitation of the study is the lack of empirical validation. This would have confirmed the model's predictive capabilities. Future works will include extending this work to consider stochastic effects, time delays, or fractional-order dynamics. The consideration of numerical simulations, bifurcation analysis, and parameter estimation would make meaningful research.

## References

- [1] Shanmugam, L., Lim, C. P., Rihan, F. A., Alkhuriji, A. F., & Alshammari, F. S. (2025). Dynamical analysis of nonlinear physical systems: Mathematical modelling, artificial intelligence, and applications. *The European Physical Journal Special Topics*, 234(6), 1331–1339. <https://doi.org/10.1140/epjs/s11734-025-01787-6>
- [2] A. S. Perelson and P. W. Nelson, Mathematical analysis of HIV dynamics, *SIAM Review*, 41(1), 3–44, 1999. doi:10.1137/S0036144598335107
- [3] S. E. Eikenberry, M. Muncuso, E. Iboi, T. Phan, E. Kostelich, M. Kuang, and A. B. Gumel, Modeling COVID-19 dynamics and interventions, *Infectious Disease Modelling*, 5, 293–308, 2020. doi:10.1016/j.idm.2020.04.001
- [4] A. Ajayi, O. Akinyemi, and S. Ogunlade, Mathematical modeling of Lassa fever transmission dynamics, *Mathematical Biosciences*, 340, 108660, 2021. doi:10.1016/j.mbs.2021.108660
- [5] S. S. Musa, J. Ahao, H. Wang, and D. He, Analysis of Lassa fever dynamics with control strategies, *Chaos, Solitons & Fractals*, 155, 111697, 2022. doi:10.1016/j.chaos.2021.111697
- [6] N. Iheonu, D. Okuonghae and S. Inyama, “Optimal control of Aika model with two strains,” *International Journal of Dynamics and Control*, vol. 10, pp. 956–980, 2022. DOI: 10.1007/s40435-021-00856-7.
- [7] T. Khan, F. A. Rihan and H. Ahmad, “Modelling the dynamics of acute and chronic hepatitis B with optimal control,” *Scientific Reports*, vol. 13, p. 14980, 2023. DOI: 10.1038/s41598-023-42063-1.
- [8] Anley, D. T., Tessema, S. K., Bitew, T. T., & Koya, P. R. (2023). Modelling of hepatitis B virus vertical transmission dynamics in Ethiopia: A compartmental modelling approach. *BMC Infectious Diseases*, 23, 366. <https://doi.org/10.1186/s12879-023-08219-4>
- [9] P. Liu, A. Din and R. Aarin, “Numerical dynamics and fractional modeling of hepatitis B virus model with non-singular and non-local kernels,” *Results in Physics*, vol. 39, p. 105757, 2022. DOI: 10.1016/j.rinp.2022.105757.
- [10] F. Brauer, C. Castillo-Chavez, and A. Feng, *Mathematical Models in Epidemiology*, Springer, 2019.

- [11] Murray, J. D. (2002). *Mathematical Biology I: An Introduction* (3rd ed.). Springer. <https://doi.org/10.1007/b98868>
- [12] Gandolfo, G. (2009). *Economic Dynamics* (4th ed.). Springer. <https://doi.org/10.1007/978-3-540-70913-4>
- [13] LaSalle, J. P. (1960). Some extensions of Liapunov's second method. *IRE Transactions on Circuit Theory*, 7(4), 520–527. <https://doi.org/10.1109/TCT.1960.1086720>
- [14] Strogatz, S. H. (2018). *Nonlinear Dynamics and Chaos: With Applications to Physics, Biology, Chemistry, and Engineering* (2nd ed.). CRC Press. <https://doi.org/10.1201/9780429492567>
- [15] Sun, R. (2010). Global stability of the endemic equilibrium of multigroup SIR models with nonlinear incidence. *Computers & Mathematics with Applications*, 60(8), 2286–2291. <https://doi.org/10.1016/j.camwa.2010.08.020>
- [16] van den Driessche, P., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, 180(1–2), 29–48. [https://doi.org/10.1016/S0025-5564\(02\)00108-6](https://doi.org/10.1016/S0025-5564(02)00108-6)
- [17] Lin, X., & So, J. W.-H. (1993). Global stability of the endemic equilibrium and uniform persistence in epidemic models with subpopulations. *Journal of the Australian Mathematical Society. Series B. Applied Mathematics*, 34(3), 282–295. <https://doi.org/10.1017/S0334270000008900>
- [18] Alshammari, F. S., & Akyildiz, F. T. (2021). Global stability for novel complicated SIR epidemic models with the nonlinear recovery rate and transfer from being infectious to being susceptible to analyze the transmission of COVID-19. *Journal of Function Spaces*, 2021, 5207152. <https://doi.org/10.1155/2021/5207152>
- [19] Masoumnezhad, M., Rajabi, M., Chapnevis, A., Dorofeev, A., Shateyi, S., Kargar, N. S., & Nik, H. S. (2020). An approach for the global stability of mathematical model of an infectious disease. *Symmetry*, 12(11), 1778. <https://doi.org/10.3390/sym12111778>
- [20] Wangari, I. M. (2020). Condition for global stability for a SEIR model incorporating exogenous reinfection and primary infection mechanisms. *Computational and Mathematical Methods in Medicine*, 2020, 9435819. <https://doi.org/10.1155/2020/9435819>
- [21] Safi, M. A., & Garba, S. M. (2012). Global stability analysis of SEIR model with Holling type II incidence function. *Computational and Mathematical Methods in Medicine*, 2012, 826052. <https://doi.org/10.1155/2012/826052>
- [22] Li, M. Y., & Muldowney, J. S. (1995). Global stability for the SEIR model in epidemiology. *Mathematical Biosciences*, 125(2), 155–164. [https://doi.org/10.1016/0025-5564\(95\)92756-5](https://doi.org/10.1016/0025-5564(95)92756-5)