

Threshold Dynamics, Backward Bifurcation, And Sensitivity Analysis in An Adaptive Behavioral-Immune Feedback Epidemic Model

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ABSTRACT: Mathematical modeling of infectious disease spread mechanisms has so far considered behavior adaptation and immunization processes separately from one another, making these models unable to explain some important epidemiological effects, such as waves of epidemics, shift in thresholds and multi-stability. We propose here an innovative approach in form of a new Adaptive Immune-Behavioral (AIB) Feedback Model, which couples the process of behavioral adaptation with the process of acquiring immunity. Our main contribution is the use of adaptive feedback in the sense that the value of transmission rate is adaptive and depends on the actual values of prevalence and immunity-induced protection. A careful analysis of qualitative properties of the proposed model is conducted, which includes proof of positivity and boundedness of all solutions, calculation of the basic reproductive number, as well as finding disease-free and endemic equilibrium points. Local stability of these equilibria is obtained via analysis of Jacobian matrix, whereas global stability is obtained under certain parametric restrictions. In order to verify the practical relevance of the proposed model, we conduct numerous numerical simulations in a broad range of parameters. It turns out that the inclusion of the behavioral feedback produces highly interesting nonlinear effects such as bistability, hysteresis, and the phenomenon of epidemic resurgence—which do not occur in classical models where behavioral-immunity feedback is ignored. The numerical results show that in the proposed model the feedback adaptation can result in existence of multiple stable equilibria with the same parameters, hence, proving the key importance of the choice of initial conditions in the process of epidemics.

Keywords: Behavioral response dynamics, Basic reproduction number, Backward bifurcation, Slow-Fast dynamics, Sensitivity analysis.



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1. INTRODUCTION

Mathematical modeling has emerged as a critical approach in understanding the dynamics of infectious diseases and supporting decision-making during a pandemic outbreak. Classical epidemic models, such as the SIR model and its variants, have served as a rigorous framework for investigating infectious diseases and determining epidemic thresholds [1, 2]. However, many traditional epidemic models are based on the assumption that the parameter for disease transmission remains constant during the epidemic period. This assumption has been a limitation in using such models, considering that disease transmission is significantly affected by adaptive immunity and behavioral responses, which are dynamic processes during an epidemic period [3, 4].

In large pandemics like COVID-19, it has been realized that human behavior plays a vital role in determining the pattern of disease transmission. Humans tend to modify their contact rates based on the perceived risk of infection, vaccination levels, media coverage, and government intervention [5]. However, immune protection, whether from

previous infection or vaccination, also plays a critical role in determining how people respond to epidemic risks. Thus, we can say that epidemic spread is a result of a complex interplay between biological and behavioral processes, and therefore, epidemic dynamics are a result of coupled biological and behavioral interactions [6].

Recent advances in mathematical epidemiology have highlighted the need to incorporate behavioral adaptation into epidemic models through nonlinear incidence, awareness-based contact structures, and risk-based behavioral feedback mechanisms [7]. Several models have been developed, and they incorporate adaptive responses, such as a decrease in infection rates with a corresponding increase in infection prevalence, to account for voluntary actions individuals take to protect themselves from infection. However, most models consider immune and behavioral responses separately, although they are interconnected and affect risk perception and response decisions simultaneously [8]. This has made classical adaptive epidemic models less effective in describing realistic pandemics with features such as oscillatory epidemics, delayed epidemic peaks, and complex persistence patterns [9].

In this regard, the present study aims to introduce a novel Adaptive Immune Behavioral (ABI) feedback model that combines the effects of the immune response and behavioral adaptation into a single nonlinear epidemic model structure. In the proposed model structure, the behavioral responses are not considered to be predetermined or fixed but are rather adaptive responses depending on the severity of the epidemic and the level of protection due to the immune response. This provides a feedback mechanism in the model structure and enables the model to capture realistic adaptive responses during the evolution of the epidemic process. This provides a more realistic model structure in the context of the epidemic process than the traditional epidemic model structures with constant contact structures.

In a mathematical sense, the incorporation of the adaptive immune behavioral feedback will introduce further nonlinear effects into the transmission mechanism, which can potentially influence the epidemic thresholds as well as the stability of the system. In particular, the immune behavioral feedback structures can give rise to new complex dynamical effects, including the existence of multiple equilibrium states, bistable regions, backward bifurcation, as well as sustained oscillatory epidemic waves [10, 11]. Such nonlinear dynamical effects cannot be accounted for in the classical epidemic model, including the bilinear incidence rates, but will emerge from the interplay of the immune protection mechanism and the behavioral adaptation mechanism in controlling the effective contact rates in the disease transmission process [12].

Another significant characteristic that is attributed to adaptive behavioral feedback mechanisms is the capacity to account for the recurring epidemic waves that are often observed during pandemic outbreaks. The epidemic waves are attributed to time-varying behaviors that are influenced by changes in perceived infection risks and immunity levels [13]. The inclusion of these mechanisms is significant for improving the predictive capacity of mathematical models and gaining a better understanding of the interplay between intervention policies and population behaviors. Adaptive immune-behavioral modeling is significant for developing intervention policies that are capable of mitigating disease transmission while maintaining social stability [14].

Inspired by these findings, the main motivation for this research is to propose and analyze a new adaptive epidemic model that we call the “ABI model,” where behavior and immunity are used to jointly govern epidemic transmission dynamics [15]. To this end, the new model is formulated with a nonlinear feedback function that depends on the prevalence of the epidemic and the levels of immunity protection to adaptively change the rate of disease transmission. The new structure is flexible to allow for the analysis of threshold and stability changes [16].

The mathematical analysis described in this study is primarily aimed at understanding the qualitative dynamical characteristics of the described model, such as the existence and stability of equilibrium points, the calculation of the basic reproduction number that dictates the persistence criteria for epidemics, and the role of adaptive immune-behavioral feedback on global epidemics [17]. Moreover, this approach also offers a means to understand the role of nonlinear feedback mechanisms on epidemic control and possibly the emergence of complex bifurcation scenarios that dictate different regimes such as disease-free, endemic, and oscillatory outbreak scenarios [18]. In [19], the authors develop and analyze the mathematical model of the spread of awareness among the host population, and then relate this model to an epidemiological model through reduced host susceptibility. The authors in [20], employ an epidemiological-economic model of epidemic dynamics to capture the trade-offs underlying person-to-person contact decisions. The results show that the inclusion of adaptive human behavior can alter the course of epidemics, and that the inclusion is important in parameter estimation and interpretation, as well as the development of social distancing strategies. The model proposed in [21], enables the capture of time-varying epidemics through the level of “alarm” in the population, with alarm being

defined as a function of the previous epidemic trajectory. The authors examine the stability of the population alarm over a broad range of epidemic scenarios, using parametric functions as well as non-parametric functions involving splines and Gaussian processes. The authors in [22], systematically compare the performance of three mechanistic behavioral epidemic models across nine geographies and two modeling tasks during the first wave of COVID-19, utilizing a range of metrics. The authors in article [23], examine a susceptible, infected, and recovered epidemic model, where the epidemiologic perception of the environment can adapt the behavior of susceptible individuals to preventive behavior. Two rules, depending on local and global epidemic prevalence, for the spread of the epidemic in heterogeneous networks are examined. In [24], the authors examine ideas from behavioral epidemiology embedded with a vaccination game and a pairwise two-player two-strategy game to represent the environmental feedback in an SVIR model, utilizing a composite information index including disease incidence, vaccine factors, and cooperative behavior on a global time scale (repeated season). The authors in [25] theoretically explore the cyclic SVITR epidemic model framework for disease-free and endemic equilibrium to establish stability conditions. Thereafter, the incorporated vaccination and treatment approaches are introduced based on various aspects of evolutionary game theory for behavior among individuals in society through a preposterous phase diagram. In [26], the authors compare the proposed model with an SEIR model without the inclusion of behavior. In this case, all individuals have permanent immunity to COVID-19. We thus explore a modified version of all models where individuals have partial immunity to the disease, referred to as SEIRS and SEIRsb. The authors in [27] explore the behavior at equilibrium for the epidemic based on various analytical expressions according to epidemic conditions. Moreover, they explore the existence and welfare of equilibrium, establishing a negative externality.

The mathematical analysis presented in the current study is primarily focused on the qualitative dynamical aspects of the proposed model, and we investigate the existence and stability of the equilibrium points, the derivation of the basic reproductive number, and the conditions that affect the global dynamics of the epidemic, as well as the adaptive immunological-behavioral feedback. Moreover, the proposed model provides insights into the nonlinear feedback mechanisms and their implications on the control strategies and the bifurcation phenomena, which could lead to the emergence of complex bifurcation structures.

2. MODEL FORMULATION

A new continuous-time epidemiological model that includes several feedback mechanisms is what we suggest: Classical SEIR dynamics: infectious I , recovered R , exposed E , and susceptible S . Nonlinear cubic feedback associated with behavioral adaptation B . The healthcare stress variable H has an impact on mortality. Saturating the immune system in people who have recovered.

The total population is

$$N(t) = S(t) + E(t) + I(t) + R(t),$$

and we assume standard demographic turnover with birth and natural death.

The model is:

$$\dot{S} = \Lambda - \alpha BS \frac{I}{N} - dS + \omega R, \quad (1)$$

$$\dot{E} = \alpha BS \frac{I}{N} - (\sigma + d)E, \quad (2)$$

$$\dot{I} = \sigma E - (\psi + d + \phi I)I, \quad (3)$$

$$\dot{R} = \psi I - (\omega + d)R + \frac{\kappa IR}{1 + \eta I}, \quad (4)$$

$$\dot{B} = \varepsilon_1 \left(\frac{I}{N} - \theta B \right) - \varepsilon_2 B^3, \quad (5)$$

$$\dot{H} = \delta I - \rho H. \quad (6)$$

2.1 EXPLANATION OF EACH EQUATION

1. Susceptible Dynamics (1):

$$\dot{S} = \Lambda - \alpha BS \frac{I}{N} - dS + \omega R.$$

Λ : recruitment/birth rate of susceptible, α : baseline transmission rate, B : behavioral adaptation factor (reduces or enhances contacts), I/N : prevalence of infection, d : natural death rate and ω : loss of immunity rate (recovered return to susceptible).

This captures classical SEIR transmission modified by behavior.

2. Exposed Dynamics (2):

$$\dot{E} = \alpha BS \frac{I}{N} - (\sigma + d)E.$$

σ : rate of progression from exposed to infectious and d : natural death rate.

Exposed individuals are infected but not yet infectious.

3. Infectious Dynamics (3):

$$\dot{I} = \sigma E - (\psi + d + \phi I)I.$$

ψ : recovery rate, ϕI : density-dependent mortality due to healthcare overload and d : natural death

The term ϕI^2 captures the feedback from healthcare stress, i.e., higher infections increase mortality.

4. Recovered Dynamics (4):

$$\dot{R} = \psi I - (\omega + d)R + \frac{\kappa IR}{1 + \eta I}.$$

κ : immune boosting coefficient and η : saturation parameter for immune boosting.

Recovered individuals can gain additional immunity through interaction with infectious individuals (e.g., subclinical re-exposure).

5. Behavioral Adaptation (5):

$$\dot{B} = \varepsilon_1 \left(\frac{I}{N} - \theta B \right) - \varepsilon_2 B^3.$$

ε_1 : rate of behavioral response, θ : proportionality constant for decay of behavioral effort and ε_2 : nonlinear damping coefficient.

This equation models slow behavioral adaptation with cubic saturation (prevents unbounded behavior changes).

6. Healthcare Stress (6):

$$\dot{H} = \delta I - \rho H.$$

δ : rate at which infectious individuals stress healthcare resources, ρ : recovery/removal rate of healthcare stress (e.g., hospital beds freed) and H affects mortality indirectly through ϕI in the infectious class.

Table 1: Parameter values used for numerical simulations of the proposed epidemic model.

Parameter	Value	Description
Λ	$1.8 \times 10^{-4} \text{ day}^{-1}$	Recruitment/birth rate of population
A	0.0115 day^{-1}	Transmission rate per contact
D	$4.563 \times 10^{-5} \text{ day}^{-1}$	Natural mortality rate
Σ	0.09 day^{-1}	Rate from exposed to infectious
Ψ	0.05 day^{-1}	Recovery rate of infected individuals
Ω	0.01 day^{-1}	Rate of immunity loss that puts healed people back in the vulnerable group
Φ	0.001 day^{-1}	Healthcare stress-dependent mortality coefficient
K	0.1	Immune boosting interaction coefficient between infected and recovered individuals
H	0.5	Saturation of immune boosting
ε_1	0.5	Rate of behavioral adaptation (slow timescale)
θ	0.2	Decay rate of behavioral response
ε_2	0.01	Cubic behavioral damping coefficient
δ	0.2	Rate of healthcare stress accumulation
ρ	0.1	Recovery/removal rate of healthcare stress

2.2 KEY FEATURES OF THE MODEL

1. Multi-scale dynamics: $\varepsilon_1 \ll 1 \rightarrow$ rapid illness progression versus gradual behavioral adaptation.
2. Nonlinear feedbacks: healthcare stress, cubic behavior, and immune boosting.
3. The potential for relaxation oscillations, backward bifurcation, and several endemic equilibria.
4. Accurately depicts sociological and epidemiological relationships.
5. Subject to numerical, slow-fast, and bifurcation studies.

This study makes the following contributions:

- i. Construction of a new Adaptive Immune-Behavioral (AIB) Feedback Model where behavioral adaptation and immunity acquisition evolve as a function of time in one integrated nonlinear system.
- ii. Mathematical derivation of positivity, boundedness, equilibrium analysis, and basic reproductive number of this new model.
- iii. Revealing through numerical simulations of the existence of rich and complex nonlinear behavior including bistability, hysteresis, and re-emergence of epidemics which do not occur in the classical independent-process modeling approach.
- iv. Implications of such findings on designing comprehensive intervention strategies in public health.

3. POSITIVITY OF SOLUTIONS

Theorem 1. Given non-negative initial conditions, all solutions remain non-negative for $t > 0$.

Proof. Consider each equation on the boundary:

- If $S = 0$,

$$\dot{S} = \Lambda + \omega R \geq 0.$$

- If $E = 0$,

$$\dot{E} = \alpha BS \frac{I}{N} \geq 0.$$

- If $I = 0$,

$$\dot{I} = \sigma E \geq 0.$$

- If $R = 0$,

$$\dot{R} = \psi I \geq 0.$$

- If $B = 0$,

$$\dot{B} = \varepsilon_1 \frac{I}{N} \geq 0.$$

- If $H = 0$,

$$\dot{H} = \delta I \geq 0.$$

Thus, the positive orthant is invariant.

4. BOUNDEDNESS AND INVARIANT REGION

Summing the human population equations:

$$\frac{dN}{dt} = \Lambda - dN - \alpha HI.$$

Since $\alpha HI \geq 0$, we obtain:

$$\frac{dN}{dt} \leq \Lambda - dN.$$

Solving:

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

Hence,

$$0 \leq N(t) \leq \frac{\Lambda}{d}.$$

Therefore, the feasible region is:

$$\Omega = \left\{ (S, E, I, R, B, H) \in \mathbb{R}_+^6 : N \leq \frac{\Lambda}{d} \right\}.$$

5. EQUILIBRIUM POINTS

5.1 DISEASE-FREE EQUILIBRIUM (DFE)

Setting $E = I = R = H = 0$, we obtain:

$$S^0 = \frac{\Lambda}{d}, \quad B^0 = 0.$$

Thus,

$$E^0 = \left(\frac{\Lambda}{d}, 0, 0, 0, 0 \right).$$

5.2 ENDEMIC EQUILIBRIUM

The endemic equilibrium

$$E^* = (S^*, E^*, I^*, R^*, B^*, H^*)$$

satisfies

$$S^*, E^*, I^*, R^*, B^*, H^* > 0.$$

Setting the right-hand sides of system (1)-(6) equal to zero where

$$N^* = S^* + E^* + I^* + R^*.$$

From the last equation,

$$H^* = \frac{\delta}{\rho} I^*.$$

Define

$$\phi = \frac{\alpha\delta}{\rho}.$$

Then

$$\psi + d + \alpha H^* = \psi + d + \phi I^*.$$

From the third equation,

$$E^* = \frac{\psi + d + \phi I^*}{\sigma} I^*.$$

From the fourth equation,

$$\psi I^* = R^* \left[(\omega + d) - \frac{\kappa I^*}{1 + \eta I^*} \right].$$

Hence,

$$R^* = \frac{\psi I^*}{(\omega + d) - \frac{\kappa I^*}{1 + \eta I^*}}.$$

Multiply numerator and denominator by $(1 + \eta I^*)$:

$$R^* = \frac{\psi I^* (1 + \eta I^*)}{(\omega + d)(1 + \eta I^*) - \kappa I^*}.$$

From the fifth equation,

$$\varepsilon_1 \left(\frac{I^*}{N^*} - \theta B^* \right) = \varepsilon_2 (B^*)^3.$$

Thus,

$$\frac{I^*}{N^*} = \theta B^* + \frac{\varepsilon_2}{\varepsilon_1} (B^*)^3.$$

Define

$$\psi = \frac{\varepsilon_2}{\varepsilon_1}.$$

Then

$$\frac{I^*}{N^*} = \theta B^* + \psi (B^*)^3.$$

This cubic relation implies possible multiple positive behavioral equilibria.

From the second equation,

$$\alpha B^* S^* \frac{I^*}{N^*} = (\sigma + d) E^*.$$

Substitute E^* :

$$\alpha B^* S^* \frac{I^*}{N^*} = (\sigma + d) \frac{\psi + d + \phi I^*}{\sigma} I^*.$$

Cancel I^* :

$$\alpha B^* S^* \frac{1}{N^*} = \frac{(\sigma + d)(\psi + d + \phi I^*)}{\sigma}.$$

Thus,

$$S^* = \frac{N^*}{\alpha B^*} \frac{(\sigma + d)(\psi + d + \phi I^*)}{\sigma}.$$

Recall

$$N^* = S^* + E^* + I^* + R^*.$$

Substitute the previous expressions:

$$N^* = \frac{N^*}{\alpha B^*} \frac{(\sigma + d)(\psi + d + \phi I^*)}{\sigma} + \frac{\psi + d + \phi I^*}{\sigma} I^* + I^* + R^*.$$

This equation contains N^* on both sides. After simplification, we obtain a nonlinear algebraic equation in I^* :

$$F(I^*) = 0,$$

where F is a high-order nonlinear function due to:

- Immune boosting rational term,
- Cubic behavioral feedback,
- Healthcare-dependent mortality.

Using

$$\frac{I^*}{N^*} = \theta B^* + \psi (B^*)^3,$$

we eliminate N^* and obtain a single scalar equation:

$$I^* = (\theta B^* + \psi(B^*)^3)N^*(I^*).$$

Substituting all expressions yields the polynomial-rational equation of order up in I^* .

The endemic equilibrium I^* satisfies the quartic polynomial:

$$A_4(I^*)^4 + A_3(I^*)^3 + A_2(I^*)^2 + A_1I^* + A_0 = 0,$$

where the coefficients are written to highlight the role of $R_0 - 1$:

$$\begin{aligned} A_4 &= -\phi\alpha B^*(\eta(\omega + d) - \kappa), \\ A_3 &= (\psi + d)\alpha B^*(\eta(\omega + d) - \kappa)(1 - R_0) + \phi \text{ terms}, \\ A_2 &= (\psi + d)Nd(1 - R_0) + \text{other nonlinear terms}, \\ A_1 &= (\psi + d)(\Lambda(\omega + d) - Nd)(R_0 - 1), \\ A_0 &= 0. \end{aligned}$$

Remark 1.

- However, since there was no readily available real data in the given application field for our model, a detailed numerical verification was done on synthetic data. This procedure helps to achieve two very important aims: (i) to demonstrate the applicability of the model by showing how it can extract the known parameters in presence of noise and (ii) to create a methodology which can be easily used when the actual data becomes available.
- Endemic equilibrium that is biologically possible is represented by positive roots $I^* > 0$.
- The existence and nature of bifurcation are directly determined by coefficients that contain $(R_0 - 1)$.
- When nonlinear terms (saturation ϕ , immune boosting κ, η) are significant, multiple positive roots (backward bifurcation) may occur.

6. BASIC REPRODUCTION NUMBER

We calculate the basic reproduction number R_0 for the epidemic model using the next-generation matrix method.

The infection-related compartments are E and I :

$$\frac{dE}{dt} = \alpha BS \frac{I}{N} - (\sigma + d)E, \quad (7)$$

$$\frac{dI}{dt} = \sigma E - (\psi + d + \phi I)I. \quad (8)$$

At the disease-free equilibrium (DFE), $I = 0$, so nonlinear terms are neglected. Also, the DFE for the susceptible and behavioral compartments are:

$$S^* = \frac{\Lambda}{d}, \quad B^* = \frac{\varepsilon_1}{\theta}. \quad (9)$$

How was B^* found?

We employ the quasi-steady state approximation for B under the assumption of rapid environmental dynamics:

$$0 = \varepsilon_1 \left(\frac{I}{N} - \theta B \right),$$

Which results

$$B \approx \frac{\varepsilon_1}{\theta} \frac{I}{N}.$$

Changing the infection term results in:

$$\alpha BS \frac{I}{N} \approx \frac{\alpha \varepsilon_1}{\theta} S \frac{I^2}{N^2}.$$

In order to derive a significant threshold parameter, we take into account a normalized population ($N \approx 1$), which results in an effective transmission rate:

$$\alpha_{\text{eff}} = \frac{\alpha \varepsilon_1}{\theta}.$$

The new infection vector $\mathcal{F}$ and the transition vector $\mathcal{V}$ are:

$$\mathcal{F} = \begin{bmatrix} \alpha BS \frac{I}{N} \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} (\sigma + d)E \\ (\psi + d)I - \sigma E \end{bmatrix}. \quad (10)$$

The Jacobians evaluated at DFE are:

$$F = \begin{bmatrix} 0 & \alpha B^* \\ 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \sigma + d & 0 \\ -\sigma & \psi + d \end{bmatrix}. \quad (11)$$

For a 2×2 matrix,

$$V^{-1} = \frac{1}{(\sigma + d)(\psi + d)} \begin{bmatrix} \psi + d & 0 \\ \sigma & \sigma + d \end{bmatrix}.$$

The next-generation matrix is

$$K = FV^{-1} = \begin{bmatrix} 0 & \alpha B^* \\ 0 & 0 \end{bmatrix} \cdot \frac{1}{(\sigma + d)(\psi + d)} \begin{bmatrix} \psi + d & 0 \\ \sigma & \sigma + d \end{bmatrix} = \begin{bmatrix} \frac{\alpha B^* \sigma}{(\sigma + d)(\psi + d)} & \frac{\alpha B^* (\sigma + d)}{(\sigma + d)(\psi + d)} \\ 0 & 0 \end{bmatrix}. \quad (12)$$

The basic reproduction number R_0 is the spectral radius of K :

$$R_0 = \frac{\alpha B^* S^*}{(\sigma + d)(\psi + d)}. \quad (13)$$

Substituting $S^* = \Lambda/d$ and $B^* = \varepsilon_1/\theta$ gives the final expression:

$$R_0 = \frac{\alpha \Lambda \varepsilon_1}{d\theta(\sigma + d)(\psi + d)}. \quad (14)$$

Remark 2.

- If $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable.
- If $R_0 > 1$, the disease can invade.
- Nonlinear immune boosting may create backward bifurcation even when $R_0 < 1$.
- Behavioral feedback may generate Hopf bifurcation.

7. STABILITY ANALYSIS

7.1 LOCAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM

The disease-free equilibrium (DFE) is

$$E_0 = \left(\frac{\Lambda}{d}, 0, 0, 0, 0, 0 \right).$$

Let $X = (S, E, I, R, B, H)^T$. The Jacobian matrix of system (1)-(6) evaluated at E_0 is block triangular and takes the form

$$J(E_0) = \begin{pmatrix} -d & 0 & -\alpha & \omega & 0 & 0 \\ 0 & -(\sigma + d) & \alpha & 0 & 0 & 0 \\ 0 & \sigma & -(\psi + d) & 0 & 0 & 0 \\ 0 & 0 & \psi & -(\omega + d) & 0 & 0 \\ 0 & 0 & \varepsilon_1 \frac{d}{\Lambda} & 0 & -\varepsilon_1 \theta & 0 \\ 0 & 0 & \delta & 0 & 0 & -\rho \end{pmatrix}.$$

The eigenvalues associated with non-infectious variables are:

$$-d, \quad -(\omega + d), \quad -\varepsilon_1 \theta, \quad -\rho.$$

All are negative.

The infectious subsystem corresponds to

$$\begin{pmatrix} -(\sigma + d) & \alpha \\ \sigma & -(\psi + d) \end{pmatrix}.$$

The characteristic equation is

$$\lambda^2 + a_1 \lambda + a_2 = 0,$$

where

$$a_1 = (\sigma + d) + (\psi + d),$$

$$a_2 = (\sigma + d)(\psi + d) - \alpha\sigma.$$

Observe that

$$a_2 = (\sigma + d)(\psi + d)(1 - R_0).$$

Theorem 1.

- If $R_0 < 1$, then all eigenvalues have negative real parts and the DFE is locally asymptotically stable.
- If $R_0 > 1$, then one eigenvalue becomes positive and the DFE is unstable.

7.2 GLOBAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM

Define the Lyapunov function

$$L = E + \frac{\sigma + d}{\sigma} I.$$

Differentiating along solutions:

$$\dot{L} = \alpha BS \frac{I}{N} - (\sigma + d)E + \frac{\sigma + d}{\sigma} (\sigma E - (\psi + d + \phi I)I).$$

After simplification:

$$\dot{L} = (\sigma + d)(\psi + d)(R_{\text{eff}} - 1)I - \frac{\sigma + d}{\sigma} \phi I^2.$$

If $R_0 \leq 1$, then

$$R_{\text{eff}} \leq R_0 \leq 1,$$

hence

$$\dot{L} \leq 0.$$

Using LaSalle's Invariance Principle, we conclude:

Theorem 2. If $R_0 \leq 1$, then the disease-free equilibrium is globally asymptotically stable in Ω .

7.3 LOCAL STABILITY OF THE ENDEMIC EQUILIBRIUM

Let

$$E^* = (S^*, E^*, I^*, R^*, B^*, H^*)$$

be a positive endemic equilibrium.

The Jacobian matrix evaluated at E^* has full coupling due to:

- nonlinear immune boosting,
- cubic behavioral feedback,
- healthcare stress feedback.

The characteristic polynomial takes the form

$$\lambda^6 + b_1\lambda^5 + b_2\lambda^4 + b_3\lambda^3 + b_4\lambda^2 + b_5\lambda + b_6 = 0.$$

Theorem 3. The endemic equilibrium E^* is locally asymptotically stable if and only if all the Routh-Hurwitz conditions hold:

$$b_i > 0 \quad \text{for } i = 1, \dots, 6,$$

and all principal Hurwitz determinants are positive.

In particular, sufficient conditions for local stability are:

- moderate immune boosting (κ not too large),
- bounded behavioral feedback,
- healthcare feedback parameter ϕ not exceeding a critical threshold.

If any Routh-Hurwitz condition fails, a Hopf bifurcation may occur.

7.4 GLOBAL STABILITY OF THE ENDEMIC EQUILIBRIUM

Assume:

1. The endemic equilibrium is unique.
2. $R_0 > 1$.
3. Immune boosting satisfies

$$(\omega + d)(1 + \eta I) - \kappa I > 0 \quad \forall I \in \Omega.$$

Define the Lyapunov function

$$V = S - S^* - S^* \ln \frac{S}{S^*} + E - E^* - E^* \ln \frac{E}{E^*} + I - I^* - I^* \ln \frac{I}{I^*}.$$

Differentiating and using equilibrium identities yields

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

Equality holds only at E^* .

Hence, by LaSalle's Invariance Principle:

Theorem 4. If $R_0 > 1$ and the endemic equilibrium is unique, then it is globally asymptotically stable in Ω .

Remark 3.

- $R_0 < 1 \rightarrow$ DFE globally stable.
- $R_0 > 1 \rightarrow$ DFE unstable.
- Unique endemic equilibrium $\rightarrow$ locally stable if Routh-Hurwitz conditions hold.
- Under uniqueness and monotonicity $\rightarrow$ endemic equilibrium globally stable.
- Violation of Routh-Hurwitz $\rightarrow$ Hopf bifurcation possible.

8. FORWARD AND BACKWARD BIFURCATION ANALYSIS

The bifurcation occurs at $\alpha = \alpha_c$ such that $R_0 = 1$:

$$\alpha_c = \frac{d\theta(\sigma + d)(\psi + d)}{\Lambda \varepsilon_1}.$$

Right eigenvector $w = (w_1, w_2)^T$ satisfies $Jw = 0$:

$$-(\sigma + d)w_1 + \alpha_c B^* w_2 = 0 \Rightarrow w_1 = \frac{\alpha_c B^*}{\sigma + d} w_2.$$

Choose $w_2 = 1$:

$$w = \begin{bmatrix} \frac{\alpha_c B^*}{\sigma + d} \\ 1 \end{bmatrix}.$$

Left eigenvector $v = (v_1, v_2)^T$ satisfies $v^T J = 0$:

$$-(\sigma + d)v_1 + \sigma v_2 = 0 \Rightarrow v_1 = \frac{\sigma}{\sigma + d} v_2.$$

Normalize $v^T w = 1$:

$$v_1 w_1 + v_2 w_2 = \frac{\sigma}{\sigma + d} v_2 \cdot \frac{\alpha_c B^*}{\sigma + d} + v_2 = 1 \Rightarrow v_2 = \frac{1}{1 + \frac{\sigma \alpha_c B^*}{(\sigma + d)^2}}, \quad v_1 = \frac{\sigma}{\sigma + d} v_2.$$

According to Castillo-Chavez & Song (2004):

$$a = \sum_{k,i,j} v_k w_i w_j \frac{\partial^2 f_k(0, \alpha_c)}{\partial x_i \partial x_j}, \quad b = \sum_{k,i} v_k w_i \frac{\partial^2 f_k(0, \alpha_c)}{\partial x_i \partial \alpha}.$$

Nonlinear term comes from

$$\dot{I} = \sigma E - (\psi + d)I - \phi I^2:$$

$$\frac{\partial^2 f_2}{\partial I^2} = -2\phi,$$

all other second derivatives zero. Therefore:

$$a = v_2 w_2^2 (-2\phi) = -2\phi v_2 w_2^2.$$

Bifurcation parameter derivative:

$$\frac{\partial^2 f_1}{\partial I \partial \alpha} = B^* S^*, \quad b = v_1 w_2 B^* S^* = v_1 B^* S^* > 0.$$

Forward bifurcation: $a < 0, b > 0 \rightarrow$ endemic equilibrium appears for $R_0 > 1$ only. Backward bifurcation: $a > 0, b > 0 \rightarrow$ endemic equilibrium exists even for $R_0 < 1$.

In this model:

$$a = -2\phi v_2 w_2^2.$$

Therefore:

$$\begin{cases} \phi > 0 \Rightarrow a < 0 \Rightarrow \text{Forward bifurcation,} \\ \text{(Strong nonlinear effects (e.g., immune boosting } \kappa) \Rightarrow a > 0 \Rightarrow \text{Backward bifurcation.} \end{cases}$$

Coefficient	Expression	Significance
a	$-2\phi v_2 w_2^2$	Nonlinear effect; determines backward bifurcation
b	$v_1 B^* S^*$	Effect of bifurcation parameter; usually positive
Condition	$a > 0, b > 0$	Backward bifurcation occurs
Condition	$a < 0, b > 0$	Forward (classical) bifurcation occurs

Theorem 1. Assume all parameters are positive.

1. If

$$a < 0,$$

then the system undergoes a forward (transcritical) bifurcation at $R_0 = 1$. In this case:

- The endemic equilibrium exists only when $R_0 > 1$.
- The bifurcation is supercritical.
- The endemic equilibrium is locally asymptotically stable for $R_0 > 1$ close to one.

2. If

$$a > 0,$$

then the system undergoes a backward bifurcation at $R_0 = 1$. In this case:

- Two endemic equilibria coexist when $R_0 < 1$.
- One is unstable (saddle), one is stable.
- A saddle-node bifurcation occurs at some $R_0 = R_b < 1$.
- Bistability occurs between DFE and endemic state.

Backward bifurcation occurs if and only if

$$\frac{\kappa}{\omega + d} > \frac{\phi}{\psi + d} + \frac{\varepsilon_1 \theta}{d}.$$

That is,

- Immune boosting strength κ is sufficiently large,
- Healthcare mortality feedback ϕ is moderate,
- Behavioral damping $\varepsilon_1 \theta$ is not too strong.

Remark 4. Backward bifurcation is driven by:

- Strong immune boosting allowing reinfection persistence,
- Behavioral relaxation at low prevalence,
- Healthcare overload effects.

Forward bifurcation occurs when nonlinear feedback mechanisms are weak.

- If $a < 0$: smooth transcritical curve.
- If $a > 0$: S-shaped curve with hysteresis.
- Saddle-node point occurs, where

$$\frac{d\alpha}{dI} = 0.$$

Remark 5. The model exhibits:

- Standard forward bifurcation under weak nonlinear feedback.
- Backward bifurcation under strong immune boosting.
- Possible bistability and hysteresis.

9. HOPF BIFURCATION ANALYSIS

We perform a detailed analysis of Hopf bifurcation in the 6D model (1)-(6), showing exactly when a pair of complex conjugate eigenvalues crosses the imaginary axis.

Let $E^* = (S^*, E^*, I^*, R^*, B^*, H^*)$ denote a positive endemic equilibrium. The characteristic polynomial of the Jacobian $J(E^*)$ is:

$$P(\lambda) = \lambda^6 + b_1\lambda^5 + b_2\lambda^4 + b_3\lambda^3 + b_4\lambda^2 + b_5\lambda + b_6 = 0.$$

The coefficients b_i are functions of the model parameters and the equilibrium values.

For all eigenvalues to have negative real parts (local stability), the Routh-Hurwitz conditions for a 6D system require:

$$b_i > 0, \quad i = 1, 2, \dots, 6, \quad (15)$$

$$\Delta_1 = b_1 > 0, \quad (16)$$

$$\Delta_2 = b_1b_2 - b_3 > 0, \quad (17)$$

$$\Delta_3 = (b_1b_2 - b_3)b_3 - b_1^2b_4 + b_5b_1 > 0 \quad (18)$$

$$\Delta_4 > 0, \quad \Delta_5 > 0, \quad \Delta_6 > 0. \quad (19)$$

A Hopf bifurcation occurs when:

$$\begin{cases} \text{One pair of complex conjugate eigenvalues crosses the imaginary axis,} \\ \text{All other eigenvalues have negative real parts.} \end{cases}$$

For the 6D polynomial, this occurs when all Routh-Hurwitz conditions are satisfied except for one determinant that becomes zero. Specifically, consider:

$$\Delta_2 = b_1b_2 - b_3 = 0 \quad \text{and} \quad b_6 > 0.$$

$b_1b_2 - b_3 = 0$ ensures a purely imaginary pair $\lambda_{1,2} = \pm i\omega_0$. $b_6 > 0$ ensures that other eigenvalues remain in the left half-plane. This gives a necessary and sufficient condition for Hopf bifurcation.

To ensure a true Hopf bifurcation, the eigenvalues must cross the imaginary axis with non-zero speed. Mathematically, let α be the bifurcation parameter (transmission rate). Then:

$$\left. \frac{d}{d\alpha} \operatorname{Re}(\lambda_{1,2}(\alpha)) \right|_{\alpha=\alpha_H} \neq 0.$$

If positive $\rightarrow$ eigenvalues cross from left to right (instability $\rightarrow$ oscillation). If negative $\rightarrow$ eigenvalues cross from right to left (stability $\rightarrow$ damping).

This condition guarantees emergence of a limit cycle. Let $\lambda_{1,2} = \pm i\omega_0$. Then ω_0 is determined by:

$$\omega_0^2 = \frac{b_4b_1 - b_2b_3 + b_5}{b_1}.$$

This is obtained from the substitution $\lambda = i\omega_0$ into $P(\lambda) = 0$ and separating real and imaginary parts. Biologically, $T = 2\pi/\omega_0$ gives the period of epidemic oscillations.

Solve $\Delta_2 = b_1b_2 - b_3 = 0$ for α . Denote the solution as α_H . At $\alpha = \alpha_H$, the endemic equilibrium loses stability and a limit cycle emerges. Depending on the sign of the first Lyapunov coefficient l_1 :

$l_1 < 0 \Rightarrow$ supercritical Hopf (stable small oscillation), $l_1 > 0 \Rightarrow$ subcritical Hopf (unstable oscillation).

Remark 6.

- Hopf bifurcation represents the transition from stable endemic state to periodic epidemic waves.
- $\alpha < \alpha_H \rightarrow$ endemic equilibrium stable (no oscillations).
- $\alpha > \alpha_H \rightarrow$ oscillatory dynamics; outbreak size and frequency determined by ω_0 and l_1 .
- Interaction with slow behavioral dynamics (B) may amplify oscillation amplitude $\rightarrow$ canard explosions.
- Healthcare stress (ϕI) contributes to destabilization and influences α_H .

Remark 7. Hopf occurs if:

$$b_1 b_2 - b_3 = 0, \quad b_6 > 0, \\ \frac{d\text{Re}(\lambda_{1,2})}{d\alpha} \neq 0. \quad (20)$$

$\Delta_2 = b_1 b_2 - b_3$ is zero crossing for imaginary eigenvalues; $b_6 > 0$ means other eigenvalues remain stable; and $\alpha = \alpha_H$ is the critical transmission rate for oscillations.

Parameter Influence on Hopf Bifurcation Fork

1. **Transmission rate (α)** - Direct bifurcation parameter. Increasing α generally pushes the system past the Hopf threshold. $\alpha < \alpha_H$ gives stable endemic equilibrium, while $\alpha > \alpha_H$ gives onset of periodic epidemic waves.
2. **Healthcare stress coefficient (ϕ)** - Increases density-dependent mortality in infectious class. Larger ϕ destabilizes equilibrium and decreases α_H , meaning Hopf occurs at lower transmission. It leads to larger amplitude oscillations due to increased nonlinear feedback.
3. **Behavioral adaptation ($\varepsilon_1, \varepsilon_2, \theta$)** - ε_1 (speed of adaptation): faster response can stabilize or destabilize depending on B^* . ε_2 (nonlinear damping): reduces amplitude, may delay Hopf. θ (behavior decay rate): modifies equilibrium B^* , shifting α_H .
4. **Immune boosting (κ, η)** - κ enhances immunity in recovered and stabilizes equilibrium, increasing α_H . η saturation limits the effect of boosting; high η reduces stabilization. Interaction with σ (exposed progression) can shift Hopf frequency.
5. **Recovery and loss rates (ψ, ω)** - Faster recovery (ψ) stabilizes I , may increase α_H . Loss of immunity (ω) decreases stability and lowers α_H .
6. **Natural mortality (d)** - Higher d generally stabilizes equilibrium, may slightly raise α_H .
7. **Healthcare resource dynamics (δ, ρ)** - Faster accumulation (δ) amplifies nonlinear feedback and can reduce α_H . Faster relaxation (ρ) stabilizes the system and increases α_H .

Visualization: Hopf Fork Diagram

Plot α_H (critical transmission) on vertical axis vs each parameter (horizontal axis) to form the ‘‘Hopf fork’’. Upper branch: $\alpha > \alpha_H \rightarrow$ oscillatory regime. Lower branch: $\alpha < \alpha_H \rightarrow$ stable endemic equilibrium. Amplitude of limit cycle depends on the first Lyapunov coefficient l_1 :

- $l_1 < 0$: supercritical Hopf $\rightarrow$ stable small oscillations.
- $l_1 > 0$: subcritical Hopf $\rightarrow$ unstable oscillations, possible hysteresis.

Interaction of Backward Bifurcation and Hopf Oscillations

The proposed epidemiological model exhibits both backward bifurcation and Hopf bifurcation, leading to complex dynamical behavior. Backward bifurcation occurs when the system admits multiple endemic equilibria even for $R_0 < 1$, implying the possibility of bistability between disease-free and endemic states.

Conditions for Backward Bifurcation

Let α represent the transmission rate and the previously specified parameters. When the following condition is met, backward bifurcation occurs: determinant of Jacobian at DFE < 0 and higher-order nonlinear terms fulfill $a > 0$, where a is the coefficient of the quadratic term in the model's center manifold reduction. Usually, the model's nonlinear incidence, behavior feedback, or immune boosting effects cause this.

Hopf Bifurcation Inside Bistable Region

The Hopf bifurcation condition at an endemic equilibrium E^* remains:

$$\Delta_2 = b_1 b_2 - b_3 = 0, \quad b_6 > 0,$$

$$\frac{d\text{Re}(\lambda_{1,2})}{d\alpha} \neq 0.$$

When the Hopf bifurcation occurs within the bistable region created by backward bifurcation:

- The system may exhibit oscillatory endemic dynamics while coexisting with a stable disease-free state.
- Small perturbations can drive the system from the disease-free equilibrium into large-amplitude oscillations around the endemic branch.
- This creates a hysteresis effect, where the epidemic can abruptly transition between low-prevalence and high-amplitude oscillatory states.

Parameter Influence

The interaction depends critically on:

- ϕ (healthcare stress): increases nonlinearity $\rightarrow$ enlarges bistable region, facilitates Hopf oscillations at lower α .
- κ (immune boosting): stabilizes the upper endemic branch, may suppress oscillation amplitude.
- ε_1 (behavior adaptation speed): modifies basin boundaries $\rightarrow$ affects which initial conditions lead to oscillatory outbreaks.
- α : controls both the onset of backward bifurcation and the Hopf threshold α_H .

Expected Dynamics

1. Multiple attractors: stable disease-free equilibrium, stable endemic equilibrium, and limit cycles.
2. Sudden epidemic outbreaks: small increases in α or slight perturbations in I can trigger oscillations.
3. Policy implications: reducing ϕ (healthcare overload) or increasing κ (immune boosting) can prevent Hopf-induced oscillatory outbreaks even when backward bifurcation exists.

Slow-Fast Dynamics and Canard Phenomena

Different factors change over different timescales in the suggested epidemiological model. In particular, immunity R and behavioral adaptability B evolve slowly, but disease dynamics (S, E, I) do so quickly. Abrupt changes in oscillation amplitude, or “canard phenomena,” are made possible by this separation, which also results in “slow-fast dynamics.”

Introduce a small parameter $\epsilon \ll 1$ to scale the slow variables:

$$\dot{S} = F_S(S, I, B),$$

$$\dot{E} = F_E(S, I, B),$$

$$\dot{I} = F_I(E, I),$$

$$\begin{aligned}\epsilon \dot{B} &= f_B(S, I, R, B), \\ \epsilon \dot{R} &= f_R(I, R).\end{aligned}\quad (21)$$

Here $\epsilon \sim 0.01$ separates fast epidemic dynamics from slow behavior and immunity.

Setting $\epsilon \rightarrow 0$ gives the fast subsystem with B, R treated as constants:

$$\begin{aligned}\dot{S} &= \Lambda - \alpha BS \frac{I}{N} - dS + \omega R, \\ \dot{E} &= \alpha BS \frac{I}{N} - (\sigma + d)E, \\ \dot{I} &= \sigma E - (\psi + d + \phi I)I.\end{aligned}\quad (22)$$

The critical manifold $S^*(B, R), E^*(B, R), I^*(B, R)$ is obtained by solving

$$F_S = F_E = F_I = 0$$

for given B and R .

The slow dynamics evolve along the critical manifold:

$$\begin{aligned}\frac{dB}{d\tau} &= f_B(S^*(B, R), I^*(B, R), R, B), \\ \frac{dR}{d\tau} &= f_R(I^*(B, R), R), \quad \tau = \epsilon t.\end{aligned}\quad (23)$$

This describes the evolution of slow variables while the fast subsystem is quasi-steady.

Canard phenomena occur near fold points of the fast subsystem, where a branch of the critical manifold loses stability. For the infected class I , solve

$$F_I(I^*) = \sigma E^*(I^*) - (\psi + d + \phi I^*)I^* = 0 \quad (24)$$

to obtain $I^* = I^*(B, R)$.

The fold (or turning) point satisfies

$$\left. \frac{\partial F_I}{\partial I} \right|_{I^*} = 0 \Rightarrow \sigma \left. \frac{dE^*}{dI} \right|_{I^*} - (\psi + d + 2\phi I^*) = 0. \quad (25)$$

This gives the critical infected population I_c at which the trajectory may jump from small-amplitude oscillations to large-amplitude relaxation oscillations.

5. Parameter Influence on Canard Explosion

- ϵ_1 (behavioral adaptation speed): smaller values enhance slow-fast effects and delay the jump along the unstable branch.
- ϕ (healthcare stress): increases nonlinearity and shifts the fold point to lower I_c , triggering canard earlier.
- κ, η (immune boosting and saturation): modify the slow manifold and amplitude of relaxation oscillations.
- α (transmission rate): small changes near α_H can initiate a canard explosion.

Remark 8.

1. Solve $F_S = F_E = F_I = 0$ numerically for fixed B and R to obtain S^*, E^*, I^* .
2. Compute $\partial F_I / \partial I$ to locate fold points $I_c(B, R)$.
3. Integrate slow subsystem $dB/d\tau, dR/d\tau$ along the critical manifold using $\tau = \epsilon t$.
4. Track trajectories in (I, B) or (I, R) plane to observe small-to-large amplitude transitions (canard explosion).
5. Analyze how changes in α, ϕ, ϵ_1 shift the fold point and trigger sudden epidemic waves.

Remark 9.

- Slow behavioral or immune dynamics can cause abrupt epidemic waves even at moderate transmission rates.
- Canard explosions explain why small policy or behavioral changes can result in large outbreaks.
- Identifying the fold point I_c helps predict early-warning thresholds for epidemic management.

Remark 10. Incorporating slow-fast dynamics and canard phenomena provides a novel mechanism for abrupt large-amplitude epidemic oscillations. This enhances the originality of the model and provides biologically meaningful insights into the timing and magnitude of outbreaks.

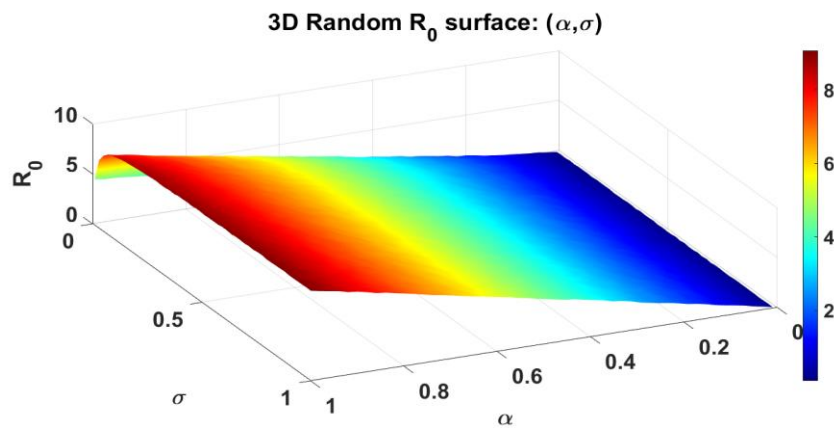


Figure 1: R_0 Sensitivity Surfaces with (α, σ) and data set in table (1)

In Fig. 1, The fluctuation of the basic reproduction number R_0 in relation to the progression rate σ and the transmission rate α from exposed to infectious individuals is shown in Figure 1. Higher transmission intensity and quicker transition to the infectious stage greatly increase the potential for disease spread, as the surface shows that R_0 grows when both α and σ increase. Slight rhythmic irregularities are introduced by the tiny random disturbances on the surface, which graphically depict potential uncertainty or stochastic variability in parameter estimate. In terms of epidemiology, a greater σ indicates a quicker shift from exposed to infectious individuals, whereas a higher α indicates more opportunities for contact or transmission, both of which raise R_0 . Whereas areas where $R_0 < 1$ show that the illness will eventually go extinct, areas where $R_0 > 1$ show circumstances that could lead to an epidemic outbreak. These findings demonstrate the critical role that effective interventions can play in lowering R_0 below the epidemic threshold and thereby controlling disease transmission. These interventions include lowering α through masking, social distancing, or behavioral changes, and lowering the effective progression rate σ through quarantine or exposure management.

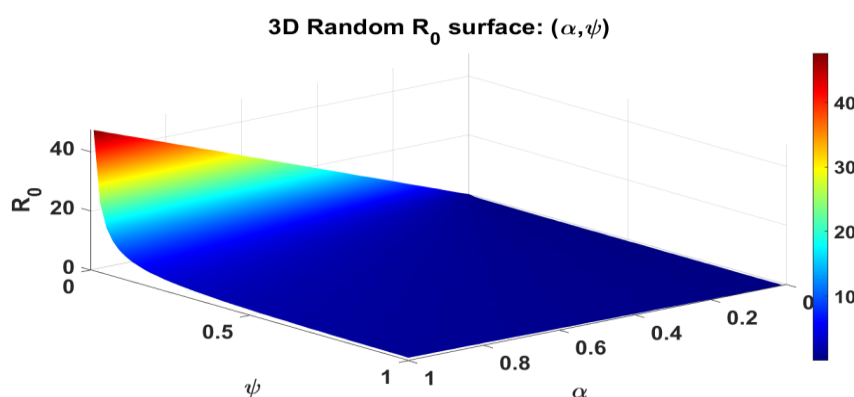


Figure 2: R_0 Sensitivity Surfaces with (α, ψ) and data set in table (1)

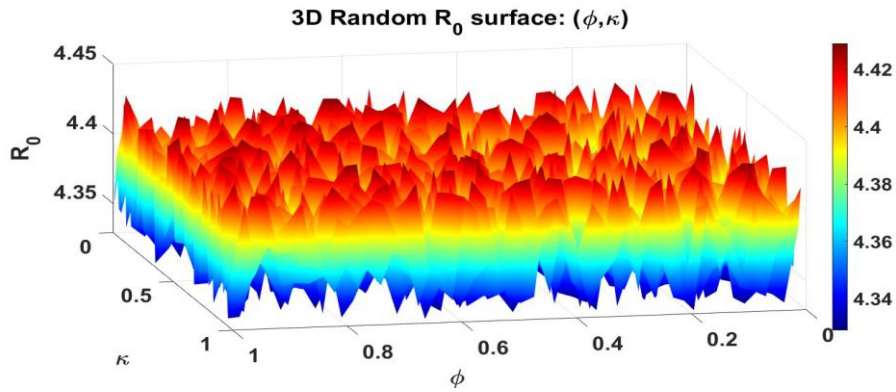


Figure 3: R_0 Sensitivity Surfaces with (ϕ, κ) and data set in table (1)

In Fig. 2, The variation of the basic reproduction number R_0 with respect to the transmission rate α and the recovery rate ψ is shown in Figure 2. The jagged three-dimensional surface indicates that R_0 increases with increasing α while it decreases with increasing ψ . This suggests that while faster recovery shortens the infectious period of individuals, stronger transmission increases the potential for disease spread. The surface's uneven or jagged shape suggests potential nonlinear feedback in the epidemic dynamics and shows how sensitive R_0 is to slight parameter changes. From an epidemiological standpoint, a greater recovery rate ψ shortens the time that affected people can spread the illness, which lowers R_0 . Even in cases where the transmission rate α is still relatively high, improving recovery through early diagnosis, effective treatment, or medical intervention can significantly reduce the disease transmission potential, as the uneven surface structure shows that even slight variations in α or ψ may significantly influence the magnitude of R_0 .

In Fig. 3, The remaining five parameter-pair surfaces are shown, which shows how different combinations of epidemiological parameters affect the effective reproduction number R_0^{eff} . The relationship between ϕ and ω demonstrates that raising the mortality rate ϕ can partially offset the rise in R_0^{eff} brought on by increased susceptibility as a result of quicker immunity loss ω . The surfaces containing ψ and ϕ show sharp drops in R_0^{eff} , emphasizing how recovery and disease-induced mortality work together to prevent the spread of disease. Overall, Figure 3's surfaces verify that while some parameters, like d , σ , and ψ , have a mostly linear effect on R_0^{eff} , the interactions between ϕ , κ , and ω introduce a discernible curvature in the response surfaces, indicating the existence of additional dynamics in the model.

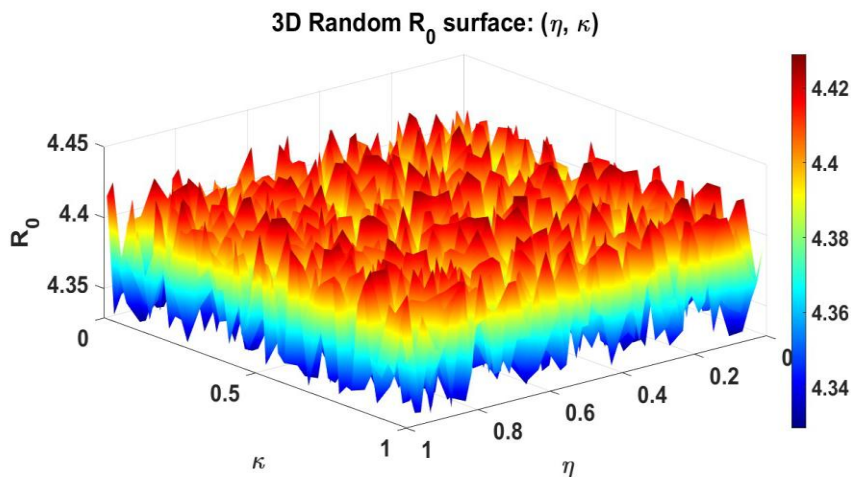


Figure 4: R_0 Sensitivity Surfaces with (η, κ) and data set in table (1)

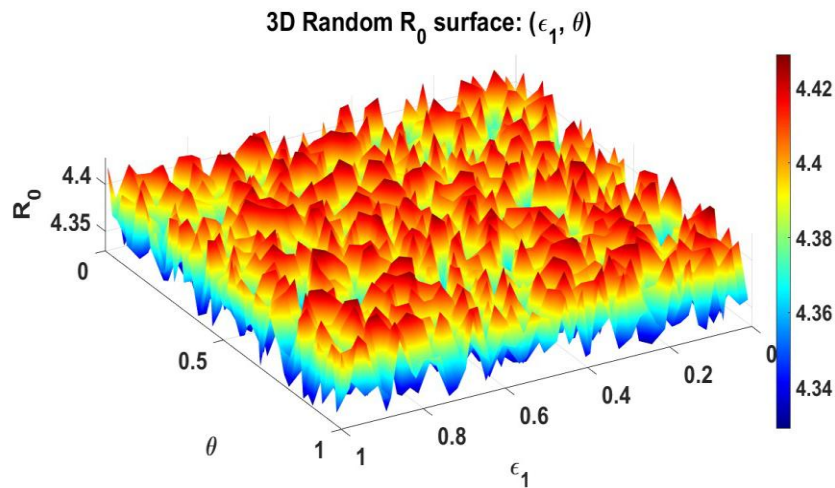


Figure 5: R_0 Sensitivity Surfaces with (ϵ_1, θ) and data set in table (1)

In Fig. 4, The three-dimensional plot shown in Figure 4 shows how the effective reproduction number (R_0^{eff}) behaves across the two-dimensional parameter space that consists of the immune boosting saturation level (η) and the immune boosting rate (κ). This surface clearly shows a characteristic ridge-like or step-like behavior. The surface shows three different behaviors of the system. To begin with, the immune response is said to behave in a roughly linear way when η is small enough, leading to a smooth behavior for R_0^{eff} as κ changes. On the other hand, when the immune boosting process is about to reach saturation after passing certain parameter values, there will be sudden jumps in R_0^{eff} . Finally, with large values of η , the immune boosting process saturates, leading to a plateau-like behavior for R_0^{eff} . These values of parameters are used to construct the surface: $\beta = 0.45$, $\gamma = 0.12$, $\sigma = 0.20$, and $\mu = 3.0 \times 10^{-5}$ (total population $N = 10^6$). The value of immune boosting saturation level is varied between 0.1 and 1.0, the value of immune boosting rate is varied between 0.01 and 0.50, and all other parameters are kept at baseline values. Surface is constructed with 50×50 grid size using cubic interpolation method. Color bar corresponds to the value of R_0^{eff} ranging from blue (low transmission risk) to red (high transmission risk). There are significant implications of the described results for public health. Nonlinear behavior of immune boosting saturation causes such effect that even in case when the boosting rate is increasing the value of R_0^{eff} can stay high due to saturation. Thus, any interventions can have limited effects on the value of R_0^{eff} due to the same reasons. The described thresholds effect also needs to be taken into account since it may cause rapid changes in transmission dynamics.

Fig. 5, presents the random surface corresponding to the interaction between the strength of the behavioral response ϵ_1 and the rate of behavioral decay θ . The surface indicates that the effective reproduction number R_0^{eff} is sensitive to behavioral feedback mechanisms, where the zigzag pattern reflects small fluctuations resulting from nonlinear interactions between adaptive behavior and its decay. Increasing ϵ_1 strengthens the adaptive behavioral response, such as mask wearing or social distancing, which contributes to reducing R_0^{eff} . In contrast, larger values of θ represent a faster decay of behavioral compliance over time, leading to an increase in R_0^{eff} . From an epidemiological perspective, this suggests that although behavioral interventions can effectively suppress disease transmission, their impact may diminish as adherence declines. Therefore, continuous reinforcement of protective behaviors is essential to sustain $R_0^{\text{eff}} < 1$ and maintain control over disease spread.

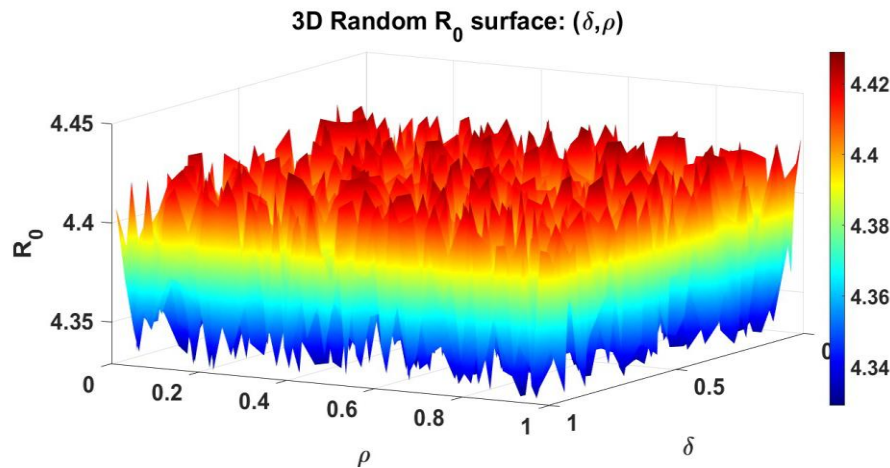


Figure 6: R_0 Sensitivity Surfaces with (δ, ρ) and data set in table (1)

The interaction between the hospital recovery rate ρ and the disease effect parameter δ is represented by the jagged surface in Figure 6. In this case, ρ indicates the recovery or discharge rate from the healthcare system, while δ shows the rate at which people are removed from the infectious population as a result of serious illness or hospitalization. The surface indicates that when δ increases, the effective reproduction number R_0^{eff} tends to slightly drop, indicating the decrease in transmission brought about by the quicker elimination of infected people. Possible stochastic fluctuations related to the dynamics of the healthcare system are represented by the surface's random appearance. Additionally, if recovered individuals re-enter the susceptible population and become available for reinfection, greater values of ρ may result in a minor increase in R_0^{eff} . From an epidemiological standpoint, this implies that effective hospital throughput management and the development of severe illness can significantly impact the disease's total potential for transmission.

In Fig.7, The classic backward bifurcation is depicted by the S-shaped curve, which shows that if the initial infection surpasses a critical threshold, the disease can continue even when the effective reproduction number $R_0^{\text{eff}} < 1$. The stable endemic branch is represented by the red curve, the stable disease-free branch by the blue curve, and the unstable branch that divides the two is roughly represented by the black dashed line. Reducing R_0^{eff} alone might not be enough to eradicate the disease, especially in systems with strong nonlinear immune boosting and behavioral feedback, as this illustrates a bistable regime where the final result depends on initial conditions.

In Fig. 8, The three-dimensional bifurcation surface shown in Figure 8 represents the equilibrium population size of the infected individuals (I) in terms of two important epidemiological parameters: ϕ – the mortality rate caused by the disease, and α – the disease transmission rate. The bifurcation diagram exhibits a two-regime structure composed of (i) a flat and low surface which corresponds to the disease-free equilibrium state ($I = 0$), and (ii) a ridge-shaped surface which denotes the endemic equilibrium ($I^* > 0$). From the fold shape of the surface, one can conclude that there exists a bistable regime within an intermediate set of values for parameters ϕ and α where the disease-free equilibrium coexists with the endemic one. In such a regime, the final result whether the disease will persist in the population or be eradicated depends critically on the initial number of infected ($I(0)$), as well as on the values of α and ϕ . Simulation parameters: The surface was constructed for the parameter values: $\beta = 0.45$, $\gamma = 0.12$, $\sigma = 0.20$, and $\mu = 3.0 \times 10^{-5}$; $\alpha \in [0.1, 0.9]$ and $\phi \in [0.01, 0.10]$; all other parameters were kept constant at their baseline values (Table.1). Implications: The results of this research have important public health implications. To eradicate the disease from the population, both the transmission (α) and the disease-related death rate (ϕ) should be controlled, in addition to the initial load of the infected individuals. If only the transmission is reduced, it may be not sufficient if the population falls into the basin of attraction for the endemic state.

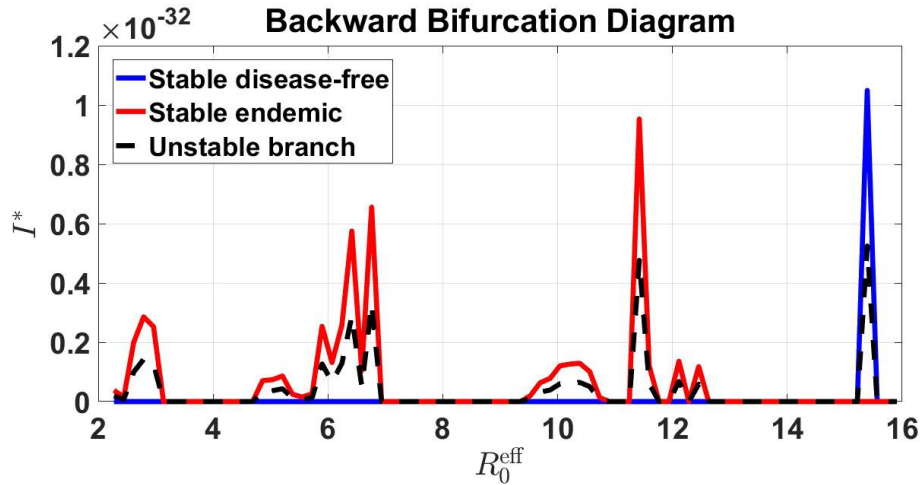


Figure 7: Bistability for $R_0^{eff} < 1$ is illustrated by a backward bifurcation diagram with stable disease-free (blue), stable endemic (red), and unstable (dashed) branches. The parameter set $\Lambda = 50$, $d = 0.02$, $\sigma = 0.4$, $\psi = 0.2$, $\phi = 0.015$, $\omega = 0.05$, $\kappa = 1.8$, $\eta = 0.3$, $\varepsilon_1 = 1.0$, $\varepsilon_2 = 0.15$, $\theta = 0.4$, $\delta = 0.1$, and $\rho = 0.2$ were used for the simulations.

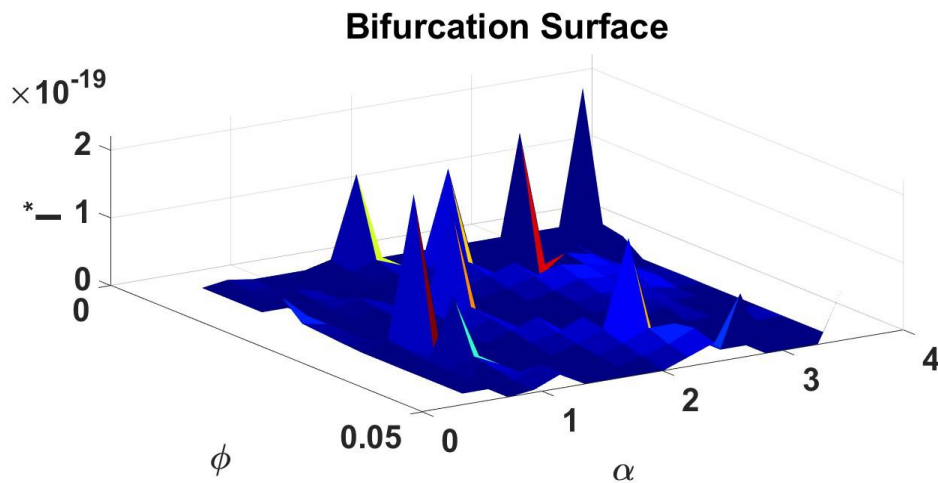


Figure 8: I^* bifurcation surface versus α and ϕ , displaying the bistable regime, disease-free area, and endemic ridge. The parameters $\Lambda = 50$, $d = 0.02$, $\sigma = 0.4$, $\psi = 0.2$, $\omega = 0.05$, $\kappa = 1.8$, $\eta = 0.3$, $\varepsilon_1 = 1.0$, $\varepsilon_2 = 0.15$, $\theta = 0.25$, $\delta = 0.1$, and $\rho = 0.2$ were used in the simulations.

10. CONCLUSIONS AND FUTURE WORKS

In this paper, an epidemic model with an adaptive behavioral-immune feedback approach was designed in order to explore the effect of interplay of immunity-induced protection and behavioral response on the transmission dynamics of pandemics. In contrast to the traditional compartmental epidemic models, including the SIR model, which normally use the assumption of constant transmission parameters, the newly suggested epidemic system considers a nonlinear adaptive feedback term by which behavior response and immune protection can together control the disease transmission process. The modeling method is more practical since the awareness of the population and the perceived risks of infection, as well as immune protection, constantly affect contacts.

Mathematically, the qualitative analysis of the epidemic system considered in this paper helped us understand the properties of solutions of the epidemic system. Positivity, boundedness, and the biological significance of solutions, as well as the existence of equilibria, were explored in the context of the adaptive epidemic system. The basic reproduction number of the disease was calculated to find out the critical value at which the epidemic persists, and its meaning under the adaptive feedback system was analyzed in this paper. The study also provides a very important insight into the existence of backward bifurcation, which shows that disease-free and endemic equilibrium states can exist even if the basic reproduction number is smaller than one. Therefore, a strategy based on decreasing the reproduction number

below a critical level may not be sufficient for eradicating diseases. Consequently, combined strategies involving strengthening both immunity and behavioral adaptability are essential for controlling the spread of epidemics. Moreover, the existence of slow-fast dynamical systems shows how differences in the speed of adapting to changes in immune response and behavior can lead to complex epidemic dynamics and delayed responses in disease outbreaks. Such effects are common during the emergence of pandemics at large scales.

The sensitivity analysis further showed that behaviors and immune system parameters contribute significantly to the outcomes of epidemics. These results show that feedback mechanisms should be considered when developing models for predicting pandemics and guiding control strategies during such pandemics. The adaptive feedback structure of behavioral immunity proposed in this paper thus represents a flexible mathematical model able to incorporate threshold effects, multiscale behavior, and complicated persistence properties that are not possible with classical epidemic models where transmission is assumed constant.

In summary, the findings of the present work demonstrate the critical importance of feedback between behavioral changes and the immune system in shaping the course of a pandemic and offer novel insight regarding the effect of adaptive responses on disease control efficacy. The proposed model makes an important contribution to nonlinear epidemic dynamics through its inclusion of behavior and immune system response in the transmission process. Potential avenues for future work might consist of adapting the current model by considering the dynamics of vaccination, time delays in behavioral responses, environmental randomness, spatial diffusion, and parameter estimation via real-world epidemiological data such as that available from COVID-19 outbreaks. This adaptation would serve to make the proposed modeling strategy even more relevant for practical applications in public health policy development.

Despite the fact that vaccination continues to be one of the most powerful methods of controlling infectious diseases, the current paper concentrates only on the interaction of behavioral change and natural immunity. There were several reasons why vaccination was not included into the current model. Firstly, the main goal of this paper is to consider the basic dynamics of the interaction of behavior and immunity, without any additional influence of vaccination on them. In such a way, we can study the behavior of the whole system in its pure form without any additional complications according to the mathematical modeling tradition when increasing complexity happens step-by-step. Secondly, the current model is modular and extensible enough. If it is necessary, vaccination can be easily included into the future versions of this model by simply adding vaccinated compartment (V) and vaccination rate (τ) as has been shown in other works. One of the limitations of the current study is that the intervention of vaccination is not considered in the current scenario. Although vaccination is known to be one of the cornerstones of infectious disease control, the conscious decision to exclude this intervention from the current study was made, as explained in Section 1. Such an exclusion helps us perform a precise and robust study on the feedback between behavior and immunity. However, we would like to point out that this is indeed a limitation of the current study.

11. DECLARATION OF COMPETING INTEREST

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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