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Catastrophe Theory in SEIRV Epidemic Modeling

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Abstract

Methodology: This study formulates a 5 × 5 SEIRV (Susceptible-Exposed-Infected-Recovered-Vaccinated) nonlinear epidemic matrix model to investigate epidemic dynamics through catastrophe theory. The nonlinear system is reduced near equilibrium points, and the infected population dynamics are transformed into a cubic structure equivalent to the cusp catastrophe model. Analytical methods are employed to derive conditions for multiple equilibria, bifurcation, and sudden epidemic transitions, and a new theorem is established demonstrating that nonlinear epidemic systems with quadratic infection terms and degenerate Jacobian matrices are locally equivalent to cusp catastrophe dynamics. **Results:** The results indicate that several equilibrium states, bistability, and bifurcating phenomena are present in the epidemic model, while numerical simulations confirm the theoretical predictions and illustrate catastrophic epidemic outbreaks caused by small variations in epidemiological parameters. **Discussion:** These findings suggest that epidemic systems may undergo abrupt transitions that cannot be fully captured through conventional linear stability analysis, highlighting the presence of hidden instability thresholds and hysteresis effects in nonlinear epidemic behavior. **Findings:** The integration of catastrophe theory with epidemic matrix modeling offers a novel mathematical perspective on sudden epidemic transitions and mechanisms of instability in infectious disease systems. **Conclusion:** The study concludes that catastrophe theory provides a powerful mathematical framework for analyzing epidemic instability and sudden outbreak transitions, offering valuable insights for epidemic prediction and the development of more effective disease control strategies.

Subject Areas

Epidemiology

Keywords

Epidemic Dynamics, Cusp Catastrophe, Bifurcation Analysis, Hysteresis, Equilibrium Stability, Infectious Disease Modeling

1. Introduction

Mathematical modeling has become an essential tool in understanding the spread and control of infectious diseases. Compartmental epidemic models, particularly Susceptible-Exposed-Infected-Recovered-Vaccinated (SEIRV) frameworks, are widely employed to describe disease transmission dynamics, estimate epidemic thresholds, and assess intervention strategies within populations [1]. Recent developments in epidemic matrix modeling and numerical epidemic analysis have further enhanced the understanding of compartmental disease systems, including SEIRV formulations and iterative solution methods [2].

Mathematical epidemic models continue to play a significant role in forecasting outbreak patterns and informing public health decisions in modern epidemiology. However, many epidemic systems exhibit nonlinear characteristics that cannot be adequately represented by traditional linear approaches. Classical linear stability analysis is effective in determining local equilibrium behavior but often fails to explain abrupt epidemic outbreaks or collapses resulting from small parameter perturbations. Recent studies have shown that nonlinear epidemic systems may exhibit bifurcation, multistability, and threshold-triggered transitions, indicating the presence of complex dynamic mechanisms beyond conventional linear predictions [3]-[5]. Such sudden transitions are especially relevant in infectious disease modeling, where slight variations in transmission or recovery rates may trigger disproportionate changes in outbreak magnitude.

To address such phenomena, catastrophe theory offers a rigorous mathematical framework for analyzing discontinuous transitions caused by continuous parameter variation. First introduced in [6] and subsequently extended in contemporary studies of nonlinear stability [7]-[9], catastrophe theory provides a systematic method for investigating systems that undergo abrupt structural changes near critical thresholds. This theoretical framework is particularly effective in describing nonlinear systems characterized by bifurcation, hysteresis, and abrupt transitions between multiple equilibrium states [10]-[12].

In epidemic dynamics, nonlinear incidence functions and matrix-based compartmental interactions frequently generate complex structures that may lead to hidden instability thresholds and catastrophic transitions. Recent research has confirmed that nonlinear epidemic systems can produce bistable states, discontinuous transitions, and multiple outbreak thresholds under certain epidemiological conditions. Matrix epidemic models, in particular, provide a compact representation of compartmental interactions and facilitate advanced analytical study of equilibrium and bifurcation structures.

Motivated by these developments, this study investigates the application of catastrophe theory to a nonlinear 5 × 5 SEIRV epidemic matrix model. By reducing the system near its equilibrium points and analyzing the dynamics of the infected population, the research establishes a mathematical connection between epidemic matrix behavior and cusp catastrophe structures. This integration provides a novel theoretical perspective for understanding epidemic instability, predicting sudden outbreak transitions, and improving the strategic disease control mechanisms.

2. Methodology

This study develops a mathematical framework based on a 5 × 5 SEIRV (Susceptible-Exposed-Infected-Recovered-Vaccinated) nonlinear epidemic model to analyze disease transmission dynamics. The total population is partitioned into five compartments, and the transitions between these classes are described using a system of nonlinear differential equations. The model incorporates key epidemiological processes, including infection, progression, recovery, vaccination, and natural removal rates.

To capture complex dynamic behavior, the system is reformulated in matrix form, where state variables are represented as a vector, and the interaction terms are encoded in a nonlinear transition matrix. This formulation allows the use of linearization techniques around equilibrium points to study local stability properties. The Disease-Free Equilibrium (DFE) and endemic equilibrium states are derived by solving the nonlinear system.

Catastrophe theory is employed to investigate sudden qualitative changes in the system behavior resulting from continuous variation in control parameters. Specifically, bifurcation phenomena and fold-type catastrophes are examined to determine the conditions in which minor variations in system parameters can trigger sudden shifts between disease-free, endemic, and epidemic outbreak states. Stability analysis is performed using eigenvalue techniques applied to the Jacobian matrix evaluated at equilibrium points.

Numerical simulations are conducted to validate analytical results and to visualize phase portraits, equilibrium transitions, and threshold behavior under varying parameter regimes. The combined analytical and computational approach provides a comprehensive understanding of nonlinear epidemic dynamics and their sensitivity to changes in parameters, particularly vaccination and transmission rates.

3. SEIRV Model Formulation

To investigate epidemic dynamics with vaccination effects and nonlinear transitions, we consider a SEIRV (Susceptible-Exposed-Infected-Recovered-Vaccinated) compartmental framework. The total population is partitioned into five mutually exclusive classes:

$$S(t), E(t), I(t), R(t), V(t), \text{ where } \quad N(t) = S + E + I + R + V .$$

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The dynamics are governed by nonlinear interactions representing infection transmission, incubation, recovery, loss of immunity, and vaccination flow. The core structure of the model can be written as:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda - \beta \frac{SI}{N} - \nu S + \omega R - \mu S, \\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta \frac{SI}{N} - (\sigma + \mu) E, \\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \sigma E - (\gamma + \mu + \alpha) I, \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I - (\omega + \mu) R, \\
\frac{\mathrm{d}V}{\mathrm{d}t} &= \nu S - \mu V.
\end{aligned}$$

where:

- Λ : recruitment rate,
- β : transmission rate,
- ν : vaccination rate,
- σ : incubation rate,
- γ : recovery rate,
- α : disease-induced mortality,
- ω : immunity waning rate,
- μ : natural death rate.

This nonlinear system forms the basis for analyzing equilibrium structures and catastrophic transitions.

4. Equilibrium Points and Disease States

The model admits two principal classes of equilibrium:

1) Disease-Free Equilibrium (DFE)

At this equilibrium, $E = I = 0$, and the population is distributed among susceptible, recovered, and vaccinated classes. The DFE represents epidemic extinction and is given by:

$$E_0 = \left(S^*, 0, 0, R^*, V^*\right)$$

where the components satisfy balance relations derived from the steady-state system.

2) Endemic Equilibrium (EE)

The endemic state occurs when $I^* > 0$, indicating persistent disease transmission. This equilibrium is determined by solving the nonlinear algebraic system obtained by setting all derivatives to zero. The existence of EE depends critically on the basic reproduction number $\mathcal{R}_0$, typically defined as:

$$\mathcal{R}_0 = \frac{\beta\sigma}{(\sigma + \mu)(\gamma + \mu + \alpha)}$$

When $\mathcal{R}_0 > 1$, the system admits a biologically feasible endemic equilibrium.

5. Linear Stability Analysis

To analyze local stability, the system is linearized around equilibrium points using the Jacobian matrix. For the DFE, stability is determined by the eigenvalues of the infection subsystem:

- If all eigenvalues have negative real parts, the DFE is locally asymptotically stable.
- If at least one eigenvalue becomes positive, the system undergoes a qualitative transition toward endemicity.

The threshold condition is again governed by $\mathcal{R}_0$, which acts as a bifurcation parameter controlling system stability.

6. Catastrophe Theory Framework

Catastrophe theory provides a geometric and nonlinear framework to analyze sudden shifts in epidemic states when parameters vary continuously. In the SEIRV system, parameters such as β, ν, or ω may act as control parameters, while the infected population I serves as a state variable.

The system can be reduced (via center manifold or fast-slow decomposition) to a lower-dimensional potential form:

$$\frac{\mathrm{d}I}{\mathrm{d}t} = -\frac{\partial V(I;a,b)}{\partial I}$$

where $V(I;a,b)$ is a potential function and a,b are control parameters derived from epidemiological rates.

Typical catastrophic behaviors include:

- **Fold catastrophe**: sudden jump from disease-free to endemic state,
- **Cusp catastrophe**: bistability between low and high infection regimes,
- **Hysteresis loops**: irreversible epidemic transitions under parameter reversal.

7. Cusp Type-Catastrophe

7.1. Cusp Catastrophe Potential (Standard Form [7]-[9])

The canonical cusp potential is:

$$V(I;a,b) = \frac{1}{4}I^4 + \frac{1}{2}aI^2 + bI$$

where:

- I = state variable (infected population)
- a,b = control parameters (derived from SEIRV rates)

7.2. Dynamical System (Gradient Form)

The dynamics are:

$$\frac{\mathrm{d}I}{\mathrm{d}t} = -\frac{\mathrm{d}V}{\mathrm{d}I} = -\left(I^3 + aI + b\right)$$

So equilibrium points satisfy:

$$I^3 + aI + b = 0$$

This cubic equation is the core of cusp bifurcation behavior.

7.3. Interpretation in SEIRV Epidemic Modeling

In an SEIRV reduction:

State variable

- **I : infected class (dominant slow variable)**

Control parameters (typical mapping)

Define:

- **a : effective linear epidemic pressure:** $a \sim (\mathcal{R}_0 - 1) - c_1 \nu + c_2 \omega$
- **b : external or asymmetry forcing:** b ~importation rate, behavioral shifts, seasonal forcing

7.4. Epidemic Meaning of Cusp Geometry

The cusp catastrophe explains:

1) Bistability

Two possible stable epidemic states:

- $I = 0$ disease-free
- $I = I^* > 0$ endemic

2) Sudden outbreak jump

A small parameter change in a or b causes:

- smooth variation → then abrupt jump in infection level

3) Hysteresis

If parameters are reversed:

- system does NOT return along the same path
- epidemic persists even after $\mathcal{R}_0 < 1$

7.5. Cusp Set (Bifurcation Surface)

The cusp bifurcation occurs when equilibria merge:

$$4a^3 + 27b^2 = 0$$

Meaning:

- inside this curve → 3 equilibria (bistability)
- outside → 1 equilibrium (monostable)

7.6. Numerical SEIRV Example (Illustrative)

Let: $a = -0.2$ and $b = 0.05$

Then:

$$V(I) = \frac{1}{4} I^4 - 0.1 I^2 + 0.05 I$$

Interpretation:

- bistable regime (near cusp region)
- system may switch between: disease-free state, endemic outbreak state

7.7. Epidemic Interpretation Summary

A cusp catastrophe in SEIRV means:

- gradual changes in vaccination or transmission
- produce abrupt epidemic transitions
- system exhibits: bistability, hysteresis loops, tipping points

7.8. Key Takeaway

The cusp potential provides a geometric explanation for sudden epidemic outbreaks in SEIRV models, in which infection dynamics are governed by a quartic landscape with two control parameters, thereby capturing bistability and hysteresis [13]-[15].

8. Bifurcation and Sudden Transition Behavior

The SEIRV model exhibits rich bifurcation structures depending on vaccination and transmission intensity. In particular:

- Increasing ν (vaccination rate) shifts the system toward the disease-free regime.
- Increasing β leads to forward bifurcation and possible endemic persistence.
- When nonlinear feedback (e.g., immunity waning ω) is strong, the system may exhibit backward bifurcation, where $\mathcal{R}_0 < 1$ still allows endemic equilibria.

Catastrophe theory explains such behavior as the crossing of a fold surface in parameter space, where equilibria disappear or suddenly emerge.

9. Interpretation of Catastrophic Transitions

In epidemiological terms, catastrophe corresponds to abrupt transitions such as:

- Sudden outbreak emergence after a gradual increase in contact rate
- Rapid collapse of infection due to vaccination threshold crossing
- Coexistence of multiple stable states (endemic and disease-free)

These transitions are not smooth and cannot be fully captured by classical linear stability theory alone.

10. Dynamical Structure

The SEIRV catastrophe model reveals that:

- Epidemic systems are inherently nonlinear and multi-stable
- Control parameters can trigger discontinuous transitions
- Vaccination and immunity loss critically shape the geometry of equilibrium surfaces
- Catastrophe theory provides a unified explanation for sudden epidemic shifts

11. Conclusions

This study demonstrated that SEIRV epidemic dynamics can be effectively reinterpreted through the framework of catastrophe theory, particularly the cusp catastrophe model. By reducing the high-dimensional SEIRV system to a lower-dimensional infection subsystem, the evolution of the infected class was shown to be representable in gradient form using a potential function with quartic structure and two control parameters.

The resulting potential landscape provides a geometric interpretation of epidemic behavior, where equilibrium states correspond to critical points of the potential. In this context, the disease-free and endemic equilibria emerge as stable minima, while unstable states act as transition barriers between them. The analysis highlights that small continuous variations in epidemiological parameters—such as transmission rate, vaccination rate, and immunity waning—can lead to abrupt qualitative changes in system behavior.

In particular, the cusp catastrophe formulation explains key nonlinear phenomena observed in epidemic systems, including bistability, hysteresis, and sudden outbreak transitions. These features cannot be fully captured by classical threshold-based approaches such as basic reproduction number analysis alone. Instead, the cusp structure reveals the existence of multiple coexisting equilibria and critical tipping surfaces that govern regime shifts between disease-free and endemic states.

Overall, the integration of SEIRV modeling with catastrophe theory provides a powerful theoretical framework for understanding nonlinear epidemic transitions. It enhances the interpretability of complex disease dynamics and offers deeper insight into how control strategies such as vaccination and behavioral interventions can prevent catastrophic outbreaks. This approach opens new directions for further analytical and computational studies in nonlinear epidemiology, particularly in the study of multistability and critical transitions in infectious disease systems.

Conflicts of Interest

The author declares no conflicts of interest.

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