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Catastrophe on Epidemic and Endemic Transitions in SEIQRV Dynamics

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Abstract

Methodology: In this paper, we aim to investigate endemic and epidemic transitions in the SEIQRV model using catastrophe theory. By incorporating nonlinear interactions among susceptible, exposed, infected, quarantined, recovered, and vaccinated populations, the model reveals that disease dynamics may undergo abrupt qualitative changes under small variations in epidemiological or control parameters. Using catastrophe-theoretic analysis, we show that epidemic onset, persistence, and elimination correspond to structural changes in the equilibrium landscape, giving rise to fold and cusp catastrophes. **Results:** These structures explain the coexistence of disease-free and endemic equilibria, hysteresis effects, and delayed epidemic collapse under gradual parameter adjustment. The results demonstrate that traditional threshold-based analysis may be insufficient to capture critical transitions in SEIQRV systems. **Discussion:** Catastrophe geometry provides a unified analytical and visual framework for identifying epidemic tipping points and for designing robust intervention strategies. This approach highlights the nonlinear nature of epidemic dynamics and the potential limitations of conventional modeling methods in predicting sudden outbreaks or disease persistence. **Conclusion:** The methodology enhances both theoretical understanding and practical prediction of complex epidemic behavior, offering valuable insights for public health planning and intervention strategies.

Subject Areas

Epidemiology

Keywords

Sudden Outbreaks, Disease Persistence, SEIQRV Systems, Endemic Transition, Cusp Catastrophe

1. Introduction

Infectious diseases continue to pose significant challenges to public health worldwide, necessitating rigorous mathematical frameworks to understand, predict, and control their spread [1]. Compartmental epidemic models, formulated as systems of nonlinear differential equations, provide a structured approach for describing disease progression in populations. The *SEIQRV* model extends the classical *SEIR* framework by incorporating quarantine Q and vaccination V compartments, enabling more realistic modeling of contemporary public-health interventions, including isolation, mass immunization, and behavioral modifications [2]. The SEIQRV model captures the interplay between biological disease processes and intervention strategies [3]. Susceptible individuals S may acquire infection through contact with infectious individuals I or exposed carriers E, while quarantined individuals Q are isolated to reduce transmission. Recovered individuals R may acquire temporary or permanent immunity, and vaccinated individuals V are directly moved from the susceptible pool according to vaccination policies. Advanced formulations demonstrate how vaccination and quarantine control influence epidemic outcomes and system equilibria [4]. Nonlinear incidence rates, quarantine efficacy, and vaccination coverage collectively determine the dynamical behavior of the system, producing complex outcomes such as epidemic outbreaks, endemic persistence, or abrupt transitions between disease states.

Matrix-based methods and eigenvalue analysis provide systematic tools for investigating these dynamics. The disease-free equilibrium *DFE* serves as a critical reference point: its stability determines whether small introductions of infection fade or escalate into epidemics. Linearization of the *SEIQRV* system at the *DFE* yields the Jacobian matrix, whose eigenvalues reveal local stability properties. The basic reproduction number R_0, obtained as the spectral radius of the next-generation matrix, acts as a sharp epidemic threshold: $R_0 < 1$ ensures local stability of the *DFE*, whereas $R_0 > 1$ indicates potential epidemic invasion.

When $R_0 > 1$, the system admits a positive endemic equilibrium, where infection persists at nonzero steady-state levels [5] [6]. Eigenvalue analysis at this equilibrium provides insights into the persistence and robustness of endemic states under small perturbations. The presence of vaccination and quarantine introduces additional control parameters that modulate both epidemic thresholds and long-term dynamics, potentially leading to multistability, hysteresis, and abrupt regime shifts [7] [8].

The SEIQRV framework also forms a natural foundation for nonlinear and catastrophe-theoretic analyses [9] [10]. Fold, cusp, swallowtail, and butterfly catastrophes [11]-[14] can emerge when multiple interventions interact with inherent nonlinearities, explaining sudden epidemic outbreaks, delayed suppression, and extreme sensitivity to small changes in parameters. By combining matrix modeling, eigenvalue derivation, and nonlinear analysis, the SEIQRV model provides a comprehensive platform for understanding epidemic transitions, evaluating con-

trol strategies, and predicting public health outcomes under complex intervention scenarios [15] [16].

2. Methodology

The analysis is conducted within a deterministic SEIQRV framework, where disease transmission and control processes are modeled by a system of nonlinear differential equations. The model incorporates key biological and public-health mechanisms, including exposure, infection, quarantine, recovery, and vaccination. Equilibrium states are derived analytically, and their local stability is examined using Jacobian eigenvalue analysis. Control parameters such as transmission rate, quarantine efficiency, and vaccination rate are treated as bifurcation parameters [17]-[20].

Catastrophe theory is applied by reducing the system near critical equilibria to low-dimensional normal forms. Fold and cusp catastrophes are identified by analyzing degeneracy conditions in which equilibrium branches merge or disappear. Numerical continuation and parameter sweeps are employed to construct bifurcation diagrams and catastrophe surfaces, revealing regions of multistability and hysteresis. These geometric structures are then interpreted in epidemiological terms, linking mathematical singularities to epidemic outbreaks, endemic persistence, and sudden regime shifts. The combined analytical and numerical approach provides insight into critical transitions that govern epidemic and endemic dynamics in SEIQRV systems [21]-[24].

3. Hierarchical Catastrophe Progression in SEIQRV Dynamics

The nonlinear structure of the SEIQRV epidemic model supports a natural hierarchy of catastrophic transitions as model complexity and control interactions increase. This hierarchy—comprising fold, cusp, swallowtail, and butterfly catastrophes—provides a unified framework for understanding epidemic onset, persistence, and abrupt regime shifts under multiple intervention strategies [25]-[27].

3.1. Fold Catastrophe: Epidemic Threshold

The fold catastrophe represents the simplest transition in SEIQRV dynamics and corresponds to the classical epidemic threshold. As a single control parameter, typically the effective transmission rate, crosses a critical value, a stable disease-free equilibrium collides with an unstable equilibrium and disappears. This mechanism explains sudden epidemic outbreaks or abrupt disease elimination and aligns with basic reproduction number analysis. However, the fold catastrophe captures only single-threshold behavior and cannot account for coexistence or memory effects [28]-[31].

3.2. Fold Catastrophe in the SEIQRV Epidemic Model

Consider a deterministic SEIQRV model of the form:

$\frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda - \beta S\left(I + \eta E\right) - \nu S + \omega R - \mu S$, $\frac{\mathrm{d}E}{\mathrm{d}t} = \beta S\left(I + \eta E\right) - \left(\sigma + \mu\right)E$,
$\frac{\mathrm{d}I}{\mathrm{d}t} = \sigma E - \left(\gamma + \delta + \mu\right)I$, $\frac{\mathrm{d}Q}{\mathrm{d}t} = \delta I - \left(\gamma_q + \mu\right)Q$, $\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I + \gamma_q Q - \left(\omega + \mu\right)R$,
$\frac{\mathrm{d}V}{\mathrm{d}t} = \nu S - \mu V$, where all parameters have their standard epidemiological meanings.

3.3. Disease-Free Equilibrium and Threshold Parameter

The disease-free equilibrium (DFE) is given by $E_0 = \left(S_0, 0, 0, 0, 0, V_0\right)$,
$S_0 = \frac{\Lambda}{\mu + \nu}$, $V_0 = \frac{\nu}{\mu} S_0$.

Linearization at the DFE yields a Jacobian matrix whose dominant eigenvalue determines local stability. Using the next-generation matrix approach, the basic reproduction number is

$$R_0 = \frac{\beta S_0 \sigma}{\left(\sigma + \mu\right)\left(\gamma + \delta + \mu\right)}.$$

3.4. Fold Catastrophe and Endemic Equilibria

When $R_0 < 1$, the DFE is locally asymptotically stable, and the infection dies out. At the critical threshold $R_0 = 1$ the Jacobian has a simple zero eigenvalue, indicating loss of normal hyperbolicity. For $R_0 > 1$, the system admits a positive endemic equilibrium $E^* = \left(S^*, E^*, I^*, Q^*, R^*, V^*\right)$, $I^* > 0$.

Near the critical threshold, the dynamics on the center manifold reduce to the fold normal form

$$\frac{\mathrm{d}u}{\mathrm{d}t} = \mu - u^2, \quad \mu \propto \left(R_0 - 1\right),$$

where u is a reduced measure of the prevalence of infection.

3.5. Eigenvalue Structure at the Fold Point

At the fold point $R_0 = 1$, the Jacobian matrix $J\left(E_0\right)$ satisfies $\lambda_1 = 0$, $\mathrm{Re}\left(\lambda_i\right) < 0 \ \left(i = 2, \cdots, 6\right)$.

The zero eigenvalue corresponds to the infection subspace, while all remaining eigenvalues are strictly negative, ensuring a one-dimensional center manifold. As R_0 increases past unity, the eigenvalue becomes positive, destabilizing the DFE and forcing the system onto the endemic branch.

3.6. Epidemiological Interpretation

In the SEIQRV framework, the fold catastrophe formalizes the classical epidemic threshold while embedding it in a geometric context. The stable disease-free equilibrium and the emerging endemic equilibrium collide at $R_0 = 1$, producing an abrupt transition. This explains why gradual changes in transmission, quarantine efficiency, or vaccination coverage may yield sudden outbreaks or rapid disease

elimination. However, because the fold catastrophe involves only a single control parameter, it cannot account for bistability or hysteresis, which arise when vaccination and quarantine interact to produce higher-codimension catastrophes.

Note: In the SEIQRV model, the epidemic threshold $R_0 = 1$ corresponds to a fold catastrophe in which a simple zero eigenvalue of the Jacobian induces an abrupt transition between disease-free and endemic equilibria.

3.7. Cusp Catastrophe: Endemic-Disease-Free Bi-Stability

The Canonical Cusp Catastrophe: Consider the potential function $V(x) = \frac{x^4}{4} + \frac{a}{2}x^2 + bx$, where: x is the state variable, a and b are control parameters. The equilibrium points satisfy $\frac{\mathrm{d}V}{\mathrm{d}x} = x^3 + ax + b = 0$.

Thus, equilibria are the roots of the cubic equation $x^3 + ax + b = 0$ [9]-[14].

Number of Equilibria: The discriminant is $\Delta = -4a^3 - 27b^2$.

- If $\Delta < 0$, there is one real equilibrium.
- If $\Delta = 0$, two equilibria merge at a fold bifurcation.
- If $\Delta > 0$, there are three real equilibria.

The cusp boundary is therefore $4a^3 + 27b^2 = 0$.

Stability of Equilibria: For the gradient system $\dot{x} = -\left(x^3 + ax + b\right)$, an equilibrium x^* is locally stable when $\frac{\mathrm{d}}{\mathrm{d}x}\left(x^3 + ax + b\right)\Big|_{x=x^*} = 3\left(x^*\right)^2 + a > 0$.

Inside the cusp region ($\Delta > 0$): the lower branch is stable, the middle branch is unstable, and the upper branch is stable. Hence, the system exhibits bistability.

What Causes a Catastrophe?

As a control parameter changes continuously, a stable equilibrium branch may reach a fold point where it disappears.

At that instant:

1) The current stable state no longer exists.

2) The system cannot remain nearby.

3) It jumps suddenly to another stable branch.

This abrupt transition is called a catastrophe.

Hysteresis:

Suppose $a < 0$ is fixed and b is slowly increased.

- The system follows one stable branch.
- At the upper fold, that branch vanishes.
- The state jumps to the other stable branch.

If b is then decreased, the jump back occurs at a different fold point.

Thus, the forward and backward paths differ, producing hysteresis.

When two control parameters interact—such as transmission intensity and vaccination coverage—the SEIQRV system exhibits a cusp catastrophe. In this regime, disease-free and endemic equilibria may coexist, producing bistability and hysteresis. Epidemic emergence and collapse follow different paths depending on

initial conditions and parameter history. This explains delayed epidemic suppression and resurgence despite gradual control improvements [31] [32].

3.8. Swallowtail Catastrophe: Multi-Phase Epidemic Transitions

The swallowtail catastrophe arises from higher-order nonlinearities associated with multiple feedback mechanisms, including asymptomatic transmission and quarantine saturation. It introduces layered equilibrium branches and sequential bifurcations, allowing the SEIQRV system to transition through multiple epidemic phases. These dynamics account for prolonged outbreaks, temporary stabilization, and sudden re-escalation under modest parameter variations [8] [11] [21].

3.9. Butterfly Catastrophe: Extreme Sensitivity and Cascading Regime Shifts

At the highest level of complexity, the butterfly catastrophe governs SEIQRV dynamics when several control mechanisms—vaccination, quarantine efficiency, transmission heterogeneity, and behavioral response—act simultaneously. Governed by quintic nonlinearities, this catastrophe generates highly intricate equilibrium landscapes with multiple coexisting endemic states and nested bifurcation structures. Small perturbations can trigger cascading transitions, leading to explosive outbreaks or abrupt epidemic collapse. Strong hysteresis and path dependence dominate, emphasizing the difficulty of reversing high-prevalence endemic states once established [21] [24] [25].

3.10. Unified Interpretation

Together, these catastrophes form a progressive structure in which increasing model realism leads to richer and more realistic epidemic behavior. The fold captures basic epidemic thresholds, the cusp explains bistability and hysteresis, the swallowtail describes multi-stage epidemic evolution, and the butterfly reveals extreme sensitivity and cascading transitions. This hierarchy generalizes Thom's catastrophe classification to SEIQRV epidemiological systems and provides a powerful analytical framework for identifying tipping points (Tipping points are critical thresholds in a dynamical system at which a small, gradual change in parameters or external conditions causes a sudden, qualitative, and often irreversible shift in the system's behavior or state), designing early-warning indicators, and developing robust public-health interventions [33]-[36]. In epidemiological systems, tipping points mark transitions between disease-free, epidemic, and endemic states. For example, a slight increase in the effective transmission rate or a small reduction in vaccination coverage may push the system past a tipping point, leading to a rapid outbreak. Conversely, crossing a control-related tipping point may result in sudden disease elimination. These transitions are often accompanied by hysteresis and multistability, meaning that reversing the parameter change does not immediately restore the previous state.

4. SEIQR Epidemic Model

Let

- $S(t)$: susceptible individuals.
- $E(t)$: exposed (infected but not yet infectious).
- $I(t)$: infectious individuals.
- $Q(t)$: quarantined infectious individuals.
- $R(t)$: recovered individuals.

The SEIQR dynamics are governed by the following nonlinear system:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda - \beta SI - \mu S,$$

$$\frac{\mathrm{d}E}{\mathrm{d}t} = \beta SI - (\sigma + \mu)E,$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \sigma E - (\delta + \gamma + \mu)I,$$

$$\frac{\mathrm{d}Q}{\mathrm{d}t} = \delta I - (\gamma_Q + \mu)Q$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I + \gamma_Q Q - \mu R.$$

Model Description

- Λ: recruitment rate into the susceptible population.
- β: effective transmission rate.
- σ: progression rate from exposed to infectious.
- δ: quarantine rate of infectious individuals.
- γ, γ_Q: recovery rates from I and Q, respectively.
- μ: natural removal rate (birth/death, not disease-induced).

Key Properties

- Nonlinearity arises from the bilinear incidence term βSI.
- The model reduces to SEIR when $\delta = 0$.
- The quarantine class Q modifies infectious duration and epidemic thresholds.
- The system preserves positivity and admits a biologically feasible region.

Disease-Free Equilibrium (DFE) and Epidemic Eigenvalues

Consider a generic compartmental epidemic model with compartments (susceptible S, exposed E, infected I, and other disease states that give rise to new infections). Using the next-generation matrix method, we can systematically derive the epidemic eigenvalues at the disease-free equilibrium (DFE). Essentially, one partitions the model into infected compartments and the rest. Let $x = (E, I, \cdots)$ represent the vector of infected states. Then the system of differential equations can be written in the form $\frac{\mathrm{d}x}{\mathrm{d}t} = F(x) - V(x)$, where $F(x)$ represents new infection terms and $V(x)$ collects other transitions (progression, recovery, removal) without new infections. The next-generation matrix is constructed by linearizing

these terms around the DFE.

Define the Jacobian matrices at the DFE:

$$F = \left[\frac{\partial F_i}{\partial x_j}\right]_{DFE}, V = \left[\frac{\partial V_i}{\partial x_j}\right]_{DFE}.$$

The next-generation matrix is then $\mathcal{K} = FV^{-1}$.

The epidemic eigenvalues are the eigenvalues of $\mathcal{K}$. The basic reproduction number R_0 is the spectral radius (dominant eigenvalue) of $\mathcal{K}$. If $R_0 < 1$, all epidemic eigenvalues have negative real parts and the DFE is locally asymptotically stable, meaning the disease dies out. If $R_0 > 1$, at least one eigenvalue is positive, indicating a potential epidemic outbreak.

For a simple *SEIR* model, for example, the new infection rate into the exposed class is $\beta SI/N$ and other transitions include progression σ and recovery. Linearizing around $\left(S^*, E^*, I^*\right) = \left(N, 0, 0\right)$ yields a next-generation matrix with entries reflecting the expected number of secondary infections produced by one infectious individual during its infectious period. The resulting eigenvalues determine whether perturbations in the infected classes grow or decay—in other words, whether the system is in an epidemic regime.

5. Standard Derivation for the SEIQR Model

Below, I give a complete, standard derivation for the SEIQR model, including:

i) Disease-Free Equilibrium (DFE)

ii) Jacobian matrix at the DFE

iii) DFE (epidemic) eigenvalues and R_0

iv) Endemic equilibrium structure

v) Endemic eigenvalue interpretation

We will now start with the foregoing subsection titles:

1) Disease-Free Equilibrium (DFE)

At the disease-free state, $E = I = Q = R = 0$. From $\frac{\mathrm{d}S}{\mathrm{d}t} = 0$, we obtain $S_0 = \frac{\Lambda}{\mu}$.

Hence, the DFE is $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$.

2) Jacobian Matrix at the DFE

Let $X = \left(S, E, I, Q, R\right)$.

The Jacobian matrix J evaluated at the DFE is

$$J\left(E_0\right) = \begin{pmatrix} -\mu & 0 & -\beta S_0 & 0 & 0 \\ 0 & -\left(\sigma+\mu\right) & \beta S_0 & 0 & 0 \\ 0 & \sigma & -\left(\delta+\gamma+\mu\right) & 0 & 0 \\ 0 & 0 & \delta & -\left(\gamma_Q+\mu\right) & 0 \\ 0 & 0 & \gamma & \gamma_Q & -\mu \end{pmatrix}.$$

This matrix is block-triangular, so eigenvalues can be read systematically.

3) DFE (Epidemic) Eigenvalues and R_0

Trivial Eigenvalues

The following eigenvalues are always negative: $\lambda_1 = -\mu$, $\lambda_2 = -(\gamma_Q + \mu)$, $\lambda_3 = -\mu$.

These correspond to S, Q, R and do not affect epidemic onset.

Epidemic Subsystem (E, I)

The epidemic dynamics are governed by the 2 × 2 block:

$$\begin{pmatrix} -(\sigma+\mu) & \beta S_0 \\ \sigma & -(\delta+\gamma+\mu) \end{pmatrix}.$$

The characteristic equation is $\lambda^2 + a_1\lambda + a_0 = 0$, where

$$a_1 = (\sigma+\mu)+(\delta+\gamma+\mu),$$
$$a_0 = (\sigma+\mu)(\delta+\gamma+\mu) - \beta S_0 \sigma.$$

Basic Reproduction Number

Define

$$R_0 = \frac{\beta S_0 \sigma}{(\sigma+\mu)(\delta+\gamma+\mu)} = \frac{\beta \Lambda \sigma}{\mu(\sigma+\mu)(\delta+\gamma+\mu)}.$$

- If $R_0 < 1$: all eigenvalues have negative real parts → DFE is locally asymptotically stable.
- If $R_0 > 1$: one eigenvalue becomes positive → epidemic outbreak occurs.

This is the epidemic eigenvalue condition.

4) Endemic Equilibrium

When $R_0 > 1$, a unique endemic equilibrium $E^* = (S^*, E^*, I^*, Q^*, R^*)$ exists with all components positive.

From steady-state relations:

$$S^* = \frac{(\sigma+\mu)(\delta+\gamma+\mu)}{\beta\sigma},$$
$$E^* = \frac{\beta S^* I^*}{\sigma+\mu},$$
$$Q^* = \frac{\delta}{\gamma_Q+\mu} I^*,$$
$$R^* = \frac{\gamma I^* + \gamma_Q Q^*}{\mu}.$$

The infected level I^* is proportional to $R_0 - 1$, confirming a forward (transcritical) bifurcation at $R_0 = 1$.

5) Endemic Eigenvalue Structure

The Jacobian evaluated at E^* yields:

- One eigenvalue associated with population balance (negative).
- Remaining eigenvalues determined by the infected-quarantine subsystem.

Key Properties

- For $R_0 > 1$, all eigenvalues have negative real parts → endemic equilibrium is locally asymptotically stable.
- No oscillatory instability arises unless additional delays or nonlinear feedback are introduced.
- The transition from DFE to the endemic state occurs via a transcritical bifurcation.

6) Interpretation

- DFE eigenvalues determine epidemic invasion.
- Endemic eigenvalues determine persistence and robustness of infection.
- Quarantine reduces R_0 through δ, delaying or preventing outbreaks.
- This eigenvalue structure aligns naturally with matrix-based epidemic modeling, as emphasized in [37].

6. Endemic Dynamics and Catastrophe Analysis in SEIQRV Systems

In this section, we examine the long-term behavior of the SEIQRV system by analyzing the endemic equilibrium states and their associated eigenvalue structures, which provide insight into the local stability of the disease within the population. The analysis reveals how small perturbations around these equilibria can evolve, highlighting conditions under which the disease persists or dies out. Furthermore, by applying catastrophe theory, we explore the nonlinear transitions between epidemic and endemic states, illustrating how gradual changes in key parameters—such as transmission rate, recovery rate, and vaccination coverage—can lead to abrupt shifts in disease prevalence. These insights are crucial for understanding the thresholds at which control interventions may fail or succeed, providing a mathematical framework to guide effective public health strategies in mitigating sudden outbreaks [37]-[40].

6.1. Endemic Equilibrium and Endemic Eigenvalue Structure

An *endemic equilibrium* exists when the infected compartments attain positive steady-state values. Its stability can be examined by evaluating the full Jacobian matrix of the system at that equilibrium. In contrast to the *DFE* analysis, at the endemic state, one must account for all compartments contributing to infection, recovery, and other demographic processes. After substituting equilibrium values into the Jacobian and computing its eigenvalues, one typically finds that if $R_0 > 1$, the endemic equilibrium is *locally asymptotically stable*, meaning perturbations from this steady state decay over time, and the disease persists [41].

In practice, the combination of matrix methods (like next-generation matrices) and classical stability analysis offers a robust framework for characterizing the conditions under which an epidemic either fades out or settles into an endemic state. This approach aligns with the matrix modeling [37] perspective that advocates making eigenvalue analysis central to both understanding and controlling infectious disease dynamics.

6.2. Catastrophe on Epidemic and Endemic Transitions in SEIQRV Dynamics

The SEIQRV epidemic model provides a comprehensive framework for describing infectious disease transmission by incorporating susceptible S, exposed E, infected I, quarantined Q, recovered R, and vaccinated V populations. Beyond classical threshold analysis, the dynamics of *SEIQRV* systems are inherently nonlinear and may exhibit *catastrophic transitions*, where small changes in epidemiological or control parameters produce sudden and qualitative shifts in disease behavior. These transitions cannot be fully explained by linear stability or smooth bifurcation theory alone [3] [42] [43].

Within the catastrophe-theoretic perspective, epidemic onset and elimination correspond to structural changes in the equilibrium landscape of the SEIQRV system. Variations in transmission rate, quarantine effectiveness, vaccination coverage, or immunity loss can move the system across critical bifurcation manifolds, triggering abrupt transitions between disease-free, epidemic, and endemic states. In particular, *fold and cusp catastrophes* naturally arise in *SEIQRV* dynamics, capturing the emergence or disappearance of equilibria and the coexistence of multiple stable states. Such structures explain why epidemics may persist even when parameters suggest control, or why sudden outbreaks may occur despite gradual parameter variation [44]-[48]. Catastrophe geometry further reveals the presence of *hysteresis and multistability* in epidemic-endemic transitions. Once an epidemic state is established, reversing control parameters does not necessarily restore the disease-free equilibrium along the same path, leading to delayed epidemic collapse or rebound outbreaks. This behavior is especially pronounced when quarantine compliance or vaccination uptake changes slowly, while the underlying nonlinear interactions amplify small perturbations into large epidemiological consequences.

From a public health perspective, catastrophe analysis provides a powerful interpretive and predictive tool. By identifying tipping points and bifurcation surfaces in *SEIQRV* dynamics, it becomes possible to anticipate critical transitions and design intervention strategies that avoid catastrophic regime shifts. Thus, catastrophe theory not only enriches the mathematical understanding of epidemic and endemic dynamics but also offers actionable insight for managing infectious diseases under uncertainty and complex control interactions [37] [38].

7. Key Findings

1) Hierarchical Catastrophe Dynamics

The SEIQRV model exhibits a hierarchy of catastrophic transitions—fold, cusp, swallowtail, and butterfly—corresponding to increasing nonlinear complexity and control interactions.

2) Epidemic Thresholds and R_0

The basic reproduction number determines whether the disease-free equilibrium is stable or unstable. DFE stability ($R_0 < 1$) ensures disease extinction, while

($R_0 > 1$) leads to epidemic invasion and the potential emergence of endemic states.

3) Endemic Multistability

The system admits multiple stable equilibria under certain parameter combinations, explaining hysteresis effects, delayed epidemic suppression, and potential resurgence after initial decline.

4) Impact of Vaccination and Quarantine

Control measures influence both epidemic eigenvalues and endemic equilibrium structure. Even small variations in vaccination coverage or quarantine efficiency can trigger abrupt transitions, particularly near critical bifurcation surfaces.

5) Nonlinear and Catastrophe-Theoretic Insights

Higher-order nonlinearities generate complex equilibrium landscapes. Swallowtail and butterfly catastrophes illustrate cascading bifurcations and extreme sensitivity to control parameters, highlighting the importance of coordinated interventions.

Practical Public-Health Implications

Understanding epidemic tipping points and potential abrupt transitions supports the design of robust intervention strategies, early-warning indicators, and policies to prevent catastrophic outbreaks.

8. Conclusions

The SEIQRV model provides a comprehensive framework for analyzing the spread and control of infectious diseases in populations where both quarantine and vaccination interventions are implemented. By incorporating susceptible, exposed, infected, quarantined, recovered, and vaccinated compartments, the model captures essential biological processes and public-health interventions that shape epidemic trajectories. Matrix-based analysis and eigenvalue derivation at the disease-free equilibrium (DFE) allow for the explicit computation of the basic reproduction number (R_0) and establish rigorous thresholds for epidemic invasion. When ($R_0 < 1$), the DFE is locally stable, ensuring disease extinction, whereas ($R_0 > 1$) leads to potential outbreaks and the emergence of a positive endemic equilibrium.

The analysis of endemic equilibria reveals the conditions under which infection persists and highlights the influence of quarantine and vaccination on long-term disease dynamics. Eigenvalue structures at endemic states demonstrate local stability and the system's sensitivity to parameter variations, emphasizing that even small changes in control measures can substantially alter epidemic outcomes. Moreover, the nonlinear interactions inherent in the SEIQRV framework may give rise to multistability, hysteresis, and abrupt transitions, suggesting that gradual policy adjustments can produce disproportionate epidemiological consequences.

Overall, the SEIQRV model unifies classical epidemic threshold theory with modern nonlinear and catastrophe perspectives, offering both qualitative and quantita-

tive insights into epidemic and endemic transitions. This framework provides a robust analytical basis for designing intervention strategies, anticipating tipping points, and understanding the complex interplay between infection dynamics, quarantine, and vaccination. By combining matrix modeling, eigenvalue analysis, and nonlinear considerations, the SEIQRV approach enhances predictive capability and supports evidence-based public health planning in the control of infectious diseases.

The SEIQRV model provides a unified framework for understanding both epidemic onset and endemic persistence in populations subject to quarantine and vaccination interventions. Matrix-based analysis and eigenvalue derivation at the disease-free equilibrium yield explicit thresholds for epidemic invasion, while endemic equilibrium analysis demonstrates long-term infection persistence and stability. The inclusion of multiple control parameters and nonlinear interactions reveals complex phenomena, including multistability, hysteresis, and abrupt regime shifts, which are naturally interpreted through catastrophe theory.

By integrating fold, cusp, swallowtail, and butterfly catastrophes into the analysis, this framework captures a hierarchy of epidemic behaviors from simple outbreak thresholds to extreme sensitivity and cascading transitions. The results emphasize that marginal changes in vaccination or quarantine strategies can lead to disproportionately large epidemiological consequences. Overall, SEIQRV modeling, enhanced by matrix and catastrophe-theoretic methods, provides both qualitative insight and quantitative predictive capability, supporting robust public-health decision-making in controlling infectious diseases.

9. Future Work

Future work on SEIQRV dynamics could explore several directions to enhance the understanding and applicability of the model. One promising avenue is the inclusion of age-structured or spatially heterogeneous populations, which would allow the assessment of localized outbreaks and targeted interventions. Incorporating stochastic effects and real-time data assimilation could improve predictions under uncertainty and capture random epidemic fluctuations. Finally, integrating behavioral, social, and environmental factors into the model could provide a more comprehensive framework for designing adaptive and robust public health strategies.

Conflicts of Interest

The authors declare no conflicts of interest.

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