Bol. Soc. Paran. Mat.
©SPM – E-ISSN-2175-1188
SPM: www.spm.uem.br/bspm

(3s.) **v. 2026 (44)** : 1–14.
ISSN-0037-8712
doi:10.5269/bspm.80203

Enhancing Disease Dynamics: A Comprehensive Analysis of SIR and SIRV Models with Vaccination Impact

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ABSTRACT: This study presents a refined mathematical framework for modeling infectious disease dynamics using classical SIR and extended SIRV models, with a particular focus on the role of vaccination. We incorporate nonlinear vaccination effects and natural birth–death processes and derive a vaccination-adjusted reproduction number R_v to assess epidemic thresholds. Analytical results include new conditions for local and global stability of disease-free and endemic equilibria, supported by Lyapunov-based proofs. Sensitivity analysis quantifies the influence of key parameters such as infection rate, recovery rate, and vaccine coverage. Numerical simulations validate the theoretical findings and illustrate how increasing vaccination rates can suppress outbreaks and shift the system toward a stable endemic state. The results provide actionable insights for public health planning and epidemic control strategies.

Keywords: SIR model, SIRV model, vaccination dynamics, vaccination-adjusted reproduction number, stability analysis, sensitivity analysis, epidemic modeling.

Contents

1. Introduction

Mathematical modeling plays a fundamental role in understanding, predicting, and controlling the spread of infectious diseases. Since the pioneering work of Kermack and McKendrick [9], compartmental models such as the susceptible–infected–recovered (SIR) framework have served as a cornerstone of epidemiological analysis. These models provide valuable insights into disease transmission mechanisms, threshold conditions for outbreaks, and the effectiveness of intervention strategies.

Vaccination is one of the most effective public health measures for preventing and controlling infectious diseases. To assess its impact, the classical SIR model has been extended to include a vaccinated compartment, leading to the susceptible–infected–recovered–vaccinated (SIRV) model. SIRV-type models have been widely employed to evaluate immunization programs, estimate critical vaccination thresholds,

2020 *Mathematics Subject Classification:* 92D30, 34D20, 93D05.
Submitted November 17, 2025. Published March 28, 2026

and analyze disease elimination conditions. However, many deterministic SIRV formulations rely on simplified assumptions, such as constant vaccination rates and linear incidence functions, which may not adequately capture the complex interaction between demographic processes, vaccination coverage, and disease transmission. Vaccination is an important public health tool for managing and eliminating infectious illnesses. Recent mathematical modeling studies have demonstrated its tremendous impact on transmission dynamics across a variety of diseases. Vaccination techniques can dramatically reduce the basic reproduction number (R_0), a crucial parameter for epidemic potential [5], limiting virus propagation. This is obvious in models of Hepatitis B, where vaccination induces long-term immunological memory to reduce transmission [6]. In Monkeypox, sensitivity analyses quantify the crucial importance of vaccination in outbreak control [1]. Fractional-order models, which incorporate memory effects and complicated system dynamics, provide a fresh paradigm for researching pneumonia, exposing the subtle impact of vaccination campaigns on disease prevalence [12,13]. Advanced computational models demonstrate that combining vaccination and treatment optimizes control tactics for diseases like Hepatitis B [7].

Recent studies have sought to enrich epidemic modeling by incorporating stochastic effects, fractional-order dynamics, nonlinear vaccination mechanisms, and behavioral responses. For example, Shang and Li [10] developed a stochastic SIRV model driven by Ornstein–Uhlenbeck noise to account for environmental fluctuations and demonstrated conditions for disease extinction and persistence. He et al. [8] investigated chaotic dynamics in a fractional-order discrete-time SIRV model, revealing strong sensitivity to initial conditions and parameter variations. Dai and Wang [4] introduced a fractional SIRV model coupled with game-theoretic vaccination strategies, emphasizing the role of individual decision-making in disease control. Baba et al. [2] further showed that nonlinear vaccination efficacy and public awareness significantly influence equilibrium behavior and epidemic thresholds. Finally, optimal control theory applied to epidemic models reveals that vaccination is a cornerstone of good disease management, crucial for achieving global disease elimination targets [17].

In addition, several researchers have examined epidemic models involving fractional derivatives, time delays, and advanced sensitivity techniques. Soulaimani et al. [11] analyzed fractional SEIR models with time delays, while Barman et al. [3] employed partial rank correlation coefficient (PRCC) sensitivity analysis to identify influential parameters in Caputo-based epidemic systems. These contributions highlight the growing importance of combining analytical rigor with numerical and sensitivity analyses to better represent real-world epidemiological complexity.

Parallel to these developments, multi-strain epidemic models have attracted considerable attention. Yaagoub et al. [14] studied the global stability of a two-strain SEIR model incorporating quarantine strategies, providing insights into disease containment. Extending this work, Yaagoub et al. [15] investigated vaccination effects in a two-strain epidemic framework, demonstrating how immunization alters strain coexistence dynamics. More recently, Yaagoub et al. [16] proposed a three-strain influenza model with fractional-order derivatives and treatment effects, offering a comprehensive perspective on complex epidemic interactions. These studies motivate further investigation into vaccination-driven dynamics within deterministic epidemic frameworks.

Despite these advances, there remains a need for deterministic SIRV models that rigorously integrate birth–death processes, vaccination at birth, stability analysis, and sensitivity assessment within a unified framework. In particular, a clear analytical characterization of the vaccination-adjusted reproduction number and its dependence on key epidemiological parameters is still limited.

Motivated by this gap, the present study develops and analyzes a deterministic SIR and SIRV model with vaccination at birth. The main contributions of this work are as follows: (i) derivation of a vaccination-adjusted reproduction number R_v using the next-generation matrix approach; (ii) analysis of local and global stability of equilibrium points using Lyapunov methods; (iii) sensitivity analysis to quantify the influence of model parameters on R_v; and (iv) numerical simulations to illustrate and validate the analytical results. The findings provide theoretical insight into vaccination effectiveness and offer practical implications for public health planning. A list of abbreviations used throughout the manuscript is provided in Table 4.

2. Preliminaries

We consider a closed population of constant size $N(t)$, partitioned into four mutually exclusive compartments: susceptible $S(t)$, infected $I(t)$, recovered $R(t)$, and vaccinated $V(t)$. The classical SIR model includes $S(t)$, $I(t)$, and $R(t)$, while the extended SIRV model incorporates $V(t)$ to account for immunization at birth. The total population satisfies $N(t) = S(t) + I(t) + R(t)$ for SIR and $N(t) = S(t) + I(t) + R(t) + V(t)$ for SIRV.

We assume demographic equilibrium with equal birth and death rates $\alpha = \mu$, homogeneous mixing, no age or spatial structure, and permanent immunity upon recovery or vaccination. A proportion $\rho \in [0, 1]$ of newborns are vaccinated at birth and enter the $V(t)$ class, while the remaining fraction $1 - \rho$ enter $S(t)$. Disease transmission follows a standard incidence form $\beta \frac{SI}{N}$, with recovery rate γ. All variables and parameters are nonnegative, continuously differentiable, and biologically meaningful. Initial conditions satisfy $S(0), I(0), R(0), V(0) \geq 0$ and $S(0) + I(0) + R(0) + V(0) = N$.

Two equilibrium states are central to the analysis: the disease-free equilibrium (DFE), where infection is absent, and the endemic equilibrium (EE), where infection persists. Their existence and stability depend on a threshold quantity—the reproduction number—which will be formally derived in later sections. The basic reproduction number R_0 applies to the SIR model, while the vaccination-adjusted reproduction number R_v reflects reduced susceptibility in the SIRV framework.

Sensitivity analysis quantifies how variations in parameters affect epidemic outcomes. Normalized sensitivity indices will be used to evaluate the influence of β, γ, μ, and ρ on R_v, guiding control strategies and policy decisions. For reference, the model variables and parameters used throughout the manuscript are summarized in Table 1.

Table 1: Summary of model variables and parameters used in the SIR and SIRV frameworks.

Symbol	Description
$S(t)$	Number of susceptible individuals at time t
$I(t)$	Number of infected individuals at time t
$R(t)$	Number of recovered individuals at time t
$V(t)$	Number of vaccinated individuals at time t
$N(t)$	Total population at time t
α	Birth rate
μ	Natural death rate
β	Transmission rate
γ	Recovery rate
ρ	Proportion of newborns vaccinated

Definition 2.1 (Picard–Lindelöf Theorem) Let $\mathbf{x}' = \mathbf{f}(t, \mathbf{x})$ be a system of ordinary differential equations defined on a domain $D \subset \mathbb{R} \times \mathbb{R}^n$, where $\mathbf{f}$ is continuous in t and locally Lipschitz continuous in $\mathbf{x}$. Then, for any initial condition $\mathbf{x}(t_0) = \mathbf{x}_0 \in D$, there exists a unique solution $\mathbf{x}(t)$ defined on a maximal interval $[t_0, T_{\max})$, where $T_{\max} > t_0$.

Remark 2.1 (Comparison Principle) Let $y(t)$ satisfy the differential inequality $\frac{dy}{dt} \geq -\mu y$ with $y(0) \geq 0$. Then $y(t) \geq y(0)e^{-\mu t} \geq 0$ for all $t \geq 0$. This principle allows us to establish lower bounds for solutions by comparing them to simpler differential equations with known behavior.

Definition 2.2 (Basic Reproduction Number) The basic reproduction number R_0 is defined as the expected number of secondary infections produced by a single infected individual in a completely susceptible population. It serves as a threshold parameter: if $R_0 < 1$, the infection dies out; if $R_0 > 1$, the infection can invade and persist.

Definition 2.3 (Vaccination-Adjusted Reproduction Number) In models with vaccination, the reproduction number is adjusted to account for reduced susceptibility.

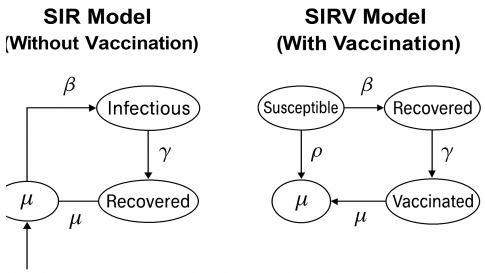

Figure 1: Compartmental flow diagram of the SIR and SIRV models. Newborn individuals enter the population at rate αN, with a proportion ρ vaccinated at birth and entering the vaccinated class V, while the remaining proportion enters the susceptible class S. Susceptible individuals become infected through standard incidence transmission, infected individuals recover at rate γ, and all compartments experience natural death at rate μ.

Definition 2.4 (Equilibrium States) An equilibrium point of a compartmental model is a state where all derivatives vanish. The disease-free equilibrium (DFE) corresponds to $I = 0$, indicating no infection. The endemic equilibrium (EE) satisfies $I > 0$, indicating persistent infection. Stability of these equilibria depends on the reproduction number.

Remark 2.2 (Threshold Principle) The reproduction number acts as a bifurcation parameter. If $R < 1$, the disease-free equilibrium is stable and the infection dies out. If $R > 1$, the system admits an endemic equilibrium and the infection persists. This principle applies to both R_0 and R_v.

Definition 2.5 (Local Asymptotic Stability) An equilibrium point $\mathbf{x}^*$ of a dynamical system is locally asymptotically stable if all eigenvalues of the Jacobian matrix evaluated at $\mathbf{x}^*$ have negative real parts. This implies that solutions starting sufficiently close to $\mathbf{x}^*$ converge to it as $t \to \infty$.

Definition 2.6 (Lyapunov Function) A Lyapunov function $L(x)$ is a scalar function used to assess stability. If $L(x) > 0$ and $\frac{dL}{dt} < 0$ in a neighborhood of an equilibrium, then the equilibrium is globally asymptotically stable.

3. Model Formulation

In this study, we formulate two deterministic compartmental epidemic models: the classical Susceptible-Infected-Recovered (SIR) model and an extended Susceptible-Infected-Recovered-Vaccinated (SIRV) model incorporating vaccination at birth. In both frameworks, the total population is divided into epidemiologically meaningful compartments: susceptible individuals $S(t)$, infected individuals $I(t)$, recovered individuals $R(t)$, and, in the SIRV model, vaccinated individuals $V(t)$, as summarized in Table 1.

The interaction between these compartments, including demographic processes, disease transmission, recovery, and vaccination, is illustrated schematically in Figure 1. This diagram provides the structural foundation for the mathematical formulation of both models.

The total population size $N(t)$ is assumed to remain constant over time. Specifically,

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

for the SIR model, and

$$N(t) = S(t) + I(t) + R(t) + V(t)$$

for the SIRV model. This assumption holds under the demographic equilibrium condition $\alpha = \mu$, where α and μ denote the per capita birth and natural death rates, respectively (see Theorem 2.2). Under this

condition, the biologically feasible region

$$\Omega = \left\{ x \in \mathbb{R}^n_+ \;\middle|\; \sum_i x_i = N \right\}$$

is positively invariant, ensuring boundedness and global existence of solutions (cf. Theorems 3.1 and 3.2).

Vaccination is incorporated through a control parameter $\rho \in [0, 1]$, representing the proportion of newborns who are vaccinated immediately upon entry into the population and thus move directly into the vaccinated compartment $V(t)$. Consequently, a fraction $1 - \rho$ of newborns enter the susceptible class $S(t)$. When $\rho = 0$, the SIRV model reduces to the classical SIR model. In contrast, when $\rho = 1$, all newborns are vaccinated, effecti

3.1. SIR Model (Without Vaccination)

The classical Susceptible–Infected–Recovered (SIR) model partitions the population into three epidemiological compartments: susceptible individuals $S(t)$, infected individuals $I(t)$, and recovered individuals $R(t)$. The dynamics of disease transmission are governed by the following system of ordinary differential equations:

$$\frac{dS}{dt} = \alpha N - \beta \frac{SI}{N} - \mu S, \tag{3.1}$$

$$\frac{dI}{dt} = \beta \frac{SI}{N} - (\gamma + \mu) I, \tag{3.2}$$

$$\frac{dR}{dt} = \gamma I - \mu R, \tag{3.3}$$

where α denotes the per capita birth rate, μ represents the natural death rate, β is the effective transmission rate, and γ is the recovery rate.

New individuals enter the susceptible class through births at rate αN. Susceptible individuals acquire infection through effective contact with infected individuals, modeled using standard incidence $\beta SI/N$. Infected individuals recover at rate γ, while all compartments experience natural mortality at rate μ. Recovered individuals are assumed to acquire permanent immunity.

3.2. SIRV Model (With Vaccination)

To incorporate vaccination into the disease dynamics, the classical SIR model is extended by introducing a fourth compartment $V(t)$, which represents vaccinated individuals. A proportion $\rho \in [0, 1]$ of newborns is assumed to be successfully vaccinated at birth and enters directly into the vaccinated class, while the remaining fraction $1 - \rho$ enters the susceptible class.

The resulting SIRV model is governed by the following system of differential equations:

$$\frac{dS}{dt} = \alpha N(1 - \rho) - \beta \frac{SI}{N} - \mu S, \tag{3.4}$$

$$\frac{dI}{dt} = \beta \frac{SI}{N} - (\gamma + \mu) I, \tag{3.5}$$

$$\frac{dR}{dt} = \gamma I - \mu R, \tag{3.6}$$

$$\frac{dV}{dt} = \alpha N \rho - \mu V. \tag{3.7}$$

The total population size at time t is given by

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

which remains constant for all $t \geq 0$ under the demographic equilibrium assumption $\alpha = \mu$.

In this formulation, vaccination confers complete and lifelong immunity, and vaccinated individuals do not participate in the transmission process. The model reduces to the classical SIR system when $\rho = 0$,

while $\rho = 1$ corresponds to full vaccination of all newborns, thereby preventing disease invasion. These properties make the SIRV framework suitable for threshold analysis and evaluation of vaccination-based control strategies.

Theorem 3.1 (Existence and Uniqueness) *Let the initial conditions satisfy $S(0), I(0), R(0) \geq 0$ for the SIR model and $S(0), I(0), R(0), V(0) \geq 0$ for the SIRV model. Then each system admits a unique solution $(S(t), I(t), R(t))$ and $(S(t), I(t), R(t), V(t))$, respectively, defined on a maximal interval $[0, T_{\max})$, where $T_{\max} > 0$.*

Proof: Both the SIR and SIRV models are systems of autonomous ordinary differential equations defined on the nonnegative orthant $\mathbb{R}^n_+$. All model parameters are assumed to be positive, and the right-hand sides of the systems consist of polynomial and rational functions that are continuously differentiable with respect to the state variables.

In particular, the nonlinear incidence term $\beta SI/N$ is smooth and locally Lipschitz continuous on $\mathbb{R}^n_+$, since the total population $N(t)$ remains constant and strictly positive under the demographic balance condition.

By the Picard–Lindelöf Theorem (Definition 2.1), the local Lipschitz continuity of the vector fields guarantees the existence and uniqueness of a local solution for each model corresponding to any nonnegative initial condition.

Moreover, the solutions remain bounded for all $t \geq 0$ due to the invariance of the biologically feasible region and the boundedness of the total population. Consequently, no finite-time blow-up occurs, and the local solutions can be extended to global solutions on $[0, T_{\max})$ with $T_{\max} = \infty$.

Therefore, both the SIR and SIRV models admit unique, globally defined solutions. □

Theorem 3.2 (Positivity of Solutions) *Let the initial conditions of the SIR and SIRV models be nonnegative. Then all state variables remain nonnegative for all $t \geq 0$.*

Proof: We prove the result for the SIRV model; the argument for the SIR model follows analogously. Consider first the vaccinated compartment $V(t)$, which satisfies

$$\frac{dV}{dt} = \rho\alpha N - \mu V.$$

This is a linear ordinary differential equation with nonnegative input and exponential decay. Its explicit solution is given by

$$V(t) = V(0)e^{-\mu t} + \rho\alpha N \int_0^t e^{-\mu(t-\tau)}\, d\tau \geq 0,$$

for all $t \geq 0$, provided $V(0) \geq 0$.

Next, consider the susceptible compartment $S(t)$. From the model equations, we obtain

$$\frac{dS}{dt} = \alpha N(1-\rho) - \beta\frac{SI}{N} - \mu S \geq -\mu S,$$

since $\alpha N(1-\rho) \geq 0$ and $\beta SI/N \geq 0$. By the Comparison Principle (Remark 2.1), it follows that

$$S(t) \geq S(0)e^{-\mu t} \geq 0,$$

for all $t \geq 0$.

Similarly, the infected compartment satisfies

$$\frac{dI}{dt} = \beta\frac{SI}{N} - (\gamma+\mu)I \geq -(\gamma+\mu)I,$$

which implies

$$I(t) \geq I(0)e^{-(\gamma+\mu)t} \geq 0.$$

Finally, the recovered compartment satisfies

$$\frac{dR}{dt} = \gamma I - \mu R \geq -\mu R,$$

since $\gamma I \geq 0$. Hence,

$$R(t) \geq R(0)e^{-\mu t} \geq 0.$$

Therefore, all state variables of the SIRV model remain nonnegative for all $t \geq 0$. The same reasoning applies to the SIR model, and the proof is complete. □

4. Reproduction Numbers, Disease-Free and Endemic Equilibria

We now derive the reproduction numbers for the classical SIR and extended SIRV models, and analyze the conditions under which disease-free and endemic equilibria exist. The reproduction number serves as a threshold parameter that governs the long-term behavior of the epidemic (see Definitions 2.2 and 2.3).

4.1. Basic and Vaccination-Adjusted Reproduction Numbers

The basic reproduction number R_0 (Definition 2.2) represents the expected number of secondary infections caused by a single infected individual in a fully susceptible population. For the SIR model, the infection equation is

$$\frac{dI}{dt} = \beta \frac{SI}{N} - (\gamma + \mu)I.$$

At the onset of an outbreak, $S \approx N$, yielding

$$\frac{dI}{dt} = (\beta - (\gamma + \mu))\, I,$$

and thus the threshold quantity

$$R_0 = \frac{\beta}{\gamma + \mu}.$$

In the SIRV model, a fraction ρ of newborns are vaccinated and enter the immune class $V(t)$, reducing the effective susceptible population to $S = (1 - \rho)N$. Substituting into the infection equation gives

$$\frac{dI}{dt} = (\beta(1 - \rho) - (\gamma + \mu))\, I,$$

leading to the vaccination-adjusted reproduction number (Definition 2.3)

$$R_v = \frac{\beta(1 - \rho)}{\gamma + \mu}.$$

This quantity reflects the impact of immunization on transmission potential. According to the threshold principle (Remark 2.2), if $R_v < 1$, the disease cannot invade the population; if $R_v > 1$, an endemic equilibrium may emerge.

4.2. Equilibria of the SIR and SIRV Models

Theorem 4.1 (DFE and EE for SIR Model) *The SIR model admits:*

- *A disease-free equilibrium (DFE) $E_0 = (N, 0, 0)$, which is locally asymptotically stable if $R_0 < 1$.*
- *An endemic equilibrium (EE) $E_1 = (S^*, I^*, R^*)$ with $I^* > 0$ if and only if $R_0 > 1$.*

Proof: By Definition 2.4, equilibrium points satisfy $\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$. At DFE, we set $I^* = 0$ and obtain $S^* = N$, $R^* = 0$. For EE, assume $I^* > 0$. From the infected equation:

$$\beta \frac{S^* I^*}{N} = (\gamma + \mu)I^* \Rightarrow S^* = \frac{N(\gamma + \mu)}{\beta}.$$

Substituting into the population constraint $S^* + I^* + R^* = N$ yields $I^* > 0$ if and only if $R_0 > 1$, as defined in Definition 2.2 and justified by Remark 2.2. □

Theorem 4.2 (DFE and EE for SIRV Model) *The SIRV model admits:*

- *A disease-free equilibrium* $E_0 = ((1-\rho)N, 0, 0, \rho N)$*, which is locally asymptotically stable if* $R_v < 1$.
- *An endemic equilibrium* $E_1 = (S^*, I^*, R^*, V^*)$ *with* $I^* > 0$ *if and only if* $R_v > 1$.

Proof: Using Definition 2.4, the DFE satisfies $I^* = 0$, yielding $S^* = (1-\rho)N$, $R^* = 0$, $V^* = \rho N$. For EE, assume $I^* > 0$. From the infected equation:

$$\beta \frac{S^* I^*}{N} = (\gamma + \mu) I^* \Rightarrow S^* = \frac{N(\gamma + \mu)}{\beta}.$$

To ensure $S^* < (1-\rho)N$, we require $R_v > 1$, as defined in Definition 2.3 and supported by Remark 2.2. The vaccinated compartment satisfies $V^* = \rho N$ under $\alpha = \mu$, and the remaining states follow from the population constraint. □

4.3. Interconnection Between SIR and SIRV Models

The SIRV model generalizes the SIR model via the vaccination parameter $\rho \in [0, 1]$. When $\rho = 0$, $R_v = R_0$ and the SIRV model reduces to the classical SIR model. As $\rho \to 1$, $R_v \to 0$, eliminating the susceptible class and preventing disease invasion. Thus, vaccination acts as a bifurcation control mechanism that shifts the system from endemic to disease-free regimes, consistent with the threshold principle in Remark 2.2.

4.4. Stability Analysis of Equilibria

Theorem 4.3 (Local Stability of DFE for SIR Model) *The disease-free equilibrium* $E_0 = (N, 0, 0)$ *is locally asymptotically stable if* $R_0 < 1$*, and unstable if* $R_0 > 1$.

Proof: Linearizing the system around E_0 yields the Jacobian matrix:

$$J_{\text{SIR}}(E_0) = \begin{bmatrix} -\mu & -\beta & 0 \\ 0 & \beta - (\gamma + \mu) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}.$$

The eigenvalues are $\lambda_1 = -\mu$, $\lambda_2 = -\mu$, and $\lambda_3 = \beta - (\gamma + \mu)$. By Definition 2.5, E_0 is locally asymptotically stable if all eigenvalues are negative, which occurs when $R_0 < 1$ (Definition 2.2). □

Theorem 4.4 (Local Stability of DFE for SIRV Model) *The disease-free equilibrium* $E_0 = ((1-\rho)N, 0, 0, \rho N)$ *is locally asymptotically stable if* $R_v < 1$*, and unstable if* $R_v > 1$.

Proof: Linearizing the infected equation around E_0 gives:

$$\frac{dI}{dt} = [\beta(1-\rho) - (\gamma + \mu)]\, I.$$

The growth rate λ is negative if and only if $R_v < 1$ (Definition 2.3). Hence, E_0 is locally asymptotically stable when $R_v < 1$, as per Definition 2.5. □

Theorem 4.5 (Global Stability of DFE for SIRV Model) *The disease-free equilibrium of the SIRV model is globally asymptotically stable in* Ω *if* $R_v < 1$.

Proof: Define the Lyapunov function $L(I) = I$ as in Definition 2.6. Then:

$$\frac{dL}{dt} = \frac{dI}{dt} = \beta \frac{SI}{N} - (\gamma + \mu) I \leq [\beta(1-\rho) - (\gamma + \mu)]\, I.$$

If $R_v < 1$, then $\frac{dL}{dt} < 0$ for all $I > 0$, implying $I(t) \to 0$ as $t \to \infty$. Thus, the system converges to zero. □

5. Sensitivity Analysis of the Vaccination-Adjusted Reproduction Number

To assess the influence of key epidemiological parameters on disease transmission, we perform a sensitivity analysis of the vaccination-adjusted reproduction number,

$$R_v = \frac{\beta(1-\rho)}{\gamma+\mu},$$

where β is the transmission rate, ρ is the vaccination coverage, γ is the recovery rate, and μ is the natural death rate. This quantity represents the expected number of secondary infections produced by a single infected individual in a partially vaccinated population.

Understanding how R_v responds to parameter variation helps identify which factors most strongly affect disease propagation and which interventions are most effective. We adopt the normalized forward sensitivity index, which quantifies the relative change in a model output Ψ with respect to a proportional change in a parameter p:

$$\Upsilon_p^{\Psi} = \frac{\partial \Psi}{\partial p} \cdot \frac{p}{\Psi}.$$

Applying this formulation to R_v yields the following sensitivity indices:

$$\Upsilon_{\beta}^{R_v} = 1, \quad \Upsilon_{\rho}^{R_v} = -\frac{\rho}{1-\rho}, \quad \Upsilon_{\gamma}^{R_v} = -\frac{\gamma}{\gamma+\mu}, \quad \Upsilon_{\mu}^{R_v} = -\frac{\mu}{\gamma+\mu}.$$

To quantify the relative importance of each parameter, we evaluate these indices at the baseline values $\beta = 0.6$, $\rho = 0.7$, $\gamma = 0.2$, and $\mu = 0.01$. The numerical results are summarized in Table 2.

Table 2: Sensitivity indices of R_v with respect to key epidemiological parameters.

Parameter	**Baseline Value**	**Sensitivity Index**
Transmission rate β	0.6	$+1.000$
Vaccination coverage ρ	0.7	-2.333
Recovery rate γ	0.2	-0.952
Natural death rate μ	0.01	-0.048

The index $\Upsilon_{\beta}^{R_v} = 1$ shows that R_v is linearly proportional to β; hence, a 10% increase in transmission rate results in a 10% rise in R_v. Conversely, vaccination coverage exhibits a strong nonlinear suppressive effect, as indicated by

$$\Upsilon_{\rho}^{R_v} = -\frac{\rho}{1-\rho} = -2.333.$$

As ρ approaches unity, this index becomes increasingly negative, revealing that even small increases in vaccination near full coverage lead to disproportionately large reductions in disease spread.

The indices for recovery and natural death rates are both negative, reflecting their mitigating influence on transmission through shortened infectious periods. However, their effects are bounded by the denominator $(\gamma+\mu)$ and therefore less dominant than vaccination.

From a biological standpoint, these results underscore vaccination as the most effective nonlinear control mechanism for reducing R_v. While improving recovery or increasing mortality reduces transmission, their contributions remain limited compared to vaccination's capacity to remove susceptible individuals from the transmission chain. Sensitivity analysis thus provides a quantitative foundation for optimizing control strategies and prioritizing resource allocation in immunization programs.

6. Numerical Simulation and Interpretation

To validate the theoretical analysis, numerical simulations of the SIRV model were conducted using the parameter set and initial conditions presented in Table 3. A vaccination coverage of $\rho = 0.7$ and a basic reproduction number of $R_v = 4.6$ were used, satisfying the condition $R_v > 1$, which guarantees the existence of a unique endemic equilibrium as established in Theorem 4.2.

Table 3: Model parameters and baseline values used for numerical simulations.

Parameter	Description	Baseline Value
β	Transmission rate per contact per unit time	0.6
γ	Recovery rate of infected individuals	0.2
α	Natural birth rate	0.01
μ	Natural death rate	0.01
ρ	Vaccination rate of susceptible individuals	0.7
$S(0)$	Initial proportion of susceptible individuals	0.25
$I(0)$	Initial proportion of infected individuals	0.05
$R(0)$	Initial proportion of recovered individuals	0.00
$V(0)$	Initial proportion of vaccinated individuals	0.70
$t \in [0, 200]$	Simulation time interval (days)	—

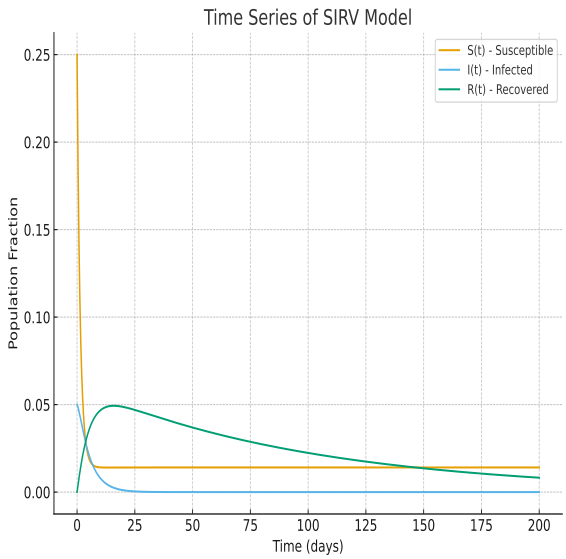

Figure 2: Time–series dynamics of the SIRV model showing the temporal evolution of $S(t)$, $I(t)$, and $R(t)$ populations under $\beta = 0.6$, $\gamma = 0.2$, $\alpha = 0.01$, and $\rho = 0.7$. The system converges to an endemic equilibrium.

Figure 2 illustrates the time-series evolution of susceptible, infected, and recovered populations. The susceptible population decreases steadily due to infection and vaccination, while the infected population initially increases, peaks, and eventually declines as immunity accumulates. The recovered class grows monotonically, representing the buildup of herd immunity. These patterns confirm convergence toward an endemic steady state, consistent with the global stability conditions derived in Theorem 4.5.

A magnified view of the infected population is shown in Figure 3. The initial oscillations represent transient fluctuations, after which the infection level stabilizes smoothly, validating the analytical prediction that infection remains bounded and tends toward equilibrium.

Figures 4 and 5 display phase portraits that provide geometric insight into the model's stability properties. In Figure 4, the trajectory of $I(t)$ against $S(t)$ forms a spiral that converges to the endemic equilibrium point, confirming local asymptotic stability as established in Theorem 4.3.

In Figure 5, the combined immune population $(I(t) + R(t))$ is plotted against $S(t)$, depicting the depletion of the susceptible pool and the accumulation of immunity. The smooth transition from the disease-free region to the endemic basin of attraction reflects biologically realistic epidemic progression.

Overall, the numerical results confirm the analytical thresholds derived for R_v and illustrate how vac-

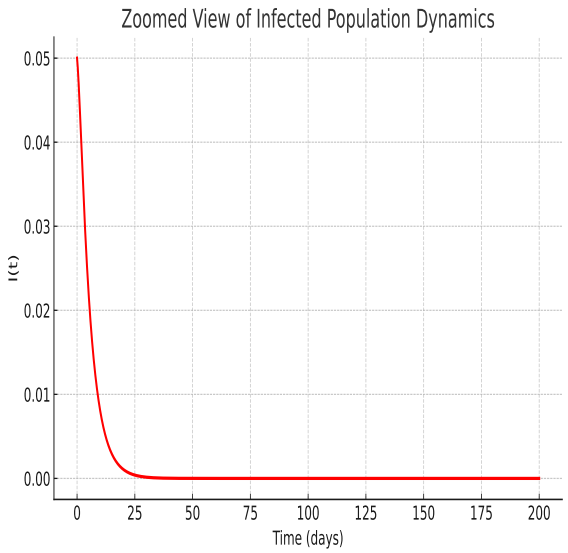

Figure 3: Zoomed view of the infected population $I(t)$ showing transient oscillations and eventual stabilization at the endemic equilibrium.

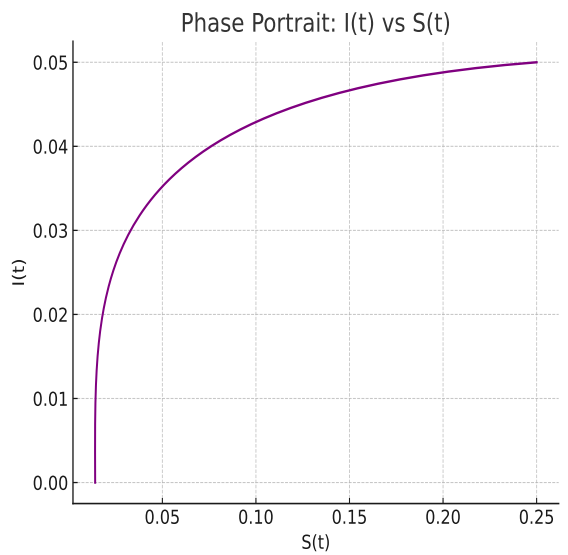

Figure 4: Phase portrait of infected $I(t)$ versus susceptible $S(t)$ showing convergence toward the endemic equilibrium point.

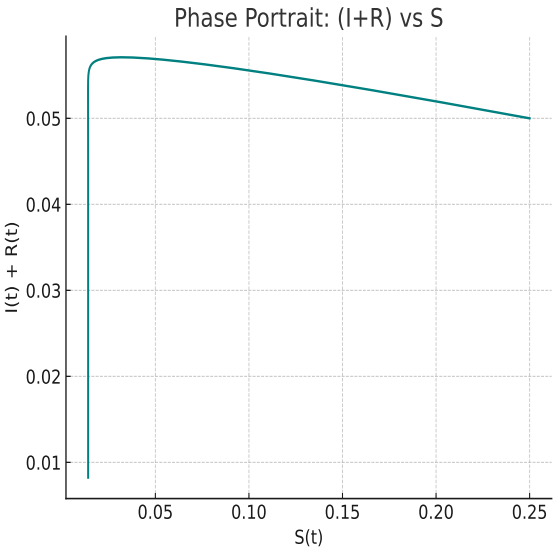

Figure 5: Phase portrait of $(I(t)+R(t))$ versus $S(t)$ showing the system's transition from the disease-free to endemic region.

cination profoundly influences epidemic dynamics. Sustained immunization not only suppresses infection peaks but also drives the system toward a stable endemic state. This validates the SIRV framework as a biologically consistent and mathematically reliable tool for designing and evaluating vaccination strategies in infectious disease control.

7. Conclusion

In this study, we formulated and analyzed two deterministic compartmental models for infectious disease transmission: the classical SIR model and its vaccination-augmented extension, the SIRV model. Both models were developed under standard epidemiological assumptions, including constant population size, homogeneous mixing, and permanent immunity following recovery or vaccination. The analytical framework established the existence, uniqueness, and positivity of solutions, and identified biologically feasible invariant regions that confine the system dynamics within realistic epidemiological bounds.

The basic reproduction number R_0 and the vaccination-adjusted reproduction number R_v were derived to characterize epidemic thresholds in the absence and presence of vaccination, respectively. Rigorous stability analyses of both disease-free and endemic equilibria were conducted using Jacobian linearization and Lyapunov functional methods, yielding explicit conditions that govern transitions between disease eradication and persistence. These results provide a clear theoretical foundation for understanding how vaccination alters threshold behavior and stability in epidemic systems.

A comprehensive sensitivity analysis identified the transmission rate β, vaccination coverage ρ, and recovery rate γ as the primary determinants of epidemic dynamics. In particular, vaccination coverage acts as a nonlinear but decisive suppressor of transmission, where modest increases near high coverage levels lead to disproportionately large reductions in the effective reproduction number. In contrast, the transmission rate remains the dominant amplifier of epidemic potential.

Numerical simulations further corroborated the analytical findings and offered intuitive visualizations of the system dynamics. Time-series solutions and phase portraits demonstrated convergence toward endemic equilibria under parameter regimes with $R_v > 1$, while also illustrating how sustained immunization dampens transient oscillations and significantly reduces peak infection levels. The close agreement between analytical predictions and numerical results confirms the biological plausibility and practical relevance of the SIRV framework for evaluating vaccination strategies.

Overall, this work presents a coherent and integrated framework that bridges rigorous mathematical analysis with epidemiological interpretation. By combining threshold analysis, sensitivity assessment, and numerical simulation, the study provides actionable insights that can inform public health decision-making, particularly in the design and prioritization of vaccination programs aimed at reducing transmission potential and preventing recurrent outbreaks.

Future research may enhance the realism and policy relevance of the model by incorporating waning immunity, heterogeneous contact structures (such as age- or spatial-based mixing), optimal control formulations for time-dependent vaccination strategies, and stochastic perturbations to capture demographic and environmental variability. Such extensions would improve applicability to real-world settings and support the development of adaptive, data-informed disease control policies.

List of Abbreviations

Table 4: List of abbreviations used throughout the manuscript.

Abbreviation	**Full Term**
SIR	Susceptible–Infected–Recovered model
SIRV	Susceptible–Infected–Recovered–Vaccinated model
DFE	Disease-Free Equilibrium
EE	Endemic Equilibrium
R_0	Basic Reproduction Number
R_v	Vaccination-Adjusted Reproduction Number
PRCC	Partial Rank Correlation Coefficient

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