

Stability Analysis of an Epidemic Model with Three Infected Compartments

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ABSTRACT

This paper proposes a deterministic compartmental epidemic model incorporating three infected classes to capture multistage disease progression and heterogeneous infectiousness. The model explicitly accounts for neonatal vaccination by introducing a fraction of newborns immunized at birth, thereby reducing the inflow into the susceptible population. Using the next-generation matrix approach, an explicit expression for the basic reproduction number $\mathcal{R}_0$ is derived. Rigorous qualitative analysis is conducted to establish the existence, positivity, boundedness, and local stability of both disease-free and endemic fixed points. It is shown that the disease-free fixed point is locally asymptotically stable when $\mathcal{R}_0 < 1$, whereas a positive endemic fixed point exists and becomes locally asymptotically stable when $\mathcal{R}_0 > 1$. Numerical simulations are performed to support the theoretical results and to explore the epidemiological impact of neonatal vaccination. The results indicate that increasing vaccination coverage significantly reduces infection prevalence, suppresses epidemic peaks, and enhances convergence toward the fixed point, even when endemicity persists. These findings highlight the critical role of early-life immunization as an effective public health strategy for controlling multistage infectious diseases and mitigating long-term epidemic burden.

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1. Introduction

Mathematical modeling has become an indispensable tool for understanding the transmission dynamics of infectious diseases and for supporting evidence-based public health decision-making. By capturing the interactions among susceptible, infected, and recovered individuals, epidemic models provide quantitative insights into disease spread, intervention effectiveness, and long-term population-level outcomes. Such models have played a crucial role in informing strategies for controlling diseases such as COVID-19, tuberculosis, hepatitis, and influenza. (Baqi et al., 2023; Bjørnstad et al., 2020; Khalaf & Flayyih, 2023; McKibbin & Fernando, 2023).

Vaccination remains one of the most effective and cost-efficient interventions for preventing infectious diseases. However, vaccine-induced immunity is not always permanent, and vaccine coverage is often incomplete due to logistical, economic, or social constraints. In particular, neonatal vaccination—administered shortly after birth—has received increasing attention because of its potential to reduce early-life susceptibility and interrupt disease transmission chains at an early stage. Despite its importance, the epidemiological impact of neonatal vaccination in complex multi-stage infection processes remains insufficiently explored (Dan & Juan, 2025).

In recent years, numerous mathematical models have been proposed to investigate epidemic dynamics under vaccination strategies. Classical compartmental frameworks such as SIR, SEIR, and their extensions have been widely studied to capture various biological and epidemiological mechanisms (Alridha et al., 2022; Din et al., 2020; Egonmwan & Okuonghae, 2019; Hadjadj & Halimi, 2023; Khan et al., 2019; Lavanchy, 2004; Means et al., 2020; Ochieng, 2025; Ottaviano et al., 2022; Pan & Zhang, 2022; Rafiq et al., 2021; Tadmon & Foko, 2019). Several authors have incorporated nonlinear incidence rates, treatment effects, and multiple infectious stages to better represent real-world disease progression (Alzaid et al., 2022; Bolatova et al., 2024; Chae & Tak, 2023; Ghasemi & Gouran, 2022; Sánchez et al., 2020; Sangotola et al., 2024; Xu et al., 2019; Yoda et al., 2024; Youssef et al., 2022). Nevertheless, many existing studies either neglect treatment failure and drug resistance or consider simplified infection structures that may underestimate long-term disease persistence. To the best of our knowledge, no previous study has simultaneously incorporated neonatal vaccination and treatment-induced

drug resistance within a three-stage infection framework and derived explicit analytical stability conditions.

Motivated by these limitations, this study develops a compartmental epidemic model that incorporates three infectious classes, representing untreated infection, treated infection, and drug-resistant infection, together with neonatal vaccination. This structure allows the model to capture heterogeneous infection pathways and treatment outcomes in a biologically realistic manner. Unlike classical models, our framework explicitly accounts for transitions between treatment success and failure, as well as waning immunity, thereby offering a more comprehensive representation of disease dynamics.

The main contributions of this work are threefold. First, we formulate a novel multi-compartment epidemic model incorporating neonatal vaccination and treatment-induced transitions. Second, we rigorously analyze the model by deriving the basic reproduction number using the next-generation matrix approach and establishing the local stability of both disease-free and endemic equilibria. Third, we perform numerical simulations to investigate the epidemiological impact of vaccination coverage on disease prevalence and persistence.

The results demonstrate that increasing neonatal vaccination coverage significantly reduces infection levels and can suppress epidemic outbreaks even in the presence of drug resistance. These findings provide valuable insights for public health policymakers seeking to design effective vaccination strategies in resource-limited settings.

The remainder of this paper is organized as follows. Section 2 presents the model formulation and underlying assumptions. Section 3 presents the analytical results, including fixed-point analysis, stability conditions, and numerical simulations. Finally, Section 4 presents the conclusions and discusses the epidemiological implications of our mathematical findings.

2. Methodology

To achieve the objectives outlined in the introduction, we employ a deterministic compartmental modeling framework. The methodology consists of formulating a system of nonlinear ordinary differential equations describing disease transmission dynamics, followed by a rigorous local stability analysis.

2.1 Model Formulation

In this part, we develop a compartmental epidemic model that incorporates vaccination. To construct the model equations, the total population N is categorized into six distinct groups: the susceptible individuals $S(t)$, the exposed individuals $E(t)$, the infective individuals $I_1(t)$, those receiving treatment $T(t)$, the infected under drug resistant $I_2(t)$, and the recovered individuals $R(t)$. The construction of the model is based on the following assumptions:

- i. Natural births occur in the population, whereby newborns enter the susceptible class and may or may not possess temporary immunity at birth.
- ii. Natural mortality takes place in every compartment, affecting all individuals regardless of their epidemiological status.
- iii. Susceptible individuals may acquire infection through effective contact with infectious individuals who are untreated, undergoing treatment, or have failed to recover after treatment. Newly infected individuals enter the exposed class, where they are infected but do not yet show symptoms.
- iv. Exposed individuals, who have contracted the pathogen but remain asymptomatic, progress to the first infectious class once the latent stage ends.
- v. Individuals in the first infectious class may enter treatment, thereby moving to the treatment compartment.
- vi. Individuals undergoing treatment may experience one of two outcomes: they may recover from the disease or fail to recover, in which case they progress to the second infectious class representing more severe infection.
- vii. Individuals who fail to recover after treatment may eventually recover from the disease, entering the recovered class.
- viii. Disease-induced mortality may occur in all infectious classes, including untreated infectious individuals, individuals under treatment, and individuals who failed to recover after treatment.
- ix. Recovered individuals may lose their immunity over time, returning to the susceptible class and becoming vulnerable to reinfection.

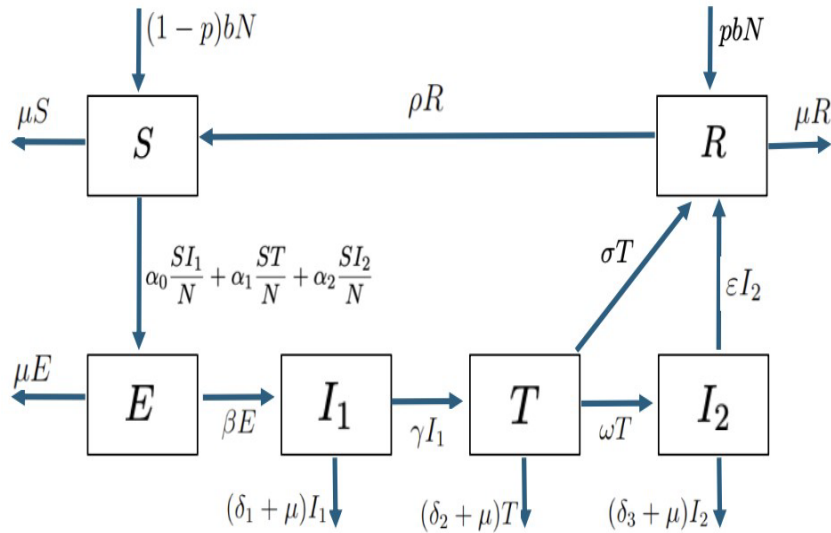
All parameters associated with the construction of the proposed model, which describes the transition of individuals between compartments, are presented in Table 1. With reference to the factors outlined previously, Figure 1 shows the compartmental flow diagram that underlies the formulation of the epidemic model.

Table 1 (a): Parameters Involved in the Formation of Epidemic Model

Notation	Parameter Description
b	Natural birth rate
α_0	Transmission rate from S to E due to contact with individuals in I_1

Table 1 (b): Parameters Involved in the Formation of Epidemic Model

Notation	Parameter Description
α_1	Transmission rate from S to E due to contact with individuals in T
α_2	Transmission rate from S to E due to contact with individuals in I_2
μ	Natural mortality rate
δ_1	Disease-induced mortality rate in I_1
δ_2	Disease-induced mortality rate in the T
δ_3	Disease-induced mortality rate in I_2
β	Transition rate from E to I_1
ω	Rate of treatment failure (drug resistance) in T
ε	Treatment rate in I_2
σ	Treatment rate in the T
ρ	Rate at which recovered individuals lose immunity and return to the S
p	Fraction of newborns vaccinated at birth
γ	Transition rate from I_1 to T (treatment initiation rate)


Figure 1: Compartmental Diagram of Epidemic Model

Referring to the flow chart, the dynamics of the model are represented through the following set of nonlinear ordinary differential equations:

$$\begin{aligned}
 \frac{dS}{dt} &= (1-p)bN - \alpha_0 \frac{SI_1}{N} - \alpha_1 \frac{ST}{N} - \alpha_2 \frac{SI_2}{N} + \rho R - \mu S \\
 \frac{dE}{dt} &= \alpha_0 \frac{SI_1}{N} + \alpha_1 \frac{ST}{N} + \alpha_2 \frac{SI_2}{N} - (\beta + \mu)E \\
 \frac{dI_1}{dt} &= \beta E - (\gamma + \delta_1 + \mu)I_1 \\
 \frac{dT}{dt} &= \gamma I_1 - (\sigma + \delta_2 + \mu + \omega)T \\
 \frac{dI_2}{dt} &= \omega T - (\varepsilon + \delta_3 + \mu)I_2 \\
 \frac{dR}{dt} &= pbN + \sigma T + \varepsilon I_2 - (\mu + \rho)R
 \end{aligned} \tag{1}$$

where

$$N(t) = S(t) + E(t) + I_1(t) + T(t) + I_2(t) + R(t), \tag{2}$$

with all of the initial conditions $S(0) = S_0$, $E(0) = E_0$, $I_1(0) = I_{01}$, $T(0) = T_0$, $I_2(0) = I_{02}$, $R(0) = R_0$ are nonnegative.

To ensure the validity of the model, it must be demonstrated that the solution of model (1) remains nonnegative and bounded, thereby confirming its epidemiological and mathematical relevance. First, let us show that the solution of the

model (1) is nonnegative. We state the following result.

Theorem 2.1. For any nonnegative initial conditions $S_0, E_0, I_{01}, H_0, I_{02}, R_0$ The solution to model (1) is nonnegative.

Proof. Consider the equation for the susceptible compartment in system (1), as presented by

$$\frac{dS}{dt} = (1-p)bN + \rho R - \left(\alpha_0 \frac{I_1}{N} + \alpha_1 \frac{T}{N} + \alpha_2 \frac{I_2}{N} + \mu \right) S \geq -CS, \quad (3)$$

Where $C = \alpha_0 \frac{I_1}{N} + \alpha_1 \frac{T}{N} + \alpha_2 \frac{I_2}{N} + \mu$. Multiplying (3) by integral factor $\phi = \exp \left(\int_0^t C(z) dz \right)$, we obtain

$$\begin{aligned} \phi \frac{dS}{dt} + \phi CS &\geq 0. \\ \frac{d}{dt}(\phi S) &\geq 0. \end{aligned} \quad (4)$$

Inequality (4) show that ϕS is increase, and it implies

$$\begin{aligned} \phi S &= \exp \left(\int_0^t C(z) dz \right) S(t) \geq \phi(0)S(0) = S(0) \\ S &\geq S(0) \exp \left(- \int_0^t C(z) dz \right) \geq 0, \end{aligned}$$

Due to nonnegativity of the natural exponential function. Furthermore, consider the equation for the exposed compartment in system (1), as presented by

$$\frac{dE}{dt} = \alpha_0 \frac{SI_1}{N} + \alpha_1 \frac{ST}{N} + \alpha_2 \frac{SI_2}{N} - (\beta + \mu)E \geq -(\beta + \mu)E. \quad (5)$$

From (5) it is obtained

$$E \geq E_0 \exp(-(\beta + \mu)t) \geq 0$$

Due to nonnegativity of E_0 and the natural exponential function. By the same procedure, the other state variables I_1, T, I_2, R are nonnegative for all time $t \geq 0$.

We now proceed to demonstrate that the solution of model (1) remains bounded. Differentiating both sides of equation (2) with respect to time t and applying equation (1) followed by algebraic simplification, we obtain the expression for the rate of change of the total population, given by:

$$\frac{dN}{dt} = (b - \mu)N - (\delta_1 I_1 + \delta_2 T + \delta_3 I_2) \leq (b - \mu)N. \quad (6)$$

Inequality (6) results

$$N(t) \leq N_0 \exp((b - \mu)t). \quad (7)$$

The equation (7) demonstrates that $N(t) \leq N_0$ for all time $t > 0$ if $b \leq \mu$, and this means the solution of the model (1) is bounded.

For the sake of tractability, we proceed to derive the dimensionless form of model (1). Setting $s = \frac{S}{N}, \tilde{e} = \frac{E}{N}, \tilde{i}_1 = \frac{I_1}{N}, \tilde{t} = \frac{T}{N}, \tilde{i}_2 = \frac{I_2}{N}, \tilde{r} = \frac{R}{N}$ as the fractions of the class susceptible, exposed, infected, those receiving treatment, resistance and reformed in the population, respectively, we get

$$\begin{aligned} \frac{d\tilde{s}}{dt} &= (1-p)b - \alpha_0 \tilde{s} \tilde{i}_1 - \alpha_1 \tilde{s} \tilde{t} - \alpha_2 \tilde{s} \tilde{i}_2 + \rho \tilde{r} - \mu \tilde{s} \\ \frac{d\tilde{e}}{dt} &= \alpha_0 \tilde{s} \tilde{i}_1 + \alpha_1 \tilde{s} \tilde{t} + \alpha_2 \tilde{s} \tilde{i}_2 - (\beta + \mu) \tilde{e} \\ \frac{d\tilde{i}_1}{dt} &= \beta \tilde{e} - (\gamma + \delta_1 + \mu) \tilde{i}_1 \\ \frac{d\tilde{t}}{dt} &= \gamma \tilde{i}_1 - (\sigma + \delta_2 + \mu + \omega) \tilde{t} \\ \frac{d\tilde{i}_2}{dt} &= \omega \tilde{t} - (\epsilon + \delta_3 + \mu) \tilde{i}_2 \\ \frac{d\tilde{r}}{dt} &= p b + \sigma \tilde{t} + \epsilon \tilde{i}_2 - (\mu + \rho) \tilde{r} \end{aligned} \quad (8)$$

with all of the initial conditions $\tilde{s}(0), \tilde{e}(0), \tilde{i}_1(0), \tilde{t}(0), \tilde{i}_2(0), \tilde{r}(0)$ are nonnegative.

2.2 Local Stability Analysis

As established in dynamical systems theory (Lynch, 2014), analyzing the local stability of model (8) involves examining the long-term behavior of its solutions near the fixed points. To determine the fixed points, we set all time derivatives in the system to zero and solve the resulting steady-state equations, i.e.

$$\frac{d\tilde{s}}{dt} = \frac{d\tilde{e}}{dt} = \frac{d\tilde{i}_1}{dt} = \frac{d\tilde{t}}{dt} = \frac{d\tilde{i}_2}{dt} = \frac{d\tilde{r}}{dt} = 0. \quad (9)$$

In the context of the epidemiological model, two fixed points are typically considered: the disease-free fixed point, denoted by

$$\mathcal{E}^0 = (\tilde{s}^0, \tilde{e}^0, \tilde{i}_1^0, \tilde{t}^0, \tilde{i}_2^0, \tilde{r}^0),$$

and the disease-endemic fixed point, denoted by

$$\mathcal{E}^* = (\tilde{s}^*, \tilde{e}^*, \tilde{i}_1^*, \tilde{t}^*, \tilde{i}_2^*, \tilde{r}^*).$$

The disease-free fixed point represents a state in which the entire population is free from disease, which is mathematically characterized by setting $\tilde{i}_1 = 0$, $\tilde{t} = 0$ and $\tilde{i}_2 = 0$. Applying these to (8), we have the disease-free fixed point

$$\mathcal{E}^0 = \left(\frac{b((1-p)\mu + \rho)}{\mu(\rho + \mu)}, 0, 0, 0, 0, \frac{pb}{\mu + \rho} \right). \quad (10)$$

The basic reproduction number, denoted by $\mathcal{R}_0$, is defined as the expected number of secondary infections generated by a single infectious individual in a completely susceptible population. The basic reproduction number of system (8) is computed by using the Next Generation Matrix method as follows:

$$\mathcal{R}_0 = \hat{\rho}(FV^{-1}),$$

where F is the Jacobian matrix corresponding to the new infection terms, V is the Jacobian matrix associated with transition terms among the infected compartments, and $\hat{\rho}(\cdot)$ denotes the spectral radius of a matrix (i.e., the dominant eigenvalue) (Brauer et al., 2008). For the model (8), we have

$$F = \begin{bmatrix} 0 & \alpha_0 \tilde{s}^0 & \alpha_1 \tilde{s}^0 & \alpha_2 \tilde{s}^0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} f & 0 & 0 & 0 \\ -\beta & g & 0 & 0 \\ 0 & -\gamma & h & 0 \\ 0 & 0 & -\omega & i \end{bmatrix},$$

where

$$\begin{aligned} f &= \beta + \mu, \\ g &= \gamma + \delta_1 + \mu, \\ h &= \sigma + \delta_2 + \mu + \omega, \\ i &= \epsilon + \delta_3 + \mu. \end{aligned} \quad (11)$$

Likewise, we have

$$\mathcal{R}_0 = \frac{(\alpha_0 h i + \alpha_1 \gamma i + \alpha_2 \gamma \omega) \tilde{s}^0 \beta}{f g h i}. \quad (12)$$

Where $\tilde{s}^0 = \frac{b((1-p)\mu + \rho)}{\mu(\rho + \mu)}$.

Differentiating $\mathcal{R}_0$ with respect to the vaccination fraction p yields

$$\frac{\partial \mathcal{R}_0}{\partial p} = - \frac{(\alpha_0 h i + \alpha_1 \gamma i + \alpha_2 \gamma \omega) b \beta}{f g h i (\rho + \mu)} < 0.$$

Hence, $\mathcal{R}_0$ is a strictly decreasing function of p . Therefore, increasing neonatal vaccination coverage always reduces the basic reproduction number and suppresses the potential for disease transmission.

By applying the Hartman's Theorem (Lynch, 2014), the disease-free fixed points $\mathcal{E}^0$ is asymptotically stable if the real part of all eigenvalues of the Jacobian matrix at $\mathcal{E}^0$ (denoted by $J_{\mathcal{E}^0}$) is negative provided $\mathcal{E}^0$ is a hyperbolic fixed point. A simple calculation yields.

$$\mathcal{J}_{\mathcal{E}^0} = \begin{bmatrix} -\mu & 0 & -\alpha_0 \tilde{s}^0 & -\alpha_1 \tilde{s}^0 & -\alpha_2 \tilde{s}^0 & \rho \\ 0 & -f & \alpha_0 \tilde{s}^0 & \alpha_1 \tilde{s}^0 & \alpha_2 \tilde{s}^0 & 0 \\ 0 & \beta & -g & 0 & 0 & 0 \\ 0 & 0 & \gamma & -h & 0 & 0 \\ 0 & 0 & 0 & \omega & -i & 0 \\ 0 & 0 & 0 & \sigma & \epsilon & -j \end{bmatrix}, \quad (13)$$

where $j = \mu + \rho$. The characteristic polynomial of $\mathcal{J}_{\mathcal{E}^0}$ is given by the following equation:

$$p(\lambda) = (-\mu - \lambda)(-j - \lambda)(a_4 + a_3\lambda + a_2\lambda^2 + a_1\lambda^3 + \lambda^4). \quad (14)$$

where

$$\begin{aligned} a_1 &= f + g + h + i \\ a_2 &= fg + fh + fi + gh + gi + hi - \alpha_0 \beta \tilde{s}^0, \\ a_3 &= fgh + fgi + fhi + ghi - \alpha_0 \beta \tilde{s}^0 h - \alpha_0 \beta \tilde{s}^0 i - \alpha_1 \beta \gamma \tilde{s}^0 \\ a_4 &= fghi - \alpha_0 \beta hi \tilde{s}^0 - \alpha_1 \beta \gamma i \tilde{s}^0 - \alpha_2 \beta \gamma \omega \tilde{s}^0. \end{aligned} \quad (15)$$

The eigenvalues of the Jacobian matrix $\mathcal{J}_{\mathcal{E}^0}$ are the roots of $p(\lambda)$, namely, $\lambda_1 = -\mu$, $\lambda_2 = -j$, and $\lambda_3, \lambda_4, \lambda_5, \lambda_6$ are the roots of polynomial

$$p_1(\lambda) = a_4 + a_3\lambda + a_2\lambda^2 + a_1\lambda^3 + \lambda^4. \quad (16)$$

It is obvious that λ_1 and λ_2 are all negative. Using Routh–Hurwitz criterion (Fisher, 1999), $\text{Re}(\lambda_k) < 0$, $k = 3, 4, 5, 6$ if and only if

$$\begin{aligned} (c1) a_1 &> 0, \\ (c2) a_1 a_2 - a_3 &> 0, \\ (c3) a_3(a_1 a_2 - a_3) - a_1^2 a_4 &> 0, \\ (c4) a_4(a_3(a_1 a_2 - a_3) - a_1^2 a_4) &> 0. \end{aligned} \quad (17)$$

Condition (c1) is obvious from the first equation of (15). Conditions (c2) and (c3) imply that $a_4 > 0$ in condition (c4). Moreover, one can observe that

$$\begin{aligned} a_4 > 0 &\Leftrightarrow fghi - \alpha_0 \beta \tilde{s}^0 hi - \alpha_1 \beta \gamma \tilde{s}^0 i - \alpha_2 \beta \gamma \omega \tilde{s}^0 > 0 \\ &\Leftrightarrow 1 - \frac{(\alpha_0 hi + \alpha_1 \gamma i + \alpha_2 \gamma \omega) \tilde{s}^0 \beta}{fghi} > 0 \\ &\Leftrightarrow 1 - \mathcal{R}_0 > 0. \end{aligned}$$

The preceding explanation implies the following result.

Theorem 2.2. The disease-free fixed point $\mathcal{E}_0$ is locally asymptotically stable if and only if the basic reproduction number $\mathcal{R}_0$ is less than one.

Now, let us proceed to the analysis of the disease-endemic fixed point. The disease-endemic fixed point corresponds to a state where the disease continues to exist in the population, which is mathematically characterized by $\tilde{t}_1 > 0$. Using this and (9), we have

$$\begin{aligned} \tilde{s}^* &= \frac{(1-p)b j h i + \rho \sigma \gamma i \tilde{t}_1^* + \rho p b h i + \rho \epsilon \omega \gamma \tilde{t}_1^*}{\alpha j h i \tilde{t}_1^* + \alpha_1 \gamma j i \tilde{t}_1^* + \alpha_2 \omega \gamma j \tilde{t}_1^* + \mu j h i} \\ \tilde{e}^* &= \frac{g}{\beta} \tilde{t}_1^* \\ \tilde{t}_1^* &= \frac{\phi_1}{\phi_2} (R_0 - 1) \\ \tilde{t}^* &= \frac{\gamma \tilde{t}_1^*}{h} \\ \tilde{t}_2^* &= \frac{h}{\omega \gamma} \tilde{t}_1^* \\ \tilde{r}^* &= \frac{p b}{j} + \frac{(\sigma r i + \epsilon \omega \gamma)}{h i j} \end{aligned} \quad (18)$$

where

$$\phi_1 = \mu f g h^2 i^2 j$$

$$\phi_2 = k[\mu g h i j + \mu \beta g h i + (\beta \rho(\sigma + \omega)(\delta_1 + \mu) + \beta \rho g(\delta_2 + \mu))i + \beta \rho \omega \gamma(\delta_3 + \mu)]$$

with $k = \alpha_0 h i + \alpha_1 \gamma i + \alpha_2 \gamma \omega$. From (18), we have that $\tilde{i}_1 > 0$ if and only if $R_0 > 1$. Using Hartman's Theorem again, the disease-endemic fixed point is asymptotically stable if the real part of all eigenvalues of the Jacobian matrix at ε^* (denoted by J_{ε^*}) is negative. The Jacobian matrix J_{ε^*} of system (8) is given by

$$J_{\varepsilon^*} = \begin{bmatrix} -(\alpha_0 \tilde{i}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{i}_2^* + \mu) & 0 & -\alpha_0 \tilde{s}^* & -\alpha_1 \tilde{s}^* & -\alpha_2 \tilde{s}^* & \rho \\ \alpha_0 \tilde{i}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{i}_2^* & -f & \alpha_0 \tilde{s}^* & \alpha_1 \tilde{s}^* & \alpha_2 \tilde{s}^* & 0 \\ 0 & \beta & -g & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -h & 0 \\ 0 & 0 & 0 & \omega & -i & 0 \\ 0 & 0 & 0 & \sigma & \varepsilon & -j \end{bmatrix} \quad (19)$$

Eigenvalues of the Jacobian matrix J_{ε^*} are the roots of its characteristic polynomial, namely

$$g(\lambda) = k_6 + k_5 \lambda + k_4 \lambda^2 + k_3 \lambda^3 + k_2 \lambda^4 + k_1 \lambda^5 + \lambda^6 \quad (20)$$

where k_j , for $j = 1, 2, \dots, 6$, are given in Appendix. Using the Routh–Hurwitz criterion (Fisher, 1999), the real part of $\lambda_k < 0$, $k = 1, 2, \dots, 6$, if and only if

$$\begin{aligned} (C1) \quad & k_1 > 0, \\ (C2) \quad & k_1 k_2 - k_3 > 0, \\ (C3) \quad & k_1(k_2 k_3 - k_1 k_4) - (k_3^2 - k_1 k_5) > 0, \\ (C4) \quad & k_1 \psi_2 - \psi_4 > 0, \\ (C5) \quad & k_1(-k_6 \psi_1 + k_5 \psi_2) + k_6 \psi_3 - k_5 \psi_4 > 0, \\ (C6) \quad & k_6[k_1(-k_6 \psi_1 + k_5 \psi_2) + k_6 \psi_3 - k_5 \psi_4] > 0, \end{aligned} \quad (21)$$

Where

$$\begin{aligned} \psi_1 &= k_2(k_3^2 - k_1 k_5) - k_1(k_3 k_4 - k_1 k_6) \\ \psi_2 &= k_2(k_3 k_4 - k_1 k_5) - k_1(k_4^2 - k_1 k_6) + (k_4 k_5 - k_3 k_6) \\ \psi_3 &= k_3(k_3^2 - k_1 k_5) - k_1 k_3 k_5 \\ \psi_4 &= k_3(k_3 k_4 - k_2 k_5) - k_5(k_1 k_4 - k_5) \end{aligned}$$

It follows from the Appendix that $k_1 > 0$ is equivalent to $\mathcal{R}_0 > 1$, thereby validating part (C1) of (21). The explicit formulation of conditions (C2) – (C6) as functions of the basic reproduction number $\mathcal{R}_0$, such that they yield the relation $\mathcal{R}_0 > 1$, results in exceedingly intricate expressions. Due to this analytical complexity, numerical validation was performed using Python. The simulations demonstrate that these relations are consistently positive, thereby ensuring that the stability requirements are rigorously satisfied. Thus, the following result is established.

Theorem 2.3. If $\mathcal{R}_0 > 1$ and conditions (C2) – (C6) in (21) are satisfied, then disease-endemic fixed point ε^* is locally asymptotically stable.

3. Numerical Simulation and Discussion

To further elucidate the theoretical findings presented in Theorems 2.2 and 2.3, numerical simulations are carried out using a fourth-order Runge–Kutta method implemented in Python. All initial conditions and the parameter values are chosen for illustrative purposes to demonstrate qualitative system behavior rather than to fit a specific epidemiological dataset. Specifically, the initial conditions are taken as $\tilde{s}(0) = 0.98$, $\tilde{e}(0) = 0.01$, $\tilde{i}_1(0) = 0.005$, $\tilde{t}(0) = 0.003$, $\tilde{i}_2(0) = 0.001$, $\tilde{r}(0) = 0.001$ and the corresponding parameter values are summarised in Table 2.

Table 2: The Parameter Values of the Model

Parameter	α_0	α_1	α_2	δ_1	δ_2	δ_3	β	ω	ε	σ	ρ	γ
Value	0.1	0.01	0.005	0.35	0.3	0.01	0.1	0.005	0.001	0.2	0.8	0.2

3.1 Stability Analysis of the Disease-Free Fixed Point

By employing the parameter values presented in Table 2. and setting parameter $b = \mu = 0.15$ and the neonatal vaccination fraction to $p = 0.5$, the basic reproduction number is computed as $\mathcal{R}_0 = 0.892$. Accordingly, the disease-free equilibrium point is $\varepsilon^0 = (0.9211, 0, 0, 0, 0, 0.0789)$.

Figure 2 illustrates the temporal evolution of each subpopulation. It can be observed that the trajectories

corresponding to the infectious individuals (I_1), those undergoing treatment (T), and individuals experiencing drug resistance (I_2) rapidly converge to zero.

Consistent with Theorem 2.2, the condition $\mathcal{R}_0 < 1$ ensures eventual disease eradication in the long-term dynamics of the system. Epidemiologically, this suggests that the implemented interventions, particularly neonatal vaccination at a 50% coverage rate, are capable of suppressing disease transmission. Nevertheless, in practical settings, total elimination may remain challenging due to additional complexities inherent in real population dynamics.

3.2 Stability Analysis of the Disease Endemic Fixed Point and the Impact of Vaccination

To represent the endemic setting, the birth rate $b = 0.05$ and natural death rate $\mu = 0.05$, respectively. The simulations are conducted under different values of the neonatal vaccination fraction (p) to evaluate its effect on disease prevalence.

(i). Scenario $p = 0.3$: The resulting basic reproduction number is $\mathcal{R}_0 = 1.4049$, and the corresponding fixed point is $\varepsilon^* = (0.6768, 0.0204, 0.0246, 0.0089, 0.0009, 0.0177)$.

(ii). Scenario $p = 0.5$: The resulting basic reproduction number is $\mathcal{R}_0 = 1.3881$, and the corresponding fixed point is $\varepsilon^* = (0.6774, 0.01959, 0.0236, 0.0085, 0.0009, 0.0294)$.

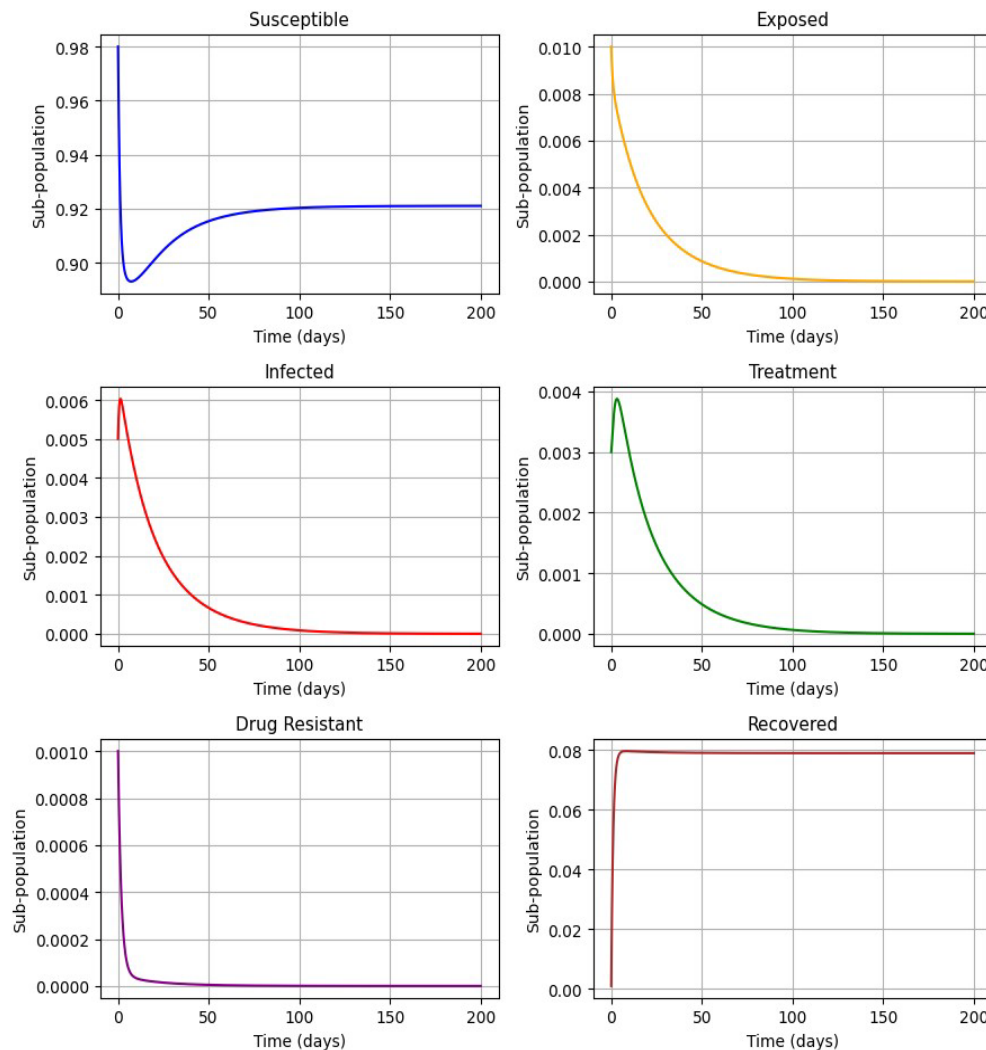


Figure 2: Curves of Each Subpopulation for $\mathcal{R}_0 < 1$

(iii). Scenario $p = 0.8$: The resulting basic reproduction number is $\mathcal{R}_0 = 1.13628$, and the corresponding fixed point is $\varepsilon^* = (0.6784, 0.0183, 0.0221, 0.0080, 0.00088, 0.0471)$.

The simulation results in Figure 3 demonstrate that higher levels of neonatal vaccination (p) lead to a consistent decline in the sizes of all infected compartments (I_1, T, I_2). While the disease remains endemic because $\mathcal{R}_0 > 1$, the total

infection burden is substantially reduced.

A key finding is that higher neonatal vaccination coverage suppresses the size of the drug-resistant compartment (I_2), despite the continued persistence of transmission. This indicates that early vaccination strategies not only reduce the initial infection burden but also indirectly mitigate the risk of treatment-induced drug resistance at the population level.

Numerically, it is found that the Routh–Hurwitz conditions (C2) – (C6) are satisfied in all considered scenarios, thereby confirming the local asymptotically stability of the endemic fixed point. This implies that small perturbations in the number of infections will cause the system to return to the endemic level rather than escalate into an uncontrolled outbreak.

From a public health standpoint, these simulation findings reinforce the effectiveness of neonatal.

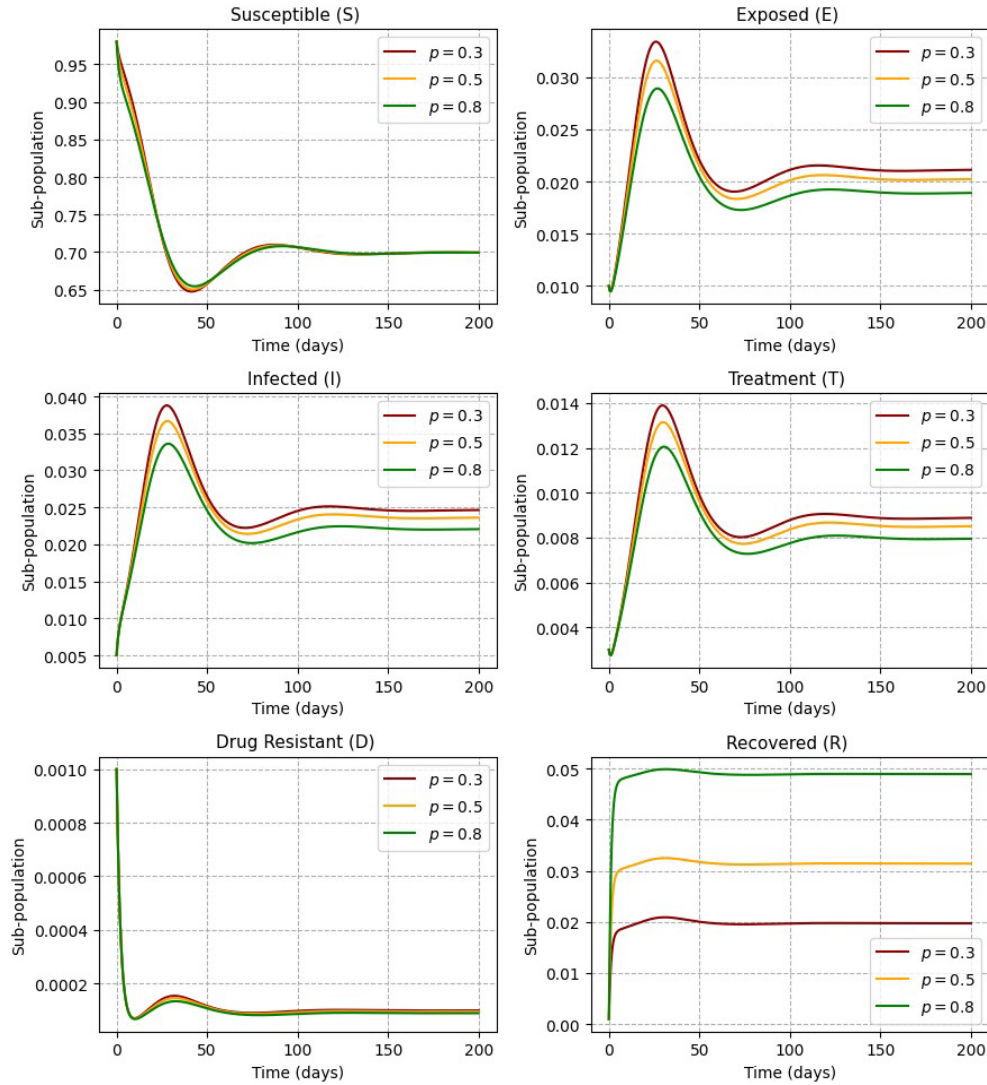


Figure 3: Curves of Each Subpopulation for $\mathcal{R}_0 > 1$ of Several Values p

Vaccination as a control strategy. Enhanced vaccination coverage at birth can substantially mitigate epidemic intensity, stabilize disease dynamics more rapidly, and indirectly limit the development of drug resistance by reducing the total infectious reservoir within the population.

4. Conclusion

In this work, we formulated a deterministic epidemic model with three infected compartments to capture multistage infection progression and heterogeneous infectiousness, thereby providing a more realistic representation of disease transmission dynamics. The model structure allowed us to analytically derive the basic reproduction number, R_0 , using the next-generation matrix and to rigorously establish the existence and local stability of both the disease-free and endemic fixed points. Our results show that the disease-free fixed point is locally asymptotically stable whenever $\mathcal{R}_0 < 1$. In contrast, a positive endemic fixed point exists and is locally asymptotically stable when $\mathcal{R}_0 > 1$. A key component of the model is

neonatal vaccination, incorporated through the fraction of newborns vaccinated at birth. The analysis demonstrates that increasing this vaccination fraction decreases the inflow of susceptible newborns into the infected classes, leading to a nonlinear reduction in $\mathcal{R}_0$ and substantial suppression of infection prevalence across all infected stages. Numerical simulations further confirm these analytical findings, illustrating how neonatal vaccination attenuates epidemic peaks, accelerates convergence to fixed point, and shortens transient dynamics even when $\mathcal{R}_0 > 1$.

Taken together, these results show that neonatal vaccination is a biologically effective and operationally feasible intervention capable of reducing multistage infection burden and mitigating long-term endemic burden.

While the present analysis establishes rigorous local stability results, deriving global stability conditions for the disease-free and endemic equilibria constitutes an important direction for future research. Such an investigation would require the construction of suitable Lyapunov functions or alternative analytical techniques and is left for subsequent study.

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7. Appendix

$$\begin{aligned}
k_1 &= \alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu + f + g + h + i + j \\
&= (R_0 - 1) \left(\alpha_0 + \alpha_1 \frac{\gamma}{h} + \frac{\omega \gamma}{hi} \right) \frac{\phi_1}{\phi_2} + \mu + f + g + h + i + j \\
k_2 &= (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu)(f + g + h + i + j) + f(g + h + i + j) + g(h + i + j) \\
&\quad + hi + hj + ij - \alpha_0 \beta \tilde{s}^* \frac{\omega \gamma}{hi} \tilde{l}_1^* \\
k_3 &= (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu)(f g + f h + f i + f j + g h + g i + g j + h i + h j + i j) \\
&\quad + f(g h + g i + g j + h i + h j + i j) + g(h i + h j + i j) + h i j \\
&\quad - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu + h + i + j) \alpha_0 \beta \tilde{s}^* - \alpha_1 \beta \gamma \tilde{s}^* \\
&\quad + (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) \alpha_0 \beta \tilde{s}^* \\
k_4 &= (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu)(f g h + f g i + f g j + f h i + f h j + f i j + g h i \\
&\quad + g h j + g i j + h i j) + f g h i + f g h j + f g i j + f h i j + g h i j \\
&\quad - ((\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu)(h + i + j) + h i + h j + i j) \alpha_0 \beta \tilde{s}^* \\
&\quad - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu + i + j) \alpha_1 \beta \gamma \tilde{s}^* - \alpha_2 \beta \gamma \omega \tilde{s}^* \\
&\quad + (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*)(h + i + j) \alpha_0 \beta \gamma \tilde{s}^* + \alpha_1 \beta \gamma \tilde{s}^* \\
k_5 &= (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu)(f g h i + f g h j + f g i j + f h i j + g h i j) + f g h i j \\
&\quad - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu)(h i + h j + i j) + h i j) \alpha_0 \beta \tilde{s}^* \\
&\quad - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu)(i + j) + i j) \alpha_1 \beta \gamma \tilde{s}^* \\
&\quad - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu + j) j \alpha_2 \beta \gamma \omega \tilde{s}^* \\
&\quad + (h i + h j + i j)(\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) \alpha_0 \beta \tilde{s}^* - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) \beta \gamma \rho \sigma \\
&\quad + (i + j)(\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) \alpha_1 \beta \gamma \tilde{s}^* + (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) \alpha_2 \beta \gamma \omega \tilde{s}^* \\
k_6 &= (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu) f g h i j - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu) h i j \alpha_0 \beta \tilde{s}^* \\
&\quad - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu) i j \alpha_1 \beta \gamma \tilde{s}^* - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^* + \mu) j \alpha_2 \beta \gamma \omega \tilde{s}^* \\
&\quad + (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) h i j \alpha_0 \beta \tilde{s}^* - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) \beta \rho \gamma \omega \varepsilon \\
&\quad - (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) \beta \rho \gamma \sigma i + (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) i j \alpha_1 \beta \gamma \tilde{s}^* \\
&\quad + (\alpha_0 \tilde{l}_1^* + \alpha_1 \tilde{t}^* + \alpha_2 \tilde{l}_2^*) j \alpha_2 \beta \gamma \omega \tilde{s}^*
\end{aligned}$$