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Dynamical Analysis of Nipah Virus Transmission Model

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Abstract

Nipah virus can be transmitted from animals to humans through contaminated food or direct human-to-human contact. In this paper, we formulate and analyze an $SEIRD$ model to describe the transmission dynamics of the Nipah virus by incorporating two significant factors: unprotected contact with the corpses of Nipah virus–infected individuals prior to burial and the incubation period during transmission. The $SEIRD$model partitions the total human population into five compartments: susceptible (S), exposed (E), infectious (I), recovered (R), and deceased (D). Two equilibrium points are identified in the system, namely the disease-free equilibrium (DFE) and the endemic equilibrium (EE). The basic reproduction number $\mathcal{R}_0$, is derived using the next-generation matrix method. Stability analysis reveals that the DFE is locally asymptotically stable when $\mathcal{R}_0 < 1$, indicating disease eradication, whereas the EE is locally asymptotically stable when $\mathcal{R}_0 > 1$, signifying the persistence of the disease within the population. Furthermore, numerical simulations are carried out to illustrate the analytical findings and to investigate the influence of key epidemiological parameters on disease transmission. The results provide new insights into the role of unprotected contact with corpses and incubation delays in Nipah virus dynamics, offering valuable guidance for the development of more effective prevention and control strategies.

Keywords: Nipah virus, equilibrium point, basic reproduction number, stability analysis, numerical simulation.

2010 Mathematics Subject Classification: 92D30, 34D20, 37N25, 65L05.

1. Introduction

Nipah disease is caused by the Nipah virus (NiV), a member of the Henipavirus genus within the Paramyxoviridae family. The virus was first identified in 1998 during an outbreak on a pig farm in Sungai Nipah, Negeri Sembilan, Malaysia, from which its name originates. Since then, Nipah virus outbreaks have

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been reported in Singapore, Bangladesh, India, and the Philippines [8]. Nipah disease is a zoonotic infection, capable of transmission between animals and humans. The spread of the virus is closely associated with ecological disturbances; for instance, stressed fruit bats—the natural reservoir hosts—may shed the virus when their habitats are disrupted by extensive deforestation [21]. Human infection can occur through direct contact with infected animals, consumption of contaminated food products, or close contact with infected individuals. Moreover, human-to-human transmission may occur via exposure to saliva, blood, or other bodily fluids of infected persons [10].

Furthermore, according to Zaki [28], the clinical manifestations of Nipah virus infection include encephalitis and respiratory illness. The symptoms of Nipah virus infection in humans vary depending on the severity of the disease. In mild cases, infection may be asymptomatic or present with nonspecific symptoms such as cough, fever, headache, myalgia, nausea, vomiting, and shortness of breath. Generally, symptoms appear between 4 and 14 days after exposure, with an incubation period that can extend up to 45 days. Severe cases often progress to acute encephalitis and seizures, which can lead to death within 24–48 hours if not treated promptly. Such severe infections may also result in permanent neurological damage or fatality. The average case fatality rate is estimated to range between 40% and 75%, depending on the capacity of local health authorities to perform effective epidemiological investigations, surveillance, and clinical management of Nipah virus cases [26]. Outbreaks of Nipah virus have been reported in several countries, including Malaysia [8], [11], Singapore [20], Bangladesh [15], [13], [18], [22], India [6], [2], [1], [12], [19], [24], and the Philippines [7]. Given the high mortality rate and recurrent outbreaks in different regions, further studies on the transmission dynamics of the Nipah virus are essential to support effective prevention and control strategies.

Mathematical models can be employed to predict the transmission dynamics of Nipah virus infection, assess the effectiveness of intervention strategies, and design more efficient prevention and control measures. Several mathematical models have been developed to study the spread of Nipah virus disease. For instance, Zewdie and Gakkar [29] proposed an $SIRD$ model, where the compartment D represents infected deceased individuals who have not yet been buried. This model was motivated by evidence that the Nipah virus can be transmitted through direct contact between susceptible individuals and the corpses of infected persons without protective equipment. Their findings demonstrated that preventing unsafe contact with infected bodies and ensuring immediate burial or cremation can significantly reduce the transmission rate of the Nipah virus. Further refinement was made by Raza et al. [23], who formulated an $SEIR$ model incorporating quarantine control for exposed individuals, isolation capacity, public awareness of healthy lifestyles, surveillance, and treatment of infected individuals. Their study revealed that these control measures can effectively reduce the spread of Nipah disease.

In this study, we extend the models of [29] and [23] by integrating both key transmission factors: direct contact with infected corpses [29] and the incubation period in exposed individuals [23]. To capture these dynamics, we introduce an additional compartment E (Exposed) and formulate an $SEIRD$ model for the spread of Nipah virus. The proposed model is expressed as an autonomous system of nonlinear differential equations. We determine the equilibrium points and analyze their stability, derive the basic reproduction number ($\mathcal{R}_0$) using the next-generation matrix approach, and perform numerical simulations to examine the effects of key parameters on the system's dynamic behavior and to validate the analytical results.

2. Preliminaries

Definition 2.1. An *equilibrium point* of a differential equation is a point at which the system remains constant over time. Mathematically, for an autonomous system of differential equations

$$\frac{d\vec{x}}{dt} = \vec{f}(\vec{x}),$$

an equilibrium point $\vec{x}^*$ satisfies

$$\vec{f}(\vec{x}^*) = 0.$$

This implies that if the system starts at $\vec{x}^*$, it will remain there indefinitely since the state variables do not change with time. Equilibrium points play a crucial role in understanding the long-term behavior and stability of dynamical systems [3].

Definition 2.2. The *stability of an equilibrium point* describes the behavior of a dynamical system in the neighborhood of that point. Specifically, an equilibrium point $\vec{x}^*$ is said to be stable if, when the system starts sufficiently close to $\vec{x}^*$, it remains close to it for all future times. Conversely, if trajectories diverge from $\vec{x}^*$ after small perturbations, the point is unstable.

Formally, for a continuous dynamical system

$$\frac{d\vec{x}}{dt} = \vec{f}(\vec{x}),$$

an equilibrium point $\vec{x}^*$ is classified as follows:

1. *Stable* if small perturbations in $\vec{x}$ lead to small deviations that remain bounded over time.
2. *Asymptotically stable* if small perturbations cause the system to return to $\vec{x}^*$ as $t \to \infty$.
3. *Unstable* if small perturbations cause the system to move away from $\vec{x}^*$, not returning to equilibrium.

The stability of an equilibrium point can often be determined by linearization or by analyzing the Jacobian matrix of the system evaluated at the equilibrium [3].

Definition 2.3. Consider a dynamical system defined by

$$\frac{d\vec{x}}{dt} = \vec{f}(\vec{x}),$$

where $\vec{x} \in \mathbb{R}^n$ and $\vec{f}(\vec{x})$ denotes the system dynamics. An equilibrium point $\vec{x}^*$ satisfies

$$\vec{f}(\vec{x}^*) = 0.$$

To examine its stability, the system is linearized around $\vec{x}^*$. The Jacobian matrix of the system evaluated at $\vec{x}^*$ is

$$J(\vec{x}^*) = \left[\frac{\partial f_i}{\partial x_j}\right]_{\vec{x}=\vec{x}^*}.$$

The stability of $\vec{x}^*$ depends on the eigenvalues of $J(\vec{x}^*)$ [3]:

1. If all eigenvalues have negative real parts, then $\vec{x}^*$ is *asymptotically stable*.
2. If at least one eigenvalue has a positive real part, then $\vec{x}^*$ is *unstable*.
3. If all eigenvalues have non-positive real parts, and at least one eigenvalue has zero real part, further (nonlinear) analysis is required to determine stability.

3. Main Results

3.1. Model Construction

The $SEIRD$ model proposed in this study is a modification of the models developed by Zewdie and Gakkhar [29] and Raza et al. [23]. The modification accounts for two important epidemiological features of Nipah virus transmission: (i) infection resulting from close contact between susceptible individuals and infected corpses without adequate protection, and (ii) the presence of an incubation period before individuals become infectious. The corresponding compartmental structure is illustrated in Figure 1.

In Figure 1, solid arrows represent transitions between epidemiological compartments due to demographic and disease-related processes, such as infection, progression, recovery, and mortality. Dashed arrows indicate indirect transmission pathways, specifically infection arising from contact between susceptible individuals and contaminated dead bodies of infected individuals.

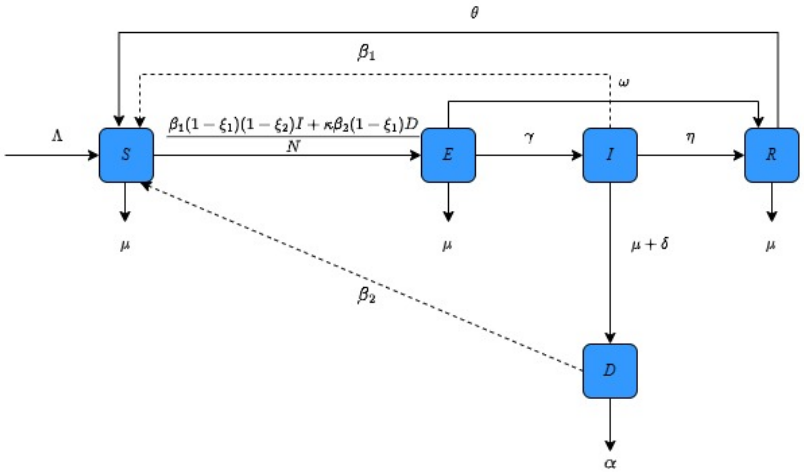

Figure 1: Compartment Diagram of the $SEIRD$ Model for Nipah Virus Transmission

The total population is divided into five mutually exclusive compartments: susceptible $S(t)$, exposed $E(t)$, infected $I(t)$, recovered $R(t)$, and deceased $D(t)$. New individuals enter the susceptible class at a constant recruitment rate Λ. Recovered individuals may lose immunity and return to the susceptible class at rate θ. Susceptible individuals become exposed through effective contact with infectious individuals or infected corpses, occurring at rates β_1 and β_2, respectively. Public health interventions are incorporated through awareness campaigns for susceptible individuals and quarantine measures for infected individuals, with effectiveness levels ξ_1 and ξ_2. Consequently, proportions $(1-\xi_1)$ and $(1-\xi_2)$ represent individuals not successfully protected by these interventions. Natural death occurs in all living compartments at rate μ.

$$\frac{dS}{dt} = \Lambda - \left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N}\right) S - \mu S + \theta R.$$

After infection, individuals enter the exposed class, where they are not yet infectious and experience an incubation period. Exposed individuals progress to the infected class at rate γ, recover through early treatment at rate ω, or die naturally at rate μ.

$$\frac{dE}{dt} = \left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N}\right) S - (\mu + \gamma + \omega)E.$$

Infectious individuals transmit the disease and leave the infected class through natural death, disease-induced death at rate δ, or recovery due to treatment at rate η.

$$\frac{dI}{dt} = \gamma E - (\mu + \delta + \eta)I.$$

Recovered individuals may lose immunity and re-enter the susceptible class or die naturally.

$$\frac{dR}{dt} = \omega E + \eta I - (\mu + \theta)R.$$

The deceased compartment $D(t)$ represents corpses of individuals who died due to infection or natural causes while infected. These corpses contribute to disease transmission until they are safely buried or managed at rate α.

$$\frac{dD}{dt} = (\mu + \delta)I - \alpha D.$$

The transmission dynamics of Nipah virus are governed by the following system of nonlinear autonomous differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N}\right)S - \mu S + \theta R,\\
\frac{dE}{dt} &= \left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N}\right)S - (\mu+\gamma+\omega)E,\\
\frac{dI}{dt} &= \gamma E - (\mu+\delta+\eta)I,\\
\frac{dR}{dt} &= \omega E + \eta I - (\mu+\theta)R,\\
\frac{dD}{dt} &= (\mu+\delta)I - \alpha D,
\end{aligned} \tag{3.1}$$

where $N(t) = S(t) + E(t) + I(t) + R(t)$ denotes the total active population.

The parameters used in the $SEIRD$ model are presented in Table 1.

Parameter	Description
Λ	Recruitment rate to susceptible class
β_1	Rate of infection from infected individuals
β_2	Rate of infection from dead bodies of infected individuals
κ	Fraction of dead bodies that are handled safely
ξ_1	Rate of individuals awareness of infectious diseases
ξ_2	Quarantine rate of infected individuals
γ	Rate of infection in exposed individuals
ω	Rate of recovery in exposed individuals due to awareness
δ	Rate of disease-induced death
η	Rate of recovery in infected individuals due to treatment
θ	Rate of loss of immunity
α	Rate of disposition (burial/cremation) of dead bodies
μ	Natural death rate

Table 1: Description of $SEIRD$ Model Parameters

3.2. Non-negativity and Boundedness of Solution

We prove the non-negativity of solution of model (3.1) to show that the model is epidemiologically meaningful.

Theorem 3.1. *All solutions of model (3.1) subject to non-negative initial values are non-negative and ultimately bounded*

Proof. We first show the non-negativity of S from equation (3.1)

The differential equation for S is given by:

$$\frac{dS}{dt} = \Lambda - \left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N}\right)S - \mu S + \theta R$$

which implies

$$\frac{dS}{dt} \geq -\left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N} + \mu\right)S.$$

Using separation of variables, we get:

$$\int \frac{dS}{S} \geq \int -\left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N} + \mu\right) dt.$$

Integrating both sides:

$$\ln S \geq -\left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N} + \mu\right) t + c.$$

Thus, we obtain:

$$S \geq e^{-\left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I+\kappa\beta_2(1-\xi_1)D}{N}+\mu\right)t+c}.$$

Letting $c_1 = e^c$, we can write:

$$S(t) \geq c_1 e^{-\left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I+\kappa\beta_2(1-\xi_1)D}{N}+\mu\right)t}.$$

Since S is non-negative for all $t > 0$, using the same method, we get $E(t) > 0$, $I(t) > 0$, $R(t) > 0$, and $D(t) > 0$.

To prove the boundedness of the solutions, we need to show that the solutions $S(t)$, $E(t)$, $I(t)$, $R(t)$, and $D(t)$ remain bounded for all $t \geq 0$. The total population $N(t)$ is given by:

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

Differentiating both sides:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \Lambda - \mu N - \delta I.$$

The solution to this linear differential equation is:

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

Since $e^{-\mu t}$ is non-negative for $\forall t \geq 0$, we obtain:

$$\lim_{t\to\infty} N(t) = \frac{\Lambda}{\mu}, \quad \text{so that } 0 \leq N(t) \leq \frac{\Lambda}{\mu}.$$

The compartment $D(t)$ in the $SEIRD$ model represents the cumulative number of individuals who have died due to the disease. This class is not included in the active population, which is defined as

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

Although $D(t)$ is cumulative, it remains bounded for all $t \geq 0$. The dynamics of $D(t)$ are governed by the linear nonhomogeneous differential equation

$$\frac{dD}{dt} = (\mu + \delta)I(t) - \alpha D(t).$$

Using the integrating factor $e^{\alpha t}$, the explicit solution is given by

$$D(t) = D(0)e^{-\alpha t} + \int_0^t (\mu + \delta)I(s)e^{-\alpha(t-s)}\, ds.$$

From the boundedness of the active population, we have

$$I(t) \leq N(t) \leq \frac{\Lambda}{\mu}, \quad \text{for all } t \geq 0.$$

Consequently, the following upper bound holds:

$$(\mu+\delta)I(s) \leq (\mu+\delta)\frac{\Lambda}{\mu}, \quad \text{for all } s \geq 0.$$

Substituting this bound into the integral expression yields

$$D(t) \leq \frac{(\mu+\delta)\Lambda}{\alpha\mu} + \left(D(0) - \frac{(\mu+\delta)\Lambda}{\alpha\mu}\right)e^{-\alpha t},$$

which implies that $D(t)$ is uniformly bounded for all $t \geq 0$.

From this solution, we conclude that $N(t)$ is bounded within the solution region:

$$\Omega = \{(S,E,I,R,D) \,|\, S(t) \geq 0,\ E(t) \geq 0,\ I(t) \geq 0,\ R(t) \geq 0, \\ N(t) = S(t)+E(t)+I(t)+R(t) \leq \frac{\Lambda}{\mu},\ D(t) \leq \frac{(\mu+\delta)\Lambda}{\alpha\mu}\Big\}.$$

□

3.3. Equilibrium point and basic reproduction number ($\mathcal{R}_0$)

The equilibrium point is obtained when $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dD}{dt} = 0$ [3]. The equilibrium points in the Nipah virus spread model are two, namely the disease-free equilibrium point at

$$E_0 = (S,E,I,R,D) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right) \tag{3.2}$$

The disease-free equilibrium point always exists because it satisfies the system of equations under the condition that there is no infection in the population. Second is the endemic equilibrium point at

$$E_1 = (S^*, E^*, I^*, R^*, D^*) \tag{3.3}$$

such that

$$S^* = \frac{\alpha N(\mu+\gamma+\omega)(\mu+\delta+\eta)}{\gamma(\alpha\beta_1(1-\xi_1)(1-\xi_2)+\kappa\beta_2(1-\xi_1)(\mu+\delta))},$$
$$E^* = \frac{\mu+\delta+\eta}{\gamma}I^*,$$
$$I^* = \frac{\alpha\gamma N(\Lambda-\mu S^*)(\mu+\theta)}{\gamma(\mu+\theta)(\alpha\beta_1(1-\xi_1)(1-\xi_2)+\kappa\beta_2(1-\xi_1)(\mu+\delta))S^* - \alpha\theta N(\omega(\mu+\delta+\eta)+\eta\gamma)},$$
$$R^* = \frac{\omega(\mu+\delta+\eta)+\eta\gamma}{\gamma(\mu+\theta)}I^*,$$
$$D^* = \frac{\mu+\delta}{\alpha}I^*.$$

3.3.1. Basic Reproduction Number ($\mathcal{R}_0$)

The basic reproduction number ($\mathcal{R}_0$) is determined by the next generation matrix method [9], [14]. The infectious class of model (3.1) is

$$X = \begin{bmatrix} E \\ I \\ D \end{bmatrix}$$

and the rate of appearance of new infection in each infection class is denote by $\mathcal{F}$ and given by

$$\mathcal{F}(X) = \begin{bmatrix} \frac{(\beta_1(1-\xi_1)(1-\xi_2)I+\kappa\beta_2(1-\xi_1)D)S}{N} \\ 0 \\ 0 \end{bmatrix}$$

The rate of other transition between infectious classes is denote by $\mathcal{V}$ and given by

$$\mathcal{V}(X) = \begin{bmatrix} (\mu+\gamma+\omega)E \\ -\gamma E + (\mu+\delta+\eta)I \\ -(\mu+\delta)I + \alpha D \end{bmatrix}.$$

where

$$D\mathcal{F} = \begin{bmatrix} \frac{\partial \mathcal{F}_1}{\partial E} & \frac{\partial \mathcal{F}_1}{\partial I} & \frac{\partial \mathcal{F}_1}{\partial D} \\ \frac{\partial \mathcal{F}_2}{\partial E} & \frac{\partial \mathcal{F}_2}{\partial I} & \frac{\partial \mathcal{F}_2}{\partial D} \\ \frac{\partial \mathcal{F}_3}{\partial E} & \frac{\partial \mathcal{F}_3}{\partial I} & \frac{\partial \mathcal{F}_3}{\partial D} \end{bmatrix} = \begin{bmatrix} c_{11} & c_{12} & c_{13} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

$$\begin{aligned} c_{11} &= -\frac{(\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2} \\ c_{12} &= \frac{\beta_1(1-\xi_1)(1-\xi_2)SN - (\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2} \\ c_{13} &= \frac{\kappa\beta_2(1-\xi_1)SN}{N^2} \end{aligned}$$

and

$$D\mathcal{V} = \begin{bmatrix} \frac{\partial \mathcal{V}_1}{\partial E} & \frac{\partial \mathcal{V}_1}{\partial I} & \frac{\partial \mathcal{V}_1}{\partial D} \\ \frac{\partial \mathcal{V}_2}{\partial E} & \frac{\partial \mathcal{V}_2}{\partial I} & \frac{\partial \mathcal{V}_2}{\partial D} \\ \frac{\partial \mathcal{V}_3}{\partial E} & \frac{\partial \mathcal{V}_3}{\partial I} & \frac{\partial \mathcal{V}_3}{\partial D} \end{bmatrix} = \begin{bmatrix} \mu+\gamma+\omega & 0 & 0 \\ -\gamma & \mu+\delta+\eta & 0 \\ 0 & -(\mu+\delta) & \alpha \end{bmatrix}.$$

At disease free equilibrium point the matrice $D\mathcal{F}$ and $D\mathcal{V}$ will give us

$$D\mathcal{F} = \begin{bmatrix} 0 & \beta_1(1-\xi_1)(1-\xi_2) & \kappa\beta_2(1-\xi_1) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$D\mathcal{V} = \begin{bmatrix} \mu+\gamma+\omega & 0 & 0 \\ -\gamma & \mu+\delta+\eta & 0 \\ 0 & -(\mu+\delta) & \alpha \end{bmatrix}.$$

Then, the inverse matrix $D\mathcal{V}$ is obtained in the form of

$$(D\mathcal{V})^{-1} = \begin{bmatrix} \frac{1}{\mu+\gamma+\omega} & 0 & 0 \\ \frac{\gamma}{(\mu+\gamma+\omega)(\mu+\delta+\eta)} & \frac{1}{\mu+\delta+\eta} & 0 \\ \frac{\gamma(\mu+\delta)}{\alpha(\mu+\gamma+\omega)(\mu+\delta+\eta)} & \frac{\mu+\delta}{\alpha(\mu+\delta+\eta)} & \frac{1}{\alpha} \end{bmatrix}$$

The next-generation matrix is defined as

$$
\begin{aligned}
K &= (D\mathcal{F})(D\mathcal{V})^{-1} \\
&= \begin{bmatrix} 0 & \beta_1(1-\xi_1)(1-\xi_2) & \kappa\beta_2(1-\xi_1) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\mu+\gamma+\omega} & 0 & 0 \\ \frac{\gamma}{(\mu+\gamma+\omega)(\mu+\delta+\eta)} & \frac{1}{\mu+\delta+\eta} & 0 \\ \frac{\gamma(\mu+\delta)}{\alpha(\mu+\gamma+\omega)(\mu+\delta+\eta)} & \frac{\mu+\delta}{\alpha(\mu+\delta+\eta)} & \frac{1}{\alpha} \end{bmatrix} \\
&= \begin{bmatrix} \frac{\gamma(\alpha\beta_1(1-\xi_1)(1-\xi_2)+\kappa\beta_2(1-\xi_1)(\mu+\delta))}{\alpha(\mu+\gamma+\omega)(\mu+\delta+\eta)} & \frac{\alpha\beta_1(1-\xi_1)(1-\xi_2)+\kappa\beta_2(1-\xi_1)(\mu+\delta)}{\alpha(\mu+\delta+\eta)} & \frac{\kappa\beta_2(1-\xi_1)}{\alpha} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}
\end{aligned}
$$

The basic reproduction number is obtained from the spectral radius of matrix K [4], i.e.

$$\mathcal{R}_0 = \rho(K) = \frac{\gamma(\alpha\beta_1(1-\xi_1)(1-\xi_2) + \kappa\beta_2(1-\xi_1)(\mu+\delta))}{\alpha(\mu+\gamma+\omega)(\mu+\delta+\eta)}. \tag{3.4}$$

The endemic equilibrium point in (3.3) can be expressed in $\mathcal{R}_0$ as follows

$$
\begin{aligned}
S^* &= \frac{\alpha N(\mu+\gamma+\omega)(\mu+\delta+\eta)}{\gamma(\alpha\beta_1(1-\xi_1)(1-\xi_2) + \kappa\beta_2(1-\xi_1)(\mu+\delta))}, \\
E^* &= \frac{\Lambda(\mu+\theta)(\mu+\delta+\eta)(\mathcal{R}_0-1)}{(\mu(\mu+\delta+\eta)(\mu+\gamma+\omega+\theta) + \gamma\theta(\mu+\delta))\mathcal{R}_0}, \\
I^* &= \frac{\Lambda\gamma(\mu+\theta)(\mathcal{R}_0-1)}{(\mu(\mu+\delta+\eta)(\mu+\gamma+\omega+\theta) + \gamma\theta(\mu+\delta))\mathcal{R}_0}, \\
R^* &= \frac{\Lambda(\omega(\mu+\delta+\eta)+\eta\gamma)(\mathcal{R}_0-1)}{(\mu(\mu+\delta+\eta)(\mu+\gamma+\omega+\theta) + \gamma\theta(\mu+\delta))\mathcal{R}_0}, \\
D^* &= \frac{\Lambda\gamma(\mu+\theta)(\mu+\delta)(\mathcal{R}_0-1)}{\alpha(\mu(\mu+\delta+\eta)(\mu+\gamma+\omega+\theta) + \gamma\theta(\mu+\delta))\mathcal{R}_0}.
\end{aligned}
$$

The endemic equilibrium point will be exists if $\mathcal{R}_0 > 1$.

3.4. Stability of equilibrium point

The stability of the equilibrium point is determined by using linearization of the system of differential equations (3.1). The Jacobian matrix in the form as follows

$$J = \begin{bmatrix} J_{11} & J_{12} & J_{13} & J_{14} & J_{15} \\ J_{21} & J_{22} & J_{23} & J_{24} & J_{25} \\ 0 & \gamma & -(\mu+\delta+\eta) & 0 & 0 \\ 0 & \omega & \eta & -(\mu+\theta) & 0 \\ 0 & 0 & \mu+\delta & 0 & -\alpha \end{bmatrix} \tag{3.5}$$

where

$$
\begin{aligned}
J_{11} &= \frac{-(\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)N + (\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2} - \mu, \\
J_{12} &= \frac{(\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2}, \\
J_{13} &= \frac{-\beta_1(1-\xi_1)(1-\xi_2)SN + (\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2}, \\
J_{14} &= \frac{(\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2} + \theta, \\
J_{15} &= \frac{-\kappa\beta_2(1-\xi_1)S}{N},
\end{aligned}
$$

$$J_{21} = \frac{(\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)N - (\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2},$$
$$J_{22} = \frac{-(\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2} - (\mu+\gamma+\omega),$$
$$J_{23} = \frac{\beta_1(1-\xi_1)(1-\xi_2)SN - (\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)S}{N^2},$$
$$J_{24} = \frac{-(\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_2)D)S}{N^2},$$
$$J_{25} = \frac{\kappa\beta_2(1-\xi_1)S}{N}.$$

The eigenvalues of the Jacobian matrix J equation (3.5) can be used to determine the stability of the equilibrium point.

3.4.1. Stability of disease-free equilibrium point

The Jacobian matrix of the disease-free equilibrium point is obtained by substituting the disease-free equilibrium point (3.2) into the Jacobian matrix (3.5), resulting in the following matrix:

$$J(E_0) = \begin{bmatrix} -\mu & 0 & c_1 & \theta & c_2 \\ 0 & c_3 & -c_1 & 0 & -c_2 \\ 0 & \gamma & c_4 & 0 & 0 \\ 0 & \omega & \eta & c_5 & 0 \\ 0 & 0 & c_6 & 0 & -\alpha \end{bmatrix} \tag{3.6}$$

where

$$c_1 = -\beta_1(1-\xi_1)(1-\xi_2),$$
$$c_2 = -\kappa\beta_2(1-\xi_1),$$
$$c_3 = -(\mu+\gamma+\omega),$$
$$c_4 = -(\mu+\delta+\eta),$$
$$c_5 = -(\mu+\theta),$$
$$c_6 = -(\mu+\delta).$$

We get eigenvalue from Jacobian matrix (3.6) are $\lambda_1 = -\mu, \lambda_2 = -(\mu+\theta)$ and characteristic polynomial

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$$

such that

$$a_0 = 1,$$
$$a_1 = \alpha - c_4 - c_3,$$
$$a_2 = -\alpha c_4 - \alpha c_3 - c_3 c_4 + \gamma c_1,$$
$$a_3 = \alpha c_3 c_4 + \alpha\gamma c_1 + \gamma c_2 c_6.$$

To determine the stability of the disease-free equilibrium, all roots of the characteristic equation must have negative real parts. This condition can be verified using the Routh–Hurwitz criteria [17], which require

$$a_0 > 0, \quad a_1 > 0, \quad a_1 - a_3 > 0, \quad \text{and} \quad a_3 > 0.$$

It is straightforward to verify that $a_0 > 0$ and $a_1 > 0$. Furthermore, the coefficient a_3 is given by

$$\begin{aligned} a_3 &= \alpha c_3 J_{33} + \alpha\gamma c_1 + \gamma c_2 J_{53} > 0 \\ \alpha(\mu+\gamma+\omega)(\mu+\delta+\eta) - \alpha\gamma\beta_1(1-\xi_1)(1-\xi_2) + \gamma\kappa\beta_2(1-\xi_1)(\mu+\delta) &> 0 \\ 1 - \mathcal{R}_0 &> 0. \end{aligned}$$

Hence, $a_3 > 0$ if and only if $\mathcal{R}_0 < 1$. It follows that the disease-free equilibrium is locally asymptotically stable whenever $\mathcal{R}_0 < 1$.

3.4.2. Stability of endemic equilibrium point

The Jacobian matrix of the endemic equilibrium point is obtained by substituting the endemic equilibrium point (3.3) into the Jacobian matrix (3.5), resulting in the following matrix:

$$J(E_1) = \begin{bmatrix} d_1 & d_2 & d_3 & d_4 & d_5 \\ d_6 & d_7 & d_8 & d_9 & d_10 \\ 0 & \gamma & c_4 & 0 & 0 \\ 0 & \omega & \eta & c_5 & 0 \\ 0 & 0 & c_6 & 0 & -\alpha \end{bmatrix} \tag{3.7}$$

where

$$\begin{aligned} d_1 &= \frac{-(\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)N + (\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)S^*}{N^2} - \mu, \\ d_2 &= \frac{(\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)S^*}{N^2}, \\ d_3 &= \frac{-\beta_1(1-\xi_1)(1-\xi_2)S^*N + (\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)S^*}{N^2}, \\ d_4 &= \frac{(\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)S^*}{N^2} + \theta, \\ d_5 &= \frac{-\kappa\beta_2(1-\xi_1)S^*}{N}, \\ d_6 &= \frac{(\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)N - (\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)S^*}{N^2}, \\ d_7 &= \frac{-(\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)S^*}{N^2} - (\mu+\gamma+\omega), \\ d_8 &= \frac{\beta_1(1-\xi_1)(1-\xi_2)S^*N - (\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*)S^*}{N^2}, \\ d_9 &= \frac{-(\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_2)D^*)S^*}{N^2}, \\ d_{10} &= \frac{\kappa\beta_2(1-\xi_1)S^*}{N}. \end{aligned}$$

Characteristic polynomial of equation (3.7) is given by

$$a_0\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0$$

such that

$$\begin{aligned} a_0 &= 1, \\ a_1 &= \alpha - d_1 - c_5 - d_7 - c_4, \\ a_2 &= c_5 d_1 + d_1 d_7 + c_4 d_1 + c_5 d_7 + c_4 c_5 + c_4 d_7 - \alpha d_1 - \alpha c_5 - \alpha d_7 - \alpha c_4 - \omega d_9 - \gamma d_8 - d_2 d_6, \end{aligned}$$

$$
\begin{aligned}
a_3 &= \alpha c_5 d_1 + \alpha d_1 d_7 + \alpha c_4 d_1 + \alpha c_5 d_7 + \alpha c_4 c_5 + \alpha c_4 d_7 + \omega d_1 d_9 + \gamma d_1 d_8 + \omega c_4 d_9 + \gamma c_5 d_8 \\
&\quad + c_5 d_2 d_6 + c_4 d_2 d_6 - \alpha\gamma d_8 - \gamma c_6 d_{10} - \alpha\omega d_9 - \alpha d_2 d_6 - c_5 d_1 d_7 - c_4 c_5 d_1 - c_4 d_1 d_7 - \eta\gamma d_9 \\
&\quad - c_4 c_5 d_7 - \omega d_4 d_6 - \gamma d_3 d_6, \\
a_4 &= \gamma c_5 c_6 d_{10} + \gamma c_6 d_1 d_{10} + \alpha\omega d_1 d_9 + \alpha\gamma d_1 d_8 + \alpha\omega c_4 d_9 + \alpha\gamma c_5 d_8 + \alpha c_5 d_2 d_6 + \alpha c_4 d_2 d_6 \\
&\quad + \eta\gamma d_1 d_9 + c_4 c_5 d_1 d_7 + \omega c_4 d_4 d_6 + \gamma c_5 d_3 d_6 - \gamma c_6 d_5 d_6 - \alpha c_5 d_1 d_7 - \alpha c_4 c_5 d_1 - \alpha c_4 d_1 d_7 \\
&\quad - \alpha\eta\gamma d_9 - \alpha c_4 c_5 d_7 - \alpha\omega d_4 d_6 - \alpha\gamma d_3 d_6 - \omega c_4 d_1 d_9 - \gamma c_5 d_1 d_8 - \eta\gamma d_4 d_6 - c_4 c_5 d_2 d_6, \\
a_5 &= \gamma c_5 c_6 d_5 d_6 + \alpha\eta\gamma d_1 d_9 + \alpha c_4 c_5 d_1 d_7 + \alpha\omega c_4 d_4 d_6 + \alpha\gamma c_5 d_3 d_6 - \alpha\omega c_4 d_1 d_9 - \alpha\gamma c_5 d_1 d_8 \\
&\quad - \alpha\eta\gamma d_4 d_6 - \alpha c_4 c_5 d_2 d_6 - \gamma c_5 c_6 d_1 d_{10}.
\end{aligned}
$$

Since the eigenvalue is difficult to determine, we use the Routh-Hurwirtz criteria to determine the stability of the endemic equilibrium point [17]. It needs to be shown that $a_0 > 0, a_1 > 0, b_1 = \frac{a_1 a_2 - a_3 a_4}{a_1} > 0, b_2 = \frac{a_1 a_4 - a_0 a_5}{a_1}, e_1 = \frac{b_1 a_3 - a_1 b_2}{b_1} > 0, e_2 = \frac{b_1 a_5 - a_{10}}{b_1}, f_1 = \frac{e_1 b_2 - b_1 e_2}{e_1} > 0$ and $g_1 = f_1 > 0$. Due to the analytical complexity of the nonlinear system, this condition cannot be rigorously established in closed form. Therefore, it is demonstrated through numerical simulations.

3.4.3. Global stability of the disease-free equilibrium point

Theorem 3.2. *The disease-free equilibrium point E_0 is globally asymptotically stable in the domain Ω if $\mathcal{R}_0 < 1$ and the following criteria are satisfied:*

1. *$\vec{X}^*$ is globally asymptotically stable for the system $\frac{d\vec{X}}{dt} = \vec{F}(\vec{X}, \vec{0})$.*
2. *$G(\vec{X}, \vec{Z}) = B\vec{Z} - \hat{G}(\vec{X}, \vec{Z})$, where $\hat{G}(\vec{X}, \vec{Z}) \geq 0$ for all $(\vec{X}, \vec{Z}) \in \Omega$, and $B = D\vec{Z}G(\vec{X}^*, \vec{0})$ is an M-matrix (Metzler matrix), which is a matrix with all the off-diagonal elements non-negative.*

Proof. We use the methods of Castilo-Chavez et al. [5] to prove this theorem. We start by rewriting model (3.1) as

$$
\frac{d\vec{X}}{dt} = F(\vec{X}, \vec{Z}) = \begin{bmatrix} \Lambda - \left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N}\right) S - \mu S + \theta R \\ \omega E + \eta I - (\mu + \theta) R \end{bmatrix},
$$

$$
\frac{d\vec{Z}}{dt} = G(\vec{X}, \vec{Z}) = \begin{bmatrix} \left(\frac{\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D}{N}\right) S - (\mu + \gamma + \omega) E \\ \gamma E - (\mu + \delta + \eta) I \\ (\mu + \delta) I - \alpha D \end{bmatrix},
$$

with $X = (S, R) \in R_+^2$ denotes the class of uninfected individuals, $Z = (E, I, D) \in R_+^3$ denotes the class of infected individuals. Let $E_0 = (\vec{X}^*, \vec{0})$ with $\vec{X}^* = \left(\frac{\Lambda}{\mu}, 0\right)$.

We will verify condition (1) in Theorem 3.2. Consider the system

$$
\frac{d\vec{X}}{dt} = F\left(\vec{X}, \vec{0}\right) = \begin{bmatrix} \frac{dS(t)}{dt} \\ \frac{dR(t)}{dt} \end{bmatrix} = \begin{bmatrix} \Lambda - \mu S + \theta R \\ -(\mu + \theta) R \end{bmatrix}. \tag{3.8}
$$

solving the equation (3.8) so that it is obtained

$$
\vec{X}(t) = \begin{bmatrix} S(t) \\ R(t) \end{bmatrix} = \begin{bmatrix} \frac{\Lambda}{\mu} + \left(S(0) - \frac{\Lambda}{\mu} - \frac{\theta R(0)}{\mu + \theta}\right) e^{-\mu t} + \frac{\theta R(0)}{\mu + \theta} e^{-(\mu + \theta) t} \\ R(0) e^{-(\mu + \theta) t} \end{bmatrix}.
$$

Because $\lim_{t \to \infty} S(t) = \frac{\Lambda}{\mu}$ and $\lim_{t \to \infty} R(t) = 0$, then the equilibrium point $\vec{X}^* = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$ is globally asymptotically stable. To verify condition (2) in Theorem 3.2 it is necessary to determine the matrix $B = D_Z G(\vec{X}^*, 0)$ which is a Metzler matrix with all the off-diagonal elements are non-negative.

$$
B = \begin{bmatrix} -(\mu + \gamma + \omega) & \beta_1(1 - \xi_1)(1 - \xi_2) & \kappa\beta_2(1 - \xi_1) \\ \gamma & -(\mu + \delta + \eta) & 0 \\ 0 & \mu + \delta & -\alpha \end{bmatrix}.
$$

Then, calculate $\hat{G}(\vec{X},\vec{Z}) = BZ - G(\vec{X},\vec{Z})$.

$$\begin{aligned}\hat{G}(\vec{X},\vec{Z}) &= BZ - G(\vec{X},\vec{Z}) \\ &= \begin{bmatrix}(\beta_1(1-\xi_1)(1-\xi_2)I + \kappa\beta_2(1-\xi_1)D)(1-\frac{S}{N}) \\ 0 \\ 0\end{bmatrix}.\end{aligned}$$

Since $S \le N$, then $\frac{S}{N} \le 1$ so that $1 - \frac{S}{N} \ge 0$. Thus, it is obtained that $\hat{G}(\vec{X},\vec{Z})$ is nonnegative. Since conditions (1) and (2) are fulfilled, we can conclude that the disease-free equilibrium point is globally asymptotically stable if $\mathcal{R}_0 < 1$. □

3.4.4. Global stability of endemic equilibrium point

Theorem 3.3. *Consider the SEIRD model system* (3.1) *with positive initial conditions. If* $\mathcal{R}_0 > 1$*, then the endemic equilibrium* $E_1 = (S^*, E^*, I^*, R^*, D^*)$ *is globally asymptotically stable in the interior of the feasible region*

$$\Omega = \left\{(S,E,I,R,D) \in \mathbb{R}^5_+ : S + E + I + R \le \frac{\Lambda}{\mu}\right\}.$$

Proof. Define the Lyapunov function $\mathcal{L} : \Omega \to \mathbb{R}_+$ by

$$\begin{aligned}\mathcal{L}(S,E,I,R,D) = &\left(S - S^* - S^* \ln\frac{S}{S^*}\right) + \left(E - E^* - E^* \ln\frac{E}{E^*}\right) \\ &+ \frac{\mu+\gamma+\omega}{\gamma}\left(I - I^* - I^* \ln\frac{I}{I^*}\right) + \frac{\theta}{2(\mu+\theta)}(R-R^*)^2 \\ &+ \frac{\kappa\beta_2(1-\xi_1)S^*}{2\alpha N^*}(D-D^*)^2.\end{aligned}$$

The function $\mathcal{L}$ is continuously differentiable in Ω, satisfies $\mathcal{L}(E_1) = 0$, and $\mathcal{L} > 0$ for all $(S,E,I,R,D) \ne E_1$.

Differentiating $\mathcal{L}$ along the trajectories of system (3.1) yields

$$\begin{aligned}\frac{d\mathcal{L}}{dt} = &\left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{E^*}{E}\right)\frac{dE}{dt} + \frac{\mu+\gamma+\omega}{\gamma}\left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} \\ &+ \frac{\theta}{\mu+\theta}(R-R^*)\frac{dR}{dt} + \frac{\kappa\beta_2(1-\xi_1)S^*}{\alpha N^*}(D-D^*)\frac{dD}{dt}.\end{aligned}$$

Substituting the right-hand sides of (3.1) and using the equilibrium relations

$$\begin{aligned}\Lambda &= \lambda^* S^* + \mu S^* - \theta R^*, \\ \lambda^* S^* &= (\mu+\gamma+\omega)E^*, \\ \gamma E^* &= (\mu+\delta+\eta)I^*, \\ \omega E^* + \eta I^* &= (\mu+\theta)R^*, \\ (\mu+\delta)I^* &= \alpha D^*,\end{aligned}$$

where

$$\lambda^* = \frac{\beta_1(1-\xi_1)(1-\xi_2)I^* + \kappa\beta_2(1-\xi_1)D^*}{N^*},$$

straightforward algebraic manipulations give

$$\begin{aligned}\frac{d\mathcal{L}}{dt} = &-\mu\frac{(S-S^*)^2}{S} - \frac{\theta(\mu+\theta)}{2}(R-R^*)^2 - \frac{\kappa\beta_2(1-\xi_1)S^*}{2N^*}(D-D^*)^2 + \lambda^* S^*\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right) \\ &+ \beta_1(1-\xi_1)(1-\xi_2)I^*S^*\left(1 - \frac{S^*NI}{SN^*I^*} + \ln\frac{S^*NI}{SN^*I^*}\right) \\ &+ \kappa\beta_2(1-\xi_1)D^*S^*\left(1 - \frac{S^*ND}{SN^*D^*} + \ln\frac{S^*ND}{SN^*D^*}\right).\end{aligned}$$

Using the arithmetic–geometric mean inequality $1 - x + \ln x \leq 0$ for all $x > 0$, with equality if and only if $x = 1$, and noting that $2 - \frac{S^*}{S} - \frac{S}{S^*} \leq 0$, it follows that

$$\frac{d\mathcal{L}}{dt} \leq -\mu\frac{(S - S^*)^2}{S} - \frac{\theta(\mu + \theta)}{2}(R - R^*)^2 - \frac{\kappa\beta_2(1 - \xi_1)S^*}{2N^*}(D - D^*)^2 \leq 0.$$

Moreover, $\frac{d\mathcal{L}}{dt} = 0$ if and only if $(S, E, I, R, D) = E_1$. By LaSalle's invariance principle, every solution of system (3.1) with initial conditions in the interior of Ω converges to E_1 as $t \to \infty$. Therefore, the endemic equilibrium E_1 is globally asymptotically stable in Ω. □

3.5. Numerical simulation

To confirm our previous analytical results, we solve the $SEIRD$ epidemic model (3.1) using the fourth-order Runge-Kutta method. If not stated otherwise, our simulation uses parameter values as in Table 2.

Parameter	**Value**	**Source**
Λ	6295.16	[27]
β_1	0.65	[16]
β_2	0.651	[29]
κ	0.35	[25]
ξ_1	0.15	Assumed
ξ_2	0.15	Assumed
γ	1	[16]
ω	0.09	Fixed
δ	0.771	[30]
η	0.091	[29]
θ	0.851	[29]
α	0.5	[29]
μ	0.16	[27]

Table 2: Parameter Values of the $SEIRD$ Model

Three initial value NA1 = (5000, 1000, 500, 50, 10), NA2 = (1500, 1000, 100, 5, 3), NA3 = (900, 40, 10, 1, 2). Using these parameter values, we get $\mathcal{R}_0 = 0.8399 < 1$. The Figure 2 represents the phase portrait of the $SEIRD$ model (Susceptible, Exposed, Infected, Recovered, Dead) under the condition $\mathcal{R}_0 < 1$. It illustrates the trajectories of population groups S (Susceptible), E (Exposed), and I (Infected) from three different initial conditions. All trajectories converge to the disease-free equilibrium ($E_0 = (39344.7, 0, 0, 0, 0)$), reflecting the absence of an epidemic. We can says that the disease-free equilibrium point E_0 is globally asymtotically stable. The phase portrait demonstrates that all trajectories are stable at the disease-free equilibrium (E_0) where the susceptible population dominates. With $\mathcal{R}_0 < 1$, the disease cannot persist in the population, preventing an outbreak. The initial states influence the path but not the final outcome, which is the eradication of the disease. This highlights the critical role of maintaining $\mathcal{R}_0 < 1$ to control and eliminate the spread of infectious diseases. For the second numerical simulation, we take a smaller value of rate of disposition dead bodies $\beta_1 = 1$, then we get the basic reproduction number in this case is $\mathcal{R}_0 = 1.1166 > 1$. The phase space diagram shows the dynamic behavior of a $SEIRD$ epidemic model for $\mathcal{R}_0 > 1$, where the basic reproduction number exceeds the critical threshold, leading to disease persistence. The trajectories indicate how the system evolves over time under different initial conditions. The disease-free equilibrium point is unstable since the trajectories move away from it, reflecting the spread of the disease in the population when $\mathcal{R}_0 > 1$. Conversely, the endemic equilibrium point exists and it hence is globally asymptotically stable. This stability properties is confirmed by our simulation depicted in Figure 3, that is the numerical solution is convergent to the endemic equilibrium point $E_1 = (32747.5, 954.797, 954.797, 151.2549, 1756.8265)$

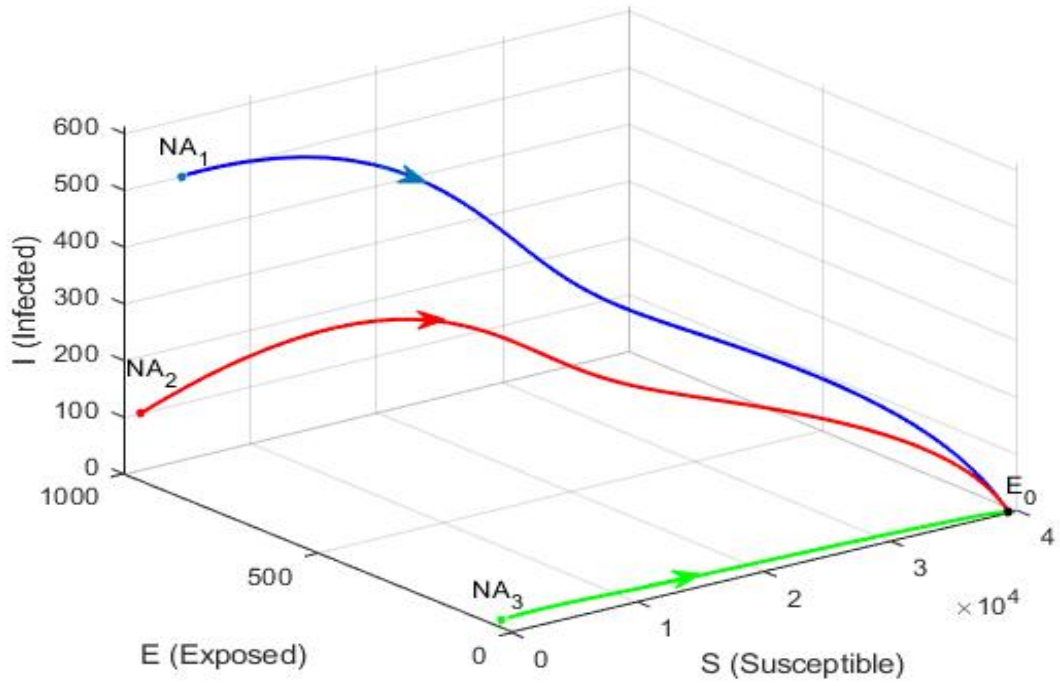

Figure 2: Phase Portrait of $SEIRD$ Model for $\mathcal{R}_0 < 1$ in SEI-space

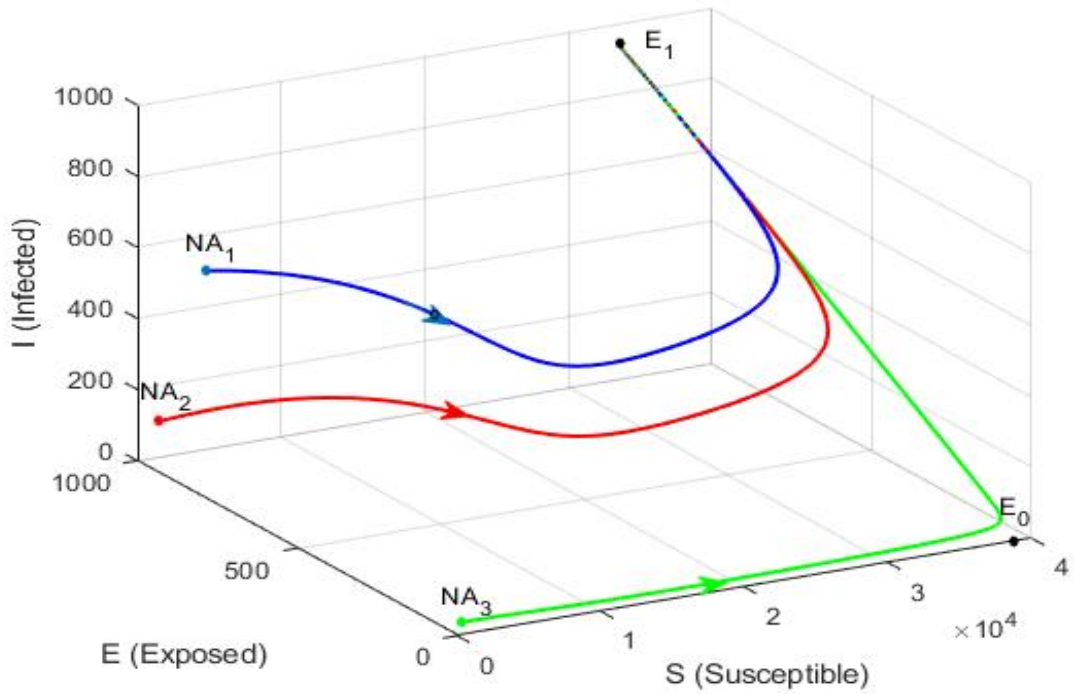

Figure 3: Phase Portrait of $SEIRD$ Model for $\mathcal{R}_0 > 1$ in SEI-space

4. Conclusion

The $SEIRD$ model of Nipah virus transmission exhibits two equilibrium points: the disease-free equilibrium (DFE) and the endemic equilibrium (EE). The DFE is asymptotically stable when $\mathcal{R}_0 < 1$, indicating that the disease will eventually be eradicated, whereas the EE is asymptotically stable when $\mathcal{R}_0 > 1$, implying that the disease will persist within the population. Numerical simulations confirm the analytical results and demonstrate how variations in key parameters influence the disease dynamics.

The findings have significant real-world implications, showing that unprotected contact with deceased individuals and the incubation period substantially affect the transmission of the Nipah virus. This suggests that public health interventions focusing on safe burial practices, timely isolation of exposed individuals, and community awareness campaigns can effectively reduce disease spread.

However, the current model assumes homogeneous mixing within the population and neglects spatial effects, stochasticity, and demographic heterogeneity, which may limit its applicability to complex real-world scenarios. Future research could extend the present model by incorporating spatial diffusion, age structure, or stochastic effects, as well as validating the model using empirical data from Nipah virus outbreaks to enhance its predictive accuracy and practical relevance.

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