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# Analyzing Vaccine Efficacy: Stability Analysis of SEIR Model for Nipah Virus in India and Nepal

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***Abstract:-***This study investigates the efficacy of vaccination in controlling Nipah virus (NiV) outbreaks using SEIR modeling in India and Nepal. Leveraging mathematical modeling, it assesses vaccination strategies' impact on NiV transmission dynamics. The SEIR model, applied to infectious diseases, facilitates understanding disease spread and intervention effects. Extending this approach to NiV outbreaks aims to inform effective public health measures. Integration of empirical data enhances model validity. Findings offer insights into vaccine effectiveness, aiding evidence-based decision-making. This research contributes to combating NiV, a lethal zoonotic pathogen.

***Keywords****: Nipah virus (NiV), Vaccination efficacy, Outbreaks, Epidemiology, Transmission*.

## 1. Introduction

The emergence and spread of infectious diseases pose significant threats to global public health, necessitating rigorous research and analysis to develop effective preventive measures. Among the various infectious agents, Nipah virus (NiV) stands out as a particularly lethal zoonotic pathogen, capable of causing severe respiratory and neurological symptoms in humans [1]. Originating from fruit bats of the genus Pteropus, NiV outbreaks have been documented primarily in South and Southeast Asia, with sporadic cases reported in other regions as well [2, 3]. The gravity of the Nipah virus stems not only from its high fatality rate but also from its potential to trigger large-scale outbreaks with devastating consequences [4].

In light of the pressing need to mitigate the impact of NiV outbreaks, understanding the dynamics of virus transmission and assessing the efficacy of potential interventions are paramount. Vaccination represents one of the most effective strategies for controlling infectious diseases by conferring immunity and preventing transmission [5, 6]. However, the development and deployment of vaccines necessitate a comprehensive understanding of vaccine efficacy and its implications for population-level disease dynamics.

This research paper aims to analyze the efficacy of vaccination in controlling NiV outbreaks through the stability assessment of a compartmental model, specifically the Susceptible-Exposed-Infectious-Recovered (SEIR) model. By leveraging mathematical modeling techniques, we seek to investigate the impact of vaccination campaigns on the transmission dynamics of NiV in two highly vulnerable regions: India and Nepal. Our study builds upon previous research on SEIR modeling of infectious diseases and extends its application to the context of NiV outbreaks, providing insights into the potential effectiveness of vaccination strategies [7, 8].

The SEIR model, a cornerstone of epidemiological modeling, divides the population into distinct compartments based on their disease status: susceptible (S), exposed (E), infectious (I), and recovered (R) [9]. This compartmental framework allows for the simulation of disease transmission dynamics by tracking the flow of individuals between different states over time. By incorporating parameters such as transmission rates, latent periods, and recovery rates, the SEIR model provides a quantitative framework for assessing the impact of interventions, including vaccination, on disease spread [10].

Previous studies have demonstrated the utility of SEIR modeling in understanding the dynamics of various infectious diseases, including but not limited to influenza, measles, and Ebola [11, 12, 13]. By extending this

approach to the study of NiV outbreaks in India and Nepal, we aim to elucidate the potential effectiveness of vaccination in mitigating the spread of the virus and reducing the burden of disease in these regions [14].

Moreover, our analysis will be informed by relevant empirical data on NiV transmission, epidemiological parameters, and vaccination coverage in India and Nepal. By integrating real-world data with mathematical modeling techniques, we can enhance the validity and applicability of our findings, thereby informing evidence-based decision-making and public health interventions [15, 16, 17].

Vaccine administration is a highly effective method of preventing and reducing viral infections [18]. Even though there is no vaccine or a specific antiviral for the treatment of patients infected with Nipah virus available, the development of ChAdOx1 NipahB vaccine offers promising prospects [19, 20]. Vaccination and optimal control are key points to manage an epidemic situation, as discussed in [21, 22, 23, 24, 25, 26]. In this study, we utilize an SEIR model equipped with the effectiveness of vaccination to forecast the Nipah virus situation upon vaccine availability. Within our model, we classify vaccines into two main types: prophylactic vaccines aimed at prevention and therapeutic vaccines administered post-infection [27].

In summary, this research paper seeks to contribute to our understanding of vaccine efficacy in controlling NiV outbreaks by employing SEIR modeling techniques. By conducting a stability assessment of the SEIR model in the context of India and Nepal, we aim to provide insights into the potential impact of vaccination campaigns on the dynamics of NiV transmission, ultimately guiding efforts to combat this deadly zoonotic pathogen.

## 2. Mathematical Model

We are examining the SEIR model, representing the fractions of susceptible (S), exposed (E), infectious (I), and recovered (R) populations at a given time (t). We disregard the trivial solution where all compartments are empty. The differential equations describing the system, depicted in a schematic diagram given in Fig.[1] are as follows:

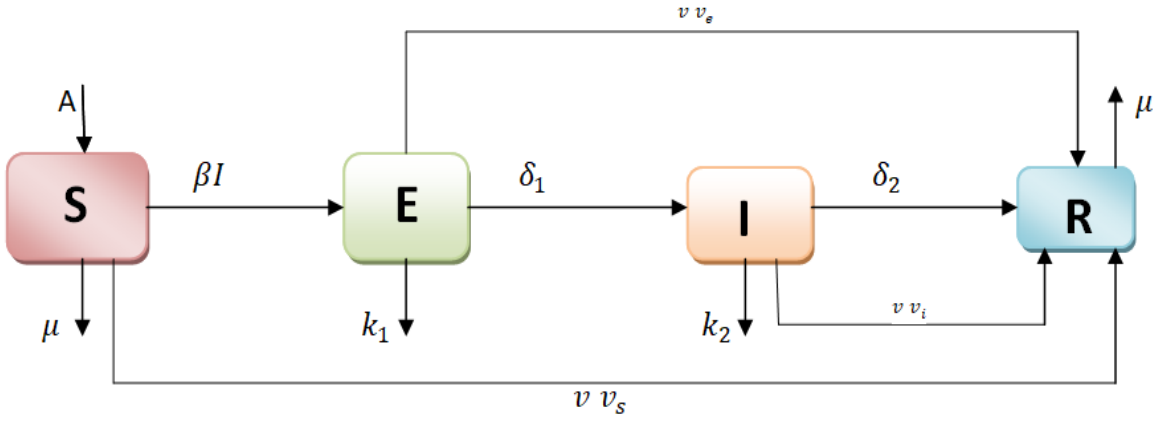



**Figure 1: Flow diagram for a model system (2.1) to (2.4).**

$$\frac{dS}{dt} = A - (v\,v_s + \mu)\,S - \beta\,(1 - v\,v_s)SI, \quad \ldots(2.1)$$

$$\frac{dE}{dt} = \beta\,(1 - v\,v_s)SI - \{k_1 + \delta_1 + (1 - \delta_1)v\,v_e\}E, \quad \ldots(2.2)$$

$$\frac{dI}{dt} = \delta_1\,E - \{k_2 + \delta_2 + (1 - \delta_2)\,v\,v_i\}, \quad \ldots(2.3)$$

$$\frac{dR}{dt} = v\,v_s\,S + v\,v_e\,(1 - \delta_1)\,E + \{\delta_2 + (1 - \delta_2)\,v\,v_i\}\,I - \mu\,R, \quad \ldots(2.4)$$

with the condition that

$$0 \leq S(0), E(0), I(0), R(0) \leq 1 \quad \ldots(2.5)$$

The density $S(t)$ at time $t$ represents the fraction of susceptible individuals, while $E(t)$, $I(t)$and $R(t)$signify similar proportions for exposed, infectious, and recovered individuals, respectively.

Table 1 provides a detailed explanation of the variables and parameters in Equation $(2.1) - (2.4)$.

**Table 1: Table of Description**

| Variable and Parameter | Description |
|---|---|
| $S$ | Fraction of susceptible case |
| $E$ | Fraction of exposed case |
| $I$ | Fraction of infected case |
| $R$ | Fraction of recovered case |
| $\beta$ | Rate of effective transmission of Nipah Virus |
| $\delta_1$ | Rate of change from exposed to infected |
| $\delta_2$ | Rate of change from infected to recovered |
| $v$ | Population's vaccination rate |
| $v_s$ | Vaccination effectiveness in S |
| $v_e$ | Vaccination effectiveness in E |
| $v_i$ | Vaccination effectiveness in I |
| $A$ | Birth rate of population |
| $\mu$ | Death rate of non-Nipah virus population |
| $k_1$ | Death rate plus μ of the exposed population |
| $k_2$ | Death rate plus μ of the infected population |

The model is motivated by the understanding that the vaccination rate per day $(v)$ cannot instantaneously halt the system's dynamics, given that the entire population cannot be vaccinated simultaneously. Individuals become eligible for vaccination once they are susceptible, exposed, or infectious. In the first equation of System (2.1) - (2.4), the change in susceptible individuals depends on both the number of vaccinated individuals, $v\,v_s\,S$, and the number of non-vaccinated individuals, $(1 - v\,v_s)S$. Notably, according to the fundamental existence-uniqueness theorem for nonlinear systems, System (2.1) - (2.4) possesses a unique solution set $(S(t), E(t), I(t), R(t))$. Furthermore, to ensure that the densities S(t), E(t), I(t) and R(t) remain non-negative at any given time $t > 0$ we establish the following lemma.

**Lemma 2.1***If* $(S(t), E(t), I(t), R(t))$*represents the continuous solution of equations (2.1) through (2.4) with the initial condition specified in (2.5), then for any positive time* $t > 0$*, the values of* $(S(t), E(t), I(t), R(t))$ *remain within the range of non-negative real numbers.*

**Proof:-** To establish this lemma, we utilize the property that a function fwith f(0) $f(0) \geq 0$ is non-negative if its derivative $\left[\frac{df}{dt}\right]_{t=u} \geq 0$when f(u) $= 0$; in other words, the function fis non-decreasing at $u$. According to condition (2.5), there exists a time $t_s$such that S(t) $\geq 0$ for$0 \leq t < t_s$ and $S(t_s) = 0$. Referring to the initial equation from (2.1) to (2.4), we find that

$$\left[\frac{dS}{dt}\right]_{t=s} = A \geq 0 \qquad \ldots(2.6)$$

This implies that $S(t) \geq 0$ for any $t \geq 0$ . Next, let $t_i$ be the time such that $I(t) \geq 0$ for $0 \leq t < t_i$ and $I(t_i) = 0$. According to the equation (2.3), we find that

$$\left[\frac{dI}{dt}\right]_{t=i} = \delta_1 E(t_i) \geq 0 \qquad \ldots(2.7)$$

As both S and $I$remain non-negative on the interval $[0, t_i]$.it can be deduced from the equation (2.2) that

$$\frac{dE}{dt} + \{k_1 + \delta_1 + (1 - \delta_1)v\,v_e\}E \geq 0 \qquad \ldots(2.8)$$

$$on\ [0, \quad t_i].$$

Hence,

$$E(t_i) \geq E(0)e^{\{-\{k_1+\delta_1+(1-\delta_1)v\,v_e\}t_i\}}$$

As, $E(0) \geq 0$ and exponential function is always positive then

$$E(t_i) \geq E(0)e^{\{-\{k_1+\delta_1+(1-\delta_1)v\,v_e\}t_i\}} \geq 0 \qquad \ldots(2.9)$$

Equations (2.8) and (2.9) indicate that $\left[\frac{dI}{dt}\right]_{t=i} \geq 0$, thus ensuring I(t) $\geq 0$ for any $t \geq 0$. Additionally, it can be readily verified that $E(t) \geq 0$when $I(t) \geq 0$. Given that S, I, and E are non-negative for t > 0, it is evident that $R(t) \geq 0$for$t \geq 0$.

This lemma allows us to conclude that the set $[0,\infty)^4$ is positively invariant concerning the equation from (2.1) to (2.4) and it acts as an attractor for all solutions of the model.

## 3. Stability of equilibrium points

An equilibrium point in a dynamical system is where there is no change over time in the system's state. Therefore, if a system initiates from an equilibrium point, its state will remain unchanged and persist in equilibrium indefinitely.

To determine the equilibrium points of the system, we solve for the values of $S$, E, I and R where the rates of change$\frac{dS}{dt}$, $\frac{dE}{dt}$,$\frac{dI}{dt}$and $\frac{dR}{dt}$are all equal to zero in equation's (2.1) to (2.4). This involves solving the following system of equations:

$$0 = A - (v\,v_s + \mu)\,S - \beta\,(1 - v\,v_s)SI,$$
$$0 = \beta\,(1 - v\,v_s)SI - \{k_1 + \delta_1 + (1-\delta_1)v\,v_e\}E,$$
$$0 = \delta_1\,E - \{k_2 + \delta_2 + (1-\delta_2)\,v\,v_i\},$$
$$0 = v\,v_s\,S + v\,v_e\,(1-\delta_1)\,E + \{\delta_2 + (1-\delta_2)\,v\,v_i\}\,I - \mu\,R, \ldots(3.1)$$

A disease-free equilibrium represents a state where the disease does not propagate, with both the exposed and infected populations effectively reduced to zero, denoted as E $\equiv 0$ and I $\equiv 0$ By solving equation (3.1), the unique form of the disease-free equilibrium is derived as

$$(S_f, E_f, I_f, R_f) = \left(\frac{A}{vv_s+\mu}, 0, 0, \frac{A}{\mu}\right) \qquad \ldots(3.2)$$

For constant parameters A, v, $v_s$ and μ.

Beyond the disease-free equilibrium, additional equilibrium points, known as the endemic equilibrium, can be determined by solving equation (3.1) while adhering to the constraints that S $\neq 0$, E $\neq 0$, I $\neq 0$, and R $\neq 0$. This is applicable because the state variables (S(t), E(t), I(t), R(t)) are within the interval $[0,\infty]^4$, as proven in Lemma 2.1. This endemic equilibrium is unique under the constraints of the equation (2.1) to (2.4) under fixed parameters and is represented in the following form

$$(\tilde{S}, \tilde{E}, \tilde{I}, \tilde{R}) \qquad \ldots(3.3)$$

Where, $\tilde{S} = \frac{A}{v\,v_s+\mu+\beta\,(1-v\,v_s)\,\tilde{I}}$,

$\tilde{E} = \frac{A-(v\,v_s+\mu)\,\tilde{S}}{k_1+\delta_1+(1-\delta_1)\,v\,v_e}$,

$\tilde{I} = \frac{\delta_1\tilde{E}}{k_2+\delta_2+(1-\delta_2)\,v\,v_i}$,

$\tilde{R} = \frac{A-\mu\tilde{S}-k_1\,E-k_2\tilde{I}}{\mu}$.

Now let $(S^m, E^m, I^m, R^m)$ … (3.4)

Commonly represent an equilibrium point expressed in the form of equations (3.2) or (3.3). The subsequent theorem elucidates the stability of the equilibrium point denoted as (3.4).

**Theorem 2.2**. *With the parameters of the equation (2.1) to (2.4) held constant along with the initial condition (2.5), the equilibrium point of the model exhibits local asymptotic stability*.

**Proof.** Examine the Jacobian matrix of system (2.1) to (2.4) concerning equilibrium point (3.4), presented as follows:

$$J_E = \begin{bmatrix} -v\,v_s & 0 & -L_1 S^m & 0 \\ L_1 I^m & -(k_1 + L_2) & -L_1 S^m & 0 \\ 0 & \delta_1 & -(k_2 + L_3) & 0 \\ v\,v_s & 8\,L_2 - \delta_1 & L_3 & -\mu \end{bmatrix}$$

In the given context, we define $L_1 = \beta\,(1 - \mathrm{v\,v_s}), L_2 = \delta_1 + (1 - \delta_1)\,\mathrm{v\,v_e}$ , $L_3 = \delta_2 + (1 - \delta_2)\mathrm{v\,v_i}$. The eigenvalues, denoted as $ \lambda $, of the matrix $J_E $ are obtained by solving the characteristic polynomial equation$|\lambda I - J_E| = 0$; i.e.the eigenvalues represent the solutions that satisfy this polynomial equation.

$$(\mu + \lambda)(M_1 + M_2\,\lambda + M_3\lambda^2 + \lambda^3) = 0$$

Where,

$$\mathrm{M_1 = L_1^3 I^m S^m + vv_s(L_2L_3 + L_3k_1 + L_2k_2 + L_1^2 S^m)},$$

$$\mathrm{M_2 = L_2L_3 + L_3k_1 + L_2k_2 + k_1k_2 + L_1^2 S^m + vv_s(L_2 + L_3 + k_1 + k_2)},$$

$$\mathrm{M_3 = L_2 + L_3 + k_1 + k_2 + vv_s}.$$

It is evident that $\mathrm{M_1} > 0, \mathrm{M_2} > 0$ and $\mathrm{M_3} > 0$ . Given that $\mathrm{M_1}$, $\mathrm{M_2}$ and M_3 are positive real numbers, it logically follows that all solutions of equation (3.5) possess negative real parts. Consequently, the equilibrium point of system from (2.1) to (2.4) exhibits local asymptotic stability.

## 4. Basic reproduction number and global stability

Utilizing the matrix generation technique {\color{red} [27], the fundamental reproductive number, denoted as $R_0$ corresponds to the principal eigenvalue, known as the spectral radius, of the matrix $FV^{-1}$ , where,

$$F = \begin{bmatrix} 0 & \beta\,(1 - vv_s)S \\ \delta_1 & 0 \end{bmatrix} \qquad \ldots(4.1)$$

and

$$V = \begin{bmatrix} k_1 + \delta_1 + (1 - \delta_1)\,v\,v_e & 0 \\ 0 & k_2 + \delta_2 + (1 - \delta_2)\,v\,v_i \end{bmatrix} \qquad \ldots(4.2)$$

Thus, the basic reproductive number, denoted as $R_0$, associated with the disease-free equilibrium (3.2), takes the following form

$$R_0 = \sqrt{\frac{\delta_1\,\beta\,(1 - \mathrm{v\,v_s})\,\mathrm{A}}{(\mathrm{k_1} + \delta_1 + (1 - \delta_1)\,\mathrm{v\,v_e})(\mathrm{k_2} + \delta_2 + (1 - \delta_2)\,\mathrm{v\,v_i})(\mathrm{vv_s} + \mathrm{A})\}}} \qquad \ldots (4.3)$$

Through equations (4.1) and (4.2), we observe that the principal eigenvalues of $FV^{-1}$and $V^{-1}$ F coincide. On the behalf of this basic reproductive number $\mathrm{R_0}$, we subsequently establish the following theorem regarding the global stability of the disease-free equilibrium (3.2).

**Theorem 4.1.** If $R_0 < 1$, the disease-free equilibrium (3.2) demonstrates global asymptotic stability. Conversely, the equilibrium becomes unstable when $R_0 > 1$.

**Proof.** Let us suppose the matrix

$$u = \left[1 \quad \frac{R_0(k_2 + \delta_2 + (1 - \delta_2)v\,v_i)}{\delta_1}\right]$$

here $k_2, \delta_2, v$ and $v_i$represent parameters as defined in Table 1. Notably, u is a $1 \times 2$ matrix consisting of positive real components. Verifying this entails a straightforward examination, confirming that

$$u\left(R_0 \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix} - V^{-1}F\right) = 0 \qquad \ldots(4.4)$$

Here $F$ and $V$ are defined in equations (4.1) and (4.2) respectively. Equation (4.4) suggests that

$$u\,R_0 = u\,V^{-1}\,F \qquad \ldots(4.5)$$

Now, consider

$$H = \begin{bmatrix} E \\ I \end{bmatrix}$$

We observe that $H$ represents a zero matrix solely at the disease-free equilibrium. By employing equation from (2.1) to (2.4), we have

$$\frac{dH}{dt} = \begin{bmatrix} \frac{dE}{dt} \\ \frac{dI}{dt} \end{bmatrix} = \begin{bmatrix} -(k_1 + \delta_1 + (1-\delta_1)\,v\,v_e) & \beta\,(1 - vv_s)\,S \\ \delta_1 & -(k_2 + \delta_2 + (1-\delta_2)\,v\,v_i) \end{bmatrix}$$

Thus, $\frac{dH}{dt} = [F - V]H.$

Now, we construct the Lyapunov function $L$ as

$$L = u\,V^{-1}H \qquad \ldots(4.6)$$

Considering that $u\,V^{-1}$ represents a $1 \times 2$ matrix comprised of positive real components, and $H$ is a non-negative matrix, it logically follows that $H \geq 0$ and furthermore, $H = 0$ if and only if $E = 0$ and $I = 0$. This suggests that $H$ is positive definite.

This indicates the positive definiteness of $R$.

$$\frac{dL}{dt} = u\,V^{-1}\frac{dH}{dt}$$

$$= u\,V^{-1}(\mathrm{F} - \mathrm{V})\mathrm{H}$$

$$= (uV^{-1}F - u)H$$

$$= u(R_0 - 1)H \qquad \ldots(4.7)$$

If the condition $R_0 < 1$ holds, the derivative of $H$ with respect to time is negative, indicating that the disease-free equilibrium (equation 6) is globally asymptotically stable. Conversely, if $R_0 < 1$ , the derivative of $H$ with respect to time is positive, suggesting instability of the equilibrium. It is noteworthy that when$R_0 = 1$ , we can infer local stability of the equilibrium as the derivative of $H$ with respect to time equals zero.

## 5. Numerical simulation

We conducted simulations of the equation from (2.1) to (2.4) under two scenarios: Case I (India) and Case II (Nepal), with initial conditions and parameters specified in Table 2. The simulations were performed using a Mathematica program, which approximates the model's solution using the fourth-order Runge-Kutta method (RK4).

**Table 2: Table of Description**

| Parameter | Case I/ Reference | Case II/ Reference |
|---|---|---|
| $S(0)$ | 0.97286 [28] | 0.994 [30] |
| $E(0) + I(0)$ | 0.00905 [29] | 3.813 [31] |
| $R(0)$ | 0.00665 [32] | 0.00554 [34] |
| $\beta$ | 0.53 [32] | 0.40 [33] |

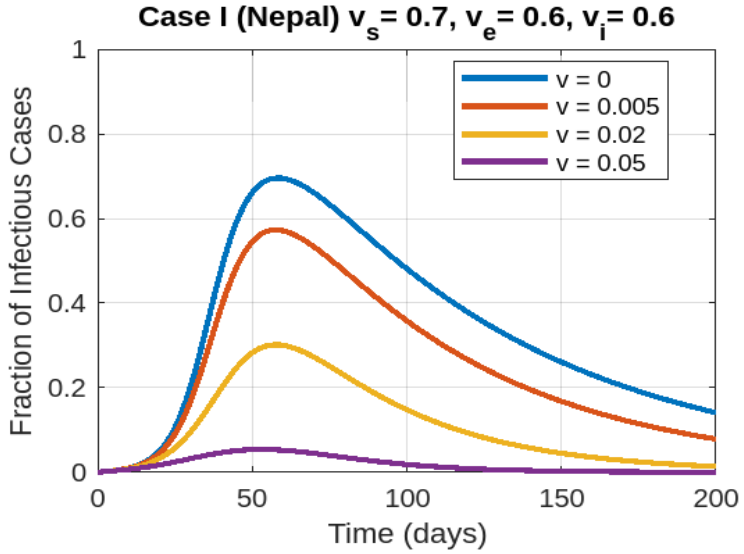
| | | |
|---|---|---|
| $\delta_1$ | 1/11.5 per day [33] | 1/11.5 per day [33] |
| $\delta_2$ | 0.0125 per day [34] | 0.0125 per day [34] |
| $A$ | $4.895 \times 10^{-5}$per day [29] | $4.589 \times 10^{-5}$per day [28] |
| $\mu$ | $1.708 \times 10^{-5}$ per day [30] | $2.04739 \times 10^{-5}$ per day [31] |
| $k_1$ | $1.735 \times 10^{-5}$ per day [32] | $2.04794 \times 10^{-5}$ per day [33] |
| $k_2$ | $1.735 \times 10^{-5}$ per day [32] | $2.04794 \times 10^{-5}$ per day [33] |

The maximum reproductive number, $R_0$, concerning the disease-free equilibrium (3.2), is observed when there is no vaccination $v = 0$.

Thus, $$R_0 = \sqrt{\frac{\delta_1 \beta A}{\mu (k_1 + \delta_1)(k_2 + \delta_2)}} \quad \ldots (5.1)$$

In Nepal, according to Table 2, the highest $R_0$ value for Case I is 1.035, and for Case II, it is 2.81. As the vaccination rate $v$ increases, the $R_0$ values decrease, reflecting the effectiveness of prophylactic $v_s$ and therapeutic $(v_e, v_i)$ vaccines, as depicted in Fig. 2. For instance, if we consider $v_s = 0.4$ it indicates 40% effectiveness of prophylactic vaccination when administered to susceptible individuals $S$. This implies that out of 100 people in the susceptible group who receive the prophylactic vaccine, 40 individuals are expected to recover.

Refer to Figure 2, as vaccination rates increase in both countries, the Nipah virus infection rate decreases. This underscores the crucial role of vaccination in controlling the Nipah virus outbreak. While vaccine efficacy is vital, it can significantly influence the risk of illness. Nipah virus is a zoonotic disease, transferring from animals to humans. During outbreaks, human infections with the Nipah virus have been more prevalent than animal infections. The virus might utilize both animals and humans as reservoirs for resurgence, akin to previous findings with other coronaviruses [35, 36]. Therefore, Nipah virus disease could be considered a re-emerging viral illness, meaning a disease previously observed within populations. To effectively manage and prevent Nipah virus infections, it's essential to explore strategies that boost vaccination rates. Achieving a high vaccination rate, aligned with vaccine potency, may require multiple vaccine doses. Furthermore, with the vaccine's efficacy rates of 70 % for prevention and 60 % for treatment, Nepal would require a higher vaccination rate than India to flatten the curve, as illustrated in Fig.2.



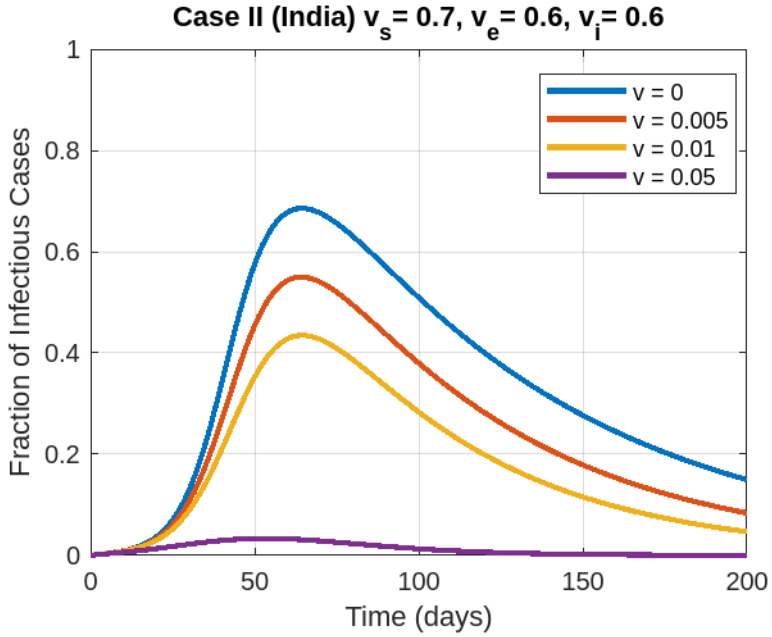



**Figure 2: Over time, we examined the proportion of infectious cases in Nepal and India under the assumption of 70% effectiveness for prophylactic vaccines and 60% for therapeutic vaccines. This was done across various vaccination rates, ranging from 0%(no vaccination), 0.1% per day etc for each population.**

The equilibrium points for Nepal's situation can be determined using Equation (3.4). Assuming a vaccination rate of 0.1%.

per day for the Nepal's population (v=0.001) and a vaccine efficiency of 90 % for both prophylactic and therapeutic vaccines, the equilibrium point corresponding to the fixed parameters in Table 2 of the Nepal case is $(S^m, E^m, I^m, R^m) = (0.000623, 0, 0, 2.865925)$. Without any vaccine, the equilibrium point for Nepal's case is $(S^m, E^m, I^m, R^m) = (0.000606, 0.006126, 0.038360, 2.910508)$, indicating that the disease will not eventually die out. In the long term, approximately 0.425 % of Nepal's population remains infectious.

For India's situation, with a vaccination rate of 0.1% With a vaccination rate of 0.1% per day and 90% vaccine efficiency, the equilibrium point is $(S^m, E^m, I^m, R^m) = (0.000014, 0, 0, 2.241390)$. Without vaccines, the equilibrium point is $(S^m, E^m, I^m, R^m) = (0.000014, 0.000672, 0.004133, 2.236598)$. Similarly to Nepal, a few percentages 0.62%of India's population remain infectious in the long term if no vaccine is available.

The difference in efficacy between prophylactic and therapeutic vaccines for Nipah virus treatment in humans is illustrated in Figure 3. Both vaccines were assumed to have the same effectiveness. The findings indicated that the prophylactic vaccine exhibits higher efficacy than the therapeutic vaccine in both Nepal and India. The prophylactic vaccine stimulates the immune system, generating long-lasting memory lymphocytes [37, 38]. As a result, the immune system can respond quickly to Nipah virus infection, leading to a decrease in the number of infected cases.

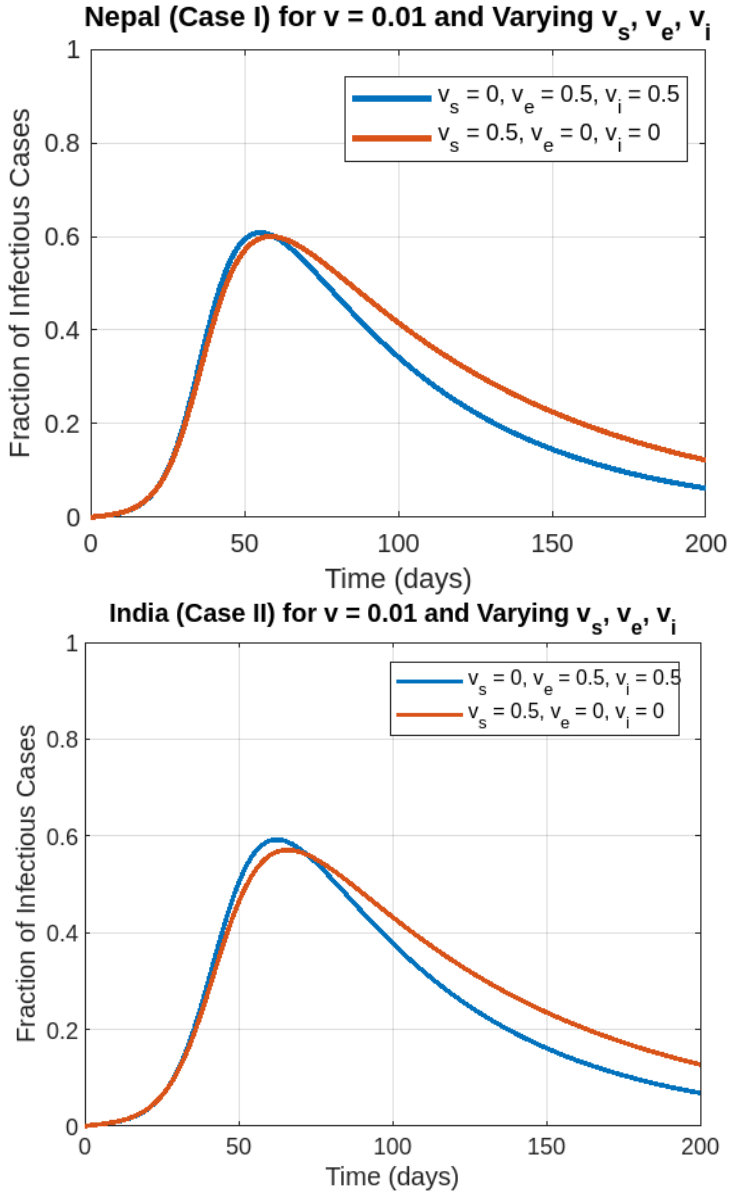



**Figure 3: Nepal and India: Cases where either prophylactic or therapeutic vaccines show equal efficiency.**

## 6. Conclusion

In conclusion, the SEIR model developed in this study provides significant contributions to understanding the future trajectory of Nipah virus control, especially in light of the emergence of the ChAdOx1 NipahB vaccine. Our simulations highlight the critical role of effective vaccination in mitigating the peak of the infectious population. However, it is essential to note that the introduction of a vaccine does not immediately halt the pandemic; rather, its effectiveness depends on various factors, including vaccine efficacy and the rate of vaccination roll out. Notably, our analysis indicates that Nepal would necessitate a higher daily vaccination rate compared to India, assuming equivalent vaccine efficacy, to achieve similar outcomes. By examining the model's formula presented in Section 2, we underscore the intricate interplay between vaccination rates and vaccine effectiveness in reducing the reproductive number $R_0$ , Theoretical insights confirm that when $R_0$ less than 1, control over the Nipah virus outbreak is attained, ensuring model stability. The equilibrium point of our

model, guided by specific parameters, anticipates a sustained decrease in Nipah virus prevalence over time, with only a marginal portion of the population remaining infectious in the absence of vaccination.

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