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## ***SEI*-MODEL FOR TRANSMISSION OF NIPAH VIRUS**

NITA H. SHAH\*, ANKUSH H. SUTHAR, FORAM A. THAKKAR AND MOKSHA H. SATIA

Department of Mathematics, Gujarat University, Ahmadabad-380009, Gujarat, India

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**Abstract:** Nipah virus (NiV) has drawn attention as an emerging infectious disease in Southeast Asia. It has become one of the most alarming threats of the public health mainly due to its periodic outbreaks and the high mortality rate. In the present work, the transmission of NiV using *SEI*-model is analysed through the system of non-linear differential equations. In addition, the reproduction number is attained which signifies the intensity of NiV outbreak. The local and global stability of equilibrium points is studied. Numerical simulation illustrates the behavior and flow of NiV infections in different compartments.

**Keywords:** mathematical model; reproduction number; local stability; global stability; simulation.

**2010 AMS Subject Classification:** 97M60.

### **1. Introduction**

Mathematical modeling has become an important tool for analysing the spread as well as control of infectious diseases. In recent years, epidemiological modeling of infectious disease transmission has had an increasing influence on the theory and practice of disease management

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\*Corresponding author

E-mail address: [nitahshah@gmail.com](mailto:nitahshah@gmail.com)

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and control. Epidemiology is the study of the distribution, determinants of health-related states or events in specified populations and the application of epidemiology is to control of health problems. The application of mathematical modeling to the spread of epidemics has a long history, which was been initiated by Bernoulli [3] work on the effect of cowpox inoculation on the spread of smallpox.

Nipah virus (NiV), belongs to the genus *Henipavirus*, a new class of virus in the Paramyxoviridae family, has drawn attention as an emerging zoonotic virus in southeast and south-Asian region. This emerging infectious disease has become one of the most alarming threats of the public health mainly due to its periodic outbreaks and the high mortality rate. NiV was been first noted in Malaysia in 1998 in pigs and pig farmers [7]. In 2001, NiV outbreaks have had been reported in Meherpurin Bangladesh and Siliguri in India. Of which, the highest mortality has occurred in Bangladesh, where the outbreak was typically noticed in winter season. In 2003–2005, the outbreak again appeared in Naogaon, Manikganj, Rajbari, Faridpur and Tangail districts [5]. In Bangladesh, the outbreaks were again been reported in subsequent years [9]. Recently, an outbreak has had been reported in the Kozhikode district of Kerala, India, wherein seventeen deaths were recorded, including one healthcare worker, which was declared to be officially ceased on June 10, 2018 [2]. The natural host of NiV is fruits bats [18]. Antibodies versus NiV have identified in fruit bats wherever they have tested including Cambodia, Thailand, India, Bangladesh and Madagascar [12], [16], [8], [9], [10]. Though NiV has caused a few outbreaks, it infects a wide range of animals and causes severe disease and death in people, making it a public health concern. Treatment is mostly symptomatic and supportive as the effect of antiviral drugs is not satisfactory. Therefore, the high mortality addresses the need for its control and mitigation. Hence, the present analysis deals with application of *SEI*-model for NiV transmission.

Wenzel have conducted a Markov Chain Monte Carlo simulation to estimate the unknown parameters of transmission of NiV [17]. Biswas have investigated the disease propagation and control strategy of NiV infections using *SIR* type mathematical model [4]. Sultana and Podar studies the optimal use of intervention strategies to mitigate the spread of NiV using optimal control technique [15]. Allen *et al.* review some mathematical models developed for the study of viral zoonoses in wildlife and identify areas [1].

In this paper, mathematical model for transmission of NiV is been formulated using system of non-linear ordinary differential equations in second section. Further, in third section, reproduction number and three equilibrium points are been derived from the system of differential equations. Local and global stability of the equilibrium points is been calculated in section four and analysis is completed by discussing numerical simulation.

## 2. Mathematical modeling

Here, we formulate a mathematical model of transmission of the NiV. Let the total population size of bats and humans at time  $t$  are denoted by  $N_B(t)$  and  $N_H(t)$  respectively. We divide total population of bats  $N_B(t)$  into two subclasses, susceptible bats  $S_B(t)$  and infected bats  $I_B(t)$ . Similarly, total population of humans  $N_H(t)$  is been divided in three subclasses, susceptible humans  $S_H(t)$ , exposed humans  $E_H(t)$  and infected humans  $I_H(t)$  and flow of NiV infection through these compartments is revealed in figure 1. Notations and parametric values used in this model are shown in table 2.1.

Table 2.1. Notations and parametric values used in the model

<i>Notations</i>	<i>Description</i>	<i>Parametric values</i>
$S_B$	Population size of susceptible bats	15
$I_B$	Population size of infected bats	12
$S_H$	Population size of susceptible humans	10
$E_H$	Population size of exposed humans	7
$I_H$	Population size of infected humans	5
$B_B$	Birth rate of bats	0.4
$B_H$	Birth rate of humans	0.2
$\beta_1$	Rate at which susceptible bats get infected	0.4
$\beta_2$	Rate at which susceptible individual turned into exposed individual.	0.4
$\beta_3$	Rate at which exposed individual is infected	0.3
$\beta_4$	Rate at which infected bats affect susceptible individuals	0.6
$\mu$	Disease escape rate	0.15
$\mu_d$	Mortality rate due to NiV infection	0.1

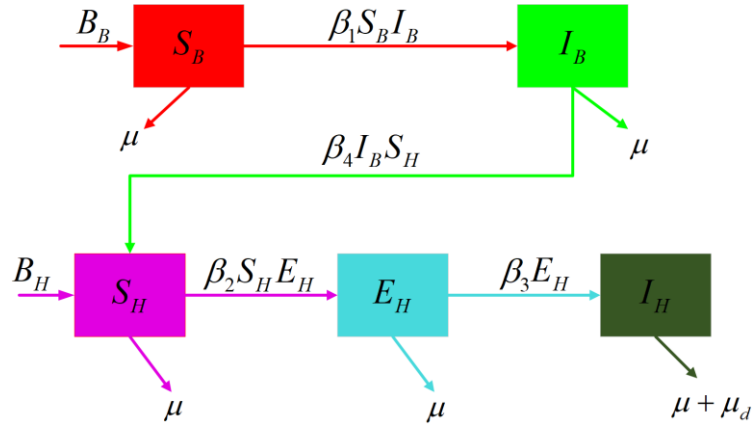


Figure 2.1. Transmission diagram for NiV infection

The system of non-linear ordinary differential equation given below describes the dynamics of NiV infection.

$$\begin{aligned}
 \frac{dS_B}{dt} &= B_B - \beta_1 S_B I_B - \mu S_B \\
 \frac{dI_B}{dt} &= \beta_1 S_B I_B - \beta_4 I_B S_H - \mu I_B \\
 \frac{dS_H}{dt} &= B_H + \beta_4 I_B S_H - \beta_2 S_H E_H - \mu S_H \\
 \frac{dE_H}{dt} &= \beta_2 S_H E_H - \beta_3 E_H - \mu E_H \\
 \frac{dI_H}{dt} &= \beta_3 E_H - \mu I_H - \mu_d I_H
 \end{aligned} \tag{1}$$

where  $N_H \geq S_H + E_H + I_H$  and  $N_B \geq S_B + I_B$ . Moreover,  $S_B, S_H > 0; I_B, E_H, I_H \geq 0$ .

Adding up all above differential equation of system (1), we obtain

$$\frac{d}{dt}(S_B + I_B + S_H + E_H + I_H) \leq B_B + B_H - \mu(N_B + N_H)$$

This implies that  $\limsup_{t \rightarrow \infty} (S_B + I_B + S_H + E_H + I_H) \leq \frac{B_B + B_H}{\mu}$

Thus, the feasible region for model (1) is:

$$\Gamma = \left\{ (S_B, I_B, S_H, E_H, I_H) : S_B + I_B + S_H + E_H + I_H \leq \frac{B_B + B_H}{\mu}, S_B > 0, I_B \geq 0, S_H > 0, E_H \geq 0, I_H \geq 0 \right\}$$

$\Gamma$  is positively invariant. *i.e.* every solution of model (1), with initial condition in  $\Gamma$  remains

there for all  $t > 0$ .

## 2.1. Reproduction number and equilibria

By solving system of non-linear differential equations (1), we get three equilibrium points:

i) *Infection free equilibrium*  $E_o = \left( \frac{B_B}{\mu}, 0, \frac{B_H}{\mu}, 0, 0 \right)$

ii) *Infectious bats free equilibrium*

$$E_1 = \left( \frac{B_B}{\mu}, 0, \frac{\beta_3 + \mu}{\beta_2}, \frac{B_H \beta_2 - \beta_3 \mu - \mu^2}{(\beta_3 + \mu) \beta_2}, \frac{\beta_3 (B_H \beta_2 - \beta_3 \mu - \mu^2)}{\beta_2 (\mu_d \beta_3 + \mu_d \mu + \beta_3 \mu + \mu^2)} \right)$$

iii) *Endemic equilibrium point*  $E^* = (S_B^*, I_B^*, S_H^*, E_H^*, I_H^*)$ , where

$$S_B^* = \frac{\beta_2 \mu + \beta_4 (\beta_3 + \mu)}{\beta_1 \beta_2}, \quad I_B^* = \frac{\beta_2 (B_B \beta_1 - \mu^2) - \beta_4 \mu (\beta_3 + \mu)}{\beta_1 (\beta_2 \mu + \beta_4 (\beta_3 + \mu))}, \quad S_H^* = \frac{\beta_3 + \mu}{\beta_2},$$

$$E_H^* = \frac{M}{\beta_1 \beta_2 ((\beta_3 + \mu) (\beta_2 \mu + \beta_3 \beta_4 + \beta_4 \mu))}, \quad \text{and} \quad I_H^* = \frac{\beta_3 M}{\beta_1 \beta_2 N}.$$

where,

$$M = (\beta_3 + \mu) (\beta_1 \beta_2 \beta_4 (B_B + B_H) - \beta_2 \mu^2 (\beta_1 + \beta_4) - (\beta_3 + \mu) (\beta_1 \beta_4 \mu + \beta_4^2 \mu)) \\ + B_H \beta_1 \beta_2^2 \mu,$$

$$N = (\beta_3 + \mu) ((\beta_3 + \mu) (\mu_d \beta_4 + \mu \beta_4) + \mu \beta_2 (\mu_d + \mu))$$

We will find the basic reproduction number  $R_0$  by the method of next generation matrix method

[6]. Let  $X = (I_B, S_H, E_H, I_H, S_B)$  then model (1) can be rewrite as  $X' = F(X) - V(X)$  such

that

$$F(X) = \begin{bmatrix} \beta_1 S_B I_B \\ \beta_4 I_B S_H \\ \beta_2 S_H E_H \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad V(X) = \begin{bmatrix} \beta_4 I_B S_H + \mu I_B \\ -B_H + \beta_2 I_H S_H + \mu S_H \\ \beta_3 E_H + \mu E_H \\ -\beta_3 E_H + \mu I_H + \mu_d I_H \\ -B_B + \beta_1 S_B I_B + \mu S_B \end{bmatrix}$$

By calculating the Jacobian matrices at  $E_0$ , we find that  $D(F(E_0)) = \begin{bmatrix} f & 0 \\ 0 & 0 \end{bmatrix}$  and

$$D(V(E_0)) = \begin{bmatrix} v & 0 \\ J_1 & J_2 \end{bmatrix} \text{ where}$$

$$f = \begin{bmatrix} \frac{\beta_1 B_B}{\mu} & 0 & 0 & 0 & 0 \\ \frac{\beta_4 B_H}{\mu} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_2 B_H}{\mu} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \text{ and } v = \begin{bmatrix} \frac{\beta_4 B_H}{\mu} + \mu & 0 & 0 & 0 & 0 \\ 0 & \mu & \frac{\beta_2 B_H}{\mu} & 0 & 0 \\ 0 & 0 & \beta_3 + \mu & 0 & 0 \\ 0 & 0 & -\beta_3 & \mu + \mu_d & 0 \\ \frac{\beta_1 B_B}{\mu} & 0 & 0 & 0 & \mu \end{bmatrix}$$

Thus, the next generation matrix is  $f v^{-1} = \begin{bmatrix} \frac{\beta_1 B_B}{B_H \beta_4 + \mu^2} & 0 & 0 & 0 & 0 \\ \frac{\beta_4 B_H}{B_H \beta_4 + \mu^2} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_2 B_H}{\mu(\beta_3 + \mu)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$  and the basic

reproduction number  $R_0$  is given by,

$$R_0 = \text{spectral radius}(\rho(f v^{-1})) = \frac{B_B \beta_1 \beta_3 \mu + B_B \beta_1 \mu^2 + B_H^2 \beta_2 \beta_4 + B_H \beta_2 \mu^2}{\mu(\beta_3 + \mu)(B_H \beta_4 + \mu^2)}.$$

### 3. Stability analysis

**Theorem 3.1.** (Local stability of  $E_0$ ) The infection free equilibrium point  $E_0$  is locally asymptotically stable if  $B_H \beta_2 < (\beta_3 + \mu)\mu$  and  $\beta_1 B_B < \beta_4 B_H + \mu^2$ .

**Proof.** Evaluating the Jacobian matrix for model (1) at point  $E_0$  gives

$$J(E_0) = \begin{bmatrix} -\mu & \frac{-\beta_1 B_B}{\mu} & 0 & 0 & 0 \\ 0 & \frac{\beta_1 B_B}{\mu} - \frac{\beta_4 B_H}{\mu} - \mu & 0 & 0 & 0 \\ 0 & \frac{\beta_4 B_H}{\mu} & -\mu & \frac{-\beta_2 B_H}{\mu} & 0 \\ 0 & 0 & 0 & \frac{\beta_2 B_H}{\mu} - \beta_3 - \mu & 0 \\ 0 & 0 & 0 & \beta_3 & -\mu - \mu_d \end{bmatrix}$$

Thus, the eigenvalues of  $J(E_0)$  are given by

$$\lambda_1 = -\mu, \lambda_2 = \frac{\beta_1 B_B}{\mu} - \frac{\beta_4 B_H}{\mu} - \mu, \lambda_3 = -\mu, \lambda_4 = \frac{\beta_2 B_H}{\mu} - \beta_3 - \mu, \text{ and } \lambda_5 = -\mu - \mu_d. \text{ Clearly, } \lambda_1,$$

$\lambda_3$  and  $\lambda_5$  are negative.

Also, if  $B_H \beta_2 < (\beta_3 + \mu)\mu$  then  $\lambda_4 < 0$ , and if  $\beta_1 B_B < \beta_4 B_H + \mu^2$  then  $\lambda_2 < 0$ .

Hence,  $E_0$  is locally asymptotically stable if  $B_H \beta_2 < (\beta_3 + \mu)\mu$  and  $\beta_1 B_B < \beta_4 B_H + \mu^2$ .

**Theorem 3.2.** (Local stability of  $E_1$ ) The infectious bats free equilibrium point  $E_1$  is locally asymptotically stable if  $\mu((\beta_3 + \mu)\beta_4 + \mu\beta_2) > \beta_1 \beta_2 B_B$  and  $B_H \beta_2 > \mu(\beta_3 + \mu)$ .

**Proof.** Evaluating the Jacobian matrix for system (1) for point  $E_1$ ,

$$J(E_1) = \begin{bmatrix} -\mu & \frac{-\beta_1 B_B}{\mu} & 0 & 0 & 0 \\ 0 & \frac{\beta_1 B_B}{\mu} - \frac{(\beta_3 + \mu)\beta_4}{\beta_2} - \mu & 0 & 0 & 0 \\ 0 & \frac{(\beta_3 + \mu)\beta_4}{\beta_2} & -\frac{B_H \beta_2 - \beta_3 \mu - \mu^2}{\beta_3 + \mu} - \mu & -\beta_3 - \mu & 0 \\ 0 & 0 & \frac{B_H \beta_2 - \beta_3 \mu - \mu^2}{\beta_3 + \mu} & 0 & 0 \\ 0 & 0 & 0 & \beta_3 & -\mu - \mu_d \end{bmatrix}$$



Clearly,  $J(E_1)$  have two negative eigenvalues, say  $\lambda_1 = -\mu$  and  $\lambda_2 = -\mu - \mu_d$ , and remaining eigenvalues  $\lambda_3$ ,  $\lambda_4$  and  $\lambda_5$  are given by the polynomial,

$$\lambda^3 + (x_2 + x_1)\lambda^2 + (x_1x_2 + x_3x_4)\lambda + x_4x_3x_1 = 0$$

Where,

$$x_1 = -\frac{\beta_1 B_B}{\mu} + \frac{(\beta_3 + \mu)\beta_4}{\beta_2} + \mu, \quad x_2 = \frac{B_H\beta_2 - \beta_3\mu - \mu^2}{\beta_3 + \mu} + \mu, \quad x_3 = \beta_3 + \mu \quad \text{and} \quad x_4 = \frac{B_H\beta_2 - \beta_3\mu - \mu^2}{\beta_3 + \mu}$$

. Hence,  $x_2 + x_1$ ,  $x_1x_2 + x_3x_4$  and  $x_4x_3x_1$  are positive if  $\mu((\beta_3 + \mu)\beta_4 + \mu\beta_2) > \beta_1\beta_2B_B$  and  $B_H\beta_2 > \mu(\beta_3 + \mu)$ . Which implies, the equilibrium point  $E_1$  is locally asymptotically stable under these conditions.

**Theorem 3.3.** (Local stability of  $E^*$ ) The endemic equilibrium point  $E^*$  is globally asymptotically stable if

$$\beta_1\beta_4(B_B + B_H) - \mu^2(\beta_1 + \beta_4) + \frac{B_H\beta_1\beta_2\mu}{(\beta_3 + \mu)} > \beta_2\beta_4(B_B\beta_1 - \mu^2) + \frac{(\beta_3 + \mu)\mu}{\beta_2}(\beta_1\beta_2 + \beta_4^2 - \beta_2\beta_4^2).$$

**Proof.** Evaluating the Jacobian matrix for system (1) for point  $E^*$ ,

$$J(E^*) = \begin{bmatrix} -x_5 & \beta_1 S_B^* & 0 & 0 & 0 \\ \beta_1 I_B^* & -x_6 & -\beta_4 I_B^* & 0 & 0 \\ 0 & \beta_4 S_H^* & -x_7 & -\beta_2 S_H^* & 0 \\ 0 & 0 & \beta_2 E_H^* & -x_8 & 0 \\ 0 & 0 & 0 & \beta_3 & -x_9 \end{bmatrix}$$

where,

$$x_5 = \beta_1 I_B^* + \mu, \quad x_6 = -\beta_1 S_B^* + \beta_4 S_H^* + \mu, \quad x_7 = -\beta_4 I_B^* + \beta_2 E_H^* + \mu, \quad x_8 = -\beta_2 S_H^* + \beta_3 + \mu, \quad x_9 = \mu + \mu_d.$$

For given the Jacobian matrix  $J(E^*)$ , the characteristic polynomial is defined as:

$$Ch_{E^*}(\lambda) = \lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5$$

where,

$$a_1 = x_5 + x_6 + x_7 + x_8 + x_9,$$

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$$\begin{aligned}
a_2 &= E_H^* S_H^* \beta_2^2 + I_B^* (S_B^* \beta_1^2 + S_H^* \beta_4^2) + (x_5 + x_9)(x_6 + x_7 + x_8) + x_5 x_9 + x_6 x_7 + x_6 x_8 + x_7 x_8, \\
a_3 &= (x_7 + x_8 + x_9)(I_B^* S_H^* \beta_1^2 + x_5 x_6) + (x_5 + x_6 + x_9)(E_H^* S_H^* \beta_2^2 + x_7 x_8) \\
&\quad + (x_5 + x_8 + x_9) I_B^* S_H^* \beta_4^2 + x_9 (x_7 + x_8)(x_5 + x_6), \\
a_4 &= E_H^* I_B^* S_B^* S_H^* \beta_1^2 \beta_2^2 + E_H^* S_H^* \beta_2^2 (x_5 x_6 + x_5 x_9 + x_6 x_9) + (x_7 x_8 + x_7 x_9 + x_8 x_9)(I_B^* S_H^* \beta_1^2 + x_5 x_6) \\
&\quad + I_B^* S_H^* \beta_4^2 (x_5 x_8 + x_5 x_9 + x_8 x_9) + x_7 x_8 x_9 (x_5 + x_6), \\
\text{and } a_5 &= E_H^* I_B^* S_B^* S_H^* \beta_1^2 \beta_2^2 + E_H^* S_H^* \beta_2^2 x_5 x_6 + I_B^* S_B^* \beta_1^2 x_7 x_8 + I_B^* S_H^* \beta_4^2 x_5 x_8 + x_5 x_6 x_7 x_8.
\end{aligned}$$

All the co-efficient of characteristic polynomial  $Ch_{E^*}(\lambda)$  are positive if  $x_i \geq 0$ , for  $i = 1, 2, 3, 4, 5$ .

Clearly,  $x_5, x_9 \geq 0$ . Moreover  $x_6, x_8$  vanishes under point  $E^*$  and  $x_7$  is positive if

$$\beta_1 \beta_4 (B_B + B_H) - \mu^2 (\beta_1 + \beta_4) + \frac{B_H \beta_1 \beta_2 \mu}{(\beta_3 + \mu)} > \beta_2 \beta_4 (B_B \beta_1 - \mu^2) + \frac{(\beta_3 + \mu) \mu}{\beta_2} (\beta_1 \beta_2 + \beta_4^2 - \beta_2 \beta_4^2).$$

Hence, equilibrium point  $E^*$  is locally asymptotically stable under this condition.

**Theorem 3.4.** (Global stability of  $E_0$ ) The infection free equilibrium point  $E_0$  is global asymptotically stable in  $\Gamma$ .

**Proof.** Consider the Lyapunov function  $L(t) = I_B + E_H + I_H + S_H$

$$\dot{L} = B_B - \mu S_B + B_H - \mu(S_H + I_B + I_H + E_H) - \mu_d I_H$$

Since,  $E_0$  belongs to the feasible region  $\Gamma$ ,  $S_H$  and  $S_B$  are bounded above by  $\frac{B_H}{\mu}$  and  $\frac{B_B}{\mu}$

respectively, this implies  $\frac{dL}{dt} \leq 0$ . Moreover  $\frac{dL}{dt} = 0$  only if  $E_H = 0$  and  $I_H = 0$ . Therefore,

the only trajectory of the system on which  $\frac{dL}{dt} = 0$  is  $E_0$ .

Hence by LaSalle's Invariant Principle (LaSalle, 1976),  $E_0$  is globally asymptotically stable.

**Theorem 3.5.** (Global stability of  $E_1$ ) The infectious bats free equilibrium point  $E_1$  is globally stable in  $\Gamma$  if  $\beta_1 \beta_2 B_B < \beta_4 (\beta_3 + \mu) \mu + \beta_2 \mu^2$ .

**Proof.** Consider the Lyapunov function  $L(t) = I_B + E_H$ ,

$$\begin{aligned} \frac{dL}{dt} &= \beta_1 S_B I_B - \beta_4 I_B S_H - \mu I_B + \beta_2 S_H E_H - \beta_3 E_H - \mu E_H \\ &= (\beta_1 S_B - \beta_4 S_H - \mu) I_B + (\beta_2 S_H - \beta_3 - \mu) E_H \end{aligned}$$

Hence,  $\frac{dL}{dt} < 0$  if  $\beta_1 \beta_2 B_B < \beta_4 (\beta_3 + \mu) \mu + \beta_2 \mu^2$  with  $\frac{dL}{dt} = 0$  if and only if  $I_B = 0$  and

$$E_H = 0.$$

Hence, the only solution of system (1) in  $\Gamma$  on which  $\frac{dL}{dt} = 0$  is  $E_1$ .

Using LaSalle's Invariance principle, it is clear that every solution of the system (1), with initial conditions in  $\Gamma$  approaches to  $E_1$  as  $t \rightarrow \infty$ . Hence,  $E_1$  is globally asymptotically stable.

**Theorem 3.6.** (*Global stability of  $E^*$* ) The endemic equilibrium point  $E^*$  is globally stable.

**Proof.** Consider the Lyapunov function:

$$\begin{aligned} L(t) &= \frac{1}{2} \left[ (S_B(t) - S_B^*) + (I_B(t) - I_B^*) + (S_H(t) - S_H^*) + (E_H(t) - E_H^*) + (I_H(t) - I_H^*) \right]^2 \\ \therefore \frac{dL}{dt} &= \left[ (S_B - S_B^*) + (I_B - I_B^*) + (S_H - S_H^*) + (E_H - E_H^*) + (I_H - I_H^*) \right] \left[ S_B' + I_B' + S_H' + E_H' + I_H' \right] \\ &= \left[ (S_B - S_B^*) + (I_B - I_B^*) + (S_H - S_H^*) + (E_H - E_H^*) + (I_H - I_H^*) \right] \\ &\quad \left[ B_B - \mu S_B - \mu I_B - B_H - \mu S_H - \mu E_H - \mu I_H - \mu_d I_H \right] \end{aligned}$$

By putting  $B_B = \mu S_B^* + \mu I_B^*$  and  $B_H = \mu S_H^* + \mu E_H^* + \mu I_H^*$ , we get

$$\frac{dL}{dt} = -\mu \left[ (S_B - S_B^*) + (I_B - I_B^*) + (S_H - S_H^*) + (E_H - E_H^*) + (I_H - I_H^*) \right]^2 \leq 0$$

Hence,  $E^*$  is globally stable.

#### 4. Numerical simulation

The changes in different compartments under influence of other compartments and different parameters can be analysed through numerical simulations.

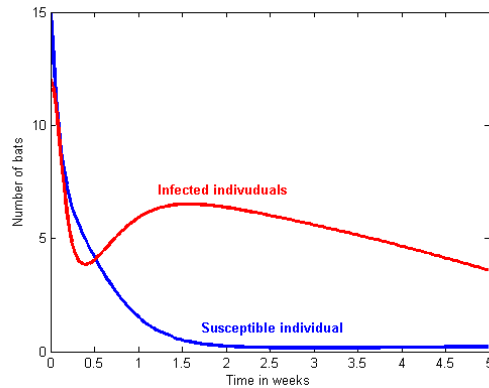


Figure 4.1. Motion of bats in compartments

Figure 4.1. demonstrates the change in compartments of susceptible bats and infected bats with respect to time. It can observe from the above figure, that population of susceptible bats decreases continuously and become negligible in two weeks. And in three days, the population of infected bats decreases up to almost 70% after which it gradually increases.

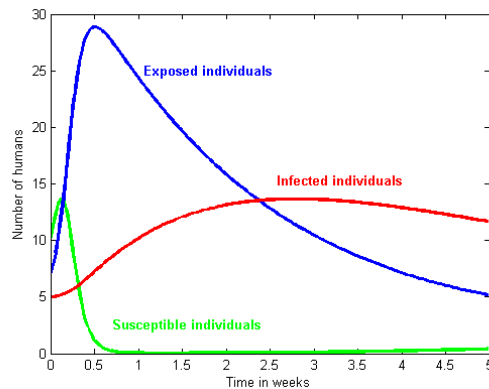


Figure 4.2. Motion of humans in compartment

Figure 4.2. depicts that population of susceptible humans' increase to some extent and then gets vanished in one week, after four weeks slight improvement in the class is observed. Highly improvement in exposed class is observed during first week, later it decreases gradually. Improvement in infected individuals is observed during NiV explosion for the parametric values

given in table2.1.

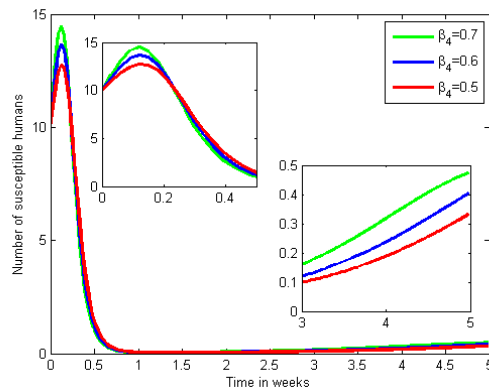


Figure 4.3. Impact on susceptible individuals due change in  $\beta_4$

Figure 4.3. indicates that the change in susceptible individuals with respect to time for three different values of  $\beta_4$  (Rate at which infected bats affect susceptible individuals). It is clearly noticeable that  $\beta_4$  is directly proportional to number of susceptible individuals. For all three values of  $\beta_4$ , it is observed that initially class of susceptible individuals get vanished together in one week and after three weeks considerable improvement is observed for all given values of  $\beta_4$ .

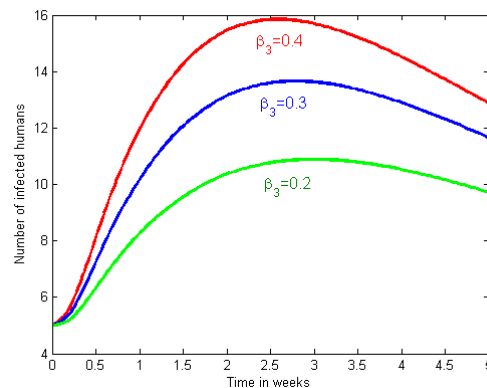


Figure 4.4. Impact of infected individual due to change in  $\beta_3$

Figure 4.4. shows the graph of change in class of infected humans with time for different values of  $\beta_3$  (Rate at which exposed individual is infected). One can observe that number of infected

humans increases as we increase value of  $\beta_3$ .

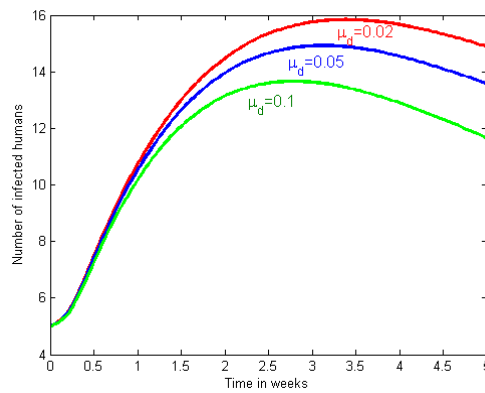


Figure 4.5. Impact on infected individual due to change in  $\mu_d$

Figure 4.5. illustrate that if infected individuals increase by 27.67%, mortality rate due to NiV infection will increase by 8%.

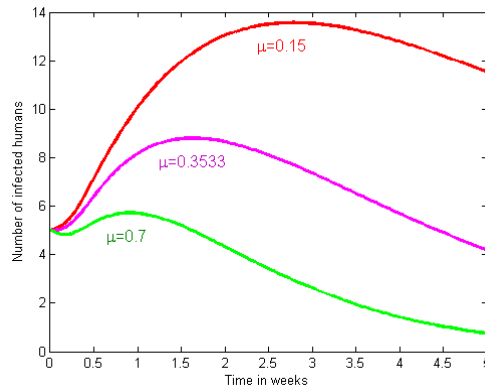


Figure 4.6. Impact on infected individual due to change in  $\mu$

In figure 4.6., graph of infected individuals is been plotted for three different values of  $\mu$  (disease escape rate). Note that,  $R_0 \approx 1$  for  $\mu = 0.3533$ , in figure-7 we can see the class of infected individual get controlled in 5-6 weeks for  $\mu > 0.3533$  and it get increased for  $\mu < 0.3533$ .

## 5. Discussion and conclusion

In the present work, a mathematical model, *SEI*-model, is used to examine the transmission of NiV in populations of bats and humans. Further, the reproduction number is attained which signifies the intensity of NiV outbreaks. Numerical simulations have illustrated the behaviour and flow of NiV infections in different compartments, which shows that how population of the infected bats can affect the susceptible and infected individuals. If escape rate is greater than 35.33% then only the system remains disease free, otherwise disease turn out to be endemic. Since the proper vaccination is not yet available, this information could be further useful in control and mitigation of NiV outbreak.

### Conflict of Interests

The authors declare that there is no conflict of interests.

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