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The dynamics of Nipah virus (NiV) transmission and analysis

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Abstract

We propose a model for NiV infection mechanisms from pigs to humans and humans to humans, with a focus on the impact of a combination vaccine, and condom as a control measure. In a biologically realistic setting, we derived the basic characteristics of our suggested model, such as boundedness and positivity. We determined the basic reproduction number to investigate both the local and global behavior of the model's various equilibria. When the reproductive number is less than one, the disease-free state of Nipah virus is locally asymptotically stable, but unstable when it is higher than one. We established that the endemic equilibrium is locally asymptotically stable near unity using central manifold theory. Nipah virus free equilibrium is stable on the global stability scale, and endemic equilibrium is asymptotically stable. We determine that vaccination and condom are effective ways for reducing Nipah virus spread. In addition, Nipah virus carriers (asymptomatic) are identified as the most infectious individuals who should be targeted by the model. Finally, numerical simulations are used to verify the efficacy of the provided findings.

Keywords: Nipah virus, vaccination, condom, isolation, asymptomatic.

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1. Nipah virus (NiV) introduction

NiV is a highly communicable zoonotic viral infection that could potentially cause disease in people and animals, especially pigs. The virus was discovered during an outbreak in Malaysia as well as Singapore in 1999, where it caused significant lung infections in pigs and encephalitis (brain inflammation) in people [3].

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The Nipah virus's natural host is fruit bats, also widely recognized as flying foxes. Infection from infected bats, polluted food or water, or intimate touch with sick pigs or people can all pass on the illness to humans. Human-to-human spread is also possible, particularly in hospital environments where infection prevention and management methods are lacking. Nipah virus exposure can cause symptoms ranging from minor flu-like symptoms to serious neurological illness, including seizures, coma, and mortality.

The Nipah virus is an enclosed, newly, negative-sense Ribonucleic acid virus that creates the novel genus Henipavirus in the paramyxoviridae group. Infection with Nipah virus is also known as NiV encephalitis [20]. Hendra and Cedar virus are intimately associated with NiV [19]. They are indeed the three species representatives of the genus Henipavirus, a novel viral infection subclass in the Paramyxoviridae group. One of the most significant bat-borne infections recently found is the Nipah Virus (NiV).

It was previously believed to be a variation of Japanese Encephalitis (JE), but was subsequently discovered to be a novel zoonotic illness and given the name Nipah in honor of the town of "Sungai Nipah," where it was discovered [14]. After a fever epidemic among swine producers and importers in Malaysia and Singapore in 1999, Doctor Chua discovered first Nipah virus, which led to the downfall of the worth billions of dollars pig export economy [4]. Cerebrospinal fluid samples from three deadly fever cases were used to inoculate vero cells, which then produced syncytia [17]. The virus was studied using electron microscopy (EM), which showed features of a virus from the virus family that does not contain the Japanese encephalitis virus [31].

Immunofluorescence antibody assays revealed that Nipah disease cells reacted strongly to Hendra virus monoclonal antibody but not to specific antibodies for other retroviruses such as measles virus, syncytial virus of the respiratory tract, parainfluenzaviruses 1 and 3, herpesvirus, enteroviruses, or JE virus [15]. According to cross-neutralization experiments, the Nipah and Hendra viruses' neutralizing antibodies differed by 8 to 16 times, suggesting that despite their close relationship, the viruses were not the same [15]. Compared to other family members such as the flu and fever viruses, which typically have a restricted host area and genetic stability with a nearly identical genome area common to every family [19], henipaviruses are distinguished from the paramyxoviruses by a larger genome. They also have a wider host range than other members of the family [22].

Like other animal paramyxoviruses, NiV is rendered inactive after 60 minutes at 60°C. Within the range of pH 4.0 and 10.0, it is stable. The Nipah virus may last in mango fruit or certain fruit juices for up to three days, and it can endure not less than seven days in synthetic date palm sap. In fruit bat urine, the virus has an 18-hour half-life [17].

The NiV epidemic in Malaysia between 1998 and 1999 was brought on after pigs consumed partially eaten fruits from the bats [23]. Due to the importation of pigs from Malaysia, the infection also reached Singapore's pig handlers [23]. Comparing the Nipah virus in Malaysia and Bangladesh, no amplification host was required in Bangladesh, unlike in Malaysia where the epidemic had spread from bats to amplification host swine and then to persons [18].

The pathogenesis and ecology of the Nipah virus infection have been investigated by many researchers, yet there are not many models that are accessible for it, and they are as follows: Nipah fever in Bangladesh, have a mathematical model that was proposed [10] to comprehend the outbreak of the Nipah virus pandemic. Sultana et al [30] created a dynamic Nipah virus model with two distinct control methods and populations of various sizes, in which increasing consciousness and getting therapy are viewed as controls. A mathematical model with vital dynamics for stopping the spread of Nipah illness has been suggested and investigated [24]. This model includes quarantines of infectious people, which are influenced by the accessibility of isolation facilities and surveillance coverage. Using the SEI model, Shah et al. [29] proposed a collection of non-linear differential equations for the transmission of NiV. The traditional differential model, according to Agarwal et al. [5], offers an explanation centered on the Markovian approach in which the development equation has no memory, which is clearly incorrect for the true situation. They substituted the local time differential operator with a Mittag-Leffler function differential operator. According to Zewdie et al. [34], the Nipah virus is likely to propagate through

unprotected touch with the deceased bodies of infected individuals. They put out the SIRD model and looked at the mechanisms of Nipah virus transmission and the effects of open contact with infected people's dead bodies prior to burial or cremation as well as their disposal rate. Omede et al. [25] developed and examined mathematical model to investigate the qualitative and quantitative aspects of the Nipah virus spread patterns. To find out how the controls affected the spread of the illness, they looked at different combination tactics. Through the quantitative outcomes, they discovered that the most effective control methods greatly lessen the impact of the diseases.

The primary goal of this study is to investigate the mechanisms of NiV infection from pigs to persons and from persons to persons, with a focus on the impact of a combination vaccine, and condom as a control measure. This paper is arranged in the following form. In Section 2 we have Nipah virus model formulation and procedure. In Section 3, analysis of Nipah virus infectious model is presented. Section 4 presents numerical solutions. In Section 5 we have discussion and conclusion.

2. Nipah virus model formulation and procedure

There are seventeen compartments in the model that was developed with the population being studied. They are $S_p(t)$: susceptible pigs, $E_p(t)$: exposed pigs, $I_p(t)$: infectious pigs, $S(t)$: susceptible human population, $S_v(t)$: susceptible vaccinated population, $S_u(t)$: susceptible non-vaccinated population, $S_{vc}(t)$: susceptible vaccinated condom users population, $S_{vn}(t)$: susceptible vaccinated non-condom users population, $S_{uc}(t)$: Susceptible non-vaccinated condom users population, $S_{un}(t)$: susceptible non-vaccinated non-condom users, $E(t)$: exposed human, $I(t)$: infectious human population, $C(t)$: NiV carrier human population, $I_i(t)$: infectious isolated undergoing treatment human population, $I_t(t)$: infectious undergoing treatment human population, $R(t)$: recovered individuals, $D(t)$: dead bodies of infectious individuals.

$E(t)$ represents the amount of people who have been exposed to the NiV but are not yet contagious, i.e., pre-symptomatic. $I(t)$ indicates the amount of NiV-infected individuals who have the ability of passing on the illness to those who are susceptible. $C(t)$ denotes the number of people who have had NiV, continue to infect others and show no symptom, i.e., asymptomatic. $R(t)$ represents the amount of people who have survived from NiV through therapy (Ribavirin) or on their own. $D(t)$ is the quantity of unprotected deceased corpses of infectious people.

The first compartment in (2.1), shows the dynamics of susceptible human. Newly susceptible individuals are recruited at the level Λ with those who became susceptible after recover for some time at the rate ε, vaccinated at the rate χ_2 and dies normally at rate μ. The susceptible non-vaccinated population is further classified as susceptible non-vaccinated condom users and susceptible non-vaccinated non-condom users at the properties η_1 and η_2, respectively with natural death rate μ while susceptible vaccinated compartment is classified as susceptible vaccinated condom users and susceptible vaccinated non-condom users at the properties τ_1, τ_2, respectively and the natural death rate μ. We consider the parameters a_1, a_2, a_3, a_4, a_5 to be the percentage of successful contact of susceptible vaccinated condom users with infectious pigs, Nipah virus human carriers, infectious individuals, infectious undergoing treatment individuals and unprotected dead bodies, respectively, and the transmission probability β_1. Furthermore, the parameters b_1, b_2, b_3, b_4, b_5 are the contact rate of susceptible vaccinated non-condom users with infectious pigs, Nipah virus human carriers, infectious individuals, infectious undergoing treatment individuals and unprotected dead bodies, respectively, and the transmission probability β_2. Again the parameters q_1, q_2, q_3, q_4, q_5 are the contact rate of susceptible unvaccinated condom users with the infectious pigs, Nipah virus human carriers, infectious individuals, infectious undergoing treatment individuals and unprotected dead bodies, respectively, and the transmission probability β_3. The parameters z_1, z_2, z_3, z_4, z_5 are the contact rate of susceptible non-vaccinated non-condom users with the infectious pigs, Nipah virus human carries, infectious individuals, infectious undergoing treatment individuals and unprotected dead bodies, respectively, and the transmission probability β_4. As a result, the infection forces indicate the efficient risk of transmission with respect to susceptible vaccinated

condom users, susceptible vaccinated non-condom users, susceptible non-vaccinated condom users and susceptible non-vaccinated non-condom users are

$$\Gamma_1 = \beta_1 \left(\frac{a_1 I_p}{N_p} + \frac{a_2 C + a_3 I + a_4 I_t + a_5 D}{N} \right), \quad \Gamma_2 = \beta_2 \left(\frac{b_1 I_p}{N_p} + \frac{b_2 C + b_3 I + b_4 I_t + b_5 D}{N} \right),$$
$$\Gamma_3 = \beta_3 \left(\frac{q_1 I_p}{N_p} + \frac{q_2 C + q_3 I + q_4 I_t + q_5 D}{N} \right), \quad \Gamma_4 = \beta_4 \left(\frac{z_1 I_p}{N_p} + \frac{z_2 C + z_3 I + z_4 I_t + z_5 D}{N} \right),$$

respectively, where N is number of human beings and N_p is number of pigs. Therefore a_i, b_i, q_i, z_i, $i = 1, 2, 3, 4, 5$ are contact rates.

The class of exposed people becomes successfully infectious and Nipah virus carriers at the rate κ and θ, respectively per unit time, and dies naturally at the rate μ. The exposed class increases in the level of infections by $\Gamma_3, \Gamma_4, \Gamma_1$ and Γ_2 rate from susceptible unvaccinated condom users, susceptible unvaccinated non-condom users, susceptible vaccinated condom users, and susceptible vaccinated non-condom users, respectively.

The infectious class group is produced by exposed people who progressed to the infectious class at the rate κ. The infectious undergoing treatment class reduces the population at a rate of ψ_2, infectious isolated undertaking treatment class at a rate of ψ_1, recovery rate γ_4, disease-induced mortality at a rate of δ_2, and natural death rate μ.

The Nipah virus carrier class population is derived from exposed people who advanced to the Nipah virus carrier class at some degree θ. The population diminishes at the rate of γ_3 recovered people, δ_1 disease-induced mortality, and μ natural death.

The infectious isolated but undergoing treatment class population is produced by infected people who advanced to the infectious isolated undertaking treatment class at a ratio of ψ_1. Individuals who recovered at a rate of γ_2, disease-induced mortality at a rate of δ_3, and normal death at a rate of μ reduced the population.

The infectious undergoing treatment class population is produced by infectious people progressing to the infectious undergoing treatment class at a rate of ψ_2. Individuals who recovered at a rate of γ_1, disease-induced mortality at a rate of δ_4, and natural death at a rate of μ all contribute to the population decline.

The recovered compartment is formed by the Nipah virus carriers class, infectious isolated undergoing treatment class, infectious undergoing treatment class, and infectious individuals who recovered from Nipah at the rates of γ_3, γ_2, γ_1, and γ_4, respectively, and dies naturally at the rate of μ.

This model incorporated dead body compartment. Contact with dead bodies of infectious individuals can expose susceptible people to Nipah virus. The dead bodies compartment is generated from Nipah virus carriers, infectious individuals, infectious isolated and infectious undergoing treatment class at the rates δ_1, δ_2, δ_3 and δ_4, respectively, and finally disposed or buried at the rate μ_d.

The susceptible pigs are recruited at the level Λ_p, exposed to the Nipah virus at σ, and perish naturally at μ_p. The exposed pigs advance to the infectious class at a rate of ρ and die of natural causes at a rate of μp, whereas the infectious class dies due to the virus at a rate of δ_p.

2.1. Nipah virus model assumptions

We assume the following.

- Natural healing from illness may occur as a result of potent antibodies [25].
- Individuals are exposed to the virus through unprotected contact with the dead corpse of a NiV-infected person [34].
- The vulnerable human population interacts with sick and contagious pigs that are carrying the NiV virus.

- Isolated people do not contribute to NiV transmission progress because they are under careful monitoring and health workers shield themselves from the virus, and infection can occur in the therapy class [25].

- Condoms, an isolation facility for the contagious, and the vaccinations are widely affordable and easily available to the entire population.

- After some time, those who have healed are once again susceptible to the infection.

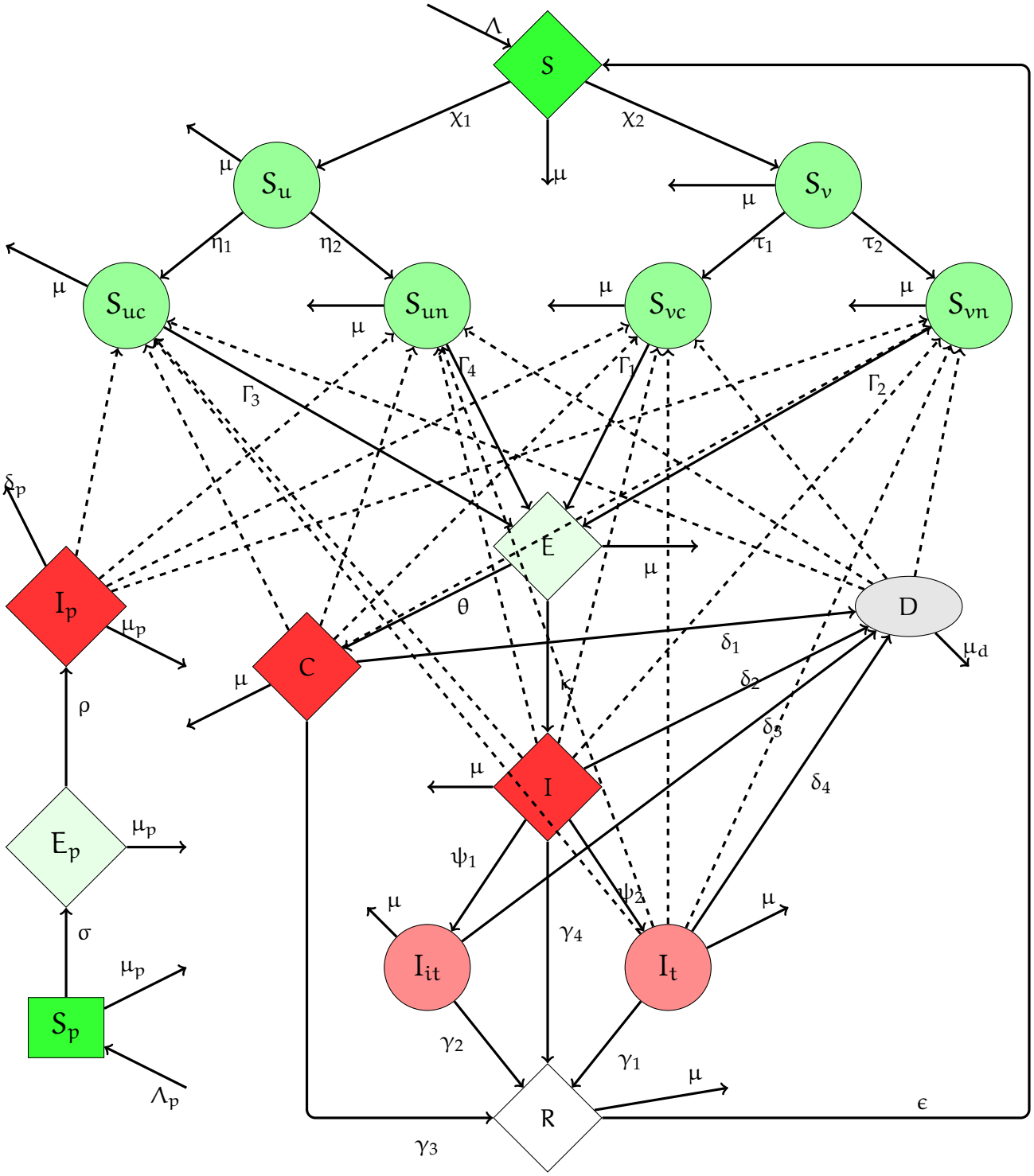


Table 1: Variables description.

Parameters	Parameter description
Λ_p	Number of newly introduced pigs
σ	Pigs susceptible rate of exposure
ρ	Rate at which exposed pigs become infectious
Λ	Level of recruitment for human
χ_1	Non-vaccination vulnerable population rate
χ_2	Vaccination rate of susceptible populations
η_1	The proportion of susceptible unvaccinated people using condoms
η_2	The proportion of susceptible unvaccinated people not using condoms.
τ_1	The proportion of susceptible vaccinated people using condoms
τ_2	The proportion of susceptible vaccinated people who do not use condoms
Γ_3	Force of infection on S_{nc}
Γ_4	Force of infection on S_{un}
Γ_1	Force of infection on S_{vc}
Γ_2	Force of infection on S_{vn}
κ	Exposed population's rate of becoming infectious
θ	Exposed population's rate of becoming NiV carriers
ψ_1	Rate of isolation undergoing treatment of the infectious people
ψ_2	Rate of treatment of the infectious people
γ_1	Rate of recovery from the infectious undergoing treatment class
γ_2	Recovery rate from the infectious separated undergoing treatment class
γ_3	Recovery rate from the NiV carrier people
γ_4	Recovery rate from the infectious people
ϵ	Rate of susceptible from the recovered individuals
δ_1	Death Rate from Illness in the NiV-Carriers group
δ_2	Death Rate from Illness in the infectious population
δ_3	Death Rate from Illness in the infectious isolated people undergoing treatment
δ_4	Death Rate from Illness in the infectious people undergoing treatment
δ_d	Death Rate from the infectious pigs
μ_d	Rate of disposal of deceased corpses (burial/cremation)
μ_p	Normal death rate of pigs
μ	Natural death rate

Given the aforementioned formulations and presumptions, the Nipah virus's evolving processes are represented by the deterministic system of ordinary differential equations shown below.

$$
\begin{cases}
\frac{dS}{dt} = \Lambda - (\chi_1 + \chi_2 + \mu)S + \epsilon R, \\
\frac{dS_v}{dt} = \chi_2 S - (\tau_1 + \tau_2 + \mu)S_v, \\
\frac{dS_u}{dt} = \chi_1 S - (\eta_1 + \eta_2 + \mu)S_u, \\
\frac{dS_{vc}}{dt} = \tau_1 S_v - (\beta_1 \left(\frac{a_1 I_p}{N_p} + \frac{a_2 C + a_3 I + a_4 I_t + a_5 D}{N}\right) + \mu)S_{vc}, \\
\frac{dS_{vn}}{dt} = \tau_2 S_v - (\beta_2 \left(\frac{b_1 I_p}{N_p} + \frac{b_2 C + b_3 I + b_4 I_t + b_5 D}{N}\right) + \mu)S_{vn}, \\
\frac{dS_{uc}}{dt} = \eta_1 S_u - (\beta_3 \left(\frac{q_1 I_p}{N_p} + \frac{q_2 C + q_3 I + q_4 I_t + q_5 D}{N}\right) + \mu)S_{uc}, \\
\frac{dS_{un}}{dt} = \eta_2 S_u - (\beta_4 \left(\frac{z_1 I_p}{N_p} + \frac{z_2 C + z_3 I + z_4 I_t + z_5 D}{N}\right) + \mu)S_{un}, \\
\frac{dE}{dt} = \beta_2 \left(\frac{b_1 I_p}{N_p} + \frac{b_2 C + b_3 I + b_4 I_t + b_5 D}{N}\right) S_{vn} \\
\qquad + \beta_4 \left(\frac{z_1 I_p}{N_p} + \frac{z_2 C + z_3 I + z_4 I_t + z_5 D}{N}\right) S_{un} + \beta_3 \left(\frac{q_1 I_p}{N_p} + \frac{q_2 C + q_3 I + q_4 I_t + q_5 D}{N}\right) S_{uc} \\
\qquad + \beta_1 \left(\frac{a_1 I_p}{N_p} + \frac{a_2 C + a_3 I + a_4 I_t + a_5 D}{N}\right) S_{vc} - (\mu + \theta + \kappa)E, \\
\frac{dC}{dt} = \theta E - (\gamma_3 + \mu + \delta_1)C, \\
\frac{dI}{dt} = \kappa E - (\psi_1 + \psi_2 + \mu + \delta_2 + \gamma_4)I, \\
\frac{dI_{it}}{dt} = \psi_1 I - (\gamma_2 + \mu + \delta_3)I_{it}, \\
\frac{dI_t}{dt} = \psi_2 I - (\gamma_1 + \mu + \delta_4)I_t, \\
\frac{dR}{dt} = \gamma_2 I_{it} + \gamma_4 I + \gamma_1 I_t + \gamma_3 C - \mu R - \epsilon R, \\
\frac{dD}{dt} = \delta_4 I_t + \delta_3 I_{it} + \delta_1 C + \delta_2 I - \mu_d D, \\
\frac{dS_p}{dt} = \Lambda_p - (\sigma + \mu_p)S_p, \\
\frac{dE_p}{dt} = \sigma S_p - (\rho + \mu_p)E_p, \\
\frac{dI_p}{dt} = \rho E_p - (\mu_p + \delta_p)I_p,
\end{cases}
\tag{2.1}
$$

where N is the total number of human beings and N_p is the total number of pigs .

3. Analysis of Nipah virus infectious model

In this section, the central qualities of the NiV model (2.1), such as the invariant area and the model's positivity, are examined using the autonomous model. We locate the two equilibrium points: the endemic and disease-free equilibrium points, and assess each one's stability. While the positivity of the solutions explains nonnegativity of the solutions, meaning that the variables are always nonnegative every times t, the invariant area represents the region in which the solutions of the model (2.1) make biological sense.

3.1. Invariant region

In the Nipah virus model, the human and pig populations are represented, and all state variables are nonnegative at all times $t \geqslant 0$ [1]. As a result, we will assess the model's autonomous model (2.1) in a sufficiently realizable range [21], which we get as follows $N(t) = S(t) + S_v(t) + S_u(t) + S_{vc} + S_{vn} + S_{uc} + S_{un} + E(t) + C(t) + I(t) + I_{it}(t) + I_t(t) + R(t)$, and the total number of pigs $N_p(t)$ as $N_p(t) = S_p(t) + E_p(t) + I_p(t)$. Therefore, we have the differential equations

$$\frac{dN}{dt} = \Lambda - \mu N - \delta_4 I_t - \delta_1 C - \delta_3 I_{it} - \delta_2 I \leqslant \Lambda - \mu N(t) \tag{3.1}$$

and

$$\frac{dN_p}{dt} = \Lambda_p - \mu_p N_p(t) - \delta_p I_p \leqslant \Lambda_p - \mu_p N_p(t). \tag{3.2}$$

Lemma 3.1. *In $\mathbb{R}^{17}_+$, the area $\Omega \cup \Omega_p \in \mathbb{R}^{14}_+ \times \mathbb{R}^3_+$ is positively invariant for the model (2.1) with nonnegative initial conditions, where Ω is the domain of the human population and Ω_p is the domain of the pigs population.*

Proof. Without loss of generality,

$$\frac{dN(t)}{dt} \leqslant \Lambda - \mu N(t),$$

which implies

$$N(t) \leqslant \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right) e^{-\mu t}, \tag{3.3}$$

where the $N(0) = N_0$ is the initial value of the total populations. As $t \longrightarrow \infty$ in (3.3), the population size $N(t)$ approaches $\frac{\Lambda}{\mu}$, i.e.,

$$0 \leqslant N(t) \leqslant \frac{\Lambda}{\mu}$$

and

$$\frac{dD}{dt} = \delta_4 I_t + \delta_3 I_{it} + \delta_1 C + \delta_2 I - \mu_d D \tag{3.4}$$

but $S(t) + S_u(t) + S_v(t) + S_{uc}(t) + S_{un}(t) + S_{vc}(t) + S_{vn}(t) + E(t) + C(t) + I(t) + I_{it}(t) + I_t(t) + R(t) \leqslant \frac{\Lambda}{\mu} \Longrightarrow C(t) \leqslant \frac{\Lambda}{\mu}$, $I(t) \leqslant \frac{\Lambda}{\mu}$, $I_{it}(t) \leqslant \frac{\Lambda}{\mu}$, $I_t(t) \leqslant \frac{\Lambda}{\mu}$ as $t \geqslant 0$. It follows that

$$D(t) \leqslant \frac{(\delta_4 + \delta_3 + \delta_1 + \delta_2)\Lambda}{\mu\mu_d} + [D(0) - \frac{(\delta_4 + \delta_3 + \delta_1 + \delta_2)\Lambda}{\mu\mu_d}]e^{-\mu_d t}. \tag{3.5}$$

As $t \longrightarrow \infty$, then $D(t) \longrightarrow \frac{(\delta_4+\delta_3+\delta_1+\delta_2)\Lambda}{\mu\mu_d}$. Thus, the model system's viable set (2.1) is

$$\Omega \cup \Omega_p = \left\{ \begin{array}{c} (S(t), S_u(t), S_v(t), S_{uc}, S_{un}, S_{vc}, S_{vn}, E(t), C(t), I(t), I_{it}(t), I_t(t), R(t), D(t), \\ S_p, E_p, I_p) \in \mathbb{R}^{17}_+ : S(t), S_u(t), S_v(t), S_{uc}, S_{un}, S_{vc}, S_{vn}, E(t), C(t), \\ I(t), I_{it}(t), I_t(t), R(t), D(t), S_p, E_p, I_p \geqslant 0, S(t) + S_u(t) + S_v(t) + S_{uc}(t) + S_{un}(t) + S_{vc}(t) \\ +S_{vn}(t) + E(t) + C(t) + I(t + I_{it}(t) + I_t(t) + R(t) \leqslant \frac{\Lambda}{\mu}, D(t) \leqslant \frac{(\delta_4+\delta_3+\delta_1+\delta_2)\Lambda}{\mu\mu_d}, \\ S_p + E_p + I_p \leqslant \frac{\Lambda_p}{\mu_p} \end{array} \right\},$$

which is a favorably invariant set under the model's flow (2.1). □

As a result, the system (2.1) is mathematically sound and has epidemiological significance in the area $\Omega \cup \Omega_p$. For this reason, it is necessary in this area to study the dynamics of the model (2.1). In addition, the usual existence, unambiguousness and continuity of results apply to the system.

3.2. Positivity and boundedness of solutions

The answers to the formulae must always be positive. This is significant because it reflects the reality that negative numbers of individuals cannot exist in the actual world. The answers to the equations must be bounded within a certain region. This is significant because it represents the reality that the number of individuals in a population is finite. Positivity and boundedness are essential because they guarantee that the model's answers (2.1) are reasonable and make sense in the actual world [13].

Theorem 3.2. *Let the initial data be* $S(t), S_p(t) > 0$ *and* $S_u(t), S_v(t), S_{uc}, S_{un}, S_{vc}, S_{vn}, E(t), C(t), I(t), I_{it}(t), I_t(t), R(t), D(t), S_p, E_p, I_p \geqslant 0$. *Then the solutions* $(S(t), S_u(t), S_v(t), S_{uc}(t), S_{un}(t), S_{vc}(t), S_{vn}(t), E(t), C(t), I(t), I_{it}(t), I_t(t), R(t), D(t), S_p(t), E_p(t), I_p(t))$ *of the Nipah virus model* (2.1) *are non-negative for all* $t > 0$. *Furthermore*

$$\lim_{t\to\infty} \sup N(t) \leqslant \frac{\Lambda}{\mu}, \quad \lim_{t\to\infty} \sup N_p(t) \leqslant \frac{\Lambda_p}{\mu_p}.$$

Proof. The first equation of the model (2.1) is

$$\begin{array}{l} \frac{dS}{dt} = \Lambda - (\chi_1 + \chi_2 + \mu)S + \epsilon R \geqslant -(\chi_1 + \chi_2 + \mu)S, \qquad \int \frac{dS}{S} \geqslant \int -(\chi_1 + \chi_2 + \mu)dt, \\ S(t) \geqslant Ke^{-(\chi_1+\chi_2+\mu)t}, \qquad S(t) \geqslant S(0)e^{-(\chi_1+\chi_2+\mu)t} > 0, \qquad K \leqslant S(0). \end{array} \tag{3.6}$$

In a similar way, all state variables are non-negative. The right hand side of (3.1) and (3.2) are both bounded by $\Lambda - \mu N$ and $\Lambda_p - \mu_p N_p$, respectively. It follows that $\frac{dN}{dt} < 0$ if $N(t) > \frac{\Lambda}{\mu}$ and $\frac{dN_p}{dt} < 0$ if $N_p(t) > \frac{\Lambda_p}{\mu_p}$. Using a standard comparison theorem [35], then

$$N(t) \leqslant \frac{\Lambda}{\mu} + (N(0) - \frac{\Lambda}{\mu})e^{-\mu t} \quad \text{and} \quad N_p(t) \leqslant \frac{\Lambda_p}{\mu_p} + (N_p(0) - \frac{\Lambda}{\mu})e^{-\mu t},$$

such that $\lim_{t\to\infty} \sup N(t) \leqslant \frac{\Lambda}{\mu}$, and $\lim_{t\to\infty} \sup N_p(t) \leqslant \frac{\Lambda_p}{\mu_p}$. In particular, if $N(0) < \frac{\Lambda}{\mu}$, then $N(t) \leqslant \frac{\Lambda}{\mu}$, and if $N_p(0) < \frac{\Lambda}{\mu}$, then $N_p(t) \leqslant \frac{\Lambda_p}{\mu_p}$. Therefore the region $\Omega \cup \Omega_p \in \mathbb{R}^{14}_+ \times \mathbb{R}^3_+$ is positively invariant and the solutions are bounded. □

3.3. Nipah virus disease free equilibrium points

Nipah virus disease free equilibrium is a special solution of the model equations (2.1) that depicts a situation in which there are no infected people in the community [32] and denoted E_0 such that $\Gamma_1 = \Gamma_2 = \Gamma_3 = \Gamma_4 = 0$. For simplicity, let $\alpha_1 = \chi_1 + \chi_2 + \mu$, $\alpha_2 = \eta_1 + \eta_2 + \mu$, $\alpha_3 = \tau_1 + \tau_2 + \mu$, $\alpha_4 = \mu + \theta + \kappa$, $\alpha_5 = \gamma_3 + \mu + \delta_1$, $\alpha_6 = \psi_1 + \psi_2 + \mu + \delta_2 + \gamma_4$, $\alpha_7 = \gamma_2 + \mu + \delta_3$, $\alpha_8 = \gamma_1 + \mu + \delta_4$, such that

$$E_0 = \left(\frac{\Lambda}{\alpha_1}, \frac{\chi_2\Lambda}{\alpha_1\alpha_3}, \frac{\chi_1\Lambda}{\alpha_1\alpha_2}, \frac{\tau_1\chi_2\Lambda}{\mu\alpha_1\alpha_3}, \frac{\tau_2\chi_2\Lambda}{\mu\alpha_1\alpha_3}, \frac{\eta_1\chi_1\Lambda}{\mu\alpha_1\alpha_2}, \frac{\eta_2\chi_1\Lambda}{\mu\alpha_1\alpha_2}, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda}{\sigma + \mu_p}, 0, 0 \right). \tag{3.7}$$

3.4. Nipah virus endemic equilibrium

The endemic Nipah virus equilibrium point is a special solution of the model equations (2.1) that depicts a situation in which the illness persists in the community but the number of infected people stays constant over time,

$$E_1 = \left(S^*, S_v^*, S_u^*, S_{vc}^*, S_{vn}^*, S_{uc}^*, S_{un}^*, E^*, C^*, I^*, I_{it}^*, I_t^*, R^*, D^*, S_p^*, E_p^*, I_p^*\right). \tag{3.8}$$

Therefore, we calculate for the state variables by equating the model equations to zero. Solving for all the state variables with respect to R, we obtain

$$R^* = \frac{\zeta\varpi(\Lambda + \epsilon R^*)}{\alpha_4\alpha_1(\epsilon+\mu)}, \tag{3.9}$$

where

$$\zeta = \frac{\gamma_2\psi_1\kappa}{\alpha_6\alpha_7} + \frac{\gamma_4\kappa}{\alpha_6} + \frac{\gamma_1\psi_2\kappa}{\alpha_6\alpha_8} + \frac{\gamma_3\theta}{\alpha_5},$$
$$\varpi = \frac{\Gamma_1\tau_1\chi_2}{(\Gamma_1+\mu)\alpha_3\alpha_4} + \frac{\Gamma_2\tau_2\chi_2}{(\Gamma_2+\mu)\alpha_3\alpha_4} + \frac{\Gamma_3\eta_1\chi_1}{(\Gamma_3+\mu)\alpha_2\alpha_4} + \frac{\Gamma_4\eta_2\chi_1}{(\Gamma_4+\mu)\alpha_2\alpha_4}.$$

Solving for R^* in (3.9), we have

$$R^* = \frac{\zeta\varpi\Lambda}{\alpha_4\alpha_1(\epsilon+\mu) - \zeta\varpi\epsilon}. \tag{3.10}$$

Finally, we substitute (3.10) in each of the state variables w.r.t R and obtain

$$S^* = \frac{\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda}{\alpha_1\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]}, \qquad S_v^* = \frac{\chi_2(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{\alpha_1\alpha_3\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]},$$
$$S_u^* = \frac{\chi_1(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{\alpha_1\alpha_2\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]}, \qquad S_{vc}^* = \frac{\tau_1\chi_2(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{(\Gamma_1+\mu)\alpha_3\alpha_1\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]},$$
$$S_{vn}^* = \frac{\tau_2\chi_2(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{(\Gamma_2+\mu)\alpha_3\alpha_1\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]}, \qquad S_{uc}^* = \frac{\eta_1\chi_1(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{(\Gamma_3+\mu)\alpha_2\alpha_1\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]},$$
$$S_{un}^* = \frac{\eta_2\chi_1(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{(\Gamma_4+\mu)\alpha_2\alpha_1\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]}, \qquad E^* = \frac{(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)\varpi}{\alpha_1\alpha_4\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]},$$
$$C^* = \frac{\theta\varpi(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{\alpha_1\alpha_4\alpha_5\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]}, \qquad I^* = \frac{\kappa\varpi(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{\alpha_1\alpha_4\alpha_6\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]},$$
$$I_{it}^* = \frac{\psi_1\kappa\varpi(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{\alpha_1\alpha_4\alpha_6\alpha_7\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]}, \qquad I_t^* = \frac{\psi_2\kappa\varpi(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{\alpha_1\alpha_4\alpha_6\alpha_8\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]},$$
$$D^* = \frac{\Phi\varpi(\Lambda\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right] + \epsilon\zeta\varpi\Lambda)}{\alpha_1\alpha_4\left[\alpha_1\alpha_4(\epsilon+\mu) - \zeta\varpi\epsilon\right]},$$

where

$$\Phi = \frac{\delta_4\psi_2\kappa}{\alpha_6\alpha_8} + \frac{\delta_3\psi_1\kappa}{\alpha_6\alpha 7} + \frac{\delta_1\theta}{\alpha_5} + \frac{\delta_2\kappa}{\alpha_6}, S_p^* = \frac{\Lambda}{\sigma+\mu_p}, E_p^* = \frac{\Lambda\sigma}{(\sigma+\mu_p)(\sigma+\mu_p)}, I_p^* = \frac{\Lambda\sigma\rho}{(\sigma+\mu_p)(\sigma+\mu_p)(\mu_p+\delta_p)}.$$

3.5. The reproduction number R_0

The reproduction number is an important epidemiological measure that defines a disease's ability to propagate within a community. It shows the typical number of secondary infections caused by a single infected person in a vulnerable community [2]. The epidemiological importance of reproduction number stems from its ability to forecast disease spread and direct public health steps to control its dissemination. If the reproduction number is higher than one (1), the disease has the potential to spread throughout

the community, and measures must be taken to limit transmission. In comparison, if the number of reproductions is less than one (1), the disease will ultimately die out, even if no action is taken.

The frequency of new cases in the compartments E and E_p was derived from the model system (2.1) using the method of the next-generation matrix [16],

$$\mathcal{F} = \begin{bmatrix} \Gamma_2 S_{vn} + \Gamma_4 S_{un} + \Gamma_3 S_{uc} + \Gamma_1 S_{vc} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ \sigma S_p \\ 0 \end{bmatrix}. \tag{3.11}$$

Using Python software, the Jacobian matrix of $\mathcal{F}$ (3.11) at the NVFE, E_0, where $N \leqslant \frac{\Lambda}{\mu}$ and $N \leqslant \frac{\Lambda_p}{\mu_p}$ to form the Jacobian matrix is

$$F = \begin{bmatrix} 0 & e_1 & e_2 & e_3 & 0 & e_4 & 0 & e_5 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{3.12}$$

where

$$\begin{cases} e_1 = \frac{a_2\beta_1\chi_2\tau_1}{\alpha_1\alpha_3} + \frac{b_2\beta_2\chi_2\tau_2}{\alpha_1\alpha_3} + \frac{\beta_3\chi_1\eta_1 q_2}{\alpha_1\alpha_2} + \frac{\beta_4\chi_1\eta_2 z_2}{\alpha_1\alpha_2}, \\ e_2 = \frac{a_3\beta_1\chi_2\tau_1}{\alpha_1\alpha_3} + \frac{b_3\beta_2\chi_2\tau_2}{\alpha_1\alpha_3} + \frac{\beta_3\chi_1\eta_1 q_3}{\alpha_1\alpha_2} + \frac{\beta_4\chi_1\eta_2 z_3}{\alpha_1\alpha_2}, \\ e_3 = \frac{a_4\beta_1\chi_2\tau_1}{\alpha_1\alpha_3} + \frac{b_4\beta_2\chi_2\tau_2}{\alpha_1\alpha_3} + \frac{\beta_3\chi_1\eta_1 q_4}{\alpha_1\alpha_2} + \frac{\beta_4\chi_1\eta_2 z_4}{\alpha_1\alpha_2}, \\ e_4 = \frac{a_5\beta_1\chi_2\tau_1}{\alpha_1\alpha_3} + \frac{b_5\beta_2\chi_2\tau_2}{\alpha_1\alpha_3} + \frac{\beta_3\chi_1\eta_1 q_5}{\alpha_1\alpha_2} + \frac{\beta_4\chi_1\eta_2 z_5}{\alpha_1\alpha_2}, \\ e_5 = \frac{\Lambda a_1\beta_1\chi_2\mu_p\tau_1}{\Lambda_p\alpha_1\alpha_3\mu} + \frac{\Lambda b_1\beta_2\chi_2\mu_p\tau_2}{\Lambda_p\alpha_1\alpha_3\mu} + \frac{\Lambda\beta_3\chi_1\eta_1\mu_p q_1}{\Lambda_p\alpha_1\alpha_2\mu} + \frac{\Lambda\beta_4\chi_1\eta_2\mu_p z_1}{\Lambda_p\alpha_1\alpha_2\mu}. \end{cases} \tag{3.13}$$

Calculating the transfer of individuals out of the compartments of the system (2.1) via E, C, I, I_{it}, I_t, D, E_p, I_p, we have

$$\mathcal{V} = \begin{bmatrix} (\mu + \theta + \kappa)E \\ (\gamma_3 + \mu + \delta_1)C - \theta E \\ (\psi_1 + \psi_2 + \mu + \delta_2 + \gamma_4)I - \kappa E \\ (\gamma_2 + \mu + \delta_4)I_{it} - \psi_1 I \\ (\gamma_1 + \mu + \delta_4)I_t - \psi_2 I \\ \mu pD - \delta_4 I_t - \delta_3 I_{it} - \delta_1 C - \delta_2 I \\ (\rho + \mu_p)E_p \\ (\mu_p + \delta_p)I_p - \rho E_p \end{bmatrix}.$$

The Jacobian matrix of $\mathcal{V}$ is

$$V = \begin{bmatrix} \alpha_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\theta & \alpha_5 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\kappa & 0 & \alpha_6 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\psi_1 & 0 & \alpha_7 & 0 & 0 & 0 \\ 0 & 0 & -\psi_2 & \alpha_8 & 0 & 0 & 0 & 0 \\ 0 & -\delta_1 & -\delta_2 & -\delta_4 & -\delta_3 & \mu_p & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & s_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\rho & s_7 \end{bmatrix},$$

where $\alpha_4 = \mu+\theta+\kappa$, $\alpha_5 = \gamma_3+\mu+\delta_1$, $\alpha_6 = \psi_1+\psi_2+\mu+\delta_2+\gamma_4$, $\alpha_7 = \gamma_2+\mu+\delta_3$, $\alpha_8 = \gamma_1+\mu+\delta_4$, $s_6 = \mu_p+\rho$, $s_7 = \delta_p+\mu_p$. Furthermore, we find the inverse of the jacobian matrix V as

$$V^{-1} = \begin{bmatrix} \frac{1}{\alpha_4} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\theta}{\alpha_4\alpha_5} & \frac{1}{\alpha_5} & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\kappa}{\alpha_4\alpha_6} & 0 & \frac{1}{\alpha_6} & 0 & 0 & 0 & 0 & 0 \\ \frac{\kappa\psi_2}{\alpha_4\alpha_6\alpha_8} & 0 & \frac{\psi_2}{\alpha_6\alpha_8} & 0 & \frac{1}{\alpha_8} & 0 & 0 & 0 \\ \frac{\kappa\psi_1}{\alpha_4\alpha_6\alpha_7} & 0 & \frac{\psi_1}{\alpha_6\alpha_7} & \frac{1}{\alpha_7} & 0 & 0 & 0 & 0 \\ \frac{\delta_1\alpha_6\alpha_7\alpha_8\theta+\delta_2\kappa\alpha_5\alpha_7\alpha_8+\delta_3\kappa\psi_1\alpha_5\alpha_8+\delta_4\kappa\psi_2\alpha_5\alpha_7}{\alpha_4\alpha_5\alpha_6\alpha_7\alpha_8\mu_p} & \frac{\delta_1}{\alpha_5\mu_p} & \frac{\delta_2\alpha_7\alpha_8+\delta_3\psi_1\alpha_8+\delta_4\psi_2\alpha_7}{\alpha_6\alpha_7\alpha_8\mu_p} & \frac{\delta_3}{\alpha_7\mu_p} & \frac{\delta_4}{\alpha_8\mu_p} & \frac{1}{\mu_p} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{1}{s_6} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\rho}{s_6 s_7} & \frac{1}{s_7} \end{bmatrix}.$$

Therefore,

$$F.V^{-1} = \begin{bmatrix} w & \frac{\delta_1 e_4}{\alpha_5\mu_p}+\frac{e_1}{\alpha_5} & \frac{e_2}{\alpha_6}+\frac{e_3\psi_2}{\alpha_6\alpha_8}+\frac{e_4(\delta_2\alpha_7\alpha_8+\delta_3\psi_1\alpha_8+\delta_4\psi_2\alpha_7)}{\alpha_6\alpha_7\alpha_8\mu_p} & \frac{\delta_3 e_4}{\alpha_7\mu_p} & \frac{\delta_4 e_4}{\alpha_8\mu_p}+\frac{e_3}{\alpha_8} & \frac{e_4}{\mu_p} & \frac{e_5\rho}{s_6 s_7} & \frac{e_5}{s_7} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

where

$$w = \frac{e_1\theta}{\alpha_4\alpha_5}+\frac{e_2\kappa}{\alpha_4\alpha_6}+\frac{e_3\kappa\psi_2}{\alpha_4\alpha_6\alpha_8}+\frac{e_4\left(\delta_1\alpha_6\alpha_7\alpha_8\theta+\delta_2\kappa\alpha_5\alpha_7\alpha_8+\delta_3\kappa\psi_1\alpha_5\alpha_8+\delta_4\kappa\psi_2\alpha_5\alpha_7\right)}{\alpha_4\alpha_5\alpha_6\alpha_7\alpha_8\mu_p},$$

such that the eigenvalues of $F.V^{-1}$ become

$$\left\{0:7,\ \frac{e_1\theta}{\alpha_4\alpha_5}+\frac{e_2\kappa}{\alpha_4\alpha_6}+\frac{e_3\kappa\psi_2}{\alpha_4\alpha_6\alpha_8}+\frac{e_4\left(\delta_1\alpha_6\alpha_7\alpha_8\theta+\delta_2\kappa\alpha_5\alpha_7\alpha_8+\delta_3\kappa\psi_1\alpha_5\alpha_8+\delta_4\kappa\psi_2\alpha_5\alpha_7\right)}{\alpha_4\alpha_5\alpha_6\alpha_7\alpha_8\mu_p}:1\right\}.$$

Therefore the dominant eigenvalue of $F.V^{-1}$ is

$$R_0 = \frac{e_1\theta}{\alpha_4\alpha_5}+\frac{e_2\kappa}{\alpha_4\alpha_6}+\frac{e_3\kappa\psi_2}{\alpha_4\alpha_6\alpha_8}+\frac{e_4\left(\delta_1\alpha_6\alpha_7\alpha_8\theta+\delta_2\kappa\alpha_5\alpha_7\alpha_8+\delta_3\kappa\psi_1\alpha_5\alpha_8+\delta_4\kappa\psi_2\alpha_5\alpha_7\right)}{\alpha_4\alpha_5\alpha_6\alpha_7\alpha_8\mu_p}. \tag{3.14}$$

Substituting the value of e_i, $i = 1,2,3,4,5$ from (3.13) in (3.14), we obtain

$$\begin{aligned} R_0 = {} & \frac{\kappa\psi_2\left(\frac{a_4\beta_1\chi_2\tau_1}{\alpha_1\alpha_3}+\frac{b_4\beta_2\chi_2\tau_2}{\alpha_1\alpha_3}+\frac{\beta_3\chi\eta_1 q_4}{\alpha_1\alpha_2}+\frac{\beta_4\chi\eta_2 z_4}{\alpha_1\alpha_2}\right)}{\alpha_4\alpha_6\alpha_8}+\frac{\kappa\left(\frac{a_3\beta_1\chi_2\tau_1}{\alpha_1\alpha_3}+\frac{b_3\beta_2\chi_2\tau_2}{\alpha_1\alpha_3}+\frac{\beta_3\chi\eta_1 q_3}{\alpha_1\alpha_2}+\frac{\beta_4\chi\eta_2 z_3}{\alpha_1\alpha_2}\right)}{\alpha_4\alpha_6} \\ & +\frac{\theta\left(\frac{a_2\beta_1\chi_2\tau_1}{\alpha_1\alpha_3}+\frac{b_2\beta_2\chi_2\tau_2}{\alpha_1\alpha_3}+\frac{\beta_3\chi\eta_1 q_2}{\alpha_1\alpha_2}+\frac{\beta_4\chi\eta_2 z_2}{\alpha_1\alpha_2}\right)}{\alpha_4\alpha_5} \\ & +\frac{\left(\frac{a_5\beta_1\chi_2\tau_1}{\alpha_1\alpha_3}+\frac{b_5\beta_2\chi_2\tau_2}{\alpha_1\alpha_3}+\frac{\beta_3\chi\eta_1 q_5}{\alpha_1\alpha_2}+\frac{\beta_4\chi\eta_2 z_5}{\alpha_1\alpha_2}\right)\left(\delta_1\alpha_6\alpha_7\alpha_8\theta+\delta_2\kappa\alpha_5\alpha_7\alpha_8+\delta_3\kappa\psi_1\alpha_5\alpha_8+\delta_4\kappa\psi_2\alpha_5\alpha_7\right)}{\mu_p\alpha_4\alpha_5\alpha_6\alpha_7\alpha_8}. \end{aligned}$$

3.6. Local stability of disease-free Nipah virus steady state

We discuss its qualitative behavior around the equilibrium points using stability analysis of the model equation to get insight regarding the long-term disease dynamics.

Theorem 3.3. *If all of the eigenvalues of $J(E_0)$ have negative real components, the Nipah virus free equilibrium point E_0 is locally asymptotically stable; otherwise, it is unstable.*

Proof. We employ the Jacobian stability technique to determining the local stability of a system (2.1).

$$J(E_0) = \begin{bmatrix}
-\alpha_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \varepsilon & 0 & 0 & 0 & 0 \\
\chi_2 & -\alpha_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\chi_1 & 0 & -\alpha_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \tau_1 & 0 & -\mu & 0 & 0 & 0 & 0 & -g_2 & -g_3 & 0 & -g_4 & 0 & -g_5 & 0 & 0 & -g_6 \\
0 & \tau_2 & 0 & 0 & -\mu & 0 & 0 & 0 & -g_7 & -g_8 & 0 & -g_9 & 0 & -j_1 & 0 & 0 & -j_2 \\
0 & 0 & \eta_1 & 0 & 0 & -\mu & 0 & 0 & -j_3 & -j_4 & 0 & -j_5 & 0 & -j_6 & 0 & 0 & -j_7 \\
0 & 0 & \eta_2 & 0 & 0 & 0 & -\mu & 0 & -j_8 & -j_9 & 0 & -j_{11} & 0 & -j_{12} & 0 & 0 & -j_{13} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -\alpha_4 & j_{14} & j_{15} & 0 & j_{16} & 0 & j_{17} & 0 & 0 & j_{18} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta & -\alpha_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \kappa & 0 & -\alpha_6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \psi_1 & -\alpha_7 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \psi_2 & 0 & -\alpha_8 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_3 & \gamma_4 & \gamma_2 & \gamma_1 & -h_{10} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \delta_1 & \delta_2 & \delta_3 & \delta_4 & 0 & -\mu_3 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -h_9 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -h_{11} & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \rho & -h_{12}
\end{bmatrix}, \tag{3.15}$$

where $h_{10} = \mu + \varepsilon$, $h_9 = \mu_p + \sigma$, $h_{11} = \mu_p + \rho$, $h_{12} = \delta_5 + \mu_p$, $j_1 = \frac{b_5\beta_2\chi_2\tau_2}{\alpha_1\alpha_3}$, $j_2 = \frac{\Lambda b_1\beta_2\chi_2\mu_p\tau_2}{\Lambda_2\alpha_1\alpha_3\mu}$, $j_3 = \frac{\beta_3\chi_1\eta_1q_2}{\alpha_1\alpha_2}$, $j_4 = \frac{\beta_3\chi_1\eta_1q_3}{\alpha_1\alpha_2}$, $j_5 = \frac{\beta_3\chi_1\eta_1q_4}{\alpha_1\alpha_2}$, $j_6 = \frac{\beta_3\chi_1\eta_1q_5}{\alpha_1\alpha_2}$, $j_7 = \frac{\Lambda\beta_3\chi_1\eta_1\mu_pq_1}{\Lambda_2\alpha_1\alpha_2\mu}$, $j_8 = \frac{\beta_4\chi_1\eta_2z_2}{\alpha_1\alpha_2}$, $j_9 = \frac{\beta_4\chi_1\eta_2z_3}{\alpha_1\alpha_2}$, $j_{11} = \frac{\beta_4\chi_1\eta_2z_4}{\alpha_1\alpha_2}$, $j_{12} = \frac{\beta_4\chi_1\eta_2z_5}{\alpha_1\alpha_2}$, $j_{13} = \frac{\Lambda\beta_4\chi_1\eta_2\mu_pz_1}{\Lambda_2\alpha_1\alpha_2\mu}$, $j_{14} = \frac{a_2\beta_1\chi_2\tau_1}{\alpha_1\alpha_3} + \frac{b_2\beta_2\chi_2\tau_2}{\alpha_1\alpha_3} + \frac{\beta_3\chi_1\eta_1q_2}{\alpha_1\alpha_2} + \frac{\beta_4\chi_1\eta_2z_2}{\alpha_1\alpha_2}$, $j_{15} = \frac{a_3\beta_1\chi_2\tau_1}{\alpha_1\alpha_3} + \frac{b_3\beta_2\chi_2\tau_2}{\alpha_1\alpha_3} + \frac{\beta_3\chi_1\eta_1q_3}{\alpha_1\alpha_2} + \frac{\beta_4\chi_1\eta_2z_3}{\alpha_1\alpha_2}$, $j_{16} = \frac{a_4\beta_1\chi_2\tau_1}{\alpha_1\alpha_3} + \frac{b_4\beta_2\chi_2\tau_2}{\alpha_1\alpha_3} + \frac{\beta_3\chi_1\eta_1q_4}{\alpha_1\alpha_2} + \frac{\beta_4\chi_1\eta_2z_4}{\alpha_1\alpha_2}$, $j_{17} = \frac{a_5\beta_1\chi_2\tau_1}{\alpha_1\alpha_3} + \frac{b_5\beta_2\chi_2\tau_2}{\alpha_1\alpha_3} + \frac{\beta_3\chi_1\eta_1q_5}{\alpha_1\alpha_2} + \frac{\beta_4\chi_1\eta_2z_5}{\alpha_1\alpha_2}$, $j_{18} = \frac{\Lambda a_1\beta_1\chi_2\mu_p\tau_1}{\Lambda_2\alpha_1\alpha_3\mu} + \frac{\Lambda b_1\beta_2\chi_2\mu_p\tau_2}{\Lambda_2\alpha_1\alpha_3\mu} + \frac{\Lambda\beta_3\chi_1\eta_1\mu_pq_1}{\Lambda_2\alpha_1\alpha_2\mu} + \frac{\Lambda\beta_4\chi_1\eta_2\mu_2z_1}{\Lambda_2\alpha_1\alpha_2\mu}$, $g_2 = \frac{a_2\beta_1\chi_2\tau_1}{\alpha_1\alpha_3}$, $g_3 = \frac{a_3\beta_1\chi_2\tau_1}{\alpha_1\alpha_3}$, $g_4 = \frac{a_4\beta_1\chi_2\tau_1}{\alpha_1\alpha_3}$, $g_5 = \frac{a_5\beta_1\chi_2\tau_1}{\alpha_1\alpha_3}$, $g_6 = \frac{\Lambda a_1\beta_1\chi_2\mu_p\tau_1}{\Lambda_2\alpha_1\alpha_3\mu}$, $g_7 = \frac{b_2\beta_2\chi_2\tau_2}{\alpha_1\alpha_3}$, $g_8 = \frac{b_3\beta_2\chi_2\tau_2}{\alpha_1\alpha_3}$, $g_9 = \frac{b_4\beta_2\chi_2\tau_2}{\alpha_1\alpha_3}$.

Evaluating $|J(E_0) - \lambda I| = 0$, where λ is the eigenvalue, we have the following: $\lambda_i = -\mu$, $i = 1, 2, 3, 4, 5$, $\lambda_6 = -\alpha_5$, $\lambda_7 = -\alpha_2$, $\lambda_8 = -\mu_d$, $\lambda_9 = -\alpha_3$, $\lambda_{10} = -\alpha_4$, $\lambda_{11} = -\alpha_6$, $\lambda_{12} = -\alpha_7$, $\lambda_{13} = -\alpha_8$, $\lambda_{14} = -h_9$, $\lambda_{15} = -\alpha_1$, $\lambda_{16} = -h_{11}$, $\lambda_{17} = -h_{12}$, $\lambda_{17} = -h_{12}$. Therefore, the Nipah virus free equilibrium point E_0 is locally asymptotically stable. It is evident that all the eigenvalues of $J(E0)$ have negative real parts [8] because the total of a matrix's eigenvalues equals the trace of the matrix and the product of a matrix's eigenvalues equals the determinant of the matrix [11]. This means that all of the eigenvalues of $J(E0)$ have negative real components. □

3.7. Local stability of Nipah virus endemic steady state

The eigenvalues of the Jacobian matrix computed in the endemic equilibrium state can be used to establish the local stability of endemic equilibrium. This technique can be mathematically complex at times [12]. We use central manifold theory method to investigate the stability of endemic equilibrium around $R_0 = 1$. It is also used to examine the existence of backward or forward bifurcation at $R_0 = 1$ ([7, 33]).

Theorem 3.4. *Take the general system of ordinary differential equations with a bifurcation constant a_2^*. shown below:*

$$\frac{dx}{dt} = f(y, a_2^*),\ f : \mathbb{R}^{17} \times \mathbb{R} \to \mathbb{R},\ f \in C^2(\mathbb{R}^{17} \times \mathbb{R}). \tag{3.16}$$

Without loss of generality, it is assumed that 0 is an equilibrium for system (3.15) for all the values of the parameter a_2^*; that is, $f(0, a_2^*) = 0$ for all a_2^*. Assume that

A1. *$A = D_x f(0,0) = (\partial f_i/\partial x_i)(0,0)$ is the linearization matrix of system (3.15) around equilibrium 0 with a_2^* evaluated at 0. Zero is a simple eigenvalue of A, and all other eigenvalues of A have negative real parts;*

A2. *Matrix A has a nonnegative right eigenvector m and a left eigenvector n corresponding to the zero eigenvalue.*

Let f_k *be the* k^{th} *component of* f *and*

$$p = \sum_{k,i,j=1}^{17} n_k m_i \frac{\partial^2 f_k}{\partial y_i \partial a_2^*}(0,0), \qquad g = \sum_{k,i,j=1}^{17} n_k m_i m_j \frac{\partial^2 f_k}{\partial y_i \partial y_j}(0,0), \tag{3.17}$$

for $k,i,j = 1,2,3,\ldots,17$. *The local dynamics of the ordinary differential equation in* (3.15) *around* 0 *is totally determined by* g *and* p *given in* (3.17).

(i.) $g > 0, p > 0$. *When* $a_2^* < 0$ *with* $|a_2^*| \ll 1$, 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium; when* $0 < a_2^* \ll 1$, 0 *is unstable and there exists a negative and locally asymptotically stable equilibrium.*

(ii.) $g < 0, p < 0$. *When* $a_2^* < 0$ *with* $|a_2^*| \ll 1$, 0 *is unstable; when* $0 < a_2^* \ll 1$, 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium.*

(iii.) $g > 0, p < 0$. *When* $a_2^* < 0$ *with* $|a_2^*| \ll 1$, 0 *is unstable, and there exists a locally asymptotically stable negative equilibrium; when when* $0 < a_2^* \ll 1$, 0 *is stable, and a positive unstable equilibrium appears.*

(iv.) $g < 0, p > 0$. *When* a_2^* *changes from negative to positive,* 0 *changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.*

Proof. Let $S = y_1$, $S_v = y_2$, $S_u = y_3$, $S_{vc} = y_4$, $S_{vn} = y_5$, $S_{uc} = y_6$, $S_{un} = y_7$, $E = y_8$, $C = y_9$, $I = y_{10}$, $I_{it} = y_{11}$, $I_t = y_{12}$, $R = y_{13}$, $D = y_{14}$, $S_p = y_{15}$, $E_p = y_{16}$, $I_p = y_{17}$. We write the system of model (3.1)-(3.17) as follows:

$$\begin{cases}
\frac{dS}{dt} = \Lambda - \alpha_1 y_1 + \epsilon y_{13} & : f_1,\\
\frac{dS_v}{dt} = \chi_2 y_1 - \alpha_3 y_2 & : f_2,\\
\frac{dS_u}{dt} = \chi_1 y_1 - \alpha_2 y_3 & : f_3,\\
\frac{dS_{vc}}{dt} = \tau_1 y_2 - (\Gamma_1 + \mu) y_4 & : f_4,\\
\frac{dS_{vn}}{dt} = \tau_2 y_2 - (\Gamma_2 + \mu) y_5 & : f_5,\\
\frac{dS_{uc}}{dt} = \eta_1 y_3 - (\Gamma_3 + \mu) y_6 & : f_6,\\
\frac{dS_{un}}{dt} = \eta_2 y_3 - (\Gamma_4 + \mu) y_7 & : f_7,\\
\frac{dE}{dt} = \Gamma_2 y_5 + \Gamma_4 y_7 + \Gamma_3 y_6 + \Gamma_1 y_4 - \alpha_4 y_8 & : f_8,\\
\frac{dC}{dt} = \theta y_8 - \alpha_5 y_9 & : f_9,\\
\frac{dI}{dt} = \kappa y_8 - \alpha_6 y_{10} & : f_{10},\\
\frac{dI_{it}}{dt} = \psi_1 y_{10} - \alpha_7 y_{11} & : f_{11},\\
\frac{dI_t}{dt} = \psi_2 y_{10} - \alpha_8 y_{12} & : f_{12},\\
\frac{dR}{dt} = \gamma_2 y_{11} + \gamma_4 y_{10} + \gamma_1 y_{12} + \gamma_3 y_9 - (\mu + \epsilon) y_{13} & : f_{13},\\
\frac{dD}{dt} = \delta_4 y_{12} + \delta_3 y_{11} + \delta_1 y_9 + \delta_2 y_{10} - \mu_d y14 & : f_{14},\\
\frac{dS_p}{dt} = \Lambda_p - (\sigma + \mu_p) y_{15} & : f_{15},\\
\frac{dE_p}{dt} = \sigma y_{15} - (\rho + \mu_p) y_{16} & : f_{16},\\
\frac{dI_p}{dt} = \rho y_{16} - (\mu_p + \delta_p) y_{17} & : f_{17},
\end{cases} \tag{3.18}$$

where

$$\Gamma_1 = \beta_1 \left(\frac{a_1 f_{17}}{N_p} + \frac{a_2 f_9 + a_3 f_{10} + a_4 f_{12} + a_5 f_{14}}{N} \right), \quad \Gamma_2 = \beta_2 \left(\frac{b_1 f_{17}}{N_p} + \frac{b_2 f_9 + b_3 f_{10} + b_4 f_{12} + b_5 f_{14}}{N} \right),$$
$$\Gamma_3 = \beta_3 \left(\frac{q_1 f_{17}}{N_p} + \frac{q_2 f_9 + q_3 f_{10} + q_4 f_{12} + q_5 f_{14}}{N} \right), \quad \Gamma_4 = \beta_4 \left(\frac{z_1 f_{17}}{N_p} + \frac{z_2 f_9 + z_3 f_{10} + z_4 f_{12} + z_5 f_{14}}{N} \right).$$

The Jacobian matrix associated with (3.18) at Nipah Virus Endemic equilibrium is given as

$$J(E_1) = \begin{bmatrix}
-\alpha_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \varepsilon & 0 & 0 & 0 & 0 \\
\chi_2 & -\alpha_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\chi_1 & 0 & -\alpha_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \tau_1 & 0 & -A_1 & 0 & 0 & 0 & 0 & -l_1 & -l_2 & 0 & -l_3 & 0 & -l_4 & 0 & 0 & -l_5 \\
0 & \tau_2 & 0 & 0 & -A_2 & 0 & 0 & 0 & -l_6 & -l_7 & 0 & -l_8 & 0 & -l_9 & 0 & 0 & -l_{10} \\
0 & 0 & \eta_1 & 0 & 0 & -A_3 & 0 & 0 & -l_{11} & -l_{12} & 0 & -l_{13} & 0 & -l_{14} & 0 & 0 & -l_{15} \\
0 & 0 & \eta_2 & 0 & 0 & 0 & -A_4 & 0 & -l_{16} & -l_{17} & 0 & -l_{18} & 0 & -l_{19} & 0 & 0 & -l_{20} \\
0 & 0 & 0 & \Gamma_1 & \Gamma_2 & \Gamma_3 & \Gamma_4 & -\alpha_4 & l_{21} & l_{22} & 0 & l_{23} & 0 & l_{24} & 0 & 0 & l_{25} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta & -\alpha_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \kappa & 0 & -\alpha_6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \psi_1 & -\alpha_7 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \psi_2 & 0 & -\alpha_8 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_3 & \gamma_4 & \gamma_2 & \gamma_1 & -A_5 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \delta_1 & \delta_2 & \delta_3 & \delta_4 & 0 & -\mu_d & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -A_6 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -A_7 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \rho & -A_8
\end{bmatrix}, \quad (3.19)$$

where $l_1 = \frac{\alpha_2\beta_1\gamma_4}{N}$, $l_2 = \frac{\alpha_3\beta_1\gamma_4}{N}$, $l_3 = \frac{\alpha_4\beta_1\gamma_4}{N}$, $l_4 = \frac{\alpha_5\beta_1\gamma_4}{N}$, $l_5 = \frac{\alpha_1\beta_1\gamma_4}{Np}$, $l_6 = \frac{b_2\beta_2\gamma_5}{N}$, $l_7 = \frac{b_3\beta_2\gamma_5}{N}$, $l_8 = \frac{b_4\beta_2\gamma_5}{N}$, $l_9 = \frac{b_5\beta_2\gamma_5}{N}$, $l_{10} = \frac{b_1\beta_2\gamma_5}{Np}$, $l_{11} = \frac{\beta_3\mu q_2\gamma_6}{N}$, $l_{12} = \frac{\beta_3\mu q_3\gamma_6}{N}$, $l_{13} = \frac{\beta_3\mu q_4\gamma_6}{N}$, $l_{14} = \frac{\beta_3\mu q_5\gamma_6}{N}$, $l_{15} = \frac{\beta_3\mu q_1\gamma_6}{N}$, $l_{16} = \frac{\beta_4\gamma_7 z_2}{N}$, $l_{17} = \frac{\beta_4\gamma_7 z_3}{N}$, $l_{18} = \frac{\beta_4\gamma_7 z_4}{N}$, $l_{19} = \frac{\beta_4\gamma_7 z_5}{N}$, $l_{20} = \frac{\beta_4\gamma_7 z_1}{N}$, $l_{21} = \frac{\alpha_2\beta_1\gamma_4}{N} + \frac{b_2\beta_2\gamma_5}{N} + \frac{\beta_3 q_2\gamma_6}{N} + \frac{\beta_4\gamma_7 z_2}{N}$, $l_{22} = \frac{\alpha_3\beta_1\gamma_4}{N} + \frac{b_3\beta_2\gamma_5}{N} + \frac{\beta_3 q_3\gamma_6}{N} + \frac{\beta_4\gamma_7 z_3}{N}$, $l_{23} = \frac{\alpha_4\beta_1\gamma_4}{N} + \frac{b_4\beta_2\gamma_5}{N} + \frac{\beta_3 q_4\gamma_6}{N} + \frac{\beta_4\gamma_7 z_4}{N}$, $l_{24} = \frac{\alpha_5\beta_1\gamma_4}{N} + \frac{b_5\beta_2\gamma_5}{N} + \frac{\beta_3 q_5\gamma_6}{N} + \frac{\beta_4\gamma_7 z_5}{N}$, $l_{25} = \frac{\alpha_1\beta_1\gamma_4}{N} + \frac{b_1\beta_2\gamma_5}{N} + \frac{\beta_3 q_1\gamma_6}{N} + \frac{\beta_4\gamma_7 z_1}{N}$, $A_1 = \Gamma_1 + \mu$, $A_2 = \Gamma_2 + \mu$, $A_3 = \Gamma_3 + \mu$, $A_4 = \Gamma_4 + \mu$, $A_5 = \mu + \varepsilon$, $A_6 = \mu_p + \sigma$, $A_7 = \mu_p + \rho$, $A_8 = \mu_p + \delta_5$.

Applying the center manifold theory, let

$$n = (n_1, n_2, n_3, n_4, n_5, n_6, n_7, n_8, n_9, n_{10}, n_{11}, n_{12}, n_{13}, n_{14}, n_{15}, n_{16}, n_{17}),$$
$$m = (m_1, m_2, m_3, m_4, m_5, m_6, m_7, m_8, m_9, m_{10}, m_{11}, m_{12}, m_{13}, m_{14}, m_{15}, m_{16}, m_{17}),$$

be the right and left eigenvector, respectively associated with the zero eigenvalue. Consider $a_2 = a_2^*$ [6] as a bifurcation parameter when $R_0 = 1$. Therefor

$$a_2^* = \frac{\alpha_1\alpha_3\alpha_2\mu_p\alpha_4\alpha_5\alpha_6\alpha_7\alpha_8 - (\alpha_5\alpha_7\mu_p\kappa\psi_2 K_2 + \alpha_5\alpha_7\alpha_8\kappa\mu_p K_3 + K_4 K_1 + \theta K_5\alpha_6\alpha_7\alpha_8\mu_p)}{\mu_p\alpha_2\beta_1\chi_2\tau_1},$$

where

$$K_1 = \delta_1\alpha_6\alpha_7\alpha_8\theta + \delta_2\kappa\alpha_5\alpha_7\alpha_8 + \delta_3\kappa\psi_1\alpha_5\alpha_8 + \delta_4\kappa\psi_2\alpha_5\alpha_7,$$
$$K_2 = \alpha_2 a_4\beta_1\chi_2\tau_1 + \alpha_2 b_4\beta_2\chi_2\tau_2 + \alpha_3\beta_3\chi\eta_1 q_4 + \alpha_3\beta_4\chi\eta_2 z_4,$$
$$K_3 = a_3\alpha_2\beta_1\chi_2\tau_1 + b_3\alpha_2\beta_2\chi_2\tau_2 + \alpha_3\beta_3\chi\eta_1 q_3 + \alpha_3\beta_4\chi\eta_2 z_3,$$
$$K_4 = \alpha_2\alpha_5\beta_1\chi_2\tau_1 + \alpha_2 b_5\beta_2\chi_2\tau_2 + \alpha_3\beta_3\chi\eta_1 q_5 + \alpha_3\beta_4\chi\eta_2 z_5,$$
$$K_5 = b_2\alpha_2\beta_2\chi_2\tau_2 + \alpha_3\beta_3\chi\eta_1 q_2 + \alpha_3\beta_4\chi\eta_2 z_2.$$

Setting $m_{10} > 0$ as a free vector, and multiplying the Jacobian matrix $J(E_1)$ in (3.19) with m^T and equate to zero, we have $m_{15} = m_{16} = m_{17} = 0$, $m_{12} = \frac{\psi_2 m_{10}}{\alpha_8}$, $m_{11} = \frac{\psi_1 m_{10}}{\alpha_7}$, $m_8 = \frac{\alpha_6 m_{10}}{\kappa}$, $m_9 = \frac{\theta\alpha_6 m_{10}}{\kappa\alpha_5}$, $m_{13} = \frac{\Phi_1 m_{10}}{\kappa\alpha_7\alpha_8\alpha_5 A_5}$, where $\Phi_1 = \theta\alpha_7\alpha_8\alpha_6\gamma_3 + \kappa\alpha_7\alpha_8\alpha_5\gamma_4 + \psi_1\kappa\alpha_8\alpha_5\gamma_2 + \psi_2\kappa\alpha_5\alpha_7\gamma_1 > 0$ and $m_{14} = \frac{\Phi_2 m_{10}}{\kappa\alpha_7\alpha_8\alpha_5\mu_d}$, where $\Phi_2 = \theta\alpha_7\alpha_8\alpha_6\delta_1 + \kappa\alpha_7\alpha_8\alpha_5\delta_2 + \psi_1\kappa\alpha_8\alpha_5\delta_3 + \alpha_7\psi_2\alpha_5\kappa\delta_4 > 0$, $m_1 = \frac{\varepsilon\Phi_1 m_{10}}{\alpha_1\kappa\alpha_7\alpha_8\alpha_5 A_5}$, $m_2 = \frac{\chi_2\varepsilon\Phi_1 m_{10}}{\alpha_3\alpha_1\kappa\alpha_7\alpha_8\alpha_5 A_5}$, $m_3 = \frac{\chi_1\varepsilon\Phi_1 m_{10}}{\alpha_2\alpha_1\kappa\alpha_7\alpha_8\alpha_5 A_5}$, $m_4 = \frac{\Phi_3 m_{10}}{\mu_d\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8 A_1 A_5\kappa}$, where $\Phi_3 = \mu_d\tau_1\chi_2\varepsilon\Phi_1 - \Psi_1$, $\Psi_1 = l_1\alpha_6\theta\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8\mu_d + l_2\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8\kappa A_5\mu_d + l_3\psi_2\alpha_1\alpha_3\alpha_5\alpha_7\kappa\mu_d A_5 + l_4\Phi_2\alpha_1\alpha_3 A_5$, $m_5 = \frac{\Phi_4 m_{10}}{\mu_d\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8 A_2 A_5\kappa}$, $m_6 = \frac{\Phi_5 m_{10}}{\mu_d\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8 A_3 A_5\kappa}$, $m_7 = \frac{\Phi_6 m_{10}}{\mu_d\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8 A_4 A_5\kappa}$, where

$$\Phi_4 = \mu_d\tau_2\chi_2\varepsilon\Phi_1 - \Psi_2,$$
$$\Psi_2 = l_6\alpha_6\theta\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8\mu_d + l_7\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8\kappa A_5\mu_d + l_8\psi_2\alpha_1\alpha_3\alpha_5\alpha_7\kappa\mu_d A_5 + l_9\Phi_2\alpha_1\alpha_3 A_5,$$
$$\Phi_5 = \mu_d\eta_1\chi_1\varepsilon\Phi_1 - \Psi_3,$$
$$\Psi_3 = l_{11}\alpha_6\theta\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8\mu_d + l_{12}\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8\kappa A_5\mu_d + l_{13}\psi_2\alpha_1\alpha_3\alpha_5\alpha_7\kappa\mu_d A_5 + l_{14}\Phi_2\alpha_1\alpha_3 A_5,$$

$$\Phi_6 = \eta_2\chi_1\varepsilon\Phi_1\mu_d - \Psi_4,$$
$$\Psi_4 = l_{16}\alpha_6\theta\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8\mu_d + l_{17}\alpha_1\alpha_3\alpha_5\alpha_7\alpha_8\kappa A_5\mu_d + l_{18}\psi_2\alpha_1\alpha_3\alpha_5\alpha_7\kappa\mu_d A_5 + l_{19}\Phi_2\alpha_1\alpha_3 A_5.$$

The left eigenvector of the Jacobian $J(E_1)$ associated with the zero eigenvalue is given by n and it satisfies $n.m = 1$. Multiplying $J^T(E_1)$ by n^T and equating to zero, we obtain $n_4 = \frac{\Gamma_1 n_8}{A_1}$, $n_5 = \frac{\Gamma_2 n_8}{A_2}$, $n_6 = \frac{\Gamma_3 n_8}{A_3}$, $n_7 = \frac{\Gamma_4 n_8}{A_4}$, $n_2 = \frac{K_1 n_8}{A_1A_2\alpha_3}$, $n_3 = \frac{K_2 n_8}{A_3A_4\alpha_2}$, $n_1 = \frac{K_3 n_8}{A_1A_2A_3A_4\alpha_1\alpha_2\alpha_3}$, where $K_1 = A_2\tau_1\Gamma_1 + A_1\tau_2\Gamma_2$, $K_2 = A_4\eta_1\Gamma_3 + A_3\eta_2\Gamma_4$, $K_3 = A_3A_4\alpha_2\chi_2K_1 + \chi_1K_2A_1A_2\alpha_3$, $n_{13} = \frac{\varepsilon K_3 n_8}{A_1A_2A_3A_4A_5\alpha_1\alpha_2\alpha_3}$, $n_{14} = \frac{K_4 n_8}{A_1A_2A_3A_4\mu_d}$, where $K_4 = l_4\Gamma_1A_2A_3A_4 + l_9\Gamma_2A_1A_3A_4 + l_{14}\Gamma_3A_1A_2A_4 + l_{19}\Gamma_4A_21A_3 - l_{24}A_1A_2A_3A_4$ and

$$n_{11} = \frac{K_5 n_8}{\alpha_1\alpha_2\alpha_3\alpha_7A_1A_2A_3A_4A_5\mu_d},$$

where $K_5 = \mu_d\gamma_2\varepsilon K_3 + \delta_3K_4\alpha_1\alpha_2\alpha_3A_5$, $n_{12} = \frac{(Q_2-Q_1)n_8}{\alpha_1\alpha_2\alpha_3\alpha_8\mu_dA_1A_2A_3A_4A_5}$, where

$$Q_2 = l_{23}\alpha_1\alpha_2\alpha_3\mu_dA_1A_2A_3A_4A_5 + \gamma_1\varepsilon K_3\mu_d + \delta_4K_4\alpha_1\alpha_2\alpha_3A_5,$$
$$Q_1 = l_3\Gamma_1\alpha_1\alpha_2\alpha_3\mu_dA_1A_2A_3A_4A_5 + l_8\Gamma_2\alpha_1\alpha_2\alpha_3\mu_dA_1A_2A_3A_4A_5 + l_{13}\Gamma_3\alpha_1\alpha_2\alpha_3\mu_dA_1A_2A_3A_4A_5 + l_{18}\Gamma_4\alpha_1\alpha_2\alpha_3\mu_dA_1A_2A_3A_4A_5,$$
$$n_{10} = \frac{(Q_4 - Q_3)n_8}{\alpha_1\alpha_2\alpha_3\alpha_6\alpha_7\alpha_8\mu_dA_1A_2A_3A_4A_5},$$

where

$$Q_4 = l_{22}\alpha_1\alpha_2\alpha_3\alpha_7\mu_dA_1A_2A_3A_4A_5\alpha_8 + \psi_1K_5\alpha_8 + \psi_2(Q_2 - Q_1)\alpha_7 + \gamma_4\varepsilon K_3\alpha_7\alpha_8 + \delta_2K_4\alpha_1\alpha_2\alpha_3\alpha_7\alpha_8A_5,$$
$$Q_3 = l_2\Gamma_1\alpha_1\alpha_2\alpha_3\alpha_7\mu_dA_1A_2A_3A_4A_5\alpha_8 + l_7\Gamma_2\alpha_1\alpha_2\alpha_3\alpha_7\mu_dA_1A_2A_3A_4A_5\alpha_8 + l_{12}\Gamma_3\alpha_1\alpha_2\alpha_3\alpha_7\mu_dA_1A_2A_3A_4A_5\alpha_8 + l_{17}\Gamma_4\alpha_1\alpha_2\alpha_3\alpha_7\mu_dA_1A_2A_3A_4A_5\alpha_8,$$
$$n_9 = \frac{(\alpha_4\alpha_1\alpha_2\alpha_3\alpha_6\alpha_7\alpha_8\mu_dA_1A_2A_3A_4A_5 - \kappa Q_4 + \kappa Q_3)n_8}{\alpha_1\alpha_2\alpha_3\alpha_6\alpha_7\alpha_8\mu_dA_1A_2A_3A_4A_5\theta}, \quad n_{17} = \frac{(l_{25}A_1A_2A_3A_4 - Q_5)n_8}{A_1A_2A_3A_4A_8},$$

where

$$Q_5 = l_5\Gamma_1A_2A_3A_4 + l_{10}\Gamma_2A_1A_3A_4 + l_{15}\Gamma_3A_1A_2A_4 + l_{20}\Gamma_4A_1A_2A_3,$$
$$n_{16} = \frac{\rho(l_{25}A_1A_2A_3A_4 - Q_5)n_8}{A_1A_2A_3A_4A_7A_8}, \quad n_{15} = \frac{\sigma\rho(l_{25}A_1A_2A_3A_4 - Q_5)n_8}{A_1A_2A_3A_4A_6A_7A_8}.$$

Next we compute the value of g and p such that

$$g = \sum_{k,i,j=1}^{17} n_k m_i m_j \frac{\partial^2 f_k}{\partial y_i \partial y_j}(0,0), \quad p = \sum_{k,i,j=1}^{17} n_k m_i \frac{\partial^2 f_k}{\partial y_i \partial a_2^*}(0,0).$$

We obtain the following:

$$\frac{\partial^2 f_4}{\partial y_4\partial y_{14}} = \frac{-a_5\beta_1}{N}, \quad \frac{\partial^2 f_4}{\partial y_4\partial y_{17}} = \frac{-a_1\beta_1}{N_p}, \quad \frac{\partial^2 f_4}{\partial y_4\partial y_9} = \frac{-a_2\beta_1}{N}, \quad \frac{\partial^2 f_4}{\partial y_4\partial y_{10}} = \frac{-a_3\beta_1}{N}, \quad \frac{\partial^2 f_4}{\partial y_4\partial y_{12}} = \frac{-a_4\beta_1}{N},$$
$$\frac{\partial^2 f_5}{\partial y_5\partial y_{14}} = \frac{-b_5\beta_2}{N}, \quad \frac{\partial^2 f_5}{\partial y_5\partial y_{17}} = \frac{-b_1\beta_2}{N_p}, \quad \frac{\partial^2 f_5}{\partial y_5\partial y_9} = \frac{-b_2\beta_2}{N}, \quad \frac{\partial^2 f_5}{\partial y_5\partial y_{10}} = \frac{-b_3\beta_2}{N}, \quad \frac{\partial^2 f_5}{\partial y_5\partial y_{12}} = \frac{-b_4\beta_2}{N},$$
$$\frac{\partial^2 f_6}{\partial y_6\partial y_{14}} = \frac{-q_5\beta_3}{N}, \quad \frac{\partial^2 f_6}{\partial y_6\partial y_{17}} = \frac{-q_1\beta_3}{N_p}, \quad \frac{\partial^2 f_6}{\partial y_6\partial y_9} = \frac{-q_2\beta_3}{N}, \quad \frac{\partial^2 f_6}{\partial y_6\partial y_{10}} = \frac{-q_3\beta_3}{N}, \quad \frac{\partial^2 f_6}{\partial y_6\partial y_{12}} = \frac{-q_4\beta_3}{N},$$
$$\frac{\partial^2 f_7}{\partial y_7\partial y_{14}} = \frac{-z_5\beta_4}{N}, \quad \frac{\partial^2 f_7}{\partial y_7\partial y_{17}} = \frac{-z_1\beta_4}{N_p}, \quad \frac{\partial^2 f_7}{\partial y_7\partial y_9} = \frac{-z_2\beta_4}{N}, \quad \frac{\partial^2 f_7}{\partial y_7\partial y_{10}} = \frac{-z_3\beta_4}{N}, \quad \frac{\partial^2 f_7}{\partial y_7\partial y_{12}} = \frac{-z_4\beta_4}{N}.$$

Therefor,

$$\begin{aligned} g &= \sum_{k,i,j=1}^{17} n_k m_i m_j \frac{\partial^2 f_k}{\partial y_i \partial y_j} \\ &= n_4 M_4 \left[m_{14}.\frac{-a_5\beta_1}{N} + m_{17}.\frac{-a_1\beta_1}{N_p} + m_9.\frac{-a_2\beta_1}{N} + m_{10}.\frac{-a_3\beta_1}{N} + m_{12}.\frac{-a_4\beta_1}{N} \right] \\ &+ n_5 M_5 \left[m_{14}.\frac{-b_5\beta_2}{N} + m_{17}.\frac{-b_1\beta_2}{N_p} + m_9.\frac{-b_2\beta_2}{N} + m_{10}.\frac{-b_3\beta_2}{N} + m_{12}.\frac{-b_4\beta_2}{N} \right] \\ &+ n_6 M_6 \left[m_{14}.\frac{-q_5\beta_3}{N} + m_{17}.\frac{-q_1\beta_3}{N_p} + m_9.\frac{-q_2\beta_3}{N} + m_{10}.\frac{-q_3\beta_3}{N} + m_{12}.\frac{-q_4\beta_3}{N} \right] \\ &+ n_7 M_7 \left[m_{14}.\frac{-z_5\beta_4}{N} + m_{17}.\frac{-z_1\beta_4}{N_p} + m_9.\frac{-z_2\beta_4}{N} + m_{10}.\frac{-z_3\beta_4}{N} + m_{12}.\frac{-z_4\beta_4}{N} \right]. \end{aligned} \tag{3.20}$$

Since the $n_i.m_j = 1$ for $i = j$, we have $n_4m_4 = n_5m_5 = n_6m_6 = n_7m_7 = 1$. Therefor we simplify (3.20) and collect the like terms

$$\begin{aligned} g = &-\frac{m_{14}}{N}(a_5\beta_1 + b_5\beta_2 + q_5\beta_3 + z_5\beta_4) - \frac{m_{17}}{N}(a_1\beta_1 + b_1\beta_2 + q_1\beta_3 + z_1\beta_4) \\ &- \frac{m_9}{N}(a_2\beta_1 + b_2\beta_2 + q_2\beta_3 + z_2\beta_4) - \frac{m_{10}}{N}(a_3\beta_1 + b_3\beta_2 + q_3\beta_3 + z_3\beta_4) \\ &- \frac{m_{12}}{N}(a_4\beta_1 + b_4\beta_2 + q_4\beta_3 + z_4\beta_4). \end{aligned}$$

Observe that $m_{14}, m_9, m_{10}, m_{12} > 0$ and $m_{17} = 0$, then

$$\begin{aligned} g = &-\frac{m_{14}}{N}(a_5\beta_1 + b_5\beta_2 + q_5\beta_3 + z_5\beta_4) - \frac{m_9}{N}(a_2\beta_1 + b_2\beta_2 + q_2\beta_3 + z_2\beta_4) \\ &- \frac{m_{10}}{N}(a_3\beta_1 + b_3\beta_2 + q_3\beta_3 + z_3\beta_4) - \frac{m_{12}}{N}(a_4\beta_1 + b_4\beta_2 + q_4\beta_3 + z_4\beta_4) < 0. \end{aligned}$$

Computing the value of p, we obtain

$$p = \sum_{k,i,j=1}^{17} n_k m_i \frac{\partial^2 f_k}{\partial y_i \partial a_2^*} = n_5 m_5 \frac{\partial^2 f_5}{\partial y_5 \partial a_2^*} = n_5 m_5 \left[\frac{-y_9}{N} \right],$$

which implies $p = \frac{-y_9}{N} < 0$. Finally, from the above theory section (ii), we conclude that the Nipah virus Endemic equilibrium of the model (2.1) is locally asymptotically stable for $R_0 > 1$, which implies that in the absence of external factors or changes in the environment, the outbreak will eventually decline and disappear within a specific geographical area.

□

3.8. Global stability of equilibrium states

Global stability is an essential idea in disease control because it suggests that once a disease epidemic has been managed and an equilibrium state has been achieved, the disease will not resurface in the future. This is due to the fact that the equilibrium state remains steady in the face of all potential perturbations, such as shifts in transmission rate or population contact patterns.

Theorem 3.5. *The system is globally asymptotically stable at Nipah virus endemic equilibrium* E_1 *and globally stable at Nipah virus free equilibrium* E_0.

Proof. We employ the quadratic lyapunov function ([26, 27]) to prove the above theorem. Let

$$G_1 = S + S_u(t) + S_v(t) + S_{uc} + S_{un} + S_{vc} + S_{vn} + E(t) + C(t) + I(t) + I_{it}(t) + I_t(t) + R(t) + S_p + E_p + I_p.$$

Consider the Lyapunov function

$$2L_f = G_1^2, \quad \frac{dL_f}{dt} = \sum_{i=1}^{16} \frac{dL_f}{di} \times \frac{di}{dt},$$

where $i \in (S, S_u(t), S_v(t), S_{uc}, S_{un}, S_{vc}, S_{vn}, E(t), C(t), I(t), I_{it}(t), I_t(t), R(t), S_p, E_p, I_p)$. This implies

$$\begin{aligned}
\frac{dL_f}{dt} &= G_1 \times \dot{G}_1 \\
&= (S + S_u(t) + S_v(t) + S_{uc} + S_{un} + S_{vc} + S_{vn} + E(t) + C(t) + I(t) + I_{it}(t) \\
&\quad + I_t(t) + R(t) + S_p + E_p + I_p) \times (\dot{S} + \dot{S}_u(t) + \dot{S}_v(t) + \dot{S}_{uc} + \dot{S}_{un} + \dot{S}_{vc} + \dot{S}_{vn} + \dot{E}(t) + \dot{C}(t) \\
&\quad + \dot{I}(t) + \dot{I}_{it}(t) + \dot{I}_t(t) + \dot{R}(t) + \dot{S}_p + \dot{E}_p + \dot{I}_p) \\
&= (S(t) + S_u(t) + S_v(t) + S_{uc} + S_{un} + S_{vc} + S_{vn} + E(t) + C(t) + I(t) + I_{it}(t) \\
&\quad + I_t(t) + R(t) + S_p + E_p + I_p) \times (\Lambda - \mu S - \mu S_u - \mu S_v \\
&\quad - \mu S_{uc} - \mu S_{un} - \mu S_{vc} - \mu S_{vn} - \mu E - \mu C - \delta_1 C - \mu I - \delta_2 I - \mu I_{it} - \delta_3 I_{it} - \mu I_t - \delta_4 I_t - \mu R \\
&\quad + \Lambda_p - \mu_p S_p - \mu_p E_p - \mu_p I_p - \delta_p I_p) \\
&\leqslant (S(t) + S_u(t) + S_v(t) + S_{uc} + S_{un} + S_{vc} + S_{vn} + E(t) + C(t) + I(t) + I_{it}(t) \\
&\quad + I_t(t) + R(t) + S_p + E_p + I_p) \times (\Lambda - \mu N - \delta_1 C - \delta_2 I - \delta_3 I_{it} - \delta_4 I_t + \Lambda_p - \mu_p N_p - \delta_p I_p).
\end{aligned}$$

Observe that $\mu N \leqslant \Lambda$ and $\mu_p N_p \leqslant \Lambda_p$, and using $\mu N = \Lambda$ and $\mu_p N_p = \Lambda_p$,

$$\begin{aligned}
\frac{dL_f}{dt} &\leqslant (S(t) + S_u(t) + S_v(t) + S_{uc} + S_{un} + S_{vc} + S_{vn} + E(t) + C(t) + I(t) + I_{it}(t) \\
&\quad + I_t(t) + R(t) + S_p + E_p + I_p) \times (\Lambda - \mu N - \delta_1 C - \delta_2 I - \delta_3 I_{it} - \delta_4 I_t + \Lambda_p - \mu_p N_p - \delta_p I_p) \\
&= -[\delta_p I_p + \delta_1 C + \delta_2 I + \delta_3 I_{it} + \delta_4 I_t](S(t) + S_u(t) + S_v(t) + S_{uc} + S_{un} \\
&\quad + S_{vc} + S_{vn} + E(t) + C(t) + I(t) + I_{it}(t) + I_t(t) + R(t) + S_p + E_p + I_p).
\end{aligned} \tag{3.21}$$

This shows that the system is globally asymptotically stable at the Nipah virus endemic equilibrium E_1. At the Nipah virus disease free equilibrium E_0, from (3.21), we have $\frac{dL_f}{dt} \leqslant 0$. Therefore the system is globally stable at the Nipah virus free equilibrium. □

4. Numerical solution

In this part, mathematical solutions of the system (2.1) for the data presented in Tables 2 and 3 are performed. We run simulations of the system (2.1) using Python software to see the influence of all the control mechanisms put into the model (2.1). Next, we approximate the initial values and parameters in the manner described below [9]: $N(0) = 1005$, $Np(0) = 150$, $\Lambda_2 = 5$, $\Lambda = 10$, $S(0) = 1000$, $Sv(0) = 420$, $Su(0) = 370$, $Svc(0) = 200$, $Svn(0) = 210$, $Suc(0) = 190$, $Sun(0) = 175$, $E(0) = 10$, $I(0) = 5$, $Iit(0) = 4$, $It(0) = 0$, $C(0) = 0$, $R(0) = 0$, $D(0) = 3$, $Sp(0) = 138$, $Ep(0) = 3$, $Ip(0) = 2$.

The findings below were produced by the Python programming language using the table's parameter values and initial values.

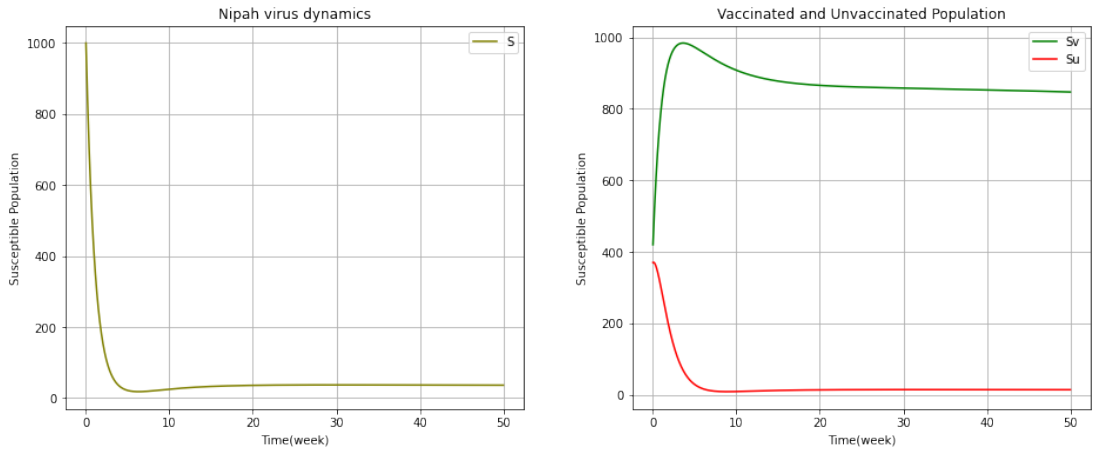
Figure 1 depicts the susceptible population S, S_v, S_u over a period of 50 weeks. In Figure 1 (a), the susceptible S decreased as time increased due to the outbreak of Nipah virus without clue of the kind of virus that emerged but never got to zero due to some level of recruitment into the susceptible population. In Figure 1 (b), we have the susceptible vaccinated and unvaccinated population. The susceptible vaccinated population increased due to the implementation of the vaccine within the first month, slightly changed and remain stable when recognized and response to the virus. However, the susceptible unvaccinated population reduced drastically and nearly got to zero because there is no vaccine implementation. Vaccines are extremely essential because they can prevent you and those around you from becoming ill. When a large number of people are immunized, pathogens and viruses find it much more difficult to disseminate from person to person and thus cannot infect as many people.

Table 2: Description of the parameter.

Parameter	Value	Source
χ_1	0.33	Estimated
χ_2	0.62	Estimated
θ	0.486	Estimated
κ	0.715	Estimated
τ_1	0.008	Estimated
τ_2	0.019	Estimated
η_1	0.45	Estimated
η_2	0.39	Estimated
ψ_1	0.825	Estimated
ψ_2	0.342	Estimated
γ_1	0.8	Inferred from [9]
γ_2	0.5	Inferred from [9]
γ_3	0.09	Inferred from [9]
γ_4	0.1	[9]
β_1	0.1134	Inferred from [9]
β_2	0.3969	Inferred from [9]
β_3	0.4455	Inferred from [9]
β_4	0.7209	Inferred from [9]
δ_1	0.02	Inferred from [9]
ρ	0.56	Estimated
σ	0.75	Estimated

Table 3: Description of the parameter.

Parameter	Value	Source
δ_2	0.15	[9]
δ_3	0.0171	Inferred from [9]
δ_4	0.2	Inferred from [9]
a_1	0.58	Inferred from [9]
a_2	0.513	Inferred from [9]
a_3	0.486	Inferred from [9]
a_4	0.513	Inferred from [9]
a_5	0.000288	Inferred from [9]
b_1	0.69	Inferred from [9]
b_2	0.522	Inferred from [9]
b_3	0.513	Inferred from [9]
b_4	0.504	Inferred from [9]
b_5	0.000324	Inferred from [9]
q_1	0.75	Inferred from [9]
q_2	0.4617	Inferred from [9]
q_3	0.531	Inferred from [9]
q_4	0.513	Inferred from [9]
q_5	0.000648	Inferred from [9]
ϵ	0.03	Estimated
z_2	0.4374	Inferred from [9]
z_3	0.504	Inferred from [9]
z_4	0.513	Inferred from [9]
z_5	0.000648	Inferred from [9]
μ_p	0.00081	Estimated
μ	0.0003421	[34]

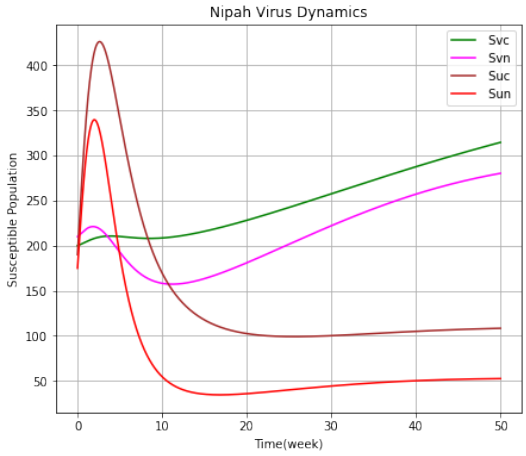


(a) (b)

Figure 1: The graph of susceptible, vaccinated, and unvaccinated populations.

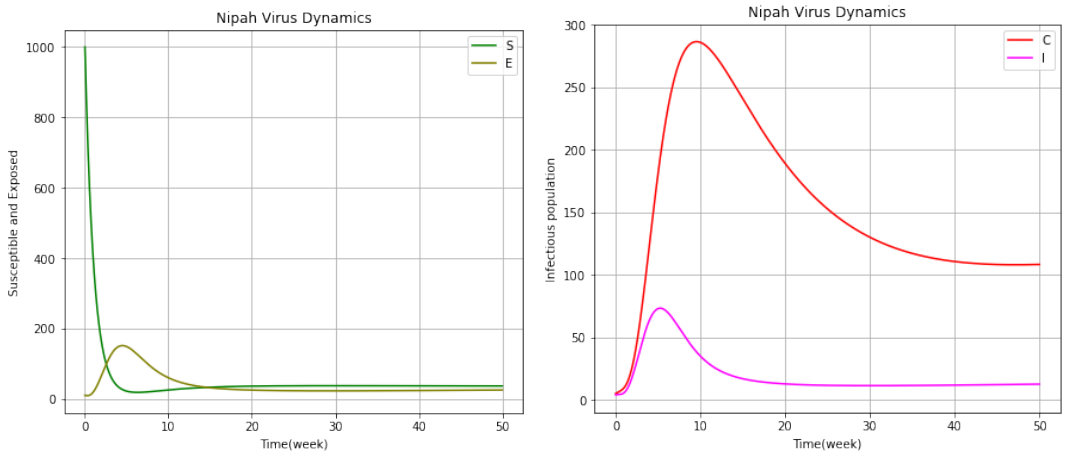
In Figure 2 (a), we have the vaccinated condom users who are susceptible S_{vc}, the vaccinated non-condom users who are susceptible S_{vn}, the unvaccinated condom users who are susceptible S_{uc}, and the unvaccinated non-condom users who are susceptible S_{un}. The susceptible vaccinated condom users S_{vc} slightly fluctuated at the beginning by recognizing the Nipah virus and increases as it responds to

the virus while gaining protection. The vaccinated non-condom users who are susceptible S_{vn} strongly fluctuated at the beginning by recognizing the Nipah virus and increases as it responds to the virus while gaining protection. The unvaccinated condom users who are susceptible S_{uc} and noncondom users who are susceptible S_{un} populations decreased with time but the unvaccinated noncondom users who are susceptible S_{un} population decreased the most and faster while the unvaccinated condom users who are susceptible S_{uc} population decreased the least and slower because of condom use implementation in the population. Combining vaccines and condoms is an efficient method to safeguard against infectious illnesses. Condoms, on the other hand, create a tangible barrier that stops the interchange of bodily fluids during sexual action. Using both vaccines and contraceptives together can provide additional protection against these illnesses. This means that with the implementation of vaccine and condom use in the susceptible population, the Nipah virus transmission can be reduced effectively.



(a)

Figure 2: The graph of susceptible populations.

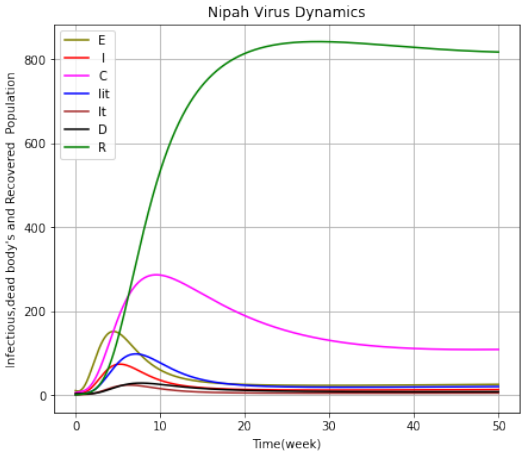


(a) (b)

Figure 3: Exposed, NiV carriers and infectious population.

Figure 3 (a) is the graph of exposed E with respect to susceptible S over a period of 50 weeks. While the exposed individuals dramatically grew from the beginning, the susceptible population declined drastically. When the exposed population reached its apex, the susceptible population is almost nearly to zero but could not due to some level of recruitment into the susceptible. In Figure 3 (b), the infectious

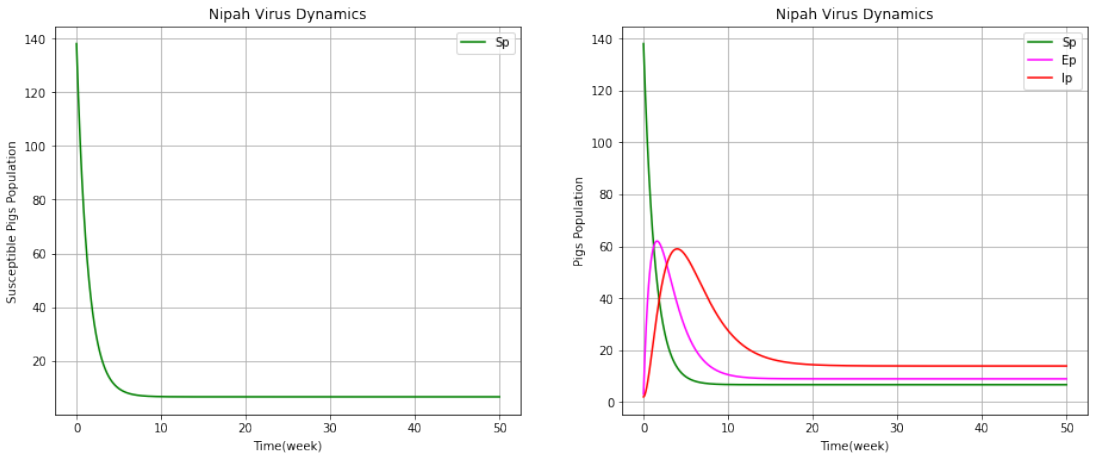
and Nipah virus carries populations increase drastically at the beginning and then shrank over time but the Nipah virus caries population grew the fastest. This is due to the fact that NiV carriers do not show symptoms and are thus more likely to interact with others without taking precautions or seeking medical attention, whereas infectious individuals are more likely to seek medical attention and be diagnosed with the disease, which can help prevent further spread of the disease through quarantine or isolation measures. As a result, NiV carriers may unintentionally raise the risk of spreading the illness to others.



(a)

Figure 4: Exposed, NiV carriers and infectious population.

In Figure 4 (a), the population of infectious patients receiving treatment I_t also grew initially and then shrank over time, but the infectious population I_{it} receiving isolation-based care saw the biggest growth because fewer infectious patients are seeking care in hospitals and more infectious patients are choosing to be treated in isolation. The dead bodies increased from the initial time of Nipah virus outbreak and got to its peak after 8 weeks before declined due to some measure but never get to zero. The recovered population increased to its peak after 30 week, and at this point the infectious and dead bodies population drastically tend to zero while the Nipah virus carries (asymptomatic) was gradually decreasing but not to zero since it shows no symptom. This shows that Nipah virus carries (asymptomatic) population needs serious attention by increasing the rate of testing to identify the individual.



(a) (b)

Figure 5: The graph of pigs population.

In Figure 5 (a), as more pigs are drawn into the pigs community, the susceptible pig population declined over time but did not reach zero. In Figure 5 (b), the numbers of exposed and infectious animals both grew and peaked around two and four weeks, respectively, and then started to decrease.

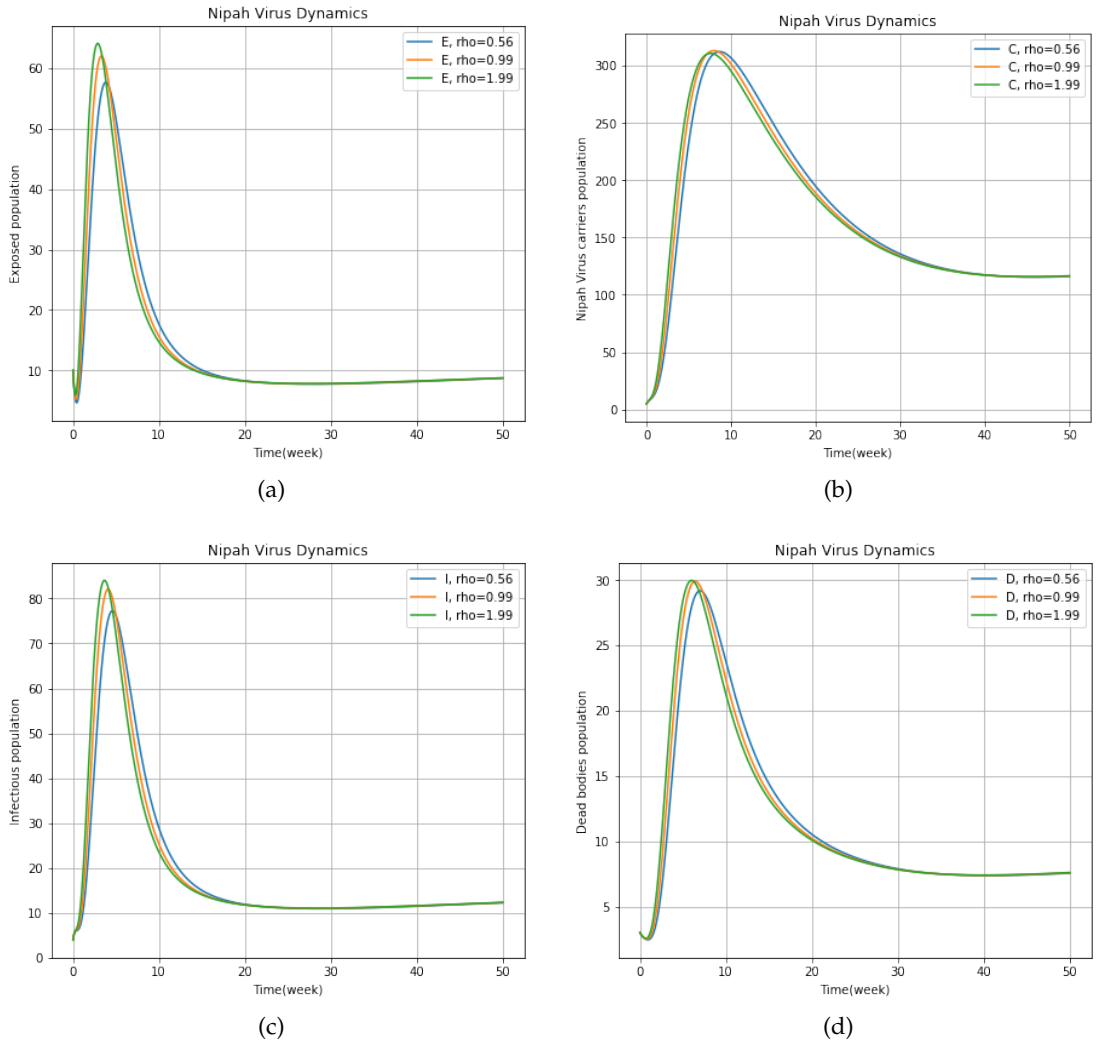
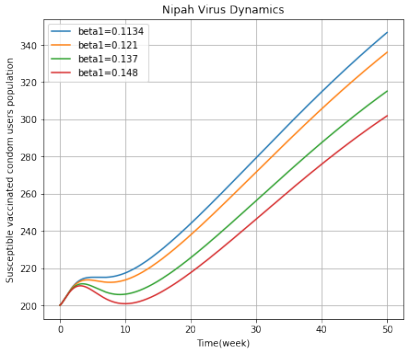


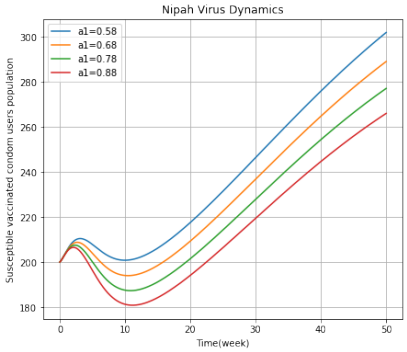
Figure 6: The effect of "rho=ρ" on E, C, I, and D.

The rate at which pigs contract an infection is "Rho=ρ". The impact of changing the pace at which pigs infect people is shown in Figure 6 shows an increase in the pigs infection rate will result to an increase in the human exposure, Nipah virus carries, and human infections. Additionally causing a rise in the number of dead bodies. Therefore, reducing the pig population's contamination rate will protect human lives.

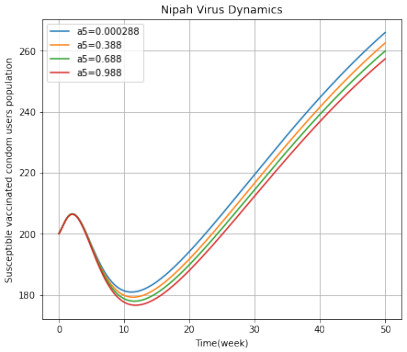
In Figure 7 (a)-(c), simulation result shows the effect of the infection transmission probability β_1, infectious pigs contact rate α_1 and unprotected Nipah virus dead bodies contact rate α_5 on susceptible vaccinated condom users. Even though vaccination and condom use are good strategy, it does not necessary mean that continuous contact with infectious pigs, unprotected NiV dead bodies will not perturb your health status. Therefore, an increase in this parameters, increases the chances of transmission thereby disturbing health status.



(a) Effects of variation of infection transmission probability β_1 with S_{vc}.

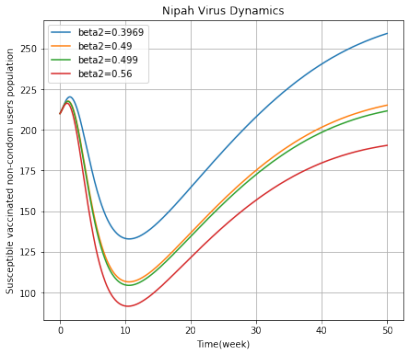


(b) Effects of variation of infectious pigs contact rate a_1 with S_{vc}.

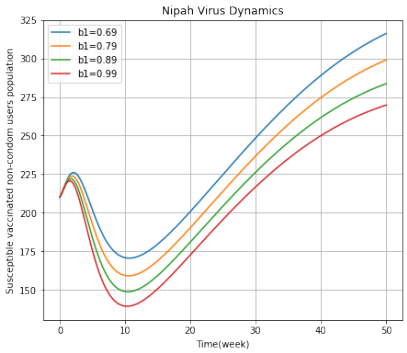


(c) Effects of variation of Nipah virus dead bodies contact rate a_5 with S_{vc}.

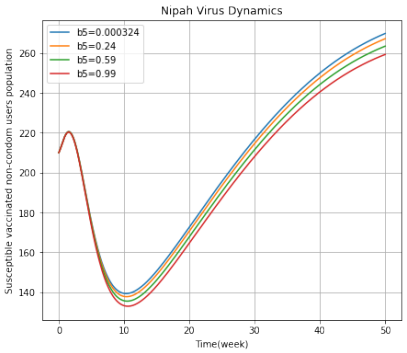
Figure 7: The effect of β_1, a_1, a_5 on S_{vc}.



(a) Effects of variation of infection transmission probability β_2 with S_{vn}.



(b) Effects of variation of infectious pigs contact rate b_1 with S_{vn}.



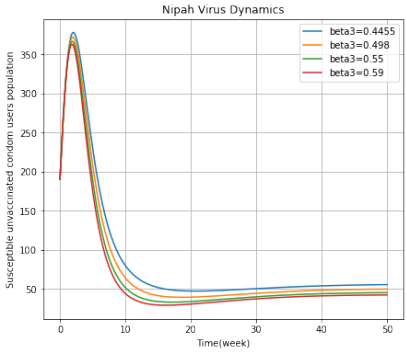
(c) Effects of variation of Nipah virus dead bodies contact rate b_5 with S_{vn}.

Figure 8: The effect of β_2, b_1, b_5 on S_{vn}.

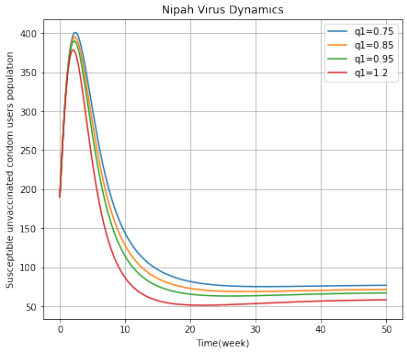
Figure 8 (a)-(c) indicates the effect of the infection transmission probability β_2, infectious pigs contact rate b_1 and unprotected Nipah virus dead bodies contact rate b_5 on susceptible vaccinated non-condom users. This effect is directly proportion to the susceptible vaccinated non-condom users. Therefore, increasing the contact rate of infectious pigs and unprotected Nipah virus dead bodies with susceptible vaccinated non-condom users will amount on an increase in the chances of transmission of Nipah virus. On the other hand, comparing Figure 7 and Figure 8 clearly show that the use of condom is an advantage to reduce the chances of transmission of Nipah virus.

In Figure 9 (a)-(c) depicts the effect of the infection transmission probability β_3, infectious pigs contact rate q_1 and unprotected Nipah virus dead bodies contact rate q_5 on susceptible unvaccinated condom users. Clearly, increasing the contact rate will strengthen the chances of Nipah virus transmission to the susceptible unvaccinated condom users. This indicates high level of transmission of Nipah virus when the susceptible populations are not vaccinated even though they apply condoms. Therefore, the effect of condoms could be relatively observed.

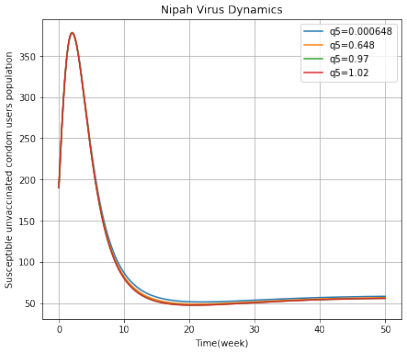
Figure 10 (a)-(c) describes the effect of the infection transmission probability β_4, infectious pigs contact rate z_1 and unprotected Nipah virus dead bodies contact rate z_5 on susceptible unvaccinated non-condom users. The population of susceptible unvaccinated non-condom users decreases drastically very close to zero at shortest time interval when compare with susceptible unvaccinated condom users. This implies that there is relative prevention of Nipah virus transmission when using condom. In addition, Figures 7-10 suggest that the implementation of vaccine and the use of condom are good strategy to minimize the spread of Nipah virus.



(a) Effects of variation of infection transmission probability β_3 with S_{uc}.

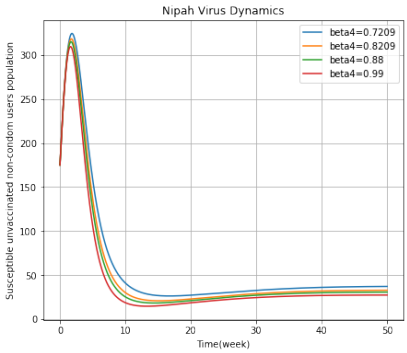


(b) Effects of variation of infectious pigs contact rate q_1 with S_{uc}.

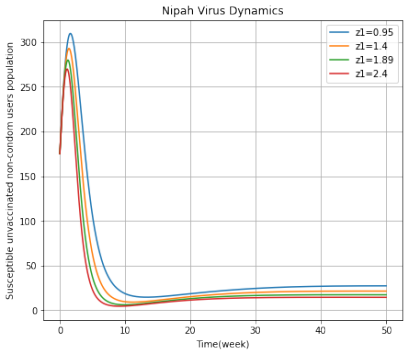


(c) Effects of variation of Nipah virus dead bodies contact rate q_5 with S_{uc}.

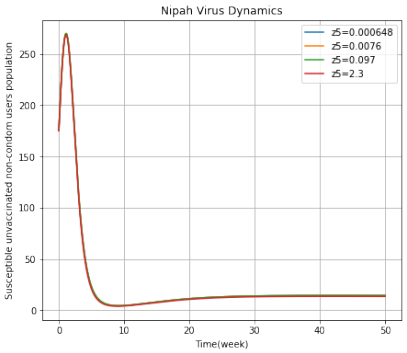
Figure 9: The effect of β_3, q_1, q_5 on S_{uc}.



(a) Effects of variation of infection transmission probability β_4 with S_{un}.



(b) Effects of variation of infectious pigs contact rate z_1 with S_{un}.



(c) Effects of variation of Nipah virus dead bodies contact rate z_5 with S_{un}.

Figure 10: The effect of β_4, z_1, z_5 on S_{un}.

5. Discussion and conclusion

The spread of the NiV from pigs to persons and persons to persons was examined in this study. Since a phase 1 clinical trial of a Nipah virus vaccine candidate (HeV-sG-V) began in February 2020, we presummated the presence of a vaccine for the Nipah virus [28].

We were inspired to add to knowledge in this field because there have been few studies on the transmission dynamics of Nipah virus in the research. We created a seventeen-compartment model to investigate the mechanisms of NiV infection from pigs to persons and persons to persons. We discussed the invariant region, positivity and boundedness of solution of the model, Nipah virus equilibria points; the Nipah virus disease free equilibrium and Nipah virus endemic equilibrium, the replication number R_0, local and global stability. While the positivity of the solution demonstrated that the epidemic system has no negative solutions and that the variables are never negative, the invariant region highlighted the region in which the model's solution makes biological sense. Positivity and boundedness are essential because they guarantee that the model's answers are reasonable and make sense in the actual world. The replication number measures disease's infectiousness and an essential epidemiological tool because it gives useful information about a disease's ability to spread through a community. If the reproduction number is higher than one (1), the disease has the potential to spread throughout the community, and measures must be taken to limit transmission. In comparison, if the number of reproductions is less than one (1), the disease will ultimately die out, even if no action is taken. With the help of Python software, we calculated the replication number R_0 of the model using the next-generation technique, and we then

used it to analyze the model's equilibrium points. The Nipah virus would be completely eliminated from the system if all of our control measures are successfully implemented because the Nipah virus free equilibrium point was locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. At the Nipah virus prevalent equilibrium, our model is globally asymptotically stable, and at the Nipah virus disease free equilibrium, it is globally stable.

Numerical simulations have instantiate the behaviour and flow of Nipah virus infections in different compartments, which shows that population of the infectious pigs and human, dead bodies of an infectious individuals can affect the susceptible individuals. Furthermore, the Nipah virus carries (asymptomatic) are most infectious than symptomatic individuals. This is due to the fact that NiV carriers do not show symptoms and are thus more likely to interact with others without taking precautions or seeking medical attention, whereas infectious individuals are more likely to seek medical attention and be diagnosed with the disease, which can help prevent further spread of the disease through quarantine or isolation measures. As a result, NiV carriers may unintentionally raise the risk of spreading the illness to others and should be targeted. It also indicates that vaccine, and condom use are an efficient method to safeguard against infectious illnesses. Therefore, combining vaccines, and condom can provide additional protection against these illnesses. Therefore we suggest the implementation of vaccine, the use of condom, safe and protected contact for healthcare workers especially the mortuary attendants, and rapid testing of individuals to identify the infectious individuals for isolation and treatment.

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