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A MATHEMATICAL MODEL TO PROVIDE INSIGHTS INTO FOOD-BORNE NIPAH VIRUS INFECTIOUS DISEASE TRANSMISSION DYNAMICS

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Abstract. This study extends the SEIR model to 16 compartments ($S_H, S_V, S_{HW}, E_H, E_{HT}, I_H, I_{HT}, I_{Hi}, R_H, S_P, S_{PV}, I_P, I_{PT}, R_P, S_B, I_B$) to analyze Nipah virus (NiV) transmission dynamics. We computed the basic reproductive number (R_0) and investigated local stability of the disease-free equilibrium using the Jacobian Matrix, and disease-endemic stability with the center manifold theorem. Global stability was assessed using LaSalle's Invariant Principle, and sensitivity analysis was performed. Our results indicated that disease-free and endemic equilibria are locally and globally stable, with the system showing a forward bifurcation. Simulations enhanced understanding of NiV's long-term behavior. We established a critical threshold of 12.2374 for the rate of consumption of NiV-contaminated food items (Λ_{HW}), beyond which the disease could escalate uncontrollably. Graphical simulations suggested that, in a food community of 1,558,025 individuals, the number consuming contaminated food should

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not exceed 12 to prevent virus spread. These insights can guide policymakers in developing targeted NiV control strategies. Sensitivity analysis identified key parameters affecting R_0: the exposed rate (β_1) and the modification parameter for decreased human infectiousness (n_0), both with significant economic implications. By focusing on these parameters, developing countries can implement initiatives to mitigate NiV spread and its economic impact. Our model offers a foundation for targeted intervention strategies.

Keywords: virus; epidemiology; stability; equilibrium-point; transmission.

2020 AMS Subject Classification: 92D30, 34D20, 34C60.

1. INTRODUCTION

Nipah virus (NiV) is a member of the Paramyxoviridae family, specifically the Henipavirus genus. The virus was first discovered in 1999 during a Malaysian outbreak (Weingartl, Berhane and Czub [21], Kaku [9]). This virus is a single-stranded RNA virus called NiV, which can cause serious sickness both in human and animals. Its two distinct strains are the NiV Malaysia (NiVM) and the NiV Bangladesh (NiVB). NiV disease has an incubation period that typically ranges between 14 to 28 days after a fully susceptible individual has been exposed to the virus (Escaffre et. al [6]). Today, there is no particular strategy or method for treating NiV disease. Due to NiV potential for attacking multiple organs (like brain, liver, and lungs), which in most cases leads to severe and often fatal diseases, its ability to be transmitted from human to human, and its high potential for nosocomial (hospital-based or laboratory) outbreaks, it has been listed as a bio-safety level-4 (BSL-4) pathogen by WHO and other international health bodies (Delamater et. al, [5]). From existing literature, there are numerous works on mathematical models for NiV transmission dynamics, but not one captured the spread of NiV as a single variant food-borne infectious disease while at the same time synchronizing the humans, bats, and pigs compartments to establish an in-depth knowledge of the NiV transmission dynamics and control (Sharma et. al, [18]). This research aimed to develop a mathematical epidemiological model for the study of the transmission and control of Niv single variant food-borne disease in humans, pigs, and bat populations. The specific objectives of the study were given as: (i) develop a mathematical model NiV single variant food-borne transmission and control; (ii) establish the equilibrium points of the NiV developed model to gain a better understanding of the long-term implications of the virus; (iii) envisage the future course of NiV epidemic through the

basic reproductive number (R_0) of NiV from the general model; (iv) perform sensitivity analysis of the model to help us pinpoint which parameter(s) has greatest effect on the transmission of the disease; (v) Simulations were done to give us more insight on the system of equations behavior.

2. PRELIMINARIES

2.1. Model Formulation and Development.

We now develop a mathematical model representing this information that can enable us to carry out some analyses and provide vital information for medical health officials about the virus-caused disease after deliberating in detail the process and mode of transmission of this virus in the community and noting the peculiar features in these areas in question that aided the spread of the virus (Sheeley [31]). To do so successfully, we have the following model assumptions:

2.2. Model Assumptions.

1. Those recruited into the system are not infected or contacted the disease.
2. We focus on a single strain Nipah virus, like the Nipah virus Malaysia (NiV_M) strain.
3. There is a susceptible class for human, susceptible human drinking palm wine, and also susceptible humans vaccinated.
4. There is an Intensive supportive care for those infected with the virus that goes for treatment.
5. The treatment given to those infected is the combinational therapy method, because as the time of this study there is no single specific approach or method for treating Nipah virus disease.
6. We anticipate the development of vaccine for treatment of the disease, which we integrated in our study.
7. We hope to use mass media campaign as well as geographical control (on areas with high alert on Nipah virus during an outbreak), as our control for this study.
8. Nipah virus is seen to be a highly emerging disease and no single drug is available yet for its treatment.

9. Humans can contact the disease by contact with infected bats, infected pigs or infected humans.

10. The infected individual can only recover by care giving/combinational therapy.

11. An infectious human that is critically ill could be isolated.

Considering the above assumptions, the total human, animal, and bird populations were grouped based on the disease or non-disease status of those involved, which led us to the following:

The human population is divided into four pairwise disjoint compartments, which are: susceptible humans (S_H), susceptible humans vaccinated (S_V), susceptible humans consuming palm wine (S_{HW}), infected humans (I_H), infected humans that go for treatment (I_{HT}), and recovered humans (R_H).

The susceptible human compartment is increased by Λ_H, it is reduced by natural death (μ_H), it also has a susceptible human vaccinated compartment which is increased by Λ_V, and susceptible human drinking palm wine compartment is increased by Λ_{HW}.

The human healthy population is also reduced by contact with the infected class by n_0, n_1, β_1, and n_2, where β_1 is the rate of infection in the pig population, while n_0 is the modification parameter resulting from various factors such as social gatherings, economy, status, and so on. Similarly, n_1 is the modification parameter resulting from various factors, and n_2 is the modification parameter resulting from our social activities in the bush or farm.

2.3. Model Flow-Diagram.

In this section, we present the flow diagram (see Figure 1), which would help us formulate our proposed Nipah virus model:

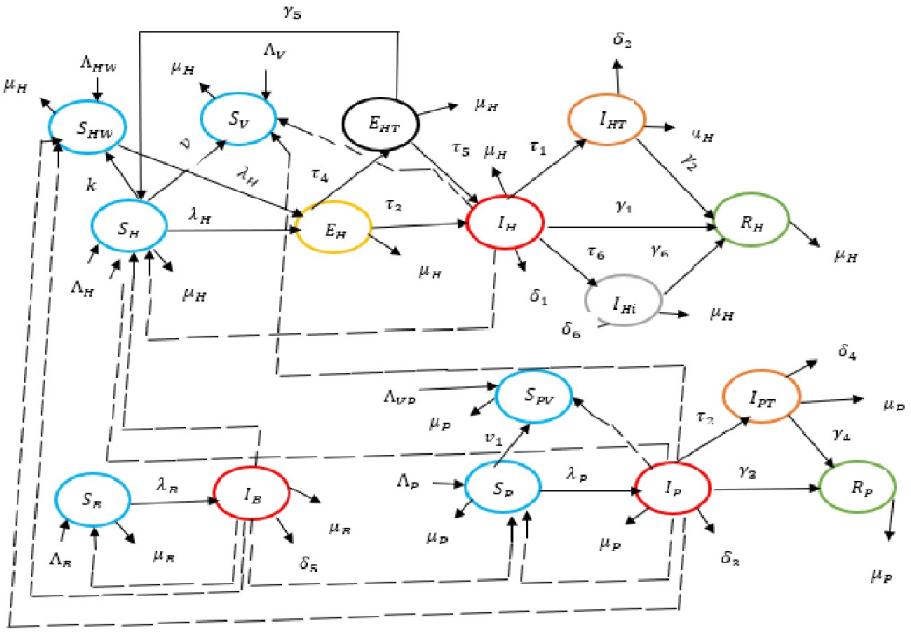

FIGURE 1. Flow Diagram for Nipah virus transmission.

We then obtained the following mathematical system of equations from the model assumptions and flow-diagram in Figure 1:

2.4. General Nipah Virus Dynamic Model.

$$\frac{dS_H}{dt} = \Lambda_H + \gamma_5 E_{HT} - \left(v + \frac{\beta_1 \left[n_0 I_H + n_1 I_P + n_2 I_B \right]}{N_H} + k + \mu_H \right) S_H \tag{1}$$

$$\frac{dS_V}{dt} = \Lambda_V + v S_H - \mu_H S_V \tag{2}$$

$$\frac{dS_{HW}}{dt} = \Lambda_{HW} + k S_H - \left(\frac{\beta_1 \left[n_0 I_H + n_1 I_P + n_2 I_B \right]}{N_H} + \mu_H \right) S_{HW} \tag{3}$$

$$\begin{aligned} \frac{dE_H}{dt} = & \left(\frac{\beta_1 \left[n_0 I_H + n_1 I_P + n_2 I_B \right]}{N_H} + \mu_H \right) S_H + \left(\frac{\beta_1 \left[n_0 I_H + n_1 I_P + n_2 I_B \right]}{N_H} + \mu_H \right) S_{HW} \\ & - \left[\tau_4 + \tau_3 + \mu_H \right] E_H \end{aligned} \tag{4}$$

$$\frac{dE_{HT}}{dt} = \tau_4 E_H - \left(\tau_5 + \gamma_5 + \mu_H \right) E_{HT} \tag{5}$$

$$\frac{dI_H}{dt} = \tau_3 E_H + \tau_5 E_{HT} - \left(\delta_1 + \tau_1 + \mu_H \right) I_H \tag{6}$$

$$\frac{dI_{HT}}{dt} = \tau_1 I_H - (\gamma_2 + \delta_2 + \tau_6 + \mu_H) I_{HT} \tag{7}$$

$$\frac{dI_{Hi}}{dt} = \tau_6 I_{HT} - (\gamma_6 + \delta_6 + \mu_H) I_{Hi} \tag{8}$$

$$\frac{dR_H}{dt} = \gamma_2 I_{HT} + \gamma_6 I_{Hi} - \mu_H R_H \tag{9}$$

$$\frac{dS_P}{dt} = \Lambda_P - \left(\frac{\beta_2 [n_1 I_P + n_2 I_B]}{N_P} + \mu_P \right) S_P - (v_1 + \mu_P) S_P \tag{10}$$

$$\frac{dS_{VP}}{dt} = \Lambda_{VP} + v_1 S_P - \mu_P S_{VP} \tag{11}$$

$$\frac{dI_P}{dt} = \left(\frac{\beta_2 [n_1 I_P + n_2 I_B]}{N_P} + \mu_P \right) S_P - [\gamma_3 + \delta_3 + \tau_2 + \mu_P] I_P \tag{12}$$

$$\frac{dI_{PT}}{dt} = \tau_2 I_P - (\gamma_4 + \delta_4 + \mu_P) I_{PT} \tag{13}$$

$$\frac{dR_P}{dt} = \gamma_3 I_P + \gamma_4 I_{PT} - \mu_P R_P \tag{14}$$

$$\frac{dS_B}{dt} = \Lambda_B - \lambda_B S_B - \mu_B S_B \tag{15}$$

$$\frac{dI_B}{dt} = \lambda_B S_B - (\delta_5 + \mu_B) I_B \tag{16}$$

Where the forces of infection for the NiV sub-model are: $\lambda_H, \lambda_P, \lambda_B$:

$$\lambda_H = \frac{\beta_1 (n_0 I_H + n_1 I_P + n_2 I_B)}{N_H} \tag{17}$$

$$\lambda_P = \frac{\beta_2 (n_1 I_P + n_2 I_B)}{N_P} \tag{18}$$

$$\lambda_B = \frac{\beta_3 n_2 I_B}{N_B} \tag{19}$$

Note: β_1, β_2, and β_3 are known as contact rates, which are for humans, pigs, and bats respectively.

3. MAIN RESULTS

3.1. Model Analysis.

After developing the model equations, we inspect the model's well-posedness by demonstrating that the model satisfied the constraints on the positivity of the model's variables as well as the constant remaining of the variables in the region of existence of the variables (Escaffre et. al [7], Mbah et. al [10], Zheng, Wang and Fu [29]). As a result, we get the following:

3.2. Invariant Region of Solution.

Since equations 1 to 16 reflect the general population, it is necessary to emphasize that the associated population sizes can never be negative. Thus, we establish the invariance of Ω as follows:

$$\Omega = \left\{ \begin{array}{l} (S_H, S_V, S_{HW}, E_H, E_{HT}, I_H, I_{HT}, I_{Hi}, R_H, \\ \\ S_P, S_{PV}, I_P, I_{PT}, R_P, S_B, I_B) \in \mathbb{R}_+^{16} \end{array} \right\} \tag{20}$$

then

$$\left\{ \begin{array}{l} (S_H, S_V, S_{HW}, E_H, E_{HT}, I_H, I_{HT}, I_{Hi}, R_H, \\ \\ S_P, S_{PV}, I_P, I_{PT}, R_P, S_B, I_B) \text{ and } N(t) \end{array} \right\} \geq 0, \forall t \geq 0 \tag{21}$$

In our proposed model, we deploy the triple helix approach of the human, pig, and bat populations, ensuring that all state variables remain non-negative at all times t.

Theorem 1. *Suppose we have a positive region, Ω, which is defined as:*

$$\Omega = \left\{ \begin{array}{l} (S_H, S_V, S_{HW}, E_H, E_{HQ}, I_H, I_{HT}, I_{Hi}, R_H, \\ \\ S_P, S_{PV}, I_P, I_{PT}, R_P, S_B, I_B) \in \mathbb{R}_+^{16} \end{array} \right\} \geq 0 \tag{22}$$

Proof. We show that Ω is positively invariant so that it is sufficient to consider the dynamics of the model system.

From (1):

$$\frac{dS_H}{dt} = \Lambda_H + \gamma_5 E_{HT} - \lambda_H S_H - kS_H - (v + \mu_H) S_H$$

Without loss of generality, we have the following inequality:

$$\boxed{\Rightarrow} \quad \frac{dS_H}{dt} \geq -[\lambda_H + k + v + \mu_H] S_H \tag{23}$$

Next, we integrate both sides:

$$\int \frac{dS_H}{S_H} \geq -\int [\lambda_H + k + v + \mu_H] dt \tag{24}$$

$$\ln(S_H) \geq -(\lambda_H + k + v + \mu_H)t + S(t) \tag{25}$$

$$e^{\ln(S_H)} \geq e^{-(\lambda_H + k + v + \mu_H)t + S(t)} \tag{26}$$

At t = 0,

$$S_H \geq S(0)$$

This implies that

$$S_H \geq 0 \tag{27}$$

The solution is bounded by zero below and is positive for all $t > 0$. We therefore continue using this same approach to prove for the remaining variables.

Continuing with our analytical exploration, we shift our focus to another aspect—the ongoing presence or absence of this infection within the population. This involves scrutinizing the equilibrium points of the disease in both the disease-free and endemic states. During this analysis, we assume a consistent virulence level, avoiding the study of various viral strains. Consequently, we examine the equilibrium state in the following manner: □

3.3. Disease-Free Equilibrium (DFE). In this section, we attempted to determine the values for parameters in which equations 1 to 16 of our Nipah virus epidemiological model accurately represent a situation where the NiVD has been eliminated, and no individuals in the population are infected (Goswami and Hategekimana [8], Boonpatcharanon, Heffernan and Jankowski [30]). This analysis is crucial for gaining insights into the long-term behavior of Nipah virus disease within a population and for making meaningful predictions about its dynamics.

Let

$$A^0(S_H^0, S_V^0, S_{HW}^0, E_H^0, E_{HT}^0, I_H^0, I_{HT}^0, I_{Hi}^0, R_H^0, S_P^0, S_{PV}^0, I_P^0, I_{PT}^0, R_P^0, S_B^0, I_B^0)$$

be the equilibrium points of the displayed system (equations 1– 16). Then, at the equilibrium state, we obtain the disease-free equilibrium (DFE) point:

$$A^0 = \left(\frac{\Lambda_H}{k+v+\mu_H}, \frac{\Lambda_V(k+v+\mu_H)+v\Lambda_H}{\mu_H(k+v+\mu_H)}, \frac{\Lambda_{HW}(k+v+\mu_H)+k\Lambda_H}{\mu_H(k+v+\mu_H)}, 0,0,0,0,0,0, \frac{\Lambda_P}{\mu_P}, \right. \tag{28}$$
$$\left. \frac{\mu_P\Lambda_{PV}+v_1\Lambda_P}{\mu_P}, 0,0,0, \frac{\Lambda_B}{\mu_B}, 0 \right)$$

3.4. Endemic Disease Equilibrium (EE).

In this section, we examine the endemic equilibrium state of the disease. This state occurs when the disease remains present within the population compartments, rather than being completely eradicated. At this equilibrium, the disease persists in the population, meaning that the numbers in the susceptible, infectious, treated infectious, recovered, and other classes are not all zero (Mbah et. al [10], Scott et. al [16]). To obtain the endemic equilibrium points of the model, we solve equations 1 to 16 simultaneously.

3.5. Basic Reproductive Number (R_0) for the Study.

In this section, we aim to discover a key epidemiological metric that may be used to assess the likelihood of a disease spreading within a population [26, 25]. This is the expected number of secondary infections produced when one infectious individual is introduced into a susceptible population [5]. Arguably, it is the most important threshold parameter that determines whether an infectious disease can invade a population [13].

Theorem 2. *Examine a set of ordinary differential equations elucidating the transmission dynamics of an infectious disease. In this context, let F denote the matrix depicting the rate of new infections, and V represents the matrix illustrating the rate at which individuals transition from the infected state to the recovered (or other removed) state. The fundamental reproductive number, denoted as R_0, is defined as the spectral radius of the matrix product FV^{-1} Boonpatcharanon, Heffernan, and Jankowski* [30]*, Obasi and Mbah* [12].

Proof. From the model of our study, equations (1) to (16), we have: Where f_i's are new infections, while v_i's are transferred infections to other classes:

$$f_i = \begin{pmatrix} f_1 & = \frac{\beta_1[n_0 I_H + n_1 I_P + n_2 I_B]S_H}{N_H} + \frac{\beta_1[n_0 I_H + n_1 I_P + n_2 I_B]S_{HW}}{N_H} \\ f_2 & = 0 \\ f_3 & = 0 \\ f_4 & = 0 \\ f_5 & = 0 \\ f_6 & = \frac{\beta_2[n_1 I_P + n_2 I_B]S_P}{N_P} \\ f_7 & = 0 \\ f_8 & = \frac{\beta_3 n_2 I_B S_B}{N_B} \end{pmatrix} \tag{29}$$

$$v_i = \begin{pmatrix} v_1 & = [\tau_4 + \tau_3 + \mu_H]E_H \\ v_2 & = -\tau_4 E_H + [\tau_5 + \gamma_5 + \mu_H]E_{HT} \\ v_3 & = -\tau_3 E_H - \tau_5 E_{HT} + [\gamma_1 + \delta_1 + \tau_1 + \mu_H]I_H \\ v_4 & = -\tau_1 I_H + [\gamma_2 + \delta_2 + \tau_6 + \mu_H]I_{HT} \\ v_5 & = -\tau_6 I_{HT} + [\gamma_5 + \delta_6 + \mu_H]I_{Hi} \\ v_6 & = -[\gamma_3 + \delta_3 + \tau_2 + \mu_P]I_P \\ v_7 & = -\tau_2 I_P + [\gamma_4 + \delta_4 + \mu_P]I_{PT} \\ v_8 & = [\mu_B + \delta_5]I_B \end{pmatrix} \tag{30}$$

Therefore, we have F to be:

$$\begin{aligned} &\frac{\beta_1 n_0 S_H}{N_H} + \frac{\beta_1 n_0 S_{HW}}{N_H} = a_1, \\ &\frac{\beta_1 n_1 S_H}{N_H} + \frac{\beta_1 n_1 S_{HW}}{N_H} = a_2, \\ &\frac{\beta_1 n_2 S_H}{N_H} + \frac{\beta_1 n_2 S_{HW}}{N_H} = a_3, \\ &\frac{\beta_2 n_1 S_P}{N_P} = a_4, \\ &\frac{\beta_2 n_2 S_P}{N_P} = a_5, \\ &\frac{\beta_3 n_2 S_B}{N_B} = a_6. \end{aligned} \tag{31}$$

$$F = \begin{pmatrix} 0 & 0 & a_1 & 0 & 0 & a_2 & 0 & a_3 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_1 & 0 & 0 & a_4 & 0 & a_5 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_1 & 0 & 0 & 0 & 0 & a_6 \end{pmatrix} \tag{32}$$

We have V to be:

$$\begin{aligned} &[\tau_4 + \tau_3 + \mu_H] = b_1, \\ &[\tau_5 + \gamma_1 + \mu_H] = b_2, \\ &[\gamma_1 + \delta_1 + \tau_1 + \mu_H] = b_3, \\ &[\gamma_2 + \delta_2 + \tau_6 + \mu_H] = b_4, \\ &[\gamma_5 + \delta_6 + \mu_H] = b_5, \\ &[\gamma_3 + \delta_3 + \tau_2 + \mu_P] = b_6, \\ &[\gamma_4 + \delta_4 + \mu_P] = b_7, \\ &[\delta_5 + \mu_B] = b_8 \end{aligned} \tag{33}$$

$$F = \begin{pmatrix} b_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_1 & 0 & 0 & a_4 & 0 & a_5 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_1 & 0 & 0 & 0 & 0 & a_6 \end{pmatrix} \tag{34}$$

Having evaluated equations 31 and 33 at DFE, we now have V^{-1} and F, we can then compute the Next Generation Matrix (NGM) for the Nipah virus disease model:

$$FV^{-1} = \begin{pmatrix} d_1 & d_2 & d_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_1 & 0 & 0 & d_5 & 0 & d_7 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & d_7 \end{pmatrix} \tag{35}$$

we then have:

$$R_0 = \frac{\beta_1 n_0 \left([\Lambda_H \mu_H] + \Lambda_H W[k+v+\mu_H] + k\Lambda_H\right)}{[\tau_4+\tau_3+\mu_H][\tau_5+\gamma_5+\mu_H][\gamma_1+\delta_1+\tau_1+\mu_H]} \\ \mu_H[k+v+\mu_H]. \tag{36}$$

□

From equation 29 of the basic reproduction number subsection, we found out that f_1 which represents the proportional rate of susceptible humans likely to consume Nipah virus-contaminated food items (such as palm wine) and get infected. This behavior significantly impacts the transmission dynamics of the virus. As f_1 increases due to higher contact rates or transmission coefficients, the basic reproduction number R_0 will also increase, indicating a higher potential for the virus to spread within the population.

3.6. Local asymptotical stability Disease free equilibrium (DFE).

Here, we try to visualize the need for local stability analysis as it provides valuable information about the potential effectiveness of various control strategies based on the basic dynamics of our Nipah virus disease model of equations 1 to 16.

Local stability analysis of the disease-free equilibrium point A^0 is obtained by the variational matrix of the model (1) to (16) corresponding to the point A^0 Escaffre et. al [7], Mbah et. al [10], Goswami, and Hategekimana [8].

Theorem 3. *Given a matrix $J(A^0)$ and that all of the eigenvalues have negative real components, then we say that the virus-free equilibrium at the point A^0 is locally asymptotically stable; otherwise, it is unstable.*

Proof. We linearize the system of equations 1 to 16 around the equilibrium point (DFE) to form the Jacobian Matrix.

(37)

$$\begin{pmatrix}
D_1 & 0 & 0 & 0 & D_2 & 0 & 0 & 0 & 0 & 0 & D_3 & 0 & 0 & 0 & 0 & D_4 \\
v & -\mu_H & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
k & 0 & -\mu_H & 0 & 0 & D_5 & 0 & 0 & 0 & 0 & 0 & D_6 & 0 & 0 & 0 & D_7 \\
0 & 0 & 0 & D_8 & 0 & D_9 & 0 & 0 & 0 & 0 & 0 & D_{10} & 0 & 0 & 0 & D_{11} \\
0 & 0 & 0 & \tau_4 & D_{12} & 0 & 0 & 0 & d_1 & d_2 & d_3 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \tau_3 & \tau_5 & D_{13} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \tau_1 & D_{14} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \tau_6 & D_{15} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \theta_1 & \theta_2 & \gamma_1 & \gamma_2 & \gamma_5 & \mu_H & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & D_{16} & 0 & D_{17} & 0 & 0 & 0 & D_{18} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -v_1 & -\mu_P & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & D_{19} & 0 & 0 & 0 & D_{20} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \tau_2 & D_{21} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_3 & \gamma_4 & \mu_P & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu_B & D_{22} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & D_{23}
\end{pmatrix}$$

Where:

(38)

$$\begin{aligned}
D_1 &= -(k+v+\mu_H); \\
D_2 &= -\frac{\beta_1 n_0 \Lambda_H}{N_H(k+v+\mu_H)}; \\
D_3 &= -\frac{\beta_1 n_1 \Lambda_H}{N_H(k+v+\mu_H)}; \\
D_4 &= -\frac{\beta_1 n_2 \Lambda_H}{N_H(k+v+\mu_H)};
\end{aligned}$$

$$D_5 = -\frac{\beta_1 n_0 \Lambda_H W(k+v+\mu_H)+k\Lambda_H}{N_H(\mu_H(k+v+\mu_H))};$$

$$D_6 = -\frac{\beta_1 n_1 \Lambda_H W(k+v+\mu_H)+k\Lambda_H}{N_H(\mu_H(k+v+\mu_H))};$$

$$D_7 = -\frac{\beta_1 n_2 \Lambda_H W(k+v+\mu_H)+k\Lambda_H}{N_H(\mu_H(k+v+\mu_H))};$$

$$D_8 = -(\tau_4+\mu_H+\tau_3);$$

$$D_9 = \frac{\beta_1 n_0 \Lambda_H}{N_H}\left(\frac{\Lambda_H}{(k+v+\mu_H)}+\frac{\Lambda_H W(k+v+\mu_H)+k\Lambda_H}{(\mu_H(k+v+\mu_H))}\right);$$

$$D_{10} = \frac{\beta_1 n_1 \Lambda_H}{N_H(k+v+\mu_H)};$$

$$D_{11} = \frac{\beta_1 n_2 \Lambda_H}{N_H(k+v+\mu_H)};$$

$$D_{12} = -(\tau_5+\mu_H);$$

$$D_{13} = -[\gamma_1+\tau_1+\mu_H+\delta_1];$$

$$D_{14} = -[\gamma_2+\tau_6+\mu_H+\delta_2];$$

$$D_{15} = -[\gamma_5+\mu_H+\delta_6];$$

$$D_{16} = -[v_1+\mu_P];$$

$$D_{17} = -\frac{\beta_2 n_1 \Lambda_P}{N_P \mu_P};$$

$$D_{18} = -\frac{\beta_2 n_2 \Lambda_P}{N_P \mu_P};$$

$$D_{19} = \frac{\beta_2 n_1 \Lambda_P}{N_P \mu_P} - [\gamma_3+\tau_2+\mu_P+\delta_3];$$

$$D_{20} = \frac{\beta_2 n_2 \Lambda_P}{N_P \mu_P};$$

$$D_{21} = -[\gamma_4+\mu_P+\delta_4];$$

$$D_{22} = -\frac{\beta_3 n_2 \Lambda_B}{N_B \mu_B};$$

$$D_{23} = \frac{\beta_3 n_2 \Lambda_B}{N_B \mu_B} - [\mu_B+\delta_5].$$

The characteristic equation for the system is given by

$|J-\theta_i I|=0$, where $\theta_i = \theta_1, \theta_2, \ldots, \theta_{23}$ are the eigenvalues and (I) is the identity matrix for our

NiV model. Below is the

computed eigenvalue results from the Jacobian of our NiV model using **Maple-2021**:

$$\theta_1 = -(k+v+\mu_H),$$

$$\theta_2 = -\mu_H,$$

$$\theta_3 = -[\theta_1+\tau_4+\mu_H+\tau_3],$$

$$\theta_4 = -[\gamma_5+\tau_5+\mu_H],$$

$$\theta_5 = -[\gamma_1+\tau_1+\mu_H+\delta_1],$$

$$\theta_6 = -[\gamma_2+\tau_6+\mu_H+\delta_2],$$

$$\theta_7 = -[\gamma_6+\mu_H+\delta_6],$$

$$\theta_8 = -[v_1+\mu_P],$$

$$\theta_9 = -\mu_P,$$

$$\theta_{10} = \left(\frac{\beta_2 n_1 \Lambda_P}{N_P \mu_P}\right) - [\gamma_3+\tau_2+\mu_P+\delta_3],$$

and θ_{10} is negative as long as $\left(\frac{\beta_2 n_1 \Lambda_P}{N_P \mu_P}\right) < [\gamma_3+\tau_2+\mu_P+\delta_3]$,

$$\theta_{11} = -[\gamma_4+\mu_P+\delta_4],$$

$$\theta_{12} = -\mu_B,$$

$\theta_{13} = \left(\frac{\beta_3 n_2 \Lambda_B}{N_B \mu_B}\right) - [\mu_B+\delta_5]$ and θ_{13} is negative, if and only if $\left(\frac{\beta_3 n_2 \Lambda_B}{N_B \mu_B}\right) < [\mu_B+\delta_5]$.

Since all our parameters ($[\beta_1, \gamma_1, \gamma_2, \tau_1, \tau_3, \tau_4, \tau_5, \mu_H, \delta_1, \delta_2, \theta_1, \theta_2, \delta_6, \beta_2, \gamma_3, \gamma_4, \gamma_5, \gamma_6, \tau_2, \mu_P, \delta_3, \delta_4, \beta_3, \delta_5, \mu_B] > 0$), therefore, we say that the disease-free equilibrium (DFE) of the general system is locally asymptotically stable. This result mathematically implies that the disease can be wiped out of the population with respect to the initial condition (population). □

3.7. Local Stability for Nipah Endemic State.

In the disease-endemic state, we investigated the existence of forward or backward bifurcation by using the center manifold theorem postulated by Castillo-Chavez, Song [32].

Then we redefine the equations 1- 16 as:

$$f_1 = \Lambda_H + \gamma_5 x_6 - v x_1 - k x_1 - \frac{x_1 \beta_1 (n_0 x_6 + n_1 x_{12} + n_2 x_{16})}{N_H} - \mu_H x_1$$

$$f_2 = \Lambda_V + v x_1 - \mu_H x_2$$

$$f_3 = \Lambda_{HW} + k x_1 - \frac{x_3 \beta_1 (n_0 x_6 + n_1 x_{12} + n_2 x_{16})}{N_H} - \mu_H x_3$$

$$f_4 = \frac{x_1 \beta_1 (n_0 x_6 + n_1 x_{12} + n_2 x_{16})}{N_H} + \frac{x_3 \beta_1 (n_0 x_6 + n_1 x_{12} + n_2 x_{16})}{N_H} - P_1 x_4$$

$$f_5 = x_4 \tau_4 - P_2 x_5$$

$$f_6 = x_4 \tau_3 + x_5 \tau_5 - P_3 x_6$$

$$f_7 = x_6 \tau_1 - P_4 x_7$$

$$f_8 = x_7 \tau_6 - P_5 x_8$$

$$f_9 = x_7 \gamma_2 + x_8 \gamma_6 - \mu_H x_9$$

$$f_{10} = \Lambda_P - \frac{x_{10} \beta_2 (n_1 x_{12} + n_2 x_{16})}{N_P} - \mu_P x_{10}$$

$$f_{11} = \Lambda_{VP} + v_1 x_{10} - \mu_P x_{11}$$

$$f_{12} = \frac{x_{10} \beta_2 (n_1 x_{12} + n_2 x_{16})}{N_P} - P_6 x_{12}$$

$$f_{13} = x_{12} \tau_2 - P_7 x_{13}$$

$$f_{14} = x_{12} \gamma_3 + x_{13} \gamma_4 - \mu_P x_{14}$$

$$f_{15} = \Lambda_B - \frac{x_{15} \beta_3 n_2 x_{16}}{N_B} - \mu_B x_{15}$$

$$f_{16} = \frac{x_{15} \beta_3 n_2 x_{16}}{N_B} - x_{16} P_8$$

Thus, we applied the central manifold theorem to get the bifurcation coefficients $a < 0$ and $b < 0$. Mbah et. al [10] This indicates that the endemic equilibrium state is locally asymptotically stable while also exhibiting forward bifurcation. We can now proceed to study the global stability.

3.8. Global stability of the NiV transmission dynamics model equilibrium states.

In this section, we try to investigate how the system as a whole behaves, rather than just near the equilibrium points, which was the case when we analyzed the local stability of the system.

As in the local stability, in the analysis of the global stability of the system, we studied the equations 1- 16 for the disease-free equilibrium and also the endemic equilibrium cases.

3.8.1. *Global stability of the disease-free equilibrium state.* To carry out this study, we use the LaSalle's invariant principle to define a Lyapunov's function:

$$\begin{aligned}
V(S_H^0, S_V^0, S_{HW}^0, E_H^0, E_{HT}^0, I_H^0, I_{HT}^0, I_{Hi}^0, R_H^0, S_P^0, S_{PV}^0, I_P^0, I_{PT}^0, R_P^0, S_B^0, I_B^0) = \\
(S_H - S_H^0 - S_H^0 \ln \frac{S_H^0}{S_H}) + (S_V - S_V^0 - S_V^0 \ln \frac{S_V^0}{S_V}) + \\
(S_{HW} - S_{HW}^0 - S_{HW}^0 \ln \frac{S_{HW}^0}{S_{HW}}) + (E_H - E_H^0 - E_H^0 \ln \frac{E_H^0}{E_H}) + \\
(E_{HT} - E_{HT}^0 - E_{HT}^0 \ln \frac{E_{HT}^0}{E_{HT}}) + (I_H - I_H^0 - I_H^0 \ln \frac{I_H^0}{I_H}) + \\
(I_{HT} - I_{HT}^0 - I_{HT}^0 \ln \frac{I_{HT}^0}{I_{HT}}) + (I_{Hi} - I_{Hi}^0 - I_{Hi}^0 \ln \frac{I_{Hi}^0}{I_{Hi}}) + \\
(R_H - R_H^0 - R_H^0 \ln \frac{R_H^0}{R_H}) + (S_P - S_P^0 - S_P^0 \ln \frac{S_P^0}{S_P}) + \\
(S_{PV} - S_{PV}^0 - S_{PV}^0 \ln \frac{S_{PV}^0}{S_{PV}}) + (I_P - I_P^0 - I_P^0 \ln \frac{I_P^0}{I_P}) + \\
(I_{PT} - I_{PT}^0 - I_{PT}^0 \ln \frac{I_{PT}^0}{I_{PT}}) + (R_P - R_P^0 - R_P^0 \ln \frac{R_P^0}{R_P}) + \\
(S_B - S_B^0 - S_B^0 \ln \frac{S_B^0}{S_B}) + (I_B - I_B^0 - I_B^0 \ln \frac{I_B^0}{I_B})
\end{aligned}$$

But at Nipah virus disease-free equilibrium (NDFE):

$$A^0 = E_H^0 = E_{HT}^0 = I_H^0 = I_{HT}^0 = I_{Hi}^0 = R_H^0 = I_P^0 = I_{PT}^0 = R_P^0 = I_B^0 = 0$$

Then, evaluate the derivative to get:

$$\begin{aligned}
\frac{dV}{dt} = &\left(\frac{S_H + S_H^0}{S_H}\right)\frac{dS_H}{dt} + \left(\frac{S_V + S_V^0}{S_V}\right)\frac{dS_V}{dt} + \left(\frac{S_{HW} + S_{HW}^0}{S_{HW}}\right)\frac{dS_{HW}}{dt} \\
&+ \left(\frac{E_H + E_H^0}{E_H}\right)\frac{dE_H}{dt} + \left(\frac{E_{HT} + E_{HT}^0}{E_{HT}}\right)\frac{dE_{HT}}{dt} + \left(\frac{I_H + I_H^0}{I_H}\right)\frac{dI_H}{dt}
\end{aligned} \tag{39}$$

$$+\left(\frac{I_{HT}+I_{HT}^0}{I_{HT}}\right)\frac{dI_{HT}}{dt}+\left(\frac{I_{Hi}+I_{Hi}^0}{I_{Hi}}\right)\frac{dI_{Hi}}{dt}+\left(\frac{R_H+R_H^0}{R_H}\right)\frac{dR_H}{dt}$$
$$+\left(\frac{S_P+S_P^0}{S_P}\right)\frac{dS_P}{dt}+\left(\frac{S_{PV}+S_{PV}^0}{S_{PV}}\right)\frac{dS_{PV}}{dt}+\left(\frac{I_P+I_P^0}{I_P}\right)\frac{dI_P}{dt}$$
$$+\left(\frac{I_{PT}+I_{PT}^0}{I_{PT}}\right)\frac{dI_{PT}}{dt}+\left(\frac{R_P+R_P^0}{R_P}\right)\frac{dR_P}{dt}+\left(\frac{S_B+S_B^0}{S_B}\right)\frac{dS_B}{dt}$$
$$+\left(\frac{I_B+I_B^0}{I_B}\right)\frac{dI_B}{dt}$$

Substituting for the derivatives in equation 39, using equations 1- 16 and simplifying, we then obtain:

$$\begin{aligned}\frac{dV}{dt}=&\left(\frac{S_H+S_H^0}{S_H}\right)\Lambda_H+\gamma_5(E_{HT}+E_{HT}^0)-\left(\frac{S_H+S_H^0}{S_H}\right)^2(\lambda_H+k+(v+\mu_H))+\\
&\left(\frac{S_V+S_V^0}{S_V}\right)(\Lambda_V+v(S_H+S_H^0))-\left(\frac{S_V+S_V^0}{S_V}\right)^2\mu_H+\\
&\left(\frac{S_{HW}+S_{HW}^0}{S_{HW}}\right)(\Lambda_{HW}+k(S_H+S_H^0))-\left(\frac{S_{HW}+S_{HW}^0}{S_{HW}}\right)^2(\lambda_H+\mu_H)+\\
&\left(\frac{E_H+E_H^0}{E_H}\right)\lambda_H((S_H+S_H^0)+(S_{HW}+S_{HW}^0))-\left(\frac{E_H+E_H^0}{E_H}\right)^2(\tau_4+\tau_3+\mu_H)+\\
&\left(\frac{E_{HT}+E_{HT}^0}{E_{HT}}\right)\tau_4(E_H+E_H^0)-\left(\frac{E_{HT}+E_{HT}^0}{E_{HT}}\right)^2(\tau_5+\gamma_5+\mu_H)+\\
&\left(\frac{I_H+I_H^0}{I_H}\right)(\tau_3(E_H+E_H^0)+\tau_5(E_{HT}+E_{HT}^0))-\left(\frac{I_H+I_H^0}{I_H}\right)^2[\gamma_1+\delta_1+\tau_1+\mu_H]+\\
&\left(\frac{I_{HT}+I_{HT}^0}{I_{HT}}\right)\tau_1(I_H+I_H^0)-\left(\frac{I_{HT}+I_{HT}^0}{I_{HT}}\right)^2[\gamma_2+\delta_2+\tau_6+\mu_H]+\\
&\left(\frac{I_{Hi}+I_{Hi}^0}{I_{Hi}}\right)\tau_6(I_{HT}+I_{HT}^0)-\left(\frac{I_{Hi}+I_{Hi}^0}{I_{Hi}}\right)^2[\gamma_6+\delta_6+\mu_H]+\\
&\left(\frac{R_H+R_H^0}{R_H}\right)[\gamma_2(I_{HT}+I_{HT}^0)+\gamma_6(I_{Hi}+I_{Hi}^0)]-\left(\frac{R_H+R_H^0}{R_H}\right)^2\mu_H+\\
&\left(\frac{S_P+S_P^0}{S_P}\right)\Lambda_P-\left(\frac{S_P+S_P^0}{S_P}\right)^2(\lambda_P+(v_1+\mu_P))+\\
&\left(\frac{S_{PV}+S_{PV}^0}{S_{PV}}\right)(\Lambda_{PV}+v_1(S_P+S_P^0))-\left(\frac{S_{PV}+S_{PV}^0}{S_{PV}}\right)^2\mu_P+\\
&\left(\frac{I_P+I_P^0}{I_P}\right)\lambda_P(S_P+S_P^0)-\left(\frac{I_P+I_P^0}{I_P}\right)^2[\gamma_3+\delta_3+\tau_2+\mu_P]+\end{aligned}\tag{40}$$

$$\left(\frac{I_{PT}+I_{PT}^0}{I_{PT}}\right)\tau_2(I_P+I_P^0)-\left(\frac{I_{PT}+I_{PT}^0}{I_{PT}}\right)^2[\gamma_4+\delta_4+\mu_P]+$$
$$\left(\frac{R_P+R_P^0}{R_P}\right)(\gamma_3(I_P+I_P^0)+\gamma_4(I_{PT}+I_{PT}^0))-\left(\frac{R_P+R_P^0}{R_P}\right)^2\mu_P+$$
$$\left(\frac{S_B+S_B^0}{S_B}\right)\Lambda_B-\left(\frac{S_B+S_B^0}{S_B}\right)^2(\lambda_B+\mu_B)+$$
$$\left(\frac{I_B+I_B^0}{I_B}\right)\lambda_B(S_B+S_B^0)-\left(\frac{I_B+I_B^0}{I_B}\right)^2[\delta_5+\mu_B]$$

Next, collect all the positive and negative terms from equation 40 to give:

$$\frac{dV}{dt}=T-Z$$

Where:

$$T=\left(1+\frac{S_H^0}{S_H}\right)(\Lambda_H+\theta_1E_H+\theta_2E_{HT})+\left(1+\frac{S_V^0}{S_V}\right)(\Lambda_V+v(S_H+S_H^0))+$$
$$\left(1+\frac{S_{HW}^0}{S_{HW}}\right)(\Lambda_{HW}+k(S_H+S_H^0))+\left(1+\frac{E_H^0}{E_H}\right)\lambda_H((S_H+S_H^0)+(S_{HW}+S_{HW}^0))+$$
$$\left(1+\frac{E_{HT}^0}{E_{HT}}\right)\tau_4(E_H+E_H^0)+\left(1+\frac{I_H^0}{I_H}\right)(\tau_3(E_H+E_H^0)+\tau_5(E_{HT}+E_{HT}^0))+$$
$$\left(1+\frac{I_{HT}^0}{I_{HT}}\right)\tau_1(I_H+I_H^0)+\left(1+\frac{I_{Hi}^0}{I_{Hi}}\right)\tau_6(I_{HT}+I_{HT}^0)+$$
$$\left(1+\frac{R_H^0}{R_H}\right)[\theta_1(E_H+E_H^0)+\theta_2(E_{HT}+E_{HT}^0)+\gamma_1(I_H+I_H^0)+\gamma_2(I_{HT}+I_{HT}^0)+\gamma_5(I_{Hi}+I_{Hi}^0)]+$$
$$\left(1+\frac{S_P^0}{S_P}\right)\Lambda_P+\left(1+\frac{S_{PV}^0}{S_{PV}}\right)(\Lambda_{PV}+v_1(S_P+S_P^0))+$$
$$\left(1+\frac{I_P^0}{I_P}\right)\lambda_P(S_P+S_P^0)+\left(1+\frac{I_{PT}^0}{I_{PT}}\right)\tau_2(I_P+I_P^0)+\left(1+\frac{R_P^0}{R_P}\right)(\gamma_3(I_P+I_P^0)+\gamma_4(I_{PT}+I_{PT}^0))+$$
$$\left(1+\frac{S_B^0}{S_B}\right)\Lambda_B+\left(1+\frac{I_B^0}{I_B}\right)\lambda_B(S_B+S_B^0)$$

$$Z=-\left(\left(\frac{S_H+S_H^0}{S_H}\right)^2(\lambda_H+k+(v+\mu_H))+\left(\frac{S_V+S_V^0}{S_V}\right)^2\mu_H+\left(\frac{S_{HW}+S_{HW}^0}{S_{HW}}\right)^2(\lambda_H+\mu_H)\right.$$
$$+\left(\frac{E_H+E_H^0}{E_H}\right)^2(\theta_1+\tau_4+\tau_3+\mu_H)+\left(\frac{E_{HT}+E_{HT}^0}{E_{HT}}\right)^2(\tau_5+\theta_2+\mu_H)+\left(\frac{I_H+I_H^0}{I_H}\right)^2[\delta_1+\tau_1+\mu_H]$$
$$+\left(\frac{I_{HT}+I_{HT}^0}{I_{HT}}\right)^2[\gamma_2+\delta_2+\tau_6+\mu_H]+\left(\frac{I_{Hi}+I_{Hi}^0}{I_{Hi}}\right)^2[\gamma_5+\delta_6+\mu_H]+\left(\frac{R_H+R_H^0}{R_H}\right)^2\mu_H$$
$$\left.+\left(\frac{S_P+S_P^0}{S_P}\right)^2(\lambda_P+(v_1+\mu_P))+\left(\frac{S_{PV}+S_{PV}^0}{S_{PV}}\right)^2\mu_P+\left(\frac{I_P+I_P^0}{I_P}\right)^2[\gamma_3+\delta_3+\tau_2+\mu_P]\right.$$

$$+\left(\frac{I_{PT}+I_{PT}^{0}}{I_{PT}}\right)^{2}[\gamma_4+\delta_4+\mu_P]+\left(\frac{R_P+R_P^{0}}{R_P}\right)^{2}\mu_P+\left(\frac{S_B+S_B^{0}}{S_B}\right)^{2}(\lambda_B+\mu_B)+\left(\frac{I_B+I_B^{0}}{I_B}\right)^{2}[\delta_5+\mu_B]\Bigg)$$

If $T<Z$, then $\frac{dV}{dt}$ will be negative definite along the sub-system's solution route.

This indicates that the NiV disease-free equilibrium will be globally asymptotically stable in Ω_{I1}.

3.8.2. *Global stability of the endemic equilibrium state.* To establish the global stability of the endemic equilibrium state of the disease within the population, we use the same method as before, but now with the values of the variables at the equilibrium state. We consider the following Lyapunov function defined as:

$$\begin{aligned}
&\Lambda(S_H^*,S_V^*,S_{HW}^*,E_H^*,E_{HT}^*,I_H^*,I_{HT}^*,I_{Hi}^*,R_H^*,S_P^*,S_{PV}^*,I_P^*,I_{PT}^*,R_P^*,S_B^*,I_B^*)=\\
&(S_H-S_H^*-S_H^*\ln\left(\frac{S_H^*}{S_H}\right))+(S_V-S_V^*-S_V^*\ln\left(\frac{S_V^*}{S_V}\right))+(S_{HW}-S_{HW}^*-S_{HW}^*\ln\left(\frac{S_{HW}^*}{S_{HW}}\right))\\
&+(E_H-E_H^*-E_H^*\ln\left(\frac{E_H^*}{E_H}\right))+(E_{HT}-E_{HT}^*-E_{HT}^*\ln\left(\frac{E_{HT}^*}{E_{HT}}\right))+(I_H-I_H^*-I_H^*\ln\left(\frac{I_H^*}{I_H}\right))\\
&+(I_{HT}-I_{HT}^*-I_{HT}^*\ln\left(\frac{I_{HT}^*}{I_{HT}}\right))+(I_{Hi}-I_{Hi}^*-I_{Hi}^*\ln\left(\frac{I_{Hi}^*}{I_{Hi}}\right))+(R_H-R_H^*-R_H^*\ln\left(\frac{R_H^*}{R_H}\right))\\
&+(S_P-S_P^*-S_P^*\ln\left(\frac{S_P^*}{S_P}\right))+(S_{PV}-S_{PV}^*-S_{PV}^*\ln\left(\frac{S_{PV}^*}{S_{PV}}\right))+(I_P-I_P^*-I_P^*\ln\left(\frac{I_P^*}{I_P}\right))\\
&+(I_{PT}-I_{PT}^*-I_{PT}^*\ln\left(\frac{I_{PT}^*}{I_{PT}}\right))+(R_P-R_P^*-R_P^*\ln\left(\frac{R_P^*}{R_P}\right))+(S_B-S_B^*-S_B^*\ln\left(\frac{S_B^*}{S_B}\right))\\
&+(I_B-I_B^*-I_B^*\ln\left(\frac{I_B^*}{I_B}\right))
\end{aligned}$$

Then, evaluate the derivative to get:

$$\begin{aligned}
\frac{d\Lambda}{dt}=&\left(\frac{S_H+S_H^*}{S_H}\right)\frac{dS_H}{dt}+\left(\frac{S_V+S_V^*}{S_V}\right)\frac{dS_V}{dt}+\left(\frac{S_HW+S_HW^*}{S_HW}\right)\frac{dS_HW}{dt}\\
&+\left(\frac{E_H+E_H^*}{E_H}\right)\frac{dE_H}{dt}+\left(\frac{E_HT+E_HT^*}{E_HT}\right)\frac{dE_HT}{dt}+\left(\frac{I_H+I_H^*}{I_H}\right)\frac{dI_H}{dt}\\
&+\left(\frac{I_HT+I_HT^*}{I_HT}\right)\frac{dI_HT}{dt}+\left(\frac{I_Hi+I_Hi^*}{I_Hi}\right)\frac{dI_Hi}{dt}+\left(\frac{R_H+R_H^*}{R_H}\right)\frac{dR_H}{dt}
\end{aligned}$$

$$+\left(\frac{S_P+S_P^*}{S_P}\right)\frac{dS_P}{dt}+\left(\frac{S_PV+S_PV^*}{S_PV}\right)\frac{dS_PV}{dt}+\left(\frac{I_P+I_P^*}{I_P}\right)\frac{dI_P}{dt}$$
$$+\left(\frac{I_PT+I_PT^*}{I_PT}\right)\frac{dI_PT}{dt}+\left(\frac{R_P+R_P^*}{R_P}\right)\frac{dR_P}{dt}+\left(\frac{S_B+S_B^*}{S_B}\right)\frac{dS_B}{dt}$$
$$+\left(\frac{I_B+I_B^*}{I_B}\right)\frac{dI_B}{dt}$$

Substituting these derivatives into equations 1- 16

$$\frac{d\Lambda}{dt}=\left(\frac{S_H+S_H^*}{S_H}\right)\Lambda_H-\left(\frac{S_H+S_H^*}{S_H}\right)^2(\lambda_H+k+v+\mu_H)+\left(\frac{S_V+S_V^*}{S_V}\right)(\Lambda_V+v(S_H+S_H^*))-\left(\frac{S_V+S_V^*}{S_V}\right)^2\mu_H$$
$$+\left(\frac{S_HW+S_HW^*}{S_HW}\right)(\Lambda_HW+k(S_H+S_H^*))-\left(\frac{S_HW+S_HW^*}{S_HW}\right)^2(\lambda_H+\mu_H)$$
$$+\left(\frac{E_H+E_H^*}{E_H}\right)\lambda_H(S_H+S_H^*+S_HW+S_HW^*)-\left(\frac{E_H+E_H^*}{E_H}\right)^2(\tau_4+\tau_3+\mu_H)$$
$$+\left(\frac{E_HT+E_HT^*}{E_HT}\right)\tau_4(E_H+E_H^*)-\left(\frac{E_HT+E_HT^*}{E_HT}\right)^2(\tau_5+\gamma_5+\mu_H)$$
$$+\left(\frac{I_H+I_H^*}{I_H}\right)(\tau_3(E_H+E_H^*)+\tau_5(E_HT+E_HT^*))-\left(\frac{I_H+I_H^*}{I_H}\right)^2(\gamma_1+\delta_1+\tau_1+\mu_H)$$
$$+\left(\frac{I_HT+I_HT^*}{I_HT}\right)\tau_1(I_H+I_H^*)-\left(\frac{I_HT+I_HT^*}{I_HT}\right)^2(\gamma_2+\delta_2+\tau_6+\mu_H)$$
$$+\left(\frac{I_Hi+I_Hi^*}{I_Hi}\right)\tau_6(I_HT+I_HT^*)-\left(\frac{I_Hi+I_Hi^*}{I_Hi}\right)^2(\gamma_6+\delta_6+\mu_H)$$
$$+\left(\frac{R_H+R_H^*}{R_H}\right)(\gamma_5(E_HT+E_HT^*)+\gamma_2(I_HT+I_HT^*)+\gamma_6(I_Hi+I_Hi^*))-\left(\frac{R_H+R_H^*}{R_H}\right)^2\mu_H$$
$$+\left(\frac{S_P+S_P^*}{S_P}\right)\Lambda_P-\left(\frac{S_P+S_P^*}{S_P}\right)^2(\lambda_P+v_1+\mu_P)$$
$$+\left(\frac{S_PV+S_PV^*}{S_PV}\right)(\Lambda_PV+v_1(S_P+S_P^*))-\left(\frac{S_PV+S_PV^*}{S_PV}\right)^2\mu_P$$
$$+\left(\frac{I_P+I_P^*}{I_P}\right)\lambda_P(S_P+S_P^*)-\left(\frac{I_P+I_P^*}{I_P}\right)^2(\gamma_3+\delta_3+\tau_2+\mu_P)$$
$$+\left(\frac{I_PT+I_PT^*}{I_PT}\right)\tau_2(I_P+I_P^*)-\left(\frac{I_PT+I_PT^*}{I_PT}\right)^2(\gamma_4+\delta_4+\mu_P)$$
$$+\left(\frac{R_P+R_P^*}{R_P}\right)(\gamma_3(I_P+I_P^*)+\gamma_4(I_PT+I_PT^*))-\left(\frac{R_P+R_P^*}{R_P}\right)^2\mu_P$$
$$+\left(\frac{S_B+S_B^*}{S_B}\right)\Lambda_B-\left(\frac{S_B+S_B^*}{S_B}\right)^2(\lambda_B+\mu_B)$$
$$+\left(\frac{I_B+I_B^*}{I_B}\right)\lambda_B(S_B+S_B^*)-\left(\frac{I_B+I_B^*}{I_B}\right)^2(\delta_5+\mu_B)$$

Next, collect all the positive and negative terms, we have the following: $\frac{d\Lambda}{dt} = M - N$

$$
\begin{aligned}
M = &\left(1+\frac{S_H^*}{S_H}\right)(\Lambda_H+\theta_1 E_H+\theta_2 E_H T)+\left(1+\frac{S_V^*}{S_V}\right)(\Lambda_V+\nu(S_H+S_H^*))\\
&+\left(1+\frac{S_H W^*}{S_H W}\right)(\Lambda_H W+k(S_H+S_H^*))+\left(1+\frac{E_H^*}{E_H}\right)\lambda_H(S_H+S_H^*+S_H W+S_H W^*)\\
&+\left(1+\frac{E_H T^*}{E_H T}\right)\tau_4(E_H+E_H^*)+\left(1+\frac{I_H^*}{I_H}\right)(\tau_3(E_H+E_H^*)+\tau_5(E_H T+E_H T^*))\\
&+\left(1+\frac{I_H T^*}{I_H T}\right)\tau_1(I_H+I_H^*)+\left(1+\frac{I_H i^*}{I_H i}\right)\tau_6(I_H T+I_H T^*)\\
&+\left(1+\frac{R_H^*}{R_H}\right)[\theta_1(E_H+E_H^*)+\theta_2(E_H T+E_H T^*)+\gamma_1(I_H+I_H^*)+\gamma_2(I_H T+I_H T^*)+\gamma_5(I_H i+I_H i^*)]\\
&+\left(1+\frac{S_P^*}{S_P}\right)\Lambda_P+\left(1+\frac{S_P V^*}{S_P V}\right)(\Lambda_P V+\nu_1(S_P+S_P^*))\\
&+\left(1+\frac{I_P^*}{I_P}\right)\lambda_P(S_P+S_P^*)+\left(1+\frac{I_P T^*}{I_P T}\right)\tau_2(I_P+I_P^*)\\
&+\left(1+\frac{R_P^*}{R_P}\right)[\gamma_3(I_P+I_P^*)+\gamma_4(I_P T+I_P T^*)]+\left(1+\frac{S_B^*}{S_B}\right)\Lambda_B\\
&+\left(1+\frac{I_B^*}{I_B}\right)\lambda_B(S_B+S_B^*)
\end{aligned}
$$

$$
\begin{aligned}
N = -\Bigg(&\frac{(S_H+S_H^*)^2}{S_H^2}(\lambda_H+k+(\nu+\mu_H))+\frac{(S_V+S_V^*)^2}{S_V^2}\mu_H+\frac{(S_H W+S_H W^*)^2}{S_H W^2}(\lambda_H+\mu_H)\\
&+\frac{(E_H+E_H^*)^2}{E_H^2}(\theta_1+\tau_4+\tau_3+\mu_H)+\frac{(E_H T+E_H T^*)^2}{E_H T^2}(\tau_5+\theta_2+\mu_H)\\
&+\frac{(I_H+I_H^*)^2}{I_H^2}(\delta_1+\tau_1+\mu_H)+\frac{(I_H T+I_H T^*)^2}{I_H T^2}(\gamma_2+\delta_2+\tau_6+\mu_H)\\
&+\frac{(I_H i+I_H i^*)^2}{I_H i^2}(\gamma_5+\delta_6+\mu_H)+\frac{(R_H+R_H^*)^2}{R_H^2}\mu_H\\
&+\frac{(S_P+S_P^*)^2}{S_P^2}(\lambda_P+(\nu_1+\mu_P))+\frac{(S_P V+S_P V^*)^2}{S_P V^2}\mu_P\\
&+\frac{(I_P+I_P^*)^2}{I_P^2}(\gamma_3+\delta_3+\tau_2+\mu_P)+\frac{(I_P T+I_P T^*)^2}{I_P T^2}(\gamma_4+\delta_4+\mu_P)\\
&+\frac{(R_P+R_P^*)^2}{R_P^2}\mu_P+\frac{(S_B+S_B^*)^2}{S_B^2}(\lambda_B+\mu_B)+\frac{(I_B+I_B^*)^2}{I_B^2}(\delta_5+\mu_B)\Bigg)
\end{aligned}
$$

If $M < N$, then $\frac{d\Lambda}{dt}$ will be negative definite along the solution path of the sub-system.

This implies that the endemic equilibrium is globally asymptotically stable in Ω_{I2}.

3.9. Sensitivity Analysis of the General NiV Model.

In this section, we determined how changes in each of the parameters affect the transmission and spread of the disease. In order to achieve this, a sensitivity analysis of the non-inhibitor model is carried out. This is also done in order for us to see the response of the model output (in this case R_0) to parameter(s) variation Yang, Liu [27], Abang et. al [1].

This enables us to ascertain the effect of parameters in our model on the dependent variable. For example, we might want to know if increasing a particular parameter will lead to an increase in the dependent variable or not.

TABLE 1. Numerical values for Variables used in implementation of our analysis

Variables	Numerical Values	Source
$S_H(0)$	1000	Zewdie, Gakkhar [28]
$S_V(0)$	350	(Estimated)
$S_{HW}(0)$	440	(Assumed)
$E_H(0)$	20	Shah et. al [17]
$E_{HT}(0)$	0	(Assumed)
$I_H(0)$	20	Shah et. al [17]
$I_{HT}(0)$	0	(Assumed)
$I_{Hi}(0)$	0	(Assumed)
$R_H(0)$	0	(Assumed)
$S_P(0)$	90	Shah et. al [17]
$I_P(0)$	5	Zewdie, Gakkhar [28]
$I_{PT}(0)$	3	Tyagi et. al [20]
$R_P(0)$	0	(Assumed)
$S_B(0)$	50	Shah et. al [17]
$I_B(0)$	37	Shah et. al [17]

TABLE 2. Numerical values of Parameters adopted for our implementation

Parameters	Numerical Values	Source
Λ_H	20	(Assumed)
Λ_P	4	(Assumed)
Λ_B	2	Shah et. al [17]
N_H	1020	(Assumed)
N_P	95	(Assumed)
N_B	50	Shah et. al [17]
μ_H	0.0000421	Zewdie, Gakkhar [28]
μ_P	0.16	Tyagi et. al [20]
μ_B	0.45	(Estimated)
β_1	0.0002	Tyagi et. al [20]
β_2	0.01	(Assumed)
β_3	0.1	Reynolds, Torremorell, Craft [15]
τ_1	0.0001 or 0.52	Shah et. al [17], Omede et. al [14]
τ_2	0.0002	Shah et. al [17], Wit et. al [23]
τ_3	0.17	(Assumed)
τ_4	0.48	Reynolds, Torremorell, Craft [15]
τ_5	0.81	(Assumed)
τ_6	0.0001	(Estimated)
δ_1	0.002	Shah et. al [17]
δ_2	0.001	(Assumed)
δ_3	0.0015	(Assumed)
δ_4	0.0018	(Assumed)
δ_5	0.75	Zewdie, Gakkhar [28]
δ_6	0.001	Shah et. al [17]
γ_1	0.58	Shah et. al [17], Wigand, Kumel [22]
γ_2	0.72	Shah et. al [17]
γ_3	0.4	Reynolds, Torremorell, Craft [15]
γ_4	0.45	Sultana, Podder [19]
γ_5	0.76	(Assumed)
γ_6	0.46	Shah et. al [17]
n_0	0.000001	Scott et. al [16]
n_1	0.02	(Assumed)
n_2	0.2	(Assumed)
λ_H	0.75	Sultana, Podder [19]
λ_P	0.075	Shah et. al [17]
λ_B	0.07	Mondal, Hanif, Biswas [11]

Our main focus in this section is to perform a sensitivity index on R_0, using the values for variables and parameters in Tables 1 and 2 respectively.

We use the normalized forward-sensitivity index of a variable, v, depends on a parameter, p, which is expressed as:

$$r_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v}$$

In particular, the sensitivity indices of the basic reproduction number, R_0, with respect to the model parameters of interest to be examined Yadav et. al [24], Aja, Omale, Mbah [2], Chinebu et. al [4]. The positive sign of sensitivity indices of R_0 to the model parameters illustrates that an increase (or decrease) in the value of each of the parameters in the model may lead to an increase (or decrease) in R_0 and asymptotically result in the persistence (or elimination) of the disease in the community Obasi, Mbah [12], Barua, Dénes [3], Zheng, Wang, Fu [29]. On the contrary, the negative sign of sensitivity indices of R_0 to the model parameter indicates that an increase (or decrease) in the value of each of the parameters in each case leads to a corresponding decrease (or increase) in R_0 of the model. Hence, with sensitivity analysis, one can gain insight into appropriate intervention strategies to prevent and control the spread of the disease described in the model.

Recall that:

$$R_0 = \frac{\beta_1 n_0(\Lambda_H \mu_H + \Lambda_H W(k+v+\mu_H) + k\Lambda_H)}{(\gamma_5+\tau_4+\tau_3+\mu_H)(\tau_5+\mu_H)(\gamma_1+\delta_1+\tau_1+\mu_H)\mu_H(k+v+\mu_H)}$$

and that $n_0 > 0$.

Using **Maple 2021**, we computed the sensitivity index of a parameter, say β_1, with respect to R_0 as:

$$r_{\beta_1}^{R_0} = \frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{R_0} \tag{41}$$

$$= \frac{\frac{\partial\left(\frac{\beta_1 n_0(\Lambda_H\mu_H+\Lambda_H W(k+v+\mu_H)+k\Lambda_H)}{(\gamma_5+\tau_4+\tau_3+\mu_H)(\tau_5+\mu_H)(\gamma_1+\delta_1+\tau_1+\mu_H)\mu_H(k+v+\mu_H)}\right)}{\partial\beta_1} \times \frac{\beta_1}{\left(\frac{\beta_1 n_0(\Lambda_H\mu_H+\Lambda_H W(k+v+\mu_H)+k\Lambda_H)}{(\gamma_5+\tau_4+\tau_3+\mu_H)(\tau_5+\mu_H)(\gamma_1+\delta_1+\tau_1+\mu_H)\mu_H(k+v+\mu_H)}\right)}}{\left(\frac{\beta_1 n_0(\Lambda_H\mu_H+\Lambda_H W(k+v+\mu_H)+k\Lambda_H)}{(\gamma_5+\tau_4+\tau_3+\mu_H)(\tau_5+\mu_H)(\gamma_1+\delta_1+\tau_1+\mu_H)\mu_H(k+v+\mu_H)}\right)} \tag{42}$$

$$= \left(\frac{n_0(\Lambda_H\mu_H+\Lambda_H W(k+v+\mu_H)+k\Lambda_H)}{(\gamma_5+\tau_4+\tau_3+\mu_H)(\tau_5+\mu_H)(\gamma_1+\delta_1+\tau_1+\mu_H)\mu_H(k+v+\mu_H)}\right)$$

$$\times \left(\frac{(\gamma_5+\tau_4+\tau_3+\mu_H)(\tau_5+\mu_H)(\gamma_1+\delta_1+\tau_1+\mu_H)\mu_H(k+v+\mu_H)}{n_0(\Lambda_H\mu_H+\Lambda_H W(k+v+\mu_H)+k\Lambda_H)} \right)$$

$$=1 \quad \text{(positive)}$$

The sensitivity index of the remaining parameters can be computed in the same way as that of β_1. The sensitivity index for other parameters was computed in the same manner, and the results are displayed in Table 3 below:

TABLE 3. Sensitivity index of R_0 with respect to parameters of the model

Parameters	**Numerical Value**	**Sensitivity Index**
μ_H	0.0000421	-0.998201
τ_1	0.0001	-0.0007234
β_1	0.0002	1.0000000
γ_1	0.58	-0.3365
n_0	0.000001	1.0000000
Λ_H	20	0.5274
δ_1	0.0001	-0.0007234
γ_5	0.76	-0.072
τ_4	0.48	-0.6768
τ_3	0.17	-0.1206
τ_5	0.81	-0.99995
k	0.2	0.4736
v	0.52	-0.3744

4. MODEL SIMULATIONS AND DISCUSSIONS

In this section, we deemed it fit to estimate and adopt some model parameters that were not readily available just for illustrative purposes. We also used some theoretical data in which the sources were strictly acknowledged. The model simulation helped us to have a better understanding of the impact of some important parameters and assumptions on the dynamics and

control of Nipah virus disease. Numerical simulations were performed to support and also illustrate our analytical results.

4.1. Showing parameter impact on our Nipah virus epidemiological model. Here, we checked the mathematical representation of our Nipah virus disease (NiVD) transmission dynamics, by varying parameters to get deeper understanding on how different factors influenced the spread of the NiVD. Also, by evaluating how these changes in parameters like disease transmission rates, morbidity, and mortality affect key variables, policymakers can make more informed decisions about interventions such as vaccination campaigns, social distancing measures, or quarantine strategies.

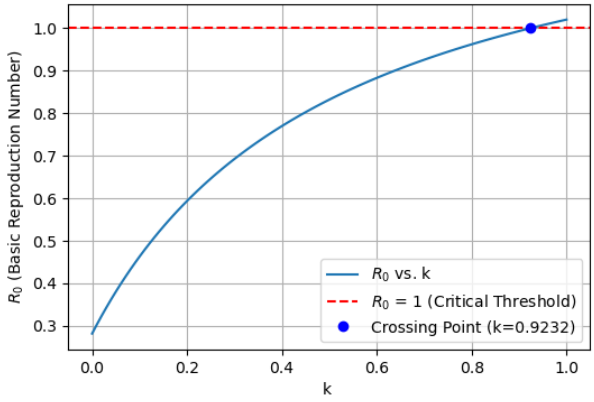

FIGURE 2. Graph of reproduction number (R_0) as a function of k.

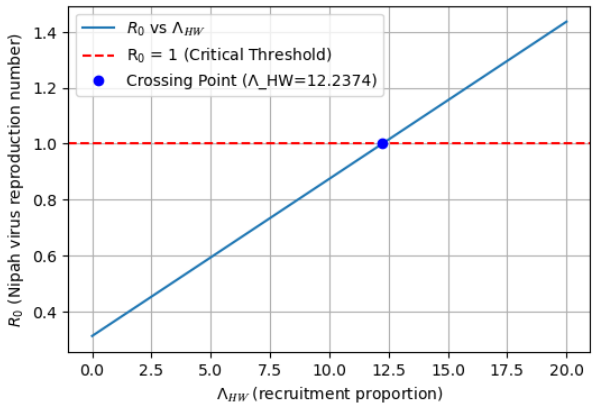

FIGURE 3. Plot of reproduction number (R_0) as a function of Λ_{HW}.

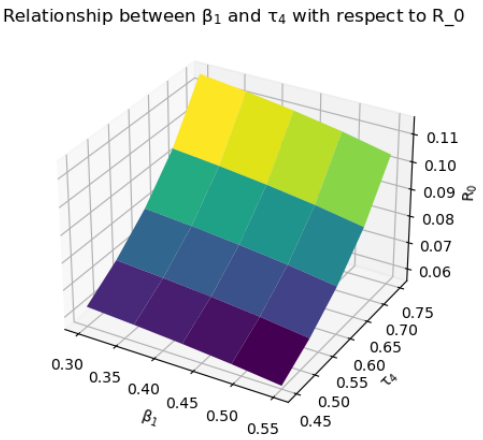

FIGURE 4. Simulation of Relationship between β_1, τ_4 and basic reproduction number (R_0).

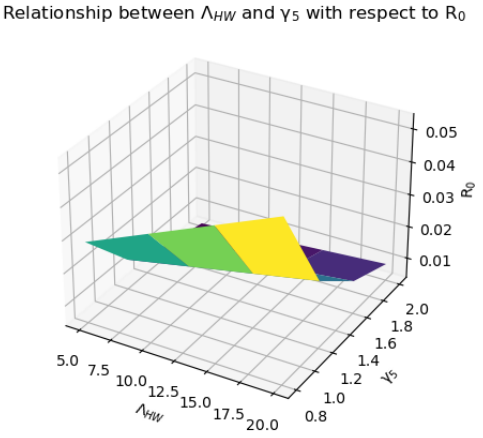

FIGURE 5. Relationship between λ_{HW}, γ_5 and reproduction number (R_0).

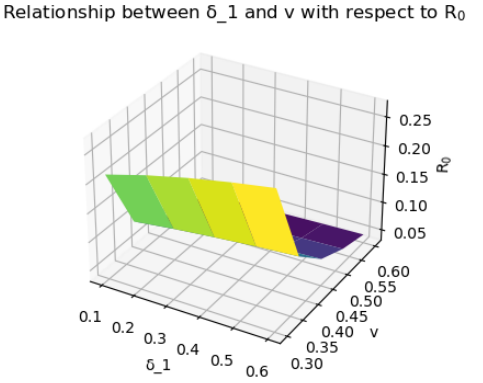

FIGURE 6. Graph of Relationship between δ_1, v and reproduction number (R_0).

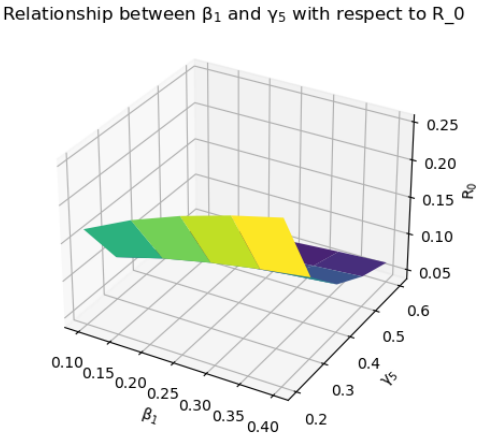

FIGURE 7. Graph of Relationship between β_1, γ_5 and reproduction number (R_0).

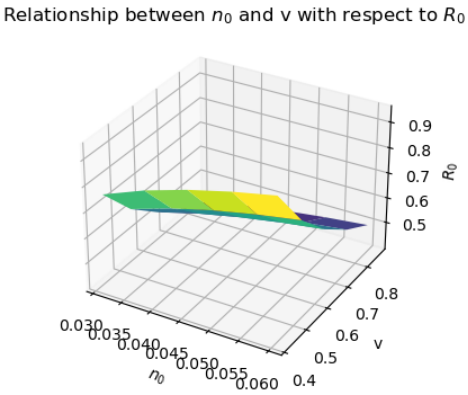

FIGURE 8. Graph of Relationship between n_0, v and reproduction number (R_0).

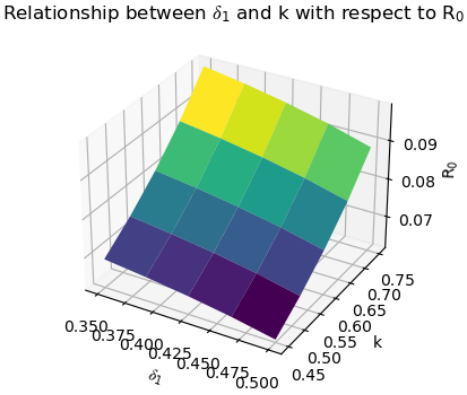

FIGURE 9. Graph of Relationship between δ_1, k and reproduction number (R_0).

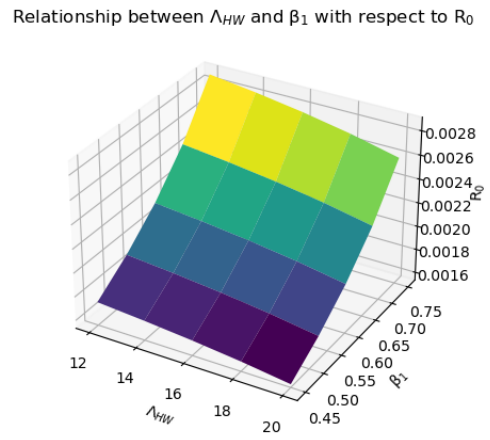

FIGURE 10. Graph of Relationship between Λ_{HW}, β_1 and reproduction number (R_0).

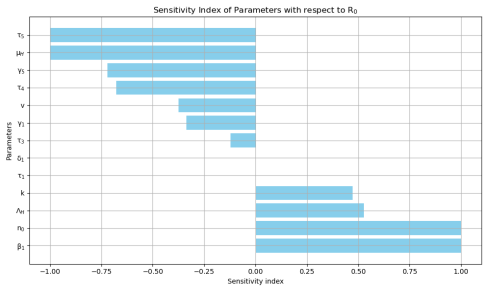

FIGURE 11. Graph of Nipah virus disease sensitivity index analysis.

In Figure 2, we observed that at $k = 0.9232$, R_0 crosses the critical threshold of 1, indicating that the disease's transmission potential (R_0) reaches this critical point. Beyond this, the disease could become a public health concern. In other words, when $R_0 < 1$, the disease is not likely to spread widely and may eventually die out. However, when $R_0 > 1$, the disease is expected to spread and may become an epidemic, requiring significant intervention. Based on the graph and the crossing point at $k = 0.9232$, we conclude:

(1) When the consumption rate of Nipah virus-tainted food items (k) is below 0.9232, the disease is not likely to cause an epidemic $(R_0 < 1)$.

(2) If the consumption rate (k) exceeds 0.9232, the disease may become an epidemic $(R_0 > 1)$.

At $k = 1$, this implies that the population is consuming Nipah virus-tainted food items at the same rate as the disease's transmission rate, significantly increasing the epidemic potential, necessitating strong preventive measures.

In Figure 3, our simulated graph suggests that when the recruitment proportion of susceptible individuals likely to consume tainted food items (Λ_{HW}) reaches approximately 12.2374, the disease's transmission potential (R_0) reaches the critical threshold of 1. Beyond this point, the disease could become a public health concern. As with $R_0 < 1$, the disease may eventually die out, while $R_0 > 1$ indicates the potential for an epidemic. This analysis highlights the role of (Λ_{HW}) in disease spread, aiding public health officials in making informed decisions about intervention strategies.

Figure 4 shows a simulation of the relationship between the parameters β_1 and τ_4 with respect to R_0. We observed that as the contact rate (β_1) increases, so does the rate at which humans become infectious (τ_4), leading to an increase in R_0 of the Nipah virus.

In Figure 5, we visualize the relationship between the parameters Λ_{HW} and γ_5 with respect to R_0. The simulation indicates that during an outbreak, a higher immigration rate of individuals likely to consume Nipah virus-contaminated food items such as unpasteurized palm wine (Λ_{HW}) could lead to a faster spread of the disease compared to when the immigration rate is low.

In Figure 6, we depict the relationship between the parameters δ_1 and v with respect to R_0. The simulation suggests that a higher vaccination rate (v) could paradoxically lead to a faster spread of the Nipah virus, unlike when the vaccination rate is low.

Figure 7 shows a simulation of the relationship between the parameters β_1 and γ_5 with respect to R_0. We observed an "L" shape in the visualization, indicating a transition between states: the darker region represents a lower R_0 (a disease-free state), while the lighter region indicates a higher R_0, suggesting an endemic equilibrium where the disease spreads faster.

In Figure 8, the simulation illustrates the relationship between the parameters n_0 and v for a given R_0. The plot shows that as n_0 and v increase, the combination's shade becomes darker, indicating a lower R_0. Conversely, a lighter shade indicates a higher R_0, signifying a faster spread of the disease.

Figure 9 presents the relationship between the parameters δ_1 and k for a given R_0. The plot indicates that as both parameters increase, the shade becomes lighter, representing a higher R_0 or an endemic equilibrium. A darker shade would indicate a lower R_0, meaning the disease would eventually die out.

In Figure 10, we visualize the relationship between the parameters Λ_{HW} and β_1 with respect to R_0. Our simulation suggests that a higher immigration rate of individuals likely to consume contaminated food items (Λ_{HW}) and a higher contact rate (β_1) increase the likelihood of a rapid spread of the Nipah virus in the community.

Figure 11 simulates our findings, showing the parameters that contribute positively or negatively to the growth of R_0. This approach helps us assess the sensitivity of the Nipah virus disease model to changes in different parameters.

5. Conclusion

By studying the Nipah virus, we were able to create an accurate mathematical model that encompassed the majority of the disease traits observed in an epidemic of the virus, which results in the rapid and widespread spread of the disease within an endemic population. Two key factors in lowering the number of people who can contract and spread the Nipah virus in the advent of an outbreak in a population, are that the rate of likelihood of consuming contaminated NiV food item (k) and the recruitment proportion of susceptible individuals likely to consume tainted food items (Λ_{HW}). In the analysis of equations 1 to 16, we obtained some threshold values in which k and Λ_{HW} should not exceed in order for us to be able to control the transmission of the NiV disease. In our further publications, we are going to solve this model using various numerical methods, as this could help us also validate our proposed model.

Conflict of Interests

The authors declare that there is no conflict of interests.

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