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MATHEMATICAL MODELLING APPROACH FOR THE STUDY OF NIPAH VIRUS DISEASE TRANSMISSION DYNAMICS

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Abstract. Ever since the first outbreak of Nipah virus disease, which occurred both in human and non-human primates in developing countries in Far East Asian between 1998 and 1999 which led to a majority of deaths, with the effect of such occurrence still witnessed up till date. We studied the spread of Nipah virus and obtained a system of equations comprising of ten equations which effectively described the transmission of Nipah Virus in a population where control measures were incorporated and two major sources of contacting the disease which are coming in contact with contaminated foods by infected bats from crop farming and pig farming, these were also incorporated. We investigated the local stability of the disease-free equilibrium using the Jacobian Matrix approach and the disease- endemic stability using the center manifold theorem. We also investigated the global

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stability of the equilibrium points using the LaSalle's invariant principle. The results showed that the disease-free and endemic equilibrium where both locally and globally Stable and we established a population immunity threshold for our model, with available data from the recent outbreak. Numerical simulations were carried out and our graphs show that vaccination and treatment are best for the infectious population to avoid further disease spread in the population and have quicker and better recovery.

Keywords: disease; model; variant; virus; Nipah; parameter.

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1. INTRODUCTION

Nipah virus (NiV) is a single-stranded RNA virus that causes severe disease both in humans and animals. It has two distinct strains, Nipah virus Malaysia (NiV_M) and Nipah virus Bangladesh (NiV_B). These two strains differ in transmission pattern, researches have shown that (NiV_B) could be more pathogenic than (NiV_M), Mire et. al [1].These two viruses are known to cause Nipah virus disease (NiVD) in humans.

Human beings can contract NiVD through but not limited to the following means: direct contact (such as broken skin, saliva, urine, or mucus membrane in the eyes, nose, or mouth) with blood or body fluids of a person who is sick with Nipah disease, or a non-human infected with the virus, Goh et. al [2]. One can equally get the disease through objects (such as syringes and needles) that have been contaminated with the virus from a person sick with Nipah disease or the body Skowron et. al [3].

Humans have been seen to be infected by direct contact with infected pigs, probably through mucous membranes, but possibly also through skin abrasions, or consumption of poorly cooked infected pig meat. In a Nipah-like outbreak in the Philippines Banerjee et.al [4], most patients had been involved in slaughtering sick horses or had eaten undercooked horsemeat from sick horses Chua et al [5]. In Bangladesh, human cases have been linked to drinking unpasteurized date palm sap (juice). Experimental infections have shown that urine is a significant route of henipavirus (HeV) excretion from fruit bats Mire et. al [1]. Therefore, the persistence of henipaviruses (HeVs) in fruit bat urine was examined Skowron et. al [3]. NiVD infection cluster was originally first identified in September 1998 in Perak, Malaysia, and this was closely

followed by a second and third cluster in the state of Negri Sembilan, with cases occurring primarily among adult men in contact with swine Chua et. al [5]; Abang et. al [6].

Observations from several clinical data for NiV outbreaks reveal some key differences between patients infected with NiV_M and NiV_B. Some of these differences are: (1) NiV_B has a shorter average incubation period and a more narrow range for the incubation period than NiV_M, (2) most of the recorded cases of NiV_B included respiratory symptoms while few patients infected with NiV_M presented with respiratory symptoms, (3) Rear cases of patients during the Bangladeshi and Indian NiVB outbreaks reported myoclonus, while a significant proportion of patients from the Malaysian outbreak presented with segmental myoclonus as well as the fatal cases in the Philippines presenting with an acute encephalitis Mire et. al [1]; Pallister et. al [7], (4) During the Bangladeshi outbreaks, sources revealed that the virus is either unknown in some cases or has been traced to be from consumption of contaminated fruit or date palm sap (or palm-wine), which was then followed by human-to-human transmission and nosocomial spread, whereas the source of the virus in the Malaysian outbreak is known to be from pigs, which served as an amplifying host Mire et. al [1]; Sultana et. al [8]. On the issue of host to the virus, it is speculated that bats are the host for Nipah virus Mondal et. al [9]; Biswas et. al [10]. Direct transmission from reservoirs or secondary infected animals may occur. non-domesticated animals may transfer the virus to humans when an infected and humans comes in contact with their infected and infectious body fluids or when their meat is eaten by humans uncooked or under-cooked. In this situation, the virus then spreads in the human population either through human-to-human, pig-to-human, or bat-to-human transmission. Airborne spread of the disease has not been detected or discovered in the observed Nipah virus outbreaks Weingartl et. al [11].

2. PRELIMINARIES

Having deliberated 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, we now develop a mathematical model representing this information that can enable us to carry out some analyses and provide vital information for the medical health officials about the disease caused by the virus. To do this successfully, we have our model assumptions as:

2.1. Model Assumptions. The development of the model is based on the following assumptions:

1. Those recruited into the system are not infected or contacted the disease.

2. We focused on a single strain, either the Nipah virus Malaysia (NiV_M) strain or Nipah virus Bangladesh (NiV_B) strain.

3. There is an intensive supportive care for those infected with the virus that goes for treatment.

4. The treatment given to those infected is a combinational therapy method, because at the time of this study there is no single specific approach or method for treating Nipah virus disease.

5. Nipah virus is seen to be a highly emerging disease and no single drug is available yet for its treatment.

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

7. The infected individual can recover either by care giving/combinational therapy or naturally.

8. One can recover from the infection without treatment.

9. Once recovered, a person or animal cannot get the same strain of the virus again. 10. We assume the latent class is merged with the infectious class.

11. In the nearest future, a globally acceptable vaccine for humen in the treatment of Nipah virus would be produced.

12. Pigs get infected by contact with infectious pigs or infectious bats.

13. People or pigs can die naturally or as a result of the disease.

14. Bats are the natural carriers of Nipah virus disease.

15. The pigs are caged so that they do not roam to eat defecation from humans that are infected.

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 leads us to the following: The human individuals that can readily contact the disease if in contact with an infectious person, infectious animal, or infectious bird, which we call the susceptible individuals, S_H; we have φ_H to be the rate of vaccination form the infection; we present (φ_H S_H), to be the proportion of the susceptible population that have been vaccinated; (1-φ_H) S_H is the proportion of susceptible population that are yet to be vaccinated;(I_H) Infected humans not yet going for

treatment; (I_{HT}) Infected humans population that are currently under-going for treatment; (R_H), human population that have recovered from the virus.

The pig population compartment is also divided into four mutually disjoint compartments, which are: (S_P), susceptible pigs; (I_P), infected pigs; (I_{PT}), virus infected pig population that has been taken for treatment; (R_P), the population of pigs that has recovered from the virus.

The bat population is also divided into two compartments, which are: susceptible bats (S_B), infected bats (I_B).

The model tracks how each individual group communicates between each other with respect to time by the application of differential equations.

We obtained the following mathematical system of equations from our above model assumptions:

$$\frac{dS_v}{dt} = \varphi_H S_H - \mu_H S_v \tag{1}$$

$$\frac{dS_H}{dt} = \Lambda_H - \frac{(\beta_1[n_0 I_H + n_1 I_P + n_2 I_B])S_H}{N_H} - \mu_H S_H \tag{2}$$

$$\frac{dI_H}{dt} = \frac{(\beta_1[n_0 I_H + n_1 I_P + n_2 I_B])S_H}{N_H} - [\gamma_1 + \tau_1 + \mu_H + \delta_1]I_H \tag{3}$$

$$\frac{dI_{HT}}{dt} = \tau_1 I_H - [\gamma_2 + \mu_H + \delta_2]I_H \tag{4}$$

$$\frac{dR_H}{dt} = \gamma_1 I_H + \gamma_2 - \mu_H R_H \tag{5}$$

$$\frac{dS_P}{dt} = \Lambda_P - \frac{(\beta_2[n_1 I_P + n_2 I_B])S_P}{N_P} - \mu_P S_P \tag{6}$$

$$\frac{dI_P}{dt} = \frac{(\beta_2[n_1 I_P + n_2 I_B])S_P}{N_P} - [\gamma_3 + \tau_2 + \mu_P + \delta_3]I_P \tag{7}$$

$$\frac{dI_{PT}}{dt} = \tau_2 I_P - [\gamma_4 + \mu_P + \delta_4]I_P \tag{8}$$

$$\frac{dR_P}{dt} = \gamma_3 I_P + \gamma_4 I_{PT} - \mu_P R_P \tag{9}$$

$$\frac{dS_B}{dt} = \Lambda_B - \frac{\beta_3 n_2 I_B S_B}{N_B} - \mu_B S_B \tag{10}$$

$$\frac{dI_B}{dt} = \frac{\beta_3 n_2 I_B S_B}{N_B} - [\mu_B + \delta_5] I_B \tag{11}$$

2.1.1. *Model Parameters.* This is shown in table 1.

TABLE 1. Model Parameters with description.

Parameters	Description
Λ_H	Proportion of new entry into the human class
Λ_P	New pig entry, either by birth or purchase into the pig society
Λ_B	Proportion of new entry into the bat society
φ_H	Rate of vaccination in the human population
μ_H	Rate of natural death in the human population
μ_P	Rate of natural death of pig
μ_B	Rate of natural death of bat
β_1	Contact rate between infected classes and humans
β_2	Contact rate between infected classes and pigs
β_3	Contact rate between infected classes and bats
τ_1	Rate of infected human goes for treatment
τ_2	Rate of infected pig that is taken for treatment
δ_1	Rate which infected humans dies of the disease
δ_2	Rate which infected humans treated dies of the disease
δ_3	Rate which infected pigs dies of the disease in infectious pig class
δ_4	Rate which infectious treated pig dies of the disease
δ_5	Rate which bats die as a result of human and other activities

The continuation for some of the parameters used and their descriptions are shown in table 2.

TABLE 2. Model Parameters with description. Continuation

Parameters	Description
γ_1	Rate of recovery in infected human
γ_2	Rate recovery in infected human undergoing treatment
γ_3	Rate at which infectious pigs recover
γ_4	Rate which infected treated pigs recover
n_0	Modification parameter related to reduced infectiousness of infected humans
n_1	Modification parameter related to reduced infectiousness of infected pigs
n_2	Modification parameter related to reduced infectiousness of infected bats
λ_H	Force of infection for the human class
λ_P	Force of infection for the pig class
λ_B	Force of infection for the bat class

3. MAIN RESULTS

Having developed the model equations, we check to see the well-posedness of the model by showing that the model satisfied the conditions on the positivity of the variables of the model as well as the continual remaining of the variables in the region of existence of the variables Castillo-Chavez and Song [12]. Thus, we have the theorem:

Theorem 1. *Let us consider the region defined as:*

$$\Omega_1 = \{S_v, S_H, I_H, I_{HT}, R_H, S_P, I_P, I_{PT}, R_P, S_B, I_B\} \subseteq \mathbb{R}_+^{11}$$

We show that the solutions to the system of equations $(2.1-2.11)$ *are positive for all* $t > 0$ *given that the initial conditions are positive. For the system of equations* $(2.1-2.11)$*, the region* Ω_1 *is positively invariant, and the solution starting in* Ω_1 *remains in* Ω_1*. Obasi and Mbah* [13].

PROOF

Suppose that all initial conditions of the variables are all positive, then, from (2):

$$\frac{dS_H}{dt} = \Lambda_H - \frac{(\beta_1[n_0 I_H + n_1 I_P + n_2 I_B])S_H}{N_H} - (\mu_H + \varphi_H)S_H$$

let

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

therefore, (2) becomes

$$\frac{dS_H}{dt} = \Lambda_H - \lambda_H S_H - (\mu_H + \varphi_H)S_H \tag{12}$$

Let

$$(\lambda_H - (\mu_H + \varphi_H)) = a_1$$

$$\therefore \frac{dS_H}{dt} = \Lambda_H - a_1 S_H \tag{13}$$

Next, we solve using the integrating factor method comparing equation (13) with the standard first order equation given as:

$$y^1 + Py = Q \tag{14}$$

Which solution is of the following form:

$$ye^{\int P dx} = [\int Qe^{\int P dx} dx + c] \tag{15}$$

where the integrating factor a(X):

$$X = e^{\int P dx}$$

Given the following from (13)

$$y^1 = \frac{dS_H}{dt}; S_H(t); P = a_1; Q = \Lambda_H; X = e^{\int a_1 dx} = e^{a_1 t}$$

then,

$$\int \Lambda_H e^{a_1 t} dt + A_1 = \frac{\Lambda_H}{a_1} e^{a_1 t} + A_1 \tag{16}$$

Therefore, the solution for equation (16) becomes:

$$S_H(t)e^{a_1 t} = \frac{\Lambda_H}{a_1} e^{a_1 t} + A_1 \tag{17}$$

Divide through by $(e^{a_1 t})$ and then apply the initial condition at $t = 0$, we have:

$$S_H(0) = \frac{\Lambda_H}{a_1} + c \tag{18}$$

$$\implies S_H(t) \geq 0$$

The solution is bounded by zero below and is positive for all t $\geq$ 0 We can continue using this same approach to prove for the remaining variables. Therefore, in general, we have that:

$$S_H(t) \neq 0, I_H(t) \neq 0, I_{HT}(t) \neq 0, R_H(t) \neq 0, S_P(t) \neq 0, I_P(t) \neq 0, I_{PT}(t) \neq 0, R_P(t), S_B(t) \neq 0, I_B(t) \neq 0$$

By this, we have shown that the solutions of the equations in the system (equations 2.2 – 2.11) are positive and in $\mathbb{R}^{10}_{+}$, provided that the initial conditions are positive. This implies that Ω_1 is positively invariant, which indicates that the model is mathematically and biologically well posed and reasonable enough to be used in the study of the transmission of Nipah virus disease.

Next, in our analytics agenda, we move to our other point which is the continuous existence and non-existence of this infection in the population. This is done by analyzing to get the equilibrium points of the disease at the disease free and endemic states. At these points, it is expected that the virulence of the disease does not change, which means we try not to study different viral strains of the virus. Thus, we study the equilibrium state as follows:

3.1. Disease free equilibrium (DFE). Let $E(S_H, I_H, I_{HT}, R_H, S_P, I_P, I_{PT}, R_P, S_B, I_B)$ be the equilibrium point of the displayed system (equations 2.2–2.11). Then, at the equilibrium state, we have that:

$$\frac{dS_H}{dt} = \frac{dI_H}{dt} = \frac{dI_{HT}}{dt} = \frac{dR_H}{dt} = \frac{dS_P}{dt} = \frac{dI_P}{dt} = \frac{dI_{PT}}{dt} = \frac{dR_H}{dt} = \frac{dS_B}{dt} = \frac{dI_B}{dt} = 0$$

This then implies that the right-hand side of equation (2.2 – 2.11) will be equated to zero, therefore, solving the resulting equations from our proposed model, we obtain the equilibrium point, E^n as:

$$E^n = (S^0_H, I^0_H, I^0_{HT}, R^0_H, S^0_P, I^0_P, I^0_{PT}, R^0_P, S^0_B, I^0_B) = \left(\frac{\Lambda_H}{(\mu_H + \varphi_H)}, 0, 0, 0, \frac{\Lambda_P}{\mu_P}, 0, 0, 0, \frac{\Lambda_B}{\mu_B}, 0\right)$$

Therefore, the disease-free equilibrium (DFE) for our Niv model is achieved when all the disease classes of humans, pigs, and bats are zero.

3.2. endemic disease equilibrium (EE). In this section, we discuss the endemic equilibrium state of the disease; this is the state where the disease is not totally absent in the various compartments of the population. We can say that the disease may persist in the population, such that the susceptible class, the infectious class, the infectious treated class, the recovered class, and others must not all be zero at this equilibrium state Gurley et. al [14]. In other, to obtain the endemic equilibrium points of the model, we solve equations (2.2 – 2.11) simultaneously. By equating :

$$\frac{dS_H}{dt} = \frac{dI_H}{dt} = \frac{dI_{HT}}{dt} = \frac{dR_H}{dt} = \frac{dS_P}{dt} = \frac{dI_P}{dt} = \frac{dI_{PT}}{dt} = \frac{dR_P}{dt} = \frac{dS_B}{dt} = \frac{dI_B}{dt} = 0$$

$$S_B^* = \frac{N_B(\mu_B+\delta_5)}{\beta_3 n_2}$$

$$I_B^* = \frac{(\beta_3 n_2)-(\mu_B+\delta_5)(N_B\mu_B)}{(\mu_B+\delta_5)(\beta_3 n_2)}$$

$$S_P^* = \left[\frac{A_P N_P(\mu_B+\delta_5)(\beta_3 n_2)}{(2\beta_2 n_2 I_B)[(\beta_3 n_2 N_B)A_B-(\mu_B+\delta_5)(N_B\mu_B)]+(\mu_P N_P)(\mu_B+\delta_5)(\beta_3 n_2)}\right]$$

$$I_P^* = \left[\frac{n_2[(\beta_3 n_2 N_B)A_B-(\mu_B+\delta_5)(N_B\mu_B)]}{n_1(\mu_B+\delta_5)(\beta_3 n_2)}\right]$$

$$I_{PT}^* = \frac{\tau_2\gamma_3(n_2[(\beta_3 n_2 N_B)A_B-(\mu_B+\delta_5)(N_B\mu_B)])}{[-(\gamma_3(\gamma_4+\mu_P+\delta_4))](n_1(\mu_B+\delta_5)(\beta_3 n_2))}$$

$$R_P^* = \frac{(\tau_1\gamma_2)-\gamma_1(\gamma_2+\mu_H+\delta_2)([(\beta_3 n_2)\Lambda_B-(\mu_B+\delta_5)N_B\mu_B])}{\tau_1\mu_H[-(\gamma_1(\gamma_2+\mu_H+\delta_2))(n_0((\mu_B+\delta_5)\beta_3 n_2))]}$$

$$S_H^* = \left[\frac{\Lambda_H-(\gamma_1+\tau_1+\mu_H+\delta_2)(2n_2[(\beta_8 n_2)\Lambda_B-(\mu_B+\delta_5)(N_B\mu_B)])}{\mu_H n_0((\mu_B+\delta_5)(\beta_3 n_2))}\right]$$

$$I_H^* = \frac{n_2[(\beta_3 n_2 N_B)A_B-(\mu_B+\delta_5)(N_B\mu_B)]}{n_1(\mu_B+\delta_5)(\beta_3 n_2)}$$

$$I_{HT}^* = \frac{\tau_2\gamma_3(n_2[(\beta_3 n_2 N_B)A_B-(\mu_B+\delta_5)(N_B\mu_B)])}{[-(\gamma_3(\gamma_4+\mu_P+\delta_4))](n_1(\mu_B+\delta_5)(\beta_3 n_2))}$$

$$R_H^* = \frac{(\tau_1\gamma_2-\gamma_1(\gamma_2+\mu_H+\delta_2))(\tau_1\gamma_1(2n_2[(\beta_3 n_2)\Lambda_B-(\mu_B+\delta_5)(N_B\mu_B)]))}{\tau_1\mu_H[-(\gamma_1(\gamma_2+\mu_H+\delta_2))](n_0((\mu_B+\delta_5)(\beta_3 n_2)))}$$

Therefore, the endemic equilibrium points for our proposed model equation (2.2 - 2.11) exists, which is given as:

$$E^p = (S_H^*, I_H^*, I_{HT}^*, R_H^*, S_P^*, I_P^*, I_{PT}^*, R_P^*, S_B^*, I_B^*) \neq (0,0,0,0,0,0,0,0,0,0)$$

Having produced an acceptable equilibrium points, we need to verify whether these equilibrium points are stable or not. But first we need to generate the basic reproductive number for our studied disease, then we would study the per capita disease growth rate, stability of the disease free and endemic states of the studied disease at the equilibrium points stability: the local and global stability of the disease states

3.3. The Basic Reproductive number (R_0) for the study. To derive the basic reproduction number for our proposed model system as described in equations (2.2 - 2.11). We then form the

next generation matrix which is F V^{-1}, where F is the new infection term and V is the matrix of all the transferred terms. Also, F is the rate of appearance of new infection; V is the difference between the transfer rate of individual out of compartment by other means and the transfer rate of individuals into the same compartment by other means Shah et. al [15].

$$F = \begin{bmatrix} \frac{\partial f_1}{\partial I_H} & \frac{\partial f_1}{\partial I_{HT}} & \frac{\partial f_1}{\partial I_P} & \frac{\partial f_1}{\partial I_{PT}} & \frac{\partial f_1}{\partial I_B} \\ \frac{\partial f_2}{\partial I_H} & \frac{\partial f_2}{\partial I_{HT}} & \frac{\partial f_2}{\partial I_P} & \frac{\partial f_2}{\partial I_{PT}} & \frac{\partial f_2}{\partial I_B} \\ \frac{\partial f_3}{\partial I_H} & \frac{\partial f_3}{\partial I_{HT}} & \frac{\partial f_3}{\partial I_P} & \frac{\partial f_3}{\partial I_{PT}} & \frac{\partial f_3}{\partial I_B} \\ \frac{\partial f_4}{\partial I_H} & \frac{\partial f_4}{\partial I_{HT}} & \frac{\partial f_4}{\partial I_P} & \frac{\partial f_4}{\partial I_{PT}} & \frac{\partial f_4}{\partial I_B} \\ \frac{\partial f_5}{\partial I_H} & \frac{\partial f_5}{\partial I_{HT}} & \frac{\partial f_5}{\partial I_P} & \frac{\partial f_5}{\partial I_{PT}} & \frac{\partial f_5}{\partial I_B} \end{bmatrix} \tag{19}$$

$$V = \begin{bmatrix} \frac{\partial V_1}{\partial I_H} & \frac{\partial V_1}{\partial I_{HT}} & \frac{\partial V_1}{\partial I_P} & \frac{\partial V_1}{\partial I_{PT}} & \frac{\partial V_1}{\partial I_B} \\ \frac{\partial V_2}{\partial I_H} & \frac{\partial V_2}{\partial I_{HT}} & \frac{\partial V_2}{\partial I_P} & \frac{\partial V_2}{\partial I_{PT}} & \frac{\partial V_2}{\partial I_B} \\ \frac{\partial V_3}{\partial I_H} & \frac{\partial V_3}{\partial I_{HT}} & \frac{\partial V_3}{\partial I_P} & \frac{\partial V_3}{\partial I_{PT}} & \frac{\partial V_3}{\partial I_B} \\ \frac{\partial V_4}{\partial I_H} & \frac{\partial V_4}{\partial I_{HT}} & \frac{\partial V_4}{\partial I_P} & \frac{\partial V_4}{\partial I_{PT}} & \frac{\partial V_4}{\partial I_B} \\ \frac{\partial V_5}{\partial I_H} & \frac{\partial V_5}{\partial I_{HT}} & \frac{\partial V_5}{\partial I_P} & \frac{\partial V_5}{\partial I_{PT}} & \frac{\partial V_5}{\partial I_B} \end{bmatrix} \tag{20}$$

$$F = \begin{bmatrix} \frac{\beta_1 n_0 s_H}{N_H} & 0 & \frac{\beta_1 n_1 s_H}{N_H} & 0 & \frac{\beta_1 n_2 s_H}{N_H} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_2 n_1 s_P}{N_P} & 0 & \frac{\beta_2 n_2 s_P}{N_P} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_8 n_2 s_B}{N_B} \end{bmatrix} \tag{21}$$

Then, we let

$$[\gamma_1 + \tau_1 + \mu_H + \varphi_H + \delta_1] = b_1, [\gamma_2 + \mu_H + \varphi_H + \delta_2] = b_2, [\gamma_3 + \tau_2 + \mu_P + \delta_3] = b_3,$$

$$[\gamma_4 + \mu_p + \delta_4] = b_4, [\mu_B + \delta_5] = b_5 \tag{22}$$

$$V = \begin{bmatrix} b_1 & 0 & 0 & 0 & 0 \\ -\tau_1 & b_2 & 0 & 0 & 0 \\ 0 & 0 & b_3 & 0 & 0 \\ 0 & 0 & -\tau_2 & b_4 & 0 \\ 0 & 0 & 0 & 0 & b_5 \end{bmatrix} \tag{23}$$

We then computed FV^{-1}, and evaluated all its eigenvalues. Where we got the following to be the R_0, and it is the same as computed by Abang et. al [6]

$$R_0 = \frac{\beta_1 n_0 \Lambda_H}{[\gamma_1 + \tau_1 + \mu_H + \varphi_H + \delta_1] N_H (\mu_H + \varphi_H)} \tag{24}$$

3.3.1. *Infected population per capita growth rate.* In this section, we try to show the relationship between R_0 and the rate at which the product of the proportion of susceptible individuals in the population who are yet to be infected or have not been vaccinated times the transmission rate of the disease (this is known as per capita growth rate for the disease). From equation (3), we have that:

$$\frac{dI_H}{dt} = \frac{\beta_1 [n_0 I_H + n_1 I_P + n_2 I_B] S_H}{N_H} - [\gamma_1 + \tau_1 + \mu_H + \varphi_H + \delta_1] I_H$$

this equation is simplified, thus, the following:

$$\frac{1}{I_H} \frac{dI_H}{dt} = \left(\frac{\beta_1 n_0 \Lambda_H}{\gamma_1 + \tau_1 + \mu_H + \varphi_H + \delta_1} - 1\right) \tag{25}$$

we then have (25), which is the rate at which infections occur in the community per infected individual. It helps us determine whether the disease will spread and grow in the population or eventually fade away.

3.4. Local stability. Here, we conduct the local stability analysis for our proposed model. Local stability analysis helps us to determine whether an equilibrium point of a dynamic system is stable or unstable Tyagi et. al [16]. We have the local stability analysis in the disease free and endemic state. In the disease-free state.

3.4.1. *Local stability for Nipah virus single variant disease free state.*

Theorem 2. *Given a matrix $J(E^0)$ and all of its eigenvalues have negative real components, we say that the virus-free equilibrium at the point E^0 is locally asymptotically stable; otherwise, it is unstable.*

$$J(E^0) = \begin{bmatrix} -\mu_H & D_1 & 0 & 0 & 0 & D_2 & 0 & 0 & 0 & D_3 \\ 0 & D_4 & 0 & 0 & 0 & -D_2 & 0 & 0 & 0 & -D_3 \\ 0 & \tau_1 & D_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -\mu_H & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_P & D_6 & 0 & 0 & 0 & D_7 \\ 0 & 0 & 0 & 0 & 0 & D_8 & 0 & 0 & 0 & -D_7 \\ 0 & 0 & 0 & 0 & 0 & \tau_2 & D_9 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_3 & \gamma_4 & -\mu_P & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_B & D_{10} \\ 0 & 0 & 0 & 0 & b_5 & b_1 & 0 & 0 & 0 & D_{11} \end{bmatrix} \tag{26}$$

$$D_i \quad \text{where} \quad i = 1,2,3,4,5...11$$

$$D_1 = -\frac{\beta_1 n_0 \Lambda_H}{N_H \mu_H}; \quad D_2 = -\frac{\beta_1 n_1 \Lambda_H}{N_H \mu_H}; \quad D_3 = -\frac{\beta_1 n_2 \Lambda_H}{N_H \mu_H}; \quad D_4 = -\frac{\beta_1 n_0 \Lambda_H}{N_H \mu_H} - [\gamma_1 + \tau_1 + \mu_H + \delta_1];$$

$$D_5 = -[\gamma_2 + \mu_H + \delta_2]; \quad D_6 = -\frac{\beta_2 n_1 \Lambda_P}{N_P \mu_P}; \quad D_7 = -\frac{\beta_2 n_2 \Lambda_P}{N_P \mu_P}; \quad D_8 = -\frac{\beta_2 n_1 \Lambda_P}{N_P \mu_P} - [\gamma_3 + \tau_2 + \mu_P + \delta_3];$$

$$D_9 = -[\gamma_4 + \mu_P + \delta_4]; \quad D_{10} = -\frac{\beta_3 n_1 \Lambda_B}{N_B \mu_B}; \quad D_{11} = -\frac{\beta_2 n_1 \Lambda_P}{N_P \mu_P} - [\mu_B + \delta_5]$$

Using a Python program, we evaluated the Jacobian matrix $J(E^0)$. We obtained the following eigenvalues: ζ_i where $i = 1,2,3,4,5,...,10$.

$$\zeta_1 = -\mu_B$$

$$\zeta_2 = \frac{D_9}{2} + \frac{\sqrt{4\tau_2 D_8 + D_9^2}}{2}$$

$$\zeta_3 = \frac{D_9}{2} - \frac{\sqrt{4\tau_2 D_8 + D_9^2}}{2}$$

$$\zeta_4 = D_5$$

$$\zeta_5 = D_4$$

$$\zeta_6 = D_{11}$$

$$\zeta_7 = -\mu_P$$

$$\zeta_8 = -(\mu_H + \varphi_H)$$

$$\zeta_9 = -\mu_P$$

$$\zeta_{10} = -(\mu_H + \varphi_H)$$

From the obtained eigenvalue results, it can be seen that all the eigenvalues have negative real parts, which implies that the disease-free equilibrium state is locally asymptotically stable.

3.4.2. *Local stability for Nipah virus single variant Endemic Equilibrium.* For the disease-endemic state, we studied the local stability using the Centre Manifold theorem by Castillo-Chavez and Song [12].

We define the transformation of equations (1) to (11) as

$$\frac{d\Phi}{dt} = F_i(\phi)$$

such that

$$F_i(\phi) = (f_1, f_2, f_3, \ldots, f_{11})(\phi)$$

and

$$(S_H, S_V, I_H, I_{HT}, R_H, S_P, I_P, I_{PT}, R_P, S_B, I_B) = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7, \phi_8, \phi_9, \phi_{10}, \phi_{11})$$

Then, we can further define equations (1) to (11) as:

$$\frac{d\phi_1}{dt} = \Lambda_H - \vartheta_1\phi_1 - (\mu_H + \varphi_H)\phi_1 : f_1 \tag{27}$$

$$\frac{d\phi_2}{dt} = \Lambda_v + \varphi_H\phi_1 - \mu_H\phi_2 : f_2 \tag{28}$$

$$\frac{d\phi_3}{dt} = \vartheta_1\phi_1 - \vartheta_2\phi_3 : f_3 \tag{29}$$

$$\frac{d\phi_4}{dt} = \tau_1\phi_3 - \vartheta_3\phi_4 : f_4 \tag{30}$$

$$\frac{d\phi_5}{dt} = \gamma_1\phi_3 + \gamma_2\phi_4 - \mu_H\phi_5 : f_5 \tag{31}$$

$$\frac{d\phi_6}{dt} = \Lambda_P - \vartheta_4\phi_6 - \mu_H\phi_6 : f_6 \tag{32}$$

$$\frac{d\phi_7}{dt} = \vartheta_4\phi_6 - \vartheta_5\phi_7 : f_7 \tag{33}$$

$$\frac{d\phi_8}{dt} = \tau_2\phi_7 - \vartheta_6\phi_8 : f_8 \tag{34}$$

$$\frac{d\phi_9}{dt} = \gamma_3\phi_7 + \gamma_4\phi_8 - \mu_P\phi_9 : f_9 \tag{35}$$

$$\frac{d\phi_{10}}{dt} = \Lambda_B - \vartheta_7\phi_{10} - \mu_B\phi_{10} : f_{10} \tag{36}$$

$$\frac{d\phi_{11}}{dt} = \vartheta_7\phi_{10} - \vartheta_8\phi_{11} : f_{11} \tag{37}$$

where

$$\vartheta_1 = \frac{\beta_1(n_0I_H + n_1I_P + n_2I_B)}{N_H}, \quad \vartheta_2 = [\gamma_1 + \tau_1 + \mu_H + \delta_1], \quad \vartheta_3 = [\gamma_1 + \mu_H + \delta_2],$$

$$\vartheta_4 = \frac{\beta_2(n_1I_P + n_2I_B)}{N_P}, \quad \vartheta_5 = [\gamma_3 + \tau_2 + \mu_P + \delta_3], \quad \vartheta_6 = [\gamma_4 + \mu_P + \delta_4],$$

$$\vartheta_7 = \frac{\beta_3 n_2 I_B}{N_B}, \quad \vartheta_8 = [\mu_B + \delta_5].$$

Next, we applied the central manifold theorem and obtained the bifurcation coefficients $a < 0$ and $b < 0$ Ahman et. al [17]; Atokolo and Mbah et al [18]. This shows that the endemic equilibrium state is locally asymptotically stable and at the same time exhibits forward bifurcation. This implies that we can reduce the reproduction number to be less than one by increasing our educational campaign. This is enough to restrain the spread of the Nipah virus disease, irrespective of the initial sizes of the infected populations. Now we can study the global stability.

3.5. Global Stability Analysis for our Nipah virus disease model.

Theorem 3. *If $W > 1$, the non-negative equilibrium point (E^0) of the model* (1) *to* (11) *is globally stable and convergence to the equilibrium point (E^0).*

Proof. In order to establish the global stability of this equilibrium point (E^0), we construct the Lyapunov function Weingartl et. al [11]; Ahman et. al [17]; Atokolo and Mbah [18].

$$W(S_H^0, S_V^0, I_H^0, I_{HT}^0, R_H^0, S_P^0, I_P^0, I_{PT}^0, R_P^0, S_B^0, I_B^0) =$$

$$\begin{aligned}
&\left(S_H - S_H^0 - S_H^0 \log \frac{S_H^0}{S_H}\right) + \left(S_V - S_V^0 - S_V^0 \log \frac{S_V^0}{S_V}\right) \\
&+ \left(I_H - I_H^0 - I_H^0 \log \frac{I_H^0}{I_H}\right) + \left(I_{HT} - I_{HT}^0 - I_{HT}^0 \log \frac{I_{HT}^0}{I_{HT}}\right) \\
&+ \left(R_H - R_H^0 - R_H^0 \log \frac{R_H^0}{R_H}\right) + \left(S_P - S_P^0 - S_P^0 \log \frac{S_P^0}{S_P}\right) \\
&+ \left(I_P - I_P^0 - I_P^0 \log \frac{I_P^0}{I_P}\right) + \left(I_{PT} - I_{PT}^0 - I_{PT}^0 \log \frac{I_{PT}^0}{I_{PT}}\right) \\
&+ \left(R_P - R_P^0 - R_P^0 \log \frac{R_P^0}{R_P}\right) + \left(S_B - S_B^0 - S_B^0 \log \frac{S_B^0}{S_B}\right) \\
&+ \left(I_B - I_B^0 - I_B^0 \log \frac{I_B^0}{I_B}\right)
\end{aligned}$$

But at the Nipah virus-free equilibrium,

$$I_H = I_{HT} = R_H = I_P = I_{PT} = R_P = I_B$$

$$\begin{aligned}
\frac{dW}{dt} &= \left(\frac{S_H - S_H^0}{S_H}\right)\frac{dS_H}{dt} + \left(\frac{S_V - S_V^0}{S_V}\right)\frac{dS_V}{dt} + \left(\frac{I_H - I_H^0}{I_H}\right)\frac{dl_H}{dt} \\
&+ \left(\frac{I_{HT} - I_{HT}^0}{I_{HT}}\right)\frac{dI_{HT}}{dt} + \left(\frac{R_H - R_H^0}{R_H}\right)\frac{dR_H}{dt} + \left(\frac{S_P - S_P^0}{S_P}\right)\frac{dS_P}{dt} \\
&+ \left(\frac{I_P - I_P^0}{I_P}\right)\frac{dI_P}{dt} + \left(\frac{I_{PT} - I_{PT}^0}{I_{PT}}\right)\frac{dI_{PT}}{dt} + \left(\frac{R_P - R_P^0}{R_P}\right)\frac{dR_P}{dt} \\
&+ \left(\frac{S_B - S_B^0}{S_B}\right)\frac{dS_B}{dt} + \left(\frac{I_B - I_B^0}{I_B}\right)\frac{dI_B}{dt}
\end{aligned} \tag{38}$$

we expand (38) and collect the positive and negative terms separately, where the positive term is N and and the negative term is M, then

$$\frac{dW}{dt} = N - M$$

$$
(39)\quad \begin{cases} N = \left(1-\frac{S_H^0}{S_H}\right)\Lambda_H + \left(1-\frac{S_V^0}{S_V}\right)(\Lambda_V+\varphi_V) + \left(1-\frac{I_H^0}{I_H}\right)\left[\frac{\beta_1[n_0I_H+n_1I_P+n_2I_B]S_H}{N_H}\right] + \\ \left(1-\frac{I_{HT}^0}{I_{HT}}\right)[\tau_1 I_H - [\gamma_2+\mu_H+\delta_2]I_{HT}] + \left(1-\frac{R_H^0}{R_H}\right)[\gamma_1 I_H+\gamma_2 I_{HT}] + \\ \left(1-\frac{S_P^0}{S_P}\right)\Lambda_P + \left(1-\frac{I_P^0}{I_P}\right)\left[\frac{\beta_3[n_1I_P+n_2I_B]S_P}{N_P}\right] + \\ \left(1-\frac{I_{PT}^0}{I_{PT}}\right)\tau_2 I_P + \left(1-\frac{R_P^0}{R_P}\right)[\gamma_3 I_P+\gamma_4 I_{PT}] + \\ \left(1-\frac{S_B^0}{S_B}\right)\Lambda_B + \left(\frac{(I_B-I_B^0)}{I_B}\right)\frac{\beta_3 n_2 I_B S_B}{N_B} \end{cases}
$$

$$
(40)\quad \begin{cases} M = \frac{\left(S_H-S_H^0\right)^2}{S_H}\frac{1}{N_H}\left(\beta_1\left[n_0I_H+n_1I_P+n_2I_B\right]\right) + \frac{\left(S_H-S_H^0\right)^2}{S_H}\mu_H + \frac{\left(S_V-S_V^0\right)^2}{S_v}\mu_V + \\ \frac{\left(I_{HT}-I_{HT}^0\right)^2}{I_{HT}}\left[\gamma_2+\mu_H+\delta_2\right] + \frac{\left(R_H-R_H^0\right)^2}{R_H}\mu_H + \\ \frac{\left(S_P-S_P^0\right)^2}{S_P}\frac{1}{N_P}\left[\beta_2\left[n_1I_P+n_2I_B\right]\right] + \frac{\left(S_P-S_P^0\right)^2}{S_P}\mu_P + \\ \frac{\left(I_P-I_P^0\right)^2}{l_P}\left[\gamma_3+\tau_2+\mu_P+\delta_3\right] + \frac{\left(I_{PT}-I_{PT}^0\right)^2}{I_{PT}}\left[\gamma_4+\mu_P+\delta_4\right] + \\ \frac{\left(R_P-R_P^0\right)^2}{R_P}\mu_P + \frac{\left(S_B-S_B^0\right)^2}{S_B}\frac{1}{N_B}\left(\frac{\beta_3 n_2 I_B}{N_B}\right) + \frac{\left(S_B-S_B^0\right)^2}{S_B}\mu_B + \frac{\left(I_B-I_B^0\right)^2}{I_B}\left[\mu_B+\delta_5\right] \end{cases}
$$

If $N < M$, then $\frac{dW}{dt}$ will be negative definite along the solution path of the sub-system. And thus, implies that , only at Nipah disease free equilibrium (E^0) would $\frac{dW}{dt} \leq 0$. This indicates that the system is globally stable at the Nipah virus disease free equilibrium.

4. RESULTS

Here, we discuss our numerical results and create simulations to give a better understanding of our model discussions.

4.1. Numerical Simulations. In this 4.1, we perform mathematical solutions for our proposed Nipah virus model system from (1) to (11), using data for the model parameters presented in 2 and 4. We conducted simulations for our system (1) to (11) using computer aided softwares (MATLAB and Python) to test the behaviour of our control parameters in the model (1) to (11), this is done by approximating our initial values and parameters as described by Jain et. al [21]: $N_H(0) = 1020$, $N_P(0) = 95$, $N_B(0) = 50$, $\Lambda_H = 20$, $\Lambda_P = 4$, $\Lambda_B = 2S_H(0) = 1000$, $S_v = 12$, $I_H = 20$, $I_{HT} = 12$, $R_H = 0$, $S_P = 90$, $I_P = 5$, $I_{PT} = 3$, $S_B = 50$, $I_B = 37$.

The graphical findings below were developed through python programming software applying the parameter values in 2 and 4, with also the initial values.

TABLE 3. Numerical values for Parameters adopted for implementation.

Parameters	Numerical value	Sources
Λ_H	20	*Estimated*
Λ_P	4	*Estimated*
Λ_B	2	Shah et. al [15]
$N_H(0)$	1020	*Estimated*
N_P	95	*Estimated*
N_B	50	Shah et. al [15]
μ_H	0.0000421	Zewdie and Gakhar [19]
μ_P	0.16	Tyagi et. al [16]
μ_B	0.45	*Estimated*
β_1	0.0002	Zewdie and Gakhar [19]
β_2	0.01	*Estimated*
β_3	0.1	Singh et. al [20]
τ_1	0.0001	Zewdie and Gakhar [19]
τ_2	0.0002	Zewdie and Gakhar [19]
δ_1	0.002	Tyagi et. al [16]

TABLE 4. Continuation of Numerical values for Parameters adopted for implementation.

Parameters	Numerical value	Sources
φ_H	20	*Estimated*
δ_2	0.001	*Estimated*
δ_3	0.0015	*Estimated*
δ_4	0.0018	*Estimated*
δ_5	0.75	Zewdie and Gakhar [19]
γ_1	0.58	Tyagi et. al [16]
γ_2	0.72	Sultana and Podder [8]
γ_3	0.4	*Assumed*
γ_4	0.45	Shah et. al [15]
n_0	0.0015	*Estimated*
n_1	0.02	*Estimated*
n_2	0.2	*Assumed*
λ_H	0.75	Tyagi et. al [16]; Jain et. al [21]
λ_P	0.0075	Shah et. al [15]
λ_B	0.07	Shah et. al [15]

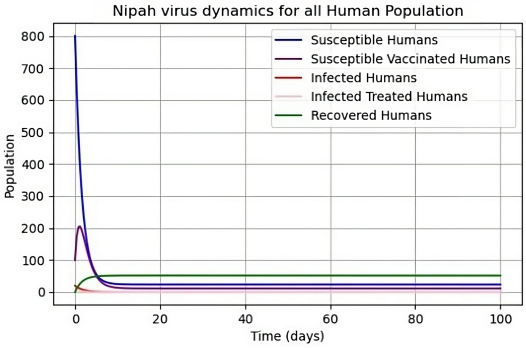
These parameters in 2 and 4 describe the transmission dynamics of the Nipah virus disease and the effects of different proposed control interventions. The parameters are given specific values that are based on the characteristics of the disease being modeled.

4.2. Simulation Figures. Figure 1 depicts the Nipah virus dynamics in the human population and that of the susceptible human population S_H, S_v, over a period of 100 days.

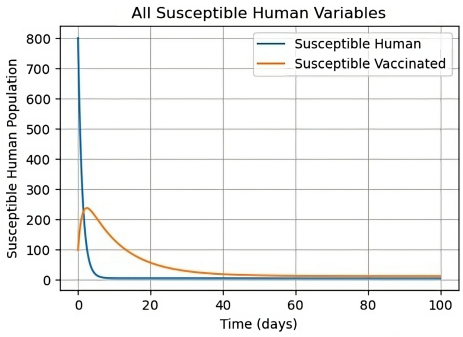
Figure 1a shows the various dynamics of Nipah virus in the human population, during the disease outbreak.

In Figure 1b, the susceptible S_H decreased as time increased due to the outbreak of Nipah virus without knowledge of the kind of virus that emerged. We discovered that the susceptible

population never got to zero due to various factors such as natural immunity to the virus by those that have suffered from disease similar to Nipah virus.



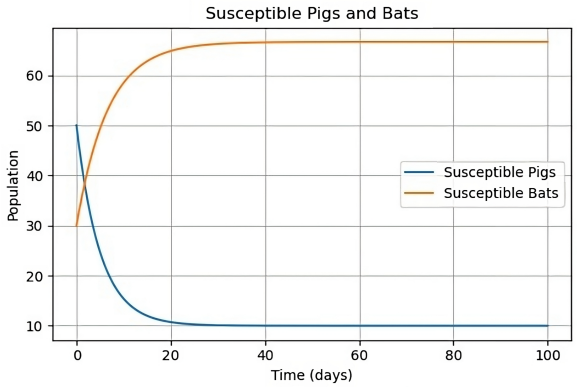
(A)



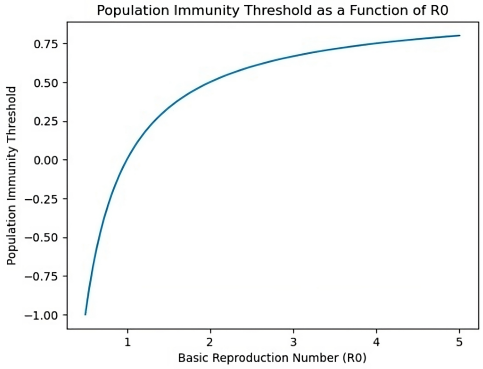
(B)

FIGURE 1.

Nipah virus disease dynamics in human population and vaccinated popupulation



(A)



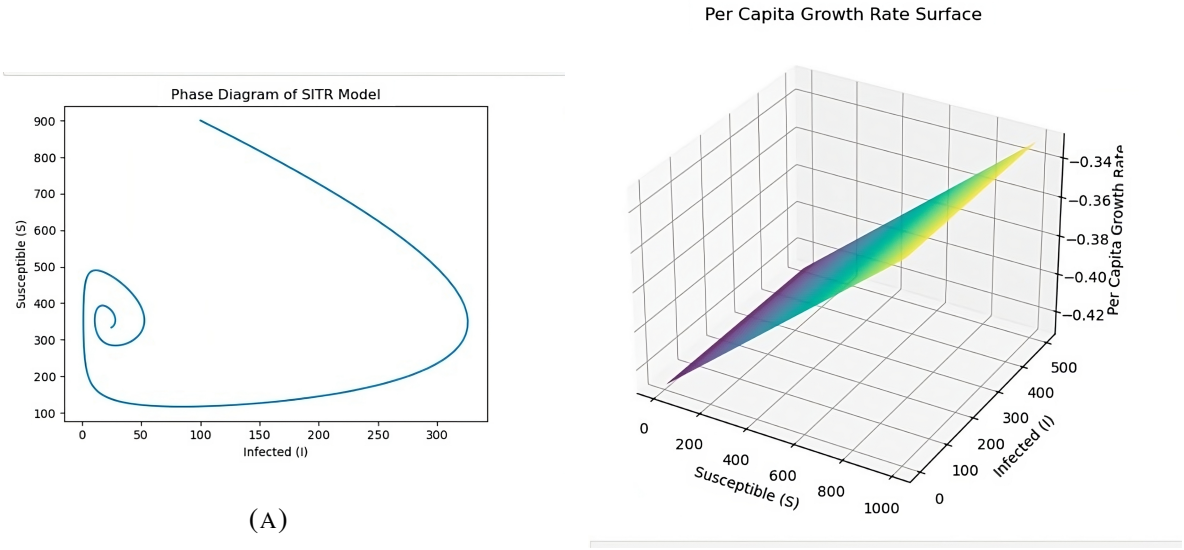
(B)

FIGURE 2.

Nipah virus disease dynamics for susceptible classes and Population immunity threshold

In Figure 2, graph of non-human susceptibles and population immunity threshold are shown. Figure 2a depicts a scenario where the susceptible bat population grows as time increases, which is inline with our model assumptions and our model of equation 10. In figure 2b we say a

graphical display of the level which must be vaccinated for the population to reach an immunity threshold, where the Nipah virus disease can no longer be a threat in the observed society. Figure 3 expresses the graphs that display the phase diagram for our proposed model 1 to equation 11 and the per capita growth for the Nipah virus disease is also shown in Figure 3. Figure 3a indicates the extent of how serious each outbreak of Nipah virus disease would be if an outbreak occurs, it shows that every subsequent outbreak would not be as devastating as the one before, but the disease might be endemic in the society. In Figure 3b the graph shows that as Nipah virus per capita growth increases, the number of susceptibles reduces, so in other for us to maintain a better healthy society, we need the disease per capita growth to be low.



(A)

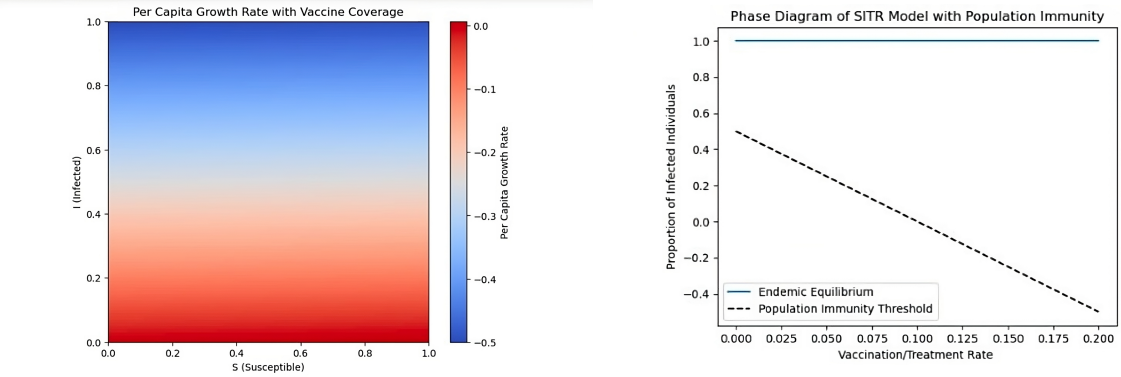
(B)

FIGURE 3.

Phase plot of Susceptible, Infected population and Nipah per capita growth rate

Figure 4 depicts visualization of effect of treatment and vaccine on our proposed model of equations (1) to equation (11). Figure 4a, indicates the extent to which administering of vaccine during outbreak of Nipah virus disease would have on both the susceptible and infected population. It shows that if the susceptible population accepts the vaccination idea, this singular act would reduce those that would become infected as a result decreasing the infected population.

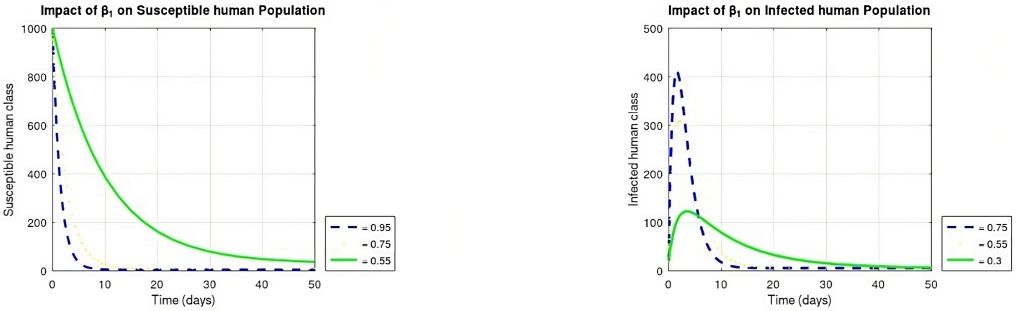
Figure 4b displays a graphical visualization of the treatment effect of treatment on our susceptible population regardless of that at any point in time there must be someone infected with the virus, but once the right treatment is administered coupled with positive vaccine penetration, the Nipah virus disease could be control.



(A) (B)

FIGURE 4.

Vaccine and treatment rate on Nipah dynamics population



(A) (B)

FIGURE 5.

Effect of β_1 on Susceptible and Infected populations

From figure 5, we have the graphical picture showing the impact of (β_1)

5. CONCLUSION

In our study of Nipah virus, we were able to formulate a clear mathematical model that contained the majority of the disease features obtainable in Nipah virus outbreak that instigates

the broad and rapid transmission of Nipah Virus disease within an endemic population. We included the issue of vaccine coverage in the community as one of the major sources for achieving population immunity and reducing contacting and spreading of the Nipah virus disease.

In the analysis of equations (1) to (11), we obtained the equilibrium points of the transmission of the disease and further analyzed these equilibrium points to obtain the conditions for the local and global stabilities of the disease free and endemic equilibriums of the disease transmission dynamics.

It can be seen from our graph that early intervention and reducing the disease contact rate is the best option for managing and treating an infectious people in order to reduce the spread and transmission of the disease, which would further lead to a healthy and more productive society. In addition, from our graphs, the population immunity threshold was identified in the population for effective administering of vaccine to a large number of the vulnerable community as it has a great effect on the disease spread and control.

In our further publications, we are going to include the susceptible population of palm-wine drinkers, how they can alter the disease dynamics and carry out sensitivity analyses to see the effect of the control, transmission, and treatment parameters on the spread of the disease in the population.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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