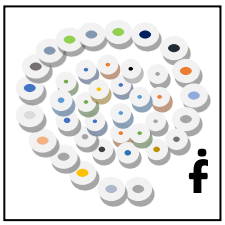

International Journal of Mathematical Analysis and Modelling

(Formerly Journal of the Nigerian Society for Mathematical Biology)

Volume 5, Issue 1 (May), 2022

ISSN (Print): 2682 - 5694
ISSN (Online): 2682 - 5708

International Journal of Mathematical Analysis and Modelling
Volume 5, Issue 1, May 2022, Pages 100 – 120

Sensitivity analysis of Nipah virus Malaysian variant (NiV_m) with transmission dynamics

S. I. S. Abang[*†], A. L. Ozioko[‡†], O. N. Nze[*§], G. C. E. Mbah[†] , and C. T. Onyia[††]**

Abstract

In this paper, we established the basic reproductive number of the Nipah virus disease, the DFE and endemic equilibrium of the SIR disease model. We deploy the next generation matrix approach to obtain sufficient conditions under which the basic reproductive number was obtained. We then perform a sensitivity analysis of the model, which helped us come up with some proposed policies to assist stake holders on the right policies to apply. The study aimed at analyzing the sensitivity of the parameters in relations to the basic reproductive number of the proposed disease model; to achieve this we use the following objectives: (1) obtain the basic reproductive number of the model (2) perform sensitivity analysis of the model. From our analysis, some of the followings were drawn as conclusions. It was discovered that some parameters were found to be positively contributing to the spread of the disease, parameters like Λ_H, which is the recruitment parameter for the model, β_1, this parameter is the effective contact rate, while some other parameters reduce the spread of the disease, parameters like τ_1, which is the proportion of infected that goes for treatment, while μ_H, natural death.

[*] Corresponding Author: National Centre for Technology Management, an Agency of the Federal Ministry of Science & Technology, PRODA Training Institute, Independence Layout, Enugu, Nigeria; sunnyscott2010@gmail.com; 07033911987
[†] Department of Mathematics, University of Nigeria Nsukka, Nigeria
[‡] Department of Mathematics, Federal University, Lokoja, Nigeria; arinze.luke@fulokoja.edu.ng; 08034198454
[§] obynnekanze@gmail.com; 08062627262
[**] godwin.mbah@unn.edu.ng; 08032198708
[††] Department of Statistics, Institute of Management Technology (IMT), Enugu, Nigeria; chukwuemekaonyia@gmail.com; 07035410292

1. Introduction

Nipah virus (NiV) infection is an emerging pathogen endemic to Southeast Asia that can be transmitted from its reservoir (wild animals, fruit bats, pigs etc) to human. NiV is a zoonotic virus (that is, it is transmitted from animals to humans) and can also be transmitted through contaminated food or directly between people [13]. It was first detected in the 1998–1999 outbreaks in Kampung Sungai Nipah, Malaysia and then Singapore, with seasonal outbreaks in Bangladesh and India since 2001, and a suspected 2014 outbreak in the Philippines. It derived its name from Sungai Nipah, a village in Malaysia where the virus was first detected in 1998 [1]. In Africa there has been an outbreak in Madagascar, which shows that Africa is not spear of this new deadly zoonotic virus [5].

In the 1998-99 Malaysia outbreak, NiV spillover occurred from bats to pigs, which led to pig-to-pig, pig-to-human, and suspected, although limited, human-to-human NiV transmission [11]. After the first noticeable outbreak in Malaysia, NiV infection has subsequently been recognized in Bangladesh in 2001 and nearly annual outbreaks have occurred in this country ever since, with disease also identified periodically in eastern India; case fatality rates during outbreaks in these countries have ranged from 75% to 100%. Other regions may be at risk for NiV infection, as serologic evidence for NiV has been found in the known natural reservoir (Pteropus bat species) and several other bat species in a number of other countries, including Cambodia, Thailand, Indonesia, Madagascar, Ghana, and the Philippines [12]; [8].

2. Model formulation

2.1. Model assumption

The development of the model is based on the following assumptions:

i. Those recruited into the system are not infected or contacted the disease.
ii. We focus on the Malaysia (NiV_M) strain.
iii. There is an Intensive supportive care for those infected with the virus that goes for treatment.
iv. 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.
v. Nipah virus is seen to be a highly emerging disease and no single drug is available yet for its treatment.
vi. Humans can contact the disease by contact with infected bats, infected pigs or infected humans.
vii. The infected individual can recover either by care giving/combinational therapy or naturally.
viii. One can recover from the infection without treatment.
ix. Once recovered a person or animal cannot get the virus again.
x. We assume the latent class is merge with the infectious class.
xi. Pigs get infected by contact with infectious pigs or infectious bats.

xii. People or pigs can die naturally or as a result of the disease.
xiii. Bats are natural carrier of Nipah virus disease.
xiv. The pigs are caged so that they do not roam to eat defecations from humans that are infected.

Now, putting these assumptions together we have a model flow diagram and thus the model equations for the transmission dynamics of the Nipah virus and its control.

Model variables for the Nipah virus model

S_H - susceptible humans
I_H - infectious humans
I_{HT}- infectious humans receiving treatment
R_H - recovered humans
S_P - susceptible pigs
I_P - infectious pigs
I_{PT}- infectious pigs receiving treatment
R_P - recovered pigs
S_B - Susceptible bats, with strong immune response capable of controlling the infection.
I_B - infectious bats

Parameters and their interpretations for the Nipah virus model

Λ_H - Constant recruitment of human through birth and migration
Λ_P - Constant recruitment of pig through birth and purchase of new pigs into the animal society
Λ_B - Constant recruitment of bats by births and migration of bats into the bat population.
N_H – Total human population
N_P – Total pig population
N_B – Total bat population
μ_H - Natural death rate for humans
μ_P - Natural death rate for pigs
μ_B – Natural death rate for bats.
β_1 - Effective contact rate for humans
β_2- Effective contact rate of pigs
β_3 - Effective contact rate of bats
τ_1 – Proportion of infected human that goes for treatment
τ_2- Proportion of infected pigs taken for treatment
δ_1 -rate at which humans die as a result of the disease in the infectious human class

δ_2- rate at which humans die as a result of the disease in the infectious treated human class undergoing treatment.
δ_3 -rate at which pigs die as a result of the disease in the infectious pig class not undergoing treatment.
δ_4- rate at which pig dies as a result of the disease in the infectious pig class undergoing treatment.

δ_5 -rate at which bats die as a result of human activities.
γ_1 - rate at which infectious human recovers
γ_2- rate at which infectious human undergoing treatment recovers
γ_3 - rate at which infectious pigs recovers
γ_4- rate at which infectious treated pigs recovers
n_0 - modification parameter associated with reduced infectiousness of infected humans, $0 \leq n_0 \leq 1$
n_1 - modification parameter associated with reduced infectiousness of infected pigs as compared to infected humans, $0 \leq n_1 \leq 1$
n_2 - - modification parameter associated with reduced infectiousness of infected pigs as compared to infected humans, $0 \leq n_2 \leq 1$.
λ – this is the force of infection

2.2. Model equations on NiV infection without inhibiting parameters

$$\frac{dS_H}{dt} = \Lambda_H - \frac{\beta_1[\, n_0 I_H + n_1 I_P + n_2 I_B] S_H}{N_H} - \mu_H S_H \,, \quad (1)$$

$$\frac{dI_H}{dt} = \frac{\beta_1[\, n_0 I_H + n_1 I_P + n_2 I_B] S_H}{N_H} - [\gamma_1 + \tau_1 + \mu_H + \delta_1] I_H, \quad (2)$$

$$\frac{dI_{HT}}{dt} = \tau_1 I_H - [\gamma_2 + \mu_H + \delta_2] I_{HT}, \quad (3)$$

$$\frac{dR_H}{dt} = \gamma_1 I_H + \gamma_2 I_{HT} - \mu_H R_H, \quad (4)$$

$$\frac{dS_P}{dt} = \Lambda_P - \frac{\beta_2[I_P + n_2 I_B] S_P}{N_P} - \mu_P S_P, \quad (5)$$

$$\frac{dI_P}{dt} = \frac{\beta_2[I_P + n_2 I_B] S_P}{N_P} - [\gamma_3 + \tau_2 + \mu_P + \delta_3] I_P, \quad (6)$$

$$\frac{dI_{PT}}{dt} = \tau_2 I_P - [\gamma_4 + \mu_P + \delta_4] I_{PT}, \quad (7)$$

$$\frac{dR_P}{dt} = \gamma_3 I_P + \gamma_4 I_{PT} - \mu_P R_P, \quad (8)$$

$$\frac{dS_B}{dt} = \Lambda_B - \frac{\beta_3 I_B S_B}{N_B} - \mu_B S_B \,, \quad (9)$$

$$\frac{dI_B}{dt} = \frac{\beta_3 I_B S_B}{N_B} - [\mu_B + \delta_5] I_B. \quad (10)$$

3. Positivity of solutions

Since there is a portion of the system of equations in (4.1) to (4.10) represents the non-inhibitor populations, it is necessary to reflect on the fact that the associated population sizes can never be negative. Thus, we establish the positive invariance of Ω_1 to be given as:

$\Omega_1 = \{ (S_H(t), I_H(t), I_{HT}(t), R_H(t), S_P(t), I_P(t), I_{PT}(t), R_P(t), S_B(t), I_B(t)) \in \Re_+^{10} \}$ then
$\{S_H(t), I_H(t), I_{HT}(t), R_H(t), S_P(t), I_P(t), I_{PT}(t), R_P(t), S_B(t), I_B(t) \text{ and } N_1(t)\} \geq 0, \forall\, t \geq 0$

There is need for all the variables to be non-negative for all time. This analysis ensures that the model is well posed and that it is realistic in representing the populations with non-negative values. A Mathematical equation is said to be well posed, if it has a solution and that solution should be unique and the solution must depend continuously on the data and parameters [9]; [2].

Theorem 1: The solutions set for the non-inhibitor sub-model $(S_H(t), I_H(t), I_{HT}(t), R_H(t), S_P(t), I_P(t), I_{PT}(t), R_P(t), S_B(t), I_B(t)) \in \Re_+^{10}$, are contained in the feasible solution Ω_1. We describe the feasible solution to be such a state, where at the origin of the disease, the $(S_H(t), I_H(t), I_{HT}(t), R_H(t), S_P(t), I_P(t), I_{PT}(t), R_P(t), S_B(t), I_B(t))$ are non-zero;

Proof: Let $\{S_H(t), I_H(t), I_{HT}(t), R_H(t), S_P(t), I_P(t), I_{PT}(t), R_P(t), S_B(t), I_B(t) \ and \ N_1(t)\} \geq 0, \forall\, t \geq 0$.

We show that Ω_1 is positively invariant so that it is sufficient to consider the dynamics of the model system

From (1):

$$\frac{dS_H}{dt} = \Lambda_H - \frac{\beta_1[\,\eta_0 I_H + \eta_1 I_P + \eta_2 I_B]S_H}{N_H} - \mu_H S_H$$
$$\Rightarrow \frac{dS_H}{dt} \geq -\left[\frac{\beta_1[\,\eta_0 I_H + \eta_1 I_P + \eta_2 I_B]}{N_H} + \mu_H\right] S_H \ .$$

Next, we integrate both sides

$$\int \frac{dS_H}{S_H} \geq -\int \left[\frac{\beta_1[\,\eta_0 I_H + \eta_1 I_P + \eta_2 I_B]}{N_H} + \mu_H\right] dt$$
$$\ln S_H \geq -\left[\frac{\beta_1[\,\eta_0 I_H + \eta_1 I_P + \eta_2 I_B]}{N_H} + \mu_H\right] t + S(t)$$

Take exponential of both sides

$$S_H \geq S_{H1}(t) e^{-\left[\frac{\beta_1[\,\eta_0 I_H + \eta_1 I_P + \eta_2 I_B]}{N_H} + \mu_H\right] t} \ .$$

At t=0,

$$S_H \geq S(0) \ .$$

This implies that

$$S_H \geq 0.$$

Similarly, we do this approach for the other variables, by this we have shown that all the solutions of (1 to 10) are in $\Re_+^{10}$, provided that the initial conditions are positive. Thus, our feasible region, Ω_1 is positively invariant. Therefore, the model is mathematically well posed and epidemiologically reasonable. Hence, it is sufficient to consider the dynamic of the model (1 to 10) in Ω_1 [7].

4. Disease free equilibrium (DFE)

Using the sub-model for the human without inhibitor as control, the force of infection for the non-inhibitor sub-model is given to be $\lambda_H = \frac{\beta_1[n_0 I_H + n_1 I_P + n_2 I_B]S_H}{N_H}$

Before we go further into the analysis, we must first determine the equilibrium points of the modelled system of equations.

To find the equilibrium point(s) for the non-inhibitor human compartment, we need to set the above equations (1), (2), (3), and (4) to zero, that is:

$$\frac{dS_H}{dt} = 0, \frac{dI_H}{dt} = 0, \frac{dI_{HT}}{dt} = 0, \frac{dR_H}{dt} = 0,$$

we have the following:

$$0 = \Lambda_H - \frac{\beta_1[n_0 I_H + n_1 I_P + n_2 I_B]S_H}{N_H} - \mu_H S_H, \quad (11)$$

$$0 = \frac{\beta_1[n_0 I_H + n_1 I_P + n_2 I_B]S_H}{N_H} - [\gamma_1 + \tau_1 + \mu_H + \delta_1]I_H, \quad (12)$$

$$0 = \tau_1 I_H - [\gamma_2 + \mu_H + \delta_2]I_{HT}, \quad (13)$$

$$0 = \gamma_1 I_H + \gamma_2 I_{HT} - \mu_H R_H \quad (14)$$

From (11), we replace β_1 with $q_1 I_H$, then solve for S_H

$$S_H = \frac{\Lambda_H N_H}{N_H \mu_H + q_1 I_H[n_0 I_H + n_1 I_P + n_2 I_B]} \quad (15)$$

From (12), we let $\beta_1 = q_1 I_H$, then solve for I_H

$$0 = \left(\frac{q_1[n_0 I_H + n_1 I_P + n_2 I_B]S_H}{N_H} - [\gamma_1 + \tau_1 + \mu_H + \delta_1]\right) I_H \quad (16)$$

From (16), we found $I_H = 0$ or $\left(\frac{q_1[n_0 I_H + n_1 I_P + n_2 I_B]S_H}{N_H} - [\gamma_1 + \tau_1 + \mu_H + \delta_1]\right) = 0$, considering this result from (16), it shows us that we would have two equilibrium points.

$$\Rightarrow \left(\frac{q_1 n_0 I_H S_H}{N_H} + \frac{q_1 n_1 I_P S_H}{N_H} + \frac{q_1 n_2 I_B S_H}{N_H} - [\gamma_1 + \tau_1 + \mu_H + \delta_1]\right) = 0$$

$$I_H = \left(\frac{N_H[\gamma_1 + \tau_1 + \mu_H + \delta_1] - [n_1 I_P + n_2 I_B]q_1 S_H}{q_1 n_0 S_H}\right).$$

Now, substitute the new value of S_H in (15) into (12).

$$\Rightarrow I_H = 0 \; or \; I_H = \left(\frac{N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1\Lambda_H}{q_1 n_0 \Lambda_H}\right).$$

Now we input $I_H = 0$ into (13), (14) and (15) to get the values for I_{HT}, R_H and S_H respectively.

For (13),

$$0 = \tau_1 I_H - [\gamma_2 + \mu_H + \delta_2] I_{HT}$$
$$I_{HT} = \frac{\tau_1 I_H}{[\gamma_2+\mu_H+\delta_2]},$$
$$I_{HT} = \frac{\tau_1 (0)}{[\gamma_2+\mu_H+\delta_2]} = 0$$
$$\therefore I_{HT} = 0.$$

For (14), we solve for R_H and then substitute $I_H = 0$ and $I_{HT} = 0$ into the result

$$0 = \gamma_1 I_H + \gamma_2 I_{HT} - \mu_H R_H$$
$$R_H = \frac{\gamma_1 I_H + \gamma_2 I_{HT}}{\mu_H}$$
$$\Rightarrow R_H = \frac{\gamma_1(0)+\gamma_2(0)}{\mu_H} = 0$$

Then $R_H = 0$.

Next, we substitute $I_H = 0$ into (15)

$$S_H = \frac{\Lambda_H N_H}{N_H \mu_H + q I_H[n_0 I_H + n_1 I_P + n_2 I_B]}$$
$$S_H = \frac{\Lambda_H N_H}{N_H \mu_H + q(0)[n_0(0) + n_1 I_P + n_2 I_B]}$$
$$\Rightarrow S_H = \frac{\Lambda_H}{\mu_H}$$

Thus, we have the values for S_H, I_H, I_{HT}, and R_H.

Hence the first equilibrium point (which is popularly referred to as the DFE), to be given as:
$(S_H, I_H, I_{HT}, R_H) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0\right)$.

For the second equilibrium point, we substitute the value $I_H^* = \left(\frac{N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H}{q_1 n_0 \Lambda_H}\right)$ into the following (13), (14), and (15) to get new values for I_{HT}, R_H, and S_H.

$$I_H^* = \left(\frac{N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H}{q_1 n_0 \Lambda_H}\right) = W_2.$$

For (13),

$$0 = \tau_1 I_H^* - [\gamma_2 + \mu_H + \delta_2] I_{HT}^*$$

$$I_{HT}^* = \frac{\tau_1 I_H^*}{[\gamma_2+\mu_H+\delta_2]},$$

$$I_{HT}^* = \frac{\tau_1(N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H)}{q_1 n_0 \Lambda_H\ [\gamma_2+\mu_H+\delta_2]} = W_3.$$

For (14), we solve for R_H and then substitute $I_H^* = \left(\frac{N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H}{q_1 n_0 \Lambda_H}\right)$ and $I_{HT}^* = \frac{\tau_1(N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H)}{q_1 n_0 \Lambda_H\ [\gamma_2+\mu_H+\delta_2]}$ into (14).

$$0 = \gamma_1 I_H^* + \gamma_2 I_{HT}^* - \mu_H R_H^*$$

$$R_H^* = \frac{\gamma_1 I_H^* + \gamma_2 I_{HT}^*}{\mu_H}$$

$$R_H^* = \frac{\gamma_1(N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H)\,[\gamma_2+\mu_H+\delta_2] + \gamma_2(\,\tau_1(N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H))}{\mu_H(q_1 n_0 \Lambda_H)\ [\gamma_2+\mu_H+\delta_2]}$$

$$R_H^* = W_4.$$

For (15), we substitute $I_H^* = \left(\frac{N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H}{q_1 n_0 \Lambda_H}\right)$ into (15):

$$S_H^* = \frac{\Lambda_H\ N_H}{N_H\,\mu_H + q_1\left(\frac{N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H}{q_1 n_0 \Lambda_H}\right)\left[\, n_0\left(\frac{N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H}{q_1 n_0 \Lambda_H}\right)+n_1 I_P+ n_2 I_B\right]}$$

$$S_H^* = \frac{\Lambda_H\ N_H\ q_1 n_0 \Lambda_H}{(N_H\,\mu_H)q_1 n_0 \Lambda_H + q_1(N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H)[\, n_0(\,N_H[\gamma_1+\tau_1+\mu_H+\delta_1]-[n_1 I_P+n_2 I_B]q_1 n_0 \Lambda_H)+(n_1 I_P+ n_2 I_B)\, q_1 n_0 \Lambda_H]}$$

We then let $S_H^* = W_1$. Now, we have the values for S_H^*, I_H^*, I_{HT}^*, and R_H^* .

Hence the second equilibrium point (that is the EE), which is given as:
$S_H^*, I_H^*, I_{HT}^*, R_H^*) = (W_1, W_2, W_3, W_4)$.

We call the first equilibrium point the disease free equilibrium (DFE), while the second equilibrium point is called the endemic equilibrium (EE).

Since we have gotten the equilibrium points for our non-linear system of equation for the uncontrolled disease sub-model for human, we will then analyze these equilibrium points, as they are necessary for our study, because it is at these points, that we will discuss the stability of the modelled system.

Next, we find the equilibrium for the pig compartment of the non-inhibitor compartment:

$$\frac{dS_P}{dt} = \Lambda_P - \frac{\beta_2[I_P + n_2 I_B]S_P}{N_P} - \mu_P S_P, \quad (5)$$
$$\frac{dI_P}{dt} = \frac{\beta_2[I_P + n_2 I_B]S_P}{N_P} - [\gamma_3 + \tau_2 + \mu_P + \delta_3]I_P, \quad (6)$$
$$\frac{dI_{PT}}{dt} = \tau_2 I_P - [\gamma_4 + \mu_P + \delta_4]I_{PT}, \quad (7)$$
$$\frac{dR_P}{dt} = \gamma_3 I_P + \gamma_4 I_{PT} - \mu_P R_P . \quad (8)$$

At this point of our discussion, we replace β_2 with gI_P, which is the rate at which susceptible pigs become exposed to the disease.

To find the equilibrium point, we need to set the above equations (5), (6), (7), (8) to zero, that is:

$$\frac{dS_P}{dt} = 0, \frac{dI_P}{dt} = 0, \frac{dR_P}{dt} = 0,$$
$$0 = \Lambda_P - \frac{\beta_2[n_1 I_P + n_2 I_B]S_P}{N_P} - \mu_P S_P, \quad (17)$$
$$0 = \frac{\beta_2[n_1 I_P + \eta_2 I_B]S_P}{N_P} - [\gamma_3 + \tau_2 + \mu_P + \delta_3]I_P, \quad (18)$$
$$0 = \tau_2 I_P - [\gamma_4 + \mu_P + \delta_4]I_{PT}, \quad (19)$$

$$0 = \gamma_3 I_P + \gamma_4 I_{PT} - \mu_P R_P. \quad (20)$$

From (17) we solve for S_P:

$$S_P = \frac{\Lambda_P N_P}{N_P \mu_P + \beta_2[n_1 I_P + n_2 I_B]} \quad (21)$$

We let $\beta_2 = q_2 I_P$, then we solve for I_P

$$0 = \left(\frac{q_2[n_1 I_P + \eta_2 I_B]S_P}{N_P} - [\gamma_3 + \tau_2 + \mu_P + \delta_3]\right) I_P. \quad (22)$$

For $\left(\frac{q_2[n_1 I_P + \eta_2 I_B]S_P}{N_P} - [\gamma_3 + \tau_2 + \mu_P + \delta_3]\right) = 0$,

$$\Rightarrow \left(\frac{q_2 n_1 I_P S_H}{N_P} + \frac{q_2 n_2 I_B S_H}{N_P} - [\gamma_1 + \tau_1 + \mu_H + \delta_1]\right) = 0$$

$$I_P = \left(\frac{N_P[\gamma_3 + \tau_2 + \mu_P + \delta_3] - [n_1 I_P + n_2 I_B]q_2 n_1 S_P}{q_2 n_1 S_P}\right).$$

From (38), we found $I_P = 0$ or $0 = \left(\frac{q_2[n_1 I_P + \eta_2 I_B]S_P}{N_P} - [\gamma_3 + \tau_2 + \mu_P + \delta_3]\right)$ considering this result from (38), it shows us that we would have two equilibrium points, one for $I_P = 0$ and the other when $I_P = \left(\frac{N_P[\gamma_3+\tau_2+\mu_P+\delta_3]-[n_1 I_P+n_2 I_B]q_2 n_1 S_P}{q_2 n_1 S_P}\right)$.

Now, substitute the new value of S_P in (21) into the other value for I_P:

$$I_P^* = \frac{q_2\,[\,n_1 I_P + n_2 I_B]\Lambda_P \;\; -[\gamma_3+\tau_2+\mu_P+\delta_3]\,N_P\,\mu_P}{q_2\,[\,n_1 I_P + n_2 I_B]} = \; W_6 \; . \qquad (23)$$

Now we input $I_P = 0$ into (19), (20) and (21) to get the values for I_{HT}, R_H and S_H respectively.

For (19),

$$0 = \;\; \tau_2 I_P - [\gamma_4 + \mu_P + \delta_4] I_{PT},$$
$$I_{PT} = \frac{\tau_2 I_P}{[\gamma_4+\mu_P+\delta_4]} \;\; ,$$
$$I_{PT} = \frac{\tau_2\,(0)}{[\gamma_4+\mu_P+\delta_4]} = 0,$$
$$\therefore \; I_{PT} = \; 0\,.$$

For (20), we solve for R_P and then substitute $I_P = 0$ and $I_{PT} = 0$ into the result

$$0 = \; \gamma_3 I_P + \; \gamma_4 I_{PT} \; - \; \mu_P R_P,$$
$$R_P = \frac{\gamma_3 I_P + \gamma_4 I_{PT}}{\mu_P}$$
$$\Rightarrow \;\; R_P = \frac{\gamma_3(0) + \gamma_4(0)}{\mu_P} = 0$$

Then $R_P = 0$.

Next, we substitute $I_P = \; 0$ into (15):

$$S_P = \frac{\Lambda_P \;\; N_P}{N_P \;\mu_P + \; q_2 I_P[\,n_1 I_P + n_2 I_B]}$$
$$S_P = \frac{\Lambda_P \;\; N_P}{N_P \;\mu_P + \; q_2(0)[n_1(0) + n_2 I_B]}$$
$$\Rightarrow \; S_P = \frac{\Lambda_P}{\mu_P}.$$

Thus, we have the values for S_P, I_P, I_{PT}, and R_P . Hence the first equilibrium point (that is the DFE), which is given as:

$$(S_P\,, I_P, I_{PT}, R_P) = \left(\frac{\Lambda_P}{\mu_P}\,, 0, 0, 0\right).$$

For the second equilibrium point, we substitute (23) into the following (19), (20), and (21) to get new values for I_{PT}, R_P, and S_P

For (19),

$$0 = \tau_2 I_P^* - [\gamma_4 + \mu_P + \delta_4] I_{PT}^*$$
$$I_{PT}^* = \frac{\tau_2 I_P^*}{[\gamma_4+\mu_P+\delta_4]} ,$$
$$I_{PT}^* = \frac{\tau_2(q_2 [n_1 I_P + n_2 I_B]\Lambda_H - [\gamma_3+\tau_2+\mu_P+\delta_4] N_P \mu_P)}{q_2 [n_1 I_P + n_2 I_B] [\gamma_4+\mu_P+\delta_4]} = W_7.$$

For (20), we solve for R_H and then substitute $I_P^* = \frac{q_2 [n_1 I_P + n_2 I_B]\Lambda_P - [\gamma_1+\tau_1+\mu_H+\delta_1] N_H \mu_H}{q_2 [n_0 I_H + n_1 I_P + n_2 I_B]}$ and $I_{HT}^* = \frac{\tau_2(q_2 [n_1 I_P + n_2 I_B]\Lambda_P - [\gamma_3+\tau_2+\mu_P+\delta_3] N_P \mu_P)}{q_2 [n_1 I_P + n_2 I_B] [\gamma_4+\mu_P+\delta_4]}$ into the result

$$0 = \gamma_3 I_P^* + \gamma_4 I_{PT}^* - \mu_P R_P^*$$
$$R_P^* = \frac{\gamma_3 I_P^* + \gamma_4 I_{PT}^*}{\mu_P}$$
$$R_P^* = \frac{\gamma_2(q_2 [n_1 I_P + n_2 I_B]\Lambda_P - [\gamma_3+\tau_2+\mu_P+\delta_3] N_H \mu_H) [\gamma_2+\mu_H+\delta_2] + \gamma_2(\tau_1(q_2 [n_1 I_P + n_2 I_B]\Lambda_P - [\gamma_3+\tau_2+\mu_P+\delta_3] N_P \mu_P))}{\mu_P(q_2 [n_1 I_P + n_2 I_B] [\gamma_4+\mu_P+\delta_4])}$$
$$R_H^* = W_8 .$$

For (21), we substitute $I_P^* = \frac{q_2 [n_1 I_P + n_2 I_B]\Lambda_P - [\gamma_3+\tau_2+\mu_P+\delta_3] N_P \mu_P}{q_2 [n_1 I_P + n_2 I_B]}$ into (21):

$$S_P^* = \frac{\Lambda_P N_P}{N_P \mu_P + q_2 I_P [n_1 I_P + n_2 I_B]}$$
$$S_P^* = \frac{\Lambda_P N_P ([n_1 I_P + n_2 I_B])}{N_P \mu_P ([n_1 I_P + n_2 I_B]) + (q_2 [n_1 I_P + n_2 I_B]\Lambda_P - [\gamma_3+\tau_2+\mu_P+\delta_3] N_P \mu_P)[n_1 I_P + n_2 I_B]}$$
$$\Rightarrow S_P^* = \frac{\Lambda_P N_P}{N_P \mu_P + (q_2 [n_1 I_P + n_2 I_B]\Lambda_P - [\gamma_3+\tau_2+\mu_P+\delta_3] N_P \mu_P)} = W_5 .$$

Now, we have the values for S_P^*, I_P^*, I_{PT}^*, and R_P^* . Hence the second equilibrium point (that is the EE), which is given as:
$(S_P^* , I_P^*, I_{PT}^*, R_P^*) = (W_5, W_6, W_7, W_8)$.

Similarly, we find the equilibrium point for the bat compartment. Recall

$\lambda_B = \frac{\beta_3 I_B S_P}{N_P}$ = FOI for bats

$$\frac{dS_B}{dt} = \Lambda_P - \frac{\beta_3 I_B S_B}{N_B} - \mu_B S_B \qquad (9)$$

$$\frac{dI_B}{dt} = \frac{\beta_3 n_2 I_B S_B}{N_B} - [\mu_B + \delta_5]I_B \ . \qquad (10)$$

At this point of our discussion, we replace β_3 with $q_3 I_B$, which is the rate at which susceptible bats become exposed to the disease.

To find the equilibrium point or fixed point, we need to set the above equations (9), (10) to zero, that is:

$$\frac{dS_B}{dt} = 0, \frac{dI_B}{dt} = 0, \frac{dB}{dt} = 0$$

$$0 = \Lambda_B - \frac{\beta_3 n_2 I_B S_B}{N_B} - \mu_B S_B \qquad (24)$$

$$0 = \frac{\beta_3 n_2 I_B S_B}{N_B} - [\mu_B + \delta_5]I_B \ . \qquad (25)$$

From (24) we have the following:

$$0 = \Lambda_B - \frac{\beta_3 n_2 I_B S_B}{N_B} - \mu_B S_B$$

$$S_B = \frac{\Lambda_B N_B}{N_B \mu_B + \beta_3 n_2 I_B} \ . \qquad (26)$$

Now, we let $\beta_3 = q_3 I_B$, then we solve for I_B in (25)

$$0 = \frac{q_3 I_B n_2 I_B S_B}{N_B} - [\mu_B + \delta_5]I_B$$

$$0 = \left(\frac{q_3 I_B n_2 S_P}{N_B} - [\mu_B + \delta_5]\right) I_B$$

$$\Rightarrow I_B = 0 \ and \ 0 = \left(\frac{q_3 I_B n_2 S_P}{N_P} - [\mu_B + \delta_5]\right).$$

For $\left(\frac{q_3 I_B n_2 S_P}{N_B} - [\mu_B + \delta_5]\right) = 0$

$$\Rightarrow \left(\frac{q_3 I_B n_1 S_P}{N_B} - [\mu_B + \delta_5]\right) = 0$$

$$I_B = \left(\frac{N_B[\mu_B + \delta_5]}{q_3 n_2 S_B}\right).$$

From (25), we found $I_B = 0$ or $0 = \left(\frac{q_3[n_2 I_B] S_B}{N_B} - [\mu_B + \delta_5]\right)$ considering this result from (25), it shows us that we would have two equilibrium points, one for $I_B = 0$ and when $I_B = \left(\frac{N_B[\mu_B + \delta_5]}{q_3 n_2 S_B}\right)$

Now, substitute (26) into the other value for I_B:

$$I_B^* = \left(\frac{N_B([\mu_B+\delta_5]N_B\,\mu_B+[\mu_B+\delta_5]\,q_3n_2I_B)}{q_3n_2\Lambda_B\;N_B}\right) = \; W_{10}\,. \qquad (27)$$

Now we input $I_P = 0$ into (26) to get the value for S_H. For (26),

$$S_B = \frac{\Lambda_B\;N_B}{N_B\,\mu_B+\,q_3\,I_Bn_2I_B}$$
$$S_B = \frac{\Lambda_B\;N_B}{N_B\,\mu_B+\,q_3\,(0)n_2(0)}$$
$$\Rightarrow S_B = \frac{\Lambda_B}{\mu_B}\;.$$

Hence the first equilibrium point is given as $(S_B\,, I_B) = \left(\frac{\Lambda_B}{\mu_B}\,, 0\right)$. Next, we compute the second equilibrium point

Put $I_B^* = \left(\frac{N_B([\mu_B+\delta_5]N_B\,\mu_B+[\mu_B+\delta_5]\,q_3n_2I_B)}{q_3n_2\Lambda_B\;N_B}\right)$ into (26) to find the endemic value for S_B

$$S_B^* = \frac{\Lambda_B\;N_B\,(q_3n_2\Lambda_B\;N_B)^2}{N_B\,\mu_B(q_3n_2\Lambda_B\;N_B)^2+\,q_3\,n_2(N_B([\mu_B+\delta_5]N_B\,\mu_B+[\mu_B+\delta_5]\,q_3n_2I_B))^2}.$$

Therefore, the second equilibrium point the bat compartment is:

$$(\,S_B^*\,, I_B^*) = \left(\frac{\Lambda_B\;N_B\,(q_3n_2\Lambda_B\;N_B)^2}{N_B\,\mu_B(q_3n_2\Lambda_B\;N_B)^2+\,q_3\,n_2(N_B([\mu_B+\delta_5]N_B\,\mu_B+[\mu_B+\delta_5]\,q_3n_2I_B))^2}\,, \left(\frac{N_B([\mu_B+\delta_5]N_B\,\mu_B+[\mu_B+\delta_5]\,q_3n_2I_B)}{q_3n_2\Lambda_B\;N_B}\right)\right).$$

Hence, the disease free equilibrium, E^0, (DFE) for the non-inhibitor Niv model are:

$$(S_H(0)^0, I_H(0)^0, I_{HT}(0)^0, R_H(0)^0, S_P(0)^0, I_P(0)^0, I_{PT}(0)^0, R_P(0)^0, S_B(0)^0, I_B(0)^0\,) =$$
$$\left(\frac{\Lambda_H}{\mu_H}\,, 0, 0, 0, \frac{\Lambda_P}{\mu_P}\,, 0, 0, 0, \frac{\Lambda_B}{\mu_B}\,, 0\;\right)$$

where, the endemic equilibrium, E^*, (EE) for the non-inhibitor Niv model are:

$$(S_H(0)^*, I_H(0)^*, I_{HT}(0)^*, R_H(0)^*, S_P(0)^*, I_P(0)^*, I_{PT}(0)^*, R_P(0)^*, S_B(0)^*, I_B(0)^*) =$$
$$\left(\frac{[\gamma_1+\tau_1+\mu_H+\delta_1]N_H}{q[n_0I_H+n_1I_P+n_2I_B]}, \frac{\Lambda_H q[n_0I_H+n_1I_P+n_2I_B]-[\gamma_1+\tau_1+\mu_H+\delta_1]N_H}{q[n_0I_H+n_1I_P+n_2I_B]}, \frac{\tau_1(\Lambda_H q[n_0I_H+n_1I_P+n_2I_B]-[\gamma_1+\tau_1+\mu_H+\delta_1]N_H)}{q[n_0I_H+n_1I_P+n_2I_B][\gamma_2+\mu_H+\delta_2]}, \right.$$
$$\gamma_1 \frac{\Lambda_H q[n_0I_H+n_1I_P+n_2I_B]-[\gamma_1+\tau_1+\mu_H+\delta_1]N_H}{q[n_0I_H+n_1I_P+n_2I_B]\mu_H}, \frac{[\gamma_3+\tau_2+\mu_P+\delta_3]N_P}{g[n_1I_P+n_2I_B]}, \frac{\Lambda_P g[I_P+n_2I_B]-[\gamma_3+\tau_2+\mu_P+\delta_3]T_P}{g[n_1I_P+n_2I_B]}, \tau_2 \frac{\Lambda_P g[n_1I_P+n_2I_B]-[\gamma_3+\tau_2+\mu_P+\delta_3]N_P}{g[n_1I_P+n_2I_B][\gamma_4+\mu_P+\delta_4]},$$
$$\left. \gamma_3 \frac{\Lambda_P g[n_1I_P+n_2I_B]-[\gamma_3+\tau_2+\mu_P+\delta_3]T_P}{g[n_1I_P+n_2I_B]\mu_P}, \frac{[\mu_B+\delta_5]N_B}{\beta_3}, \left[\frac{\Lambda_B-S_B\mu_B}{S_B} \right] \frac{N_B}{\beta_3} \right)$$

5. The basic reproductive number (R_0) for the study

Epidemiologists calculate R_0 using various approaches, like individual-level contact tracing data obtained at the beginning of the pandemic. Once a person is diagnosed, his/her contacts are traced and tested. R_0 is then figured out by averaging over the number of secondary cases of all diagnosed individuals. If one or more infected person(s), is introduced into the community, the disease could either spread or die out [10]. This approach is based upon the definition of R_0, but it does not ensure that the calculated R_0 is also an epidemic doorstep parameter.

Now, rewriting the inhibitor free model, starting with the disease compartment, we have:

$$\frac{dI_H}{dt} = \frac{\beta_1[n_0I_H+n_1I_P+n_2I_B]S_H}{N_H} - [\gamma_1+\tau_1+\mu_H+\delta_1]I_H,$$
$$\frac{dI_{HT}}{dt} = \tau_1 I_H - [\gamma_2+\mu_H+\delta_2]I_{HT},$$
$$\frac{dI_P}{dt} = \frac{\beta_2[I_P+n_2I_B]S_P}{N_P} - [\gamma_3+\tau_2+\mu_P+\delta_3]I_P,$$
$$\frac{dI_{PT}}{dt} = \tau_2 I_P - [\gamma_4+\mu_P+\delta_4]I_{PT},$$
$$\frac{dI_B}{dt} = \frac{\beta_3 n_2 I_B S_B}{N_B} - [\mu_B+\delta_5]I_B$$
$$\frac{dS_H}{dt} = \Lambda_H - \frac{\beta_1[n_0I_H+n_1I_P+n_2I_B]S_H}{N_H} - \mu_H S_H,$$
$$\frac{dS_P}{dt} = \Lambda_P - \frac{\beta_2[I_P+n_2I_B]S_P}{N_P} - \mu_P S_P$$
$$\frac{dS_B}{dt} = \Lambda_B - \frac{\beta_3 n_2 I_B S_B}{N_B} - \mu_B S_B$$
$$\frac{dR_H}{dt} = \gamma_1 I_H + \gamma_2 I_{HT} - \mu_H R_H$$
$$\frac{dR_P}{dt} = \gamma_3 I_P + \gamma_4 I_{PT} - \mu_P R_P.$$

From the above model of our study, we have:

X = vector of the infected classes, such as exposed, carrier, infectious etc.
Y = vector of uninfected classes, such as susceptible, recovered etc.

Therefore,
$$X = \begin{pmatrix} I_H \\ I_{HT} \\ I_P \\ I_{PT} \\ I_B \end{pmatrix},\ Y = \begin{pmatrix} S_H \\ S_P \\ S_B \\ R_H \\ R_P \end{pmatrix}$$

$$f = \begin{bmatrix} f_1 \\ f_2 \\ f_3 \\ f_4 \\ f_5 \end{bmatrix},\ v = \begin{bmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \end{bmatrix}$$

$$f = \begin{bmatrix} \frac{\beta_1[\,n_0 I_H + n_1 I_P + n_2 I_B]S_H}{N_H} \\ 0 \\ \frac{\beta_2[n_1 I_P + n_2 I_B]S_P}{N_P} \\ 0 \\ \frac{\beta_3 n_2 I_B\, S_B}{N_B} \end{bmatrix},\quad v = \begin{bmatrix} [\gamma_1+\tau_1+\mu_H+\delta_1]I_H \\ -\tau_1 I_H + [\gamma_2+\mu_H+\delta_2]I_{HT} \\ [\gamma_3+\tau_2+\mu_P+\delta_3]I_P \\ -\tau_2 I_P + [\gamma_4+\mu_P+\delta_4]I_{PT} \\ [\mu_B+\delta_5]I_B \end{bmatrix},$$

$$F = \begin{bmatrix} \frac{\partial f_1}{\partial I_H} & \frac{\partial f_1}{\partial I_{HT}} & \frac{\partial f_1}{\partial I_P} & \frac{\partial f_1}{\partial I_{PT}} & \frac{\partial f_1}{\partial I_B} \\ \frac{\partial f_2}{\partial I_H} & \frac{\partial f_2}{\partial I_{HT}} & \frac{\partial f_2}{\partial I_P} & \frac{\partial f_2}{\partial I_{PT}} & \frac{\partial f_2}{\partial I_B} \\ \frac{\partial f_3}{\partial I_H} & \frac{\partial f_3}{\partial I_{HT}} & \frac{\partial f_3}{\partial I_P} & \frac{\partial f_3}{\partial I_{PT}} & \frac{\partial f_3}{\partial I_B} \\ \frac{\partial f_4}{\partial I_H} & \frac{\partial f_4}{\partial I_{HT}} & \frac{\partial f_4}{\partial I_P} & \frac{\partial f_4}{\partial I_{PT}} & \frac{\partial f_4}{\partial I_B} \\ \frac{\partial f_5}{\partial I_H} & \frac{\partial f_5}{\partial I_{HT}} & \frac{\partial f_5}{\partial I_P} & \frac{\partial f_5}{\partial I_{PT}} & \frac{\partial f_5}{\partial I_B} \end{bmatrix},\ V = \begin{bmatrix} \frac{\partial V_1}{\partial I_H} & \frac{\partial V_1}{\partial I_{HT}} & \frac{\partial V_1}{\partial I_P} & \frac{\partial V_1}{\partial I_{PT}} & \frac{\partial V_1}{\partial I_B} \\ \frac{\partial V_2}{\partial I_H} & \frac{\partial V_2}{\partial I_{HT}} & \frac{\partial V_2}{\partial I_P} & \frac{\partial V_2}{\partial I_{PT}} & \frac{\partial V_2}{\partial I_B} \\ \frac{\partial V_3}{\partial I_H} & \frac{\partial V_3}{\partial I_{HT}} & \frac{\partial V_3}{\partial I_P} & \frac{\partial V_3}{\partial I_{PT}} & \frac{\partial V_3}{\partial I_B} \\ \frac{\partial V_4}{\partial I_H} & \frac{\partial V_4}{\partial I_{HT}} & \frac{\partial V_4}{\partial I_P} & \frac{\partial V_4}{\partial I_{PT}} & \frac{\partial V_4}{\partial I_B} \\ \frac{\partial V_5}{\partial I_H} & \frac{\partial V_5}{\partial I_{HT}} & \frac{\partial V_5}{\partial I_P} & \frac{\partial V_5}{\partial I_{PT}} & \frac{\partial V_5}{\partial I_B} \end{bmatrix}.$$

Therefore, we have

$$F = \begin{bmatrix} \frac{\beta_1 n_0\, S_H}{N_H} & 0 & \frac{\beta_1 n_1\, S_H}{N_H} & 0 & \frac{\beta_1 n_2\, S_H}{N_H} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_2 n_1 S_P}{N_P} & 0 & \frac{\beta_2 n_2 S_P}{N_P} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_3 n_2 S_B}{N_B} \end{bmatrix},$$

$$V = \begin{bmatrix} [\gamma_1+\tau_1+\mu_H+\delta_1] & 0 & 0 & 0 & 0 \\ 0 & -\tau_1 I_H + [\gamma_2+\mu_H+\delta_2] & 0 & 0 & 0 \\ 0 & 0 & [\gamma_3+\tau_2+\mu_P+\delta_3] & 0 & 0 \\ 0 & 0 & -\tau_2 & [\gamma_4+\mu_P+\delta_4] & 0 \\ 0 & 0 & 0 & 0 & [\mu_B+\delta_5] \end{bmatrix}.$$

Next, we evaluate F at DFE. Therefore, we have

$$F = \begin{bmatrix} \frac{\beta_1 n_0 \Lambda_H}{N_H \mu_H} & 0 & \frac{\beta_1 n_1 \Lambda_H}{N_H \mu_H} & 0 & \frac{\beta_1 n_2 \Lambda_H}{N_H \mu_H} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_2 n_1 \Lambda_P}{N_P \mu_P} & 0 & \frac{\beta_2 n_2 \Lambda_P}{N_P \mu_P} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_3 n_2 \Lambda_B}{N_B \mu_B} \end{bmatrix},$$

$$V = \begin{bmatrix} [\gamma_1+\tau_1+\mu_H+\delta_1] & 0 & 0 & 0 & 0 \\ 0 & [\gamma_2+\mu_H+\delta_2] & 0 & 0 & 0 \\ 0 & 0 & [\gamma_3+\tau_2+\mu_P+\delta_3] & 0 & 0 \\ 0 & 0 & -\tau_2 & [\gamma_4+\mu_P+\delta_4] & 0 \\ 0 & 0 & 0 & 0 & [\mu_B+\delta_5] \end{bmatrix}.$$

We then compute V^{-1}, using Maple 2019:

$$\begin{bmatrix} \left[\frac{1}{\gamma 1 + \tau 1 + \mu H + \delta 1}, 0, 0, 0, 0\right], \\ \left[0, \frac{1}{\gamma 2 + \mu H + \delta 2}, 0, 0, 0\right], \\ \left[0, 0, \frac{1}{\gamma 3 + \tau 2 + \mu P + \delta 3}, 0, 0\right], \\ \left[0, 0, \frac{\tau 2}{(\gamma 3 + \tau 2 + \mu P + \delta 3)(\gamma 4 + \mu P + \delta 4)}, \frac{1}{\gamma 4 + \mu P + \delta 4}, 0\right], \\ \left[0, 0, 0, 0, \frac{1}{\mu B + \delta 5}\right] \end{bmatrix}$$

Since we have gotten V^{-1} and we also have F, then, we can then compute the Next Generation Matrix (NGM) for the uncontrolled disease model.

$$\left[\left[\frac{\beta 1(n0)\,\Lambda H}{(\gamma 1+\tau 1+\mu H+\delta 1)\,NH(\mu H)},\ 0,\ \frac{\beta 1(n1)\,\Lambda H}{(\gamma 1+\tau 1+\mu H+\delta 1)\,NH(\mu H)},\ 0,\ \frac{\beta 1(n2)\,\Lambda H}{(\gamma 1+\tau 1+\mu H+\delta 1)\,NH(\mu H)}\right],\ \left[0,0,0,0,0\right],\ \left[0,0,\frac{\beta 2(n1)\,\Lambda P}{(\gamma 3+\tau 2+\mu P+\delta 3)\,NP(\mu P)},0,\frac{\beta 1(n2)\,\Lambda P}{(\gamma 3+\tau 2+\mu P+\delta 3)\,NP(\mu P)}\right],\ \left[0,0,\frac{\tau 2\,\beta 2(n1)\,\Lambda P}{(\gamma 3+\tau 2+\mu P+\delta 3)\,(\gamma 4+\mu P+\delta 4)\,NP(\mu P)},0,\frac{\tau 2\,\beta 1(n2)\,\Lambda P}{(\gamma 3+\tau 2+\mu P+\delta 3)\,(\gamma 4+\mu P+\delta 4)\,NP(\mu P)}\right],\ \left[0,0,0,0,\frac{\beta 1(n2)\,\Lambda B}{(\mu B+\delta 5)\,NB(\mu B)}\right]\right]$$

Next, we determine the eigenvalues of our non-inhibitor model. Now we compute $|FV^{-1}-A_iI|=0$, where A_i is given as the Eigen values.

The eigenvalues for the non-inhibitor model is given as:

$$\begin{bmatrix} 0 \\ 0 \\ \dfrac{\beta 2(n1)\,\Lambda P}{(\gamma 3+\tau 2+\mu P+\delta 3)\,NP(\mu P)} \\ \dfrac{\beta 1(n0)\,\Lambda H}{(\gamma 1+\tau 1+\mu H+\delta 1)\,NH(\mu H)} \\ \dfrac{\beta 1(n2)\,\Lambda B}{(\mu B+\delta 5)\,NB(\mu B)} \end{bmatrix}$$

Therefore, $R_0 = \frac{\beta_1 n_0\,\Lambda_H}{[\gamma_1+\tau_1+\mu_H+\delta_1]\,N_H\mu_H}$. This is the basic Reproductive number for our non-inhibitor model. Result for R_0, was first recognized by [10], many other works have been done on R_0, such as [6].

In practice, R_0 does not depend on the assumptions about immunity subsequent to the infectious period. The R_0 concept is based on an assumed situation where one infective is introduced into a large susceptible population [4] In reality, such situations hardly ever occur, such that one has to base estimates of Ro on data available in endemic situations where the infection is present for a long time. However, this requires detailed knowledge about the density-dependent regulation of the

transmission process which determines the equilibrium endemic level. It also assumes knowledge about the heterogeneities in transmission patterns, as it is obvious that estimates of Ro based on equilibrium data are highly dependent on the availability of this required information or on the degree to which the assumed mechanisms reflect reality.

6. Sensitivity indices of NiV infection model parameters

In this section, we try to determine 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.

In 2017, [3], gave us a clear succinct definition of sensitivity analysis where he stated that the normalized forward- sensitivity index of a variable, v, depends on a parameter of interest, p, which is defined as:

$$\mathrm{r}_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v}.$$

In particular, the sensitivity indices, of the basic reproduction number, R_0, with respect to the model parameters are examined.

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 parameter in the model, may lead to an increase (or decrease) in R_0 of the model (1-10) and asymptotically result into persistency (or elimination) of the disease in the community. On the contrary, the negative sign of S.I of R_0 to the model parameter indicates that an increase (or decrease) in the value of each of the parameter in each case leads to a corresponding decrease (or increase) in R_0 of the model (1-10). Hence, with sensitivity analysis, one can get insight on the appropriate intervention strategies to prevent and control the spread of the disease described in model (1-10).

Recall that

$$R_0 = \frac{\beta_1 n_0 \Lambda_H}{[\gamma_1+\tau_1+\mu_H+\delta_1] N_H \mu_H},$$

and that $n_0 > 0$.

Hence:

$$\boldsymbol{r}_{\mu_H}^{R_0} = \frac{\partial R_0}{\partial \mu_H} \times \frac{\mu_H}{R_0}$$
$$= -\left(\frac{\beta_1 n_0 \Lambda_H}{[\gamma_1+\tau_1+\mu_H+\delta_1] N_H \mu_H^2} + \frac{\beta_1 n_0 \Lambda_H}{[\gamma_1+\tau_1+\mu_H+\delta_1]^2 N_H \mu_H}\right) \times \frac{[\gamma_1+\tau_1+\mu_H+\delta_1] N_H \mu_H^2}{\beta_1 n_0 \Lambda_H}$$

$$= -\left(\frac{\beta_1 n_0 \Lambda_H[\gamma_1+\tau_1+\mu_H+\delta_1] + \beta_1 n_0 \Lambda_H \mu_H}{[\gamma_1+\tau_1+\mu_H+\delta_1]^2 N_H \mu_H{}^2}\right) \times \left(\frac{[\gamma_1+\tau_1+\mu_H+\delta_1]\, N_H \mu_H{}^2}{\beta_1 n_0 \Lambda_H}\right)$$

$$= -\left(\frac{[\gamma_1+\tau_1+\mu_H+\delta_1] + \mu_H}{[\gamma_1+\tau_1+\mu_H+\delta_1]^2 N_H \mu_H{}^2}\right) \times ([\gamma_1 + \tau_1 + \mu_H + \delta_1]\, N_H \mu_H{}^2)$$

$$= -\left(\frac{[\gamma_1+\tau_1+\mu_H+\delta_1] + \mu_H}{[\gamma_1+\tau_1+\mu_H+\delta_1]\mu_H}\right) \quad (negative)$$

Summarily, the table below shows the signs of S.I. of R_0:

$\beta_1 - positive$
$\Lambda_H - positive$
$n_0 - positive$
$\delta_1 - negaive$
$N_H - negative$
$\tau_1 - negative$
$\gamma_1 - negative$
$\mu_H - negative$

Thus, if we increase (decrease) any of the parameters β_1, Λ_H, n_0, then R_0 will increase (decrease). Also, if we increase (decrease) any of N_H, μ_H, γ_1,τ_1, δ_1, then R_0 will decrease (increase). Sensitive index of other parameters that did not appear in R_0 are zero, that is, the increase or decrease of such parameter has no influence on the reproductive number.

6. Conclusion

The periodic outbreaks of deadly Nipah virus infections in Malaysia have become the most public health concerns because of the highly mortality rates. Once an outbreak occurs in any region of the country, the virus can be transmitted to the whole family and/or even in the community. The study derives an estimate of R_0 based on Nipah virus epidemic and endemic situations to interpret R_0, from our assumptions and theories of existing researches. During the last twenty years R_0 has emerged as a basic concept in infectious disease epidemiology, but it has also become apparent how difficult it is to apply in actual field situations [4]. It was discovered that some parameters were found to be positively contributing to the spread of the disease, parameters like Λ_H, which is the recruitment parameter for the model, β_1, this parameter is the effective contact rate, while some other parameters reduce the spread of the disease, parameters like τ_1, which is the proportion of infected that goes for treatment, while μ_H, natural death. It is our hope that this survey will motivate further research in this direction.

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