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OPTIMAL NIPAH VIRUS (NIV) SPREAD CONTROL DYNAMICS

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Abstract. In this work, we look at the vaccine and condom Nipah virus model using an optimum control analysis. We implemented measures to limit infection dissemination and control. We examine four distinct controls: personal protection, rapid testing, burying infected pigs, and therapy control. We construct an ideal control model and demonstrate the mathematical results using the suggested controls. The results of optimum control suggest that measures can be useful in decreasing infected individuals and increasing the health of society.

Keywords: Nipah virus; optimal control; numerical results; protection.

2020 AMS Subject Classification: 00A71,92B05, 68U20.

1. INTRODUCTION

The Nipah Virus (NiV) is a transmissible from animal virus which may trigger deadly encephalitis and serious lung conditions in humans. It is a contagious virus that primarily affects Pteropodidae fruit bats, but it can also impact swine and humans. During an epidemic among

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Malaysian swine producers in 1999, the Nipah virus was detected. There have been no new cases recorded in Malaysia since 1999. It was discovered in Bangladesh in 2001, and outbreaks have occurred practically every year thereafter. East India has also been ravaged by the illness on a regular basis. The virus has been discovered in the recognized native reservoir (*Pteropus* bat species) as well as a variety of additional bat species in Cambodia, Ghana, Indonesia, Madagascar, the Philippines, and Thailand [14].

Humans are affected with the virus through interaction with sick animals especially pigs or their bodily secretions, or through ingestion of contaminated food items. Close contact with an infected person may result in from person to person. spread, particularly in hospital settings. According to Tan et al [15], patients hospitalized to the University Hospital in Kuala Lumpur and their family members served as the study's subjects. 110 people from 14 houses were analyzed. Asymptomatic Nipah infection was seen in 30 out of 110 (27%) of the household members.

Although no drugs or vaccines are presently available for treatment, a phase 1 clinical research of a Nipah virus vaccine candidate (HeV-sG-V) began in February 2020 [13]. The vaccine will be administered to normal individuals aged 18 to 49 to test its effectiveness and safety in triggering an immune response [13].

During NiV epidemics in South Asian nations, interactions between animals, people, and the environment were very important [2]. Numerous variables influence human-animal interactions, including prolonged droughts, reduced animal habitats as a result of deforestation, manmade fires that destroy forests in Indonesia, and pig rearing mixed with farmland. [1].

For the study of the dynamics of disease spread, researchers have developed mathematical models like the one in [7],[8],[6], which has changed our knowledge of these contagious illnesses and aided in the development of sufficient and effective countermeasures. Biswas investigated disease mechanisms by employing the SIR fundamental mathematical model. He investigated this model further and investigated potential control and avoidance techniques with an optimum solution [10],[9] and [3]. Sultana [4] used optimum control theory to create a quantitative study of Nipah viral infections. Control tactics in their work included raising consciousness and providing treatment. They proved the presence of optimum controls, and the ideal controls

are defined using Pontryagin's utmost concept. According to the numerical modeling, the optimum control method is far more effective at reducing infectious people and the associated costs of both measures. Furthermore, Mondal et al[16] propose and evaluate a mathematical model for Nipah viral management with vital mechanics. They [11], [16] include the quarantine of contagious people based on the existence of isolation facilities alongside monitoring coverage. According to the numerical modeling, better sanitation and confinement of infectious people are sufficient to contain Nipah viral spread. As Nipah virus control plans, Omede et al [12] weighed avoidance measures to keep people in the vulnerable area from coming into contact with the virus, Nipah virus treatment for people in the area that is infectious compartment, and Nipah virus treatment for people in the curative area.

The fundamental purpose of this research is to investigate the four control techniques; personal prevention(pp), rapid testing, burying infected piglets, and therapy control in the vaccines and condoms Nipah virus model. This paper is structured as follows: Section 2, Methodology of NiV model. In Section 3, Existence of the optimal control of NiV. Section 4, optimal control numerical simulation. Section 5, discussion and conclusion.

2. METHODOLOGY OF NiV MODEL

From the humans and pigs population, we have $S_p(t)$: the number of pigs who are not yet infected with the NiV at time t , but may get it if they come into contact with other infected pigs or eat contaminated fruits, $E_p(t)$: the number of pigs that have come into contact with the infectious agent or pathogen that causes the Nipah virus, $I_p(t)$: the number of pigs that are capable of transmitting the virus to others including human, $S(t)$: susceptible with respect to human beings, $S_v(t)$: the susceptible persons who are vaccinated, $S_u(t)$: the susceptible persons who are not yet vaccinated, $S_{vc}(t)$: the susceptible persons who are vaccinated and use condoms, $S_{vn}(t)$: the susceptible persons who are vaccinated and do not use condoms, $S_{uc}(t)$: the susceptible persons who are not yet vaccinated and use condoms, $S_{un}(t)$: the susceptible persons who are not yet vaccinated and do not care for condoms, $E(t)$: people that are in touch with the infectious agent(human and pigs) or pathogen that causes the Nipah virus, $I(t)$: people that are capable of transmitting the virus to others, $C(t)$: they are people who have been infected with the virus but do not develop any symptoms of the disease, and still carry and

transmit the virus to others, $I_i(t)$: they are isolated individuals undergoing treatment who are capable of transmitting the virus to others, $I_t(t)$: they are not isolated but undergoing treatment as individuals that are capable of transmitting the virus to others, $R(t)$: they are individuals who have recovered from the Nipah virus and are capable of contacting the virus again, $D(t)$: the bodies of those who died due to the virus.

We assume the following: Natural sickness recovery can take place because of powerful antibodies[12]. Casual touching of the dead bodies will expose the individuals to the virus [17]. There is an interaction between the farmer and the infectious pigs[5]. Since they are continuously watched, medical personnel safeguard themselves against the virus, and infection can happen in a therapy class, isolated individuals do not aid in the spread of NiV[12]. The general public has easy access to and can afford condoms, an infected isolation facility, and vaccinations[5]. After some time, people who have recovered become susceptible to infection once more.

TABLE 2.1. Describe the Variables

parameters	Parameter Description
Λ_p	The proportion of new pigs introduced
σ	Exposure rate of pigs
ρ	The rate at which infected pigs become exposed
Λ	Human resource recruitment level
χ_1	Rate of vulnerable non-vaccinated people
χ_2	Vaccination coverage among vulnerable populations
η_1	The fraction of unvaccinated vulnerable people who use condoms
η_2	The fraction of unvaccinated vulnerable people who do not use condoms.
τ_1	The fraction of vaccinated people who use condoms
τ_2	The fraction of vaccinated vulnerable people who do not use condoms
Γ_3	Infection force on S_{nc}
Γ_4	Infection force on S_{un}
Γ_1	Infection force on S_{vc}
Γ_2	Infection force on S_{vn}

κ	The rate at which an exposed population becomes infected
θ	The proportion of the exposed population who becomes a NiV carrier
ψ_1	Rate of isolation of infected people having treatment
ψ_2	Treatment rate of infected persons
γ_1	Recovery rate from the disease therapy class
γ_2	Recovery rate from the infectious isolated undergoing treatment class
γ_3	The NiV carrier recovery rate
γ_4	The infectious recovery rate
ε	Rate of susceptibility among recovered persons
δ_1	Illness-related death rate in NiV-Carriers
δ_2	Illness-related death rate in infectious population
δ_3	Illness-related death rate in infectious isolated people undergoing treatment
δ_4	Illness-related death rate in infectious people undergoing treatment
δ_d	Illness-related death rate in infectious pigs
μ_d	The rate at which deceased bodies are disposed of (burial/cremation)
μ_p	Pig mortality rate
μ	Natural death rate

We assume the following: Natural sickness recovery can take place because of powerful antibodies [12]. Casual touching of the dead bodies will expose the individuals to the virus [17]. There is an interaction between the farmer and the infectious pigs[5]. Since they are continuously watched, medical personnel safeguard themselves against the virus, and infection can happen in a therapy class, isolated individuals do not aid in the spread of NiV [12]. The general public has easy access to and can afford condoms, an infected isolation facility, and vaccinations[5]. After some time, people who have recovered become susceptible to infection once more.

The above formulations and assumptions result in the deterministic system of nonlinear ordinary differential equations that characterize the dynamics of Nipah virus infection spread in two groups.

$$\begin{aligned}
(2.1) \quad & \frac{dS}{dt} = \Lambda - (\chi_1 + \chi_2 + \mu)S + \varepsilon R \\
& \frac{dS_v}{dt} = \chi_2 S - (\tau_1 + \tau_2 + \mu)S_v \\
& \frac{dS_u}{dt} = \chi_1 S - (\eta_1 + \eta_2 + \mu)S_u \\
& \frac{dS_{vc}}{dt} = \tau_1 S_v - (\Gamma_1 + \mu)S_{vc} \\
& \frac{dS_{vn}}{dt} = \tau_2 S_v - (\Gamma_2 + \mu)S_{vn} \\
& \frac{dS_{uc}}{dt} = \eta_1 S_u - (\Gamma_3 + \mu)S_{uc} \\
& \frac{dS_{un}}{dt} = \eta_2 S_u - (\Gamma_4 + \mu)S_{un} \\
& \frac{dE}{dt} = \Gamma_2 S_{vn} + \Gamma_4 S_{un} + \Gamma_3 S_{uc} + \Gamma_1 S_{vc} - (\mu + \theta + \kappa)E \\
& \frac{dC}{dt} = \theta E - (\gamma_3 + \mu + \delta_1)C \\
& \frac{dI}{dt} = \kappa E - (\psi_1 + \psi_2 + \mu + \delta_2 + \gamma_4)I \\
& \frac{dI_{it}}{dt} = \psi_1 I - (\gamma_2 + \mu + \delta_3)I_{it} \\
& \frac{dI_t}{dt} = \psi_2 I - (\gamma_1 + \mu + \delta_4)I_t \\
& \frac{dR}{dt} = \gamma_2 I_{it} + \gamma_4 I + \gamma_1 I_t + \gamma_3 C - \mu R - \varepsilon R \\
& \frac{dD}{dt} = \delta_4 I_t + \delta_3 I_{it} + \delta_1 C + \delta_2 I - \mu_d D \\
& \frac{dS_p}{dt} = \Lambda_p - (\sigma + \mu_p)S_p \\
& \frac{dE_p}{dt} = \sigma S_p - (\rho + \mu_p)E_p \\
& \frac{dI_p}{dt} = \rho E_p - (\mu_p + \delta_p)I_p
\end{aligned}$$

Such that

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

2.1. Optimal Control Procedure. The Nipah viral model is subjected to optimum management theory given in (2.1). We examine four distinct controls to reduce Nipah viral infection and further spread in the population. The following definition applies to these controls: The safety control u_1 calls for reducing interaction with contagious persons, including dead victims of the Nipah virus, cleaning hands frequently, using hand sanitizer, and wearing masks. Further, Avoid meeting and limit their movement in areas with a high number of cases. Rapid testing of people in the vulnerable stage to detect Nipah virus carriers (C) and symptomatic individuals constitutes the second control u_2 . To further reduce the infection, the people should be isolated or confined to their house after recognition through testing. The control u_3 is burying infected piglet. The fourth control u_4 is the therapy control. The use of Ribavirin and any other substances that can boost immune system especial different species of vegetables. Also bitter kola could also be very relevant. Following is a system of optimal control issue that may result from the talk above:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\chi_1 + \chi_2 + \mu)S + \varepsilon R \\
\frac{dS_v}{dt} &= \chi_2 S - (\tau_1 + \tau_2 + \mu)S_v \\
\frac{dS_u}{dt} &= \chi_1 S - (\eta_1 + \eta_2 + \mu)S_u \\
\frac{dS_{vc}}{dt} &= \tau_1 S_v - (\Gamma_1 + \mu)S_{vc} \\
\frac{dS_{vn}}{dt} &= \tau_2 S_v - (\Gamma_2 + \mu)S_{vn} \\
\frac{dS_{uc}}{dt} &= \eta_1 S_u - (1 - u_1)\Gamma_3 S_{uc} - \mu S_{uc} \\
\frac{dS_{un}}{dt} &= \eta_2 S_u - (1 - u_1)\Gamma_4 S_{un} - \mu S_{un} \\
(2.2) \quad \frac{dE}{dt} &= \Gamma_2 S_{vn} + (1 - u_1)\Gamma_4 S_{un} + (1 - u_1)\Gamma_3 S_{uc} + \Gamma_1 S_{vc} - (u_2 + \mu + \theta + \kappa)E \\
\frac{dC}{dt} &= (u_2 + \theta)E - (u_4 + \gamma_3 + \mu + \delta_1)C \\
\frac{dI}{dt} &= \kappa E - (\psi_1 + \psi_2 + \mu + \delta_2 + \gamma_4 + u_4)I \\
\frac{dI_{it}}{dt} &= \psi_1 I - (u_4 + \gamma_2 + \mu + \delta_3)I_{it}
\end{aligned}$$

$$\begin{aligned}
\frac{dI_t}{dt} &= \psi_2 I - (u_4 + \gamma_1 + \mu + \delta_4) I_t \\
\frac{dR}{dt} &= (I_t + I_{it} + I) u_4 + \gamma_2 I_{it} + \gamma_4 I + \gamma_1 I_t + \gamma_3 C - \mu R - \varepsilon R \\
\frac{dD}{dt} &= \delta_4 I_t + \delta_3 I_{it} + \delta_1 C + \delta_2 I - \mu_d D \\
\frac{dS_p}{dt} &= \Lambda_p - (1 - u_3) \sigma S_p - \mu_p S_p \\
\frac{dE_p}{dt} &= (1 - u_3) \sigma S_p - (\rho + \mu_p) E_p \\
\frac{dI_p}{dt} &= \rho E_p - (\mu_p + \delta_p) I_p
\end{aligned}$$

with the initial conditions $S(0) \geq 0, S_u(0) \geq 0, S_v(0) \geq 0, S_{uc}(0) \geq 0, S_{un}(0) \geq 0, S_{vc}(0) \geq 0, S_{vn}(0) \geq 0, E(0) \geq 0, C(0) \geq 0, I(0) \geq 0, I_{it}(0) \geq 0, I_t(0) \geq 0, D(0) \geq 0, R(0) \geq 0, S_p(0) \geq 0, E_p(0) \geq 0, I_p(0) \geq 0$

Our goals are to decrease the overall number of contagious people in the community and the costs associated with protective gear, vaccine production and dissemination, quick testing, and treatment at predetermined intervals. We reduce the target function provided by,

(2.3)

$$J(u_i, \Omega_1) = \int_0^T \left(A_1 E + A_2 C + A_3 I + A_4 I_{it} + A_5 I_t + A_6 D + \frac{1}{2} [k_1 u_1^2 + k_2 u_2^2 + k_3 u_3^2 + k_4 u_4^2] \right) dt$$

where $i = 1, 2, 3, 4$, and T is final time, Ω_1 stands for the collection of all contagious compartments, $A_i, i = 1, \dots, 6$ represents the sections' non-negative weight values E, C, I, I_{it}, I_t, D respectively and k_1, k_2, k_3, k_4 are the weight constant for the control variable u_1, u_2, u_3, u_4 respectively. The weights k_1, k_2, k_3, k_4 which are constant parameters for u_1, u_2, u_3, u_4 will standardized using the optimal control condition.

The variables defined above have bounds on Lebesgue integrable functions. We strive for optimum settings u_i^* for $i=1, \dots, 4$, such that $J(u_i^*) = \min(J(u_i, \Omega_1))$ where $u_i \in U$ the control set defined as

$$(2.4) \quad U = \{(u_i) : [0, T] \longrightarrow [0, 1], (u_i), \text{ is Lebesgue measurable}\}$$

We explain the terms in the integrand in (2.3) above as follows: The term $A_1E + A_2C + A_3I + A_4I_{it} + A_5I_t + A_6D$ indicates the expense involved with tracking infectious people. The term $k_1u_1^2 + k_3u_3^2$ reflects the expense of all kinds of protection against infectious people and pigs. The term $k_2u_2^2$ reflects the expense of all types of rapid testing of exposed people. The term $k_4u_4^2$ indicates the total expense of all kinds of therapy for all infected people in the infectious groups.

3. EXISTENCE OF THE OPTIMAL CONTROL FOR NIPAH VIRUS MODEL

3.1. Existence of the State. From the equation (2.2), we have

$$\begin{aligned}
\frac{dN}{dt} &= \Lambda - \mu S - \mu S_u - \mu S_v - \mu S_{uc} - \mu S_{un} - \mu S_{vc} - \mu S_{vn} - \mu E - \mu C - \delta_1 C - \mu I \\
&\quad - \delta_2 I - \mu I_{it} - \delta_3 I_{it} - \mu I_t - \delta_4 I_t - \mu R \\
&= \Lambda - \mu S - \mu S_u - \mu S_v - \mu S_{uc} - \mu S_{un} - \mu S_{vc} - \mu S_{vn} - \mu E - \mu C - \mu I \\
&\quad - \mu I_{it} - \mu I_t - \mu R - \delta_4 I_t - \delta_1 C - \delta_3 I_{it} - \delta_2 I \\
&= \Lambda - \mu N - \delta_4 I_t - \delta_1 C - \delta_3 I_{it} - \delta_2 I \\
(3.1) \quad &\leq \Lambda - \mu N(t),
\end{aligned}$$

$$(3.2) \quad \frac{dD}{dt} = \delta_4 I_t + \delta_3 I_{it} + \delta_1 C + \delta_2 I - \mu_d D$$

Solving (3.1), we obtain

$$(3.3) \quad N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}$$

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

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

$$\begin{aligned}
&S(t) + S_u(t) + S_v(t) + S_{uc}(t) + S_{un}(t) + S_{vc}(t) + S_{vn}(t) + E(t) + C(t) + I(t) \\
&+ I_{it}(t) + I_t(t) + R(t) \leq \frac{\Lambda}{\mu} \implies S(t) \leq \frac{\Lambda}{\mu}, \dots, I(t) \leq \frac{\Lambda}{\mu}, I_{it}(t) \leq \frac{\Lambda}{\mu}, I_t(t) \leq \frac{\Lambda}{\mu}, \dots, R(t) \leq \frac{\Lambda}{\mu} \text{ as} \\
&t \geq 0.
\end{aligned}$$

Therefore,

$$\begin{aligned}
 \frac{dD}{dt} &= \delta_4 I_t + \delta_3 I_{it} + \delta_1 C + \delta_2 I - \mu_d D \\
 (3.4) \qquad &\leq (\delta_4 + \delta_3 + \delta_1 + \delta_2) \frac{\Lambda}{\mu} - \mu_d D
 \end{aligned}$$

implies $D(t) \leq (\delta_4 + \delta_3 + \delta_1 + \delta_2) \frac{\Lambda}{\mu_d \mu}$ and

$$\begin{aligned}
 \frac{dN_p}{dt} &= \Lambda_p - \mu_p S_p - \mu_p E_p - \mu_p I_p - \delta_p I_p \\
 &= \Lambda_p - \mu_p N_p(t) - \delta_p I_p \\
 (3.5) \qquad &\leq \Lambda_p - \mu_p N_p(t)
 \end{aligned}$$

implies $N_p(t) \leq \frac{\Lambda_p}{\mu_p}$.

Finally the system (2.2) is bounded and the solutions exist.

3.2. Existence of the Objective Functional. Pontryagin's optimal concept reduces the state system (2.2) to an issue of reducing the Langrangian L and Hamiltonian H with regard to (u_1, u_2, u_3, u_4) at each position. The integrand of the goal functional makes up the Langrangian of the management problem, which is the Hamitonian enhanced with penalty terms for control restrictions and is given by

$$L = A_1 E + A_2 C + A_3 I + A_4 I_{it} + A_5 I_t + A_6 D + \frac{1}{2} [k_1 u_1^2 + k_2 u_2^2 + k_3 u_3^2 + k_4 u_4^2]$$

We look for the lagrangian's minimal number. This can be done by specifying the Hamiltonian H for the control problem, comprised of the integrand of the function with the goal and the result of the inner product of the right hand sides of the state equations and the co-state variables or adjoint variables λ_i , $i = 1, ..17$ with respect to state variable as

$$\begin{aligned}
 H &= A_1 E + A_2 C + A_3 I + A_4 I_{it} + A_5 I_t + A_6 D + \frac{1}{2} [k_1 u_1^2 + k_2 u_2^2 + k_3 u_3^2 + k_4 u_4^2] \\
 &+ \lambda_1 \{ \Lambda - (\chi_1 + \chi_2 + \mu) S + \epsilon R \} + \lambda_2 \{ \chi_2 S - (\tau_1 + \tau_2 + \mu) S_v \} \\
 &+ \lambda_3 \{ \chi_1 S - (\eta_1 + \eta_2 + \mu) S_u \} + \lambda_4 \{ \tau_1 S_v - (\Gamma_1 + \mu) S_{vc} \} \\
 &+ \lambda_5 \{ \tau_2 S_v - (\Gamma_2 + \mu) S_{vn} \} + \lambda_6 \{ \eta_1 S_u - (1 - u_1) \Gamma_3 S_{uc} - \mu S_{uc} \} \\
 (3.6) \qquad &+ \lambda_7 \{ \eta_2 S_u - (1 - u_1) \Gamma_4 S_{un} - \mu S_{un} \}
 \end{aligned}$$

$$\begin{aligned}
& + \lambda_8 \{ \Gamma_2 S_{vn} + (1 - u_1) \Gamma_4 S_{un} + (1 - u_1) \Gamma_3 S_{uc} + \Gamma_1 S_{vc} - (u_2 + \mu + \theta + \kappa) E \} \\
& + \lambda_9 \{ (u_2 + \theta) E - (u_4 + \gamma_3 + \mu + \delta_1) C \} + \lambda_{10} \{ \kappa E - (\psi_1 + \psi_2 + \mu + \delta_2 + \gamma_4 + u_4) I \} \\
& + \lambda_{11} \{ \psi_1 I - (u_4 + \gamma_2 + \mu + \delta_3) I_{it} \} + \lambda_{12} \{ \psi_2 I - (u_4 + \gamma_1 + \mu + \delta_4) I_t \} \\
& + \lambda_{13} \{ (I + I_t + I_{it}) u_4 + \gamma_2 I_{it} + \gamma_4 I + \gamma_1 I_t + \gamma_3 C + u_2 C - \mu R - \epsilon R \} \\
& + \lambda_{14} \{ \delta_4 I_t + \delta_3 I_{it} + \delta_1 C + \delta_2 I - \mu_d D \} \\
& + \lambda_{15} \{ \Lambda_p - (1 - u_3) \sigma S_p + \mu_p S_p \} + \lambda_{16} \{ (1 - u_3) \sigma S_p - (\rho + \mu_p) E_p \} \\
& + \lambda_{17} \{ \rho E_p - (\mu_p + \delta_p) I_p \}
\end{aligned}$$

The system's status and control variables (2.2) have positive values. Convex and closed characterize the control set U . The presence of optimal control is demonstrated by Corollary 4.1 in [18] because the integrand of the objective cost function U given by (2.2) is a convex function of (u_1, u_2, u_3, u_4) on the control set U . As a result, there are positive integers ξ_1, ξ_2 and ς such that

$$(3.7) \quad J(u_1, u_2, u_3, u_4) \geq \xi_1 (|u_1|^2 + |u_2|^2 + |u_3|^2 + |u_4|^2)^{\frac{\varsigma}{2}} - \xi_2$$

where $\xi_1, \xi_2 > 0$, and $\varsigma > 1$

Since the answers are bounded, the state system's Lipschitz condition is satisfied. Considering that the state variables are constrained, this proves the presence of an optimum control.

3.3. An Optimal Control's Characteristics. The essential criteria for Pontryagin's Optimal Concept serves as the foundation for the important standards for optimal management [19]. The systems (2.2) and (2.1) are reduced by this idea. to the problem of reducing a Hamiltonian H pointwise with respect to the constraints $u_i, i = 1, 2, 3, 4$. The set of adjoint equations can be derived using the correct partial derivatives of H with regard to the state variables.

Theorem 3.1. *The given optimal controls u_i^* and solutions $S(t)^*, S_v(t)^*, S_u(t)^*, S_{vc}(t)^*, S_{vn}(t)^*, S_{uc}(t)^*, S_{un}(t)^*, E(t)^*, C(t)^*, I(t)^*, I_{it}(t)^*, I_t(t)^*, R(t)^*, D(t)^*, S_p(t)^*, E_p(t)^*, I_p^*$ of the control system (4.409)-(4.425) that minimizes $J(u_i^*)$ over U . Then there exists adjoint variables λ_i satisfying*

$$(3.8) \quad \frac{\partial \lambda_i}{dt} = - \frac{\partial H}{\partial i}$$

with the transversality conditions,

$$(3.9) \quad \lambda_i(T) = 0$$

$$i = S(t), S_v(t), S_u(t), S_{vc}(t), S_{vn}(t), S_{uc}(t), S_{un}(t), E(t), C(t), I(t), I_{it}(t), I_t(t), R(t), \\ D(t), S_p(t), E_p(t), I_p$$

The optimality condition is given by

$$(3.10) \quad \frac{\partial H}{\partial u_i} = 0, i = 1, 2, 3, 4$$

Furthermore, we have the controls u_i^* ,

$$(3.11) \quad u_1^* = \min \left\{ 1, \max \left[0, \frac{\lambda_8 (Suc\Gamma_3 + Sun\Gamma_4) - Suc\lambda_6\Gamma_3 - Sun\lambda_7\Gamma_4}{k_1} \right] \right\}$$

$$(3.12) \quad u_2^* = \min \left\{ 1, \max \left[0, \frac{E(\lambda_8 - \lambda_9) - \lambda_{13}C}{k_2} \right] \right\}$$

$$(3.13) \quad u_3^* = \min \left\{ 1, \max \left[0, \frac{Sp\sigma(\lambda_{16} - \lambda_{15})}{k_3} \right] \right\}$$

$$(3.14) \quad u_4^* = \min \left\{ 1, \max \left[0, \frac{I\lambda_{10} + I_{it}\lambda_{11} + I_t\lambda_{12} - \lambda_{13}(I + I_t + I_{it})}{k_4} \right] \right\}$$

Proof. The findings from the [18] cases provide proof that an optimal control issue exists. Differentiating H with regard to each state variable results in the system ruling the adjoint variables.

Consequently, the adjoint system is expressed as,

$$\dot{\lambda}_1 = -\frac{\partial H}{\partial S} = \lambda_1(\chi_1 + \chi_2 + \mu) - \chi_1\lambda_3 - \chi_2\lambda_2$$

$$\dot{\lambda}_2 = -\frac{\partial H}{\partial S_v} = \lambda_2(\mu + \tau_1 + \tau_2) - \lambda_4\tau_1 - \lambda_5\tau_2$$

$$\dot{\lambda}_3 = -\frac{\partial H}{\partial S_u} = \lambda_3(\eta_1 + \eta_2 + \mu) - \eta_1\lambda_6 - \eta_2\lambda_7$$

$$\dot{\lambda}_4 = -\frac{\partial H}{\partial S_{vc}} = \lambda_4(\Gamma_1 + \mu) - \lambda_8\Gamma_1$$

$$\dot{\lambda}_5 = -\frac{\partial H}{\partial S_{vn}} = \lambda_5(\Gamma_2 + \mu) - \lambda_8\Gamma_2$$

$$\dot{\lambda}_6 = -\frac{\partial H}{\partial S_{uc}} = \lambda_6(1 - u_1)(\Gamma_3 + \mu) - \lambda_8(1 - u_1)\Gamma_3$$

$$\dot{\lambda}_7 = -\frac{\partial H}{\partial S_{un}} = \lambda_7(1-u_1)(\Gamma_4 + \mu) - \lambda_8(1-u_1)\Gamma_4$$

$$\dot{\lambda}_8 = -\frac{\partial H}{\partial E} = -A_1 - \lambda_{10}\kappa + \lambda_8(\kappa + \mu + \theta + u_2) - \lambda_9(\theta + u_2)$$

$$\begin{aligned} \dot{\lambda}_9 = -\frac{\partial H}{\partial C} = & -A_2 - \delta_1\lambda_{14} - (\gamma_3 + u_4)\lambda_{13} \\ & - \lambda_8 \left(\frac{Suc\beta_3q_2(1-u_1)}{N} + \frac{Sun\beta_4z_2(1-u_1)}{N} + \frac{Svca_2\beta_1}{N} + \frac{Svnb_2\beta_2}{N} \right) + \lambda_9(\delta_1 + \gamma_3 + u_4 + \mu) \\ & + \frac{Suc\beta_3\lambda_6q_2(1-u_1)}{N} + \frac{Sun\beta_4\lambda_7z_2(1-u_1)}{N} + \frac{Svca_2\beta_1\lambda_4}{N} + \frac{Svnb_2\beta_2\lambda_5}{N} \end{aligned}$$

$$\begin{aligned} \dot{\lambda}_{10} = -\frac{\partial H}{\partial I} = & -A_3 - \delta_2\lambda_{14} - (\gamma_4 + u_4)\lambda_{13} + \lambda_{10}(\delta_2 + \gamma_4 + u_4 + \mu + \psi_1 + \psi_2) - \lambda_{11}\psi_1 \\ & - \lambda_{12}\psi_2 - \lambda_8 \left(\frac{Suc\beta_3q_3(1-u_1)}{N} + \frac{Sun\beta_4z_3(1-u_1)}{N} + \frac{Svca_3\beta_1}{N} + \frac{Svnb_3\beta_2}{N} \right) \\ & + \frac{Suc\beta_3\lambda_6q_3(1-u_1)}{N} + \frac{Sun\beta_4\lambda_7z_3(1-u_1)}{N} + \frac{Svca_3\beta_1\lambda_4}{N} + \frac{Svnb_3\beta_2\lambda_5}{N} \end{aligned}$$

$$\dot{\lambda}_{11} = -\frac{\partial H}{\partial I_{it}} = -A_4 - \delta_3\lambda_{14} - (\gamma_2 + u_4)\lambda_{13} + \lambda_{11}(\delta_3 + \gamma_2 + u_4 + \mu)$$

$$\begin{aligned} \dot{\lambda}_{12} = -\frac{\partial H}{\partial I_t} = & -A_5 - \delta_4\lambda_{14} - (\gamma_1 + u_4)\lambda_{13} + \lambda_{12}(\delta_4 + \gamma_1 + u_4 + \mu) \\ & - \lambda_8 \left(\frac{Suc\beta_3q_4(1-u_1)}{N} + \frac{Sun\beta_4z_4(1-u_1)}{N} + \frac{Svca_4\beta_1}{N} + \frac{Svnb_4\beta_2}{N} \right) \\ & + \frac{Suc\beta_3\lambda_6q_4(1-u_1)}{N} + \frac{Sun\beta_4\lambda_7z_4(1-u_1)}{N} + \frac{Svca_4\beta_1\lambda_4}{N} + \frac{Svnb_4\beta_2\lambda_5}{N} \end{aligned}$$

$$\dot{\lambda}_{13} = -\frac{\partial H}{\partial R} = \varepsilon\lambda_1 + \lambda_{13}(\varepsilon + \mu)$$

$$\begin{aligned} \dot{\lambda}_{14} = -\frac{\partial H}{\partial D} = & -A_6 + \lambda_{14}\mu_3 - \lambda_8 \left(\frac{Suc\beta_3q_5(1-u_1)}{N} + \frac{Sun\beta_4z_5(1-u_1)}{N} + \frac{Svca_5\beta_1}{N} + \frac{Svnb_5\beta_2}{N} \right) \\ & + \frac{Suc\beta_3\lambda_6q_5(1-u_1)}{N} + \frac{Sun\beta_4\lambda_7z_5(1-u_1)}{N} + \frac{Svca_5\beta_1\lambda_4}{N} + \frac{Svnb_5\beta_2\lambda_5}{N} \end{aligned}$$

$$\dot{\lambda}_{15} = -\frac{\partial H}{\partial S_p} = \lambda_{15}(\mu_2 + \sigma(1-u_3)) - \lambda_{16}\sigma(1-u_3)$$

$$\dot{\lambda}_{16} = -\frac{\partial H}{\partial E_p} = \lambda_{16}(\mu_2 + \rho) - \lambda_{17}\rho$$

$$\begin{aligned} \dot{\lambda}_{17} = -\frac{\partial H}{\partial I_p} = & \lambda_{17}(\delta_5 + \mu_2) - \lambda_8 \left(\frac{Suc\beta_3q_1(1-u_1)}{Np} + \frac{Sun\beta_4z_1(1-u_1)}{Np} + \frac{Svca_1\beta_1}{Np} + \frac{Svnb_1\beta_2}{Np} \right) \\ & + \frac{Suc\beta_3\lambda_6q_1(1-u_1)}{Np} + \frac{Sun\beta_4\lambda_7z_1(1-u_1)}{Np} + \frac{Svca_1\beta_1\lambda_4}{Np} + \frac{Svnb_1\beta_2\lambda_5}{Np} \end{aligned}$$

With the transversality conditions at time T : $\lambda_1(T) = \lambda_2(T) = \dots = \lambda_{17}(T) = 0$

For $t \in [0, T]$, the optimal controls u_1, u_2, u_3 , and u_4 can be solved from the optimality condition

$$\begin{aligned}\frac{\partial H}{\partial u_i} &= 0, i = 1, 2, 3, 4 \\ \frac{\partial H}{\partial u_1} &= Suc\lambda_6\Gamma_3 + Sun\lambda_7\Gamma_4 + 1.0k_1u_1 - \lambda_8(Suc\Gamma_3 + Sun\beta_3) = 0 \\ \frac{\partial H}{\partial u_2} &= C\lambda_{13} - E\lambda_8 + E\lambda_9 + 1.0k_2u_2 = 0 \\ \frac{\partial H}{\partial u_3} &= Sp\lambda_{15}\sigma - Sp\lambda_{16}\sigma + 1.0k_3u_3 = 0 \\ \frac{\partial H}{\partial u_4} &= -C\lambda_9 - I\lambda_{10} - Iit\lambda_{11} - It\lambda_{12} + 1.0k_4u_4 + \lambda_{13}(I + Iit + It) = 0\end{aligned}$$

where

$$\begin{aligned}\Gamma_3 &= \beta_3 \left(\frac{Ipq_1}{Np} + \frac{Cq_2 + Dq_5 + Iq_3 + Itq_4}{N} \right) \\ \Gamma_4 &= \beta_4 \left(\frac{Ipz_1}{Np} + \frac{Cz_2 + Dz_5 + Iz_3 + Itz_4}{N} \right)\end{aligned}$$

Substituting

$$u_1 = u_1^*, u_2 = u_2^*, u_3 = u_3^*, u_4 = u_4^*$$

and solving for the optimal control $(u_1^*, u_2^*, u_3^*, u_4^*)$ we obtain

$$\begin{aligned}u_1^* &= \frac{\lambda_8(Suc\Gamma_3 + Sun\Gamma_4) - Suc\lambda_6\Gamma_3 - Sun\lambda_7\Gamma_4}{k_1} \\ u_2^* &= \frac{E(\lambda_8 - \lambda_9) - \lambda_{13}C}{k_2} \\ u_3^* &= \frac{Sp\sigma(\lambda_{16} - \lambda_{15})}{k_3} \\ u_4^* &= \frac{I\lambda_{10} + Iit\lambda_{11} + It\lambda_{12} - \lambda_{13}(I + It + Iit)}{k_4}\end{aligned}$$

Hence, the required optimal control condition is obtained as

$$\begin{aligned}u_1^* &= \min \left\{ 1, \max \left[0, \frac{\lambda_8(Suc\Gamma_3 + Sun\Gamma_4) - Suc\lambda_6\Gamma_3 - Sun\lambda_7\Gamma_4}{k_1} \right] \right\} \\ u_2^* &= \min \left\{ 1, \max \left[0, \frac{E(\lambda_8 - \lambda_9) - \lambda_{13}C}{k_2} \right] \right\} \\ u_3^* &= \min \left\{ 1, \max \left[0, \frac{Sp\sigma(\lambda_{16} - \lambda_{15})}{k_3} \right] \right\}\end{aligned}$$

$$u_4^* = \min \left\{ 1, \max \left[0, \frac{I\lambda_{10} + I_{it}\lambda_{11} + I_t\lambda_{12} - \lambda_{13}(I + I_t + I_{it})}{k_4} \right] \right\}$$

It is discovered that the optimality requirements derived by considering the derivatives of the Hamiltonian (3.6) with regard to the controls only hold in the innermost regions of the control set U . □

4. OPTIMAL CONTROL MODEL NUMERICAL SIMULATIONS

The Python programming language is used in this part to implement numerical answers to the optimum system. We looked at four factors that rely on the neighbors of the state variables: $u_1, u_2, u_3,$ and u_4 . We run the model both with and without supervision, and then we contrast the outcomes. Because the limits are not completely effective, we took into account their number values between zero(0) and one(1): $u_1, u_2, u_3,$ and u_4 . The parameter values used in the models are displayed in Tables 4.1 and 4.2. The visually optimized system's numerical answer, obtained using Python software, is displayed below. Unlike state variables, which have starting conditions, adjoint variables have final conditions [[5], [9]].

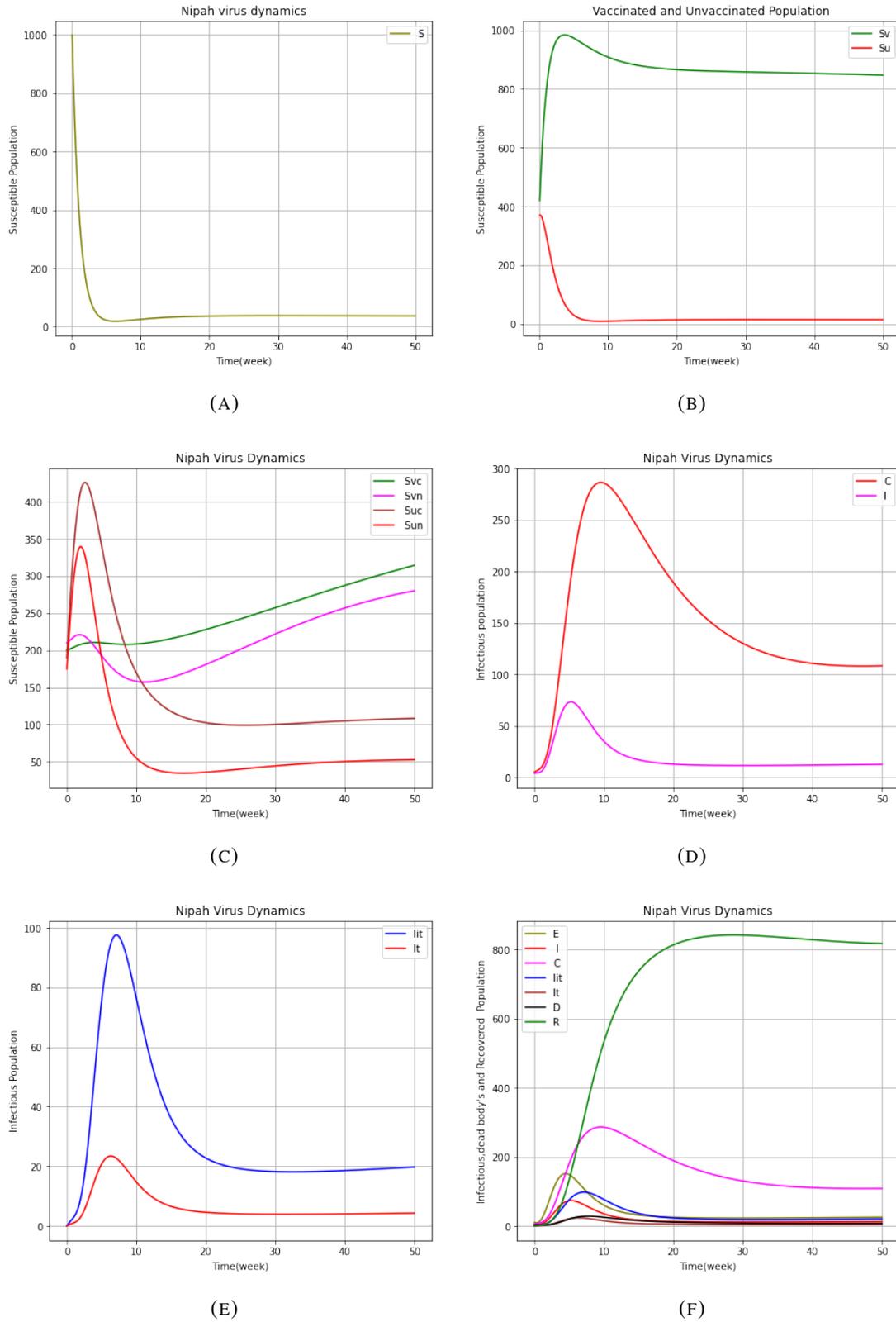


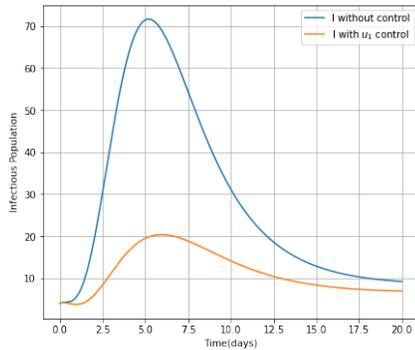
FIGURE 4.1. The solution of the model

Parameters	Value	Source
χ_1	0.33	Estimated
χ_2	0.62	[5]
θ	0.486	[5]
κ	0.715	[5]
τ_1	0.008	Estimated
τ_2	0.019	Estimated
η_1	0.45	Estimated
η_2	0.39	Estimated
ψ_1	0.825	Estimated
ψ_2	0.342	Estimated
γ_1	0.8	Inferred from [9]
γ_2	0.5	Inferred from [9]
γ_3	0.09	Inferred from [9]
γ_4	0.1	[9]
β_1	0.1134	Inferred from [9]
β_2	0.3969	Inferred from [9]
β_3	0.4455	Inferred from [9]
β_4	0.7209	Inferred from [9]
δ_1	0.02	Inferred from [9]
ρ	0.56	[5]
σ	0.75	[5]

TABLE 4.1. Description of the parameters

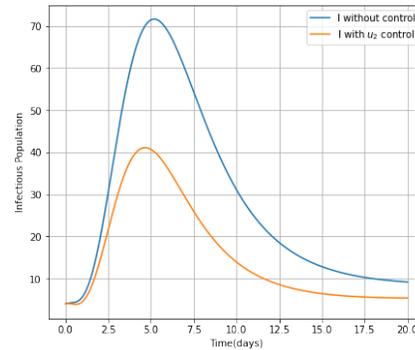
Parameters	Value	Source
δ_2	0.15	[9]
δ_3	0.0171	Inferred from [9]
δ_4	0.2	Inferred from [9]
a_1	0.58	Inferred from [9]
a_2	0.513	Inferred from [9]
a_3	0.486	Inferred from [9]
a_4	0.513	Inferred from [9]
a_5	0.000288	Inferred from [9]
b_1	0.69	Inferred from [9]
b_2	0.522	Inferred from [9]
b_3	0.513	Inferred from [9]
b_4	0.504	Inferred from [9]
b_5	0.000324	Inferred from [9]
q_1	0.75	Inferred from [9]
q_2	0.4617	Inferred from [9]
q_3	0.531	Inferred from [9]
q_4	0.513	Inferred from [9]
q_5	0.000648	Inferred from [9]
ε	0.03	Estimated
z_2	0.4374	Inferred from [9]
z_3	0.504	Inferred from [9]
z_4	0.513	Inferred from [9]
z_5	0.000648	Inferred from [9]
μ_p	0.00081	Estimated
μ	0.0003421	[17]

TABLE 4.2. Description of the parameter



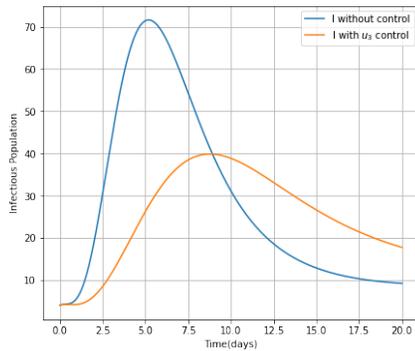
(A) Protection control

$$u_1 \neq 0, u_2 = u_3 = u_4 = 0$$



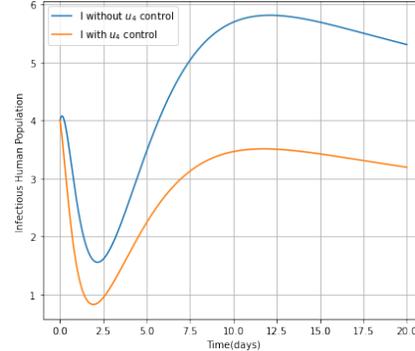
(B) Rapid testing $u_2 \neq 0$

$$0, u_1 = u_3 = u_4 = 0$$



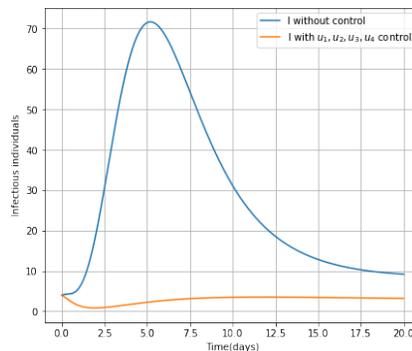
(C) Burying infected pigs

$$u_3 \neq 0, u_1 = u_2 = u_4 = 0$$



(D) Therapy control

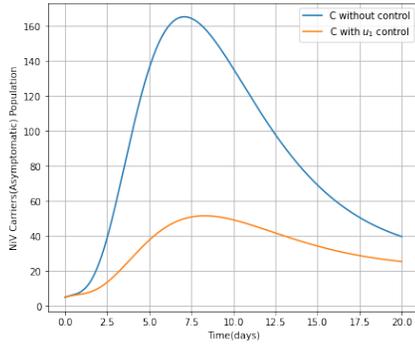
$$u_4 \neq 0, u_1 = u_3 = u_2 = 0$$



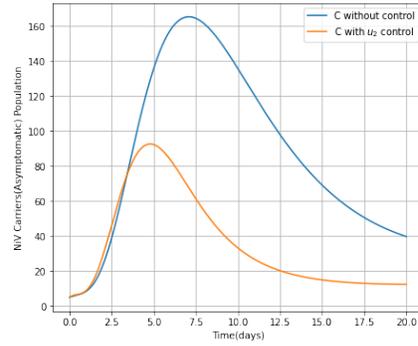
(E) Combing controls

$$u_2 = u_1 = u_3 = u_4 \neq 0$$

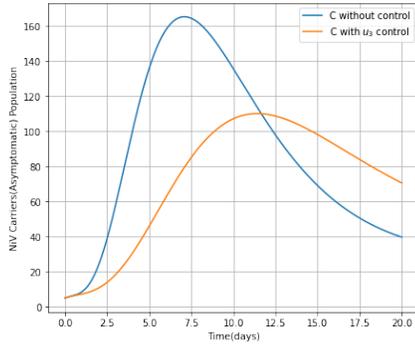
FIGURE 4.2. Simulations Showing The Effect of Controls u_1, u_2, u_3, u_4 on Infectious Human Population I



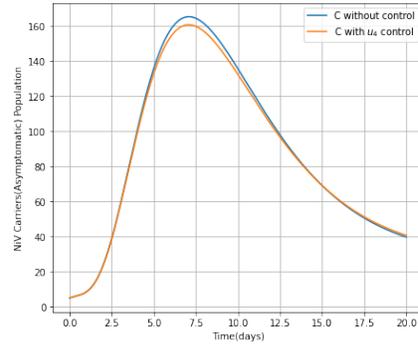
(A) Protection control
 $u_1 \neq 0, u_2 = u_3 = u_4 = 0$



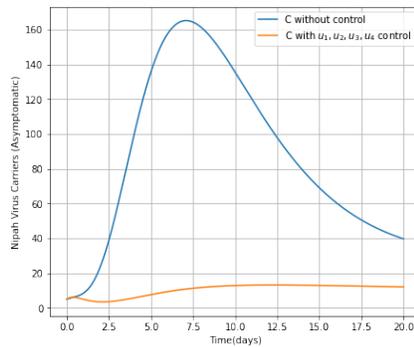
(B) Rapid testing $u_2 \neq 0, u_1 = u_3 = u_4 = 0$



(C) Burying infected pigs $u_3 \neq 0, u_1 = u_2 = u_4 = 0$

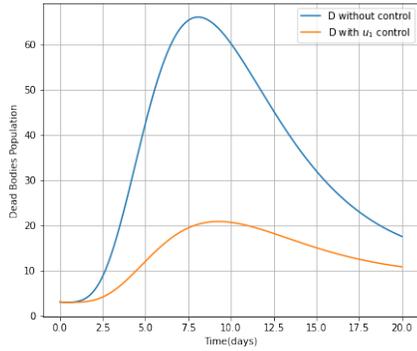


(D) Therapy control $u_4 \neq 0, u_1 = u_3 = u_2 = 0$



(E) Combing controls $u_2 = u_1 = u_3 = u_4 \neq 0$

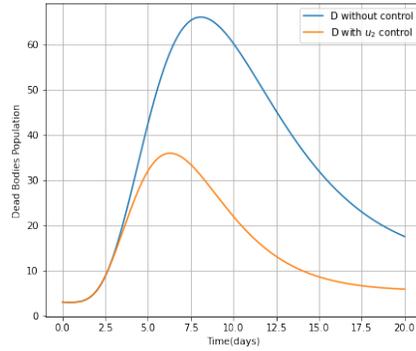
FIGURE 4.3. Simulations Showing The Effect of Controls u_1, u_2, u_3, u_4 on NiV carriers(Asymptotic) Population C



(A) Protection control

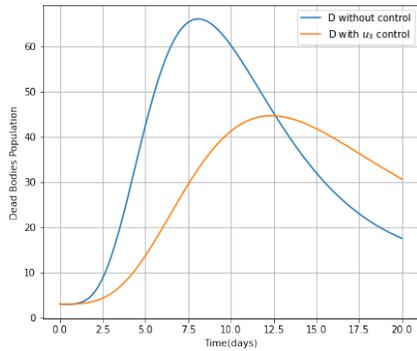
$$u_1 \neq 0, u_2 = u_3 = u_4 =$$

$$0$$



(B) Rapid testing $u_2 \neq$

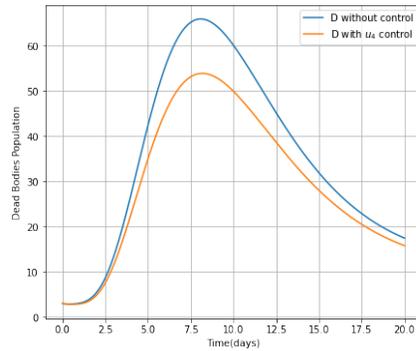
$$0, u_1 = u_3 = u_4 = 0$$



(C) Burying infected

$$\text{pigs } u_3 \neq 0, u_1 = u_2 =$$

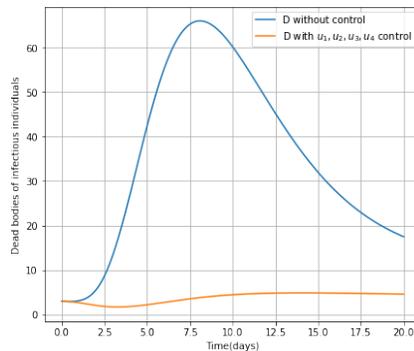
$$u_4 = 0$$



(D) Therapy control

$$u_4 \neq 0, u_1 = u_3 = u_2 =$$

$$0$$



(E) Combing controls

$$u_2 = u_1 = u_3 = u_4 \neq 0$$

FIGURE 4.4. Simulations Showing The Effect of Controls u_1, u_2, u_3, u_4 on Dead bodies Population I

Figure 4.1 depicts the schematic solutions of the model system. Figures 4.1(A) and 4.1(C) plainly show the importance of vaccine in a vulnerable community. Furthermore, figure 4.1(C) shows the fact that combining vaccine and condom use are beneficial management strategy for reducing NiV transmission. Furthermore, as demonstrated in figure 4.1(D), asymptomatic people are more infectious than symptomatic persons. This is because infectious people are more likely to go for healthcare assistance and be diagnosed with the illness, which can help hinder further spread of the disease through treatment or isolation measures, whereas asymptomatic people are more likely to interact with others without taking precautions or visiting a doctor because they do not show symptoms figure 4.1(E). This shows that, without vaccination and the use of condom, so many people are exposed to disease

Figures 4.2, 4.3 and 4.4 depict the impact of various control measures, including therapy controls, burying sick piglets, fast testing, and protection, on the infectious population I and NiV carriers (asymptomatic) population C , respectively. Each measure helps to contain the spread of NiV by lowering the population's proportion of NiV carriers (asymptomatic) and infectious individuals and dead bodies population. However, as shown in figure 4.2(E), 4.3 (e) and 4.4 (e), there is a substantial effect when combining the four controls at once. Such results are also experienced when apply to the exposed E as it drastically reduces the exposed individuals.

4.1. Conclusion and Recommendation. Public health policymakers, especially those in Cambodia, Ghana, Indonesia, Madagascar, the Philippines, and Thailand who are at risk for infection due to confirmation that the virus has been discovered in known natural reservoirs (Pteropus bat species) and many other bat species, will benefit from the availability of methods that can be used to determine the best way to stop the spread of Nipah Virus [14].

In this study, we investigated the optimal combination of personal protection, quick testing, burying sick pigs, and therapy control methods to restrict infection spread in the vaccine and condom Nipah virus model. The goal function of the optimal control issue was then defined. We create a vaccine and condom Nipah virus model and display the pictorial results. The four control strategy have been examined in the form of personal protection control(prevention) which involves minimizing the contact among the infectious people including death body of

Nipah virus individuals, restrict traveling where the cases are high, washing hands regularly using sanitizer and masks. Secondly, rapid testing of individuals to identify the Nipah virus carriers (C) and the infectious individuals for isolation and treatment. Thirdly, is burying sick pigs and finally the therapy control. We explored the presence of optimum control and then used the Hamiltonian and the Pontryagin's maximum principle to accomplish our goal. A contrast between a control plan with and without one is being watched. The impact of control factors is very noticeable in decreasing the number of infected people and controlling disease dynamics. The models show that the optimum combination of the four controls is very important for Nipah virus eradication. However, we recommend fast production of vaccine and its implementation with the use of condom.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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