OPTIMAL CONTROL MODEL FOR THE TRANSMISSION DYNAMICS OF MALARIA-PNEUMONIA CO-INFECTION WITH MASS ACTION INCIDENCE

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ABSTRACT

Malaria and Pneumonia are leading causes of serious illness in children and adults worldwide with their death rate and prevalence on the rise. Such alarming statistics may retard the milestones so far achieved in meeting the Millennium Development Goals 4 and 6 whose targets are to improve child survival and reverse the high prevalence of diseases such as pneumonia and malaria respectively. Two sub-models of malaria-pneumonia co-infection namely malaria model and pneumonia model were considered first and then followed by the full malaria-pneumonia co-infection model. The malaria model, pneumonia model and co-infection model basic reproduction numbers denoted by R_m, R_p and R_{mp} respectively was obtained using the Next Generation Matrix method. The model disease free equilibrium's local and global stability was analysed using Descartes' Rule of signs and Comparison method. The bifurcation analysis for the malaria, pneumonia and co-infection models was studied using the Centre Manifold Theory. The sensitivity indices of the model basic reproduction numbers R_m, R_p and R_{mp} to the parameters in the models were calculated. Optimal control theory was applied using the Pontryagins' Maximum Principle to investigate optimal strategies for controlling the spread of malaria, pneumonia and co-infection models using insecticide treated bed nets $(u_1(t))$, spraying of mosquitoes insecticides $(u_2(t))$, sanitation $(u_3(t))$, vaccination $(u_4(t))$, anti-malaria drugs $(u_5(t))$, anti-pneumonia drugs $(u_6(t))$, both anti-malaria drugs and anti-pneumonia drugs $(u_7(t))$ as the system time control variables. Numerical simulations using a set of parameter values were provided to validate the analytical results.

Keyword: Malaria, Mass Action, Malaria-Pneumonia, Optimal Control, Pneumonia

1.0   INTRODUCTION

Pneumonia and Malaria are two of the most deadly diseases of our time. The geographic overlap of these diseases in sub-Saharan Africa facilitates their co-infection. Although the consequences of the co-infection with pneumonia and malaria parasites are not fully understood in literature, available evidence now suggests that the infections act synergistically and their combination results in worse outcomes which poses a major public health concern. Malaria in an individual is an illness or disease that is caused by the parasites of the genus *Plasmodium* (Phylum Apicomplexa) in the blood or tissues [22]. The parasitic disease malaria is transmitted to the human through a biting from an infected female *Anopheles* mosquito [3]. The female *Anopheles* mosquito gets infected when it takes a blood meal from a person carrying the malaria parasite. There are four species of the plasmodium parasites, namely *Plasmodium falciparum*, *Plasmodium ovale*, *Plasmodium vivax* and *Plasmodium malariae*, of the four species, *Plasmodium Falciparum* is the most virulent, lethal and responsible for the majority of morbidity

and mortality due to malaria [10]. Evidence of human infections with several species of *Plasmodium* from higher apes has been documented except for *P. knowlesi*—a zoonotic species that causes malaria in macaques these are mostly of limited public health importance [14]. Many children living in malaria-endemic areas are frequently exposed to other diseases such as pneumonia. Children have a higher risk of developing pneumonia if they have weakened immune system. A weak immune system may be as a result of prolonged malaria exposure, malnutrition among other factors. Statistics has shown that of all children out patients suffering from respiratory complications 25% of the cases are confirmed to be pneumonia [24].

Pneumonia is one of the forgotten killer diseases [37]. It is one of the major causes of children's' death in Africa and it kills more children per year than any other illness in the world [37]. The death rates are around 2 million children worldwide; every year [37]. Pneumonia is an inflammatory condition of the lungs; it is an airborne disease acquired through inhalation or aspiration of pulmonary pathogenic organisms into a lung segment or lobe [26]. Pneumonia could be caused by bacterial, virus, fungi and parasites [26]. The parasites infections usually enter the body through the skin or the mouth and progresses through the body to the lungs, usually through the blood [32]. Prevention of diseases such as pneumonia includes vaccination, environmental measures and appropriate treatment of other health problems [32].

.   Malaria and Pneumonia account for about 8% and 19% of annual deaths in children [21]. The combination of both diseases contributes about 40% of the deaths in children below five years of age in sub-Saharan Africa [21]. Over six million new cases of pneumonia are estimated annually in Nigeria while childhood fevers presumed to be malaria account for 30% of all childhood deaths [31]. The WHO/UNICEF guidelines defines "malaria" as the presence or history of fever and symptoms that also occur in children with pneumonia, while "pneumonia" includes history of cough or difficulty in breathing in the presence of increased respiratory rate according to age and symptoms that may also indicate malaria [17]. Children who have malaria-pneumonia symptom overlap are given dual integrated management of childhood illness (IMCI) classifications and are treated with both antimalarial and antibiotics [19]. In Eastern Africa, the extent of the overlap has been documented but not in routine IMCI practice at the health centre level in Nigeria [22].

          Mathematical models has been widely used to study and explains the transmission dynamics of the spread of malaria following the WHO position statement [36] that it is important to carry out modelling studies to determine the impact of various combinations of control strategies on the transmission dynamics of malaria. Series of studies have been done to quantify the impact of malaria infection in humans ([8], [18] and [20]). Many of these studies discusses only the transmission of the malaria disease in human and the vector populations, however,   recent studies by Chiyaka *et al.*[13] focused on a deterministic system of differential equations involving two latent periods in the non-constant host and vector populations in order to analytically assess the potential impact of personal protection, treatment and possible vaccination strategies on the transmission dynamics of malaria. Blayneh *et al.* [7] formulated a time dependent model to consider the effects of prevention and treatment on malaria, while in similar fashion, Okosun *et al.* [28] studied a time dependent model on the possible impact of vaccination with treatment strategies in controlling the spread of malaria in a model that incorporate treatment and vaccination with waning immunity. Agusto *et al.* [2] studied a deterministic system of differential equations for the transmission of malaria and thereafter considered the optimal control strategies to investigate optimal strategies for controlling the spread of malaria disease using treatment, insecticide treated bed nets and spraying of mosquito insecticide as the system control variables. The possible impact of using combinations of the three controls either one at a time or two at a time on the spread of the disease was also examined in their study.

Many mathematical model already exist describing malaria infection or pneumonia infection but the best control for malaria infection, pneumonia infection and the co-infection of both diseases still remain a subject of debate. Previous mathematical models have considered treatment, sleeping under mosquito treated bedside nets (MTBN) and the use of insecticide coil as controls. However, these have their limitations. The insecticides used for treating bedside nets is lethal to the mosquitoes, other insects and also repels the mosquitoes, thus reducing the number of mosquitoes who attempt to feed on people in the sleeping areas with the nets [2]. However, the mosquitoes can still feed on humans outside these protective areas, hence; Agusto *et al* [2] included the spraying of insecticides in their model. The latter control by [2] will be more effective in a closed area, hence, the inclusion of sanitation as control in the present work. The preventive and treatment controls for pneumonia includes vaccination, environmental measures and appropriate treatment of other health problems [32]. It is believed that, if appropriate preventive measures were instituted globally, mortality among children could be reduced by 400,000; and, if proper treatment were universally available, childhood deaths could be decreased by another 600,000 [38].

The rest of the paper are organised in the following way: The mathematical formulation for the full co-infection model was presented in section 2, section 3 focused on the analysis of the sub models namely: malaria only model and pneumonia only model, this was later followed by the analysis of the full model on malaria-pneumonia co-infection. The optimal control analysis for the malaria only, pneumonia only and the co-infection model was considered in section 4 while the numerical simulations and the discussion of results was presented in section 5.

2.0   MATHEMATICAL FORMULATION

Lawi *et al* [24] studied the following model on malaria-pneumonia co-infection with standard incidence:

$$
\begin{aligned}
&S_h{'}(t) = \Lambda_h - \frac{\alpha\beta_m I_v}{N_h}S_h - \beta_p c(I_p + kI_{mp})S_h - \mu_h S_h + \pi I_m + \tau I_p + \phi I_{mp} \\
&I_m{'}(t) = \frac{\alpha\beta_m I_v}{N_h}S_h - \frac{\vartheta\beta_p c(I_p + kI_{mp})}{N_h}I_m - (\sigma_m + \pi + \mu_h)I_m \\
&I_p{'}(t) = \frac{\beta_p c(I_p + kI_{mp})}{N_h}S_h - \frac{\varepsilon\alpha\beta_m I_v}{N_h}I_p - (\sigma_p + \tau + \mu_h)I_p \\
&I_{mp}{'}(t) = \frac{\varepsilon\alpha\beta_m I_v}{N_h}I_p + \frac{\vartheta\beta_p c(I_p + kI_{mp})}{N_h}I_m - (\sigma_m + \sigma_p + \sigma_{mp} + \phi + \mu_h)I_{mp} \\
&S_v{'}(t) = \Lambda_v N_v - \frac{\alpha\beta_v c(I_m + \delta I_{mp})}{N_h}S_v - \mu_v S_v \\
&I_v{'}(t) = \frac{\alpha\beta_v c(I_m + \delta I_{mp})}{N_h}S_v - \mu_v I_v
\end{aligned} \right\} \quad (2.1)
$$

Cai *et al* [11] replaced the standard incidence with the mass action incidence in their study of malaria model with partial immunity to reinfection. Thus, following the modification of the work of Lawi *et al* [24], Oluyo and Adeniyi [29] studied the following model (2.2) on the co-infection of malaria-pneumonia using the mass action incidence.

$$S_h{'}(t) = \Lambda_h - \alpha\beta_m I_v S_h - \beta_p c(I_p + kI_{mp})S_h - \mu_h S_h + \pi I_m + \tau I_p + \phi I_{mp}$$

$$I_m{'}(t) = \alpha\beta_m I_v S_h - \vartheta\beta_p c(I_p + kI_{mp})I_m - (\sigma_m + \pi + \mu_h)I_m$$

$$I_p{'}(t) = \beta_p c(I_p + kI_{mp})S_h - \varepsilon\alpha\beta_m I_v I_p - (\sigma_p + \tau + \mu_h)I_p$$

$$I_{mp}{'}(t) = \varepsilon\alpha\beta_m I_v I_p + \vartheta\beta_p c(I_p + kI_{mp})I_m - (\sigma_m + \sigma_p + \sigma_{mp} + \phi + \mu_h)I_{mp}$$

$$S_v{'}(t) = \Lambda_v - \alpha\beta_v(I_m + \delta I_{mp})S_v - \mu_v S_v$$

$$I_v{'}(t) = \alpha\beta_v(I_m + \delta I_{mp})S_v - \mu_v I_v$$

(2.2)

One intervention recommended to control malaria transmission in the work of [29] was keeping clean environment. For pneumonia, intervention can be in two ways: reducing the number of susceptible individuals through vaccination or reducing the rate of contact by good sanitation. Based on the above submission, the model in (2.2) was further extended to include the exposed classes for malaria, pneumonia and malaria-pneumonia, vaccination for those exposed to pneumonia and the sanitation function for malaria and pneumonia. Thus, the improved model is:

$$S_h{'}(t) = \Lambda_h - \alpha\beta(H)I_v S_h - \beta_p c(H)(I_p + kI_{mp})S_h - \mu_h S_h + \pi I_m$$
$$+ \pi E_m + \tau I_p + \tau E_p + V_p E_p + \phi I_{mp} + \phi E_{mp}$$

$$E_m{'}(t) = \alpha\beta(H)I_v S_h - (k_m + \pi + \mu_h)E_m$$

$$I_m{'}(t) = k_m E_m - \vartheta\beta_p c(H)(I_p + kI_{mp})I_m - (\sigma_m + \pi + \mu_h)I_m$$

$$E_p{'}(t) = \beta_p c(H)(I_p + kI_{mp})S_h - (k_p + \tau + V_p + \mu_h)E_p$$

$$I_p{'}(t) = k_p E_p - \varepsilon\alpha\beta(H)I_v I_p - (\sigma_p + \tau + \mu_h)I_p$$

$$E_{mp}{'}(t) = \varepsilon\alpha\beta(H)I_v I_p + \vartheta\beta_p c(H)(I_p + kI_{mp})I_m - (k_{mp} + \phi + \mu_h)E_{mp}$$

$$I_{mp}{'}(t) = k_{mp} E_{mp} - (\sigma_m + \sigma_p + \sigma_{mp} + \phi + \mu_h)I_{mp}$$

$$S_v{'}(t) = \Lambda_v - \alpha\beta_v(I_m + \delta I_{mp})S_v - \mu_v S_v$$

$$E_v{'}(t) = \alpha\beta_v(I_m + \delta I_{mp})S_v - (k_v + \mu_v)E_v$$

$$I_v{'}(t) = k_v E_v - \mu_v I_v$$

(2.3)

The model (2.3) subdivides the total human population of interest into two sub population depending on the malaria and pneumonia status of individuals. The classes consist of Susceptible (S) representing the number of individuals who are at risk of acquiring malaria or pneumonia or both infections, Individuals Exposed to malaria or pneumonia or both infections (E), Infective (I) representing infectious malaria individuals or infectious pneumonia individuals or infectious malaria-pneumonia individuals capable of transmitting infection to susceptible individuals. The constant per capita recruitment rate into susceptible human population is Λ_h while humans die naturally at a rate μ_h, the modification parameter according to the increased susceptibility to infection with pneumonia is denoted ϑ, humans die as a result of malaria infection at rate σ_m while humans die of pneumonia infection at a rate σ_p. Individual become infected with pneumonia by coming in contact with one infectious individual at rate β_p. Malaria and pneumonia induced mortality occur at rate σ_{mp}, rate of recovery from malaria, pneumonia and malaria-pneumonia back to the susceptible class are denoted by π, τ and ϕ respectively while α denote the number of bites per human per mosquito. The per capita rate of progression of humans from the exposed class E_m to the infectious class I_m is k_m with $\frac{1}{k_m}$ taking as the average duration of the latent period, k_p represent the per capita rate of progression

of humans from the exposed class E_p to the infectious class I_p with $\frac{1}{k_p}$ denoting the average duration of the latent period while k_{mp} is the per capita rate of progression of humans from the exposed class E_{mp} to the infectious class I_{mp}. $\frac{1}{k_{mp}}$ is the average duration of the latent period.

Exposed individuals to pneumonia are vaccinated at constant rate V_p. The mosquitoes (vector) population are sub-divided into susceptible mosquitoes(S_v), mosquitoes exposed to malaria (E_v) and infected mosquitoes (I_v) respectively. The per capita recruitment rate of mosquitos into susceptible vector population is Λ_v, mosquito become infected with malaria after taking a blood from any infected human with malaria at a rate β_v, exposed mosquitoes (E_v) progress to the infectious class (I_v) at a constant rate k_v with $\frac{1}{k_v}$ being the average duration of the latent period, the mosquito natural death rate is μ_v. ε, denote the expected decrease in contact due to ill health as a result of pneumonia disease such that $0 < \varepsilon < 1$. The modification parameters accounting for the relative infectiousness of the co-infected individual as compared to their counterparts are δ and k respectively. $\beta(H)$ is the transmission rate of malaria in humans expressed as function (linearly decreasing function) of the effectiveness of sanitation H and is defined by

$$\beta(H) = \beta_{max} - \gamma H \tag{2.4}$$

γ represent the expected reduction / increase in transmission rate of malaria as sanitation level H increases / decreases. H is the sanitation level of the community and is defined to be $H \in [0,1]$, so that if $H = 0$, then there is maximum transmission of malaria in the community and if $H = 1$ means there is access to maximum sanitation facilities in the community, hence, minimum transmission rate of malaria is achieved. $C(H)$ is the contracting rate of pneumonia expressed as function (linearly decreasing function) of the effectiveness of sanitation H and is defined by

$$C(H) = C_{max} - \gamma H \tag{2.5}$$

when $H = 0$, then there is maximum transmission of pneumonia in the community and when $H = 1$ means there is minimum contacting rate of pneumonia in the community.

The assumptions for the model in system of equations (2.3) are as follows:

i. This model assumes a homogeneous mixing of individuals in the population where all individuals have equal likelihood of contracting the infection if they come into effective contact with infectious individuals or infectious mosquitoes and that transmission of the infection occurs with a mass action incidence rate.

ii. Infected individuals are assumed to recover with no permanent immunity and return to the susceptible class.

iii. Human population is assumed not to be constant since birth, immigration, emigration and death occur in the population.

iv. The probability of survival till the infectious state for individual exposed to malaria as well as those exposed to pneumonia is less or equal to unity.

v. That as a result of the assumption (iv) above, the exposed class of individuals in the population are therefore included.

3.0   Malaria – only sub model

Before the full model of system (2.3) is analysed, it is important to gain insight into the dynamics of the malaria – only sub model obtained by setting $E_p(t) = E_{mp}(t) = I_p(t) = I_{mp}(t) = 0$ in (2.3) given by

$$S_h{'}(t) = \Lambda_h - \alpha\beta(H)I_v S_h - \mu_h S_h + \pi E_m + \pi I_m$$

$$E_m{'}(t) = \alpha\beta(H)I_v S_h - (k_m + \pi + \mu_h)E_m$$

$$I_m{'}(t) = k_m E_m - (\sigma_m + \pi + \mu_h)I_m$$

$$S_v{'}(t) = \Lambda_v - \alpha\beta_v I_m S_v - \mu_v S_v$$

$$E_v{'}(t) = \alpha\beta_v I_m S_v - (k_v + \mu_v)E_v$$

$$I_v{'}(t) = k_v E_v - \mu_v I_v$$

$$(3.1)$$

Let the total human population and vector population at any time t be denote by $N_h(t)$ and $N_v(t)$ respectively, then the total human and vector population for the malaria – only model of (3.1) are given by

$$N_h(t) = S_h(t) + E_m(t) + I_m(t) \tag{3.2}$$

and

$$N_v(t) = S_v(t) + E_v(t) + I_v(t) \tag{3.3}$$

respectively. It is easy to show that both human and vector population will reach a constant value over time. i.e. $N_h(t) = \frac{\Lambda_h}{\mu_h}$ and $N_v(t) = \frac{\Lambda_v}{\mu_v}$.

For system (3.1), it is straightforward to verify that the region $\Gamma_{hv} = \{(S_h, E_m, I_m, S_v, E_v, I_v) \in$ $\mathcal{R}_+{}^6 : S_h + E_m + I_m \le \frac{\Lambda_h}{\mu_h}, S_v + E_v + I_v \le \frac{\Lambda_v}{\mu_v}\}$ is positively invariant and attracting. Thus the dynamics of malaria – only model will be analysed in Γ_{hv}.

The malaria – only model of (3.1) has a disease-free equilibrium point given by

$$M_0 = (S_{h0}{}^*, E_{m0}{}^*, I_{m0}{}^*, S_{v0}{}^*, E_{v0}{}^*, I_{v0}{}^*) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0\right) \tag{3.4}$$

and endemic equilibriums $M_1{}^* = (S_{h1}{}^*, E_{m1}{}^*, I_{m1}{}^*, S_{v1}{}^*, E_{v1}{}^*, I_{v1}{}^*)$ and $M_2{}^* = (S_{h2}{}^*, E_{m2}{}^*, I_{m2}{}^*, S_{v2}{}^*, E_{v2}{}^*, I_{v2}{}^*)$ given by the positive roots of

$$AI_m{}^{*2} + BI_m{}^* + C = 0 \tag{3.5}$$

with

$A = \alpha^3\beta(H)\beta_v{}^2 k_m k_v \Lambda_v \pi(\sigma_m + \pi + \mu_h) + \alpha^3\beta_v{}^2 k_m \pi - (k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)\alpha^3\beta(H)\beta_v{}^2 k_v \Lambda_v - (k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)\alpha^2\beta_v{}^2\mu_h\mu_v(k_v + \mu_v)$

$B = \alpha^3\beta(H)\beta_v{}^2 k_m k_v \Lambda_v \Lambda_h + \alpha^2\beta(H)\beta_v k_m k_v \Lambda_v \mu_v \pi(\sigma_m + \pi + \mu_h) + \alpha^2\beta(H)\beta_v k_m{}^2 k_v \Lambda_v \mu_v \pi - 2(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)\alpha\beta_v\mu_v{}^2\mu_h(k_v + \mu_v) - \alpha^2\beta(H)\beta_v\mu_v k_v \Lambda_v(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)$

$C = k_m k_v \alpha^2\beta(H)\beta_v \Lambda_v \Lambda_h \mu_v - \mu_h\mu_v{}^3(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)(k_v + \mu_v)$

The local stability of the equilibrium M_0 is governed by the basic reproduction number R_m. The basic reproduction number for the malaria model of (3.1) will be investigated using the next generation approach ([15], [35]). Using the notations in [35] on system (3.1), the matrices F and V, representing the new infections terms and transferred terms respectively are given by

$$F = \begin{pmatrix} 0 & 0 & 0 & (\alpha\beta_v\Lambda_v/\mu_v) \\ 0 & 0 & 0 & 0 \\ 0 & (\alpha\beta_v\Lambda_v/\mu_v) & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix};$$

$$V = \begin{pmatrix} (k_m + \pi + \mu_h) & 0 & 0 & 0 \\ -k_m & (\sigma_m + \pi + \mu_h) & 0 & 0 \\ 0 & 0 & (k_v + \mu_v) & 0 \\ 0 & 0 & -k_v & \mu_v \end{pmatrix}$$

Thus, the basic reproduction number for system (3.1), denoted by R_m is given by the spectral radius ρ of FV^{-1} (the dominant eigenvalue in magnitude)

$$R_m = \rho(FV^{-1}) = \sqrt{\frac{\alpha^2 \beta(H)\beta_v k_m k_v \Lambda_h \Lambda_v}{\mu_h \mu_v{}^2 (k_v + \mu_v)(\sigma_m + \pi + \mu_h)(k_m + \pi + \mu_h)}} \qquad (3.6)$$

The reproduction number R_m is the average number of secondary malaria infectious cases produced by a single infected mosquito during its infectious period when introduced in a population of mostly malaria susceptible individuals.

Theorem 3.1: The malaria disease - free equilibrium M_0 of system (3.1) is locally asymptotically stable if $R_m < 1$ and unstable if $R_m > 1$.

Proof: The Jacobian matrix of system (3.1) evaluated at M_0 is

$J(M_0) =$

$$\begin{pmatrix} -\mu_h & \pi & \pi & 0 & 0 & (-\alpha\beta(H)\Lambda_h/\mu_h) \\ 0 & -(k_m + \pi + \mu_h) & 0 & 0 & 0 & (\alpha\beta(H)\Lambda_h/\mu_h) \\ 0 & k_m & -(\sigma_m + \pi + \mu_h) & 0 & 0 & 0 \\ 0 & 0 & (-\alpha\beta_v\Lambda_v/\mu_v) & -\mu_v & 0 & 0 \\ 0 & 0 & (\alpha\beta_v\Lambda_v/\mu_v) & 0 & -(k_v + \mu_v) & 0 \\ 0 & 0 & 0 & 0 & k_v & -\mu_v \end{pmatrix} \qquad (3.7)$$

The characteristic equation of equation (3.7) is

$(-\mu_h - \lambda)(-\mu_v - \lambda)g(\lambda) = 0 \qquad (3.8)$

where

$g(\lambda) = (\lambda^4 + (k_m + 2\pi + 2\mu_h + 2\mu_v + \sigma_m + k_v)\lambda^3 + (k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma_m)(k_v + \mu_v) + (k_m + 2\pi + 2\mu_h + 2\mu_v + \sigma_m + k_v)\mu_v) \lambda^2 + \big(\mu_v((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma_m)(k_v + \mu_v)) + (k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)(k_v + \mu_v)\big) \lambda + \mu_v(k_v + \mu_v)(\sigma_m + \pi + \mu_h)(k_m + \pi + \mu_h) - \frac{\alpha^2\beta(H)\beta_v k_m k_v \Lambda_h \Lambda_v}{\mu_h \mu_v}$

Clearly, $\lambda_1 = -\mu_h$, $\lambda_2 = -\mu_v$ while $\lambda_3, \lambda_4, \lambda_5$ and λ_6 are obtained from $g(\lambda) = 0$ i.e

$\lambda^4 + (k_m + 2\pi + 2\mu_h + 2\mu_v + \sigma_m + k_v)\lambda^3 + \big((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma_m)(k_v + \mu_v) + (k_m + 2\pi + 2\mu_h + 2\mu_v + \sigma_m + k_v)\mu_v\big)\lambda^2 + \big(\mu_v((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma_m)(k_v + \mu_v)) + (k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)(k_v + \mu_v)\big)\lambda + \mu_v(k_v + \mu_v)(\sigma_m + \pi + \mu_h)(k_m + \pi + \mu_h) - \frac{\alpha^2\beta(H)\beta_v k_m k_v \Lambda_h \Lambda_v}{\mu_h \mu_v} = 0 \qquad (3.9)$

Equation (3.9) will have four negative real roots (by Descartes rule of positive solutions) if

$$\frac{\alpha^2\beta(H)\beta_v k_m k_v \Lambda_h \Lambda_v}{\mu_h \mu_v} < \mu_v(k_v + \mu_v)(\sigma_m + \pi + \mu_h)(k_m + \pi + \mu_h)$$

$$\Rightarrow \frac{\alpha^2\beta(H)\beta_v k_m k_v \Lambda_h \Lambda_v}{\mu_h \mu_v{}^2(k_v + \mu_v)(\sigma_m + \pi + \mu_h)(k_m + \pi + \mu_h)} < 1$$

$R_m{}^2 < 1$

$\therefore R_m < 1$

Hence, the malaria disease – free equilibrium M_0 of system (3.1) is locally asymptotically stable if $R_m < 1$. The result follows immediately that M_0 is unstable if $R_m > 1$.

Theorem 3.2: The malaria disease - free equilibrium M_0 of system (3.1) is globally asymptotically stable if $R_m < 1$ and unstable if $R_m > 1$.

Proof: The Comparison method as implemented in Lashmkantham, *et al* [23] and Mushayabasa *et al* [27] is used here. The rate of change of the exposed and infected components of system (3.1) can be written as

$$
\begin{pmatrix} \dfrac{dE_m}{dt} \\ \dfrac{dI_m}{dt} \\ \dfrac{dE_v}{dt} \\ \dfrac{dI_v}{dt} \end{pmatrix} = (F - V) \begin{pmatrix} E_m \\ I_m \\ E_v \\ I_v \end{pmatrix} - \left(1 - \dfrac{S_h}{N_h}\right)\left(1 - \dfrac{S_v}{N_v}\right) F \begin{pmatrix} E_m \\ I_m \\ E_v \\ I_v \end{pmatrix}
\tag{3.10}
$$

where F and V are as defined above. Since at the disease free $E_m = I_m = E_v = I_v = 0 \rightarrow (0,0,0,0)$ and $S_h \leq N_h, S_v \leq N_v$ as $t \rightarrow \infty$ in Γ_{hv}. Thus,

$$
\begin{pmatrix} \dfrac{dE_m}{dt} \\ \dfrac{dI_m}{dt} \\ \dfrac{dE_v}{dt} \\ \dfrac{dI_v}{dt} \end{pmatrix} \leq (F - V) \begin{pmatrix} E_m \\ I_m \\ E_v \\ I_v \end{pmatrix}
\tag{3.11}
$$

Then all eigenvalues of the matrix $(F - V)$ have negative real parts i.e

$$
\lambda^4 + (k_m + 2\pi + 2\mu_h + 2\mu_v + \sigma_m + k_v)\lambda^3 + \Big((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma_m)(k_v + \mu_v) + (k_m + 2\pi + 2\mu_h + 2\mu_v + \sigma_m + k_v)\mu_v\Big)\lambda^2 + \Big(\mu_v\big((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma_m)(k_v + \mu_v)\big) + (k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)(k_v + \mu_v)\Big)\lambda +
$$

$$
\mu_v(k_v + \mu_v)(\sigma_m + \pi + \mu_h)(k_m + \pi + \mu_h) - \dfrac{\alpha^2 \beta(H)\beta_v k_m k_v \Lambda_h \Lambda_v}{\mu_h \mu_v} = 0
\tag{3.12}
$$

Equation (3.12) have four negative roots by Descartes rule of signs if $R_m < 1$. It follows that the linearized differential inequality (3.11) is stable whenever $R_m < 1$. Consequently, $(E_m, I_m, E_v, I_v) \rightarrow (0,0,0,0)$ as $t \rightarrow \infty$. Evaluating system (3.1) at $E_m = I_m = E_v = I_v = 0$ gives, $S_h \rightarrow 1$ and $S_v \rightarrow 1$ for $R_m < 1$. Hence, the malaria diseases free equilibrium M_0 of system (3.1) is globally asymptotically stable if $R_m < 1$. The result also follow immediately that the malaria disease – free equilibrium M_0 of system (3.1) is unstable if $R_m > 1$.

3.1   Local Asymptotic Stability of Malaria Endemic Equilibrium

The Centre Manifold theorem is now applied to study the local stability of the malaria endemic equilibrium.

Let $\beta(H) = \beta(H)^*$ be a bifurcation parameter and if the case $R_m = 1$ is considered, then

$$
\beta(H) = \beta(H)^* = \frac{\mu_h \mu_v^2 (k_v + \mu_v)(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)}{\alpha^2 \beta_v k_m k_v \Lambda_h \Lambda_v}
\tag{3.13}
$$

Firstly, the Jacobian matrix of system (3.1) at point $(M_0, \beta(H)^*)$ is

$J(M_0, \beta(H)^*)$

$$
= \begin{pmatrix}
-\mu_h & \pi & \pi & 0 & 0 & (-\alpha\beta(H)^*\Lambda_h/\mu_h) \\
0 & -(k_m + \pi + \mu_h) & 0 & 0 & 0 & (\alpha\beta(H)^*\Lambda_h/\mu_h) \\
0 & k_m & -(\sigma_m + \pi + \mu_h) & 0 & 0 & 0 \\
0 & 0 & (-\alpha\beta_v\Lambda_v/\mu_v) & -\mu_v & 0 & 0 \\
0 & 0 & (\alpha\beta_v\Lambda_v/\mu_v) & 0 & -(k_v + \mu_v) & 0 \\
0 & 0 & 0 & 0 & k_v & -\mu_v
\end{pmatrix} \quad (3.14)
$$

The characteristic equation of (3.14) has a simple zero eigenvalue i.e.
$\lambda_1 = -\mu_h$, $\lambda_2 = -\mu_v$, $\lambda_3 = 0$, while the remaining equation is written as

$$
\lambda^3 + (k_m + 2\pi + 2\mu_h + 2\mu_v + \sigma_m + k_v)\lambda^2 + \big((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma_m)(k_v + \mu_v) + (k_m + 2\pi + 2\mu_h + 2\mu_v + \sigma_m + k_v)\mu_v\big)\lambda + \Big(\mu_v\big((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma_m)(k_v + \mu_v)\big) + (k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)(k_v + \mu_v)\Big) = 0
$$
$$(3.15)$$

Equation (3.15) has three negative eigenvalues as its roots (by Descartes rule of signs). Thus, $\lambda_3 = 0$ is a simple zero eigenvalue and the other eigenvalues are real and negative, then the assumptions of theorem A.1 (Centre Manifold theorem) in the appendix is then verified. Furthermore, the right and left eigenvectors associated with the zero eigenvalue $\lambda_3 = 0$ are

w

$$
= \left[\frac{(\pi\mu_h\mu_v{}^2(k_v + \mu_v)(\sigma_m + \pi + \mu_h) + \pi\mu_h\mu_v{}^2 k_m(k_v + \mu_v) - \alpha^2\beta(H)^*\beta_v k_m k_v \Lambda_h \Lambda_v)w_2}{\mu_h(k_v + \mu_v)(\sigma_m + \pi + \mu_h)}, w_2, \right.
$$
$$
\left. \frac{k_m w_2}{(\sigma_m + \pi + \mu_h)}, -\frac{\alpha\beta_v\Lambda_v k_m w_2}{\mu_v{}^2(\sigma_m + \pi + \mu_h)}, \frac{\alpha\beta_v\Lambda_v k_m w_2}{\mu_v(k_v + \mu_v)(\sigma_m + \pi + \mu_h)}, \frac{\alpha\beta_v k_m k_v \Lambda_v w_2}{\mu_v{}^2(k_v + \mu_v)(\sigma_m + \pi + \mu_h)} \right]^T
$$

and

$$
\bar{v} = \left[0, \bar{v}_2, \frac{(k_m + \pi + \mu_h)\bar{v}_2}{k_m}, 0, \frac{\mu_v(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)\bar{v}_2}{\Lambda_v k_m \alpha\beta_v}, \right.
$$
$$
\left. \frac{(k_v + \mu_v)(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)\mu_v\bar{v}_2}{\Lambda_v k_v k_m \alpha\beta_v} \right]
$$

where $w_2 > 0$ and $\bar{v}_2 > 0$ are free right and left eigenvectors.

Computation of the Coefficient a and b for the Malaria Model
After some rigorous mathematical manipulations using the associated partial non-zero partial derivatives of (3.1) at M_0, it can be shown that

$$
a = \frac{2\bar{v}_2\alpha w_2{}^2\beta_v(k_m + \pi + \mu_h)}{\mu_v(\sigma_m + \pi + \mu_h)}[a_0 - 1], \quad b = \bar{v}_2\frac{\alpha^2\beta_v k_m k_v w_2}{\mu_v(k_v + \mu_v)(\sigma_m + \pi + \mu_h)}
$$

where

$$
a_0 = \left(\frac{(\pi\mu_h\mu_v{}^2(k_v + \mu_v)(\sigma_m + \pi + \mu_h) + \pi\mu_h\mu_v{}^2 k_m(k_v + \mu_v) - \alpha^2\beta(H)^*\beta_v k_m k_v \Lambda_h \Lambda_v)}{\Lambda_h k_m(k_v + \mu_v)} \right)\mu_v
$$

According to theorem A.1 in the appendix it is the sign of the coefficient a - which depend on a_0 - that decides the local dynamics around the disease free equilibrium for $\beta(H) = \beta(H)^*$. Thus,
1. If $a_0 > 1$, then $a > 0$, thus a backward bifurcation occurs
2. If $a_0 < 1$, then $a < 0$, thus a forward bifurcation occurs
The foregoing discussion is summarized in the following results from theorem A.1 items (a) and (d):
Lemma 3.1: The malaria only model of (3.1) has a positive endemic equilibrium which is unstable

if $R_m < 1$ otherwise stable.

Lemma 3.2: The malaria only model of (3.1) has a unique positive endemic equilibrium which is locally asymptotically stable (LAS) if $R_m > 1$ otherwise unstable.

3.2 Sensitivity Analysis of (R_m)

The sensitivity index of the reproduction number is used to assess the impact on the relevant parameters to disease transmission. The sensitivity of the reproduction number, R_m is analysed to determine the parameter that are most sensitive to R_m. Following the approach in [6], the forward sensitivity index with respect to biting rates of the mosquitoes, α and treatment rate, π used in the malaria only model is presented below using the following formula

$$r_q^{R_m} = \frac{\partial R_m}{\partial q} \times \frac{q}{R_m} \tag{3.16}$$

$$\frac{\partial R_m}{\partial \alpha} \times \frac{q\alpha}{R_m} = 1 \tag{3.17}$$

$$\frac{\partial R_m}{\partial \pi} \times \frac{\pi}{R_m} = -\frac{\pi(k_m + \sigma_m + 2\pi + 2\mu_h)}{2(\sigma_m + \pi + \mu_h)(k_m + \pi + \mu_h)} \tag{3.18}$$

From the calculation in (3.17), it was observed that R_m is most sensitive to changes in the biting rates of the mosquitoes, α. An increase in number of mosquito bites α will bring about an increase of the same proportion in R_m and a decrease in α will result in a decrease in R_m with about an equivalent magnitude. Equation (3.18) imply that an increase in treatment of malaria infected individuals have a positive impact in controlling malaria in the population.

3.3   Pneumonia only Disease Transmission Model

Here, pneumonia disease transmission model only are investigated and analysed.

Let $E_m(t) = I_m(t) = E_v(t) = I_v(t) = E_{mp}(t) = I_{mp}(t) = 0$ in model system (2.3), then we have the following system of equations is obtained

$$\left.\begin{aligned}
S_h{}'(t) &= \Lambda_h - \beta_p c(H)I_p S_h - \mu_h S_h + \tau E_p + \tau I_p + V_p E_p \\
E_p{}'(t) &= \beta_p c(H)I_p S_h - \left(k_p + \tau + V_p + \mu_h\right)E_p \\
I_p{}'(t) &= k_p E_p - \left(\sigma_p + \tau + \mu_h\right)I_p \\
S_v{}'(t) &= \Lambda_v - \mu_v S_v
\end{aligned}\right\} \tag{3.19}$$

Note that the fourth equation in system (3.19) is independent of the other three equations then equation (3.19) can be re-written as

$$\left.\begin{aligned}
S_h{}'(t) &= \Lambda_h - \beta_p c(H)I_p S_h - \mu_h S_h + \tau E_p + \tau I_p + V_p E_p \\
E_p{}'(t) &= \beta_p c(H)I_p S_h - \left(k_p + \tau + V_p + \mu_h\right)E_p \\
I_p{}'(t) &= k_p E_p - \left(\sigma_p + \tau + \mu_h\right)I_p
\end{aligned}\right\} \tag{3.20}$$

with $N_h(t) = S_h(t) + E_p(t) + I_p(t)$. For system (3.20) it can be shown that the region $\Gamma = \Gamma_h \subset \mathcal{R}_+{}^3$ with $\Gamma_h = \left\{(S_h, E_p, I_p) \in \mathcal{R}_+{}^3 : S_h + E_p + I_p \leq \frac{\Lambda_h}{\mu_h}\right\}$

is positively invariant. Thus, in this region the model system (3.20) can be considered to be epidemiologically well-posed. The pneumonia free equilibrium is obtained to be

$$P_0 = \left(S_{h0}{}^*, E_{p0}{}^*, I_{p0}{}^*\right) = (\frac{\Lambda_h}{\mu_h}, 0, 0) \tag{3.21}$$

Following the approach in [15] and [35] as in section 3.0 on the analysis of malaria model. It can be shown that the reproduction number for system (3.20) denoted by R_p is given by

10

$$R_p = \frac{k_p \beta_p c(H) \Lambda_h}{\mu_h (\sigma_p + \tau + \mu_h)(k_p + \tau + V_p + \mu_h)} \tag{3.22}$$

The basic reproduction number R_p is the average number of new infections caused by a single pneumonia infected individual (but not infected with malaria) during his or her infectious period in a population of pneumonia susceptible s who have no malaria.

3.4.1 Sensitivity Analysis of the Reproduction Number, R_p

Here, the impact of treatment on pneumonia is investigated using the sensitivity of the reproduction number for pneumonia R_p. It follows from (3.22), that the sensitivity of R_p with respect to τ can be obtained as

$$\frac{\partial R_p}{\partial q} \times \frac{q}{R_p} = -\frac{\tau(k_p + \sigma_p + 2\tau + 2\mu_h)}{(k_p + \tau + \mu_h)(\sigma_p + \tau + \mu_h)} \tag{3.23}$$

Hence, the treatment of pneumonia patients will have a positive impact in reducing pneumonia burden. Thus the foregoing discussion is summarized in the following result:

Theorem 3.3: The pneumonia disease - free equilibrium P_0 of system (3.20) is locally asymptotically stable if $R_p < 1$ and unstable if $R_p > 1$.

Theorem 3.3 implies that pneumonia can be eliminated from the population (when $R_p < 1$) if the initial size of the sub-population of the model system (3.20) are in the basin of attraction of the disease free equilibrium P_0. In order to ensure that pneumonia elimination does not depend on the initial size of the sub-populations, it is important to establish that the DFE is globally asymptotically stable.

3.4.2 Global Stability of Pneumonia - Free Equilibrium

The rate of change of the exposed and the infected components of system (3.25) can be written as

$$\begin{pmatrix} \frac{dE_p}{dt} \\ \frac{dI_p}{dt} \end{pmatrix} = (F - V)\begin{pmatrix} E_p \\ I_p \end{pmatrix} - \left(1 - \frac{S_h}{N_h}\right)F\begin{pmatrix} E_p \\ I_p \end{pmatrix} \tag{3.24}$$

where $F = \begin{pmatrix} 0 & \frac{\beta_p c(H)\Lambda_h}{\mu_h} \\ 0 & 0 \end{pmatrix}$, $V = \begin{pmatrix} (k_p + \tau + V_p + \mu_h) & 0 \\ -k_p & (\sigma_p + \tau + \mu_h) \end{pmatrix}$

Since at the disease free $E_p = I_p = 0 \rightarrow (0,0)$ and $S_h \leq N_h$ as $t \rightarrow \infty$ in Γ_h. Thus,

$$\begin{pmatrix} \frac{dE_p}{dt} \\ \frac{dI_p}{dt} \end{pmatrix} \leq (F - V)\begin{pmatrix} E_p \\ I_p \end{pmatrix} \tag{3.25}$$

Then all eigenvalues of the matrix $(F - V)$ have negative real parts i.e

$$\lambda^2 + (k_p + 2\tau + V_p + 2\mu_h + \sigma_p)\lambda + (k_p + \tau + V_p + \mu_h)(\sigma_p + \tau + \mu_h) - k_p\beta_p c(H) = 0 \tag{3.26}$$

Equation (3.26) has its entire roots negative and real if $R_p < 1$

It follows that the linearized differential inequality (3.25) is stable whenever $R_p < 1$. Consequently, $(E_p, I_p,) \rightarrow (0,0)$ as $t \rightarrow \infty$. Evaluating system (3.20) at $E_p = I_p = 0$ gives $S_h \rightarrow 1$ for $R_p < 1$. The result is summarized in the following theorem:

Theorem 3.4: The pneumonia diseases free equilibrium P_0 of system (3.20) is globally asymptotically stable if $R_p < 1$ and unstable if $R_p > 1$.

3.4.3   Local Asymptotic Stability of Pneumonia Endemic Equilibrium

The Centre Manifold Theorem as used in [9] is employed here to establish the local asymptotic stability of the endemic equilibrium. The following change of variables are made in order to apply the Centre Manifold theory: Let $S_h = x_1$, $E_p = x_2$, $I_p = x_3$; so that $x_1 + x_2 + x_3 = N_h(t)$. Furthermore, let the vector $X = (x_1, x_2, x_3)^T$, then the model in system (3.20) can now be written in the form

$\frac{dX}{dt} = f(x)$, where $f = (f_1, f_2, f_3)^T$. It implies that system (3.20) can be written in term of the new variables as

$$\left.\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \Lambda_h - \beta_p c(H) x_3 x_1 - \mu_h x_1 + \tau x_2 + V_p x_2 + \tau x_3 \\
\frac{dx_2}{dt} &= f_2 = \beta_p c(H) x_3 x_1 - (k_p + \tau + V_p + \mu_h) x_2 \\
\frac{dx_3}{dt} &= f_3 = k_p x_2 - (\sigma_p + \tau + \mu_h) x_3
\end{aligned}\right\} \tag{3.26}$$

It can be shown that system (3.26) has a right and left eigenvectors associated with zero eigenvalue at $c(H) = c(H)^*$ given by

$$w = \left(\frac{\left(\mu_h(\tau + V_p)(\sigma_p + \tau + \mu_h) + k_p(\mu_h \tau - \beta_p c(H)^* \Lambda_h)\right) w_2}{\mu_h{}^2 (\sigma_p + \tau + \mu_h)}, w_2, \frac{k_p w_2}{(\sigma_p + \tau + \mu_h)} \right)^T \tag{3.27}$$

and

$$\bar{v} = (\bar{v}_1, \bar{v}_2, \bar{v}_3) = \left(0, \bar{v}_2, \frac{(k_p + \tau + V_p + \mu_h) \bar{v}_2}{k_p} \right) \tag{3.28}$$

where $w_2 > 0$ and $\bar{v}_2 > 0$ are free right eigenvector and left eigenvector.

Computation of the coefficient a and b

For system (3.26), the associated non-zero partial derivatives at P_0 can be shown to be

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_3} = \beta_p c(H)^* \tag{3.29}$$

From (3.29), it follows that

$$a = \frac{2\bar{v}_2 (k_p + \tau + V_p + \mu_h) w_2{}^2 a_1}{\Lambda_h (\sigma_p + \tau + \mu_h)} \quad , \qquad b = \bar{v}_2 w_3 \beta_p = \frac{\bar{v}_2 k_p \beta_p w_2}{(\sigma_p + \tau + \mu_h)} \tag{3.30}$$

where $a_1 = \mu_h(\tau + V_p)(\sigma_p + \tau + \mu_h) + k_p(\mu_h \tau - \Lambda_h \beta_p c(H)^*)$

Thus, (i) If $a_1 > 0$, a backward bifurcation occurs   (ii) If $a_1 < 0$, a forward bifurcation occurs.

The above analysis is summarized in the following results from theorem A.1 item (d):

Lemma 3.3: The pneumonia only model of (3.20) has a unique positive endemic equilibrium which is locally asymptotically stable (LAS) if $R_p > 1$ otherwise unstable.

3.5   Analysis of Malaria-Pneumonia Co-infection Disease Transmission Model

The full model on the co-infection of Malaria and Pneumonia disease transmission (2.3) is now considered. Firstly, the co-infection dynamics of the full model system (2.3) is reduced to an eight dimensional system by neglecting the susceptible classes for both human and vector populations respectively and also using the fact that

$S_h(t) = 1 - E_m(t) - I_m(t) - E_p(t) - I_p(t) - E_{mp}(t) - I_{mp}(t)$ and
$S_v(t) = 1 - E_v(t) - I_v(t)$. Thus,

$$
\left.
\begin{aligned}
E_m{}'(t) &= \alpha\beta(H)I_v\big(1 - E_m - I_m - E_p - I_p - E_{mp} - I_{mp}\big) - (k_m + \pi + \mu_h)E_m \\
I_m{}'(t) &= k_m E_m - \vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m - (\sigma_m + \pi + \mu_h)I_m \\
E_p{}'(t) &= \beta_p c(H)\big(I_p + kI_{mp}\big)\big(1 - E_m - I_m - E_p - I_p - E_{mp} - I_{mp}\big) - \big(k_p + \tau + V_p + \mu_h\big)E_p \\
I_p{}'(t) &= k_p E_p - \varepsilon\alpha\beta(H)I_v I_p - \big(\sigma_p + \tau + \mu_h\big)I_p \\
E_{mp}{}'(t) &= \varepsilon\alpha\beta(H)I_v I_p + \vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m - \big(k_{mp} + \phi + \mu_h\big)E_{mp} \\
I_{mp}{}'(t) &= k_{mp}E_{mp} - \big(\sigma_m + \sigma_p + \sigma_{mp} + \phi + \mu_h\big)I_{mp} \\
E_v{}'(t) &= \alpha\beta_v\big(I_m + \delta I_{mp}\big)(1 - E_v - I_v) - k_v E_v - \mu_v E_v \\
I_v{}'(t) &= k_v E_v - \mu_v I_v
\end{aligned}
\right\}
\tag{3.31}
$$

The disease-free equilibrium is given by
$$
P_{mpf} = \big(E_m{}^0, I_m{}^0, E_p{}^0, I_p{}^0, E_{mp}{}^0, I_{mp}{}^0, E_v{}^0, I_v{}^0\big) = (0,0,0,0,0,0,0,0)
\tag{3.32}
$$

It can be shown that the basic reproduction number, denoted by R_{mp} for the full malaria-pneumonia co-infection model (3.31) by using the next generation matrix approach is given by
$$
R_{mp} = max\{R_m, R_p\}
\tag{3.33}
$$
So that the following result follows from theorem 2 in [35]:

Theorem 3.5: The malaria-pneumonia free equilibrium $\boldsymbol{P_{mpf}}$ of system (3.31) is locally asymptotically stable if $R_m < 1$, $R_p < 1$ and unstable if $R_m > 1$ and $R_m > 1$.

3.5.1   Global Stability of Malaria-Pneumonia Free Equilibrium $\boldsymbol{P_{mpf}}$ Model

Theorem 3.6: The malaria-pneumonia free equilibrium $\boldsymbol{P_{mpf}}$ of system (3.31) is globally asymptotically stable if $R_p < 1$, $R_m < 1$, otherwise unstable.

Proof:

The rate of change of the exposed and infected components of system (3.31) can be written as

$$
\begin{pmatrix}
\dfrac{dE_m}{dt} \\[4pt]
\dfrac{dI_m}{dt} \\[4pt]
\dfrac{dE_p}{dt} \\[4pt]
\dfrac{dI_p}{dt} \\[4pt]
\dfrac{dE_{mp}}{dt} \\[4pt]
\dfrac{dI_{mp}}{dt} \\[4pt]
\dfrac{dE_v}{dt} \\[4pt]
\dfrac{dI_v}{dt}
\end{pmatrix}
= (F - V)
\begin{pmatrix}
E_m \\ I_m \\ E_p \\ I_p \\ E_{mp} \\ I_{mp} \\ E_v \\ I_v
\end{pmatrix}
- \left(1 - \frac{S_h}{N_h}\right)\left(1 - \frac{S_v}{N_v}\right)F
\begin{pmatrix}
E_m \\ I_m \\ E_p \\ I_p \\ E_{mp} \\ I_{mp} \\ E_v \\ I_v
\end{pmatrix}
\tag{3.39}
$$

where F and V are given by

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha\beta(H) \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_p c(H) & 0 & \beta_p k c(H) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha\beta_v & 0 & 0 & 0 & \alpha\beta_v\delta & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} D_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -k_m & D_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & D_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_p & D_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & D_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_{mp} & D_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & (k_v + \mu_v) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -k_v & \mu_v \end{pmatrix}$$

with $D_0 = (k_m + \pi + \mu_h)$, $D_1 = (\sigma_m + \pi + \mu_h)$, $D_2 = (k_p + \tau + V_p + \mu_h)$,
$D_3 = (\sigma_p + \tau + \mu_h)$, $D_4 = (k_{mp} + \phi + \mu_h)$, $D_5 = (\sigma_m + \sigma_p + \sigma_{mp} + \phi + \mu_h)$

Since at the disease free $E_m = I_m = E_p = I_p = E_{mp} = I_{mp} = E_v = I_v = 0 \to (0,0,0,0,0,0,0,0)$
with $S_h \le N_h$ and $S_v \le N_v$ as $t \to \infty$ in Γ. Thus,

$$\begin{pmatrix} \frac{dE_m}{dt} \\ \frac{dI_m}{dt} \\ \frac{dE_p}{dt} \\ \frac{dI_p}{dt} \\ \frac{dE_{mp}}{dt} \\ \frac{dI_{mp}}{dt} \\ \frac{dE_v}{dt} \\ \frac{dI_v}{dt} \end{pmatrix} \le (F - V) \begin{pmatrix} E_m \\ I_m \\ E_p \\ I_p \\ E_{mp} \\ I_{mp} \\ E_v \\ I_v \end{pmatrix} \tag{3.35}$$

According to [35], all eigenvalues of the matrix $(F - V)$ have negative real parts i.e.
$$|(F - V) - \lambda I| = 0 \tag{3.36}$$
Equation (3.41) simplifies to give

$(-D_4 - \lambda)(-D_5 - \lambda)[(-D_0 - \lambda)(-D_1 - \lambda)(-\mu_v - \lambda)(-(k_v + \mu_v) - \lambda) - \alpha^2\beta_v\beta(H)k_m k_v][(-D_2 - \lambda)(-D_3 - \lambda) - k_p\beta_p c(H)] = 0$
Clearly, $\lambda_1 = -D_4$, $\lambda_2 = -D_5$ and
$(-D_0 - \lambda)(-D_1 - \lambda)(-\mu_v - \lambda)(-(k_v + \mu_v) - \lambda) - \alpha^2\beta_v\beta_m k_m k_v = 0$

$$\lambda^4 + (D_0 + D_1 + \mu_v + (k_v + \mu_v))\lambda^3 + (D_0D_1 + D_0\mu_v + D_0(k_v + \mu_v) + D_1\mu_v + D_1(k_v + \mu_v) +$$
$$\mu_v(k_v + \mu_v))\lambda^2 + (D_0D_1\mu_v + D_0\mu_v(k_v + \mu_v) + D_0D_1(k_v + \mu_v))\lambda + D_0D_1\mu_v(k_v + \mu_v) -$$
$$\alpha^2\beta_v\beta(H)k_mk_v = 0 \qquad\qquad (3.37)$$

Equation (3.37) will have all its eigenvalues to be real and negative (Descartes Rule of signs) if $R_m < 1$ and $R_p < 1$. Therefore, all eigenvalues of the linearized differential inequality are negative if $R_p < 1$ and $R_m < 1$. Consequently, $(E_m, I_m, E_p, I_p, E_{mp}, I_{mp}, E_v, I_v) \to$ $(0,0,0,0,0,0,0,0)$ as $t \to \infty$. Evaluating system (3.31) at $E_m = I_m = E_p = I_p = E_{mp} = I_{mp} = E_v = I_v = 0$ gives $S_h \to 1, S_v \to 1$ for $R_p < 1$ and $R_m < 1$. Hence, the malaria-pneumonia disease free equilibrium $\boldsymbol{P_{mpf}}$ is globally asymptotically stable if $R_p < 1$ and $R_m < 1$. The result follows that the malaria-pneumonia disease free equilibrium $\boldsymbol{P_{mpf}}$ is unstable if $R_p > 1$ and $R_m > 1$.

3.5.2   Local Asymptotic Stability of Malaria-Pneumonia Endemic Equilibrium $\boldsymbol{P_{mpe}}$

We establish the stability of the endemic equilibrium of the malaria – pneumonia model (3.31), using the Centre Manifold theory. The following result follows.

Theorem 3.6: The full malaria – pneumonia co-infection model of (3.31) has a unique endemic equilibrium which is locally asymptotically stable if $R_{mp} > 1$ and unstable if $R_{mp} < 1$ whenever item (d) of theorem A.1 in the appendix is satisfied.

Proof:

To apply the Centre Manifold theory, the following change of variables is made. Let $E_m = x_1$, $I_m = x_2, E_p = x_3, I_p = x_4, E_{mp} = x_5, I_{mp} = x_6, E_v = x_7, I_v = x_8$

Furthermore, introducing the vector $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8)^T$,

and then the model in system (3.31) can now be written in the form $\frac{dX}{dt} = F(x)$, where $F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8)^T$. It implies that system (3.31) can be written in term of the new variable as follow:

$$\frac{dx_1}{dt} = f_1 = \alpha\beta(H)x_8(1 - x_1 - x_2 - x_3 - x_4 - x_5 - x_6) - (k_m + \pi + \mu_h)x_1$$

$$\frac{dx_2}{dt} = f_2 = k_mx_1 - \vartheta\beta_pc(H)(x_4 + kx_6)x_2 - (\sigma_m + \pi + \mu_h)x_2$$

$$\frac{dx_3}{dt} = f_3 = \beta_pc(H)(x_4 + kx_6)(1 - x_1 - x_2 - x_3 - x_4 - x_5 - x_6) - (k_p + \tau + V_p + \mu_h)x_3$$

$$\frac{dx_4}{dt} = f_4 = k_px_3 - \varepsilon\alpha\beta(H)x_8x_4 - (\sigma_p + \tau + \mu_h)x_4$$

$$\frac{dx_5}{dt} = f_5 = \varepsilon\alpha\beta(H)x_8x_4 + \vartheta\beta_pc(H)(x_4 + kx_6)x_2 - (k_{mp} + \phi + \mu_h)x_5$$

$$\frac{dx_6}{dt} = f_6 = k_{mp}x_5 - (\sigma_m + \sigma_p + \sigma_{mp} + \phi + \mu_h)x_6$$

$$\frac{dx_7}{dt} = f_7 = \alpha\beta_v(x_2 + \delta x_6)(1 - x_7 - x_8) - (k_v + \mu_v)x_7$$

$$\frac{dx_8}{dt} = f_8 = k_vx_7 - \mu_vx_8$$

(3.38)

It can be shown that system (3.39) has the right and left eigenvectors given by

$$w = \left(\frac{D_1}{k_m}w_2, w_2, 0,0,0,0, \frac{\alpha\beta_v}{(k_v + \mu_v)}w_2, \frac{\alpha\beta_vk_v}{\mu_v(k_v + \mu_v)}w_2\right)^T \qquad\qquad (3.39)$$

and

$$\bar{v} = \left(\frac{k_m}{D_0}\bar{v}_2, \bar{v}_2, 0,0, \frac{\alpha^2\beta_v\delta\beta(H)^*k_mk_vk_{mp}}{\mu_v(k_v + \mu_v)D_0D_4D_5}\bar{v}_2, \frac{\alpha^2\beta_v\delta\beta(H)^*k_mk_v}{\mu_v(k_v + \mu_v)D_0D_5}\bar{v}_2, \frac{\alpha\beta(H)^*k_mk_v}{\mu_v(k_v + \mu_v)D_0}\bar{v}_2, \frac{\alpha\beta(H)^*k_m}{\mu_vD_0}\bar{v}_2\right)$$

(3.40)

where $w_2 > 0$ and $\bar{v}_2 > 0$ are free right and left eigenvector.

Computation of the coefficient $\boldsymbol{a}$ and $\boldsymbol{b}$

For system (3.43), the associated non-zero partial derivatives of (3.38) at P_{mpf} can be shown to be

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_8} = \frac{\partial^2 f_1}{\partial x_2 \partial x_8} = -\alpha\beta(H)^*, \quad \frac{\partial^2 f_7}{\partial x_2 \partial x_7} = \frac{\partial^2 f_7}{\partial x_2 \partial x_8} = -\alpha\beta_v, \frac{\partial^2 f_1}{\partial x_8 \partial\beta(H)} = \alpha \qquad (3.41)$$

From (3.41), it follows that

$$a = -2\alpha(\bar{v}_1 w_1 w_8 \beta(H)^* + \bar{v}_1 w_2 w_8 \beta(H)^* + \bar{v}_7 w_2 w_7 \beta_v + \bar{v}_7 w_2 w_8 \beta_v) < 0 \qquad (3.42)$$
$$b = \bar{v}_1 w_8 \alpha > 0 \qquad (3.43)$$

3.6   The Effect of Sanitation on Disease Transmission Dynamics

The aim and ultimate goal of a public health worker is to change the transmission dynamics of a disease in such a way that if an infected individual enters into a community he/she will not trigger an epidemic in the community. Mathematically, it is reasonable to assume that if
$R_0 < 1$, then
$$\frac{dI}{dt} < 0 \qquad (3.44)$$

For malaria, intervention can be in the following ways: (i) Treatment using anti-malaria drugs (ii) Reducing the rate of contact between susceptible individuals and infected mosquitoes and vice-versa. The latter can be achieved through sleeping under the mosquito treated nets, clean environment (maintaining high level of sanitation), etc. The system of equations in (2.3) introduces the functions $\beta(H)$ and $c(H)$ to describe the effect of sanitation on the malaria and pneumonia transmission respectively.

The functions $\beta(H)$ and $c(H)$ in equations (2.4) and (2.5) predicts that malaria and pneumonia transmissions are reduced proportionally to the improvement of sanitation conditions. Using the functions defined in (2.4) and (2.5), a required level of sanitation to prevent the outbreak and low transmission of malaria and pneumonia respectively are established as follows:

If $R_m < 1$

$$\sqrt{\frac{\alpha^2 \beta_v \beta(H) k_m k_v \Lambda_h \Lambda_v}{\mu_h \mu_v{}^2 (k_v + \mu_v)(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)}} < 1$$

$$\beta(H) < \frac{\mu_h \mu_v{}^2 (k_v + \mu_v)(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)}{\alpha^2 \beta_v k_m k_v \Lambda_h \Lambda_v} \qquad (3.45)$$

$$\beta_{max} - \gamma H < \frac{\mu_h \mu_v{}^2 (k_v + \mu_v)(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)}{\alpha^2 \beta_v k_m k_v \Lambda_h \Lambda_v}$$

Thus, the level of sanitation required for malaria reduction / eradication is

$$H > \frac{1}{\gamma}\left[\beta_{max} - \frac{\mu_h \mu_v{}^2 (k_v + \mu_v)(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)}{\alpha^2 \beta_v k_m k_v \Lambda_h \Lambda_v}\right] \qquad (3.46)$$

Also, if $R_p < 1$

$$c(H) < \frac{(\sigma_p + \tau + \mu_h)(k_p + \tau + V_p + \mu_h)\Lambda_h}{\mu_h k_p \beta_p}$$

$$c_{max} - \gamma H < \frac{(\sigma_p + \tau + \mu_h)(k_p + \tau + V_p + \mu_h)\Lambda_h}{\mu_h k_p \beta_p}$$

Thus, the level of sanitation required pneumonia reduction / eradication is

$$H > \frac{1}{\gamma}\left[c_{max} - \frac{(\sigma_p + \tau + \mu_h)(k_p + \tau + V_p + \mu_h)\Lambda_h}{\mu_h k_p \beta_p}\right] \qquad (3.47)$$

4.0   Application of Optimal Control to the Co-infection of Malaria-Pneumonia

The main objective of this study is to suggest possible(s) optimal method of reducing/minimizing malaria and pneumonia transmission. Many mathematical model already exist describing malaria infection or pneumonia infection but the best control for malaria infection, pneumonia infection and the co-infection of both diseases still remain a subject of debate. Previous mathematical models have considered treatment, sleeping under mosquito treated bedside nets (MTBN) and the use of insecticide coil as controls. However, these have their limitations. The insecticides used for treating bedside nets is lethal to the mosquitoes, insects and also repels the mosquitoes, thus reducing the number of mosquitoes who attempt to feed on people in the sleeping areas with the nets [2]. However, the mosquitoes can still feed on humans outside these protective areas, hence; Agusto *et al* [2] included the spraying of insecticides in their model. The latter control by Agusto *et al* [2] will be more effective in a closed area, hence, the inclusion of sanitation as control in the present work.

The preventive and treatment control for pneumonia includes vaccination, environmental measures and appropriate treatment of other health problems [32]. It is believed that, if appropriate preventive measures were instituted globally, mortality among children could be reduced by 400,000; and, if proper treatment were universally available, childhood deaths could be decreased by another 600,000 [37].

4.1 Formulation of Optimal Control Model for the Co-infection of Malaria-Pneumonia with Mass Action Incidence

We now introduce into system (2.3) time dependent preventive measures $\big(u_1(t), u_2(t), u_3(t), u_4(t)\big)$ and treatment efforts $\big(u_5(t), u_6(t), u_7(t)\big)$ as controls to curtail the spread of malaria and pneumonia infection. Thus, system (2.3) becomes

$$
\left.
\begin{aligned}
S_h'(t) &= \Lambda_h - (1 - u_1 - u_2 - u_3)\alpha\beta(H)I_v S_h \\
&\quad - (1 - u_3 - u_4)\beta_p c(H)\big(I_p + kI_{mp}\big)S_h - \mu_h S_h \\
&\quad + u_5 I_m + u_5 E_m + u_6 I_p + u_6 E_p + u_4 E_p + u_7 I_{mp} + u_7 E_{mp} \\
E_m'(t) &= (1 - u_1 - u_2 - u_3)\alpha\beta(H)I_v S_h - (k_m + u_5 + \mu_h)E_m \\
I_m'(t) &= k_m E_m - (1 - u_4)\vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m - (\sigma_m + u_5 + \mu_h)I_m \\
E_p'(t) &= (1 - u_3 - u_4)\beta_p c(H)\big(I_p + kI_{mp}\big)S_h - \big(k_p + u_4 + u_6 + \mu_h\big)E_p \\
I_p'(t) &= k_p E_p - (1 - u_1 - u_2 - u_3)\varepsilon\alpha\beta(H)I_v I_p - \big(\sigma_p + u_6 + \mu_h\big)I_p \\
E_{mp}'(t) &= (1 - u_1 - u_2 - u_3)\varepsilon\alpha\beta(H)I_v I_p + (1 - u_4)\vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m \\
&\quad - \big(k_{mp} + u_7 + \mu_h\big)E_{mp} \\
I_{mp}'(t) &= k_{mp}E_{mp} - \big(\sigma_m + \sigma_p + \sigma_{mp} + u_7 + \mu_h\big)I_{mp} \\
S_v'(t) &= \Lambda_v - (1 - u_1 - u_2 - u_3)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v \\
&\quad - (u_1 + u_2 + u_3 + \mu_v)S_v \\
E_v'(t) &= (1 - u_1 - u_2 - u_3)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v - (k_v + u_1 + u_2 + u_3 + \mu_v)E_v \\
I_v'(t) &= k_v E_v - (u_1 + u_2 + u_3 + \mu_v)I_v
\end{aligned}
\right\}
\quad (3.48)
$$

where

$u_1(t)$: is the time preventive control using mosquito treated bedside nets (MTBN) for malaria

$u_2(t)$: is the time preventive control using insecticides spray on mosquitoes

$u_3(t)$: is the time preventive control through sanitation for malaria and pneumonia control

$u_4(t)$: is the time preventive control using vaccine for pneumonia control

$u_5(t)$: is the treatment effort using anti-malaria drugs for malaria

$u_6(t)$: is the treatment effort using anti-pneumonia drugs for pneumonia

$u_7(t)$: is the treatment effort using both anti-malaria and anti-pneumonia drugs

The following cases are considered:

Case 1: The optimal control strategy for malaria transmission model

Case 2: The optimal control strategy for pneumonia transmission model

Case 3: The optimal control strategy for the co-infection of malaria and pneumonia disease transmission model

4.2   The Optimal Control Strategy for Malaria Transmission Model Case 1

Here, the formulation of the optimal control problem, analysis of the optimal control problem, adjoint conditions, optimality conditions and the optimality system for the malaria model are considered. Let $I_p(t) = I_{mp}(t) = E_p(t) = E_{mp}(t) = 0$ in model system (3.48) gives the following system of equations

$$S_h'(t) = \Lambda_h - (1 - u_1 - u_2 - u_3)\alpha\beta(H)I_vS_h - \mu_hS_h + u_5I_m + u_5E_m$$
$$E_m'(t) = (1 - u_1 - u_2 - u_3)\alpha\beta(H)I_vS_h - (k_m + u_5 + \mu_h)E_m$$
$$I_m'(t) = k_mE_m - (\sigma_m + u_5 + \mu_h)I_m$$
$$S_v'(t) = \Lambda_v - (1 - u_1 - u_2 - u_3)\alpha\beta_vI_mS_v - (u_1 + u_2 + u_3 + \mu_v)S_v$$
$$E_v'(t) = (1 - u_1 - u_2 - u_3)\alpha\beta_vI_mS_v - (k_v + u_1 + u_2 + u_3 + \mu_v)E_v$$
$$I_v'(t) = k_vE_v - (u_1 + u_2 + u_3 + \mu_v)I_v \qquad (3.49)$$

The controls u in (3.49) is defined to be $u \in [0,1]$, where u ranges from no control ($u = 0$) to maximum control ($u = 1$). Note that $u_1, u_2, u_3, u_5 \in u$.

The main objective of this research is to find the optimal control strategy u throughout the length of $0 \le t \le t_f$ such that the numbers of infected humans I_m and infected vectors are minimized while minimizing the cost of control u. Thus, the objective function is

$$J(u_1, u_2, u_3, u_5) = \int_0^{t_f}(MI_m + NI_v + m_1u_1{}^2 + m_2u_2{}^2 + m_3u_3{}^2 + m_5u_5{}^2)dt \qquad (3.50)$$

where coefficients M, m_1, m_2, m_3 and m_5 are positive weights to balance the factors. Thus, we seek an optimal control $u^* = \{u_1^*, u_2^*, u_3^*, u_5^*\}$ such that

$$J(u_1^*, u_2^*, u_3^*, u_5^*) = \min_{u_1, u_2, u_3, u_5}\{J(u_1, u_2, u_3, u_5)|_{u_1, u_2, u_3, u_5} \in u\} \qquad (3.51)$$

where

$$u = \{(u_1, u_2, u_3, u_5)|_{u_1, u_2, u_3, u_5}: [0, t_f] \to [0,1]\} \qquad (3.52)$$

is Lebesgue measurable and convex on u, then there exist an optimal control u satisfying the conditions in Appendix A.2

Since there exist an optimal control for minimizing the functional (3.50) subject to system of equations (3.49), the Pontryagins' Maximum Principle [16] is used to derive necessary conditions for this optimal control. The Hamiltonian is defined as follows:

$$\bar{H} = MI_m + NI_v + m_1u_1{}^2 + m_2u_2{}^2 + m_3u_3{}^2 + m_5u_5{}^2 + \lambda_1[\Lambda_h - (1 - u_1 - u_2 - u_3)\alpha\beta(H)I_vS_h - \mu_hS_h + u_5I_m + u_5E_m] + \lambda_2[(1 - u_1 - u_2 - u_3)\alpha\beta(H)I_vS_h - (k_m + u_5 + \mu_h)E_m] + \lambda_3[k_mE_m - (\sigma_m + u_5 + \mu_h)I_m] + \lambda_4[\Lambda_v - (1 - u_1 - u_2 - u_3)\alpha\beta_vI_mS_v - (u_1 + u_2 + u_3 + \mu_v)S_v] + \lambda_5[(1 - u_1 - u_2 - u_3)\alpha\beta_vI_mS_v - (k_v + u_1 + u_2 + u_3 + \mu_v)E_v] + \lambda_6[k_vE_v - (u_1 + u_2 + u_3 + \mu_v)I_v] \qquad (3.53)$$

where $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5$ and λ_6 are the adjoint variables or co-state variables.

In order to attach the system of ordinary differential equation in (3.49) on to the objective function in (3.50), the adjoint functions (or co-state variables) were used. The Pontryagins' Maximum Principle gives the necessary conditions that the adjoint functions must satisfy. Thus, the differential equations satisfied by system (3.49) are:

$$\frac{d\lambda_1}{dt} = -\frac{\partial\bar{H}}{\partial S_h} = (\lambda_1 - \lambda_2)(1 - u_1 - u_2 - u_3)\alpha\beta(H)I_v + \lambda_1\mu_h$$

$$\frac{d\lambda_2}{dt} = -\frac{\partial\bar{H}}{\partial E_m} = \lambda_2(k_m + u_5 + \mu_h) - \lambda_1u_5 - \lambda_3k_m$$

$$\frac{d\lambda_3}{dt} = -\frac{\partial \bar{H}}{\partial I_m} = (\lambda_4 - \lambda_5)(1 - u_1 - u_2 - u_3)\alpha\beta_v S_v + \lambda_3(\sigma_m + u_5 + \mu_h) - \lambda_1 u_5 - M$$

$$\frac{d\lambda_4}{dt} = -\frac{\partial \bar{H}}{\partial S_v} = (\lambda_4 - \lambda_5)(1 - u_1 - u_2 - u_3)\alpha\beta_v I_m + \lambda_4(u_1 + u_2 + u_3 + \mu_v)$$

$$\frac{d\lambda_5}{dt} = -\frac{\partial \bar{H}}{\partial E_v} = \lambda_5(k_v + u_1 + u_2 + u_3 + \mu_v) - \lambda_6 k_v$$

$$\frac{d\lambda_6}{dt} = -\frac{\partial \bar{H}}{\partial I_v} = (\lambda_1 - \lambda_2)(1 - u_1 - u_2 - u_3)\alpha\beta(H)S_h + \lambda_6(u_1 + u_2 + u_3 + \mu_v) - N \quad (3.54)$$

with the boundary conditions (or Transversality conditions) at the final time, t_f:

$$\lambda_1(t_f) = 0, \ \lambda_2(t_f) = 0, \ \lambda_3(t_f) = 0, \ \lambda_4(t_f) = 0, \ \lambda_5(t_f) = 0, \lambda_6(t_f) = 0 \qquad (3.55)$$

The Hamiltonian in (3.53) is minimized with respect to the controls u_1, u_2, u_3 and u_5 separately in order to obtain the optimal value of $u_1{}^*, u_2{}^*, u_3{}^*, u_5{}^*$. At these controls values, the maximum Hamiltonian is obtained. The derivative of the Hamiltonian with respect to u_1, u_2, u_3 and u_5 is thus zero, since at the absolute minimum or maximum the slope of a function is zero. Thus,

$$u_1 = \frac{(\lambda_2 - \lambda_1)\alpha\beta(H)I_v S_h + (\lambda_5 - \lambda_4)\alpha\beta_v I_m S_v + \lambda_5 E_v + \lambda_6 I_v + \lambda_4 S_v}{2m_1} \qquad (3.56)$$

$$u_2 = \frac{(\lambda_2 - \lambda_1)\alpha\beta(H)I_v S_h + (\lambda_5 - \lambda_4)\alpha\beta_v I_m S_v + \lambda_5 E_v + \lambda_6 I_v + \lambda_4 S_v}{2m_2} \qquad (3.57)$$

$$u_3 = \frac{(\lambda_2 - \lambda_1)\alpha\beta(H)I_v S_h + (\lambda_5 - \lambda_4)\alpha\beta_v I_m S_v + \lambda_5 E_v + \lambda_6 I_v + \lambda_4 S_v}{2m_3} \qquad (3.58)$$

$$u_5 = \frac{(\lambda_3 - \lambda_1)I_m + (\lambda_2 - \lambda_1)E_m}{2m_5} \qquad (3.59)$$

At the absolute minimum $u = u^*$, therefore the optimality conditions are

$$\left.\begin{aligned}
u_1{}^* &= min\{1, max(0, u_1)\} \\
u_2{}^* &= min\{1, max(0, u_2)\} \\
u_3{}^* &= min\{1, max(0, u_3)\} \\
u_5{}^* &= min\{1, max(0, u_5)\}
\end{aligned}\right\} \qquad (3.60)$$

4.3   The Optimal Control Strategy for Pneumonia Transmission Model Case 2

If $E_m(t) = I_m(t) = E_{mp}(t) = I_{mp}(t) = E_v(t) = I_v(t) = 0$ in model system (3.48) gives the following system of equations

$$\left.\begin{aligned}
S_h{}'(t) &= \Lambda_h - (1 - u_3 - u_4)\beta_p c(H)I_p S_h - \mu_h S_h + u_6 E_p + u_6 I_p + u_4 E_p \\
E_p{}'(t) &= (1 - u_3 - u_4)\beta_p c(H)I_p S_h - (k_p + u_6 + u_4 + \mu_h)E_p \\
I_p{}'(t) &= k_p E_p - (\sigma_p + u_6 + \mu_h)I_p \\
S_v{}'(t) &= \Lambda_v - (u_1 + u_2 + u_3 + \mu_v)S_v
\end{aligned}\right\} \qquad (3.61)$$

Observe that the fourth differential equation in (3.61) is independent of the first three differential equations; hence, system (3.67) can then be written as

$$\left.\begin{aligned}
S_h{}'(t) &= \Lambda_h - (1 - u_3 - u_4)\beta_p c(H)I_p S_h - \mu_h S_h + u_6 E_p + u_6 I_p + u_4 E_p \\
E_p{}'(t) &= (1 - u_3 - u_4)\beta_p c(H)I_p S_h - (k_p + u_6 + u_4 + \mu_h)E_p \\
I_p{}'(t) &= k_p E_p - (\sigma_p + u_6 + \mu_h)I_p
\end{aligned}\right\} \qquad (3.62)$$

where the controls u_3, u_4 and u_6 retain their original meaning as defined in system (3.48) The controls u in (3.62) is defined to be $u \in [0,1]$, for $u_3, u_4, u_6 \in u$

The objective is such that the numbers of infected humans with pneumonia I_p is minimized while minimizing the cost of control u. Hence

$$J(u_3, u_4, u_6) = \int_0^{tf}\left(DI_p + m_3 u_3{}^2 + m_4 u_4{}^2 + m_6 u_6{}^2\right)dt \tag{3.63}$$

where coefficients D, m_3, m_4 and m_6 are positive weights to balance the factors.

Thus, we seek an optimal control $u^* = \{u_3{}^*, u_4{}^*, u_6{}^*\}$ such that

$$J(u_3{}^*, u_4{}^*, u_6{}^*) = \min_{u_2,u_4,u_6}\left\{J(u_2, u_4, u_6)|_{u_2,u_4,u_6} \in u\right\} \tag{3.64}$$

where

$$u = \left\{(u_2, u_4, u_6)|_{u_2,u_4,u_6}: [0, t_f] \to [0,1]\right\} \tag{3.65}$$

is Lebesgue measurable and convex on u, then there exist an optimal control u satisfying the conditions in Appendix A.2. We define an Hamiltonian as follows:

$$\bar{H} = DI_p + m_3 u_3{}^2 + m_4 u_4{}^2 + m_6 u_6{}^2 + \lambda_1\big[\Lambda_h - (1 - u_3 - u_4)\,\beta_p c(H)I_p S_h - \mu_h S_h + u_6 E_p + u_6 I_p + u_4 E_p\big] + \lambda_2\big[(1 - u_3 - u_4)\,\beta_p c(H)I_p S_h - (k_p + u_6 + u_4 + \mu_h)E_p\big] + \lambda_3\big[k_p E_p - (\sigma_p + u_6 + \mu_h)I_p\big] \tag{3.66}$$

with $\lambda_1, \lambda_2, \lambda_3$ defined as the adjoint variables or co-state variables and obtained as

$$\left.\begin{aligned}
\frac{d\lambda_1}{dt} &= -\frac{\partial \bar{H}}{\partial S_h} = (\lambda_1 - \lambda_2)(1 - u_3 - u_4)\beta_p c(H)I_p + \lambda_1 \mu_h \\[2mm]
\frac{d\lambda_2}{dt} &= -\frac{\partial \bar{H}}{\partial E_p} = \lambda_2(k_p + u_4 + u_6 + \mu_h) - (u_4 + u_6)\lambda_1 - \lambda_3 k_p \\[2mm]
\frac{d\lambda_3}{dt} &= -\frac{\partial \bar{H}}{\partial I_p} = (\lambda_1 - \lambda_2)(1 - u_3 - u_4)\beta_p c(H)S_h + \lambda_3(\sigma_p + u_6 + \mu_h) - D
\end{aligned}\right\} \tag{3.67}$$

with the boundary conditions (or Transversality conditions) at the final time, t_f:

$$\lambda_1(t_f) = 0,\ \lambda_2(t_f) = 0,\ \lambda_3(t_f) = 0 \tag{3.68}$$

The Hamiltonian in (3.72) is minimized with respect to the controls u_3, u_4 and u_6 separately in order to obtain the optimal value of $u_3{}^*, u_4{}^*, u_6{}^*$. Thus,

$$u_3 = \frac{(\lambda_2 - \lambda_1)\beta_p c(H)I_p S_h}{2m_3} \tag{3.69}$$

$$u_4 = \frac{(\lambda_2 - \lambda_1)\beta_p c(H)I_p S_h + (\lambda_2 - \lambda_1)E_p}{2m_4} \tag{3.70}$$

$$u_6 = \frac{(\lambda_2 - \lambda_1)E_p + (\lambda_3 - \lambda_1)I_p}{2m_6} \tag{3.71}$$

The optimality conditions in this case are

$$\left.\begin{aligned}
u_3{}^* &= min\{1, max(0, u_3)\} \\
u_4{}^* &= min\{1, max(0, u_4)\} \\
u_6{}^* &= min\{1, max(0, u_6)\}
\end{aligned}\right\} \tag{3.72}$$

4.4 The Optimal Control Model for the Co-infection of Malaria-Pneumonia with Mass Action Incidence Model Case 3

Recall the system of equations in (3.48) written below as

$$
\left.
\begin{aligned}
S_h{}'(t) &= \Lambda_h - (1 - u_1 - u_2 - u_3)\alpha\beta(H)I_v S_h \\
&\quad - (1 - u_3 - u_4)\beta_p c(H)\big(I_p + kI_{mp}\big)S_h - \mu_h S_h \\
&\quad + u_5 I_m + u_5 E_m + u_6 I_p + u_6 E_p + u_4 E_p + u_7 I_{mp} + u_7 E_{mp} \\
E_m{}'(t) &= (1 - u_1 - u_2 - u_3)\alpha\beta(H)I_v S_h - (k_m + u_5 + \mu_h)E_m \\
I_m{}'(t) &= k_m E_m - (1 - u_4)\vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m - (\sigma_m + u_5 + \mu_h)I_m \\
E_p{}'(t) &= (1 - u_3 - u_4)\beta_p c(H)\big(I_p + kI_{mp}\big)S_h - (k_p + u_4 + u_6 + \mu_h)E_p \\
I_p{}'(t) &= k_p E_p - (1 - u_1 - u_2 - u_3)\varepsilon\alpha\beta(H)I_v I_p - (\sigma_p + u_6 + \mu_h)I_p \\
E_{mp}{}'(t) &= (1 - u_1 - u_2 - u_3)\varepsilon\alpha\beta(H)I_v I_p + (1 - u_4)\vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m \\
&\quad - (k_{mp} + u_7 + \mu_h)E_{mp} \\
I_{mp}{}'(t) &= k_{mp}E_{mp} - (\sigma_m + \sigma_p + \sigma_{mp} + u_7 + \mu_h)I_{mp} \\
S_v{}'(t) &= \Lambda_v - (1 - u_1 - u_2 - u_3)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v \\
&\quad - (u_1 + u_2 + u_3 + \mu_v)S_v \\
E_v{}'(t) &= (1 - u_1 - u_2 - u_3)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v - (k_v + u_1 + u_2 + u_3 + \mu_v)E_v \\
I_v{}'(t) &= k_v E_v - (u_1 + u_2 + u_3 + \mu_v)I_v
\end{aligned}
\right\} \quad (3.73)
$$

where the controls $u_1, u_2, u_3, u_4, u_5, u_6$ and u_7 retain their original meaning as defined in system (3.48). The objective functional is

$$
J(u_1, u_2, u_3, u_5) = \int_0^{tf}\big(MI_m + DI_p + QI_{mp} + NI_v + m_1 u_1{}^2 + m_2 u_2{}^2 + m_3 u_3{}^2 + m_4 u_4{}^2 + m_5 u_5{}^2 + m_6 u_6{}^2 + m_7 u_7{}^2\big)dt \quad (3.74)
$$

where the coefficients $D, M, N, Q, m_1, m_2, m_3, m_4, m_5, m_6$ and m_7 are positive weights to balance the factors. Thus, an optimal control $u^* = \{u_1{}^*, u_2{}^*, u_3{}^*, u_4{}^*, u_5{}^*, u_6{}^*, u_7{}^*\}$ is sought such that

$$
J(u_1{}^*, u_2{}^*, u_3{}^*, u_4{}^*, u_5{}^*, u_6{}^*, u_7{}^*) = \min_{u_1, u_2, u_3, u_5}\big\{J(u_1, u_2, u_3, u_4, u_5, u_6, u_7)|_{u_1, u_2, u_3, u_5} \in u\big\}
$$

where

$$
u = \big\{(u_1, u_2, u_3, u_4, u_5, u_6, u_7)|_{u_1, u_2, u_3, u_4, u_5, u_6, u_7} : [0, t_f] \rightarrow [0,1]\big\} \quad (3.75)
$$

is Lebesgue measurable and convex on u, then there exist an optimal control u satisfying the conditions in section 2.7. The Hamiltonian is defined as

$$
\begin{aligned}
\bar{H} &= MI_m + DI_p + QI_{mp} + NI_v + m_1 u_1{}^2 + m_2 u_2{}^2 + m_3 u_3{}^2 + m_4 u_4{}^2 + m_5 u_5{}^2 + m_6 u_6{}^2 \\
&\quad + m_7 u_7{}^2 + \lambda_1\big[\Lambda_h - (1 - u_1 - u_2 - u_3)\alpha\beta(H)I_v S_h - (1 - u_3 - u_4)\beta_p c(H)\big(I_p + kI_{mp}\big)S_h \\
&\quad - \mu_h S_h + u_5 I_m + u_5 E_m + u_6 I_p + u_6 E_p + u_4 E_p + u_7 I_{mp} + u_7 E_{mp}\big] + \lambda_2[(1 - u_1 - u_2 \\
&\quad - u_3)\alpha\beta(H)I_v S_h - (k_m + u_5 + \mu_h)E_m] + \lambda_3\big[k_m E_m - (1 - u_4)\vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m \\
&\quad - (\sigma_m + u_5 + \mu_h)I_m\big] + \lambda_4\big[(1 - u_3 - u_4)\beta_p c(H)\big(I_p + kI_{mp}\big)S_h - (k_p + u_4 + u_6 + \mu_h)E_p\big] \\
&\quad + \lambda_5\big[k_p E_p - (1 - u_1 - u_2 - u_3)\varepsilon\alpha\beta(H)I_v I_p - (\sigma_p + u_6 + \mu_h)I_p\big] + \lambda_6\big[(1 - u_1 - u_2 \\
&\quad - u_3)\varepsilon\alpha\beta(H)I_v I_p + (1 - u_4)\vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m - (k_{mp} + u_7 + \mu_h)E_{mp}\big] + \lambda_7\big[k_{mp}E_{mp} \\
&\quad - (\sigma_m + \sigma_p + \sigma_{mp} + u_7 + \mu_h)I_{mp}\big] + \lambda_8\big[\Lambda_v - (1 - u_1 - u_2 - u_3)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v \\
&\quad - (u_1 + u_2 + u_3 + \mu_v)S_v\big] + \lambda_9\big[(1 - u_1 - u_2 - u_3)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v - (k_v + u_1 + u_2 + u_3 + \\
&\quad \mu_v)E_v\big] + \lambda_{10}[k_v E_v - (u_1 + u_2 + u_3 + \mu_v)I_v] \qquad (3.76)
\end{aligned}
$$

with the following adjoint variables (or co-state variables) satisfying the Pontryagin Maximum Principle condition:

$$\frac{d\lambda_1}{dt} = -\frac{\partial \bar{H}}{\partial S_h} = (\lambda_1 - \lambda_2)(1 - u_1 - u_2 - u_3)\alpha\beta(H)I_v + (\lambda_1 - \lambda_4)(1 - u_3 - u_4)\beta_p c(H)\big(I_p + kI_{mp}\big) + \lambda_1\mu_h$$

$$\frac{d\lambda_2}{dt} = -\frac{\partial \bar{H}}{\partial E_m} = \lambda_2\big(k_m + u_5 + \mu_h\big) - \lambda_1 u_5 - \lambda_3 k_m$$

$$\frac{d\lambda_3}{dt} = -\frac{\partial \bar{H}}{\partial I_m} = (\lambda_3 - \lambda_6)(1 - u_4)\vartheta\beta_p c(H)\big(I_p + kI_{mp}\big) + (\lambda_8 - \lambda_9)(1 - u_1 - u_2 - u_3)\alpha\beta_v S_v$$
$$+ \lambda_3\big(\sigma_m + u_5 + \mu_h\big) - \lambda_1 u_5 - M$$

$$\frac{d\lambda_4}{dt} = -\frac{\partial \bar{H}}{\partial E_p} = \lambda_4\big(k_p + u_4 + u_6 + \mu_h\big) - \lambda_1(u_4 + u_6) - \lambda_5 k_p$$

$$\frac{d\lambda_5}{dt} = -\frac{\partial \bar{H}}{\partial I_p} = (\lambda_3 - \lambda_6)(1 - u_4)\vartheta\beta_p c(H)I_m + (\lambda_5 - \lambda_6)(1 - u_1 - u_2 - u_3)\varepsilon\alpha\beta(H)I_v$$
$$+ (\lambda_1 - \lambda_4)(1 - u_3 - u_4)\beta_p c(H)S_h + \lambda_3\big(\sigma_p + u_6 + \mu_h\big) - \lambda_1 u_6 - D$$

$$\frac{d\lambda_6}{dt} = -\frac{\partial \bar{H}}{\partial E_{mp}} = \lambda_6\big(k_{mp} + u_7 + \mu_h\big) - \lambda_1 u_7 - \lambda_7 k_{mp}$$

$$\frac{d\lambda_7}{dt} = -\frac{\partial \bar{H}}{\partial I_{mp}} = (\lambda_1 - \lambda_4)(1 - u_3 - u_4)\beta_p c(H)kS_h + (\lambda_3 - \lambda_6)(1 - u_4)\vartheta\beta_p kc(H)I_m$$
$$+ (\lambda_8 - \lambda_9)(1 - u_1 - u_2 - u_3)\alpha\beta_v\delta S_v + \lambda_7\big(\sigma_m + \sigma_p + \sigma_{mp} + u_7 + \mu_h\big) - \lambda_1 u_7 - Q$$

$$\frac{d\lambda_8}{dt} = -\frac{\partial \bar{H}}{\partial S_v} = (\lambda_8 - \lambda_9)(1 - u_1 - u_2 - u_3)\alpha\beta_v\big(I_m + \delta I_{mp}\big) + \lambda_8\big(u_1 + u_2 + u_3 + \mu_v\big)$$

$$\frac{d\lambda_9}{dt} = -\frac{\partial \bar{H}}{\partial E_v} = \lambda_9\big(k_v + u_1 + u_2 + u_3 + \mu_v\big) - \lambda_{10} k_v$$

$$\frac{d\lambda_{10}}{dt} = -\frac{\partial \bar{H}}{\partial I_v} = (\lambda_1 - \lambda_2)(1 - u_1 - u_2 - u_3)\alpha\beta(H)S_h + (\lambda_5 - \lambda_6)(1 - u_1 - u_2 - u_3)\varepsilon\beta(H)I_p$$
$$+ \lambda_{10}\big(u_1 + u_2 + u_3 + \mu_v\big) - N$$

$$(3.77)$$

with boundary conditions
$$\lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = \lambda_4(t_f) = \lambda_5(t_f) = \lambda_6(t_f) = \lambda_7(t_f) = \lambda_8(t_f) = \lambda_9(t_f) = \lambda_{10}(t_f) = 0$$

The derivative of the Hamiltonian with respect to $u_1, u_2, u_3, u_4, u_5, u_6$ and u_7 is thus zero, since at the absolute minimum or maximum the slope of a function is zero. Then

$$u_1 = \frac{(\lambda_2 - \lambda_1)\alpha\beta(H)I_v S_h + (\lambda_6 - \lambda_5)\varepsilon\alpha\beta(H)I_v I_p + (\lambda_9 - \lambda_8)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v + \lambda_8 S_v + \lambda_9 E_v + \lambda_{10} I_v}{2m_1}$$

$$u_2 = \frac{(\lambda_2 - \lambda_1)\alpha\beta(H)I_v S_h + (\lambda_6 - \lambda_5)\varepsilon\alpha\beta(H)I_v I_p + (\lambda_9 - \lambda_8)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v + \lambda_8 S_v + \lambda_9 E_v + \lambda_{10} I_v}{2m_2}$$

$$u_3 = \frac{(\lambda_2 - \lambda_1)\alpha\beta(H)I_v S_h + (\lambda_4 - \lambda_1)\beta_p C(H)\big(I_p + kI_{mp}\big)S_h + (\lambda_6 - \lambda_5)\varepsilon\alpha\beta(H)I_v I_p + (\lambda_9 - \lambda_8)\alpha\beta_v\big(I_m + \delta I_{mp}\big)S_v + \lambda_8 S_v + \lambda_9 E_v + \lambda_{10} I_v}{2m_3}$$

$$u_4 = \frac{(\lambda_4 - \lambda_1)\beta_p c(H)\big(I_p + kI_{mp}\big)S_h + (\lambda_4 - \lambda_1)E_p + (\lambda_6 - \lambda_3)\vartheta\beta_p c(H)\big(I_p + kI_{mp}\big)I_m}{2m_4}$$

$$u_5 = \frac{(\lambda_3 - \lambda_1)I_m + (\lambda_2 - \lambda_1)E_m}{2m_5}$$

$$u_6 = \frac{(\lambda_5 - \lambda_1)I_p + (\lambda_4 - \lambda_1)E_p}{2m_6}$$

$$u_7 = \frac{(\lambda_7 - \lambda_1)I_{mp} + (\lambda_6 - \lambda_1)E_{mp}}{2m_7}$$

Therefore the optimality conditions are

$$
\left.
\begin{aligned}
u_1{}^* &= min\{1, max(0, \ u_1)\} \\
u_2{}^* &= min\{1, max(0, \ u_2)\} \\
u_3{}^* &= min\{1, max(0, \ u_3)\} \\
u_4{}^* &= min\{1, max(0, \ u_4)\} \\
u_5{}^* &= min\{1, max(0, \ u_5)\} \\
u_6{}^* &= min\{1, max(0, \ u_6)\} \\
u_7{}^* &= min\{1, max(0, \ u_7)\}
\end{aligned}
\right\}
\qquad (3.78)
$$

5.0   Numerical Results and Discussion

In this section, we study numerically for the purpose of illustration the malaria only model, pneumonia only model, the malaria-pneumonia co-infection model and their corresponding optimal control models. The numerical computations were performed using MAPEL 18 program with computation times of 5.0s on a windows 7 operating system core i5. The optimal control was obtained by solving the optimality systems for malaria only, pneumonia only and malaria-pneumonia co-infection models respectively. An iterative forward and backward finite difference scheme are used to solve the optimality system; the forward finite difference was used to solve the state equations and the backward finite difference scheme was used to solve the co-state (adjoint) equations using the current iterations solutions of the state equations because of the transversality conditions. Then the controls are updated by using a convex combination of the previous controls and the value from the characterization (3.60), (3.72) and (3.78). Thus, the process is repeated and the iterations are stopped at the final time t_f .

The analytical results of the study are illustrated by simulating the model systems using values from other literature. The scarcity of data on the malaria-pneumonia co-infection limits our ability to scale our analytical results, however, for the purpose of illustration, other parameter values are assumed to vary within realistic means as given in Table 1

Table 1 Values of Parameters for the Malaria, Pneumonia and Co-infection Model

Parameters	Value	Sources
k_m	$\frac{1}{17}$	[4]
β_{max}	0.003	Assumed
γ	0.009	Assumed
β_v	0.001	[5]
μ_h	0.0000457	[39]
μ_v	0.04	[13]
k_v	$\frac{1}{18}$	[33]
Λ_h	0.00011	[40]
α	0.5	[5]
σ_m	0.01	Assumed
Λ_v	0.071	[4]
π	0.5	Assumed
k_p	0.5	Assumed
c_{max}	0.005	Assumed
β_p	0.5	Assumed

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V_p	0.5	Assumed
τ	0.5	Assumed
ϑ	0.1	Assumed
ε	0.1	Assumed
δ	0.5	Assumed
σ_{mp}	0.1	Assumed
σ_p	0.01	Assumed
k	0.5	Assumed
k_{mp}	0.5	Assumed

It was observed that as the biting rate, $\boldsymbol{\alpha}$ of the infected mosquitoes increase, there is a rapid decline in the number of susceptible human population as shown in **Figure 1**. This is expected from the result of the sensitivity analysis on the malaria model in case 1 that the biting rates of the mosquitoes, $\boldsymbol{\alpha}$, is the most sensitive of all the parameters in the malaria model being considered. This explains the rapid reduction in the number of susceptible individuals in the population as the rate of mosquito biting rates $\boldsymbol{\alpha}$ increases. This result is also in perfect agreement with the result obtained in Tumwiine *et al* [34] that reduction in contact between infected mosquitoes and humans was necessary in bringing the basic reproduction number below unity. The effect of treatment rate π using anti-malaria drugs on infected individual with malaria were also considered and it can be seen from **Figure 2** that as treatment rate π of malaria infected individuals increases, the proportion of infected humans decreases because treated individual leave the infected class and move to the susceptible class upon recovery from the malaria disease, a result which is in agreement with those obtained in [34] that there was need for effective drugs to reduce the spread of malaria disease.

Figure 3 is the backward bifurcation diagram for the malaria model where an endemic equilibrium co-exist (dotted lines) with the disease-free equilibrium. The backward bifurcation phenomenon explains that for total malaria eradication in the population $\boldsymbol{R_m < 1}$ is not sufficient rather $\boldsymbol{R_m}$ must be brought below certain threshold $\boldsymbol{R_c}$ such that $\boldsymbol{R_m < R_c < 1}$. $\boldsymbol{R_m}$ is defined here as the basic reproduction number for malaria. The impact of backward bifurcation on malaria transmission is to ensure that proper diagnosis are carried out on patients rather than treating patients based on symptoms and individuals who had being diagnosed to have malaria should go through complete treatment. From **Figure 4**, it was observed that if the contact rate $c = 0$ between susceptible and an infected individual with pneumonia, it was noticed that the number of susceptible human population increases considerably, however, as the rate of contact c increases, there was a sharp decline in the number of susceptible human population into the exposed human to pneumonia and subsequently progress to infected pneumonia class. This affirms the result of the sensitivity analysis that the contact rates c between susceptible individuals and infected individual is the most sensitive parameter**.** This claim was also supported by Mandell *et al* [25]. Therefore, reduction in effective contacts between susceptible and an infected individual is recommended through good sanitation among other measures.

The effect of treatment rate τ was considered in **Figure 5**. It was observed that the number of treated individuals with pneumonia decreases as the treatment rate τ using anti – pneumonia drugs on the infected individuals in the population increases. **Figure 6** explains the role vaccination played in individuals exposed to pneumonia disease. The anti-pneumonia vaccines prevent exposed individuals from progressing to the infectious class. Increase in the vaccination rate, $\boldsymbol{V_p}$, result in decrease in the number of exposed individuals. This result is supported by the claim of ([30], [32]).

The pneumonia model discussed in case 2 exhibited a forward bifurcation which suggest that it is sufficient to reduce $\boldsymbol{R_p}$ below unity to guarantee total eradication of pneumonia disease in

the population while the disease persist in the population if R_p is greater than unity as shown in **Figure 7**. As established in the malaria model and pneumonia model of this study, that increase in treatment rate of malaria and pneumonia diseases reduce the transmission rates of the two diseases, hence, **Figure 8** also establish that using the combination of anti-malaria and anti-pneumonia drugs reduces the co-infection of malaria - pneumonia diseases. The result of [22] affirm our assertion that individual with malaria – pneumonia symptoms overlap are treated with both anti – malaria and anti – biotics. The bifurcation diagram for the malaria – pneumonia co – infection is presented in **Figure 9**. The diagram above is in perfect agreement with our analytical result; that where malaria and pneumonia co – exist the condition $R_m < R_c < R_p < 1$ is required for malaria and pneumonia to be eradicated in the population. Note that $R_{mp} = max\{R_m, R_p\}$, where R_{mp} is defined to be the basic reproduction number for co – infection of mlaria and pneumonia.
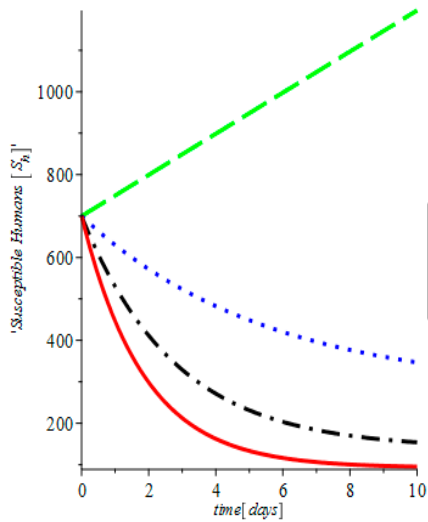
Figure 1: The variation of proportion of Susceptible Human population for different values of α
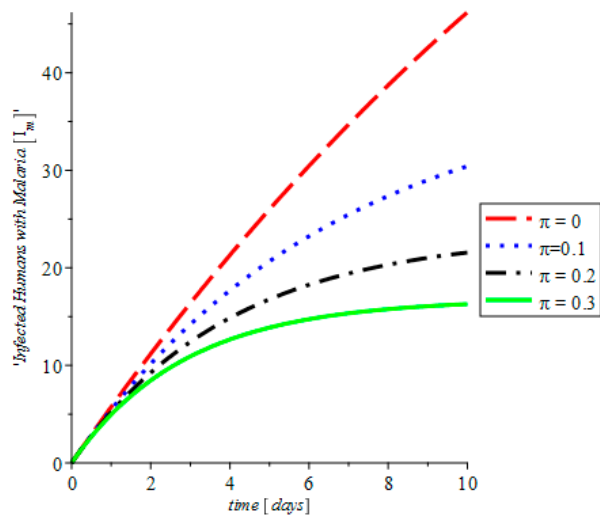
Figure 2: The variation of proportion of malaria infected humanpopulation for different values of π
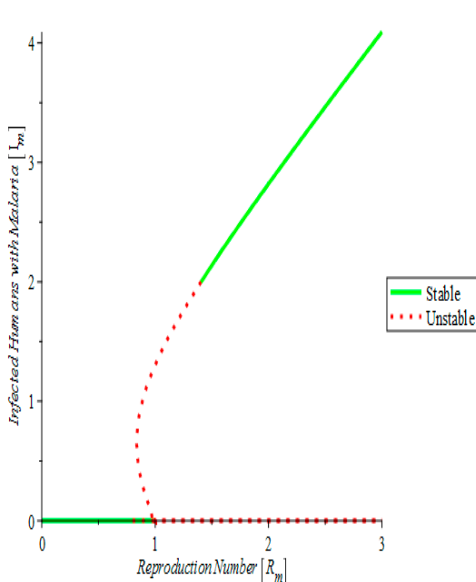
Figure 3: The backward bifurcation diagram for malaria infection
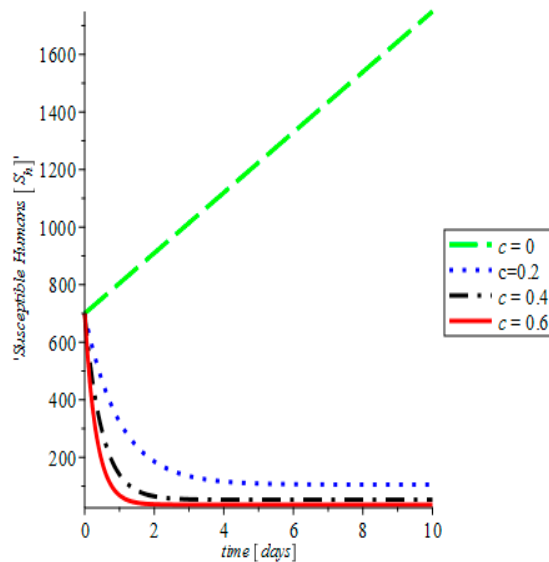
Figure 4: The variation of proportion of susceptible human population for different values of c

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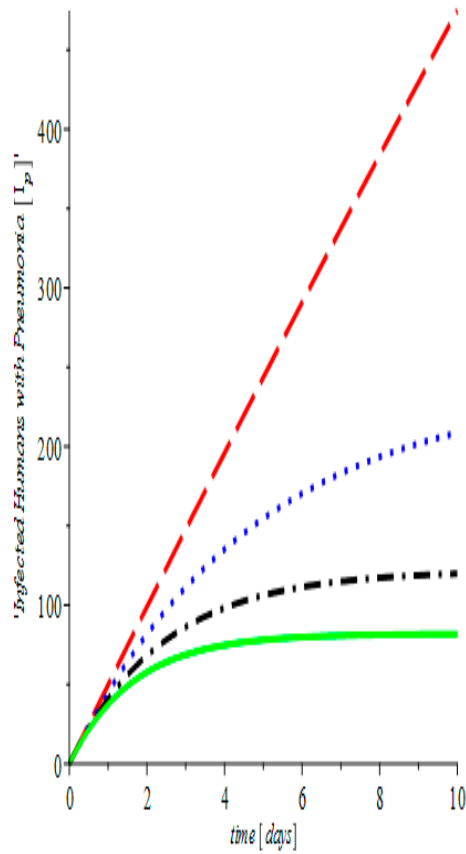
Figure 5: The variation of proportion of susceptible human population for different values of τ
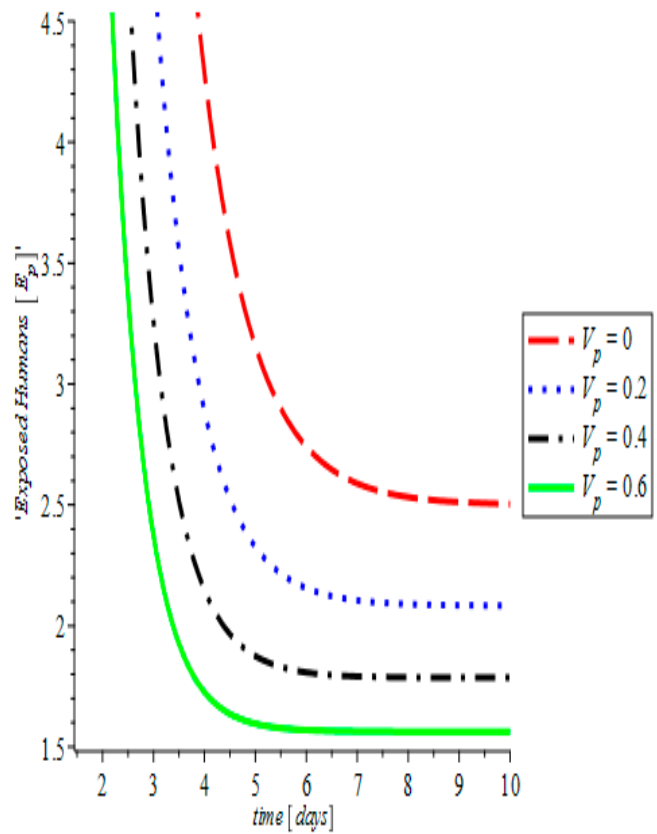
Figure 6: The variation of proportion of exposed human population to pneumonia for different values of vaccination rates (V_p)
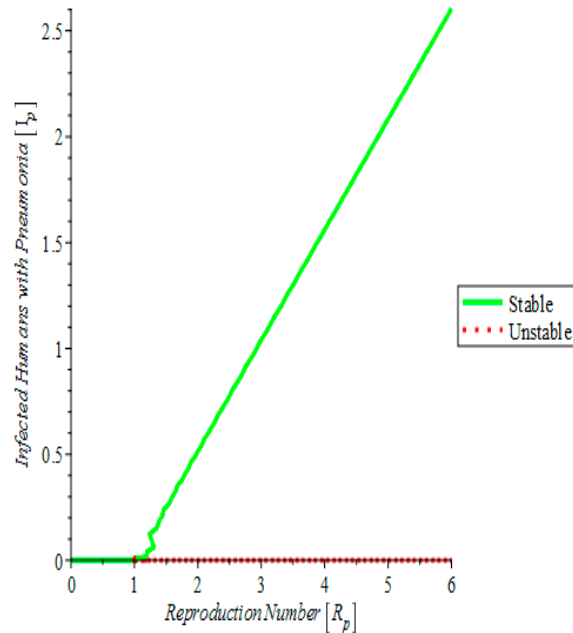
Figure 7: The forward bifurcation diagram for pneumonia infection
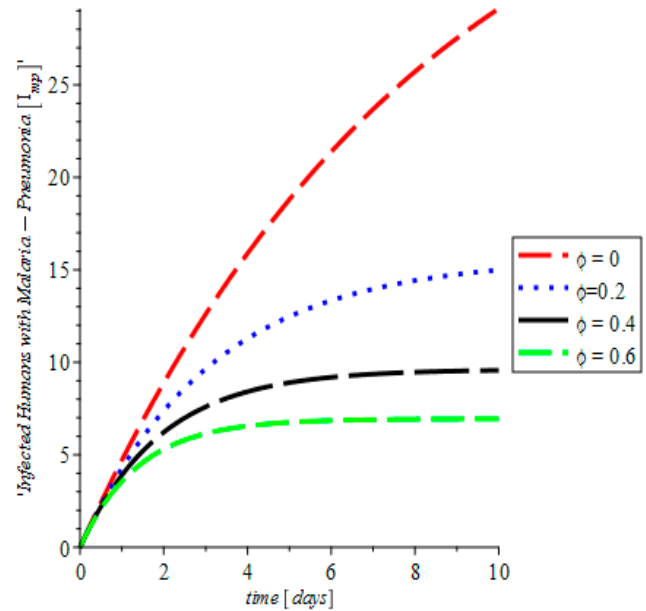
Figure 8: The variation of proportion of malaria-pneumonia infected human population for different values of ϕ

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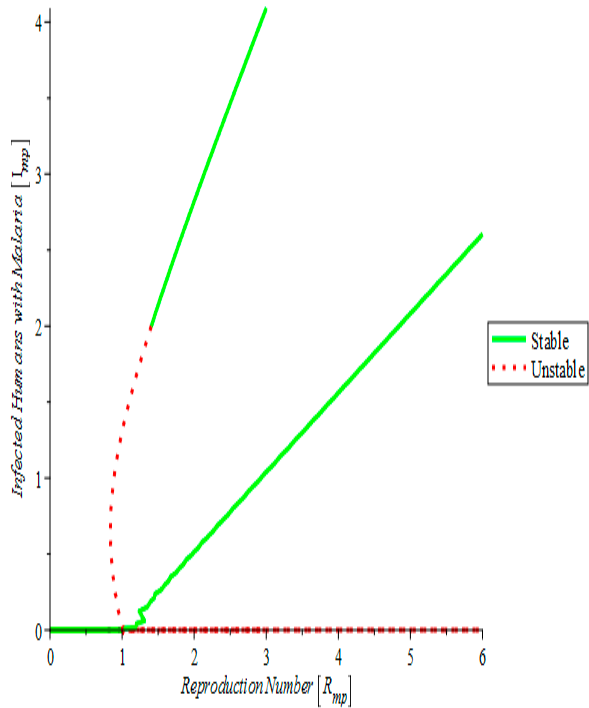
Figure 9: The co-infection bifurcation diagram for malaria and pneumonia infection
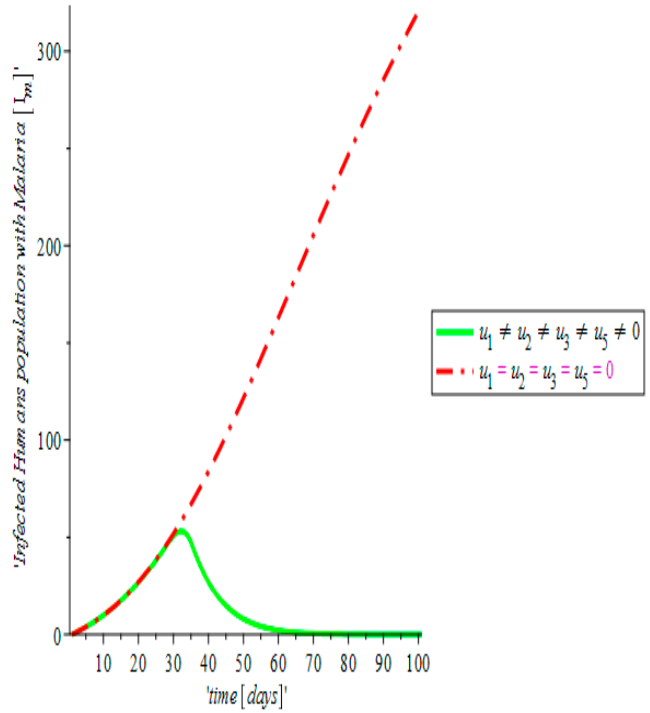
Figure 10: The variation of proportion of malaria infected population with and without control
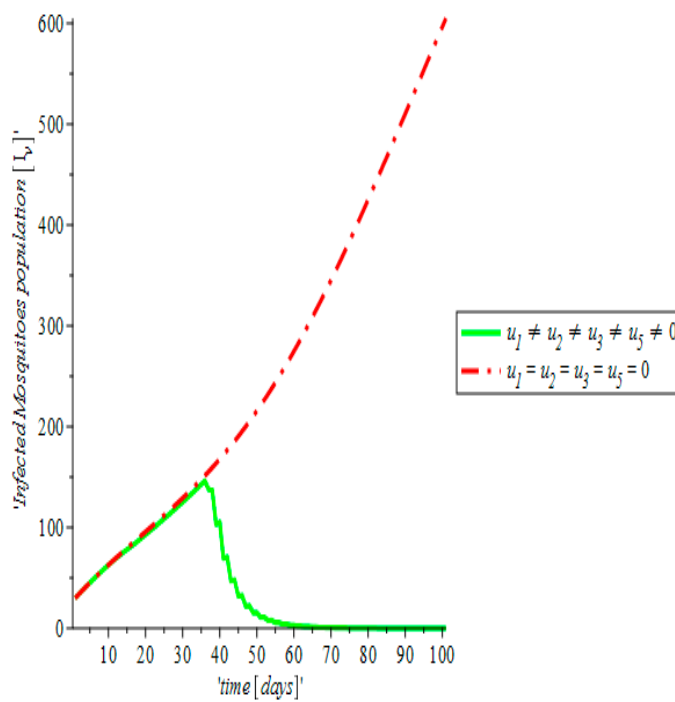
Figure 11: The variation of proportion of mosquito infected population with and without control
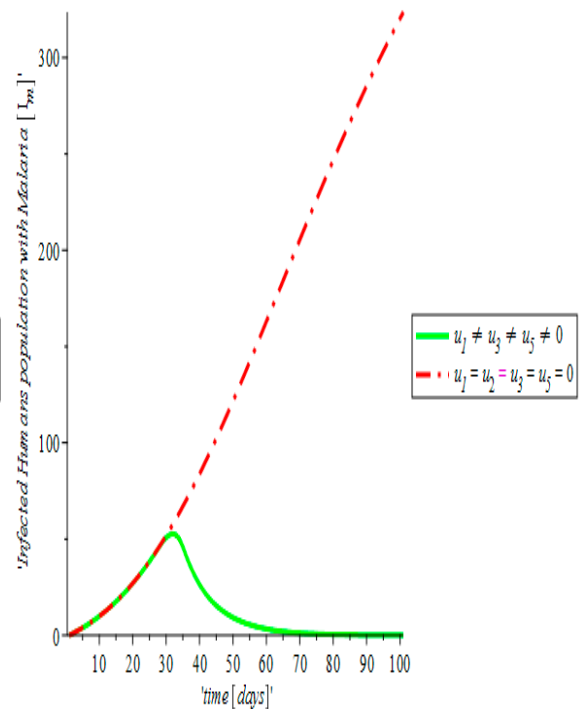
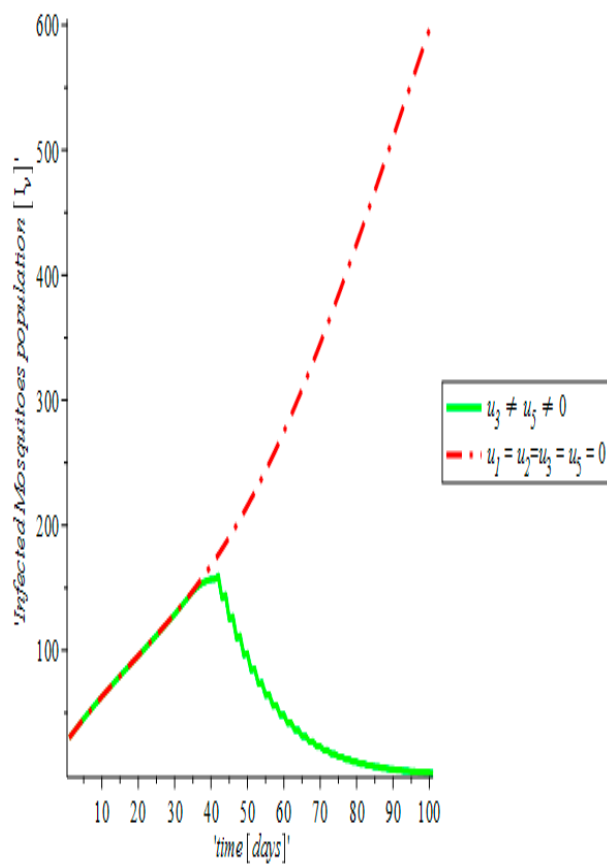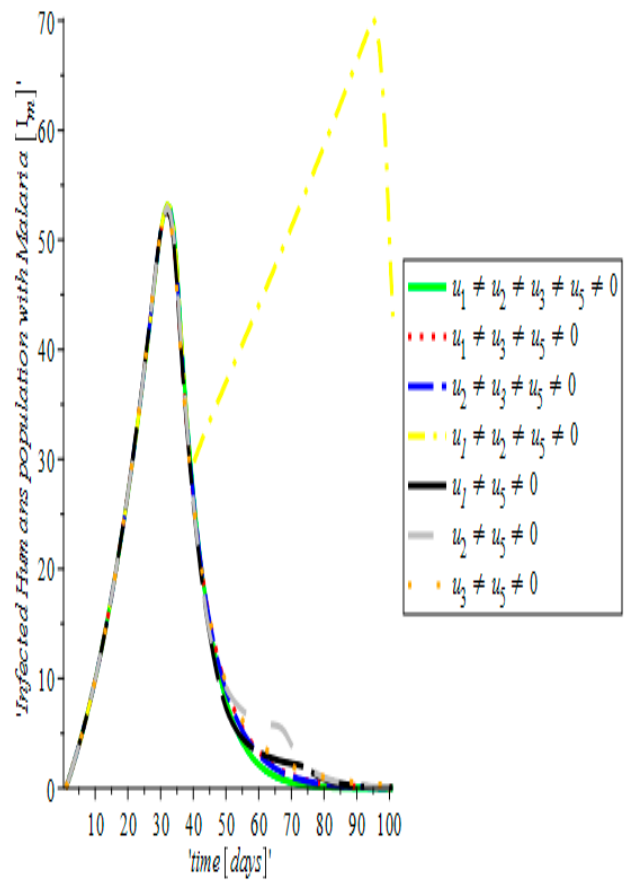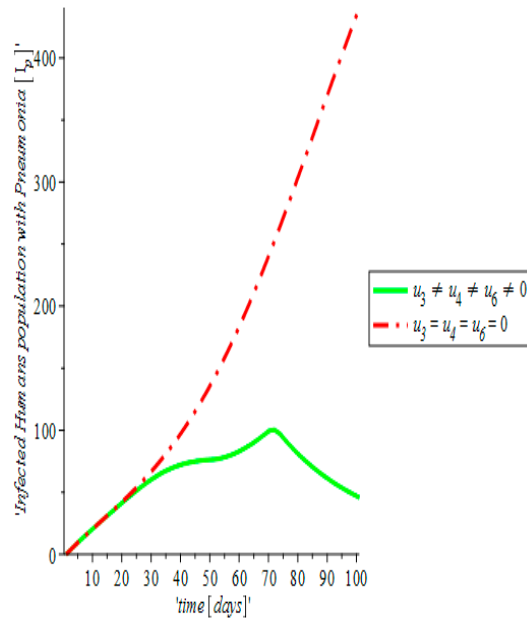Figure 12: The variation of proportion of malaria infected population with and without control
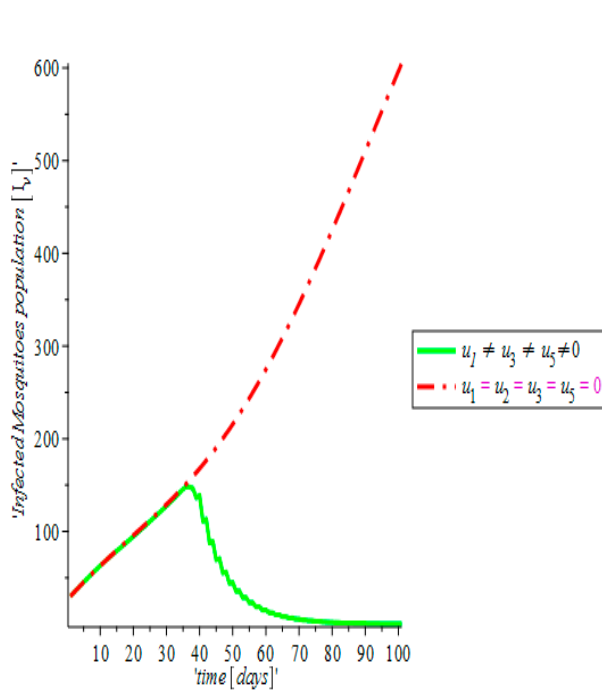
Figure 13: The variation of proportion of mosquito infected population with and without control
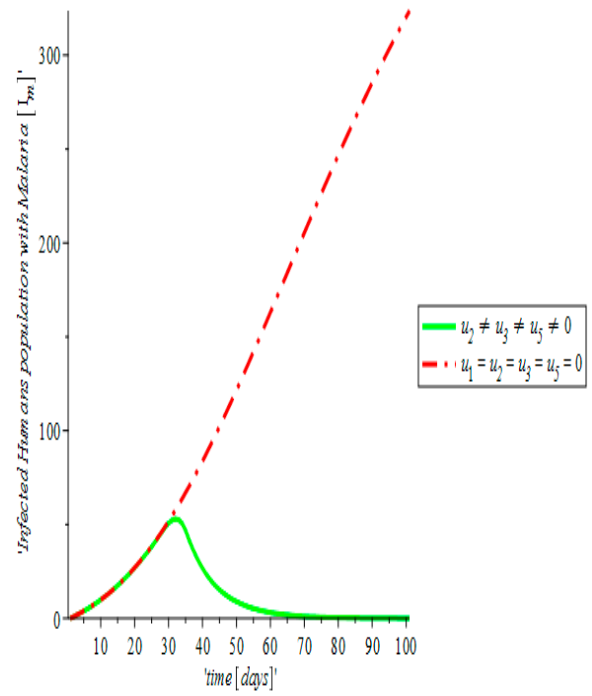
Figure 14: The variation of proportion of malaria infected population with and without control
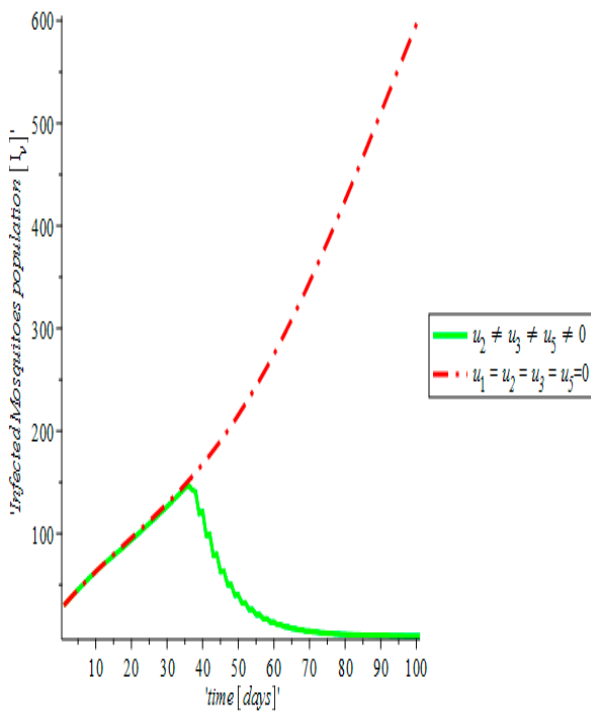
Figure 15: The variation of proportion of mosquito infected population with and without control
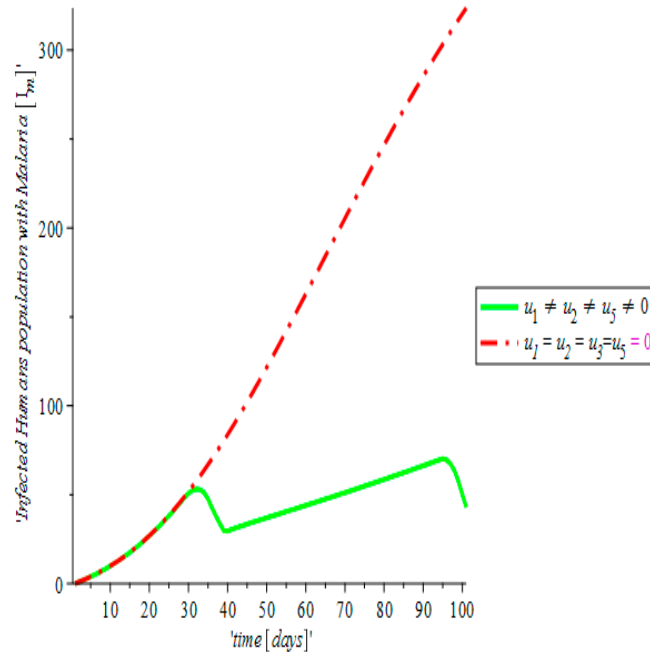
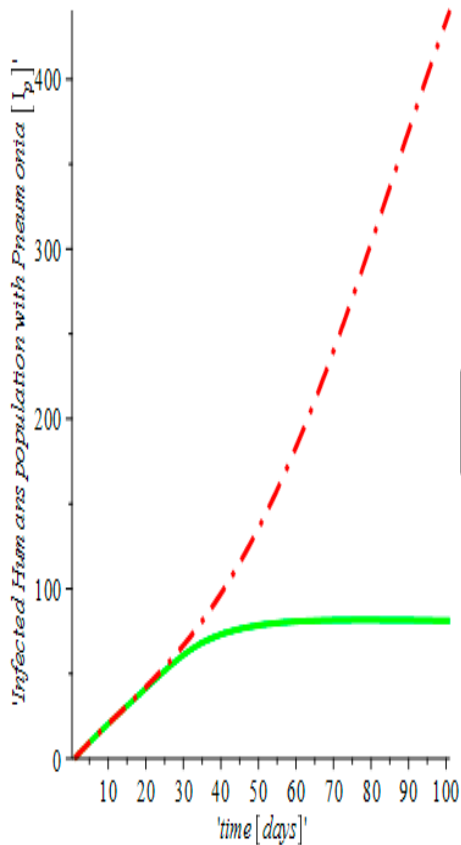Figure 16: The variation of proportion of malaria infected population with and without control
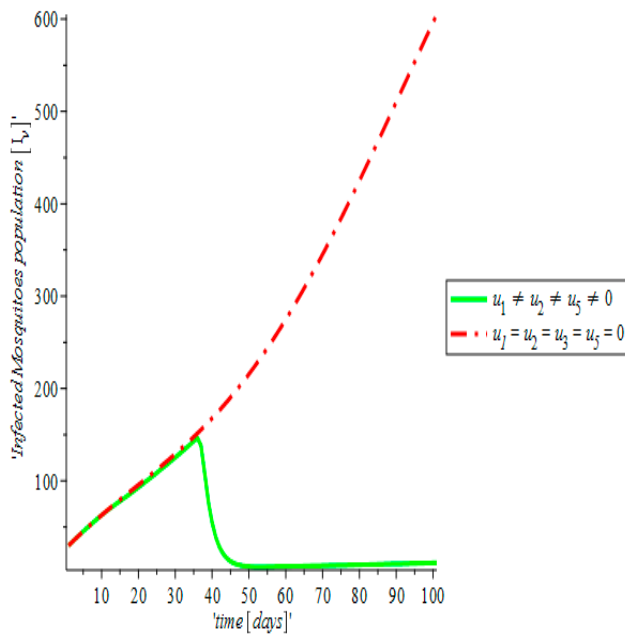
Figure 17: The variation of proportion of mosquitoes infected population with and without control
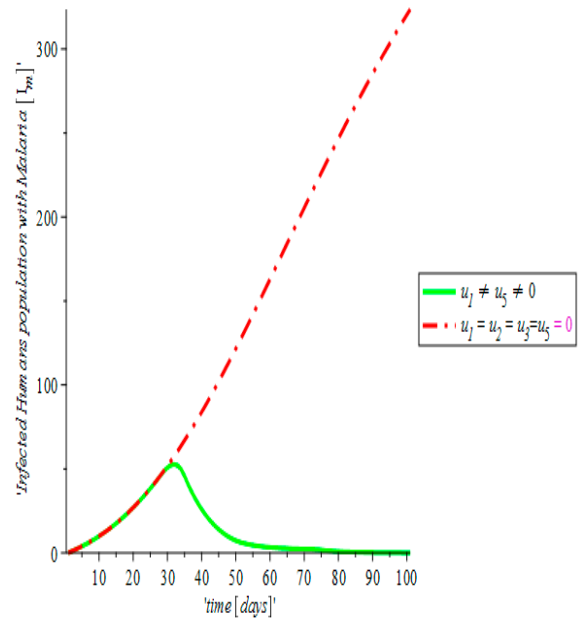
Figure 18: The variation of proportion of malaria infected population with and without control
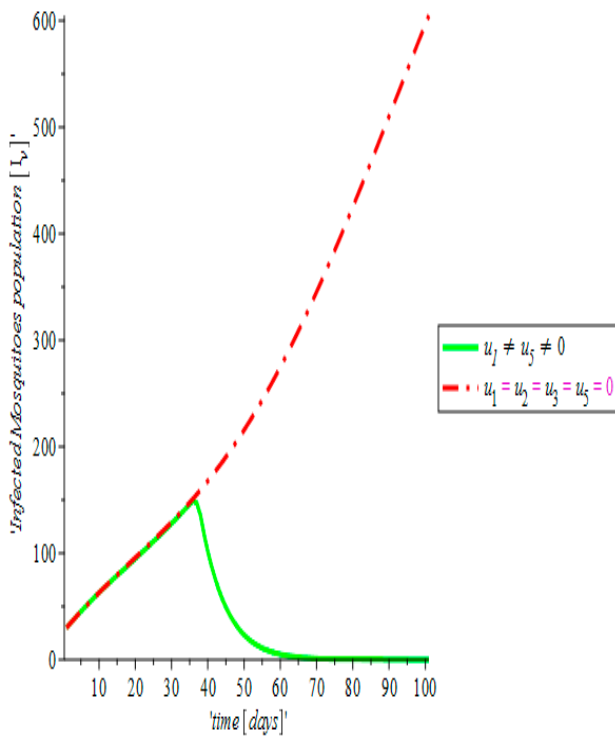
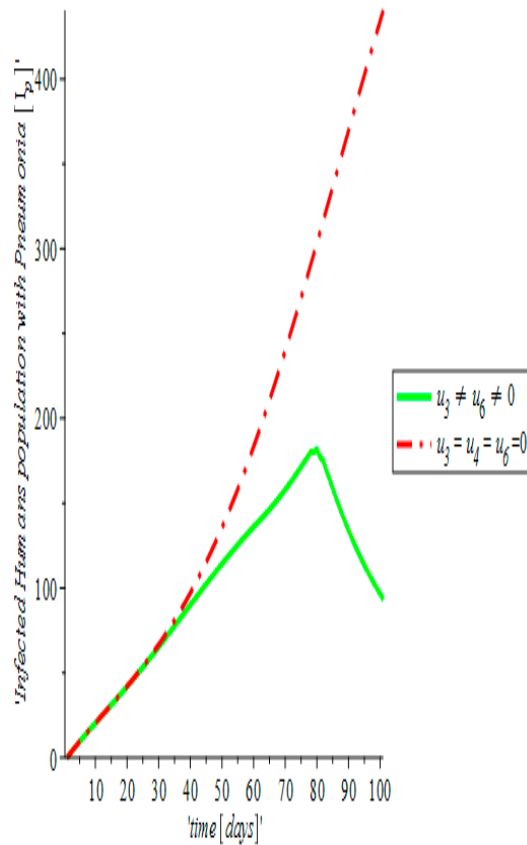Figure 19: The variation of proportion of mosquito infected population with and without control
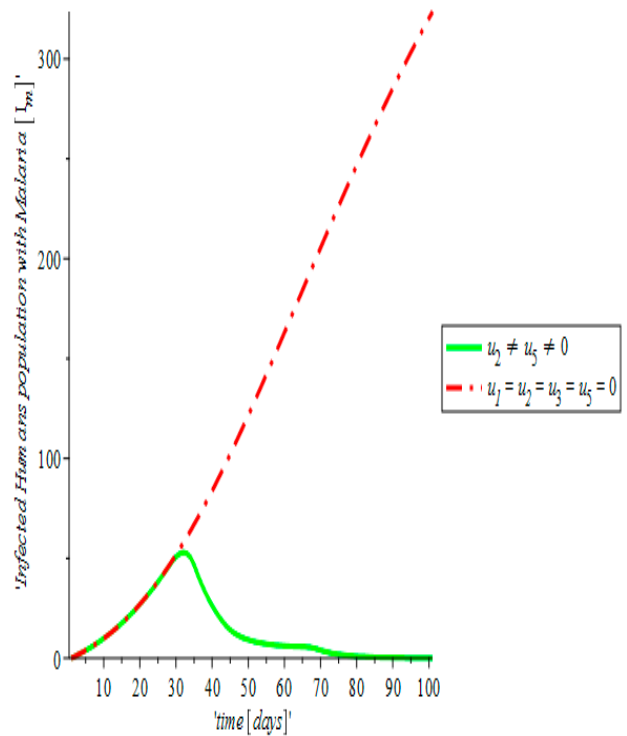
Figure 20: The variation of proportion of malaria infected population with and without control
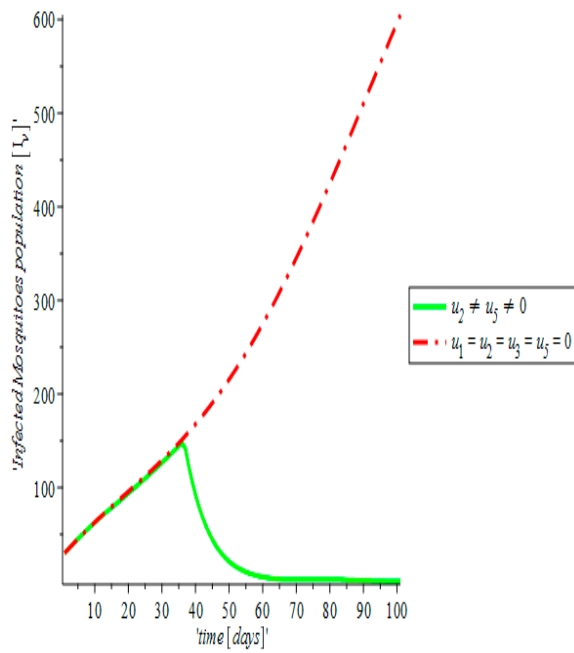
Figure 4.21: The variation of proportion Of mosquito infected population with and without control
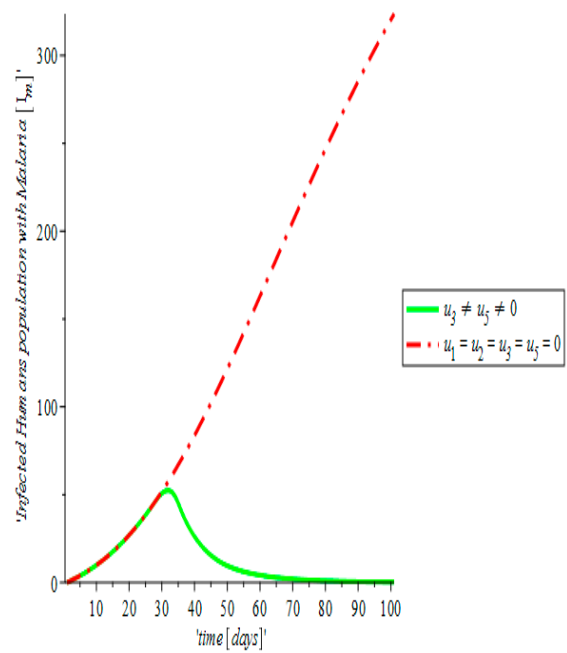
Figure 4.22: The variation of proportion of malaria infected population with and without control

Figure 23: The variation of proportion of mosquito infected population with and without control

Figure 24: The comparison of the effect of the different control strategies on infected humans with malaria for the malaria model

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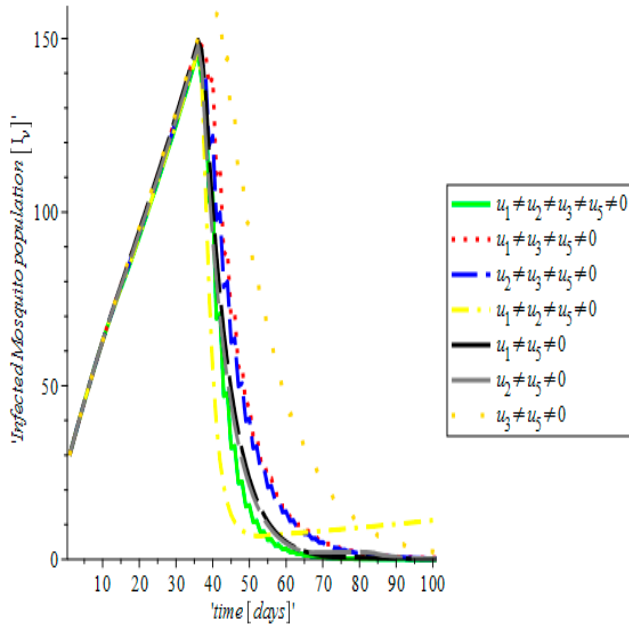
Figure 25: The comparison of the effect Of the different control strategies on infected mosquitoes for the malaria model

Figure 4.26: The variation of proportion of pneumonia infected population with and without control

Figure 27: The variation of proportion of pneumonia infected population with and without control

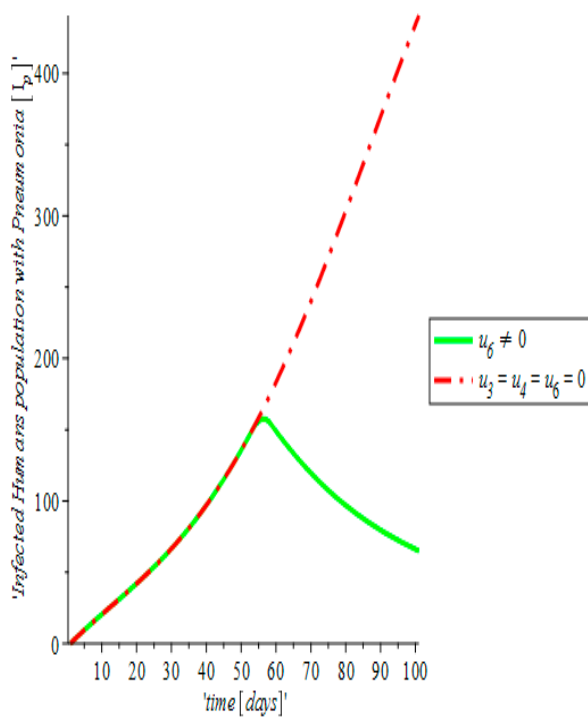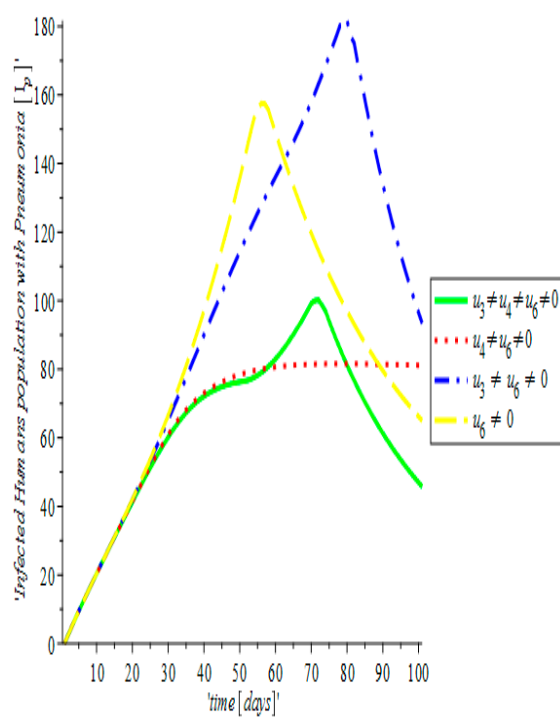Figure 28: The variation of proportion of pneumonia infected population with and without control

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Figure 29: The variation of proportion of pneumonia infected population with and without control

Figure 30: The comparison of the effect of the different control strategies on infected humans with pneumonia for the pneumonia model
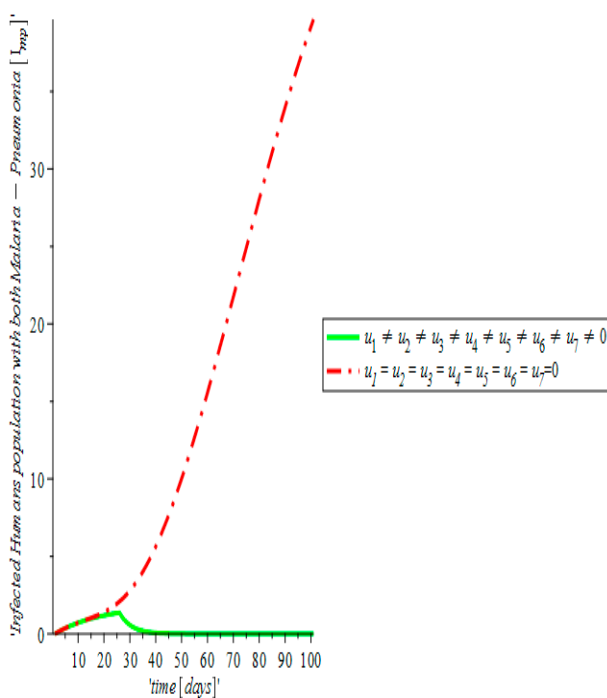

Figure 31: The variation of proportion of malaria-pneumonia infected population with and without control
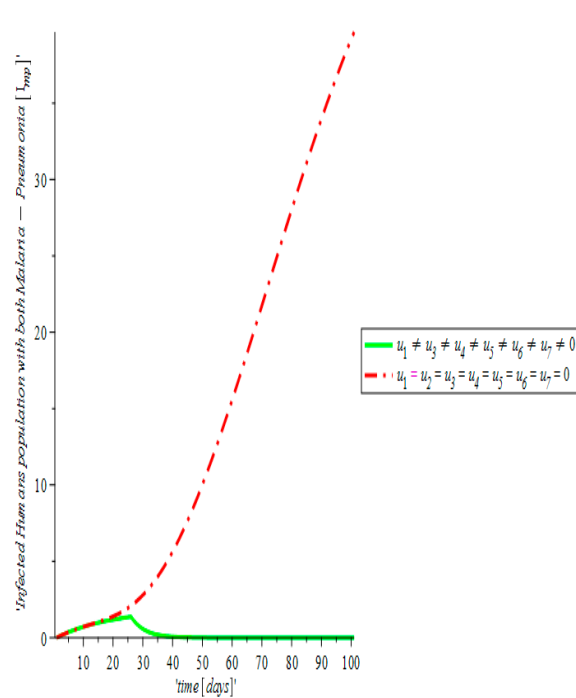

Figure 32: The variation of proportion of malaria-pneumonia infected population with and without control
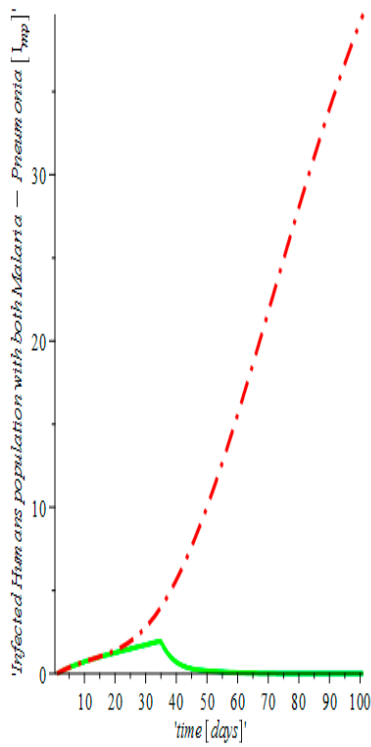
Figure 33: The variation of proportion of malaria-pneumonia infected population with and without control
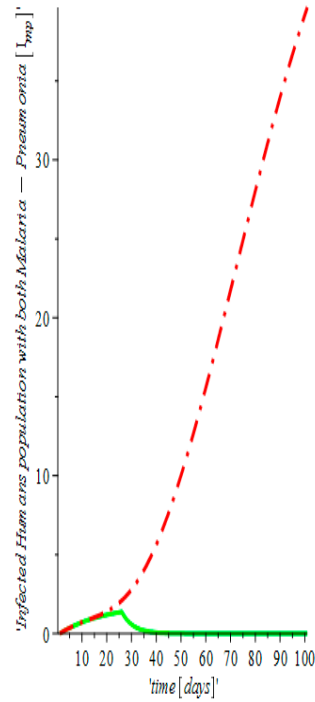

Figure 34: The variation of proportion of malaria -pneumonia infected population with and without contro

Figure 35: The variation of Proportion of malaria-pneumonia infected population with and without control

Figure 36: The variation of proportion of malaria-pneumonia infected population with and without control

Figure 37: The variation of proportion of malaria-pneumonia infected population with and without control

Figure 38: The variation of proportion of malaria-pneumonia infected population with and without control
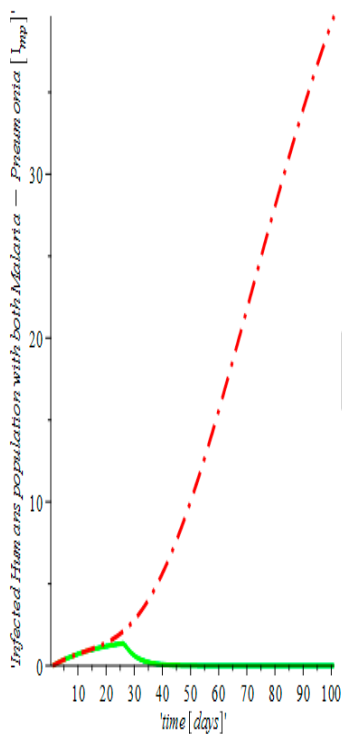

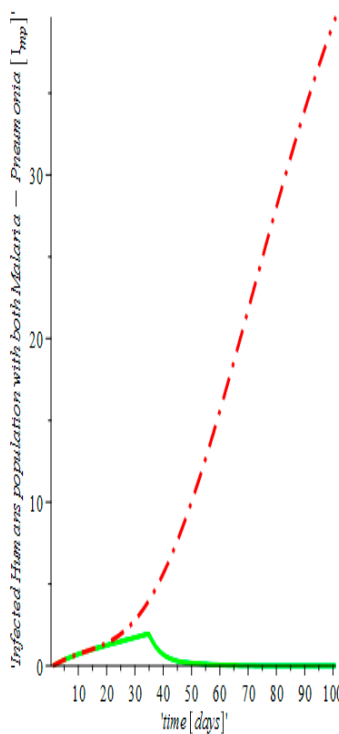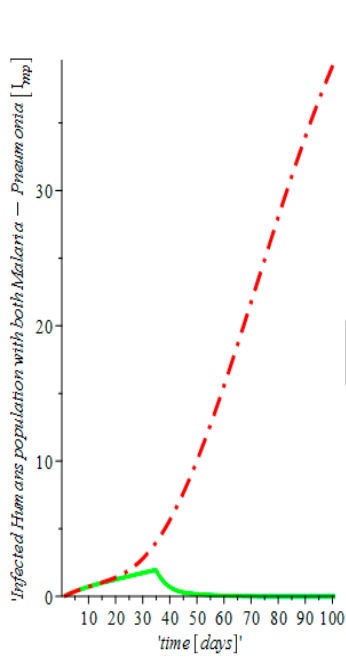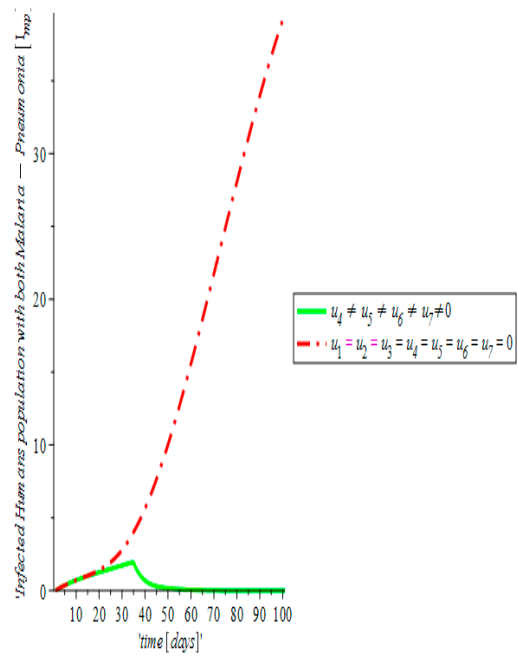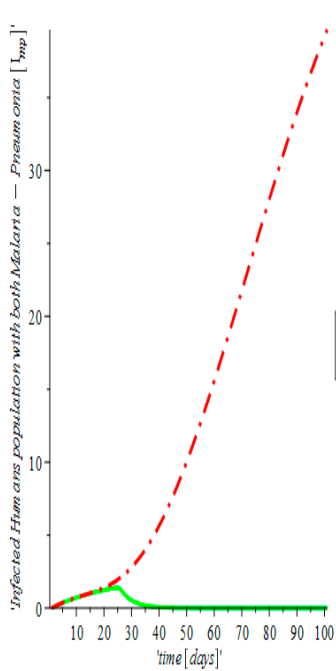Figure 39: The variation of proportion of malaria-pneumonia infected population with and without control
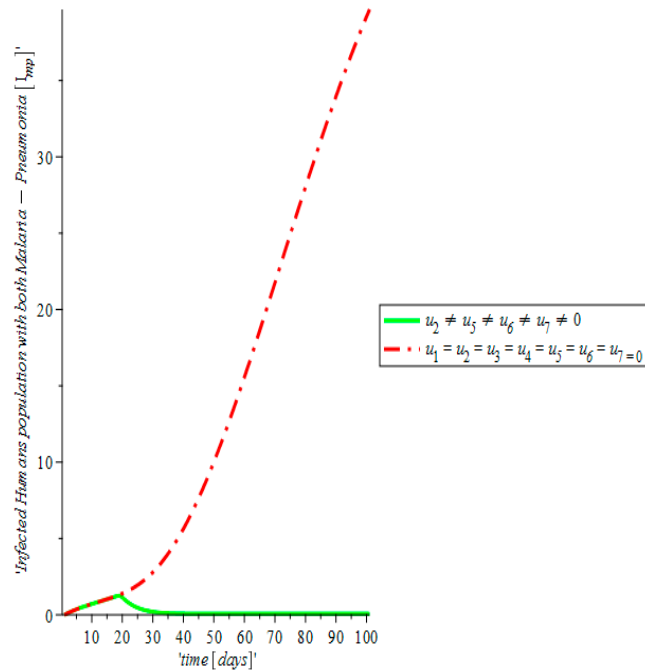

Figure 40: The variation of proportion of malaria-pneumonia infected population with and without control
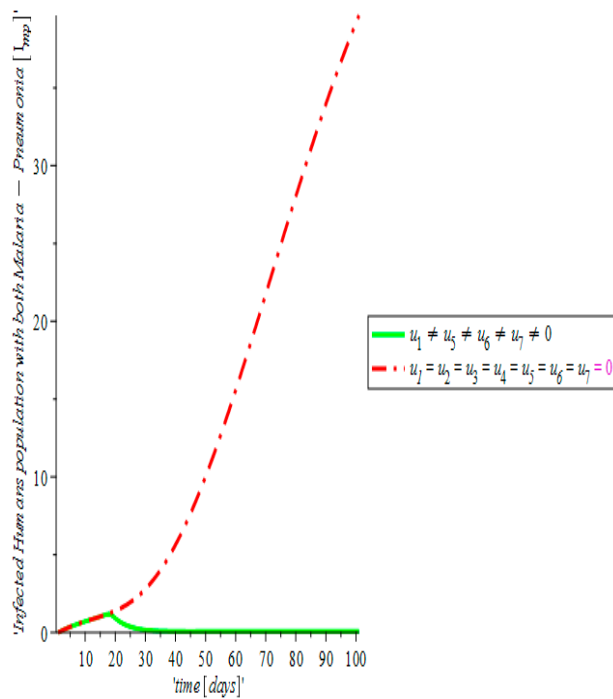
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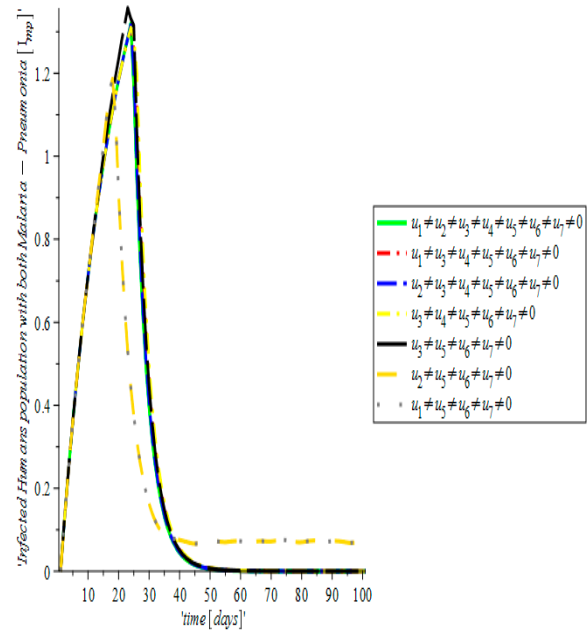Figure 4.41: The variation of proportion of malaria-pneumonia infected population with and without control

Figure 4.42: The comparison of the effect of the different control strategies on infected humans with malaria-pneumonia for the malaria-pneumonia model

We further explore an optimal control model with time preventive (sleeping under treated mosquito nets $u_1(t)$, Insecticide spray $u_2(t)$ and sanitation $u_3(t)$) and treatment (use of anti-malaria drug $u_5(t)$) strategies as control measures on the transmission of malaria disease. Various combinations of the controls are considered as follows:

(a)   Optimal control using: sleeping under treated mosquito nets $u_1(t)$, insecticide spray $u_2(t)$, sanitation $u_3(t)$ and treatment $u_5(t)$.

Here, all the four controls are used to optimize the objective function J. It was observed in **Figure 10** and **Figure 11** that the combination of the four controls resulted in significant decrease in both the number of infected humans with malaria (I_m) and infected mosquitoes (I_v) (represented by the green solid line) as against the increased number of infected humans (I_m) and infected mosquitoes (I_v) (denoted by the red dash dot line) in the uncontrolled case.

(b)   Optimal control using: sleeping under treated mosquito nets $u_1(t)$, sanitation $u_3(t)$ and treatment $u_5(t)$.

In this case, the control on spraying insecticide $u_2(t)$ is set to zero while the controls on sleeping under treated mosquito nets $u_1(t)$, good sanitation $u_3(t)$ and treatment $u_5(t)$ are used to optimize the objective function J. Using this strategy, it was observed in **Figure 12** and **Figure 13** that the number of infected humans with malaria (I_m) and infected mosquitoes (I_v) decreases as against the increase observed in the uncontrolled case.

(c)   Optimal control using: insecticide spray $u_2(t)$, sanitation $u_3(t)$ and treatment $u_5(t)$.

Here, the controls on spraying insecticide $u_2(t)$, good sanitation $u_3(t)$ and treatment $u_5(t)$ are used to optimize the objective function J while the control on sleeping under mosquito treated nets $u_1(t)$ is set to zero. Considering this strategy, it was observed in **Figure 14** and **Figure 15** that the number of infected humans with malaria (I_m) and infected mosquitoes (I_v) decreases as against the increase observed in the uncontrolled case respectively.

(d)   Optimal control using: sleeping under treated mosquito nets $u_1(t)$, insecticide spray $u_2(t)$ and treatment $u_5(t)$.

The control on sleeping under mosquito treated nets $u_1(t)$, insecticide spray $u_2(t)$ and treatment $u_5(t)$ are used to optimize the objective function J while the control on sanitation was set to zero. It was observed in **Figure 16** that the control measure employed here decreases the number of infected humans with malaria (I_m) but not to the level of the controls involving good sanitation as against the increase observed in the uncontrolled case. Although, a decrease in the number of infected mosquitoes (I_v) was observed as against the uncontrolled case in **Figure 17** but due to poor or no sanitation, the number of infected mosquitoes (I_v) began to rise.

(e)   Optimal control using: sleeping under treated mosquito nets $u_1(t)$ and treatment $u_5(t)$.

With this strategy, the controls sleeping under treated mosquito nets $u_1(t)$ and treatment $u_5(t)$ are used to optimize the objective function J with the control on insecticide spray $u_2(t)$ and good sanitation $u_3(t)$ all set to zero. For this strategy, it was shown in **Figure 18** and **Figure 19** that the number of infected humans with malaria (I_m) and infected mosquitoes (I_v) reduces considerably from the uncontrolled case

(f)   Optimal control using: insecticide spray $u_2(t)$ and treatment $u_5(t)$

With this strategy, the controls insecticide spray $u_2(t)$ and treatment $u_5(t)$ are used to optimize the objective function J, while the control on sleeping under mosquito treated nets $u_1(t)$ and good sanitation $u_3(t)$ all set to zero. The result in **Figure 20** and **Figure 21** showed a significant difference in the number of infected humans with malaria (I_m) and infected mosquitoes (I_v) with optimal strategy compared to (I_m) and (I_v) without control. It was observed in **Figure 20** that the control strategies resulted in a decrease in the number of infected humans with malaria (solid green line) as against an increase in the uncontrolled case (red dash dot line). Also, in **Figure 21**, the uncontrolled case resulted in increased number of infected mosquitoes (I_v), while the control strategy lead to a drastic decrease in the number of infected mosquitoes (I_v).

(g)   Optimal control using: sanitation $u_3(t)$ and treatment $u_5(t)$.

The objective function J are optimized in this case using the control strategy on good sanitation $u_3(t)$ and treatment $u_5(t)$, while the control on sleeping treated mosquito nets $u_1(t)$ and insecticide spray $u_2(t)$ are set to zero. It was observed in **Figure 22** that the control strategies resulted in a decrease in the number of infected humans (I_m) with malaria as against increase in the uncontrolled case. Similarly, in **Figure 23**, the uncontrolled case resulted in increased number of infected mosquitoes (I_v), while the control strategy lead to a decrease in the number of infected mosquitoes.

(h)   Comparing all the control strategies

A comparison of all the control strategies for our malaria model case 4 was considered on both infected humans (I_m) and mosquitoes (I_v) in **Figure 24** and **Figure 25** respectively. It was observed that all the four controls led to a decrease in the number of infected humans (I_m) and mosquitoes (I_v) respectively. The control strategies ($u_1(t), u_3(t), u_5(t)$) and ($u_2(t), u_3(t), u_5(t)$) also led to a decrease in the number of infected humans and mosquitoes respectively. The strategy ($u_1(t), u_2(t), u_5(t)$) yield a poorer result on both the infected humans and mosquitoes. This result showed that with individuals sleeping under mosquito treated nets, spraying of insecticide in the sleeping area and have access to treatment may not be sufficient to control the spread of the disease in the community if poor sanitation is encouraged. The optimal strategies involving two controls namely: ($u_1(t), u_5(t)$), ($u_2(t), u_5(t)$) and ($u_3(t), u_5(t)$) decreases the number of infected humans (I_m) and mosquitoes (I_v). From **Figure 24** and **Figure 25**, it was also observed that the optimal control strategies involving good sanitation gave a better result in terms of reducing the number of infected humans (I_m) and mosquitoes (I_v).

The result of the optimal control for pneumonia is discussed using time dependent preventive and treatment controls to investigate the effect of these controls on the transmission dynamics of pneumonia disease. The following scenarios of the controls are considered:

(a)   Optimal control using: sanitation $u_3(t)$, vaccination $u_4(t)$ and treatment $u_6(t)$

The objective function J is optimized here using all the three controls sanitation $u_3(t)$, vaccination $u_4(t)$ and treatment $u_6(t)$. We observed from **Figure 26** that the combination of the three controls resulted in significant reduction in the number of infected humans with pneumonia (I_p) (represented by solid green line) as against the increased number of infected humans with pneumonia disease (I_p) (denoted by red dash dot line) in the uncontrolled case.

(b)   Optimal control using: vaccination $u_4(t)$ and treatment $u_6(t)$

In this case, the control on using vaccination $u_4(t)$ and treatment $u_6(t)$ are used to optimize the objective function J while the control on sanitation $u_3(t)$ is set to zero. For this strategy, it was observed in **Figure 27** that the number of infected human with pneumonia reduce considerably and maintain a constant level over time while the number of infected humns with pneumonia increases in the uncontrolled case.

(c)   Optimal control using: sanitation $u_3(t)$ and treatment $u_6(t)$

Here, the control on using sanitation $u_3(t)$ and treatment $u_6(t)$ are used to optimize the objective function J with the control on vaccination $u_4(t)$ set to zero. The number of infected humans with pneumonia was observed to be decreasing as shown in **Figure 28** while the number of infected humans with pneumonia increases considerably in the uncontrolled case.

(d)   Optimal control using: treatment $u_6(t)$

With this strategy, the control on using only treatment $u_6(t)$ was used to optimize the objective function J while the controls on sanitation $u_3(t)$ and vaccination $u_4(t)$ are set to zero. The result in **Figure 29** revealed that for the controlled case, the number of infected humans with pneumonia reduces (green line) while the number of infected human with pneumonia increases for the uncontrolled case (red dash dot line).

(e)   Comparing all the control strategies

A comparison of all the control strategies for the pneumonia model case 5 was considered on the infected humans (I_p) **Figure 30**. It was observed that all the four controls led to a decrease in the number of infected humans (I_p) this was closely followed by the strategy of using anti-pneumonia as treatment. The strategy involving vaccination and treatment also gave a good result in reducing the number of infected humans (I_p) and also ensure that exposed humans do not progress to the infected class. Lastly, the strategy involving the use of good sanitation and treatment also gave a good result in decreasing the number of infected humans (I_p).

The malaria-pneumonia model with time dependent preventive and treatment control strategies is considered in this section. The following scenarios are considered as follows:

(a)   Optimal control using: sleeping under mosquito treated nets $u_1(t)$, insecticide spray $u_2(t)$, sanitation $u_3(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

The objective function J for malaria-pneumonia model is optimized in this case using all the seven controls. It was seen in **Figure 31** that the combinations of the seven controls resulted in significant decrease in the number of infected humans with malaria-pneumonia (I_{mp}) (solid green line) as against the increase observed in the number of infected humans (I_{mp}) (dash dot red line) in the uncontrolled case.

(b)   Optimal control using: sleeping under mosquito treated nets $u_1(t)$, sanitation $u_3(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

In this case, the control on insecticide spray $u_2(t)$ is set to zero while the controls on sleeping under mosquito treated nets $u_1(t)$, sanitation $u_3(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ are used to optimize the objective function J. Using this strategy, it was observed in **Figure 32** that the number of infected humans with malaria-pneumonia (I_{mp}) decreases as against the increase observed in the number of infected humans (I_{mp}) in the uncontrolled case.

(c)   Optimal control using: sleeping under mosquito treated nets $u_1(t)$, insecticide spray $u_2(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

Here, the control on sleeping under mosquito treated nets $u_1(t)$, insecticide spray $u_2(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ are used to optimize the objective function J while sanitation $u_3(t)$ is set to zero. For this strategy, it was observed in **Figure 33** that the number of infected humans with malaria-pneumonia (I_{mp}) decreases as against the increase observed in the number of infected humans (I_{mp}) in the uncontrolled case.

(d)   Optimal control using: insecticide spray $u_2(t)$, sanitation $u_3(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

The control on sleeping under mosquito treated nets $u_1(t)$ is set to zero while the controls on insecticide spray $u_2(t)$, sanitation $u_3(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ are used to optimize the objective function J. Using this strategy, it was observed in **Figure 34** that the number of infected humans with malaria-pneumonia (I_{mp}) decreases as against the increase observed in the number of infected humans (I_{mp}) in the uncontrolled case.

(e)   Optimal control using: sanitation $u_3(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

For this strategy, the controls on sanitation $u_3(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ are used to optimize the objective function J while the controls on sleeping under mosquito treated nets $u_1(t)$ and insecticide spray $u_2(t)$ are set to zero. It was observed as shown in **Figure 35** that this strategy decreases the number of infected humans with malaria-pneumonia (I_{mp}) while the number of infected humans with malaria-pneumonia (I_{mp}) increases for the uncontrolled case.

(f)   Optimal control using: insecticide spray $u_2(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

The control on insecticide spray $u_2(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ optimizes the objective function J with the controls on sleeping under mosquito treated nets $u_1(t)$ and good sanitation $u_3(t)$ set to zero. For this control measure, it was shown in **Figure 36** that the number of infected humans with malaria-pneumonia (I_{mp}) reduces significantly from the uncontrolled case.

(g)   Optimal control using: sleeping under mosquito treated nets $u_1(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

With this strategy, the control on sleeping under mosquito treated nets $u_1(t)$, vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ are used to optimize the objective function J, while the control on insecticide spray $u_2(t)$ and sanitation $u_3(t)$ are all set to zero. The results in **Figure 37** revealed a significant difference in the number of infected humans with malaria-pneumonia (I_{mp}) compared to infected

humans with malaria-pneumonia (I_{mp}) without control. It was observed in Figure 37 that the control strategies resulted in a decrease in the number of infected humans (I_{mp}) as against an increase in the uncontrolled case.

(h)   Optimal control using: vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

The objective function J is optimized in this case using the control strategy on vaccination $u_4(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$, while the controls on sleeping under mosquito treated nets $u_1(t)$, insecticide spray $u_2(t)$ and sanitation $u_3(t)$ are all set to zero. We observed in Figure 38 that the control strategies resulted in a decrease in the number of infected humans with malaria-pneumonia (I_{mp}). Similarly, From **Figure 38**, the uncontrolled case resulted in increased number of infected humans with malaria-pneumonia (I_{mp}).

(i)   Optimal control using: sanitation $u_3(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

Using this strategy, the control on using sleeping under mosquito treated nets $u_1(t)$, insecticide spray $u_2(t)$ and vaccination $u_4(t)$ are all set to zero, while the control on sanitation $u_3(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ are used to optimize the objective function J. The number of infected humans with malaria-pneumonia (I_{mp}) was observed to be increasing for the uncontrolled case (dash dot red line) as shown in **Figure 39** while the number of infected humans with malaria-pneumonia (I_{mp}) decreases for the controlled case (solid green line).

(j)   Optimal control using: insecticide spray $u_2(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

The objective function J here is optimized using insecticide spray $u_2(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ as controls while the controls on sleeping under mosquito treated nets $u_1(t)$, sanitation $u_3(t)$ and vaccination $u_4(t)$ are all set to zero. It was observed in **Figure 40** that this strategy resulted in significant decrease in the number of infected humans with malaria-pneumonia (I_{mp}) while an increase was observed in the uncontrolled case.

(k)   Optimal control using: sleeping under mosquito treated nets $u_1(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$

For this strategy, the control on insecticide spray $u_2(t)$, sanitation $u_3(t)$ and vaccination $u_4(t)$ are set to zero while the controls on sleeping under mosquito treated nets $u_1(t)$, treatment for malaria $u_5(t)$, treatment for pneumonia $u_6(t)$, treatment for both malaria and pneumonia $u_7(t)$ was used to optimize the objective function J. It was shown in **Figure 41** that the controlled strategies resulted in a decrease in the number of infected humans with malaria-pneumonia (I_{mp}) as against an increase observed in the uncontrolled case.

(l)   Comparing all the control strategies

A comparison of all the control strategies for the malaria-pneumonia model case 3 was considered on infected humans with malaria-pneumonia (I_{mp}) in **Figure 42**. It was observed that all the seven controls, followed by the combinations of six controls ($(u_1(t), u_3(t), u_4(t), u_5(t), u_6(t), u_7(t))$ and $(u_2(t), u_3(t), u_4(t), u_5(t), u_6(t), u_7(t))$), five controls $(u_3(t), u_4(t), u_5(t), u_6(t), u_7(t))$ and four controls $(u_3(t), u_5(t), u_6(t), u_7(t))$ all led to a significant decrease in the number of infected humans with malaria-pneumonia (I_{mp}). The strategies $(u_1(t), u_5(t), u_6(t), u_7(t))$ and $(u_2(t), u_5(t), u_6(t), u_7(t))$ yield a poorer result on the infected humans with malaria-pneumonia (I_{mp}) because of poor or no sanitation. This result showed that with individuals sleeping under mosquito treated nets, spraying of insecticide in the sleeping area, vaccination and access to

treatment may not be sufficient to control the spread of the disease in the community if poor or no sanitation is encouraged.

6.0   Conclusion

A nonlinear mathematical model has been developed and analyzed to study the dynamics of the co-infection of malaria-pneumonia using the mass action incidence. Here we incorporated the dynamics of sanitation by means of a linearly decreasing function both in terms of the transmission rate for malaria and contracting rate for pneumonia. The analysis of the model systems were performed using stability theory. Qualitative analysis of the model showed that the disease free equilibrium is locally asymptotically stable at threshold parameter less than unity and unstable at threshold greater than unity. Global stability of the model disease free equilibrium points were established using the comparison approach. Furthermore, the local stability of the model endemic equilibrium points were investigated by using Centre Manifold theory which showed that the malaria, pneumonia and the full malaria-pneumonia models endemic equilibriums are locally asymptotically stable whenever the associated basic reproduction number is greater than unity.

Sensitivity analysis of the model showed that the biting rate of mosquitoes, α, contact rate c and transmission rate β_p of pneumonia are the most sensitive parameters in the present study, which suggest that more attention should be focused on these parameters. As a result, an optimal control strategy to curtail the spread of malaria, pneumonia and their co-infection was further studied in this work by incorporating treatments, sleeping under insecticide nets, spraying of insecticides and good sanitation as controls measures. The optimal analysis was carried out using the Pontryagin's Maximum Principle.

Finally, numerical results are provided to validate the analytical results.

CONFLICT OF INTEREST
.The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT
The authors declare that all relevant data supporting the findings of this study are available within the article.

APPENDIX A

1. Centre Manifold Theorem [9]

Consider a general system of ODEs with a parameter ϕ
$$\dot{x} = f(x, \phi); f: R^n \times R \to R^n; f \in C^2(R^n \times R) \qquad (A.1)$$
Without loss of generality, assume that $x = 0$ is equilibrium for system (A.1).
Theorem A.1
Assume:
(i) $A = D_x f(0,0)$ is the linearization matrix of system (2.1) around the equilibrium $x = 0$ with ϕ evaluated at O. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts.
(ii) Matrix A has a (nonnegative) right eigenvector w and a left eigenvector v corresponding

to the zero eigenvalue. Let f_m denote the mth complements of

f and $a = \sum_{m,j,i=1}^{n} v_m w_i w_j \frac{\partial^2 f_m(0,0)}{\partial x_i \partial x_j}$; $\qquad$ $b = \sum_{m,j,i=1}^{n} v_m w_i \frac{\partial^2 f_m(0,0)}{\partial x_i \partial \phi}$

Then the local dynamics of system (2.1) around $x = 0$ are totally determined by a and b

(iii) $a > 0$, $b > 0$. When $\phi < 0$, with $|\phi| \ll 1$, $x = 0$ is locally asymptotically stable and there exist a positive unstable equilibrium; when $0 < \phi \ll 1$, $x = 0$ is unstable and there exist a negative and locally asymptotically stable equilibrium

(iv) $a < 0$, $b < 0$. When $\phi < 0$, with $|\phi| \ll 1$, $x = 0$ is unstable; when $0 < \phi \ll 1$, $x = 0$ is locally asymptotically stable and there exist a positive unstable equilibrium.

(v) $a > 0$, $b < 0$. When $\phi < 0$, with $|\phi| \ll 1$, $x = 0$ is unstable and there exist a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, $x = 0$ is stable and a positive unstable equilibrium appears

(vi) $a < 0$, $b > 0$. When ϕ changes from negative to positive, $x = 0$ changes its stability from stable to unstable. Correspondingly, a unstable equilibrium becomes positive and locally asymptotically stable.

The proof of theorem A.1 is found in [12].

2. Pontryagin's Maximum Principle

Theorem A.2: The necessary conditions that $\left(x_0{}^*, u^*(t)\right)$ be an optimal initial condition and optimal control for the optimal control problem are the existence of a non-zero k-dimensional vector λ with $\lambda_1 \leq 0$ and an n-dimensional vector function $P(t)$ such that for $t \in [t_0, t_1]$:

(i) $P(t)' = -P(t)' f_x\left(t, x^*(t), u^*(t)\right)$; for $t \in (t_0, t_1)$ and $u \in U$

(ii) $P(t)' \left[f(t, x^*(t), u) - f\left(t, x^*(t), u^*(t)\right) \right] \leq 0$;

(iii) $P(t_1)' = \lambda' \phi_{x_1}(e)$;

(iv) $P(t_0)' = -\lambda' \phi_{x_0}(e)$;

(v) $P(t_1)' f\left(t_1, x^*(t_1), u^*(t_1)\right) = -\lambda' \phi_{t_1}(e)$;

(vi) $P(t_0)' f\left(t_0, x^*(t_0), u^*(t_0)\right) = \lambda' \phi_{t_0}(e)$;

If $f(t, x, u)$ has a continuous partial derivative $f_t(t, x, u)$, then the condition

(vii) $P(t)' f\left(t, x^*(t), u^*(t)\right) = \lambda' \phi_{t_0}\left(t_0, t_1, x^*(t_0), x^*(t_1)\right) + \int_{t_0}^{t} P(s)' f_t\left(s, x^*(s), u^*(s)\right) ds$

holds for each $t \in [t_0, t_1]$.

The prove of theorem A.2 can be found in [15]

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