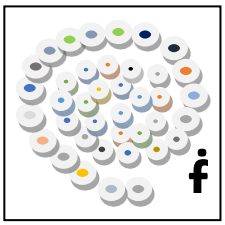

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On the reproduction number of a model for the transmission dynamics of Malaria and Pneumonia co-infection with mass action incidence

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Abstract

Pneumonia and malaria are two of the world's most lethal diseases. The geographic overlap of these diseases in Sub-Saharan Africa facilitates co-infection. The effect of the basic reproduction number on the transmission dynamics of malaria and pneumonia co-infection is investigated in this study. The co-infection model for malaria and pneumonia revealed that there is a relationship between the reproduction number for malaria and pneumonia. The study also discovered that pneumonia disease can aggravate malaria in the population. Considering the global stability of the model, the disease free equilibrium (DFE) account for co-infected persons must be less than unity to ensure global stability of the model.

Keywords: malaria; malaria-pneumonia; disease-free equilibrium; global stability; basic reproduction number; pneumonia

1 Introduction

Mathematical models of infectious diseases allow us to deduce the state and progression of an outbreak based on the information available. Although the burden of infectious diseases has surpassed the burden of chronic diseases and psychiatric illnesses in many countries, infectious diseases and health conditions continue to cause morbidity and mortality in many countries and communities. Malaria, measles, hepatitis B, cholera, and pneumonia epidemics are characterized in Sub-Saharan Africa owing to poor hygiene, sanitation, malnutrition, and perinatal conditions, with poverty being the most visible feature of disease spread in the region [20, 18, 17].

Malaria is a febrile illness caused by Plasmodium parasites that are transmitted to humans through mosquito bites from infected female Anopheles mosquitoes [3]. Human malaria is caused by five parasitic species, of which Plasmodium falciparum is the deadliest and most common on the African continent. According to the most recent WHO report, approximately 241 million cases of malaria were recorded in 2020, a 24 percent increase over the previous year, resulting in a 35 percent increase in deaths compared to the previous year [24].

Pneumonia is the leading cause of respiratory morbidity in over two million children under the age of five. It's a lung infection brought on by bacteria, viruses, fungi, or other pathogens [10]. Streptococcus pneumoniae, also known as pneumococcus, is the most common cause. Pneumococcus spreads through micro aspiration of oropharyngeal organisms and inhalation of aerosols. Children become severely ill with high fevers and rapid breathing. Infants usually suffer from convulsions, unconsciousness, hypothermia, lethargy, and feeding problems [10, 12,

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21]. Fungal pneumonia is uncommon, but it can occur in people who have weakened immune systems due to AIDS, immuno-suppressive drugs, or other medical issues [7]. Infections caused by parasites typically enter the body through the skin or mouth and travel through the body to the lungs, usually through the blood [23]. Some people are more susceptible to pneumonia than others. Infection is especially dangerous for people over the age of 65, infants, and children under the age of five [6]. People with weakened immune systems are more likely to become infected with cytomegalovirus (CMV) [8].

Pneumonia and malaria are two of the world's most lethal diseases. In Sub-Saharan Africa, the geographic overlap of these diseases facilitates co-infection. Oluyo and Adeniyi [15] showed the consequences of co-infection of pneumonia and malaria parasites and that their combination leads to worse outcomes, posing a major public health concern.

1.1 Related works

Lawi et al. [11] presented and analyzed a deterministic model of malaria and pneumonia dynamics. The findings revealed that increasing the treatment rate leads to a reduction in new disease incidences. Due to increased mobility, a decrease in pneumonia cases would result in an increase in malaria cases. A study by Niger and Gumel [13] examined the role of repeated exposure on the transmission dynamics of malaria in a human population using a deterministic model. The study revealed that when the associated reproduction threshold is less than unity, a stable disease-free equilibrium coexists with a stable endemic equilibrium. According to a thorough qualitative analysis of the model, which includes three immunity stages and the absence of reinfection, it was shown that when the reproduction threshold exceeds unity, mass action incidence has a globally asymptotically stable endemic equilibrium. Olaniyi and Obabiyi [16] developed a model considering the transmission of Plasmodium falciparum malaria between humans and mosquitoes using a seven-dimensional ordinary differential equation with non-linear forces of infection in the form of saturated incidence rates. It was shown that antibodies were produced in both human and mosquito populations in response to the presence of parasite-causing malaria. Consequentially, humans must increase their antibody production in order to combat parasite invasion in the bloodstream. Also, an individual's immunity state, that is, the infected person's general health and nutritional status, is a factor in preventing or aiding the occurrence of malaria. Under different levels of sanitation strategy, a deterministic mathematical model for malaria was considered in [19]. According to the study,the results revealed that the malaria model has a backward bifurcation, implying that malaria eradication requires more than just lowering $Rm < 1$, and that other factors such as sanitation must be considered and maintained, better sanitation reduces the number of mosquito bites and the rate of malaria transmission. Oluyo and Adeniyi [15] conducted a study on a thorough mathematical analysis of the malaria-pneumonia co-infection model. The local and global stabilities of the malaria only, pneumonia only, and full malaria-pneumonia models were investigated. Their result showed that the malaria only model exhibits a backward bifurcation, implying that malaria will be cleared from the population if a critical threshold is met, while the pneumonia only model reveals that pneumonia disease is cleared from the population if a critical threshold is met. Using a nonlinear mathematical model,[14] proposed and analyzed the transmission dynamics of malaria disease in a population, the model was qualitatively evaluated to incorporate time-dependent variable controls, with the goal of reducing the spread of malaria disease.

This paper is organized as follows; the section 2 entails the mathematical formulation of the malaria and pneumonia co-infection model. In Section 3, the mathematical analysis of the model which is sub sectioned into section 3.1 as the feasible region and well-posedness of the model, section 3.2, the equilibrium analysis of the model, the basic reproduction number of the model was shown in section 3.3,the stability analysis of the malaria and pneumonia co-infection was shown in section 3.4. Section 4, present numerical simulation, discussion of result and concluding remarks are presented in Section 5.

2 Mathematical formulation

The development of the malaria and pneumonia co-infection model in the host(human) and vector(mosquito) populations starts with dividing the total human-mosquito population into two distinct compartments written as $N_h(t)$ and $N_m(t)$, representing the total population sizes for humans and mosquitoes at a given time t, respectively. Consequently, within the scope of this discussion from this point on,the use of the time t dependency is suppressed. Following the modification of the work of Lawi et al [11], Oluyo and Adeniyi [15] studied a mathematical model on the co-infection of malaria and pneumonia with mass action incidence. In this study, the human population will be decomposed further into the following seven epidemiological compartments namely: susceptible humans S_h, exposed humans to malaria E_h, exposed humans to pneumonia E_p, exposed humans to both malaria and pneumonia E_{hp}, infected humans with malaria I_h, infected humans with malaria I_h, infected humans with pneumonia I_p and infected humans with both malaria and pneumonia I_{hp} such that $N_h = S_h + E_h + E_p + E_{hp} + I_h + I_p + I_{hp}$. For clarity of exposition, the states variables are identified with their corresponding class. For example, taking into consideration human from the class E_h, it is considered that E_h is a function and not a class. In general, the vector population is sub-divided the population into three classes: susceptible mosquito S_m, exposed mosquito E_m and infected mosquito I_m. Thus, the total mosquito population is $N_m = S_m + E_m + I_m$.

According to Filipe et al [9], the rate of infection between human and mosquito populations depend on many factors such as biting rate of the mosquito α (i.e. the number of bites per mosquito) and the transmission rate β_{mh}. The force of malaria infection for humans (i.e. from mosquitoes to humans)is defined as the product of the average number of bites per human α and the transmission rate β_{mh} from a mosquito in the I_m class to a susceptible humans in S_h class i.e.,

$$\lambda_h = \alpha\beta_{mh}I_m. \tag{1}$$

Similarly, the force of infection for mosquitoes (i.e. from human to mosquito) is given as

$$\lambda_m = \alpha\beta_{hm}(I_h + \delta I_{hp}), \tag{2}$$

where β_{hm} is the transmission rate from an infected human in the I_h or I_{hp} class to a susceptible mosquito in S_m class with δ defined as a modification parameter that accounts for the infectiousness of co-infected individuals in comparison to their counterparts, such that $0 < \delta < 1$. Force of infection for pneumonia denoted by λ_p is defined as the contact rate per human C, the transmission rate of pneumonia from a human in I_p or I_{hp} class to a susceptible human in $S_h$class i.e.

$$\lambda_p = \beta_{hm}C(I_p + gI_{hp}), \tag{3}$$

where g denote the modification parameter for the co-infected individual's relative infectiousness in comparison to their counterparts such that $0 < g < 1$.

From these assumptions a flow diagram is drawn which is summarized using a system of nonlinear ordinary differential equations as follows:

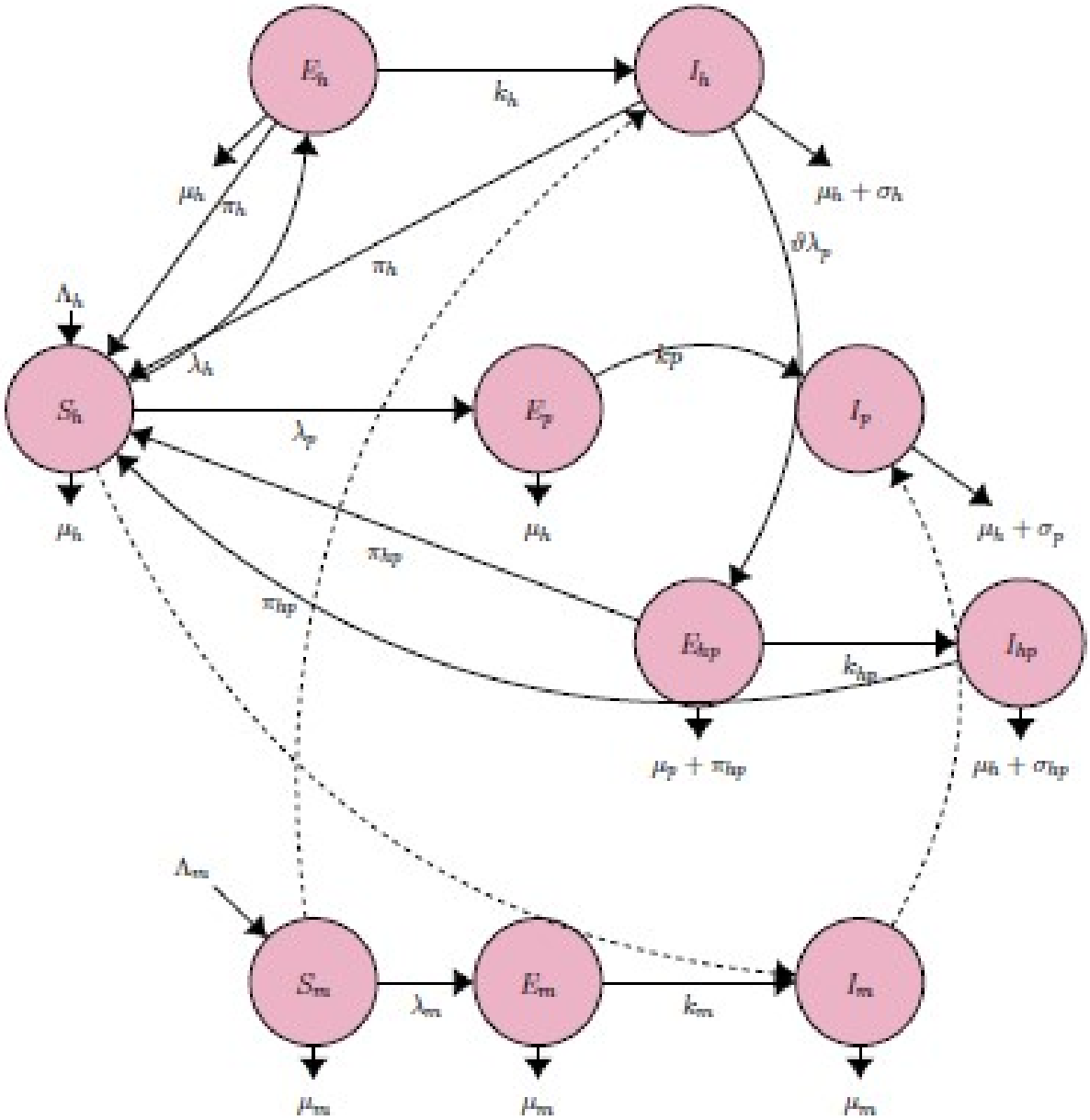

Figure 1: Model flow diagram

$$
\begin{aligned}
S_h^{'} &= \Lambda_h - \lambda_h S_h - \lambda_p S_h - \mu_h S_h + \pi_h E_h + \pi_p E_p + \pi_{hp} E_{hp} + \pi_h I_h + \pi_p I_p + \pi_{hp} I_{hp},\\
E_h^{'} &= \lambda_h S_h - A_0 E_h,\\
I_h^{'} &= k_h E_h - \vartheta \lambda_p I_h - A_1 I_h,\\
E_p^{'} &= \lambda_p S_h - A_2 E_p,\\
I_p^{'} &= k_p E_p - \varepsilon \lambda_h I_p - A_3 I_p,\\
E_{hp}^{'} &= \vartheta \lambda_p I_h + \varepsilon \lambda_h I_p - A_4 E_{hp},\\
I_{hp}^{'} &= k_{hp} E_{hp} - A_5 I_{hp},\\
S_m^{'} &= \Lambda_m - \lambda_m S_m - \mu_m S_m,\\
E_m^{'} &= \lambda_m S_m - A_6 E_m,\\
I_m^{'}(t) &= k_m E_m - \mu_m I_m,\\
&\Big(S_{h0}, E_{h0}, I_{h0}, E_{p0}, I_{p0}, E_{hp0}, I_{hp0}, S_{m0}, E_{m0}, I_{m0}\Big)^T,
\end{aligned}
\tag{4}
$$

where $A_0 = (k_h + \pi_h + \mu_h)$, $A_1 = (\sigma_h + \pi_h + \mu_h)$, $A_2 = (k_p + \pi_p + \mu_h)$, $A_3 = (\sigma_p + \pi_p + \mu_h)$, $A_4 = (k_{hp} + \pi_{hp} + \mu_h)$, $A_5 = (\sigma_{hp} + \pi_{hp} + \mu_h)$, $A_6 = (k_m + \mu_m)$.

For convenience, the model parameter descriptions is shown in Table 1 . Further, the following assumptions are made:

(i) The model's parameters are all strictly positive.

(ii) This model assumes that all individuals in the population are equally likely to become infected if they come into effective contact with an infectious individual or infectious mosquito, and that infection transmission occurs at a mass action incidence rate.

(iii) It is assumed that an infected individual will recover with no permanent immunity and revert to the susceptible class.

(iv) Since birth, immigration, emigration, and death occur in the population, it is assumed that human populations are not constant.

(v) Individuals exposed to malaria as well as those exposed to pneumonia have a less than one in a million chance of survival until the infectious state.

(vi) The chance of a mosquito exposed to malaria surviving until the infectious state is less than or equal to one.

(vii) The exposed class of human and mosquito in the population is therefore included as a result of assumptions (v) and (vi) above.

Variable and Parameter Descriptions	
S_h	Susceptible class of human population to malaria
E_h	Human population exposed to malaria
I_h	Human population infectious with malaria
E_p	Human population exposed to pneumonia
I_p	Human population infectious with pneumonia
E_{hp}	Human population exposed to both malaria and pneumonia
I_{hp}	Human population that is infectious with malaria and Pneumonia
S_m	Susceptible class of vector population to malaria
E_m	Vector population exposed to malaria
I_m	Infectious class of the vector population
Λ_h	Constant per capita recruitment rate into susceptible human population
Λ_m	Constant per capita recruitment rate into susceptible vector population
μ_h	Natural death rate of human population
μ_m	Natural death rate of vector population
ϑ	Modification parameter according to the increased susceptibility to infection with Pneumonia
σ_h	Malaria induced mortality rate
σ_p	Pneumonia induced mortality rate
σ_{hp}	Malaria and pneumonia induced mortality rate
ε	The expected decrease in contact due to ill health as a result of pneumonia disease; $0 < \varepsilon < 1$
π_h	Rate of recovery from malaria to the susceptible class
π_p	Rate of recovery from pneumonia to the susceptible class
π_{hp}	Rate of recovery from malaria and pneumonia to the susceptible class
λ_h	Force of malaria infection for humans
λ_m	Force of infection for mosquitoes
λ_p	Transmission rate for pneumonia
k_h	The per capita rate of progression of humans from the exposed class E_h to the infectious class I_h.
k_p	The per capita rate of progression of humans from the exposed class E_p to the infectious class I_p.
k_{hp}	The per capita rate of progression of humans from the exposed class E_{hp} to the infectious class I_{hp}.
k_m	The per capita rate of progression of mosquitoes from the exposed class E_m to the infectious class I_m.

Table 1: Parameter description

3 Mathematical analysis of the model

3.1 Feasible Region and Well-posedness of the Model

In this section, the feasibility and well-posedness of the model are studied using the approach in [2].

Suppose system (4) can be rewritten compactly as

$$\begin{aligned} X^{'}(t) &= \phi\Big(X(t)\Big), \\ X(0) &= X_0. \end{aligned} \tag{5}$$

It is assumed that $\phi \in \mathbf{C}^{'}$ provides sufficient conditions to ensure that system (5) is well-posed; however, because the concern is with the model's stability near equilibrium points, additional assumptions on ϕ are required in order to invoke a variation of the centre manifold theorem, which was proven by Castillo-Chavez, 2004 [4]] . Consequently, it is assumed that ϕ is twice continuously differentiable i.e. $\phi \in \mathbf{C^2} \subset \mathbf{C}^{'}$. In addition, the functions under investigation should have a bounded first derivative $\mathbf{C_B^{'}}\big(\Re_+^{\mathbf{10}}\big)$ for system (5) to be feasible in an epidemiological sense.

Theorem 3.1 *(Feasible region of the model)*
Suppose $\Big(S_h, E_h, I_h, E_p, I_p, E_{hp}, I_{hp}, S_m, E_m, I_m\Big)^T$ *with initial data* $X(0) \geq 0$*, then, the solutions* x *of the malaria and pneumonia model* (5) *will remain non-negative for all time* $t > 0$*. Further, define the following region of attraction* Γ_{hm} *given by*
$\Gamma_{hm} = \Big\{x \in \mathbf{C_B^{'}} \cap \mathbf{C_B^{'}}\Big(\Re_+^{\mathbf{10}}\Big) : \mathbf{\Gamma_h} \times \mathbf{\Gamma_m} \subset \Re_+^{\mathbf{7}} \times \Re_+^{\mathbf{3}}\Big\}$,
where $\Gamma_h = \Big\{\Big(S_h, E_h, I_h, E_p, I_p, E_{hp}, I_{hp}, S_m, E_m, I_m\Big) : S_h + E_h + I_h + E_p + I_p + E_{hp} + I_{hp} \leq \frac{\Lambda_h}{\mu_h}\Big\}$,
and $\Gamma_m = \Big\{\Big(S_m, E_m, I_m\Big) : S_m + E_m + I_m \leq \frac{\Lambda_m}{\mu_m}\Big\}$
attracts all solution initiating in the interior positive invariant.

The proof of Theorem 1 follows from [15].

3.2 Equilibrium analysis of the model

The feasibility of all equilibrium points of the model (4) are considered in this section. The equilibrium points of system (4) are solutions of the dynamical system (4) obtained by equating the first derivative of all system variables to zero. In particular, for the malaria and pneumonia model in (4), the following feasible steady states are obtained:

(i) **Malaria and pneumonia free equilibrium** $(MPFE)$ denoted by ξ_0, this represent the solutions of the dynamical variable of system (4) corresponding to the case where there is no presence of both malaria and pneumonia diseases in the population. The $(MPFE)$, ξ_0 is feasible if the associated basic reproduction number for malaria and pneumonia diseases denoted by R_{hm} and R_p respectively are both less than unity or otherwise. Thus,

$$\xi_0 = \Big(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0\Big), \tag{6}$$

where $R_{hm} = \sqrt{R_h R_m}$, $R_h = \frac{\alpha\beta_{mh}k_h\Lambda_h}{\mu_h A_0 A_1}$, $R_m = \frac{\alpha\beta_{hm}k_m\Lambda_m}{\mu_m^2 A_6}$ and $R_p = \frac{\Lambda_h k_p \beta_p C}{\mu_h A_2 A_3}$ respectively. The co-infection reproduction number denoted by R_c is defined to be

$$R_c = max\{R_{hm}, R_p\}. \tag{7}$$

A detailed insight and explanation on the basic reproduction number is considered in section 3.3.

(ii) **Malaria free endemic equilibrium** $(MFEE)$ written as ξ_1. $(MFEE)$, ξ_1 depicts the solution of (4) corresponding to the case where there is absence of malaria disease but there is presence of pneumonia disease in the population. This scenario is feasible if the basic reproduction for pneumonia infection R_p is greater than unity. Then,

$$\xi_1 = \Big(S_{h1}, 0, 0, E_{p1}, I_{p1}, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0\Big). \tag{8}$$

(iii) **Pneumonia free endemic equilibrium** $(PFEE)$ denoted by ξ_2, is a situation in the population representing the absence of pneumonia but persistence of malaria disease. This equilibrium is feasible if $R_{hm} > 1$. Thus,

$$\xi_2 = \Big(S_{h2}, E_{h2}, I_{h2}, 0, 0, 0, 0, S_{m2}, E_{m2}, I_{m2}\Big). \tag{9}$$

(iv) **Malaria and Pneumonia endemic equilibrium** $(MPEE)$ written as ξ^* is a scenario where both malaria and pneumonia diseases persist in the population. ξ^* is feasible if both $R_{hm} > 1$ and $R_p > 1$. We define

$$\xi^* = \Big(S_h^*, E_h^*, I_h^*, E_p^*, I_p^*, E_{hp}^*, I_{hp}^*, S_m^*, E_m^*, I_m^*\Big). \tag{10}$$

Remark 3.1 *The detailed calculations on how the equilibrium points in (6), (8), (9), and (10) were derived are provided in Appendix A.*

3.3 The basic reproduction number

The basic reproduction number R_{hm} and R_p are important dimensionless threshold in epidemiology. They are used to predict whether a disease goes to extinction or persist in the affected population. According to [5], the term basic reproduction number, R_{hm} is defined as the average number of secondary infections produced by a typical single infection in a completely susceptible population. Particularly, if $R_{hm} > 1$ (or $R_{hm} < 1$), it means that on average, an infected human(mosquito) will produce more than (or less than) one secondary infected mosquito (human) during the whole infectious period in an entirely susceptible population and thus the disease will persist (or die out) in the population, respectively. R_h and R_m represent the dynamics of the model when only malaria disease is transmitted in the population through cross-infection of humans and mosquitoes in the absence of no control, while R_p captures the dynamics of the model when only pneumonia disease is transmitted in the population through contact between a susceptible and infected individual when no control is available. R_c describe the co-infection dynamics of the model when malaria and pneumonia are transmitted among people in the population. The basic reproduction number R_0 can be obtained by applying the next generation matrix technique [5, 25] and implemented in [1, 14].The derivation of the basic reproduction numbers R_{hm} and R_p are shown in the Appendix.

A deep insight is sought to understand the qualitative information embedded in the basic reproduction number. The basic reproduction numbers R_{hm} and R_p is decomposed further to obtain information that are epidemiologically meaningful. From epidemiological point of view, it is of interest to have R_{hm} and R_p below unity. R_{hm} is decomposed to give

$$R_{hm} = \alpha\sqrt{\tfrac{\Lambda_h}{\mu_h}}\sqrt{\frac{\beta_{mh}}{\Big(\sigma_h+\pi_h+\mu_h\Big)}}\sqrt{\frac{k_h}{\Big(k_h+\pi_h+\mu_h\Big)}}\sqrt{\frac{k_m}{\Big(k_m+\mu_m\Big)}}\sqrt{\tfrac{\beta_{hm}}{\mu_m}}\sqrt{\tfrac{\Lambda_m}{\mu_m}},$$

$$R_{hm} = \alpha\prod_{i=1}^{6}\sqrt{Q_i} \quad , \quad i = 1, 2, ...6. \tag{11}$$

Similarly,

$$R_p = C\frac{\Lambda_h}{\mu_h}\frac{k_p}{\left(k_p+\pi_h+\mu_h\right)}\frac{\beta_p}{\left(\sigma_p+\pi_h+\mu_h\right)},$$

$$R_p = C\prod_{j=1}^{3} q_j \quad , \quad i = 1, 2, 3. \tag{12}$$

The first factor of Eqn. 11, α, the biting rates of the *anopheles* mosquitoes. This is usually less than unity because the *anopheles* mosquito normally spread less than 100 *sporozoites* per bite [22].The size of each of the other factors in Eqn. 11 is analyzed as follows:

(i) The term $Q_1 = \frac{\Lambda_h}{\mu_h}$ refers to the ratio of the human recruitment rate to the natural death rate of humans. In normal settings, the human recruitment rate rank high than the natural death rate of humans on the sensitivity order of many epidemic models documented in the literature.

(ii) The term $Q_2 = \frac{\beta_{mh}}{\left(\sigma_h+\pi_h+\mu_h\right)}$ is the ratio of the transmission probability of the mosquito-to-human to the reciprocal of the sum of the malaria-induced death rate, recovery rate and natural death rate of humans. This quantity is always greater than zero monotonically decreasing with respect to the malaria induced death in humans plus the recovery rate and the natural death in humans. This is true if the probability of infection is less than unity while the term in the denominator of Q_2 increases. However, because increasing σ_h and μ_h is not practical in a real scenario, this particular term Q_2 provide no control over R_{hm}.

(iii) The term $Q_3 = \frac{k_h}{\left(k_h+\pi_h+\mu_h\right)}$ is the reciprocal of the host (human) progression rate to malaria infected class divided by itself and the sum of human recovery π_h and the death rates, μ_h. The upper bound of this term is one and is monotonically decreasing with respect to the sum of the human recovery π_h and mortality rates, μ_h. This fact is consistent if there are few humans exposed to the parasites to invade, then fewer malaria infections will arise. Since increasing μ_h is not realistic, Q_3 does not offer any control over R_{hm}.

(iv) The term $Q_4 = \frac{k_m}{\left(k_m+\mu_m\right)}$ depicts the reciprocal of the vector latent period k_m divided by itself plus the vector death rate, μ_m. As in Q_3, the quantity Q_4 is bounded above, always less than unity and monotonically decreases with respect to the mosquito death rate μ_m. Hence, the higher the mosquito mortality rate, the smaller Q_4. Thus, Q_4 will offer realistic control over R_{hm} if the fatality rate of the mosquitoes can be increased.

(v) The term $Q_5 = \frac{\beta_{hm}}{\mu_m}$ is the ratio of the transmission probabilities of human-to-mosquito to the mosquito natural death rate. Since the mosquito natural death rate μ_m is always smaller compared to the transmission probability of malaria from infected human to a susceptible mosquito, then Q_5 is always greater than unity, hence this term will increase R_{hm}.

(vi) Q_6 is the ratio of the mosquito recruitment rate to mosquito mortality rate μ_m. From other studies, $\Lambda_m > \mu_m$, thus Q_6 will have a positive effect on R_{hm}.

R_p is decomposed and analyzed as follows:

The parameter C, the contact rate between susceptible humans and infected human with pneumonia. This is normally less than unity from literature. The other factors are considered as follows:

(i) q_1 is the same as Q_1, hence the result follows.

(ii) q_2 is the term $q_2 = \frac{k_p}{\left(k_p+\pi_p+\mu_h\right)}$ representing the reciprocal of the human progression rate to pneumonia infected class divided by itself and the sum of human recovery from pneumonia π_p and the human natural death rate μ_h. Clearly, 1 is the upper bound of q_2 and is monotonically decreasing with respect to the sum of the human recovery rate from pneumonia and the fatality rate μ_h. Indeed, this is the case when there are few humans exposed to the bacteria invasion which may lead to few pneumonia infections cases. Further, since increasing μ_h is not practical in a real life situation, q_2 control over R_p is insignificant.

(iii) $q_3\frac{\beta_p}{\left(\sigma_p+\pi_p+\mu_h\right)}$ refer to the ratio of the pneumonia transmission probability to the reciprocal of the sum of the pneumonia induced deaths in humans, pneumonia recovery rate and natural death rate of humans. As in the case of Q_2, q_3 is always greater than zero and monotonically decreasing with respect to the pneumonia-induced and natural death rates in humans. This is the case whenever the probability of pneumonia infection is less than unity while the reciprocal of q_3 has no significant control on R_p.

By combining Eqns. (3.7) and (3.8), it can be shown that the basic reproduction number for malaria infection, R_{hm} and pneumonia infection, R_p is related by

$$R_{hm} = Q^{\star}\sqrt{R_p}, \tag{13}$$

where $Q^{\star} = \sqrt{\frac{\alpha^2 Q_2 Q_3 Q_4 Q_5 Q_6}{C q_2 q_3}}$.

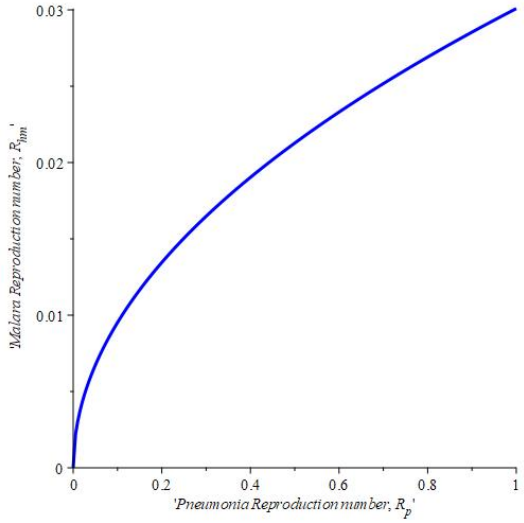

Figure 2: The graph of the Malaria Reproduction number R_{hm} against Pneumonia Reproduction number R_p

It can be seen from Figure 2 that increase in the basic reproduction number for pneumonia, R_p will increase the basic reproduction number for malaria. This implies that increase in pneumonia can induce malaria. In Figure 3, the contour plot showing the relationship between R_{hm} and R_p is shown.

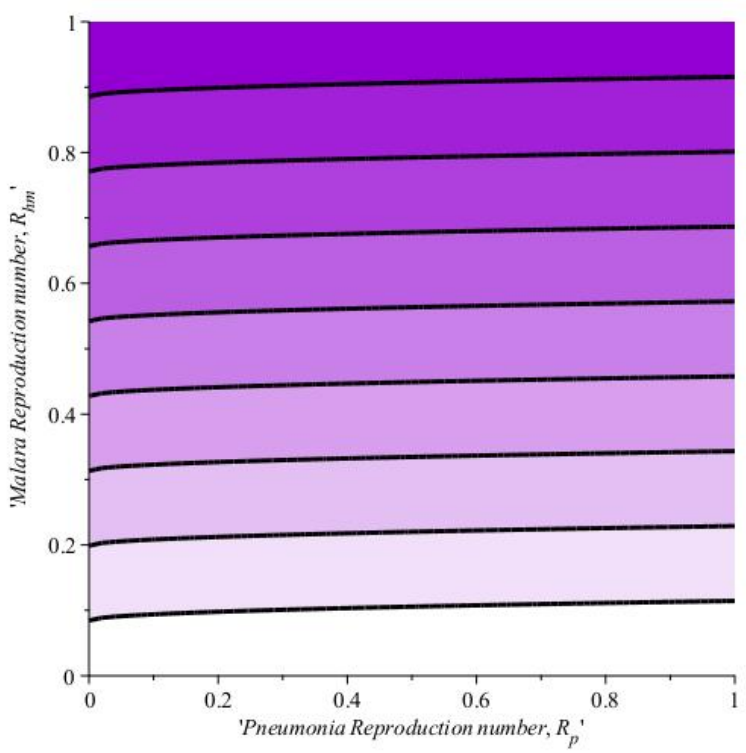

Figure 3: Contour plot of the malaria and pneumonia co-infection

Eqn. (3.7) is re-emphasized on as follows: First, if the biting rate of the mosquitoes $\alpha = 0$, it follows that $R_{hm} = 0$ as the quantity totally nullify R_{hm} which give rise to the following epidemiological scenario:

1. Humans are not bitten by mosquitoes and
2. Mosquitoes are not transmitting the disease

Further, suppose the transmission probability from human-to-mosquito β_{hm} is equal to zero, then R_{hm} is zero meaning that infected humans are not transmitting the malaria disease to susceptible mosquitoes. However, if certain quantities such as the death rate of the vector, μ_m and human death rate μ_h are zero then R_{hm} is undefined. Realistically, μ_h and μ_m cannot be zero because all mosquitoes and humans experience death at a stage in their life cycle. The study on control strategies will form the foundation of a separate research.

3.4 Stability analysis of the model

In the following section, the local asymptotic stability (LAS) of ξ_0, ξ_1 and ξ_2 is computed through the following theorem:

Theorem 3.2 *Consider the model system* (2.4)*, then the following holds:*

(i) The malaria and pneumonia free equilibrium, ξ_0 is feasible and locally asymptotically stable if $R_{hm} < 1$ and $R_p < 1$ otherwise unstable.

(ii) The malaria-free endemic equilibrium, ξ_1 is feasible and locally asymptotically stable if $R_p > 1$.

(iii) The pneumonia-free endemic equilibrium, ξ_2 is feasible and locally asymptotically stable if $R_{hm} > 1$.

The proof to Theorem 3.2 is given in Appendix A.

Parameter	Values	Source
Λ_h	0.071	[15]
Λ_m	0.00011	[15]
μ_h	0.04	[15]
μ_m	0.0000457	[15]
ϑ	0.5	[15]
σ_p, σ_h	0.01	[15]
σ_{hp}	0.1	[15]
ε	0.1	Estimated
π_p ,π_h	0.1	[15]
k_{hp},k_p	0.5	[15]
α,π,τ	0.5	Estimated

Table 2: Parameter values

3.5 Global stability analysis of the Malaria and Pneumonia co-infection free equilibrium, ξ_0

Theorem 2 (i) suggest that the malaria and pneumonia co-infection free equilibrium ξ_0 is locally asymptotically stable if R_{hm} and $R_p$holds, however, this does not capture the co-infection scenario in details. It is therefore important to consider the global asymptotic stability (GAS). To do this, the global asymptotic stability analysis is motivated through the following theorem:

Theorem 3.3 *The malaria and pneumonia co-infection free equilibrium ξ_0 is globally asymptotically stable if ξ_0 is locally asymptotically stable and $R_{mp} \leq 1$, where R_{mp} is the co-infection threshold defined as*

$R_{mp} = \frac{k_{hp}}{A_4 A_5}\Big(A_3 g R_p + A_1 \delta\Big)$.

The proof of Theorem 3.3 is provided in the Appendix B.

Remark 3.2 *It can be deduced that the global stability of ξ_0 depends on R_{mp}. Indeed, R_{mp} take into account the average number of secondary infections arising from co-infectious individual with pneumonia plus malaria infected humans.*

3.6 Endemic equilibrium point and bifurcation analysis

When a disease persist in the population in the course of time, then an endemic equilibrium occurs. The persistence of a disease depend on a threshold called the reproduction number R_0 whether it is less or greater than unity. Recent studies, [[2]] has revealed that for disease eradication, bringing $R_0 < 1$ may not be sufficient but may also depend on other parameters.The endemic equilibrium usually arise from other studies in the literature when $R_0 > 1$, however, there are situations where the endemic equilibrium co-exist with the disease free equilibrium for $R_0 < 1$,this phenomenon is called bifurcation. Bifurcation can be either forward (super-critical) or backward(sub-critical) which have serious implications in epidemiology. A forward bifurcation is shown when $R_0 > 1$ from below, and looses its stability so that a small positive super-threshold equilibrium that is asymptotically stable, then the disease free equilibrium become unstable. However, when $R_0 < 1$ and a small positive sub-threshold equilibrium appears, and the disease free equilibrium together with a larger positive equilibrium are locally asymptotically stable.

Considering the components of the endemic equilibrium $\xi^\star$, it is desirable to study in details the stability of the endemic equilibrium around the malaria and pneumonia co-infection

free equilibrium using bifurcation analysis through the well known Centre Manifold Theorem discussed in [[4]]. In the following, the bifurcation analysis of the malaria and pneumonia co-infection model is studied. Particularly, the following theorem is proven to study the bifurcation behaviour of the model:

Theorem 3.4 *(Bifurcation analysis for the malaria and pneumonia co-infection model) Let* $R_{hp} = 1$ *and the positive quantities a and b defined as follows*

$$a = -\frac{2\nu_4\left(\mu_h^2+(k_p+\pi_p+\sigma_p)\mu_h+k_p\sigma_p\right)r_5^2A_2A_3\left(gA_2A_3k_{hp}\vartheta+A_4A_5k_p\right)}{\Lambda_h k_p^3A_4A_5}, \quad b = \frac{\nu_4r_5\beta_p\Lambda_h\left(gA_2A_3k_{hp}\vartheta+A_4A_5k_p\right)}{A_4A_5k_p\mu_h},$$

then the malaria and pneumonia model exhibits a forward bifurcation if $a<0$ *and* $b>0$.

The proof of Theorem 3.4 can be found in Appendix C.

4 Discussion and conclusion

In this work, the effect of the basic reproduction number'R_0' on the transmission dynamics of malaria and pneumonia co-infection is studied. The malaria and pneumonia co-infection model (2.4) revealed that there exist a relationship between the reproduction number for malaria (R_{hm}) and reproduction number for pneumonia (R_p) given by (3.9). The study further revealed that pneumonia disease may induce malaria disease in the population. Considering the global stability of the Disease Free Equilibrium (DFE) of the model, the threshold (R_{mp}) that account for the co-infected persons is required to be less than unity to guarantee global stability of the model. A short summary highlighting the conclusions of this work is given below:

1. In section 3.1, the mathematical and epidemiological wellposedness of the model was shown given that the initial conditions are satisfied. Further, in Theorem 1, a feasible region Γ_{hm} that is mathematically and epidemiologically suitable was provided.

2. In Section 3.2, the model equilibrium points were obtained.

3. In Section 3.3, the basic reproduction number for malaria (R_{hm}) and (R_p) and the co-infection reproduction number (R_c) were discussed in details by decomposing them into components in order to carefully analyze the size contribution of each component involved. The analysis revealed that increasing the mosquito mortality rate (μ_m) will offer realistic control over R_{hm}. Also, the analyses revealed that the transmission probabilities of human-to-mosquito, β_{hm} and the recruitment rate of mosquitoes (Λ_m) will increase the size of the reproduction number.

4. In Section 3.4, stability analysis of the model were discussed. Precisely, in Theorem 2 conditions for the local stability of ξ_0, ξ_1 and ξ_2 using the associated basic reproduction number was established.

5. In Section 3.5, the global stability of the malaria and pneumonia free equilibrium, ξ_0 using the method of Lyapunov function was established. Theorem 3 also provide that for the global stability to be established, the co-infection threshold R_{mp} must be less or equal to unity.

6. In Section 3.6, the Centre Manifold Theorem was used to study the stability of the endemic equilibrium around the malaria and pneumonia co-infection free equilibrium. The analysis revealed that the co-infection model exhibit a transcritical (forward) bifurcation.

As a guide for future research, it will be of interest to study the quantities that are related to control measures that can effectively increase mosquito fatality, reduce mosquito recruitment while also reducing the transmission rates of malaria and pneumonia.

Conclusively, the malaria and pneumonia co-infection model has provided us with a mathematical understanding the relationship between the basic reproduction numbers for malaria and pneumonia transmission dynamics.

Appendix A. Feasibility of equilibrium points

The feasibility of $MPFE$, ξ_0 is trivial. The next focus is on the feasibility of ξ_1, ξ_2 and $\xi^\star$

Feasibility of ξ_1

Consider the case when $\lambda_h = \lambda_m = 0$ in (4), so that the following equation are obtained at equilibrium:

$$\Lambda_h - \beta_p C I_p S_h - \mu_h S_h + \pi_p E_p + \pi_p I_p = 0, \tag{A.1}$$

$$\beta_p C I_p S_h - A_2 E_p = 0, \tag{A.2}$$

$$k_p E_p - A_3 I_p = 0, \tag{A.3}$$

$$\Lambda_m - \mu_m S_m = 0. \tag{A.4}$$

It is clear from $(A.4)$ that $S_{m1} = \frac{\Lambda_m}{\mu_m}$. This is the case as there are no malaria infection in the population. Thus, the susceptible vectors are not infected and pose no malaria threat. From eqn. $(A.3)$,

$$E_p = \frac{A_3}{k_p} I_p. \tag{A.5}$$

Using $(A.5)$ in $(A.1)$ yield

$$S_h = \frac{k_p \Lambda_h + \pi_p\big(k_p + A_3\big) I_p}{k_p\Big(\beta_p C I_p + \mu_h\Big)}. \tag{A.6}$$

Taking into account of $(A.5)$ and $(A.6)$ in $(A.2)$,

$$f(I_p) = \Lambda_h k_p \beta_p C - \mu_h A_2 A_3 - \beta_p C\Big(\sigma_p\big(k_p + \mu_h\big) + \mu_h A_2\Big) I_p = 0. \tag{A.7}$$

Now,

$$\begin{aligned} f(0) =& \Lambda_h k_p \beta_p C - \mu_h A_2 A_3 \\ =& \mu_h A_2 A_3\Big(R_p - 1\Big) > 0 \quad if \quad R_p > 1. \end{aligned} \tag{A.8}$$

Further,

$$f(H_0) = -\mu_h A_2 A_3 < 0, \tag{A.9}$$

where $H_0 = \frac{\Lambda_h k_p}{\Big(\sigma_p\big(k_p + \mu_h\big) + \mu_h A_2\Big)}$. Also,

$$f^{'}(I_p) = -\beta_p C\Big(\sigma_p\big(k_p + \mu_h\big) + \mu_h A_2\Big) < 0. \tag{A.10}$$

Thus, $f(I_p)$ has a unique positive root $I_p = I_{p1}$ in the interval $(0, H_0)$ if $R_p > 1$. In particular, eqn $(A.7)$ yield

$$\begin{aligned} I_p = & I_{p1} = \frac{\Lambda_h k_p \beta_p C - \mu_h A_2 A_3}{\beta_p C\Big(\sigma_p\big(k_p + \mu_h\big) + \mu_h A_2\Big)} \\ = & \frac{\mu_h A_2 A_3 (R_p - 1)}{\beta_p C\Big(\sigma_p\big(k_p + \mu_h\big) + \mu_h A_2\Big)}. \end{aligned} \tag{A.11}$$

Using $(A.11)$ in $(A.5)$ yield

$$E_p = E_{p1} = \frac{\mu_h A_2 A_3^2 (R_p - 1)}{k_p \beta_p C\Big(\sigma_p\big(k_p + \mu_h\big) + \mu_h A_2\Big)}. \tag{A.12}$$

Further, using (3.17) in (3.12) yield

$$S_h = S_{h1} = \frac{k_p \Lambda_h + \pi_p\big(k_p + A_3\big) I_{p1}}{k_p\Big(\beta_p C I_{p1} + \mu_h\Big)}. \tag{A.13}$$

Thus, the malaria-free endemic equilibrium, ξ_1 is

$$\xi_1 = \Big(S_{h1}, 0, 0, E_{p1}, I_{p1}, 0, 0, S_{m1}, 0, 0\Big). \tag{A.14}$$

Feasibility of ξ_2

Suppose that $\lambda_p = 0$ which implies that $E_p = I_p = E_{hp} = I_{hp} = 0$ in (2.4), the following algebraic equations are obtained at equilibrium:

$$\Lambda_h - \alpha \beta_{mh} I_m S_h - \mu_h S_h + \pi_h E_h + \pi_h I_h = 0, \tag{A.15}$$

$$\alpha \beta_{mh} I_m S_h - A_0 E_h = 0, \tag{A.16}$$

$$k_h E_h - A_1 I_h = 0, \tag{A.17}$$

$$\Lambda_m - \alpha \beta_{hm} I_h S_m - \mu_m S_m = 0, \tag{A.18}$$

$$\alpha \beta_{hm} I_h S_m - A_6 E_m = 0, \tag{A.19}$$

$$k_m E_m - \mu_m I_m = 0. \tag{A.20}$$

From $(A.20)$

$$E_m = \frac{\mu_m I_m}{k_m}. \tag{A.21}$$

By adding $(A.18)$ and $(A.19)$ taking into account $(A.21)$ yield

$$S_m = \frac{k_m \Lambda_m - \mu_m A_6 I_m}{\mu_m k_m}. \tag{A.22}$$

Using $(A.21)$ and $(A.22)$ in $(A.19)$ while making I_h subject gives

$$\alpha \beta_{hm} I_h \left(\frac{k_m \Lambda_m - \mu_m A_6 I_m}{\mu_m k_m} \right) - \frac{A_6 \mu_m I_m}{k_m} = 0.$$

Thus

$$I_h = I_{h2} = \frac{\mu_m^2 A_6 I_m}{\alpha\beta_{hm}\big(k_m\Lambda_m - \mu_m A_6 I_m\big)}. \tag{A.23}$$

In view of $(A.23)$, Equation $(A.17)$ can be written as

$$E_h = E_{h2} = \frac{\mu_m^2 A_1 A_6 I_m}{\alpha k_h\beta_{hm}\big(k_m\Lambda_m - \mu_m A_6 I_m\big)}. \tag{A.24}$$

From $(A.15)$, the expression for S_h is

$$S_h = \frac{\Lambda_h + \pi_h E_h + \pi_h I_h}{\alpha\beta_{mh} I_m + \mu_h}. \tag{A.25}$$

The substitution of $(A.23)$ and $(A.24)$ in $(A.25)$, implies that

$$S_h = S_{h2} = \frac{\Lambda_h\alpha k_h\beta_{hm}\big(k_m\Lambda_m - \mu_m A_6 I_m\big) - \big(k_h + \mu_h\big)A_1 A_6\mu_m^2 I_m + k_h\pi_h\mu_m^2 A_6 I_m}{\mu_h\alpha k_h\beta_{hm}\big(k_m\Lambda_m - \mu_m A_6 I_m\big)}. \tag{A.26}$$

By substituting $(A.24)$ and $(A.26)$ in $(A.16)$ gives

$$\beta_{mh}\left[\frac{\Lambda_h\alpha k_h\beta_{hm}\big(k_m\Lambda_m - \mu_m A_6 I_m\big) - \big(k_h + \mu_h\big)A_1 A_6\mu_m^2 I_m + k_h\pi_h\mu_m^2 A_6 I_m}{\mu_h k_h\beta_{hm}\big(k_m\Lambda_m - \mu_m A_6 I_m\big)}\right] - \frac{A_0 A_1 A_6\mu_m^2}{\alpha k_h\beta_{hm}\big(k_m\Lambda_m - \mu_m A_6 I_m\big)} =$$

Further simplification of the above equation yield

$$\begin{aligned} f(I_m) &= \alpha^2\beta_{mh}\beta_{hm}k_m k_h\Lambda_m\Lambda_h - \mu_h\mu_m^2 A_0 A_1 A_6 - \mu_m\alpha A_6\beta_{mh}\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_3 + k_h\sigma_h + k_h\mu_h\big)\Big]I_m = 0 \\ &= \mu_h\mu_m^2 A_0 A_1 A_6\left(\frac{\alpha^2\beta_{mh}\beta_{hm}k_m k_h\Lambda_m\Lambda_h}{\mu_h\mu_m^2 A_0 A_1 A_6} - 1\right) - \mu_m\alpha A_6\beta_{mh}\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_3 + k_h\sigma_h + k_h\mu_h\big)\Big]I_m \\ &= \mu_h\mu_m^2 A_0 A_1 A_6\big(R_{hm}^2 - 1\big) - \mu_m\alpha A_6\beta_{mh}\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_3 + k_h\sigma_h + k_h\mu_h\big)\Big]I_m = 0. \end{aligned} \tag{A.27}$$

Then

$$f(0) = \mu_h\mu_m^2 A_0 A_1 A_6\big(R_{hm}^2 - 1\big) > 0 \quad if \quad R_{hm} > 1, \tag{A.28}$$

$$f(H_1) = \mu_h\mu_m^2 A_0 A_1 A_6 < 0, \tag{A.29}$$

where $H_1 = \dfrac{\mu_h\mu_m^2 A_0 A_1 R_{hm}^2}{\mu_m\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_1 + k_h\big(\sigma_h + \mu_h\big)\big)\Big]}$.

Also,

$$f^{'}(I_m) = -\mu_m\alpha\beta_{mh}A_6\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_1 + k_h\big(\sigma_h + \mu_h\big)\big)\Big] < 0. \tag{A.30}$$

Thus, $f(I_m)$ has a unique positive root $I_m = I_{m2}$ in $(0, H_1)$ if $R_{hm} > 1$. In particular, from $(A.27)$ we have that

$$I_m = \frac{\mu_h\mu_m A_0 A_1\big(R_{hm}^2 - 1\big)}{\alpha\beta_{mh}\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_1 + k_h\sigma_h + k_h\mu_h\big)\Big]}. \tag{A.31}$$

Substituting $(A.31)$ in $(A.21)$, yields

$$E_m = E_{m2} = \frac{\mu_h\mu_m^2 A_0 A_1\big(R_{hm}^2 - 1\big)}{k_m\alpha\beta_{mh}\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_1 + k_h\sigma_h + k_h\mu_h\big)\Big]}. \tag{A.32}$$

Further, from $(A.22)$ using $(A.31)$, S_m is non-negative if $R_{hm} > 1$. i.e.

$$\begin{aligned} S_m = S_{m2} =& \frac{1}{\mu_m k_m}\left[k_m\Lambda_m - \frac{\mu_h\mu_m^2 A_0 A_1 A_6\big(R_{hm}^2 - 1\big)}{\alpha\beta_{mh}\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_1 + k_h\sigma_h + k_h\mu_h\big)\Big]}\right] \\ =& \frac{\mu_h\mu_m^2 A_0 A_1 A_6 + \mu_h k_m\Lambda_m\big(\mu_h A_1 + k_h\sigma_h + k_h\mu_h\big)}{\mu_m k_m\alpha\beta_{mh}\Big[\alpha\Lambda_h k_h\beta_{hm} + \mu_m\big(\mu_h A_1 + k_h\sigma_h + k_h\mu_h\big)\Big]}. \end{aligned} \tag{A.33}$$

By using $(A.31)$ in $(A.23)$, $(A.24)$ and $(A.26)$ respectively gives

$$I_h = I_{h2} = \frac{\mu_h\mu_m^3 A_0 A_1 A_6\big(R_{hm}^2 - 1\big)}{\alpha\beta_{hm}\Big[\mu_h\mu_m^2 A_0 A_1 A_6 + \alpha^2\beta_{mh}\Lambda_m k_m\mu_m\big(\mu_h A_1 + k_h\sigma_h + k_h\mu_h\big)\Big]}; \tag{A.34}$$

$$E_h = E_{h2} = \frac{\mu_h\mu_m^3 A_0 A_1^2 A_6\big(R_{hm}^2 - 1\big)}{k_h\alpha\beta_{hm}\Big[\mu_h\mu_m^2 A_0 A_1 A_6 + \alpha^2\beta_{mh}\Lambda_m k_m\mu_m\big(\mu_h A_1 + k_h\sigma_h + k_h\mu_h\big)\Big]}; \tag{A.35}$$

and

$$S_h = S_{h2} = \frac{\Lambda_h + \pi_h E_{h2} + \pi_h I_{h2}}{\alpha\beta_{mh} I_{m2} + \mu_h}. \tag{A.36}$$

Thus, the pneumonia-free endemic equilibrium is feasible if $R_{hm} > 1$ i.e.

$$\xi_2 = \Big(S_{h2}, E_{h2}, I_{h2}, 0, 0, 0, 0, S_{m2}, E_{m2}, I_{m2}\Big). \tag{A.37}$$

Feasibility of $\xi^\star$

It is of note for this case that $\lambda_h > 0$, $\lambda_p > 0$ and $\lambda_m > 0$, implies that each of the state variables is non-negative. Thereafter, solving the full system of (2.4) as follows by equating the left-hand side of (2.4) to zero:

$$\Lambda_h - \lambda_h S_h - \lambda_p S_h - \mu_h S_h + \pi_h E_h + \pi_p E_p + \pi_{hp} E_{hp} + \pi_h I_h + \pi_p I_p + \pi_{hp} I_{hp} = 0, \tag{A.38}$$

$$\lambda_h S_h - A_0 E_h = 0, \tag{A.39}$$

$$k_h E_h - \vartheta\lambda_p I_h - A_1 I_h = 0, \tag{A.40}$$

$$\lambda_p S_h - A_2 E_p = 0, \tag{A.41}$$

$$k_pE_p - \varepsilon\lambda_h I_p - A_3 I_p = 0, \tag{A.42}$$

$$\vartheta\lambda_p I_h + \varepsilon\lambda_h I_p - A_4 E_{hp} = 0, \tag{A.43}$$

$$k_{hp}E_{hp} - A_5 I_{hp} = 0, \tag{A.44}$$

$$\Lambda_m - \lambda_m S_m - \mu_m S_m = 0, \tag{A.45}$$

$$\lambda_m S_m - A_6 E_m = 0, \tag{A.46}$$

$$k_m E_m - \mu_m I_m = 0. \tag{A.47}$$

From $(A.45)$,

$$S_m = S_m^\star = \frac{\Lambda_m}{\lambda_m + \mu_m}. \tag{A.48}$$

Using $(A.48)$ in $(A.46)$ to get

$$E_m = E_m^\star = \frac{\Lambda_m\lambda_m}{A_6\big(\lambda_m + \mu_m\big)}, \tag{A.49}$$

In view of $(A.47)$, I_m is obtained as

$$I_m = I_m^\star = \frac{k_m\Lambda_m\lambda_m}{\mu_m A_6\big(\lambda_m + \mu_m\big)}. \tag{A.50}$$

It can be seen from $(A.39)$ and $(A.41)$ that

$$E_h = \frac{\lambda_h}{A_0}S_h, \tag{A.51}$$

and

$$E_p = \frac{\lambda_p}{A_2}S_h. \tag{A.52}$$

The substitution of $(A.51)$ and $(A.52)$ in $(A.40)$ and $(A.42)$ respectively yields

$$I_h = \frac{k_h\lambda_h}{A_0\big(\vartheta\lambda_p + A_1\big)}S_h, \tag{A.53}$$

and

$$I_p = \frac{k_p\lambda_p}{A_2\big(\varepsilon\lambda_h + A_3\big)}S_h. \tag{A.54}$$

From $(A.43)$ using $(A.53)$ and $(A.54)$, gives

$$E_{hp} = \frac{\Big[k_h\vartheta A_2\big(\varepsilon\lambda_h + A_3\big) + k_p\varepsilon A_0\big(\vartheta\lambda_p + A_1\big)\Big]}{A_0A_2A_4\big(\varepsilon\lambda_h + A_3\big)\big(\vartheta\lambda_p + A_1\big)}\lambda_p\lambda_h S_h. \tag{A.55}$$

Using $(A.55)$ in $(A.44)$ to get

$$I_{hp} = \frac{k_{hp}\Big[k_h\vartheta A_2\big(\varepsilon\lambda_h + A_3\big) + k_p\varepsilon A_0\big(\vartheta\lambda_p + A_1\big)\Big]}{A_0A_2A_4A_5\big(\varepsilon\lambda_h + A_3\big)\big(\vartheta\lambda_p + A_1\big)}\lambda_p\lambda_h S_h. \tag{A.56}$$

By substituting $(A.51)$, $(A.52)$, $(A.53)$, $(A.54)$, $(A.55)$ and $(A.56)$ in $(A.38)$ while making S_h subject yield

$$S_h = \frac{A_0A_2A_4\big(\varepsilon\lambda_h + A_3\big)\big(\vartheta\lambda_p + A_1\big)\Lambda_h}{D_0\lambda_p\lambda_h^2 + D_1\lambda_p\lambda_h + D_2\lambda_h^2 + D_3\lambda_h + D_4\lambda_h\lambda_p^2 + D_5\lambda_p^2 + D_6\lambda_p + \mu_h A_0A_2A_4}, \tag{A.57}$$

where

$D_0 = A_2\vartheta\varepsilon\big(k_{hp}\big(1-\pi_{hp}\big) + \mu_h\big) + \mu_h A_2A_4\vartheta\varepsilon,$
$D_1 = \Big(k_hA_2A_3\vartheta\vartheta\varepsilon + \mu_h\big) + k_pA_0A_1\varepsilon\Big)\varepsilon\big(k_{hp}\big(1-\pi_{hp}\big) + \mu_h\big) + \mu_hA_2A_4A_3\vartheta + \mu_hA_0A_1A_4\varepsilon + \mu_hA_0A_2A_4\vartheta\varepsilon$, $D_2 = A_2A_4k_h\varepsilon\big(\sigma_h+\mu_h\big)+\mu_hA_1A_2A_4\varepsilon$, $D_3 = k_hA_2A_3A_4\varepsilon\big(\sigma_h+\mu_h\big)+\mu_hA_2A_4\big(A_1A_3+\varepsilon A_0\big)$,
$D_4 = k_p\vartheta\varepsilon A_0\Big(\big(1-\pi_{hp}\big)k_{hp} + \mu_h\Big) + \mu_hA_0A_4\vartheta\varepsilon$, $D_5 = k_pA_0A_4\vartheta\big(\sigma_p + \mu_h\big) + \mu_hA_0A_3A_4\vartheta A_0$,
$D_6 = k_pA_0A_1A_4\vartheta\big(\sigma_p + \mu_h\big) + \mu_hA_0A_4\big(A_1A_3 + \vartheta A_2A_3\big)$.

Substituting $(A.57)$ in $(A.51)$, $(A.52)$, $(A.53)$, $(A.54)$, $(A.55)$ and $(A.56)$ yield the corresponding values for E_h, E_p, I_h, I_p, E_{hp} and I_{hp} as a functions of λ_h and λ_p. Thus, the components of malaria and pneumonia endemic equilibrium, $\xi^\star$ has a unique positive endemic equilibrium if $\lambda_h > 0$, $\lambda_m > 0$ and $\lambda_p > 0$. Hence

$$\xi^\star = \Big(S_h^\star, E_h^\star, I_h^\star, E_p^\star, I_p^\star, E_{hp}^\star, I_{hp}^\star, S_m^\star, E_m^\star, I_m^\star\Big). \tag{A.58}$$

Appendix B. Prove of the stability analysis of the model

Proof.

(i) The Jacobian matrix of system (2.4) evaluated at ξ_0 is

$$J(\xi_0) = \begin{bmatrix}
-\mu_h & \pi & \pi & \tau & -\frac{\beta_pC\Lambda_h}{\mu_h} + \tau & \phi & -\frac{\beta_pCg\Lambda_h}{\mu_h} + \phi & 0 & 0 & -\frac{\alpha\,\beta_{mh}\Lambda_h}{\mu_h} \\
0 & -a_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\alpha\,\beta_{mh}\Lambda_h}{\mu_h} \\
0 & k_h & -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -a_2 & \frac{\beta_pC\Lambda_h}{\mu_h} & 0 & \frac{\beta_pCg\Lambda_h}{\mu_h} & 0 & 0 & 0 \\
0 & 0 & 0 & k_p & -a_3 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -a_4 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & k_{hp} & -a_5 & 0 & 0 & 0 \\
0 & 0 & -\frac{\alpha\,\beta_{hm}\Lambda_m}{\mu_m} & 0 & 0 & 0 & -\frac{\alpha\,\beta_{hm}\delta\,\Lambda_m}{\mu_m} & -\mu_m & 0 & 0 \\
0 & 0 & \frac{\alpha\,\beta_{hm}\Lambda_m}{\mu_m} & 0 & 0 & 0 & \frac{\alpha\,\beta_{hm}\delta\,\Lambda_m}{\mu_m} & 0 & -a_6 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & k_m & -\mu_m
\end{bmatrix}. \tag{A.59}$$

By inspection, it is easy to see that two eigenvalues of $(A.59)$ are $\lambda_1 = -\mu_h$ and $\lambda_2 = -\mu_m$ while the remaining eigenvalues are obtained from the following 8×8

$$A = \begin{bmatrix} -A_0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\alpha\,\beta_{mh}\Lambda_h}{\mu_h} \\ k_h & -A_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -A_2 & \frac{\beta_p C\Lambda_h}{\mu_h} & 0 & \frac{\beta_p Cg\Lambda_h}{\mu_h} & 0 & 0 \\ 0 & 0 & k_p & -A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & k_{hp} & -A_5 & 0 & 0 \\ 0 & \frac{\alpha\,\beta_{hm}\Lambda_m}{\mu_m} & 0 & 0 & 0 & \frac{\alpha\,\beta_{hm}\delta\,\Lambda_m}{\mu_m} & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_m & -\mu_m \end{bmatrix}. \quad \text{(A.60)}$$

By Routh-Hurwitz condition, all the eigenvalues of $(A.60)$ are negative if

(i) $Trace(A) < 0$ (ii) $Det(A) > 0$.

Clearly,

- $Trace(A) = -A_0 - A_1 - A_2 - A_3 - A_4 - A_5 - A_6 - \mu_m < 0$.
-

$$Det(A) = \frac{A_4A_5\left(k_h\alpha^2\beta_{mh}\Lambda_h k_m\beta_{hm}\Lambda_m - A_0{\mu_m}^2A_6A_1\mu_h\right)\left(\beta_p C\Lambda_h k_p - A_2A_3\mu_h\right)}{{\mu_h}^2\mu_m}$$

$$= \frac{A_0A_1A_2A_3A_4A_5A_6\mu_m^2\mu_h^2\left(\frac{k_mk_h\alpha^2\beta_{mh}\beta_{hm}\Lambda_h\Lambda_m}{A_0A_1A_6\mu_m^2\mu_h} - 1\right)\left(\frac{\beta_pC\Lambda_hk_p}{A_2A_3\mu_h} - 1\right)}{\mu_h^2\mu_m}$$

$$= A_0A_1A_2A_3A_4A_5A_6\mu_m\Big(R_{hm}^2 - 1\Big)\Big(R_p - 1\Big) > 0 \quad if \quad R_{hm} < 1 \quad R_p < 1.$$

So that the eigenvalues of $matrixA$ are all negative, and hence, the eigenvalues of $(A.59)$ are all negative if $R_{hm} < 1$ and $R_p < 1$. Thus, malaria and pneumonia free equilibrium, ξ_0 is locally asymptotically stable otherwise unstable.

(ii) It is easy to obtain the upper triangular matrix of the Jacobian matrix of the model system (2.4) evaluated at ξ_1 as

$$J\Big(\xi_1\Big) = \begin{bmatrix} -G_1-\mu_h & \pi & \pi & \tau & -G_2+\tau & \phi & -kG_2+\phi & 0 & 0 & -G_3 \\ 0 & -A_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & G_3 \\ 0 & 0 & -G_1-A_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_2 & G_2 & 0 & kG_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_3 & 0 & 0 & 0 & 0 & -G_5 \\ 0 & 0 & 0 & 0 & 0 & -A_4 & 0 & 0 & 0 & G_5 \\ 0 & 0 & 0 & 0 & 0 & 0 & -A_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -G_4-\mu_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m \end{bmatrix} \tag{A.61}$$

where $A_7 = \frac{\mu_h A_2 A_3}{\beta_p C(\sigma_p(k_p+\mu_h)+\mu_h A_2)}$, $G_1 = \beta_p C A_7\,(R_p-1)$, $G_2 = \frac{\beta_p C\big(k_p\Lambda_h+\pi_{hp}(k_p+A_3)A_7(R_p-1)\big)}{k_p(\beta_p C A_7(R_p-1)+\mu_h)}$,
$G_3 = \frac{\alpha\,\beta_{mh}\big(k_p\Lambda_h+\pi_{hp}(k_p+A_3)A_7(R_p-1)\big)}{k_p(\beta_p C A_7(R_p-1)+\mu_h)}$,

$G_4 = \alpha\,\beta_{hm} A_7\,(R_p-1)$, $G_5 = \alpha\,\varepsilon\,\beta_{mh} A_7\,(R_p-1)$. It is easy to see that

$Trace\Big(\xi_1\Big) = -2\,G_1-\mu_h-A_0-A_1-A_2-A_3-A_4-A_5-G_4-2\,\mu_m-A_6 < 0 \quad R_p > 1$

$Determinant\Big(\xi_1\Big) = (-G_1-\mu_h)\,A_0\,(-G_1-A_1)\,A_2A_3A_4A_5\,(G_4+\mu_m)\,A_6\mu_m > 0 \quad R_p > 1$. Hence all eigenvalues of $(A.61)$ are negative which implies that the malaria-free endemic equilibrium, ξ_1 of system (2.4) is feasible and locally asymptotically stable if $R_p > 1$ otherwise unstable.

(iii) The upper triangular matrix of the Jacobian matrix of the model system (2.4) evaluated at ξ_1 gives

$$J\Big(\xi_2\Big) = \begin{bmatrix} -G_6-\mu_h & \pi & \pi & \tau & -G_7+\tau & \phi & -kG_7+\phi & 0 & 0 & -G_8 \\ 0 & -A_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & G_8 \\ 0 & 0 & -A_1 & 0 & -G_9 & 0 & -kG_9 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_2 & G_7 & 0 & kG_7 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -G_{10}-A_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -A_4 & kG_9 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -A_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m \end{bmatrix} \tag{A.62}$$

where $A_8 = \frac{\mu_h\mu_m A_0 A_1}{\alpha\,\beta_{mh}(\alpha\,\Lambda_h k_h\beta_{hm}+\mu_m(\mu_h A_1+k_h\mu_h+k_h\sigma_h))}$, $A_9 = \frac{\mu_h{\mu_m}^2 A_0A_1A_6+\mu_h k_m\Lambda_m(\mu_h A_1+k_h\mu_h+k_h\sigma_h)}{\mu_m k_m\alpha\,\beta_{mh}(\alpha\,\Lambda_h k_h\beta_{hm}+\mu_m(\mu_h A_1+k_h\mu_h+k_h\sigma_h))}$,

$A_{10} = \frac{\mu_h {\mu_m}^3 A_0 A_1 A_6}{\alpha\,\beta_{hm}(\mu_h {\mu_m}^2 A_0 A_1 A_6 + \alpha^2 \beta_{mh}\Lambda_m k_m \mu_m(\mu_h A_1 + k_h \mu_h + k_h \sigma_h))}$, $G_6 = \alpha\,\beta_{mh} A_8\left({R_{hm}}^2 - 1\right)$,

$G_7 = \frac{\beta_p C}{\alpha\,\beta_{mh} A_8\left({R_{hm}}^2 - 1\right) + \mu_h}\left(\Lambda_h + \frac{\pi_h A_1 A_{10}\left({R_{hm}}^2 - 1\right)}{k_h} + \pi_h A_{10}\left({R_{hm}}^2 - 1\right)\right)$,

$G_8 = \frac{\alpha\,\beta_{mh}}{\alpha\,\beta_{mh} A_8\left({R_{hm}}^2 - 1\right) + \mu_h}\left(\Lambda_h + \frac{\pi_h A_1 A_{10}\left({R_{hm}}^2 - 1\right)}{k_h} + \pi_h A_{10}\left({R_{hm}}^2 - 1\right)\right)$,

$G_9 = \vartheta\,\beta_p C A_{10}\left({R_{hm}}^2 - 1\right)$, $G_{10} = \alpha\,\varepsilon\,\beta_{mh} A_8\left({R_{hm}}^2 - 1\right)$. Straight forward calculation yield

$\lambda_1 = -\left(G_6 + \mu_h\right)$, $\lambda_2 = -A_0$, $\lambda_3 = -A_1$, $\lambda_4 = -A_2$, $\lambda_5 = -\left(G_{10} + A_3\right)$, $\lambda_6 = -A_4$, $\lambda_7 = -A_5$, $\lambda_8 = -\mu_m$, $\lambda_9 = -A_6$ and $\lambda_{10} = -\mu_m$. Observe that λ_1 and λ_5 are both negative if $R_{hm}>1$, thus, all eigenvalues of $(A.62)$ are negative meaning that the pneumonia-free endemic equilibrium, ξ_2 of system (2.4) is feasible and locally asymptotically stable if $R_{hm} > 1$ and unstable if $R_{hm} < 1$.

Appendix B. Prove of the global stability of Malaria and Pneumonia co-infection free equilibrium point

Proof. Consider the following Lyapunov function

$$L(E_h, I_h, E_p, I_p, E_{hp}, I_{hp}, E_m, I_m) = \frac{k_h}{\mu_m^2 A_0}E_h + \frac{1}{\mu_m^2}I_h + \frac{k_p}{\mu_m^2 A_2}E_p + \frac{1}{\mu_m^2}I_p + \frac{1}{\mu_m^2}E_{hp} + \frac{A_4}{\mu_m^2 k_{hp}}I_{hp} + \frac{A_1}{\mu_m \alpha\beta_{hm}\Lambda_m}E_m + \frac{A_1 A_6}{\mu_m \alpha\beta_{hm}\Lambda_m k_m}I_m. \tag{B.1}$$

The differentiation of $(B.1)$ with respect to time t along the solutions of system (2.4) yield

$$L' = \left(\frac{k_h\alpha\beta_{mh}S_h}{\mu_m^2 A_0} - \frac{A_1 A_6}{\alpha\beta_{hm}\Lambda_m k_m}\right)I_m + \left(\frac{A_1 S_m}{\mu_m\Lambda_m} - \frac{A_1}{\mu_m^2}\right)I_h + \left(\frac{k_p\beta_p C S_h}{\mu_m^2 A_2} - \frac{A_3}{\mu_m^2}\right)I_p + \left(\frac{k_p\beta_p C g S_h}{\mu_m^2} + \frac{A_1\delta S_m}{\mu_m\Lambda_m} - \frac{A_4 A_5}{\mu_m^2 k_{hp}}\right.$$

At the DFE, $S_h = \frac{\Lambda_h}{\mu_h}$ and $S_m = \frac{\Lambda_m}{\mu_m}$

$$L' = \frac{A_1 A_6}{\alpha\beta_{hm}\Lambda_m k_m}\left(\frac{\alpha^2\beta_{hm}\beta_{mh}k_h k_m \Lambda_h \Lambda_m}{\mu_h \mu_m^2 A_0 A_1 A_6} - 1\right)I_m + \frac{A_3}{\mu_m^2}\left(\frac{k_p\beta_p C\Lambda_h}{\mu_h A_2 A_3} - 1\right)I_p + \frac{A_4 A_5}{k_{hp}\mu_m^2}\left(\frac{k_{hp}k_p\beta_p C g\Lambda_h}{\mu_m^2 A_2 A_4 A_5} + \frac{k_{hp}\delta A_1}{A_4 A_5} - 1\right)$$

$$L' = \frac{A_1 A_6}{\alpha\beta_{hm}\Lambda_m k_m}\left(R_{hm}^2 - 1\right)I_m + \frac{A_3}{\mu_m^2}\left(R_p - 1\right)I_p + \frac{A_4 A_5}{\mu_m^2 k_{hp}}\left(R_{mp} - 1\right)I_{hp}, \tag{B.2}$$

where

$$R_{mp} = \frac{k_{hp}}{A_4 A_5}\left(A_3 g R_p + \delta A_1\right). \tag{B.3}$$

Thus, the malaria and pneumonia free equilibrium, ξ_0 is globally asymptotically stable if $R_{hm}<1$, $R_p<1$ and $R_{mp}<1$

Remark 4.1 *The condition for the global stability of the malaria and pneumonia free equilibrium, ξ_0 require that $R_{mp}<1$ in addition to $R_{hm}<1$ and $R_p<1$. $R_{mp}<1$ account for those individuals who are infectious with both diseases.*

Appendix C. Prove of Theorem 3.4

Proof. Consider the dynamical system (2.4) with contact rate C as a bifurcation parameter, then the stability of the endemic equilibrium can be studied by considering the existence and stability of non-trivial equilibrium states in a neighbourhood of the bifurcation point. The idea here is that the Disease Free Equilibrium (DFE), ξ_0 is considered to be a manifold $\left(\xi_0, C\right)$ such that the local stability of ξ_0 changes at the point $\left(\xi_0, C^\star\right)$. Suppose from (3.3) at the critical point $R_c = R_p = 1$, then

$$\frac{\Lambda_h k_p \beta_p C}{\mu_h A_2 A_3 = 1}.$$

Therefore, the bifurcation parameter at the critical value $R_p = 1$ is

$$C = \frac{\mu_h A_2 A_3}{\Lambda_h k_p \beta_p = C^\star}. \tag{C.1}$$

This means that xi_0 is locally asymptotically stable when $C<C^\star$ and unstable if $C>C^\star$. Further, for easy representation, we rewrite system (2.4) as

$$\dot{x} = F(x, C),$$

where $x = \left(S_h, E_h, I_h, E_P, I_P, E_{hP}, I_{hP}, S_m, E_m, I_m\right)^T$ such that X_i is the ith component of X and $F = \left(F_1, F_2, F_3, F_4, F_5, F_6, F_7, F_8, F_9, F_{10}\right)^T$, i.e.,

$$\begin{aligned}
F_1 &= \Lambda_h - \alpha\beta_{mh} x_{10} x_1 - \beta_p C\left(g x_7 + x_5\right) x_1 - \mu_h x_1 + \pi_h x_3 + \pi_h x_2 + \pi_p x_5 + \pi_p x_4 + \pi_{hp} x_7 + \pi_{hp} x_6,\\
F_2 &= \alpha\beta_{mh} x_{10} x_1 - A_0 x_2,\\
F_3 &= k_h x_2 - \vartheta\beta_p C\left(g x_7 + x_5\right) x_3 - A_1 x_3,\\
F_4 &= \beta_p C\left(g x_7 + x_5\right) x_1 - A_2 x_4,\\
F_5 &= -\varepsilon\alpha\beta_{mh} x_{10} x_5 + k_p x_4 - A_3 x_5,\\
F_6 &= \vartheta\beta_p C\left(g x_7 + x_5\right) x_1 + \varepsilon\alpha\beta_{mh} x_{10} x_1 - A_4 x_6,\\
F_7 &= k_{hp} x_6 - A_5 x_7,\\
F_8 &= \Lambda_m - \alpha\beta_{hm}\left(\delta x_7 + x_3\right) x_8 - \mu_m x_8,\\
F_9 &= \alpha\beta_{hm}\left(\delta x_7 + x_3\right) x_8 - a_6 x_9,\\
F_{10} &= k_m x_9 - \mu_m x_{10}.
\end{aligned} \tag{C.2}$$

The Jacobian of $(C.2)$ evaluated at $(\xi_0, C^\star)$ denoted by $J(\xi_0, C^\star)$ is obtained as

$$J(\xi_0, C^\star) = \begin{bmatrix}
-\mu_h & \pi_h & \pi_h & \pi_p & -\frac{a_2 a_3}{k_p} + \pi_p & \pi_{hp} & -\frac{a_2 a_3 g}{k_p} + \pi_{hp} & 0 & 0 & -\frac{\alpha\beta_{mh}\Lambda_h}{\mu_h}\\
0 & -a_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\alpha\beta_{mh}\Lambda_h}{\mu_h}\\
0 & k_h & -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & -a_2 & \frac{a_2 a_3}{k_p} & 0 & \frac{a_2 a_3 g}{k_p} & 0 & 0 & 0\\
0 & 0 & 0 & k_p & -a_3 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & -a_4 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & k_{hp} & -a_5 & 0 & 0 & 0\\
0 & 0 & -\frac{\alpha\beta_{hm}\Lambda_m}{\mu_m} & 0 & 0 & 0 & -\frac{\alpha\beta_{hm}\delta\Lambda_m}{\mu_m} & -\mu_m & 0 & 0\\
0 & 0 & \frac{\alpha\beta_{hm}\Lambda_m}{\mu_m} & 0 & 0 & 0 & \frac{\alpha\beta_{hm}\delta\Lambda_m}{\mu_m} & 0 & -a_6 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & k_m & -\mu_m
\end{bmatrix}. \tag{C.3}$$

According to the Centre Manifold Theorems as proved by Castillo-Chavez and Song [4], $J(\xi_0, C^\star)$ have a zero simple eigenvalue while others have negative real parts. In order to determine the coefficients a and b, we first obtain the values of the left and right eigen vectors of

$J(\xi_0, C^\star)$ denoted respectively as

$$\nu = \Big(\nu_1, \nu_2, \nu_3, \nu_4, \nu_5, \nu_6, \nu_7, \nu_8, \nu_9, \nu_{10}\Big),$$

and

$$r = \Big(r_1, r_2, r_3, r_4, r_5, r_6, r_7, r_8, r_9, r_{10}\Big)^T.$$

Observe that the $J(\xi_0, C^\star)$ is not symmetric, thus the left and right eigen vectors are not equivalent. The solutions of the following equations

$$\nu J(\xi_0, C^\star) = 0, \tag{C.4}$$

and

$$J(\xi_0, C^\star) r = 0, \tag{C.5}$$

yields the following solutions

$$\nu = \left(0, 0, 0, \nu_4, \frac{A_2\nu_4}{k_p}, \frac{gA_2A_3k_{hp}\nu_4}{A_4A_5k_p}, \frac{gA_2A_3\nu_4}{A_5k_p}, 0, 0, 0\right), \tag{C.6}$$

and

$$r = \left(-\frac{\left(\mu_h^2 + (k_p + \pi_p + \sigma_p)\,\mu_h + k_p\sigma_p\right)r_5}{\mu_h k_p}, 0, 0, \frac{a_3 r_5}{k_p}, r_5, 0, 0, 0, 0, 0\right), \tag{C.7}$$

where ν_4 and r_5 are free positive left and right eigenvectors respectively. The following nonzero partial derivatives of F at xi_0 for system $(C.2)$ are obtained as

$$\frac{\partial^2 F_1}{\partial x_1 \partial x_5} = -\beta_p C, \frac{\partial^2 F_1}{\partial x_1 \partial x_7} = -\beta_p C g, \frac{\partial^2 F_1}{\partial x_1 \partial x_{10}} = -\alpha\beta_{mh}, \frac{\partial^2 F_2}{\partial x_1 \partial x_{10}} = \alpha\beta_{mh} C, \frac{\partial^2 F_3}{\partial x_3 \partial x_5} = -\vartheta\beta_p C, \frac{\partial^2 F_3}{\partial x_3 \partial x_7} = -\vartheta\beta$$

$$\frac{\partial^2 F_4}{\partial x_1 \partial x_5} = \beta_p C, \frac{\partial^2 F_4}{\partial x_1 \partial x_7} = \beta_p C g, \frac{\partial^2 F_5}{\partial x_5 \partial x_{10}} = -\varepsilon\alpha\beta_{mh}, \frac{\partial^2 F_6}{\partial x_1 \partial x_{10}} = \varepsilon\alpha\beta_{mh}, \frac{\partial^2 F_6}{\partial x_1 \partial x_5} = \vartheta\beta_p C, \frac{\partial^2 F_6}{\partial x_1 \partial x_7} = \vartheta\beta_p C g$$

$$\frac{\partial^2 F_8}{\partial x_3 \partial x_8} = -\alpha\beta_{hm}, \frac{\partial^2 F_8}{\partial x_7 \partial x_8} = -\alpha\beta_{hm}\delta, \frac{\partial^2 F_9}{\partial x_3 \partial x_8} = \alpha\beta_{hm}, \frac{\partial^2 F_9}{\partial x_7 \partial x_8} = \alpha\beta_{hm}\delta, \frac{\partial^2 F_1}{\partial x_5 \partial C} = -\frac{\beta_p\Lambda_h}{\mu_h}, \frac{\partial^2 F_1}{\partial x_7 \partial C} = -\frac{\beta_p g\Lambda}{\mu_h}$$

$$\frac{\partial^2 F_4}{\partial x_5 \partial C} = \frac{\beta_p\Lambda_h}{\mu_h}, \frac{\partial^2 F_4}{\partial x_7 \partial C} = \frac{\beta_p g\Lambda_h}{\mu_h}, \frac{\partial^2 F_6}{\partial x_5 \partial C} = \frac{\vartheta\beta_p\Lambda_h}{\mu_h}, \frac{\partial^2 F_6}{\partial x_7 \partial C} = -\frac{\vartheta\beta_p g\Lambda_h}{\mu_h}.$$

Thus, the computation of the coefficient of b yield

$$\begin{aligned}
b &= \sum_{i,k=1}^{10} \nu_k r_i \frac{\partial^2 F_k}{\partial x_i \partial C}\Big(\xi_0, C^\star\Big) \\
&= \nu_1 r_5 \frac{\partial^2 F_1}{\partial x_5 \partial C}\Big(\xi_0, C^\star\Big) + \nu_1 r_7 \frac{\partial^2 F_1}{\partial x_7 \partial C}\Big(\xi_0, C^\star\Big) + \nu_4 r_5 \frac{\partial^2 F_4}{\partial x_5 \partial C}\Big(\xi_0, C^\star\Big), \nu_4 r_7 \frac{\partial^2 F_4}{\partial x_7 \partial C}\Big(\xi_0, C^\star\Big) \\
&+ \nu_6 r_5 \frac{\partial^2 F_6}{\partial x_5 \partial C}\Big(\xi_0, C^\star\Big) + \nu_6 r_7 \frac{\partial^2 F_6}{\partial x_7 \partial C}\Big(\xi_0, C^\star\Big) \\
&= \frac{\Lambda_h\beta_p\left(\vartheta\nu_6 - \nu_1 + \nu_4\right)\left(g r_7 + r_5\right)}{\mu_h} \\
&= \frac{\nu_4 r_5 \beta_p \Lambda_h \left(g\vartheta A_2 A_3 k_{hp} + A_4 A_5 k_p\right)}{A_4 A_5 k_p \mu_h} > 0.
\end{aligned} \tag{C.8}$$

Note that b is always positive since ν_4 and r_5 are positive. It then follows that the bifurcation behaviour of the dynamical system $(C.2)$ is totally determined by the sign of the coefficient a.

$$
\begin{aligned}
a &= \sum_{i,k=1}^{10} \nu_k r_i r_j \frac{\partial^2 F_k}{\partial x_i \partial x_j}\Big(\xi_0, C^\star\Big) \\
&= 2\Bigg[\nu_1 r_1 r_5 \frac{\partial^2 F_1}{\partial x_1 \partial x_5}\Big(\xi_0, C^\star\Big) + \nu_1 r_1 r_7 \frac{\partial^2 F_1}{\partial x_1 \partial x_7}\Big(\xi_0, C^\star\Big) + \nu_1 r_1 r_{10} \frac{\partial^2 F_1}{\partial x_1 \partial x_{10}}\Big(\xi_0, C^\star\Big) + \nu_2 r_1 r_{10} \frac{\partial^2 F_2}{\partial x_1 \partial x_{10}}\Big(\xi_0, C^\star\Big) \\
&+ \nu_3 r_3 r_5 \frac{\partial^2 F_3}{\partial x_3 \partial x_5}\Big(\xi_0, C^\star\Big) + \nu_3 r_3 r_7 \frac{\partial^2 F_3}{\partial x_3 \partial x_7}\Big(\xi_0, C^\star\Big) + \nu_4 r_1 r_5 \frac{\partial^2 F_4}{\partial x_1 \partial x_5}\Big(\xi_0, C^\star\Big) + \nu_4 r_1 r_7 \frac{\partial^2 F_4}{\partial x_1 \partial x_7}\Big(\xi_0, C^\star\Big) \\
&+ \nu_5 r_5 r_{10} \frac{\partial^2 F_5}{\partial x_5 \partial x_{10}}\Big(\xi_0, C^\star\Big) + \nu_6 r_1 r_5 \frac{\partial^2 F_6}{\partial x_1 \partial x_5}\Big(\xi_0, C^\star\Big) + \nu_6 r_1 r_7 \frac{\partial^2 F_6}{\partial x_1 \partial x_7}\Big(\xi_0, C^\star\Big) + \nu_6 r_1 r_{10} \frac{\partial^2 F_6}{\partial x_1 \partial x_{10}}\Big(\xi_0, C^\star\Big) \\
&+ \nu_8 r_3 r_8 \frac{\partial^2 F_8}{\partial x_3 \partial x_8}\Big(\xi_0, C^\star\Big) + \nu_8 r_7 r_8 \frac{\partial^2 F_8}{\partial x_7 \partial x_8}\Big(\xi_0, C^\star\Big) + \nu_9 r_3 r_8 \frac{\partial^2 F_9}{\partial x_3 \partial x_8}\Big(\xi_0, C^\star\Big) + \nu_9 r_7 r_8 \frac{\partial^2 F_9}{\partial x_7 \partial x_8}\Big(\xi_0, C^\star\Big)\Bigg] \\
&= -\frac{2\nu_3 r_3 r_7 \vartheta \mu_h A_2 A_3 g}{\Lambda_h k_p} + \frac{2\nu_6 r_1 r_7 \vartheta \mu_h A_2 A_3 g}{\Lambda_h k_p} - \frac{2\nu_1 r_1 r_7 \mu_h A_2 A_3 g}{\Lambda_h k_p} + \frac{2\nu_4 r_1 r_7 \mu_h A_2 A_3 g}{\Lambda_h k_p} - \frac{2\nu_3 r_3 r_5 \vartheta \mu_h A_2 A_3}{\Lambda_h k_p} \\
&+ \frac{2\nu_6 r_1 r_5 \vartheta \mu_h A_2 A_3}{\Lambda_h k_p} - 2\nu_8 r_7 r_8 \alpha \beta_{hm} \delta + 2\nu_9 r_7 r_8 \alpha \beta_{hm} \delta - 2\nu_5 r_5 r_{10} \alpha \varepsilon \beta_{mh} + 2\nu_6 r_1 r_{10} \alpha \varepsilon \beta_{mh} \\
&- \frac{2\nu_1 r_1 r_5 \mu_h A_2 A_3}{\Lambda_h k_p} + \frac{2\nu_4 r_1 r_5 \mu_h A_2 A_3}{\Lambda_h k_p} - 2\nu_8 r_3 r_8 \alpha \beta_{hm} + 2\nu_9 r_3 r_8 \alpha \beta_{hm} - 2\nu_1 r_1 r_{10} \alpha \beta_{mh} + 2\nu_2 r_1 r_{10} \alpha \beta_{mh} \\
&= -\frac{2\nu_4\big(\mu_h^2 + (k_p + \pi_p + \sigma_p)\,\mu_h + k_p \sigma_p\big) r_5^2 A_2 A_3 \left(g A_2 A_3 k_{hp} \vartheta + A_4 A_5 k_p\right)}{\Lambda_h k_p^3 A_4 A_5} < 0.
\end{aligned}
\tag{C.9}
$$

It follows that the sign of a is negative, thus the bifurcation is transcritical (forward).

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