

MATHEMATICAL MODELLING OF THE DYNAMICS OF A MOSQUITO-BORN DISEASE WITH THE ACCOUNT OF INSECTICIDE TREATED NETS AND IMMUNITY BUILD-UP

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Abstract

Mosquito-borne diseases are a great concern in many areas in the world, including Sub-Saharan Africa, South America, and Southeast Asia, claiming thousands of lives annually. These diseases are transmitted to humans through the bites of female mosquitoes carrying the pathogen and seeking a blood meal. Some examples of mosquito-borne diseases are Zika, Chikungunya, Dengue, and Malaria. Preventing mosquito-borne diseases lies in avoiding mosquito bites. To achieve this objective, insecticide-treated bed nets (ITNs) are widely distributed in the population. However, the bed nets reduce immunity development. Indeed, regular exposure to infection is crucial for immunity building. In this work, we propose a new mathematical model with the influence of bed nets on lowering immunity build-up. From the mathematical point of view, we derive the well-posedness of the model and investigate the stability of the disease-free equilibrium. Numerically, we prove that the basic reproduction number is a sharp threshold for disease elimination and that the immunity build-up is a decreasing function of the bed net used. This analysis suggests the need to address supplement control measures, such as vaccination, to balance the trade-off between bed nets and immunity development.

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Keywords: Mosquito-Borne Disease; Bed nets; Immunity; Breeding sites; Questing mosquitoes.

Mathematical Subject Classifications: 34C60, 37N25, 37N30, 65L12.

1 Introduction

Mosquito-borne diseases pose significant public health challenges, particularly in regions such as Sub-Saharan Africa, South America, Southeast Asia, and the Western Pacific. Annually, these diseases contribute to millions of infections and thousands of fatalities [18], with Africa being disproportionately affected due to factors such as poverty, limited access to healthcare, and low adherence to preventive measures. These diseases are transmitted to humans through the bites of female mosquitoes that carry pathogens when they seek out to obtain blood meal necessary for reproduction. Infected humans can transmit the disease to mosquitoes through the same process. Often, once vectors become infectious, they are capable of transmitting the pathogen for the rest of their life through [18]. Some examples of mosquito-borne diseases include Zika, Chikungunya, Dengue, Leishmaniasis, yellow fever, West Nile fever, Japanese encephalitis, and Malaria [18].

Preventing mosquito-borne diseases primarily focuses on minimizing mosquito bites, which can be achieved by door and window screens, indoor residual spraying (IRS), peridomestic space spraying, the use of insect repellents, and insecticide-treated bed-nets (ITNs) [3, 10]. Among these control measures, ITNs are the most common and are widely used in the world. In various countries, ITNs have been extensively distributed and have been made freely accessible, allowing poor populations to be protected against mosquito-borne diseases [16, 17]. ITNs serve as a physical barrier, protecting individuals from mosquito bites during sleep while simultaneously killing or repelling mosquitoes through the insecticide-infused fabric. A picture of a bed net is given in Figure 1. However, the widespread use of ITNs has unintended consequences on immunity development. Regular exposure to pathogens, such as the *Plasmodium* parasites responsible for malaria, is crucial for building immunity, particularly in endemic regions [7]. By lowering infection rates, ITNs hinder immunity development, making individuals more vulnerable to severe mosquito-borne diseases that can lead to death when they eventually encounter these pathogens. Hence, it is of paramount importance to assess the influence of bed nets on immunity build-up and related consequences.

An outfit of mathematical models has been addressed to understand the dynamics of mosquito-borne diseases (see, for instance, [1, 5, 6, 8, 12] and references therein), and some of them investigated the relevance of bed net use in decreasing the number of malaria cases [1, 14]. The concern in these works is that none has considered the influence of bed nets on immunity build-up. We want to fill this gap. More precisely, this project aims to investigate the long-term effects of ITNs' usage on mosquito-borne disease transmission and immunity development through mathematical modeling. Our model involves the following key aspects:



Figure 1: A mosquito bed net

- The transmission dynamics of mosquito-borne diseases between humans and mosquitoes, with a clear distinction between mosquitoes in the breeding sites and those questing for a blood meal.
- The role of ITNs in reducing mosquito biting rates and increasing mosquito mortality.
- The lifecycle of mosquitoes, including their development from eggs and larvae to pupae in breeding sites, and their pursuit of blood.
- The process of immunity development, built on disease incidence.

Through mathematical analysis, we establish the model's well-posedness and compute the basic reproduction number, which allows us to establish the local asymptotic stability of the disease-free equilibrium. Numerical simulations support the theory, show the existence and the global asymptotic stability of the unique endemic equilibrium, demonstrate the effectiveness of bed nets in lowering the disease burden, while highlighting their detrimental impact on immunity development.

The rest of the paper is structured as follows. In Section 2, the model is formulated under some assumptions. Section 3 presents the quantitative and qualitative analysis of the model. In Section 4, we provide the model simulations. Section 5 gives the conclusion of the paper and provides future work.

2 Model formulation

2.1 Main assumptions and model variables

For the sake of simplicity, we consider the following assumptions:

- A1. Recovered humans do not transmit the disease to mosquitoes.
- A2. The proportion of mosquito bed nets used in the population and their effectiveness remain constant.
- A3. All the mosquitoes that successfully acquire a blood meal return to the breeding sites to rest and reproduce.

For $t \geq 0$, we consider the following mutually exclusive variables:

- $S := S(t)$, susceptible humans.
- $I := I(t)$, infected humans.
- $R := R(t)$, recovered humans.
- $S_b := S_b(t)$, susceptible mosquitoes in breeding sites.
- $I_b := I_b(t)$, infected mosquitoes in breeding sites.
- $S_q := S_q(t)$, susceptible questing mosquitoes.
- $I_q := I_q(t)$, infected questing mosquitoes.
- $I_m := I_m(t)$, immunity building in the population.

Set

$$N_h = S + I + R \text{ and } W = S_b + S_q + I_b + I_q ,$$

the human and the mosquito populations, respectively.

2.2 Derivation of equations

the use of mosquito bed nets reduces interactions between humans and mosquitoes. Let us denote by κ the proportion of bed nets used in the population and ε their effectiveness. Through the use of bed nets, the mosquito biting rate $\beta(\kappa)$ can be modelled as (see [1] for a similar biting rate)

$$\beta(\kappa) = \beta_M - \kappa\varepsilon(\beta_M - \beta_m) ,$$

where β_M and β_m are the maximum and the minimum biting rates, respectively.

In addition, the ITNs increase the death rate, μ_q , of questing mosquitoes. Let us denote by μ_0 the natural death rate of questing mosquitoes. Then μ_q can be expressed as

$$\mu_q(\kappa) = \mu_0 + \mu_1\kappa,$$

where $\mu_1\kappa$ is the ITNs-induced death rate.

The biting rate $\beta(\kappa)$ is the same for both human and mosquito populations, so the average number of bites per human per unit time is $\beta(\kappa)(S_q + I_q)/N_h$ [1]. Therefore, the force of infection in the human population is given by

$$\lambda_h = \frac{p\beta(\kappa)(S_q + I_q)}{N_h} \frac{I_q}{(S_q + I_q)} = \frac{p\beta(\kappa)I_q}{N_h},$$

where p is the probability for a human to become infected after being bitten.

The force of infection in the mosquito population is given by

$$\lambda_q = \frac{p_q\beta(\kappa)I}{N_h},$$

where p_q is the probability for a mosquito to become infected after a blood meal.

The interactions between mosquitoes in I_q and S_q with humans are depicted by

$$\frac{\beta(\kappa)I_q(S + a_1I + b_1R)}{N_h} \text{ and } \frac{\beta(\kappa)S_q(\hat{a}_1S + I + \hat{b}_1R)}{N_h}, \text{ respectively}$$

where $a_1(\hat{a}_1)$ measures the mosquitoes' modification attraction due to interactions between mosquitoes in $I_q(S_q)$ and humans in the $I(S)$ compartment; $b_1(\hat{b}_1)$ measures the mosquitoes' modification attraction due to interactions between mosquitoes in $I_q(S_q)$ and humans in the R compartments.

The interactions between mosquitoes in I_q and humans lead to:

- the infection of susceptible humans, which may occur at the infection force λ_h , i.e, a number

$$\frac{p\beta(\kappa)SI_q}{N_h} \tag{1}$$

of susceptible humans move from S to I .

- the moving of all these mosquitoes to the breeding sites, i.e, a number

$$\frac{\beta(\kappa)I_q(S + a_1I + b_1R)}{N_h} \tag{2}$$

of mosquitoes move from I_q to I_b .

The interactions between mosquitoes in S_q and humans lead to:

- the infection of mosquitoes, which may occur at the infection force λ_q .
- the moving of all these mosquitoes to the breeding sites. Among the mosquitoes that return to the breeding sites,

$$\frac{p_q\beta(\kappa)IS_q}{N_h} \tag{3}$$

move to I_b and the other to S_b . The latter group of mosquitoes encompasses all of those which interact with susceptible and recovered humans, and the proportion $(1 - p_q)$ of those which do not acquire the disease after interacting with infected humans. Namely, the proportion of mosquitoes in S_q which move to S_b is given as:

$$\frac{\beta(\kappa)S_q(\hat{a}_1S + \hat{b}_1R)}{N_h} + \frac{(1 - p_q)\beta(\kappa)IS_q}{N_h}. \quad (4)$$

The susceptible human population is recruited at the constant Λ due to births or migrations and undergoes a natural mortality rate μ_h . These susceptible individuals can be infected after interacting with disease-infected mosquitoes, with the infection force λ_h . Once infected, these individuals move to the compartment I , where they can die due to the disease at the rate δ or recover at the rate γ . Recovered individuals have a temporary immunity that lasts for $\frac{1}{\alpha}$. The dynamics of the human population is given by the system:

$$\begin{cases} \dot{S} = \Lambda - \frac{p\beta(\kappa)SI_q}{N_h} - \mu_h S + \alpha R, \\ \dot{I} = \frac{p\beta(\kappa)SI_q}{N_h} - (\mu_h + \gamma + \delta)I, \\ \dot{R} = \gamma I - (\mu_h + \alpha)R. \end{cases} \quad (5)$$

The mosquito recruitment rate in the breeding site is assumed to follow a logistic growth

$$\eta\theta\phi(S_q + I_q)\left(1 - \frac{S_q + I_q}{K}\right)$$

that depends on questing mosquitoes, where θ is the number of eggs laid by female mosquitoes; η is the probability of eggs surviving from the egg to the adult stage, ϕ is the rate at which eggs develop to the adult stage, and K is the carrying capacity. These mosquitoes can die naturally at the rate μ_b or quest for blood at the rate ν_1 . Taking into account the number of questing mosquitoes that return to breeding sites, as given in Eq. (4), leads to the dynamics of S_b below:

$$\dot{S}_b = \eta\theta\phi(S_q + I_q)\left(1 - \frac{S_q + I_q}{K}\right) - (\mu_b + \nu_1)S_b + \frac{\beta(\kappa)S_q(\hat{a}_1S + \hat{b}_1R)}{N_h} + \frac{(1 - p_q)\beta(\kappa)IS_q}{N_h}.$$

The infected mosquitoes compartment in the breeding sites is supplied by the questing mosquitoes which return to the breeding sites and given by Eq. (2) and Eq. (3). Mosquitoes in I_b quest for blood at the rate ν_2 . Taking into account the natural mortality, one has the following dynamics of I_b :

$$\dot{I}_b = \frac{p_q\beta(\kappa)IS_q}{N_h} - (\mu_b + \nu_2)I_b + \frac{\beta(\kappa)I_q(S + a_1I + b_1R)}{N_h}.$$

The questing mosquitoes in S_q and I_q compartments are supplied by the numbers ν_1S_b and ν_2I_b mosquitoes that left the S_b and I_b compartments, respectively, because they are

looking for a blood meal. Questing mosquitoes in S_q and I_q compartments undergo mortality at the rate $\mu_q(\kappa)$ or return to the breeding sites S_b and I_b at the numbers

$$\frac{\beta(\kappa)S_q(\hat{a}_1S + I + \hat{b}_1R)}{N_h} \text{ and } \frac{\beta(\kappa)I_q(S + a_1I + b_1R)}{N_h}, \text{ respectively,}$$

leading to the dynamics of S_q and I_q below:

$$\begin{cases} \dot{S}_q = \nu_1 S_b - \mu_q(\kappa) S_q - \frac{\beta(\kappa)S_q(\hat{a}_1S + I + \hat{b}_1R)}{N_h}, \\ \dot{I}_q = \nu_2 I_b - \mu_q(\kappa) I_q - \frac{\beta(\kappa)I_q(S + a_1I + b_1R)}{N_h}. \end{cases} \quad (6)$$

Mosquito-borne immunity is built up per mosquito bite at the rate σ . We denote by c the factor that measures the immunity level, which gives 50% chance to prevent infection, and ω the natural decay rate of immunity. Taking into account the fact that human mortality leads to immunity loss, one has the following dynamics of immunity build-up:

$$\dot{I}_m = \frac{\frac{\sigma\beta(\kappa)pI_q}{N_h} S_h}{c + \frac{\beta(\kappa)pI_q}{N_h} S_h} - \left(\omega + \frac{\delta I_h}{N_h} + \mu_h \right) I_m.$$

Gathering all the equations leads to System (8) for which the flow diagram is given by Figure 2.

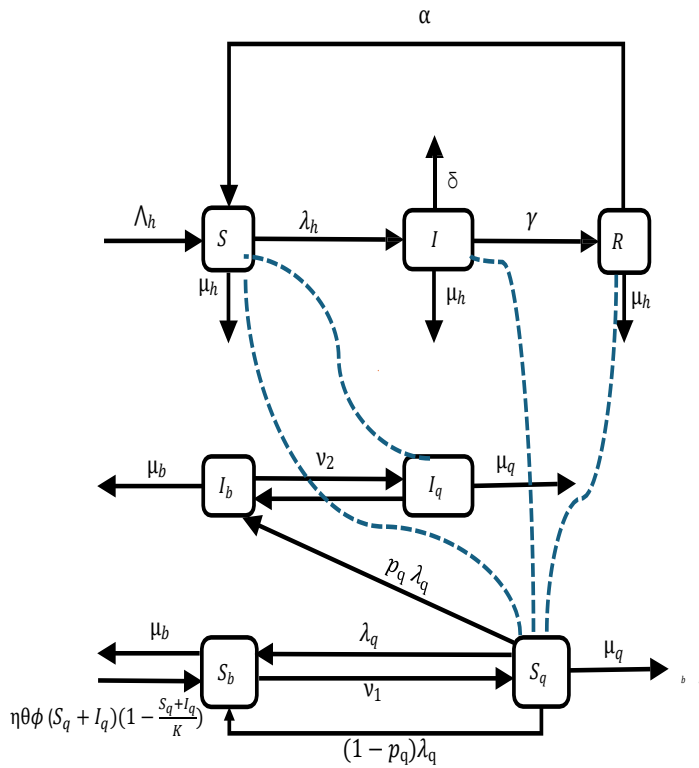


Figure 2: Flow diagram of the model

$$\left\{ \begin{array}{l} \dot{S} = \Lambda - \frac{p\beta(\kappa)SI_q}{N_h} - \mu_h S + \alpha R, \\ \dot{I} = \frac{p\beta(\kappa)SI_q}{N_h} - (\mu_h + \gamma + \delta)I, \\ \dot{R} = \gamma I - (\mu_h + \alpha)R, \\ \dot{S}_b = \eta\theta\phi(S_q + I_q) \left(1 - \frac{S_q + I_q}{K}\right) - (\mu_b + \nu_1)S_b \\ \quad + \frac{\beta(\kappa)S_q(\hat{a}_1 S + \hat{b}_1 R)}{N_h} + \frac{(1 - p_q)\beta(\kappa)IS_q}{N_h}, \\ \dot{I}_b = \frac{p_q\beta(\kappa)IS_q}{N_h} - (\mu_b + \nu_2)I_b + \frac{\beta(\kappa)I_q(S + a_1 I + b_1 R)}{N_h}, \\ \dot{S}_q = \nu_1 S_b - \mu_q(\kappa)S_q - \frac{\beta(\kappa)S_q(\hat{a}_1 S + I + \hat{b}_1 R)}{N_h}, \\ \dot{I}_q = \nu_2 I_b - \mu_q(\kappa)I_q - \frac{\beta(\kappa)I_q(S + a_1 I + b_1 R)}{N_h}, \\ \dot{I}_m = \frac{\sigma\beta(\kappa)pI_q S_h}{cN_h + \beta(\kappa)pI_q S_h} - \left(\omega + \frac{\delta I_h}{N_h} + \mu_h\right)I_m, \end{array} \right. \quad (7)$$

subject to the initial conditions:

$$\begin{array}{llll} S(0) = S_0 > 0, & I(0) = I_0 \geq 0, & R(0) = R_0 \geq 0, & S_b(0) = S_{b0} > 0, \\ I_b(0) = I_{b0} \geq 0, & S_q(0) = S_{q0} > 0, & I_q(0) = I_{q0} \geq 0, & I_m(0) = I_{m0} \geq 0. \end{array}$$

The dynamics of System (7) is the same as that of the reduced system (since I_m does not intervene in the other equations):

$$\left\{ \begin{array}{l} \dot{S} = \Lambda - \frac{p\beta(\kappa)SI_q}{N_h} - \mu_h S + \alpha R, \\ \dot{I} = \frac{p\beta(\kappa)SI_q}{N_h} - (\mu_h + \gamma + \delta)I, \\ \dot{R} = \gamma I - (\mu_h + \alpha)R, \\ \dot{S}_b = \eta\theta\phi(S_q + I_q) \left(1 - \frac{S_q + I_q}{K}\right) - (\mu_b + \nu_1)S_b \\ \quad + \frac{\beta(\kappa)S_q(\hat{a}_1 S + \hat{b}_1 R)}{N_h} + \frac{(1 - p_q)\beta(\kappa)IS_q}{N_h}, \\ \dot{I}_b = \frac{p_q\beta(\kappa)IS_q}{N_h} - (\mu_b + \nu_2)I_b + \frac{\beta(\kappa)I_q(S + a_1 I + b_1 R)}{N_h}, \\ \dot{S}_q = \nu_1 S_b - \mu_q(\kappa)S_q - \frac{\beta(\kappa)S_q(\hat{a}_1 S + I + \hat{b}_1 R)}{N_h}, \\ \dot{I}_q = \nu_2 I_b - \mu_q(\kappa)I_q - \frac{\beta(\kappa)I_q(S + a_1 I + b_1 R)}{N_h}. \end{array} \right. \quad (8)$$

Throughout this work, we assume $\mu_q \leq \eta\theta\phi$. If this condition is not fulfilled, the population of mosquitoes will disappear in the long run.

Table 1: Parameters and their epidemiological interpretations.

Parameter	Epidemiological interpretation	Values	Units	References
Λ	Recruitment constant in S .	0.0015875	hum.day ⁻¹	[15]
κ	Proportion of bed nets used.	0.6	-	Assumed
ε	Efficacy of bed nets used.	1	-	Assumed
p	Probability for a human to be infected after being bitten.	0.75	mos ⁻¹	[15]
p_v	Probability for a questing mosquito to be infected after a bite.	0.75	hum ⁻¹	[15]
β_M	Maximum contact rate between susceptible in S and infected questing mosquitoes.	0.1	hum.day ⁻¹	[1]
β_m	Minimum contact rate between susceptible in S and infected questing mosquitoes.	0	hum.day ⁻¹	[1]
$a_1(\hat{a}_1)$	Modification parameter for the contact between mosquitoes in $I_q(S_q)$ and humans in $I(S)$ as compared to β .	1	-	Assumed
$b_1(\hat{b}_1)$	Modification parameter for the interaction between mosquitoes in $I_q(S_q)$ and humans in R as compared to β .	1	-	Assumed
ν_1	Mosquitoes questing rate in S_b .	0.005	day ⁻¹	Assumed
ν_2	Mosquitoes questing rate in I_b .	0.005	day ⁻¹	Assumed
μ	Natural mortality rate of humans.	0.00004	day ⁻¹	[15]
μ_b	Mortality rate of mosquitoes in breeding sites.	0.05	day ⁻¹	[15]
μ_0	Natural mortality rate of questing mosquitoes.	1/21	day ⁻¹	[1]
μ_1	Mortality rate of questing mosquitoes due to bed nets.	0.01	day ⁻¹	Assumed
θ	Number of eggs laid by female mosquitoes.	200	day ⁻¹	Assumed
η	Probability of eggs surviving from the egg to the adult stage.	0.4	-	Assumed
ω	Immunity decay rate.	0.06	-	Assumed
ϕ	Rate at which an egg develops into an adult.	0.6	-	Assumed
γ	Disease recovery rate.	1/4	day ⁻¹	[1]
δ	Disease-induced death rate.	10 ⁻³	day ⁻¹	[1]
α	Immunity waning rate after recovering.	0.1	day ⁻¹	[2]
σ	Disease immunity built up rate per mosquito bite.	0.2	-	Assumed
c	Measures the immunity level, which gives 50% chance to prevent infection.	100	hum.day ⁻¹	Assumed
K	Mosquito carrying capacity	100,000	mos	Assumed

3 Quantitative and qualitative analysis

For the sake of notational simplicity, we define the following parameters:

$$\psi_1 = \frac{(\eta\theta\phi - \mu_q)K}{\eta\theta\phi}; \quad \psi_2 = \max(\hat{a}_1 + \hat{b}_1, 1 + a_1 + b_1).$$

3.1 Well-posedness of the model

The following theorem ensures the well-posedness for Model (8).

Theorem 3.1 *Model (8) is a dynamical system on the following biologically feasible compact region, which is attractive:*

$$\Omega := \left\{ (S, I, R, S_b, I_b, S_q, I_q) \in \mathbb{R}_+^7 : N_h \leq \frac{\Lambda}{\mu_h}, S_q + I_q \leq \psi_1, S_b + I_b \leq \frac{\psi_1[\eta\theta\phi + \beta(\kappa)\psi_2]}{\mu_b} \right\}. \quad (9)$$

Proof

Model (8) is a dynamical system on Ω following the three steps below:

- Model (8) admits a unique local solution, due to the differentiable property of its right-hand side.
- The positive cone $\mathbb{R}^7$ is forward-invariant with respect to System (8). The proof of this item is obtained by the the Barrier theorem [13] (the reader interested in much detail is referred to [9, 11]).
- Any solutions initiated in Ω satisfies the *priori* estimate

$$N_h \leq \frac{\Lambda}{\mu_h}, \quad S_q + I_q \leq \psi_1, \quad S_b + I_b \leq \frac{\psi_1[\eta\theta\phi + \beta(\kappa)\psi_2]}{\mu_b}.$$

The inequality $N_h \leq \Lambda/\mu_h$ can be easily obtained by adding the first three equations of System (8) and applying the Gronwall inequality.

Adding the last four equations of System (8) gives:

$$\begin{aligned} \dot{W} &= \eta\theta\phi(S_q + I_q) \left(1 - \frac{S_q + I_q}{K} \right) - \mu_b(S_b + I_b) - \mu_q(\kappa)(S_q + I_q) \\ &\leq \eta\theta\phi(S_q + I_q) \left(1 - \frac{S_q + I_q}{K} \right) - \mu_q(\kappa)(S_q + I_q) \\ &= (\eta\theta\phi - \mu_q(\kappa))(S_q + I_q) \left(1 - \frac{S_q + I_q}{\psi_1} \right). \end{aligned}$$

Hence, the population $S_q + I_q$ of questing mosquito can be taken less or equal to the new carrying capacity ψ_1 .

Adding the fourth and fifth equations of System (8) leads to

$$\begin{aligned}\dot{S}_b + \dot{I}_b &\leq \eta\theta\phi(S_q + I_q) - \mu_b(S_b + I_b) - \nu_1 S_b - \nu_2 I_b + \beta(\kappa)S_q(\hat{a}_1 + \hat{b}_1) \\ &\quad + \beta(\kappa)I_q(1 + a_1 + b_1) \\ &\leq [\eta\theta\phi + \beta(\kappa)\psi_2](S_q + I_q) - \mu_b(S_b + I_b) \\ &\leq \psi_1[\eta\theta\phi + \beta(\kappa)\psi_2] - \mu_b(S_b + I_b).\end{aligned}$$

By the Gronwall inequality, one gets

$$(S_b + I_b)(t) \leq \frac{\psi_1[\eta\theta\phi + \beta(\kappa)\psi_2]}{\mu_b} \text{ whenever } (S_b + I_b)(0) \leq \frac{\psi_1[\eta\theta\phi + \beta(\kappa)\psi_2]}{\mu_b}$$

and

$$\limsup_{t \rightarrow \infty} (S_b + I_b)(t) \leq \frac{\psi_1[\eta\theta\phi + \beta(\kappa)\psi_2]}{\mu_b},$$

which shows that Ω is attractive. $\square$

3.2 Disease-free equilibrium and basic reproduction number

Setting the right-hand side of System (8) to zero, when

$$\lambda_h = \lambda_q = 0$$

the disease-free equilibrium (DFE), E_0 , of System (8) is given as

$$E_0 = (S^0, I^0, R^0, S_b^0, S_q^0, I_b^0, I_q^0)$$

where

$$\begin{aligned}S^0 &= \frac{\Lambda_1}{\mu_h}, \quad I_q^0 = I^0 = I_b^0 = R^0 = 0, \quad S_q^0 = \frac{\nu_1 S_b^0}{\mu_q + \beta(\kappa)\hat{a}_1}, \\ S_b^0 &= \frac{K(\mu_b + \nu_1)(\mu_q + \beta(\kappa)\hat{a}_1)^2}{\eta\theta\phi\nu_1^2} \left(\frac{\nu_1\eta\theta\phi}{(\mu_q(\kappa) + \beta(\kappa)\hat{a}_1)(\mu_b + \nu_1)} - 1 \right),\end{aligned} \quad (10)$$

whenever

$$\mathcal{T} = \frac{\nu_1\eta\theta\phi}{(\mu_q(\kappa) + \beta(\kappa)\hat{a}_1)(\mu_b + \nu_1)} \geq 1.$$

The basic reproduction number, $\mathcal{R}_0$, for Model (8) is the average number of cases generated by one human or mosquito infected introduced into a completely susceptible population during all his/her/its infectious period. We use the next generation method to compute $\mathcal{R}_0$. Let us order the infected compartments as in the vector $\mathcal{X}$ defined by

$$\mathcal{X} = (I_1, I_b, I_q)$$

and $\mathbf{y}$ denote the disease free compartments,

$$\mathbf{y} = (S_1, R_1, S_b, S_q).$$

The infected components of the system (8) can be written in the form,

$$\frac{dX}{dt} = \mathcal{F} - \mathcal{V}, \quad (11)$$

where $\mathcal{F}$ is the vector of appearance of new infections and $\mathcal{V}$ the transfer term vector between compartments.

The Jacobian matrices, F and V , of $\mathcal{F}$ and $\mathcal{V}$, at the disease-free equilibrium are given as

$$F = \begin{pmatrix} 0 & 0 & \frac{p\beta(\kappa)}{N_h^0} \\ \frac{p_q\beta(\kappa)S_q^0}{N_h^0} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \mu_h + \gamma + \delta & 0 & 0 \\ 0 & \mu_b + v_2 & \frac{-\beta(\kappa)}{N_h^0} \\ 0 & -v_2 & \mu_q(\kappa) + \frac{\beta(\kappa)}{N_h^0} \end{pmatrix}, \quad (12)$$

respectively and

$$FV^{-1} = \begin{pmatrix} 0 & \frac{p\beta(\kappa)v_2}{N_h^0 \left(v_2\mu_q(\kappa) + \mu_b \left(\mu_q(\kappa) + \frac{\beta(\kappa)}{N_h^0} \right) \right)} & \frac{p\beta(\kappa)(\mu_b + v_2)}{N_h^0 \left(v_2\mu_q(\kappa) + \mu_b \left(\mu_q(\kappa) + \frac{\beta(\kappa)}{N_h^0} \right) \right)} \\ \frac{p_q\beta(\kappa)S_q^0}{N_h^0(\mu_h + \gamma + \delta)} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The basic reproduction number, $\mathcal{R}_0$, is computed as

$$\mathcal{R}_0 = \rho(FV^{-1}) = \sqrt{\frac{p\beta(\kappa)v_2}{N_h^0 \left(v_2\mu_q(\kappa) + \mu_b \left(\mu_q(\kappa) + \frac{\beta(\kappa)}{N_h^0} \right) \right)} \frac{p_q\beta(\kappa)S_q^0}{N_h^0(\mu_h + \gamma + \delta)}} \quad (13)$$

with $\rho(FV^{-1})$ the spectral radius of the matrix, FV^{-1} .

The relevance of the basic reproduction number is provided in the following result:

Theorem 3.2 *The disease-free equilibrium is locally asymptotically stable when the basic reproduction number $\mathcal{R}_0 < 1$, and is unstable when $\mathcal{R}_0 > 1$ provided that $\mathcal{T} > 1$.*

- The biological interpretation of Theorem 3.2 is that the disease will be overcome locally when $\mathcal{R}_0 < 1$ and may persist when $\mathcal{R}_0 > 1$ provided that $\mathcal{T} > 1$.
- $\mathcal{R}_0$ can be written as

$$\mathcal{R}_0 = \sqrt{\mathcal{R}_0^H \cdot \mathcal{R}_0^q},$$

where

$$\mathcal{R}_0^H = \frac{p\beta(\kappa)v_2}{N_h^0 \left(v_2\mu_q(\kappa) + \mu_b \left(\mu_q(\kappa) + \frac{\beta(\kappa)}{N_h^0} \right) \right)}, \text{ and } \mathcal{R}_0^q = \frac{p_q\beta(\kappa)S_q^0}{N_h^0(\mu_h + \gamma + \delta)}$$

are the basic reproduction numbers of disease-human infection and disease-mosquito infection, respectively.

- $\mathcal{T}$ can be defined as the basic offspring number for the mosquito population [4]. The condition $\mathcal{T} > 1$ in Theorem 3.2 is a necessary criterion for the coexistence of humans and mosquitoes in the population. Indeed, from Equation (10), we observe that when $\mathcal{T} = 1$, only the human population persists at the disease-free equilibrium (DFE), whereas when $\mathcal{T} < 1$, the DFE is not defined.
- Moreover, simple computations show that

$$\frac{\partial \mathcal{R}_0^H}{\partial \kappa} = \frac{-pv_2 N_0^h [\varepsilon \mu_q(k)(\beta_M - \beta_m) + \beta(k)\mu_1]}{(N_h^0)^2 \left(v_2 \mu_q(\kappa) + \mu_b \left(\mu_q(\kappa) + \frac{\beta(\kappa)}{N_h^0} \right) \right)^2} < 0, \text{ and } \mathcal{R}_0^q = \frac{-p_q \varepsilon (\beta_M - \beta_m) S_q^0}{N_h^0 (\mu + \gamma + \delta)} < 0.$$

This highlights that the basic reproduction number decreases as bed-net usage increases; in other words, the more people use bed nets, the more malaria transmission is reduced within the population.

4 Numerical simulations

In this section we carry out some simulations to determine our model's behavior in the long run and to support the mathematical theory. In Figure 3, we investigate the asymptotic stability of the DFE of Model (7) when $\mathcal{R}_0 < 1$. This figure highlights that, in the long run, all the infected will disappear in the population, suggesting the global asymptotic stability of the DFE. In this regard, the Theorem 3.2 can be improved as follows: The DFE of Model (7) is globally asymptotically stable (GAS) when $\mathcal{R}_0 < 1$ and it is unstable when $\mathcal{R}_0 > 1$.

In Figure 4, we investigate the existence and the stability of an interior equilibrium for Model (7), an aspect which has not been addressed from a mathematical point of view. This figure shows that when $\mathcal{R}_0 > 1$, different trajectories will converge to the same positive equilibrium point, suggesting the existence, the uniqueness, and the global asymptotic stability of the endemic equilibrium for Model (7) when $\mathcal{R}_0 > 1$.

In Figure 5, we examine the impact of bed net coverage on the development of immunity in humans. The figure shows three coverage levels: (a) $\kappa = 0.2$ (black curve), (b) $\kappa = 0.4$ (blue curve), and (c) $\kappa = 0.6$ (red curve). As bed net use increases, population-level immunity declines, which may hinder disease control. This result highlights the negative effect of widespread bed net use on immunity build-up and underline the importance of finding a balance between protection through bed nets and the preservation of natural immunity. This balance can be achieved by the use of the vaccine to increase the level of immunity within the population.

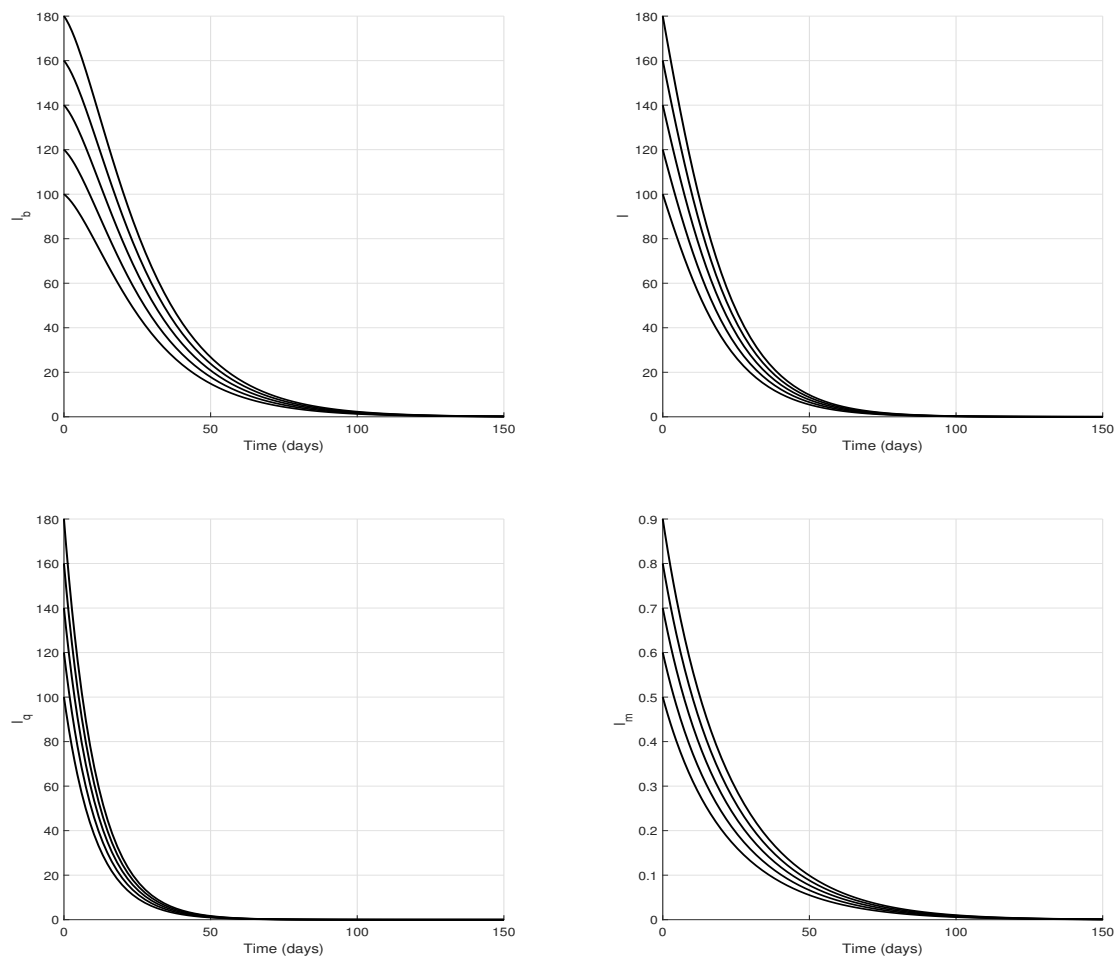


Figure 3: Global asymptotic stability of the DFE for $\mathcal{R}_0 < 1$. This figure is plotted with $K = 10000$. The other parameters are gathered in Table 1. These parameters give $\mathcal{R}_0 = 5.0058$.

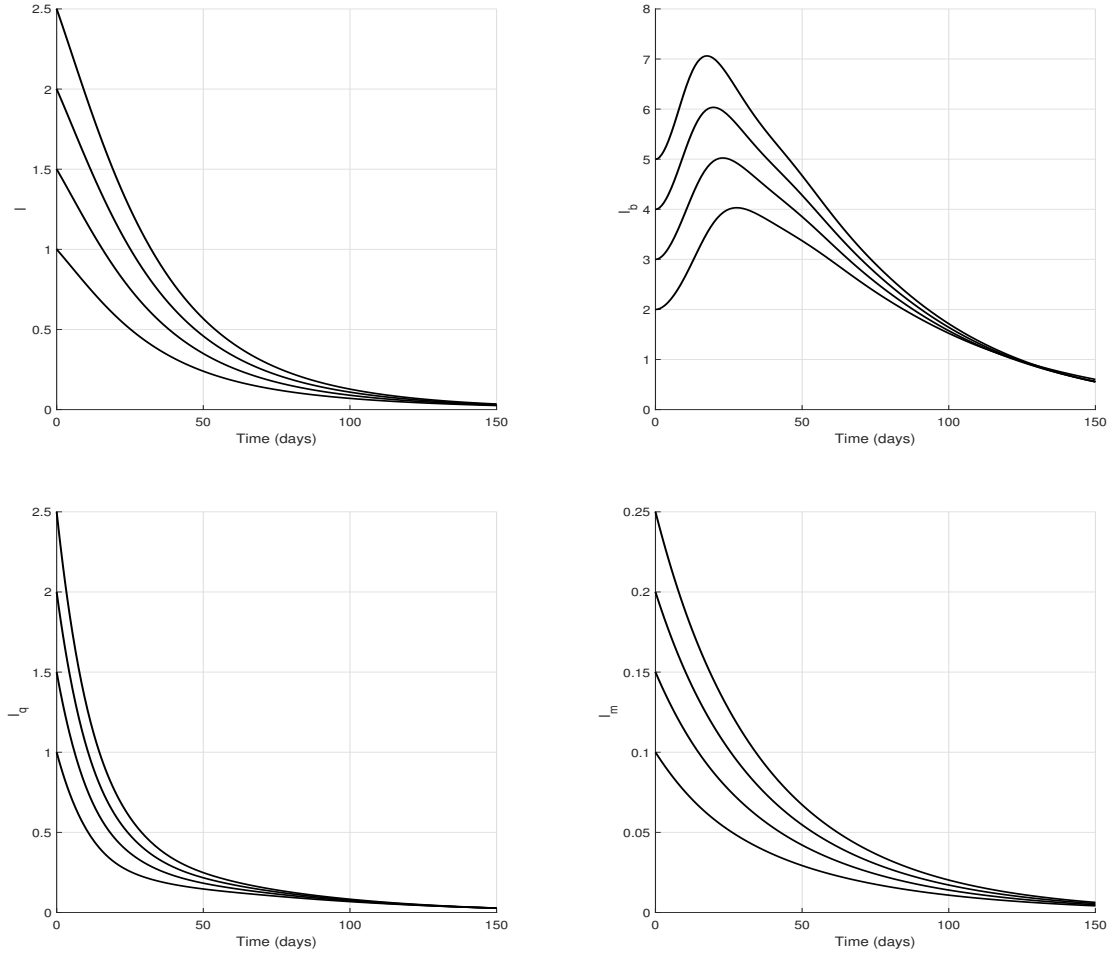


Figure 4: Existence and global asymptotic stability of the endemic equilibrium for $\mathcal{R}_0 > 1$. This Figure is plotted with $\mu_b = 0.03, \delta = 0.0001; \gamma = 0.04; \mu_0 = 0.001; \beta_M = 0.32; v_1 = 0.0015; v_2 = 0.0025; \eta = 0.9; \theta = 100; \phi = 0.3; p = 0.3; p_v = 1; \sigma = 0.4; \omega = 0.03; \kappa = 0.8$. The other parameters are gathered in Table 1. These parameters give $\mathcal{R}_0 = 3.7935$.

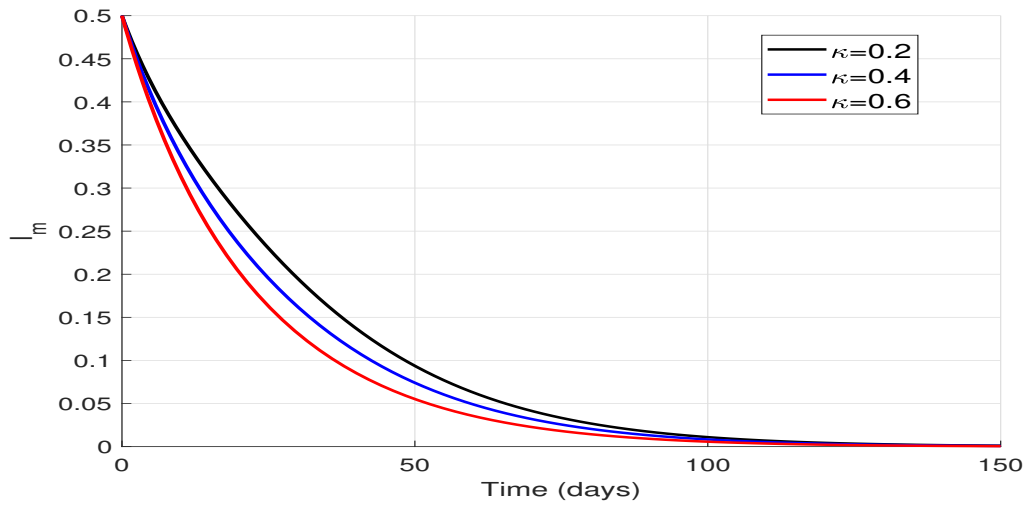


Figure 5: Influence of bed net used on immunity build up. This figure is plotted with the parameters gathered in Table 1.

5 Conclusions and future work

Mosquito-borne diseases pose a significant threat to public health, causing considerable human, social, and economic damage. To mitigate the spread of these diseases, international organizations, in collaboration with governments, provide subsidies for the free distribution of impregnated mosquito nets. These nets have played a crucial role in reducing mortality and morbidity associated with these diseases. However, their use can also lead to a decrease in population immunity, as immunity is built when individuals are exposed to infections [7]. A direct consequence of reduced immunity is that individuals who contract the infection may develop more severe forms of the disease, potentially increasing the death rate.

In this study, we proposed a model that incorporates the use of mosquito nets and their counterproductive effect on the development of immunity. We derived that the model is well-posed and established the conditions under which the disease can be eliminated from both human and mosquito populations. Through numerical methods, we explored the existence and uniqueness of an endemic equilibrium and showed that the basic reproduction number serves as a sharp threshold for effective disease control. Additionally, we illustrated the negative impact of mosquito net usage on immunity development.

However, this study represents only a preliminary step and does not yet account for the role of immunity in determining disease severity. In future work, we plan to refine the model to incorporate this important factor while keeping it mathematically tractable. Including immunity will enable a theoretical analysis of how bed net use influences immunity build-up. Combined with numerical simulations, this extension will offer new insights to healthcare practitioners for improving malaria control strategies. Furthermore, we intend to incorporate supplementary control measures,

such as vaccination, to balance the trade-off between mosquito nets and immunity development.

Acknowledgments

The author thanks the anonymous reviewer for valuable suggestions that have helped improve the article. He thanks also the organizers of the “Mathematics Industry Study Group, 2025” for their kind invitation and support for this work. He is particularly grateful to Prof. C. Ngonghala and Prof. G. Ngwa, from the University of Florida, USA, and the University of Buea, Cameroon, respectively, who proposed this topic as part of this event.

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