Available online at http://jprm.sms.edu.pk/
Journal of Prime Research in Mathematics,
Special Issue on Mathematical and Computational Methods in Nonlinear Systems: Theory and Applications (2026), 41–67

Journal of Prime Research in Mathematics

A Mathematical Approach to Malaria Eradication: Insights from Vaccination Strategies

Saheed Ajao[a,*], John O. Akanni[b,*], Titilayo Akinwumi[c], Musibau A. Omoloye[d], Isaac Olopade[e], Olusola A. Odebiyi[f], F. Fatmawati[g]

[a]*Department of Mathematics, Obafemi Awolowo University, Ile-Ife, Osun State, Nigeria.*
[b]*Department of Mathematics, Saveetha School of Engineering, Saveetha Nagar, Chennai, 602 105, Tamil Nadu, India.*
[c]*Department of Mathematics and Computer Science, Elizade University, Ilara-Mokin, Akure, Ondo State, Nigeria.*
[d]*Department of Mathematical Science, Nigerian Defence Academy, Kaduna, Nigeria.*
[e]*Department of Mathematics and Statistics, Federal University, Wukari, Taraba State, Nigeria.*
[f]*Department of Pure and Applied Mathematics, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.*
[g]*Department of Mathematics, Universitas Airlangga, Kampus C Mulyorejo, Surabaya, Indonesia.*

Abstract

In the continued effort to eradicate malaria, the World Health Organisation recently recommended the use of matrix RS1 as a malaria vaccine, the second vaccine after the launch of the first one in 2021. However, efficacy has been a critical issue with vaccines. This study used mathematical modelling to assess the potential impact of vaccines on malaria eradication. The aim of this study is to provide quantitative information on the impact of vaccines on malaria eradication. The formulated model considers vaccination and vaccine efficacy in studying their impact on malaria control. The malaria-free equilibrium and the metric that determines the spread of malaria and its extinction are obtained. Analysis of the existence of an endemic state reveals that there are multiple equilibria, which leads to a backward bifurcation when the bifurcation coefficient $a > 0$. Global endemic stability is established when $R_{vo} > 1$. Furthermore, the global sensitivity analysis conducted identifies parameters such as the probability of transmission from humans to mosquitoes, the probability of transmission from mosquitoes to humans, biting rate, mosquito recruitment rate, treatment rate, mosquito death rate, and vaccine efficacy as significant determinants of malaria spread and control. Further numerical simulations highlight the importance of vaccine effectiveness in controlling malaria, as a highly effective vaccine is required, along with other interventions, to successfully combat the spread of malaria.

Keywords: Vaccination, Vaccine efficacy, Malaria, Control reproduction number, Stability, Centre manifold theory.

*Corresponding author
Email addresses: goajao@gmail.com (Saheed Ajao), johnakanni06@gmail.com (John O. Akanni), titilayo.akinwumi@elizadeuniversity.edu.ng (Titilayo Akinwumi), am.omoloye@nda.edu.ng (Musibau A. Omoloye), isaac.olopade@fuwukari.edu.ng (Isaac Olopade), oaodebiyi@lautech.edu.ng (Olusola A. Odebiyi), fatmawati@fst.unair.ac.id (F. Fatmawati)

Received : 2 July 2025*; Accepted:* 15 January 2026*; Published Online:* 27 March 2026

2010 Mathematics Subject Classification: 92D30, 92C60, 34D20, 34C60, 37N25, 92B05

1. Introduction

Malaria is a vector-borne disease that is caused by the parasite *Plasmodium* and is transmitted through bites from an infected female Anopheles mosquito [1]. It is a great public health issue that affects children under the age of five, pregnant women, and their unborn children [2]. In sub-Saharan Africa, malaria remains one of the leading causes of disease and death in children [3]. In 2023, there were 263 million cases of the disease globally that occurred in 83 malaria-endemic countries, of which 246 million cases came from the WHO African Region [4, 5].

Recently, the global malaria deaths for the year 2023 remained at 597,000 deaths, with more than 50% of the deaths occurring in Tanzania (4.3%), Niger (5.9%), the Democratic Republic of the Congo (11.3%), and Nigeria (30.9%) [5]. In Africa, where malaria is a substantial burden, controlling malaria is difficult. Some of the reasons for this include the presence of mosquitoes that are efficient in transmitting the parasite, favourable climate conditions for mosquito breeding, a high prevalence of the deadliest parasite species, a high cost of intervention that rules out full-scale-up in low-income countries, and inadequate infrastructure to combat malaria [6].

The burden of malaria in Africa remains high despite various interventions. Hence, the need for other intervention tools, such as vaccines, to combat the disease is important. Vaccines are important for controlling and preventing outbreaks of infectious diseases. They give support to global health security, and they are also important tools in the fight against antimicrobial resistance[7]. Vaccines have helped eliminate diseases such as smallpox, poliomyelitis, rubella, and congenital rubella syndrome in Americans [8].

In 2021, the WHO approved the use of RTS, S / AS01, the first vaccine to prevent malaria in regions where transmission is high and moderate [3]. In addition, in 2023, the WHO approved another malaria vaccine called R21 / Matrix-M for use in malaria-endemic countries [9]. The World Health Organisation (WHO) plans to introduce two malaria vaccines, RTS,S/AS01 (RTS,S) and R21/Matrix-M (R21), into regular immunisation programmes for children aged 5 months in 19 African countries. The implementation of the malaria vaccine in Africa represents an important step in the fight against the disease [10]. One of the main concerns regarding vaccines is their efficacy. Venkatesan [10] reported that vaccine efficacy decreases considerably over 12–18 months. However, malaria is a curable and preventable disease.

Mathematical models have become imperative tools in public health decision-making. In the aspect of chronic disease, they are used to make predictions about changes in population size, distributions, risk factor patterns, and the impact of intervention measures in a population [11]. Mathematical modelling has been applied to study the transmission dynamics of infectious diseases (see [12, 13, 14, 15, 16, 17]). Modellers and researchers have provided many mathematical frameworks to study the spread dynamics of malaria and control (see [18, 19, 20, 21, 22, 23]). In the work of Adeniyi *et al.* [24], a mathematical model was developed to study the influence of the response of people to information on the dynamic spread of malaria. The results of the study indicated that awareness of the usage of insecticide-treated nets causes behavioural changes in susceptible humans and, therefore, reduces the burden of the disease.

Furthermore, Ibrahim *et al.* [25] explored the contribution of awareness to malaria control. In this study, the authors divided the infected class into unaware and aware infected people, with the assumption that the growth rate of the awareness campaign impacting the population is proportional to the unaware infected group. It was also assumed that aware infected people always avoided contact with vectors as a result of the effect of the awareness programme. The study results highlighted the need for effective awareness programmes to reduce the burden of malaria in society.

Similarly, a study of the effect of awareness and control interventions on malaria dynamics was conducted by Ojo and Goufo [26]. The authors performed a sensitivity analysis and found that bed net use and residual spray were the most impactful parameters on the reproduction number. The authors concluded that the proportion of bed net use, awareness, and residual spray should be increased to at least 75% and prioritised to combat malaria. In addition, Onifade *et al.* [27] provided a model for quantifiable predictions of the

impact of malaria testing on malaria prevalence. The authors matched the model with malaria data, and the results revealed that the test will prevent the prevalence of malaria from increasing, drastically retard the change of the wild strain to the resistant strain, and prevent more treatment failures.

Ochieng [28] presented a seasonal deterministic SEIRS model to measure the impact of awareness on the dynamics of malaria. The study's findings revealed that awareness through social networks, in conjunction with other control strategies, is the most cost-effective approach to controlling the disease. In another study, Mangongo *et al.* [23] analysed the global asymptotic stability of a malaria model. The authors incorporated ignorant infected humans into the model and performed a global sensitivity analysis of the model. The results of the sensitivity analysis indicated that the fraction of infectious persons who recovered and the rate of recovery of infectious persons are the most significant factors in the dynamics of the disease. Furthermore, it was revealed that reducing the size of the ignorant population and the number of mosquito bites can subdue malaria transmission. In the same vein, Witbooi *et al.* [29] considered the influx and outflow of migrants in the modelling of malaria transmission. They also factored in the effect of residual indoor spraying on mosquitoes. Their results showed the effect of infected immigrants on disease in South Africa and the process of determining the influx rate responsible for the observed number of malaria incidences in South Africa. Furthermore, Wang *et al.* [30] applied optimal control to study an age-structured malaria model. The authors accommodated relapse and vaccination in their model. Insecticide spraying during the epidemic period was concluded to be more effective than other preventive strategies. In addition, it was revealed that the combination of all other strategies with insecticide spraying was more efficient in mitigating the burden of the disease.

Furthermore, Ngonghala [31] studied the effect of temperature and deterioration of net efficacy treated with insecticides on malaria dynamics and control. The author conducted a sensitivity analysis on the model, and the results showed that parameters related to temperature are more important in the dynamics of the disease. It was also concluded that control efforts that ensure that insecticide-treated nets are changed frequently and early enough, and that educate populations at risk on the correct care and use of ITNs, are important for reducing the number of malaria cases. In a study by Collins and Duffy [32], a mathematical model of malaria was proposed that incorporated treatment, drug resistance, and the use of mosquito nets to prevent malaria. The results of the study showed that good control strategies that focus on improved treatment, the dominant resistant strain, and the use of insecticide-treated nets should be prioritised to achieve a malaria-free situation.

Similarly, Woldegerima [33] formulated and analysed a mathematical model of malaria to evaluate the impact of transmission-blocking drugs. The findings of their study revealed that treating 35% of the sub-Saharan African population with effective transmission-blocking drugs with 95% efficacy will produce an 82% reduction in malaria mortality by 2035. Furthermore, Massad *et al.* [34] employed a mathematical model to investigate the risk of infection among visitors to the Brazilian Amazonian region. The authors calibrated the model with malaria incidence cases in Lábrea, Amazonas state. The study revealed that the highest risk was experienced towards the end of the rainy season, and the relationship between infection risk and arrival time was linear, positively correlated, and relied on the local effects of the seasonality.

The mathematical model presented by Olaniyi *et al.* [35] incorporated a saturated treatment function and transmission through blood transfusion. In addition, the authors included controls such as antimalarial drug treatment, blood screening, insecticide-treated nets, and vector control with insecticide spray to study the optimal control measures for malaria. It was concluded that the most efficient and cost-effective combination of the four controls should be implemented to mitigate the spread of malaria. Tchoumi [36] proposed a mathematical framework for the dynamics of malaria spread, taking into account the differential susceptibility of humans, with the assumption that immunity is acquired partially after contracting the disease. The results of the study indicated that people who have experienced malaria many times are less likely to become infected over time.

In addition, Adegbite *et al.* [37] presented a deterministic model of malaria that considered the effects of adopting traditional control methods by vigilant individuals. Their study suggested that total vigilance in the use of traditional malaria control strategies and control strategies approved by the World Health

Organisation are the best control strategies against malaria importation. In the work of Traore *et al.*, [38], a malaria model was formulated and analysed, in which the mosquito population was classified into four metamorphic phases. The authors also incorporated a seasonal effect into the model. The authors concluded that a reduction in the carrying capacity of mosquitoes will have a huge impact on the spread of the disease, as this will lower the transition rate of immature mosquitoes and increase their mortality. In addition, malaria elimination is guaranteed if the mosquito population can be eradicated or if the basic reproductive number does not exceed one. The results of the study further revealed that a change in climatic conditions can trigger the recurrence of diseases in some US and European countries. Haile *et al.* [40] presented a malaria model that incorporates infected ignorant individuals and relapse. The results revealed that mosquito protection strategies are more efficient in combating malaria than treatment. Kaboré *et al.* [39] studied the role of male mosquitoes in the transmission dynamics of malaria. The results of the study show how changes in mating-related parameters influence the population of mosquitoes and the spread of malaria.

Recently, Naandam *et al.* [41] modelled malaria transmission with vaccine-induced immunity and vaccination proportion. However, the authors used mass action incidence to capture malaria transmission from mosquitoes to humans and vice versa.

The mathematical study of the impact of vaccination and the efficacy of vaccines on the dynamics and control of malaria is limited. Therefore, we present a mathematical model focused on vaccines to gain quantitative insights into the impact of vaccines on eradicating malaria. This mathematical model considers vaccination to study the effects of vaccines and their efficacy on the dynamic spread and control of malaria. The remaining parts of the paper are organised as follows: Section 2 describes the methods, Section 3 presents the results and discussion, and the conclusion follows in Section 4.

2. Methods

2.1. Model Assumptions and Formulation

Malaria is a vector-borne disease which is transmitted through the bite of mosquitoes, so to model the dynamics of malaria, two interacting populations are required, namely: the population of humans and the population of mosquitoes. The total population of humans, denoted by N_h, is classified into the following classes: the susceptible class of people (S_h), the vaccinated class (V_h), the exposed class (E_h), the infectious class (I_h) and the recovered class of people (R_h). The total population of mosquitoes denoted by N_v is classified as susceptible (S_v), exposed (E_v) and infectious (I_v). The susceptible class S_h of humans is assumed to be populated at the rates Λ_h (recruitment rate), χ by people who return to susceptible after the vaccine wanes, and θ by those who lost their immunity after recovering from malaria. The class size of the susceptible human decreases due to infection following contact with infectious mosquitoes at a rate λ_M, where

$$\lambda_M = \frac{\beta_h \phi I_v}{N_h}, \tag{2.1}$$

β_h is the probability of transmission from an infectious mosquito to a human, and ϕ is the biting rate of mosquitoes. The population further declines due to the vaccination of susceptible individuals at a rate ω. The vaccinated class (V_h) is populated by individuals from the susceptible class (S_h) who are vaccinated at a rate ω. It is assumed that the vaccine is imperfect, which makes vaccinated individuals (V_h) acquire malaria at a rate $(1-\sigma)\lambda_M$, where σ accounts for the vaccine efficacy and lies in the range $0 < \sigma < 1$. It is further assumed that the vaccine wanes off at a rate χ, which thereby reduces the vaccinated (V_h) class. The exposed class (E_h) is composed of those who newly acquire malaria at the rates λ_M and $(1-\sigma)\lambda_M$ in the susceptible class (S_h) and vaccinated class (V_h), respectively. The decline in the size of the exposed class (E_h) occurs due to progression at a rate κ. The infectious class (I_h) is generated at a rate κ from the exposed class (E_h), and a reduction is experienced in the class as a result of treatment and malaria-induced death (at rates τ and δ). The recovered population (R_h) contains those who are treated in the infectious class (I_h) (at a rate τ). The size of the recovered population (R_h) shrinks as a result of the loss of immunity (at a rate θ). The mosquitoes are recruited into the susceptible class (S_v) at a rate Λ_v. The susceptible

mosquito compartment (S_v) reduces following contact with exposed and infectious individuals at a rate λ_v, where

$$\lambda_v = \frac{\beta_v \phi (E_h + \eta I_h)}{N_h}, \tag{2.2}$$

β_v is the probability of transmission from the infected individuals to the mosquito, and η represents the relative infectiousness of the infectious humans (I_h) when compared with the exposed humans (E_h). The exposed class of mosquitoes (E_v) has the newly infected mosquitoes that change their status from susceptible to infected, and the progression at a rate γ reduces the population size of the exposed mosquitoes. The infectious mosquito population (I_v) is generated by the exposed mosquitoes (E_v) that progress into the class (at a rate γ). It is assumed that both the human and mosquito subclasses experience natural mortality at the rates μ_h and μ_v, respectively, which reduces all the subpopulations of humans and mosquitoes. The interplay among the variables and parameters is presented diagrammatically in Figure 1. The parameters associated with the model are defined in Table 1. All the above assumptions result in the following system of ordinary differential equations (ODEs):

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \lambda_M S_h + \theta R_h + \chi V_h - (\omega + \mu_h) S_h, \\
\frac{dV_h}{dt} &= \omega S_h - (1-\sigma)\lambda_M V_h - (\chi + \mu_h) V_h \\
\frac{dE_h}{dt} &= \lambda_M S_h + (1-\sigma)\lambda_M V_h - (\kappa + \mu_h) E_h, \\
\frac{dI_h}{dt} &= \kappa E_h - (\tau + \mu_h + \delta) I_h, \\
\frac{dR_h}{dt} &= \tau I_h - (\theta + \mu_h) R_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \lambda_v S_v - \mu_v S_v, \\
\frac{dE_v}{dt} &= \lambda_v S_v - (\gamma + \mu_v) E_v, \\
\frac{dI_v}{dt} &= \gamma E_v - \mu_v I_v,
\end{aligned} \tag{2.3}$$

where

$$\begin{aligned}
\lambda_M &= \frac{\beta_h \phi I_v}{N_h}, \\
\lambda_v &= \frac{\beta_v \phi (E_h + \eta I_h)}{N_h}.
\end{aligned} \tag{2.4}$$

and

$$N_h = S_h + V_h + E_h + I_h + R_h.$$

For computational purposes, the malaria model in (2.3) can be rewritten as follows:

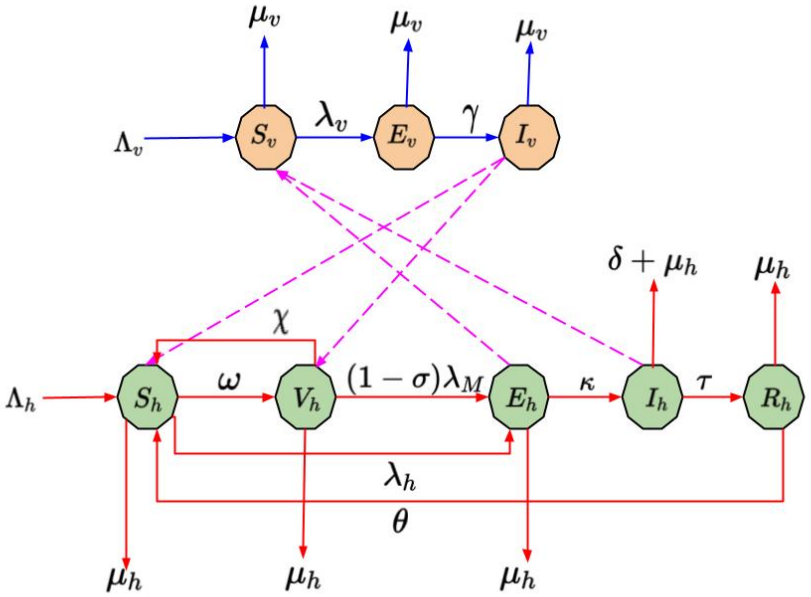

Figure 1: Schematic diagram of malaria model (2.3)

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \lambda_M S_h + \theta R_h + \chi V_h - C_1 S_h,\\
\frac{dV_h}{dt} &= \omega S_h - (1-\sigma)\lambda_M V_h - C_2 V_h\\
\frac{dE_h}{dt} &= \lambda_M S_h + (1-\sigma)\lambda_M V_h - C_3 E_h,\\
\frac{dI_h}{dt} &= \kappa E_h - C_4 I_h,\\
\frac{dR_h}{dt} &= \tau I_h - C_5 R_h,\\
\frac{dS_v}{dt} &= \Lambda_v - \lambda_v S_v - \mu_v S_v,\\
\frac{dE_v}{dt} &= \lambda_v S_v - C_6 E_v,\\
\frac{dI_v}{dt} &= \gamma E_v - \mu_v I_v,
\end{aligned} \tag{2.5}$$

where

$$\begin{aligned}
C_1 &= \omega + \mu_h, & C_4 &= \tau + \mu_h + \delta,\\
C_2 &= \chi + \mu_h, & C_5 &= \theta + \mu_h,\\
C_3 &= \kappa + \mu_h, & C_6 &= \gamma + \mu_v.
\end{aligned}$$

2.2. Fundamental Properties of the Model

The required properties of the malaria model (2.3) are explored here.

Table 1: Definition of the parameters of the model (2.3)

Parameter	Definition.
μ_h	Humans' natural mortality rate
η	Amplification factor
κ	Transition rate
ω	Vaccination rate
β_h	Probability of transmission from infectious mosquitoes to humans
ϕ	Biting rate of mosquitoes
γ	Progression rate for mosquitoes
δ	Rate of death due to malaria
χ	Vaccines wane off rate
τ	Recovery rate due to treatment
Λ_h	Inflow rate into the susceptible class of humans
Λ_v	Inflow rate into the susceptible class of mosquitoes
β_v	Probability of transmission from infected humans to susceptible mosquitoes
σ	Vaccine efficacy rate
μ_v	Mosquitoes' natural death rate
θ	Rate of immunity loss

2.2.1. Non-negativity of solutions

The populations involved in the model (2.3), which are the populations of humans and vectors, cannot be negative. Therefore, it is imperative to verify that they are non-negative. This is achieved by proving Theorem 2.1

Theorem 2.1. *The solutions* $S_h(t), V_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t)$ *and* $I_v(t)$ *of the model* (2.3) *with regard to the starting values* $S_h(0) > 0, V_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_v(0) > 0, E_v(0) \geq 0$ *and* $I_v(0) \geq 0$ *are all non-negative for all possible values of* $t > 0$.

Proof. Let $t_1 = sup\{t > 0 : S_h(t) > 0, V_h(t) > 0, E_h(t) > 0, I_h(t) > 0, R_h(t) > 0, S_v(t) > 0, E_v(t) > 0$ and $I_v(t) > 0\} > 0$, and arising from the S_h class of the model (2.3),

$$\frac{dS_h}{dt} = \Lambda_h - \lambda_M S_h + \theta R_h + \chi V_h - (\omega + \mu_h) S_h. \tag{2.6}$$

The following inequality holds

$$\frac{dS_h}{dt} \geq -(\lambda_M + \omega + \mu) S_h, \tag{2.7}$$

$$\int_0^{t_1} \frac{dS_h}{S_h(t)} \geq \int_0^{t_1} -(\lambda_M + \omega + \mu) dt, \tag{2.8}$$

$$S_h(t_1) \geq S_h(0) e^{-(\omega+\mu)t_1 - \int_0^{t_1} \lambda_M(\varepsilon) d\varepsilon} > 0 \text{ as } t_1 \to \infty. \tag{2.9}$$

Adopting the same process, it can be demonstrated that the remaining state variables $V_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_v(0) > 0, E_v(0) \geq 0$ and $I_v(0) \geq 0$ for all future values of t $(t > 0)$, and therefore, the proof ends. □

2.2.2. The invariant region

The region where the model's solutions are feasible is examined here, and such a region is bounded. This is accomplished by proving Theorem (2.2).

Theorem 2.2. *If $S_h(t), V_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t)$, and $I_v(t)$ are the solutions of the malaria model* (2.3) *having the corresponding starting conditions*

$$S_h(0), V_h(0), E_h(0), I_h(0), R_h(0), S_v(0), E_v(0), \text{ and } I_v(0),$$

and are non-negative for all values of time (i.e. $t > 0$). Then, the closed set

$$D = D_h \times D_v = \left\{ (S_h, V_h, E_h, I_h, R_h, S_v, E_v, I_v) \in R_+^8 : N_h \leq \frac{\Lambda_h}{\mu_h}, N_v \leq \frac{\Lambda_v}{\mu_v} \right\},$$

is positively invariant with non-negative starting values in R_+^8. Where

$$D_h = \left\{ (S_h, V_h, E_h, I_h, R_h) \in R_+^5 : N_h \leq \frac{\Lambda_h}{\mu_h} \right\},$$

and

$$D_v = \left\{ (S_v, E_v, I_v) \in R_+^3 : N_v \leq \frac{\Lambda_v}{\mu_v} \right\}.$$

Proof. The additions of the subclasses of humans and mosquitoes in model (2.3) are given by

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta I_h \quad \text{and} \quad \frac{dN_v}{dt} = \Lambda_v - \mu_v N_v.$$

It follows that

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h \quad \text{and} \quad \frac{dN_v}{dt} \leq \Lambda_v - \mu_v N_v.$$

Then

$$N_h(t) \leq N_h(0) \exp(-\mu_h t) + \frac{\Lambda_h}{\mu_h}(1 - \exp(-\mu_h t))$$

and

$$N_v(t) \leq N_v(0) \exp(-\mu_v t) + \frac{\Lambda_v}{\mu_v}(1 - \exp(-\mu_v t)).$$

If $N_v(0) \leq \frac{\Lambda_v}{\mu_v}$, then $N_v(t) \leq \frac{\Lambda_v}{\mu_v}$. In addition, if $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$, then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$. Thus, all solutions of the malaria model with starting values in D dwell in D for all times $t > 0$. This implies that D is positively invariant, and in D, the model is deemed biologically meaningful and mathematically well-posed. Therefore, the dynamics of the model (2.3) can be explored in region D. □

2.3. Malaria-free Equilibrium and Reproduction Number

Malaria-free equilibrium occurs when malaria is not present in the population. If

$$\Sigma_o = (S_h^o, V_h^o, E_h^o, I_h^o, R_h^o, S_v^o, E_v^o, I_v^o)$$

represents the malaria-free equilibrium. Then, finding the steady-state solution of (2.5) leads to

$$\Sigma_o = (S_h^o, V_h^o, 0, 0, 0, S_v^o, 0, 0), \tag{2.10}$$

where

$$S_h^o = \frac{\Lambda_h C_2}{C_1 C_2 - \chi\omega}, \qquad V_h^o = \frac{\omega \Lambda_h}{C_1 C_2 - \chi\omega}, \qquad S_v^o = \frac{\Lambda_v}{\mu_v}.$$

The method used in [42], [43], [44], [45], and [46] is adopted to calculate the reproduction number. If we define the matrices F (transmission terms) and V (transition terms) as follows:

$$F = \begin{bmatrix} 0 & 0 & 0 & \frac{\beta_h[(1-\sigma)V_h^o + S_h^o]}{N_h^o} \\ 0 & 0 & 0 & 0 \\ \frac{\beta_v \phi S_v^o}{N_h^o} & \frac{\beta_v \phi \eta S_v^o}{N_h^o} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \tag{2.11}$$

$$V = \begin{bmatrix} C_3 & 0 & 0 & 0 \\ -\kappa & C_4 & 0 & 0 \\ 0 & 0 & C_6 & 0 \\ 0 & 0 & -\gamma & \mu_v \end{bmatrix} \tag{2.12}$$

.

Then, $R_{vo} = \rho(FV^{-1})$, where ρ is the spectral radius of the dominant eigenvalue. Thus,

$$R_{vo} = \sqrt{\frac{\gamma\, \beta_h \beta_v \phi^2 S_v^o \left[(\eta\, \kappa\, + C_4)[(1-\sigma)\, V_h^o + S_h^o]\right]}{C_3 C_4 C_6 \mu_v N_h^{o^2}}}. \tag{2.13}$$

However, when vaccination is absent (that is, $V_h = 0$, with $\chi = 0, \omega = 0$), the control reproduction number becomes

$$R_o = \sqrt{\frac{\gamma\, \beta_h \beta_v \phi^2 S_v^0\, (\eta\, \kappa + C_4)}{C_3 C_4 C_6 \mu_v N_h^0}}. \tag{2.14}$$

Then, (2.13) can be written in terms of (2.14) as follows:

$$R_{vo} = R_o \sqrt{\frac{(1-\sigma)V_h^o + S_h^o}{N_h^o}}. \tag{2.15}$$

The fundamental quantity R_{vo} represents the control reproduction number which is the average number of malaria-infected cases resulting from a single infectious source when placed in a population where a fraction of susceptibles have been vaccinated, while R_o is the basic reproduction number which is the measure of the average number of malaria-infected cases arising from a single infectious source when brought in contact with the susceptible population [42, 47, 48, 49].

To qualitatively assess the impact of vaccines, we let the fraction of people vaccinated be f_v, then, from (2.15), we have

$$R_{vo} = R_o \sqrt{1 - \sigma f_v}. \tag{2.16}$$

Then, differentiating (2.16) partially with respect to f_v leads to

$$\frac{\partial R_{vo}}{\partial f_v} = -\frac{\sigma R_o}{2\sqrt{1-\sigma f_v}}. \tag{2.17}$$

For $0 < \sigma < 1$ and $0 < f_v < 1$, the equation in (2.17) illustrates the impact of the vaccine, which indicates that R_{vo} decreases as the population of vaccinated people increases. Thus, vaccination will have a positive effect on decreasing the burden of malaria.

Moreover, if the critical vaccination proportion needed to curb malaria is represented by f_{vc}, then from (2.16), when $R_{vo} = 1$, the critical number that must be vaccinated to control malaria is given by

$$f_{vc} = \frac{1}{\sigma}\left(1 - \frac{1}{R_o^2}\right), \tag{2.18}$$

provided that $R_{vo} < 1$ and $f_v > f_{vc}$. As a consequence of the above, the following result is obtained based on Theorem 2 of [50]

Lemma 2.3. *If $f_v > f_{vc}$, the malaria-free equilibrium is locally asymptotically stable when $R_{vo} < 1$ and otherwise, when $R_{vo} > 1$.*

Lemma 2.3 can be interpreted to mean that malaria can be curbed if control reproduction $R_{vo} < 1$.

The implication of the disease-free equilibrium (DFE), represented by (2.10), describes a state in which the infection has been completely eliminated, leaving only susceptible and vaccinated individuals in the population. To maintain this condition, the control reproduction number (2.13) must be kept below one ($R_{vc} < 1$).

2.4. Endemic Equilibrium of the Malaria Model

The equilibrium where malaria is prevalent in the population is explored. In the endemic case, the infected classes are nonzero. This is obtained by setting the right-hand side of (2.5) to zero and then finding the solutions for the values of the variables in the endemic state. Then, let the endemic equilibrium be represented by $\Sigma^e = (S_h^e, V_h^e, E_h^e, I_h^e, R_h^e, S_v^e, E_v^e, I_v^e)$. Therefore, we have

$$
\begin{aligned}
S_h^e &= \frac{\Lambda_h C_5C_4C_3(A\lambda_M^e + C_2)}{C_5C_4C_3(A\lambda_M^e + C_2)(\lambda_M^e + C_2) - [(A(\omega + \lambda_M^e) + C_2)\theta\tau\kappa\lambda_M^e + C_5C_4C_3\omega\chi]},\\
V_h^e &= \frac{\Lambda_h \omega C_5C_4C_3}{C_5C_4C_3(A\lambda_M^e + C_2)(\lambda_M^e + C_2) - [(A(\omega + \lambda_M^e) + C_2)\theta\tau\kappa\lambda_M^e + C_5C_4C_3\omega\chi]},\\
E_h^e &= \frac{\big(A(\omega + \lambda_M^e) + C_2\big)\Lambda_h\lambda_M^e C_5C_4C_3}{C_3\Big[C_5C_4C_3(A\lambda_M^e + C_2)(\lambda_M^e + C_2) - [(A(\omega + \lambda_M^e) + C_2)\theta\tau\kappa\lambda_M^e + C_5C_4C_3\omega\chi]\Big]},\\
I_h^e &= \frac{\big(A(\omega + \lambda_M^e) + C_2\big)\Lambda_h\kappa\lambda_M^e C_5C_4C_3}{C_4C_3\begin{bmatrix}\Big[C_5C_4C_3(A\lambda_M^e + C_2)(\lambda_M^e + C_2)\\ -[(A(\omega + \lambda_M^e) + C_2)\theta\tau\kappa\lambda_M^e + C_5C_4C_3\omega\chi]\Big]\end{bmatrix}},\\
R_h^e &= \frac{\big(A(\omega + \lambda_M^e) + C_2\big)\Lambda_h\kappa\tau\lambda_M^e C_5C_4C_3}{C_5C_4C_3\begin{bmatrix}\Big[C_5C_4C_3(A\lambda_M^e + C_2)(\lambda_M^e + C_2)\\ -[(A(\omega + \lambda_M^e) + C_2)\theta\tau\kappa\lambda_M^e + C_5C_4C_3\omega\chi]\Big]\end{bmatrix}},\\
S_v^e &= \frac{\Lambda_v}{\lambda_v^e + \mu_v},\\
E_v^e &= \frac{\Lambda_v\lambda_v^e}{C_6(\lambda_v^e + \mu_v)},\\
I_v^e &= \frac{\gamma\Lambda_v\lambda_v^e}{\mu_v C_6(\lambda_v^e + \mu_v)}.
\end{aligned}
\tag{2.19}
$$

If the forces of infection in equilibrium are represented by λ_M^e and λ_v^e and are given by

$$\lambda_M^e = \frac{\beta_h\phi I_v^e}{N_h^e}, \tag{2.20}$$

$$\lambda_v^e = \frac{\beta_v\phi(E_h^e + \eta I_h^e)}{N_h^e}. \tag{2.21}$$

Substituting (2.19) into (2.20) and (2.21) gives

$$\lambda_M^e = \frac{\beta_h\phi\gamma\,\lambda_v\Lambda_v\begin{bmatrix}C_5C_4C_3(A\lambda_M^e + C_2)(\lambda_M^e + C_2)\\ -\left[(A(\omega + \lambda_M^e) + C_2)\theta\tau\kappa\lambda_M^e + C_5C_4C_3\omega\chi\right]\end{bmatrix}}{\mu_v\Lambda_h C_6\,(\lambda_v + \mu_v)\begin{bmatrix}\Big(\lambda_M^e\,(\kappa\,\tau + \kappa\,C_5 + C_4C_5)\,(A(\omega + \lambda_M^e) + C_2)\\ +\omega\,C_3C_4C_5 + (A\lambda_M^e + C_2)\,C_5C_4C_3\Big)\end{bmatrix}}, \tag{2.22}$$

$$\lambda_v^e = \frac{\beta_v \phi \lambda_M^e C_5 \left(\eta\,\kappa + C_4\right)\left(A(\omega + \lambda_M^e) + C_2\right)}{\lambda_M^e \left(\kappa\,\tau + \kappa\, C_5 + C_4 C_5\right)\left(A(\omega + \lambda_M^e) + C_2\right) + \omega\, C_3 C_4 C_5 + \left(A\lambda_M^e + C_2\right) C_5 C_4 C_3}. \tag{2.23}$$

Substituting (2.23) into (2.22) leads to

$$P_1 \lambda_M^{e^4} + P_2 \lambda_M^{e^3} + P_3 \lambda_M^{e^2} + P_4 \lambda_M^e + P_5 = 0. \tag{2.24}$$

Where

$$\begin{aligned}
P_1 &= LC_6 A^2 \mu_v \Lambda_h \big(C_5 \phi \beta_v \left(\eta\,\kappa + C_4\right) + \mu_v L\big),\\
P_2 &= C_5 \phi \beta_v \left(\eta\,\kappa + C_4\right)\Big(A\left(\Lambda_v \gamma\, \beta_h \phi \left(\kappa\,\tau\,\theta - J\right) + C_6 \Lambda_h \mu_v \left(2\,L\omega + J\right)\right) A\\
&\quad + 2\, C_6 \Lambda_h \mu_v L C_2\Big) + 2\,{\mu_v}^2 \Lambda_h C_6 L\Big(A\left(L\omega + J\right) + C_2 L\Big),\\
P_3 &= C_5 \phi \beta_v \left(\eta\,\kappa + C_4\right)\Big(\big[\beta_h \Lambda_v \gamma\, \phi\left(\left(-\omega - C_1\right) J + 2\,\omega\,\theta\,\tau\,\kappa\right) + \omega\, C_6 \Lambda_h \mu_v \left(L\omega + J\right)\big] A^2\\
&\quad + \big[2\, C_2 \Lambda_v \beta_h \gamma\, \phi\left(\kappa\,\tau\,\theta - J\right) + C_6 \Lambda_h \mu_v \left(\left(\omega + C_2\right) J + 2\,\omega\, C_2 L\right)\big] A + C_6 \Lambda_h \mu_v L {C_2}^2\Big)\\
&\quad + {\mu_v}^2 \Lambda_h C_6 \Big(\left(L\omega + J\right)^2 A^2 + 2\, L\big[\left(\omega + 2\, C_2\right) J + \omega\, C_2 L\big] A + {C_2}^2 L^2\Big),\\
P_4 &= C_5 \beta_v \phi \left(\eta\,\kappa + C_4\right)\Big(J\big(C_6 \Lambda_h \mu_v \left(\omega + C_2\right)\left(A\omega + C_2\right)\\
&\quad - \gamma\, \beta_h \Lambda_v \phi\big({C_2}^2 + AC_2\left(\omega + 2\, C_1\right) + A\omega\ \left(AC_1 - \chi\right)\big)\big)\\
&\quad + \theta\, \Lambda_v \beta_h \phi \tau\, \gamma\, \kappa\ \left(A\omega + C_2\right)^2\Big) + 2\,{\mu_v}^2 \Lambda_h C_6 J\big(AJ + L\left(A\omega + C_2\right)\big)\left(\omega + C_2\right),\\
P_5 &= \Lambda_h \mu_v^2 C_3 C_4 C_5 C_6 J(\omega + C_2)^2 (1 - R_{vo}^2).
\end{aligned} \tag{2.25}$$

and

$$\begin{aligned}
L &= C_4 C_5 + C_5 \kappa + \tau\kappa\\
A &= 1 - \sigma,\\
J &= C_3 C_4 C_5.
\end{aligned} \tag{2.26}$$

Clearly, from (2.24) and (2.25), $P_1 > 0$ and $P_5 > 0$ when $R_{vo} < 1$, and $P_5 < 0$ when $R_{vo} > 1$. Then, the number of roots that will be positive will depend on the signs of P_4, P_3, P_2. This can be determined by employing the Descartes rule of signs in (2.24). Table 2 lists the possible numbers of positive roots that (2.24) can have.

Thus, from Table 2, the following theorem is established.

Theorem 2.4. *The malaria model* (2.5) *possesses:*

1. *no endemic equilibrium if Case* 1 *holds and that* $R_{ov} < 1$.
2. *two or four endemic equilibria if the Cases* $2 - 8$ *hold and* $R_{ov} < 1$.
3. *one or three endemic equilibria if the Cases* $9 - 16$ *hold and* $R_{ov} > 1$.

Hence, the presence of multiple endemic equilibria when $R_{ov} < 1$ indicates that the model may exhibit a backward bifurcation. This is a scenario in which a stable malaria-free state coexists with an endemic equilibrium. In the next Subsection 2.5, the possibility of the occurrence of a backward bifurcation is investigated.

Table 2: The possible number of sign changes.

Cases	P_1	P_2	P_3	P_4	P_5	R_{vo}	No of sign changes	No of possible positive roots
1	+	+	+	+	+	<1	0	0
2	+	+	+	−	+	<1	2	0 or 2
3	+	+	−	+	+	<1	2	0 or 2
4	+	−	+	+	+	<1	2	0 or 2
5	+	−	−	+	+	<1	2	0 or 2
6	+	−	−	−	+	<1	2	0 or 2
7	+	−	+	−	+	<1	4	0 or 2 or 4
8	+	+	−	−	+	<1	2	0 or 2
9	+	+	+	+	−	>1	1	1
10	+	+	+	−	−	>1	1	1
11	+	+	−	+	−	>1	3	1 or 3
12	+	−	+	+	−	>1	3	1 or 3
13	+	−	−	+	−	>1	3	1 or 3
14	+	−	−	−	−	>1	1	1
15	+	−	+	−	−	>1	3	1 or 3
16	+	+	−	−	−	>1	3	1

2.5. Bifurcation Analysis

Bifurcation is a phenomenon associated with a change in the behaviour of the system as a result of a change in one of the parameters of the system. The methodology used by Castillo-Chavez and Song [51] is used to investigate this phenomenon. The Centre Manifold Theory [52] used by Castillo-Chavez and Song [51] and implemented in [53], is used to establish the form of bifurcation that the model will exhibit. Let the variables in the model be written as follows:

$$S_h = l_1, V_h = l_2, E_h = l_3, I_h = l_4, R_h = l_5, S_v = l_6, E_v = l_7, I_v = l_8,$$

and that

$$N_h = l_1 + l_2 + l_3 + l_4 + l_5. \tag{2.27}$$

Thus, the malaria model (2.5) can be rewritten as follows:

$$\begin{aligned}
l_1' &= \Lambda_h - \lambda_M l_1 + \theta l_5 + \chi l_2 - C_1 l_1 = h_1,\\
l_2' &= \omega l_1 - (1-\sigma)\lambda_M l_2 - C_2 l_2 = h_2\\
l_3' &= \lambda_M l_1 + (1-\sigma)\lambda_M l_2 - C_3 l_3 = h_3,\\
l_4' &= \kappa l_3 - C_4 l_4 = h_4,\\
l_5' &= \tau l_4 - C_5 l_5 = h_5,\\
l_6' &= \Lambda_v - \lambda_v l_6 - \mu_v l_6 = h_6,\\
l_7' &= \lambda_v l_6 - C_6 l_7 = h_7,\\
l_8' &= \gamma l_7 - \mu_v l_8 = h_8,
\end{aligned} \tag{2.28}$$

where

$$\begin{aligned}
\lambda_M &= \frac{\beta_h \phi l_8}{l_1 + l_2 + l_3 + l_4 + l_5},\\
\lambda_v &= \frac{\beta_v \phi (l_3 + \eta l_4)}{l_1 + l_2 + l_3 + l_4 + l_5}.
\end{aligned} \tag{2.29}$$

If β_h=β_h^f is assumed to be the bifurcation parameter at $R_{vo}=1$, then from (2.13), we have

$$\beta_h = \beta_h^f = \frac{C_3C_4C_6\mu_v {N_h}^{o^2}}{\gamma\,\phi^2\beta_v S_v^o\left([1-\sigma]\,V_h^o + S_h^o\right)(\eta\,\kappa + C_4)} \tag{2.30}$$

The Jacobian of (2.28) at Σ_o denoted by $J_{|_{\Sigma_o}}$ is given by

$$J_{|_{\Sigma_o}} = \begin{bmatrix} -C_1 & \chi & 0 & 0 & \theta & 0 & 0 & \frac{-\beta_h^f\phi S_h^o}{S_h^o+V_h^o} \\ \omega & -C_2 & 0 & 0 & 0 & 0 & 0 & \frac{-(1-\sigma)\beta_h^f\phi V_h^o}{S_h^o+V_h^o} \\ 0 & 0 & -C_3 & 0 & 0 & 0 & 0 & \frac{\beta_h^f\phi(S_h^o+[1-\sigma]V_h^o)}{S_h^o+V_h^o} \\ 0 & 0 & \kappa & -C_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & -C_5 & 0 & 0 & 0 \\ 0 & 0 & \frac{-\beta_v\phi S_v^o}{S_h^o+V_h^o} & \frac{-\beta_v\phi\eta S_v^o}{S_h^o+V_h^o} & 0 & -\mu_v & 0 & 0 \\ 0 & 0 & \frac{\beta_v\phi S_v^o}{S_h^o+V_h^o} & \frac{\beta_v\phi\eta S_v^o}{S_h^o+V_h^o} & 0 & 0 & -C_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma & -\mu_v \end{bmatrix}, \tag{2.31}$$

and has a simple zero eigenvalue at $R_{vo}=1$, and thus we can apply the Center Manifold Theory [52]. Also, $J_{|_{\Sigma_o}}$ has a right eigenvector $\mathbf{p}=(p_1,p_2,p_3,p_4,p_5,p_6,p_7,p_8)^T$ where

$$\begin{aligned} &p_1 = p_1 > 0, && p_2 = \frac{(S_h^o+V_h^o)\omega p_1 - (1-\sigma)\beta_h^f\phi V_h^o p_8)}{C_2(S_h^o+V_h^o)}, \\ &p_3 = \frac{C_4p_4}{\kappa}, && p_4 = \frac{C_5p_5}{\tau}, \\ &p_5 = p_5 > 0, && p_6 = -\frac{\beta_v\phi S_v^o(p_3+\eta p_4)}{\mu_v}, \\ &p_7 = \frac{\beta_v\phi S_v^o(p_3+\eta p_4)}{C_6(S_h^o+V_h^o)}, && p_8 = \frac{\gamma p_7}{\mu_v}. \end{aligned} \tag{2.32}$$

and left eigenvector $\mathbf{q}=(q_1,q_2,q_3,q_4,q_5,q_6,q_7,q_8)^T$, such that $\mathbf{p}\cdot\mathbf{q}=1$, where

$$\begin{aligned} &q_1 = \frac{\omega q_2}{C_1}, && q_2 = q_2 > 0, && q_3 = \frac{\kappa q_4(S_h^o+V_h^o)+\beta_v\phi S_v^o q_7}{C_3(S_h^o+V_h^o)}, \\ &q_4 = \frac{\tau q_5(S_h^o+V_h^o)+\beta_v\phi\eta S_v^o q_7}{C_4(S_h^o+V_h^o)}, && q_5 = \frac{\theta q_1}{C_5}, && q_6 = 0, \\ &q_7 = q_7 > 0, && q_8 = \frac{\beta_h^f\phi(S_h^o+[1-\sigma]V_h^o)q_3 - \beta_h^f\phi(S_h^o q_1+[1-\sigma]V_h^o q_2)}{\mu_v(S_h^o+V_h^o)}. \end{aligned} \tag{2.33}$$

By finding the associated non-zero partial derivative of $h(l)$ in (2.28) at Σ_o, the associated bifurcation coefficients a and b defined by

$$a = \sum_{k,i,j=1}^{n} q_k p_i p_j \frac{\partial^2 h_k}{\partial l_i \partial l_j}(0,0) \qquad \text{and} \qquad b = \sum_{k,i=1}^{n} q_k p_i \frac{\partial^2 h_k}{\partial l_i \partial \beta_h^f}(0,0), \tag{2.34}$$

are obtained as follows:

$$a = \frac{2\phi}{(S_h^o + V_h^o)^2}\begin{bmatrix} \beta_v(\eta p_4 + p_3)\big[p_6(S_h^o + V_h^o) - S_v^o(p_1 + p_2 + p_3 + p_4 + p_5)\big] \\ +\beta_h^f p_8\Big((p_3 + p_4 + p_5)\big[V_h^o(1-\sigma)(q_2 - q_3) + S_h^o(q_1 - q_2)\big] \\ +(p_1 V_h^o - p_2 S_h^o)[q_2(1-\sigma) + q_3\sigma - q_1]\Big) \end{bmatrix}, \tag{2.35}$$

and

$$b = \frac{\phi p_8\big[(q_3 - q_2)(1-\sigma)V_h^o + (q_3 - q_1)S_h^o\big]}{S_h^o + V_h^o}. \tag{2.36}$$

From (2.36), $b > 0$ if $q_3 > q_2$ and $q_3 > q_1$, and thus we have the following result:

Theorem 2.5. *The malaria model* (2.3) *will exhibit a backward bifurcation if* $a > 0$ *and otherwise if* $a < 0$

The system exhibits backward bifurcation when $a > 0$, which implies that even if the basic reproduction number (R_0) is less than 1, malaria can still persist. This requires stronger intervention strategies beyond the simple reduction of R_0.

The implication of the model exhibiting a backward bifurcation phenomenon is that the basic necessary condition of having $R_{vo} < 1$ for the elimination of the disease will not suffice for the control of the disease. This implies that the control of the disease will be too cumbersome.

2.6. Global Stability of Endemic Equilibrium of the Malaria Model

The global asymptotic property of the endemic equilibrium of the malaria model is investigated to determine whether the initial values can influence the persistence of malaria when $R_{vo} > 1$.

Theorem 2.6. *If* $\Sigma^e = (S_h^e, V_h^e, E_h^e, I_h^e, R_h^e, S_v^e, E_v^e, I_v^e)$ *represents the endemic equilibrium and* $D^o = \{(S_h, V_h, E_h, I_h, R$ $D : E_h, I_h, R_h, E_v, I_v = 0\}$ *is a stable manifold of the malaria-free equilibrium, then* Σ^e *is globally asymptotically stable in* $D \backslash D^o$ *when* $R_{vo} > 1$.

Proof. The Lyapunov function stated in (2.37) is used to prove the global stability of the endemic equilibrium.

$$\begin{aligned} Z =& \frac{1}{2}\Big[(S_h - S_h^e) + (V_h - V_h^e) + (E_h - E_h^e) + (I_h - I_h^e) + (R_h - R_h^e)\Big]^2 \\ &+ \frac{1}{2}\Big[(S_v - S_v^e) + (E_v - E_v^e) + (I_v - I_v^e)\Big]^2 \end{aligned}. \tag{2.37}$$

Differentiating (2.37) gives

$$\begin{aligned} Z' =& \Big[(S_h - S_h^e) + (V_h - V_h^e) + (E_h - E_h^e) + (I_h - I_h^e) + (R_h - R_h^e)\Big]\times \\ &\Big(\frac{d}{dt}\big(S_h(t) + V_h(t) + E_h(t) + I_h(t) + R_h(t)\big)\Big) + \Big[(S_v - S_v^e) + (E_v - E_v^e) + (I_v - I_v^e)\Big]\times \\ &\Big(\frac{d}{dt}\big(S_v(t) + E_v(t) + I_v(t)\big)\Big). \end{aligned} \tag{2.38}$$

By substituting the right-hand side of (2.3) and adding, we have

$$\begin{aligned} Z' =& \Big[(S_h + V_h + E_h + I_h + R_h) - (S_h^e + V_h^e + E_h^e + I_h^e + R_h^e)\Big]\times \\ &\big(\Lambda_h - \mu_h(S_h + V_h + E_h + I_h + R_h) - \delta I_h\big) \\ &\Big[(S_v + E_v + I_v) - (S_v^e + E_v^e + I_v^e)\Big] \times \big(\Lambda_v - \mu_v(S_v + E_v + I_v\big) \end{aligned} \tag{2.39}$$

$$\begin{aligned} Z' \leq & \Big[(S_h + V_h + E_h + I_h + R_h) - (S_h^e + V_h^e + E_h^e + I_h^e + R_h^e)\Big] \times \\ & \big(\Lambda_h - \mu_h(S_h + V_h + E_h + I_h + R_h)\big) \\ & \Big[(S_v + E_v + I_v) - (S_v^e + E_v^e + I_v^e)\Big] \times \big(\Lambda_v - \mu_v(S_v + E_v + I_v\big), \end{aligned} \tag{2.40}$$

$$\begin{aligned} Z' \leq & -\mu_h\Big[(S_h + V_h + E_h + I_h + R_h) - (S_h^e + V_h^e + E_h^e + I_h^e + R_h^e)\Big] \times \\ & \left((S_h + V_h + E_h + I_h + R_h) - \frac{\Lambda_h}{\mu_h}\right) - \mu_h\Big[(S_v + E_v + I_v) - (S_v^e + E_v^e + I_v^e)\Big] \times \\ & \left((S_v + E_v + I_v) - \frac{\Lambda_v}{\mu_v}\right). \end{aligned} \tag{2.41}$$

But $N_h^e = \dfrac{\Lambda_h}{\mu_h}$ and $N_v^e = \dfrac{\Lambda_v}{\mu_v}$, then we have

$$\begin{aligned} Z' = & -\mu_h\Big[(S_h - S_h^e) + (V_h - V_h^e) + (E_h - E_h^e) + (I_h - I_h^e) + (R_h - R_h^e)\Big]^2 \\ & -\mu_v\Big[(S_v - S_v^e) + (E_v - E_v^e) + (I_v - I_v^e)\Big]^2. \end{aligned} \tag{2.42}$$

Therefore, $Z' < 0$ if $\mathcal{R}_{vo} > 1$. In addition, $Z' = 0$ if and only if $S_h = S_h^e, V_h = V_h^e, E_h = E_h^e, I_h = I_h^e, R_h = R_h^e, S_v = S_v^e, E_v = E_v^e, I_v = I_v^e$. Then, by the LaSalle Invariance Principle [54], every trajectory of the model converges to Σ^e when $\mathcal{R}_{vo} > 1$. Therefore, the endemic equilibrium is globally asymptotically stable when $\mathcal{R}_{vo} > 1$.

. □

The inference that can be drawn from Theorem 2.6 is that malaria will remain in the population regardless of the size of the initial population when $R_{vo} > 1$. This underscores the need for continuous large-scale intervention efforts rather than temporary measures to eliminate the disease. The global stability of the endemic equilibrium in a malaria model has significant implications for disease control and public health strategies.

2.7. Uncertainty and Sensitivity Analysis

Due to the many parameters involved in the model, uncertainty will be experienced in the system. The Latin Hypercube Sampling (LHS)/Pearson Rank Correlation Coefficient (PRCC) technique is applied to measure the level of uncertainty and examine how parameter changes affect the output. The Latin Hypercube Sampling (LHS) method is used to sample the sixteen parameters that constitute the expression of the control reproduction number, and the PRCC method is used to rank the sensitivity indices of the control reproduction number. The LHS/PRCC approach is a powerful method for conducting global sensitivity analysis. The signs in the PRCCs indicate the relationship between the output R_{ov} and the parameters. It should be noted that the sensitivity analysis helps in making predictions for the effective measures to be considered to mitigate the burden of the disease in the population. A parameter with a negative PRCC indicates that it is negatively correlated, and a parameter with a positive PRCC indicates that it is positively correlated with the control reproduction number. In addition, when the absolute value of the PRCC of a parameter is equal to or greater than 0.5, that parameter is statistically significant for the spread dynamics and control of the disease. The results of the calculated PRCCs are presented in Table 3 and are also represented by a bar graph in Figure 2.

Table 3: Partial Rank Correlation Coefficients (PRCCs) values

Parameter	PRCC	Parameter	PRCC
β_h	0.57813	κ	0.00272
ϕ	0.75494	η	0.47751
β_v	0.50371	σ	-0.62608
Λ_v	0.56738	ω	-0.07270
δ	-0.00426	χ	0.02314
γ	0.34302	Λ_h	-0.08375
τ	-0.50687	μ_h	0.11146
μ_v	-0.78322		

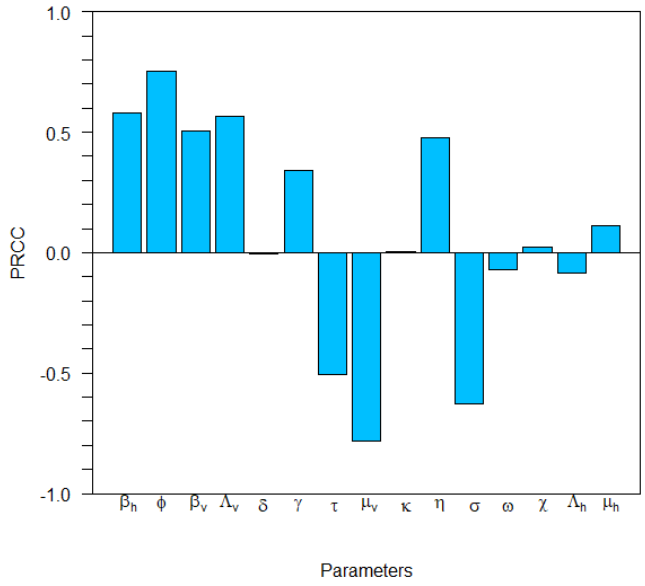

Figure 2: Bar plot of the PRCC when R_{vo} acts as the response function

3. Results and Discussion

A mathematical model of malaria that takes into account vaccination is formulated and analysed. The control reproduction number was obtained. The existence of the endemic state was studied, and it was discovered that multiple equilibria exist when $R_{vo} < 1$. Further analysis revealed that the system may undergo a phenomenon known as backward bifurcation, in which malaria-free and endemic states occur simultaneously when the associated bifurcation parameter $a > 0$. The stability analysis of the endemic state shows that it is globally asymptotically stable. From the results of the sensitivity analysis shown in Table 3, the most significant parameters in the dynamics of malaria are the probability of transmission from infected individuals to mosquitoes (β_h), biting rate (ϕ), probability of transmission from mosquitoes to humans (β_v), mosquito recruitment rate (Λ_v), treatment rate (τ), mosquito death rate (μ_v), and vaccine efficacy (σ). Parameters $\beta_h, \phi, \beta_v,$ and Λ_v are positively correlated, which means that any increase in the value of these parameters will exacerbate the transmission of malaria in the population. Therefore, measures such as the use of long-lasting insecticidal nets and spraying of insecticides should be encouraged to reduce contact between mosquitoes and humans. Furthermore, the parameters $\tau, \mu_v,$ and σ are negatively correlated, indicating that these parameters are capable of reducing the transmission of malaria when they are increased. Hence,

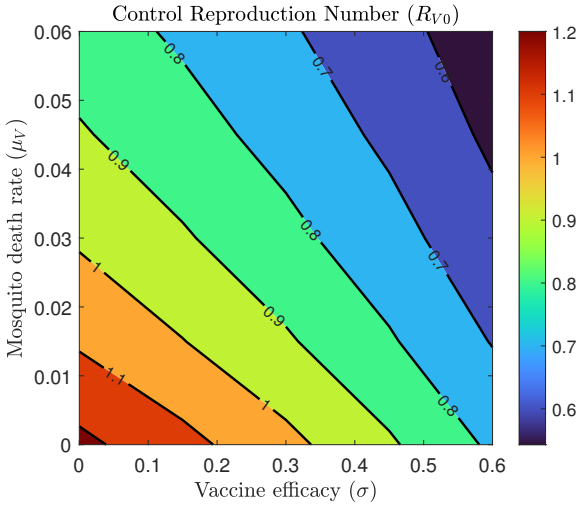

Figure 3: Contour plot of the control reproduction number (R_{vo}) of malaria as a function of mosquito death rate (μ_V) and efficacy of vaccine (σ)

serious attention should be given to these parameters that can reduce the control reproduction number by implementing a robust treatment programme for infectious individuals, adopting insecticides to depopulate the mosquito population, and utilising highly efficacious vaccines.

In order to measure the impact of the vaccine and other notable parameters associated with the model, numerical experiments are carried out. The values in Table 4 are used to simulate the model. The simulation results are visualised in Figures 3–14

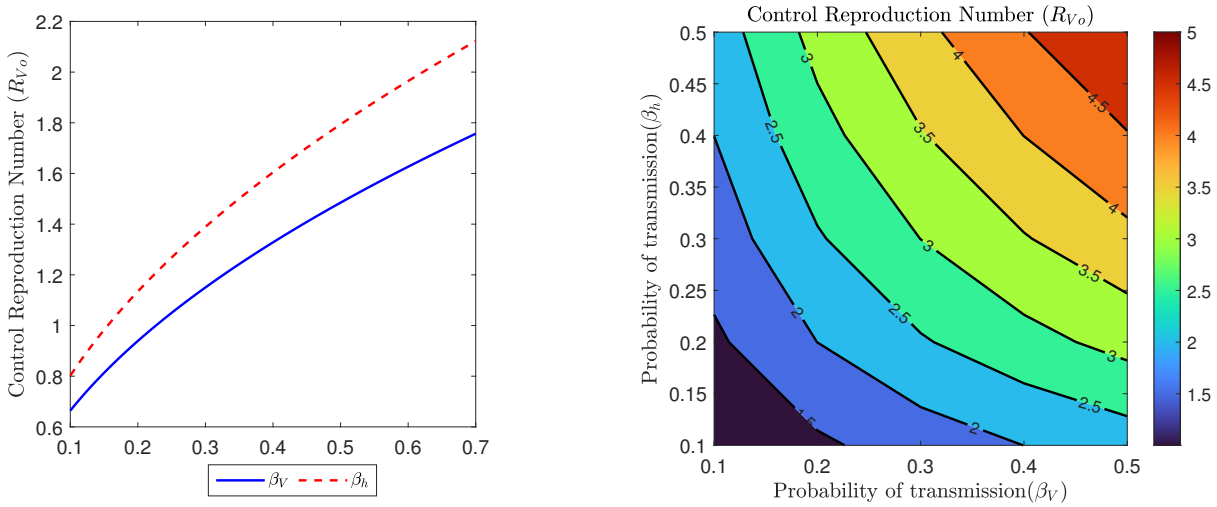

Figure 4: Plots of (R_{vo}) as a function of transmission probabilities (β_v) and (β_h)

Table 4: Values of the model's parameters

Parameter	Range	Value	Ref.
μ_h	[0.0000342-0.0000472]	0.0000421	[26]
η	$[1-10]$	7	Assumed
κ	[0.067-0.1]	0.1	[55]
ω	[0.2-0.8]	0.2	Assumed
ϕ	[0.1-1.0]	0.5	[56]
β_h	[0.01-0.27]	0.22	[26]
γ	[0.029-0.33]	0.0555	[26, 57]
δ	[0.00001-0.001]	0.000505	[36]
χ	[0.00055-0.0056]	0.003074	Assumed
τ	[0.0014-0.017]	0.0092	[18, 58]
Λ_h	[0.034-0.047]	0.0421	[26]
Λ_v	[0.05-1.0]	0.525	[59]
β_v	[0.072-0.64]	0.321	[18]
σ	[0-1)	0.1	Assumed
μ_v	[0.048-0.33]	0.048	[60]
θ	[0.0001-0.009]	0.0005275	[26, 61]

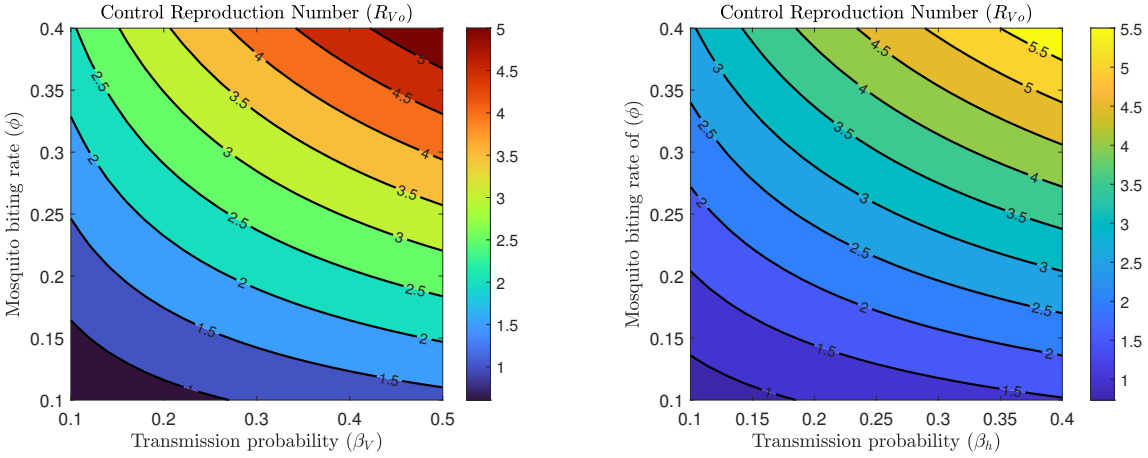

Figure 5: Contour plots of R_{vo} as a function of transmission probabilities (β_h, β_v) and biting rate (ϕ)

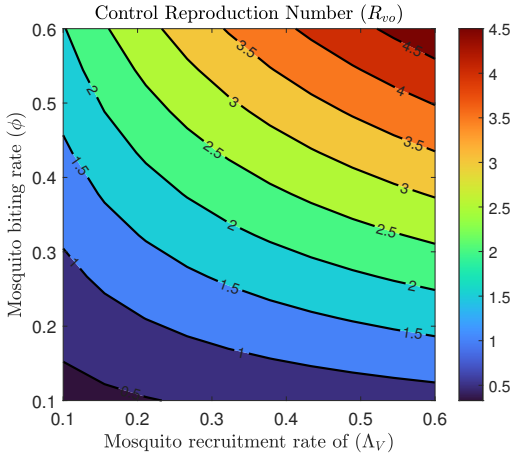

Figure 6: Contour plot of R_{vo} with respect to mosquito recruitment rate (Λ_V) and biting rate (ϕ)

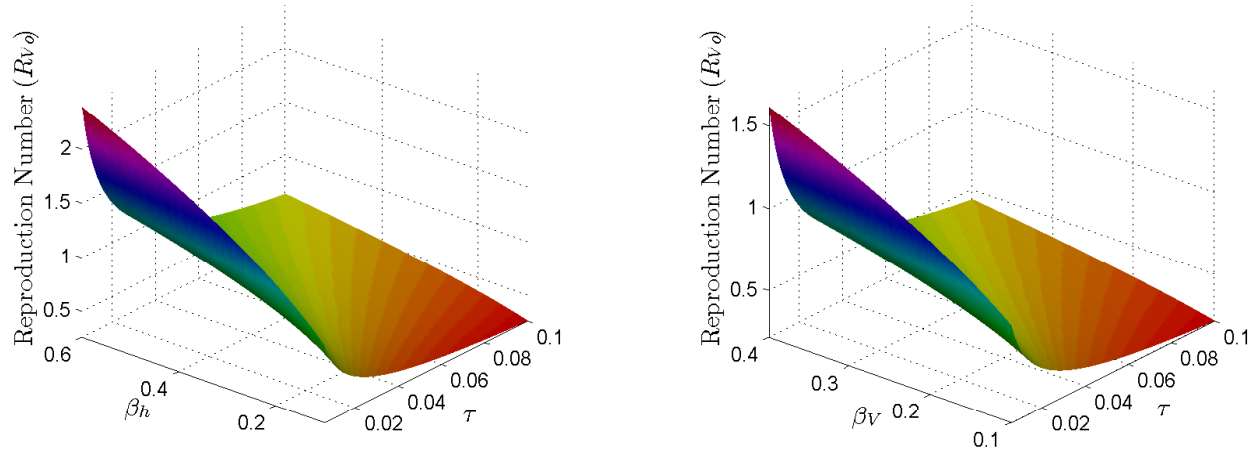

Figure 7: 3-D graphs showing the effects of transmission probabilities (β_h, β_v) and treatment rate (τ) of the infectious people on the control reproduction number

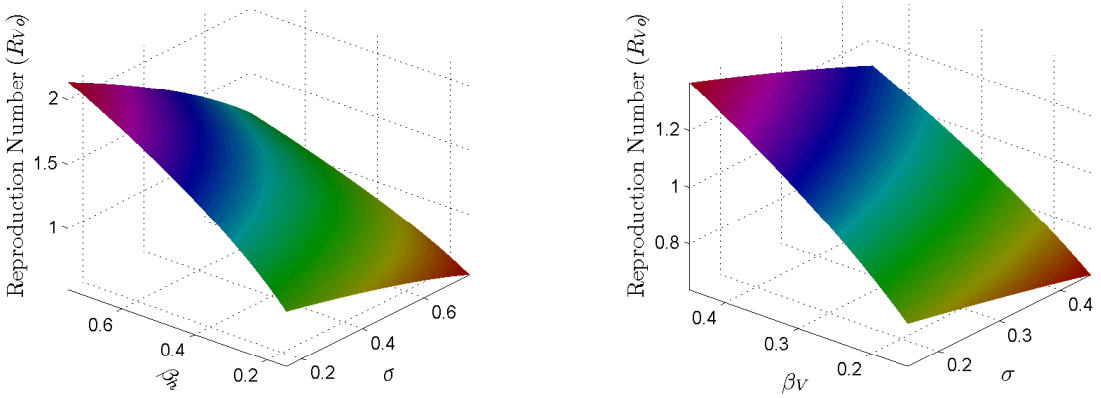

Figure 8: Effects of transmission probabilities (β_h, β_v) and the vaccine efficacy on the control reproduction number (R_{vo}) in 3-D

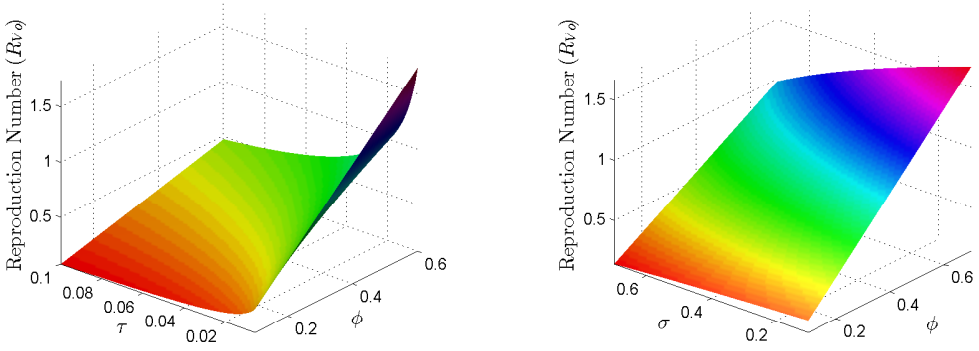

Figure 9: 3-D graphs showing the effects of treatment rate, vaccine efficacy and mosquito biting rate on the control reproduction number (R_{vo})

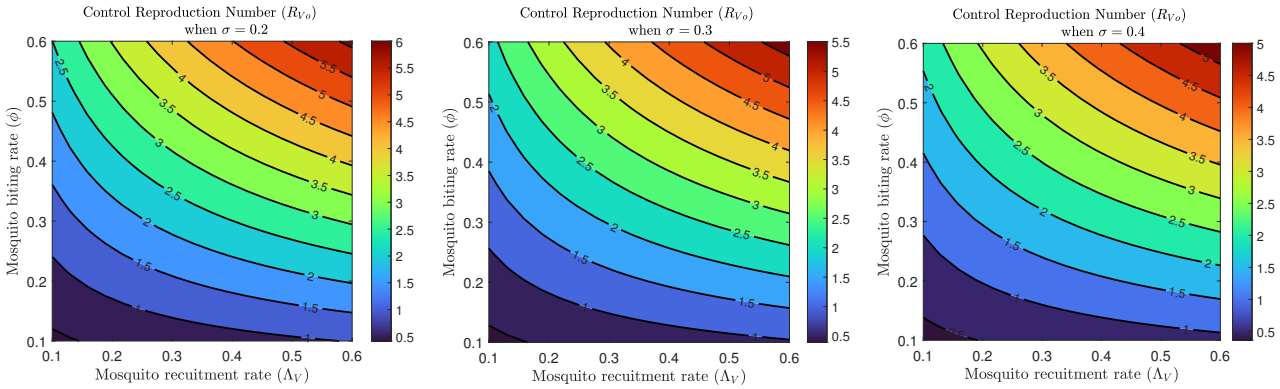

Figure 10: Contour plots of R_{vo} as a function of mosquito recruitment rate (Λ_V) and biting rate (ϕ) for different values of vaccine efficacy (σ)

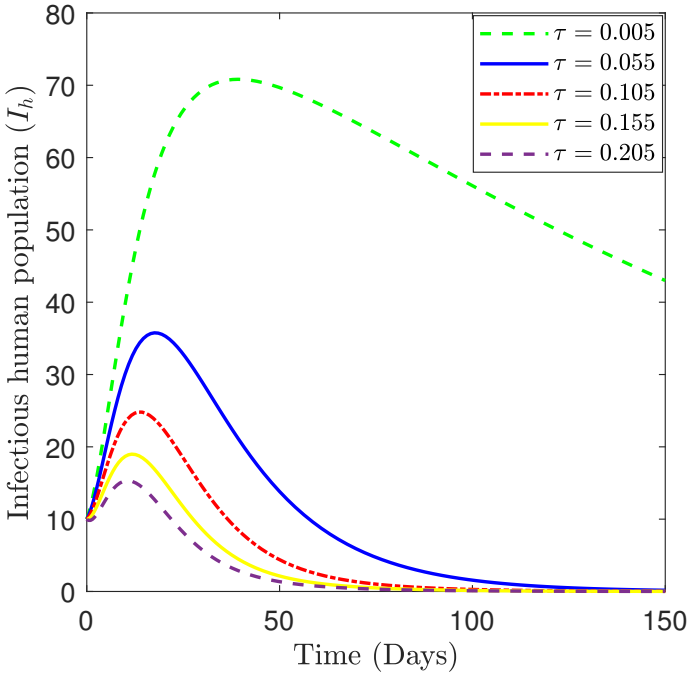

Figure 11: Effect of treatment on infectious human population

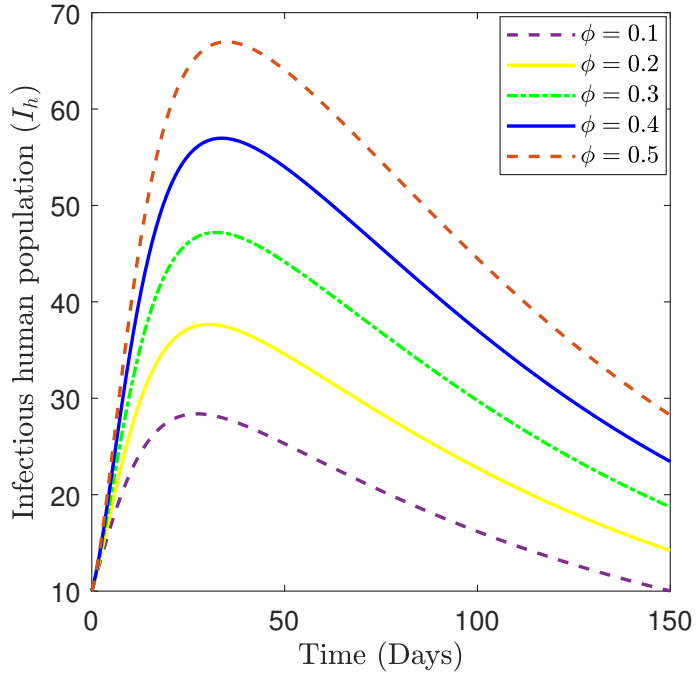

Figure 12: Dynamics of the infectious human population at different biting rates

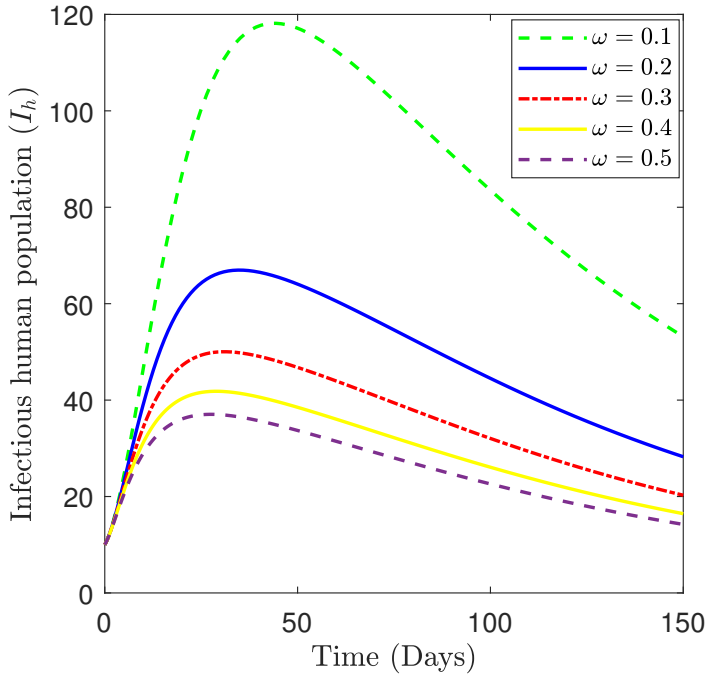

Figure 13: Solution curves for the infectious human population at different vaccination rates

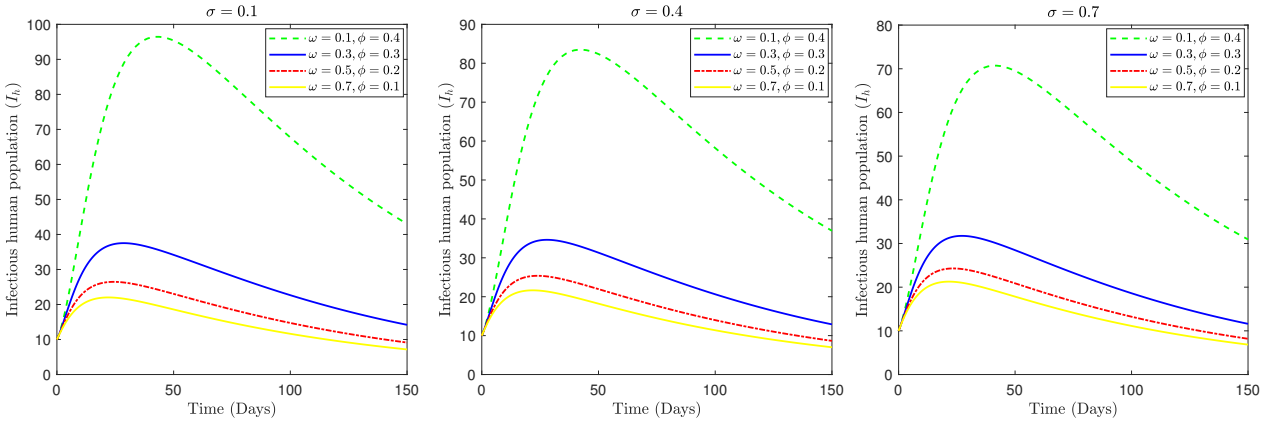

Figure 14: Dynamics of infectious human compartment when the vaccination rate (ω), biting rate (ϕ), and vaccine efficacy (σ) are varied.

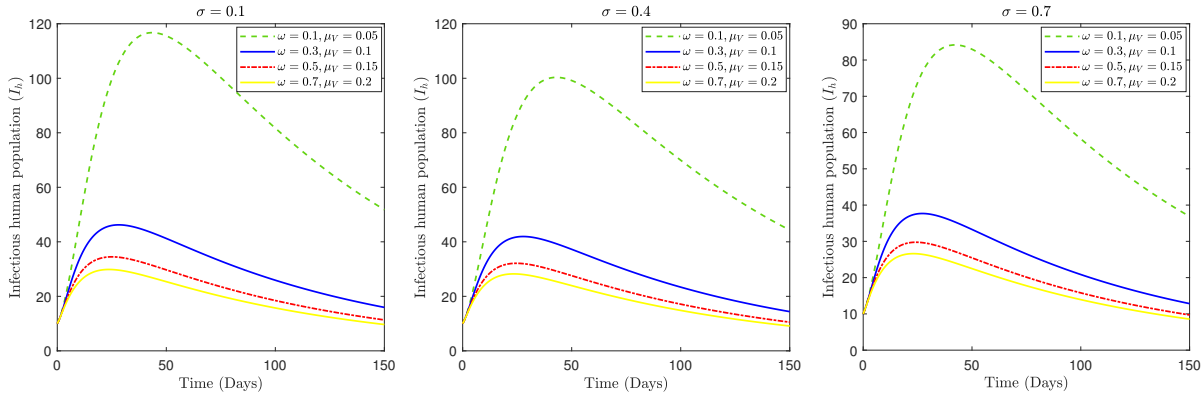

Figure 15: Graphs of the population sizes of infectious individuals when the vaccination rate (ω), mosquito death rate (μ_v), and vaccine efficacy (σ) are varied.

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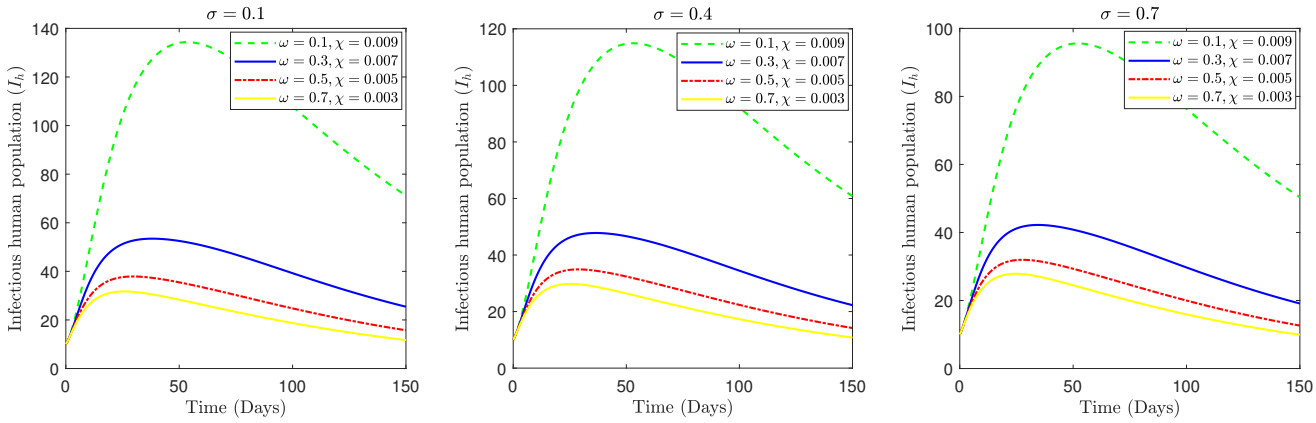

Figure 16: Trajectories of the infectious human compartment when the vaccination rate (ω) and rate at which the vaccine wanes (χ) are varied, for different values of vaccine efficacy (σ)

The plot in Figure 3 displays the contour diagram of the control reproduction number in terms of the mortality rate (μ_v) of mosquitoes and the efficacy of the vaccine (σ). The figure reveals that the control reproduction number can be reduced if both vaccine efficacy and the mosquito death rate are high. Therefore, efforts must be directed toward the production of high-quality vaccines and the increase of mosquito mortality rate to reduce malaria endemicity. The illustrations in Figure 4 indicate that the control reproduction number (R_{vo}) increases as the transmission probabilities (β_h, β_v) increase, which agrees with the interpretation of the results of the sensitivity analysis obtained through PRCC. In addition, Figure 5 illustrates the contour plots of the control reproduction number (R_{vo}) as a function of the transmission probabilities (β_h, β_v) and mosquito biting rate (ϕ). The plots show that malaria can be mitigated if the bite rate of mosquitoes can be lowered, as well as the probabilities (β_h, β_v) of malaria transmission. Therefore, control efforts should be geared toward reducing the mosquito biting rate (ϕ) and transmission probabilities (β_h, β_v) to reduce malaria cases. Furthermore, the graphical illustration in Figure 6 shows the effects of the mosquito recruitment and biting rates on the control reproduction number. It can be deduced that with low mosquito recruitment and biting rates, there will be no substantial malaria outbreaks. Control measures like the destruction of the mosquito breeding places and the use of ITNs should be encouraged to reduce the spread of malaria. The 3D plots in Figure 7 illustrate the effects of transmission probabilities (β_h, β_v) and treatment rate (τ). These graphs can be interpreted to mean that if the malaria transmission probabilities (β_h, β_v) are low and the rate of treating infectious individuals is high, then malaria transmission will be low in the society. Similarly, Figure 8 represents the impacts of transmission probabilities (β_h, β_v) and vaccine efficacy on the control reproduction in 3D. Increasing the transmission probabilities (β_h, β_v) and decreasing vaccine efficacy increase the control reproduction number, thereby making malaria endemic in the society. The graphs in Figure 9 demonstrate the impact of the treatment rate (τ), the vaccine efficacy (σ) and mosquito biting rate ϕ on the threshold metric (R_{vo}). It is observed that increasing the treatment rate and vaccine efficacy, while reducing the biting rate, would lead to a malaria-free situation. Hence, a robust treatment programme, the production of highly effective vaccines, and strategies to reduce the mosquito

bite rate should be considered in the fight against the disease. The contour plots in Figure 10 indicate that with low vaccine efficacy, malaria transmission can be subdued if the mosquito bites and recruitment rates are low in the population.

Moreover, Figure 11 shows that with vaccination put in place, the infectious human population can be reduced if supported by a high treatment rate of infectious individuals. The graphical illustration in Figure 12 shows the population of infectious individuals when the biting rate is varied. The biting rate with the highest value has the highest peak and the largest final population size, which means that the biting rate must be controlled to minimise malaria transmission. This can be achieved through the use of long-lasting insecticidal nets. In addition, the increasing effect of vaccination is shown in Figure 13, and the population of infectious individuals decreases as the rate of vaccination increases. Therefore, a robust vaccination programme must be considered to vaccinate more susceptible people. The graphs presented in Figure 14 show that malaria burden can be mitigated when a highly effective vaccine is used, coupled with a high vaccination rate among susceptible individuals and a low mosquito biting rate. Thus, control efforts aimed at having a highly efficacious vaccine, increasing the vaccination rate, and using long-lasting insecticidal nets to reduce the biting rate should be adopted to curb the transmission dynamics of malaria. In addition, Figure 15 shows that the efficacy of the vaccine, vaccination rates, and the death of mosquitoes must be high to reduce the population of infectious individuals. Therefore, control measures, such as the use of highly effective vaccines and spraying insecticides to increase mosquito mortality, should be considered. Finally, Figure 16 shows that with high vaccine efficacy, a high vaccination rate, and a low vaccine waning rate, malaria transmission can be reduced. Hence, emphasis should be placed on the production and use of highly efficacious vaccines with low waning rates, as well as on increasing vaccination coverage among susceptible individuals.

4. Conclusion

A mathematical model of malaria with vaccination has been developed and studied to obtain quantitative information on the impacts of vaccination and the efficacy of the vaccine in eradicating malaria. We obtained the malaria-free equilibrium state and control reproduction number. The study of the endemic state of the malaria model shows that there exist multiple equilibria when $R_{vo} < 1$, which thus gives rise to the appearance of a backward bifurcation when the bifurcation coefficient $a > 0$. The endemic state is shown to be globally asymptotically stable when $R_{vo} > 1$. The sensitivity analysis conducted using $LHS/PRCC$ indicates that transmission probabilities (β_h and β_v), mosquito biting rate, mosquito recruitment rate, treatment rate, mosquito death rate, and vaccine efficacy are the main components that can significantly alter the dynamics of malaria spread and control. The simulation results indicated that to effectively combat the spread of malaria, emphasis should be placed on the use of highly effective vaccines, increasing the treatment rate of infectious persons, the death rate of mosquitoes, and reducing the recruitment rate, biting rate of mosquitoes, the probability of transmission from humans to mosquitoes and probability of transmission from mosquitoes to humans. The findings of the study revealed that malaria spread can be curbed with the combined efforts of other preventive and treatment measures in conjunction with the use of highly efficacious vaccines. Hence, the public health makers should consider the above-mentioned measures in the fight against malaria to reduce its transmission.

Statements and Declarations

Funding

This work did not receive any specific grants from funding agencies in the public, commercial, or non-profit sectors.

Competing interests

The authors declare no conflicts of interest.

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