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The Impact of Antimalarial Drug on the Dynamics of Malaria Transmission Using Nonlinear Mathematical Model

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Abstract

Malaria remains a major threat in many parts of the tropical and subtropical regions of the world. Even though significant effort has been implemented through the use of treated nets, indoor residual spraying, and antimalarial drugs, the disease remains chronic in many endemic communities. This paper studies the impact of antimalarial drug on the dynamics of malaria transmission using a nonlinear mathematical model. The model incorporates both human host and mosquito vector populations. The human population is divided into susceptible, exposed, infected, and recovered classes, while the mosquito population is divided into susceptible, exposed, and infected classes. Epidemiological mathematical analyses are carried out on the model system, including the disease-free equilibrium, endemic equilibrium, basic reproduction number, and local asymptotic stability. Additional numerical and graphical analyses are included to show the behaviour of the system under different levels of antimalarial drug effectiveness. Using the threshold imposed by the basic reproduction number, it is found that if $R_0 < 1$, the disease dies out, while if $R_0 > 1$, the disease persists in the environment. The graphical results further show that antimalarial drug has a significant effect on reducing malaria infection when the drug effectiveness is sufficiently high.

Keywords: Malaria; antimalarial drug; nonlinear mathematical model; disease-free equilibrium; basic reproduction number; graphical analysis.

1. Introduction

Malaria is a life-threatening illness that affects millions of people all over the world [2]. The disease is transmitted when an infected female *Anopheles* mosquito bites a human, injecting parasites into the bloodstream and initiating the infection cycle. Malaria infection has an incubation period of about 7 to 30 days after an infective mosquito bite, and its symptoms include fever, chills, vomiting, diarrhoea, headache, and weakness. If malaria is not treated on time, it may result in severe complications and death [8].

According to malaria reports, global malaria cases increased from 227 million in 2019 to 241 million in 2020, while deaths also increased during the same period [8]. Sub-Saharan Africa bears the heaviest burden, accounting for most malaria cases and deaths, with children under five years being the most vulnerable. Nigeria remains one of the countries with the highest malaria burden in the world [8, 9].

Malaria infections are normally prevented through insecticide-treated bed nets, indoor residual spraying, intermittent preventive treatment, vector control, and effective antimalarial treatment [5, 9]. Artemisinin-based combination therapy is commonly used for uncomplicated malaria. Antimalarial drugs are important because they reduce the infectious period of infected humans and lower the parasite burden available for onward transmission to mosquitoes. However, the success of antimalarial drug intervention depends on early treatment, correct dosage, compliance, and absence of strong parasite resistance [3, 4].

Mathematical models have become significant tools for understanding the transmission of malaria infection and for suggesting possible control measures. Models help to explain the movement of individuals between susceptible, exposed, infected, and recovered classes, and also describe the role of mosquitoes in sustaining transmission [1, 6, 7]. In this study, the original nonlinear malaria model is retained, while additional analysis and graphical interpretation are added to strengthen the work.

2. Model Formulation

The model compartments are divided into seven sub-compartments namely susceptible human population $S_h(t)$, exposed human population $E_h(t)$, infected human population $I_h(t)$, recovered human population $R_h(t)$, susceptible mosquito population $S_m(t)$, exposed mosquito population $E_m(t)$, and infected mosquito population $I_m(t)$. The model is given

by

$$\frac{dS_h}{dt} = \pi_h + QR_h - \beta_h I_m S_h - \mu_h S_h, \tag{1}$$
$$\frac{dE_h}{dt} = \beta_h I_m S_h - (\delta_h + \mu_h) E_h, \tag{2}$$
$$\frac{dI_h}{dt} = \delta_h E_h + \gamma I_h - (\mu_h + \alpha + K\phi) I_h, \tag{3}$$
$$\frac{dR_h}{dt} = K\phi I_h - (\mu_h + Q) R_h, \tag{4}$$
$$\frac{dS_m}{dt} = \pi_m - \beta_m I_h S_m - (\mu_m + \tau) S_m, \tag{5}$$
$$\frac{dE_m}{dt} = \beta_m I_h S_m - (V_m + \mu_m + \tau) E_m, \tag{6}$$
$$\frac{dI_m}{dt} = V_m E_m - (\mu_m + \tau) I_m. \tag{7}$$

This system is retained as the original model structure. The antimalarial drug effect is represented by $K\phi$, where K is the human recovery rate and ϕ is the effectiveness of antimalarial drug. The term γI_h represents additional infection contribution as stated in the original model.

3. State Variables and Parameters of the Model

Table 1: State variables of the model.

Variable	Definition
S_h	Susceptible human population
E_h	Exposed human population
I_h	Infected human population
R_h	Recovered human population
S_m	Susceptible mosquito population
E_m	Exposed mosquito population
I_m	Infected mosquito population

Table 2: Parameters of the model.

Parameter	Definition
π_h	Recruitment rate of humans
π_m	Recruitment rate of mosquitoes
μ_h	Natural death rate of humans
μ_m	Natural death rate of mosquitoes
Q	Loss of immunity by recovered individuals
β_h	Transmission rate from infected mosquitoes to humans
β_m	Transmission rate from infected humans to mosquitoes
δ_h	Progression rate from exposed humans to infected humans
V_m	Incubation/progression rate of exposed mosquitoes
α	Disease-induced death rate
K	Human recovery rate
ϕ	Effectiveness of antimalarial drug
τ	Death rate of mosquitoes due to insecticide spray
γ	Additional infection contribution in the infected human class

4. Positivity and Boundedness of Solutions

For the model to be biologically meaningful, all state variables must remain non-negative. If any state variable is zero, the corresponding equation in (1)–(7) gives a non-negative inflow at the boundary, provided all parameters and initial conditions are non-negative. Hence, the solution of the model remains in the non-negative region.

Adding the human equations gives

$$\frac{dN_h}{dt} = \pi_h - \mu_h N_h - \alpha I_h + \gamma I_h, \tag{8}$$

where $N_h = S_h + E_h + I_h + R_h$. If $\gamma < \alpha + \mu_h$, the human population is bounded. Similarly, for mosquitoes,

$$\frac{dN_m}{dt} = \pi_m - (\mu_m + \tau)N_m, \tag{9}$$

where $N_m = S_m + E_m + I_m$. Therefore,

$$0 < N_m(t) \leq \frac{\pi_m}{\mu_m + \tau}. \tag{10}$$

Thus, the model is epidemiologically well posed in a positively invariant region.

5. Disease-Free Equilibrium

To solve for the disease-free equilibrium, all infected and exposed populations are set to zero. Therefore,

$$E_h = I_h = R_h = E_m = I_m = 0. \tag{11}$$

From equations (1) and (5),

$$S_h^0 = \frac{\pi_h}{\mu_h}, \qquad S_m^0 = \frac{\pi_m}{\mu_m + \tau}. \tag{12}$$

Hence, the disease-free equilibrium is

$$E_0 = \left(\frac{\pi_h}{\mu_h}, 0, 0, 0, \frac{\pi_m}{\mu_m + \tau}, 0, 0 \right). \tag{13}$$

6. Basic Reproduction Number

The basic reproduction number is the number of secondary infections produced by a single infective individual in an entirely susceptible population. To compute the basic reproduction number, the infected compartments are taken as

$$x = (E_h, I_h, E_m, I_m)^T. \tag{14}$$

The new infection terms and transfer terms are

$$\mathcal{F} = \begin{pmatrix} \beta_h I_m S_h \\ 0 \\ \beta_m I_h S_m \\ 0 \end{pmatrix}, \qquad \mathcal{V} = \begin{pmatrix} (\delta_h + \mu_h) E_h \\ (\mu_h + \alpha + K\phi - \gamma) I_h - \delta_h E_h \\ (V_m + \mu_m + \tau) E_m \\ (\mu_m + \tau) I_m - V_m E_m \end{pmatrix}. \tag{15}$$

At the disease-free equilibrium, the Jacobian matrices are

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_h S_h^0 \\ 0 & 0 & 0 & 0 \\ 0 & \beta_m S_m^0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \tag{16}$$

$$V = \begin{pmatrix} \delta_h + \mu_h & 0 & 0 & 0 \\ -\delta_h & \mu_h + \alpha + K\phi - \gamma & 0 & 0 \\ 0 & 0 & V_m + \mu_m + \tau & 0 \\ 0 & 0 & -V_m & \mu_m + \tau \end{pmatrix}. \tag{17}$$

Therefore,

$$R_0 = \rho(FV^{-1}), \tag{18}$$

and after simplification,

$$R_0 = \sqrt{\frac{\beta_h \beta_m S_h^0 S_m^0 \delta_h V_m}{(\delta_h + \mu_h)(\mu_h + \alpha + K\phi - \gamma)(V_m + \mu_m + \tau)(\mu_m + \tau)}}. \tag{19}$$

Using $S_h^0 = \pi_h/\mu_h$ and $S_m^0 = \pi_m/(\mu_m + \tau)$, this becomes

$$R_0 = \sqrt{\frac{\beta_h \beta_m \left(\frac{\pi_h}{\mu_h}\right) \left(\frac{\pi_m}{\mu_m + \tau}\right) \delta_h V_m}{(\delta_h + \mu_h)(\mu_h + \alpha + K\phi - \gamma)(V_m + \mu_m + \tau)(\mu_m + \tau)}}. \tag{20}$$

When $R_0 < 1$, the disease dies out. When $R_0 > 1$, the disease persists in the environment. The formula also shows that increasing ϕ decreases R_0, provided $\gamma < \mu_h + \alpha + K\phi$.

7. Disease Endemic Equilibrium

The endemic equilibrium is the point where the infected populations are not set to zero. Let

$$E^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*) \tag{21}$$

be the endemic equilibrium. From equation (3),

$$E_h^* = \frac{\mu_h + \alpha + K\phi - \gamma}{\delta_h} I_h^*. \tag{22}$$

From equation (4),

$$R_h^* = \frac{K\phi}{\mu_h + Q} I_h^*. \tag{23}$$

From equation (5),

$$S_m^* = \frac{\pi_m}{\beta_m I_h^* + \mu_m + \tau}. \tag{24}$$

From equation (6),

$$E_m^* = \frac{\beta_m I_h^* S_m^*}{V_m + \mu_m + \tau}. \tag{25}$$

From equation (7),

$$I_m^* = \frac{V_m E_m^*}{\mu_m + \tau}. \tag{26}$$

Also, from equation (1),

$$S_h^* = \frac{\pi_h + Q R_h^*}{\mu_h + \beta_h I_m^*}. \tag{27}$$

This gives a biologically meaningful endemic equilibrium only when all components are positive. In particular, the condition

$$\mu_h + \alpha + K\phi - \gamma > 0 \tag{28}$$

is necessary for a positive exposed human class. If $\gamma = 0$, the usual threshold condition shows that the endemic equilibrium exists when $R_0 > 1$.

8. Jacobian Matrix at Disease-Free Equilibrium

The Jacobian matrix of the model at the disease-free equilibrium is obtained by linearizing equations (1)–(7). The infection subsystem is governed by the matrices F and V above. By the next-generation matrix method and the Routh-Hurwitz criterion, the disease-free equilibrium is locally asymptotically stable if

$$R_0 < 1, \tag{29}$$

and unstable if

$$R_0 > 1. \tag{30}$$

At $R_0 = 1$, a threshold change occurs. This corresponds to a transcritical bifurcation between the disease-free and endemic states. If $\gamma > 0$, the effective removal term in the infected human equation is reduced from $\mu_h + \alpha + K\phi$ to $\mu_h + \alpha + K\phi - \gamma$. Hence, the effective threshold condition becomes

$$R_0 < 1 \quad \text{and} \quad \gamma < \mu_h + \alpha + K\phi. \tag{31}$$

If $\gamma \geq \mu_h + \alpha + K\phi$, then the disease-free state is not stable because infected humans do not decline sufficiently.

9. Numerical Simulation and Graphical Analysis

Numerical simulation is carried out to illustrate the analytical results of the original model. The parameter values used are hypothetical but epidemiologically reasonable for demonstrating the qualitative behaviour of the model. They are not intended to represent a specific field survey. The purpose of the graphical analysis is to show how antimalarial drug effectiveness influences the infected human class, the infected mosquito class, and the basic reproduction number.

Table 3: Human parameter values used for simulation.

π_h	μ_h	Q	β_h	δ_h	α	K	γ
20	0.02	0.04	0.000035	0.20	0.01	0.35	0

Table 4: Mosquito parameter values used for simulation.

π_m	β_m	μ_m	τ	V_m
120	0.000020	0.10	0.05	0.25

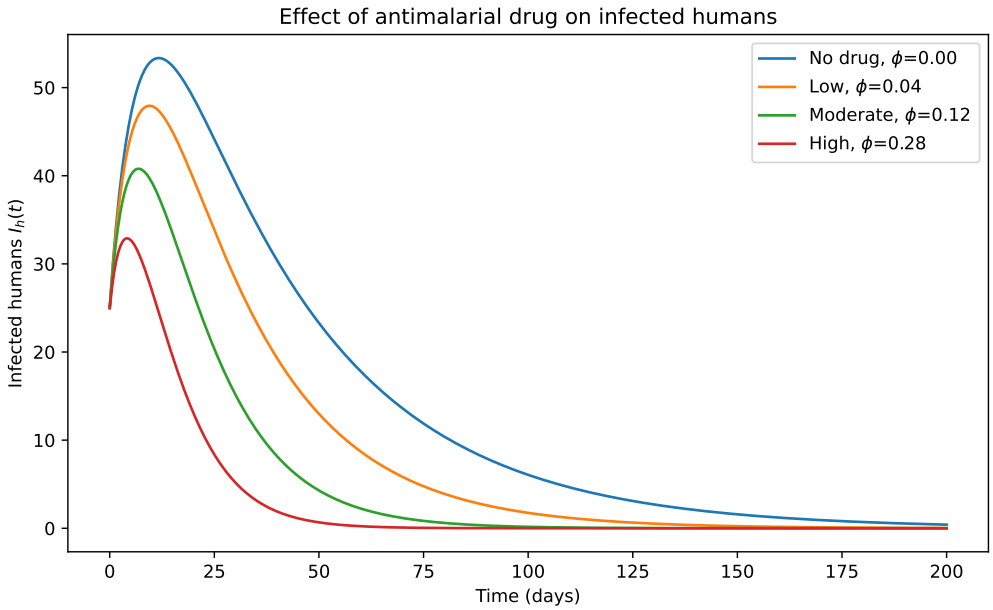

Figure 1: Comparison of infected humans under different levels of antimalarial drug effectiveness.

Figure 1 shows that the infected human population decreases faster as the effectiveness of antimalarial drug increases. In the absence of drug intervention, infected individuals remain longer in the infectious class. When the drug parameter increases, infected humans recover more rapidly, and the infection curve declines earlier.

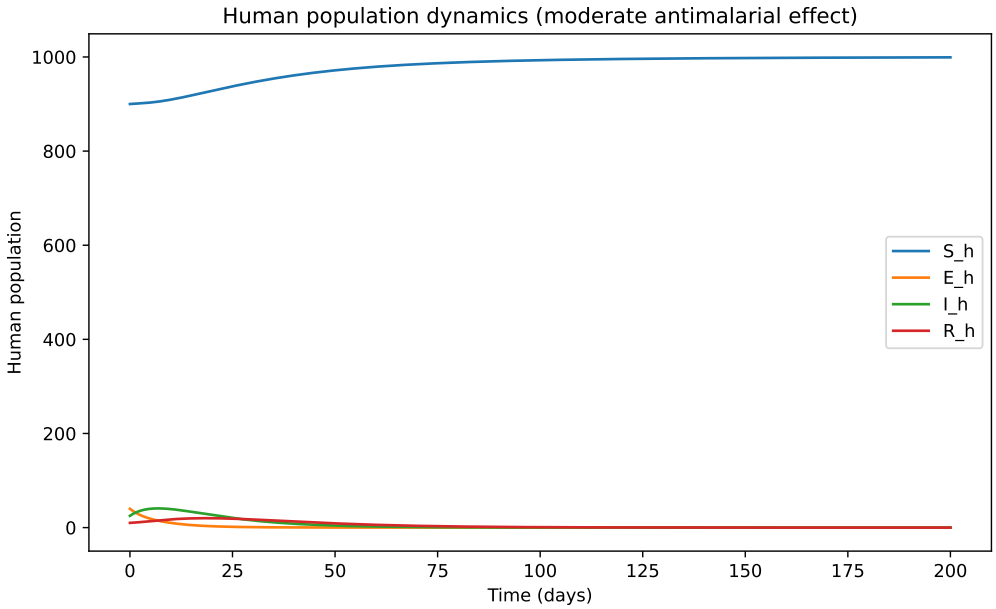

Figure 2: Human population dynamics under moderate antimalarial drug effectiveness.

Figure 2 shows the movement of humans between susceptible, exposed, infected, and recovered classes. The exposed and infected classes decline with time, while the susceptible class approaches a stable level. This supports the analytical result that reducing the infectious period helps to drive the system towards disease elimination when $R_0 < 1$.

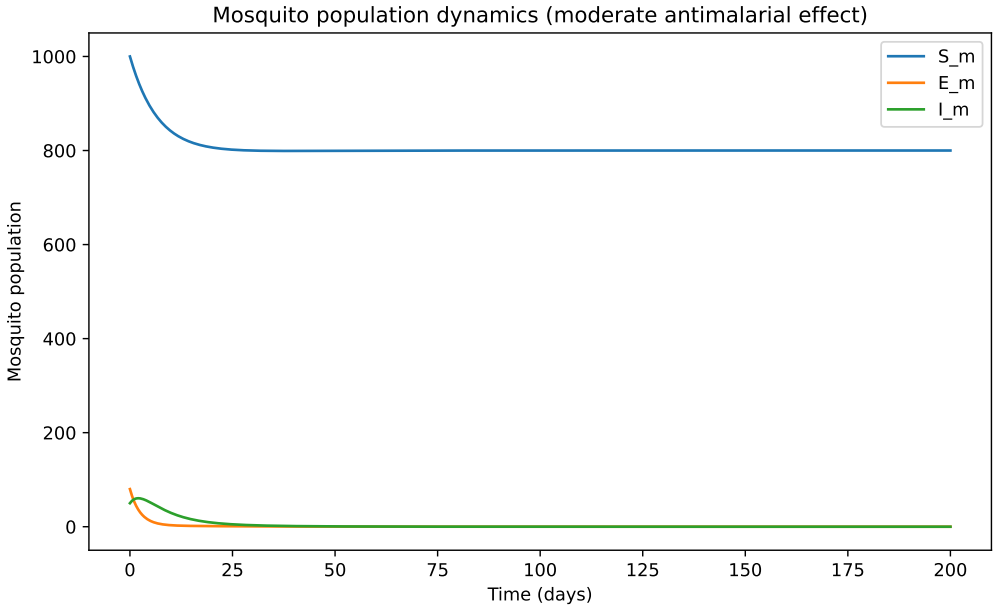

Figure 3: Mosquito population dynamics under moderate antimalarial drug effectiveness.

Figure 3 indicates that mosquito infection also declines when the infected human population declines. Since mosquitoes acquire malaria from infected humans, effective treatment in humans indirectly reduces exposed and infected mosquitoes.

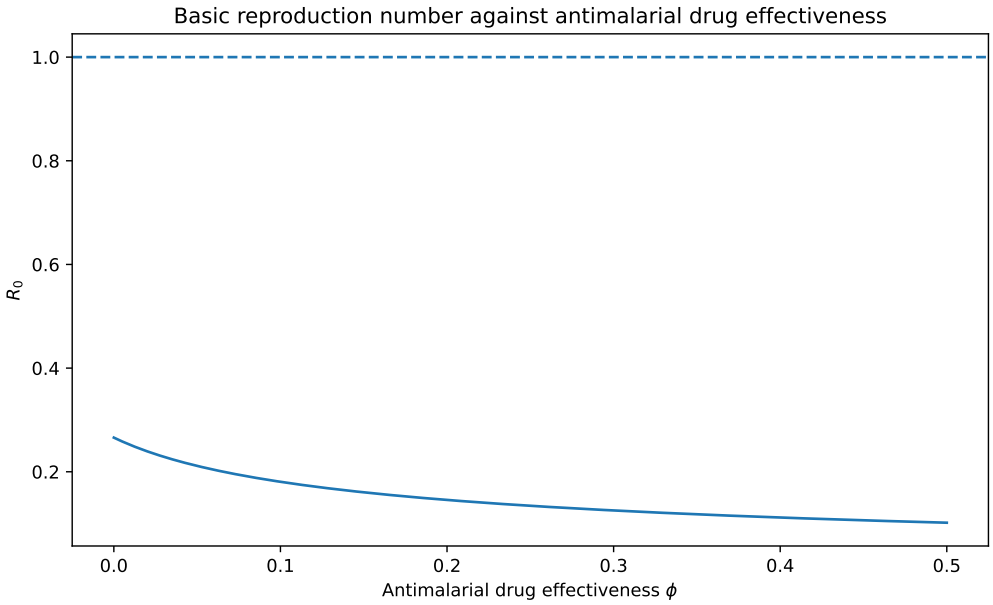

Figure 4: Basic reproduction number against antimalarial drug effectiveness.

Figure 4 shows that R_0 decreases as ϕ increases. This agrees with equation (19), where ϕ appears in the denominator through the term $\mu_h + \alpha + K\phi - \gamma$. Therefore, stronger antimalarial drug effectiveness reduces the expected number of secondary infections.

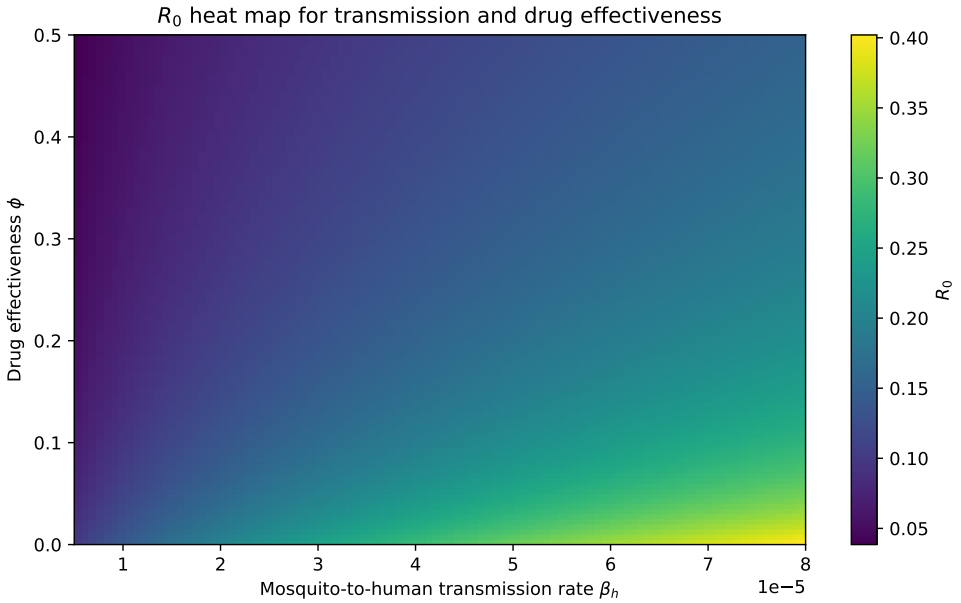

Figure 5: Heat map of R_0 for mosquito-to-human transmission rate and antimalarial drug effectiveness.

Figure 5 shows the combined effect of transmission and drug intervention. High mosquito-to-human transmission increases R_0, while high drug effectiveness decreases R_0. The contour line $R_0 = 1$ separates the disease-free region from the persistence region.

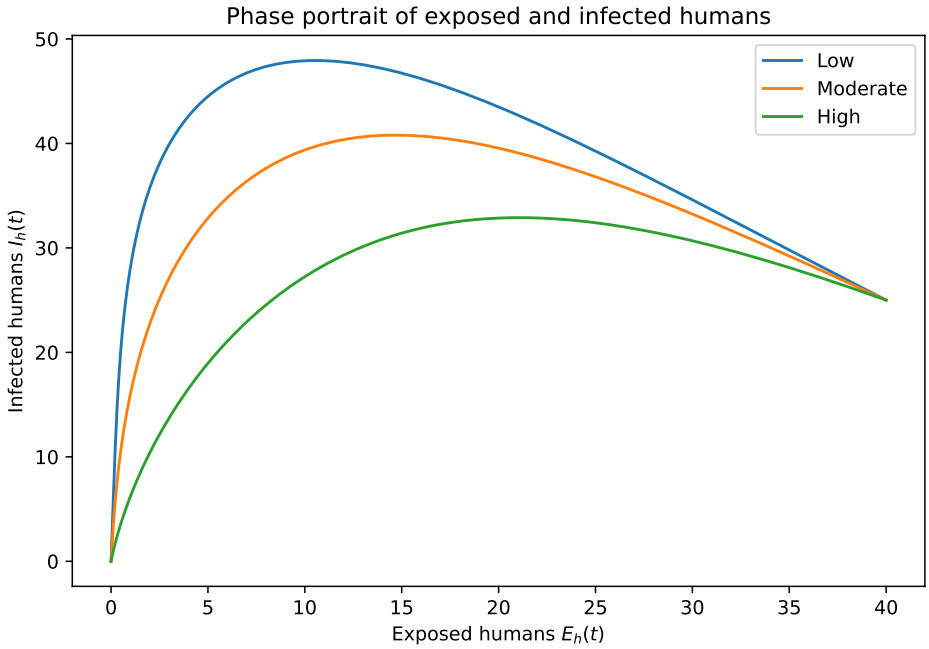

Figure 6: Phase portrait of exposed and infected human classes.

Figure 6 shows the phase portrait of exposed humans against infected humans. The curves move toward lower infection levels as treatment effectiveness increases. This confirms that antimalarial drug reduces both the exposed and infected human populations.

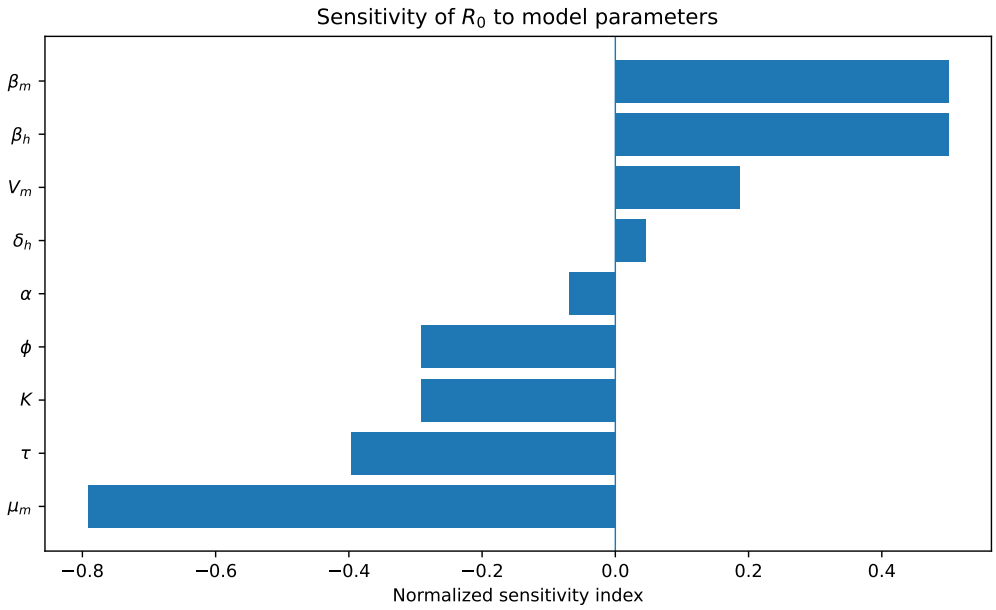

Figure 7: Sensitivity of R_0 to selected model parameters.

Figure 7 shows that parameters that increase transmission have positive sensitivity indices, while parameters that increase removal or reduce infection have negative sensitivity indices. The drug effectiveness parameter has a negative effect on R_0, meaning that an increase in antimalarial drug effectiveness decreases malaria transmission.

10. Findings

The findings from this study are as follows. The study establishes the local asymptotic stability of the disease-free equilibrium point of the model. The study also deduces the basic reproduction number, and it is found that when $R_0 < 1$, malaria dies out, while when $R_0 > 1$, malaria persists in the environment. The graphical analysis shows that increasing antimalarial drug effectiveness reduces the infected human population, reduces infected mosquitoes, and lowers the reproduction number. The heat-map analysis further indicates that drug intervention must be supported by reduction in mosquito-to-human transmission in order to achieve stronger malaria control.

11. Conclusion

This study analysed the impact of antimalarial drug on the dynamics of malaria transmission using a nonlinear mathematical model. The model comprises susceptible, exposed, infected, and recovered human populations together with susceptible, exposed, and infected mosquito populations. The disease-free equilibrium, endemic equilibrium, basic reproduction number, and stability conditions were investigated. The analysis shows that when $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable and malaria dies out. When $R_0 > 1$, malaria persists in the environment. Numerical simulation and graphical analysis show that antimalarial drug has a significant effect on malaria transmission because increasing drug effectiveness reduces infected humans and infected mosquitoes. Therefore, effective antimalarial treatment, when combined with vector control measures such as insecticide spraying and treated nets, can greatly reduce malaria transmission.

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Conflict of Interest

The authors declare no conflict of interest.

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